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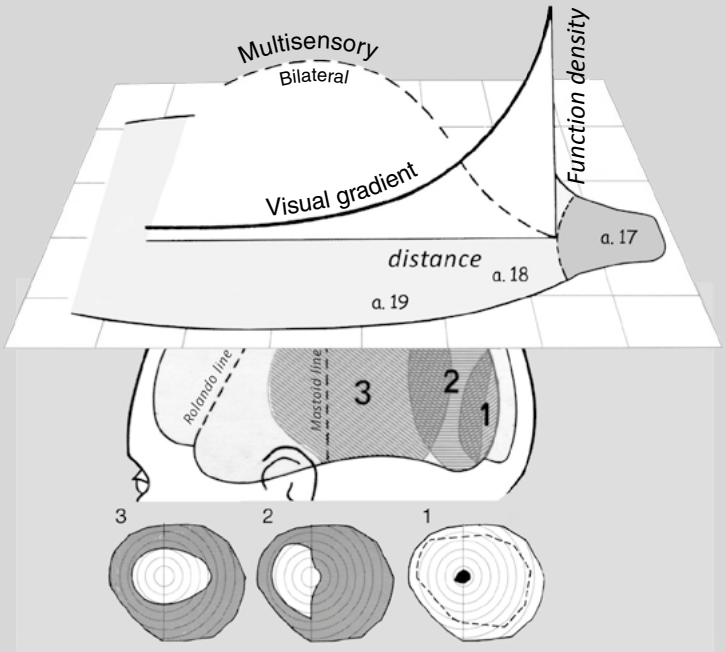
Brain Dynamics

The brain activity according to the dynamic conditions of nervous excitability

Justo Gonzalo

Isabel Gonzalo Fonrodona (ed.)



CONSEJO SUPERIOR DE INVESTIGACIONES CIENTÍFICAS

Justo Gonzalo y Rodríguez-Leal (Barcelona, 1910-Madrid, 1986) graduated in Medicine in Madrid (1933), and specialized in neurology in Austria and Germany (1933-1935) with H. Hoff, O. Pötzl and K. Kleist, funded by the Junta para Ampliación de Estudios (JAE). He worked as a clinical neurologist, and also conducted anatomoclinical research at the Cajal Institute (Madrid 1936-1937). He was assigned as a physician in the Republican front (1937) during the Spanish Civil War (1936-1939), and as a neurologist in a military hospital of Valencia (1938-1939), where he identified peculiar phenomena in some of the war brain-injured patients.This was the beginning of a broad and novel research that Gonzalo developed on the human cerebral cortex. The first results were presented to the Consejo Superior de Investigaciones Científicas (CSIC) in 1941, institution to which he was adscribed until his retirement. He was head of the Laboratory of Brain Pathophysiology at the Cajal Institute, in the Faculty of Medicine in Madrid, where he examined a large number of brain-injured subjects, and taught doctoral courses on Brain Pathophysiology (1945-1966) also exposing his research. His two-volume book in Spanish on Brain Dynamics was well received internationally, and awarded by the Real Academia Nacional de Medicina (1950) and the Sociedad Española de Psicología (1958). His whole life was inseparable from study and research, delving into disciplines such as biology, physics, mathematics and philosophy.

Isabel Gonzalo Fonrodona (Madrid, 1949), PhD in Physics from the Complutense University of Madrid (UCM), is currently professor emeritus at the UCM, in the Optics Department of the Faculty of Physics, where she has developed an extensive teaching and research work in fields such as Optics, Quantum Physics, Molecular Physics and Photonics, publishing her reseach in international journals. She continues her research activity in Quantum Physics and Optics. As connoisseur of Justo Gonzalo's research, she has also published several works on his research, presenting many of them at international meetings, and has edited the present book.

Cover figure: Schematic of functional cortical gradient for vision, multisensory curve, sites of injuries and visual syndromes (adapted from a figure by Justo Gonzalo).

BRAIN DYNAMICS

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Brain Dynamics

The brain activity according to the dynamic conditions of nervous excitability

Volumes 1 and 2

Supplements I and II

by

Justo Gonzalo

Compiled and edited by

Isabel Gonzalo Fonrodona

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Preface

THIS book is the first English translation of the Spanish book published in 2010 that included the facsimile edition of Volume 1 (1945) and Volume 2 (1950) of the book 'Dinámica Cerebral' [Brain Dynamics] by Justo Gonzalo, a 1952 article by the same author (as Supplement I) and the first edition of some of his later contributions (as Supplement II).[1]

The interest of the research described here lies in the fact that it is surprisingly of current relevance, apart from its undoubted historical interest. Some aspects were ahead of discoveries that were made later. It is remarkable that some of the phenomena exposed are still unknown, others have only been observed in the last decades, and the functional dynamic unity of the cortex proposed by the author is closely related to the current trends in the study of the brain.

Justo Gonzalo y Rodríguez-Leal (Barcelona, 1910 – Madrid, 1986) after completing his medical studies at the University of Madrid, specialized in neurology at the University of Vienna (1933-34) with Hans Hoff and Otto Pötzl, and in brain pathology at the University of Frankfurt (1934-35) with Karl Kleist funded by the Junta para Ampliación de Estudios e Investigaciones Científicas (JAE) [Comitee for extension of Studies and Scientific Research]. The research presented here began under the adverse conditions of the Spanish Civil War (1936-1939) at a neurological hospital in Valencia (Spain), and was later continued in Madrid (Spain) at the S. Ramón y Cajal Institute and the Consejo Superior de Investigaciones Científicas [Spanish National Research Council], institution to which he was adscribed until his retirement. More data about the author and his research, and publications by various authors on his contributions from a historical and scientific perspective are available in open digital support.[2]

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[1] GONZALO, J. (2010). *Dinámica cerebral. La actividad cerebral en función de las condiciones dinámicas de la excitabilidad nerviosa.* Edición facsimilar del Volumen 1 (1945), Volumen 2 (1950), Suplemento I (1952) y 1.ª edición del Suplemento II. I. Gonzalo Fonrodona (ed. de los suplementos). Publicado por la Red Temática en Tecnologías de Computación Natural/Artificial (RTNAC) y la Universidad de Santiago de Compostela, España 2010. Open Access: http://hdl.handle.net/10347/4341

[2] https://en.wikipedia.org/wiki/Justo_Gonzalo

From the study of brain-injured patients with unilateral lesion in an association area in the left parieto-occipital cortex, equidistant from the visual, tactile and auditory projection areas, the author characterized a multisensory, bilateral and symmetrical disorder that he called *central syndrome*. This is a multisensory (visual, tactile, auditory) alteration with the lesion not involving the specific areas, all functions being affected, from simple excitability to more complex functions, bilaterally and symmetrically, all being incompatible with the rigid traditional theory of brain localization. The syndrome has a clear dynamic character, such as the separation or disaggregation of a sensory function into partial functions or responses as the intensity of the stimulus decreases. These partial functions are united in normal perception, and are gradually lost according to their demands of nervous excitability, thus revealing the different components that make up the sensorium. One of these components turns out to be the orientation of the visual image. Thus, inverted or tilted vision appears, together with other disorders such as degradation of the visual image regarding shape, size and color. The first in-depth study of tilted or inverted vision is part of this research. A related dynamic phenomenon is the partial disappearance of the disorders by intensification of the stimulus, or by the phenomenon of facilitation according to which the perception of a stimulus is improved by the presence of another stimulus of the same or of a different sensory modality (cross-modal effect), or by a motor stimulus, muscular effort being one of the most efficient and least known means. For example, a strong muscular contraction can improve perception by straightening and improving the visual image. This capability is greater as the brain excitability deficit increases. The first detailed study on multisensory and motor facilitation, providing new observations and an interpretation of this effect, is also part of this research. Noticeably, multisensory facilitation is an extremely current research topic, as evidenced by the large number of scientific articles.

From the new approach that the author gave to the research, his conception of brain dynamics emerged.[3] To the best of my knowledge, this was the first time that the term 'brain dynamics,' so widely used today, was introduced to describe brain mechanisms in relation to sensory organization. As the author pointed out, this research filled the gap then existing between brain pathology and the physiology of the nervous system, since the phenomena were explained on a physiological basis governed by the laws of nervous excitability. This provided a dynamic solution to the rigid theory of brain localization, and established a continuous transition between lower and higher sensory functions, all based on the same physiological laws, thus highlighting the functional unity of the cortex.

As prototypes of the central syndrome, two patients with brain lesions presenting different loss of neural mass in the same area and different intensity in their disorders were specially studied. The deep and detailed analysis of these patients

[3] In 1941, Justo Gonzalo submitted a 95-page report to the Consejo Superior de Investigaciones Científicas [Spanish National Research Council] entitled: "Investigaciones sobre Dinámica Cerebral. La acción dinámica en el sistema nervioso. Estructuras sensoriales por sincronización cerebral" [Investigations on Brain Dynamics. Dynamic action in the nervous system. Sensory structures by brain synchronization], which was awarded by this institution.

allowed the author to generalize the concept of brain dynamics. In subsequent research, the author found 35 cases that also fit the central syndrome.[4] A reference case is the famous Schneider case of Goldstein and Gelb studied in 1918 and 1919, which deserves publications even at present, and which the author re-interpreted as a case of central syndrome.

As the author pointed out, the scarcity of cases of central syndrome is not due to their exceptionality, as they would be numerous, but because they remain hidden due to the difficulty in examining this type of patients. They are usually unaware of their own anomalies, and they unconsciously develop facilitation mechanisms that improve their perception. In fact, as noted by the author, the observations were not understood and quantified until permeability to both facilitation and iteration became evident in this syndrome.

In Volume 1, the first part deals with general aspects of the research (findings, new syndrome, methodology, etc.). The second part of this volume deals with the experimental and quantitative analysis of sensory dynamics of visual functions (excitability, colors, shapes, image orientation, schema). Some singular phenomena are described with extreme detail, such as inverted vision, facilitation, delocalization of colors, reversal of motion and orientation disorder. Concerning schema, a striking phenomenon of spatial orientation is that these patients were able to read a text with the same ease whether it was in a normal position or upside down without noticing any difference in orientation. Also remarkable is the alteration in allocentric orientation.[5]

Volume 2 deals with the analysis of sensory dynamics of tactile functions (general excitability, sensations, tactile space, orientation, schema) in central syndrome and extension of concepts. For example, among other noteworthy phenomena, tactile inversion is observed and studied in detail. Inverted perception is thus generalized in the central syndrome to all sensory systems of a spatial nature once confirmed in the auditory system. There was no precedent in the literature for this type of tactile and auditory inversion, and there is still none. This type of tactile inversion, more difficult to detect than the visual one, must be distinguished from the often described tactile allochiria. In the process of tactile localization of a stimulus, up to five phases are distinguished as stimulation increases, from simple sensation to specific localization passing through the inversion phase. As in vision, this process is described as a spiral development of the sensory field since in the inversion phase there is a marked deviation towards the midline of the body (or towards the center of the visual field).[6] Tactile inversion is studied for both cutaneous and joint stimulation including complex processes such as walking. Analogous to vision, a continuity is also found between

[4] Some cases are reported in: GONZALO, J. (1952). "Las funciones cerebrales humanas según nuevos datos y bases fisiológicas" [Human brain functions according to new data and physiological basis], *Trabajos del Instituto Cajal de Investigaciones Biológicas,* XLIV: 95-157. Its English translation is Supplement I of the present edition. Other cases are reported in Supplement II.

[5] At the beginning of the analysis of sensory dynamics, the author states that the research described in both volumes is original, and whenever a precedent exists, it is duly indicated.

[6] See also Supplement I and the article: GONZALO, J. (1951). "La cerebración sensorial y el desarrollo espiral" [Sensory cerebration and spiral development], *Trabajos del Instituto Cajal de Investigaciones Biológicas,* XLIII: 209-260.

sensory functions and gnosis, since they are all based on the same physiological laws. In this volume, Gerstmann's syndrome is discussed and put in relation to the central syndrome. The schema function is studied in detail, and considered in varying degrees according to the somatic model, postural model and praxis model. The text parts in small print, as in the original, include experimental details and supplementary data.

Particularly remarkable is the author's description of the careful and thorough way of examining patients in order to reveal varied and unusual phenomena that help shed light on sensory processing. Also noteworthy is the rich bibliographic documentation on various schools of thought, clinical data, experiments and precedents related to the observed phenomena, which adds interest and amenity to the book.

As in the 2010 Spanish edition, in the present edition the two volumes are presented together, as conceived by the author, and with complete bibliographical references. Supplements I and II provide an overview of the author's subsequent research, which completes and unifies the work as a whole.

The original book of 1945 received the attention of relevant authors at that time. For example, Köhler (representative of the Gestalt theory) wrote in a letter to Gonzalo:[7] "*The book contains many observations which are both entirely new and very important. I also believe that at several points your interpretations are more convincing than those of Gelb and Goldstein.*" Also Bing (Prof. of neurology at the University of Basel, Switzerland) wrote to Gonzalo:[8] "*It is a work of the utmost importance and originality, worthy of the traditions established by the immortal sage* (Ramón y Cajal) *to whose memory it has been dedicated*" (translated from French). Some extracts from the review in the journal edited by Buscaino are:[9] "*The book is very rich in objective observations, most of them original and of great interest. It is also rich in theoretical deductions ... . A series of very interesting and important facts … . Particularly noteworthy is the phenomenon of tilted or inverted vision, this being the first case in the international literature of almost chronic duration ...*" (translated from Italian). Soon after, Bender and Teuber, in a work on visual functions state:[10] "*Thus far, the American and English literature has failed to produce a monograph similar in scope to Gonzalo's Dinámica Cerebral which was based on experiments with brain injured casualties of the Spanish Civil War.*" Other authors such as Ajuriaguerra and Hécaen (1949), Guiraud (1950), Critchley (1953), devoted attention to this research in their specialized books.[11] The two-volume book soon went out of print and was not reissued until the facsimile edition of 2010.

In a subsequent publication in 1952, here Supplement I (see footnote 4), the author proposed, on the basis of new cases, a model of functional gradients across the cerebral cortex. The specificity of the cortex is thus understood as distributed,

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[7] Family archive: Letter from W. Köhler to J. Gonzalo, 1946.

[8] Family archive: Letter from R. Bing to J. Gonzalo, 1946.

[9] VIEMBI, (1946). Recensioni, *Acta Neurologica (Napoli),* Anno I, 5: 368-371.

[10] BENDER, M. B. and TEUBER, H. L. (1948). "Neuro-ophtalmology," in E.A. Spiegel (ed.) *Progress in Neurology and Psiquiatry,* III, Chap. 8: p. 171.

[11] AJURIAGUERRA, J. and HÉCAEN, H. (1949). *Le cortex cérébral. Étude neuro-psycho-pathologique,* Paris: Masson 1949.

GUIRAUD, P. (1950). *Psychiatrie Général,* Paris: Le François 1950.

CRITCHLEY, MCD. (1953). *The Parietal Lobes,* London: Arnold 1953.

in continuous gradation, in accordance with the continuous transition observed between the central syndrome and other cortical syndromes. This allowed the author to interpret other syndromes.[12] The concept of brain gradients is now highly topical and based on imaging techniques.

Subsequently (1952-54), the author selected, among a large number of brain-injured patients, nearly 200, mostly wounded from the Spanish Civil War. This gave him further support for the central syndrome, for the concept of cortical gradients, and for other concepts he introduced from dynamic systems, such as similarity and allometry. The latter led him to find different (allometric) relationships between different sensory functions, both in their loss and recovery.

The concepts of gradient, similarity and allometry were presented by the author from 1951 onwards in the doctoral courses on brain pathophysiology at the University of Madrid (1945-66), and are now part of the contents of Supplement II. This supplement also includes 30 selected illustrations with their explanations, and was published for the first time in the Spanish edition of 2010. In the present edition, additional information has been included in the explanatory texts of such illustrations.

Data and concepts of this research have been exposed and developed in a more recent context by other authors.[13] This research was also echoed in the field of cybernetics and artificial intelligence, where some authors consider this research together with that of K. S. Lashley and A. R. Luria as a neurophysiological basis for the functional organization of nervous tissue and behavior.[14] It is in this context that

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[12] As the author points out in the Introduction to his book, several syndromes resulting from lesions in cortical areas beyond the projection pathways would belong to some extent to a more or less pure central syndrome.

[13] For example: GONZALO, I. (1997). "Allometry in the Justo Gonzalo's model of the sensorial cortex," *Lect. Not. Comp. Sci. (LNCS),* **1240**: 169-177.

ARIAS, M. and GONZALO, I. (2004). "The neuroscientific work of Justo Gonzalo: the center syndrome and reversal metamorphopsia," *Neurología,* **19** (8): 429-433.

GONZALO-FONRODONA, I. (2007, 2009). "Inverted or tilted perception disorder," *Rev. Neurol.,* **44** (3): 157-165 (2007). "Functional gradients through the cortex, multisensory integration and scaling laws in brain dynamics," *Neurocomputing,* **72**: 831-838 (2009).

Open Access: https://doi.org/10.1016/j.neucom.2008.04.055

GONZALO-FONRODONA, I. and PORRAS, M. A. (2014). "Nervous excitability dynamics in a multisensory syndrome and its similitude with normals. Scaling Laws," in A. Costa and E. Villalba (eds.) *Horizons in Neuroscience Research,* Vol. 13, Chap. 10: 161-189.

OA: https://novapublishers.com/wp-content/uploads/2019/06/978-1-62948-426-6_ch10.pdf

GARCÍA-MOLINA, A. (2015). "Justo Gonzalo's groundbreaking contributions to the study of cerebral functional organization," *Neurosciences and History,* **3** (2): 61-67.

OA: *https://nah.sen.es/vmfiles/abstract/NAHV3N2201561_67EN.pdf*

GARCÍA-MOLINA, A. and GONZALO-FONRODONA, I. (2023). "Redescubriendo al paciente M: Justo Gonzalo y su teoría de dinámica cerebral", *Rev. Neurología,* **76** (7): 231-241.

OA: *https://neurologia.com/articulo/2023062*

[14] For example: DELGADO, A. E. (1978). *Modelos neurocibernéticos de Dinámica Cerebral,* PhD thesis, Polytechnic University of Madrid.

MIRA, J. and DELGADO, A. E. (2003). "Neural modeling in cerebral dynamics," *Biosystems,* **71**: 133-144.

the 'Red Temática en Tecnologías de Computación Natural/Artificial (RTNAC),' together with the University of Santiago de Compostela (Spain), published in 2010 the aforementioned Spanish edition, cited at the beginning of this Preface whose open online version maintains a significant readership.

The present English translation aims to expand the dissemination of this pioneering research, in the hope that the inquiring minds of scientists will appreciate the originality and profoundness of a research of current interest. Obviously, the text should be understood in the context of the time it was written, particularly the expressions of a temporal character.

The text that appears in the figures has been translated maintaining the original figures even if some of them are not of high quality. Changes with respect to the 2010 edition are: this Preface (specific to this edition), the tables of contents of Volumes 1 and 2 together, and the author's Introduction to these volumes as a synthesis of the author's prefaces, all to highlight the continuity and unity between the volumes. Regarding the original edition, let's mention that at the beginning of Volume 1 (1945) the author wrote the following dedication: "To the memory of the great researcher of the nervous system Santiago Ramón y Cajal" (translated from Spanish).

I thank José R. Álvarez, José M. Ferrández and Félix de la Paz (founders of RTNAC), as well as the University of Santiago de Compostela, all publishers of the Spanish edition of 2010, for their kind assistance in providing the information and permissions necessary for the present edition. I am also grateful for the work of inserting the English text in the figures, done by Cristina Teijeiro in Vols. 1 and 2 and by Barbara Wang in Supplement II, the help of Celia Blanco de Torres during the translation of the first part of Supplement II, and the work of Enrique Barba Gómez and Leopoldo Ortiz in improving many of the figures. I also wish to thank Pura Fernández for her helpful and kind advice when I proposed this publication, as well as the researches Manuel Arias Gómez, Ana E. Delgado García, Alberto García-Molina and Jesús Hernández Gallego for their support in expressing the relevance and suitability of publishing this edition. I am especially grateful to Miguel A. Porras for his continuous and highly valuable advice throughout the process of translation and preparation of this edition. Finally, I am very grateful to the Consejo Superior de Investigaciones Científicas (CSIC) [Spanish National Research Council] for making this publication possible through 'Editorial CSIC.'

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BRAIN DYNAMICS

The brain activity according to the dynamic conditions of nervous excitability

Volumes 1 and 2

by

Justo Gonzalo

Introduction[1]

It is noteworthy the remarkable divergence between the conceptions of the physiology of the nervous system and those of brain pathology, due to the lack of physiological basis of the current brain theory, based on an anatomical criterion.

The brain-injured cases presented here are totally incompatible with the brain theory currently in use in brain pathology. By studying the phenomena in detail, this research has spontaneously led to a strictly physiological conception. Thus, the whole pathological process has been understood on the basis of very simple conceptions and laws of nervous excitability, within the laws of the general physiology of the nervous system. Phenomena that at first seemed exceptional and unacceptable in brain pathology, duly studied, become the general rule of brain functioning in man, these phenomena constituting an *experimentum crucis* that has determined the basis of brain activity.

Establishing a fundamental concept through a few cases is justified when the analysis of these cases has been deep enough to get to the roots of the issue. According to the theory of localization of brain functions, the character of each pathological case depends entirely on the specific site –damaged by the lesion– that houses a given function. However, when the physiological laws governing the general functioning of the brain are established, we see that in different cases, the different pathological manifestations are variants of a general type differing from each other quantitatively, and a gradual transition from one case to another can be observed. The discovery of the phenomena of dynamic action in the cases presented here (modifications of brain excitability by subordination and repercussion

[1] Considering here the two volumes as a whole and in order to avoid some repetitions, this introduction is a synthesis of the following original texts written by the author: preface and introduction at the beginning of Vol. 1 (1945) and preface and final Note of Vol. 2 (1950). Such final Note is a summary of the research carried out up to 1950. Yet, sections 1-3 of Vol. 1 provide a detailed introduction to this research and its meaning. In addition, a summary of the research contained in these two volumes is given in sections 1-6 of the 1952 article (Supplement I).

effects, asynchronism, summation actions, etc.) entails such an essential change that it leads to the establishment of a new conception of brain theory. This conception is *brain dynamics*, in which the functional unity resulting from the interrelation of the whole brain mass is crucial. Moreover, the singular dynamic conditions of nervous excitability, originated by a brain injury, open the way to the experimental method, which is the only way to properly address the mechanism of brain functions and their quantitative evaluation by determining the level of excitability of each brain process.

The dynamic conception constitutes a physiological conception. The static conception hardly leaves the simply anatomical approach. The physiological conception is broader and simpler than the anatomical one. The latter, rightly considered, is only valid for a small number of cases of a given type and, even then, only as a first approximation. For many other cases it is insufficient, its generalization being an abuse of the relationship between a given function and the anatomical structure that presumably houses it. Such a relationship is extremely complex and problematic, and could only be established in extreme cases. The transition from a static to a dynamic conception, i.e., from topographical anatomical consideration to physiological functional understanding by changes in nervous excitability in the brain, falls within the natural development of sciences, where purely static explanations must give way to functional or dynamic ones.

Within the back and forth that the scientific process sometimes presents, old ideas that have already been discarded regain relevance, and thus the ideas of Flourens (1824) on brain activity might now be rehabilitated. These ideas propose the functional unity of the cerebral cortex, which implies a firm opposition to anatomical localizations as in the phrenology of his time. However, no conception is completely excluded in the course of scientific development. Different theories are rather constructions based on incomplete observations and extreme views of a same thing. Rather than an absolute return to old doctrines already forgotten, it is matter of considering them under new and more precise aspects of a more general scope. The problem of brain localization, which has almost completely dominated brain theory, has always given rise to numerous discussions, considerably exacerbated over time. Thus, numerous authors from very diverse sectors have expressed their opposition to the anatomical criterion of brain localization and in favor of the functional or dynamic point of view, such as Bethe and Fischer (1931), Monakow (1914 a, 1914 b), Head (1918, 1920, 1923), Goldstein (1910), Stein (1928, 1930), Weizsäcker (1923, 1931), Lashley (1929, 1933, 1937) etc., among the most significant up to the time of presenting this research. But it should also be noted that on the side of the supporters of localization we have the great work of Kleist (1934), of the purest orthodox character as regards anatomical localizations and construction of brain maps. This proves that this is not such an easy question to resolve. A brain theory of functional character, opposed to the localizations, was developed by Jackson (1884) when studying the evolution and dissolution of the nervous system. The fact is that a sufficiently clear and coherent solution has never been reached. It should be noted that Goldstein (1910), known for his profound analyses of brain functions and his justified and important arguments against the anatomical theory of localiza-

tion, has missed the exceptional opportunity presented to him in one of his patients to provide compelling arguments, as will be analyzed here.

The two most notable features of the localization theory, the specific centers and the nosological syndromes, both the result of anatomical and psychological "atomism," are not capable of giving a correct interpretation of known facts. If we add to this the dynamic manifestations found in our cases, which force a functional and physiological interpretation, the classical traditional theory remains, at best, very insufficient. In the brain dynamics that we shall describe, brain activity constitutes a functional unit in which there is no room for autonomies, since cortical lesions affect the entire brain system, according to certain laws of nervous organization.

As for the structure of sensory activities and the nervous mechanism underlying them, the traditional classical theory always refers to the principle of the specificities of the centers which, by themselves or by their associations, account for all types of structures. The result is the primary and secondary identifications of Wernicke (1874, 1895), the associative disorders such as agnosia, aphasia, apraxia, etc., as a means of explaining the alteration of the most complex brain functions. On the other hand, in some studies that focus more on the functional analysis of symptoms, there is a tendency to consider sensory activity as the result of a progressive differentiation without resorting to any kind of associations. However, nothing is indicated regarding the conditions of the nervous mechanism that performs such differentiation.

The task of dealing with sensory organization in its two aspects, phenomenological and physiological, fills the major part of the research presented here, and probably constitutes the most original contribution together with the theory of the dynamic brain system, evidenced by the type of overall repercussion of the lesions.

With regard to the nervous mechanism in brain activity, the notion of isochronism of Lapicque (1934 a) is applied with great benefit. Within the physiological conception, a pathological alteration only results in a reduction or simplification of the brain function, i.e., a lower level of organization due to a lower synchronization, but still governed by the same laws of nervous excitability. The difference between normal and pathological thus results from purely quantitative variations. The dynamic conception of the brain presented here aims to unify and synthesize aspects belonging to disciplines such as brain pathology, sensory physiology, experimental psychology, etc., on the common basis of nervous physiology.

An attempt has been made here to establish brain activity in man on a physiological basis, thus filling the important gap hitherto existing between brain pathology and the physiology of the nervous system.

* * *

The development of the research presented in these two volumes has been carried out without interruption over more than ten years. A simplified chronology of some of the findings is as follows:

In 1938, inverted vision was observed in patient M and clinical data was obtained on him. The important dynamic action phenomena (excitability, asynchrony,

facilitation and repercussion) were found in 1939. In 1941, an unpublished communication was presented to the Spanish National Research Council [Consejo Superior de Investigaciones Científicas],[2] where important experiments, the principle of *magnitude and position* of the lesion, and fundamental concepts of brain dynamics are exposed, all of which systematically developed in the present book. Progress was relatively rapid at the beguinning; subsequently, it has been slow and laborious, given the multifaceted nature of the topic. Experimental work from 1942 onwards took much longer than expected, both because of the extent and complexity of the issues, and because of the difficulty of obtaining the most indispensable experimental instruments. In 1942, the effect of the magnitude of the lesion was verified on patient T, and greater quantitative precision was obtained in various phenomena (excitability curves) in M and in T. This led to the characterization of the *central syndrome,* which presents a bilateral and symmetrical disorder. In 1943, the recruitment logarithmic curve was determined for visual image orientation, extending the Fechner-type law to sensory pathological phenomena. In 1944, the effect of binocularity was measured by means of visual image orientation curves, important for understanding summation effect by different facilitations. Curves for touch and hearing were determined, and some cases of *paracentral syndrome* (asymmetric disturbance) were studied. In 1945 the problems of tactile space were addressed, and in 1946 both tactile inversion and auditory inversion were discovered in patient M. Systematic research on spatial inversion led to the formulation in 1947 of a *spiral development* of the sensory field. In 1948 concepts such as residual field, sensory dimensions, functional growth, etc., were introduced.

* * *

The first volume presents some fundamental findings as well as general concepts of brain dynamics, followed by the systematic study of visual functions on the new basis.[3] A multitude of pathological phenomena, many of them new, as inverted or tilted vision, are analyzed for the first time on the basis of nervous excitability (excitability level, asynchrony, facilitation, summations, etc.), supported by quantitative determinations of intensity-duration excitability curves as well as those of sensory recruitment, which complement each other. The starting point is the dynamic action phenomena in nervous excitability, thus providing a simple and unified interpretation of our two cases, M and T, and the discussed Schneider case of Goldstein and Gelb (1918, 1919). These cases belong to the central syndrome characterized here, whereas for the usual theory they constitute limit cases that are im-

[2] As mentioned in a footnote in the Preface, Justo Gonzalo submitted in 1941 a 95-page report to the Spanish National Research Council [Consejo Superior de Investigaciones Científicas] entitled: *Investigaciones sobre Dinámica Cerebral. La acción dinámica en el sistema nervioso. Estructuras sensoriales por sincronización cerebral* [Investigations on Brain Dynamics. Dynamic action in the nervous system. Sensory structures by brain synchronization].

[3] At the beginning of the part devoted to *Sensory Dynamics* (first visual and then tactile), J. Gonzalo states that the research described is original, and whenever a precedent exists, it is duly indicated.

possible to resolve. On the other hand, the most typical syndromes of brain localization theory are considered in the brain dynamics here exposed as limiting cases (marginal syndromes) compared to the central syndrome. In them, rather than cortical lesion of gray mass, there is a lesion of projection pathways. The various syndromes that are usually described in more central cortical areas would instead belong to some extent to the more or less pure central syndrome. The effects of a cortical lesion depend on its magnitude and position. A sensory system is always altered as a whole when a cortical lesion is involved, but if the magnitude of the lesion is small, it may falsely seem, on an inaccurate examination, that only the most superior activity (gnostic type) is involved.

The second volume, on tactile functions, deepens on several general issues and, above all, initiates a new theoretical stage in the brain dynamics presented here by considering spatial inversion at the center of the sensory organization. The research, as in Vol. 1, is original (see footnote 3), the most relevant topics to the new conception being *tactile space* and perceived *tactile orientation,* with the findings on tactile localization phenomena and tactile space inversion. New dynamical concepts are stablished, such as sensory dimensions (intensity, space and time) dependent on the active brain mass (i.e., on the magnitude of the lesion), residual sensory field where the dimensions are reduced, functional individuality derived from organization, central action, functional growth by brain recruitment, etc. All this together with the generalization of inverted perception (in touch and hearing) has led to establish a principle of *spiral development* of the sensory field, susceptible to account for all kinds of pathological sensory phenomena. Inversion is related to the anatomical configuration, whereas size and re-inversion (organization) are related to brain mass, to functionality. The sensory organization presents a development as a whole from which the various functions arise by differentiation. Therefore, it is necessary to focus on the degree of differentiation (functional level) of the sensory system, which can be properly expressed by the dimensions of the sensory field. Between mere sensory function and gnostic activity there is a continuity over the same pattern of organization, and therefore gnosis (or schema function) is determined by the dimensions of the field.

Finally, we would agree with Priestley (1777), who already in his time stated that physics should be applied to the nervous system.

* * *

We are grateful to the Cajal Institute for funding this work since 1942.

Volume 1

General aspects

Fundamental findings and the conception of brain dynamics

In this part, we study the fundamental findings that constitute the starting point for the new conception of brain activity. These findings are the various phenomena of *dynamic action,* found at the end of 1939, which lead to an understanding of brain functions completely different from that sustained by classical theory.

Significant new data lead not only to a new general point of view but also to innovation in working and research methods. We must therefore deal with three outstanding aspects: dynamic action phenomena, a new conception of brain activity and research methods followed. These aspects are completely interdependent since they are based on the same principle, which is developed systematically.

This part on general aspects is necessary both to show the theoretical meaning of this research and to present basic phenomena which will constantly appear in a second part when studying in detail the different sensory functions.[1] In this way, it is possible to maintain a certain balance between a deductive and an inductive exposition that allows a better understanding of the present work.

This exposition on general aspects and their theoretical meaning will find its completion in a third part.[2]

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[1] Such second part deals with visual functions in Volume 1 and with tactile functions in Volume 2.

[2] A third volume was not published in spite of the material prepared for it. In addition to Vol. 2 (first published in 1950), there are these two later publications by the author:

GONZALO, J. (1951, 1952). *Trabajos del Instituto Cajal de Investigaciones Biológicas*, XLIII: 209-260 (1951). XLIV: 95-157 (1952). (The latter is Supplement I).

A very brief exposition of the last part of the research is included in Supplement II. There are works by other authors on this part in a more recent context, as mentioned in a footnote of the Preface, for example:

GONZALO-FONRODONA, I. (2009). "Functional gradients through the cortex, multisensory integration and scaling laws in brain dynamics," *Neurocomputing,* **72**: 831-838.

OA: https://doi.org/10.1016/j.neucom.2008.04.055

GONZALO-FONRODONA, I. and PORRAS, M. A. (2014). In: *Horizons in Neuroscience Research,* Vol. 13, Chap.10: 161-189, and references therein. OA: https://doi.org/10.48550/arXiv.2006.01666 and https://novapublishers.com/wp-content/uploads/2019/06/978-1-62948-426-6_ch10.pdf

1. Fundamental findings

1.1. DEVELOPMENT OF THE RESEARCH

The elaboration of the topics we are going to expose has taken a long time; the studies began in the summer of 1938 and continued without interruption up to now, that is, six years. The research was initially oriented towards the complex problem of agnosia syndrome. However, based on new agnosia phenomena, the initial orientation was changing as the mechanism of these phenomena was investigated in depth. A broader base has been established by achieving a transition from brain pathology to brain physiology. The latter has been developed in an entirely systematic way, since by starting from a small number of fundamental facts it has been possible to carry out a development that encompasses all types of brain activity. Thus, a human brain physiology is established which is directly based on the laws of nervous excitability, and due to the special dynamic conditions that determine such excitability, it can be called *Brain Dynamics.*

New syndromes have constituted the first starting point. Thus, in a certain subject affected by a left parieto-occipital brain injury, who initially seemed to present only a concentric reduction of the visual field, a series of singular disorders were found in the summer of 1938, such as inverted vision, loss of visual perception of motion, *flat color* vision (Gelb 1920), as well as other more elementary alterations such as greenish *chromatopsia,* etc. The first remarkable phenomenon found in that subject at that time was the perception of flat colors; the last one was inverted vision, a couple of months later. This proves how hidden or ignored these kinds of disorders can remain for the reasons we shall indicate further on. The study of these phenomena, providing new findings, gradually became more problematic. Thus, a finding related to the inverted vision was the remarkable phenomenon of loss of *orthogonal* function in visual shapes.[1] Due to this latter phenomenon, the subject was able to

[1] Orthogonal function means correct image orientation on the plane orthogonal to the line of sight.

read letters and numbers both in their normal position and upside down, and claiming that the position in both cases is completely identical and normal. Along with these unusual or totally unknown phenomena there were the usual visual agnosia disorder in a very diffuse and unstable way. This was a motley complex of bizarre phenomena which, despite giving rise to very significant disturbances, went completely unnoticed at first glance, since they did not seem to alter the ordinary behavior of the affected individual, who was almost the most unaware of his disorders. However, it was soon discovered that agnosia and other sensory changes were not limited to visual functions; touch and praxic functions were also affected, therefore a very special disturbance had to be dealt with.

There were precedents for some of these agnosia phenomena, especially concerning the flat color disorder, first described by Gelb (1920) in two patients with occipital damage occurred in World War I, although it has not been studied in detail since then. Some cases of inverted vision had also been observed, already at the beginning of the 20th century, but these were simple notes about this disorder suffered sporadically by certain patients with a brain pathology, without being directly observed and objectively examined by the authors.

Initially, no matter how interesting the question of the cerebral localization of these disorders was according to the usual conception of brain pathology, much more attention was paid to the study of pathogenesis, since very favorable conditions were offered for this. Thus, in the case of inverted vision, it was discovered that an object appearing in a normal position in near vision, was seen gradually tilted when moving away, to the point of getting totally inverted at a certain distance. It was also found that the bigger the size of the object, the greater the distance needed to reach the inversion. These tests, among many others, made the research focus on the pathophysiological study. This study made possible, after many detours and patient observations, the discovery of nervous phenomena of greater scope and scientific meaning, which gave a new course to the research.

During the summer of 1938, a large amount of different observations on the brain-injured patient in question had been gathered, although their systematization and, above all, their theoretical explanation presented great difficulties. In 1939, attention was paid to the relationships of brain excitability, which were so evident in various experiments about inverted vision. Thus, a strong stimulation, i.e., near vision, resulted in a correct and normal image, whereas a weak stimulation, i.e., far vision, resulted in an inverted image, according to the especial physiological conditions of the patient – what we shall later call functional asynchrony –. Although a decisive step had been taken, there were certain difficulties that needed to be solved before a proper and conclusive explanation could be reached. An important issue to be addressed was the great instability of the phenomena in a short period of time and even within the same examination. A large margin of variation made every meticulous determination difficult and any quantitative assessment useless.

The persevering and in-depth analysis of the aforementioned brain-injured patient, undertaken at the end of 1939, had as a most important result the unexpected discovery of the nervous phenomena of *dynamic action,* leading to a profound transformation of all views prior to this finding. The physiological criterion of excit-

ability became indispensable, acquiring a fundamental significance since, as it will be seen, the dynamic action phenomena are based on special changes in brain excitability. Consequently, the research was set up on a strictly physiological basis, addressing the study of brain activity in a very different way from that followed by brain pathology from the time of Wernicke (1874) to the present day.

Having shown that a single small left parieto-occipital lesion had caused a generalized and uniform alteration of the whole brain, i.e., of all the sensory systems, in both halves of the body, and that the different disorders responded to special dynamic conditions of nervous excitability (cerebral repercussion, asynchrony, summation, etc.), it was no longer meaningful to search for brain localizations that were against all logic. This is the negative aspect of the problem, and as for the positive aspect, it was necessary to take advantage of the great prospects offered by the dynamic manifestations of nervous excitability to unravel the fine nervous mechanism of brain functions.

Finally, in 1940, a systematic research plan was established in view of the new facts, which made it possible to successfully address several disciplines which, although close to each other, had always been quite isolated from each other, such as sensory physiology, physiological psychology and brain pathology. All of them should be derived from the physiology of the nervous system and, above all, from the laws of nervous excitability.

In 1941, it was finally possible to establish a conceptual framework that gave rise to the *Brain Dynamics* presented here.[2] However, due to insufficient working resources, a huge task remained to be done in the experimental domain. This task has been performed during the last three years (1942, 1943, 1944), in which, by means of limited instrumental resources, it has been possible to develop the experimental analysis and make a quantitative assessment of the phenomena. Thus, where before there were only simple descriptions from the clinical observation of patients, now a varied experimentation is carried out, whose results, expressed by means of graphic representations, show in the simplest and most accurate way the physiological laws that govern brain activities.

1.2. SINGULAR PRECEDENTS ON AGNOSIA RESEARCH

Before setting out the fundamental finding of the dynamic action, it is necessary to discuss certain precedents that will help to understand the meaning of the findings. Thus, we must refer to the works of Goldstein and Gelb (1918, 1919) on the well-known case of *Schneider*, a patient with brain injury from the First World War. These authors published two important research works on agnosia in this patient. These

[2] As mentioned in a footnote in the Preface and in the Introduction, Justo Gonzalo submitted in 1941 a report to the Consejo Superior de Investigaciones Científicas [Spanish National Research Council] entitled: *Investigaciones sobre Dinámica Cerebral. La dinámica en el sistema nervioso. Estructuras sensoriales por sincronización cerebral* [Investigations on Brain Dynamics. Dynamic action in the nervous system. Sensory structures by brain synchronization].

works have been significant both for the singular observations and for the theoretical consequences that the authors claim. The first one deals with visual function: *Zur Psychologie des optischen Wahrnehmungs- und Erkennungsvorganges* (Goldstein and Gelb 1918), a very detailed and extensive study, also highly cited and discussed; the second one addresses tactile functions: *Über den Einfluss des vollständigen Verlustes des optischen Vorstellungsvermögens auf das taktile Erkennen* (Goldstein and Gelb 1919).

With regard to the first of the works, on visual function, the patient with a left parieto-occipital lesion showed an *apperceptive visual agnosia* (blindness to visual forms), a type of visual agnosia supposed by Lissauer (1890) and demonstrated for the first time by Goldstein and Gelb (1918, 1919). The greatest novelty of this work is that the patient, being totally blind to visual forms, should not see more than amorphous spots, but thanks to a special mechanism discovered by these authors in this patient, he was able to behave apparently in a quite normal way with regard to his visual functions.

These authors found, after many persevering and meticulous observations, that the patient needed to move his head to see shapes or recognize them visually. He needed to move his head contouring the object to realize its configuration, otherwise he only perceived a chaos of shapeless spots. But the most surprising thing is the authors' explanation that visual recognition is fictitious because the patient is only able to recognize the object by a motor process and not by sight. That is to say, if the subject is able to find out the configuration or form of objects is not due to his sight but to the representation of the set of movements he makes with his head by contouring the diffuse forms of the objects. Such movements are so insignificant that they go unnoticed by the most attentive observer, as happened for a long time to these authors and also to the patient, who was initially completely unaware of them because he performed them involuntarily.

The mentioned authors consider that their Schneider case shows a kind of motor imagination and a surprising disposition for that kind of perception. If all spontaneous head movement was prevented, the patient in question was totally unable to see or visually recognize any object.

Such characteristics represent the most outstanding feature in visual perception and shape recognition by a person who is blind to visual shapes. In addition, various psychological processes related to the visual disorder in question are discussed by these authors, and all kinds of evidences are provided in order to maintain the above explanations.

In the second work of these authors, also very extensive and detailed, published shortly after the first one, they maintain that the loss of visual spatial sense and visual representation entails secondarily a disorder of the same nature in the sense of touch. In this way they aim to explain the disturbances of tactile space in both sides of the body (location of the stimuli, spatial discrimination of two stimuli, posture, etc.) as well as those of tactile recognition, since the patient presented a very special tactile asymbolia. They think that tactile functions are basically intact, and if they can be so severely affected it is due to the loss of the visual influence that would normally be exerted on touch, since touch alone would be unable to organize spatial and gnostic structures. Appealing to this hypothesis of visual influence on touch, they renew

a theory that was in vogue long time ago among psychologists and philosophers, in which it is claimed that there is no space other than that provided by sight, and that tactile space is generated secondarily by the influence or support provided by sight.

All these considerations refer to the general thesis. As for the particular disturbances of tactile function, there is again a disorder very similar to that of visual function, with very similar motor help phenomena. The authors find that thanks to certain muscular jerks or twitching movements that the subject makes unconsciously, it is possible to supply the functional deficit and achieve the localization of stimuli or even to perform other more complex functions. In short, a series of mechanisms allow the patient to reach a behavior of rather normal appearance, without which the patient presents important disorders in all the spatial and gnostic structure. According to the authors, such motor mechanisms only seemingly accomplish the spatial tactile function, and the indirect way of achieving this function is a subterfuge that in no way changes the true state of the patient, who lacks spatial sense in both sight and touch.

Such works, as much cited as discussed, have been the seed of a number of more or less theoretical conceptions. Thus, Schilder (1923/1935), for example, relies on these works on the Schneider case for his conception on *body schema,* and likewise Stein (1928, 1930) for his motor theory of perception.

On the other hand, several contradictions were soon indicated, and there are very important works devoted to refute the whole building of hypotheses of the authors, especially the work of Poppelreuter (1923), although in fact nothing positive has been established. Here, in the reduced space that we have to deal with many issues, we cannot expose in detail the research of Goldstein and Gelb (1918, 1919) and the criticisms it has promoted; thus, we shall expose only some general ideas. Aside from the important criticism by Poppelreuter (1923), the Schneider case has been discussed in various aspects by authors such as Pötzl (1928), Kleist (1922), Stein (1928), etc., as well as by many other scholars who have exposed it in detail.

Certain contradictions are indicated which make the Schneider case difficult to understand and, in general, it is thought that alongside remarkable and interesting observations, there are extremely hypothetical ideas.

The Schneider case was subsequently the subject of further research from a psychological point of view about general intelligence (Benary 1922) and language peculiarities (Hochheimer, 1932); and after twenty years his brain injury, Schneider was still the subject of conferences and various studies, maintaining the characteristics initially described by Goldstein and Gelb (1918, 1919). It can be stated that no other brain patient has so far given rise to as many works and comments as the Schneider case.

After twenty years, in 1939, the veracity of certain findings in the Schneider case of Goldstein and Gelb was confirmed for the first time in other cases, thanks to the evidence provided by the M and T cases, whose detailed study has provided the material for the present work. It should be noted that, although the great thoroughness of the mentioned authors has revealed very special phenomenological aspects of the Schneider case, they have not taken the decisive and indispensable step for a complete understanding of the disorders. It can be said that they have just arrived

to the borderline. As is often the case, a certain number of well observed facts are confirmed and maintained in subsequent investigations by other authors, whereas other facts of decisive nature remain latent and go unnoticed due to the influence of theoretical prejudices always present in any investigation. Thus, speculative constructions turn out to be alien to reality and further impede the discovery of significant facts. But all this is inherent to the scientific development whose progress is the result of the sum of several authors' efforts.

From the viewpoint of clinical and symptomatic manifestations, the Schneider case is the only precedent we can indicate to our current investigations on the M and T cases. This is not because such cases are exceptional; on the contrary, we think they manifest a modality of a very general nature, which, if it has remained hidden is due to an insufficient examination of the patients with brain injuries. However, the Schneider case is more a starting point in our research than a true precedent, since all the work presented here starts precisely from the experimental rebuttal of the theoretical explanations of Goldstein and Gelb (1918, 1919) about motor recognition and the alleged visual influence on touch. This is due to the finding of the phenomena of dynamic action, which in addition to offering a solution to the problems posed by the Schneider case, they open up new and wide-ranging perspectives in brain research.

1.3. THE FINDING OF THE DYNAMIC ACTION PHENOMENA

At the beginning of this research, in August 1938, the M case began to be significant, initially for the discovery of the phenomenon of *flat colors,* and especially later for the series of experiments on the curious disorder of inverted vision. But all this did not seem to indicate a possible similarity between the M case and the Schneider case, since neither of these two phenomena had been described in the Schneider patient. It was a year later, at the end of 1939, when trying to rationally explain the series of syndromes found in our patient M, an attempt was made to find a certain parallelism between the disorders of both subjects. In fact, although the M case had initially attracted all our attention to certain very remarkable symptoms never mentioned in the Schneider case, they had a good number of disorders in common, some of them extremely rare such as the loss of visual perception of motion and, in a more general sense, both presented, in very special conditions, the disorders of visual and tactile agnosia. The fruitful approach to the Schneider case was a consequence of a very careful examination of patient M to resolve certain difficulties that made the results of his examination very changeable.

1.3.1. The singular functional disaggregation

It often happens that some phenomena encountered at the beginning of an investigation remain almost unnoticed in terms of their main significance, and only stand out in a preferential place at the end of many detours, without anything essential having occurred in them. It all depends on the right choice between what is essen-

tial and what is accessory. This is what happened to us with the issue of functional disaggregation, which is the first phenomenon found of dynamic action. It will be dealt with in detail when studying *nervous asynchrony*. Only by combining it with the other phenomena, it is possible to understand the true situation of brain activity in patient M.

In the summer of 1938, by studying experimentally the disorder of inverted vision in patient M, we had enough data to establish, as a general rule, the remarkable functional disaggregation caused by the brain excitability disorder. The sensory response obtained depended entirely on the intensity of the stimulus used. Thus, if an object was seen at a short distance, its initially correct perceived position was progressively tilted as the distance to the object increased, until a total inversion occurred. The larger the size of the object, the greater the distance required to achieve the inversion. Likewise, even if the object was located at a short distance where it was seen correctly, the object could be seen inverted or strongly tilted if the exposure time of the object was only about one second. Therefore, it was possible to study with great ease the transition from the normal function (correct vision) to the pathological one (inverted or diversely tilted vision) just by changing the experimental conditions. This phenomenon was evidently based on especial alterations in brain excitability, with a different response to stimuli according to their intensity. The stimulation was weak in far vision due to low luminous intensity and small visual angle; the opposite for near vision. In the case of inversion or strong tilt in near vision but with a brief exposure of the test object, another stimulation factor took part besides the intensity: the exposure time of the stimulus. This fact was already an indication that the reaction time was extremely increased in this patient. For all this, the state of brain excitability gave the real explanation for all these disorders. However, the full importance of the excitability process did not become evident until a year later when the necessary maturity was reached by observing other dynamic action phenomena.

The functional disaggregation is the phenomenon that has allowed the important study of the physiological structure of the various sensory organizations. This disaggregation is based on an asynchrony of the nervous elements as a consequence of the special dynamic alteration in brain excitability. The characteristics mentioned above in inverted vision were common to all types of functions; thus, for example, in the case of tactile sensitivity, a weak stimulus was only perceived without producing any sensation of localization, whereas a more intense one was perceived in a localized manner. The results were dependent on the intensity and duration of the stimulus applied; hence, a tactile stimulus of a certain intensity but short duration was felt only as a simple contact, whereas a more prolonged excitation of the same stimulus caused localization.

These partial effects, out of phase with each other, are instead a single function (as an all-or-nothing effect) in a normal individual. In these partial effects it is clearly shown that the simplest elements of the function are the most easily excitable (have the highest excitability); thus, the more complex organizations are delayed because they need more excitation. These are only activated by stimulation of a certain intensity and of longer duration than those necessary to arouse the most elementary

activities. Thus, if the stimulus is weak, even if it lasts a long time, the level of excitation of the more complex elements (little excitable) is not reached and the only response obtained is that of the simpler elements of the disaggregated function.

No indication of all this is found in the detailed descriptions of the Schneider case by Goldstein and Gelb. They only emphasize that the behavior in the various visual and tactile activities is completely different depending on whether or not motor recognition intervenes in visual activities, and certain muscular jerks in tactile ones. The meaning of this difference will be seen later on.

This singular disaggregation was progressively observed in all kinds of altered functions, greatly facilitating how to address the research within the multiform set of symptoms that changed at every step due to circumstances that went easily unnoticed. It was precisely by trying to obtain the highest possible accuracy in quantitative determinations, that we reached the true understanding of the special state of brain excitability in subject M, in whom the functional asynchrony indicated above was only a particular aspect.

1.3.2. Facilitation (reinforcement[3]) or summation phenomenon

The great instability of the symptomatology in neurological patients is a well-known fact, but in our case, the lack of constancy of the phenomena when we tried to determine quantitatively with precision the effect of the stimuli was so sudden and of such a wide range that it could not be explained by fatigue or changes in the recovery from the pathologic syndrome, as could be admitted in other cases. Perhaps special mechanisms were involved, and for a long time they were searched for. At the same time, since certain analogies had been found between the general symptomatology of subject M and that of subject Schneider, even the special muscular jerks or twitching movements that appeared spontaneously in some examinations of the tactile function, we tried first to determine whether the motor recognition phenomena for sight existed in subject M, and then to see whether such phenomena were the cause of such remarkable variations in the behavior of the subject. This type of muscular jerks had been clearly observed in the second patient studied here, subject T, who was examined before subject M, and before knowing in detail the studies of Goldstein and Gelb (1918, 1919).

Whereas for the sense of touch (tactile localization for example, etc.), muscular jerks or twitching movements were evident on occasions and in an irregular manner, for the sense of sight they could not be verified in very repeated and detailed observations, and nothing seemed to confirm the existence of a motor recognition and a full analogy with the Schneider case. All these new analyses were undertaken by the end of 1939 in order to clarify and complete a number of details. It happened by chance that the patient M, lying down and in complete inactivity, he made so many errors when objects were presented to him for their visual recognition that it was possible to conclude that he barely had vision, which appeared to improve noticeably

[3] Reinforcement is 'refuerzo' in Spanish, term used in the original work, and has been translated in most cases as 'facilitation.'

as soon as he sat up and returned to his ordinary attitude. Such an unexpected change made attention focus again on the determination of motor phenomena or something similar. When trying earlier to examine the existence of motor recognition, it was not possible to find an active movement of the subject's head, even though it was known, from the indications of Goldstein and Gelb (1918, 1919), that such recognition movements went easily unnoticed due to their small amplitude. When the patient was told not to move his head at all, no significant influence on his functions was observed, or at any case, its effect was not very important, but a new test with the head kept fixed did give decisive results. With the patient's head firmly fixed, his visual perception of objects worked as badly as in the previous position, lying down and inactive. However, when the head was released, the usual behavior appeared again, that is, very slow but quite viable perception. Our patient became very surprised but did not seem to give much importance to such results, and only when he failed the visual tests with his head fixed, he insisted on having his head released, and said that it was because the blood did not reach his head and he was not able to see well. If such movements of his head were there before it was fixed, they had to be extremely slight, rather small muscular tensions producing insignificant displacements.

For a moment it was thought that the motor recognition of Goldstein and Gelb (918, 1919) had been found, thus confirming for the first time that phenomenon in another subject. However, in a multitude of further tests performed to study the issue accurately, very remarkable divergences began to appear, since our patient behaved as follows: he was able to recognize objects visually by just moving his head in any direction without corresponding in any way to the shape of the object presented. Therefore, such behavior should not correspond to the contouring referred to by the mentioned authors. In addition, when the subject's head was immobilized, and he was lying down in complete muscular relaxation and immobility, if objects were presented to him, his visual perception was initially unclear, but after ten to fifteen seconds the perception had improved considerably. In his ordinary state of total freedom and sitting up, this time was reduced to about half, although he was still very slow compared to a normal subject.

All these results led to the opinion that there was something that influenced the brain activity of visual perception, modifying nervous excitability. Other tests showed in the same way that the motor recognition according to Goldstein and Gelb (1918, 1919) was here inadmissible; in fact, keeping the subject's head immobile and asking him to clench his fist strongly, his visual perception worked as well as with the free movements of the head. No movement of any kind was required; tension or a strong muscular contraction of any part of the body was enough.

In the Schneider case, visual perception was rather slow, as in our patient in his ordinary state, and the authors thought that this delay was due to the time spent on performing the contouring movements, without which the subject was not able to see but shapeless spots. Therefore, in tachistoscopic exposure of figures, he was not able to see defined shapes because of the brevity of the exposure. But having ourselves arrived at the above results on changes in excitability due to a possible extra-visual facilitation, we tried to determine the effect of an intensification of this

type of facilitation on latency time. The result was that a strong contraction of all the voluntary body musculature reduced sensory time to such an extent that it seemed to completely normalize visual function. Patient M thought he was cured, he stated that he felt as before suffering the brain wound, although later, when he experienced that on finishing the intense muscular effort he returned to his usual precarious state, his enthusiasm diminished, but he kept on thinking that healing would be a matter of a few days. As expected, the patient was impressed by all these tests. In fact, when in previous experiments the effect of keeping still the head was found, and he had at last to discard the unfounded explanation of circulatory difficulty, he fell into a state of prostration as he realized the severity of his disorders.

By studying the possible existence of the motor recognition of Goldstein and Gelb (1918, 1919), we ended up discovering that sensory activity is considerably influenced, in its own excitability, by nervous actions foreign to such activity, as is the case of muscular tension. This type of facilitation, which represents a nervous summation phenomenon, can show gradations. Thus, it happens that already the upright attitude entails a natural muscular tension causing a certain summative action to which can be added the action due to slight tensions of the neck muscles (performed unconsciously during visual perception), reinforcing the previous summative effect. When the subject is in complete inactivity (lying down and in a state of muscular relaxation), all facilitating action ceases and the true basal state of brain excitability is achieved. Finally, by means of maximum contraction of the whole musculature, the effect of summation is so considerable that the visual behavior of the subject becomes quite normal. Although in the latter case, we shall see that by examining the subject thoroughly, he still shows a certain deficit, though very small in comparison with the two previous situations: complete inactivity, and moderate muscular effort in ordinary life.

In summary, from what is exposed in this section, it can be inferred that for the proper understanding of the pathological manifestations of our subject, it is necessary to take into account, besides the intensity and duration of the applied stimulus, the summation effect by muscular action, which increases brain excitability. With this last finding of facilitation by muscular effort, the problem of instability and inconstancy of the experimental results, which had made so difficult all attempts at quantitative determination of disorders, was solved. It was then possible not only to obtain precise and stable results, but also to obtain a large number of them, since a new procedure was available to easily experiment on nervous excitation relationships.

However, the investigation on summation did not stop here since, given that the effect of muscular contraction seemed to be so non-specific, there was reason to believe that perhaps any other type of nervous action would produce the same result. Indeed, the result of the tests performed was entirely in favor of this hypothesis. Both auditory and tactile stimulation were able to improve visual function, however the effect obtained was much smaller than that produced by an intense contraction of the whole musculature, as is easily understandable. Certain phenomena were to be chosen to clearly show the summation effect. To this end, the inverted vision disorder is very appropriate. Thus, an object placed at a convenient distance is seen inverted (upside down) if the subject is free from any facilitating action, but by means

of an intense contraction of all his musculature, the image of the object rotates quickly becoming correctly oriented and at the same time the sharpness of its shape improves considerably. Under these same conditions of object presentation, and being free of any facilitation, a tactile excitation of a certain importance, such as brushing or rubbing his back, without causing any pain to avoid defensive contractures, has the effect of re-inverting the image of the object in a certain number of degrees, i.e., to change from the inverted position to a tilted or even horizontal one. This change lasts only as long as the tactile stimulation is applied. Similarly, a strong auditory excitation by means of an intense whistling at the subject's ear causes the inverted image to oscillate by causing a small re-inversion.

This means that any type of nervous action, both muscular contraction (in a constant tension or performing movements) and any sensory excitation, modifies the state of brain activity as a whole when it reaches the cortical centers, reinforcing by a summation action the excitability of the brain. There is no specificity in the effects of the various types of facilitation mechanisms. It should be note that there are also significant types of summation phenomena in certain situations, and their knowledge is of great importance for the full understanding of the behavior of the brain-injured people we shall study. Thus, especially in vision but also in hearing, the result of the tests is very different depending on whether the person uses only one eye or one ear keeping the other one shut, or whether he uses both eyes or both ears. In the latter case, a significant improvement of the corresponding function occurs due to the resulting summation effect. This effect will be called *bi effect,* and is of great importance in vision and in any other sensory system as well. For example, in the sense of touch, a weak contact can be made perceptible by means of a simultaneous stimulation in another region of the skin. In this way it is also possible to reduce, or even suppress, the pathological time lag between the sensation of mere contact and that of localization.

Several types of facilitation can act simultaneously and thus produce a very important summation effect; for instance, binocular vision and maximum muscular contraction. Therefore, different types of facilitation must be considered in nervous facilitation; otherwise, important gaps would remain in both the accuracy of experimentation and the theory of nervous summation.

So far, we have referred to the visual system. As regards tactile functions, the results were entirely similar with respect to the conditions of functional asynchrony depending on the intensity and duration of the stimulus applied (as mentioned above), as well as with respect to the facilitation effect. Therefore, the twitching movements referred to by Goldstein and Gelb (1918, 1919) were not indispensable to localize stimuli and reach other more complex functions. Complex functions such as stimulus localization were achieved both by increasing stimulus intensity without facilitation and by a weak stimulus with facilitation via a strong muscular contraction.

At the same time, all these relationships and excitability phenomena showed that the sensory disturbance involving vision and touch was global, from the simplest elementary sensation to the most complex perceptual and gnostic functions. By contrast, in the Schneider case, disturbances had been indicated only in the

latter two. The rebuttal of the explanations of motor recognition for vision and muscular twitching for touch, as well as the study of excitability relationships, and the fact that the sensory disturbance was extended to all kinds of functions of these two sensory systems, led to questioning the hypothesis of the supposed visual influence on touch.

We now proceed to complete the study of the dynamic action.

1.3.3. Cerebral repercussion

This is about the finding of the disturbance of all brain functions and on both sides of the body, produced by a single not very extensive lesion in the left parieto-occipital region, against all that could be expected from the theory of brain localization.

Excitability relations and central summation phenomena, although generally ignored in brain pathology, are on the contrary well-known in the general physiology of the nervous system. Thus, it is enough here to extend them for the first time to the domain of human brain functioning. As for the nervous repercussion, which consists of the general brain alteration due to a small lesion in an area of *association*, it presents perhaps a much greater theoretical interest. This is because nervous repercussion phenomena (effects at a distance from the lesions), although they had been discovered some time ago, the research on them had not involved the human brain. But now, in our case, these phenomena have raised questions of fundamental importance, radically modifying current trends and, above all, compromising the theory of brain localization.

The difficulty in understanding a tactile disorder of equal severity on both sides of the body caused by a lesion in an area that has nothing to do with the brain region attributed to touch, may have led in the Schneider case to the hypothesis of visual influence mentioned above. But now, having shown that the disorder in our M case was much deeper since not only spatial functions (localization, two-point discrimination, motion, position, posture, etc.) were involved, but also the simplest sensitive manifestations (pressure, pain, temperature, etc.), the thesis of a secondary disorder of tactile space did not answer the whole question. In order to explain the tactile disturbance, the aforementioned visual influence should then have been extended to more elementary functions; but, to think that the simple sensation of contact or pressure needs visual influence is meaningless. Therefore, the question that tactile function was affected in both halves of the body following a brain injury on the left side and outside the tactile area was still a problem. Nevertheless, the same problem arose with regard to visual function, since the brain damage did not involve the calcarine cortex, but the patient suffered from a very large concentric reduction of the visual field, that is, the two halves of the field were involved. This patient as well as a large number of our wounded subjects with concentric visual field reduction of different severity, presented brain lesions outside the calcarine cortex or visual field projection area, and so did the Schneider case as well. This disorder, without any possible explanation within the theory of brain localization, despite being sufficiently known, has not led to the necessary research. The difficulties to explain this type of disorder in both visual and tactile systems were the same, and it was not possible

to progress by trying to understand a disorder by means of another disorder, since both disorders were equally in need of further research.

On the other hand, the finding of facilitation by muscular effort, and the evidence that it exerted a dynamic action "at a distance" on brain excitability, opened the way to consider these disorders from a dynamic point of view. If facilitation acts "at a distance," the lesion could also do so; in this way nervous activity should offer a much more unitary character than what might be assumed within the generally accepted anatomical and localization concepts of brain pathology.

If such a hypothesis of action "at a distance" of the lesion (dynamic repercussion) was correct, it seemed conceivable that other hitherto unexplored sensory systems might have been equally impaired, and due to the special characteristics of the disorder they would have gone unnoticed. Based on this, we decided to determine the patient's hearing status in all its aspects, and found that he showed a kind of alteration completely identical in intensity and characteristics to that shown in vision and touch. The summation effect due to the upright attitude and other weak muscular efforts, as well as the habitual slight examination had made all these hearing disorders unnoticeable. The hearing impairment was in all respects in line with the characteristics already indicated for other sensory systems with regard to intensity-duration excitability relationships, sensory function delay (functional disaggregation), summation effect, etc. The disorder also affected both ears, which showed severe deafness when the subject was inactive, the acoustic stimulation was weak and, above all, of short duration. There was also inability to perceive sound rhythms from a metronome, to distinguish musical tones, etc.

Thus, our view that there was a general alteration of the brain by a dynamic repercussion effect was reinforced by the empirical confirmation of the theoretical deductions. The three most important sensory systems, vision, touch and hearing, were deeply altered on both sides of the body, that is, in their full extent, and also in all their functional aspects from the simplest elementary sensation to the more complex gnostic functions. The disorder showed the exceptional feature, within the usual concepts, of being the effect of an action "at a distance" of a brain lesion located in a central area (association area).This establishes a new type of brain disorder that we shall study later on under the name of *central syndrome* of the cerebral cortex, and which also constitutes a starting point for the fundamentals of brain dynamics.

Other sensory disturbances, although much less important, appeared in lower functions such as taste and smell, to which disturbances in orientation and body position corresponding to a function of the labyrinth had to be added. All these fundamental findings on dynamic action were well established, as mentioned above, at the end of 1939 in patient M, confirmed much later in the second patient (the T case), and expanded and refined in a number of details during the following years. The general perturbation of the sensory systems has made it possible to study in a single individual a multitude of phenomena, such as the complete series of sensory activities and their physiological characteristics revealed by a sensory lag (functional disaggregation). Furthermore, by means of the summation effect (facilitation), an important experimental resource has been provided to expand and control this type of analysis.

1.4. REBUTTAL AND EXPERIMENTAL EXTENSION OF THE TWO RESEARCH STUDIES OF GOLDSTEIN AND GELB ON AGNOSIA

The two previous sections have dealt, firstly, with the origins of these studies in the research on the Schneider case and, secondly, with the finding of new data on dynamic action that have profoundly changed the initial orientation of the research. Now is the time to specify conclusions and considerations on these matters.

Long after the finding of the dynamic action in patient M, the other injured patient (subject T) was sought since, due to the symptoms he presented in 1938, he had certain analogies with M. The purpose was to submit him to the same analysis to which the patient M had been submitted. The brain lesion was in the same region as in the M case, but it was less deep. According to these circumstances, the behavior of subject T was found to be entirely analogous to that of subject M, except that all the excitability disorders were much less pronounced. Thus, less slowness, less asynchrony (less sensory lag) and less summation effect. As for the general disorder of sensory systems (cerebral repercussion), the distribution was entirely identical to that of patient M.

In short, we are dealing with two cases with a dynamic disturbance of the brain, different in their intensity but identical in their essential characteristics. As for the Schneider patient, in view of his more accentuated manifestations, he can be considered to present the type of dynamic alteration described here. Moreover, in view of certain data, the Schneider case can be considered as intermediate between the T and M cases as regards the intensity of the brain disturbance. In this way, it is possible to know approximately the true state of alteration of sensory functions in the Schneider case on the basis of a small number of indications, and it is also possible to predict the existence of a number of disorders that have been completely unnoticed by the authors.

In their final comments, Goldstein and Gelb (1918, 1919) insist on the inalterability of the symptoms in their Schneider case; but after all that has been said here about the changes according to the conditions of excitation and facilitation, their statement can be considered to be the result of an incomplete observation. They also found that certain complex functions of a spatial nature (form, motion, localization, etc.), as well as the higher functions of visual and tactile recognition, failed or were missing in the face of stimuli of ordinary intensity. A more attentive examination of the conditions and relationships of excitability is enough to know that the disorder originates, above all, from a general alteration of the excitability of the brain, encompassing all kinds of sensory functions, both simple and complex.

For the same reasons that the authors think that the Schneider case is a man who lacks spatial perception for both sight and touch, they could have argued that he was blind or anaesthetic (without sensation), since stimuli of a certain intensity but of very short duration (easily detected by a normal individual) would have gone completely unnoticed by him, as in the M and T cases. However, increasing the intensity or the excitation time is enough for even complex functions to appear.

The special modification of nervous excitability leads to the generation of iterative excitation, i.e., a weak stimulus sufficiently repeated at short intervals can pro-

duce not only elementary sensory responses (which do not appear under a single excitation), but also sensory responses to complex functions. This iteration capability is very notorious in the M case and much less in the T case, in line with the intensity of the other nervous excitability disorders. Hence, the intermediate degree of iteration that the Schneider case should present can be found with a certain approximation.

The research on the Schneider case remains exclusively at a psychological level, while the mere description of the symptoms is at a clinical level. As interesting as the results are, the decisive step towards a physiological basis of the disorders (the only way to fully and correctly explain the important questions raised by the Schneider case) has not been taken.

The need for a physiological basis becomes even more peremptory with regard to phenomena such as the supposed motor recognition and the alleged secondary tactile alteration by abolition of the visual influence. These two explanations are completely psychological. In one, everything is reduced to a singular facility for motor representation. In the other, it is admitted that the only true space is that provided by sight, and that spatial structures of touch are an effect provided by sight. The authors went so far as to pose the problems, but the psychological orientation has perhaps blocked the way to a proper solution. A physiological orientation would have led to logical arguments capable of explaining all kinds of phenomena in a unitary and simple way. Therefore, we can say that our research rebut the results of Goldstein and Gelb (1918, 1919) on agnosia in the Schneider case, at the same time that it extends their research by continuing it under a more experimental aspect, even though the result spoils the explanation of these authors.

In particular, with regard to the motor aids that all these subjects use unconsciously, the physiological point of view becomes irreplaceable; the only explanation is that of the nervous summation effect. Moreover, it is necessary to take into account nervous summation in order to know the true state of the brain disorder, since the weak summation generated in the ordinary state of these injured patients is enough to improve their functions and mask the severity of their impairments. We know that depending on the intensity of the muscular effort, the intensity of the nervous excitability disorder can be very different, and the subject can then present different types of states. This makes it possible that different cases regarding the intensity of their nervous disorder can be equated to some extent. As an example, patient M under maximum facilitation by strong muscular contraction can be very similar to patient T free of all facilitation, and M with somewhat less muscular effort becomes similar to patient Schneider. The psychological conceptions of Goldstein and Gelb (1918, 1919) that have induced them to leave aside such important questions about nervous excitability, show how far brain pathology research is from its true physiological basis.

The other fundamental result of the second work on the Schneider case is quite paradoxical. It is remarkable that Goldstein (1910), particularly known for his justified criticisms of the classical theory of brain localization based on the anatomical-clinical point of view, did not take advantage of the exceptional opportunity he had with the Schneider case to provide an observational fact as simple as decisive.

That fact was much more eloquent than all kinds of theoretical arguments, always very diffuse in this matter. By resorting to the theory of inter-sensory influence, Goldstein (1910) has remained within the associationist psychology, one of the mainstays of the theory of brain localization. The development of this issue has led him to establish primary alterations for sight and secondary alterations for touch. However, in both our cases and in the Schneider case, vision, touch and hearing are basically altered at their origin, in all their functions, with the simplest functions being altered on both sides of the body, a fact that is totally incomprehensible according to the theory of brain localization. The solution to the problem of a general disturbance of sensory systems affecting the whole body, due to a left parieto-occipital brain lesion (which does not involve visual, tactile and auditory projection areas at all) is to radically abandon the usual concept of a rigid and static anatomical localization, since brain lesions affecting the truly central areas (the so-called association areas) disturb equally, by dynamic action, the entire brain system that is in functional relationship with those centers.

The cerebral cortex can be considered as a dynamic structure in which the functional level depends on the amount of nervous mass, mass that would act as an unspecific activator of brain excitability. From this point of view, it is easy to understand that the exclusion of nervous mass due to destructive injuries should reduce functional capacity, i.e., impair the excitability level of activities subordinated to the dynamic action of the nervous centers. The effect of facilitation is then to add activity to the remaining brain activity, thus partially replacing the brain mass excluded by the lesion.

In short, within the physiologic criterion, the brain disorder of the three cases, Schneider, M and T, can be explained by a unitary dynamic disorder. By virtue of this functional dynamic unit, the disorder of nervous excitability spreads to the entire brain, with regard to sensory functions. This disorder is characterized not only by a necessary increase in the stimulus intensity and duration, but also by *permeability* to iteration and facilitation (summation effect), and additionally by a functional asynchrony (leading to a functional disaggregation) arising from the excitability deficit, as we shall study further on.

1.5. SPECIAL FEATURES IN THE EXAMINATION OF THESE WOUNDED PATIENTS

At first glance, although it might seem paradoxical, these cases present a scarce symptomatology. Patient M spontaneously complains only of a great loss of vision, which otherwise does not bother him greatly in his daily life. Apart from tests and clinical determinations, nothing in his ordinary behavior would suggest such serious disorders as those shown by his different sensory systems. Even more scarce are the manifestations shown at first glance by the second subject (T), who in principle has not shown any particular brain disorder outside the usual general discomfort typical of brain-injured convalescents. This means that the finding of the numerous disorders that these subjects have is almost exclusively due to very patient examinations which have made clear, over the years, the condition of these subjects, without them explicitly cooperated to the knowledge of their disorders.

Thus, disorders such as flat color vision or the inverted vision phenomenon have been unexpected results in examinations aimed at other purposes, and when they were found, the patient initially did not seem to give great importance to any of them. With respect to the important phenomenon of inverted or strongly tilted vision, subject M merely commented: "these are things that sometimes appear in my sight." He also explained that sometimes, before we found his disorder, he had been surprised by his abnormalities, for instance, seeing some men working upside down on a scaffold. In general, the alterations go completely or almost unnoticed by the injured subjects themselves, and even when they are discovered, these subjects do not seem to be concerned, and rather consider them as something transitory that does not affect their daily life.

Throughout the study of these injured subjects, the great difference in behavior during the experimental tests and outside them has been evident. Such divergence has been completely clarified by the special phenomena of dynamic action in these individuals. In fact, when in their daily lives they pay attention only to the most intense stimuli, the disorder of sensory lag that produces a disaggregation of functions is practically excluded. For example, visual stimuli of a certain intensity produce vision that is correctly oriented or very little tilted. But above all, the facilitation action is decisive, since it considerably reduces the brain disorder, as has been said. If out of the tests, binocular vision is used as usual, this is already a very important facilitation due to the double action of the eyes. If a certain muscular activity is added when walking, etc., as well as a good illumination of natural light, a series of actions are obtained that, when added, produce a considerable reducing effect on the disorder. In the first tests after the fundamental findings, only a few kinds of behavior were identified. Later it became clear that various simultaneous combinations of different types of facilitation are possible, allowing for a rather normal behavior in everyday life.

As for the attitude that the subjects adopt in the face of such singular and numerous symptoms, it is not completely indifferent in the long run. On the contrary, the finding of facilitation by muscular effort has produced a very deep impression on them, both because of the importance of its effect and because it is inexplicable and strange for them. On the other hand, they have gradually become accustomed to all kinds of tests, although they have certainly found it unpleasant to be affected by so many different disorders. The degree of acceptance of their situation was very different depending on the type of disturbance. They have adapted fairly well to detailed tests for quantitative determinations of sensory disorders and so on, but they become extremely irritated when a more intellectual activity disorder is demonstrated. In this sense, failures in visual recognition of simple figures or drawings, or certain very subtle defects in understanding language, are very badly received because they realize the great extent of their brain disorder.

Another notable aspect of the research on these injured subjects is the type of interpretation and appreciation they make about their pathological manifestations. In general, brain patients tend to give all kinds of explanations about the symptoms or phenomena observed in them, and since they are usually not very well founded, great errors would be made by relying on their explanations and on the subjective

phenomenology of the patient without further control. It is necessary to submit the patient to an objective control by means of very varied tests, by which we can perfectly realize his functional state by the logical correspondence between the different tests. Moreover, it must be taken into account that within the instability or lability of his sensory abnormalities, a certain equalization may occur between very different phenomena. Thus, patient M claims that a piece of writing is in the same position whether it is presented in the correct position or upside down, due to the loss of orthogonal spatial orientation,[4] which is to some extent independent of the inverted vision disorder. In this case, to be more precise, the patient should have said that he does not know about positions in reading and does not distinguish the correct position from the inverted position of writing. In this behavior also intervene sensory organization laws generating the mentioned equalization. It is conceivable that the mechanisms involved are analogous to those that cause Babinski's anosognosia syndrome (Babinski 1914, 1918) in many brain lesions, according to which the patient tends to involuntarily exclude the existence of the defect. For example, the hemiplegic patient who ignores his paralysis, the receptive aphasic with logorrhea and jargon aphasia who is unaware of the lack of intelligibility of his language, or the brain-injured blind person who obstinately insists on his visual capacity, which is the so-called Anton syndrome (Anton 1898, 1889) of which we have study several very instructive cases in brain-injured patients.

What can really hinder the examination, and made it indeed difficult at the beginning of this study for insufficient knowledge of the condition of the injured patients, is the lack of vivacity, that is, the great slowness of all the brain processes due to the excitability deficit that gives rise to a long latency for stimuli as well as to an abnormal persistence of them. The latter can lead to fusions of stimuli from successive tests very close in time. This excitability deficit, which entails functional slowness, also causes mental clumsiness or viscosity, which obliges to carry out a paused and meticulous examination if useful results are to be obtained. It is very important to keep in mind that patients respond to tests in an approximate way, especially if the brain disorder is very pronounced. Therefore, quantitative determinations may be of little value unless patients are properly instructed and attention is paid to all details.

In addition, given the complexity and extent of the sensory disturbance, tests that may not be useful or lose the significance they would have in ordinary cases should be avoided. For example, when investigating color vision, it must be kept in mind that there is a very complex disorder involving very different aspects of color function. Thus, color weakness is accompanied by greenish chromatopsia as well as flat color vision (which can significantly alter the perception of colors located next to each other), and finally by color agnosia (difficulty to understand color names). Only by considering the disorder as a whole is it possible to establish the examination methods to be followed, and how much value should be given to the results.

[4] Orthogonal orientation means correct orientation on the plane orthogonal to the line of sight.

2. The new central syndrome of the brain

2.1. THE M CASE AND THE T CASE

Patient M and patient T were examined for the first time in 1938. Due to various circumstances, patient M has been studied more extensively and with whom this research work has been initiated. As already indicated, both show the same general characteristics, differing only in the quantitative degree of their disorders. This allows a more extensive investigation since it can be developed by considering different levels of excitability. The general characteristics of these cases will allow us to describe the new *central syndrome* of the cerebral cortex, which means a considerable transformation in the theories of the brain.

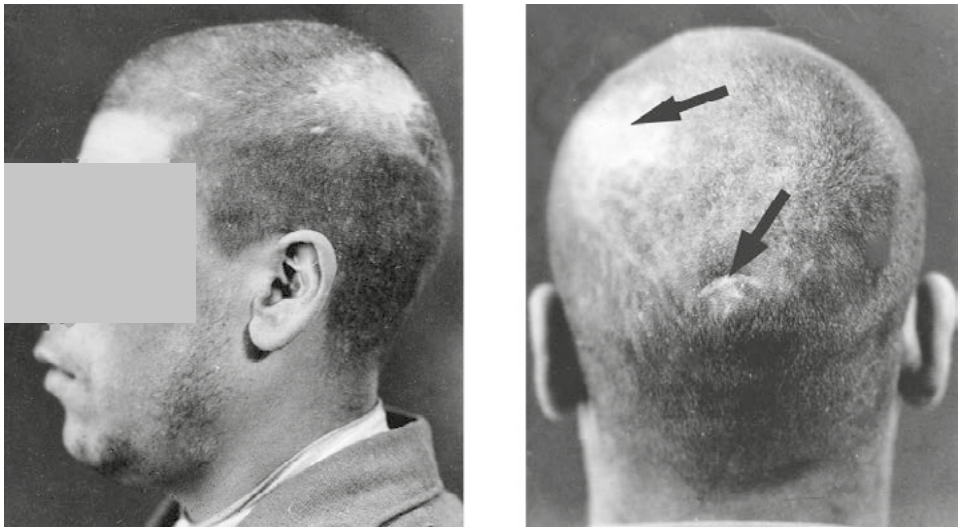
2.1.1. The M case

Subject M suffered a brain wound in May 1938, when he was 25 years old; he has two cranial scars in the left parieto-occipital convexity that corresponds to the entrance and exit orifices of a bullet. A very small entrance hole located 1 cm from the midline and above the external occipital protuberance. Another much bigger one corresponds to the exit, star-shaped and located three fingerbreadths from the middle line and two behind the mastoid line (Figs. 2.1 and 2.2). Despite the importance of these wounds, the subject healed easily and quickly without the need for surgery or other special care. He only had a little suppuration at the edges of the exit wound, which delayed full healing somewhat.

From the location of the entrance and exit cranial wounds, it can be stated that the brain area destroyed by the bullet corresponds approximately to the angular gyrus, close to the posterior end of the inter-parietal sulcus. Therefore, it corresponds to the parieto-occipital, medial and somewhat upward region, i.e., the area in the middle of area 19 of the Broadmann nomenclature and perhaps the anterior portion of area 18 and the most posterior portion of area 39, both areas around area 19.

The lesion is placed on a fully *association* brain region whose destruction, given the short intracranial trajectory of the bullet somewhat tangential to the brain, must not have been very deep although the tear of the cerebral cortex convolutions in that region must be admitted.

He was observed by us a few weeks after being wounded, at which time he presented the usual residual phenomena in brain injured people (severe headaches, slight meningism, etc.). It seems that during the first days after the injury, he completely lost consciousness; later when he regained it, he presented a great loss of vision, especially in the left eye with which he could hardly see.



Figures 2.1 and 2.2. Patient M. The arrows indicate the two cranial entry and exit scars of the bullet that crossed the left parieto-occipital convexity in its middle and upper portion. The direction of the bullet is from behind forward and from below upward.

Recovery was slow and after some months, he presented an intense concentric reduction of the visual field in both eyes: on the perimeter, with a white test of 0.5 cm in diameter, the visual field did not reach more than 4 to 6 degrees for the right eye and one or two degrees less for the left eye. In addition, he had a very slight paresis on the entire right side of his body that disappeared in a short time. Except for the great loss of vision, he did not complain about anything nor did he present any other significant neurological disorders in the ordinary examinations. His general condition was good and the only thing that called our attention was his great need for sleep and his indifference.

He was more carefully examined during the summer months, and several agnostic and apraxic disorders were found, as well as a remarkable slowness for all sensory and intellectual functions. The most notable findings in the visual system, apart from the marked concentric reduction and a very complex and diffuse agnosia,

were: *triplopia* (triple image perception of a single object in monocular vision), a phenomenon that we have also studied in other wounded, and which we shall address in a special publication; *flat color* vision (a chromatic alteration that makes colors appear swollen and as if they had been detached from their objects); a special chromatic blindness in which, in addition to mixing certain colors, everything was seen with a green tint, being impossible the correct vision of white that appeared as light green (*chromatopsia*); and finally the inverted vision, intensively investigated during the months of August and September 1938. This anomaly was related to very complex disorders of visual spatial orientation, loss of the *orthogonal* function, etc. As for the sense of touch, apraxic alterations and a certain tactile agnosia were found, as well as an altered perception of posture and movement, etc., affecting the entire body.

He was examined again a year later and at the end of 1939; the above-mentioned phenomena of dynamic action were found, which revealed a much deeper and more extensive brain alteration than that first presumed in 1938. Since then, the research on this patient has become more systematic and experimental, he being subjected to periodic studies as the research work progressed. With regard to the evolution of the disorders, it can be said that only during the first year after being injured, there has been a certain recovery progressively slower; later, the degree of the disturbances has become more stable, and these can now be considered invariable in their evolution.

2.1.2. The T case

The second patient, the T case, was injured in January 1938 at the age of 20, in the left parieto-occipital region, upper third. He was operated the same day he was injured because of the sinking of the cranial vault in that region. During the first month, it seems that he suffered from paresis of the entire right side of the body accompanied by loss or decreased sensitivity on the same side. Two months later, when he began to be observed by us, his general condition was very good and he did not present any significant neurological disorder, the paresis and sensitivity disorder having disappeared. Because of the copious suppuration of the head wound, he had to be operated two or three times to remove the infected bone edges until the wound was completely clean. In the surgical interventions, the dura mater was always intact and in good physiological condition. He never presented any complications due to the surgical interventions, and his good overall condition allowed him to get up and lead an almost normal life. He never had cerebral vomiting nor meningism, only some headaches and dizziness of the type of small absences. Five months after the injury, the suppuration of the bony edges ceased completely, with the dura always having a good pulsation, healing the wound definitively.

As for the state of the scar, there is now a large hollow of about 3 cm in diameter due to the loss of cranial vault; inside, the scar tissue is thickened over the dura mater, which shows no pulsation because of the thickness and hardness of the scar. The depression is located two fingerbreadths to the left of the midline. The anterior prolongations of the scar reach almost the bi-mastoid line and behind, the scar is four fingerbreadths above the cranial external occipital protuberance (Fig. 2.3).

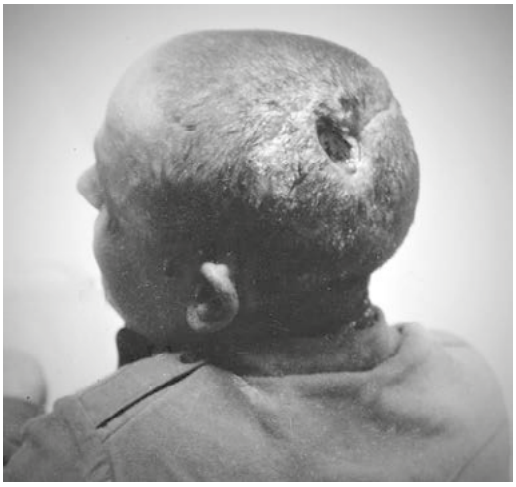


Figure 2.3. Patient T. Aspect of the head injury after healing. Left upper parieto-occipital bony depression that does not directly affect the brain matter.

This is a shrapnel wound that has fractured and destroyed a segment of the cranial vault in the left parieto-occipital area without tearing the dura mater nor causing anatomical subdural lesions, according to the numerous surgical operations in which the dura mater was always found intact and with a good pulsation. For all this, the brain disorders that the patient has presented, and the current residual effects, can be considered to be caused by the brain contusion produced by the bony sinking. This makes the brain disorder in patient T much less than in patient M; subject T has suffered a contusion whereas subject M has suffered a significant tear in the cortex due to the passage of the bullet. It must also be noted that the recovery from the brain disorder has been much more important in the T case than in the M case, which is well explained by the respective type of brain injury.

The location of the brain lesion in the T case is very similar to that of the previous case, and can be considered practically at the same place. It can be admitted that the brain lesion is located in the center of the intracranial trajectory of the bullet in the M case.

During the examination of patient T in the first months of observation, it was remarkable that such an important wound, even if it was only a contusion, caused so scarce neurological symptoms, even though it could be said that the wound was in an area of the so-called *silent* areas of the brain. He presented besides a concentric reduction of the visual field of moderate intensity in both eyes, a slight hypoesthesia in the right half of the body and a certain insecurity for the other tactile functions (localization, deep sensitivity, recognition, etc.). It is noteworthy in this subject that when he tried to localize tactile stimuli he slightly shook his body and limbs by means of almost imperceptible muscular contractions without which he made many mistakes.

A few months later, during the summer of 1938, he was subjected to a detailed study for a long time because of the important phenomena found in his visual field.

In addition to the concentric reduction and alterations of the type of the so-called *hemianopic weakness of attention* according to Poppelreuter (1917), he presented monocular *polyopia* phenomena in such a pronounced way that it served to unravel much of the mechanism of this phenomenon of multiple vision in a single eye, which we had already observed in many other brain-injured patients. Added to this, there were still very diffuse alterations of visual agnosia type, optic ataxia, etc.

Having been discharged when the reversed vision was fortuitously found in case M, and presenting both cases some clinical similarity, subject T was sought and re-examined for a few days. It was not found to present the phenomenon of inverted vision, but rather an important tilt of the image of objects when looking at them at a certain distance. He also presented the curious disorder of the loss of what we call orthogonal function (see Sec. 16), which allowed him to read just as easily whether the writing was in a normal position or upside down without noticing the change in position. Later we learned that before he was examined by us, after he had been wounded, he was often seen reading the newspaper with the newspaper upside down, and he was very upset when the anomaly was pointed out to him because it went completely unnoticed by him. Despite the absence of the phenomenon of fully inverted vision, although with a tendency to it (tilted vision), we found that after the epileptic seizures he suffered as a consequence of the cortical contusion, he was so confused by the visual orientation of objects that he preferred to remain in bed during this condition which lasted no more than 24 hours. It was also observed that his tactile and visual disorders were more pronounced than initially observed. For example, he had a significant postural disorder due to a deficit in the deep sensitivity, and a visual agnosia manifested by slow visual recognition and by the very special efforts he made to see and perceive objects, which could well be considered as the singular muscular jerks or twitches mentioned above in order to localize tactile stimuli. This last symptom was observed several months before examining patient M for the first time, and made us turn our attention to the Schneider case of Goldstein and Gelb (1918, 1919).

Due to the finding of the dynamic action in the M case at the end of 1939, and considering the similarity between the M and T cases, subject T was required to verify if he also presented the dynamic phenomena. But due to external circumstances, he could not be found until the year 1942 in which the phenomena of dynamic action were confirmed in the T case, in a similar way to the M case. These verifications allowed us to determine more precisely the extent and depth of the brain disorder. Thus, by studying in detail the excitability state, it was observed that tactile sensitivity was altered on both sides of the body, although slightly more on the right side. For the other sensory systems, the same alterations as those already know in the M case were found.

2.1.3. Comparison between the M, T and Schneider cases

From the finding of the dynamic action in both patients M and T we have been able to confirm more and more the similarity between them during the numerous periodical investigations they have been subjected to since then until now. If we compare

these two cases with each other and then with the Schneider case, we find that they all represent different degrees of the same type of brain disturbance.

Anatomically, the brain injury occupies approximately the same place in the patients M and T, although perhaps a little more upward in T. In the first case, considerably more destruction of brain matter can be admitted than in the T case, due to the tear produced by the intracranial trajectory of the bullet, although the damage has been limited to the most superficial zones. In the T case, there is less disturbance because the injury is a contusion type. As for the possibility of deeper or more distant anatomical lesions, there is no reason in the series of examinations of either case to indicate their existence. On the contrary, it can be established with great certainty that in both patients the brain injuries are confined to the site of the wound. In the Schneider case, he also presented a left parieto-occipital injury in its middle and upper portion with direct damage of the brain mass by the bullet.

As far as the recovery from the symptoms and residual signs, subject T has improved much more in relation to his initial condition than subject M. For example, the loss of orthogonal function (see Sec. 16) has disappeared since 1942-43, and polyopia is much less severe nowadays than it was in 1938. In any case, his disturbances have always been quantitatively much less pronounced than in the M case. The different degree of recovery, which can be explained by the different type of brain injury, more recoverable in the T case than in the M case, now further separates these two cases from each other in terms of the intensity of their brain disturbance. Residual phenomena, especially cortical epileptic seizures, have occurred very rarely in patient M, a total of a couple of seizures in more than five years. Instead, they are more frequent in patient T who suffers them every six months or more, due both to possible cortical irritations of the scar and to the type of irregular life he leads (occasional drinker, etc.). These seizures, at the moment, increase the intensity of the disorders, but it is a transitory state that lasts a very short time. In both cases, it can be stated that the degree of brain disturbance is completely stable and fixed since 1942, and measurements on sensory functions from one year to the next are usable. The same stability is presented by subject Schneider who after twenty years continues in the same situation as that described in the first two studies dedicated to him.

Concerning the symptomatic manifestations, we already know that the dynamic action, according to the characteristics exposed above, is fulfilled in our two cases, and gives the correct explanation to the Schneider case. But at the same time, the two patients studied here are quantitatively very different types of the same brain syndrome. Apart from the cerebral repercussion of the lesion, which causes a disorder common to all sensory systems and in both halves of the body, the excitability disorder is very different due to the different amount of brain mass excluded by the lesion, resulting in two types of physiological level. Thus, considering the visual field whose condition can serve as an index of the degree of disturbance of visual functions, subject M shows an intense reduction since the vision of the usual test of 0.5 cm reaches only up to 6 degrees, whereas in the T case it is possible to reach up to 50 degrees, which represents a small reduction. The same applies to other functions, e.g. with regard to visual image orientation, M presents an inverted vision of 170 degrees, whereas T presents a maximum tilt of about 30 degrees; and so on for

all the other visual functions and any other sensory system. The dynamic manifestations of functional sensory lag (functional disaggregation), facilitation effect, etc., which we shall see further on, are also related to the different degree of disturbance. Nonetheless, thanks to the summative effect of facilitation, an approximate quantitative similarity between these two cases can be obtained. For example, with respect to vision, subject M in binocular vision and under maximum muscular contraction can remarkably resemble subject T without facilitation and using only his worse eye.

As regards the Schneider case, several facts we know about it allow us to place it somewhere in between our two cases, the M, which is more severe, and the T, which is less intense. Thus, Schneider would reach up to 25 degrees in the concentric reduction of the visual field. The loss of visual perception of motion was quite remarkable in Schneider, as well as in M, whereas it is extremely small in the T case. For all this, it is perfectly feasible not only to ascribe to the Schneider subject an intermediate position between our two patients, but also to attribute him a series of disorders that have not been found in him, and that he should necessarily present. On the other hand, subject M under facilitation can be easily equated to subject Schneider, thus we can predict with great accuracy and detail the complex brain disturbance that corresponds to Schneider.

2.2. THE CENTRAL SYNDROME

On the basis of our two cases and the one studied by Goldstein and Gelb (1918, 1919), we have enough reasons to establish a new well-defined type of brain disorder that we shall briefly call *central syndrome*, and more precisely, central syndrome of the cerebral cortex.

It is clear that, according to all the above, the central syndrome is characterized by a new physiological and dynamic conception of brain activity in opposition to current theories of brain pathology.

The central syndrome is defined by the properties that we shall expose in what follows. First of all, the most characteristic feature of the symptomatology is the effect of the cerebral repercussion that causes the typical *symmetric disorder*, that is, the equal alteration of the two halves of the body. Regarding the anatomical lesion, it involves *central* cortical areas that correspond to the so-called association areas, different from the projection areas which occupy a marginal or peripheral position. Added to this is the excitability disorder which, according to dynamic actions, causes a *dynamic reduction* in brain functions. Next, we shall detail the characteristics of this syndrome that, ultimately, in its multiple aspects and developments, represents the topic of this work, and the starting point of the brain dynamics described here.

2.2.1. Total and symmetrical repercussion disorder

The lesion of central areas of the cerebral cortex, from which this syndrome takes its name not only because of the anatomical position of the lesion but mainly because it damages brain areas that function as true nervous centers, affects regions that

disturb the functional dynamic unit of the brain to a maximum degree. These regions are what were called association areas in the past, which are surrounded peripherally by the projection areas (that we call marginal zones) where the long pathways of the brain end (or originate). It could be admitted that these central areas only act by increasing brain excitability in a non-specific way, and thus subordinating the activity of all other functionally related brain regions, which means the dynamic unity of all regions. Thus, the destruction of brain matter in central areas causes, by dynamic repercussion, a disorder that affects all the functionally unified territories. In our two cases and also in the Schneider case, it has been shown that a lesion in the most central region of the sensory part of the brain disturbs all sensory systems in all their functions, both the simpler (lower) functions and the more complex (higher) ones. Furthermore, there is something perhaps more special, which is the alteration on both sides of the body and equally, that is, on the whole extension of the sensory field. Thus, the left lesion affects both the contralateral and the homolateral side, and both in a fairly equal way resulting in a symmetrical disturbance. This alteration of all sensory systems in both halves of the body is due both to the dynamic unity of one cerebral hemisphere and to the dynamic unity of the two hemispheres with each other.

It can be said that the more symmetrical the alteration in both halves of the body, the more central is the lesion, and it can also be expected that the alteration is extended to more than one sensory system. Thus, when we find a concentric reduction of the visual field, we should expect alterations of the same type for other sensory systems (tactile, auditory, etc.). In contrast, as the asymmetry becomes more noticeable, i.e., one side is more altered than the other, the central syndrome tends to become a syndrome marginal (i.e., of the projection zone), and there will be fewer altered sensory systems. In the extreme case, there will be only one alteration with maximum asymmetry and for only one sensory system.

Regarding symmetry, the M, T and Schneider cases can be considered as prototypes of the central syndrome.

2.2.2. Excitability disorder

If we consider a physiological basis and use the concept of nervous excitability, we find very interesting aspects in the central syndrome.

First of all, excitability is reduced as expected following the destruction of brain mass that directly influences excitability dynamically. The reaction time becomes longer, and an increase in the intensity of the stimulus is necessary; in other words, the most immediate change is a greater demand for the two factors involved in nervous excitation: time and intensity. This change causes a series of remarkable phenomena, depending on excitability, that we already know from the exposition of dynamic action phenomena in previous pages. In addition to a long reaction time, other phenomena such as iterative excitability and facilitation arise, which are different manifestations of nervous summation. Thus, where there was only excitation by a single stimulus in a normal individual, there is now also iterative excitation by rhythmically repeated stimuli, whose action lies in the accumulation of residual ef-

fects that add up until they trigger the reaction of the organ in question. It is similar to the changes that occur in experimental animals when parts of the spinal cord are separated from the brain. An iterative excitability then appears for reflexes, which were obtained by single excitation in a normal situation. In our cases, iterative excitation allows significant savings in the intensity of the stimuli compared to a single stimulation.

As for the action of facilitation, it increases the excitability of the centers, and thus compensates for a large part of the brain mass destroyed, thereby significantly reducing the various alterations of excitability, and establishing a more favorable physiological level.

With regard to the intensity of the nervous excitability disorder, it depends entirely on the amount of brain mass excluded by the lesion. Thus, the disorder is important in the M case, and much less pronounced in the T case. As for the deficit in elementary excitability (increased rheobase and chronaxie), the two types of summation, by iteration and facilitation, have a corrective effect on it.

A very important manifestation of the nervous excitability disorder is *asynchrony*, by which sensory functions are out of phase leading to the dynamic reduction described below.

2.2.3. Dynamic reduction

The excitability disorder causes a functional disaggregation or asynchrony. Depending on the physiological demands of sensory functions, an asynchrony arises that leads to a simplification or dynamic reduction of these functions.

As already mentioned, a sensory function that in a normal subject behaves practically as an all-or-nothing effect, i.e., it shows up in its entirety or fails completely, is disaggregated here into partial functions with different degrees of excitability. This gives rise to a partial effect. The greater the excitability disturbance, the easier this effect becomes evident. This is the case, for example, with the inverted vision phenomenon: a strong stimulation generates normal vision, a moderate one produces tilted vision, and a weaker one causes inverted vision. The same applies to any other aspect of visual function, such as the visual field, which shows in patient M a considerable concentric reduction by exclusion of the less sensitive peripheral areas due to the excitability deficit.

As the more complex (less excitable) activities are progressively excluded from the action of habitual stimuli of medium intensity, the sensory functions are simplified, i.e., dynamically reduced, since the most physiologically demanding activities are lost. Since dynamic reduction is closely dependent on the degree of excitability deficit, the reduction in subject M is much greater than in subject T.

In short, dynamic reduction is a natural consequence of nervous asynchrony.

The syndrome opposite to the central syndrome is the *marginal* (or peripheral) *syndrome;* both are the two main forms of a brain disorder. The marginal syndrome corresponds to a lesion of the cortical projection areas, so it is well known in brain pathology, unlike the central syndrome. A lesion of the projection areas, which are point of origin of the long pathways, is almost equal to the destruction or interruption

of these pathways, resulting in a *localized* disturbance in contrast to the general alteration of the central syndrome because of the dynamic repercussion of the centers. In the case of a marginal lesion, which occupies a peripheral position in relation to the central zones as its name indicates, there is not the symmetry effect that we have seen in the central syndrome. On the contrary, it causes a localized effect that disturbs in a very circumscribed way contralateral functions, for whose activity the conductibility of the long pathways traversing the marginal zone is vital. Taking the visual field as an example, the marginal visual lesion causes a hemianopic alteration in which the homolateral halves on the side opposite the lesion are blind whereas the other two halves remain unscathed. Nor should we expect other alterations outside the visual domain, i.e., touch and hearing, for example, can remain totally free.

This would be a type of extreme marginal disturbance, and it can be said to be more of a nervous pathway lesion than a lesion of the cerebral cortex, the latter being regarded as a nervous center. Thus, the marginal syndrome shows a type of disturbance that is essentially asymmetrical. Between the pure central syndrome and the marginal syndrome there is a whole series of intermediate types, and it could be said that most brain lesions belong to these intermediate types. In fact, it could be stated that every cortical lesion that does not exclusively affect the above-mentioned pathways, shares to a greater or lesser extent properties of the central syndrome. This will be studied later, and now the indication made about the types of brain disorder is enough, which helps to better characterize the central syndrome.

2.3. BRAIN DYNAMICS ISSUES RAISED BY THE CENTRAL SYNDROME

The characteristics of the central syndrome constitute, above all, a true *experimentum crucis* to decide on the functioning of the brain, and what then results leads directly to a dynamic conception of the brain. Thus, the brain lesions of the two cases studied here enlighten us about the mechanism of brain centers, completely ignored until now. This has entailed a profound change in the general ideas and raises fundamental issues in the theory of the nervous system that can only be addressed from the point of view of the dynamics of the brain. Next, we shall deal with three of the main issues: physiological level, sensory structures and brain system. We shall now restrict ourselves to indicate little more than their existence to highlight the most important theoretical consequences derived from the central syndrome. They will be the content of the third part of this work, devoted to the development of the general fundamentals of brain dynamics.

2.3.1. Physiological level

The excitability criterion is of primary interest for the physiological rationale of brain activity, hence, the first and most elementary issue to be considered in brain dynamics is the *physiological level,* which is an expression of nervous sensitivity to stimuli. The phenomena found here allow us to consider the cerebral cortex as a dynamic unit, that is, as a functional unit resulting from the activity of all the elements that

compose it. In this ensemble, the amount of nervous mass of the centers determines the excitability level (physiological level); hence the exclusion of a portion of such mass causes a decrease in the functional level due to the subtraction of nervous energy that this entails. This is what happens, as we have already seen, in the central lesion of our two cases and also of the Schneider case.

There are precedents of this approach, some very old, for example in the doctrine of Loeb (1899) by considering in a very broad way the activity of the nervous system as a simple action of reinforcement of the natural sensitivity of the organism to the stimuli of the external world. In a more precise and concrete way, there is a precedent in the experiments and explanations of the physiologist Lashley (1929) who continued the work of Flourens (1824), confirming and extending it. Lashley (1929), working with rats trained in the labyrinth's intelligence tests, found a close relationship between intelligent behavior and the amount of brain mass destroyed. Consequently, he formulated the *mass action* law, considering that this brain mass is *equipotential,* i.e., of non-specific physiological activity, and with the only effect of reinforcing brain function. More modern research on general physiology of the nervous system is also going in the same direction. In particular, Lapicque M (1923), Lapicque L and Lapicque M (1928), Lapicque L (1934 b), and his many followers demonstrated the interaction between the nervous elements with each other by means of nervous *subordination* effects, which highlight a much more dynamic functional structure than that assumed by the static anatomical theory of pathways and centers. The end of the predominance of the anatomical view is evidenced by the constant progress of such dynamic conceptions in the physiology of the nervous system.

The activity of the central mass, resulting from the sum of actions of the nervous elements that compose it, can be replaced by various other nervous actions, as seen in the phenomenon of facilitation, thus making it possible to largely compensate for the destruction of the nervous mass. All these circumstances prove the non-specificity of the nervous influx, which acts simply by summation, increasing sensitivity to external stimuli.

2.3.2. Sensory structures

The study of sensory activity in brain pathology has so far been based on an intellectualist psychology, in addition to trying to determine an anatomical localization in the cerebral cortex for each type of function. Thus, an endless series of "atomized" brain syndromes have been established psychologically and anatomically. Contrary to such theories, the phenomena found here evidence the functional unity of each sensory system, whose activity depends on the state of excitability of the system (receptor and nervous mass linked to it).

As for the sensory organization, it is carried out by means of the nervous mechanism of synchronization, i.e., excitability levelling of functional elements, thus obtaining more differentiated structures. This view, systematically adopted in the study of our cases, is an application of the theory of nervous isochronism of Lapicque (1934 a) to the human brain.

From a phenomenological point of view, the dynamic conceptions of the Gestaltists, such as Köhler (1930), Wertheimer (1912), Koffka (1919/ 1935), etc., can be usefully applied.

As a precedent, it should be noted the indications of Stein (1928, 1930) in favor of basing all kinds of sensory activity, from the most elementary to the most complex, on the characteristics of nervous excitability.

In any case, it is clear that both *psychologism* and *anatomism* are insufficient guidelines that must give way to more physiological conceptions.

2.3.3. Brain system (brain organization)

The central syndrome has very important consequences for the theory of cerebral cortex functioning, hitherto dominated almost entirely by anatomical localization theory. Nonetheless, this conception has always had its weak points and, although in general it is dominant, it can be said that authors have been divided into localists and anti-localists from its beginning. Although the theory of anatomical localization seemed to be well founded in restricted areas (projection cortex), there were still large areas to be explored, and the physiological laws of the nervous mechanism were very little known. Thus, generalizations have been premature and highly questionable. Even before physiologists and pathologists established the theory of brain localization, Flourens (1824), whose conception could be very well rehabilitated here, had considered the cerebral cortex as a functional unity, based on his experiments in laboratory animals, in which a destruction of brain mass caused a reduction of all brain activity and not of a particular function. We have already indicated how Lashley (1929, 1933, 1937) has corroborated and specified such a conception. In a similar way to Flourens, the physiologist and nervous system pathologist Goltz (1881) soon opposed to the nascent theory of brain localization, considering that function goes beyond structure.

The non-specificity is a consequence of the physiological level we have discussed above, assuming that the central mass activates nervous excitability, without any qualitative specificity. Thus, the rebuttal of brain localization is a direct consequence of the phenomena indicated on brain excitability.

However, the cerebral cortex does not have a completely homogeneous activity. For this reason, the cerebral cortex could be understood as a dynamic system, both because it constitutes a functional unit and because it changes its effects according to its different regions. The two extreme types of cerebral syndrome (central and marginal) limit a series of intermediate cases that prove the existence of a dynamic system layout.

We can say that the effects of a lesion on the brain system depend on the two following factors: the amount of brain mass excluded and the position of the lesion. Thus, the effect produced is in relationship with the *magnitude* and *position* of the lesion, fact that perfectly fulfills the conditions of a dynamic system. The magnitude, i.e., the amount of mass destroyed, determines the intensity of the excitability disorder and the degree of dynamic reduction suffered by sensory functions. The position of the lesion determines the type of distribution of the disorder, which is symmetric

for the central syndrome and asymmetric for the marginal syndrome, counting also with the intermediate types such as a disorder of general distribution but with asymmetrical predominance.

This is how the problem of brain functioning can be addressed, and the thorny issue of brain function localization theory solved. It is important to note that, except in the extreme marginal syndrome, which represents in fact a direct lesion of the nervous pathways, we should not speak of focal symptoms when referring to the cerebral cortex, contrary to conventional ideas. It is, of course, completely impossible when it concerns the central syndrome, since the theory of brain localization then fails completely. For this reason, it might be said that the dynamic conception developed here is the end of the controversy about localization of brain functions.

This dynamic conception of the brain system is the most immediate and simplest way possible to understand the facts derived from experiments. We can concretely illustrate the theory by resorting, as in other occasions, to the behavior of the visual field in different types of brain injuries.

In the patients studied here, we have the type of lesion corresponding to a concentric reduction, that is, a disorder that can only be understood from a dynamic conception, and that conforms perfectly to all indicated in the central syndrome.

For a marginal lesion, we have the hemianopic alteration, in which only the halves of the opposite side of the lesion are blind, and here, of course, the theory of localization is valid since we are dealing with an interruption or damage to the nervous pathways in the cerebral cortex (calcarine) where they end up. However, the symptoms may appear more complex in the first moments due to a certain nervous repercussion on the rest of the undamaged field, indicating that the dynamic mechanism is always present in all types of lesion.

Finally, we can still consider an intermediate type of disorder produced by lesions that, being in the projection areas or bordering areas, do not cause a direct alteration of the pathways. In these cases, if the patient is examined with due precision, it is usual to find a somewhat general distribution of the brain disorder but with asymmetric predominance. Most of the best known cortical syndromes undoubtedly belong to this type. In such a case the visual field shows the following characteristics: for each eye, the half opposite the lesion is blind except for the central zone, which can be restricted to the macular region; then the dividing line between the blind and healthy parts of the field of each eye is not a straight line but has a small concavity in the center, which corresponds to the most central zone (*Aussparung* of the macula according to German authors, whose point of view has been highly discussed). But the other half of the visual field, presumably intact, is not, since when it is properly examined, some degree of alteration can always be found. This alteration may range from a slight narrowing of color isopters to a concentric reduction affecting the most peripheral zones of the visual field of that half. Therefore, it turns out that both halves of the field are involved, albeit unequally, but according to the same mechanism. In the contralateral half, the functional reduction is so intense that vision is limited to the macular area (the most sensitive), whereas in the other half, the repercussion effect (action at a distance) is so weak that the alteration of the field

manifests itself only in the most peripheral regions of the field, which are more difficult to excite, i.e., less sensitive.

Through these three examples, the action of the brain dynamic system we advocate is specified. The dynamic action applies to all cases.

For the moment, it suffices the indications made on fundamental issues arising from the central syndrome, and to point out its theoretical scope for a theory of the brain.

3. Dynamic analysis

3.1. DYNAMIC ACTION AND EXPERIMENTAL ANALYSIS. PHYSIOLOGICAL LEVELS

The special conditions of excitability make it possible to carry out experiments of great importance to address the study of hitherto unknown brain mechanisms. Experiments of a great demonstrative effect can be carried out on broad aspects of sensory activities, in such a way that the factors involved in their formation can be discovered.

Basically, the exposition we shall make of brain dynamics is just the detailed study of the central syndrome. This special type of disorder offers very broad perspectives for research because, firstly, it allows any sensory system to be addressed in a single individual since all sensory systems are affected to the same degree, and secondly, because the special change in nervous excitability reveals the nervous mechanism that governs brain activity.

By supporting all research on a strict physiological basis, the study is developed free from psychological views and procedures that only obtain hypothetically interconnected data without reaching a direct and simple explanation of the phenomena. Even having to rely on subjective phenomena in the examined injured subjects, the analysis is ultimately well-established on the objective behavior of these subjects facing tests and experiments that, properly combined, allow a reliable control of the brain disorder.

Along with the experimental analysis of the *qualitative* factors that determine an activity, there is a *quantitative* assessment of them, this being the only way to complete the information, and to specify accurately the characteristics of the phenomena. Thus, in our two cases we try to express the different relationships of nervous excitability that intervene in the sensory functions by means of curves and graphs.

The dynamic nature of the fundamental disorder of nervous excitability determines the conditions of the experimental analysis to be carry out. This is not the place to explain in detail the causes of that fundamental alteration originated by the loss of central mass, but just to anticipate that it is characterized by a great *slowness in nervous reaction,* which gives rise to a special *asynchrony* of the nervous elements that

integrate brain activity. A manifestation of that slowness is the alteration of the intensity-duration threshold curve of the elementary excitation,[1] and a manifestation of the asynchrony is the fundamental experiment of sensory lag that causes a disaggregation of sensory functions into partial reactions.

On the other hand, summation effects by iteration and facilitation, caused by the essential disorder of excitability, act by partially avoiding this slowness as well as much of the asynchrony. In particular, the action of facilitation by replacing to some degree the loss of central mass, especially in the more severely altered M case, allows new types of physiological levels to be generated, thus providing a wide range for experimentation. Thus, there are several factors to consider in the experiments, factors that we shall use systematically in the study of brain functions. Given the general characteristics indicated, the study can be called dynamic analysis.

3.1.1. Physiological levels

Figure 3.1 schematically shows the change in the intensity-duration excitability curve of any sensory receptor, due to the loss of central brain mass. The lower curve (the one closest to the axes) corresponds to the excitability of a normal individual, and the upper curve represents the activity of subject M, who has the greatest brain mass deficit. Between these two curves we have the curve corresponding to the facilitated state by maximum muscular effort in the M case, and those expressing the state of excitability of the T case. Slightly below the T-curve is the curve for the subject T under maximum facilitation which causes a very small descent, as can be seen.

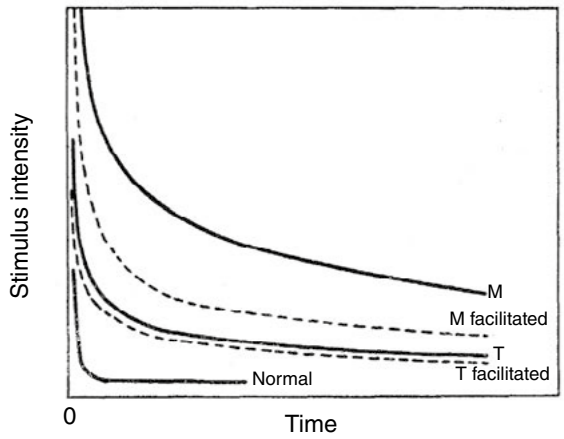


Figure 3.1. Schematic representation of the state of brain excitability according to the intensity-duration curve (called strength-duration curve for electrical stimulation), in the M and T cases, without facilitation and under its maximum action. A stepped series of different physiological levels with different excitability is obtained.

[1] Intensity-duration curve for threshold stimulation is called strength-duration curve for electrical stimulation. For other types of stimulation we shall call it intensity-duration curve.

Thus, a number of excitability states or *physiological levels* result depending on both the loss of brain mass and the facilitation that replaces this loss to some extent. Facilitation by maximum muscular effort produces a large increase in excitability in patient M and a much smaller increase in patient T. It is observed that the more pronounced the disorder, the greater the effect that can be expected from facilitation. The same happens with iteration, whose saving action in stimulus intensity is much more pronounced in M than in T, as we will see later on. However, there is not a complete recovery to the normal level of excitability but rather, as we have seen, the maximum facilitating effect is still quite far from the normal level.

The curves are characterized by two excitability factors: rheobase, which is the lowest value of intensity that produces a response, and chronaxie, which is the time corresponding to twice the rheobase. It should be noted the degree of curvature. In this series of curves, there is a gradation in the types of excitability in which the deficit is due to an approximately parallel increase in the two excitability parameters, time and intensity. Consequently, the curvature of the curve becomes less pronounced. Thus, the reaction speed is lower in the curves further away from the axes.

This series of excitability levels offers the possibility of investigating brain activity under very different conditions and, as we see, this is due not only to the existence of two subjects with a very different intensity of their lesion, but also to the combinations that can be made by means of facilitation, especially in the case of M. In Fig. 3.1, only the curves for the subjects in inactive state (without any facilitation) and under facilitation by maximum muscular contraction are indicated. However, other types of facilitation are still possible. By combining the effects of various facilitations, it is possible for the M case to become physiologically equivalent to the totally inactive T case. Although this equivalence may not be fully achieved, the physiological levels of both cases become very close to each other under these conditions. Thus, we have the possibility of converting, for example, the visual perception of subject M into that of subject T as long as the former is under the action of maximum muscular contraction and in binocular vision.

The very diverse types of facilitation (muscular contraction, sensory action, binocular vision, movements, etc.) give rise to very numerous types of physiological levels, whose exact knowledge is indispensable to establish with certainty the quantitative determinations in the experiments. In addition, facilitation is a very important means of extending and controlling all kinds of experiments, as we shall see later on.

3.2. FUNDAMENTAL EXPERIMENT OF ASYNCHRONY

The previously mentioned nervous asynchrony has its most immediate manifestation in the *fundamental experiment* on sensory lag (or delay), of great importance in the analysis of sensory functions.

We have previously dealt with the excitability deficit due to a loss of brain mass. This deficit affects differently the nervous elements that are involved in brain activity, and gives rise to a functional asynchrony that is easily revealed in the fundamental experiment. Nervous asynchrony, the degree of which depends on the

excitability deficit, is also greatly reduced by facilitation in the same way that the intensity-duration excitability curve becomes lower in Fig. 3.1. But the normalization of asynchrony is not complete either, although the pathological alteration is significantly reduced.

Using excitability relationships, the fundamental experiment is presented under two types of graphs that are different expressions of the same asynchronous process: one type is the asynchronous bundle of curves, each one corresponding to the different (now independent) partial functions resulting from functional disaggregation by sensory lag (dephasing); the other type is the sensory recruitment curve of these disaggregated functions. These relationships are complemented in a very important way by the summative action of facilitation which, by correcting the disorder through central action, tends to synchronize the disaggregated functions.

In the following, we shall briefly indicate the characteristics of these excitability relationships in the fundamental experiment.

3.2.1. Asynchronous bundle of disaggregated functions

Functional sensory lag (asynchrony) spreads to all kinds of activities of any system: vision, touch, hearing, etc. As already indicated, it was first found in the phenomenon of inverted vision in which we already know the different effect of strong and weak stimuli. Strong stimuli give normal or almost normal vision whereas weak stimuli, on the contrary, give inverted or strongly tilted vision. If in this phenomenon, which can now be considered a paradigm of sensory lag or asynchrony, we determine the intensity-duration threshold curves for the various functional levels (different orientations of the perceived visual image), we find a series of curves like those shown in Fig. 3.2.

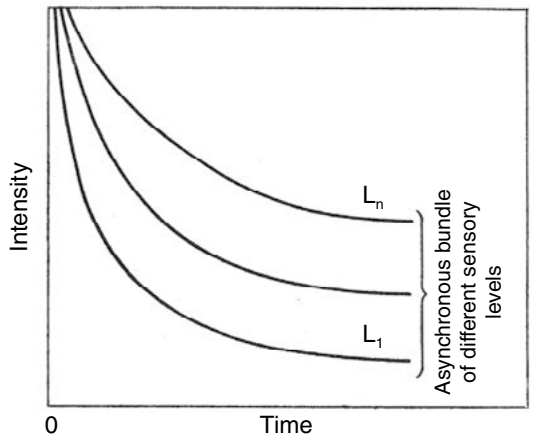


Figure 3.2. Schematic representation of the asynchronous bundle of curves corresponding to the fundamental experiment of asynchrony. These intensity-duration curves are threshold curves corresponding to the disaggregated partial functions, L_i, that would form a single curve (single sensory level) in a normal individual.

What in a normal individual would give a single curve, now, due to asynchrony, becomes a bundle of curves placed far above the normal value; the greater the asynchrony, the wider the bundle. In this family of curves, the lowest curve corresponds to the most sensitive sensory function or level (highest excitability), which in our case is inverted or strongly tilted vision, and the uppermost curve corresponds to normal oriented vision, which requires more excitation. Between these two curves there is a continuous series of curves corresponding to the different tilts (intermediate functional levels). The schematic representation in Fig. 3.2 shows that the higher the curve, the lower the curvature (indicating a lower reaction speed); all the characteristics of the higher curve indicating a lower sensitivity.

In the asynchronous bundle of curves there is a series of sensory levels corresponding to the disaggregated partial functions, sensory level being understood as the rheobase threshold of the sensory manifestation. Due to the property of the intensity-duration law, the curves of the asynchronous bundle tend to be closer together for strong stimulus intensities, and to separate from each other for small intensities or long times, showing then their respective rheobase levels.

The greater the excitability deficit, the more pronounced the asynchrony and therefore the greater the separation between the curves. Hence, this separation is much more pronounced in the M case than in the T case, and by means of facilitation it is possible, especially in the first case, to shorten such separation by a considerable degree.

For the moment we only consider, for simplicity, the general aspect of the asynchronous bundle without paying attention to the restrictions imposed by each case according to the intensity of the lesion and permeability to facilitation.

3.2.2. Recruitment of desynchronized levels

The other aspect of the fundamental experiment lies in the recruitment of the disaggregated (desynchronized) partial functions.

In the recruitment experiment, the rheobase levels of the disaggregated functions can be reached by progressively intensifying the stimulus. That is, each sensory level is related with a certain stimulus intensity, without intervention of time, which is assumed to be unlimited because we are dealing with the rheobase threshold. In the case of visual image orientation, changes in the intensity of the stimulus easily occur when an object moves away from or towards one of these patients because the visual angle at which the object is seen changes as well as the intensity of the light reflected by the object. By increasing the intensity of the stimulus in this way, it is possible to go from the strongest inversion to the most normal upright orientation, the sensory level being determined by the degrees of rotation of the visual image.

This is an experiment of sensory growth as a function of the intensity of the stimulus, expressed by Fechner's law (Fechner, 1860) which is also fulfilled in this pathological case of nervous asynchrony, as can be seen in Fig. 3.3 where the recruitment experiment is schematically represented. By taking the logarithm of the stimulus intensity at the abscissa, the sensory level grows directly proportional to the logarithm of the stimulus, although this relationship is only fulfilled exactly for the central part of the curve.

As can be easily understood, this recruitment curve is related to the curves that form the asynchronous bundle. Both types of experimental curves are aspects of the same phenomenon.

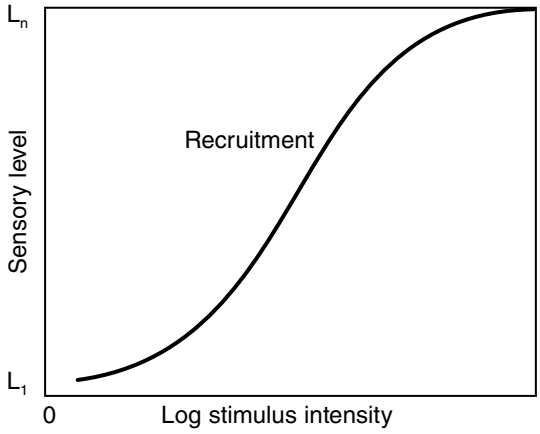


Figure 3.3. Recruitment of sensory levels L_i as a function of the logarithm of stimulus intensity. The levels correspond to the rheobase thresholds of the desynchronized (disaggregated) functions shown in Fig. 3.2.

Another curve of the same type is obtained if, instead of relating the sensory level to the intensity of the stimulus acting for an indefinite time, the action time of the stimulus is the variable on the abscissa for a fixed medium intensity. In this case the recruitment is determined as a function of time, i.e., we obtain the development in time of the series of desynchronized functions, from the simplest (and fastest) to the most complex (and slowest). In this type of experiment, easy to carry out due to the great slowness in the reaction of the subjects, it is necessary to fix a medium intensity of the stimulus that cannot be lower than the rheobase threshold of the highest sensory level, otherwise only lower levels would be reached by the action of time, and an incomplete recruitment would be obtained. We can see in Fig. 3.2 the basis of the two types of recruitment exposed, that is, either by the action of increasing stimulus intensity or by the action of increasing time (a much slower process), a succession of sensory levels appears from the most anomalous function to the most normal one which appears last.

3.3. SYNCHRONIZATION BY FACILITATION

The new type of experiment that completes the sensory analysis by making use of dynamic phenomena, is synchronization by means of facilitation. The latter acts in a summative way on the centers of the cerebral cortex, thus obtaining an action that is opposite, and to a certain extent symmetrical to that of asynchrony.

Taking again the example of inverted vision, we consider the situation in which subject M, being in an inactive state (free of any facilitation), perceives the inverted (or almost inverted) image of an object due to its distance, size and illumination conditions. If then he makes a muscular contraction, a re-inversion is produced; the greater the muscular effort, the more pronounced the re-inversion. This type of facilitation can easily be measured if the subject holds a certain weight under conditions that will be detailed later on when the different experiments will be explained in detail.

Figure 3.4 represents the effect of synchronization by facilitation due to muscular effort, showing the growth of the sensory level as a function of the logarithm of muscular effort, this one measured by the weight held by the subject. The starting point is the state of maximum asynchrony, in our example it is the most inverted image, which corresponds to the lowest sensory level, and also to a state of absolute inactivity of the subject; then the degrees of tilt of the visual image corresponding to each weight held are determined. If these degrees are indicated on the ordinate, and the logarithm of the held weight on the abscissa (the weight can be up to 80 kg to reach maximum synchronization), a sensory growth curve is obtained analogous to that obtained in the previous experiment of sensory recruitment by stimulus intensification. But whereas in the recruitment the increase of the stimulus intervenes, in the experiment with facilitation the sensory increase is due to the summative effect exerted on the nervous centers, synchronizing the sensory levels, that is, equalizing their excitabilities and forming again a unitary function.

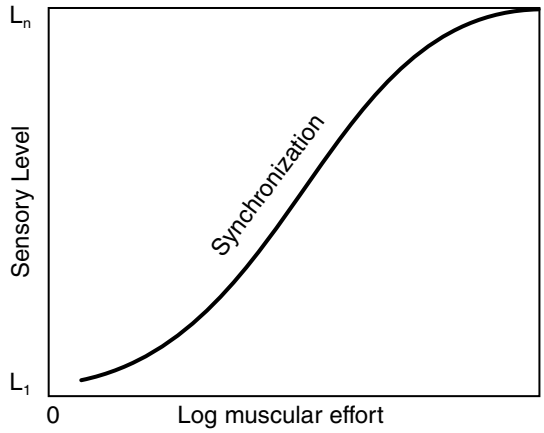


Figure 3.4. Diagram of the effect of synchronization by means of facilitation due to muscular effort for a fixed stimulus intensity, showing the growth of the sensory level by the progressive action of the applied effort.

However, even maximum facilitation does not achieve *ad integrum* recovery. Thus, if in the described experiment we start from the most strongly tilted or inverted image, it is not possible to achieve by means of maximum facilitation a complete re-inversion of the image, although one very close to it. Let us recall that the degree

of permeability to facilitation is very diverse according to the degree of brain disturbance; it has a great effect in the M case, and is much weaker in the T case, diminishing the effect notably as the excitability deficit decreases. In any case, the action of facilitation in both subjects is so great that in the conditions of ordinary life, it practically counteracts the asynchrony defect. Only in the most accurate determinations of experimental tests, it can be shown that a small asynchrony persists even with maximum facilitation.

In addition to the two ways of growth of the sensory level, by intensification of the stimulus and by facilitation, there is a third type due to the existence of iteration. In this type of stimulation, a stimulus of constant intensity corresponding to the lowest sensory level is applied repeatedly, resulting in increasingly higher sensory levels as the number of stimuli increases. This is an iterative recruitment in which the accumulation of stimulus residues, which slowly fade due to the state of nervous excitability, causes the stimulation effect to progressively increase. The curve thus obtained is similar to that of recruitment by intensification of a single stimulus. Therefore, there are three different forms of sensory growth depending on the stimulation conditions: recruitment by intensification of a single stimulus, recruitment by iterative stimulation and synchronization by facilitation, with similar curves being obtained in all cases. We can say that sensory growth is proportional to the logarithm of nervous stimulation provided in one way or another.

Whereas iteration is in fact a special mode of stimulation, i.e., it is a sum of successive stimuli acting directly on the receptor in question, facilitation acts by modifying the fundamental excitability disorder (the excitability deficit and asynchrony). The change in the excitability level (or physiological level) by facilitation due to maximum muscular effort was already indicated in Fig. 2.3, bringing the intensity-duration curve of the elementary response of the sensory receptor closer to the normal value. Facilitation works by reducing asynchrony in the fundamental experiment, and decreases iterative capability. The latter effect is due to the fact that the sensory system becomes faster, more sensitive and more synchronous; hence the saving obtained by iteration (in the intensity of the stimulus) with respect to a single stimulus, and the degree of iterative recruitment are reduced. In summary, it is sufficient to emphasize that the fundamental experiment of asynchrony and the experiment on the effect of synchronization by the progressive action of facilitation, are experiments that close a complete cycle of tests in the dynamic analysis of sensory functions.

3.4. SENSORY ASYNCHRONY

We have indicated the characteristics of the fundamental experiment, and next we shall expose the meaning of the sensory lag (asynchrony) that arises in that type of experiment.

As already mentioned, the nervous origin of this type of phenomena lies in the asynchrony of the nervous elements that constitute the centers of the cerebral cortex. By virtue of asynchrony, not only the interval or gap of excitability existing in the normal individual between the diverse functions increases, but also other intervals

or gaps are created where there was no separation, thus appearing new modalities of reaction to the stimuli. An example of the pathological increase of the normal interval (gap), due to asynchrony, is found in the photochromic interval of colors, which being minimal and even almost imperceptible for some of them in a normal individual, is extremely increased, and clearly appreciable in most colors. The same occurs in other differential intervals such as those that exist naturally along the visual field meridian in which sensitivity decreases rapidly from the center to the periphery, or in other functions such as spatial discrimination as a function of stimulation intensity, etc. In all these cases, due to the increased interval, a higher intensity of stimulation is needed to overcome it, which means a loss of differential sensitivity.

Therefore, the most remarkable characteristic of asynchrony in the fundamental experiment is to generate new intervals (gaps) of excitability, thus causing the disaggregation of functional complexes that in a normal subject react "all at once," that is, according to the all-or-nothing effect. Due to the new intervals generated, the functional complexes are dissociated or fragmented into independent functions, giving rise to partial reactions or effects.

It can be admitted that, both for the normal intervals (normal differences in excitability) that become larger and for the new intervals generated, we are dealing with an abnormal increase in excitability differences, which within normality only some of them are evident whereas others are insignificant without establishing discontinuity. These new intervals, by fragmenting the unitary functional complexes, provide a kind of analysis of the sensory structures. In this analysis, we must distinguish the phenomenic aspect of the sensory manifestations and the level of excitability (physiological characteristic) of each of them. In this way, the mechanism of formation of the sensory organization is revealed through the separation of the factors that compose it. These factors are ordered in terms of their excitability according to their degree of sensory differentiation. For example, the process of tactile spatial localization is disaggregated, first appearing the sensation of simple contact without any indication of the location of the stimulus, and then appears, as a last sensation, the function of precise localization, both processes being completely independent and separated from each other, which is completely impossible in a normal individual. Between the two processes there are several intermediate states corresponding to diffuse ("irradiated") localization, similar to what happens in flat color vision (chromatic "irradiation"), as we shall see later.

Particularly important for the analysis of functions are cases of asynchrony in which extremely unusual manifestations occur that have no precedent in normal sensory activity, such as the phenomenon of inverted vision and chromatopsia (colored vision), for example. From the dynamic analysis of the first example it follows that there are two different types of orientation of the visual image; one normal, guided by the position and movements of the body, and another inverted on the retina which would decide the orientation.[2] In the example of chromatopsia, in which affected subjects perceive white as pale green, the various colors that contribute to

[2] A more complete analysis of this issue is provided in Volume 2 and in Supplement I.

the formation of white do not come into action simultaneously because of their different excitabilities. In this way, it is possible to explore the brain mechanism in the formation of both white and other colors, thus determining which are primary and which are composite (mixed).

These examples among many others show the significance of asynchrony in the research of sensory structures, and the useful applications of the dynamic analysis. Functional types that appear independent as a result of functional disaggregation are not always mere partial functions that in a normal situation are integrated into the total unified function. If this were so, one would have to admit that inverted vision is included in normal right vision, and yet both types of vision are mutually exclusive. More accurately, all these facts must be understood on the one hand as expression of the factors involved, and on the other hand as new physiological predispositions derived from the fragmentation, constituting simpler and less differentiated manifestations.

Thus, in the fundamental experiment of sensory asynchrony, we not only witness an analysis of the sensory structures but also a development and differentiation of sensory activity that goes through different stages in line with their own level of excitability. What is shown throughout the new intervals is a series of functions that in going from one to the other, an increasing differentiation and sensory organization are developed.

3.5. DYNAMIC REDUCTION

A consequence of asynchrony is dynamic reduction, which means a functional simplification arising from the nervous asynchrony disorder. The latter, by producing an increase in the differential interval, and even creating new ones, causes a large part of the sensory activities to be excluded under stimuli of ordinary intensity, resulting in a reduced function, but reduced according to its nervous excitability level; this is why the process can be called dynamic reduction.

Complex functions with a high level of excitability, i.e., requiring high intensity and long duration of the stimulus, rapidly increase their excitation threshold and are replaced, in the face of ordinary stimuli, by other simpler and more easily excitable functions. The greater the asynchrony that causes the separation between the various functional levels, the more intense the reduction. All these functional levels suffer from an excitability deficit, but in greater proportion the higher ones, corresponding to more complex and differentiated functions.

It can be stated that the dynamic reduction, generated according to the exposed mechanism, is the characteristic type of disturbance of brain activity for any type of anatomical lesion and, in general, for any type of alteration in the nervous system. It is a well-known fact in nervous pathology that in the impairment of a sensory system, mainly the most complex and differentiated functions are altered, and this occurs quite independently of the anatomical location of the lesion. However, such a significant fact has not acquired all the importance it deserves on a theoretical level, and with the exception of authors such as Stein and Weizsäcker (1927, 1928), it

has hardly been taken into consideration, since it is in clear conflict with the theory of anatomical localization. The mentioned authors advocate a functional unity for a given system, and reject the specificity of pathways and centers of the anatomical theory of nervous functioning. According to them, the excitability of the system is altered, and its functional character varies. However, they do not sufficiently specify this change of the functional character, but we can say on the basis of our experiments that this change is entirely due to nervous asynchrony, which raises differently the excitability thresholds of the brain functions, thus causing a dynamic reduction. For example, concentric reduction of the visual field is easily explained from the point of view of dynamic reduction; by contrast, it represents an insurmountable obstacle to any attempt to explain it according to the theory of anatomical localization in the brain. This dynamic reduction of the visual field is not an isolated case but the general rule for all kinds of nervous functions.

In such simplification or functional reduction, there is no inextricable chaos of symptoms as would be the case if we had to strictly abide by the theory of anatomical specification. The lesion, by provoking the excitability deficit that gives rise to the above-mentioned asynchrony, establishes a type of nervous organization at a different excitability scale from the normal one but according to the same general laws. Therefore, there is no radical separation between a normal and a pathological state, and from a broad point of view it is possible to determine the quantitative variations that establish a rational transit from one state to another.

A notable feature of dynamic reduction in the cases of central syndrome studied here is the reversibility of this reduction; this allows recruitment of the more delayed functions by sufficiently increasing the intensity of the stimulus. Thus, in subject M and using the worse eye, it is possible to obtain vision in the most peripheral part of the visual field and even to obtain a correctly oriented image without applying any facilitation and only by increasing the intensity of the stimulus. It is clear that dynamic reduction depends on the excitability deficit, that is, on the degree of asynchrony; thus, in our two cases we find very different degrees of dynamic reduction, which in addition can be modified by facilitation. However, in a very pronounced nervous disorder, asynchrony would distance the levels considerably from each other, and reversibility disappears.

The indicated reversibility presents a type of behavior very similar to that of Pflüger's strong and weak reflexes (Pflüger, 1859, 1877), in which a weak stimulation produces a partial or restricted reaction, and a strong one causes an extended and general reaction. In all cases, the relationships of excitability and asynchrony are fulfilled in the same way.

3.6. SENSORY STRUCTURES

If asynchrony leads to a functional disaggregation and a simplification or de-differentiation of sensory structures, its opposite effect consists in the synchronization of the constitutive factors and in sensory differentiation. Therefore, the degree of differentiation runs parallel to the degree of synchronization.

The structures of a given sensory system respond to a unitary global activity in which it is not possible to separate autonomous processes. For example, functional vision impairment occurs in brightness, colors, visual field, visual acuity, image orientation, and recognition and understanding of shapes and schematic drawings. The alteration affects the whole system without exception, and each function suffers according to its particular conditions of physiological demand. At the beginning of these studies, following the criterion of nosological syndromes of the usual brain pathology, it seemed that we were dealing with a series of independent disorders, but a more precise quantitative examination demonstrating a general disorder, gave way to the idea of a functional unity, thus ruling out any attempt to establish nosological units and anatomical localizations.

Unlike the *anatomical psychology* exercised by classical authors of brain pathology, we shall now study all kinds of sensory activities on a strictly physiological basis, from the point of view of nervous excitability. For each sensory system, discrimination and functional organization depend directly on its dynamic conditions, each system constituting a compact activity in which it is impossible to point out processes independent from the general activity. This conception is contrary to the rigid separation of brain activities into independent faculties such as sensation, perception, recognition, etc., used by classical psychology and adapted by brain pathology to clinical nosology. Likewise, the exclusion of the principle of brain localization means the rebuttal of the usual nosology that follows the criterion that a disturbance corresponds to a focal symptom.

Instead of classifying brain disorders according to a series of entities or nosological syndromes whose number grows endlessly (they are only partial aspects of a more extensive and general disorder that goes unnoticed), this issue would be greatly simplified by taking into account the *degree of alteration of the sensory system*, i.e., the level of differentiation and organization to which it is reduced as a result of the lesion. Such a functional level is expressed both by sensory manifestations and by nervous excitability relationships. In sensory differentiation, we witness a progressive and unitary qualitative-structural development, that is, a multiplication of qualities (e.g., development of the chromatic series in innumerable hues) and an increase in the spatio-temporal relationships that give rise to forms and structures such as localization, shape, motion, etc. This long sequence is obtained through gradual transitions in which one function is transformed into another, without any discontinuity. There is a continuous transition from the most elementary sensations to the most complex intellectual activities.

However, for a clear exposition we need to establish divisions that have only a circumstantial value, and do not compromise the unitary functional principle that has been advocated. Therefore, in the next part, the following classification of sensory functions will be used for all the sensory systems studied (visual, tactile and auditory): *sensations*, *forms* and *schemas*. Sensations correspond to specific manifestations of each system (luminosity and colors; pressure, pain and temperature; noises, sounds, etc.); forms refer to spatial and temporal structures of all kinds (localization, shapes, motion, rhythm, etc.), and finally, schemas represent a special type of structures directly derived from the last group, and constitute the basis of superior or intelligent behavior.

Sensory dynamics. Visual functions

Sensory structures according to brain synchronization

ONCE the general orientation of these investigations has been established in the preceding pages, we shall proceed to study more specific issues concerning the sensory dynamics of the three most important sensory systems: visual, tactile and auditory.[1]

This study consists essentially of investigating the organization of sensory activities, which are presented under two fundamental aspects. Firstly, the phenomenological aspect, which is the sensory expression itself, and which we address either through the subjective manifestations of the patient under examination or by objectively monitoring his behavior in the face of a wide variety of tests. The second aspect is the determination of the excitability level and other physiological characteristics corresponding to each sensory manifestation. Thus, both qualitative and quantitative manifestations of excitability are addressed. The latter is of the utmost importance for brain dynamics.

The working method consists of the application of the dynamic analysis already described. Therefore, it is mainly a matter of determining the conditions for the fundamental experiment of asynchrony and dynamic reduction in all types of sensory functions. It is then shown that sensory organization has its physiological basis in the degree of nervous synchronization of the functional elements of the cerebral cortex.

The investigation described below is entirely original, and whenever there is a precedent it will be duly indicated. In the exposition we shall limit ourselves to the essential facts, excluding details and discussions that would demand a much greater extension.

The sensory activity of the visual system has always provided the greatest number of problems to research, given the complexity and variety of its functions, and its easier accessibility compared to other sensory receptors. Here we have a wide field of experimentation that presents aspects as diverse as: general excitability conditions, color vision, structure of the visual field, forms, motion, mechanism of the visual image orientation, etc., and finally, the most complex activity of schema function that corresponds to the so-called visual recognition.

In all these topics we present new facts and experiments. As far as possible, the experimental results are represented by graphs and curves that show in a simple and immediate way the character of the phenomena. This is performed in the most complete way, especially in the experiments on visual image orientation. Of the two patients studied here, subject M is the most relevant, and subject T is more like a complement.

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[1] The auditory system is not covered in this book. Only brief rereences are made to it.

General excitability

4. Electrical excitability

4.1. Strength-duration curve (Hoorweg's law)

Of the two main ways of determining the state of elementary excitability in the visual system, electrical stimulation and luminous stimulation, we shall first deal with the former, which provides more important and accurate data.

Excitability behavior for the most elementary response function in both types of stimulation is the starting point for the study of more complex and differentiated functions.

In the subjects we study, we can get a very precise idea of the physiological state of the visual system by electrically stimulating the retina. The simplest and most elementary of all manifestations is the strength-duration curve,[1] which we shall examine first. Next, we shall study the very special phenomena concerning facilitation and iteration. This set of quantitative determinations shows with great accuracy the degree of nervous excitability disturbance in the central syndrome.

Of the two procedures to determine the strength-duration curve, the one of Weiss (1901) by means of a ballistic rheotome to obtain a direct measurement of time, and the other, of Hoorweg (1892) by capacitor discharge, we utilize the latter in all our tests, employing the usual device of Bourguignon (1923) for chronaxie determinations. As is well known, time is measured in this method by the discharge duration of the capacitor used; the greater the capacitance of the capacitor, the longer the discharge duration. Thus, the strength-duration curve is replaced by that of voltage-capacitance. The active electrode or cathode is applied to the upper eyelid, and the anode to any limb. The active electrode, which ends in a small sphere of 5 mm in diameter, should be applied gently on the upper eyelid to avoid the production of photomas that could hinder the perception of minimum luminosity, which is used as a test when the electric current is acting. In order to have a higher voltage, the final resistance of the circuit is reduced to 3000 ohms instead of the usual 10 000 or 20 000 ohms, so we will refer to

[1] Since the threshold stimulus is electrical, the curve is called strength-duration curve.

the capacitance in microfarads, without converting them into time units. This is not a disadvantage, and instead measurements can be made that would otherwise be very restricted, given the excitability conditions in the subjects studied.

Instead of restricting ourselves to finding only the rheobase and chronaxie capacitance, a series of values are determined to construct the entire excitability curve, thus showing in the most complete way the state of sensitivity of the sensory system. Given the characteristics of nervous excitability in our subjects, a series of precautions must be taken, without which the result obtained would be completely wrong in terms of the strength-duration law. The three main aspects to be watched are described as follows.

First of all, it is necessary to avoid any possible facilitation phenomenon, which would increase visual excitability, and thus hide its true state. To this end, the tests must be performed when the subject is in complete muscular relaxation and free from all activity, i.e., in an inactive state. This condition must be fulfilled especially in subject M, who has a great capability for facilitation.

Secondly, given the extraordinary latent addition (or iterative excitability) capability, each determination for a single stimulus must be suitably distanced from other excitations. Otherwise, the stimulus residuals, which fade very slowly, are easily added up, and a much lower value of the applied stimulus intensity than that corresponding to a single excitation would be obtained. The stimulations should be at least 30 seconds apart, and sometimes even more, depending on the subjects. Lapicque (1926) has already warned about the considerable errors that can be made in determining rheobase in tissues of slow excitation, as is the case of these subjects. In them it is not indifferent whether the determination is made by increasing the voltage or decreasing it, obtaining in the second case much lower values, and thus obtaining a measure not of the characteristic excitability of the prepared state but of the transitory excitability due to a previous operation.

Finally, we must take into account the easy fatigability of these subjects, which raises the necessary stimulation values, constituting another source of errors. For all these reasons, it is understood that electrical measurements require great patience since each point of the curve must be determined by successive approximations.

Table 4.1. Rheobase in volts (V) with direct current, and chronaxie in microfarads (μF), in patients M and T in inactive and facilitated states.

	M, right eye	**M, left eye**	**T, right eye**
Inactive	Rheobase: 14 V Chronaxie: 3.5 μF	Rheobase: 15.2 V Chronaxie: 3.7 μF	Rheobase: 7.8 V Chronaxie: 1.4 μF
Facilitated by muscular effort	Rheobase: 9.5 V Chronaxie: 2.7-2.9 μF	Rheobase: 10 V Chronaxie: 3-2.9 μF	Rheobase: 7.2 V Chronaxie: 1.2 μF

Beginning with M, Table 4.1 shows the values found for both eyes in the excitation by means of capacitors. The sensitivities of both eyes are quite similar al-

though somewhat higher in the right eye. The strength-duration excitability curves for the right eye of patient M, both in inactive state and under maximum facilitation by strong muscular effort, are shown in Fig. 4.1. The upper curve corresponds to the inactive state (complete muscular relaxation); the middle one corresponds to the state of facilitation by maximum muscular effort, the subject contracting all the musculature with maximum strength every time an electrical determination is made; finally, the lowest curve represents the excitability of a normal subject under the same general experimental conditions.

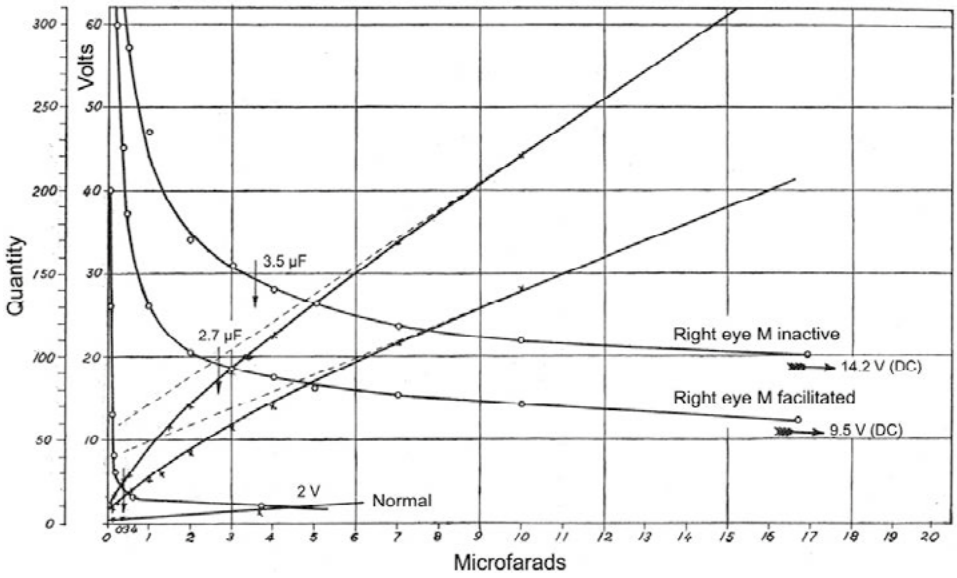

Figure 4.1. Strength-duration curves according to the Hoorweg' law (volts versus microfarads) for the right eye of subject M. Upper curve: inactive state. Middle curve: under facilitation by strong muscular effort. Lowest curve: normal subject. Note the different rheobase values in direct current (DC), and the different chronaxie capacitances (indicated by vertical arrows), as well as the different bend in the strength-duration curves and in the electricity quantity lines (left scale).

The results obtained show the huge difference between the values of the inactive state of subject M and those of the normal subject. In the M case, the two excitability factors, strength and duration of the stimulus, increase considerably and in a more or less parallel manner, since chronaxie is increased by about ten times, and slightly less rheobase. The pathological curve also shows a much smaller curvature than that of the normal subject, which corresponds to a lower speed of nervous reaction. This can also be seen in the electricity quantity lines starting near the coordinate origin and crossing the figure. Whereas it is practically a straight line in the normal subject, it has a strong curvature towards the origin in the pathological case.

As for the facilitation curve, i.e., the curve of the excitability state reached under maximum contraction of all the voluntary musculature, it is situated between the curve of the inactive state and that of the normal subject, although much closer to

the former. Therefore, facilitation has the effect of bringing the pathological curve of the inactive state closer to the normal one, reducing rheobase and chronaxie values, and consequently increasing the reaction speed. Therefore, the curve has a more pronounced curvature than that of the inactive state, but still shows a very slow reaction speed compared to that of a normal subject.

It is important to indicate that the facilitation permeability, and therefore the rapprochement to the normal values of excitability, presents a limit that is reached relatively soon, the values of the facilitated state still being very distant from the normal values.

Concerning the other subject, patient T, the values of excitability in his right eye, which in this subject is somewhat less excitable than the left eye, are shown also in Table 4.1. The values are considerably lower than those of the previous case, since the parallel increase of the two excitability parameters is approximately four times the normal value, as compared to about ten times for the inactive state of case M. The T case also presents the facilitation phenomenon, but much less pronounced because of his less severe disorder. The facilitation effect is manifested sufficiently, and can be shown in the electrical excitation tests (Fig. 4.2). Thus, the rheobase voltage drops a little more than half a volt by the action of maximum muscular contraction; this drop is maintained throughout the curve, and being so small, no change in the chronaxie value is indicated in Fig. 4.2.

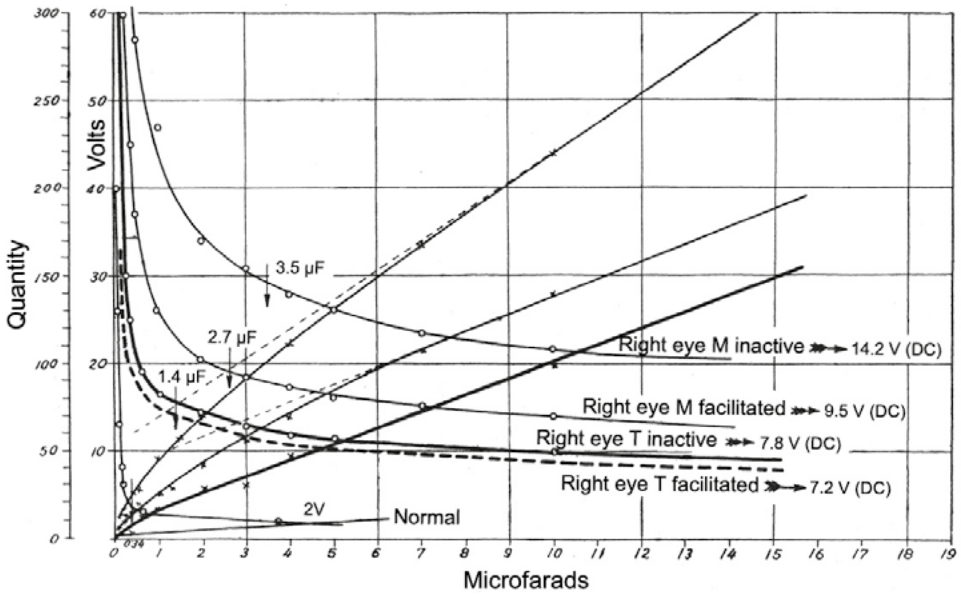

Figure 4.2. Strength-duration curves according to the Hoorweg' law, as in Fig. 4.1 but with the T case added: right eye of T inactive (thick solid line), and right eye of T under facilitation (thick dashed line) which shows a small reduction of the disturbance. Note the different values and the different curvatures, in particular in the electricity quantity lines (left scale); in the T case, only a small curvature close to the origin.

In this Fig. 4.2 we can see the state of excitability of subject T compared to that of subject M and that of a normal subject, as well as the very different effect of facilitation by muscular effort in the two studied cases. The closer the strength-duration curve to the normal position, the lower the permeability to facilitation. The small descent of the curve for subject T under facilitation is obtained both by intense muscular contraction and by very active movements of the head and extremities, and when both types of facilitation are combined, a much more evident descent is obtained. Much more care is needed in subject T than in M to highlight the small difference between the inactive state and the facilitated state in electrical excitation tests. This difference appears much more easily in other experiments, in particular in the phenomenon of inverted vision, which appears only as tilted vision in subject T, and by means of facilitation the tilt varies very clearly.

In the T case, the excitability curve has a more pronounced bend, hence the excitability state is quite fast compared to the two states of the M case, especially the inactive state, although when compared to the normal subject, T still offers a rather considerable slow reaction time since the chronaxie is about four times higher. The different curvatures of the electricity quantity lines corresponding to the respective strength--duration curves also characterize the different reaction speed in the cases studied. The greater the curvature of the quantity line, the lower the speed of reaction.

In the excitability disorder we therefore find a functional decrease since there is an increase in the excitability parameters, rheobase and chronaxie, and therefore lower curvature of the voltage-capacitance (strength-duration) curve. Such a decrease is dependent on the amount of brain mass destroyed by the lesion, and constitutes a loss of nervous action of central subordination (Lapicque M 1923; Lapicque L and Lapicque M 1928; Lapicque L 1934 b). Thus, there are different degrees of excitability disorder which, including the states under facilitation, can be sorted by decreasing disorder as shown in Table 4.2.

Table 4.2. Different degrees of excitability disorder in patients M and T.

	Rheobase (V)	**Chronaxie (μF)**
M, inactive state	14.2	3.5
M, under facilitation	9.5	2.7
T, inactive state	7.8	1.4
T, under facilitation	7.2	1.2
Normal subject	2.0	0.34

This type of alteration of excitability (increase in the required intensity and duration of the stimulus) is also produced both by the action of curare and by cooling, since both agents slow down functional speed. Thus, we find a similarity between the behavior of the excitation curves in our cases and in the case of curare poisoning in rapid response tissues such as striated muscle.

In Fig. 4.3, adapted from that of Lapicque (1938), it can be seen how curare poisoning modifies muscle excitability by increasing rheobase and shifting chronaxie to long times, the curve showing a much smaller curvature, which makes the quantity line concave downwards. Such a decrease in the speed of reaction is what we find in the central syndrome, and the greater the excluded brain mass the more pronounced the alteration. We also find that facilitation can to some extent compensate for this deficit depending on the case, thus showing a certain equivalence with the action of the lost nervous tissue mass.

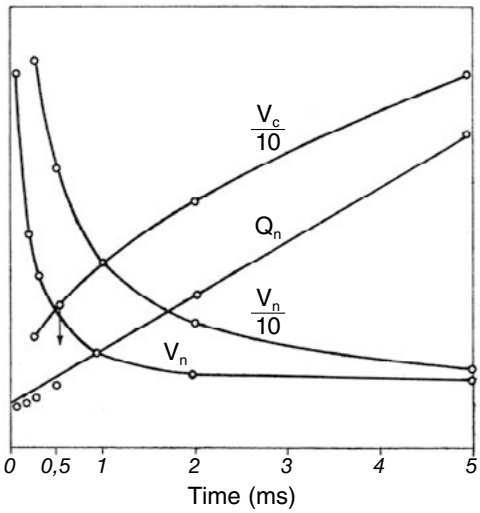

Figure 4.3. Strength-duration curves for the gastrocnemius striated muscle of a normal frog, and poisoned by curare, according to Lapicque (1938). Vn and Qn: voltage and quantity curves respectively in the normal state of the muscle. The quantity curve is practically a straight line. Vc and Qc: voltage and quantity curves respectively for the same muscle after a strong dose of curare. They are divided by 10 to bring the curves closer to the normal curves. The quantity curve Qc is clearly bent downwards.

4.2. THE FACILITATION PHENOMENON

From the two curves of subject M in Fig. 4.2, inactive and under facilitation, it can be seen the saving in the intensity of the stimulus for the transition from the inactive to the facilitated state. Thus, the lower the intensity (voltage), the greater the saving in the duration (in μF) of the stimulus. This saving is very small at high intensities and indefinitely large at low intensities.

Facilitation has a very different effect in the two cases here studied. From the curves in Fig. 4.2, it can be inferred that the permeability to this type of summation decreases rapidly when the excitability deficit is lower, i.e., when the brain system shows a more favorable physiological level. If we consider the saving by facilitation in the rheobase voltage, for example, the reduction of the disorder in the M case is

1/3 or slightly more, and one tenth or less in the T case. It should be noted that the values indicated on the previous pages about the facilitated state do not show the entire permeability to nervous summation, since other types of facilitation are still possible (binocular effect, for example). But in electrical excitation tests, it is not easy to use other types of facilitation different from muscular contraction. We shall now refer only to this type of facilitation, leaving the study of the simultaneous action of various types of facilitation for other experiments.

Between the two curves of M (for inactive state and for the facilitated state by maximum muscular effort) in Fig. 4.2, there would be a series of curves corresponding to different degrees of facilitation (different degrees of muscular effort), which would represent other excitability states of the brain system or, more specifically, of the visual system.

A very interesting issue is to know how excitability increases as the facilitating stimulus increases. This issue is of fundamental importance in order to know the action of nervous influx on the activity of the centers. Since facilitation can be equated to the action of the nervous mass destroyed by the lesion, the functional action of that nervous mass could thus be determined indirectly.

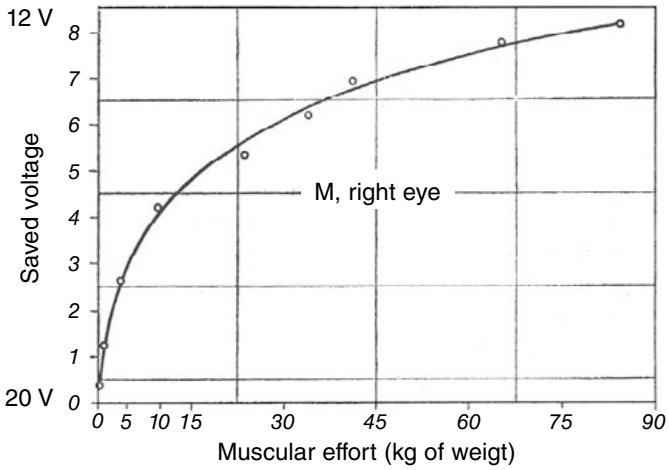

Figure 4.4. Voltage saved as a function of muscular effort measured in kg of weight held by patient M. Test on the right eye for the transition from the electrical excitability curve of the inactive state to that of the facilitated state, for a fixed stimulus duration (17 μF).

To this end, the voltage saved is determined as a function of the muscular effort made by the subject when changing from the inactive state curve of Fig. 4.2 to that of facilitation by maximum muscular effort. The effort made by the subject can be measured quite accurately by having the subject hold known weights. In the standing position, the subject holds a sturdy handle in each hand from which weights of 5 and 10 kg are suspended until a total of about 80 kg is reached, an extremely large amount although necessary for maximum facilitation. The weights in both hands must be

properly balanced to ensure that the effort consists only of the action of holding the weights. In addition, care is taken to maintain regularity in all weight tests, the weights always held at the same height. Under these conditions it is not difficult to perform electrical measurements of the retina with a good degree of precision in order to obtain usable results such as those indicated in the curve of Fig. 4.4, which shows the voltage saved as a function of muscular effort measured in kg of weight held. In this experiment, a slightly higher voltage than that of the rheobase was taken as the starting point, using the discharge of a capacitor of about 17 microfarads (fixed stimulus duration). Analogously, for a constant stimulus intensity (voltage), the time saved in the duration (capacitance) of the stimulus could be determined as a function of muscular effort, when passing from one curve to the other in Fig. 4.2.

It can be seen in Fig. 4.4 that there is a rapid growth in saved voltage for very small muscular efforts, and a slower growth in saved voltage as the muscular effort is intensified. It is important to point out that relatively very small muscular efforts in comparison with the total effort that can be made, produce a considerable economy in the voltage, that is, an increase of excitability (or sensitivity) to stimuli. This explains that in patient M, small muscular contractions or other muscular actions in his ordinary life greatly facilitate his sensory activity.

Thus, by the muscular action exerted in different positions or activities of the body, the voltage corresponding to a fixed capacitance can show an important reduction in the stimulation of the retina, as can be seen in the results shown in Table 4.3. We find that the greater the muscular innervation used, the greater the increase in excitability.

Table 4.3. Voltage required in electrical stimulation of the retina, for a given capacitance, in the right eye of subject M in different positions and activities.

Subject M	Voltage (V)
Lying down	19
Standing	18.4
Walking briskly	16.2
Crouching down with incomplete leg flexion	15-14.5

Coming back to the curve of the mentioned experiment (see Fig. 4.4), if the logarithm of the weights is taken, the points fit well a straight line, as can be seen in Fig. 4.5. It can then be stated that the voltage saved (i.e., the increase in excitability or the increase in the physiological level) is proportional to the logarithm of the muscular effort applied. The nervous energy provided to the cerebral cortex by the proprioceptive receptors of the muscles modifies the state of excitability by influencing the nervous element that determines the elementary response of the visual system (minimal sensation of luminosity evoked by electrical stimulation). This law on the action of facilitation is of great importance for brain dynamics, because if the nervous influx of facilitation can be equated to the influx of nervous centers, the latter could be measured indirectly. Therefore, the level of nervous activity might be determined

as a function of the amount of nervous mass. But these are general issues that we shall address later. For the time being, it suffices to mention that this law on the action of facilitation will be also found in the case of other receptors (tactile, auditory, etc.), and also for other types of facilitation different from muscular effort.

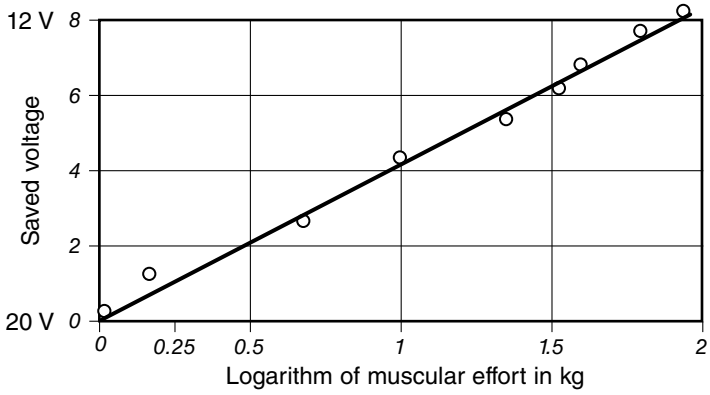

Figure 4.5. The same experiment as in Fig. 4.4 but taking the logarithm of the weights held by subject M. The voltage saved (increase in excitability) grows proportionally to the logarithm of muscular effort.

The facilitation action we have now studied does not refer to the synchronization experiment exposed when dealing with the dynamic analysis, since now the sensory response is always the same (minimum sensation of luminosity). Therefore, in this experiment we study the ability of facilitation to reduce the excitability threshold, that is, to increase the sensitivity of the system. It is clear that this entails a general action on the entire visual system, and therefore the synchronization of different abnormally asynchronous sensory functions, which become synchronized by different increase in their sensitivities. But all this corresponds to another type of experiments that we shall deal with later.

We have already indicated that facilitation is not an exclusive effect of muscular effort; similarly, any other type of nervous stimulation produces summation effect. Auditory or tactile stimuli can enhance visual activity, although no matter how intense such stimuli are, their effects are still far below the effect of a strong muscular contraction. Examples of summation effects by intersensory facilitation for the visual receptor in the M case, are shown in Table 4.4.

Table 4.4. Reduction of the voltage required in electrical stimulation of the retina in the right eye of subject M, by means of different intersensory facilitations.

Whistling strongly next to his ear: from 19 V to 17-16.5 V
Weight of 20 kg held by hand: from 19 V to 15 V
Pressing down on his hand until causing pain: from 19 V to 14 V and even less

Finally, we make some considerations about the characteristics of the facilitation phenomenon. Facilitation increases excitability, lowering the Hoorweg curve (Hoorweg 1892) in which both rheobase and chronaxie diminish, modifying the curvature of the curve and coming closer to the normal curve. The transition from the inactive state of excitability to the facilitated state is carried out in the experiment in the proximity of the rheobasic level, and follows a logarithmic relation (saved voltage proportional to the logarithm of the applied muscular effort). Facilitation exerts a central action whereby the fundamental disorder (slowness or excitability deficit and asynchrony) is reduced from the root. The excitability deficit has already been seen in the Hoorweg curve, and more details will be given when studying iteration. The reduction of asynchrony, which we will deal with later, is the result of a decrease in the dissociated threshold values. As facilitation increases these threshold values tend to become more equalized. Synchronization can then be represented by a curve similar to that of the transit from the inactive state to the facilitated state, studied in this section.

From a strictly physiological point of view, facilitation corresponds to an action of nervous *subordination* by means of the influx of peripheral excitations on nervous centers. This action partly replaces the loss of action of nervous centers on nervous regions that are dynamically subordinated to them. Thus, the exclusion of some nervous centers produces a decrease in excitability, i.e., loss of *central subordination* as was found by Lapicque M. (1923) in laboratory experiments. Facilitation is thus a reciprocal effect, i.e., external influx on the centers. These phenomena are studied in humans for the first time, without any precedent as far as we know.

4.3. ITERATIVE EXCITABILITY OR LATENT ADDITION

The fundamental disorder, with its considerable deficit of excitability, gives way to nervous summations by facilitation (indirect summation) and by iteration (direct summation). Iteration consists of stimulation by rhythmically repeated stimuli with the peculiarity that a single stimulus produces no effect but a series of stimuli does. The parameters involved in this type of stimulation are: intensity of the stimulus, i; duration of a single stimulus, t; time interval between two stimuli, ε; and number of stimuli, n; the intensity being a function of t, ε and n, that is, $i = f(t, \varepsilon, n)$, according to Lapicque (1925). This author established two important relationships, the intensity-number relationship (*law of numbers*) and the intensity-interval relationship (*law of intervals*), for given values of the other two variables in each relationship.

As for the technique to determine in our cases the laws of iterative excitability, we use the Lapicque iteration cylinder (Lapicque 1925), which we made build for this purpose. The cylinder consists of a roller that when rotating activates a system of tabs that produce an automatic charge and discharge of the capacitors, with a widely adjustable rhythm in which the time interval and the number of stimuli can be set. The intensity and duration of the stimulus are regulated according to the usual procedure of varying voltage and capacitance of the capacitors, as for the Hoorweg curve. The iterative cylinder is connected to or disconnected from the capacitor circuit depending

on the needs of the experiments, allowing one set-up for single stimulation and another for iterative stimulation.

Firstly, it should be noted that the normal human retina responds to a single excitation without showing the slightest tendency to latent addition or iteration. However, in the two patients we have studied, the iteration is extremely pronounced, and to a different degree depending on the intensity of their lesions. It happens that a non-iterative organ becomes, due to the brain lesion, an iterative organ, although not exclusively since it also responds to a single excitation. The basis of the iteration lies in the sum of residuals of successive stimuli which fade away more slowly as the iteration capability of the organ increases. The accumulation of residuals thus increases progressively until a new stimulus triggers a response from the organ examined.

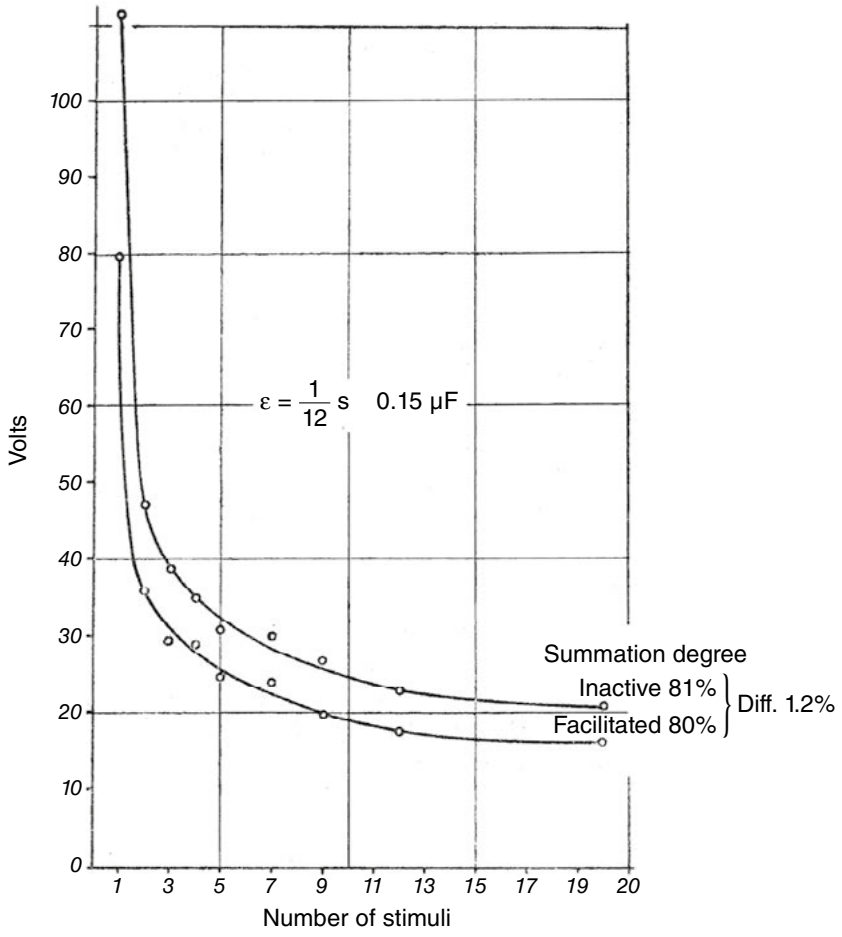

Figure 4.6. Law of numbers in patient M. Upper curve: inactive state. Lower curve: with facilitation by strong muscular effort. Time interval ε = 1/12 seconds, capacitance 0.15 μF. Corresponding degrees of summation and the difference in percentage are indicated.

Summation depends largely on the time of action of the stimulus, as well as other conditions on time interval and number of stimuli. The shorter the duration of the stimulus, in relation to the nature of the organ being examined, the greater the summation. As for the time interval, the smaller it is, the greater the degree of summation, i.e., the greater the difference between the required intensity of a single stimulus and that of a stimulus from a series of them.

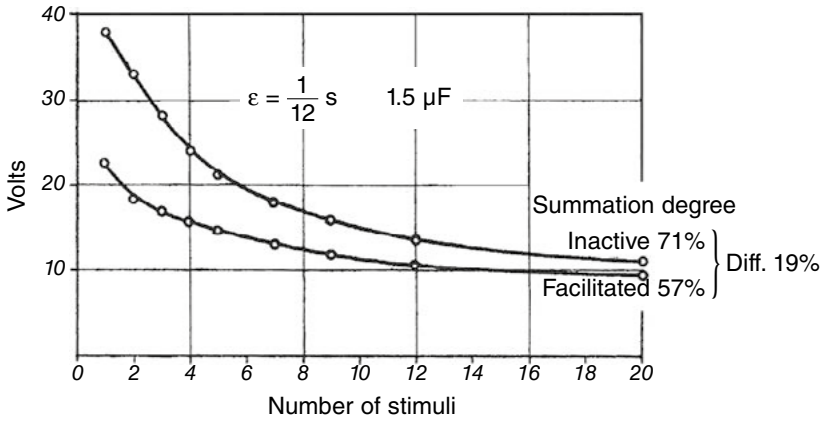

Figure 4.7. Law of numbers in patient M under the same conditions as the previous Fig. 4.6, but modifying the capacitance, now ten times greater. Note the different slope of the two curves, inactive and facilitated, and the degree of the corresponding summation.

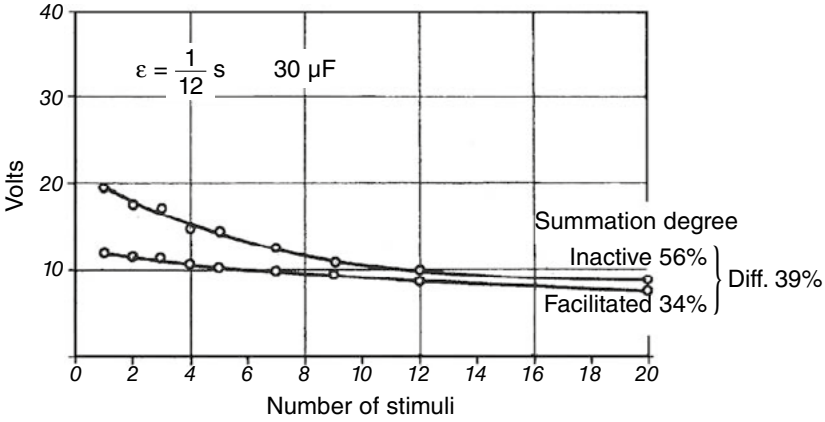

Figure 4.8. Law of numbers in patient M using an extremely large capacitance. The difference in iteration between the two curves is maximum.

To find the law of numbers in patient M, the intensity (voltage) of the stimulation is determined as a function of the number of stimuli used, for constant values of stimulus duration (capacitance of the capacitor) and time interval between two

stimuli (1/12 second). Figure 4.6 shows the results of the iteration in patient M, for the inactive and the facilitated state. Note the decrease in voltage required as a function of number of stimuli. Using a very small action time (very small capacitance), a high degree of summation is obtained in this case. *Degree of summation* is the difference between the voltage of a single stimulus that produces sensation and the lowest voltage when a series of stimuli produces sensation, expressed as a percentage. Using stimuli whose duration corresponds to a capacitance of 0.15 μF, very high degrees of summation are obtained, both without and with facilitation; the respective values obtained of 81% and 80% indicate that the system is strongly iterative. The proportional difference of summation between both curves is insignificant, 1.2 %. This is because we are dealing with a capacitance for which the two Hoorweg curves, inactive and facilitated, are almost together. This small difference in iteration between the inactive and facilitated states is considerably enlarged by using higher capacitances, 1.5 μF and 30 μF, as shown in Figs. 4.6 and 4.7 respectively. As the stimulus duration increases, the degree of iteration decreases, but the difference in iteration between the inactive state and the facilitated state increases (in accordance with Hoorweg curves). In short, it can be said that iteration is very significant and that facilitation reduces it, especially in the range of excitation where facilitation acts most strongly. Note the different slope of the iteration curves without facilitation and with it in Figs. 4.7 and 4.8, especially in Fig. 4.8 where the difference in iteration between the two curves is maximum.

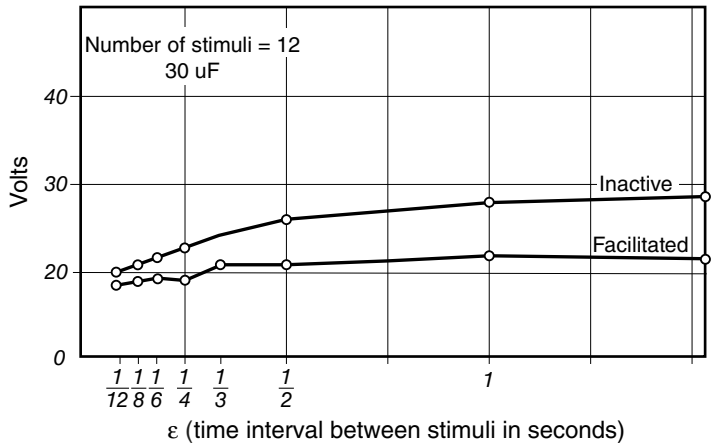

Figure 4.9. Law of intervals in patient M for the inactive and the facilitated state, using a capacitance of 30 μF and 12 stimuli in each determination. The curve of the facilitated state is much more horizontal than that of the inactive state.

Here we also find two different types of degree of summation, greater degree for the inactive state than for the one facilitated by muscular effort. As with strength-duration curves, facilitation reduces the excitability deficit in the iteration

curves, thus making the system less iterative. But since facilitation is not able to completely restore the function to normality, some iteration capability remains.

Summation time is the product of the number of stimuli used by the time interval between them (ε), when the voltage is no longer reduced. The summation time for patient M is approximately 1.5 seconds in the inactive state, and perhaps somewhat less in the facilitated state. As is known, this is the time in which the latent addition of stimuli produces maximum voltage saving.

The other important relationship of iterative excitability is the law of intervals, in which stimulus intensity is a function of the ε interval, with the number of stimuli and their duration being constant. We use 12 stimuli, and a time interval between two stimuli from 1/12 second to more than one second. For the law of intervals, it is now sufficient to indicate the results shown in Fig. 4.9, for which a capacitance of 30 μF was used, as in the last case of the law of numbers. It can be seen how the voltage

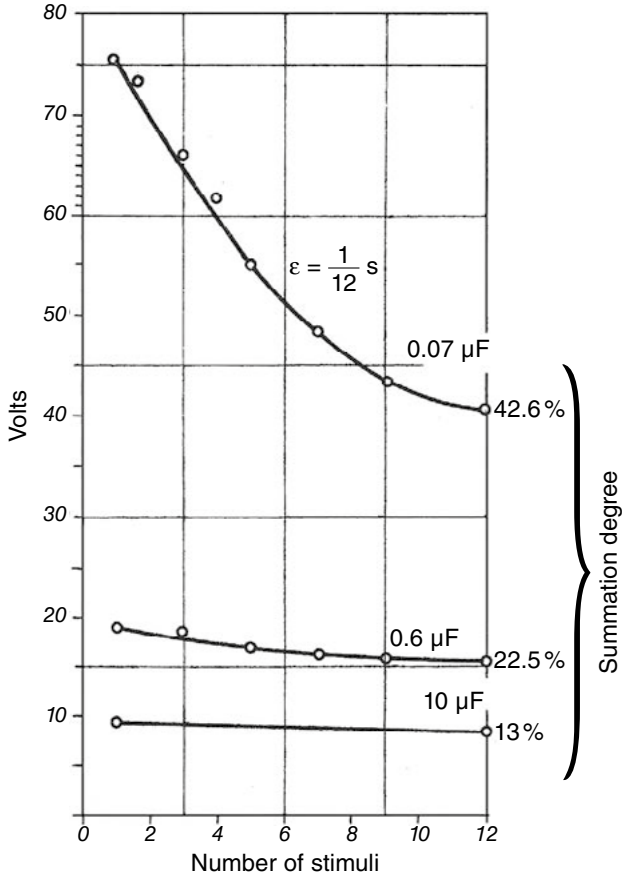

Figure 4.10. Law of numbers in patient T for three capacitance values (stimulus duration). The value of the time interval ε is the same as in the M case. Note the slope of the curves, and the corresponding summation degree values.

decreases as the time interval gets shorter. The more iterative a nervous organ is, the steeper the slope of the curve and the greater the voltage economy achieved with very short time intervals. Fig. 4.8 shows rather horizontal curves due to the long duration of the stimulus used. The same considerations made about Fig. 4.8 (third example of the law of numbers) are valid now since the phenomena are the same under another aspect; with facilitation, the curve is more horizontal than in the inactive state. By using smaller capacitances, results analogous to those shown for the law of numbers are obtained.

As for patient T, it is shown to have a not inconsiderable iteration capability, although much less pronounced than in patient M, as expected from the different behavior of their Hoorweg curves. Iteration in the T case is very easy to study, and can be determined with much less difficulty and more accurately than the phenomenon of facilitation by muscular effort in this subject. This is because iteration lowers the excitation threshold much more than facilitation by maximum muscular effort, especially if very short stimuli are used in iteration.

Figure 4.10 shows the behavior of the law of numbers in the T case for three different values of stimulus duration (in μF), and an interval of 1/12 second as in the M case. Under these conditions, the degree of summation varies from 42.6 % for a very small capacitance, to 13 % for a very large capacitance in relation to the characteristics of the excitation time in subject T.

A much lower degree of summation is obtained than in patient M, even when M is under facilitation; thus, T is much less iterative. However, whereas in the M case, using an interval of 1/48 second in the law of numbers, no greater summation capability is achieved, in the T case such capability clearly increases, as shown in Table 4.5.

Table 4.5. Iteration capability in patient T.

Capacitance (μF)	**Voltage needed with a single stimulus**	**Voltage needed with unlimited number of stimuli with interval $\varepsilon = 1/12$ s**	**Voltage needed with indefinite number of stimuli with interval $\varepsilon = 1/48$ s**
10	9.6 V	8.3 V	7.6 V
2	15 V	12 V	9 V
0.6	19.5 V	14 V	11 V
0.07	75 V	39 V	23 V

Thus, using a capacitance of 0.07 μF, the degree of summation can be increased from 42.6 % using a time interval of 1/12 s (Fig. 4.10) to 66 % using a time interval of 1/48 s, and analogously for the other capacitance values shown in Table 4.5. But even so, the degree of summation remains much lower than that of M even when M is under facilitation by maximum muscular effort.

Regarding the law of intervals, subject T presents a behavior similar to that shown in the law of numbers, so we shall not insist on it. As for the summation time in subject T, it is a little less than one second.

In summary, if we try to characterize as a whole the emergence of iterative excitability of an organ such as the retina, which does not have it at all in a normal situation, we can say that the greater the deficit in nervous excitability (the slower the system, i.e., the greater its chronaxie), the more pronounced the capability for iteration. Considering three main types of excitability, two in subject M (in the inactive state and under facilitation by maximal muscle effort) and that of subject T, different iteration capabilities are obtained corresponding to the three respective strength-duration excitability curves for a single stimulus (Fig. 4.2).

Comparing the effect of facilitation with that of iteration, the latter lowers the threshold of the inactive state somewhat more than facilitation by maximum muscular contraction. As said, facilitation, by its central summation action, reduces the degree of summation by iteration since it makes the system to react faster. Once facilitation by muscular effort has acted, iteration lowers the threshold voltage very little, and vice versa, once a series of stimuli have acted up to reach the lower voltage limit, facilitation lowers the threshold weakly. Both types of summation can act simultaneously but the final effect is not the arithmetic sum of the action of the two types separately but much less, since facilitation modifies iteration capability.

The permeability to these two types of summation, iteration and facilitation, the former being direct and the latter indirect, depends entirely on the slowness in the reaction of the brain system, as already mentioned, although their respective mechanisms are very different. As for iteration, we must point out that its occurrence is a consequence of the loss of reaction speed in nervous organs or in any other tissue. Striated muscle tissue, which normally does not show any iteration, when it becomes slower due to incomplete curare intoxication (see Fig. 4.3), offers a very important iteration.

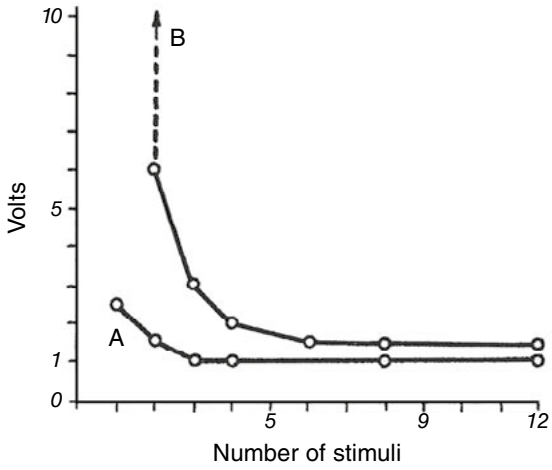

Figure 4.11. Law of numbers in the hemispherectomized frog (A) and in the spinal frog (B) for the reflex of crossed shortening of the lower extremity. Excitation of the open sciatic nerve using rhythmic capacitor discharges. Constant capacitance of the order of the chronaxie capacitance of the sensitive fibers of the sciatic nerve. Time interval between stimulus: 1/12 s. (Schriever 1933).

The slower the reaction of an organ, the slower the disappearance of the electrical modification produced by a stimulus. This results in the accumulation of the residual action of closely spaced stimuli, an accumulation that grows up to a certain limit.

We know that in our two cases, the excitability deficit must be put in relation to the loss of brain mass, and in this respect, the experiments of Schriever (1933) (see Figs. 4.11 and 4.12) are very illustrative. The crossed reflex of shortening of the frog's lower limb, which normally responds to a single stimulus and does not present any iteration, becomes an iterative reflex due to the removal of the central nervous mass. The more extensive the removal of nervous centers, the more pronounced the iterative capability. Thus, if only the cerebral hemispheres are removed, it is still possible to trigger the reflex with a single excitation, as seen in curve A of the law of numbers, although iteration allows it to be obtained with lower voltage. However, if there is an extraction just below the midbrain, i.e., if a spinal frog is prepared, the reflex becomes totally iterative, and the B curves of the laws of numbers and intervals not only have steeper slopes, but also show that a single excitation is not capable by itself of triggering the reflex, no matter how much the stimulation voltage increases.

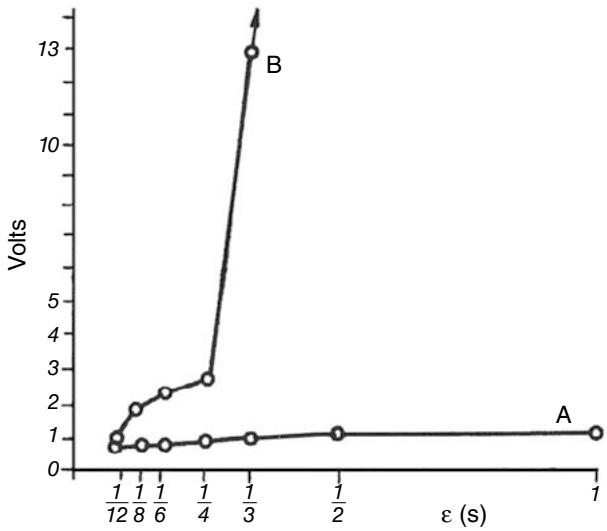

Figure 4.12. Law of intervals in the frog in accordance with the indications in previous Fig. 4.11. Constant number of stimuli equal to 12. (Schriever 1933).

In subjects M and T two different degrees of iteration are also obtained in accordance with the amount of brain mass destroyed by the lesion. In addition, another intermediate type of curve is obtained due to the facilitation action in subject M, showing how facilitation can replace the action of the centers. This latter effect offers a wide field for experimentation, reinforcing the dynamic thesis about the nervous system.

4.4. CONCLUSIONS ON ELECTRICAL EXCITABILITY

In general, it can be said that we are dealing with systems with different excitability degree, i.e., different physiological activity depending both on the amount of nervous mass destroyed by the brain injury and on the action of facilitation. In relation to the physiological level, the results of electrical stimulation of the retina can be grouped as shown in Table 4.6.

In such systems, the excitability deficit caused by the loss of brain mass is partly counteracted by types of summation such as latent addition and facilitation, not at all present in a normal subject, and which are generated by the slowness of nervous reaction. The greater the excitability deficit, the more pronounced the iteration capability. Iteration acts directly by producing a special mode of stimulation that allows saving stimulus intensity, and facilitation acts indirectly by increasing the excitability of the nervous centers, i.e., by increasing the reaction speed and making efficient the sub-rheobase stimulus of the inactive state. At the same time, by increasing the speed of the system, the capacity for latent addition is reduced. There is therefore a margin of permeability to summation which can be filled either by iteration (direct addition of stimuli) or by facilitation (indirect summation on the nervous centers).

Table 4.6. Results of electrical stimulation of the retina in patients M and T.

Patient M (severe brain lesion)		**Patient T (mild brain lesion)**
Inactive	**Facilitated by maximum muscular effort**	**Inactive**
Very slow reaction system. Rheobase & chronaxie ~ 10 times larger than normal.	Faster system. Rheobase & chronaxie ~ 4 times larger than normal.	Even faster system. Rheobase & chronaxie 4 times larger than normal.
Very iterative system. 71 % summation degree using half chronaxie capacitance.	Less iterative system. 57 % summation degree using half chronaxie capacitance of the inactive state.	Much less iterative system. 22.5 % summation degree using half chronaxie capacitance.
System very permeable to facilitation, saves 1/3 of rheobase voltage.	Does not admit more facilitation by muscular effort, but it does of any other type.	System very little permeable to facilitation, saves ~ 1/10 of rheobase voltage by muscular effort and movement.

As for facilitation, in the case of visual excitability, there is a reinforcement of the stimulus on the retina by means of another extra visual stimulus, thus, there is an indirect summation of both stimuli through the nervous centers. If the margin of permeability to summation is almost filled with iteration, then little action is left for facilitation, and vice versa, since the system can be saturated by either type of sum-

mation. Therefore, their effects can be considered similar, although the mechanism is not the same.

In conclusion, it can be said that if brain excitability decreases due to loss of brain mass, this deficit is partially overcome by the forms of summation we have discussed. The central mass normally acts by reinforcing or increasing by summation the excitability of the various receptors and sensory systems, that is, by exerting an action of central subordination over them, whereas in the case of summation by facilitation and iteration, it is an inverse case, of peripheral subordination over the nervous centers, i.e., an action of stimuli over the centers. Therefore, for all cases it could be said that excitability depends on nervous summation. We have already seen in the curves about facilitation by muscular effort (Figs. 4.4 and 4.5) how excitability changes by the action of summation. If such a facilitation is similar to the action of summation normally exerted by the nervous centers, then the dependence of excitability on the logarithm of muscular effort (Fig. 4.5) can be also extended to the action exerted by the amount of brain mass. This means stating a law of primary importance for the dynamics of the brain, as it will be seen in subsequent work.

5. Light excitability

5.1. EXCITATION WITH ADEQUATE STIMULUS

In the same way we have studied excitability as a function of time using electrical stimulation, we can now do so by means of light stimulation. For this purpose, we use a white test 1 cm in diameter, diversely illuminated, and a simple tachistoscopic shutter mechanism that regulates the exposure time of the stimulus. Due to various circumstances, the results cannot be as accurate as those obtained in electrical stimulation, but they are good enough to determine the state of excitability in the two patients we are studying.

Fig. 5.1 shows the curves of the minimum light excitation necessary to produce sensation in central vision in subject M in his two extreme states, inactive and under facilitation by maximum muscular effort, and in subject T, together with the curve for a normal subject used as reference. For easier representation of these curves, the luminous intensity values are expressed logarithmically. Several properties can be appreciated in the different curves: different reaction speed according to their concavity, rheobase thresholds that place the curves at different heights, and different useful time (duration of the stimulus of minimum intensity that produces sensation).

Due to the imprecise determination of the rheobase threshold by its normal lability, which is even more pronounced in these pathological cases, and due to the simplicity of the measuring instruments, we can only expect approximate values, yet good enough to distinguish the types of excitability of the cases we are studying. Thus, the useful time for the right eye of subject M in the inactive state can be assessed in approximately six seconds, i.e., the lowest intensity light stimulus that can be detected by the subject requires action for about six seconds. For the same eye but applying facilitation by maximum muscular effort, besides being possible to reduce the intensity of the stimulus to about half, a useful time of about three or four seconds is sufficient.

In the case of T, the rheobase threshold and the useful time are lower, the latter being about 1.6 second. For the normal subject the useful time is about half a

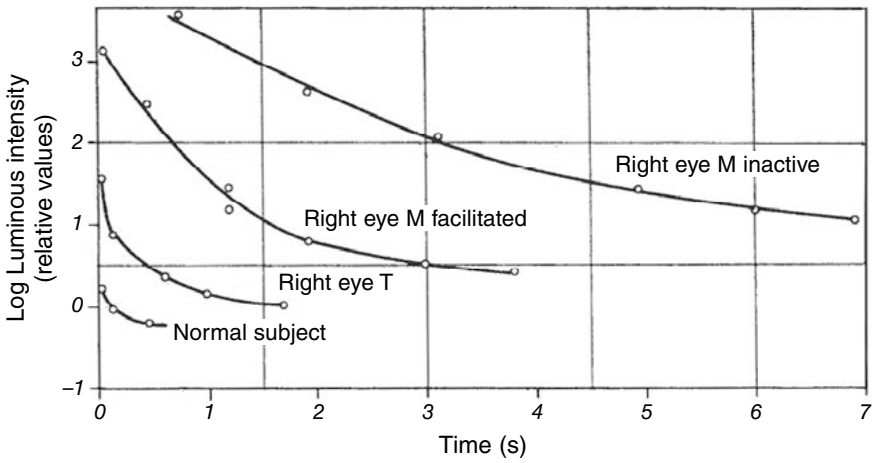

Figure 5.1. Light excitability curves in central vision of the right eye of: M in the inactive state, M under facilitation by maximum muscular effort, and T in the inactive state; compared to the lowest curve for a normal subject.

second, and with a much lower intensity threshold than in the previous cases. These different useful time values show the corresponding reaction speed for each case. The values keep approximately the same proportions as in the chronaxie capacitances in electrical excitation using capacitors. In the curves of patient M, a considerable saving in time and intensity is observed in the facilitated state with respect to the inactive state, as in the tests of electrical excitation.

It should be noted that the duration of the stimulus is usually several times longer than that of electrical stimulation. This increase is attributable to the intermediate photochemical process that develops to elicit nervous excitation, hence it is a more indirect stimulation than electrical stimulation. As with electrical stimulation, care must be taken to avoid iteration of stimuli, facilitation and fatigue, in order to find the true state as regards light excitability in these subjects, especially in subject M in the inactive state. The importance of facilitation has already been seen in Fig. 5.1. As for fatigue, it easily arises as soon as experiments are prolonged for a certain period of time, leading to an increase in intensity and time values. As for iteration, the same relationships exist as in electrical stimulation, although in this type of light stimulation they are difficult to evaluate. It is observed that single isolated stimuli that do not produce sensation can trigger it by reiteration of the same stimulus. The difference in intensity required between a single stimulus and several is greater the shorter the duration of the stimulus and the shorter the time interval between them.

The sensation of luminosity develops along different phases whose duration depends on the speed of nervous reaction. In this evolution we distinguish three stages: latency or time elapsed from the initiation of the stimulus until the emergence of sensation, the stage of establishment of the sensation during which it remains stable after having gone through certain fluctuations and, finally, the stage of progressive fading until the sensation disappears. When nervous processes are very slow

due to a deficit of excitability, as in the M case, this evolution of sensation occurs with remarkable slowness; latency is very long as well as the fading phase which can reach up to four seconds, especially for weak stimuli.

This slowness in reaction makes both subject T and subject M not perceive rapid medium-intensity light stimuli as light sparks, even when subject M is under facilitation. This subject in the inactive state does not detect even intense electric light from a distance of a couple of meters and duration less than half a second. The reaction is so slow that the duration of the light stimulus can easily be measured with a stopwatch for most of the excitability curve (see Fig. 5.1), and equally for any other type of test with colors, shapes, visual image orientation, etc.

In the genesis of sensation, both stimulus and receptor intervene. In our subjects, excitation with a single stimulus and iterative excitation must be considered, and in both cases with extremely increased time and intensity, as well as savings in iterative excitation. The receptor conditions the sensation according to the degree of adaptation to the stimulus, and according to the sensitivity of the region of the receptor (foveal or peripheral vision). These circumstances are also present in normal individuals but are extremely pronounced in our cases. There are also special circumstances such as phenomena of summation by facilitation. These can be of an extra visual type, such as muscular effort, acoustic stimulation, tactile stimulation, etc., or of a visual type, such as the effect of one eye on the other eye that is stimulated, binocular vision, light on the eye being examined, etc. In short, actions that modify the central state.

All these circumstances must be carefully considered in order to properly establish the various tests, and to maintain uniformity and constancy in the course of them. Since many factors intervene in the result of the sensation in our subjects, it is possible to perform a very varied experimental analysis which shows the complexity of nervous activity governing sensory function.

To highlight the conditions that intervene in visual sensation (analogous for other receptors) we can group them as shown in Table 5.1.

Table 5.1. Stimulus and receptor conditions in visual sensation.

Visual sensation		
Stimulus	**Receptor**	
	Own state	**Summations (central state)**
Single excitation: Intensity and time		Extra visual facilitation: muscular effort, etc.
	Region of the visual field	
Iterative excitation: Time interval, etc.	Light adaptation	Bi effect: binocularity, light over the eye not being examined.
		Light on the eye being examined.

For the curves in Fig. 5.1, relatively simple conditions have been considered, which are the ones we shall use in general. These are: vision only in the right eye with the other eye closed, and considering in patient M a facilitated state by maximum muscular contraction, or an inactive state, although sitting in front of the measuring instruments always implies a small facilitation that can be considered negligible. In addition, unless otherwise indicated, the tests refer to central or macular vision and to the state of adaptation to darkness, i.e., of maximum sensitivity. The values thus obtained differ significantly from those obtained in binocular vision due to the facilitating effect of one eye on the other. In the latter case, the rheobase threshold can be lowered almost as much as in monocular vision combined with facilitation by maximum muscular effort. In order to obtain a binocular summation effect, it is not necessary that both eyes receive the same stimulus (which is the case of maximum summation effect), but a rather similar effect is obtained when one eye receives the stimulus and the other eye (impeded by a screen to see the stimulus) receives a certain amount of light.

These types of conditions will only be systematically analyzed when dealing with inverted vision phenomena, which are very appropriate for all kinds of determinations. For other phenomena, we shall follow the conditions already exposed (vision in the right eye of subject M, central vision, adapted to a certain darkness, inactive state, or under facilitation by maximum muscular effort). A similar procedure will be followed for subject T, who will be useful mainly as a complement in the experiments.

5.2. LIGHT AND DARK ADAPTATION

The threshold for perceiving a sensation of light depends on the previous state of adaptation of the visual receptor, i.e., on its degree of sensitivity or capacity to adapt to changes in illumination. In patients M, T and many others with occipital lesions that we have studied, adaptation to darkness is markedly impaired. This alteration consists mainly of a great slowness in adaptation and a decrease in the degree of adaptation. Here we shall only expose the tests of patient M, in whom the alteration in adaptation is maximum. This disorder must be related to the deficit of excitability of the centers, i.e., to the slowness in nervous reaction, and not to an alteration of peripheral photochemical mechanisms which must be admitted as intact.

The procedure for studying the adaptation is as follows. First, intense dazzle with a white light lamp of 250 foot-candles for two minutes, and then determination with the photometer of the minimum intensities of light that can be perceived along half an hour during which the subject remains in darkness and adapts to the minimum intensities. A normal individual under the same experimental conditions serves as a comparison. The measurements are made at first every one or two minutes due to the initial speed of adaptation, and after ten minutes, they are made more separated in time because the change is slower. In these determinations, although central vision is used, the visual field is not limited exclusively to the macular region but extends slightly to the periphery due to the aperture of the photometer eyepiece.

These circumstances are not indifferent since the adaptation curve depends on several factors such as the wavelength of the light, the region and size of the visual field examined, the duration of the dazzle, etc.

Figure 5.2 shows the dark adaptation curves obtained in subject M in the inactive state and in a normal subject, according to the experimental conditions mentioned above. Between both curves there would be another one corresponding to the facilitated state which has not been drawn for the sake of simplicity. It is observed that the process of adaptation to darkness lasts a long time, and although we have taken the values up to thirty minutes, the process is prolonged for about forty-five minutes although by then the decline is almost insignificant.

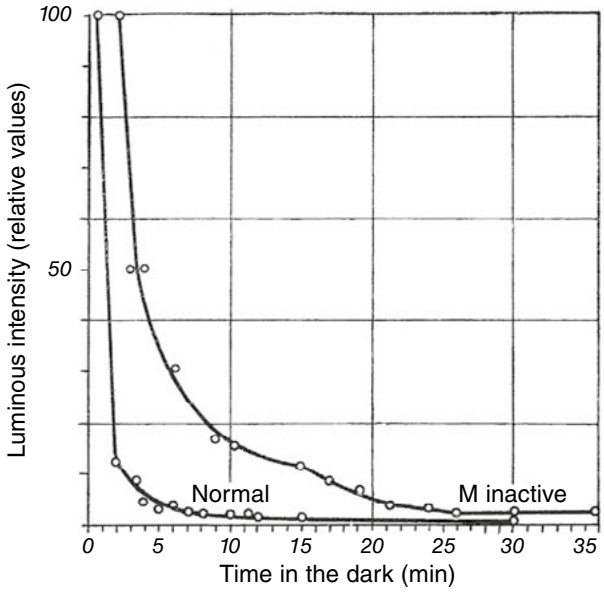

Figure 5.2. Dark adaptation of the right eye of subject M in the inactive state, and of a normal subject, both dazzled under the same conditions. In the case of M, observe the much slower adaptation, and the change of behavior after fifteen minutes.

The process of dark adaptation in the normal subject takes place very quickly in the first two or three minutes, and after five minutes the decrease of the threshold is very considerable. By contrast, adaptation is much slower in subject M, and he takes about fifteen minutes to reach a threshold like that of the normal subject after two minutes. The elevation of the curve in the M case over that of the normal subject, and the smaller curvature, indicate a decrease in the degree and speed of adaptation. The pathological curve also shows a small change in behavior after about 15 minutes, corresponding to a small variation in the adaptation speed. This change is not seen in the normal curve in this type of representation but it can be seen by plotting the logarithm of the intensity, as we shall see below.

Another way of expressing dark adaptation is by displaying sensitivity (inverse of luminous intensity threshold) as a function of time in the dark. In this way, the curves of Fig. 5.2 become those of Fig. 5.3 showing how sensitivity increases.

Sensitivity increases rapidly in the normal subject and very slowly in subject M. The pathological curve has a smaller slope, which means a slower evolution of the nervous process. The final value reached by the curves indicates the maximum sensitivity. In the M case, this value (about a quarter of the normal value) is only approximate due to the poor detection of the photometer in very low light.

A third way of expressing dark adaptation measurements is shown in Fig. 5.4, where the intensity values of Fig. 5.2 are represented logarithmically. This makes it easy to distinguish two phases with different rates of adaptation in both the pathological and the normal curve.

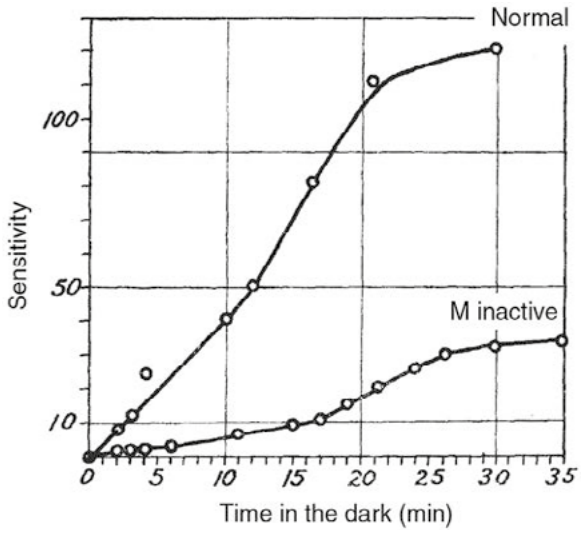

Figure 5.3. Luminous sensitivity (inverse of luminous threshold) in dark adaptation, for the right eye of a normal subject and of subject M in an inactive state. Note the different slop of the curves, and the different values at large times.

In Fig. 5.2, only a small change in the evolution of the curve is seen for the M case, whereas in the logarithmic representation (Fig. 5.4), each of the two curves clearly show two regions with different adaptation rates. In the normal subject, there is a rapid adaptation phase during the first ten minutes and then a slower adaptation phase. In the M case, the change in behavior occurs later, around seventeen minutes, and adaptation in the second phase is faster than in the normal subject.

In the duplicity theory of vision, of cones and rods, this change in the curves is explained as the result of two systems that react differently. The first part of the curve corresponds to cones, which adapt quickly, and this part ends promptly; the second part corresponds to rods, which react slowly and act in low light. When only central

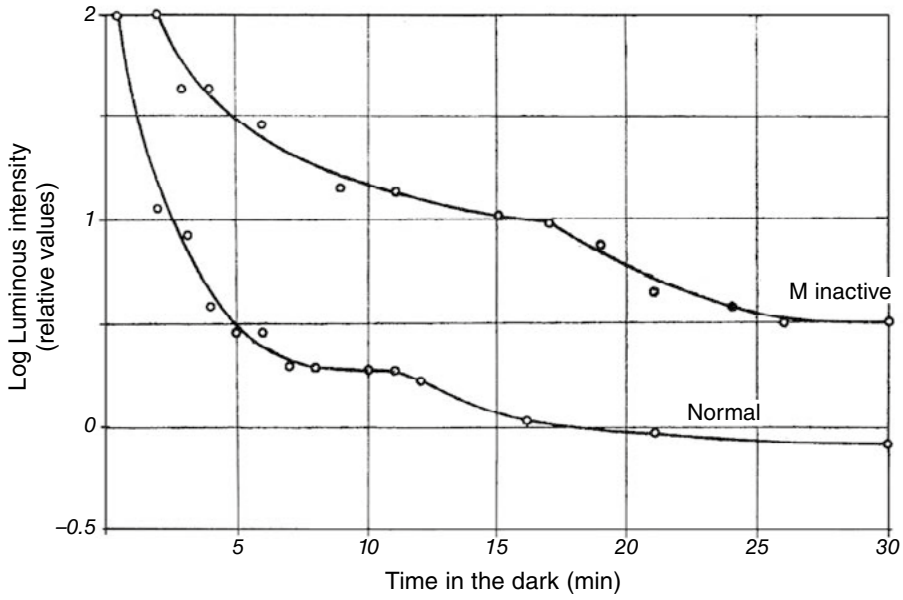

Figure 5.4. Dark adaptation of the right eye of subject M in the inactive state, and of a normal subject, as in Fig. 5.2 but taking the logarithm of luminous intensity. The adaptation process is divided into two phases with different adaptation rates.

vision is used, and therefore only cones are involved, dark adaptation is limited to the first part of the curve. In our test, the visual field is very reduced, and therefore few elements of the periphery, rods, come into play, this being the cause that the transition from the first part of the curve to the next one is not too pronounced.

The slowness of adaptation in subject M must be explained by the deficit of nervous excitability of the centers, and not by the photochemical mechanism of the retina.

Due to their slow adaptation, patients M and T have difficulties in ordinary life to adapt to sudden changes in luminosity. Such a delay makes them appear to be affected by night blindness (nyctalopia), that is, difficulty in adapting to twilight vision, but the curves show that the adaptation is rather good although with much less speed than in the normal individual, so it is a moderate nyctalopia. We have also observed this adaptation deficit in many patients with brain lesions in the visual domain. They show difficulty in the transition from strong to weak illumination, but they are not blind in the dark in a lasting way as is the case in genuine nyctalopia. However, it should be noted that the two subjects we are studying as well as others with an occipital lesion (or near that region) and visual alterations of variable intensity, present, in addition to this slowness and some deficit in the degree of dark adaptation, the peculiarity of feeling better under weak illumination than under strong illumination because of the easy fatigue they experience under the latter. Many of these brain-injured people tend to avoid sunlight because it causes them eye fatigue

too soon, and instead they feel very well during the twilight hours. Thus, they show a certain tendency to day blindness (hemeralopia), i.e., better visibility in dim light than in strong light. In all these cases, such disorders have a brain origin, and are produced by an excitability deficit, a cause not mentioned so far. However, alterations in adaptation to darkness in purely nervous injuries are not new. In fact, Best (1919) and Igresheimer (1918, 1919) report a slow adaptation in some cases of hemianopic defect, and especially, in the research of Kaltwasser (1920) on subjects with occipital injuries, a diminished adaptation is also indicated.

5.3. INTERMITTENT STIMULATION. FLICKER-FUSION FREQUENCY

We shall complete the study of light stimulation by determining the behavior of subject M under intermittent stimulation in order to find the visual critical flicker-fusion frequency. For these tests, we use a rotating disc of adjustable speed, activated by a synchronous electric motor; the speed is indicated by a revolution counter. The disc consists of two equal semicircles, one white, one black, and when it rotates, an alternating sensation of light and darkness is produced. When the speed is increased, a weak oscillation appears corresponding to the flutter point, and when the speed is increased even more, a uniform sensation is established (luminous surface without the least oscillation), which corresponds to the flicker-fusion point.

This critical point depends on the alternating frequency of the phases (white and black) and on the luminous intensity on the disc. The relationship between frequency and luminous intensity is governed by the law of Ferry (1892) and Porter (1902), according to which critical flicker-fusion frequency is proportional to the logarithm of luminous intensity. As in the previous case of dark adaptation, the results depend on the region and size of the stimulated retinal field, and on the nature of the radiation used. The different curves thus obtained are also interpreted according to the duplicity theory of vision, that is, by different action of central cones and peripheral rods.

In the tests performed in subject M and in a normal subject for comparison, white light and a central visual field of 6 degrees in diameter are used. The curves obtained for critical flicker-fusion frequency as a function of illumination intensity are shown in Fig. 5.5. The lower curve corresponds to subject M in the inactive state, the middle curve to M under facilitation, and the upper curve to a normal subject. The ordinate indicates rotation frequency in cycles per second, and the abscissa indicates the luminous intensity on the disk to obtain the critical flicker-fusion point. At this point, the weak oscillation corresponding to the flutter point disappears, and a constant uniform surface appears whose brightness is equivalent to the average brightness of stimulation, according to the law of Talbot (1834). Intermittent stimulation, since it involves time, provides insight into the kinetic properties of the visual system. The two halves of the disc, white and black, correspond respectively to a succession of light stimulus and absence of stimulus, both phases being of equal duration as the semicircles are identical. Therefore, the intermittent stimulus informs us about the degree of persistence of the stimulus, that is, about the speed of visual reaction, which

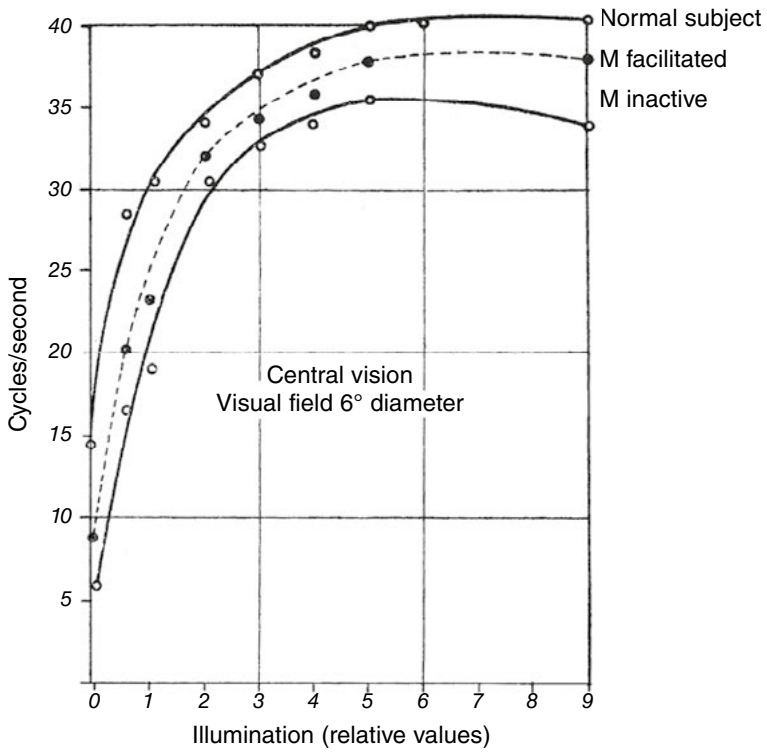

Figure 5.5. Critical flicker-fusion frequency as a function of illumination, for the right eye of subject M in the inactive state, under facilitation by maximum muscular contraction, and for a normal subject. White light and central visual field of 6 degrees in diameter.

constitutes the basis of the flutter point and the critical flicker-fusion point. The two curves of subject M are below the normal curve due to his slow reaction time. Given his alteration in excitability, the evolution process of the sensation of luminosity is very slow, leading to an increase in the persistence of sensation and an easy fusion of intermittent stimuli. For a given value of luminous intensity, the fusion is obtained in subject M with a much lower frequency than in the normal subject; and for a given frequency, subject M needs more luminous intensity than the normal subject since the persistence in M decreases with the intensification of the stimulus. The curve corresponding to the facilitated state lies between the normal curve and the curve of the inactive state. Thus, facilitation either saves luminous intensity or allows for an increase in the critical flicker-fusion frequency because it makes the visual system to react faster, i.e., as excitability increases, the persistence of the sensation decreases.

If in the graphic representation we take the logarithm of the luminous intensity, we obtain an almost straight line which is an expression of the law of Porter (1902): critical flicker-fusion frequency is proportional to the logarithm of the luminous intensity. But here we give the intensity values arithmetically because it is more appropriate to the values found experimentally, since at the bottom of the curve there are

very few values – because of the device used – to obtain logarithmically a straight line with sufficient accuracy.

Let us note that for a given luminous intensity, when subject M reaches the flicker-fusion point, the normal subject sees a fluttering or a rather strong oscillation of luminosity. It should also be noted that intermittent stimulation produces a latent addition of stimuli in M, significantly reducing its excitability deficit, and thus shortening the separation between his curves and that of the normal subject. Thus, if there are only two successive light stimuli, subject M does not perceive any interruption or visual oscillation even if the interval reaches two seconds. If the light from a low intensity electric bulb is suddenly interrupted for one second or less, subject M does not perceive any oscillation of luminosity, even under facilitation, and the same happens to subject T (although to a lesser extent), thus both subjects show a great difference in behavior with respect to the normal subject. They present a great persistence as a consequence of a slow nervous reaction.

Therefore, visual sensitivity to time discrimination of stimuli is evidenced by the flicker-fusion frequency. There is a deficit in the appreciation of short time intervals since consecutive sensations merge easily whereas the normal subject still has a wide margin to perceive the separation between stimuli. The pathological curves of the M case are similar to the curves of a normal case in peripheral regions of the visual field which show a lower flicker-fusion frequency than in central regions.

As it has been mentioned, the alteration in the flicker-fusion frequency is due to an increase in the persistence caused by the excitability deficit. Therefore, it has a central nervous origin, as does the alteration in dark adaptation and also the alteration of the strength-duration curve in light stimulation, and should not be considered as alterations of the photochemical mechanism of the receptor. In this regard, it should be noted that in contrast to the photochemical theory of these phenomena developed by Hecht and Shlaer (1935), other authors are in favor of a purely central nervous mechanism. Thus, according to Brecher (1932), the flutter rhythm perceived as a vibration, and which is independent of intensity, is 18 c/s and has the same value for the different sensory domains (visual, tactile, auditory), depending exclusively on the characteristic refractory period of the nervous centers. In the same direction is the research of Bartley and Bishop (1933) in the rabbit. From the delay of retinal potentials when the eye is intermittently illuminated, these authors laid the foundations for a clearer explanation by involving mainly nervous mechanisms.

With the exposition of all these tests about light excitability (intensity-duration, dark adaptation and critical flicker-fusion frequency) we conclude the study on general behavior under adequate stimulus, and we shall now undertake the study of the various visual functions.

Color vision

6. Alteration of the chromatic spectrum

6.1. COLOR VISION RESEARCH

The color disorder we are dealing with here presents very profound alterations, especially in subject M, on whom we shall focus most of this study; the other subject T being a complement in certain issues.

In addition to color disorder per se, i.e., all that has to do with the important issue of color vision and color blindness, there are other types of disorders such as flat color vision and color agnosia (difficulty in understanding the names of colors), which will be discussed later in other chapters along with disorders of a similar nature.

Since we shall refer directly to the discriminatory activity of brain centers in the face of light, the present study on color function is different from other studies on color vision performed in subjects with retinal abnormalities but with normal nervous centers. Here, the study of color vision is based on the relationships of nervous excitability by means of the dynamic analysis we already know.

Given the complexity of color alteration, certain methodological precautions must be taken since, in addition to the strictly sensory disorder, there are also perceptual disorders and agnosia. The sensory disorder has a very special character, and changes the appearance of colors; thus, blue and yellow are seen as green (*dyschromatopsia*), and in the same way, white and different grays are seen as green (*chromatopsia*). Together with this strictly sensory disorder, there is the spatial color alteration called *flat color* vision in which colors appear as fogs or colored masses that are detached from (or "irradiated"[1] by) colored objects. Moreover, color agnosia hinders the understanding of color names, i.e., the abstract concept of colors. It is therefore imperative to take into account all these alterations in order to properly design tests and experiments. Thus, for example, the examination of subject M

[1] The term 'irradiation' is used to indicate that the perceived localization is widespread and diffuse. Hereafter, the term will not be written in quotation marks.

by means of Stilling's pseudo-isochromatic plates (Stilling 1883) to detect color blindness is not at all suitable since dyschromatopsia and chromatopsia make the examination difficult, and in addition, color irradiation from the plates due to the spatial color disorder makes them impossible to use. It should also be noted that due to the agnosic disorder, instead of using the usual color names which correspond to a conceptual abstraction, it is much more appropriate to use terms of a more concrete or immediate character; for example, straw color instead of yellow, or color of the sky instead of blue.

The complexity of this set of alterations and their unstable character depending on the conditions of stimulation have not facilitated color research in these patients, especially in the M case, until the alteration in brain excitability and the corresponding process of chromatic degradation was understood.

At the beginning of the examination of subject M, in the summer of 1938, once his wounds had healed, the state of his color disorder was about the same as now. In general, he saw all objects as greenish although he was able to distinguish red. While walking down the street he came to believe that he had been lost in the city and had arrived in the countryside because he was seeing the ground with a greenish color like that of the grass. Seeing white and gray tones as green is what constitutes green chromatopsia, i.e., colored vision of objects that normally have neutral or white color. This occurs in violet-blind people, called *tritanopes*. It is an already known phenomenon although observed in just a few rare occasions. Although subject M is violet-blind, his color alteration is much more extensive. The considerable decrease in color threshold even in the colors he best perceives as red and green, establishes a general chromatic degradation with predominance in certain regions of the spectrum. This decrease in color threshold depends on the type of sensory activity, so the result varies according to luminous intensity, facilitation, etc.

Latency and color persistence must also be carefully taken into account to obtain reliable results during the experiments. In the case of M, the chromatic deficit is considerably improved by a very intense illumination of colors, although the characteristic disturbances do not completely disappear. However, under facilitation by maximum muscular effort and medium illumination, color vision is normal. It is thus crucial the facilitation through intense muscular contraction of the whole body, thanks to which the subject instantly recovers normal color vision and is able to distinguish both the so-called primary colors and the subtlest color shades of any kind, chromatopsia disappears, and white and gray recover their corresponding hues. Therefore, by changing facilitation intensity or stimulus intensity, the chromatic activity can be analyzed in a very diverse way.

Patient T case seems to show negligible disturbances in color vision; thus, in medium or low illumination, he perceives the yellow-blue pair with some difficulty, confuses blue with green very often, and is rather uncertain in naming colors. It seems that he does not present greenish chromatopsia, but a careful examination of this phenomenon shows that when a white sheet of paper is moved far enough away, there is a tendency for the margins to be seen with small pale green spots. In the same way, yellow and blue colors tend to be perceived as green. But even in near vision, this phenomenon of greenish chromatopsia can easily occur in subject T by

illuminating a white paper for an instant while the subject is in darkness. In this case, the paper appears full of green spots on a white background. This phenomenon is only noticeable for the right eye whose vision is weaker than that in the left eye. If the illumination, instead of being for an instant, is prolonged for a while, the greenish spots on white background are only perceived during the first instants, fading very quickly. This process indicates that the development of white color sensation in the T case shows a slowness that allows the observation of certain partial phases. In the M case under ordinary conditions, these phases are not completely overcome and give rise to greenish chromatopsia.

Similarly, if subject M, who might seem to have completely normal color vision under facilitation, is examined in the facilitated state in conditions similar to those of subject T (very brief stimulation), also presents greenish chromatopsia, somewhat more pronounced than in T. We already know that no matter how intense facilitation is, it is not capable of completely restoring the normal function, and both subject M under facilitation and subject T inactive show a certain slowness in reaction time giving place to some functional lag or asynchrony which must be conveniently searched for in order to make it evident.

The Schneider case of Goldstein and Gelb (1918), which would correspond to an intermediate degree of disorder between the two cases studied here, does not seem to have had any color vision disorder, according to these authors. However, we believe that their case, examined following the principles and methods here exposed, would show much more significant disorders than patient T and very similar to those of patient M under facilitation.

6.2. PERCEPTION OF THE COLOR SPECTRUM

By studying the perception of the color spectrum in patient M by means of a monochromator spectroscope, color vision in his right eye has been determined for different luminous intensities, in the inactive state and under facilitation by strong muscular effort.

By using the minimum luminous intensity under which the right eye of subject M in the inactive state can perceive color, the color spectrum he perceives is then reduced to red and green: red is seen from a wavelength of 680 nanometers (nm) up to 605 nm where green appears, and extends up to approximately 540 nm. This means that under such conditions, the perceived red corresponds almost entirely to the normal orange region, and the perceived green includes entirely the normal yellow region. The remainder of the spectrum does not provide any color sensation. This first result indicates that red and green are the colors for which our subject is most sensitive, and that he has a special dyschromatopsic disorder since the colors seen do not correspond to their place in the spectrum, but are the result of a chromatic alteration and degradation of other colors. There are therefore no neutral regions as often described in color blindness; thus, yellow, to which the subject is blind in these conditions, suffers the indicated degradation or dyschromatopsia instead of showing a colorless region in the spectrum. It should be noted that the colors for

which a normal subject in these conditions has more sensitivity are yellow and blue-green.

In a medium illumination intensity, the perceived red ranges from 750 nm to 595 nm where green appears, and green extends up to 435 nm. Thus, red and green expand their regions considerably, the first covering the normal red and orange regions, the second covering the normal regions of yellow, green and part of the blue region (see Fig. 6.1).

Finally, in very strong illumination, the spectrum expands a little more towards the ends; colors become much brighter and saturated, and new hues appear, although we are still far from the normal aspect. In the normal spectral region of red, in addition to red, a faint orange hue is seen. In the normal yellow region, there is a pale green region dotted with paler green and even yellowish spots that show a tendency to generate yellow. Likewise, in the normal spectral region of blue, there are small spots or traces of pale blue on a pale greenish background. In the normal region of violet, the subject perceives reddish and greenish spots as stripes. Thus, although red and green in general dominate under very strong illumination, there are

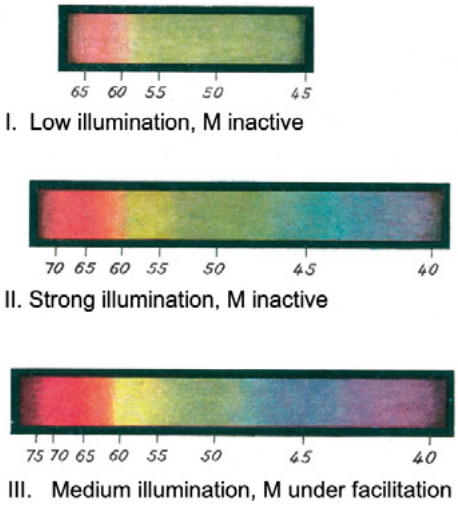

Figure 6.1. Perception of the color spectrum in subject M, right eye, according to illumination intensity and facilitation.

I: Illumination somewhat low and M inactive: he perceives red and green in the central region of the spectrum; thus, yellow is also seen as green, like the green of its own spectral region. These colors appear as little saturated and not very bright.

II: Strong illumination and M inactive. The perception of the color spectrum expands and now the missing colors in I appear: yellow and blue on a paler green background than the real green. The same happens in the violet region. (Due to the poor reproduction of the image, the greenish background in the yellow and blue regions is hardly appreciated.) Red and green look brighter.

III: Ordinary (medium) illumination and M under facilitation by strong muscular effort. The perceived spectrum becomes normal, and colors show good saturation, so it is practically normal vision.

traces of the other yellow-blue pair, and violet shows a very special mixture. In this situation, dyschromatopsia and weakness to yellow and blue-violet become very evident. The former because of the tendency to a greenish tint in the background; the latter because yellow and blue-violet only appear weakly and in a very reduced region. The mixed coloration of violet becomes green as soon as the illumination decreases a little, and reddish traces disappear.

These results show that depending on the illumination used, the subject is blind or almost blind to all spectral colors except red and green, and that the other colors do not become neutral or colorless, but change to an immediate color that is more stable according to the physiological characteristics of the subject.

In all these tests, the increase of visual field by the enlargement of the viewfinder slit does not seem to change the results.

By means of facilitation, color vision becomes normal, even in medium illumination. Not only does any trace of dyschromatopsia disappear, but colors appear completely normal in terms of brightness, saturation, etc., and red and green are now noticeably enhanced.

Results similar to those obtained with the spectroscope are obtained by other means, for example, using the color circle of Hering (1880) which includes twelve different colors. Subject M in the inactive state only perceives two colors, red and green, each filling one half of that circle. If the illumination is greatly increased, the perception is analogous to that already mentioned in relation to the spectrum: a greater number of hues can be distinguished. With facilitation by strong muscular effort, vision becomes normal, the twelve colors of the circle are perfectly distinguished, and the brightness of the colors already perceived in the inactive state increases. Thus, as said above, the color disorder extends to all colors and is very pronounced for some of them.

When examining color vision in subject M by means of different colored pieces of paper in very near vision and in the inactive state, it is observed that, as the illumination increases strongly, yellow, blue and white acquire more and more their true tones tending to dominate in extension over a green background, even though this background is not completely erased no matter how much the illumination is increased. Conversely, when the illumination decreases, the true tones of the mentioned colors weaken, reduce in extension, and a greenish tone tends to dominate. Violet never becomes dominant over the altered background, even with very intense illumination and near vision; therefore, violet is the color with the greatest deficit in the color disorder of subject M.

Experiments with color mixtures provide results fully consistent with what has been exposed. The behavior of the violet in its transition to purple by the mixture of red and violet, is remarkable. Violet and purple are seen as green or red, depending on their spectral conditions and the degree of illumination. In ordinary illumination, violet is seen as green and purple as red; in strong illumination, violet has green and red stripes, the green strips being dominant; and purple also has green and red stripes but red stripes are dominant. Under facilitation, each color goes to its true tone and the strips disappear. By mixing on a screen light rays of the three primary colors, red, green and violet, in such a way that they partially overlap each other, it

results that, from the seven different colors presented (red, yellow, green, blue, purple, violet and white), subject M in the inactive state can only distinguish two dominant colors, red and green that include all the others, as in the Hering circle. By increasing illumination, the subject sees red and various green tones with different intensities and traces of yellow and blue. By means of facilitation by muscular effort, all colors acquire their normal tone, the seven tones formed are perfectly distinguished, and colors that were already perceived well (red and green) increase their saturation and brightness losing the dirty and pale aspect with which they are usually perceived by the subject. The primary colors, red, green and violet, are then distinguished, as well as the colors resulting from the different combinations, i.e., yellow by a mixture of red and green; blue by confluence of green and violet; and white by a mixture of red, green and violet (the three primary colors).

All these tests show that subject M presents a chromatic degradation for the whole spectrum, although much more pronounced in certain spectral regions, which means a different increase in color thresholds, due to the excitability disorder. Color discrimination increases as excitability is improved by intensification of the visual stimulus and also by facilitation. Thus, red and green, first limited to the central zone of the spectrum, expand towards the extremes of this one, encompassing other diverse tones which appear in increasingly evident traces when the illumination increases, the normal vision being reached only by means of facilitation, provided the first moments of sensation have elapsed.

The behavior of white and grays goes in parallel with that perception of the spectrum. A white paper in weak illumination, no matter how big the paper is, appears with a dark green tone that when illumination increases becomes pale green or yellowish green; and finally, in strong illumination, an increasing number of larger and larger whitish spots are seen on a pale green background. Only by means of facilitation color vision becomes normal. The series of grays and even black behave in a similar way and present a more or less dark greenish tint; black always contains a certain amount of albedo, thus, it also presents a more or less dark greenish tint. Likewise, mirrors and objects with metallic shine are seen with a greenish tone of varying intensity depending on the circumstances. The greenish tint in white (greenish chromatopsia) resembles a pale green close to yellow, according to the comparison made by the subject between the color perceived and the twelve colors of Hering's circle.

6.3. DIFFERENTIAL COLOR SENSITIVITY

From all that has been said, it can be understood that color discrimination in a subject suffering from the previously exposed chromatic degradation should be extremely reduced, since the vision of the spectrum in medium or low illumination is reduced to a couple of colors due to dyschromatopsia in colors such as yellow, blue and violet.

By means of the aforementioned monochromator spectroscope, which allows the isolation of colors and the determination of their wavelengths along the entire spectrum, the different color bands perceived by subject M and by a normal subject

(as a reference) are determined. Thus, the boundaries of the different color bands, from extreme red to violet, are determined. Some 165 different tones, and even more, can be established in the whole spectrum. But following a successive procedure with a single spectroscope, twenty to thirty different bands can be distinguished by taking advantage of the fact that differentiation increases as the illumination increases.

The results of these differential color sensitivity tests under different degrees of illumination are shown in Fig. 6.2 for patient M and a normal subject. These tests are not at all easy to perform even in a normal subject, and for this reason and greater certainty, different luminous intensities are used in the M case in order to ensure the results. In the figure, the abscissa of a given experimental point indicates the central wavelength of a given color band, and the ordinate indicates the difference between the wavelengths of the boundaries of that color, i.e., the spectral width corresponding to that color.

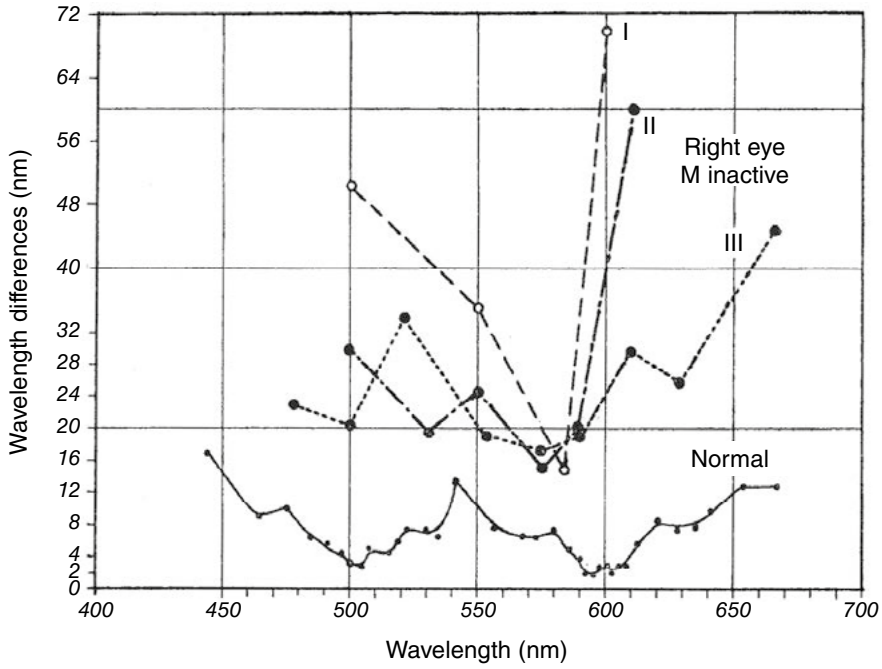

Figure 6.2. Differential sensitivity to colors. Perceived spectral widths versus color wavelength (see the text). The lowest curve refers to a normal subject, the other three refer to the right eye of M inactive, for different luminous intensities of the spectrum: I, low-medium intensity; II, strong intensity; III, very strong intensity. The curve of the normal subject corresponds to an intensity slightly stronger than type I.

In the lowest curve (normal subject) more than thirty hues are perceived whose spectral widths have very different values; widths are smaller (and therefore color changes are more abundant) in the yellow region and the green-blue region, two

regions where color discrimination is greater. But instead, subject M using the right eye and in the inactive state shows a very reduced differential sensitivity since he perceives very few colors and with very broad spectral widths. In the normal individual, more than thirty spectral bands of different hues are obtained whereas in M, only five to ten bands depending on the illumination used. Concerning the greatest color discrimination in a normal individual, it only occurs in the region of yellow, where the spectral widths are smaller and therefore the hues are more numerous. This region corresponds to the change from red to green in subject M. This maximum differentiation coincides in the M case for the three types of illumination employed: type I (medium-low intensity), type II (strong intensity) and type III (extremely strong intensity). The curve of the normal subject corresponds to an intensity a bit stronger than type I. Differentiation increases in subject M as the illumination increases: the spectral bands become narrower, and therefore there are more different spectral bands but, even in maximum illumination, the spectral band widths in the region of maximum differentiation are approximately ten times the smallest spectral width perceived by the normal subject.

Concerning the other maximum of differentiation in the blue-green region of the normal curve, it is present only in very slight signs in M with very strong illumination. This lack of the other maximum can be interpreted as a result of blindness or strong weakness to the third primary color, blue-violet. Thus, the single maximum is established by color combinations in the transition from red to green, i.e., in the yellow region.

In summary, in the tests to determine the threshold of differentiation of color changes, it is found that the color bands in the M case are too wide and scarce, thus showing a very high threshold, and the obtained curve shows only a single maximum of differential sensitivity in the yellow region.

As regards the distribution of brightness in the spectrum, i.e., the amount of white or resemblance to white shown by the different colors, no major anomalies were observed in the M case in the simple tests performed by the direct method of comparing the brightness of each color with white using a photometer. The characteristic bell-shaped curve shows the maximum brightness in the yellow region, as in a normal subject. Concerning the Purkinje phenomenon (Purkinje 1825), when illumination is reduced, the brightness of the spectrum shifts towards short wavelengths, as in a normal subject. However, more complete and precise quantitative experiments are still needed in this type of phenomenon. It should be noted that at the beginning of the examination of patient M, the Purkinje phenomenon was inverted, i.e., in low light, red seemed brighter than blue. We have also observed a clear trend towards the reversal of the Purkinje phenomenon in other brain-injured people with occipital lesion and visual field disorder.

7. Chromatic dynamics

7.1. PHOTOCHROMIC AND PHOTO-HETEROCHROMIC INTERVALS

From the features previously studied on the perception of the chromatic spectrum, we can get an idea of the nature of the alteration in color vision. Here, we shall expose more accurately the conditions of brain excitability in the M case in relation to the different colors.

When the luminous intensity of a color decreases greatly, it appears as colorless; in fact, any color with very tiny luminosity is seen only as whitish (simple luminosity), and only with greater luminosity, it is chromatic. Thus, there is a colorless interval (*photochromic* interval) between the threshold of simple luminosity and that of color perception. Only with respect to red there is controversy about whether it shows a colorless interval or not; followers of the dualist theory of vision such as Kries (1899), Köning (1894) and others deny it, whereas researchers such as Hering (1874/1878, 1880) claim it. The photochromic interval is minimal or nil for red, and increases progressively as the color wavelength becomes shorter. The fundamental disorder of brain excitability, consisting of slow reaction time, excitability deficit and especially asynchrony, alters the photochromic interval in a very special way. In addition to the increased threshold due to the excitability deficit, the colorless interval expands considerably and becomes very conspicuous even for red. Red and green, although with a photochromic interval larger than normal, do not show any other particularity. Instead, all other colors, including white (although it is not a spectral color in the true sense), show what could be called a *photo-heterochromic* interval, meaning that they go through a different color than their own before reaching their true normal tone. In these colors, the interval between simple luminosity and the color itself is not as simple as in a normal subject, and shows an intermediate phase or very singular dyschromatopic alteration that evidences a color decomposition for a certain luminous intensity, giving rise to sensory manifestations (sensory levels) that do not exist in a normal individual.

By determining the intensity-duration excitability curves for the different phases of the photochromic and photo-heterochromic interval, bundles of curves are obtained similarly to what was already described when dealing with the fundamental experiment in the general part (Fig. 3.1). The curves for these intervals are obtained by the same procedure as for the light excitability curve shown in Fig. 5.1.

The results obtained for the photochromic interval of red and green are shown in Figs. 7.1 and 7.2 respectively. In each figure, the lower curve corresponds to minimum sensation of luminosity and the upper one to minimum sensation of color. The photochromic interval corresponds to the separation between the two curves. The scales for these two and subsequent figures are the same, taking the logarithm of luminous intensity in the ordinate to facilitate the representation of the curves.

All these tests refer to case M, right eye, the subject being totally inactive without any facilitation, which makes the experiment easier. The same subject under facilitation would give a smaller photochromic interval, i.e., curves closer together, and also lower threshold and faster reaction time. These last features would be even more pronounced in the T case. For a normal individual, the two curves in each of the two colors considered would be extremely close, would have a more pronounced curvature, and the useful time would not exceed half a second at most. Thus, as the brain

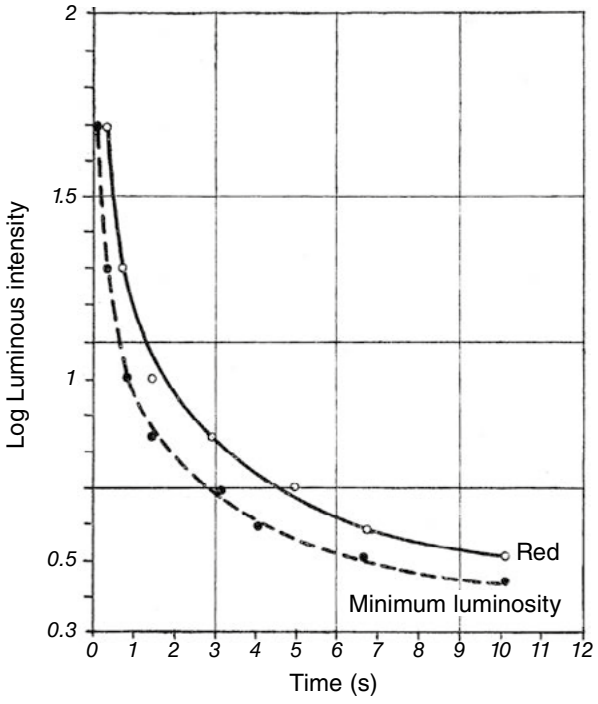

Figure 7.1. Photochromic interval for red in pagient M inactive, right eye. Upper curve: intensity-duration excitability curve for minimum color sensation. Lower curve: intensity-duration curve for sensation of minimum luminosity.

excitability deficit increases, the curves show a higher rheobase threshold, a longer useful time, and the separation between the curve for sensation of minimum luminosity and that for the color in question is greater, i.e., the photochromic interval increases. All these relationships are expected given the different light excitability curves for M inactive, M under facilitation, T (inactive) and a normal subject, shown in Fig. 5.1.

The photochromic intervals for red and green in the M case are very similar, with the interval for green slightly larger. In all these curves, each experimental point is the mean value of several measurements. The duration of the stimulus results perhaps a little longer due to the experimental conditions imposed in order to discriminate between the sensation of luminosity and that of color, but this is not relevant.

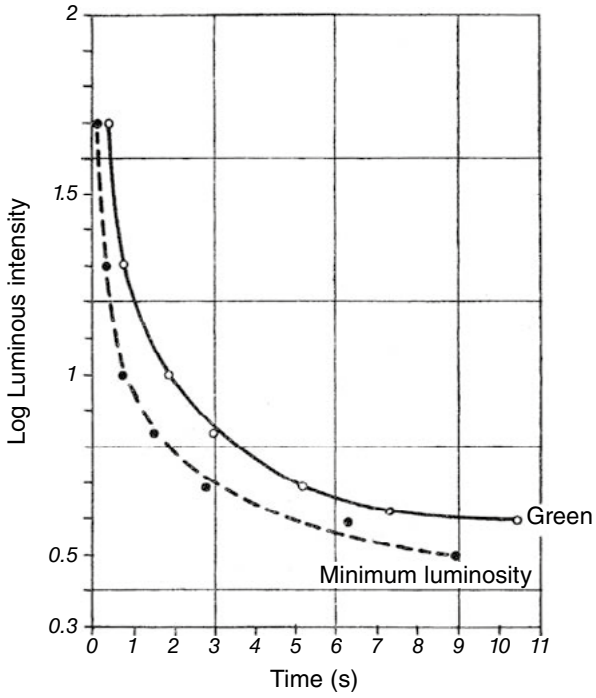

Figure 7.2. Photochromic interval for green in subject M and conditions as in Fig. 7.1.

Examining the interval in question for yellow, blue and white, under the same conditions as before, more phases appear. In the determination of the corresponding curves, only three sensory levels (phases) will be chosen: the phase of minimum luminosity, the phase of color that appears when the intensity increases and which does not correspond to the color tone we are studying, and finally the appearance of the color itself, which only appears in traces by means of small spots over the color of the preceding phase. Therefore, it is a complex interval that we have called 'photo-heterochromic' to highlight the intermediate phase of altered color.

The phases in the M case for each of the colors mentioned are as follows. The yellow color, after having passed the colorless phase, goes on to light green when the luminous intensity increases; and when the intensity increases even more, some yellow stripes are seen over the green background; and finally when the illumination is considerably increased, the area of yellow can dominate over that of green. For blue, there is an analogous process: colorless phase, green phase and phase of blue spots over a green background. Violet color changes from simple luminosity to green, and later shows a tendency to reddish and lilac. Finally, white, after the colorless phase, changes to light green, and later displays white stripes over some green. It should be noted that in the case of M, in the inactive state, no matter how much the light intensity is increased, he cannot perceive colors fully normally except red and green. For white and for the yellow-blue pair, when illuminated very strongly, the most that can be achieved is to reduce considerably the abnormal greenish hue, and that it appears in less quantity than the color under study. In order to reach the completely normal color, facilitation must be applied.

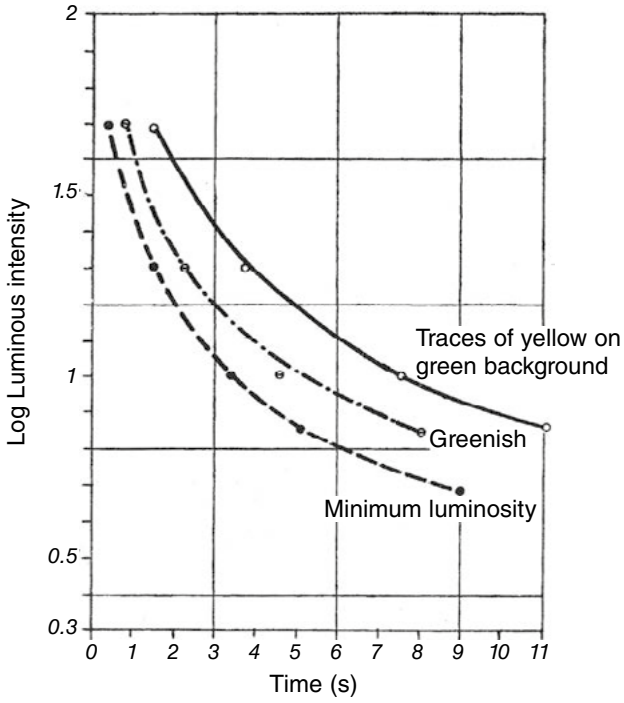

Figure 7.3. Photo-heterochromic interval for yellow in subject M and conditions as in Fig. 7.1.

The photo-heterochromic intervals for yellow, blue and white are respectively shown in Figs. 7.3, 7.4 and 7.5. The corresponding curves have, for all the phases, a smaller concavity and a higher rheobase threshold than those for red and green shown in Figs. 7.1 and 7.2. Therefore, yellow, blue and white are more difficult to

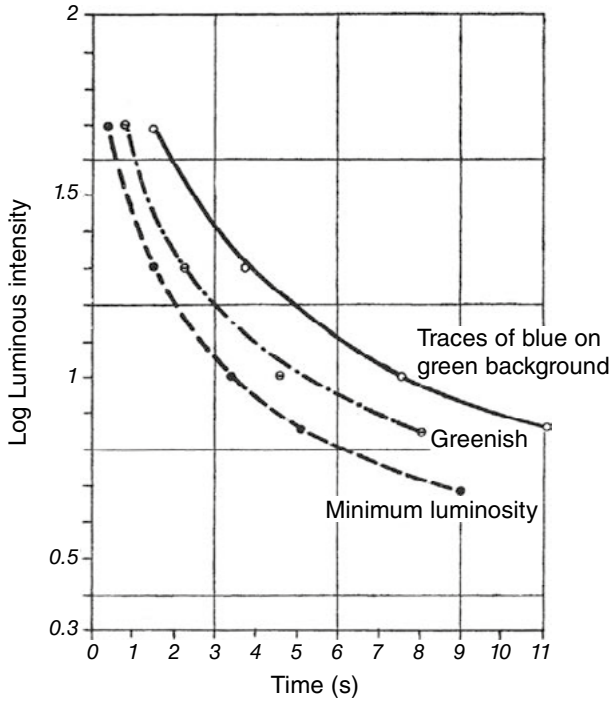

Figure 7.4. Photo-heterochromic interval for blue in subject M and conditions as in Fig. 7.1.

excite, i.e., slower to react. Due to the triple phase that these colors present, the interval between the sensation of minimum luminosity and the color itself, even if only traces, is considerable, and would be even much greater if the threshold for a more complete display of the color itself is reached. Thus, for white it is necessary to increase from fifteen to twenty times the light intensity threshold corresponding to the phase of white traces in order for the green-white ratio to be inverted and white to appear with green traces. If light intensity increases even more, white becomes more pronounced but there are always traces of green. Thus, a fourth curve could be added, which would be placed well above the corresponding curve of traces of color over a green background, and which would indicate an inverse relationship, i.e., the color itself and some traces of green. Even so, this new curve would not be fully similar to those obtained for red and green, since these two colors appear without any mixture.

By facilitation, the bundle of curves for a photo-heterochromic interval is considerably narrowed, the curves tend to approach the axes and each other, the intermediate dyschromatopsic phase is considerably reduced, and in subject T the synchronization of these phases (out-of-phase functions) is so large that each dyschromatopsic phase is practically impossible to determine because it only appears in minimal signs.

In short, this type of experiment shows that the excitability deficit affects the entire chromatic system, but certain colors are affected more intensely than others. The decrease in brain excitability, thus causing asynchrony, leads to an increase in

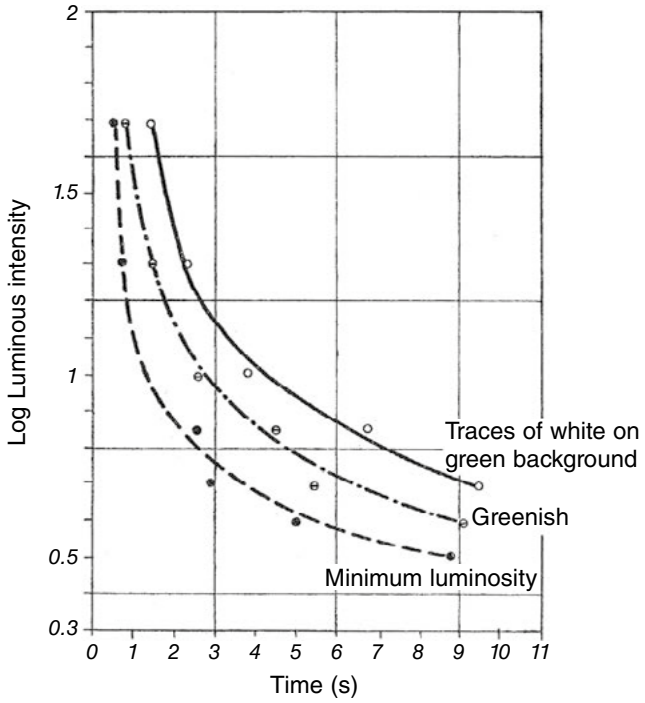

Figure 7.5. Photo-heterochromic interval for white in subject M and conditions as in Fig. 7.1.

the colorless interval; the more affected the color, the greater the interval is. Furthermore, due to this asynchrony, a decomposition of certain complex colors into their chromatic components is generated during the photo-heterochromic interval.

In a normal individual there are very small differences in excitability for the various color sensations, and according to Kleitman and Piéron (1925) and Piéron (1932, 1934), colors are sorted according to the speed of perception, red first, green very soon after, and blue shows a much lower speed. Pathologically these differences become considerably more pronounced, affecting in greater proportion the less excitable colors, so blue is one of the most altered colors. Whereas in a normal individual the small differences in color excitability are easily overcome, in subject M the asynchrony is so great that it even tends to exclude colors that are more difficult to be excited, thus reducing the number of hues perceived.

7.2. DYSCHROMATOPSIA AND CHROMATOPSIA. SIMPLE AND COMPOSITE COLORS

The aforementioned tests on photochromic and photo-heterochromic intervals, and especially the alteration of certain colors when passing through the dyschromatopsic phase, constitute the fundamental experiment in the dynamic analysis of colors. The

bundle of curves that can be obtained, especially for colors with photo-heterochromic interval, is just the asynchronous bundle of desynchronized (or partial) functions described in Sec. 3.2.1 when dealing with the fundamental experiment.

It results from these tests that only the red-green pair maintains its true color whereas the other yellow-blue pair and also white show phases whose tones are completely different from their true colors, depending on the degree of illumination. This color change is called dyschromatopsia; thus, blue and yellow are seen as green but of a different lightness to that of real green. White in weak illumination is also seen as green, and in ordinary and even strong illumination it maintains this greenish shade although paler, turning towards yellowish green or pale green. This case is also a dyschromatopsia, but due to the special character of white, which belongs to the neutral series (grey-white), this alteration of white that produces a colored vision is also called chromatopsia. This is in fact a consequence of dyschromatopsia in certain colors, so all these color alterations result from the same process.

We are therefore faced with a special chromatic degradation that affects all types of colors except red and green; these remain unaltered and evolve directly to the achromatic phase when the intensity of the stimulation decreases. The other colors, for example, yellow and blue (the other chromatic pair), are seen as green in ordinary or medium luminous intensity, that is, they go through a somewhat close hue that must be considered as originated in the disaggregation of the chromatic function by virtue of an asynchrony of normally synchronized (united) functions.

7.2.1. Primary and composite colors

The perception of the spectrum by subject M under ordinary conditions of stimulation is essentially reduced to two unalterable colors: red for the long wavelength region, and green from the yellow region to the shorter wavelength region. The studied chromatic degradation and decomposition would allow us to classify colors in simple and composite colors. Simple colors, of higher excitability, persist. However, composite colors, of lower excitability, tend to be excluded and replaced by more stable hues resulting from the decomposition; these hues would therefore contribute to the formation of the composite color. If the simple colors red and green are called primary colors, the others can be called composite colors since in their decomposition by asynchrony they change to one of the two primary colors. Composite colors could also be considered as differentiated colors, which means an increase in differential color sensitivity.

In the process of degradation, the differential yellow-blue pair is lost, and these colors change to the next simpler hue or to certain mixtures, depending on the colors. Thus, yellow and blue become light green, and violet becomes green or red depending on its spectral position. In the case of white, considered the maximum composite color, a yellowish green or light green color appears as a result of the mixture that is then established. The primary red-green pair subsists but somewhat weakened as we know, losing brightness and saturation.

In our brain-injured people, all kinds of sensory functions experience a special disaggregation due to asynchrony, and the most complex functions are delayed or

even excluded. Thus, in the case of colors, white is one of the most altered functions due to its most complex character. Even in normal individuals, there are different reaction speeds for different colors, and although the differences are small, they have been accurately determined by Kleitman and Piéron (1925) and Piéron (1932, 1934) using a special spectrocolorimeter.

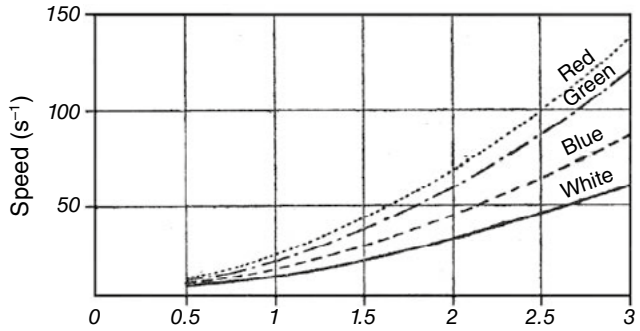

Figure 7.6. Speed in establishing color sensation as a function of the logarithm of luminous intensity, for different colors in foveal vision. Figure adapted from that of Kleitman and Piéron (1925).

In foveal vision, the development of color sensation until a steady state is reached has maximum speed for red, a little lower for green, much lower for blue and even lower for white, as can be seen in Fig. 7.6.

Due to the deficit in brain excitability, these differences increase considerably, the colors of lower speed being the most affected, which tend to be excluded or to appear incomplete even in extremely high stimulation. Thus, the pathological alteration follows the same sequence that exists in the normal subject; red and green are much less deteriorated than blue or white.

If we observe what happens in the recovery from the chromatic disorder in brain-injured people who have become almost blind and slowly recover their vision by regression of the disorder, we find a similar sequence. Although our observations are not sufficiently precise in this matter, we can count on the indications of Economo, Fuchs and Pötzl (1918), Best (1920), etc., who point to red as the first color restored in the recovery from cortical blindness, followed by green and yellow, and much later by blue which may be missing for a long time. Although this recovery has not been possible for us to study it in detail, we can now specify the exact order in which it should be carried out in view of the characteristics of color excitability. For the moment, we shall simply point out that the order in color recovery coincides and perfectly fits the degree of color weakness in our cases.

By means of special tests, a similar order in color disturbance is obtained. Thus, in the experimental intoxication with mescaline in normal subjects, very diverse sensory alterations are shown, and with respect to colors, Beringer (1923, 1927) describes certain phenomena which are very important for the knowledge of the diverse excitability of colors: "The chromatic qualities disappear, the whole space is seen uniformly

gray and colorless." As for the dominant colors, the subject concerned says: "everything looks either green-blue or green-red. I see everywhere only red and green, and I look for blue and yellow." Mescaline, a drug that reduces brain excitability, therefore produces a chromatic alteration in which the yellow-blue pair is most affected.

Therefore, in all the cases considered of chromatic alteration by modification of the nervous system, such as our brain-injured patients, cases of recovery from cortical blindness and cases of mescaline intoxication, the sequence of color alteration is always the same. The only basis for this is the alteration of brain excitability by which the small differences existing in the normal subject increase considerably and in different proportions.

7.2.2. Dyschromatopsias

We think that the dyschromatopsic degradation of composite colors presents a fundamental interest for the theory of color vision since it seems to be the usual form of color alteration in brain lesions. Weakness to yellow and blue-violet, and the confusion of blue with green or, rather, the perception of both colors as green, which occurs in cases of minor disorders such as in the T case, are phenomena that we have detected in a large number of injured people with occipital or other central lesions that disturb visual perception to varying degrees. This type of alteration was not only a predominant and constant phenomenon in these brain-injured people, but it was the only one observed. However, in the literature on chromatic disorders due to brain injuries, this type of alteration in the yellow-blue pair is rarely mentioned. This is probably due to a defect in the examination of patients because red-green color blindness from retinal anomalies is much better known and frequent, and it is possible that attention has been focused only to this type of color disorder.

Two types of dyschromatopsia can be distinguished; one for colors that become green such as blue and yellow, and another for those that become red such as purple and sometimes violet.

The intensity of dyschromatopsia depends on the degree of brain disorder and the dynamic conditions involved in sensory excitation (facilitation by muscular effort, by binocular effect, etc.). Thus, subject M can exhibit various degrees of impairment depending on the conditions of the experiment. However, it should be noted that even in the optimal state of excitability (binocular vision and facilitation by maximum muscular effort), asynchrony is still present to some extent. This allows to obtain still a photo-heterochromic interval (dyschromatopsia) by sufficiently reducing stimulation intensity. Subject T usually distinguishes the twelve colors of Hering's circle; but in weak illumination, yellow and especially blue tend to become green to some extent, as does white, although the process is much less pronounced than in subject M. In a very brief exposition of such colors, there is also some tendency towards green, and even in ordinary conditions of medium illumination the mutual confusion of green with blue is very frequent. Detailed indications about the various types of vision (or levels of excitability) in relation to colors will be given further on when dealing with chromatopsia.

In relation to dyschromatopsia, it is interesting to comment on the syndrome that Poppelreuter (1917) called *Psychische Farbenschwäche* [psychic color weakness], according to which the different tones of a given color are not perceived in a first stage, i.e., there is a reduction in differential sensitivity; and in more acute cases, colors with some similarity get confused with each other. This author states that the cardinal symptom of psychic color weakness in occipital lesions consists of the confusion of green and blue with each other, and he sees in this symptom a completely different behavior from that of not distinguishing colors due to retinal abnormalities. From the descriptions of Poppelreuter, it seems that he ascribes the referred syndrome to a psychic deficit for the concept of color (*Schwäche in der Auffassung des Farbentones* [weakness in the perception of color tone]). On the contrary, we think that it is not necessary to admit complex intellectual disorders since it is simply a color weakness of sensory origin, a deficit in differential sensitivity due to an alteration in brain excitability, and a dyschromatopsia that turns blue into green. The disorders described by the aforementioned author refer to subjects with much lighter disturbances than those of subject M; at most, the chromatic alteration consists of the confusion of colors with similar shades such as blue and green. These are subjects with disorders of similar intensity to those of patient T and even much less, but properly examined would show more complex alterations, as has been seen in T concerning greenish chromatopsia when the stimulation with white color is extremely brief.

7.2.3. Chromatopsia

The fact that white and the whole series of greys up to black are seen as green constitutes green chromatopsia or colored vision, which in our cases acquires a more or less intense green hue for reasons that will be seen later. This singular disorder contributed to considerably hamper the interpretation of the color alteration in the M case, already so complex due to the peculiar physiological conditions. This greenish perception of colorless objects, i.e., of the neutral series (white, gray and black), is known to be characteristic of subjects with *tritanopia* (violet blindness), an extremely rare disorder.

Chromatopsia disappears completely by means of maximum facilitation, although due to some slow development of white sensation, there is still some greenish tint in the first moments, which disappears quickly being replaced with full white. In the inactive state of subject M, a very important attenuation of the greenish tint is achieved when white is strongly illuminated and also observed in very near vision, although there are always small traces of green. Even the solar disk would be seen with some traces of green. As for grey and black, subject M sees them as green too; the medium grey is perceived as medium green, darker than that perceived in white, and black is perceived as a very dark green. If black is very strong and bright, as on a shiny satin surface, then mixed green and dark strips appear. All kinds of white light show some green. It is not surprising then that black is seen with a bit of green, since almost all blacks reflect a certain amount of light and never fully meet the conditions of absolute black.

Chromatopsia was first investigated in subject M, and much later in subject T, who only shows it when the stimulation is very brief. In this subject it is revealed by illuminating a white paper for an instant in darkness; the greenish tone appears only in the first instants of the establishment of the sensation of white, since his asynchrony is extremely small compared to M. More recently we have verified the existence of chromatopsia in a former brain-injured patient with significant visual field alteration of an intermediate type between concentric reduction and homonymous hemianopsia. This subject presents a state of brain excitability for all visual functions very similar to that of subject M in monocular vision and under facilitation by muscular effort. We have therefore proven accurately the existence of chromatopsia, and in different degree in three different brain-injured subjects.

In both patients, M and T, there are various types of vision according to the conditions of excitability (facilitation, binocularity, etc.) and also to the eye being used, since there is a small difference in vision between the two eyes. Therefore, by combining the various factors, very different states of excitability can be obtained in which chromatopsia is present to varying degrees. Examining carefully such types of excitability, the following phenomena are observed in subject M, under intense illumination of a white piece of paper:

In binocular vision and under facilitation by maximum muscular effort, no green is seen; only by paying close attention he can perceive traces of green in very peripheral vision and at the beginning of the sensation.

Using only the right eye and facilitation by maximum muscular effort, the central part of the sheet is seen white, and peripheral green spots over white background are more abundant than in the previous case.

In binocular vision and in the inactive state, greater amount of green is seen in peripheral vision than in the previous test, and some green dots over white background are seen in the center.

Using only the right eye and in the inactive state, white predominates but there is a large amount of green.

Using only the left eye and in the inactive state, white also predominates but with even more amount of green than in the previous case.

Even under the best conditions of brain excitability (binocularly and with facilitation by muscular effort), a greenish hue is perceived if the observation time is very short. By strongly reducing the illumination, full and permanent green coloration is also obtained even in binocular vision and with maximum facilitation.

There are therefore important differences with subject T who, in order to perceive a greenish hue using only his right eye, a white paper in the dark must be illuminated only for an instant. This means that only in the first moments of the sensation there is a chromatic asynchrony causing chromatopsia. If the stimulation lasts longer, the green hue disappears very quickly, and a normal white appears. In this subject, greenish perception is minimal and only appears as green spots, very close together, over a grey background, and only in the right half of the visual field while the left half is completely white. Chromatopsia appears in the weakest part of the visual field, and although there is a concentric reduction involving the two halves of the field, the right side (contralateral to the brain lesion) is somewhat

weaker. If in the test with illumination for an instant, facilitation is applied (whose effect on this subject is much weaker than in M), the green spots become much paler changing to yellowish and appear more spaced out. Using the left eye, which has slightly better vision, no trace of green is obtained even if the white paper is illuminated for an extremely short time. It must be noted that if the right eye (the worse eye) is used, even by reducing illumination conveniently, chromatopsia cannot be obtained in a stable or permanent way, therefore it is not possible to determine excitation curves corresponding to the different stages as we did in the M case.

As for the third subject mentioned above who has presented greenish chromatopsia, a behavior very similar to that of subject M in monocular vision under facilitation is observed. This means that only under very intense illumination, the greenish hue of white disappears, so in medium or ordinary illumination, the abnormal greenish hue appears in a permanent and pronounced way. A yellow or blue sheet of paper is also altered presenting an intensely green area in the lower left quadrant of the visual field (weaker region in this third subject).

Chromatopsia, like so many other new phenomena studied here, goes easily unnoticed by patients who suffer from it, and even in well-educated people, as in the case of our third patient indicated, we have been able to discover it quite pronounced after several years of having suffered the brain lesion, without the subject being aware of its existence during all that time.

The fundamental experiment of varying the illumination of a white object to obtain different intensities of green, ranging from total dark green in very low illumination to traces of extremely pale green or yellowish in extremely strong illumination, is equivalent to perform the transition from a reduced perception of the color spectrum to a more complex and differentiated perception of it.

The M case and all these brain-injured people who present the chromatopsia described above, can be considered either tritanopes or tritanomalous, depending on their brain excitability, giving rise respectively to blue blindness or to blue weakness, blue being the third primary color according to Young-Helmholtz trichromatic theory (Young 1802, Helmholtz 1852). However, we know that the color disorder is more general and not circumscribed to blue; yellow also disappears, and the red-green pair loses saturation and brightness. We must also consider the dyschromatopsia of the yellow-blue pair, which degrades these composite colors giving rise to green without the appearance of neutral spectral zones as occurs in many other cases of retinal type blindness. Thus, we find that under the usual conditions of subject M, he sees a large part of the spectrum (much more than half) only green, and the rest of the spectrum, red, and both colors with a weak saturation. For this reason, the excitation of the entire spectrum, which normally produces white, cannot give in that subject more than a mixture of the indicated colors giving rise to a yellowish green or a pale green. Thus, the explanation of green chromatopsia is simple, a direct consequence of the chromatic deficit in this subject, since his inability to see white is produced by the lack of appropriate ingredients in the spectrum. In this case there is not enough provision of luminosity nor chromatic neutralization of colors with each other.

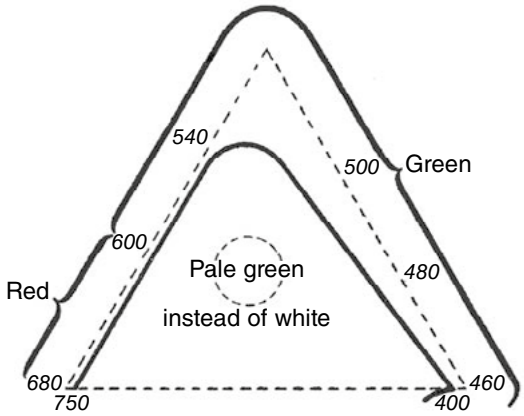

Figure 7.7. Triangle of colors in patient M in the inactive state. Wavelengths are indicated in nanometers. The braces indicate dyschromatopsia, and the central circle represents green chromatopsia.

By representing the dyschromatopsia of subject M in the triangle of colors (Fig. 7.7), it is easy to appreciate the difficulty to form white. In the Maxwell triangle of colors (Maxwell 1860), each color located on one side of the triangle has its complementary color in the diametrically opposite position with respect to the center, therefore, the combination of both colors in the center produces white. According to the representation of this triangle for patient M in Fig. 7.7, white cannot be achieved by joining complementary colors, and the fusion of the whole spectrum only gives a pale yellowish green hue.

Therefore, chromatopsia is not due to some special contrast phenomenon, as has sometimes been hypothesized, nor to even more problematic absorption phenomena, but depends solely on the dynamic conditions of brain excitability. The dynamic conditions, by altering color excitabilities and degrading the spectrum, modify the conditions of spectral addition. Chromatopsia is therefore at the heart of color disturbance and constitutes the synthesis of the whole disorder. It is also shown that the process of formation of white is in agreement with the theory of fusion of the simplest colors (Young 1802, Helmholtz 1852), thus white is the most complex color or function.

7.3. INVERSION OF COLOR ISOPTERS

The special chromatic alteration in the subjects we are studying, especially subject M, still allows us to deal with a very interesting complementary aspect which is the arrangement of colors in the visual field, i.e., color isopters. In a normal situation, the periphery of the visual field is blind or extremely weak to color, and the thresholds for the different colors have different boundaries giving rise to color isopters. From the periphery to the center, the outermost color is white; the next are blue and yel-

low; more centrally, red, and finally green is the innermost, the latter being the least extensive in the visual field.

In our two cases, the layout of these boundaries (color isopters) is totally different; the outermost color is red, even more peripheral than withe. Thus, all the colors are distributed in two types of isopters, one more eccentric for red and another more central for the different greens resulting from the alteration, arranged according to their degree of lightness. Since sensitivity in the visual field decreases rapidly from the center to the periphery and colors show very different excitabilities, they are positioned along the visual field meridian according to their new excitability levels.

Due to the strong concentric reduction of the visual field, especially in subject M, colors occupy a very small area in the field, although by means of facilitation a considerable enlargement is achieved; but despite this enlargement, the position of color isopters is still inverted, i.e., the red-green pair more peripheral than the yellow-blue pair. By studying color isopters in subject M, inactive and under facilitation, and in subject T, the development of the alteration of color isopters can be easily followed because the different cases show different arrangements of them.

The position of color isopters in the right eye of subject M in the inactive state is shown in Fig. 7.8. The red isopter is the most external, the green is much more

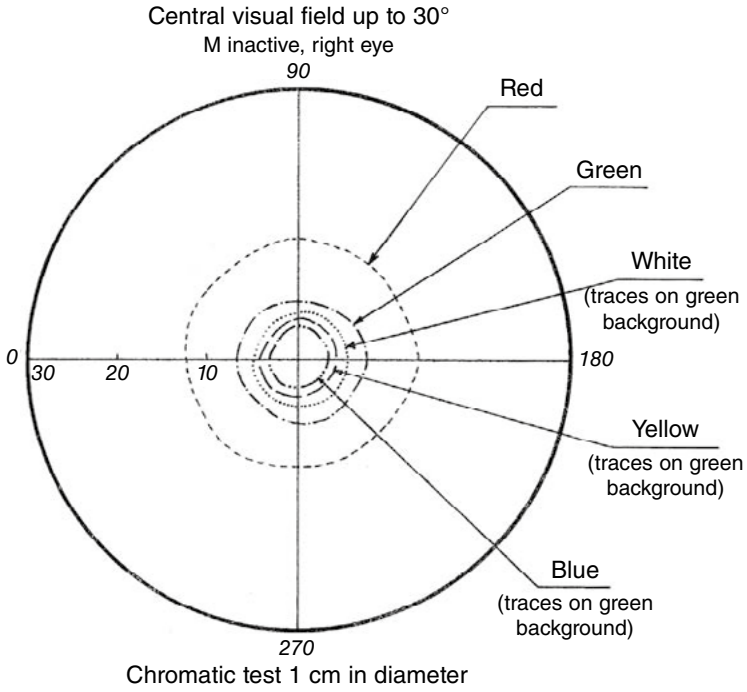

Figure 7.8. Position of color isopters in the visual field of the right eye of subject M in the inactive state. Boundaries are indicated for colors only; each color can produce a sensation of simple colorless luminosity at a much higher eccentricity.

inside, and the isopters for white, yellow and finally blue are situated from 5 to 3 degrees. These three colors are shown only as traces of their true color on green background. The different hues can be perceived by using colored tests of 1 cm in diameter under good illumination. The isopters shown in the figure only reveal one of the aspects of color function since, due to the excitability conditions, very diverse changes occur in the visual field. Thus, by carefully performing the chromatic test from the periphery to the center, either the photochromic or the photo-heterochromic interval is found again, depending on the type of color. The values obtained for the right eye of subject M in the inactive state are shown in Table 7.1.

Table 7.1. Degrees of visual field for boundaries of minimal simple luminosity and minimal color sensation in the right eye of subject M in the inactive state, using colored tests of 1 cm in diameter.

Color	Minimal simple luminosity	Minimal color sensation
Red	24°	..... 13° pure red
Green	14°	..... 6° pure green
White	23°	12° dyschromatopsic green; 5° traces of white
Yellow	17°	9° idem ; 3° traces of yellow
Blue	14°	7° idem ; 3° traces of blue

As indicated in Table 7.1, with regard to the sensation of minimal luminosity only, red and white are almost together although red is more eccentric than white, and yellow is more eccentric than green. With regard to the emergence of color sensation, dyschromatopsic greens emerge for white, yellow and blue (in order of lightness), and are situated more externally than the real green. The emergence of the true color for these three composite colors occurs only in traces and more centrally than the real green. We can then say that color isopters show an inverted position since the red-green pair is much more external than the yellow-blue pair. As for white, it is in an intermediate position between both pairs of colors, always bearing in mind that white, yellow and blue are only perceived as traces. Colors are then arranged according to their characteristic excitability, complying with the above-mentioned relationships on chromatic intervals of diverse complexity.

The same tests for the right eye of subject M but now under facilitation by strong muscular effort result in more enlarged color isopters (Fig. 7.9), approximately double or triple depending on the colors, but the sequence remains the same as in the inactive state. However, there is the important novelty that pure white without green can be seen at about 3°, and yellow and blue are seen in the center (0°) almost entirely devoid of discromatopsic green hue. As in the previous test, photochromic intervals of diverse complexity are obtained, as shown in Table 7.2.

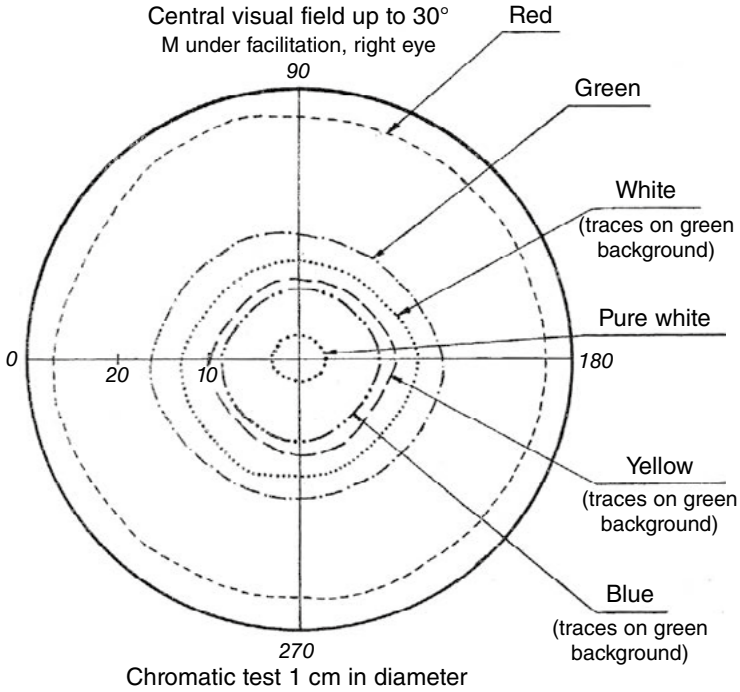

Figure 7.9. Position of color isopters in the visual field of the right eye of subject M under facilitation by strong muscular effort. Same sequential arrangement of color isopters as in Fig. 7.8 but they are more eccentric here. There is the important novelty that pure white can be seen at 3°, and yellow and blue are almost completely free of green at 0°.

Table 7.2. Degrees of visual field for boundaries of minimal simple luminosity and minimal color sensation in the right eye of subject M under facilitation by strong muscular effort, using colored tests of 1 cm in diameter.

Color	Minimal simple luminosity	Minimal color sensation
Red	45°	..... 27° pure red
Green	35°	..... 17° pure green
White	40°	22° dyschr. green; ..... 13° traces of white; 3° pure white
Yellow	32°	20° " " ; ..... 10° " yellow; 0° almost pure yellow
Blue	32°	20° " " ; ..... 9° " blue; 0° almost pure blue

Compared to the previous test in the inactive state, it can be seen that the intervals are smaller, especially the photo-heterochromic intervals; pure white is obtained close to the center, and both yellow and blue with almost no green trace are obtained at the center. A larger number of phases can be determined in the compos-

ite colors, as is indicated in Table 7.2: simple luminosity, dyschromatopsic green, traces of color, and pure or almost pure color. We see that facilitation makes that the boundary for traces of white in inactive state (at 5°) becomes the boundary for full white (at 3°), and similarly for the other composite colors. But as we know, facilitation is not able in the M case to completely restore the normal function, even if excitability improves very significantly. A shorter interval under facilitation means, as has been said several times, less asynchrony. By means of facilitation, the interval is reduced to a little less than half the interval in the inactive state.

For subject T, an arrangement of color isopters intermediate between the normal case and the M case is found; thus, there is a trend toward a more normal color function. There is no photo-heterochromic interval for yellow and blue or it is insignificant and difficult to appreciate; only for white, a certain dyschromatopsic phase appears in the first moments of the sensation, already mentioned in previous pages. In addition, given the difficulty in differentiating blue from green, there may be something like a dyschromatopsic phase in the former.

By examining, as in the previous cases, the temporal meridian of the right eye (of worse vision in subject T) using the same illumination in the perimeter as in the previous tests, the boundaries obtained for the chromatic intervals in the visual field are shown in Table 7.3.

Table 7.3. Degrees of visual field for the boundaries of photochromic and photo-heterochromic intervals in the right eye of subject T, using colored tests of 1 cm in diameter.

Color	Minimal simple luminosity	Minimal color sensation
White	42°	37° fleeting traces of green; 34° pure white
Red	39°	..... 32° pure red
Yellow	29°	..... 22° pure yellow
Green	29°	..... 21° pure green
Blue	21°	16° green; ..... 12° pure blue

Color isopters for this case are shown in Fig. 7.10. Their positions only undergo some alterations without reaching such important inversions as in the M case. In subject T, only a single photo-heterochromic interval can be considered (in the blue color) since in white, chromatopsia is extremely fleeting and unstable. In addition, photochromic intervals are very small compared to subject M under facilitation; for example, for the red-green pair.

The anomaly in color isopters lies in the fact that yellow is inside the red and at some distance from it, that blue is the most internal and that white is very close to the red although white is the most peripheral. There is, then, a tendency to a certain inversion of color isopters since the yellow isopter is more constricted than the red isopter, and the blue isopter more constricted than the green one, whereas the isopters for the yellow-blue pair are more peripheral than the red isopter in a normal case.

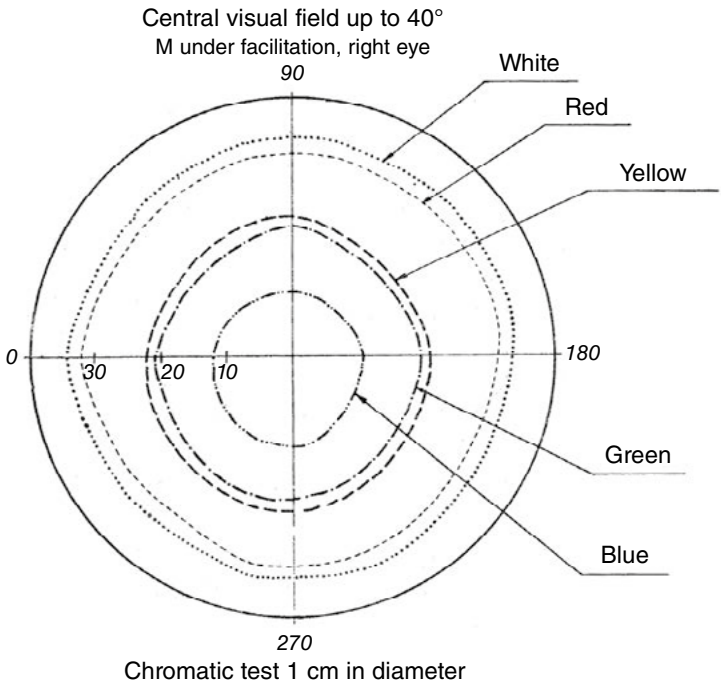

Figure 7.10. Color isopters in subject T. Abnormalities: the yellow isopter is more constricted than the red isopter, and the blue isopter is the innermost.

Such alterations in color isopters, from total to moderate inversion, are clearly in relation to the excitability characteristics in each case.

All these results suggest certain considerations about the theory of functional duplicity of the retina. This theory postulates a different functionality for the two types of receptors in the retina, cones and rods. Cones are ascribed to diurnal vision, especially to colors, and rods to night vision (Parinaud 1898, Kries 1905). The Purkinje phenomenon (Purkinje 1825) is invoked as particularly demonstrative of that theory, and it is generally accepted that in low luminosity, vision is ensured by sensitive blue and blue-green rods, whereas in high luminosity, only cones excitable by long wavelength radiation would intervene.

However, the inversion of color isopters does not fit in any way with this theory of duplicity. Even in cases of less severe color disorders than those mentioned above, a large number of brain-injured patients had an inversion consisting of seeing red more peripherally than blue. Some sporadic cases can also be found in the literature, with no special comments added. In extreme cases such as subject M and the third brain-injured subject mentioned in the preceding pages, the red isopter is the outermost, much more peripheral than the isopter for white and all the others. In contrast, the blue isopter is the most internal even in cases of very moderate alteration. Such an arrangement should not be attributed to anatomical differences of the

receptors but rather to the differential activities of the brain centers that undergo a major change by varying their nervous excitability.

Normal color blindness at the periphery of the retina does not seem to be caused by simple anatomical changes in the receptor elements. Its origin is probably to be looked for in the functional characteristics of colors, from a global conception of color vision in relation to excitability phenomena.

7.4. ALTERATION OF CHROMATIC INDUCTION PHENOMENA

We shall complete the study of chromatic dynamics by examining the behavior of chromatic contrast phenomena, especially in the M case, and in its two main forms: simultaneous (*edge contrast*) and formation of afterimages (*successive contrast*).

Edge contrast presents in subject M the peculiar effect of a very important magnification. When a red and a green paper are presented to him together, strips of a more intense color appear in the edge zone. These edge strips are very bright and very wide; they are 22 mm wide at the red side and about 15 mm wide at the green side; widths that could never be reached in a normal subject. This effect is obtained without difficulty and quite rapidly. The same occurs with yellow and blue. Under the same conditions, a color-enhanced edge strip 12 mm wide is obtained at the yellow side, and 15 mm at the blue side. These colors, which the subject usually perceives as pale green, look almost normal in the enhanced edge zone, although greenish dyschromatopsia does not completely disappear.

By means of facilitation, a significant reduction in the increase of edge contrast is obtained, thus resembling the behavior of a normal subject. The width of the contrast strip is reduced to 1/5 of the previous value, and its saturation is also reduced. This singular behavior in edge contrast is known as typical of people with color weakness (or *anomalous*), in whom edge contrast acquires great intensity. Where normal people see only a narrow edge contrast, anomalous perceive wide areas of intense color; and whereas a normal subject needs a fairly long time for the edge contrast to appear, in anomalous it is almost instantaneous (Nagel 1907, Guttmann 1920, Koffka 1922, and others).

Subject M can be considered as suffering from color weakness, and in particular he would be tritanomalous given his weakness to blue-violet even under optimal stimulation conditions. In his usual state, he should really be considered as affected by *tritanopia* (blue-violet blindness), and since color blindness goes in pairs, blind to the blue-yellow pair. The T case fulfills the conditions of tritanomalous given his blue weakness, blue being the third primary color in the trichromatic theory. This tritanomaly is a very rare alteration and was reported by Engelking (1925).

Regarding the interpretation of the enhanced edge contrast, it could perhaps be explained by an increase in color fatigue, which would favor an increase in the complementary color, although more detailed research would be needed.

Concerning the successive contrast, i.e., the formation of afterimages, we find in the M case the disappearance of the negative afterimage at the expense of an en-

hancement of the positive afterimage which persists for a long time due to the slowness of the visual function.

If subject M stares at a red surface for a certain time and then immediately looks at a neutral surface, he does not see green, as would be normal, but continues to see red for a period of time. The same result is obtained with any other color. In all cases, it is impossible to obtain in any way the complementary color that forms the negative afterimage, and he always sees the same color he was initially looking at. If he is instructed to look at a yellow surface until yellowish stripes are generated on a green background, and then look at a neutral surface, the image remains the same, and after a while the yellowish stripes on green background disappear, the whole surface becoming green until the whole image disappears completely. By means of facilitation by muscular contraction, which accelerates the development of sensation thus considerably decreasing its persistence, a negative after image can be obtained although weakly developed.

Pötzl (1916, 1928) also found a chromatic induction disorder in occipital lesions altering brightness and color, especially in patients who being blind in the acute phase recover their vision. An increase in positive afterimages and a decrease in negative ones can then be observed, without the author providing more specific data on these phenomena.

Finally, the alteration of the afterimages suggests the hypothesis that such subjects could behave like the *eidetic* subjects of Jaensch (1923, 1930), that is, they would have a form of perception (without negative afterimage) that many children normally present, which leads them to take representations and perceptions at the same level, thus contributing to create a special state of fantasy; later, but before puberty, the negative afterimage appears, and perceptions are better distinguished from representations. In our subjects, the change suffered in sensory activity with the emergence of eidetism could be the cause of the distortion of perceptions, pseudoagnosia, autotopagnosia, exclusion of pathological defects, etc. In other patients, singular phenomena such as the phantom limb of the limb amputee, hemianopsic hallucinations, or the mental state of blind subjects by central lesion who assert good vision (Anton syndrome, of which we have studied some cases in brain-injured people), and many other manifestations of anosognosia (ignorance of one's own disorder), may be attributable to eidetic-type changes in the sensory structure. But all this is conjecture, and precise investigations would be needed before the mechanism of such phenomena could be determined. In any case we would have a precise physiological and experimental base for further elucidation.

8. Theory of color differentiation

8.1. COLOR DIFFERENTIATION ACTION

The two subjects studied here, as well as others whose degree of alteration is very diverse, show many types of color degradation which can be gradually classified from color *anomaly* to color *blindness*. This can already be exemplified with only the two cases, M and T, putting into play various summation effects (binocular effect, muscular effort, etc.). In the most favorable conditions of excitability, the color vision obtained corresponds to that of *anomalous trichromats* according to Kries (1899), Guttmann (1908, 1909,1920) and Nagel (1907), whose characteristics are: the times necessary to recognize colors are extremely increased, distinguishing between shades of a color becomes quite difficult, colors are not perceived in the periphery of the visual field but are seen relatively well in the center. Such characteristics are fulfilled very well by subject T who can see the three primary colors but he presents blue color weakness, and can therefore be considered as affected by tritanomaly, a rare anomaly so far. The same happens to some extent in subject M under optimal vision conditions (binocular effect and facilitation by muscular effort). But this subject in an inactive state already presents the characteristics of a tritanope (blue color blindness), a defect that includes the opposite color, yellow, without all the other colors being totally undamaged. Therefore, all kinds of transition forms are obtained, from normal color vision to the deepest deficit, but always based on the fundamental alteration of the third primary color according to the trichromatic theory.

Both tritanomaly and tritanopia are little known disorders, but this does not prevent them from being common forms of color alteration in lesions affecting the visual area. In several cases we have studied with slight disorder, what is characteristic is a certain blue-yellow weakness with a predominance of deficit for blue, as well as a certain inversion of color isopters with red being more eccentric than blue in the visual field. This form of alteration is, as stated, the result of changes in nervous excitability of colors.

A theory of color vision or a meticulous interpretation of all these types of phenomena cannot be expected here, where only the description of the outstanding manifestations and a general explanation of brain excitability conditions have a place.

Considering the facts observed from the point of view of the two most important color theories, the trichromatic theory of Young-Helmholtz (Young 1802, Helmholtz 1852) and the complementary pair theory of Hering (1874/1878, 1880), we find that these theories only partially agree with the phenomena studied. Let us take as an example the case of the white color. In the trichromatic theory it is accepted that white is the result of the excitation of the three primary colors; red, green and blue-violet. In the case of blindness to the third primary color, the fusion of the remaining colors cannot produce white, and a more or less greenish-yellow or pale green is obtained due to the fusion of red, green and the green from the altered yellow and blue, thus explaining chromatopsia perfectly. In this case, Hering's hypothesis that a visual white substance is responsible of the formation of all the grey series and white, cannot be sustained. If the activity of that substance were to decrease, grey would be obtained instead of white, but not a greenish hue.

A different situation is that of yellow. In trichromatic theory, yellow is a composite color obtained by fusing red and green. In Hering's theory, yellow is part of the yellow-blue visual substance, and both colors are altered simultaneously. In our subject M, there is no difficulty in thinking that yellow is a composite color since it appears degraded, it is seen as green, and therefore it is not primary. We also know that the type of blindness in M is predominantly to the yellow-blue pair, although the remaining colors are not totally undamaged. This paired blindness is the best argument in favor of Hering's theory, although it fails in relation to chromatopsia and white, and this is important.

In cases of slight alteration, such as the T case, the predominant alteration is found in blue; then there is no chromatic pair disorder, and Hering's theory is again questioned. The fact that the yellow-blue pair is perceived altered by the dyschromatopsia of subject M, i.e., with a green tint, is not in favor of these colors being simple nor of the yellow-blue substance of Hering. Thus, it can be said that the phenomena, in general, are more easily explained by the trichromatic theory, although it is necessary to appeal to the theory of Hering for the alteration of the chromatic pair. However, the third primary color, blue or blue-violet, also is altered by dyschromatopsia, which is not a fact in favor of this color being a simple or primary color as accepted by the trichromatic theory.

Each of the two main theories of color vision, which we consider sufficiently well known, is based on important facts and arguments, but both also face difficulties that neither of them has been able to overcome from their own perspective. In general, physiologists side with the trichromatic theory, and psychologists side with Hering's theory. Many attempts have been made to bring the two closer together by trying to involve different stages, both peripheral and central, in the chromatic process (Hecht and Shlaer 1936, Müller 1917, Piéron 1939, Troland 1920, 1921, etc.), giving increasing importance to the chromatic processes that should take place in the centers of the cerebral cortex. Now, it is clear that in view of the nature of the cases studied here, it is only at this stage of nervous centers that we should place ourselves,

and for the same reason the whole chromatic issue must be focused in this study on the alterations in the brain excitability of colors. Colors already present small differences in a normal individual with respect to reaction speed and color establishment, and these differences are pathologically amplified, the slower colors being more impaired and tending to be excluded.

Thus, blue (the third primary color of the trichromatic theory), being the slowest color in a normal person, is the first to be altered, so tritanomaly and, in more pronounced cases, tritanopia, should be considered typical forms of color disorder in brain lesions involving vision. As has been said, a characteristic of the alterations studied in our brain-injured patients is that the most complex functions are the most affected, splitting into partial functions and being reduced to simpler activities. This is mainly fulfilled for white, which is the maximum composite color in the Young-Helmholtz theory, since the three primary colors must participate in its formation. As for the other colors, it is observed that red and green are the only colors that remain unaltered, whereas yellow and blue are green dyed due to dyschromatopsia. Therefore, it can be said that red and green are the only two primary colors and would form the only elementary pair, whereas the other yellow-blue pair would be formed from the primary pair: yellow by combination of red and green, as assumed in the trichromatic theory, and blue from green with some other hue and in special conditions of luminosity. This pair, yellow-blue, would be composite colors resulting from the action of *color differentiation* in brain centers.

The conditions for color vision depend entirely on the excitability relationships established according to the degree of activity of the nervous centers. Red and green are the most stable colors because they have the highest reaction speed, and the other colors would be generated by progressive differentiation from them. Yellow, for example, would need the previous existence of red and green, with a certain degree of intensity. If this pair is diminished, there would not be enough chromatic contribution to originate the yellow color because the combination of red and green would only form a yellowish green that cannot be differentiated from the real green. Likewise, if blue is weakened, it can easily be confused with green which seems to intervene in its formation. In this way, these two colors, yellow and blue, appear little differentiated, rather degraded, adopting a tone of simpler colors that participate in their formation. If the excitabilities of colors are very close as in a normal individual, the colors that we can consider composite (yellow-blue pair) are well differentiated and easily formed, as well as white and grays. Instead, if asynchrony occurs, all composite colors succumb because their processing is incomplete, showing abnormal hues that reveal their composite nature.

If such an interpretation proves to be valid, it could be said that all the colors, more completely or incompletely, are developed from red and green which seem to be simpler. In short, it can be said that chromatic differentiation has its physical brain base in the degree of nervous synchronization of the nervous centers. This differentiation by means of the combination of simple colors would be more in line with the trichromatic theory than with that of complementary colors. Finally, color degradation according to the conditions created by asynchrony generates a dynamic reduction of color differentiation by excluding the more complex and less excitable colors.

8.2. THE PROBLEM OF COLOR PROCESSING IN THE BRAIN

Under the influence of the theory of brain localization, attempts have been made, using the anatomical-clinical method, to determine a localization for color vision within the occipital lobe, on the basis of subjects with occipital lesions who showed more or less intense color disorders. However, because of all that is indicated in the general part about the action of nervous centers and the dynamic conception of the brain, such a localization, which has never crystallized into something certain, is difficult to admit.

From our observations in many brain-injured people, and especially in the subjects studied here, it follows that any localization in areas of the cerebral cortex, excluding the marginal areas which are practically the projection pathways, seems impossible. Brain centers seem to act only as nervous masses that activate the receptors, without determining specificity of any kind. We have seen quite intense chromatic alterations in lesions of the left hemisphere, right hemisphere, and in zones both close and distant to the calcarine cortex. In all cases the intensity of the disorder depends on the amount of brain mass destroyed, provided that the lesions are not too far from the occipital lobe, since for a given intensity of injury, its effect on vision decreases as the distance to the visual projection cortex increases. In favor of excitability processes as a basis for color vision, and ruling out any kind of anatomical localization, we can only cite Stein (1928, 1930) who admits or supposes a chronaxic characterization of colors similar to that of the different tactile sensations. But this functional conception represents a rarity in the midst of the theories of anatomical localization which, being more or less diffuse, are widespread everywhere.

A rigorous research shows that there is no reason to think about localization of color or any other type of activity in the visual domain (form, motion, orientation, etc.), and we can only talk about the greater or lesser degree of organization and differentiation of the visual function in relation to the level of brain excitability. The central lesion dynamically reduces all sensory activity in the brain, and a dynamic reduction is established in the visual system by virtue of which the less excitable functions are the first to be altered, they tend to be delayed or totally excluded. This applies both to colors and to any other type of function as we shall see next. The alteration is therefore global and according to the excitability of each function.

Visual forms

9. Visual field

9.1. Concentric reduction

In this chapter, we shall study visual forms, a type of sensoriality that corresponds in psychological language to perceptions, that is, to the spatio-temporal organization of visual stimuli. We shall start with a matter of primary importance which is the structure and modifications of the visual field. Then we shall deal with spatial localization, which in our subjects appears under the remarkable phenomenon of flat color vision (or color irradiation). Finally, we shall address phenomena of greatest differentiation such as spatial discrimination (acuity), motion perception and conceptualization of complex forms and configurations of objects.

The study of the visual field in the cases presented here is very appropriate, given its simplicity and characteristics, to demonstrate the physiological disorder that leads to asynchrony and dynamic reduction. In our two cases as well as in the Schneider case of Goldstein and Gelb (1918), there is a visual field alteration of the type *concentric reduction,* whose different intensity shows the degree of brain alteration in each of these three cases; highly intense in patient M, of medium intensity in the Schneider case and moderate in patient T.

As already mentioned in the first part of this volume, the concentric reduction of the visual field poses a serious and unsolvable problem to the theory of brain localization of functions, within which there is no possible interpretation since it is impossible to imagine an anatomical disposition in the cerebral cortex that concentrically disturbs both eyes equally, being the lesion in only one hemisphere. Moreover, in this type of visual field alteration, it is characteristic that the brain lesion is located outside the visual projection area of the cortex, generally in the parieto-occipital region, i.e., at a great distance from the calcarine cortex or striated area. By contrast, the explanation of the concentric reduction is very easy and immediate within the dynamic conception we advocate, since it is only a dynamic reduction in its simplest form.

In the study of the concentric reduction in our two cases, using a white test of 0.5 cm in diameter at the perimeter, we find very different degrees of reduction.

Subject M in the inactive state presents a very intense reduction since the limit of vision only reaches up to 6° in the temporal meridian of his right eye, and up to 4° in that of his left eye (Fig. 9.1), whereas a normal subject in the same illumination conditions reaches up to 90°.

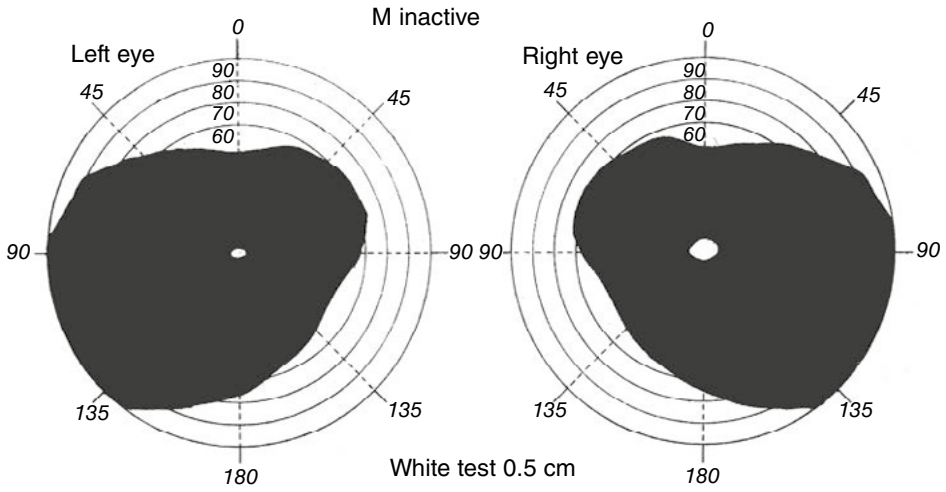

Figure 9.1. The visual fields of subject M in inactive state. White test of 0.5 cm in diameter. Note the intense concentric reduction, somewhat more pronounced in the left eye.

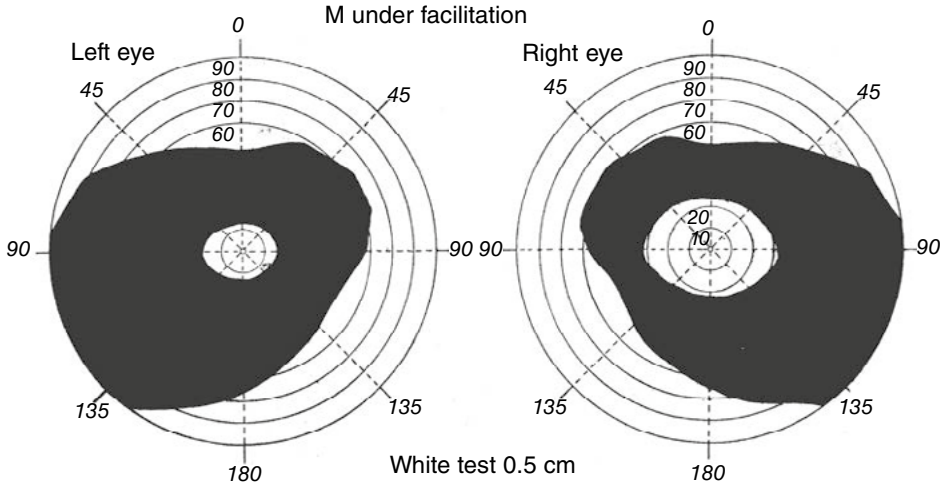

Figure 9.2. The visual fields of subject M under facilitation by maximum muscular effort. An enlargement of about five times with respect to the previous inactive state is obtained.

Therefore, vision is reduced to macular vision approximately. In the study of color isopters described above, the limits are wider because a test of 1 cm in diameter and greater perimeter illumination were used to properly investigate color manifestations. For a proper comparison with a normal individual, the limit of vision is determined not by the simple sensation of luminosity produced by moving the test from the periphery, but by a vision that provides a more defined sensation which in M corresponds to the appearance of a greenish tint on the white test. This occurs at 6° with the 0.5 cm diameter test and at 10° with the 1 cm diameter test, always using sufficient illumination for a normal subject to reach 90° with the 0.5 cm diameter test.

The concentric reduction is not perfectly circular but the visible field maintains a somewhat elliptical shape, i.e., the area of vision extends a little more along the horizontal meridian. This significant reduction does not mean that peripheral areas are absolutely blind. As said, it is enough to increase the diameter of the test to achieve a wider area of vision. Thus, by sufficiently intensifying the luminous stimulus, the area of vision is extended to the entire visual field even if the subject is free of any facilitation, as we shall see later. Therefore, the disturbance is relative and there is no absolute blindness in the rest of the field. Excitability decreases rapidly from the center to the periphery of the field in a normal case, and the brain lesion causes a general disturbance in the field that given its functional structure, the more peripheral the area the more pronounced the disturbance.

If facilitation is applied by means of a strong muscular contraction of the whole body, the increased brain excitability leads to an enlargement of the visual field of about five times that of the inactive state. For the right eye, for example, the limit of the field of vision is increased from 6° to 30° (Fig. 9.2). This effect of facilitation on the visual field was already found at the end of 1939, when the action of muscular contraction was discovered and applied to the investigation of a large number of functions. However, the enlargement obtained does not completely eliminate the field reduction because this type of facilitation is not capable of restoring brain activity *ad integrum*, as we know from other examples. But somewhat larger enlargements of the visual field can be obtained by combining several types of facilitation (muscular effort, binocular effect, etc.), although we shall now consider only the above effect for simplicity. It is necessary to point out that the enlargement of the field by means of facilitation, with respect to the inactive state, increases as the intensity of the stimulus used decreases. Thus, for example, a stimulus perceived in the inactive state at 6°, can be perceived at 30° (five times higher) with facilitation, whereas another more intense stimulus perceived in the inactive state at 12°, can be perceived at 35° or 40° (only about three times higher) with facilitation. Therefore, enlargement through facilitation decreases as stimuli become more intense, as we shall see in the corresponding examinations below. In general, for stimuli normally used in the visual field examination, magnification of the field through facilitation is four to five times the value in the inactive state.

As regards the second case, subject T, the concentric reduction is much more moderate, as can be seen in Fig. 9.3. The field of vision can reach up to 50° with the 0.5 cm white test, the field being slightly larger in the left eye than in the right eye, and showing an elliptical contour as in the above case. However, visibility is dimin-

ished in this case by the presence of a small *annular scotoma* which was much more widespread at the beginning of the examination of this subject. This type of scotoma results from the special dynamic conditions of the concentric reduction. In the examination with the perimeter, moving the test from the periphery to the center, the test becomes visible at a certain moment, and continuing slowly towards the center, the test is no longer visible because the subject's vision has become fatigued due to testing in weak areas. But by continuing towards the center, the test object is seen again due to the stimulation of increasingly sensitive areas. The zone in which vision is interrupted is the scotoma, which appears in all meridians giving rise to an annular scotoma, fully functional, dependent on the conditions of examination in an unstable and easily fatigued field.

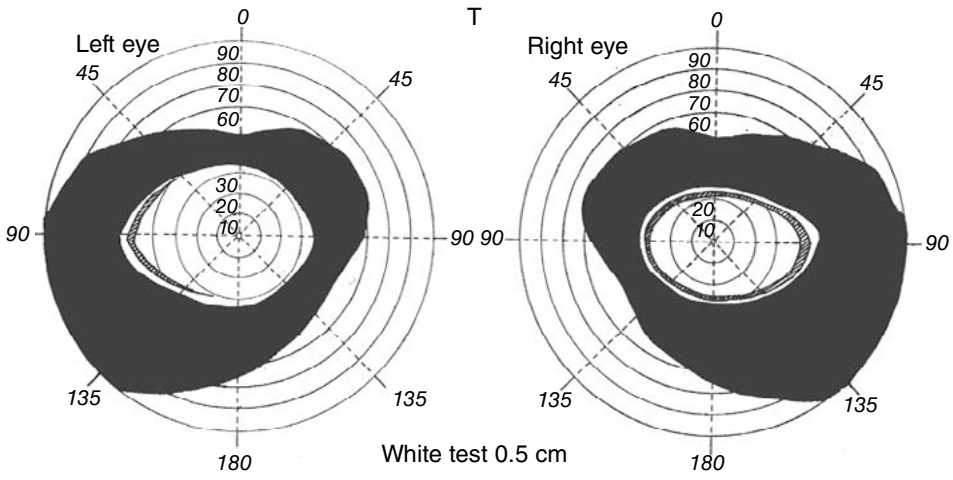

Figure 9.3. The visual fields of subject T. Compare with Figs 9.1 and 9.2. Note the small annular scotoma in right eye and an incomplete one in left eye (the better eye).

Goldstein and Gelb (1918), in a series of brain-injured people from the First World War, with visual alteration of the type concentric reduction of the visual field, were the first authors to give a rational explanation of the annular scotoma. Previously, these scotomas had been verified on different occasions, but without a plausible explanation they were wrongly admitted as a hysterical manifestation. This erroneous belief also occurred in the first observed cases of concentric reduction of the visual field, although the enormous number of subsequent observations in brain-injured people led that view to be abandoned. In other publications, we shall deal with different types of annular scotomas that occur along with other dynamic visual field alterations such as polyopia, macular deviation, etc.

As we already know, the T case offers the possibility of a small facilitation, so it is possible to obtain a small magnification of the visual field which demonstrates precisely the weak effect of facilitation on this subject, also due to the larger size of

the field in the inactive state. Thus, in the case of a quite peripheral vision, the enlargement of the visual field by facilitation would be minimal.

Finally, Fig. 9.4 shows the visual field of the Schneider case of Goldstein and Gelb (1918). It can be seen that the degree of concentric reduction is between that of the more severe M case and the less severe T case. It can also be said that the M case with facilitation is less severe than the Schneider case. Hence, it would be easy to deduce the intensity of the disorders in the Schneider subject for all types of visual functions and for any other sensory system. The above-mentioned authors have described only a very small number of disturbances and with a very different interpretation, as indicated in the general part of this work.

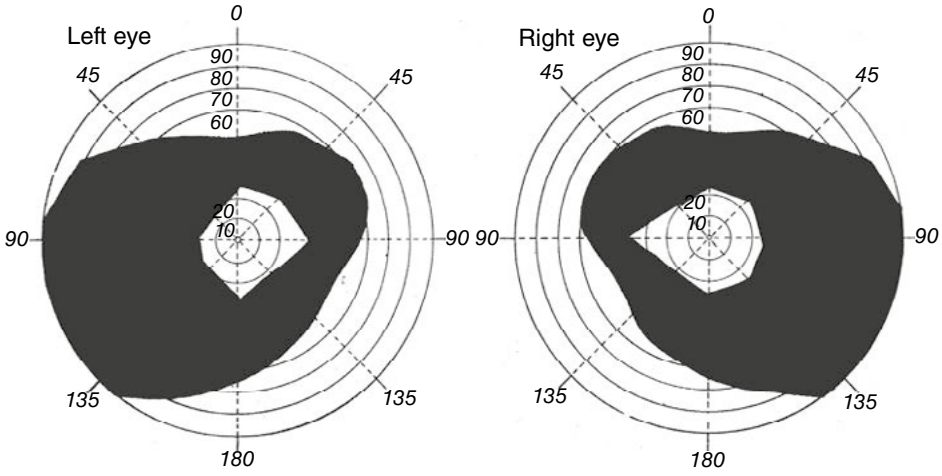

Figure 9.4. The visual fields of the Schneider case of Goldstein and Gelb (1918). Concentric reduction to an intermediate degree between that of the M and T cases. Subject M under facilitation can be similar to the Schneider case.

9.2. EXCITABILITY RELATIONS IN THE VISUAL FIELD

We shall now deal with the experimental research of the concentric reduction of the visual field by applying the dynamic analysis (fundamental experiment, synchronization by facilitation, etc.). The tests to be performed are easy since they are limited to examining vision with the test object along the perimeter in different conditions of stimulation: varying time, intensity, facilitation, etc. However, this simplicity of analysis is hampered by the irregularities that peripheral vision may present. The patients sometimes have great difficulty in keeping the eye stationary looking at the central fixation point, and easily look at the peripheral stimulus. In addition, we must expect the appearance of peripheral fatigue, changes in attention, etc. For these reasons, these examinations can be very painful, and must be prolonged in time and repeated sufficiently until usable data are obtained. It is not realistic to expect a com-

pletely exact match between different examinations, but only approximate, as there is always some margin of variation, even in a normal individual.

First of all, it should be recalled that the visual field is not functionally uniform even in a normal situation, and all kinds of functions are quickly weakened from the center to the periphery. Figure 9.5, taken from Charpentier (1903), shows how the sensitivity of the retina decreases from the center to the periphery; it shows the additional illumination that must be given to the test object to distinguish it from the background. Sensitivity decreases very rapidly from 40°. It is therefore understandable that in an overall impairment of visual function, the peripheral zones of the visual field fail much more easily than the more excitable central zones, and thus a concentric reduction of the visual field occurs.

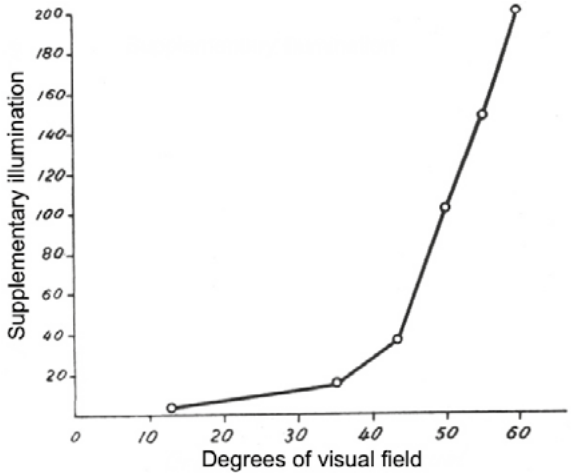

Figure 9.5. Additional illumination of the test object versus the degrees of the visual field. Sensitivity decreases strongly from 40° (Charpentier 1903).

Asynchrony affects all kinds of functions in such a way that normally less excitable functions are proportionally more disturbed. This explains that the weakest functions are excluded (dynamic reduction), and can only be recovered by extremely high intensity stimuli.

Due to this asynchrony, the already existing excitability differences in the normal individual undergo a considerable enlargement, as we have mentioned above regarding the photochromic interval in colors. An example of this enlargement of the normal interval is shown in Fig. 9.6, which shows the intensity-duration curves obtained with stimulation at 0° and 20° of the visual field, for the right eye of subject M in the inactive state, using a white test object 1 cm in diameter. In addition to the large difference (interval) in the rheobase level, the different curvature of the curves indicates the different reaction speed, much lower at 20° of the visual field. By applying facilitation by muscular effort, the mentioned interval decreases considerably and is reduced to approximately one quarter, but it is still very large compared to the value of the nor-

mal excitability interval under the same experimental conditions. As the interval decreases by means of facilitation, the corresponding curves show a more pronounced curvature, increasing the reaction speed as indicated above in other experiments.

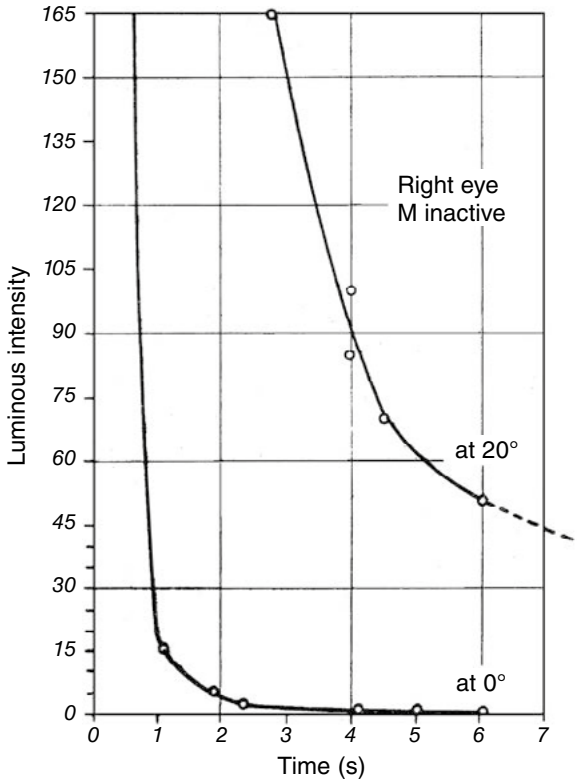

Figure 9.6. Curves of luminous excitability (intensity-duration of threshold stimulus) at 0° and at 20° of the perimeter using a white test object 1 cm in diameter, for the right eye of subject M inactive. There is a considerable widening of the normal interval.

The great decrease in excitability at 20° that subject M experiences in the inactive state is understandable given the intense concentric reduction, so to achieve vision in this zone, very intense and long-lasting light stimuli are necessary compared to the more central zones. If these types of intensity-duration curves are determined for a series of even more peripheral degrees, a bundle of curves is obtained which, compared with the analogous bundle using facilitation and with that of a normal subject, would provide a complete description of the enlargement of the various intervals. But now we must restrict ourselves to simpler relationships since such determinations would mean a huge task in this kind of brain-injured subjects, and such comparisons can be obtained much more simply as we shall see later.

Another excitability relationship to be considered is the one existing between the time elapsed until the sensation is obtained and the perimeter degrees where a stimulus

of certain intensity is placed; thus, the reaction speed in different regions of the visual field can be obtained. In the previous figures on the concentric reduction of the visual field, it is not entirely correct to directly compare normal vision with that in pathological subjects M, T and Schneider, since at the limit of peripheral vision, the time required to obtain sensation is very different in these cases. Therefore, to correctly establish the comparison, it is important to note that at the peripheral limit, whereas a normal subject takes only a few tenths of a second to perceive the stimulus, subject T needs a minimum exposure of about two seconds, subject M under facilitation needs four seconds or more, and about seven seconds if he is in an inactive state.

The increase in time, i.e., the loss of reaction speed, is not exclusive of the most peripheral areas since the excitability disorder, being global, is present in all regions of the visual field, as shown in Fig. 9.7 for a given stimulus along the horizontal meridian.

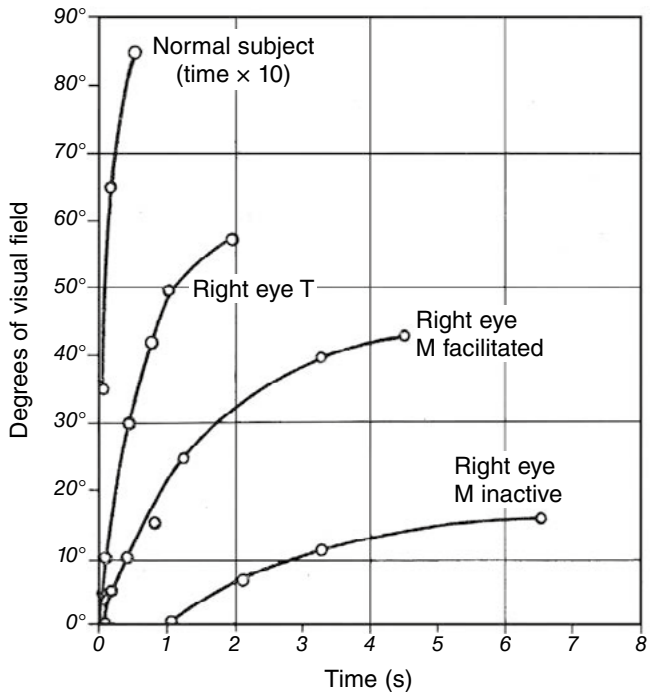

Figure 9.7. Reaction time in the perimeter for the right eye of: subject M inactive and under facilitation, subject T and a normal subject, using the same stimulus in all cases. The time for the normal subject is multiplied by 10 in order to indicate it in the graph.

For subject M in the inactive state, the speed of reaction is very low along the meridian that he can see: at 0°, about one second of exposure is needed, and at about 13° (the limit of the visible field), six seconds are needed. By contrast, under the action of facilitation by maximum muscular contraction, a remarkable economy of time is achieved with the same stimulus, as well as an enlargement of the visible zone. However, he is still very delayed compared to subject T, and the latter is still very

delayed compared to a normal individual, as shown in Fig. 9.7. These tests show us how the excitability in the visual field decreases from the canter to the periphery in each case, expressed by the decrease in the slope of the curves as well as by the useful time at the limit of peripheral vision.

As indicated, the intense concentric reduction in the M case, and the more moderate reduction in the T case, do not mean absolute blindness in the remaining visual field; it is sufficient to properly intensify the light stimulus to reach vision in the most peripheral regions, even if the subject is free of any facilitation. That is, an increase in stimulation is sufficient to compensate for the large decrease in excitability. This is shown in Fig. 9.8, where the degrees of peripheral vision are related to the intensity of the stimulus needed to produce sensation in the various cases we have studied.

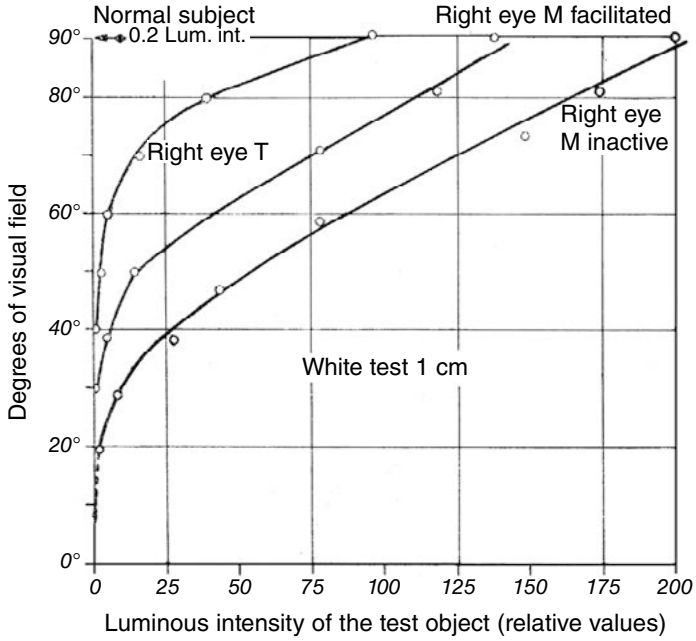

Figure 9.8. Degrees of the visual field of the right eye versus required stimulus intensity to produce sensation, for three types of excitability: subject M inactive, subject M under facilitation by maximum muscular effort, and subject T inactive.

As in Fig. 9.7 above, the slope of the first part of the curves in Fig. 9.8 indicates the visual field excitability status. The curve for the T case presents a greater slope, thus with a moderate increase in the stimulus intensity, the most peripheral zones of the field are easily reached. By contrast, the increase in stimulus intensity in the other two cases (M inactive and facilitated) needs to be very large, especially in subject M inactive, which needs a significant stimulus intensification from 30° onwards. As for M with facilitation, he occupies an intermediate position between T and M inactive, showing a saving of stimulus intensity, with respect to the inactive state, much higher at low intensities than at very high ones. Such curves would correspond

to the recruitment of the rheobase levels of the intensity-duration curves at different degrees of the visual field (Fig. 9.6). The necessary values of the stimulus in the most external regions of the field are extraordinarily high in the case of M inactive, reaching, for 90° of peripheral vision, about one thousand times the stimulus in the normal subject. However, with facilitation, about six hundred times or little more is enough. As mentioned several times, the normally less excitable functions undergo a proportionally much greater deficit than other activities of less physiological demand. For this reason, there is a progressive increase in the functional deficit from the center to the periphery of the visual field, as shown in all the tests carried out. This explains the origin of the concentric reduction of the visual field, without this meaning a complete abolition of the extreme peripheral regions but only their lack of excitability in the face of ordinary stimuli of moderate intensity.

Finally, we must also consider how the peripheral visibility of a stimulus of constant intensity increases by increasing the intensity of the facilitation applied by means of muscular effort. For this purpose, subject M must stand facing the perimeter which is placed at a sufficient height, with his right eye looking at the central point. Once the outer limit of vision is reached without any muscular effort, the enlargement achieved is determined as a function of the weights held by the subject, in the same way as described in the experiments on electrical excitability of the retina. The results obtained are shown in Fig. 9.9, which shows two curves for test objects of 0.5 cm and 1 cm diameter respectively.

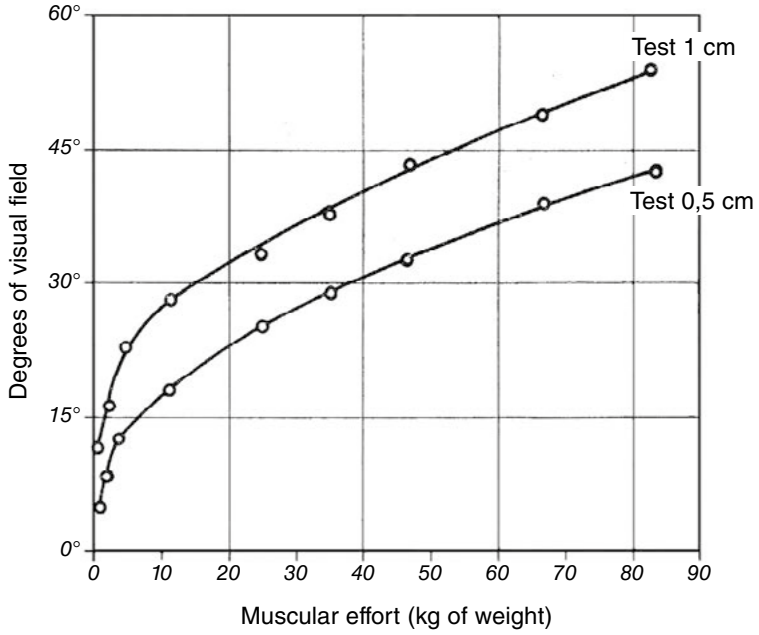

Figure 9.9. Enlargement of the visual field by facilitation. Degrees of the visual field of the right eye of subject M as a function of the muscular effort measured in kg of weight held by subject M, with white tests of 0.5 cm and 1 cm in diameter.

As in the curve showing the voltage saved by facilitation (Fig. 4.4), the visual field increases rapidly with facilitation at the beginning of the curve, and then more and more slowly. In proportion, the smaller test leads to a larger increase. Thus, the limit (in degrees) of the visual field becomes about four times larger for the 1 cm test object, whereas it becomes about seven times larger (extremely high value) for the 0.5 cm test object.

The experimental data in the curves are the average of several determinations, since the conditions of the experiment (fixation at the central point and attention to the peripheral vision, in addition to the considerable effort to hold very high weights) do not allow very precise assessments. However, they are good enough for our current aim of graphically expressing the course of the visual field enlargement as a function of facilitation by muscular effort.

These facilitation curves are similar to those shown in Fig. 9.8 on recruitment of peripheral degrees by intensification of the stimulus luminosity, but in this facilitation experiment, the stimulus remains unchanged. The fact that it can be perceived in increasingly peripheral areas because of an increase in their sensitivity, is due to the central summation produced by the muscular effort.

9.3. VISUAL FIELD ORGANIZATION

The visual field represents a functional unit which is globally impaired in the central disorder studied here. Only superficial and incomplete determinations can lead to the idea that only peripheral regions are altered in concentric reduction. There is an alteration in excitability throughout the visual field, but the more peripheral the regions, the greater their deficit. Thus, we can consider a dynamic reduction in which excitability is modified throughout the entire functional field, although with the indicated degree of diversity. There is an increase, in diverse degree, of the excitability differences (intervals) that already exist from one region to another in a normal individual.

In relation to the unitary functional character of the visual field we can cite the observations by Poppelreuter (1917). This author points out that there are no absolute scotomas in the visual field as a consequence of cortical lesions, and it is sufficient to increase the intensity of a luminous stimulus to obtain signs of vision in regions where they would otherwise appear blind. In concentric reduction, the field is enlarged by increasing the luminosity of the test object. This author also found that the "healthy" half in hemianopsia does not remain absolutely undamage, and may show a certain concentric reduction or at least a reduction of color isopters. There is thus a functional unit in the activity of the visual field. However, more specific considerations on excitability or more varied experimental analyses are not mentioned by this author.

Other phenomena, which we are merely mentioning, also reveal the dynamic peculiarities of the visual field, i.e, the interdependence between its parts. Such phenomena are monocular *polyopia,* macular deviation (*pseudofovea*), *autokinetic* phenomenon, *metamorphopsia* (distorted vision), etc., all of which are manifestations we have been able to observe in the subjects studied here and in many others. Very remark-

able and of great theoretical importance is monocular polyopia, which we have studied for the first time in an injured subject with parieto-occipital lesion at the end of 1936, later in some other cases, and in 1938 we determined the physiological laws of the phenomenon. This allowed us to collect a large number of cases with brain injuries and lesions of other nature. The cases described of polyopia are very few in total, having been reported by very few authors (Goldstein 1939, Quensel 1927, Pötzl 1933, Hoff and Pötzl 1935 a, etc.). However, although very few patients describe the disorder spontaneously, when attention is paid to certain circumstances of the visual field impairment, the phenomenon can be brought to light in a large number of cases in a constant and regular way. Some hypotheses about the occurrence of the phenomenon have been formulated by Pötzl (1933), and more positive interpretations by Goldstein (1939); however, these interpretations are not sufficient to explain the most complex variants of polyopia that we found. For the moment we shall only indicate that changes in the localization of stimuli occur, and a single stimulus in monocular vision may cause multiple localization due to an anisotropic visual field configuration.

Polyopia in the M case is shown to be related to the intensity of the luminous stimulus; the weaker the stimulus the more intense the monocular polyopia, and facilitation reduces it. With a stimulus of suitable intensity, the following three types of vision are obtained in subject M as well as in many other subjects:

1) The test object in central vision gives rise to triplopia; the real stimulus is seen in the middle, and somewhat less intense virtual images are seen on both sides.
2) In peripheral vision, only diplopia (double vision from a single stimulus) is obtained, with the virtual image less peripheral than the real image.
3) In a paracentral region at a certain distance from the central point of the visual field, a single image is obtained from a single stimulus, so there is a region without polyopia.

In subject T, polyopia is not very pronounced at present but it was very pronounced when this patient was studied for the first time in 1938, allowing us to unravel to a large extent the mechanism of this phenomenon. At present, the virtual images in this subject are reduced to faint shadows; apart from this, he complies with the general rules set out in the previous case.

The phenomenon of macular deviation can be easily studied in subject T due to the greater extension of his visual field. Central vision, with maximum visual acuity, can be moved to about 15 degrees under certain experimental conditions that we cannot detail now. Fuchs (1922), a psychologist like Gelb and collaborator of Goldstein at the Brain Injury Research Institute during World War I, described the formation of a pseudofovea in the healthy half of the visual field of hemianopics. We found in 1938-1939 that, without the need to have hemianopsia, many subjects with concentric reduction of the visual field show a certain deviation of the foveal vision that does not correspond to the anatomical fovea, this deviation being much more pronounced in certain experimental circumstances. Thus, the entire functional layout

of the retina is altered, relocating its functions (colors, luminosity, acuity, etc.) to regions that do not normally perform them.

We can also mention metamorphopsia (alteration of the shape of objects) and the autokinetic phenomenon consisting of the apparent motion of motionless light stimuli. This last alteration was initially extremely pronounced in subject M, and currently is only minimally displayed by means of low-intensity stimuli. All these peculiar manifestations of the visual field, along with other cases, will be exposed in specific publications. We only mention them here to emphasize the functional complexity and nervous dynamism exhibited by the visual field, which seems to be a simple and static structure.

10. Flat colors. Visuospatial localization

10.1. FLAT COLOR VISION

In addition to the color sensory disorder described in previous pages, our subjects present a singular chromatic spatial alteration: *flat color* vision. This is a color irradiation[1] that makes objects appear wrapped in a colored atmosphere; thus, when an object is grabbed or a colored surface is touched with the hand, the subject has the visual impression that his hand is sinking into the color. Colors therefore do not seem to be adhered to the surface of objects, the vision of a colored surface is then lost and, in its place, a color irradiation appears that tends to blur the relief and contour as well as the orientation of the surface, the color appearing on a frontal plane (flat color vision).

This disorder is extremely pronounced in subject M, and was one of the first notable phenomena found in this subject in the summer of 1938. It is also present in subject T but less markedly so according to his less severe brain lesion.

This disorder was first described by Gelb (1920) in the work entitled *Über den Wegfall der Wahrnehmung von Oberflächenfarben* [On the loss of surface color perception], when he was working with Goldstein at the Institute for Brain Injury Research. In this work, the loss of surface color was studied in two subjects with a parieto-occipital lesion. They presented flat color vision in accordance to the research of Katz (1911/1930/1935). From then until now, although some authors have dealt in passing with somewhat similar disorders (especially color irradiation), the flat color disturbance has not been researched. Gelb's descriptions are entirely from the psychological point of view. But here, in our subject M, who presents a much more intense chromatic spatial alteration than the two cases of Gelb, we shall address the disorder in a direct and simple way on a strictly physiological basis, showing that flat color vision is the result of a visuospatial localization disorder. Something similar occurs also in touch in these subjects, and could be called flat touch or irradiated localization

[1] As stated in the footnote to Sec. 6.1, this term must be understood as a diffuse spatial localization of the color of an object.

due to the loss of precise localization. As said, both sensory systems are disturbed to the same degree in the central syndrome, therefore, in the case of spatial localization, the alterations that we now study in the visual system will be found again in a more or less similar way in the tactile system.

As for the patients of Gelb (1920), in addition to flat color vision, they also presented a concentric reduction of the visual field, a decrease in acuity, less adaptation to light, color weakness and severe visual agnosia. It should be noted that the type of visual field alteration is sufficient to assign the central syndrome to these patients. That is to say, they are types of brain disorder of the same type as that of our two subjects and subject Schneider, and as for their intensity, already Gelb indicates that they show a much less pronounced agnosia than that of the Schneider case studied by this author together with Goldstein (Goldstein and Gelb 1918, 1919). Indeed, in the most pronounced case of flat color vision, the concentric reduction is somewhat less intense than in Schneider, and can be equated with that of subject M under facilitation by maximum muscular effort. Thus, that case shows a much more intense brain alteration than that of our second case, subject T. Since these patients of Gelb can be included in the central syndrome, they would have all the characteristics studied (alteration in all sensory systems in both halves of the body, asynchrony, permeability to summation, etc.). Flat color vision is only one aspect of the visual system disorder, and this in turn is a part of the brain disorder that necessarily involves other sensory systems.

Concerning the interpretation of this spatial color disorder, other authors (Pötzl 1925, 1928, Stein 1928, 1930, Kleist 1934) besides Gelb (1920) have discussed it without achieving a simple and satisfactory explanation. In general, the disorder is assumed to be dependent on both a sensory impairment of color vision and an associated apperceptive visual agnosia; all interpretations being made from a psychological standpoint, and discarding the possibility of finding a physiological basis for the disorder. In this brief study, we shall prove that the pathological flat color vision results simply from a disorder in visuospatial localization, more specifically, from the loss of the normal precise localization, which is replaced with a diffuse or irradiated localization, in the same way that we will have the opportunity to study it in the tactile system.

10.2. SPATIAL LOCALIZATION DISORDER

Normally, colors and luminosities occur under various spatial aspects. When colors are adhered to objects and surfaces, they appear as *surface colors* with a defined location. In contrast, there are other situations such as the colors in the spectrum, the blue of the sky, etc., that show a less precise spatial location, they appear to us as in a plane, hence their *flat color* character (Hering 1880, Katz 1911/1930/1935). When this characteristic increases, they acquire an aerial character, and flat colors become *spatial colors* appearing as colored masses or fogs. As already mentioned, the pathological disturbance in our subjects lies in the fact that surface colors (with well-defined location) are lost, and it is not possible to perceive them other than as flat colors.

10.2.1. Properties of the pathological flat color vision

In Gelb's patients, the darker the colors were, the more swollen they became, and this also happens in our two subjects, especially in subject M, who will be our main reference. When colors are detached from objects, they show a soft character that makes them penetrable, thus, when touching a colored surface, the hand sinks into the color and gets tinged with it. Following the nomenclature used by the patients of Gelb, colors can be classified into *thick* and *thin* according to their penetrability; thus, the colors less luminous "irradiate" much more than the clearer ones. The former, besides easily tinting the objects in real contact with them, produce a sensation of certain softness to the touch, whereas thin colors tinge much less and are harder to the touch. In the M case, red is the thickest and softest color because it "irradiates" to the maximum, and white, which is perceived as light green, is the one that irradiates least. In between these two colors there are all the others regarding their irradiation capacity.

The color detached from a piece of colored cardboard is perceived on a frontal plane even when the cardboard is tilted with respect to that plane, unless the tilt is quite pronounced. The tilt necessary for this frontality to be lost depends on the color of the cardboard. Thus, red tolerates deviations of the cardboard from the front plane much more than yellow or white.

The concept and recognition of objects and forms is altered due to the disorder of irradiation, although they are already intrinsically altered in our subjects as we shall see later. The detachment of colors from objects tends to suppress the relief and contours of them, making their form more uniform and simple.

When writing with a pencil on white paper, our subject M has the sensation that the tip of the pencil is sunk in the light green of the altered white. The tip is as if impregnated with color, which being light color, its irradiation (delocalization) is very weak. If the pencil strokes are thin, they disappear under the irradiation, and no difference with the background is perceived.

Subject M has the most intense color irradiation anomaly known so far, which decreases considerably under very strong illumination. In ordinary illumination and at the accommodation distance, the irradiation is so manifest that even under the most intense facilitation it cannot disappear from the less light colors such as red, instead it disappears for the other colors. In the other subject T, irradiation is very small and only pronounced in red.

10.2.2. Irradiated[2] localization

The above phenomenology depends on the conditions of color irradiation, which varies greatly with excitability, and this in turn depends on the particular color, its illumination, exposure time and facilitating action.

When subject M is shown cards of different colors, and is asked to sort them according to the intensity of irradiation, he does it as follows (from highest to lowest

[2] As stated in previous footnotes, this term is used to indicate a type of spatial delocalization.

irradiation): red, green, blue, yellow and white. All these colors irradiate upwards or towards the subject who is looking at the card, and also towards the sides, although somewhat less. With regard to black, which looks as a dark, somewhat greenish fog, he has the sensation that instead of irradiating upwards, the color is as if it were sunken, i.e., irradiating towards the bottom, and irradiating in a slightly greater proportion than red. This distal character of a black surface's irradiation may be a secondary subjective alteration, and if we discard this peculiarity of black, it can be stated that the degree of irradiation depends on color luminosity.

Frontal irradiation is much more intense than lateral irradiation, the latter being about one third of the former. By measuring the irradiation of colors according to the indications of subject M at a distance of 25 cm and in medium illumination, the values obtained are shown in Table 10.1.

Table 10.1. Spatial irradiation of colors in the M case in the inactive state, using colored cards 20 × 20 cm.

Color	Frontal irradiation	Lateral Irradiation
Red	6 cm	2.5 cm
Green	4.5 cm	2 cm
Blue	3.5-4 cm	2 cm
Yellow	2.5 cm	1.5 cm
White	2 cm	0.8 cm

Under these conditions, and applying facilitation by means of maximum contraction of the entire musculature, irradiation decreases considerably to about one fifth, resulting in a red irradiation of only about 8 mm.

A little more irradiation is perceived using only one eye than using both eyes, but in these tests the differences are small, and the most noticeable change is obtained by going from the inactive state to the facilitated state.

Subject T presents this anomaly to a much lesser degree, and only perceives irradiation when looking at the object with the right eye (the one with worse vision). Irradiation intensity in the T case is approximately similar or somewhat lower than in the M case under facilitation. Subject T also sorts colors according to their irradiation, similar to M, from highest to lowest as: red, blue, green, medium gray, yellow and white. As he sees blue much more intense and saturated than subject M, he places it after red. As for black, he insists on placing it between green and medium grey. It should be noted that the irradiation appears only to the right side of the visual field, as it happens with green chromatopsia, since the right half is somewhat more amblyopic. In binocular vision, the irradiation practically disappears due to the mutual facilitation between the two eyes. As said, irradiation is perceived mainly in the right half of the visual field of the right eye, and the more peripheral the vision, the greater the irradiation, which causes that a completely flat red surface is perceived as quite concave on the right giving the sensation that it is raised by that side.

We already know that of the chromatic series that includes white, subject M only perceives red, and all the other colors are perceived as greens of varying lightness: he sees blue as a pale green, yellow as a paler green, and finally, white as the palest green. Therefore, we can say that the lower the lightness of a color (i.e., the more saturated it is), the larger the color irradiation will be; hence the larger irradiation is obtained for red. Likewise, irradiation occurs in subject T for whom the most saturated colors (least pale) are red followed by blue. We must therefore admit as a general rule in these subjects that the spatial localization of the different colors is altered according to their lightness. According to Katz (1911/1930/1935), a factor such as the intensity with which attention is awakened may be involved in the irradiation perceived; but according to what is said above, such a factor should be the intensity of color saturation and not its lightness. It should be noted that when a normal subject looks at the spectrum, he already perceives a more aerial character at the more saturated ends of the spectrum, red and violet, whereas this character is minimal at yellow.

Color irradiation changes significantly depending on the stimulation conditions. It is enough to move away a piece of colored cardboard, for irradiation to increase progressively both frontally and at the edges, a fact already observed by Gelb (1920), although he mentions it briefly without giving it any more importance. It is clear that in this case there is a decrease in the intensity of the stimulus, thus the disorder of spatial localization becomes more acute, i.e., irradiation increases. This change can be observed both by moving the colored surface away from the subject or by diminishing the illumination of the colored surface while it remains at the same distance. In this last case, varying degrees of irradiation then arise successively: almost normal surface color vision is obtained under very strong illumination, color irradiation (flat color) is obtained in medium illumination, and aerial color with a very diffuse spatial localization is obtained in very weak illumination. In this last stage, it is no longer possible to distinguish the frontal from the lateral irradiation, since everything looks like a colored aerial mass much larger than the real colored surface. By diminishing the illumination even further, the color, which had already become fainter, disappears completely and is replaced by a kind of dull fog that fills the entire visible field, such fog being much larger than the preceding colored mass.

In this way, a joint variation in the intensity and spatial extent of the color is seen when the stimulation intensity changes. When the colored surface is strongly illuminated, its color is perceived intensely and as a surface color, and when the illumination decreases, it appears more and more weak and with a flat character, and finally spatial or aerial, as spherical. At this point, we can only consider the volume of the color, so it is not possible to distinguish any differentiation or spatial organization, the localization has been completely lost, and this happens when the color is about to disappear completely and be replaced with a simple colorless fog more or less dark. It is important to note that in flat colors, the lateral and frontal irradiation zones show a less intense color tone than the real colored surface.

Even in medium illumination, if the exposure time is very short (one second), a strong irradiation (i.e., spatial color) is obtained in the M case, and equally in the T

case but to a lesser degree. If the exposure time is prolonged, the irradiation decreases significantly. Therefore, it would be possible to determine excitation (strength-duration) curves corresponding to the different levels of visuospatial localization: one curve for spatial color, another for flat color and another curve for surface color. Thus, we have different types of spatial localization: from the complete loss of localization in aerial (spatial) color to the total localization in surface color, passing through an intermediate state of diffuse or irradiated localization corresponding to flat color.

Under facilitation by maximum muscular effort and in ordinary illumination of colored cards, irradiation disappears only in the lightest colors: first in white, which even without facilitation presents very weak irradiation, and also in yellow, etc. Instead, a small irradiation always remains in red, which only tends to disappear under strong illumination and facilitation. In average illumination and in the inactive state, when small objects are placed on a red paper, the frontal irradiation in which they are immersed makes them almost disappear. If facilitation is then applied, the irradiation decreases considerably, and the objects become visible because the *thickness* of the color decreases.

Due to the different irradiation of different colors, changes in shapes or configurations can be perceived, depending also on the stimulation conditions. For example, let us take two partially overlapping squares, one red and one white (Fig. 10.1) with the white over the red. In weak illumination, subject M sees a full red square on the white one, that is, the corner of the red square completed by irradiation covers the corner of the white square that irradiates much less, as shown in Fig. 10.1a). If the illumination is high enough, the subject perceives then the real configuration, and the white square appears complete [(Fig. 10.1b)], covering the corner of the red square, since the weak irradiation of red in strong illumination does not achieve to complete the shape of the red square. These changes are also obtained by means of facilitation, without altering the illumination; thus, in weak illumination, if the subject is in an inactive state, the red square is seen as complete, and under facilitation it is seen incomplete and the white square complete.

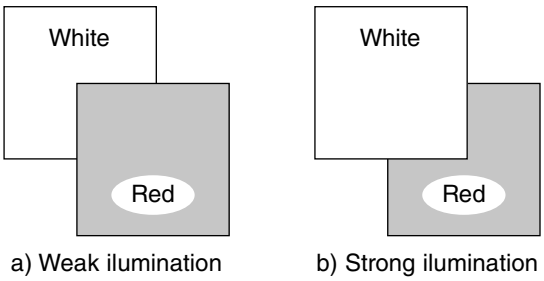

Figure 10.1. Completing the shape of the red square by irradiation (see text).

The best way to complete the shape under the indicated conditions is by combining a strong irradiation color with a very weak irradiation color, provided that the area to be completed is not excessively large. Then, the stronger irradiation color

tends to complete the shape of the square because this shape is more regular, of greater meaning or pregnancy[3] according to Gestaltists, than the configuration based on right angles that results from covering the corner of the square.

Since irradiation alters spatial localization, it also modifies the perceived size of objects. By moving the object away or decreasing its illumination, the diameter of the test object tends to diminish as the lateral irradiation increases. This is because the irradiation has a weaker luminous intensity that allows it to be distinguished from the innermost real area. When illumination is quite low or the distance very long, the flat color is lost and evolves to spatial color, disappearing any distinct perception of the object and remaining only a diffuse irradiation over an area much larger than that of the object.

10.2.3. Asynchrony between simple sensation and spatial localization

As already mentioned, pathological flat color vision is only the effect of an irradiated spatial localization caused by the failure of the normal localization. The latter can be achieved by intensifying the visual stimulus (e.g. by increasing light intensity). For this reason, light colors produce much less irradiation than more saturated colors (less whitish) under the same illumination conditions. For all colors, irradiation is weakened under strong illumination, and also by facilitation which reduces the asynchrony (or separation) between simple visual sensation and its spatial localization. Irradiation (diffuse spatial location) occurs in both vision and touch since the characteristics of this functional disturbance are the same for both sensory systems. In both systems there is a remarkable interval between minimal visual or tactile sensation and spatial localization. Weak stimuli that in a normal situation are always perfectly localized, are left without localization in our subjects, giving rise to gusts of tactile sensation without spatial character, or to aerial colors in vision. In more intense stimulation, a diffuse (irradiated) localization occurs, which is a rudimentary undefined spatial localization and occupies a certain area. Finally, in very intense stimuli, the normal defined and precise localization occurs. Irradiated localization in touch extends not only on surface but also in depth; the subject has the sensation that the stimulus reaches the deep layers of the skin, in the same way as in frontal irradiation of flat colors, which alters the perception of depth.

Spatial localization, being a more complex function that requires a greater physiological demand than simple sensation, is altered in our subjects in a much greater proportion, and is therefore delayed presenting a much higher intensity threshold. Then a disaggregation occurs, and alterations appear such as flat color vision, etc., alterations that we shall return to study quantitatively in a more precise way when dealing with tactile functions. Thus, flat colors result from a color-spatial asynchrony (disaggregation) leading to an abnormal interval. This disaggregation is governed

[3] Pregnancy (Prägnant in German) or "good form" is a concept introduced by the Gestaltists, and refers to the quality of the visual forms that capture the attention of the observer because of their simplicity, balance, concision, accuracy or stability of their structure.

by a mechanism common to all activities: excitability deficit but in greater proportion in the more complex ones.

We have also observed some weak irradiation in other brain-injured people with reduced visual field or other alterations in the visual field, the irradiation being especially towards the peripheral regions of the visual field, as in the T case. In a normal individual, traces of flat color can also be noticed in the outermost zones of the visual field. During the phase of recovery from the disorder in the two cases of Gelb, the irradiation disappeared first in the central zone and, much later, it was attenuated in the peripheral zones.

As a complement, it is interesting to mention that in a normal individual, pathological flat color vision appears under the effect of mescaline (Beringer 1923, 1927), a drug that significantly decreases brain excitability. In this case, colors seem swollen and penetrable, wrapping objects in an atmosphere of color. However, the transient conditions of intoxication and the irregularities that occur are not as conducive to research as brain lesions, which show a much more constant and uniform symptomatology.

11. Visual form perception

11.1. VISUAL ACUITY

In the study of visual forms, we include visual acuity, visual perception of motion, and perception of object shapes and visual configurations.

Visual acuity is a complex function of spatial discrimination (minimum separable) of stimuli, and already includes aspects of shape perception. Visual acuity is extraordinarily altered in the two cases we are studying, especially in the M case. Due to the diffuse localization of visual stimuli by the irradiation mentioned above, it is easy to understand that spatial discrimination between stimuli is very difficult. A rather poor function can only be expected under intense illumination or by facilitation through muscular effort or other types of summation. In the usual clinical tests using optotypes, the visual acuity values obtained in subject M for different types of vision are shown in Table 11.1. Very different degrees of acuity are obtained depending on the types of vision. These types are determined by the facilitation due to muscular effort, by the joint action of the two eyes facilitating each other, and also by the small difference in vision between the two eyes. By combining the two types of facilitation, i.e., that of maximum muscular contraction and that of binocular vision, considerable increases in acuity are achieved, which can be as much as six times the value of the weakest eye in the inactive state.

Table 11.1. Visual acuity in both eyes of subject M in full sunlight.

Type of vision in subject M	Visual Acuity
Left eye, inactive state	1/25
Right eye, inactive state	1/16-1/20
Left eye, facilitation by muscular effort	1/16-1/20
Binocularly	1/10
Right eye, facilitation by muscular effort	1/8-1/10
Binocularly and muscular effort	1/6-(1/4 ?)

If the illumination is lower than that used for the results of Table 11.1, but still sufficient to examine a normal individual, the acuity values in subject M decrease significantly, to more than half of the above acuities. Under intense illumination (as indicated in Table 11.1), it is still possible to obtain further improvement in acuity if, in addition to the binocular effect and maximum muscular contraction, the subject also receives intense light over his own eyes. Then it is easy to reach acuity of 1/4. If he is additionally told to make vigorous movements of his head, limbs, etc., 1/3 is reached. In this case, several types of facilitation (binocular vision, muscular effort, movements, intense light on both the test object and on both eyes) are combined. When determining visual acuity with the letter test or other optotypes, if the test is not seen with sharp vision, the test is perceived to be somewhat tilted due to the orientation disorder that leads to inverted or tilted vision. In sharp vision, the test object is seen in the correct position, but if the test object is smaller, it is perceived as both blurred and tilted, and so on for even smaller test objects. The T case behaves like the M case, but as the brain deficit is less intense, the various acuity values, shown in Table 11.2, are much more favorable.

Table 11.2. Visual acuity in both eyes of subject T in good illumination.

Type of vision in subject T	Visual acuity
Right eye	1/3-1/2
Left eye	1/2-2/3
Right eye, facilitation by muscular effort	2/3
Left eye, facilitation by muscular effort	2/3-1
Binocularly	2/3-1
Binocularly and muscular effort	1

Subject T behaves similarly to subject M, for example, with the right eye, the one with weaker vision, optotypes corresponding to visual acuity 1/2 are seen sharp and correctly oriented, whereas those corresponding to 1/3 can be recognized and read, but are somewhat blurred and tilted. At the beginning of the examination of this subject, his visual acuity was 1/8 to 1/6 or even less. Later, some general recovery occurred, and his visual acuity improved significantly. As for the Schneider case of Goldstein and Gelb (1918), acuity values reported are essentially variable from one test to another, since three different ophthalmologists found different data; one found normal acuity, another found it was somewhat reduced, and the third gave 1/10. It is probable that the last one was the most accurate, that value being between that of subject M and that of T. It must be noted that the acuity values in the Schneider case can be affected by his characteristic head contouring movements which, as we know, are merely central summation phenomena, contrary to the assumptions of the mentioned authors. If such movements are missing, the acuity can be very poor, a fact pointed out by the authors who consider the patient to suffer from apperceptive visual agnosia, i.e., inability to perceive shapes.

In ordinary life, such wounded people behave with much better visual acuity than one would expect from clinical trials, especially the more severely affected subject M. We have already seen the big difference between using only one eye in an inactive state and using binocular vision along with facilitation by muscular effort. In fact, in binocular vision and good illumination, some muscular tension and small movements are sufficient to obtain an acuity within fairly usable limits. As is known, visual acuity in a normal individual is already markedly influenced by illumination, growing proportionally to the logarithm of luminous intensity. The curves of acuity as a function of illumination for the two subjects we are studying have the same general characteristics as for a normal subject, but with very different values according to their different degree of brain alteration. We determine for central vision, the minimum separable as a function of illumination, and we obtain visual acuity by taking the reciprocal of the minimum separable.

In the curves of the minimum separable (Fig. 11.1), it can be seen the considerable difference between the values of the pathological cases and that of a normal subject, for a given intensity of illumination. The more pronounced the nervous excitability deficit, the greater the difference. The bending of the curves indicates the speed in the functional variation; the more pronounced the brain disorder, the lesser the speed.

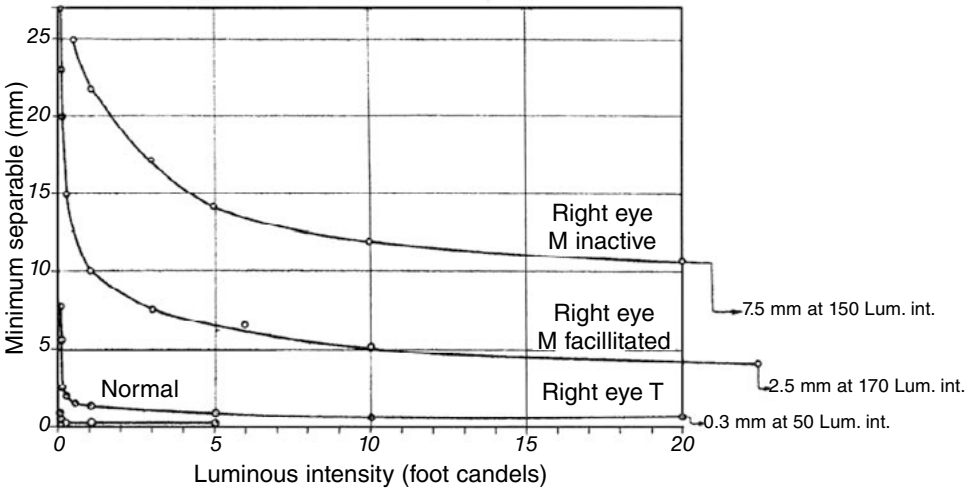

Figure 11.1. Minimum separable as a function of luminous intensity, in central vision of the right eye of M inactive, M under facilitation, T inactive and a normal subject.

Taking the reciprocal of the minimum separable, the corresponding curves of acuity as a function of luminous intensity are obtained. They are shown in Fig. 11.2, where some corrections have been made to show all the curves in the same graph. It can be noticed in these acuity curves that, for the luminous intensity at which the normal subject reaches acuity 1, the pathological cases show extremely low acuities that only improve very slowly along the curve. Extremely high intensities are necessary to achieve a more favorable discrimination.

Finally, taking the logarithm of the illumination, more complete curves of the variation of visual acuity in the different cases are obtained (Fig. 11.3). For the right eye of subject M inactive, the acuity increases up to 1/25 for maximum illumination, and up to 1/12 applying facilitation by muscular effort. Subject T inactive, in strong illumination but much lower than in the previous case, reaches an acuity of 1/2. Also noteworthy is the different luminous intensity at the lowest values of acuity in the three pathological functional variants (M inactive, M under facilitation and T inactive). The curves of Fig. 11.3 tend to become straight lines, and for the middle values of acuity it can be said that acuity grows proportionally to the logarithm of illumination.

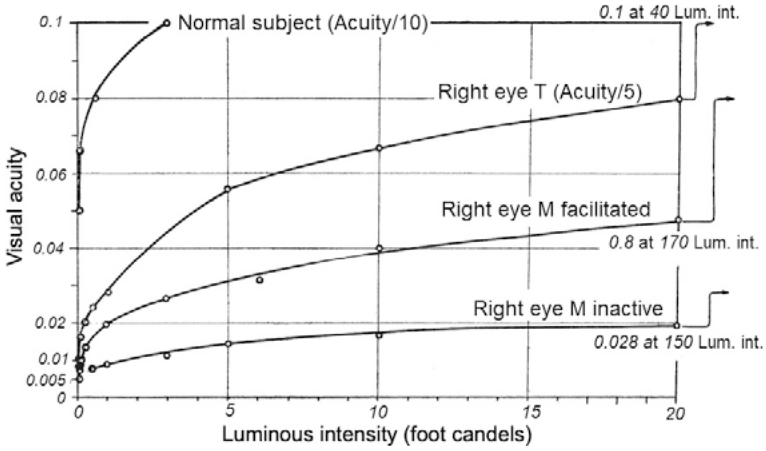

Figure 11.2. Visual acuity as a function of luminous intensity. Acuity is here the reciprocal of the minimum separable (see Fig. 11.1). The acuity of the normal subject has been divided by 10, and the acuity of subject T by 5, to represent them on the same graph.

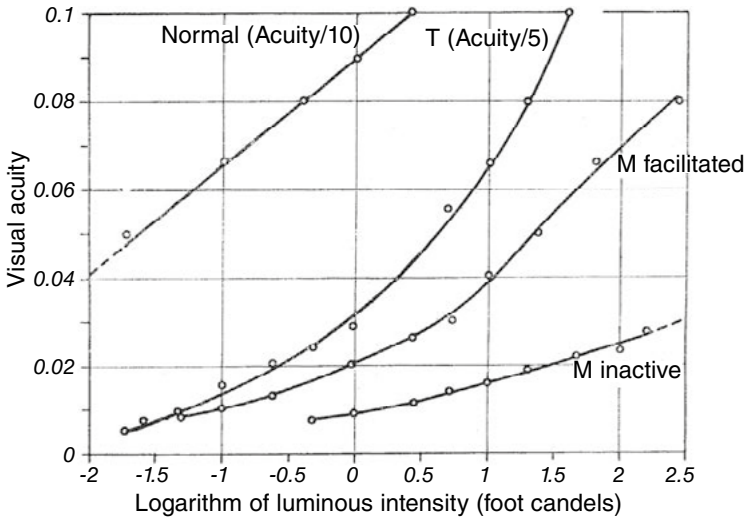

Figure 11.3. As in Fig. 11.2 but taking the logarithm of luminous intensity.

These curves give a clear account of the status of the acuity function. In a normal subject, the variation is rapid even for small light intensities. In the pathological cases, especially in M, the growth of acuity is very slow, and the curve for M inactive begins at high intensities due to the deficit of excitability in the lower threshold and also in the differential threshold, the latter due to an enlargement of the normal interval by the asynchrony already discussed.

If visual acuity is determined not only for central vision but also along the meridian of the visual field, with constant illumination, the so-called Wertheim curve (Wertheim 1894) is obtained, which shows that the acuity deficit extends to the entire visual field, as was to be expected. In subject M, not only the acuity corresponding to foveal vision is very low, but an equally intense decrease also occurs in somewhat peripheral zones, the curve ending very soon due to the concentric reduction of the visual field. A diagram of the behavior of the Wertheim curve in correspondence with concentric reduction of the visual field and with electrical excitability of the retina in subject M, inactive and under facilitation, is shown in Fig. 11.4. This highlights the functional dependence of acuity and visual field on the state of brain excitability. Such functionality must be assessed from a physiological point of view, dismissing the idea of preformed anatomical structures.

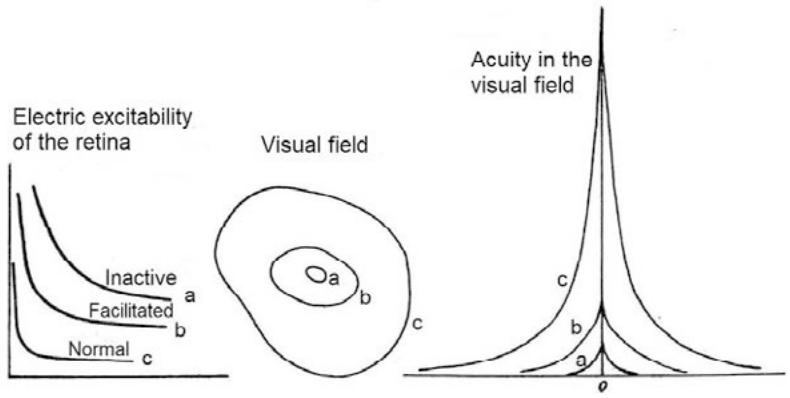

Figure 11.4. Schematic representation for the M case, compared to the normal one, of the functional correspondence between the visual field (center), the acuity in the field given by the Wertheim curves (right side), and the state of excitability of the retina (left side). a: inactive; b: facilitated by strong muscular affort; c: normal case.

The mere fact that visual acuity in a normal individual depends so much on light intensity proves that acuity cannot simply be inferred from the anatomical arrangement of the sensitive elements in the retina (separation between cones, etc.). Helmholtz (1867/1896) and later Hartridge (1918, 1922) suggested that visual acuity could be inextricably linked to intensity discrimination. Therefore, visual acuity must always be put in relation to the type of stimulation, or rather to the states of brain excitability.

It is understandable that in the above-mentioned tests and determinations, visual acuity (or minimum separable) is hampered in subject M by the pronounced spatial irradiation described in previous pages. In fact, when this irradiation decreases under strong luminous intensity, acuity improves, i.e., the minimum separable decreases. The fact that visual acuity is reduced so considerably in these subjects, even in very intense illumination, is due to the character of the sensory disorder, which, being global, appears more pronounced in functions of greater differentiation. In short, there is an enlargement of the excitability intervals between the different acuity levels, leading to the exclusion of the highest levels (of highest acuity).

11.2. ALTERATION OF VISUAL MOTION PERCEPTION

The loss of visual perception of motion was one of the first singular phenomena that subject M showed as a result of a careful examination, shortly after the finding of his inverted vision. It was precisely this alteration of visual perception of motion, already observed in 1938, that led us to search for some resemblance between M and Schneider. This type of disturbance is extremely rare, and cases of this type clearly detected to date would only be the Schneider case of Goldstein and Gelb (1918) and a much earlier case of Pötzl and Redlich (1911). The patient studied by the latter authors was a woman with an occipital lesion who, instead of perceiving the motion of an object, she perceived a series of objects; a sort of decomposition of the motion into successive, static images of the object along the path. In the Schneider case, the disorder appeared differently, and the subject did not see moving objects, but perceived them either here or there, that is, at the departure and arrival points, lacking the sensation of displacement. When this singular alteration was found in subject M, he behaved very similarly to subject Schneider, but lacking at that time an appropriate understanding of the brain disorder, it was not possible to advance in the investigation of the phenomenon. In the two above-mentioned cases, the authors did not go further either. In the earliest case, the authors merely stated the disorder, and as for the Schneider case, the authors related the loss of visual perception of motion to apperceptive visual agnosia, searching for a primary basis for all this in the failure of the *transverse function* of Wertheimer (1912), whose proof of seeming motion was not obtained in this subject. This is perhaps the only time that Goldstein and Gelb got close to a physiological interpretation of the phenomena, although they did not go beyond suggesting it. In general, it can be said that the studies of these authors do not leave the field of psychology.

Subject M in binocular vision, at accommodation distance and in ordinary illumination does not perceive the gestural movements of a person in front of him such as winking, rapid opening and closing movements of the hands, etc. He does not perceive any change, which is understandable considering the brevity of these movements and his slow reaction time in all sensory processes. However, he can perceive these movements under facilitation by muscular effort, although normalization is not complete since, for example, very rapid winks may still be excluded from his perception.

Subject T behaves much better in the visual perception of these types of movements, and it is necessary to resort to fast and very short movements to demonstrate that there is also a certain alteration in motion perception.

Visual perception of motion, like all other types of functions, is altered in such a way that it depends on the conditions of stimulation. For example, the motion of strongly illuminated moving stimuli can be perceived, whereas others of lower luminous intensity, which in a normal situation are perceived quite well, are totally excluded for subject M. In the case of low illumination, if subject M perceives motion, the speed and path length are altered. If a moving stimulus of suitable luminous intensity is observed from very near, its motion is perceived although only in the central region of the path. However, if it is observed from a slightly greater distance, any perception of motion disappears due to the strong decrease in light intensity according to the inverse of the square of the distance, besides the fact that the angle of vision is reduced.

All these variations are studied in a very simple way by means of a metronome, placing a small white disc of 1 cm in diameter at the end of the pendulum that moves according to the chosen frequency of oscillation. The regulation of the luminous intensity of the moving disc allows us to study quantitatively the excitability relationships for the perception of motion by subject M.

Under the indicated experimental conditions and with metronome oscillations of 1 beat per second, subject M in an inactive state and using only the right eye perceives the motion decomposed into various sensory phases of different excitation level, as shown in Fig. 11.5. The main phases from the simplest to the most complex are: sensation of a motionless point in the middle of the path between two beats; sensation of signs of motion only in the most central zone of the path and with reverse direction of motion; finally, sensation of a larger path and normal direction of motion.

Since the time between two beats is 1 second and the path is about 10 cm long, the average speed is 10 cm/s. To perceive a single movement from one side to the other (duration 1 second) at a distance of 25 cm, very intense illumination would be necessary, and only a sensation of motionlessness is obtained even at high intensities, as shown by the lower curve in Fig. 11.5. The small disc is perceived static in the central zone of the path. Furthermore, it is devoid of any color, only a motionless light is perceived.

A second phase appears with signs of motion if the observation is prolonged. Thus, in less than two seconds, a sensation of motion appears at high luminous intensities, as shown in the intermediate curve of Fig. 11.5. If the illumination is reduced, it is necessary to increase the number of oscillations sufficiently to maintain the initial perception of motion. This intermediate curve of kinetic sensation is higher than the previous one, and is less concave, corresponding to a sensory phase of higher functional level. As for the concomitant color changes of the test object, when the slightest sign of motion appears, the white disc may still be colorless, but immediately afterwards it shows a dark greenish tint. The perceived motion in this phase is limited to the most central zone, showing a very short path and much greater speed than the actual motion, thus the speed is overestimated. Moreover, this motion

presents the important peculiarity of being seen in the reverse direction. This is easy to verify since the speed of the metronome pendulum is slow enough, and there is more than enough time for the subject to verbally indicate the direction of motion between two beats. The reversal of motion is due to the special asynchrony in the brain mechanism of visual orientation; thus, weak stimuli are seen in the opposite direction, and stronger ones in the correct direction. This will be discussed in subsequent chapters.

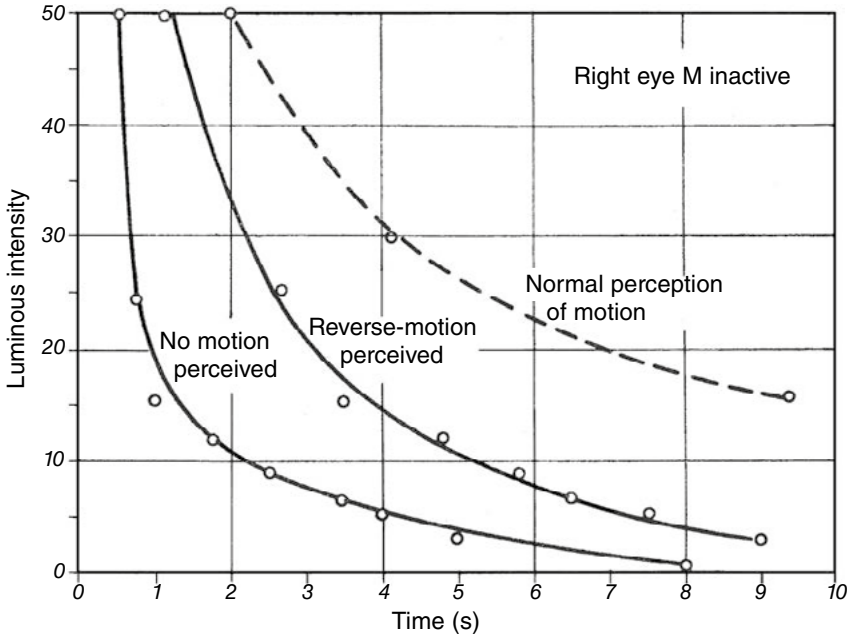

Figure 11.5. Intensity-duration threshold curves in the perception of motion by the right eye of M inactive. Test object: white disc 1 cm in diameter at the end of the metronome pendulum, at 60 beats per minute and at a distance of 25 cm from the observer. Lowest curve: only sensation of motionlessness, and in the middle of the path. Intermediate curve: very short motion with reverse direction. Dashed curve: larger and slower motion with correct direction.

Finally, it is still possible to obtain a third phase by appropriately increasing intensity and time of observation, which results in a motion perception closer to normal. In this phase, the perceived motion is of much longer path, slower, in the correct direction, and the color of the test object (the disc) becomes pale green with a certain amount of white. This last phase is the only one that resembles the motion perception by a normal individual, although it is still far from being identical. In fact, the amplitude of motion is smaller and the speed somewhat greater than for a normal subject. For a complete identification with a normal subject, it is necessary to apply facilitation, in which case the perceived motion reaches all the amplitude, becomes a little slower, and the disc appears completely white.

These experiments indicate that visual perception of motion is extremely altered in the M case. However, motion can be perceived under special conditions such as high luminous intensity of the moving object, or also at lower intensities if the motion is observed for a sufficiently long time. For this reason, the motions that occur in ordinary life necessarily go unnoticed because they are below the threshold of necessary excitation since they are brief motions, and even in good illumination they are too brief to produce any effect. It is therefore necessary to increase brain excitability by facilitation in order that motion can be perceived in some way.

The curves in Fig. 11.5 refer to vision in a single eye and in the inactive state, and similarly could be obtained for the facilitated state, in which case the separation (or asynchrony) between the phases would be much less. When the subject in the inactive state is in the first kinetic phase, it is sufficient to apply facilitation by muscular contraction to jump to the third phase or to a more normal stage. If the subject is in the second phase (slight sign of motion and inversion), facilitation action gets much more easily the change, i.e., re-inversion, path enlargement and speed reduction. This speed-reducing effect, which could be considered paradoxical, is not at all so if we consider the nature of the excitability disturbance. Because of the shorter perceived time, the motion seems faster. However, if perception is more favorable, either by simply increasing luminous intensity or by facilitation, the velocity decreases. Signs of this paradoxical effect are already present in a normal subject, thus for a constant speed motion, the perceived speed is higher when illumination decreases; and the same motion seems faster in peripheral vision than in central vision according to Exner (1886), which must be explained by a lower excitability in the peripheral regions of the visual field.

In the tests with metronome, in addition to facilitation by muscular effort, other weaker types of facilitation can be revealed, such as those produced by sensory stimulation of touch or hearing. Thus, when the subject is in the akinetic phase, a loud whistle next to his ear is sufficient for the motion to appear immediately, disappearing as soon as the sound stops. Likewise, by brushing the subject's back smoothly and with some speed, a similar effect on motion perception is obtained. The action of these facilitations is much less intense than that of strong contraction of the entire musculature, but in this type of experiment where there is little separation between the akinetic phase and that of signs of motion, these facilitations are sufficient to bridge the abnormal interval by means of central synchronization. Thus, an intersensory summation is obtained.

In relation to the luminous intensity of the kinetic stimulus, the effect of the different colors should now be considered. The same as discussed previously with regard to visual localization of colors in relation to their different spatial color irradiation according to their luminosity, is now applicable to visual perception of motion. The lighter a color, the better motion perception. For this reason, motion perception is easier with a white test object than with a red one. In this regard, the following test can be performed: a white test is placed on the metronome at a rhythm and distance suitable for reaching the threshold of minimum motion perception; motion is no longer perceived when viewed through a red glass, but is perceived again when the glass is removed.

As for subject T, since he presents a much smaller brain lesion and a correspondingly smaller alteration in excitability, he behaves normally with respect to motion perception in ordinary life, and only if illumination is very low, a deficit in his visual perception of motion can appear. In the metronome, very fast oscillations are necessary for them to cease to be perceived. Whereas a normal subject can very well see a side-to-side path of 1/3 of a second duration, subject T fails completely when illumination is very low, with both the right eye (the worst) and the left eye. He may need three or four side-to-side paths in monocular vision or two in binocular vision to perceive motion. But this only happens in very low illuminations in which subject M would not even perceive a motionless stimulus. In the T case there are also changes in the perceived velocity according to the conditions of excitability, for example, in binocular vision and facilitation by muscular effort, the perceived motion is slower than that perceived only by the right eye and in an inactive state.

With regard to the Schneider case of Goldstein and Gelb (1918), the degree of his motion perception disorder is, as in all other functions, intermediate between that of the more severe M case and the milder T case, and must conform to all the conditions described for these cases. Thus, it is possible for him to perceive motion if the stimulation conditions meet certain requirements of sufficient intensity and exposure time. There is therefore no absolute loss of visual perception of motion as thought by the authors mentioned.

The origin of the disorder we are studying is the same as for other functions: because of asynchrony, activities of higher physiological demand are less accessible to ordinary stimuli, and can only be awakened by more intense and prolonged excitation.

In the M case, for example, even in monocular vision and inactive state, a moving electric lamp observed at a short distance can be perfectly perceived even when the movements are rapid and of short duration. But apart from special cases like this, brief ordinary object motions under the usual much less intense illumination are discarded because of the extraordinary slowness of nervous reaction in this subject.

Stein (1928, 1940) already suggested that the loss of visual perception of motion must be related to an increase in sensory chronaxie, and we have experimental confirmation of this view in our cases. What in a normal subject is perceived as motion, is split into a series of stages in subject M: irradiated light, static localization, signs of rapid and inverted motion and slower and larger motion. The greater the deficit of brain excitability, the more different (or asynchronous) are the excitability levels of these stages.

As we shall see in the appropriate place, this alteration of the kinetic function occurs similarly in touch, both in joint movement and in the surface sensitivity when a stimulus moves over the skin. Likewise, a certain relationship could be established with the disorder of the perception of successive stimuli. The perception of motion not only implies perfect pinpoint localization, but also the ability to spatially discriminate between stimuli (minimum separable). It could then be possible to register a continuous series of very brief successive stimuli. This requires great sensory sensitivity; for this reason, motion perception disorder is one of the first and easiest to occur.

It should be noted that motion perception is not an independent function as some authors have admitted, but, as is easy to understand, it is part of the process of discrimination and sensory organization, constituting one of its most complex stages. In the language of the Gestalt, the disorder we have studied would be explained by a destruction of the structure of the *field*, by the loss of coherence and the failure of the *transverse function* (Wertheimer 1912, Benussi 1914, Witasek 1910, Koffka 1919/1935, etc.). But this is a consideration from another point of view that leaves the physiological problem of excitability untouched, and cannot directly address the quantitative characteristics of the disorder such as changes in intensity-duration curves, acceleration of motion, reduced trajectory, etc. In short, the usual moving stimuli are not perceived because they are too fast and too weak in luminosity in relation to the excitability characteristics of the subject, especially subject M.

11.3. VISUAL PERCEPTION OF FIGURE AND OBJECT SHAPES

In view of what has been exposed in previous pages about visual acuity, one could infer in some way the behavior of our two subjects in visual perception of figures and objects. A very remarkable pathological feature that should be highlighted here is what we call *metamorphopsic pseudoagnosia.* With this name we refer to a special alteration of forms and configurations due to more favorable meanings that arise spontaneously due to the lability of perception. Many examples can be mentioned about this type of disorder, but perhaps the simplest and most illustrative are found in the perception of letters. In the M case, it is frequent that a 'C' is perceived as an 'O' (shape totalization), a 'J' as an 'I' (simplification and symmetry), a large size 'a' as two superimposed zeros, and an 'S' as an '8.' It is not a simple alteration of visual acuity that changes shapes, but an instability in perception that tends to change the shapes into others of greater pregnancy (simpler and more meaningful) according to the laws of organization of the field of perception exposed by the Gestaltists (see footnote in Sec. 10.2.2). This type of alteration can considerably change the appearance of shapes and their meanings. For example, a calligraphic 'A' large enough to be easily recognized (Fig. 11.6) is perceived by subject M as an 'o' and an 'A,' as shown in Fig. 11.6. This disaggregation that occurs in this example is very typical, and is observed quite regularly in all kinds of somewhat complex figures. Also noteworthy is the difficulty in perceiving sets in a unitary way.

Figure 11.6. Alteration of the visual perception of the figures in subject M. The calligraphic 'A' (model) is perceived broken down into an 'o' and an 'A,' as seen in the copy.

By using geometric shapes cut out from cardboard, it is observed that perception is guided by the most striking details such as angles, sharp points, corners, etc., and much less by sides and surfaces, so normal consistency and unity is not achieved. This is the reason for the distinction between geometric shapes often fails, and different shapes appear to be equal when they are perceived simultaneously. For example, an oval and a circle 3 cm in diameter, or a circle and a square, appear to be equal to subject M when shown to him at the same time, but he perceives them correctly when shown separately. The perception of shapes is carried out in a diffuse and unstable way, being subjected to many circumstances that significantly alter the perception of shapes; in addition, the slowness in perception is considerable. This type of perception is common in the M case in the inactive state, in medium illumination and at the visual accommodation distance. Also subject T showed this kind of perception when he began to be examined in 1938 shortly after suffering the brain injury, but nowadays he does not show such a marked deficit, and only by conveniently shortening the exposure time he makes the same mistakes as M. Subject M does not make mistakes only if he appeals to facilitation, which not only greatly reduces the reaction time but also makes the above-mentioned phenomena of alteration of shapes disappear, thus the distinction between shapes can be made correctly. The various errors in shape perception by subject M in an inactive state never appear under facilitation by muscular effort.

In the Schneider case of Goldstein and Gelb (1918), the perception of shapes was completely different if they were shown in the tachistoscope very quickly or under ordinary conditions. In the first case, vision was blurred and shapeless, only diffuse color spots, whereas in the second case the perception and recognition of shapes was quite feasible. This difference is based on the different exposure time of the stimulus, and since his reaction time is increased, there is not enough time in the tachystoscopic exposure for the shape sensation to develop, being reduced to the primary stage of shapeless spots. However, the mentioned authors think that there is no possibility in tachystoscopic vision for the patient to make his characteristic head movements and, therefore, shape recognition is not possible. The movements of the head have effect although not in the sense admitted by the authors but by the central summation action as we know. Without any kind of movement, it is sufficient to lengthen the observation time enough, or to increase the illumination or the size of the object, for normal perception to emerge. For example, subject M under facilitation by maximum muscular effort can well recognize figures of a convenient size with good illumination in a short time (even tachistoscopically in 1/5 second), whereas in the inactive state he would need up to 3 or 4 seconds. But in medium illumination, he may need much more than 6 or 7 seconds to recognize very simple shapes that a normal subject recognizes in about 1/10 second.

In relation to shape perception, the behavior of these subjects is of interest in the face of geometrical-optical illusions that occur in normal individuals, but here, especially in subject M, they hardly occur at all. Thus, in the well-known figure of Müller-Lyer (1889) (Fig. 11.7), the optical illusion that the line segment with open ends seems longer is very difficult to perceive, and only after a long time of very careful attention is it perceived, although not steadily.

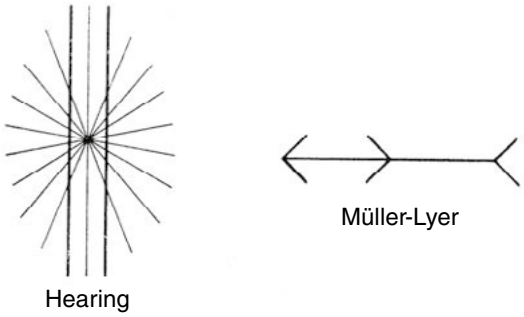

Figure 11.7. Geometrical-optical illusions of Hearing (1861) and Müller-Lyer (1889), not perceived by subject M in the inactive state.

In other more complex figures such as the figure of Hering (1861), the illusion of curvature of the parallel lines never occurs in the inactive state, but is very well perceived by means of facilitation, as in the other mentioned illusion or other analogous illusions. Geometrical-optical illusions do not manifest themselves in the inactive state because perception probably does not occur with sufficient unitary character or coherence, perhaps also because of the lack of precision in shape discrimination. In short, the perception of shapes without facilitation is always more or less diffuse and labile, there is not enough coherence and there is a tendency to alter the shapes.

Similar characteristics to those described for figure perception can be found in object perception. Object perception is very slow in our subjects, even with good illumination and proper object size, because it is a complex process which develops less directly than in a normal individual. These subjects usually show a peculiar inability to perceive an object as a whole, and often have to resort to an indirect way of comprehension by means of an analytical-deductive procedure. The perception of an object is not carried out at once, but these subjects carry out a kind of *reading of the configuration (constellation)* of the object; that is to say, the perception is carried out by paying attention successively to the diverse specific aspects of the object in order to subsequently achieve its comprehension in a deductive way. Often the interpretation is only approximate when the objects are little known, but in general it is correct. Goldstein and Gelb (1918), in their case Schneider, have been the first to study this type of indirect perception which they put in relation to the difficulties of shape perception. However, this type of perception should be considered more precisely as the result of the inability of simultaneous complex recognition, as happens, for example, in Wolpert's simultaneous agnosia (Wolpert 1924).

Some examples of the mode of perception indicated are the following responses in quotation marks of subject M, inactive, facing different objects:

Measuring tape rolled up: "Something messed up ... that *should be* a tape measure."

Shell: "Something flattened and striped ... *should be* the lid of a clam."

Natural sponge: "This … a bunch of wool … ." Questioned again: "… no, a sponge."

A white bean: "Something squashed, whitish, I don't know what it is … ." Insisting: "… a bean."

Apple: "A lemon." Then he smells it and realizes it is an apple.

The understanding of the object can be quite late because, apart from the slowness of reaction, the subject first makes an examination of the visual configuration of the object and then attempts the interpretation, which is deduced either from a series of partial aspects or from some characteristic detail of the object. In objects of less frequent use the subject usually asks the question "what could this be?" In special tests with slightly modified objects, both the diffuse perception and the difficulty of interpretation when characteristic aspects are missing are clearly demonstrated. For example:

A table knife with the top half of the blade missing: He recognizes it immediately. He is asked about possible defects and answers: "It is okay and complete." Only when he touches it he realizes that it is shorter than he thought; he says that with his sight he noticed that it was short, but not that short.

Big key without teeth: (Puzzled) "I don't know, if it is a key it would have something at the bottom."

Knife handle: "I don't know either … a piece of white iron that I don't know what it is."

The blade of the previous handle: "This … yes, a knife." The previous handle is placed next to it, and he immediately says: "Ah! this, the handle."

Apart from the slowness and indirect recognition, it can be said that objects are quite well understood, and only in special tests like the ones above, some difficulty arises. Instead, under facilitation by intense muscular contraction, the perception of objects is rapid and unitary, and modified objects are also correctly interpreted.

All these tests are performed in near vision and in very good illumination; the perception of the configuration is then reached in about three or four seconds, which means a very large increase in time compared to a normal individual. It is evident that in the examination of certain objects, although the configuration can be seen well, an additional time is spent in appreciating characteristics. But when the vision is less good because of the distance, or in near vision but with weak illumination, the perception of the object requires more time, going through a series of sensory stages. In such conditions, a pair of scissors needs about eight or nine seconds to be perceived; first, a dark spot appears which soon becomes smaller, then something elongated with a greenish tint is perceived, next a more defined shape such as "something long with two rings underneath," and the subject thinks: "it can't be a key, maybe a pair of scissors." Given the rather long time needed for this development, the process can be followed quite easily in subject M, but if the exposure time of the object is shortened, the perception stops in the primary phases, which are insufficient to reach the interpretation of the object. Under the same conditions, subject T in monocular vision using the right eye needs only about two seconds, sometimes almost three, whereas in binocular vision only one second. Also in the first moments, objects are perceived as swollen and shapeless, and later more shaped. Thus, with low intensity

of stimulation, the perception of objects develops slowly and goes through various phases due to asynchrony.

In subject M, who is the most impaired, very different types of object perception are easily obtained depending on whether only one or both eyes are used, and whether the state is inactive or facilitated by muscular effort. In general, in ordinary illumination, binocularly and inactive, or only with the right eye and with facilitation by muscular effort, perception shows some deficit; it is not very coherent and stable, and defects in objects go unnoticed or very attenuated. In contrast, in binocular vision and with facilitation, perception is very good, and small objects like a dice are recognized in one second at most; and if they are bigger and easier, half a second is enough. However, if the illumination is reduced or the distance is increased, the difficulties of the other inferior types of vision appear. In ordinary life, the perception of objects, although a little slow, is not bad at all because binocular vision is used, and there is always some facilitation by muscular effort that the subject consciously increases when necessary since the discovery of such phenomenon. But if summations are excluded, a great difficulty arises; thus, in monocular vision, and medium illumination and distance, a felt hat can easily be perceived as a beret. Subject M can see the time on a pocket watch at a maximum distance of 14 cm in good illumination, binocularly and with facilitation by muscular effort; and although the thinnest lines of the Roman digits disappear, this is not a problem because he knows the location of the digits in relation to the watch lug, and if the hands of the watch seem too short because their thinner ends are invisible, this is not a great impediment either. Even with facilitation by muscular effort but in monocular vision, he can no longer perceive either the numbers or the hands of the clock. Therefore, to use the watch he should resort to all possible facilitation effects to overcome the situation. Naturally, in examples of this type, the significant visual acuity defect must be added to the agnosia disorder.

In summary, it can be said that visual perception of objects behaves in the same way as other functions, i.e., there is a deficit which can be very significant, but perception also depends on the conditions of stimulation, the state of central excitability, the type of facilitation, etc. In general, the interpretation of an object is almost always possible except in the case of special tests, although the process is slow, indirect and less consistent than in a normal individual. It is important to point out that the true capacity to interpret objects is properly revealed in drawings where, due to the necessary schematic simplifications, the difficulties of conception and understanding become very evident, which we will deal with later. Changes in perception of the shape of letters, for example, can already be considered as an alteration of alexic type, which introduces us to the study of perception of symbols, drawings, etc. A rigid separation cannot be established between the recognition of objects and that of their abbreviated representations by means drawings. We shall continue the study of the understanding of objects when dealing later with the cognitive schema. There we shall also address visual agnosia, which is not an isolated alteration but only a final and complex stage in the global and unitary disorder of the visual function.

Visual image orientation

12. Visual image orientation disorder

12.1. PRECEDENTS ON THE ISSUE OF VISUAL IMAGE ORIENTATION

Given the importance of the orientation of the perceived visual image, especially because of the exceptional phenomena exhibited by our wounded patients, in particular subject M, a chapter is devoted to the study of this topic. The orientation of the perceived visual image is only a special aspect of spatial localization. Due to the functional complexity of such localization, a very peculiar disaggregation appears due to nervous asynchrony, giving rise to the phenomenon of inverted vision in the most altered cases, and diversely tilted in the lightest ones. The dynamic analysis regarding excitability conditions offers more possibilities here than in any other phenomenon because quantitative determinations can easily be performed. In addition, different aspects of the visual functions analyzed in previous pages find application in the research on visual image orientation. A systematic and as complete as possible study of the orientation of the perceived visual image is therefore conducted.

The issue of visual image orientation has given rise to many discussions and works since long time ago. Porta (1589/1593) and Scheiner (1619) having discovered at the end of the Renaissance that the visual image was formed inverted on the retina, the question immediately arose as to how it was possible for visual perception to be right. Kepler (1604) addressed this issue, and later Descartes (1637) in his "Dioptrique" attempted an explanation by thinking that the inverted image on the retina is counteracted by the corresponding inversion of the optic nerve fibers in the brain, thereby making vision appear right. Ramón y Cajal (1898, 1899) also tried to explain the crossover of the optic chiasm and all the decussations of the pathways of the nervous system as an anatomical arrangement designed to counteract the inversion on the retina. But we shall see that the physiological experiments of Stratton (1896, 1897) do not give weight to this anatomical criterion.

Continuing the development of the issue, we find in the sensualist philosophy of the 18th century a marked interest in the nascent psychology of senses as a basis for the theory of knowledge, and again the problem of right vision being the image

inverted on the retina appears as a fundamental issue. Particularly noteworthy are Berkeley (1709/1910) and Condillac (1754/1821), who address this problem in relation to the theories of spatial perception in which either touch or vision intervene predominantly, according to various authors. Essentially, it is thought that from the coordination of these two senses should be derived the perception of space and orientation in it. Especially Condillac (1754/1821), studying the formation of spatial orientation using his well-known simile of the statue that gradually acquires the different senses, points out that sight relies on touch to achieve the primary orientations. In this way, the inversion on the retina does not mean any difficulty for the right vision, since as long as the eyes have not been instructed by touch, there is neither up nor down for them.

The issue becomes even more significant within the physiology of senses established in the 19th century. Nativists and empiricists, represented respectively by Müller (1826) and Helmholtz (1867/1896), dispute the solution of this issue. According to the latter author, even the adult eye needs constant information from experience to maintain agreement between visual and tactile perceptions. The debated question of why objects appear right while their retinal images are inverted seems to be solved within such a criterion. The sense of touch is able by itself to give us complete notions about space, even without the sense of sight; the behavior of those born blind is enough to convince us. Moreover, the sense of gravity which determines up and down is not immediately obtained by sight but exclusively by touch.

Finally, the issue acquired an experimental character at the end of the 19th century thanks to the important experiments of Stratton (1896, 1897) which show that the orientation of the visual image is only the result of the functional relationship between visual and tactile (haptic) stimuli on our own body. Thus, it is possible to see just as well with an inverted image on the retina as with a right image on it, since there is a correlation and adaptation based on the upright position of our own body. Stratton performed the experiments on himself, closing one eye and placing in front of the other a combination of lenses that gave an inverted image of the object and, therefore, an upright image on the retina. The experiment lasted two and a half days, and finally he was able to achieve a more or less perfect right (correct) vision despite the fact that the image on the retina was not inverted in relation to the object. In other experiments, the duration was much longer, and he described in detail a number of very interesting phenomena. When he removed the lenses, a muddled scene appeared before his eyes which lasted several hours until normality was restored.

Throughout this historical development of the issue, it was established that the orientation of the visual image must result from a correlation between visual and tactile sensations, the latter being the point of reference. Thus, the anatomical conditions, in particular the inversion on the retina, have no meaning since all is decided by the visual-tactile correlation. However, the pathological manifestation of inverted vision leads to a new general situation. The question of whether the retina could by itself determine the orientation of the image is sometimes overlooked, sometimes left in suspense, and only very rarely assumed as possible by some isolated author, but we find the specific answer to that question in the brain lesions that cause inverted or diversely tilted vision. It is possible to affirm that inverted vision

is due to the local sign of the retina because of an alteration in the brain mechanism of the visual-tactile correlation. Thus, the old question of the inverted image on the retina becomes active again.

From the above indications, it should be noted that the issue of visual image orientation has so far been a study of the domain of classical psychologists and also of some physiologists of senses. Such an important issue for brain pathology is still foreign to it today, and not because it could be claimed that visual orientation disorders have not appeared in brain-injured patients. On our part, we can state that it is an extremely common alteration which, properly examined, can easily be found, if not the most exceptional complete inversion, at least the tilt of the image to a greater or lesser extent. This reveals the disturbance of the nervous mechanism of orientation of the visual image and also the role played by the inverted image on the retina. References on inverted vision in brain disorders are already found in Pick (1908 a), Kolb (1907), Phleps (1908), etc., and in other more modern observations by Gerstmann (1926), Wilders (1928), Halpern (1930), Pötzl (1943), etc. All the indications of these authors are very brief, and it is characteristic that in none of the cases known before ours cases, the alteration in the orientation of the visual image has been objectively observed, because the alteration had occurred sporadically in cortical epileptic seizures, angiospastic crisis, etc. This is why the authors limited themselves to recording the phenomenon, obtaining the information from the anamnesis of the patients or from their spontaneous statements. Therefore, there is a lack of any kind of test or experiment aimed at elucidating the disorder.

From time to time hypothetical considerations were made about the mechanism of the disorder trying to include it in the mechanism of agnosia. In some cases, the existence of tumors or other types of lesions in the parieto-occipital region was verified by autopsy. Despite the singularity of this disorder, it has remained so unnoticed or ignored that it is not mentioned in the main works on brain pathology such as the two by Monakow (1914 a, 1914 b) and the recent one by Kleist (1934), nor in the various monographs on visual agnosia (Stauffenberg 1914, Poppelreuter 1917, 1923, Pötzl 1943, Quensel 1931, Lange 1936, etc.), although it can be assumed from some descriptions, that patients diagnosed with visual agnosia of spatial orientation would be affected by a visual image orientation disorder that went unnoticed.

However, we must point out, as an exception and very interesting physiologically, the following experiment conducted by Hoff (1929). A patient who presented a large cranial laceration due to a war wound in the parieto-occipital region, presented cortical epileptic seizures during which, without loss of consciousness, objects appeared to be tilted about 30°. When the patient was seizure free, a similar tilt occurred by cooling the brain through the cranial laceration. No details were provided about the concomitant visual disturbances (e.g., shape, etc.), the author limiting himself to localize the mechanism of visual image orientation in the mentioned brain region.

In the two cases we are dealing with, especially in case M, we shall expose for the first time in an objective way the complex phenomena of disaggregation in the orientation of visual image, according to tests and experiments performed during several years. These experiments are crucial for understanding the mechanism of visual image orientation in humans.

12.2. INVERTED VISION TO VARYING DEGREES: M, T AND OTHER CASES

In the summer of 1938, the phenomenon of inverted vision in the M case was discovered by chance a few months after the subject suffered his brain injury and while he was still convalescing. He did not seem to be very surprised by the finding, and explained that, since some time ago, he was seeing occasionally tilted or even inverted objects. He seemed to have seen men upside down working on a scaffold; without giving much importance to the fact, he used to say: "these are things that sometimes come into my sight." Otherwise, the disorder did not make his ordinary life at all difficult; he was able to walk down the street and orient himself perfectly without suffering any accident. This is understandable if one considers the phenomena of summation that usually occur spontaneously: binocular vision (mutual eye facilitation) and standing or walking, the latter acting as a moderate but very efficient facilitation by muscular effort.

In addition, if the subject only pays attention to close or large objects, which appear sharper, perception is correct even without facilitation by muscular effort. Objects that are seen well, appear correctly oriented, so the subject cannot find any divergence with tactile information when handling them. However, if an object is far away and its motion is perceived as reversed, the perception of its form is very poor or completely blurred, and only the reverse direction of motion can be perceived, and since it is far away, orientation by the sense of touch is not possible, and no discordance with this sense is manifested. For all these reasons, in ordinary life the phenomenon easily goes unnoticed by the subject, and when it occurs in unexpected situations it is so fleeting that it does not alarm the subject who gives more importance to the healing of his wounds, to his general state, to headaches, etc.

In August 1938, a considerable amount of new data was obtained in a short period of time during a very laborious and sometimes not at all easy research work. We were completely ignorant about the origin and mechanism of the disorder, and finding ourselves at every step in the midst of an endless change of phenomena, experimentation and observation were done rather randomly. At last, simple rules were established to produce inverted vision: objects appeared correctly oriented when seen distinctly because they were close, appeared tilted when they were far away and vision was worse, and appeared inverted when they were even further away and vision was very poor. Of two very different sized objects placed at the same distance, the smaller one was more tilted. If the exposure time of the object was very short, it was perceived as strongly tilted or inverted, even in near and distinct vision. These conditions of stimulation that allowed us to obtain inverted vision with ease and security in subject M, were not properly valued until much later, since the attention was initially focused on finding new phenomena close to the orientation disorder, such as recognizing numbers and letters equally well in any orientation, and other complex alterations of spatial orientation. One year later, this patient was examined again in detail, the excitability conditions were specified and the dynamic action phenomena were found. When a sufficiently distant object was perceived as inverted, it was found that by applying facilitation through muscular effort, re-inversion was possible. Since that time, visual orientation disorder and all other disorders

have been systematically investigated through a large number of experiments, both in the M and T cases.

A more precise examination shows that inverted vision is not complete in subject M, since in the eye with worse vision (left eye) the maximum inversion that can be reached is about 170°, and in the right eye it is 135°, the subject being in an inactive state, and examining each eye separately. The difference up to 180° is small enough that, under certain circumstances, the vision is considered inverted for practical purposes. Other maximum tilt limits are obtained through different facilitations (bi effect, muscular contraction, etc.), due to the correction of asynchrony and subsequent improvement of the orientation disorder. In this way, a same individual can present alterations of different intensity in the image orientation, according to his brain excitability state (his physiological level).

Because the brain lesion in subject T is less severe, his physiological level is less altered, as we have already seen in various visual functions. In this subject, we find a maximum tilt of only about 25° in the eye with worse vision, and different limits of less asynchrony are also obtained by applying different types of facilitation. Conversely, the degree of asynchrony can be increased either by brain cooling through the cranial wound or more simply by the effect of alcoholic drinks; in both situations brain excitability is reduced and consequently the degree of asynchrony is increased. At the beginning of the examination of this second subject in 1938, the tilt of the image was somewhat greater but not reaching 90°. However, during his first cortical epileptic seizures due to his cranial scar, his vision seemed to be strongly altered, and even after the seizures had ceased, visual image orientation was very disturbed, so the patient preferred to stay in bed until the disorder disappeared. In his own words, the position of objects "appeared very messy." It also seems that he had presented this altered state when he regained consciousness a few days after being injured. It is very likely that in such a situation this subject has presented the phenomenon of inverted vision or at least close to it, and that after a rather rapid recovery, the disorder has been reduced to a small tilt that now remains stable and can be revealed, as in subject M, by minimal stimulation and other conditions. Subject T also showed until 1939, the peculiar and important phenomenon of loss of orthogonal function (see Sec. 16), in the same way as it is presented today by subject M in an inactive state and even with good vision. Subject T was able to read the newspaper equally well whether it was in a normal position or upside down, without noticing any difference, and affirming that the letters were always in a normal position. This disorder disappeared probably since 1940, and a further detailed examination in 1942 did not reveal the slightest alteration in this respect.

In the Schneider case of Goldstein and Gelb (1918), there is no mention of inversion or rotation of the visual image, but it should certainly be somewhat tilted under conditions of minimal vision. This tilt could be evaluated in at least approximately 80°, perhaps more, based on several data on concentric reduction of the visual field and other disturbances.

We can also mention other cases found by us, some previous to this study on brain dynamics in the M and T cases, and others after it. In this last group there are two brain-injured subjects; one of them, already carefully examined by us in

1938, has a small shrapnel brain injury in the right occipital region, two or three centimeters from the midline. He presents a somewhat *asymmetric* type of brain disorder; therefore, he is not a pure central syndrome like M, T and Schneider, although he clearly shows dynamic alterations of a central type (general repercussion, summation, asynchrony, etc.). The importance of this case and the other (see below) will be discussed in the last part of this study. For the moment, we only indicate that the visual field of this first subject shows an intermediate alteration between hemianopsia and concentric reduction, which could be more precisely termed *asymmetric concentric reduction.* Limiting ourselves to the visual system, he presents, besides disorders in all functions (colors, luminosity, acuity, etc.) including an extremely evident green chromatopsia already mentioned, a maximum tilt limit of the visual image in the eye with worse vision of about 45°, much more than in the T case. This significant alteration in image orientation was recently discovered by us and has gone completely unnoticed by him for about five years, as well as other alterations, despite he being a professionally qualified individual who performs well in his activities.

The other case is that of a student who was examined long after being wounded in the left parieto-temporal region, and who also shows an asymmetric brain disorder, as the previous case but with a different location of the lesion. In the former case, the lesion is near the right occipital pole; in the latter, the lesion is near the left superior parietal lobe. Due to the dynamic phenomena of repercussion, which also occur in this subject, the rather anterior parieto-temporal lesion has caused a general disturbance that affects, although weakly, the visual function. His vision seems completely normal at first, and only sometimes he gets tired easily. But when the subject is duly examined, he shows a deficit, more evident in the right eye (contralateral to the lesion) in which a tilted vision of 5° to 7° can be detected. This subject must have had a more intense repercussion shortly after he was wounded; when questioned about this, he said that while he was hospitalized, he was surprised at seeing the baseboard strip of the room quite tilted instead of horizontal.

As for the cases we found prior to our brain dynamics research, we can cite numerous cases. It is remarkable that among all the cases studied so far, including M and T, only one wounded man with an anatomical occipital lesion and visual disorders very similar to those of the above-mentioned subject with asymmetric concentric reduction, spontaneously complained of seeing things tilted. Indeed, it was enough to tilt an elongated object in the opposite direction for him to see it vertically. At that time (1938) we did not know how to interpret this disorder, and therefore neither did we know how to determine the maximum tilt. Since the compensating opposite tilt had to be about 40° under ordinary conditions, much higher limits were certainly possible with minimal stimulation. Other cases with some tilted vision were found when various tests were carried out on wounded people with either an occipital or parietal convexity injury, but we did not determine the degree of disorder either, limiting ourselves in the protocols to recording the tilted vision together with other phenomena of a different nature. At least 6 cases were in these conditions, and surely some of them with vision close to the inversion as in the M case. In addition, many other cases must have gone unnoticed to us.

As an end to this section, we should draw attention to the following points. Firstly, it is clear that cases with tilted vision should be considered as frustrated types of inverted vision or rudimentary forms of inversion. Even in subject M, an exact 180° inversion is not reached although his vision can practically be considered inverted under appropriate stimulation conditions. The degree of asynchrony determines the maximum tilt limit, and the disorder in all cases lies only in the disaggregation of the brain's mechanism of localization. If the perceived orientation is somewhat tilted, such a mechanism is more or less asynchronous, and the retinal factor (with the image inverted) comes into play to a greater or lesser extent. In short, different tilts are quantitative aspects of the same alteration.

Secondly, the alteration of the orientation of the visual image does not represent an autonomous syndrome, as is also the case with any other type of disorder, since a functional disorder is always for the whole of brain activity. Within all visual functions, it is not possible to consider the function of visual image orientation independent of all others, and the intensity of the orientation disorder runs parallel with the disorder of the other visual activities. It is also very important to point out that the orientation of the visual image is disturbed from anywhere in the sensory brain. In fact, we have detected image tilts of different degrees in patients with either right or left parieto-occipital lesions, lesions in the occipital pole or far from the occipital region, such as lesions in the very anterior parieto-temporal region. Thus, there is no need to search for any kind of localization, an issue already addressed in part in the first part of this book on general aspects.

Finally, it can be stated that tilted vision is a very common disorder which has to be conveniently searched for. Any brain injury that affects visual functions in some way will immediately affect the orientation of the visual image by causing at least very small tilts. Tilt measurement can even be useful as a simple and accurate method to determine the intensity of the visual system disturbance.

12.3. GENERAL FEATURES OF THE VISUAL IMAGE ORIENTATION DISORDER

Visual image orientation disorders, and especially the phenomenon of inverted vision, will be studied in subject M, considering subject T as a complement to the different tests.

The first experiment consists of the tilt and inversion that occurs when an object, which is correctly seen at a short distance, is progressively moved away. This is nothing more than the fundamental experiment of asynchrony, in which the sensory level (perceived orientation) depends on the intensity of stimulation (visual angle subtended and light reflected by the object). This change in perceived orientation as a function of the stimulus intensity occurs in conjunction with changes in other visual functions such as color and shape; thus, the alteration of the visual system is global, which is important to bear in mind. If we use an upright white cardboard arrow about 10 cm high and 1.5 cm wide (3 cm at the head), it can be seen practically upright in very near vision and in very good illumination. In these conditions, the perception of its shape is good and correct, and as for its color, al-

though there are some green hues, most of the arrow looks white. But if the arrow moves away, always in the same upright position and with its head upwards, a tilt is produced, and at the same time the shape becomes blurred and the white of the arrow tends to disappear while green dominates more and more. This usually occurs when the perceived tilt is about 90°. Moving the arrow further away, all these characteristics are quickly accentuated, and the arrow finally looks almost inverted, dark green, and very altered in shape, like something elongated where the arrow head is distinguished only by a small bulk little different from the rest of the shape. Then the maximum inversion occurs, which does not exceed about 140° in the right eye, whereas it can reach 170° in the left eye. By moving the arrow a bit further away, all visual structure disappears, and the preceding perception becomes a dark, rounded and diffuse colorless spot. Consequently, as a general empirical rule it can be stated that in sharp vision, the perceived orientation is normal, and the more blurred the image becomes, the more pronounced the alteration of the perceived orientation. In terms of excitability it means that intense stimulation results in normal orientation, and weak stimulation results in altered orientation.

It is clear that the degree of asynchrony, and therefore the degree of image tilt, depends on the severity of the brain disorder, and in all cases, the maximum tilt limit must be determined under minimum stimulation, i.e., when the perceived shape is just at the point of being lost.

In the perception of orientation (and also in any other type of perception), the following three factors must be borne in mind: *stimulus*, *receptor* and *central* nervous condition. As for the stimulus, it is necessary to consider mainly its intensity and duration; the receptor influences the sensation according to its state (light adaptation degree) and by the site of the visual field (central or peripheral vision); finally, the nervous centers show different physiological level (different level of excitability and asynchrony) according to the summation effects to which they are subjected.

As for the stimulus, the excitation produced by the retinal image of an object depends on its size and luminosity. Thus, two arrows of different size and at the same distance will have different tilts; the larger one should be moved away to equal their tilts. In addition, since subject M's left eye has somewhat less functional capability than the right eye, an arrow at a given distance will be perceived as more tilted if seen by the left eye than if seen by the right eye. Stimulus duration should also be considered. Even in very near vision and very good illumination, if the exposure time is shortened, the sensation does not develop sufficiently, and a tilt appears; the shorter the exposure time, the more pronounced the tilt. Together with the tilt, there is blurring of the shape, color alteration, etc., since the entire visual function is reduced. It should be noted that, due to the slow reaction time, even at high stimulation intensities, a time of several seconds is required to achieve completely a right vision. A certain amount of stimulus (combination of intensity and duration) is therefore needed to perceive a normal orientation of the visual image.

The same is also true when we consider the receptor, which intervenes through the degree of adaptation to light and the stimulated zone of the visual field. An object correctly perceived in central vision can appear tilted in peripheral vision; the more peripheral the vision, the more tilted the object appears. Therefore, to

maintain correct orientation, the intensity of stimulation must be increased in peripheral vision.

As for the central nervous state, its action depends on the summation effects that reduce asynchrony. If subject M in the inactive state and in monocular vision perceives the arrow at the limit of inversion, a strong contraction of all his musculature is enough to cause a significant re-inversion that brings the arrow to an almost normal position. A complete re-inversion from the maximum limit of inversion never occurs because the indicated facilitation does not erase completely the asynchrony; however, the re-inversion is considerable, about 120°. Nonetheless, by adding other types of facilitation such as binocular vision, the re-inversion can be further improved in such a way that almost full re-inversion can be reached. Reducing stimulation appropriately until imperfect vision is obtained, a tilt can be achieved again even in binocular vision and under facilitation by muscular contraction, but in this case the maximum tilt is about 30°, whereas only using the left eye and in the inactive state the tilt is 170°.

By means of iterative stimulation, a certain re-inversion of the image is also achieved from the maximum tilt in monocular vision and inactive state. This can easily be done by quickly covering and uncovering the white arrow with a black card. However, in the various experiments described below, only summative facilitations allowing accurate determinations will be used.

The great asynchrony exhibited in visual image orientation and the great ease to measure image tilt offer an opportunity to clearly show the weak facilitations produced by tactile or acoustic stimuli on visual perception. A loud whistle next to the ear of subject M causes the tilted vision to straighten out by about 15°, in such a way that he perceives a small turn with the sound stimulus, and an opposite turn when the summative excitement (the sound) ceases. A similar effect is obtained with tactile stimulation, for example, by brushing the subject's back softly and quickly, without causing pain to avoid defensive muscular contraction that could mask the experiment.

Finally, we must pay attention to the type of rotation in the perceived image orientation. The rotation occurs in the frontal plane, since this is the only way in which the visual image can be preserved. The visual image changes its orientation by rotating around the central point of fixation in central vision. In the T case, in addition to the rotation in the frontal plane, the upper part of the object appears to be slightly tilted towards the subject when the rotation is at its maximum (30° to the right in the right eye). This is because that part of the image is in a weaker region of the visual field, and due to spatial irradiation (flat color), that part seems to be tilted towards the subject. It should be remembered that when studying the vision of flat colors in the T case, it was indicated that a colored surface situated frontally is perceived by this subject with frontal irradiation only in the right half of the visual field, so this part seems closer. For both M and T subjects, and in central vision, when the intensity of the stimulus decreases, the rotation of the perceived visual image is clockwise in the right eye and counter-clockwise in the left eye. However, when the object is situated peripherally in the visual field, the sense of rotation is different for each half of the field in such a way that in the two right halves the image rotates in

the same sense, and in the two left halves it rotates in the opposite sense to the previous one, thus obtaining two homonymous hemianopsic layouts with a mutually opposite rotation. The fact that for each eye the rotation of the image is opposite in central vision gives rise to the special phenomenon of *binocular duplication of orientation* of the image when the image is not normal due to the stimulation conditions. A double image of a single object is then seen, the two images positioned in an X-shape, since the tilt perceived by each eye is different. In addition, because vision in each eye is impaired differently, we have that in subject M for example, the image in the left eye is more blurred and more tilted than in the right eye.

As for the method followed to determine the alterations in visual image orientation, it is very simple. The characteristics of the stimulus (luminous intensity, duration, etc.) can be easily and directly measured, and for the quantitative evaluation of the image orientation (or sensory level) produced by a certain stimulus, the compensation effect is used, i.e., rotation of the test arrow in the opposite sense until the subject perceives the arrow as vertical. By measuring then the degrees of the compensatory tilt on a graduated circle, we know exactly the rotation experienced by the perceived image. In this way it is easy to get an experimental curve with as many points as desired, obtaining very complete determinations.

13. Dynamics of visual image orientation

13.1. FUNDAMENTAL EXPERIMENT ON VISUAL IMAGE ORIENTATION. STIMULUS DEPENDENCE

The multiple excitability relationships that arise in the tests on the alteration of visual image orientation can be classified into three main groups: perceived orientation as a function of stimulus, as a function of the central nervous state and as a function of the state of the receptor. Of special interest is the degree of the disorder according to the different types of vision which in fact have their origin in the state of the nervous centers. We shall begin with experiments dealing with perceived orientation as a function of stimulus, the other two factors (central nervous state and receptor) being constant.

The perceived orientation (sensory level) as a function of the stimulation is no more than the fundamental experiment we exposed in the general part of this work when dealing with the dynamic analysis of sensory functions. There are three fundamental types of tests related respectively to recruitment, to the asynchronous set of disaggregated functions and to the development in time of the sensory level in visual image orientation.

An experience close to those of ordinary life and the first one detected in subject M is the tilting and inversion of the perceived image as the distance from the test object to subject M increases. The test object is the above-mentioned white cardboard arrow. In order to obtain a greater degree of asynchrony and to better evaluate visual function, monocular vision is used, choosing now the right eye of subject M because it is the eye usually studied in this subject, although his left eye presents a somewhat more intense alteration.

Fig. 13.1 shows the curves of tilt degrees (sensory level) as a function of the intensity of stimulation given by the visual angle subtended by the arrow at different distances from the observer's eye (angle obtained from the trigonometric tangent determined by the distance and height of the arrow), in a uniformly illuminated space (outdoors with light intensity of 125 foot-candles). To be more precise, it should also be taken into account that when the arrow moves away, the luminosity

reflected by it decreases rapidly as the inverse of the square of the arrow-observer distance. In this test, the duration of the stimulus is not a variable since it is considered indefinite, i.e., long enough for the sensation to develop as much as possible in each measurement. Moving the arrow away, the sense of rotation of the image is clockwise, and counterclockwise when the arrow approaches. In addition, with a small visual angle that causes a strong tilt, there is blurred vision of the arrow, green chromatopsia, etc., as indicated on previous pages.

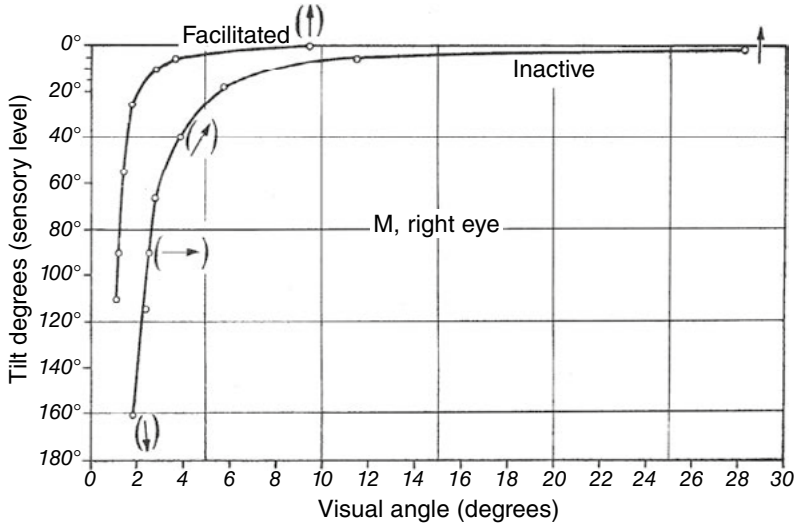

Figure 13.1. Perceived orientation of a 10 cm upright test arrow as a function of the subtended visual angle, for the right eye of subject M, in inactive state and under facilitation by strong muscular effort.

By comparing the two curves represented for the right eye of M, in inactive state and under facilitation by maximum muscular effort, the corresponding functional differences are easily appreciated. Both curves are of the same type but with different specific characteristics. The facilitation curve has a more pronounced curvature than that of the inactive state, and develops within much lower values of visual angle, i.e., there is a saving in stimulus intensity. Subject M inactive at 3 meters perceives a tilt of 160°, but under facilitation the tilt is 25° at 3 meters and 100° at 5 meters. The different degree of asynchrony under facilitation makes the maximum tilt limit much less, about 50° less. It is observed in the curves that starting from the inactive state and low stimulus intensity corresponding to a tilt of 160°, if facilitation is applied, a very significant, although not complete, re-inversion is achieved; there are still about 20 degrees left, i.e., a correction of about 140° is achieved. It is important to note that it is very difficult to achieve completely correct arrow orientation in the inactive state and using only one eye. Even with very high intensities, a small tilt of about 5° remains, especially using the left eye which is the

worse eye. Careful determinations also show the difficulty of achieving absolutely normal orientation using the right eye; even in very near vision (greater visual angle), the curve remains somewhat distant from the 0° value, as shown in Fig. 13.1.

The type of the curves shown corresponds approximately to Fechner's law (Fechner 1860), i.e., sensory growth is proportional to the logarithm of stimulus intensity, with the specific quantitative differences of the two extreme states of brain excitability (inactive and under facilitation) in the M case.

The excitability relationships are studied more accurately in the following tests by evaluating the intensity of stimulation more precisely by fixing a visual angle and varying the illumination of the test arrow. To this end, we used the experimental setup shown in Fig. 13.2. On a table there is a holder with a white arrow on a black background surrounded by a circle marked in degrees, and another holder with a luxometer whose photocell is at the same height as the center of the arrow. There is also an electric lamp with a screen illuminating both the arrow and the luxometer equally. Luminous intensity, regulated by a resistance, is measured by the luxometer, and the perceived tilt of the arrow is measured by the compensatory opposite rotation for the subject to perceive it as fully vertical and pointing upwards. The examined subjects are placed at a distance of 25 to 50 cm from the arrow, depending on the type of experiment. Thus, while the subtended visual angle remains fixed, the sensory level of visual image orientation (degrees of tilt) depends solely on the light reflected by the arrow.

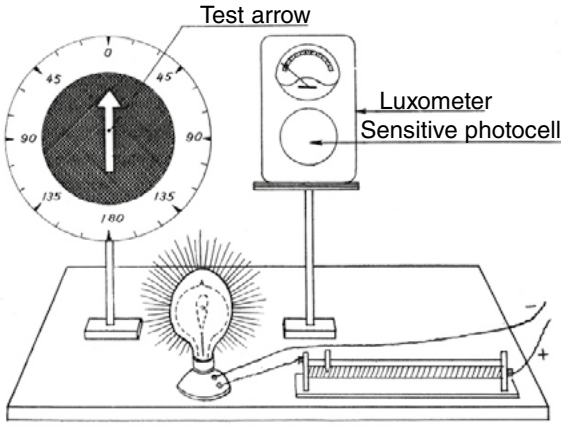

Figure 13.2. Experimental setup used in the test on visual image orientation as a function of light intensity. The illumination received by the arrow is measured by the luxometer, and the tilt perceived by the subject is measured by the degrees of rotation of the arrow in opposite sense in order to be perceived as vertical and pointing upwards.

By means of the indicated experimental setup, we have performed in our two subjects the fundamental experiment on asynchrony in its three main modalities: recruitment, asynchronous bundle of out-of-phase sensory levels and development in time of visual image orientation.

13.1.1. Recruitment of sensory levels as a function of the stimulus

We first present the results of patient M, who is particularly suitable for a more extensive analysis due to his greater asynchrony. A comparison is always made between his inactive state and the facilitated state by muscular effort, as shown in Fig. 13.3 where the two curves corresponding to these states are displayed. Both curves are of the same type, and only differ quantitatively because they correspond to different physiological levels. In the facilitation curve, not only the required intensity of the stimulus is reduced, but due to less asynchrony of the nervous elements, the curvature is more pronounced, and the curve is significantly higher than that of the inactive state for the same eye; the tilt limit of the visual image is much smaller (less inversion). With a luminous intensity corresponding to the tilt limit in the inactive state (about 145°), a decrease in tilt degrees of about 100° to 110° is possible by means of facilitation, but are missing about 30° or more to reach normal orientation.

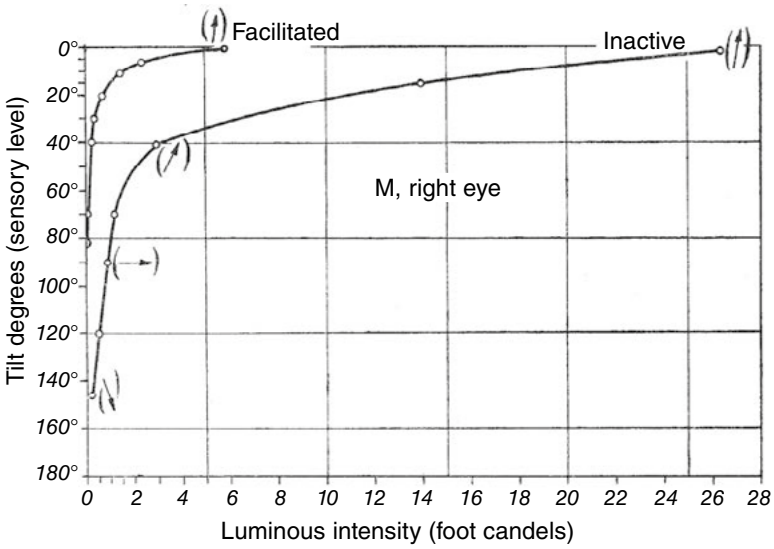

Figure 13.3. Recruitment of sensory levels: visual image orientation as a function of stimulus intensity (test arrow illumination) for the right eye of subject M, in the inactive state and under facilitation by strong muscular effort. Note the reduction in the required stimulus and the maximum tilt limit when M is under facilitation. The test arrow is 40 cm from the observer.

We have already said when dealing with the perceived orientation as a function of the visual angle subtended by the arrow, that a completely normal orientation without the slightest deviation is difficult to achieve with large stimuli in the inactive state. Even in the right eye, the one with better vision, always seem to lack about three degrees for the correct orientation. This can only be determined by very meticulous examinations, since patients, especially the M case in the inactive state, only respond with sufficient precision when insisted upon; otherwise a deviation of up

to 5° is neglected and it is then difficult to determine the values at the ends of the curves. It can be seen in the curves that under facilitation and illumination of about 6 foot-candles, 0° of deviation is reached, however, in the inactive state and with an illumination intensity 5 times greater, the arrow does not arrive at the exactly vertical position, the superior end of the curve being slightly below 0°.

In these tests we cannot establish a comparison with a normal subject, as we have done for other functions studied previously, since the referred orientation function responds to an all-or-nothing effect in a normal subject; thus, a minimal stimulus does not produce the slightest deviation in the perceived orientation. It is only possible to compare the different pathological cases with each other, as we shall do further on. For the moment, we shall limit ourselves to a comparison between the two extreme states of excitability, inactive and facilitated, for the same eye.

If a colored arrow, e.g., red, is used instead of a white arrow, under identical stimulation conditions (visual angle and illumination) the red arrow is tilted about 10 degrees more. The white arrow, although seen as a very pale green, reflects more light than the red one and, therefore, the tilt is smaller because the stimulus is greater. This is the same effect as the one already exposed when dealing with the effect of color on motion perception using a metronome.

As for the changes in shape and color that occur along with the change in orientation of the perceived image, it is convenient to give more details, as shown in Table 13.1.

Table 13.1. Correlation between orientation, shape (visual acuity) and color of the arrow perceived by the right eye of subject M in the inactive state. Experiment corresponding to Fig. 13.3.

Orient. (degrees)		Shape (acuity)		Color
Inversion ↓	0°	Irradiation	Sharp vision of the entire arrow	Chromatopsia	Mostly white, very little green
	10°		Sharp tips of the arrow head are reduced		White and green equally
	30°		Slightly blurred		Green dominates over white
	40°		The arrowhead triangle not well-defined		Green dominates even more
	70°		Idem but more deficient		Still traces of white
	90°		Very blurry arrowhead		Completely green
	130°		Smaller arrow, very fuzzy shape		Dark green
	145°	↓	All shape structure just about to be erased	↓	Dark green limit

In general terms, it can be said that the shape and color of the arrow appear already altered when it is perceived with a tilt of 10°, the alteration being quite marked around 45°. In this regard, it should be noted that when visual acuity is tested with optotypes, those of small size are perceived as tilted, and when the deviation is about 30° to 40°, they are already at the limit of distinction of their shape. The perception is extremely altered at 90° and worsens very rapidly thereafter, as

can be seen in the recruitment curves and in Table 13.1. At the limit of inversion, shape structure is extremely rudimentary, appears as a faint hint, and the same happens with color perception. This sensory level corresponds in the M case to a perception of motion only as weak signs and in the opposite direction. Thus, maximal inversion, shape rudiments, minimal color emergence and threshold perception of motion correspond to the same functional level.

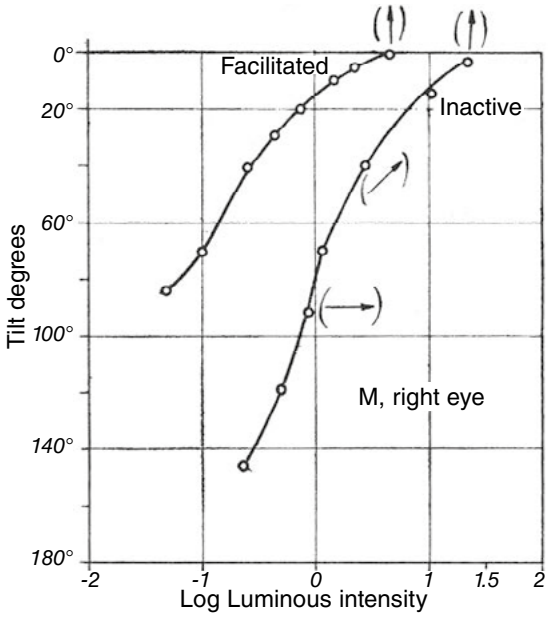

Figure 13.4. The same curves as in Fig. 13.3 but represented versus the logarithm of luminous intensity. The sensory level of the perceived orientation of the visual image increases approximately proportionally to the logarithm of the stimulus intensity. This is Fechner's law, which is fulfilled for most of the curve.

The sensory growth of the visual image orientation as a function of the intensity of the stimulus follows Fechner's logarithmic law (Fechner 1860). It is sufficient to take the logarithm of the illumination in the graphs of Fig. 13.3 to transform them into those of Fig. 13.4, which show a characteristic sigmoidal shape expressing the degrees of image tilt as a function of the logarithm of the stimulus intensity. Fechner's law is fulfilled except for the extreme values (very low and very high intensities), since most of the S-shaped curve resembles considerably a straight line. The recruitment of nervous elements as the basis for sensory growth is carried out in this type of alteration according to the normal general law of sensory variation, but in our cases the excitability values are very different from those of a normal subject. In the case of facilitation, the mentioned logarithmic relationship is fulfilled in the same way as in the inactive state, although at different intensities because the facilitated state corresponds to another value of brain excitability.

As regards the determination of the curves in this type of experiment, it is necessary for the subject examined to adapt to darkness first, and it is also necessary to proceed slowly enough in the determinations since the stimulus is supposed to act indefinitely or for a sufficient time. It is also very important that the distance of the arrow is fixed and, above all, that the subject's head is upright and immobile, otherwise the orientation of the visual image is easily altered for reasons that we shall mention later on. Likewise, any small dazzle or sudden change to very different illumination must be carefully avoided. If these conditions are fulfilled, the determinations are very accurate and reliable.

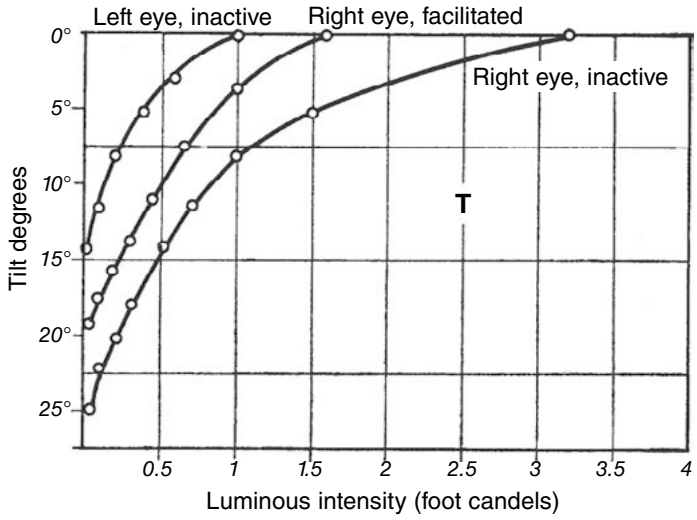

Figure 13.5. Orientation of the visual image as a function of illumination of the test arrow, for subject T, left eye in the inactive state, and right eye in the inactive state and facilitated state by strong muscular effort, under the same experimental conditions as for subject M (Fig. 13.1).

Regarding subject T, we already know that this individual presents much less asynchrony and, therefore, the orientation disorder of the visual image only consists of small tilts with respect to the correct orientation. Fig. 13.5 shows various types of alteration of the image orientation in subject T: for the right eye (the worse eye) in inactive state and under facilitation, and for the left eye in inactive state. The tests are carried out under the same experimental conditions as in the M case; therefore, the results of both cases can be directly compared. For this purpose, Fig. 13.6 shows various types of curves, including the one for the eye with worse vision in T inactive and the one for the right eye in M inactive. The great difference between the two subjects can then be easily appreciated. Given the lesser brain disturbance in subject T, sensory asynchrony in visual image orientation is only obtained with very low stimuli, and the maximum tilt value is small. This value is about 25° in the right eye in the inactive state, and 18° with facilitation by muscular effort. In the left eye, which behaves somewhat

better, the tilt limit is about 15°, and the effect of facilitation here is much smaller. Subject T also shows an overall alteration of the visual function as that described for subject M, although much more attenuated. Like in M, the maximum tilt limit corresponds to the shape alteration limit. A little before this limit and only in the right eye, fleeting greenish spots appear on the right side of the arrowhead, which disappear after a few seconds. These signs of chromatopsia appear mainly when the illumination is reduced.

Subject T can exhibit much more severe alterations as a consequence of epileptic seizures, alcohol ingestion or cooling of the cerebral cortex with ethyl chloride through his cranial wound.

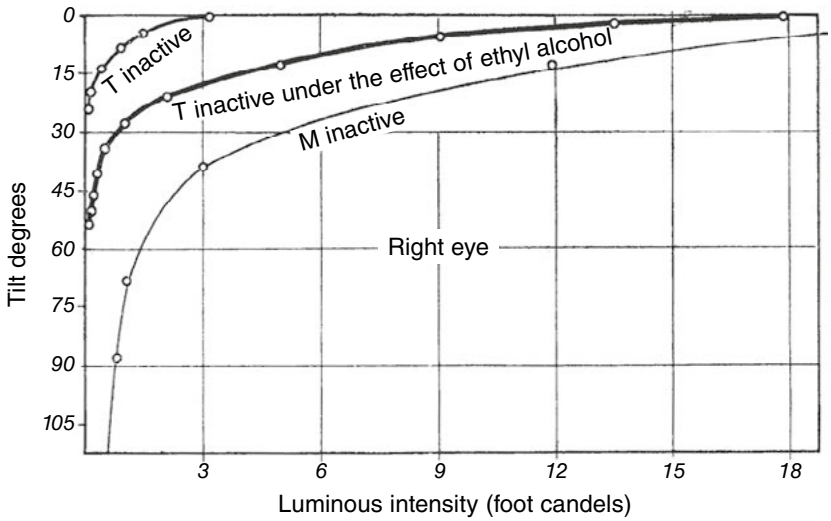

Figure 13.6. Orientation of the visual image as a function of illumination of the test arrow, for the right eye of subject T in the inactive state under the moderate effect of ethyl alcohol (bold middle curve), to be compared with the highest curve (same eye but alcohol-free) and with the lowest curve (right eye of subject M in the inactive state).

The effect of alcohol is remarkable. It is known that it increases nervous chronaxie, so it produces in this subject a transitory intensification of all his brain disorders when he falls into a state of alcoholic intoxication. In such a state and in binocular vision, he says he perceives objects as much more tilted and diffuse, and everything a little darker, with a tendency for green color to dominate. This alteration, in a state of acute alcoholic intoxication, disappears in about one hour, and in less time if he drinks a cup of strong coffee. In order to examine the effects of alcohol, we have studied in this subject the effect of moderate doses by means of the following test: a small glass of sherry is dispensed to him and the change in the maximum tilt limit is then measured. After five minutes, this limit has already increased considerably, reaching about 50°, whereas alcohol-free it was 25°. After ten minutes, the maximum effect seems to be reached, with the tilt being about 52° - 58°, which slowly decreases until the recovery of the initial state in about half an hour. During this ethylic effect, greenish chro-

matopsia increases, becoming less fleeting, although without reaching in any way that of the M case. Fig. 13.6 shows the curve of visual image tilt under the maximum effect of alcohol at a moderate dose. Compared to the curve for the same eye before the ethyl test, it can be seen that not only is the maximum tilt limit more than doubled, but that the curve has a flatter course, indicating that fully normal vision requires very high luminous intensities compared to that required in the ordinary state of subject T.

From the result of this moderate dose of alcohol, the effect of intense drunkenness, which subject T has suffered more than once, can be predicted. But the effect, no matter how excessive, always disappears completely. A similar effect to that observed with alcohol is produced by cooling the cerebral cortex with ethyl chloride through the loss of cranial bone that this subject presents. In this case, a moderate functional incapacity is produced that is not easy to determine with precision because the cooling becomes unbearable for the subject, who presents some clouding of the sensorium and of all activity of the waking state. The test arrow is perceived to be tilted more than 60°, and the whole vision becomes very dark and greenish. This experimental method of cooling the nervous centers, introduced by Trendelenburg (1910), offers many possibilities in experimental animals, the function being fully recovered when cooling ceases. However, this type of test in humans must be carried out with caution and for a short time, because it can produce a rather accentuated general clouding of all kinds of mental activities (darkening of vision, clumsiness of language, etc.), although it is harmless when used properly, and recovery occurs in a few minutes without leaving behind the slightest trace.

Of greater interest is the effect produced by epileptic accesses. Our patient reported that just after epileptic seizures, he suffers from very poor vision, especially in the right eye, perceiving a very large increase in the tilt of objects. These cortical epilepsy seizures, consequence of the cranial scar, appear from time to time, two or three a year, and are favored by states of alcoholic intoxication. In 1944, we examined our patient two days after he suffered a severe epileptic seizure that resulted in facial contusions from falling on his face. Although he had improved considerably in the days before the seizure, we were able to verify extremely intense disturbances that he never presented in his usual state. Thus, the tilt limit in his right eye was 120° whereas it was 25° in his usual state, and the tilt limit in his left eye was 70° whereas it was 14° in his usual state. Permeability to facilitation was also quite increased, since using the right eye and by muscular contraction, he was able to go from 120° to 60° of perceived tilt. At the same time, there was an analogous change in his visual acuity (from 1/8 to 1/6 with facilitation), in the very pronounced and stable green chromatopsia, in the concentric reduction of the visual field (he goes from a field reaching only 14° in the right eye with 0.5 cm test to a field reaching 22° with facilitation), etc. Tactile and auditory functions also showed greater alterations and significant changes under facilitation. In summary, when subject T was examined two days after a strong epileptic seizure, he showed an intermediate functional level between that of M inactive and M under facilitation by strong muscular effort; therefore, it can be admitted that just after the seizure, his brain alteration must have been probably as pronounced or more than that of M inactive. These alterations tend to disappear slowly, and after one week of having the seizure, he returns to his usual state.

13.1.2. Asynchronous bundle of out-of-phase (disaggregated) functions

The strength-duration curves for the right eye of subject M in the inactive state, for different sensory levels of visual image orientation are shown in Fig. 13.7. The tests are performed with the same experimental setup used in the previous experiments, determining for a given tilt (sensory level), the necessary duration of the stimulus according to the intensity of illumination of the test arrow. Due to the very slow reaction time of subject M, even the minimum exposure times of the arrow with high illumination intensities can easily be measured with a chronometer, without the need for a tachistoscopic display.

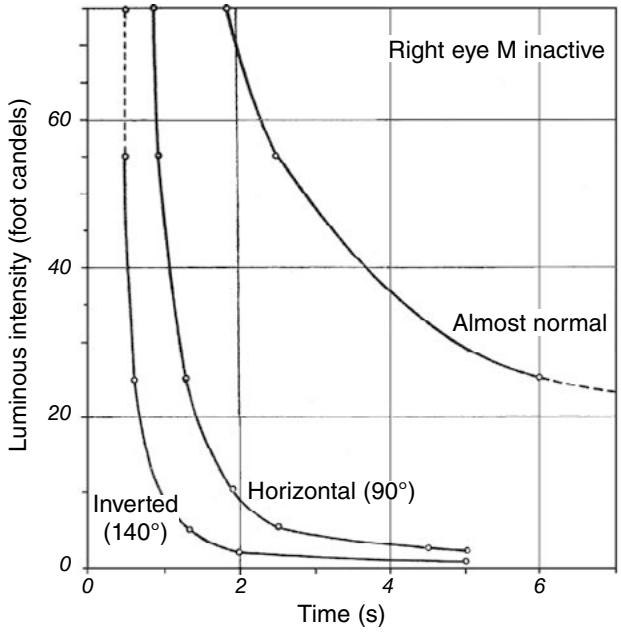

Figure 13.7. Intensity-duration threshold curves for three different sensory levels corresponding to three different perceived orientations of the visual image in the right eye of subject M inactive. Note the high rheobase threshold of the almost correct vision and the lower curvature of the corresponding curve. (Experiment with the test arrow used in previous examinations.)

Three different orientations of the perceived image are tested: almost normal vision (about 3° image tilt), maximum tilt (140°) and an easily recognizable intermediate situation which is the horizontal orientation (90° tilt). The result is three curves with different characteristics (different rheobase threshold and curvature). The curve for almost normal vision, being much higher than the other two curves and showing less concavity, indicates a functional level of great physiological demand, i.e., a high threshold and a slow reaction time, so its alteration is the first one to appear. In cases with mild lesion, as in subject T, it is the only alteration that occurs. The other

two curves corresponding to pronounced image tilts are much more similar to each other although there is a noticeable difference between them in tilt degrees.

We have already seen in the recruitment curves in Fig. 13.3 that the increment in stimulus intensity required to go from maximum inversion to horizontal orientation is relatively small, whereas it increases considerably to go from 90° to the right (correct) orientation, because the re-inversion increases proportionally to the logarithm of illumination of the test arrow. The two lower curves in Fig. 13.7 correspond to more easily excitable levels in time and intensity. This bundle of asynchronous curves, corresponding to out-of-phase functional levels (disaggregated functions), has no precedent in normal subjects, for whom we could only determine a single curve for the correctly oriented image, which would be well below the lowest curve of subject M and with a much shorter useful time (half a second maximum).

Under facilitation and certain types of excitability for vision, we would obtain, for both subject M and subject T, bundles of curves with less asynchrony, which would show a shorter useful time and a lower rheobase threshold. The separation between the curves in the rheobase level between the correct (or almost correct) orientation and the maximum tilt, is a measure of the asynchrony (or degree of functional delay) and, of course, is the difference observed in Fig. 13.3 between the intensity of the stimulus for the inversion limit and the corresponding intensity for correct vision.

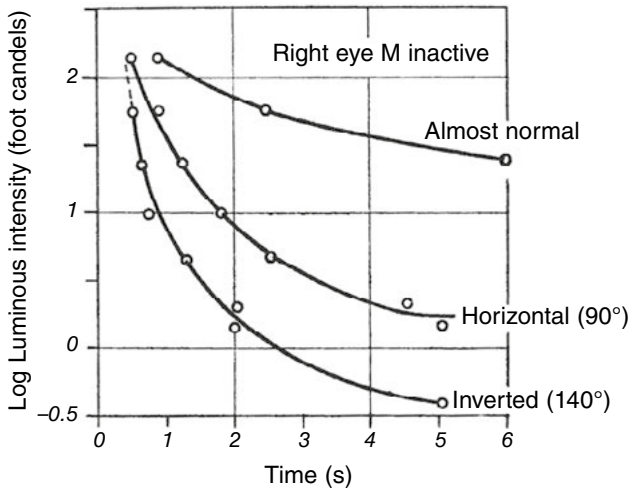

Figure 13.8. The same curves as in Fig. 13.7 but taking the logarithm of the luminous intensity of the stimulus.

Taking the logarithm of the luminous intensity of the stimulus, the asynchronous curves in Fig. 13.7 become those shown in Fig. 13.8. The rheobase levels in these new curves correspond to values of the recruitment curve for M inactive in Fig. 13.4, where the sensory level (perceived orientation) increases almost proportionally to the logarithm of luminous intensity.

13.1.3. Development in time of visual image orientation

Finally, by establishing a correspondence between degrees of perceived tilt and exposure time of the stimulus for a constant luminous intensity, a curve is obtained for the development in time of the orientation of the perceived image, thus completing the modalities of the fundamental experiment on sensory asynchrony in visual image orientation.

Due to the considerable slow reaction time in subject M (and also to a lesser degree in subject T) in monocular vision and inactive state, when any type of mid-intensity stimulation acts for a certain time, both subject M and subject T can perceive the development in time of the sensation since it goes through different stages until a final steady sensory level is reached. It is verified that subject M takes about six or seven seconds to correctly perceive a sufficiently illuminated vertical test arrow. But before reaching this final stage, the arrow is perceived fuzzy and somewhat tilted in the first moments, and a rotation of the image occurs as the image of the arrow improves over a few seconds. Normally, subject M can only perceive a rotation of about 80° or perhaps somewhat less, since the rotation is extremely fast at first, and only a change in small tilted orientations, which evolve much more slowly, is easily perceived. By studying the evolution in time of the perceived orientation, with the appropriate device, this evolution can be conveniently fragmented and, thus, the complete evolution can be determined quite accurately.

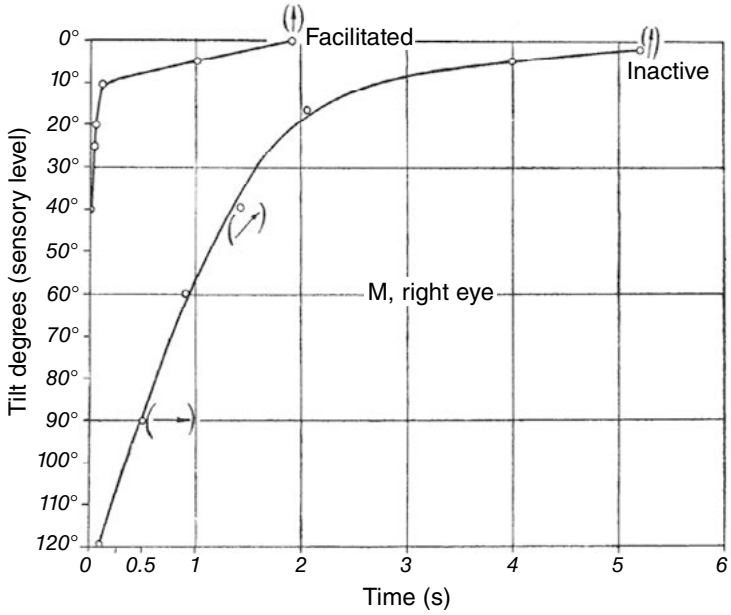

Figure 13.9. Time evolution of the orientation (tilt degrees) of the perceived image of the vertical test arrow, for the right eye of subject M, in the inactive state and under facilitation by strong muscular effort. Note the different time evolution and the different degree of asynchrony in these two states.

Without resorting to complicated methods, we already know that an object is perceived correctly if its stimulation capacity (size and lighting) as well as the exposure time are sufficient. However, it is perceived to be quite tilted if this time is reduced to less than one second or half a second. In order to obtain the time evolution curves of the perceived orientation, shown in Figs. 13.9 and 13.10, the duration of the stimulus is determined by means of a tachistoscopic display adjustable to the required time, and the degrees of image tilt are determined by means of the compensation method, i.e., by rotating the arrow in the opposite direction until it is perceived vertically pointing upwards. The distance and the illumination of the arrow remain constant during the determination of the entire curve of evolution in time of the sensation. In these tests we have used slightly higher luminous intensity than that of the rheobase level for correctly oriented vision. This is the reason why subject M inactive reaches practically correct vision in less than six or seven seconds, and similarly for the other cases depicted. In the curves, we can easily appreciate the very different evolution speed and the different degree of asynchrony according to brain excitability in each case.

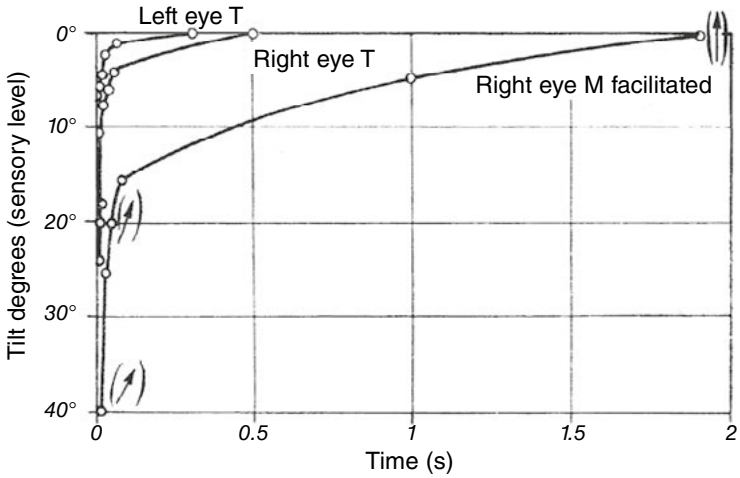

Figure 13.10. Time evolution of the orientation (tilt degrees) of the perceived image of the vertical test arrow, for subject T inactive, left eye and right eye, and for the right eye of subject M under facilitation by strong muscular effort (for comparison). Note the different evolution in time and the different degree of asynchrony in each case.

The time evolution curve is approximately of the same type as that of sensory growth by increasing stimulus intensity (Fig. 13.3), although less precision is obtained here due to a greater experimental difficulty.

13.2. VISUAL IMAGE ORIENTATION DEPENDING ON "TYPES OF VISION"

We shall see that with only these two subjects, M and T, a large number of brain excitability levels can be obtained by combining the effect of different types of facilitation.

Binocular effect in these subjects results in very interesting phenomena. When both eyes are used, a mutual facilitation in visual excitability occurs, improving in each eye the orientation of the image. The image becomes tilted only when the stimulus is less intense, reaching a much smaller tilt limit, although not as small as under facilitation by muscular effort. Since the image is tilted in opposite directions in each eye, it turns out that when the intensity of the stimulus is decreased by reducing the illumination of the arrow or simply moving it away, a binocular duplication occurs and two crossed X-shaped arrows appear, each arrow tilted to a different degree due to the different sensitivity in each eye. The following describes different stages of perception in subject M under different conditions.

1) The initial conditions are: binocular vision, inactive state and illumination adjusted to obtain 0° deviation (vertical arrow). If he closes his right eye, the arrow tilts 28° to the left (tilt corresponding to the left eye only). But if he closes his left eye, the arrow tilts 13° to the right (tilt corresponding to the right eye only).
2) By suitably decreasing the illumination, the following image duplication is obtained binocularly: a somewhat diffuse arrow at 30° corresponding to the left eye and another still vertical one corresponding to the right eye. If he closes his right eye, the arrow in his left eye tilts even more.
3) By further reducing the illumination and binocularly, the image corresponding to the left eye is at 90° and that to the right eye is oblique. In addition, the first one is smaller, more diffuse and darker.
4) By further reducing the illumination, the image corresponding to the left eye disappears at 122°, while the right eye perceives the arrow at 100°. Thus, single vision is again obtained instead of double vision.

Any type of facilitation modifies the image orientation disorder; the aspect of the corresponding curves changes and the tilt limit is reduced, as seen in the previous figures.

Fig. 13.11 shows different curves for the image orientation perceived by subject M according to the kind of facilitation used. In this figure, a considerable change in the sensory function can be appreciated when comparing very different *types of vision*, such as that of the left eye in the inactive state, and that of the right eye under binocular effect and facilitation by muscular effort.

As we already know, facilitation exerts an action on the nervous centers by modifying the central state and, thus, making up for the deficit caused by the destruction of brain mass. When muscular effort is combined with binocular effect, the action exerted is considerable and the visual defect is significantly corrected. Therefore, it is understandable that the usual behavior of subject M in ordinary life, using binocular vision and performing some muscular activity such as walking and other movements, results in a quite reduced image orientation disorder compared to what is obtained in monocular tests in the inactive state.

Figure 13.11 shows how the curves tend to approach 0° of deviation as more facilitations are applied, so lower and lower stimulus intensities are needed to reveal

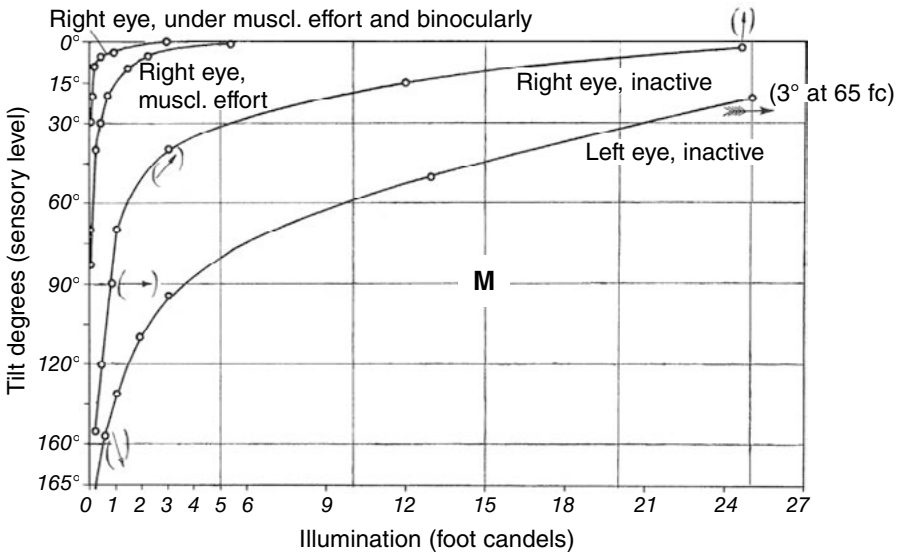

Figure 13.11. Orientation (tilt degrees) of the image perceived by subject M as a function of luminous intensity of the stimulus, for different *types of vision* in the right eye according to different types of facilitation, and for the left eye in the inactive state. Note the different slopes and different maximum tilt limits. The more facilitations are applied, the greater the reduction of the stimulus to produce correct vision, and the smaller the tilt limit.

deviations from the vertical line and, at the same time, the maximum tilt that can be achieved is much smaller. In addition, the curves tend to have a more pronounced curvature, which means that the variation of image orientation as a function of stimulus (differential sensitivity) tends to be more pronounced. In short, asynchrony is largely corrected by the action of summative facilitations that bring the excitabilities of the central nervous elements involved in image orientation closer, i.e., more synchronous, and the disorder is thus greatly reduced.

Note that above the curve in Fig. 13.11 for the most favorable state of subject M, there are still three curves for the T case shown in Fig. 13.5. Hence, with only two subjects, we obtain a series of functional types that allow us to investigate the perception of visual image orientation in very different states. It is possible to obtain, for each subject, eight types of vision. The two eyes having a somewhat different functionality, different curves are already obtained monocularly in the inactive state; in addition, we must also consider facilitation by muscular effort, by binocular effect and the combination of both facilitations for each eye. Therefore, with both subjects, 16 types of vision are obtained with the corresponding different curves.

From the data of the curves shown in Figs. 13.5 and 13.11, different degrees of tilt of the image are obtained with respect to the best vision taken as a reference. Thus, by adjusting light intensity so that in the left eye (the better eye) of subject T the exact upward orientation of the arrow is reached, other types of vision provide the tilts shown in Table 13.2. Thus, with the same intensity of stimulus (illumination

of the arrow at a fixed distance) required for the left eye of subject T to see the arrow completely vertical upwards, the left eye of subject M perceives the arrow at an angle of 135°, i.e., close to full inversion.

Table 13.2. Perceived tilt degrees for different types of vision in comparison with the best vision taken as a reference (see the text). OS = left eye, OD = right eye.

Type of vision	Tilt
T inactive, OS	0° (reference)
T facilitated by muscular effort, OD	3° to the right
T inactive, OD	6° to the right
M facilitated by muscular effort and binocular effect, OD	6° to the right
M facilitated by muscular effort, OD	16° to the right
M inactive, OD	75° to the right
M inactive, OS	135° to the left

Although we do not provide all the types of curves of perceived orientation that can be obtained in these two subjects, we shall indicate the maximum tilt limit of the image according to the types of vision in each subject. The tilt limits in the eight types of vision provided by the M case are shown in Fig. 13.12. These data are not the tilts shown in Table 13.2, since a different stimulus intensity is needed in Fig. 13.12 for each type of vision. Thus, as more favorable types of excitability are considered, the maximum tilt becomes smaller and corresponds to a lower stimulus intensity, as can be appreciated in the curves shown in Fig. 13.11. As seen in Fig. 13.12, subject M in the inactive state with his worse vision eye does not achieve a complete 180° inversion, although it is quite close and can be practically admitted as inverted vision. It can also be appreciated that the action of the binocular effect is weaker than muscular effort. By the combination of both facilitations, the functional state is largely improved, showing a very reduced tilt limit in comparison with the other states. It should be recalled that beyond the maximum tilt limit, the arrow loses every vestige of shape, even the aspect of something elongated, being impossible to appreciate its orientation since the arrow becomes a diffuse rounded spot.

The types of vision that can be determined in the T case are shown in Fig. 13.13, which refer to the ordinary state of this subject, i.e., free from all effects of epileptic seizures and alcohol intoxication. The types of better vision, that is, left eye facilitated by binocular effect and left eye facilitated by both binocular effect and muscular effort, are not indicated as they do not seem to present any image tilt. As regards the tilt limit of the image, it can be seen that the types of vision in the M case are lower than those in the T case. Note that the most favorable state in M can almost be equated with the most deficient type in T. Thus, OD of M facilitated by strong muscular effort and binocular effect, with 27° maximum image tilt, is practically equivalent to OD of T inactive, with 25° maximum image tilt.

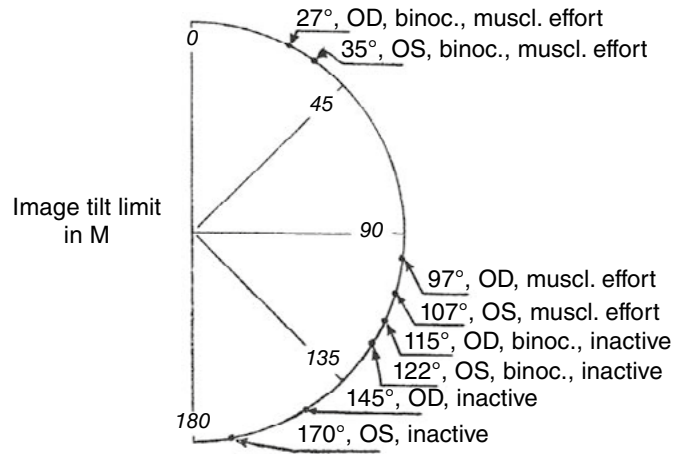

Figure 13.12. Image tilt limit according to the *types of vision* in subject M, corresponding to different excitability states obtained by means of diverse facilitations. OD = right eye; OS = left eye; binoc. = binocularly; muscl. effort = muscular effort. A different stimulus intensity is needed for each type of vision.

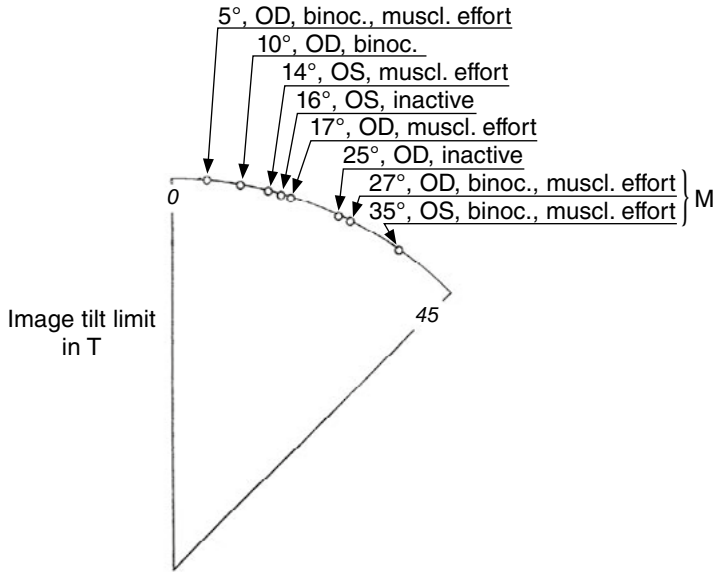

Figure 13.13. Image tilt limit according to the *types of vision* in subject T, corresponding to different excitability states obtained with diverse facilitations. Compare with the more favorable types in subject M. OD = right eye; OS = left eye; binoc. = binocularly; muscl. effort = muscular effort. A different stimulus intensity is needed for each type of vision.

Therefore, if subject M applies all the facilitations, his type of vision becomes equivalent to that of the poorest vision of subject T. This means not only an equal-

ization in the orientation function but also in all other visual functions (color, acuity, motion, etc.), as well as a same chronaxie value in the electrical excitation of the retina. However, although the mentioned types can be considered equal as far as functional level is concerned, a small difference is found in accurate determinations.

We have already indicated that in the T case, excitability is greatly altered by the action of alcohol drinks, leading to an increase in image tilt. Excitability is also particularly altered by the functional exclusion produced by epileptic seizures whose residual effects last a few days. In this situation, subject T may show a very similar state to that of subject M, and several types of vision are the same in both subjects.

13.3. SYNCHRONIZATION THROUGH VARIOUS FACILITATIONS. VARIATION IN THE CENTRAL NERVOUS STATE

As was said, the different degrees of alteration corresponding to the different types of vision are due to the action of facilitations by synchronization effect. Now we shall deal with the evolution of synchronization as a function of the intensity of the facilitation applied. First, we shall study the action of muscular effort on the re-inversion of the image. For the M case inactive, using the right eye and illumination such that the arrow is perceived with maximum inversion, the degrees of re-inversion (or sensory growth) of the orientation of the arrow are determined in relation to the muscular effort made by the subject by holding different weights. This is carried out in the same way as in previous experiments on saving voltage in electrical excitation of the retina, or on enlargement of the visual field by means of muscular effort.

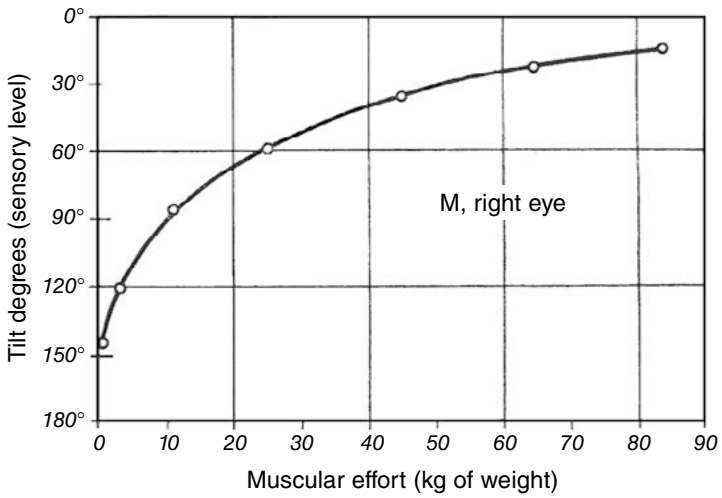

Figure 13.14. Synchronization (re-inversion of the image) in the M case, right eye, as a function of facilitation by muscular effort measured in kg of weight held by subject M.

Figure 13.14 shows the result obtained, which is a type of curve similar to that of perceived image orientation as a function of stimulus intensity. However, the conditions are quite different here since the stimulus (illumination and distance of the arrow) remains invariable, and the re-inversion (or sensory growth of the orientation) occurs by a central nervous modification produced by muscular effort. This facilitation works by increasing brain sensitivity (brain excitability) and bringing the asynchronous levels closer together. Hence, by increasing muscular effort, the asynchronous levels, lowering their thresholds, enter successively within the sphere of action of the invariable stimulus used in the experiment.

As we know, a decrease in image tilt entails a parallel improvement of visual function in other aspects such as colors, shape, etc. The points of the curve here represented constitute the average of several determinations, since this test is less stable than the preceding ones. Considerable re-inversion is observed for small weights, and becomes less and less noticeable as weights increase, without reaching, even with extremely large efforts (holding 85 kg), complete re-inversion. In fact, there are still about 15° to reach the vertical upward orientation. But we see that by means of great muscular efforts, a very important re-inversion is achieved, namely, from 150°-145° to 15°, thus reaching a decrease in tilt somewhat greater than that indicated in other facilitation curves in previous figures. For example, in Fig. 13.3, the maximum tilt of the image in the inactive state changes to about 35°- 40° by means of the ordinary facilitation by muscular contraction, producing a re-inversion of about 105°-110°, whereas the efforts of holding large weights make possible a re-inversion of 130° or more. These end positions are always difficult to determine, but we shall for the moment stick to the data indicated. In any case, starting from the maximum tilt, muscular effort does not attain to completely erase the asynchrony and to restore the function *ad integrum*, although here, in the case of visual image orientation, greater effects seem to be obtained by this type of facilitation than in other experiments such as that of saving voltage (Fig. 4.4), or enlargement of the visual field (Fig. 9.9), etc.

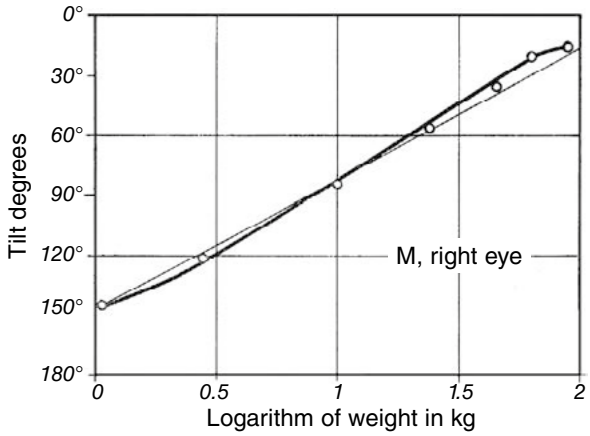

Figure 13.15. Re-inversion as a function of the logarithm of muscular effort (transformation of the curve of Fig. 13.14). Note that synchronization is proportional to the logarithm of muscular effort.

Taking the logarithm of the sustained weights, the curve of Fig. 13.14 becomes a rudimentary 'S' which approximates a straight line (Fig. 13.15), in the same way as in the case of orientation as a function of stimulus intensity (Figs. 13.3 and 13.4). This enables us to establish that the sensory level grows directly proportional to the logarithm of the facilitating muscular effort, due to the synchronization effect. Therefore, the growth of the sensory level as a function of stimulus intensity or as a function of the summative muscular effort is produced in both cases in the same way, although by a different mechanism. In the first case, there is a recruitment of levels by peripheral action, whereas in the second case there is a synchronization of levels by a central effect of summation which increases and unifies brain excitability.

Another very interesting form of facilitation is the effect of one eye on the other one. When dealing with the different types of vision, we have already studied the double image obtained binocularly (each image has a different tilt) and the correction of image tilt. Thus, by adjusting the illumination of the arrow so that the inactive subject M sees it pointing upward in binocular vision, if he closes his right eye, the arrow tilts 28° to the left, which is the tilt corresponding to the left eye only. These tests have led us to determine the effect on the image seen by only one eye when the other one is not looking at the arrow and is illuminated directly and individually. Indeed, a very remarkable effect is produced. If, for example, the right eye looks at the upright arrow which, due to its weak illumination, is perceived quite tilted, and the left eye, not looking at the arrow, receives a luminous beam of sufficient intensity, a significant re-inversion of about 75°-80° of the image of the arrow is perceived by the right eye. Monitoring suitably the illumination of the facilitating eye, the curve of the orientation of the image perceived by the right eye as a function of the light on the left eye (which has been duly separated from the arrow by a screen) is easily obtained with high precision. The illumination of the arrow remains constant. The curve obtained, shown in Fig. 13.16, has the same form as the previous curve obtained with facilitation by muscular effort, shown in Fig. 13.14, except that in the present case of ocular facilitation the re-inversion is much smaller. This similarity between the curves is very important since it ensures and confirms the action mechanism of muscular effort, whose measurement is less easy and less stable than the measurement of facilitation by light, the latter being performed with great ease and regularity.

Taking the logarithm of the illumination applied over the facilitating eye, the curve in Fig. 13.17 shows the same aspect as in the previous case with muscular effort (Fig. 13.15). Thus, the action of the two types of facilitation are identical. It should be noted that in order to achieve a full re-inversion by means of this type of ocular facilitation, the illumination of the facilitating eye must be increased considerably becoming much higher than the one needed to have normal vision only in the right eye. Let us emphasize that the left eye does not receive any shape information from the test arrow, but is stimulated only by light.

Experiments to study the effect of the various types of stimulation still need to be conducted and will be the subject of further studies. For the moment, we shall restrict ourselves to the referred experiment, which proves how facilitation action of one eye on the other one, under the indicated conditions, produces a synchronizing

central summation of the same type as that originated by facilitation by muscular effort.

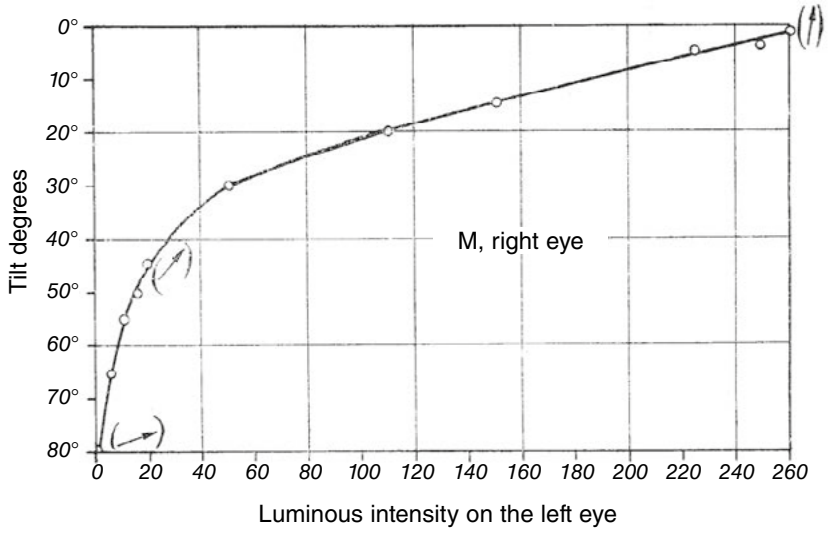

Figure 13.16. Orientation of the image perceived by the right eye of subject M, as a function of the luminous stimulation on the left eye not looking at the arrow, the illumination of the arrow being constant. The vertical upward arrow is perceived as rotated 80° at the beginning of the test.

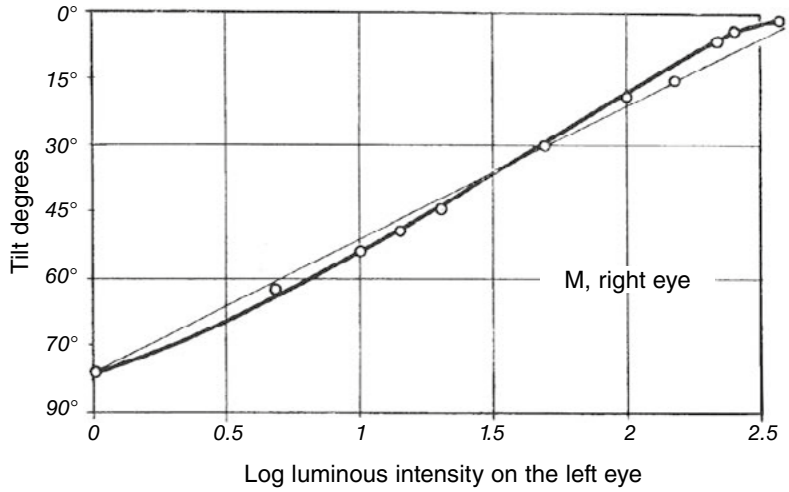

Figure 13.17. Same conditions as in the previous Fig. 13.16 but taking the logarithm of the illumination received by the facilitating eye (the one not looking at the arrow), isolated from the right eye. The sensory level (or synchronization) is proportional to the logarithm of the illumination on the facilitating eye.

Visual perception can still improve remarkably by other means since visual function is activated not only by the action of one eye on the other eye, but also by the light that bathes the eye being examined. Thus, in addition to the indirect facilitation of one eye on the other, it is necessary to take into account the direct facilitation by the action of the light that surrounds and bathes the examined eye, and which is independent of the light coming from the test arrow. If the arrow is illuminated in such a way that it is seen somewhat tilted by the right eye of subject M inactive, it turns out that when this right eye is directly illuminated without changing the illumination of the arrow, a certain degree of re-inversion is achieved, which varies in the way we already know by the previous experiments. However, there is a point in which, there being a great difference between the direct illumination on the eye and that of the arrow, re-inversion ceases and tilt reappears, increasing progressively in conformity with the mentioned difference. This new tilt under strong illumination of the examined eye is due to the fact that the eye is dazzled, and the arrow, which continues under much lower illumination, is seen increasingly darker and indistinct. This type of experience teaches us that, even in monocular vision, in order to determine the functional state correctly, it is necessary to pay attention not only to the luminous intensity of the test object, but also to the light that surrounds the eye being examined. Thus, it was already mentioned that visual acuity varies depending on whether the tests are performed indoors under medium illumination and strong brightness of the optotypes, or outdoors in full sunlight. In the latter case, the light surrounding the eye should be added to the light coming from the optotypes, in which case visual acuity would be much better.

In short, eye functionality in these subjects (M and T) depends on stimulation intensity. Stimulation includes not only that coming from the observed object (size, brightness, etc.), but also the intensity of the diffuse light that surrounds and excites the eye contributing to the final result of the perception. Up to a certain point, because the surrounding luminosity does not correspond to the test object, the effect of such luminosity can be considered as a facilitation effect, that is, a nervous stimulation foreign to the sensory stimulation from the test object. Likewise, it occurs in these subjects that a tactile stimulus is facilitated by another adjacent stimulus.

Concerning the general characteristics of synchronization by any type of facilitation, it was already said that re-inversion as a function of facilitation follows a behavior similar to re-inversion as a function of stimulus intensity; that is, the orientation perceived is directly proportional to the logarithm of facilitation or stimulus intensity, respectively. That said, facilitation cannot be considered only as a foreign stimulus that is added to the stimulus itself of the examined sensation, since facilitation is characterized by its central effect; good proof of this is the diminution of the maximum tilt limit of visual image orientation. We also know that subject M in binocular vision and with muscular effort resembles considerably subject T in monocular vision and inactive (Fig. 13.13). Thus, the loss of brain mass (central mass) is compensated by facilitation, and subject M, with a considerable central deficit, can be equated to subject T, with a small central deficit. Likewise, we know from the study carried out on general excitability, that facilitation reduces the chronaxie and rheobase values, i.e., excitability is increased and iteration capability is reduced. This increase in excitability brings asynchronous levels closer together, i.e., there is a synchronization of the

abnormally independent partial functions; however, such synchronization is never complete because the deficit in brain excitability cannot be fully corrected.

13.4. VISUAL IMAGE ORIENTATION DEPENDING ON THE STATE OF THE RECEPTOR

We have already exposed the variations in visual image orientation due to changes in stimulus intensity or changes in the central nervous state (i.e., by facilitation). We shall now deal with the influence of a third factor, namely the state of the retinal receptor, in which two main types of variation must be distinguished: topographic and light adaptation.

The different experiments analyzed above have been carried out using central vision. However, a test arrow, correctly perceived in central vision due to stimulation conditions, is perceived as tilted as soon as it is placed peripherally in the visual field. The tilt increases to the limit of inversion as the position of the arrow is increasingly peripheral. This change is of the same type as the one shown by other functions, such as colors and visual acuity. Because of the fast decrease in excitability from the center to the periphery of the visual field, the same stimulus that in central vision produces normal perception, peripherally produces an increasingly tilted image. This is due to an increase in the excitation threshold towards the peripheral field that makes that the invariable stimulus becomes undervalued and, consequently, the response of the nervous centers is reduced.

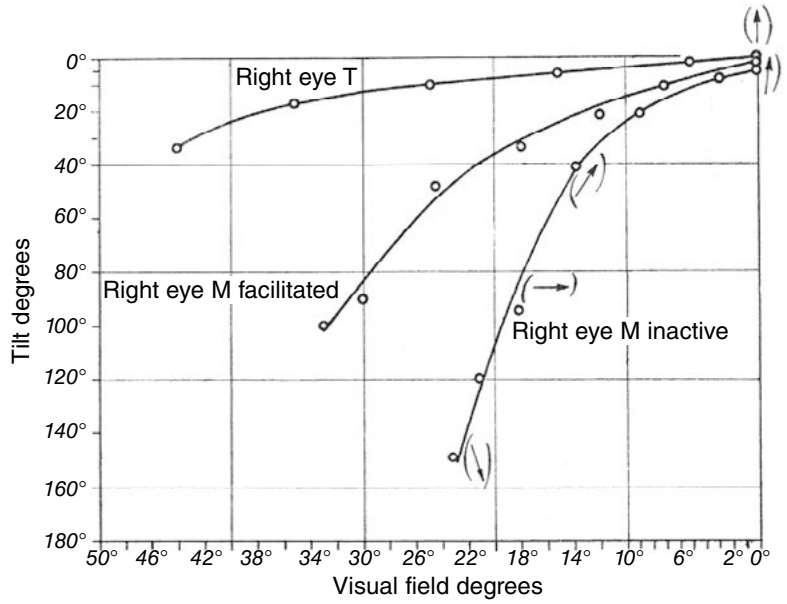

Figure 13.18. Orientation perceived of a vertical upward test arrow as a function of visual field degrees on the perimeter, for the right eye of subject T, subject M inactive and under facilitation by strong muscular effort. Note the different slope of the curves, the limit of vision on the perimeter and the maximum value of the tilt perceived.

Figure 13.18 shows the curves obtained for the right eye in the two extreme states of subject M, inactive and under facilitation by strong muscular effort, and in subject T. In order to obtain the curves, it is sufficient to provide enough illumination to a test arrow for it to be perceived as upright as possible in central vision. Then, by sliding the arrow towards the periphery, the degrees of tilt of the image are determined along the horizontal meridian, the illumination of both the arrow and the perimeter being constant. Thus, three different curves result, corresponding to different types of brain excitability. Each curve shows different slope, different maximum tilt of the image, and different site of the perimeter where this maximum tilt is reached. Due to the illumination conditions and the size of the arrow used, subject M in the inactive state reaches up to about 25° of visual field. If facilitation is then applied by muscular effort, the visual field enlargement is much less pronounced than using the 0.5 cm diameter test object, as we saw when studying the concentric reduction of the visual field. Here, in the inactive state, the arrow turns about 150° when it is at 25° on the perimeter, whereas under facilitation, the maximum tilt is about 100° when it is at 35° on the perimeter.

For the T case, the maximum tilt is only about 30° when the arrow is at about 45°-48° on the perimeter. All these tilt limits are the same as the ones obtained in central vision by reducing suitably the stimulation intensity. If in the M case, instead of the right eye, the left eye is used (not indicated in Fig. 13.18) in the same illumination conditions, the tilt limit is about 170° when the arrow is at about 20° on the perimeter.

According to what we already know, when the arrow is moved along the perimeter and is perceived as tilted, its shape changes, becoming blurry, and its color, initially white, gets increasingly greener. When the limit of maximum tilt is reached for each case, the perceived shape has worsened so much that, when the arrow is moved to an even more peripheral zone on the perimeter, its whole structure disappears, and nothing but a shapeless spot is perceived without the least element of directionality. The different slopes of the curves are of the same type as those we have seen in the experiments on orientation perceived as a function of stimulus intensity or stimulus duration, and are in relation with the other characteristics of the physiological level of each of the cases examined.

As it can be seen in Fig. 13.18, only subject T reaches in central vision (0° on the perimeter) completely normal perception without the least deviation, whereas in subject M there is still a deviation of 1° or 2° under facilitation, and 5° to 7° in the inactive state, as is the case in other types of experiments on perceived orientation.

It follows from these tests that in order to keep normal visual perception it would be necessary to increase the illumination when the arrow becomes more peripheral in the visual field. If the stimulus intensity is constant, as in the curves shown in Fig. 13.18, the topographic variation of excitability in the retina is the cause of the stimulus being registered diminished towards the periphery, leading to partial reactions in visual orientation function, given the asynchrony of the nervous centers. Therefore, it can be said that these centers need to receive a constant

amount of stimulation in order to achieve the most correct image orientation possible, within the circumstances of excitability in each individual. Not only the stimulus factor must be taken into account but also the type of receptor, that is, its excitation threshold (sensitivity to pick up the stimulus). For this reason, the same stimulus will cause very different reactions depending on the excitability of the receptor that picks it up, as it was shown in the experiments performed. In a normal subject, no alterations in visual image orientation are expected although excitability in the peripheral visual field is much lower than in the central field. This is because in the normal individual there is no asynchrony in the centers, and a minimum luminous stimulus already provides a correct perception of the orientation. Thus, in a normal subject there is an all-or-nothing response, whereas in pathological cases there is a partial reaction more or less complete depending on the kind of stimulation.

Identical considerations must be made regarding the influence of another type of variation in the receptor: light adaptation. Already without using special tests, it can be verified that if subject M is looking at a vertical upward arrow illuminated more than enough to be seen correctly, and the illumination is suddenly slightly reduced, a tilt is perceived. However, after 30 to 60 seconds the arrow is again perceived correctly due to adaptation to the new illumination intensity, still sufficient to achieve the correct orientation of the image.

The influence of light adaptation (change in sensitivity of the receptor) on the functional level of visual image orientation is studied more accurately in the following experiment. First, a suitable illumination is provided to a vertical upward test arrow for it to be seen as correctly as possible by the right eye of subject M in the inactive state, at a certain distance. Next, the eye is dazzled with a lamp of 250 foot-candles for two minutes. Subsequently, the perceived tilt of the arrow is determined during the time taken to adapt to the intensity of the light from the arrow (much less than that of the dazzle). Immediately after the dazzle, the luminosity of the test arrow turns out to be too low for the high threshold established, and the arrow is not perceived at all; however, in a few minutes, the adaptation process starts to recover the situation previous to the dazzle, and the arrow tends to take shape, going through different degrees of perceived tilt, from the maximum tilt to the initial orientation at the beginning of the experiment. Figure 13.19 shows the two recovery curves corresponding to the inactive state and the facilitated state by strong muscular effort. In the latter state, the recovery is made in about ten minutes, and in the inactive state in about twenty minutes, without reaching in this case a vertical upward direction as perfect as with facilitation. In addition, the recovery under facilitation begins earlier than in the inactive state. The maximum tilt limit at the beginning of the recovery is different in the inactive state and in the facilitated state, as in previous tests.

Therefore, even in central vision, changes in the receptor according to the state of adaptation to light must be taken into account. When there is a change in the excitation threshold, i.e., in the receptor sensitivity, the same stimulus is detected differently and, consequently, causes very different responses in the nervous centers.

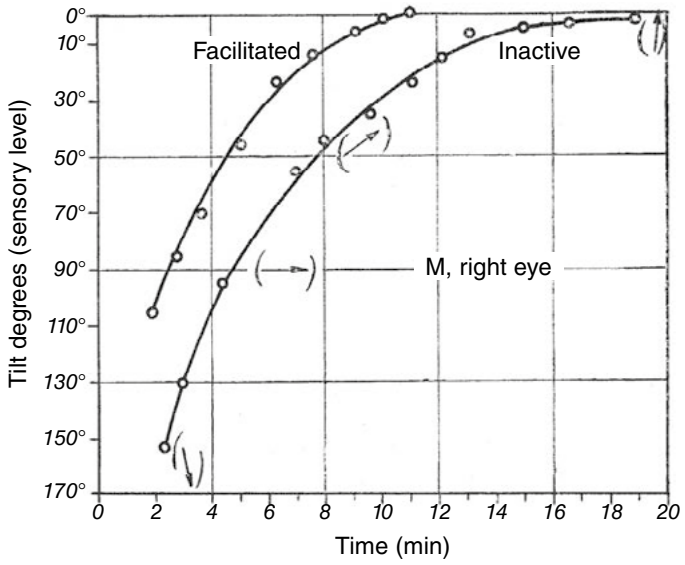

Figure 13.19. Recovery of the orientation of the image after a strong dazzle in the right eye of subject M. As light adaptation to lower intensities is developing, the image of a vertical upward test arrow is straightening until reaching the orientation previous to the dazzle.

The recovery of the initial vertical upward orientation prior to the dazzle is related to the light adaptation curve studied in the M case on very previous pages. However, in the case under discussion, we are not dealing with adaptation to the minimum threshold of light, but to a gradual increase in the visibility of the arrow (illuminated with a fixed intensity), as a result of the process of accommodation to light. At the beginning of the recovery, nothing is seen, but after about one minute and a half under facilitation, the perceived arrow has a diffuse shape and maximum tilt corresponding to that state. In the inactive state, that situation occurs a bit later, after two minutes and a half, and with larger tilt than in the facilitated state. As adaptation increases, so does the re-inversion, which takes place mainly in the phase of rapid adaptation (see Fig. 5.2). This phase lasts three to five minutes in the normal subject, whereas it lasts ten to fifteen minutes in the inactive subject M, and more than twenty minutes must elapse for this subject to reach a level of adaptation that a normal subject achieves in four minutes. Under facilitation by strong muscular effort, intermediate values between those of the inactive state and a normal subject, somewhat closer to the former, are obtained. In any case, although the recovery (re-inversion) occurs in the phase of rapid adaptation, subject M takes a long time to reach the state previous to the dazzle due to the slowness of his nervous processes.

As adaptation increases, the stimulation produced by the arrow has a greater effect; the arrow looks brighter, less green, more defined in its shape and less tilted. This is because the stimulus reaches the nervous centers with greater intensity.

13.5. VARIOUS COMPLEMENTARY TESTS

There are still some tests that should be exposed as a complement to those performed on the dynamics of visual image orientation, although we shall now restrict ourselves to simple descriptions without indication of measurements and experimental curves.

a) Of interest for the behavior in ordinary life is the following test on drawing and perceived orientation. Subject M in inactive state, using his right eye, draws a vertical line with a pencil on white paper, at the accommodation distance and in medium illumination. At the beginning, he does not see the line which is 1 cm long and less than 1 mm thick. If the line is made bigger, making it thicker and 0.5 cm longer, it is perceived, although tilted about 60°. Subsequently, if the line is intensified by drawing it about fifteen times larger (both in width and length) it is finally seen correctly, that is, in its real aspect. Therefore, the perceived orientation of the line depends on its intensity and size. When the line begins to be seen, it is already perceived as much less tilted than the maximum inversion. This is due to the fact that the progression from the tilt limit to 60° occurs with a very small difference in stimulus intensity, as can be seen in the curves of orientation perceived as a function of the stimulus, e.g., in Fig. 13.3. But if the line is drawn very carefully, enlarging and enhancing it very little by little, it is it is found that the line can be perceived with a tilt of about 140°, which is the inversion limit in the right eye for the inactive state, as we know. Later, with an insignificant enhancement of the line, the tilt goes easily to 90° and to 70°. In contrast, it is noteworthy the considerable enlargement necessary (about fifteen times) of the initial line to change from being perceived as maximally tilted to normal (straightened) orientation. Even then, there is still a weak tilt, since normal orientation is never fully achieved monocularly and in an inactive state. A warning of practical importance in orientation tests in subject M is that, if one does not proceed with appropriate meticulousness, the perceived arrow easily goes from a tilt close to 90° to the disappearance of its shape and orientation, thus leaving the maximum tilt undetermined. In connection with this and with the development in time of perception, it was already said that at the beginning of the perception, the time elapsed between the maximum tilt and a tilt of about 50° is so minimal that these orientations cannot be distinguished from each other. Thus, when M looks at an object, he perceives it tilted at less than 90° in the first moments, and soon after he perceives it almost correctly oriented.

b) Other remarks we should make refer to changes in the visually perceived direction of motion. When visual perception of motion was studied by metronome tests in subject M, we saw that the first oscillation perceived (only signs of motion), in addition to being very short in relation to the real trajectory, it is seen in reversed direction of motion. Thus, if the pendulum of the metronome moves from left to right covering about 10 cm, the perceived motion is from right to left and only in the most central region, covering only 2 - 3 cm. In ordinary life, when subject M perceives vehicles or people moving in an inverted direction, he perceives them as

ill-defined, and can guess which objects they are by the size of the blurred spot he perceives. We know that for the inverted perception of motion to take place, poor vision conditions are necessary. That is, the same circumstances apply as we already know for the visual inversion of an object according to its capacity for visual stimulation. Therefore, depending on visual conditions, the apparent direction of a same motion can be different. It will be inverted in very bad vision, but it will be seen transverse to the real direction (tilt of 90°) if vision is somewhat better and, finally, if vision is better still, only a small deviation from the real direction of motion will be perceived.

In the perception of motion direction, the different types of vision should be considered, as in the perception of the orientation of objects. The result is very different depending on the type of vision used. In subject T, the inversion of the direction of motion never occurs since the maximum tilt in his worse eye and inactive state is only about 25°, and therefore, under conditions of minimal vision, motion is perceived with only a small deviation in relation to the real direction. It should be noted that to obtain low vision in the perception of motion, it is sufficient to accelerate it properly, since given the slowness of reaction of these subjects, minimal vision is easily achieved for very fast motions.

Proceeding in this manner, it is shown that in subject M, binocularly and under facilitation by muscular effort, inversion never takes place, and only a very small deviation from the real direction of the moving object occurs. If the motion speed is increased, the moving object is no longer visible, without reaching a greater deviation. In monocular vision and under the same facilitation and initial conditions, a very important deviation can be perceived, greater than 90°. In binocular vision and the inactive state, the deviation is very pronounced, close to inversion, and in ordinary life (vehicles, persons, etc.), it can practically be taken as a reversal of the direction of motion. Finally, in monocular vision and the inactive state (minimum stimulation), the inversion comes up to the maximum; however, in careful determinations, it is verified that it is not a 180° inversion but a bit less, in accordance with the tilt limits we have indicated when studying the types of vision. In short, it can be roughly said that subject M in binocular vision and under muscular effort facilitation does not perceive deviation from the real direction of motion, even with minimal stimulation. Under the same conditions but in monocular vision, a strong deviation from the real direction of motion occurs; and in the inactive state, both in binocular and monocular vision, inversion appears, although a little less in the first case.

In all these tests, to determine the maximum deviation perceived for each type of vision, it is necessary to see the displacement of the moving object, even if it is only in the most central part of the trajectory and there are only faint indications of motion.

In motion directions fully or partially perpendicular to the frontal plane, inversion can also occur under conditions of minimum vision, monocularly and in the inactive state; thus, a motion towards the subject turns into distancing from him, i.e., a motion in depth. The above-mentioned tests deal with motions in the frontal plane, but also in any other plane (e.g., the sagittal plane as indicated) the direction of mo-

tion can be reversed, since it is always the same process, i.e., visual inversion of the trajectory of the moving object, due to an asynchrony in the brain mechanism of visual image orientation.

c) A very different issue is the lability or instability of the orientation of the visual image regardless of stimulation conditions. We have already mentioned that when the test arrow is seen clearly because of appropriate conditions of luminosity and distance, it appears correctly oriented, without (or negligible) deviation; however, such orientation is less stable than one might think at first glance. It is true that if we do not add any other experimental factor, the orientation of the arrow is maintained inalterable, but using certain contrivances we shall see that the orientation can be modified considerably. In this regard, we have prepared the following test for subject M. It consists of placing in front of the eye that is looking at the test arrow, a cardboard with a slot in the middle of which the arrow can be seen. When this slot is rotated in any direction, the perceived arrow follows the rotation of the slot, either clockwise or counterclockwise. It is not that the arrow takes exactly the same orientation as the slot, but rather it is dragged by the rotation of the slot, which demonstrates the instability of the orientation of the arrow even under optimal visual conditions. In this way, a rotation of the arrow of about 90° can be achieved if its initial position without slot is very close to the vertical upward orientation. The tilt can be much greater if the arrow was already seen quite tilted before using the slot; in this case, the arrow always takes the orientation given to the slot. But with the exception of this latter case, there is only some deviation induced by the rotation of the slot, the orientation of the arrow being lagged behind the orientation of the slot. Under facilitation, the influence of the slot decreases considerably, and if initially the arrow is correctly perceived, the rotation of the slot only induces a deviation of the arrow of very few degrees.

With regard the interpretation of the deviation induced by the rotation of the slot, we must first consider the instability or malleability of the orientation of visual image as a result of asynchrony, which makes orientation dependent on the balance between the haptic factor (tactile sensations due to body movements), inducing correct orientation, and the retinal or inverted factor. When the normal mechanism is broken, visual image orientation is adrift, and depending on the stimulation conditions, either correct orientation or inverted one is established, the latter being guided by the retinal factor. Such instability causes that any external factor intervening in the visual sensory field can change the orientation of the visual image, given its lack of firmness. Thus, if the slot regulates the field vision according to a certain orientation, this will influence the perceived orientation of the object. The more labile and unstable the structure of the sensory field, the greater the effect of the slot, therefore the effect will be greater in the inactive state than under facilitation.

Another modification in the orientation by influence of a similar mechanism over the visual field is presented in the test of tubular vision. If for the right eye of subject M, inactive, the visual conditions are such that the vertical upward arrow is seen with a tilt of about 50°, and then he looks at the arrow through a tube 9 mm in diameter and 13 cm in length, the perception of the arrow improves somewhat, it is

seen less tilted, diminishing from 50° to 32°. It is known that looking through a very narrow tube significantly improves vision in a normal subject. Colors appear more intensely saturated, and the details of a drawing become sharper and more distinct. In the M case, the tubular effect should be interpreted as produced by concentration of the functional capability of the visual field in a very small central zone, which would produce an accumulation or enhancement of activity, thus increasing the sensitivity of the receptor and therefore improving the orientation of the arrow.

d) Finally, it is necessary to point out some considerations about the influence of the own body of the subject on the mechanism of visual image orientation, although this topic will be discussed later when dealing with egocentric and allocentric spatial orientation in visual schema. When performing all the experiments concerning visual image orientation under very different conditions, care has been taken that the subject examined keeps his head completely vertical (immobile in his natural position) when looking at the test arrow. This detail is not unimportant since the slightest lateral tilt of the head produces a deviation in the perceived position of the arrow.

This phenomenon has given rise, especially in subject T, to a remarkable tic or small movement of the head that the subject makes instinctively, without becoming aware of it. When the image of an object tilts towards the right side, the subject tilts his head towards the left in such a way that the image appears straightened out and even fully upright in many instances. This tic was very characteristic at the beginning of the study of this subject, and its meaning was not easy to elucidate since he used to make this movement involuntarily for all types of perceptions: visual, tactile, auditory, etc. This is due, on the one hand, to the fact that a tilt to the left of the head and even of the trunk counteracts the deviation of the image orientation to the right and, on the other hand, it is also due to the fact that this movement or tic executed with some abruptness constitutes a summative facilitation. This fact has made that the tic be associated unconsciously to all kind of perceptions, although it has nothing specific regarding facilitation and can be replaced with any type of muscular shake.

Not only in this subject T but in other two brain-injured people different from those studied here and with similar disturbances, we were able to verify the compensatory tilt of the head, although for different circumstances, it has not given rise to the tic that subject T presented for a long time. People with brain injuries who do not have a significant visual orientation disorder can correct any image deviation by compensatory tilt of the head and even the trunk. When they look at an object, they usually try to adapt the position of the head in relation to the orientation of the image perceived. To this end, they perform a series of trial and error movements until they find a position that allows them the best possible vision and orientation.

One of our patients (not M or T) not only tilted the head to the side opposite to the deviation of the visual image but he also bent at the waist, curving strongly to one side to achieve an optimal straightening of the image orientation. But when the orientation disorder is relatively small, as in the T case, and the deviation can be counteracted by a small lateral tilt of the head, an associative mechanism is easily estab-

lished involuntarily, which ends up accompanying every kind of sensory perception, although in many cases it acts only as facilitation by muscular effort. In this way a tic arises, somewhat different from the contouring movements described by Goldstein and Gelb (1918) in their Schneider case. In the cases above described, the tic acts by the mentioned double action of counteracting the tilt of the visual image and by central summation.

The phenomenon of counteracting the deviation by the opposite tilt of the head reveals the disaggregation of the brain mechanism of visual orientation. In a normal subject, head tilt does not cause any deviation in the orientation of the visual image, except in very special conditions, for example a bright line seen in the dark, i.e., the Aubert phenomenon (Aubert, 1865). It can be said that these brain-injured patients present this phenomenon in any situation without the need for experiments in the dark.

Hence, the maintenance of the natural upright position of the head in visual orientation experiments is of utmost importance for correct determinations. It should still be noted that the re-inversion of the visual image by lateral deviation of the head is very diverse according to the intensity of the brain disorder. Thus, in subject M inactive, it appears at its maximum, greatly diminishing its effect by facilitation. For subject T inactive, it is much less than for subject M under facilitation, and is more intense using his right eye than his left eye.

14. Theory of visual image orientation

14.1. FUNCTIONAL COMPLEX OF VISUAL IMAGE ORIENTATION

We have already indicated at the beginning of this study that the orientation of the visual image can be considered a spatial localization; it is a complex function that easily succumbs and disintegrates because of the dynamic conditions that originate asynchrony of the nervous elements of the cerebral cortex. The phenomena resulting from such disintegration or asynchrony depend on the sensory factors taking part in the formation of the functional complex of visual image orientation.

Here we limit ourselves to the most essential aspects in order to obtain a rational explanation of the experiments described above. It is known that the issue of visual image orientation has given rise to innumerable discussions and multiple studies, mostly because of the inverted image on the retina. Opinions of all kinds can be found. There are also authors, such as Mach (1886/1906), who think that this kind of controversy is pointless, since the fact that the image is correctly oriented despite the inversion in the retina is a pseudo-problem, and does not merit the endless discussions to which it has given rise. According to Mach, the essential fact is that all sensory systems of spatial character, no matter how different, are related by a common functional link, that is, the movements used for orientation. Thus, the same spatial orientation is established for all kinds of sensory systems since they jointly participate in spatial perception.

Some classical authors already mention the visual-tactile functional complex that is involved in visual image orientation, and for those with an empirical conception, visual orientation seems to depend entirely on tactile support, the visual factor playing no role. Visual orientation is then conditioned by touch, understood in a wide sense (movements, static sensations, etc.). That interpretation is adopted by authors of the 18th century, for example Condillac (1754/1821), and those of the late 19th century such as Helmholtz (1867/1896). Likewise, the important experiment of Stratton (1896, 1897) seems to support this view; it does not matter if the image formed on the retina has one direction or another, since in both cases the proper

correspondence between visual and tactile sensations of movement is acquired through experience, and the result will always be the same.

In such theories, visual image orientation is established entirely by the tactile component, thus the visual factor, by itself, is overridden as regards orientation. This means to discard fully and definitely the question of retinal inversion. Nevertheless, since opinions of all kinds are not lacking, we could also mention, as a curious detail, in favor of the visual factor (visual image orientation provided by the retina), the opinion of Buffon (1880), who imagined that newborns, lacking associations or proper references, would have inverted vision because they would use the information from the retina for spatial localization. Leaving the appraisal of this hypothesis aside, it happens that the issue of visual image orientation and the old associated problem of the inverted image on the retina acquire a new perspective in view of the phenomena about pathological inverted vision studied here. In this way, the retinal factor turns back to recover its meaning because it is able by itself to decide visual image orientation, since the inversion in our brain-injured patients has to be put in relation to the inverse localization of the spatial retinal local sign.

The theory of the functional visual-tactile complex, according to which visual image orientation is determined by the second factor (touch), is still acceptable on condition of recognizing that the visual factor can also determine the orientation, a condition that has been generally denied by all supporters of the commonly accepted theory of the mechanism of visual image orientation. In the pathological cases with inverted vision, something similar to the first phase of adaptation in the Stratton experiment occurs, that is, incongruent orientations for sight and touch coexist until adaptation is acquired again and correspondence between sight and touch is recovered. However, in our pathological cases the genesis is very different; sight and touch are in opposite orientation because the retinal factor gains autonomy and conditions by itself spatial orientation of visual sensations. The emergence of this autonomy is due to brain asynchrony, which breaks the functional link between the visual factor and tactile factor. However, if the stimulation intensity becomes much higher and the whole brain mechanism of the visual-tactile complex comes into action, the correspondence between the orientations is obtained again, and the orientation determined by the retinal factor is excluded.[1]

14.2. DISAGGREGATION AND DEGRADATION IN VISUAL IMAGE ORIENTATION

As we know, nervous asynchrony gives rise to the fundamental experiment of functional time lag and dynamic reduction. Functional complexes become disaggregated and simplified, and in the case of visual image orientation, the orientation of the image is no longer adapted to tactile orientation, body movements, etc. Consequent-

[1] This issue, along with other inverted perceptions such as tactile perception, is more fully reviewed in Sec. 26.2 in Vol. 2. Visual inversion is discussed in relation to the secondary areas at the end of Sec. 4 in: GONZALO, J. (1952). *Trab. Inst. Cajal Invest. Biológ.,* XLIV: 95-157. (Supplement I of the present edition.)

ly, we could say that visual image orientation is as if it were drifting, with no other rule of orientation than that provided by the visual system itself, thus manifesting the retinal inversion.

The inverted orientation is therefore the result of a dynamic reduction of the activity of the visual-haptic complex, in such a way that the simplification (or reduction) of the mechanism of visual orientation excludes the secondary or less immediate factor (tactile factor). The simplification of complex functions, which means their *disaggregation* and *degradation,* is the rule in brain alterations; and here, in the case of visual image orientation, we again find similar conditions to those of green chromatopsia in the perception of white. In this last case, due to a considerable difference between the excitabilities of the primary colors (these ones needed to generate white by their joint stimulation), white cannot be obtained, and a pale green appears in its place. Analogously, in the case of visual image orientation we are facing a difference between the excitability for normal and inverted (or variably tilted) image orientation; and instead of the unitary function that reacts according to all-or-nothing, a wide interval is established between the factors that make up the functional complex.

At the lower end of the interval is the isolated visual factor that originates inverted vision, and at the upper end (highest level) is the correct vision caused by the unitary action of the brain mechanism in visual image orientation, i.e., by the tactile support. Depending on the intensity of stimulation, the respective sensation level is reached, and either visual inversion (retinal action) or normal vision (tactile or haptic action) is obtained. This pathological interval creates a new situation that has no antecedent in the sensory activity of a normal subject, thus appearing visual inversion in the same way as green chromatopsia in the case of colors and as replacement of white.

The magnitude of the abnormal interval depends on the degree of asynchrony, which is not always so pronounced as to cause a complete disaggregation of the visual-tactile complex. Even in subject M, a complete 180° inversion is not exactly reached, and if the cerebral deficit is compensated by various central summations, the inversion limit is greatly reduced and only moderate tilts appear. We have already seen that depending on stimulation conditions, it is possible to gradually go from the inversion to the correct image orientation, following specific sensory laws. Thus, a certain balance is always stablished between the two modes of orientation, correct and inverted, depending on the intensity of the visual stimulus. If a deviation occurs, even if small, it means a failure of the haptic factor (which enables the correct orientation) and at the same time a certain autonomy of the retinal factor. If there is complete inversion, it means that only the retinal factor is acting, the tactile or para-visual influence being totally excluded.

In cases of severe disorders, such as M, even without total exclusion of the tactile factor since inversion is somewhat incomplete, the orientation of the visual image does not easily go from one extreme to the other in the interval mentioned above; there are always a few degrees missing to reach a completely correct orientation even if stimulus intensity is greatly increased. Asynchrony is so pronounced in this case that even with intense stimuli it is not possible to recruit all the nervous elements of the tactile factor. The same happens in the case of white, which does not

appear completely clean of a green hue, even under the most intense illumination. However, with facilitation, the magnitude of the interval (or asynchrony) is much less, and normal orientation can then be achieved with appropriate stimulus intensity.

Since visual activity is altered in its totality and the different functions present the same asynchronous disorder, alterations in visual image orientation do not remain isolated from alterations in other visual activities which appear simultaneously. Let us recall that when the image is tilted, the greater the tilt, the greater the alteration of color and shape.

The empirical rule that a correct orientation can be expected in sharp vision already indicates a solidary activity, and also leads easily to the idea that sharp vision and correct orientation of the visual image are approximately isochronous functions, i.e., of the same excitability. Thus, in our subjects, as soon as color irradiation destroys or disaggregates image details of the test arrow, a deviation of its orientation appears.

Disaggregation and degradation of sensory activity do not represent a sensory chaos, but they manifest themselves according to the dynamic reduction mechanism, that is, sensory activities appear in a particular sequence according to their new characteristics of nervous excitability; those of greater complexity being the most difficult to be stimulated. The general law of Fechner (sensory perception proportional to the logarithm of stimulus intensity) (Fechner 1860), is still valid along the new abnormal interval that includes such sensory activities. Therefore, there is no sensory chaos or annulment of fundamental laws, but another order with the same laws, in other words, a nervous organization at a different scale from the normal one.

As for the nosological meaning of inverted vision and other similar alterations, they do not represent independent entities or syndromes, and only represent multiple aspects of the general disturbance suffered by visual functions due to a single lesion. Moreover, it is not even necessary for the lesion to be located in optical or para-optical areas since, due to the dynamic repercussion caused by the joint activity of the entire cerebral sensory cortex, a parietal lesion, for example, easily alters visual activity and, with it, visual orientation of the image. It should still be noted that we have observed alterations in visual image orientation in both left and right brain lesions, and in both cases in both eyes.

For the moment, without pretending to have conducted a complete study on the orientation of the visual image, we put an end to this issue.

Schema in visual perception

15. Schema in visual forms

15.1. COGNITIVE SCHEMA

The cognitive schema corresponds to the highest degree of organic differentiation of sensory structures by accomplishing a stylized and more properly characteristic perception of objects and sets of them. Cognitive schema makes it possible intelligent behavior, that is, abstraction. The function 'schema' does not imply any singular activity; it is derived directly from the shape structure of which it constitutes a special feature.

Although it is now impossible to determine experimental curves of the phenomena, given the nature of this topic, it is feasible to perform certain dynamic variations by means of facilitation that allow this topic to be studied in depth, in the same way as in the previous study of visual perception of shapes.

As we have observed when studying the perception of objects, these are almost always correctly interpreted by subject M provided he counts on sufficient time and favorable conditions for vision. However, his perception under unfavorable conditions does not occur in an immediate or unitary way, but by means of successive partial aspects. In many instances, the correct interpretation is reached mainly through characteristic details (e.g., the handle to recognize a key) without getting a clear idea of the whole object under examination. If agnosia can often be overcome by indirect understanding tricks in the case of physical objects, the situation is very different when it concerns the understanding of schematic drawings of these objects.

It is well known that patients with visual agnosia fail much more frequently in understanding drawings of objects than in understanding those same physical objects when they look directly at them, in which case they hardly make any significant mistakes. It means, using the nomenclature of brain nosology, that visual *asymbolia* occurs more easily than visual *agnosia* of objects. The distinction between the different behaviors when subjects are faced with physical objects and schematic drawings is of utmost importance for the correct understanding of visual agnosia, using this term in a general sense. Comprehension of physical objects and comprehension of schematic drawings are successive phases of sensory organization which are altered

simultaneously in agnosia, and only by meticulous examination and appropriate testing is it possible to clearly discover agnosia for physical objects. By contrast, the defect in schemes is much more easily revealed in view of their shortened or summarized character and the imaginative effort required for their understanding. Schemes are originated directly from physical objects through a stylization process that gives rise to "*strong forms or figures*" in the language used by the Gestaltists. The demonstration that such stylization exists and it is not pure explanatory phantasy can be found not only in certain experiences of simplification of forms such as in tachistoscopic vision or when they are represented in the imagination, but also in the fact that representation through drawing would be impossible without it.

The disaggregation and diffuse conception that we have already observed when previously studying the perception of forms and objects, is much more pronounced and with very remarkable features in schemes, as it results from the following tests with simplified drawings to be recognized by subject M and subject T.

When M is faced with the drawings of Fig. 15.1, in binocular vision and good illumination, the following results are obtained.

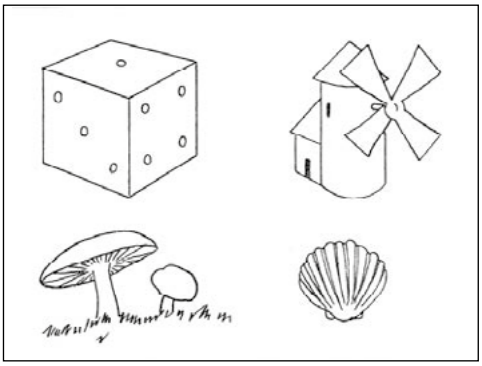

Figure 15.1. Simplified drawings to test cognitive schema. In general, they are well recognized by subject M under facilitation by muscular effort, whereas failure is the rule in the inactive state (see the text).

With respect to the die, M in inactive state does not know, he declines to interpret the figure after having looked at it intensely. Finally, he believes it is about a house with windows, but he cannot be sure. M under facilitation by muscular effort takes just one second to answer "it′s a die."

With regard to the windmill, M inactive only perceives a house, and when asked about the sails, he finds it very hard to catch sight of them. First, he only sees the two sails on the right side. He confuses the other two sails with the drawing of the house. They are perceived *in a different way*. At last, he succeeds in recognizing all of them independently from the rest of the figure, but he does not get to see anything but "a house and a cross." The meaning of the whole escapes him. Under facilitation by muscular effort he realizes after a short time that it is a windmill.

As for the mushrooms, M inactive perceives them as trees, but under facilitation the interpretation is correct.

As for the sea shell, M inactive at first does not know, a bit later he takes it for a fan. Under facilitation he says "a clam."

When subject T is faced with these same drawings shows a certain deficit; nonetheless, using only his right eye, the one with worse vision, he interprets correctly the die and the shell, although the latter perceives it at first as something shapeless. As for the mushrooms, he believes they are just "a flowerpot," and as for the windmill, he makes a misinterpretation of the same type as that made by subject M. Initially, he only perceives a house, but realizing that his interpretation is not quite right, he tries new exams of the drawing. The sails have gone unnoticed to him, partly confused with the figure of the house, as in the M case, and only after much attention, when asked about their presence, he answers: "It is a sentry box and a cross" (he has been a soldier for a long time). He also thinks of a church and a castle, and long after he understands it is a windmill. All these difficulties do not appear in binocular vision, the interpretation being then quick and correct.

Figure 15.2. Simplified drawings to test cognitive schema. In the M case, although an interpretation close to the correct one is achieved under facilitation by muscular effort, mistakes still persist in all the drawings (see the text).

When M is faced with the drawings of Fig. 15.2, in binocular vision and good illumination, the following results are obtained.

As for the candle, M inactive finds very difficult to know what it might be, even after a long time; it seems to him it might be a tower or a tree (cypress?), and he finally opts for a chimney. Under facilitation by muscular effort, his perception does not improve much, and although he does not reach the correct interpretation, his understanding of the drawing is enriched to a certain degree; thus, he thinks of a smoking chimney or a fountain for street decoration.

As for the weight, M inactive says "a bottle," and under facilitation, "a glass jar." Facilitation does not correct the defect although the response seems a bit more appropriate than in the inactive state.

As for the cheese, M inactive says "something … ," and he cannot say anything else. But under facilitation he says "it's not a thing, it looks like a die, but it's not sure."

As for the whistle, M inactive thinks it looks like a shoe or also a bottle. Under facilitation he says "something like a bottle, but it's not sure."

In these other figures, it is verified that even in binocular vision and under facilitation by maximum muscular effort, the correct interpretation of the schematic figures remains impossible for subject M, although more or less plausible and approximate interpretaions are achieved. The perception is more accurate with facilitation than in the inactive state, but in any case, the difference in perception between these two states is not as clear as in the drawings of Fig. 15.1, which are however of the same type of simplification.

However, subject T, even using only his right eye, recognizes the four drawings of Fig. 15.2 with relative ease.

With regard the drawings shown in Fig. 15.3, when subject M is faced with these figures, in binocular vision and in good illumination, the following results are obtained.

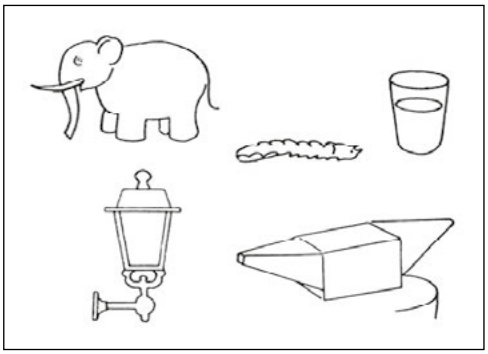

Figure 15.3. Drawings to test cognitive schema. Remarkable illusions or figure deformations are perceived by M, disappearing under facilitation by muscular effort (see the text).

As for the elephant, M inactive says "a dog," but under facilitation says "elephant" without any difficulty. In the inactive state, he did not see the trunk and the head seemed to him to be prolonged towards the tusks. Under facilitation, this illusion disappears and in addition he perceives the tail well.

As for the street lamp, M inactive says "a pitcher," and under facilitation "a street lamp." In the inactive state he does not pay attention to the device holding the lamp, which changes its meaning, but under facilitation the attention is equally distributed throughout the figure.

As for the caterpillar, M inactive does not know and says "perhaps it's a spike," but under facilitation says "worm"; he distinguishes well the head and even the horns.

As for the glass with water, M inactive says "two glasses" (one inside the other!), and under facilitation "glass with water." The change in his interpretation is remarkable.

As for the anvil, M inactive has no idea, even after a long time. Under facilitation, he has sometimes failed, renouncing to any interpretation, but occasionally has achieved the correct interpretation. It seems that he does not pay enough attention to the pointed ends and concentrates on the central block.

Regarding the T case, he makes similar mistakes, also due to paying or distributing the attention heterogeneously on the drawing. Such mistakes are more easily made in monocular vision, but much less so in binocular vision and provided there are good conditions of luminosity and distance. For example, in monocular vision he takes the elephant for a wild boar, whereas in binocular vision he quickly realizes that it is an elephant. It seems that in the first attempt the trunk went unnoticed to him, hence he got the optical illusion of perceiving a boar. In the case of the glass, using only the right eye he makes the same mistake as the previous subject by thinking that there were two glasses (he says "one glass … and a smaller one inside"), whereas with the two eyes he says "glass with liquid." In the case of the caterpillar, he only sees "one line … two lines with sharp point making curve … ," even binocularly. Other figures, such as the street lamp and the anvil, although he generally interprets them correctly, has sometimes failed in tests on different dates, thus, he has taken the anvil occasionally for a Civil Guard three-cornered hat, and the street lamp for a watchtower, which means not having paid attention to the support of the lamp which is what gives its character. Sometimes he has also thought that it is a jar; all confirming that his perception has been fragmentary. However, when insisting, he always comes to understand the figure correctly ("of this … that illuminate the streets … a street lamp"). There are, then, undoubtedly disturbances of the same type as in the M case, that is, disaggregation and fuzzy understanding, although attenuated.

The deficit in understanding such schematic drawings is due to the peculiar alteration in the organization of forms, and not merely to defects in visual acuity, defects that must also be taken into account. This is confirmed by the behavior of these subjects when faced with overlapping drawings, as in the Poppelreuter figure (Poppelreuter 1917) shown in Fig. 15.4.

Subject M sees only many lines in that figure at first, and desists from interpreting it. By insisting and encouraging him to pay more attention, his perception in the inactive state, in binocular vision and with good illumination is as follows. After quite a long while of looking and searching, it seems to him that there is a jug. He is told there are more things to find, and after a long time, he only gets to perceive part of the cleaver that he takes for a saw. He thinks that the rest of the figures are drawings or ornaments of the jug. These tests, and especially the figure of overlapped drawings, bother him greatly when he fails, and he complains strongly. Paying much attention and patience, sometimes he has been able to find, after many minutes, other objects in the figure, such as the cleaver and the hammer, but not the iron which appears particularly difficult for him. In any case, he loses many details that he does not properly understand, e.g., he considers the handle of the jug as something different and independent of the jug because he had not perceived it in the first recognition of the jug.

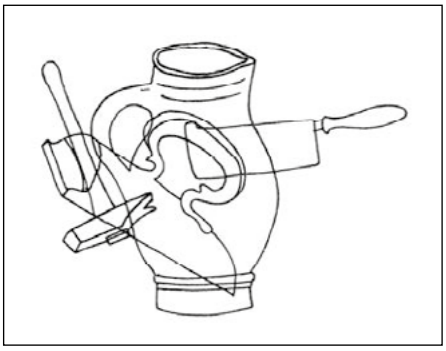

Figure 15.4. Poppelreuter overlapping figure (Poppelreuter 1917) to test visual agnosia (see the text).

Under facilitation by muscular effort, his perception becomes firmer and he finds successively the various objects. First the jug with its handle, then the cleaver or handsaw or sawblade (he cannot be sure which of the three would be the correct answer). Occasionally, in the inactive state, he takes the cleaver handle for the jug handle, but this never occurs in the facilitated state. Next, he usually finds the hammer, which he perceives only exceptionally before the cleaver, and afterwards some difficulty appears, even under facilitation, to find the iron, and after a long time he says "another thing with a handle … an iron." It is necessary to indicate that initially, when he has shown resistance to interpret the figure because it seemed very difficult to him, the objects to be looked for (hammer, cleaver, iron) were named to encourage him to look for them, which has undoubtedly facilitated his task; otherwise it is possible that they had remained unrecognized.

Subject T also presents serious difficulties in understanding this figure; on one occasion he saw only "a drum with sticks, everything mixed up," without attempting to interpret anything more. On another occasion, he perceived a jug with two or three handles (the ones sticking out). It is remarkable and quite characteristic in subject T as well as in the previous one (M), the naturally way they respond and the seemingly certainty of having been right. When subject T is asked about the other drawings on the figure, he says, after some thought, that the jug is cracked. All this occurs commonly in monocular vision, which as we know corresponds to a functional level slightly higher than that of subject M in binocular vision and under facilitation by muscular effort. In binocular vision perception improves, but still he hesitates long before interpreting the overlapped drawings of the figure. In addition, he makes many lateral movements of his head and moves or turns the drawings as if trying to find favorable positions for the interpretation, apart from the fact that, as we have said, the movements or shakes of his head favor visual function by summation. Finally, he finds the hammer and then the cleaver, but fails with the iron, and only by telling him his name and that he must find it, does he find it after some time of searching.

This last test of the overlapped drawings shows even more clearly than the previous figures that, in fact, the mistakes in the recognition of schematic drawings are due to true misunderstandings or illusions in the appreciation of the configura-

tion because, given the instability of the perception, there is a tendency to formation of figurations that respond to a simpler state of equilibrium in the sensory field.

The data shown on the behavior of these two subjects in the interpretation of schematic drawings are only the most characteristic extract among the numerous tests carried out during the several years that they have been examined. Therefore, the answers shown have not been obtained at random, but represent a very clear and constant indication of the agnosic disorder in these subjects. It is clear from the above tests that agnosia in subject M is very evident and does not disappear completely even with facilitation. For subject T, the behavior is better than that of M under facilitation, but there is still a significant deficit.

Regarding the Schneider case of Goldstein and Gelb (1918), the authors point out that when the patient inspects the figure he indicates the various characters but does not understand the figure in its entirety and only interprets partial aspects, which segregated from the whole acquire a new meaning. These authors, adopting the viewpoint of the Gestaltists, highlight the confusion and instability of the background and shape in the figure, in such a way that new aspects appear that the normal subject would never see. In more recent studies, Gelb (1937) attributed most of the alteration to a fragmentation of shapes as well as to a reduction of meaning or pregnancy (gestaltist term related to meaning) in their organization. All these circumstances are also present in our two brain-injured patients, M and T, and in some others we have studied suffering from visual agnosia. The interpretation of Schneider's supposed motor recognition, which must be discarded even if it is still erroneously admitted in later publications, does not change the intrinsic phenomena of schematic figure recognition, although it overlooks the all-important question of nervous excitability disorders.

Considering all the results of the previous tests, it must be admitted that the wrong interpretation is mainly due to a change in the configuration, i.e., to a metamorphopsia which leads to a metamorphopsic pseudo-agnosia, as we have indicated when dealing with shape perception in previous sections. One might think that the subjects invent the answers at random, but this would be a very superficial assessment, and nothing even remotely supports it when the nature of the various responses and, above all, the responses as a whole are examined closely. A remarkable feature we have already pointed out is the attitude of conviction with which they assume certain faulty or somewhat approximate interpretations. This suggests that they have indeed perceived a certain illusion in the figure, which for a normal subject would be impossible, but in them easily takes place given the instability of the sensory organization. Therefore, there is no fortuitous invention, but an *altered view* of the configuration of the drawing. In other words, the patient usually answers in accordance with what he is capable of perceiving, and if it looks like an unfounded invention, it is due to the fact that figures lend themselves to be seen under new aspects. However, the impairment in understanding schematic drawings does not lie only in the perceived distortions; there is also an imaginative difficulty, that is, a deficit in the *construction of mental representations* from highly stylized or simplified drawings, which would constitute the intrinsic disorder of schema. In any case, a rigid separation should not be made between perceptual alteration (alteration of what has been seen) and agnosia in the strict sense of the word (constructional deficit). Both

contribute equally to the failure in understanding schematic drawings, and although they are of a somewhat different type, they are stages that condition each other in the organization of shapes in the sensory field.

By examining more closely the circumstances involved in this disorder, it can be noted that some factors influence each other and contribute jointly to determine the alteration. Thus, we find *illusions* or deformations of the figure (an 'a' is seen as two superimposed zeros, the elephant is perceived as deformed, etc.), *disaggregation* or fragmentation of the whole (the case of the windmill broken down into a house and a cross, the case of the street lamp, etc.), and also *inability to construct* or imagine a proper shape from schematic lines (example of the cheese, candlestick, etc.). All these factors can occur separately, depending on the kind of drawing, or simultaneously as in the Poppelreuter figure wherein the overlapping drawings increase the instability of perception and understanding, causing both illusions (jug with several handles) and disaggregation (cracked or decorated jug), and also difficulty for appropriate imaginative construction.

In short, perception is diffuse and labile, so the balance of shapes is altered and, consequently, their phenomenic aspect, as it occurs in visual-geometric illusions in normal subjects, although here it is due to other reasons. There is also constructive inability, e.g., difficulty in imagining relief in flat drawings without shadows, deficit in the assessment of the whole, etc. Thus, there is both a reduced sensory activity in perception and in the imaginative construction, and both are expression of the same fact, *reduction of pregnancy (gestaltist term related to meaning) in the organization of shapes*. Mere perception and mental construction are both continuation and reflection of each other and, ultimately, aspects of a same process whose basis lies in the functional reduction of the sensory field. A radical separation or distinction between both would then be rather contrived. The facilitation phenomenon, which in the M case modifies in a very important way the functional level, improves clearly the perception of schemes due to the reorganization that is established in the sensory field, this one broadly understood as the set of sensory organization processes.

15.2. VISUAL BEHAVIOR

An alteration in the cognitive schema distorts to a high degree the intelligent behavior of these subjects, since this kind of activity, intelligence, is solidary of the structure of the sensory field. Following the Gestaltists, there is no possible distinction between sensory and intellectual functions, to the point that it might be admitted that "intelligence is a spontaneous mode of organization." Thus, the general alterations we have found in the conceptualization of schematic figures, again participate in various alterations of higher visual behavior giving rise to what is commonly considered a visual intelligence disorder. Such alterations are produced, for example, in the recognition of situations or scenes in drawings, in the construction of drawings and puzzles, etc., and very especially in the abstract behavior regarding colors, i.e., everything related to color agnosia. We shall deal with these topics succinctly, by restricting ourselves to the essential aspects.

A characteristic example of agnosia in subject M, taken from ordinary life, is the recognition of banknotes. These are recognized not by their value numbers printed on them but by the general format, size and ornamentation aspect. Thus, he knows that those of small size have a certain value, the largest and longest ones have another value, those with a nearly square shape have a different value, etc. In many cases, the figures illustrating them are recognized more or less correctly, but a typical difficulty is to find the numbers that indicate the value of the bill since they are in the middle of all kinds of arabesques which seriously alter their recognition. Thus, for some types of bills, it seems impossible he finds out the numbers in spite of being visible in a variety of ways. When he tries to find those numbers, he seems to be in a situation similar to that of a normal subject in the face of an ambiguous image (Vexierbild by German authors) within which there is another hidden figure different from the one seen at first. This difficulty appears even in the best functional visual conditions, that is, in good illumination, at as near vision as necessary, binocularly and under facilitation by muscular effort. But these conditions do not prevent the defect to subsist, and the numbers are almost impossible to be perceived. When, in view of this result, he is shown the drawing containing the number 5 on certain five-pesetas bills (see Fig. 15.5), the situation does not improve. He only sees many lines, and by assuring him there is a 5 and he should see it, no matter how hard he tries, he does not see anything other than a 3. He only perceives the strongest shading of the number 5 which, indeed, resembles remarkably a 3 if some fragments are omitted. The rest of the drawing of the number 5 remains mixed up with the background, without acquiring individuality, making impossible for him to determine its shape, no matter how much help be given to him. He affirms that he only sees the indicated number 3. In short, in this test, the subject even in his optimal functional state cannot distinguish or segregate the figure of the number from the ornamental background; he can only isolate the more solid shadow which adopts a different meaning. Therefore, there is a marked organizational deficit in perception due to its instability and, especially, its diffuse character.

Figure 15.5. Bill of five pesetas.[1] Subject M binocularly and with facilitation by muscular effort cannot see the '5,' but only a '3' corresponding to part of the shading of the '5.'

[1] The peseta was the currency of Spain between 1868 and 2002.

These same facts are demonstrated in many other examples such as in incomplete drawings of the face of a person. In the inactive state, the defect goes easily unnoticed, and even if the mouth or the nose is missing, he recognizes the figure without noticing any defect, due to a diffuse perception. However, by means of facilitation by muscular effort, the defect is immediately pointed out. It is also the case that the negative and the positive image of a same photographic portrait of a person are taken as identical in the usual state of the patient. He also does not distinguish between a photography taken from a living person and a photography taken from a sculpture, he only recognizes that in both cases there is a person. In photographs of big size and pictures easily visible by the subject, he only perceives the general characteristic and always responds "one person," but he hardly indicates gender or age. For that purpose, he is guided by details, long hair in women, moustache and tie in men, etc. For this reason, in an old picture showing a man with long hair, he thinks the person is a woman, even though he has been made aware that he has a moustache, but he is not able to solve the problem and has no interest in giving further explanations. All these difficulties disappear automatically with facilitation by muscular effort, and all the peculiarities of portraits, drawings, prints, etc. are perceived. In this case the perception becomes more defined and secure, as we know from other tests. However, in certain tests, some difficulty arises even under facilitation, both in subject M and subject T. Thus, these subjects generally fail when trying to distinguish between very simple drawings that are a little different, because they seem to them visually the same.

Much greater difficulty is shown in the well-known ambiguous images (drawings with double meaning), i.e., figures in which the whole configuration can be changed by varying the attention or manner of looking at them. It is clear that already for the normal subject, certain difficulty arises, and it is no surprise that these subjects fail when they try to move from a type of visual configuration to a very different one, since it is already enough achievement for them to grasp the most evident or immediate configuration. The remarkable thing in them is rather the difficulty to admit the double meaning in spite of all explanations and aid given to them. There is a sort of adhesiveness to the first meaning which prevents the transit to a new understanding. Such mental viscosity or stickiness (called *Gebundenheit* by German authors) is very typical of mental operations in all kinds of brain diseases and, particularly, it is very pronounced in brain-wounded patients suffering from agnosic disorders. It is more patent in subject M than in subject T who, by means of very detailed explanations is able to understand the double meaning of a simple ambiguous image.

A not easily accessible exercise for these brain-injured patients is putting together a puzzle, no matter how simple it is, for example an almost life-sized face, in color, divided obliquely into four parts. Subject M desists from the first moment to do the test in view of the difficulties. In another test on a different date, when he finally achieves a certain regularity in the construction of the figure, it results incorrectly oriented in its whole, although he believes it is correct. Subject T also shows similar but more moderate difficulties, which vary according to the complication of the test.

In the constructional difficulty concur both perceptive and schema defects. In the first group we consider, for example, in addition to the deficit of shape appreciation, the disorder in visual image orientation, which can hinder the orientation of the fragments, and also a somewhat different kind of defect that we call orthogonal disorder, discussed further below (see Sec. 16). As said, the intrinsic disorder of schema acts as well, leading to an ideational agnosia in the sense given by Liepmann (1908), which makes construction very difficult because the schematic conception is precisely here essential and indispensable to arrange the parts.

Similar considerations could be made regarding the difficulties to copy drawings, which reveal disorders of the type of *optic ataxia* and also of the *constructional apraxia* of Kleist (1911) in either of the two subjects M and T.

Finally, in the tests with the well-known sheets of Binet (1903) for clinical examinations of intelligence, the complex agnosic disorder of these subjects can be clearly seen.

Figure 15.6. The sheet of Binet (1903) "Snowball." Subject M interprets separately and somewhat incorrectly the different figures, but the meaning of the scene escapes him. He does not find any explanation for many of the details, others give rise to illusions, e.g., he believes that the boy's cap near the window frame is a broom. With facilitation by muscular effort, the partial understanding improves a little, without important advances.

Subject M, in the first sheet entitled "snowball," shown in Fig. 15.6, gives the following interpretation: "one man scolding another man, grabbing him by the hair

and pulling upwards … and another one leaning out of a door or window (he refers to the hidden boy), I don't know what it is, window or door; … when he lets go of the other one, he is going to catch him." He is asked "what is going on up here?" (indicating the left angle to him). He answers "a woman or a man behind a window, and here there is another small window but there is nothing." Asking him again "and what is this?" (pointing to the boy's cap), he responds "I don't know what it'll be." After a long time, he says that it looks like a broom because he extends it upwards along the window frame, and asks if it really is. Questioned about the boy's blackboard, he thinks it is a book.

These results are obtained after looking closely at the sheet for a long time and helping him to pay attention at what he should see. He does not understand the meaning of the scene at all, mistaking the boy with the adult, the woman with the man, etc. Under facilitation by muscular effort, everyone is better distinguished, but he continues ignoring what had happened, and even the illusion of the cap is maintained for a while or, in any case, he does not know what it could be. There are therefore perceptual defects, and also an inability to understand the situation as a whole since only partial aspects are interpreted, which shows the inability to construct a schema or idea about the scene represented.

Subject T, although a bit better, does not go farther either in the interpretation. He answers "the mother is hitting the child and another who is hidden." He does not recognize either the snow that is hanging, or the blackboard or the cap that is falling down. Feeling that his answer is not well received, he adds "she grabs him by the hairs for him to go to school." As in other tests of this type, subject T resorts to all types of head movements and involuntary efforts when examining the scene. In the other two sheets, subject T succeeds relatively easily in the second one (it is the game blind man's bluff) and he fails in the third sheet (the gentleman's greeting), more difficult, since he says "he is talking to his girlfriend and he has a fight with someone, and a woman arrives and separates them," although he gets it pretty close. Subject M with facilitation by muscular effort, and in a very indirect way, understands the scene of the second sheet, although many details escape him. Regarding the third sheet, he does not properly understand who the people on the scene are, and he does not get the meaning of the situation at all, although he tries different explanations.

In these tests, apart from a diffuse perception, which harms the recognition of important details for a full understanding, the main defect surely lies in the inability to pay attention to the whole, i.e., to integrate it into a unit, as we have already noticed in previous tests (e.g., in the windmill and the street lamp), although the scenes of Binet are more complex. In any case, we find different degrees of a same type of functional disorder. This inability to integrate or apperceive the whole scene hinders the understanding of the situation. Understanding can also be done successively, as we mentioned when studying the perception of forms and objects. However, in this way only juxtaposed aspects or partial actions are grasped, at most, without reaching the true global meaning. Therefore, the defect is not only a lack of unitary visual perception, but more exactly, difficulty of constructing an ideational schema corresponding to the whole of the scene. There is, then, a constructional inability, like in

the case of putting together a puzzle or copying a somewhat complicated geometric drawing, the figure of a house, etc. This kind of disability occurs in the so-called *simultagnosia* of Wolpert (1924) in which the understanding of the set of objects representing a scene fails, while each of them, individually, can be recognized quite well.

As for the ability of visual mental representation, i.e., the ability to evoke images in the absence of the object in question, it is not abolished, even in subject M, although it can be slow and defective. Therefore, agnosia for mental visual representation (Charcot 1889) is not present in our subjects, contrary to what Goldstein and Gelb (1918) argue about their patient Schneider, in whom the difficulty for mental representation would play an essential role in the genesis of most of his disabilities, according to these authors.

In relation to the alteration of the cognitive schema, i.e., the inability for abstract ideas, we must mention the manifestations of *color agnosia* in our two subjects, namely, anomalies in understanding the name of colors. Besides the fact that colors are altered from a sensory point of view (dischromatopsias, chromatopsia, etc.) and also spatially (flat colors), our subjects present a disorder of agnosic type that fits very well with a failure in the cognitive schema. When our subjects can distinguish colors from each other, they often cannot name them. The patient in question spends a long time until he gets the name right, which he does after some exercise, because at the beginning of the test he usually does not get the name right or gives the wrong one. Likewise, when a color is named for them to choose among several other colors, they do not immediately understand the denomination, and remain puzzled for a while without knowing what is it about.

Evocation of colored objects is also deficient. Subject M, for example, says from memory that violets (flowers) are green. It is typical in these two brain-injured patients and others we have studied, the indirect procedure to understand the name of a color. For example, when they are asked to separate the orange color from a pile of colors (M being under facilitation), they get perplexed, without being able to take a decision, showing that the indicated name does not mean anything to them. However, after a period of time, they decide to examine the colors and can finally extract the color in question. Interrogated about the cause of the first indecision and the correct behavior much later, it appears that initially the orange color does not make sense to them and is not conceived as corresponding to something with color, but later the name of the color has evoked the fruit of that name, which does offer a meaning of color to them, and this has been the starting point to reach the color in question. Therefore, they need to make a detour by means of the concrete representation of "orange fruit," since it is impossible for them to understand the color directly through "orange color." Due to these circumstances, they commonly use, in the denomination of colors, much more concrete names than the more conceptual or abstract ordinary ones; examples are blood color instead of red, sky color instead of blue, grass color instead of green, straw color instead of yellow, etc. They get oriented immediately with these concrete designations whereas the usual name generally seems to them vague or indeterminate, and some names, such as blue or yellow, seem particularly difficult.

The impairment of color naming comprehension in these brain-injured patients is much more pronounced for those colors to which they present blindness or weakness, i.e., yellow, blue and violet in subject M, and blue in subject T who tends to confuse it with green. Thus, there is an agnosia for colors with marked predominance for some of them, which corresponds to partial color agnosia, described by Peritz (1918) in a case of lesion in the angular gyrus. This shows that agnosia is more severe where sensory disturbance is also more pronounced, thereby revealing the unitary nature of the disorder.

In the M case, the effect of facilitation in this color agnosia is much less than in other functions, and although some improvement can be achieved, it is not as regular as in other functions. We have already observed the same in other tests on cognitive schema.

As we said, color agnosia is influenced by the alteration of the schema, i.e., by the alteration of the abstract behavior. Gelb and Goldstein (1924), in a very important study devoted to color name amnesia, base this disorder on a specific deficit of categories, that is, on a loss of the *category* function. This conception is nothing more than the psychological aspect of the schema disorder (disorder in the ability to construct ideas). In fact, the disorder is parallel or identical to the one that is revealed in the tests of Binet (1903), i.e., the whole framework fails, and behavior becomes more concrete, particular or partial, as expression of a deficit in the structural differentiation process.

For now, we shall limit ourselves to these brief indications about color agnosia in our patients. This disorder will be further studied when dealing with language disorders in relation to auditory functions.

15.3. STRUCTURE OF VISUAL AGNOSIA. SCHEMA DISSOLUTION

Theories on the structure of visual agnosia, and of all types of agnosia in general, could be classified into two main groups. In a first group we consider theories that, starting from the ideas and methods close to those of Wernicke (1895), are based on the theory of primary and secondary *identifications*, resulting in the two forms of visual agnosia of Lissauer (1890), apperceptive agnosia (inability to perceive forms correctly) and associative agnosia (inability to recognize forms), depending on the degree of difficulty. Various authors have elaborated somewhat different theories, but always according to these general types, and involving in different ways complex psychological mechanisms of associative and reproductive type. At the same time, they assume specific anatomical brain localizations, where the theory of specialized centers and association pathways is decisive. A second very different and more modern group is initiated with Monakow (1914 a, 1914 b), assuming, contrary to the earlier anatomic-psychological conception, a functional biological viewpoint in which the possibility of obtaining a specific anatomical localization for agnosia is rejected. The disorder is conceived, from a structural viewpoint, as a result of the disorganization of visual processes of very diverse complexity.

Both frames of reference (anatomic-psychological and functional) which assume at the same time very different brain conceptions, have contributed variously to the

advancement in the knowledge of visual agnosia. The former, more used with clinical objectives and as a starting point for the description of new agnosic syndromes, has lagged behind in deepening the mechanism of visual agnosia. Instead, the functional point of view of the second theory has undoubtedly facilitated such deepening. In this theory, the more modern conceptions of Poppelreuter (1923), Goldstein and Gelb (1918), etc., could be included, and also Stein (1928, 1930) in relation to physiological aspects of great significance.

The conception about agnosia of Goldstein and Gelb (1918) derives mainly from the study of the Schneider case. Regarding the structure of visual agnosia, they stress the defect of organization between ground and figure, which corresponds to the viewpoint of the Gestaltists about the perception mechanism of figures. It is also considered very characteristic of agnosia, and in general of all kinds of brain disorders in which higher functions are damaged, the loss of abstract behavior and the destruction of categorial function, so patients only understand tangible or concrete situations. As for the physical basis of agnosia, it is thought to be a general brain disorder, but it is not sufficiently precise. However, the Schneider case has been considered by these authors as a very characteristic focal defect. There is not then a sufficiently delimited conception concerning the anatomical issue. As a whole, their idea on agnosia, and also on the brain in general, pays deep attention to the altered behavior of patients, although at times, like in Schneider, they introduce very questionable psychological hypotheses. On the other hand, they build a theory of the brain based on biological characteristics that emphasizes both its functional character and its unity. However, despite all this, a physiological basis is still lacking, which gives a diffuse character to the theory of these authors. Apart from that, it can be said that such authors are the ones who have made the most progress in brain research within the more or less usual currents of thought.

The criterion of Poppelreuter (1923) is also of interest. He considers visual agnosia as a set of perceptual and intellectual defects of a very diverse and complex nature, which can hardly be explained by anatomo-clinical conceptions and associative theories. Through an appropriate study of experimental psychology in numerous patients with brain lesions, this author has brought to light the defects of the higher visual functions which, in a wide variety of forms, contribute to agnosia. This author is the one who revealed the slowness of perception in this type of patients, but he still explains it as a result of a higher psychic disorder. If we try to understand the meaning of the research of Poppelreuter (1923) as a whole, we find mainly the following features: psychological character although based on experimentation, extension of agnosic disorder to various activities (notation, attention, representation, etc.) and lack of a concrete physiological basis. According to this author, visual agnosia is not a perfectly delimited or theoretically founded set of clinical symptoms, but rather, the extraordinary complexity of the higher process of visual recognition results in a multitude of isolated disorders in the differentiation process. As for the delimitation that he proposes, he believes that visual agnosia must be considered the summarized expression of all kinds of visual disorders that are not visual field alterations and sensory defects. This delimitation is criticized and rejected by Stein (1928, 1930) who, enlarging even more the agnosia concept proposed by Poppelreuter (1923), relates

agnosia to basic alterations in brain excitability, thus involving all kinds of alterations in the visual system.

Therefore, in the work of Stein (1928), we find a physiological explanation for the first time, and the various psychological behavioral alterations would be derived from a brain excitability disorder. Thus, he states that a series of phenomena that until now only seemed susceptible of a psychological explanation, such as events referring to attention deficits and others, must find a physiological explanation in alterations of brain nervous excitability (Stein 1928). Consequently, he rejects, rightly in our opinion, that the slowness of perception in patients with agnosia is due to disorders of higher psychic functions as proposed by Poppelreuter (1923), and considers that it depends directly on simple alterations in brain excitability, i.e., on an increase in excitation time (visual chronaxie). Thus, sensory excitability disorder causes a destruction of sensory organization in a different way depending on the degree of the alteration.

One might wonder what then remains of the individuality of agnosia, as assumed in the theory of anatomical centers and pathological associations, and we must answer that nothing remains, since everything is reduced to a certain degree of sensory disorganization dependent on physiological conditions of brain excitability. The important physiological approach of Stein (1928, 1930) is largely confirmed in our studies, and the two cases exposed here fully agree with it. We must then consider Stein as a precursor of this kind of research. It should be emphasized that the ideas of Stein, with exception of his motor theory of perception based largely on the incomplete and partly erroneous studies on the Schneider case of Goldstein and Gelb (1918), are fully in line with reality and considerably simplify the problem of the sensory organization.

Even leaving the physiological basis of agnosia aside, attentive examination of the patients reveals that it is by no means an alteration circumscribed to the intellectual functions of recognition or higher understanding. On the contrary, careful, quantitative determinations always show there are more elementary sensory alterations, such as in visual acuity, visual perception of motion, stability of the visual field, etc., in addition to slowness and sensitivity deficits in all types of functions, all of which is only revealed by means of appropriate examinations. Such general alteration has been pointed out by the more modern previously mentioned authors, even though it was already exposed in older studies (Monakow 1905, Siemerling 1890, etc.). Hence, the idea expressed by Lissauer (1890) that a visual agnosia disorder only occurs when any other sensory and perceptual alteration can be ruled out, should be considered inadmissible because it must necessarily be impossible. This last point of view is already exposed in posthumous publications of Gelb (1925/1926), when addressing the issue of the difficulty in separating seeing from recognizing, since each of these processes is already sufficiently complex, and in no way can be explained by simple formulas of classical psychology.

Regarding our two cases, it is clear that a general disorder of their visual functions is very evident from what has been explained in previous chapters. In addition, we must not forget that the central lesion disturbs the sensory brain in its two halves equally, causing in other sensory systems similar alterations to those studied in the

visual system. Agnosia appears as a consequence of the deficit in the sensory organization of visual functions; this deficit is in turn based on the nervous excitability disorder of the cortex. Since functions are reduced according to their degree of physiological demand, shape or figure structures, requiring a great complexity of organization, are seriously altered, and even more so the cognitive visual schema that is directly derived from them and constitutes a more complex and differentiated aspect.

In the same way that the ability to discriminate between two simultaneous tactile stimuli is more easily lost than if they were successive stimuli (due to a reduction in functional capacity), the perception of forms and their recognition are altered. In other words, the more complex the functions, the more easily they are lost. Therefore, they all are disorders of the same nature and depend on the degree of the functional level of visual activity, and there is no need to resort to secondary or associative influences of other sensory systems, since the different visual functions are based solely on the degree of differentiation or organization of their *own* activity. Agnosia involves both an altered perception of forms (causing a diffuse and metamorphopsic vision) and an inability to construct a schema. The latter is broken down into more specific or accessory activities with no order other than juxtaposition, lacking a general framework or ideation. This does not mean that a visual chaos be established, but rather a change in vision that prevents or hinders the comprehension of reality, as we have had the opportunity to verify in the various tests. As in other functions, disaggregation of the function 'schema' is characteristic of agnosia, leading to a degradation in the organization.

Summarizing what was exposed in the different tests to our injured patients, the alterations in visual schema can be grouped as shown in Table 15.1.

Table 15.1. Alterations in visual schema.

Dissolution of visual schema: visual agnosia	
Changes in vision	**Constructional deficit**
Illusions by: totalization, equalization, etc.	Incomplete apperception Disaggregated apperception Failure of figurative schema
Metamorphopsia	Simultaneous agnosia (Binet's tests) Constructional apraxia (build figures, copy, etc.)
(In general, new dynamic equilibrium in figures)	Color agnosia (In general, concrete behavior replaces abstract or schematic behavior)
Changes in vision and *constructional deficit* influence each other No clear separation can be established between the effects of both factors	

All these alterations represent a reduction in the *degree of organization*. This reduction is just a dynamic reduction caused by the nervous asynchrony that we are

already familiar with. Concerning such organization, it should be emphasized that there is only a differentiation or progressive discrimination that depends entirely on the dynamic state afforded by the sensory visual field, which in turn is an expression of the level of brain excitability. Such visual organization is developed by itself, that is, without needing support from other sensory systems, contrary to what is generally supposed in the so-called associative agnosias in which a disorder in the association with other senses is assumed (Wernicke 1895, Lissauer 1890, etc.). Depending on the visual structural level, motion is perceived or not, acuity increases or decreases, visual schema is more diffuse or more coherent, but in all these cases it is not necessary to resort to the secondary help of touch or hearing to explain the disorder.

This approach to visual agnosia as a failure in the structure of the schema (schema dissolution) applies equally to tactile and auditory agnosias, as we shall see later on. Much more complete considerations on the general problem of sensory organization will be developed in the third part of this work.

16. Schema in spatial orientation

16.1. ALTERATION IN ORTHOGONAL[1] ORIENTATION

We shall now analyze the signification of the various objects in the external world in relation to their position and orientation in space. In this case, a spatial orientation schema is developed which imprints a specific character on perception, since perception then presents new and important properties. Restricting ourselves to the essentials, we shall expose the most striking characteristics of the alteration of the spatial orientation schema. These characteristics can be reduced to two types: loss of the orthogonal orientation and alteration of the allocentric spatial orientation. In both cases, the general nature of the disorder is of the same type as that already studied in the visual schema for shapes and figures; that is, changes in vision, constructional deficit, etc.

16.1.1. Orthogonal property of figures

Spatial orientation of figures and objects is a very important attribute in their aspect and recognition, to such an extent that it may be decisive. This property appears in many different degrees. An extreme case is that of figures and objects whose appearance depends almost essentially on their spatial orientation, for example, writing characters (letters and numbers). A significant alteration of their usual orientation makes them difficult to recognize, at least initially. These figures then have a strongly developed *orthogonal property*, i.e., the meaning of a figure is highly linked to its usual vertical upward orientation.[2] These kinds of figures can be denoted as figures of "orthogonal" signification. Many objects also acquire that property in varying degrees (e.g., furniture), whereas for other figures and objects that continually adopt varied orientation in ordinary life (e.g., tools, cutlery, etc.), their "orthogonal" signi-

[1] Orthogonal orientation is understood as orientation on the plane orthogonal to the line of sight.
[2] This is a particular orientation in the plane orthogonal to the line of sight.

fication can be nil. Accordingly, figures and objects can be classified, in a simplified manner, into "orthogonal" and indifferent.

In the M case, in the inactive state and with good illumination, an indifferent object such as the arrow used previously for the orientation test, is seen in the same position as a normal subject. However, another object with orthogonal property, such as a writing, which a normal subject is not able to read it or does it very slowly when it is rotated 180° in the frontal plane, is read by subject M with the same ease whether it is presented in normal position or rotated 180°. In addition, he believes and repeatedly asserts that it is in a normal position. Therefore, the orthogonal property has been lost in our case M, and for this reason, figures are recognized independently of their spatial orientation, without offering any change in their aspect. If he believes that the writing is in normal position despite being actually upside down, it is precisely because of this lack of change in its aspect. Such a change is characteristic for a normal subject, who is not used to reading an inverted writing. However, this result does not merely imply that all figures behave as indifferent for the M case, since those that are really indifferent, like the test arrow, can be perceived in many different positions and admitted that it is in different positions, provided there is enough illumination to avoid the asynchrony studied. By contrary, writing is always admitted in normal position no matter its real orientation.

We face a complex disorder because, on the one hand, the anomalous orientation does not hinder the perception of the characteristic aspect of the object, contrary to what happens in the normal subject, but on the other hand, since the change in the position is not registered, all orientations are considered equally correct. There is a loss of "orthogonal" signification and, in addition, lability and diffuse orientation perception.

Initially, in 1938, this type of disorder hampered the study of visual image orientation disorder or inverted vision, in the same way as chromatopsia hampered the analysis of color disturbances until chromatopsia was sufficiently clarified. In the previous experiments on visual image orientation, we intentionally used a very simple arrow as a test object, which in addition to the ease of perceiving its shape, allow us to exclude effects such as those provided by objects with "orthogonal" signification.

Although it is not possible to graphically represent this type of phenomena by means of curves due to the nature of the issue, the phenomena are perfectly in line with the dynamism of stimulation conditions and changes in excitability through facilitation. The results of the tests performed on our patients can be considered among the most remarkable. For instance, in visual perception of numbers or letters by subject M in inactive state, in good illumination for the shape to be correctly perceived, he recognizes them easily in any orientation, and in addition he believes that they are in normal position. However, as soon as facilitation by muscular effort acts, the illusion disappears right away and their orientations are perceived correctly. It is a sudden change that allows correct perception, but no data could be obtained from the patient about the transformation process. There are also different types of vision as in other visual functions, as well as intermediate states between correct perception and absolute loss of the "orthogonal" signification. Thus, we are dealing

with phenomena susceptible of being studied in accordance with the same general characteristics (excitability, gradations, etc.) as the other visual functions already discussed.

We have observed the orthogonal disorder quite clearly in our two patientss; in 1938 in M, and in the beginning of 1939 in T, but at present, only subject M has it, generally in the inactive state, although also under facilitation by muscular effort when illumination is weak. There is then a difference in behavior in both patients similar to that found in chromatopsia; very pronounced and completely steady in M, and only traces and very fleeting in T, according to the different functional level of both subjects, which implies a different degree of asynchrony or disaggregation of the sensory structure. Nevertheless, although the T case, due to the greater recovery from his brain disorders, does not present this peculiar alteration in recent years, there is a marked tendency to it in transient states of greater brain deficit (e.g., after epileptic seizures). In these states, it seems necessary to admit a somewhat pronounced degree of asynchrony, at least similar to that of M under facilitation and in monocular vision. It is very likely that the Schneider case of Goldstein and Gelb (1918) presents this alteration as well.

We are not aware that this singular disorder, or at least an alteration as pronounced as that of our two brain-injured patients, has ever been described. However, it is conceivable that many described cases of constructional apraxia, optic ataxia, etc., may be related to this remarkable disorder and even find their main origin in it. We have only found a mention that refers to the Ziegel case of Kleist (1934, p 579), a war-wounded patient with a biparietal bullet shot (entry and exit). In that reference, two lines of the medical history of the patient indicate that he recognizes figures or pictures presented upside down with somewhat more difficulty than in the correct position, but he does not know if they are in correct position, upside down or toward what side they are oriented. This case and others that also present diverse disturbances of the optical apraxia type are considered by Kleist (1934) as cases of location blindness ('Ortblindheit' in German).

16.1.2. Loss of orthogonal orientation

We shall expose here the most outstanding manifestations of this complex disorder, which lends itself to a wide variety of studies. It was largely studied in subject M in the summer of 1938; his disorder being more or less in the same state at present.

When diverse figures, especially portraits of people, pictures, etc., are presented to subject M in the inactive state, he recognizes them in the correct or upside down position indistinctly, and he does not perceive the slightest difference, believing that in both cases they are in a normal or correct position. Asking him to place those objects in the most favorable position for him, after having turned them over and over, he ends up often placing them upside down. Instead, he easily recognizes the horizontal position as anomalous, although this position neither is very problematic for him. When he leafs through an illustrated newspaper in the correct position and he finds a difficult figure for him, he spontaneously inverts the newspaper. In his pocket watch, although he handles it in correct position, he reads the time in any

position without noticing any change. In general, the correct position and upside down position are indifferent for him, and he only recognizes the horizontal position as anomalous. These tests are carried out in binocular vision and under as good illumination as desired, he being in the inactive state. If facilitation by muscular effort is activated, he immediately realizes the real position of the drawings or pictures, and his behavior then does not differ from that of a normal subject.

Therefore, without facilitation, abnormal orientation of figures does not hamper their recognition, and the patient tends to admit they are in normal position in almost all situations. Two big mutually inverted portraits, one in normal position, the other one upside down, are both recognized with the same ease, and he considers them equally in correct position. It is evident that the orientation of figures does not prevent their recognition; however, it is not so evident that any orientation be perceived as normal. Is there really an inner re-inversion in the imagination and hence do figures tend to be seen in their usual position? Or is it just that the patient expresses his perceptions in a diffuse way? Or do both things happen in part? Experimental proofs in favor of either opinion are not easy to obtain, given the lability of the process. Thus, when in the case of inverted portraits that the subject affirms to be in correct position, he is asked to point to the forehead or hair, he indicates with his finger rather automatically toward the upper part (the forehead or hair being in reality on the lower part); however, pressing him to indicate more definitely the details of the asked object, he appears insecure and finally changes direction towards the lower part. Therefore, it is doubtful that a re-inversion be established. It seems more likely that the recognition of the figure per se is so independent of its position that its orientation is left practically excluded, and the figure, being perceived as unaltered, is admitted to be identical both in the correct position and upside down, an erroneous assessment that is secondarily and unconsciously added to the perception. The recognition of the figure is so independent of its orientation that recognition is the dominant sensation for the patient; and if there is no alteration of the appearance by modification of the orientation, there is no reason to believe that the figure is in an abnormal position. This is the simplest explanation which is also in agreement with the nature of the general disorder in our patient.

In general, one should not rely on the explanations or viewpoints of brain-injured patients, but only taken them as data to be verified. Admitting that the patient truly sees the inverted figure in the correct position, we would have to assume a re-inversion of the image in the imagination, which would correspond to a structural process similar to the one the Gestaltists call law of good form. That is, ultimately, the figures would really be perceived in the right position because it is the most appropriate one, and this easy re-inversion would be favored by instability in the perception and lack of firmness in their position and orientation. However, this would entail a strong activity of the orthogonal property described above, which fails precisely in this patient. On the other hand, the instability in the perception of orientation could also be considered to be the cause for excluding the influence of orientation on the figure. In short, it must be admitted that the deficit in the organization of orientation is the cause of its lack of influence on the aspect of figures, contrary

to what happens in the normal subject. For the patient, the aspect of the figure is always the same, and is right in any position.

However, one might then ask why the behavior with respect to objects or figures that are indifferent from the point of view of orientation, is so different. This is probably because they are not subjected to a change of aspect according their orientation and, above all, because they are very simple objects or figures whose positions or orientations are much easier to determine. Thus, in the case of an arrow, it is not difficult to know whether it is in a vertical position or tilted since it is only just about the position of a straight line, and the same would apply in the case of a spoon, for example. Whereas for the normal subject a difference can be established between forms with "orthogonal" and indifferent signification, for case M, all forms behave as indifferent. Nevertheless, he can recognize the orientation in the indifferent ones; in the others, the orientation seems the same to him. This is due to the fact that in the most complicated figures (portraits, pictures, letters, numbers, etc.) the interest of the figure per se predominates over anything else and the perception of form is realized independently of the spatial background, contrary to very simple figures in which the figure-background relationship is easier to establish. Therefore, it really has to do with the diverse complexity to organize forms in the space or background that contains them. In all cases there is lability or difficulty of orientation, but in the case of extremely simple figures, their orientation can still be recognized, whereas in the case of more complex figures, there is a greater requirement for perception since, in addition to understanding them, they must be oriented with respect to the background, and the latter does not take place (lost orthogonal property), so all orientations seem approximately identical. For this reason, it is very difficult to admit that a portrait is upside down since it offers the same aspect as when it is in normal position.

This type of orientation fault may contribute to hide in part the visual image orientation disorder studied in precedent chapters; e.g., objects that should be perceived as tilted due to poor illumination and small size may not be perceived as tilted. Thus, subject M being in the theater realized that the actors were upside down or at least that they were highly tilted. This happened to him immediately after the beginning of the performance. However, the inverted scene did not feel totally strange to him, and there were moments where he failed to notice the inversion.

He has very remarkable confusions about the position of objects of daily use, for example, chairs. In inactive state he makes mistakes constantly, and no matter whether chairs are in correct position or upside down, they always seem to him that they are in a good position. Invited to sit in a chair upside down rested on the wall (whit his hands in his pockets to avoid being oriented by touch), he tries to sit resolutely without any qualms, and is very surprised when he slips. He believes that the chair might be broken, and then he does not agree to sit on another one in normal position under the excuse that it is a hoax. These mistakes disappear as soon as facilitation by muscular effort acts. But also in the inactive state, when the observation lasts a long time and he is asked to look hard, he ends up by showing doubts about the true position of a chair put upside down. He finds certain abnormalities, for example, that sticks come out too much or that there is a crossbar that does not correspond; he just gets to say that the chair is a little crooked. Similar mistakes arise when

he tries to know if a table is in normal position or upside down. A table upside down with a chair next to it in normal position seem to him in a good position to sit down to eat. It seems that the set surprises him, but he is not able to understand the situation; then, after a long time, he thinks the chair is fine and the table legs a bit crooked. If he comes very close to objects, he ends up recognizing their position correctly. In general, he confuses very easily the positions up and down in all these tests; instead, he perceives rather well a chair lying on its side.

The confusion about spatial orientation occurs more commonly in drawings and pictures, and less in tangible objects. Thus, whereas a portrait is perceived equally in any orientation, especially vertical up and vertical down, there is less confusion with tangible objects of moderate size, and generally the subject knows their true position. A particularly difficult case is to recognize an orientation among others in a set of variously oriented objects or drawings; it usually occurs that all of them rank equally as regards orientation. Moreover, whereas he is able, after paying close attention, to recognize the orientation of a single object isolated from the set, if the object is in the set, he may equate its orientation with that of any other object in a very different position. Differentiation for any class of function is a complex activity, hence its failure is easy. Therefore, it is understandable the great difficulty when trying to assemble a puzzle or to build a drawing that requires many lines.

All these kinds of disorders are presented to the maximum in writing characters (letters and numbers), where they can be easily studied.

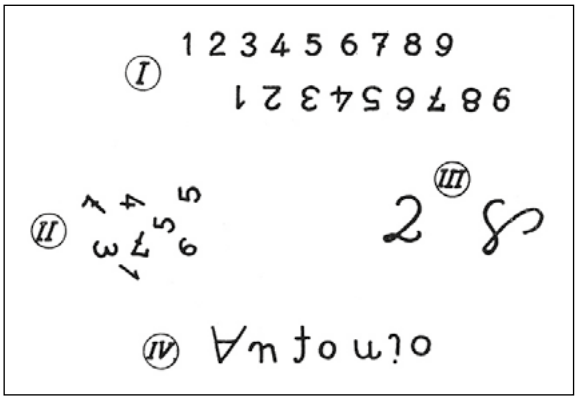

Figure 16.1. Numbers and letters to test the loss of orthogonal orientation in subject M, in the inactive state. I) The two rows of numbers are recognized with the same ease, and he claims they are in the same position. II) He names correctly all the numbers claiming they are all equally in normal position and in two rows. III) He has no difficulty in recognizing two numbers '2' with very different aspects, whereas a normal subject can easily take the altered '2' for an '8.' IV) He reads his name perfectly without noticing any anomaly. By means of facilitation by muscular effort, he becomes aware of the inversions and other orientations.

Figure 16.1 shows some of these tests. Subject M recognizes and names the series of natural numbers in any orientation, both in normal and upside down posi-

tion, without noticing any difference and, of course, he thinks they are fine in both cases (Fig. 16.1 I). Numbers variously oriented and placed disorderly, are all of them read by him with the same ease, and he believes they are all equally oriented and in two rows (Fig. 16.1 II). A very demonstrative case of the loss of influence of spatial orientation on the aspect of the figure is the test we carried out with the number '2.' This number drawn in a very anomalous position, as indicated in Fig. 16.1 III, is not recognized by a normal subject or he thinks it might be an '8' in any case. However, our patient takes it for a '2' with the same ease as when it is placed correctly. Besides, he believes it is in normal position, as the '2' written correctly. In this example, whereas the normal subject tends to see an '8' due to its orthogonal property which appears then in favor of that number, subject M keeps on seeing a '2' because the "orthogonal" signification does not intervene, and there is no confusion with an '8.' Under facilitation by muscular effort, the different position is well distinguished; the normal '2' is more easily recognized whereas the altered '2' appears strange at the beginning and, at times, it is taken for an '8,' as it happens to a healthy subject.

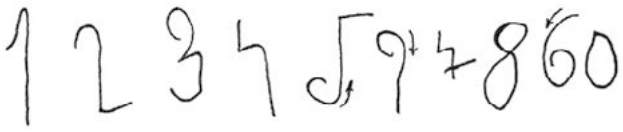

Figure 16.2. Spontaneous writing by patient M of a series of numbers. He reverses the '6' and the '9,' which he confounds with each other. In addition, perseveration of a number in another one is observed (he begins the '3' as a '2,' the '5' as a '6,' the '7' as a '4'). The difficulty for the numbers '6' and '9' is quite persistent.

Also noteworthy is the way in which subject M writes the series of natural numbers (Fig. 16.2), the maximum difficulty being in numbers '6' and '9,' which he draws almost always mutually changed. In addition, some perseveration of the shape is observed when passing from one number to another number; the number '2' reminds the number '1,' the '3' is written as a '2' initially, the '5' is written imperfectly and beginning from the bottom; the '6' is made like a '9'; the '7' reminds the '4' and, finally, the number '9' has been mistaken for '6.' This last mistake occurs constantly. When he is asked to write '96,' he writes '69' most frequently, and when is asked separately to write '6' and '9,' very rarely he can do it correctly and writes them inverted and also reflected. Furthermore, anyone of these two numbers is taken for a '6' or for a '9' indistinctly, the same number thus offering two very different meanings. This situation is similar to that of the normal subject with regard to ambiguous images ('Vexierbild' in German) and drawings with interpretation either in hollow or in relief. By means of facilitation by muscular effort, he easily realizes the confusion between '6' and '9,' and then writes them correctly.

When he is asked to copy a number that is inverted and that he admits as being in correct position, the copy may be a true copy, i.e., inverted, although he still maintains it is in the normal position. If he is then asked to write the same number by

heart, without looking at the inverted model, he does not draw it completely normal, the result being intermediate between the previous inverted copy and its normal position. These remarkable peculiarities can be observed in the various stages displayed in Fig. 16.3. He immediately recognizes the '2' of the very abnormally oriented model, and when he is asked to copy it, he writes a '2' in normal position, which he does automatically, without worrying about the original model; he finds them both identical. Urging him to copy the model in a second attempt, he begins the copy faithfully, but ends up writing the '2' automatically in normal position. A third time he tries the copy and does it rather well, and a fourth time almost completely well. If he is now asked to write the number without looking at the model, he does it but in a reflected way. In every case, the numbers seem to him to be in the same position even though the drawing has been different.

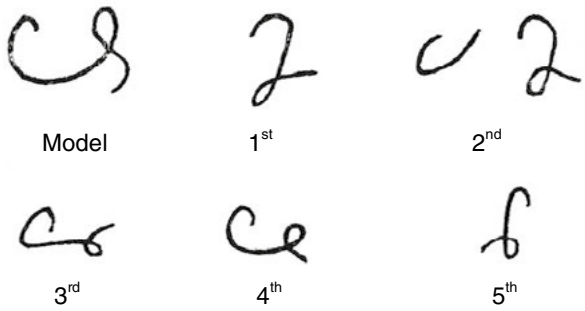

Figure 16.3. M case. Several stages in the copy of the abnormally oriented '2' which subject M considers in correct position.1st attempt: he writes automatically a normal '2.' 2nd attempt: asked to concentrate on the model, he tries to copy it but he starts again and writes again a normal '2.' 3rd attempt: the copy resembles more the model. 4th attempt: finally, the copy is almost equal. 5th attempt: if told to write the number without paying attention to the model, he draws a mirrored '2.'

These tests show the antagonism between automatic writing and model imitation, as well as the lack of influence of writing movements on the perception of the orientation of the writing. The exact copy can be reached after many attempts and very slowly, but the change of movements is not sufficient to establish a different understanding despite the modifications in the motor attitude during the attempts, as is shown by writing finally the number without looking at the model. The lability of spatial orientation perception is therefore extensive to writing movements, this being also the reason for the mutual confusion between '6' and '9' when drawing them.

Concerning the particular conditions of stimulation and functional level which determine the failure in the spatial orientation of figures, we must note that there are several types of vision similar to those already discussed in other visual functions. Generally, in good illumination and appropriate size of figures, subject M in binocular vision and inactive state recognizes what it is all about, but fails in orientation which always accepts as normal. However, under facilitation by muscular effort, the interpretation is perfect. However, even binocularly and with muscular effort, when the illumination or

the size of the figures is considerably reduced, the same failure as in the inactive state appears. Here too, especial visual facilitations are possible. Thus, subject M in binocular vision, with muscular effort, good illumination and sufficiently near vision, recognizes the different meanings of the numbers and letters shown in Fig. 16.1, but does not perceive the orientation defects, or only succeeds with some number. However, as soon as intense light is directed toward his eyes, without changing the illumination of the figure, he perceives correctly the different orientation of the numbers and letters. This is then the result of binocularity, muscular effort, light over the eyes and good illumination of the figures. Such combination of facilitations may be needed in the case of small figures, or in tests of differentiation which are always most difficult. It can also happen that with muscular effort but looking at the figure with only one eye, the patient does not know its orientation, and by intense illumination over the other eye, which although open does not look at the figure, a normal visual function is obtained. Thus, a series of combinations can be carried out. In short, the functional level depends on both facilitation and intensity of the stimulus (size and illumination of the figure).

This series of tests allows us to establish different stages in the development of the normal function. There are mainly the following stages, from the simplest to the most complex: 1st, all orientations are indifferent; 2nd, only opposite orientations in the vertical, upward and downward, are confused with each other; 3rd, orientation is correctly perceived. All these stages depend on the stimulation conditions and the excitability level by the action of different types of facilitation. At all stages, the interpretation of the figure can be as good as desired, perceiving details in a portrait about sex, age, etc., but this does not prevent the failure in recognizing its orientation.

As for the subject T, in his usual state at present, he does not show any disorder of this type, not even drastically reducing the illumination of the figures; or in any case, the disorder is unimportant and is quickly corrected. Nevertheless, we had the opportunity to observe a very evident alteration in his perception of orientation in early 1939, when trying to verify whether he presented inverted vision, previously discovered in patient M. Although he did not present inverted vision but only a significant tilt whose magnitude was not accurately determined at that time, it was quite clear the loss of orthogonal orientation, since he could read the newspaper fluently and with the same ease in the normal position and upside down, although in both cases somewhat slowly because of the cerebral deficit. In both positions he found the newspaper to be in the normal position, without noticing any change. Later, we learned that this patient, shortly after having suffered the brain lesion and long before being referred to us for observation, was often seen reading the newspaper upside down, without realizing it, and it seems that he became annoyed when, for this reason, he suffered the mocking of his fellows, mocking he could not understand at all since the change of position of the newspaper was indifferent to him. This singular disorder must have disappeared in this patient T around 1940, and when he was re-examined in 1942, there were no signs of it. In the tests on ethyl action described previously, this alteration was also not found, or in any case, to a negligible degree. However, when he was observed in 1944 by chance, two days after a severe epileptic seizure which had reduced considerably his brain activity, he did show certain alterations in visual orientation close to being pathological. For example, he was able to read in-

verted writing without realizing it, and he used to believe that correctly oriented writing was badly oriented, but finally he was able to realize the real spatial orientations despite he easily confused some of them. Regarding his functional ability, he appeared to be in the intermediate state indicated above in which mutually opposed orientations (normal and upside down) are easily confused with each other.

16.1.3. Structure of orthogonal orientation. Spatial level

Regarding the structure of the 'figure orientation' function, it is important to point out certain ontogenic and phylogenic peculiarities, i.e., its development in children and primitive humans, who show a behavior very similar to that of our two patients M and T.

Preyer (1882, 1890) discovered his 5-year-old son drawing most of the numbers either reflected or inverted. This behavior lasted a long time with the numbers 1 and 4. Bühler (1918) relates in his work that it is a very curious fact, which has caught the attention of many people, that many children look at their books of drawings with the same interest whether the books are in a normal position or upside down; and when this fact is investigated it is found that they distinguish and name representations of men, women, animals, trees, houses, ships, etc, with the same ease in the normal position and also rotated at an angle of 90° to 180° from the natural position. This is consistent with the fact that when they start drawing, they often confuse the orientation in space, representing things upside down; for example, they draw men and animals upside down, or a car with the wheels upward, and this is also shown in their first attempts at writing, where another type of disorientation is even more frequent, which is the reproduction of writing as seen in a mirror, changing the right side for left side.

All this constitutes a complex set of facts from which it follows that the concept of spatial form can be represented, up to a certain point, independently of orientation, and children often ignore orientation even when they already know how to draw forms. A more detailed theoretical study of this issue could significantly contribute to the general problem of perception of figures. Also for Stern (1909), form is for a child much more independent of its absolute position than for adults. Children look at drawings and pictures in any position, they copy the letters from a model in all possible orientations without any difficulty, and it is not an effort for them to read a text reflected in a mirror, which they do as well as those who are trained to do it.

A finding of Oetjen (1915) is very interesting in this regard; he observed that a rotation of 90° of reading material represented a much less difficulty for children aged nine to thirteen and a half than for adults. The influence of writing orientation on the time spent on writing recognition by adults is shown in the research of Prandtl (1927), according to which the positions that offer most difficulty are the two inverted oblique positions (↙ ↘), ad the next in difficulty is the upside down (↓), whereas the slightly tilted positions (↖ ↗) allow reading the text in an almost normal time. Such differences are not present in children, neither in primitive people, since in both, recognition is independent of orientation, as is also the case in our two patients. Concerning primitive people, there is an observation of Peschüel (1877) on the in-

digenous population of Loango (Bantu Negroes) in whom he verified that they saw and understood images representing familiar things equally well in both normal and upside down orientations. Likewise, the few who knew how to read a printed text, they did it fluently both with the text in normal position and upside down.

Therefore, the disorder of our two patients presents a remarkable parallelism with the behavior of children and primitive people, since in all these cases the meaning of a figure is independent of its spatial orientation. In these cases, there is a *spatial level* (according to the Gestaltists) scarcely differentiated because the corresponding relationship between the figure and the spatial background that contains it is not established; thus, there is no tendency to establish the *constancy of the figure* corresponding to its normal orientation.

In subjects M y T, the destruction of the brain mechanism of visual orientation must also be taken into account, which already implies an extremely severe alteration of spatial organization. We know that even when a normal visual image orientation is obtained by appropriate stimulation, there is certain instability revealed by the influence of secondary factors (as in the slot experiment and others). This meansthat even in very simple shapes, we cannot count on a totally firm orientation, although in general, for elementary shapes (e.g., stripes) the orientation is generally well perceived. However, in the case of more complex figures, there would be a *functional overload* that would prevent the development of the entire structural plan, the figure being perceived as segregated from its spatial orientation attributes. In this case, any type of orientation does not change at all the aspect of the figure, which conventionally can take on different meanings (case of '6' and '9').

Therefore, it can also be said that there is a dissolution of the spatial orientation schema because, as was seen when studying the schema with respect to forms, there is both an altered or unstable vision and a constructional deficit. The former because the abnormal orientation does not modify the aspect of the figure, hence visual perception is altered compared to a normal subject; the latter because all orientations are perceived as similar due to a failure in the spatial schema, that is, in the construction of references. There is thus a mutual influence between the two factors, and it is impossible to establish a clear separation between them. In short, the *spatial level* (figure-background) is not established, and everything is the same as regards orientation. The sensory field is too deficient to present then the differentiated function 'schema,' which depends entirely, like other functions, on the type of stimulation and, above all, on the established physiological level. By means of facilitation, such level improves and at the same time increases the coherency of perception, i.e., a more differentiated sensory organization is produced.

16.2. ALTERATION OF THE ALLOCENTRIC SPATIAL ORIENTATION

Finally, we shall briefly discuss a spatial orientation disorder with general features very similar to those of the disorder described above. It lies basically in the fact that the spatial references are not established in an absolute and constant way according to the space coordinates, i.e., according to an *allocentric orientation*, but rather an *ego-*

centric orientation is generated in which the position of the subject's body decides on the orientation at every moment. This disorder is very pronounced in subject M in inactive state, and is notably corrected by facilitation in the same way as in the loss of orthogonal orientation of figures. Subject M is one of the few cases in brain pathology in which *visual agnosia of spatial orientation* has been observed. A certain similarity is found with the patient studied by Siekmann (1932).

The difficulty for allocentric orientation derives both from instability of visual image orientation and from the intrinsic alteration of spatial schema by constructional deficit. Concerning the former, we have already indicated in previous pages that a tilt of the head causes the upright test arrow to be observed tilted even when the luminous stimulation is intense enough to exclude asynchrony in visual image orientation. In the normal subject, such a tilt only occurs in the case of a luminous line observed in the dark, which constitutes the so-called *Aubert phenomenon* (Aubert 1865). According to Müller (1917), the following three systems cooperate in the mechanism of spatial localization:1, the gaze system (with the three coordinate axes of the cyclops-like eye); 2, the head system and 3, the system originated by the trunk position. Although the three systems generally coincide, they may be different in special situations, for example in the *Aubert phenomenon.* In the wounded patients studied here, this mechanism is so labile and unstable that the dissociation occurs at all times. The above-mentioned systems neither inform nor correct each other, and the compensations that take place in the normal subject fail. In this way, spatial orientation depends on the orientation of the body of the subject, remains adhered to it, and the egocentric reference controls the situation.

This new type of orientation is evidenced in several tests. For example, when subject M moves while sitting on a swivel chair, what seems to him to be turning or moving is not his own body but the visual scene, thus appearing a "movement induced" by a change in the spatial references. This change is due to the fact that the turn of the own body is not felt because of a deficit in the nervous centers, and what is felt then in movement is the outside, i.e., the visual scene. The coordination or mutual adaptation of the different sensations involved in orientation can be so weak that even when walking, the subject has the sensation that the ground is moving. All these anomalies are excluded by means of facilitation by muscular effort, or at least they are so minimal that they can be disregarded, appearing only in special tests. The information provided by the haptic domain is very weak and diffuse because the tactile mechanisms (pressures, joint movements, etc.) are as altered as the visual function, and only by means of facilitation is it possible to have a more efficient haptic activity.

Therefore, the orientation is not fixed as it is in the normal subject, but is determined in a circumstantial way. This can also be shown in the following experiment. Subject M with his eyes closed is fixed to an operating table that can be rotated, and he is placed vertically with his head down. In this position he is asked to look at a vertical arrow pointing upwards and to indicate its direction (the vision being very clear and distinct); he then responds that the arrow is vertical pointing downwards. That is, he does not localize the object in relation to the absolute space, which is normally independent of the body position, but he localizes it egocentrical-

ly, i.e., in relation to his own body. When he is asked to make a strong muscular contraction, his perception changes and he answers: "No, the arrow is upward, and I am downward." Facilitation, by improving brain activity, modifies the spatial reference system and replaces the pathological egocentric orientation with the normal allocentric one. In this experiment, it is understood that the abnormal body position is not detected by the subject in the inactive state but is detected under facilitation by muscular effort, which causes the localization of the arrow to change. It is clear that here the change is not due to an asynchrony in the perception of visual image orientation (since the subject always sees the arrow very clearly and distinctly), but to a change in the reference.

These alterations in spatial orientation involve, as has been seen clearly, a severe tactile deficit which we shall deal with further on in the appropriate place. This deficit, together with the phenomenon of asynchrony in visual image orientation, can give rise to very singular disorientations of the own body, as occurs in subject M in the inactive state. For example, a finger is taken from the patient and moved away from him about half a meter so that he perceives it with blurred vision in low light. It then turns out that a passive movement of the finger from left to right is perceived in the opposite direction, and when the finger has reached the right side and the patient is asked to take it with the other hand, he tries to take it from the opposite side, i.e. on the left. Since the passive joint movement of his own arm is not perceived by him in the inactive state, he only detects the finger visually, and in order to take it he must necessarily be guided by the perceived movement of the finger which is in the reverse direction, giving rise to a severe disorientation of his own body. These experiments can be made more complicated in several ways, showing effects that constitute a true *experimental autotopagnosia*. We shall deal with this later, in the part devoted to tactile functions.

Apart from the alteration caused by the lack of correlation between the various factors involved in spatial orientation, subject M has difficulty in appreciating the relative orientation of several objects. If there is only a single object, the subject knows whether it is to the right or left of his body, but if he sees two or three objects at the same time and their relative positions change, he does not notice any difference, at least at first and when he is in an inactive state. This difficulty increases when dealing with drawings. Thus, he believes with respect to Fig. 16.1 (II) that all the numbers are simply in two rows, being unable to appreciate the diversity of their positions and orientations. He can also get very disoriented in special situations of ordinary life, for example on the top of a staircase. Thus, when he was at the top of the steps of an operating room, near the celling, he perceived the celling as the wall, confusing most directions of the room. Nevertheless, after many verifications he can get oriented, even without facilitation.

These alterations show analogous characteristics to other disorders previously indicated in spatial schema. These are, alteration in vision and constructional deficit; the former is originated as a consequence of the alteration of references, for example in the mentioned case of "induced movement"; the latter prevents absolute references and breaks up spatial schema, evidenced for example in the difficulties to appreciate the relative positions between several objects. All positions tend to be perceived

as being the same, as it occurs in the loss of orthogonal orientation of figures. In general, it can be said that allocentric orientation is replaced by egocentric one, and the subject does not move within a space of fixed orientation, but the orientational space changes according to circumstances and follows the own body of the subject.

The alteration of the spatial orientation schema in the two aspects studied, loss of orthogonal orientation and loss of allocentric orientation, reveals the complexity of the visual orientation disorder. Whereas orientation in the normal subject is firmly unified, here, in our patients, especially in case of M, it is split, due to nervous asynchrony, into a series of stages or independent partial functions with very different levels of excitability. These levels can be recruited either by increasing stimulation or by synchronization by means of facilitation. Considering the most pronounced changes in the structure of spatial orientation, we can distinguish the following stages: 1) inverted vision (with indistinct vision); 2) normal oriented visual image although labile (with distinct vision) but orthogonal and allocentric failure; 3) normal stage, only reached by means of facilitation from the second stage.

Dynamic reduction, by excluding the most complex functions, follows an inverse order to the one we have exposed in the three stages. It must be noted that functional disaggregation is conditioned by the degree of asynchrony; thus, subject M with distinct vision may show allocentric and orthogonal failure, whereas subject T with also distinct vision does not present such alterations due to a significantly lower asynchrony.

Recapitulation on visual functions

A brief recapitulation will serve to establish an overview of the multiple phenomena studied in visual functions. Many of the phenomena are new, and the way they are addressed is entirely new.

The alteration of the visual system is only a part of the sensory disorder in the central syndrome; disturbances of the same nature and intensity will be studied in other sensory systems. Sensory structures depend on the degree of nervous synchronization. A brain injury, causing an excitability deficit that affects functional elements differently, leads to an asynchrony between them. New functions then appear without any precedent in the normal subject (chromatopsia and dyschromatopsia, flat colors, inverted vision, metamorphopsia or change in shape perception, egocentric spatial orientation, reading upside down as easy as normal, etc.). However, an inextricable chaos does not occur, but a more reduced and simple organization where the general physiological laws remain valid, although at a different scale.

1) Regarding *general excitability*, there is a considerable increase in rheobase and chronaxie, in addition to the important anomaly of permeability to nervous summations such as facilitation and iteration. Luminous sensation presents long latency and also high persistence. Light adaptation is very slow and to a lesser degree than in the normal subject; there is therefore a certain hemeralopia of central origin. It is very important to note that visual sensation depends on the three following factors: stimulus, receptor and central state, the latter depending on the deficit created by the lesion and the compensation provided by facilitation.

2) In *color vision,* there is a general alteration, but with very great predominance in the yellow-blue pair which is perceived very altered. There is a great increase in the photochromic interval; and in yellow, blue and white, there is a special decomposition that gives rise to a photo-heterochromic interval. The significant asynchrony and different excitability of the different colors prevent the formation of white, and chromatopsia appears in its place. It should also be noted the inversion of color

isopters and the alteration of chromatic induction phenomena (increased edge contrast and abolition of negative afterimages). The color disorder corresponds to tritanopia (according to the nomenclature of the trichromatic theory). According to the physiological properties of colors, they can be classified into simple or primary (red and green) and composite (yellow, blue-violet, white), the last ones being differentiated from the first ones.

3) Regarding *visual forms*, we can mention phenomena such as concentric reduction of the visual field (prototype of dynamic reduction), irradiated localization which causes the pathological vision of flat colors and severely alters visual acuity, severe deficit in visual perception of motion, etc. Motion perception is broken down into different phases (static sensation, inverted motion, seeming acceleration, etc.). Perception of figures and objects is very diffuse and unstable, there is a tendency to metamorphopsia, which creates a more stable and simpler figure. Understanding or recognition of objects is indirect and insecure, as well as slow.

4) With regard to *visual image orientation,* there is the very singular phenomenon of inverted or variously tilted vision, as a result of asynchrony in the visual-haptic complex[1]. Distinct vision (due to intense stimulus) results in correct orientation, whereas blurry vision (due to weak stimulus) causes inverted vision. Perceived orientation as a function of stimulus intensity follows the law of Fechner (1860), which can also be applied to other visual asynchronies. In relation to the factors involved in sensation, important phenomena appear such as re-inversion by means of facilitation, inverted vision in peripheral vision and also due to dazzling, types of vision with different maximum image tilt, etc.

5) Concerning *visual schema* (understanding of simplified figures and also of complex visual structures), there is a schema dissolution, both by an alteration of vision resulting from an unstable perception, and by a constructional deficit that disintegrates structures; drawings are seen differently, and there is no abstract behavior capable of understanding sets of them. Due to the alteration of the spatial schema there is a loss of orthogonal orientation (which leads to the recognition of figures independently of their orientation such as reading a text upside down without being aware of it), as well as a loss of allocentric orientation, which is replaced by egocentric orientation, due to a change of spatial references.

The dynamic unity governing brain activity is what causes the lesion to alter sensory organization as a whole, although in different degrees the different visual functions due to their different asynchrony; the greater their physiological demand, the greater the alteration suffered. The pathological asynchrony can be avoided by intensifying the stimulus. This can be achieved more easily the lower the degree of asynchrony. In highly altered cases, such as M, no matter how much the intensity of the

[1] Visual inversion is more fully reviewed in Sec. 26.2 in Vol. 2, and related to the secondary areas at the end of Sec. 4 in: GONZALO, J. (1952). *Trabajos del Instituto Cajal de Investigaciones Biológicas,* XLIV: 95-157. (Supplement I of the present edition.)

Table R1. Visual functions of subject M in the inactive state.

1. General excitability	2. Colors	3. Forms	4. Image orientation	5. Schema
Rheobase and chronaxie 10 times greater than normal – 71% summation degree in iteration – Great permeability to facilitation, saving 1/3 of rheobase voltage – Long latency and persistence of sensation	In medium light: yellow-blue blindness (dyschromat.) – Chromatopsia – Inversion of color isopters. – Very increased edge contrast, etc.	Visual field up to 6° (OD), 4° (OS) – Acuity monoc.: 1/25 binoc.: 1/10 – Strong color irradiation – Loss of motion perception except with very intense stimulus – Unstable shape perception, successive understanding	OD max. tilt: 145°. In very strong light: about 5° for normal vision. – OS max. tilt:170°. Binocularly max. Tilt: 115° – Binocular image splitting – Change by: facilitation, dazzling, peripheral vision	Binocular and strong light: illusions, disaggregation and concrete behavior in the face of shapes and complex figures – Spatial orientation: loss of orthogonal and allocentric functions (only egocentric orientation).

stimulus is increased, in the inactive state there are always abnormal remnants (certain green hues of chromatopsia, deviation of visual image orientation by a few degrees, irradiation from red, somewhat unstable shapes and orthogonal and allocentric failure).

Such functional unity allows, from a single pathological datum, to presume the alteration of the rest of functions. In that regard, the overall state of visual activity in subject M in the inactive state can be summarized in Table R1.

Moreover, by counting on two patients with brain lesions of unequal intensity, it is possible to study sensory organization according to different physiological levels, which implies different degrees of asynchrony and of dynamic reduction. Since in addition, in each patient both eyes are somewhat dissimilar, new different functional levels can be obtained which, together with facilitation by muscular effort and binocular effect, make possible very varied combinations that lead to about fifteen or more types of vision with different functional activity. For example, in the M case, a very substantial change is obtained by switching from monocular vision in an inactive state to binocular vision and maximum muscular effort. The change is even greater between binocular vision in the T case and monocular vision in M inactive. The remarkable differences between the three most frequently mentioned physiological levels in this research are shown in Table R2.

Table R2. Visual functions according to three types of physiological level.

	M inactive	M under facilitation by muscular effort	T inactive
Excitability[2]	OD: rheobase 14.2 V, chronaxie cap. 3.5 µF. OS: more disturbed.	OD: rheobase 9.5 V, chronaxie cap. 2.7 µF.	OD: rheobase 7.8 V, chronaxie cap. 1.4 µF.
Colors	*In medium light*: yellow-blue blindness, etc., intense chromatopsia, pronounced inversion of color isopters, intense alteration of chromatic induction phenomena.	*In medium light*: practically normal color vision. *In very low light*: phenomena of the inactive state.	*In very low light*: tritanomaly (weakness to blue), traces of fleeting chromatopsia, partial inversion of color isopters.
Forms	Visual field up to 6°. Acuity: monoc. 1/25, binoc. 1/10. Strong color irradiation *in medium light*. Severe loss of motion perception. Unstable and diffuse shape perception. Very slow and successive perception.	Visual field up to 40°. Acuity: monoc. 1/8, binoc. 1/6 - 1/4. Irradiation only from red *in medium light*. Motion perception much better than in the inactive state. Better and faster shape perception than in the inactive state.	Visual field up to ~ 50°. Acuity: monoc. 1/3 - 1/2, binoc. 2/3. Weak irradiation only from red. Slightly altered motion perception. Somewhat unstable shapes, and somewhat slow perception *in very low light*.
Orientation	OD max. tilt image: 145°. OS max. tilt image: 170°. Binoc. idem: 115°. *In very strong light*: tilt image about 5°.	OD: from max. tilt image in inactive state and same stimulus, a strong re-inversion is obtained (from 145° to 30° and even 20°). OD max. tilt image: 97°. Binoc. max. tilt image: 27°.	OD max. tilt image: 25°. OS max. tilt image: 16°. Binoc. max. tilt image: 10° or less.
Schema	*Binocularly and in strong light*: illusions, disaggregation, concrete behavior, orthogonal failure, egocentric orientation.	*Binocularly and in strong light*: fairly well in general, but serious defects in complex tests, orthogonal and allocentric orientation. *In low light*: as in the inactive state.	Mistakes only in complex tests, although there are traces of weakness in all of them. Normal orthogonal orientation even in very low light.

[2] More data on excitability of the three types of physiological levels can be found in Table 4.6.

Volume 2

Sensory dynamics. Tactile functions

Sensory structures according to brain synchronization

THE significance of touch is understandable since, for many authors and since ancient times, it has been the primary and fundamental sense.

The similarity between tactile and visual functions is very pronounced; not in vain vision has been referred to as touch at a distance. Thus, we shall find a striking parallelism between the physiological organization of touch and vision in issues such as general excitability, sensation, space and forms, orientation and finally schema.

However, despite the apparent uniformity of the tactile constituent elements, there is a functional complexity perhaps absent in the case of vision. Especially tactile space is a broad field of investigation, and any function developed to some extent must ultimately refer to it, from rudimentary localization to the body schema, going through the remarkable phenomena of spatial inversion.

Tactile functions have always been studied by neurologists given their importance in neurological diagnosis, and recently, after some partial attempts at renewal, these functions seemed to be an exhausted field for research. However, in the case of touch, the dynamic analysis conducted here has once again revealed a multitude of unknown phenomena in all kinds of functions, phenomena that can easily be interpreted from a strictly physiological point of view. We therefore believe that this means a profound transformation, in data and theory, in the knowledge of the sense of touch.

General excitability

17. Electrical excitability

17.1. Strength-duration curve. Hoorweg's law

The determination of tactile electrical excitability has been carried out in identical technical conditions as for the retina, and we refer back to it for methodology. The results are likewise of the same type as for the retina in our two patients M and T.

Due to the special characteristics of excitability in these subjects, it is necessary, as in the case of the retina, to avoid fatigue, every action of facilitation, as well as latent addition of stimuli if determinations are made close in time. In this way we obtain the strength-duration (voltage-capacitance) threshold curve for single stimulation, which corresponds to the true tactile excitability state, that is, in the inactive state free of any summative influence. For the curve of facilitation by muscular effort, we request the subject to contract his entire musculature with the maximum possible strength every time a stimulus is applied.

Disturbances of tactile sensitivity are spread through the entire surface of the body according to the characteristics of the central syndrome. The curves shown in Fig. 17.1 refer to the electrical stimulation of the lip. This zone offers an unusually remarkably lower rheobase threshold, which allows to take better advantage of the voltage scale in this elemental excitability curve and, above all, in other curves corresponding to more complex functional levels, as we shall see later.

Figure 17.1 shows the two curves for subject M, in the inactive state and under facilitation by maximum muscular effort, as well as the curve for a normal subject, all obtained under the same technical conditions, the cathode stimulating the mucosa of the middle part of the lower lip. It should be warned that the sensations obtained are not the same in all cases although we are dealing with the minimal tactile sensation perceptible to electrical stimulation. In the normal subject, a sensation of perfectly localized and circumscribed minimal skin contact is achieved, whereas in the pathological cases, a diffusely spread pressure, like a gust of wind, without any spatial localization is felt, in such a way that the subject feels stimulated on some undetermined part of his body. This occurs both in subject M and subject T. The

sensation of minimal intensity that in a normal case goes together with spatial localization, in our cases is devoid of any kind of spatial organization due to asynchrony. This will be studied in depth in the chapter on tactile space.

As seen in the curves, the rheobase and the chronaxic capacity of the inactive state (the true excitability state) in subject M, are about ten times the normal value. Under facilitation, these parameters are significantly lowered, but the decrease is no more than one third. As can be observed, the behavior of tactile excitability in subject M is of the same quantitative order in all aspects as the retinal excitability already studied. The slower reaction time is reflected in the lower curvature of the voltage-capacitance curves compared to the normal subject. It is also seen in the slight concavity towards the origin of the electricity quantity lines for the inactive and facilitated states, whereas in the normal subject it is a straight line.

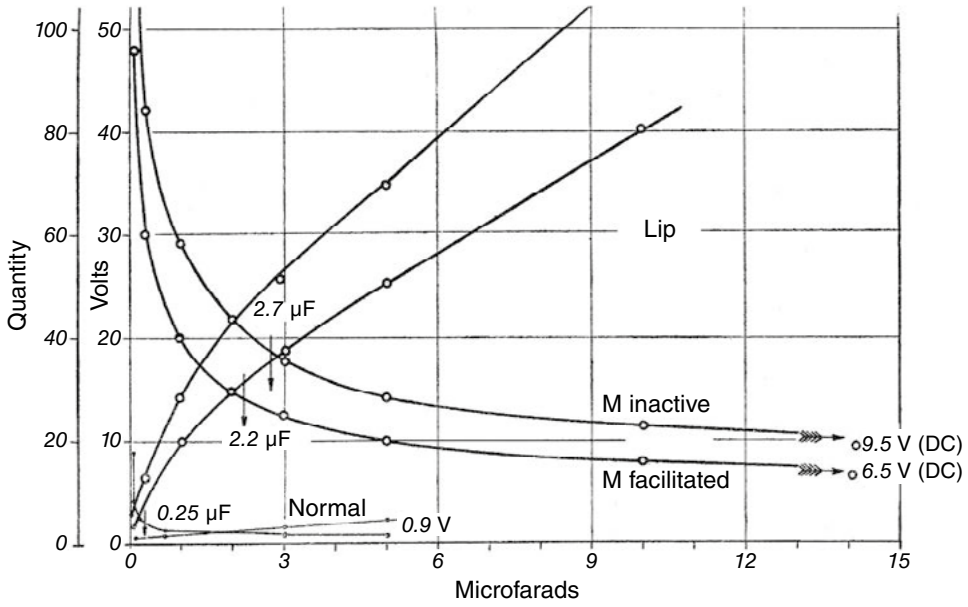

Figure 17.1. Strength-duration curves according to Hoorweg' law (volts vs. microfarads) for the M case with cathode on the midline of the lower lip, in direct current (DC). Note the different curves for the inactive state and the state under facilitation by strong muscular effort, with rheobase and chronaxie values much higher than in a normal subject. Also compare the respective electricity quantity lines (left scale) for the inactive and the facilitated state.

For the T case, the mentioned quantitative parallelism with vision is also valid; thus, in touch, in the inactive state, we found the same increase of four times the normal rheobase and chronaxie values (Fig. 17.2), as in vision. As for facilitation by muscular effort, the decrease in values is very small but sufficiently pronounced to be evident. In Fig. 17.2, the two extreme states of subject T can be compared with the analogous states of subject M and with the normal subject.

As we know, the different curves for subjects M and T correspond to different losses of brain matter destroyed by the lesion. The values obtained are shown in Table 17.1.

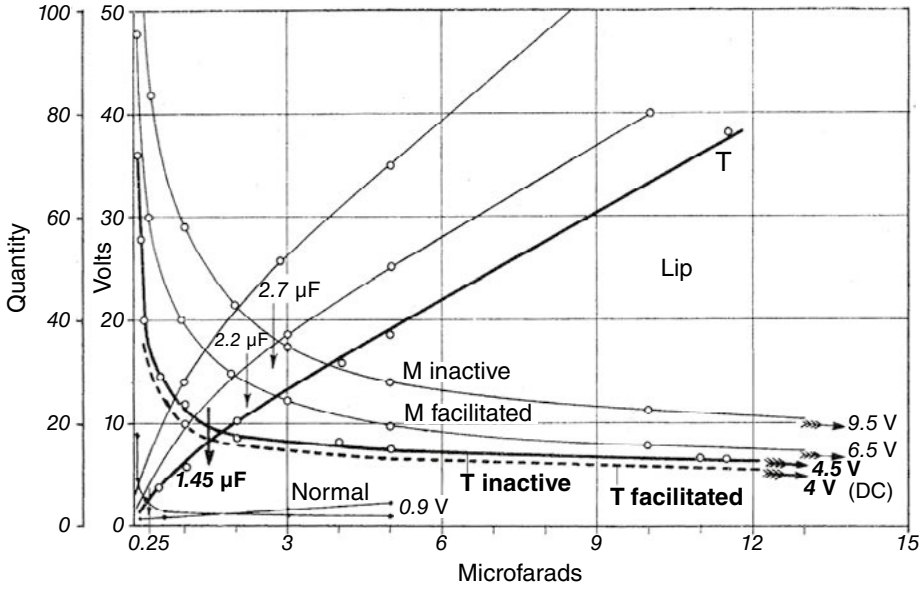

Figure 17.2. Strength-duration curves according to Hoorweg' law (volts vs. microfarads) with cathode on the midline of the lower lip, in direct current (DC), for subject T (thick curves), compared with subject M (thin curves). Note the different degrees of facilitation in these subjects as well as their different rheobase and chronaxie values.

Table 17.1. Electrical excitability (midline of lower slip).

	Rheobase (V)	**Chronaxie (μF)**
M, inactive state	9.5	2.7
M, under facilitation	6.5	2.2-2
T, inactive state	4.5	1.45
T, under facilitation	4	1.25
Normal subject	0.9	0.25-0.3

If this table is compared with the analogous table on electrical excitability of the retina in these M and T cases (Table 4.2 in Vol. 1), it can be seen that the excitability disorder in touch is quantitatively of the same order. Therefore, the excitability curves for the retina (Fig. 4.2 in Vol. 1) and touch (Fig. 17.2) show an identical arrangement in the set of excitability levels studied. It is important to highlight this similarity because it shows that the central syndrome is revealed in a very pure form,

since the excitability deficit is the same quantitatively for both vision and touch. The values obtained in the inactive subject M are about 10 times those of the normal subject, and in subject T they are about four to five times those of the normal subject. We also see that both subjects present in touch the same permeability to facilitation by muscular effort as in the retina.

17.2. THE FACILITATION PHENOMENON

The figures and table above show the different facilitation action in the two patients M and T, i.e., their different *permeability* to *summative effect* on the nervous centers. This facilitation, by lowering the elementary excitability curve, saves energy of the stimulus. For example, the saving at the rheobase threshold is approximately one third in the M case and one tenth in the T case, with respect to their respective inactive states. Permeability to the summative effect is the same for touch as for vision. It was already said that the impact of the central syndrome is the same on the different receptors.

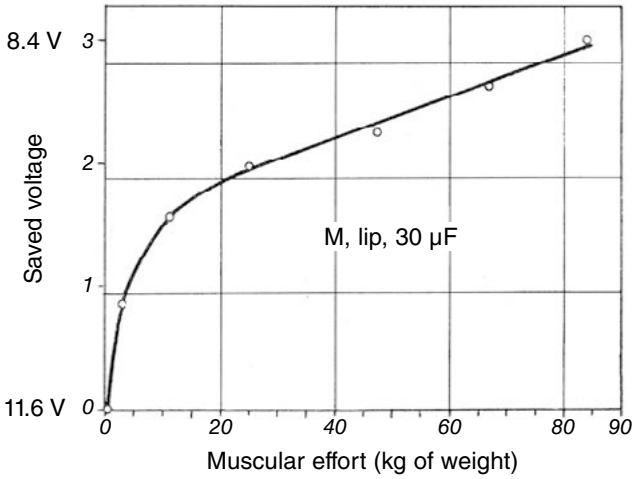

Figure 17.3. Voltage saved as a function of intensity of facilitation by muscular effort (kg of weight held by subject M). Transition from the inactive state to the facilitated state, for a fixed stimulus duration (30 µF).

To know how this saving varies as a function of the facilitation applied, it is sufficient to measure the decrease in voltage (with fixed capacitance close to that of the rheobase threshold) when subject M holds different weights that give the measure of the muscular effort made, analogously to what was done in the case of the retina. Figure 17.3 shows the curve of this voltage saving. Starting from a voltage of 11.6 volts (corresponding to the inactive state), increasing muscle efforts are applied and the voltage for each new minimum stimulation is determined. At the beginning the saving is fast, as shown by the sharp rise of the curve, becoming slower and

slower thereafter. The limit is reached at around 8.4 volts, i.e., after having saved about 3 volts, corresponding to the effort of holding the high load of 80 kg weight. It is notable that low-intensity facilitation already exerts a very favorable action on excitability, as shown in the graph. In fact, small muscular actions, that go unnoticed in ordinary life, act as a not negligible facilitation.

Taking the logarithm of the facilitation, i.e., of the held weight, the relationships between saving and logarithm of facilitation approximates a straight line (Fig. 17.4), so it can be stated that the level of excitability is directly proportional to the logarithm of the applied facilitation. This is a law of great importance for the dynamics of nervous centers. The relationships obtained are of the same kind as those corresponding to this type of experience in vision, and all the considerations made then with respect to vision, are equally valid now.

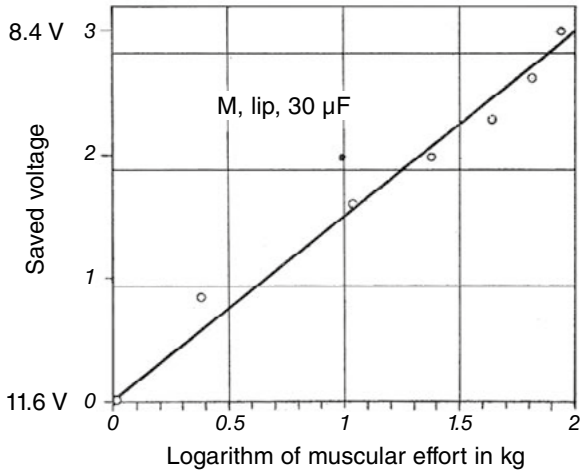

Figure 17.4. The same experiment as in Fig. 17.3 but taking the logarithm of the weights held by subject M. The voltage saved (increase in excitability) grows proportionally to the logarithm of muscular effort.

The same behavior is found for other non-muscular facilitations, i.e., of the inter-sensory (cross modal) type, although their action is much weaker than muscular effort, and can only be clearly evidenced when applied in pathological phenomena with a wide margin of variation, for example diverse alterations of tactile localization, which corresponds to other chapters.

17.3. ITERATIVE EXCITABILITY

Tactile behavior in iterative excitability is of the same kind as that described in the case of vision. Since we have already studied this issue (Sec. 4.3 in Vol. 1), a few brief indications will suffice.

First of all, it should be noted that, unlike the normal retina, normal cutaneous sensitivity has a certain tendency to iteration (addition of successive stimuli), although it is small. Several authors such as Altenburger (1933), Schriever (1933), etc., found an iterative effect only for tactile functions such as pain or temperature, but not at all for pressure or mere contact sensation. However, we have found a certain iteration for mere contact by stimulating the lower lip of normal subjects in order to compare the values with those of our patients. Piéron and Segal (1938) also defend the existence of iteration for mere contact by stimulation with capacitors. The results obtained by the different authors show a certain consensus, since the iterative capability for pressure or contact is notably lower than that for pain and temperature. The low iteration for mere contact compared to the other functions is probably the reason why it has gone unnoticed.

We shall now deal only with iterative excitability for contact (or pressure) in our two cases. It is clear that, as in vision, the iterative disorder is very pronounced, the iteration in the normal subject being almost negligible compared to these cases. By examining subject M (with greater disorder) at the midline of his lower lip, as in the previous tests, we can determine the curves for the law of numbers and the low of intervals in the extreme inactive and facilitated states, as shown in Figs. 17.5 and 17.6.

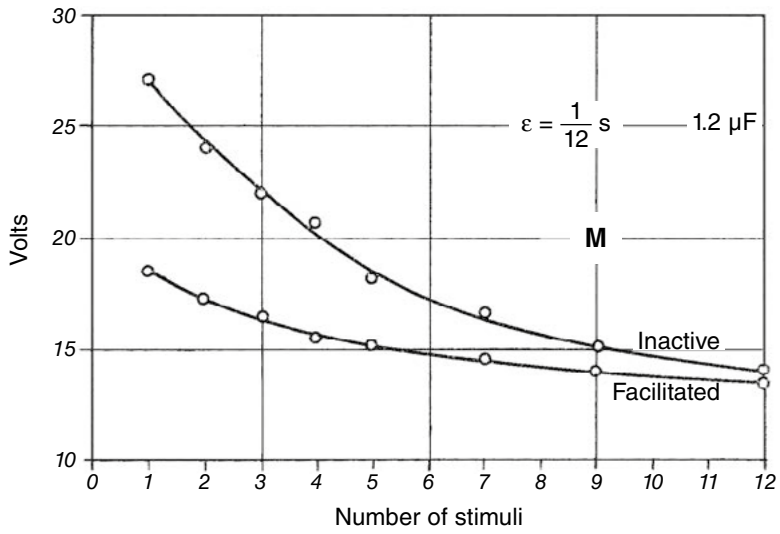

Figure 17.5. Law of numbers in the M case, lower lip midline. Note the different slopes of the curves for the inactive state and the facilitated state. Time interval $\varepsilon = 1/12$ s, capacitance 1.2 μF (half the chronaxie capacitance for the inactive state). The voltage saved is 50% in the inactive state and 30% in the facilitated state.

In Fig. 17.5 it can be seen that the succession of a number of stimuli causes a marked decrease in the voltage necessary to awaken the tactile sensation which, as mentioned above is not localized as in a normal subject. The capacitance used is approximately half that of the chronaxie in the inactive state, and the time interval

between stimuli is $\varepsilon = 1/12$ s. In this way, after 12 stimuli, the voltage for the single stimulus is reduced by about half. With facilitation by strong muscular effort and under identical experimental conditions, a more horizontal curve is obtained, corresponding to a lower degree of summation. Whereas 50% of the voltage is saved in the inactive state, only 30% is saved with facilitation. These values correspond to an iteration of medium degree, in relation to the duration (microfarads) of the stimulus used. Different values can be obtained by varying the time interval between stimuli, as shown in Table 17.2.

Table 17.2. Iterative excitability in subject M (law of numbers).

Cap. (μF)	ε (s)	No. stim.	Volts	Condition	Summat. degree
0.1	1/12	1 12	67.5 16	Inactive	76 %
		1 12	49 14	Facilitated	71 %
1.2	1/12	1 12	27 14	Inactive	50 %
		1 12	18.5 13.5	Facilitated	30%
20	1/12	1 12	10.6 8	Inactive	27 %
		1 12	8 7.3	Facilitated	10 %

A behavior similar to that described in the iteration by electrical stimulation of the retina is thus obtained. Suffice it to say that subject M presents in the sense of touch a strong latent addition of stimuli that can reach a degree of summation of 76% or more.

As for the law of intervals in this case, Fig. 17.6 shows the curves for the inactive and facilitated states using a capacitance equal to that used in the law of numbers in the previous figure. Twelve stimuli are used for each determination. By varying the time interval between successive stimuli, it is obtained that the smaller the interval the greater the saving of the threshold voltage. Considerations made on the law of numbers are valid for this law of intervals since it presents the same characteristics under another aspect.

Concerning subject T, of faster excitability than M, we only show Fig. 17.7 on voltage saving as a function of the number of stimuli. Using a capacitance half that of chronaxie in this subject T, a degree of summation similar to that of subject M under facilitation is obtained, although now the capacitance is much lower. Subject T requires 12 volts for a single stimulus whereas he requires only 8 volts for 12 successive stimuli.

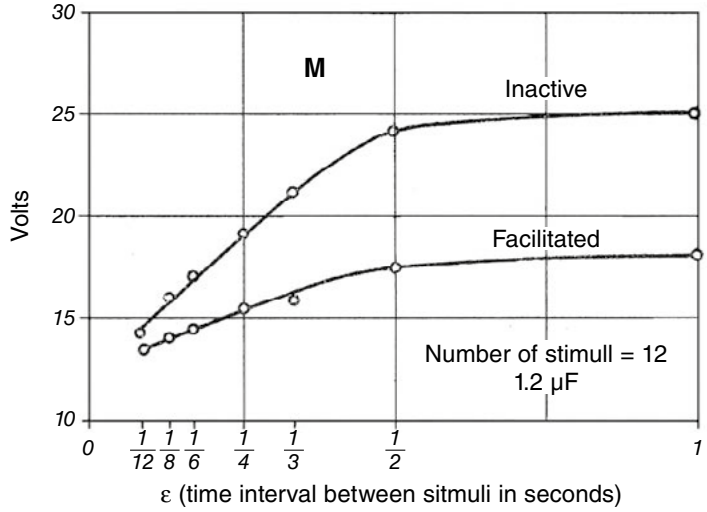

Figure 17.6. Law of intervals in the M case, lower lip midline. Capacitance (stimulus duration) as in the previous figure. Note the different slopes of the curves for the inactive and facilitated state.

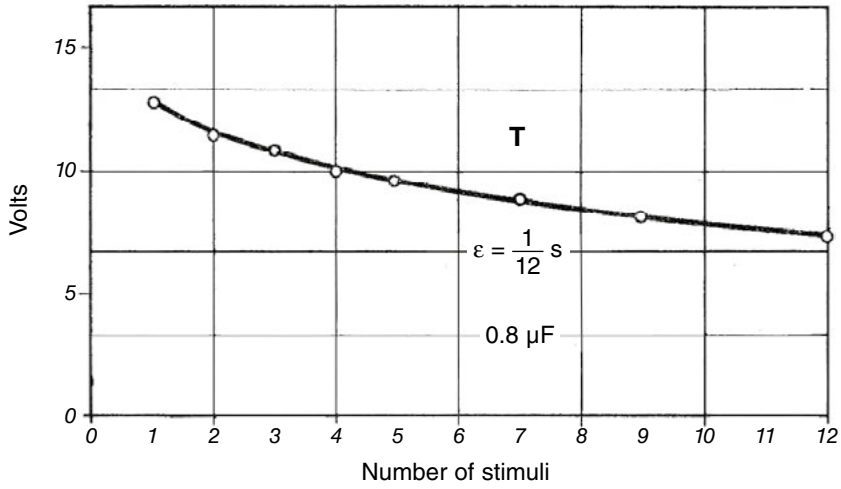

Figure 17.7. Law of numbers in the T case, lower lip midline. The capacitance is half that of chronaxie. Degree of summation about 30%.

With respect to the relationship between the increase in chronaxie and the degree of iterative summation, as well as other considerations on variation by facilitation, etc., we refer to the corresponding section on vision in Vol. 1 in order to avoid repetition. The most important conclusion now lies in the quantitative similarity between the tactile excitability disorder and that of the retina, a consequence of the *homogeneous impact* in pure central syndrome.

18. Mechanical excitability

18.1. GENERAL REMARKS ON ADEQUATE STIMULUS

The above data on electrical excitability, which allow us to appreciate with great accuracy the functional state of touch in the two cases studied, should be completed with mechanical stimulation. We shall constantly make use of the data under this type of stimulation in the study of complex functions in following chapters.

By applying an esthesiometer to the skin of a hand, with a pressure of a few grams, the required application time (useful time) to reach the sensation threshold in subject M in inactive state is about six to seven seconds. This is an extremely high value compared to the normal subject, who in addition to reaching the sensation with a stimulus of much lower intensity, needs at most a useful time of 1/3 second. For the state of maximum facilitation in M, the useful time is reduced to about four seconds in addition to lowering the stimulus intensity. In the much less affected subject T, the useful time is about 2 seconds. Such data according to the different intensity of the brain lesion and different facilitation compensation, follow the law of Hoorweg (1892), and show a behavior similar to that of the light excitability of the retina.

It is important to indicate the different modes of application of a stimulus on the skin: 1st, by a single, fixed contact of desired intensity and duration; 2nd, by reiteration of a stimulus at given time intervals; and 3rd, by a kinetic stimulus sliding over the surface of the skin. The above data on useful time correspond to the first method. As for reiteration, a single stimulus that does not cause sensation, applied repeatedly in the same place produces a cumulative effect, and sensation is easily achieved by "temporal summation," provided that the reiteration is sufficiently rapid in relation to the characteristics of the subject's excitability. It should be noted that iterative stimulation does not need to be applied at the same site on the skin; thus, successive stimuli can be applied at different sites close to each other, with a small time interval, until a minimal contact sensation is triggered. In this case, in addition to temporal summation, there is a "spatial summation" effect as in the case of the facilitation phenomenon.

This leads us to consider stimulation by a kinetic stimulus, for example, a piece of cotton sliding over a limb or the whole face. If the stimulus is motionless on the skin, it does not cause sensation no matter how much time elapses, due to its weak pressure, whereas by sliding the stimulus over the skin, there comes a moment when contact sensation is triggered. This requires that the moving stimulus covers a certain space (sufficient number of receptors) and at a certain speed (sufficient frequency of receptors). If this speed is extremely small, the result is the same as if the exciting agent were completely motionless. It is then clear that the conditions in a moving stimulus are the same as in the iteration with a single stimulus repeated at different locations (temporal and spatial summations). A stimulus of subliminal intensity that slides over the skin, causes successive nervous modifications of a certain duration that are accumulated (temporal summation) until, after a certain distance (spatial summation), the sensation appears. The slow reaction time of the nervous centers allows the kinetic stimulus to give rise to the accumulation of the different spatio-temporal residues. In these subjects, stimulation with a moving object on the skin is therefore a mixture of excitation by temporal and spatial summation. In all these stimulation modes and when the energy does not exceed a certain limit, only a sensation of contact with an extensive irradiation[1] and lack of spatial localization is obtained, as we have already indicated with respect to electrical stimulation. Therefore, with a kinetic stimulus, no line or movement is perceived on the skin, and the sensation thus obtained does not differ from that obtained with the other modes of stimulation. Usually, with a kinetic stimulus of a certain speed, a path of about 10 cm in length is sufficient to obtain a contact sensation.

According to what is said, the development of tactile sensation is rather slow depending on the case. In the inactive subject M, maximum latency (delay) is obtained between the application of the stimulus and the appearance of the sensation, which, in addition, lasts for a certain time after the stimulus has been suppressed. Of particular interest is this persistence of the sensation, which in the inactive subject M can reach about two seconds at most. This persistence gives rise to very remarkable phenomena if facilitation acts on the sensory residue. Thus, a sensation of simple contact (without any localization in these cases) during the period of persistence after the stimulus has been removed, can be markedly intensified if facilitation by intense muscular contraction of the whole body is rapidly applied. This intensification not only increases the clarity of the sensation, but also endows it with a spatial nature, making its localization possible. Obviously, the new effect achieved is extremely brief but sufficient for the change to be felt.

Such "reactivation" of the sensory residue is also obtained in the case of vision. For example, let us consider a vertical arrow in conditions such that the image perceived by the inactive subject M is tilted 90°; it turns out that when the arrow is removed from the visual field and facilitation by muscular effort is immediately introduced, a tilt of about 30° is obtained and all perception disappears very rapidly, since the facilitation has acted only on the residual image of the sensation, i.e., on

[1] As in vision, the term 'irradiation' indicates a type of widespread and diffuse spatial localization.

the residue close to extinction. The longer the time elapsed between the exclusion of the stimulating agent and the application of facilitation, the lower the sensory level obtained, since summation by facilitation is exerted on an increasingly weaker sensory residue. In both vision and touch, if the elapsed time is 1.5 seconds, reactivation is almost nil, and several tests indicate that in both sensory systems, the maximum sensory persistence does not exceed 2 seconds. As for the dependence of the degree of intensification and localization of the tactile residue on the time at which facilitation is applied, see the chapter on tactile space for understanding the phenomena of desynchronization in spatial localization.

Concerning the facilitation effect, we know from electrical excitability its different effect in the M and T cases. In the T case, facilitation also exerts an unquestionable summative effect on mechanical tactile stimulation, although to a lesser degree than in the M case. Except for muscular contraction, other types of cross-modal or inter-sensory facilitation are of little importance in touch. Thus, both acoustic and visual stimulation hardly facilitate tactile function, due to the minimal central effect they probably exert. By contrast, the sensation of a tactile stimulus is significantly improved when a second tactile stimulation coexists, such as a gentle touch on another region of the skin. Thus, a bi-tactile effect (spatial summation of stimuli) is obtained.

Sensory fatigue is very noticeable in our cases. Using a minimal stimulus in subject M to obtain a sensation of simple contact, and maintaining that stimulus indefinitely, it happens that after about 30 seconds from the emergence of the sensation, it ceases to be perceived. If the maximum facilitation by strong muscular effort is then applied, the sensation reappears and, moreover, it is localized, a fact that did not occur before. But when facilitation stops, the localization disappears immediately and the remaining sensation of contact is lost more quickly than in the previous experiment, in just about 10 seconds. This fatigue may be due in part to a phenomenon of sensory accommodation (Adrian 1928), but to a greater extent is due to an increased threshold caused by sensory fatigue.

Regarding tactile adaptation, it is worth mentioning the process of sensitive adaptation or tactile recovery after some alteration of the sensory organ. For example, when altering a fingertip by rubbing it over a rough, hard surface for half a minute, the normal subject recovers most of its pre-alteration sensitivity within a minute after the end of the rubbing, whereas the inactive subject M may take up to four minutes. Since we have not quantitatively determined the evolution of this adaptation process, we can only give rudimentary data, but it is to be expected that it follows an analogous behavior to the adaptation process studied in vision.

The different behaviors of subject M in relation to tactile sensation can be illustrated by considering weight sensation. He greatly underestimates a considerable weight held in his hand. This is demonstrated because under facilitation by strong muscular contraction, the sensation of weight increases greatly, well over twice, as indicated by the subject. The differential threshold also increases; thus, when successively holding different weights with the same hand, or with both hands at the same time, weights of 0.5 kg and 1 kg seem nearly the same. Only when he makes a few small movements to compare the weights, does he perceive a minimal differ-

ence. Under facilitation by muscular effort and without movements to compare the weights, both are perceived as heavier and very different from each other, to the point of presuming that one is twice as heavy as the other.

In the inactive state and without any type of movement, subject M perceives a difference, although not excessive, when comparing weights of 0.5 kg and 2 kg. This difference increases when performing movements to compare weights. With facilitation, the perceived difference can become three times greater than in the inactive state, and even greater with movements to estimate weights. Thus, in the M case, there is a decrease in the perceived weight (absolute threshold) and in the comparative differences (differential threshold), varying according to the corrective effect of facilitation, which never entirely normalizes the function.

To obtain sensations of similar intensity in the inactive state and under facilitation, different weights are needed to compare them successively. Thus, 1 kg in the inactive state can be equated to 300 grams under facilitation. Sometimes an equivalence is also obtained between 1 kg in the inactive state and 0.5 kg under facilitation. However, the subject rejects that 1 kg in the inactive state is equal to 200 grams under facilitation because he perceives that the latter weighs less. All this means that weights are perceived to be about three times heavier under facilitation than in the inactive state.

All these determinations refer to evaluation of strengths, since the weights hang from a ring that the subject grasps with his fingers. It is clear that these measurements are not very accurate, but they are useful to get an idea of the subject's functional capacity. In particular, the equating relationship between inactivity and facilitation seems to be well established since it is verified in other types of tests described below.

18.2. VIBRATION SENSITIVITY. INTERMITTENT STIMULATION

The examination of vibration sensitivity in our patients by means of tuning forks of different frequencies will show important features about intermittent stimulation, appreciation of time intervals, fusion of successive stimuli, etc.; all related to the nervous excitability disorder.

Regarding vibration sensitivity, there are very discordant opinions among authors. Dejerine and Dejerine-Klumpke (1914) and Egger (1899) consider it to be a specific sensitivity of the osseous system. Later it was shown that the sensation was equally obtained on soft tissues. In particular, Frey (1910, 1913, 1916/1917, 1928) argues that it is a modality of pressure sensitivity that belongs to cutaneous sensitivity and has no relation to deep sensitivity. Bones are particularly sensitive to vibrations because they transmit sensation to a large surface of skin. Piéron (1936) states that there is no independent sense of vibration, but a vibratory excitability as a type of mechanical excitability involving superficial and deep receptors, predominating on bones for anatomical reasons. More modern neurologists think, in line with Frey, that vibration is not a sensory individuality, although there are still many who believe otherwise.

Given the excitability characteristics of our brain-injured patients, the mere application of a tuning fork on the skin (soft or bony parts) is not sufficient to get an idea of the state of vibration sensitivity, since we could make the mistake of accepting a loss of it. As in other determinations, the correct approach is to study the function along a quantitative scale, accurately determining excitability characteristics. For this purpose, we use a series of tuning forks for clinical use with frequencies between 32 Hz and 1024 Hz. The tuning fork is struck at maximum and immediately placed softly on the left styloid process of the radius in such a way that the contact or pressure of the tuning fork does not mask the sensation of vibration. The results obtained in both subjects are shown in Table 18.1 where the duration of the perceived vibration for tuning forks of different frequency is indicated.

Table 18.1. Vibration sensitivity. Duration (seconds) of the perceived vibration.

Frequency (Hz)	M inactive (s)	M facilitated (s)	T inactive (s)	T facilitated (s)	Normal subject (s)
32	4.5	7.5	12	18	25
64	4	6.5	12	18	30
128	2 – 1.5	3	8.5	13	18
265	0.7	1.5	5	8	12
435	–	?			
512	–	–	4	6	9
1024	–	–	–	–	??

It should be noted that the tuning forks have unequal mass, so the duration of vibration is different. However, the normal subject is a good reference with which to compare the pathological cases. First of all, the different duration of vibration perception in the different cases is noted, as well as the different upper limit of frequency, all related to the already known characteristics of excitability. Under maximum facilitation by strong muscular effort, the duration in subject M almost doubles that of the inactive state, whereas in subject T the duration increases 1/2 that of the inactive state. Note that the duration in subject T under this type of facilitation is still well below that of the normal subject, although T is able to perceive all the frequencies of the normal subject. In contrast, M loses high frequencies, and for those at which he perceives vibration sensation, the duration is very brief, especially in the inactive state. Subject M in the inactive state feels vibration only in very low frequencies, always during a brief period of time, but when examined with a tuning fork for clinical use such as the one of 435 Hz, he is not able to feel the least sensation of vibration, and there is also great difficulty when using the 256 Hz tuning fork because of the short duration of the sensation. However, it would be a mistake to deny him capability to feel vibration sensation, since this is possible using very low frequencies. As for the normal subject, with the 1024 Hz tuning fork, it is very difficult to obtain a vibration on the styloid process of the radius, and one can practically say

that there is no perception. Only by paying close attention and striking the tuning fork strongly, it is perhaps possible to feel a very slight vibration signal of negligible duration.

Apart from the considerable reduction of vibratory sensitivity in our patients, especially in the inactive M, we must consider the evolution of the sensory process just after the tuning fork is applied, in order to understand the nature of the disorder. In the normal subject, the latency time from application to vibration perception is negligible compared to pathological cases, in which this time can be easily measured with a stopwatch. In subject M inactive, using a low frequency tuning fork, there is a latency of 1 to 2 seconds. However, it is very important to note that this latency time is not to awaken the sensation of bone tremor, but to obtain only uniform contact sensation without any vibration. This sensation of uniform contact lasts in turn for some time, and is followed by the sensation of vibration as a small tremor. In the above Table 18.1, the duration values refer exclusively to this true vibration phase, not to the whole sensory process. The process ends by evolving from tremor to uniform sensation again and, finally, to the complete loss of all sensory activity. It should not be considered extraordinary that even the sensation of contact ceases even if the tuning fork is still applied to the skin, since the tuning fork does not press on the skin with its full weight, but is applied laterally to transmit exclusively the vibratory movement. In addition, it should not be forgotten the high threshold of excitability due to the brain disorder, especially in the inactive state. When the rather large tuning fork of 32 Hz is applied, without vibrating, with its full weight on the skin of the inactive subject M, it takes him 5 to 6 seconds to perceive the sensation of contact, whereas when it vibrates, it takes only 1 to 2 seconds to have the same kind of sensation. This clearly indicates that in vibratory stimulation there is an accumulation (summation) of discontinuous stimuli.

By studying in detail the evolution of the vibratory sensory process when a 32 Hz tuning fork is used in the inactive subject M, several phases are determined that depend on the changes in nervous excitability during the tuning fork action time. A diagram of this process is shown in Fig. 18.1.

In addition to the aforementioned sensory latency, there is a phase of continuous sensation and then a phase of vibratory sensation. The first corresponds to the fusion of stimuli, that is, to the fusion of the vibratory tremor. In this phase the excitability is still low, and the vibratory impulses are perceived so slowly that there is no possibility to discriminate between them and they seem fused. However, since sensitivity tends to improve by accumulation of discontinuous impulses, there comes a time when the tremor can be perceived because the sensation becomes more intense and rapid. However, the interval that can be perceived is much smaller than that perceived by the normal subject; therefore, the vibratory tremor results to be overestimated since it is perceived very quickly. Thus, facilitation not only intensifies the sensation but also makes the perceived rhythm slower, although it is still faster than in the normal subject.

The situation is the same as for intermittent stimulation in vision. In a normal subject, it already happens that when fusion is obtained for a given rhythm and luminous intensity, if the illumination is increased, the discontinuity of the flickering

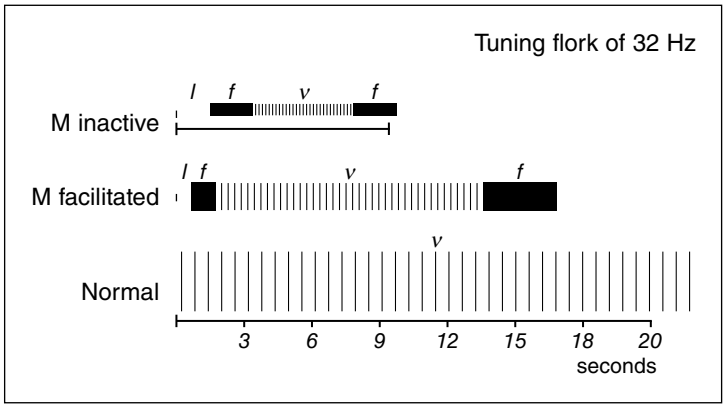

Figure 18.1. Qualitative diagram of vibratory sensory phases. Latency, *l*; fusion, *f*; vibration, *v*. In the diagram, the scale is not the same for the three cases. See data in Table 18.1. Note the different duration of the phases in the three cases considered. The height of the lines indicates the intensity of the sensation.

appears because the reaction becomes faster. Here, in tuning fork vibration, the indicated change is due to the improvement of the sensory level as a result of the accumulation of discontinuous stimuli, which is very easy in view of the high iterative capacity of subject M.

As for the disappearance of sensation, the process occurs in reverse order, i.e., from vibration to fusion and, finally, to sensory silence even though the tuning fork is still applied on the skin. To interpret this process, one must realize that after the tuning fork has begun to vibrate at its maximum by the action of a strong blow, it tends to dampen the intensity of the vibration and therefore, the stimulus becomes less and less intense and the process of evolution of the sensation reaches a point where it is reversed, ending as it began. The duration of the whole process is very short in the M case compared to the T case, and much shorter than in the normal subject, either because the small decrease of the tuning fork vibration intensity influences very soon to lower the level of functional activity prematurely being impossible to maintain an indispensable minimum, or because in these subjects there is a very rapid adaptation to vibration which tends to erase it (Adrian 1928, Piéron 1936) as it happens also in the normal subject. In either case, both factors could act together (see Fig. 18.2).

It should be noted that the phases correspond to different spatial levels. In the phases of mere contact or fusion there is no localization, whereas in the vibratory tremor phase there is a spatial localization that is more perfect (in accordance with the real place stimulated) the clearer and more lasting the vibratory sensation is. Quantitative data on this point are given in the chapter on spatial localization further on. Regarding the indicated phases, when a tuning fork of 435 Hz is applied laterally on the styloid process of the inactive subject M, he perceives neither tremor nor contact or fusion; instead, under facilitation, he goes beyond the fusion phase but does not clearly perceive the vibration, which seems practically null.

The exclusion of high vibration frequencies (see Table 18.1) can be explained by the impossibility of latent addition due to the extremely small interval between stimuli that would appear in the refractory period of the preceding stimulus. We know that the smaller the interval, the greater the degree of summation, but this occurs up to a certain frequency ("optimum frequency") beyond which, if the time interval decreases, the degree of summation also decreases. Finally, the degree of summation becomes null when the time interval is equal to the duration of the refractory period of the first stimulus (Bremer 1930 a, Bremer 1930 b, Koehnlein 1934). Given the great slowness of nervous processes (hence increased refractory period) in our brain-injured patients, especially in the inactive subject M, the limit for latent addition is reached very soon, and for this reason tuning forks of medium and even low vibration frequency (such as 256 Hz) hardly produce a slight and short-lived vibration sensation (Table 18.1). Since tuning forks are not of the same mass, it is difficult to correctly establish comparative relationships, but it is perhaps possible to state that almost all tuning forks whose vibration can be perceived by the inactive subject M are in a range in which the latent addition tends to decrease when the frequency of vibration increases.

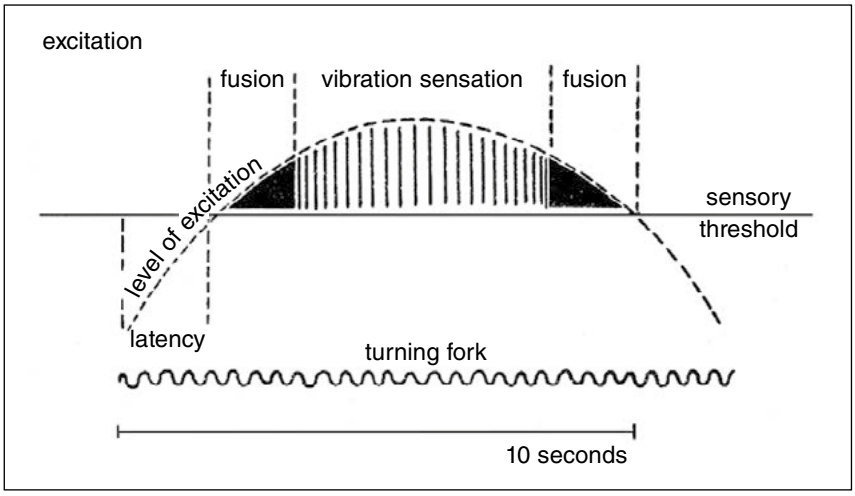

Figure 18.2. Diagram of the sensory periods according to the level of excitation (dashed curve) during vibration stimulation in subject M inactive. Same conditions as in Fig. 3.4.

This same slowness in nervous reaction explains that when the tremor is finally felt, it is perceived much faster than it really is, due to the fact that the interval between stimuli becomes very short. This perception is similar to that in intermittent visual stimulation with respect flickering and fusion (Sec. 5.3 in Vol. 1), and in general with respect to the perception of movement, which in both vision and touch become accelerated. Facilitation, by improving excitability, corrects in part this acceleration, lengthening the sensory interval somewhat.

Thus, it is possible to obtain the same vibratory sensation in the inactive state and the state under facilitation; for example, it is possible to feel tremor of equal frequency both with a 32 Hz stimulation in the inactive state and with a 128 Hz stimulation in the facilitated state by strong muscular effort. In general, facilitation produces a decrease in speed two or three times that perceived in the inactive state (for a suitable tuning fork); therefore, when the tuning fork frequency perceived under facilitation is two or three times higher than that perceived in the inactive state, equality of sensation can easily be achieved by means of facilitation. It can then be easily inferred that the vibration felt by a normal subject with a low-frequency tuning fork corresponds to an extremely high vibration sensation in subject M inactive. And given this correspondence, when using medium or somewhat high frequencies, whereas the normal subject still perceives them easily, they produce only fusion of stimuli or no sensation at all in subject M inactive. In relation to what said, the following equivalence between frequencies that produce the same sensation is plausible: 32 Hz in M inactive =128 Hz in M under facilitation = 435 Hz in T = more than 512 Hz in normal subject. The relationships between the values are very similar to those of the respective chronaxies.

As for frequency discrimination by the inactive subject M, the 32 and 64 Hz tuning forks at first seem the same to him, and later he thinks there is some difference, although very small. He appreciates the difference between the 32 and 128 Hz tuning forks much better. Under facilitation, the difference between 32 and 64 Hz is easily perceived.

All the above tests refer to the application of the tuning fork on the bone (styloid process of the radius), but can be approximately valid as well for a soft part of the skin, such as the fingertip. In this last case, the sensation is weaker and more pressure is required when applying the tuning fork.

The study of the factors that come into play in the perception of vibration by tuning forks in these subjects shows quite clearly that vibration is only a modality of excitation of the ordinary sense of pressure or contact, since the properties of vibration correspond entirely to the characteristics of pressure or contact in rhythmic stimulation. Consequently, any specificity of a presumed sense of vibration must be rejected.

Some clinicians think that there may be a separation between vibratory sensitivity and surface touch sensitivity, as if they were independent senses, for example, with regard to certain spinal syndromes. In an observation of Guillain (1905), the elementary sensitivity was abolished, but the vibration of a tuning fork was perceived. In such a case, it is to be thought that, whereas a single stimulation by pressure was not perceived, the accumulation of stimuli by the tuning fork finally resulted in a sensation, even a tremor sensation. There is therefore no real separation between specific senses, but rather different reactions to different modes of stimulation, and the severed or injured spinal cord is only able to respond to iterative stimulation. We give this example to highlight the inconsistency of establishing many clinical "specificities" without having studied in depth the physiological nature of sensory and nervous processes.

As for the cortical disorders of tactile sensitivity, it can be stated that tactile sensitivity is very easily altered since it is a discrimination function (perception of

very short intervals), showing in general a behavior similar to that of sensitivity to surface or deep movement. We must not lose sight of the fact that, as already stated in this research, the alteration of a given sensory system is always global, thus affecting all types of activities, which appear reduced according to their physiological demands. The degree of vibration disturbance will then depend on the degree of brain excitability disturbance, as indicated in Table 18.1. This table demonstrates the need to test vibration across a range of frequencies to provide a clear picture of the state of the function.

APPENDIX: OTHER CASES WITH BILATERAL TACTILE DISORDER IN UNILATERAL CORTICAL LESION (CENTRAL SYNDROME)

In this section we draw attention to a number of cases reported in the neurological literature in which different tactile disturbances have been observed in both halves of the body despite presenting a unilateral lesion. Such manifestations have remained without proper interpretation, and for our part we can assert that they simply constitute alterations of the central syndrome type, i.e., the prototype of disturbance in brain dynamics.

A first case of this nature is the one described by Oppenheim (1906). Over the course of a year, the subject presented weakness for movements on the entire right side of the body, but only abolition of fine movements of the fingers. He also had Jacksonian seizures on the right side with subsequent generalization to the whole body. Deep tendon reflexes were slightly hyperactive on the right side, with doubtful Babinski's sign. As for sensitivity: on the *right side,* slight decrease of tactile sensitivity; in addition, considerable alteration in perception of attitudes and very great astereognosis. On the *left side,* normal motor functions and sensitivity, but astereognosis was present.

The author was inclined to admit the existence of a unilateral lesion on the left parietal region and he tried to explain the bilateral astereognosis on the basis of the functional dominance of the left hemisphere. Therefore, surgery was advised. A tumor was found, the size of an egg, on the middle part of the postcentral gyrus and posterior parietal lobe. Five days after tumor resection, the astereognosis of the left hand had diminished considerably.

In 1915, a similar observation was made by Goldstein (1915). A 53-year-old man developed a right incomplete hemiparesis, bilateral apraxia and bilateral astereognosis, within a period of one year. Language was possible for simple words, and affected for more complex functions. Elementary sensitivity to touch, pain and temperature were little altered. By contrast, the appreciation of positions was very disturbed on both sides of the body. The author states that the alterations in sensitivity are not so great as to explain the patient's severe astereognosis.

Necropsy revealed a large cystic cavity in the left hemisphere extending to the postcentral, supramarginal and inferior parietal gyri. Histologically, no anatomical abnormality in the right hemisphere was found to explain the left astereognosis.

In these two first cases of bilateral astereognosis with a single left lesion, we are dealing with an incompletely examined central syndrome. In the former, the side opposite to the lesion is slightly more affected (as in our case T), and elementary alterations of tactile sensitivity, although slight, are clearly observed; instead, in the homolateral side, only the perception of spatial forms is affected. In this left side, touch is less impaired, but it is possible that elementary disturbances have gone unnoticed, and a quantitative examination of excitability would have shown a significant generalized disturbance.

As for the second case, alterations of the elementary sensitivity for contact, pain and temperature are observed, and if they are less pronounced than the perception of position or configuration of objects is due to the exclusion of different functions according to their different physiological demands (dynamic reduction). The dominant action of the left hemisphere is not enough to explain everything. In addition, we have verified the dynamic repercussion of the central syndrome in both left and right unilateral lesions, as will be seen in the corresponding place. The supposed particularity of the left hemisphere is already a repercussion, but this issue is left for other chapters. Since these cases are of the central syndrome type, many other bilateral disturbances of other sensory systems -and in all their activities- should be present, but are easily overlooked in ordinary examinations. However, in the case of Goldstein (1915), more varied symptoms (astereognosis, aphasia, apraxia, etc.) are mentioned.

The characteristics of the central syndrome are even more evident in the four cases collected by Foix (1922) with a unilateral brain lesion and a clinical syndrome characterized by: hemiplegia and aphasia of variable intensity, ideomotor apraxia and bilateral alterations of sensitivity. Regarding the latter, the author specifies that these alterations predominate in the notion of position, and secondarily on the stereognostic sense; other modes of sensitivity being only affected in an occasional way. The author interprets the impairment of touch homolateral to the lesion as anesthesia due to agnosia. He insists on asserting that the whole set of symptoms undoubtedly depends on a single lesion. We note that in these cases the bilateral sensory alterations evolve in a completely parallel way on one side and on the other, as well as with respect to the rest of the symptoms.

> In one of these cases of Foix (1922), the examination of sensitivity shows on the right side (with hemiparesis): moderate hypoesthesia for all modalities (evident alterations of the notion of position, tactile sensitivity, stereognostic sense, etc.). On the left side: very evident hypoesthesia, slight touch lost on all the side except on the face, very altered position in small joints, delay of heat perception and evident astereognosis.

The same features are present in the other cases of Foix, in which the intensity of their involvement varies somewhat, but they always show bilateral pathological manifestations in sensitivity, and almost always a little more pronounced on the paretic side. The most severe disorder is for position sense, but on the left side, homolateral to the lesion, minor alterations of elementary sensitivity (omission of some contacts, delay in the perception of pain and temperature) are also found in a very constant way in most cases.

The location of the lesions in the cases of Foix (1922) is very similar to that of the case of Oppenheim (1906) and the cases of Goldstein (1915).

We can say that in all these observations we are dealing with tactile disorders of the central syndrome type and in a rather pure form, i.e., with a well pronounced symmetrical distribution (bilateral parallelism highlighted by Foix), since the small difference in favor of the contralateral side to the lesion is almost negligible, as is the case in our T case. The difficulties to explain such symptomatology within the brain localization system are obvious. Foix (1922) arbitrarily mentions "anesthesia by agnosia" to interpret the tactile disorder homolateral to the lesion, also assuming the functional supremacy of the left hemisphere. But it is evident that in this way, the sensitivity disorder for elementary sensations (pressure, pain, temperature) remains unexplained, and as for other perceptual activities, the interpretation is rather forced. Proof of this is that some authors try to understand the cases of Oppenheim (1906) and Goldstein (1915) (with bilateral astereognosis due to a left tumor) as due to bilateral lesions, even if they are minimal, in order to explain the homolateral tactile alteration within the brain localization system.

In short, for all these cases, some authors invoke the dominance of the left hemisphere (when the lesion is on that side) while others, not believing that this is sufficient to explain the variety and depth of the symptoms, are in favor of the existence of bilateral lesions. Finally, within brain dynamics, such cases are easily interpreted as types of central syndrome, which even allows assuming the existence of many other symptoms in other sensory systems.

We find again all these difficulties in the study of Goldstein and Gelb (1919) on the tactile functions of the Schneider case, increasing even more the complication with new theories that far from solving the problem, make it more obscure. But before commenting on some circumstances of the Schneider case, it is worth noting that many of the cases of visual agnosia studied by Stauffenberg (1914), also with bilateral astereognosis, correspond to the central syndrome as well. Elementary tactile sensitivity should also have been more or less impaired, and if the disorder of form recognition was more prominent than others, it was only due to the characteristics of the dynamic reduction, already repeatedly indicated.

Moreover, certain experiments of resection in the cerebral cortex of vertebrate animals allow to obtain favorable evidences on the importance of the quantity of removed gray matter (magnitude of the lesion) for the intensity of the functional disorder. This supports both the investigations of Lashley (1929) and the fact that the effects of lesions depend on their magnitude and position according to the present brain dynamics investigation. For example, Rothmann (1914) obtained in the macaque, by simultaneous resection of the postcentral gyrus and the supramarginal gyrus, much more severe and lasting alterations than those obtained by the isolated removal of either of them. There are also analogous observations by Minkowski (1917), in which the more complete the resection of the parietal lobe, the more intense and long-lasting the tactile sensitivity disorder.

Likewise, we must bear in mind the experiments of Pavlov (1927) on the destruction of the cortical area of the analyzers. Such experiments show that discrimi-

nation of stimuli decreases as the quantitative destruction of this area increases, this being valid for any sensory system.

In the solution of these problems, particularly in the human brain, careful clinical observations and proper analysis of the altered functions play the main role. The Schneider case of Goldstein and Gelb (1918, 1919), although lacking a proper physiological examination, has been significant for the very detailed clinical study of the intricate visual and tactile disturbances, but above all, for the singular characteristics of the disorder. In touch, there is an alteration of spatial functions (localization, spatial discrimination, forms, etc.) and of recognition, all of these alterations varying with certain muscular jerks or twitching movements.

In the Schneider case there was also a bilateral alteration of touch as in a case of Goldstein (1915); but whereas in the case of 1915 the author is inclined to admit the predominance of the left hemisphere, as Oppenheim (1906) in his case, in the Schneider case the interpretation changed radically. This is because upon detection of a complex visual disorder, the entire bilateral spatio-tactile disorder was made dependent on the loss of the visual spatial influence. Therefore, touch is considered basically intact. Thus, the disorder lies in the visual system, and this in turn has psychological effects on touch, depriving it of its spatial quality.

Besides the fact that this hypothesis of visual influence is a matter to be proved, it must be noted that in both the case of 1915 and Schneider, elementary sensitivity is not completely intact. Hence, the debatable hypothesis of this influence cannot even be accepted as an explanation for the tactile disorder. In the first case (1915), elementary sensitivity to touch, pain and temperature is little altered. As for the Schneider case, the second work on this patient points out that the elementary sensitivities (pressure, pain, temperature), although essentially intact, are somewhat reduced over the entire surface of the body, i.e., on both sides. But Goldstein and Gelb (1919) think that this decrease in elementary sensitivities should be interpreted as a deficit of attention on the part of the patient during the examination, and in an explanatory footnote they ask themselves "how else could a decrease in sensitivity over the entire surface of the body occur?". This question ceases to be problematic in the brain dynamics we present, since the alteration of elementary excitability for the whole body in a unilateral lesion corresponds precisely to the central syndrome pattern. The explanations based on an attentional déficit or a visual influence can be excluded for the following reasons: first, the significant concentric reduction of the visual field in Schneider already indicates a disturbance in visual elementary excitability which, as in touch, does not derive from any attention deficit; second, the tactile disturbances encompass the most elementary functions, which are outside the presumed spatial influence; third, the intensity of the disorder in both systems, vision and touch, is the same and equally distributed in both halves of the body (concentric reduction and decrease of elementary sensitivity in the whole body). Therefore, the classification into primary disorders for vision and secondary disorders for touch is arbitrary. The above arguments show the difficulties and theoretical obscurity of the interpretation of the Schneider case by Goldstein and Gelb (1918, 1919).

A strong argument is the finding of dynamic action phenomena in our M and T patients and the characterization of the central syndrome (see Vol. 1), which clear-

ly determines the state of brain excitability and the distribution of the disorder (on both sides of the body and for all sensory systems). It is then possible not only to easily understand the multiple disorders of the Schneider patient but also, by including him in the central syndrome, to predict many others that have not been found in him.

A brief review of a series of cases with brain lesions published at very different times (Oppenheim 1906; Goldstein 1915; Foix 1922; Stauffenberg 1914, 1918; Goldstein and Gelb 1918, 1919), only a few among many others, shows that if the examination of the patients is rather careful, the interpretation of the paradoxical symptoms encountered presents great difficulties within the theory of brain localization. In order to maintain this theory, the authors need to resort to additional hypotheses at the expense of arbitrarily diminishing the value of certain alterations or even denying or disregarding them.

As for astereognosis as an isolated or independent defect, the position of Stein and Weizsäcker (1927) against such autonomy is noteworthy. They rely on experimental facts about lability of the threshold of sensation, abnormal fatigability, etc., all of which refers the defect to a basic disturbance of nervous excitability.

A detailed discussion of each case would take a long time and would lead us to the same results and conclusions that in general terms have already been stated. These are: all these cases show more or less fragmentarily the characteristics of the central syndrome, and the difficulties of interpretation they raise are automatically solved within the brain dynamics exposed. The more complete the examination of all brain functions and, especially, the better the *quantitative* determination of the different excitabilities, the more accurate is the inclusion of a case in the central syndrome.

This last requirement is of great importance in order to reveal all the elements of the central syndrome, especially the alteration of the most elementary functions, thus demonstrating the overall alteration of a given sensory system. Otherwise, since in ordinary examinations attention is usually paid only to the abolition or presence of functions but not to the *degree* of impairment, many functions are easily admitted as intact, particularly the more or less elementary ones, leading to erroneous conclusions about the specificity and localization of certain functions. In addition, there is the masking of symptoms due to facilitation actions, ways of applying stimuli, usual examination techniques, etc., as has been repeatedly indicated throughout the exposition of this research (see Vol. 1), and as will be seen in the next chapters on tactile functions. All this is to point out that, before raising fundamental theoretical questions, careful investigation is required, which is not easy, and it is only with much patience that sufficient data are obtained to understand the characteristics of the disorder. In fact, at the beginning of these investigations, our M and T patients did not seem to be as profoundly affected as the detailed study of them later revealed.

TACTILE SENSATIONS

19. Dynamics of tactile sensations

19.1. GENERAL ASPECTS. HETEROTACTILE INTERVAL

The so-called *fundamental tactile sensations (or modalities)* are *pressure, pain and temperature*, which suffer in our brain-injured patients a dynamic alteration evidenced both by a considerable decrease in excitability and by a peculiar disaggregation due to a functional desynchronization. A certain parallel can now be established with the disorder in colors although the organization of these tactile functions is much simpler. A characteristic of colors and tactile sensations is the reduction of excitability and the noticeable interval between the initial sensation and the more defined sensation, called photochromic and photo-heterochromic interval in colors (see Sec. 7 in Vol. 1), and now heterotactile interval.

According to the results of our study, the three fundamental tactile sensations mentioned above should not be considered as belonging to the same functional level, unlike what is usually accepted. In both vision and touch (and the same is true for hearing), the most primitive sensation can be separated from the more defined sensations. In vision we have as first sensation, simple colorless luminosity, and as a more defined sensation, the different colors. In touch we have as initial or elementary tactile sensation, simple contact (pressure), analogous to simple colorless luminosity, and in another more elaborate range are the tactile sensations of pain and temperature. Since the alteration of any system is global, these two types of sensory activity, initial and more defined sensation, are affected in different ways due to their peculiar excitability characteristics.

The behavior of the initial sensation (contact) in our brain-injured patients is established in the previous sections on general excitability, either by electrical or mechanical stimulation. In addition to the considerable deficit of excitability, there is a spatial disorder that impedes spatial localization of the elementary sensation of contact. Thus, there is a pronounced hypoesthesia, the degree of which depends on the intensity of the cortical lesion, and is partly attenuated by facilitation. The deficit of excitability is clearly defined, so it is inappropriate and arbitrary to attribute the hypoesthesia to at-

tention disorders, as Goldstein and Gelb (1919) have assumed for their Schneider case in order to avoid the serious theoretical obstacle of impairment of sensitivity in both halves of the body when there is only a unilateral lesion (see Sec. 18.3).

Hypoesthesia also affects the sensations of pain and temperature (hot and cold); thus, a pinprick as well as the application of heat or cold, when not very intense, may be felt only as an unclear contact without any trace of spatial localization. To reach the physiological level of well-defined sensations, the stimulation must be more intense and so, whereas a normal subject perceives a tactile modality of remarkable intensity, the patients describe only a slight qualitative sensation. In short, pain and temperature are less perceived due to a reduced sensitivity. Well-defined sensations are accompanied by a certain spatial localization, which is diffuse (irradiated) when the sensation is weak, and more perfectly localized when the sensation is very intense.

The most remarkable aspect of the dynamic reduction is a peculiar decomposition (disaggregation) of tactile sensation, thus appearing the *heterotactile interval*. A sufficiently intense thermal or painful stimulus is chronologically decomposed into two stages: a first stage of simple contact sensation and a second stage of the tactile sensation in question. In this way, we obtain what we call *tango-algic interval* for pain and *tango-thermal interval* for temperature, thus, the fundamental experiment for tactile sensations is similar to the one studied for colors. This asynchrony makes it possible to separate the well-defined sensations of pain and temperature from the primitive tactile sensation of contact. Moreover, they correspond to spatial categories of very different development in our pathological cases, since only the well-defined modality shows a certain localization. In short, with adequate stimulus, a well-defined tactile modality is not established from the outset, but is preceded by a primitive tactile sensation devoid of qualitative differentiation and spatial localization.

This type of asynchrony through the aforementioned heterotactile interval is of great theoretical significance in questioning the physiological individuality of tactile sensations since the interval shows the existence of a primitive component from which algic and thermal sensations evolve and differentiate. Similarly, simple primitive luminosity is the starting point for color sensations. Dynamic reduction, through the asynchrony of functions, is therefore the keystone for approaching the analysis of seemingly simple or individual functions. From tests in our brain-injured patients, it is shown that there are really no specific or independent functions, but rather, the different sensory levels (many of them usually considered irreducible to other elements) appear as the result of a progressive evolution and organization developed from a very primitive element. It is simply conventional to try to identify individualized functions throughout this development. More properly, it is only possible to consider different degrees of differentiation or of sensory organization; not the existence of different individual functions.

19.2. PRESSURE AND PAIN

The data previously shown on the state of general excitability in touch, already suggest that there is a notable hypoesthesia extended to the whole body and on both

halves approximately equally or with minor differences. The intensity of hypoesthesia is related to the magnitude of the brain lesion; thus, it is severe in M and moderate in T. Especially in case M, it is necessary to examine the subject in a fully inactive state in order to uncover the whole magnitude of the defect. It is noteworthy that despite the deficit in tactile sensitivity (in stimulus intensity and timing), these subjects were never spontaneously aware of this disorder nor of the significant lag that leads to a considerable alteration in stimulus localization. This unawareness is due both to the compensatory action of facilitation and to the habit of paying attention only to stimuli that are well perceived, discarding all others, because they are too weak or cause a fragmentary perception.

As already mentioned, the sensation of contact or pressure can be considered a primary activity of the tactile function, like simple luminosity in vision. Whereas for some authors this sensation is completely independent of the other two modalities (pain and temperature), other authors, especially Goldscheider (1917), accept a gradual transition between pressure and pain, in such a way these sensations are extreme modalities of the same function. Therefore, Goldscheider (1917, 1925) admits that a touch on the skin can provide either pressure or pain depending on the intensity of the stimulation. This is equivalent to deny the existence of different specific points for pressure and for pain as Frey maintains (Frey 1910, 1913, 196/1917, 1928). Goldscheider (1917, 1925) admits the existence of certain points where weak stimulation produces only pressure, and more intense stimulation causes pain; and in analogy with the photochromic interval of colors, he mentions a tango-algic interval. This interval, originated by the duality of the response according to the stimulation energy, seems to be present even in regions where only pain points are usually admitted (e.g., the cornea). Thus, threshold excitations cause only contact sensation according to Kiesow (1924). Likewise, Hoefer and Kohlrausch (1924) report that under threshold electrical excitation at pain points, they always obtain an initial sensation of contact. It has also been observed that in sensory adaptation to pain, when pain disappears, only a sensation of pressure or simple contact remains.

In our cases, mainly in the inactive subject M, it is repeatedly proven that stimuli that are clearly painful for a normal subject are only perceived as contact, and when a more intense stimulus triggers pain, the sensation goes through a previous phase of contact. The tango-algic interval is extremely marked and when a skin site is stimulated, the sensory interval that appears is so large that it is very easy to observe it. A prick of a certain intensity with a fine needle, if it is brief, only gives rise to a perception of contact, and if the time of application is prolonged, the pain phase is reached. With facilitation by muscular effort, the interval also exists but it is not so long. Analogously in subject T.

Cutaneous chronaxie determinations in a normal subject already show different values for pressure and for pain. Bourguignon (1929, 1933) finds that the chronaxie for the so-called "superficial light pain" is twice that for pressure (or pushing); and for "deep blunt pain" it is up to four times that for pressure. By determining with electrical stimulation the curves for pressure and pain in the two extreme states of subject M and subject T, as well as in the normal comparison subject, it is found that the greater the deficit of excitability, the greater the tango-algic interval. The tests are

performed, as in general excitability, on the middle of the lower lip. Since a 0.5 cm diameter electrode is used, the stimulation is on surface and not on selected contact points, but this does not prevent distinguishing between pressure and pain sensations in relation to the stimulation energy.

The strength-duration curves for pressure and pain sensations in Fig. 19.1 show the large difference between subject M in the inactive state and the normal subject. The curves of M are markedly higher, corresponding to a very large rheobase increase, they also have less curvature than in the normal case due to the significant increase in chronaxie, and they are also much farther apart between them than in the normal subject, which means a considerable enlargement of the tango-algic interval. In electrical stimulation, the sensation of pressure corresponds to a sensation of very slight shock due to the passage of current when the capacitors are discharged. The sensation of pain corresponds to a very slight and superficial pain but clearly distinguishable from the previous sensation.

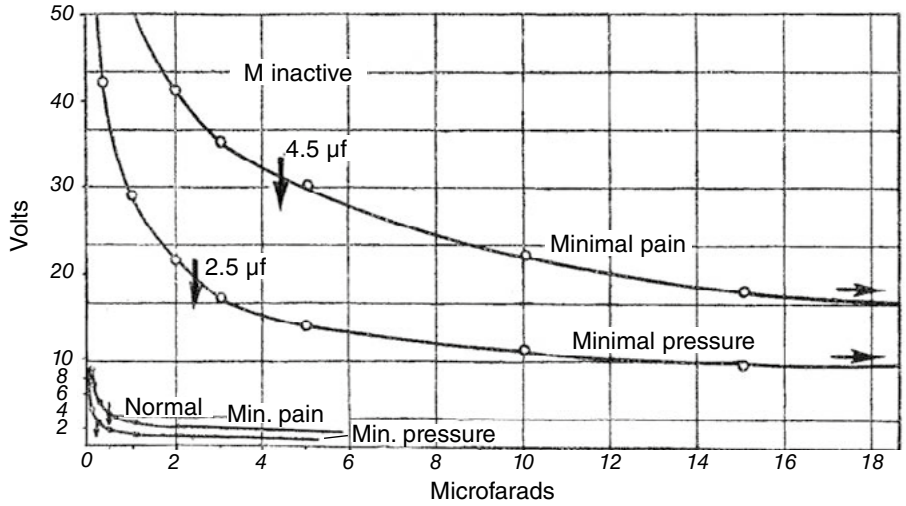

Figure 19.1. Strength-duration threshold curves for pressure and pain in the inactive subject M (upper curves) and for the normal subject (lower curves). Electrical stimulation on the midline of the lower lip. Cathode 5 mm in diameter. Note the large elevation of the curves in subject M and the large separation between the two curves compared to the normal subject. Note also the different chronoaxie values.

In these tests, the chronaxie capacitances in the normal subject for pressure and pain are 0.25 μf and 0.4 μf respectively. In subject M in the inactive state, the respective values are 2.5 μf and 4.5 μf. Thus, the two values in M inactive are about ten times greater than in the normal subject. Hence, the relative pressure-pain ratio remains unchanged in the pathological case, i.e., in both M and normal, the chronaxie for pain is almost twice the chronaxie for pressure. As for the absolute values, the difference is very large, since in the normal subject the variation in chronaxie is of

tenths whereas in M inactive it is of several units. In short, there is pathologically an extremely wide tango-algic interval, which explains that in the stimulation of a same cutaneous location, pressure and pain are obtained with great separation of time (μf) or intensity (volts), the latter due to the fact that the rheobases suffer an increase parallel to the chronaxies both in electrical and mechanical excitation.

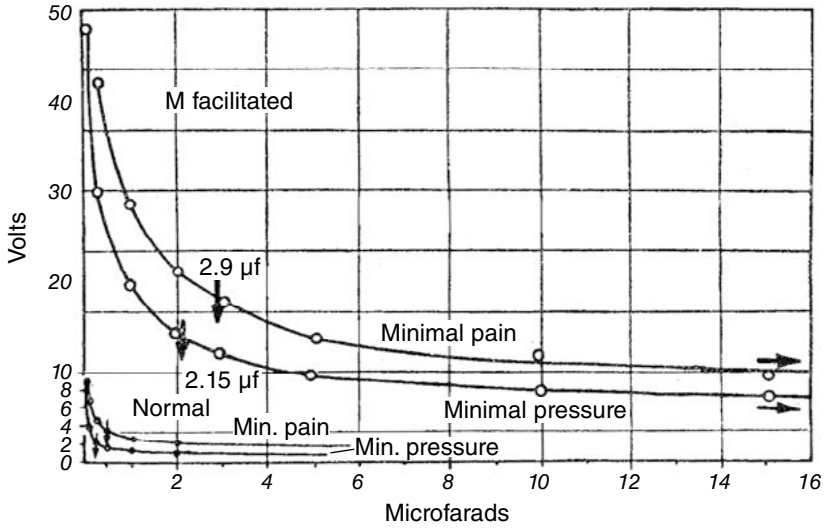

Figure 19.2. Strength-duration threshold curves for pressure and pain for subject M under facilitation (maximum contraction of the entire musculature), and for a normal subject. Stimulation conditions as in Fig. 19.1.

For subject M under facilitation by strong muscular effort, the above considerations are valid. This case corresponds to an intermediate physiological level between the inactive state and the normal subject, therefore the tango-algic interval now has an intermediate amplitude (see Fig. 19.2). The pressure and pain curves are lower than in the inactive state and are also closer together, but the interval still has a considerable amplitude.

Finally, in subject T the interval is narrower as shown by the closer proximity of both curves (Fig. 19.3), which correspond to much faster chronaxies than in the two mentioned states of M.

In these cases, the amplitude of the interval is closely related to the level of excitability, i.e., to the characteristics of the pressure and pain curves. The ratio between the values for pressure and pain remains approximately constant in all cases, including the normal subject. However, the absolute value of the differences increases as the deficit of excitability is greater. Table 19.1 shows the values corresponding to Figs. 19.1, 19.2 and 19.3.

One of the most remarkable features accompanying the sensation of pressure and pain in our subjects is, as stated, the alteration of spatial localization. Pressure

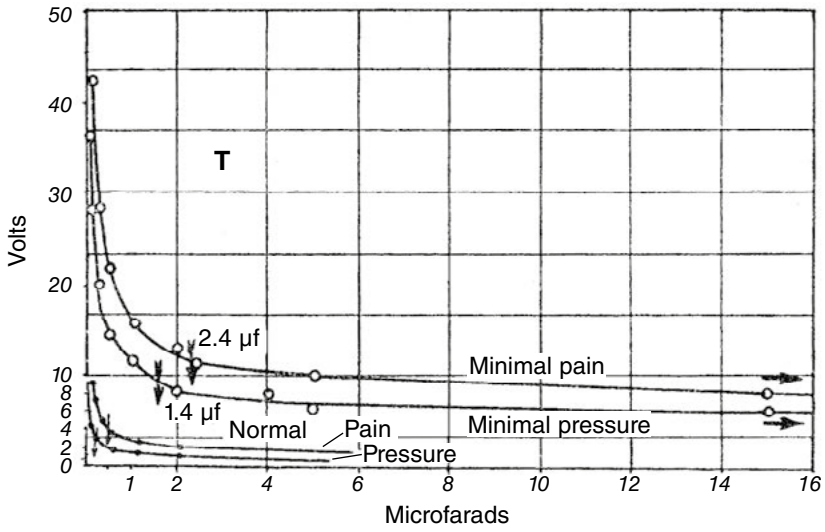

Figure 19.3. Strength-duration curves for pressure and pain in subject T and in a normal subject. Same conditions as in Figs. 19.1. Note the differences with the two aforementioned cases.

Table 19.1. Values corresponding to Figs. 19.1, 19.2 and 19.3.

	Pressure		**Pain**	
	Chronaxie (µf)	Rheobase (volts)	Chronaxie (µf)	Rheobase (volts)
M inactive	2.5	9.5	4.5	16
M under facilitation	2.1	6.5	2.9	9
T	1.4	4.5	2.4	5.6
Normal subject	0.25	1.2	0.4	1.6

or simple contact does not cause any sensation of localization, and a pinpoint pressure causes a kind of cutaneous irradiation of indeterminate localization on the surface of the skin. Rudiments of spatial localization appear only with pain and thermal sensations, and in the case of pain, as soon as the sensation of pain begins, even if it is very slight, a certain localization appears. But this one is quite deviated from the stimulus application site; in addition, the mentioned irradiance gradually decreases, although it is still prominent. The more intense the pain (e.g., in a pinprick), the more normal the spatial structure of the perceived stimulus. In this way both deviation and irradiation gradually decrease according to certain laws. A pinprick causing almost unbearable pain is necessary for the localization to become completely normal, at least in subject M inactive, who presents maximum functional lag (asynchrony). This question will be studied in a separate chapter, and now it suffices to mention

the general characteristics of pressure and pain sensations. Therefore, in addition to the huge enlargement of the tango-algic interval, there is a sensory-spatial interval due to the lag or asynchrony in the nervous centers involved in the localization mechanism. This last interval does not exist in the normal subject since any tactile stimulus, no matter how weak or brief, is always perfectly localized. This fact is completely new, without any precedent in functional normality.

We must mention the special type of pain admitted by Piéron (1935) consisting of a stinging sensation (piqûre in French). Indeed, it is a tactile sensation close to pain but extremely spatially circumscribed, producing a very slight pain of pinpoint character. Due to the considerable spatial irradiation in our pathological cases, such a stinging sensation is completely abolished since the sensation of minimal or incipient pain occurs in a very spatially diffuse manner. This defect is detected in the clinical test of distinguishing between pinhead contact and pinpoint contact. This distinction based on sting-like perception is impossible in subject M inactive, within rather wide limits, even if a certain degree of pain occurs with the corresponding stimulus. The sting is thus essentially a sensory complex of mild pain according to a certain spatial characteristic, and there is no reason to consider it an individualized function within the sensation of pain. More details on this issue are given in the chapter on spatial localization.

As for the excitation conditions for pressure and pain, it should be noted that strong pain is very easily obtained by iterative stimulation of a mere pressure sensation. In this way, not only the wide sensory gap of the tango-algic interval disappears, but an intense, almost unbearable pain sensation is reached, which is very close to the normal localization. Already in a normal subject, the sensation of pain shows some aptitude to iterative stimulation (Schriever and Cebulla 1938, Altenburger 1933, Piéron 1935) but in our cases, particularly in M, it is greatly increased. In relation to this behavior are the old observations of Egger (1899), who points out that no tabetic analgesia withstands stimulation by means of a needle fixed to a tuning fork providing 60 pulses per second. Pain is either triggered or recruited by summation in time (latent addition), as in our brain-injured patients.

Analogous to iterative summation, although less effective, is facilitation by muscular effort. Thus, a stimulus at the pressure threshold is transformed into a sensation of pain by means of a vigorous muscular effort, overcoming the tango-algic interval and sometimes reaching a sensation of accentuated pain if the effort is very intense.

Finally, as for sensory adaptation to pain in subject M, this is evidenced within the limits already indicated when discussing tactile adaptation in general. With stimulus applied continuously with just enough intensity to produce pain, the pain ceases after half a minute, and only the sensation of pressure remains, which in turn disappears after about half a minute or a little less. This adaptation that excludes algic sensation, occurs very easily, probably due to the increased fatigability in these pathological cases. During adaptation, the tango-algic interval occurs in the opposite direction.

From all that has been said so far, it follows that pressure and pain are closely related physiologically, giving rise to the idea that pain is a differentiated modality derived from pressure.

19.3. THERMAL SENSATIONS

Thermal sensations show in our cases a full parallelism with pressure-pain sensation, namely, a deficit of excitability with required increase in intensity and time of stimulation, and also a tango-thermal interval. The deficit is related to the magnitude of the brain lesion and the conditions of the central nervous system by facilitation.

When touching a cold or hot object, only contact is felt, without any thermal sensation when the temperature of the object is not excessively far from the neutral point of thermal sensation. This occurs in both subject M and subject T, although with the difference due to the different level of excitability in each subject. If the temperature of the touched object is very different from the body temperature, thermal sensation finally occurs but it is clearly observed that in the slow sensory development, the thermal sensation is preceded by one of simple contact or pressure. This shows the existence of the tango-thermal interval, which does not seem to exist in a normal subject. In our cases it is clearly obtained by both the difference in intensity thresholds (higher for temperature than for pressure) and the different speed of reaction (slower for temperature). The heterotactile interval is thus common to pain and temperature, both sensations developing from the primitive pressure sensation.

There is no agreement among authors on how to obtain thermal sensation by electrical stimulation. Some, like Schriever (1929), consider to have achieved it for cold. Bourguignon (1929, 1933) as well as Schriever (1929) found that the chronaxie for temperature is eight to ten times the chronaxie for pressure, and about four times the chronaxie for surface pain. Thus, thermal sensation is by far the tactile sensation with the highest chronaxie. We shall now limit ourselves to stimulation with thermal, not electrical, stimulus. The tests are performed with water at different temperatures in a test tube, applying its base on a small area of the skin. Other ways of stimulation, water drops, radiant heat, etc., serve to complete the study. In this way, it is not difficult to get an idea of the sensitivity to temperature in its heat and cold modes, and intensity-duration[1] curves showing the state of the function can be determined.

Figures 19.4 and 19.5 show the results obtained in subject M inactive. A same thermal stimulus gives rise to two different sensations corresponding to respective curves of different excitability. The higher excitability is for pressure sensation (simple contact), the lower excitability is for thermal sensation, either warm or cold. This duplicity with the same stimulus shows the existence of the tango-thermal interval. In addition, the alteration of excitability for cold and for warm are of the same quantitative order, and the range of the pressure-temperature interval is about 5° Celsius. With facilitation (by strong muscular effort), the interval is much smaller, only 1 °C or 2 °C between contact sensation and warm sensation. The interval is also found in subject T, although much smaller than in M under facilitation. Therefore, the behavior is similar to that indicated for the tango-algic interval in the cases considered.

[1] Since the stimulation is not electrical, the term intensity-duration will be used instead of strength-duration.

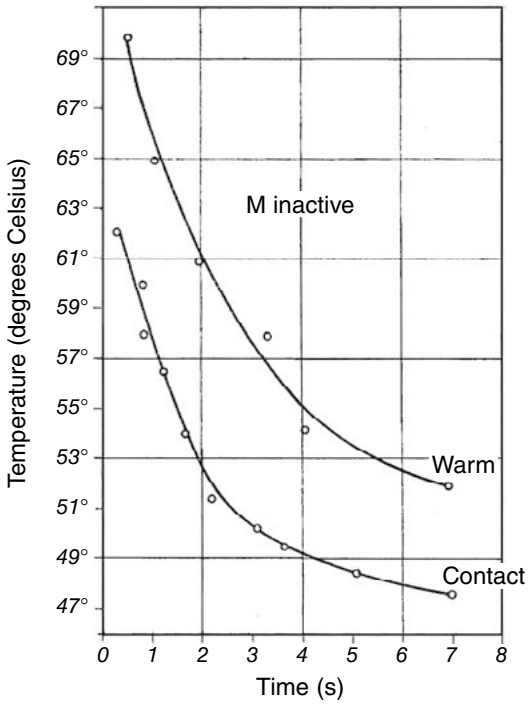

Figure 19.4. Intensity-duration (temperature-duration) threshold curves for contact sensation and warm sensation in subject M inactive. Note the tango-thermal interval. Stimulation by applying a test tube with hot water over the midline of the forehead, over a surface of approximately one square centimeter and avoiding pressure. The curves correspond to the average values of a series of tests.

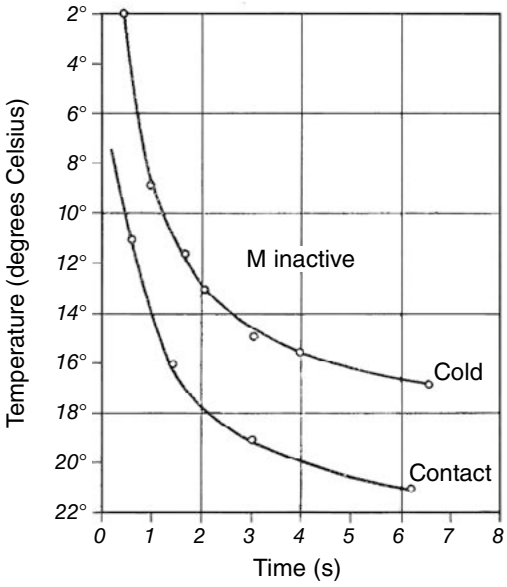

Figure 19.5. Temperature-duration threshold curves for contact sensation and cold sensation in subject M inactive. Note the tango-thermal interval. Stimulation conditions as in Figure 19.4.

In all these cases, when the intensity of the stimulus is not high enough, the thermal sensation is not produced, only the first contact sensation is obtained, that is, the aforementioned interval is not overcome. Moreover, if the thermal intensity is even lower, although the wall of the test tube is resting on the forehead of the subject, not even the sensation of contact is obtained. Thus, the thermal stimulus is what determines the sensation of contact, not the pressure (contact) of the test tube, which only serves (especially in inactive M) to transmit heat. In order to verify this remarkable finding, we proceeded to perform further tests in the absence of any possible pressure stimulus. For this purpose, the test using drops of water at a certain temperature on the skin is easy to perform. The drops, due to their very low weight, do not act as a pressure stimulus in these subjects. In this way, the tango-thermal interval can be obtained for a single drop at a certain temperature, since, as we know, the development of sensation is very slow in subject M. If the temperature of the drop is reduced, only contact sensation is obtained, and below that temperature it is no longer possible to achieve any pressure sensation even if drop after drop is deposited on the skin.

Another even more conclusive test involves the use of thermal radiation. By directing radiant heat from an incandescent electrical resistor onto a certain surface of the skin, it is possible to determine a distance at which the thermal flux on the skin is felt as a contact without any heat. It is clear that lacking all mechanical contact, the sensation of contact (pressure) must be attributed to the sensory degradation suffered by the pure thermal sensation because of the heterotactile interval.

In addition to the important phenomenon described on the tango-thermal interval, there is a characteristic disorder of temperature perception which is the considerable enlargement of the neutral temperature range (intermediate between cold and warm) to which the surface of the human body is adapted and which does not give rise to thermal sensations. In the normal subject, this range is about two degrees, and in the most usual conditions it ranges from 32 °C to 34 °C. Enlargement of the neutral range was first reported by Head and Riddoch (1920) in tactile disorders due to parietal cortical lesion. Head found decreased thermal differential sensitivity, i.e., increased thermal differential threshold, and enlargement of the neutral range as a consequence of also increased absolute threshold for cold and heat. The authors give as a general explanation a deficit of discrimination, without mentioning at all nervous excitability.

Figure 19.6 shows the variations undergone by the neutral range in the various cases considered. It can be seen that the widening of the range is related to the deficit in nervous excitability. The thresholds for cold and heat increase, and also their useful time, as we know. Whereas the neutral range in the normal subject is about 2 °C, in M inactive, the most extreme case, the range reaches 36 °C, i.e., 18 times higher.

It is also observed that the alteration is the same for cold and heat, the corresponding curves being symmetrically located with respect to the central point of the neutral range (33 °C). A comparison of the different threshold values shows that the threshold increases considerably in subject M, both when inactive and under facilitation, but especially in the former state. This is consistent with the profound deficit suffered by a function much less sensitive than pressure or pain. In subject M (of greater brain deficit) the disorder in temperature sensation is very pronounced, since

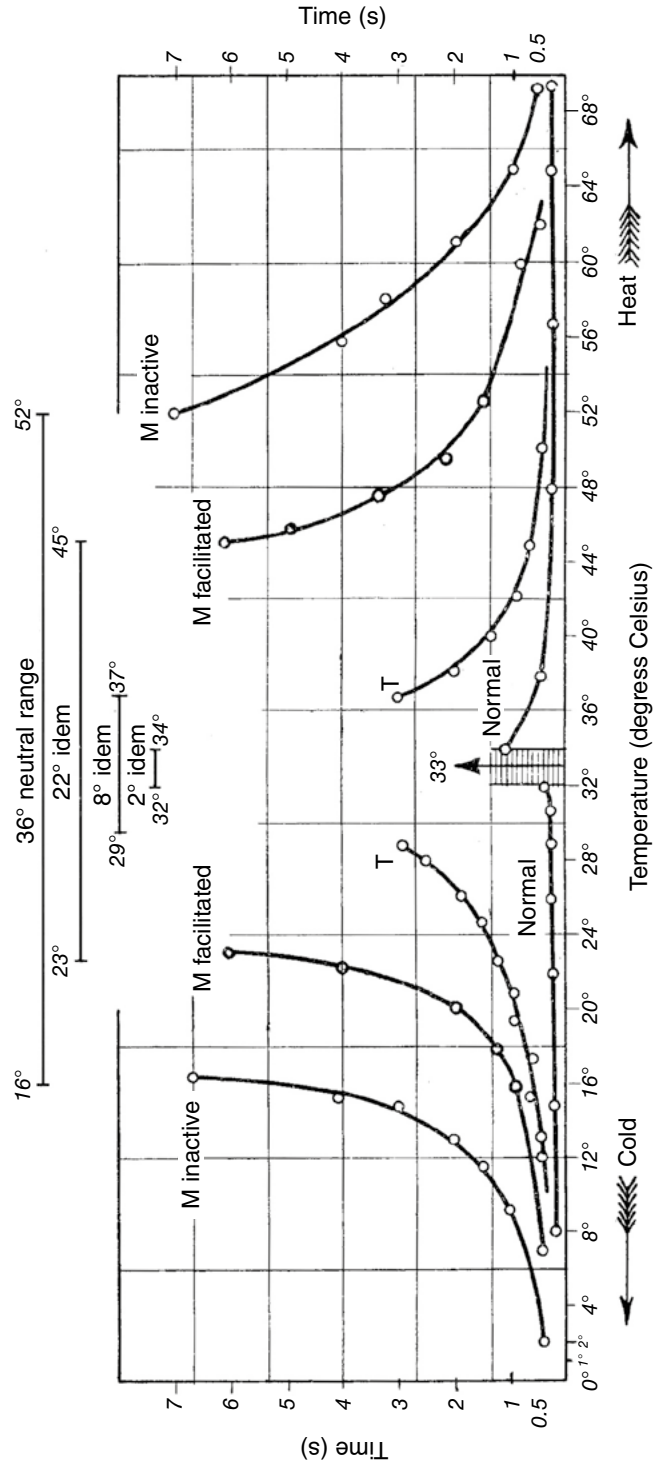

Figure 19.6. Arrangement of the intensity-duration (temperature-duration) threshold curves for cold and heat allowing us to observe the enlargement of the neutral range as a function of the deficit of excitability, related to the magnitude of the lesion. Curves for M inactive, M under facilitation by strong muscular effort, T inactive and normal subject [data for the latter according to Piéron (1935)]. Stimulation on the forehead with a thermal surface of one square centimeter.

functions suffer greater disturbance as they are less excitable. Thus, within the tactile sensations, those of temperature show to be the most intensely altered.

The values indicated by the curves correspond to laboratory experiences somewhat distant from ordinary life conditions. For a normal subject, a temperature of 50 °C applied to a very small surface of the skin is perceived as heat after about 1/3 s, whereas in subject M inactive, it must be about 52 °C to feel heat after a few seconds. After this period of time, the normal subject would feel an acute sensation of strong heat, while M is at the beginning of the thermal sensation, i.e., at the sensation threshold. Thus, it must be concluded that there is an acute thermal hypoesthesia in M. However, in the conditions of ordinary life when touching objects, this does not occur so pronouncedly since the contact usually takes place on larger surfaces, and the thermal deficit is corrected significantly by the summative action of the surface. In the experiment with the test tube filled with hot water, if instead of applying the bottom of the tube on the forehead or on a cheek on a surface of one square centimeter or less, the tube is applied laterally over its entire length, the values obtained in the inactive state become equal or even lower than those of the curve in the facilitated state when the surface is one square centimeter. In ordinary life, the thermal defect is greatly reduced, both by the more or less intense muscular effort that the subject exerts spontaneously and which acts as facilitation, and by the action of the surface, thus achieving two types of summation that significantly compensate for the defect.

The spatial properties of thermal sensations are analogous to those discussed in the case of pain. In the tango-thermal interval, the first sensation of simple contact is not accompanied by spatial localization and only a fleeting sensation more or less extensive is perceived in some part of the body impossible to specify. But as soon as the second phase is reached, thermal sensations become localized and irradiation tends to decrease. As in the case of pain, very intense thermal sensations are required for localization to be normal, i.e., without irradiation and without deviation with respect to the stimulation point, as we shall study later on. In short, only pain and temperature have a spatial character, which means localization of the corresponding stimulus, and only at high intensities localization becomes close to normal.

Thermal adaptation and differential threshold have not been studied systematically enough to obtain usable data. In addition, a number of circumstances make this type of testing difficult. Nevertheless, it can be said that thermal differential sensitivity in M inactive is severely impaired. The intensity threshold for the sensation of heat and cold is markedly increased, as we have seen, but so is the differential threshold. Whereas the normal subject can appreciate differences of fractions of a degree between certain temperatures, subject M needs up to 2 °C or 3 °C in the most favorable conditions (certain thermal ranges in which differential sensitivity tends to be better). As for thermal adaptation, this occurs more easily than in the normal subject due to the enlargement of the neutral range, and may also be due to increased sensory fatigability. More complete experiences would be needed to determine more precisely these phenomena.

20. Organization of tactile sensations

20.1. CRITICISM OF THE SO-CALLED "TACTILE DISSOCIATIONS"

Neuroanatomical concepts often find an important theoretical and practical application in the tactile system. Anatomical specificity consists in admitting certain receptors with a well-determined function (tactile points), as well as different conduction pathways and terminal centers also linked to specific tactile activities. This anatomical independence of the different functions should be particularly evident in the multiple "clinical dissociations" of the tactile functions according to the type of anatomical lesion. Therefore, tactile dissociations are considered very important in clinical diagnosis.

This view certainly responds to clinical needs, but it should not be set up as a systematic theory or general law. It constitutes only an acceptable approximation for certain particular cases, whereas it fails completely in the interpretation of many others. A revision of this approach began with the studies of Head and Riddoch (1920) and Head *et al.* (1920), who reduces all tactile activity to only two types of sensitivity, namely, primitive or protopathic and more differentiated or epicritic. But, apart from this, the anatomical aspect remains quite firm in this new theory since protopathic and epicritic pathways and centers are assumed. The organization of functions is simplified, but the hypothesis of anatomical specificity is preserved for the two types of sensitivity.

Later on, Foerster (1936), an author closely linked to the theory of localization and anatomical specificities, was compelled, in order to interpret many clinical observations, to accept a *functional principle* in addition to the principle of localization. Thus, it can be said that when the tactile system is affected, a constant regularity is shown in the way its different functions are altered, and this occurs independently of the anatomical type of lesion. The sensory modality that is always the most affected is temperature followed by pain and finally pressure. When a lesion significantly impairs tactile activity, and after a certain time functional recovery occurs, the following order is always observed in the appearance of sensations: pressure, pain

and finally temperature. However, Foerster (1936), a consistent localist, interprets this functional order of affection by resorting either to diffuse biological hypotheses on a greater robustness of the most basic functions, or by means of the anatomical argument of a supposed greater spread of the anatomical elements corresponding to the best preserved functions. This argumentation in order to maintain the approach of anatomical specificity is not very resistant to criticism, but it is not appropriate to address this issue here.

Finally, we find a new approach in Stein and Weizsäcker (1927, 1928) much more explicit, coherent, and rational than the previous ones. These authors consider the tactile system as a functional unit that has a number of activities, not as much because it contains different sensory elements, as because it is activated in many different ways. Such considerations lead them to refute in depth the dissociated alterations of sensitivity, as well as the usual teaching of clinical neurology on sensitivity as set forth in classic treatises such as that of Dejerine and Dejerine-Klumpke (1914) among others. For them, it is absurd to seek for each new function a special organ, i.e., a specific pathway or a new anatomical-physiological element. Due to the special *change of function* that occurs in lesions of the tactile system, it results that while the sensitivity is suitable for a certain function, it remains unable for others. It is therefore a futile effort to try to infer from these changes (dissociations) conclusions about the autonomy of receptors and diverse specific pathways. In short, these authors establish that when there is a certain disorder of the tactile system, its excitability is modified, and the changes in the response do not correspond to anatomical dissociations of specific elements or neuronal regions for a certain function, but are due to physiological disorders of the whole tactile system, which by altering its excitability, disturbs the capability of functional differentiation.

These theories of the aforementioned authors show a growing tendency to replace the old and classical tactile dissociations (according to multiple anatomical specificities) by more functional or physiological conceptions that tend to simplify the problem, either by reducing the number of sensitivities (Head and Riddoch 1920, Head *et al.* 1920) or by establishing a constant sequence of alterations (Foerster 1936), and finally, by focusing the problem on the excitability process of a functionally unitary tactile system (Stein and Weizsäcker 1927, 1928). This latter approach is thus the most radically opposed to the classical theory of anatomical dissociations. For Head et *al*, sensitivity is either primitive and weakly differentiated (protopathic), or more discriminative and evolved (epicritic). For Foerster, there is also a sequence in the evolution of the alteration, and finally, for Stein and Weizsäcker, the principle of functional alteration (type of differentiation, sequence in the evolution, etc.) is made to depend simply on the excitability disorder of the tactile system, without resorting to further anatomical considerations as is still the case for the previous authors.

Taking into account that in a normal subject there are already very different chronaxies for the different sensations, Foerster's functional order of alteration is easily explained. Thus, the less excitable functions (of higher chronaxie) are more easily excluded. This does not mean independent alteration of some functions with respect to others since the alteration of excitability is always global. It is therefore

not possible to obtain absolute dissociations, but only relative ones. Some functions suffer more than others, but all of them are affected to some degree, as shown in the cases studied here. The alleged absolute dissociations are then more apparent than real, and they result from a purely qualitative examination. By making quantitative measurements, as in our cases, and determining the level of excitability for all types of activities, the global character of the disorder is demonstrated, although diversely manifested in the different functions, thus giving rise to the dynamic reduction that we already know. The issue of tactile dissociations and, in general, of all types of functional dissociations, acquires a meaning quite different from that usually accepted.

20.2. ORGANIZATION OF TACTILE SENSATIONS

The dynamic reduction in tactile sensitivity presents several aspects in our two patients that should be remembered. Vibratory sensitivity is greatly reduced due to a great loss of reaction speed. Pressure, pain and temperature show a remarkable deficit, more pronounced in pain and especially in temperature. In addition, the dynamic disorder shows very special characteristics due to functional asynchrony, giving rise to tango-algic and tango-thermal intervals, which are clearly evidenced. To this must be also added the different type of spatial localization that accompanies each phase of these intervals. As for temperature, it should not go unnoticed that the considerable enlargement of the neutral range takes place at the expense of an equal deficit for heat and cold. All these phenomena lead us not only to a negative criticism of tactile dissociations, as already mentioned, but also, on the positive side, to establish a new concept about tactile organization, mainly concerning tactile sensations.

We must consider the system as a functional unit which, according to the various modalities of the central excitation process, gives rise to the different activities. As for the modes of tactile sensitivity, an important issue about physiological organization arises with the heterotactile interval. As already mentioned, pressure on one side, and pain and temperature on the other, are shown to belong to very different sensory levels. Not only do they have different chronaxies, but also phenomenic features with very different evolution, which is of great importance for classifying these tactile modalities and getting an idea of their physiological organization. These distinguishing features are the heterotactile interval and spatial localization.

Pressure is the specific elementary activity (tangibility) in the sense of touch, like luminosity in vision and sonority in hearing, but not as a modality per se. The meaning of modality should be reserved for pain and temperature. This is analogous to the difference between luminosity and colors, since these also show in our cases a physiological organization quite different from luminosity, such as the photochromic interval and spatial phases.

To determine the meaning of these two well-defined sensations (pain and temperature), one must examine: specificity, functional nexus with the elementary sensitivity and origin of these sensations; all aspects of a same problem. If in order to accept the specificity of a given function one adopts the usual principle of physiolog-

ical individuality, i.e., the non-dissociable character of the function, one must then pronounce against the specificity of the algic and thermal tactile sensations, due to the aforementioned heterotactile interval. Particularly in the case of temperature, it is unquestionable that a pure thermal stimulus, i.e., devoid of any mechanical pressure (e.g. thermal radiation), can be reduced to a simple sensation of contact under appropriate conditions.

This non-individuality of the well-defined sensations results in the acceptance of a functional nexus between the elementary activity of simple pressure and the well-defined sensation that arise through a process of differentiation from the primitive function. That nexus leads to examine the possible origin of pain and temperature. Since certain functions can be "isolated," they can be assigned at least a relative specificity, since it could not be absolute as stated above. This relativity allows us to suppose some evolutionary process by which the sensations we are concerned with arise. If this were not the case, we would have to accept them as irreducible and primary manifestations, as undoubtedly is luminosity in vision and tangibility (simple pressure) in touch. But a process of progressive organization always adds something new, and the well-defined sensations would participate simultaneously of primitive function and evolutionary function, hence a relative specificity is assigned to them. This type of specificity seems to be different for pain and for temperature. In fact, the gradual transition from pressure (primary manifestation) to pain (secondary manifestation) seems phenomenologically very clear and natural, as it occurs in the gray scale or in the transition from weak light to bright light. However, it is very difficult for temperature to find such a type of transition. Therefore, thermal specificity seems much more evident than pain specificity. Moreover, thermal specificity is not entirely homogeneous since it includes cold and heat, admitted by many authors as independent sensations. In this respect, it is observed in our cases that both thermal modalities undergo an identical alteration (producing an enlargement of the neutral region by an equal deficit for cold and heat) which leads to incline the opinion towards the unity of the thermal modality and not its duality, as believed by some authors (Hahn 1928, Goldscheider 1886) who indicate that duality is only an appearance.

As for the origin of thermal sensation, Bourguignon (1929, 1933), in a study on tactile chronaxies, makes the bold hypothesis of supposing that thermal sensation is the result of a combination of the sensation of tingling and of pressure, or in any case, as a differentiating effect from both. If this were admitted, a rather gradual transition between primordial tactile sensation and thermal sensation would result, but at present there is insufficient basis for this hypothesis, and any relationship between primordial sensation and well-defined modality is limited to the phases of the tango-thermal interval.

Concerning the so-called 'fine touch' as another modality whose alteration is easier and of longer duration, it should be noted that the persistent deficit of sensitivity to light pressure and to fine contact discrimination only means an increase in absolute and differential thresholds, which corresponds to the basic excitability disorder (increased rheobase). Likewise, the deficit in the appreciation of very small time intervals (increased chronaxia and therefore refractory period) leads to the in-

ability to perceive high frequency vibrations, even in cases of mild brain lesion. There is thus no reason to think that fine touch deserves a special place as a differentiated function.

All these considerations clearly show the new dynamic approach, applicable to any type of function and valid for any sensory system.

With respect to the sensory specificity of the peripheral receptors (whose current theory is totally opposed to the dynamic considerations maintained here), such specificity would be rather a preliminary to the dynamic action of the centers. This applies to colors as well as to tactile sensations and the rest of the more complex sensory functions.

As a general conclusion, it could be said that instead of having an anatomical-physiological individuality for the various functions (giving rise to well-defined absolute dissociations depending on the nature of the lesion), there is a global disorder and neither functional individuality nor isolated disorders can be considered. Dissociations, if they exist, would in any case be relative. The functions arise according to the degree of organization of the sensory field determined by the physiological level of excitability. The development of this organization makes it possible to establish different physiological levels. Therefore, the tactile functions we have discussed can be dynamically classified into two levels: at an elementary level we have tangibility (pressure) as an elementary function, and at a higher level, the sensations of pain and temperature.

Tactile space

21. Tactile localization

21.1. SPATIAL DISORDER

So far, knowledge about spatial localization disorder is scarce, both in its manifestations and in the physiological mechanism of the pathological process. In certain lesions of the parietal cortex (tactile projection area), the usual clinical observation comes to establish that, for example, the patient localizes the stimulus with error or deviation, that he mistakes the place of stimulus application, that he is unable to pay attention, etc. In more severe cases, and also rarer, it has been observed that the patient is barely able to localize the stimuli, no matter how intense they may be. He knows that he is touched somewhere on his body, but is totally unable to indicate where. This last type of deficiency corresponds to an extreme degree of impairment. In moderate disorders, some irregularity in localization is found, as well as a tendency to irradiation under a point stimulus, indicated by Head and Riddoch (1920), Head *et al.* (1920) and later by Stein (1928, 1930) and Stein and Weizsäcker (1926). A point stimulus is localized somewhat uncertainly, and it is perceived as a sensation in a wider region, not as a point. More systematic and precise determinations are lacking.

In a good number of wounded patients with parietal lobe lesions and diverse tactile symptomatology, studied with some accuracy in 1938, we have been able to establish a certain sequence in the localization of stimuli in relation to the severity of the disorder. This sequence, from lesser to greater severity, is as follows. First, the patient makes more errors when pointing at the location than when mentioning it verbally, i.e., naming is better than pointing because pointing forces to be more precise. Second, stimuli are localized more towards the center of the body than they really are ('proximal deviation'), thus, in the extremities, the localization is perceived more towards their origin, with an evident deviation of a few centimeters. An opposite result, i.e., a distal deviation, is very rarely found, and therefore proximal deviation can be accepted as a rule. Third, the patient is only able to vaguely point at the place where the stimulus has been felt, merely indicating with one hand a

region of the body, but keeping the hand in the air without deciding to specify a place (localization in the air). Thus, some patients indicate large areas of their body without giving further details. Similar to this state but much more disturbed is the fourth stage in which there is a complete loss of localization of tactile stimuli and the subject says "I am pricked, but I do not know where." We have observed this type of response (besides in the two cases studied) only in brain-injured patients in the acute phase, when even very intense stimuli are not able to change the situation. But this complete absence of localization is not usually maintained for a long period, and in patients who survive, after a few days there is a tendency for some rudiment of spatial localization to reappear, especially with very intense or repeated stimuli.

These four stages show that localization does not disappear as a whole; on the contrary, depending on the severity of the disorder, different phases appear which are of great importance for unraveling the mechanism of spatial localization. To the above specifications must still be added the frequent tendency to irradiation under pinpoint stimuli, since the stimulus with the tip of a pin is felt as a tingling of some size, sometimes covering a large part of a limb, a fact already first pointed out by Head and Riddoch (1920) and Head *et al.* (1920). We were also able to observe various phenomena qualified by some authors as attention deficit, ['Beachtungsmangel' in German (Kleist 1934)] or lability, fatigue, etc., which further complicate the results when the examination lasts a long time. It should also be noted that the different brain-injured patients we examined presented a global disorder of tactile sensitivity. Spatial functions were more affected than simple sensations, and within the spatial functions, localization was much less impaired than discrimination between two points (Weber), passive movement and posture, the latter two being maximally affected in all cases. To reach a considerable disorder of localization (third or fourth stage), a very deep impairment of the tactile system is necessary. All these data do not go beyond the empirical clinic, however interesting they may be, since a systematic study and particularly a physiological investigation are lacking. These last two aspects are fulfilled in the brain dynamics developed here on the two cases we are dealing with. The significance of the mentioned stages will be discussed later, and now they are only a preliminary to show the existence of gradations in localization disorder.

These gradations acquire importance when considering the only existing case (apart from those presented here) with lasting abolition of tactile localization, i.e., the Schneider case of Goldstein and Gelb (1919). Many years after being injured, this subject showed a complete abolition of localization, as he only felt contact without any possible indication of localization, unless certain movements or involuntary muscular jerks or twitching movements intervened, in which case localization was perfect, according to the authors. Despite the detailed study of the phenomenology of the patient, no intermediate forms are described, which in our opinion must necessarily exist, as will be seen in our cases. Even more than in the work on vision, in the second study on touch, the authors focus exclusively on highlighting the great difference when twitching movements are absent compared to when they are present. These are two extreme states corresponding respectively to a total abolition of spatial functions and to a complete normalization of them (only seemingly according

to the authors); and no gradual transition between these two extremes is reported. The change is abrupt and does not allow us to unravel the phenomenology, much less the development of the localization and spatial function in general. This issue (presence of stages with and without muscular action, the latter being only a facilitation), and the fundamental importance of the development of localization and other spatial activities, will be discussed again throughout this chapter.

The general thesis of the two studies by Goldstein and Gelb (1918, 1919) on the Schneider case has been exposed in the first part of this work, as well as the experimental rebuttal of their interpretations together with the rational physiological interpretation of this patient. Although it is not the main purpose of this work to criticize the Schneider case, given his enigmatic symptomatology and antecedents in relation to our cases, it is very necessary to know his characteristics, both for the necessary remarks we shall make and to highlight the essential differences in the method of study and in the results obtained.

As already indicated, patient Schneider also presents some impairment of elementary tactile sensations, although the authors try to play it down and relate it to an attention disorder. Hence, in all the transcriptions of this case, it is said that tactile sensations were completely intact, which is not the case as stated above. According to the authors, kinesthetic perceptions (muscle, tendon and joint perceptions) show a behavior similar to that of the other tactile sensations. In other words, without resorting to any facilitation (muscular contractions, etc.), a passive movement of a joint is clearly perceived, and it can even be appreciated whether it is fast or slow. This statement entails several important contradictions. Perception of the passive movement of a joint is already a spatial function that reveals an advanced degree of spatial organization, which is inconsistent with the general and absolute loss of tactile space that the aforementioned authors postulate. Stein (1928, 1930) and Stein and Weizsäcker (1926), who generally agree with what has been established regarding this patient, note the difficulty that arises in accepting passive movement as intact. They think that it should be as impaired as the other spatial functions, and that the contradictory result indicated would be due to a compensation produced by possible movements such as muscular jerks or twitches, even if minimal. Moreover, this preservation of passive movements in contrast to the abolition of the rest of spatial functions is in contradiction with all that is known about the sequence in the alteration of tactile activities, according to the most relevant authors.

Head and Riddoch (1920) and Head *et al.* (1920) established that position perception and passive movements are affected first and greatly, followed by spatial discrimination (with Weber's compass), and afterwards by spatial localization. The same was established by Foerster (1916, 1930), as well as by us based on brain-injured patients that we have had the possibility to study prior to this research on brain dynamics. This sequence of alterations is based on the dynamic reduction of functions, and it is understandable that since movement, both articular and cutaneous (mobile stimulus on the skin), is a very complex spatial activity, it must necessarily derive from more elementary spatial structures such as localization and even spatial discrimination. It is inconceivable that the more complex structures subsist whereas the elementary ones are abolished. In conclusion, the supposed preservation of the

aforementioned movement perception in the Schneider case is an error of observation, since in an inactive state, it would certainly be abolished under certain conditions of stimulation. It is also a serious lapse about the functional organization of tactile space.

As for tactile localization of stimuli, the study of Godstein and Gelb (1919) reveals certain phenomena entirely favorable to our interpretation, phenomena not duly considered by these authors because they have completely ignored the disorder of nervous excitability. Localization as well as other spatial functions manifest a radical change depending on whether muscular contractions are present or the subject is inactive.

These authors find: (1) Even at rest (inactive state according to us), if the stimulus is applied for a long time, the subject ends up localizing it. But they think this is due to the subject's inability to remain immobile under prolonged stimulation. In our opinion, the localization is due to the action of the stimulation time which in this patient, as in ours, is greatly increased (increased chronaxie). (2) Even in the case of allowing the patient muscular twitches (state of moderate facilitation), they note as a remarkable fact that they must use very strong stimuli for the patient to be able to localize them, much stronger than when the patient is only asked to say whether he has been touched or not. It is evident, although the authors have not noticed it, that simple contact and localization correspond to stimuli of different intensities (because of asynchrony) and that even with muscular jerks or twitches, such separation of excitabilities is patent. They try to attribute these singularities to attention deficit, but since attention would also be impaired for simple contact without localization, the explanation is not clear. (3) Even when there are twitching movements, the stimulus must last for a certain time to be localized. If this time is too short, localization is impossible. In this respect, the authors think that the patient does not have enough time to perform well the muscular mechanism. However, taking into account the probable excitability disorder, it is easy to understand that even with moderate facilitation the chronaxie is still increased. Thus, to achieve a complex function such as localization, a certain stimulus intensity and a certain excitation time are necessary, both of which increased with respect to a normal subject.

In point (1), it was argued that in the inactive state, if the stimulus lasted long enough, it was finally localized, the authors admitting that it was impossible for the patient to remain still. However, in point (3), they think that if the duration of the stimulus is very short, there is no localization because the muscle movements do not act long enough. All this seems a bit arbitrary, needing auxiliary hypotheses at every step, and if they had proceeded to rigorous observations measuring the intensity and duration of the stimuli applied in the different states, a single and simple explanation would have spontaneously emerged. We refuse as a whole the interpretations given by the authors on the three observations mentioned above, and we strictly adhere to the disorder of excitability relationships (increase of rheobase and chronaxie, asynchrony, summative facilitation, etc.). Concerning the theory of Goldstein and Gelb on that twitching movements, as well as other issues, we shall have occasion to discuss them later.

Having briefly reviewed the knowledge thus far on tactile localization, it will be very instructive to consider now the two patients studied in this work. In the course of several years of study of these subjects, we have gained insight into different aspects of spatial tactile disorder, particularly the fundamental one of tactile localization, until the complex and difficult problem of stimulus localization has been fully elucidated.

In the first observations made in 1938, the tactile examination of subject M was performed very superficially. The disorders found were limited to alterations of posture and passive joint movement. Given the predominantly occipital lesion, no attention was paid to sensory disorders that were not expected to be found. Sometime later, when inverted vision and other visual spatial orientation disturbances were discovered, we found that tactile space also showed pathological changes.

In contrast, in patient T, who had been examined earlier, it was possible to observe from the outset that tactile localization was based on certain muscular jerks or twitches without which localization was almost completely lost. At that time, we did not know in detail the investigations of Goldstein and Gelb (1918, 1919) on the Schneider case. Subject T showed the muscular jerks so flagrantly that we were able to prevent them easily in order to observe their effect on localization, and indeed the failure was then very great and constant. In such a circumstance, it was extremely unpleasant for the patient to sense the contact of the stimulus without being able to localize it. He was aware that thanks to the artifice of muscular contraction he was able to succeed in the localization tests. The same behavior was shown in passive joint movement and posture. All these alterations especially concerned the contralateral side of the brain lesion, at least at the beginning of the examinations. Later it was observed that both sides were impaired, with little difference. The patient became aware of his particular tactile disorder during the examinations, and then tried to contract the musculature in one way or another, which made the tests very difficult, and he was even reluctant to do them for fear of failing them. Such muscular contractions, of enigmatic significance for us at the time, were also observed by us with varying clarity in some other brain-injured patients with tactile disorders (1938). Also Kleist (1937), commenting on Schneider's case, reports having noticed them in some of his patients.

As for patient M, in 1939, shortly before the finding of the dynamic action, it was already extraordinarily striking how long it took him to localize a stimulus. In trying to find the muscular contractions, already observed earlier in the T case and so much studied in the Schneider case, it turned out that although they could be found occasionally in M, they were not as constant as in the mentioned cases. In M, there was a certain degree of spontaneous and unconscious sustained muscular tension that he performed spontaneously and unconsciously. However, what was very noticeable was the long time it took him to localize a stimulus whereas it seemed as if he had perceived simple contact much earlier. But once the phenomenon of facilitation by muscular effort in vision had been discovered (at the end of 1939), and the other questions of visual excitability elucidated, all these new data were applied to the investigation of touch, which showed characteristics entirely identical to those of vision. It is remarkable the phenomenon observed in the tests that led to the discovery of the facilitation phenomenon.

When the patient's head was held firmly, in order to avoid jerks and even certain movements of muscular tension, it was impossible for him to localize stimuli that at other times he was able to do. The patient then believed to explain the process by saying that in order to localize a certain location in his body he first had to know its situation with respect to his head, and therefore he had to "activate" his head first. This interpretation does not strictly correspond to the facts, but it is a fact to keep in mind when studying the body schema disorder in this subject. In fact, by means of facilitation, nervous summation immediately brings out the spatial function as a whole, without the patient having to resort to indirect orientations. Moreover, a precise knowledge of the excitability conditions revealed that, even without any facilitation, a sufficiently intense and long-lasting stimulus was perfectly localized. Thus, the conceptions of Goldstein and Gelb (1919) were discarded in this and other matters. In addition, being able to exclude any facilitating action, made it possible to study the patient in his true natural state, i.e., in the inactive state, thus bringing out the full extent of asynchrony (time lag). In this situation, intense stimuli that would be painful for a normal subject, if applied briefly, could go unnoticed, usually being perceived as a simple contact without reaching the slightest trace of localization. However, it was sufficient to prolong the application time for the function to be normal.

During the following years we devoted our attention almost exclusively to the study of visual functions in accordance with the new ideas of dynamic action, and to systematizing the new theory, as well as to checking the dynamic phenomena in case T. For these reasons, the phenomenology of localization in touch and other associated circumstances on excitability made little progress. However, it is worth mentioning the observation of tactile irradiation, which was compared with color irradiation (pathological vision of flat colors). In subject M, the two extreme cases of localization (simple contact and normal pinpoint localization) were already known, as well as the fact that simple contact, besides being devoid of a specific localization, was perceived as an extensive burst when stimulation was, for example, with a pin. Likewise, when the subject indicated a location, this did not usually correspond to a point, as it was in reality, but to a certain cutaneous surface.

The study of all kinds of new phenomena is, in any case, very arduous, and a progress in providing data and their rational interpretation needs a multitude of examinations and verifications. In addition, patients are unwilling to give details of the phenomena, and when they are obtained, very careful control is necessary in a series of tests, so as not to fall into considerable errors at every step. Thus, effective progress is a very slow work which must necessarily extend to numerous observations over several years. As mentioned elsewhere, the Schneider patient underwent a continuous study for about two years; however, as we know, neither a large number of pathological phenomena have been discovered in him nor has it been possible to take the necessary step to diagnose the patient from a physiological basis. Such a basis is what provides a correct and simple solution to the disorders found, and to many others that the subject should necessarily present.

By 1943-44, continuing the analysis of tactile localization disorder, a quantitative determination of the asynchronous beam of curves for both M and T under electrical stimulation was performed. This beam is the set of strength-duration curves correspond-

ing to the stages of spatial asynchrony, already known at that time: 1st, simple contact without localization; 2nd, diffuse localization (irradiation), and 3rd, normal localization without irradiation. Such rigorous determinations provided a great advance in the knowledge of the question, especially with regard to the different physiological levels due to the difference in the magnitude of the lesion in both cases, and to the variations within each case through the action of maximal facilitation, mainly in the M case, as we already know. Subsequently it seemed convenient to complete this already encouraging set of data, and a meticulous work yielded very valuable results although it took a great deal of patience on our part and that of the patient. To the three stages of evolution in the localization process, others were added that provided new phenomena of extraordinary interest, especially inverted localization. Thus, a pathological situation was obtained in touch similar to that in vision. The significant deviation of the localization of a stimulus from the real location of the stimulus, a phenomenon that was not sufficiently clear before, was also studied in great detail. The finding of new well-characterized stages in the asynchronous process of localization meant not only an enrichment in the phenomena but also a readjustment of previous data.

In summary, the continued study of our brain-injured patients since 1938 has gradually led, through a series of stages, to a full understanding of the nature of the localization of tactile stimuli. These stages of improvement are as follows:

1938: Some disperse findings on tactile spatial disorder (peculiar muscular twitches in T and lengthening of localization time in M) difficult to explain according to the usual theories.

1939: Finding of the phenomena of dynamic action and their application to problems of tactile space, especially localization. Experimental rebuttal of the ideas of Goldstein and Gelb (1919) on the Schneider case by interpreting all the disorders according to dynamic changes in excitability (strength-duration threshold curves, asynchrony or time lag, facilitation, etc.).

1941: Observations on tactile irradiation, a phenomenon analogous to color irradiation (pathological vision of flat colors).

1943-44: Correct determination of the strength-duration curves for three phases of the localization process (simple contact, diffuse or irradiated localization and normal localization), thus forming a set of asynchronous curves with varying degrees of asynchrony depending on the patients considered. These quantitative measures are complemented with other types of graphs such as recruitment, etc.

1945-46: An even more precise phenomenological analysis supported by a very complete series of tests and quantitative determinations, finding the important phenomenon of tactile inversion, various aspects of localization deviation, and types of irradiation, etc., until reaching a great systematic coherence in the process of tactile localization.

Such study, so long in time, reveals the complexity of a brain function as seemingly simple as tactile localization, and the difficulty in identifying the fundamental features of the process. Certainly, the analysis of tactile functions is much less amenable to objective evaluation than visual functions, and therefore, the separation between different stages in the asynchrony process is difficult and can lead to multiple

errors. The main procedure consists of subjecting patients to very particular conditions of both state of excitability and stimulation, in order to conduct a sufficiently detailed investigation. The effort and guidance of the work must fall entirely on the person doing the observation and not on the patient, who in principle is completely unaware of his own anomalies.

If we have dwelt on the general aspects of spatial disorder, highlighting the background of the localization problem as well as the development of the research presented here, it is due to the utmost importance of knowing the localization disorder and its mechanism for understanding all kinds of spatial structures in touch. It is harder in touch than in vision to separate some disorders from others, and at every step it is necessary to pay attention to a multitude of aspects that evolve at the same time and in which localization is always involved. This is so regardless of the tactile activities, from the most elementary tactile response to the most complex organization of schema.

The study that we shall now present on tactile localization, and tactile space in general, constitutes a completely novel part of brain pathophysiology.

21.2. PHENOMENOLOGY OF ASYNCHRONY IN TACTILE LOCALIZATION

Because of asynchrony, the tactile localization of a pinpoint stimulus is broken down into a series of intermediate phases, thereby showing the evolution of localization as a function of stimulation energy. Thus, there is no all-or-nothing behavior, as in a normal subject, i.e., either a specifically localized contact or the complete absence of any sensation. Instead, there are pathological partial functions that have no precedent in a normal subject.

The partial phases that can be distinguished are theoretically indefinite, but in order to describe the process by focusing on the most salient aspects, it is possible to distinguish five quite individualized phases which, as the intensity of a given tactile stimulus increases, appear in the following sequence: I, primitive perception; II, medial deviation; III, inversion; IV, proximal deviation; and V, specific localization. Only the latter is normal whereas the others are pathological effects of asynchrony, and therefore, the greater the asynchrony, the better the phases are manifested. In the development of localization, several factors are present, among the main ones are: irradiation, resulting from the destruction of localization at one point; spatial deviation due to a reduction of the body schema; and contralateral inversion of localization due to a disorder of tactile spatial orientation. The evolution of pain and temperature during the gradual process of localization must also be considered. Thus, at each moment we obtain a very diverse evolutionary set, a multiplicity of interwoven sensory factors showing the full complexity of tactile localization. The five phases indicated above are a fundamental starting point for understanding all types of spatial activities, and in this case, in relation to touch. In subsequent chapters, we shall continually refer to these phases to understand other types of tactile functions.

The phenomenological description of these phases has been carried out almost entirely on the basis of subject M, who is the one who presents maximum asynchro-

ny, and is therefore easier to study. M is also the patient who has undergone more frequent and detailed studies, not only because of his characteristics, but also because of the many difficulties to access the second subject (case T). The considerable amount of coherent details and their constant manifestation throughout multiple observations during the last years is a guarantee that allows us to describe entirely new phenomena, in which it is very easy to make observational errors, as we shall see. Within this section, we shall proceed to expose the phenomenology of the alteration, and in the following sections we shall study quantitative aspects by means of numerical relations on excitability and related graphs. In the following we describe the five phases mentioned above.

21.2.1. Phase I: Primitive sensation

By means of a mechanical stimulus on the skin of any part of the body, a tactile perception without any possible localization is obtained in both subject M and subject T when the intensity and duration of the stimulus are conveniently reduced. A sensation in the form of a gust spread diffusely over the skin is perceived. The subject can say that he has been touched but does not know where.

The test is performed on subject M in the inactive state and with eyes closed to avoid distractions. When the stimulus has the intensity to reach the first sensation, he perceives a band of irradiation that he estimates to be one hand span long, i.e., 20 to 23 cm, and about two fingerbreadths wide, i.e., 3 to 4 cm. He also believes that the band has a certain depth (1 to 2 cm?), as shown in Fig. 21.1. This irradiation is very uniform and it is not possible to distinguish individual zones or points. It is completely impossible for the patient to localize it on the body and to indicate which orientation it adopts, if along the body or perpendicular to it. He only feels a cutaneous situation that seems to penetrate somewhat into the tegument and also protrude from it as swelling, the former being more pronounced than the latter.

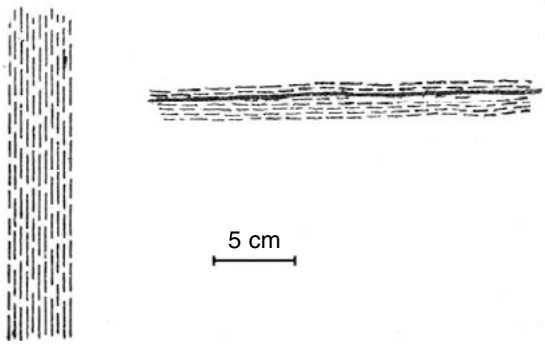

Figure 21.1. Diagram of the primitive sensation in subject M in the inactive state. A pinpoint stimulus gives rise to a band of irradiation (left) in some part of the body. Note the depth of the band (right) which penetrates and also protrudes from the tegument. Dashed lines indicate faint sensation, as a gust of air of the size indicated by the scale.

While it is true that such a diffuse irradiation sensation may share certain characteristics of a cramp or paresthesia, it does not correspond at all to a protopathic or hyperpathic sensation. The band of irradiation is essentially neutral, and it is impossible to arouse thermal or painful sensations at this stage. The intensity of the sensation thus obtained is the lowest possible.

Regarding the mode of stimulation, whether by single pinpoint or iterative stimulation, scratching, application of a given surface or blowing on the skin, the result is always the same: a similar band of irradiation for all cases provided that the appropriate level of excitation is not surpassed.

All these stimuli are very high in relation to the threshold of the normal subject who by functional synchronization correctly localizes the stimuli even with the tactile threshold. This large sensory deficit explains that if the subject is given a light slap on the face with a few fingers, he does not feel anything, reaching only the primitive sensation phase. The different regions of the body have no effect on the result, so it is possible to obtain this primitive phase both in the stimulation of the body surface and inside the cavities (stimulation of the oral cavity or the surface of the tongue). Even in the sclera it is possible to obtain the first phase, and sometimes also in the cornea by stimulating with a hair, although due to the reflex movements of the eyelids the second phase is often reached. The cutaneous reflexes are therefore triggered by the mere sensation of contact, without any localization. The same occurs when the wing of the nose or the cheek near the eye is lightly and briefly tapped, since the reflex response of palpebral contraction is elicited. This means that such reflexes take place subcortically and are completely independent of the sensory localization of the stimulus. Thus, the reflex is localized but the sensation is not. However, this does not entail any difficulty of interpretation. This is a common observation in certain lesions of the nervous system. Different nervous centers are also involved: from the brainstem for the reflexes mentioned, and from the cerebral cortex for sensory organization. In any case, the reflex muscle activity that may appear is very slight, enough to be observed by the researcher but not by the subject who is totally unaware of it. Only in the case of somewhat abrupt stimulation of the cornea can it happen that repeated contraction movements of the eyelids promote more generalized defense movements, and by acting as facilitation, the simple contact phase changes to other more evolved phases.

In relation to the different zones, it seems that perhaps the central zones of the body (face, chest) are somewhat more sensitive than the distal zones (extremities, especially the most distal part). When the same stimulus is applied to all of them, a slightly more intense sensation and perhaps a little less irradiation is sometimes felt in the central zones, without exceeding the limit of the first phase.

> At the beginning of the examinations in these subjects (1938-1939), this first phase of simple contact was difficult to be observed, not only because of the partial and automatic effect of facilitation by muscular action, but also because it was an unusual and strange sensation for the patients. They tend to exclude or disregard a tactile sensation that is not accompanied by a more or less specific localization; thus, it was possible that facial contacts able to elicit reflex contractions were considered by the examined subject

as stimuli without any effect. When the stimulus was of sufficient intensity, the subject's sensation was waited for to develop until it reached at least a rudimentary localization. In this case he took a long time to give a response to the stimulation. All these circumstances can give rise to many errors, especially if the excitability conditions are not well known, or in cases with little asynchrony such as the T case. It is therefore necessary to instruct the testing subjects conveniently, indicating to them the need to give their responses with great detail and precision. At the same time, it is necessary to adjust the stimulation mode to their excitability characteristics.

Under the action of facilitation by maximum muscular effort, the first phase can also be obtained in subject M, but it is necessary to use very slight and brief stimuli (for example, rubbing with cotton), otherwise the second phase is very easily reached. In areas of the body that seem to be more sensitive, such as the face, especially the lips, eyelids, etc., as well as the midline of the chest, the first phase is difficult to obtain under the aforementioned facilitation and is often almost confused with the second phase. In contrast, it is still easy to obtain simple contact on the limbs as long as the intensity of the stimulus is suitably reduced. It should be added that in the hands, facilitation by maximum contraction of the musculature reduces considerably the duration of the first phase, i.e., the first and second phases become very close to each other. If other types of facilitation are added, such as abrupt and wide movements of the limbs, a weak and brief stimulus (rubbing with cotton) on the hand is felt only as a simple contact, and sometimes as a second phase, but does not go beyond that.

As already mentioned, slight differences can be obtained within the first phase depending on the areas, i.e., the irradiation surface and the intensity of the sensation may vary slightly in skin areas of very different sensitivity (limbs and lips, for example). Likewise, there is a difference when comparing the inactive state with the state under facilitation: the irradiation of the simple contact under reinforcement seems to be somewhat smaller (perhaps three or four fingerbreadths shorter), and is very close to the second phase, which, apart from other characteristics, shows a smaller irradiation than the first phase.

In the T case, the first phase also seems to exist, although the size of the irradiation was not precisely determined. The essential point is that by using a weak stimulus and, above all, a brief one, contact is perceived without knowing where. Because of the smaller excitability deficit, the size of the irradiation is more difficult to obtain than in the M case.

A convenient and reliable procedure to correctly determine the thresholds in this first phase when the subject is under facilitation by muscular effort, is the stimulation by moving a single hair on the back of the hand by means of a stylus. In this case there is no reaction, whereas in the normal subject a complete sensation of localization is produced. By repeating the stimulus, the first phase is obtained, and if continued a little longer, the second phase is reached. In the inactive state, this hand hair procedure does not produce the first phase no matter how long the repetition of moving the hair is maintained. However, it is possible to obtain it using eyebrow hairs, which are stiffer, especially if several are stimulated at the same time.

Finally, we point out the sensory change that occurs by facilitation. Let us consider a stimulus of appropriate intensity such that in prolonged duration it does not lead to overcome the first phase. If the patient makes maximum muscular effort, he instantly passes from the first phase to the third or fourth. However, he does not reach normality, since from the fourth to the fifth (normal phase) there is a considerable gap (interval).

The primitive sensation phase lacks the possibility of differentiation, and stimulation with moderate cold or heat only provides primitive sensation. It is also not possible to register spatial and temporal intervals (vibration). Regarding the characteristics of general excitability in this primitive phase (useful time, fatigue, persistence, etc.), we refer to Section 18.1 on general excitability under mechanical stimulus, which corresponds to this phase studied.

Summarizing the characteristics of the first phase, there is maximum cutaneous irradiation in the form of an elongated gust of a certain thickness and uniform texture, and is impossible for the patient to indicate which orientation it adopts. There is no trace of localization nor organization of sensations and spatial or temporal structures.

21.2.2. Phase II: Medial deviation

This second phase, which is difficult to explore, has distinctive characteristics that allow its identification. A rudiment of localization or spatial organization appears whose most important manifestation consists in the deviation of the perception of all types of stimuli towards areas of the anterior midline of the body.

This phase was impossible to be distinguished at first, and then confused with the first phase or with more advanced phases such as the third and even the fourth (see Fig. 22.10). It was finally possible to uncover it in subject M by conveniently fractionating the examination of the localization process and trying to note the smallest details accurately. Quantitatively, the stimulus is very little higher than in the first phase, and moreover, the step width for the next phase is very small, even in inactive M. A convenient method of adjusting the necessary stimulation consists in iterative excitation, by which, with repeated contacts or gentle rubbing with the tip of a piece of paper or absorbent cotton, different phases are recruited slowly and successively, stopping then at the appropriate one.

For the first time a certain degree of spatial localization appears, although due to the maximal proximal deviation at this stage, stimulation anywhere on the body is sensed only on the body axis, i.e., there is an anterior medial localization. Careful tests show that in the midline there are three zones, head, thorax and abdomen, which collect stimuli coming from the head, upper limbs and lower limbs respectively. The sensation is still, as in the first phase, an elongated band or gust of a certain thickness, perhaps about four fingerbreadths shorter than in the first phase, with analogous reduction for the other dimensions. All these measurements are approximate and subjective since the patient under examination can only indicate them indirectly. The irradiation band is still homogeneous, with no differentiation of a central point. Being slightly smaller in size than in the first phase, it should offer a

slightly higher intensity of sensation. An irradiation of about 17-18 cm in length can be attributed to subject M in inactive state, especially in the case of stimuli on a limb which then produce a band of irradiation on the midline of the thorax. In this phase, unlike the first one, the irradiation band shows a well-defined orientation on the body, in the direction of the body axis.

At this stage it is still impossible to obtain tactile sensations. Painful or thermal stimulation only produces a sensation of contact or pressure. Perception of intermittent stimulation (vibration) is also not possible. Spatial discrimination in the usual sense (with Weber's compass) is abolished. However, sensation of duplicity in simultaneous stimuli may be obtained under special circumstances which we shall see later.

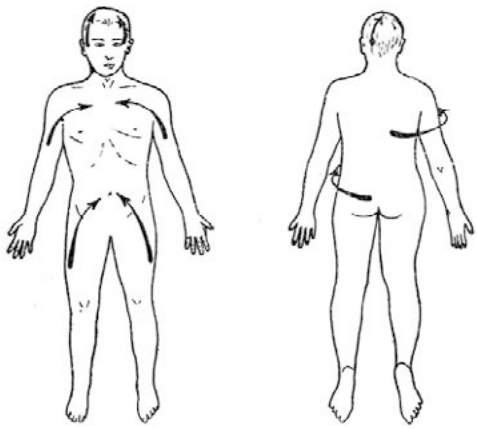

Figure 21.2. Phase II of localization (medial deviation). Deviation toward the midline and anterior plane of the body. (See also Fig. 21.5.)

The *medial deviation* that sensations undergo in response to stimuli has several important peculiarities, and deserves to be described in some detail (see Fig. 21.2). In this phase, whatever the site of stimulation on the limbs, the sensation is perceived by subject M, inactive, on the midline of the trunk, and with such a special distribution that the upper limbs are represented on the thorax and the lower limbs on the abdomen. If the stimulation is on the trunk, e.g., on the sides, the stimulus will be perceived on the abdomen or on the thorax, depending on whether the site of application is low or high, respectively. In all these cases the irradiation band is located on the trunk axis. When the most distal part of a limb (fingers or toes) is stimulated, a maximum spatial deviation is obtained. This can be considered the *maximum effect of proximal deviation,* which is more moderate in more advanced phases, as we shall see. In any case, both in the case of a stimulus on the side of the trunk and on the fingers, there is no other possible localization than on the midline of the body, as already indicated. Moreover, it makes no difference whether the stimulus is applied on the anterior or posterior part of the limbs, since in all cases the patient gives the same response: on the anterior midline of the trunk, not on the posterior. When the back is stimulated, either on the

midline or on the sides, a localization is also obtained on the anterior part, either on the thorax or on the abdomen, depending on whether the height of the stimulation point is on the upper back or on the lower lumbar region. It should be noted that this localization sensation cannot be compared in clarity to that experienced by a normal subject when stimulated in the chest or abdomen. These pathological sensations are diffuse and labile, and it is necessary to pay great attention to localize them in the midline of the trunk, and even more so to localize them on the anterior plane of the trunk. Several tests seem to demonstrate that this plane constitutes a zone of predilection, and that the posterior plane is systematically excluded. Such a deviation from the back towards the anterior plane of the trunk is confirmed in subsequent phases, although more moderate, as we shall see later on.

The prevalence of the anterior midline or of the anterior plane of the body over the exclusion of the dorsal plane is also demonstrated by the recent experiments of Rey (1947) on cutaneous localization by the stroboscopic method. Already in the normal subject, under special conditions, the anterior predominance is demonstrated by certain dynamic continuity effects (stroboscopic phenomenon) as follows. Two electrodes on the skin, distant from each other and successively excited in a short time interval, cause a sensory continuity between them. If the stimulating electrodes are placed one on the inner side of the thigh and the other on the outer side, at the same height, continuity is perceived on the anterior side of the thigh. The same result is obtained in the leg, arm and also the trunk. In the latter case, the electrodes were placed on the sides, their dorsal separation being shorter than the ventral. Despite such a difference, the dorsal region remained neutral and the sensory continuity was perceived in the abdomen following a ventral arc. Sensory effects seem therefore to be polarized towards the anterior part of the body. Thus, our findings on anterior predominance or deviation, obtained prior to those cited, and in another type of phenomena, may find support in the observations reported by Rey (1947).

As for the *head*, a stimulus anywhere on the head causes a sensation in an area located on the midline of the cranial vault. Facial stimuli on the sides or in the center are felt above the forehead with sensation of irradiation in the region from the cranial vertex to the root of the hair on the forehead. Thus, it can be said that the head behaves as a region independent of the rest of the body; and within the head, the face appears as a distal region, and the top of the skull up to the forehead, as a proximal or central zone. As for the neck, it is part of the zone that includes the head, and only in the lower part does the sensation deviate towards the chest.

There is therefore in this phase a strong deviation of stimuli anywhere on the body towards the mentioned three zones of predilection in the anterior midline of the body (Fig. 21.3), which collect excitations from their ascribed cutaneous regions. The body schema in this phase is reduced to three segments along the body axis, independent of each other and of very reduced width. The length of the segment corresponds to the length of the irradiation band, seeming equal in the thorax and abdomen, perhaps somewhat shorter in the cranial segment. The size of the body schema is considerably reduced, since the irradiation bands are only about 3 cm wide, resulting in an almost filiform body schema of about 50 cm in height. When

the stimulus is more intense and the third phase is reached, the segments lateralize away from the midline.

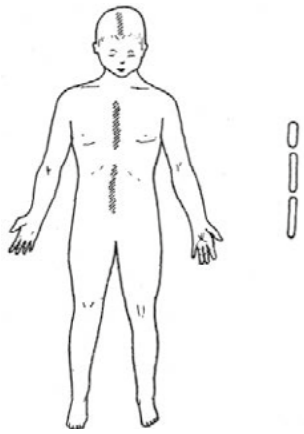

Figure 21.3. Phase II (medial deviation) with indication of the three small regions (striped part) in the anterior midline: cranial vault, thorax and abdomen, working as areas of predilection to localize stimuli. On the right, size of the body schema in this phase.

The independence of the three medial sectors is also corroborated by the fact that it is possible to obtain double or even triple sensation by simultaneously stimulating the different medial segments or their dependent distal cutaneous territories.

Thus, two stimuli, one on the hand and the other on the foot, or one on the thorax and the other on the abdomen separated by more than 24 cm, are perceived as two on the anterior midline. In the case of the hand and foot, the separation between sensations on the medial zone does not exceed 3-4 fingerbreadths. In contrast, if the two stimuli fall in the same subsidiary territory (foot and abdomen, or leg on one side and thigh on the other), only a single stimulus is perceived on the medial zone. When several points on the head or face are stimulated, only a single homogeneous irradiation sensation is perceived on the cranial vault. When the anterior plane of the trunk is directly stimulated and the separation between stimuli is less than 20 cm, it is not possible to achieve double sensation, but with more separation, a band of irradiation on the chest and a similar one on the abdomen are perceived sufficiently well. If one stimulus is applied at an extreme point of the thorax (near the neck) and the other at an extreme point of the abdomen (e.g., the groin), the interval between the corresponding longitudinal irradiations is not much greater than in the previous case. In the first case, of hand and foot stimulation, the separation between the two sensations on the midline is similar to that in the previous test.

Spatial discrimination is therefore quite rudimentary, and some sign may only appear under very particular conditions such as those indicated. Thorax and abdomen appear closer than thorax and head when stimulated simultaneously. By stimulating

distal areas of the three medial segments (e.g. ear, forearm and knee), three longitudinal irradiations are obtained along the body axis, independent of each other and separated from each other. It can then be stated that the total length, including gaps, can be about 50 cm, and the width about 3 cm. In this second spatial phase, the body schema is reduced to such dimensions (Fig. 21.3). That is, only on the surface determined by these dimensions is it possible to perceive tactile stimuli, regardless of their origin. It should be noted that all this is blurred and unstable, at the limit of functional capability, and also difficult to evidence. In no way can it be compared with stimulus localization in a normal subject.

Finally, we shall mention some aspects on the action of facilitation. In order to obtain the second phase in subject M under facilitation, the stimulus intensity must be markedly reduced with respect to the inactive state. Especially on the face, the stimulation must be very brief, otherwise, the third or even fourth phase can be easily reached. This phase II was not explored in subject T since he was examined for the last time long before the finding of the medial deviation. Subject M inactive, at this second phase, can reach the fourth phase by means of maximum muscular effort. For example, if being in the inactive state and conveniently stimulated in his right hand to obtain sensation in the thorax, with the mentioned facilitation, the irradiation becomes much smaller and with a central point, all in the right forearm (therefore, moderate proximal deviation), which corresponds to fourth spatial phase. With less facilitation, the third phase would be reached, which consists of localization in the left arm, i.e., lateral inversion and more pronounced proximal deviation than in the fourth phase.

In summary, the main features of the second phase (medial deviation) are as follows. There is only a weak localization in the anterior midline in the form of a band of homogeneous irradiation, slightly smaller than in the first phase and oriented along the body axis. There are three segments of predilection in the anterior midline (cranial vault, thorax and abdomen) which pick up the stimuli applied in the ascribed territories, including the dorsal plane of the trunk. The sensory body schema is greatly reduced (filiform about 50 cm long). There are no well-defined sensations nor intermittent sensations. There is double sensation of stimulation only when stimuli are applied simultaneously in autonomous regions.

21.2.3. Phase III: Inversion

This is a spatial phase of great physiological importance because it is equivalent to the inversion of the orientation of the visual image. For this reason, and given the complexity of the phenomenon, we shall devote a special chapter to it. We limit ourselves now to continuing the study on the development of spatial localization.

This phase, like the previous one, shows very small functional separation with the adjacent phases (see phase curves further on in Fig. 22.10). Therefore, only in subsequent more detailed tests, phases II and III were revealed. In spite of having some resemblance with the second phase (irradiation, labile localization, very reduced

and poorly differentiated body schema, etc.), phase III already shows a significant progress in the localization process. It is remarkable the strong proximal deviation of the sensation without reaching the medial deviation of the previous phase and, above all, the inverted sensation with respect to the midline (*contralateral localization*).

For the first time, tactile specific sensations appear, as well as certain rudiments of spatial organization (movement, duplicity, etc.). In addition, different gradations of the phase are obtained by varying the stimulus energy, i.e., some differentiation within the phase is possible. For this phase to appear, a significant asynchrony is probably necessary, as in subject M, inactive. This subject, under maximum facilitation, no longer presents the contralateral inversion of the third phase. Therefore, it is presumable that subject T, in whom this phase was not explored, does not present it, as will be discussed in other chapters. The following exposition thus refers to subject M in inactive state.

An iterative stimulus (repeated rubbing with the tip of a somewhat hard piece of paper) applied to the back of the right hand, for example, causes a sudden change from phase II (irradiation on the thorax) to phase III (left shoulder, contralateral), and then continues along the arm on the same left side (Fig. 21.4). It is difficult, even when the stimulus is regulated, to achieve a gradual transit between the midline and the contralateral shoulder. Instead, it is possible to obtain, by varying the stimulus a little, some recruitment within this phase III, tending to lower the localization from the shoulder to the elbow, until it is finally located homolateral to the stimulus.

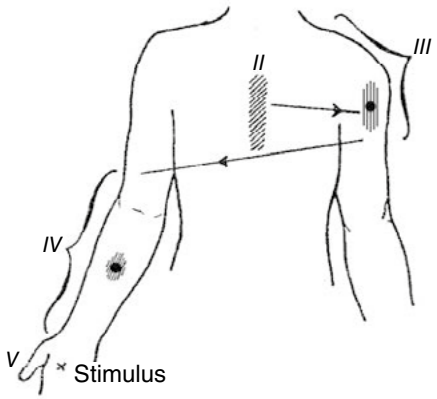

Figure 21.4. Diagram showing contralateral localization in phase III. Note its relation to phase II and phase IV.

Whatever the degree of the third phase, there is always irradiation but it is no longer homogeneous due to the presence of a point in the center of the irradiation band. This band maintains its orientation along the axis of the body or of the limbs when it is located in the latter (only in the most proximal zone). The area of irradiation in this phase is smaller than in previous phases and, within the subjectivity of all these measurements, can be estimated at 16 and 10 cm in length at the begin-

ning and end of the phase respectively. When stimulating in one hand, the irradiation in the contralateral shoulder would be the greatest, and when passing to the elbow (contralateral to the stimulus) it would be the least. Analogously, the width and thickness of the irradiation band are reduced. A probable moderate intensification of the sensation with respect to the previous phases would be due to the appearance of a central point in the band, which stands out just enough to be noticed.

The most appropriate way to study the characteristics of this phase is to observe it in the case of stimulation on the *extremities,* as has been indicated in part in the above examples. If a stimulus is applied on one hand and its intensity is gradually increased, a progressive transition from the second to the third phase can sometimes be found with great care. From the midline of the thorax, a slight contralateral deviation of three to four fingerbreadths begins, with a faint central point appearing in the band of irradiation. By increasing the stimulation, irradiation is directed first to the shoulder and then to the arm (both contralateral). Three different degrees of proximal deviation are thus obtained, all contralateral to the stimulation site. When the stimulus is applied to the dorsum of the hand, the transit to the homologous side (fourth phase) usually occurs at the level of the elbow. All this is valid for both upper and lower extremities and for both left and right sides.

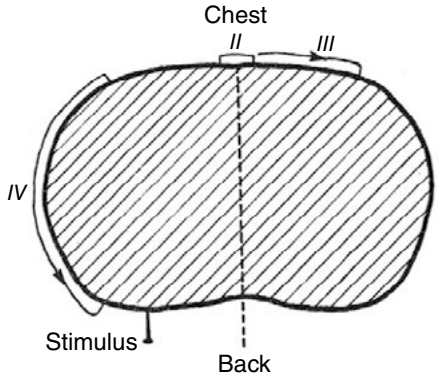

Figure 21.5. For stimulation on the back, the corresponding localizations are strongly deviated towards the chest (phase II), and also contralaterally in phase III.

For the *trunk*, the same type of proximal deviation appears, which is strong and contralateral. A stimulus on one side of the thorax, toward the flank, is localized centrally in the second phase, and on the contralateral side in the third phase. In this phase, the distance to the center is much smaller than the distance from the stimulus to the midline. When the excitation is intensified, the localization moves to the fourth phase, and the lateral inversion is suppressed, but some deviation toward the midline still remains. This behavior is also observed when the excitation is applied to the back (Fig. 21.5). In this case, the proximal deviation is more pronounced, and in the third phase the subjective localization falls on the anterior plane (the chest), towards the contralat-

eral side, even when the stimulus is applied three or four fingerbreadths away from the midline on the back. Therefore, the back behaves as a distal sector, and since the anterior plane predominates in the proximal deviation phenomenon, the back is excluded in the third phase. As we shall see in the next phase, the distal sectors of the body and the back begin to become involved.

As for the *head*, taking into account what was said above (Sec. 21.2.2) about the face (as a distal region) and the cranial vault (as a proximal or central region) we can guess what happens in this phase. For example, a stimulus at the ear is perceived at the vertex in the second phase, and contralaterally two or three fingerbreadths away from the midline in the third phase. Even if there is a transition to the fourth phase, there is still deviation, and the sensation is homolateral toward the temple. The third phase is therefore localized exclusively in the cranial vault, regardless of where the stimulus is applied on the head.

In addition to the inversion, the strong proximal deviation of the third phase implies a notable reduction of the body schema, which, although increased with respect to the second phase, is far from reaching its normal shape and size (Fig. 21.6). Thus, the possible body schema, besides being very small, is diffuse and unstable; the posterior plane does not yet exist, the extremities only in their proximal half, and the face and neck are also absent.

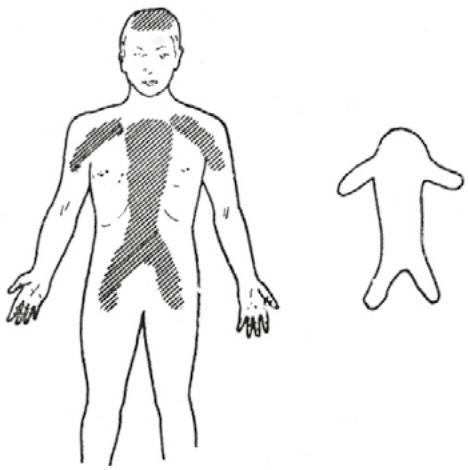

Figure 21.6. Body schema in phase III (striped area). On the right, the corresponding homunculus. In addition to the general reduction in size, all distal parts of the limbs are excluded, as well as the face and back.

When the localization is contralateral (inverted) in the proximal part of the limbs, it is important to note that it is not possible to distinguish whether the stimulus is anterior or posterior (palm or dorsum of the hand). It seems that the subjective location of the irradiation band with central point is either indeterminate in the proximal extrem-

ity, or it is located dorsally (external). In the latter case, it would be a *dorsal predominance,* in the same way as the aforementioned predominance of the anterior plane of the body. In relation to the lability of localization in this phase, it should be noted that shortly before the discovery of the phenomenon of localization inversion, the subject, stimulated on one limb, responded that he had felt something on one limb, but he was unable to tell which side. Later, it was found that a localization with lateral inversion had occurred.

Another characteristic of the third phase is that pain and temperature appear for the first time, albeit very slightly, and more precisely towards the middle of the phase; that is, for a painful stimulus in one hand, the algic sensation appears weakly when the subjective localization corresponds to the contralateral shoulder-arm. The same occurs with temperature.

As for spatial organization or differentiation, a certain aptitude for duplicity of stimuli already appears at this stage, for example, within the same limb. However, considerable separation is required between the two simultaneous stimuli; thus, for a distance of at least one stimulus on the hand and another on the elbow, the subject perceives the duplicity contralaterally and towards the shoulder. All these circumstances are most probably due to the fact that the irradiation is smaller than in the previous phases, but above all to the existence of a central point, and to the progress in the whole functional organization. There is also perception of motion, although with spatial characteristics that we shall see later. This perception is proximal and contralateral (lateral inversion) and of inverse direction to that of the real moving stimulus. However, the intermittency of stimuli (vibration) belongs rather to the fourth phase, and if it existed at the end of the third phase, it would be difficult to appreciate because it would be a very weak indication.

Finally, we shall examine the effect of facilitation on this phase. By means of maximum muscular effort, the stimulus, which in the inactive state gives rise to the third phase, causes the passage to the final part of the fourth phase, i.e., to almost normal localization. Nevertheless, some proximal deviation still appears, although minimal. This occurs in the case of stimuli on a hand, an ear, etc. Thus, the stimulated site is not completely reached by such facilitation, but it is very close. When the subject is under facilitation by maximum muscular effort, and the aim is to obtain the third phase by considerably reducing the energy of the stimulus with respect to the inactive state, it is impossible to obtain it; either phases I and II arise or there is a transition to the fourth phase, but the characteristic lateral inversion of the third phase is never reached. This absence persists in many other tests, so the exclusion seems to be confirmed. This does not preclude that on rare occasions a third phase under facilitation is achieved, which can be interpreted as due to a submaximal effort that allows some margin, albeit very small, for the inverted third phase to appear.

In summary, the most salient features of the third phase are as follows. The irradiation band is smaller than in the second phase and with a central point, contralateral localization to the stimulus, and strong proximal deviation. It is possible to differentiate several degrees within the phase. The body schema is very small and diffuse, excluding the distal half of the limbs, as well as the posterior plane of the

trunk, face and neck. There is perception of motion (kinetic stimulus on the skin), some spatial duplicity and pain and temperature sensations. There is not yet aptitude for intermittent sensations (vibration).

21.2.4. Phase IV: Proximal deviation

This phase is easy to demonstrate in both subjects (M and T). Already in the first years of special examinations (1940-41) it was evidenced. It is present even in cases of mild asynchrony, therefore its incidence in brain tactile disorders must be very high, in contrast to the previous phases, especially intermediate II and III.

This phase, with a larger step in excitability (see phase curves further on in Fig. 22.10), starts when the localization is no longer inverted, i.e., just after the transit from contralateral to homolateral localization, and ends when the localization is practically normal, i.e., without deviations or irradiations of any kind, which corresponds to the fifth phase. It involves considerable recruitment, for which a progressive increase in stimulus intensity is necessary. The degree of proximal deviation and irradiation, much lower than in the previous phases, is related to such intensity. In subject M, inactive, the heterotactile interval of the tactile modalities is overcome, and there can be a more or less intense well-defined sensation. With mechanical or electrical stimulation, a very slight pain is awakened at the beginning of the phase, becoming very intense at the end of the phase, this end being indispensable for recruiting the more advanced degrees of sensation, at least in the inactive state. The most notable characteristic of this phase is a moderate proximal deviation, always homolateral. In addition, the irradiation tends to adopt the shape of an oval of variable size, depending on the intensity of stimulation.

As soon as the localization moves from the contralateral side (third phase) to the homolateral side (fourth phase), or shortly thereafter, the spatial localization tends to become clear, i.e., it is known whether the stimulation corresponds to an anterior or dorsal area of a limb, or whether it corresponds to the face or towards the back (considered as distal areas), even when there is a noticeable proximal deviation at the beginning. In contrast, on the contralateral side, the subject tends either to locate the site on the dorsal (outer) side of the limb, or it remains undetermined, as mentioned above. The irradiation that occurs is highly variable. At the beginning the irradiation band is 10 cm or a little more, with the central point of the previous phase more intensified. Towards the middle of the phase it is about 6-7 cm, and oval-shaped, which means a type of irradiation with a spatial organization. At the end of the phase, if the stimulus is a painful pinprick, the oval decreases considerably, although a residue of 1.5 to 2 cm in diameter always remains (Fig. 21.8), impossible to remove in the inactive state. If, for example, subject M in the inactive state is moderately pricked on the dorsum of one hand for a time long enough not to enter into consideration in the excitability relations, he feels pain in the middle of the contralateral arm (Fig. 21.4); but with very slight intensification of the stimulus, the sensation moves from the level of the elbow to the homolateral limb, appearing a band of irradiation of about 10 cm in length on the upper forearm. By increasing the stimulus,

the proximal deviation and irradiation decrease, and the above-mentioned oval of irradiation appears in the lower third of the forearm (Fig. 21.7).

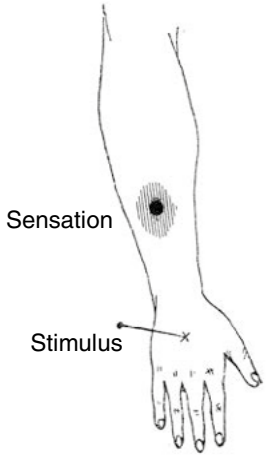

Figure 21.7. Proximal deviation in phase IV. Stimulation on the back of the hand and sensation on the forearm (oval-shaped irradiation with a more intense central point).

The irradiation is shortened at the ends, and the contour tends to acquire an elliptical shape approximately. In any case it can be known that it is neither a band, as before, nor a circle. The distance from the point of application of the stimulus to the center of the oval is in this situation 8 to 10 cm. When the oval has become as small as possible by increasing the stimulus, it still has a certain size (1.5 cm), and it is already localized on the hand with a very small deviation, almost negligible, like that of a finger. The pain due to the prick is at that moment extremely annoying.

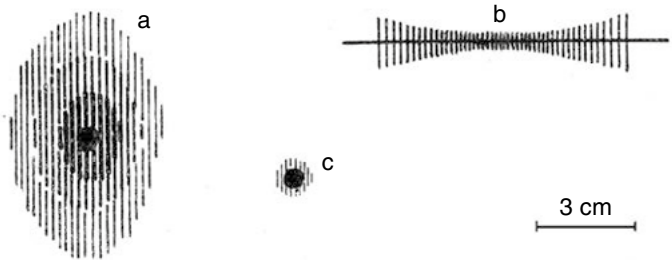

Figure 21.8. Irradiation in phase IV. (a) Characteristic oval shape with a more intense center. (b) Oval profile (thickness in the skin). (c) Irradiation at the end of phase IV.

The characteristics of the oval-shaped irradiation deserve comment in some detail (Fig. 21.8). The irradiation presents a point in the center where the sensory intensity is

greater and with some moderate superficial pain that gradually decreases towards the periphery. It seems that there is still a more eccentric zone devoid of well-defined sensation. Since the irradiation is not only superficial, but also in depth to some extent, it can be felt that the thickness of the oval is very small or almost null in the center, and that it increases significantly towards the periphery, i.e., it adopts a biconcave shape (Fig. 21.8 b). It should be noted that the oval shows some organization, and all its characteristics can be best perceived when it is at its maximum size. The subject is also able to perceive the orientation adopted by the oval, especially as long as it does not reach too small a size, in which case it cannot be distinguished from a circle. Like the irradiation band in other phases and in this one at the beginning, the oval is oriented on the skin generally following the body axis, and along the limbs.

At the beginning of the oval (maximum oval), its size varies somewhat according to the regions of the body: very small on the eyelids and tongue (size between a coin and a lentil), somewhat larger on the lips and cheeks, and much larger on the limbs and trunk. The oral cavity presents special characteristics. When the inner wall of a cheek (mucosa) is stimulated and the third phase is reached, contralaterally, the sensation is localized externally, up on the skull; and in the fourth phase, it is localized homolaterally on the inner wall. If facilitation is then applied, the sensation is localized closer to the stimulus. All this has been tested several times with stable results.

In the fourth phase it is possible to localize stimuli on the various areas excluded in the previous phases (face, back, neck, more distal part of the limbs, etc.), but a certain moderate proximal deviation persists, which diminishes with intensification of the stimulus but does not disappear completely, nor does the small oval or circle. A very painful stimulus on the earlobe is always localized a little in front of the stimulus site or on the cheek, no matter how painful the stimulus is. However, by means of facilitation, the localization coincides with the stimulus application site. All this corresponds to the end of the phase, since at the beginning, the localization may be on the homolateral temple. The same occurs with other parts of the face. A stimulus on the lips may be felt, depending on its intensity, either on the forehead or on the nose, and only by painful stimulus the localization comes close to the place of its application. Excitation at the tip of the tongue seems to be localized towards its base, and although the deviation is small, it is sufficient to be felt. With more intense stimulation, or with facilitation, the deviation disappears.

In order to indicate the subjective localization of the irradiation oval, subject M, inactive, answers verbally with eyes closed or without looking at the examined area. It is relatively easy to indicate the localization on very significant areas (elbow, wrist, tip of the nose, ear, chin, etc.), but on other regions he can only give somewhat approximate answers (along the limbs, trunk, etc.). In the latter case, M may be asked, for the sake of accuracy, to compare the localization in the fourth phase with the practically normal localization of a strong stimulus under facilitation by maximal muscular effort. To inform about the differences, he can do so either verbally or by pointing on a graphic model of the examined part, or on his own body or on that of the examiner. Thus, it is possible to specify precisely the localization deficit at the end of the fourth phase with an observation of great interest (Fig. 21.9). For example, if a pinpoint stimulus is applied to a fingertip (calloused skin) with a weight of 100 g, it is localized at the prox-

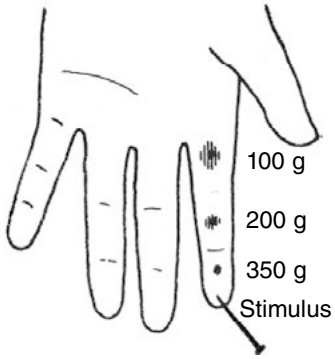

Figure 21.9. Phase IV. Proximal deviation in very distal areas. Deviation and irradiation vary according to the intensity of the stimulus.

imal end of the first phalanx. If the weight is 200 g, it is localized at the end of the second phalanx (rather painful). If the weight is 350 g, it is located with intense pain in the center of the third phalanx or fingertip. There is always some proximal deviation which at the end of the phase may be 1-1.5 cm with an irradiation of the size of a lentil.

Absolute normality therefore does not belong to the fourth phase for subject M inactive. Under facilitation by muscular effort, complete normalization of localization and size is obtained, even with a stimulus lower than the maximum in the inactive case, about 300 g or less. In any case, a fairly accurate localization can be obtained with much less painful stimuli than in the active state, since what is achieved with 350 g in the inactive state, is achieved with 200 g under facilitation, a stimulus saving that varies between one-third and one-half.

Finally, it should be noted that most of the qualitative and spatio-temporal tactile discriminations take place in the fourth phase. Already from the beginning of this phase, pain and temperature are perceived, as well as motion over the surface and duplicity of stimuli. The latter two sensations tend to improve considerably towards the end of the phase. Intermittency (vibration) appears for the first time, most probably towards the middle of the phase. In addition, the body schema increases considerably in size and especially in coherence, compared to the third phase. However, this situation is far from being similar to the normal state, and in addition to the necessary increase in stimulus intensity and application time, a series of functional reductions appear, which we shall deal with further on.

In summary, the characteristics of the fourth phase are as follows. There is localization with moderate proximal deviation, and homolateral to the stimulus. The irradiation has an oval shape with a certain structure consisting of an intense central point with decreasing tactile sensation towards the periphery, and a much smaller thickness of the oval in the center, etc. There is localization in the area close to the stimulation, being possible the localization in previously excluded zones (face, back, distal zones of the limbs, etc.). Motion, intermittency (vibration), duplicity, as well

as pain and temperature are perceived, and the body schema reaches a near normal size. However, there is still a marked functional reduction.

21.2.5. Phase V: Specific (normal) localization

In the previous phase, a completely normal specific localization is not achieved in subject M inactive. No matter how much the stimulus is intensified, some irradiation and deviation remains. This is what happens in vision, for example. If M is in the inactive state, it is neither possible to completely suppress color irradiation nor to achieve a completely correct visual orientation in monocular vision. The recruitment of the asynchronous levels is not complete, and there is always a remnant of asynchrony, albeit very small. When the functional state is more favorable and the asynchrony much smaller, for example in M under facilitation or in subject T, the mere increase of the stimulus is sufficient to reach a specific (normal) localization. In this case, the fourth phase will lead directly to the fifth (normal) phase.

It follows from the above that fully specific localization without deviation and irradiation is only possible in subject M by facilitation and a fairly intense stimulus, although its intensity may be somewhat less than that needed to achieve the end of the fourth phase in the inactive state. Examples of this have already been indicated (Fig. 21.9). Anomalous traces of irradiation and deviation that may exist in other areas, such as the back or others, are more difficult to examine because these areas are extensive and with low specificity. Such remnants should also disappear and the specific localization reach the whole body. If even with intense stimulation and facilitation, some irradiation of the order of millimeters seems to persist, it should be considered within the limits of perception.

All other tactile functions improve considerably when specific localization is achieved (two-point discrimination, vibration, motion, body schema, etc.). However, functional activity is far from normal, although the difference from normal is much smaller than at the end of the previous phase. A detailed description of such behavior will be presented in another chapter.

21.2.6. Sumary

As a general summary of the asynchrony phenomenon in spatial localization, it can be stated that it is mainly characterized by *irradiation* and *deviation*. Both progressively decrease as the function approaches normality (Fig. 21.9). This developmental process is not limited to a few functions, but involves all tactile activities without exception. This global process entails a great functional complexity, as seen for example in contralateral inversion, which involves tactile orientation function. Hence, a multiplicity of reactions appears in pathological localization. This can lead to a multitude of important errors when studying patients, as they tend to respond in a diffuse or vague manner at first, and there can also be errors on the part of the examiner if he does not keep the test conditions sufficiently constant.

The localization process exposed needs to be completed at several points with the study of other more complex spatial functions that have so far only been men-

Table 21.1. Tactile spatial development in subject M in the inactive state. First row: modalities of differentiation or functional organization in increasing order of development. First column: localization phases, I-V. Phase V only appears with facilitation. (See the text.)

	Irradiation	**Deviation**	**Body schema**	**Side**	**Intens. phase discrim.**	**Pain, temp.**	**Spatial discrim.**	**Motion on skin**	**Vibrat.**	**Forms**
I *Primit. projection*	Max. uniform band – Indist. project.									
II *Medial deviation*	Smaller uniform band – Along body axis	Anterior medial line – Maximal proximal deviation	Max. reduct. – Three filiform segments							
III *Inversion*	Smaller band with central point	Large proximal deviation	Reduct., difuss. – Distal exclus., and face, neck, back	Contralat.	Signs	Signs at mid-phase	Signs	Signs of mov. (inverted)	?	
IV *Proximal deviation*	Oval with structure	Moderate proximal deviation – Distal localiz.	Almost normal size – Coherence	Homolat.	Marked	Very strong at end of the phase	Weber (initiat.)	Larger but sub-normal	In the middle of the phase	Unstab.
V *Specific localiz.*	Negl. – Point localiz.	Specif. localiz. – Negl. deviation	Almost normal	Normal	Almost normal	Almost normal	Almost normal	Almost normal	Sub-normal	Almost normal

tioned. The phenomenology now established is fundamental as a starting point for further studies and a summary of it is given in Table 21.1. In the first column of Table 21.1, the five phases of localization are indicated. The first row shows the modalities of differentiation or functional organization in approximately increasing order, although some belong to the same functional level. The 'Irradiation' and 'Localization' columns show the main localization phenomena, partly also shown in the 'Body schema' column. In the table it can be seen that functional differentiation is null in phases I and II, there are signs in phase III, it progresses greatly in phase IV and tends to normality in phase V.

21.3. MEANING OF THE SPATIAL DISORDER. STRUCTURAL CHANGE OF THE SENSORY FIELD

In view of the set of the above phases, it is now appropriate to give an interpretation of the essential features of the spatial disorder. Leaving aside special circumstances, such as inversion, it is evident that in any of the phases, localization is determined by the degree of proximal deviation and by irradiation. It is observed that with pinpoint stimulation the disorder is twofold, since there is an absolute alteration of localization (due to deviation) and a relative alteration (due to irradiation) that modifies the pinpoint modality of the stimulus.

21.3.1. Relative disorder

The analysis of the *relative* disorder (irradiation) shows first of all that its size as well as its thickness are inversely related to the intensity of the sensation (Fig. 21.10). To the very large irradiation of the first phases corresponds a very weak tactile sensation, whereas in the later phases the irradiation tends to zero, i.e., the sensation tends to become punctiform, corresponding then to a very painful intensity.

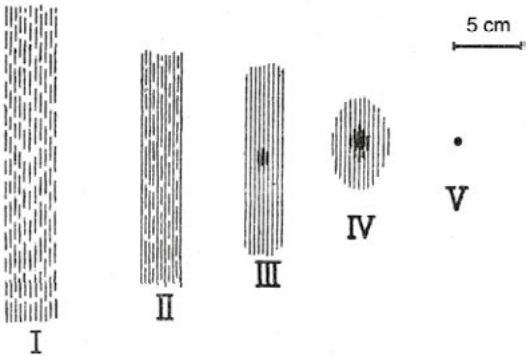

Figure 21.10. Diagram showing the irradiation in the five phases of localization as the intensity of the stimulus increases.

This disorder of pinpoint localization gives rise to both color irradiation (vision of flat colors) and tactile irradiation, the tactile system showing phenomena similar to those studied in the visual system. In vision, we find that color can appear as "aerial color," "flat color" and "surface color," with irradiation and voluminosity decreasing in that order. There is still an extreme pathological phase that appears as achromatic fog, where there is no color due to the photochromic interval, and the faint luminous sensation is voluminous, diffuse and wide, even though the stimulus is chromatic and almost point-like.

In the same way, touch presents, according to the phases of localization, different degrees of irradiation in depth and on surface. The achromatic fog mentioned corresponds to the primitive projection in touch (phase I), whereas the aerial colors correspond to the tactile phases with a band of irradiation (phases II and III). Flat color vision (usual in subject M), corresponds in touch to irradiation in a more organized space (phase IV), and normal surface color vision corresponds to the pinpoint localization of phase V. As in vision, now in touch we also find irradiation in all directions (surface and depth), but examining the detail of the phenomena, we also find certain differences that are important to note. In vision, irradiation predominates in the direction of depth (frontal irradiation), being much less pronounced in the other directions (lateral irradiation). In contrast, in the case of touch, the irradiation thickness is considerably smaller than the irradiation surface (lateral). Moreover, whereas in vision, lateral or surface irradiation is similar in all directions, in touch it adopts the marked form of a band. Even when irradiation in the form of a diffuse band tends to diminish, it adopts an oval shape, persisting the predominance of longitudinal irradiation.

Such differences do not seem difficult to interpret if one takes into account the different functionality of vision and touch. The former is closely related to spatial depth, whereas touch is very weakly so. The visual field has a circular structure, whereas the tactile field or body schema is clearly elongated. It is therefore understandable that in the case of touch, irradiance is band-shaped and only moderately thick. Thus, the spatial disorder of irradiation finds, both in vision and in touch, certain *previous molds*. Such an elongated arrangement in the case of touch, due to the influence of the body schema, exists even in the most primitive spatial stages, where all reference of localization in the body is lacking. The *shape of the sensory field*, and therefore the action of the body schema, determines the *irradiation form*.

In the comparison between color irradiation and tactile irradiation, it should be noted that although in the study of flat colors, reference was made to the vision of colored surfaces (piece of cardboard), and now in touch, to mainly pinpoint stimuli, such a difference in stimulation does not essentially prevent direct comparison. In the lower phases of tactile localization (I to III), the size of the stimulus applied is indifferent, influencing a little more in the later phases according to their characteristics.

A special issue in relation to irradiation is the stinging sensation, which corresponds to the well-known clinical test to distinguish between touching and pricking (with the head and the point of a pin respectively), and although it has been discussed when studying pain sensation (Sec. 19.2), the issue should now be clarified in relation

to the phases of irradiation. In the first phases (at least I and II) such a distinction is impossible since these phases correspond to homogeneous irradiation. This distinction is possible during part of phase III and during all of phase IV, but indirectly, since a true pinprick sensation is never obtained, but rather a somewhat extended pain. When either the head or the tip of a pin is applied on the skin with the same pressure, the sensation is certainly different; for example, the first phases are obtained for the head, and phase IV for the tip, but this distinction is not the one perceived by a normal subject after being touched or pricked. In M inactive, it is possible to obtain in phase IV the same sensation (equal irradiation and deviation) by applying the tip of the pin as by applying the head of the pin with greater pressure, being impossible any distinction that in a normal subject is feasible. All these circumstances have consequences for the acuity function (Weber), for the appreciation of tactile microstructures (texture of objects), for tactile recognition, etc., as will be seen below.

21.3.2. Absolute disorder

The *absolute* localization disorder (proximal deviation), diversely pronounced according to the different phases (Fig. 21.11), is in fact a *concentric reduction of the body schema* (Fig. 21.14). Here again, the similarity of behavior between the visual and tactile

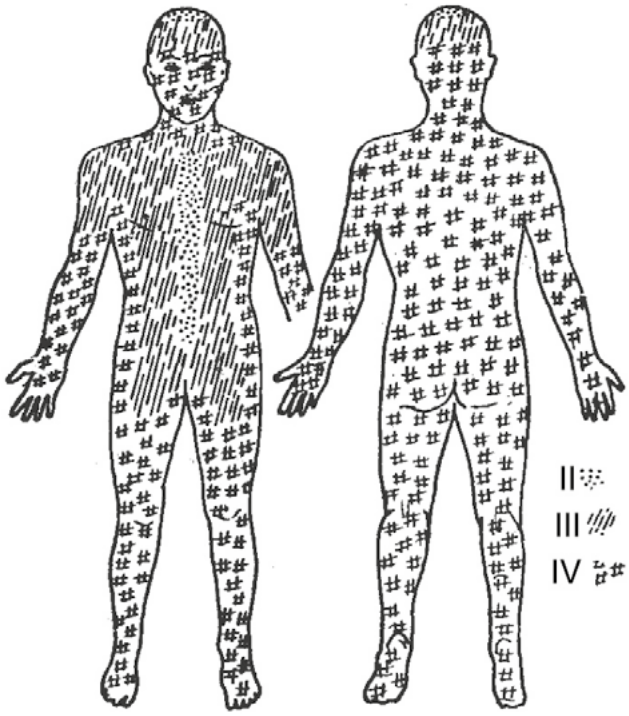

Figure 21.11. Localization zones for phases II, III and IV. Note their different extension and the tendency to concentric reduction and narrowing of the body schema in the first phases.

systems is evident. Concentric reduction of the visual field and concentric reduction of the body schema just mean concentric reduction of the sensory field in any of the alluded systems.

Using a suitable stimulus on any part of the body, simple contact sensation can be obtained, but the stimulus can only be localized in phase II in the anterior midline and in a very narrow area. If the stimulus is applied on a finger, this medial localization is an extremely large deviation. This means that the field for simple contact sensation and the field for spatial localization are very dissimilar. The field for spatial localization is reduced to a narrow band in phase II, whereas the field for simple excitability extends over the whole body (Fig. 21.12). This size disparity is maximal in phase I, since the spatial field is null because localization is completely abolished. Instead, the disparity tends to decrease through phases III and IV, until in phase V the disparity disappears and the fields coincide, the sensation being localized at the same site of stimulus application. Thus, the spatial field begins to develop, at least in M inactive, when the field for simple contact sensation already exists in its entirety, which indicates that the latter is much more resistant to functional reduction. It is clear that the importance of proximal deviation is related to the degree of *incongruence of the aforementioned fields* (*simple contact field* and *spatial field*). The body schema is thus identified with the spatial sensory field, whereas it is alien to the simple sensory field, a difference which is only possible pathologically, due to asynchrony, since in the normal subject there is not the slightest sign of this difference.

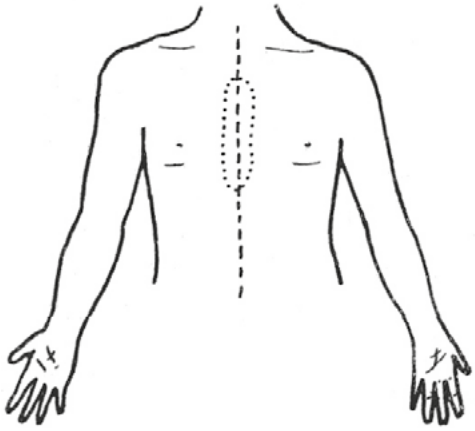

Figure 21.12. Incongruence of fields because of abnormal sensory interval (gap) due to asynchrony. For a functional level of phase II, the whole body surface is excitable, but localization is deviated and restricted to the anterior midline area (delimited by dotted line).

To be more complete about the important process of concentric reduction, it should be noted that this reduction can also occur in the simple (sensitive) field, since in all cases the development is from the center to the periphery because of the *medial privilege*. When describing phase I, it was already indicated that the central regions

of the body seem to be more sensitive than the peripheral ones and, of course, the same is true for the other phases. Thus, let us consider a stimulus applied in the central zone, with threshold intensity to obtain phase I; if this same stimulus is applied in the periphery or in a more distal region, it must be below the threshold for this phase. This is a centripetal narrowing of the sensitive field, resulting in a situation identical to that of the concentric reduction of the visual field. In this respect we have not performed sufficiently precise quantitative determinations about the tactile field, but the different sensitivity from the center to the periphery is evident.

It is a well-known fact that in tactile hemianesthesia and hemi-hypoesthesia, distal areas suffer more than central areas, not only in cortical lesions but at any level of the neuraxis, such as lesions of the thalamus, or of the spinal cord. Moreover, in mild lesions or in advanced stages of recovery of the disorder, symptoms are easily manifested only in the more distal areas of the body such as hands and feet.

Redlich (1915) carefully studied the topography of the trunk of the body in cerebral hemianesthesia, and found two or three parallel zones of different intensity of anesthesia; the more distal, the more severe (Fig. 21.13 left). Goldstein (1915) later confirmed this arrangement of the disorder (Fig. 21.13 right). The distal predominance of the disorder has also been evidenced by different authors, such as Kleist (1934) and Foerster (1916). The *privilege of the midline,* i.e., its lower intensity of alteration as opposed to the greater distal vulnerability is an important issue, just as in vision is the preservation of the macula in cortical hemianopsias and in the concentric reduction of the visual field. Both phenomena are aspects of the same process for both vision and touch, which

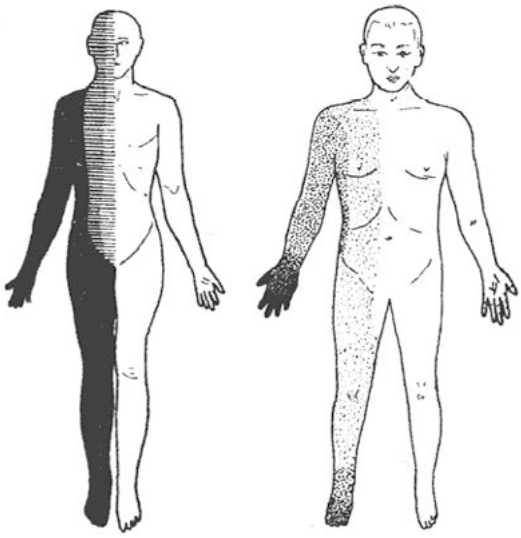

Figure 21.13. Residual cortical hemianesthesia. On the left, after Redlich (1915); on the right, after Goldstein (1915). Note the greater distal involvement, and in the figure on the right, the intact midline.

poses an insoluble problem within the theory of anatomical localization, but not so within our dynamic conception, as we know. Kleist (1934), for example, tries to interpret anatomically the aforementioned type of disorder by assuming that the bilateral midline innervation would gradually diminish towards the periphery, accentuating the functional deficit of the more distal areas, although he also believes that the hand and foot suffer more because of their greater differentiation. Other authors also go no further, such as Foerster (1916) and also Goldstein (1915).

Without entering into a detailed discussion of the various views, it is worth noting the difference in clarity and simplicity between either of the two hypotheses, localist or diffuse biological, and the dynamic physiological interpretation. This could already be illustrated in a hemianesthesia. For concentric reduction by unilateral lesion, as in our cases, both in sight and touch, any explanation outside brain dynamics fails completely. Bourguignon (1933) finds that already in the normal subject, the chronaxie for pressure increases from the center to the periphery of the body, behaving similarly to the chronaxie of the underlying muscle groups. Thus, the situation is entirely similar to that of the visual field whose sensitivity decreases from the center to the periphery. Pathologically, the alteration is more pronounced in the periphery because the less excitable function is affected in a greater proportion. Thus, concentric reduction as well as greater distal involvement appear as a direct consequence of dynamic reduction according to a very simple physiological process, without the need to resort to complicated anatomical hypotheses that have never been experimentally corroborated.

21.3.3. Structural change of the tactile sensory field

As we have seen, the phenomenon of irradiation as well as the narrowing of the sensory field evolve in unison (compare Figs. 21.10 and 21.11). It shows the profound *structural change of the tactile sensory field* with the reduction in the organization of phenomena, physiologically explained by the asynchronism between functions that are united in a normal situation.

The pathological alteration is for the entire field, and dynamic reduction is manifested in concentric narrowing of the field by virtue of the physiological privilege of the medial zone, as well as in spatial dedifferentiation. Since the central or proximal area of the body is more sensitive, excitability arises first in this area, and spreads peripherally as functional capacity improves. However, since spatial localization of stimuli requires a higher functional level, absent in peripheral areas, stimuli are diverted to medial areas of greater sensitivity. In other words, the abnormal interval between the sensitive field and the spatial field becomes evident. This means a functional dedifferentiation, also corroborated by tactile irradiation, since spatial specificity is altered by both deviation and irradiation. In short, the *residual field* is a narrowed field due to deviation, and a lax field due to irradiation.

It should also be noted that the cohesion of the body schema, i.e., of the tactile spatial field, may be poorly determined since, in addition to the narrowing, irradiation (on the surface and in depth) tends to blur the cutaneous aspect of localization in such a way that the body itself feels malleable, with diffuse boundaries.

Functional reduction indicates, as in other cases, a tendency to homogeneity in phenomenology, in contrast to the functional diversity of the normal sensory field (Fig. 21.14). Thus, irradiation tends to fill the field and not to stand out as a point. The narrowing of the body schema leads to the elimination of spatial values for a large part of the field, reducing the complexity of the spatial organization. An extreme case of homogeneity is the band of maximum irradiation without any spatial reference (phase I). However, a certain structure or influence of the tactile field always persists. For example, in phase I, the shape of the irradiation (as a band) is like that of the tactile field (elongated).

Other aspects of this topic pertain more to the theory of localization and of spatial function in general, and although they are not very different from what is described above, they will be dealt with in another section.

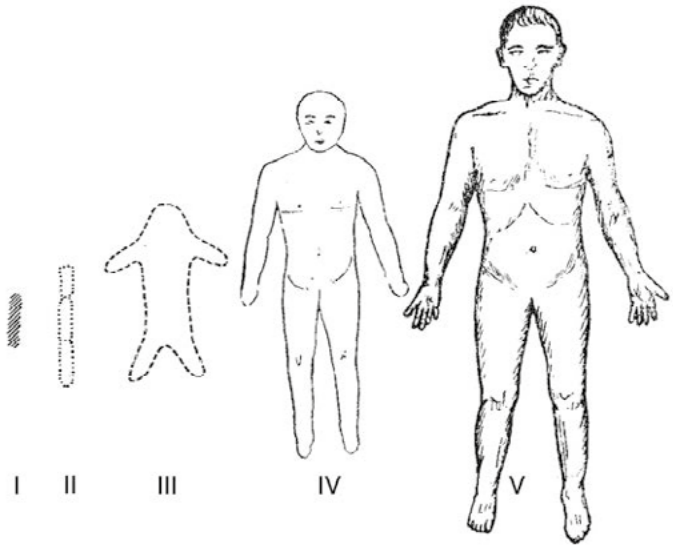

Figure 21.14. Reduction of the body schema according to the spatial phases. In phase IV, the distal zones of the limbs are excluded. In III, there is only an anterior plane of embryonic morphology. In II, the schema is filiform. In I, there is no representation. As a whole, there is concentric reduction of the tactile field and functional dedifferentiation.

22. Localization dynamics

22.1. RECRUITMENT OF PHASES IN THE LOCALIZATION PROCESS

After having exposed in detail the phenomenology of spatial asynchrony (or lag), as well as its fundamental characteristics, we can now undertake the study on the dynamic relations of excitability involved in the localization process. These relations describe the fundamental experience in the process of tactile localization by means of recruitment, the asynchronous beam, etc., relations already mentioned in the general part of Vol. 1 and which were applied to several visual functions such as visual orientation, etc. As we know, such quantitative data are what give the most peculiar character to the brain dynamics presented here, allowing us to establish the physiological laws that govern the different brain functions.

Within the experimental possibilities, the curves to be studied refer mainly to recruitment processes and to the asynchronous beam of partial (out-of-phase) functions, using either a proper mechanical stimulus or electrical stimulation by capacitor discharge. Tactile measurements cannot in many cases reach the precision of measurements of other sensory functions, since the phenomena of irradiation and proximal deviation involved in tactile localization are difficult to measure accurately. Nevertheless, the data obtained in various tests are sufficient to give an idea of the localization process with both mechanical and electrical stimuli. Altenburger (1933) already warns of the difficulty of performing tactile chronaxie determinations under normal conditions by experienced authors; even more so for pathological determinations.

22.1.1. Recruitment with single stimulus

By intensifying the stimulus, the functional reduction diminishes, and a sensory growth is obtained as a result of the recruitment of abnormal sensory intervals. Irradiation and proximal deviation of tactile localization then decrease simultaneously and correlatively, as we know. However, this is not an obstacle to inde-

pendently consider these two phenomena, as will be done below, starting with irradiation.

Figure 22.1 shows the functional relationship between stimulus intensity and irradiation of the cutaneous sensation in subject M inactive, under gradual point stimulus on the dorsum of a hand. It is observed that with the initial increase of the stimulus, irradiation decreases rapidly and then more and more slowly. The different phases already described thus appear, i.e., irradiation in the form of a band, a smaller band with central point of sensation, an oval, a very small oval, and finally an almost pinpoint sensation. The data are mean values of several measurements. As already indicated, the extent of irradiation can only be appreciated indirectly, the patient noting down the extent he feels on a ruler. Thus, the curve shows the transition from an irradiation of 20 cm or slightly more to the minimum possible irradiation, in the inactive state and using a very strong stimulation. It seems impossible to rule out certain tactile irradiation residuals, difficult to evaluate accurately and which probably measure less than 1 cm.

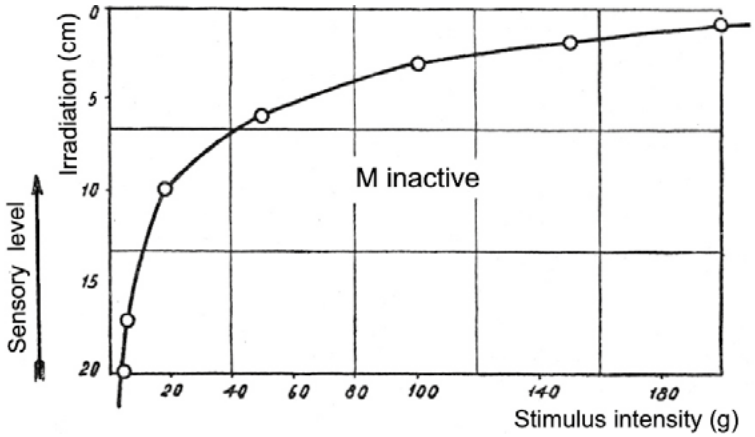

Figure 22.1. Tactile irradiation (in cm) as a function of the stimulus intensity (in grams). Point stimulus applied to the dorsum of a hand of patient M inactive. The arrow indicates the growth of the sensory level, which is inverse to the growth of irradiation. Note that it is not possible to reach zero irradiation in the inactive state.

This curve follows the general behavior of sensory recruitment (e.g., in visual orientation). Taking the logarithm of the stimulus intensity, a slightly sigmoid line is obtained that approximates a straight line, showing the proportionality between sensation and logarithm of the stimulus (Fig. 22.2).

As already indicated, tactile irradiation and irradiation in vision are similar phenomena, and the recruitment curve for tactile irradiation could also be applied to visual phenomena. Due to the inherent characteristics of irradiation in vision, a quantitative experimentation sufficiently complete to be expressed graphically is not possible.

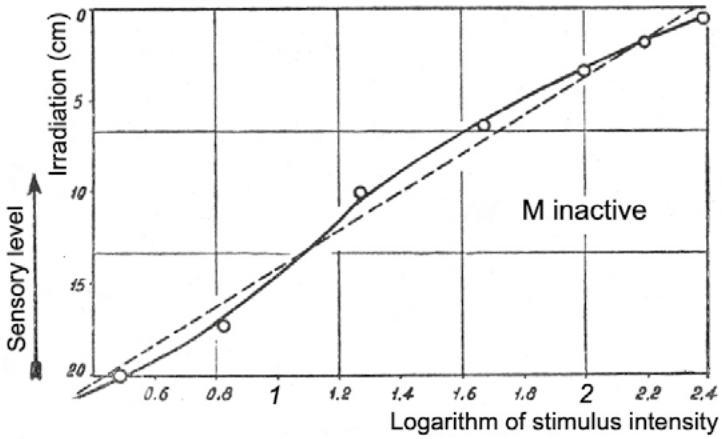

Figure 22.2. The same data as in the previous curve (Fig. 22.1), but taking the logarithm of the stimulus intensity. Almost a direct proportionality is obtained between sensory growth and the growth of the logarithm of the stimulus (Fechner's law).

The other important aspect of localization disorder is that of proximal deviation, which is the most interesting and complex aspect due to lateral inversion, body schema, etc. The functional relationship between the degree of deviation and stimulus intensity gives rise to a recruitment curve. To obtain this type of curve, lateral inversion has been ignored, indicating the distance as if the localization were always on the same side of the body, thus obtaining the curves shown in Fig. 22.3. The location of the perceived sensation cannot be determined as precisely as, for example, the degrees of tilt of the visual image. It is necessary to stick to verbal indications that allow only an approximate measurement ("in the middle of the arm," "towards the shoulder," etc.) or to proceed to indirect determinations on body models or using other aids. Nevertheless, the results thus obtained after several tests are good enough to illustrate the process.

Figure 22.3 shows the process in subject M, both in the inactive state and under facilitation, using a practically point stimulus on the dorsum of a hand. The mechanical stimulus in this experiment is somewhat less punctiform than in the one corresponding to the previous curves (Figs. 22.1 and 22.2), and that is the reason for a small difference in thresholds. The curve is of the same type as the previous one on irradiation, although it is not so perfectly simplified by taking the logarithm of the stimulus, which is attributable to the peculiar experimental difficulties. As shown in Fig. 22.3, proximal deviation and extent of irradiation evolve together, as can be seen in the corresponding scales. For any other part of the body, a similar recruitment must be admitted, but by choosing a distal part of a limb, there is both a wider field for measuring the deviation and an easier reference to indicate the site where the abnormal localization is felt. On the head and even more so on the trunk, many difficulties would arise for a measurement of this type; in the first case, because of the small size, in the second, because of the difficulty in finding references.

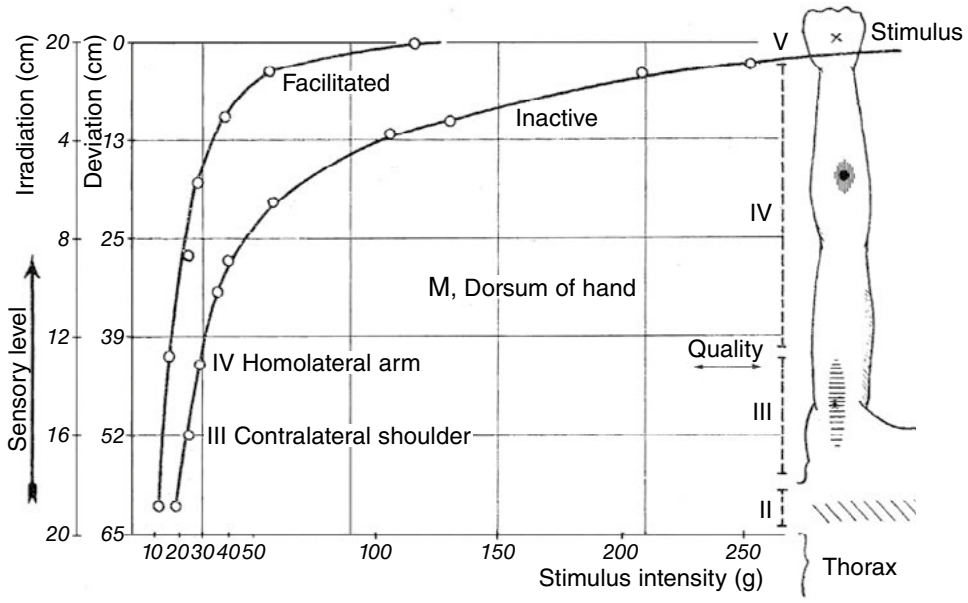

Figure 22.3. Curves of proximal deviation for subject M in the inactive state and under facilitation by strong muscular effort, as a function of the intensity (in grams) of a point stimulus on the center of the dorsum of a hand. The deviation (in cm) is the distance between the real stimulus site and the subjectively felt site. The irradiation scale (in cm) allows to appreciate the parallel behavior of irradiation and deviation. The indication of the phases (from II to V) and of the body anatomy facilitates the understanding of the process. Note the different slopes of the curves in the inactive state and under facilitation, as well as their positions.

Since these measurements are susceptible to error, it is convenient to indicate the verbal method for obtaining the data, as shown in Tables 22.1 and 22.2. All these data are the mean values of the data obtained in several tests performed under as equal conditions as possible, using mainly the subject's verbal information to obtain the degree of deviation. This method seems to be the most appropriate given the patient's characteristics. In such tests, only the sensory level (deviation and irradiation) and the intensity of the stimulus are involved, whereas the stimulation time is sufficiently long (unlimited time) and is not taken into account, as in Fig. 22.1. For a given stimulus, the sensation reaches steady state in about seven seconds in the inactive state, and in about four seconds under facilitation. Even the first phases can be obtained under facilitation with a permanent stimulus if its intensity is suitably reduced. (See curves of the asynchronous beam further on.)

In Figure 22.3 we can appreciate the different thresholds and slopes of the curves for the inactive state and the state under facilitation. Given the rapid initial growth of the sensory level, the first phases of localization show extremely small transit intervals and can only be individualized by very careful experiments. For this reason, phases II and III may initially remain unnoticed. By contrast, phase IV, which corresponds to a slow sensory increase, is very large and easy to obtain

Table 22.1. Subject M, inactive state. Experiment of Fig. 22.3. Stimulus applied for 7-10 seconds.

Stimulus intensity (g)	Phase	Sensory deviation from application site (cm)	Sensory irradiation (cm)
16	I		~ 22
19.5	II	~ 60 (Chest)	~ 19
25.5	III	~ 52 (Contralat. shoulder)	~ 15 Mild pain
27	IV	~ 42 (Homolat. mid arm)	~ 12
30	IV	~ 30 (Toward elbow)	~ 9
39	IV	~ 27 (Upper third forearm)	
59	IV	~ 19 (Mid forearm)	~ 7 Oval shape
109	IV	~ 12 (Two fingers above wrist)	~ 6
130	IV	~ 10 (Wrist)	~ 3 – 4 Strong pain
209	IV	~ 5 (Below wrist)	~ 2
250	IV	~ 1.5 –1 (Still lower)	~1.5–1 Very strong pain

Table 22.2. Subject M under facilitation by maximum muscular effort. Experiment of Fig. 22.3. Stimulus applied for 4 seconds.

Stimulus intensity (g)	Phase	Sensory deviation from application site (cm)	Sensory irradiation (cm)
10	I		~ 20
12.5	II	~ 60 (Chest)	~ 17
17	III	~ 40 (Homolat. mid arm)	~ 12 Mild pain
26	IV	~ 27 (Upper third forearm)	~ 8
29	IV	~ 19 (Mid forearm)	~ 6 Oval shape
40	IV	~ 11 (Wrist)	~ 4
59	IV	~ 3 (Hand)	Like a lentil
109	V	Without deviation ?	~ 0 ? Bearable pain

even in the T case. Although the first two phases can be obtained in M under facilitation, phase III seems impossible to be appreciated, at least with maximum facilitation, given the sharp slope. The patient suddenly passes from phase II to the onset of phase IV, and contralateral deviation is never obtained, the first two phases remaining very close to the onset of phase IV. It also happens that phase IV is much shorter under maximum facilitation than in the inactive state, about half, and since the asynchrony is lower, the abnormal phases are much more reduced. During this sensory recruitment, the intensity of the sensation also varies, which at first is like a weak pressure, and with much greater stimulus the sensation is painful, presenting a highly increased tango-algic interval, as we already know. The sensation of pain appears towards the end of the third phase in the inactive state, and at the beginning of phase IV with facilitation, in both cases the localization is approximately towards the middle of the arm. With facilitation, the curve reaches the normal localization (phase V), but in the inactive state, some abnormal residual remains which seems impossible to overcome no matter how much the stimulus is intensified.

From the arrangement of the curves in Fig. 22.3, we can appreciate the stimulus saving involved in the facilitated state. However, the normalizing action of facilitation is limited, and by using a stimulus to obtain phase I or II in the inactive state, the action of facilitation only makes possible to reach the onset of phase IV. This means that a stimulus that is not at all localized, or localized on the chest, moves to the elbow or more distally, i.e., from about 60 cm of deviation it moves to 30 cm of deviation, and sometimes less.

This type of recruitment was not determined in subject T, but given his characteristics, it can be admitted that his curve would be above that of M with facilitation and with a greater slope (see further on).

22.1.2. Recruitment with iterative stimulation

Given the high iterative capacity of subject M, another type of spatial tactile recruitment experiments can be performed by iteration instead of using a single stimulus. The curves in Figs. 22.4 and 22.5 correspond to this type of stimulation according to the number of stimuli and the interval between stimuli, respectively. For iterative stimulation, electrical stimulation is more precise and appropriate. The active electrode of other tests is applied on the back of a hand, and the characteristics of the excitation are conveniently chosen to obtain the most evident effect possible. Figure 22.4 shows that iterative recruitment in the inactive state is very large, but while achieving a much greater effect than facilitation with a single stimulus, complete recruitment is not achieved, although it comes very close to it. This experiment begins already in phase II with a single stimulus to have a higher sensory level. Using very short stimuli in relation to the subject's excitability characteristics, it is necessary to have a very high voltage. For an inter-stimulus interval of 1/12 second, the proximal deviation decreases as the *number* of stimuli increases, first rapidly and then very slowly.

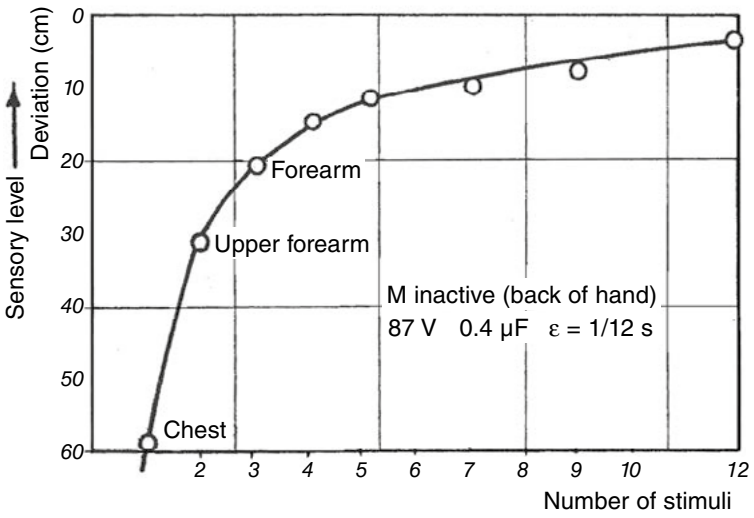

Figure 22.4. Iterative recruitment of spatial localization, i.e., decrease of centripetal deviation as a function of the number of stimuli in M inactive. The characteristics of the stimulation (voltage, capacitance and time interval, ε, between stimuli) are shown in the figure.

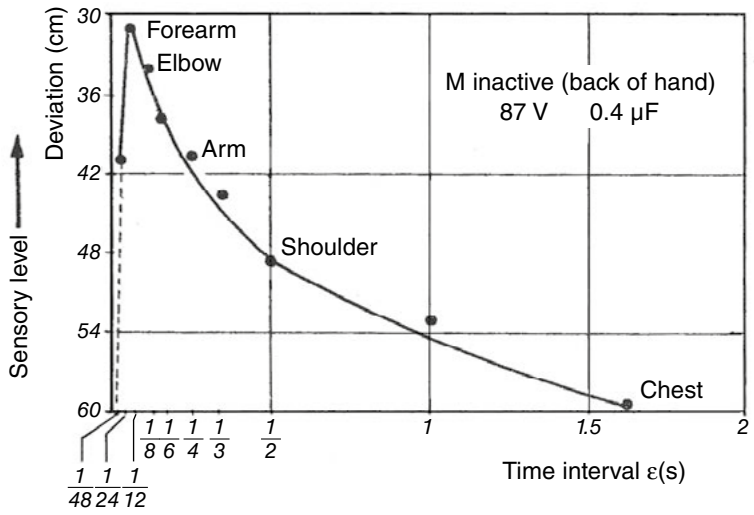

Figure 22.5. Spatial recruitment (localization deviation) as a function of the time interval (ε) between two stimuli. Intensity and duration of a single stimulus as in Fig. 22.4. The maximum effect of latent addition occurs with an interval of 1/12 s, becoming null when the interval is about 1.5 s. With intervals shorter than 1/12 s, recruitment tends to decrease, since the refractory period of the first stimulus should intervene, making temporal summation difficult.

With two stimuli, the sensory growth is so strong that phase III of contralateral localization is eliminated, and the sensation shifts from the chest (phase II) to the homolateral elbow or lower (phase IV). After a number of stimuli (12 or more), the limit of sensory recruitment is reached, and a residual asynchrony remains that cannot be overcome. If the experiment were started at the threshold corresponding to phase I, the final sensory level would be even lower and localization would not go beyond the wrist.

A complement to the previous experiment is obtained with the test corresponding to Fig. 22.5, with the same electrical excitation conditions as in Fig. 22.4 but now the sensory level is a function of the time interval between two stimuli. The highest point of the curve corresponds to the sensory level for an interval of 1/12 s, and the localization is toward the upper part of the homolateral forearm, as in the preceding Fig. 22.4. However, as the time interval increases, recruitment becomes less and less, passing through the contralateral phase III, obtained in its full amplitude, until reaching the chest (phase II) with the same effect as with a single stimulus. Due to the exponential decay of the excitation of a single stimulus, if the interval ε increases, the second stimulus adds to a smaller and smaller residual of the excitation of the first stimulus that seems evident ends after about 1.5 s in inactive M. If the interval ε is reduced to less than 1/12 s, the sensory level does not increase but remains constant or rather tends to decrease. Since the iterative cylinder (the mechanism used in these tests) does not allow rigorous control of intervals of about 1/48 s and smaller, it has been impossible to properly study the effect of very brief intervals.

In any case, it can be admitted that the decrease in the degree of summation with extremely short intervals is due to the fact that the second stimulus falls on the refractory period of the first stimulus (relative refractory period). It should be noted that as the chronaxie increases, the refractory period increases as well, thus, within the uncertainty of the present curve at very small intervals, it would perhaps be possible to estimate the refractory period for electrical stimulation at around 1/24 to 1/50 s on average. This interval-dependent summation test is similar to that of Bremer (1930 a) by exciting the nerve of a muscle under the moderate effect of curare, which confers to the muscle the condition of an absolutely iterative organ. Depending then on the interval between only two stimuli, the contraction recorded varies reaching a maximum and two minima (null contraction). One of these minima is for a very small interval (absolute refractory period) and the other is for a large interval (addition time limit). Other methods have been adopted by different authors.

In relation to the effects of iteration on tactile localization recruitment, it is worth noting briefly the *mechanical vibratory stimulation* with different tuning forks in subject M. A normal subject perceives the vibration of a 512 Hz tuning fork applied to the styloid process of the radius for at least 8-9 seconds, after having strongly struck the tuning fork. However, in subject M, due to his excitability deficit which increases the refractory period thus discarding high frequencies, it is necessary to use a 32 Hz tuning fork and apply maximum facilitation to obtain a similar result (see Table 18.1). This means that only low-frequency tuning forks can be used to obtain sufficient summation effect. The number of stimuli is not taken into account

(it is unlimited), but instead the time interval between stimuli is decisive, as in the electrical stimulation experiment shown in Fig. 22.5. Thus, the sensory level (here localization phase) depends on the tuning fork frequency used, as shown in Table 22.3.

Table 22.3. Subject M. Localization depending on vibration frequency of tuning fork on the styloid process of the radius.

Frequency (Hz)	Inactive state		With facilitation	
	Phase	Localization	Phase	Localization
32	IV	Upper third forearm	IV	~4 cm above wrist
64	IV	Upper third forearm	IV	~4 cm above wrist
128	IV	Elbow or higher	IV	Forearm
256	III	Contralat. shoulder	IV	Middle homolat. arm
435		No sensation	II	Chest (fusion)

A tuning fork of 32 Hz in strong vibration is perceived tactilely for 7-8 seconds by subject M in the inactive state, although only half of that time corresponds to vibratory sensation (see Figs. 18.1 and 18.2), which appears in phase IV of localization (upper third of the forearm); the rest of the time, he only feels stimulus fusion, hence some pressure uniform in time, and spatially, he does not go beyond phase II or less.

From the data in Table 22.3, it can be seen that the recruitment of localization by means of tuning forks is very small, which indicates that the frequencies, even being in the low range, are too high for the excitability characteristics of subject M (increased chronaxie and therefore increased refractory period); therefore, only under facilitation by maximum muscular effort and with the 32 Hz tuning fork, the proximal deviation is reduced to a minimum. For higher frequencies (i.e., very small interval), the proximal deviation is very large, especially in the inactive state. The decrease in sensory recruitment should then be understood as an effect of the refractory period, which reduces or prevents iterative summation. At least for the inactive state, it is presumed that the tuning forks indicated in the table act within the relative refractory period, and the last of them (435 Hz) even in the absolute refractory period. Thus, there can be no latent addition or temporal summation of any kind, and the sensory effect is null since not even phase I of spatial localization is reached.

Vibratory sensation appears at the onset of phase IV, the presence of sensation during phase III being questionable since a weak vibration seems to be initiated in the contralateral shoulder in inactive M at 256 Hz. In short, localization worsens as the vibration frequency increases, also increasing the perceived vibration speed which is always greatly overestimated (Fig. 18.1). Thus, the greater the tendency to fusion, the greater the alteration of spatial localization, i.e., the greater the deviation.

22.2. ASYNCHRONY IN SPATIAL LOCALIZATION

To complete the study of asynchrony, let us analyze the strength-duration curves referring to the most significant phases of the abnormal localization, i.e., the asynchronous bundle of curves corresponding to the different sensory levels. The determination of these curves is much easier with electrical stimulus than with mechanical stimulus, given the need to measure time.

Figure 22.6 shows the strength-duration curves for two quite different spatial localization phases in subject M inactive: phase I, without localization, and phase IV of proximal deviation. Comparing these two curves with those corresponding to M under facilitation (Fig. 22.7), it can be seen that in the latter case the curves have lower values, i.e., the excitability for these phases has increased, and the interval between them is markedly reduced.

From the chronaxie values in Figs. 22.6 and 22.7, it can be noted that the chronaxie interval with facilitation (0.7 μf) is one third of the chronaxie interval in the inactive state (2 μf). We also see that the approximation of the levels (phases) to each other is obtained mainly at the expense of the increased excitability of the most out-of-phase sensory level (phase IV). It is important to insist on the action of facilitation on asynchrony, since it reveals the physiological mechanism of the nervous

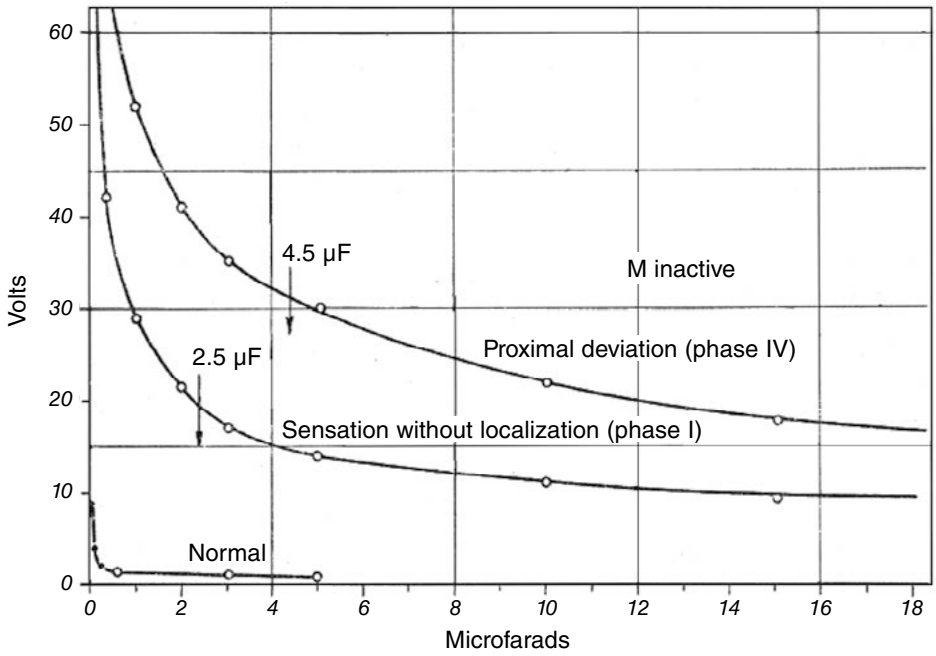

Figure 22.6. Strength-duration curves for phase I of localization and for the onset of phase IV, in subject M inactive. Lip stimulation according to the characteristics of previous electrical tests (Figs. 17.1 and 17.2). Note the large excitability interval (asynchrony). The lowest curve correspond to a normal subject.

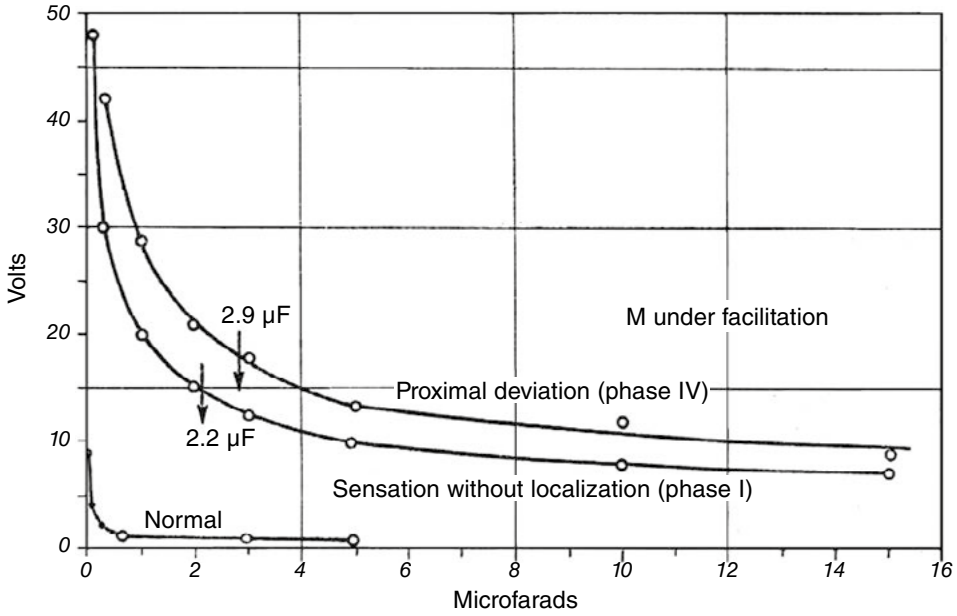

Figure 22.7. Strength-duration curves for phase I of localization and for the onset of phase IV, in subject M under facilitation by maximum muscular effort. Note the decrease in the chronaxie values of both curves, mainly of the upper one, which results in a remarkable closeness of the phases to each other reducing the interval between them. Test conditions similar to those in Fig. 22.6. The lowest curve correspond to a normal subject.

alteration, which consists in the fact that the higher levels become more altered than the lower levels as the excitability deficit increases. In both Figs. 22.6 and 22.7, as in most tactile stimulations by capacitor discharge, the lip has been used since it allows the best voltage utilization. The curves for simple contact (phase I) are therefore the same as those in Fig. 17.1 on general excitability. Likewise, the curves of the higher sensory level (phase IV) in both Figs. 22.6 and 22.7 correspond to the initiation of the tactile sensations, which show a tango-algic interval with respect to those of the lower level, being the same curves as those in Figs. 19.1 and 19.2.

As can be seen from Figs. 22.6 and 22.7, facilitation tends to convert the simple sensation curve (phase I) of the inactive state into a curve of proximal deviation (phase IV), as indicated in previous sections about the change of sensory level by facilitation. The sensory level only rises until the beginning of phase IV, with a wide margin of asynchrony still remaining and therefore being far from reaching normalization. The subjective localization corresponding to the described phases when the labial mucosa is electrically stimulated is as follows. For phase I, no localization as we already know; for phase IV, the localization is towards the temple or at most on the upper part of the cheek on the same side of the stimulation (stimulation on the labial commissure), therefore with marked deviation towards the vertex.

A more complete display of the asynchrony in patient M is shown in Fig. 22.8, comprising from phase I to the end of phase IV, which is all that can be determined

in M inactive. To the two curves of the phases previously studied, the highest and final curve is now added, which is very close to the normal localization, corresponding to the lower part of the cheek, close to the stimulation site on the lip and with a weak irradiation. The separation between this high curve and the one corresponding to the onset of phase IV determines the extent (amplitude) of phase IV.

The *abnormal sensory interval* from phase I to point localization is of course much wider than that from phase I to the onset of phase IV. Phases II and III are not represented in Fig. 22.8 since this figure as well as Figs. 22.6, 22.7 and also 22.9 for subject T were determined in the first half of 1944, when we were still unaware of the existence of phases II and III, whose values, differing little from the adjacent phases, made the identification of these intermediate levels difficult.

From the set of curves in Fig. 22.8, we note that whereas in a normal subject an all-or-none effect is obtained (localization without irradiation nor deviation, or total lack of sensation), in M inactive, the normal function is decomposed, according to the stimulation, into *partial reactions* (phases) with a marked deficit of excitability. This deficit is considerably accentuated for the partial phases of higher functional level, as already mentioned. Thus, with respect to the chronaxie for normal function, the chronaxie for simple contact (phase I) is about ten times greater, for the onset of phase IV it is twenty times greater, and for the end of phase IV about fifty times

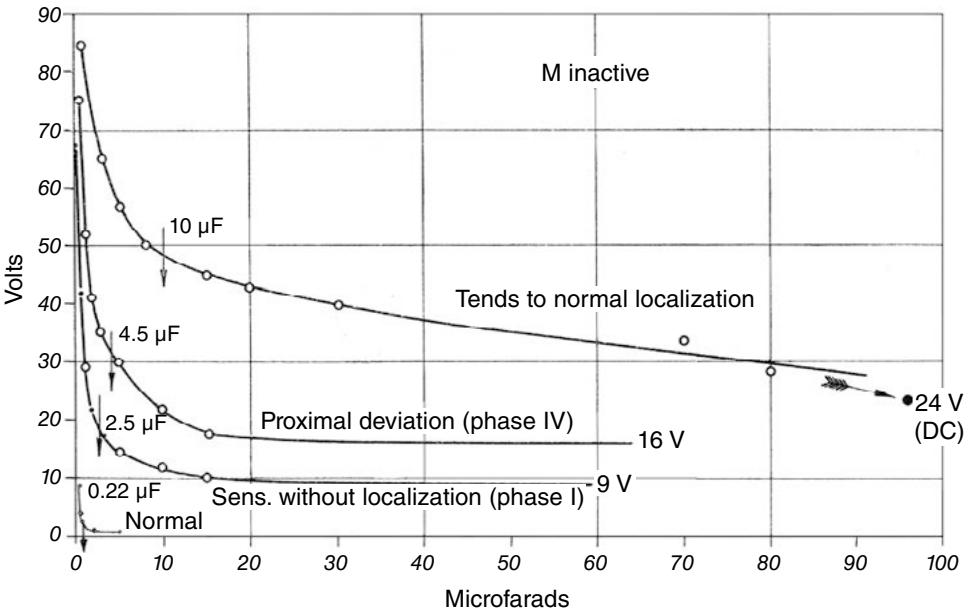

Figure 22.8. Electrical stimulation on the lower lip of M in the inactive state. Strength-duration curves for the different phases of spatial localization. The separation between the lowest and highest curves of M indicates the degree of asynchrony. The separation between the two higher curves indicates the extent (amplitude) of phase IV. Note the diversity of values compared to the curve of a normal subject, which includes all phases in itself.

greater. As for phase V (total normalization), the increase would be indefinite since the asynchrony in M inactive cannot be completely overcome.

Although a direct comparison of excitability values between Fig. 22.8 (strength-duration curves) and Fig. 22.3 (sensory recruitment as a function of stimulus) is not possible since the stimulation is at different sites and of different nature, the correspondence regarding the amplitude of the different phases is remarkable.

Subject T was also examined in this respect in early 1944, and the asynchronous curves of Fig. 22.9 were obtained. Although asynchrony in T is much less than in M inactive, it is sufficient to determine electrically different phases.

In subject T, the contact phase I without localization can be individualized even though it is more excitable than in M with facilitation, and the separation between the phases is small in comparison with M inactive. In T, normal localization is easily achieved, as in M under facilitation. In M, reversed (contralateral) phase III is absent, at least with maximal facilitation (Fig. 22.3), and should also be absent with more reason in subject T although he was not examined in this respect. Phase II, present in M under facilitation, would also be excluded in T because of very rapid sensory variation. Therefore, only phases I, IV and V were determined in subject T. The arrangement of the curves is approximately the same as in M; that is, the excitability deficit is greater in the higher sensory levels. Likewise, the onset of the well-defined sensations takes place at the onset of phase IV (see Fig. 19.3). It should

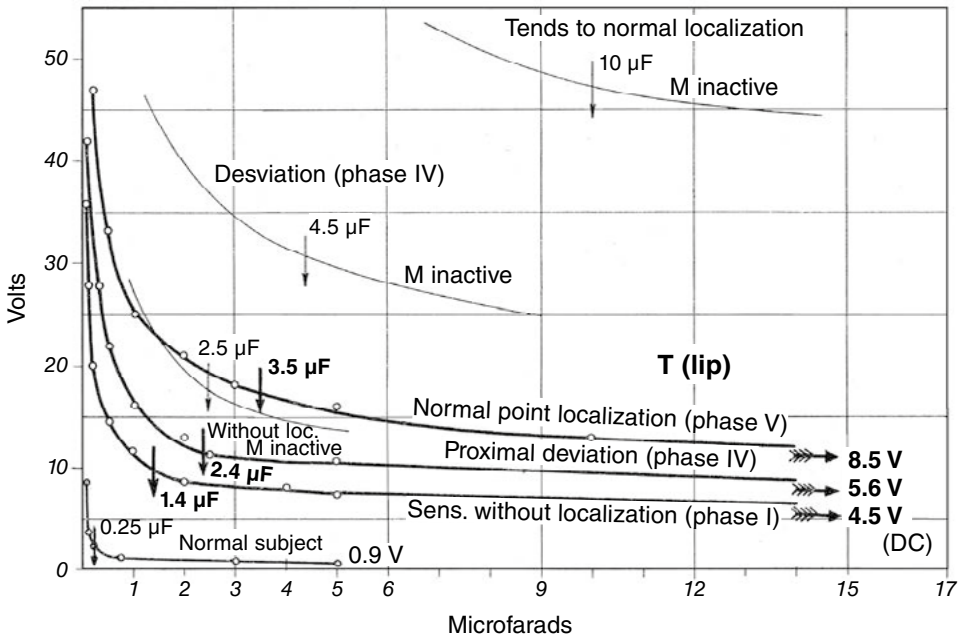

Figure 22.9. Strength-duration curves for the three existing spatial phases in subject T (thick lines). Experimental conditions similar to those of subject M. Compare with the curves for subject M inactive (thin lines), partially depicted.

be noted that the distribution of the sensory defect is not completely symmetrical in T, the right side (contralateral to the lesion) being somewhat more affected than the left. The data in Fig. 22.9 refer to measurements at the midline of the lower lip (to take a midpoint). The action of facilitation in T, although much weaker than in M, reduces asynchrony and also allows passage from phase I to phase IV.

Finally, it should be noted that subject T can show a much greater asynchrony as a consequence of epileptic seizures, as was indicated when studying vision. In such circumstances it was observed that his state of excitability was intermediate between M inactive and M under facilitation, tending to return to the usual state within a few days. Similarly, other modifications of excitability due to the effect of alcohol, or brain cooling, etc., already mentioned in relation to the tilt of the visual image, must also affect tactile functions for the same reasons as in the postictal state.

To conclude, a more complete representation of all possible phases in subject M inactive is given in Fig. 22.10, where the curves for phases II and III, difficult to determine in their entirety because they show values very close to each other, have been added. These latter curves were obtained after 1944, and the others maintained the values in the repeated tests up to that time. The figure shows the respective localization deviations on the head of the different phases when stimulating on the left labial commissure.

The curves correspond to the thresholds of the respective phases, that is, to the onset of the phases. The localizations indicated on the head are as precise as possible

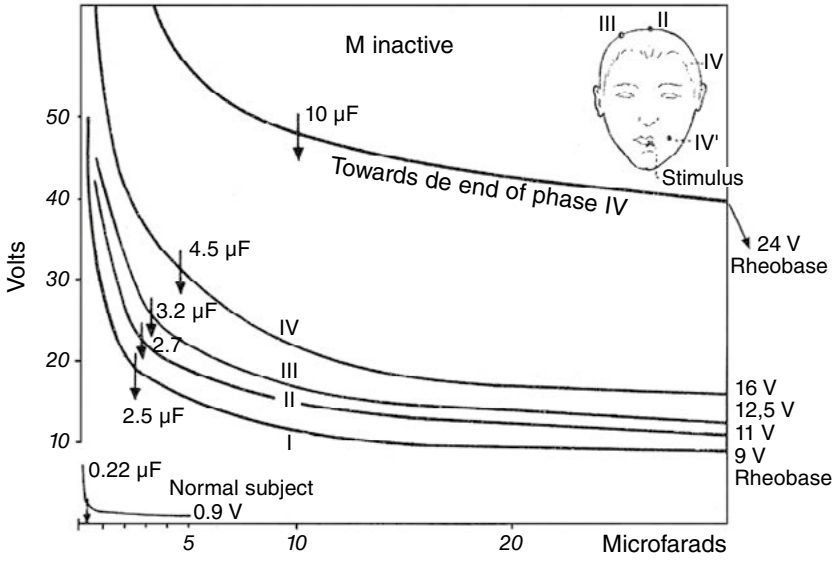

Figure 22.10. Strength-duration curves for all the phases that can be determined in subject M in the inactive state, stimulated on the left labial commissure. Note the great asynchrony and the deficit of excitability in relation to the curve of the normal subject, as well as the small differences between the lower phases (I, II, III), which make their determination difficult. On the upper right a diagram shows the localization of the different phases with deviation towards the vertex.

in an experiment of this nature. They are: for phase II, on the vertex or upper part of the cranial calotte; for phase III, on one side of the calotte, contralateral to the stimulated labial commissure; for phase IV, on the homolateral temple, at the limit of hair growth; and for phase IV (the highest sensory level attainable), the contact is perceived towards the lower cheek, close to the point of stimulus application.

22.3. TEMPORAL DEVELOPMENT

Some brief comments about time in both a) spatial sensory development and b) certain summation effects, will conclude the study on dynamic relationships.

a) Due to the slow reaction time, the development of a given sensory level can often be perceived through the different phases by the examined subject himself if he pays special attention to it. Thus, by using a weak but sufficiently intense stimulus to reach a well-developed phase IV, subject M inactive takes 1-2 seconds to reach phase I and a further 2 s to reach phase IV (a total of 4.5-5 s for phase IV). During this time, he can sense the change in the extent of irradiation and the change in localization. However, the transition from phase I to phase IV is abrupt, the intermediate phases being excluded. In order to obtain a more gradual development, it is necessary to resort to iterative stimulation and select the intermediate phases with a certain number of stimuli. For example, with mechanical stimulation by means of a piece of soft cotton, phase I is obtained after two or three successive contacts, phase II after the fifth or sixth contact, phase III after the tenth, etc. Undoubtedly, this type of development can be obtained more accurately and completely by electrical iteration, and we refer the reader to the corresponding curves presented in Sec. 22.1.2. In addition, the *temporal development of the spatial process* can be inferred from the curves of the different phases (especially from Fig. 22.10) for a stimulus of sufficient intensity to reach the highest sensory level. The reaction speed increases in M with facilitation, and is even higher in the T case. For ease of understanding, this can be related to the time evolution of visual image orientation (Figs. 13.9 and 13.10).

A reverse development can also be obtained by studying sensory degradation, either by the fatigue process with permanent stimulation, or by residual persistence after removing the stimulus. In both cases the results are quite similar. Using the minimum stimulus necessary to obtain a well-developed phase IV, and keeping the stimulus unchanged, it results that after approximately one minute of having reached phase IV, it disappears abruptly and leaves a sensation corresponding to phase I which is still maintained for a few minutes. Depending on the intensity of the high phase reached at the beginning of the test, the process during *fatigue* is different; sometimes the final phase I seems to be maintained indefinitely while other times it ends up disappearing, although much later. Fatigue progressively and gradually raises the excitation threshold, therefore the high functional level reached tends to present dynamic reduction through asynchrony.

The fading of the persistent sensory residue in the absence of stimulus occurs as follows. Once the stimulus that has allowed reaching a well-developed phase IV

has been removed, the sensation persist only for a brief moment, passing immediately and abruptly to phase I, being impossible to notice inversions and intermediate phases. The persistence does not last in total more than 2 seconds at most, and is on average about 1.5 s for the two phases mentioned above. In this process of temporal degradation, a certain *sequence of phases* is obtained which, as in the temporal development, is limited to the most accessible phases I and IV, given the brevity and instability of the process.

b) It is now worth mentioning the action of facilitation on the residual sensation once the stimulus removed, which causes the remarkable phenomenon of *post-stimulus summation*, already study in M since 1940 both in vision (effect on the tilt of the visual image) and in touch (spatial localization). The effect certainly takes place as long as the time interval between the removal of the stimulus and the application of facilitation (strong muscular effort) is very small (less than 2 s), otherwise the facilitation has no effect because the residual sensation has vanished.

Facilitation exerts a summative action on the persistent sensory residual and intensifies it for brief instants, raising it even to a sensory level higher than that produced by the stimulus. Of course, the degree of this increase is a function of the above-mentioned time interval, all other experimental conditions being constant. For example, when M inactive has a constant sensation of simple contact without localization (phase I) and then the stimulus is removed, the sensation lasts slightly more than 1.5 s. Thus, depending on the delay in the application of facilitation, different sensory levels are reached as shown in Table 22.4.

In this reactivation process, the time that the subject under examination takes from the moment he receives the order until he makes the muscular effort should also be taken into account; this time is usually less than one second and is somewhat variable according to the subject's degree of attention and other circumstances.

Table 22.4. Localization corresponding to the sensory level reached according to the delay (in seconds) in applying facilitation after removing the stimulus. The stimulus is a 19 g permanent weight on a 0.7 mm diameter surface on the dorsum of a hand of M inactive, triggering phase I only. Simultaneous application of maximal facilitation would result in sensation at the homolateral elbow (advanced phase IV).

Delay in facilitation (s)	Localization (sensory level)
0.1	Homolat. elbow (phase IV), same as no delay
0.2	Homolateral arm (onset of phase IV)
0.9	Contralateral shoulder (phase III)
1	Chest (phase II)
1.5	Only phase I
More than 1.5	No sensation

Table 22.4 shows that the sensory level decreases as the time interval between stimulus removal and the application of facilitation increases, since the sensory residual of phase I decreases rapidly until it disappears completely shortly before 2 s. For long time intervals (1 s or a little more), facilitation acts on a subliminal residual that is below phase I and, therefore, facilitation only manages to elevate the residual to phase II, or even lower, restoring phase I. It is understandable that in all cases the level reached is of short duration, since the tactile stimulus has been removed.

The characteristics of this post-stimulus summation by facilitation are very similar formally to those of the experience in Fig. 22.5 (sensory level as a function of the interval between two identical electrical stimuli) although the experimental conditions are different. In both cases a certain sensory level is recruited from the residual trace of a previous stimulus. Such a residual trace is activated either by a second identical stimulus or by facilitation. In both cases, the maximum time interval for the summation effect to exist is of the same order (about 1.5 s), and even the degree of sensory enhancement is very similar, despite the different type of stimulation in each case. The fundamental difference lies therefore only in the type of summation, direct for iteration (*temporal summation*) and indirect for facilitation (*spatial summation*), the effects of both being equivalent. Whereas in temporal summation the two stimuli are of the same nature and travel to the nervous centers using the same pathway, in spatial summation the two actions (stimulus and facilitation) not only follow different pathways, but in addition, facilitation involves a nonspecific central action. In short, the residual imprint of a first tactile stimulus can be activated, and even raised above its corresponding level, by very different nervous actions.

Finally, some brief comments are pertinent about facilitation as a synchronizing action of desynchronized sensory levels. The transition to a higher tactile sensory level by facilitation due to muscular effort has been dealt with descriptively and graphically (Fig. 22.3), and the analogy with the behavior of the visual function with respect to facilitation (enlargement of the visual field, correction of the orientation of the visual image, etc.) can easily be admitted. For this reason, we do not add for the moment the corresponding graph of tactile sensory growth as a function of the weights held by subject M, since it is a great inconvenience (although not an impossibility) to check how the subject, holding weights up to 80 kg, perceives an improvement in the sensation of a constant weak tactile stimulus. It is also possible to state that recruitment as a function of facilitation (by muscular effort) has similar characteristics to recruitment as a function of stimulus intensity, noting that synchronization by maximum muscular effort is not complete and there is always an important residual that seems to be much greater than in the case of stimulus increase.

Concerning other non-muscular facilitations, namely cross-modal actions, only touch (bi-tactile effect) is of some importance. Thus, a pronounced mechanical pressure on the skin, or brushing gently over the skin, reinforces the sensation awakened by a weak tactile stimulus elsewhere, raising its level, reducing the irradiation, etc. This fact is of particular interest when studying spatial discrimination between two stimuli, as will be seen further on. On the other hand, it is difficult to demonstrate the summation of visual and auditory facilitations on touch, although perhaps auditory facilitation is somewhat clearer, being issues that still need to be investigated.

22.4. THEORY OF TACTILE LOCALIZATION. SPATIAL ORGANIZATION

Although the theoretical issues will be developed in depth in the last part of this research, we cannot refrain now from making some comments on tactile localization in the light of all that has been said about our cases, as well as about the Schneider case of Goldstein and Gelb (1919) according to the interpretations of these and other authors. All this will help to specify the properties of *spatial organization,* properties that are deduced from our research.

Clinical neurology has contributed little to the elucidation of this issue, i.e., to the physiological mechanism of tactile localization. At most it is said (Head and Riddoch 1920, Head *et. al.,* 1920) that the diffuse or incorrect localization provided by the nerve fibers of protopathic sensitivity is improved and refined by the special nerve fibers of epicritical sensitivity. The issue is thus focused on the anatomical specificity of the conducting fibers which, besides being a problematic hypothesis, leaves the problem of the mechanism in question untouched.

It is in the domain of theoretical psychophysiology that this problem develops and where the theories most in conflict with each other are conceived, namely 'empiricism' or 'genetism,' and on the other hand 'nativism.' For the founders of empiricism (Lotze 1852, Wundt 1862, 1874, Helmholtz 1867/1896) the 'local sign' arises *secondarily* through the association of cutaneous sensations with muscular movements in the course of life, and visual representations of one's own body also contribute to it. This interpretation, more or less modified, reaches Henri (1898) and from there reaches Goldstein and Gelb (1919) who apply it to solve the problem posed by their Schneider case. In contrast to them, the nativist trend appears with Hering (1874/1878, 1880) and especially with Stumpf (1873), arguing that the spatial structure is *primarily* inherent to the sensation. Later, it became very significant the development and acceptance of the concept of extensity as an immediate phenomenic datum, considering it an attribute of sensation (Külpe 1893/1909, Titchener 1910, James 1890). Likewise, on the part of the Gestalt theory, Wertheimer (1912) and Koffka (1919/1935) clearly argue that extensity is as primary as tactile sensation, and that shape is a mere mental organization given in the extensity. As these orientations become more and more prevalent, there is a tendency to admit that the perceived space is in correspondence with the spatial relationships established in the nervous system independently of learning.

Turning our attention now to the examination of the facts, we shall first deal with the controversy about the Schneider case in relation to the pathophysiological problem of spatial localization, following the attempted interpretation of Goldstein and Gelb (1919) and other authors. The characteristics of this case are already known to us, and now we shall only refer to the way of achieving localization by means of particular muscular twitches, according to these authors. These authors distinguish between normal or "voluntary" localization and "automatic" or reflex localization, following Henri (1898). According to this author, the first type (voluntary) involves secondary auxiliary aids such as the visual representation of the touched place, which is never lacking, and a certain kind of attention ["genaue Beachtung der Tastqualität" Henri (1898)]. The second type involves movements to local-

ize the touching finger and contact sensations when touching with the finger. It is admitted that these localization movements have a congenital reflex character, seemingly of a spinal cord nature, providing only a very rough localization, corrected in part by the referred contact sensations which by trial and error guide the localization. Goldstein and Gelb (1919), without judging the obscure statement of Henri (1898), take advantage of the automatic localization to attempt an explanation of their case, in addition to the general conceptions (visual influence on tactile space, etc.) on which they base the tactile disorder. Thus, as soon as subject Schneider feels contact (perceives that he is touched), he automatically performs movements of very short amplitude, such as muscular twitches or small jeks, all over his body, reaching a moment in which, by colliding with the applied stimulus, the referred movements are more specifically directed towards it. The authors then think that a kind of "kinematic modality," completely hypothetical, must intervene, leading to a certain sensation of location, or rather a kind of guidance of the body, according to the authors.

In fact, the authors state that, even in the case of achieving localization, a tactile spatial structure is still absolutely absent. The same occurs in vision: during the movements around objects, a true visual sensation of shape would not take place. Thus, in both touch and vision, the function occurs indirectly but without being achieved in reality. Without going into details, it is clear that the authors' interpretation seems non-transparent or very arbitrary (qualitative specificity of the movements), and even contradictory (the patient can localize perfectly but lacking a spatial notion), as recognized without exception by all authors, psychologists, neurologists, etc. who have commented on this issue.

Goldstein and Gelb (1919) admit that tactile space takes place only through visual representations of the body supported in turn by movement sensations. This is the genetic point of view defended and most extensively developed by Henri (1898), whose origin goes back to Lotze (1852), Wundt (1862, 1874), etc. Goldstein and Gelb think that given the lack of visual space in Schneider, touch suffers in this subject a "transcortical alteration of sensitivity" which they claim to have demonstrated for the first time. Finally, ascribing a character of generality to their conclusions, they maintain that there is no space other than visual space. This opinion was formerly held by Platner (1793), revived by Hagen (1844) and defended again after the Schneider case. However, the consequences of this opinion for the congenitally blind, to whom all spatial perception would have to be denied, were impossible to be maintained even by Goldstein and Gelb who have attempted to establish a certain parallelism with their Schneider case. A fully recognized fact in this type of blind people is precisely the ability to sense tactile space, therefore independent of any visual influence.

Regarding the formation of tactile space in a normal subject, Goldstein and Gelb accept a pronounced empiricism, and thus, in children, only automatic localization would be present, which would gradually give way to voluntary localization. Therefore, during the first stage of their development humans would have a tactile state similar to that of the Schneider subject.

In the Schneider patient, it has been rightly reached to distinguish his different behavior in the two different states, that of rest (inactive) and that of muscular twitch-

ing. On our part, we know from our research that this can be explained, for vision as well as for touch and for any other activity, by simple excitability relations, and that muscular twitching is a central summative facilitation. This summative action can equally well be achieved by immobile muscular tension or by any other type of facilitation. On the other hand, the authors have lacked knowledge of the phenomenology of localization (stages according to irradiation and proximal deviation) which provides essential data to address the spatial problem.

Regardless of our findings, we have followed the course of the ideas of Goldstein and Gelb (1919) in their attempt to explain the Schneider case, to show that their explanation is not possible. Paying attention to certain particularities, the contradictions become even clearer. Thus, it seems that in the supposed automatic localization, spatial guidance is possible thanks to the hypothetical kinesthetic ability mentioned above, namely, to muscular movements that are guided towards the stimulus, which is casually found by the sensation of increased pressure. However, it is impossible that, normal localization being abolished, the ability to perceive joint movements and others still exists, since it is known (Head *et al.* 1920) that joint movements are much more severely impaired than any other spatial function.

The specific features of motion in touch will be studied later, but in Sec. 21.2 we saw that motion, either on the skin or involving joints, appears only weakly in phase III (inversion). Some peculiarities are also indicated in Sec. 21.1. If we apply this criticism on the perception of joint movement to the contouring movement around objects for visual perception of them, the explanation given by Goldstein and Gelb (1918) for vision is also problematic. As for the hypothesis about the specificity of those movements that seem to have the property of spatial localization, no fundament or effective proof has been provided. Concerning the way of achieving localization (or as it should be called according to the authors' thought), it is not understandable. For instance, the kinematic sensation would awaken in the subject the indication 'arm'; he does not know *where* the arm is but abstractly (?) he knows that it has something to do with the arm.

> Goldstein and Gelb (1919) incorporate normal localization into automatic localization, probably impressed both by the spasmodic muscular twitches of subject Schneider in order to localize and by the ideas of Henri (1898) which, in principle, seem to fit perfectly with the interpretation of the subject's behavior. However, they feel compelled to postulate in addition the existence of a "kinematic sensation" in the aforementioned muscular twitches, in such a way that some movements would be qualitatively different from others, that is, they would carry in themselves significance of location. This means admitting local sign for the muscular twitching. But then, why not admit in a primary way the cutaneous local sign, i.e., the static sensation, which is much simpler? According to Henri (1898), from automatic localization (a mere spinal reflex of a rough spatial character), there would be a transition to voluntary localization, more perfect, thanks to learning through experience by means of a functional transition that does not seem at all clear and, on the contrary, involves new requests of principle. In order to solve the situation, Goldstein and Gelb (1919) are forced to admit the new kinematic factor, as already mentioned. The importance of movements for the perception of sensory

space seems to them so decisive that they apply it also to visual recognition, although in this case it has to be done with tactile movements, i.e., by means of the tactile space which the subject in question lacks. All this entails many complications and new hypotheses at every step, to leave the theoretical construction finally unresolved.

In relation to the above, we shall briefly examine the matter of automatic localization. This is well known in animals (frog, dog, etc.) brainless or spinal. In the spinal frog, the stimulus of an acid placed on one side of the lumbar region provokes defensive contractions of the hind leg on that side, as if to get rid of the excitant. If the reflex is prevented by holding the referred leg, the defense reflex is produced in the hind leg on the opposite side. Similar behavior occurs in the spinal dog with respect to the scratching reflex elicited by a tactile stimulus on the animal's flanks (Sherrington 1906). Likewise, defense reflexes similar to the mentioned scratching reflex can be observed in humans with central affectations (Guillain 1905). Such automatisms in humans and animals are localized reflexes in conformity with the nervous connections in the nervous centers. They are therefore entirely native mechanisms, and thus independent of previous experience. In some brain-injured patient with acute disorders in tactile localization, we have been able to observe similar synergies when stimulating a severely hypoesthetic area, or in any case devoid of the sense of location; but without having made a sufficiently complete study, it is difficult to draw conclusions of any importance on this issue. In such cases a number of factors may come into play (allochiria, automatosis, diversity of response according to the intensity of the stimulus, etc.) which cannot be assessed in a superficial examination. The best studied example is the spinal automatisms, either in spinal man or in physiological preparations of animals. Such automatisms constitute segmental reflex activities that depend on pre-existing structures in the nervous system. They lack any sensory effect, and it does not seem possible to establish a transit towards specific or normal tactile localization. Already in subject M we have had occasion to observe that cutaneous stimuli that do not go beyond phase I of primitive projection (without localization in the body), may trigger reflex responses such as eye closure (see Sec. 21.2.1).

In our opinion, automatic (reflex) localization and normal specific tactile localization are clearly completely different activities that have no relationship or transition of any kind between them. Even rejecting any relationship between the two types of localization, it could be assumed that, if reflex responses are localized primordialy (in origin) due to the corresponding neural structures and connections, the same could be true for cutaneous localizations at a higher level of nervous organization. However, the structure of the sensory field is generated by the brain, both by the action of the nervous connections or cortical projections and by the action of the central mass which, by influencing the physiological level, determines the value of the spatial sensory dimension. Thus, in the central syndrome, spatial localization, whether tactile or otherwise, will have several stages with a sensory field reduced according to the amount of the lesion and the action of the pre-existing nervous connections. Therefore, the tactile field (body schema) will be more or less wide, and will be subjected to the spatial inversion effect due to the inverted cortical projection. The brain lesion gives rise to a spatial dedifferentiation until a point is reached where progressive loss of structure results in complete destruction of spatial localization, corresponding to phase I of spatial localization, as shown in our cases, especially subject M.

Turning now to other approaches, we must consider the theory of Stein (1928, 1930) and Stein and Weizsäcker (1926), which, in addition, is closely related to the studies on the Schneider case. These authors postulate that perceptions (sensations of spatial character) arise through the action of certain motor activities. It is clear that Stein (1928, 1930) seems to refer especially to what he calls, in a vague way, "virtual movements," and in such a case these movements correspond to the phenomena of Wertheimer (1912) and Benussi (1913, 1916) on apparent movement, which are rather an effect of the spatial structure and not its cause, as Stein thinks. If they are admitted as a support of spatial perception, they are an unnecessary complication, if not a theoretical misrepresentation, at least in our opinion. Apart from all this, we strongly disagree when he states that the Schneider case is explained according to his theory by a dissociation of movements and sensations. He also thinks that the most interesting finding of Goldstein and Gelb (1919) lies in the observation of the auxiliary movements that the patient uses to achieve visual and tactile perceptions. He therefore thinks that there must exist a certain primary relationship between virtual and real movements, and he even admits a genetic dependence between them. The child would start with real movements, then their virtual imprints would remain. Pathologically, there would be the corresponding regression.

Note that Stein (1928, 1930) seems to admit that Schneider's auxiliary movements do indeed allow him spatial perception, which Goldstein and Gelb (1919) reject since according to them the subject, with or without movements, would lack all visual and tactile space, and furthermore, they admit no space other than visual space.

The interpretation of Stein (1928, 1930) on sensory space differs in part from the previous ones (he also does not admit the visual influence on touch), but it is not satisfactory. The arguments and proofs he takes from the Schneider case, slightly changing its explanation to make it somewhat simpler, make his interpretation refuted by all that has been exposed of our research. Stein has been right in assuming the physiological basis of nervous excitability for sensory organization, but his questionable motor theory of perception, referring to and relying on the Schneider case, does not work. The same must be said, as far as the spatial question is concerned, of all the authors [Schilder (1923/1935) among others] who have been influenced by the conclusions of the Schneider case, or of this case and Stein at the same time, such as Buscaino (1946).

In summary, after reviewing the ideas on spatial theory, particularly tactile, we must reject, for being inconsistent, the theories of Henri (1898), Goldstein and Gelb (1919) and Stein (1928, 1930), who in one way or another have tried to interpret the Schneider case. All these theories are genetic, empiricist or associationist. For tactile sensations, Henri, and Goldstein and Gelb admit muscular movements and vision influence; and Stein admits movements and sensations for space in general. With regard to a hypothetical visual influence, it is a well-known fact that congenitally blind people have a perfect sense of tactile space, and even deaf-blind people have it too (such as Laura Bridgman and Helen Keller), therefore any sense can develop higher structures *directly* without any help from other senses.

The solution to the problem of the structure of the tactile space and localization of stimuli lies in the phenomena described above concerning asynchrony in tactile localization. The existing abnormal sensory interval gives rise, depending on the intensity of stimulation, to a series of spatial stages, completely ignored in the long study of the Schneider case carried out by Goldstein and Gelb (1919).

We already know that the fundamental phenomena of irradiation, proximal deviation, narrowing and inversion, are manifested through the different stages (phases), and such spatial phenomena must be admitted as *primary* or innate, i.e., inherent to the activity of the nervous centers, independently of genetic or learning mechanisms. Thus, irradiation means a reduction in the specificity of nervous reaction, being an expression of loosening of the local sign in the sensory field. The proximal deviation or concentric reduction of the field indicates a reduction in the sensory dimension, and the sensory inversion is a reflection or copy of the inverted projection of the sensory pathways in the cerebral cortex. In short, if the lesion causes a reduced brain activity, this results in a *residual function field* (irradiation, narrowing and inversion). It is not the time now to examine in detail the psychophysical basis of these phenomena; suffice it to point out that the sensory organization in the brain centers depends both on the connections (inverted projection) and on the action of the central mass (excitability, cohesion, sensory dimension, etc.). Thus, it is clear that the nature of the spatial phases is in *correspondence* with the pre-existing central nervous substrate, so we could easily connect with the psychophysiological views of the authors already mentioned at the beginning of this section, stating that the perceived space is in agreement with the spatial relations contained in the nervous system, independently of learning. The same can be said about certain very singular spatial alterations, such as monocular polyopia, polyesthesia, and also the formation of a pseudofovea in hemianopsia.

The phenomena studied reveal that the local sign (the spatial value), far from being an easily identifiable and isolable invariable entity, is subject to gradations that depend on the state of sensory organization. That is to say, a functional entity, of spatial type, or of tactile sensation or of any other type, does not exist by itself but its *individualization is due to sensory organization.* In any case, such functional entities are not primary elements, but on the contrary, functions that derive from a degree of complexity (structural development), and that starting from what is homogeneous and continuous, evolve towards heterogeneity and discontinuity, thus generating distinctness and individuality, as shown throughout the five phases of tactile localization.

A similar situation is found in the phenomenon of irradiation of conditioned reflexes, according to Anrep (1923). At the beginning of the development of a tactile conditioned reflex, a response can be elicited from cutaneous sites other than the point in question. There is then a certain area of irradiation, but as the development of the reflex is consolidated, the area of irradiation narrows, being almost reduced to the first stimulated site. It can then be said that the reflex is practically specific for a same receptor. In view of such behavior, it is possible to conceive that a stimulus influences the whole tactile field, since an area of irradiation arises which may cover the whole field;

> but as the central organization of the reflex progresses, the excited site tends to become specific for the reaction. It is thus clear that localization depends solely on the state of central organization, with total exclusion of any other factor.

In conclusion, spatial localization (spatial organization) presents very different phases in relation to the nervous state of central organization, that is, of the sensory field. This organization is intrinsic (innate), and therefore, alien to external circumstances.

The above is sufficient to give an overview of the thesis defended here, which will be studied more extensively in a third part of this research.

23. Spatial discrimination

23.1. SPATIAL ACUITY (WEBER)

The spatial discrimination of two points on the skin constitutes the tactile spatial acuity, similar to the minimum separable in vision. Such discrimination is, of course, conditioned in our subjects by the functional level of the phases of spatial localization (irradiation, deviation, etc.), maintaining close correlation with them (see Sec. 21.2.4 and 5.2.5). The characteristics of the stimulation are always decisive, as well as the central factor (facilitation).

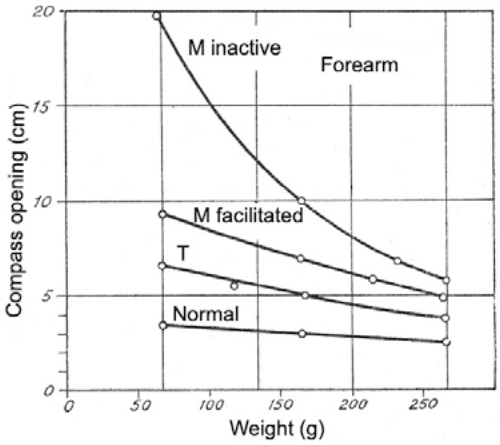

Figure 23.1. Spatial threshold (Weber's compass opening) as a function of stimulation intensity (compass pressure) on the middle part of the dorsal forearm, for M inactive, M under facilitation by strong muscular effort, subject T and normal subject. Note that the threshold hardly varies in the normal subject, whereas the variation is very pronounced in the other cases, mainly in the one with the highest deficit, i.e., subject M in the inactive state. Even under high pressure, the pathological cases show a threshold much higher than normal. Compare with Fig. 11.1 concerning vision.

In phase I there is absolutely no sensation of duplicity whatever the spatial distance between two stimuli. In phase II, duplicity may be possible, but in circumstances that are so special that it is not comparable to its usual character; in addition to the great instability it presents, it is necessary to stimulate regions of different medial representation (see Sec. 21.2.2). A more normal-like discrimination, but with a greatly increased threshold, corresponds to phase III, but the duplicity of stimuli is perceived very centripetally deviated and contralaterally inverted. For example, a stimulus on one hand and another on the elbow of the same side, 18-20 cm apart, are perceived as two contacts almost together towards the shoulder of the opposite side. It can be said that spatial discrimination appears towards phase III, in which, as we know, there is a certain rudiment of organization in the corresponding sensation (irradiation band with a weak central point).

Figure 23.1 shows the relationship between spatial threshold and stimulation intensity for subject M in his two main states, for subject T and for a normal subject. For the two extreme curves in the figure (M inactive and normal subject) the different behavior is clearly shown, i.e., when increasing the pressure of the two points of the Weber compass (Weber 1834/1846), there is a marked reduction of the threshold in M, and very little variation in the normal subject (see Fig. 11.1 concerning vision).

The compass pressure is then decisive in the pathological cases and especially in subject M, the most affected. Even using pressures that produce unbearable pain, the reduction of the threshold in pathological cases presents a limit much higher than normal values. The thresholds, deviations, phases and localization area reached by simultaneously applying the two tips of the compass are shown for subject M in Tables 23.1 and 23.2.

Table 23.1. Subject M in the inactive state, stimulated in the middle of the forearm. Simultaneous Weber threshold (in cm) as a function of intensity (in grams).

Stimulus Intensity	Spatial threshold	Phase, Area of localization
65 g	18 cm	III, Contralateral duplicity toward the shoulder
115 g	14.5 cm	IV, Above the homolateral elbow
165 g	11 cm	IV, Toward the homolateral elbow
215 g	8 cm	IV, Toward the homolateral elbow
265 g	7 cm	IV, Below elbow, upper third forearm

As shown in Table 23.1, in M inactive, spatial duplicity with the Weber test begins in phase III (inverted), since with less pressure only phase II (medial deviation) is obtained, which only gives a single sensation. It is also observed that each spatial phase corresponds to a very different spatial threshold (minimum separation between the points of the compass to perceive duplicity). In phase III, with a sepa-

Table 23.2. Subject M under facilitation by strong muscular effort, stimulated in the middle of the forearm. Simultaneous Weber threshold (in cm) as a function of intensity (in grams).

Stimulus Intensity	Spatial threshold	Phase, Area of localization
65 g	8-9 cm	IV, Upper third forearm
165 g	6.5 cm	IV, More distally
265 g	5 cm	IV, Between the points of the Weber compass

ration of 18-20 cm between the points of the compass, the perception is two cutaneous sensations almost together; however, at the end of phase IV in M inactive, a separation of 7 cm is sufficient for this same result. This shows the different *tactile spatial shrinkage* according to the functional level reached. Thus, the small subjective separation in phase III must be greatly increased if the corresponding pressure is applied to reach the end of phase IV, resulting in a widening of the tactile space. The same occurs when, at a threshold, in the inactive state, facilitation is applied; the points are no longer perceived almost together but widely separated, especially if the stimulus is weak (see Fig. 23.1).

In the *simultaneous Weber* test, the spatial threshold values in different skin areas of subject M are given in Table 23.3.

Table 23.3. Spatial threshold (cm) in different areas for *simultaneous* Weber in subject M and a normal subject.

Areas	M inactive	M under facilitation	Normal (after Weber)
Forearm	7-8 cm	5 cm	4 cm
Hand	6 cm	3 cm	1.5 cm
Finger	3 cm	1 cm	0.2 cm
Tongue tip	1.5 cm	0.65 cm	0.1 cm

In this Table, the thresholds for the M case correspond to the maximum pressure with the two points of the Weber compass. By comparing the inactive state, the state under facilitation by strong muscular effort and that of the normal subject, it is observed that facilitation reduces the threshold to approximately half that of the inactive state. Both inactive and facilitated states present a threshold well above the normal one, this difference being much more marked for the cutaneous areas with higher spatial sensitivity. For example, in a fingertip, the threshold in the inactive state is about ten times that of normal despite the considerable pressure of the compass, whereas in the normal subject, a gentle contact is suffi-

cient. As with other functions, by means of stimulus intensification, or facilitation, the less fine sensory levels (case of the forearm) can be recruited almost completely, but the more differentiated ones are impossible to reach even by combining facilitation with very intense stimulation.

In all these tests, it is necessary to pay attention, in order to be more precise, to the temporal development of the sensation of duplicity, which is quite rapid under facilitation, but slow, up to four seconds, in the inactive state. There is an evolution through the various phases among which it is easy to distinguish simple contact, single point localization and finally duplicity. In general, in all pathological cases, the perception of duplicity does not occur immediately after applying the two points of the compass, and it is perfectly possible to perceive at least the interval between the sensation of a single point and that of two.

As for the *successive Weber* test (successive application of the compass points), the threshold in the normal subject is one third or less of the threshold for the simultaneous Weber. In pathological cases, a significant reduction in threshold is also obtained with respect to that of the simultaneous case, although somewhat less than the reduction in the normal subject. For example, the threshold for the successive case on the back of the hand of subject M is 3.5 cm in the inactive state and 1.8 cm with facilitation. Comparing these values with the respective ones for Weber simultaneous shown in Table 23.3, we see that the reduction is almost 50%. Therefore, even within the notable reduction of functional capacity, and using very intense stimuli, the diversity of thresholds according to the types of tests tends to be maintained. But it is important to note that, given the slow reaction time of subject M, a considerable time interval is necessary between successive applications of the two points of the compass. Thus, whereas in the normal subject the time interval between the successive application of the two points must be between 1/10 and 1/5 s to be perceived, in M, even with very intense stimuli, the interval must be at least 0.5 to 1 s, otherwise he will perceive fusion of stimuli (remember the fusion in vibratory sensation). A consequence of all this is that as the temporal interval increases, the successive spatial threshold is clearly reduced. Thus, with an interval of about 2 s, the threshold on the back of the hand in the inactive state can drop to 2.2 cm, which is smaller than the value indicated above (3.5 cm) corresponding to an interval of 1 s or less. Likewise, under facilitation and an interval of 1 s, the successive spatial threshold is 1 cm on the back of the hand.

Clinically, a dissociation between simultaneous Weber and successive Weber has often been suggested, as if they were independent functions. However, according to the criterion of dynamic reduction, it is understandable that simultaneous Weber having a higher threshold than successive Weber, is more affected, being difficult to achieve it in extreme cases. Instead, successive Weber, with a lower threshold, would still be possible, especially if the time interval in the application of the compass points is relatively long. If the conditions of stimulation in both types of spatial testing are carefully studied, the alleged dissociation or independence cannot be accepted, just as for any other type of functional activity. Apparent dissociations due to the effect of functional diversity exist, but an effective and absolute dissociation is only an observation or judgment error.

Other types of phenomena are of interest because of their dynamic characteristics of the spatial threshold. Thus, when the second point of the Weber compass is applied while keeping the first point in its place, the subject perceives a virtual motion of the first point towards the second. This motion effect can be considered an *induction* of the second stimulus on the first one, and the intensity of the effect is related to the distance and the intensity of the second stimulus. This induction seems to exist in a normal subject according to Frey (1910, 1913, 1916/1917, 1928), but in pathological states, such action is much more clearly manifested because it is increased. In subject M, it can easily be shown that the induction is much more pronounced in the inactive state than in the state under facilitation, and seems to be related to the instability of the sensory field.

In the inactive state, for two stimuli producing duplicity with a distance of 7 cm between them on the back of the hand or on the lower part of the forearm, the above-mentioned induction of the second stimulus on the first (which decreases with increasing distance) seems to disappear when the separation between stimuli is about 40 cm. With facilitation, it is much less, about 16-20 cm. The maximum induction effect in the inactive state corresponds to a distance below the duplicity threshold. For example, by placing the second stimulus 6 cm from the first stimulus, this one can undergo a virtual motion of about 3 cm toward the second stimulus. This sensation is only a single one, since it does not reach the threshold of duplicity, and also seems to gain in intensity, which should be understood as a "bi" effect varying with distance. In addition, due to the strong induction, the proximal deviation of the localization disorder can be substantially corrected, the single sensation tending to be localized between the two points of the compass. According to the above, it is understandable that when facilitation is applied, the single point sensation becomes double, i.e., duplicity appears as a result of reaching a more favorable functional level.

With respect to the action of facilitation it should be added that if facilitation is applied at the spatial threshold of the simultaneous Weber for the inactive state, the small separation perceived between the two points is clearly enlarged, i.e., a spatial dilation occurs.

A similar modification occurs when, the subject being always in the inactive state and first applying simultaneously the two tips of the Weber compass, they are then applied successively while maintaining the same aperture. In the latter case a greater separation is perceived. Here we could attribute the change to the fact that in the simultaneous modality there is a mutual attraction or induction of the stimuli, whereas in the successive modality, such induction is non-existent or much weaker, due to the split in the application of the two stimuli.

According to all the above, the disorder in tactile acuity (spatial threshold) is mainly explained by a spatial reduction, and perhaps partially by an increase in the mutual spatial induction of stimuli due to the spatial instability of the sensory field. All this leads to a *reduction of spatial acuity,* i.e., to an increase in the spatial threshold, according to the functional level of the nervous centers which depends on the magnitude of the central lesion, facilitation, degree of recruitment by the stimulus, etc. The correlation with tactile localization is clear, and it has already

been seen how Weber's threshold evolves in parallel with the localization phases, i.e., according to the degree of sensory organization. Thus, the increase in threshold is an immediate result of the *reduction of the body schema* (see Fig. 21.14).

23.2. MOTION ON THE SKIN SURFACE

The motion perception of a mechanical stimulus sliding over the skin is closely related to the state of spatial discrimination (tactile acuity). In addition to the spatial alteration, time disorder has to be considered. Both perfectly explain the different phenomena of motion disorder in touch, entirely similar to those occurring in visual motion perception.

In subject M inactive, motion on the skin begins to be perceived, as in Weber's test, in spatial phase III, although in a very rudimentary form and disturbed to the maximum, i.e., contralateral localization with strong proximal deviation, inverted direction of motion, maximum speed and considerable shortening of the trajectory. As for all types of functions, the sensation depends on the intensity of stimulation and other characteristics such as asynchrony, facilitation, etc.

In a moving stimulus (e.g., point sliding on the skin), the pressure on the skin, trajectory and speed, i.e., the intensity and duration of the stimulus, must be taken into account. Very fast motions need a long trajectory or a high intensity to awaken the sensation of displacement, as is understandable due to the characteristics of subject M. If a moving stimulus on the skin does not produce a certain level of excitation, only the static phase is obtained, as in vision, either phase I or II. As said, the first rudiment of motion perception corresponds to phase III, although still very far from normal sensation. As the stimulation intensity increases, the motion sensation tends to resemble, in phase IV, that of a normal subject (the trajectory increases, the speed decreases, contralateral localization and reversal direction disappear although some tilt remains), being impossible in the inactive state to reach complete normality no matter how much the stimulation is increased.

By letting a large drop of cold water fall along the arm of subject M inactive, he can perceive during phase III conditions (when hardly any thermal sensation is perceived) a certain motion sensation on the skin. But if the drop is either very cold or very hot, or the stimulation conditions are such that a first drop already awakens a clear thermal sensation, the perception of motion has the characteristics of phase IV, the more intense the temperature sensation, the more accentuated motion perception. It can be seen that in spatial phase III a series of functional differentiations appear, albeit weakly, such as lateral localization –although contralateral to the stimulus–, two-point discrimination, well-defined tactile sensation and motion, thus establishing a certain functional correlation between these diverse activities (see Sec. 21.2).

Under facilitation, phase III is not obtained and there is a sudden transition from static phases I and II to motion phase IV. Moreover, it is sufficient to stimulate with a piece of hard paper, whereas in the inactive state a pin is needed for the same purpose. Thus, there is a major change, and if there was already some per-

ception of motion in the inactive state, the perception approaches normality under facilitation.

Examining in more detail the characteristics of the altered motion perception, and leaving aside the changes in localization and direction, which will be studied in Sec. 24, we shall now consider only the *seeming acceleration* and the *shortening of the trajectory*. Both factors appear in inverse ratio to the degree of sensory development. Thus, at the onset of motion perception in phase III, the subjective speed becomes the maximum that the subject is able to feel, the true speed of the stimulus being greatly overestimated, and at the same time the perceived trajectory is the minimum in which the subject can feel translation of a moving object. The length of the trajectory in such circumstances is difficult to assess accurately, but can be estimated at one tenth of the true trajectory of the stimulus, i.e., if the true trajectory is about 10 cm, it is perceived as little more than 1 cm. For a given trajectory and speed of the moving object, as the pressure of the object on the skin increases, the perceived trajectory increases and the perceived speed decreases, although both are always out of normal by defect and excess respectively. The study of this type of alteration is easily done by the subject's description of his perception, since in extreme cases of alteration the perceived motion differs considerably from the real one. It can also be studied by observing the effects of facilitation in the inactive state under a constant stimulation.

The emergence of motion perception is in relation to the degree of tactile irradiation. In the static phase, only a uniform and widespread sensation is obtained (spatial phase II). In phase III, there is some irradiation, but a central point of more marked intensity already emerges which determines the motion sensation within the surrounding irradiation. Finally, in phase IV, as the spatial character is better recognized, the irradiation decreases, and the motion is perceived with a longer trajectory and lower speed, getting closer to normal. There is thus a joint evolution for several factors.

To analyze the genesis of the disorder it is necessary to consider some essential factors in both trajectory length and speed. The former depends on the degree of spatial reduction revealed by the increased spatial threshold in the Weber test. The overestimation of speed results directly from the alteration of excitability over time. Chronaxie is increased, i.e., more time of excitation is required, which means that time is appreciated in less than in the normal subject, therefore, the motion seems faster, as well as any other time perception process (e.g., vibratory sensation). In other words, there is a *contraction in space and time,* the latter being the one that mainly disturbs the perception of motion on the skin, or other kind of motion perception (e.g. visual). It follows from the above that the higher the sensory chronaxie, the greater the overestimation of speed.

Finally, we should highlight the perfect similarity between motion disorder in touch and in vision, as summarized for subject M inactive in Table 23.4.

Table 23.4. Similarity between motion perception disorder in touch and in vision for subject M in inactive state.

Phase	Spatial irradiation	Sensation	Motion		
			Speed	Trajectory	Direction
I & II	Homogeneous irradiation – Achromatic fog				
III	Irradiation with central point – Spatial color	Initiated	Maximum	Minimum	Inverted
IV	Point, weak irradiation – Flat color	More defined – Green chromat.	Lower	Longer	Tilted
V Normal	Point localization – Surface color	Normal – White is possible	Normal	Normal	Normal

23.3. SHAPES ON THE SKIN SURFACE

The perception of shapes and figures either applied or drawn on the skin is related to all that has been said about tactile acuity (Weber) and motion. *Static shapes*, i.e., those applied to the skin in one single action, have little relevance even in the normal subject. They are very difficult to be perceived by subject M in the inactive state even when resorting to high-pressure stimuli. It is understandable considering his remarkable alteration in the simultaneous Weber. The blade of a knife applied by its cutting edge or by its blunt edge needs to be pressed rather hard on the back of the hand to elicit the sensation of a static line. When it is perceived, it always seems to be shorter than the stimulus, apart from other alterations of position and orientation which will be studied further on. The perceived length is related to the intensity (pressure) of the stimulus, in a similar way to that described for motion perception. What can be studied by means of static shapes is very elementary, but it is of great importance for studying orientation disorder (orientation of a line).

The interest of tactile shapes lies therefore in *kinematic shapes,* i.e., shapes drawn by means of a point on the skin. Kinematic shapes are much easier to be perceived, requiring much less pressure. This difference with static shapes corresponds to that between simultaneous threshold and successive threshold in Weber's compass test. Kinematic shapes present the characteristics studied above on the perception of motion on the skin. Most of the somewhat complex shapes need a rather advanced spatial phase, so they belong to phase IV (see Sec. 21.2), where spatial contraction and speed overesti-

mation are less pronounced than in the onset of motion sensation in phase III. The subjective speed may give the sensation that shapes are drawn quickly, but it is not the cause of an alteration of the shapes. Instead, spatial contraction leads to a reduction in the size of the lines, which is more pronounced at the beginning of phase IV.

In addition to the decrease in size, the shapes show a peculiar disorder that results in dysmorphism, originated by various causes. In phase IV there is still an alteration of the orientation that causes changes in the perceived orientation to varying degrees depending on the stimulus, giving rise to changes in the shape. Moreover, the lines are deformed due both to the particular conditions of excitability causing that the beginning of the lines can be excluded due to a slow reaction time, and to the instability of the tactile sensory field giving rise to certain dominant balances in the sensory field which imprint their peculiarity to the drawn shapes. All this will be studied below.

These different factors often act together in the transformation of the figure perceived, but it is not difficult to analyze them separately by appropriate tests. One of the most remarkable effects consists of a deformation that could be called *longitudinal dysmorphism,* caused by an abnormal spatial balance in the sensory field. When drawing, with a toothpick, the largest possible circle on the back of either of the two hands of subject M, in a continuous way, rather slowly and with enough pressure, he perceives an oval of half the size of the circle, oriented in the direction of the axis of the limb. If instead of the circle, a cross 4-5 cm long is drawn, the sensation is of an X-like shape shortened on the sides, more or less oriented also in the direction of the axis of the limb. To better know the sensation perceived by the subject, he afterwards draws the perceived figure, as shown in Fig. 23.2. A verification test consists of drawing on the back of the hand an oval and an X-shaped cross, transversal to the axis of the limb; they are perceived as a circle and a cross respectively, although smaller than the original model. The figures are perceived with a proximal deviation that, given the strength of the stimulus, is of little importance. The deformation practically disappears by the action of facilitation, at least using a stimulus of energy similar to that of the previous test in the inactive state. Under facilitation there is also no size reduction.

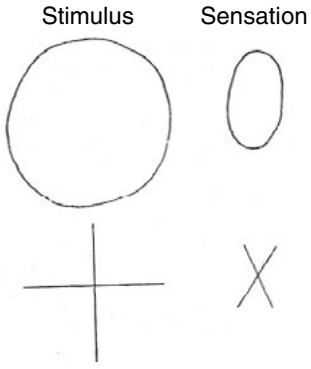

Figure 23.2. Longitudinal dysmorphism in subject M in the inactive state. On the left, figures of about 4-5 cm drawn on the back of the hand. On the right, perceived figures showing deformation in the longitudinal direction of the limb, as well as reduction in size.

As for the interpretation of this type of dysmorphism, there may be some relation with the longitudinal irradiation (irradiation band) in the localization disorder, and most probably with an increased effect of the longitudinal spatial predominance already observed in the normal subject with the circles of Weber. It would thus result, as indicated above, in a heterogeneous spatial balance, i.e., an anisotropy of spatial values in the sensory field. This same type of deformation is also present in vision (dismorphopsia), and we have been able to observe it clearly and with precision in many wounded with anomalies in the visual field, before the study of the two present cases who also present this deformation under special conditions of experimentation. This visual disorder has been deliberately omitted in this work because it is not essential for our present purpose, leaving it for a special publication on various very singular optical phenomena (polyopia, pseudofovea, etc.). We should point out that this visual dysmorphism was first described by Gelb and Goldstein (1923) in a series of brain-injured patients with visual field alteration.

Other types of deformations can be produced by the action of factors of different nature, either in conjunction with the effect described above or independently. A couple of examples are shown in Figure 23.3. A large cross may become an acute angle if the drawing is faster than in the previous test, then the initial line strokes do not have time to produce sensation, the longitudinal deformation subsisting for the remainder of the lines. The result is then a longitudinally oriented acute angle, and if the residual is even smaller, only two more or less parallel lines remain. A more considerable transformation occurs when the figures drawn are small (2 cm). In such a case, a cross usually becomes a more or less closed curve. Here it is necessary to appeal to various effects to explain it. Short line strokes mean less stimulation, preventing a differentiated perception. In addition, due to the orientation disorder, the

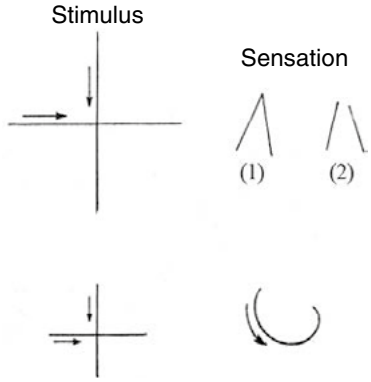

Figure 23.3. Different dysmorphisms on the back of the hand of subject M in the inactive state. At the top, a cross is perceived in a first test (1) as an acute angle by elimination of the initial line strokes (excitability deficit), and lateral narrowing due to longitudinal dysmorphism. Repeating the test, it is perceived as two more or less parallel lines (2). At the bottom, a smaller cross is perceived as a more or less closed curve due to a complex effect involving excitability disorder, orientation disorder, form´s pregnancy (in gestaltist terms), etc.

line stroke is perceived as tilted. Above all, as this process corresponds to a low functional level and to a great instability of the sensory field, the different line strokes tend to be joined and curved by the action of pregnancy of the form (in gestaltist terms, see footnote in Sec 10.2.2).

Similar deformations were described by Stein and Weizsäcker (1927, 1928) in central tactile lesions. A simple drawing that is repeated at the same skin site elicits different perceived shapes each time because of sensory fatigue and lability, until a point at which the drawing stimulus no longer elicits any sensation due to a continuous increase of the excitation threshold. It should be emphasized that the dysmorphism in all the cases indicated obeys perfectly regular and easily explainable laws; therefore, we are not dealing with errors of interpretation of the forms but rather with a certain modification of the forms, as already studied in visual perception of forms (see Sec. 11.3 and 15.1 in Vol. 1).

As for the action of facilitation, it suffices to add that in small figures it eliminates deformation and also increases their size.

Tests of greater complexity are performed with letters and numbers, showing the instability of the figures even under optimal stimulation conditions. The lines should be made with great pressure and a certain slowness, the figures should be written on the skin (back of the hand) in a correct and regular way, and the size should be as large as possible (5 to 6 cm). In this way, subject M in the inactive state can recognize a '0' an '8' or an 'A,' even though the perceived size is half or even less than the true size. However, recognition alterations are very frequent, generally due to shape simplification and similar effects. Thus, many numbers are taken for a '1,' a '2' is perceived as an '8,' a '3' as a '2,' etc. The shapes of '6' and '9' are well perceived by him but he confuses them mutually, correcting under facilitation. For the perception of numbers, it makes no difference whether they are written normally or as a mirror image; the result is the same.

By contrast, deformations or changes in meaning are markedly reduced under facilitation: errors tend to be scarce, the size of the numbers is larger (a little more than two thirds of the true size), and drawing on the skin can be faster and with much less pressure. Moreover, the subject (M) notices if the numbers are abnormally oriented. However, some remnant of abnormality persists (small decrease in size, some errors, etc.).

23.4. SENSITIVITY TO JOINT MOVEMENT

The perception of joint movement shares the general characteristics of the perception of motion on the surface of the skin, presenting in addition peculiar aspects determined by the examination method. In the case of motion on the skin, when a moderate pressure stimulus does not elicit motion sensation in a short path, the path can be prolonged until the effect is obtained. However, in joint movement, the amplitude for stimulation of movement is limited by joint excursion. If the examination is restricted to the simple test of passively moving an articular segment, it could be erroneously concluded that the perception of articular movement is completely abolished

in somewhat intense alterations. However, in this function, as in all other functions, the impairment occurs in a gradual manner and the aim is to determine the *quantitative value of the functional reduction.*

In what follows we shall deal with the perception of the so-called *passive joint movement.* The active movement, more proper to another chapter, will be dealt with briefly only as a complement. As for the two subjects studied here, it should be noted, as always, that subject T in the inactive state is approximately similar to subject M with facilitation. Subject M in the inactive state presents at first sight a considerable loss of perception of joint movement; an arm can be passively raised from the vertical to the horizontal position without him perceiving any movement.

The same result is obtained in any other joint. With facilitation, the sensation of movement is lacking when the displacement is small. In general, there are many possibilities for examination or stimulation. Mild and repeated stimulation is possible as well as single energetic stimulation. The subjective experience is much more complex than is commonly recognized. Suffice it to say that, as in motion on the skin, intrinsic motion disorder (shrinking and acceleration) is present together with spatial localization disorder (proximal deviation and inversion).

We shall begin with the most common joint movements in ordinary activity. In our subjects, these movements must be *repeated* to elicit the sensation of movement. The following experiment gives a fairly complete picture of the state of this function. While subject M is in the inactive state, the tip of his index *finger* is held by its nail and alternate flexion and extension movements of the finger are imparted. With two oscillations of medium speed and amplitude, he feels only a simple contact of spatial phase I type. With four or five oscillations he reaches phase II, thus continuing to feel no movement, and localizes the contact toward the chest, as expected. With ten or twelve oscillations (these numbers are approximate depending on the stimulation method), a joint movement is perceived in the contralateral elbow, but only two or three very brief movements are felt (corresponding to the multiple oscillations of the index finger). Here, phase III has been reached, in which movement with contralateral localization is initiated. If the oscillating movement of the finger is maintained, the joint movement can be localized in the homolateral wrist joint. For this to occur, it is necessary to make the finger oscillate widely and rapidly. Phase IV is then reached, and the movement is perceived a little slower and wider than in phase III. Finally, no matter how long the oscillation of the finger is maintained, a new change is not achieved. Thus, it is impossible to bring the sensation of movement to the metacarpophalangeal joint (origin of the true movement). Table 23.5 summarizes the perception process.

From all this it can be inferred that in this type of stimulation the recruitment of the spatial level is obtained by *iteration*, similar to what happens in visual perception of motion in the metronome test (see Sec. 11.2 in Vol. 1). Recruitment is incomplete and normality is not reached. The movement is intrinsically altered in both speed and amplitude, is spatially deviated and undergoes the same spatial asynchrony studied in other tactile excitations.

Given the large number of oscillations necessary to obtain a sensation of movement even in signs, and also the strong proximal deviation and contralateral inver-

Table 23.5. Iterative joint sensitivity in subject M in the inactive state. Passive oscillatory movement of the index finger.

No. oscillations	Joint sensation	Localization
1	None	None
2	Static	None (phase I)
4	Static	Medial (phase II)
10	Signs of movement	Contralat. elbow (phase III)
15-20 very strong	Mov. wider and slower than before	Homolat. wrist (phase IV)
Indefinite	Idem	Idem

sion, it is understandable that one can easily make the mistake of assuming a complete abolition of the sensation of joint movement. Such a mistake may be favored by the subject who knows that the test consists of the movement of the finger (even if during the experience he remains with his eyes closed), since, not feeling sensation of movement in that finger, he tends to exclude any other perception of movement, which in fact is very weak. This shows how necessary it is to pay close attention to all kinds of circumstances, both to the factors of excitation and to the sensory experience of the subject, in order to get a true picture of his functional condition.

It should also be noted that joint movement can elicit a sensation of simple contact, of phase I, fully identical to that produced by a weak mechanical pressure on the skin, or by a thermal stimulus that does not arouse a sensation of temperature, etc. Thus, stimuli of a very different nature produce the same sensory effect within certain limits, the specific response being lost. Along with the decomposition of movement into static and kinematic phases (the latter of varying speed), there is no doubt that one of the major novelties in the disturbance of the perception of joint movement lies in its *localization* anomalies, which shows an identical process to that of the perception of a stimulus on the skin, whether it is moving or static (see Fig. 21.4). Given that both localization and the sensation of movement evolve in a completely parallel way, it is easy to understand how unacceptable it is to consider dissociations between the different activities, an issue that arises throughout the different chapters of this book. It follows from all the above that the so-called dissociations have arisen from the partial and incomplete analysis of a set of phenomena; and in reality, there is only a functional reduction in the series of activities of a given sensory system.

Tests in other parts of the body show characteristics analogous to those indicated for the finger, but it is convenient to examine the particularities of each case. Regarding the *elbow* joint, different phases are obtained with increasing number of oscillations; first phases I and II, then the sensation of movement appears in the contralateral shoulder (phase III), and finally the sensation shifts to the moving elbow, with the already described subjective change of speed and amplitude. Thus, the change of localization from the contralateral side to the homolateral one is not symmetrical and occurs with a certain deviation. In the case of the finger, from the elbow to the wrist; and in the elbow test, from the shoulder to the elbow. In the lat-

ter case there is no final proximal deviation because the stimulation is not at an extreme distal location. In the first case of the finger, it is useless to pretend that by stimulating all the fingers (opening and closing the hand passively with force and quickly), the perception of movement descends below the wrist; but the change of side from the elbow to the wrist can be made easier than with the stimulation of a single finger, a change that is sometimes difficult to achieve.

For the case of *wrist* joint movement, in addition to the non-movement perception phases, the perception of movement moves from the opposite shoulder to the homolateral elbow, and finally to the stimulated wrist, although the latter occurs with considerable difficulty since very intense stimulation is necessary. In this case, localization at the stimulation site is achieved because the latter is not very distal, although the difficulty is greater than in the case of the elbow.

For the *shoulder*, the localization goes from the contralateral shoulder, where the perception of movement is initiated, to the stimulated shoulder, with the corresponding difference in speed and amplitude. In this test, the transit from one side to the other is at the same level because there is no other possibility. For the lower limb, the same behavior is found as described for the upper limb in all cases.

Finally, the case of the *head* is even simpler, without any transit from one side to the other. After phases I and II with no perception of movement in the medial region of the head (vertex), the movement is perceived in the neck or occiput. No new change of localization of the movement occurs here, since it is impossible, but a phase III with reversal of the direction of the joint movement can be found, as will be seen in another chapter. Due to the characteristics of the onset of movement sensation (phase III), it can be perfectly distinguished from the more developed (slower and wider) movement of phase IV.

Facilitation causes in all the above tests a change as remarkable as in any other function. Subject M in the inactive state under continuous stimulation of the finger, does not achieve localization beyond the wrist. With facilitation, however, the perception of movement is shifted to the actual site of stimulation (metacarpophalangeal joint); at the same time, a doubling or tripling of the trajectory and a significant decrease in speed are felt. A greater number of oscillations is also recognized. Therefore, there is much more agreement between the stimulus and the subjective sensation.

If the above tests are performed from the beginning with subject M under facilitation by maximal muscular effort, the significant functional improvement results in reducing the excitation threshold, suppressing intermediate phases (especially phase III) and greatly reducing abnormal residuals (proximal deviation), as well as reducing movement impairment. Thus, the following is obtained for the finger test. A slight shaking is enough to obtain a sensation of movement without the need for repetition. If the stimulation is properly regulated, it is possible to obtain asynchrony and to find phases I or II, but phase III is never possible. From phase II, localization goes directly to the homolateral wrist, but if the intensity of stimulation is increased a little, the sensation of movement arises in the finger. Thus, there is no residual proximal deviation, and it should be noted that a wide finger oscillation of about 40° is sufficient for the sensation of movement to arise. However, two or three oscillations of the finger are necessary if the amplitude of the oscillation is small.

When the passive movement takes place in the interphalangeal joints of the finger, an intense muscular effort enables the movement to be felt in the same location of the motor origin, but several oscillations (7-8), somewhat intense, are necessary. Therefore, when it is an extremely distal movement there is a tendency for proximal deviation, even with facilitation, although the deviation can be overcome by reiteration. In contrast, in the inactive state the localization cannot go beyond the wrist.

For the other joints of the upper limb, facilitation makes it possible that a single small oscillation is sufficient to obtain the perception of movement in the examined joint (wrist, elbow, shoulder). These are large joints and more proximal (to the center of the body), factors that facilitate excitation. In tests with facilitation by generalized muscular contraction, the joint segment whose passive movement is being explored must, of course, be left free. When the area under examination is also part of the strong facilitating muscular contraction, the threshold of sensitivity to movement decreases even more than in the previous case, but here it is not a sensitivity to purely passive movement since the provoked displacement of the limb must overcome the strong muscular contraction.

Subject T showed at the beginning of being examined (1938) the anomaly of perceiving passive joint movement as simple pressure (static phase), together with a certain difficulty in localizing stimuli of medium intensity. It was also evident that he did not perceive slow movement even of a certain amplitude. However, these disorders disappeared through involuntary muscular twitching (spontaneous facilitation). Later, we observed that this subject behaves in everything like subject M under maximum facilitation. Thus, passive finger movements of very small amplitude are not perceived, but when the amplitude is increased a little, the sensation of movement arises, although modified since it is clearly felt to be of smaller amplitude than the real one.

We have seen above that the sensation of joint movement is only possible by prolonged repetition of moderate oscillations, especially in subject M. However, by stimulating vigorously it is possible to achieve the perception of movement in the inactive state with a *single oscillation.* Of course, this oscillation has very different characteristics from the previous ones. For example, a single oscillation with all possible amplitude of the metacarpophalangeal joint and with great speed can result in a sensation of movement at the wrist. For this to occur, it is very important to forcefully reach the point of maximum excursion of the joint. Otherwise, the proximal deviation is stronger and the localization results in the homolateral elbow or the contralateral shoulder. It is therefore possible to regulate the test and obtain different localizations. In short, with a single oscillation the same result can be achieved as with 15-20 moderate oscillations, but the single oscillation must be very strong, especially to achieve the maximum effect that is possible in the inactive state.

The conditions under which the sensation of movement is elicited by single stimulation deserve close examination. A necessary characteristic, common to both single and repeated oscillations, is the *rapidity of the oscillation.* In this respect there is a great difference with visual perception of motion and tactile perception of motion on the skin, since in these cases an excess of speed of the moving stimulus tends to

impede the perception of its motion. By contrast, in the multiple tests performed over the years on our subjects, it has been found that a rapid joint movement is necessary to cause any effect. The fact that the sensation of movement is better when the speed of the movement is higher was pointed out by Goldscheider (1889), who proved that at low speeds the threshold value to perceive joint movement is somewhat increased.

The *range of movement* and the *pressure on the joint end-stop* must also be considered. The importance of the former is clearly seen in the single oscillation test where it is desirable to involve the entire joint excursion; it should also be noted that in the less disturbed cases (subject T inactive and M with facilitation) there is a greater sensitivity for large joints than for small ones. As for the pressure on the joint end-stop, it seems to play a decisive role in generating the sensation of joint movement. In the theory of joint movement, there are two opposing hypotheses: the *purely articular* or articular surfaces theory (Goldscheider 1889), and the *para-articular* theory (Frey 1918) which attributes the process to the changes and tractions performed by the muscles, tendons, articular capsules and even other deep and superficial soft parts including the skin. In any case, the articular movement would be the result of an organic complex. In our cases, the fact that the pressure on the joint end-stop is important, forcing the joint capsules, ligaments, etc. to the maximum, seems to support the para-articular theory of Frey. The influence of the speed and the range of movement could also support it; high speed would cause an abrupt change in muscle tension, and a wide range of motion would work in the same way. Thus, if the passive movement is the effect of the activity of ligaments, tendons and muscles, and not of the simple displacement of the articular surfaces, the sensation of movement would develop in a very indirect way in comparison with the case of an object moving on the skin or with the case of visual perception of motion.

The pure articular theory (displacement of the articular surfaces) seems to have a little less support in our tests. The artificial increase of the articular pressure (of one surface against the other) which, according to experiments by Lewinski (1879), would notably favor the sensitivity to movement, has given us irregular but rather confirmatory results. However, it is questionable whether the facilitation action is due to pressure between the articular surfaces or perhaps to an indirect summative effect due to the pressure exerted on the skin when gripping tightly the finger being tested, since this gripping may result in localization at the wrist even if there is no actual movement. Without being able to completely attribute the perception of joint movement to certain factors by excluding others, it should be noted that the important effect of pressure at the joint limit and also the effect of the speed of movement seem to directly support the para-articular theory of ligament tensions.

However, the importance of the amplitude of movement can be a support for both para-articular theory and purely articular theory. In short, the process is complex and a very considerable part seems to involve para-articular organs, in accordance with the predominant orientation at present.

Finally, we should briefly indicate some characteristics about perception of *active* joint movement, i.e., voluntary movement. This shows in general the same disturbances as the passive movement, although somewhat reduced. For subject M to be able to make a voluntary movement, he needs a certain degree of facilitation

to bring out the body schema, and then he can make use of his own movements (as will be seen in Sec. 27). When he is asked to move any finger of his hands, he needs a significant muscular effort to orient himself on his own body and choose the requested movement. But, if the movement of his finger has not been very vigorous, the sensation differs considerably from the real movement made. If the movement is slight, it may not be perceived as such movement (phase I) or in any case be very deviated, or contralateral, since the facilitation is not maximal. If the movement is more intense, it may be localized in the homolateral wrist, etc. Of course, the greater the deviation, the greater the speed and brevity of the perceived movement, as in passive movement. Under repetition of the movement, changes in the localization and amplitude of the movement are obtained by increasing the number of oscillations.

It is therefore understandable that there can be considerable incongruities between the voluntary action and the corresponding proprioceptive sensation. Different questions of great importance will be studied in depth in the section on orientation in touch (Sec. 26) and in the section on the body schema (Sec. 27), where the issue now initiated will be continued.

23.5. BODY PERCEPTION AND MANUAL TOUCH

Body perception, and in part manual touch, are issues of a more complex order than those discussed above, and already belong to the function 'tactile schema' which will be dealt with in the last part of this book. However, as is always the case, there are transitions between the different categories. Here, both the *schema of the own body* and manual touch (*active touch*) will be briefly discussed in relation to tactile space, thus completing the study of this topic.

23.5.1. Reduction in the schema of the own body

Body perception derived from the set of tactile sensations, both cutaneous and deep, presents remarkable phenomena due to asynchrony. Among the most characteristic phenomena are: fragmentation of the body schema, abnormal flexibility of the body, sensation of lightness (loss of weight), reduction of size, etc.; of which a brief description will be made although many of these phenomena are difficult to be fully described.

As the coherence of the tactile spatial functions is impaired, a certain *fragmentation of the body schema* arises, that is, a kind of independence of its parts, which acquire a singular degree of lightness or flexibility, all similar to the phenomena described by Beringer (1923, 1927) in experimental mescaline intoxication. This author describes that the body continuity is lost, and when shaking hands, the affected person has the feeling that the hand is detached from the rest of the body, the body is felt to be smaller, as if concentrated in a homunculus, etc. When a limb is moved passively and the inversion phase is obtained, the subject feels the movement of the limb as something more or less disconnected from the body and says that it seems to be

a little loose; whereas in the next phase (IV), this disconnection tends to disappear quickly. From the examination of the patient's perceptions it seems that in the first case the moving limb has no references with respect to the rest of the body, especially the trunk; but as the organization improves by increasing stimulation, the rest of the body schema tends to emerge, although it is a process difficult to express clearly. The less developed phases (II and I) give an even more blurred sensation of the body schema, which is reduced to spatial references that are always static and highly ill-defined.

Subjective *flexibility* of the body is another aspect of body schema disorder. When a limb moves, either passively or actively, it gives rise, within certain functional level limits, to the sensation of being somewhat diffuse or swollen, soft or malleable; in part, somewhat similar to the sensation a normal subject has when a limb falls "asleep" due to nerve compression. In relation to the decreased sensation of pressure, both *weight* and *strength* seem greatly diminished (see end of Sec. 18.1), the body is felt much lighter and without resistance to thrust, thus contributing to the lack of body definition. When rising from the sitting position, in an inactive state or with the minimum possible facilitation, the weight of the body is perceived as very light compared to the sensation when performing the same movement under maximum facilitation. The same occurs when walking, the movements are felt light (or without force) as well as fast and short. If the subject actively lifts a limb under maximum facilitation (by muscular effort), the subject feels it three times heavier than if he lifts it in an inactive state (but with minimal effort to produce a voluntary movement). Under facilitation, a hand is felt as heavy as the whole leg in an almost inactive state.

The *size* of his own body is felt to be reduced due to spatial contraction (see Sec. 23.1), and in the usual state, the subject feels himself considerably smaller than under facilitation (by maximum muscular effort). In addition, and as an effect of proximal deviation, the more distal parts of the limbs are excluded, resulting in a further decrease in body size and volume. These distal parts need maximum facilitation to recover their presence (see Fig. 21.14).

All these anomalies are easily manifested in activities such as walking, getting up, sitting, etc. When rising from sitting, in the usual way (without maximum facilitation), the body is felt to be smaller, lighter and softer; but if then the subject starts to walk, the normal characteristics tend to become progressively established. In these subjects, walking is a very complex process from the proprioceptive sensory point of view, and will be studied in detail in the section on orientation. In the following, only some characteristics about corporeality during gait are indicated. In the first steps, the body is felt to be much smaller and lighter in weight, and the steps are felt to be much shorter and faster. At the same time, the footstep feels soft "like on a carpet." A great change occurs when the subject is under the action of maximum facilitation since the functions are considerably normalized. As for the resistance of the floor when treading, the subject in the inactive state is unable to distinguish between treading on a floorboard and on a carpet. However, with facilitation he immediately notifies when he treads on a carpet or on a hard floor (always without seeing the floor). When he stands and rests his body on one leg, this leg seems to

him longer than the other; the same effect is obtained when, standing on both legs equally, he moves one of them or contracts its muscles.

Finally, to be complete in our description, we should mention the recognition of body postures or attitudes, especially of the limbs; but this issue requires an in-depth study of the body schema in order to be sufficiently clarified, which will be done in the sections on tactile schema. Suffice it to point out now that the perception of body attitudes requires a highly developed body schema, and therefore such perception is usually suppressed unless a fairly intense facilitation is applied, and even then, there are significant reduction of attitudes, also in the less impaired subject T. In addition, there is a special tendency to ignore different postures or attitudes, which are perceived as similar to the usual posture of the body and limbs (pseudoagnosia).

In short, the perception of the body is closely related to all kinds of tactile space functions, and its study will be completed when dealing with orientation, and mainly with tactile schema.

23.5.2. Reduction in manual (active) touch

As a result of the various alterations of the tactile space, superficial and deep (in joints), active touch with the fingers can be quite affected even under the usual conditions of the subjects. The disturbances are, as for body perception and other sensory functions, of the type described by Beringer (1923, 1927) in relation to mescaline (the author says: "hard objects feel as if they were rubbery, soft and ill-defined in shape.")

These alterations of active touch with the fingers due to the spatial disorder of touch could be called *flat touch,* by analogy with flat colors. Thus, due to the thickness of the tactile irradiation that dulls the cutaneous surface, there is a certain sensation of penetrability or flexibility of hard objects and a reduction in the appreciation of microstructures. Hard objects, such as a glass jar, do not provide to the hand's touch a strong sensation of hardness, but are perceived rather as something malleable that could be squashed with the fingers, but not like a rubber ball but like a solid rubber. Similar sensation may be elicited by a wooden table top, seeming less hard, or as if lined with a padded tablecloth. All these abnormal sensations are greatly diminished with a fairly intense facilitation (by muscular effort), but they are quite evident to the testing subject when facilitation is weak, and even if he exerts strong pressure with the fingertips when touching objects.

It also happens that all touched objects give the sensation of having a smaller size. This must be caused by the contraction of tactile space (superficial and articular) already studied, in such a way that the size of an object becomes equal to that of the hand. As tactile acuity is reduced, roughness and other characteristics of the microstructure of objects are erased, giving the sensation of being smooth to the touch. In all these tests, the subject is free to touch objects with his fingers by moving the fingers as he wishes and pressing the objects at will; and although this involves some muscular effort (facilitation), the above-mentioned anomalies are clearly present. Only with facilitation by vigorous muscular effort as well as active touch with strong pressure, a noticeable change towards normal sensation occurs, regarding hardness, roughness, size, etc.

All these phenomena show us that stereognosis is very impaired, although not abolished, but reduced and altered by 'flat touch.' It is then understandable that tactile recognition of objects is severely altered. When the subject slides his hand over a brush, he only feels a soft surface that deforms greatly, comparable to a rubber ball. He feels the same when he sinks his fingers into the brush. When he slides his hand repeatedly over the brush or his hand is rubbed with the brush, he perceives at most a slight roughness but as coming from a compact mass. When the subject is in an inactive state, the rubbing of the brush on the cheek gives him the sensation of a rough cloth, but on repeating the action, he ends up having a normal perception. A somewhat thick pocket chain is perceived as a cord, i.e., more compact and softer than it really is. However, under facilitation all these anomalies disappear.

It should be noted that touch must be combined with movement since in this way the advantages of the successive Weber test are exploited, in addition to other facilities provided by joint movement. Thus, even if the tips of a fork are strongly applied to a finger, the subject feels only one tip and must move the fingertip over the tips to perceive two or three of the four tips. But this active touch (kinetic or successive) is still very diminished in the ordinary conditions of the subject since if he touches the tines of a fork lying on a table with the tips downwards, not upwards as before, he says he perceives only "two things, two thin sticks"; and this is achieved with very active movements and a strong pressure exerted with his fingers. Only with facilitation by maximum muscular effort does he recognize several times and immediately understands that it is a fork. The test is performed, of course, with the eyes closed.

When many ordinary manual activities are examined by allowing the subject only a weak facilitating action, such activities present great difficulties. Thus, when buttoning a shirt or any other article of clothing, he finds great difficulty in locating the buttonholes, distinguishing them from the rest of the cloth and, above all, in channeling the buttons through the buttonholes, etc., all effects of the reduction of tactile space.

Impairment of tactile object recognition involves several anomalies, namely the so-called 'flat touch' anomaly that influences the loss of acuity, other intrinsic anomalies in shapes on the skin surface and in joint movements, and in a very important way the alteration in body perception (e.g. distal exclusion). The disorder of tactile object recognition is therefore the result of a global disorder of the tactile system, and will be dealt with in the part devoted to the schema.

Perceived tactile orientation

24. Dynamic disorder of orientation in touch

24.1. General aspects

The phenomenon of inverted vision was discovered in subject M by chance in 1938, and only one or two years later could it be rationally understood within the framework of this brain dynamics research. However, the *inversion of tactile space* was not found until much later (1945-1946). Its finding, also partly by chance, was a consequence of certain theoretical assumptions but mainly of very meticulous examinations of various tactile functions such as spatial localization and motion perception of a stimulus moving on the skin. With the discovery of the dynamic action phenomena (1939) it was shown that the nature of sensory disturbance was the same in intensity and manifestations for the three most important sensory systems, vision, touch and hearing. An exception, however, was inverted or tilted vision, unparalleled at that time in touch and hearing. From a theoretical point of view, if pathological inverted vision was to be linked to the normal inversion of the visual image on the *visual retina* and thus transferred to the "cerebral retina" (sensory projection), it is also true that the inversion of sensory projections is not a particular case of vision, since an analogous anatomical arrangement exists for touch and hearing. Precisely in the case of touch, it is elementary knowledge that the projection of each half of the body is contralateral and upside down. Therefore, it seemed that an even greater parallelism in the alterations of the mentioned sensory systems should be expected. In principle, both a tactile and an auditory inversion were expected, but it was difficult to predict what modality they should adopt.

The long-standing and persistent debate on how vision can be right (normal), knowing that the visual image is inverted on the retina, has already been pointed out in the appropriate place (Secs. 12.1 and 14.1 in Vol. 1). In contrast, no trace is found about touch, although a very similar problem can be posed if one considers the inversion of the sensory projection in the cortex. This is because the knowledge of the contralateral and inverted projection of the tactile space (or of the body) belongs to a time (late19th century) when all these questions of the sensualist philoso-

phy about the orientation of space and other related questions had fallen into the most absolute oblivion among scientific concerns; and moreover, these issues had never given rise to significant manifestations in brain lesions. As already mentioned, the long dispute about the mechanism for a correct orientation of the perceived visual image has not had any impact on brain pathology either despite the existence, since very ancient times, of some clinical reports about inversion and tilt of the perceived visual image, although without objective verification or detailed study of the phenomenon, always described in a few lines.

When dealing with visual image orientation, we have mentioned some clinical precedents on pathological inverted vision, whereas it is almost impossible to find them for tactile inversion, except perhaps in the brief, vague and unexplained observations of what is called allochiria (tactile localization contralateral to the stimulus), which we have been able to observe in a brain-injured patient in 1948. In accordance with what will be expounded on the theory of orientation in Sec. 26, it seems certain that in many of the known cases, allochiria correspond to phenomena of a very different kind from that now being studied on tactile inversion. If some cases could perhaps show some relationship, it is clear that they would constitute a minimal and much less direct precedent than what we have found in inverted vision.

Within the general difficulty of investigating the different abnormal manifestations, the fact that in subject M tactile spatial inversion is more difficult to be detected than visual inversion deserves a comment. As Katz (1920/1925) points out, already under normal circumstances, visual sensations and functions are more objective than tactile phenomena, since the visual ones refer to the external space, outside the body and the eyes. However, in tactile phenomena there is inevitably a subjective factor in relation to both our own body and the object; this is why Katz calls tactile phenomena "bipolar."

We can admit that such tactile circumstances make it difficult to clearly perceive pathological phenomena which fit poorly into the normal routine, and which subjectively would be suppressed or diminished. But moreover, there is a general tendency, not only in touch, to exclude any defect both by the singular and inscrutable phenomenon of anosognosia and by the summative mechanism of facilitation by muscular effort exerted unconsciously. In view of all these characteristics, it could perhaps be said that tactile inversion has been more difficult to discover due to the lesser objectivation of tactile perception. Probably, other peculiarities of tactile space are also involved, such as its greater heterogeneity compared to visual space. In short, tactile inversion is a very hidden disorder despite the multiple and important manifestations to which it gives rise. When inverted vision was found in subject M, he said spontaneously that he had on occasions before seen more or less marked inversion of the visual scene. In the case of touch, however, he was not able to say something similar, and the complex and diverse phenomena of tactile inversion, painstakingly brought to light, seemed to him as surprising and strange as the phenomenon of facilitation by muscular effort at the beginning of being examined.

Tactile inversion is linked to the finding of phase III in the localization of a stimulus, a phase that arises due to nervous asynchrony as an extreme consequence of the disorder of orientation in space, as with vision. However, depending on the

level of recruitment, the orientation disorder appears differently pronounced, with different degrees of tilt, belonging to phase IV. These tilts can be easily appreciated when the stimulus is a straight line whose orientation on the skin is possible to perceive. This is obviously not possible with a point stimulus. Therefore, the orientation disorder is shown through phases III and IV until phase V (normal) is reached. The less developed the spatial phase is, the greater the disorder, although the first two phases (I and II) do not count at all in the study of orientation because they have a too elementary sensory organization. It should be noted that this disorder presents a very broad course. Phase III only corresponds to the extreme alteration with practically complete inversion of the orientation and contralateral localization, whereas in phase IV (very broad and with homolateral localization of the stimulus as we know), the orientation of a straight line can show from very large tilts exceeding 90°, to very small or practically null tilts. Therefore, the exact localization of a line also involves its orientation, so the following must be taken into account: the side of the body, the proximal deviation and the orientation of the line. Moreover, as the line is perceived in different sizes depending on the phases, the localization process is very complex, evolving in its entirety as is always the case in the dynamic reduction.

All the processes of orientation in touch were studied only in subject M since subject T was not available to be examined after 1944, and the disorder of tactile inversion was glimpsed toward the end of 1945 and extensively investigated in 1946. However, given that two quantitatively very different sensory levels can be studied in subject M, inactive and under facilitation by maximal muscular effort, and given that subject T is very similar to M under maximal facilitation, it can be presupposed the degree of the disorder in tactile orientation that subject T should suffer from. Subject M presents a tactile inversion of the same intensity as that of the visual inversion, in accordance with the characteristics of the central syndrome. This allows us to conjecture the degree of the tactile orientation disorder in subject T since it must also be equal to the degree of the visual orientation disorder that we already know well. Hoping to be more complete on a more favorable occasion, we now stick to the experimental data of subject M.

Because of the two extreme states of excitation of subject M, it is possible to make very broad generalizations to other cases with less brain impairment. We know that subject M in the inactive state presents almost complete inversion in vision and touch, whereas under maximal facilitation he only presents tilts of moderate intensity, phase III (in touch) resulting then excluded. These moderate tilts, such as that of subject T in vision, are manifestations of incomplete or frustrated inversions because the asynchrony is not sufficiently intense, and are therefore included in the same type of disorder. Thus, the Schneider patient of Goldstein and Gelb (1918, 1919), of whom nothing is mentioned about visual or tactile inversion or tilt, should present a tilt of 80 to 90 degrees in both sensory systems. It should be noted that since the sensory disorder is always of an overall type, as already exposed in this research, the alteration in tactile orientation should be a quite frequent phenomenon (at least in a moderate form) even in cases of a not very pronounced brain tactile disorder, in the same way as we stated about visual image orientation.

The *general laws* of the inversion or tilt of the perceived orientation in touch are the same as in vision, but the study in touch is much more difficult. This is due to the fact that in addition to the aforementioned difficulty in being objective, the heterogeneity of the tactile field both in texture and form, highly irregular in comparison with the visual field, greatly hinders accurate determinations. The position of a straight line in space can be perceived visually with remarkable accuracy; but the orientation of a straight line on the skin surface can only be recognized approximately even by a normal subject. It is also not easy in touch to perform a wide variety of tests and quantitative determinations such as those related to the study of visual image orientation, for example, those related to the "bi" effect (Sec. 13.3 in Vol.1), the sensory state of the receptor (Sec. 13.4 in Vol. 1), sensory adaptation, etc. In touch, the tests have to be restricted to the recruitment of orientation by intensifying the stimulus for the two sensory states, inactive and under facilitation. However, this experimental restriction is compensated by the complexity of tactile space, namely, spatiality on surface, spatiality in deep sensitivity (joints), and processes in the orientation of the body schema, all this offering a very wide field for the investigation of tactile orientation, whose phenomena can be considered among the most novel ones in this research.

Regarding the theoretical development of this research on brain dynamics, we should note the important progress made by finding in the central syndrome the spatial inversion for all the sensory systems that allow localization and orientation in space (vision, touch and hearing). In fact, the discovery of tactile inversion led us to search for a possible auditory inversion (localization in the contralateral ear) and confirm it. Thus, it is no longer possible to consider reversed vision as an isolated process without parallel in other sensory systems. Within the perfect concordance between the pathological manifestations of the various sensory systems, the privilege that vision seemed to have in altering orientation seemed strange indeed. Therefore, the generalization of spatial inversion to the remaining senses was enormously gratifying, especially because of finding such a remarkable regularity in sensory organization. The material basis of the stimulus for each sense is specific for each of them (light, touch, sound), but the functional architecture is identical for all of them. In the central syndrome studied here, the reduction of functions is similar for all of them, showing a perfect parallelism between the different sensory systems involved.

Finally, we must emphasize the great importance of the finding of tactile inversion and the generalization of the phenomenon of spatial inversion for the development of this research on brain dynamics. It means not only complementing the study of the disorders following what has already been established in vision, bringing a greater homogeneity to the central syndrome, but mainly it establishes a new theoretical stage of great interest for this research. One of the reasons for the interest is that the investigation of tactile inversion has led to a deeper understanding of the phenomenon of inversion (also in vision) according to a *spiral development*. Another reason for the interest, perhaps more important, is that the topic of spatial inversion acquires a great scope by being linked to general issues of remarkable theoretical importance, such as that of the anatomical crossings of the long pathways, and the *psychophysical correspondence* involved, as will be seen in the theory of tactile inversion

and mainly in the third part of this research. As a result, this research does not change its characteristics but undergoes an important extension of its dynamic postulates.

24.2. PERCEIVED ORIENTATION IN CUTANEOUS STIMULATION

In the case of cutaneous stimulation, it would have to be a pure chance that the orientation disorder might be perceived by the subject spontaneously, outside experimental determinations. This is because for phase III (inverted) to appear, a stimulus of moderate and precise intensity is required. If the intensity is slightly higher or lower, this phase does not appear. This refers mainly to point stimuli, since rectilinear stimuli, whether static or kinetic, very easily present very diverse tilts (rotations), at least within phase IV, which is a very broad phase. However, these rotations cannot be appreciated as directly as in the case of vision, requiring a certain amount of attention which is not usually paid to tactile perceptions, apart from the fact that isolated straight lines are not usual tactile stimuli in ordinary life. Thus, the disorder remains largely hidden and is only revealed by very thorough and varied special tests.

The general empirical conditions are the same as for vision, i.e., the sharper the tactile perception, the less the orientation disorder; and vice versa, as the perception becomes diffuse (irradiation, proximal deviation, etc.) there is a greater tendency for the inversion of the perceived spatial orientation of a tactile stimulus over the body. We shall now study, in increasing order of complexity, the inverted perception of a point stimulus, of a rectilinear stimulus and of motion on the skin.

24.2.1. Inverted perception of a point stimulus

Given the close relationship between phenomena, the study of inversion and localization in point stimulation over the skin is entirely the same as that presented on localization phases III and IV. Therefore, we shall now only complete it with some particularities, especially regarding the head.

As we know, the *head* behaves in localization tests as an independent element (see Sec. 21.2.2), and tends to deviate the stimuli towards the vertex or cranial vault (proximal sector) in medial phase II, the face acting as a distal sector. Let us recall that the sagittal midline belongs to phase II, a small parasagittal area on the cranial vault belongs to phase III, and finally the rest of the head and face belong to phase IV. In phase III, the stimuli are localized contralaterally, but always within a narrow area of the cranial vault. The characteristics of the contralateral localization can be seen in Fig. 24.1. A stimulus on the lower cheek is perceived contralaterally on the cranial vault and very close to the midline; but if the stimulus is on the temple, the localization is also contralateral on the cranial vault and less close to the midline than in the previous case, i.e., a little below, the distance between the two inverted points being less than the distance between the referred stimuli (see Fig. 24.1). All this refers to the vertical direction, and as for the horizontal, the disposition is analogous: very anterior stimuli in the head are localized contralaterally backwards in the cranial vault, and vice versa. Thus, the tendency is towards contralateral localization and with inversion

of all positions, although due to the strong proximal deviation towards the cranial vault, stimuli at the upper part are not localized at the opposite bottom part. In general, it can be stated that the point of inverted localization corresponds to the opposite end of the straight line that starts from the stimulus site and passes through the center of the head, considering in addition the strong deviation towards the vertex. Thus, there is no possibility for the inverted perception to be localized on the face, since the *inversion area* is much smaller than that of the stimulus application, and is restricted to the area most proximal to the midline of the head.

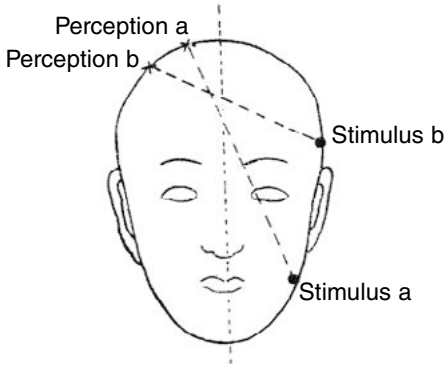

Figure 24.1. Schematic drawing of the inverted perception of stimuli in the head of subject M in the inactive state. In phase III there is a contralateral localization and always on the cranial vault, showing a certain inverted symmetry: stimuli on the lower part are localized on the upper part and vice versa. The inverted symmetry is rather distorted by the strong proximal deviation towards the vertex.

As for the transition from inverted to homolateral localization with increasing stimulus intensity, it suffices to refer to what has already been said about stimulus localization. The tests referred to in Fig. 22.10 show the asynchrony between the different phases of deviated localization and these tests are also valid to show the stages in orientation. In the case of a point stimulus, it is only possible to study the position of the felt point, whereas if the stimulus is a straight line it is also possible to study its orientation.

Let us now analyze in more detail the trajectory that links the various phases of stimulus localization as stimulus intensity varies. When the stimulus intensity decreases, localization tends towards the medial zone (centripetal trajectory) and if it increases, the opposite occurs (centrifugal). For example, if we consider a stimulus in the center of the left cheek (see Fig. 24.2), as the intensity of the stimulus increases, we obtain after phase III (contralateral localization on the cranial vault about two fingerbreadths from the midline) a forward translation on the upper part of the forehead; from here it crosses the midline toward the homolateral temple (or external angle of the left eye), and finally, with painful stimulation, the localization tends to move down the cheek approaching the stimulation site.

It should be noted that it is very difficult for the tested subject to describe verbally the exact trajectory followed by the localizations deviated from the stimulus site. This is because these are lower stages of poorly differentiated sensations that change rapidly, and also because of the difficult spatial discrimination in the cranial vault even in normal subjects. Therefore, we have to stick to a few data that give us a rough indication of the general path: upper contralateral zone, somewhat lower mid-anterior, mid-height homolateral, and finally homolateral in lower zone. All this, including phase II of medial localization, results in a *spiral development* of the sensation from the vertex to the stimulated cheek because of the combination of inversion and proximal deviation effects, with the spiral becoming more and more open. This spiral development can be considered valid within the imprecision of perception in this test since a similar trajectory is shown in other tests, thus constituting a general characteristic of spatial inversion. Moreover, this is valid for both touch and vision.

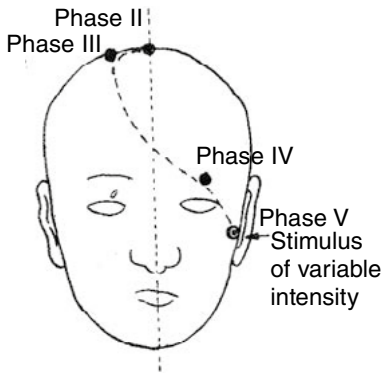

Figure 24.2. Trajectory followed by the perceived localizations when varying the intensity of a stationary point stimulus in subject M in the inactive state. Due to the contralateral translation and proximal deviation towards the cranial vault, the trajectory tends to be a spiral.

Under maximal facilitation, we already know that the complete inverted phase (contralateral localization) is not possible, and localization abruptly shifts from phase II to phase IV. Phase IV includes orientation changes that can only be demonstrated by a rectilinear stimulus.

As for the case of the *limbs*, there is nothing special to add to what was stated in Sec. 21.2.3 on the inverted phase of spatial localization, the process of inversion and deviation being illustrated in Fig. 21.4. In this figure it can be seen that, given the morphological circumstances, although it is not possible to clearly uncover a spiral-shaped trajectory, the two effects that determine it are still valid: contralateral inversion and proximal (centripetal) deviation. As will be seen, the study of orientation can be easily performed by means of a rectilinear stimulus on a limb.

Finally, the inverted perception of stimuli on the *trunk* is analogous to that on the head, although much simpler. Only the most anterior plane of the thorax corresponds to the inverted localization, since the lateral and the back already belong to

phase IV, i.e., to a distal zone. The contralateral localizations are inverted in such a way that stimuli on the upper part are perceived on the lower part, and vice versa, as shown in Fig. 24.3. Due to the special shrinkage of the inversion region, the distance between the perceived localizations is much smaller than the real distance between the stimulation points (Fig. 24.3). In this figure, the proximal deviation in phase III can also be seen, as in Fig. 24.2.

In short, as we already know from the study of tactile localization, we find *three independent zones for inverted perception.* One is the head, another is the trunk (thorax) and upper extremities, and the other is the abdomen and lower extremities. Thus, the body is segmented into these three zones in relation to tactile inversion. This is a notable difference with the visual field where the inversion occurs for the whole field (each retinal half). Therefore, a stimulus on the head cannot be perceived inverted on a lower limb or on the contralateral side of the trunk, but only on the head towards its proximal midline region, as we have seen.

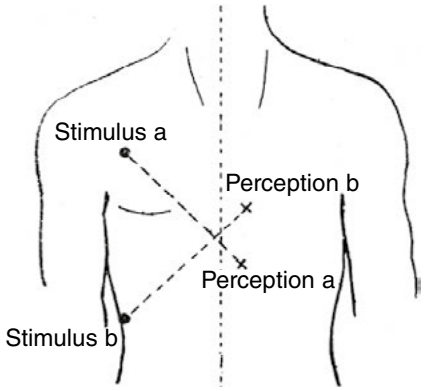

Figure 24.3. Inverted perception of point stimuli on the trunk of subject M in the inactive state. During the inversion phase, contralateral localization is obtained with opposite localization (up-down) in the vertical axis. Inversion symmetry is reduced by the effect of proximal deviation and constriction.

In relation to the meticulous tests on the inversion of point tactile stimuli is the finding of *polyesthesia* (multiple localization of a single stimulus), analogous to monocular polyopia. As in the latter, polyesthesia requires a particular study and now only the most elementary features will be mentioned. In the inactive subject M, polyesthesia has only been demonstrated on the midline of the body including the cranial vault and trunk, especially on the chest. When a point stimulus is applied on the midline of the body, we know that the phase III sensation is in the form of a band of irradiation with a central point located on the midline, i.e., at the same place of application of the stimulus, without deviation. In this situation, another point usually appears, much weaker in intensity and always on the right side, about two finger-breadths away from the first point, just at the lateral boundary of the irradiation band. If the stimulation is intensified, the sensation tends to change to an oval shape

(always in the midline) and the double localization disappears. It has not been possible to observe this additional localization on the left side. There are strong analogies with polyopia since in the latter the polyopic deviation appears on the right in central vision and when the tilt of the image of the test arrow is about 30°-40°. Polyopia disappears in good vision and without image tilt perception, whereas on the contrary, it becomes triplopia (a virtual image on each side) when vision worsens and the perceived tilt of the central image is greater but without reaching 90°. Whereas in the visual field polyopia is easily obtained in peripheral vision, in touch it has been impossible to obtain polyesthesia outside the referred midline. There are certain similarities between vision and touch in the way in which polyopia and polyesthesia appear, but the greater difficulty of examination and objectification of tactile phenomena may perhaps be the cause of an incomplete parallelism in this phenomenon of multiple localization. From the results obtained, this phenomenon seems to be less pronounced in touch than in vision.

24.2.2. Inverted perception of a rectilinear stimulus

If the stimulus has the form of a straight line and is applied statically on the skin, a deviated and contralateral perception is also obtained, and in addition, a tilted perception analogous to the already studied tilted visual perception of a vertical arrow. Thus, the study of perceived orientation in touch is more complete by means of a rectilinear stimulus.

This type of test can be performed in any region of the body. However, it is easier on the upper limbs since, as in the study of stimulus localization, they provide ample space to carefully follow the process if the stimulation is applied quite distally. In the test, the straight edge of a metal sheet is placed on the back of the left hand, being in contact about 6 cm along the hand (see Fig. 24.4). The intensity of the stim-

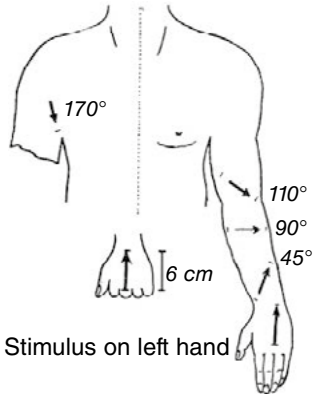

Figure 24.4. Perceived position of a 6-cm straight line stimulus applied with varying pressure on the back of the left hand of subject M in the inactive state. The localization and orientation of the straight line in the course of the process is indicated. The maximum rotation takes place contralaterally. The degree of rotation correlates with the degree of proximal deviation (see Table 24.1).

ulus is determined by applying to the metal sheet weights ranging from 200 g to 3 kg. In some tests it is possible to use the compensation method analogous to that used in vision (tilting the test arrow in the opposite direction to be perceived vertically upward). But due to the normal tactile imperfection to perceive orientation accurately and, above all, due to the irregularities of the back of the hand (tendon prominences, etc.), such a procedure is much less useful than in vision. To overcome certain shortcomings and confirm some data, it is advisable to use a moving stimulus by tracing a straight line on the skin, which is easier and quicker to apply on most areas.

Table 24.1. Perception of a 6-cm straight line stimulus applied with varying pressure (applied weights in grams) on the back of the left hand of subject M in the inactive state. See Fig.24.4.

Stimulus (grams)	Perceived orientation	Phase, Localization	Size	Tactile sensation
280	Looks vertical	III, Contralat. arm close to shoulder	~0.7 cm?	Pressure
340	Tilted?	IV, Lower half homolat. arm	May be larger	Very slight pain?
500	Oblique	IV, Homolat. elbow or below	~1.5 cm?	Mild pain
850	Transversal	IV, To the half of the homolt. forearm	Larger	Painful
1000	Somewhat oblique	IV, More distal	Like previous	More painful?
1300	Very oblique	IV, To the wrist	~ 3 cm	Severe pain
2000	Close to vertical	IV, To the hand	Longer	Acute pain
3000	Almost or totally vertical	V, Hand dorsum	~ 4 cm	Unbearable pain

By varying the intensity of pressure on the skin, the corresponding perception evolves in a complex way, changing in unison the following features: localization of the perceived straight line, its size, tactile irradiation (diffuse perception of the edges), orientation, and the corresponding sensation (diverse pain). The evolution of the orientation is roughly as follows as the intensity increases: In the inactive state, only the maximum inversion is contralateral (upper third of the contralateral arm), although homolaterally the rotation of the straight line is very considerable, probably at least 110°, until it straightens while proximal deviation decreases. Under maximum facilitation by muscular effort the straight line is always localized homolateral-

ly, but the maximum limit of rotation is quite large, about 110°. Proceeding carefully, it is not difficult to obtain a large number of positions and orientations of the perceived line, which allows to get a very good idea of the development of the process. But due to the characteristics of the test, the tested subject must verbally report the situation reached, which entails obtaining less accurate data than in analogous experiences on visual image orientation, in which the subject must only report when the arrow is seen in a vertical upward position (compensation method). However, repeated tests have shown us that it is possible to obtain sufficiently reliable data to evaluate the process quantitatively and represent it graphically. Figure 24.4 graphically illustrates the process schematically, and Table 24.1 shows the results obtained.

The following development is obtained when the pressure is increasing. At first, when a well-developed phase III is reached, something like a very short line in the center of a more elongated and diffuse irradiation can be perceived in the upper part of the contralateral arm. Its orientation is difficult to perceive accurately given the small length and faintness of the line, and may give the sensation that the line is oriented vertically, i.e., following the axis of the limb. However, by appealing to the inversion of motion on the skin when the stimulus has the intensity of phase III, it is shown that a motion on the dorsum of the hand from top to bottom (from proximal to distal) gives rise to a sensation of brief and rapid motion on the upper part of the contralateral arm and with practically inverted direction, i.e., from bottom to top. There is possibly, as in vision, a tilt limit with respect to the vertical, without a complete inversion of 180°, but it is technically difficult to prove this in touch. Phase III ends soon, followed by the transition to the homolateral side and the beginning of phase IV. As already noted in the tests of localization of a point stimulus, it is difficult to determine accurately at what level of the limb the change of side occurs when the stimulus intensity on the dorsum of the hand increases, but it most likely takes place somewhat above the elbow. Thus, a proximal part of the arm corresponds to the contralateral localization, and the remaining distal part is in the side homolateral to the stimulus.

The most noteworthy feature of phase IV now is the significant orientation disorder involved, perfectly demonstrated with the test of perceiving a straight line. It turns out that the localization defect (centripetal deviation) is always correlated with the orientation defect. Thus, in the case of maximum facilitation, which does not allow contralateral localization, it is possible to find a strong rotation of the straight line greater than 90°, which would allow to set the limit of orientation disturbance in homolateral localization.

A rotation of the perceived line that is easy to determine is that of 90°, since when the straight line stimulus is applied on the longitudinal direction of the limb, the altered sensation must correspond to a totally transversal orientation, without obliquities, and which is localized well below the homolateral elbow. It can be said that this transverse subjective line (midway of full inversion) is situated towards the middle of the limb. As the intensity increases, the perceived length of the straight line increases, which is perceived more clearly from the transverse position onwards. Finally, very high intensities are necessary to try to suppress the centripetal deviation and the inversion residue. Unbearable pain is then produced which easily triggers defensive muscular contractions, impurifying the state of inactivity. When reaching the maximum recruitment

(sensory growth), it is very difficult to say if there are abnormal remnants of deviation and tilt, but in analogous tests in vision, their existence is verified with relative ease, and here in touch they should also be admitted. Thus, at the end of the process, the perceived line probably does not exceed 4 cm (compared to 6 cm for the test line), to which must be added a weak centripetal deviation of about 2 cm and a rotation of about 10°, although all this has been deduced theoretically and according to other tests.

These tests are completed by considering the action of facilitation by maximum muscular effort. The results obtained are shown in Table 24.2.

Table 24.2. Same conditions as in the previous Table 24.1 except that subject M is under facilitation by maximum muscular effort.

Stimulus (grams)	Perceived orientation	Phase, Localization
150	Somewhat tilted	IV, Lower third homolat. arm
200	About the same	IV, Homolat. elbow
300	Transversal	IV, Mid homolat. forearm
370	Oblique	IV, Toward homolat. wrist
500	More vertical	IV, Homolat. wrist
1000	Vertical?	V?, Hand
1500	Even more vertical	V, More central on hand

It was already indicated that under maximum facilitation the maximum rotation goes beyond 90°, i.e., a behavior analogous to that observed in vision, but the line does not pass to the other side and is positioned homolaterally, a little above the elbow. This maximum rotation is reached under facilitation with about 150 g on the stimulus, whereas about 500 g are required in the inactive state. Thus, facilitation saves about two-thirds of the stimulus intensity, this being valid for all other tests more or less. Apart from the difference in stimulation for the two states referred to (inactive and under facilitation), it can be stated that the correlation between the degree of rotation and the position on the limb is the same for both states; for example, the transversal orientation of 90° corresponds towards the middle of the homolateral forearm for the two states, and so on for the other orientations.

Under facilitation it is observed that not only are thresholds much lower but also the intervals between sensory levels are much smaller than in the inactive state. In short, there is an increase in excitability leading to greater sensitivity and faster variation in the process under consideration.

The considerable rotation of the perceived stimulus in a state of relatively moderate functional impairment such as that of subject M under maximum facilitation,

points to the fact that orientation disorder in touch should be very frequent, as is also the case in vision, even if only as small tilts, less than 30° as should correspond to subject T. In subject T, the tilt should be strongly intensified after epileptic seizures, or more moderately intensified by the ingestion of alcohol, as occurs in other tests already described in the case of vision (see Sec. 13.1.1 in Vol. 1).

In summary, whenever a proximal deviation in tactile localization occurs, an alteration of the perceived tactile orientation, which always accompanies such a deviation, can be presumed. Thus, it is shown that the localization disorder is more complex than it appears in a simple examination with a point stimulus, since spatial localization (exact localization of a sensation) involves both proximal deviation and rotation. The latter only revealed by a rectilinear stimulus as shown in Fig. 24.4. By plotting the data from the above tables, not forgetting that it is not possible to achieve as much precision as in vision, we obtain the curves of Fig. 24.5, which undoubtedly provide a valuable indication of the functional law of tactile orientation disorder.

It is evident that these curves are similar to those of visual image orientation recruitment as a function of stimulus intensity (Fig. 13.3). It can be seen that at the beginning of the curves the sensory growth is fast and then becomes slower and slower following the general type of logarithmic growth according to the law of Fechner (1860). All that has been said about curves of a similar type, as those obtained in vision, is now applicable. Since it is difficult to use the compensation method with the same accuracy as in vision, it is impossible to determine exactly the maximum limit of rotation (inversion) in each of the two states studied. However, in view of the approximation that can be obtained, it is quite certain that it is of the same order as in vision, i.e., in the inactive state, 160° to 170°, and under facilitation, about 110°. When starting from the inactive state with very pronounced rotations, and changing to the state of maximum facilitation, the facilitation effect is very easily noticed, as can be seen in the curves of Fig. 24.5, the correction in the rotation being about 80° to 90°.

Since the development of normal orientation (re-inversion) with increasing stimulus intensity is correlated throughout its course with the growth of the body schema (correction of the deviation), both phenomena can be depicted simultaneously, as shown in Fig. 24.6. In fact, the development of localization (Fig. 22.3) and that of orientation (Fig. 24.5) are quantitatively similar. Such Fig. 24.6 provides us with a complete illustration of the actual process that takes place in the perception by subject M of a rectilinear stimulus.

As for the *direction of rotation,* the characteristics are also the same as in vision (see Sec. 12.3 in Vol. 1). Thus, rectilinear stimuli in both halves of the body rotate in opposite directions as the stimulus intensity decreases. On the right half of the body the rotation is counterclockwise and on the left half it is clockwise, i.e., outward in both cases. In the midline of the body, whether it be the forehead, chest, etc., the rotation is counterclockwise, i.e., toward the right side. As we have already seen, the rotations in each half of the retina of a single eye are in opposite senses. However, in the center, the rotation is outward in each eye. The sense of rotation can be easily determined on the different parts of the body by stimulating with a rectilinear motion over the skin and exerting a little facilitation in such a way that only a moderate rotation of about 40° occurs.

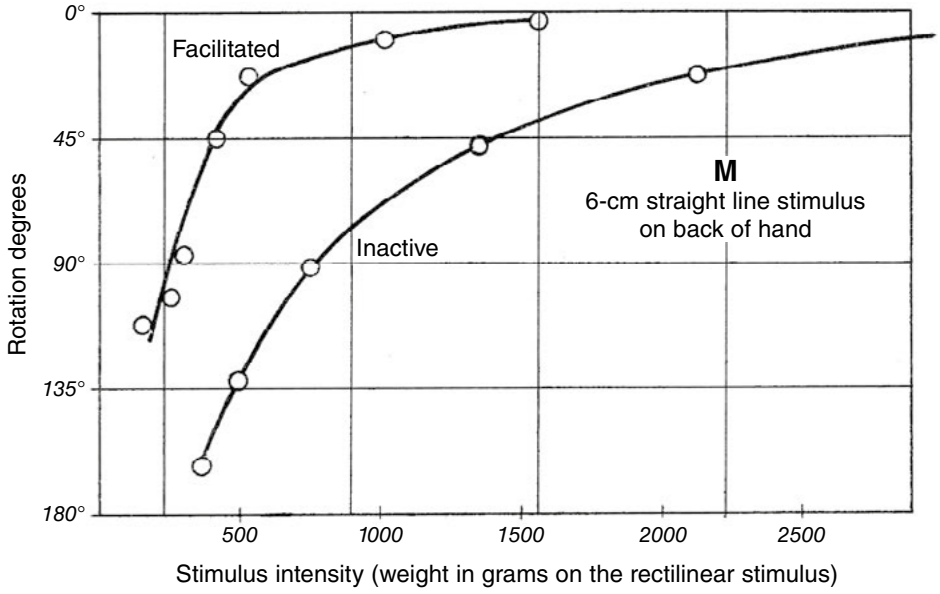

Figure 24.5. Perceived orientation (in rotation degrees) by subject M of a straight line stimulus as a function of its intensity (weight on the stimulus), in the inactive state and under facilitation by maximal muscular effort. Note the difference between the two curves, especially the different maximum rotation limit and the slope at that point (the lowest one) in the corresponding curve.

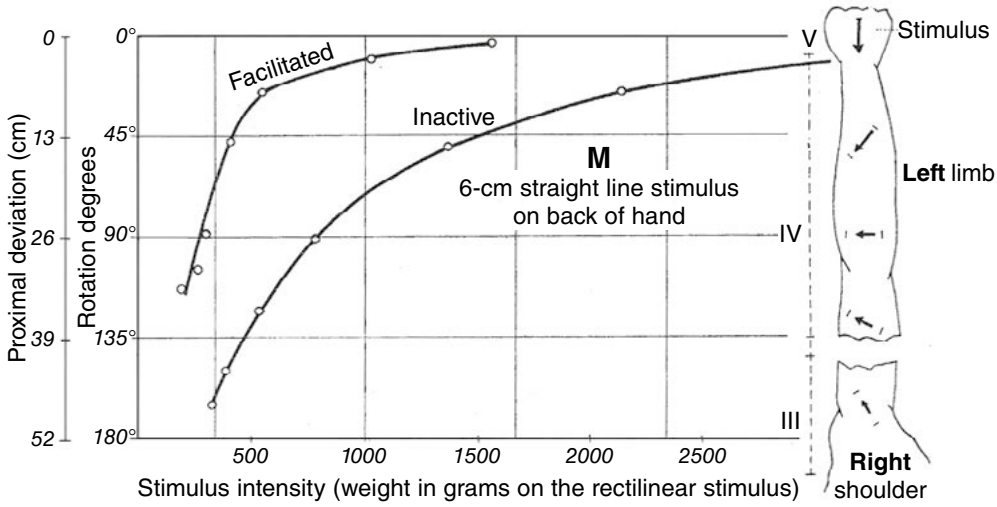

Figure 24.6. Same as Fig. 24.5 but including proximal deviation (in cm). On the right, a schematic drawing of the size, deviation and corresponding perceived orientation on the right shoulder and left arm.

The pressure test on the rectilinear stimulus can be performed on any part of the body, although with varying experimental ease and accuracy, obtaining results of the same type as in the hand. On the cheek, the test can be performed with a much lower stimulus intensity than on the back of the hand, either because of thinner skin or greater nerve sensitivity. The subject must be appropriately positioned in the inactive state such that the edge of the metal sheet rests horizontally on the left cheek.

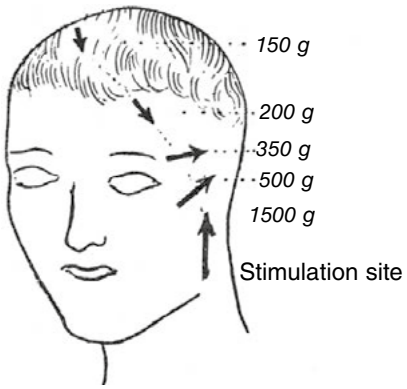

Figure 24.7. Test with a rectilinear stimulus on the left cheek of subject M in the inactive state. As in the upper extremity, most of the rotation corresponds to the homolateral side. Note the localization corresponding to 90°.

Figure 24.7 shows the stimulus location and the perceived localizations and orientations according to the weight applied on the rectilinear object. With a weight of 150 g, a weak localization is felt on the opposite cranial vault (right); with 200 g, a sensation of oblique line is obtained on the left forehead; with 350 g, the line is better felt, localized horizontally between the eye and the homolateral ear (left temple); finally, with 500 g, the line is felt as oblique (Fig. 24.7), more toward the cheek, without reaching the place of application of the stimulus; being necessary about 1500 g to reach this place. Thus, the values obtained correspond to those of the hand test under facilitation (see Table 24.2). Compare Fig. 24.7 with Fig. 24.2 with respect to the path followed by the localization.

24.2.3. Inverted perception of motion on the skin

In the case of a moving stimulus on the skin, the same characteristics as in the static straight line case are present, but in addition it provides other information such as the direction of the perceived motion and the alteration of the perceived trajectory. It should also be considered that intrinsic changes in trajectory and velocity occur in parallel with the degree of inversion, as mentioned previously in Sec. 23.2.

The direction of motion and the corresponding localization are of course dependent on the stimulation conditions, namely stimulus intensity (cutaneous pres-

sure of the moving stimulus) and its duration (path length and speed). Any variation of these factors influences the sensory recruitment (sensory development) and thus the direction of motion. For a given motion on the skin with a fixed trajectory, constant intensity and moderate speed, capable of eliciting a very little altered perception (in localization, direction of motion, length and speed) in the inactive subject M, it happens that as the speed increases, the anomaly in orientation and other associated factors become clearly evident until descending to phase III. In this phase, the localization becomes contralateral and very close to the midline, the speed felt is the maximum possible and the direction of motion is felt practically inverted (close to 180°). The fact of increasing the speed of the stimulus implies a shortening of the stimulation time, thus preventing the development of the function, which then shows asynchronous levels with different degrees of inversion. The result obtained is the same as in the tests on visual perception of the direction of motion of a moving object at different speeds (see Sec. 13.5 in Vol. 1). Obviously, any variation of the other stimulus parameters (intensity, path length) produces a similar effect.

When the maximum inversion is obtained, the felt path is approximately 1/10 of the real path of the moving stimulus. Figure 24.8 shows the complete inversion of motion on different parts of the body. In addition to the direction of motion, the other concurring features are explained by the fact that this stage belongs to phase III. There is an intense proximal deviation towards the midline, as well as a sensation of both strong path reduction and high speed, all as a result of the dynamic reduction in the *space and time sensory dimensions,* according to what has already been studied. The motions represented in Fig. 24.8 have vertical orientation, but any other orientation shows the same result, so a motion from front to back originates a sensation from back to front, and so on for other cases.

The test with the static stimulus of the straight line is particularly suitable for a more precise determination of the degrees of tilt (rotation) as a function of stimulus

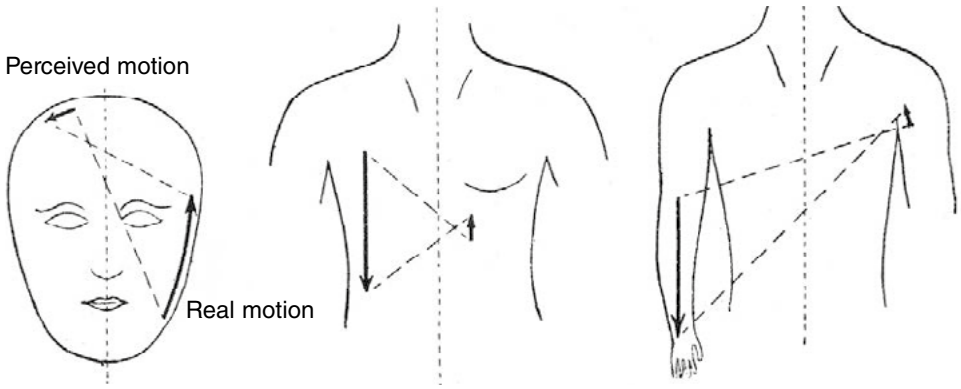

Figure 24.8. Total reversal of motion over the skin in different parts of the body of subject M in the inactive state. The large arrow corresponds to the moving stimulus and the small arrow to the perceived motion, which shows reversal of motion direction, intense centripetal deviation (phase III) and strong spatial and time shortening that produces a sensation of increased speed.

intensity, i.e., for the detailed study of the evolution of the process, whereas the test with moving stimuli is more appropriate for obtaining general data on the direction of rotation and inversion. The examination of the direction of the felt motion by means of moving stimuli offers an important complement to the test with the static straight line, both because of the ease of performing it and because it reveals more clearly the perceived orientation.

When the moving stimulus can promote a greater recruitment (sensory growth), there is no longer such a complete inversion nor, consequently, contralateral localization, but only different tilts on the side homolateral to the stimulus. An interesting test is the one performed by moving a stylus with moderate pressure on the skin, uniform moderate speed and very long trajectory over almost the entire upper limb of subject M in the inactive state. A homolateral, non-inverted trajectory that deviates considerably from the real trajectory is then obtained subjectively, as shown in Fig. 24.9.

Because of the great length of the real path and the moderate pressure, it results that at the beginning of the perception, the direction of the felt motion undergoes a great deviation, and subsequently, due to the continued development of the function, the alteration of the direction tends to be progressively corrected (see Fig. 24.9). There is undoubtedly a cumulative action on the nervous centers, with the result that the final effect differs from the initial one, even though the stimulation is uniform in all its aspects. The path length is about 35 cm, from the lower third of the arm to the middle of the back of the hand. However, the path felt by the subject is only about 10 cm, so it has been reduced to about 1/3 of the real value. At the beginning of the perception, the direction of motion is felt rather inverted, it could be evaluated at about 120°, but it lasts very short, and the path then becomes somewhat larger and transverse (90°), from which it gradually changes to less and less rotated and longer paths. Finally, a certain deviation of about 10° always remains. In short, the sensory level is always growing during the motion of the object, and since the length of the path and the degree of inversion evolve together (the greater the inversion the greater the spatial shrinkage), the mentioned deviated trajectory is obtained, which in reality would be the *spiral deviation* referred to above. Likewise, the uniform speed of the real motion should undergo a change similar to that of space, as we know; thus, a high speed of the moving stimulus is felt at first, and gradually decreases as the trajectory straightens out and increases in length. As for localization, a proximal deviation of the entire trajectory is obtained, which is located between the lower third of the arm and the upper forearm, as shown in Fig. 24.9.

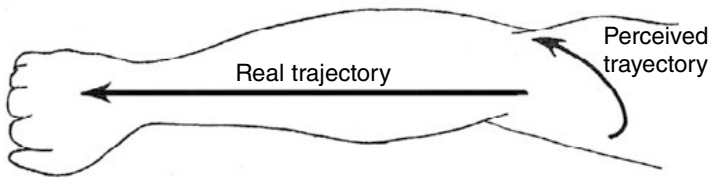

Figure 24.9. Real trajectory and perceived trajectory of a stimulus moving over the skin of subject M in the inactive state. The perceived trajectory is deviated proximally, with a shorter path (1/3), and is perceived with higher speed, especially at the beginning.

This alteration of motion that involves so many aspects is a logical consequence of the impairment of various factors which come into play, and a great deal of experimental thoroughness is necessary to bring out such an alteration. If the subject under examination disregards the initial path due to its smallness and sensory weakness, he will only state to have felt a somewhat oblique motion on the end of the arm; but if he pays more attention, he will become aware of the initial path resulting from the initial inversion.

In relation to this alteration of the trajectory is the deformation of figures drawn on the skin (see Sec. 23.3), unless the drawing is of large size and is made with great pressure.

24.3. PERCEIVED DIRECTION OF JOINT MOVEMENT. DEEP SENSITIVITY

Here we study the perceived direction of joint movements produced both passively and actively. We already know that there is a parallelism between the perception of motion on the surface of the skin and the perception of deep (joint) movement. The most important phenomena are manifested in passive movements of the limbs, the head, and in the peculiarities of active (voluntary) movements.

24.3.1. Inversion of passive movements in the extremities

For the study of the direction of movement, it is not possible to use repeated stimulation by means of an oscillatory movement of an extremity. It is indispensable to perform only a single movement in order to observe in a well-defined manner the anomaly in the direction of the perceived movement. Such movement must be of large amplitude and vigorous in order to elicit sensation of movement in the inactive subject M, as explained in Sec. 23.4, which is now of great importance. Depending on the energy used, very large in all cases, the localization of the movement undergoes a more or less strong proximal deviation. In conjunction with this deviation there is a certain degree of inversion in the direction of the perceived movement as well as intrinsic modifications of it, analogous to what occurs with a stimulus moving over the skin.

When testing the index finger (metacarpophalangeal joint) of subject M in the inactive state, the following is obtained. For a given energy applied to the finger in a passive movement, the felt movement is localized in the contralateral shoulder joint in such a way that when the finger performs an extension, a flexion of the arm is perceived as sown in Fig. 24.10, *1*. This movement is felt to be very rapid and very short in its path compared to the real movement of the finger. If, for example, the amplitude of the finger movement is about 100°, the amplitude of the perceived movement in the arm would be about 10°, just enough to be perceived by the patient. The direction of the movement perceived is practically inverted, being impossible, due to the experimental conditions, to appreciate exactly what may be missing to reach the absolute inversion.

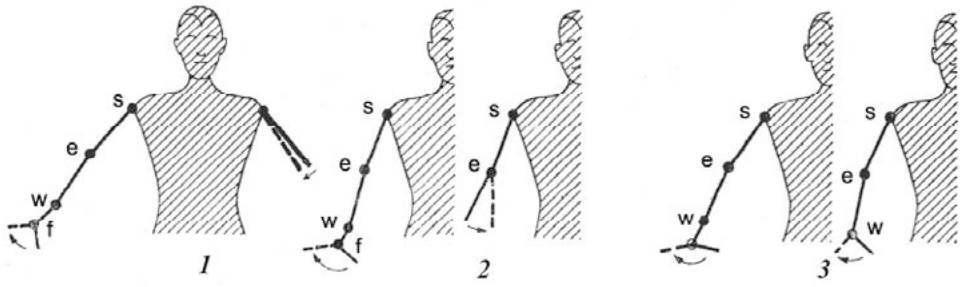

Figure 24.10. Schematic drawing of inversion and proximal deviation in passive joint movement of a finger in subject M in the inactive state. ***1***: A vigorous finger extension produces a sensation of weak flexion of the arm at the contralateral shoulder. ***2***: A more vigorous extension of the finger causes sensation of forearm flexion at the homolateral elbow. ***3***: Much greater amplitude of the finger extension and with more energy produces a sensation of extension in the homolateral wrist.

If the passive extension of the finger is performed more strongly, the sensation of movement is perceived as a flexion of the homolateral elbow (Fig. 24.10, *2*), that is, there is a considerable, albeit incomplete, reversal of the direction of movement. Finally, a finger extension with the maximum possible amplitude and energy (it is convenient to press on the articular limit) produces the sensation of a deep movement at the wrist, with approximately the same direction of the finger movement (extension), as shown in Fig. 24.10, *3*. We already know that no matter how strong and large the movement is, it is not possible for subject M to localize it at the real stimulus site (metacarpophalangeal joint).

These abnormal localizations (contralateral shoulder, homolateral elbow, wrist) and their respective directions of movement are in perfect agreement with the results of the test with the straight line stimulus, in which the tilt felt correlates with the proximal deviation. Thus, at the level of the elbow, the degree of inversion of the straight line is still very pronounced, whereas at the wrist, the line feels rotated only about 30°. In joint movement, it is difficult to obtain further data from the patient on the degree of inversion and whether it is clockwise or counterclockwise according to the intensity of the stimulus, so we must be satisfied with a rough indication of the joint extension and flexion. However, it is very evident that the perceived movement on the homolateral side is much larger and slower than on the contralateral side. At the wrist, the movement is felt at least three times larger and slower than when felt at the shoulder.

The same test performed under facilitation by maximal muscular effort excludes contralateral inversion at the shoulder, as we know. Depending on the stimulation energy, flexion is felt at the elbow, or extension at the wrist and even the finger, the latter movement being possible to be perceived under facilitation. When the localization is at the wrist, in addition to extension, possible rotation is felt, whereas at the finger, the perceived extension is more correct. When the localization is felt in the finger, the perceived movement is the largest amplitude and slowest of all those that can be felt in the different joints with proximal deviation.

Further tests in the inactive state using passive motion of major joints serve to complete the data on movement direction. Thus, by passively abducting the right arm about 90°, a sensation of adduction of the contralateral arm of about 10° and with very rapid movement is obtained (Fig. 24.11, *1*). The subject cannot say whether he also feels the arm deviates a little backwards or forwards, but in some test he has felt that kind of deviation, in which case there would not be a complete 180° inversion. If the passive movement of the arm is forward, the felt movement is backward in the contralateral arm as shown in Fig. 24.11, *2*. But if the passive abduction movement of the right arm is much more intense than in case 1, then there is neither contralateral localization nor reversal of movement direction. The perceived movement is localized in the same shoulder and undergoes a partial inversion of only about 90° in such a way that the abduction is felt as a backward movement, as shown in Fig. 24.11, *3*.

The same happens for the other joints, both of the upper and lower extremities. No further details seem to be necessary.

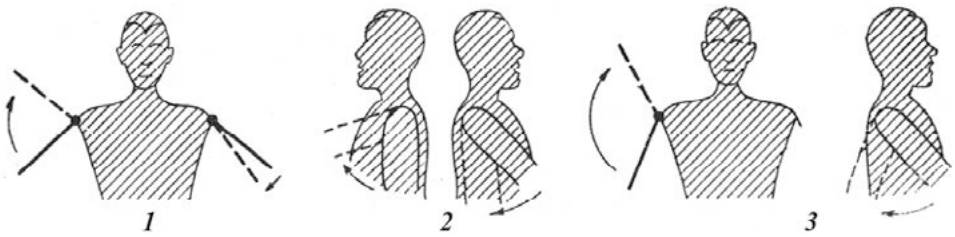

Figure 24.11. Schematic drawings of the reversal of direction of passive articular movement in the arm of subject M in the inactive state. ***1***: Abduction of the arm is perceived as adduction of the opposite arm (complete reversal of direction in the contralateral arm) and in one tenth of amplitude. ***2***: Arm forward is perceived as a weak and rapid backward movement of the other arm (complete reversal of direction in the contralateral arm). ***3:*** Very vigorous abduction is perceived as a backward movement of the same arm (partial reversal of movement in the same arm).

24.3.2. Inversion of passive movements of the head

The direction of the articular movements of the head can be studied more easily and accurately than in the case of the extremities, and because of its importance, it deserves a new subsection.

As we already know, in this case no deviation of localization is possible, and in all tests the movement remains referred to the same site (neck joint). Therefore, it is necessary to pay attention only to the direction of the joint movement according to the intensity of the stimulation.

In subject M in the inactive state, a passive head movement forward (flexion), or backward (extension), with moderate intensity but sufficient to produce excitation, is perceived as follows. If the movement is forward, the perceived movement is reversed although not completely, so the exact direction of the perceived movement is backward and to his right as shown by the black arrow in Fig. 24.12, *1*. If

the movement is backward, the perceived movement is forward and to his left as shown by the black arrow in Fig. 24.12, *2*. Therefore, the rotation that takes place is always clockwise, the head behaving as an organ of the midline of the body.

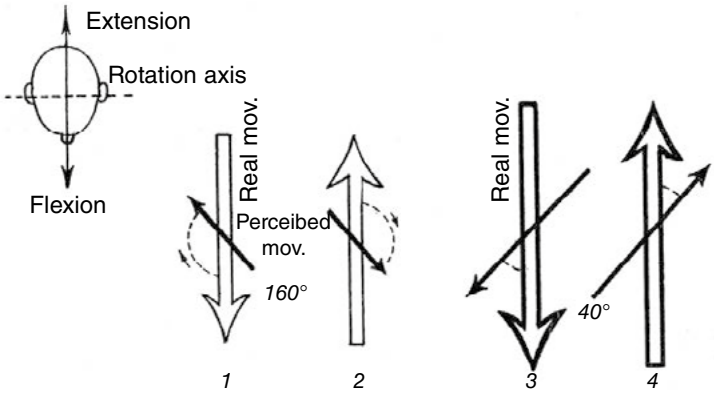

Figure 24.12. Diagram on the inversion of the passive movement of flexion and extension of the head of subject M in the inactive state. If the movement is large but not vigorous, the perceived movement is rather inverted (thin small black arrows in 1 and 2). If the movement is very large and very energetic, the change in direction of the perceived movement is much smaller, as indicated in 3 and 4 (thin black arrows). Note that the rotation perceived is always clockwise, and that the different perceived length of the path is correlated with the degree of inversion.

A very large (to the limit) and extremely intense movement is necessary for there not to be such an accentuated inversion, and then the perceived direction is rotated about 40°-50° with respect to the direction of the real movement. Thus, the flexion movement is felt in the forward direction and to his right (thin black arrow in Fig. 24.12, *3*), and the extension movement is felt in the backward direction and to his left (thin black arrow in Fig. 24.12, *4*). These perceived directions are accompanied by a longer and slower felt movement than when there is an almost complete inversion. When the passive movement is of intermediate intensity between the two previous types, the direction of the perceived movement is rotated about 90°, and therefore the flexion-extension movement causes a sensation of movement with a transverse direction from side to side. Flexion causes a sensation of movement to the right, and extension, to the left.

Under facilitation by muscular effort, if the head movement is very weak, some inversion can still be obtained, although to a lesser degree (about 100°) than in the inactive state. If the amplitude and speed of the movement is moderate, the perceived direction of the movement usually changes only by about 40°. If in addition the facilitation is very strong, the perceived direction changes only very slightly, and if the passive movement is a little larger, there is practically no subjective change in the direction of the movement. All this is verified by means of the compensation method, that is, by giving some real lateral tilt to the passive flexion or extension movement so that the sensation is not laterally deflected.

Other tests by means of passive head movement in other directions provide similar results. Thus, in the movements of rotation from side to side in the frontal plane, it is observed that a movement to the left is felt as a movement with some forward flexion and to the right, i.e., there is almost complete inversion. It should be noted that for the subject to be able to describe the felt movement with ease, the most appropriate movement is flexion or extension.

Aside from these directional disorder in passive head movements, this region has been especially significant for the remarkable phenomena of sensory-motor incongruence in active (voluntary) head movements, which will be studied below.

24.3.3. Inversion in active (voluntary) movements. Incongruences

When studying the disorder of the direction of movement through passive movements, the patient could remain indefinitely ignorant of his disorder. This is because with his eyes closed, ignoring the type of stimulus and limiting himself only to the perceived sensation, it is impossible for him to notice discrepancy between cause and effect. A very different situation occurs when the patient takes an active part in the stimulus by producing it himself. Thus, when examining the alteration of the perceived direction of voluntary movements, it is observed that, in the range of stimulation intensity that causes subjective inversion, a significant disparity appears between the direction of the executed movement and that of the perceived movement, opposite to each other due to the effect of inversion. This is a *sensory-motor incongruence of the movement direction,* and can be noticed by the patient himself. It is clear that, from the above, this disparity extends to the intrinsic character of the movement, since it is perceived as having a shorter path and higher speed than the movement performed.

The mechanism of execution of a voluntary movement by subject M will be studied in detail in a separate section. Suffice it to say now that if this subject is in the inactive state, any voluntary movement is impossible for him. He needs a certain degree of facilitation for the body schema to be activated and thus to guide the motor impulse. If the facilitation is weak (sustained muscle contraction of moderate intensity), the movement short and of a single joint excursion (appropriate to arouse a kinetic sensation in the initial phase), then a reversal of the direction of movement is perceived along with a proximal deviation and a contralateral localization. For example, closing one hand produces the sensation of abduction of the opposite arm. It should be noted that in the above circumstances, facilitation is used rather to "find" and perform the movement that the subject is asked to execute. This facilitation, being weak, seems to modify very little the movement in question, allowing a very pronounced inversion to be perceived.

A discordance occurs between the motor command, usually well executed, and the sensory response indicating a completely different location and direction of the movement. Let us analyze such a curious phenomenon in order to determine what kind of independence exists between the motor function and the sensory function involved in the voluntary action, and what states of adaptation can be achieved.

The order to perform the movement is carried out by the subject with a certain delay due to the time spent in activating the body schema and selecting the move-

ment. This movement, being very simple, does not necessarily present pathological deficits (ataxia, etc.), although the proprioceptive control is not normal. In such conditions, the subject may become aware of the aforementioned sensory-motor incongruence. In fact, if asked if he is sure he performed the assigned command, or if he is confused by the disparity, he responds that he knows he moved his hand as commanded even though he felt the movement in the contralateral shoulder. He seems to be sure of the specific movement he performs, but various tests attest that this conviction is very shaky, as seen in the following test. Instead of telling him to close a certain hand, he is commanded to move an arm (without specifying the side), so the verbal command is nonspecific. He perceives an opposite movement in the contralateral arm, but spontaneously tends to believe that the moved arm is the arm where he perceives the movement. This is because the executed movement has been improvised at random without taking on a definite character, and it seems that only the sensory effect acquires prominence. Thus, when he is asked about the movement performed, he answers with total certainty referring to the sensory sensation and not to the motor action. This means that he ends up ignoring his own voluntary movement because a *change* of reference is established to overcome the aforementioned sensory-motor incongruence which is thus suppressed, and a certain functional balance appears. In this test there are special conditions, but even in the case of ordering the subject to execute a well-specified movement that he succeeds in carrying out, it always happens that finally, when continuing the test, the movement felt "absorbs" the one executed voluntarily. Thus, it may happen that the subject believes that he has made a mistake in executing the order, or that he does not remember which side was indicated to him.

This process is studied in a more complete and simple way by means of *active head movements.* In particular, the aforementioned change of reference with the consequent functional balance excluding incongruences becomes clearer. Of course, all these phenomena, like many others, refer to tests within certain limits, and outside them, in the ordinary life of the subject, these phenomena do not arise, or go unnoticed, not compromising his activity. Since the subject must already apply some facilitation to promote the action of voluntarily moving the head, the movement commanded to him must be of small amplitude to obtain an inversion of about 150°. Thus, when the subject, complying with the order, performs a small flexion of the head, he feels an extension. The motor impulse has been well guided, but if he continues to pay attention to the order, he must feel an incongruence due to the sensation of inverted direction when the impulse of the movement performed almost disappears. He then tries to rectify by himself to fulfill the command, in the following way. He is spontaneously guided by the sensory information, and automatically performs the opposite movement to the one ordered to achieve concordance between the order and the sensation, disregarding the motor engram.

When he remains sufficiently attentive to the course of the process, he is often seen to first move his head in the direction ordered; then when he notices sensory incongruence with the order, he makes small trial-and-error movements to obtain articular information, and finally initiates the opposite movement to that ordered, which suppresses the aforementioned incongruence. In such a situation, an automat-

ic change of reference has occurred, i.e., the motor order ceases to prevail, and the action takes as reference the sensory factor which is the only one that controls it. The patient does not really know what voluntary movement he has executed, since he always responds according to the sensory information to which he is totally adapted. At this point, if the patient continues to receive orders indefinitely, he will constantly have the sensation of fulfilling them correctly by performing a movement opposite to the one ordered. The sensory-motor incongruence is thus overcome, and a balance is reestablished by disregarding the motor element, since it is not possible otherwise in such circumstances. This leads to the remarkable consequence that the active (voluntary) movement has become, through this change of reference, a *pseudo-active movement* bordering on passive movement. This whole singular process is illustrated by the diagram of Fig. 24.13.

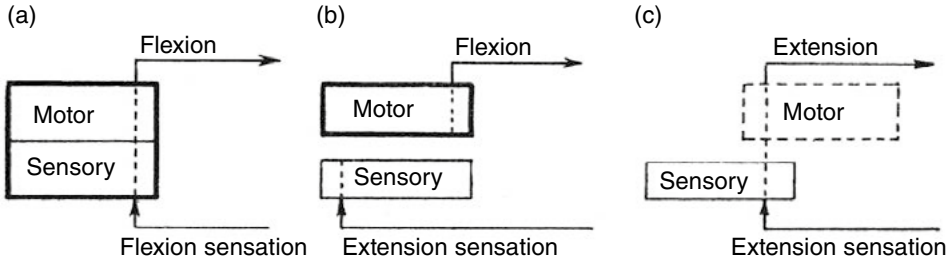

Figure 24.13. Diagram to illustrate the incongruences and the change of reference in active (voluntary) movement of the head. (a) Normal subject: an active flexion movement produces a sensation of flexion. (b) Sensory inversion stage in subject M under the command to perform a flexion: an active flexion movement produces a sensation of extension. There is a sensory-motor incongruence due to the sensory inversion. (c) Subject M, who undergoes inversion, continues to receive the command to perform a flexion. However, once the sensory factor is manifested (extension), it ends up absorbing (dominating) the sensory-motor complex. The motor system then suppresses its own direction and takes as reference the sensory perception which guides the action.

In short, it can be stated that the motor action can run by itself, but then it is very unstable and diffuse since it lacks the support of the epicritical proprioceptive sensitivity, and in addition, it is interpreted in a contrary way due to the sensory inversion. Thus, an articular adaptation (sensory predominance) is automatically established as the only resource to achieve congruence. At that point the active action becomes a quasi-passive action since the character of the executed movement is ignored. The impulse is "promoted" by the motor system but is "guided" by the inverted sensory cue.

This automatic change of reference is not an isolated fact in the series of anomalies of our patient M. It could be compared to the visual phenomenon that occurs in tests on "induced motion," i.e., seeming movement of the visual scene, which is fixed, as the subject's body rotates (Sec. 16.2 in Vol. 1). This latter phenomenon is due to a failure of tactile information in the process. Such a phenomenon may also occur in a normal subject, for example, in the well-known seeming motion of the

moon through the clouds, among many other cases. Thus, a new type of perception appears due to a reorganization in the set of factors involved in the process.

This "accommodation" to the sensory factor is maintained as long as the inversion is sufficiently pronounced, but it can easily be destroyed as soon as the subject is asked to make, instead of a small head movement, a large movement that no longer provides any inversion or only produces a small tilt. In this situation, incongruence appears for a moment because the patient still continues with an abnormal reference system that is no longer necessary for congruency. The return to normality, i.e., the complete reestablishment of the motor reference, does not occur suddenly but through trial-and-error movements and confusions, as in the abnormal process of sensory adaptation. Thus, at first, he does not easily notice the incongruence that appears when the inversion is suppressed, due to the fact that the exclusion of the motor system has been very strong.

All these tests are performed with eyes closed, but even with eyes open the result does not change much since he does not pay attention to his surroundings nor would it benefit him in orientation, given his visual image orientation disorder and other disorders.

Although the patient gets used to the unexpected and paradoxical phenomena he experiences, all these tests on active movement make a strong impression on him. This is because the confusion in which he is immersed due to changes of reference, inconsistencies, etc., in an activity as immediate as voluntary actions, probably affects him more than many other disorders that manifest more indirectly.

Other types of sensory-motor incongruences in active movement can still be mentioned as a consequence of sensory deformation of the direction of very large movements, especially in the extremities. A curve-shaped deformation appears subjectively, similar to that studied in motion over the skin surface (Fig. 24.9). An example is the following test. The patient (M) is asked to raise an arm from the natural hanging position of rest, forward to the horizontal, without deviating from the lateral sagittal plane of the body, lifting the entire limb in a uniform and non-violent manner. The movement perceived by the patient, standing and with moderate muscular effort, is as follows. He first feels a short backward and outward movement, then a forward and upward curve, finally being out of the sagittal plane, outward. The amplitude of the perceived movement (about 30°) is much smaller than that of the real movement (90°).

In addition to the *change of shape* of the movement trajectory, its shortening and the change of speed (higher at the beginning) entail new disparity factors. The cause of the deformation of the active movement of the limb is the one exposed in the case of an object moving on the skin. The cause is the different degree of inversion according to the cumulative action of the stimulus on the nervous centers, etc., and we will not insist on this any further.

Finally, we must mention the disorder of orientation in mandibular movements, whether passive or active. Already in the normal subject it is very difficult to obtain a complete relaxation of the mandible that makes possible a proper examination of its passive movements. Therefore, we will refer mainly to active movements, which allows us to mention this case in this section. When the patient (M) opens his

mouth voluntarily, i.e., makes a depression movement of the mandible, he can feel a movement in the head, towards the neck, due to the proximal deviation that accompanies the phenomenon, and in a quite inverted direction (backwards and to his right). Under moderate facilitation by muscular effort, the movement is usually localized in the mandible, and when he opens his mouth he feels a very incomplete inversion (sensation of lateral movement of the mandible). For all this to occur, it is necessary to properly instruct the subject on the type and strength of the movement to be performed, and to urge him to pay attention to the sensations he perceives from his voluntary movements.

24.4. PERCEIVED ORIENTATION OF THE BODY SCHEMA

The alteration of deep (joint) sensitivity due to a defect in the direction of movement, among other disorders, affects the perception of the spatial orientation of the body schema (subjective position of the body in space). Moreover, as all sensory manifestations are closely linked to each other, this disturbance of the direction of movement is associated with a certain sensory level of the body schema, determined by body shrinkage, fragmentation, etc. (see Sec. 23.5.1). If this sensory level is very low, any sense of spatial orientation of the body is suppressed, mainly due to the loss of body coherence.

If subject M is sitting on a soft chair, he does not feel his body at all if he is immobile and inactive. Only at the first moment of sitting down he has a diffuse non-localizable sensation (spatial phase I). In that situation it is impossible for him to determine the spatial orientation of his body. It is necessary that the subject (seated and in a natural upright position) apply a certain degree of facilitation by means of a moderate and sustained muscular contraction, especially of the trunk, for some rudiment of body schema to emerge. When the body schema is initiated under such conditions, he feels it quite deviated from the vertical, tilted to the right about 60°. If the subject is standing, well upright, requiring of course some muscular action to maintain that attitude, the subjective deviation of the body schema can be quite smaller, around 40°. Therefore, such a position involves a greater facilitation than that required to diffusely bring out the schema when seated.

These results show how easily the *subjective tilt limit* is reached in the body schema. If in the sitting position the subject reduces the facilitation a little, all sensation of spatial orientation is lost because the body schema fades away. The maximum limit of subjective tilt could reach about 70°. These facts correspond to the general behavior in the perception of figures of all types (including the body figure), which require a fairly developed sensory level in order to be perceived, and at that level the spatial orientation function is only moderately affected. We have already seen this during sensory phase IV in the case of figures drawn on the skin (see Sec. 23.3), and also in the case of visual image inversion (Sec. 13, Table 13.1 in Vol. 1). In the latter case we saw that figures (e.g., optotypes used in visual acuity tests) already become blurred when the perceived image is tilted about 40°, all details disappearing before reaching 90°. If the degree of inversion

is higher, only a diffuse shape is perceived (see case of the test arrow in Sec. 13 in Vol. 1).

From all that has been said, it can be inferred that for the subject to perceive a certain orientation of the body schema, he must reach a sensory development of at least spatial phase IV, to which corresponds a tilt of less than 90°. The rudiments of the body schema in more elementary phases are not enough to arouse a coherent sensation of the schema, which besides being very small and diffuse, tends to be fragmented (independence of its parts).

Examining the deviation of the body schema more precisely, it results that in addition to the rightward tilt, there is a certain backward rotation, and at the same time it seems that the whole perceptible body is somewhat tilted forward. It is therefore a complex deviation in several spatial directions that should be interpreted as a residue of the inversion of the whole body according to a *spiral turn*. The patient senses that the tilt is for the body as a whole, i.e., the body does not bend at the waist forming an angle, but rotates through the center of the trunk changing the whole body's orientation in space. The spiral deviation (a backward turn on himself) is better perceived when the tilt is very pronounced, about 60°-70°, but it is less evident at tilts of about 40°. As for the correction by means of facilitation, it should be noted that when standing and with maximum facilitation, the body schema is much more defined, but nevertheless there remains a small residual deviation to the right that may be about 10° or a little more. The different deviations according to the subject's states of activity are described by the subject, either verbally in an approximate way, or by indicating the position of an arrow rotating on a graduated circle.

As said, together with the alteration of the felt orientation of the body, the body schema evolves in shape and size. In a seated position and with weak facilitation by muscular effort but sufficient to awaken the body schema, the deviation felt is 60°-70° with respect to the vertical. In this case the schema is at the beginning of spatial phase IV (Fig. 21.14); the trunk is what takes on most relevance, the head and extremities probably less, the latter being only diffusely perceptible up to their middle, as shown in Fig. 24.14 (a). The *size of the body* is felt to be greatly reduced, both by the exclusion of distal parts and by the general spatial shrinkage (recall the reduction of the spatial threshold). As for these data, it should be noted that the perception of the body in a normal immobile subject is already somewhat diffuse, and this feature is even more pronounced in pathological cases, being difficult a precise description. The data must be appropriately "extracted" from the patient, since, as will be seen when studying postures, the defect in the perception of the body and postural alterations in the body schema tend to be ignored by a mechanism of pseudoagnosia that is very frequent in all types of pathological manifestations.

For a deviation of about 40°, the size of the body schema increases a little more, the head and limbs are felt more clearly although the hands and feet are probably still missing [Fig. 24.14 (b)]. Finally, with maximum facilitation by muscular effort, there is only a subjective tilt of the body of about 10°, the size increases and tends to normal; although if the subject does not move, the most distal part of his fingers may be excluded [Fig. 21.14 (c)]. When standing, the difference in subjective height between the state of moderate facilitation and that of maximum facilitation can be

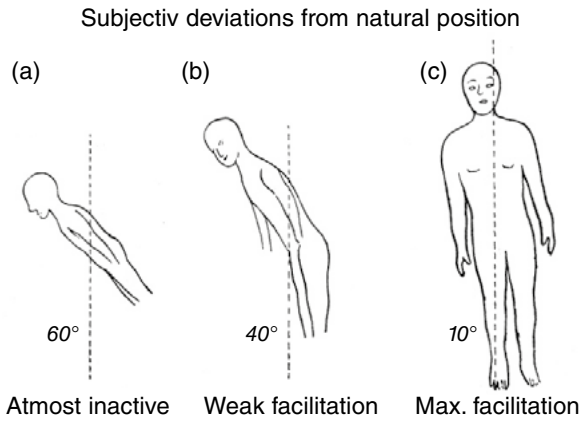

Figure 24.14. Subjective orientations of the body schema in patient M. (a) M seated and almost inactive: body tilted to the right 60°-70° and notable reduction in size corresponding to the onset of phase IV. It is the maximum deviation that can be felt at the beginning of the sensation of the body as a whole. (b) M standing and with more facilitation through muscular effort: 40° of deviation, phase IV more developed. (c) M standing and with maximum facilitation by muscular effort: residual deviation of 10°-15°.
Note in the figures a tendency to spiral rotation (head turned).

about 20-25 cm. Sometimes, however, there may be a greater variation, since if he is standing and as inactive as this attitude allows, it may seem to him that he is as reduced in height as if he were kneeling. This reduction probably corresponds more to the sitting position, which is even more inactive.

We can get a more precise idea of the nature of the deviation of the body schema by resorting to more objective methods. One of them consists of the subject under maximum facilitation imitating the deviation he feels in an almost inactive state. Another method is that of compensation of the subjective deviation by a real tilt in the opposite sense when the subject is almost inactive. Although the maximum facilitation by muscular contraction does not completely suppress the deviation, it is so small that it can be disregarded in these objective methods. The patient is then able to imitate the posture he feels when he is inactive, as shown in Fig. 24.15 (a): he turns his head to his right and a little backward, the right ear is farther back and lower than the left, the trunk and upper extremities adopt a similar position, and the whole body leans forward. If the compensation method is used, the standing patient, as inactive as possible, should be positioned tilted about 40° or more to his left and backward in order to give him the sensation that his body is upright and in a normal frontal position, as shown in Fig. 24.15 (b).

As already stated, the origin of the tilts and turns felt by the patient is the disorder of inverted tactile orientation. The perceived degree of inversion is related to the perception of the body, and below a certain limit, this perception fades easily and prevents to feel a large tilt. Thus, a moderate tilt and rotation correspond to an inversion following a spiral that is partially developed here. It can be said that the

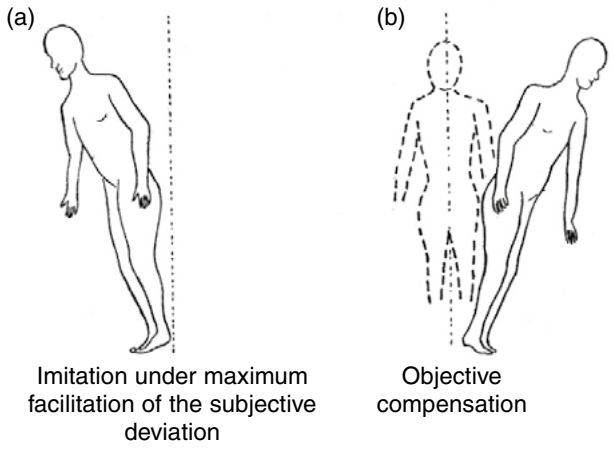

Figure 24.15. Tests for body orientation disorder in subject M. (a) Patient M under maximum facilitation imitates the posture felt in the inactive standing state. He tilts objectively 40° to the right, with some backward rotation. (b) Subject M, in an almost inactive state, is objectively tilted 40° to his left to obtain the sensation of verticality of the body, thus compensating the subjective deviation to his right.

sensation of deviation of the position of the body reflects the state of deep sensitivity and of other functions involved in the formation of the body schema. Spatial values are modified by shrinkage and inversion, due to asynchrony. Thus, proprioceptive excitation (via joints, muscular tension, etc.) corresponding to the static attitude of the body results in incomplete spatial recruitment, leading to an inversion process of the body and a reduction of its size. The less the facilitation acts, the more pronounced these effects are.

As for the *direction of rotation* in the inversion, it is always to his right because the rotation concerns the body as a whole. It is like in the inversion tests of rectilinear stimuli on the skin in the midline of the body.

The issue of body posture deviation does not end here and we shall return to it further on under other also novel aspects.

25. Complex processes of orientation in the sense of touch

25.1. PERCEIVED INVERSION AND DEVIATION DURING WALKING

Having finished describing the deviation of the orientation of the body schema in a static situation, we must now consider the disorder kinetically. It can be studied in a very appropriate and complete way when the subject is walking. Very remarkable phenomena of great importance appear which clearly illustrate the process of the so complex tactile inversion. The main interest lies in the phenomena that occur when the patient starts walking, i.e., during the first ten steps at the most, under certain conditions. These phenomena are very numerous due to the multiple factors involved in the process. Not all the steps actively taken by the subject are equally perceived and valued, although objectively they are the same. The first steps are perceived as if they were much shorter, much faster, and with the direction completely inverted with respect to the objective direction of the walk. Nothing remains fixed in the course of walking: the step size tends to increase, the speed to decrease and the reversed direction tends to straighten out by a process of latent addition in the nervous centers as the subject continues to walk. In parallel, the body schema varies greatly. The movement of the lower extremities is localized in different ways, the perceived body size gradually increases and the body schema tends to emerge, evolving also the orientation of the body in space. At first, the phenomena are very abnormal and diffuse, but gradually a more complex and more normal organization emerges. Thus, there is a very extensive development of tactile orientation. As intricate as this set of phenomena may seem, examination of the process reveals a fairly simple mechanism that finds a logical explanation in matters already studied for the most part in previous pages.

For a thorough analysis of the process we shall study in the following subsections: iterative excitation by successive steps, subjective space and time in walking, subjective trajectory, subjective localization of the movement in the body, size of the body schema, etc., and finally the felt orientation of the body during walking.

25.1.1. Iterative excitation by successive steps

The process of walking is originated by the proprioceptive stimulus of the steps, so we must first analyze their characteristics and effects. Subject M, making no more effort than the usual effort to stand and walk (semi-inactive state for our purpose) takes a step or a step and a half, i.e., advances one foot normally and then moves the other to meet the first, the length of the step being the usual one. Up to this point, the tactile sensation usually barely exceeds phase I, i.e., a diffuse pressure sensation without localization, or at most localized medially (phase II), but without arousing the sensation of movement. It is only after he has taken two and a half or three steps that he perceives signs of movement. Of the several steps taken, he only perceives the last one, and he senses it in the totally opposite direction, that is, backward, without being able to indicate whether there is some deviation to any side. This subjectively inverted step is felt as extremely fast and short (phase III), and the corresponding movement is localized only in the hips. It is worthwhile to compare the occurrence of the different phases here with the data in Table 23.5. The body schema presents certain deviations and reduction in size, which will be study further on.

Continuing now with the process of walking, the patient is able to perceive two or three steps after four or five steps. After six or seven steps, he feels four steps as follows: the first one very short and totally backward, the next two also backward but somewhat tilted to the right and slower and longer than the first, and finally, the fourth step is felt as transversal, or something similar more or less towards the right. This happens successively; thus, as the number of steps during uninterrupted walking increases, the subjective direction tends to straighten out and resemble the real gait (phase IV). At the same time, the steps are felt to be longer and slower than at the beginning. Since the initial steps are not perceived at all and the following steps are felt to be very short, the subjective path traveled must necessarily be much shorter than the real one (see below). If, in addition, the subject walks with his eyes closed, he will have no other reference for the path traveled than his steps reduced in length.

Performing the same test in a state of maximum facilitation, i.e., walking with the body under strong generalized muscular tension, results in fewer ignored steps, less inversion, and greater subjective distance traveled. Walking at the same speed as in the previous case, it may sometimes happen that he ignores the first step taken. This occurs when the step is short or weak, or when the facilitation by muscular effort is moderate. But in general, the first step is already perceived and is felt less inverted than in the previous inactive state. It is perceived as going backwards and to the right, i.e., rotated about 110° approximately. The second step may already be at 90°, and the successive steps quickly tend to be felt more towards the front. After seven or eight steps, the step is perceived in a direction very close to the one really executed. Therefore, the evolution of the subjective walking is quite different from that in the quasi-inactive state.

These two tests of walking, in the inactive state and under facilitation, correspond to a real rate of one step per second or slightly less. For example, the subject takes six steps in just over 4 seconds, or five, being steps of normal length, of about 40-50 cm (the subject is a tall individual), resulting in a rather slow walking pace. In

such conditions we have seen that the sensation that the subject has tends to change continuously during the course of the gait, intervening, as already said, an action of accumulation (latent addition) in the nervous centers, due to the excitations involved in the successive steps. Thus, it results in a *recruitment of the direction of walking as a function of the number of steps*. This iterative character is demonstrated by studying the two main factors involved in this type of stimulation: the energy of each step, and the iterative aspect related to the number and frequency of steps.

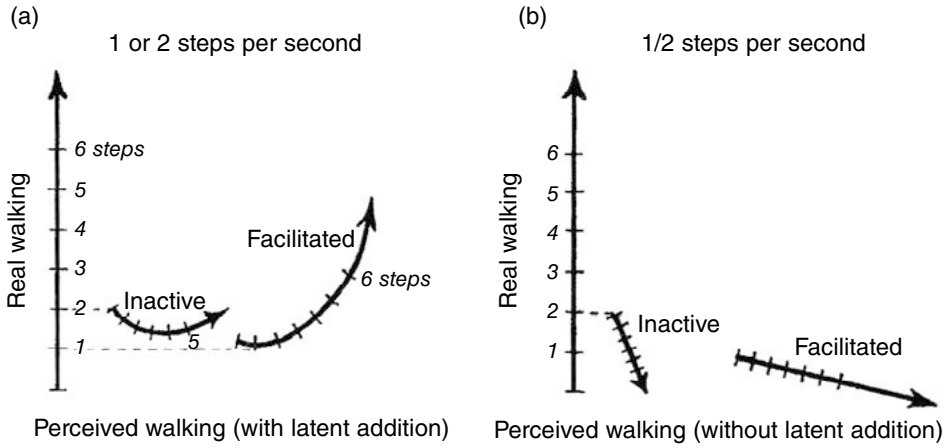

Figure 25.1. Diagram on the subjective perception of walking according to step frequency in patient M. (a) With an average of 1 to 2 steps per second, a latent addition effect and thus sensory recruitment is obtained. The steps vary subjectively in a continuous manner in direction and length. In the inactive state, recruitment is slower than under facilitation. In the former, only the second step is perceived, the direction felt is more inverted for the whole path, the length of which is felt to be shorter as well as the time spent. The subjective spatiotemporal shortening in the inactive state is one-third of the real value, and almost half of that perceived under facilitation. The tick marks for the steps indicate the spatial shortening. (b) At a frequency of half a step per second (one step every two seconds) there is no addition effect, and since there is no sensory recruitment, the subjective perception of walking remains unchanged as regards direction and spatiotemporal value, which are much more altered than in the previous case. There may be some trace of latent addition, especially in the inactive state, as the perception of movement occurs in the second step.

Note in the different cases the different subjective shortening of the step in relation to the degree of inversion, the difference between inactive state and under facilitation, and the different subjective trajectory with or without latent addition.

The action of the number of steps has already been seen. As for the frequency, if the subject walks very slowly, and there is a long time interval from one step to the next, it is not possible to achieve accumulation and the same result is obtained for both the first and the last step. Thus, the subjective direction of walking is approximately equally abnormal throughout the gait. Two different types of subjective gait can thus be obtained, of uniform direction if latent addition is not involved, and of variable direction if it is involved, as shown in Fig. 25.1. The latent addition tests

described above correspond to a small frequency of about *one step per second*, since this was convenient for the initial analysis of the phenomena in subject M. But this frequency is too low to obtain full recruitment of the direction of walking. Much better recruitment is achieved with a double frequency of *two steps per second*, as will be seen later. Instead, to eliminate the central effect of accumulation (latent addition) of the steps, the subject, in an almost inactive state, should walk rather slowly. Tests performed on patient M show that a frequency of one step in one and a half seconds results in a perception of continuously inverted walking direction, with the steps being felt as very short and fast. In the state under facilitation by muscular effort, the nervous reaction being more rapid and therefore the residue shorter, the frequency can be somewhat greater without giving rise to latent addition. Excluded the central action of latent addition, the recruitment obviously disappears and the subjective perception does not change no matter how long the walking lasts, or the change is so slow as to be negligible.

These step frequency values for the latent addition limit vary greatly in relation to the other fundamental factor involved in the process, the *step energy*, which depends on the length of the step and its strength. Thus, in the test under facilitation, no latent addition is obtained when the subject, at a rate of one step per second, walks with a very small step length (almost half of normal) and with very little energy (slow and not vigorous). In this case, the subject does not perceive a straightening of the gait and has to be led by the hand because he does not know how to walk so slowly on his own in this situation. On the contrary, if the subject under facilitation takes a single high-energy step, with great strength and force of the lower extremity, this single step reaches a considerable functional level, and the subjective direction deviates only about 60° from the real direction. Even less subjective deviation is obtained if the subject kicks hard in the air with as much force as possible. In this case he can achieve a practically normal direction. However, in the inactive or almost inactive state, no matter how strong the kick is, he only achieves 90° of subjective deviation, and there is probably a brief inverted path at the beginning of feeling movement. Figure 25.2 shows these results.

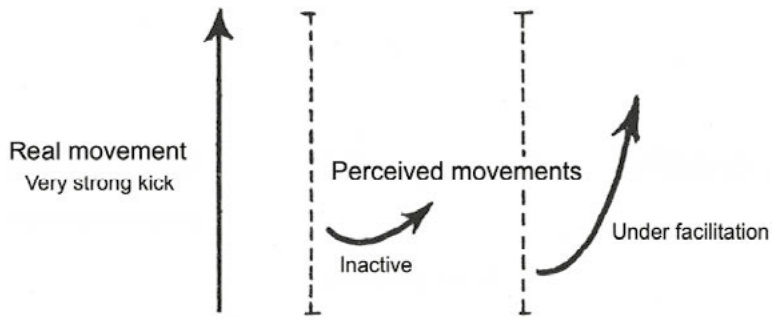

Figure 25.2. Subjective direction of a very energetic single step (with kick in the air) of subject M. Note that in the inactive state, the subject cannot straighten the subjective direction beyond 90°, whereas under facilitation, the subjective deviation is very small. Note also the different subjective path in the inactive state and in the state under facilitation.

In the case of the strong kick, it turns out that with a single step the same effect is obtained as with many. It is clear that in the kick, the joint movement comes into play in all its range, and also abruptly, which means a great excitation which results in a high sensory level, within the current asynchrony. In contrast, during ordinary walking, joint movement is quite moderate in range and velocity, so recruitment has to be developed slowly and successively through iteration.

In short, the disorder of direction during walking is consistent with what has been stated above (Sec. 24.3) about perception and direction in deep (joint) movement, whether passive or active, and for both repeated stimulation and single strong excitation. A single step must be very long and vigorous to awaken the sensation of movement and, above all, to overcome the inversion of direction. This is partially achieved in the inactive state, and much better under facilitation. However, under ordinary walking conditions, the joint excitations are too weak, and only the addition of steps produces a re-inversion within certain limits of step frequency. Thus, very slow walking (one step every one and a half or two seconds) always provides the same perceived direction, since the residue of each step is dissipated before the next one arrives. Instead, fast walking (two steps per second) allows the residue of the steps in the nervous centers to add up successively, and thus the perceived direction (sensory level) varies with each step. The interval between steps to produce summation is related to the level of excitation, and therefore should be somewhat shorter under facilitation than in the inactive state. All these circumstances on the subjective perception of walking are illustrated in Figs. 25.1 and 25.2.

25.1.2. Perceived space and time during walking

The space and time of the perceived steps is changing during walking (due to latent addition) in parallel to the change occurring in the subjective direction. That is to say, the first perceived steps are very short and fast compared to their real value; then, as the direction of the gait is being straightened and re-inverted, the steps gradually resemble their real value, although without reaching it as long as the felt direction is not completely normal. These manifestations of space and time in the steps correspond to the intrinsic alteration of the perception of movement (path and speed) already studied in several chapters for both touch and vision. This alteration depends directly on the elementary disorder of nervous excitability in the nervous centers. Such manifestations are the result of a functional dynamic reduction, reflected here in a *spatiotemporal reduction*, i.e., a contraction of perceived space and time, which results in a reduction of the path and an overestimation of speed, giving rise to a sensation of walking with small and fast steps.

Due to this intrinsic modification of the perceived movement, it turns out that the path perceived by the subject after six steps is greatly reduced in relation to the real path. However, this remarkable difference between what is felt and what is executed tends to be initially disregarded by the subject, and he only recognizes it by means of appropriate tests.

Of course, the subjective reduction of the path is more clearly noticeable in the inactive state, but the subject first believes that the path has been reduced by only

two-thirds of the real path. However, it is understandable that due to the steps lost initially (if he walks at one step per second he ignores the first two steps and if he walks at two steps per second he ignores the initial one) and the enormous contraction that most of the steps undergo due to the strongly inverted direction (Fig. 25.1), the perceived decrease in the path should be much greater, as is indeed the case. What happens is that very abnormal sensations, being more diffuse, brief and of less frequent occurrence for the subject (M), tend to be excluded from perception and easily not taken into consideration. This happens both for strong inversion and for the shortening phenomena we are now dealing with.

If the patient in an almost inactive state walks through a room six meters long and crosses it by taking ten steps, he later believes that the perceived shortening of the path is not much, especially if he has kept his eyes open. But if he walks this path with his eyes closed, alone or led by the hand, and is asked to pay attention to his sensation of the length of the path, he will easily realize how short the felt path is. Since with closed eyes he has no information other than his altered touch, he has to stick only to that information. It is difficult to determine with certain accuracy the reduction of the path, and it can only be known in an approximate and indirect way: the subject, after having walked the six meters in ten steps with his eyes closed, must indicate on the ground, opening his eyes, what distance he thinks he has covered. It happens then that the six meters can be reduced to 1.80 meters, that is, less than one third. This value is by no means small and may even be too large if the following is taken into account: at the end of the run the perceived direction is about 80°, therefore the steps must have greatly reduced their perceived size; moreover, as two or three steps are felt strongly inverted, the perceived shortening must be even greater, apart from one or two first steps that are lost. It should be noted that the total or almost inverted movement corresponds to a perception of movement only as slight signs, and in this case a normal step of 40 cm is reduced to about 4 cm, i.e., one tenth. The same test under facilitation results in a much smaller subjective reduction because the steps are not lost, and they are perceived to be longer as there is less inversion. In this case, the subject indicates on the ground a distance of about 4 or 5 meters, but it can probably also be excessive, although at the end of the path he feels a deviation of only about 20°.

It is understandable, after all that has been said above about iteration, that the step frequency has a significant influence on the spatial reduction. If the subject walks very slowly, there is no addition and the reduction is maximum; thus, in the inactive state, six meters should feel like half a meter. Figure 25.1 attempts to illustrate the spatial reduction roughly, as well as the close relationship between the degree of inversion and the spatial reduction of the steps. (These steps in the non-addition case are perhaps represented too large.)

As for the temporal contraction (increase of the perceived speed in the sensory process), it is related to the spatial reduction, and is manifested in a significant increase in the perceived frequency of the steps. As the perceived gait changes continuously when there is a latent addition effect, the subjective rate cannot be uniform but progressively decreasing, without reaching the objective step frequency which is always lower than the subjective one. This is the same as in other rhythmic stimu-

lation tests, for example in vibratory stimulation with tuning forks. For both vibration and footsteps, at the beginning of the perception, the subjective frequency is so high that what is perceived is a fusion (phases I and II). Subsequently, the perception becomes intermittent with a very short time interval that tends to enlarge progressively throughout the process. In the case of walking, if the first step is lost, it is due to the fusion effect that prevents the appreciation of the time interval, indefinitely reduced when the development of the sensory process of walking begins.

The sensation of time contraction would mean a shorter time felt by the subject in walking the aforementioned path. The real path is six meters, traveled in ten steps, which at two steps per second results in about five meters in five seconds, i.e., a rate of one meter per second. Assuming that the contraction of time is of the same order as that of space, it follows that if 5 meters of real path are subjectively reduced to 1.80 meters (approximately one third), the perceived time will offer a similar reduction, i.e., five real seconds would be reduced to 1.80 subjective seconds. It could then be said that the speed has not changed, since space and time are altered equally, but it is clear that the subjective frequency or rhythm of the steps will have tripled, since the same number of steps will be felt in one third of the real time. Similarly, the corresponding reduction under facilitation would be obtained. Thus, the spatial contraction of the steps indicated in the different figures also represents a temporal contraction.

25.1.3. Perceived trajectory

We know that the *subjective trajectory* of walking depends on the conditions for latent addition of steps and on the spatiotemporal dimension, which determine the corresponding development of the process. In the following, we shall take a closer look at this development by paying attention to both the immediate manifestations of the subjective trajectory and the functional quantitative relations. It is also important to demonstrate the existence of phenomena of this type in other sensory systems, for example in the visual system, which means that they have a general character.

When patient M is walking and there is progressive recruitment according to the number of steps, the deviation felt by the subject is always to his right (Figs. 25.1 and 25.2), as is the rule in this individual for stimuli affecting the whole body schema. Regarding the course of the trajectory, as the sensation of step direction changes from an almost complete inversion to a very pronounced straightening while also changing the sensation of size, the perceived trajectory follows a more or less open spiral depending on the speed of recruitment, as we have already seen in other types of tests.

By repeating the tests and studying the meaning of the different factors, keeping the subject attentive and properly informed about the nature of the tests, it is possible to obtain valuable data despite the drawbacks and inaccuracies of the method. In the experiment, subject M walks in a straight line forward with a frequency of about two steps per second, a frequency almost double that in previous tests, thus resulting in an efficient latent addition, well suited for our current objective. The step length is 40-50 cm, i.e., a common length. Naturally, it is necessary to maintain max-

imum regularity in all these factors during the test. The results are shown in Tables 25.1 and 25.2.

Table 25.1. Walking perceived by subject M in an inactive state, according to number of steps. Frequency of 2 steps per second. Step length 40-50 cm. (See Figs. 25.3 and 25.4.)

No. of steps	Perceived direction, angle rotated	Movement localization
1		Not perceived
2	Backward, 160°?	Signs in contralateral hip
3	Idem	Idem
4	Backward and to the right, 110°?	Idem homolateral
6	To the right, 90°	Idem
8	Weakly forward, 80°	Idem
12	Almost oblique, 70°?	Idem and signs in the knees
15	Oblique, 45°	Idem
20	35°?	Well in the knees
25	Almost forward, 20°?	Idem and signs in the feet

Table 25.2. Walking perceived by subject M under facilitation by strong muscular effort, according to number of steps. Frequency of 2 steps per second. Step length 40-50 cm. (See Figs. 25.3 and 25.4.)

No. of steps	Perceived direction, angle rotated	Movement localization
1	Somewhat backward, 110°	Homolateral hip
2	Almost transverse, 80°	Idem and signs in the knees
3	Almost oblique, 50°	Idem
4	35°?	Well in the knees
5	30°?	Idem
6	Almost straightened, 20°?	Strong in the knees, signs in the feet
10	Very little to straighten out, 10°?	Hips, knees and well in the feet

Tables 25.1 and 25.2 show, for each number of steps, the corresponding sensory level expressed by the perceived direction of the steps and also by the localization of the movement. This localization initially undergoes strong proximal deviation, and after more steps it is more distal (knees and feet). In this way, the growth in the size of the body schema is also evidenced. Once the subjective gait deviation is known through global determinations, partial tests are performed to find out which subjective deviation corresponds to each step, in order to know more precisely the type of trajectory. After the subject has taken one or more steps, he must indicate the degree of deviation he feels at the end of this partial walk. Due to the iteration effect, it is not possible to resort to the compensation method because it would be too cumbersome to divert the real gait in various ways. Therefore, there is no choice but to stick to the verbal information of the subject. The disorder in step direction causes steps

taken forward to be felt as deviating in various ways, as if the step were taken with a certain lateral, transverse, backward deviation, etc., and the patient should indicate this type of deviation. Some directions can be indicated with some precision, for example in the case of a 90° deviation in which case the subject feels as if he has taken a step in a completely transverse direction. For other cases, the patient must be guided by the degree of backward or forward obliquity, and only approximate determinations can be obtained (indicated in the tables with a question mark) but very useful and sufficient for our purpose in the absence of a better method.

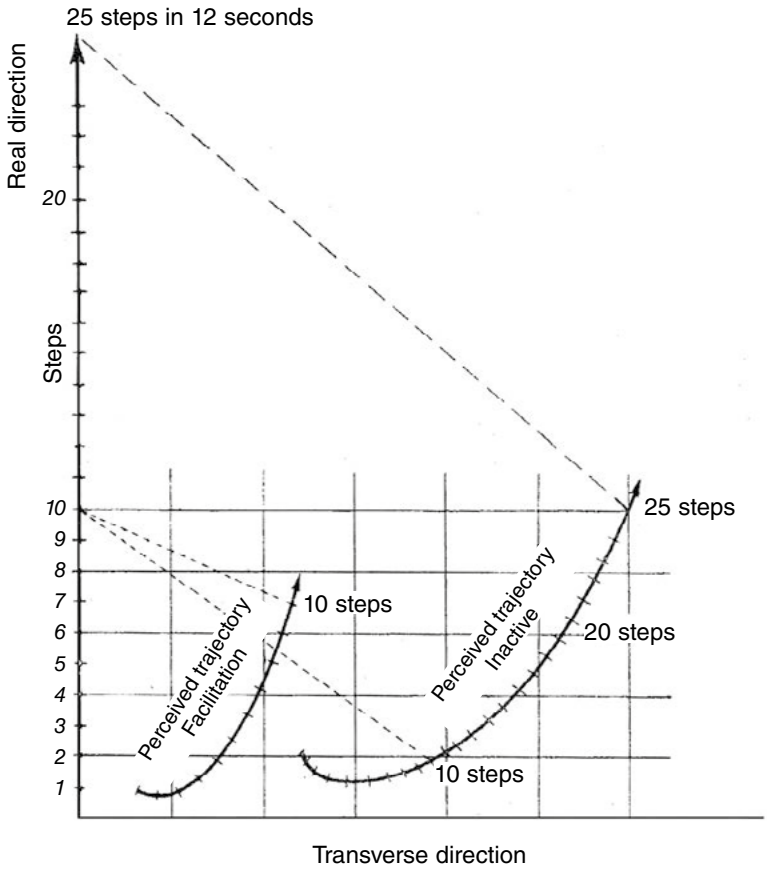

Figure 25.3. Diagram of the trajectory perceived by subject M as a function of the number of steps. Frequency of two steps per second, step length 40 to 50 cm. Note the spiral development (very open spiral branch), the perceived shortening of the trajectory, the suppression of the initial steps, etc.; for the inactive state and for the state under facilitation. Compare with the curves in Fig. 25.4.

From the data in the two preceding tables and taking into account the corresponding spatial contraction (more pronounced at high degrees of inversion), the trajectories shown in Fig. 25.3 are determined in an approximate way. Direction,

space and time of the steps vary continuously, and this is perceived by the subject directly. In Fig. 25.3, the perceived direction is indicated by the tilt of the line corresponding to each step, and the perceived space and time is indicated by the length of that line, very short at the beginning and longer at the end, tending to resemble the real size.

The trajectory tends to take the form of a spiral, more pronounced in the inactive state, to which corresponds a slower variation throughout the process. Comparing the results of the inactive state with that of facilitation, it is observed that in the latter state, the perceived walking after ten steps is almost normal (see Table 25.2 and Fig. 25.3). In contrast, in the inactive state and with the same number of steps, the patient feels that he has traveled half the path as with facilitation, and about one third of the real path. In the inactive state, the tenth step taken is felt with a deviation around 90°. The disorder is more evident in the inactive state, and the subject needs to take 20-25 steps to achieve an effect similar to that obtained with ten steps under facilitation. Therefore, the difference is two times greater or more.

These important defects have been extracted from the subject by means of much patience. The subject never noticed them spontaneously since in ordinary life they are easily ignored, either by the corrective effect of facilitation by more or less unconscious muscular effort, or because they are only very noticeable disorders at the beginning of walking and of short duration. Furthermore, there is a natural tendency to exclude abnormal phenomena, and even more so if they are incongruent with voluntary activity, as in the case of walking.

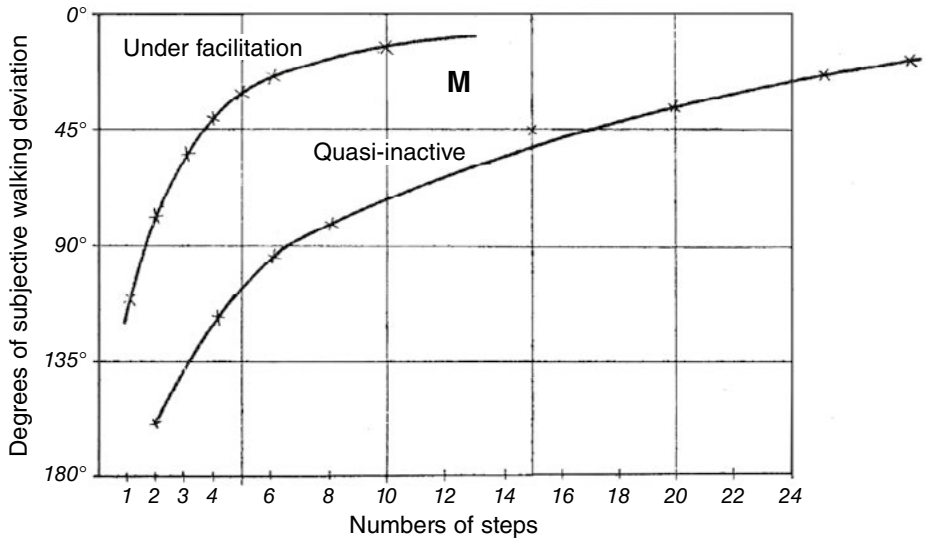

Figure 25.4. Recruitment curves of the sensed direction of walking as a function of the number of steps, according to the data in Tables 25.1 and 25.2. Note the different slope and position of the curves for the quasi-inactive state and the state under facilitation. The subject walks objectively in a straight line, with equal steps of ordinary length (40-50 cm) and a frequency of two steps per second.

In the trajectories studied, both the variation of the direction and the variation of the length of the steps are involved, depending on the number of steps. From the data in Tables 25.1 and 25.2 we can obtain functional relationships between the direction of the steps and their number, which are the iterative recruitment curves shown in Fig. 25.4. This type of recruitment has already been analyzed in different experiments on touch, as shown in Fig. 22.4 (localization recruitment as a function of the number of stimuli at a given location on the skin). All curves of sensory level recruitment are of the same type, regardless of the type of excitation, whether it is a single increasing stimulus (Fig. 22.3), a constant stimulus iteration or an increasing facilitation by increasing muscular effort. They correspond more or less to logarithmic curves of the type of Fechner's law of sensory variation. These curves in Fig. 25.4 can be compared with the data from the test on the perceived orientation of a rectilinear stimulus on the back of the hand as a function of pressure (Fig. 24.4). In the curves of Fig 25.4, the iterative stimulation conditions are the same for the quasi-inactive state and for the state under facilitation. As asynchrony is lower under facilitation, recruitment in that state is much higher, as can be seen from the position and slope of the curves.

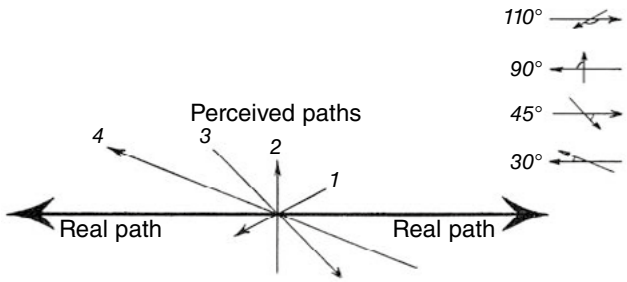

Figure 25.5. Visually perceived directions by subject M of the oscillatory motion of a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome. Note the increase of the perceived path when the perceived direction is straightened. To simplify the representation, this diagram corresponds to the state under facilitation according to Table 25.4. For the inactive state, a similar diagram can also be obtained but with other luminous stimulation values. See text for experimental conditions.

This iterative recruitment of spatial orientation in the sense of touch is not an isolated event in the set of central syndrome disorders. We also find it in vision, as it is to be expected, since touch and vision are equal systems in structure and excitability defect. To observe the phenomenon in vision, we place a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome that oscillates at a constant number of beats per minute. There is a beat for which the subject reaches the phase in which he begins to perceive motion. From that beat on, each consecutive beat corresponds to a new perceived direction of motion, the direction evolving in the same way as that of the steps when walking. That is to say, not only the perceived direction changes but also the amplitude of the oscillation and the speed, apart from a chromatic change in this case due to chromatopsia of the white

color of the small disc. The experimental technique is analogous to that of walking, although with some particularities that make the visual test more difficult. At the beginning of the perception process, the perceived motion is strongly inverted, i.e., almost horizontal but in the opposite sense to the real motion, and it can be indicated by the patient since the real oscillation is not very fast. Subsequently, the motion tends to be perceived as vertical (rotated 90°), to become again perceived as horizontal in the almost correct sense as the recruitment increases, as shown in Fig. 25.5.

The experiment is conveniently fragmented into several partial trials in order for the subject to indicate as accurately as possible, after a given beat, the perceived direction. The experimental conditions are: metronome at 25 cm distance, white disc of 1 cm in diameter at the end of the pendulum illuminated with medium natural light, displacement of the small disc about 10-12 cm between each beat, rate of 1 beat per second, and vision only with the right eye of patient M. The mean values of several tests are shown in Tables 25.3 and 25.4.

Table 25.3. Visually perceived direction by subject M, in the inactive state, of the motion of a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome, depending on the number of beats elapsed. Experimental conditions in preceding text.

No. of beats	Perceived direction, color. (Sensory level)
1	Dim motionless luminosity, achromatic sensation
2	Very short inverted motion, dark green
3	Idem but more oblique
4	Idem and still more oblique 120°?
5	Close to vertical 90°? pale green
6	80°? scarce white
8	60°?
12	Rather horizontal, not inverted, more white than green
14	Idem but more horizontal

Table 25.4. Same as in Table 25.3 but subject M under maximum facilitation by means of maximum muscular effort.

No. of beats	Perceived direction, color. (Sensory level)
1	Inverted oblique motion 100-120°? pale green with hints of white
2	Close to vertical 80-90°
3	Close to oblique 45°?
4	Approaching horizontal but not inverted
6	Idem but even more horizontal
7, 8	Idem, completely white

As for the color change indicated in Tables 25.3 and 25.4, dependent on the degree of inversion and contraction of the path perceived by subject M (especially

in the inactive state), it has already been studied in the part concerning visual functions (Sec. 11.2 in Vol. 1). As for the simultaneous gradual variation of the different factors, it may be useful to compare the data in Tables 25.3 and 25.4 with those in Table 13.1 in Vol. 1 on visually perceived orientation of a vertical white arrow pointing upward. Much more relevant now is to compare Tables 25.1 and 25.2 on the perceived direction of walking depending on the number of steps, with Tables 25.3 and 25.4 on the visually perceived direction of motion depending on the number of metronome beats.

We then find that such iterative recruitment of the direction of motion is possible in both vision and touch, which allows us to realize that this process has a very general significance. Furthermore, it is shown that, by properly adjusting the experimental conditions in vision and touch, the development of sensory recruitment is, in quantitative terms, completely identical in both sensory systems. This is expressed by means of the curves in Fig. 25.6, where it can be seen that the tactile and visual curves are very similar. The visual curves were made several months after the tactile curves in 1946, since having first made the recruitment curve for step direction, it was thought much later that a similar effect could be determined in vision. In both the metronome beats method and the step method certain imperfections are unavoidable, and these methods cannot compete in accuracy with the compensation

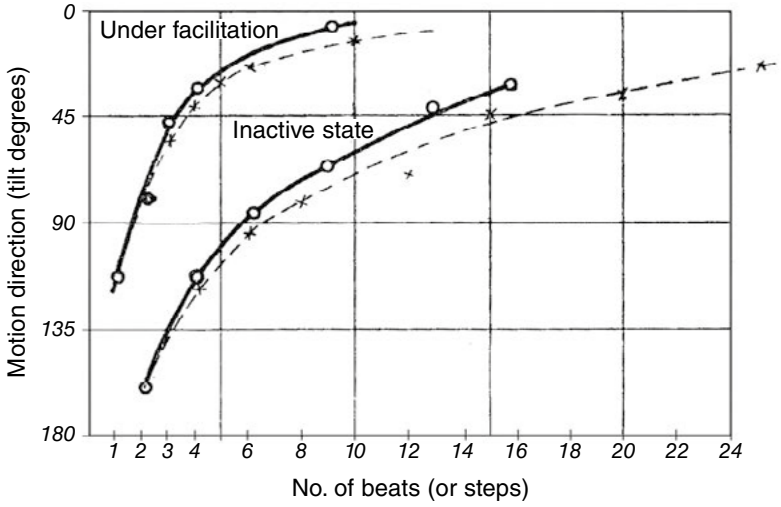

Figure 25.6. Iterative recruitment of the direction of motion perceived by subject M in the inactive state and in the state of maximum facilitation. Solid curves: visually perceived direction of motion of a white disc of 1 cm in diameter at the end of the pendulum of a mechanical metronome, as a function of the number of beats (see Tables 25.3 and 25.4). Metronome at 25 cm distance, white disc illuminated with medium natural light, displacement of the small disc about 10-12 cm between each beat, rate of 1 beat per second, and vision only with subject M's right eye. Dashed curves: perceived direction of walking as a function of the number of steps (Fig. 25.5); see data and experimental conditions in Tables 25.1 and 25.2. Note the high similarity between visual and tactile curves.

method used in the curves on the image orientation of a vertical arrow. However, we have already seen in other tactile tests how it is possible to achieve valuable results despite the shortcomings of the methods.

25.1.4. Localization of movement in the body, size of body schema, displacement on the ground

During the initiation of walking, the localization of the movement of the lower extremities varies significantly due to the proximal deviation of stimulus localization, resulting in a change in the size of the body schema. The first perceived steps only elicit a sensation of movement around the most proximal part of the limbs, i.e., the hips. Thus, we can only refer to perceived steps in a broad sense. At the same time, the perception of the body is diffuse, rather soft and poorly delimited, difficult to describe accurately by the subject. After several steps, the subject tends to feel the movement in the knees and much later in the feet, the previous localizations being preserved.

Tables 24.2 and 25.1 provide details on this *evolution of localization* in tests of walking at a rate of two steps per second. In the inactive state, after two steps the subject feels movement in the hips, after twelve steps the sensation of movement in the knees is initiated, and after 24 steps, signs of movement in the feet are felt. Instead, under maximum facilitation, with a single step the movement is already felt in the hips, with two steps, it is felt a little in the knees, and after six or seven it is felt in the feet. When the localization of the movement begins to be felt in the feet, it is felt only as small indications, as a movement of much smaller magnitude than the actual movement of the foot, and deviated by about 20° (see Tables 25.1 and 25.2). With more steps, the movement felt in the feet becomes more intense up to a certain limit.

The decrease in the proximal deviation effect as the localization of the movement becomes more and more distal is equivalent to an *increase in the size of the body schema*, and the body is felt to grow in the course of walking. The shortening of the body schema with the distal exclusion of the lower parts of the limbs does not necessarily mean that the subject feels supported on the leg stumps. Here the support is indifferent, it is simply that the body does not feel complete. The consequences of this incomplete and diffuse sensation are addressed in the following experience.

A complementary test, very significant for the new phenomena it reveals, consists of the patient making the movement of taking steps but without moving from the site. At a rate of two "steps" per second, the subject, in a quasi-inactive state and with eyes closed, takes 12 seconds (25-27 "steps") to realize that he is not moving from the site. Under facilitation by means of strong muscular effort, the same result is achieved with 10 "steps" in 4.5 seconds. This result is achieved when in both cases the localization of the movement is quite evident in the feet, and the body schema is well developed. At that point, the deviation of the perceived direction of the step is about 20° or less. During the previous steps, the subject is convinced that he is walking displacing himself over the ground, and not that he is in the same place (as is actually the case). This result shows how much the perception of walking is destroyed.

It is likely that for the subject to distinguish the type of walking (with or without translation movement) it is not so important the localization of the movement in the feet, but rather that the body schema is developed reaching sufficient internal coherence to establish references between the movements of the limbs and trunk in the different types of steps. It is therefore a matter of reaching a certain level of organization that allows sufficient discrimination. The spontaneous *sensory illusion* of believing that there is displacement, together with the corresponding tendency to automatically exclude the defect, is typical of diffuse and unstable sensory activity.

From all that has been said, it follows that ordinary walking in a more or less inactive state remains for a certain time at a very primitive level from the point of view of the subject's own perception. In a strict sense, walking steps are only perceived after some time, when the sensory level has increased considerably.

25.1.5. Subjective body orientation during walking

The different subjective size of the body is accompanied by a different *subjective orientation of the body in space*, as occurs with the steps. The subjective position of the body undergoes certain turns that we have already studied when dealing with the orientation of the body schema statically (Sec. 24.4). The body seems to rotate in the three directions of space, as follows: it rotates on itself to the right and backward and also tilts to the right side and forward. These sensations are not easy to specify by the subject, especially in the first sensory phases. It should be noted that not all phenomena manifested at a given moment are equally evident. We must therefore stick to simple indications of the patient.

The aforementioned body deviations tend to become evident when the body schema begins to emerge. When subject M is in a quasi-inactive state and perceives the direction of walking strongly inverted, he cannot say whether it is the whole body that is moving in the opposite direction to the real one. Rather, it seems that the inversion is restricted to the lower limbs, which are as if disconnected, without reference to the body schema. Recall the test of moving the feet without translational movement, in which the subject was unaware that he was actually at the same place. However, although the sensations about his own body are extremely diffuse at the beginning of walking, as soon as the subject feels his body more defined, he clearly feels that the gait trajectory does not take place on a horizontal plane, but as if going down a slope. When the body is felt more clearly, the different deviations are better felt. The subject feels his body tilted forward and to the right, and somewhat rotated on itself to the right, such deviations corresponding to the felt deviations of the steps between 80° and 40°.

It is reasonable to interpret these tilts and rotations as residual phenomena of body schema inversion, an inversion that is not possible to obtain more pronounced due to the disintegration that the body schema undergoes when the sensory level is very low (see Sec. 24.4). It could be assumed that the body schema tends to make a spiral turn in space, following a helical trajectory. Given the tendency to inversion, it is conceivable that there is a moment in which, being the tilt of the body schema very strong, the slope referred to above would be very steep. However, it has been repeatedly found that the subject does not perceive such anomalous orientations, which

seems logical since he would lack any reference to the body schema at such low sensory level. Thus, it is understandable that the subject always decisively rejects the question we ask him if at the beginning of walking it seems to him that he is walking almost upside down, or at least that the slope he perceives is so steep that it seems to be going down a vertical wall with his body tilted 90°. None of this can be perceived because the body schema does not subsist at such a low sensory level, and in order to study the issue it is necessary to stick to the data of the more attenuated disorder. It is also possible that difficulties may arise for the subject to localize his own body very inverted in space, or that mechanisms of exclusion or attenuation of the defect may intervene. We have already seen in the test of moving the feet as if walking but without moving from the site, the deficit that arises to appreciate the lack of translational movement. Therefore, there is a limit to how far we can go in analyzing the process, and at present it has not been possible to make any further progress on this issue.

However, we have found it possible to study to some extent the type of tilt felt by subject M. He is asked to walk in an almost inactive state on a board inclined about 45° to the horizontal. If he walks ascending he tends to perceive a horizontal path. But if he walks downward he feels that there is a large almost vertical drop. These sensations are greatly diminished under facilitation, and also vary with the length of the path. A similar situation occurs when going up or down a staircase. If he goes up the staircase in an inactive state, he senses a lesser slope than the actual slope and that the path tends to be horizontal. But if he goes down the stairs, he feels a much steeper slope than the actual one, and protects himself by holding on to the handrail. Even with his eyes open, he cannot descend a single step without helping himself with the wall or grasping the stair handrail. In contrast, under facilitation by maximum muscular effort he can descend more easily.

In general, in the conditions of ordinary life (medium facilitation), going up or down a staircase involves considerable difficulty for the subject, since the tactile proprioceptive information is deficient or unclear, the range of motion is felt to be reduced and there is imprecision about the displacement of the own body, apart from the deviation of the direction of walking, incongruities, etc. Under intense facilitation, these difficulties usually appear only at the beginning of walking. The difficulties are aggravated if the staircase is carpeted, because the steps are cushioned and the steps are less visible. On stairs with turns, without corners, with steps with uneven bottoms, the subject can easily fall backwards. For these reasons, it is very common to see him, outside the tests, descend a staircase with great caution and without ever letting go of the handrail. This could be interpreted as a visual difficulty, but in this case the major defect corresponds rather to touch, and here most likely to the subjective increase of the slope. Despite the many significant sensory impairments of patient M, his behavior in ordinary life at first glance is entirely normal, with the important exception of the case of stairs where he is at risk for many mishaps.

After dealing with numerous issues related to the disorder of perceived direction (orientation) during walking, all of them simple consequences of phenomena already studied, the most relevant characteristics are summarized as follows: We have first examined the perceived trajectory during walking and then the changes in the body schema during this process, aspects that evolve in parallel. Thus, a certain

felt size of the steps is accompanied by a certain felt size of the body, and likewise, a felt direction of walking is accompanied by a felt orientation of the body schema in space. We have only referred here to two very relevant aspects: *size and direction (orientation)*. Synthesizing further, it can be stated that the whole process follows a *spiral development in space* (helical) due to the different reduction of both the body schema and the steps taken, and to the corresponding inversion in the steps and in the body.

25.2. INDUCED OBJECTIVE POSTURAL DEVIATION

All the orientation disorders described so far are strictly sensory in nature, and the phenomena on the body schema, both static and during walking, are subjective and can only be studied indirectly through the sensations of the patient. Something quite different occurs in the phenomena we are now going to describe. These are motor deviations of the body, i.e., alterations in the postural tone that result in changes in the attitude of the body, either in its entirety or partially in its segments, depending on the conditions. These phenomena are thus revealed in an objective and direct way. Such postural motor deviations, of a spontaneous nature, are well-known phenomena in clinical neurology. These are: head tilts, deviation of the whole body (Romberg's sign), deviation of the index finger in finger-pointing tests, and deviation of the whole body during walking.

In all these motor phenomena the deviation always consists of a rightward rotation, as in cases of subjective deviation. Facilitation also has a considerable effect in correcting postural motor deviation. Thus, these phenomena could be related to the sensory disorder of orientation in touch. Such a disorder would alter in a primary way the sensomotility (Exner 1894), and would induce secondarily a motor deviation.

First, the motor phenomena will be described, and then their interpretation will be addressed.

25.2.1. Postural deviation of the head

The head of patient M is usually spontaneously tilted to the right and rotated a little around its axis. In this test, patient M is in an inactive state (or semi-inactive, as corresponds to his usual state) and his head is passively moved in various directions, with the neck relaxed, leaving the head free in any position. If he is then asked to put his head in a natural position, he ends up putting it in the position indicated at the beginning of the test. If at that point he is asked to apply facilitation by means of intense generalized muscular effort, the deviation is corrected immediately by straightening the head. The objective deviation of the head in the inactive state is about 30°. If there is ever any objective head deviation with the mentioned facilitation, it will be barely perceptible. Sometimes a leftward deviation can be observed, also small, probably to counteract the small subjective sensory deviation felt even under facilitation. During the test, the subject is seated with eyes closed, although with eyes open the result hardly changes. It is very important to note that the subject is not asked to give indications about the position he perceives of his body, but is

only asked to try to adopt the most natural or comfortable posture. Thus, an almost reflex function is allowed to act.

25.2.2. Body deviation. The Romberg test

If the subject is standing, in frontal position, with eyes closed, after a few seconds he tends to deviate with the whole trunk to his right and somewhat backwards, showing a positive Romberg's sign (Romberg 1846/1853), although moderately. The degree of deviation is measured by the displacement of the tip of the nose in the course of the test. After one minute, the deviation is 18 cm, the whole body gradually turning in the direction indicated above. Under facilitation by strong muscular effort, the deviation is corrected, and the tip of the nose is only two or three centimeters away from the normal position. If the facilitation persists, there is a real deviation to his left that counteracts the subjective deviation disorder. Performing the test applying facilitation from the beginning, the deviation obtained is 4-5 cm in 18 seconds, i.e., about one third of the value for the inactive state, in about one third of the time. Under facilitation he also deviates a little backwards. In other tests in an inactive state, it can happen that after he has deviated a few centimeters, he almost falls to the right, but this is not common.

As in the previous case of the head, the deviated position is more natural than any other, although when a certain degree of postural deviation is reached, an action can cause a static imbalance and a fall.

25.2.3. Deviation of index fingers

In this test, subject M is seated with eyes closed and arms extended forward pointing with his index fingers. If asked to maintain this posture, the index fingers tend to deviate slowly and continuously to the right, the right index finger somewhat more than the left. The values are similar to those of the Romberg test. In the inactive state the index deviates about 30 cm without any perceived displacement, but if facilitation by strong muscular effort intervenes at the end, he spontaneously corrects the deviation even without having received any command to do so, and brings the finger towards the center. Performing the test applying facilitation from the beginning, the same finger deviates only 5 to 12 cm in different tests, and in a time of 15 to 25 seconds, without increasing the deviation no matter how long the test is prolonged, the average being about 10 cm to the right.

In the test of moving the index finger with the entire upper limb rigid, in the direction from bottom to top in the corresponding sagittal plane, the same lateral deviation occurs, moving away from that plane. In all these tests, the position of the upper limbs deviated to the right is also the most comfortable or natural position, i.e., the one that corresponds to the neutral motor position, i.e., that of functional balance.

The usual clinical test of touching the tip of the nose with the index finger shows the same type of deviation. In the semi-inactive state, both index fingers deviate in such a way that the finger touches the right cheek near the nose, at other times it touches under the right eye, etc. In such state, the subject feels neither the finger nor the place touched, since the pressure applied (semi-inactive state) only

allows him to obtain a tactile sensation of the type of phase I or perhaps even phase II. In addition, the pointing movement is very peculiar (see its mechanism in the section on body schema), and when the subject initiates the requested movement, it does so with an abrupt impulse and without defined direction.

The aforementioned deviation would be even greater if it were not accompanied by some facilitation due to the effort required to give some coordination to the finger-pointing movement. Under facilitation (always by muscular effort), the deviation is almost completely suppressed and both index fingers can easily reach the tip of the nose, although sometimes there is still some deviation of the right index finger touching the right wing of the nose. Apart from this, when facilitation is applied, the sensation of finger and nose contact is much better than in the inactive state, although normalization is not reached since there is proximal deviation for the finger (it is felt shorter) and also for the nose (it is as if he touches towards the root of the nose). Under facilitation, the initial impulse referred to occurs to a much lesser degree and the movement is better coordinated.

25.2.4. Deviation during walking

In the previous tests on direction of walking, the reversal or deviation of direction is felt by the subject, walking the subject in a straight line and forward, either with eyes open to comply with the command not to deviate, or with eyes closed but being guided appropriately. However, if with eyes closed (and sometimes even open) he is allowed to walk freely, a great tendency to deviate objectively to the right is evidenced, more so in the inactive state than under facilitation. In a walk of about 5 meters taking 7 or 8 steps with eyes closed and the instruction to walk forward by himself, he deviates to the right by 1.70 meters at the end of the walk. In contrast, under facilitation, he deviates only 0.60 meters. As in the other examinations, in the inactive state he shows three times more deviation than under facilitation (see Table 25.5). In this type of test, it is easy to obtain, in the inactive state, a "star walking" by asking the subject to walk a few meters alternately forwards and backwards, as he will deviate respectively to the right and to the left.

It is noteworthy that, as in the previous motor deviation tests, spontaneous gait with objective deviation seems to the subject to be more natural and comfortable than correct gait when guided by the hand, regardless of whether there is a subjective deviation. What is important now is the most natural posture that the subject can feel, whether static or in movement; and the objectively deviated gait is felt by the subject to be naturally correct, and he is not bothered by the postural anomaly. Instead, when he is guided and walks correctly, there is something that disturbs him causing the effect of walking in a situation of imbalance.

The deviated posture to the right in patient M is so natural that if he is lying supine, in the inactive state, and is urged to adopt a natural position, he usually ends up bent at the waist to the right. But if he applies facilitation by muscular effort, he corrects the deviation spontaneously without receiving any special indication.

Considering all the phenomena, the general characteristics are the same in all cases, the following two aspects being noteworthy. One is the different deviation in

the inactive state and under facilitation. The other is that the deviation appears as a neutral motor state, i.e., as a posture of preference. As for the difference between the inactive and facilitated state, the respective quantitative values of deviation maintain a practically constant ratio for the different tests (see Table 39). The deviation in the inactive state is in all cases about three times that under facilitation. In degrees of deviation (index finger and gait tests), about 25°-30° is obtained in the inactive state, and about 10° under facilitation. It can therefore be stated that there is a *deviation of the postural model* according to the respective states. Of course, the fact that the aforementioned motor anomaly appears segmentally, i.e., in the head, limbs, or in the whole body (Romberg test and walking), depends only on the degree of freedom of movements in the corresponding tests.

Table 25.5. Motor deviation values in patient M, in inactive state and under facilitation by strong muscular effort. Se text in Secs. 25.2.1, 25.2.2, 25.2.3 and 25.2.4.

Test	Inactive	Under facilitation
Head	30°	10°?
Romberg	18 cm in 60 s	4-5 cm in 18 s
Index fingers	30 cm in 60 s	10 cm in 20 s
Gait (6.5 m)	1.70 m	0.60 m

Patient T has also presented deviations to the right side in the Romberg test, index fingers test, finger-nose test, etc. The Schneider patient (Goldstein and Gelb 1919) has also presented them to some extent and they have been attributed by these authors to the involvement of the cerebellum in the brain lesion, in our opinion with insufficient foundation.

Addressing at last the interpretation of the postural anomalies, it is clear that, if the other phenomena shown by our patients are not considered, those postural anomalies would have the appearance of a motor tone disorder, either cerebellar or cerebral, particularly of the frontal lobe in the latter case. Apart from this, other characteristic defects of a cerebellar lesion are entirely lacking.

> The cerebellum and frontal lobe affect the red nucleus which is an essential organ in the distribution of postural tone. Lesions of this nucleus cause postural abnormalities of the same type (torsion of the head and trunk), according to several authors and ourselves (Gonzalo 1935, Kleist and Gonzalo 1938).
>
> Such postural anomalies do not bother the patient at all, but on the contrary, they constitute new equilibrium positions of the static system, or "new comfortable postures" according to Goldstein (1926).

In our patients, any cerebellar or frontal lesion is excluded, since there is only a parieto-occipital lesion corresponding to the central syndrome here studied. However, postural deviations are also known in parieto-occipital lesions, as in the cases reported by Hoff and Schilder (1927) and others; although it could be hypothesized that cere-

bral repercussion somehow affects the frontal lobe, which is more related to postural tone than the parietal lobe. However, the previously studied sensory disorder of orientation in touch does not enter into this argumentation at all. If we have included the phenomena of postural deviation in this Sec. 25, it is precisely because of their formal similarity with the mentioned sensory disorder, thus trying to obtain an interpretation as unitary and simple as possible for the whole of the various phenomena. In this regard, it should be admitted that postural function, being linked to sensomotility, depends both on the purely motor element and on sensitivity. It could then be thought that the orientation disorder exists primarily in tactile sensitivity and exerts a parallel influence on postural motor function, which is thus secondarily altered.

By examining certain alterations described in the functional complex of sensomotility, we find aspects of great interest for our purpose. Thus, in both cerebellar and frontal lesions, which present the corresponding defect of postural tone, Goldstein (1926) has also observed special perceptual disorders in several sensory systems, such as tilted perception of the visual image, deviations in the localization of tactile and auditory stimuli, etc. This symptomatic complex has also been confirmed in some cases of similar lesion by Hoff and Schilder (1927), Fischer and Pötzl (1924), Marburg (1931) and other authors. It happens that postural tone deviation due to cerebellar or frontal lesion induces some sensory spatial deviation, towards the same side in cerebellar lesion and towards the opposite side in frontal lesion.

In the cases reported by Goldstein (1926) with a cerebellar lesion, the visual deviation from the vertical is mainly perceived by the homolateral eye as a rotation to the same side of the lesion. There may also be certain deformations of the visual image of the object, in some of its axes, etc.; and in addition, different sensory stimuli (tactile or auditory) can influence the aforementioned visual deviation. Analogous manifestations are also found in touch, for example, drawings on the skin are felt deformed in the direction of the pathologically deviated motor tone. There is therefore a spatial sensory alteration due to the motor postural alteration, and such sensory alteration is diversely influenced by intersensory action. But there is also the reciprocal effect that the sensory factor exerts an influence on muscular tone, thus closing a circle of mutual effects. Thus, it is possible to obtain deviation in pointing, and deviation in body position, by means of acoustic, tactile, visual stimuli, etc., the deviation being towards the same side of the body on which the sensory excitation acts [phenomena studied by Goldstein (1926) and later confirmed by Hoff and Schilder (1927)].

In short, the postural deviation due to cerebellar lesion has an effect in the same direction on the sensory space (deviation of the localization of a tactile stimulus, tilted vision, etc.); and reciprocally, modifications in the sensory field by its excitation determine changes in the distribution of postural tone. That is, mutual sensorimotor inductions occur due to the special lability of the sensorimotor functional complex.

Two cases of Weizsäcker (1924), seemingly suffering from a unilateral labyrinth lesion, would belong to the first type of induction mentioned (sensory spatial deviation secondary to a primary deviation of postural tone). In these cases, the alteration of postural tone secondarily caused a tilted visual image. We have observed a similar induction phenomenon in a patient diagnosed with left cerebellar abscess and successful-

ly operated on. This patient was affected by very marked cerebellar motor disorders on the left side of his body, and showed episodic disorders of visual image orientation during the acute period before surgery. He spontaneously said that sometimes, upon waking up in the morning, he had seen for a few moments the whole visual scene tilted 90° to his left (side of the lesion). However, his vision was not blurred and did not show any color change, contrary to what occurred in subject M.

With respect to these spatial sensory alterations secondary to postural deviation, it should be noted that, for example, the visual image tilt is moderate and, above all, it is a disorder that has nothing to do with asynchrony and dynamic reduction of the sensory field. The induced disturbances are nothing more than orientation changes in the sensory space due to the effect of the sensory-motor correlation that acquires a new aspect due to an alteration of the motor element. Regardless that we may return to this topic in another part of this work, for the moment we bring it to an end.

Coming back to the starting point, and in the light of the various phenomena considered (mai nly sensory deviation in postural abnormalities due to a cerebellar lesion), it seems appropriate to reciprocally interpret the motor disorder in subjects M, T and even Schneider, as an effect induced by the inverted or tilted sensory field. Although inversion in patient M occurs in all sensory systems (visual, tactile and auditory), when dealing with motor deviation it is sufficient to refer to the disorder of orientation in touch since it is most strongly linked to the sensorimotor complex. Thus, the interpretation is different from that of Fischer and Pötzl (1924) and other authors. These authors believe that the lesion of the angular gyrus or neighboring regions causes the same postural tone disorders as the cerebellar lesion, thus postulating the existence of a primary motor center in that brain region which, however, is in a fully sensory area. Instead, the explanation given here, much simpler and unitary, of an *induced postural deviation* (motor deviation secondary to the deviation of orientation in touch) would suffice. Thus, the body schema of subject M is rotated to the right both subjectively and by posture (objectively), by the sole action of the sensitivity deficit.

Finally, for the sake of clarity, we summarize in Table 25.6 the different sensorimotor pathological phenomena, especially in the case of cerebellar lesion (primary motor disorder) and in the case of central syndrome (primary sensory disorder).

Table 25.6. Sensory-motor dynamic phenomena.

Type of lesion	Primary manifestations	Induced manifestations
Cerebellum	Homolateral deviation of the postural muscular tone	Homolateral deviation of spatial values in the sensory field (tactile, visual, etc.). Reciprocally, sensory stimuli may deviate postural tone.
Parieto-occipital (central syndrome)	Contralateral deviation of the sensory field (tactile, visual, etc.)	Motor postural deviation in the same direction as in the sensory field.

26. Theory of orientation in the sense of touch

26.1. ORIENTATION AND LOCALIZATION IN THE SPIRAL DEVELOPMENT

We have seen how the orientation disorder is interconnected with that of stimulus localization. Both functions, inversion and proximal deviation, always go hand in hand, and in certain phases, such as contralateral localization, they are so closely linked that they are equivalent. In fact, a change of laterality is a change of orientation. Such characteristics deserve a careful analysis in order to address the theory of spatial orientation.

26.1.1. Differences from allochiria

Contralateral localization of stimuli in subject M leads us to some brief considerations on allochiria (contralateral localization of tactile stimuli), in order to properly delimit these various contralateral manifestations. Allochiria was first described by Obersteiner (1892) in certain migraine auras, then studied by Jones (1908), by Kramer (1915, 1917) in brain and medullary lesions, by Redlich and Bonvicini (1911) in brain lesions, and finally by Schilder (1923/1935) in a brain case and two cases of tabes dorsalis. This phenomenon has also been studied experimentally, first by Mott (1893) in monkeys with spinal cord hemisection, and more accurately by Dusser de Barenne (1913) in dogs and cats by combining spinal cord hemisection and strychnization. The clinical observations reported are brief and do not exhaust the study of the phenomenon, apart from the fact that it is often a transitory phenomenon. Patients are uncertain to distinguish the side of the body that is touched, and sometimes tend to refer the sensation to the symmetrically opposite side (allochiria). Although it is a rather rare symptom, other cases have been subsequently reported by various authors but without much more precision than in the above-mentioned observations.

We have observed a case of allochiria in a subject with biparietal brain lesion (Fig. 26.1), who described the phenomenon spontaneously during the examination since he had become aware of it under special conditions.

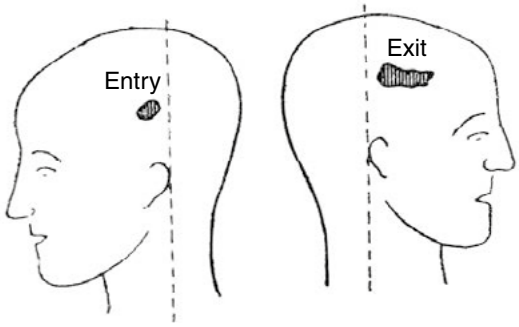

Figure 26.1. Patient U.G. Biparietal brain injury (transverse trajectory of the bullet, somewhat oblique forward and to the right). The patient showed moderate left hemiparesis and hemihypoesthesia, various disorders related to agnosia, apraxia and aphasia, some concentric reduction of the visual field, etc., and allochiria: tactile stimulation on the right side (healthy side) was localized either on the left (affected) side only, or on both sides at the same time. The reverse case (from affected to normal side) never occurred.

Patient U.G. suffered a gunshot wound (see Fig. 26.1) in 1938 during the Spanish Civil War. He first suffered from a left hemiparesis with sensitivity disorder on the same side, language difficulty (especially nominal amnesia), visual fatigue, etc.; and remained so for about two weeks. He also showed a set of symptoms of agnosia and apraxia such as left-right confusion, finger agnosia, constructive apraxia, agraphia (or dysgraphia), acalculia, amnesic aphasia especially for colors, and dyspraxia, especially in the left hand. All this was possible to be considered as Gerstmann's syndrome (Gerstmann 1931) with predominance of apraxia.

The left hemiparesis diminished rapidly, while hemihypoesthesia on the same side was still evident. He spontaneously reported during the examination that when walking in company and carried by the right arm, he felt the contact on the left side (the affected side). During the examination he spontaneously reported that while walking in company held by the right arm, he felt the contact on the left side (the affected side). On examining touch, it was found that stimuli on the right side were indeed localized symmetrically on the left side. The opposite never occurred. The contralateral localization, symmetrical to the excited site, took half a minute or less to occur, but at the same time it was possible to perceive the stimulus at the stimulated site. Thus, the sensation was double, on the normal side and on the affected side. This occurred mainly when the stimulus was strong, the sensation being weaker and diffusely localized (as deep currents) on the contralateral side. If the stimulus applied was very weak, it was only perceived contralaterally. By lightly rubbing the right cheek with a very thin paper (tickling), he felt after about 15 seconds the need to scratch the left cheek, without perceiving anything at all on the right cheek. When the change of side occurs, a dizzying sensation may appear due to confusion in distinguishing which side is actually stimulated.

Examination of tactile sensitivity on the left side revealed some impairment of all functions; for example, localization with proximal deviation, a deficit in perception of

passive joint movement and postures, a deficit in Weber's test, a tendency to contract the musculature to enhance perception, some failures in manual recognition of objects, and indirect way of recognizing them, etc.

The visual field showed a very moderate concentric reduction and several annular scotomas due to fatigue. There was no inversion of chromatic isopters.

In summary, as far as allochira is concerned, there is a tendency to localize stimuli applied on the healthy (or less affected) side on the contralateral hypoesthetic side, and in a symmetrical way. This type of examination, carried out rapidly during the daily care of a large number of war wounded with brain injuries, cannot be considered complete for an exhaustive study of allochiria, but it will suffice for the moment as a guideline. This case examined by us can be equated to the case of Schilder (1923/1935) suffering from hemiplegia and hemihypoesthesia, apparently due to a capsular and perhaps also a cortical lesion. This patient also suffered from allochiria only when the healthy side was stimulated. We discard the numerous hypotheses to explain the phenomenon, especially those related to the body schema, as in the case of Schilder, as being very poorly founded. Instead, it seems clear that the simplest interpretation of these cases is to relate them to hemiplegic synkinesias. The affected side has an abnormal permeability to stimuli from the healthy side. This would occur either by muscular innervations that also provoke reflex or synkinesic contractures on the impaired side, or by tactile stimuli on the healthy side that tend to be localized on the affected side. Furthermore, both in synkinesias and in these cases of allochira, the transfer is symmetrical. In the light of the above, it is not possible to identify this type of allochiria with the one obtained in the tactile inversion studied here, since both the mechanism and the phenomena are completely different.

However, it cannot be ignored that other described cases of allochiria do not fit the type of the two above. Thus, in the two cases of Kramer (1915), one with bulbar apoplexy and the other with cortical hemiplegia, as well as in the case of Redlich and Bonvicini (1911) with cortical hemiplegia, the change of stimulus localization was, contrary to what was indicated above, from the affected to the healthy side, also symmetrically. Here, as in some tests of monocular polyopia, it might be thought that the stimulus is deviated toward the most sensitive site. The same occurs in the mentioned experiments of Dusser de Barenne (1913), although strychnization then intervenes in a special way.

Singular phenomena have also been described, such as the alloparalgia of Fuchs (1908) in peripheral nerve lesions, which consists of paresthesias and localized pain contralateral to the lesion. This is a disorder that could be similar to the contralateral "repercussions" of the functional disorder in peripheral nerve injuries, first described by Bourguignon (1923, 1932) (in radial nerve injury, excitability is altered in the radial nerve of the opposite side), and later confirmed by Altenburger (1933, 1937) in different ways.

In conclusion, all these types of contralateral localizations do not seem to have anything to do with the contralateral localization in the tactile inversion described in the present book. In the latter tactile inversion, there is not only contralateral but

also proximal deviation, whereas in classic allochiria the localization is symmetrical, a fact that all authors have always insisted on and that in our case of allochiria is very clear (slight excitation of the healthy cheek leads the patient to scratch the affected cheek). Trying to find similarities, one could suppose that there is an inverted orientation in the cases in which the perception of the stimulus moves from the affected zone to the healthy one, as occurs in the cases of Kramer (1915) and Redlich and Bonvicini (1911). But the transposition should not be symmetrical but with a strong proximal deviation towards the body axis. Until more complete and conclusive analyses are available, all known cases of allochiria cannot correspond to the tactile orientation disorder described in the present work. This last disorder does not refer, of course, only to the lateral transposition of a simple touch, but also to a whole set of phenomena and tests described here on the inversion of a cutaneous rectilinear stimulus, motion inversion of a moving object on the skin, inversion of joint movement, etc., all these phenomena following well-defined rules.

With respect to vision, similar considerations must be made with respect to the visual alloesthesia of Hermann and Pötzl (1928)[1]. This disorder consists, as its name indicates, of an allochiria of the visual field. The visual scene or peripheral images from one side of the visual field are perceived on the opposite side. Even more than in the described cases of tactile allochiria, in these visual cases the phenomenon is sporadic and very ill-defined, like change of site in scotomas due to migraines and similar states. The cases reported by these authors correspond to old clinical observations that are very superficial and inappropriate for drawing conclusions, apart from the transience of the visual disturbances. More evident is the last of these cases, observed in 1925, with several areas of softening, mainly in the right parieto-occipital convexity, where visual allochiria appears more clearly: objects in the periphery of the left field tend to be perceived on the right side. This phenomenon also appears sporadically. If the change of side is from the amblyopic to the healthy side, a reversal of orientation could be admitted, although much data is lacking to be sure. Given the imprecision of the tests, it is not known whether there was proximal deviation (towards the center of the visual field) when changing from side to side, and nothing is indicated about inverted or strongly tilted vision with respect to the vertical. Regarding the interpretation, the aforementioned authors make extensive comments, in contrast to the exiguous clinical observations, establishing a parallelism with tactile allochiria, and especially with the characteristics of the experimental studies of Dusser de Barenne (1913). Probably, as the authors were impressed by the lateral transposition, they do not consider the general problem of inverted orientation in vision.

26.1.2. Spiral development in vision and touch

Having established the difference between true spatial orientation inversion and allochiria, we now return to the issue of orientation as an essential factor in spatial localization. In our patients with brain lesions, the spatial disorder is of such a nature

[1] These authors use the term alloesthesia in their work.

that, by combining inversion with proximal deviation, a *spiral development* of localization (spiral deviation) is obtained along the process. We have already referred to this spiral trajectory above. Suffice it to recall Figs. 21.14 and 24.2 for a point stimulus on a hand and on the face respectively, Figs. 24.4 and 24.7 for a rectilinear stimulus under analogous conditions, Fig. 24.9 for a stimulus moving on the skin, and Figs. 25.1, 25.2 and 25.3 for articular movement leading to a subjective trajectory during walking shown in these figures. Thus, the spiral trajectory is a feature of general significance that we must use as a starting point in the theory of tactile orientation.

This type of spiral trajectory is as characteristic of the tactile system as it is of the visual system. The spatial similarity of both systems allows a more complete study of the different phenomena. Proximal deviation is more satisfactorily determined in touch, whereas vision is more appropriate for studying the regularity of the inversion rotation and its quantitative evaluation. Apart from this, there are other differences described next. For the visual field, the demonstration of the spiral trajectory is simpler than for the tactile field, since the structure of the latter is less homogeneous and lacks a central point (or fovea) on which to center the mentioned spiral development.

In cases 2 and 3 of Fig. 26.2, a diagram of the spiral development for the visual field is shown, in the case of a stimulus of varying intensity and situated at the visible end of a horizontal meridian. By supplying sufficient luminous intensity, the stimulus can be seen in that meridian more or less peripherally; but as the intensity decreases, the perceived stimulus tends to follow a spiral course, progressively approaching the center of the visual field and moving to the opposite side, finally coming very close to the center. Thus, a contralateral centripetal translation is obtained, i.e., the aforementioned spiral deviation is obtained as a combined effect of the rotation of the visual field and the concentric reduction of the field (proximal deviation).

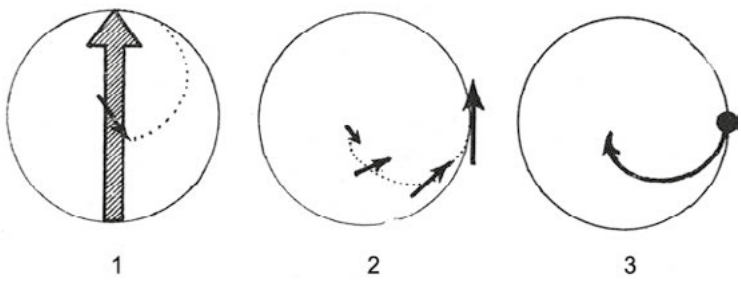

Figure 26.2. Diagram of the spiral trajectory in the visual field. 1: Arrow in the center of the visual field whose perceived image is becoming inverted and decreasing in size as the illumination is suitably reduced. In this process, the upper end of the arrow (as well as the lower end) describes a spiral curve due to the joint effect of the orientation reversal process and the reduction of the size of the arrow. 2: Arrow located at the visible end of a horizontal meridian of the visual field. When the illumination decreases, the image perceived during the inversion process follows a spiral with some displacement with respect to the previous case. 3: Spiral trajectory as in case 2 but here the test object is a small circle situated peripherally.

It should be noted that when studying the inversion of the visual image orientation, the test was made with the arrow in the center of the visual field (case 1 of Fig. 26.2), so the mentioned spiral translation was not obtained. However, since the size of the arrow image is progressively reduced during the image inversion process, the result is that each end of the arrow image follows a spiral trajectory progressively approaching the center. Recall the reduction of the perceived trajectory in the inverted visual perception of a moving object, and also in the steps perceived as inverted. In the above-mentioned test with central position of the test arrow, if the position of the arrow is vertical, when the image of the arrow is inverted, its upper end will be down and close to the center; but if the position of the arrow is horizontal, the change of the image will be from one side to the other side, which corresponds to the typical case of inversion in the tactile field. In relation to visual inversion, it should be noted that in the corresponding sections (Secs. 12-14 in Vol. 1) only one experiment on peripheral inversion (Fig. 13.18) had been reported, pointing out only the tilt of the image with respect to the vertical test arrow, but not its absolute position in the visual field, i.e., its spiral translation had been omitted. But now this can be shown in case 2 of Fig. 26.2.

As for the tactile field, the spiral deviation does not seem to show such a simple and unitary character, since in addition to the irregularity of the field in question, both morphologically and functionally, certain fragmentations take place. Thus, a stimulus in the cephalic area, for example in the face, is not perceived in the lower part of the body (the feet or even in the pelvic region). In this case, the inverted perception, in its contralateral rotation around the vertical axis of the body, is localized also in the head and toward the vertex (Figs. 24.2 and 24.7). A deviation more similar to that of the visual field is only obtained when the stimulus acts on the anterior plane of the trunk, over the chest, as shown in Fig. 24.3.

Thus, the difference in the spiral rotation between the visual and tactile fields lies in the fact that for the former it always takes place around the central point of the field, whereas in the latter there are, as we know, *independent rotations* around the axis of the body, since there is no central point as in the former case. During the spiral reduction, the tactile field is fragmented into the three zones already studied, as shown in Fig. 26.3. Apart from this peculiarity, there is a similarity between vision and touch in the inversion rotation, and each lateral half of the sensory field (visual and tactile) can act independently by rotating in opposite directions, as is schematically depicted in Fig. 26.3.

26.1.3. Spiral development of the sensory field. Sensory organization law

The spiral process, considered more broadly, involves a *spiral development of the sensory field* (tactile or visual) that affects the whole sensory organization. Thus, the greater the degree of inversion, the more pronounced the proximal deviation, i.e., the greater the reduction of the sensory field. This reduction reaches all types of functions, thus there is a structural change of the field resulting in a *residual organization field*. Its characteristics refer mainly to inversion, reduction, irradiation and also to other sensations, although in the spiral process the spatial organization is of special significance.

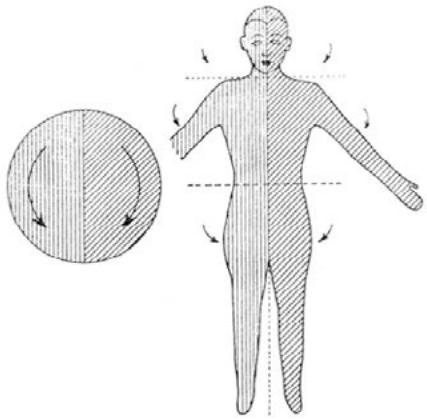

Figure 26.3. On the left: In the inversion of the visual field, each half of the field rotates around the center independently. On the right: In the inversion of the tactile field or body schema, each half of the body has 3 independent zones for inversion, namely, head, upper limbs and lower limbs. Note that the opposite rotations in both halves have the same sense in vision and touch.

It is not possible in such a situation to determine independent factors, such as orientation inversion, field reduction, or irradiation. On the contrary, these factors are aspects of a same process in which the spatial structure of the field tends to become a residual structure. Inversion is a particular aspect of localization, changed in sign by special circumstances that we shall discuss later. The reduction derives from the contraction of the sensory dimension affecting both space and time, thus explaining the alterations in motion perception, spatial acuity, size, etc. Irradiation means a reduction of local specificity, i.e., dilution of the local sign or spatial value. This dilution manifests itself as a loss of cohesion between the parts of the field, this being more evident in touch than in vision. Naturally, the greater the residual character, the greater the loss of functional differentiation and the greater the destruction of functional entities (individualities). In this way, the normal complexity of what is heterogeneous or discontinuous is changed, by effect of the reduction, into homogeneous and continuous, where all traces of organization end up vanishing.

This overall, unified process of sensory field reduction is illustrated in Figure 26.4, where different phases of the process are depicted for both the visual and tactile systems in patient M. In both systems the rotation is to the right side (clockwise). In vision, this occurs for the right eye in foveal image. For touch it occurs when the whole tactile field (i.e., the body schema) is considered, either static or during walking.

In the case of vision, when the visual image is gradually tilted until it ends in an almost complete inversion, the field becomes smaller and smaller, i.e., the perceived image reduces in size and at the same time becomes diffuse due to irradiation, until a moment arrives in which the entire configuration is lost and only a weak, amorphous and achromatic luminosity remains. As for the color, the almost white arrow in optimal vision, passes through gradually darker green colors due to chro-

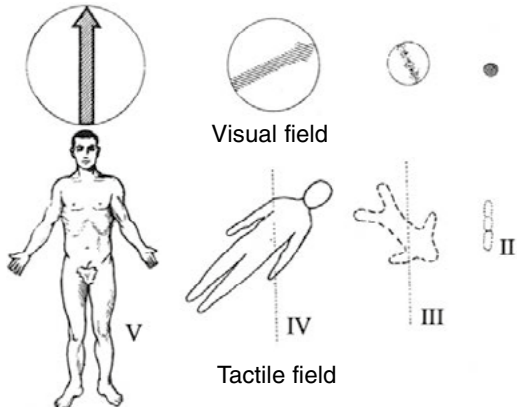

Fig. 26.4. Spiral reduction process of the sensory field through the different phases of residual organization. Note the similarity of phenomena for the visual field and for the tactile field. In both, the degree of inversion is parallel to spatial contraction (concentric reduction, proximal deviation) and to irradiation. At the end, all differentiation or organization disappears. See text for further details.

matopsia, until even the color disappears when the whole configuration vanishes. Thus, at the end, all traces of organization disappear.

Similarly occurs in touch, although the whole process cannot be determined as directly and simply as in vision. Indeed, in moderate reduction, the tilt of the body schema and its reduction in size are obtained immediately with suitable experimental conditions. But we know that with a subjective tilt of more than 50°-60°, the body schema vanishes due to loss of cohesion (independent rotations of its parts, etc.), being then impossible to follow directly the course of the reduction. For this reason, phase III as depicted in Fig. 26.4 is an ideal construct based on fragmented determinations, since by means of local excitations in that phase the size of the body schema can be inferred (by means of proximal deviation), as well as the subjective orientation. Apart from that, the corresponding irradiation and alterations derived from it end up, as in vision, erasing all functional differentiation, as it occurs already in phase II, and mostly in phase I.

In relation to the reduction process, some peculiar aspects should be pointed out. First of all, it should not be forgotten that the distal sectors of the field lack inverted representation since the proximal deviation is then present to the maximum, so particularly in touch, the extremities cannot suffer any inversion. This constitutes an essential difference with the usually known allochiria, where the side transposition is symmetrical. It is also important to note, for subsequent theoretical interpretations, the noticeable reduction of the field size as inversion occurs, and especially in touch, the fragmentation into parts (loss of mutual references).

Finally, we must emphasize that a deeper understanding of the inversion phenomenon relates this anomaly to a disturbance of the whole spatial structure, especially associated with spatial reduction (field shrinkage, etc.). Inversion is thus one of the fundamental aspects of the *spiral development of the sensory field.* This process

accounts for all types of sensory activities and the various possibilities of general organization of the field. This statement thus constitutes a law of sensory organization, the psychophysical basis of which will be discussed in the next section. We now point out that this statement constitutes one of the two fundamental laws of the brain dynamics described in this book. The other law refers to the repercussion effect of the lesion depending on its magnitude and position, as we already know. All this will have to be dealt with more extensively in another work, but its importance can already be appreciated now.

26.2. MECHANISM AND STRUCTURE OF SPATIAL ORIENTATION[2]

In the section on visual image orientation (in Vol. 1), the inversion was interpreted as an effect of retinal autonomy (with primary image inverted) when the functional visual-haptic complex (visual-tactile correlation) that would normally provide normal orientation was broken. Later it was shown that the orientation disorder also occurs in other sensory systems, as now in touch. This type of disorder is thus of a general nature, and needs to be studied more extensively to satisfy the various questions that arise in a general way. Also in touch, inverted orientation is interpreted by referring to the primary image of the inverted projection in the brain. Instead, it would be impossible in a sense as fundamental and primary as touch to derive normal orientation from the influence of another sensory system, as was done in vision. Because of these and other similarities and differences between vision and touch, the orientation issue needs a more careful examination and a new, much broader approach. The following points will therefore be addressed below: origin of spatial inversion, mechanism by which it appears, theories on orientation, and general principles involved in our interpretation.

26.2.1. Origin of spatial inversion

The origin of spatial inversion must be sought in the layout of the tactile field in the brain, more specifically, in the projection cortex (marginal zone). Not only is there a transposition of sides due to decussation of the sensitive pathways, but there is also a vertically inverted arrangement in the cortical projection (see schematic in Fig. 26.5). The same is known to occur in vision, although the inversion of the visual field is considered to result from the optical system of the eyeball, and the inverted orientation that already exists in the ocular retina is projected anatomically point by point onto the calcarine cortex or "cerebral retina" of Henschen (1926). Thus, the chiasm intervenes only to achieve binocular correspondence, i.e., to produce the cyclopean eye. Therefore, cortical sensory projection areas show a contralateral and vertically inverted arrangement with respect to their sensory fields (visual, tactile,

[2] The issue of spatial inversion is addressed and related to the secondary areas in Secs. 4 and 5 in: GONZALO, J. (1952). *Trabajos del Instituto Cajal de Investaciones Biológicas,* XLIV: 95-157. (Supplement I of the present edition.)

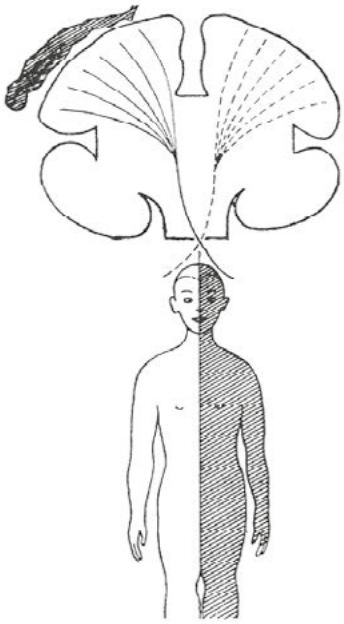

Figure 26.5. Diagram to recall the brain projection of the somatic sensitivity (tactile field) contralateral and vertically inverted for each half.

auditory). Hence, there is a spatial congruence on the brain surface between the different sensory systems; for example, tactile and visual stimuli from the lower right are projected into the upper and left brain.

Thus, in the interpretation of inverted perception in vision we have first considered the local effect of retinal inversion, being equivalent to refer to ocular retina or to cerebral retina for such spatial effects, as was said. But now, having discovered inverted perception in other senses, touch and hearing, the question of inversion is focused on the projection onto the cortex. Thus, it is curious that the old and much discussed issue of ocular image inversion (see Sec. 12.1 in Vol. 1) has led, through the study of our brain-injured patients, to a cerebral effect encompassing all sensory systems (inversion occurs in vision as well as in the other spatial senses). Inversion has therefore ceased to be merely ocular and exclusive to vision, becoming a strictly cerebral effect of a general nature. From the anatomical point of view, the starting point is the ocular crystalline lens that produces the optical inversion, transferred in the same way to its cortical projection. According to the theory of Ramón y Cajal (1898) on nerve crossings, the decussation and inversion of the other sensory pathways achieves a congruent spatial projection (of the same sign) in the brain.

As for the remarkable phenomenon of fragmentation of the body into three autonomous zones (head, trunk-upper limbs and pelvis-lower limbs) showing independent rotations in inverted perception, this phenomenon probably has a physiological basis in the special somatotopic projection of the sensitive cortical area, ac-

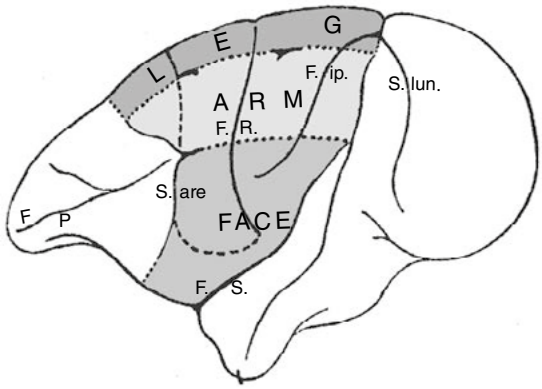

Figure 26.6. Sensory cortex in the convexity of the hemisphere of the macaque monkey, as revealed by the method of local strychnization of the cortex (Dusser de Barenne 1924). Note the three independent areas (face, arm and leg), clearly delimited by dotted lines. The dashed lines indicate the frontal boundary of the electrically excitable cortex.

cording to the cortical strychnization experiments of Dusser de Barenne (1924). As described by the author himself, "these experiments reveal a well-defined subdivision of the sensitive cortex into three sectors: area of the face, area of the trunk and arm, and area of the leg (Fig. 26.6). The boundaries between these areas are precise and respected even by strychnine, which generally causes a diffuse discharge when introduced into the central nervous system. Strychnine has never been observed to spread from the leg to the arm or to other subdivisions when applied locally to the postcentral circumvolution."

Leaving aside for the moment the analysis of these morphological issues, we simply emphasize that the acceptance in our theory of the effect of projection pathways, is an important concession to the anatomical aspect of the functions. This stance cannot surprise the attentive reader of this work, since already at the beginning of it (Sec. 1.2.3 in Vol. 1) the syndrome of cortical projection zones (which we have called marginal syndrome) was accepted as an extreme case fully characteristic of brain localization since it affects the conduction of the long pathways. However, such acceptance of brain anatomy does not imply that we are going to develop a situation of compromise with the brain localization theory whose statements we should reject. Rather, we merely state that along with mass action (excitability and organization) the anatomical nerve texture must be taken in account, especially for the spatial structure. This anatomical factor has often seemed to play a minor role in our conception, but in certain issues such as the present one, it acquires a major significance. Thus, the pathophysiological question of spatial orientation (and of space in general) can have an anatomical-physiological basis.

In this sense, it is convenient to indicate the role that the anatomical-physiological complex plays in the orientation function, according to the possible cases (or syndromes). Let us consider the following cases in gradation: normal subject (localization sensation congruent with stimulus), central syndrome (sensation preserved

but inverted localization), and projection or marginal syndrome (suppression of sensation contralateral to the lesion). In a normal subject, the anatomical factor of contralateral inversion is present in a latent form, being supplanted by the physiological action of the central mass, which, by reorganizing the field, excludes any spatial discordance between stimulus and sensation. Instead, in the central syndrome, which arises from a brain or hemisphere with reduced activity, the marginal (projection area) effect tends to emerge, and the greater the brain deficit, the more evident this effect becomes. Thus, with a great loss of central nervous mass (great asynchrony as we have seen), the primitive action of the anatomical factor of the marginal projection becomes evident. In this case, simple sensation or elementary excitability is possible to some extent for the corresponding "sensitive field," but its spatial localization undergoes a change, being guided by the contralateral and inverted orientation of the marginal arrangement (in the projection area). That is, the orientation of the "spatial field" tends to reflect the orientation of the projection area. There is thus a functional incongruence between the sensitive field and the spatial field. Finally, if the brain lesion reaches the projection (marginal) area, the ordinary projection syndrome (which we call marginal) appears, in which the absolute sensory defect remains contralateral to the lesion according to the conduction pathways on the projection area. As is well known, in a marginal (projection) lesion, defects are produced on the contralateral side and inverted vertically; thus, a lesion in the left superior calcarine causes blindness in the right and inferior quadrants.

In view of the above, we see how the anatomical texture of the marginal projection can give rise, depending on the circumstances, to two manifestations which, despite their external dissimilarity, have a certain internal unity. That is to say, in one case we have the classic projection (or marginal) syndrome with totally contralateral and inverted defect due to marginal "passivity," and in the other case we have the inverted perception of orientation in the central syndrome, as a result of marginal "activity." We therefore see that two brain theories as antagonistic as the classical one of anatomical localizations in the brain and the one of brain dynamics presented here, find for once a common nexus in basing the issue of orientation on the anatomical-physiological complex.

Returning to the configuration of the projection area, it should be noted that in order to fully explain the tactile inversion in patient M, each body half should be located in the totally opposite cortical area, i.e., in addition to lateral and vertical inversions, there should also be inversion between front and back (e.g. the anterior part of the body represented backwards in the cortex).

26.2.2. Inversion mechanism

Here we analyze the conditions for each of the two extreme orientations, the normal one and the inverted one, and how the transit between them occurs. For this, both the brain mechanism and the symptomatic changes in the sensory field must be considered.

As already indicated above, inversion is produced by the action of the marginal brain disconnected from the influence that the central area must normally exert. Such an inverted orientation genetically corresponds to a primary (nativist) orienta-

tion that could also be called autocentric or idiocentric, i.e., an orientation that arises directly from the marginal structure, being independent and prior to any external experience. On the contrary, the joint action of the whole brain (central action), correcting the marginal action, gives rise to an allocentric orientation, normal and congruent with the stimulus, that is, adapted to the experience by virtue of the reorganization of the sensory field.

As for the brain mechanism of inversion, the experiments described above show that there is an asynchrony of excitability between the two orientation systems, marginal and central, resulting in an abnormal sensory interval between the two extreme orientations. According to the rule of dynamic reduction, inversion appears with weak stimulation because it is a more elementary function, whereas the correct orientation corresponds to strong stimulation. Starting from the marginal action, with inverted orientation, the activity of the central area can be achieved by increasing the stimulation. In this way there is a recruitment of brain mass that corrects the orientation following Fechner's law. This recruitment occurs through different balance states between marginal and central action.

Parallel to this recruitment, the phenomenology of the sensory field evolves, and not only in orientation but in the whole organization, as indicated by studying the process of spiral reduction in Fig. 26.4. This parallelism or correspondence between the state of the brain and the state of the sensory field is illustrated by means of the diagram in Fig. 26.7. It shows that a residual field corresponds to a brain functional-

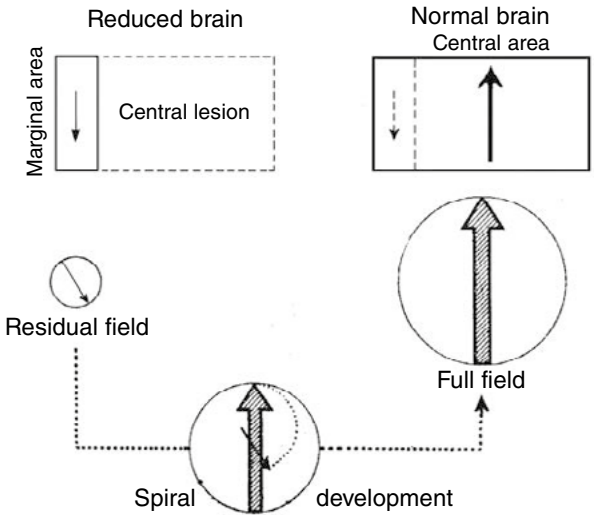

Figure 26.7. Diagram to illustrate the brain mechanism of orientation and the corresponding sensory field (both visual and tactile). A very small *residual field* with inverted orientation, as in the marginal area, corresponds to a brain reduced to the marginal area by a central lesion. An *entire (full) field* of maximal size and functional reorganization such that orientation is congruent with the stimulus, corresponds to a normal brain, i.e., one with a larger brain mass. The transition from one brain state to the other by recruitment of brain mass is reflected in the spiral development of the sensory field (field enlargement and re-inversion).

ly reduced by a central lesion. This field is considerably reduced, has an inverted orientation analogous to the marginal projection of the field and other pathological features that we shall see. Reciprocally, an entire or full field corresponds to a normal brain (with active central zone). Such a field, with full integrity of the sensory functions, is a larger field because it responds to a greater activity of brain mass than in the previous case, and has a normal orientation congruent with the stimulus, all due to the sensory reorganization determined by the central mass. The transition from one extreme state to the other by recruitment of brain mass gives rise to the aforementioned spiral development of the sensory field by the joint effect of progressive re-inversion and enlargement. The *neurosensory correspondence* (between brain organization and sensory field organization) is therefore evident with respect to both orientation and size. Such characteristics of the field and the brain are valid for both vision and touch since orientation phenomena and organization are similar in both.

In trying to understand the phenomenon of re-inversion (normalization of orientation) new questions arise. As for vision, we admitted that tactile or haptic support is involved in the formation of normal orientation, thus establishing the visual-tactile complex for normal (correct) orientation. But now, in the case of touch, where does the support come from in order to establish an orientation congruent with the stimulus? It is clear that it is not possible to resort to intersensory supports when all systems suffer from the same inversion disorder. Besides, it would seem inadmissible in a sense as fundamental and primary as touch, so intimately linked to the corporeality and orientation of the own body. Therefore, the explanation in the case of touch must be sought in the tactile system itself, i.e., necessarily in its state of organization. Thus, the orientation problem is simplified and unified.

The question raised of how touch re-inverts its orientation by itself can be solved by analyzing the characteristics of the sensory field. Thus, we see that the characteristics of the field for the correct orientation and for the inverted one are very different. The inverted field has a residual organization, it is an extremely reduced field, presenting an inverted local sign, and on the verge of losing all structure. In contrast, the normal field, by virtue of its reorganization, has the opposite properties, such as large size, cohesion, orientation congruent with a stimulus, etc. Thus, the sensory field corresponding to inversion does not resemble the normal field at all, but would rather resemble phenomena of instability and incoherence of a pseudo-hallucination. Within all these characteristics, the essential difference could be found in the respective continuity of the field. In the normal case, the field, of large size and cohesion, can form a total and unitary continuity in such a way that spatial references related to each other are established, and then there must necessarily be spatial congruence between a stimulus and the corresponding sensation. A different situation is that of a residual field which, restricted to a reduced sector (the periphery, extremities, etc., are excluded), without internal cohesion (recall the independent rotations in tactile inversion), lacks the necessary amplitude to establish continuity between spatial references and to form stable and congruent links with the stimulus. This field being thus disconnected from all support and abandoned to itself, tends to show its primordial marginal orientation (inverted). That is to say, if the field is complete and continuous it must show spatial congruence; but if it is very reduced,

there is no internal continuity and it has an idiocentric orientation, being incongruent with the outside. In short, orientation depends on the continuity of the organization.

26.2.3. Theories on orientation

As a complement to the above considerations, it is worthwhile to make a quick review of the theories on orientation and localization in touch, mentioning also the problem of the orientation of the visual image. According to the above, the primary factor in spatial determination (localization and orientation) lies in the cohesion and functional integrity of the sensory field, and not in diverse associative mechanisms, intersensory or not. This means a clear opposition to the empiricist conceptions of space in general, whose critique was exposed in Sec. 22.4. Such empiricist and associationist conceptions had a great impact on the issue of visual image orientation, first theoretically and later experimentally (Helmholtz 1867/1896), especially after the famous experiment of Stratton (1896, 1897) (see Sec. 12.1 in Vol. 1). It is clear that the pathological inversion (spiral reduction) is of a primordial (nativistic) nature, totally independent of experience and without influence from other senses. In the experiments of the cited authors, especially in the one by Stratton, new values in visual orientation could be obtained due to the fact that the sensory field being intact, its continuity gave rise to a reorganization adapted to the configuration artificially promoted in the experiment. For this reason, the Stratton experiment (re-inversion by habituation to the artificially inverted visual scene with lenses) was not possible in the case of our patients, with residual field, since there was already difficulty in adapting to the conditions of ordinary vision.

According to the ideas indicated in the preceding subsection on orientation as a function of field organization, the interpretation that can then be given to the experiment of Stratton (1896, 1897) does not refer to the formation of new associations between vision and touch (visual-haptic complex), but only to the sensory reorganization provided by the central mass of a healthy brain. Such a mass would allow adaptation to the artificial inversion given by the lenses, as normally occurs for the natural inversion of the latent marginal (inverted) action. Thus, the process would be entirely visual without any other sensory aid. Already Müller (1903, 1923) is in favor of this visual independence to change the organization in Stratton's experiment, discarding the primary necessity of visual association with touch. All this means that also for the problem of visual orientation, a mechanism similar to the one described for touch should be admitted, granting a large autonomy to the visual field in the formation by its own of spatial orientation. The visual-haptic complex would then be reduced to a totally accessory role, if its acceptance is in any way justified. A more detailed examination of similar problems, especially concerning visual orientation - which is somewhat out of place here - is reserved for another work.

26.2.4. General principles in our interpretation

Finally, given the theoretical importance of the topics addressed, it seems appropriate to add some indications on the general principles involved in our interpretation. The

different phenomena of the brain dynamics presented here have been interpreted from a strictly physiological point of view based on the excitability factor, which in turn depends on the damaged central nervous mass. In this respect, the orientation disorder followed a behavior analogous to that of other functions. Even inverted vision seemed to be explained by the particular effect of ocular inversion, without having to resort to new fundamentals. But the discovery of inverted perception in other senses, and the attribution of such a phenomenon to marginal action (action of the projection area), has brought the anatomical factor (configuration of the nervous matter) into the picture. Thus, the orientation function, due to its peculiarity, is not like many other altered functions, but is of fundamental importance because it plays a decisive role in the second law (on spiral development) of the brain dynamics described here.

The orientation function reveals by itself the nature of brain activity, composed of both the anatomical factor and the physiological factor (nervous excitability in relation to brain mass). Indeed, no other function could have evidenced the anatomical factor, since in the spatial functions all we find is the change of size: reduced or enlarged space, depending on the activity of the brain mass. In fact, the anatomical arrangement is manifested in a very characteristic way in the alteration of the orientation function, precisely because the anatomical arrangement is opposite to that of the physiological organization. In short, for the complete interpretation of the phenomena of sensory organization it is necessary to resort to the physiological principle of mass action together with the principle of anatomical configuration (anatomical connections), each of which corresponds respectively, in a broad sense, to the functional principle and the principle of localization.

The aforementioned principles, which are indispensable for understanding the different functions of the nervous system, intervene in different ways in the system. Thus, the anatomical principle of connections is particularly relevant in all phylogenetically older structures (spinal cord and subcortical formations in general), with a more fixed function, although without dispensing with the functional principle (remember that the isolated spinal cord becomes iterative). However, these structures have an extremely restricted activity at the cortical level, especially centrally, and such activity only has an effect in the marginal (projection) area, as indicated when describing the various brain syndromes in relation to orientation, etc. In the central cortical mass, the anatomical connections do not play a role comparable to that of other nervous structures, so it is possible to consider this central area as an undifferentiated (non-specific), rather plastic area, whose function is to enhance excitability and reorganize activity. This is seen both in the re-inversion that must normally occur to correct the anatomical marginal inversion and in the adaptation evidenced by the experiment of Stratton (1896, 1897) showing the maneuverability of the central mass, regardless of whether a visual-tactile association or our interpretation is accepted.

According to such considerations, it can be said that, whereas inverted orientation corresponds rather to a *static* spatial localization, re-inversion (correct localization), congruent with the stimulus, corresponds to a *dynamic* localization by means of the central effect of reorganization of the field, either by normal synchronism in a healthy individual or by recruitment in patients with brain lesions. We can then

characterize the central action as a maneuvering mass in contrast to the primitive and anatomically fixed function of the marginal (projection) area.

Inverted orientation corresponds to a primordial orientation, which is in favor of nativism, as already said, whereas correct orientation would be in favor of empiricism, even more so in the case of re-inversion in the Stratton experiment. This antinomy is resolved by going beyond the nativism-empiricism dispute; and then, what is evident in all cases is the *correspondence* between brain organization and the structure of the sensory field.

Such a correspondence, already pointed out in dealing with the mechanism of orientation (see Fig. 26.7), brings our study on orientation and sensory organization to the plane of natural philosophy with the issue of psychophysical correspondence. We are facing the very remarkable result that sensory organization and brain organization are identical or superimposable: the inversion of the residual field copies the marginal organization (reduced and inverted), whereas the normal field with correct orientation is supported by a larger brain mass with possibility of physiological reorganization. Such a type of correspondence refers to a psycho-physical isomorphism. This isomorphism must be understood as equivalence of spatial structure between the sensory field and the brain action. This action constitutes a dynamic field influenced by the anatomical configuration and the maneuvering of the central mass. Then, the law of sensory organization referring to the spiral development of the sensory field (see Sec. 26.1.3) constitutes a law of psychophysical organization, which can be stated more causally and explicitly as "spiral development of the sensory field due to a psycho-physical isomorphism in the cerebral recruitment."

Further discussion of all these theoretical issues will be found in a future work. For the moment, the issue of orientation has been sufficiently examined in its various consequences so as to see that it occupies a privileged place at the basis of the brain dynamics described here.

Tactile schema

27. Body schema

27.1. Degrees of body schema

The theory of the body schema results rather confusing, regardless of the usefulness of this concept, since phenomena of very different types are mixed indistinctly. Indeed, this concept refers both to the phantom limb syndrome of amputees and to the inverse syndrome such as asomatognosia and autotopagnosia (disorientation of the own body in many different forms). The concept of body schema also refers to localization defects of the allochiria type and, above all, to the postural disorder (disorder in the recognition of body attitudes); finally, it is deeply related to the complex and varied apraxic and apractognosic syndromes.

The body schema concept was first formulated by Head and Holmes (1911), supported by the rich clinical material of Pick (1908 a, 1908 b, 1915, 1922), developed by Schilder (1923/1935), who introduced the term body image, and by many other authors such as Anton (1893), Hartmann and Schilder (1927), Pötzl (1924), Pineas (1926), Stockert (1934), not always coinciding in the delimitation of the concept and much less in the nature and generation mechanism of such a function. According to Head and Holmes (1911), the body schema is an essential function for determining the site of skin excitation on any part of the body, and especially for recognizing its position. But later, such function has been widely extended both to the spatial activity of the own body and to its connection with the external space. It has been assumed that this function is due to many different factors, such as cutaneous and also deep tactile sensitivities, visual influence, vestibular factors, motor factors (muscle tone), sensomotility, etc., all of them integrated in a superior complex of spatial structure. On the other hand, its nature has often been overloaded with psychological intellectualisms (representations, comparisons, judgments, etc.), as is usual in gnosic type functions.

For our part, on the basis of the phenomena observed in our patients, providing new data (which systematize the body schema process), as well as on the assumption that there are no functional individualities per se but continuity of organization

and gradual development, we distinguish several degree*s* in the body schema, the latter consisting essentially in the spatial tactile organization. This organization is based on the progressive differentiation of the tactile field without the need to resort to other elements of an indeterminate nature, so there is perfect continuity between the diverse structures of the tactile sensory field. Without clamming a rigid delimitation of functional stages of the body schema, since the different activities are often intermingled, we will be guided by the following classification according to three degrees of increasing complexity in the spatial organization: corporeality or *somatic model*, *postural model,* and spatial schema or *praxis model.*

The most rudimentary degree is corporeality, i.e., *somatic model,* which only expresses how much it is related to the size and conformation of the body (soma), which implies a certain sensation of body orientation and also praxic or voluntary innervation. It has nothing to do with attitudes or kinetic form of the body, the definition being restricted to the mere static form (simple conformation of the body in its most stable aspect). The *postural model* refers to the perception of the various attitudes or postures of the body, and the localization of these postures in space. It is a more developed and subsequent degree to the simple corporeality. Finally, the spatial schema or *praxis model* mainly represents the functional structure (spatial action plan) where both the orientation about the own body and the orientation with respect to the exterior are articulated.

In a normal individual, these three main modalities (levels) are fully fused in the same activity, but in brain-injured patients, these levels become rather independent due to asynchrony, allowing a dissection of the structure of the body schema. In this situation, there is a pathological reduction of the body schema that tends to exclude the more developed stages, that is to say, it tends to convert them into preceding, simpler stages. The process of progressive reduction is then as follows: first, the structure of the spatial schema is simplified showing a reduction of the praxic behavior; next, the postural model shows more and more a character of simple corporeality by reduction to the most stable or habitual activity; subsequently, corporeality is reduced in size and conformation to finally vanish completely. A reverse process can be obtained to some extent by recruitment of the asynchronous levels, either by intensification of the stimulus (activation of the schema "from outside") for corporeality and posture stages, or by the much more effective effect of facilitation by muscular effort (activation of the schema "from within") for all stages of the body schema.

27.2. SOMATIC MODEL

In the study of the simplest stage of the body schema we must pay attention to the following aspects that arise in the analysis of our cases. First, the somatic development (development of corporeality) according to the size and conformation resulting from the experiments already discussed on tactile space (Secs. 21-23). Second, the important aspect on the capability for elementary voluntary movements (praxic impulse). Third, the very special feature of facilitation, which activates the model. Finally, we shall give a brief theoretical summary.

27.2.1. Somatic development

In the previous chapters on tactile space and orientation we have often had to refer to the body schema in its most rudimentary manifestations of simple corporeality, this one being involved in any spatial tactile activity however simple it may be. The *somatic development* corresponding to the different phases of localization of a cutaneous stimulus is described in Sec. 21.2, also valid for deep sensitivity.

Looking at the stages of localization of a cutaneous stimulus, we see that, depending on the energy of the stimulus, a certain degree of body schema emerges, which means an activation of the soma "from the outside." The degree to which it emerges, according to *size* and *conformation,* is shown in Fig. 21.14. Given that even in the most normal-like phase, there is a certain proximal deviation of tactile stimuli, especially for stimuli in the most distal areas of the extremities, there is a certain concentric reduction of the somatic model, i.e., a more or less reduced model according to the development of phase IV in spatial localization. A greater reduction in size and conformation presents stage III, with a shape corresponding to an "embryonic model," losing also the posterior plane and other features. Finally, in phase II the simplification is so accentuated that at most a "filiform model" is produced (Fig. 21.3) in which all more or less defined corporeality (tactile spatial field) is close to disappearing. However, even in phase I, an extremely residual activity with no possible spatial localization persists in the aroused sensation; it is a certain influx of the body schema that determines the elongated shape of the irradiation (Fig. 21.10).

Likewise, several phases of corporeality are aroused with deep (joint) stimulation (activation "from within"), either in tests with joint segments (Sec. 23.4), or during initiation of walking (Sec. 25.1.4), where the size and coherence of the model grow as gait progresses. When starting to walk, as there is no corporeality or model to support the steps, there is only fusion of movements, that is, indistinct sensation of phase I or phase II, which, in addition, shows the lack of distinction between cutaneous and deep (articular) sensitivities, due to the low level of organization. Afterwards, it is already possible to distinguish movements, and finally steps and sensation of translation, due to the recruitment of the model and the emergence of spatial references. Such recruitment (somatic growth) as a function of stimulation can be measured and illustrated graphically, as shown, for example, in the curves of recruitment of localization (Fig. 22.3) and recruitment of orientation (Fig. 24.6) for a stimulus on a hand. In the case of walking, the subjective direction of the steps varies according to their perceived length and the size of the model (Sec. 25.1). Fig. 25.4 shows some recruitment curves in the case of walking. It can be noted in such examples that the recruitment of the somatic model, approximately of logarithmic type, is simply a recruitment of the tactile field since the proximal deviation (spatial shrinkage) is corrected. In all these cases of cutaneous or articular activation of the model, its emergence tends to be partial or fragmentary, as we already know, and the situation cannot be compared to the situation of a normal subject. Indeed, a major disturbance is the lack of continuity and cohesion of the model, whose parts are as if loose (disconnected). Other factors such as abnormal lightness of the body,

flexibility, etc., contribute to the sensation of abnormality. All this has been indicated when dealing with corporeality in a general way (Sec. 23.5).

Another mode of somatic emergence to be considered corresponds to the state of natural passivity, without any particular movement nor cutaneous stimulus. When a normal subject is in this state, a diffuse sensation of body schema is produced in response to low-intensity cutaneous and articular tactile excitations. In the brain-injured patients, mainly in subject M because of his excitability deficit, any sensation of corporeality is then hardly awakened, arising only under a more intense general excitation, either directly or indirectly through facilitation by muscular effort. As for patient M, the degree of corporeality is thus related to the degree of general tactile activity. For example, when he is sitting comfortably, relaxed and completely inactive, he does not perceive any corporeal sensation, unlike the normal subject. Instead, when he is standing, he can feel a diffuse corporeality that, at most, would reach up to his knees. He does not feel the head and arms at all, the body being reduced to an indistinct sensation of the trunk and the proximal sector of the lower limbs. This is a very faint corporeality and of a size that would be at most half that of normal. This sensation does not improve no matter how long the patient stands. A fuller emergence of the somatic model can only be achieved by increased tactile activity, such as during walking, or by vigorously applying facilitation through muscular tension.

27.2.2. Praxic impulse

It should be noted that the state of absolute inactivity in patient M, seated and relaxed, is difficult to achieve in most examinations, since when he is asked or given instructions, he automatically exerts, without being able to avoid it, a certain facilitation by muscular effort. It must therefore be borne in mind that there is almost always a minimum of facilitation. However, by conveniently instructing the patient about the conditions of the experiment, he can maintain the state of complete passivity and relaxation during certain tests, which entails total absence of any sensation of corporeality, as said.

An expression of the degree of corporeality is certainly the capacity to voluntarily make use of one's own body, of the *praxis innervation,* manifested in the aptitude to voluntarily move or activate any part of the body. When a state of absolute inactivity is achieved in patient M, a test of relevant importance can be performed which allows us to understand the significance of the somatic model in relation to the praxic impulse (initiation of elemental voluntary movement). We thus arrive at the remarkable and unsuspected finding that when subject M is absolutely inactive he is completely unable to perform the most insignificant voluntary movement, no matter how little energy is required, such as opening his eyes. This is verified with absolute regularity in repeated trials. This shows that if the model is absent, it is not possible to promote any voluntary action no matter how elementary it may be, resulting in akinesia or rather total apraxia, due to the absence of the most elementary orientation about his own body. Faced with such a result, it seems at first sight that the possibility of freely applying facilitation by means of muscular effort to activate the

model is in contradiction with the above. However, the incoherence disappears when we consider that this type of facilitation is exercised in a diffuse way and almost as a reflex, very involuntarily, like any other automatic action of ordinary life, as will be seen.

In examining praxis in relation to the emergence of the somatic model, we first find an abolition of the praxic impulse when all corporeality is absent. This is in itself a datum of great importance. It is necessary to go a little further, to the initial limit of activation of the model under minimal facilitation, in order to "catch" the emergence of the praxic impulse. The following tests allow us to assess this process.

Patient M being in the inactive state with eyes closed is asked to open them upon hearing an acoustic signal (loud enough to be noticed even in the inactive state). The patient is allowed to use some facilitation by means of minimal muscular effort. In this situation it takes him four or five seconds to open his eyes, even though he thinks he does it instantly! But if, when receiving the signal, he is under the sustained action of the facilitation by maximum muscular effort (very intense and permanent contraction of the whole musculature), he executes the order quickly, in tenths of a second, like a normal subject. However, it is remarkable that he claims that he was equally fast in both cases (quasi-inactive and with intense facilitation), and he states this emphatically in several trials.

This latency time to perceive and activate the body varies according to the muscular effort made. However, the different objective times thus achieved, corresponding to different levels of nervous activity, are considered to be the same by the subject. Thus, when he is allowed to make a weak effort, the latency time can drop to about three seconds, although in tests performed on a different day he has taken up to nine seconds, maximum value reached in such conditions. This should be attributed to the different degree of muscular effort applied during the test rather than to a different degree of attention. Nevertheless, in this test, the change in the subject's attention is equivalent to the change in excitability due to muscular effort. When he takes nine seconds to respond, he still believes that he has executed the order quickly, as in three seconds, or in tenths of a second under maximum facilitation by strong muscular effort. He is very surprised and complains when he is told that he has needed quite a long time. Finally, when he is very inactive, he may spend a minute without opening his eyes even though he does not stop trying to comply the order. In this case, it does not seem to him that a long time has elapsed either, only about two seconds in comparison with the test under maximum facilitation.

Regarding the process of following the order, it is observed that a little before opening the eyes, slight movements in the upper eyelids may occur, as frustrated opening movements. For this, it is necessary that, upon receiving the acoustic signal, the subject applies a certain facilitation by muscular effort in the form of a reflex that involves an activation of the trunk and, weakly, of the head. At this point it is then possible to initiate movement of the eyelids. The limbs are not felt at all, i.e., the model is activated somewhat similarly to when the subject is standing, but with more tendency to include the head and lose the limbs.

Other examples of this type of voluntary movements in patient M are the following. Opening the mouth using the facilitation effect as little as possible. The pa-

tient takes three or four seconds to do this although he thinks he does it instantly. Another example is moving a finger under the same conditions. The patient takes 3.5 seconds, showing shortly before slight movements (the referred innervations or frustrated movements) in some other finger of the hand or in the same one that subsequently moves. The same behavior is observed for the head movement; he also takes 3.5 seconds. Larger movements such as moving a whole limb obviously lengthen the latency time, reaching seven to nine seconds. For example, lifting an arm takes six seconds, and lifting a leg and foot takes seven seconds. He also feels now that he executes the order very quickly, although he admits that perhaps he executes it less quickly than in the case of opening the eyes or other simple movements with half the latency time. When in these tests he is asked to move a very distal part, a finger, a foot, etc., the model must be activated at least up to the middle of the limb (up to the elbow or knee), and the somatic emergence may be regionally diverse. Thus, the distal area of a limb may be activated considerably while the head or other limbs are activated much less. This means that facilitation by muscular effort is directed, albeit diffusely, towards the region to be moved, as we will see later on.

It should be noted that such elementary voluntary movements may be subjectively ignored, given the weakness of both the facilitation applied (by muscular effort) and the movement performed, as well as suffering various subjective alterations of inversion, localization, etc.

Summarizing the development of the praxic impulse, i.e., of voluntary movements of elementary character, it can be said that such movements are closely related to the degree of corporeality. Particularly in the initial stage of the somatic model, the praxic impulse shows the typical features of a low functional level. These are: noticeable slowness of the process (to search for the model, to transmit the movement, etc.), movements initially frustrated by disintegration of the process, and even reduction of the subjective time of the process as it corresponds to a brain state of reduced excitability. In short, depending on the degree of corporeality, the praxic impulse can be either abolished (absence of the somatic model in a state of total inactivity), or emerge slowly and inarticulately (initiation of the somatic model under weak facilitation), or be practically the same as in a normal subject (somatic model fully activated by maximal facilitation).

27.2.3. How facilitation by muscular effort is exerted and its effects

The phenomenon of facilitation by muscular effort shows important aspects in tactile functions. In relation to our study on corporeality, we must examine how such facilitation is performed and how the body schema is activated.

In view of the abnormal manifestations in the different sensory systems, it seems evident that the phenomenon of facilitation by muscular effort has spontaneously originated in tactile activity. It is clear that daily movements, both manual and of the body, already normally require diffuse muscular tension. This leads not only to improve proprioceptive function due to increased stimulation of the receptors involved, but also to improve any tactile function by central nervous summation effect. In this sense, since patient M in the inactive state has extremely impaired

spatial tactile functions, these must always have been accompanied by muscular tensions of considerable intensity (tensions, contractions, movements, etc.). These tensions, although involuntary and unnoticed by the subject, are absolutely indispensable for his daily life. Even in patient T and in other brain-injured patients with less brain deficit, some degree of facilitation by muscular effort is necessary to activate tactile space (localization, deep sensitivity, etc.). This facilitation usually consists of diffuse muscular tension or muscular jerks or twitches (see Sec. 21.1), being also spontaneous and of unknown significance to the patient. This explains the great functional difference shown, for example, in joint sensitivity: very disturbed when examined in an inactive state by passive tests, and instead quite functional in spontaneous voluntary activity. It is then understandable that in the long run, a certain degree of facilitation by muscular effort is automatically associated with all types of tactile activity, thus considerably improving such a fundamental sense for the organism as touch.

Facilitation by muscular effort has originated mainly in the tactile system, and has been extended by applying it to the other sensory systems since they normally work together. However, facilitation by muscular effort is much more necessary for touch than for other senses such as vision or hearing. The latter are already partially compensated in their brain deficit in a simple and natural way by the summation effect provided by the pair of organs, and are not as impaired in their activity as might otherwise be expected. It can then be said that patient M and patient T automatically exert some degree of muscular facilitation to any type of sensory activity, having developed an unconscious habit over time after suffering the brain injury. This would also apply to the Schneider case of Goldstein and Gelb (1918, 1919). However, the subjects being unaware of the application of this type of facilitation and even more of its nervous summation effect, they were unable to take advantage of its voluntary application and its significant maximum effect, until this phenomenon was discovered. As already mentioned (Sec. 1.3.2 in Vol. 1), the phenomenon of facilitation by muscular effort was discovered in 1939 after persevering research. Since then, especially patient M benefited extraordinarily from the voluntary application of facilitation by means of maximal contraction of the entire musculature. Thus, his sensory horizons were instantly broadened in all kinds of activities, achieving a very considerable correction of the defect.

The application of the facilitating muscular effort occurs, as already indicated, almost as a reflex as soon as some sensory activity arises, therefore the state of inactivity in the tests is difficult to achieve, and a proper instruction of the subject is required to achieve complete relaxation and passivity. It is at this point when the useful time of subject M in the different tests of sensory excitability with adequate stimulus achieves up to about seven seconds.

The loss of the somatic model, absence of corporeality, does not seem to be an impediment to exert muscular tension since, as observed and as the patient states, he performs a diffuse muscular tension which, since it is not localized, does not need to be guided by the somatic model. This is a very different situation from that of elementary movements associated with praxis, where the movement is specifically chosen. In conclusion, the application of facilitation by muscular effort, whether involun-

tary or conscious, arises as an undifferentiated action impulse that provokes a diffuse muscular tension of the body without the need to be guided by the somatic model.

It should be noted that although the execution of facilitation by muscular effort is rather diffuse, the site of origin is in the trunk musculature, as evidenced by observation of the patient. There are several reasons for this preference. First, the trunk is the central area of the body, and as such, acts as a support for all haptic activity. Second, it is a very undifferentiated region in terms of touch and therefore lends itself to diffuse activities. Finally, it is more resistant to dynamic reduction in the fading of the body schema, since, being a central region, its survival is possible on many occasions. It is also plausible that the area of spontaneous thoracic breathing movements is an attraction to more or less automatically exert facilitation by muscular effort. Indeed, it is very common that when subject M voluntarily applies facilitation, he starts mainly by making an effort with the breathing musculature (thoracic muscles and abdominal pressure). It is possible to perceive his suffocation produced in such a situation, especially when asked to apply a strong muscular effort of maximum effect. These conditions occur especially when the subject is seated and totally inactive. When he is standing and needs muscular effort to increase any sensory activity, he tenses strongly the musculature of the legs and thighs, since when standing, this region emerges sufficiently in the body schema, and thus hides the muscular tension from the sight of other people. The patient acts in this way from the time he is aware of the effect of this type of facilitation.

Another issue to be studied is the type of "corporal activation" according to the amount of facilitating muscular effort. Within the trunk there may be muscular contractions and tensions of varying intensity which, at best, only cause a partial activation of the somatic model. Thus, in maximum muscular contraction of the trunk due to strong tension of the thoracic and abdominal musculature, the model emerges only up to approximately the knees and elbows, although the head is also usually activated by the tension of the neck. This type of facilitation is the one usually used by patient M when he is asked to apply it in tests, and the one he also usually applies in his daily life if he has to overcome difficulties in perception, comprehension, etc. But it is possible to achieve a greater effect by adding a strong tension of the limb musculature, thus obtaining the maximum limit of muscular effort.

It is very remarkable the fact that in order to clench the fist strongly, he must begin by contracting the trunk, and next the whole corresponding upper limb. It seems that it is absolutely necessary to maintain the same muscular tension in the trunk and limb as in the fist. Thus, if he is asked to continue clenching his fist tightly but relaxing the trunk, he is completely unable to do so, and by relaxing the trunk the hand loses all its strength. It seems natural that the trunk can be innervated (activated) without the limb, but the reciprocal seems impossible.

As has been said, no matter how considerable the muscular tension of the trunk (central area of the body) is, the activation of the somatic model does not reach the more distal areas of the limbs, and it is necessary to distribute the muscular tension more peripherally to make them emerge in the model. Various tests on how to activate (to perceive) the hand show the need to contract the entire musculature of the limb, with the help of the trunk, and also to clench the fist strongly. Even so, it seems

that the distal half of the last phalanx may be excluded, according to tests with tactile stimuli at the fingertip that are perceived with some proximal deviation. If instead of closing the fist, the open hand is tensed, the activation is less complete and only reaches up to half of the fingers. In the feet, by making an intense contraction of the toes, the activation of the model is likely to reach the beginning of the toes. This emergence of the somatic model is the same as the one reached iteratively when walking, after many steps (see Sec. 25.1.4).

Other consequences of facilitation by muscular effort will be discussed when dealing with praxia, manipulations, etc.

27.2.4. Theoretical summary

Making a theoretical summary on the somatic model (corporeality), we can say that through asynchrony and therefore dynamic reduction, the process of emergence of the model directly shows the level of organization of the somatic field (tactile field).

Somatic emergence is determined by its size and, in part, by its configuration which is linked to the postural model, as will be seen further on. Such emergence is restricted to the central zones of the body in the most rudimentary phases, and subsequently expands following a process that is entirely identical to the recruitment of the spatial tactile field. Thus, reduction of the somatic model is nothing more than a concentric reduction of the tactile field. The localization of stimuli, either cutaneous or articular, is therefore the most direct expression of the development of the model, as pointed out by Head and Holmes (1911) when dealing with the body schema. As for the shape of the model, a certain general shape tends to be preserved at all stages of model development, even in the irradiation band without any possible localization.

Both cutaneous and deep (joint) sensitivities contribute to the formation of somatic corporeality, however these sensitivities are only distinguished as such if there is a certain degree of development and organization of the model, otherwise they are perceived in an indistinct and diffuse way.

A very low or moderately developed level of organization also serves to address the nature of other properties of the somatic model, such as orientation and belongingness of one's own body, linked to the elementary praxis activity. This type of activity is completely disabled in absence of corporeality; and in stages of very moderate activation of the model, such activity becomes defective, slow and disjointed. The remarkable subjective perception of the reduction in the duration time of the processes is another sign of the low functional level.

The muscular effort that activates corporeality, that is, that brings out the somatic model, does not resemble the praxis activity, since the former corresponds to an instinctive impulse carried out in a diffuse way, mainly in the trunk, without choice nor local specificity.

In short, the disorder affecting the emergence and consolidation of the somatic model is due to an increase in the sensory threshold and to disorganization, and would correspond more or less to asomatognosia. This represents the highest degree of disturbance in the body schema and is one of the various stages that can be delimited in our cases depending on the conditions of the test.

It should be noted that the term asomatognosia was used by Lhermitte and Trelles (1933) and Lhermitte (1939) precisely to denote the total fading of the body image or body sensation in the cases of Foerster (1903) and Deny and Camus (1905). These cases were considered rather of a psychiatric type by the respective authors, although these cases undoubtedly presented alteration of postural sensitivity, for example, and had shown a very typical symptomatology of absence of any somatic corporeality. It should be noted that when we use the term asomatognosia for our cases, especially M, we simply refer to the loss of corporeality by abolition of the tactile (somatic) field, discarding here hypotheses and more complex conceptions of other authors in this regard.

27.3. POSTURAL MODEL

The next degree to the somatic model is the postural model, which refers to the perception of the different attitudes of the body. Postures (position sense) play a major role in body schema theory. Thus, for Head and Holmes (1911), the concept of body schema arises as support for the attitudes of the body. Postures also play an important role in our study of body schema, with new empirical data. Postures do not involve a different situation from what has been explained so far about the schema, but rather they are only a further stage in the tactile spatial organization.

Although the interest is focused on the study of segmental postures, for a more complete and systematic study of the postural model, we shall address other aspects of postures, dealing successively with: postures in relation to spatial localization of cutaneous stimuli, general body posture, segmental postures, and finally, theory of the postural model.

27.3.1. Localization of tactile stimulus and postural model

It is clear that for a correct localization of a tactile stimulus on a limb displaced from its natural position, it is necessary to have a sense of attitude, i.e., localization must occur on the postural model. If due to the circumstances of the pathological case, or to the test conditions, the perception of posture is abolished (as may occur in subject M), the localization of the stimulus is possible to some extent in a diffuse corporeality (somatic model) but not in relation to the surrounding space.

Two levels of localization can be distinguished. The simplest level corresponds merely to the site on the body, and statically abides to the somatic model, neutral in postural terms. The other more developed level of localization corresponds to an absolute spatial placement by intervention of the postural sensory model (not to be confused with the postural motor function mentioned in Sec. 25.2.4). However, this classification into two levels of localization does not mean a complete independence between them, as they are degrees of the same organizational process that varies as a whole. That is, if there is a deficit in the schema, the somatic model and the postural model suffer alterations both at the same time, but the latter to a greater extent. This is analogous to what happens in other functions, as mentioned in different

sections. Hence, if postural localization is abolished, the stimulus will not be perceived somatically at the exact location of stimulus application but will be perceived with some proximal deviation.

These conclusions about modes of localization and about schema are deduced from the examination of our brain-injured patients. It is clear that localization under residual sensory field conditions (localization with strong proximal deviation, inversion, etc.) corresponds only to a more or less reduced somatic model. However, the levels of organization, although considerably out of synchronization, can be recruited by intensifying the stimulation. This recruitment is "from outside," i.e., by the action of stimuli. Thus, a painful pinprick that can markedly reduce proximal deviation in patient M inactive, also produces postural activation of the corresponding limb, albeit in an incomplete and diffuse manner. Indeed, an intense prick on a finger can produce a very little deviated cutaneous localization and at the same time awaken more or less the perception of the attitude of that finger, but it is not able to provide a general activation of the whole limb. In fact, the activation reaches at most only up to the wrist in this case. Instead, if the prick is slight and the corresponding sensation stays in very elementary spatial phases, no postural activation is possible and only a certain diffuse corporeality is awakened. In general, it can be stated that when somatic localization is rather good, attitude (posture) emerges, but only for the stimulated segment, either an arm or a finger (see segmental postures in Sec. 27.3.3 below).

In short, segmental posture can be activated by recruitment under intense cutaneous stimulation. In this way, the somatic model evolves to a certain extent to the postural model, thus almost completely overcoming the asynchrony between both models, and therefore achieving the localization of the stimulus in space.

27.3.2. General posture of the body

The *general posture* of the body deserves a separate study. Such a posture is somewhat different from segmental postures which will be discussed in the next subsection. The issue of the general posture of the model arises in our study because of the phenomenon of orientation inversion in tactile space. As we know, such inversion also appears in the body schema (see Sec. 24.4).

According to what has been said about the alteration of orientation in the body schema (Sec. 24.4), the subjective rotation of the body felt by the patient constitutes a *deviation of the somatic model*, i.e., an alteration of the verticality and the normal position of the body as a whole. This alteration connects the stage of the somatic model with that of the postural model, perhaps closer to the former than to the latter. Indeed, the subjective rightward tilt of the somatic model in the form of a spiral turn of the body, when patient M is standing or sitting under weak facilitation by muscular effort, is more pronounced the more reduced the somatic model is (Figs. 24.14 and 24.15). This occurs up to a certain point where it is not possible to feel any position of the body as a whole due to the vanishing of the model.

This subjective deviation of the somatic model thus corresponds to a functional level at which segmental postures can hardly be felt. That is to say, it is almost

impossible for the postural model, in its common meaning, to arise in such a situation. Trying to link the different phenomena (since everything refers to sensory posture), the *general* posture of the body can be considered as the first step in the development of a postural model, model that finally reaches its greatest differentiation with the segmental postures.

Accepting the above, it can be stated that the postural model comes from the somatic model, since the general posture refers to the position and orientation of the somatic model in space. This orientation depends on the state of the local spatial sign which, due to the inversion process in our patient, tends to produce a rotation (tilt) of the model. Specifically, the postural model derives from spatial localization, and thus, an uninterrupted line of phenomena can be established between stimuli localization and postures; *posture is localization*. We again find continuity in the organization. Thus, posture, generally considered as an intellectual or associative phenomenon, is merely an extension of localization or of the local sign. It means that posture is established in a simpler and more direct way.

Certain alterations of the *general* posture of the body have been described in some special cases involving "lack of perception by left hemiplegia." Thus, in one case of Kramer (1915), two cases of Pötzl (1924), others of Stockert (1934), etc., there was a subjective sensation of certain turning or deviation of the affected half of the body towards the healthy half. The case of Kramer also had tactile allochiria (mentioned in Sec. 26.1.1), but not those of Pötzl. The cases of Stockert presented different complications of the body schema, and in addition to the rotation of the model towards the healthy side, they had alterations of localization and orientation in vision, proximal deviation in touch, and auditory allochiria when stimulating the ear of the hemiplegic side. From our point of view, the clinical descriptions of such cases are insufficient to establish that the rotation of the somatic model is the result of an inversion process, and for the moment, without going further into these cases, we merely cite them. Other more or less clear cases of deviation of the model correspond to induced manifestations on the body schema, as in the cases with cerebellar and frontal lesions described by Goldstein (1926) and others, as already mentioned in Sec. 25.2.4.

27.3.3. Segmental postures

With the study of segmental postures we enter fully into the specific activity of the postural model and into one of the most characteristic manifestations of the body schema. Attitude recognition is severely disturbed in our brain-injured patients in the usual state of activity. In general, this ability is always one of the most disturbed functions in cases of cortical involvement of tactile sensitivity, as was first pointed out by Head and Holmes (1911) and Head and Riddoch (1920). Many authors have confirmed this later, and we have been able to verify it in a large number of brain-injured subjects.

As mentioned above, the postural model comes from the somatic model, and the general posture of the body becomes the initial postural activity from which local or segmental attitudes are derived by differentiation. The relationship of the postural model with localization has also been indicated, as well as the role that a segmen-

tal posture plays in the correct localization of tactile stimuli, with the possibility of activation of such posture by intensification of such tactile stimuli (Sec. 27.3.1). However, in the usual recognition of postures, recognition is not activated "from outside" but "from within" by the proper organization of tactile sensitivities, particularly the deep one, up to the "figure" level.

The pathological degradation of segmental postures clearly reveals the nature of the process. Thus, within the parallelism between localization and posture, it is found that in both localization and posture the spatial perturbation is of the same nature, i.e., a proximal deviation. That is, in tactile localization there is deviation towards the midline, and in posture, deviation towards the habitual position of the body (somatic configuration), i.e., towards the *neutral posture*. In short, in the first case, somatic shrinkage is produced, and in the second, postural shrinkage, in such a way that the change in attitudes tends to be neutralized or underestimated.

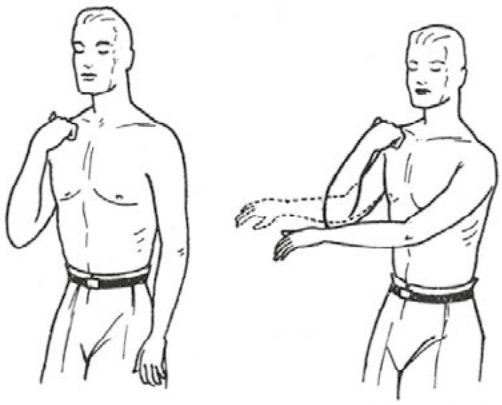

Figure 27.1. Typical phenomenon of postural deviation towards the neutral posture. The passively induced attitude of the right upper limb (left drawing) is perceived, by means of some facilitation, as very deviated towards its most habitual posture; that is, the complete flexion is perceived as a weak flexion, the fist being forward (dotted line in the drawing on the right). Thus, in the active imitation with the other limb, this deviation is displayed (drawing on the right), objectifying the subjective postural deviation.

As early as 1938, long before the discovery of the phenomena of dynamic action excitability (facilitation by muscular effort, etc.), patient M was examined in his habitual state, corresponding to a weak or moderate facilitation by muscular effort. It was then evident that neutral positions predominated in many tests. For example, in the following test, first the forearm is passively flexed over the arm until his fist reaches his shoulder (Fig. 27.1 left), and he is asked to imitate that movement with the other arm. In trying to do so, he tends to raise it stretched forward (Fig. 27.1 on the right), placing it horizontally; that is, he places it in an intermediate position between the habitual one (hanging inert next to his body) and the fully flexed one that he must imitate, thus showing a deviation towards the neutral posture.

Likewise, if keeping one hand completely open, even by his own voluntary movement, he is asked to imitate that attitude with the other hand with eyes closed, the second hand adopts a semi-flexed neutral posture corresponding to the inert attitude of the hand (Fig. 27.2). On opening his eyes, he is surprised by the difference. If what he has to imitate is a clenched fist, the resulting attitude is likewise a semi-flexed hand (Fig. 27.2). Already before the discovery of the facilitation by muscular effort, the patient needed to tense the hand, open or closed, in order not to make mistakes. Otherwise only a diffuse perception was possible, and the usual semi-flexed attitude was predominant.

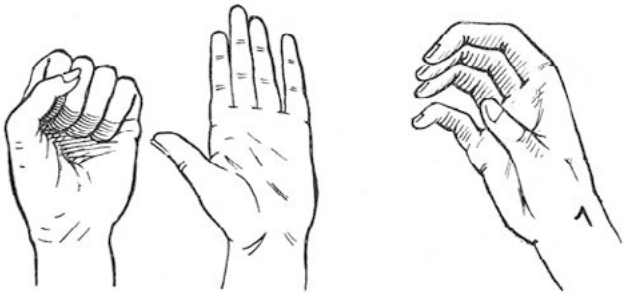

Figure 27.2. Phenomena of postural deviation to the neutral position. On the left, closed fist and completely open hand. On the right, imitation of both positions with the hand semi-flexed (neutral position of the inert hand).

At present, patient M exhibits similar behavior in ordinary circumstances. In the inactive state, he is completely unaware of posture since, as we know, the somatic model does not provide any sensation in that state. However, he maintains the tendency to respond with a neutral position rather than manifesting his inability to perceive a change of attitude in his limbs. For example, with the hands hanging naturally on both sides of the body, if one of them is passively raised, he still believes that both are in the same position, even if he applies some diffuse facilitation. However, when asked insistently, he ends up answering that he does not know for sure but that he thinks they are fine. This postural illusion disappears when facilitation by strong muscular effort is applied. But if the effort is not very intense, several errors may occur, both by deviation to the neutral position and by defects corresponding to the low spatial level (centripetal deviation, inversion, etc.). In order to perceive postural changes in very distal regions, such as the fingers, facilitation must be applied in a generalized manner with the muscular tension being more intense than in the case of asking the patient to voluntarily move a finger.

The most direct tests are those in which the patient describes the posture felt, a posture that has been produced passively. Depending on the intensity of the facilitation applied and the type of posture, the following results are obtained in patient M.

A test that may be useful for examining general posture is head posture as well as trunk postures, as these are midline regions. The head is passively placed backwards (the subject ignores the maneuver because he is in inactive state). The patient is asked to moderately contract the musculature of the body (weak facilitation); then, after five or six seconds he gives notice of the perceived posture, explaining that the head is slightly forward. Due to the low functional level reached, the position (localization) is inverted and with a small deviation from the vertical, since this phase corresponds to a shrunken somatic model.

A test on complex segmental posture is as follows. The forearm is passively placed in flexion over the chest. After five seconds of diffuse muscular effort (weak facilitation), the patient feels the posture and describes it as the forearm somewhat flexed, but not on the chest but somewhat separated from it. He tends to give as a response an intermediate posture between the neutral posture and the passively established posture, showing the phenomenon of deviation towards the neutral position. There is thus a sensory reduction in posture.

A test of differentiation in simple segmental posture is as follows. The thumb is passively maintained in extension, the other fingers in semi-flexion in an inert position. After five seconds with some facilitation by muscular effort he states that all fingers of the hand are more or less extended. Thus, there is a postural differentiation deficit, since in diffuse perception the position of the thumb is generalized to the other fingers.

Under intense facilitation by strong muscular effort, the errors of appreciation disappear or are considerably reduced in all these cases, although some errors may persist in very complex tests.

Another mode of examination corresponds to the usual clinical test in which one limb actively imitates the passively produced posture of the symmetrical limb. Similar results to the previous ones are obtained, as indicated below.

Imitation of the left hand placed on the left parietal: after seven seconds he places his right hand in front of his head, almost at the same height as the other hand. With a very intense facilitation, he performs the appropriate correction.

Imitation of two extended fingers (index and little finger) of his left hand: he extends all the fingers of his right hand. With intense facilitation he corrects with some difficulty, but he is able to distinguish the two fingers from the others.

In these tests it is easily observed that postural imitation produces some deviation towards the neutral position, albeit slight, since the imitation does not complete the full path despite the use of a fairly intense, although not maximal, facilitation.

Given the tactile alteration for the whole body in the central syndrome, postural imitation in the ordinary state of the patients is easily altered, as seen in Figs. 27.1 and 27.2. Both the attitude of the passive model limb and that of the active limb that has to move the patient suffer a sensory reduction. However, it can be admitted that the main defect comes from the passive limb, since the other is more activated by the facilitation. In short, a very intense facilitation is needed in patient M to overcome all defects. But this is not always achieved in very complex attitudes, being in these cases necessary for him to open his eyes to perceive a postural discrepancy in the tested limbs.

27.3.4. Theory of the postural model

The theory of posture was initiated by Head and Holmes (1911) who introduced the notion of body schema as something plastic and hypothetical to which to refer changes in body attitude, which in reality are produced by the postural model. This theory also emphasizes that the perception of position and passive movements depends on the ability to relate perceptions to each other, which is accomplished through the plastic function of schema. It also points out that for the localization of cutaneous stimuli, another type of schema such as a model of the body surface is required.

Such a classification corresponds to what we have explained here about the somatic model (for localization) and the postural model (for attitudes), but these being conceived in a much simpler sense than that expressed by Head and Holmes (1911), since we are dealing purely and simply with tactile spatial organization without sensory influences other than touch. Thus, in contrast to the emphasis on the ability to compare or relate perceptions to each other, in our conception, phenomena are rather a direct expression of the state of the sensory field (degree of organization, spatial dimension, etc.).

As for the various sensory influences, there is an old opinion about the collaboration of visual images to the sense of position. This opinion is also shared by Head and Holmes (1911), but becomes more prominent after the interpretations of the Schneider case by Goldstein and Gelb (1919). These authors think that the whole disorder of tactile space is due to the loss of visual influence on touch. This point of view was later adopted by Schilder (1923/1935) to support his study on the body schema. This issue has already been discussed in Sec. 22.4, devoted to the theory of tactile localization and spatial organization. The explanation of the visual influence in the Schneider case has been discarded in the first part of this book, when exposing the central syndrome (Sec. 1.4 in Vol. 1).

As for localization, it has been shown that it depends on the state of the tactile sensory field as a direct expression of neural organization (localization phases according to neural mass, decussations, etc.). There is thus a simple interpretation for tactile localization of stimuli, and something analogous is valid for passive movements and postures since posture can be understood as localization. About this we have said in Sec. 27.3.1 how posture can be recruited by intensification of a skin stimulus in the same way that proximal deviation is overcome in the somatic model. We have also indicated (in Sec. 27.3.2) that the subjective lateral deviation (rotation) experienced by the general posture of the body (Fig. 24.14) is due to the effect of inversion of the local sign. Finally, it was indicated in Sec. 27.3.3 that segmental postures are pathologically reduced in the sense that they are perceived to be closer to the habitual (neutral) positions. This reduction runs parallel to the proximal deviation in the somatic model. In conclusion, considering posture as localization is fully justified by the observations.

It should be noted that in the detailed studies of Goldstein and Gelb (1919) on the Schneider case nothing is mentioned about the phenomenon described here on the subjective postural deviation towards the neutral position. Nor is it mentioned in the meticulous observations of Head and Holmes (1911/1912). In all these authors the concept (and facts) about degrees of disturbance of a given function seems to be

completely absent. This concept plays instead a fundamental role (asynchrony, new phases, abnormal steps, etc.) in the brain dynamics presented here, as we have repeatedly shown.

> According to Goldstein and Gelb (1919) the Schneider patient was not aware of the position of a given limb as long as he did not move it. When he was holding the arm in a horizontal position, he tried to get a clear idea of its position by oscillatory movements of the shoulder joints, then of the elbow joints, and then of the whole body. He recognized postures by their critical points (*ausgezeichneten Stellungen* according to these authors); for example, in the fact that the elbow can no longer move in a certain direction. When he was in a horizontal position, he recognized it by the pressure he felt on his back. When he was in a vertical position, he recognized it by the pressure on the soles of his feet and a certain sensation in his knees. He did not perceive deviations from the horizontal up to 45°, and he considered this deviation as horizontal. He was able to imitate the posture of a limb with the symmetrical limb when the positions were critical and by moving both limbs.
>
> Postures were thus "recognized" by means of contrivances and in a very indirect way. Although the patient could seem to behave normally by allowing him to perform movements or muscular twitches, the authors claim that he was not able to perceive the posture as such, but only to have some idea by means of indirect processes. As already indicated, in our opinion, muscular twitches constitute a facilitation of nervous summation in the nervous centers that improves the general level of excitability. In addition, they intensify joint proprioception, as has been observed in many brain-injured patients and even in a normal subject after a long rest. Therefore, the Schneider patient, to the extent that he achieves facilitation according to the intensity of movements and muscular tension, is able to perceive the posture directly. The same occurs in subject M with a greater brain injury.

Considering more specifically the phenomenon described here of postural deviation towards the neutral position, we have a very remarkable precedent in the normal individual under certain conditions. It does not invalidate this pathological phenomenon, but rather gives it greater support. In a normal subject, neutral body attitudes exert a great influence on spatial sensation and even cause sensory confusions. It is a topic that comes from Aristoteles (384-322 BC), and is developed especially by a series of authors such as Czermak (1852), Henri (1900), Kramer (1915), Tastevin (1937), Ponzo (1910), Rupp (1912), and mainly Spearman (1907) and Skramlik (1937) among others. The influence of the natural positions of the fingers explains the well-known 'Aristotle's illusion' and the so-called 'Japanese illusion' (error when moving a finger having the hands crossed and the sides changed), as well as other illusions. Thus, a stimulus on deviated lips tends to be felt in normal position although not completely.

The observations of greatest significance for our purpose are the following. According to Henri (1900), there is a tendency for the position of the excited arm to be perceived as too low, with preference for a position close to the body. Kramer (1915) also indicates that by placing one arm in a symmetrical position with re-

spect to the other, there is a deviation towards a more comfortable or habitual position. Mainly Spearman (1907), studying normal illusions, finds that there is some proximal deviation with shortening of the arm as well as an illusion about the angle which is perceived as smaller. This illusion increases notably when the joint is at rest. This is also pointed out by Rupp (1912) when referring to the tendency to prefer the closest positions to the body. As Skramlik (1937) states, every habitual (neutral) posture acts sensorially so intensely that deviated postures are perceived as being close to the neutral posture, and are thus underestimated. For example, different tests with the hand prove that the perceived postural deviation is toward the semi-flexed position of the fingers, i.e., to the neutral posture resulting from inert muscle tone (Fig. 27.3). It should be noted that in the normal subject such postural illusions occur mostly under certain conditions, such as attention focused in a particular way, a long period of rest, etc. In a long period of rest, joint positions do not produce any appreciable sensation and then the image of the neutral position predominates. Sensory illusions disappear when moving the joints, as for example in the finger-crossing test. Thus, postural illusions in the normal subject appear easily due to a certain diffuse perception, thus giving rise to the neutral posture, which corresponds to the most frequent configuration of the body by virtue of its architecture and muscle tone.

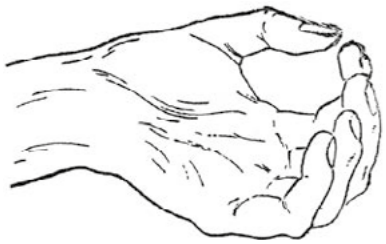

Figure 27.3. Normal hand position according to Skramlik (1937). When the perception of the hand attitude is very diffuse, this habitual (neutral) position tends to be perceived. See Fig. 27.2.

Thus, it is observed that the difference between the pathological cases and a normal individual is only quantitative. Whereas in the normal there is a certain deviation under special conditions, in the pathological cases the predominance of the neutral position in perception is persistent and very noticeable. The more diffuse and unstable the tactile function is, the more the neutral posture is manifested in perception. Thus, when the sensation of touch is about to disappear, any type of induced posture is perceived as a neutral position. As we have seen in patient M, when there is no perception of the passively acquired posture, he responds according to the neutral posture, which, in addition, implies a certain unawareness of his defect.

When the perception of the posture is incomplete, the response is deviated towards the neutral posture. This process of deviation towards neutral posture is part of the reduction mechanism in the different activities of form perception. We

have already seen that figures drawn on the skin tend to be perceived as deformed along the longitudinal axis of the limb (Fig. 23.2), or simplified to more stable figures (Fig. 23.3). The latter is more clearly demonstrated in the visual system with respect to visual forms (metamorphopsic pseudoagnosia, see Sec. 11.3 in Vol. 1), and in different schematic drawings (Figs. 15.1-15.6) that give rise to a greater sensory illusion the greater the perceptual instability.

In conclusion, posture is considered a type of localization. It develops from the general posture of the body to the segmental postures. Pathologically the perception of posture is not suppressed at once but presents, like all functions, degrees of reduction that are manifested in the degree of postural deviation towards the neutral position, giving rise to the corresponding underestimation. This conception is very simple and consistent with the postural mechanism in a normal individual. Such reduction tends to convert the postural model into the somatic model, since the latter constitutes a static configuration and the postural model involves an increase in the spatial organization of the somatic model. There is a reduction of the schema that first affects the postural model and then continues with the somatic model according to the degree of organization of the sensory field.

27.4. PRAXIS MODEL

Voluntary movements are linked to the body schema both in motor initiation and in the subsequent development of the action. Thus, praxis obeys an organizational plan derived from the body schema, justifying the term 'praxis model' used here.

> Such a conception, which is now very essential, already existed more or less in many authors, such as Schilder (1923/1935), Grünbaum (1930), Conrad (1933), Lhermitte and Trelles (1933), Lhermitte (1939), etc. Earlier classical authors on apraxia such as Liepmann (1900, 1920) or Pick (1905) could also adhere to such a concept if the notion of body schema had existed at that time. Schilder considers that apraxia depends on a deficit in the evaluation of the body schema, and Lhermitte thinks similarly. In Grünbaum and Conrad we find complementary concepts. According to Grünbaum, handling tools or utensils is equivalent to an extension of the body schema, whereas for Conrad, the concept of body schema involves both the conceived space and the space of action. Similarly, Bogaert (1934) interprets the postural model as active, extending it to praxis activity.

In our study, the concept of praxis model becomes necessary because the action plan runs parallel to the body schema in its various degrees, until the normal praxis behavior is reached by virtue of the corresponding reorganizations. Given the nature of the praxis model, in its pathological reduction, praxis behavior is simplified becoming diffuse and discontinuous, the coupling with the environment becoming disrupted. In severe alteration, phenomena of previous stages are manifested, appearing a great disorganization of movements as well as a series of incongruities due to the effect of somatic inversion.

The purpose here is not to study the classical apraxia phenomena or their variants, but to study peculiar disorders in relation to the alteration of the body schema, according to different functional levels that show us the internal structure of action. To this end, the following issues will be addressed: dyspraxia in rudimentary model, dyspraxia in the coupling of the model with external space, and dyspraxia due to instability of the model. Finally, a theoretical summary on general aspects will be given.

27.4.1. Dyspraxia in rudimentary model

It can be stated in general that actions are derived from the spatial orientation provided by the body schema, and thus, praxis model means to a large extent motor orientation. Especially in the most elementary phases, such as the first motor attempts, orientation with respect to the body acquires a major significance. Therefore, we must take up again certain aspects of the already studied phases of the body schema.

27.4.1.a) Asomatognosia and motor action

As we know, in the inactive state of subject M, there is an effective abolition of corporeality (asomatognosia), and of course also of all orientation and capacity for voluntary selective movements (see Sec. 27.2.2). Such a deficit is hardly perceived by the subject, so we can consider that there is anosognosia, i.e., lack of perception of his deficit, as is the general rule for the various disorders in our brain-injured patients. In such conditions, it seems that this deficit is replaced with a diffuse sensation of the neutral model, as mentioned above. That is, the illusion of the neutral postural model dominates, giving rise to a pseudognosis regarding the orientation of the own body. Similar to the appearance of a phantom limb in amputees, a whole phantom body, or at least an illusory model, now appears.

In these circumstances, patient M makes all kinds of orientation errors with respect to himself. For example, he is asked to find with one hand the other hand that had been passively placed at the level of the head. He then directs the active hand towards the habitual (neutral) position of the other hand, and not finding it, he tries it in the vicinity, and in view of the failure, he looks for the elbow which he does not find either, and so he goes on until he reaches the shoulder, bumping into the observer's hand which he mistakes for his own and he tries to grasp it being fully convinced that it is his own. Meanwhile, with his own hand on his head he is passively scratched until he is able to perceive localization (phase II); then he says that someone is touching his head. If he applies facilitation by maximal muscular effort, he immediately becomes aware of the confusions.

In the test of one hand seeking the other, the action is rather *automatic* and not a selective movement in response to a specific request. For this reason, the action occurs in the absence of an effective model, without the need of activation through facilitation by muscular effort. Automatic action and anosognosia occur at the same functional level of undifferentiation of the body schema, neither the anomalous po-

sition of the limb nor any other type of corporeality being perceived. If the observer's head comes between the head of the subject under examination and his actively seeking hand, this head is taken as his own, as is the case with the alien hand as mentioned above. In short, automatic action is guided by the illusion of the neutral postural model according to the state of the rudimentary model.

In connection with the above, it is worth mentioning the very frequent tendency in various tests to maintain the postures that have been given to the limbs no matter how painful they may be, and to believe that all is well. There is a certain postural perseveration, which is unnoticed by the subject due to the lack of sensory control. However, pain arises with fatigue, and this can lead to the perception of various postures.

> We have been able to observe this type of disorientation with respect to the own body in countless war brain injuries, and also in civilian cases due to diverse lesions (vascular, tumoral, etc.). In these cases, the subjects were cognitively unaware of their own defect. Most of our cases examined during the Spanish Civil War (1936-1939) had parietal lesions causing contralateral hemiparesis and abolition of spatial and deep sensitivity on the same side. Many of them had a spontaneous sensation of "missing" limb, more rarely of "foreign" limb or of total absence of the involved side. Most of them confused the observer's limbs with their own in the tests, and even failed to visually recognize their own hand (the affected one). One case presented the illusion of believing that the totally paralyzed limb was moving. In tests to find the affected limb, passively hidden, some of these subjects always searched for the limb (or hand) by going down from the shoulder on the same side. In the most severe cases, the subjects did not search systematically, or had little conviction when encountering the member. Other subjects were not even able to look for their own hand, etc. Ranking the cases according to their behavior in these tests, from least to most impairment, we obtain the following: 1, the patient moves the limb to activate the internal perception; 2, he searches systematically; 3, he does not search in an organized way, or has little conviction when he encounters the limb; 4, he does not know how to search for the hand, or does not even try to, he moves the hand without being aware of it.
>
> In connection with the above, a subject injured in the parietal area, examined in 1938, with right hemiparesis and hemihypoesthesia, presented phenomena of special interest, as follows. Hemihypoesthesia affected preferentially, as expected, spatial functions, mainly in the distal part of the upper limb. In the hand, the simultaneous Weber was abolished for any separation, whereas the successive Weber was preserved, although very enlarged (5 to 8 cm). Joint movement was rarely felt, and only in the shoulder. When the arm was passively moved, he perceived only a static contact in the shoulder. In the localization of skin stimuli, he made many errors in *naming* the site, and usually with proximal deviation. When *pointing* with his healthy hand he made the same type of errors, perhaps with greater proximal deviation, and often pointed diffusely from the air (see Sec. 21.1). Particularly noteworthy for the matter at hand is that if the affected limb was stimulated and then moved from its habitual position, he tended to point to the habitual position since the subject had no perception of the change. In other words, he localized according to the more or less preserved somatic model but not in

relation to the postural model. Thus, having his hand next to its corresponding shoulder, when stimulating each of these parts separately, shoulder or hand, the *verbal* response was correct, but if he *pointed* to the place touched, he did well for the shoulder but very incorrectly for the hand. For the hand, he pointed from the air toward the natural (neutral) place of the hand. That is, as in the previous test, he localized according to the neutral posture. Such a condition of the patient, of course disturbed his orientation with respect to his own body. If the affected hand was displaced, he was looking for it irregularly all over the body and in the air, although he did not confuse it with a foreign hand. After many attempts, he used to go to the shoulder, and going down the arm he was able to reach the corresponding hand. He couldn't feel the hand at all, lacking any spontaneous sensation from the shoulder down. When he was asked about the hand, he did not know where it was. If it was in front of his eyes he was able to recognize it correctly although not very quickly. In bed he had the feeling that his arm was missing.

These cases and similar ones described by Stockert (1934), Hoff and Pötzl (1935 b), Lhermitte (1939), etc., generally deal with diverse disorders of the body scheme, in which the subject is either unaware of his own deficit (hemiplegia, hemianesthesia, etc.), or suffers from anosognosia of Babinski (1914, 1918) [syndrome of Anton (1898, 1899)], or disorientation with respect to his own body or autotopagnosia of Pick (1908 b, 1915, 1922) in a more or less pure form.

Although further study of these phenomena is still necessary, the different cases can be classified with respect to the perception of the defect into subjects aware of it and those unaware of it, as Stockert (1934) does, making an analogy with subjects with hemianopsia having "vision noire" (visual blackness) and "nulle vision" (null vision) respectively. In those who are aware of their defect, the sensation that a part of the body does not exist (opposite effect to the phantom limb of amputees) would be due to deep brain lesions (subcortical), which according to Hoff and Pötzl (1935 b) and Stockert (1934), among other authors, would be parietal and thalamic lesions. In those who are unaware of their defect, the lesions would be more superficial, although Stockert (1934) refers them to the corpus callosum. In these last cases, the body defect is replaced with diverse totalizations, corresponding to true anosognosia, being the most common manifestation in these brain-injured patients, although with different variants as shown by our cases mentioned just before.

27.4.1.b) Inversion

At the sensory level considered in the precedent subsection *a*) (subject in an inactive state), movements occur almost automatically, without the need for any great initiative or plan of action on the part of the subject. Many daily actions are, of course, of this type, so the body schema deficit is not a major obstacle.

However, when relaxation is not so extreme and the body scheme is not so deficient due to the effect of certain facilitation by muscular effort, elementary voluntary movements can be initiated according to the characteristics already studied. When these movements are either not large, or the sensory level is very reduced, the sensation of movement is not felt, or if it is felt, this sensation is very altered: with proximal deviation and inversion of the direction of the movement.

For example, if subject M is asked to raise a hand or arm very quickly while being as inactive as possible, he spends about two seconds to raise it 90° to horizontal, but he only senses a phase of simple contact and therefore does not sense the movement. Under facilitation by muscular effort, he takes less than a second to perform the same action, but when he is asked to drop the arm immediately, the sensation of movement, although reached, does not develop sufficiently due to the brevity of the test, and he always answers that the movement is of the opposite arm. Since the movement has been improvised at random, he is then guided by the movement felt and not by the one carried out. This implies a remarkable disorientation about himself. Only when the facilitation by muscular effort and the action last longer, there is no disparity between his action and his perception, the behavior being then practically normal.

In conditions of low functional level in which there is inversion of the direction of movement, the subject presents a great disorientation about his own body, resulting in an unprecedented type of *autotopagnosia by inversion.* This is what we have already studied as *sensory-motor incongruence of the movement direction* (Sec. 24.3.3). This is a notable situation of conflict in the praxis model, a situation that tends to disappear spontaneously by means of an automatic change of references in the sensory-motor complex which becomes entirely governed by the sensory factor now inverted. It can then be said that the voluntary movement of the subject becomes a pseudo-active movement since he is unaware of its true character. Thereby, elemental voluntary movements show aspects unknown until now, revealing further aspects of sensory-motor coordination in the genesis of voluntary movements.

Another very singular type of inversion in praxis, as a result of the transposition of sides in the body schema, is observed in tests in which the subject has to choose one of his fingers in response to a verbal request under certain experimental conditions. The results are shown in Table 27.1.

Table 27.1. Response action of subject M to the verbal request to show a particular finger, in a semi-inactive state and under facilitation by maximum muscular effort. Response time in seconds.

Finger to be shown	Response in semi-inactive state. Response time	Response under max. facilitation
Left index	Right index. 7 s	Corrects quickly
Right thumb	Left index, then left thumb. 7 s	Idem
Right little finger	Left little finger. 7 s	Idem
Right middle	Left index, then left middle. 7 s	Idem
Left thumb	Right thumb. 7 s	Idem

In Table 27.1 it can be seen that in the semi-inactive state (with weak facilitation), after some delay, the remarkable phenomenon of inversion (lateral transposition) is *persistently* present. There is also some confusion about the kind of finger,

but on a second attempt he gets it right although not about the side which remains changed. Keeping the eyes open or closed does not noticeably influence the response. By contrast, if he applies facilitation by maximal muscular effort in each test, the order is executed immediately and correctly. Interestingly, the subject believes that no variation has occurred, which means that he is unaware of the errors in the semi-inactive state.

The persistent contralateral inversion of the response is somewhat shocking and unexpected, apart from the confusion between the fingers, since when studying the execution of an active movement (Sec. 24.3.3) we have seen that the requested action was correctly performed, although the sensation of inversion was felt immediately afterwards. The explanation for this difference may lie in the character of the movement in question, quite automatic and simple there (Sec. 24.3.3), and much more selective now. If the requested movement is not particularly selective, such as lifting an arm or moving the head (midline organ), the movement has a basically automatic motor character. By contrast, if the request is very selective, such as moving a particular finger of a particular hand, a much greater bodily discernment is inevitably required - to choose first the side and then the finger - which forces the subject to be guided by his own body schema. Thus, the motor engram predominates in the first case whereas proprioceptive or sensory control predominates in the second one.

Everything seems to indicate that due to the rudimentary state of the body schema and the great importance that the sensory factor acquires in the sensory-motor complex of the praxic impulse (as explained above), the impulse for the corresponding movement suffers a *contralateral canalization* due to the sensory inversion inherent to a low functional level. The sensory-motor incongruity of other tests involving simpler or more automatic movements, which subsequently tends to disappear, does not occur in the situation we are now considering. In this latter situation, the change of reference arises from the beginning, and the motor factor is entirely subordinated to the orientation determined by the sensory factor.

Due to the high specificity of the requested movement, as well as the subject's condition, the subject has no choice but to try to orient himself by his bodily sensation in order to activate the praxic impulse. This change of side is only the initial process of the motor impulse, which in order to reach a certain finger needs a new activation more distal of the scheme, which if sufficient, can achieve the mobilization of the requested finger. The process is therefore discontinuous.

Deepening in this phenomenology, we find that, in order to avoid confusions between the fingers, the activation of the schema must reach the root of the fingers, otherwise he confuses them because he cannot discriminate between them. It seems that when the subject is asked to move any finger, without specifying the side of the body, it is not necessary that the body schema reaches beyond the shoulder, since no specific movement is requested. If the schema reaches up to the elbow, the request to move the right little finger results in a movement of the left middle finger. As for the easiness to confuse fingers, it seems to be much lower in the marginal fingers (thumb and little finger), a little more in the index finger, and the maximum confusion occurs with the ring and middle fingers. This is somewhat related to what usually occurs in the normal subject.

Another issue is that when the subject applies maximum facilitation immediately correcting the previous errors, he has the sensation that there is no change with respect to the inactive state condition, the change of side being totally ignored. This can be explained by two reasons. One reason is that the subject has been guided, both in the inactive state and under facilitation, by the sensation of laterality that he has been able to have in each case, sufficiently understanding the existence of such laterality. The other reason is that, although he understands that there are different body sides, he does not distinguish between them sufficiently when he has *to compare them with each other*, at least in his usual (semi-inactive) state. In fact, the "left-right spatial arrangement" (rather than the lateral orientation of the body) is usually of little relevance to him, and therefore he does not perceive much difference with the result of the test under facilitation.

As seen, the degree of failure in the tests depends on the degree of somatic activation. It is noteworthy that subject M, when first examined in 1938, already showed some evident disorder of the finger agnosia type, although of a different nature from that now described. In that disorder, lateral confusion was not very frequent (20% of the tests), and confusion between the fingers was quite persistent (70% of the tests) although he was sometimes able to correct spontaneously. The result was not much different if the eyes were open or closed. This type of disturbance, corresponding to another functional level, consists of a left-right deficit (not primary spatial inversion) and confusion between fingers, being part of the usual syndrome of Gerstmann (1924, 1927, 1930, 1931, 1940). However, in the case of persistent contralateral canalization, the disorder is deeper, beyond that syndrome.

In the light of our research, these old results should be interpreted as the effect of a habitual facilitation (by muscular effort) that produces an incomplete but sufficient activation to overcome the somatic inversion. By contrast, in the later tests, lateral inversion has arisen without being sought, probably because the subject has been trained over many tests to avoid facilitation or to graduate it variably, spontaneously giving rise to favorable conditions for this phenomenon.

27.4.2. Dyspraxia in the coupling of the praxis model with external space

At a functional level higher than the one studied above, praxis activity is related to the degree of coupling of the body with external space. A certain organization of the area of activity is necessary in order to reach a more complex level. As before, the alterations we found are both agnosia and apraxia, without being able to establish priorities, but rather, the whole disorder of the praxis model can be understood as an apractognosia which we study here at different levels of alteration.

27.4.2.a) Spatial orientation and left-right actions

The ability of spatial orientation in praxis is determined by pointing tests, either towards oneself or towards external directions. As for the first type, patient M in his habitual state (with moderate spontaneous facilitation) makes mistakes when asked to point at certain parts of his body, with his eyes closed and sometimes also open.

For this reason, in the tests on stimulus localization in Sec. 21, it was preferable to resort to the verbal statement of the sensed location rather than to point at it. The mistakes made by the patient are usually a simple effect of the reduction of the body schema, with a tendency to point towards the center of the body, as can be appreciated in the following results:

If asked to point at the chin, he points about 4 cm below and 5 cm away, but on the midline. If asked to point at the knee, he points at the thigh. If asked to point at the shoulder, he points at the middle of the arm. If asked to point at the wrist, he points at the hand. If asked to point at the forehead, he points at the nose. The subject corrects these defects spontaneously and easily by applying facilitation through maximal muscular effort.

This same diffuse or imprecise character in the action of pointing is also present when subject M must indicate with the finger directions in space. When he is sitting or standing, in his habitual state of activity (little facilitation), he indicates spatial directions very incompletely, both with his eyes closed and open, although especially in the first case, as shown in the following tests:

If asked to point upwards, he points with the right index finger obliquely: upwards and to the right with a tilt of about 45°. If asked to point forward, he points index finger forward and slightly to the right. If asked to point downward, he points obliquely to the right and 30° below the horizontal. As in the previous tests, a complete and spontaneous correction is obtained by applying facilitation through maximal muscular effort.

As can be seen, the requested directions are indicated partially and with deviation to the right. The latter is interpreted by the deviation of the body schema to that side (see Sec. 24.4 and Sec. 25.2.3). In both types of tests, on oneself and on external space, there is an effect of reduction (shrinkage) of the praxis model since the deviation of the direction occurs in parallel to the reduction (shrinkage) of the somatic model, as we know.

As for the *left-right orientation* and the corresponding action, there are numerous different phases of alteration. We have already seen that elementary actions, such as briefly lifting a limb, remain disconnected from the whole and provide no sensation of movement and laterality. At other times, when the subject pays attention to what he feels and not to what he has moved, the action is perceived contralaterally due to the effect of inversion. In other cases, when the fingers are moved in response to a given order, the action is produced from the opposite side, due to an inversion effect by insufficient activation of the model. However, the subject is not aware of the change, as demonstrated when correcting by means of facilitation by muscular effort. In better conditions, even if the subject has a certain idea about laterality, he is not able to sufficiently distinguish both sides, especially when he has to relate or compare them with each other. As long as simple situations are involved, such as indicating a particular side of his body or of the environment, a fairly acceptable action can often be achieved if his functional level is not too low (moderate facilitation); but as soon as the test becomes more complicated or different relationships have to be established, the defect arises. A somewhat complicated test is to ask the subject to indicate the left or right side of the examiner in front of him. At the begin-

ning, the subject usually has difficulty and confuses the sides, but soon he manages to orient himself better, either because he applies a greater facilitation by muscular effort or because he understands the situation better. However, if after a while the tests are repeated, the subject makes the same mistakes again at the beginning, being able to correct them by applying more facilitation.

The distinction between left and right, and in general the discernment of spatial orientation is very unstable, which is demonstrated in many ways. Let us begin by recalling that letters and numbers drawn on the skin, either in normal form or as mirror writing, are perceived in the same way as long as subject M does not apply facilitation by maximal muscular effort (see Sec. 23.3), showing a behavior similar to that shown with respect to the orientation of visual figures.

Even more remarkable is the lack of spatial orientation of subject M when he draws any number in the air. Thus, if he is asked to draw a 2 in the air, in a semi-inactive state, there is a latency of 6 seconds preparing for the action; then, with the index finger of the right hand he usually draws the 2 correctly, but with the left hand he draws the mirror image. Sometimes he simplifies very much the 2 and draws a kind of 9. In the execution of the drawing probably contributes both a certain automatism with a diffuse orientation (see below Sec. 27.4.3) and a moderate facilitation that corrects the latent praxis deficit. It is noteworthy that sometimes he can draw the 2 in the correct position with the left hand, but without having previously drawn it with the right hand; otherwise, he writes it as a mirror image. However, with sufficient facilitation (by muscular effort), there is no inversion with the left hand even if he had previously drawn it correctly with the right hand under facilitation. It has also happened that writing correctly with the left hand under facilitation, subsequently in a semi-inactive state he does it as a mirror image with the same hand. In conclusion, both in the case of numbers drawn on the skin (to test orientation on oneself) and when drawing them in the air with any hand (to test orientation on external space), there is an important orientation defect, the errors are not noticed, everything looks the same and laterality is blurred.

This disorder is not only for tactile space, it is a spatial reduction that also affects visual space, as studied in the corresponding place. Thus, for example, subject M knows whether an isolated object is located to the right or to the left of his body, but if several objects modify their relative positions, he does not notice the change (see Sec. 16.2 in Vol. 1). It is also worth remembering the 'orthogonal disorder' in drawings, prints, etc., in which very different and anomalous orientations of drawings, letters, numbers, etc., are perceived as normal and always the same, without noticing any difference (Sec. 16.1.2 in Vol.1). It is remarkable that even the right hand can invert or change noticeably the writing of numbers (e.g., 6 and 9). Even more noticeable are the special changes in the praxis of writing (which would be a singular paragraphia) when the subject is copying a certain symbol in an anomalous position which he perceives as in normal position (see Fig. 16.3 in Vol. 1).

In order to complete the present complex syndrome, we include various data from brain-injured patients examined in 1938 according to ordinary clinical procedures (prior to this research on brain dynamics).

A case with *mild* disorders of the type now studied is that of patient U. G., whose abbreviated clinical history is mentioned in Sec. 26.1.1 and Fig. 26.1. This case was considered as one of syndrome of Gerstmann (1924, 1927, 1930, 1931, 1940), with predominance of apraxia, in addition to allochria and other disorders of unilateral sensitivity. Six months after he was injured, he presented various symptoms of Gerstmann's syndrome. As for finger agnosia (showing fingers as requested), he made only minor mistakes, especially at the beginning of the test, the mistake being only between fingers (changing the fingers of the same hand) and not changing the side of the body, which occurs only rarely. In addition, there was some deficit in naming the fingers shown to him, as well as amnesia for colors, etc. In relation to the left-right orientation, there was only some confusion at the beginning of the examination. Thus, movements that should be performed with the left hand were executed with the right hand (left hand apraxia). When executing orders to make a "cross" movement (e.g., touching with one hand the opposite ear), he tried to activate in an elementary way the parts to which the movements referred, in order to improve his orientation. In addition, according to his account, he sometimes inadvertently wrote as mirror image from right to left with his right hand. In order to make visible in some way the left-right disorder, it was necessary to resort to more complicated tests. Thus, he made several mistakes when he was asked to point out the right and left sides on himself and on the examiner placed in front of him, since due to his functional lability, it was difficult for him to get adapted. He also presented some constructional apraxia (copying drawings with wrong orientation or changing the perspective, etc.), some dyspraxia with eyes closed at the beginning, agnosia, and discrete acalculia. There was a great improvement after several months, especially in agraphia, which practically disappeared.

Another case in which the praxis model was *severely* affected is that of the brain-injured patient J. C., who suffered from a deep brain abscess in the right upper parieto-occipital area as a consequence of shrapnel. It was drained for quite some time and the patient was examined at the end of 1938. No paralysis or anesthesia was evident with ordinary tests, presenting a patent severe autotopagnosia which was the cause of innumerable mistakes in orientation on his own body. He also presented an elementary difficulty in praxis, predominantly in the left hand, which made it difficult for him to initiate the movements he was instructed to do. There were also several apraxia disorders closely related to schema alteration, left-right disorientation, etc.; some selected examples are described in the following.

As for orientation on his own body, he pointed at his elbow when asked to point at his knee. When asked to point at his hip, he pointed at his chest and afterwards at his flank. When asked to raise his left hand, he always moved his right hand and only much later did he get it right. In cross movements such as touching his right ear with his left hand, he was unable to do so, and he touched his right ear with his right hand. When he had to take his hands with his eyes closed, it was easy for him to confuse one of his hands with those of the examiner who put his own hands in between. Moreover, he remained firm in his confusion even with his eyes open. Disorientation and even the feeling of not belonging to his body seemed to exist especially for the left side. Thus, with both hands hidden behind his head, when asked for his left hand, he only said, "here," and then said that it was in front of his head. In contrast, he was more easily

able to indicate his right hand. To know where his left hand was, he moved it but did not know how to say more than "it is here." When asked to look for his left hand, he did not know what to do and tapped his chest with his right hand. When asked again, he looked to the left and pressed his left hand. With his right hand he grasped the first hand he found, even if it was not his own. If he was asked to lower his left hand when both hands were raised, he lowered his right hand. He made serious mistakes in the position of both hands, needing to move them to activate the body schema. He confused the sides of the objects and those of the examiner in front of him. When he was asked to point at the parts of the examiner's body, he always did it with the right hand and with great difficulties and mistakes, continually getting upset by his failure. With his left hand he had great difficulty in all kinds of actions, even on himself, since he had great inability to initiate movements and even more inability to direct those movements. He looked for his own shoulder in the air, and then all around his body, etc. He imitated the military salute in any manner. He was extraordinarily apathetic and inattentive. When copying a square, he showed a great lack of initiative, then he drew a total of three lines, one separate and two at right angles, etc.

After a little over a month he had less difficulty orienting himself on his own body, finding his hands, etc., although he still needed to move his left hand to find it. The feeling of belonging to his body was practically normal (no confusion with other people's limbs). Instead, some left-right confusion persisted among their own limbs, and also when orienting in external space. There was almost no apraxia in the onset of movements but there was still a moderate general ideational apraxia (in the test of sending a kiss to someone with his hand, he only kissed the fingers together without any other action). He correctly performed automatic actions such as threatening, combing his hair, military salute, etc., but failed when trying to perform a more complicated action such as crossing his hands to imitate a butterfly, crossing his hands to grasp the opposite ears, etc. With his eyes open, he imitated positions of the limbs, slowly and with difficulty. He did not know the names of the fingers and had no interest in it. In general, left hand apraxia was dominant. He was unable to decide to copy simple geometrical drawings, or finally did it very badly. He was rather euphoric, somewhat indifferent and lacking general motivation.

As a whole, it is a case of severe disorganization of the praxis model, with pure autotopagnosia generalized to the whole body, and with apraxia of basic character. The disorders can be sorted according to their severity as follows, starting with the most severe ones: disorders of belonging to one's own body, disorders of orientation on one's own body, disorders of orientation on external space, apraxia-type disorders and constructional apraxia. In a partial improvement, the most severe alterations disappeared, but there remained a certain disorder of orientation in external space and in left-right orientation, as well as dyspraxia, especially of the left hand, and constructional apraxia, the latter being one of the most difficult alterations to disappear because it corresponds to the most elaborated function of the praxis model.

Finally, we describe a third case (patient A. C.) of *moderate* praxis disorder, somewhat different from the previous ones since it is a Gerstmann`s syndrome due to a typical lesion of the left angular gyrus in a brain-injured man, examined in 1938. Immediately after being wounded, he presented some sensory aphasia (he only understood

isolated words and had jargon aphasia, among other disorders), evolving later to amnesic aphasia, in addition to agraphia (at the beginning he was unable to write even his name) which over time became paragraphia. On closer examination, he presented the various phenomena of Gerstmann's syndrome, with a predominance sometimes of amnesia or agnosia (left-right confusion, finger agnosia, agraphia, constructional apraxia, some acalculia and amnesic aphasia, especially for colors).

Regarding orientation and left-right handling, the execution of commands to move the limbs were correct, but confusions appeared in more complex actions such as crossed movements, and even more in actions performed on the examiner in front of him. Thus, the number of mistakes grew as the tests got a little more complicated. For example, when asked to touch the examiner's right eye (in front of him) with his left hand, the subject touched the examiner's right eye but with his right hand. However, when asked to raise his left hand, he did it correctly despite mistaking the hand a little earlier. As for the test of showing the fingers on request to check finger agnosia, he never mistook the side, but almost always mistook the finger of the hand. The constructional apraxia was of the same type as in the first case described above, as well as agraphia, paragraphia and acalculia; in the latter, he had difficulty in reading quantities and performing elementary arithmetic operations, writing 11 instead of 101, etc. The left-right disorientation was again evident when reading quantities with many digits; he did not know whether he should start ordering them from the left or from the right. He also had some general dyspraxia which was very clear when he tried to put on his gloves.

During the recovery from this disorder, the most resistant alterations have been, as always, finger agnosia and constructional apraxia, in addition to amnesic symptoms and difficulty in calculation, all corresponding to higher levels of the schema or of the 'categorial function.'

In relation to recovery, it is worth mentioning what was established by Zutt (1932), perfectly in line with our observations. Thus, in the recovery from the orientation disorder in the present case, the left-right orientation is recovered first for the body but not for external space, therefore the orientation relations between the body and external space, and the spatial articulation actions that derive from it, remain altered. In another type of phenomena, we should distinguish the disorder in writing (agraphia) from the disorder in drawing. Writing is a more automated activity, and the second is more free and constructive. It is in the constructive action where the spatial defect of praxis is more noticeable and persistent, being the constructional apraxia the most persistent defect.

In summary, the most genuine form of left-right disorder corresponds to a general disorder of insufficient differentiation in spatial orientation. This can be observed in praxis as well as in the visual scene, in writing, drawing, etc. The disorder is analogous in nature to the orthogonal disorder studied in vision, consisting of various forms of apractognosia, or simply of a deficit in the formation of the spatial schema. The more complex the test or experimental situation is, the more this deficit tends to appear, being practically absent in elementary or immediate situations. The comparison between situations, complex relations, constructions, writing, transitive actions projected to external space, etc., serve to demonstrate the deficit in the spatial schema. In these actions there is spatial confusion or insecurity of choice because the difference between

orientations is blurred and therefore the mistakes are ignored. All these disorders are highly objective as they are manifestations of praxis that are visible to the observer.

The fact of not distinguishing between orientations constitutes what we may call "neutral praxis," which, due to its character of functional reduction, has perhaps a certain analogy with the illusion of the neutral model; in both cases, there is, as always, a reduction in sensory organization. In neutral praxis, the praxis model is modified losing its specific orientations, becoming subordinated to the circumstances of the action proposed to the patient.

27.4.2.b) Spatial orientation and egocentric-allocentric action

Concerning spatial orientation, the above tests do not show all of subject M's disorder, i.e., all of the reduction of his action space. To show this clearly, it is enough to change the position of the subject in space, either to ordinary positions (e.g. lying down) or to very unusual ones (the body upside down). In this way it can be observed that when modifying the position of his body, spatial orientation is linked to his own body, that is, it is exclusively egocentric, thus, 'up' is always towards his head and 'down' is towards his feet. Hence, there is *egocentric prevalence* in spatial orientation.

If subject M is in the inactive state and seated in an articulated armchair, it is very easy to place him in a completely horizontal position without him hardly noticing the change. This is done with the subject M's eyes closed, but sometimes also with his eyes open, and in this case he still believes (or has the illusion) that he is in the initial position or rather in the habitual position, since at his functional level, he does not perceive bodily sensations clearly enough. When asked to point with one hand to a certain direction (up or down), he does so with reference to himself, i.e., egocentrically. If the instruction is to point toward the ceiling, he directs one hand toward the head as in Fig. 27.4. Up and down are always in relation to his head and feet respectively, whatever the position of the body in space. Instead, with facilitation by strong muscular effort, he immediately corrects the defect, acquiring the normal allocentric orientation where the directions in space are no longer linked to his own body and become fixed and independent. It has already been mentioned, in Sec. 16.2 in Vol. 1, that the patient of Siekmann (1932) behaved similarly with his eyes closed.

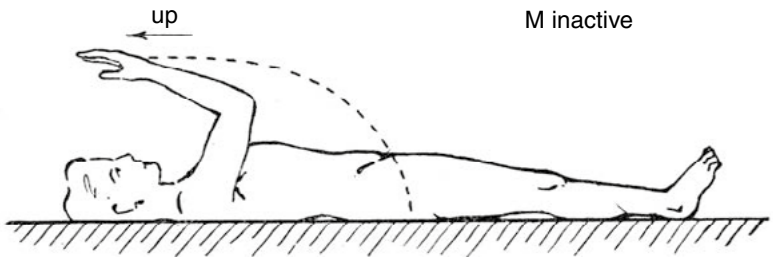

Figure 27.4. Prevalence of egocentric orientation. When subject M is in a horizontal position, with eyes closed, and is asked to point upward, he directs his hand toward his head. Likewise, downward is towards his feet. With facilitation by strong muscular effort, he corrects immediately.

The mentioned egocentric behavior was already observed in patient M at the beginning of being examined in 1938, shortly after the finding of his disorders of visual inversion and of the orthogonal function, all of which led to a general examination of his spatial visual orientation, as described in Sec. 16.2 in Vol. 1. Years later (in 1946) he presented similar behavior under the same experimental conditions.

> Orientation changes may vary according to the circumstances of each test. For example, in an armchair with a movable backrest, the subject can be reclined backwards without hardly noticing it, even with his eyes open. In this situation, if he sees the ceiling well and is asked to point upward, he does well. However, until he gets up and leaves the chair, he does not realize that instead of sitting, as he thought, he was lying down.

The lability of spatial orientation and the predominance of egocentric orientation can appear in special situations although close to ordinary life. For example, when patient M is at the top of a staircase, close to the ceiling, he may take his proximity to the ceiling (where he can lean) as a wall, and the far wall in front of him as the ceiling (Sec. 16.2 in Vol. 1). Here there is first a situation of chaotic orientation and finally a predominance of egocentric disposition (e.g., what is next to the body cannot be the ceiling or 'up').

If the subject is placed, by means of a tilting table, with the body vertically inverted (head down), trying to prevent him from perceiving the situation (eyes closed), he still admits that he is in a normal upright position as long as he does not apply facilitation by enough muscular effort. Moreover, if in this situation of body inversion he is asked to walk, he may at first believe that he is walking on the ground, seeming to him that the ground is soft (see Sec. 23.5.1). But with the movements (which iteratively activate the body schema), he realizes the situation and believes that after walking a distance he has been turned upside down without him realizing it. He does not know if he was really walking, he thinks he was, although considering that he was not moving, he also admits that he had taken a few steps without moving from the place. Such a confusing perception at the beginning of walking should not come as a surprise after what has already been indicated in Secs. 9.1.4 and 9.1.5. As for the actual inversion of the body, this is fuzzily replaced by the normal position, as in other tests in which the position was less unusual. Concerning the vestibular factor in this patient, see below.

Another experience of egocentric orientation, also with the body objectively inverted, is that described in Sec. 16.2 in Vol. 1 and which can be illustrated by Fig. 27.5. When the patient, being in the inactive state, is placed vertically upside down, a cardboard arrow 10 cm long pointing upwards is shown to him. The arrow is perceived correctly because it is in near vision, but he says that it is pointing downward (toward his feet) because of the egocentric reference. Instead, when implementing facilitation by intense muscular effort, he immediately describes the situation correctly: arrow upwards and the own body downwards, the normal allocentric orientation being established. Thus, as soon as the perception of the body schema

in space has been possible, the reference system has changed from being himself to being in external space.

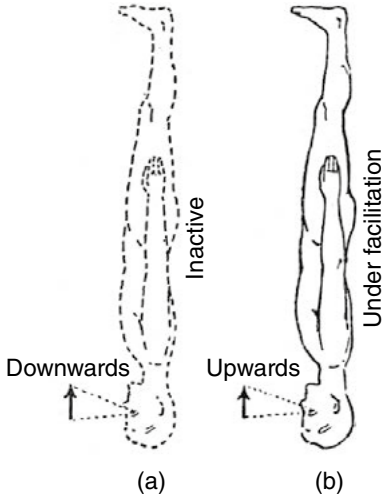

Figure 27.5. Egocentric orientation and allocentric orientation, according to changes of reference. (a) Patient M inactive: the body schema is not perceived (dotted line), so it cannot be localized in space; the clearly visible arrow is perceived as pointing downwards *egocentrically* (towards the feet). (b) Patient M under facilitation: the body schema is perceived correctly, inverted in space; the arrow is also referred to external space and is perceived pointing upwards, i.e., *allocentrically*.

The lack of connection with external space and the consequent predominance of the egocentric reference appears in many other situations already mentioned (Sec. 16.2 in Vol. 1). Thus, if subject M looks at the ground while walking, what seems to him to be moving is the ground, at least during the first part of the gait. Sitting on a swivel chair rotating moderately, it is the visual scene that appears to him to be moving while his own body appears to him to be at rest. These phenomena disappear if the subject applies facilitation by means of suitable muscular effort. Of the same category is the Aubert (1865) phenomenon [Sec.13.5 d) and 16.2 in Vol. 1] which in our cases is pathologically present with light (in normal subjects only in the dark). It consists in the fact that when the head is tilted, objects are perceived as deviating from the vertical. In subject M, the deviation is in the opposite direction to the movement of the head, and the greater the tilt of the head, the more pronounced the tilt of the object.

In all these tests, egocentric orientation is the effect of the blurring of the body schema, due to the more or less inactive state (Fig. 27.5), thus suppressing reference to the surrounding space. The possible activities in this state must then necessarily refer to the residual model, which gives no reference other than to itself, and therefore the directions of space are linked to himself. Any change of the body in space is ignored because it is not perceived, and instead there is the sensation

of a habitual situation (an "illusion of neutral position"), i.e., the *egocentric prevalence is a consequence of referring to the neutral model.* Under such conditions, if the subject is asked to point upwards, he will always point towards his head even if he is lying down or upside down, and he will not mind doing the walking action, believing at first that he is doing it normally. When walking, the ground appears to move, and when sitting on the swivel chair, the visual scene appears to move. In fact, the images of the objects move on the retina in these cases, but the inconsistency of the body schema does not inform about the displacement on the ground or about the rotation of the body and therefore the body is considered motionless (kinetically neutral). Thus, the visual scene that moves with respect to the body is perceived as moving (induced movement). The normal references have changed, and as the subject is isolated, the egocentric orientation is established. Finally, the same happens in the pathological Aubert phenomenon, which differs from the normal Aubert phenomenon only quantitatively, according to the interpretations of Müller (1903).

> According to Müller (1903), if a luminous line is observed in the camera obscura with the head tilted 90° to the right, the line appears generally tilted at the top to the left, which is the phenomenon of Aubert (1865) of type A. There is a type EA, where with less tilt of the head, the deviation of the line is towards the same side as the head. The position of the perceived image is influenced by both the visual condition and the head position. It should be noted that the subjective localization of the head may be different from the objective position (neutral deviation), and that the subject forgets that his head is tilted, etc. All these factors give rise to type A, as a result of the egocentric orientation.
>
> This type A is the one that occurs in subject M when tilting the head; the visual scene is deviated to the opposite side (here the phenomenon of inversion by asynchrony does not intervene because the vision is clear and there is good luminosity). As we said, in the normal subject this phenomenon only occurs under special experimental conditions (camera obscura, special attention, etc.), and in a very attenuated form compared to our injured patients.

Among other types of allocentric alteration, of a somewhat different type from the previous phenomena, it is worth remembering experimental autotopagnosia, already mentioned in the study of vision (see Sec. 16.2 in Vol. 1). In subject M, one of his hands is taken and passively moved from side to side in front of his eyes under appropriate conditions to obtain inversion of the direction of movement (brief movement, at some distance and with weak illumination). If he is then asked to grasp with the other hand the one that is moving, he tries to do it in the opposite direction to the real one, and uselessly tries to grasp in empty space. This experiment, besides objectively demonstrating the phenomenon of inverted vision, causes disorientation about one's own body (autotopagnosia of a very special type) and about external space, with the consequent disorder just described. This is due to the various anomalous phenomena in the visual-tactile multisensory complex: the subject's own hand moving passively in front of his sight is visually perceived in the opposite direction,

and the slight articular movement of a single oscillation is not perceived at all; in such circumstances there is only visual information, and now it is inverted.

> In comparison with the phenomenon of egocentric orientation, one could consider a type of "super-egocentric" orientation due to the effect of inverted vision (a much more profound orientation disorder). This type occurs in the body schema during *subjective* deviation in walking and other anomalies, such as the sensation of the body going down a slope (see Sec. 25.1.5). It also occurs, albeit *objectively*, in the phenomenon of inversion in praxis, in which proprioceptive inversion of the body schema induces an error in the praxic impulse.

To complete this topic on spatial orientation, we must comment on an aspect related to vestibular excitation. In the tests performed with patient M, a swivel and articulated chair or simply a rocking chair has been used, the patient being comfortably seated, leaning on the backrest, and in the inactive state. The body is made to rotate on itself or to oscillate in the forward-backward direction. The following is then observed. With a half-turn or full-turn clockwise rotation, the subject feels a brief counterclockwise thrust, and vice versa. With a single, wide and forceful oscillation from front to back, the subject feels a brief push forward, and vice versa. The thrust in the vertical direction has not been tested for lack of a suitable device, but similar inversion phenomena should also be present. Under facilitation by maximum muscular effort, inversion has not been obtained even with the slightest perceived oscillation.

All these tests, which admit other varieties, show that the body impulse to be picked up by the vestibular system is perceived in different directions (and therefore also in strength, amplitude, etc.) according to the state of central activity (inactive or with facilitation), i.e., the stimulus picked up is inserted differently on the body scheme, producing inversion of the impulse in the inactive state and impulse of practically normal direction under facilitation.

Trying now to pass judgment on the vestibular action on spatial orientation, both in these tests of body impulse and in the previous ones on changes of body position in space, it should be noted first of all that in the Schneider patient of Goldstein and Gelb (1919) these authors came to suppose the existence of a disorder of the labyrinth function in view of the patient's inability to perceive very large changes of body position in space. They also think that there is undoubtedly a cerebellar disorder. As for the latter, it has already been said (Sec. 25.2.4) that it is unfounded, and as for the vestibular disorder, it should also be excluded, as explained below. In preliminary tests of vestibular chronaxie, subject M has shown no difference from the normal subject (neither increased chronaxie nor facilitation effect), and the vestibular orientation mechanisms must be considered completely normal. However, only in the case of a sufficiently developed body schema, a correct vestibular effect is possible since this effect depends on the vestibular connection with the body schema. Thus, if there is no body schema in the tests, the vestibular effect plays no role. However, depending on the intensity of vestibular excitation, the altered schema can be recruited to varying degrees, as observed

in the impulse tests. In the inactive state, if the impulse is weak there is no effect. If the impulse is strong, there is sensation of impulse in the reversed direction because the vestibular action is connected with a body schema in a spatial inversion stage. With facilitation, the body schema is sufficiently developed and the connection with the vestibular system is normal, the vestibular system remaining unchanged within its normality.

According to the above we can conclude that the normal body schema and external space are connected to each other, and that allocentric orientation and the corresponding action require a fully developed schema. The egocentric orientation is the result of a reduction of the body schema, and the references are built on the neutral model. It is thus a "spatial neutralization," as is the rule in many other functions of a similar nature (segmental postures, orthogonal figures, etc.).

27.4.3. Dyspraxia due to instability of the praxis model

Finally, we shall study the praxis model with some functional development in order to consider common dyspraxia symptoms. The instability of the body schema and the lability of the spatial schema result in a reduction of "praxis behavior" in the sense that voluntary actions are performed according to a diffuse plan. Two pathological aspects can be considered, the defect in the *initiation of praxis* and in the *constructional praxis* (fragmentation of the action, simplification, etc.). Thus, the lack of unity of action, typical of ideational apraxia and ideomotor apraxia, becomes evident.

27.4.3.a) Defect in the initiation of praxis

The initiation of voluntary action constitutes a very significant aspect, since it reveals by itself the state of the body schema, as previously studied, especially in Sec. 27.2.2. As we know, for the state of total inactivity there is no possibility of selected voluntary action, resulting in akinesia secondary to the loss of the body schema. With some activation of the schema, the action arises slowly, after a long latency period.

The Schneider patient of Goldstein and Gelb (1919) presented above all great difficulty in initiating any movement that was asked to him. If he was asked to move a certain limb or to perform any other precise movement, he was at first completely stumped. As described by the authors, the patient used to repeat verbally what he was asked to do, as if he wanted to have it clearer; then, it was observed that in order to prepare himself to perform the action, he performed movements of the whole body (head, neck, legs, etc.); then, the movements were gradually restricted to the limb to be moved until finally only the requested movement remained. What the patient was doing was not really complying with the indicated request, i.e., he was not performing a voluntary movement with a given limb, but rather this movement appeared as a final effect.

> Since the patient lacked spatial appreciation of his own body, he had to start by finding the limb that had been indicated to him. Likewise, if he had been instructed to

draw a square or a circle in the air (a motor action known to him), the beginning was particularly difficult until he was able to orient himself on the limb to be moved and on an external plane, and then the rest of the action proceeded with relative ease, in a very automatic way. Only the initiation caused difficulties.

All these results refer to tests with the eyes closed. However, the process was essentially the same with eyes open since the patient had to search for the limb in more or less the same way as with eyes closed, only the action proceeded more quickly and accurately. The aforementioned authors naturally distinguish between movements of ordinary life, however complicated they may be, and voluntarily selected movements that respond to a given order. In the former, the action is executed quickly and easily even if the movements are complex and the eyes are closed or not looking (e.g., taking out a handkerchief and blowing the nose), since these movements are automated by habit. As for the difficulty described in the second type of movements, especially in their initiation, the mentioned authors explain it by the loss of visual influence, stating that the patient's behavior undoubtedly shows that visual data in the form of visual representations or perceptions are necessary for the realization of such movements. The authors leave open the issue about the nature of such data, insisting on the fact that visual data are particularly necessary for the initiation of movement whereas they are of little or no significance for the subsequent activity.

We must point out that certain praxis disorders of the Schneider case (e.g., in the initiation of praxis) coincide with our observations in the wounded patients, however we differ completely in the interpretation, which we make in a simpler form. For us, the visual influence does not count at all, i.e., it is not essential (congenitally blind people do not suffer from dyspraxia or difficulty in initiating conscious and selective movements). This point of view has been expressed several times throughout this book. In the Schneider case we would only have the loss of the body schema due to a lack of tactile organization, which hinders the initiation of voluntary movement, being necessary for the subject to activate the schema by means of muscular twitches for example, which is nothing more than applying a non-specific nervous reinforcement (facilitation). The difficulty is greater when trying to initiate the movement since it starts from a "dead point," although there may also be difficulty in the subsequent development of the activity (praxis discontinuity), as we shall see in our cases.

In subject M, in addition to the aforementioned initial difficulty (considerable latency in initiating the ordered movement), it should be noted that the first movement is usually particularly strong; the action is initiated with abrupt and accelerated impulse in most of the tests. This should be attributed to the deficit of proprioceptive control or kinesthetic sensitivity, the subject having the need to make a vigorous movement to perceive it in some way in the initial stage, which is the most affected.

In general, in the requested actions the movements are somewhat clumsy and rigid. The latency time for the semi-inactive state is five to seven seconds to initiate the movement. The action is influenced by insufficient proprioceptive control (resulting in a certain dystaxia in the initial movement) as well as by dyspraxia (inabil-

ity to combine, sequence and unify the various movements), due to the weakness of the body schema.

27.4.3.b) Defect in constructional praxis

In the study of actions, it is necessary to consider their diversity due to the degree of automation. It is known that very common and almost involuntary actions are correctly executed by apraxic subjects, since they are performed automatically and instinctively. Dyspraxia, instead, occurs in voluntary actions that respond to a specific command. Within these actions some are more habitual and automated than others and also have different degrees of complexity. The action of greeting is rather automated and rather simple; drawing or writing in the air is less automated and more complex; transitive actions, as well as the construction of movements, learning, etc., are devoid of any automatism and are also complex sequences of movements. It is easy to understand that defects in praxis are better manifested when the automatism is lower and the need for a construction is greater, as illustrated in the following tests in patient M, from lesser to greater difficulty:

i) A first type of voluntary actions shows the most elementary tests, of highly automated action and very simple movements, in which subject M shows no defect other than an initial latency and a certain discontinuity of action only in the most complex test.

A highly automated action is the military salute. In the semi-inactive state or with weak facilitation to activate the body schema, subject M takes five seconds to "get ready" but his salute is correct although the initiation of the movement is performed with a sharp and accelerated impulse which is typical in many tests. At the end of the action, he only has felt an arm movement of about 30°, as if he were raising his arm less than horizontal. This same test under facilitation by strong muscular effort, in addition to practically suppressing the initial latency, produces a much smaller reduction in the sensation of movement, since the movement felt reaches about 150°, compared to the 180° of the real movement of the salute. It is remarkable that the considerable reduction in the movement felt in the inactive state hardly disturbs the execution of the movement, since it is a rather habitual (automatic) action.

Similar results are obtained with the test of sending a kiss with the hand. In semi-inactive state the latency time is 5 seconds and correct execution. Under facilitation the latency time is 1 second.

A slightly less automated test is to wave goodbye in a semi-inactive state. After a few seconds, the subject raises the arm and a little later moves one or more fingers of the hand in a clumsy and rigid manner. Sometimes he moves all his fingers abruptly as if to catch something in the air. He only feels that he raises his arm a little, as in the previous test, and does not feel the movements of the fingers. Under facilitation, the latency is reduced to a quarter of that of the semi-inactive state, and although the movement is more correct, there is still some rigidity, and finger movements are perceived very slightly. In this test, in addition to other defects, a certain praxis discontinuity al-

ready appears (certain time between raising the hand and waving it), i.e., deficit in the unity of action, which becomes important in other tests.

ii) Other tests on less automated actions, such as drawing or writing in the air, reveal more clearly the various aspects of dyspraxia (spatial dyskinesia, mirror writing, etc.):

> For example, subject M is asked to draw a cross in the air, being in a semi-inactive state (usual state of weak facilitation). He takes about six seconds to start and does so with some ataxia and drawing only one angle (spatial dyskinesia). In addition, he does not feel the movement while drawing. With facilitation by strong muscular effort, he takes only two seconds to start and draws the cross correctly with the index finger, but the felt movement is about one half or one third of the actual size. About writing numbers in the air, it has already been dealt with in Sec. 27.4.2. Without facilitation by muscular effort there is a constant tendency to produce mirror writing with the left hand, in addition to other errors (simplifications such as in the drawing of the cross). The automatism plays an important role in writing, failing rather the orientation of the writing.

iii) Difficulties increase in actions devoid of habitual automatism and requiring above all a good spatial scheme such as combined transitive movements. In this situation, *praxis discontinuity* due to a deficit in the unity of action is fully displayed, as described below:

A transitive action is to bring the left hand up to his right ear. If he is almost inactive he has considerable difficulty in doing this. After a certain time, he raises his hand as instructed, but only to the middle of his chest, pauses there for a moment and then undertakes a more lateral movement, then tilts his head to favor reaching the indicated ear and goes groping along his face until he reaches the ear. In total, preparation (latency) and execution, he takes about twelve seconds, whereas with facilitation by muscular effort he takes only one second, performing direct movement without pauses.

Another test of the same type, in an almost inactive state, is to place the left hand on the right shoulder. The subject first raises the left hand toward the chest and stops there, then moves the head a little or slightly the shoulder as if to orient himself. Next, the stopped hand is abruptly directed toward the shoulder, and finally he corrects by trial and error the wrong positions that might have resulted.

A very illustrative test is that of grasping the ears by means of crossed hand movements, in a semi-inactive state, as shown in Fig. 27.6. From the moment the subject is asked to perform this action, he takes about 15 seconds to perform it, and does not initiate it until after 7-8 seconds. The action is broken down into a series of successive phases as follows. Starting from the natural position, with both hands lowered along the sides of his body and after the aforementioned latency, in a first attempt he raises both hands to his chest, pauses, and in a new impulse crosses his hands and raises them almost to his face; there is another pause to try to grasp an ear with the opposite hand, for which he first moves his head bringing an ear close

to the hand. Next, he tilts his head to the opposite side to bring the other ear closer to the other hand. This results in a total of five or six independent phases to perform the whole action. It seems to be impossible for him to reach both ears simultaneously no matter how hard he tries. At other times, in a more clumsy way, he slides his hands crossed over the opposite arms, advancing little by little up to the face.

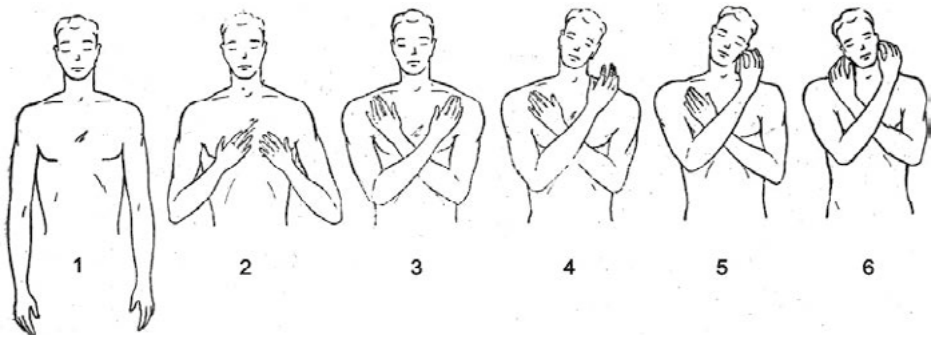

Figure 27.6. Typical praxis discontinuity in transitive actions carried out by patient M. In his habitual state (almost inactive), the action of grasping the ears with the opposite hands presents an initial latency of seven seconds, and also a breakdown into five or six independent phases over another seven seconds. Under facilitation by strong muscular effort, the action is direct and brief.

All these tests can be performed with the eyes closed or open, and in the latter case the results do not vary essentially, although they may be somewhat faster. Praxis discontinuity in patient M, and also partly in patient T, was observed already in 1938, before the discovery of muscular effort facilitation and other dynamic phenomena. At present it is also evident if the patient suppresses a large part of muscular effort facilitation. Such a fragmentation is very characteristic of the disturbance of schema function of any kind (visual, tactile, etc.). However, as far as praxis is concerned, it is not highlighted in Goldstein and Gelb's Schneider patient (Goldstein and Gelb 1919), and the authors focus the disorder on movement initiation, as stated above.

Sometimes a certain perseveration is observed in some tests, but it is not frequent and could be regarded as a misunderstood order. Thus, if subject M has to perform a transitive action and at the same time stick out his tongue, in a following test of a different type and which he executes well, he also sticks out his tongue even though he has not been ordered to do so. This is an example of a fuzzy state (difficulty of functional selection), a classic feature of praxis disorder.

iv) The defect becomes very flagrant in those actions that require a previous spatial scheme, a construction or "pensée spatiale" (Lhermitte and Trelles 1933). In the absence of this, partial actions and indirect attempts are produced, as shown in the following test:

In the common two-handed "thumbing one's nose" test, subject M in 1938 (before discovering facilitation by muscular effort) first gradually joins the hands raised in the air and contacts both thumbs; then, groping with the palm of the

hand, he manages to join the thumb of one hand with the little finger of the other; finally, having his hands in this way, he brings them close to his face until he touches his nose with one of them. At present, the result depends on the facilitation applied by muscular effort. When he is in an almost inactive state, the behavior is as described, although it may vary somewhat, taking about 12 seconds for the whole action. After 7 seconds of receiving the order, the subject first places one hand extended in good position, but over the mouth, then the other hand, also extended, tries to contact the little finger, but does so with the ring finger of the hand already positioned; finally, he succeeds in finding the little finger, and then shakes the fingers of both hands, but one of them is still over the mouth, and they are not in the same sagittal plane, the first one is medially and the second one is to one side. With facilitation by maximum muscular effort, the subject places the first hand on the tip of the nose correctly from the first moment, but he also places the second hand on the nose in the same way (simplification). However, he corrects immediately by moving the second hand and placing it in line with the first hand.

v) Other tests are very difficult because they consist of actions that are totally new to the patient, thus entailing a true learning process. They offer great difficulty even with maximum facilitation, at least at the beginning, and also in the much less affected patient T. Thus, neither of the two brain-injured patients (M and T) can, even with intense facilitation, imitate the "flying butterfly" (crossed hands hooked by the thumbs, showing upward the back of the hands and waving the fingers). Only after being taught many times and after multiple attempts, they finally succeed, always under facilitation, in performing the action. If once the action is learned and executed, they are asked to perform it without great facilitation by muscular effort, the action results very imperfect, but applying maximum facilitation it becomes correct.

As for the state of maximum facilitation by muscular effort, although it notably improves the praxis model, it does not achieve full normality. As we know, full normality is also not achieved for the simpler activity of elementary excitability, and if the functions are complex, the correction by facilitation is naturally less efficient. Despite the fact that in patient M there is an important difference in praxis between the ordinary state (weak facilitation) and the state under maximum facilitation by intense muscular effort, it has been observed that in the latter state there are certain errors in the action, as described above, although they tend to be corrected immediately and spontaneously. The lack of agility is much more pronounced when the tests are totally new or very complicated where learning is necessary, as in the "flying butterfly" test. In short, the spatial schema or "pensée spatiale" is not entirely achieved by maximal facilitation. The same happens in other types of schema, and was already mentioned in the visual schema (see Sec. 15.1 and Fig. 15.2 in Vol.1). Thus, only by trial and error and in an indirect way can the activity be performed correctly or, in any case, in an approximate way.

Summarizing this third subsection on the praxis model, it can be said that the praxic reduction in actions of a certain complexity reveals a disorder rooted in the elementary function of the local space (somatic model, postural model) and thus compromises the development of the spatial schema, that is to say, of the of the action plan or constructive action. Therefore, this reduction in praxis is above all an

inability in constructing, and the characteristic phenomenon is the fragmentation of the action.

27.4.4. Theoretical summary

We highlight below the most significant aspects of the praxis model. The multiple and varied phenomena of disorganization of the praxis model participate in both apraxia and agnosia, and although one or the other predominates depending on the type of phenomenon, they are always closely related. Thus, the disintegration (reduction) of the action space is part of the general syndrome of apractognosia of classical brain pathology.

Different levels in the degree of spatial organization of the praxis model have been observed, corresponding respectively to severe impairment (severe asomatognosia and somatic inversion), moderate impairment (disruption with the external space), and somewhat milder impairment (praxis discontinuity of voluntary actions that require certain development). The phenomena corresponding to these stages result from internal factors (inversion, neutral image) and external or adaptation factors (reorganization), and depending on their contribution determine the character of the 'spatial reduction.'

It is worth highlighting some general aspects of this spatial reduction. The reduction is mainly manifested by functional *undifferentiation*, more or less pronounced depending on the levels of organization considered. At the lowest level of asomatognosia, the model is replaced by the neutral model. At a higher stage, the types of lateral confusion and undifferentiation appear. Apart from changes in the movements, we find first of all, as a profound alteration of the praxis model, a constant primary lateral inversion in simple tests. It is a primary phenomenon resulting from spatial inversion. In more complex tests, lateral confusion is observed due to lack of lateral differentiation. It is a more frequent phenomenon resulting from the lability of the left-right organization, similar to the so-called orthogonal disorder in vision. Finally, with less deficit, the confusion and undifferentiation is no longer lateral but only on the fingers of the same hand. Egocentric orientation that replaces normal (allocentric) orientation, as well as praxis simplification of voluntary actions and praxis discontinuity, also constitute a spatial reduction, that is, a functional undifferentiation.

Another general aspect of importance lies in the phenomenon of the *neutral illusion* (spatial neutralization of the praxis model), which plays a more fundamental role the greater the reduction of the model. Neutral illusion therefore plays a significant role in asomatognosia, and very especially in egocentric orientation; it even constitutes a feasible hypothesis for mirror writing in left-right undifferentiation. As already mentioned when dealing with the postural model, the neutral illusion lies in the predominance of the most habitual attitude of the body. When the organization of the body is labile, the tendency to the most stable or preferred situation appears spontaneously, which can be none other than the habitual (neutral) model. This is the general phenomenon of reduction of meaning in sensory organization, similarly to what we have observed in vision in orthogonal disorder (any orientation of draw-

ings is good or natural) or in the understanding of schematic drawings (change to a simpler interpretation, etc.).

A third general aspect is the *unawareness of the defect* in spatial perception, which coexists with any kind of functional reduction, leading naturally to a limitation of the organization. In praxis inversion as well as in egocentric orientation, lateral undifferentiation, praxis discontinuity, etc., the examined subject does not perceive any defect. On the contrary, the corresponding activity seems to him the most appropriate and normal. This very general aspect has been mentioned repeatedly throughout this book.

Finally, it is possible synthetically to understand the disorders of the praxis model as a *constructional deficit* of the action space, which is the most representative defect of the alteration of the schema function in general. Naturally, this defect is particularly evident in functions of certain complexity, and so it is both in left-right disorganization and in the failure of allocentric orientation and organization of the external space, but above all, it is evident in the development of somewhat complex voluntary actions. Thus, the typical phenomenon of praxis discontinuity (fragmentation of the action) is a consequence of the reduction of the action plan. Since there is no construction of the whole, the action is developed by juxtaposition of fragmentary effects. It is the same situation as in simultaneous visual agnosia, in which the comprehension of the scene as a whole is lost and replaced by partial aspects. The degree of organization is lower, and it could be said, considering the "pensée spatiale," that there is no speech but adjacent sentences or loose words. The most constructive activities are always the most affected and the last to recover. To conclude, the schema (here, spatial schema) can once again be defined as a construction by reorganization of elements in the sensory field (here, action space), which essentially means a functional growth as we shall see below.

27.5. GRADUAL SERIES IN APRACTOGNOSIA AND FUNCTIONAL GROWTH

After having studied the multiple manifestations of the body schema in our brain-injured patients, we shall address the nosological and physiological meaning of such manifestations. In presenting above the somatic model, the postural model and the praxis model, we have described phenomena that constitute a gradual series of disorders in apractognosia. This series indicates a functional unity, despite the autonomous entities of classical brain pathology.

> Among the disorders with predominance of apraxia are mainly: innervatory apraxia (Kleist 1907, 1911), the different varieties of apraxia of Liepmann (1900, 1920), constructional apraxia (Kleist 1907, 1911; Strauss 1924), with its close type, visual ataxia (Poppelreuter 1917) and agraphia. Of a rather agnosic character we can mention: autotopagnosia (Pick 1908 b, 1915, 1922) and its varieties [anosognosia (Babinski 1914, 1918; Anton 1898, 1899, Redlich and Bonvicini 1909, 1911), the different types of Stockert (1934). etc.], asymbolia for pain (Schilder and Stengel 1931/1932), left-right disorientation (Pick 1908 b, 1915, 1922; Bonhoeffer 1923, Pötzl 1924) and the complex syndrome of finger agnosia (Gerstmann 1924, 1927, 1930, 1931, 1940).

There are also some more recent cases included in the denomination of 'apractognosia of spatial structure,' according to the observations of Ranschburg and Schill (1932), Schlesinger (1928), Zutt (1932), etc., comprising disorders of the visual system and tactile system, which in certain aspects are very similar to those of our cases.

It should be noted that in all these disorders the lesions are "central," involving the supramarginal and angular gyri or their surroundings, i.e., the classic territories of apraxia and agnosia, either cortical or subcortical.

Many of the manifestations described as individual nosological entities can be found throughout the series of phenomena presented by our brain-injured patients, which argues very little in favor of the existence of such individual entities, and rather suggests different degradations of a same and single process. Within the wide range of phenomena in our cases, we find first of all that the so-called 'innervatory apraxia' is closely related to the disorder in our cases described in Sec. 27.2.2 and the praxis initiation defect [Sec. 27.4.3. a)], although the interpretation is very different from that of Kleist (1907, 1911), since in our cases it constitutes an elementary movement difficulty due to a severe deficit of the body schema. We also think that asymbolia for pain corresponds to a very elementary stage of the body schema. In the only case described to our knowledge, it is already interpreted as a disorder of the body schema. In our opinion, such a symptom, which seems to be very poorly defined, would involve both alterations of localization on the skin (disorder of the somatic model) and alteration of the action plan (difficulty in reacting to provoked pain), perhaps in relation to defects in praxis innervation.

Schilder and Stengel (1931/1932) report a case of dissociation between body schema and pain perception. Their case was that of a 73-year-old woman with cerebral arteriosclerosis who after a stroke presented sensory aphasia, mild apraxia disorders, some motor disorder, and asymbolia to painful stimuli consisting of a lack of reaction to painful stimuli, although pain perception did not seem to be altered.

Within the insufficient precision of such a phenomenon, we could provide an observation of this nature in 1935 in a woman with sensory aphasia, apraxia and, seemingly, asymbolia for pain. She did not understand most verbal and mimic commands (e.g., the invitation to be silent), presented intense logorrhea and paraphasia, was unable to imitate any action (in the two-handed "thumbing one's nose" test she waved her hands separately in the air), wrote indistinctly with the fountain pen open or closed, as well as with a pin which at the end seemed to recognize it, did not know how to handle scissors, nor cut paper, etc. With a strong and repeated prick she said: "It pains me ... it hurts me a lot." This *contrasted with the lack of defensive movements, absolute passivity and no opposition.* Her mood was euphoric.

As for the manifestations of the type of autotopagnosia, asomatognosia, left-right disorientation, etc., they are in fact different degrees of elaboration of the body schema in the process that goes from the simple somatic model to the coupling of the body with external space, passing through stages such as egocentric orientation, hardly known until now. Thus, it can be seen that these are not nosological entities

but modalities of a same function that we have been able to study in our brain-injured patients according to the conditions of central excitability.

Analogous considerations can be made with respect to apraxia phenomena, ranging from ordinary ideational apraxia to the subtlest types of constructional apraxia and visual ataxia. These are different stages of schema construction between the most automatic level and the least automatic level, as is already indicated by Sittig (1931), as well as by Zutt (1932) with respect to the sequential order of praxis destruction [see Zutt in Sec. 27.4.2.a)].

Special attention should be paid to finger agnosia in the syndrome of Gerstmann (1924, 1927, 1930, 1931, 1940), due to a lesion of the left angular gyrus, and which must be examined in relation to our cases.

In Gerstmann's syndrome, in addition to finger agnosia, there is usually left-right disorientation, agraphia, acalculia, and often there is also constructional apraxia and amnesic aphasia. In some cases there are also more widespread disorders, such as visual field defects, balance disturbances, etc. For this author, this syndrome constitutes a pure individuality, although he also recognizes that it appears as a recovery stage (residual syndrome) of more complex brain disorders. He tries to explain it as a selective disorder of the body schema, ascribed to the left-right orientation, and also as a disorder of the finger schema from which agraphia and acalculia would derive.

However, it seems to us more correct and simple to consider such manifestations as a broad apractognosia defect, as a deficit of the general schema function not only limited to the body schema. In particular, acalculia goes far beyond the body schema, even if one tries to associate aptitude for calculus and the handling of numerical signs with counting fingers. Even in the case of interpreting the syndrome more broadly than Gerstmann, as a defect of conceptual structure of space ["begrifflichen und schöpferischen Gliederung des Raumes" (Lange 1936)], which allows to understand agraphia and even constructional apraxia, there is always something else in acalculia that cannot be included in this conception, apart from the very common existence of amnesic aphasia that also escapes from such an interpretation.

As for the individuality of the syndrome and its functional independence, it is already questioned when considering that it can appear as a residual defect of an even more varied disorder, as mentioned above. Its unity is also compromised by the fact that even the most typical symptoms of the syndrome (finger agnosia, left-right disorientation, agraphia, acalculia) are not inseparable and some of them may be missing depending on the case.

About such an incomplete syndrome and the varieties of the syndrome, some cases from the literature deserve to be mentioned briefly. In the subject studied by Scheller and Seidemann (1932), there was only some left-right disorientation, diffuse apraxia and pronounced constructional apraxia plus dyslexia, hemianopsia, etc. A similar case is that of Zutt (1932), also lacking finger agnosia, and which undoubtedly must have presented orthogonal disorder of figures and letters, left-right disorder and very evident constructional apraxia, in addition to pure agraphia. In contrast, the case of Schlesinger (1928), in addition to some concentric reduction of the visual field, visual agnosia and various tactile symptoms, had moderate finger agnosia, constructional

apraxia of the own body, but no agraphia nor acalculia. In the case of Conrad (1932) with finger agnosia and aphasia, agraphia was very moderate. A case of Schilder (1931) with finger agnosia did not show left-right disorientation. It should be noted that the syndrome in question, more or less complete, can be characterized by more fundamental defects (aphasia, agnosia, apraxia, etc.). Thus, Conrad's case showed an aphasic trait with finger-naming disorder; in Schilder's and Lange's observations there was a certain agnostic-visual predominance; in Schlesinger's case, an apraxic predominance; and in the case of Wagner (1932), the underlying disorder was a severe autotopagnosia. Schilder (1931) distinguishes up to five varieties of Gerstmann's syndrome according to the predominant feature, admitting all of them as perfectly localizable in the brain: the typical syndrome in the angular gyrus; the apraxic type, forward in the supramarginal gyrus; the aphasic type, towards the language area; the visual-agnosic type, towards the occipital area, etc.

Apart from the cases related to the brain dynamics described in this book, there are other patients that we have had the opportunity to examine, some of which are described in Sec. 27.4.2.a), in which different modalities of the syndrome in question are observed according to the predominant feature (rather apraxia in the first case, severe autotopagnosia in the second, and a very typical syndrome perhaps with some agnosia and amnesia in the third case). Also noteworthy in our cases was the late onset of the syndrome, which was rather a residual stage of a deeper disorder (of greater disorganization) of the schema. On the contrary, in another brain-injured patient (not mentioned above), who along with tactile disorders manifested from the beginning a moderate Gerstmann's syndrome, this disappeared in a few days. Thus, there are all kinds of degrees depending on the different localization of the lesion and the stage of the recovery process. This leads to admit a certain degree of apractognostic disorganization rather than a pure individuality of the syndrome.

One might expect, based on classical empirical data, that the M and T cases studied throughout this work would correspond in some way to Gerstmann's syndrome, since they have a lesion of the angular gyrus or close to it (see Sec. 2.1 in Vol. 1). Indeed, when subject M was first examined in mid-1938, he had the symptom of finger agnosia [see the end of Sec. 27.4.1.b)], constructional apraxia, certain diffuse disorders of agnosia, severe visual field constriction, etc. Likewise, subject T presented during the first months of 1938, several visual field disorders, tactile agnosia, constructional apraxia, very pronounced optic ataxia, agnosia for geometric shapes, etc. Thus, our brain-injured patients fulfilled to a certain extent what is usually established about a lesion in the angular gyrus or in the left parieto-occipital area. However, as established throughout this book, a lesion in a 'central' region of the brain gives rise to the 'central syndrome' (bilateral and symmetrical repercussion). In this central syndrome all sensory systems are equally involved and in all their activities, i.e., the entire sensory field is affected as a whole (for each of the systems, visual, tactile, auditory, etc.). The "classic" parieto-occipital syndrome is only the part corresponding to the disorder of the schema function in the central syndrome that we have characterized. That part can stand out more than other disorders either when the magnitude of the central lesion is small, or in larger lesions compensated

with facilitation by muscular effort. And if more extensive and more elementary alterations are then hidden, it is due, as we have repeatedly indicated, to the inaccuracy and quantitative insufficiency of ordinary examinations, which is what happened to us during the first examination of our two patients (M and T) in whom we were only able to detect agnosia disorders.

It should be noted that if a diversity of manifestations of Gerstmann's syndrome is admitted when the location of the lesion is slightly different, it does not contradict what has been established in the present research, since the type of dynamic repercussion in the central syndrome depends on the position of the lesion. Thus, the fact that apraxia predominates if the lesion is anterior to the angular gyrus, or that visual agnosia predominates if the lesion is posterior towards the occipital region, or that aphasia predominates if the lesion is in the inferior region of that gyrus, is perfectly compatible with what is established in the brain dynamics exposed here, since such cases would be paracentral (asymmetrical) syndromes.

Regarding the reduced schema organization in subject M, we now see better that it includes phenomena similar to Gerstmann's syndrome or related to it, such as some left-right disorientation, finger agnosia, occasional mirror writing as well as inverted writing, and considerable changes in writing under special test conditions (Fig. 16.3 in Vol. 1). In addition, subject M presents a more profound disorder of body orientation due to the primary phenomenon of praxis inversion, which goes beyond the described Gerstmann's syndrome.

To understand the physiological significance of the phenomena we are concerned with, it is necessary to consider the functional unity of the disorders, which leads to the establishment of the important concept of functional growth.

The gradual series of apractognosic symptoms is interpreted here from a dynamic point of view as stages of a unitary functional development. Therefore, the ordinary nosological individualities appear as fictions, resulting from a superficial clinical empiricism. Moreover, the schema function does not emerge as something different from the other functions (of a lower level); on the contrary, there is a perfect *continuity* between all of them. It is therefore no longer possible to accept the ordinary separation between "purely sensory" functions and "intellectual" functions (gnosis).

It is still noteworthy that in the usual classical brain pathology, all interest is focused mainly on the higher functions, and there seems to be hardly any other topics of interest apart from aphasia, apraxia and agnosia, i.e., schema functions, whereas the most elementary sensory and spatial phenomena (localization, shape, etc.) receive only brief attention and are interpreted superficially in a very simple mechanical way. Here in our study the opposite is true. We have first investigated the nervous excitability state (the sensory state) and then the spatial functions (localization, orientation, space, time, forms, etc.), that is, the structure of the sensory field, considered to be of the utmost importance. It is in this structure of the sensory field where the most fundamental and characteristic questions of the brain dynamics presented here are raised, and to which we have devoted the most attention.

All this means that, without ignoring the complexity of the higher functions, the importance of spatial activities (commonly called perceptions) must be recognized and put in relation with the schema function. Thus, if the schema is a construction

based on reorganization in the sensory field, this can also be applied to the most elementary function, for example, of the local sign, especially in the re-inversion process. Functional unity results from a single pattern of organization of the sensory field, organization that varies only quantitatively according to the complexity of the activities.

Such unit and quantitative variation are best expressed by means of *functional growth.* In this respect, we find in the body schema a natural continuity between the three types of model (somatic model, postural model and praxis model), which appear as *phases of growth of the model,* and strictly, as stages of growth of the tactile field. Moreover, we can say that such growth of the body schema is linked to the law of psychophysical organization of the spiral development of the sensory field. This law can easily account for both the most severe alterations corresponding to a residual field (somatic shrinkage, distortion of the model, praxis inversion, etc.) and the deficit in the organization of the higher functions, always linked to a certain reduction of the sensory field due to the overall alteration.

In short, gnosis or normal schema is the last stage of the growth of the sensory field, as a result of integration or recruitment of central brain mass. Such growth entails both an *increase in the sensory dimensions* of the field (intensity, space, time) and an *increase in functional complexity* (organization), i.e., a progressive individualization of functions (individualization is the result of organization, as indicated at the end of Sec. 22.4). It is clear that, within such a simple and general biological concept, the schema function is subjected to the general organizational characteristics of any other type of less complex activities, from which it does not essentially differ. All activity depends on the state of growth of the field. Thus, the characteristic constructional deficit of any agnosia implies both a reduction in dimension (size) and organization.

27.6. DIFFERENCE BETWEEN CORTICAL AND SUBCORTICAL LESIONS

The fundamental problems will be dealt with elsewhere, but now we must advance some brief clarifications in relation to the above. It should be noted that all of the above is based on the observation of our cases and refers to *cortical* lesions, i.e., to the cerebral cortex involved in the activity of the sensory systems and thus in the development of the sensory field corresponding to each system.

In contrast, *subcortical* lesions (of the deep white matter) seem to correspond to a very different behavior. They leave the sensory field intact with all its varied characteristics, and the sensory systems seem practically unharmed; instead, they maximally disrupt complex activities, i.e., complex functions of mixed sensory-motor character (handling, writing, speaking, etc.). There is then a radical contrast between the severe affection of these higher functional complexes and the normality of the sensory structures strictly speaking; not being here applicable the functional unity for the whole of it. Deep (subcortical) lesions in the parieto-occipital-temporal region give rise to serious behavioral disorders in the sense of classical brain pathology. At the same time the sensory excitability and physiological level for any type of sensory function is normal, there is also no primary motor disorder but the connection

between the sensory and motor domains is hindered, leading to a considerable impairment of the actions in which the anterior motor region and the posterior sensory region must intervene in unison.

At first glance, the above behavior may appear to be a major limitation to the fundamentals of the brain dynamics presented here, but although it precludes a generalization to all types of cases or brain lesions, it does not undermine the dynamic principles as long as the difference between cortical and subcortical lesion is understood. The first type of lesion (cortical) is related to the *disorganization of the sensory field* due to altered nervous excitability (excitability deficit, summation permeability, desynchronization, spiral reduction by inversion and narrowing, etc.). Depending on the position of the lesion, it may affect all sensory systems equally (central syndrome) or some more than others (paracentral syndrome).

The second type of lesion (subcortical) is related to the *brain disarticulation* that disrupts the coupling of the higher sensory-motor complex involved in the complerx activities mentioned above, while preserving the own motor and sensory activities, which do not undergo alterations in excitability and in the own structure. It is understandable that admitting the latter type of disorders means a consolidation of certain ideas of classical brain pathology, whereas the first type of disorders means a major pathophysiological and nosological transformation. The difference between the two types is justified by the type of anatomical lesion: nervous centers (gray matter) in the first type, long intracerebral pathways (white matter) in the second type. Their different nature is not an obstacle for both types to complement each other in the whole brain activity, leaving open the question of the close relationship between both types; for example, the possibility of a slight deficit of cortical excitability in a subcortical lesion, diaschisis effects or others that we have not yet studied with precision.

As an illustration of subcortical lesions, we present the following cases studied in 1947-48.

> *Deep left parieto-occipital glioma.* General symptoms: headache, very frequent vomiting, pain on percussion of the left parietal region, pain on pressure on eyeballs and trigeminal points, especially on the left side. There was no papilledema. Local brain symptoms: homonymous hemianopsia (explored without perimeter, in bed), amnesic aphasia and some sensory aphasia (great deficit in naming and evoking and poor comprehension), marked ideational apraxia, complete agraphia, acalculia, dyslexia, finger agnosia, difficulty with transitive movements (difficulty in left-right, etc.) and minimal visual agnosia (partial failure with overlapping figures).
>
> As for complex activities, the following stands out: when writing, the patient had great difficulty in handling the pencil, which he placed inverted, and had difficult to hold it between his fingers, as well as to place it on the paper [agnosia of utilization (Morlaás 1928)], he strongly deviated towards the left edge (visual side preserved) of the sheet of paper, placing the tip of the pencil on the fingernail of the finger holding the paper. When trying to write his name, there was perseveration of the initial line of the first letter, which prevented any further action. He failed all tests of naming, handling, etc., although he was able to perform simple tests on command such as sticking

out his tongue, closing his eyes, raising his hand (therefore, there was no innervatory apraxia). He was also unable to lift his head off the pillow or did so slowly and clumsily. He was totally unable to perform more complex commands such as sending a kiss with his hand or simply putting his lips in a kissing position. In the two-handed "thumbing one's nose" test, after much insistence and watching someone else do it, he attempted to bring both outstretched hands together, but without any relation to his face. In many praxis tests there was a strong tendency to perseveration, much less in language. Thus, there could be pseudoparaphasia. Spontaneous automatic language (ordinary stereotyped conversation, greetings, farewells, etc.) was correct, but more voluntary and selective language revealed an amnesic defect as well as a partial defect in comprehension. There was no paraphasia nor jargon aphasia.

The special examination on dynamic phenomena (excitability, asynchrony, etc.) did not give any positive results, although it was carried out while the patient was in bed, without the use of special equipment and without great precision. In any case, any dynamic deficit, even moderate, was ruled out with reasonable certainty. Some of the results of the dynamic tests were as follows. For the vision of the two healthy halves there was no delay. He perceived winks and fast movements even in low illumination or at some distance, there were no color and orientation disorders of the visual image (no slight deviation from the vertical of a suitable test object, even by moving it away or dimming its illumination to the point of barely perceiving it, and in monocular vision). In accordance with all this, there was no facilitation effect by strong contraction of the whole musculature. There was also no disturbance of tactile sensitivity in its various aspects including gnosis.

The clear clinical diagnosis was confirmed by ventriculography (disappearance of the posterior horn of the left lateral ventricle).

Another very similar case, although with a more inferior deep lesion, is that of an otogenic left temporal abscess, confirmed by surgery in the middle somewhat posterior part of the temporal gyrus (temporo-occipital region), quite deep and with a large purulent discharge. Symptoms in the acute phase, of general type (of intracranial hypertension, irritation) were: intense headache, fever, meningismus, slight papilledema, paresis of the left third cranial nerve, involvement of the left trigeminal nerve, etc. Local symptoms were: homonymous right upper quadrantonopsia and very marked amnesic aphasia with very slight difficulty in comprehension and occasional paraphasia. There was no dyspraxia, perhaps very slight signs. Dynamic tests on vision and other sensory systems gave no result, or it was so weak that it was within the error margin.

After several drainage punctures, symptoms remitted almost completely, except for residual deficits in previously blind quadrants.

Twenty days later, there was a relapse with the same symptoms that slowly increased, and further interventions were necessary. Over time, he developed pronounced intracranial hypertension with tendency to cerebral herniation. Brain involvement was still limited to the blindness of the quadrants mentioned above and to language in which the naming defect predominated in addition to some semantic and comprehension alteration. There was no apraxia, not even in the copying of drawings.

In new dynamic tests, it was not possible to clearly find any visual defect (no delay nor small tilt with respect to the vertical, no improvement with muscular effort, etc., even overlapping figures were well recognized). There were no problems in touch

either. However, perhaps there was some deficit of excitability in hearing; the right ear showed some hearing loss on audiometry. In the moderate comprehension deficit there was a certain lag because a word was heard well but its meaning was not immediately understood, even in the case of very common words; however, there was no facilitating action. The abscess became deeper and deeper and more difficult to drain. He had strong trigeminal pain (pain in the middle of the face, especially in the teeth, which were healthy).

In summary, the exposed subcortical cases did not show fundamentally perceptual defects, except for some "marginal" blindness (in relation to the projection área). Nor did they show comprehension defects, but the patients were unable to perform complex tasks (praxis disorder in the first case and verbal naming deficiency in the second) although they lacked a true motor lesion. It is not the time now to give further explanations, and we merely indicate the agreement between our observations and the point of view of the neurologist Pierre Marie when he considers that the neuropathology of war is a "polio-pathology" or pathology of the gray matter, whereas during peacetime there is rather a "leuko-pathology" or pathology of the white matter.

28. Schema in manual touch

28.1. TACTILE OBJECT RECOGNITION

After having studied the body schema in our brain-injured patients, we shall now deal with the tactile schema itself, i.e., the schema in manual touch (tactile gnosis). It is possible to establish relationships between both types of schema, the body schema and the manual schema, and to consider the latter as a particular aspect of the former; thus, the manual schema would be the body schema referred to the hand. Some authors have already pointed out the affinities of tactile gnosis with somatognosia (Schilder 1931), as well as with praxis in a broad sense (Lange 1936), considering especially tactile agnosia as a constructional agnosia. In our study we shall see the close link between manual recognition and body schema.

For a complete study of the present issue in our brain-injured patients, we shall first examine the characteristics of tactile manipulation of objects, and secondly, tactile recognition of objects (tactile gnosis).

28.1.1. Tactile manipulation

The study of tactile manipulation is the starting point for tactile object recognition. In view of what has been said about tactile localization and especially about the body schema, the first question that arises is how manual activity (manipulation and recognition) is possible given that the hand and especially the fingertips are preferential sites of distal involvement in the concentric reduction of the body schema.

It is understandable that for subject M, ordinary object manipulation (gentle hand contact) is of little use in obtaining tactile information about them. However, the subject uses spontaneously, and without receiving any external indication, various procedures that tend to counteract the disorder. These procedures are of two types; a muscular effort more or less generalized to the whole body (as in any voluntary action) which activates the somatic body model, and a touch with energetic pressure of the hand and fingers in addition to rubbing the object, which entails a

considerable increase in the tactile sensations aroused. This spontaneous correction of the preferential distal touch impairment has been present in subject M since he was wounded, and is independent of the finding of the phenomenon of facilitation by muscular effort. However, after this finding, such spontaneous action can be performed in a more energetic and secure manner. Proper manipulation of objects depends on the body scheme of the hand in which somatic, postural and praxic aspects of the hand are integrated.

It is clear that the correction of the defects, as indicated above, varies according to the degree of muscular effort and contact pressure. The hand may turn out to be more or less enlarged and sensitive in the body schema, as can be deduced from the errors in object manipulation in the following tests with patient M.

One of the tests is to count cards from a deck of cards with eyes closed and applying maximum facilitation by muscular effort. He acts slowly to ensure that he passes the cards from one hand to the other to count them. Out of 15 cards he only counts 14 in these conditions because one of the times he puts two of them together. This small error disappears with the eyes open. But if the facilitation by muscular effort is less intense in such a way that the body scheme makes the hand very incomplete (according to the subject's sensation), then more mistakes are made: out of 15 cards, only 11 are counted because more than one card is taken at a time. If the facilitation is even more moderate and the hand is probably only felt as "sketched," he counts only 7 cards out of 15, as he sometimes takes up to four cards together as if they were a single card.

A finer test, applying maximum facilitation by muscular effort, consists of counting the pages of a notebook with one hand. Out of 15 pages, he counts 12 because he puts two of them together several times. The same happens when leafing through a book with thin pages or a large-sized magazine. The difficulty can be so great that instead of grabbing the page by brushing the edge of the page with the fingertips, he decides to put his hand in the center of the page, trying to scrunch it up and grab it by the relief formed by the creases.

> The defect in manipulation, which is never completely erased, can be very diverse depending on the possibility of counteracting the distal deficit. Depending on the type of objects, it is sometimes possible to use maximum facilitation and contact pressure, as in the case of cards in a deck of cards where the cardboard is resistant. In contrast, with the thin pages of a book, the subject can only apply facilitation by muscular effort, as tactile pressure plays no role and the pages cannot be handled as independently as the cards in the previous test. It should be borne in mind here that the patient is a manual worker with rather rough hands, and although this has some influence it cannot be the cause of the errors made, as demonstrated by the degree of error in relation to the facilitation applied.

The test now consists of counting cigarette rolling papers. Out of 8 papers he only counts 5. He performs the test applying maximum muscular effort and also great tension in his hands to achieve maximum facilitation. At the same time, he uses all kinds of controls, such as rubbing the papers to make sure if there are several or just one, and putting the papers in one hand carefully to extract them with the other. However, as

can be seen in the result, he has pulled out two papers together, and the most remarkable thing is that he does not take the last one because he believes they are finished.

These types of tests can be varied indefinitely, and a similar result is always obtained. For example, another test is to take toothpicks from a toothpick holder by the tip. From a total of 11 toothpicks he picks up 8 of them (sometimes he picks up two together, and throws away another one without noticing it). In another test he has to pick up (and count) with one hand the pins he has in the hole of the other hand. Out of 35 pins he only counts 31. Here he makes less mistakes than in other tests because after picking up the pins he takes the time to touch them with three fingers to check if there is more than one, but if he does it with only two fingers, he always picks up several pins at once. As in the case of the cigarette paper, he considers the test finished when he still has two pins left in his hand; when warned, he then picks up one of them and drops the other without realizing it. As in the previous tests, he applies maximum facilitation and is left in full freedom to act except for opening his eyes, although sometimes sight does not help much in such subtle tests. Subject M performs all these tests with remarkable slowness despite maximum facilitation. Under weak facilitation it is almost impossible for him to perform them.

According to what has been said above, it is easy to understand that any daily manual activity must always be undertaken under the action of maximum facilitation including maximum muscular tension in his hands. He usually presses hard objects with his fingers or rub them repeatedly. This is what happens when he tries to button his vest; he uses muscular effort facilitation and both hands, although it is also possible for him to do it with one hand but with great difficulty and slowness. With weak facilitation he has great difficulty finding the buttonhole, less trouble finding the button, but then putting the button through the buttonhole is very cumbersome, taking altogether about 30 seconds.

The Schneider patient of Goldstein and Gelb (1919) used a tactile manipulation for object recognition analogous to that of patient M in the above test with pins, and in addition to multiple fingertip contacts, probably exerted some pressure on the object and was also aided by some diffuse muscular effort throughout the body, although his alteration was not as pronounced as in our patient M.

28.1.2. Tactile object recognition

Tactile recognition of objects is pathologically conditioned by a reduction in stereognosis that includes the aforementioned manipulation defect. Recognition is also affected by a defect in the tactile schema that hinders a synthetic conception of the object. This leads to a successive recognition of partial aspects of the object, a fragmentation that is typical of all types of disturbance of the schema (visual schema, body schema, etc.).

We shall therefore deal with the two defects mentioned, astereognosis and tactile asymbolia. The first has already been studied in part when dealing with the reduction in manual (active) touch (Sec. 23.5.2), and we shall now complete it in relation to the issue at hand. In the *stereognostic defect* there is no complete loss of the spatial characteristics of the object, nor of its form, but rather a reduction and a transformation are involved. *Stereognosis reduction* is a natural consequence of the gradual disorder of the

sensory function, and is manifested in modifications of the hardness, size and texture of the object, due to a reduction of the sensory dimensions of intensity, space and time. The object is then perceived as softer, smaller, smoother, etc., than it really is. Connected to this disturbance is the *stereognosis transformation*, a metamorphosis illusion that signifies a change towards more stable forms and that follows certain laws of configuration.

We have been able to find these characteristics in other brain-injured patients, previous to the ones we are now dealing with. These other patients always feel in the affected hand that the examined object has decreased in size compared to when they examine it with the healthy hand. They also feel that there are certain changes in the conformation of the objects as they may seem to be deformed in a certain direction, etc.

An illustrative example in subject M is as follows. Subject M holds a 7 × 5 cm wooden egg in any one hand (Fig. 28.1). If he applies maximal muscular effort facilitation, he perceives it as oval and feels that he is touching it along the fingers, although the fingers may perhaps be felt as slightly incomplete at their distal end. However, if the facilitation is much weaker, the egg is perceived as spherical, smaller in size and more localized towards the hollow of the hand or maybe more proximal, depending on the circumstances of facilitation, palpation, etc. (Fig. 28.1). If the facilitation were too weak and the palpation of the object too soft, all sensation of size and shape would vanish, and there would be even greater proximal deviation.

In conclusion, in diffuse perception, forms tend to be gradually simplified in accordance with the reduction of the organization. That is, it is not a matter of perceptual errors but of a regular change towards simpler and more stable forms, just as we have found in visual forms and in tactile forms. Thus, the modifications felt correspond to the state of the tactile field, and in this case, of the tactile field of the hand. If the size of the object is felt to be reduced, this is an expression of a similar decrease in the size of the hand, and the same is true for the change of form. Ultimately, it can be said that gnosis of the object is gnosis of the hand.

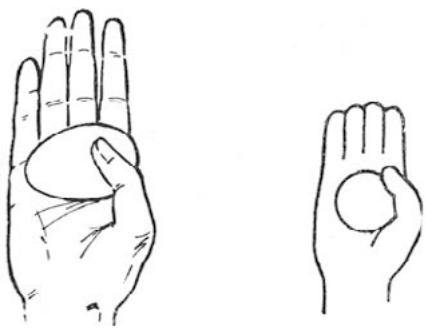

Figure 28.1. Schematic representation to illustrate the change in size and shape of a wooden egg. On the left, practically normal stereognosis with maximum facilitation by muscular effort. On the right, reduced stereognosis, with weak facilitation, perceiving a sphere of smaller volume and located more towards the center of the hand, corresponding to a certain reduction of the hand. (In the test, the hand is closed over the object. Here it is shown open for clarity of illustration.)

With regard to the *disturbance of the tactile schema* itself (tactile agnosia or tactile asymbolia), our patients, M and T, only seemed to have, at the beginning of being examined, the defect of loss of meaning of the manipulated objects. A few months after subject T was injured in April 1938, he identified a shell as a snail with the left hand, and as a "hollow thing" with the right hand, thus showing a tactile asymbolia mainly of the hand opposite to the brain lesion. But this asymbolia was not pure, since the subject already presented some defect of postural sensitivity and particular disorders of spatial sensitivity, among others. Patient M, studied extensively during the summer of 1938 following the finding of inverted vision, orthogonal disorder, etc., also showed bilateral tactile agnosia. A key held in either hand was interpreted as "a piece of iron with a handle up here and a point down below." This was not a pure asymbolia, since at that time he was found to have very pronounced disorders of deep and postural sensitivity throughout his body.

> However, failure in recognition was not always the rule in patient M. In certain cases he got it right, albeit indirectly, recognizing objects such as a buckle, a fork, a brush, a pair of scissors. In other cases he assigned a very different meaning, as in a sink stopper with a chain or a rifle bullet. Finally, in other cases he only indicated its shape or some character, without taking a decision. With respect to the different objects, his answers in quotation marks are indicated as follows:
>
> Buckle with belt: "I don't know ... with corners," (touches the buckle) "looks like a buckle ..." (pulls the belt, touches the holes) "a belt, it has here something to hook" (speaks very slowly).
>
> Fork: "It has points here … a fork."
>
> Nail brush: "Soft here" (the bristles) "and hard here" (the wood). It makes the bristles sound, but by making noise at the same time it cannot recognize the nail brush, and only when it stops making noise can it recognize the nail brush by repeatedly moving the bristles.
>
> Pocket scissors: "A piece of iron with two handles," (he recognizes them by making noise with them, otherwise he doesn't know it).
>
> Sink stopper with chain: "clock with chain."
>
> Rifle bullet: "A nail ... here the head and here the tip, a round nail."
>
> Key: "A piece of iron with a handle up here and a spike down here."
>
> Candle: "A piece of wood ... a stick ... it has a rope here."
>
> In the cases in which he responds correctly, he needs much time and the procedure is not correct and simple, since he uses an indirect analytical-deductive method instead of the immediate and synthetic method of the normal subject. This indirect way of proceeding was mainly emphasized by Goldstein and Gelb (1918, 1919) in their patient Schneider of whom they said that he did not acquire a simultaneous representation of the object since he only provided a description as a sum of parts. Such tests show the need to pay attention to the smallest details and to the entire process, and not to be satisfied with just recording the final effect, which could give the false idea that tactile recognition is normal. This is true for many other functions, as we have repeatedly pointed out throughout this book.

Our patient M behaves in all these tests like patient Schneider. In the case where neither subject was able to recognize an object by touch, they had a fairly good idea of its shape. If the object was offered to them again much later under the same conditions, they came to identify it as the same object as before, even though they did not recognize it. However, in the case of an object that they had not recognized by touch (e.g., a key in the case of subject M) they were of course able to recognize it immediately afterwards by sight, but were reluctant to believe that it was the same object of which they had only recognized the shape by touch a few minutes before seeing it.

One of the most remarkable phenomena consists in being able to draw an object with one hand while the object is tactilely examined with the other hand, although not recognized. The drawing, rather acceptable although schematic, does not allow the subject to recognize the object either. Subject M was examined in this respect in 1938: While he was manipulating with his left hand an object that he was unable to recognize, he was drawing its approximate shape with his right hand by looking only at the drawing (see Fig. 28.2). When finished, he still was unable to recognize the object. The same happened with subject Schneider for several objects, in spite of drawing much better than our subject, as can be seen in Fig. 28.3. The work of Goldstein and Gelb (1918, 1919) contains other similar drawings of a key, a printer's stamp, etc., very well drawn despite their difficulty.

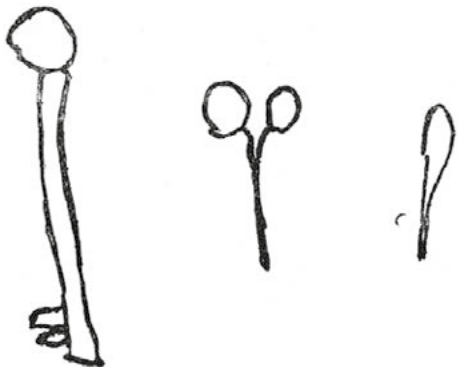

Figure 28.2. Drawings by patient M in 1938. Original size drawings of objects (key, scissors, bullet) drawn with his right hand and looking at the drawing while the objects are manipulated with his left hand. At the end, he does not recognize the meaning of his own drawings which, although deficient, convey the main ideas.

Apart from these similarities between the phenomena, it is not possible to accept the authors' explanation of the method followed by their patient (regarding the way of obtaining sizes, proportions, etc.) to make the drawings. Their explanation is aimed at supporting the idea that their patient is completely unable to acquire spatial relations. On the contrary, it is clear that subject Schneider, like subject M, was able to achieve some spatial perception, albeit more or less reduced. We already know that functional reduction, which affects complex and constructional activities, hinders the idea of the

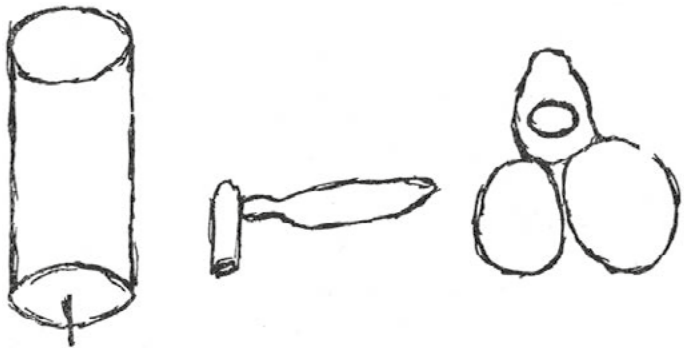

Figure 28.3. Drawings by patient Schneider of Goldstein and Gelb (1918, 1919). Original size drawings of objects (candle, percussion hammer, cigar scissors) drawn with one hand looking at the drawing while the objects are manipulated with the other hand. He is not able to recognize the object either tactilely or visually.

whole. The subject is drawing step by step, without having much idea of the unity of the whole but being able to conceive different parts. The disorder thus lies in the asymbolia of the object, in the inability to integrate the whole process of tactile recognition. The fact that the drawing made of the object is also not visually recognized is due to the fact that it is extremely schematic and even deformed, as occurs in subject M, according to what has been said about schema in visual forms (Sec. 15 in Vol. 1). This also applies to the Schneider patient. A year later, when the facilitation by maximum muscular effort was discovered in subject M, he was able under such facilitation to recognize the drawings of the key and the scissors (Fig. 28.2) although not that of the bullet because it was very deficient.

The finding of facilitation by muscular effort introduced relevant changes, and nowadays, the defect in the manual touch schema (tactile agnosia) can be counteracted to a great extent allowing subject M to easily recognize many objects. Difficulties or confusions arise only in special tests. For a complete study of the issue we shall first deal with the behavior of subject M in his usual state, that is, with moderate facilitation which he already applied involuntarily before discovering the phenomenon, and the possibility of considerably intensifying it. Afterwards, we shall deal with the changes introduced by maximum facilitation (by maximum muscular effort).

Subject M, whatever his state, uses a series of tactile aids in the tactile recognition of objects, such as pressing them with the fingers, rubbing them with the tense hand, exploring them successively in multiple directions, thus trying to discover their various aspects, and all this is done with remarkable speed. However, the subject in his usual state takes a certain time until he considers the process finished, a process that is rather an inspection than a true recognition. This time is variable depending on the familiarity of the object, the more or less moderate degree of facilitation by diffuse muscular effort, etc., and can range from 7 to 20 seconds, whereas a normal

subject reaches the meaning at once and immediately. Thus, in a first phase he makes an analytical examination of the object, which does not go beyond providing a certain idea about its shape, spending 5 to 7 seconds, sometimes much more. In a second phase, the subject, who has only a descriptive conception of the parts (not simultaneous like that of the normal subject) passes from the analytical to the deductive phase and wonders what the object in question can be. Sometimes he succeeds in finding out by indirect means, sometimes he remains in suspense without being able to give more than a description of the shape found before. In this second phase he needs another seven seconds. Finally, sometimes a third phase is necessary to find the name of the object when somehow its meaning has already been found, but this is a process that goes beyond the simple recognition of the object.

These phases are similar to those of visual object recognition (Sec. 11.3 in Vol. 1), although the circumstances are not quite the same, since tactile recognition is already somewhat less simultaneous than visual recognition in a normal situation. All of the above is illustrated below with examples of recognition of the following objects by subject M in his usual condition; (his answers are in quotation marks).

Ordinary metal spoon: after handling it a lot, he says "... it has a bowl, it must be a spoon." He has taken 20 seconds because he has applied little facilitation by muscular effort.

Easily recognizable objects, such as a thimble, a ring, etc.: he usually takes five to seven seconds to recognize their shape. In the case of the thimble, he takes five seconds to know that "it is a round thing, hollow at one end." A few seconds later he names it correctly.

Pocket watch: "this looks like a box, but it has a ring up here. It's most likely a watch."

Hat brush with handle: "... a toothbrush."

Metal medal: "a round thing equal to ten centavos."

Open safety pin: "here together and here apart, if a piece were removed it would be a hairpin."

(See other examples in the fine print above.)

The way to *deductively* find the meanings lies, more than in joining the various aspects, in relying on some very typical detail of the object, such as the concavity of the spoon, the hollow of the thimble, the ring of the watch, or being able to insert a finger in the case of a pipe or a flask, etc. When this is not possible, the meaning is greatly altered, as in the case of the medal, or even more so in the case of the safety pin. At other times, the integration of the whole is more necessary, even if there are characteristic details, as in the case of the key. When it is not possible to achieve the synthetic meaning, it is not possible to go beyond a purely additive description of the parts, which is characteristic of tactile agnosia. The resulting conception is then fragmentary and diffuse. That is why changes of meaning or approximations occur, such as taking small watchmakers' pliers for scissors. The subject also does not get a correct idea of the size, which leads him to take the brush with handle for a toothbrush. Other times the defect of recognition borders on astereognosis, for example, when taking a fork for a spoon, a button for a coin or a screw for a nail. In short, tactile recognition is very slow, requires a lot of successive tactile

inspection and, above all, fails to grasp the whole and the specificity of the object. It must therefore be concluded that, although the correct answer is sometimes given, the recognition is somewhat deceptive and, in any case, different from the normal one.

Maximal facilitation by maximum muscular effort considerably reduces such defects, and then only abnormal signs are found in some tests. The recognition time is reduced to one second as a minimum. If there are difficulties, due to the smallness of the objects, the time may be increased to three or four seconds (this remarkable brevity is due to being quite familiar with repeatedly tested objects). He also applies strong finger pressure and rubs the object, but in any case, the examination of the object is much faster and safer, without the need to inspect in multiple directions and repeatedly check the details. Thus, for the previously described case of recognizing the spoon, now with maximum facilitation it is enough for him to hold it with one hand by the handle and place a finger in the concavity next to the handle to know what it is in a second, without the need to resort to other manipulations. There is therefore now a synthetic (normal) conception, but with a facilitation due to a maximum contraction of all the musculature including that of the hands, which for a normal subject is totally unnecessary. Under such conditions, both astereognosis and asymbolia defects are erased, and he quickly recognizes the shell, the buckle, the medal, the cap, the chain, the glasses, the candle, the brush, the key (whole or without teeth), the bullet, the smoking pipe, etc. However, some insecurities or confusions may still arise, such as taking a button for a coin, or a screw for a nail; but a small warning is enough for him to correct the error. He recognizes small sculptures of human and animal figures; however, he almost failed with a porcelain elephant of about 18 cm, and only after much thought was he able to recognize it. We already know that the maximum facilitation cannot erase all the defect, and also now, some lability can be evidenced in special recognition tests.

In the same way that overlapping drawings constitute a very selective visual test for discovering agnosia or schema defects (Fig. 15.4), we can follow a similar procedure with touch by trying that the subject recognizes two or more objects together in one hand. Indeed, subject M with maximal facilitation recognizes well separately a belt buckle and a pin to fix shirt collar, but if given both together hooked to each other in some way, he fails completely. With four or five objects together he takes a long time and makes mistakes and omissions.

> At the beginning of the test with several objects at the same time, the subject tries to select them by removing them from the palm of the hand one by one to explore each one with the fingers, but then the test is like on a single object and does not correspond to the required demand. He takes a long time, but all the objects (four) are recognized correctly. Instead, if he is not allowed to make that selection and is asked to handle them all at the same time, he makes mistakes. From five small objects that he must pick up all at once with one hand (two buckles, a pin to fix shirt collar, a button and a 3 cm key) he claims to recognize "two buckles and two buttons" in 20-30 seconds, unable to resist making a certain selection. Therefore, he has omitted the key and has mistaken the pin to fix shirt collar for a button.

In another test with larger objects, such as a small pliers, a branch of scissors, a key, a medal, he names: medal, key and scissors. In another test with the same objects he answers: key, scissors leg, medal and bullet (this last word probably refers to the sharp head of the pliers).

It may be objected that these simultaneous recognition tests with the hand may be very difficult for many normal subjects, but in fact they are within the reach of any normal subject, at least after some training in a few trials. Such is the case with our patients who have been repeatedly subjected to a multitude of tests in long sessions, and end up adapting quite well to the tests, within their sensory deficits.

The tactile reduction that occurs when an object is covered with a handkerchief causes the perception time to lengthen even under maximum facilitation. For example, for each of the separate objects: medal, buckle, etc., the time increases from one second without the handkerchief to twelve seconds with it, since a single contact is not sufficient for the subject, and he must use rubbing and so on to make up for the loss of tactile finesse due to the interposition of the handkerchief. In perception speed tests, the tactile recognition deficit can also be evidenced. In this type of tests, subject M is only allowed to hold the object in his hand for a short time (the object is put in his hand, touched in some way, and then the object is immediately removed). The results may be difficult to be accurately assessed. Subject M seems to need a second for recognition under the best possible conditions, what the normal subject does in much less time, perhaps a third of a second or less, at least for usual objects. Subject M always fails in quick tests, for example in the case of the button, which takes it by a coin, he needs almost two seconds to have time to rub it between his fingers.

Patient T, much less examined but very similar to patient M when M is under maximum facilitation, usually recognizes all types of objects (last exploration in 1944), but tends to use successive touch and an analytical-deductive procedure, generally taking two seconds and sometimes much longer in the tactile recognition of objects. If the objects are not very easy, he takes up to five seconds and more. This recognition is therefore not equivalent to that of the normal subject even though the objects are identified, since a certain degree of initial synthesis is missing, and this occurs in both hands although the right hand is somewhat more affected.

Summarizing all this section on tactile recognition of objects, we see that the alteration of this process involves a complex of disorders including distal reduction, astereognosis and schema alteration. The latter is the most evident and consists in a fragmented conception, i.e., in the inability of simultaneous activity, characteristic of any type of alteration of the schema (visual, praxic, etc.). Normal simultaneous comprehension is replaced with successive comprehension.

28.2. CRITICAL EXAMINATION OF TACTILE AGNOSIA

The nature and individuality of the classic syndrome of tactile agnosia raises similar problems to those of visual agnosia (Sec. 15.3 in Vol. 1) and perhaps with a longer

tradition of controversy. We shall first examine the historical background, then the interpretation of the brain dynamics presented here and, finally, our refutations.

28.2.1. Historical background

The question at hand begins with the old Wernicke - Strümpell - Dejerine controversy, in which the first of these authors tries to maintain the independence of the tactile agnosia syndrome whereas the other two refute such autonomy because they consider that tactile agnosia, erroneously called tactile paralysis ('Tastlähmung' in German) of Wernicke (1895) is only the result of a more elementary sensitivity disorder.

> This controversy deserves a careful examination since it is the origin of several tendencies and also of many misunderstandings. Wernicke (1895) described two cases of cortical lesion in which the subjects were unable to recognize objects by touch, despite showing scarce alterations of tactile sensitivity. There was then a remarkable disproportion between such alterations and object recognition; that is, the recognition defect went far beyond what could be expected from the small alteration of the simplest tactile functions. Wernicke also pointed out that in cervical tabes with significant alterations in sensitivity, tactile recognition is relatively well preserved, whereas on the contrary, in cortical lesions, slight defects in sensitivity cause an almost absolute impossibility of recognition. Thus, this disproportion led him to establish tactile recognition disorder (tactile asymbolia) as an independent nosological entity. Shortly thereafter, Strümpell (1918) in Germany and Dejerine (1907) in France took a position against the independent existence of tactile agnosia. They were followed by many authors who refused to admit a manifestation of pure agnosia without any other sensitivity disorder. Vouters (1909), following Dejerine (1907), went so far as to affirm that non of the cases of tactile agnosia published up to that time was genuine.
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> It is remarkable to note that while all the authors of the time accepted the hypothesis of anatomical localization according to the specificity of the nervous centers, mutual associations, corresponding nosological entities, etc. (anatomical and associationist theory of brain functions), Dejerine who was no exception in this respect for aphasia and so on, adhered to a very different unitary approach for cortical tactile functions, more appropriate to current times. In the two cases of Wernicke (1895) the presence of elementary sensitivity disorders was quite patent. The question is then centered on the disproportion of alteration of the various functions. For Wernicke, agnosia cannot come entirely from the most elementary defect, whereas for Dejerine, agnosia would be explained by the elementary defect and does not care much about the lack of parallelism in intensity between elementary sensitivity disorder and agnosia. Subsequently, the meaning of this lack of parallelism was highlighted by different authors such as Heilbronner (1905, 1910), Bonhoeffer (1918) and Goldstein (1915, 1925). Further on in this section we shall see its scope and modalities.

Certainly, in cortical lesions the most frequent cases are mixed forms of astereognosis and tactile agnosia, with diverse disorders of the simplest sensitivities. In

most cases, astereognosis predominates. Of this type are almost all the war wounded with parietal lesions studied by us.

> In about 40 injured subjects with parietal brain lesions, studied in detail by the usual clinical procedures, a global alteration of the tactile system was always found, sometimes with a great astereognosis disorder easily explained by the deficit of more elementary functions, these cases being the most abundant. Other cases had a greater disproportion between the astereognosis defect and the elementary deficit in tactile sensations, spatial functions of localization, movement, spatial discrimination, etc. Rarely there was astereognosis disorder mixed with tactile agnosia and with minor impairment of elementary functions. In this type of injured subjects, it was possible to determine all kinds of forms of alteration, e.g., partial astereognosis of the hand or limited to a few fingers, in very circumscribed parietal lesion. The diagnosis we made in 1938 for most of these cases was "reduced critical sensitivity," affecting the whole set of tactile functions but always in much greater proportion to the higher functions, for example, spatial functions more impaired than elementary sensations, and the meaning of forms even more affected than spatial functions. It was impossible to establish the disorder of a single function, and the most that could be done within this general syndrome was to determine the predominance of astereognosis or of agnosia.
>
> It is noteworthy that in the few cases that could be framed between astereognosis and agnosia (perhaps more of the latter), the elementary and spatial functions appeared almost normal at the beginning of the ordinary clinical examination, but in the course of such examination an easy fatigue and functional lability appeared for Weber's test, posture, small passive movements, etc. In addition, whereas the patients recognized the shape of large objects quite well, they failed with smaller objects; many errors were also made in the meaning of objects and only an approximate meaning was possible, such as confusing a fork with tweezers or a lighter that opens with scissors, this being a disorder of the type "disjunctive" agnosia of Liepmann (1909). In such cases with great disproportion between the elementary defect, which was minimal (only lability), and the very patent defect of shape and agnosia, it should be noted that the patients were in the recovery phase and shortly before had presented significant hypoesthesia.
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> Cases that at first only appeared to be of pure asymbolia are our two patients, M and T, but a more precise analysis showed in the long run much more widespread disorders. This occurred before the discovery of facilitation by muscular effort, which masked the degree of abnormality, and despite the fact that the subjects were in the recovery phase.

Although pure cases of tactile agnosia are certainly very rare (admitting that they may exist), there is a tendency to accept that some cases have indeed been described, despite the assertion of Dejerine and his followers as stated above. The cases of Kutner (1907), Gerstmann (1918), Bonhoeffer (1918), Raymond and Egger (1906), Guillain and Bize (1932) (among many other authors) are often mentioned as the most representative cases of pure tactile agnosia. Thus, we would have to add here somehow our two patients M and T, or at least the second one in which disorders other than agnosia are more difficult to be detected, and also the Schneider case

presuming the view of Goldstein and Gelb (1918, 1919) that touch is not basically affected but only by the loss of visual influx. But it should be noted at once that while many authors consider the above reported cases as being of totally pure tactile agnosia, others, more cautious, consider them to be *relatively* purer cases. This is because certain more elementary alterations can be detected in special determinations in "very pure cases," and other cases already show at first glance small elementary defects (see fine print below).

Indeed, in the case of Kutner (1907), considered as transcortical tactile paralysis, the subject was suffering from epileptic seizures and had some general mental disorders, which already clouded the diagnosis of tactile agnosia. In addition, a considerable functional variability was noted in the tests, i.e., a remarkable fatigability not only in tactile recognition but also already in sensitivity tests, showing slight disturbances of joint sensitivity and of localization. Thus, there was no absolute purity.

The case of Gerstmann (1918), one of the purest among all those mentioned, was a war wounded man with a parietal lesion presenting tactile agnosia as a residual form of a preceding astereognosis; therefore, at least the course of the disorder was not pure. In addition, it is noted, as in the previous case, a great fatigability of the subject in tactile recognition tests and a variability of the results from one day to another. Such patent fatigability in these cases makes them object of the criticism of Weizsäcker (1923, 1931) (see below).

Other cases are much less pure. In that of Bonhoeffer (1918), also a war wounded man, there was agnosia only in four fingers of one hand (the thumb was not), and there was also a very small defect in temperature and pain sensations, as well as an increase in Weber's test. The case of Higier (1916) also belongs to this type of cases, although more impure than the previous one, with alterations of sensitivity and localization errors. These partial-type cases also pose difficulties for the classical conceptions of the gnostic areas and their transcortical action, as indicated by Kleist (1934), although Bonhoeffer (1918) claims the existence of special connections between the gnostic area and each of the fingers. We have also found these partial types in war wounded, although much more impure than those mentioned, and more mixed with astereognosis and elementary disorders of sensitivity. Similar to the case of Bonhoeffer (1918), although extended to the whole hand, is the case of Poggio (1908), a subject who after surgery for cysticercosis presented a slight astereognosis and subsequently tactile agnosia and a small increase in Weber.

Rather less pure is the case of Raymond and Egger (1906), erroneously considered by these authors as a case of "tactile aphasia," although it would only correspond to a tactile asymbolia. The patient was a 64-year-old woman who had suffered a stroke with language disorders and monoplegia of one hand. In addition to the typical defect of recognition of the meaning of objects with the affected hand, she presented many other defects of sensitivity, mainly of spatial nature (increased Weber, tactile localization errors, decreased vibratory sensitivity, decreased sensations of weight and pressure).

Other cases sometimes mentioned in the literature on tactile agnosia, besides showing small defects of other kinds that make them impure, were transient forms of deeper alterations of sensitivity, residual states of earlier more elementary astereognosis, or

conversely, early stages of progressive disorders that were disintegrating more deeply the cortical tactile system. To the latter type belongs the observation of Marie, Bouttier and Bogaert (1924) in a prefrontal tumor with various mental disturbances, and also the case of André-Thomas and Courjon (1917) with a parietal tumor. To the type of residual agnosia belongs the brain-injured patient of Magnus-Alsleben (1924). Already Villaret (1916) rightly understood the tactile manifestations in brain-injured patients as stages of recovery from deeper disorders. However, a more modern author such as Delay (1935) still tries to interpret the various modalities along the course of the disorder as changes in the association of syndromes independent of each other. The latter cannot be accepted after all that has been said in the above-mentioned series of cases, since it is seen that the disorder affects the tactile organization as a whole. Only in seemingly pure cases the defect in elementary functions is very small in comparison with the defect in higher functions because the rule that higher functions are affected in greater proportion is always fulfilled, this being an experimental fact independent of any theoretical orientation.

At this point it is unnecessary to mention more cases, since with those already mentioned it can be seen that tactile agnosia is not pure since it is always more or less clouded with more elementary tactile defects. However, we shall mention again the case of Guillain and Bize (1932) because of its special circumstances, since it was later subjected again to a long and detailed study by Delay (1935) using very precise psychophysiological methods. This case is that of a young woman with a parietal wound caused by a bullet resulting in hemiparesis, transient speech defects, etc., with easy recovery. Given the type of injury, she must have also presented hypoesthesia, which would allow considering this case as residual agnosia, but this is not indicated in the summary we have used. A year later this case was presented by Guillain and Bize (1932) as 'pure astereognosis due to a traumatic parietal cortical lesion,' and two years later she was thoroughly examined by Delay (1935). The tactile sensitivity of her hand was as follows: intact sensations, very small defect of tactile localization, clear defect of simultaneous and successive Weber (the first increased five times the normal value), small defect of shape appreciation and complete loss of object recognition, although she can slowly recognize stereometric objects by deduction. From all this it can already be seen that the case is far from being pure; in fact, there is a rather global impairment of the tactile organization, although the maximum defect is in the higher function (gnosis).

It is notable that the very accurate pressure, pain and temperature tests in this case, which had provided normal results with mechanical methods that only measured intensity, showed significant defects with electrical methods capable of measuring intensity and time. The chronaxie of the sensations, determined by Bourguignon (1933) himself, was increased in the affected hand about three times above the normal value for mild pain and for temperature, but pressure was shown to be intact. The chronaxie measured was for the palm of the hand. Perhaps pressure was affected in the fingertips. In short, the more complete and precise the study of the cases, the better the impurity of tactile agnosia can be demonstrated. The disorder is of the whole organization, according to certain characteristics of physiological hierarchy.

From a careful examination of the circumstances of the so-called pure cases, the Wernicke-Dejerine controversy cannot be considered over. The so-called pure

cases being really impure somewhat favors Dejerine's interpretation. But at the same time, the defects of elementary sensitivity in some cases are so minimal that these cases could fit Wernicke's interpretation of the real existence of a tactile asymbolia not derived exclusively from simpler defects. What is evident in these last cases is the great disproportion between the weak alterations of the elementary sensitivity and the total lack of capability for tactile recognition of objects.

Let us now address the current state of the issue. An author like Foerster (1936), always in line with classical brain theory and not at all suspicious of sharing more modern anti-localist conceptions, recognizes the limitations of the classical approach. With regard to tactile agnosia he states that the more precise the physiological methods for the determination of thresholds in hand sensitivity tests are, the better we can demonstrate almost without exception that along with tactile agnosia there is an alteration of the pressure points on the skin, an elevation of their intensity thresholds for mechanical and electrical stimuli, an increase in the chronaxie of the pressure points and a fatigability of these points under multiple repeated tests in a short time. He also states that he fully agrees with Weizsäcker's criticism on the concept of pure tactile agnosia and especially on the thesis that sensitivity as such might be completely intact. Weizsäcker (1923, 1931), in part associated to Stein (Stein and Weizsäcker 1927, 1928), considers the usual clinical examination of tactile sensitivity to be insufficient. Where the usual methods fail to discover elementary disorders, appropriate physiological methods (chronaximetry, functional analysis, etc.) can reveal the existence of clear disorders of excitability, a functional change ('Funktionswandel' in German) expressed in threshold lability, increase of the characteristic time, etc. In short, there is a modification in the excitability state which alters tactile perception and would provide an explanation for the agnosia disorder. This leads Weizsäcker to declare that the Wernicke-Dejerine controversy is over, and he denies of course the existence of the tactile agnosia postulated by Wernicke (1895).

Another aspect of the current status of the question concerns the phenomenological characteristics of tactile asymbolia. Since the analysis of the Schneider case mainly, we have not only paid attention to the lack of understanding of the meaning, but also to the indirect way of arriving at it in the tests in which the agnosic subject gets it right.

> From very diverse perspectives, attempts have been made to understand the mind with agnosia, either philosophically by Cassirer (1923, 1925, 1929), or psychologically, especially by Révész (1926) in the case of tactile agnosia, or pathophysiologically by Delay (1935). From all these approaches, the result is always what Goldstein and Gelb (1918, 1919) had already indicated with respect to their patient Schneider: replacement of simultaneity with the addition of parts (see Sec. 28.1.2).
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> For Cassirer (1923, 1925, 1929), the world of the agnosic subject (tactile, visual, etc.) is threatened to break up into fragments. The superior unity is lost, the syntactic structure of the whole phenomenon disappears, a sort of "agrammatism" of the rules of perception prevails, similar to the defect studied by Pick (1905) in language. Delay (1935) goes so far as to say that tactile asymbolia is characterized not so much by null

recognition as by a radical change in the recognition process, and includes tactile asymbolia within the semantic alterations, while still admitting the autonomy of tactile agnosia.

Half a century after Wernicke, the theories about the brain have undergone many transformations, but the Wernicke-Dejerine controversy is not entirely satisfactorily resolved by what has been said so far, even if it seems otherwise, and at least the arguments of the great German author retain their full value.

It is now up to us to develop the issue by means of the brain dynamics presented in this book.

28.2.2. Interpretation according to the brain dynamics

The findings and considerations of Weizsäcker (1923, 1931) seem to put an end to the controversy on tactile agnosia, this being admitted to a certain extent by authors as prominent as Foerster (1936) and Lange (1936), among others. However, going back to the roots of the issue and being consistent, it must be admitted that the problem remains more or less as it was raised by Wernicke (1895) with his two cases, i.e., the great disproportion between the defects of elementary sensitivity and the abolition of tactile recognition of the meaning of objects is still the problem to be solved. The two cases of Wernicke (1895) were already impure, then came other purer ones (Kutner 1907, Gerstmann 1918) among others, which presented some doubts. Finally, Weizsäcker (1923, 1931), in more precise determinations, found that all the cases that can be studied are perfectly impure. This has two important consequences. One is that we should not expect to find a pure case. The other, which is essential, is that tactile agnosia would derive from the disorder of elementary excitability leading to a disorganization in perception, since it is the tactile system as a whole that would be impaired. Although all this is important, the problem of the mentioned disproportion is not solved. As valuable as the conception defended by Weizsäcker (1923, 1931) on the unity of the sensory system is (experimentally confirmed in the brain dynamics presented here), it does not explain the mechanism by which a very small disturbance of elementary excitability leads to a suppression of higher functions (much more complex).

The above-mentioned disproportion (non-parallelism in the intensity of alteration), which corresponds to the principle of Jacksonian ascendancy on the greater vulnerability of the higher functions, constitutes a gap in the ideas of Stein and Weizsäcker (1927, 1928) and Weizsäcker (1923, 1931). Already at the beginning of this book we warned that the "Funktionswandel" [functional change] of these authors was not able to specify sufficiently the mechanism of perturbation of the different functions. This instead is possible here in our study thanks to the experimental finding of nervous asynchrony (Sec. 3.5 in Vol. 1) which increases the excitability thresholds of the different brain functions differently. Excitability characteristics are also determined here not only for the most elementary activity as in Stein and Weizsäcker, but also for many other more complex functions and in a systematic way. Therefore, from the *quantitative* characteristics of the different altered functions, the problem

about the non-parallelism is immediately solved. The mentioned disproportion comes from the fact that desynchronization follows a Fechner-type logarithmic law showing the change in the sensorium, as summarized below.

> The curves on desynchronization of functions, either sensory recruitment curves or the asynchronous bundle of curves of various sensory levels, illustrate accurately the physiological basis of the non-parallelism in the intensity of alteration between elementary and more complex functions. For example, with respect to tactile functions, in Fig. 22.8 or 22.10 it can be observed in subject M that for the sensation of *simple touch or pressure*, the chronaxie increases about ten times the normal value, whereas for a *near-normal localization* sensation, the chronaxie increases about fifty times, which results in a considerable abnormal separation between the respective strength-duration curves of these two extreme types of sensation. From the localization recruitment curve (proximal deviation as a function of stimulus intensity, Fig. 22.3) and also from the orientation recruitment curve (Fig. 22.9), it can be seen that the lower levels (intense proximal deviation and strong inversion, lower part of the curves) are recruited with a weak increase of the stimulus whereas the states closer to normal (high part of the curves) require a considerable increase of the stimulus. The same occurs in vision, for example in the recruitment of the visual image orientation: in order to go from almost 180° of deviation (inversion) to 90°, a small increase in stimulation is sufficient; but to go from 90° to 0° (normal), the increase in stimulation must be very much greater.

In a given sensory sphere, the whole system is impaired, all its functions are affected, but in greater proportion the complex activities (the most physiologically demanding). Thus, the Jacksonian principle of disproportion in functional alteration is physiologically explained by generalizing the law of Fechner (1860) on sensory growth to pathological manifestations. Considering more broadly the problem of the mentioned disproportion, we see that it is related to the question of the so-called clinical "dissociations," which we have addressed more than once when studying tactile functions, especially tactile sensations (Sec. 20.1 and 20.2). We stated that there are no *absolute* but *relative* dissociations since the impairment is global although some functions are much more severely affected than others according to their physiological characteristics (see also Sec. 23.1 on simultaneous and successive Weber test). Dissociations correspond to the phenomenon of asynchrony that takes place in the series of differentiations of sensitivity (i.e., in the sensory organization), which leads to a dynamic reduction of the system.

It is understandable that the activity of the sensory system is a whole involving the entire system, and that the various syndromes that are presumed to be well-defined and independent entities (agnosia, astereognosis, individualized disturbance of this or that special function) are in fact predominant alterations but not the only ones, thus the usual nosological classifications have at best only a relative value. In fact, what is found is a concrete state of excitability and organization of the system (Sec. 3.6 in Vol. 1), corresponding to a particular stage of development of the sensory field in which the different functions, apparently independent, are only a consequence of the level of organization (see final part of Sec. 22.4), and therefore do not have a

reality of their own. Thus, it does not make much sense to pay attention to the multitude of seemingly independent sensory phenomena, nor to broader classifications of phenomena such as the usual one into sensations, perceptions and gnosis. To specify in a simple and accurate way the new ordering of phenomena we must refer to the perception of *sensory dimensions* (intensity, space and time), which can be expressed numerically and reflect the underlying physiological state. As for the manifestation of the phenomena, it is framed within the spiral development of the field, an immediate result, as we know, of neuro-sensory isomorphism.

Restricting ourselves to the question of tactile recognition, we state that it is the final result of the organization of the tactile field (of the hand). It is thus not necessary to resort to theories about secondary identifications, inter-sensory supports or visual associations for the object's meaning to emerge. Otherwise, those blind from birth would necessarily remain in a state of tactile agnosia, and this does not happen. More important now is to highlight the characteristic fragmentation of the recognition process that we have repeatedly observed in both vision and touch. This is an agnosic, semantic, categorical, schema disorder, very commented within the brain pathology by Goldstein and Gelb (1918, 1919), Gelb and Goldstein (1922, 1923, 1924), Gelb (1925/1926, 1937), Head and Holmes (1911), Woerkom (1925) etc., and outside of it by Cassirer (1923, 1925, 1929), Benary (1922). Delacroix (1930), among others. What needs to be specified is the origin of such *fragmentation* in recognition, i.e., the successive recognition process followed by a subject with agnosia as opposed to the immediate (synthetic) process followed by a normal subject. We have already said that this type of abnormal recognition derives from a deficit in organization (e.g., see Sec. 15.3 in Vol. 1 regarding visual agnosia), and this is what we intend to define more precisely.

By carefully examining the type of perturbation in the schema function, we see that if there is fragmentation it is because there is still some vestige of interpretation, that is, some vestige of schema. The schema subsists but reduced, as happens in any other type of function. This means that the understanding of the meaning is poor, that the schema has lost intensity (development), thus it cannot encompass the whole but only parts or fragments. In such a situation only the successive (discontinuous) interpretation appears, which in reality is nothing more than a juxtaposition of partial interpretations independent of each other. A subject with agnosia cannot integrate or construct (as in simultaneous visual agnosia, praxis discontinuity, constructional apraxia, etc.), cannot conceive of the whole and the higher unity, and therefore remains in a series of unconnected details.

According to what has been said, we think that the origin and basis of all this behavior in agnosia is that the schema has been reduced, i.e., there is a *reduction in the dimension of the schema.* In this way, we believe to have solved the proposed problem. It could be objected that such a solution was already implicit in the idea of fragmentation in recognition, but it can be replied that it was not clearly expressed. In any case, the importance of the mentioned solution lies mainly in the consequences it has, orienting the issue of agnosia in a physiological direction very different from the psychological one followed so far. The concept of schema dimension means framing the issue in a strictly physiological domain, since the mentioned

reduction in the dimension of the schema is no different from the reduction in the dimensions of the sensory field as referred to above. Therefore, what occurs for all functions is a *change of scale* (another dimension value). Everything is thus established on a physiological basis since the dimension depends on the active brain mass. This simplifies the issue, gives it a firm basis and, consequently, excludes many sterile discussions.

For example, the disputed question of the so-called "noetic" function as to whether it is a disorder of the general psychic (noetic) activity or a disturbance of the physiological mechanism is meaningless in our conception, since we have to refer only to the dimensions of the sensory field and its organization, specified precisely by its dimensions. Moreover, this means that there is the same pattern of organization for all phenomena, there being no opposition between sensory functions and gnostic functions, but rather a continuity, as indicated in Sec. 27.5, for example. Thus, the change that occurs in schema by replacing simultaneous perception with successive perception is analogous to the change from simultaneous Weber to successive Weber. Let us recall that when the simultaneous Weber is almost abolished, the perception of duplicity with the successive Weber is still possible.

28.2.3. Discussion. Refutations and assertions

Finally, we must consider several questions about the nature of tactile agnosia, which will lead us to refute or assert some ideas, thus defining more precisely the scenario we have arrived at.

First of all, there is the question of the Schneider case, of which numerous indications and criticisms have been made throughout this book. Regarding the singular tactile disorder in this case, Goldstein and Gelb (1919) literally state: "clinically, we must speak here of a *transcortical sensitivity disorder*, a type of sensitivity disorder whose possibility had not been considered until now, and which is observed and clarified here for the first time." As is known, the authors think that touch itself is basically intact in all its functions, the loss of visual influence (due to a greater visual impairment) being what determines such a transcortical tactile disturbance. For all that we have exposed, this idea falls apart and it is no longer possible to accept the general conceptions of these authors about their patient.

However, the facts discovered in that patient have been verified in our patients (M and T) and in many others later on, demonstrating the true nature of the disorder. It should be noted that with the hypothesis of visual influence and the consequent transcortical defect of touch, Goldstein and Gelb proceed entirely within the classical theory of associations between cortical centers. Let us mention that a transcortical alteration of tactile agnosia had already been proposed ten years earlier by Kutner (1907) in the case he reports, although it refers to a frontal lesion.

In contrast, we dynamically interpret the whole set of sensory alterations in the Schneider case as an effect of the central syndrome. In this syndrome, dynamic repercussion is a mechanism that could be assimilated to transcortical action if the latter were understood not as an influence of specific centers (e.g., visual centers) that shape psychological functions, but rather as a nonspecific nervous influence that

determines the physiological state of general brain excitability. But this idea is not even suspected in the classical conception of transcortical disturbance nor in the interpretation of the Schneider case. Such an idea belongs entirely to the brain dynamics presented here.

It is convenient to bring up Schneider's case because the hypothesis of visual influence on touch has given rise to a certain state of opinion. The observations of Stauffenberg (1914) on visual agnosia have also been considered in the same sense by modern authors such as Kleist (1934), Lange (1936) or Delay (1935). Stauffenberg found that of 20 cases of visual agnosia, 15 also showed impaired tactile recognition without major sensitive defects that could be responsible for it. However, Stauffenberg himself makes no reference to specific influences (his observations predate the Schneider case). He rightly considers, following the ideas of his master Monakow (1905) that visual agnosia represents an "asemic disorder of the highest level" which never constitutes a single symptom, being accompanied by other tactile, language, orientation, intelligence, etc., alterations. For our part we must add that such multiple alterations are only an expression of the central syndrome, as already indicated at the beginning of the study of tactile functions (Sec. 18.3), as is the case in our patients and in Schneider's case. The objection of Thiele (1928) to Stauffenberg (1914), based on the fact that the referred multiple alterations can be explained by considering that in most of such cases there were vascular alterations (often with multiple cerebral foci), could reduce the value of Stauffenberg's interpretation. But this means nothing in our patients and in Schneider, in whom the lesion was only one and well circumscribed, which leads us to fully accept the point of view of Stauffenberg (1914, 1918).

The issue of influences still finds in Lange (1936) a very particular point of view. After interpreting Stauffenberg's observations from the point of view of visual influence (like Goldstein and Gelb in the case of Schneider), he wonders about the reverse question, i.e., in what way a tactile disorder should influence vision. He believes that this problem is of great importance even though it has hardly been raised. To this we reply that influences of any kind are non-existent; if the influence of vision on touch has come from an inconsistent psychological domain, for the reverse influence (touch on vision) not even that reason can be found, and ultimately it is the central syndrome that provides a solution for all these cases. In this regard, it should be noted that our patient T seemed to be only a "tactile case" in the early examinations of 1938; and much later on, visual disturbances were discovered (moderate concentric reduction, polyopia, etc.) which, according to the usual conceptions, could not be related to the same cortical lesion causing the tactile defects.

Was it necessary then to think that touch was affected primarily and vision secondarily? Not at all; both types of disorders existed by themselves and had to be attributed to a common fundamental disorder since they were caused by the same lesion. The fact that in T the touch defect was found first is explained by the fact that the lesion was in a parietal region of the skull quite far from the occipital pole and the subject did not complain of any visual defect. Thus, clinical attention was somehow directed to touch, particularly when considering that just after being injured, he had presented a clear loss of sensitivity on the contralateral side of the lesion (although the lesion did

not correspond to the tactile projection area). However, in patient M, the visual defect attracted attention first since the patient complained of visual symptoms and also the entry orifice of the projectile was in the occipital area, whereas the tactile defects were brought to light much later. In both patients (M and T) the disorder of elementary excitability (tactile and visual) was demonstrated when the dynamic action was discovered a year later, which also led to finding the auditory defect in them, as we exposed in the first part of this book (Sec. 1 and 2 in Vol.1).

In connection with this, it must be said that in the Schneider case the visual defect was the first to be discovered and only much later did the authors become aware of the existence of a similar defect in touch. In fact, the first publication on vision (1918) makes no reference to tactile disorders. When tactile defects were later found (second publication in 1919), it seems natural that they were put in relation to the earlier study on vision, and perhaps this order in the discovery of visual and tactile disturbances has had something to do with the idea of visual influence on touch. In any case, it is clear how much important data can remain hidden in the usual clinical observations that seem to exhaust the question, and therefore the impossibility of a theoretical interpretation in such a situation. A theory is viable only when the data are complete, the examination is exhaustive in all directions, and the results are consistent with each other in such a way that they can be interpreted from a common and simple point of view without the need to continually appeal to new hypotheses or principles.

The existence of multiple disorders poses a profound theoretical problem, and attempts to resolve it within the classical conceptions have been futile. Hesitations and contradictions are not infrequent in a same author. Thus, at the beginning of the detailed study on astereognosis by Delay (1935), when dealing with the question of the lack of parallelism between the alteration of the upper and lower tactile functions, this author considers that in the simultaneous alterations of anesthesia and astereognosis there is only a fortuitous coincidence, explainable for reasons of anatomical proximity and not for functional reasons. This is not admissible after all that has been exposed. However, in the final part of Delay's work, when referring to the recovery of tactile syndromes in which some are transformed into others, he rightly thinks that it would be justified to wonder whether tactile asymbolia represents only the most superficial and minimal alteration of the cortical tactile region. He goes on to say that to a higher degree would be alterations in spatial perceptions, shape agnosia, etc.; and to an even higher degree, cortical anesthesia. But then he states that it is impossible to decide, and finally he sticks to the hypothesis of a "gnostic center" in the brain different from that of the most elementary functions. Thus, he again moves infinitely far away from the functional and dynamic conception to accept the fundamental features of the classical theory. However, we know from our research that such a "gnostic center" corresponds to a central region that is responsible both for gnosis and for the most elementary excitability of contact sensation, as it results from the central syndrome.

All that has been exposed so far, namely: the impurity of the so-called pure cases, their revision in the light of the "Funktionswandel" [functional change], our study of logarithmic desynchronization, the multiplicity of sensory spheres affected

in agnosia, the data of the Schneider case and the research on our brain-injured patients, clearly demonstrate from multiple perspectives the shortcomings of classical conceptions. We could state that there are no pure cases of tactile agnosia or anything else, but rather a global impairment that, when the lesion is central, affects all sensory systems equally.

What is usually diagnosed and isolated in brain cases is only the most pronounced alteration, the predominant disorder, which will be the disorder of the most complex function. This is at the expense of neglecting other simpler and less evident alterations in the usual examinations, but which demonstrate the true nature of the disorder and the true mechanism of brain activity. The classical conception is therefore only a rough approximation, which in many cases does not meet all the requirements, and fails completely when dealing with *privileged cases* of the dynamics of the brain, such as our two brain-injured patients and Schneider.

Therefore, our dynamic conception not only interprets special cases but any other case, and fully satisfies the requirements of a coherent and simple explanation.

Recapitulation on tactile functions

TACTILE functions have been systematically investigated in all their aspects, resulting in a large number of new facts and new approaches derived from them. The global alteration of the tactile system in our brain-injured patients is, as we know, only one particular aspect of the multisensory alteration in the already exposed central syndrome. Given the properties of this syndrome, the tactile disturbances are of the same quantitative order as those previously studied in vision. As for the phenomena, the finding of spatial inversion in touch in 1946 (subsequent to inversion in vision) makes it possible to establish a complete analogy between tactile and visual sensory manifestations, and also leads to new advances in the research and to broad generalizations. Therefore, the general characteristics of visual functions in the central syndrome, summarized at the end of Vol. 1, are now equally applicable with the appropriate specific modifications.

1) *General excitability* shows an increase in chronoaxie and rheobase, as well as permeability to spatial summation (facilitation) and temporal summation (iteration) of the same order of magnitude as in vision, which is consistent with the homogeneous repercussion of the central syndrome. The tactile sensation presents a remarkably slow evolution, and in the extinction phase (where there is persistence) the sensation can be reactivated by summation. It is worth noting the underestimation of weights. Vibration sensitivity shows loss of high frequencies and overestimation of rhythm, due to the effect of increased fusion of intermittent stimuli, phenomena that can be explained by the decrease in excitability speed (increased chronaxie, increased refractory period, etc.). This is similar to what happens in vision with flicker-fusion frequency.

2) *Tactile sensations,* of a much simpler nature than colors in vision, show a notable decrease in sensitivity and a marked heterotactile interval by functional disaggregation (asynchrony). The sensation of pressure or contact appears as the primary activity of touch. The tactile sensation of pain presents a considerable enlargement

of the pressure-pain (tango-algic) interval. Similarly, thermal sensation is easily decomposed into pressure sensation and thermal sensation giving rise to the pressure-thermal (tango-thermal) interval. The increase in the thermal threshold, heat and cold, explains the significant enlargement of the neutral range of temperature.

Clinical dissociations of sensitivity are relative, not absolute; the explanation is functional and within the overall disturbance of the system. Sensitivities are dynamically classified into tangibility (pressure as primary tactile activity) and well-defined sensations (pain and temperature). A functional nexus between the former and the latter two is suggested.

3) *Tactile space* presents phenomena of special importance. In tactile localization, asynchrony causes a large abnormal interval between the simple sensation of contact and specific localization. The stimulation intensity determines intermediate effects such as various phases of spatial localization, which are: primitive projection (contact sensation without any localization), medial deviation, inversion, homolateral proximal deviation and specific (normal) localization. Each of these phases has several particularities. Such effects derive from two general phenomena: irradiation and proximal deviation. The weaker the stimulus and the earlier the localization phase, the greater the irradiation and proximal deviation. Tactile irradiation is homologous to color irradiation (pathological flat color vision). The elongated shape of tactile irradiation is related to the elongated shape of the tactile field or body schema. Proximal deviation is a manifestation of a concentric reduction of the body schema, with a preferential impairment of the distal regions and a functional privilege of the medial line. All this implies a structural change of the sensory field, giving rise to a field of residual activity, i.e., a poorly defined field (irradiation) and constricted field (centripetal deviation).

As for quantitative relationships, both irradiance and deviation (the latter easier to quantify) vary as a function of stimulus intensity, following logarithmic recruitment curves. Iteration curves are determined electrically as a function of the number of stimuli and as a function of the time interval between stimuli. Mechanical iteration with tuning forks only generates recruitment at low frequencies, due to the aforementioned characteristics of vibration sensitivity. These relationships are ratified in the asynchronous beam of strength-duration curves corresponding to the different localization phases (Sec. 22.2), where the higher levels suffer a disturbance several times greater than that of the lower levels. In addition, it is worth noting the post-stimulus summation effect that facilitation by muscular effort exerts on the residual trace of a tactile stimulus. The reactivation varies depending on the delay in the application of this facilitation, and is measured by the degree of deviation of the localization sensation (Table 22.3). The theory of tactile localization and sensory space in general is discussed, and the empiricist theory of the associative-kinetic local sign is refuted. Abnormal localization phenomena (deviation, irradiation, inversion) are the expression of a sensory field of residual function, in correspondence with the central nervous substrate, prior to any experience. Nor is there a local sign as a functional individuality, but rather multiple gradations according to the degree of organization of the nervous centers.

Several functions concerning spatial discrimination are studied. Spatial acuity (Weber) shows, as in vision, a considerable increase in the spatial threshold (reduction of acuity), which is interpreted as a spatial shrinkage (shrinkage of the body schema). The motion sensation produced by a moving object on the surface of the skin reveals a subjective acceleration and spatial shortening in inverse relation to the intensity of the pressure of the moving object. This is a result of spatial and temporal shortening. The spatial phases that arise are identical to those arising in the visual perception of motion. In the perception of figures drawn on the skin, systematic errors, illusions, dysmorphisms, etc., are produced due to the instability of the sensory field. In joint sensitivity (deep movement), as in the perception of a moving object on the surface of the skin, there are also intrinsic modifications (acceleration and shortening of the perceived movement) together with changes in joint localization by deviation and inversion. All this depends on the type of passive stimulation, which can be iterative (multiple oscillations of the same joint) or single (in this case very energetic and large, since an ordinary oscillation does not elicit sensation). Finally, it is worth mentioning the alteration of one' s own corporeality regarding reduction in size, weight, continuity, etc., as well as the reduction in stereognosis, "flat touch" due to tactile irradiation which erases the microstructure of the object being handled, etc.

4) *Perceived tactile orientation* presents phenomena of the utmost importance, which complement the study on tactile space. The finding of the inversion of tactile space (and also of auditory space) entails the generalization of the orientation issue and the establishment of new dynamic postulates of brain activity. The general laws are the same as for the orientation of the visual image, and now the process is studied more completely by combining the development of the inversion with that of the proximal deviation leading to a spiral development. The spiral trajectory of the inversion is determined by varying the intensity of a point stimulus in the three areas of the body (head, thorax with upper limbs and abdomen with lower limbs) that are autonomous regions of inversion. The most complete study of the gradual changes in perceived orientation and localization is performed by varying the pressure of a rectilinear stimulus on a distal part of a limb. The lower the pressure of the rectilinear stimulus on the hand, the greater the proximal deviation and tilt until reaching the inversion of the orientation with contralateral localization over the trunk while reducing the size of the line. The characteristic curves of sensory recruitment are obtained quantitatively.

Tests on changes in the direction of motion on the skin serve to ratify and complete the previous ones. In the perception of joint movement, there are complicated changes in both the direction of movement and proximal deviation of localization depending on the amplitude and energy of the passive joint oscillation, at the same time as the perception of the intrinsic aspects of the movement (speed, amplitude) is changing. Particularly notable are the phenomena of sensory-motor incongruence in the direction of movement in certain circumstances due to sensory inversion in voluntary movement. The orientation disorder is also manifested in the whole body schema, but only appears as a moderate tilt, since the body figure does not subsist (fades away) beyond a certain tilt.

Orientation disorder is also studied in more complex processes, the multiple sensory alterations that arise in gait being very demonstrative. The successive steps act iteratively on the nervous centers giving rise to a sensory recruitment of the walking direction depending on the number of steps, their frequency (addition effect in fast steps and no addition effect in very slow steps), and also on the step energy, with effects similar to those mentioned in joint sensitivity. Space and time in walking are reduced by the aforementioned spatiotemporal shrinkage. The subjective walking trajectory follows a spiral path. Typical sensory recruitment curves of the direction of walking by iteration as a function of the number of steps are obtained, and are analogous to the curves in vision on the perceived direction of motion as a function of the number of metronome oscillations. Finally, alterations in the localization of the movement of the lower extremities, the sensation of translation on the ground, the subjective orientation of the whole body during walking (turns, subjective slope, etc.) are also studied. Of another type are the singular sensory-motor phenomena in objective postural deviations, static (head, trunk, limbs) and in movement (finger-pointing, walking, etc.), interpreted as an induced deviation secondary to a primary sensory deviation.

Orientation theory is a topic of general significance, and poses very fundamental brain questions. It can be seen that both proximal deviation and inversion are involved in spatial localization, and when both are combined in the developmental process by increasing stimulation, a spiral development of the sensory field is obtained (which constitutes a law of sensory organization in the brain dynamics presented here). Within asynchrony, the origin of inversion lies in the marginal areas of the cerebral cortex, with inverted and contralateral sensory projection, whereas re-inversion is the result of what we have called central areas. There is thus a direct correspondence between the brain field and the brain state: reduced and inverted sensory field corresponds to brain reduced to marginal activity; enlarged field with normal orientation (reorganized) corresponds to an enlarged brain with the mass of the central areas. It should be noted that the process of sensory organization involves, to different extent depending on the case, both the anatomical configuration and the action of the mass (maneuvering mass of the mentioned central areas). Ultimately, the spiral development of the sensory field reveals a psychophysical isomorphism.

5) *Tactile schema* comprises a set of functions that are in continuity with the previous ones. The body schema presents different levels of organization: somatic model, postural model and praxis model. The somatic model is the most rudimentary level and corresponds to the simple corporeality that only gradually emerges through tactile stimuli or through facilitation by muscular effort, since it is originally vanished. Due to this absence of somatic model, the initiation of praxis is hindered, the facilitation by muscular contraction appears in a quasi-reflex form, activating the model to varying degrees; it is a disorder of asomatognosia type. The postural model means further development, which is indispensable for the correct spatial localization of tactile stimuli. The general posture of the body suffers a subjective deviation due to the orientation disorder. More important is the disorder of segmental pos-

tures, which are perceived as deviated towards the neutral posture (sensory reduction). This phenomenon presents diverse degrees of reduction, and also certain illusions in a normal subject. The praxis model is considered in a broad sense, and many different types of action alterations have been addressed. Dyspraxia in the rudimentarily developed model consists of automatic actions guided by illusions about one's own body (phantom body, anosognosia, etc.). There are also several inversions in praxis that cause autotopagnosia by sensory inversion of the direction of movement, as well as the peculiar phenomenon of contralateral canalization of movement initiation under special conditions.

Dyspraxia in the coupling of the praxis model with external space refers to defects in spatial orientation (pointing with the finger to oneself and to external directions), and to instability in left-right differentiation, which is a general visual and tactile spatial disorder (mirror writing, etc.). Very noticeable is the egocentric prevalence due to the failure of allocentric orientation and action. This is interpreted as a result of taking as a reference only the neutral model of the body (illusion of neutral position). The dyspraxia in the requested actions comes from the instability of the body schema. It is worth mentioning the defect in the initiation of praxis as well as the defect in constructional praxis, this varying according to the degree of automation of the actions, praxis discontinuity being the most characteristic phenomenon. The various disorders of the praxis model correspond to various forms of apractognosia with certain general defects such as neutral illusion, unawareness of the defect and constructional deficit. The gradual series in apractognosia shows functional unity, there is continuity between phenomena of different functional levels and there is no opposition between sensory and gnostic functions, but they all show the same pattern of organization and are part of a functional growth (growth of the sensory field).

The schema in manual touch constitutes a particular aspect of the body schema. Tactile recognition of objects is impaired due in part to distal tactile disorder affecting manual activity, being spontaneously compensated by facilitation by muscular effort and pressure in handling objects, although errors in manipulation remain in special tests. Object recognition can undergo stereognosis reduction and stereognosis transformation, and at a higher level, a defect in the tactile schema (asymbolia). Tactile recognition goes through different phases (analytical, deductive, etc.), and there is fragmentation of the function, indirect recognition, etc. The typical fragmentation (discontinuity), constructional inability, etc. of the agnostic condition is the result of a reduction in the dimension of the schema, schema that becomes "small" and does not encompass the whole, but only independent parts. This is no different from the alteration of other dimensions of the sensory field; there is in fact a change of scale.

Finally, several controversies and problems about tactile agnosia are easily clarified by the facts and concepts of the brain dynamics presented here. Thus, the disproportion of impairment between higher and lower functions is consistent with a logarithmic asynchrony, and the multisensory disorders are consistent with the central syndrome described here. Therefore, this conception of brain dynamics is supported by its own data and by issues raised outside of it.

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SUPPLEMENT I*

Human brain functions according to new data and physiological basis

An introduction to the studies conducted on brain dynamics

by

Justo Gonzalo

* **Original editions (in Spanish):**

GONZALO, J. (1952). "Las funciones cerebrales humanas según nuevos datos y bases fisiológicas. Una introducción a los estudios de dinámica cerebral", *Trabajos del Instituto Cajal de Investigaciones Biológicas,* XLIV: 95-157.

Suplemento I in:

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> … I have been of the opinion that all the knowledge which men can have of nature must necessarily be derived from the principles of geometry and mechanics; for all other notions we have of sensible things, being confused and obscure, cannot serve to give us knowledge of anything outside of ourselves. (Translated from French.)
>
> René Descartes

> Until a physical-chemical explanation is found for a biological phenomenon, it will be commonly incomprehensible. If the veil is ever lifted, we shall be surprised not to have guessed from the beginning what was hidden.
>
> Jacques Loeb

> If one can measure what one speaks about and express it as a number, something is known about it. If not, the knowledge is poor and quite unsatisfactory, whatever one is dealing with.
>
> Lord Kelvin

INTRODUCTION

These quotes are doubtlessly a difficult demand to be placed on brain pathology, and may seem to some to be utopian. Nevertheless, they refer to the general nature of our work and avoid many explanations from the outset. This orientation is not at all a criticism of more conventional paths, but rather points towards a subsequent stage which should be reached in a natural way.

The study many years ago of a multitude of cases of brain injury has given rise to new data which help to determine human brain functions according to much more physiological *principles* than those established to date. The present report is restricted to highlighting the most demonstrative aspects of the research, and refers the reader to the original publications for more details. I shall first present a summary of my

work on brain dynamics [1941[1], (1945-50)[2]] followed by indications of complementary studies conducted in the period 1950-1952[3].

The progress may come from the use of other methods, and also from an unbiased examination of simple observed facts. Both approaches have a place in our study. Many issues have been advanced here thanks to a more physiological and experimental analysis than is customary, and especially to the benefits of *quantitative* procedures. Measurements that provide precision to observations serve as objective controls on sensory phenomena and provide quantitative laws of the processes.

The two published volumes of *Dinámica Cerebral* [Brain Dynamics] (see footnote 2) present studies on two brain-injured patients, patient M and patient T, with a high degree of topographic similarity in their left parieto-occipital cortical lesion, although the *magnitude of the lesion was greater in* M. The observations cover a time period between 1938 and 1948. The innovative nature of this research appeared in 1939 with the discovery of dynamic action phenomena. In 1941, the most important experiments and arguments were reported (see footnote 1). The first volume was published in 1945. It covers general findings and the systematic study of visual functions. The second volume, published in 1950 covers tactile functions and expands on the principles introduced in the first volume.

1. PHENOMENA OF DYNAMIC ACTION

This type of phenomena is treated in the first place due to its general nature, because it clarified different agnosic disorders in the prototype M and T cases and, above all, because it indicated a physiological direction from the outset of this research in 1939. The *dynamic action phenomena* (asynchrony, facilitation[4] and repercussion) consist of transformations of central nervous excitability.

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[1] GONZALO, J. (1941). "Investigaciones sobre Dinámica Cerebral. La dinámica en el sistema nervioso. Estructuras sensoriales por sincronización cerebral" [Investigations on Brain Dynamics. Dynamic action in the nervous system. Sensory structures by brain synchronization]. Unpublished report presented to the Consejo Superior de Investigaciones Científicas [Spanish National Research Council]. Madrid 1941.

[2] GONZALO, J. (1945, 1950, 2010). *Investigaciones sobre la nueva Dinámica cerebral. La actividad cerebral en función de las condiciones dinámicas de la excitabilidad nerviosa.* Vol. 1 (1945), Vol. 2 (1950). Consejo Superior de Investigaciones Científicas, Madrid. Included in the facsimile edition: *Dinámica Cerebral,* published in Spanish by the Red Temática en Tecnologías de Computación Natural/Artificial (RTNAC) and the Universidad de Santiago de Compostela (USC), 2010. Open Access: http://hdl.handle.net/10347/4341

[3] This new part with respect to Volumes 1 and 2 of "Brain Dynamics" is described in Secs. 7-11 of this Supplement. In addition to the new cases reported on central syndrome and chronic tilted vision, section 7 is of special interest because it is devoted to the important topic of functional cortical gradients first published in the 1952 article of which this Supplement is its English translation. This alone justifies the inclusion of this Supplement in the present edition.

[4] The term used in the original Spanish version is 'refuerzo' (reinforcement in English), and has been translated as facilitation.

Asynchrony is the separation of sensory phenomena which are normally united and not dissociated, in such a way that pathological sensory intervals appear, and the normal process of all-or-nothing is broken down into phases which are partial reactions. This sensory desynchronization, originated by a nervous asynchronism, permits an analysis of the sensory complex by penetrating its structure. A very weak tactile stimulus is only perceived as a mere sensation of contact lacking the possibility of spatial localization, whereas localization is normal when the stimulus is stronger. Small, distant or weakly illuminated objects appear to be tilted or even inverted although when stimulation is much greater (large visual angle, more intense illumination etc.) the object is perceived as being upright, and so on with other examples. The abnormal interval is then understood to be recruited or surpassed by the intensification of stimulus. The amplitude of such a pathological interval is dependent on the magnitude of the cortical lesion, then being quite pronounced in patient M.

It must be pointed out that the most striking abnormal phenomena (inversion) *only occur under minimal stimulus* and are absent under a certain intensity of stimulus. Subjects may thus behave normally in everyday life and even be unaware of a great part of their anomalies. The excitability disorder is general because there is an important deficit of elementary excitability (in intensity and time), and the mentioned asynchrony is only a more complex aspect.

Excitability disorder has a special feature in the *facilitation* phenomenon (by muscular effort or by multisensory effect). It corresponds neurophysiologically to the "spatial" or simultaneous summation. It was found that intense muscular contraction in the subject was able to replace the loss of elementary excitability and thus reduce the desynchronization. When the image of an object appears to be greatly tilted, this type of facilitation straightens it instantly and simultaneously clears the vision, dilates the visual field, and is similarly shown in touch and hearing. A distinction must thus be made between the "inactive" state of the subject and the state under facilitation which is much more favorable. However, no matter how intense the facilitation, it is unable to annul the entire deficit. Together with this muscular effect there are other types of spatial (cross-modal) summation such as the "bi" effect, i.e., the effect of binocular summation in which one eye reinforces the other, i.e., all visual functions being better when both eyes are used instead of one. The same occurs with the other senses: the combination of muscular effort and the bi effect results in increased action. Experiences of multisensory (cross-modal) summation are also described. For example, a certain kind of tactile stimulation can improve visual function. This facilitation has nil effect in a normal subject, and whereas it is quite clear in subject M, it is scarcely notable in subject T.

Along with abnormal permeability to spatial summation, there is also permeability to temporal or successive summation, i.e., to iterative excitation by accumulation of successive stimuli, a phenomenon which is also absent in a normal subject. It is highly significant in subject M and only quite clear in subject T.

Finally, the phenomenon of cerebral *repercussion* of the lesion is what is most opposed to the traditional ideas of specific anatomic localization in the brain. The cortical brain lesion in M and T is located in a "central zone" equidistant from the visual, tactile and auditory areas, their projection areas being not directly impli-

cated although it can be demonstrated that all these sensory systems are clearly affected as their activity is reduced in all types of function, from simple excitability to the most complex ones. Furthermore, both sides of the body are affected almost equally in spite of it being a unilateral lesion. This form of repercussion gives rise to the new 'central syndrome' of the cortex.

These phenomena of dynamic action may be said to initiate the transition from the traditional or static brain concept (simple clinical empiricism) to a dynamic one (physiological causal analysis), proven not only in cases M and T, until now models of this research on *brain dynamics*, but also in many others.

2. MAGNITUDE AND POSITION. THE PROBLEM OF BRAIN FUNCTION LOCALIZATION

From the examination of patient M, patient T and others, it was established in the aforementioned 1941 report (see footnote 1 in the Introduction) that the effect of cortical lesions depends on just two factors: the *magnitude* and the *position* of the lesion. Position determines the type of disorder distribution in the brain system, i.e., the topography of the above mentioned 'repercussion.' Magnitude or degree of cortical destruction determines the intensity of the disorder.

2.1. Position

With respect to the position of the lesion, three general cortical syndromes can be distinguished: central, paracentral and marginal (or of the projection area), as shown in Fig. 1.

The *central syndrome* is the new syndrome mentioned in the description of repercussion, and is characteristic of this research of brain dynamics. The site of the lesion is geometrically "central" or equidistant from the visual, tactile and auditory projection areas. These sensory systems are equally affected on both sides and in all their aspects. It is thus a homogeneous repercussion in the whole sensory brain in both hemispheres. The sensory affection thus has maximum symmetry. For example, concerning the visual system, a symmetric concentric reduction of the visual field is found.

The *paracentral syndrome* is somewhat similar to the central syndrome. It is a form of transition as its name indicates, and the repercussion (distribution of the disorder) is asymmetric. The lesion is in an intermediate site between the "central" zone and the projection area, or closer to the latter. There are thus three paracentral syndromes: visual, tactile and auditory. Functional involvement may be somewhat general while naturally predominating over the nearest projection, with the contralateral side predominating more than the homolateral side of the lesion. The type of alteration in the visual field is asymmetric concentric reduction, corresponding to a hemianopsia, with varying degrees of macula conservation and homolateral constriction: the two halves of the field undergo constriction by the identical mechanism but much more so on the contralateral side. In touch and hearing, a quite small deficit may be shown contralaterally, whereas it is practically null homolaterally.

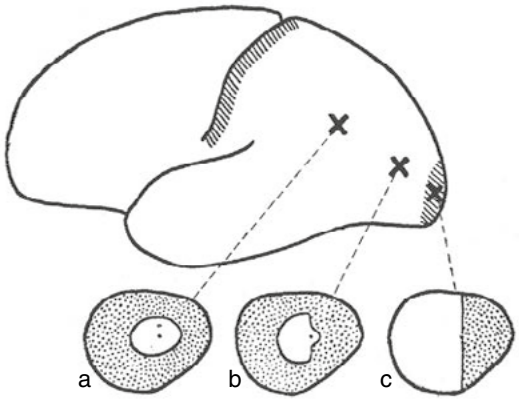

Figure 1. Scheme of positions of cortical lesions (x) and corresponding types of visual field. a: symmetric concentric reduction in the central syndrome. b: asymmetric concentric reduction in the paracentral visual syndrome. c: hemianopsia in the projection path syndrome (here called marginal or peripheral). The more central the syndrome, the greater the lesion must be to provoke a deficit in the visual field.

The *marginal syndrome* lacks repercussion. The lesion is in the projection area (visual, tactile or auditory) with the affectation completely restricted to the contralateral half of only one sensory system. In the visual field, the defect is a simple hemianopsia lacking other components.

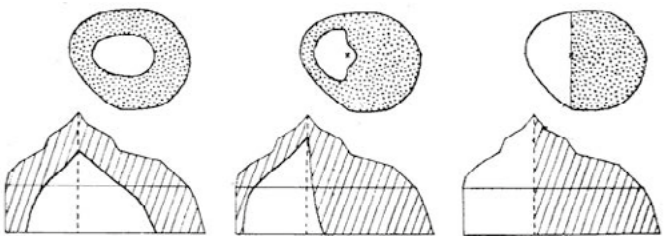

Figure 2. The pathological forms of each visual field (above), and the respective visual profile intensity. In symmetric concentric reduction, i.e., in central syndrome, there is a *depression* (decrease) in the sensitivity profile with the form more or less maintained (left). In paracentral syndrome (in the middle), the depression is less homogeneous (asymmetrical). In the projection path syndrome (right), there is a visual *suppression* for one half, according to the anatomical disposition of the cortical projection.

Thus, when the lesion moves from the "central" zone of the cortex to the "marginal" zone (Fig. 1), the repercussion is reduced, and the general disorder (bilateral and extended to all systems) becomes restricted to only one system on its contralateral half. The central syndrome is something totally new. The marginal syndrome traditionally refers to the projection areas. The paracentral syndrome is

also known but not correctly interpreted and now, considered involved in the asymmetrical repercussion effect, it is presented as justly interpreted. Furthermore, it is worthwhile noting that whereas the marginal syndrome is more a syndrome of projection paths, the other two syndromes correspond to the nervous centers. In a more physiological consideration, the marginal syndrome is a functional *suppression* (interruption) whereas the others should be regarded as syndromes of functional *depression* (decrease) (Fig. 2) which present the above mentioned phenomena of dynamic action.

2.2. Magnitude

The degree of alteration, i.e., the degree of functional decrease (depression) depends on the quantity of cortex destroyed. The best examples of this in the whole work are the M and T cases, which, belonging to the central syndrome, are distinguished by the intensity of the symptoms in close concordance with the different magnitude of their respective cortical lesions. The concept of magnitude is characteristic of brain dynamics given its extreme *quantitative* nature, radically opposed to the traditional, somewhat *qualitative* doctrines. The parallel between the magnitude of the lesion and the degree of alteration leads to the establishment of continuous transitions in the various abnormal phenomena, and the exclusion of independent or genuine qualitative defects. This is related to a problem which was raised long ago in brain pathology although generally avoided by most authors. Thus, the difference between paralysis and paresis, anesthesia and hypoesthesia, hemianopsia and hemiambliopia, etc., may only lie in different degrees of involvement of the same function.

The simplification of the thorny problem of functional localization in the brain by taking into account the two factors discussed here (magnitude and position) leads to a dynamic solution of the problem. The repercussion phenomenon, particularly in the central syndrome, may seem to put an end to the traditional theory of the specific centers. It would lead to the postulation of a dynamic unit of the whole brain system whose degree of involvement only depends on the magnitude of the lesion. The position factor, on the other hand, determines different paracentral syndromes, and firmly supports the heterogeneity of the brain system. It may thus be concluded that *instead of specific centers there are dynamic effects in the brain system according to the magnitude and position of the lesion.* This statement has been considered as the 'first principle' of the brain dynamics developed in this research. We shall see that the concept of brain gradients, introduced in Sect. 7, includes and combines in a more complete way these two aspects, magnitude and position.

3. VISUAL FUNCTIONS. THE PROBLEM OF SENSORY ORGANIZATION

A further fundamental problem is that of functional organization which, although related to the problem of brain function localization, has its own character. Here we shall address the problem only for sensory functions, which offer a wealth of

manifestations difficult to match by motor functions. Given the central syndrome in M and T, a structural analysis is possible in any of the three main sensory systems- visual, tactile and auditory. Apart from their individual peculiarities, they show a common organizational plan. Moreover, a great number of manifestations may be unified under a general principle which is dealt with in a further section.

The analysis of the functions is focused here on the activity of the sensory field, which in our subjects undergoes a functional "depression" derived from a deficit of cerebral excitability. The most elementary excitability is reduced, and there is an abnormal capability to spatial and temporal summation. The normal sensory intervals of differentiated sensitivity are greatly dilated, and further intervals arise where they are normally absent, as set out in the paragraph on *asynchrony* in Sec. 1.

Within each sensory system, the excitability disorder involves all types of activity, and increases with increasing physiological demand. This gives rise to the typical exclusion of more complex activities according to a well-defined physiological order, constituting the so-called 'dynamic reduction.'

In synthesis, the depression (reduction) of the sensory field does not imply a mere smaller field but a *field of residual function* in which every activity is affected, whereas the general scheme of organization is conserved, although functioning on a different numerical scale from the normal one. In short, the magnitude of cortical lesion causes a *change in the magnitude of the sensory scale*, i.e., the "sensory dimensions" become different, as we shall see further on.

The monographic study of visual functions on the basis of the M and T cases occupies a large part of the precedent book *Dinámica Cerebral*.[5] More attention is placed on patient M due to his more intense disorders, considering the T case as a complement. Other cases are occasionally reported, and the most characteristic phenomena of this study have been verified in paracentral cases.

Many phenomena are new or almost unknown, and an effort is made to provide a physiological and quantitative basis for them and to give, even in the details, a rational explanation. Much of Vol. 1 of the mentioned book, from Sec. 8 to Sec. 16, including Fig. 4.1 to Fig. 16.3, deals with visual functions. A summary of the issues addressed in each of the five visual functions studied is as follows:

i) *General excitability*: Electrical excitability (strength-duration curves), laws of iteration, effect of facilitation phenomenon. Light excitability curves, light adaptation, flicker fusion frequency.

ii) *Color vision*: Vision of the spectrum, differential color sensitivity, photochromic interval, dyschromatopsias and chromatopsia, inversion of color isopters, chromatic induction (abnormal increase of edge contrast, abolition of negative afterimages). Color processing in the brain.

[5] *Brain Dynamics*, Volume 1.

iii) *Visual forms*: Concentric reduction of the visual field, relationships of excitability in the visual field, organization of the visual field (monocular polyopia, pseudofovea, etc.). Flat colors or color irradiation (distorted color perception). Visual acuity. Perception of motion. Perception of figures and objects (metamorphopsia, etc.).

iv) *Visual image orientation*: Inverted vision to varying degrees, characteristics of this disorder, fundamental experiment, synchronization by facilitation (variation according to the state of the central nervous system), orientation of the visual image according to the state of the receptor, various complementary tests. Mechanism in the orientation of the visual image.

v) *Schema* in visual perception: Agnosia, visual behavior. Structure of the visual agnosia. Alteration of orthogonal orientation.[6] Allocentric orientation disorder.

Table 1 is intended to give a quick overview of the study conducted on the M and T cases, their disorders, and the effect of facilitation on M.

In general terms, chronaxie and visual rheobase are ten times greater than normal in M, and four times in T. The phenomena of summation are very evident in the former, and much less so in the latter. M's visual field is considerably reduced but recoverable (recruitable) by both intense stimulation and facilitation. In contrast, the reduction of the visual field is moderate in T. The same happens in the reduction of visual acuity, in the abnormal irradiation of colors due to weakness in specific spatial localization, in the color alteration (blue-violet is more affected because it is the slowest or that of greatest chronaxie), in the instability of forms or figures and in the visual perception of motion.

The orientation disorder in patient M consists in the fact that the visual image of objects is considerably tilted, almost reaching inversion. Accurate measurements indicate a rotation of the image of up to 150°, whereas only reaching 20° in patient T. Visual agnosia phenomena are extreme in case M and moderate in T. The former is able to interpret very few drawings of objects, and failed completely in mixed or overlapping drawings, whereas the latter only has difficulties with this type of drawings.

The following brief remarks refer to the five visual functions indicated in the left column of Table 1:

As for *general excitability*, the corresponding experiments provide a very objective basis. The electrical stimulation of the retina and the pathological summation curves (Figs. 3 and 4) are highly accurate. Similar results were obtained on light stimulation. Light adaptation is slower and less wide than in normal cases. Flicker fusion frequency was also measured.

[6] See Sec. 16 in Volume 1 of *Brain Dynamics*.

Table 1.[7] Visual functions according to three types of physiological level (M inactive, M under facilitation by strong muscular effort, T inactive). OD: right eye; OS: left eye.

	M inactive	**M under facilitation by muscular effort**	**T inactive**
Excitability	OD: rheobase 14.2 V, chronaxie cap. 3.5 µF. OS: more disturbed.	OD: rheobase 9.5 V, chronaxie cap. 2.7 µF.	OD: rheobase 7.8 V, chronaxie cap. 1.4 µF.
Colors	*In medium light*: yellow-blue blindness, etc., intense chromatopsia, pronounced inversion of color isopters, intense alteration of chromatic induction phenomena.	*In medium light*: practically normal color vision. *In very low light*: phenomena of the inactive state.	*In very low light*: tritanomaly (weakness to blue), traces of fleeting chromatopsia, partial inversion of color isopters.
Forms	Visual field up to 6°. Acuity: monoc. 1/25, binoc. 1/10. Strong color irradiation *in medium light*. Severe loss of motion perception. Unstable and diffuse shape perception. Very slow and successive perception.	Visual field up to 40°. Acuity: monoc. 1/8, binoc. 1/6-1/4. Irradiation only from red *in medium light*. Motion perception much better than in the inactive state. Better and faster shape perception than in the inactive state.	Visual field up to ~ 50°. Acuity: monoc. 1/3 - 1/2, binoc. 2/3. Weak irradiation only from red. Slightly altered motion perception. Somewhat unstable shapes, and somewhat slow perception *in very low light*.
Orientation	OD max. tilt image: 145°. OS max. tilt image: 170°. Binoc. idem: 115° *In very strong light*: tilt image about 5°.	OD: from max. tilt image in inactive state and same stimulus, a strong re-inversion is obtained (from 145° to 30° and even 20°). OD max. tilt image: 97°. Binoc. max. tilt image: 27°.	OD max. tilt image: 25°. OS max. tilt image: 16°. Binoc. max. tilt image: 10° or less.
Schema	*Binocularly and in strong light*: illusions, disaggregation, concrete behavior, orthogonal failure, egocentric orientation.	*Binocularly and in strong light*: fairly well in general, but serious defects in complex tests, orthogonal and allocentric orientation. *In low light*: as in the inactive state.	Mistakes only in complex tests, although there are traces of weakness in all of them. Normal orthogonal orientation even in very low light.

[7] This Table is the same as Table R2 in Vol. 1 of *Brain Dynamics*.

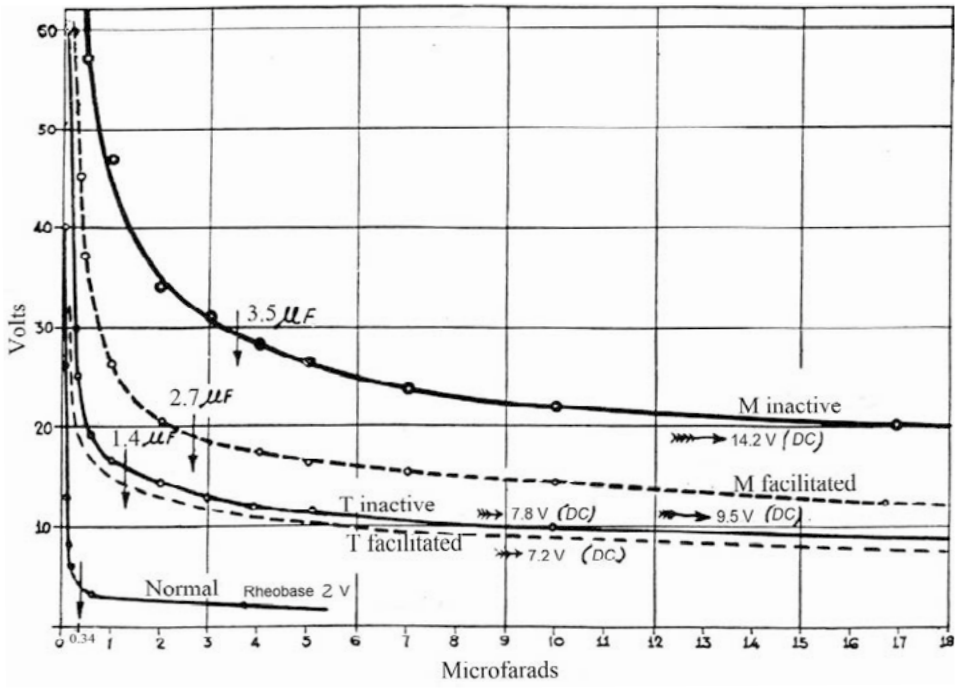

Figure 3. Strength-duration curves (volts versus microfarads) obtained by retina stimulation, with capacitor discharge (cathode on eyelid) to obtain minimum phosphene. Vertical arrows indicate chronaxie capacitances. Compare the values of M and T with those of a normal case, the change of the curve in state of facilitation under strong muscular effort in M, and the smallness of facilitation effect in T. This figure is similar to Fig. 4.2 in Vol. 1 of the book *Brain Dynamics* except for the missing quantity curves here.

The study of *colors* is somewhat difficult in these patients due to their color sensory defects and also to their perceptive and agnosic defects. In the first aspect, color weakness occurs in the whole spectrum but is stronger in the yellow-blue pair, particularly in the latter color (tritanopia-tritanomaly). The photochromic interval is greatly increased and it becomes complex in some colors. Sensitivity to color discrimination is highly diminished. White has a green tinge (chromatopsia) due to a deficit of color ingredients etc., each phenomenon fitting the known rules of color blindness.

Concerning visual *form* perception, defects in the *visual field*, *acuity* etc. (Fig. 5) become highly useful numerical data for brain dynamics. We focus on concentric reduction as it is generally an obscure and overlooked matter. Here it is defended in its organic nature in accordance with the central syndrome. *Color irradiation* or "flat colors" described by Gelb,[8] is studied physiologically according to the relationships of excitability. The peculiarity of the disorders in visual perception of *motion* lies in

[8] GELB A. (1920), reference in Vol. 1 of *Brain Dynamics.*

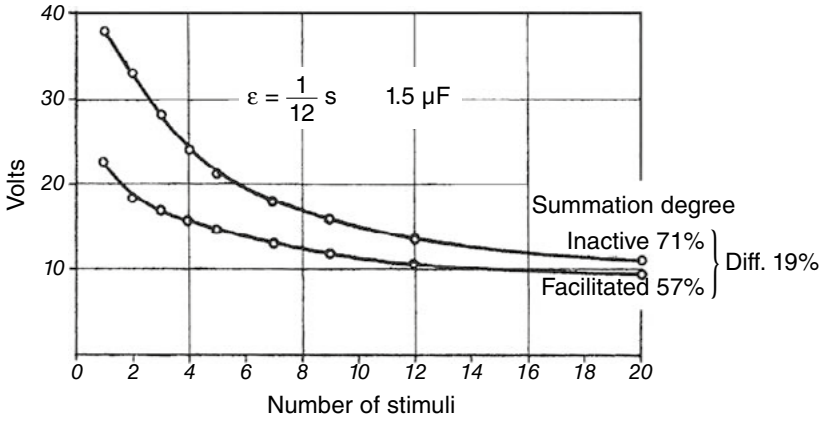

Figure 4. Retinal iterative aptitude in subject M, which is null in a normal subject. Curves for inactive and facilitated states (volts versus number of stimuli) with stimulation as in Fig. 3, for a constant time interval between stimuli $\varepsilon = 1/12$ second and a constant duration of stimulus (capacitance = 1.5 μF). Degree of summation is the difference between the voltage of a single stimulus that produces sensation and the lowest voltage when an unlimited number of stimuli produces sensation, expressed in %. This figure is the same as Fig. 4.7 in Vol. 1 of *Brain Dynamics*.

the phenomena of apparent acceleration and reduction of traveled distance when the stimulus is weak. It is a spatial and temporal contraction. In addition, there is a reversal of the direction of motion. It is the starting point for fundamental issues.

Orientation disorder in relation to visual image inversion has weak but numerous precedents. However, the first objective observation of such an unusual phenomenon and its in-depth experimental study belong entirely to this research on brain dynamics. It is treated separately for many reasons (see next section on *spiral development*).

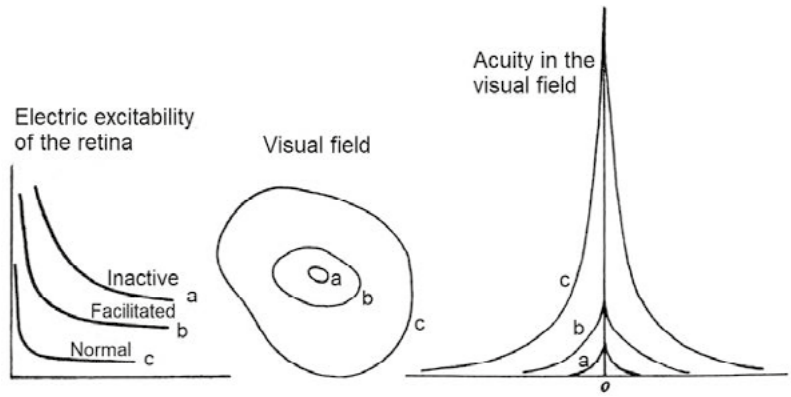

Figure 5. Correspondence between curves of excitability, visual field size and acuity in the visual field, for inactive state (a) and facilitated state (b) in subject M, and in a normal case (c). Acuity in central vision in M descends to 1/20 in the inactive state, and 1/10 in the facilitated state. This figure is similar to fig. 11.4 in Vol. 1 of *Brain Dynamics*.

Visual agnosia, approached here according to the *schema* concept, is caused by a disaggregation (disintegration) of schema function, and a diffuse conception of objects and their schematic representations. More importantly, although it is the defect of a peculiar stage of sensory organization, it can by no means be admitted as an independent phenomenon. Contrary to the conventional idea, the defect is considered parallel to the remaining sensory defects of the system, as shown in Table 1. The most outstanding contribution of the new phenomena in this field is probably the finding of the *orthogonal disorder*,[9] a singular defect of spatial orientation in which objects and figures are recognized independently of their orientation, i.e., inverted or upright portraits seem the same; written text, in particular, can be read whether it is oriented normally (upwards) or rotated 180°, without the patient noting any difference. The modifying effect of summations (bi effect, facilitation by muscular effort) on this disorder is very strong. For several months after being injured, patient T clearly showed this disorder. Afterwards, it disappeared completely with some recovery from the brain damage, and only appeared fleetingly in a rudimentary form as a consequence of a strong epileptic seizure. Another original contribution is the description of the substitution of *hallocentric* spatial orientation in M by mere *egocentric* orientation.[10]

4. SPIRAL DEVELOPMENT

This final part on visual functions refers to the phenomenon of inverted vision. To address this phenomenon, the experimental and quantitative analysis that characterizes this research on brain dynamics is extensively deployed. The spatial inversion process is connected to other sensory manifestations already analyzed (visual field, colors, acuity, motion perception, etc.). It is a functional complex which develops in accordance with the inversion and re-inversion process, revealing the structure of the sensory field according to a spiral development, and in accordance with both physiological relationships of brain excitability and anatomic textures of the cortex.[11]

Inverted vision was discovered by chance in patient M in 1938. This inversion is not complete, and although it is difficult to determine its precise value, it seems to reach a rotation of 160°, a very considerable alteration that is in relation to his other disorders. In patient T there is only a slight tilt of some 20°. Other cases of brain injuries with small tilts were observed by the author prior to M. The phenomenon in M consists in seeing objects greatly tilted and even inverted, rotated in the frontal plane, the rotation being dependent on the size and distance of the object observed, i.e., on the subtended angle of vision. There is also a dependence on the intensity of illumination and exposure time. Thus, a nearby object appeared to be greatly rotated if it was only seen for an instant. The phenomenon was only properly understood and studied in depth when the action of facilitation was understood. Then, case T

[9] See Sec. 16 in Vol. 1 of *Brain Dynamics*.
[10] See Sec. 16.2 in Vol. 1 and Sec. 27.4.2 in Vol. 2 of *Brain Dynamics*.
[11] See Secs. 12-14 in Vol. 1 and Sec. 26 in Vol. 2 of *Brain Dynamics*.

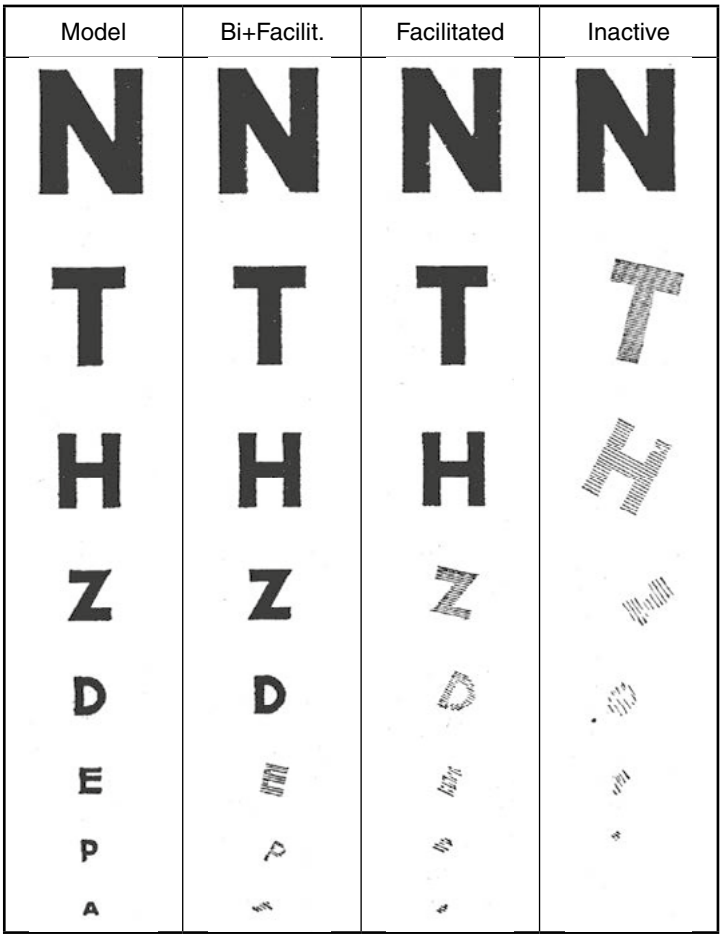

Figure 6. Tilt and fading of the configuration perceived by patient M, according to size of letters and states of summation. Left column is the model to look at. The model is situated at a convenient and fixed distance in such a way that patient M, with the right eye and in inactive state, obtains completely upright (correct) vision only for the tallest letter (N). Then, the smaller letters seem tilted and are illegible beyond a tilt of 40° (fourth column). For the state under facilitation by strong muscular effort an analogous effect is obtained but the perception is improved (third column). If there is also binocular vision, perception is further improved (second column).

was interpreted as an attenuated manifestation of the phenomenon. It is not merely an exchange between above and below, but essentially a gradual rotation according to the energy of stimulation, and it occurs for any orientation of the object (vertical, horizontal). In fact, the phenomenon was discovered in horizontally moving objects that were perceived as moving in the opposite direction.

In patient M, the orientation disorder does not disturb daily life and, paradoxically, almost goes unnoticed, since *no tilt is perceived in objects seen with good vision.* A notable rotation of 50° corresponds to hazy vision which impedes the recognition of

the object, and beyond 90° and nearing inversion, the object is reduced to a shadow that is ignored; thus, great rotations are practically excluded. Subject M usually perceives an almost correct image with small tilts that do not cause disturbance. Thus, to obtain large tilts and inversions it is usually necessary to provoke them, but without having to using any special instruments; it is sufficient for the subject to concentrate on peripheral vision in an elongated object moving away or from one side to another or from above to below etc. Such simple tests helped to establish the appropriate stimulation conditions.

In order to give an idea of the questions arising from spatial inversion, in this section we analyze 1) the phenomenological aspect, 2) the quantitative aspect, 3) spiral development of the sensory field, 4) brain mechanisms and 5) nerve decussations.

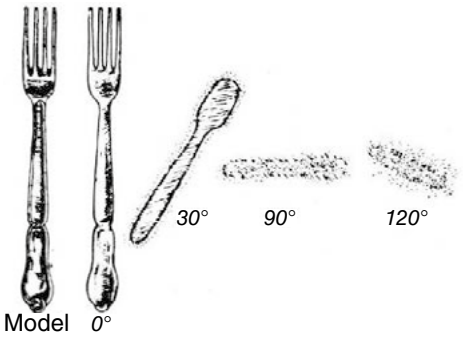

Figure 7. Fading of the shape through the inversion process. There is also a reduction in size and in light contrast, etc. A metal fork of 20 cm at a distance of 1.5 m is perceived as nearly inverted by M with only one eye, in an inactive state and in medium illumination.

1) Several examples serve to give a *phenomenological* idea of the process. Once beyond a perceived tilt of 40°, letters appear to be unrecognizably blurred (Fig. 6). A more pronounced rotation can be studied in more elongated objects. A vertical fork moving away from the subject, may be taken for a spoon when is perceived as rotated 30°. At 90° it looks like a smaller, elongated diffuse object; at 140°, it is on the verge of becoming a shapeless spot which is hardly distinguished from the background. The experimental conditions are: M in an inactive state at 1.5 m from the object, using one eye and under medium lighting (Fig. 7). A similar perception occurs for human figures but at 5 m and with low illumination. If a moving vehicle is seen in an inverted direction of motion, it appears as a mere blurred spot moving over a much smaller trajectory and at an overestimated speed. Thus, together with the change of direction of motion, there is a parallel alteration of sensory *intensity*, *space* and *time*.

2) A more extensive experimental study shows that the process conforms perfectly to certain *quantitative relationships*, as suggested in 1941 (see footnote 1) and developed in 1943. Asynchrony causes an abnormal sensory interval between pathological inversion and the normal perception, an interval which must be recruited (overcome) by an increment in the stimulus. The basic relationship to be determined

is simple (orientation perceived versus stimulus intensity), although it can show a great diversity of aspects due to the participation of many factors in the excitation (stimulus, receptor, central nervous state), and other experimental conditions.[12] Here we shall focus on the simplest and most important aspects (Figs. 8-10). Fig. 8 illustrates the device used to obtain the curves shown in Fig. 9.

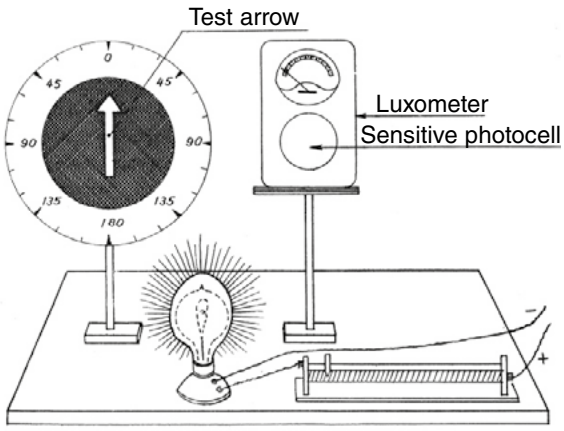

Figure 8. Upright white cardboard arrow on black background, graduated circle for each half, illuminated with dimmable light measured by the luxometer. When lighting is reduced, the arrow seems to be rotated in the frontal plane. The rotation is measured by rotating the arrow in the opposite direction until it is perceived vertical pointing upwards. Arrow 10 cm size placed 40 cm from the subject. This figure is the same as Fig. 13.2 in Vol. 1 of *Brain Dynamics*.

The perceived rotation by the patient of the upright test arrow depends on the illumination of the arrow at a given subject-arrow distance. The process of sensory *recruitment* of the orientation of the image is shown in Fig. 9 as a function of illumination intensity. It can be seen that from the upright correct perception, light must be reduced significantly to obtain a perceived tilt of 45° in the inactive state. At this point, however, the rotation increases quickly when the illumination is reduced very little more. In the state under facilitation by strong muscular effort, all occurs identically but with less light than in the inactive state. The sensory recruitment curves for orientation can also be obtained by varying the subtended visual angle, either by distancing the object or using different sized objects at the same distance (as in the previous test with letters or optotypes). When the curves are drawn taking the logarithm of light intensity (Fig. 10), they have a sigmoidal shape that approximate straight lines. This allows to say that *the sensory growth of the orientation of the visual image is proportional to the logarithm of the stimulus.* This conclusion falls within the Fechner law, which is therefore still valid for pathological stages. In the T case, a similar but much higher recruitment curve than in M under

[12] See details in Vol. 1 and Vol. 2 of *Brain Dynamics.*

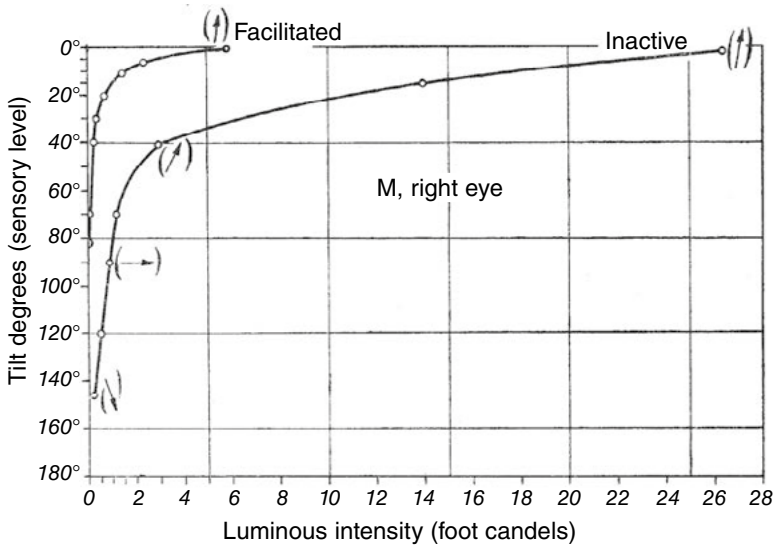

Figure 9. Sensory recruitment curves for the perceived orientation (tilt degrees) of the visual image as a function of the luminous intensity, for subject M in inactive state and under facilitation by strong muscular effort. (Experimental device of Fig. 8.) The same figure as Fig. 13.3 in Vol. 1 of *Brain Dynamics*.

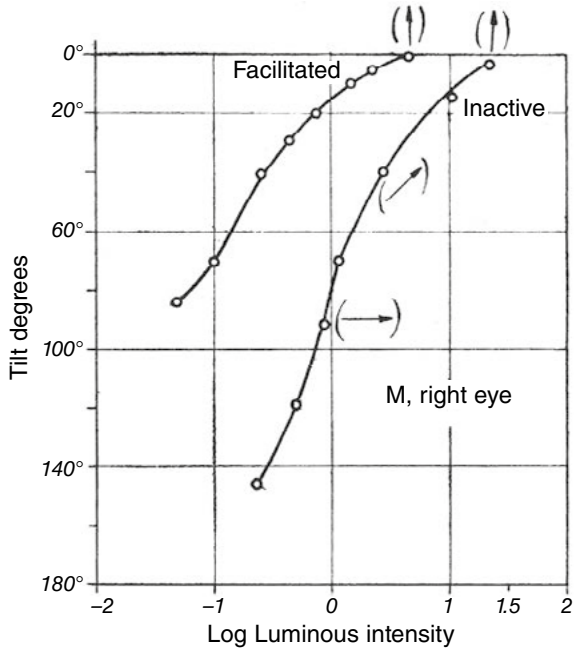

Figure 10. Relationship between angle of the perceived tilt and logarithm of the stimulus intensity (Fechner law). The same figure as Fig. 13.4 in Vol. 1 of *Brain Dynamics*.

facilitation is found. It means that in the three levels (M inactive, M under facilitation, T inactive) the law is the same but with a different parameter, whose value depends either on the magnitude of the lesion or on the state of the centers due to the summation effect.

3) The perceived tilt involves a complex process of sensory degradation. Thus, when the upright test arrow is perceived as rotated, it also appears narrowed and shortened among other changes. Several measurements, which are only indirect, seem to indicate that this reduction of the size of the arrow, i.e., of the visual field, is quite progressive (Fig. 11). Following the perceived trajectory of one extreme of the arrow during the rotation, we obtain a quite open *spiral* branch which expresses the actual spatial field disorder (combining rotation and constriction), constituting what we call *spiral development of the sensory field.*

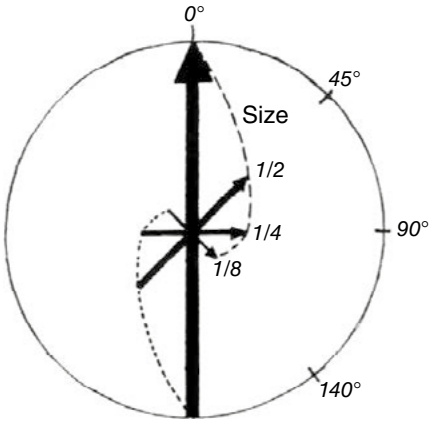

Figure 11. Reduction of arrow size (reduction of the s ensory field) when the image is tilted. The extreme of the arrow thus describes a spiral; there is a spiral field development.

4) Concerning the *mechanism* of the disorder, apart from the unspecific asynchrony, the problem lies in determining the cause of the image rotation. In Vol. 1 of *Brain Dynamics*,[13] after referring to the historical controversy over how upright (correct) vision is possible given optical inversion in the ocular globe, it is admitted that the retina factor is a spatial reality which can become independent in patients with brain injuries when that optical inversion is not corrected by some brain mechanism, the problem being thus restricted to a mere particular question of the vision system. However, on the discovery of tactile and auditory inverted perception in M in 1946, the process was extended to all the sensory systems with a spatial character (Vol. 2 of *Brain Dynamics*).

There had to be a structural factor conditioning the spatial inversion. This effect was soon attributed to the respective cortical projection areas with an inverted and contralateral disposition. The anatomical configuration thus acquired an unex-

[13] Secs. 12-14 in Vol. 1.

pected roll in this research on brain dynamics. Within this dynamic concept, the spiral development has the following brain basis: inversion and constriction extremes correspond to individual action of the projection (or "marginal") area; magnification and re-inversion are linked to the activity of more "central" areas (magnification due to an increase in recruited neural mass, re-inversion due to some effect of brain plasticity). There is then a sensory growth or progressive recruitment where the spiral trajectory represents a series of successive balances between "marginal" (projection area) and "central" action, which can also be expressed indicating that in *size* and *orientation* there is a sensory-brain correspondence, i.e., a psychophysical isomorphism. Due to the important consequences of this formulation in the sensory organization, it is the 'second principle' in this research on brain dynamics: *Spiral development of the sensory field due to a psychophysical isomorphism.* However, this left a gap with respect to the process of image re-inversion, a process that seems to be filled in this research by linking the recently discovered 'secondary areas' (of unknown significance) to this re-inversion process. Such areas represent the sensory field in the re-inverted position, and have a bilateral action.

Just before this publication, the spiral development has been studied specifically.[14] In relation to previous findings on spatial inversion in tactile system (see next section), the spiral development was specifically investigated for the case of the test object being situated in peripheral vision to one side of the visual field. When sensory degradation is in progress, the image of the test object undergoes rotation, centripetal deviation, reduction in size and intensity, and finally remains in a contralateral and inverted position, although very close to the center of the visual field.

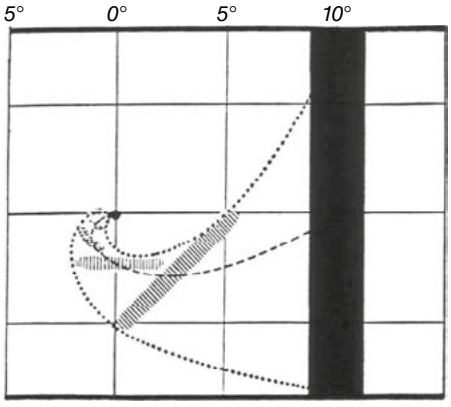

Figure 12. Experiments on visual spiral development in patient M. The test object is placed 10º from the central point of fixation. As visual excitation is suitably reduced, the image of the object decreases in size and intensity, rotates, and ends contralaterally close to the center. The drawn images represent phases obtained during the process. The trajectories correspond to logarithmic spirals.

[14] GONZALO, J. (1951). "La cerebración sensorial y el desarrollo espiral" [Sensory cerebration and spiral development], *Trabajos del Instituto Cajal de Investigaciones Biológicas,* XLIII: 209-260.

It is as if one half of the field, by rotating around the center of the visual field, has become progressively constricted and dulled in visual intensity (Fig. 12). The spiral trajectory obtained is like a field "force line" representing the *physiogenesis of the 'local sign.'* Geometrically, it has the aspect of a logarithmic spiral, and can be expressed dynamically according to the "forces" it is subjected to. It can be broken down into two vectors: one for size and the other for orientation (see Fig. 13).

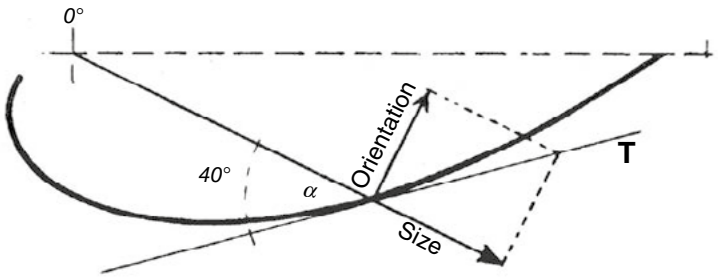

Figure 13. Dynamic characteristics of the spiral in the field development. The two vectors in which the tangent T to a point on the curve is decomposed signify the two "forces" of growth causing the spiral: *size* to enlarge the field, *orientation* to re-invert it.

5) Finally, the problem of inversion and re-inversion is taken to its anatomical roots, and related to Cajal's well-known theory of nerve crossings (chiasm, decussation of long pathways), in which the following three processes would occur:

i) The ocular inversion caused by the presence of the crystalline lens makes chiasmatic crossing necessary to achieve binocular congruence in the nervous centers (Cajal).[15]

ii) But the brain's visual projection remains inverted and crossed with respect to the exterior. According to Cajal, however, a crossing of the pathways of the other sensory and motor systems is functionally an uncrossing of vision; thus, a visual stimulus may elicit a motor reflex response on the same side, i.e., congruently from a spatial point of view.

iii) However, in the sensorium, the crossing and inversion of the projection (primary) areas still holds. In accordance with this order of ideas on crossings, I have proposed that re-inversion and bilateralization of the image occurs in the secondary areas. This fact eliminates any spatial incongruence in this latter process, since the brain centers could obtain a copy or image of the external world in the same order and orientation.

Thus, the pathologic process of image rotation in our patients would be produced by an asynchronism between primary and secondary areas (see details in *Brain Dynamics*).[16]

[15] See the reference RAMÓN Y CAJAL (1898) in References of Vol.1 and 2 of *Brain Dynamics*.
[16] See Sec. 26.2 in Vol. 2 of *Brain Dynamics*.

5. TACTILE FUNCTIONS. OTHER FUNCTIONS

Touch and vision have a remarkable structural analogy, but the former presents more difficulty in providing objective results, which makes it analysis more difficult. The analogy between touch and vision allows us now to omit many comments and the exposition here will be very brief. For more details, see Volume 2 of *Brain Dynamics*.

The following brief remarks refer to the five tactile functions studied:

General tactile excitability behaves in the same way as visual excitability with a similar quantitative deficit, due to the homogeneous repercussion of the central syndrome. Vibration sensitivity presents a similar loss of high frequencies analogously as in flicker fusion frequency in vision. The study is made on electrical, mechanical and vibration excitability, in inactive state, under facilitation and in iteration. Pathological bilateral tactile cases with unilateral lesions, unexplained by other authors, are interpreted in an appendix. (See Secs. 17, 18 and its appendix, in Vol. 2 of *Brain Dynamics*.)

Tactile sensations, much simpler than colors, have also a heterotactile interval due to asynchrony. Pressure or touch appears as the primary tactile activity (similar to luminosity in vision). Pain and temperature as differentiations. Clinical dissociations are relative and not absolute.

Tactile space shows many important phenomena. There is a large abnormal interval in cutaneous spatial localization of a stimulus between simple contact sensation and specific localization, with five phases distinguished according to the energy of the stimulus: I, primitive projection or contact sensation without localization; II, deviation towards the middle line of the body; III, inversion; IV, proximal homolateral deviation; V, specific or normal localization (see Fig. 14). These phases are a

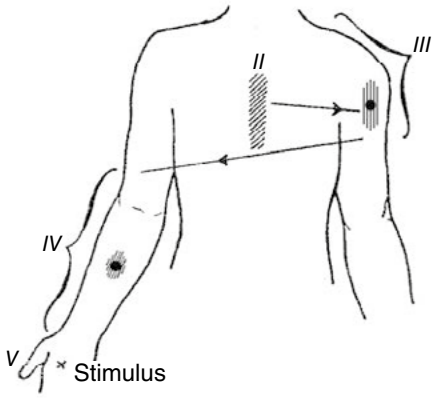

Figure 14. Phases of tactile localization in M in inactive state for a mechanical pressure stimulus on a hand. Depending on the intensity of the stimulus, the following is obtained: Sensory threshold intensity originates tactile sensation lacking localization (phase I, which is not in the figure); slight raise in intensity leads to phase II (medial deviation and quite irradiation), and to phase III (inversion phase). Phase IV is the homolateral phase with proximal deviation corresponding to moderate stimulus. Phase V is a normal sensation of localization, requiring intense stimulus or moderate stimulus and facilitation by strong muscular effort. Same figure as Fig. 21.4 in Vol. 2 of *Brain Dynamics*.

consequence of general factors such as *proximal deviation*, *spatial inversion* and *irradiation*, becoming more manifest as the intensity of the stimulus decreases since the delay in the localization phase increases. Tactile irradiation (spatial diffusion instead of a normal point sensation) is similar to color irradiation. The quantitative relationships are determined by the curves of logarithmic recruitment (proximal deviation as a function of stimulus intensity) and, more precisely, strength-duration curves for each phase of localization. The higher levels, closer to normal localization, are perturbed to a much greater extent than the lower levels. The lower phases (I, II and III) are very close, making them difficult to distinguish at the start of the study.

With respect to the local sign theory, the abnormal phenomena (deviation, irradiation, inversion) are an expression of a sensory field of residual function in correspondence with the central nervous substratum, and there is no local sign as genuine individuality, but there are multiple gradations according to the functional state of the organization of the nervous centers.

Acuity (Weber), movement, tactile figures, present similar defects to those of the corresponding visual functions.

Perceived *orientation in touch* is closely linked to the process of localization, and inversion was discovered when the third phase was identified. The study of all these phenomena is much less accessible than in vision and more difficult to objectify, leading potentially to many errors in superficial examination. The general laws are the same as in visual image orientation. The phenomena of *tactile inversion* are studied in cutaneous, articular (deep) stimulation, as well as in complex processes such as walking. Fig. 15 illustrates the inversion phenomenon of a stimulus moving on the skin. A moving stimulus descending on one side of the body is perceived contralaterally (inversion phase) and close to the midline of the body, with a very shortened trajectory and an opposite direction of motion. If such a stimulus is along an arm, reversed motion is perceived in the contralateral shoulder; if it is on the face, the third phase (or inversion) will correspond to the cranial calotte. In summary, a distal

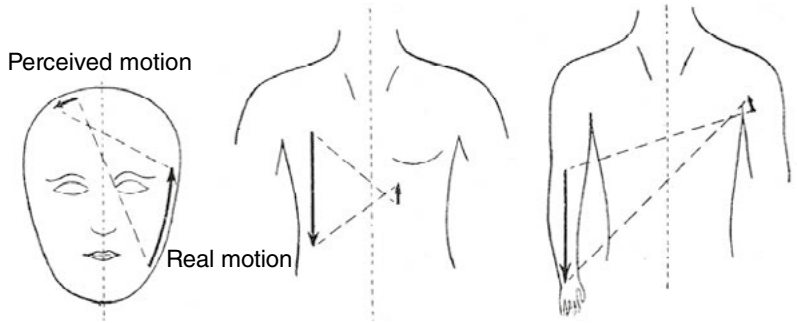

Figure 15. Inversion of motion over the skin in different parts of the body of subject M in the inactive state. The large arrow corresponds to the moving stimulus and the small arrow to the perceived motion. In all cases, the perceived inverted direction of movement is localized contralaterally, approaching to the middle line (proximal deviation) and with much smaller trajectory than the real movement (reduced to 1/10 approximately). Same figure as Fig. 24.8 in Vol. 2 of *Brain Dynamics*.

sector that undergoes inversion is located contralaterally, in the opposite direction, highly constricted and thus close to the middle line of the body, verifying the conditions of a sensory field of residual function. The inversion is not for the whole field as in vision but is more regional according to *three autonomous zones* (head, trunk-upper limbs and pelvis-lower limbs). The quantitative results are shown in the curves of orientation recruitment (rotation and deviation perceived of a rectilinear stimulus as a function of the pressure applied on the skin) although the same precision as in vision cannot be obtained.

In walking, the process shows unique characteristics: in moderate walking, the first step is ignored, the second feels inverted, the third transversal, the fourth oblique, etc. This is a progressive recruitment of direction of perceived steps by iterative action of steps. Instead, in slow walking there is no summation effect, and the direction of the steps remains inverted for each step. The inverted steps feel very short and fast in conformity with the residual field. The energy of the step modifies the result according to the rules of excitation.

The mechanism and theory of spatial orientation has been extensively addressed in vision, and has been generalized to all spatial sensory systems. Tactile research has promoted the union between the constriction of the field (proximal deviation) and inversion, leading to the spiral development described above.

Tactile schema comprises many functions which present continuity with the previous ones. There are two fundamental aspects: the corporal model or body schema and tactile recognition. The former can be considered gradually according to the *somatic, postural* and *praxis* models, which show notable anomalies when studied in detail. Tactile recognition shows defects such as stereognosis reduction and stereognosis transformation, and tactile asymbolia in a higher phase. Recognition is indirect, and the typical fragmentation or discontinuity of agnosia appears, which is only an effect of the reduction of schema dimensions (small schema that does not encompass the whole but juxtaposed parts).

With respect to *auditory functions*, their dynamic phenomena and other manifestations occur in patients M and T in the same way as in vision and touch, except for the peculiarities of each system. Auditory excitability presents a deficit and is permeable to summations (facilitation by muscular effort, bi effect, iteration). There is a certain hypoacusia, perhaps stronger in high tones. Acoustic quality (musical tone) has an abnormal interval, as in vision and touch. It is a singular interval between simple sonority and real tone. Contralateral localization or inversion of a sound stimulus only occurs in M when the intensity of the stimulus is weak and the subject is in an inactive state. The inverted perception always lacks tonal quality; it is a residual function.

In *language*, different aphasic aspects occur depending on stimulation, this being in fact a semantic defect.

6. PARAMETERS OF THE SENSORY FIELD

At this point, several further observations will be made on the residual field and sensory dimensions.

Firstly, the clinical aspect of the two types of cortical syndromes will be indicated. In a "marginal" (or projection area) syndrome, patients have a quite ostensible defect because the lesion of a determined sensory system is massive. There is an anatomic suppression of the system which prevents any type of study of the functional structure, and we are reduced to outlining a "gap" in the sensory organization. It is over these gaps that specific localizations in the brain are established. On the contrary, in the more central syndromes (central and paracentral) even in extreme magnitude, the individuals show few symptoms at first sight and sometimes are unaware of them themselves. However, examination reveals certain gradual alterations in a series of functions (physiological "depression"). Under minimum stimulus or under a situation of sensory threshold, anomalies of utmost importance then appear. Only in these cases can we penetrate the sensory structures. The anodize appearance of these cases is derived from the fact that the essential disorder consists in a *scale shift of sensory magnitudes* and thus, if the stimuli are significant there are no functional deficits or failures.

Such a systematic alteration of multiple functions in "central" affection, even in the case of being limited to only one sensory system (paracentral syndrome), immediately runs up against several nosological entities admitted as independent and specifically localized, which in classical brain pathology are categorized into *sensory*, *perceptual* and *intellectual* disorders (e.g., anesthesia or blindness, astereognosis and agnosia), classification system more or less derived from the doctrine of mental faculties. Table 1 on visual functions shows the great regularity in the alteration of the whole system, a regularity that makes it possible, on the basis of a single pathological datum, to presume the alteration of the other functions. Contrary to traditional ideas, there is no isolated effect on any of these functions, nor do they recognize genuine nature (see asynchrony). This issue must be addressed taking into account our patients, in whom there is simply a sensory field governed by other *sensory dimensions*. These dimensions can be reduced to intensity, space and time, simple physical concepts common to natural sciences and with a quantitative character. *Intensity* refers both to the simplest sensory threshold (rheobase) and to intensity changes in the differential threshold. *Space* or spatial dimension covers place (local sign), spatial acuity, extent and size, shape or figure, etc. Sensory *time* includes reaction speed, chronaxie, rhythm frequency (fluttering, vibration etc.) and movement processes. The values of these parameters tend to zero in a very residual field, whereas they increase as the sensory field develops until the normal field value is reached. They are thus field parameters, and depend on the active neural mass; i.e., they are dynamic parameters. All of this responds to pathophysiological needs, and considerably simplifies the sensory problem, avoiding bothersome classifications.

These parameters permit a natural description of the state of the sensory field. It could be thought that they only apply to the lower sensory functions, whereas the higher or intellectualized functions (figures, gnosis, etc.) would be left out. However, experience shows that the system is disturbed as a whole (see Table 1 on visual functions), and the schema function is reduced according to the other activities of the field. It is thus possible to postulate that *between mere sensory function and gnosis activity there is a continuity through a same functional pattern*, even when there are successive

stages of increasing complexity. The traditional separation and even opposition between higher and lower functions, as well as the notable preponderance of the former in classical brain pathology, are questionable criteria after this study. As mentioned, both types of functions have the same basis, and only the lower functions allow us to elucidate this basis.

As for agnosia, it should be noted that whereas traditional theory emphasizes the amnesic defect, i.e., a "re-production" (re-cognition) disorder, here agnosia is approached as a "production" defect (as clearly shown in orthogonal disorder, in allocentric disorder and, very simply, in the overlapping figures test) which greatly facilitates its linkage with simpler functions. In sum, gnosis is thus basically a mere capability of the field to gather information. It would be very useful to analyze the agnosic process according to the concept of *quantity of information* belonging to the theory of communication.

7. BRAIN GRADIENTS

According to the effect of the position of the lesion we have accepted the functional topographic heterogeneity of the cortex, and also accepting the classic anatomoclinical method (skull-clinical in the injured patients), three syndromes have been distinguished: marginal (projection area), paracentral and central. In Fig. 1, these syndromes refer to the visual system, taking the visual field as an index. This schematic illustration is the starting point for the concept of brain gradients which are based on a continuous variation.

When the visual field is used as a general pattern for all syndromes, the problem of brain localization is simplified substantially, offers new perspectives, and the issue seems to flow along its natural course. In traditional theory of localization, the three above mentioned syndromes, marginal, paracentral and central (Fig. 1) would be referred respectively to, the field, perceptive defects and agnosia; completely heterogeneous terms which are useless for mutual comparison. Nevertheless, the syndromes are expressed by the same factor in the brain dynamics here developed: *the field*, whose shape and size vary from one syndrome to another according to defined rules. The common denominator (the field) provides homogeneity and transition from one to another, i.e., it makes possible a continuity in their assessment. It would seem difficult to find anything better than the *field* to comply with the strictest requisites of functional localization since this is a spatial effect, and it is better covered when an entity of an essentially spatial nature such as the visual field is used. Thus, the problem of functional localization in the brain is addressed here according to cortical gradients thanks to the previous work of determining a) what is *localizable*, and b) the *transition* between the syndromes.

We shall first present several cases to adequately demonstrate the three visual syndromes illustrated in Fig. 1. From a great number of first hand observations of war injuries, I have selected the following three series of visual cases. Fig. 16 shows cases of *central scotoma* and notches due to occipital pole lesion (macular projection in striate area); they are examples of a marginal syndrome which is now partial. Fig. 17

shows cases of *hemianopsia with homolateral constriction*, i.e., of the paracentral visual syndrome due to unilateral lesion in the occipital convexity at a certain distance from the posterior midline. Fig. 18 refers to cases of central syndrome, with *concentric reduction* from unilateral lesion in a more "central" position than the previous type.

These are not equally common types. Cases of central scotoma require a very circumscribed lesion at both occipital poles, and although numerous civil and war cases are known, they are rather rare. Cases of hemianopsia with some homolateral constriction and quite moderate concentric reductions are quite common in war injuries. On the contrary, significant constrictions are rare, both in paracentral and central syndromes. In the figures we have designated our most affected cases but none reach the intensity of the M case although case 1 in Fig. 18 is close. Each case is a permanent disorder continuing many years after the time of injury. It should be pointed out that the visual defect is purely cortical, in the scotomas and in cases of constriction; thus, any peripheral cause such as papillary stasis is completely discarded.

Cases of *scotoma* (Fig. 16) have an anatomic explanation (macular projection), although in view of the different visual acuity and the density of the scotoma, the defect should be understood somewhat functionally, and rather referred to the number of destroyed neurons in the macular area. These purely marginal cases are closest to ocular defects. There is no reduction in visual organization and less in other sensory systems.

Contralateral *hemianopsia* of the paracentral syndrome (Fig. 17) would seem to be explained by purely anatomic causes, in accordance with the traditional homonymous cortical lateral hemianopsia. However, this could be excessively inexact since the lesion may lie *outside the calcarine* in many cases, and the contralateral hemianopsia is only a hemiambliopia with the defect disappearing under intense stimulus, thus being only a large lateral constriction of the same nature as the small homolateral constriction. It would then be an asymmetric concentric reduction which is a transition towards the symmetric concentric reduction due to more central lesion, or even further from the calcarine, and completely unadaptable to a traditional anatomic explanation.

Concerning the transitions, the field in paracentral cases winds up taking a more rounded or symmetric form when using a highly intense visual stimulus or test, whereas one finds a trend towards hemianopsia in some symmetric concentric reductions when the central isopters are determined. Case 1 in Fig. 18 shows an intense asymmetric constriction in the 3/300 ordinary isopter, and a notch in the upper quadrant, corresponding to the type of cortical lesion. But, under a very bright stimulus, the field widens and tends to be more regular although with a certain lateral asymmetry. This is a typical intermediate case between the paracentral and central syndromes.

The paracentral and central syndromes are satisfactorily explained by the physiological functional depression we have seen, and being dynamic syndromes, are fully ascribed to the system of gradients. Such depression is accompanied by phenomena of dynamic action, reduction of functions etc., as set out for the M and T cases. In case 1 of Fig. 17, the constriction is significant and the acuity descends

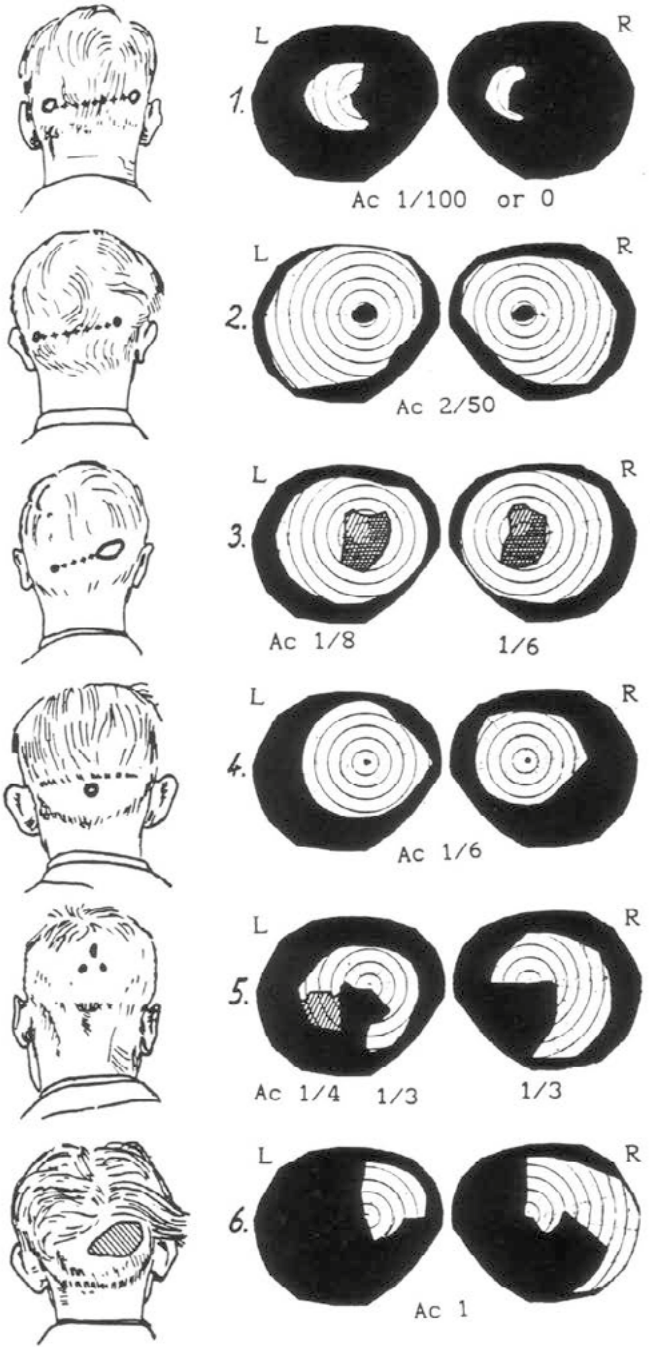

Figure 16. Series of *central scotoma* cases (first four) and central notches (final two), due to war lesion in both occipital poles. Note the considerable acuity (Ac) deficit due to cortical involvement of the macula. These cases present an incomplete *marginal* syndrome.

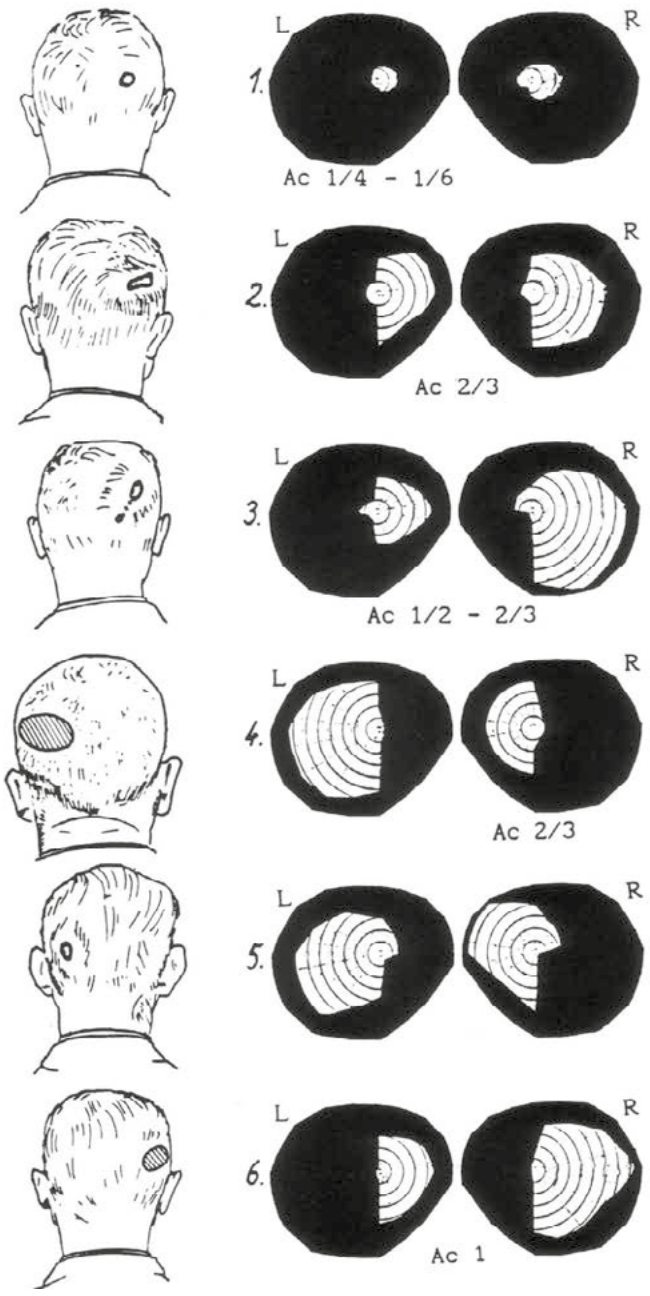

Figure 17. Various cases of lateral cortical *hemianopsia* due to occipital war lesion on one side of the midline. In all cases there is a reduction of the conserved field. The more the reduction the greater the deficit of visual acuity and the more accused asynchrony (case 1 presents a rotation of the visual image of 40°; case 2 only 6°). In this research, all these cases correspond to the *paracentral* visual syndrome with intensity varying from one to another.

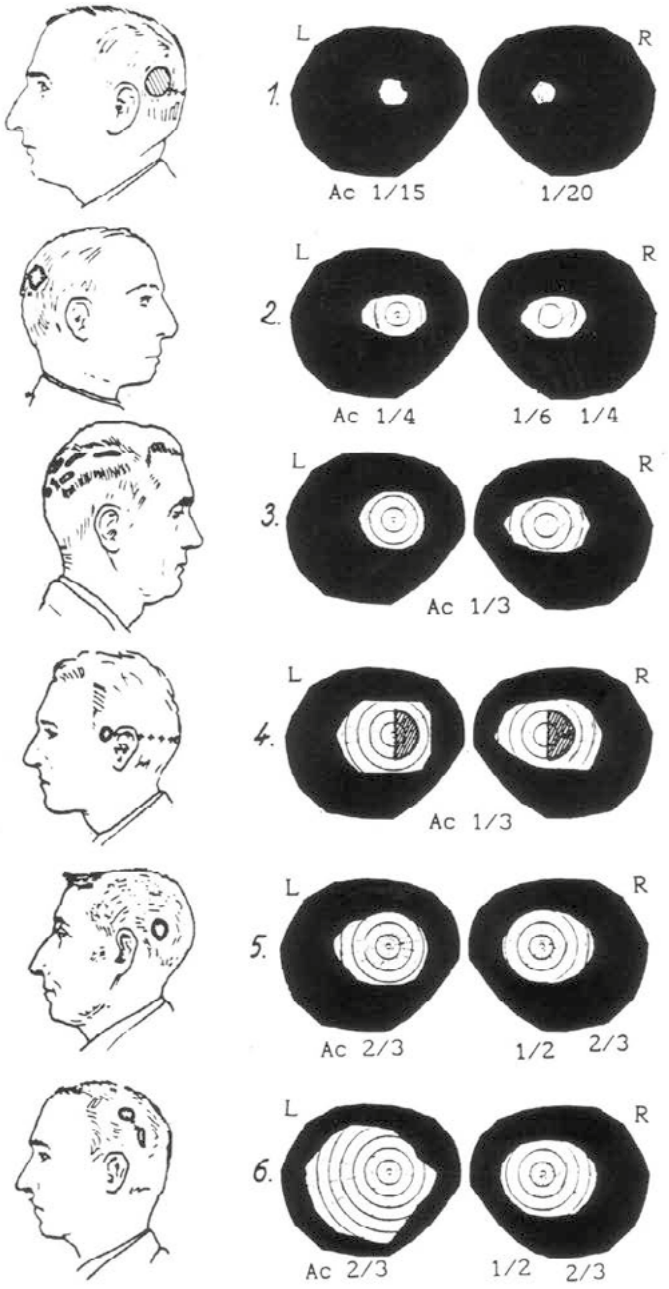

Figure 18. Cases of *concentric reduction* of the visual field due to parieto-occipital lesions which may be very distant from the striate area. A large lesion which is close to the visual area produces much greater reduction (case 1) than when further away (case 6). All are cases of *central* syndrome, with the corresponding dynamic phenomena (case 1 presents a rotation of the visual image of 90°; case 6 of 12°).

proportionally, the dynamism being evident since this case presents a pathological visual image rotation of 40°. Furthermore, the patient shows a certain tactile and auditory repercussion in the contralateral side. In contrast, case 2 of Fig. 17 only reaches a visual image tilt of some 6°. In Fig. 18, all cases (central syndromes) present a significant dynamic defect. The first one reaches a rotation of the visual image of 90-100°, and the sixth reaches 12° with the worse eye, i.e., that contralateral to the lesion.

Symmetrical and asymmetrical visual field reductions depending on the position of the lesions are found in both war injury cases and civilian cases by other authors, and although some authors have pointed out it, this issue is still generally ignored. In the literature we have found numerous cases, both war injuries and vascular lesions as well as surgical cases, which fit our concept.

A schematic illustration of the syndromes according to the double cranium-clinical aspect is shown in Fig. 19, which has a meaning similar to that of Fig. 1. One of the most notable results is the important participation of the extravisual cortex (occipitoparietal, parietal, temporal, etc.) in the maintenance of the visual field. Such a result is also valid for touch and the rest of the senses. Thus, the traditional separation between projection and association areas is in serious crisis, as here it is demonstrated that the "central" zone participates to some extent in the formation of the field. This situation should also lead to refute the distinction between higher and lower functions as set out in Sec. 4. The idea of functional *continuity* through the cortical areas is thus imposed, albeit with a certain *variation*, leading to the system of gradients.

In the sense used here, a functional gradient is defined as a function that varies with distance. Since 1951 I have used in the doctoral courses the schematic representation shown in Fig. 20, where the different syndromes can be interpreted according to two types of gradients: specific gradient and integration gradient.

The *specific* gradient referred to vision means that close to area 17 (visual projection area), the density of the visual function is maximum (there is a critical zone at this point) and decreases progressively towards more central areas and beyond. It corresponds in Fig. 20 to the curve descending from the visual extreme towards the central zone (in the simplest form, the function would decrease with the square of distance). This specific gradient only has *contralateral* action. The same may be said for the specific tactile gradient, and we could also add an auditory gradient, omitted to simplify the diagram. This type of gradient involves all sensory activity in such a way that for the visual field to be normal, acuity with a value of 1, etc., the action of the critical or higher density zone is not enough, but all the visual gradient through the cortex must be involved. This gradient is essential, and is understood to involve and combine the factors of magnitude and position referred to above.

The *integration or unspecific* gradient is more complex. It is a consequence of the overlapping of the specific gradients, given the extent they occupy. It is therefore reasonable that in the central zone where they overlap, there must be an action of mutual integration which is nonspecific, let us say, equipotential. This action is maximum in the central zone and minimal towards the projection areas (bell curve). In addition, this gradient contains bilaterality or interhemispheric effect due to the ac-

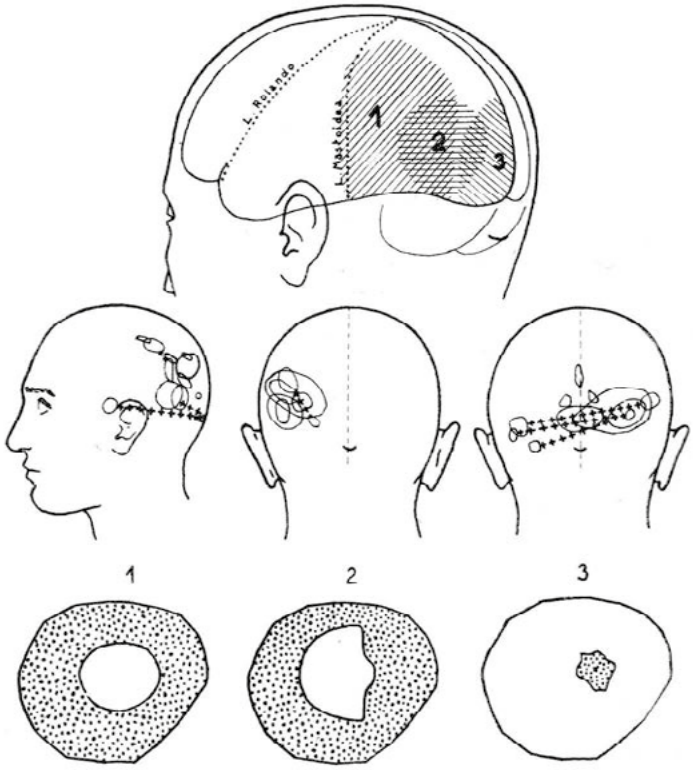

Figure 19. Cranium-clinical correlation showing a synthesis of the three series of cases in Figs. 16-18. 1: concentric reduction; 2: asymmetric reduction; 3: central scotoma. Note the ample participation of the parietal zone in the visual field.

tion of the callous, from which the projection areas are excluded, as is known from anatomical and neurographic studies.

In general terms, in lesions towards the maximum of the specific gradient functions, the defect is predominantly contralateral and unisensory. In "central" lesions, the defect is bilateral and multisensory, i.e., general. With respect to the intensity of the defect, what is achieved in the specific maximum with a small lesion requires extensive lesions in the central area, as indicated in Figs. 20 and 20'.

The gradients system is a system of *quantitative localizations according to fields of action*, offering an eminently dynamic conception. However, this representation of gradients is no special hypothesis but an abbreviated formulation of the syndromes described in this research of brain dynamics. It is a mere abstraction of the observed facts, and is an attempt to place them in a certain order.

As opposed to the rigid separation into areas or centers according to the traditional theory, here a functional continuity with regional variation is offered. For each point of the cortex, the combination of a *specific* action with a *central* action leads to characterize that point by a determinate value. Each point acquires then different

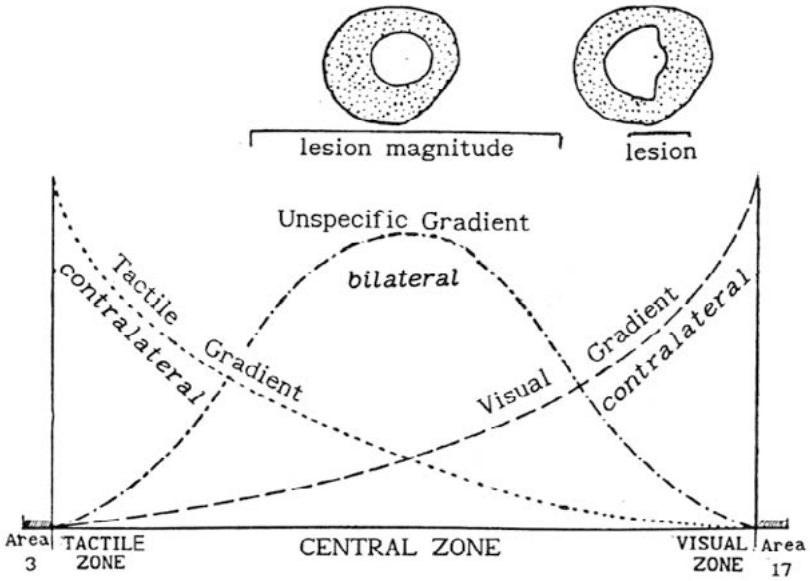

Figure 20. Scheme of cortical gradients. Maximum visual "density" is in area 17, decreasing towards the central zone and beyond. It determines the *specific visual gradient*, of contralateral action. Analogous for the tactile gradient. The *integration or unspecific gradient* (equipotential) arises from the overlapping of the tactile and visual gradients, with maximum action in the central zone and minimal in the extremes. It represents also the bilaterality. The upper visual fields refer to the position and magnitude of the respective lesion.

properties from the neighboring points. In spite of this diversity, even in the most specific areas, there seem to be a certain unity with the rest of the cortex. For example, in a shrapnel injury in the tactile area which produced a complete permanent hemianaesthesia (a rare occurrence), in addition to anartria, apraxia of the mouth, etc., there was a clear constriction of the more internal isopters in the visual fields, to a greater degree in the contralateral eye. On the other hand, in a similar case but with less acute tactile symptoms, the visual fields were completely normal. This leads one to think that even the tactile projection area affects vision to some extent. This influence is quite small, and the tactile loss must thus be very great for the influence to become manifest. Similarly, there is a reciprocal action of the visual area on touch, hence the final decline of the specific gradient must reach the opposite extreme of the horizontal axis in Fig. 20.

These gradients come into play in the topographical disposition of a particular sensory system. The corresponding specific gradient evidently plays a role, but also counting on the bilaterality provided by the other gradient; thus, the system tends to be elaborated and integrated towards the central zone. In some cases, a gradient with a hemispherical dominance also has to be added. A further aspect is that of certain complex functions which could arise from the fusion of gradients of different systems. Thus, the genuine or primary alexia would have its own characteristic field

obtained from the fusion of visual and auditory gradients, giving rise to a bell-shaped lexical gradient between the two systems. It is clear that local fields and gradients with more restricted effects must arise along with the general types.

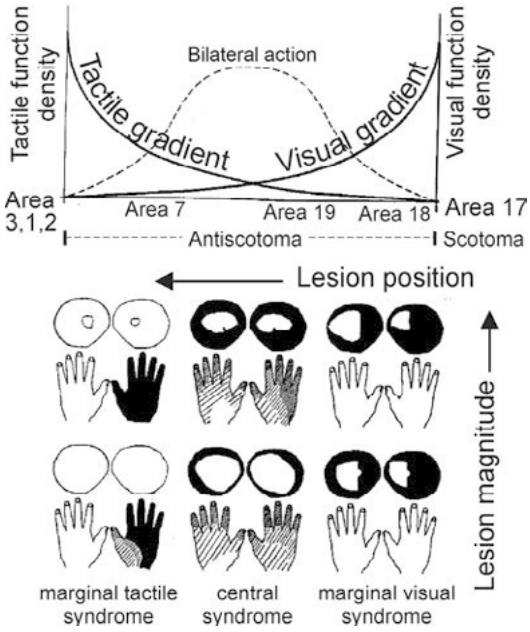

Figure 20'.[17] Visual fields and tactile sensitivity of cases ordered according to the position and magnitude of the lesion. The degree of the defect is greater in darker regions.

With respect to the effect of lesions on the gradients, it should be admitted that in the central syndrome at least, the pattern is maintained even though the values are reduced. It is questionable whether the same occurs or the distribution varies in paracentral syndromes, but in any case it is essential that the change affects the whole system whose potential is reduced.

Finally, it must be noted that the gradients have an anatomic base (terminal paths, contralaterality, corpus callosum) and represent its dynamic aspect. The whole cortex is subjected to a common principle of organization (action field and its gradient), signifying a "deintellectualization" of the cortex. The sensory field projected on the marginal area (projection area) is only an outline which must be magnified and elaborated (*integrated*) towards the central zone. The ample activation of territory

[17] This figure is made by the author after the publication of the article of 1952. It has been included here to better illustrate de concept of cortical gradients. It was published in the article:

GONZALO, I. and GONZALO, A. (1996). In *Brain Processes, Theories and Models,* R. Moreno-Díaz and J. Mira-Mira (eds.), The MIT Press, pp. 78-87. The figure is included here with permission of The MIT Press.

with strychnine, the large extension of the EEG response without anesthesia, the series of accessory projection areas (supplementary, secondary, mixed) with characteristic physiological properties (greater threshold and latency, easy exclusion with anesthesia, etc.) would lead, through statistical results, to the gradients.

8. BRAIN LESIONS

Brain lesions deserve several comments, particularly in the basic aspect of the so-called *lesion magnitude*. In this research of brain dynamics, the exclusively quantitative differences between M and T patients are perfectly explained by the different quantity of cortical destruction on the same site, both patients showing the same syndrome in different intensity.

The lesion in M is a grazing cortical destruction, with entry and exit orifices of the projectile in the parieto-occipital convexity. In T there is a notable cranial hollow or gap resulting from fracture and sinking in the upper zone of the same convexity. However, the surgical operations revealed the dura mater intact and pulsating. A cortical contusion of considerable extension but without profound functional abolition must therefore be admitted. Recovery from the disorders was marked in T and quite small in M (see details in Sec. 2.1 in Vol. 1 of *Brain Dynamics*.

At this point it is worth providing some indication of the types of cranial injuries and the corresponding brain disorder, thus we will have an adequate criterion to judge the magnitude of the lesion. Within our aims, and as a mere empirical orientation, gaps should be distinguished from injuries with an *entry* and *exit* orifice. Contrary to what could be expected, large gaps only cause weak, but possibly numerous or varied symptoms. These gaps are bone losses due to limited fractures and sinking which only cause contusions in the brain, and the functions generally recover well. The T case may belong to this group although his gap has a medium extension. There are also small gaps resulting from large fragments of shrapnel located at varying depths. In this case the subjects at times show symptoms of great intensity.

Cases of projectile entry and exit are more appropriate for comparative study. In these cases, injuries with two biparietal or bilateral orifices must be discarded as they do not result in lasting disorders, as is the case of injuries in one hemisphere with two very distant orifices. In contrast, in those with a short trajectory and a grazing tendency, the destruction of the cortex tends to be quite large with intense and permanent symptoms such as in patient M. Several of the cases in Fig. 18 fall in this category. Apart from these ordinary circumstances, complicated cases may occur due to scar sclerosis, secondary infections etc., in which it is impossible to foresee the lesion magnitude. There are also cases of small but deep injuries, such as the first case in Fig. 17, which initially showed a disorder of low intensity but with time worsened without any apparent cause.

Brain war injuries constitute the most favorable material for brain research, given the immense variety of injuries, the youth of the subjects, the well delimited lesions, etc., complementing, and at times overrunning, civilian material. Brain pathology caused by war is more a "polio-pathology" or grey matter pathology, where-

as brain pathology in peacetime is a "leuko-pathology" or white matter pathology (Pierre Marie). The first one thus has a particular character and is quite important with respect to the cerebral cortex.

The sets of cases in Figs. 16, 17 and 18, grouped according to similarity in lesion position and type of corresponding sensory defect (Fig. 19), are what we may call *lesion families*, the differences within the same group or family being the degree of involvement.

Results may be obtained from the more homogeneous and easily compared cases of concentric reduction of the visual field to confirm the thesis that the degree of reduction of the field parameters is more remarkable as more extensive is the lesion determined by the trajectory of the projectile.

If the question of the lesion magnitude is still considered insufficiently proven in brain injuries, more direct and precise data may be provided by neurosurgical cases which fit our thesis perfectly. Results from abundant experimentation on animals also favors this.

With respect to surgical excisions, it is worth mentioning some aspects that may have a great theoretical scope, and for the time being provide new information on the functional coupling and dynamism of the brain hemispheres. Thus, a small parastriate lesion may be more perturbing functionally than an occipital lobectomy which suppresses a larger quantity of brain mass than in the first case. In the first case, both occipital lobes can be admitted to be perturbed, one significantly reduced in function (hemianopsia) and the other somewhat depressed (homolateral constriction) due to the interoccipital correlation. In the second case, however, complete excision of one lobe (even striate area) suppresses the function on one side whereas the function of the other side remains intact since the corresponding lobe is untouched and isolated from obstacles. This result leads to the interesting suggestion that a paracentral visual syndrome may benefit functionally from an occipital lobectomy despite the increased loss of brain mass, as a new state of dynamic equilibrium is promoted in the interhemispheric correlation.

Many occipital lobectomies do not cause the repercussion in the other hemisphere we are used to observe in paracentral syndrome due to parastriate lesion. However, when dealing with broad lobectomies, this effect is presented anew, probably due to a more central action. The question may pass on to a broader problem, and thus we may think that a central syndrome (bilateral disturbance due to unilateral central lesion) could be "remedied" to some extent by hemispherectomy of the side holding the organic lesion. Absolute defects would remain (in vision at least), but intact functions without the least desynchronization or alteration would result, as shown in the cases of hemispherectomy I have observed. Thus, it seems to be a question of the amount of energy to be shared by the two hemispheres according to the functional unity. It is noteworthy that the suppression of repercussion could perhaps be achieved more simply by sectioning the corpus callosum.

Finally, we should note that the correlation between the magnitude of the lesion and the magnitude of the disorder has long been expressed by outstanding authors in both human pathology and animal experimentation. It contributes to the criticism of nosological units, to the problem of functional restitution, to neurophysiological

matters, etc. However, such a correlation has not been highlighted enough due to the restriction imposed by the classical theory of specific centers. The classical theory of brain localization still could be used in small lesions as an approximation, but in large lesions, as is the case of patient M and others, the viewpoint of this research of brain dynamics is imposed in a natural way. Perhaps it could be said that the transition from de the classical to the dynamic conception derives from the magnitude of the lesion.

9. DYNAMIC REDUCTION AND NEW CASES OF THE VISUAL INVERSION PHENOMENON

Although cases of intense disorders, where the dynamic phenomena can be examined conveniently, are not common because large lesions are rare, we shall briefly refer to a more extensive casuistry in order to consolidate the concepts of this research of brain dynamics. We focus on the visual inversion process.

Fig. 21 covers more than twenty cases with visual disorders presenting a *chronic* manifestation of the pathological visual inversion phenomenon in varying degrees.

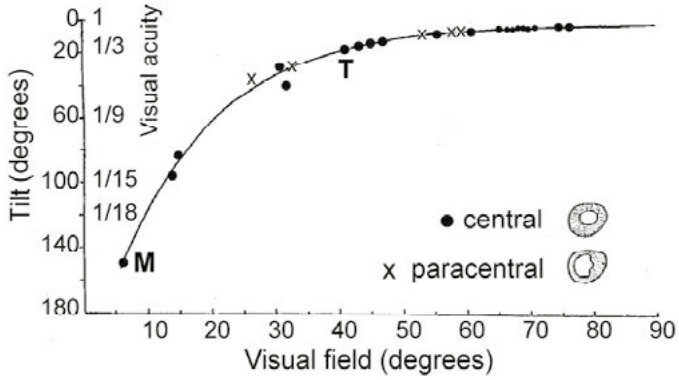

Figure 21. The curve illustrates the ordering of a series of new cases in relation to the perceived tilt (rotation) of the visual image (vertical axis) and to the width of the visual field (horizontal axis). The Mand T cases are included for comparison. The corresponding visual acuity values are displayed vertically.

The cases are ordered along the curve according to the degree of the perceived rotation of the image of a vertical text arrow and to the constriction of the visual field. The majority were injuries with more or less intense concentric reduction (central syndrome), a few others with hemianopsia and homolateral constriction (paracentral visual syndrome). In comparison with the Mand T cases, we see that there are few cases with considerable image rotation, and none reaches the intensity of the M case. Only twelve cases present a clear rotation of more than 10°, half of which are moderate, and even less have a sufficiently marked tilt to allow the study of the spiral devel-

opment in them. In contrast, many cases are found with little tilt; some of which are easy to identify whereas others are less accessible, almost within the observation error.

Several injuries with a large tilt (rotation) are included in Fig. 18 (the first case presents 90° of rotation, the sixth almost 15°), others are in Fig. 17 (the first case reaching 40°, the second only 6°). From these figures and the curve in Fig. 21 we see that the greater the constriction of the visual field, i.e., the more reduced the sensory dimensions, the greater the image rotation under minimal stimulus and the greater the dynamic reduction, i.e., the greater the trend towards a residual function field (as seen in the corresponding visual acuity). The curve in Fig. 21, which is only an approximation, shows a functional continuity between the reduction of the visual field and the degree of image tilt (rotation), highlighting the nonlinear quantitative relationship between them. If the field is significantly reduced up to 45º, the tilt is only 10º. But for greater constrictions of the field, the tilt (i.e., asynchrony) becomes highly significant. As a practical rule, one cannot expect to find cases with very marked rotations of up to 100°.

In the most acute cases, orientation recruitment curves were determined as a function of the visual angle subtended by a vertical test arrow, leading to results similar to those found in patient M (Fig. 9), and fitting the respective curve parameters quite well. The most acute cases also showed considerable agnosic defects and accentuated color weakness, visual fatigability, phenomena of summation and other disorders. The characteristics of the inversion process comply with that established in this research of brain dynamics: the greater the blurredness, the greater the rotation, accompanied by micropsia. It is noticeable that sometimes subjects with high image rotation were unaware of their disorder. In some cases, as well as the rotation in the frontal plane, there was a combination with rotation on the sagittal plane, which has already been observed partially in patient T. The majority of the important cases showed several peculiarities besides the rotation of the visual image, especially the most extreme case (case 1 in Fig. 18). In this case, in addition to suffered from a significant constriction of the visual field and acuity deficit, he showed a complex and quite accentuated syndrome of visual agnosia, color agnosia, amnesic aphasia, and particularly primary alexia of the verbal-literal type. Furthermore, he was close to the orthogonal disorder since with numbers normally oriented and rotated 180º the subject noticed something strange but was unable to specify the change in orientation.

As already said, all cases in Fig. 21 belong to the central or paracentral syndrome, whose localization in the parieto-occipital convexity is indicated in Fig. 19. It should be pointed out that the image rotation is obtained in both left and right lesions.

Besides these cases of *permanent* disorder in the visual image orientation, we have observed transitory cases, mainly during epileptic auras. Naturally these cases are not as important as the permanent ones, although they do contribute to some extent to our knowledge of the inversion process. Apart from the disorder during the seizure, the subjects (former brain injuries) show no visual disturbance worth noting. There are permanent cases with small image tilts which intensify during the auras, but other subjects only perceive tilt of the image during the seizure, either in

the form of grand mal or simple absences, during which the actual visual scene appears suddenly tilted, and at other times almost inverted. In this type of sudden tilt, visual forms undergo less deterioration than in the tilt due to permanent asynchrony, although they present always some blurredness. The subjects with this type of attack are aware of their disorder in visual orientation and tend to refer to it spontaneously. A quite curious case is that of a subject with an old injury with shrapnel lodged in parieto-occipital subcortical zone, entering through the frontal region. He suffered for a long time, and always in the same way, from absences during which he had hallucinations oriented in an inverted manner. Shortly after entering the trance, he visualized that a door appeared near the ceiling, the door opened and soldiers passed through it parading upside down for a certain period of time.

We have collected more than 100 cases of visual image tilt in the bibliographic research we have conducted which covers a period of about 50 years. However, this phenomenon is practically unknown and goes unnoticed even in the specialized works[18]. The vast majority of the cases collected present sporadic manifestations of tilted or even inverted vision in several attacks. Permanent cases are less abundant, and in them a moderate tilt of the visual image is more frequent. In general, all these cases are referred to in publications as simple isolated notes or common clinical data. It is interesting to note that the cases where it was possible to determine a lesion, showed a clear predominance of the parieto-occipital-temporal zone.

Finally, we make simple reference to the auditory system concerning the *dynamic reduction*, i.e., the involvement of the whole sensory system, and according to a physiological order. Indeed, in many cases of aphasia, particularly sensory aphasia, it is shown that the aphasic defect is not an isolated disorder; thus, the greater the language disturbance, the greater the deficit shown by the audiogram curve expressing the simple excitability to the scale of sounds. The semantic defect thus shows much less simple hearing deficit than the amnesic defect, residual to a sensory aphasia.

10. BILATERAL TACTILE INVOLVEMENT IN UNILATERAL LESION

Some comments on the *paracentral tactile syndrome* will be made here. Fig. 22 illustrates a case with an injury in the right cortical tactile area in its upper part, i.e., towards the region of the lower extremity, presenting a residual disorder consisting in a slight contralateral hemiparesis predominating in the lower extremity, and also in a hypoesthesia not only on such contralateral side but partially on the homolateral side, as shown in the figure.

In accordance with the localization of the cortical lesion, the greatest tactile defect corresponds to the lower contralateral extremity, a defect of distal predominance which reaches its maximum intensity in the toes, and extends more weakly up to the middle of the thigh and even higher. In contrast, it is much less intense and

[18] The following article provides a review of inverted perception based on this research: Gonzalo-Fonrodona, I. (2007). Inverted or tilted perception disorder. *Rev. Neurol.,* **44** (3): 157.

extensive in the contralateral upper extremity, where the degree of impairment is close to or somewhat greater than that of the lower extremity homolateral to the lesion. The contralateral face showed a slight tactile deficiency, as does the homolateral hand. In accordance with the dynamic reduction (physiological "depression"), the defect involved the various tactile sensations in a well-defined order (most important defect in temperature, then pain, then pressure), vibratory sensitivity, sensation of passive articular movement, etc., and with an intensity of the involvement in relation to the topography described above.

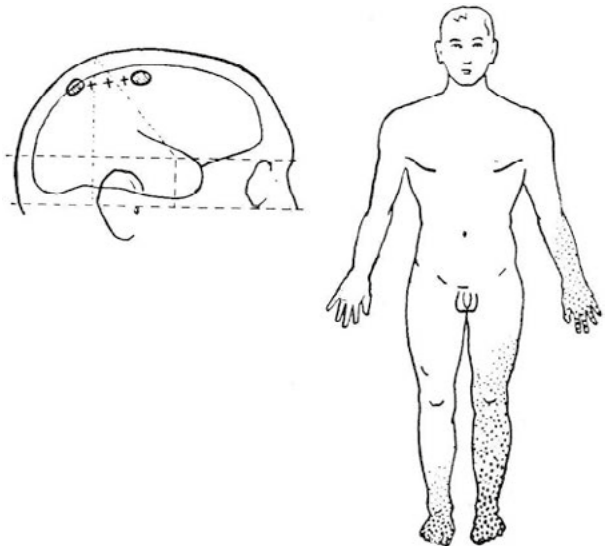

Figure 22. Case of paracentral tactile syndrome, i.e., asymmetric bilateral disorder of tactile sensitivity in unilateral "para-marginal" (paracentral) lesion. The density of the dots indicates the intensity of the disorder.

We are basically facing an *asymmetric concentric reduction of the tactile field*, similar to that of the visual field, or expressed otherwise, this is an unequal bilateral disorder in unilateral cortical lesion, with the maximum defect or greatest constriction of the sensory field on the contralateral side. When the involvement is sufficiently intense, the repercussion of the lesion (typical of a paracentral syndrome) can reach not only the whole tactile field (both sides) but other more distant brain systems depending on the type of asymmetric brain repercussion. To some extent, this is the case at hand, since there is a small constriction of the visual field, the contralateral eye reaching 75° amplitude of the visual field. This is the final case in the curve in Fig. 21, presenting a 2° tilt of the visual image.

Such tactile cases are more abundant than one might believe. If the examination of the patients were sufficiently detailed and complete, the more or less acute bilateral defect could be generalized in at least some types of lesions.

Fig. 23 shows a case of smaller tactile cortical lesion than the previous one, with a more paracentral position, close to the area of the hand. This subject had certain paresis in the fingers of the contralateral (left) hand. Tactile examination revealed a hypoesthesia in that hand expressed by the correlative deficit in functions (sensitivity, passive articular movement, Weber, pain threshold, vibratory threshold, a certain astereognosis), but there were also indications of disturbance in the homolateral hand. This is an asymmetric bilateral disorder which could be exemplified better using the corresponding thresholds of vibratory sensitivity (with electric vibrator of variable frequency) as indicated in Fig. 23. The distribution of the disorder is typical, with the defect predominating peripherally, i.e., in a cubital and distal direction. The defect is thus maximum in the contralateral little finger, although the homolateral little finger is affected in a similar manner to the contralateral thumb, and the homolateral thumb may not be completely intact. A similar result was obtained when determining the Weber thresholds.

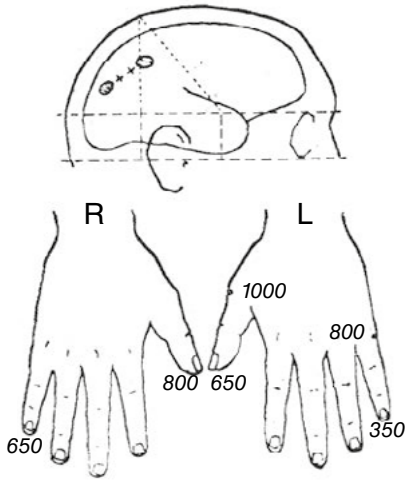

Figure 23. Asymmetric bilateral tactile disorder expressed according to the thresholds of vibratory sensitivity. Normal value: 900-1000 vibrations per second.

The other case in Fig. 24 is of the same type, but presents a disorder which is more extended to the whole body in a similar manner to the first subject (Fig. 22), although the defect in this case (Fig. 24) is much less intense. These two cases (Figs. 22 and 24) show bilateral asymmetry and also asymmetry along the axis of the body, the maximum defect corresponding to a lower extremity. According to the traditional theory, the latter defect (that of the lower extremity) is the only one that could be explained anatomically, but the disorder is really more dynamic and reaches the whole tactile field, although heterogeneously, i.e., according to a doubly asymmetric disturbance distribution.

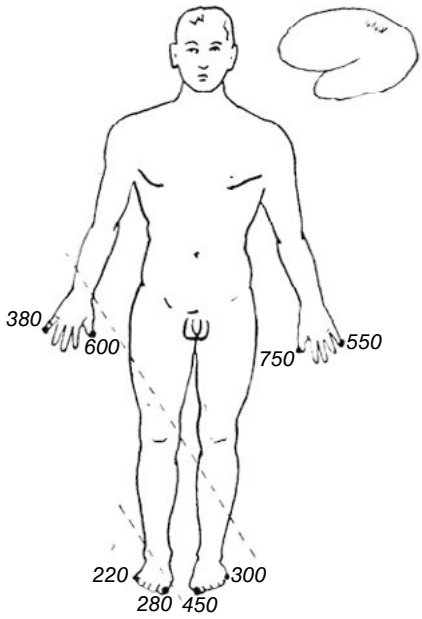

Figure 24. Bilateral tactile disorder involving a larger surface than in Fig. 23. It is determined by the same method of thresholds of vibratory sensitivity. Normal value: 900-1000. Note the predominance of cubital and peroneal disorder and the double asymmetry (lateral and vertical).

Although there are some clinical observations on this type of affection in the bibliography, it appears as an obscure matter. Foerster[19] in particular insists on the bilateral action of area 5a and 5b (area 7 according to other authors), or 'supplementary tactile area.' Many of our cases fit well this paracentral localization. It can be said that the bilateral defect does not exist in strictly "marginal" (projection paths) lesions. The asymmetric bilateral tactile defect also occurs in other paracentral zones, and even in a more symmetric manner in the central zone, as we know. One cannot ignore the fact that an important aspect of the paracentral syndrome is its transitional character to the central syndrome. Finally, it must be recalled that there are several very important classical proofs of the bilateral tactile effect from a brain hemisphere in animal physiology.

11. CONCLUDING REMARKS

The most outstanding feature of these studies on brain dynamics is its physiological basis, which is manifested from the outset through the *phenomena of dynamic action* in brain excitability, relying on far-reaching principles that lead to simpler

[19] See the reference FOERSTER (1936) in References of Vol. 1 and 2 of *Brain Dynamics*.

and more systematic notions than those of the traditional theory. To pass from a mere empiricism to a more causal theory signifies the transition from an anatomoclinic or "static" conception, to a *physiologic or dynamic* conception, common evolution to all types of knowledge. Dynamism alludes to transformation, development, function generation, etc., and in an abstract sense to continuity. Here, dynamism involves a physical and quantitative "mechanism," without resorting to other principles, and is expressed by the *value of the excitability parameters as a function of the neural mass*, by the *spiral development* and the vectors that determine it, by the *brain gradients*, etc.

Of more practical interest is to situate this brain dynamics research with respect to localist and anti-localist theories. The former, more traditional, is accepted here within the general criterion of functional regional *heterogeneity*, which should figure as a definitive acquisition, but is relegated when innumerable specific anatomic centers are to be detailed, as well as the series of autonomous nosological entities which would result from it. It can be said that localism, being starting point, it is surpassed. Functional or anti-localist orientation, at times brighter than the former but much more diffuse, in addition to being opposed to the excesses of localism, tends towards the functional analysis of syndromes and complexes which naturally must be assumed. However, it is quite imprecise in all matters, as the analysis is primarily aimed at the higher functions, ignoring the pillars of sensory organization, and is far from all physiological basis. Furthermore, the problem of functional localization in the brain is unattended, and a completely obscure and indeterminate action of totality is proposed.

Each orientation represents an approximation and plays a role within certain aims. Thus, traditional theory continues to play its role in one form or another in clinical diagnosis, theoretically evolving towards an attenuated, albeit ill-defined, localism, as a result of the conflict between opposing theories that naturally tend to complement each other.

In this research of brain dynamics, the issue of brain localization is properly approached according to the two factors of the lesion, *magnitude* and *position*, where localism (position) and anti-localism (magnitude) can be assembled. It is even more correctly approached in the *cortical gradients*, where the separation between projection and association areas is erased. In this way, the divergence between higher and lower functions also disappears. If we had to define brain activity according to these results, we would simply assert that it consists of the organization of the sensory field.

Finally, it may be said that this research of brain dynamics is a new stage connected to previous ones. As mentioned, as well as being physiological it is still anatomoclinical. In fact, it includes *anatomic* concepts (hemispheres, corpus callosum, primary and secondary areas, decussations, etc.), *physiological* concepts (excitability, summation, desynchronization, sensory parameters, cortical gradients, etc.) and *pathophysiological* concepts (central, paracentral and marginal syndromes, residual field, spiral development, etc.). It gives a new meaning to known facts and provides more original notions. It is related to well established knowledge but is directly supported by its own data. This research is in formation and in evolution. It begins in

1939 with the phenomena of dynamic action, takes further shape with the concepts of magnitude and position in 1941, and of spiral development in 1947. It later focuses on cortical *gradients* in relation to brain function localization, and on *crossings* and field *dimensions* in relation to sensory structures.

In short summary: *According to the gradient system, the more "peripheral" (closer to the projection pathway) the cortical lesion is, the greater the predominance of unisensory and contralateral defects. Conversely, the more "central" the lesion, the greater the tendency towards a bilateral and multisensory defect. The more central the lesion, the more considerable it must be to produce a functional depression. Such functional depression is manifested in the corresponding sensory field by the reduction of sensory parameters (intensity, space and time) and by the inversion process of orientation (related to decussations). Thus, the sensory field shows a spiral development, the amplitude of which is a function of the magnitude of the lesion.*

SUPPLEMENT II*

Note on Gradients, Similarity and Allometry in Brain Dynamics

Illustrations on Phenomenology and Concepts in Brain Dynamics

by

Justo Gonzalo

*** Original edition (in Spanish):**

Nota sobre gradientes, similitud y alometría en dinámica cerebral. Ilustraciones sobre fenomenología y conceptos en dinámica cerebral. Published as Suplemento II in:

GONZALO, J. (2010). *Dinámica cerebral. La actividad cerebral en función de las condiciones dinámicas de la excitabilidad nerviosa*. Edición facsimilar del Volumen 1 (1945), Volumen 2 (1950), Suplemento I (1952) y 1.ª edición del Suplemento II. I. Gonzalo Fonrodona (ed. de los suplementos). Publicado por la Red Temática en Tecnologías de Computación Natural/Artificial (RTNAC) y la Universidad de Santiago de Compostela, España 2010. Open Access: http://hdl.handle.net/10347/4341

Introduction

THIS supplement consists of a text entitled *Note on Gradients, Similarity and Allometry in Brain Dynamics,* written by Justo Gonzalo to summarize these concepts, and a selection of illustrations designed by the same author with explanatory texts by Isabel Gonzalo Fonrodona, which expand and complement the Note. Both parts were published for the first time in Spanish as 'Suplemento II' in the 2010 facsimile edition with supplements.[1]

The Note is made up of literal extracts from the reports that J. Gonzalo presented to different Spanish institutions from 1960 onwards. The text is reproduced here exactly except for minor changes and footnotes, in many of which reference is made to the illustrations. The use of certain terms and temporal references must be framed in the time in which they were written. The author introduced the concept of functional cortical gradients in 1951, published in 1952 (Supplement I), and shortly thereafter (1951-59) introduced the concepts of similarity and allometry in the alteration of sensory functions. These concepts were presented by him in doctoral courses until 1966, and have been exposed in relation to this research by other authors in a more current context.[2]

The illustrations presented here were made by J. Gonzalo between 1960 and 1975 approximately. They constitute a very small selection compared to the large

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[1] GONZALO, J. (2010). *Dinámica cerebral. La actividad cerebral en función de las condiciones dinámicas de la excitabilidad nerviosa.* Edición facsimilar del Volumen 1 (1945), Volumen 2 (1950), Suplemento I (1952) y 1.ª edición del Suplemento II. I. Gonzalo Fonrodona (ed. de los suplementos). Publicado por la Red Temática en Tecnologías de Computación Natural/Artificial (RTNAC) y la Universidad de Santiago de Compostela, España 2010. Open Access: http://hdl.handle.net/10347/4341

[2] GONZALO-FONRODONA, I. (2009). "Functional gradients through the cortex, multisensory integration and scaling laws in brain dynamics," *Neurocomputing,* **72**: 831-838.

OA: https://doi.org/10.1016/j.neucom.2008.04.055

GONZALO-FONRODONA, I. and PORRAS, M. A. (2014). In: *Horizons in Neuroscience Research,* Vol. 13, Chap.10: 161-189, and references therein. OA: https://doi.org/10.48550/arXiv.2006.01666 and https://novapublishers.com/wp-content/uploads/2019/06/978-1-62948-426-6_ch10.pdf

number of figures made for didactic purposes and for potential publications. The selected figures are related to the phenomena and concepts presented by the author in his book "Dinámica Cerebral" [Brain Dynamics] [Vol.1 (1945), Vol. 2 (1950)], in the article of 1952 (Supplement I), and are also related to concepts elaborated later, such as those presented in the following Note. In some of the figure captions, clarifying information has been added with respect to the 2010 Spanish edition.

The set of illustrations with their corresponding explanations constitutes a unit which is independent of the Note. The order of the illustrations does not follow the order in which they are cited in the Note, but according to their relationship to each other. The visual system is addressed first, then the tactile system, and finally the motor and language systems. Reference is made in the figures to the book *Brain Dynamics* and to the publication that constitutes Supplement I.

The original illustrations were made by expert draftsmen. Especially Gregorio López Sánchez and Andrés Sánchez Navarro deserve recognition for their interest and careful work during their long time dedication. The illustrations are, in general, of large size, and are formed by multiple figures connected to each other. The selection for this edition is limited to a small sample of 30 figures sufficiently general and representative, many of them being part of more complex compositions. The texts that are part of the figures have been translated and inserted trying to maintain the same style as in the original. The style is not homogeneous because the figures were made at different times and for different purposes.

In some illustrations, especially in Figures 2 and 3, the first-hand cases studied by J. Gonzalo are identified with a number (between 1 and 117). In other figures, some cases are marked with the initial of the surname. Most of the cases are wounded from the Spanish Civil War (1936-39).

ISABEL GONZALO FONRODONA

Note on Gradients, Similarity and Allometry in Brain Dynamics

LOCALIZATION OF BRAIN FUNCTIONS ACCORDING TO BRAIN GRADIENTS

Localization according to cortical gradients is an original conception which should be discussed first, and which addresses the fundamental and controversial issue of the localization of brain function in the cerebral cortex. In contrast to the dispute between localists and anti-localists authors, the conception of brain gradients undertakes more fundamental approaches in close connection with new personal observations. It comes from the brain repercussion, observed in 1939,[1] from the effect of the magnitude and position of the lesion (1941),[2] from the gradients introduced in 1951,[3] and subsequent development.[4]

Brain gradients, introduced in 1951 during doctoral courses to interpret certain visual syndromes, have now been generalized. Contrary to the numerous centers of classical localism, brain gradients represent a simple and single mechanism capable of multiple syndromes and variants. In its simplest concept, it means that in a region there is a quantitatively graded action. That is, in a region of the cortex there would be a certain functional gradient. Brain gradients would provide the spatial distribution in the cortex of the functional value of sensory or other systems.

The simplest way to represent them is by means of graded functions in an anatomical framework where the projection areas are at the extremes, and the areas of association and those of interhemispheric callosal connection are in the middle (cen-

[1] The brain repercussion consists in the multisensoriality and bilaterality of the central syndrome. See Sec. 1.3 in Vol. 1 of *Brain Dynamics*.

[2] See Sec. 2 and footnote 1 of Supplement I. Fig. 1 of Supplement II shows the types of syndromes according to the position of the lesion.

[3] See Sec. 7 of Supplement I.

[4] Figs. 2-5 show several case series according to the different types of syndromes.

tral zone) (see 1952 publication).[5] Each sensory (and also motor) system has its own gradient, with the maximum intensity next to its projection area, area which is merely the entrance to the brain, the origin. The gradient descends along the aforementioned central zone, which acts in the elaboration of all kinds of functions of the system in question.[6]

The set of projection areas is only equivalent to an inert "mosaic," according to the arrangement of nerve fibers point by point; and a lesion limited to a projection area will cause a sensory syndrome of scotoma type (partial or local suppression).[7] By contrast, the gradient zone acts very differently, as a "field," as a gradient field; and a lesion limited to a sensory gradient zone produces not only an agnostic defect, but also a sensory defect (although to a much lesser degree), thus producing a sensory syndrome of anti-scotoma type (general functional depression),[8] and according to the similarity mechanism explained below. This type of sensory gradient (there are gradients of other types that we do not deal with here) is very important to accurately determine various properties. The decrease of the gradient with the distance to the origin (projection zone) determines the unilateral-bilateral action: maximum contralateral action at the origin, and maximum callosal action in the central zone. It also determines the unisensory or multisensory action; the multisensory action by the superposition of other gradients, maximum towards the central zone. They are thus different actions that extend through the cerebral cortex and have a value that varies at each point, i.e., we have a point function or field action, in mathematically terms. It is understandable that, depending on the position and magnitude of the lesions, multiple types of syndromes are produced as a result of the combined action of the gradients. Gradients are thus the framework for describing the series of syndromes (*central, paracentral, marginal* and their intermediate transitions). These syndromes do not correspond to centers but to brain configurations of symmetrical or asymmetrical type, with different intensity of involvement, and whose anatomo-physiological explanation is immediate. The gradient, which already implies position, also encompasses the magnitude (of the lesion) given the continuity action of the gradient.[9]

The sensory gradients would be the most straightforward (well-defined sensory field in the projection area), and are gradients of more accurate determination, constituting a fundamental basis for the whole study. Other gradients for complex

[5] The 1952 publication is the one reproduced in Supplement I. In it, the author exposes the concept of functional cortical gradients in Sec. 7.

[6] See Fig. 6 and its details in Figs. 7 and 8. See also Figs. 9, 10, 21, 27, 28 and 30.

[7] See for example the right part of Figs. 1, 10, 11 and Fig. 22.

[8] 10 The central and paracentral syndromes belong to this sensory syndrome of anti-scotoma type. See, for example, the cases with concentric visual field reduction in Fig. 3, and some more asymmetric cases in Fig. 2. Figs. 6-11 and 23-26 illustrate anti-scotoma in more detail for the visual and tactile systems.

[9] See for example Figs. 6, 9, 10 and 11, where the different syndromes are shown in horizontal and the effect of magnitude on each type of syndrome in vertical. Fig. 12 illustrates the transitions between the different syndromes. The influence of lesion magnitude is shown in detail in Figs. 13 and 14.

functions can be established by analogy, and so it happens in aphasia to explain the great extension of the language area, etc.[10] In motor function, a gradient is determined with maximum precision by registering the various numerical values of electrical stimulation (in amperes) from the rolandic pyramidal area to the frontal pole.[11] Very abundant collections of first-hand cases and also from other authors are provided for demonstration, cases that are ordered in different series to show the gradual transitions inherent to the gradient.[12] Many syndromes "discarded" since long ago, as well as recent unexplained data and cases from the literature, are now easily clarified. In general, everything that was an obstacle for "localists" (there were many obstacles) becomes easy with the gradients, which are supported by copious material (clinical, experimental, etc.), and simplify and solve multiple problems.

It is important to point out that a sensory function originating in the projection area only reaches its normal value through the support of the entire gradient spread across the central zone of the cortex. This cumulative action towards the central zone constitutes the "cerebration" of such function, its growth and development.[13] Another indirect and less simple way to consider the gradient is as the *variation* at each point of the accumulated action from the origin (projection area) to that point, i.e., the gradient is the derivative of the "cerebration"[14] (of the integration). Thus, it can be said that the system is integrated (it grows and differentiates) towards the central zone. This has important applications in brain localization problems yet to be solved. Indeed, in classical localization maps, nosological syndromes are located and ordered towards the central zone as their functional complexity increases (they are further away from the origin of the system). According to what was said, such syndromes and their localization should be considered as mere "cerebration" (integration) stages of the system. Thus, the classical representations would be "cerebration" (integration) maps. The specific centers would be only an appearance, without anatomical existence, and erased by the presence of the gradient.

In conclusion, all of the above leads to a *neolocalism* that is a *quantitative* localization system according to a physical-dynamic gradient field (gradient is "anti-center"), as opposed to a *qualitative* brain localization forming a "mosaic" of anatomical centers throughout the cortex. This neolocalism also means that cortical heterogeneity, in addition to being maintained, is enriched and at the same time refined at every point, rejecting both the substantialist specifications of the "localists" authors and the psychovitalist indeterminacy of the "anti-localists" authors.

Finally, it should be noted that brain gradients lead directly, as far as the physiological mechanism of a function is concerned, to the theory of similarity. Gradients and similarity are here mutually dependent. The fact that a given activity takes different values in the different syndromes described by the gradient leads to the concept of similarity (see below).

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[10] See Fig. 28.
[11] See Fig. 27.
[12] See for example series II and III in Figs. 2 and 3, and some cases in Figs. 6, 10-12.
[13] See Figs. 14 and 18, where the term `brain integration´ is used instead of "cerebration."
[14] Whenever the term "cerebration" is used, the term `integration´ will be added in parentheses.

IMPAIRMENT OF FUNCTIONS ACCORDING TO SIMILARITY AND ALLOMETRY[15]

This conception, also original, is developed in a totally physiological and quantitative domain, and is more abstract than the previous one. It comes from the studies on asynchrony in 1939, dynamic reduction (1941-43), allometry (1951-56), similarity (1957-59), and later ones. Similarity and allometry are linked to the gradient field, and are basic mechanisms that develop and give complexity to the phenomena as well as unity to the phenomena as a whole. Classically, lesions would leave "gaps" in the brain "mosaic" destroying the corresponding specific functions, but what really happens is that lesions affect the potential of the system, which preserves its organization maintaining the functional texture but with other values, varying only in its size, that is, in its "cerebration" (integration) value. The different functions vary in this case differently, i.e., allometrically. There is thus, pathologically, similarity in the physiological texture, the functional plan is the same, and the underlying mechanism lies in a change of scale in the excitability of the system. There is similarity between the different cases, which would correspond to a homeostasis of the second type.

Excitability, which in the classical theory has no significance, is here the essential fundament, and provides a strict physiological basis for all phenomena. A first aspect, the most basic, is that a residual system shows a shift towards higher thresholds in all types of functions, which is related to the reduction of cortical mass (reduction in neuronal population). This reduction is also correlated with the most intrinsic alteration of excitation permeability, i.e., spatial and temporal summations (which depend on the degree of threshold shift). All excitability curves shift, whatever their type, following the same laws as in the normal subject but varying the parameters according to the number of subsisting neurons. Thus, a similarity of curves is obtained, i.e., a family of curves in mathematical terms.[16] Many phenomena or syndromes now find an explanation. One of the most typical phenomena of similarity is the anti-scotoma type defect (mentioned in the previous section) in which the configuration of the sensory field is preserved but at a smaller size (general depression, constriction, etc.). Thus, different degrees of pathological cases of a sensory system are formally similar, or rather, isomorphic to each other.[17] That is to say, the physiological organization is flexible, can redistribute its excitability values, can vary its dimensions, reaching a new equilibrium while maintaining the same organizational plan. Nothing simpler and different from what is usually admitted. In short, the system maintains the same functional plan although its mass is reduced.

A second, somewhat more complex aspect arises when dealing with the heterogeneity of a system affected by the aforementioned change of scale. The result is

[15] The concept of scale change as well as the associated similarity and allometry are discussed in the publications referenced in footnote 2 in the Introduction of the present Supplement II.

[16] See Figs. 3, 4 and 5 in Supplement I, and Fig. 15 in the present Supplement II, showing different degrees of central syndrome.

[17] See, for example, the similarity between different cases of anti-scotoma to different degree due to different magnitude of the lesion in Figs. 9, 11, 23 and 26.

that the different functions of the system vary to a different degree since each of them would have its own neuronal quantity. Therefore, they behave allometrically, causing the disaggregation and asynchrony of functions -some of them being excluded-, which leads to the *dispersion* of the system.[18] Any function that could be taken as the simplest and most irreducible one, undergoes a disaggregation or decomposition into its simplest elements. In this way, everything is composite, or in other words, the decomposition is unlimited. It is much more important to emphasize now that in such dispersion there is a constant order (sequence) in the decomposition of functions because each element has its physiological characteristic.[19] Physiological differences between functions already exist in some degree (in signs) in the normal subject, and the change (reduction) in the size of the system just amplifies them. Such a well-determined fixed order is a very significant property of the maintenance of the organization in the sensory system. Although some "distortion" may occur due to the allometry, nevertheless the sequence and plan persist.

Nosological syndromes deserve a separate mention. They are not "pure" since the whole system is affected, albeit allometrically ("localist" authors only pay attention to the most affected function). Nor is there the "all-or-nothing" alteration admitted by the classical authors, but rather degrees of alteration (insufficiency, and more severely, abolition). In other words, there is a residual system of size according to the lesion suffered, and the syndromes or symptoms arise according to an allometric dispersion. Finally, the exceptional cases with a very residual (i.e., very small) system in which the functions are highly dispersed (disaggregated), reveal through their pronounced dispersion the root of the system, which involves the remarkable phenomenon of spatial inversion (see the book *Brain Dynamics*).[20]

A third and last aspect leads to a higher order of similarity by converting the above-mentioned qualitative allometry into quantitative allometry. In the change of excitability of the system, the differently varying parts (functions) show an equilibrium between them, being linked by the allometry law of the type $y = b\,x^n$, where x and y are different brain functions, and b is a constant. This leads to a bundle of curves where n has a different value in each curve and gives the allometric coefficient for each particular function.[21] Such a relationship is very fundamental since it governs the morphological and even biochemical growth of organisms, and now the brain sensory growth. An allometric relationship was empirically demonstrated in a series of 24 cases of different intensity. This relationship was between the tilt of the visual image and the (reduced) amplitude of the visual field.[22] The allometric coefficient n of each function, which is the dispersion (dissociation) coefficient, is also a valuable indicator of the true specificity of each function.

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[18] See Figs. 19, 20, 24 and 29.

[19] See lower part of Fig. 18, and Figs. 24, 25 and 26 showing anti-scotoma type involvement.

[20] Vol. 1 and Vol. 2 of the book *Brain Dynamics* are included in the present edition. Spatial inversion is also covered in summary form in Supplement I.

[21] See Figs. 17, 19, 20 and 29.

[22] See Figs. 17 and 19.

Synthetically, gradients give the brain localization of the systems, whereas similarity and allometry reveal the functional mechanism of such systems. A functional system is composed of a bundle of homologous gradients, more or less united, which are pathologically affected and dissociated (separated) giving rise to allometric gradients.[23]

The brain dynamics developed in these studies lead to a neolocalism of allometric gradients. It constitutes a neurophysics of the cerebral cortex. The cortex would be a system organized in gradients, which changes its metric scale in lesions, preserving the same functional plan (functional similarity), and whose multiple particular functions change allometrically according to their respective allometric coefficients. In these studies, functional variation in all activities is studied phenomenologically, giving rise to series of syndromes, families of curves, etc., in contrast to the isolated syndrome of classical pathology. The facts increase and become more complicated, but the explanation has been simplified and unified, i.e., rationalized. The progress achieved lies in providing a maximum of data (phenomena, syndromes, etc.) with a minimum of explanatory bases. Inversely, by suppressing phenomena, and remaining in simple empiricism, one returns to the localism of a simple substantialist "mosaic" of centers.

JUSTO GONZALO

[23] See Fig. 28.

ILLUSTRATIONS ON PHENOMENOLOGY AND CONCEPTS IN BRAIN DYNAMICS

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ILLUSTRATIONS ON PHENOMENOLOGY AND CONCEPTS IN BRAIN DYNAMICS

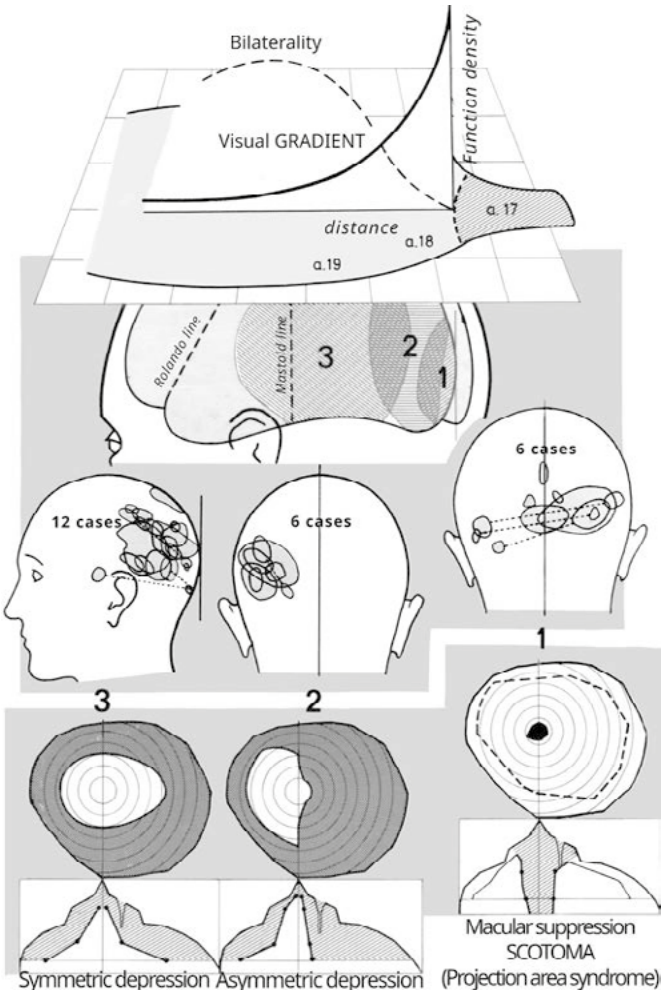

Figure 1. Lesions and syndromes. Different types of cortical lesions corresponding to 24 cases of war wounded studied by J. Gonzalo are shown. In the figure, only visual impairment is shown in the visual field and in the sensitivity profile, and in only one eye. **1:** Syndrome here called *marginal* (or of the projection area), which in these cases presents macular scotoma in the visual field. **2**: Syndrome here called *paracentral* syndrome, with asymmetric anti-scotoma. **3**: Syndrome here called c*entral* syndrome. It is multisensory and bilaterally symmetric, with concentric reduction of the visual field (symmetric anti-scotoma). It is remarkable the wide parietal area that participates in the visual field. Bottom: Visual sensitivity profiles where the shaded part means lost sensitivity. Upper part: Schematic of visual function density in gradation across the cortex (visual gradient).

This figure is related to Secs. 2 and 7 (Fig. 19) of Supplement I.

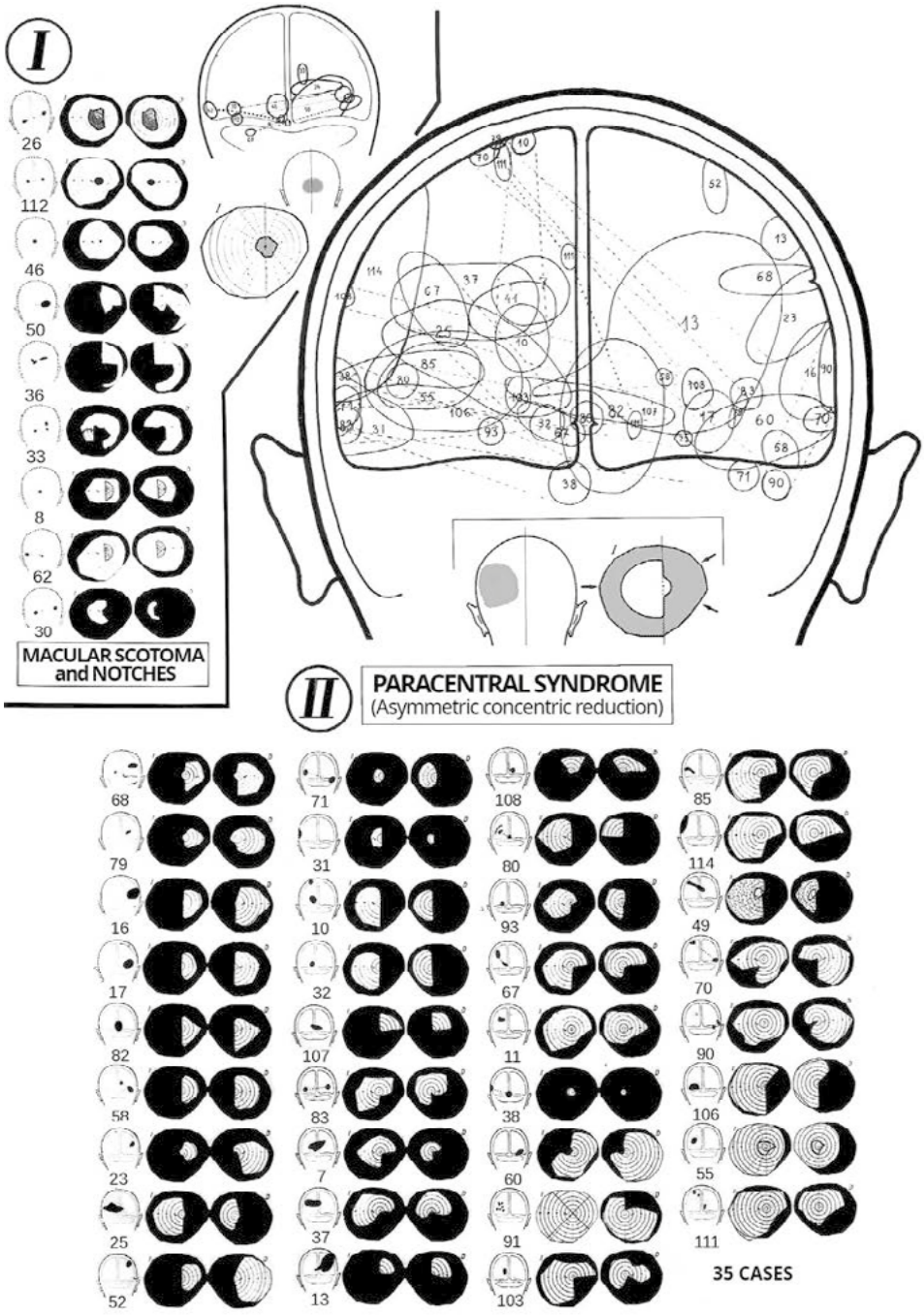

Figure 2.

Figure 2. Series of cases studied by J. Gonzalo. Lesions and visual fields. The different cases are denoted by a number. The dark parts are the altered parts.

Series **I**: Nine cases with macular scotoma and notches in the visual fields. The area of lesion and visual fields of both eyes are indicated. At the top of this series all the lesions are shown together. Just below, the area of the lesion and the type of visual field alteration are shown schematically. The syndrome presented by these cases is called by the author: *impure marginal* syndrome. Fig. 4 shows this series in more detail.

Series **II:** Thirty-five cases of *paracentral* syndrome with various *asymmetric* concentric reductions of their visual fields. In the upper part, the areas of the corresponding lesions are indicated with the number that identifies each case. In smaller size and for a single hemisphere, the area of the lesions and the type of visual field alteration are shown schematically.

These series complete those of Figs. 16 and 17 in Supplement I.

The original of this figure together with the original of the next Fig. 3 form a composition of large size (approximately 150 x150 cm), and is reproduced here from a reduced photograph.

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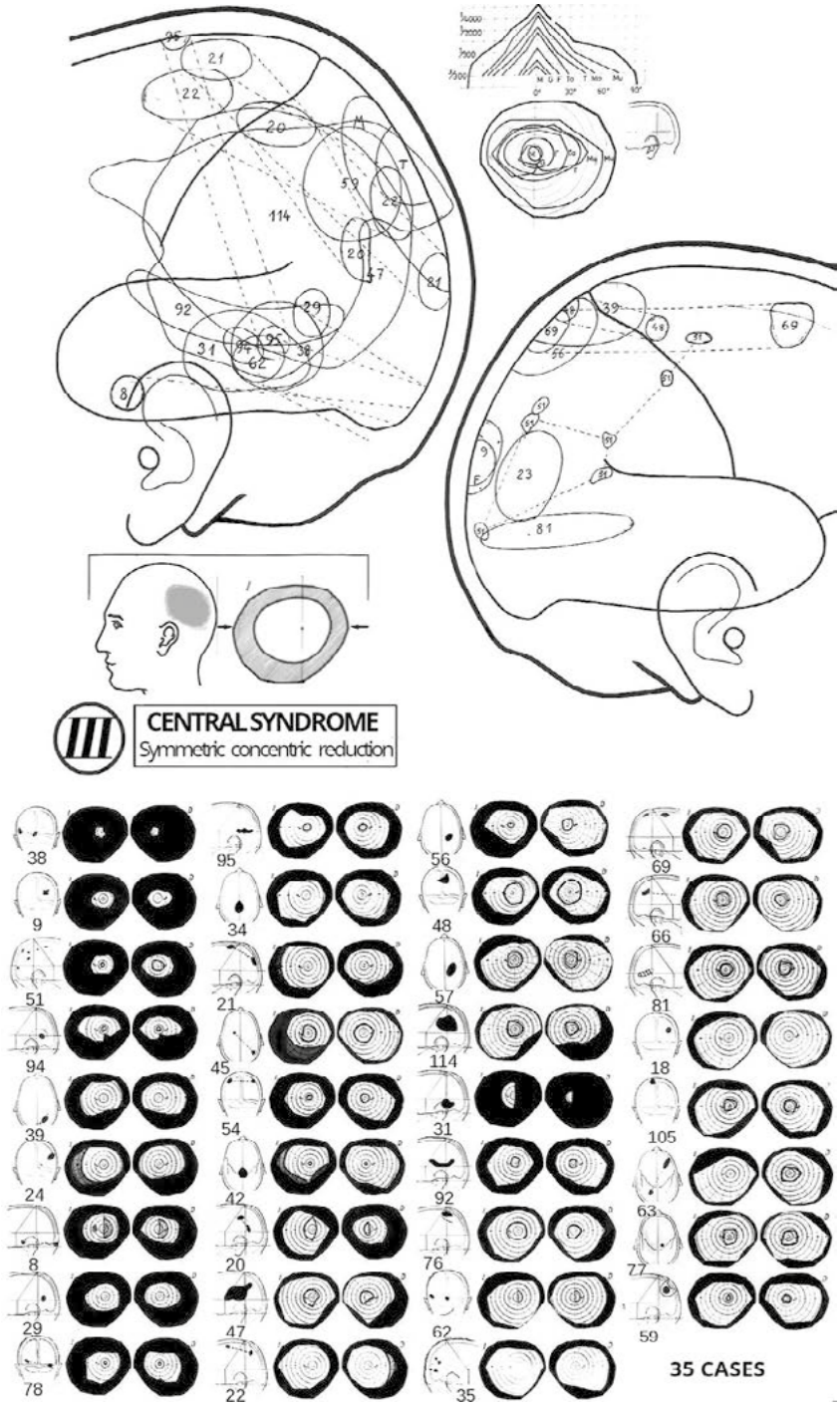

Figure 3

Figure 3. Series of cases studied by Gonzalo (continued from Fig. 2).

Series **III**: Thirty-five cases of multisensory and symmetric syndrome, i.e., *central* syndrome, of varying intensity, with *symmetric* concentric reduction of the visual fields. The numbers identify the cases. This series completes the series shown in Fig. 18 in Supplement I, and is in close connection with Vol. 1 of the book *Brain Dynamics*. In the upper part of the figure the areas of the corresponding lesions in both the right and left hemispheres are indicated. A small schematic diagram shows in a general way the area of the lesion for a single hemisphere and the type of visual field alteration. At the top of the figure, visual sensitivity profiles have been plotted for different cases with different concentric reductions, which is further detailed in Fig. 15.

(Same conditions of reproduction of the figure as Fig. 2.)

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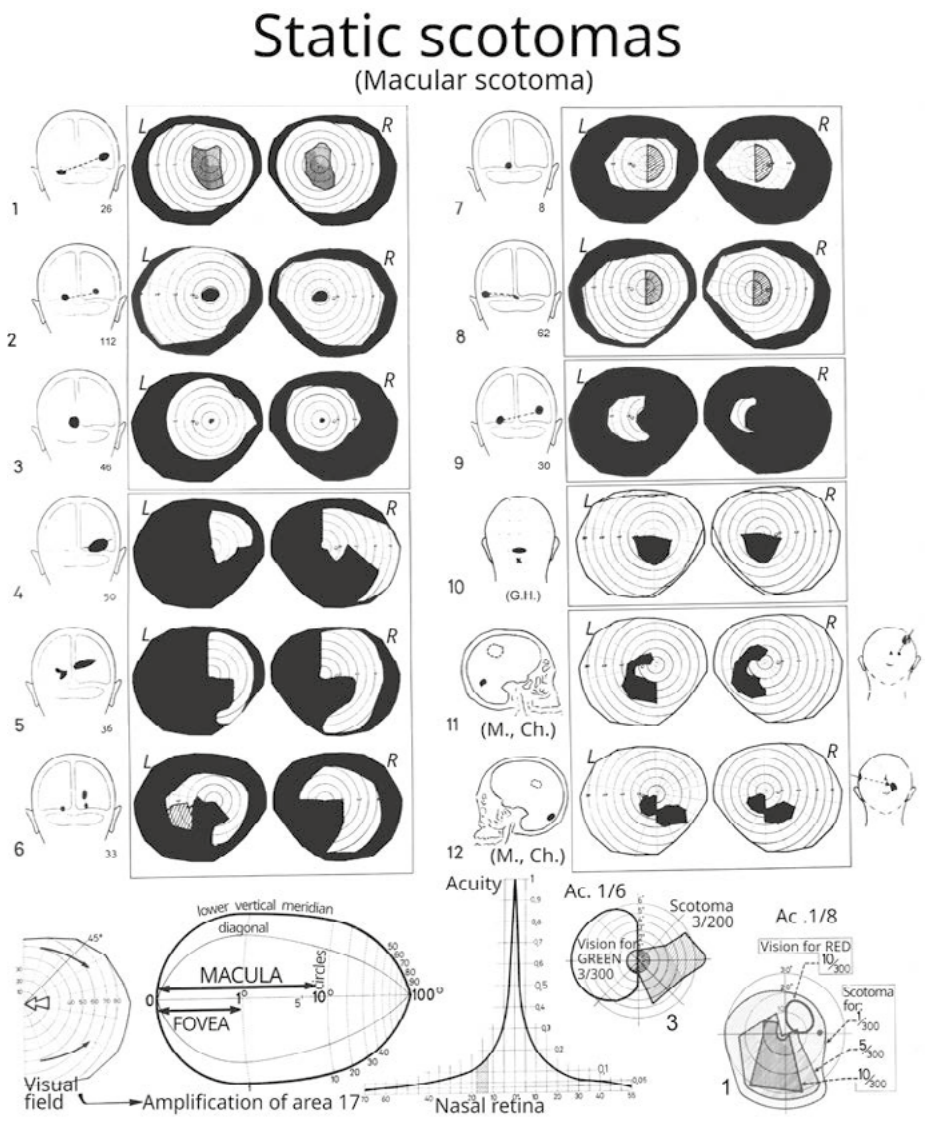

Figure 4. Macular scotoma. Cases 1 to 9 constitute series **I** in Fig. 2. All cases present macular visual scotoma, and are grouped by similarity of the disorder. As they have no dynamic effects, they are called *static* scotomas. Cases 11 and 12 are from P. Marie and J.C. Chatelin [*Rev. Neurologique*, **I**, 882 (1915)]. Below left is a neural enlargement of area 17 (Brodman's terminology). On the right, acuity as well as sensitivity of vision is indicated for green (in the third case) and red (first case). The expression 3/200 means that the test is a disk of 3 mm diameter at a distance of 200 mm from the observing eye. Similarly, *n*/300 corresponds to a test of *n* mm in diameter at a distance of 300 mm from the observing eye. The figure is in relation to Fig. 16 of Supplement I.

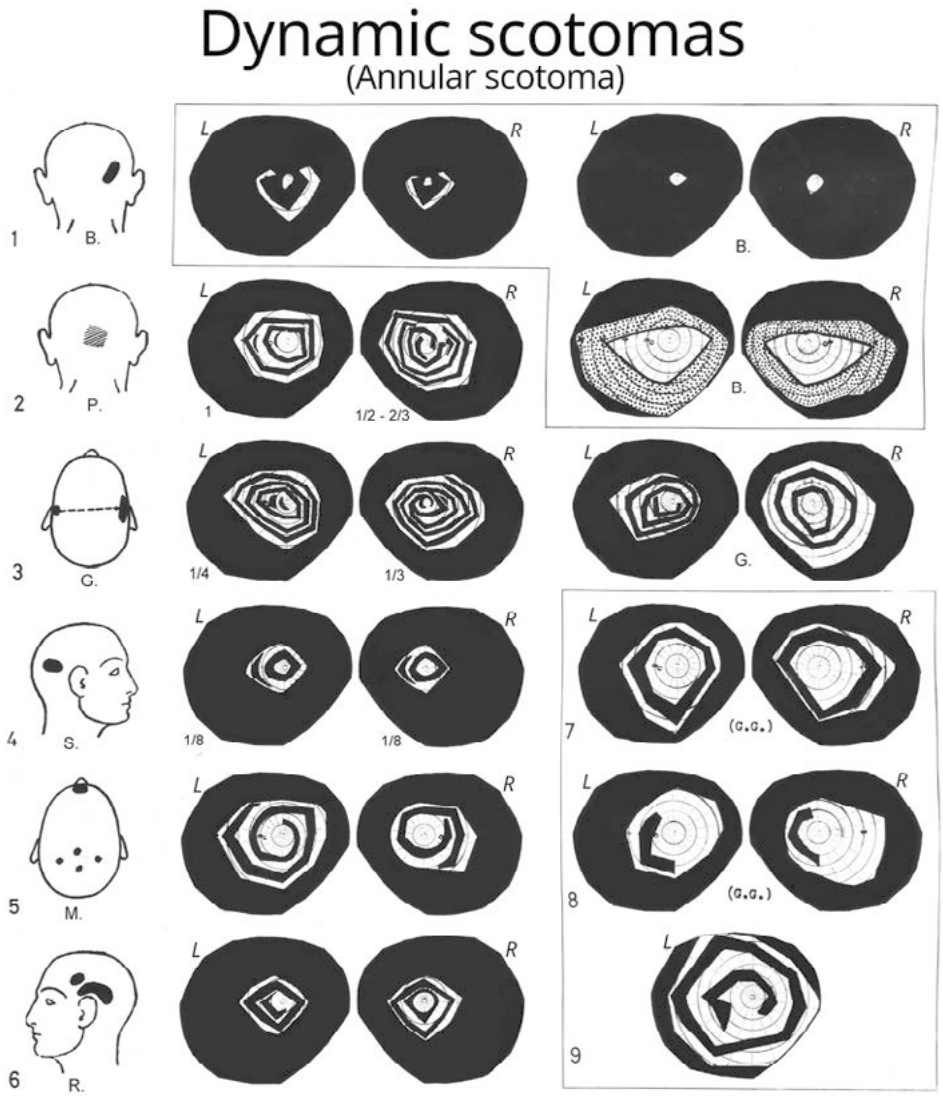

Figure 5. Annular scotoma. Nine cases presenting annular visual scotoma, changing according to the intensity of the stimulus (dynamic scotoma). Three different situations are shown for the first case, and two for the third case. The first six cases are from Gonzalo, not included among the 117 mentioned in the Introduction. In cases 2, 3 and 4, visual acuity is indicated below the visual field. Cases 7, 8 and 9 are from A. Gelb and K. Goldstein [*Albrecht von Graefes Archiv für Ophthalmologie*, **109**, 387 (1922)].

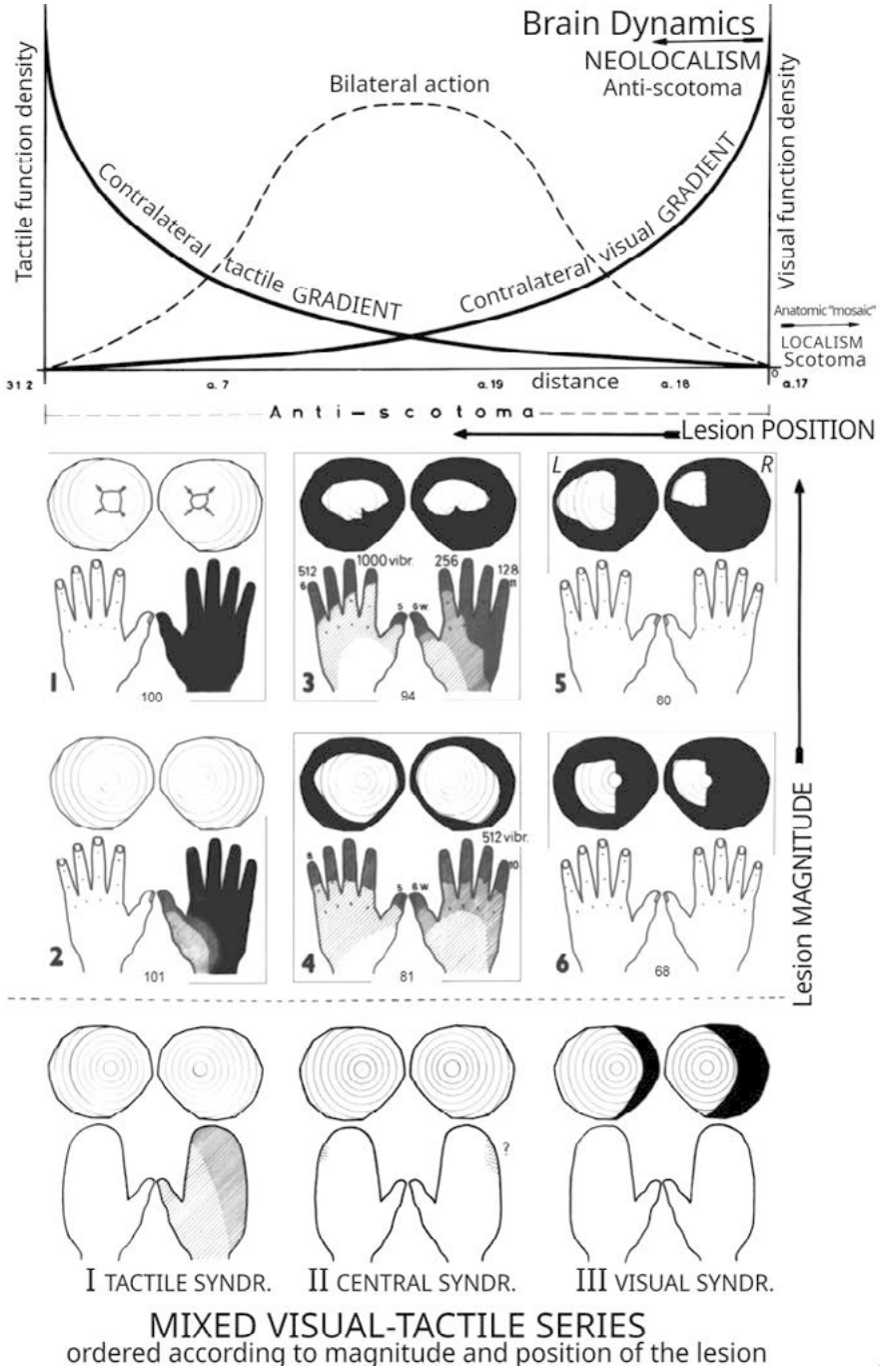

Figure 6

Figure 6. Diagram of gradients and syndromes. Top: Schematic diagram of visual and tactile gradients. The curves that take maximum value in the visual and tactile projection areas represent respectively the densities of visual and tactile function, spreading in gradation across the cortex. The areas (in Brodman's terminology) are indicated on the horizontal axis. These curves are the *specific tactile and visual gradients*, with contralateral action. The central bell-shaped curve represents the bilateral action (by the corpus callosum), and the multisensoriality due to the overlapping of the specific gradients. That curve is maximum in the zone called "central" by the author, and minimum in the projection areas. The arrow at the top of the graphs indicates that brain dynamics is manifested in the zone indicated by the arrow.

Bottom: Six cases studied by Gonzalo. They are arranged according to the position of the lesion (horizontal axis of the upper diagram), and magnitude of the lesion as indicated by the vertical arrow. The most affected parts are darker. Some vibrational sensitivity data on the hands as well as weber two-point discrimination threshold are indicated (see details in the following Fig. 7). Cases 1 and 2 in the figure (denoted by the small numbers 100 and 101 in the set of 117 cases) are tactile cases in which there is usually no visual involvement unless the magnitude of the lesion in the tactile area is sufficiently large as is the case in case 1 in which there is a very slight impairment in the visual field. Cases 3 and 4 are central syndromes with symmetrical multisensory involvement. Case 3 is more intense than case 4 because the lesion is of greater magnitude. Cases 5 and 6 present only asymmetric visual involvement. These six cases are detailed in the following Fig. 7. The last arrow schematically depicts three hypothetical cases with smaller lesions.

The present figure is closely related to Sec. 7 and Fig. 20 of Supplement I, to the articles mentioned in footnote 2 in the Introduction of this Supplement II, and to the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

This figure is an adaptation of figure 5 of the article by I. Gonzalo and A. Gonzalo (1996) in R. Moreno-Díaz and J. Mira-Mira (eds.), *Brain Process, Theories and Models. An International Conference in Honor of W.S. McCulloch,* The MIT Press, pp 78-87. The figure is reproduced here with permission of The MIT Press.

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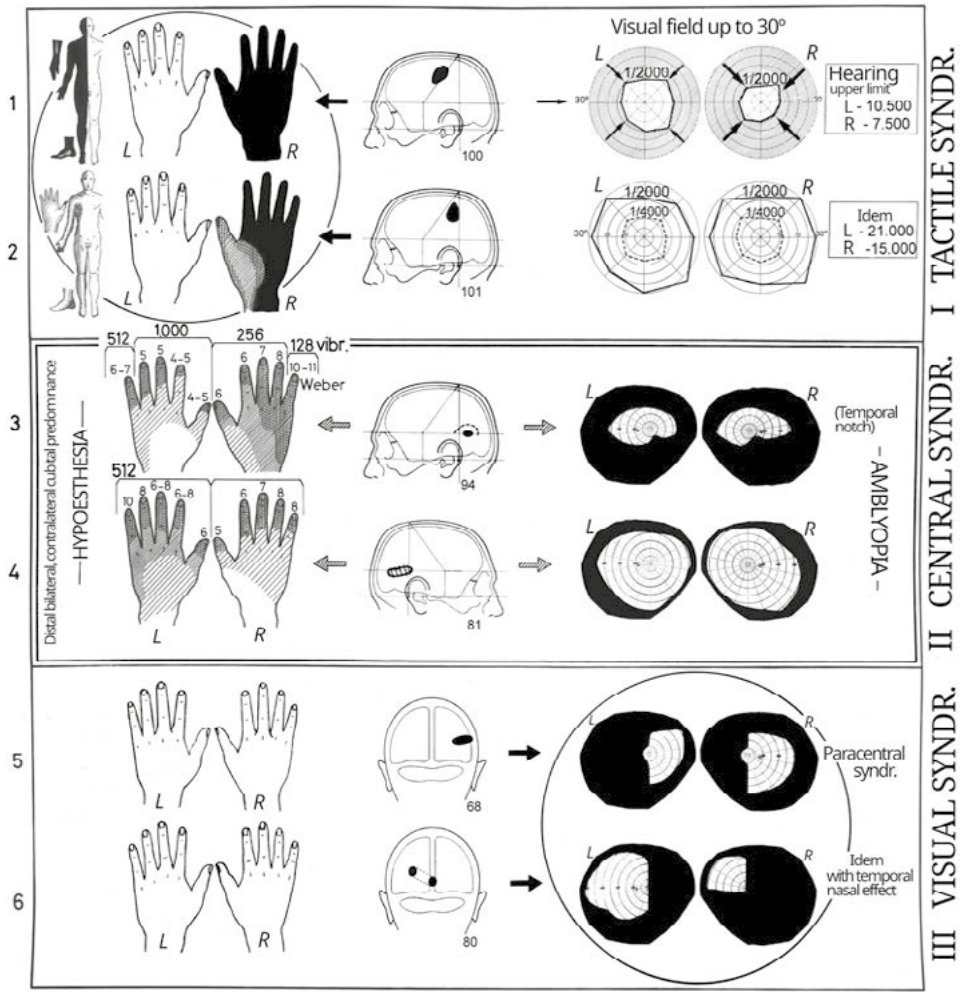

Figure 7

Figure 7. Detail of the cases of Fig. 6. The cases are indicated with the same number as in Fig. 6. The area of the lesion is shown. The cases are grouped according to the three types of syndromes (tactile, central, visual), showing tactile and visual disorders. In each group there is one case more intense than the other. The most affected parts are darker.

In tactile syndrome we have two cases. They are not strictly pure tactile syndromes but rather *tactile paracentral* syndromes, especially the more intense one. Visual fields are shown only up to 30°, with shading indicating the defect in the most intense case. Contours of equal visual sensitivity are also shown to appreciate how this sensitivity is altered in the most severe case (the upper case). The expressions 1/2000 and 1/4000 mean that the test is a white disk of 1 mm diameter at a distance of 2000 mm and 4000 mm respectively from the observing eye. In hearing, the upper limit values for high frequencies (in Hz) are indicated for each ear. The hearing impairment is more pronounced in the more severe case.

In central syndrome, the threshold frequency (in Hz) to vibratory sensitivity as well as Weber two-point discrimination threshold (in mm) –smaller numbers– are indicated on the hands.

In visual syndrome we have two cases that are *visual paracentral* syndromes.

This figure is closely related to Sec. 7 of Supplement I.

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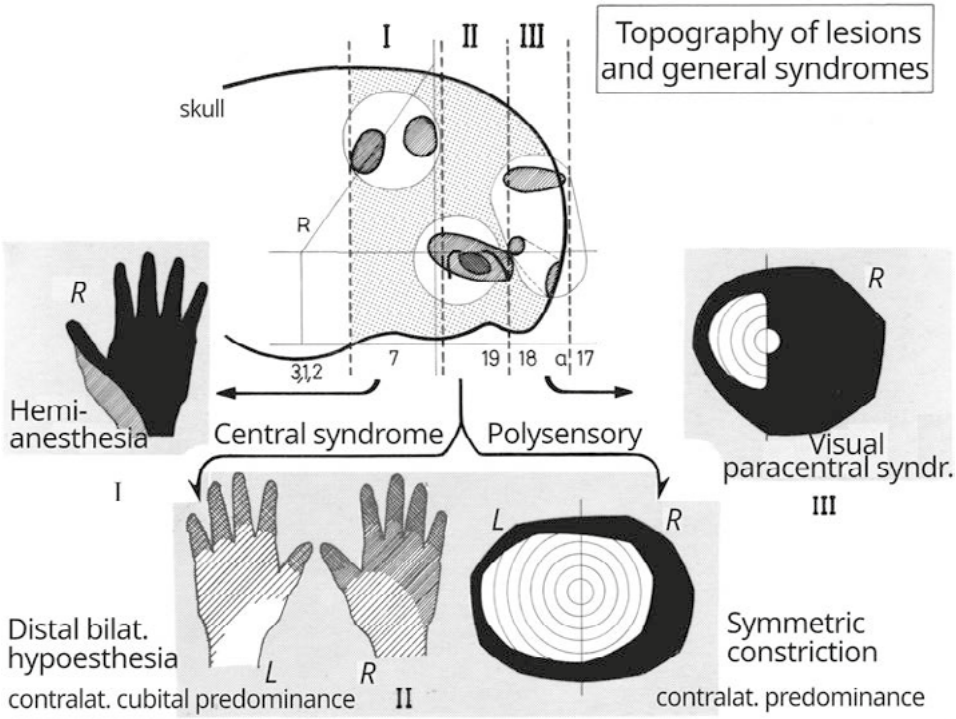

Figure 8. Topography of lesions and syndromes. The upper part shows the lesions in the indicated areas I, II and III in the skull. These areas correspond to the following syndromes: I, tactile type; II, central syndrome (multisensory and bilateral) with symmetric concentric reduction of the visual field; and III, visual paracentral syndrome. In the case of central syndrome, the concentric visual field reduction shows some contralateral predominance. Below the skull, the areas are denoted according to Brodman's nomenclature.

Figure in connection with Secs. 2, 7 and 8 of Supplement I.

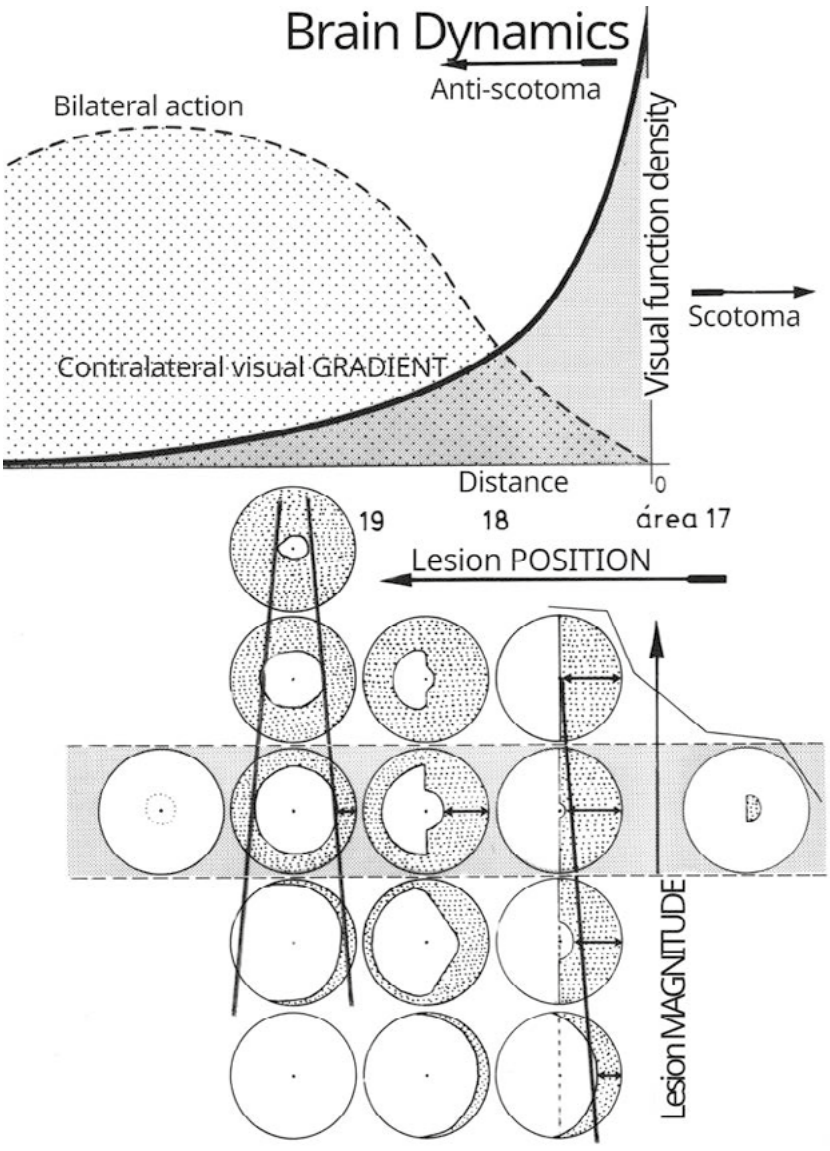

Figure 9. Schematic diagram of the visual gradient. The effect of the position (horizontal axis) and magnitude (bottom to top) of the lesion on the visual field is shown schematically. The tilted straight lines emphasize the *continuity* in the constriction of the visual field as the magnitude of the lesion increases. The series on a horizontal shaded background shows the *continuous* transition between the different syndromes as the position of the lesion varies.

Figure in connection with Secs. 2 and 7 of Supplement I, with the articles mentioned in footnote 2 in the Introduction of this Supplement II, and with the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

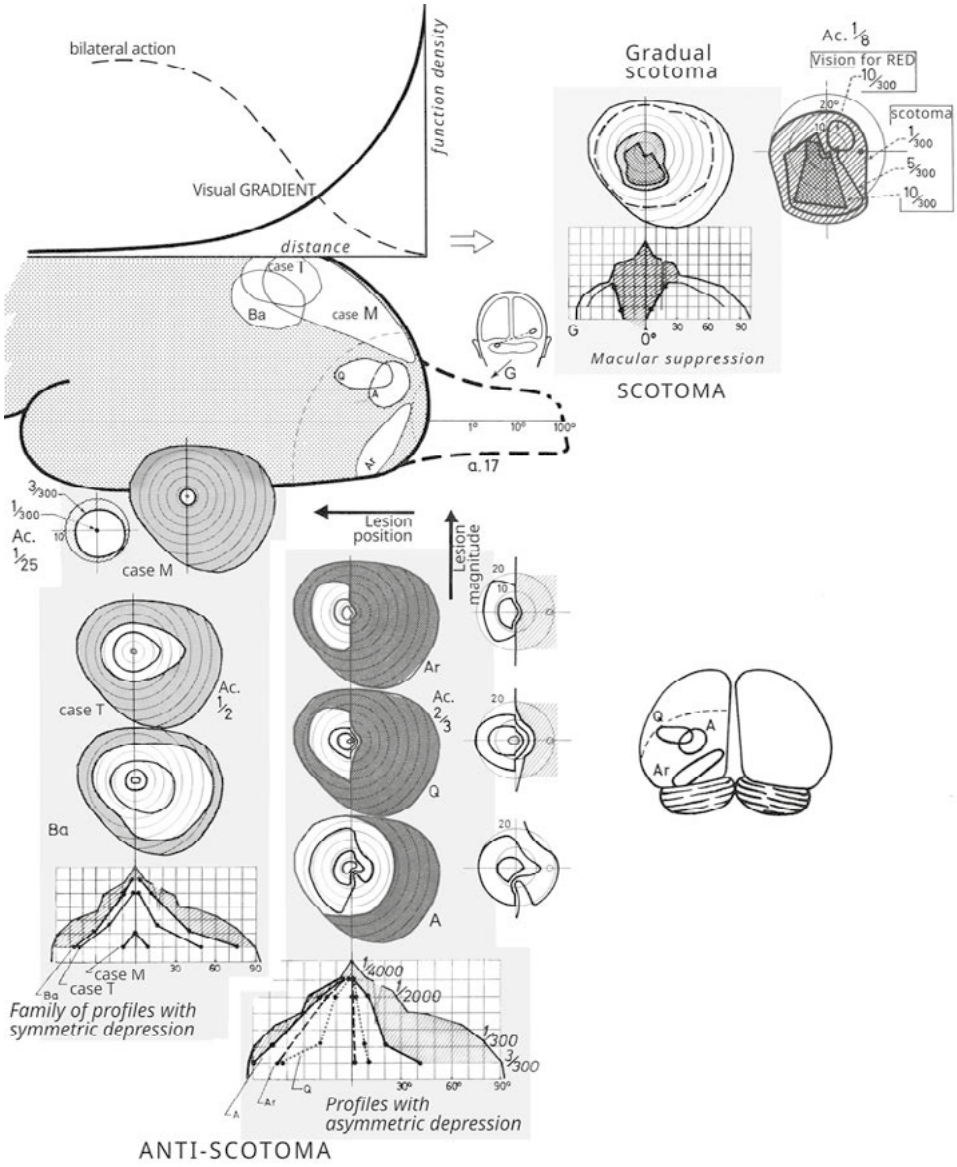

Figure 10

Figure 10. Effect of the position and magnitude of the lesion. Below the diagram of the visual gradient function (upper left part), seven cases from Gonzalo are shown, indicated by the initial of the name; among them, the M and T cases of central syndrome, studied in great detail in the book *Brain Dynamics*. This figure illustrates with real cases the scheme of the previous Fig. 9. The diagram of the brain shows the areas of the corresponding lesions and thus their distances to the visual projection zone (right origin of the horizontal axis). Only the alteration in the right eye is shown for simplicity. The dark parts are the affected ones.

The first column of visual fields corresponds to three cases of *central* syndrome ordered by their intensity. It can be seen that the disorder is rather symmetrical.

The second column shows three *visual paracentral* cases, also ordered by intensity. In these cases, the disorder is asymmetrical. For these paracentral cases their corresponding lesions are again indicated in a small sketch on the right. The visual sensitivity profiles are shown below. The expressions 1/4000, 1/2000, 1/300, 3/300 mean that the test is a white disk of 1 mm (or 3 mm in the latter case) diameter at respective distances of 4 m, 2 m, and 0.3 m from the observing eye.

A case with visual macular scotoma is shown in the upper right of the figure. It corresponds to the first case of Fig. 4. As said, the notation *n*/300 means that the test is a disk of *n* mm diameter at a distance of 300 mm from the observing eye.

Detail of isopters and visual sensitivity profiles are shown in the following Fig. 11.

Figure in connection with Secs. 2, 3 and 7 of Supplement I, Vol. 1 of *Brain Dynamics*, and with the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

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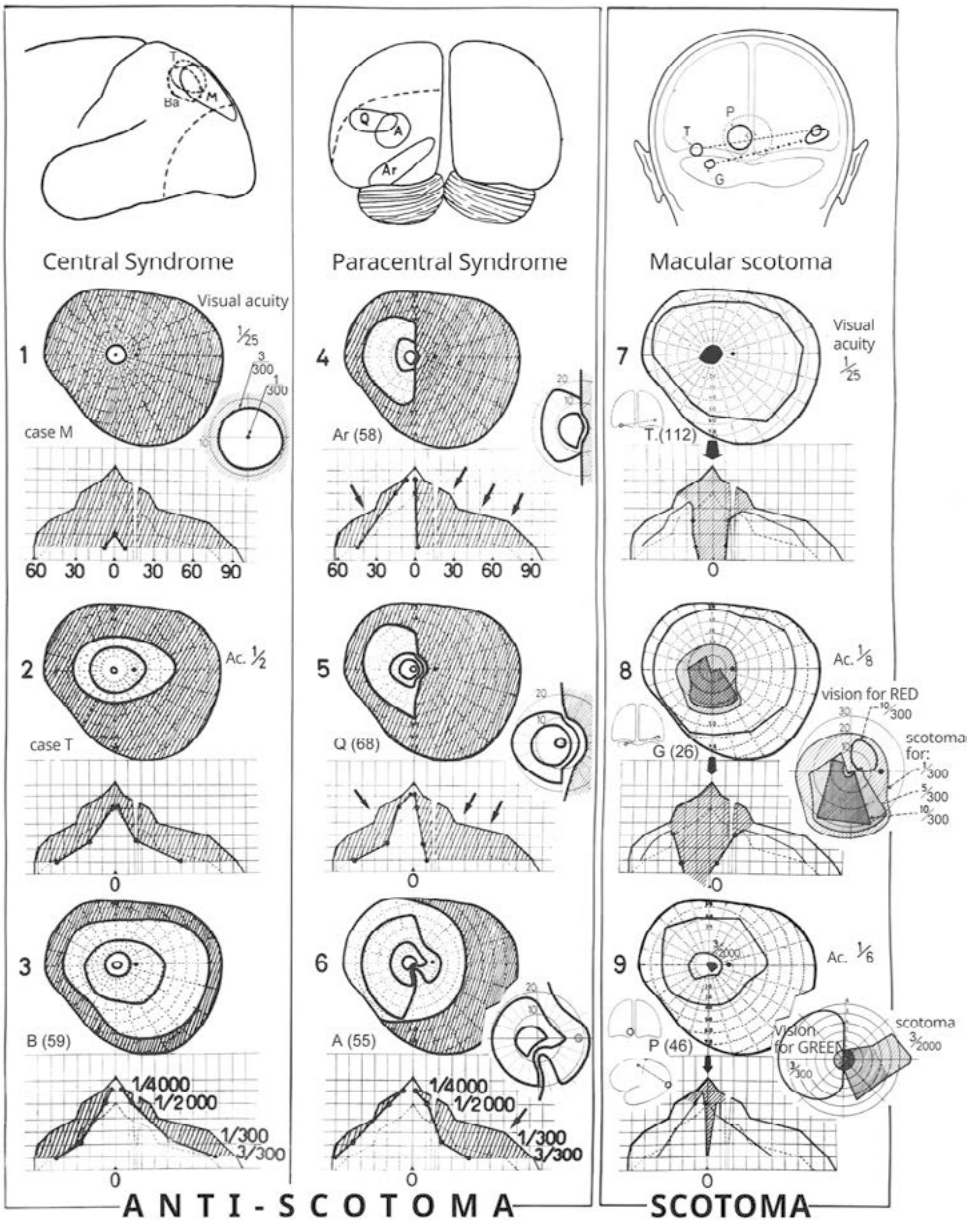

Figure 11

Figure 11. Effect of the position and magnitude of the lesion (continued). Variation of visual sensitivity with the magnitude of the lesion. This figure shows the same cases as in the previous Fig. 10 and two more cases studied by Gonzalo with macular scotoma. Isopters and visual sensitivity profiles are shown. As in Fig. 10, only the alteration in the right eye is shown for simplicity. Shaded surfaces correspond to altered parts. The intensity of the disorder decreases from top to bottom. For each of the three types of syndrome (central, paracentral and macular scotoma), it can be seen how the visual sensitivity varies according to the intensity of the disorder. The expressions 1/4000, 1/2000, 1/300, etc., ($n/\mathcal{N}$ in general), mean that the test is a disk of n mm diameter at a distance of $\mathcal{N}$ mm from the observing eye.

First column (*central* syndrome): The M, T and B (59) cases. The isopters and the sensitivity profiles maintain approximately the shape of the normal case but showing a symmetrical depression, extremely pronounced in the M case.

Second column (*paracentral* syndrome): Cases named Ar (58), Q (68) and A (55). The depression of the visual sensitivity profile is asymmetric, as well as the shape of the isopters.

Third column (macular scotoma): Cases named T (112) (different from the T case in the first column), G (26) and P (46), already shown in Figs. 2 and 4. In these cases the sensitivity profile is altered in the center. For G (26) the sensitivity is shown for red (as in Fig. 4 and 10), and for case P (46) it is shown for green (as in Fig. 4). The T (112) case is also shown in Fig. 1 as an example of projection syndrome, presenting blindness in the center.

The lesions of the corresponding cases are indicated at the top of each column.

This figure is in connection with Secs. 2, 3 and 7 of Supplement I, and Vol. 1 of *Brain Dynamics*.

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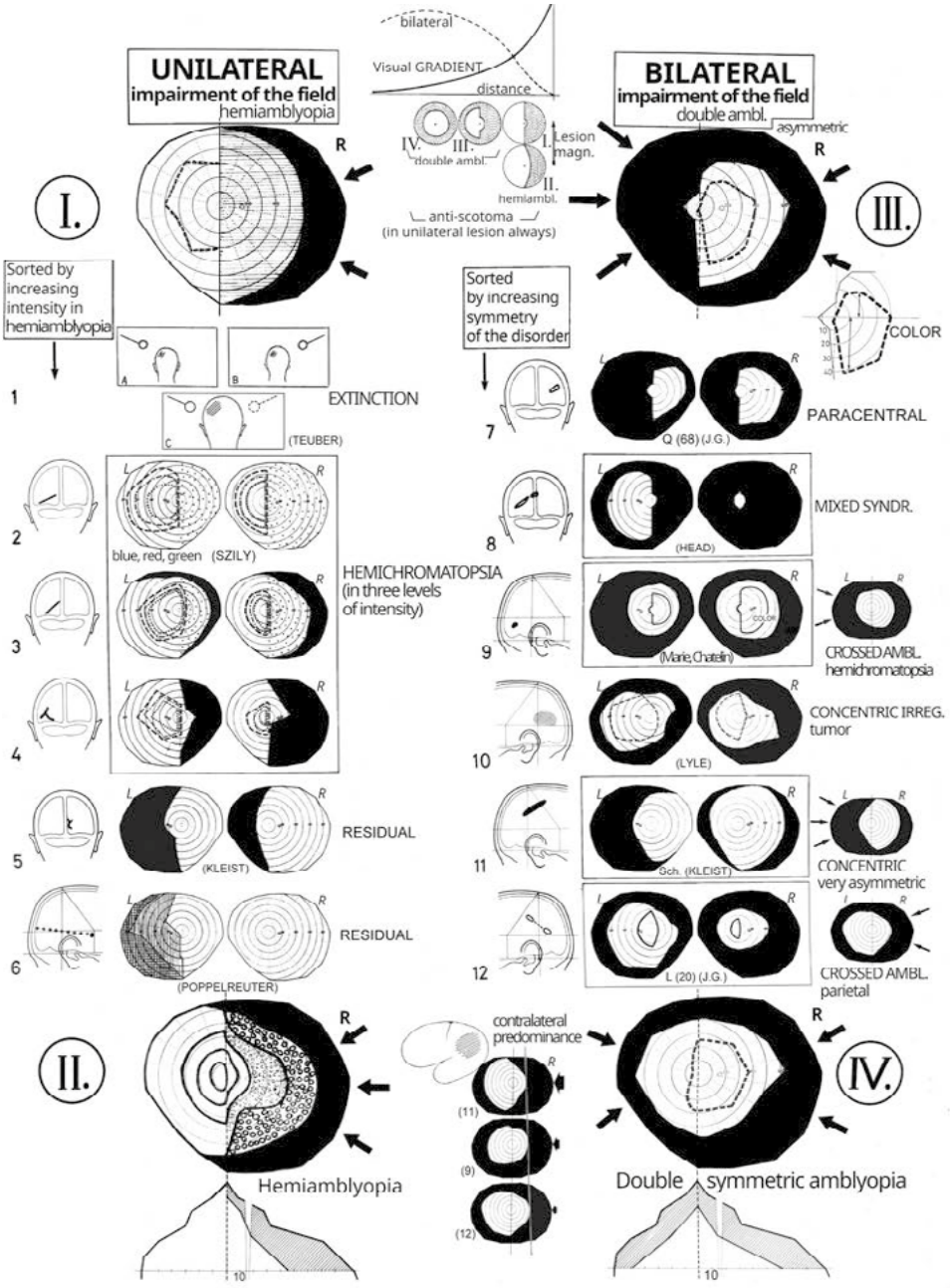

Figure 12

Figure 12. Transition between different syndromes. Cases from different authors are shown. The initials J.G. stand for J. Gonzalo. The cases are ordered according to the types I, II, III and IV, indicated in the upper central small diagram. The four large visual fields in the corners of the figure show respectively types I, II, III and IV. The darkest part is the most severely affected.

The unilateral series from type I to II consists of 6 cases that differ from each other mainly in the *magnitude* of the lesion. Case 1 is from H. L Teuber and M. B. Bender [*J. Gen. Psychol.,* **40**, 37 (1949)], cases 2, 3 and 4 are from A. von Szily (*Atlas der Kriegsaugenheilkunde,* Stuttgart 1916), case 5 is the Häfke case reported by K. Kleist (*Gehirnpathologie*, Leipzig: Barth 1934), and case 6 is from W. Poppelreuter (*Die Psychischen Schädigungen durch Kopfschuss in Kriege 1914-1916,* Leipzig: Voss 1917).

The transition from type III (asymmetric defect) to type IV (symmetric defect, central syndrome), which differ from each other mainly by the position of the lesion, is illustrated by the bilateral series of cases numbered from 7 to 12, being 7 and 12 reported by Gonzalo. The others are from H. Head [*Aphasia and Kindred Disorders of Speech,* Cambridge: Univ. Press 1926, (clinical case nº 11)], P. Marie and J. C. Chatelin [*Rev. Neurologique,* **1**, 138 (1916)] and K. Kleist [*Gehirnpathologie,* Leipzig: Barth 1934 (the Schmidt case)], as indicated. Three visual fields of three cases from J. Gonzalo [(11), (9), and (12)] are added to this last type of progression, below in the figure, in smaller size.

Figure in connection with Secs. 2, 3 and 7 of Supplement I.

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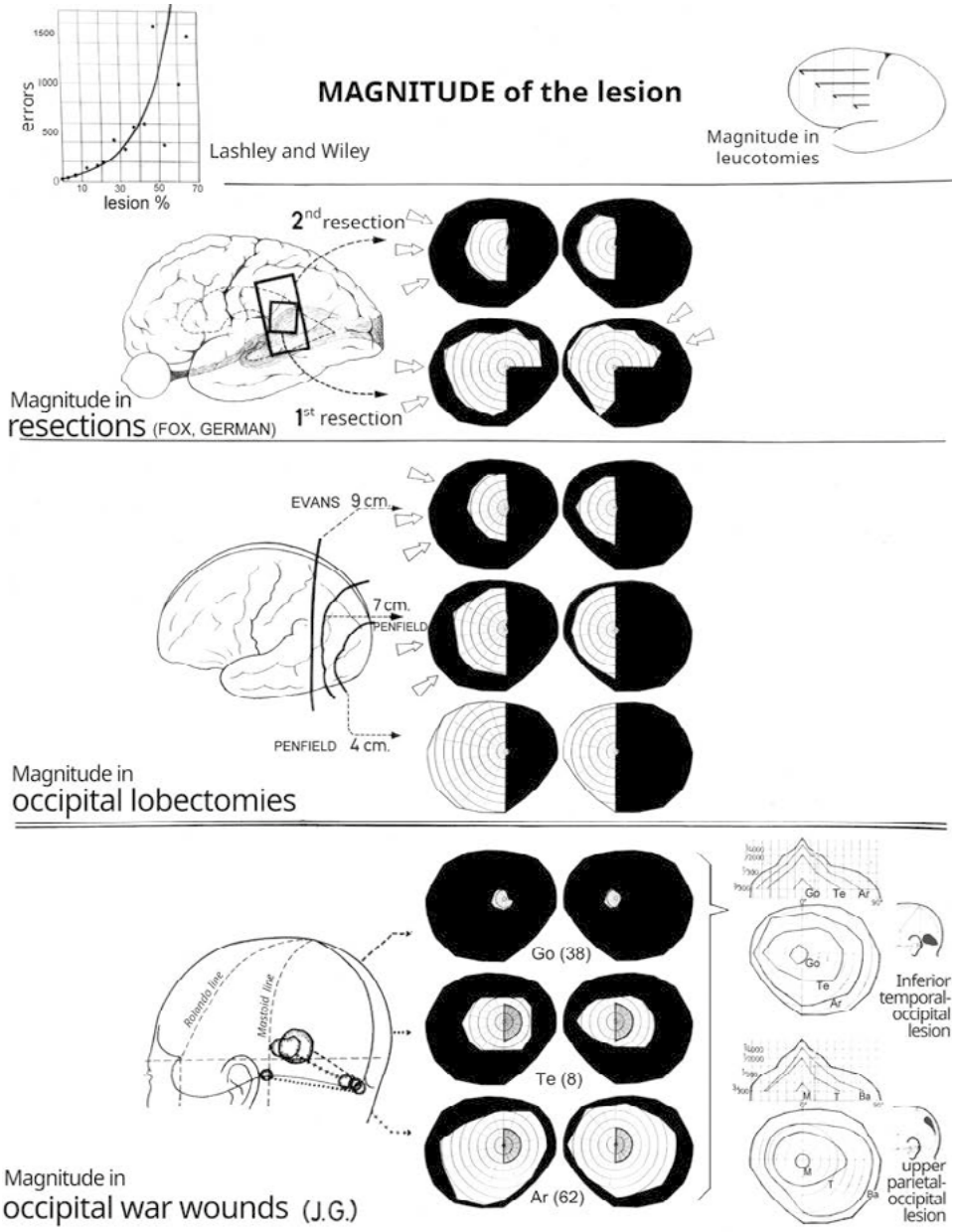

Figure 13

Figure 13. Effect of lesion magnitude on anomalies. At the top is an indication of the influence of the magnitude of *leucotomies* on the errors made by a rat [after Lashley and Wiley, *Biolog. Symposium*, (1950)].

The effect of the magnitude of the indicated *resections* on the visual fields is shown below [data from J. C. Fox and W. J. German, *Arch. Neurol. Psychiat.*, **35**, 808 (1936)]: For the larger resection (2nd resection), there is a greater reduction of the visual field.

In the *occipital lobectomies* shown (data from W. Penfield and J. Evans, 1932), for the smaller lobectomy (4 cm) the visual fields are affected only contralaterally, whereas for the 9 cm lobectomy there is a greater reduction of the visual fields with marked bilateral involvement.

Three cases from Gonzalo with similar *temporal-occipital war lesions* in the same area, with entry and exit orifices but with different degrees of lesion in the temporal orifice are shown. The case of greater lesion presents greater concentric reduction of the visual fields. It is noteworthy in these cases of *central* syndrome type, the great difference in symmetric concentric reduction of the visual field according to the magnitude of the lesion. For these three cases with *inferior temporal-occipital* lesion, the corresponding visual sensitivity profiles are shown on the right, which is also shown in the next Fig. 14. The lower right corner shows the visual sensitivity profiles corresponding to the concentrically reduced visual fields of three other central syndrome cases from Gonzalo (M, T, and Ba), also comparable to each other, with an *upper parietal-occipital* type of lesion, already shown in the first column of Fig. 10. The expressions 1/4000, 1/2000, 1/300, etc., (in general n/N), in the profiles, mean that the test is a white disk of n mm diameter at a distance of N mm from the observing eye.

The figure is in relation to Secs. 2, 7 and 8 of Supplement I.

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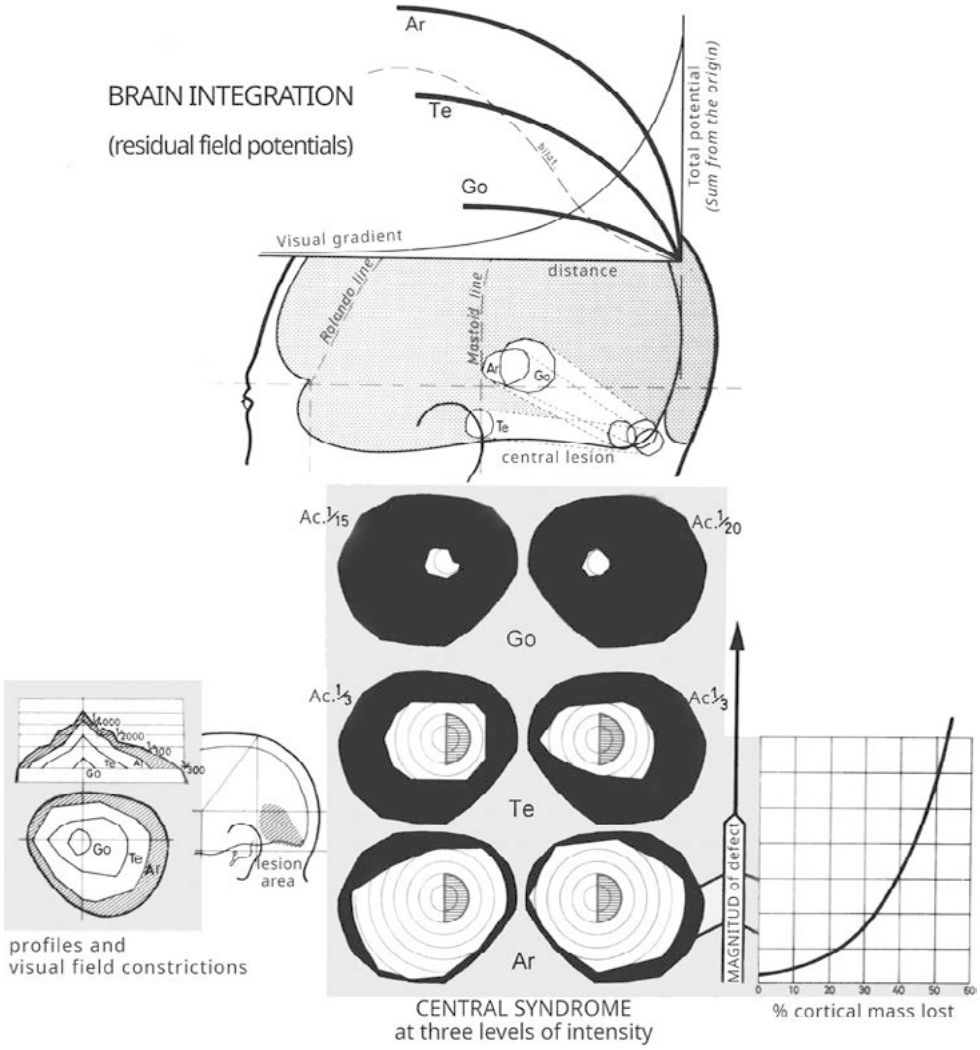

Figure 14

Figure 14. Brain integration (here called "field potential"). Three *central* syndrome cases described by Gonzalo are shown to illustrate incomplete brain integration ("residual field potential") according to the magnitude of the lesion. These cases, also shown in the previous Fig. 13, are easily comparable with each other with respect to the magnitude of their lesion. Bottom left, expressions of the type n/N in the sensitivity profiles mean that the test is a disk of n mm diameter at a distance of N mm from the observing eye.

In the upper part of the figure, each curve in thick line represents the value of the brain integration (or field potential) for each case. In a more formal way, each of these curves would be the sum or integration from the origin of coordinates of the thin curve representing the visual function density (visual gradient). If this function is incomplete due to the lesion, its integration from the origin is also incomplete. The maximum value of the integration is then no longer the normal value, but the "residual field potential." A greater lesion (Go case) leads to a lower integration (lower potential). All this would be applicable to any visual function.

The figure is related to Secs. 7 and 8 of Supplement I, to the articles referenced in footnote 2 in the Introduction of this Supplement II, and to the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

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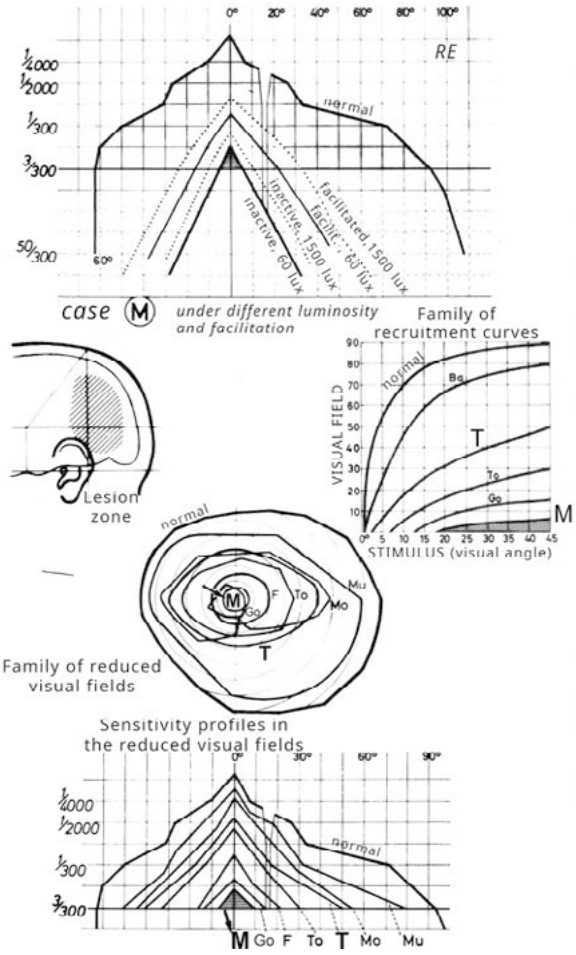

Figure 15. Similarity in the *central* syndrome. Top: Similarity between visual sensitivity profiles of patient M, in the inactive state, and under different conditions of light intensity and facilitation by muscular effort, compared to the normal case. Center and bottom: Similarity between the concentrically reduced visual fields and between the corresponding sensitivity profiles of several *central* syndromes (cases reported by Gonzalo including the most severe, M, and the intermediate T), compared to the normal case. Expressions such as 1/4000, 1/2000, 1/300, etc., (in general *n*/*N*), to the left of the profiles, mean that the test is a white disk of *n* mm diameter at a distance of *N* mm from the observing eye. The functional reduction maintains the same organizational plan as in the normal individual.

Right: Similarity between the different cases for the visual field recruitment curves as the visual stimulus increases with increasing subtended visual angle.

Figure in relation to Fig. 5 of Supplement I, to Secs. 3.5 and 14.2 in Vol. 1 of *Brain Dynamics*, to the articles referenced in footnote 2 in the Introduction of this Supplement II, and to the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

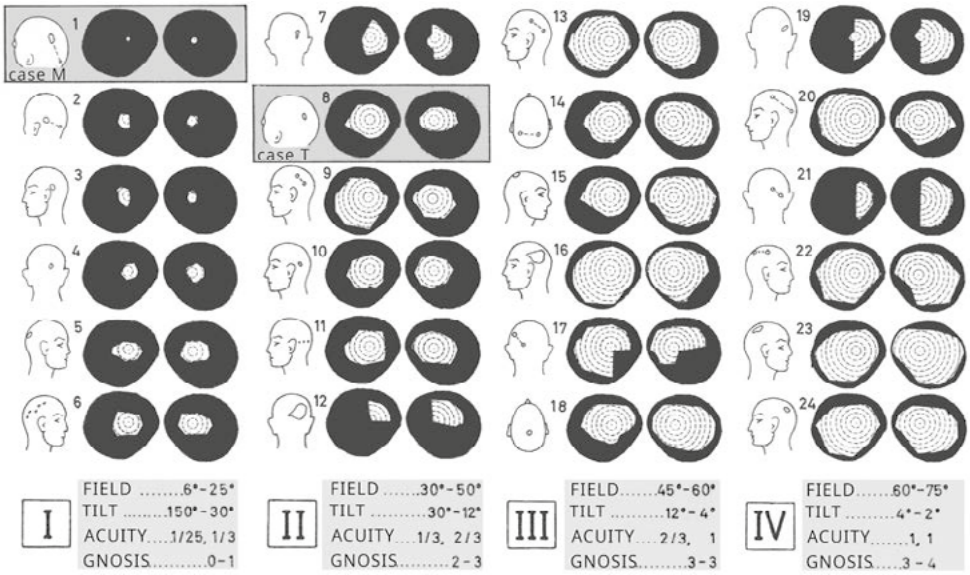

Figure 16. Twenty-four cases reported by Gonzalo classified into four groups (I, II, III and IV) according to the intensity of their permanent impairment in the functions indicated below. These functions are: Degrees of visual *field*, visual image *tilt*, *acuity*, and *gnosis*. All these cases, with *central* or *paracentral* syndrome, present visual image tilt. From column I (most affected group) to column IV (least affected group) the anomalies decrease in the four functions considered. Each case is numbered according to the order it occupies in the intensity of the anomalies, and is not the one that identifies the case (here omitted for simplicity). The M and T cases are highlighted.

The figure is related to Sec. 9 of Supplement I, to Vol. 1 of *Brain Dynamics*, and to the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

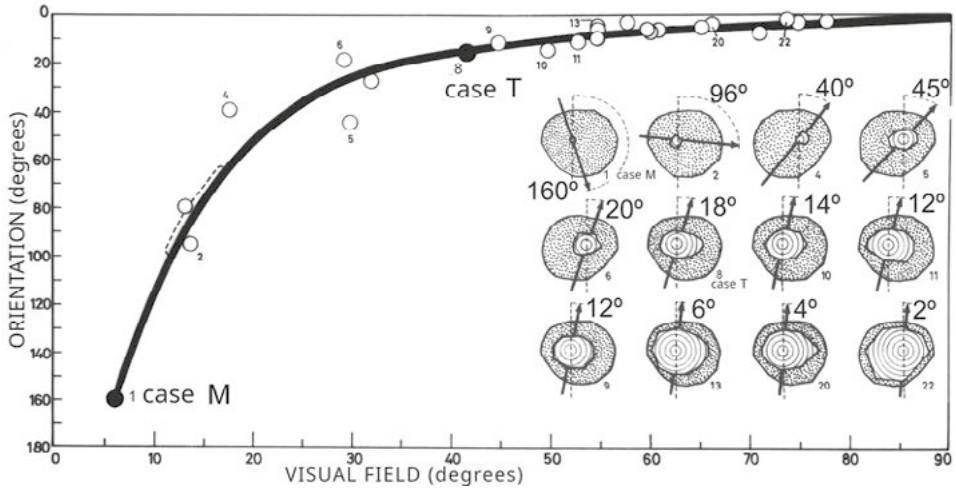

Figure 17. Allometry. Correlation between visual image orientation (tilt) and visual field size, in permanent cases under minimal stimulus. The twenty-four cases of the previous Fig. 16 are represented here according to the size (degrees of amplitude) of their visual field (horizontal axis) and the degrees of tilt of the visual image (vertical axis) of a test arrow pointing upwards. The small white circles represent the cases, highlighting the M and T cases with black circles. Patient M has almost inverted vision. For details of the M and T cases and experimental measurement conditions, see the book *Brain Dynamics* Vol. 1. Of the twenty-four cases, only those whose visual fields are shown together with an arrow indicating the tilt of the perceived visual image are numbered. They are cases of *central* syndrome.

In the figure, all image-arrows are rotated clockwise, although in the left visual fields they should be rotated counterclockwise, being central vision. However, in the way shown it is better appreciated how the absolute value of the rotated angle decreases as the size of the visual field increases.

The correlation found obeys a *potential-type allometry law* (see Fig. 19), as expected in the change of scale involved in a central syndrome with respect to the normal case.

Figure in connection with Sec. 9 of Supplement I, Secs. 12-14 in Vol. 1 of *Brain Dynamics,* the articles referenced in footnote 2 in the Introdu ction in the Introductonof this Supplement II, and the preceding *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

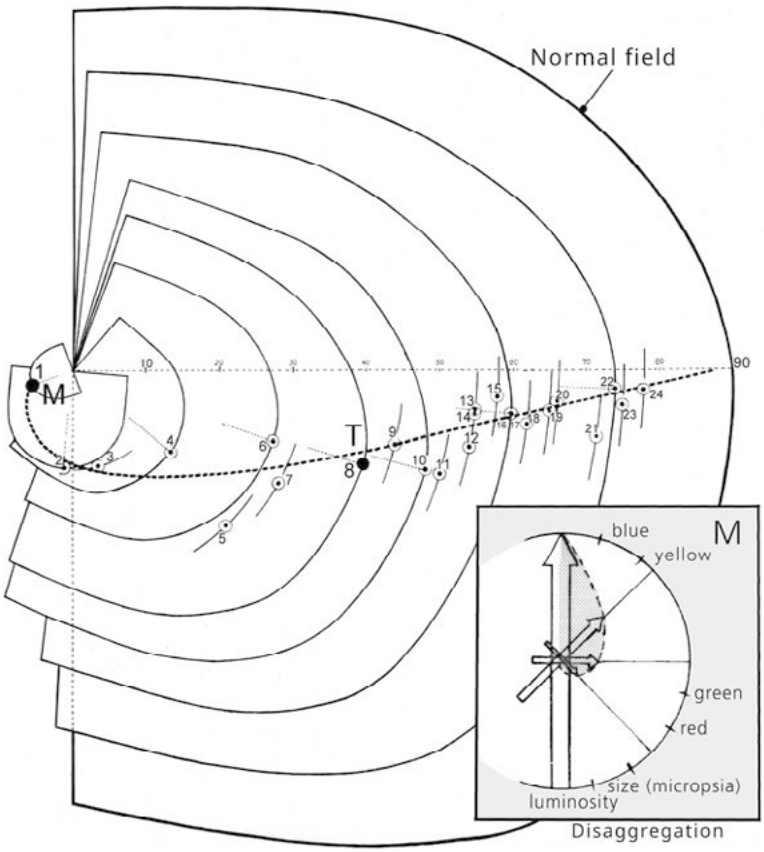

Figure 18. Spiral development of the visual field. The twenty-four cases of Figs. 16 and 17 are indicated by circled dots. The M and T cases are highlighted by small black circles. The position of each dot depends on the size of the corresponding visual field (in degrees along the horizontal axis), and on the tilt of its visual image (shown in Fig. 17). This tilt is indicated here by the angle rotated by the half of the visual field considered, with its error. The half visual field is small and almost inverted in the most pronounced pathological case, patient M, and becomes magnified and re-inverted as the cases are less severe, i.e., when the brain integration process is more complete. The dashed line through the points approximates a logarithmic spiral branch. The visual field would thus develop following a spiral development in the process of brain integration.

Bottom right: Different images perceived by patient M of a white test arrow pointing upwards, according to the intensity of the stimulus or on the facilitation by muscular effort. The various attributes of the arrow (some of which are indicated at the edge of the circle) are gradually lost (at the same time it decreases in size), as the intensity of the stimulus or facilitation decreases. The ends of the arrows also follow a logarithmic spiral. Figure in relation to Sec. 4 (Figs. 11-13) and Sec. 9 of Supplement I, Secs. 12-14 in Vol. 1 of *Brain Dynamics* and Sec. 26 in Vol. 2 of *Brain Dynamics*.

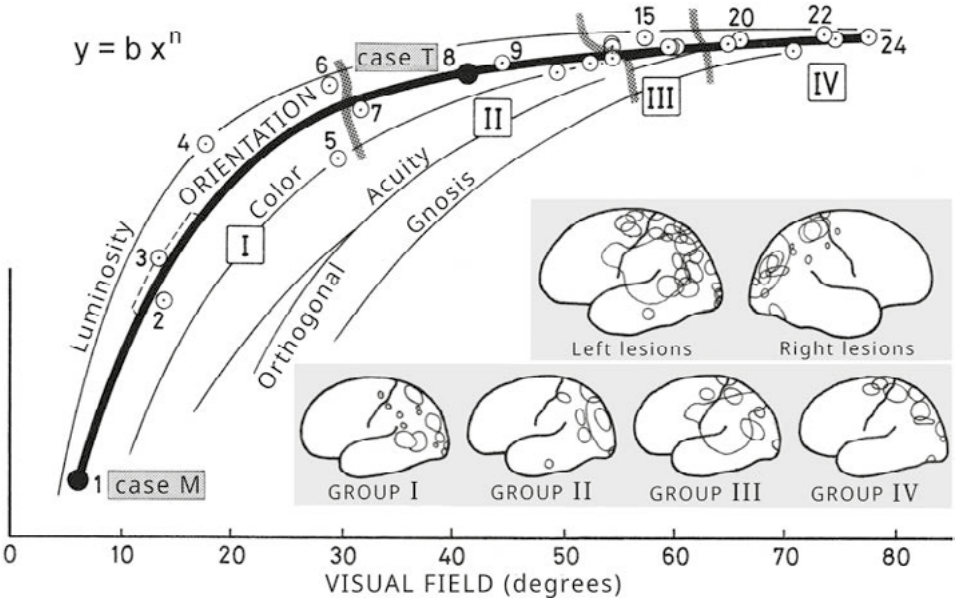

Figure 19. Allometric relationships of various functions with the size (amplitude degrees) of the visual field. The twenty-four cases of Figs. 16-18 are shown on the correlation curve between visual image orientation and visual field degrees, as in Fig. 16, but now indicating the distribution of cases in the 4 groups (I, II, III, IV) shown in Fig. 16. The most severe case, M, and the intermediate case, T, are highlighted. The other curves show qualitatively the correlations that may be established also for these cases between other visual functions (luminosity, color, acuity, orthogonal, gnosis) and the visual field. Regarding the orthogonal property, see Sec. 16 in Vol. 1 of *Brain Dynamics*.

These correlations are of the type $y = b\,x^n$, where x is here the size of the visual field, y is any of the visual functions considered, b is a constant, and n is the exponent (with positive value less than 1), different for each function, as can be seen from the data, and as expected in an allometric law.

The lesions indicated are in both the left and right hemispheres, as shown in the upper brains of the figure. However, to better compare the position of the lesions with respect to the occipital pole, all lesions are indicated in the left hemisphere in the four groups shown below.

Figure in connection with Sec. 9 of Supplement I, the articles referenced in footnote 2 in the Introduction of this Supplement II, and the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

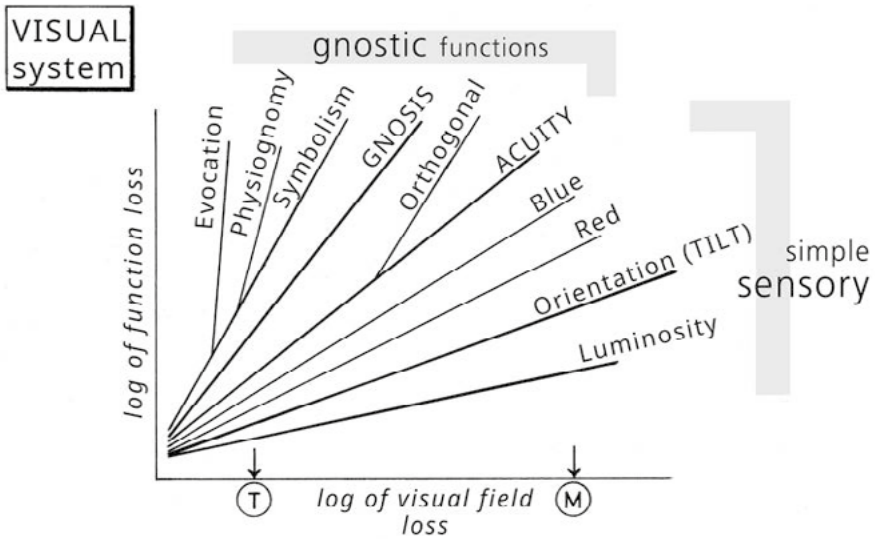

Figure 20. Allometry in the progressive loss of disaggregated functions. The correlations shown by the curves in the previous Fig. 19 are shown here as loss of the different functions versus visual field loss. Since the curves of Fig. 19 are of the potential type, when taking logarithm they become straight lines of different slopes according to the value of the exponent *n*, and taking in account that what is represented here are function *losses*, the arrangement of the straight lines is as shown schematically in the figure. We see in the figure that patient M with central syndrome, who has the most visual field loss, also has a greater loss of luminosity (for example) than patient T. The greatest losses are for the most complex functions (gnostic functions), then for acuity, a little less for blue color, even less for red, up to the simple luminosity which is the least lost. This loss of functions occurs in a constant sequence, both in the M case and in any other central syndrome case such as T. In the latter the losses are smaller, and the disaggregation of functions is much less pronounced, being practically nil in the normal case (origin of the coordinate axes). This unfolding or dispersion of functions is what is manifested in the dynamic phenomenon of functional asynchrony in the central syndrome when, as the intensity of the stimulus decreases, the functions are gradually lost according to the aforementioned sequence, which is called dynamic reduction. The M case can become closer in intensity to the T case by means of high intensity of the stimulus or by means of *facilitation* either by multisensoriality or by muscular effort (see for example Sec. 3 in Vol.1 of *Brain Dynamics*). Among the various visual functions is the orientation function, and its loss causes a tilted perception of objects. Other function is the orthogonal property (see Sec. 16 in Vol. 1 of *Brain Dynamics*).

The figure is closely related to the articles referenced in footnote 2 in the Introduction of this Supplement II, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

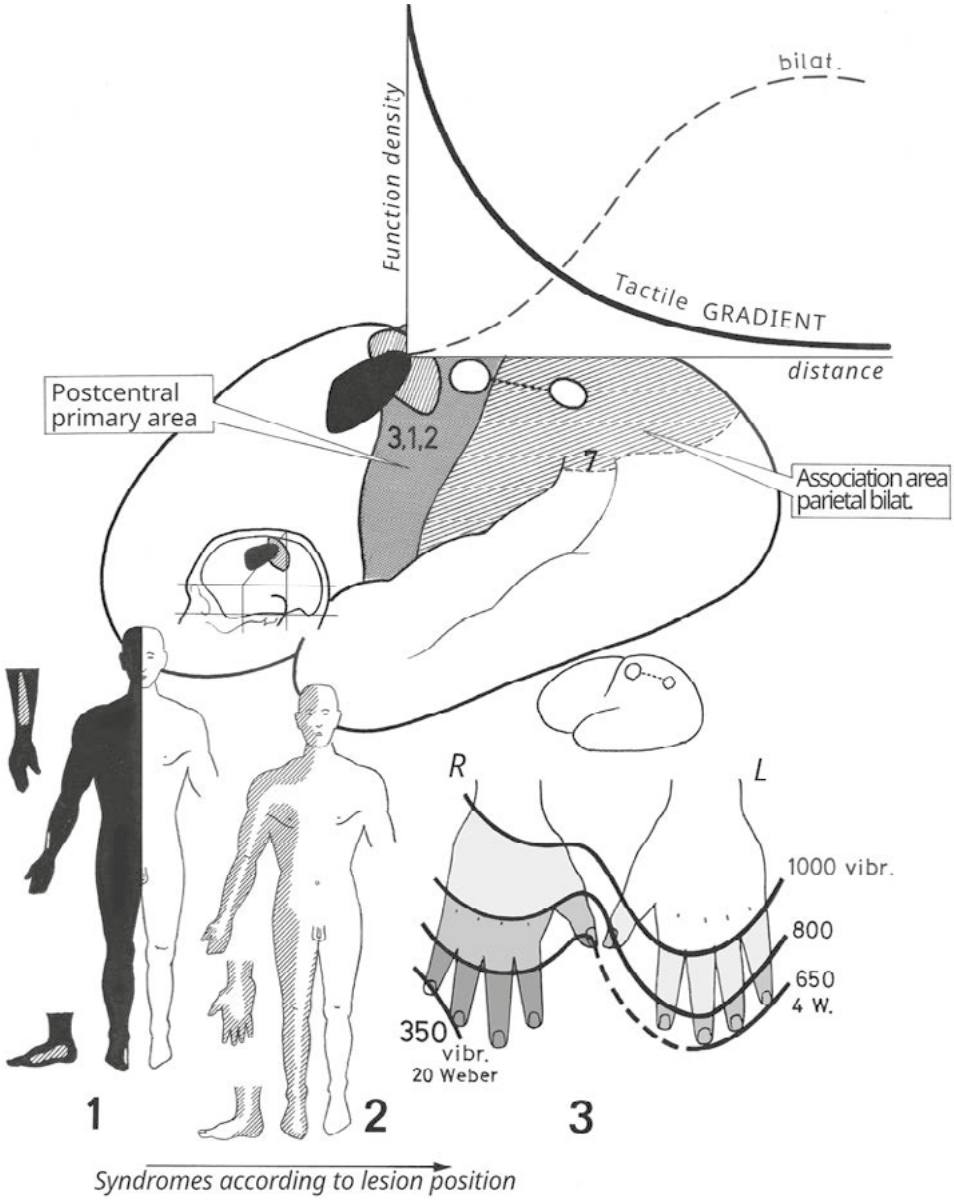

Figure 21

Figure 21. Tactile gradient. Three cases are shown (numbered below in the figure) corresponding to different syndromes according to the position of the lesion. The darkest parts of the body are the most severely affected.

In case **1** (case n.º 100 reported by Gonzalo), the lesion is indicated in black in the brain, in the primary tactile area (indicated by 3,1,2), where the tactile density function (tactile gradient) reaches its maximum value. The impairment in the right half of the body is almost total. Total avocalia is also present among other disorders.

In case **2** (case n.º 101 reported by Gonzalo), the lesion is indicated in gray in approximately the same area; but being of lesser magnitude, the anomaly is smaller, with distal predominance. He also presented semantic aphasia.

In case **3** (case n.º 109 reported by Gonzalo), the lesion is indicated in white, and involves a part of the association area. As a result, only the hands are affected in the tactile system, but now bilaterally, approximating the central syndrome type. However, the curves of equal vibratory sensitivity in the hands show the asymmetry of the disorder (*tactile paracentral* syndrome). Spatial discrimination in mm (Weber) is also indicated (undersized numbers).

In the upper part of the figure the density of tactile function in gradation across the cortex (tactile gradient) as well as the bilateral action curve are schematically represented. These representations are an abstraction of the observed syndromes, as in the visual system (e.g., Figs. 6, 9, 10).

This figure is in relation to Sec. 10 of Supplement I.

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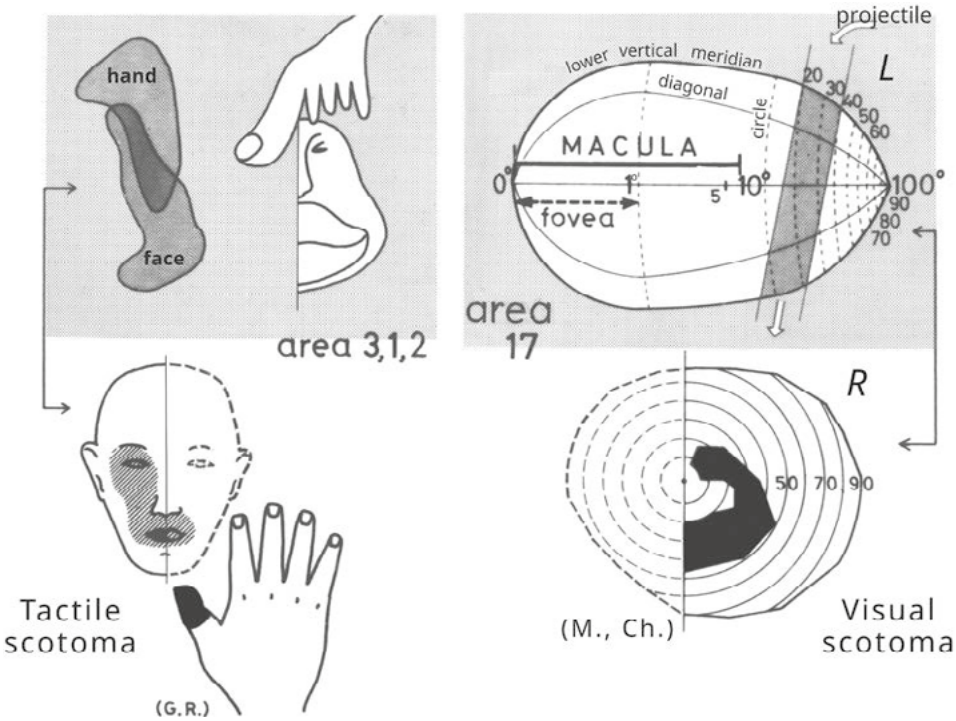

Figure 22. Tactile scotoma and visual scotoma. The dark parts are the affected parts.

Left: Tactile scotoma due to lesions in the projection areas of the face and hand, indicated above. Also shown are the hand and mouth of Penfield's homunculus showing their proportion of representation in the cerebral cortex.

Right: Visual scotoma due to bullet lesion indicated in the neural enlargement of area 17 [case reported by P. Marie and J. C. Chatelin, *Rev. Neurologique*, p 882 (1915)]. In this case, the macula is not damaged.

These scotomas do not exhibit the dynamic effects of anti-scotomas.

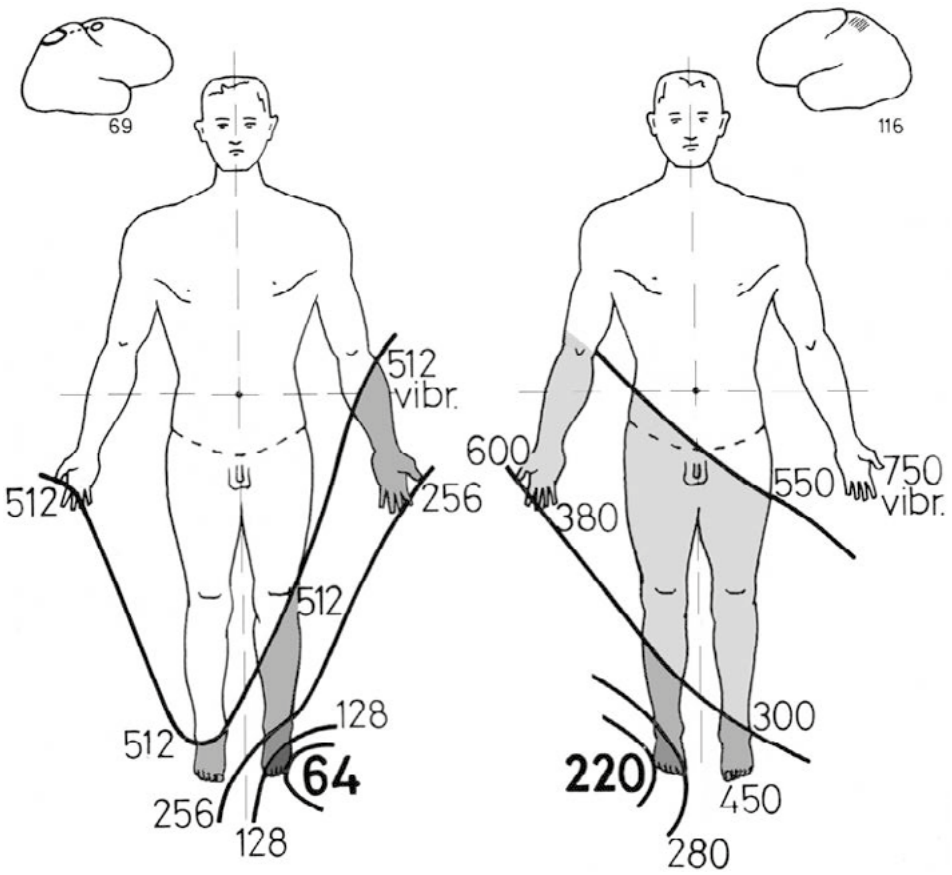

Figure 23. Tactile anti-scotomas. Two cases, n.º 69 and n.º 116, reported by Gonzalo with *tactile paracentral* syndrome, therefore showing anti-scotoma, i.e., the impairment increases progressively towards the periphery, and with tendency to bilaterality, presenting dynamic effects. The lines of equal vibration sensitivity show that the disorder increases towards the periphery and presents bilateral asymmetry. The respective lesions are schematically indicated above the figure.

Figure in relation to Sec. 10 of Supplement I (Figs. 22, 24), and to Sec. 18 in Vol. 2 of *Brain Dynamics*.

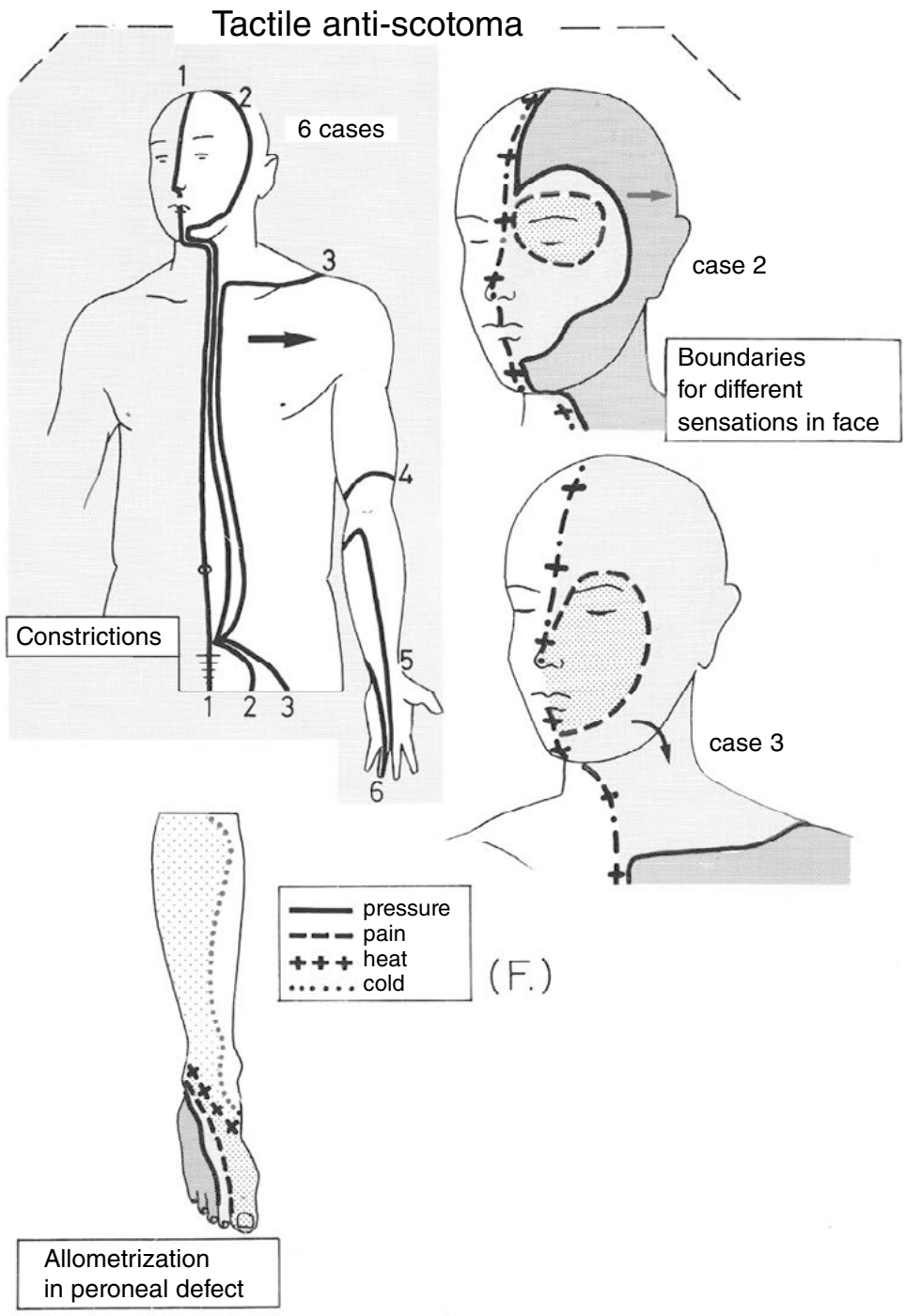

Figure 24

Figure 24. Allometric disaggregation in tactile anti-scotoma. Cases of *tactile paracentral* syndrome. The impairment increases towards the periphery, as indicated by the arrows.

On the left side, six cases (numbered 1 to 6) of decreasing intensity from 1 to 6 are shown together. The boundary line for pressure sensitivity is shown for each case.

The right side shows in detail the involvement of the facial region for cases 2 and 3. The boundaries for sensitivity to pressure, pain, heat and cold are indicated with different types of lines. In these cases, cold and heat have the same boundary [Ch. Foix, *Rev. Neurol.*, I, 322 (1922)].

Below left, the different boundaries for all the mentioned sensations can be clearly seen. This illustrates more clearly than in cases 2 and 3 the allometric disaggregation (separation) of the different types of sensations.

The figure is in relation to Secs. 19 and 20 in Vol. 2 of *Brain Dynamics*. Concerning allometry, the figure is in relation to the articles indicated in footnote 2 in the Introduction of this Supplement II, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

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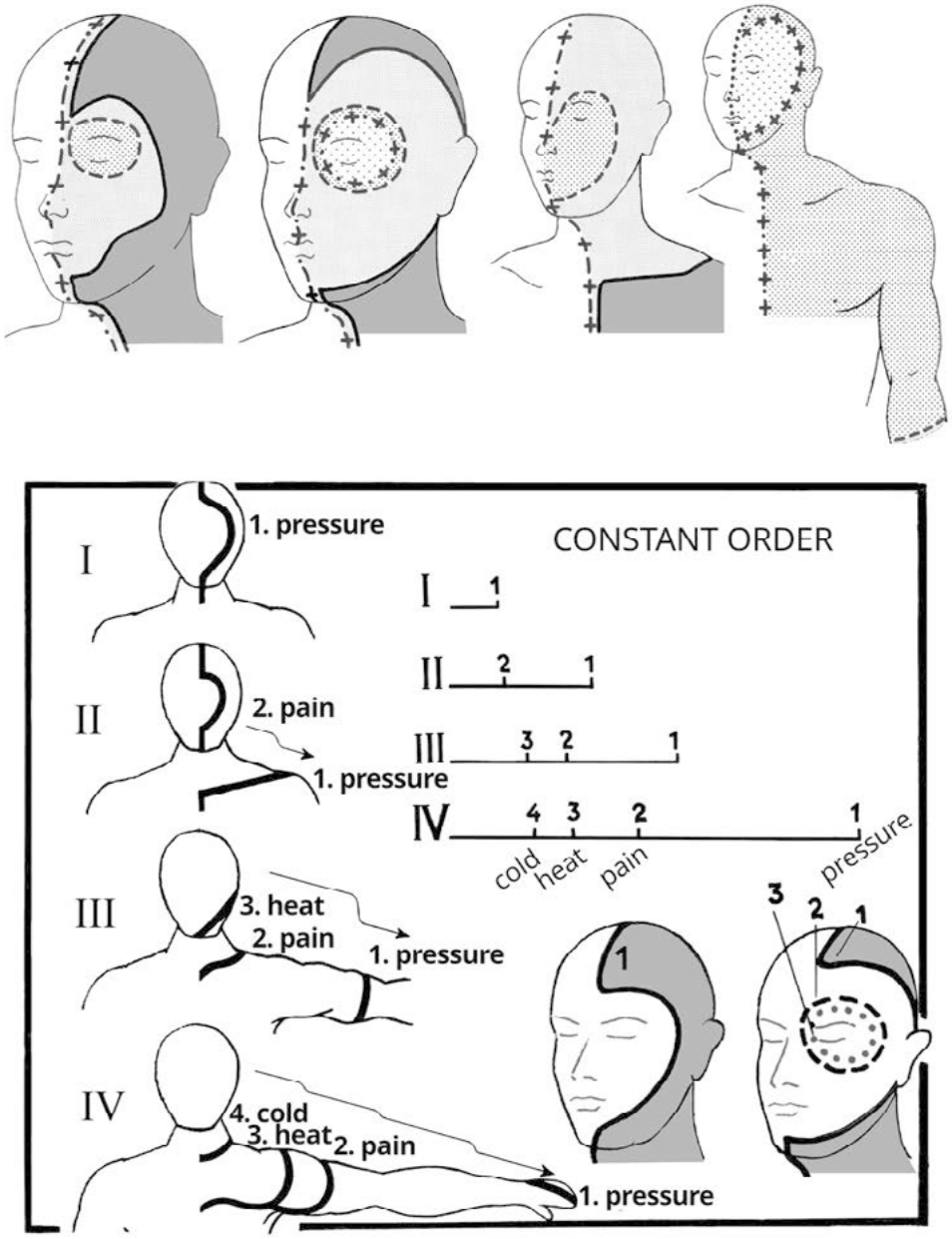

Figure 25

Figure 25. Constant order in loss of sensations. The sequence in which tactile sensations are lost in cases of different intensity of the disorder is shown.

Upper part: Four cases of *tactile paracentral* syndrome ordered by decreasing intensity of impairment from left to right. The meaning of the different line types is as shown in the previous Fig. 24. The first and third cases are those indicated as 2 and 3 respectively in Fig. 24.

Lower part: It shows the constant sequence in which the sensations of cold (4), heat (3), pain (2) and pressure (1) are lost in four cases of decreasing severity from I to IV. The most intense affectation is in the direction of the arrows, towards the periphery. The thick lines indicate the boundaries for the different sensations. The loss of the sensation of pressure in a zone (external to the line) implies the loss of the other sensations in that zone. The slightest disorder is to lose only the sensation of cold. The horizontal lines show the progressive distancing between the boundaries of the lost sensations (hot and cold lines are closer to each other than pressure and pain lines). The same sequence is shown in the facial cases in the lower right part of the figure.

The figure is in relation to Secs. 19 and 20 in Vol. 2 of *Brain Dynamics,* to the articles indicated in footnote 2 in the Introduction of this Supplement II, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics.*

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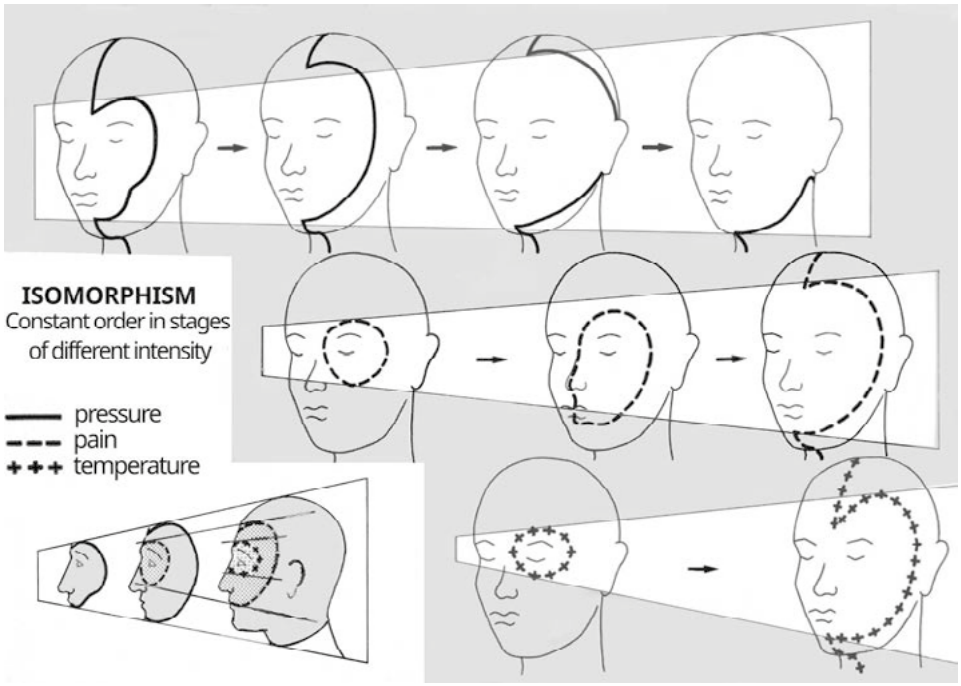

Figure 26. Isomorphism between disorders of the same type but of different intensity. Comparison between cases in which the intensity of the disorder decreases from left to right. It shows how the area unaffected by the loss of a given sensation increases. At the top, pressure sensation; in the middle, pain sensation; and at the bottom, temperature sensation. The lines marking the boundaries of sensation loss maintain approximately the same shape. The behavior is similar for each type of sensation. This shape maintenance is analogous to that occurring, for example, in the isopters of the visual fields in the *central* and *visual paracentral* syndromes shown in Figs. 10, 11, and also in the *central* syndromes shown for example in Fig. 15.

Figure in relation to Secs. 19 and 20 in Vol. 2 of *Brain Dynamics*, to the articles referenced in footnote 2 in the Introduction of this Supplement II, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

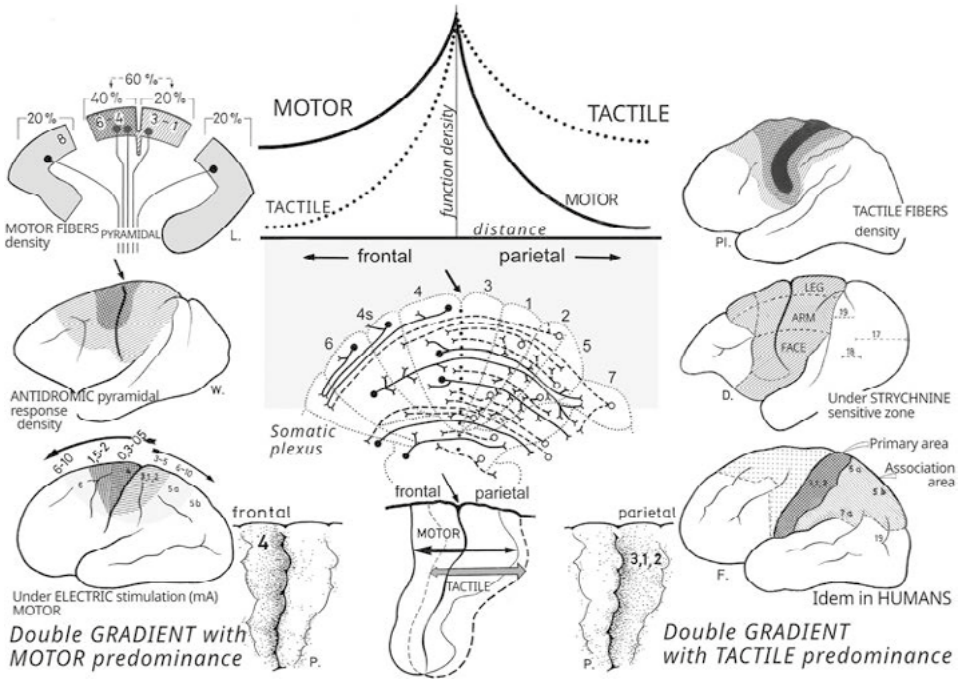

Figure 27. Double tactile and motor gradients. On the right and left, various known cortical maps, motor (on the left), tactile (on the right). Top center: Schematic of the densities of tactile function (dotted curves) and motor function (solid curves), in gradation along the cortex. On the right side of the graph, tactile and motor gradients are shown with predominance of tactile function. On the left side, tactile and motor gradients are also shown but with predominance of motor function.

This figure is related to Sec. 7 of Supplement I, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

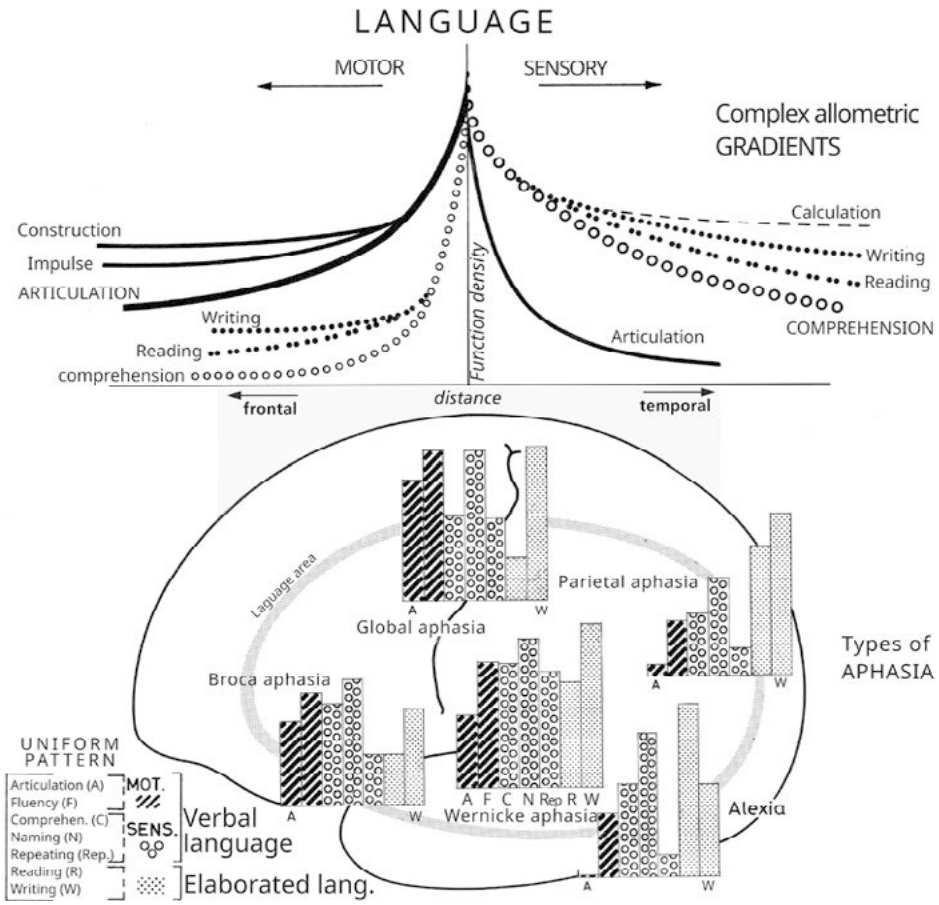

Figure 28. Cortical gradients in language. Lower part: The intensity of the types of aphasia according to the different language properties is indicated by histograms in the language area based on 214 observations (H. Hécaen and R. Angelergues, *Pathologie du langage,* Paris: Larousses 1965). The histograms indicate the intensity of impairment in articulation (A), fluency (F), comprehension (C), naming (N), repeating (Rep.), reading (R) and writing (W). The striped histograms refer to motor aphasia, those with small circles refer to sensory aphasia, and those with dots refer to reading and writing disorder.

Based on this distribution and also on some thirty of his own cases among the 117 numbered cases, Gonzalo proposes a scheme of gradients that extends towards the frontal and temporal areas, as shown in the upper part of the figure. Since the densities of the different functions have different slopes, the author calls them allometric gradients. In the lower part of the figure, the author delimits a wide language area by a thick gray line.

Figure in relation to Sec. 7 of Supplement I, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

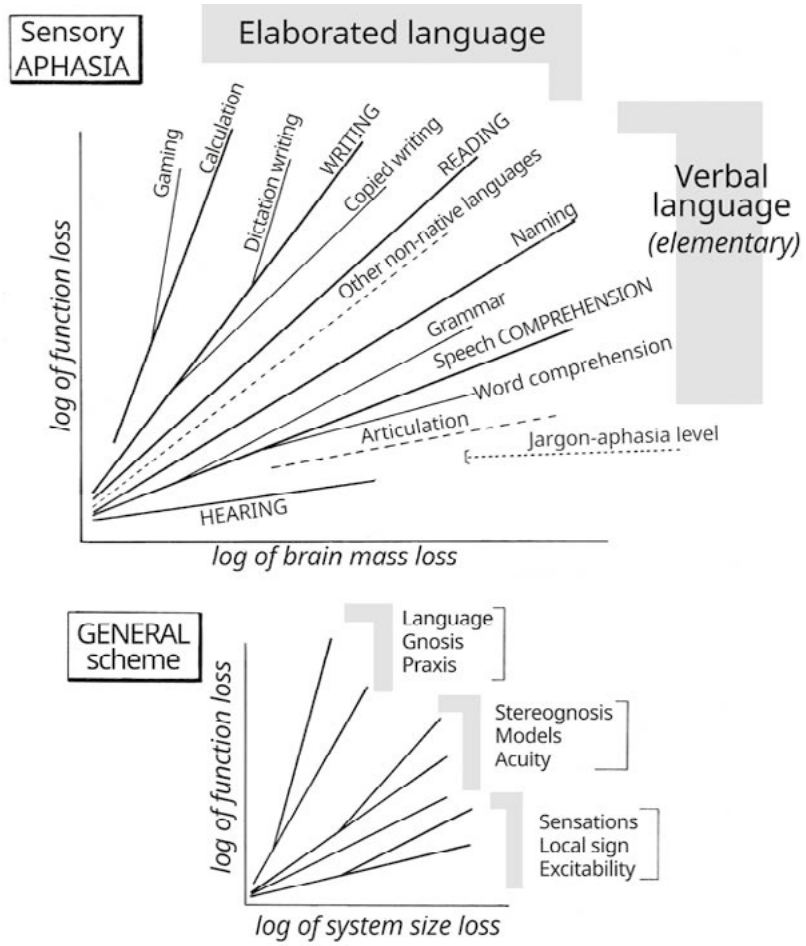

Figure 29. Allometry in sensory aphasia, and in general. Upper part: Analogous to Fig. 20 with respect to the visual system, here the logarithm of loss of function in sensory aphasia is qualitatively plotted versus the logarithm of brain mass loss in "central" type lesions causing central syndrome. The different straight lines come from different relationships giving rise to different (allometric) losses for each function. What is said in Figs. 19 and 20 regarding the graphical representation applies here as well. The straight lines with greater slope correspond to more complex functions, with greater excitability demand, and are the first to be lost.

Lower part: Schematic general diagram for the loss of the different functions in relation to the loss of size of the brain system due to central lesion in central syndromes. This diagram formalizes and explains the dynamic allometric disaggregation (dispersion) of functions in the central syndrome as lesion magnitude increases or stimulus decreases.

Figure in relation to the articles indicated in footnote 2 in the Introduction of this Supplement II, and to the *Note on Gradients, Similarity and Allometry in Brain Dynamics*.

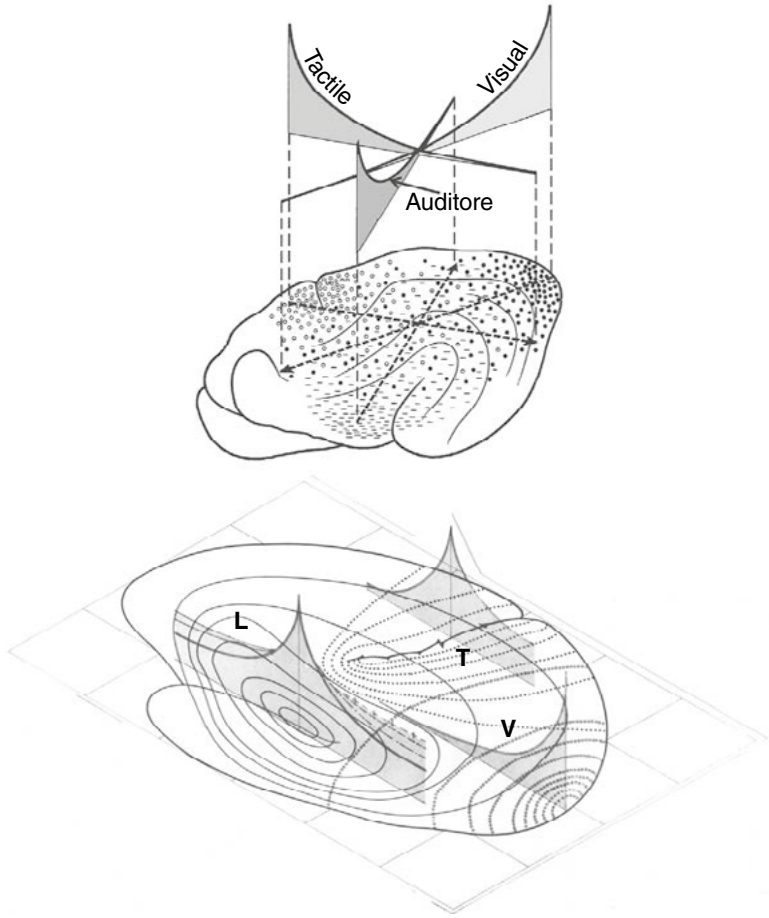

Figure 30. Schematic diagrams of cortical gradients. Upper part: Schematic of the densities of the visual, tactile and auditory functions in gradation on a dog brain. The figure of the dog brain is a modified composition of the figures from the pioneer authors L. Luciani and G. Seppilli (*Le localizzazioni funzionali del cervello,* Napoli, Italia: L. Vallardi 1885, p. 362), where the densities of the small marks represent the importance of the areas for vision (dots), touch (small circles) and hearing (dashes), according to their experiments with dogs. Lower part: Simplified scheme of gradients for language (L), touch (T) and vision (V) on a human brain, with corresponding lines of equal function density. These diagrams simply try to illustrate the idea of *continuity and heterogeneity* of the cortex, as well as the multisensory character of each point to a different degree.

Figure in relation to Sec. 7 of Supplement I, to the *Note on Gradients, Similarity and Allometry in Brain Dynamics,* and to the articles referenced in footnote 2 in the Introduction of this Supplement II. The upper part of this figure is adapted from Fig. 5 of the article in *Neurocomputing* by Gonzalo-Fonrodona I. (2009) (referenced in the Mentioned footnote 2), with permission of Elsevier.

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Justo Gonzalo

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Supplement I is the 1952 publication in which the author reports additional cases, and proposes, for the first time, that the specificity of a human brain function is distributed in gradation across the cortex, resulting in brain gradients and a unitary conception of the cortex. Supplement II deals with the concepts of dynamic similarity and allometric scaling power laws applied to the central syndrome, and shows later illustrations by the author.

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