THE PIONEERING RESEARCH OF JUSTO GONZALO (1910–1986)

ON BRAIN DYNAMICS

Isabel Gonzalo Fonrodona, Departamento de Óptica, Facultad de Ciencias Físicas
Universidad Complutense de Madrid, 28040-Madrid, Spain. igonzalo@fis.ucm.es

June 2015, revised May 2022

Abstract

This work is the English translation of an extract from the pioneering research of Justo Gonzalo (1910–1986) on brain dynamics. This is an article published in Spanish in the scientific journal 'Trabajos del Instituto Cajal de Investigaciones Biológicas', Vol. XLIV, pp. 95-157 (1952), and entitled *Human brain functions according to new data and physiological basis. An introduction to the studies conducted on brain dynamics* in its English version. As his author pointed out there, the article is a brief summary of the extensive preceding work 'Dinámica Cerebral´, Volume 1 (1945) (English translation, Open Access at: https://eprints.ucm.es/id/eprint/63730/), Volume 2 (1950) (English translation, Open Access at: https://eprints.ucm.es/id/eprint/72118/), and also includes original work performed later (from Sec. 7 of the article), as a cortical gradients model. From the study of patients with unilateral lesion in the parieto-occipital cortex, J. Gonzalo characterized what he called the *central syndrome of the cortex*, a multisensory, bilateral and symmetric affection presenting dynamic phenomena dependent on the intensity of the stimulus, such as the separation of sensory qualities united in normal perception, noticeable facilitation by motor and cross-modal effects, and tilted or inverted perception, among other disorders. He interpreted these phenomena under a dynamic physiological concept, and from a model based on functional gradients through the cortex and scaling laws of dynamical systems, thus highlighting the functional unity of the cortex and offering a dynamic solution to the traditional theory of brain function localization. In the 2000s, phenomena (related to multisensory integration, inverted vision) have been reported and models have been proposed in close relation to this research. In particular, brain gradients are a hot topic. Therefore, this research is of relevant current interest. A preface introduces some aspects of the translated article, its author and his research, including several references. It is followed by the translated article.

Key words: Brain dynamics, Asynchrony, Neurophysiology, Excitability, Multisensory, Crossmodal, Facilitation, Inverted vision, Tilted vision, Inverted perception, Visual perception, Tactile perception, Brain injury, Central and paracentral syndromes, Cortical lesions, Parieto-occipital cortex, Plasticity, Sensory field, Cortical gradients, Functional gradients, Brain gradients.

PREFACE

The present work is an English translation of a small but substantial part of the research of Justo Gonzalo (1910–1986), first published in Spanish [1] and included as 'Suplemento I' in the facsimile edition in Spanish published in 2010 "Dinamica Cerebral" [2]. The article here translated is a brief summary of the extensive preceding works (Vol. 1 [3] and Vol.2 [4] of the book `Dinámca Cerebral´ [3, 4, 2], which are now translated into English) to which the author makes frequent reference, and also includes original work performed later [5]. In particular, the proposed model of cortical gradients is presented in the article here translated. A reduced and modified version of the present translation was distributed in the Workshop on *Alpha Processes in the Brain*, in 1994 in Lübeck (Germany).

Justo Gonzalo Rodríguez-Leal (Barcelona 1910 – Madrid 1986) was a Spanish neuroscientist. After finishing his degree in medicine, he specialized in neurology at the University of Vienna (1933-34) with H. Hoff and O. Pötzl, and in Frankfurt (1934-35) with K. Kleist. During the Spanish Civil War (1936-39), he studied a large number of brain injured patients and then described what he called dynamic action phenomena. From 1941 he was a full-time researcher exploring patients with brain lesions and holding PhD courses on Physiopathology of the Brain at the University of Madrid. He belonged to the 'Instituto Cajal' and to the Official Research Council, also in Madrid. Data about the author and his research are in: http://en.wikipedia.org/wiki/Justo%20Gonzalo

From the study of patients with unilateral lesion in the parieto-occipital cortex, he characterized what he called the *central syndrome of the cortex*. This syndrome, with multisensory, bilateral and symmetric affection, presented dynamic phenomena dependent on the stimulus intensity, such as the separation of sensory qualities (united in the normal perception), noticeable facilitation by motor and cross-modal effects, and tilted or inverted perception, among other disorders. The pathological sensory stages in perception can be synchronized by facilitation, whose first detailed study was carried out in this research. In this syndrome a strong muscular contraction, for example, can improve the perception in any sensory system. In particular, dynamic phenomena were manifested in the process of inverted perception, whose first deep experimental study and interpretation also belongs to this research. The author interpreted these phenomena under a dynamic physiological concept, and from a model based on functional gradients through the cortex and scaling laws of dynamical systems, thus highlighting the functional unity of the cortex and offering a dynamic solution to the traditional localization of brain functions. He developed the concepts of similarity and allometry based on the biological principles of development and growth, and extended the model to the auditory system and language. This extension is summarized and published for the first time in 'Suplemento II' of the edition of 2010 [2].

The details about data and methods that firmly support what is established in this article are extensively presented in the mentioned book `Dinámica Cerebral´, in which Vol. 1 [3, 2] covers general aspects and the systematic study of visual functions, whereas Vol. 2 [4, 2] is devoted to tactile functions and to expand on the principles introduced in Vol. 1.

The first edition of that book had a significant reception by the international neurologist community at that time. To our best knowledge, this is the first time that the term 'brain dynamics' ('dinámica cerebral') was used to describe brain mechanisms in relation to sensory organization. In the 2000' s decade, some phenomena have been reported which are similar to those that Gonzalo had described, related to tilted or inverted vision, multisensory interactions, cross-modal, and

multisensory integration. Also, modelling of the cortex has been proposed that are closely related to Gonzalo's model. In particular, cortical gradients model is a very active topic nowadays. Some works deal with this research in a more current context [6-9]. It is noteworthy that this research has aroused special interest in the field of Artificial Intelligence (see [10] and references therein).

As is usual in texts written many decades ago, some aspects of the original title, contents, and the way of expressing it, reflects the context of that time. The original work whose translation we present here does not contain references, the author making always reference to Vol. 1 and Vol. 2 of the book "Brain Dynamics" in which the author refers to 350 works of international specialized literature. Let us mention here just a few, such as the animal experimentation by Flourens [11], the isochronisme of Lapicque [12], physiological activity of the cerebral cortex by Pavlov [13], the theory of Jackson [14] on the unity and functional levels of the brain, the diaschisis phenomena of Monakow [15], the mass action of Lashsley [16], precedents on visual agnosia [17- 24], in particular the much-discussed Schneider case of Goldstein and Gelb [18], other works on brain injuries and brain pathology [25], nerve crossings [26], brain organization [27], apraxia [28], pathology of the sensitivity and sensory perceptions [29], body perception (e.g. [30]), aphasia [31], gestalt theory and psychophysical isomorphism [32], etc.

Acknowledgements: I acknowledge the permission of the publisher of the first edition (1952) ['Consejo Superior de Investigaciones Científicas' (CSIC) (The Spanish National Research Council)], and of the facsimile Spanish edition of 2010 (RTNAC and University of Santiago de Compostela). I am also very grateful for the continuous help and advice of Miguel A. Porras.

References

[1] 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**: pp. 95–157. Included in the facsimil edition in Spanish (see [2]) as 'Suplemento I'. English translation in this document.

[2] Gonzalo J. (2010), "Dinámica Cerebral". Edición facsímil con suplementos. Volumen 1 (1945), Volumen 2 (1950), Artículo 1952 (Suplemento I), Suplemento II (1ª ed.), Santiago de Compostela: Red Temática en Tecnologías de Computación Artificial/Natural (RTNAC) y Universidad de Santiago de Compostela (USC) 2010. ISBN 978-84-9887-458-7. (Spanish edition).
Open Access at: http://hdl.handle.net/10347/4341

[3] Gonzalo J. (1945), "Dinámica Cerebral. La actividad cerebral en función de las condiciones dinámicas de la excitabilidad nerviosa". Volumen 1: pp. 1–392. Madrid: Consejo Superior de Investigaciones Científicas, Instituto S. Ramón y Cajal, 1945. (Included in the facsimile edition in Spanish [2]). English translation, Open Access at: https://eprints.ucm.es/id/eprint/63730/

[4] Gonzalo J. (1950), "Dinámica Cerebral. La actividad cerebral en función de las condiciones dinámicas de la excitabilidad nerviosa". Volumen 2: pp. 393–827. Madrid: Consejo Superior de Investigaciones Científicas, Instituto S. Ramón y Cajal, 1950. (Included in the facsimile edition in Spanish [2]). English translation, Open Access at: https://eprints.ucm.es/id/eprint/72118/

[5] Gonzalo, J. (1951). *La cerebración sensorial y el desarrollo espiral*. Trabajos del Inst. Cajal de Investigaciones Biológicas, **XLIII**: pp. 209–260.

[6] Gonzalo, I. (1997). *Allometry in the J. Gonzalo's model of the sensorial cortex.* Lecture Notes in Computer. Science. (LNCS) **1240**: 169, OA: https://doi.org/10.1007/BFb0032475 . Gonzalo, I

(1999). *Spatial Inversion and Facilitation in the J. Gonzalo's Research of the Sensorial Cortex. Integrative Aspects*. Lect. Not. Comp. Sci. (LNCS) **1606**: 94103, OA: https://doi.org/10.1007/BFb0098164 . Gonzalo, I.; Porras, M.A. (2001). *Time-dispersive effects in the J. Gonzalo's research on cerebral dynamics*. Lect. Not. Comp. Sci. (LNCS) **2084**: 150, OA: https://doi.org/10.1007/3-540-45720-8_18 . Gonzalo, I.; Porras, M.A. (2003). *Intersensorial summation as a nonlinear contribution to cerebral excitation*. Lect. Not. Comp. Sci. (LNCS) **2686**: 94, OA: https://doi.org/10.1007/3-540-44868-3_13 . Gonzalo, I.; Porras, M.A. (2007). *Physiological Laws of Sensory Visual System in Relation to Scaling Power Laws in Biological Neural Networks.* Lect. Not. Comp. Sci. (LNTCS) **4527:** 96, OA: https://doi.org/10.1007/978-3-540-73053-8_10

[7] Gonzalo I. (2007), *Inverted or tilted perception disorder*. Rev. Neurol. **44**(3): 157: https://www.researchgate.net/publication/6518309_Inverted_or_tilted_perception_disorder . Gonzalo I. (2009), *Functional gradients through the cortex, multisensory integration and scaling laws in brain dynamics*. Neurocomputing **72**: 831, OA: https://doi.org/10.1016/j.neucom.2008.04.055

[8] Gonzalo, I.; Porras, M.A. (2009). *Scaling Power Laws in the Restoration of Perception with Increasing Stimulus in Deficitary Natural Neural Network*. Lect. Not. Comp. Sci. (LNTCS) **5601**: 174, OA: https://doi.org/10.1007/978-3-642-02264-7_19 **.** Gonzalo, I.; Porras, M.A. (2011). *Scaling Effects in Crossmodal Improvement of Visual Perception.* Lect. Not. Comp. Sci. (LNTCS) **6687**: 267, OA: https://doi.org/10.1007/978-3-642-21326-7_29 . Gonzalo, I.; Porras, M.A. (2013). *Scaling effects in crossmodal improvement of visual perception by motor system stimulus*. Neurocomputing **114**: 76, OA: https://doi.org/10.1016/j.neucom.2012.06.047 . Gonzalo I.; Porras, M.A. (2013). *Deficitary nervous excitability and subjective contraction of time: Time-dispersive model*. Lect. Not. Comp. Sci. (LNTCS) **7930**: 368, OA: https://doi.org/10.1007/978-3-642-38637-4_38

[9] Gonzalo I., Porras M.A. (2014), *Nervous excitability dynamics in a multisensory syndrome and its similitude with a normal state. Scaling Laws*. In: Costa, A.; Villalba, E. (Eds.) Horizons in Neuroscience Vol. 13: Chap.10, pp. 161–189. OA at: https://doi.org/10.48550/arXiv.2006.01666 and also at: https://novapublishers.com/wp-content/uploads/2019/06/978-1-62948-426-6_ch10.pdf

[10] Mira J., Delgado A.E. (2003), *Neural modeling in cerebral dynamics.* BioSystems 71:133, OA: https://doi.org/10.1016/S0303-2647(03)00121-7 .

[11] Flourens M.J.P. (1824), Recherches Experimentales sur les Propiétés et les Fonctions du Système Nerveux dans les Animaux Vértebrés. Paris: Chevol 1924.

[12] Lapicque L. (1926), *L'excitabilité en function du temps*. Press Universitaires, Paris 1926. Lapicque L. (1937), *L'isochronisme comme condition intercellulaire de l'excitation*. Compt. Rend., **205**: 530–534.

[13] Pavlov I. P. (1927), Conditioned Reflexes: An Investigation of the Physiological Activity of the Cerebral Cortex. Anrep G. V. (Transl. and Ed.). London: Oxford Univ. Press 1927.

[14] Jackson, J. H. (1874), On the nature of the duality of the brain, Medical Press and Circular 1, 19, 41-63, London 1874. Jackson, J. H. (1874), *The Croonian lectures on evolution and dissolution of the nervous system*. Br. Med. J., **1**: 591–593, 660–663, 703–707.

[15] Monakow C. von (1914), Die lokalisation im Grosshirn und der Abbau der Funktion durch kortikale Herde. Wiesbaden: Bergmann 1914.

[16] Lashley K.S. (1929), Brain mechanisms and Intelligence: A Quantitative Study of Injuries to the Brain. Chicago, Illinois: The Univ. Chicago Press 1929. Lashley K.S. (1937), *Functional determinants of localization*. Archives of Neurology and Psychiatry, **38**: 371–387.

[17] Lissauer H. (1890), *Ein Fall von Seelenblindheit nebst einem Beitrag zur Theorie derselben*. Archiv für Psychiatrie und Nervenkrankheiten, **21**: 222–270. Poppelreuter W. (1923), Zur Psychologie und Pathologie der optischen Wahrnehmung. Zeitschrift für die gesamte Neurologie und Psychiatrie, **83**: 26-152.

[18] Goldstein K., Gelb A. (1918), *Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzer. I. Abhandlung: Zur Psychologie des optischen Wahrnehmungs und Erkennungsvorganges*. Zeitschrift für die gesamte Neurologie und Psychiatrie, **41**: 1–142. Goldstein K., Gelb A. (1919), *Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter. II. Abhandlung: Über den Einfluss des vollständigen Verlustes des optischen Vorstellungs vermögens auf das taktile Erkennen. Zugleich ein Beitrag zur Psychologie der taktilen Raumwahrnehmung und der Bewegungsvorstellungen*. Zeitschrift für Psychologie, **83**: 157–250.

[19] Gelb A. (1920), *Über den Wegfall der Wahrnehmung von Oberflächenfarben*. Zeitschrift für Psychologie, **84**: 193–258.

[20] Gelb A., Goldstein K. (1922), *Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter: VII. Über Gesichtsfeldbefunde bei abnormer "Ermüdbarkeit" des Auges (sog. "Ringskotome")*. Albrecht von Graefes Archiv für Ophthalmologie. (Graefe's Archive for Clinical and Experimental Ophthalmology), **109**: 387–403.

[21] Goldstein K. (1942), Brain Injuries in War. London: Heinemann, 1942.

[22] Piéron H. (1923), Physiologie de la vision. Paris 1923. Piéron H. (1934), *Le problème du mécanisme physologique impliqué par l' échelon differential de sensation*. Année Psychologique, **34**: 217.

[23] Economo C. von, Koskinas G. N. (1925), Die Cytoarchitektonik der Hirnrinde des erwachsenen Menschen. Wien: Springer Verlag 1925.

[24] Pötzl O. (1928), *Die optish-agnostichen Störungen* in: Handbuch der Psychiatrie, Aschaffenburg, Wien 1928. Pötzl O. (1933), *Polyopie und gnostische Störung. Ein Beitrag zur Pathophysiologie der cerebralen Sehstörungen*. Jb. Psychiat. Neurol., **50**: 57–77.

[25] Inouye T. (1909), Die Sehstörungen bei Schuβverletzunge der kortikalen Sehsphëre. Nach Beobachtungen an Verwundeten der letzten japanischen Kriege. Leipzig: Engelman 1909. Cameron N (1928), *Cerebral destruction in its relation to maze learning*. Psychol. Monogr., **39** (1): 1-68. Kleist K. von (1934), Gehirnpathologie. Leipzig: Barth J. 1934.

[26] Ramón y Cajal S. (1899), Textura del Sistema Nervioso del Hombre y de los Vertebrados. Tomo II, Madrid 1899.

[27] Lashley K. S. (1933), *Integrative functions of the cerebral cortex*. Physiological Reviews, **13**: 1-42. Lashley K. S. (1930), *Basic neural mechanism in behaviour*. Psychol. Rev., **37**: 1-24. Lahsley K. S. (1942) *The problem of cerebral organization in vision*. Biol. Symp., **7**: 301-322.

[28] Lange J. (1936), *Agnosien und Apraxien*. Handbuch der Neurologie, Bumke, Foerster (Eds.), Berlin: Springer Verlag, **6**: 807–960.

[29] Stein H, Weizsäcker V. von (1928), *Zur Pathologie der Sensibilität*. Reviews of Physiology, Biochemistry and Pharmacology, **27** (1): 657–708. Stein J. (1928), *Über die Veränderung der Sinnesleistungen und die Entstehung von Trug-wahrnehmungen.* In: Pathologie der Wahrnehmung. Handbuch der Geisteskrankheiten, Bumke O. (Ed.), Bd I, Allgemeiner Teil I, Berlin: Springer 1928. p 351–426. Weizsäcker V. von (1931), *Kasuistische Beitäage zur Lehre von Funktionswandel bei statooptosensiblen Syndromen.* Deutsche Zeitschrift für Nervenheilkunde, 117.

[30] Lhermitte J. (1939), L'image de notre corps. Nouvelle Revue Critique, Paris 1939.

[31] Wernicke C. (1874), Der aphasiche symptomenkomplex: Eine psychologische Studie auf anatomischer Basis. Breslau: Cohn and Weigert 1874.

[32] Koffka K. (1919/1935), Beitrage zur Psychologie der Gestalt. Bd., 1, Leipzig 1919 (Principles of Gestaltpsychology. New York 1935). Köhler W. (1930), Gestalt Psychology. London 1930. Köhler W (1940), Dynamics in Psychology. New York: Liveright 1940.

Human brain functions according to new data and physiological basis

An introduction to the studies conducted on brain dynamics

by **Justo Gonzalo**

Edited by Isabel Gonzalo Fonrodona

English translation by Jamie Benyei and Isabel Gonzalo Fonrodona

Data of the Spanish edition

Title: Las funciones cerebrales humanas según nuevos datos y bases fisiológicas. (Una introducción a los estudios de dinámica cerebral).

First published in: *Trabajos del Instituto Cajal de Investigaciones Biológicas*, Vol. **XLIV**, 95-57 (1952). Included in the facsimile edition in Spanish published by Red Temática en Tecnologías de Computación Artificial/Natural (RTNAC) and Universidad de Santiago de Compostela (USC), 2010. Open Access in http://hdl.handle.net/10347/4341

Contents

"*I have judged that man's knowledge of nature must be taken from the principles of geometry and mechanics, for all other notions we have of sensitive matters, being confused and obscure, cannot provide us with knowledge of anything.*"

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 at not having guessed what was hidden from the outset.*"

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 a poor and quite unsatisfactory thing, whatever it is that one is dealing with.*"

Lord Kelvin

Introduction

These quotes are doubtlessly a difficult demand to be placed on cerebral 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. Firstly, I shall present a summary of my work on brain dynamics [1941[1], (1945-50)[2]], which will be followed by indications of complementary studies conducted in 1950-1952 (see Sec. 7 *et seq*.).

---

[1] Gonzalo, J. (1941). *Investigaciones sobre Dinámica Cerebral. La dinámica en el sistema nervioso. Estructuras sensoriales por sincronización cerebral* (Research on Brain Dynamics. Dynamics in the nervous system. Sensory structures by brain synchronization). Unpublished report presented to the Spanish National Research Council (Consejo Superior de Investigaciones Científicas). Madrid 1941.

[2] Gonzalo, J. (1945, 1950). *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 published in Spanish by Red Temática en Tecnologías de Computación Artificial/Natural (RTNAC) and Universidad de Santiago de Compostela (USC), 2010 (Open Access in http://hdl.handle.net/10347/4341). *Brain Dynamics*, Volume 1 English translation (2021), Open Access at: https://eprints.ucm.es/id/eprint/63730/ ; Volume 2 English translation (2022), Open Access at: https://eprints.ucm.es/id/eprint/72118/

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 on previous page) 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 on previous page). 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 prototype cases M and T, and above all, because it indicated a physiological direction from the outset of this research in 1939. The *dynamic action phenomena* (asynchrony, facilitation[3] and repercussion) consist of transformations of central nervous excitability.

Asynchrony involves the excision of sensory phenomena which are normally united and non-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.

[3] The term used in the original Spanish version is `refuerzo´ = reinforcement in English, and has been translated as facilitation.

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 reinforcement straightens them 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. Regardless of the intensity of the facilitation, however, it is unable to annul the entire deficit. Together with this muscular effect there are other types of spatial 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 summation are also described. For example, a certain class 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.

As well as abnormal sensitivity to spatial summation, there is also sensitivity to "temporal or successive summation", i.e. to the accumulation of successive stimuli, a phenomenon which is also absent in a normal subject. It is highly significant in case M and only quite clear in case T.

Finally, the phenomenon of cerebral *repercussion* of the lesion is the most contrasted concept to the traditional ideas of specific anatomic brain localization. In M and T, the cortical brain lesion is located in a "central zone" which is equidistant from the visual, tactile and auditory zones, their projection areas being not directly implicated 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 of *brain dynamics*, but also in many others.

3. 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 conditions the type of disorder distribution in the brain system, i.e., the topography of the above mentioned `repercussion´. The magnitude or extension of cortical destruction determines the intensity of the disorder.

3.1 Position

Three general cortical syndromes must be distinguished with respect to position: central, paracentral and marginal (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.

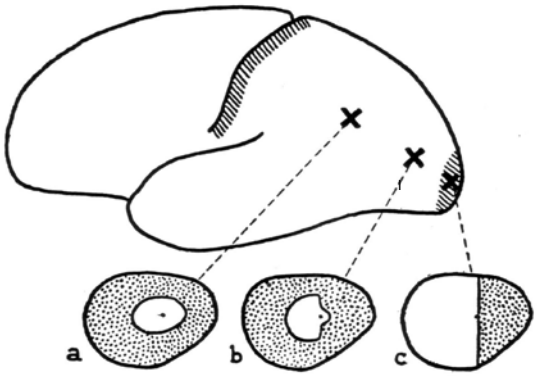

Figure 1: Scheme of positions of cortical lesions (x) and corresponding types of visual field. **a**: symmetric concentric reduction in the central syndrome. **b**: asymmetric concentric reduction in the paracentral visual syndrome. **c**: hemianopsia in the projection path syndrome (marginal or peripheral). The more central the syndrome, the greater the lesion must be to provoke a deficit in the visual field.

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.

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.

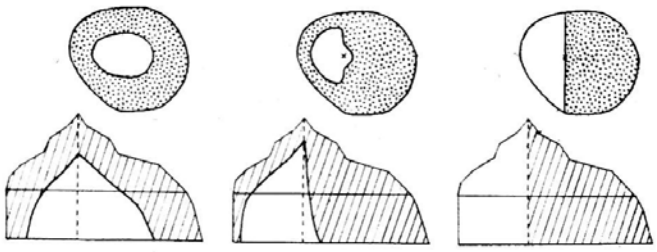

Figure 2: The pathological forms of each visual field (above) and the respective visual profile intensity. In symmetric concentric reduction (central syndrome), there is a *depression* in the sensitivity profile with the form more or less maintained. In paracentral syndrome, the depression is less homogeneous (asymmetrical). In the projection path syndrome, 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* (descent) (Fig.2) which present the above mentioned phenomena of dynamic action.

3.1 Magnitude

The intensity of affectation, i.e. the degree of functional descent (depression) depends on the quantity of cortex destroyed. The best examples of this in the whole work are cases M and T, 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 affectation leads to the establishment of continuous transitions in the various abnormal phenomena

and the exclusion of independent or genuine qualitative defects. This is 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 diverse 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 was the `first principle´ of the brain dynamics developed in this research.

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 the sensory functions, which offer a wealth of manifestations difficult to match by the motor functions. Given the central syndrome in M and T, a structural analysis is possible in any of the three primary sensory systems- visual, tactile and auditory. Apart from their individual peculiarities, they show a common organizational plan. Furthermore, a great number of manifestations may be united under a general principle which is dealt with in a further section.

The structure of the functions is analyzed here under the activity of the sensory field suffering a functional “depression” in our subjects, derived from a deficit in brain 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 system, the excitability disorder involves all types of activity and increases with rises in 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 cases M and T occupies a large part of the precedent book *Dinámica Cerebral.*[4] 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 there is an effort to provide a physiological and quantitative basis of them even in the details.

Much of Vol. 1 of the mentioned book, including from Fig. 8 to Fig. 81, deals with visual functions.[5] 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. Strength-duration curves of light excitability, 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 borderline contrast, abolition of negative afterimages). Color processing in the brain.

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 chromatic irradiation (distorted chromatic perception). Visual acuity. Perception of motion. Perception of figures and objects (metamorphopsia, etc.).

iv) *Visual image orientation*: Inverted vision in diverse degree, 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. 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, whereas reduction is moderate in T. Similar occurrences are found in the reduction of visual acuity, in the abnormal chromatic irradiation due to weakness of specific spatial localization, in the color alteration where blue-violet suffers more as a result of being the slowest or that of greatest chronaxie, in the instability of forms or figures and in the visual perception of motion.

---

[4] Volume 1 English translation (2021), Open Access at: https://eprints.ucm.es/id/eprint/63730/ ;
Volume 2 English translation (2022), Open Access at: https://eprints.ucm.es/id/eprint/72118/

[5] See Sec. 4 -16 in the English translation of Volume 1: https://eprints.ucm.es/id/eprint/63730/

Table 1[6]. 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, chronaxic cap. 3.5 µF. OS: more disturbed.	OD: rheobase 9.5 V, chronaxic cap. 2.7 µF.	OD: rheobase 7.8 V, chronaxic 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 6º Acuity: monoc. 1/25, binoc. 1/10. Strong chromatic irradiation *in medium light.* Severe loss of motion perception. Unstable and diffuse shape perception. Very slow and successive perception.	Visual field up to 40º 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 ~ 50º 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. deviation 145º OS max. deviation 170º Binocularly max. deviation 115º *In very strong light*: deviation about 5º.	OD: from max. deviation in inactive state and same stimulus, a strong re-inversion is obtained, from 145º to 30º and even 20º. OD max. deviation 97º, Binocularly max. deviation 27º.	OD max. deviation 25º OS max. deviation 16º Binocularly max. deviation 10º 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.

[6] This Table is the same as Table 18 in the book *Brain Dynamics*, Vol. 1: https://eprints.ucm.es/id/eprint/63730/

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 150º, whereas only reaching 20º 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.

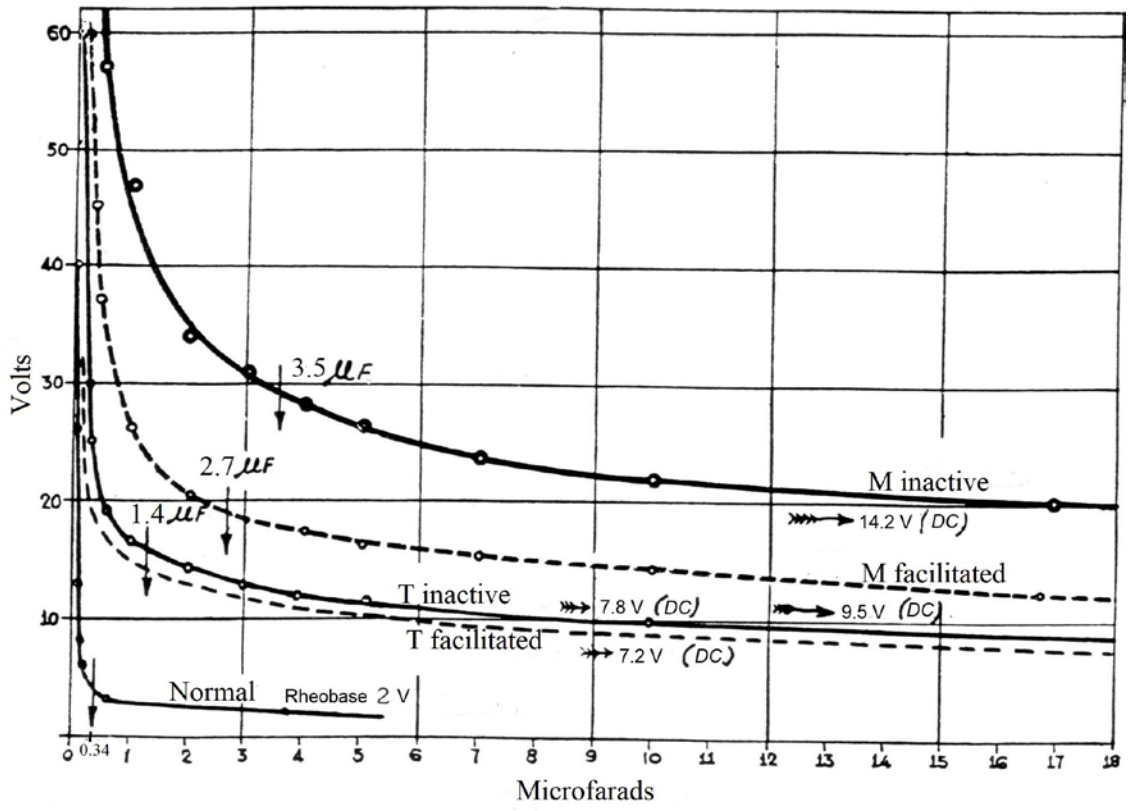

Figure 3: Strength-duration curves (volts versus microfarads) obtained by retina stimulation, with capacitor discharge (cathode on eyelid) to obtain minimum phosphene. Vertical arrows indicate chronaxie capacitances. Compare the values of M and T with those of the normal case, the change of the curve in state of facilitation under strong muscular effort in M, and the smallness of facilitation effect in T.

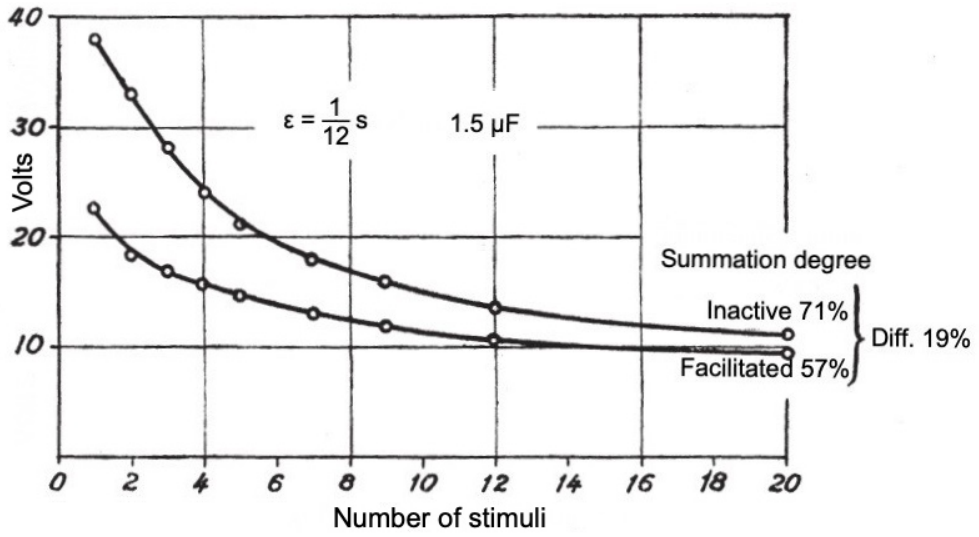

Figure 4: Iterative aptitude in retina in subject M, which is null in a normal subject. Curves for inactive and facilitated states (volts versus number of stimuli) with stimulation as in Fig. 3, for a constant time interval between stimuli ε = 1/12 second and a constant duration of stimulus (capacitance = 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 %.

The study of *colors* is somewhat difficult in these patients due to their chromatic 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.

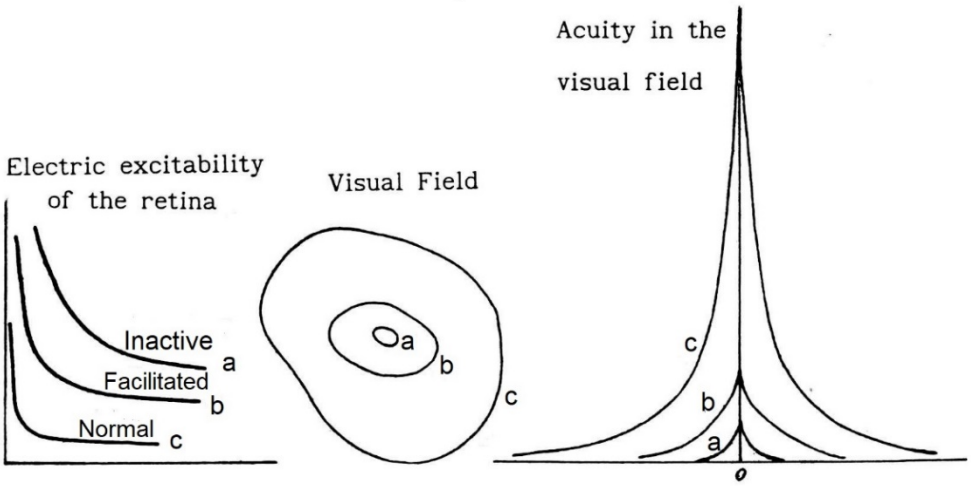

Figure 5: Correspondence between curves of excitability, visual field size and acuity in the visual field, for inactive state (a) and facilitated (b) state in subject M, and in a normal case (c). Acuity in central vision descends to 1/20 in the inactive state and 1/10 in the facilitated state.

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.

Chromatic irradiation or "flat colors" described by Gelb[7], is studied physiologically according to the relationships of excitability. The peculiarity of the disorders in visual perception of motion lies in the phenomena of apparent acceleration and reduction of distance covered 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 the profound experimental study places it entirely within the scope of this research of brain dynamics. It is treated separately for many reasons (see next section on *spiral development*).

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*, 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.[8]

4. Spiral development

This final part on visual functions refers to the phenomenon of inverted vision. Through this phenomenon it can be amply demonstrated the experimental and quantitative analysis which characterizes this research of brain dynamics. The spatial inversion process is connected to other sensory manifestations already analyzed (visual field, colors, acuity,

[7] Gelb A. (1920), reference in *Brain Dynamics* Vol.1: https://eprints.ucm.es/id/eprint/63730/

[8] See Sec. 16.2 in Vol. 1 of *Brain Dynamics*: https://eprints.ucm.es/id/eprint/63730/
and Sec. 27.4.2 in Vol. of 2 *Brain Dynamics*: https://eprints.ucm.es/id/eprint/72118/

motion perception, etc.). It is a functional complex which develops in accordance with the inversion-reinversion process, highlighting the intimate structure of the sensory field according to a spiral development and in accordance with physiological relationships of brain excitability and anatomic textures of the cortex.[9]

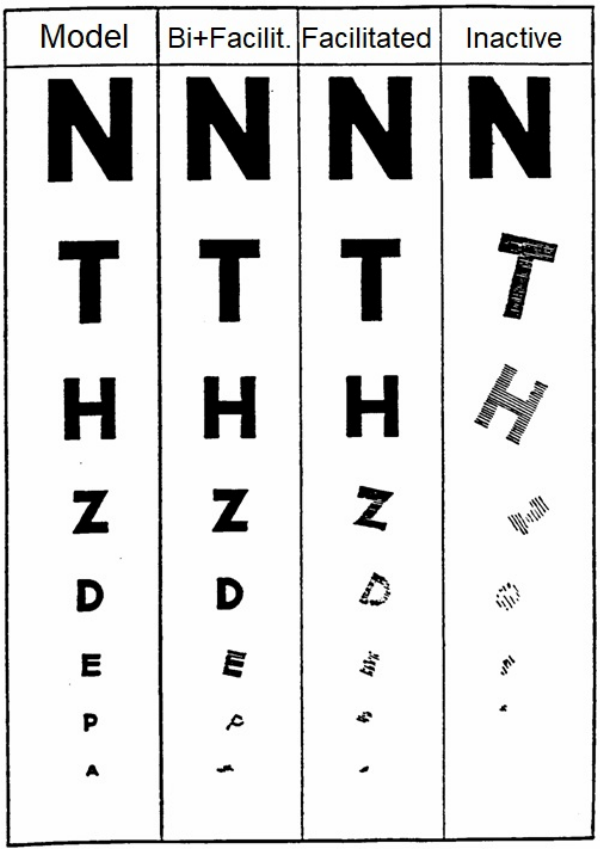

Figure 6: Tilt and fading of the configuration perceived by patient M, according to size of letters and states of summation. Left column is the model to look at. The model is situated at a convenient and fixed distance, so that patient M, with the right eye and in inactive state obtains completely upright (correct) vision only for the tallest letter (N). Then, the smaller letters seem tilted and are illegible beyond a tilt of 40º (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).

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 160º, a very considerable alteration that is in relation to his other characteristics. In patient T there is only a slight tilt of some 20º. 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

[9] See Secs. 12-14 in Vol. 1 of *Brain dynamics*: https://eprints.ucm.es/id/eprint/63730/ and Sec. 26 in Vol. 2 o *Brain Dynamics*: https://eprints.ucm.es/id/eprint/72118/

exposure time. Thus, a nearby object appeared to be greatly rotated if it was only seen for an instant. Case T was included in the same process and interpreted as an attenuating manifestation or frustrated inversion. 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 50º corresponds to hazy vision which impedes the recognition of the object, and beyond 90º and nearing inversion, the object is reduced to a shadow that is ignored, so that great rotations are practically excluded. Subject M usually perceives an almost correct image with small tilts that do not cause disturbance. Thus, large tilts and inversion should be provoked, 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 lead to the discovery of the previously mentioned factors of stimulation.

In order to give an idea of the questions arising from spatial inversion, in this section we analyze 1) the phemonenological aspect, 2) the quantitative aspect, 3) spiral development of the sensory field, 4) brain mechanisms and 5) nerve decussations.

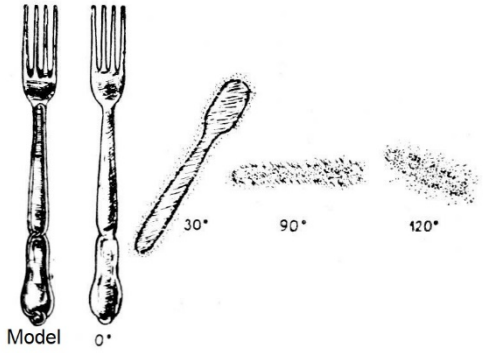

Figure 7: Fading of the configuration through the inversion process. There is also a reduction in size and in light contrast, etc. A metal fork of 20 cm at a distance of 1.5 m is perceived as nearly inverted by M with only one eye, in an inactive state and in medium illumination.

1) Several examples serve to give a *phemoneological* idea of the process. Once beyond a perceived tilt of 40º, 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 30º. At 90º it looks like a smaller, elongated diffuse object; at 140º, 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) More extensive experimental study shows that the process matches certain *quantitative relationships* perfectly, 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.[10] 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.

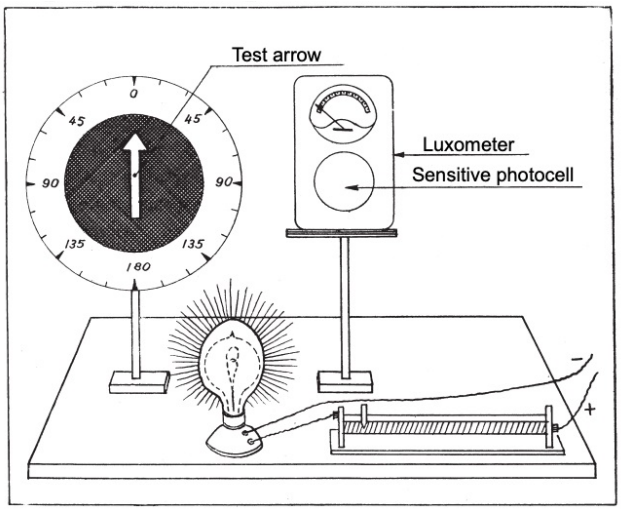

Figure 8: Upright white cardboard arrow on black background, graduated circle for each half, illuminated with dimmable light measured by the luxometer. When lighting is reduced, the arrow seems to be rotated in the frontal plane. The rotation is measured by rotating the arrow in the opposite direction until it is perceived vertical pointing up. Arrow 10 cm size placed 40 cm from the subject.

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 45º 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

[10] See details in *Brain Dynamics*, Vol. 1: https://eprints.ucm.es/id/eprint/63730/ and Vol. 2: https://eprints.ucm.es/id/eprint/72118/

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 case T, a similar but much higher recruitment curve than in case M facilitated 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.

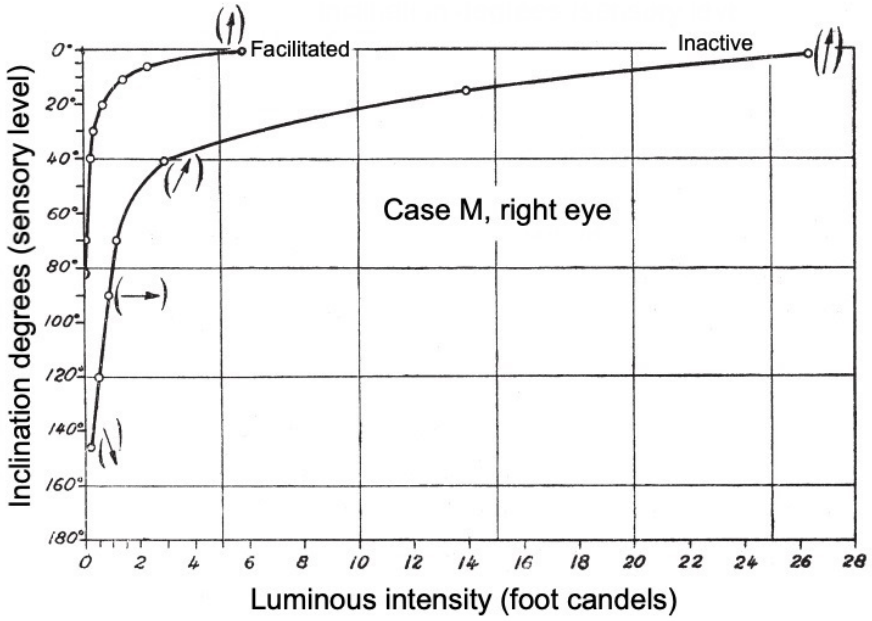

Figure 9: Sensory recruitment curves for the perceived orientation (inclination degrees) of the visual image as a function of the luminous intensity, for subject M in inactive state and under facilitation by strong muscular effort. (Experimental device of Fig. 8).

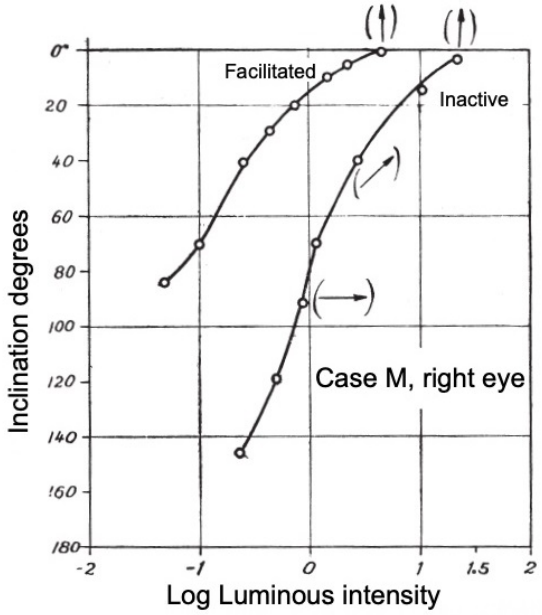

Figure 10: Relationship between angle of the perceived tilt and logarithm of the stimulus intensity (Fechner law).

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 a *spiral development of the sensory field*.

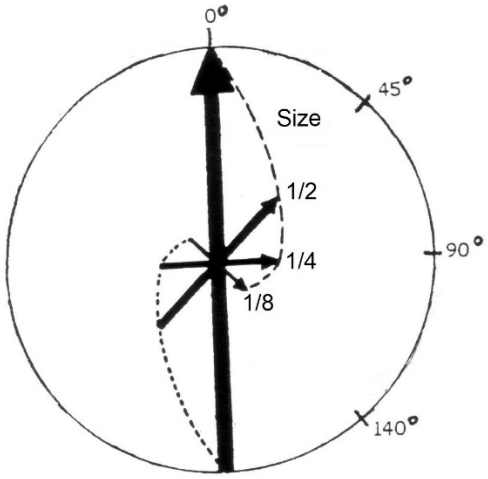

Figure 11: Reduction of arrow size (reduction of the sensory field) when the image is tilted. The extreme of the arrow thus describes a spiral (there is a spiral field development).

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*[11], 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 unexpected 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

[11] Secs. 12-14 in Vol. 1: https://eprints.ucm.es/id/eprint/63730/

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[12]. 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. 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).

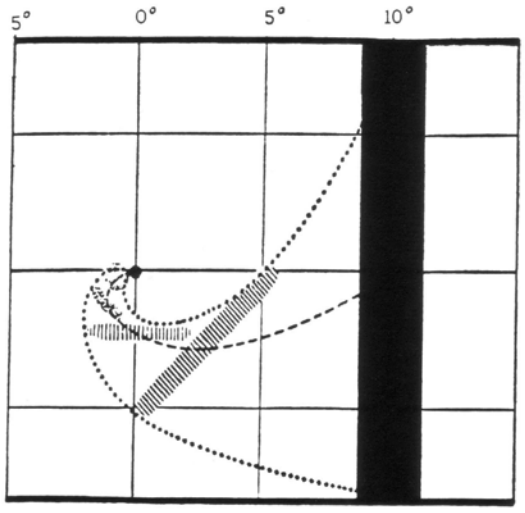

Figure 12: Experiments on visual spiral development in patient M. The test object is placed 10º from the central point of fixation. As visual excitation is conveniently reduced, the image of the object wanes in size and intensity, rotates and ends contralaterally, close to the center. The drawn images represent phases obtained during the process. The trajectories correspond to logarithmic spirals.

[12] Gonzalo, J. (1951). La cerebración sensorial y el desarrollo en espiral. Cruzamientos, magnificación, morfogénesis. *Trabajos del Instituto Cajal de Investigaciones Biológicas*, Vol. **XLIII**, 210-260.

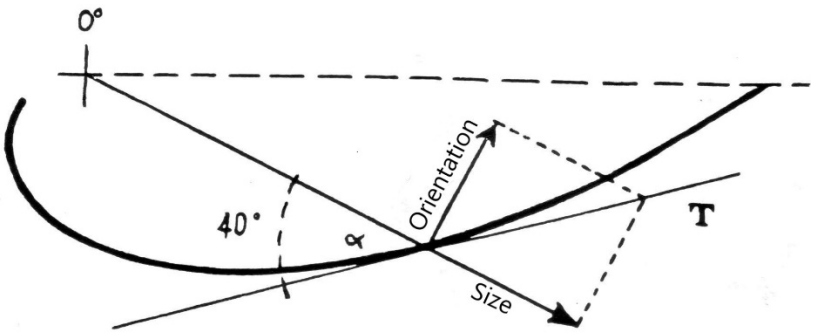

Figure 13: Dynamic characteristics of the spiral in the field development. The two vectors in which the tangent to a point on the curve is decomposed signify the two "forces" of growth causing the spiral: "size" to enlarge the field, "orientation" to re-invert it.

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.

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 the vision so that 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*[13]).

5. Tactile functions. Other functions

Touch and vision have a remarkable structural similarity. However, the former is less objetivable, making its analysis more difficult. The visual-tactile similarity allows now to omit many comments so the exposition here will be very brief. For a quick overview of the topics covered, see the table of contents of volume 2 of the book *Brain Dynamics*.[14]

The following brief remarks refer to the five tactile functions studied. (See details in *Brain Dynamics* Vol. 2).

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.

[13] See Sec. 26.2 in Vol. 2: https://eprints.ucm.es/id/eprint/72118/

[14] Vol. 2: https://eprints.ucm.es/id/eprint/72118/

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.

Tactile sensations (qualities), 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 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 chromatic 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.

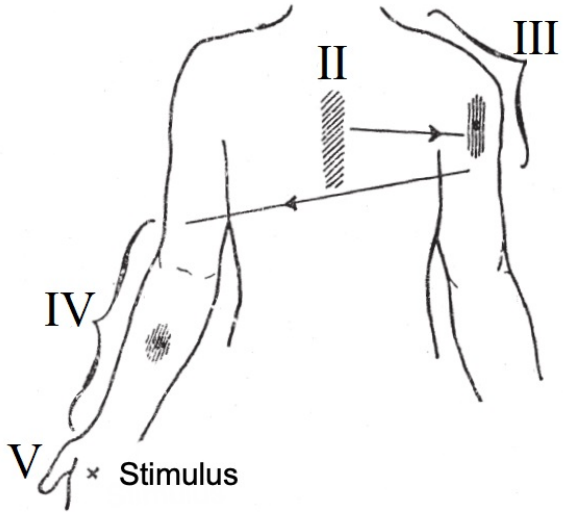

Figure 14: Phases of tactile localization in M in inactive state for mechanical pressure stimulus on a hand provides (according to its intensity): centripetal deviation, contralaterality, and irradiation. Sensory threshold intensity originates tactile sensation lacking localization (phase **I**, which is not in the figure); slight raise in intensity leads to phase **II** or medial deviation, and to phase **III** or 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.

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 coming down the side 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, the inverted movement remains on the contralateral shoulder, and; if it is on the face, the third phase (or inversion) will correspond to the cranial calotte. In summary, a distal 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 rotation or 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.

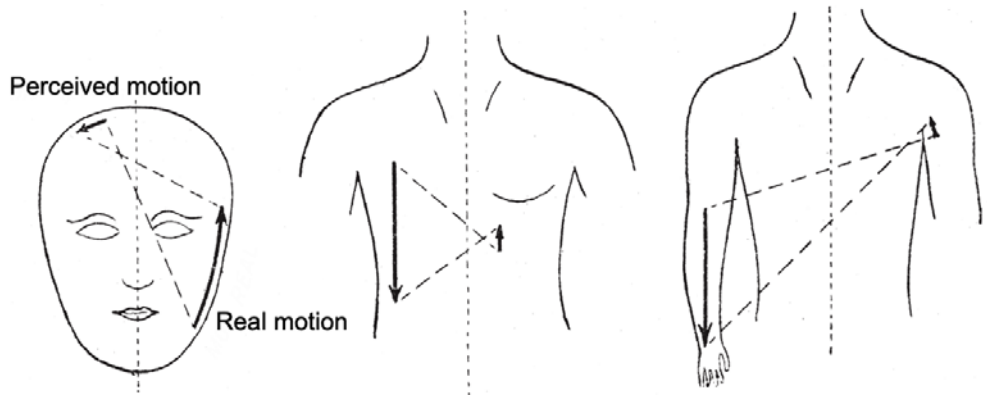

Figure 15: Inversion of motion over the skin in different parts of the body of subject M in the inactive state. The large arrow corresponds to the moving stimulus and the small arrow to the perceived motion. In all cases, the perceived inverted direction of movement is localized contralaterally, approaching to the middle line (proximal deviation) and with much smaller trajectory than the real movement (reduced to 1/10 approximately).

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.

Ttactile scheme 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 scheme 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 hypoacousia, 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 "sonorousness" 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*, diverse 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 the "marginal" 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. (i.e., anesthesia or blindness, astereognosis, agnosia), classification system more or less derived from the doctrine of mental faculties. Table 1 on visual functions shows the great regularity of the affectation of the whole system, a regularity that allows, from 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 desynchronization). This issue should be channeled, taking into account our cases, as 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. In a highly residual field, the values of these parameters tend towards zero, 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 physiopathological needs and considerably simplifies the sensory problem, avoiding bothersome classifications.

These parameters permit a natural description of the state of the sensory field, although they could be believed to only have applications in “lower” sensory functions, whereas the “higher” or intellectualized functions (figures, gnosis, etc.) would be outside. 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 permit its elucidation.

As for agnosia, it should be noted that whereas traditional theory emphasizes the amnesic defect, i.e. the "re-production" (re-knowledge) disorder, here agnosia is approached as a "production" defect (as clearly shown in the orthogonal disorder, in the 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

Accepting the traditional anatomic-clinical method, three syndromes are distinguished: marginal, paracentral and central, which in Fig. 1 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 localizations is simplified substantially and offers new perspectives so that the question seems to flow along its natural course. In traditional localizations, 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 of appreciation. 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.

First, we shall 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 presents cases of *central scotoma* and cutouts due to occipital pole lesion (macular projection in striate area), examples of a marginal syndrome which is now partial. Fig. 17 illustrates 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 highly circumscribed lesion in both occipital poles. Even after numerous civilian and war cases are studied, they are still somewhat 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 the paracentral and central syndromes. In the figures we have designated our most affected cases but none reach the intensity of case M 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, so that any peripheral cause such as papillary stasis is completely discarded.

Cases of *scotoma* have an anatomic explanation (macular projection), although given the diverse degree of visual acuity and 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 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 then 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 cutout 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 depression we have seen and, being dynamic syndromes, are fully ascribed to the system of gradients. The depression is accompanied by phenomena of dynamic action, reduction of functions etc., as set out for cases M and T. In case 1 of Fig. 17, the constriction is significant and the acuity descends proportionally, the dynamism being evident since this case presents a pathological visual image rotation of 40º. 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 6º. In Fig. 18, all cases have a significant dynamic defect. The first one reaches a rotation of the visual image of 90−100º and the sixth reaches 12º 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.

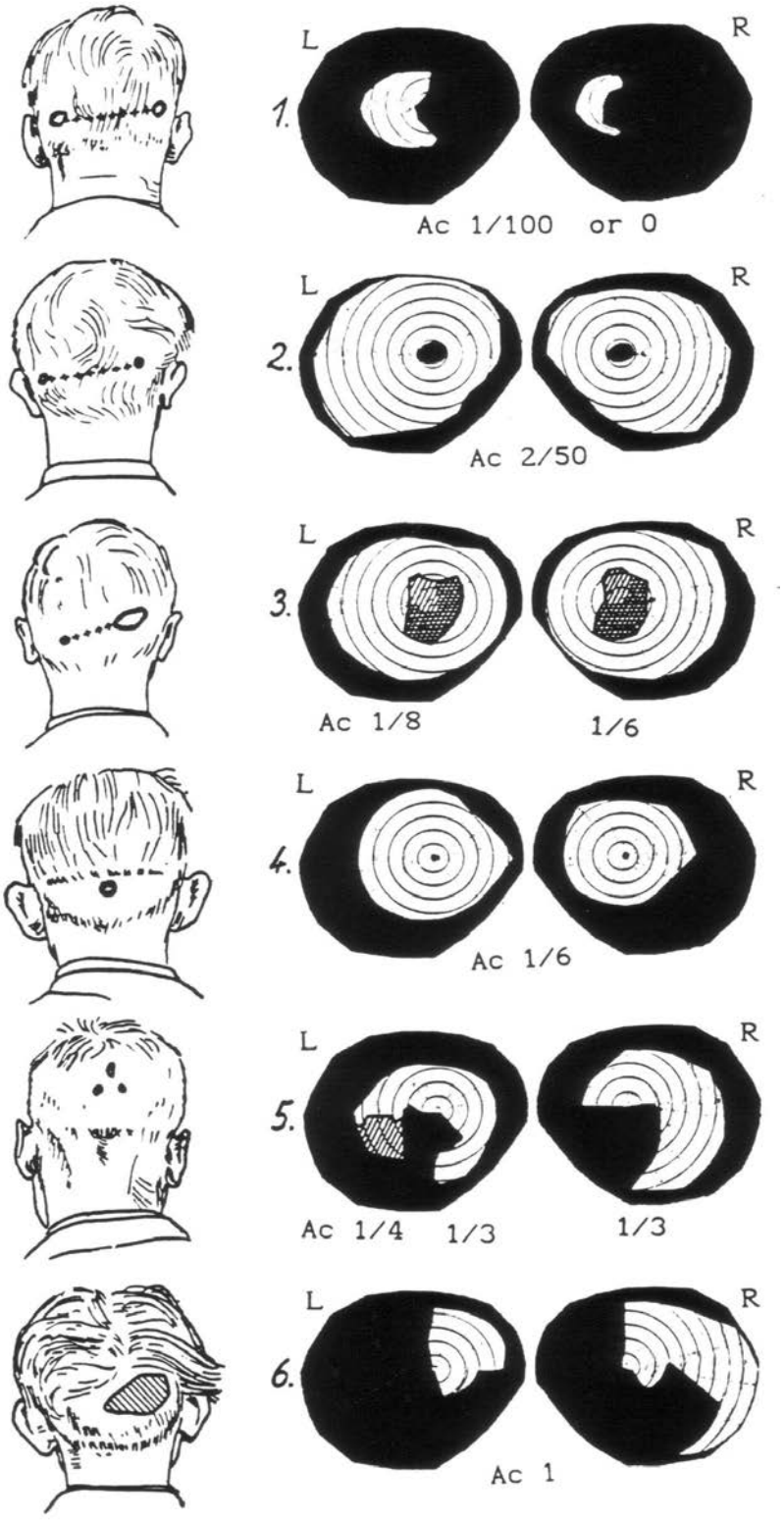

Figure 16: Series of *central scotoma* cases (first four) and central cutouts (final two), due to war lesion in both occipital poles. Note the considerable acuity (Ac) deficit due to cortical involvement of the macula. These cases present an incomplete *margina*l syndrome.

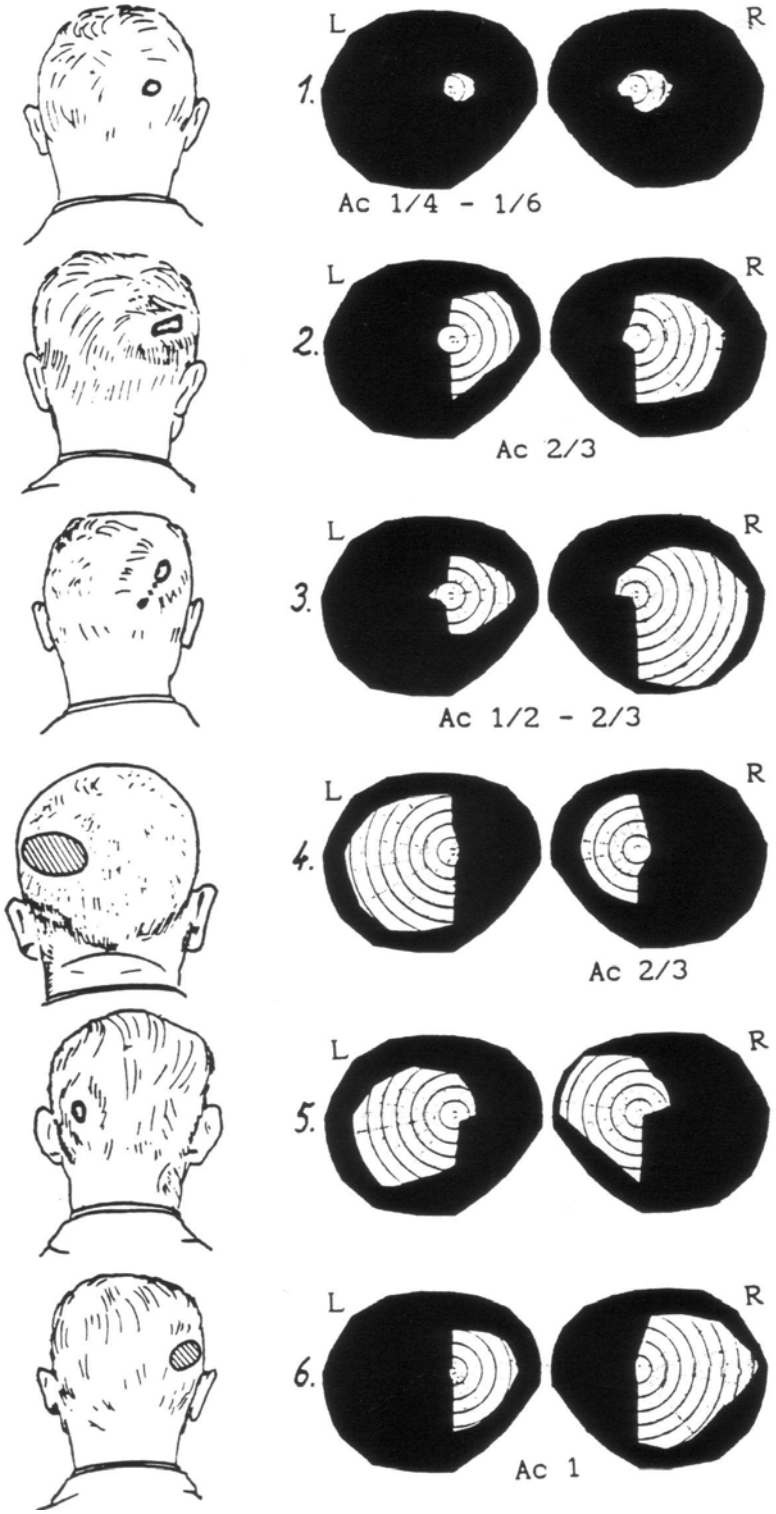

Figure 17: Various cases of lateral cortical *hemianopsia* due to occipital war lesion on one side of the midline. In all cases there is a reduction of the conserved field. The more the reduction the greater the deficit of visual acuity and the more accused desynchronization (case 1 presents a rotation of the visual image of 40º; case 2 only 6º). In this research, all these cases correspond to the *paracentral* visual syndrome with intensity varying from one to another.

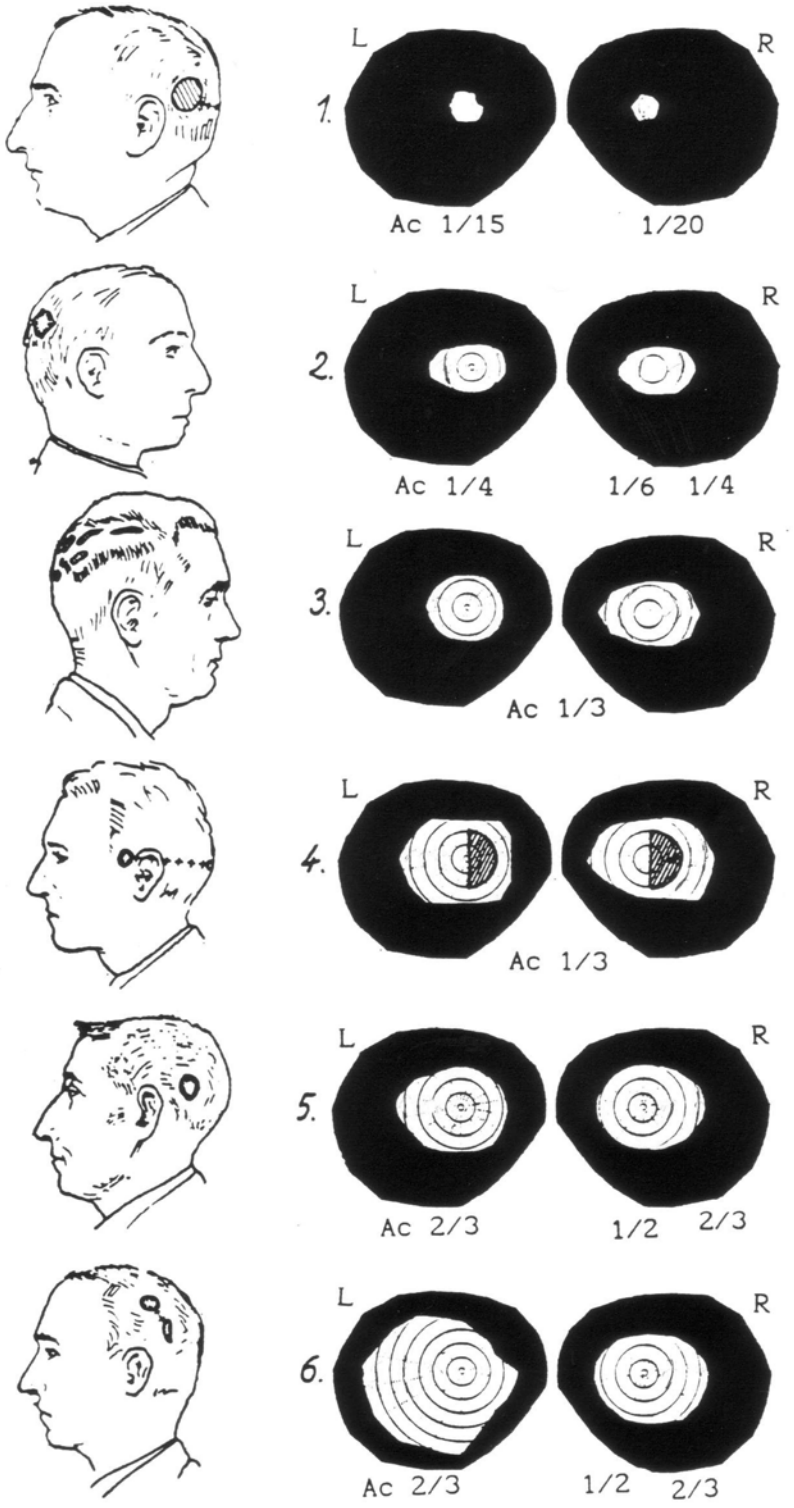

Figure 18: Cases of *concentric reduction* of the visual field due to parieto-occipital lesions which may be very distant from the striate area. An extensive lesion which is close to the visual area produces much greater reduction (case 1) than when further away (case 6). All are cases of *central* syndrome, with the corresponding dynamic phenomena (case 1 presents a rotation of the visual image of 90º; case 6, 12º)

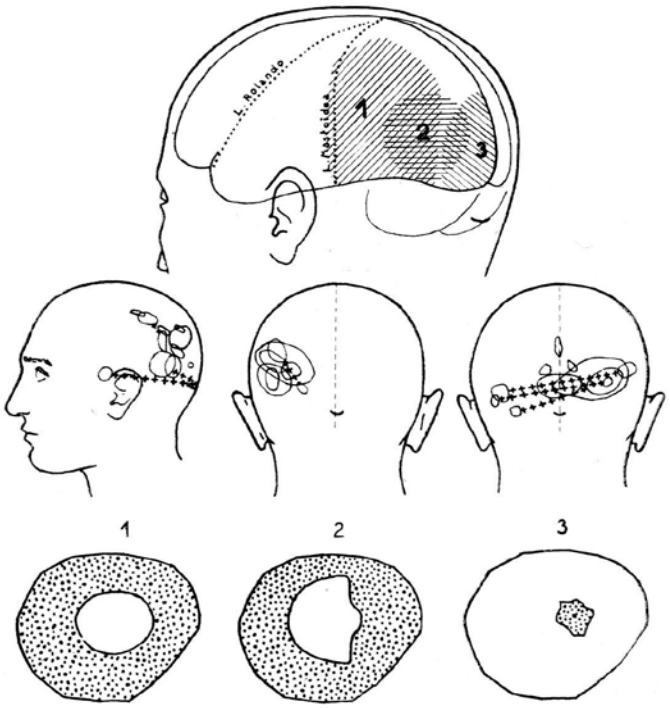

Figure 19: Cranium-clinical correlation showing a synthesis of the three series of cases in Figs. 16-18. 1: concentric reduction; 2: asymmetric reduction; 3: central scotoma. Note the ample participation of the parietal zone in the visual field.

An outlined image of the syndromes according to the double craneo-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, in spite of a certain variation, leading to the system of gradients.

In the sense used here, a functional gradient is defined as function varying 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, beside 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. Such a type of gradient involves all sensory activity, so that for the visual field to be normal, acuity with a value of 1, etc., the action of the critical zone or that of greatest

density is not enough: the whole visual gradation 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.

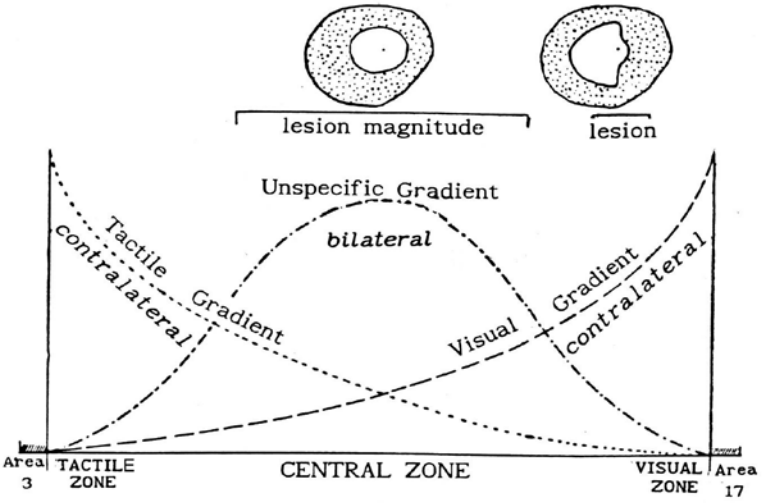

Figure 20: Scheme of cortical gradients. Maximum visual "density" is beside area 17, decreasing towards the central zone and beyond. It determines the *specific visual gradient*, of contralateral action. Analogous for the tactile gradient. The *integration or unspecific gradient* (equipotential) arises 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.

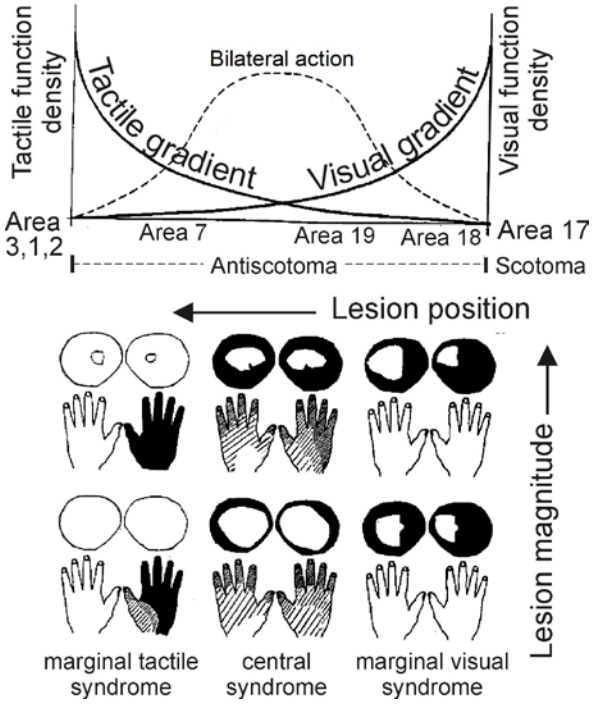

Figure 20' [15] **:** Visual fields and tactile sensitivity of cases ordered according to the position and magnitude of the lesion. The degree of the defect is greater in darker regions

[15] This figure is made by the author after the publication of the article of 1952. It has been included in this edition to better illustrate de concept of cortical gradients.

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 action of the callous, from which the projection zones are excluded, as is known from anatomical and neuronographic 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 Fig. 20.

The gradients system is one of *quantitative localizations according to action fields*, 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 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 so that 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.

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 "desintellectualization" 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 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 cases M and T are perfectly explained by the varying quantity of cortical destruction on the same site, showing the same syndrome in different intensities.

In M it 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 opening consecutive to fracture, and a 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 the book *Brain Dynamics* [16]).

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* point. Contrary to what could be expected, large openings or 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. Case T 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, biparietal or bilateral shots must be discarded as they do not result in lasting disorders, as is the case of shots in one

[16] See Sec. 2.1 in Vol. 1: https://eprints.ucm.es/id/eprint/63730/

hemisphere with 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 case 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, and 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, whereas brain pathology in peacetime is a “leuco-pathology” or white matter pathology (P. Marie). The first one thus has a particular character and is quite important with respect to the cerebral cortex.

The sets of cases in Figures 16, 17 and 18, grouped according to similarity in lesion position and type of corresponding sensory defect, 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 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 may be admitted to be perturbed, one significantly reduced in function (hemianopsia) and the other somewhat depressed (homolateral constriction) due to the interoccipital correlation, whereas in the second case, the complete excision of a 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 or free from superimposed links. This result leads to the interesting suggestion that a paracentral visual syndrome may benefit functionally through an occipital lobectomy in spite of the greater 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 observing in the 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 or depression 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 nor depression would result, as shown in cases of hemispherectomy I have observed. Thus, it seems to be about the amount of energy to be distributed between the two hemispheres according to the functional unit. It should be noted that the suppression of the repercussion might perhaps achieved more simply through the section of the corpus callosum.

We should also indicate that the `magnitude of lesion - magnitude of disorder´ relationship 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 relationship 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 process

Although cases of intense disorders, where the dynamic phenomena can be examined conveniently, are not common because large lesions are rare, we will 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 process in varying degrees.

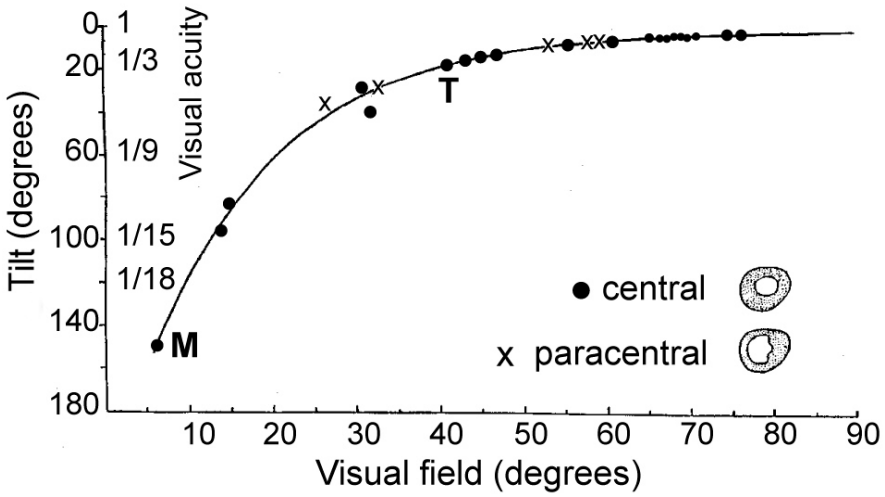

Figure 21: The curve illustrates the ordering of a series of new cases in relation to the perceived tilt (rotation) of the visual image (vertical axis) and to the width (constriction) of the visual field (horizontal axis). Cases M and T are included for comparison. The corresponding visual acuity values 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, a few others with hemianopsia and homolateral constriction (paracentral visual syndrome). In comparison with cases M and T, we see that there are few cases with considerable image rotation and none reaches the intensity of case M. Only twelve cases present a clear rotation of more than 10º, half of which are moderate and even less have a sufficiently marked tilt to be used in a study on spiral development. 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 rotation are included in Fig. 18 (the first case presents 90º of rotation, the sixth almost 15º), others are in Fig. 17 (the first case reaching 40º, the second only 6º). 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 minimum 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 rotation, highlighting the nonlinear quantitative relationship between them. If the field is significantly reduced up to 45º, the rotation only reaches 10º. But for greater constrictions of the field, the rotation (desynchronization) becomes highly significant. As a practical rule, one cannot expect to find cases with significantly marked rotations of up to 100º.

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 case 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 case T. The majority of the important cases showed several peculiarities besides the rotation of the visual image, especially in 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, amnestic 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 (old brain injuries) show no visual disturbance worth noting. There are permanent cases with small image rotations which intensify during the auras, but other subjects only show rotation 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 desynchronization, 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 rotation in the bibliographic research covering a period of about 50 years. However, this phenomenon is practically unknown and goes unnoticed even in the specialized works. The vast majority of the cases collected present sporadic manifestations of tilted or even inverted vision in diverse attacks. Permanent cases are less abundant and a moderate tilt vision is then more common. 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 a 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, so that 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 “amnestic” 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 until the mid-thigh and further up. In contrast, it is much less intense and 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 (temperature > pain > pressure), the vibratory sensitivity, the sensation of passive articular movement, etc., and with an intensity of the involvement in relation to the topography described above.

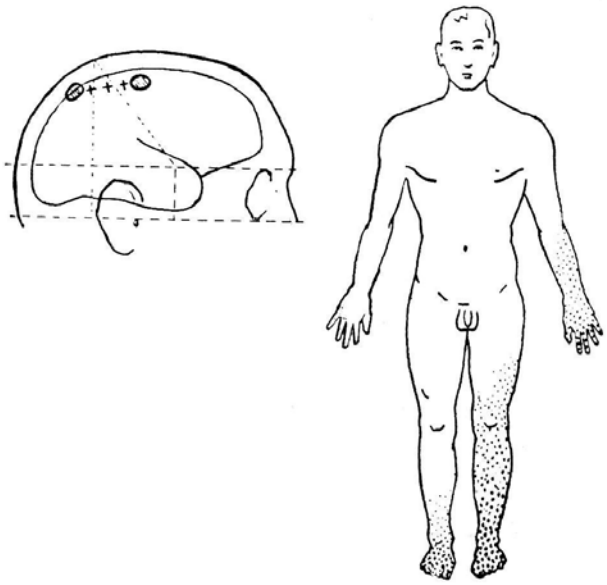

Figure 22: Case of paracentral tactile syndrome, i.e. asymmetric bilateral disorder of tactile sensitivity in unilateral "paramarginal" (paracentral) lesion. The density of the dots indicates the intensity of the disorder.

We are basically facing an *asymmetrical 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), may reach not only the whole tactile field (both sides) but other more distant brain systems according to the type of asymmetrical 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° rotation 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 situation and 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.

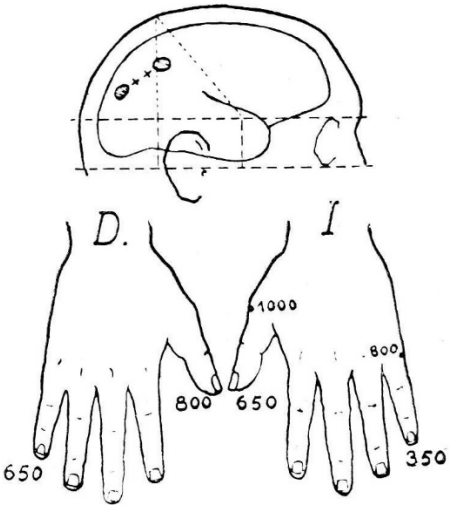

Figure 23: Asymmetric bilateral tactile disorder expressed according to the thresholds of vibratory sensitivity. Normal value: 900–1000 vibrations per second.

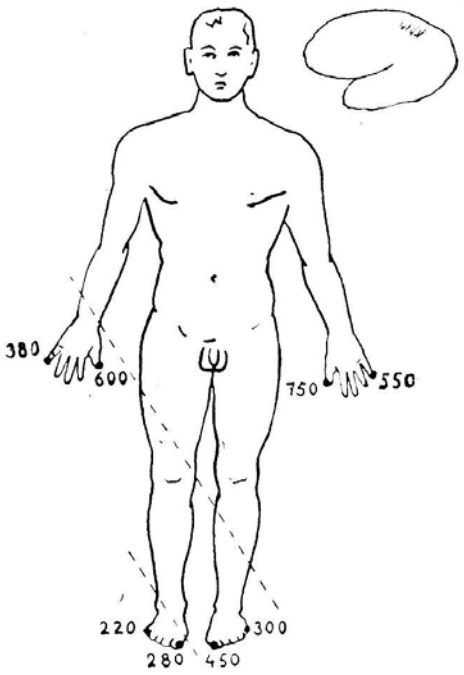

Figure 24: Bilateral tactile disorder involving a larger surface than in Fig. 23. It is determined by the same method of thresholds of vibratory sensitivity. Normal value: 900–1000. Note the

predominance of cubital-peroneal disorder and the double asymmetry, laterally direction and from below to upwards.

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

Although there are some clinical observations on this type of affection in the bibliography, it appears as an obscure matter. Foerster[17] 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 manifest from the outset through the *phenomena of dynamic action* in brain excitability, instituting far-reaching principles which lead to simpler 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* anatomoclinic 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 the localism, being starting point, it is surpassed. Functional or anti-localist orientation, at times brighter than the former but much more diffuse, as well

[17] See references in Vol. 2 of *Brain Dynamics*: https://eprints.ucm.es/id/eprint/72118/

as being in opposition 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, progressively becoming, through a theoretical approach, 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 clearly focused 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 the 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 *physiopathological* concepts (central, paracentral and marginal syndromes, residual field, spiral development, etc.). It provides new signification and interpretation 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 the 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 (in relation to decussations). Thus, the sensory field shows a spiral process, the amplitude of which is a function of the magnitude of the lesion.*
