Short Title: Acoustic diagnosis: apraxia vs dysarthria

Title: Differential Diagnosis between Apraxia and Dysarthria Based on Acoustic Analysis

Authors: Natalia Melle and Carlos Gallego

Universidad Complutense de Madrid (Spain)

Corresponding Author: Natalia Melle. Dpto. Psicología Básica II. Facultad de Psicología. Universidad Complutense de Madrid. Campus de Somosaguas 28223 –Madrid (Spain). E-mail: nmelle@psi.ucm.es

Resumen

El análisis acústico proporciona medidas cuantitativas objetivas del habla que permiten una comprensión global y más exacta de sus trastornos motores complementando a las medidas tradicionales. En el presente trabajo se realiza un estudio diferencial entre normalidad motora del habla y habla patológica y, dentro de ésta, entre apraxia del habla y disartria espástica basado en parámetros acústicos en pacientes hablantes nativos de español. Los participantes (4 afásicos con apraxia del habla, 4 con disartria espástica y 15 sin patología) realizaron tres tareas: repetir la secuencia silábica [pa-ta-ka], repetir la sílaba aislada [pa] y repetir la secuencia vocálica [i-u]. Los resultados mostraron que los valores normativos de control motor coinciden en general con los obtenidos en investigaciones con hablantes nativos de inglés y que la afectación de los procesos de control motor da lugar a un decremento de la tasa de movimientos alternantes y de movimientos secuenciales así como a un incremento de los tiempos intersilábicos para ambos tipos de movimientos. Un subconjunto de los parámetros acústicos analizados, aquellos que reflejan los procesos de planificación motora, permite diferenciar entre la norma y los pacientes apráxicos y disártricos, y a su vez entre estos. Las diferencias encontradas entre ambos grupos patológicos apoyan la distinción entre planificación motora y programación motora descrita en el modelo de procesamiento sensoriomotor de van der Merwe (1997).

Palabras clave

Acústica, apraxia, control motor, disartria.

Abstract

Acoustic analysis provides objective quantitative measures of speech that enable a comprehensive and accurate understanding of motor disorders and complement the traditional measures. This paper aims to distinguish between normal and pathological speech, more specifically between apraxia of speech and spastic dysarthria in native Spanish speaking patients using acoustic parameters. Participants (4 aphasic with apraxia of speech, 4 with spastic dysarthria, and 15 without speech disorders) performed three different tasks: repeating the syllable sequence [pa-ta-ka], repeating the isolated syllable [pa] and repeating the vowel sequence [i-u]. The results showed that the normative values of motor control, in general, coincide with those obtained in previous research on native English speakers. They also show that damage to motor control processes results in a decrease in the rate of alternating and sequential movements and an increase in the inter-syllabic time for both types of movements. A subset of the acoustic parameters analyzed, those that measure motor planning processes, enable differentiation between normal population and apraxic and dysarthric patients, and between the latter. The differences between the pathological groups support the distinction between motor planning and motor programming as described by van der Merwe's model of sensorimotor processing (1997).

Key words

Acoustic, apraxia, dysarthria, motor control**.**

All those alterations resulting from lesions to the peripheral or central nervous system affecting speech motor control processes are included under the heading of motor speech disorders. Two major categories can be distinguished within the motor speech disorders: apraxia of speech and dysarthria (Duffy, 1995; Roig-Quillis & Rodriguez-Palmero, 2008; Gamboa, Jiménez-Jiménez, Mate, & Cobeta, 2001).

To understand and differentiate these two conditions, and given that verbal production is a complex process, it is necessary to use a multidimensional explanation that will provide complementary points of view. Specifically, the emphasis in the explanation and characterization of these conditions can be made on verbal, cognitive or neuroanatomical elements. Each point of view allows for a partial analysis that, when integrated with the others, provides a comprehensive understanding of the pathology. This also enables a more robust differential diagnosis. Thus, the behavioural definition provides information about verbal symptoms, the cognitive definition explains the disorders in terms of altered processes in the context of speech motor control models and finally, the neuroanatomical definition relates behavioural symptoms to the location of neurological injuries detected by neuroimaging techniques (Croot, 2002).

Apraxia of speech is a neurological disorder of central nervous system resulting in a phonetic-motor deficits caused by an inefficiency in the translation of well-formed phonological representations in previously learned kinematic parameters that must be assembled to produce the desired sounds. It is characterized by the presence of intra- and inter-articulatory temporal and spatial distortions of sounds and prosodic distortions. These difficulties are not attributable to deficits in muscle tone or reflexes or do they respond to auditory, tactile, kinesthetic, proprioceptive, or language processing deficits (McNeil, Robin, & Schmidt, 1997). Therefore, apraxia of speech may occur in isolation but may also be present as a symptom in Broca's aphasia syndrome, given the proximity of the brain areas responsible for speech motor control and language production. In this case, extra morphosyntactic alterations of lexical access, reading, writing and others are evident in the patient's condition.

Dysarthria is a neurological disorder produced by an impairment of the central nervous and / or peripheral system that causes changes in the implementation of the motion needed for speech. It is characterized by difficulties in coordination, range, direction, strength and speed of movement of the muscles involved in speech. This leads to problems at the respiratory, phonatory, articulatory, resonator and / or prosodic level, which may appear individually, or in various combinations, depending on the extent and location of the lesion (Darley, Aronson, & Brown, 1969a, 1969b, 1975). Specifically, spastic dysarthria, or pseudobulbar palsy, results from bilateral alterations to the upper motor neuron, both to the pyramidal and the extrapyramidal tract. It is characterized by weakness, hypotonia, hypo- and hyperreflexia, spasticity, reductions in the range and speed of movement of the velopalatal, lingual, labial, mandibular and costodiaphragmatic muscles, and the presence of pathological reflexes (jaw jerk, sucking, snout and gaging) (Love, Webb, & Kirshner, 2004). These neuromuscular alterations translate into a slow speech rate, an excessive and equitable stress for all the syllables of the word, imprecise articulation, vocal distortion, hypernasality, reductions in stress, use of short sentences, small variations in pitch and intensity, breaks in pitch, aggravated tone and hoarse, strained and strangled voice (Darley et al., 1969a, 1969b, 1975).

Both diseases can be explained from a cognitive point of view following the sensorimotor speech control model proposed by van der Merwe (1997). This model integrates concepts derived from neuroanatomy, information processing and motor control theories to establish the existence of five levels of processing (*intentional-communicative, symbolic-linguistic, motor planning, motor programming and motor execution*). Therefore, the motor control is divided into three distinct processes (motor planning, programming and execution), compared with the two traditionally established (programming and execution). Each process represents a different motor skill that can be altered independently thus leading to different motor disorders such as those depicted in Figure 1.

Figure 1

Motor planning basically consists in recovering, from sensorimotor memory, the temporal and spatial specifications (place and manner of articulation) invariant of the phones and their sequential organization. Therefore, during motor planning, there is access to the specific articulator motor plans, not muscle-specific, resulting in an adaptation of the spatial specifications to the phonetic context and the phone production rate, and an adaptation of the temporal specifications to the duration of sound, to the potential coarticulation and to the interarticulatory synchronization, always within the limits of equivalence. The difficulties that may occur at this level include problems in the recovery of the invariant motor plan, in identifying the motor goals of the phonemes, in the sequential organization of movements for each phoneme and for the movements for series of phonemes, in the synchronization between articulators, etc., all of which are characteristic for apraxia of speech.

On the other hand, motor programming involves the determination of muscle-specific programs in spatiotemporal terms and in dimensions such as pitch, rate, direction and range of movement. Motor programs are updated or modified in real time, thanks to sensory feedback and proprioceptive feedback provided by neuroanatomical structures involved in the process. The difficulties detected at this level are associated with defects in muscle tone, rate, direction and range of movements that result in sound distortions, defects in speech rate and / or problems in the initiation of the movements characteristic of spastic, ataxic, hypokinetic and hyperkinetic dysarthria.

Finally, the level of motor performance implies the effective implementation of planned movements through a final common pathway. Injuries that affect this final common pathway lead to the presence of flaccid dysarthria.

Traditionally, the differential diagnosis between apraxia of speech and spastic dysarthria is based on perceptual parameters, on aspects of neuroanatomical lesions and on physiological parameters that reflect the movements of speech mechanism. However, over the last decade, acoustic analysis techniques have become an indispensable tool for the specialist in the diagnostic-intervention process of motor speech disorders. Thus, assuming that apraxia of speech and spastic dysarthria are the result of the alteration of two different speech motor processes (planning and programming, according to van der Merwe's model), such differences should be evident in the acoustic variables that reflect the performance of each of these processes. This has stimulated a growing interest in the scientific community to determine objective and reliable acoustic parameters that reflect speech alterations, as well as to develop standardized protocols that include not only perceptual and physiological criteria but also provide significant acoustic parameters that aid differential diagnosis between normal and pathological and between different types of pathologies (Auzou et al., 2000; Kent & Kim, 2003; Wang, Kent, Duffy, Thomas, & Weisman, 2004). As a result of this, there has been a breakthrough in the construction of new measuring instruments which are increasingly accurate and oriented to the study of specific aspects of the acoustic signal of speech (Roy, Leeper, Blomgren, & Cameron, 2001; Chen & Stevens, 2001; Gonzalez, Cervera, & Miralles, 2002; Schalling & Hartelius, 2004). In this regard, Weisman and Martin (1992) studied the trajectory of vowel formants and found that F2 is the formant that establishes the most direct relationship with the degree of speech intelligibility. F2 localisation indicates the anterior-posterior position of the tongue, situated at higher frequencies when the position is anterior and at lower frequencies when it is posterior. Both in Spanish and in English, the F2 path varies more across the vowels than that of F1. Thus, the Spanish vowels [i - u] represent the two extremes that F2 can reach in the formant map

This study proposes, using the acoustic analysis utilities of the *Sona-Speech II* program, to elucidate: 1) if the normative values in motor planning and motor programming coincide or not with those found in the English language, 2) if the acoustic variables (intersyllabic space duration in alternating and sequential emission of syllables or regularity, amplitude and speed of changes in the F2 of the vowels) allow to distinguish between pathological and normal speech in Spanish speakers, 3) also, if these variables allow to distinguish between speech apraxia and spastic dysarthria, and finally, 4) whether the differences, if found, can be interpreted in the context of the sensorimotor control model proposed by van der Merwe (1997) based on the distinction between motor planning and motor programming.

Method

Participants

Three groups of subjects were selected: 4 patients diagnosed with spastic dysarthria (age range: 14-49 years, $\bar{X} = 26$), 4 patients diagnosed with apraxia of speech with concomitant aphasia (age range: 31-55 years, $\bar{X} = 44.5$) and 15 normal subjects with no history of neurological, psychiatric or language disorders, which formed the control group (age range: 22-52 years, $\bar{X} = 32.46$). All participants were native Spanish speakers and they all gave their consent for the recordings and participated in the study altruistically.

Patients were selected through standard clinical diagnostic criteria that included medical and speech therapy criteria. Among the latter, diadococinetic tasks or sequential movements were not used as diagnostic criteria in order not to alter the results, since these tasks are part of the research design. Three therapists, specializing in the diagnosis of neurological speech and language disorders, carried out an initial assessment to select the patients who would form the sample. The authors confirmed the diagnosis independently.

To select participants with spastic dysarthria, the medical criteria used were the location of the lesion and the presence of neuromuscular disorders associated with this pathology. Regarding the location of the lesion, it was decided to include patients with lesions to the cortex, the corona radiata, basal ganglia, internal capsule and / or the brain stem. Regarding the presence of alterations, the existence of the following neuromotor abnormalities was considered as an inclusion criterion: hypertonia or spasticity, reduced muscle strength, range and rate of movement, salivation, hyperreflexia (gag reflex) and dysphagia (Duffy, 1995; Kent, 1997). The initial speech therapy examination was conducted from a short speech sample, using, as a criterion for inclusion in the investigation, the appearance of more than one perceptual dimension of those identified by Darley et al. (1975) for this condition. Consequently, consonant inaccuracy, vowel distortions, monopitch, monoloudness, hypernasal, strained – strangled voice quality, were used to identify patients that would be part of the study sample.

For the selection of aphasic participants with apraxia of speech, a similar procedure was carried out. Regarding the medical aspects, the site of injury and neuromotor abnormalities associated with these disorders were considered. For the first aspect, patients who had lesions to the left frontal lobe, specifically in Broca's area, to the supplementary motor area, to the primary motor cortex, to the prefrontal cortex and / or to the insula were included as sample. Regarding the associated neuromotor alterations, it was observed that they were not so severe that could explain the presence of the observed speech difficulties. Therefore, the presence of mild facial and lingual weakness and / or concomitant non-verbal oral apraxia were admitted, but not the existence of abnormal reflexes, significant alterations in muscle tone, involuntary movements, dysmetria and / or kinetic tremors. In connection with the speech therapy evaluation, mainly, the existence of Broca's aphasia was identified, through the *Aphasia and Related Disorders Evaluation Test* (Goodglass & Kaplan, 2005), and a small sample of spontaneous speech was analyzed. In this sample, articulatory features characteristic of apraxia of speech were investigated. These are: the presence of substitutions, omissions, additions and repetitions of sounds, the greater amount of articulation errors as the length of words increases, a slow speech rate, pauses between sounds, syllables or words that give an impression of segregated speech, hesitation, position searching, error awareness and / or correction attempts (Duffy, 1995).

Material

For this study, the KayPENTAX's Sona-Speech II program (Model 3650) was used. This software is specifically designed to aid the clinician with analysis and feedback of the acoustic signal produced by the patient. It allows the clinician to make and store speech recordings, edit speech samples, to obtain objective acoustic parameters in both numerical and / or picture form and to make comparisons with the normal parameters of certain variables. This software is also useful in designing specific protocols for assessment and intervention and in graphically displaying the patient's execution to make the intervention more effective. It has eight independent modules that specifically address different aspects of the acoustic signal (habitual pitch, maximum phonation time, pitch range, phonation/respiration control, amplitude perturbation, diadochokinetic rate, etc.). These modules are *real-time pitch, voice games, sona-match multi-dimensional voice program, motor speech profile, waveform editor, real-time spectrogram and auditory feedback tools*. Two modules were used for the purposes of this studio: *real-time pitch and motor speech profile*.

Specifically, the *real-time pitch* module is a specific tool used to explore and work with aspects related to the fundamental voice frequency, intensity, stress patterns, intonation and diadochokinetic rate.

In a similar way, the *motor speech profile* module provides an analysis of the aspects related to speech motor disorders present in patients with dysarthria or apraxia of speech through a numerical and graphical display of the results. Both modules have a set of pre-designed protocols, for example, the measurement of second formant transitions in the [i-u] vowel sequence or the analysis of the diadochokinetic rate (rate at which a predetermined sequence of syllables is performed). In these modules, the patient listens to a sample of what he/she must reproduce to then proceed with the recording and quantitative analysis of their speech. Patient results are contrasted with the values given by an extensive database that includes normative values.

Procedure

Previously, participants were trained to perform three different tasks: repetition of sequential [i-u] vowel movements, repetition of alternating [pa] syllabic movements and repetition of sequential [pa-ta-ka] syllabic movements. To record the speech sample, a Shure SM48 microphone, placed at an approximate distance of 15cm from the mouth and at a 45 ° angle on the mouth horizontal axis, was used. The Sona-Speech II program, installed on a conventional computer, was used to perform the recording. The procedure was performed in different ways according to the task. For the first two tasks, the pre-designed models provided by the Sona-Speech II program included in the *motor speech profile* module were used. For the third task, the in-situ performance of one of the researchers was used as a model. Depending on the performed task, aspects such as the second formant transitions, the inter-syllabic time and / or number of syllables per second were analysed as detailed below.

In the sequential vowel movement task, participants were asked to repeat the sequence [i-u] as quickly as possible without losing intelligibility during eight seconds as they were shown by the model provided by the *second formant transition* evaluation protocol of the *motor speech profile* module. Once the speech sample was taken, the program enabled the calculation of the variations in hertz of the second formant in the realization of the [i-u] vowels (*F2magn* variable, *Magnitude of F2 Variation*), the rate of variation change that occurs in the second formant when changing vowels between [i-u] in Hz / sec (*F2rate* variable, *Rate of F2 Variation*) and the percentage of regularity with which changes occur in the second formant in the production of the [i-u] vowels (*F2reg* variable, *Regularity of F2 Variation*).

On the other hand, in the alternating syllabic movements task, the participants had to repeat, as quickly as possible, the syllable [pa] during eight seconds after hearing the pattern given by the computer from the *diadochokinetic rate* evaluation protocol of the *motor speech profile* module. From the obtained speech sample, the average time, in milliseconds, lapsed between the end of a syllable and the beginning of another was calculated (*DDKavp* variable, *Average DDK period*). The rate or number of syllabic vocalizations in a given time determined by number of syllables per second (*DDKavr* variable, *Average DDK rate*) was also calculated.

Finally, in the sequential syllabic movements task, participants were asked to repeat, as quickly as possible, the sequence [pa-ta-ka] during eight seconds after hearing the sample provided by one of the researchers. The speech sample collected was recorded using the *diadochokinetic rate* protocol of the *real-time pitch* module. This was analysed manually using the execution display on the computer screen, the measure of the average time in milliseconds between the end of a syllable and the beginning of the next (*PTKavp* variable, *Average PTK period*) and the average rate or number of syllables that the participant is capable of performing in a given time, measured in number of syllables per second (*PTKavr* variable, *Average PTK rate*).

Due to the difficulties that these patients show in motor planning and programming, the speed of movement is expected to be slower and with more inter-syllabic spaces in the performance of both pathological groups versus the normal group. It is expected that the above measures will allow discrimination between pathological (apraxia of speech and dysarthria) and normal groups, except in the case of F2reg, as alterations in the regularity of the movement are not characteristic of spastic dysarthric or apraxic patients, because the injured neuroanatomical structures responsible for these conditions are not involved in such skill.

It is also expected that the dysarthric group will present less changes in F2magn during vowel sequences than the other two groups (apraxic and normal) due to the neutralization of the second formant caused by the presence of spasticity. This spasticity prevents rapid and wide changes in the muscle movements which results in a reduction of the lingual muscular route.

Finally, it is expected that the apraxic group will present greater alternating and / or sequential inter-syllabic times compared to the dysarthric group. Also, they will present slower alternating and / or sequential movement rates than the dysarthric. These difficulties are due to the limitations of the apraxic to retrieve the syllabic and / or vowel motor patterns of sounds as well as to establish the sequence of movements involved in them, which are accentuated as the structural complexity of the production increases. On the contrary, the inter-syllabic times and rates of the dysarthric group should be closer to the norm and the differences between them would be explained by the presence of spasticity which would affect the range and rate of movement.

Results

The data were analyzed using the SPSS 17.0 statistical analysis program. Nonparametric statistical analyses were carried out due to the small number of subjects in the pathological groups. Descriptive statistics, mean and standard deviation of each of the variables studied are shown in Table I. In the values shown in Table I, it is observed that both groups of patients show lower rates in alternating [pa] and sequential [i-u] and [pa-ta-ka] movements (DDKavr, F2rate and PTKavr) than the control group and higher inter-syllabic times in the performance of both types of movements (DDKavp and PTKavp).

Table I

Figure 2 shows the differences in the response patterns of each of the groups for the variables that reflect the ability to perform sequential vowel (F2rate), alternating and sequential syllabic (DDKavr and PTKavr) movements, characteristic of motor planning. In the rate of sequential vowel movements, it is observed that the slowest subjects are those with apraxia of speech, appearing on the left of the graph. On the contrary, non-pathological subjects appear on the right side, indicating higher speed in their movements whereas subjects with dysarthria show intermediate speed. As can be observed, the same occurs with alternating and sequential syllabic tasks. Thus, this subject distribution highlights the differences between pathologies and between normal and pathologic patients consistent with the distinction between planning and programming proposed by van der Merwe's (1997) model.

Figure 2

A closer analysis of the data using the Kruskal-Wallis variance by range analysis allows to conclude that all variables except F2magn, and perhaps F2reg, show significant differences (p <.05) between groups (Table II).

Table II

The Kolmogorov-Smirnov correction test was used to observe the direction of differences shown by comparing the groups two by two on all those variables that were significant in the first analysis (Table III).

Table III

As shown in Table III, the apraxic group performs significantly worse than the normal group in the execution of alternating and sequential syllabic movements, both in terms of the rate and the inter-syllabic duration (DDKavr: ZA.N = 1.659, p = .008; DDKavp: $Z_{A.N}$ = 1.659, p = .008; PTKavr: $Z_{A.N}$ = 1.777, p = .004; PTKavp: $Z_{A.N}$ = 1.777, p = .004). Similarly, they also perform significantly worse in the execution of sequential vowel movements (F2rate: $Z_{A.N}$ = 1.777, p = .004).

Comparing the dysarthric and the normal groups, only the reduction in the rate of sequential movements and the increase of the inter-syllabic time allow to differentiate between the two groups (PTKavr: $Z_{D.N}$ = 1.414, p = .037; PTKavp: $Z_{D.N}$ = 1.422, p = .035). Meanwhile, the reduction in the rate of diadochokinetic movement and the increase in the inter-syllabic time found for this task, does not permit differentiation between groups (DDKavr: $Z_{D.N}$ = 1.214, p = .105; DDKavp: $Z_{D.N}$ = 1.214, p = .105).

Finally, the results show that the variables involved in the more complex motor planning processes, particularly PTKavr ($Z_{A.\ D}$ = 1.422, p = .035), PTKavp ($Z_{A.D}$ = 1.414, p = .037) and F2rate ($Z_{A.D}$ = 1.414, p = .037), allow to significantly distinguish between both pathologic groups.

Discussion

In general, the results obtained by the control group are similar to those found in other research with an English-speaking standard sample. In this way, Deliyski and DeLasuss (1997) found values close to those obtained in this investigation both for: the syllabic rate and the average inter-syllabic duration in alternating movements ([pa]), and the variability, consistency and rate of change in F2vocals. Similarly, Kent (1997) reported similar results in a review of studies on rates of alternative movements, including [pa] and sequential [pa-ta-ka] movements.

From the results obtained for all groups, it can be concluded that there are acoustic variables that allow distinguishing between pathologic and normal speech. Specifically, the variables related to the sequencing of articulatory syllabic movements (PTKavr and PTKavp) allow distinguishing between apraxia of speech and spastic dysarthria from normal. Furthermore, the rate of change of the second formant changes in the [i-u] sequence (F2rate) and alternating syllabic movements (DDKavr and DDKavp) allow distinguishing between apraxia of speech and normal. Thus, the values obtained for these variables show their clinical utility for the diagnosis of motor speech disorders in Spanish speakers in a manner that is analogous to that previously tested for English speakers (Ackermann, Hertrich, & Hehr, 1995; Wang et al., 2004; Nishio & Niimi, 2006).

On the contrary, and as was expected, the results collected in the percentage of regularity with which changes occur in the second formant in [i-u] vowels (F2reg) do not reflect differences between pathologic and normal conditions as the regularity of movement is not affected in the analysed pathologies.

The results are less clear with respect to the F2magn variable that measures the magnitude of the changes in the second formant. It was expected that this variable would allow distinguishing between spastic dysarthria and normal even if it did not allow distinguishing between apraxia of speech and normal. While the latter is confirmed, the first statement has not been verified, F2magn does not allow distinguishing between dysarthria and normal. Even though the magnitude of change in F2 is lower in the dysarthric group than in the normal group, this difference is not significant;

therefore, this variable is not clinically useful with Spanish speakers. These results contradict those obtained for English-speakers, which show that the appearance of the centralization of vowel formants in patients with spastic dysarthria is clinically significant (Ziegler & von Carmon, 1986a, Ziegler & von Carmon, 1986b; Roy et al., 2001). This could be explained by the existing differences between the two languages with respect to F2 of the vowels involved. The distance of the F2 in English vowels is less than that for the Spanish vowels and therefore, the formant is more easily centralized. Another possible explanation could be the difference in the severity of the pathology experienced by patients in the various studies. The level of spastic dysarthria in patients selected for this research was less severe than the level of dysarthria in the patients studied by the mentioned authors. Therefore, the movement ability of these patients would be greater and would not result in formant centralization.

Questioning whether these measures distinguish between motor speech disorders, it can be noted that these results show that some variables not only allow to distinguish between pathology and normal, but, as expected, they also reflect differences between spastic dysarthric and apraxic patients.

Thus, the variation rate of the second formant changes in the [i-u] sequence (F2rate) and the sequential syllabic movement measures (PTKavr and PTKavp) show significant differences between both groups. The spastic dysarthric group was much faster, with higher execution rates and shorter inter-syllabic time than the apraxic group.

These differences can be explained within the framework of the sensorimotor model proposed by van der Merwe (1997). The poor results for these measures shown by the apraxic group are due to deficits in motor planning – recovery processes, sequential planning and motor pattern update. Meanwhile, the minor difficulties observed in the spastic dysarthric group for PTKavr and PTKavp variables, which significantly differentiate them from both apraxic and normal groups, could be explained simply by deficits in motor programming processes. That is, the slowing of transitions between articulatory positions characteristic of the change in the oral tract configuration which reflect the difficulties shown by these patients, described by other authors (Ziegler & von Carmon, 1986a; Ziegler & von Carmon, 1986b). Indeed, the absence of difficulties for the dysarthric group in high-level motor control and planning, has determined that their scores for F2rate, which also involve recovery, sequential planning and motor pattern update processes, differ significantly from those for apraxic group but not for the normal group, even if their performance is worse than the normal group.

The results, however, are not so clear with respect to the measures in the alternating syllabic movement task (DDKavr and DDKavp). Similarly to what happened in the sequential movement task (PTKavr and PTKavp), the apraxic group has performed significantly lower than the normal group in the alternating movement task, which also involves retrieval and motor pattern update, and, therefore, planning processes. However, although the dysarthric group has performed better than the apraxic group for this task, their differences were not significant. Therefore, these measures do not allow discrimination between the two groups. In this case, the poor performance of the dysarthric group could be attributed to planning difficulties because, as was noted earlier, this task involves motor pattern retrieval and update processes. However, as there are also no significant differences with the normal group, it would be more plausible to interpret the difficulties of the dysarthric group as a consequence of motor slowing and therefore as scheduling difficulties.

In conclusion, the nature of the differences found between the apraxia of speech and spastic dysarthria groups for those variables that best reflect motor planning processes (PTKavr, PTKavp, F2rate) could corroborate the existence of two distinct levels of motor control, planning and programming, as proposed by van der Merwe's (1997) sensorimotor model. That is, the difficulties observed in individuals with spastic dysarthria seem to respond more to the limitations in movement range of the speech muscles characteristic for these patients than in motor planning problems, as is evidenced by the normal rate of sequential vowel movement and the reduction, although not significant, of the F2 variation in such movements. On the other hand, the problems present in persons with apraxia of speech are evident in the recovery and planning of alternating syllables tasks and, even more so, in the sequence planning of sounds, vowels or syllables.

However, these results should be viewed with caution as the sample of apraxic and dysarthric patients of the study is small. In addition, each patient was selected at different times of the injury process, and therefore have participated in rehabilitation programs with different intensities, which could beaffecting the results.

Future studies should consider increasing the sample size of subjects, controlling the severity and time after injury at the time of inclusion in the experiment and combining the acoustic analyses that have been significant with physiological analysis of the articulatory organs involved to give a more complete and accurate explanation of the problem. Thus, they could be combined with kinematic measures resulting from the use of, for example, pressure transducers applied to the jaw and lips that analyze anterior-posterior and upper-lower movements during speech, or with the use of electropalatography or electroglottography techniques which provide data on the movement of the tongue inside the oral cavity and on the vocal cords, respectively.

In any case, the acoustic parameters analyzed are shown to be valuable in the differential diagnosis of speech pathologies. They also show a potential value in terms of intervention. It is clear that a more accurate knowledge of pathological speech based on these parameters can achieve a better diagnosis and a greater effectiveness in the design of specific intervention programs and procedures.

References

Ackermann, H., Hertric, I., & Hehr T. (1995). Oral diadochokinesis in neurological dysarthria. *Folia Phoniatrica et Logopaedica*, *47*, 15-23. http://dx.doi.org/10.1159%2F000266338

Auzou, P., Özsancak, C., Morris, R., Jan, M., Che, F., & Hannequin, D. (2000). Voice onset time in aphasia, apraxia of speech and dysarthria: a review. *Clinical linguistics & phonetics, 14*, 131-150.

Chen, H., & Stevens, K. (2001). An Acoustical Study of the Fricative /s/ in the Speech of Individuals With Dysarthria. *Journal of Speech, Language, and Hearing Research, 44*, 1300-1314. http://dx.doi.org/10.1044%2F1092-4388%282001%2F101%29

Croot, K. (2002). Diagnosis of apraxia: Definition and criteria. *Seminars in Speech and Language*, *23*, 267-281.

Darley, F.L., Aronson, A.E., & Brown J.R. (1969a). Clusters of deviant speech dimensions in the dysarthrias. *Journal of Speech, Language, and Hearing Research*, *12*, 462-469.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1969b). Differential diagnostics patterns of dysarthria. *Journal of Speech, Language, and Hearing Research*, *12*, 246-269.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1975). *Motor speech disorders*. Philadelphia. Pennsylvania: W. B.Saunders.

Deliyski, D., & Delassuss, C. (1997). *Characteristics of motor speech performance: normative date*. Boston, Massachusetts: ASHA.

Duffy, J. R. (1995). *Motor speech disorders: substrates, differential diagnosis, and management.* St. Louis. Missouri: Mosby.

Gamboa, J., Jiménez-Jiménez, F. J., Mate, M. A., & Corbeta, I. (2001). Alteraciones de la voz causadas por enfermedades neurológicas [Voice disorders caused by neurological diseases]. *Revista de neurología, 33*, 153-168.

González, J., Cervera, T., & Miralles, J. (2002). Análisis acústico de la voz: Fiabilidad de un conjunto de Parámetros Multidimensionales [Acoustic analysis of the voice: Reliability of a set of multidimensional parameters]. *Acta Otorrinolaringológica Española*, *53*, 256-268.

Goodglasss, H., & Kaplan, E. (2005). Evaluación de la afasia y de trastornos relacionados [Assessment of aphasia and related disorders]. Madrid, España: Editorial Panamericana.

Kent, R. (1997). The perceptual sensorimotor examination for motor speech disorders. In M.R. McNeil (Eds.), *Clinical management of sensorimotor speech disorders*. New York, New York: Thieme.

Kent, R. D., & Kim, Y. J. (2003). Toward an acoustic typology of motor speech disorders. *Clinical linguistics & phonetics* , *17*, 427-445.

Love, R., Webb, W., & Kirshner, H. (2004). *Neurology for the speech-language pathologist*. Butterworth-Heinemann: Elsevier.

McNeil, M. R., Robin, D. A., & Schmidt, R. A. (1997). Apraxia of speech: definition, differentiation and treatment. In M.R. McNeil (Eds.), *Clinical management of sensorimotor speech disorders*. New York, New York: Thieme.

Nishio, M, & Niimi, S. (2006). Comparison of speaking rate, articulation rate and alternanting motion rate in dysarthric speakers. *Folia Phoniatrica et Logopaedica, 58*, 114-131.

Roig-Quillis, M., & Rodríguez-Palmero, A. (2008). Trastornos oromotores en una unidad de neurología pediátrica. Clasificación y evolución clínica [Oral-motor disorders in a pediatric neurology unit. Classification and clinical course]. *Revista de neurología, 47*, 509-516.

Roy, N., Leeper, H. A., Blomgren, M., & Cameron, R. (2001). A description of phonetic, acoustic, and physiological changes associated with improved intelligibility in a speaker with spastic dysarthria. *American Journal of Speech-Language Pathology*, *10*, 274-290.

Schalling, E., & Hartelius, L. (2004). Acoustic Analysis of Speech Tasks Performed by Three Individuals with Spinocerebellar Ataxia. *Folia Phoniatrica et Logopaedica, 56*, 367-380.

Van der Merwe, A. (1997). A theorical famework for the characterization of pathological speech sensorimotor control. In M.R. McNeil (Eds.), *Clinical management of sensorimotor speech disorders*. New York, New York: Thieme.

Wang, V. T., Kent, R. D., Duffy, J. R., Thomas, J. E., & Weismer, G. (2004). Alternanting motion rate as an index of speech motor disorders en traumatic brain injury. *Clinical linguistics & phonetics* , *18*, 57-84.

Weismer, G., & Martin, R. (1992). Acoustic and perceptual approaches to the study of intelligibility. In R. D. Kent (Eds.), *Intelligibility in Speech Disorders*. Amsterdam. Philadelphia: Benjamins Pub.

Ziegler, W., & Von Cramon, D. (1986a). Spastic dysarthria after acquired brain injury: an acoustic study. *The British journal of disorders of communication*, *21*, 173-187.

Ziegler, W., & Von Cramon, D. (1986b). Disturbed coarticulation in apraxia of speech: acoustic evidence. *Brain & Languag*e, *2*, 34-47.

Figure 1: Outlined Summary of the sensorimotor model with the pathologies associated with each level of processing

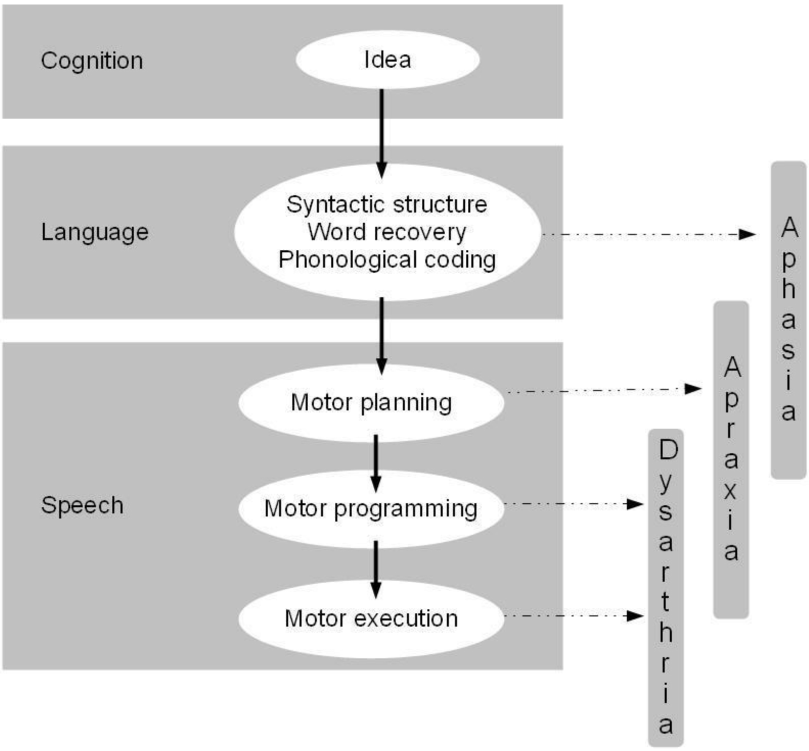
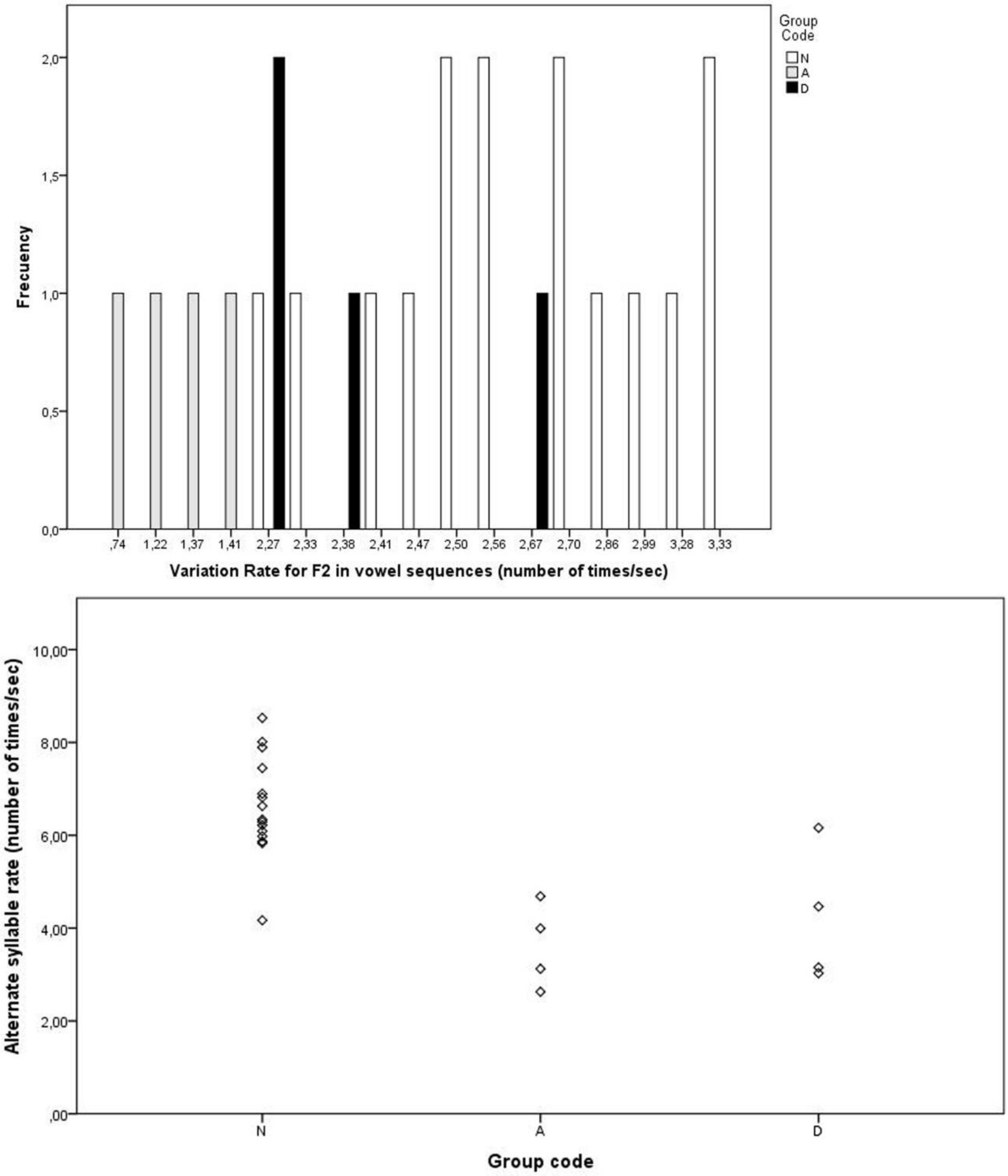


Figure 2: Group Distribution for F2rate (upper graph), PTKavr (central graph) and DKavr (lower graph) variables



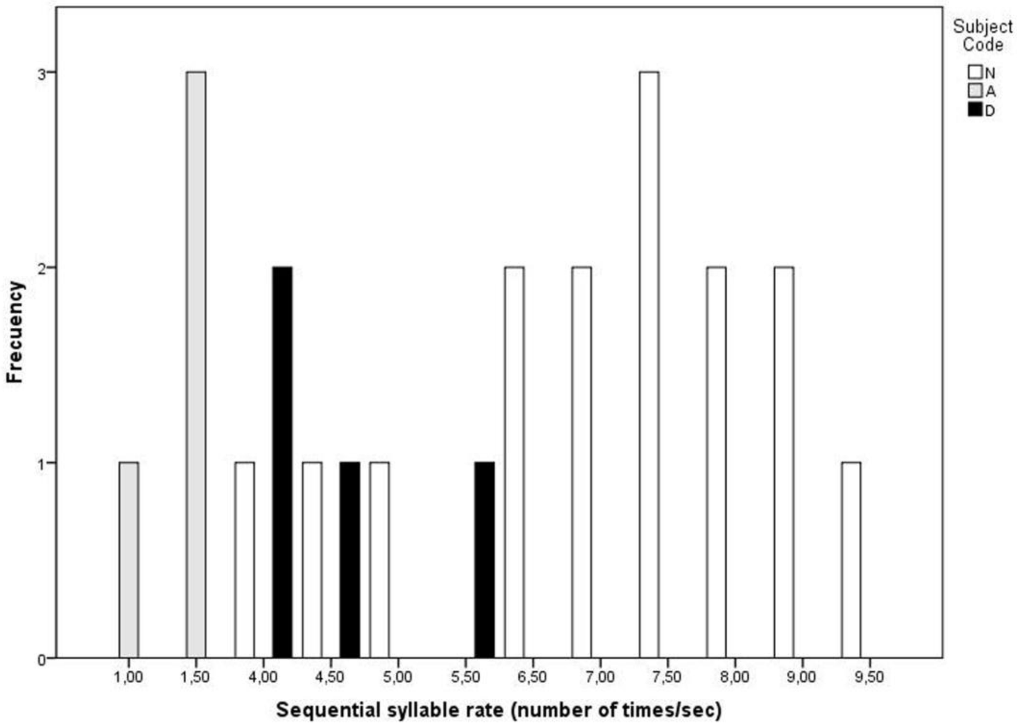

Subject Code
N
A
D
Frecuency
3
2
1
0
1,00
1,50
4,00
4,50
5,00
5,50
6,50
7,00
7,50
8,00
9,00
9,50
Sequential syllable rate (number of times/sec)

Table I. *Descriptive statistics of the variables studied*

Group	F2magn	F2rate	F2reg	DDKavp	DDKavr	PTKavp	PTKavr
A	524.12 (104.66)	1.19 (0.31)	85.78 (4.59)	276.47 (71.68)	3.61 (0.91)	386.04 (52.41)	1.38 (0.25)
D	430.39 (106.93)	2.40 (0.19)	89.35 (9.93)	258.42 (79.68)	4.20 (1.46)	87.72 (18.75)	4.50 (0.71)
N	576.42 (106.04)	2.72 (0.36)	94 (3.80)	159.36 (41.01)	6.60 (1.08)	58.67 (20.05)	7.10 (1.62)

The first number of each column represents the mean. The values within brackets show the standard deviation

Table II. *Analysis of variance by Kruskal-Wallis ranges*

Variable	A Mean Range	D Mean Range	N Mean Range	ChiQ	p
F2magn	11.25	6.00	13.80	4.236	.120
F2rate	2.50	9.25	15.27	12.027	.002*
F2reg	5.00	10.75	14.20	5.975	.050*
DDKavp	19.00	17.75	8.60	10.905	.004*
DDKavr	4.25	6.50	15.53	11.924	.003*
PTKavp	21.50	15.75	8.47	13.142	.001*
PTKavr	2.50	7.88	15.63	13.748	.001*

*significance level *p* < .05 (two-tailed)

Table III. Kolmogorov-Smirnov Analysis

Groups	***Absolute F2rate***	***Z K-S***	***p***	***Absolute F2reg***	***Z K-S***	***P***
A-N	1.000	1.777	.004*	.750	1.333	.057
D-N	.617	1.096	.181	.283	.503	.962
A-D	1.000	1.414	.037*	.500	.707	.699

Groups	***Absolute DDKavp***	***Z K-S***	***P***	***Absolute DDKavr***	***Z K-S***	***P***
A-N	.933	1.659	.008*	.933	1.659	.008*
D-N	.683	1.214	.105	.683	1.214	.105
A-D	.250	.354	1.000	.250	.354	1.000

Groups	***Absolute PTKavp***	***Z K-S***	***P***	***Absolute PTKavr***	***Z K-S***	***P***
A-N	1.000	1.777	.004*	1.000	1.777	.004*
D-N	.800	1.422	.035*	.800	1.414	.037*
A-D	1.000	1.414	.037*	1.000	1.422	.035*

* significance level $p < .05$ (two-tailed)