

Comparing Parkinson's Disease Dysarthria and Aging Speech using Articulation Kinematics

A. Gómez-Rodellar¹, D. Palacios-Alonso², J. Mekyska³, A. Álvarez-Marquina¹ and P. Gómez-Vilda¹

¹*Neuromorphic Speech Processing Lab, Center for Biomedical Technology, Universidad Politécnica de Madrid, Campus de Montegancedo, 28223 Pozuelo de Alarcón, Madrid, Spain*

²*Escuela Técnica Superior de Ingeniería Informática, Universidad Rey Juan Carlos, Campus de Móstoles, Tulipán, s/n, 28933 Móstoles, Madrid, Spain*

³*Department of Telecommunications, Brno University of Technology, Technická 10, 61600 Brno, Czech Republic*

Keywords: Parkinson's Disease, Neuromorphic Speech Processing, Neurotechnology, Aging Speech, e-Health.

Abstract: Speech is being considered a pervasive and costless means to detect and monitor neurodegenerative disease progression. Many different approaches have been reported to differentiate normative subject speech from neurodegenerative patient speech. Most of them are focussed on statistical pattern recognition approaches to improve detection results on a baseline, considering only patient speech and normative controls. The definition of a normative control is not well established in itself, usually being subjects free of any pathology aligned in the same age range as patients. But one question which is not taken into account is the effects of aging in healthy controls, as usually neurodegenerative diseases may include mostly patients affected by certain effects, as dysphonia or dysarthria, as a consequence of aging. The present research introduces a methodology based on information theory to compare the effects produced by aging dysarthria with those due to Parkinson's Disease, using the statistical distribution of speech articulation kinematics as a marker. On the one hand, it may be concluded that articulation kinematics is substantially different for PD and HC with respect to normative subjects. On the other hand, this does not seem to be the case between PD and HC subjects, as these subsets may share some dysarthric features which may be contributed more by aging than by neuromotor degeneration. This differentiation problem needs to be evaluated as well in the case of phonation features, otherwise there will not be full guarantee in using phonation features to assess neuromotor degeneration. In this sense new methodologies have to be designed to distinguish neurodegenerative from aging speech granting better guarantees.

1 INTRODUCTION

Neurodegenerative diseases have a clear effect on speech, both in phonation, articulation, prosody and fluency. Parkinson's Disease (PD) is among the most prevalent neurodegenerative diseases, affecting around 5 million people over age 50 in the 15 world most populated countries in 2005, doubling by 2030 (Dorsey et al., 2007). Typical symptoms associated to PD are bradykinesia, rigidity, freezing of gait, frozen facial mask (hypomimia), postural sway, and distal limb resting tremor, among others (Dauer and Przedborski, 2003; Jankovic, 2008; Sapir, 2014; Anizah et al., 2018). It is well known that speech is strongly related to axial symptoms (Goemann, 2005; Cantiniaux et al., 2010; Ricciardi et al. 2016). Phonation, articulation, prosody and fluency are speech characteristics strongly affected by PD.

Phonation symptoms (musculus vocalis hypotonia), vocal fold unbalance and tremor (altered neuromotor feedback) are some ways in which the neurodegeneration manifests. Articulatory instability is observed mainly as reduced vowel space and vowel centralization distortion (Sapir et al., 2010). Dysprosody and dysfluency are also common symptoms having received attention (Goerman, Blomgren and Metzger, 2010; Martens, et al., 2015). A view of the most comprehensive studies in the field can be found in Tsanas et al. (2010), Rusz et al. (2013), Mekyska et al. (2015), and Brabenec et al. (2017). The objective of this study is to compare articulation in PD patients and aging healthy controls against a normative population, using kinematic features estimated from formants, relying on Information Theory to determine if the steady jaw control necessary to maintain a vowel in its precise articulation place is similarly affected by aging voice

than by PD or not and quantify in thus wat the divergence of pathological and aging articulation with respect to normative subjects, to pinpoint differences and similarities. Recent studies have shown that a relationship can be established between formant-based articulation features and jaw-tongue kinematic activity. This relationship allows to estimate the jaw-tongue kinematics from formant dynamics. The mutual information contents from probability density functions of jaw-tongue kinematic activity estimated from formant-based articulation features may be used as dysarthria markers when comparing PD speech with normative speech (Gómez, P. et al., 2018). The neuromotor character of these markers has been validated by facial surface electromyography and accelerometry (sEMG and 3DAcc), as shown in (Gomez, A. et al., 2018). Building on this relationship, the purpose of the present work is to explore if these dysarthria markers are affected differently in presbyphonic dysarthria (characteristic of aging speech) than in PD dysarthria. The structure of the paper is as follows: Section 2 is devoted to explain the foundations of the jaw-tongue kinematics related with speech articulation modelling, the distributions describing the statistical behaviour of the kinematic variables associated with articulation, and the validation of the kinematic correlates. Section 3 describes the databases of normative, healthy controls and patients, used in the differentiation experiments, and the mutual information estimation methods. Section 4 gives a complete description and discussion of the results produced by the differentiation experiments, both as tables and as figures representing the proximity or distance of each sample to the average of the reference sets (nominally, normative and healthy controls). Finally, section 5 is devoted to highlight the conclusions derived from the presented results.

2 ARTICULATION KINEMATICS

2.1 Jaw-Tongue Biomechanical Model

Speech articulation depends on the position and shape of vocal tract structures, such as the jaw, tongue, lips and velo-pharynx, among others (Buchailleard, Perrier and Payan, 2009). These structures are controlled by different muscles, which are activated by neuromotor pathways from cranial nerves (Jürgens 2002). The acoustical characteristics of speech sounds depends on the positions of these structures and on their dynamic displacement. In the present paper, the role of the jaw-tongue system, as depicted in Figure 1 will

be studied when affected by neuromotor degeneration induced by PD. The jaw-tongue biomechanical system is considered to be a third-order lever with lumped mass load concentrated in the reference point $P_{rJT} \{x_r, y_r\}$ (Hannam et al., 2008). Harmonic oscillation $\{\Delta x_r, \Delta y_r\}$ around the fulcrum (F: attachment to the skull) is assumed under forces acting on this system. A very relevant kinematic correlate of the jaw-tongue neuromotor activity is the Absolute Kinematic Velocity (AKV) of the reference point PrJT:

$$|v_r| = \left[\left(\frac{dx_r}{dt} \right)^2 + \left(\frac{dy_r}{dt} \right)^2 \right]^{\frac{1}{2}} \quad (1)$$

The statistical distribution of the AKV will contribute valuable information in characterizing unstable articulation, as explained in the sequel.

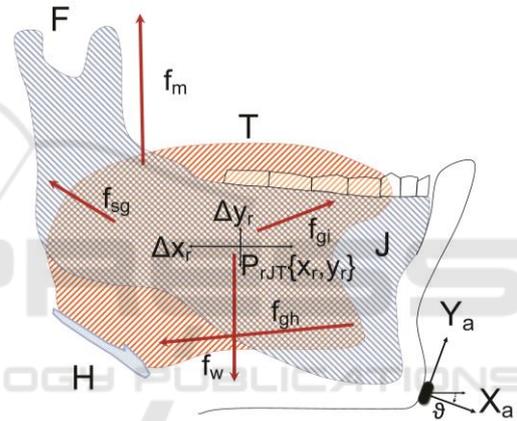


Figure 1: Jaw-Tongue Model. F: Fulcrum; T: Tongue; J: Jaw bone; H: Hyoid bone; f_{sg} : stylo-glossus force; f_m : masseter force; f_{gi} : glosso-intrinsic forces; f_{gh} : genio-hyoid force; f_w : gravity; X_a , Y_a : accelerometer normal and tangential; Δx_r , Δy_r : horizontal and vertical displacements of the reference point (P_{rJT}) in the sagittal plane.

2.2 A Kinematic Articulation Correlate

The methodology of this research is based on representing speech articulation kinematics (positions, speeds, forces and accelerations) by means of acoustically-derived information (speech formants; Dromey, Jang and Hollis, 2013). An important question on the use of kinematic features derived from acoustic correlates (the first and second formants: F_1 and F_2) is to which extent formant dynamics can be related to articulation kinematics (positions and velocities of the jaw-tongue centre of masses). The assessment of the AKV as a reliable kinematic correlate of articulation is carried on the multi-signal recording framework described in

Figure 2. The experimental validation of using acoustic information (formant-based dynamics) to represent articulation kinematics was based on a diadochokinetic exercise, consisting in the fast and continuous repetition of the diphthong [aj:], at a rate of 2-3 repetitions per second. Inverse adaptive filtering was used to estimate the vocal tract transfer function from running speech in real time (Deller, Proakis and Hansen, 1993). F_1 and F_2 are evaluated from the vocal tract transfer function obtained from inverse filtering. Surface electromyography on the masseter (sEMG) and three-channel accelerometry (3DAcc) were recorded synchronously with speech. Sampling rates of sEMG and 3DAcc were equalized to 500 Hz, as well as formant estimates.



Figure 2: Recording set-up for Signal acquisition of speech, accelerometry and surface electromyography (sEMG).

The validation of formant dynamics to represent kinematic variables was based on linear regression according to the following relational chain: surface electromyography (sEMG) is related to the force on the masseter (f_m), which on its turn is related to vertical acceleration (y_{Acc}), resulting in vertical displacement (Δy_r), changing the vertical articulation position, which induces changes in the first two formants ($\Delta F_1, \Delta F_2$). The results of regression studies among the different dynamic variables are given in Table 1.

Table 1: Regression results for the diadochokinetic validation exercise. r: correlation coefficient; p: p-value; S: Spearman’s coefficient; P: Pearson’s coefficient.

Correlation	r (S)	p (S)	r (P)	p (P)
Δy_r vs f_m	0.83	<0.001	0.81	<0.001
ΔF_1 vs Δy_r	-0.89	<0.001	-0.89	<0.001
ΔF_2 vs Δy_r	0.78	<0.001	0.79	<0.001

The correlation between the masseter force estimate from sEMG (f_m) and the vertical displacement of the reference point (Δy_r) is high and statistically relevant (0.83/0.81), showing that a strong relationship exists between neuromotor

activity and movement, as expected. The correlation between vertical displacement (Δy_r) and formant changes are also high and relevant, stronger and counter-related with respect to ΔF_1 (-0.89), than with respect to ΔF_2 (0.78/0.79). These results are aligned with the relationship between the variable controlling the phonation opening (Δy_r) and the variation of the first formant (ΔF_1). Once the relationship between kinematics and acoustics has been established and validated, the displacement of the reference point of the jaw-tongue system when observed over time could be described from an estimate of (1) as:

$$|\hat{v}_r| = \left[B_1 \left(\frac{dF_1}{dt} \right)^2 + B_2 \left(\frac{dF_2}{dt} \right)^2 + B_{12} \frac{dF_1}{dt} \frac{dF_2}{dt} \right]^{\frac{1}{2}} \quad (2)$$

where F_1 and F_2 are the first two formants, and B_1, B_2 and B_{12} are quadratic scaling factors relating movement and acoustics (Gómez, A., et al., 2018).

The distribution of the AKV values as a probability density function (AKV pdf) gives a full statistical description of the jaw-tongue kinematics, and of the kinetic energy which is involved in speech production. The shape of the AKV probability density function will be that of a χ^2 (Chi-square) distribution with two degrees of freedom, which is typically associated with thermodynamic processes, justifying the use of the term “low articulation temperature” associated to hypokinetic dysarthria, an example of these distributions shown in Figure 3.

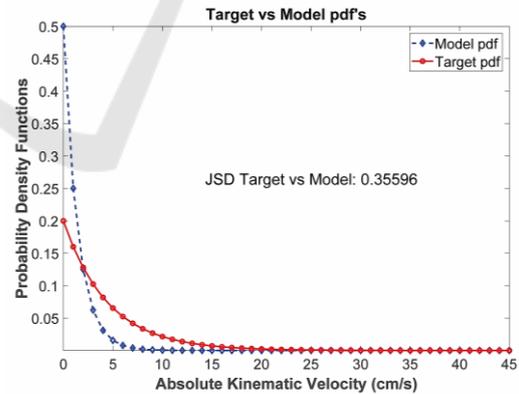


Figure 3: Two ideal probability density functions associated to the AKV in terms of their respective “temperatures”. The model distribution would be associated to the AKV pdf of a maintained vowel from an idealized normative speaker, whereas the target distribution is the typical behaviour of a vowel from a PD patient.

It must be considered that the behaviour of the AKV pdf is quite different according to the kinematic study being carried on. When steady vowels are

produced, as in single maintained vowel exercises, it is expected that a normative speaker would keep a highly stable jaw-tongue position (low temperature) with most of the absolute velocities under a given value (dash-diamond curve), whereas the PD patient will produce unstable oscillations of the articulation point (high temperature) extending along the horizontal axis (full-bullet curve). The situation in running speech, where wide oscillations of the reference point will be expected is to be the opposite: the normative speaker will produce wider and faster oscillations (higher temperature) than the PD patient affected by hypokinetic dysarthria (lower temperature). This fact points to a complete different strategy in testing sustained vowels than in running speech or dyadochokinetic exercises.

3 MATERIALS AND METHODS

3.1 Patient Data Sets

In the present study the articulation stability in maintained vowels has been used to assess the capability of these tests in differentiating the behaviour of PD patients from healthy controls within the same age range, when compared with a normative reference set considered the *golden rule* in maintained vowel phonation. For such, a three band study has been conducted in terms of the mutual information between AKV pdf's from PD patients and paired healthy controls, and with respect to normative speakers, using correlation results from confronting the three sets of speakers among themselves in terms of Jensen-Shannon Distance (JSD). Estimates of the AKV pdf have been used to evaluate the JSD between two different distributions. Vowel utterances [a:, i:, u:] from 8 male and 8 female PD patients randomly selected from male and female databases within an age range of 66.3 ± 8.6 and 69 ± 7.7 years (respectively) have been processed and statistically modelled to produce a PD database (MPD from male subjects, and FPD for female ones). Similar vowel utterances from another set of 8 male and 8 female control subjects randomly selected from male and female databases within an age of 65.6 ± 8.9 and 61.8 ± 9.1 years old (respectively) have also been processed and statistically modelled to produce a healthy control database (MHC from male subjects and FHC from female ones). Recordings were taken at 16 kHz and 16 bits. The database (PARCZ) was collected at St. Anne's University Hospital in Brno (Czech Republic), including also demographic and clinical information from each patient as gender, age, time

since first diagnosis, scores of the Unified Parkinson's Disease Rating Scale, part III (UPDRS-III: motor examination), and part IV (UPDRS-IV: complications of therapy), freezing of gait questionnaire (FOG-Q), non-motor symptoms scale (NMSS), REM sleep disorders (RBDSQ), minimal state examination (MMSE), Addenbrooke's cognitive evaluation revised (ACE-R), Beck depression inventory (BDI), faciokinesis and phonorespiratory competence. All patients signed an informed consent form that was approved by the local ethics committee. The speakers extracted from the PARCZ database are PD patients with code P1xxx (females) and P2xxx (males), and paired healthy controls with code K1xxx (females) and K2xxx (males), as described in Table 2.

Table 2: PD patient and HC subject set lists (PD: PD patient subject; HC: healthy control subject; UPDRS: Evaluation according to UPDRS-III scale).

Code	Gender	Age	Cond	UPDRS
K1003-aiu	F	63	HC	-
K1004-aiu	F	65	HC	-
K1005-aiu	F	59	HC	-
K1006-aiu	F	64	HC	-
K1007-aiu	F	59	HC	-
K1012-aiu	F	67	HC	-
K1017-aiu	F	61	HC	-
K1018-aiu	F	45	HC	-
K2001-aiu	M	59	HC	-
K2002-aiu	M	68	HC	-
K2009-aiu	M	68	HC	-
K2010-aiu	M	83	HC	-
K2011-aiu	M	55	HC	-
K2013-aiu	M	54	HC	-
K2014-aiu	M	62	HC	-
K2015-aiu	M	76	HC	-
P1006-aiu	F	59	PD	24
P1007-aiu	F	76	PD	55
P1008-aiu	F	78	PD	23
P1020-aiu	F	64	PD	8
P1021-aiu	F	65	PD	5
P1022-aiu	F	72	PD	6
P1025-aiu	F	64	PD	8
P1026-aiu	F	76	PD	12
P2005-aiu	M	46	PD	25
P2009-aiu	M	66	PD	14
P2010-aiu	M	66	PD	39
P2012-aiu	M	71	PD	35
P2017-aiu	M	63	PD	19
P2018-aiu	M	63	PD	32
P2019-aiu	M	73	PD	12
P2023-aiu	M	73	PD	13

Finally, 8 male and 8 female subjects have been randomly selected from a normative database recorded at Hospital Gregorio Marañón, of Madrid, Spain, within an age range of 34 ± 12.95 and 37 ± 13.37 (years) respectively. The list of subjects is given in table 3.

Table 3: Normative subject set (NS).

Code	Gender/Age	Code	Gender/Age
N1004-aiu	M/23	N1105-aiu	F/43
N1005-aiu	M/21	N1108-aiu	F/22
N1008-aiu	M/45	N1112-aiu	F/20
N1009-aiu	M/33	N1116-aiu	F/45
N1011-aiu	M/49	N1117-aiu	F/25
N1018-aiu	M/29	N1120-aiu	F/33
N1020-aiu	M/35	N1121-aiu	F/57
N1026-aiu	M/39	N1125-aiu	F/38

3.2 Data Processing

The methodology proposed in the present study is based on the mutual information between two given probability density functions, $p(x)$ and $q(x)$ estimated as a Jensen-Shannon Divergence (Endres and Schindelin, 2003):

$$D_{JS} = \frac{D_{KL}(p|m) + D_{KL}(q|m)}{2} \quad (3)$$

where DKL is a modified version of Kulback-Leibler's Divergence (Salicrú et al., 1994; Georgiou and Lindquist, 2003) expressed as:

$$D_{KL}(p|q) = \int_0^\infty p(x) \text{abs} \left\{ \log \frac{p(x)}{q(x)} \right\} dx \quad (4)$$

and $m(x)$ is the average of $p(x)$ and $q(x)$. In the present case, the probability functions $p(x)$ and $q(x)$ are defined in the positive part of the real axis ($x \geq 0$). Jensen-Shannon's Divergence is symmetrical with respect to $p(x)$ and $q(x)$, and it is normalized to the interval $[0, 1]$, a feature which is very helpful in implementing clustering and classification. The following procedure is used to estimate the JSD's between the PD set, the HC set and the NS set using their AKV pdf's:

- Recordings of the vowel set [a:, i:, u:] were downsampled to 8 kHz.
- The vocal tract transfer function of the speech segment was evaluated by an 8-pole adaptive inverse lattice-ladder filter (Deller, Proakis and Hansen, 1993) with a low-memory adaptive step to grasp fine time variations. A complete description of the adaptive filtering details can be found in Gómez et al. (2009).

- The first two formants were estimated by evaluating the maxima and slenderness of the vocal tract transfer function (LP spectrogram). The formant estimation resolution used was 2 Hz. Formants were estimated every 2 ms.
- The derivatives of the first two formants were used to estimate the AKV following (2).
- The probability density function of the AKV was estimated from the normalized histogram of counts on the definition interval of the AKV (in this case $0 \leq |v_r| \leq 45$ cm/s).
- The histograms were used to estimate probability density functions by Kolmogorov-Smirnov approximations (Webb, 2003).
- The average pdf for each subset was estimated. It may be shown that the average of a set of pdf's shares the same properties of individual pdf's. Six average pdf's were estimated: avMNS, avFNS, avMHC, avFHC, avMPD and avFPD, for the respective male and female normative, controls and PD subsets.
- The Jensen-Shannon Divergence between each patient's histogram-derived distribution vs that of the control subject were estimated as by (3)

4 RESULTS AND DISCUSSION

JSD's between avMNS, avMHC and avMPD on one side, and avFNS, avFHC and avFPD were estimated. The divergences of the MPD vs MNS averages are shown in table 4.

Table 4: JSD between male and female subset averages.

Datasets	JSD
avMPD vs avMNS	0.226
avMHC vs avMNS	0.244
avMPD vs avMHC	0.083
avFPD vs avFNS	0.311
avFHC vs avFNS	0.329
avFPD vs avFHC	0.092

The top template in Figure 4 shows the actual appearance of the PD male sample AKV pdf's in dash-red, whereas the NS male AKV pdf's are given in full-blue. It may be easily seen that the NS set is more concentrated towards the vertical axis, most of the distributions having decayed on the interval between $5-10 \text{ cm.s}^{-1}$, whereas the PD set is more spread over, with some activity still seen between $10-30 \text{ cm.s}^{-1}$ and even beyond. The upper-right legend gives the codes of the speaker samples included in the tests. The bottom template in Figure 4 gives the

average pdf's of the samples in the top template. The different spreads of both average pdf's may be clearly seen now. The central legend gives the JSD between both sets, as well as the results of the p-values after Student's, Kolmogorov-Smirnov's, and Mann-Whitney's tests on Target and Model sets rejecting the null hypothesis of equal means. The AKV pdf's from samples in the male and female subsets have been obtained to be compared against the respective

normative and control subset averages (avMNS, avMHC, for male samples, and avFNS and avFHC, for female samples). Table 5 gives the JSD for each sample. It may be seen that the divergence of the male PD and HC average pdf's with respect to the normative one (MPD vs MNS and MHC vs MNS) is quite similar and larger than when comparing PD and HC (MPD vs MHC).

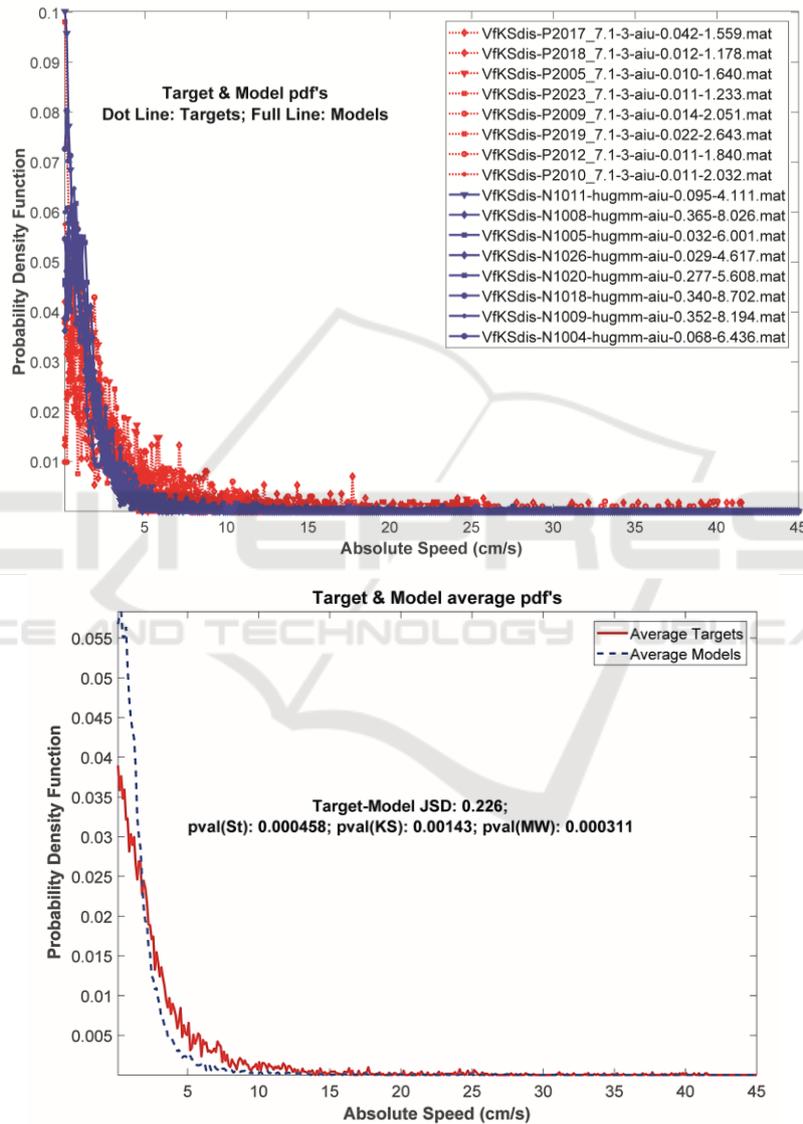


Figure 4: Top: AKV pdf's of the PD male subset (dash-red lines) and the NS male subset (full-blue lines). The normative subset is confined to lower absolute values than the PD subset. Bottom: Averages of normative (dash-blue) and PD (full-red) pdf's, showing the same behaviour. The JSD divergence between avMPD and av1MNS (0.226), and the p-values rejecting the equal mean hypothesis by t-Student, Kolmogorov-Smirnov and Mann-Whitney tests are given in the middle.

Table 5: JSD's between PD, healthy control and normative sets with respect to normative and control averages.

MPD	avMNS	avMHC	MHC	avMNS	avMHC	MNS	avMNS
P2005-aiu	0.30570	0.18871	K2001-aiu	0.24403	0.16737	N1004-aiu	0.074096
P2009-aiu	0.23901	0.14816	K2002-aiu	0.28961	0.15321	N1005-aiu	0.103380
P2010-aiu	0.13407	0.18628	K2009-aiu	0.32015	0.17684	N1008-aiu	0.124710
P2012-aiu	0.20950	0.21612	K2010-aiu	0.19482	0.16206	N1009-aiu	0.075808
P2017-aiu	0.40723	0.27555	K2011-aiu	0.31634	0.19989	N1011-aiu	0.153810
P2018-aiu	0.32686	0.18397	K2013-aiu	0.35297	0.17601	N1018-aiu	0.086608
P2019-aiu	0.23515	0.14976	K2014-aiu	0.23895	0.14632	N1020-aiu	0.088998
P2023-aiu	0.24250	0.23716	K2015-aiu	0.21416	0.17490	N1026-aiu	0.090407
FPD	avFNS	avFHC	FHC	avFNS	avFHC	FNS	avFNS
P1006-aiu	0.38868	0.18533	K1003-aiu	0.37033	0.17377	N1105-aiu	0.072714
P1007-aiu	0.25251	0.19843	K1004-aiu	0.34321	0.16863	N1108-aiu	0.093113
P1008-aiu	0.49834	0.27077	K1005-aiu	0.23298	0.19368	N1112-aiu	0.115570
P1020-aiu	0.21117	0.22827	K1006-aiu	0.47693	0.24286	N1116-aiu	0.120860
P1021-aiu	0.37974	0.17551	K1007-aiu	0.39329	0.15067	N1117-aiu	0.110290
P1022-aiu	0.28630	0.19981	K1012-aiu	0.26424	0.18785	N1120-aiu	0.104500
P1025-aiu	0.34688	0.17790	K1017-aiu	0.29882	0.17209	N1121-aiu	0.165080
P1026-aiu	0.29915	0.20053	K1018-aiu	0.44165	0.21344	N1125-aiu	0.076065

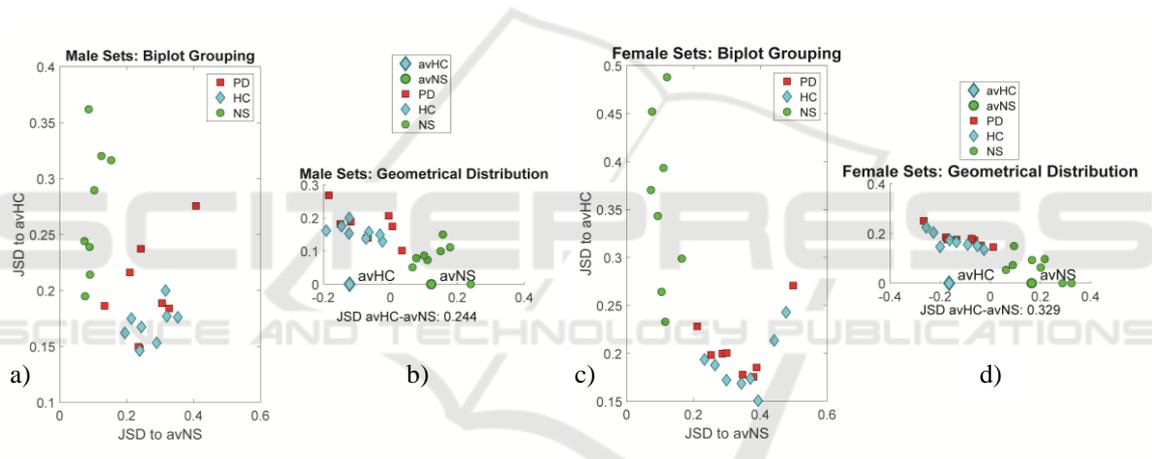


Figure 5: Distribution of each male sample with respect to the male HC and NS averages. a) Bi-plot in terms of JSD respect to the normative and healthy control sets (males). b) Geometrical distribution with respect to the centroids avHC and avNH. c) and d) Similar representations for the female sets. Red squares: PD samples. Blue diamonds: HC samples. Green bullets: NS samples.

This is a first advancement on the difficulty of separating subsets which are much closer themselves than with respect to a *golden rule* set as NS. The same observation may be derived for the female subset, where FPD vs FNS and FHC vs FNS are much more divergent than between themselves (FPD vs FHC). It may be seen that the divergences of the PD subsets with respect to the normative set averages are much larger than their divergences with respect to the healthy controls. The divergences of healthy controls with respect to the normative subsets are almost as large than those ones from PD subsets. This observation may indicate that the healthy controls are farther away from normative sets than expected in

terms of articulation kinematics. On the other hand, the normative samples are closer to their average, as expected. These results show that PD samples are clearly diverging from normatives, and to some extent from healthy controls. The question now is if this divergence is statistically significant to assume different information contents among pathologic, control and normative subsets. The graphical representation of the divergence among the different subsets may help in understanding better the relationships involved. The divergence between each sample in the study and the normative and control subset averages (avMNS and avMHC) is represented graphically in the plots shown in Figure 5.

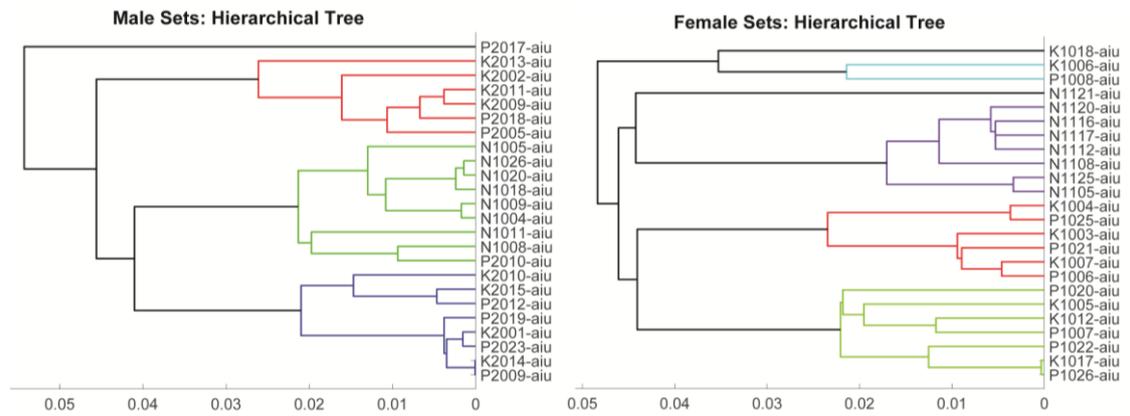


Figure 6: Hierarchical clustering of speaker samples by JSD to their respective average normative distribution. Left: Male subsets. Right: Female subsets.

It may be seen that the distance of the normative set NS with respect to its centroid avNS is small, but it is significantly larger with respect to the healthy control centroid avHC, both for male and female samples. On its turn, the situation of samples from healthy controls and PD patients is the reverse, they are far from the normative centroid avNS, but at short distance from the healthy control average avHC. This reflects the difficulty in separating both sets of samples as far as vowel sustained dysarthria is concerned. This situation is also illustrated by hierarchical clustering in terms of each sample JSD with respect to their respective average normative sets (avMNS and avFNS), as reflected in figure 6.

The male set is separated into four main clusters, one including a single sample, the second one including six ones in which healthy controls are a majority of 4/2 (in red), the third one grouping normative samples and a PD sample (in green), and a fourth one integrated by healthy controls and PD samples in equal proportion of 4/4 (in blue). The situation for the female subsets is a bit more complex. There are also four main clusters, the first one composed of three samples, two healthy controls and a PD sample (black and cyan), the second one integrated by the whole normative subset (one in black and seven in purple), a third one integrated by three healthy controls and three PD samples (in red), and a fourth one including three healthy controls and four PD samples. Again, the similarity between healthy controls and PD patients is manifested in sustained vowel dysarthria. This situation is confirmed by t-Student, Kolmogorov-Smirnov and Mann-Whitney tests considering equal-means null hypothesis conditions. As it may be seen in 0, the tests including healthy controls and PD samples vs the normative set reject the null hypothesis, both for

males and females, pointing to strong differences with respect to normative speakers as far as vowel dysarthria is concerned. But the situation is completely different when PD sets are compared with healthy controls. Whereas for male sets t-Student and Kolmogorov-Smirnov tests reject the null hypothesis, Mann-Whitney fails in doing so. In the case of female sets, all the mentioned tests fail in rejecting the null hypothesis, pointing to more similarities than expected between healthy controls and PD patients. Aging voice could be behind the problem.

Table 6: Estimated p-values from inter-subset tests. t-St: t-Student; KS: Kolmogorov-Smirnov; MW: Mann-Whitney. The cases where the null hypothesis is not fulfilled under a 5% level are printed in bold.

Datasets	t-St	KS	MW
MPD vs MNS	0.000458	0.001430	0.000311
MHC vs MNS	0.000017	0.000156	0.000155
MPD vs MHC	0.0229	0.0497	0.1300
FPD vs FNS	0.000249	0.000156	0.000155
FHC vs FNS	0.000062	0.000156	0.000155
FPD vs FHC	0.366	0.188	0.195

These results show that PD datasets are clearly separable from normative and healthy controls at highly significant levels, both in the case of male and female subsets. HC are also significantly different than normative sets. But separability between PD and age-paired HC is not granted under acceptable standards, possibly due to the aging characteristics of HC articulation kinematics. This is not clear in the male set, where two tests (t-Student and Kolmogorov-Smirnov) avail separability whereas MW does not. But in the female case, the three tests fail in rejecting the null hypothesis, pointing to the difficulty in distinguishing both sets on the basis of articulation

kinematics, a fact which is also observable in figure 6. HC shows a closer kinematic nature with respect to PD, which results in some confusion and separation difficulties. It may be observed that this similarity is of aging nature, i.e., healthiness of healthy controls cannot be assimilated to normative articulation. Age-paired HC show certain similarities with PD patients due to the effects of aging in articulation, although this assumption must be proven. A comparison of PD datasets with respect to normative sets may be not resolving enough, as anticipated by the kinematic analysis of PD and presbyphonic voice. It may be argued that articulation kinematics is not sharp enough to establish this differentiation, but it must be taken into account that articulation instability is quite well modelled by AKV pdf (Gómez, P., et al., 2017). On the other hand, studies based on phonation features, whether linear or non-linear, should be subject to the same three-band tests to ensure that they are sensitive to this separability problem. These considerations raise immediate methodological concerns regarding tests including PD patients and healthy controls paired in age. It is unclear if this separability problem is due to aging voice in healthy controls, and in that case, if distortions found in PD samples could be due also to aging, and not only to pathology. The conclusion is that more tests with larger number of samples should be conducted to confirm or reject this observation, and that sharper methods should be designed, both for the study of vowel and speech dysarthria, as well as for studies involving phonation, classically based on distortion correlates as jitter, shimmer, signal-to-noise and non-linear features. Especial care on this respect should be observed regarding MFCC's (mel-frequency cepstral coefficients), as these features are known to be sensitive both to dysphonia and to dysarthria. In this sense, resolute features are to be sought and tested using three-band benchmarks in the way shown in the present study.

5 CONCLUSIONS

From what has been discussed, the most relevant conclusions to be summarized are the following:

- Paired tests show that articulation kinematics is substantially different for PD and HC with respect to normative subjects.
- This does not seem to be the case between PD and HC subjects, as these subsets may share some dysarthric features which may be due to aging more than to neuromotor degeneration.

- This differentiation problem needs to be evaluated as well in the case of phonation features, otherwise there will not be full guarantee in using phonation features to assess neuromotor degeneration.

As a final remark, it must be stressed that these conclusions are conditioned by the low size of the datasets used, and require further validation with larger number of subjects to be generalized.

ACKNOWLEDGEMENTS

Funded by grants TEC2016-77791-C4-4-R (MINECO, Spain), CENIE_TECA-PARK_55_02 INTERREG V-A Spain – Portugal (POCTEP), 16-30805A (CZ.1.05/2.1.00/03.0072), and LOI401 from the Czech Republic Government.

REFERENCES

- Anizah, S., et al. (2018). Objective Evaluation of Bradykinesia in Parkinson's Disease using Evolutionary Algorithms. *Proceedings of the 11th International Joint Conference on Biomedical Engineering Systems and Technologies (BIOSTEC 2018)*: 63-69. DOI: 10.5220/0006601700630069
- Buchaillard, S., Perrier, P. and Payan Y., 2009. A biomechanical model of cardinal vowel production: muscle activations and the impact of gravity on tongue positioning. *Journal of the Acoustical Society of America*, 126(4): 2033-2051.
- Brabenec, L., et al., 2017. Speech disorders in Parkinson's disease: early diagnostics and effects of medication and brain stimulation, *J. Neural Transm.*, 124(3): 303-334.
- Cantinaux, S., et al., 2010. Comparative analysis of gait and speech in Parkinson's disease: hypokinetic or dysrhythmic disorders? *J. Neurol. Neurosurg. Psychiatry*, 81(2): 177-84.
- Dauer, W. and Przedborski, S., 2003. Parkinson's disease: Mechanisms and models. *Neuron*, 39(6): 889-909.
- Deller J. R., Proakis J. G. and Hansen J. H. L., 1993. *Discrete-Time Processing of Speech Signals*, NewYork, Macmillan.
- Dorsey, E. R., et al., 2007, 'Projected number of people with Parkinson disease in the most populous nations, 2005 through 2030' *Neurology*, 68(5): 384-386.
- Dromey, C., Jang, G. O. and Hollis, K., 2013. Assessing correlations between lingual movements and formants, *Speech Communication*, 55(2): 315-328.
- Endres, D. M. and Schindelin, J. E., 2003. A New Metric for Probability Distributions. *IEEE Trans. on Information Theory*; 49(7): 1858-1860.
- Georgiou T. and Lindquist, A., 2003. Kullback-Leibler Approximation of Spectral Density Functions. *IEEE Trans. on Information Theory*; 49(11): 2910-2917.

- Goberman, A. M., 2005. Correlation between acoustic speech characteristics and non-speech motor performance in Parkinson's disease, *Med. Sci. Monit.*; 11(3): 109–116.
- Goberman, A. M., Blomgren, M., Metzger, E., 2010. Characteristics of speech disfluency in Parkinson disease. *J. Neurolinguistics*, 23: 470-478.
- Gómez, P. et al., 2009. Glottal Source biometrical signature for voice pathology detection. *Speech Communication*, 51: 759-781.
- Gómez, P. et al., 2017. Parkinson Disease Detection form , A. R.Speech Articulation Neuromechanics. *Frontiers on Neuroinformatics*, doi: 10.3389/fninf.2017.00056.
- Gómez, P. et al., 2018. Neuromechanical Modelling of Articulatory Movements from Surface Electromyography and Speech Formants. *International Journal on Neural Systems* (in press), doi: 10.1142/S0129065718500399.
- Gómez A., et al., 2018. Estimating Facial Neuromotor Activity from sEMG and Accelerometry for Speech Articulation. *Proc. of the IEEE Int. Symp. on Medical Measurements and Applications*, 287-292.
- Hannam, A. G., et al., 2008. A dynamic model of jaw and hyoid biomechanics during chewing, *J. Biomechanics*, 41: 1069-1076.
- Jankovic, J., 2008. Parkinson's disease: clinical features and diagnosis, *J. Neurol. Neurosurg. Psychiatry*, 79(4): 368–376.
- Jürgens, U., 2002. Neural pathways underlying vocal control. *Neurosci. and Behav. Rev.* (26): 235-258.
- Mardsen, C. D., 1994. Parkinson's disease. *J. Neurol. Neurosurg. Psychiatry*, 57: 672–681.
- Martens, H. et al., 2015. The effect of intensive speech rate and intonation therapy on intelligibility in Parkinson's disease. *J. Comm. Disorders*, 58: 91.105.
- Mekyska, J., et al., 2015. Robust and complex approach of pathological speech signal analysis, *Neurocomputing*, 167: 94-111.
- Ricciardi, L., et al., 2016. Speech and gait in Parkinson's disease: When rhythm matters, *Park. Relat. Disord.*, 32: 42–47.
- Rusz, J. et al., 2013. Imprecise vowel articulation as a potential early marker of Parkinson's disease: effect of speaking task, *J. Acoust. Soc. Am.*, 134: 2171–2181.
- Salicrú, M., et al., 1994. On the Applications of Divergence Type Measures in Testing Statistical Hypotheses, *J. of Multivar. Anal.* 51(2): 372-391.
- Sapir, S., Ramig, L. O., Spielman, J. L. and Fox, C., 2010. Formant Centralization Ratio: A Proposal for a New Acoustic Measure of Dysarthric Speech, *Journal of Speech, Language and Hearing Research*, 53(1): 114-125.
- Sapir, S., 2014. Multiple factors are involved in the dysarthria associated with Parkinson's disease: a review with implications for clinical practice and research, *Journal of Speech, Language, and Hearing Research*, 57(4): 1330-1343.
- Tsanas, A., et al., 2010. Novel speech signal processing algorithms for high-accuracy classification of Parkinson's disease, *IEEE Trans. on Biomed. Eng.* 59: 1264-1271.
- Webb, A. R., 2003. *Statistical pattern recognition*. John Wiley & Sons.