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# Speech motor control from its impairment: the relevance of studying dysarthria in Parkinson's disease

Dysarthria is a collective name for a group of neurologic motor speech disorders, resulting from central and/or peripheral nervous system abnormalities. Hypokinetic dysarthria in Parkinson's disease presents with prosodic insufficiency, related to a monotony of pitch and intensity, a reduction of accentuation, variable speech rate and possible phoneme imprecision. In most cases, the voice is harsh and breathy. Many patients complain about speech impairments, which affect their communication in daily living activities. There is still a need for further research of dysarthria in Parkinson's disease, for a better understanding of its pathophysiology, treatment response, and relationship with other functions/symptoms. The aim of this paper is to provide an insight into the investigation of dysarthria in Parkinson's disease from two complementary perspectives: from a clinical perspective, to help design patient individualised management; from a neurolinguistics point of view, to help better understand the relative contribution of basal ganglia in speech motor control.

*Keywords*: Parkinson's disease, dysarthria, speech, treatment, challenges.

## 1. *Introduction*

Speech motor control is an important part of communication, and the breakdown of such motor control can result in speech impairment. Motor speech disorders refer to a set of signs affecting the control and production of speech consequent to neurological impairment underlying movement disorders. Therefore, they can be distinguished from higher-order language difficulties (i.e., aphasias) and characterized by an approach that dichotomizes them into two entities: apraxia of speech and dysarthria.

Authors regularly emphasize historical elements, specifying that the term "dysarthria" does not only refer to "arthritic" or supra-laryngeal articulatory disorders (e.g. Auzou, 2007; Duffy, 2007). This was the case for many years; dysarthria was then used to describe a primary disorder of speech articulation (Brain, 1965). Since the early 20th century, several authors (cited in Duffy, 2013) grouped the consequences of lesions of the sensorimotor speech system under the terms either dysarthria, i.e. articulation disorders (with the prefix *-dys* for dysfunction and the Latin root *arthri* for articulation), or anarthria, i.e. absence of articulation, the prefix *-a* being privative (Liepmann, 1913; Head, 1926). This Anglo-Saxon terminology of speech disorders, based on the stance of Darley, Brown & Aronson (1975), has been mainstream since the second half of the 20th century.

Dysarthria is "a collective name for a group of speech disorders resulting from disturbances in muscular control over the speech mechanism due to damage of the central or peripheral nervous system. It designates problems in oral communication due to paralysis, weakness, or incoordination of the speech musculature" (Darley, Brown & Aronson, 1969a: 246). Thus, dysarthria refers to "a group of neurologic speech disorders resulting from abnormalities in the strength, speed, range, steadiness, tone, or accuracy of movements required for control of the respiratory, phonatory, resonatory, articulatory, and prosodic aspects of speech production" (Duffy, 2005: 3). This is a current definition of dysarthria, confined to a neurological origin of the disorder, specific, and excluding mechanically induced impairments.

The multiple and often different uses of the term dysarthria can be confusing, and it remains important to remember that dysarthria is a sign that is part of a broader clinical picture, related to the overall symptomatology of a specific movement disorder, associated with a lesion of the central or peripheral nervous system. Clinically, the concept of arthritic disorder encompasses all supra-laryngeal articulatory disorders, in both adults and children, of neurological (e.g., neurodegenerative disease) or non-neurological origin (e.g., ablative surgery of the tongue). Dysphonic disorders, on the other hand, include all forms of laryngeal dysfunction, regardless of their origin. This dissociation between conceptual and clinical terminology would require interdisciplinary consensus to clarify certain aspects of this terminology; in fact, the term "neurodysarthrophony" frequently appears to account for both articulatory and phonatory disorders related to Parkinsonian dysarthria (Ackermann & Ziegler, 1989; Klostermann, Ehlen, Vesper, Nubel, Gross, Marzinzik, Curio & Sappok, 2008), or even the term "neuroarthropneumophony" (Moreau, Pennel-Ployart, Pinto, Plachez, Annic, Viallet, Destée & Defebvre, 2011), used since 1957 but rarely used since (Grewel, 1957). The term "neurodysarthrophony" might be the one that most precisely defines the current concept of dysarthria, i.e., an arthritic (articulatory) and/or pneumo-phonatory dysfunction of neurological origin. The terminological orthogonality between dysarthria/dysphonia can be problematic because, while the term dysphonia is identified as a voice disorder, dysarthria is often mistakenly equated with an exclusive arthritic disorder, an erroneous analogy since articulatory disorders resulting, for example, from ablative orofacial surgery do not fall within the scope of dysarthria (Pinto & Ghio, 2008).

Among other possibilities, two approaches to examining dysarthria in movement disorders could be considered (Pinto, Chan, Guimarães, Rothe-Neves & Sadat, 2017). On the one hand, one can adopt a disease-based approach, which implies that pathophysiological processes are at the origin of the motor signs that contribute, maybe exclusively, to speech alterations; On the other hand, a neurolinguistics-based approach considers that motor speech disorders are the result of alterations dependent on modifications of linguistic processes that have emerged along the progression of the disease. From the former language-independent perspective (i.e., disease-based), dysarthria needs to be assessed, to be managed. From the latter language-specific perspective (i.e., neurolinguistics-based), dysarthria is a model

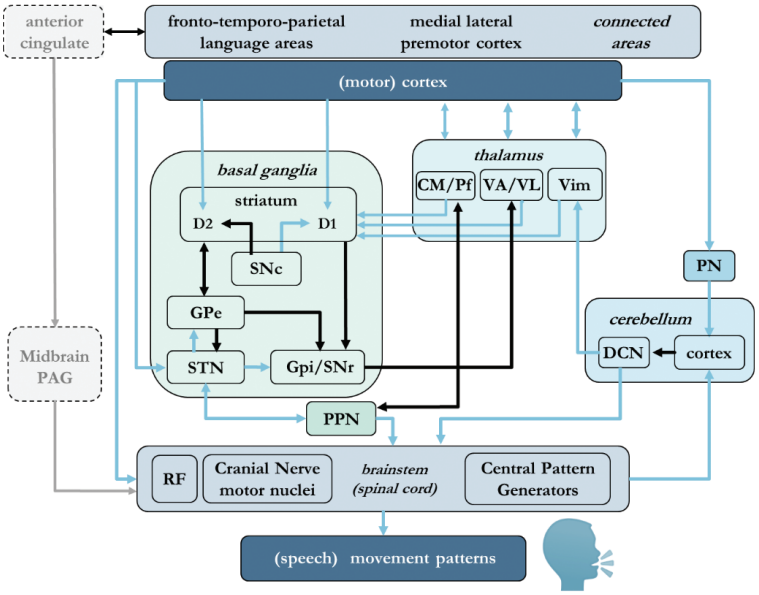
of pathological state which study could help refining neurolinguistics models of speech production. Biasing towards one of the two approaches only would narrow the impact of the findings, and it is reasonable to argue that both approaches are complementary and much needed to provide the most thorough description and analysis of dysarthria understanding.

## 2. *From speech motor control to motor speech disorders*

As a very subjective starting point, we could remember that "speech motor control refers to the systems and strategies that regulate the production of speech, including the planning and preparation of movements (sometimes called motor programming) and the execution of movement plans to result in muscle contractions and structural displacements" (Kent, 2000). A thorough neuroimaging review synthesis provided by Price (2012) summarized from a historical perspective the brain areas showed to be involved in spoken and written language, as well as auditory processes. As regards speech production, named in the review overt articulation, she highlighted cortical regions such as the anterior cingulate cortex, the supplementary motor area and motor/sensorimotor cortices. She also mentioned that the cerebellum, the insula and subcortical areas, such as the putamen and the thalamus, were involved in the timing and control of motor activity. This statement was quite important since mainstream psycholinguistic models, at that time, mainly focused on cortical areas to the detriment of subcortical ones (e.g., Hickock & Poeppel, 2004; Poeppel, Emmorey, Hickok & Pylkkänen, 2012).

A more recent review spotted similar findings, though presented differently (Hertrich, Dietrich & Ackermann, 2020). Notably, the authors provided an illustrative sketch that links brain areas among a "language network" and implicated in several linguistic processes: auditory, motor, lexical, semantic, syntactic, and aspects related to broader social cognition. Again here, the authors highlighted the importance of subcortical areas such as the basal ganglia, the thalamus and the cerebellum. Focusing even more on subcortical contributions to motor speech, Ziegler & Ackermann (2017) summarized nicely the concept in a motor, neurophysiological model that integrates, in addition to an innate limbic vocal system (involving the anterior cingulate and the midbrain periaqueductal grey matter), the two classic motor regulatory loops (the cortico-striatal and cortico-cerebellar ones), which modulate the pyramidal motor tract. Such a model can be implemented in another one (Wichmann, 2019), which is more traditionally considered for the understanding of movement disorders like parkinsonism (Figure 1).

Figure 1 - *A neurophysiological/neurolinguistics view of speech production. Adapted by combining figures from Ackermann (2008), Ziegler & Ackermann (2017) and Wichmann (2019). CM/Pf: CentroMedian-ParaFascicular nuclei complex; DCN: Deep Cerebellar Nuclei; D1/D2: striatal dopamine D1 and D2 receptors; GPe: external Globus Pallidus; GPi: internal Globus Pallidus; PAG: PeriAqueductal Gray; PN: Pontine Nucleus; PPN: PedunculoPontine Nucleus; RF: Reticular Formation; SNc: Substantia Nigra pars compacta; SNr: Substantia Nigra pars reticulata; STN: SubThalamic Nucleus; VA: Ventral Anterior nucleus; VL: Ventral Lateral nucleus; Vim: Ventral intermediate nucleus*




Connecting speech motor control to motor speech disorders would mean trying to complement neurophysiological models by integrating specificities – and dysfunctions – related to speech motor control, or vice-versa, to refine neurolinguistic models by integrating neurophysiological motor control circuitries – and their dysfunctions. It is the objective of many recent neurolinguistics or computational models, such as the ACTion-based model of speech production, speech perception, and speech acquisition (ACT) (Kröger *et al.*, 2012) or the Directions Into Velocities of Articulators (DIVA) model (Guenther, 1994; Golfinopoulos, Tourville & Guenther, 2010; Tourville & Guenther, 2011). Furthermore, as reminded by Hertrich *et al.* (2020), the role of subcortical circuits for speech motor control is known principally from clinical studies on dysarthrias after basal ganglia or cerebellar disorders. There is indeed a very tight connection and a long history between the elaboration of fundamental models of speech motor control and the report of clinical cases.

Different forms of motor speech disorders correspond to different aetiologies and pathophysiologies. They are part of a broader range of motor deficits inherent to a specific clinical picture: the idea is, therefore, to match the specific combination of signs defining the observed motor speech disorder to a precise topology of brain

lesion. Several attempts to classify dysarthrias have been initiated, both benefiting from and encountering challenges in multi- and interdisciplinary approaches: it is indeed difficult to synthesize neuroanatomical, etiological, semiological, and perceptual data into a simple and exhaustive classification. However, it has been acknowledged that the perceptual characteristics of each dysarthria depended more on the location of the neurological lesion, the anatomical substrate of the associated pathology than on its aetiology (Luchsinger & Arnold, 1965).

## 3. *Clinical-perceptual classification of dysarthrias by Darley and colleagues*

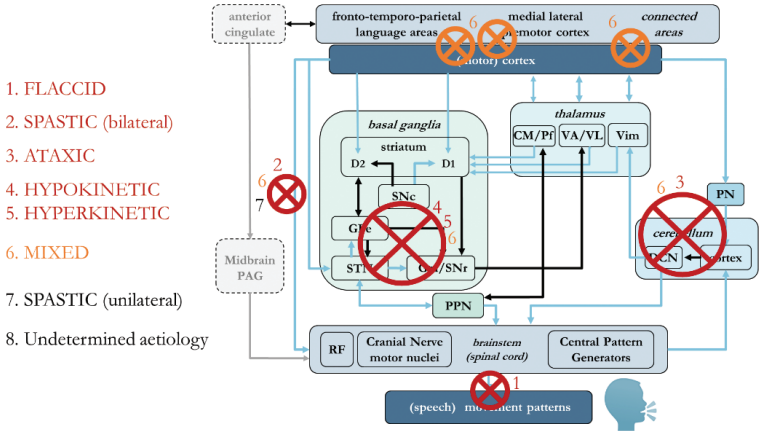
There is no classification of dysarthrias based solely on their semiotic description. The one originating from the Mayo Clinic in Cleveland (USA) remains the most widely recognized and used (Darley *et al.*, 1969a; 1969b; 1975). It offers a pragmatic description based on the grouping of perceptually evaluated anomalies and still represents a consensual, easy-to-understand and practical way to describe speech impairment in movement disorders. The description of six types of dysarthrias laid the groundwork for pathophysiological hypotheses aiming to associate a characteristic speech motor disorder with a precise anatomical lesion (Figure 2). Two additional dysarthria types have been added by Duffy (2005) to this classification: dysarthria resulting from unilateral upper motor neuron disorder (while spastic dysarthria is consequent to bilateral dysfunction), and dysarthria from undetermined aetiology.

### 3.1 Classification procedure

The classification was made from the study of 212 patients distributed into seven pathological groups, each of them meant to represent a typical pathophysiology, associated with one of the six dysarthria types (Figure 2):

- flaccid dysarthria for bulbar palsy (dysfunction of the motoneuron);
- spastic dysarthria for pseudo-bulbar palsy (dysfunction of the upper motoneuron);
- ataxic dysarthria for cerebellum dysfunctions;
- hypokinetic dysarthria for parkinsonism (basal ganglia dysfunction);
- hyperkinetic dysarthria for both chorea and dystonia (basal ganglia dysfunctions);
- mixed dysarthria for amyotrophic lateral sclerosis, ALS (diffuse brain dysfunctions).

Figure 2 - *A pathophysiological/neurolinguistic view of dysarthrias. Localisations of cerebral lesions/dysfunctions related to the different pathological groups have been identified in the normal functioning model proposed in Figure 1. Such localisations are identified in coloured circles, and associated to dysarthria types*




Among the 212 patients studied, 30 were included in each group, except for the Parkinsonian group which comprised 32 patients. Patients were required to read a short text, the Grandfather passage, and speech productions were recorded. The evaluation consisted of the three authors of the classification (Darley, Brown, and Aronson) paying attention to the characterization of 38 dimensions covering seven speech features: pitch (n=4 dimensions), loudness (n=5), voice quality (n=9), respiration (n=3), prosody (n=10), articulation (n=5), and an "overall" feature (n=2) that provided a general impression of the intelligibility and the "bizarreness" of speech (Table 1).

Table 1 - *Speech and voice features perceptually evaluated by Darley, Brown and Aronson for the classification of dysarthrias*

| Pitch | |
|---|---|
| 1 | Pitch level |
| 2 | Pitch breaks |
| 3 | Monopitch |
| 4 | Voice tremor |

| Loudness | |
|---|---|
| 5 | Monoloudness |
| 6 | Excess loudness variation |
| 7 | Loudness decay |
| 8 | Alternating loudness |
| 9 | Loudness (overall) |

| "Overall impression" | |
|---|---|
| 37 | Intelligibility |
| 38 | Bizareness |

| Vocal quality | |
|---|---|
| 10 | Harsh voice |
| 11 | Hoarse (wet) voice |
| 12 | Breathy voice (continuous) |
| 13 | Breathy voice (transient) |
| 14 | Strained-strangled voice |
| 15 | Voice stoppages |
| 16 | Hypernasality |
| 17 | Hyponasality |
| 18 | Nasal emission |

| Respiration | |
|---|---|
| 19 | Forced inspiration-expiration |
| 20 | Audible inspiration |
| 21 | Grunt at end of expiration |

| Prosody | |
|---|---|
| 22 | Rate |
| 23 | Phrases short |
| 24 | Increase of rate (in segments) |
| 25 | Increase of rate (overall) |
| 26 | Reduced stress |
| 27 | Variable rate |
| 28 | Intervals prolonged |
| 29 | Inappropriate silences |
| 30 | Short rushes of speech |
| 31 | Excess and equal stress |

| Articulation | |
|---|---|
| 32 | Imprecise consonants |
| 33 | Phonemes prolonged |
| 34 | Phonemes repeated |
| 35 | Irregular articulatory breakdown |
| 36 | Vowels distorted |

Some criteria are not discriminating at all: a hoarse voice, monotony, and imprecision of consonants describe most types of dysarthria. Conversely, excessive intensity variations are typical of dystonic voice, as are vocal arrests; Parkinsonian speech, on the other hand, exhibits very characteristic paroxysmal accelerations. By correlating certain deviant criteria referring to the same physiological function, eight syndromic clusters were described, which characterise the pathological groups considered in the study and their associated dysarthrias (Table 2). One may question why, in Parkinson's disease, for example, only the syndromic cluster of prosodic incompetence has been highlighted. This classification certainly shows limitations (see below § 3.3).

### 3.2 Brief description of dysarthrias

Flaccid Dysarthria is characteristic of the bulbar pathological group. It results from damage to the final common motor pathway, which includes peripheral nerves (arising from the corticobulbar and/or corticospinal tracts), the neuromuscular junction (e.g., as in myasthenia), and/or the speech effector muscles (e.g., as in myotonic dystrophy of Steinert, oculopharyngeal myopathy, Duchenne's dystrophy). It is characterised by low tone, paralysis, or hypotonia.

Spastic Dysarthria is the pseudobulbar one, resulting from bilateral damage to the first neuron connecting the motor cortex to the brainstem relay. The corticobulbar tract is thus bilaterally affected, with the conditions mainly related to lacunar states (small strokes). The term "spastic" in this context refers to an exaggerated and permanent increase in resting muscle tone. Pseudobulbar syndromes appearing in other pathologies (e.g., ALS, multiple sclerosis [MS], progressive supranuclear palsy [PSP]) and involving impairment of speech production from more diffuse lesions lead to mixed dysarthria (cf. below).

Ataxic Dysarthria results from damage to the cerebellum and/or cerebellar pathways. Lesions can be of various natures (degenerative, vascular, traumatic, neoplastic, inflammatory, toxic, or metabolic). The lack of coordination of voluntary movements, characteristic of cerebellar deficit, is expressed notably in articulatory imprecision and prosodic excesses.

Hypokinetic Dysarthria results from degenerative damage to a part of the basal ganglia, causing a more global dysfunction of the motor regulation loop involving the basal ganglia. Akinesia (inability to move or slow initiation of movement), bradykinesia (slow execution of movement), and hypokinesia (movements of low amplitude) characterize the pathological group centred around Parkinson's disease (PD). Dysarthrias of other parkinsonian syndromes (multiple system atrophy [MSA], PSP, Lewy body dementia [LBD], or even corticobasal degeneration [CBD]) share some hypokinetic characteristics with other types of dysarthria (hyperkinetic, spastic, etc.), often taking on a mixed character.

Hyperkinetic Dysarthria is characteristic of the dystonia and choreoathetosis pathological groups. Dystonia, properly speaking, refers to rather slow movements and twists leading to abnormal postures. Dystonia can be focal (laryngeal, oro-mandibular, cervical), more extensive (generalized or segmental dystonia), primary,

or secondary (resulting from neuroleptic intake, perinatal anoxia, post-traumatic, post-stroke, metabolic). Abnormal choreiform movements are involuntary, rapid, and non-stereotyped. Their aetiology can be degenerative, as in the case of Huntington's disease, toxic, or post-infectious. Hyperkinetic dysarthria also results from dysfunction of the basal ganglia, involving, as the name suggests, the occurrence of large, involuntary, and uncontrollable movements. Spasmodic dysphonia, often associated with essential tremor, is also an example of hyperkinetic dysarthria.

In Darley, Brown, and Aronson's classification, the pathological group that illustrated mixed dysarthria consisted of ALS patients. Mixed dysarthrias combine speech disorders resulting from the involvement of several neurological systems; mixed dysarthric speech is characteristic of pathological groups such as ALS, MS, traumatic brain injuries, but also atypical parkinsonian syndromes (e.g., LBD, MSA, PSP).

Later, two additional categories complemented this classification (Duffy, 2005: 13-14): dysarthrias due to unilateral damage to the first motor neuron, allowing for a distinction concerning a specific hemisphere compared to spastic dysarthria involving bilateral damage; and dysarthrias of undetermined aetiology.

Table 2 - *Syndromic clusters described by Darley, Brown et Aronson (according to Auzou, 2007). AD: Ataxic Dysarthria; ALS: Amyotrophic Lateral Sclerosis; Bulb.: Bulbar; Cereb.: Cerebellar; Park.: Parkinsonism; Chor.: Chorea; Dyst.: Dystonia; FD: Flaccid Dysarthria; HoD: Hypokinetic Dysarthria; HyD: Hyperkinetic Dysarthria; Ps-bulb.: Pseudobulbar; SD: Spastic Dysarthria*

| Pathological group | Bulb. | Ps-bulb. | Cereb. | Park. | Chor. | Dyst. | ALS |
|---|---|---|---|---|---|---|---|
| Dysarthria type | FD | SD | AD | HoD | HyD | | Mixed |
| Articulatory imprecision | | | | | | | |
| Prosodic excess | | | | | | | |
| Prosodic insufficiency | | | | | | | |
| Articulatory and resonatory incompetence | | | | | | | |
| Phonatory stenosis | | | | | | | |
| Phonatory incompetence | | | | | | | |
| Resonatory incompetence | | | | | | | |
| Phonatory and prosodic insufficiency | | | | | | | |

### 3.3 Limits of such dysarthria classification

The classification by Darley aims to be exhaustive but unfortunately does not allow the systematic placement of the observed disorder into a predefined category: some evaluation criteria are non-discriminatory, some pathological expressions take different forms depending on the patients, and the aetiologies are diverse for the same pathology. For example, hypokinetic dysarthria is expected to characterise PD speech, but it has been described, without any precision, from patients suffering from

"parkinsonism". Oppositely, mixed dysarthria has been described while evaluating patients with ALS only, whereas other affections such as atypical parkinsonism are also examples of mixed dysarthria.

A critical view of this classification of dysarthrias was proposed by Auzou (2007), emphasizing its undeniable contribution to the detailed description of dysarthrias but also highlighting the limitations of this classification: difficulty in the differential diagnosis of dysarthrias based on deviant criteria, lack of duplication work, approximate methodologies for constituting pathological groups and clusters, listening conditions that were not blinded to the diagnoses. Nevertheless, even though the results of this perceptual analysis are difficult to reproduce, this evaluation method is still considered the method of choice (Duffy, 2005; 2013). All these considerations pave the way for increasingly numerous studies on dysarthria, whose description and classification are still to be perfected. Over time and despite limitations, including the almost absence of reproducibility, this description by Darley and colleagues remains highly favoured in clinical practice (Kent, Kent, Weismer & Duffy, 2000), at least concerning hypokinetic dysarthria. Auditory-perceptual evaluation remains an important and appropriate clinical tool, as it allows for the description of the multiple dimensions of dysarthric speech: a congruence study among different listening judges using Mayo Clinic criteria strengthens this argument (Bunton, Kent, Duffy, Rosenbek & Kent, 2007).

Certainly, the clinical-perceptive description and classification from the Mayo Clinic have been contextually important from a diagnostic perspective; however, concerning the aetiology and management of these disorders, studying and assessing physiological dysfunctions at each level of production (breathing, phonation, resonance, articulation, prosody) remains crucial for the physiological understanding of dysarthrias. Indeed, the instrumental approach to studying dysarthrias is essential, readily focusing on exploring movement disorders of speech organs: aerodynamic, electromyographic, cineradiographic, kinematic, acoustic measurements, functional brain imaging, or even involving modelling may be useful (Duffy, 2007: 18).

## 4. *Features of dysarthric speech in Parkinson's disease*

In the seminal description of the disease made by Parkinson (1817), who described thoroughly six patients aged between 50 and 72 years old, three of them presented with speech deficits. The words of James Parkinson were the following: "His words are now scarcely intelligible", referring to a marked loss of speech intelligibility; "The power of articulation is lost", suggesting a disorder of supra-laryngeal articulation; and "Speech was very much interrupted" reporting a deficit of fluency and alteration of prosody in its temporal aspects.

### 4.1 The timeline of dysarthria in Parkinson's disease

When speech impairment in PD was reported by James Parkinson, no reference to the timing of appearance was made. It was only mentioned that such impairment occurs "as time and the disease proceed" (Parkinson, 1817: 5). Selby (1968) proposed a caudorostral progression of dysarthria, from the respiratory system to the larynx and the supralaryngeal articulation. Selby reported that "*The initial defect in the untreated patient is a failure to control respiration for the purpose of speech and there follows a forward progression of articulatory symptoms involving larynx, pharynx, tongue and finally lips*". The speech deficits in PD seem to start early in the disease timeline with hypophonia and worsen with time to include dysphonia, dysprosody and supralaryngeal dysarthria. From a functional point of view, speech changes in PD spread from loudness to voice and intelligibility. So dysarthria is not a late symptom in PD, but rather a behavioural marker.

The timeline for dysarthria may be tightly connected to the one proposed by Braak with the stage progression of PD (Braak, Del Tredici, Rüb, de Vos, Jansen Steur & Braak, 2003). Particularly, one may find here some pathophysiological origin of the first stage of dysarthria in PD, so namely hypophonia, with the reported early dysfunction of the vagus nerve. Indeed, vagal innervation is crucial for the diaphragm, the core muscle involved in respiration, and by extension to the respiratory control of speech and speech loudness.

### 4.2 Characteristics of speech in PD: a summary

Table 3 summarizes the ten deviant dimensions identified perceptually by Darley *et al.* (1969a; 1969b) that characterised the most speech in PD patients. Hypokinetic dysarthria involves laryngeal deficits, the voice being harsh, often with a low pitch. Voice breaks, including tremor, can occur, and voice can be also breathy, mainly since vocal folds are quite rigid and patients have difficulties in closing the vocal tract when voicing, implying consequently an air leak that can be heard during speech. A feature that has not been pointed out by Darley and colleagues is the hypophonic nature of PD speech (Canter, 1963). Imprecise articulation contributes to the lack of intelligibility, together with the deficit in respiratory control. From a more suprasegmental perspective, patients have a monotonous voice, use reduced stress and produce short rushes of speech. In other words, speech temporal organisation is disturbed, and speech festination, freezing, or even pseudo-stuttering can also be observed. Several recent reviews can be consulted for further details (e.g., Dashtipour, Tafreshi, Lee & Crawley, 2018; Brabenec, Mekyska, Galaz & Rektorova, 2017; Moreau & Pinto, 2019).

Table 3 - *Deviant dimensions of speech in Parkinson's disease patients, identified perceptually by Darley, Aronson & Brown (1969a; 1969b). Alterations impact prosodic dimensions (in blue), phonatory charateristics (in dark red), temporal organization (in grey) and supralaryngeal articulation deficits (in orange)*

| Rank | Dimension |
|---|---|
| 1 | Monopitch |
| 2 | Reduced stress |
| 3 | Monoloudness |
| 4 | Imprecise consonants |
| 5 | Inappropriate silences |
| 6 | Short rushes |
| 7 | Harsh voice |
| 8 | Breathy voice (continuous) |
| 9 | Low pitch |
| 10 | Variable rate |

## 4.3 The challenge of acoustic automatic analyses of dysarthric speech

Studying dysarthria might start often by identifying the communication deficits reported by the patients, and the consequences of speech impairment in their daily living activities. However, this needs to take into account the contributing factors that may exacerbate speech and communication deficits. It is also of utmost importance to measure the speech impairment per se, and for that, several means are available. Among them, a renewed interest has been paid to automated acoustic analyses in recent years. The strength of such analyses is to relate specific acoustic measures, obtained with dedicated speech tasks, to specific dimensions of speech. Therefore, together with dedicated guidelines for speech recording (Rusz, Tykalová, Ramig & Tripoliti, 2021), automated analyses can help to assess and monitor speech changes across PD. This is of particular interest for PD diagnosis and management, and to disentangle acoustic specificities or ambiguities between PD and atypical parkinsonism.

Some specific signs are often reported when referring to atypical parkinsonism (Mulroy, Stamelou & Bathia, 2019), which includes more bilateral signs, that appear earlier in the course of the disease; axial signs (such as gait disorder, postural instability, dysarthria, dysphagia, neuropsychological deficits, etc.) are often more severe than idiopathic PD, and the response to levodopa treatment is usually reduced. Regarding speech impairment, dysarthria is mixed, with additional spastic or ataxic features to the hypokinetic signs that characterise PD. Clinical assessment may refer to 'growling' dysarthria that can become constant groaning in PSP. In the case of MSA, a high-pitched and squeaky voice can be noted. Some striking abnormalities can be noted: Involuntary productions in PSP, and Stridor (MSA). In CBD, orofacial apraxia is very frequent. Also, in atypical parkinsonism, dysarthria is often associated with dysphagia that is early severe when compared to PD, and with

neuropsychological deficits that encompass language difficulties that can also be present in PD, but in later stages and later on in the progression of the disease. One recent study, for example, confirmed the predominance of hypokinetic dimensions in PD dysarthria, ataxic features in MSA and spastic in PSP (Kowalska-Taczanowska, Friedman & Koziorowski, 2020). As the authors stated, acoustic voice analysis is a very sensitive and non-invasive tool that can provide objective information for the assessment of different speech components. Such data, at some point, can be essential for the differential diagnosis of Parkinsonian syndromes. In the same vein, it is possible to go further, trying to distinguish and quantify specific features of MSAp and MSAc for example. Interestingly, in this study by Rusz, Tykalová, Salerno, Bancone, Scarpelli & Pellecchia (2019), audible inspirations observed in MSAc was the only individual speech pattern that allowed the discrimination of parkinsonian and cerebellar variants, despite strong overlaps between both MSA groups.

## 5. *Effects of treatments on speech in Parkinson's disease*

It is a fact: dysarthria is a symptom difficult to manage in PD (Moreau & Pinto, 2019). Dysarthria is usually considered as a symptom partially responsive to treatments that may, or may not, induce additional speech deficits unrelated to the disease itself, as treatment side effects.

### 5.1 Levodopa and medication strategies

Medication strategies have mitigated effects; dopaminergic medication may improve some speech dimensions but also contribute to the exacerbation of others. When it comes to medication, it is all about disease and treatment durations: the longer, the worse. Speech response to levodopa is variable, and depends on both disease and treatment durations, as well as the stage of the disease (De Letter, Santens, De Bodt, Van Maele, Van Borsel & Boon, 2007; Skodda, Grönheit, Mancinelli & Schlegel 2013). While levodopa can improve some components of articulatory deficits, it is not the case for overall speech intelligibility (e.g., Brabenec *et al.*, 2017). Considering longitudinal studies, speech performance well-reflected improvement/deterioration of motor manifestations in dependence on the L-dopa doses in the early stages of the disease (Rusz, Tykalová, Klempíř, Čmejla & Růžička, 2016), while continuously declined in later stages with poor response to L-dopa (Skodda, Visser & Schlegel 2010).

### 5.2 Neurostimulation

Managing dysarthria following deep brain stimulation is often restricted to avoid a worsening of speech disorders and to hope for potential positive effects on speech dimensions, such as respiratory control and/or loudness (Tsuboi, Watanabe, Tanaka, Ohdake, Yoneyama, Hara, Nakamura, Watanabe, Senda, Atsuta, Ito, Hirayama, Yamamoto, Fujimoto, Kajita, Wakabayashi & Sobue, 2015). Following subthalamic

nucleus deep brain stimulation (STN-DBS), there is a discrepancy between the clinical/neurological assessment of speech, which mainly focuses on intelligibility, and the measurement of physiological markers connected to speech/voice dimensions. While a typical loss of intelligibility (of about 15 %) is found one year post-surgery, improvements in speech sub-components can also be observed, like for loudness (Tsuboi *et al.*, 2015; Tripoliti, Zrinzo, Martinez-Torres, Frost, Pinto, Foltynie, Holl, Petersen, Roughton, Hariz & Limousin, 2011). Lower preoperative speech intelligibility on medication, long disease duration and also medial left hemisphere active electrode contacts were reported as predictive factors of poor speech outcome following STN-DBS (Tripoliti, Limousin, Foltynie, Candelario, Aviles-Olmos, Hariz & Zrinzo, 2014). Stuttering and breathy voice have been attributed to disease progression and unrelated to STN-DBS (Tsuboi *et al.*, 2015), whereas strained voice and spastic-like dysarthria seem to be the result of current diffusion towards the cortico-bulbar tract (Pinto, Gentil, Krack, Sauleau, Fraix, Benabid & Pollak 2005; Tsuboi *et al.*, 2015; Atkinson-Clement, Maillet, LeBars, Lavenne, Redouté, Krainik, Pollak, Thobois & Pinto, 2017). Current diffusing towards cerebello-thalamo-cortical fibres was also considered to alter speech following STN-DBS (Tripoliti *et al.*, 2011; Tsuboi *et al.*, 2015; Atkinson-clement *et al.*, 2017). To improve speech outcome, neurostimulation parameters can be modulated, either by using low-frequency in conventional STN-DBS (Moreau *et al.*, 2011) or by using adaptive DBS (Little, Tripoliti, Beudel, Pogosyan, Cagnan, Herz, Bestmann, Aziz, Cheeran, Zrinzo, Hariz, Hyam, Limousin, Foltynie & Brown, 2016). Also, negative synergistic effects between STN-DBS and levodopa exist, and should be considered in treatment management (Pinto, Ferraye, Espesser, Fraix, Maillet, Guirchoum, Layani-Zemour, Ghio, Chabardès, Pollak & Debû, 2014). STN-DBS effects on speech in PD seem related to subjective cost over-valuation of the actual impairment: in a recent study, we demonstrated that vowel production effort was estimated easier to perform with STN-DBS alone, but harder when associated with levodopa (Atkinson-Clement, Cavazzini, Zénon, Legou, Witjas, Fluchère, Azulay, Baunez, Pinto & Eusebio, 2021). Finally, early STN-DBS may avoid side-effects on speech intelligibility (Pinto, Nebel, Rau, Espesser, R., Maillochon, Niebuhr, Krack, Witjas, Ghio, Cuartero, Timmermann, Schnitzler, Hesekamp, Meier, Müllner, Hälbig, Möller, Paschen, Paschen, Volkmann, Barbe, Fink, Becker, Reker, Kühn, Schneider, Fraix, Seigneuret, Kistner, Rascol, Brefel-Courbon, Ory-Magne, Hartmann, Wojtecki, Fradet, Maltête, Damier, Le Dily, Sixel-Döring, Benecke, Weiss, Wächter, Pinsker, Régis, Thobois, Polo, Houeto, Hartmann, Knudsen, Vidailhet, Schüpbach & Deuschl, 2023): in patients operated at an earlier stage of the disease, STN-DBS did not result in a consistent deterioration in blinded speech intelligibility assessment and patient-reported communication, as observed in studies of more advanced PD.

### 5.3 The importance of speech therapy

More than 90 % of the patients with PD complain about their low voice intensity and poor quality of communication, leading to a dramatic impairment in quality of

life and social isolation (Fox & Ramig, 1997). It has been shown that between 70 and 79 % of patients report a speech impairment (Hartelius & Svensson, 1994; Miller, Noble, Jones., Deane & Gibb, 2010). Among these patients, around 29 % stated that this symptom was the major one among all (Hartelius & Svensson, 1994). Despite that fact, only a few patients were referred to speech and language therapists, with percentages ranging from 1.6 to 4.4 % which seems to represent an underestimated need (Nesbitt & Thompson, 1995; Stewart, 2000; Trail, Fox, Ramig, Sapir, Howard & Lai, 2005; Hartelius & Svensson, 1994; Miller *et al.*, 2010). The question of patient referral to speech therapy remains a hot topic, even if such referral tends to increase in very recent years (Schalling, Johansson & Hartelius 2017) and depend largely on countries and areas. As part of multidisciplinary management (Goldman, Volpe, Ellis, Hirsch, Johnson, Wood, Aragon, Biundo, Di Rocco, Kasman, Iansek, Miyasaki, McConvey, Munneke, Pinto, St. Clair, Toledo, York, Todaro, Yarab & Wallock, 2024), speech therapy ought to be provided earlier and more frequently.

Despite insufficient evidence to support the efficacy of speech and language therapy for dysarthria in PD (Herd, Tomlinson, Deane, Brady, Smith, Sackley & Clarke, 2012), possibly also due to individual differences (Spurgeon, Clarke & Sackley, 2015), notable benefits have been observed (Atkinson-Clement, Sadat & Pinto, 2015) and intensive therapy demonstrated its ability to significantly and consistently improve dysarthria (Atkinson-Clement *et al.*, 2015; Ramig, Halpern, Spielman, Fox & Freeman, 2018).

## 6. *Future challenges*

Motor impairment in PD speech is not isolated. Dysarthria is also the result of auditory deficits, which appear to be quite specific in PD (Vitale, Marcelli, Allocca, Santangelo, Riccardi, Erro, Amboni, Pellecchia, Cozzolino, Longo, Picillo, Moccia, Agosti, Sorrentino, Cavaliere, Marciano & Barone, 2012; Vitale, Marcelli, Abate, Pianese, Allocca, Moccia, Spina, Barone, Santangelo & Cavaliere, 2016). This contributes probably to the degradation of auditory feedback processing (Arnold, Gehrig, Gispert, Seifried & Kell, 2014), as part of brain correlates underlying pathomechanisms of PD speech: 1) a striato-prefontal hypo-connectivity and dysfunctional self-monitoring mechanisms, underpinning the diminished motor drive of hypophonia; 2) a reduced external auditory feedback, affecting speech motor representations, and 3) a disturbed modulation of speech routines and affective prosody. These modifications could reflect either compensatory mechanisms or brain dysfunctions of PD speech (Arnold *et al.*, 2014). Higher-order language deficits are still underestimated in PD patients (e.g., Letanneux, Viallet, Velay & Pinto, 2021) and also require future investigation. In addition, rhythmic difficulties, emerging from probable generalised dysrhythmia ought to be considered to participate in the alteration of the temporal organisation of speech (Puyjarinet, Bégel, Gény, Driss, Cuartero, Kotz, Pinto & Dalla Bella, 2019; Puyjarinet, Bégel, Geny, Driss, Cuartero, Cochen De Cock, Pinto & Dalla Bella 2022).

From a theoretical point of view, the cross-linguistic approach to studying dysarthria is concerned with the question of whether disorders of dysarthric speech may differentially disturb the communication in a given language. Thus, dysarthric patients may fail to communicate important meaning differences in a certain language, but not in others in which these contrastive features are not distinctive. Studies examining the impact of speech difficulties from a cross-language perspective are increasingly numerous, providing arguments for language-universal or language-specific aspects of dysarthric speech. So far, cross-language studies suggest more similarities than differences in how dysarthria affects the speech of PD patients speaking different languages (e.g., Whitehill, Ma & Lee, 2003; Whitehill, 2010; Orozco-Arroyave, Hönig, Arias-Londoño, Vargas-Bonilla, Daqrouq, Skodda, Rusz & Nöth, 2016; Rusz, Hlavnička, Novotný, Tykalová, Pelletier, Montplaisir, Gagnon, Dušek, Galbiati, Marelli, Timm, Teigen, Janzen, Habibi, Stefani, Holzknecht, Seppi, Evangelista, Rassu, Dauvilliers, Högl, Oertel, St. Louis, Ferini-Strambi, Růžička, Postuma & Šonka, 2021; Verkhodanova, Coler, Jonkers, Timmermans, Maurits, De Jong & Lowie, 2022; Verkhodanova, Coler, Jonkers & Lowie, 2022). Despite these universals, García, De Leon, Tee, Blasi & Gorno-Tempini (2023) report that linguistic idiosyncrasies have also been found in PD speech (Kim & Choi, 2017; García *et al.*, 2023; García, Bocanegra, Herrera, Moreno, Carmona, Baena, Lopera, Pineda, Melloni, Legaz, Muñoz, Sedeño, Baez & Ibáñez, 2018; Møller, Høj, Østergaard, Wallentin & Højlund, 2023; García & Ibáñez, 2023).

In a recent work (Pinto, Cardoso, Atkinson-Clement, Guimarães, Sadat, Santos, Mercier, Carvalho, Cuartero, Oliveira, Welby, Frota, Cavazzini, Vigário, Letanneux, Cruz, Brulefert, Desmoulins, Pavão Martins, Rothe-Neves, Viallet & Ferreira, 2024), we aimed at identifying the relative contribution of acoustic variables to distinguish PD patients and controls who spoke varieties of two Romance languages, French (from France) and Portuguese (from Portugal). In a large cohort that included 129 PD patients and 124 healthy controls, we showed that acoustic variables that distinguished between speakers of the two languages were mainly related to phonation and voice quality. They contributed less than variables related to pneumo-phonic coordination and articulation rate at discriminating between PD patients and controls, confirming that respiration and diadochokinesis tasks seem the most appropriate to pinpoint signs of dysarthria, which are largely homogeneous and language-universal. In contrast, identifying language-specific variables was less conclusive with the speech tasks and acoustic variables studied here. Further research on that point is still required.

## 7. *Conclusive remarks*

Studying dysarthria starts often by identifying the communication deficits reported by the patients, and the consequences of speech impairment in their lives. It is of utmost importance to measure/document speech with a standardized assessment, early in the patient management, repeatedly across the disease progression, accompanied

by patient-reported assessments of voice/speech and/or communication deficits, and completed by systematic audiometric and higher-order cognitive-linguistic evaluations. Simply reporting global perceptual deficits or intelligibility decline fails to address important deficits that potentially contribute largely to speech impairment. Assessments that aim at providing a holistic evaluation are widely encouraged.

There is still a need for further research of dysarthria in PD, for a better understanding of its pathophysiology, treatment response, and relationship with other functions/symptoms, etc. Language-universal and language-specific dimensions also need to be further explored.

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