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Stuttering and phonetic theory: An introduction

Stuttering has been traditionally defined by making reference to the auditory detection and qualitative assessment of the dysfluencies (some of them being abnormal for number, type, duration and position) and their distributional patterns have been explained by invoking the same dysfunctions of the linguistic representations and processes which generate lapsus and disfluencies in non-stuttering speakers. However, fluency is multidimensional: not only the fluent speech is (relatively) devoid of discontinuities, but it is also produced with a regular rhythmic beat, at fast rate and without an excessive physical and mental effort. As a phonetician, two main questions about stuttering are worthwhile. The first is: "Why would Phonetics be so important in the study of stuttering?" A possible answer could be that Phonetics is at the convergence of different scientific realms, and for such a role it holds a privileged key for unifying and simplifying the understanding of the multidimensional aspects of stuttering (made of sociocultural, psychological, physiological and genetic factors). In order to accomplish a causal function in stuttering, each of these variables must at the end interact with the motor control processes of the speech apparatus, traditionally studied by Phonetics. As to the second question "Why should a phonetician be interested in stuttering?" we could answer that phoneticians could feel a potential attraction towards a speech disorder that selectively affects fluency, in individuals that are judged to be healthy and normally endowed with reference to cognitive and emotional aspects. At the same time, the speech aspects under investigation in stuttering are at the heart of a number of theories of speech production, for what it is about the conceptualization of the time dimension and of the speech variability.

Key words: disfluencies, stuttering, phonetics, psycholinguistics.

1. *Introduction*

Within the realm of Phonetic Sciences, over the last thirty years, an informal community of researchers begun to take shape and grow around a series of conferences known with the general name of "Laboratory Phonology" (the first of them was organized and the proceedings edited by Kingston, Beckman, 1990). This community was later celebrated by three of their most representative founders in a programmatic paper (Pierrehumbert, Beckman & Ladd, 2000), which I will refer to, because it makes clear how phoneticians and people working on stuttering may benefit the ones from the others. What is shared by Laboratory Phonology is the belief that Phonology (a label covering much of the Phonetics, in the authors' point of view) is one of the natural sciences, and that everything in language, including language-specific features and sociolinguistic variation, is part of the natural world. This attitude is contrary to the mind-body dualism, and believes that categorization underlying

the phonological constructs is ultimately based on physical non-linearity grounded in the systems of speech production, speech perception and in the acoustic medium, which, acting together through speech variability, can lead to the emergence of the abstract categories. For this reason, researchers who recognize themselves in Laboratory Phonology's approach make use of laboratory methods to discover and explain the phonetic form of language. According to Lindblom (1995), Phonetics is in a privileged position compared to the other domains of Linguistics (e.g. Syntax) to develop this program. It can invoke a type of knowledge that is relevant to language but which has been acquired independently of it, such as information on general mechanisms of hearing and motor control (i.e., it uses facts and principles whose empirical motivation is independent from the data to be explained). Hence we can see a first difference with those speculating on language "starting from syntax", who believe that languages are constructed in arbitrary and unnatural ways. A second fundamental difference is that, contrary to what is claimed by those who speculate on language starting from syntax, these physiological mechanisms would not be modular, that is, specific to the language of *Homo Sapiens*, as they also serve other physiological functions, evolved naturally from mammals. Starting from the assumption that speech forms are the means the languages provide to make interpersonal communication possible and are therefore public actions (and not mental categories; Fowler, 2014), a theory of Phonology should then explain the properties of these actions; a theory of speech production, how these actions are accomplished; a theory of speech perception, how these actions are perceived. Finally, since humans are subject to natural evolution, the theoretical construction have to explain how these actions could emerge through interpersonal communication exchanges in the child, and how these actions could be reduced or distorted in speech and language disorders (as it happens in stuttering).

The research activity underlying the elaboration of these theories must be based on the cooperation of scientists of different extraction, on a common vocabulary, on the existence of auxiliary theories (such as those relating to the functioning of the instruments used), and on a mathematical formulation, specifically of continuous type (as opposed to discrete). In this perspective, clinical disorders should be considered as an opportunity for basic scientific research: "When basic science research is integrated with knowledge about clinical disorders, the disorders are viewed as natural experiments and opportunities to observe factors that are not normally available for experimental manipulation in humans, and/or that can not be viewed across a sufficient range of settings under normal conditions" (Bernstein, Weismer, 2000: 225).

Stuttering has been defined as a "disorder in the rhythm of speech, in which the individual knows precisely what he wishes to say, but at the time is unable to say it because of an involuntary repetition, prolongation or cessation of a sound" (International Classification of Diseases and Related Health Problems, ICD-9, World Health Organization, 1977: 202). We prefer this somehow dated definition

to the current ICD 10 (2007) definition[1], because it marks explicitly the peculiar nature of stuttering disfluencies, their being "involuntary" and their being perceived by People Who Stutter (henceforth PWS) as a "loss of controls" on the articulators (Perkins, 1990; Perkins, Kent & Curlee, 1991). This single characteristic would suffice to suggest the reference to a motor disorder rather than a language disorder (Perkins, 1990).

Previously, we declared our proximity to Laboratory Phonology, but our inquiry on noble ancestors would be incomplete without quoting Clinical Linguistics, which in Crystal's words "concerns the application of linguistic science to the study of communicative disabilities, as they meet in clinical situations" (Crystal, 1981: 31). According to Ball, Kent (1987), in their preface to the first issue of the *Clinical Linguistics and Phonetics* journal, it covers the application of analytical linguistic/phonetic techniques to clinical problems, or the demonstration of how clinical data could contribute to theoretical issues in Linguistics/Phonetics. Following these reasoning, as a phonetician, two main questions about stuttering are worthwhile. The first is: "Why would Phonetics be so important in the study of stuttering?" A possible answer could be that Phonetics is a borderline discipline, both in the sense that it has a theoretical as well as an applicative character, and in the sense that it is at the convergence of different scientific realms, such as Communication Engineering, Physical Acoustics, Psychology, Anatomy, Physiology, Linguistics, Applied Linguistics, Computer Science and Poetry (Ladefoged, 1988). As a borderline discipline, it holds a privileged key for unifying and simplifying the understanding of stuttering that presents itself as a multidimensional phenomenon, in which sociocultural, psychological, physiological and genetic factors are involved. In fact, one can say that, in order to accomplish a causal function in stuttering, each of those variables must at the end interact with the motor control processes of the speech apparatus, whose defective functioning can be considered the proximal cause of stuttering (Smith, Kelly, 1997).

The second, and specular, question is: "Why should phoneticians be interested in stuttering?" Once again, we could answer that phoneticians could feel attraction towards a speech disorder that selectively affects fluency, leaving essentially intact the syntactic and grammatical structures in individuals that are judged to be healthy and normally endowed with reference to cognitive and emotional aspects (remember the ICD 9 definition which suggests a speech motor, not a language,

[1] F98.5: Other behavioral and emotional disorders with onset usually occurring in childhood and adolescence: "Speech that is characterized by frequent repetition or prolongation of sounds or syllables or words, or by frequent hesitations or pauses that disrupt the rhythmic flow of speech. Minor dysrhythmias of this type are quite common as a transient phase in early childhood, or as a minor but persistent speech feature in later childhood and adult life. They should be classified as a disorder only if their severity is such as markedly to disturb the fluency of speech. There may be associated movements of the face and/or other parts of the body that coincide in time with the repetitions, prolongations, or pauses in speech flow. Stuttering should be differentiated from cluttering (see below) and from tics. In some cases, there may be an associated developmental disorder of speech or language, in which case this should be separately coded under F80."

problem). At the same time, the speech aspects under investigation in stuttering are at the heart of a number of theories of speech production (Weismer, Tjaden & Kent, 1995). In fact, these theories can be affiliated to two great families on the basis of their solutions to the problem of speech timing: the Extrinsic and Intrinsic timing theories (Fowler, 1980), also named, in the terminology of Weismer *et al.* (1995), Translational and Gestural theories, respectively. The first ones postulate the existence of a timer, possibly not specific to the speech mechanism, which puts in sequence a series of discrete and timeless units (i.e. columns of distinctive features); the second ones, bring the timing organization back to the general dynamic property of the articulatory system. The validity of these theories could – at the end – be proved by their power in explaining timing phenomena that are characteristics of the motor speech disorders, like stuttering: articulatory slowness, abnormal scaling (in magnitude) of the articulatory gestures, variability of speech production (across repetitions), abnormal degree of coarticulation (see Weismer *et al.*, 1995; Kent, 1997; Van Lieshout, Goldstein, 2008).

Turning to the ICD approach to speech disorders like stuttering, it is too restricted and has been criticized because its most central assumption is that an underlying clinical entity or medical condition is responsible for stuttering. As a consequence, the goal of Medicine is to intervene on such alterations, which are generally represented by the objective symptoms, i.e. the stuttering disfluencies, and they must be treated independently from any other associated behavioural, psychological and social factors.

These factors are all taken into consideration by another type of broad-based classification system, the International Classification of Functioning, Disability and Health (ICF, WHO, 2001), which starts from assumptions that are radically different from the ICD's ones. ICF could offer an alternative classification for stuttering because emphasis is placed on the fact that disorders like stuttering involve more than just observable behaviours. Specifically, the speaker's experience of stuttering can involve negative emotional, behavioural, and cognitive reactions (both in the speaker and in the communication partner(s)), as well as significant limitations in the speaker's ability to participate in daily activities and a negative impact on the speaker's overall quality of life (Yaruss, Quesal, 2004). According to the ICF's definition, if there is a known sensory, neurological or craniofacial impairment (e.g. hearing loss, cerebral palsy, cleft lip and/or palate), the speech impairment is classified at the Body Structure level. If there is no known cause, the speech impairment is classified at the Body Function level.

The ICD and ICF classification systems are well representative of the philosophical division that, in the word of Tetnowski, Scaler Scott (2010) "is driving both research and clinical interests." [...] This issue "is the dilemma regarding the impact of Behaviourism [as represented by ICD] versus Social Constructivism [as represented by ICF]".

Returning to the ICD classification system (World Health Organization's ICD-10, 2007), this is one of the two broad aetiological-based classification systems

(or nosographic systems) in speech pathology, the other being the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). They provide a classification system for more than communication disorders[2].

Beyond these, specific classification systems exist for specific clusters of speech disorders: as to stuttering, Yairi (2007) and Seery, Watkins, Mangelsdorf & Shigeto (2007) enlist a great number of them, and we invite the reader to refer to these authors in order to know the assumptions of these systems in more detail. For sake of simplicity, I think that they could be easily redistributed under the same three main labels used by Waring and Knight (2013) about the Speech Sound Disorders (SSD): aetiologically based models, linguistic-descriptive models, and psycholinguistic models.

Aetiologically based models are atheoretical and start from a position of pathology rather than normality. Their underpinning premise is that an unvarying relationship exists between an identifiable genetic anomaly and a specific type of speech behaviour. This kind of model is also known as the model of the Mayo Clinic (Darley, Aronson & Brown, 1975), and has been recently questioned by Weismer (2006), which challenged the legitimacy and usefulness of a direct link between medical diagnosis and linguistic symptoms, and, in clinical practice, the reinforcement of non-linguistic oral functions in order to improve the linguistic ones.

The descriptive-linguistic approach to stuttering aims at classifying the subgroups of PWS according to the influence of speech sounds on the specific locations and types of stuttering (see the reviews of Zmarich, 1991; Zmarich, 2012; Zmarich, 2015). The approach is developmental (because of differences between children up to 7-8 years and older subjects) and it relies on the identification and description of the differences between PWS's speech compared to PWNS peers. Nonetheless, the descriptive-linguistic approach is unsuitable when it affords to data based on phonetic transcriptions delicate questions about the nature of disfluencies: as Smith (1999: 27) once highlighted, "static units of disfluency counts, for example, part-word repetitions or sound prolongations, are convenient fictions [...]" but "stuttering is not a series of 'stutter events' [...]", because "stuttering is a dynamic disorder". Any explanation based on transcriptional data is also undermined by the opacity introduced by the relative distance between the more or less central cause of the pathology and the more or less distal periphery in which the acoustic or perceptive events are measured. Moreover, the phonetic-phonological theories based on perceptive or acoustic targets are not suitable for explaining motor events of intrinsically dynamic nature.

This traditional view identifies the dysfluent loci in the stuttered utterance and explains these distributional patterns by invoking the same dysfunctions of the mental representations and processes which generate lapsus and disfluencies in normal speakers (Wingate, 1988; Zmarich, 1991). Apart from the methodological error of attributing causal relationships to the statistical associations of two events

[2] The difference between ICD and DSM rests on the wider scope of the first, which applies to all kind of diseases, with respect to the second, which applies only to mental disorders.

(the disfluency occurrence and the linguistic structure affected by the disfluency), the main defect of this interpretation rests on the exclusive attention to the disfluencies, which render the speech "discontinuous". But fluency is a multidimensional concept (see Lickley, this issue): not only the fluent speech is devoid of discontinuities, but it is also produced with a regular rhythmic beat, in rapid rate and without excessive physical and mental effort (Starkweather, 1987). We know that exist stutterers without disfluencies: they are affected by "covert/subperceptual stuttering" (Bloodstein, Bernstein Ratner, 2008) and often perceive in speaking excessive levels of muscular effort and "cognitive tension" that can pass unobserved to the eye and the ear of the clinician, because they are undetectable without laboratory instrumentation (like those used in experimental phonetics). From my point of view, Phonetics is in a privileged position for measuring the fluency reductions at the level of rhythm (Harrington, 1988) and rate (Andrade, Cervone & Sassi, 2003) and in a good position to measure them at the level of physical (Ingham, Warner, Byrd & Cotton, 2006) and mental effort (Panico, Healey, 2009). As a last criticism, research has demonstrated that, at least in adult stutterers, we cannot easily distinguish between signs of learned coping strategies (the secondary behaviours of stuttering) and signs of an underlying disorder (i.e. the primary behaviours of stuttering). In conclusion, we can not avoid to do the phonetic transcription of PWS's speech, but we have to take this as a starting point, not as an ending point, in order to do phonetic research on stuttering.

The psycholinguistic processing approach employs models of speech processing in children in order to explain 'how' speech impairment arises. Warin, Knight (2013: 34) described the psycholinguistic processing approach to SSD as a bridge between aetiological classification and linguistic descriptions: "Psycholinguistic speech processing models vary considerably in their complexity; however, the application to individuals is the same: a series of hypotheses are developed and systematically tested to find where the breakdown(s) [in the flow of information] is occurring".

Psycholinguistic and phonetic studies have a long tradition in stuttering research, and the advances in knowledge permitted by them have not lost any significance even in recent years when there has been an explosion of genetic and neurophysiologic studies (Bloodstein, Bernstein Ratner 2008). Genetic research established that the predisposition to stuttering is genetically transmitted, although the responsible mechanism at this level is still unknown (for a review, Kraft, Yairi, 2012). A number of brain structural and functional anomalies have been evidenced in adult stutterers (Etchell, Civier, Ballard & Sowman, 2017; Busan, Battaglini & Sommer, 2017; see also the contribution of Busan, this issue). Since similar findings have been documented also in children (Weber-Fox, Wray & Arnold, 2013; Chang, Zhu, Choo & Angstadt, 2015) both developmental stuttering and adult stuttering seem to rely on possibly shared dysfunctional cerebral mechanisms. This don't exclude complete recovery in early childhood, probably due to high neural plasticity (favourable fac-

tors are young age and short time interval from the onset, see Ludlow, Hoit, Kent, Ramig, Shrivastav, Strand, Yorkston & Sapienza, 2008).

However important they may be, these results risk to be incomplete and difficult to interpret: on the one hand, genetic studies face with distal causes at such a "molecular" level that they can not at present provide an explanation of the "proximal" causes of the stuttering behavior. Brain-imaging research aiming at testing a certain kind of linguistic process must be guided by psycholinguistic and phonetic hypotheses (Indefrey, Levelt, 2004; Indefrey, 2007), independently formulated. The model of speech production (and perception) most widely adopted by scholars is that of P.I.M. Levelt and colleagues (Levelt, 1989; Levelt, Roelofs & Mejer, 1999; Cholin, Levelt, 2009). The model was elaborated by considering evidences coming from different research fields: (1) speech hesitations (Mahl, 1956; Johnson, 1961; Goldman Eisler, 1968); spontaneous or provoked speech errors (Fromkin, 1973); self-repairs, (Levelt, 1983); (2) temporal reactions (RT) in simple tasks involving descriptions (Oldfield, Wingfield, 1965), or RT task complicated with priming (Lupker, 1979), and with different types of SOA (stimulus onset asynchronies, cf. Schriefers, Meyer & Levelt, 1990); (3) articulatory behaviors, inferred from acoustic analysis (Kent, Kim, 2003) and/or achieved directly through kinematic analysis (Gracco, 1992).

A psycholinguistic model like that of Levelt and coll. consists in a flow diagram that identifies the processing units, explicitly mentions the linguistic information and highlights the stages and processes of information processing that take place (e.g. activation, selection, monitoring, correction, etc.). Information processing proceeds in a unidirectional (from top to bottom levels), and incremental way (i.e., a lower level process is activated as soon as it receives an initial part from the higher stage).

Recently, two psycholinguistic theoretical hypotheses have been advanced which incorporate the view that stuttering is a dynamical and multidimensional phenomenon (as put forward by Smith *et al.*, 1997; Smith, 2016). The first one, *the Packman and Attanasio3-factor causal model of moments of stuttering* (Packman, 2012), states that (1) a deficit in the neural processing underpinning speech production renders the speech production system unstable and prone to perturbation, (2) the perturbation is triggered by some inherent features of speech (like stress or linguistic complexity) that increase the motoric task demands on that system, and (3) it is modulated by intrinsic factors (like physiological arousal) which determine the triggering threshold. The second one is the *Variable Release Threshold hypothesis of stuttering* (Brocklehurst, Lickley & Corley, 2013). It takes the best from two previous models (*Anticipatory Struggle Hypothesis*, Bloodstein, 1975) and *EXPLAN revised model* (Howell, 2003), whereby "the anticipation of upcoming difficulty leads to the setting of an excessively high threshold for the release of speech plan".

Regarding the part about the articulatory preparation and execution stages, which is not very detailed in the model of Levelt, you have to look for elsewhere, but luckily not too far. The model of speech motor control that have received most attention over recent years was developed by F.H. Guenther and colleagues, GODIVA

(Bohland, Bullock & Guenther, 2010), as a specialized derivation from the so-called Hybrid Motor Control or also State Feedback Control models (Hickok, Houde & Rong, 2011; Tian, Poeppel, 2012). It has been recently used in order to account for disfluent production in stuttering (Civier, Bullock, Max & Guenther, 2013), by simulating the consequences over the time course of blood flow caused by deficits in the basal ganglia (excessive levels of dopamine) and in white matter (low density), in a cerebral region below the left precentral gyrus.

Before finishing this excursus, I would like to come back again to the problem of distinguishing between the direct manifestations of stuttering and the reactions of the subjects to it. Regarding this point, it could be very important to study the affected subjects before they develop coping reactions, that is, we must study stuttering in young preschool children. In fact, stuttering could be defined as a typically childhood disease, because it begins between 16 and 66 months of age and less than 5% of PWS begin to stutter after they pass the 5th year of age (Yairi, Ambrose, 2005). This is the period of the greatest and fastest development in anatomo-physiological structures and functions, and in linguistic, cognitive and motor abilities as well. Its incidence is around 10% of all the children but its prevalence is only around 1%, due to the overwhelming probability of spontaneous recovery (around 90%, Yairi, Ambrose, 2013). Anyway, if spontaneous recovery does not happen within four years from the stuttering onset, that child is very likely "destined" to persistence. Maybe the key for solving the mystery of stuttering resides in the understanding why 9 children out of 10 which start to stutter, later recover spontaneously.

We know that non stuttering children are more disfluent when they attempt to produce new syntactic structures (Colburn, Mysak, 1982; Wijnen, 1990). According to Rispoli (Rispoli, 2003; Rispoli, Hadley & Holt, 2008), increases in Mean Length of Utterance parallel increases in revisions, and increases in utterance length parallel increases in "stallings" (i.e., part-word repetitions and/or prolongations). After the children are four years-old, only children who stutter continue to produce stallings consistently (Wagovich, Hall & Clifford, 2009). Moreover, research has established that, as to linguistic structures or psycholinguistic skills, stuttering children are not different from nonstutterers, in the average, but lower and upper performers are over-represented (Seery *et al.*, 2007). There are more frequent dissociations in stuttering children than nonstuttering ones among language capacity (often higher than normal), and articulatoy skills (often lower than normal, Coulter, Anderson & Conture, 2009).

As to childhood stuttering, a recent review (Sasisekaran, 2014) suggested a relationship between stuttering and phonology (excluding Phonetics, considered by the author as a low-level motor production of sounds) in 3 areas:

1. effects of phonological complexity on the location (loci) of stutter events;
2. outcomes of standardized test measures in children who do and do not stutter;
3. studies of phonological encoding in children and adults who stutter.

The results from the loci studies, according to Sasisekeran (2014: 95), "offer some support for the role of phonological complexity in the occurrence of stutter-

ing. Studies of performance in standardized tests of phonology have not identified differences between children who do and do not stutter. Studies of phonological encoding have been equivocal in reporting differences between children and adults who stutter and those who do not stutter".

We can integrate the results from this review on phonological factors with the experimental findings on the articulatory skills of preschool children: in the words of the authors, "they provide new evidence that preschool children diagnosed as stuttering lag their typically developing peers in maturation of speech motor control processes" (Smith, Goffman, Sasisekaran & Weber-Fox, 2012: 344; see also Walsh, Mettel & Smith, 2015; Smith, 2016).

We can conclude this introduction on stuttering and phonetic theories by presenting the Speech Motor Skills (SMS) model of van Lieshout and colleagues (see, among others, Namasivayam, van Lieshout, 2011): according to them, stutterers lay at the lower end of a hypothetical non-pathological continuum that characterizes speech motor skills. Disfluencies reflect errors in motor control, but stuttering is not a motor disorder (such as dysarthria or dyspraxia), but reflects an "innate" limitation of the verbal motor control system (clumsiness). Clumsiness emerges when programming and performing complex motor tasks in the presence of emotional, motor, cognitive and linguistic influences, and/or when demands are increasing for both accuracy and rapidity of movement. Most of the time these resources are crashed by dual-task processes. Not surprisingly, the linguistic condition that causes a worsening of stuttering is the "ecological" communication exchange (i.e. a conversation) where the PWS must simultaneously handle feedback at different levels and plan at the cognitive and syntactic level while performing in real time phonological planning and articulatory execution processes. Phonological coding can be considered a sort of dual-task process, because it requires simultaneous planning of subsequent language units during the articulatory execution of previous units (in addition to handling various feedback), PWS are limited in motor skills: they receive poor benefits from practice and are not able to generalize acquisitions to similar tasks, and/or to maintain them over time.

2. *The organization of the special session on stuttering and phonetic theory*

The organization of the individual contributions at this special session will proceed from more general and more "peripheral" (in the sense of the analysis of stutterers' speech based on the "auditory perceptions" of the hearers), to the most recent instrumental researches, passing from acoustics and kinematics to arrive to neurophysiology. Robin Lickley opens the special session by laying the foundations for every analysis of stuttered speech, that is the analysis of disfluencies: blocks, prolongations and repetitions, which are the hallmark of the disorder (see the ICD 9 definition at beginnings; see also Lickley, 2015). Lickley shows that "while typical disfluencies are mostly influenced by cognitive issues in the planning of speech and only rarely by motor control issues, stuttered disfluencies result from a break down

in the coordination of the complex motor commands necessary for successful articulation" (from the slide presentation). In other words, drawing from his presentation: "Typical disfluency is due to problems with planning, lexical access, word finding, errors, while stuttering is due to neurological problems".

As reported above, in recent years, the focus on the motor aspects of stuttering led to considering disfluencies as only one of the many ways speech could become disfluent. Considerations like these led speech researchers to concentrate on the perceptually fluent speech of stutterers, on the belief that PWS' speech could be abnormal even when the person is not openly stuttering. Acoustic and kinematic analysis, often associated to brain imaging or to electrophysiological techniques, have been used more and more, and speech motor control theories have become the preferred theoretical frame for most of the scientists. Moreover, for the reasons I explained above, researchers focussed on the affected subjects before they could possibly develop coping reactions, that is, in the preschool years. The contribution by Giovanna Lenoci illustrates a particular application of this methodological recommendation, such as the acoustic and kinematic analysis of coarticulation, which has been so much in the focus of research on stuttering that it was included in more than one definition: "[...] the difficulty is not manifested in the articulatory postures essential to that sound, but instead in moving on the succeeding one(s) (Wingate, 1964)"; "the lack of anticipatory coarticulation is probably the primary elements in the core behaviours of stuttering" (Stromsta, 1986). The degree of intra-syllabic anticipatory coarticulation also maintains a great potential as early predictor of stuttering persistence (Subramanian, Yairi & Amir, 2003), and Lenoci presents the first results of the CNR longitudinal project on early predictive indexes of persistent stuttering in early infancy. Forty pre-school children at high risk to develop stuttering due to familiar antecedents were tested when they were 2-years old for a number of linguistic, cognitive and physiological aspects. Those who developed stuttering were later followed longitudinally in order to ascertain the predictive power for stuttering persistence of some phonetic indexes, degrees of anticipatory coarticulation included (Zmarich, Bernardini, Lenoci, Natarelli & Pisciotta, in press). Although the predictive value of this last variable is currently still not clear, the authors found significant differences between peer-aged controls and all the PWS, without distinction between recovered or persistent sub-groups. Lenoci then presents some preliminary insights about her new project about the use of Ultrasound Tongue Imaging (UTI) for investigating anticipatory coarticulation and, more generally, speech (in)stability in primary school-aged PWS and PWNS. This focus on childhood helps to remember that all of us, as adult fluent speakers of one or more language, attained this unique capacity through a long process of acquisition and learning, during the pre-school years. This is the period of the greatest and fastest development in anatomo-physiological structures and functions, and in linguistic, cognitive and motor abilities as well, and Phonetic Sciences could tell us a lot about the possible ways this acquisition process could go wrong and produce stuttering.

Pierpaolo Busan will conclude the special session by illustrating his innovative research on PWS' neurophysiology, and he will show how the most recent models of speech motor control, as the State Feedback Control models (see above), have been applied to stuttering, which has been interpreted as a defect in sensory-motor integration. He states that "stuttering may result from speech components that are not properly synchronized, also because of time pressure; it may be the result of a series of errors that are present before, after, or during word execution; it could be influenced from load of cognitive processing as well as from phonological complexity; the anticipation of upcoming difficulties may cause the setting of higher thresholds for the release of speech motor plans" (from the slide presentation). His own work based on Transcranial Magnetic Stimulation (TMS) paired with EEG recordings is illustrated and the results support specific speech models of motor control in stuttering. The neural mechanism causing stuttering is a deficit in motor-to-sensory transformation: both dopamine dysfunction and white matter impairment may cause stuttering during speech motor control (see Civier *et al.*, 2013 and its simulation of stuttering starting from the GODIVA model). Even in this case, "findings may be useful for new treatment solutions for stuttering, ranging from neuromodulation to neurofeedback".

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