



BIROn - Birkbeck Institutional Research Online

Whurr, R. and Lorch, Marjorie (2002) Perceptual aspects of spasmodic dysphonia across different languages. *Stem-, Spraak- en Taalpathologie* 11 (1), pp. 22-29. ISSN 0924-7025.

Downloaded from: <https://eprints.bbk.ac.uk/id/eprint/15626/>

Usage Guidelines:

Please refer to usage guidelines at <https://eprints.bbk.ac.uk/policies.html>
contact lib-eprints@bbk.ac.uk.

or alternatively

Perceptual Aspects of Spasmodic Dysphonia Across Different Languages

Renata Whurr¹ and Marjorie Lorch²

¹*National Hospital for Neurology and Neurosurgery, London, England*

²*Birkbeck College, University of London, London, England*

Adductor spasmodic dysphonia (ADSD), an action induced dystonia, results in abrupt initiation and termination of voicing. Anglophone and Francophone vocal characteristics of ADSD are reviewed. Perceptual judgements were obtained from the symposium participants with a new Laryngeal Dystonia Feature Analysis checklist on videotaped ADSD interviews from 4 French, 1 Malaysian, 1 Greek, and 2 English. Clear differences were perceived in the presence of pitch breaks in various languages. This group confirmed their absence in French (replicating Lorch & Whurr, 1999) and presence in the other language samples. Language-specific phonotactics are suggested as a source of cross-linguistic differences in ADSD.

1. Introduction

Spasmodic dysphonia (SD) is a chronic phonatory disorder of neurological origin. It was originally termed "spastic dysphonia." It has been re-defined as an action induced focal dystonia of the laryngeal muscles and is an acquired movement disorder. Spasmodic dysphonia can be differentiated from vocal tremor and myoclonus and there is no evidence of spastic disease. It is rare, and of unknown aetiology. It is now thought to be linked to a genetic factor (Sakoda, 1993). It is more common in woman than in men and the onset is usually in the 4-5 decade. There are reports of 52 cases per million in the USA (Nutt et al., 1988). On this estimate there may be between 2-3000 cases in the UK. SD has been ill understood and often misdiagnosed in the past thus these figures may be an under estimate.

In spasmodic dysphonia the movements of the vocal cords during speech are abnormal, involuntary, irregular and variable. The irregular vocal cord contractions affect voice quality and control of speech, causing effortful production, abrupt interruptions to voicing, with abrupt breaks in phonation and changes in pitch. There is a broken speech pattern, resulting in a strained strangled voice quality as described in English speakers. The vocal symptoms in SD may be predictable and systematic from physiological rules governing production of phonation (Izdebski, 1992). Symptoms occur in loud modal phonation and when the fundamental frequency of the voice is raised within 1-2 octaves above the original speaking fundamental fre-

quency. It is noted that laryngeal spasms are not apparent during high pitch voice production and or during whispering (non phonated) speech. These two voice alterations are often used as compensatory strategies.

The site of the neurological lesion is thought to originate in the extrapyramidal system as an abnormality of neurotransmitters in the basal ganglia. There is a faulty processing of afferent information from the larynx and inappropriate discharges from the basal ganglia in response to the afferent discharges. It is thought that the cause of over pressured phonation is the result of faulty afferent-efferent motor control in the larynx during phonation (but not in vegetative function). There is a faulty reaction of the neuronal pools in the basal ganglia to arriving sensory signals resulting in excessive adductor forces in the vocal cords.

2. Classification and Characteristics

There are two main types of SD, adductor and abductor:

Adductor—In adductor spasmodic dysphonia (ADSD) there is abnormal involuntary contraction of the adductor laryngeal muscles (thyroarytenoids) leading to sustained adduction of the vocal cords (i.e., spasms). The thyroarytenoid and interarytenoid muscles of the vocalis complex are hyperactive. The vocal arrests and strain may be generated by the hyperadduction of both the true cords and the ventricular folds. Approximation of the ventricular folds occurs in severe cases or in compensatory phonation. The velum, tongue lips and face sometimes move abnormally. The excessive spasms and hyperadduction of the thyroarytenoid muscles produces constriction of the glottic airway resulting in abrupt initiation and termination of voicing. Glottal spasms reduce airflow and increases subglottal pressure during phonation. Voice quality is explosive, forced and effortful. There are phonatory breaks, pitch breaks and variations in pitch.

Abductor—Abductor spasmodic dysphonia (ABSD) is less frequent, about (15%). There is abnormal involuntary hyperadduction and contraction of the abductor laryngeal muscles (posterior cricoarytenoids) leading to sustained abduction of the vocal cords producing weak, breathy, effortful voice particularly on voice onset. There is abrupt termination of voicing resulting in aphonic or whispering lapses in speech.

Compensatory modes and strategies for adductor and abductor spasms—To avoid laryngospasms, patients may adopt laryngeal gestures in which spasms are less likely to occur, such as in whispering (to avoid all voicing) or when producing high pitched (falsetto) voice. Compensatory modes for abductor spasms may involve inspiratory speech patterns. To reduce effort patients may use short words and reduce phrase length.

There have been a number of descriptions of the vocal characteristics of this pathology over the past 2 decades. The earliest modern descriptions in the 1980's include a mixture of perceptual, acoustic, physiological and sensori-motor descriptors:

"Strangulated" (Dedo & Shipp, 1980).

"Voiceless sounds become voiced if vocal folds remain adducted during voiceless segments. Prosody is disturbed." (Dedo & Shipp, 1980).

"Staccato, jerky, squeezed, laboured, hoarse groaning with voice arrests (from hyperadduction of the true and false cords) intermittent phonation, segmented vowels, difficulty with loudness control, deviated pitch, vocal tension, intermittent aphonia, strangled voice, breathiness, glottal fry, glottal spasms, syllable repetition, vowel prolongations, whispered speech, choked vocal attacks and hard glottal initiation." (Blitzer & Brin, 1991).

"The universally accepted symptom is described as 'strain-strangled' or over pressured voice quality...and other vocal features are as a result of compensatory activity." (Izdebski, 1992).

Physiological features—The hyperadduction of the laryngeal muscles produces physiological changes in laryngeal postures during phonation resulting in adductor or abductor spasms, constriction of the glottic airway and glottal resistance as well as changes in sub glottal air pressure.

Acoustic measures—There are abnormalities in acoustic measures such as shimmer, jitter, signal to noise ration and voice break factor. There is a high standard deviation in measures of fundamental frequency due to the variability in pitch.

Paralinguistic features—Due to the phonatory aperiodicity, as a result of phonatory breaks and pitch breaks patient may compensate by selecting short words, reducing sentence length and altering the prosodic features in sentence production.

Listener perception—To the listener, the hyperadduction of the vocal cords and breaks in the speech signal are perceived as a voice that is "strained, strangled, squeezed, hoarse, intermittently choked, effortful, jerky, and staccato."

Patient perception—Patients describe their voices: "words came out with no emphasis or sentiment"; "I sounded like a robot" (Lorch & Whurr, 1999).

Voice quality is an accumulative abstraction of perceptual features involving laryngeal and supralaryngeal gestures. Descriptions of perceptually distinguishable components can be specified as distinct articulatory, acoustic and physiological corre-

lates. The salient vocal features that are the focus of the present analysis are: 1) phonatory breaks, 2) pitch breaks, 3) harshness, 4) creak, 5) breathiness, 6) whispery voice, and 7) falsetto as characterised in Laver (1980).

Phonatory breaks (also called vocal fry, voice breaks or laryngealizations) are created by a sudden contraction of the laryngeal muscles which are erratic in occurrence and over which the patient has no control. Phonatory breaks occur both within and between syllables, and produce the impression of a jerky and staccato delivery of speech.

Pitch Breaks are abrupt changes in the fundamental frequency of vocal cord vibration due to changes in vocal cord length resulting from spasm. This will be perceived as irregularity (and/or aperiodicity) in vocal pitch.

Harshness is distinguished from creak as an irregular rather than periodic noise with a modal fundamental frequency. Harshness gives an impression of effortful production with a strained-strangled voice quality. In extreme cases of harshness there is evidence of tension in the neck muscles sometimes even extending to the upper body.

Creak 'refers to a vocal effect produced by a very slow vibration of only one end of the vocal cords and ... very low pitch level' (Crystal, 1985, p.80). In the present data analysis, creak was generally applied to the description of the quality of an individual segment while harshness was applied to voice quality over longer speech events.

Breathiness. In instances of breathiness, the vocal cords are vibrating, but there is also a significant amount of air escaping through the glottis, causing turbulence. Breathiness is actually a subcategory of whispery voice but breathiness is the more typically used perceptual descriptor (Crystal, 1985).

Whispery Voice refers to instances which affect individual segments while breathiness will be used to refer to this quality in longer speech events. This is distinct from true whisper which is characterised by the vocal chords remaining in the open position, and there is no phonation.

Falsetto is produced when the vocal cords are stretched tightly so the resulting vibrations can have over twice the frequency of that when producing their modal voice.

3. Cross-linguistic variation

These characteristics have all been identified to some extent in the description of English speakers with ASD (Whurr et al., 1993). However, it has come to our attention that there may be language specific features which affect the manifestation of this vocal pathology in other languages. In the characterisation of ASD in French

speaking subjects that appeared in the English literature the relevant vocal symptoms were identified as: voice stoppage, laryngealisation, tremor, and breathy phonation (Chevrie-Muller et al., 1987). In a more detailed description of SD in French, Klap et al. (1993) described their speech as follows: "La dysphonie spasmodique...en adduction pure, elle est responsable d'une voix saccadée avec des arrêts vocaux fréquents et une incoordination pneumophonique." (Klap et al., 1993, p. 282). In this article, the authors provide a translation of the abstract in English which includes this description of SD: "adductor form with a jerky voice, pitch breaks, vocal arrests and pneumophonatory incoordination" (Klap et al., 1993, p. 281). It is notable that these two descriptions are not identical. In the French text, the diagnostic features emphasised are frequent phonatory breaks ("des arrêts vocaux fréquents"), while the English translation includes the symptom of pitch breaks, which is not mentioned in the French. The authors go on to state:

"La dysphonie spasmodique en adduction (la plus fréquente) est caractérisée par une voix étranglée, forcée, ponctuée d'arrêts vocaux, avec des spasmes respiratoires en inspiration ou en expiration lors de la voix conversationnelle et une mauvaise coordination pneumophonique. On note par ailleurs un forçage vocal, des difficultés d'attaque du son et des désonorisations intermittentes sur un rythme irrégulier et lent." (Klap et al., 1993, p. 282)

[Spasmodic dysphonia of the adductor type (the most frequent) is characterised by a voice that is strangled, forced, punctuated by phonatory breaks, with respiratory spasms on inspiration or expiration during conversational speech and poor pneumophonatory coordination. Vocal effort, difficulty with voice onset and intermittent devoicing in irregular and slow speech rhythm are notable. (translated by ML)]

These discrepancies in characterisation of ADSD speech in Francophones calls into question how phonotactic properties interact with laryngeal gestures in ADSD speech. The phonotactics of any given language represent the phonetic constraints of a given language for the possible sounds used, their ordering in sequences, position in syllables and in word formation. Given that the vocal pathology of ADSD affects the coordination of laryngeal and other supra and subglottal structures in speech production, the phonotactic constraints will necessarily restrict the range of required gestures needed to produce the linguistic forms of a given language. We were interested in exploring this question in a variety of ADSD speakers from languages with phonotactic characteristics which differ from English to broaden what is currently a restricted Anglophone characterisation in this vocal pathology. How do language-specific features place differential demands on the vocal cords? How might the linguistic environment trigger specific speech abnormalities? How is communication in general affected by this pathology?

4. Assessment of perceptual features

Previous phonetic and perceptual analysis by our lab had confirmed Klap et al.'s description of French ADSD as being comprised of harshness, breathiness and phonatory breaks without the presence of pitch breaks unlike the characterisations of English (Lorch & Whurr, 1999). We were interested to ascertain whether this perceptual characterisation would be upheld by a heterogeneous group of over 50 speech scientists with a range of clinical experience and language backgrounds.

Naturalist speech samples were extracted from videotaped interviews with clinicians from ADSD patients who were speakers of French, Malaysian, Greek and English. There were 4 French, 1 Malaysian, 1 Greek and 2 English samples each of approximately 30 seconds in duration. These samples were played for the audience of the symposium who were a mixture of clinicians and neurolinguists. They were also mixed as to their language background including mono- and multilingual speakers of French, Dutch, Flemish, German and English amongst others.

Participants were asked to listen to the speech samples and rate them according to a new checklist devised to identify the presence of various vocal features in ADSD: the Whurr Laryngeal Dystonia Feature Analysis checklist displayed in the Appendix.

5. Results and conclusions

The audience did not perceive pitch breaks in the samples of the 4 French ADSD samples, but phonatory breaks were perceived to be a prominent feature of the French samples. In contrast, the audience was able to perceive pitch breaks in the Greek and Malaysian, as well as in the English ADSD speaker samples. This confirms Lorch and Whurr's (1999) findings showing French ADSD speakers to have vocal pathology which is perceptually distinct from that of English ADSD speakers. It also raises questions about the degree of variability in perceptual characteristics of vocal pathology in languages with phonotactic patterns which diverge from French and English such as languages with phonemic tone contrasts (e.g., Malaysian), consonant clusters (e.g., Slavic languages) and phonemic contrasts in vowel duration (e.g., Dutch).

The development of a perceptual checklist may facilitate the clinical diagnosis of ADSD. Characteristics found to be typical in describing this vocal disorder in English speakers do not seem to be present in those samples we have examined for French speakers with ADSD. It is not clear to what extent the manifestation of this vocal pathology may differ across languages. The perceptual characteristics used to diagnose ADSD appear to be easily judged by specialist listeners such as the attendees of this symposium. Their responses suggest that language specific lists of vocal pathology will need to be developed to accurately characterize this disorder in languages which employ phonotactic features differently than English.

Samenvatting

Spasmodische dysfonie van het adductietype (SDAD), een door actie geïnduceerde dystonie, resulteert in een abrupt begin en einde van de stemgeving. Er wordt een overzicht gegeven van anglofone en francofone stemkenmerken van SDAD. Perceptuele beoordelingen werden verkregen van de deelnemers aan het symposium, die, met behulp van een nieuwe checklist voor de Kenmerken-Analyse van Laryngeale Dystonie, op videoband opgenomen interviews van 4 Frans, 1 Maleisisch, 1 Grieks, en 2 Engels sprekende SDAD-patiënten beluisterden. Er werden duidelijke verschillen waargenomen in het voorkomen van stembreuken in verschillende talen. Deze groep bevestigde hun afwezigheid in het Frans (replicatie van Lorch & Whurr, 1999) en hun voorkomen in de andere taalsamples. Taal-specifieke fonotactiek wordt gesuggereerd als een bron van cross-linguïstische verschillen in SDAD.

References

- Blitzer, A., & Brin, M. (1991). Laryngeal dystonia: a series with botulinum toxin therapy. *Annals of Otolaryngology, Rhinology, and Laryngology* 100, 85-89.
- Chevrie-Muller, C., Arabia-Guidet, C., & Pfauwadel, M.-C. (1987). Can one recover from spasmodic dysphonia? *British Journal of Disorders of Communication*, 22, 117-128.
- Crystal, D.A. (1985). *Dictionary of Linguistics and Phonetics*. 2nd Edition. Oxford: Blackwells.
- Dedo, H., & Shipp, T. (1980). *Spastic Dysphonia: A surgical and voice therapy treatment program*. Houston: College Hill Press.
- Izdebski, K. (1992). Symptomatology of adductor spasmodic dysphonia: a physiologic model. *Journal of Voice*, 6, 306-319.
- Klap, P., Marion, H., Perrin, A., Fresnel-Elbaz, E., & Cohen, M. (1993). Indication de la toxine botulique en laryngologie. *Revue de Laryngologie, Otologie, Rhinologie*, 114, 281-287.
- Laver, J. (1980). *The phonetic description of voice quality*. Cambridge: Cambridge University Press.
- Lorch, M., & Whurr, R. (1999). Cross-linguistic issues in speech production disorders: spasmodic dysphonia in French speaking subjects. In Pinto, M., Veloso, J., & Maia, B. (Eds.), *Psycholinguistics on the Threshold of the Year 2000* (pp. 637-638). Porto: University of Porto.
- Nutt, J., Muentzer, M., Aronson, A., Kurland, L., & Melton, L. (1988). Epidemiology of focal and generalised dystonia in Rochester, Minnesota. *Movement Disorders*, 3, 188-194.
- Sakoda, S. (1993). Genetics in movement disorders. *Nippon Rinsho*, 51, 2935-2939.
- Whurr, R., Lorch, M., Fontana, H., Brookes, G., Lees, A., & Marsden, C.D. (1993). The use of botulinum toxin in the treatment of adductor spasmodic dysphonia. *Journal of Neurology, Neurosurgery, and Psychiatry*, 56, 526-30.

Appendix

Whurr's Laryngeal Dystonia Feature Analysis ©2001

1. *Phonation* Breaks
2. *Pitch Breaks* High / Low
3. *Volume* Soft / Loud
4. *Voice Quality* Harsh / Hoarse / Rough
Raspy / Breathy / Creak
Whispery / Forced / Shaky
Tremulous
5. *Speed / Rate* Slow / Fast
6. *Prosody* Prolongation of vowels
Delayed voice onsets
7. *Concomitant Movements*
Strap muscles of the neck
Diaphragm movement
Eyebrow movement
Eyelid movement (blinking)
Facial expression
Hand movement