PERIPHERAL NERVOUS SYSTEM IN THE LARYNX AN ANATOMICAL STUDY IN SPASMODIC DYSPHONIA

THESIS

SUBMITTED IN PARTIAL FULFILLMENT FOR M.D. DEGREE IN PHONIATRICS

PRESENTED BY

AMANY AHMED DAWOD
(M.B., B.ch., M.S)

SUPERVISED BY

PROF. DR. MOHAMED NASSER KOTBY
Professor of Phoniatrics
Ain Shams University

PROF. DR. BEN COLBERT WATSON
Associate Professor of Speech Science
and Director of Research
New York Medical College, USA

PROF. DR. MAHMOUD YOUSSEF EL SAMAA
Professor of Otolaryngology
Ain Shams University

PROF. DR. TAHA KAMEL ALLOUSH
Professor of Neurology
Ain Shams University

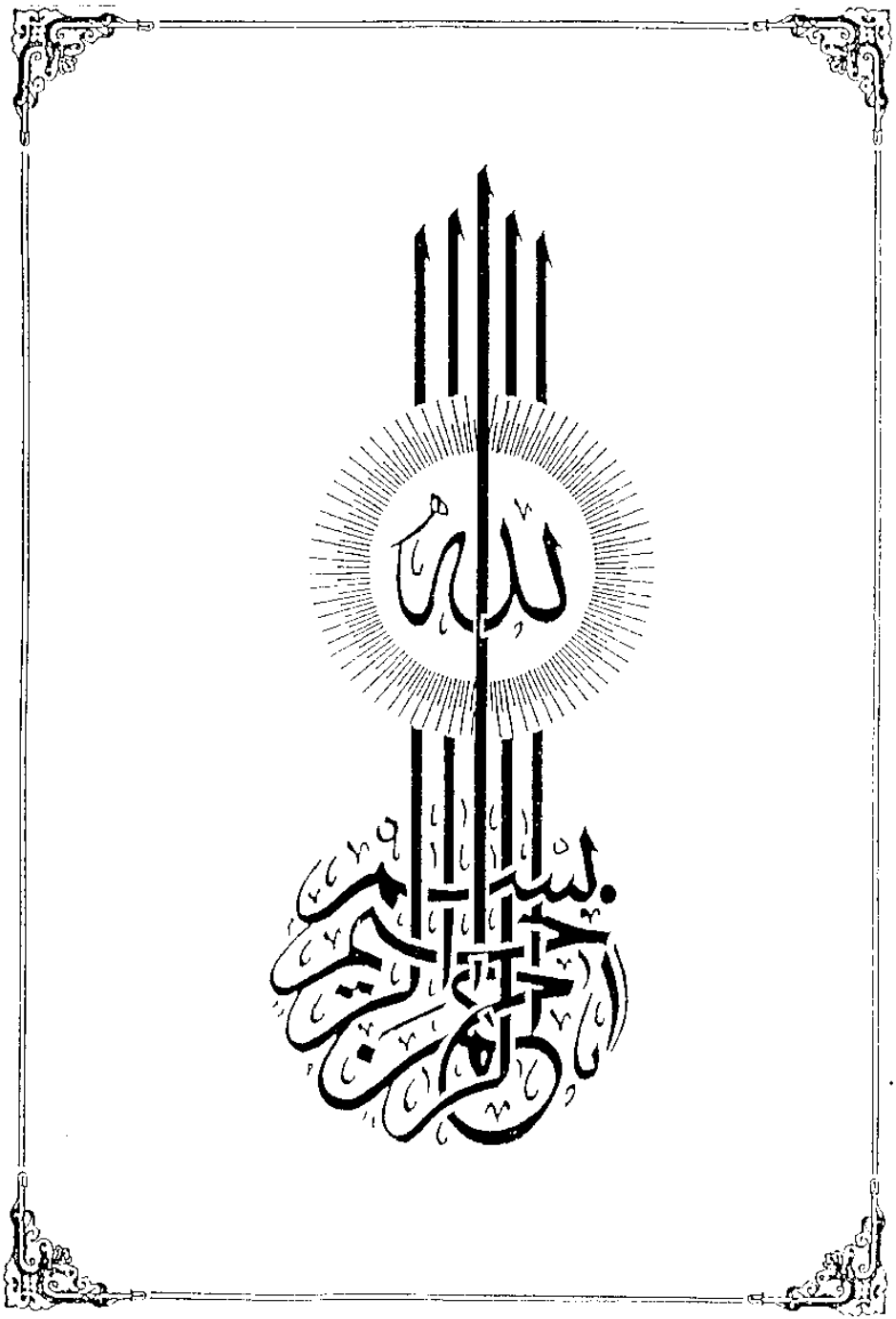
Faculty of Medicine
Ain Shams University
Arab Republic of Egypt
1999

بسم الله الرحمن الرحيم

« قالوا سُبحانك لا علمَ لنا إلا ما علمتنا إنك أنتَ العليمُ الحكيمُ »

صدق اللهُ العظيم

(سورة البقرة ، الآية ٣٢)



To My Family

With Love and Appreciation

Acknowledgement

I would like to thank Prof. Dr. Mohamed Nasser Kotby for his guidance, encouragement and support. Also, I would like to express my respect and thanks to Prof. Dr. Ben Watson for his efforts, cooperation and valuable supervision. Finally, my appreciation and thanks to Prof. Dr. Steve Schaefer, Prof. Dr. Rick Roark, Prof. Dr. Ron Baken, Prof. Dr. Mahmoud El Samaa, Prof. Dr. Taha Alloush and all the professors who helped me to continue this work.

CONTENTS

Page

Introduction——1

Aim of the work——4

Specific targets of the study——5

Spasmodic dysphonia——6

Historical background——6
Etiology——8
Pathophysiology——13
Clinical picture——23
Vocal fold behavior in laryngeal dystonias——28
Acoustic and physiologic characteristics of SD——35

Fundamental frequency control——43

Muscular forces——43
Aerodynamic forces——46
Structural forces——48

Neural control of the larynx——51

Motor nervous system——52
Sensory nervous system——59
Autonomic nervous system——61
Muscle spindles——64

Principals of laryngeal electromyography——72

Approaches——72
Apparatus and technique——74
The electromyographic signal——79
Kinesiologiocal electromyography in laryngeal physiology——80
Electromyographic analysis——83

Material and Methods——86

Perceptual analysis——87
Electromyographic recording——88

common compensatory strategy because it requires high levels of activity in intrinsic and extrinsic laryngeal muscles.

Bloch et al. (1985) found that while many vocal tasks enable patients to be free of vocal spasms, some other tasks have little, if any, effect on vocal spasms.

Non-communicative vocalizations such as coughing and throat clearing and primitive communicative vocalizations such as laughing resulted in a marked improvement of vocal symptoms in most cases. Tasks such as yawing, singing and chewing upon which communication was superimposed also resulted in improved phonation. Varied mode of vocal fold vibration (for example, breathy, glottal fry, falsetto and inspiratory phonation) also resulted in elimination or reduction of vocal spasms in most cases. Phonation produced at unusually high or low pitches brought about easier phonation in many cases, but the degree of improvement was less than for the other tasks described above. There was no difference in the effects of phonation produced at low or high pitches. Shouting brought about easier phonation in many cases, but to a lesser degree than the use of an unusual pitch. The results were poor for speaking by assuming foreign or regional accent patterns. Whispered speech produced spasm-free phonation. As the linguistic complexity of utterances increased, it became increasingly difficult for patients to achieve improved voicing. Tasks that elicited very little improvement in the severity or frequency of vocal spasms included choral reading and speaking a foreign language. Evidence of task-sensitivity points towards a cortical and/or subcortical abnormality in SD.

Upon consideration of the tasks reported by Bloch et al (1985), it appears as though altered vocal pitch is a good candidate for use in remediation of patients with SD.

Aim of the work

The aim of this work is to apply spectral analyses of electromyographic signal in the study of laryngeal spasm in spasmodic dysphonia in order to examine the neuromotor bases of vocal symptoms in that disorder.

Specific targets of the study

The specific targets of this study is to examine relations between amplitude-based changes in thyroarytenoid (TA) and cricothyroid (CT) activity across pitch conditions and the perceived severity of vocal symptoms in persons with spasmodic dysphonia.

Potential extension of the study

The potential extension of this study is to measure glottal resistance across pitch conditions as a preliminary study.

Review of Literature

Spasmodic dysphonia

Historical background

Spastic dysphonia was first reported by Traube in 1871 (cited by Blitzer et al., 1985) to describe patients with nervous hoarseness. Fraenkel (1887, cited by Blitzer and Brin, 1992) later described this vocal abnormality as mogiphonia, which he characterised as a slowly developing disorder with increasing vocal fatigue, spastic constriction of the throat muscles, and pain around the larynx. Critchley (1939, cited by Blitzer and Brin, 1992) described the voice pattern as one in which the patient seemed to be trying to talk while being chocked. Arnold (1959) warned against confusing spastic dysphonia with spastic (pseudobulbar) dysarthria in which the voice often sounds like that of spastic dysphonia. He also mentioned the necessity of excluding multiple sclerosis, cerebellar disease and parkinsonism. Bloch (1965) described adductor SD as related to a hysterical conversion disorder. Many other authors including Aronson et al. (1968b), Dedo & Izdebski (1983) and Feldman et al. (1984) described various observations linking 'spastic dysphonia' to a central nervous system disease. Marsden and Sheehy (1982) first noticed a relationship between SD, Meige's disease (blepharospasm / oromandibular dystonia) and torsion dystonia. When Marsden and Sheehy reviewed Critchley's (1939) case reports, they found that the first case had SD as well as a tremor of the left arm and torticollis and the third case had SD and a tremor of the jaw. In Marsden's (1982) series of Meige's disease, they also noted multifocal variations in the presentation. They concluded that all evidence points to the conclusion that Meige's disease is another manifestation of adult onset torsion dystonia. Since dysphonia may occur in the same syndrome, it is quite likely that isolated

dysphonia itself may be the sole manifestation of dystonia. Golper et al. (1983) analyzed cases of Meige's syndrome and found that aside from the blepharospasm and oromandibular involvement, some patients also had involvement of platysma, soft palate, tongue, pharynx, oesophagus, and respiratory muscles. Those with voice involvement had voice stoppages, inspiratory phonation, strained strangled voice, harsh voice, vocal tremor, and audible grunts. They also believed that most of these signs were consistent with dystonia. Schaefer (1983) in his analysis of 28 cases of dysphonia, found some cases with tremor and others with associated velar, labial or mandibular spasm. There were also two patients with tremors of the hand. Two thirds of the patients in Schaefer's series had dysphagia or complaint of a lump in the throat and 50% of the patients had neck pain. He drew the analogy between dysphonia and spasmodic torticollis.

Blitzer et al. (1985) recognized a dysphonia similar to that in patients with generalized and multifocal dystonia. Both clinical and electromyographic (EMG) characteristics led them to the conclusion that most cases clinically diagnosed as dysphonia are a form of cranial dystonia.

Much controversy surrounds the etiology of this disorder, so it is important to review divergent schools of thought regarding the etiology of this condition. Such review will illustrate the evidence which strongly supports the concept that spasmodic dysphonia can be a laryngeal sign of dystonia.

The etiology of spasmodic dysphonia

There are two divergent schools of thought regarding the etiology of this condition. Spasmodic dysphonia was long considered as a psychogenic disorder and was usually classified as a conversion reaction (Bloch, 1965). Then Robe et al. (1960) found abnormal electroencephalographic patterns in most of their patients with SD. Later, Aronson et al. (1968a) described neurologic aspects of this disorder that implicated the extrapyramidal tracts as the possible site of lesion. Aronson and DeSanto (1983) attributed SD to several possible sources:

1) Central nervous system diseases involving movement disorders that hyperstimulate or disinhibit centers that control laryngeal closure;
2) Acute or chronic emotional stress that can increase laryngeal musculoskeletal tension (conversion reaction); and
3) Idiopathic (if no etiologic factor can be identified).

In sum, etiological theories of SD are either functional (psychogenic), organic (neurologic) or idiopathic. Regarding organic (neurologic) origins, several possibilities include:

(1) Spasmodic dysphonia as a manifestation of disordered function of the central nervous system:

Robe et al. (1960) found that ninety percent of their patients had electroencephalograms abnormalities. Aronson et al. (1968b) found abnormal neurologic signs in 20 of the 27 patients.

(2) Spasmodic dysphonia as a manifestation of peripheral demyelination:

Dedo et al. (1978) reported 30 percent of the 89 nerves examined were abnormal and had bundle-like groups of fibers that did not stain for either

myelin or axons. Bocchino and Tucker (1978), examined 18 recurrent laryngeal nerves by both light microscopy and electron microscopy. Fourteen specimens showed evidence of demyelination. Their findings of segmental demyelination were not verified by the more sensitive technique "teased-fibre morphology" reported by Ravits et al. (1979). Their results led Ravits et al., to favor the view that SD is a disorder of the extrapyramidal system.

(3) Spasmodic dysphonia may be a sign of essential tremor:

Essential tremor is an intention type of tremor that may affect the larynx in isolation and that appears with an average frequency of 4-12 Hz during prolongation of vowels.

Aronson and Hartman (1981) concluded that "the presence of voice tremor or rhythmic voice arrests on clinical examination, despite the absence of other neurologic signs of tremor, should arouse suspicion of essential tremor" (p. 55, 57).

Both adductor and abductor spasmodic dysphonia are often associated with a vocal tremor of approximately 7 Hz (Dordain & Dordain, 1972).

(4) Spasmodic dysphonia is an organic disorder which involves both somatic and visceral brainstem nuclei and pathways (Schaefer et al., 1983):

Patients with SD show abnormalities that may reflect dysfunction of the brain stem. Such abnormalities include abnormal brain stem auditory evoked responses, reduced stomach acid responses to sham feeding, reduced fluctuation in heart rate during Valsalva maneuver (Schaefer et al., 1983; Feldman et al., 1984) and increased excitability of blink reflexes (Tolosa & Motserrat, 1985; Cohen et al., 1989a).

(5) Spasmodic dysphonia is a supranuclear movement disorder:

Schaefer et al. (1985) and Finitzo and Freeman (1989) believe that SD is a