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**An acoustic and perceptual study of spastic dysphonia**

**Blaustein, Steven H., Ph.D.**

**City University of New York, 1990**

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**AN ACOUSTIC AND PERCEPTUAL STUDY OF SPASTIC DYSPHONIA**

by

**STEVEN H. BLAUSTEIN**

**A dissertation submitted to the Graduate Faculty in  
Speech and Hearing Sciences in partial fulfillment of  
the requirements for the degree of Doctor of  
Philosophy, The City University of New York.**

1990

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This manuscript has been read and accepted for the Graduate Faculty in Speech and Hearing Sciences in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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**Abstract**

**AN ACOUSTIC AND PERCEPTUAL STUDY OF SPASTIC DYSPHONIA**

by

**Steven H. Blaustein**

**Advisor: Professor Katherine Harris**

**Adductor Spastic Dysphonia is a debilitating voice disorder characterized perceptually by a choked, effortful, strained-strangled vocal quality. The vocal cords appear normal on direct examination. The purpose of this study was to determine if patients with adductor spastic dysphonia exhibited differences in voice onset time and measures of devoicing when compared to normals. The study also determined whether subjective perceptual ratings for overall severity and strain-strangle quality in spastic dysphonic patients would correlate with objective acoustic measures.**

**Subjects were six patients each with a confirmed diagnosis of spastic dysphonia and a matched group of control subjects. Samples of spontaneous speech and a reading segment were obtained from each subject.**

**Data collection consisted of measurements of voice onset times for /p/, /t/, and /k/, and determining devoicing intervals for /s/, /ʃ/, and /f/. Measurements were obtained from targeted segments. Samples were also perceptually rated on a 7 point scale by six trained listeners.**

Results showed that, as a group, spastic dysphonic subjects demonstrated significantly shorter voice onset time measurements for stops /p/, /t/, and /k/. Significantly shorter devoicing intervals were found for /f/. A tendency was also noted for mean subjective ratings of severity to increase with increases in instances of devoicing failures.

Interjudge reliability measures showed that a reading sample is as reliable as an elicited spontaneous speech sample. Strain-strangle proved to be a more reliable perceptual rating dimension than a less specific judgement of "severity".

Results suggest that measurements of voice onset time and devoicing intervals can be used effectively to distinguish spastic dysphonic from normal subjects. These measures were also found to be effective in objectively specifying differences within the spastic dysphonic population.

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## Chapter I

### Introduction

Spastic dysphonia was first described over 100 years ago by Traube. Since then, even with significant advances in medicine, research and technology, it still remains an enigma. Brodnitz (1976) characterized the disorder as "the most mysterious, the most poorly understood, and the most resistant to successful therapy of all the disorders of the human voice." Aronson (1980) describes spastic dysphonia as the most serious of all voice disorders with catastrophic psychologic, social, and emotional consequences.

Spastic dysphonia is a debilitating disorder of phonation. The resultant voice quality has been described with such adjectives as strained, strangled, creaky, hoarse, effortful, constricted, choked, groaning, staccato, and laborious. The diagnosis is usually based on the very characteristic vocal quality in the absence of any other symptoms. Dedo (1983) reports that the major complication of spastic dysphonia is misdiagnosis, because the disorder must be diagnosed subjectively . . . with a "trained ear".

The incidence of the disorder is described as "rare" and accounts for only 4% of all voice disorders (Brodnitz 1976, Aronson 1980). It occurs almost exclusively in middle age. The disorder affects twice as many women as men according to Dedo and Shipp (1980), while Aronson (1980) reports a male to female ratio ranging from 1:1 to 1:1.8.

Spastic dysphonia is a somewhat controversial diagnosis and it remains unclear whether the label of "spastic dysphonia" refers to an actual disorder or merely describes a symptom. Spastic dysphonia may also be used as a general term that describes a number of underlying entities. Aronson (1983) notes that the laryngospasm responsible for spastic dysphonia can arise from one of several sources. These may include acute or chronic emotional stress and diseases of the central nervous system. Wilson, in a 1984 ASHA interview, stated, "I think that the concept of one spastic dysphonia is just not true . . . we have to be more careful in our evaluation of the patient (p.22)." It is not surprising then, that numerous researchers have attempted to better delineate "spastic dysphonia" by suggesting a variety of more specific diagnostic terms.

Aronson (1974) delineated spastic dysphonia into two large subgroups when he first suggested the use of the terms adductor spastic dysphonia and abductor spastic dysphonia.

The abductor type referred to a "mirror opposite" of the classic adductor type. The resultant "abductor" type of spastic dysphonia was characterized by abductor laryngospasms resulting in intermittent breathiness. McCall (1971) suggested that "spasmodic dysphonia" should be the appropriate label to describe the disorder. Aronson and Hartman (1981) suggest using the diagnosis of "spastic dysphonia of essential tremor" or a similar term whenever organic tremor can be confirmed as existing. The term "idiopathic spastic dysphonia" is recommended for forms of spastic dysphonia where no cause can be found. Cannito and Johnson (1981) further added to an already confused diagnostic classification

schema when they showed that abductor and adductor symptoms could coexist in one patient and suggested that spastic dysphonia is, in fact, one disorder existing on a continuum. Cooper (1973) describes incipient and chronic spastic dysphonia. "Incipient" spastic dysphonia is supposedly characterized by similar but less severe symptoms than "chronic" spastic dysphonia. Incipient spastic dysphonia is further thought to be a forerunner of chronic spastic dysphonia.

Thus, at least seven different names are found in the literature that refer to what may, or may not be, one disorder. The original diagnostic term of spastic dysphonia clearly persists most strongly and the controversy continues.

One area of consistent agreement between researchers, speech pathologists, and anyone else confronting this disorder is the extremely poor prognosis for recovery (Aronson, 1975; Dedo, 1980). There is currently no definite cure for this disabling disorder of phonation and therefore, research actively continues. This paper will contribute to that research.

## Chapter II

### Statement of Purpose

A large amount of research, attention, and speculation has surrounded spastic dysphonia. The one, often referred to, outstanding feature of the disorder is the unmistakable perceptual impression it leaves. Dedo and Izdebski (1983) state, "attempts to make the diagnosis with such objective laboratory techniques as acoustic analysis, glottography, electromyography, cineflouroscopy, aerodynamics, etc., may, at best, verify the aural diagnosis, but by themselves they are not diagnostic". The diagnosis must be made with a "trained ear" and indirect mirror laryngoscopy adds little. Yet "trained ears" have heard so many varieties of spastic dysphonia that a myriad of diagnostic terms have been suggested. Further investigation into the acoustics of the disorder is clearly warranted.

It would seem that such a unique voice disorder would compel researchers to systematically analyze the acoustics of the disorder. Yet, this has not been done. Fewer than six objective acoustic studies exist among the over seventy articles published about spastic dysphonia. Researchers continue to rely almost entirely on perceptual evaluations even when weighing the merits of surgery (Dedo, 1976, 1979, Biller, Som and Lawson, 1979, Izdebski and Dedo, 1981). This is surprising considering the known problems inherent in perceptual analysis and the presently widespread availability of objective acoustic measurement techniques such as spectrographic analysis.

The purpose of this investigation is, therefore, to specify an acoustic correlate of adductor spastic dysphonia that may aid in the diagnosis and remediation of the disorder, and perhaps account for the unique clinical impression. A second part of the investigation will determine if a relationship exists between acoustic measures and perceptual impressions.

The subjects used in this study have all been diagnosed as having adductor spastic dysphonia. To avoid confusion, therefore, let it be stated from the beginning that the disorder being investigated is adductor spastic dysphonia. It is generally agreed by researchers and clinicians that the disorder of adductor spastic dysphonia is primarily characterized by hyperadduction of the vocal cords causing the classic "strain-strangle" voice quality. One laryngeal event that may prove useful in analyzing spastic dysphonia is voice onset.

Voice onset time measures and measures of "devoicing" would appear logical methods of measurement for spastic dysphonia in that the disorder is largely one of initiating and maintaining appropriate vocal fold adduction. Furthermore, voice onset time measures have demonstrated differences between normal and abnormal populations of various kinds (Freeman, Sands, Harris, 1978; Metz, 1983; Agnello, Wingate, Wendell, 1974).

Voice therapy for spastic dysphonia is often aimed at establishing an "easy onset" of voicing. The most helpful treatment, thus far, has been to paralyze one vocal cord so as to physiologically limit a patient's ability to adduct the cords. This allows for easier onset, greater air flow through the glottis, and generally less "spastic" voice quality. The problem of spastic dysphonia is first, and

foremost, one of the inability to control adduction. Thus, voice onset time, and measures of devoicing, should be compromised in patients with spastic dysphonia.

This investigation will specifically determine if subjects identified as having adductor spastic dysphonia exhibit differences in voice onset time and measures of devoicing when compared to a matched group of control subjects. It will further determine if such measures correlate with subjective perceptual impressions of spastic dysphonia.

## Chapter III

### Literature Review

#### Adductor Spastic Dysphonia: The Disorder and Its Causes

Spastic dysphonia, as a diagnosis, has been known for over 100 years. Its cause remains unknown and diagnosis is still largely performed by subjective perceptual impression. Four areas of research into etiology have been pursued: psychogenic, neurogenic, histopathology and electromyography. A brief review of these areas is helpful to gain some appreciation of the nature of the problem of spastic dysphonia and the puzzling nature of its etiology.

A diagnosis of adductor spastic dysphonia is often made by the speech pathologist based on the characteristic perceptual impression of a voice that has typically been described in the literature as grunting, choked, effortful, staccato, strain-strangle, squeezed, groaning, jerky choppy, and harsh. Aronson (1980), in his text on clinical voice disorders cites the following conditions as indicative of adductor spastic dysphonia:

- 1) presence of squeezed, staccato, or groaning dysphonia.
- 2) absence of mass vocal fold lesions or paralysis.
- 3) absence of abnormal speech signs in the remainder of the peripheral speech mechanism.
- 4) brief periods of normal voice during laughter, singing, shouting or anger.

5) resistance to therapy. (pp. 157-158)

The etiology of spastic dysphonia remains unclear. Early literature on the disorder postulated a psychogenic cause as is often the case when a physiologic cause remains undetected. While it is generally agreed upon today that spastic dysphonia has such a physiologic base, a review of some of the earlier theories regarding spastic dysphonia provides an historical perspective.

Luchsinger and Arnold (1967) stated that since spastic dysphonia was first described, it has been generally agreed that it represents a psychoneurotic disorder of respiratory-phonatory incoordination. Segre (1951), in discussing what he terms "Spasmodic Aphonia", reported that in the majority of cases there exists only one neurotic element favored by different psychic factors, which fix the patient's attention of the phonatory organ, and thus determine the appearance of the vocal syndrome each time that factor repeats itself. This neurotic element, sometimes obsessive, sometimes weaker, always exists. In other words, spastic dysphonia is a form of psychological conversion reaction localized to phonation.

According to Arnold (1959) spastic phonation represents a regressive phenomenon. The underlying primitive sphincter mechanism of the larynx may be activated by subcortical secondary centers of phonation. These may take over the function of the primary cortical centers when the latter become inhibited by the unconscious withdrawal of a shattered personality from the threats of daily life. Arnold believes that the etiology and syndrome of spastic dysphonia are associated with serious problems in the psychopathology of the patient and that

this voice disorder should be primarily handled by a psychiatrist. Heaver (1959) concurs that professionally qualified psychiatric treatment should be the therapy of choice for patients with spastic dysphonia. Block (1965) concludes "spastic dysphonia has a clear psychogenic basis, but this does not exclude, absolutely, neurologic signs, symptoms, and so on. The difficulty in treating these patients results from the fact that they are often on the borderline between neurosis and psychosis". Finally, Brodnitz (1976) after reviewing 130 cases of spastic dysphonia with special emphasis on the etiology of the disease concluded that "overwhelming proof speaks for a psychogenic disorder".

Early researchers, however, did not totally ignore the possibility that spastic dysphonia was a neuropathology. Critchly (1938) in an article entitled Spastic Dysphonia ("Inspiratory Speech") mentions "an uncommon and peculiar type of speech affection". A constrained, forced, barely intelligible, imperfectly modulated voice frequently accompanied by tic-like contractions of the face, neck, and even upper limbs is described. Critchly noted that this type of voice is found in the literature in association with various disorders, such as progressive cerebellar degeneration and double athetosis. Three patients were described exhibiting a "spastic dysphonia" but in each case presented, a clear clinical diagnosis was never arrived at with confidence.

In 1944, Luchsinger (cited in Luchsinger and Arnold, 1967) studied the influence of the neuro-vegetative system through the pupillary reflex in spastic dysphonics. His finding indicated excessive dilation of the pupil of the eye during phonation suggesting an "abnormal neural mechanism".

Rabuzzi and McCall (1972) view spastic dysphonia as a reflection of at least one type of central nervous system dysfunction and Dedo (1976) suggests that spastic dysphonia is caused by a disturbance in the proprioceptive control of the vocal cords. He specifically proposes that neurotrophic viral infection damages the a and c fibers either peripherally or in the central nervous system while sparing the larger diameter motor nerve fibers to the intrinsic laryngeal muscles. The a fibers are the motor fibers that are also thought to be affected in Bell's Palsy and spontaneous recurrent laryngeal nerve paralysis. The c fibers are thin unmyelinated nerve fibers involved in proprioceptive control. The exact proprioceptive vocal cord mechanism is admittedly poorly understood. Over one-half of Dedo's clinical patients had a clinically apparent viral illness of the respiratory or gastrointestinal system at, or shortly before, the onset of spastic dysphonia.

Dedo, Townsend, and Izdebski (1978) report on results of neurologic examinations on 12 patients with spastic dysphonia. Six patients showed signs of neurologic disturbance apart from the laryngeal symptom. Postural or familial tremor was seen in 3 patients, one patient showed a disorder of idiopathic torsion dystonia, one showed blepharospasm, and the sixth showed buccolingual dyskinesia. It is noted that electroencephalograms for all 12 patients were normal.

Aronson, Brown, Litin, and Pearson (1968) used neurologic examination, psychiatric interview, and descriptive criteria to evaluate 27 patients. They were able to detect positive neurologic signs in 74% of the patients studied.

Schaefer (1983) found associated neurologic symptoms in all but one of 27 patients he studied. He cites three independent evaluations of brain stem function to conclude that spasmodic dysphonia is a descriptive term for a heterogeneous group of patients. "Spasmodic dysphonia" patients may be "united" by their speech signs despite either a functional or organic etiology. Spasmodic dysphonia is believed to be one of several spasmodic brain stem disorders that present themselves variably.

Perhaps the strongest support for the neurogenic basis of spastic dysphonia comes from the work being done at The Dallas Center for Vocal Motor Control. Finitzo and Freeman (1989) report on seven years of research. A total of 75 spastic dysphonic patients served as subjects in a variety of experiments. These included auditory brain stem response, tests of gastric and cardiac vagal function, fiberoptic studies of vocal tract movements, electromyographic studies, motor performance, and brain imaging studies (MRI, BEAM, SPECT). Results showed 35% of the patients to have abnormal auditory brain stem signs, approximately 50% showed reduced vagal activity, and abnormal vocal tract movements were observed in patients with spastic dysphonia. Abnormalities were consistent with a clinical diagnosis of tremor and/or dystonia.

Electromyographic findings included abnormal cluster bursts in velar (Levator Palatini) recordings and disruptions in neuromuscular activity of laryngeal muscles (thyroarytenoid, cricothyroid). Greater than 80% of the spastic dysphonic patients exhibited multifocal, structural, metabolic, and/or physiologic

abnormalities of the central nervous system as indicated on various brain imaging studies.

The authors conclude that "spastic dysphonia is a supranuclear movement disorder primarily, but not exclusively, affecting the larynx". The authors continue" . . . at least one form of spastic dysphonia reflects combinations of focal cortical dysfunction--specifically, left frontal/temporal cortex, paramedian frontal cortex, and right posterior temporal/parietal cortex (p. 553)".

In 1977, the emergence of the recurrent laryngeal nerve section for spastic dysphonia enabled researchers to more directly study spastic dysphonia from a histopathological point of view. Izdebski (1977) examined sections of recurrent laryngeal nerve (RLN) from 89 patients with spastic dysphonia and 21 age matched controls. Thirty percent of RLNs from the patients with spastic dysphonia were found to exhibit large groups of small non-myelinated axons. This was not seen in the normal control group nor has this been reported in any human or non-human study of the RLN. The exact nature of these histologic changes, the cause, and their relationship to the phoniatic symptoms remains to be clarified (p. 122). In 70% the patients the RLNs were apparently normal. The authors suggest a sampling error or multiple etiology to account for this discrepancy.

Bocchino and Tucker (1978) also studied RLN pathology in spasmodic dysphonia. Eighteen RLN segments from patients with spastic dysphonia and four normal RLN segments were studied. The normal segments were not identified to the neuropathologist. By light microscopy, 6 nerve segments demonstrated

minimal abnormalities and 12 were normal. Electron microscopy revealed 14 specimens to have pathological findings and tissue preparation was unsatisfactory in 4 segments. Segmental demyelination was the lesion consistently noted. The axons were normal except for occasional secondary changes resulting from extensive demyelination. No viral inclusion bodies or inflammatory infiltrates were observed. All control RLN segments were normal in all respects. These results suggest that spasmodic dysphonia is a demyelinating disease of peripheral nerves. Similarity of findings in studies dealing with cranial nerve syndromes other than spastic dysphonia suggest that several cranial nerve syndromes may be differing manifestations of one disease process. These include trigeminal neuralgia, glossopharyngeal neuralgia, blepharospasm, hemifacial spasm, and possible Bell's Palsy.

Ravits, Aronson, Desanto, and Dyck, (1979) did biopsies of RLN's obtained from nine patients who underwent laryngeal nerve sections for spastic dysphonia. Morphology, median fiber diameter, density, and size distribution of fibers was evaluated for various regions and the entire nerve segment. No significant differences were found between the diseased group and a control group. The authors were unable to verify previous reports of neuropathic abnormalities in spastic dysphonia and question such findings.

It is clear from the studies cited that recent evidence clearly points to a neurogenic etiology for spastic dysphonia. Yet, the exact underlying neural abnormality continues to be elusive and the idea of multiple underlying mechanisms remains a strong possibility. Cortical (Finitzo and Freeman, 1989),

brain stem (Schaefer, 1983) and peripheral (Boccino and Tucker, 1978; Dedo, Townsend, & Izdebski, 1978) sites have all been suggested. Further studies regarding neural etiology are warranted.

Electromyographic (EMG) studies of spastic dysphonic patients have sought to discover physiologic correlates that might be helpful in determining etiology or aid in diagnosis. Rabuzzi and McCall (1972) studied extrinsic laryngeal muscles in spastic dysphonic patients using EMG and found intermittent muscle contractions during nonvocal tasks. Shipp, Izdebski, Reed, and Morrissey (1985) sampled EMG activity from the thyroarytenoid, posterior cricoarytenoid, in a spastic dysphonia patient. Results indicated intermittent sudden increases in adductory muscle activity that coincided with momentary voice arrests. Blitzer, Lovelace, Brin, Fahn, and Fink (1985) studied patients with spastic dysphonia using EMG of the cricothyroid and thyroarytenoid muscle independently. EMG of the laryngeal muscles failed to show any abnormal spontaneous activity in 14 patients. Seven patients, however, showed EMG abnormalities that included increased unilateral amplitude of motor unit potentials (MUP's) on phonation, asynchronous activity characteristic of tremor, and bursts of activity suggesting extrapyramidal disease of diffuse myoclonus. The patients in this study evidenced no EMG symptoms that were felt to be traditionally associated with clinical spasticity such as slowed MUP's. The findings noted, however, do seem to present in patients with dystonia, a neurologic disorder characterized by abnormal involuntary movements. The authors therefore conclude that spastic dysphonia is not a spastic disease but rather a laryngeal dystonia.

Ludlow, Baker, Naunton, and Hallet (1986) inserted EMG needles percutaneously into the right thyroarytenoid, left thyroarytenoid, right cricothyroid, and left cricothyroid in five patients with "idiopathic" spastic dysphonia and five normal controls. Results showed significantly higher levels of muscle activity compared to normal subjects during quiet respiration and phonation. Abnormally high muscle tone is suggested. Abnormal laryngeal muscle activation timing is also suggested. Freeman and Finitzo (1989) also noted high amplitude Cricothyroid activity and task specific variations.

One apparent feature of spastic dysphonia that continues to prevail is the inability for any one study to find a symptom that is applicable to all patients with spastic dysphonia. This diversity of finding in spastic dysphonic patients becomes even greater when comparing one study to the next and as a result, the enigmatic nature of this disorder continues. Table 1 summarizes the various electromyographic and neurologic findings in patients with spastic dysphonia.

#### Spastic Dysphonia: Acoustic and Perceptual Studies

Numerous articles exist where perceptual measures alone have been used —to make judgements regarding various aspects of spastic dysphonia. Perceptual measures have most frequently been used to measure the effectiveness of some treatment by comparing pre- and post-treatment conditions rather than using these perceptual measures to determine parameters of the disorder itself. This treatment has usually been related to the recurrent laryngeal nerve section for spastic

Table 1

Summary of Electromyographic and Neurologic Findings in Spastic Dysphonia

Researcher	Finding
Aronson, Brown, Litin, Pearson, (1968)	Vocal tremor, facial/labial twitches, head and hand tremor
Rabuzzi and McCall (1972)	Abnormal EMG/s/Extrinsic laryngeal muscles
Dedo (1976)	Peripheral laryngeal nerve damage
Izdebski (1977)	Nonmyelinated axons in recurrent laryngeal nerve
Bocchino and Tucker (1978)	Segmental demyelination of recurrent laryngeal nerve
Dedo, Townsend, Izdebski, (1978)	Myelin abnormalities, brainstem or basal ganglia disturbances
Schaefer (1983)	Abnormal auditory brain stem response; visceral efferent dysfunctions

(table continues)

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Researcher	Finding
Blitzer, Lovelace, Brin, Fahn, Fink (1985)	Abnormal EMG findings including increased asynchronous activity, bursts
Ludlow (1986)	Abnormal EMG findings including high level muscle activity and abnormal activation timing
Shipp, Izdebski, Reed, Morrissey, (1985)	Abnormal EMG findings including intermittent increased adductor activity
Finitzo and Freeman (1989)	Multifocal, structural, metabolic, and/or electrophysiologic abnor- malities of central nervous system

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dysphonia. Thus, the thrust of a great deal of recent research has been to determine any perceived change in the degree of spastic dysphonia following some aspect of the surgery rather than attempting to specify any underlying cause or infer any new characteristics about the disorder.

Ratings of voice pre- and post-laryngeal nerve section (Dedo, 1976, 1977; Levine, Wood, Batza, Rusnov, and Tucker, 1979), ratings post anesthesia versus post RLN section (Izdebski, et al., 1979) ratings of voice post-laryngeal nerve crush (Biller, et al., 1979) ratings of voice as a function of the side of laryngeal paralysis (Izdebski & Dedo, 1981), long term follow-up ratings (Izdebski, et al., 1980; Aronson and Desanto, 1981) and ratings following a "selective" RLN section (Carpenter, Snyder, and Henley Cohen, 1981) have relied on solely perceptual impressions to make judgements about changes in patients. It is remarkable that not one of the aforementioned studies utilized any type of objective acoustic measure. We know the voice improves, or remains the same, or spastic dysphonia recurs after a while. We know little of the accompanying laryngeal or acoustic events associated with these changes.

What then do we know about the acoustics of spastic dysphonia? Wolfe and Bacon (1976) were the first to objectively study acoustic correlates of spastic dysphonia. Voice samples from two patients diagnosed as having spastic dysphonia were studied via broad band spectrographic analysis. Results were supportive of there being two types of spastic dysphonia as first suggested by Aronson (1973). An abductor type of spastic dysphonia was characterized by breathiness and viewed as partial disintegration of resonance bars and "fricative

fill" (vertical striations between resonance bars). A second patient demonstrated an adductor type of spastic dysphonia characterized by a "strain-strangle" voice which was viewed spectrographically as widely and irregularly spaced vertical striations. "To determine the relative occurrence of the three different types of phonation, the number of voiced sounds with spectrographic characteristics representing breathy strain-strangle, or normal phonation was expressed as a percentage of the total of all voiced sounds for each patient (p.327)." Analysis of these characteristics on spectrograms revealed that one patient demonstrated 40% breathy, 52% normal and 8% strain-strangle, for all voiced segments, whereas the second patient demonstrated 80% strain-strangle and 20% normal for all voiced segments. These findings would support the idea of there being at least two types of spastic dysphonia.

Zwitman (1979) presented voice symptoms, spectrographic data, indirect laryngoscope observations, and case histories for two cases of abductor type spastic dysphonia. Voice characteristics in this type of spastic dysphonia are weak voice, impaired ability to maintain normal loudness and increase volume, and decreased intelligibility, especially in ambient noise. Pitch is normal but quality is breathy. Phonation is intermittent and normal voice/voiceless patterns may be altered.

"Spectrographically abductor type spastic dysphonia is characterized by:

1. Aspiration times for initial stop consonants are two to three times normal. This is true for both voiced and voiceless consonants.

2. Less energy in the higher formant regions than for normal speakers. Energy in the area of the first fundamental and the first formant is similar to that seen in normals.
3. Voiceless stop consonants seem distinguished from voiced stop consonants by frication of the voiceless consonant. The total duration in milliseconds for words marked by aspiration is the same as words spoken normally. Aspiration time occupies and depletes vowel energy so that vowel production is shorter than normal." (p. 375)

Wolfe, Ratusnick and Feldman (1979) cite Cooper's (1973) descriptions of incipient and chronic spastic dysphonia. "Incipient" spastic dysphonia is characterized by similar but less severe symptoms than "chronic" spastic dysphonia. Incipient spastic dysphonia is further thought to be a forerunner of chronic spastic dysphonia. Wolfe, et al., sought to compare chronic and incipient spastic dysphonia on the basis of acoustic and perceptual characteristics.

Tape recorded speech samples were elicited imitatively from four patients with incipient spastic dysphonia and four patients with chronic spastic dysphonia. Diagnosis of "incipient" or "chronic" was based on length of time patients had the symptoms. Those with symptoms lasting longer than one year were classified as "chronic".

Analysis consisted of perceptual ratings along a seven point scale for eight parameters. Ratings were made by seven speech pathologists. Acoustic analysis consisted of evaluation of "laryngealization" and harmonic changes during selected voiced segments. Judgements were made from narrow and wide band spectrograms.

Comparison of mean perceptual values showed that differences between chronic and incipient spastic dysphonic patients were a matter of degree. No unique perceptual pattern was able to differentiate groups. Chronic patients were 30% more severe on ratings of "strain-strangle", 25% more severe on "effort" and 24% more severe for "stress". Chronic patients also had 24% less "fluency". Incipient patients were judged 19% more harsh. Definitions of the perceptual terms were not provided, thus their interpretation is left to the reader.

Acoustically, measures of laryngealization did not differentiate between the two groups. Laryngealization is described as biphasic vocal cord vibration characterized by widely and irregularly spaced striations on wide band spectrograms. Harmonic change is defined by the addition of noise components to the "main format range". Differences in total amount of harmonic change were also not significant between the two groups. Findings suggest symptoms of spastic dysphonia exist on a continuum of severity.

Cannito and Johnson (1981) cite the earlier spectrographic studies by Wolfe and Bacon (1976) and Zwitman (1979) and propose that spastic dysphonia may, in fact, not be amenable to a strict binary classification of abductor and adductor types. It is suggested that the characteristics of irregular harshness and

breathiness can exist along a continuum and both characteristics may be found in a single case of spastic dysphonia. A 58 year old female is presented as an illustration. Tape recordings were made of sustained phonation and conversational speech and broad band spectrograms were made and analyzed in a manner similar to that previously described by Wolfe and Bacon (1976). One hundred voiced phonemes were identified from the spectrograms and classified. Forty-two were classified as breathy, 28 were judged as "strain-strangle" characterized by irregularly spaced vertical striations, and 30 were judged as normal. There was no consistent pattern and strained and breathy often co-existed within one syllable. In addition, prephonation glottal clicks (glottal stoppages) were noted preceding "releasing" vowels, glides and nasals with an average of .3 seconds between the occurrence of the clicks and phonatory onset. Phonation was also noted, at times, to be initiated using hard glottal attack characterized by a plosive-like vertical striation. Speech rate was also found to be significantly slower than normal. It is suggested that under conditions of extreme laryngeal tension, there is a variable spasmodic adductor-abductor dysrhythmia associated with voice production.

In 1982, Fritzell, Feuer, Hagland, Knuttson, and Schiratzki, described their first experiences with nerve sectioning and evaluated the results subjectively as well as by acoustic and EMG analysis. Four subjects with spastic dysphonia were studied pre- and post-laryngeal nerve section. Results indicated that fundamental frequency was slightly raised in two patients, slightly dropped in one patient and markedly dropped in the fourth patient following laryngeal nerve section. Long-time-average-spectra (LTAS) showed minor and nonsystematic

differences between the pre-and post-operative tape recordings. Subjective evaluations of immediate results showed intermittent voice spasms to be gone and the patients reported it "easier to talk". Two patients had "normal voice", one patient had a voice tremor and a fourth patient's voice was judged "breathy" based on perceptual judgements.

Ludlow and Conner (1986) sought to undertake the most in-depth objective analysis of spastic dysphonia to date. Nine patients with spastic dysphonia were compared to 15 normal control subjects on different laryngeal tasks including maintaining phonation, intensity and frequency control, speed of phonation onset, coordination with supralaryngeal articulation and rate and length of phonation onset and onset during speech. Simultaneous audio tape and laryngograph recordings were obtained for each subject. Additional analysis included measurements of sound pressure levels and fundamental frequencies derived from spectrograms.

Results indicated that the only significant difference on measures of fundamental frequency and intensity (SPL) control was on the range in SPL. Spastic dysphonic patients tended to have a reduced range of intensity. A tendency to produce higher intensity when attempting the "soft condition" was also noted. It is hypothesized that the higher subglottal pressures that are usually associated with increased intensity may be compensatory mechanism to overcome excessive adductory force characteristic of vocal fold vibrations in spastic dysphonia. It was also speculated that spastic dysphonic patients may have poor control in achieving precision of adduction required for soft phonation.

Results also found that phonatory off times, measured by the length in milliseconds of stop-gaps in phonations, were increased. The rate of phonation on-off, defined as the number of glottal stop-gaps in phonation during 5 seconds, was also reduced. These results indicate that spastic dysphonic patients only have difficulty with vocal fold adduction for phonation onset in speech. Since there was no difference between spastic dysphonic and normal patients on any fundamental frequency control tasks, it was felt by these authors that cricothyroid and strap muscle function was not affected.

Results of the simple reaction time task showed a significant group effect for a greater time interval between laryngeal movement onset and phonatory onset in spastic dysphonic patients. The patients seem to take longer to initiate phonation following initiation of laryngeal movement. Difficulty is hypothesized to occur in obtaining correct adductory laryngeal position and force. Finally, laryngographic results showed extensive laryngeal pre-positioning a vertical movement prior to phonation in spastic dysphonia patients.

Izdebski (1984) used long-term-average-spectrum (LTAS) analysis and perceptual measures to investigate "overpressure" and breathiness in spastic dysphonia. Overpressure is described as inappropriately high subglottal air pressure for the resultant vocal output level, caused by hyperadduction. This is equivalent to "vocal strain" or "hyperfunctional voice". Breathiness indicates air loss irrespective of a fundamental tone presence. To obtain LTAS the speech signal was fed to 51 band pass filters, each 250 Hz wide. Results were analyzed by computer yielding LTAS plots showing sound power band level as a function

of frequency. Pre-and post-surgical audio recordings of 23 patients with spastic dysphonia were analyzed. Not surprisingly, perceptual comparisons of pre-surgical (laryngeal nerve section) and post-surgical samples using analysis of variance showed a significant decrease in overpressure and a significant increase in perceived breathiness. The LTAS analysis of the breathy, or post-surgical voice showed dominant or overriding white noise components due to air friction at the glottis. This friction occurs if glottal closure is incomplete or slow. The authors state that the average spectral slope of breathy voice will be steeper in decline than a "normal voice" and the opposite slope characteristic will apply to the voice with overpressure. The LTAS analysis did, in fact, support these findings. Statistically significant correlations were found between listeners' perceptual ratings of voice and LTAS ratings of both overpressure and breathiness. The authors caution, however, that the voice in spastic dysphonia may not always be predominantly overpressured, but may include degrees of breathiness, tremor and periodicity, as well. Perceptual judgements are, therefore, complex and difficult, but possible. Careful pretraining of judges is recommended. The study shows that perceptual judgements may be related to actual characteristics of the acoustic spectrum.

One study was done that utilized perceptual measures alone to establish spastic dysphonia as a unique entity that clearly differed from similar conditions. Aronson, Brown, Litin, and Pearson, (1968) compared spastic dysphonia to essential (voice) tremor and other neurological and psychogenic dysphonias using listener's perceptual judgements. Comparison of spastic dysphonia to essential

tremor showed that essential tremor had significantly lower distributions of mean ratings for irregular voice stoppage and intermittent strain-strangle dysphonia. Ratings were significantly higher in essential tremor for regular voice stoppage, constant strain-strangle, voice tremor in contextual speech, and voice tremor on prolonged vowels. Comparing spastic dysphonia and essential tremor to pseudobulbar palsy, ALS, cerebellar ataxia, and Parkinsonism showed many similarities among the groups but also revealed certain voice and articulation features that could be used to differentiate them. These features, including low pitch, pitch breaks, monopitch, harshness, strain-strangle, voice stoppage, and voice tremor in speech, vary with each specific disease. Comparison of neurologic to psychogenic voice disorders showed that the voice symptom alone is useful in differentiating between mute patients (writes notes) or aphonic patients (whispers) and neurologic patients. However, psychogenic patients who voice intermittently or who have constant dysphonia, cannot be separated from many patients with neurologic dysphonias.

### The Need for Objective Measurements of Voice

The majority of speech pathologists rely on perceptual impressions in diagnosing and managing voice disorders. Patients will often enter and terminate therapy based on the way their voice "sounds". Spastic dysphonia patients not only enter therapy based on perceptual judgements about their condition, but may end up with surgery as a result of their vocal condition. There currently exists

no objective index, test or rating scale for spastic dysphonia. The question must, therefore, be raised as to the reliability of subjective perceptual judgements in describing voice disorders.

The perception of "hoarseness" as a voice quality is a good example. According to Cooper (1973) it is the most common vocal symptom described. Of 956 patients seen in his clinic for voice problems, 91% of the functional voice patients and 94% of the organic voice patients complained of hoarseness. Yet, there are over 100 possible causes of hoarse vocal quality (Frank, 1940). Hoarseness can actually encompass several kinds and degrees of vocal abnormality (Moore and Thompson, 1965) with the only common factor being that it is phonatory in nature. Thus, to subjectively label a patient, as "hoarse" tells us little, if anything, about the patient's underlying laryngeal condition or function.

Thurman (1953) found that among trained listeners there was a tendency to confuse hoarseness and breathiness in labeling speech samples. It becomes evident, therefore, that one cannot make any underlying assumptions about the condition of vocal cords or the physiology of the phonatory mechanism based on a perceptual impression of a "hoarse" voice. Furthermore, even judgements about the degree or severity of hoarseness would be questionable without some sort of objective or quantitative measure.

Other questions of the reliability of perceptual assessment of voice have been raised. Wilson (1979) reported that voice ratings by speech pathologists show "varying reliability". Wilson cites Villareal (1950) who found a low percentage of agreement among judges agreed in rating voice quality. Wynter

(1974), is also cited, who found that judges agreed in judging pitch deviations and demonstrated "reasonable" agreement in judging hoarseness and stridency. Wynter found judges to be in low agreement on assessing breathiness and nasal resonance. Blaustein and Bar (1983) had three speech pathologists listen to randomly presented speech samples of 161 school aged children. Results indicated poor inter-judge agreement between listeners in just identifying the presence or absence of a possible voice disorder. Of 161 children screened, 45 children (28%) were identified as having a possible voice disorder by at least two of three judges. Yet, there were an additional 51 children (31.7%) that were "failed" by only one of the three judges.

Ludlow and Bassich (1983) note that perceptual measurements are difficult to standardize over time and across different settings and prevent the maintenance of adequate inter-rater reliability. Furthermore, perceptual systems are difficult to replicate and limit the comparability of different investigator's results.

Ludlow and Bassich sought to determine whether acoustic measures of the speech of dysarthric patients differentiated their patterns of impairment from normals in the same manner as perceptual ratings of the same speakers. Two groups of dysarthria patients (She-Drager Syndrome and Parkinson's) and one group of matched controls were subjects. Results indicated that both the acoustic and perceptual assessment systems were capable of discriminating accurately between the two types of dysarthric patients. It is noteworthy, however, that only the acoustic system was capable of noting vowel voicing errors, consonant voicing errors, and rate of phonation onset and offset as an impairment to both groups of

dysarthric patients. There was no perceptual rating capable of distinguishing these characteristics. It is suggested that acoustic measures may be more specific for the analysis of different speech production factors in a patient's speech.

Moran and Gilbert (1984) correlated the relationship of the Wilson Voice Profile (WVP), a perceptual rating system to the specific aerodynamic and acoustic characteristics of voice and found that the correlation "may not be as strong as users of the WVP might expect". A follow-up study by these investigators correlated WVP ratings to objective assessments of laryngeal function. Acoustic measures included fundamental frequency, noise-to-harmonic component ratio, intensity and maximum rate of change of fundamental frequency. Rate of airflow and volume flow per syllable were also measured. The perceptual data consisted of ratings of laryngeal function (tension and air loss) and pitch of each of 25 subjects according to the Wilson Voice Profile. Five subjects had normal voices and 20 subjects demonstrated "disordered voices". The judges were instructed to use a one to four point severity scale whenever a hoarse voice was encountered. Results indicated that perceptual ratings were often related to interactions of various aerodynamic and acoustic variables and not to single isolated factors. "Undetermined" factors were felt to contribute to perceptual ratings on the Wilson Voice Profile.

Bassich and Ludlow (1986) conducted a study where they sought to assess the amount of listener training required to achieve adequate reliability, the stability of intra- and inter-judge reliability over time, and the validity of the rating system used for differentiating pathological from normal voices.

Results indicated that extensive training procedures were required and including the judges' participation in the development of the rating system used. The authors further report that even after inter-judge agreement across all dimensions met the training criteria, the judge's ratings did not remain stable. The mean percentage of inter-judge agreement was shown to be below 80%. It was not specified how inter-judge agreement was arrived at. Intra-judge reliability was below 75% on one half of 13 dimensions used "Strain-strangle" was one of only three dimensions with an interjudge reliability agreement coefficient greater than .90. "Strain-strangle" also had a high intra-judge reliability coefficient. The authors conclude that the task of rating voice quality perceptually is difficult and requires professional experience and sophistication.

One can draw only certain conclusions from the limited research available on the reliability of perceptual voice assessment. It is agreed that perceptual assessment is an important measure to the speech pathologist. It is also apparent, however, that the inference of acoustic and physiologic abnormalities from perceptual dimensions can be difficult and uncertain (Lebrum, Buysens, Henneaux, 1973). It is the vocal signal that can, and should, be objectively quantified and described to allow for more precise diagnosis and greater reliability. Perceptual assessment alone can in no way meet these needs.

#### Voice Onset Time as a Measure of Pathological Speech

It is well documented that voice onset time can be used as a sensitive means to distinguish normal from pathological speech production systems.

Lesions to the nervous system resulting in aphasia, dysarthria, and apraxia, fluency disorders, cleft palate speech, normal aging and voice disorders; have been shown to result in voice onset time durations that are significantly different from unimpaired matched populations.

When researchers began to focus attention to the larynx of stutters, voice onset time was used as an easily obtainable, objective, non-invasive, acoustic measure of laryngeal coordination.

Metz, Conture and Caruso (1979) demonstrated that mean VOT's for 6 of 18 sound clusters studied were significantly different from those of fluent speakers. The results, however, were felt to be equivocal and it was felt that VOT can probably be as useful in providing insights into stutterers' fluent productions, as well as their dysfluent utterances. Metz, et al., 1983, further demonstrated that changes in voiced stop consonant VOT's and voiceless stop consonant VOT's were noted following five weeks of daily stuttering therapy.

Agnello, Wingate and Wendell (1979) showed that children who stuttered had longer VOT than children who did not stutter. Adult stutterers were also significantly slower in VOT than non-stuttering controls. Hillman and Gilbert, (1977), used 10 adult stutterers and 10 controls to demonstrate a 9 msec mean difference in VOT with stutterers being slower. Stutterers have clearly been shown to exhibit VOT differences when compared to normal speakers.

Healey and Ramig (1986) also showed that VOT can be used to differentiate between normal and abnormal speakers. Results of VOT measurements from 22 stutterers repeating the phrase "ipi saw ipi" were compared

to VOT measurements from a matched group of non-stutterers. Results showed a significant between group effect with the stutterers exhibiting longer VOT's. The authors did not account for this difference.

Voice onset time measures were used in 1971 (Rolnick & Hoops, 1971) to study the effects of wearing a palatal appliance on the duration of plosives in cleft palate speakers. Measurements obtained from 20 subjects showed significant increases in the duration of plosive parameters when the subject removed the oral prosthetic appliance designed to aid in velopharyngeal function. No measurements were obtained from normals.

In 1974, Kent and Netsell used VOT measurements as part of their acoustic analysis of a 59 year old patient with ataxic dysarthria. Voice onset time for initial /p/ and duration of frication for /f/ were not significantly different from normal speakers. The durations of vowel steady states were found to be abnormally prolonged.

Kent, Netsell and Abbs (1979) studied acoustic characteristics of dysarthria associated with cerebellar disease. Voice onset time was one measurement used. Results showed that "in most severe forms [of dysarthria] segment durations were as much as seven times the segment duration recorded for normals. Increasing severity of the dysarthria was marked by increased duration of all segments, including some, like voice onset time, that were of normal duration in the speech of less severe dysarthrics (p. 643).

Freeman, Sands and Harris (1978) examined VOT used to test the hypothesis that an apraxic's problem was in coordinating upper articulatory and

laryngeal events. Results showed that VOT's of an apraxic subject were markedly different from those of normal subjects. While Freeman, et al., found VOT lag times larger than normals for voiced stops and shorter than normals for voiceless stops, Collins, Rosenberg, and Wertz, (1983) found no significant differences in voice onset times for initial /p/ between 11 normal and apraxic subjects.

Voice onset time has also been used to differentiate varying degrees of "production deficit" in adult aphasics. Blumstein, Cooper, Goodglass, Statleader, and Gottlieb, (1980) were able to show that normal control subjects made neither phonetic nor phonemic errors based on VOT definition. Aphasic subjects showed varying degrees of deficit with Wernicke's aphasics having the mildest impairment, conductor aphasics having more errors, and Broca's aphasics having the greatest number of errors based on VOT measurements. It was further suggested that the impairment in Broca's aphasics are manifested by altered VOT might be considered as a "deficit in articulatory timing of vocal fold vibration".

The use of voice onset time as a measure of temporal control as it relates to changes in the neural, respiratory, phonatory and articulatory system was recently used to study a geriatric population. Although not considered "pathological", there is evidence that fine motor coordination required to maintain the articulatory laryngeal adjustments necessary for voice and speech production change with normal aging. Hence, Sweeting and Eaken (1982) felt that VOT would be an appropriate measure to identify these changes.

Results showed that although subjects' mean VOTs did not change significantly as a function of age, subjects, individually and as a group, showed

significantly increased variability in temporal control. The older subjects had more difficulty in hitting targets.

It is evident that researchers have used voice onset time measures to distinguish between a variety of normal and aberrant conditions. It is a useful temporal measurement to specify the coordinations between laryngeal and supra-laryngeal events during speech. Voice onset time has proven to be a sensitive measure enabling a micro-analysis of the acoustic output of the patient (Blumstein, et al., 1980).

## Chapter IV

### Method

#### Subjects

The experimental group was comprised of six subjects diagnosed as having "adductor spastic dysphonia". Three males and three females were included. Each subject was diagnosed as having adductor spastic dysphonia by at least two otolaryngologists and two speech pathologists. The subjects were individually interviewed to collect pertinent case history information. All subjects were in reportedly good health except for the voice disorder. Recent indirect laryngeal examination of each of the subjects revealed no visible vocal cord pathology. No subject reported any prior history of positive vocal findings (e.g. nodule, polyp, contact ulcer, bowed vocal cord, etc.). No history of neurological illness was reported in any of the subjects. Three of the subjects reported having neurological examinations with normal results. Subjects ranged in age from 51 years to 63 years with a median age of 54 years. Reported duration of spastic dysphonia ranged from 7 years to 18 years. Table 2 summarizes age, sex, and duration of spastic dysphonia for each of the subjects.

Three matched controls were used for the study. Two males (54 years and 58 years) and one female (55 years) were included. Each of the controls had no present or prior history of voice disorder or neurological illness. All were in reportedly good health. Indirect laryngoscopic screening of the control subjects revealed no laryngeal pathology.

Table 2

Age, Sex, and Duration of Disorder by Subject

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Subject	Sex	Age	Duration of Disorder
HZ	F	59	18 years
RM	M	63	10 years
KH	M	54	6 years
EK	F	51	7 years
GG	F	54	11 years
MS	M	52	9 years

---

### Spectrographic Analysis

Data Collection. Stimulus material consisted of the six sentences comprising the "Rainbow Passage" (Appendix A ). Each subject was seated in a soundproof room and was instructed to read the passage at a comfortable rate and loudness. Subjects were recorded on an Ampex (AG - 440B) tape recorder running at 7 1/2 CPS. A constant distance of approximately 12 inches was kept from an Altec (681A) microphone. Each of the six sentences of the Rainbow Passage was then spliced sentence by sentence to allow for easier analysis.

Selection of Target Segments. Specific targets were identified which consisted of consonant segments where a voiceless consonant immediately preceded a vowel, /r/, or /l/. Segments where two voiceless consonants occurred in combination prior to a vowel were excluded (e.g. stay). The resulting segments selected for analysis are presented in Appendix B.

Voice Onset Time and Devoicing Measurements. Wideband spectrograms were made of phrases containing the targeted segments. Tapes were played on a Teac tape recorder and spectrograms were made on a Kay Digital Sona-Graph (7800) with a Kay Sona-Graph Printer (7900). The targeted segments were further specified by identifying their locations on the spectrograms and listening to the segments. A second set of wide band spectrograms was then produced

using the 1/2 speed expansion capabilities of the Sona-Graph. This would allow for more precise measurements.

Voice onset time measurements, such as those described by Klatt (1975) and Lisker and Abramson (1964) were used for /p/, /t/, and /k/. These consisted of measuring the spectrographic interval from oral release to the onset of voicing striations in the spectrogram. In those cases where voicing was ongoing at oral release of the stop, VOT was given a value of zero.

A second measure of voicing was obtained for /s/, /f/, and /ʃ/. This was calculated by measuring the devoiced interval from the onset of frication to the onset of voicing for the vowel. Thus, if the frication interval was entirely voiced, the "devoicing interval" would again be zero. Voice onset time and devoicing interval measurements are presented in Appendices C and D.

### Perceptual Judgement Task

Listeners. Judges consisted of 6 speech pathologists. All judges are employed within a hospital setting and have experience in the diagnosis and treatment of a wide variety of voice disorders. All judges held certificates of clinical competence from the American Speech and Hearing Association.

Presentation of Voice Samples. Voice samples from each experimental subject were prepared for presentation to listeners. Two samples were obtained from each subject. One sample consisted of the reading of the Rainbow Passage

from which the VOT and devoicing measurements were obtained. The second sample consisted of two minutes of spontaneous speech from each subject.

Two tapes were prepared for presentation, with each tape consisting of three reading segments and three spontaneous speech segments. Each subject was heard only once on each tape, either reading or speaking. Samples were presented randomly, but a reading segment alternated with a speaking segment on each tape. Table 3 shows the order of presentation of samples for the two stimulus tapes.

Stimulus Presentation and Rating of Voice Sample. Taped samples were presented to each listener in sound field from a SONY TC-270 reel-to-reel tape recorder at 7 1/2 CPS. A total of two listening sessions were scheduled three weeks. Tape A was presented at the first listening session and Tape B was presented three weeks later. At each session judges were instructed to listen to each voice sample and to assign a numerical rating from one to seven. Number one represented "normal speech" and a rating of seven represented a "severe deviation from normal". This system is commonly in use for perceptually rating dimensions of speech and was first suggested by Darley, Aronson, and Brown in 1969. Each judge was asked to rate each sample for 2 separate dimensions. Dimension I was a rating of overall severity for the voice disorder "spastic dysphonia" and dimension II was a rating for "strain-strangle" quality. The "strain-strangle" rating is a phonatory quality also first described by Darley, Aronson & Brown (1969).

Table 3

Tape Presentation for Perceptual Task

Sample	Tape A		Tape B	
	Subject	Task	Subject	Task
1	MS	Reading	RM	Spontaneous
2	EK	Spontaneous	EK	Reading
3	HZ	Reading	HZ	Spontaneous
4	GG	Spontaneous	KH	Reading
5	RM	Reading	MS	Spontaneous
6	KH	Spontaneous	GG	Reading

Judges rated only one dimension ("Strain-Strangle" or "Overall Severity") for each tape presentation. After the initial two presentations of Tape A, there was a 10 minute interval and the procedure was once again repeated with listeners re-rating the tape presented. Three weeks later the entire procedure was repeated for the second presentation tape. Judges' individual ratings are presented in Appendix E.

## Chapter V

### Results

#### Voice Onset Time Measurements for /p, t, k/

Table 4 presents voice onset time measurement distributions and the percentage of each distribution that falls within each 25 millisecond ranges for the normal subjects. Table 5 presents the same information for the six spastic subjects. All voice onset time measurements are grouped by sound. Each "#" represents the total number of instances of a VOT measurement falling within the millisecond range noted.

Histograms were plotted to illustrate the data from Table 4 and Table 5. Plotting the two histograms together (Fig. 1) enables comparison of distribution percentages between spastics and the control group for p, t, and k phonemes.

#### Devoicing Interval Measurements for /s, f, ʃ/

Table 6 presents measurements of devoicing intervals for s, f, and ʃ for the normal group. Table 7 presents the same information for the spastic group. As with the stops, a histogram (Fig. 2) was plotted to enable comparison of distributions between the control and the spastic group.

#### Group Comparisons of VOT and Measures of Devoicing by Sound

Analysis of raw data sought to determine if significant differences could be found when comparing normal speakers versus spastic dysphonic speakers with

Table 4

Distribution and Percentage of Voice Onset Times for /p, t, k/ for Normals

Range (msec)	p		t		k	
	#	%	#	%	#	%
0 - 25	2	13.3	0	0	1	11.1
26 - 25	6	40	6	66.7	4	44.4
51 - 75	5	33.3	2	22.2	3	33.3
76 - 100	2	13.3	1	11.1	1	11.1
Totals	15	99.9%	9	100%	9	99.9%

Table 5

Distribution and Percentage of Voice Onset Times for /p, t, k/ for Spastics

Range (msec)	p		t		k	
	#	%	#	%	#	%
0 - 25	21	70.0	8	44.4	11	61.1
26 - 50	4	13.3	4	22.2	3	16.7
51 - 75	0	0	2	11.1	0	0
76 - 100	0	0	1	5.6	0	0
101 - 125	0	0	1	5.6	0	0
126 - 150	0	0	2	11.1	1	5.55
151 - 175	3	10.0	0	11.1	1	5.55
176 - 200	2	6.7	0	0	1	5.55
> 200	0	0	0	0	1	5.55
Totals	30	100%	18	100%	18	100%

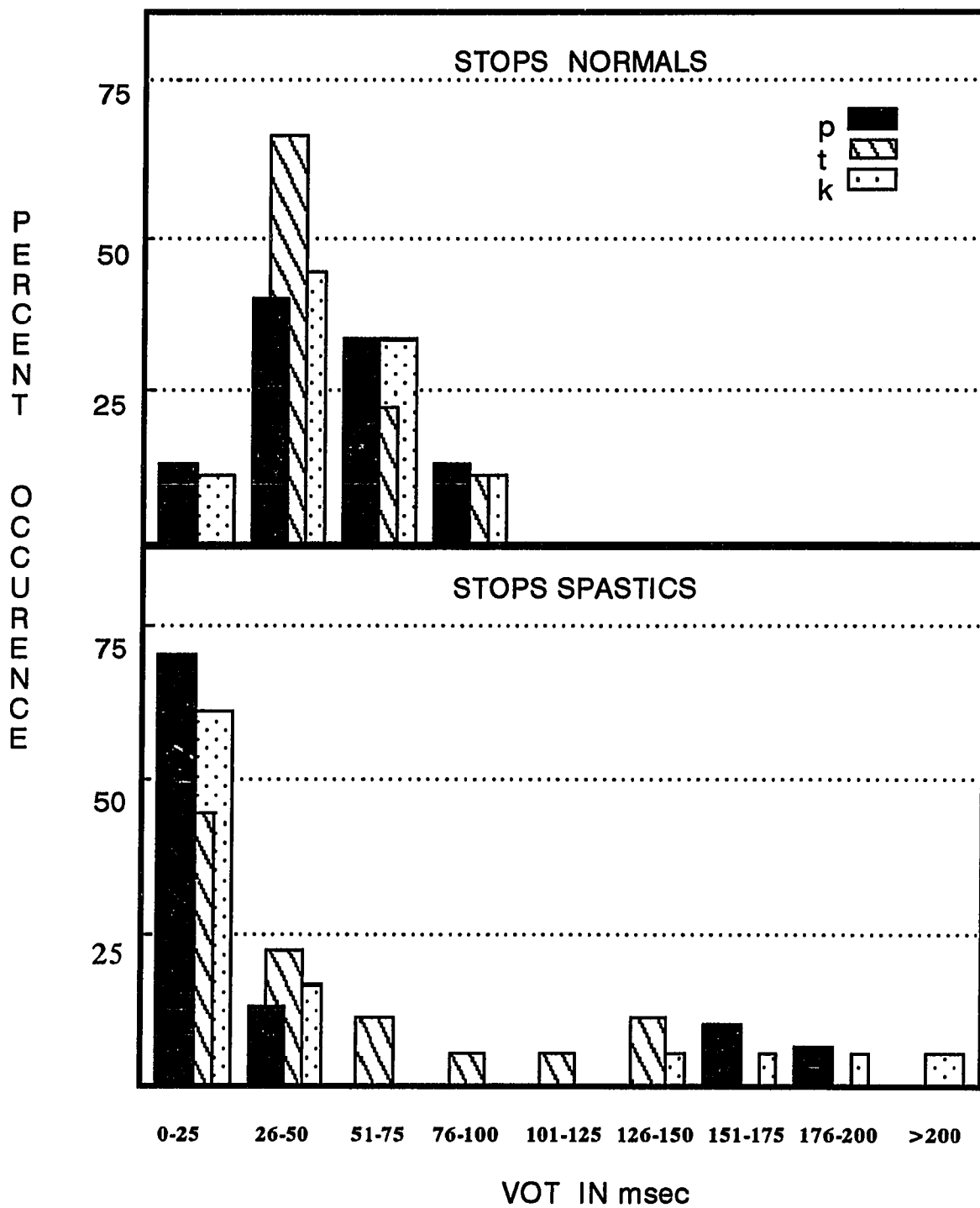


Figure 1. Percent Occurance of VOT by Duration for p, t, k

Table 6

Distribution and Percentage of Devoicing Intervals for s, f, ʃ, for Normals

Range (msec)	s		f		ʃ	
	#	%	#	%	#	%
0 - 50	0	0	0	0	0	0
51 - 100	0	0	7	46.7	0	0
101 - 150	6	66.7	6	40.0	2	66.7
151 - 200	3	33.3	2	13.3	1	33.3
201 - 250	0	0	0	0	0	0
> 250	0	0	0	0	0	0
Total	9	100%	15	100%	3	100%

Table 7

Distribution and Percentage of Devoicing Intervals for s, f, ʃ for Spastics

Range (msec)	s		f		ʃ	
	#	%	#	%	#	%
0 - 50	3	16.7	13	43.3	0	0
51 - 100	0	0	3	10.0	0	0
101 - 150	5	27.7	4	13.4	2	33.3
151 - 200	3	16.7	6	20.0	0	0
201 - 250	3	16.7	3	10.0	1	16.7
>250	4	22.2	1	3.3	3	50.0
Totals	18	100%	30	100%	6	100%

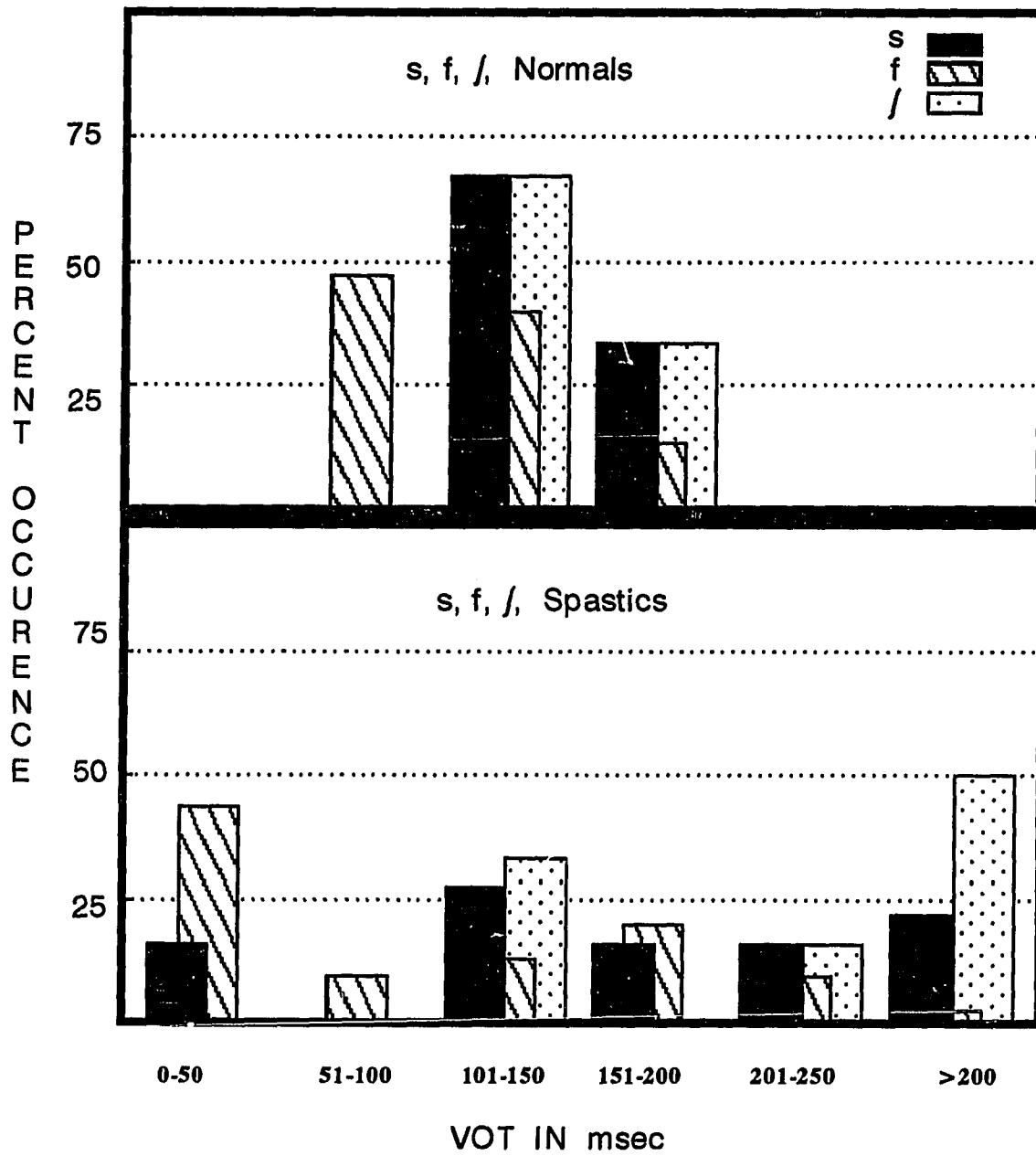


Figure 2. Percent Occurrence of Devoicing Intervals by Duration for s, f, /

respect to VOT measurements and measures of devoicing intervals on a sound by sound basis.

Mann - Whitney U - Wilcoxon Rank Sum W Tests were calculated for normals versus spastics on a sound by sound basis. Significant differences were found for /p/ ( $u=79$ ,  $p < .05$ ), /t/ ( $u=34.5$ ,  $p < .05$ ), /k/ ( $u=18$ ,  $p < .05$ ), and /f/ ( $u=89.5$ ,  $p < .05$ ). Group comparisons of /j/ and /s/ did not find a significant difference. Results of Mann - Whitney U tests are presented in Table 8.

When spastics are compared to normals for all stops (p, t, k) using a Kruskal-Wallis 1-way Anova (Table 9), results indicate a significant difference in VOT measurements. Mean ranks significantly differed between /p/, /t/, and /k/ for normals versus spastics (Chi Square = 30.04,  $p < .01$ ). A significant difference between groups was also noted when all sounds measured (p, t, k, j, f, s) were compared as a group (Chi Square = 45.37,  $p < .01$ ).

#### Analysis of Perceptual Judgement Task

An overall mean perceptual rating of spastic dysphonia was calculated for each subject in the following manner. Separate mean perceptual ratings for Trial 1 and Trial 2 for both "Overall Severity" and "Strain-Strangle" quality were obtained. The four mean perceptual ratings were then used to compute a single mean for each subject. Means to be used in the acoustic comparison part of the study were computed for the reading task, as the segments from which VOT and devoicing measurements were obtained came directly from that task. Table 10 presents mean ratings for individual tasks and the overall mean for each subject.

Table 8

Comparison of VOT/Devoicing Measures - Normal vs. Spastics, Mann -Whitney U

Sound	u	p
p*	79.0	< .05
t*	34.5	< .05
k*	18.0	< .05
ʃ	4.5	> .05 (.2619)
s	45.5	> .05 (.0672)
f*	89.5	< .05

\* significant

Table 9

Kruskal-Wallis 1 Way Anova VOT/Devoicing Measures - Normals vs. Spastics

Sound	X <sup>2</sup>	p
ptk	30.04	< .01
ptkfs	45.37	< .01

Table 10

Perceptual Ratings and Overall Means for Reading Task

Subject	Overall Severity		Strain-Strangle		Overall
	1	2	1	2	Mean
MS	4.5	3.3	3.3	3.3	3.6
EK	5.2	5.2	4.2	4.2	4.7
HZ	4.7	5.0	4.8	4.7	4.8
KH	5.7	5.7	5.2	5.2	5.5
RM	5.3	5.0	4.7	4.7	4.9
GG	6.8	6.8	7	7	6.9

Mean ratings were also computed for listeners' ratings of Overall Severity and Strain-Strangle judgements. Table 11 displays comparisons of Overall Severity and Strain-Strangle by subject.

Pearson Correlation Coefficients were computed to determine the degree of interjudge reliability in perceptually assessing the parameters of "Strain-Strangle" and "Overall Severity" for the reading task. Each individual judge's rating for every listening task was used to calculate the reliability measures.

Table 12 displays the interjudge reliability for the reading task as determined by the correlation between the judges collapsed across trials and overall severity and strain-strangle qualities.

Table 13 displays the reliability of overall severity ratings for reading collapsed across both trials and tasks. Table 14 displays the reliability of strain-strangle ratings for reading.

Perceptual ratings obtained for the spontaneous speech task were also analyzed. Table 15 presents the mean listeners' ratings for Overall Severity and Strain-Strangle quality by trial. An overall mean for the spontaneous speaking task was then computed for each subject by averaging tasks and trials.

Pearson Correlation Coefficients were then computed to determine interjudge reliability for the speech task collapsed across trials. Table 16 presents the resultant coefficients.

Table 11

Comparison of Mean Overall Severity Rating to Mean Strain-Strangle Rating

Subject	Overall Severity	Strain-Strangle
MS	3.90	3.30
EK	5.20	4.20
HZ	4.85	4.75
KH	5.70	5.20
RM	5.15	4.70
GG	6.80	7.00

Table 12

Interjudge Reliability for Reading Task

	J1	J2	J3	J4	J5	J6
J1	1.000	.4128	.5588	.5375	.4478	.6390
J2		1.000	.7667	.7772	.8043	.7579
J3			1.000	.7778	.6745	.7659
J4				1.000	.5953	.8002
J5					1.000	.6266
J6						1.000

(Average interjudge reliability .6753)

Table 13

Interjudge Reliability for Overall Severity Ratings

	J1	J2	J3	J4	J5	J6
J1	1.000	.4698	.5404	.5789	.5825	.7430
J2		1.000	.8574	.7021	.7327	.3855
J3			1.000	.8182	.5205	.5371
J4				1.000	.4049	.6037
J5					1.000	.4385
J6						1.000

(Average Interjudge reliability .5912)

Table 14

Interjudge Reliability of Strain-Strangle Ratings

	J1	J2	J3	J4	J5	J6
J1	1.000	.5476	.6623	.6070	.6182	.6802
J2		1.000	.8795	.8517	.4277	.7390
J3			1.000	.8767	.5235	.9162
J4				1.000	.5388	.8882
J5					1.000	.5444
J6						1.000

(Average Interjudge reliability .6867)

Table 15

Perceptual Ratings and Overall Means for Spontaneous Speech Task

Subject	Overall Severity		Strain-Strangle		Overall
	1	2	1	2	Mean
MS	4.5	4.5	4.3	4.3	4.4
EK	5.5	4.7	3.2	3.5	4.2
HZ	5.0	5.0	4.8	4.8	4.9
KH	6.3	6.2	6.3	6.2	6.3
RM	5.5	5.3	4.6	4.7	5.0
GG	6.8	7.0	7.0	7.0	6.9

Table 16

Interjudge Reliability Ratings for Spontaneous Speech Task

	J1	J2	J3	J4	J5	J6
J1	1.00	.588	.651	.589	.687	.764
J2		1.00	.908	.798	.328	.776
J3			1.00	.905	.351	.837
J4				1.00	.390	.780
J5					1.00	.536
J6						1.00

(Average Interjudge reliability .6592)

### Comparison of Perceptual Ratings and Acoustic Measures

A salient feature that became apparent during analysis of acoustic data was the inability of the spastic dysphonic subject to devoice. This "devoicing failure" occurred for all spastic dysphonic subjects. Table 17 presents a listing of each subject's mean perceptual rating and the number of instances of devoicing failure (zero VOT).

Analysis sought to determine if a significant relationship could be established between mean severity ratings and instances of devoicing failures. Pearson correlation coefficients failed to establish a significant correlation between the two measures.

A plot of devoicing failures against mean perceptual ratings is presented in Figure 3. A tendency for mean perceptual severity rating to increase as the number of devoicing failures increases is clearly evident for 5 of the 6 subjects, but one subject is clearly out of line.

Table 17

Perceptual Ratings and Devoicing Failures

Subject	Mean Rating	Devoicing Failures (0's)
MS	3.6	7/20
EK	4.7	9/20
HZ	4.8	8/20
KH	5.5	4/20
RM	4.9	9/20
GG	6.9	14/20

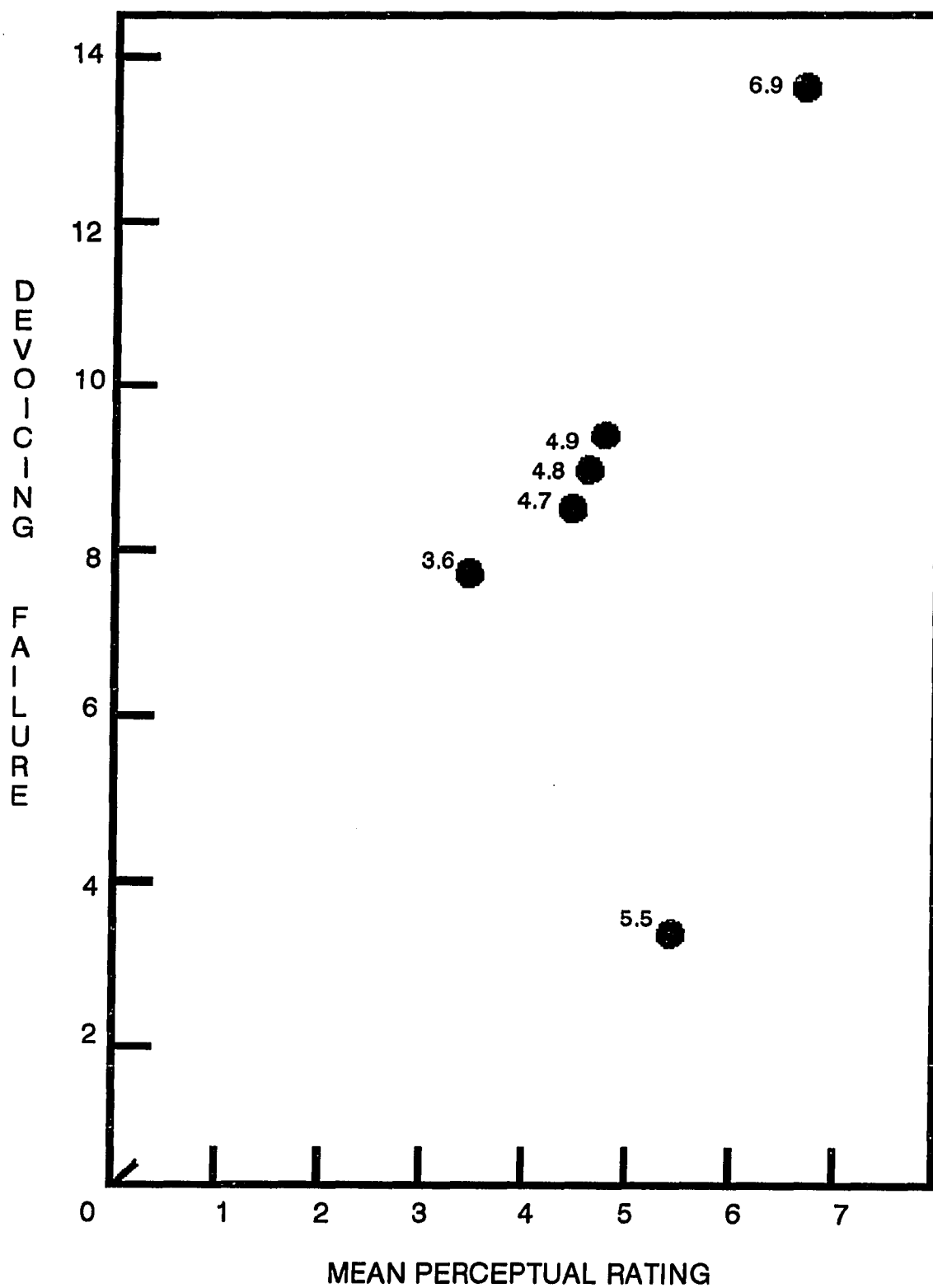


Figure 3. Perceptual Ratings Versus Devoicing

## Chapter VI

### Discussion

The first question addressed by the present study was to determine whether or not patients with adductor spastic dysphonia differed from normals in their ability to "devoice" during a connected speech task. Results indicated that spastic dysphonic patients differed significantly in measurements of "devoicing". Spastic dysphonic patients, as a group, demonstrated significantly shorter devoicing intervals than their matched control group. Difficulty with the devoicing gesture was noted in each of the six subjects studied with each subject also demonstrating the complete inability to devoice in at least two instances.

To date, there has been no actual report of VOT or devoicing measures in spastic dysphonic patients. The number of objective acoustic studies in relation to the amount of all published data on spastic dysphonia is quite small. There have, however, consistently been some suggestions that the moment of "voicing" or "devoicing" may be of specific interest in spastic dysphonia.

Wolfe and Bacon (1976) in the first objective spectrographic study of spastic dysphonia make short mention of the moment of voicing. They state "the disruption of articulation in the speech produced by the patient . . . was demonstrated also by the delayed onset of voicing of some vowel sounds (p. 330)". They state that voicing delays in one patient were observed in 15 instances following an initial plosive or fricative consonant and that in 14 of the 15 instances the phonation of the vowel was produced without breathiness. These

early observations, describing abnormalities at voicing onset, are supported by the results of the present study which also found voice onset abnormalities. One subject in this study, KH, clearly had delayed voicing onset as described by Wolfe and Bacon.

Zwitman (1979) spectrographically looked at abductor spastic dysphonia and noted aspiration times for initial stop consonants to be two or three times normal. Two years later Cannito and Johnson (1981) described prephonation glottal "clicks" (stoppages) preceding releasing vowels, glides and nasals. An average of .3 seconds occurred between the click and phonatory onset. Phonation was also noted to be initiated using a "hard" glottal attack characterized by a plosive-like vertical striation. Thus, while no specific mention of VOT or devoicing measures has been made, there frequently exists a recurring suggestion that a disruption may occur at the onset of voicing in spastic dysphonic patients. This line of research was not carried through.

Reich and Till (1983) studied reaction times in idiopathic spastic dysphonia and found that SD patients were significantly slower than normals in phonatory reaction times in producing the word "upper", but not during vowel production or non-speech related tasks. The complex motor programming for "upper" requiring "precise coordination of a variety of linguistic rule-governed behaviors in the respiratory, laryngeal, and articulatory systems", is believed to be compromised in SD patients. This was further supported by Ludlow et al. (1986) who speculated SD as a movement - control disorder affecting vocal fold adduction for phonation onset.

Finitzo and Freeman (1989), in the article previously referenced, also discuss the issue of voicing onset in these patients. Results of fiberoptic studies of vocal tract movements showed "multiple repetitions of prephonatory posturing proceeded onset of phonation (p. 546)". Their subjects appeared to "initiate phonation, and failing, restarted the process". The authors felt that these observations were consistent with subjective reports of difficulties or "blocking" when attempting to initiate phonation.

The ability to voice and devoice during spontaneous speech is a task requiring precise, rapid, laryngeal adjustments. The results of this study show that spastic dysphonic patients differ from normals by exhibiting abnormal VOT measurements and devoicing intervals. Numerous studies exist noting abnormal muscle activation patterns and timing irregularities in spastic dysphonia. There is strong evidence that the muscles involved in vocal fold adduction are affected in SD patients (Ludlow, 1986, Shipp, Izdebski, Reed, and Morrissey, 1985), and there is recent evidence that the PCA may also be affected in SD patients. Hartman (1987) reports increased PCA activity timed in SD patients. The shorter VOT and devoicing measures obtained in this study are further support for spastic dysphonia being a voluntary movement disorder with the complex voicing gestures being poorly timed, compensated for, or eliminated totally in spastic dysphonic patients.

One of the many controversial issues surrounding spastic dysphonia concerns the notion of there being an abductor type, an adductor type, or as Cannito and Johnson (1981) suggest a "continuum" type of spastic dysphonia

where breathiness and strain qualities can coexist within one patient. The results of this study show individual spastic dysphonic patients to be consistent in their symptoms. Although some inconsistency in symptom manifestation may be noted across patients, individual patients were highly consistent in their voicing abilities. The idea of abductor and adductor spasms rapidly cooccurring within one patient during one syllable, as previously reported, appears highly unlikely.

A careful scrutiny of the entire abductor/adductor controversy reveals a literature base of only three articles where the classification schema is supported spectrographically. The total number of patients involved was five and the patient who was described as having the "continuum" disorder was noted to have had three hours of speech therapy per week for two years. The patient also had previous diagnoses of hysterical aphonia and two prior surgeries for vocal nodules. Both of which are never reported in association with spastic dysphonia. The etiology is described as psychogenic. It is from this perspective that the controversy over diagnostic "types" of spastic dysphonia emerged.

The subjects in this study all had confirmed diagnoses of adductor spastic dysphonia and all presented with the classic strain-strangle, choked, voice quality. All subjects were found to exhibit difficulty with the timing of the initiation of voice following a voiceless plosive, difficulty devoicing for a voiceless consonant, or exceeding long voiceless consonants prior to a vowel. These difficulties are viewed as being related to a deficit in the ability to properly execute the properly timed adduction of the vocal folds and improper maintenance of hyperadduction during intervals when the cords should abduct. Thus the term "adductor" spastic

dysphonia would appear an accurate diagnostic nomenclature, pin pointing the nature of the problem. There certainly may exist a rarer, "mirror opposite", abductor spastic dysphonia but further documentation is called for. The existence of a mixed spastic dysphonia would seem unlikely. It is more likely that a primary and predominate adductor or abductor dysfunction exists and that compensatory mechanisms may produce random opposite characteristics as the individual struggles to maintain phonation.

The heterogeneity of spastic dysphonic patients, effects of voice therapy, and each patients' own compensatory strategy, remain variables to be accounted for in any research into spastic dysphonia. Thus, while the noted difficulties with voicing in spastic dysphonic patients were seen across all subjects, individual differences were noted. Differences among spastic dysphonic subjects seem to be the most consistent finding in research into this disorder.

While five of the six subjects performed similarly with respect to VOT and devoicing data, a sixth subject's spectrographic analysis was unlike any of the others. Subject KH had fewer devoicing failures (20%) than any of the other subjects studied and had the second highest mean perceptual severity rating. KH's attempt at voice onset time maneuvers and devoicing gestures showed numbers that ranged from 136 msec to 308 msec and were often three times higher than the normal group. This spastic dysphonic was clearly performing differently from the normals and the spastic dysphonic group. Whether this different laryngeal behavior is a direct manifestation of a specific type of muscle abnormality or a compensatory strategy on the part of the patient can only be speculation. It is,

however, additional support for the idea that spastic dysphonia is a disorder characterized by subgroups demonstrating various laryngeal symptoms. More significantly, though, it illustrates how measurements of VOT might be used not only to differentiate spastic dysphonics from normals, but also to identify differences in laryngeal behavior within the spastic dysphonic group.

The present study also sought to assess spastic dysphonia perceptually and to determine if the perceptual measures obtained would in some way correlate with objective measures of voice. Although a statistically significant correlation was not demonstrated, a strong tendency was noted for the mean perceptual rating of severity to increase as measures of voicing difficulty increased.

Though the main purpose of the perceptual portion of this study was to attempt to correlate an objective acoustic measure with a perceptual measure, there are a number of interesting findings in the perceptual data that are noteworthy.

The first observation is that for five of the six subjects the mean perceptual rating for the "strain-strangle" judgement was lower than the rating for "Overall Severity". This lower rating was one full point lower for one subject and at least .5 points lower for an additional 2 subjects. The most severe subject was rated .2 points higher for Strain-Strangle.

One speculation is that judges tend to be freer in assigning a more severe rating of a voice disorder than when asked to rate a specific aspect of a vocal quality. Yet, in a very severe case, it is apparently not as difficult to rate at the most severe end of the rating scale.

A second observation concerning the perceptual data is that for all but one subject, overall mean perceptual ratings of severity for the spontaneous speech task were greater than the reading task. In two instances this difference was as high as .8 points. In two instances it was just .1 point. Once again, the most severely rated subject, GG, attained a 6.9 average rating for speech and 6.9 average rating for reading. This again points out that it is easier in the most severe cases to assign high severity ratings despite the task or condition. The higher severity ratings for spontaneous speech against reading may also point to some degree of task difficulty as it relates to spastic dysphonia.

A third area of interest concerns the degree of interjudge reliability with regard to various tasks (overall severity versus strain-strangle) and samples (reading versus speech). Average interjudges reliability for the reading task was .6592 compared with an average interjudge reliability of .6753 for the spontaneous speech task. There is no appreciable difference between averaged reliability ratings. Results suggest that a reading sample is as reliable as a spontaneous speech sample for judge's evaluations. Judges were equally reliable in assessing both samples.

Average interjudge reliability was found to be .6867 for the task of evaluation "strain-strangle" quality compared to an average of .5912 for the "overall severity" judgement. This difference suggests that judges are more reliable when asked to note a more specific parameter such as strain-strangle, than when asked to simply determine a level of severity. The less specific subjective indicator, severity, seems to allow for greater variability among judges. The

greater reliability for the strain-strangle rating also suggests this parameter to be an appropriate descriptor for adductor spastic dysphonia.

The results of this study when viewed as a whole have implications for future research and clinical management of spastic dysphonia. Voicing difficulty is clearly a feature of adductor spastic dysphonia and is a feature that to some degree accounts for part of the perceptual impression of this unique disorder. Voice therapy that addresses the voicing difficulty and in some way trains easy onset and smooth transitions, as in stuttering, is clearly suggested if therapy is to be tried. This is currently the therapy generally in practice and its continued use is therefore supported.

An avenue of research to further delineate the abductor/adductor/continuum dilemma would be to further use voice onset time and devoicing measures with simultaneous videoendoscopic laryngeal studies. The issue of voicing and devoicing is critical to the classification schema.

Lastly, perceptual measures should be used with caution. The results of this study suggest that interjudge reliability measures for various tasks and samples can vary. When we consider that the current mode of treatment for spastic dysphonia may involve a recurrent laryngeal nerve section or injection of a botulism toxin for a disorder whose diagnosis is based largely on subjective criteria, then extreme care in diagnosis is called for. The use of perceptual judgments, objective measures, and clinicians highly experienced in the management of spastic dysphonia is essential.

## Appendix A

### The Rainbow Passage

When the sunlight strikes raindrops in the air, they act like a prism and form a rainbow. The rainbow is a division of white light into many beautiful colors. These take the shape of a long round arch, with its path high above, and its two ends apparently beyond the horizon. There is, according to legend, a boiling pot of gold at one end. People look, but no one ever finds it. When a man looks for something beyond his reach, his friends say he is looking for the pot of gold at the end of the rainbow.

## Appendix B

Segments Selected for Spectrographic AnalysisP

a prism  
 apparently  
 boiling pot

PeopleK

colors  
 according  
 looking

S

the sunlight  
 friends say  
 for something

T

into  
 these take  
 according to

ShshapeF

and form  
 beautiful  
 ever finds  
 his friends  
 looking for

## Appendix C

Voice Onset Time and Devoicing Interval Measurements  
for Normals (in milliseconds)

Segment	Subject		
	SF	MR	AB
p (prism)	28	60	76
p (apparently)	52	24	60
p (pot)	32	40	68
p (people)	32	48	52
p (pot)	20	28	80
t (into)	40	36	76
t (take)	32	40	56
t (to)	40	48	68
k (colors)	32	68	72
k (according)	36	52	88
k (looking)	60	92	64
s (shape)	116	148	172
f (form)	180	140	172
f (beautiful)	76	64	116
f (finds)	112	120	96
f (friends)	132	128	56
f (for)	60	92	64
s (sunlight)	128	144	156
s (something)	108	128	148
s (say)	128	172	192

## Appendix D

Voice Onset Time and Devoicing Interval Measurementsfor Spastics (in milliseconds)

Segment	Subject					
	MS	RM	HZ	EK	GG	KH
p (prism)	36	0	0	0	44	232
p (apparently)	0	28	0	0	0	160
p (pot)	0	0	0	0	0	368
p (people)	0	32	0	0	0	164
p (pot)	0	0	0	0	0	172
t (into)	52	0	48	100	0	136
t (take)	44	0	32	124	24	0
t (to)	0	0	48	0	60	140
k (colors)	44	0	0	0	0	184
k (according)	0	32	232	0	0	148
k (looking)	36	0	0	160	0	0
s (shape)	112	104	248	260	328	308
f (form)	0	92	115	160	0	224
f (beautiful)	48	0	40	320	168	0
f (finds)	0	0	248	104	0	176
f (friends)	120	108	200	112	0	208
f (for)	24	96	0	200	0	172
s (sunlight)	128	136	164	268	920	300
s (something)	100	124	220	164	0	244
s (say)	116	152	224	300	0	0

## Appendix E

Individual Judges Perceptual Ratings - Strain-Strangle

Subject	Task/ Trial*	Judge					
		NT	BO	JC	SK	RR	GC
MS	o s 1	5	4	3	5	5	5
	o s 2	4	4	2	3	5	4
	s s 1	4	4	2	4	4	3
	s s 2	4	4	2	4	5	3
HZ	o s 1	5	5	4	7	4	5
	o s 2	4	5	4	6	4	4
	s s 1	4	5	4	6	6	5
	s s 2	3	5	5	5	5	5
RM	o s 1	5	5	3	6	5	6
	o s 2	4	6	5	6	5	6
	s s 1	5	6	5	6	5	5
	s s 2	4	6	6	6	6	5
KH	o s 1	5	6	5	6	7	5
	o s 2	5	6	5	6	6	5
	s s 1	6	4	5	5	5	6
	s s 2	6	3	3	5	5	6

(Appendix E continues)

Subject	Task/ Trial*	Judge					
		NT	BD	JC	Sk	RR	GC
EK	o s 1	5	6	3	5	7	5
	o s 2	5	6	3	5	7	5
	s s 1	5	4	4	4	5	3
	s s 2	5	4	4	4	5	4
GG	o s 1	6	7	6	7	7	7
	o s 2	6	7	7	7	7	7
	s s 1	7	7	7	7	7	7
	s s 2	7	7	7	7	7	7
<u>Spontaneous Speech</u>							
MS	ov sv 1	5	5	3	4	6	4
	ov sv 2	4	5	3	4	6	5
	s s 1	4	5	3	5	7	3
	s s 2	4	4	3	5	5	3
HZ	o s 1	5	6	4	5	6	4
	o s 2	5	6	4	5	6	4
	s s 1	6	5	4	5	6	3
	s s 2	6	5	4	6	5	6
RM	o s 1	6	7	6	6	6	4
	o s 2	4	7	5	6	6	4
	s s 1	4	6	5	6	6	4
	s s 2	4	6	5	6	6	4

(Appendix E continues)

Subject	Task/ Trial*	Judge					
		NT	BD	JC	Sk	RR	GC
KH	o s 1	6	7	6	7	6	5
	o s 2	6	7	6	7	7	5
	s s 1	7	7	6	6	7	5
	s s 2	7	7	6	5	7	5
EK	o s 1	5	6	5	7	6	4
	o s 2	4	6	5	5	4	4
	s s 1	3	5	3	4	4	2
	s s 2	3	4	3	4	4	2
GG	o s 1	6	7	7	7	7	7
	o s 2	7	7	7	7	7	7
	s s 1	7	7	7	7	7	7
	s s 2	7	7	7	7	7	7

\* os = overall severity  
 ss = strain-strangle

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