SPASTIC DYSPHONIA: A CASE REPORT

LESLEY WOLK, B.A. (SP & H THERAPY) (WITWATERSRAND)
Department of Speech Pathology and Audiology, University of the Witwatersrand, Johannesburg.

SUMMARY

An in-depth investigation of a 55-year-old woman with spastic dysphonia was performed. The findings from otological, neurological and psychological investigations assisted in making a differential diagnosis and served to provide evidence for the etiology of this disorder. Subjective-perceptual evaluations of the voice revealed a strained, harsh-breathy voice quality with frequent breaks in phonation, a variable pitch and visible tension in the face and neck muscles. Objective spectrographic evaluations revealed much turbulence, ill-defined harmonics, a breakdown in formant structure, rapid pitch fluctuations and evidence of diplophonia, which was confirmed on a fiberscopic examination. Post-treatment spectrographic evaluations indicated an improved phonatory ability with significant improvement in the above parameters. Results are discussed in terms of the etiology and symptomatology of this disorder; and clinical implications for diagnosis and treatment are considered.

OPSOMMING

'n 55-jarige vrou met spastiese disfonie is in diepte ondersoek. Die bevindings van otologiese, neurologiese en sielkundige ondersoeke het bygedra tot 'n differensiële diagnose en het ook gedien om bewyse te verskaf vir die etiologie van hierdie afwyking. 'n Gestremde grof-ruiserige stemkwaliteit met frekwente onderbrekings in fonasie, veranderlike toonhoogte en sigbare stremming in gesig- en nekspiere blyk uit subjektiefperseptuele evaluasie van die stem. Objektiewe spektografiese evaluasies bring baie omstuimigheid, vaag bepaalde harmoniese tone, 'n afbraak in formant struktuur, vinnige wisseling van toonhoogte en bewyse van diplofonie aan die lig. Dit is bevestig deur 'n fiberskopiese ondersoek. 'n Verbeterde fonetoriese vermoë met opmerklike vooruitgang in die bogenoemde parameters blyk uit spektografiese evaluasies wat na behandeling opgeneem is. Resultate word bespreek in die lig van die etiologie en simptomatologie van hierdie afwyking en kliniese implikasies vir diagnose en behandeling word oorweeg.

Spastic dysphonia has long been recognized as the most mysterious and the most poorly understood of all voice disorders. It is a rare disorder and literature on the subject is limited, which has led to some confusion with regard to differential diagnosis and treatment.

Spastic dysphonia was first described by Traube in 1871.[6] Since then many terms have been used to describe the disorder. Aronson, Brown, Litin and Pearson,[1] for example, prefer the term 'spasmodic' dysphonia to avoid the confusion that arises from the use of the term 'spastic', which implies a specific neurological disorder. It is often referred to as 'stammering of the vocal cords' or 'laryngeal stuttering' because of the similarity between the glottal symptoms of spastic dysphonia and the oral ones of the stutterer. For the purpose of this paper, the term 'spastic dysphonia' will be used.

The onset of this disorder is reported to be relatively abrupt and to

occur almost exclusively in middle age, with women being affected slightly more frequently than men.[16, 25] Berendes has defined 'spastic dysphonia' as a disorder of phonation which is characterized by a *strained, creaking and choked vocal attack, a tense and squeezed voice sound . . . accompanied by extreme tension of the entire phonatory system* (cited in Fox,[12] p.275). He further noted that the disorder disturbs primarily the communicative function of speech, with other expressive functions of the voice (such as laughing, speaking to oneself or to animals) remaining intact. Spastic dysphonia represents the most extreme form of overadduction of the vocal folds. There is effortful voice production, intermittent voice stoppage, a reduced vocal range and inappropriate change in pitch, quality and/or volume.[10] The patient may complain of 'a choking sensation' or a 'tightness of throat'.[7] Several visible features as described, such as flushing of the face and frowning accompanying the vocal spasm and tic-like contractions of the neck muscles.[1, 2, 8] Severe contractions of the thoracic and abdominal musculature accompany the laryngeal spasm and is probably a compensatory forcing response for overcoming the resistance to exhalation created by the glottal closure. These frustrated vocal efforts result in a pronounced alteration of pneumophonic (phonatory-breath) control.[11, 16]

A basic controversy seems to emerge from the literature, reflecting two divergent schools of thought about the etiology of the disorder. One point of view, typified by Brodnitz,[6] suggests that spastic dysphonia is a psychogenic voice disorder, a form of conversion hysteria. Here spastic dysphonia is seen as one of two extreme forms on the continuum of functional voice disorders: functional aphonia being the one extreme of hypofunction, and spastic dysphonia representing the extreme of hyperfunction. Both disorders are interpreted as the physical manifestations of a deep-rooted emotional conflict.[4, 13] Luchsinger and Arnold[16] propose that this voice disorder may be explained as the intrusion of a subcortical primitive mechanism of phonation after the regular cortical phonatory system has become inhibited by the unconscious withdrawal of a shattered personality from the threats of daily life. In opposition to this point of view are others exemplified by Robe, Brumlik and Moore,[23] who claim that spastic dysphonia is a manifestation of disordered function of the central nervous system. This organic view was later advocated by Aronson et al[2] who feel that spastic dysphonia has a neurologic substrate and may be related to the essential tremor syndrome.

In view of the diversity of opinions regarding its etiology and intervention strategies, further research in this area seemed to be indicated. Furthermore, the literature on this subject has been derived mainly from subjective-perceptual evaluations and qualitative descriptions. Little attempt has been made to delineate the acoustic characteristics of spastic dysphonia or to describe the nature of the disorder by other objective means.

In this paper, the author's intention is thus (a) to highlight the clinical picture of a case with spastic dysphonia and repaired cleft of the soft palate, (b) to attempt to delineate some of the acoustic parameters in this disorder of spastic dysphonia, and (c) to present some practical implications for diagnosis and treatment.

CASE BACKGROUND

Vocal History: Mrs. G, aged 55 years, was first seen at the Speech and Hearing Clinic, University of the Witwatersrand, Johannesburg in June, 1979. She was referred by an ear, nose and throat (ENT) specialist who reported a dysphonia with no organic pathology evident upon laryngeal examination.

She reported that she suffered from a period of chronic laryngitis and experienced a sudden onset of voice difficulty approximately twelve months prior to the initial assessment. She stated that 'I find difficulty getting out my words, especially if I'm a bit agitated. . . . I try to force my words out and I get so cross with myself'. She complained of no pain apart from some discomfort in the laryngeal region, fatigue of the upper chest muscles and severe tension in the abdominal area as she forces the air for speaking. She reported that conversational speech, telephone calls, reading long passages and supervising others at work were difficult situations for her, and that her voice deteriorated when she was agitated, angry or upset. There was evidence of moderate depression and extreme anxiety at the time of initial assessment. No previous speech assessments or treatment were reported.

Medical History: She was born with a cleft of the soft palate which was not repaired until recently (lips and hard palate were not affected). Mrs. G. reported that although she has always had 'a nasal problem' related to the cleft, it never worried her. Recently she decided to undergo surgery as she hoped that this would correct her voice problem. The velar cleft was successfully repaired in April, 1979. According to Mrs. G, some improvement in her voice was noted in the immediate post-operative period, but a gradual deterioration has since occurred. She has undergone no other surgery, apart from a gynaecological operation in 1962.

Psychological and Family History: Mrs. G. described several significant 'family stress' factors which may be associated with the onset of her voice problem. No family history of speech, language or hearing problems was reported. She has been working for a major oil company for twelve years. At present she holds a senior position as a sub-accountant and appears to achieve job satisfaction. She has tended to avoid social contact since the onset of her voice difficulty.

She described herself as a tense person and a perfectionist both at work and at home.

CLINICAL OBSERVATIONS

Mrs. G. presents as an attractive, well-groomed, intelligent middle-aged woman. From an informal assessment, she displayed a reserved, controlled introvert-type personality. She exhibited traits of conscientiousness, keen awareness and discharge of her responsibilities. She maintains an upright, rigid posture with general body tension. Extreme tension of her facial muscles, forehead and lips was noted to occur during speech; reflected in much frowning and facial contortions (while this is typical to spastic dysphonia,[1] it may also be associated with inadequate velopharyngeal closure). These informal observations are consistent with Damste[9] who characterizes patients with spastic dysphonia as *reliable persons with strong sense of duty and a rigid personality, sensitive and easily hurt* (p. 173).

EXAMINATIONS CARRIED OUT

Examinations were carried out by an ENT specialist, a neurologist and a psychologist respectively, in order to assist in making a differential diagnosis and to further provide evidence for the etiology of this disorder. Results are summarized in Table 1.

TABLE I: Summary of findings from otological, neurological and psychological examinations.

EXAMINATION	FINDING
Otological	— no evidence of anatomical or functional disorder of larynx — normal vocal cord movement (indirect laryngoscopy) — no abnormality of tongue — slight nasal escape — difficulty in phonating
Neurological	— no major neurological deficits apart from: — increased reflexes in upper and lower limbs — slightly awkward digital movements. — Normal EEG — Normal EMI Scan
Psychological	— Evidence of marital tension — Strong sense of duty; rigid personality — Various sources of emotional conflict

The neurological findings seen in Table I, bear some relationship to the findings of Critchley[8] and Aronson et al[1] which suggest evidence of hypertonus and hyperreflexia in patients with spastic dysphonia. No conclusions can be drawn in relation to the EEG findings as inconsistent results are reported in the literature. Robe et al[23] found that 90% of their subjects showed abnormal EEG recordings; whereas

Aronson et al[1] found that 17 out of 22 patients showed normal EEG recordings, mild dysrhythmic activity in four others and independent spike foci in one patient.

The findings from otological, neurological and psychological investigations seem to provide some support for the theory proposed by Aronson and his co-workers.[1, 2] It is their belief that perhaps the type of personality described, superimposed on the 'unstable' motor system may be the adequate combination for the development of the disorder. They further suggest that these patients may have a basic predisposition or instability of innervation within the motor system, which is manifested through dysphonia when life events or personality abnormalities precipitate the symptoms.

Currently, Damste[10] has found no evidence to assume that one single organic factor can be held responsible for causing spastic dysphonia. He contends that organic factors contribute in bringing the patients to a disadvantaged position. This, together with other external, constitutional and personality factors may trigger the 'pathologic defense system' of which he feels the symptoms of spastic dysphonia are a part.

ASSESSMENT OF COMMUNICAION BEHAVIOUR

The assessment of communication behaviour was carried out by the author over a period of 3 months. This included a hearing assessment, an oral peripheral examination, a speech and language assessment. Results are summarized in Table II. Overall intelligibility thus appeared to be impaired by jerky rhythmic patterns, disturbed intonation patterns, periodic breaks in phonation and the frequent substitution or insertion of glottal stops. For the purpose of this paper, only a more detailed discussion of the acoustic impedance measurement will be included.

Acoustic Impedance Measurement

Studies have shown that middle ear muscle contraction precedes vocalisation by 65–100 ms or coincides with it.[20, 26] The findings of recent studies provide evidence for a high incidence of apparent middle ear dysfunction in patients with spastic dysphonia.[19] The use of acoustic impedance measurement has thus been advocated in order to further assess this phenomenon.

Briefly, the procedure used involved stimulating the acoustic reflex with sound stimuli at a 20 dB suprareflex threshold level for approximately 30 seconds. The Madsen Z070 electroacoustic impedance bridge was used to observe the changes in middle ear impedance secondary to reflex contraction of the tympanic muscles. The focus in data analysis was concerned with certain dynamic response characteristics of the middle ear muscles as evidenced by the acoustic impedance measurements. Particular attention was given to (1) on-time, (2) steady-state and (3) off-time characteristics of the acoustic reflex

TABLE II: Summary of findings from an assessment of communication behaviour.

Hearing (a) Pure-Tone Audiometry	— Normal —
(b) Acoustic Impedance Audiometry	— Apparent abnormalities in middle ear muscle function (see below)
Oral Peripheral Examination (OPE)	— Structural and functional adequacy of lips, teeth and jaw, tongue and hard palate. — Slight asymmetry of soft palate and uvula. — Soft palate is short — Oropharynx is slightly shallow with excess width — Nare constriction — Adequate velopharyngeal closure since cleft repair — Slight nasal escape (on plosives).
Speech	— Minimal articulation errors related to history of congenital velar cleft: — substitution and insertion of glottal stops. — distortion of fricatives (e.g. /s,z/). — substitution of voiceless sounds for their voiced cognates.
Language	— Normal

response. A qualified audiologist acted as a second observer to provide a measure of reliability.

The findings revealed normal tympanograms peaking at zero pressure level (mm w/s) bilaterally, and acoustic reflexes within normal limits. Three types of apparent abnormalities in the middle ear muscle function were observed:

1. An apparent abnormality in the steady-state portion of the acoustic reflex was observed at 4 000 Hz in both ears i.e. reflex contraction of the middle ear muscles was not sustained for the duration of acoustic stimulation at 4 000 Hz. Instead, the muscles apparently contracted briefly at the onset of stimulation and again at the cessation of stimulation. However, in view of the fact that this phenomenon only occurred at 4 000 Hz, which has been found to occur in some normal subjects,[14] no conclusions can be drawn.
2. An abnormality in the off-time characteristics of the acoustic impedance response was noted to occur at 1 000 Hz in the right (R) ear and at 500 Hz in the left (L) ear. This refers to a prolonged time involved in the return to the baseline, which McCall[19] feels might be suggestive of a problem in muscle relaxation.

3. There was evidence of possible tremor or shivering of the middle ear muscles at the following frequencies: 250 Hz, 1 000 Hz and 2 000 Hz in the (R) ear, and 250 Hz, 500 Hz and 1 000 Hz in the (L) ear.

It is important to mention at this point, that these results may be affected by the presence of the velar cleft, as a high incidence of middle ear pathologies in cleft palate patients is well documented in the literature.[3] Nevertheless, the results discussed above are consistent with those of McCall[19] who found similar results in his patients with spastic dysphonia. The observation of possible difficulty in muscle relaxation and tremor of the middle ear muscles might add further support to suggest neurogenic involvement of the extrapyramidal motor system in this patient.[19] Damste[9] has further suggested that the combined activity of vocal cords, velum and middle ear muscles in normal speech, might serve to explain that all three may be affected simultaneously when there is a supernuclear excitation inhibition disorder.

ASSESSMENT OF VOICE DISORDER:

Subjective-Perceptual Evaluations

An analysis of the tape recorded speech sample and general observations from the initial assessment revealed the following:

1. A disturbance of respiratory control and a lack of pneumophonic co-ordination. This is reflected by a predominant use of thoracic and clavicular breathing, shallow inhalations, jerky movements and a difficulty in sustaining phonation.
2. A variable pitch in conversational speech and a difficulty in regulating the pitch level.
3. A reduced intensity level.
4. A harsh voice quality with periodic breaks in phonation, accompanied by visible tension of the muscles in the face and neck. A glottal stroke was heard at the onset of phonation. There was evidence of apparent breathiness throughout all phonation.
5. The reflex actions of phonation were intact. Thus, coughing, laughing and crying were carried out normally. Singing, talking to herself and her pet cat were reported by her to facilitate improved phonation. This was confirmed by the clinician who listened to tape recordings of her speaking in these situations at home.

Objective Evaluation — Spectrographic Assessment

Spectrographic analyses were carried out as a diagnostic tool in an attempt to gain further insight into the nature of this disorder.

A Kay Sonagraph Model 6061-B (Kay Elemetrics 6, Pine Brook, N.J.) was used to produce type B/65 spectrograms from the tape recordings of conversational speech. The data in this study were derived from broad-band and narrow-band spectrograms. Practically, it was not possible to analyze spectrographically all utterances recorded during

diagnostic evaluations. Thus only a few utterances from the spontaneous speech sample taken at the initial assessment, were randomly chosen. The analysis of spectrographic data was descriptive in nature. For the purpose of this paper, only a few of these utterances were selected in order to highlight the major findings.

The spectrographic representation for the utterance 'and when I asked him' is illustrated in *Figure 1.* On the broad-band spectrogram *(Fig. 1a)* there is evidence of much breathiness and ill-defined harmonics. Sections were taken during the steady-state of the vowel, which showed that the filter function is operating in such a way as to dampen the harmonics. Spectral energy is entirely missing where Formant 1 (F1) was expected. While F2 is present, there is no evidence of F1 and F3. This finding may be explained by a reduced driving force from the lungs, resulting in a reduced subglottal pressure at the vocal cords;[15] or possibly inadequate adjustment of the vocal folds which would also affect the range of harmonics. It can be noted in *Figure 1a* that there are bursts of energy which probably occur after the initial glottal attack or 'laryngeal spasm'. The narrow-band spectrogram *(Fig. 1b)* confirms the breathiness evident on the broad-band spectrogram, reflecting a lack of vocal cord approximation. This is illustrated by a sporadic and inconsistent occurrence of harmonics.

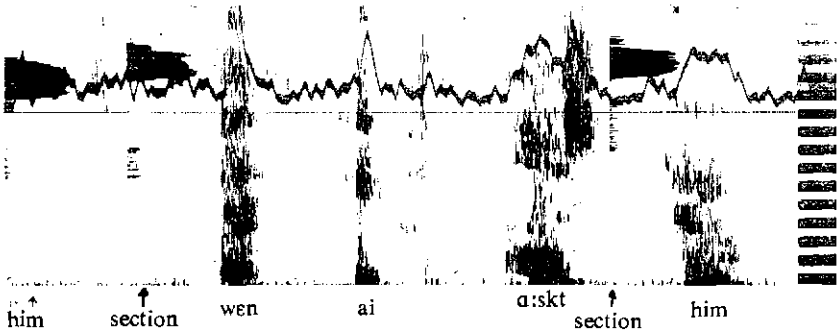

Figure 1a. Broad-band spectrogram showing utterance /and wen ai ɑ:skt him/.
Note: Some of the noise evident is from the tape-recording.

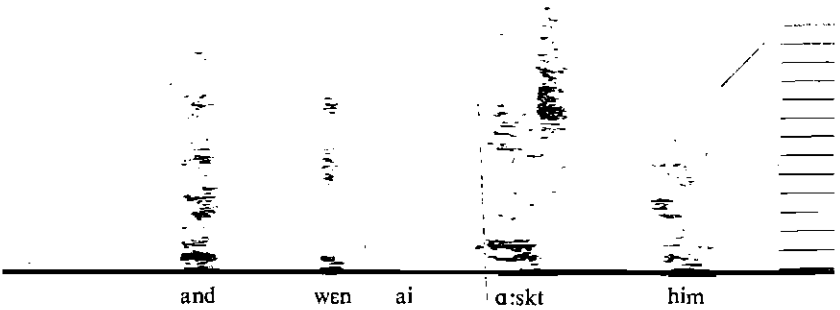

Figure 1b. Narrow-band spectrogram showing utterance /and wen ai ɑ:skt him/.

The scale magnified narrow-band spectrographic representation of the utterance 'I asked my husband' is illustrated in *Figure 2.* Marked fluctuations in pitch can be seen on the vowel /a:/ in 'asked', with a relatively high fundamental frequency (±200 Hz). There is a rise in pitch from 250 Hz to 350 Hz (100 Hz in approximately 24 ms) indicating that pitch change is very rapid. It seems then, that rapid pitch fluctuations in this patient give rise, at least in part, to the perceptual effect of harshness.[28]

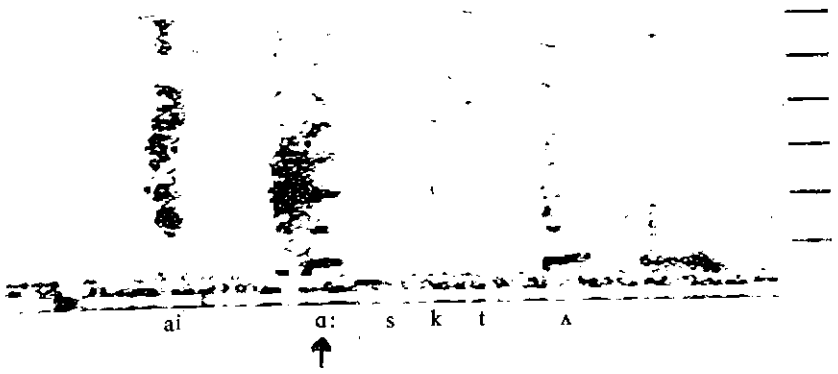

Figure 2. Scale-magnified narrow-band spectrogram showing utterance /ai ɑ:skt mai hʌzbənd/.

Figure 3 represents the utterance 'how did the voice sound?' An examination of the scale magnified narrow-band spectrogram *(Fig. 3)* alerted the author to another diagnostic feature which was hardly available during periodic subjective-perceptual evaluations. The harmonic structures represent the characteristic picture of diplophonia, which is clearly illustrated on the vowel /aw/ in 'sound'. Here it can be seen that the harmonic at 1250 Hz bears no relationship to the harmonic starting at 1000 Hz and moving down to 800 Hz. Similar pitch fluctuations to those discussed in *Figure 2* are evident. It is interesting to note that there are simultaneous rising, falling and level pitches, indicating that the vocal cords are vibrating in at least two distinct modes at the same time.[27] At the level pitch, the rate of vibration is maintained, whereas at the falling pitch there is a slowing down of the vocal fold vibration. It can be concluded therefore that in this case of diplophonia, there are (a) different modes of vibration and (b) different rates of vibration for each mode. Boone[5] has stated that diplophonia is frequently a symptom of general hyperfunctional usage of the vocal mechanism (p. 173). This diagnostic evaluation would thus seem to correlate significantly with the general diagnostic picture of spastic dysphonia and the frequent use of glottal stops resulting from the cleft palate.

Figure 3. Scale-magnified narrow-band spectrogram showing utterance /haw dɪd ðə vɔis sawnd/.

After obtaining valuable diagnostic information from the spectrographic data discussed above, it was decided to extend the objective evaluation as a measure of therapeutic success. Spectrographic analyses were carried out two months after the onset of treatment and at the end of treatment. The patient was treated for a total period of four months. Utterances were again chosen randomly from tape-recorded spontaneous speech samples. For the purpose of this discussion, spectrographic analyses carried out during the course of treatment will not be discussed in detail. They reflected a steady progression towards improved phonatory ability.

Figure 4 represents the utterance 'there were three factors', which was taken from a sample at the end of treatment. Here it can be seen that there is some evidence of breathiness, although this is considerably reduced when compared with the utterance 'and when I asked him' in *Figure 1*. The harmonics are clearly defined and there is no evidence of diplophonia. Similar results were obtained from several other utterances analyzed spectrographically after treatment. The fact that the characteristic of 'diplophonia' was not evident on subjective-perceptual evaluations, leads one to assume that it may in fact be a symptom commonly associated with spastic dysphonia, but one which might have previously been overlooked because of the reliance upon subjective evaluations.

Figure 4. Post-therapy scale-magnified narrow-band spectrogram showing utterance /ðɛə w3: ərɪ: fæktəz/.

Fiberscopic Examination:

Modern instrumentation such as the flexible fiberoptic nasolaryngoscope (fiberscope) provides a means for making direct observations of laryngeal behaviour during voice production.[24]

The entire procedure was carried out in the Department of Phonetics and General Linguistics, University of the Witwatersrand, Johannesburg.[27] The fiberscope was connected by a C-mount adapter to a camera. A diagnostic fiberoptic light source provided necessary illumination. The fiberscope was inserted into one nostril until the tip of the scope reached a level slightly rostral to the superior margin of the epiglottis, which allowed for observation of the patient's vocal folds and laryngeal structures. A tape recorder Uher 4200 Report Stereo IC with an M816 directional microphone, was prepared to run concurrently with this examination so as to record all utterances which could then be carefully examined with its corresponding slide. The patient was asked to (1) breathe at rest, (2) sustain voicing on the vowel /i:/, (3) converse with the investigator and (4) read a passage from a book.

A review of the slides revealed the following findings:

1. The vocal cords were normal at rest, as illustrated in *Figure 5*.
2. At the onset of phonation for the vowel /i:/ there seemed to be a tight sphincteric closure, which subsequently disappeared and continued as normal voicing.
3. During connected speech, there was evidence of an inconsistent picture, variable in the attitude of one vocal fold to the other, i.e. the two vocal folds did not lie equal to each other; one fold appeared larger and thicker as if it was pulled slightly over the other vocal fold. This is clearly illustrated in *Figure 6*.

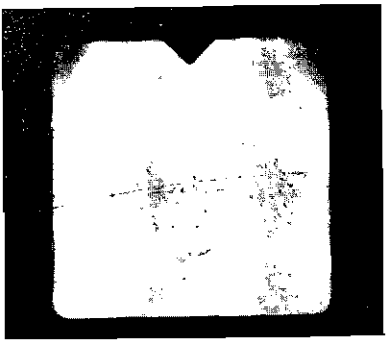

Figure 5. Subject's laryngeal behaviour during quiet breathing, showing that the vocal cords are normal at rest.

Figure 6. Subject's laryngeal behaviour during connected speech (voicing).

(Note that the two vocal cords are not lying symmetrically to each other. The (R) fold is larger and thicker, as if it is pulled slightly over (L) fold. This picture of asymmetry was seen repeatedly in connected speech.)

There was a general picture of 'shuddering' or 'laryngeal spasms' as expected for this voice disorder. Throughout all conversational speech and reading tasks, a slight 'chink' in the vocal cords was evident, which would account for the continual breathy escape. Overall results thus served to confirm the diagnosis of 'diplophonia' noted on the spectrographic assessment; and to validate the information obtained from subjective-perceptual evaluations.

In summary, subjective-perceptual evaluations of the voice revealed a strained, harsh-breathy voice quality with frequent breaks in phonation, a variable pitch and visible tension in the face and neck muscles. Objective spectrographic evaluations revealed much turbulence, ill-defined harmonics, a breakdown in formant structure ('filtering-out' of formants), rapid pitch fluctuations and evidence of diplophonia which was confirmed on the fiberscopic examination. Post-treatment spectrographic evaluations indicated an improved phonatory ability. There was a minimum turbulence, clearly defined harmonics, increased energy, increased sub-glottal pressure and an absence of diplophonia; giving rise to a less breathy, less harsh, less choked voice quality. This was combined with improved pneumophonic co-ordination, reduced tension of the face and neck muscles, an elimination of glottal attacks and a better adjusted social personality.

CONCLUSIONS AND IMPLICATIONS

It seems premature to make a general assumption that a patient with spastic dysphonia is suffering either from a psychogenic disorder or a neurologic one. As Aronson et al[1] proposed, the issue must be resolved on the basis of findings that can be established for each particular patient.

The author has thus postulated that in this single case, the onset of spastic dysphonia may be attributed to long term vocal misuse in compensation for the velar cleft,[18, 21] possibly precipitated by the physical trauma of 'secondary laryngitis'[16] and the cleft repair operation, combined with the psychological stresses of life which occur during middle age. Furthermore both the neurological and psychological findings in this study might point to an underlying organic or psychogenic predisposition. This explanation is supported by Cooper[7] who recently concluded that: the onset and development of incipient spastic dysphonia and spastic dysphonia are due to long term vocal misuse, with psychological or physical trauma often being the catalyst (p. 173). An extension of this may lead one to postulate that in this single case, both the congenital velar cleft and the predisposition for the occurrence of spastic dysphonia may be related to a single common constitutional or embryological factor.

Clinical Implications

The spectrographic analyses and fiberscopic examination carried out in this study served to provide an accurate diagnosis of the voice disorder

and an evaluation of voice improvement both during and after treatment.

Most authors agree that the overall prognosis in patients with spastic dysphonia is poor.[5,7] Currently however, it is still felt that the combination of voice therapy with psychotherapy offers the best chances for improvement.[4,6] Several voice therapy techniques have been advocated, some of which were found to be useful in the treatment of this patient. These include procedures such as physical relaxation, respiration training and the correction of pneumophonic co-ordination;[7,10,13] the yawn-sigh technique, humming and vowel exercises, Froeschel's chewing method, the elimination of hard glottal attacks and the use of a hierarchy analysis to assist the patient in recreating those environments where relaxed phonation is best achieved.[5,25] EMG biofeedback has recently been proposed as an effective adjunct to traditional therapy methods for hyperfunctional voice disorders.[22] Its applicability and effectiveness in patients with spastic dysphonia, however, is still uncertain, so that further investigation into this area seems to be indicated.

The findings of this study point to an important implication regarding habilitative procedures employed in the treatment of cleft palate children. There seems to be a tendency in some cases to overlook the vocal dimension in the treatment of these patients, focusing mainly on the improvement of velopharyngeal closure and increased flexibility of the articulators. Luse, Heisse and Foley[17] showed that all their cleft palate cases had a spasm of the hypopharynx prior to rehabilitation, indicating excessive hypopharyngeal and laryngeal tension. They thus contend that cleft palate quality can be eliminated by reducing tension in the laryngeal and pharyngeal areas, so that the manner of phonation or vocalisation would seem to be the starting point for rehabilitation.

While this study was concerned primarily with an evaluation of the vocal parameters, it would be of interest and probably essential to further investigate speech breathing in patients with spastic dysphonia. Further research employing spectrographic analyses and fiberscopic examinations, working optimally within a team approach, would be of value in the evaluation and rehabilitation of a variety of voice disorders.

ACKNOWLEDGEMENTS

The author wishes to thank Mrs. M. Wahlhaus, Senior Lecturer and Ms. C. Penn, Lecturer, in the Department of Speech Pathology and Audiology of the University of the Witwatersrand for their invaluable guidance and support, and Mr. A. Traill, Senior Lecturer of the Department of Phonetics and General Linguistics for his valuable assistance in the spectrographic and fiberscopic testing of this patient. The author also acknowledges the Human Sciences Research Council for its financial assistance.

REFERENCES

1. Aronson, A. E., Brown, J. R., Litin, E. M. and Pearson, J. S. (1968): Spastic Dysphonia I. Voice, neurologic and psychiatric aspects. *J. Speech Hear. Dis.* 33, 203-218.
2. Aronson, A. E., Brown, J. R., Litin, E. M. and Pearson, J. S. (1968): Spastic dysphonia II. Comparison with essential (voice) tremor and other neurologic and psychogenic dysphonias. *J. Speech Hear Dis.* 33, 219–231.
3. Arora, M. M. L., Sharma, V. L., Gudi, S. P. and Balakrishnan, C. (1979): Acoustic impedance measurements and their importance in cleft palate patients. The *J. Laryngol and Otol.* 93, 443–445.
4. Bloch, P. (1965): Neuro-psychiatric aspects of spastic dysphonia. *Folio Phoniat.* 17, 301–364.
5. Boone, D. R. (1971): *The voice and voice therapy.* Prentice-Hall, Inc. Englewood Cliffs, New Jersey.
6. Brodnitz, F. S. (1976): Spastic dysphonia. *Ann. Otol.* 85, 210–214.
7. Cooper, M. (1973): *Modern techniques of vocal rehabilitation.* Charles, C. Thomas Publishers, Springfield, Illinois.
8. Critchley, M. (1939): Spastic dysphonia; ('Inspiratory Speech'). *Brain.* 62, 96–103.
9. Damste, P. H. (1977): Spastic dysphonia; untieing or cutting the knot. *O.R.L.* 39(3), 173.
10. Damste, P. H. (1978): *Spastic dysphonia: An interpretation of the symptoms.* Communicative Disorders in Audio Journal for Continuing Education. 3(5) May. Bradford, L. J. (Ed) Grune and Stratton, Inc., New York.
11. Dedo, H. H. (1976): Recurrent laryngeal nerve section for spastic dysphonia. *Ann. Otol.* 85, 451–459.
12. Fox, D. R. (1969): Spastic dysphonia: A case presentation. *J. Speech Hear. Dis.* 34, 275–279.
13. Greene, M. C. L. (1964): *The voice and its disorders.* Pitman Medical Publishers, Co., Ltd., 2nd Edition.
14. Jerger, J. (Ed). (1973): *Modern developments in audiology.* 2nd Edition Academic Press Inc. A subsidiary of Harcourt, Brace Jovanovich. Publishers.
15. Ladefoged, P. (1967): *Three areas of experimental phonetics.* Chapter 1. Oxford University Press. Ely House, London.
16. Luchsinger, R., and Arnold, G. E. (1965): *Voice-speech-language.* Belmont, California. Wordsworth.
17. Luse, E., Heisse, J. and Foley, J. (1964): The vocal approach in the correction of cleft palate speech. *Folio Phoniat.* 16, 123–129.
18. Marks, C. R., Barker, K. and Tardy, M. E. (1971): Prevalence of perceived acoustic deviations related to laryngeal function among subjects with palatal abnormalities. *Cleft Palate J.* 8(2) 201–211.

19. McCall, G. N. (1973): Acoustic impedance measurement in the study of patients with spasmodic dysphonia. *J. Speech Hear. Dis.* 38(2), 250–255.
20. McCall, G. N. and Rabuzzi, D. D. (1973): Reflex contraction of middle ear muscles secondary to stimulation of laryngeal nerves. *J. Speech Hear Res.* 16, 56–61.
21. McDonald, E. T. and Koepp-Baker, H. (1951): Cleft palate speech. An interpretation of research and clinical observations. *J. Speech Hear Dis.* 16, 9–20.
22. Prosek, R. A., Montgomery, A. A., Walden, B. E. and Schwartz, D. M. (1978): EMG Biofeedback in the treatment of hyperfunctional voice disorders. *J. Speech. Hear. Dis.* 43(3), 282–294.
23. Robe, E., Brumlik, J. and Moore, P. (1960): A study of spastic dysphonia: Neurologic and electroencephalographic abnormalities. *Laryngoscope.* 70(3), 219–245.
24. Saito, S., Fukuda, H., Kitahara, S. and Kokawa, N. (1978): Stroboscopic observation of vocal fold vibration with fiberoptics. *Folio Phoniat.* 30(4) 241–244.
25. Segre, R. (1951): Spasmodic aphonia. *Folio Phoniat.* 3, 150–157.
26. Shearer, W. M., and Simmons, F. B. (1965): Middle ear activity during speech in normal speakers and stutterers. *J. Speech Hear. Res.* 8, 203–207.
27. Traill, A. (1979): Senior Lecturer in the Department of Phonetics and General Linguistics, University of the Witwatersrand, Johannesburg. *Personal Communication.*
28. Wendahl, R. W. (1963): Laryngeal analog synthesis of harsh voice quality. *Folio Phoniat.* 15, 241–250.