

INTERVENTION PLANNING FOR ADULTS WITH COMMUNICATION PROBLEMS

ADULTS WITH VOICE DISORDERS

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The Contribution of the Voice to Form, Content and Use

Language can be thought of as a conventional linguistic code which -- in an individual with normal vocal tract structure and function -- is communicated largely in the form of speech. Speech, then, is the medium through which language is most commonly expressed. An important sound source for speech production is the larynx. Within the larynx are two small shelves of tissue known as the vocal folds. When a speaker sets the vocal folds into vibration, sound is produced. This process of setting the vocal folds into vibration to produce sound is called phonation. The acoustic product of vocal fold oscillation is a complex quasiperiodic signal. Let's review briefly the characteristics of complex periodic vibration. A complex quasiperiodic signal consists of multiple frequencies of vibration. That is, it consists of a set of sinusoidal

waveforms, each having a different period or frequency of vibration. In addition, the pattern of the complex quasiperiodic waveform almost repeats itself exactly. Figure 1a shows the waveform of a complex quasiperiodic signal. Note that the pattern of vibration appears to repeat itself, but not exactly. Slight differences in cycle-to-cycle fundamental period and amplitude exist. Hence, it is considered to be a quasi-periodic signal, rather than a periodic signal. Figure 1b reveals the three lowest frequency sinusoidal waveforms of a larger set of sinusoidal waveforms, that, when added together, constitute the complex quasiperiodic waveform shown in Figure 1a. Each of these sinusoidal waveforms has a different frequency, amplitude and phase relationship with the others. Illustrated in figure 1c is an amplitude spectrum. It shows the frequency and amplitude of the first three sinusoidal waveforms, or harmonics, of the complex quasiperiodic signal. The amplitude spectrum shown in Figure 1c is known as a glottal spectrum because it reveals the amplitude and frequency characteristics of sound produced at the level of the glottis.

Vocal fold vibration, then, is an important speech sound source. It provides the acoustic energy for all American

English vowels, diphthongs, liquids, glides, and nasal sounds. In addition, vocal fold vibration is combined with noise generated within the oral cavity to produce the voiced fricative and affricate sounds. Thus, the voice makes an important contribution at the segmental level of language form. In other words, the human voice is responsible for producing many of the individual sound segments of speech.

In addition to creating many speech sounds, the voice is largely responsible for the suprasegmental features of speech, which, in turn, interact with language content. For example, variations in the amplitude and the fundamental frequency (f_0) of vocal fold vibration (that accompany changes in intonation and stress) are accomplished at the level of the larynx (Atkinson, 1978). Consider the phrase "Ben and Jerry love ice cream" which a speaker produces with a rising intonation pattern, and again with a rising-falling intonation pattern. These two productions are identical at the segmental level of speech. That is, they have the same phonemic content. Yet, by varying the pattern of f_0 change, the speaker can alter meaning. The phrase produced with a rising intonation pattern (increasing f_0 level) signals a closed-ended question. The speaker's use of a rising-falling

intonation pattern (rising f_0 level followed by a falling f_0 level) reveals a declarative statement. Similarly, the content of an identical phrase can be altered by systematically varying word stress. Consider three productions of the following simple phrase, in which the underlined word receives primary stress.

1. Ben loves Jerry.
2. Ben loves Jerry.
3. Ben loves Jerry.

In the first example, it is Ben (not Edy or Steve) who loves Jerry. In the second example, Ben doesn't merely like Jerry, he loves him. Finally, in the third example, Ben loves Jerry (not someone else). Atkinson (1978) discusses the different mechanisms of laryngeal control used by speakers, to alter the suprasegmental features of stress and intonation.

The voice, then, is an acoustic signal that carries the meaning of the linguistic code. The voice should not interfere with or detract from this primary function. If the voice interferes with the speaker's intended message, it will compromise the speaker's ability to communicate effectively. A

voice disorder secondary to vocal tract dysfunction, can interfere with both language form and content. For example, a patient with significant velopharyngeal dysfunction will present with what is often classified as a resonance-based voice disorder. Such a speaker has difficulty achieving adequate velopharyngeal closure at the appropriate moment within the flow of speech. In order to appreciate the importance of the velopharyngeal valve to normal speech production, consider a simple consonant-vowel-consonant (CVC) syllable construct, e.g., "mom." In order to produce an acoustically acceptable "mom", the speaker must maintain an open velopharyngeal valve during the initial /m/ production. The velopharyngeal mechanism must then achieve significant closure for the vowel / / and then must open again for the final /m/ sound. If the speaker cannot effect adequate opening and closing of this articulatory valve at the appropriate moment in time, an imbalance of oropharyngeal-nasal resonance will occur. The voice will sound hypernasal and, if velopharyngeal dysfunction is severe enough, audible nasal airflow may be perceived during speech production. In addition, as a consequence of a grossly incompetent velopharyngeal valve, a speaker will have difficulty producing

consonant sounds that require high levels of intraoral pressure (specifically, the stop, fricative and affricate sounds). The unusual vocal quality and imprecise articulation that will result are likely to interfere significantly with speech intelligibility. A voice disorder then, may constitute a substantial barrier to effective communication and can interfere with language form.

The majority of laryngeal-based voice disorders are characterized by the presence of noise, which results from inadequate closure of the vocal folds during phonation. In such cases, the line spectrum that should characterize the complex quasiperiodic signal will be replaced, at least in part, by a continuous spectrum (i.e., noise). Figure 2a illustrates a power spectrum for a vowel produced with normal voice quality. Note that, as expected, the section analysis yielded a line spectrum. Note also, the relative absence of a noise component in the power spectrum of a good quality vowel. Figure 2b shows a power spectrum for a vowel produced with a hoarse vocal quality. Note the presence of noise which is characterized by interharmonic and high frequency energy above 3000 Hz. In effect, a noise component (continuous spectrum) is added to the complex

quasiperiodic signal (line spectrum).

An additional consequence of the presence of a mass lesion on the vocal folds is the alteration of vocal fold stiffness and mass (Hirano, 1981; Hirano and Bless, 1993). Such changes in the physical properties of the vocal folds will alter the patient's habitual f_0 of vocal fold vibration.

A severe voice disorder can also interfere with the social functions of language. An extreme voice disorder, such as advanced spasmodic dysphonia, usually has severe vocal symptoms associated with it. The ability of an individual with spasmodic dysphonia to function in the world can be compromised considerably. For example, the significant disruption in speech flow associated with untreated spasmodic dysphonia can be so severe that the individual may ultimately cease attempts to communicate verbally. The social and professional consequences of a voice disorder with such severe symptoms can result in dramatic changes in lifestyle, such as the need to change professions or the decision to withdraw from or avoid social contacts.

WHAT CONSTITUTES A VOICE DISORDER?

Traditionally, a voice disorder is identified and defined by the perceived alteration in vocal quality that accompanies it. Laryngeal-based voice disorders are characterized by abnormal glottal resistance during phonation. Those voice disorders that are marked by reduced glottal resistance (e.g. - vocal fold paralysis, vocal nodules, polyps) result in inadequate vocal fold approximation during phonation which can add a significant amount of noise to the signal. Consequently, the voice is likely to be perceived as hoarse, harsh, breathy or rough, with varying degrees of severity. Vocal quality and the severity of the dysphonia will depend upon the size and location of the lesion (or the position of a paralyzed vocal fold) as well as on the clinician's definition of the terms used to designate quality and severity. Other voice disorders are characterized by abnormally high glottal resistance (e.g. - adductor spasmodic dysphonia). The voice of an individual with advanced spasmodic dysphonia is often described in the clinical literature as having a strained, strangled quality.

Laryngectomy has severe communication consequences, since

the individual has lost one of the primary sound sources for speech. Normal phonation is no longer possible and an alternative sound source must be found. An extrinsic source of vibration (an artificial larynx) or use of an intrinsic vibrating source (use of tissue at the pharyngo-esophageal junction in e esophageal speech or following tracheo-esophageal shunt) can serve as the patient's new "voice."

Velopharyngeal dysfunction can alter the normal balance of oral-nasal resonance during speech and phonation, which will result in abnormal vocal quality. Poorly timed and inadequate velopharyngeal closure will result in perceived hypernasality. If velopharyngeal resistance is excessive, a hyponasal quality would be expected. Consequently, resonance-based disorders secondary to velopharyngeal dysfunction are often classified as voice disorders due to the alteration in the quality of speech and voice that results.

Classifying Voice Disorders

Aronson (1990) advocates classifying voice disorders into

two broad categories: organic and psychogenic. According to Aronson, a voice disorder caused by vocal fold tissue changes or neurologic or metabolic disease would be considered an organic voice disorder. Organic voice disorders can result from a variety of congenital conditions (e.g., laryngeal web, laryngomalacia, cysts, stenosis) or viral conditions (e.g. - papilloma). In addition, acquired organic voice disorders can be secondary to tumor formation, blunt or inhalation trauma, neurologic disease or endocrine dysfunction.

A psychogenic voice disorder, according to Aronson, is one in which the voice is abnormal in the absence of any significant structural or physiologic abnormalities. Examples include conversion voice disorders, puberphonia, and those voice disorders secondary to vocal abuse/misuse or due to excessive musculoskeletal tension. Psychogenic voice disorders may have as their cause a psychoneurosis (e.g. -conversion voice disorders, mutational falsetto); or vocal abuse/misuse (e.g. -vocal nodules, polyps, contact ulcers). According to Butcher, Elias, and Raven (1993):

Factors often associated with psychogenic voice disorder[s] are high levels of anxiety, acute or chronic life stresses, interpersonal relationship or marital difficulties, personal overcommitment or assuming the onus of family

responsibilities, difficulties in setting appropriate personal and interpersonal limits, inhibition concerning the expression of thoughts or feelings and associated high levels of helplessness or powerlessness. (pp. 106-107)

Although a psychogenic voice disorder does not have, as its cause, a structural or physiologic abnormality, organic tissue changes can, and often do, occur. For example, vocal nodules constitute a significant structural change in the vocal folds. However, nodule development is the consequence of faulty habits of voice use. In other words, the cause of the dysphonia is vocal abuse or misuse which results in laryngeal tissue changes.

There is some disagreement among voice specialists regarding the classification of abuse related dysphonias as psychogenic voice disorders. Morrison and Rammage (1994) argue that the diagnosis of a psychogenic voice disorder "

... should be reserved for those muscle misuse voice disorders that clearly have a primary psycho-emotional etiology, as defined by current standards for psychiatric evaluation. (p.53)

SOURCES OF INFORMATION

In order to develop a comprehensive and well reasoned intervention plan, the clinician needs to have available to her critical sources of information. These include:

- o Knowledge of Normal Vocal Tract Structure and Function
- o Knowledge of Maintaining Factors
- o Baseline Data
- o Performance Demands
- o Principles of Learning and Rehabilitation

Knowledge of Normal Vocal Tract Structure and Function

A working knowledge of vocal tract structure and function is essential to the development of a rational treatment protocol for patients with voice disorders. A common therapeutic goal for patients who present with dysphonia is the development of "improved breath support." However, before considering the development of long-term, short-term and session goals to improve breath support, the voice clinician must determine

whether or not it is appropriate to direct intervention efforts to the ventilatory mechanism. Consider the following clinical case:

M.L., a twenty six year old male (6' 0" tall) presented with a dysphonia characterized by severe hoarseness. The clinician, equipped only with a stopwatch and a hand-held spirometer, measured M.L.'s vital capacity (VC) and maximum phonation time (MPT). His measured MPT (the duration of maximum sustained phonation) was 9.4 seconds. Using a spirometer, she determined M.L.'s VC to be 5.4 liters. M.L.'s expected vital capacity based on his age, gender and body size was 5.34 liters. Given these simple clinical measures, what clinical decision can be made regarding the appropriateness of directing therapy efforts to increasing breath support?

For this patient, the obtained MPT of 9.4 seconds, was excessively low. According to Yanagihara, Koike and von Leden (1966) An average expected MPT for an adult male is approximately 22.5 s (S.D. = 6.1 s). A reduced MPT can be explained by either a reduced vital capacity or (in the case where the patient's vital capacity is adequate) by a laryngeal valving problem. This voice clinician, having a fundamental understanding of the vocal tract dynamics underlying phonation, recognized that she could not interpret the meaningfulness of a 9.4 second MPT without additional diagnostic data. She understood that measurement of the patient's VC was necessary in

order to determine if the reduced MPT was due to inefficient laryngeal valving during phonation, direct ventilatory system involvement, or both. In this example, M.L.'s reduced MPT was most likely the result of an incompetent laryngeal valve, since his vital capacity was within normal limits for his age, gender and body size.

The clinician lacking a basic understanding of the dynamics underlying voice production, might have designed an intervention plan to increase breath support. In this case, rehabilitation efforts directed at an incompetent laryngeal valve would have been more appropriate.

Clearly, an appreciation of ventilatory and phonatory function can assist the clinician in the decision making process when developing appropriate therapeutic goals.

Too often, unnecessary work on breath support is undertaken with dysphonic patients because the clinician does not understand normal aspects of voice production. Consider the following two clinical cases in which the clinician developed a long term therapy goal "to improve breath support."

Case Example #1

T.B., a 35 year old female, presented with a hoarse voice. Monitoring of chest wall movements during speech production revealed that she typically initiated speech of normal conversational loudness at a point between 60% and 65% of her vital capacity. The patient's expiratory limbs typically ended at or slightly below resting expiratory level (REL), that is, around 40% of her vital capacity.

Case Example #2

L.T., a 29 year old female, presented with a hoarse voice. An examination of her breathing revealed that she typically produced speech of normal conversational loudness at lung volume levels between 30% and 45% of her vital capacity.

The voice clinician who has a basic understanding of normal ventilatory function will be in a position to determine whether or not a goal to improve breath support was indicated for either patient. The informed voice clinician would recognize that a goal directing intervention efforts on increasing "breath support" would be appropriate for L.T., but would very likely be contraindicated for patient T.B. Speech of normal conversational loudness is usually produced within the midvolume range (between approximately 35 - 60% of the vital capacity (Hixon, Goldman and Mead, 1973). Within this lung volume range a speaker can take advantage of the small positive relaxation pressures that exist, thus achieving the subglottal pressures

required for speech with minimal muscular effort. Spirometric recordings of T.B.'s speech breathing, revealed that she used an appropriate lung volume range (re: REL) during speech production. Patient L.T., on the other hand, appears to have adopted a speech breathing pattern that is highly inefficient. Monitoring of chest wall movements (variable inductance plethysmography) revealed that patient L.T. initiated speech at lung volume levels that were inappropriately low, and that she terminated speech at a lung volume level well below REL. Intervention efforts directed at modifying this patient's breathing pattern for speech appear to be worth exploring.

Again, the voice clinician who has a basic appreciation of the physiological factors underlying speech and voice production, will be in the enviable position of developing appropriate therapeutic goals. In addition, acoustic and physiologic measures of speech can serve as a means of documenting a patient's progress in therapy, which is an important objective means with which to evaluate therapy efficacy.

The voice clinician who has an adequate background in the basic communication processes should be able to develop

appropriate, well reasoned, therapeutic goals. Objective measures of voice can assist the clinician in making rational clinical decisions. Consider the following case in which a voice clinician obtained objective measures of the voice and attempted to use those data to guide intervention efforts.

N.G., a 22 year old female, presented with a large nonfibrotic unilateral sessile polyp. The clinician measured the mean speaking frequency (MSF) and mean f_0 during sustained phonation using a commercially available fundamental frequency analyzer. The clinician noted that MSF and mean f_0 during sustained phonation were unusually low. N.G. presented with a MSF of 168 Hz while reading the "Rainbow Passage", and a mean f_0 during sustained phonation of 171 Hz. Because the patient's f_0 level was too low, the clinician developed the following long term goal: the patient will increase vocal f_0 to a more appropriate level given the patient's age and sex.

Based on the limited information provided above, does it appear that the voice therapist made an appropriate clinical decision?

According to Stoicheff (1981), an expected MSF for a woman N.G.'s age is approximately 224 Hz (normal MSF ranges between 192 Hz - 275 Hz). Therefore, N.G.'s f_0 during reading was lower than expected. If you consider that the presence of a nonfibrotic polyp on the vocal fold will result in a decrease in the stiffness and an increase in the mass of the vocal fold cover, a lowered f_0 is not unexpected. For this patient, it is

likely that the habitual use of a reduced f_0 is not the cause of the voice disorder, but rather, a lowered f_0 was the consequence of the changes in the physical properties of the vocal fold, due to the presence of the polyp. Obviously, it would be desirable to have some information regarding the patient's habitual speaking f_0 prior to the onset of the voice disorder. Since such information is rarely available, patient and family member reports of any changes in f_0 since the onset of the voice disorder often can be helpful in speculating about the premorbid speaking frequency levels. Consequently, a goal directed at increasing vocal fundamental frequency would be contraindicated for this patient. In fact, attempts to increase the patient's MSF may exacerbate the patient's current condition, resulting in additional vocal strain. Rather, therapy directed at eliminating abusive vocal behaviors, if successful, should result in a reduction in the size of the polyp. As the size of the polyp is reduced, the physical properties of the vocal fold will change, and the f_0 of vocal fold vibration should increase to a more appropriate level. Again, knowledge of normal laryngeal structure and function can assist the voice therapist in the clinical decision making process.

Maintaining Factors

Ideally, voice therapy would be initiated only after the etiology of the voice disorder has been determined. A desirable, yet sometimes unattainable, goal of the diagnostic voice evaluation is the determination of the specific etiology of the dysphonia. Unfortunately, some voice evaluations include little more than a subjective description of vocal quality. A statement such as "... the patient presented with severe hoarseness..." provides the clinician with little useful information regarding the etiology, nature or severity of the voice disorder. Further, such perceptual descriptions do not provide useful information to the voice clinician who must answer critical questions related to clinical management, such as: What is the basis for this patient's dysphonia?; What intervention strategies might be indicated or contraindicated?; Is voice therapy indicated?, and if so, what should be the initial focus of therapy?; What is the prognosis for improvement?; What are reasonable expectations for voice

production following intervention?. Perceptual descriptions of the voice alone are extremely imprecise and are of little diagnostic value to the voice clinician.

Knowledge of the etiology of a voice disorder can assist the clinician in the development of an appropriate therapy plan. For example, treatment for a psychogenic voice disorder should be initiated only after the possibility of an organic etiology has been systematically ruled out. Of course, the identification of an organic etiology of a voice disorder does not rule out the possibility, however remote, of a concomitant psychogenic component.

Even when it has been determined that the etiology of a voice disorder is psychogenic (e.g., a voice disorder secondary to vocal abuse or misuse), structural changes in vocal fold tissues can, and usually, do occur. If a voice disorder secondary to vocal abuse or misuse is diagnosed, the clinician must identify those factors that are likely to have contributed to the development of the voice disorder; as well as any behaviors that serve to maintain vocal dysfunction. These factors may be exogenous (e.g., if the patient works in an environment in which there are great vocal demands in the

presence of excessive and intense ambient noise). Endogenous factors such as idiosyncrasies of the patient's personality, can serve as the basis of dysphonia as well.

Successful intervention planning for adults with voice disorders requires that the clinician identify those variables that can serve to maintain the voice disorder. Among the maintaining factors that need to be considered by the clinician working with the dysphonic patient are sensorimotor and psychosocial factors. In some cases, a patient's dysphonia may be maintained by a significant cognitive impairment.

Sensorimotor Factors

Voice disorders can result from isolated instances of abuse (for example, screaming at a football game) or from chronic vocal misuse or abuse. Dysphonia can also be a symptom of neurological disease. In the case of an isolated peripheral lesion of the recurrent laryngeal nerve, for example, dysphonia may be the only significant clinical symptom. With more extensive lesions of the nervous system, dysphonia may

constitute merely one of many symptoms observed throughout the speech mechanism. In addition, voice disorders can result from benign and malignant tumors, tissue changes due to trauma, the inhalation of caustic materials, viral conditions, metabolic disease and endocrine dysfunction.

Vocal Abuse or Misuse

Colton and Casper (1996) provide a comprehensive review of the effects of vocal abuse and misuse on the laryngeal mechanism. The broad category of vocal misuse can include behaviors that result in excessive laryngeal strain or tension (e.g. - excessive use of hard attack, chronic maintenance of a high vertical position of the larynx in the neck); habitual use of an inappropriate pitch level; excessive voice use and singing without proper vocal technique. The definition of vocal abuse, as adopted by Colton and Casper (1996) includes behaviors such as habituated use of an excessively loud voice; excessive throat clearing and coughing; and screaming or cheering. Continued misuse and abuse of the voice will often lead to the development of structural and physiological changes in the larynx and subsequently, the development of dysphonia. Some examples of

tissue changes that are the consequence of abuse or misuse of the voice include vocal nodules, polyps, polypoid degeneration (edema) and contact ulcers. Morrison and Rammage (1994) provide the reader with suggestions for maintaining good vocal hygiene. They identify specific vocal behaviors and environmental factors to avoid and those to adopt in order to promote efficient voice production.

Neurogenic Voice Disorders

Upper motor neuron lesions, resulting from CVA, traumatic brain injury or diseases such as multiple sclerosis will have wide ranging consequences. The symptoms associated with such global neuropathology are not confined to the phonatory mechanism, but are observed throughout the entire speech mechanism. Some examples of neurogenic disorders that present with significant vocal symptoms include pseudobulbar palsy, Parkinson's disease, Huntington's disease, myoclonus, cerebellar ataxia, myasthenia gravis, essential tremor, multiple sclerosis, laryngeal dystonia (spasmodic dysphonia), amyotrophic lateral sclerosis, and peripheral lesions of the vagus (X Cranial) nerve.

Organic Voice Disorders/ Laryngeal Trauma

Many voice disorders are the result of organic disease or trauma (including exposure to noxious environmental stimuli). Some examples are benign neoplasms; hyperkeratosis; leukoplakia; granuloma and hyperacidic contact ulcer; papilloma; laryngeal carcinoma; cricoarytenoid ankylosis; and a variety of voice disorders secondary to endocrine dysfunction (e.g. - hypothyroidism, hyperthyroidism, Addison's disease).

Hearing Impairment

The voice can be adversely affected by the presence of a hearing impairment. The effects of a hearing impairment on the voice will depend on the degree and time of onset of the hearing loss. For example, the consequences on voice -- as well as speech and language -- would be greatest for an individual with a profound, congenital hearing loss. Individuals who present with an organic condition, such as hearing impairment, may be unable to monitor their voice effectively due to impaired auditory feedback. As a result, the hearing impaired individual will have difficulty controlling pitch, loudness, flexibility

and vocal quality, particularly if the hearing loss is congenital or its onset is prelingual. Resonance problems have been identified with the hearing impaired population, most likely due to the maintenance of a posterior tongue position (Subtelny, Li, Whitehead and Subtelny, 1989). Differences in f_0 level and f_0 variability have been reported between hearing impaired speakers and individuals with normal hearing (Horii, 1982). Significant problems in ventilatory behavior among hearing impaired speakers have been reported as well (Forner and Hixon, 1977).

Evaluating the Integrity of the Peripheral Nervous System

A comprehensive assessment of the peripheral speech mechanism is critical to the planning and implementation of an efficacious intervention plan. This is particularly true for patients with neurogenic voice disorders. For example, patients who present with dysphonia secondary to peripheral lesions of the vagus nerve, require a thorough assessment of cranial nerve function. This evaluation can assist the voice clinician in planning appropriate intervention strategies. Consider the following case example.

Patient R.O. presented with a hoarse voice and hypernasal speech. An examination of the oropharynx revealed asymmetrical movement of the velum during phonation. During vowel production, extensive movement of the velum upward and back was observed only on the right side. The ENT report stated that the left vocal fold was paralyzed between the intermediate and gentle abduction positions.

Based on this information, it is reasonable to conclude that the lesion of the X cranial nerve was high, above the level of the pharyngeal nerve. What are the implications of this information for the planning of therapy? Since R.O.'s vocal fold paralysis was idiopathic, and there was some possibility of spontaneous recovery, a trial period of voice therapy was undertaken. However, due to the fact that the vocal fold was paralyzed such a significant distance from midline (since both the RLN and SLN were affected on the left side), the prognosis for improvement with voice therapy was guarded. In addition, resonance problems could be expected for patient R.O., since motor function to the velum was affected due to involvement of the pharyngeal branch of the vagus nerve on the left side. Since R.O. presented with two incompetent articulatory valves (velopharyngeal and laryngeal valves), it would not be unreasonable to anticipate difficulties in R.O.'s ability to manage subglottal pressure. His ability to generate and

maintain adequate subglottal pressure would be impaired as a consequence of excessive airflow and the premature expenditure of lung volume due to reduced velopharyngeal and laryngeal resistance during speech production.

A patient with an isolated lesion of the recurrent laryngeal nerve (RLN), resulting in a paralysis of the vocal fold in the paramedian position, (closer to midline than for patient R.O.), would have a more favorable prognosis for improved vocal function with voice therapy.

Psychosocial Factors

The voice has long been considered a barometer of the human psyche. The larynx is extremely sensitive to an individual's physical, psychological and emotional health. Vocal function is affected by a person's attitudes, emotions, and personality. Anxiety, fear, anger, depression, personality disorders, psychosexual conflict, low self esteem, disturbed interpersonal relationships, and unresolved conflicts may contribute to the development and maintenance of a voice disorder. The tension and stress associated with everyday life can contribute to vocal

dysfunction, particularly in those individuals who respond to anxiety by maintaining increased muscle tension in the larynx and perilaryngeal area, pharynx, jaw, tongue, and throughout the ventilatory system. The laryngeal structures appear to be particularly susceptible to emotional stress. Individuals who habitually maintain excessive musculoskeletal tension in response to the psychological stress and tension associated with modern life are at risk for the development of a variety of inflammatory vocal fold tissue changes. Such a response to stress may manifest as nodules, polyps, polypoid degeneration, contact ulcers, or chronic laryngitis. Common sources of anxiety include family problems and disturbed interpersonal relationships. The case histories of patients who present with psychogenic voice disorders may reveal an unfulfilled or disturbed relationship with a parent, child, spouse or significant person in their lives. In addition, a patient's conversion dysphonia may be related to his or her difficulty expressing feelings of anger, resentment, frustration or hopelessness.

Many patients who present with psychogenic voice disorders, such as conversion dysphonia, do not require intensive

psychological counseling or psychotherapy. Aronson (1990) stresses the need for clinicians who treat patients with psychogenic voice disorders to understand psychological and psychiatric aspects of voice disorders. He argues, quite properly, that voice clinicians should develop improved skills in the conduct of psychosocial interviewing and psychological therapy. A thorough discussion of the psychological aspects of psychogenic voice disorders is provided by Butcher, Elias and Raven (1993). The voice therapist should be sensitive, however, to the need to refer for psychological counseling those patients who are unable to deal with the stresses of life on their own or who present with emotional and psychological issues that are beyond the scope of practice for the speech-language pathologist.

The nature of an individual's occupation, job responsibilities, work and home environments, family constellation and lifestyle are important factors in the development and maintenance of a voice disorder. Certain occupations place greater vocal demands on an individual. For example, teachers, clergy, salespersons, professional users of the voice (singers and actors), stock brokers, telemarketers all

engage in daily activities which require extensive use of the voice. In addition to a person's occupation, the nature of the work environment is another important factor that can contribute to a voice disorder. Does the individual need to use his voice under conditions of significant background noise? Does the job require long hours and extended periods of voice use? Do airborne pollutants exist in the workplace? Does the workplace have adequate humidification? In addition, psychosocial issues such as job satisfaction, the quality of relationships with peers, superiors and subordinates, and the magnitude of job responsibilities are all potential sources of stress and anxiety. For some individuals, their occupation and work environment interact to place them at great risk for the development of voice problems. An extreme example of such an interaction is patient R.L., an untrained aspiring singer in an alternative band. He performed nightly in a smoke-filled room without adequate amplification, and smoked cigarettes and consumed alcohol between sets while mingling with members of the audience.

The dynamics of a person's home environment is also an important consideration for patient's with dysphonia. It is not

uncommon for adults with several young children or pets to signal them or gain their attention by yelling. In such active home environments, it is not unusual for family members to abuse their voices regularly as they attempt to communicate under conditions of excessive ambient noise. Some examples include: a parent who attempts to communicate with a teenage child who is playing music at high intensity, or a parent who attempts to mediate a dispute between fighting siblings. If a member of the family has a hearing impairment, it is not uncommon for other family members to speak habitually at excessively high vocal intensities. Extraordinary vocal demands, such as speaking in a noisy environment, reduces the availability of auditory feedback which can lead to poor monitoring of vocal intensity and unchecked vocal abuse.

Cognitive Factors

In cases of isolated laryngeal pathology, with no central nervous system involvement, concerns regarding a significant cognitive disability generally do not exist. However, when treating a neurologically-based voice disorder with central nervous system involvement, dysphonia may be only one -- and

often a minor -- component of a more global neuropathology. Such patients present with multiple disabilities. For the patient who presents with disorders in cognition, language, resonance, articulation, ventilatory and laryngeal function, vocal rehabilitation may constitute only one aspect of a larger intervention program. For example, a patient with severe, untreated hypothyroidism may present with myxedema with concomitant lingual edema resulting in dysphonia and articulatory imprecision. If the central nervous system is affected, impairments in language and cognitive function and changes in personality may exist as well. Patients with multiple disabilities who have a severe intellectual disability, may present with laryngeal or velopharyngeal dysfunction which results in a voice disorder. Given the significant deficits in language and cognition, intervention for dysphonia or a resonance imbalance is often among the lowest priorities in the treatment program.

Whenever a patient presents with limited cognitive abilities, the clinician should expect the patient to experience difficulty understanding instructions, monitoring the various parameters of the voice and implementing behavioral changes

required for improved vocal function.

Baseline Data

Current Vocal Function

In order to develop and implement a rational intervention plan for adults who present with voice disorders, it is essential that the clinician have available current and comprehensive diagnostic data. Even for those patients who have received a recent voice evaluation, a brief period of diagnostic therapy should not be ruled out. Ongoing assessment is desirable for the following reasons: (1) to confirm initial diagnostic impressions, and (2) to evaluate relevant issues related to vocal function that either had not been addressed, or were not resolved at the time of the initial voice evaluation. Comprehensive diagnostic data are essential to the development of appropriate rehabilitation techniques, and to the formulation of reasonable prognoses. Consider the following clinical example:

M.H., a 56 year old female, was seen by an otolaryngologist due to concerns about her voice. The patient complained

that her voice was hoarse and that it became "tired" with continued use. Indirect laryngoscopy revealed small, newly formed, bilateral vocal nodules. M.H. was referred to a local university Speech and Hearing Clinic for voice therapy. During the initial therapy session with M.H., the student clinician initiated a vocal hygiene program. At one point during the session she responded to the patient's expressed concern that her voice might not improve with the comment "Don't worry, we'll fix you right up."

In this example, the student clinician was implementing an intervention strategy based entirely on the otolaryngologist's impressions from a visualization of the larynx. In addition, she attempted, quite properly, to convey to the patient reasonable expectations regarding her voice following vocal rehabilitation efforts. Unfortunately, in this case, the prognosis offered to the patient was based on the fallacious assumption that the presenting problem was simply the presence of vocal nodules. As a result, the student clinician offered a prognosis which proved to be both premature and inaccurate. Additional perceptual vocal symptoms existed which were overlooked by the clinician, perhaps because she failed to look beyond the otolaryngologist's diagnosis of vocal nodules. These symptoms, which became more severe over several weeks, included intermittent phonation breaks and a strained, strangled vocal quality. Subsequently, airflow and electroglottographic data

obtained during diagnostic therapy confirmed instances of intermittent glottal closure during phonation, which resulted in the cessation of phonation for brief periods of time. Ultimately, adductor spasmodic dysphonia was diagnosed. This example illustrates the dangers inherent in planning intervention on the basis of a single diagnostic measure. The clinician can make appropriate treatment decisions only after a comprehensive assessment of the patient's vocal function. Such an evaluation includes -- in addition to a direct examination of the larynx -- the acquisition and interpretation of ongoing perceptual, acoustic and physiologic data. Appropriate and efficacious treatment techniques cannot be developed and implemented, until a comprehensive evaluation of the voice has been conducted.

The importance of maintaining ongoing assessment throughout voice therapy cannot be overemphasized. The acquisition of noninvasive acoustic and physiologic measures of vocal function can be incorporated into the therapeutic process. Such objective measures are sensitive to changes in the status of the larynx and can provide the clinician with indirect objective evidence of the effects of therapeutic intervention. Since it

is neither feasible nor desirable to refer patients to the otolaryngologist for frequent visualizations of the larynx, ongoing objective assessment of laryngeal function should be carried out by the voice clinician. Ongoing assessment by the voice therapist is an activity that can be reasonably implemented in therapy. The result will be the availability of information which is critical to the evaluation of therapy efficacy.

Patient Characteristics

The characteristics and capabilities of the dysphonic patient can influence the course and outcome of therapy. Two major determining factors in the success of intervention with abuse related voice disorders are: (1) the ability of the patient to identify and monitor abusive behaviors and (2) the ability to adopt and habituate new, nonabusive behaviors. Objective voice analysis (i.e., acoustic and physiologic assessment) is a useful means of evaluating the efficacy of voice therapy or medico-surgical intervention.

Patients who possess good auditory discrimination skills and mental representation abilities, for example, can often

quickly achieve the behavioral changes necessary to discard poor vocal habits and adopt new behaviors that promote improved vocal function. The degree to which the adult dysphonic possesses these abilities may have important implications for intervention planning. The use of imagery can be a very effective technique for modifying vocal behaviors. Although the descriptions provided to the patient using this method may have little or no relationship to underlying physiological processes, imagery has a long history of use in vocal pedagogy and has proven highly successful in the training of professional users of the voice. Voice therapists should not overlook this technique, which can often result in desired changes in vocal function, where physiologically accurate descriptions of voice production or the use of more traditional intervention techniques have failed. For example, traditional symptom modification therapy with Parkinson's patients have not been terribly successful. Ramig (1997) describes the effect of providing the simple cue "think loud" to a patients with Parkinson's disease.

Typically, the patient "automatically" takes a deeper breath, improves vocal fold adduction, opens her mouth more, and uses larger articulator movements. (p.34)

Since most patients with voice disorders need to learn new

motor patterns, their ability to make use of auditory, visual and tactile and kinesthetic cues are important factors in the outcome of therapy. The patient's ability to identify and discriminate among different voice qualities, intensities, frequencies and types of vocal attack and their ability to use such sensory information to effect changes in motor function will determine, in part, the success of therapy. The patient's intra-and-interpersonal discrimination abilities are critical to their adoption and use of new vocal behaviors which are consistent with good vocal hygiene and improved vocal function. Ramig (1997) reports that patients with Parkinson's disease are typically unaware of the extraordinary low vocal intensity of their voices. When voice of normal conversational loudness is elicited in therapy, it is not uncommon for a patient to respond "I feel like I am shouting." Ramig evaluated the effects of an intensive therapy program for patients with Parkinson's disease, the Lee Silverman Voice Treatment (LSVT). An important focus of the LSVT program is on the sensory self-perception problems characteristic of this population.

The patient's level of motivation to change the voice has important implications for the success or failure of therapy.

As a general rule, those patients for whom the onset of the voice disorder is sudden and the symptoms are severe, will be very concerned about the voice. Patients who are concerned about their voices are usually highly motivated to undertake the often difficult task of modifying habituated behaviors that are inconsistent with good vocal hygiene. On the other hand, patients who enter therapy with a long standing, chronic dysphonia, are generally less concerned about their voices. Consequently, these patients may be less motivated to eliminate habituated patterns of voice use and adopt new behaviors that promote improved vocal function. In some instances, patients with psychogenic voice disorders receive secondary gain from their voice problems. The clinician may find these patients to be both unmotivated and uncooperative.

The clinician should also be aware of the difficulty some patient's may experience in carryover. A patient may be successful in modifying vocal behaviors in the therapeutic milieu and can produce a new, more efficient voice. However, since a person's voice is so intimately related to their image of self, it is not uncommon for some patients with chronic dysphonia to reject even an improved voice and resist adopting

it. The rejection of a new, more acceptable voice is sometimes reported for patients with a nonorganic mutational falsetto whose vocal fundamental frequency has failed to adjust downward during puberty. Although a more appropriate fundamental frequency may be facilitated quickly in therapy, it is not uncommon for the patient to reject the more appropriate voice because "it doesn't sound like me."

Performance Demands

There are a number of contextual variables within the therapy session that the clinician will systematically manipulate in order to increase or decrease performance demands as intervention progresses. The selection of which variables to manipulate, that is, which performance demands to alter, is usually determined by the skills and abilities of the patient, the parameters of the voice targeted for change and the nature of the dysphonia. Some examples of the need to alter complexity

performance demands based on the patient characteristics follow. If a patient has difficulty charting several abusive behaviors simultaneously, the clinician may decide to make the initial performance demands less complex, by requiring that the patient initially record only one or two behaviors. The charting of the frequency of occurrence of additional abusive behaviors can be delayed until the patient's monitoring skills have improved. Similarly, the clinician will routinely need to alter the complexity of the linguistic context at various points throughout therapy. Whenever a patient learns a new motor task, for example, learning to produce voice with an easy, soft attack, the clinician may develop an early session goal that requires use of the soft attack only during the production of sustained vowels. As the patient's ability to produce a soft attack improves, new goals requiring the patient to produce this type of attack in more complex linguistic contexts, such as single words, short phrases and ultimately, discourse, may be developed.

Alternatively, there may come a point in the rehabilitation program at which the clinician will manipulate contextual variables to increase performance demands. If, for example, a

therapy objective is to produce speech with increased vocal intensity, the clinician may -- during a later stage in therapy -- require the patient to maintain vocal intensity in the presence of increasing amounts of background noise. For a patient who produces speech at an inappropriately low lung volume level, the clinician would work on altering the patient's ventilatory behavior first during speech of normal conversational loudness. Speech production at greater intensity levels, a more difficult task, requiring the patient to achieve and maintain higher lung volume levels, would be postponed until a later point in the therapy program.

Principles of Learning and Rehabilitation

There are a number of general approaches to vocal rehabilitation that have widespread applicability, irrespective of etiology. These include auditory training, vocal hygiene, counseling and the direct modification of faulty or inefficient vocal behaviors.

Auditory Training

Auditory training is a critical component of therapy with the dysphonic patient. Patients who can identify and discriminate among a variety of vocal qualities -- normal voices and those representing varying degrees of dysphonia -- are more likely to effect changes in voice production and successfully carryover new vocal behaviors outside of the therapeutic setting. Initially, patients need to heighten their awareness of those features of the voice in need of remediation, whether they be quality, flexibility, pitch or loudness. In addition, the patient needs to have a model of a target voice. As behavioral therapy approaches are introduced, the clinician needs to point out those instances in which the patient produces voice that approximates the target that has been agreed upon by both the patient and clinician. Eventually, the patients serve as their own clinicians, having learned to monitor and evaluate various aspects of their voice production. Fawcus (1986) highlighted the importance of auditory training as a priority in therapy:

The patient must become a critical and discriminating listener, and this is probably the single most important therapeutic goal. Unfortunately, concentration on breathing in the early stages of treatment -- which is normally part of a more traditional approach -- inevitably distracts the patient from the more important task of listening to his own voice. (p. 162)

Vocal Hygiene

Improved vocal hygiene should be a goal in therapy for all patients presenting with dysphonia. For those patients in which laryngeal tissue change has occurred as a result of vocal abuse or misuse, a vocal hygiene program should be a central component of therapy. A thorough examination of environmental and psychosocial factors that are likely to have contributed to the voice disorder should guide the development of the vocal hygiene program. Together, the clinician and patient should identify abusive vocal behaviors and develop strategies for their elimination. At the same time, alternative behaviors that promote good laryngeal health should be identified and substituted for those abusive behaviors that have been discarded. It is not enough to simply eliminate bad vocal habits. Given the limitations of the patient's vocal mechanism, the clinician and patient need to search for the most efficient

voice the patient can produce. As a general rule, the goal is to produce voice with minimal effort and strain. In addition, environmental factors that contribute to vocal abuse -- such as speaking under conditions of excessive environmental noise -- need to be identified and modified. Additional examples of environmental control which promote improved vocal hygiene include: increasing humidity in the environment, increasing fluid intake and removing airborne pollutants and irritants from the environment, identifying and eliminating dehydrating drugs and chemicals. Colton and Casper (1996) describe some essential components of a vocal hygiene program which include: reducing the amount of talking, reducing loudness (which may involve not speaking under conditions of high ambient noise), adopting nonvocal and nonverbal means of signalling others, and learning and adopting a variety of vocal techniques that reduce vocal effort and strain.

Counseling

The voice clinician's role as counselor is an important one. All intervention programs should include an explanation of

the problem and a discussion of the course, expectations and objectives of therapy. For most adults with voice disorders, counseling will serve as an important component of intervention. In other cases, when working with patients who present with spasmodic dysphonia, for example, counseling will constitute a major role of the voice therapist. Since the prognosis for improvement with behavioral voice therapy techniques is poor with spasmodic dysphonia, a critical role of the voice clinician is to explore and evaluate with the patient the various treatment options. The advantages and disadvantages of surgical intervention (recurrent laryngeal nerve section, recurrent laryngeal nerve crush) or medical treatment (such as botulinum toxin injection) need to be carefully considered. In the case of advanced laryngeal carcinoma requiring laryngectomy, it is desirable to consult with the patient pre-and-postoperatively. Preoperatively, the consequences of a laryngectomy on speech and voice production is often not a major concern of these patients who are, quite appropriately, more concerned with the issue of their survival. Following surgery, however, the patient may be more responsive to the services of the speech-language pathologist as the realities of alaryngeal communication emerge.

Voice therapy with the adult dysphonic represents a cooperative venture. The prognosis and objectives of therapy should be discussed and agreed upon by the patient and clinician. If, following a phase of treatment, satisfactory improvement has not been achieved, the client and clinician need to evaluate the reasons for the lack of progress. Possible explanations for the failure to achieve success could be the administration of inappropriate behavioral techniques, lack of adequate motivation or cooperation on the part of the patient, inability of the patient to carry out the behaviors specified in the management plan, a mismatch in the objectives of the clinician and patient, or the concomitant existence of an unresolved medical or psychological condition. In any case, the clinician needs to either perform additional tests, design and implement a new treatment protocol or refer to other health professionals for counseling or medical management, as appropriate.

Direct Modification of Speech and Phonatory Behaviors

For a variety of voice disorders, it may be necessary to change specific speech and vocal behaviors. This may require

direct intervention to modify faulty habits of voice use or learned behaviors that either are inefficient or maintain vocal dysfunction. For example, if vocal pathology results from chronic and excessive use of a hard vocal attack, the patient first must learn to reliably discriminate among various types of vocal attack. Then, the patient needs to learn to consistently identify their use of hard attack. Once the patient can intrapersonally scan for their use of hard attack, they need to modify their vocal attack, substituting a more efficient, less traumatic form of vocal initiation such as the soft attack. Another example is the rare instance in which a patient has learned and habituated an aberrant speech breathing pattern. Assuming an intact and competent set of ventilatory structures, any atypical chest wall movement patterns or lung volume expenditures will require modification. Imagery and visual biofeedback paradigms can be very effective therapeutic methods for eliminating undesirable learned speech behaviors and substituting for them, more appropriate ones. In the case of mutational falsetto, the patient is taught specific techniques to modify habituated speech, phonatory and ventilatory patterns that are presumed to maintain the disorder. According to

Aronson (1990),

Therapy for mutational falsetto is based on learning to inhale with increased air volume and to exhale with increased force simultaneous with a sharp glottal attack. (p. 345)

Biofeedback-Based Therapy

Historically, voice clinicians have made liberal use of auditory feedback in therapy by evaluating the voices of the patient and others using audio tape recorders and by providing the patient with immediate auditory feedback. Commercial instruments have long been available to permit patient monitoring of vocal intensity level outside of the therapy setting. A variety of visual light displays have also been used in therapy to help patients monitor vocal intensity. With the increased availability of more sophisticated, commercially available instrumentation systems, clinicians have increased opportunities to incorporate biofeedback paradigms into vocal rehabilitation programs. For example, many fundamental frequency (f_0) analyzers, such as the Kay Elemetrics Visipitch, provide real-time visual records of f_0 and relative intensity waveforms. Instruments such as these are noninvasive, menu-

driven systems making them extremely "user friendly" and well suited for clinical use. Patients can be provided with immediate visual feedback of f_0 and intensity which can assist the patient in modifying vocal parameters such as pitch and loudness as well as suprasegmental features of speech. If a voice therapist has access to sophisticated instrumentation arrays that transduce relevant physiological events, these visual signals can be used in a biofeedback-based therapy program. Some physiological signals that might be of interest to the voice clinician for use in therapy include: airflow, air pressure, medial contact area of the vocal folds, and chest wall kinematic data. The clinician can provide the patient with immediate visual feedback of these signals. Visual biofeedback, although not routinely embraced by speech-language pathologists, can serve as a valuable clinical adjunct. Netsell and Daniel (1979) provide an excellent example of the successful use a visual biofeedback paradigm with a dysarthric patient who successfully modified speech behaviors when provided with visual signals of physiologic adjustments. A caveat should be introduced to the clinician considering the use of visual biofeedback in therapy. She must be certain that the signal

used in a biofeedback paradigm is reliable, particularly if the patient is required to duplicate or match visual data. Further, the use of biofeedback as a therapeutic tool demands that the clinician has a thorough understanding of the dynamics underlying speech and voice production.

Operant Conditioning

Operant conditioning has a long history among speech-language pathologists as a method of treatment for many speech and language disorders. A therapy program that incorporates operant conditioning techniques typically shapes the behavioral responses of a patient by providing or withholding reinforcement. It is presumed that behaviors will increase in frequency when reinforcement is provided. Conversely, behaviors that are not reinforced should decrease in frequency. Operant conditioning techniques are applied with successful results for patients with nonorganic voice disorders. Butcher, Elias and Raven (1993) describe how cognitive-behavioral treatments -- of which operant conditioning is one example -- can be applied to patients presenting with psychogenic voice disorders. The rationale underlying cognitive-behavioral treatment is: by improving the patient's self confidence and feelings of control,

his level of anxiety or depression will decrease. As stress, anxiety or depression decrease, it is presumed that the patient's vocal symptoms will subside as well.

Intervention with Conversion Voice Disorders

Boone and McFarlane (1996) describe conversion aphonia as a somatoform disorder: one

... in which symptoms suggesting physical etiology occur for which no identifiable organicity can be demonstrated, no physiological basis is inferred, and symptoms are linked through positive evidence or strong presumption to psychological disturbance or conflict. (pp. 227-229)

When a conversion voice disorder is suspected, it is important to get ENT clearance, to wit, confirmation of the absence of organic disease. Therapy should begin, as always, with a brief period of patient education. The patient needs to understand the etiology of the voice disorder. The negative ENT findings should be revealed and their significance explained. Specifically, the patient needs to understand that there is no physiological or anatomical explanation for their inability to

adduct the vocal folds. The clinician must also explain to the patient that the larynx is a common site for the expression of emotional problems and stress. Patients presenting with a conversion voice disorder need to be convinced that they are capable of producing normal voice. Therefore, instances of vegetative phonation (e.g. - cough, sigh, yawn, laughter) need to be pointed out immediately. These vegetative vocalizations can then be shaped into phonation and speech. In the search for the voice the patient has lost, symptomatic voice therapy, using a variety of facilitating techniques may result in the patient's rediscovery of volitional phonation. Aronson (1990) points out:

Conversion aphonia and dysphonia are treated according to the principle of re-establishment of the patient's conscious awareness of greater phonatory capability and discussing the emotional conflicts that have generated the voice disorder ... (p. 345)

Butcher, et al. (1993) describe the application of cognitive-behavior treatment to psychogenic voice disorders.

According to these authors, the clinician chooses:

an appropriate combination of strategies from the following possibilities: anxiety or stress-management training ...; target setting; record keeping; guided evaluation or analysis of dysfunctional or negative thinking; guidance in challenging unhealthy thinking and finding healthy alternatives, sometimes employing cue cards which summarise positive thoughts; giving information about the nature of stressful life events; giving advice; marital therapy

focused on improving communication and rewards in relationships; role playing and assertiveness training ...; and behaviour modification techniques such as prescribing a half-hour worry period and programmes employing operant conditioning principles. (p.107)

Intervention with Neurogenic Voice Disorders

The dysphonia secondary to neurological disease often represents only one aspect of more global speech system dysfunction. The voice problems associated with central nervous system lesions, for example, may present more severe symptoms, including dysarthria, ataxia, apraxia, dysphagia, and aphasia. Typically, lower motor neuron lesions, depending on their location, have a more specific influence on the voice. Patients with voice disorders associated with central nervous system diseases are generally resistant to voice therapy. Lesions of the peripheral nervous system, are, as a general rule, more responsive to vocal rehabilitation techniques. A peripheral lesion of the vagus nerve, for example, will have more localized effects. The primary symptom of a unilateral lesion of the recurrent laryngeal nerve will be dysphonia. A vagal lesion above the level of the pharyngeal nerve, however, will result in resonance problems in addition to dysphonia.

For patients with neurological disorders that affect vocal function, Ramig and Scherer (1992) advocate a treatment approach that focuses on the underlying laryngeal pathophysiology. The diagnosis of specific neurologic disorder is less important to management than the nature of laryngeal dysfunction. Common problems in laryngeal function accompanying neurologic disorders identified by Ramig and Scherer (1992) include:

... problems in adducting the vocal folds (hypoadduction, hyperadduction), producing a stable voice (phonatory instability), and coordinating movements (phonatory incoordination). (p.163)

If a voice disorder is associated with a degenerative neurological disease, or if normal laryngeal function is not a reasonable expectation, the goal of voice therapy may be to maintain the current level of vocal function. In such cases, voice improvement may not be a therapeutic objective. Rather, maintaining current vocal function or delaying further deterioration of the voice may be realistic therapeutic expectations. In many cases of dysphonia associated with degenerative neurological disease, augmentative communication devices need to be considered when functional speech and phonation are no longer reasonable objectives. Realistic

expectations of voice production need to be conveyed to the patient early in the therapeutic process.

Decisions Regarding Surgical Intervention

Often, management of a voice disorder requires a combination of medical, surgical and behavioral intervention. In cases of laryngeal trauma in which the cartilaginous framework of the larynx is crushed, compromising the patency of the airway or if the vocal folds are lacerated, surgical intervention may be required immediately. It is possible that voice therapy may be helpful, after surgery and healing have taken place, in order to reestablish the best possible voice given the vocal capabilities possible with the reconstructed larynx.

In the case of recurrent laryngeal nerve paralysis of idiopathic etiology, voice therapy may be the initial choice for intervention. If voice therapy proves unsuccessful or if spontaneous recovery of the recurrent laryngeal nerve is not observed after six to twelve months, phonosurgery in the form of medialization of or injection of an inert substance into the affected vocal fold may be considered.

For those cases of vocal misuse or abuse in which large lesions were surgically removed, voice therapy is an essential post-surgical procedure. Obviously, simply removing large vocal nodules or polyps will not resolve the underlying cause of the tissue pathology: vocal abuse/misuse. A period of voice therapy focusing on vocal reeducation, if successful, will reduce the probability that the lesion will recur, thus eliminating the need for additional surgery.

Symptomatic Voice Therapy

As Boone and McFarlane (1994) point out:

the abusive vocal behaviors of adults are likely to be more difficult to isolate than those of children. It is the relatively rare adult voice patient whose vocal abuses are bound only to particular situations. (p. 150)

These authors advocate the use of various "facilitating approaches" as therapy "probes" in the search for the patient's best voice. Such a voice, they assert:

... allows an individual to produce voice with less effort and strain ... (pp. 151-152).

Table 1 summarizes twenty five facilitating approaches, that, when appropriately applied to patient's with dysphonia,

can be used effectively to modify vocal parameters such as pitch, loudness and quality.

INSERT TABLE I HERE

The focus of symptomatic voice therapy is on vocal symptoms, which, when eliminated, should result in more efficient voice production. Morrison and Rammage (1994, p. 81) stress the importance of the "... auditory, visual and tactile-kinesthetic processing systems" to the success of symptomatic voice therapy. It is important that the clinician who incorporates symptomatic voice therapy as part of a vocal rehabilitation program not arbitrarily select a particular facilitating technique. Rather, the choice of a facilitating technique should be based on the clinician's understanding of the nature of the disorder and selected only after an appropriate rationale for its use has been developed.

Boone and McFarlane (1994) point out:

The selection of a particular approach [facilitating technique] should not be an arbitrary, trial-and-error

decision. Rather, the possible effects on the parameters of pitch, loudness and quality must be considered. (p.152)

In addition, the voice therapist engaged in direct symptom management should -- as a general rule -- focus on one vocal parameter at a time. Bless (1988) points out:

A shotgun approach that provides several therapeutic strategies simultaneously may be both confusing and counterproductive. (p.140)

Implications for Intervention Planning

Management of a voice disorder -- depending on its nature and etiology -- may require voice therapy, medical-surgical intervention, or some combination of rehabilitation strategies. Input from various disciplines may be necessary, before an appropriate, comprehensive and efficacious therapy plan can be developed. Ideally, adults with voice problems would be treated in a comprehensive manner by a team of specialists. Such a team might include a speech-language pathologist and otolaryngologist. Depending on the nature and etiology of the voice disorder, additional team members could include a psychiatrist, psychologist, endocrinologist, neurologist,

gastroenterologist, or oncologist. Unfortunately, the team approach to the management of voice disorders is not routine. This is the case even in many medical settings in which potential interdisciplinary team members are on staff, but do not subscribe to a model of joint data collection and interpretation. For the vast majority of practicing speech-language pathologists, a team approach, however appealing a concept, cannot be realistically achieved in clinical practice. It is therefore essential for the speech-language pathologist to initiate communication, and seek consultation with appropriate allied medical personnel when treating patients with voice disorders.

Voice therapy may be undertaken in each of the following instances: (a) when medical-surgical management is contraindicated; (b) when medical or surgical intervention has been postponed to evaluate the effects of voice therapy; or (c) following medical-surgical intervention of a voice disorder secondary to vocal abuse, misuse or excessive musculoskeletal tension. It is important to remember that when surgical intervention is undertaken for an abuse-related voice disorder, it must be followed up with a period of voice therapy. The

failure to introduce vocal retraining as a post surgical adjunct, will most likely result in the recurrence of tissue pathology. Unless abusive vocal behaviors are eliminated, the long-term success of surgical intervention is unlikely. For those voice disorders not responsive to voice therapy, the clinician's primary responsibility -- if she is the first professional to consult with a patient -- is to make an appropriate medical referral. For example, if an examination of phonatory function and review of a comprehensive case history, suggest a dysphonia secondary to endocrine dysfunction, a referral to an endocrinologist for medical management would be indicated.

Medical clearance, minimally in the form of a recent visualization of the larynx, is essential prior to initiating any form of intervention with the dysphonic patient. For some forms of laryngeal pathology, more detailed assessment (such as stroboscopic endoscopy) can provide detailed information about the nature of vocal fold vibratory behavior and can yield more accurate and precise diagnoses. Such objective diagnostic data can assist the clinician in the development of appropriate intervention strategies as well.

In general, voice therapy with patients whose dysphonia is secondary to vocal abuse (e.g. - vocal nodules), should be guided by a search for any contributing or maintaining factors. A primary goal of intervention, is the identification of any behaviors or factors that have contributed to the development of vocal nodules. In addition, any factors that serve to maintain the voice disorder must also be identified. Once identified, a major focus of therapy is the implementation of a vocal hygiene program. The vocal hygiene program involves the systematic reduction, or ideally, elimination of abusive behaviors. As abusive factors are reduced or eliminated, the effect on the voice should be evaluated both subjectively and objectively. In addition to identifying and eliminating abusive vocal behaviors and misuse of the voice, the patient should be provided with alternative behaviors that promote good vocal hygiene. For example, if screaming has been identified as an abusive factor, in addition to eliminating screaming, the patient will need to adopt nonverbal/nonvocal means of gaining the attention of others, disciplining children, etc..

Patient Education

An important component of all vocal rehabilitation programs for adult dysphonics is a brief period of instruction regarding normal vocal fold structure and function. In addition, it is advisable to demonstrate to the patient the adverse effects of specific vocal or verbal behaviors in which they engage that are likely to have an adverse effect on vocal function. The use of graphic illustrations, functional models of the larynx, and high speed films or stroboscopic images contrasting normal and abnormal vocal fold vibration (in slow or apparent slow motion) can be very effective adjuncts to patient education. Plausible explanations for the patient's condition should be discussed and acknowledged. If, for example, it has been determined that a patient has a conversion aphonia, the patient needs to be presented with evidence of their ability to achieve vocal fold approximation. The clinician should provide the patient with immediate feedback of any instances of voicing (e.g. - coughing, laughing, etc.). Obviously, all clinical instruction must be individualized so that descriptions are appropriate to a particular patient's background. The clinician needs to be sensitive to differences in socioeconomic and educational level, cognitive function, age, gender, and culture

when counseling or providing instruction to patients.

CASE STUDY

Background Information

Marsha, a 36 year old female, was referred to an otolaryngologist (ENT) by her internist because of a hoarse voice. Indirect laryngoscopy revealed "... good mobility of the vocal cords, however, at the junction of the anterior and mid-third of the cords there are small nodules which are seen at rest." Visualization of the larynx by the ENT, resulted in the medical diagnosis of bilateral vocal nodules. The patient was subsequently referred for voice therapy. During a telephone consult with the otolaryngologist, it was reported that the nodules were newly formed with no evidence of fibrosis at the time of the examination. The initial responsibility of the voice clinician was to determine whether or not voice therapy alone was indicated. Due to the fact that the vocal nodules were small and newly formed, the ENT's recommendation of an initial period of voice therapy alone was appropriate. The voice clinician also needs to confirm the ENT's diagnosis of isolated vocal nodules, ruling out the possibility, however

remote, of the existence of other laryngeal or speech system pathology. Such confirmation can be accomplished during a brief period of diagnostic therapy.

Summary of Results

Marsha's initial therapy sessions were diagnostic in nature. In addition to the perceptual and objective assessment of the voice, a comprehensive case history was obtained. The purpose of obtaining a thorough case history was to identify any potential precipitating and maintaining factors. Since vocal nodules had already been confirmed visually, the focus of initial intervention efforts was to review Marsha's daily life activities in order to identify any sources of vocal abuse or misuse. Once abusive factors are identified, a major thrust of therapy is to systematically eliminate them while evaluating the effect of their elimination on the voice. An additional objective of diagnostic therapy for this patient was to obtain objective data regarding vocal function prior to the initiation of the vocal rehabilitation program. Several acoustic and

physiologic measures of the voice were obtained during the initial diagnostic therapy sessions. These measures later served as baseline data to which subsequent measures of vocal function were compared. The acquisition of repeated measures of vocal function every few weeks during therapy permitted an objective evaluation of therapy efficacy.

Based on information obtained from the case history, physical examination, and the objective evaluation of Marsha's voice during two sessions of diagnostic therapy, the following significant factors were identified:

Maintaining Factors Identified Through the Case History

- o Marsha's occupation as a part-time physical education teacher and summer camp counselor and the extraordinary vocal demands associated with these professional roles placed her at high risk for maintaining vocal nodules.
- o Marsha engaged in excessive throat clearing.
- o Marsha reported excessive instances of "raising" her voice and "yelling", particularly when at home fulfilling her role as the primary caretaker of her

three young children.

- o Marsha had adopted an habitually "explosive" speech pattern, which was characterized by high vocal intensity and a hard vocal attack.
- o Marsha operated a small business out of her home which required extended hours of daily telephone use.

The patient's history was negative with regard to: (1) the use of any prescription or over the counter drugs; (2) food or airborne allergies, and (3) any significant medical or health problems.

Maintaining Factors Identified Through Objective Measures of
Voice Production

Physiologic Assessment

- o Lung volume change, estimated from chest wall

movements, revealed Marsha's habitual use of an inappropriate lung volume range during speech production. Marsha's conversational speech typically took place between 25-50% of her vital capacity.

- o Marsha's measured vital capacity was 2.6 liters, which represents 83% of her expected VC, based on her height, age and gender.
- o For maximum sustained phonation tasks, Marsha used oppositional prephonatory chest wall movements (that is, rib cage expansion was accompanied by abdominal contraction throughout the prephonatory inhalation).
- o Mean airflow during sustained phonation at a comfortable pitch and loudness level was on the order of 280 ml/s.
- o The patient's average laryngeal airway resistance (Smitheran and Hixon, 1981) was calculated to be 25 cm H₂O/LPS.
- o Marsha's Maximum Phonation Time (MPT) was 8.7 seconds.

Acoustic Analysis

- o Acoustic analysis during sustained phonation at comfortable pitch and loudness yielded Relative Average Perturbation (RAP) values which were consistently in excess of 0.003.
- o Spectrographic analysis revealed excessive noise above 3000 Hz and the presence of significant levels of interharmonic energy.
- o Marsha's Mean Speaking Frequency (MSF) during reading of the Rainbow Passage was 179 Hz.
- o Acoustic analysis during sustained phonation and speech production revealed an average rise time of the amplitude envelope on the order of 28 ms.

Results of the Physical Examination

Physical examination and manipulation of laryngeal structures -- which consisted of digital manipulation of the hyoid bone and thyroid cartilage -- revealed no evidence of excessive musculoskeletal tension in the perilaryngeal area. In addition, no observable tension was noted upon palpation of the suprahyoid muscles in the region of the submandibular arch

during phonation at various pitches.

Assessment of Laryngeal Valving

The physiologic assessment of vocal function obtained prior to the initiation of therapy included measures of mean airflow during sustained phonation. Marsha's mean airflow during phonation was approximately 280 ml/sec. According to Koike and Hirano (1968), average airflow during phonation for an adult female is on the order of 93.7 ml/sec (S.D. = 31.6 ml/sec.). At therapy onset, Marsha's average airflow of 280 ml/s during phonation was too high.

The acquisition of simultaneous airflow and air pressure measures permitted the calculation of an estimate of laryngeal resistance during phonation. Marsha's estimated average laryngeal airway resistance during phonation was on the order of 25 cm H₂O/LPS. Marsha's average laryngeal resistance of 25 cm H₂O/LPS during phonation was too low. Smitheran and Hixon (1981) reported average values of glottal resistance on the order of

35.7 cm H₂O/LPS (S.D. =3.3 cm H₂O/LPS).

Marsha's high mean airflow values and reduced glottal resistance during phonation are consistent with the condition of bilateral vocal nodules. The high airflow levels and reduced glottal resistance during phonation are expected as a result of inadequate vocal fold closure due to the presence of the nodules (Yanagihara, 1970; Yanagihara and von Leden, 1967).

Additional evidence of inefficient laryngeal valving during phonation was obtained from pretherapy measures of vital capacity and MPT. Marsha's vital capacity was grossly within normal limits for her age, sex and body size (Kory, Callahan and Boren, 1961). Consequently, the patient's reduced MPT appeared to be the result of incomplete glottal closure during phonation, rather than due to inadequate ventilatory support.

The acquisition of physiologic data during speech and phonation served two important functions. First, they represented objective measures of vocal function which described the status of the ventilatory and phonatory mechanisms prior to the initiation of therapy. Second, they served as a useful yardstick for the ongoing assessment of therapy progress.

Acoustic Measures

Spectrographic analysis of sustained vowels revealed the presence of acoustic energy above 3000 Hz and excessive interharmonic energy. In addition, measures of Relative Average Perturbation (RAP), an index of vocal jitter, consistently exceeded 3%. These acoustic features of Marsha's vowel production are consistent with the perception of vocal roughness (Deal and Emanuel, 1978; Yanagihara, 1967; Koike, 1973; Takahashi and Koike, 1975; Emanuel and Whitehead, 1979; Emanuel and Sansone, 1969). Spectral analysis of normal vowel production results in a line spectrum with most of the acoustic energy between 0 and 3000 Hz. In other words, a vowel produced with good vocal quality has little energy above 3000 Hz. Vowels, after all, are low frequency speech sounds. However, due to the presence of vocal nodules, excessive glottal airflow and turbulence occur during phonation as a result of inadequate vocal fold closure (Isshiki, Yanagihara and Morimoto, 1966). Incomplete glottal closure during phonation results in the addition of a noise component to the complex quasiperiodic signal. In essence, a hoarse voice involves the addition of a noise component (which can be seen as a continuous spectrum) to

the complex quasiperiodic signal that characterizes normal phonation. Figure 2 compares the power spectra for a normal quality vowel (a) and that of a vowel produced with a hoarse vocal quality (b). The presence of interharmonic and high frequency energy are both characteristic of a hoarse voice. These acoustic features served to quantify the noise component in Marsha's vocal signal. As Marsha's therapy progressed, spectrographic analysis of sustained phonation would serve to document changes in the status of the vocal folds. Specifically, as the size of the nodules reduced, a concomitant reduction in the noise component would be expected. Thus, noninvasive objective acoustic measures of vocal function, rather than a more invasive endoscopic procedure, could be used to evaluate therapy efficacy.

A pretherapy assessment of vocal f_0 revealed a MSF of 179 Hz during reading. Although f_0 was low for a woman Marsha's age, it was within the expected range given the fact that bilateral vocal nodules were present. A lowered MSF was expected since the vocal nodules increased the mass of the vocal folds. Direct intervention to alter f_0 would therefore be an inappropriate therapeutic goal and an attempt to modify f_0 was contraindicated.

THE MANAGEMENT PLAN

LONG-TERM PLANNING PHASE

Long-term goals

The development of Marsha's long term therapy goals was guided by: (1) the medical diagnosis of vocal nodules, (2) objective baseline acoustic and physiologic data obtained during diagnostic therapy that quantified the effect of vocal nodules on vocal function, and (3) the clinician's ability to identify relevant maintaining factors from an evaluation of case history information, diagnostic therapy findings and the clinician's experience and knowledge base.

As a general rule, the initial focus of therapy with vocal nodules should center on the identification of abusive vocal behaviors. Once these behaviors have been identified, a vocal hygiene program should be developed by the voice clinician in consultation with the patient. The primary objective of a vocal hygiene program is to systematically reduce, or ideally, eliminate, all of the abusive vocal behaviors identified. Given the nature of Marsha's voice disorder, it was expected that

voice therapy would yield significant positive changes in the status of the larynx within approximately eight to twelve weeks. Consequently, Long Term Goal (LTG) achievement for this patient was expected within 3 months of the initiation of voice therapy. At this time -- if voice therapy had been successful -- the vocal nodules should be eliminated or significantly reduced in size.

One long term goal (LTG) was established a priori:

LTG 1: Marsha will gain an improved understanding of:
(a) normal laryngeal structure and function and
(b) the effect of vocal abuse on the vocal fold tissues and on her ability to produce good quality voice.

Procedural Approach: LTG 1

All voice therapy should begin with a period of instruction in which the patient has an opportunity to visualize the structure and function of normal vocal folds and compare these images with those characteristic of the patient's specific laryngeal pathology. In the case of Marsha, she received instruction -- in terms that she could understand -- regarding the anatomy and physiology of the vocal mechanism. This instruction was replete with still photographs and videotapes of

high speed cinematography, showing vocal fold vibration in slow motion. In addition, based on information obtained from the case history, potential abusive factors were identified and their effect on vocal fold structure and function was discussed. Based on this instruction, Marsha was required to identify common etiological factors associated with vocal nodule development and describe to the clinician the effect of vocal nodules on vocal fold vibration. Following a brief tutorial regarding good vocal hygiene, the patient was asked to identify desirable vocal behaviors that are not harmful to the laryngeal mechanism and that would most likely facilitate a reduction in the size of her vocal nodules.

As part of an extensive case history, the patient described, in great detail, her daily routines. With the guidance of the clinician, Marsha's vocal habits were examined within the context of the daily demands placed on her voice. Any potential instances of vocal abuse or misuse were identified and discussed.

Decision-making regarding LTG 2

The finding of an average rise time of the amplitude

envelope of 28 ms is consistent with the use of a hard vocal attack (Koike, 1967, 1973). The decision to establish a long term therapy goal to promote use of a soft vocal attack was made after objective acoustic evaluation confirmed the clinician's perception of Marsha's frequent use of a hard vocal attack during conversational speech. The following LTG was established:

LTG 2: Marsha will substitute for abusive vocal behaviors those that promote good vocal hygiene. Specifically, she will adopt and habituate use of a soft vocal attack in discourse.

Procedural Approach: LTG 2

The same instrumentation used to evaluate the rise time of the amplitude envelope was incorporated into a visual biofeedback paradigm. Marsha was instructed in the use of a "confidential voice" (Colton and Casper, 1996). Real-time visual records of her phonatory rise time were monitored during the production of carefully prepared phrases. Negative practice was used in which the patient contrasted different types of

vocal attack while evaluating simultaneously the quality and visual representation of the type of vocal attack used. During therapy sessions, Marsha used this visual feedback to help establish an awareness of auditory and tactile and kinesthetic cues associated with the more efficient soft vocal attack which characterized the "confidential voice."

Decision-making regarding LTG 3

Marsha's vital capacity, although somewhat low given her age, gender and body size, was grossly within normal limits (83% of her expected vital capacity). The patient's vital capacity served as a gross screening measure which ruled out the possibility of significant ventilatory system pathology. During a maximum effort task, Marsha was able to exchange 2.6 liters of air, which suggested that she had adequate breath support for the purposes of speech production. However, further assessment of ventilatory function revealed that Marsha habitually adopted an inefficient breathing pattern for speech. It should be emphasized that the vast majority of patients who present with vocal nodules generally do not require intervention specifically directed at the ventilatory system. In Marsha's case,

diagnostic therapy revealed the need to modify her speech breathing pattern. A brief review of normal speech breathing behavior should help the reader understand the nature of Marsha's problems in ventilatory support. During quiet tidal breathing (at rest breathing), a person begins and ends each ventilatory cycle around Resting Expiratory Level (REL). In the upright posture, REL corresponds to a lung volume level of approximately 37 - 40% of a person's vital capacity (VC). During vegetative breathing, the size of the chest wall is increased only slightly beyond its resting size at REL. Specifically, lung volume level increases on the order of 400 - 500 ml of air beyond REL. For most speech of normal conversational loudness, a person only needs to increase lung volume an additional 250 - 350 ml of air beyond the level required for quiet tidal breathing. Thus, most speech of normal conversational loudness begins at around 60% of a person's vital capacity (which represents an increase in lung volume to approximately 10 to 20 percent of VC above REL (Cavallo, 1988). Most speakers will terminate speech of normal conversational loudness at or slightly below REL (that is, around 35 - 40% of the vital capacity). Figure 3 shows typical lung volume

excursions during speech at normal conversational loudness for a normal speaker. Marsha typically initiated speech around 50% of her vital capacity. In effect, she did not take in enough air to support speech adequately. In addition, Marsha frequently terminated speech at a lung volume level well below REL (to approximately 25% of her vital capacity). Figure 4 illustrates the lung volume change re: REL that characterized Marsha's pretherapy speech breathing behavior. As a consequence of using an inappropriate lung volume range, Marsha's ability to generate the required subglottal pressures for speech was compromised. By not initiating speech at an appropriate lung volume level, Marsha failed to take advantage of the recoil forces which provide relatively small, positive relaxation pressures. Invariably, Marsha's adoption of an inefficient speech breathing pattern, added an additional strain on the vocal mechanism during speech production.

Another problem identified during diagnostic therapy was Marsha's use of oppositional rib cage and abdominal movements throughout inhalation prior to performing maximum effort tasks, such as the Maximum Sustained Phonation (MSP). Normal prespeech inspirations involve cooperative movements of the rib cage and

abdomen, with the exception of a brief oppositional movement of the chest wall components that may occur just before phonatory onset. Specifically, the size of both the rib cage and abdomen increase until approximately 100 ms prior to phonation, at which time, a sudden and small gesture of the chest wall characterized by rib cage expansion and abdominal contraction may occur (Baken, Cavallo and Weissman, 1979; Baken and Cavallo, 1981). For maximum effort tasks, Marsha consistently increased the size of the rib cage (an inspiratory gesture) and a decreased the size of the abdomen (an expiratory gesture) throughout the entire prephonatory inspiration. This represents an idiosyncratic breathing pattern that proves to be highly inefficient. It should be noted that Marsha did not typically use this abnormal breathing pattern during speech and reading tasks, although she occasionally demonstrated oppositional movements of the two chest wall components when taking a deeper breath to support louder speech utterances.

To summarize, physiologic assessment of Marsha's breathing for speech, revealed an abnormal and inefficient breathing pattern. Consequently, direct intervention in the form of breathing exercises was appropriate for this patient. An

additional long term goal (LTG) was developed for this patient.

LTG 3: Marsha will consistently produce speech of normal conversational loudness within an appropriate lung volume range (approximately 35 - 60% of the vital capacity)

Procedural Approach: LTG 3

A visual biofeedback paradigm was introduced to monitor and modify Marsha's speech breathing pattern. Using variable inductance plethysmography, a real time lung volume estimate was derived from chest wall movements and displayed on a cathode ray oscilloscope. REL was determined at the beginning of the therapy session while the patient was engaged in quiet tidal breathing. This level was marked on the oscilloscope, providing the patient with a record of lung volume change re: REL. An estimate of the midvolume range was marked on the oscilloscope screen and the patient was directed to produce speech within that range. The effect of this modification of breathing on voice quality and the patient's "feeling" of reduced laryngeal effort and tension were evaluated by the patient and discussed with the clinician. Eventually, the patient was required to speak using an appropriate lung volume range without visual

cues.

SHORT-TERM PLANNING PHASE

In the development of short term goals (STGs), the following performance demands were manipulated by the clinician: linguistic complexity and vocal intensity. For STG 2, linguistic complexity was manipulated as a performance demand. Marsha was initially required to monitor instances of vocal abuse in order to develop a heightened sense of awareness regarding their existence and frequency of occurrence. Session goals designed to reduce the frequency of occurrence of abusive behaviors -- a more complex task -- were not initiated until after Marsha demonstrated the ability to consistently identify instances in which she engaged in vocal abuse. For STG 3, intensity was manipulated as a performance demand. Marsha was required to produce speech within an appropriate lung volume range at a normal conversational loudness level (STG 3a) prior to initiating goals designed to produce speech within an appropriate lung volume range at a louder level (STG 3c).

Short-Term Goals

The short term goal (STG) developed to achieve LTG 1 was:

STG 1: Marsha will demonstrate the ability to identify common etiologic and maintaining factors of vocal nodules and describe their effect on the vocal fold tissues and on voice production.

Delimited Procedural Approach: STG 1

Early in the therapeutic process, considerable time was devoted to improving the patient's understanding of those factors that caused and maintained the nodules, and the effect of vocal nodules on the quality of her voice. In addition to providing the patient with critical information about the nature of her dysphonia, activities such as contrasting normal and dysphonic voices and observing visual images which illustrated

differences in the vibratory behavior of normal and pathologic vocal folds had the effect of increasing the patient's motivation to eliminate the nodules. Marsha's initial reaction to seeing vocal nodules was "My God, is that what mine [vocal folds] look like?" After a few sessions reviewing normal and abnormal vocal fold structure and function, and comparing normal and dysphonic voices, Marsha was highly motivated to make the behavioral changes required for a successful therapeutic outcome.

Decision-Making regarding STGs 2a - 2f

Following extensive discussion and consultation with the patient, a number of behaviors were identified as likely maintaining factors. Marsha's position as a physical education teacher placed considerable demands on the voice as did her role as primary caretaker of her three children. Marsha reported great "chaos" in the home which resulted in "having to raise my voice from the moment I get home, until the children are asleep." From the case history information, specific behaviors were identified that were inconsistent with good vocal hygiene. In addition to routinely raising her voice and shouting at home

and at work, Marsha reported that she used the telephone frequently -- often in a noisy home environment -- as a function of a private, home-based business. Clinical observation revealed excessive throat clearing that was not associated with an upper respiratory infection or allergy and frequent use of a hard vocal attack. These four behaviors: shouting, telephone use, throat clearing, and use of a hard attack were targeted as abusive vocal behaviors in need of elimination. As a substitute for these behaviors, Marsha was instructed in the use of a soft attack and the "confidential" voice.

Specific short term goals (STG's) established to achieve LTG 2 included:

- STG 2a: Throughout the week, Marsha will monitor instances of vocal abuse (specifically, instances of raising her voice, throat clearing and the frequency and duration of telephone use).
- STG 2b: Marsha will produce speech without "raising her voice" and "yelling", both at home with her children and in the work environment.
- STG 2c: Marsha will eliminate nonproductive throat clearing.
- STG 2d: Marsha will reduce telephone use and engage in telephone conversations only under conditions of low background noise.
- STG 2e: Marsha will substitute use of a soft vocal attack

for hard vocal attack during the production of short, prepared phrases.

STG 2f: Marsha will adopt and use a "confidential voice" throughout the therapy session and on a daily basis when engaged in discourse on a one-to-one basis with others.

Delimited Procedural Approach : STGs 2a - 2f

Initial session objectives toward the achievement of the long term goal to adopt healthy vocal behaviors and eliminate vocal abuse were designed to increase Marsha's awareness of the degree to which she engaged in abusive vocal behaviors.

Increased awareness of vocal abuse was achieved by requiring the patient to identify and chart all instances of these behaviors. Instances of throat clearing were monitored both in and out of the therapy milieu. Instances of telephone use and "raising her voice" were tracked outside of the therapeutic setting by the patient. In the case of telephone use, Marsha was required to record the duration of each telephone call and describe the level of ambient noise in the immediate environment during telephone use. Self-monitoring of her voice remained an integral goal throughout therapy.

After several weeks of charting, Marsha had increased her

sensitivity to those abusive vocal behaviors identified jointly by the patient and clinician. At this point in therapy, an additional session goal was introduced which required that -- in addition to charting these behaviors -- Marsha would reduce their frequency and duration.

Instruction in the production and adoption of the "confidential" voice was introduced early in therapy. The clinician trained Marsha to produce a less forceful form of phonation during speech production. Although Marsha was initially quite resistant to carrying over this voice outside of the therapy situation, she did succeed in using the "confidential" voice more consistently during her activities of daily living. When engaged in those activities which Marsha believed she could not use the "confidential" voice, she was provided with other alternatives to raising her voice or shouting. For example, when leading her physical education classes, she would use nonverbal signals whenever possible. When instructing the students regarding the activities for each gym class, she would instruct one student using the "confidential" voice and designate that student as class leader who was given the responsibility to provide instruction to the

class. In addition, she had her school district purchase a megaphone, which she used during physical education classes and school sports events.

Another set of goals which received direct attention during therapy sessions, was implemented to improve Marsha's ability to differentiate between an abusive, "hard" vocal attack and a more efficient "soft" vocal attack. This goal was accomplished quickly, since Marsha was provided with immediate, real time visual feedback of the type of vocal attack she had used. Over a period of several weeks, she was able to consistently produce spontaneous speech using a "soft" vocal attack without the aid of a visual signal.

Decision-making regarding STGs 3a - 3c

Objective assessment of Marsha's ventilatory behavior during speech revealed that she had an adequate lung volume reserve. However, she habitually used a lung volume range that was inappropriately low both for speech of normal conversational loudness and for speech at increased loudness levels. She also

used an inefficient ventilatory pattern which was characterized by oppositional chest wall movements during maximal effort phonatory tasks.

Specific STG's designed to accomplish LTG 3 included:

STG 3a: Marsha will consistently produce conversational speech (at a normal loudness level) within the midvolume range.

STG 3b: During a Maximum Sustained Phonation (MSP) task, Marsha will consistently accomplish prespeech inspiration using cooperative movements of the rib cage and abdomen.

STG 3c: During conversational speech production at high vocal intensity level, Marsha will consistently accomplish prespeech inspiration using cooperative movements of the rib cage and abdomen.

Delimited Procedural Approach: STGs 3a - 3c

Marsha's breathing problems were addressed within the context of a visual biofeedback paradigm. Using variable inductance plethysmography, records of real time rib cage and abdominal movements as well as an estimate of lung volume change were available. In order to accomplish STG 3b, rib cage and abdominal movements were monitored on an oscilloscope screen.

An estimate of lung volume change was monitored by the patient on the oscilloscope during speech production in those session activities devoted to the achievement of STG 3a.

Again, the availability of visual records of chest wall movements and lung volume change, resulted in Marcia's ability to modify her breathing pattern within a few therapy sessions. Her ability to consistently produce speech within the midvolume range in the absence of visual cues was achieved somewhat more slowly, over a period of several weeks.

SESSION PLANNING PHASE

Session Goals

In the development of the session goal (SG) sequence, the following variables were manipulated in order to alter performance demands: task complexity, linguistic complexity, biofeedback, intensity level of speech or phonation. Marsha was required to identify the existence of vocal nodules and differentiate vocal folds with and without nodules from visual images before undertaking the more complex tasks: identifying

etiologic and maintaining factors; describing the effect of vocal nodules on the voice; and identifying vocal behaviors that are consistent with good vocal hygiene.

In the development of SG 2, Marsha was first required to monitor the frequency of occurrence of abusive behaviors. When she was unable to do so reliably, the number of behaviors she was required to monitor was reduced.

Before Marsha was required to alter the type of vocal attack used, she was first required to master less complex tasks: interpersonal discrimination between hard and soft attack; intrapersonal discrimination between hard and soft attacks with visual cues; intrapersonal discrimination between hard and soft attacks without the benefit of visual cues; production of soft attack in short phrases; production of soft attack in discourse.

For SG 3, the variables linguistic complexity, biofeedback (visual record of lung volume change), and intensity level were manipulated by the clinician in the development of SGs 3 a-f. Marsha was required to produce short phrases, and then conversational speech within the midvolume range with the benefit of visual biofeedback. (SGs 3a and 3b). After

successful achievement of these session goals, Marsha was required to produce spontaneous speech in the midvolume range without the benefit of visual biofeedback (SG 3c).

SGs 3d - 3f, required Marsha to use cooperative movements of the rib cage and abdomen before phonation, short phrases and finally, in discourse. For SGs 3g - 3i both linguistic context and intensity were manipulated: Marsha had to produce phonation, short phrases and discourse using cooperative rib cage and abdominal movements at greater than normal conversational loudness levels.

The following session goals (SGs) were developed to achieve this LTG 1 and STG 1:

- SG 1a: Following clinician instruction, Marsha will identify vocal nodules and will contrast vocal folds with and without nodules from still and videotape images with 100% accuracy.
- SG 1b: Following clinician instruction, Marsha will identify etiologic and maintaining factors associated with nodule development.
- SG 1c: Following clinician instruction, Marsha will describe accurately the effect of vocal nodules on voice production.

SG 1d: Following clinician instruction, Marsha will identify vocal behaviors which promote a reduction in the size of vocal nodules.

Session Procedures: SGs 1a - 1d

A significant portion of the initial therapy sessions was devoted to educating the patient about the nature of her disorder by contrasting visual representations of vocal fold vibration and auditory recordings of phonation for individuals with and without vocal nodules. Following a brief period of instruction from the clinician, Marsha achieved the criterion of identifying and contrasting normal and disordered phonation with 100% accuracy very quickly.

The following session goals were established to achieve LTG 2 and STG's 2a - 2f:

SG 2a: On a daily basis (a minimum of 3 times per day for 30 minute periods), Marsha will monitor and maintain a frequency count of the following vocal behaviors: incidents of throat clearing; number and duration of telephone calls; incidents in which the voice was used with excessive loudness.

SG 2b: Marsha will reduce -- by at least 25% -- the number of instances in which the following behaviors occur during the week: nonproductive throat clearing; use of an excessively loud voice; and the frequency and duration of

telephone calls.

- SG 2c: Marsha will discriminate between the clinician's use of hard and soft vocal attacks during conversational speech with 90% accuracy.
- SG 2d: With the assistance of visual biofeedback, Marsha will demonstrate the ability to discriminate between her use of hard and soft vocal attacks with 90% accuracy.
- SG 2e: Without the assistance of visual cues, Marsha will demonstrate the ability to discriminate between her use of hard and soft vocal attacks while reading short phrases with 90% accuracy.
- SG 2f: Without visual cues, Marsha will produce a soft attack while reading short phrases with 90% accuracy.
- SG 2g: Marsha will produce a soft attack during spontaneous speech production throughout the therapy session with greater than 80% accuracy.
- SG 2h: Marsha's spontaneous speech during the therapy session will be characterized by the "confidential" voice greater than 90% of the time.

Session Procedures: SGs 2a - 2h

Initially, Marsha was required to monitor outside of therapy the frequency at which those abusive behaviors identified. After one week, it became apparent that Marsha was having difficulty charting behaviors on a consistent basis, and specifically, monitoring instances of throat clearing. In an

attempt to simplify the task for the patient, she was provided with a preprinted charting pocket notebook prepared by the clinician. In addition, she was not required to monitor throat clearing outside the therapy setting until she could consistently identify the behavior in therapy. Consequently, an additional session goal had to be developed:

SG 2a₁: Marsha will identify instances of throat clearing during the therapy session with 90% accuracy.

The clinician was less concerned with the accuracy and reliability of Marsha's charting than with the heightened awareness she would gain regarding these abusive behaviors as a result of this activity.

Marsha was not required to reduce the number of instances of abusive vocal behaviors (SG 2b) until she had charted the frequency of occurrence of those behaviors for a few weeks.

With regard to replacing the hard vocal attack with a soft attack, Marsha was first required to discriminate between the two types of vocal attack in the clinician's speech (interpersonal discrimination). Goals directed at intrapersonal discrimination between hard and soft attack were not attempted until SG 2b had been achieved. Intrapersonal discrimination and

between and production of hard and soft attacks were addressed within therapy sequentially in the following contexts:

intrapersonal discrimination between hard and soft attacks while reading short phrases first with and then, without, visual feedback; production of soft attack while reading short phrases with visual cues; and finally, producing soft attack during discourse.

A session goal was also introduced requiring Marsha to use a "confidential" voice in therapy 90% of the time. Although not a formal goal, Marsha was encouraged to use this voice outside of therapy whenever communicating on a one-to-one basis with others.

The following session goals were developed to achieve LTG 3 and STG's 3a - 3c:

SG 3a: Within a visual biofeedback paradigm, Marsha will consistently produce short phrases between 35 - 60% of her vital capacity (within the midvolume range) with 90% accuracy.

SG 3b: With the assistance of visual cues, Marsha will produce speech at a normal conversational loudness level between 35 - 60% of her vital capacity (within the midvolume range) with 90%

accuracy.

SG 3c: Without the assistance of visual cues, Marsha will produce spontaneous speech at a normal conversational loudness level between 35 - 60% of her vital capacity (within the midvolume range) with 80% accuracy.

SG 3d: During prephonatory inspirations, Marsha will consistently use a cooperative breathing pattern (rib cage and abdominal expansion) in preparation for phonation at a comfortable loudness level with 90% accuracy.

SG 3e: During prespeech inspirations, Marsha will consistently use a cooperative breathing pattern (rib cage and abdominal expansion) in preparation for short phrases produced at a comfortable loudness level with 90% accuracy.

SG 3f: During prespeech inspirations, Marsha will consistently use a cooperative breathing pattern (rib cage and abdominal expansion) in during conversational speech produced at a comfortable loudness level with 90% accuracy.

Therapy activities that required the patient to produce speech and phonation at high intensity levels were postponed until the later stages of therapy at a time when the size of the vocal nodules had been reduced significantly.

SG 3g: During prephonatory inspirations, Marsha will consistently use a cooperative breathing pattern (rib cage and abdominal expansion) in preparation for loud phonation with 90% accuracy.

SG 3h: During prespeech inspirations, Marsha will consistently use a cooperative breathing pattern (rib cage and abdominal expansion) in preparation for short phrases produced at greater than normal loudness level with 90% accuracy.

SG 3i: During prespeech inspirations, Marsha will consistently use a cooperative breathing pattern (rib cage and abdominal expansion) in during conversational speech at greater than normal loudness level with 90% accuracy.

BIOFEEDBACK-BASED INTERVENTION

Progress in therapy was facilitated, in part, by the availability of biofeedback procedures to assist Marsha in changing behaviors. The instrumentation used to evaluate Marsha's ventilatory support for speech and the type of vocal attack were well suited for a biofeedback paradigm. Marcia was provided with real-time visual representations of lung volume change and the type of vocal attack used. Consequently, changes in the lung volume range used for speech and the type of vocal attack used were accomplished quickly. Once an appropriate lung volume range within the vital capacity was used consistently by the patient and a "soft" vocal attack had been consistently

adopted, visual feedback was gradually withdrawn. The integration of a biofeedback paradigm into the vocal rehabilitation program maximized the efficiency with which new, nonabusive vocal behaviors were taught and adopted.

MONITORING THERAPY EFFICACY

The following measures of vocal function were obtained weekly throughout therapy in an effort to evaluate therapy efficacy: fundamental frequency (f_0) and Relative Average Perturbation (RAP) during 3 second midsegment samples of phonation at high, low and comfortable loudness levels. Throughout therapy, mean f_0 level during sustained phonation increased steadily from approximately 175 Hz to 194 Hz at the conclusion of voice therapy 3 months later. RAP values decreased from 3% and greater at therapy onset to approximately 0.23% at the time therapy was terminated. Marcia's Maximum Phonation Time (MPT) increased from 8.7 s at therapy onset to 15.8 s at the time of discharge. In addition, spectral analysis of vowels at the conclusion of therapy revealed substantially lower levels of interharmonic and high frequency energy. These

objective measures of vocal function were consistent with a reduction in the size of the nodules. This conclusion was confirmed by indirect laryngoscopy 3 months after the initiation of therapy. According to the otolaryngologist's report, "The left vocal cord has minimal thickening at the junction of the anterior and mid-third of the cord. There is no evidence of a nodule on the right vocal cord. The cords approximate well."

Although complete resolution of tissue pathology had not been accomplished, the patient was asymptomatic and very pleased with her voice. Consequently, she opted to terminate therapy. During the final few therapy sessions, the importance of maintaining good vocal habits was stressed in order to minimize the possibility of a recurrence of tissue pathology.

SUMMARY

The clinical case described in this chapter is representative of the decision making process the voice clinician engages in when working with an adult who presents with abuse-related dysphonia. It should be stressed that every patient presents with their own unique circumstances. Every

individual has a unique set of laryngeal structures; presents with different problems and abilities, and motivation level. Adult dysphonics, depending on their personalities and lifestyle, place different demands on their voices. Consequently, although certain general principles may apply to all patients, each voice case should be approached on an individual basis, taking into account the different characteristics and circumstances each patient presents.

The initial decision the clinician needs to make is whether or not voice therapy alone is indicated. In order to make this decision in an informed manner, the voice therapist needs to have comprehensive evaluation data available. It is simply not enough to receive a report from an otolaryngologist in which a specific tissue pathology is identified and voice therapy is recommended. Additional diagnostic data and consultation with the otolaryngologist are required if the voice clinician is to be in a position to generate appropriate therapeutic goals. In this case, well informed clinical decisions could not have been made without the acquisition of objective physiologic measures. This required access to and a working knowledge of a fairly sophisticated instrumentation array. For example, if the voice

clinician limited the evaluation of Marcia's ventilatory function to the acquisition of a vital capacity -- or if ventilatory behavior had not been assessed at all -- important target goals would not have been identified. In Marcia's case, the vital capacity obtained was grossly within normal limits. In the absence of other objective data, the voice clinician might have been tempted to conclude that ventilatory function for speech was adequate and decide not to direct intervention efforts toward the ventilatory system. Although a vital capacity that is significantly below an expected value usually suggests some form of ventilatory dysfunction, it does not reveal how a patient manages the lung volume reserve for the purposes of speech production. A more precise examination of Marcia's ability to manage lung volume during speech production, revealed an abnormal speech breathing pattern. Consequently, specific goals to modify speech breathing behavior were established.

The integration of acoustic and physiologic measures of phonation and speech into voice therapy provided objective measures of vocal function which the clinician and patient were able to use to monitor progress in therapy. For Marcia, the

availability of quantitative data regarding the status of her vocal nodules, proved highly motivating. She had received instruction regarding the relationship between the objective measures of vocal function and the presumed status of her vocal nodules. Once Marcia had seen the RAP values drop and had visualized the reduction of signal noise in the power spectra of her vowels, she became extremely motivated to continue making positive changes regarding her vocal behaviors. In addition, several instrumentation systems were used in therapy to provide Marcia with visual biofeedback of several speech behaviors that were in need of modification. The availability of these visual signals helped facilitate positive changes in the patient's breathing pattern and the type of vocal attack used.

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