# The Psychopathology of Voice Disorders

L.A. Rammage, H. Nichol and M.D. Morrison


## Abstract

*Various psychopathological processes that may be active in symptom formation involving the voice have been identified. Often etiological factors producing a dysphonia include a complex mixture of organic and psychogenic processes. The specific etiological relationships need to be understood so that treatment and therapy are directed most appropriately.*

*The voluntary muscles used for speech and phonation are the final common path of dysphonias of psychogenic origin (Aronson, 1985; Barlow, 1973); therefore, the identification and reduction of muscle misuses contributing to psychogenic voice disorders is a primary part of the therapy program. An interdisciplinary approach to assessment/diagnosis, classification and treatment of patients with psychogenic dysphonias facilitates comprehensive voice care.*


## Introduction

The function of voice is principally to communicate with other people. It is thus seen to have a major social component which serves to disperse feelings of psychological isolation. In order to speak one requires an organic apparatus capable of producing sound, psychological intent to communicate and a social context — since most people do not talk much to themselves, they think instead — in which one feels the desire to talk. Voice production, therefore, clearly rests upon the outcome of the interaction of factors that can be conceptualized as being at organic, psychological and social levels. In this discussion we are concerned principally with factors operating at the psychological level.

The term psychopathology is used here to refer to the abnormal or maladaptive behavioural or mental activities of patients displaying dysphonias in which psychogenic factors play a substantial role. Various types of pathogenetic processes are involved in the production of these dysphonias; these will be reviewed and their impact on the voluntary musculature of phonation, which is the final common path in the production of the dysphonias they produce, will be emphasized. A brief comment on therapeutic approaches will be made.


Linda A. Rammage, M.Sc.
Speech-Language Pathologist, Voice Clinic,
Vancouver General Hospital.

Hamish Nichol, M.B., F.R.C.P.(C)
Consulting Psychiatrist, Voice Clinic,
Vancouver General Hospital; and Associate Professor,
Department of Psychiatry, University of British Columbia.

Murray D. Morrison, M.D., F.R.C.S.(C)
Director, Voice Clinic, Vancouver General Hospital;
and Associate Professor, Division of Otolaryngology,
Department of Surgery, University of British Columbia.


## Types of Pathogenesis

The following are the principal types of pathogenesis concerned with somatic signs and symptoms that have a psychogenic basis which may produce dysphonia (Tyhurst, 1978).

1. Tensional symptoms arise from the overactivity of the autonomic and voluntary nervous system in individuals who are unduly aroused and anxious. This leads to voluntary muscle misuse because of generalized muscular hypertonicity, and to conditions variously diagnosed as functional dysphonias, vocal hyperfunction and muscular tension dysphonia (Morrison et al, 1983; Belisle, Morrison, 1983). These are often associated with psychiatric conditions such as adjustment disorders, anxiety disorders or personality trait disturbances. This category comprises the greatest proportion of patients with voice disorders having a psychogenic basis.

2. Symbolic symptoms occur on the basis of an unconscious substitution of a somatic symptom involving the sensory or voluntary motor nervous system for a psychological conflict. This is the conversion disorder referred to so frequently in the psychiatric literature and commonly labeled hysterical aphonia in speech-language pathology and otolaryngology texts. It gives rise to dysphonia when the muscles involved are those of phonation.

3. Hypochondriacal symptoms, or the self-fulfilling anticipation of poor voice production, occur in those who are unduly aware of, or responsive to, sensations arising from a particular portion of their anatomy — in the case of those with dysphonias, usually their mouth, throat and respiratory system. In these cases the associated psychiatric diagnoses are often personality trait disturbances involving obsessive-compulsive and dependent features, as well as hypochondriacal ones.

4. Depressive equivalent symptoms may arise in those individuals who are not complaining overtly of any depressive symptomatology but who are suppressing the impulse to cry or to express anger verbally; adjustment, dysthymic and affective disorders of the depressive type are the psychiatric diagnoses found in these cases.

5. The next pathogenesis of psychogenic origin is frequently revealed in taking a history from the patient. The same patient may show symptoms of symbolic, tensional and hypochondriacal origin at any particular time. One type of pathogenesis may reinforce another: for

example, there may be a hypochondriacal exaggeration of a tensional symptom. It is relevant to note these differing types of pathogenesis since the treatment of the individual symptoms they produce may well need to be quite different.

6. Combined organic and psychogenic processes are frequently found and obviously require differing therapeutic approaches. The factors producing a dysphonic voice, then, are often a complex mixture of organic, psychological and social factors, any one of which may be *predisposing, precipitating* or *perpetuating* agents. A relatively minor organic change such as edema, infection, polypoidal change or neoplasia may trigger functional misuse, particularly if the individual is unduly anxious about his or her voice or health, so that most of the voice problem is of psychological etiology; this is particularly likely to happen if there is an organic predisposing condition such as reflux esophagitis or acid laryngitis. In a more dramatic example, the hoarseness associated with an early vocal cord cancer may be due as much to the generalized hypertonicity of laryngeal muscles as to the malignancy itself. Interestingly, when the carcinoma has resolved after radiation, the dysphonia of psychological etiology causing most of the hoarseness may also be improved; but this dysphonia may persist and lead to continuing concern about the persistence of the tumour which will then require psychotherapy for its resolution. Conversely, psychologically and socially induced voice misuse may lead to a secondary laryngeal organic problem, such as polypoidal degeneration.

## *Interrelationships among Etiological Factors in Psychogenic Dysphonias*

We have tried to model diagrammatically the ways in which the various etiological factors discussed above may interact with each other in predisposing, precipitating or perpetuating a dysphonia (Figure 1). A vertical line dropped through the layers of the diagram represents a given patient at a particular time. Portions of the line lying within the layer represent the respective proportion of a factor giving rise to the dysphonia. For example, a line drawn far to the right would represent a condition almost totally psychologically based, and a line far to the left would be almost all organic in etiology. The ebb and flow over time of the severity of both organic and psychological stressors in a patient are depicted by waves at the top and the bottom.

## *The Function of Psychogenic Dysphonia*

The function of the symptom pf psychogenic dysphonia may vary substantially from patient to patient, and these differences need to be understood for therapy to be directed appropriately. The term *primary gain* refers to the reduction of anxiety, tension and conflict within the awareness of the individual which is provided by the production of a symptom of psychogenic origin, such as a dysphonia, through the employment of various defence mechanisms like regression, repression, denial, reaction formation and isolation of affect. This psychogenic symptom of dysphonia, while unpleasant in itself, constitutes for the patient the lesser evil than the personal problem from which it arose.

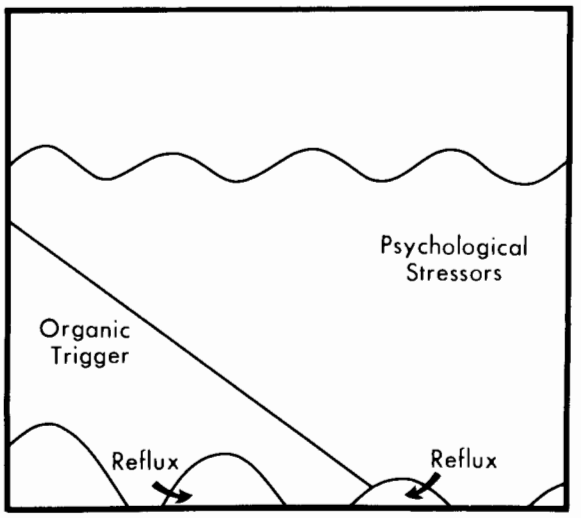


**Figure 1: Interrelationships among etiological factors in psychogenic dysphonias**

We have tried to model diagrammatically the ways in which the various etiological factors discussed above may interact with each other in predisposing, precipitating or perpetuating a dysphonia. A vertical line dropped through the layers of the diagram represents a given patient at a particular time. Portions of the line lying within the layer represent the respective portion of a factor giving rise to the dysphonia. For example, a line drawn far to the right would represent a condition almost totally psychologically based, and a line far to the left would be almost all organic in etiology. The ebb and flow over time of the severity of both organic and psychological stressors in a patient are depicted by waves at the top and the bottom.

The following case provides an example of primary gain:

> A middle aged woman had a respiratory tract infection that produced a dysphonia during the 3 months while she was awaiting her triple bypass cardiac surgery about which she was very apprehensive. The infection resolved but her dysphonia persisted even after her operation, which she did not accept as having been as successful as her cardiologist maintained. Her inability to accept the cardiologist's positive prognosis was in part due to recent recurrent and persistent sensations of burning and constriction in her chest and throat. It emerged that a hiatus hernia had been previously diagnosed and she had been observing a protocol to treat it in the past but had not followed the regimen since her cardiac problem was identified. During this period of acute illness and anxiety then, the symptoms of gastroesophageal reflux were intensified.
>
> The primary gain that she accrued by substituting anxiety about her throat and voice for anxiety about her heart was reinforced by her husband, who was often

heard saying to her: "Well it's better to have a hoarse voice to worry about, than a bad heart."

The term *secondary gain* refers to the benefit derived by an individual from the external environment on the basis of others' perceptions of his or her evident distress; this may take the form of monetory compensation, increased attention or sympathy, and the satisfaction of dependency needs. These secondary gains may serve to reinforce the patient's disorder and perpetuate its persistence.

Sociologists have emphasized that the assumption by an individual of the "sick role" and the display of "invalid behaviour" may convey many valuable privileges on the individual in our society (Kendell, 1983). Invalids may not only be exempted from normal social obligations but may also be freed of responsibility for their behaviour. Those about them often feel under the obligation to be kind and sympathetic to them and to take over some of their responsibilities. Society requires of the individual that he or she should seek appropriate treatment for the disability so that he or she exercises the privileges of the sick role for as brief a time as possible. Failure to do this is perceived correctly as the employment of the symptom for secondary gain.

In appraising our patients and their responses to treatment it is necessary to seek to differentiate the extent to which the symptom of dysphonia has primary or secondary gain attributes since this will influence the manner in which treatment needs to be directed so that they may best respond to it. It is often necessary to alter the behaviour of those in their immediate social network in order to diminish the rewards conferred by the assumption of the sick role; this is frequently not easy to do.

In many cases referred to a voice clinic the dysphonia has been present for a long time. This often leads to difficulty in determining the precise etiological factors in the dysphonias of psychological origin. Not only do patients tend to forget some of the important anxiety-laden events surrounding the onset of their dysphonia, but the natural psychological adaptive mechanisms lead to the resolution of these conflicts with the passage of time. If this referral pattern is typical of that to many speech-language pathologists and otolaryngologists, they will be faced with the difficult situation of not being able to elicit readily the hard facts indicative of psychological conflicts that will enable them to make a positive psychiatric diagnosis. At the time of the consultation, the patient will seem relatively free of psychological conflict while still burdened with the dysphonia, the latter being the residuum of the muscle misuse that arose during the earlier time of the acute psychological conflict. The habitual patterns of muscle misuse, irrespective of the type of pathogenesis of the symptom, persist even in situations where the discomfort of the psychological conflicts seem to have receded. This situation strongly suggests that voice disorders should be evaluated as soon as possible after their onset for one to be able to identify the psychological etiological factors. Providing therapy to assist the resolution of the psychopathological disorder that produced the symptom of dysphonia is obviously more readily done at this earlier time. It cannot be emphasized too strongly that, in the absence of organic structural change in the organs of phonation, the muscles of phonation are the final common path of all dysphonias of psychogenic origin and these dysphonias will certainly persist if the misuse is not remedied (Aronson, 1985; Barlow, 1973).

## *Therapeutic Approaches*

In the treatment of dysphonias of psychogenic origin the value of an interdisciplinary approach between the otolaryngologist, speech-language pathologist and psychiatrist is very substantial (Rammage, Nichol, Morrison, 1983). When both patient and psychiatrist know that each have seen a videotape of the patient's vocal cords showing the absence of organic pathology but the presence of muscle misuse characteristic of a psychogenic voice disorder such as muscular tension dysphonia or ventricular band dysphonia, they have the basis of a therapeutic alliance to address the underlying psychological problems. This is particularly true when the psychiatrist invariably works in the voice clinic as a member of the team; this tends to diminish the patient's reluctance to delve into the psychological factors that predisposed, precipitated or are perpetuating the dysphonia. Discussions among various professionals in the interest of the voice disordered patient facilitates consistency of information and reinforcement given to the patient, during treatment or therapy, and reduces the probability of recurrence of the problem. Frequent contact with the speech-language pathologist during voice therapy teaches the psychiatrist that symptom substitution is a very rare phenomenon. It also alerts him or her to a neglected field of observation in psychiatric practice; namely, that of abnormal posture and the misuse of the voluntary musculature as a significant intervening variable or as a final common path of psychological conflict.

Several patterns of voluntary muscle misuse are commonly associated with voice disorders in which associated psychogenic factors are also identified.

In inhibited individuals apprehension about the expression of aggression may be recognized in postural constraints. Assertive arm movements are effectively inhibited by scapulae adduction and elevation (Figure 2).

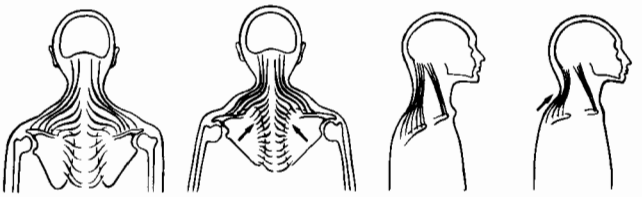

**Figure 2: Muscle misuse**
The scapulae (shoulder blades) are adducted and elevated. This reduces the effective use of assertive arm movements.

An inhibited or self-protective attitude may also be reflected in head retraction with associated extension or flexion of the neck (Figure 3). In addition, the chest may be held in inspiratory position or the abdominal muscles held tensely in an anxious individual resulting in incoordinate breathing. The patient who is recognized to be suppressing the perception of his or her impulse to cry or shout may clench the jaw; with the mandibular elevating muscles hypertonic, attempts at depressing the jaw to speak or sing may result in misuse of the TM joint with "jaw jut" or extension (Figure 4). This is often observed in relation to a retracted tongue position, and hypertonic suprahyoid muscles, which can be identified by palpation (Figure 5).

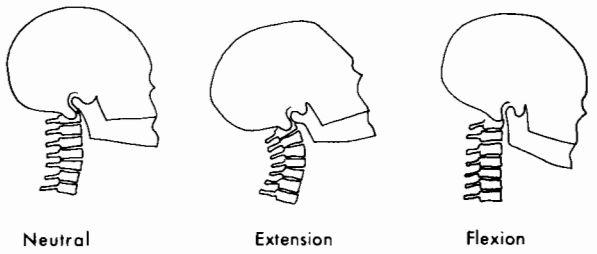


**Figure 3: Muscle misuse**
The neck may be hyperextended or flexed, reflecting an inhibitive or self-protective attitude.

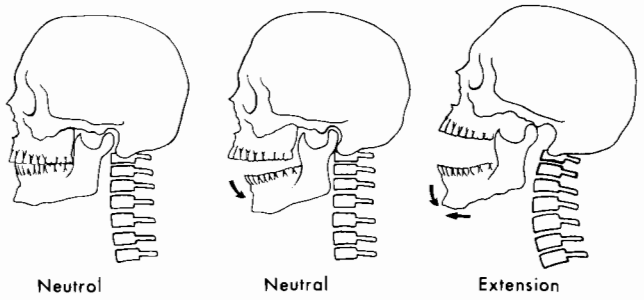


**Figure 4: Muscle misuse**
Misuse of the muscles governing movements at the temporomandibular joint results in reduced jaw movements and, commonly, extension of the jaw ("jaw jut") in association with mandibular depression. When seen in professional voice users, jaw jut tends to become increasingly apparent with rising pitch, often is associated with neck extension and head retraction and usually corresponds to hypertonicity in suprahyoid musculature.

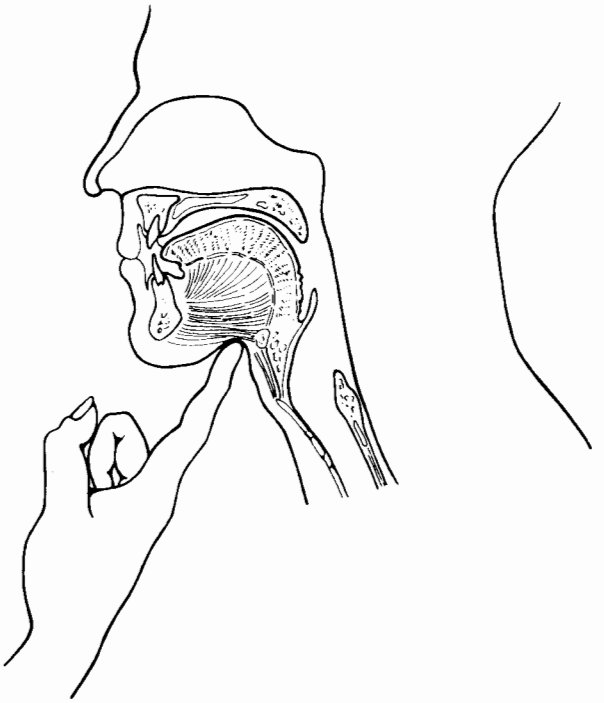

**Figure 5: Muscle misuse**
Hypertonicity of the suprahyoid muscles during phonation can be confirmed by palpation. This muscle misuse is commonly associated with poor tongue posturing — often the tongue is held in a retracted position. During singing, increasing suprahyoid muscle tension may contribute to larynx rise — a tendency for the entire larynx to be pulled superiorly with rising pitch.

In patients with a psychogenic dysphonia, extralaryngeal muscle misuses are characteristically associated with laryngoscopic manifestations of intrinsic laryngeal muscle misuses (Morrison, Rammage, Nichol, 1986). In professional voice users, anxiety (often about one's voice or career) is commonly manifested in hypertonic laryngeal and supralaryngeal muscle groups so that the characteristic signs of muscular tension dysphonia are observed: a functional posterior glottic chink — with or without mucosal changes such as vocal cord nodules (Figure 6); compensatory hyperadduction of the anterior one-third to one-half of the vocal cords; and a tendency for the larynx to rise significantly with pitch (Morrison et al, 1983). Also frequently seen is adducting activity in the ventricular bands — either in association with hyper- or hypoadducted true cords (Figure 7). This generally reflects some subconscious inhibition of verbal expression of a strong emotion. In some instances, characteristic of conversion reaction aphonia, the true cords are hypoadducted along their entire length during attempts at phonation, but adduct normally during spontaneous coughing (Figure 8).

Although no direct consistent correlations between psychiatric diagnoses and specific features of psychogenic dysphonias can be made, clearly certain important interrelationships between the various signs and symptoms of these dysphonias may be identified in an individual. The quality of care of voice disordered patients can therefore be elevated by a comprehensive evaluation of etiological factors that may be predisposing, precipitating or perpetuating voice disorders; use of a common terminology in reference to the various organic, functional, and psychological signs and symptoms of voice

disorders; and cooperation among the various professionals involved in voice care during rehabilitation.

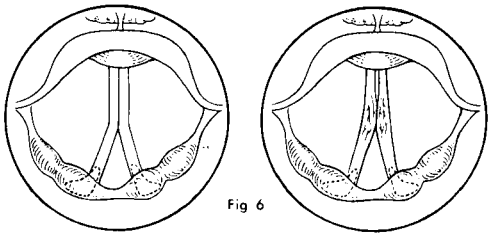

Fig 6

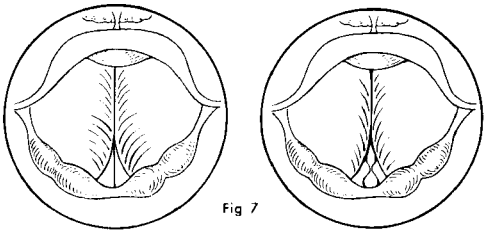

Fig 7

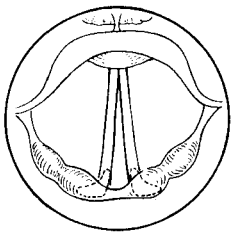

Fig 8

**Figures 6, 7, 8: Laryngoscopic features of muscle misuse during phonation**

Patients with psychogenic dysphonias usually exhibit laryngoscopic manifestations of intrinsic laryngeal muscle misuses. The most common laryngoscopic signs include: a posterior glottic chink (Figure 6) on phonation with either normal mucosa (Figure 6, left) or bilateral mucosal changes on the vocal cords, such as vocal nodules (Figure 6, right). A functional posterior glottic chink is the most salient laryngoscopic feature of muscular tension dysphonia, which is a tensional voice disorder; varying degrees of adduction of the ventricular folds of the larynx (Figure 7) with the true vocal cords either partially or entirely abducted (Figure 7, right), or firmly adducted (Figure 7, left). This sign frequently reflects some subconscious inhibition of verbal expression of a strong emotion; abduction of the true vocal cords from the anterior to posterior commissures during attempts at phonation is characteristic of many conversion aphonias (Figure 8).

## *References*


Aronson, A.E. (1985). *Clinical Voice Disorders — An Interdisciplinary Approach.* New York: Thieme Inc., 127-154.

Barlow, W. (1973). *The Alexander Principle.* London: Jutor Gollancy.

Belisle, G., Morrison, M.D. (1983). Anatomical correlation for muscle tension dysphonia. *Journal of Otolaryngology 12 (5),* 319-321.

Kendell, R.E. (1983). Hysteria. In: Russel, G.F.M., Hersov, R. (Eds.). *Handbook of Psychiatry, Vol. 4: The Neuroses and Personality Disorders.* Cambridge: Cambridge University Press.

Morrison, M.D., Rammage, L.A., Belisle, G.M., Nichol, H., Pullen, B. (1983). Muscular tension dysphonia. *Journal of Otolaryngology 12 (5),* 302-306.

Morrison, M.D., Rammage, L.A., Nichol, H. (1986). *A Management Guide to the Disordered Voice.* Self Instructional Package (SIPAC). Washington: American Academy of Otorhinolaryngology — Head and Neck Surgery Foundation Inc.

Rammage, L.A., Nichol, H., Morrison, M.D. (1983). The Voice Clinic — An Interdisciplinary Approach. *Journal of Otolaryngology 12 (5),* 315-318.

Tyhurst, J.S. (1978). Types of Pathogenesis. Personal communication in mimeographed material.