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Muscle Misuse Voice Disorders: Description and Classification*

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It is apparent that voice disorders frequently labelled "functional" are associated with laryngeal muscle misuse. This use of the word "functional" is, however, intrinsically ambiguous, and so we propose an alternative term based on descriptive features of dysfunction: "muscle misuse voice disorders". Persistent phonation with an abnormal laryngeal posture can lead to organic changes such as nodules or polyps, particularly in females with posterior glottic chink. We hypothesized that the chink was related to an overall increase in laryngeal muscle tension, and more directly due to inadequate relaxation of the posterior crico-arytenoid muscle during phonation. We employed the term "muscular tension dysphonia" (MTD) to note this condition, but it may be that the term "laryngeal isometric" is superior since there are other misuses of the larynx that obviously are manifestations of abnormalities of muscular tension. With this in mind we have evolved a new classification based on the laryngeal isometric, glottic and supraglottic lateral contraction states, antero-posterior contraction states, conversion aphonia, phychogenic bowing, and adolescent transitional dysphonia. *Key words: functional dysphonia, psychogenic voice disorders, muscular tension dysphonia, glottic posture.*

INTRODUCTION

Patients with so-called "functional dysphonia" who have structurally normal larynges and demonstrate muscle misuse in the larynx, and those with several interacting causes including habituated muscle tension, are probably better defined as having a "muscle misuse voice disorder".

Commonly occuring organic triggers of muscle misuse voice disorders are upper respiratory tract infections and gastro-esophageal reflux. The task of the voice team is a difficult one: to define and "sort out" pathological processes that are predisposing, precipitating and perpetuating an individual's dysphonia, and to apply terminology that best describes the relevant ongoing pathophysiological processes for each patient.

The clinician's diagnostic task may be further complicated by signs of organic change secondary to misuse. Vocal nodules are mucosal changes thought to be secondary to vocal abuse and misuse. Nodules often can be identified with a laryngeal mirror, and if assignment of the diagnosis is based solely on this readily identified clinical sign it seems logical to label the disease process "vocal nodules". However such an approach to classification focuses on organic pathology, often out of context with the individuals' habitual voice use patterns, and does not allow one to differentiate among the predisposing, precipitating and perpetuating factors involved in the etiology. The consequences for effective management are considerable.

A diagnostic classification scheme should be unifying, grouping disorders with common primary causes, yet flexible and expandable, so that individual factors playing etiological roles can be included in descriptions of single patients. The preferred treatment hierarchy may then be implied in a diagnosis. We have attempted to amalgamate a classification system that is comprehensive and promotes the use of diagnostic labels to best reflect all currently recognized patterns of muscle misuse associated with dysphonias. Our long-term goal is a data-based descriptive classification scheme that encompasses the most efficacious treatment practices.

Table I. *Classification of muscle misuse voice disorders.*

1. The laryngeal isometric
2. Lateral hyper-adduction states
a. Glottic
b. Supra-glottic
3. Supra-glottic anteroposterior contraction
4. Conversion aphonia
5. Psychogenic bowing
6. Adolescent transitional dysphonia

PROBLEMS OF CLASSIFICATION

Although clinical patterns described by different authors may bear resemblances, the assumed underlying pathological processes are often different. In addition, the use of terminology is inconsistent. The same term may be used to infer quite different pathological processes, for example, "plica ventricularis", "ventricular phonation", or "ventricular dysphonia" may designate a degree of adducting movement of the ventricular folds simultaneous with adduction of and phonation with the true vocal folds (1). However, these terms may be employed by others who refer to complete or near complete adduction of, and

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phonation with, the ventricular folds in the absence of true vocal fold phonation (2). Conversely, an apparently similar set of processes may be designated by very different terminology by different authors. "Puberphonia", "mutational dysphonia", "falsetto phonation" and "adolescent transitional voice disorder" may all refer to a functional dysphonia seen in teen-age males who are experiencing difficulty using modal register phonation during or following pubertal growth of the larynx and vocal tract.

Finally, the controversy continues as to the interchangeability of the terms "pychogenic" and "functional" when referring to dysphonias not primarily due to organic pathology. Aronson (2) argues for the use of "psychogenic" to refer to the broad group of voice disorders that exist in the absence of organic laryngeal pathology. His argument is based on the assumption that "psychogenic" is broadly synonymous with "functional" in its application to voice disorder classification, but that it more directly implies an inevitable underlying psycho-emotional desequilibrium. Brodnitz (3) in employing the term "psychogenic voice disturbances" has emphasized the importance of the interaction between organic and psychodynamic factors, which he believes belong together "like two sides of the coin", in the formation of all "functional" dysphonias.

Our bias is re-stated here: the terms "functional" and "psychogenic" are not synonymous: "psychogenic" diagnoses 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 (4). Such a conclusion is not reached solely by exclusion of organic pathogeneses, and often requires a formal psychological evaluation.

HISTORY OF CLASSIFICATION

Vocal dysfunction has been classified in many ways. A broad distinction was made formerly between "hyperfunctional" and "hypofunctional" voice disorders (5). Other authors have referred to a diagnostic polarity between "hyperkinesia" and "hypokinesia" (3).

Many authors have agreed that a hyperfunction or hyperkinesia of the laryngeal muscles results in a variety of dysphonia patterns. Further, the secondary organic signs of prolonged misuse of this nature have been highlighted: nodules; polyps; chronic laryngitis; contact ulcers and/or hypofunction of muscles (3, 5–8).

Attempts to classify hypofunctional or hypokinesic voice disorders have resulted in terms like "paretic hoarseness", "lateralis paralysis", "transversus triangle" and "bowing" of the vocal folds (9). While some authors referred to "hypofunctional dysphonia", Jackson (6) employed the term "myasthenia laryngis" to represent the secondary response to hyperfunction, a response he felt accounted for the problems of many professional voice users complaining of voice difficulty. Still other authors have used "phonasthenia" to describe a similar set of signs and symptoms, which were thought to be related to dysfunction of the thyroarytenoid muscles.

In more recent publications, the sub-classes "hyperfunctional" and "hypofunctional" seem to have yielded to a broader classification of musculoskeletal voice disorders (2, 8, 10, 11).

A MUSCULOSKELETAL APPROACH TO CLASSIFICATION OF DYSPHONIAS

Voice disorders can be caused by misuse of the voluntary muscles of phonation including muscles of the larynx, pharynx, jaw, tongue, neck and respiratory system. General postural misalignment is also common. Some dysphonias can be attributed primarily to incorrect vocal techniques such as poor coordination among respiratory, phonatory, resonatory and articulatory gestures; excessive or inadequate laryngeal valving; improper resonance focus; and improper control of pitch and loudness dynamics.

Some of the disorders associated with muscular misuses are not associated with observable organic changes to the vocal folds, while others commonly have associated secondary pathologies, including nodules, polypoidal degeneration, chronic laryngitis or scarring. In some individuals, muscle misuses may be the direct result of psychological stressors, in which case the resulting dysphonia can legitimately be labelled "psychogenic". Psychogenic problems represent failure of an individual to adapt physiologically to a psychological stimulus, usually a conflict based one (12–14). Whatmore & Kohli (15) have described a variety of maladaptive disturbance patterns ("dysponeses") in the signalling system for voluntary muscles which are responses to a variety of psychological states. The dysponetic signals originate in the pre-motor and motor cortices and influence action potentials in descending pathways, side branches, lower motor neurons, skeletal muscles and the various feedback pathways. Others, including Jacobson (16) and Barlow (17) have also focused on misuse of the voluntary muscles as the final common pathway for psychological conflict and dysfunction. The muscle misuse and misalignment patterns associated with symbolic conversion dysphonias or aphonias lead to severe dysfunction that is primarily psychogenic.

Psychological conflict or distress may be an interacting factor with vocal abuse and misuse in the precipitation and perpetuation of dysphonia, thus

contributing to pressor responses in the nervous system which in turn create hypertonicity of the voluntary muscles. Psychological distress may also be secondary to dysphonia, being the direct result of a dysfunctional state. The final result is the same: psychological stimuli lead to inappropriate muscle tone, disturbed feedback, and poor coordination of movements in the voluntary muscle system (14).

CLASSES OF MUSCLE MISUSE DYSPHONIA

Type 1: The laryngeal isometric. (Muscular tension dysphonia, (MTD) as described by Morrison et al. (8))

The laryngeal isometric pattern is most commonly seen in untrained occupational and professional voice users, and represents a generalized increase in muscular tension throughout the larynx and paralaryngeal areas. The etiology usually includes a combination of poor vocal technique, extensive and extraordinary voice use demands, and interacting or secondary psychological factors. Anxiety is most commonly identified, and in some cases, the diagnosis of generalized anxiety disorder is made based on criteria listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IIIR) (4). Psychological components may be secondary to dysphonia, rather than a primary etiological factor and this implies a perpetuating influence in sign-symptom formation.

A key feature of the isometric pattern of laryngeal and paralaryngeal hypertonicity relates to the characteristics and role of the posterior cricoarytenoid muscle (PCA) in abducting the glottis. The histological structure of the PCA appears to be well adapted to this role. For example it has more type I muscle fibres than all other intrinsic laryngeal muscles (18). Thus when the larynx is in a general hypertonic state, the sustained contraction of the PCA may deflect the arytenoid cartilages down the cricoarytenoid joint and open the posterior commissure, creating a posterior glottal chink (PGC). This hypothesis was supported by a laryngeal muscle pull experiment performed by the authors in 1983 (19). Dissected fresh human cadaver larynges were held in a frame with the intrinsic muscles attached to strings pulled in the direction of the muscle contraction. It was easily seen that when the glottis was closed with firm lateral cricoarytenoid and inter-arytenoid muscle pulls it took only a light traction on the PCA to open the posterior chink in such a way as to produce a glottal shape like that seen in this group of patients. PGC magnitude also may be associated with hypertonicity in supra-hyoid musculature (8).

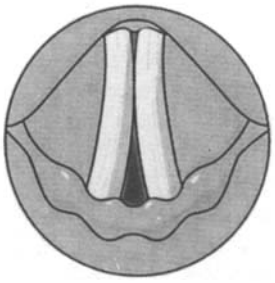

Fig. 1. The laryngeal isometric. Generalized tension in all the laryngeal muscles is often associated with an open posterior glottic chink due to persistent posterior crico-arytenoid muscle pull during phonation. This leads to secondary mucosal vocal fold changes including nodules, chronic laryngitis of polypoidal degeneration.

Studies of clinical populations and normal subjects simulating muscle misuse dysphonias have demonstrated that magnitude of the PGC is directly related to phonatory airflow rate, "whispery/breathy" perceptions, spectral noise, and distinctive intensity profiles in the acoustic spectra (8, 11, 20–22). In clinical populations, the laryngeal isometric may be accompanied by other forms of muscle misuse, such as lateral contraction states, which can confound aerodynamic and acoustic profiles.

Disease of the vocal fold mucosa is often identified as a component of the diagnosis, and generally assumed to be secondary to the specific pattern of muscle misuses associated with the laryngeal isometric posture. Hirano (23) has demonstrated the five-layered vocal fold structure in which the superficial two layers, including mucosa and superficial lamina propria (Reinke's space) make up the cover of the vocal fold and the deeper three layers, namely the middle elastic lamina propria, the deep collagenous lamina propria, and the vocalis muscle itself, make up the body of the vocal cord. The muscles contract during phonation, with the strength of the contraction dependent on various factors discussed above. During phonation, the cover flows around the leading edge of the vocal fold body, producing the so-called "mucosal wave" effect seen on stroboscopy. Looseness in the cover overlying the muscle body is necessary for clear phonation. In cases of tense or very loud phonation particularly in the presence of tense vocal folds, shearing stresses can injure the delicate tissue of the superficial lamina propria leading to edema, haemorrhage or fibrosis. These stresses tend to lead to the development of mid-membranous vocal nodules most commonly identified in premenopausal females and prepubertal children. These are also the clinical sub-groups that demonstrate the largest PGC magnitudes, and widest inter-arytenoid spaces relative to vocal fold length. The posterior margin of the nodules corresponds to the anterior margin of the PGC. This leads one to hypothesize a specific causal

relationship between PGC magnitude and bilateral nodules. Perhaps strong adduction forces employed to overcome exaggerated abduction in the posterior glottis lead to greater shearing stresses on the mid-membranous vocal folds at the position where nodules typically develop. Further, lack of adduction posterior to the mid-membraneous vocal folds would inhibit development of the mucosal disease there. Subepithelial edema or polypoidal degeneration is the usual form of secondary organic disease found in postmenopausal females, particularly in those who smoke. There is evidence that PGC magnitude and age in women are inversely related (21, 24). This could provide some explanation for the mucosal disease posterior to the typical nodule site in the older female population. Most adult males will develop a diffuse thickening of the cover that is referred to as "chronic laryngitis".

The laryngeal isometric is frequently associated with palpable increases in suprahyoid muscle tension on phonation particularly in higher pitch ranges during singing, and during high vowels and phoneme transitions in connected speech.

Type 2: Lateral contraction and/or hyperadduction

This dysfunctional pattern is a type of tension fatigue syndrome in which the larynx tends to be squeezed or hyperadducted in a side to side direction. It may exist either at the glottic or supraglottic level, or both. The glottic form is usually related to technical errors, and sometimes acute anxiety states may be identified. Supraglottic squeezing, or "plica ventricularis" on the other hand, is often associated with ongoing psychogenic factors.

Subtype a: Glottic contraction. Simple vocal misues with hyperadduction of the vocal folds produce a tense-sounding voice due to incorrect vocal technique. Phonation is probably associated with high laryngeal resistance forces, which explains why patients complain of "vocal fatigue" and discomfort at the end of a working day. In some situations the problem is triggered by an organic illness such as an upper respiratory infection, but persistent hoarseness remains many weeks after the viral illness has resolved. Koufman has used the term "habituated hoarseness" to note this relationship (10).

The lateral compression in the glottis is generally accompanied by incoordinate breathing such that the larynx functions more like a valve, controlling the rate of expiratory air flow. Proper breath control for speech entails the maintenance of a degree of inspiratory effort during exhalation so that a "push-pull" mechanism exists in the abdominal and thoracic areas to maintain steady flow of air in the trachea. In this situation, the larynx is not required to function as a flow regulator. When speech breathing is incoordinate there may be surges of uncontrolled expiratory air, which must then be valved by the glottis during phonation to allow for continued self-oscillation. An effortful and harsh voice results, with rapid fatigue. With ongoing compression, the voice pitch may drop, and vocal fry register may become prominent suggesting that there may be associated A-P constriction. The fatiguing voice may also be accompanied by general fatigue as well as discomfort or pain in the throat.

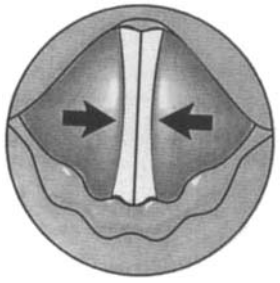

Fig. 2. Lateral hypercontraction at the glottic level. Frequently seen with generalized postural misuses and tension. May be triggered by an infection or chronic reflux.

Laryngoscopy with regular light will generally reveal normal looking structures, although erythema or diffuse thickening of the mucosa may be noted in addition to tight closure of the posterior glottis. Use of the stroboscope will show a prolonged closed phase, reduced vibratory amplitude and suppression of the mucosal wave. Ventricular fold adduction may be seen to a limited degree in association with the glottic level lateral contraction state. In this situation, it is important to differentiate between primary glottic level contraction and primary supra-glottic contraction because relegation of a patient to the supraglottic contraction category carries a much stronger inference of psychogenic etiology.

Subtype b: Supra-glottic adduction. This pattern tends to predominate in psychogenic dysphonia and can exist either with tightly adducted true vocal folds leading to a high-pitched squeaky voice, or with loose partially abducted vocal folds, in which case the voice is breathy or a tense whisper.

When the voice is strident or squeaky it may be difficult to identify whether the sound source is at the level of adducted ventricular folds, i.e. true false fold phonation, from obscured tightly adducted true folds below, or whether the true and false folds are functioning more as a valvular unit to create the voice. The latter two situations are likely more common since in most cases the ventricular bands do not adduct fully to the midline.

It has been our experience that the lateral supraglottic squeeze pattern seen on laryngoscopy in patients with a muscle misuse dysphonia is usually

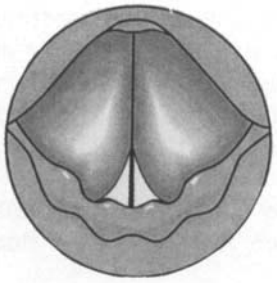

Fig. 3. Supra-glottic lateral contraction, or hyper-adduction of the false vocal folds. Tends to be strongly psychologically based.

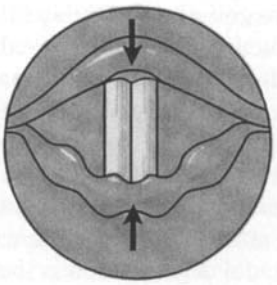

Fig. 4. Antero-posterior supra-glottic contraction. A common technical misuse seen in mild, moderate and severe forms.

associated with unresolved psychological conflict. Therapy approaches must combine correction of specific misuses with a careful evaluation and management of the psychological factors.

Type 3 – Anteroposterior contraction of the supraglottic larynx.

Koufman has presented a voice type labeled "Bogart-Bacall" syndrome (10) in which patients exhibit a tension-fatigue dysphonia with phonation at the very bottom of their vocal dynamic ranges. He describes a contraction pattern which results in reduced space between the epiglottis and the arytenoid prominances in the anteroposterior (AP) direction during phonation. Individuals using this posture complain of effortful voice and rapid fatigue when speaking at a low pitch but are able to talk more clearly and freely at a higher pitch.

This laryngoscopic pattern is not readily seen with mirror examination or with the rigid telescope because the tongue pull may extend the ary-epiglottic length. Transnasal examination during connected speech or song is the most effective way to demonstrate this misuse, which may then be subclassified as mild, moderate or severe.

Type 4. Conversion aphonia

The anxiety that leads to conversion hysteria has produced such mental pain that a physical symptom such as aphonia is much more bearable to the individual (4, 14). The type of psychological stressors and the resulting muscle misuse pattern differs from anxiety related tension misuses associated with type 2a described above. In conversion disorder the misuse may be beyond the awareness of the patient, hence the typical "la belle indifference" facial features. The vocal folds have full movement and can adduct normally for cough or other types of vegetative phonation such as laughter. But they stop short of sufficient adduction for voicing with an attempt to speak. Generalized hypertonicity can be identified in the larynx and when sound does come out it is usually high pitched, squeaky, or breathy.

Type 5. Psychogenic dysphonia with bowed vocal cords

In older patients, presbyphonia is associated with loss of muscular bulk and tone, as well as weakening and fragmentation of elastin and collagen fibres. This so-called "senile" atrophy is not necessarily the principal factor in patients who are seen to have the appearance of bowed vocal folds on indirect laryngoscopy. Occasionally, patients who appear to have a psychogenic functional dysphonia will present with a bowed glottis but may resume normal phonation and laryngoscopic appearance after voice and/or psychotherapy. This may also represent one of the forms of dysphonia in "habituated hoarseness" that follows an upper respiratory tract infection of other organic trigger.

Type 6. Adolescent transitional dysphonia

The normal adolescent voice change during puberty is often accompanied by pitch breaks, register breaks

Fig. 5. Less common forms of misuse. (left) Conversion aphonia presents a normal larynx in which the vocal folds are held away from the midline during phonation but function well for other duties such as cough. (middle) Psychogenically based dysphonia is not always associated with hyperadduction, and bowing may be seen even when senile atrophy or sulcus vocalis have been excluded. (right) The voice disorder associated with difficulties making the transition from child to adult male is usually a perpetuated falsetto, and the tension in the posterior glottis is accompanied by a larynx that is held highly and tightly in the neck.

and a degree of embarrassment. Psychological factors may lead to inhibition of the transitional event and establishment of perpetual falsetto phonation. Laryngoscopy reveals a tense glottis and the cartilaginous glottis may be hyperadducted, restricting phonation to the anterior membranous vocal folds. The larynx is generally drawn up tightly into the hyoid bone or base of the tongue. Downward traction on the thyroid cartilages usually results in modal register phonation at a pitch that is more representative of the adult male voice.

DISENTANGLING MULTIPLE ETIOLOGIES IN VOICE DISORDERS

Notwithstanding the principal cause of muscular tension in voice disorders, the severity of the symptoms may fluctuate with changes in physiological shift factors including vocal technique, variable psychological stressors, and altered muscle tone related to chronic gastro-esophageal reflux disease. Figure 6 illustrates this interrelationship. The line represents the operant laryngeal muscle tone without taking into account the changes produced by the shift factors, i.e. due to dystonia or similar processes. This hypothetical trace of muscular tension has many individuals lined up along the abscissa, a point close to which represents normal tone. Since most people are normal this line conceptually extends a great distance to the left. At the far right the muscle tone is very high as it would be in a person suffering from a focal dystonia, and this degree of hypertonicity would produce consistent spasmodic dysphonia. The pathway from the low tension normal level towards the right side likely follows a typical exponential curve.

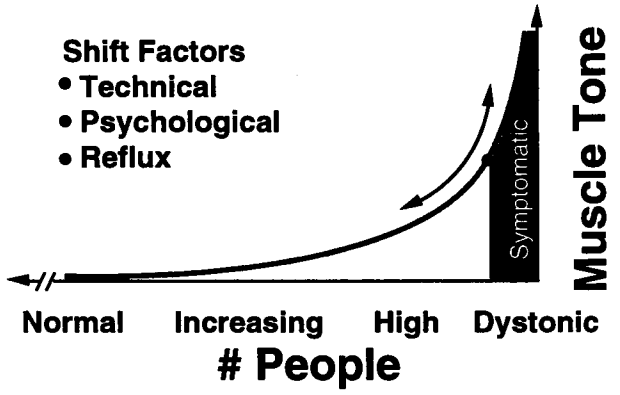

Fig. 6. Shift factors in dysphonia. After the baseline muscle tone, normal, dystonic or otherwise, has been set on the curve. The symptom severity can be shifted up or down by alterable factors including technique, psychological stressors, or reflux.

If a line is dropped from midway up the curve down to the abscissa we may assume that individuals on the right of this point will have muscular tension that will produce symptoms. But once a person's tension level has been set on the curve it can be shifted up or down (left or right) by a number of important alterable shift factors. If the central basal ganglia control and the laryngeal sensori-motor reflexes are relatively constant then the main clinically important shift factors are *i)* technical ability, *ii)* psychological tension, and *iii)* reflux. Each of these factors can be addressed in therapy, regardless of the classification category represented by a patients profile.

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