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PSYCHOGENIC DISORDERS OF VOICE — SOME PHYSIOLOGICAL CONSIDERATIONS*

Hajime Hirose

1. Introduction

Psychogenic dysphonia is not a common voice disorder but a very unique condition of laryngeal dyskinesia associated with loss or distortion of voice of psychogenic origin. Although no unanimous agreement has been reached, many authors tend to include functional (hysterical) aphonia or dysphonia, functional ventricular dysphonia and spastic dysphonia in the criteria of psychogenic dysphonia. We are of the opinion that functional disorders of pitch (fundamental frequency of voice or F_0) regulation should also be included.

During the 10 years from 1970 to 1979, 89 cases of psychogenic dysphonia were seen at the Voice and Speech Clinic of the University of Tokyo Hospital. The number corresponds to 4.2% of the total of 2,137 cases with voice disorders seen during the same period. Final diagnoses were made based on phoniatric examinations with the aid, in most cases, of psychiatric consultations. Age and sex distributions of these cases are shown in Table I.

In the present paper, general clinical pictures of each pathological group will be described and the results of some physiological studies of voice production mechanism in the spastic dysphonia group will be presented with special reference to possible therapeutic approaches.

2. General clinical pictures

i. Psychogenic aphonia

As shown in Table I, there were 22 cases of psychogenic aphonia in the present series. The age range of the 22 cases was wide (8-60 years) and incidence was higher in females.

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(1) Psychogenic aphonia (22 cases)

Age	Male	Female
- 10		1
11 - 20	4	1
21 - 30		4
31 - 40		2
41 - 50	1	6
51 - 60		2
61 -		1
	5	17

(2) Ventricular dysphonia (8 cases)

Age	Male	Female
- 10		
11 - 20		
21 - 30		
31 - 40	1	2
41 - 50		1
51 - 60	1	2
61 -		1
	2	6

(3) Spastic dysphonia (28 cases)

Age	Male	Female
- 10		
11 - 20		2
21 - 30	1	1
31 - 40	3	4
41 - 50	2	5
51 - 60	1	3
61 -	1	5
	8	20

(4) Others: including functional disorders of F_0 regulation (31 cases)

Age	Male	Female
- 10		
11 - 20		1
21 - 30		6
31 - 40	1	8
41 - 50	2	5
51 - 60	1	5
61 -	2	
	6	25

Table I. Age and sex distributions of clinical cases in the present series.

In typical cases, aphonic episodes occur suddenly, overnight for example, and the patient loses his or her voice completely. As a result, the patient is only able to phonate in whispers. Interestingly, however, sound production at the larynx for non-communicative purposes, e.g., coughing or laughing, usually remains unimpaired.

On laryngeal examination, no organic changes are observable. The mobility of the vocal folds is preserved but the glottal view during phonatory effort is quite variable. The vocal folds may show abduction rather than adduction at voice onset or they appear to close only at the very beginning of phonatory effort but reopen thereafter. Despite the fact that glottal closure is incomplete during phonation, the glottis shows complete closure

with audible sound for reflex activity in irritative cough. General signs of hysteric reaction may also be observed in other parts of the body.

In most cases, psychiatric consultation revealed a definite motivation for the development of the aphonic state associated with the basic character of the patient. As a direct trigger of an acute onset of aphonia, remarkable stress or psychological trauma was often noted. The psychological background is quite significant not only for diagnostic purposes but also for establishing therapeutic measures.

As for diagnosis, there is usually no difficulty in the diagnosis of psychogenic aphonia when organic laryngeal disorders such as laryngeal paralysis are ruled out on laryngeal examination. However, simple absence of organic changes is not necessarily conclusive enough for a final diagnosis of psychogenic origin. Careful history-taking and psychiatric evaluation are often mandatory. As mentioned above, a thorough inquiry of the clinical history of the patient is quite helpful for the relief of the aphonic condition.

In the past, many techniques were proposed to elicit reflex phonation for the relief of the aphonic condition. They were often called "surprise methods." However, it has been shown that it is not appropriate to remove only the surface symptom of aphonia; psychodynamic approaches are very necessary, although systematic vocal training or reeducation is helpful as a supportive measure.

Psychogenic aphonia has often been classified as a representative type of the hypofunctional or hypokinetic group of so-called functional dysphonia of psychogenic origin in previous reports (e.g. Brodnitz, 1969). In addition to typical aphonic cases, there is a considerable number of cases with a symptom of weak, asthenic voice without any comparable organic changes in the larynx. In addition to the symptom of dysphonia, most of these cases complain of discomfort in phonation: sore throat, sensation of irritation or dryness of the throat, etc. A term, "phonasthenia," has often been applied to these cases, but they may well be included in the group of hypofunctional dysphonia.

The clinical approach to these cases is essentially the same as that to psychogenic aphonia and systematic voice training, and

continuous encouragement for voice usage combined with psychiatric consultation is usually very effective.

ii. Ventricular dysphonia

Ventricular dysphonia, often called *dysphonia plicae ventricularis,* usually occurs as another type of hysterical reaction in which the communicative function of the larynx is hampered by more primitive biologic protective needs. Thus, venticular dysphonia can be considered a form of regression of function symptomatic of emotional disturbance. The overcontraction of the false vocal folds can occur as evidence of a simple compensatory effort of the subject to make up for the insufficient glottal closure. It is not always easy to distinguish which of the two levels is the causative, but the result in either case is ineffective voice production.

Laryngeal examination reveals adduction of the bilateral false vocal folds hiding a good glottal view. Voice quality may differ depending on whether the adducted false vocal folds are involved in active sound production, i.e., if the false vocal folds are vibrating as the sound source, the voice of the patient is rough, low-pitched and monotonous, whereas the voice will be strongly breathy when the adducted false vocal folds are not vibrating. In the latter case, the laryngeal condition is quite close to that of whispering.

Since ventricular dysphonia usually occurs as a type of hysterical reaction, the psychodynamic approach is the approach of choice, but simultaneous voice reeducation is often necessary. There have been attempts to use a special apparatus to mechanically open the adducted false vocal folds or, in extreme cases, to surgically excise the false vocal folds. For example, Kosoković, et al. (1977) used laryngomicroscopy to excise the hypertrophic ventricular folds in 35 patients with hypertrophy of the ventricular folds but in whom prolonged phoniatric treatment of normally functioning vocal folds had not resulted in regression of the hoarseness. With the patient under general endotracheal anesthesia, the hypertrophic ventricular folds were pulled with instruments toward the median line and excised somewhat more laterally from the median margin of the vocal fold and parallel to it. They reported that in 26 of the 35 patients restoration

of the normal voice was immediate, whereas 9 had poor results due to insufficient excision. These 9 underwent further microsurgical intervention with successful results. All patients received post-operative phoniatric therapy and inhaled antibiotics and cortisone for several days to protect the raw surfaces from infection and cicatricial alterations.

However, it has generally been claimed that the surgical approach does not seem to be appropriate for dealing with venticular dysphonia, unless there is a definite organic condition to elicit laryngeal closure at the level of the false vocal fold.

iii. Spastic dysphonia

Spastic dysphonia is a severe vocal disability in which a person usually speaks with a choked, strained quality of voice and a tightly constricted glottis. In this pathological condition, communication is markedly interfered and speaking requires great effort, although the patients are often able to produce normal phonatory sounds that do not have the character of direct communication. Almost all can laugh normally and a few can sing normally.

Spastic dysphonia has usually been thought of as one of the two extreme forms of the continuum of functional dysphonia representing the extreme of hyperfunction as the physical manifestation of a deep-rooted emotional conflict. This view is supported by the fact that many patients can talk naturally under the effect of Amytal. The presence of normal singing ability also suggests its emotional background. However, a considerable number of authors feel that at least some cases of spastic dysphonia are caused by neurological disorders. Robe, Brumlik and Moore (1960), for example, reported abnormal EEG patterns in their clinical cases. Some authors also claim that the neurological disorder is most likely in the extrapyramidal system.

Recently, Dedo (1976) reported that spastic dysphonia may be caused by a disturbance in the proprioceptive control of the vocal folds. Specifically, he proposed that neurotropic virus infection damaged the A and C fibers either peripherally or in the central nervous system but did not affect the large-diameter motor nerve fibers innervating the intrinsic laryngeal muscles. He and his colleagues further reported that electron-microscopic survey re-

vealed a consistent tendency toward demyelinization of the recurrent laryngeal nerve. This view was supported by Tucker (1979), although he was not conclusive as to whether the changes were primary or secondary. Tucker simply claimed that it was only evidence that the disease process could indeed be a pathophysiologic condition, not merely one of psychosomatic origin, and that there was consistent change occurring in these nerves.

DeSanto (1979), on the other hand, did not support the view of peripheral nerve pathology in the cases of spastic dysphonia based on his own histological studies. In his histogram of the control and the spastic dysphonic nerve, DeSanto did not find any differences in the numbers of large and small myelinated or unmyelinated nerve fibers. He claimed that clinical observation and the other disease models kept him skeptical of a peripheral nerve theory of spastic dysphonia.

Voice symptoms and local findings are very characteristic in spastic dysphonia. As briefly mentioned above, spastic dysphonia is characterized by a strained and quavering voice accompanied by intermittent aphonia. The normal flow and tempo of speech are interrupted and most patients attempt to obtain better sound by frequent clearing of the throat. In some cases, inspiratory phonation is also noted.

On laryngeal examination, a tendency toward strong glottal constriction often accompanied by abnormal adduction movement of the false vocal folds is observed. The false vocal folds may show tremorous adduction-abduction movements synchronously with voice quavering.

In most cases, clinical signs are so characteristic that the diagnosis of spastic dysphonia is usually made through clinical observations of voice symptoms. Strained voice quality may be noted in the cases of neurological disorders such as pseudobulbar palsy. However, these cases can be differentiated from spastic dysphonia by careful history-taking and neurological examinations.

More detailed physiological characteristics of spastic dysphonia and the recent trends in its treatment will be discussed in the following sections.

3. Pathophysiology of spastic dysphonia

i. Laryngeal observation by use of flexible fiberscope

As described above, examination of the larynx of cases with spastic dysphonia via indirect laryngoscopy very often reveals excessive constriction of the glottis usually associated with the intermittent adduction of the false vocal folds, although the larynx appears to be normal, at least structurally. It can be claimed, however, that conventional indirect laryngoscopy only provides a laryngeal view under limited conditions of fixed head position and protruded tongue, and hardly gives a view of natural laryngeal performance during running speech. We believe, in this connection, that more definitive information regarding structural movements during speech might be useful for better understanding of the pathological processes of spastic dysphonia.

In 1968, we designed a flexible fiberscope for laryngeal observation which allows direct visualization of the larynx during running speech (Sawashima and Hirose, 1968). With this technique, we are able to observe the laryngeal conditions during connected discourse with relatively little discomfort to the subject and with very little disturbance to the movements of the supraglottal articulatory organs during speech production. The instrumentation has been in wide use at present both in speech research and in clinical practice, and the application of this technique has also been attempted in the study of spastic dysphonia. One of the advantages of this technique is that this system can easily be combined with videotape recording and videoscreen observations in which the examiner and the patient are able to observe abnormal laryngeal behaviors and to record them when necessary.

Figure 1 illustrates the positioning of a flexible fiberscope inserted through the nasal cavity to the pharynx. The level of its tip is at the tip of the epiglottis. The fiberscope which is now in use (Olympus ENF-L) houses a straight viewing objective lens with a view angle of 75 degrees which gives an observation depth of 5 to 50 mm. The scope can be inserted through the nostril after application of a minimum amount of local anesthetics combined with nasal decongentant to the nasal cavity. Once the scope is inserted to an appropriate position, it can be connected to a cine- or videocamera.

Most characteristic findings in spastic dysphonia are spastic movements of the true vocal folds even during breathing and variable degrees of false vocal fold adduction during speech. Tendency

Fig. 1 Positioning of a flexible laryngofiberscope inserted through the nose. Metal Marking under the jaw indicates length of 25 mm. Arrow indicates the vocal fold.

toward tight glottal closure and false vocal folds adduction is most dominant during vowel production with relatively low pitch of voice. In many cases, an attempt to produce a sustained vowel appears to be most difficult due to spastic glottal constriction. On the other hand, use of a breathy voice or a voice with higher voice pitch usually results in less tight or apparently normal closure of the glottis with lateralization of the false vocal folds resulting in better voice quality. These findings are comparable to those reported by Parnes, Lavorato and Myers (1978).

A close observation of initiation of voicing also reveals that there is inappropriate glottal closure well before the real voice onset, which may suggest a disruption of coordinated timing of laryngeal and respiratory functions. A similar tendency has been observed in the larynx of stutterers (Yoshioka and Löfqvist, 1979), and this type of discoordination between the two levels of phonation and respiration may be an important physiological basis in spastic dysphonia as in stuttering.

Although Aronson et al. (1968) suggested that there is a specific type of spastic dysphonia in which sudden abductory movements of the glottis is characteristic, we have not been able to detect such movements in our cases. In comparison to ventricular dysphonia, the adduction of the false vocal folds appears to be more or less momentary but tighter in spastic dysphonia, particularly in low pitched voice production.

Brodnitz (1976) pointed out that it may be difficult to establish a definite diagnosis of spastic dysphonia based on overt

visual symptoms alone. As suggested by Parnes, Lavorato and Myers (1978), however, we are of the opinion that the difficulty might be overcome by obtaining detailed visual information of laryngeal behavior particularly by combining other physiological studies including EMG as described below. The validity of application of fiberoptic laryngoscopy in diagnosis and description of spastic dysphonia should be appreciated.

ii. Laryngeal electromyography in spastic dysphonia

Electromyography (EMG) is a technique for providing graphic information about the time course of the electrical activity of the muscle fibers which accompanies muscle contraction and subsequent effects such as tension development. Since Faarborg-Anderson reported his extensive study on human laryngeal EMG in 1957, a good number of reports on both clinical and research applications have been published.

Clinical EMG was developed initially in the fields of neurology and physical medicine because of the need for improved diagnostic and prognostic methods. In particular, a major application of EMG has been in the differential diagnosis between neurogenic and myogenic causes of muscular weakness. In the field of laryngology, the clinical use of EMG is aimed at the following: (1) the differential diagnosis of immobile vocal folds, (2) the prognostic evaluation of laryngeal paralysis, (3) the diagnosis of laryngeal involvement in motor neuron diseases, and (4) the examination of abnormal laryngeal kinesiology. The application of EMG to the study of spastic dysphonia is aimed at (4) of the above, i.e. the study of abnormal laryngeal kinesiology (Hirose, 1979).

A percutaneous approach using a bipolar concentric needle electrode has been used for laryngeal EMG in our clinic and laboratory, in which the two innervation territories, i.e. the cricothyroid and the thyroarytenoid, are examined bilaterally.

The percutaneous insertions are preceded by the intradermal administration of a small amount of 0.5% Xylocaine solution through a Panjet at the site of needle insertion.

The directions of needle insertion are schematically shown in Figure 2. To reach the thyroarytenoid, the needle is passed through the cricothyroid space near the midline and advanced cranially and slightly laterally in the submucous tissues near the

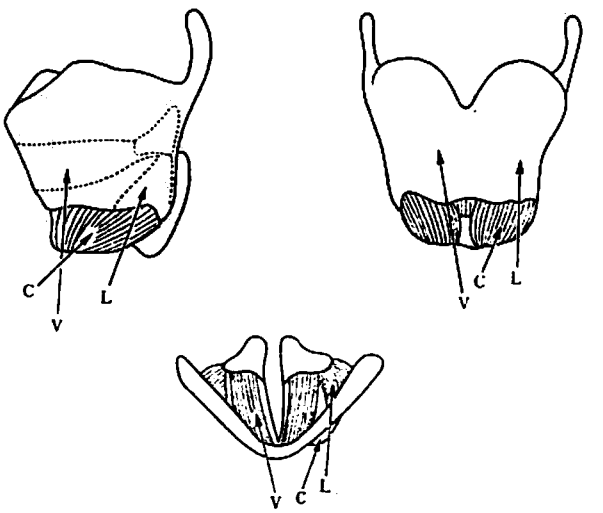

Fig. 2 Directions of insertion of the needle electrode into the thyroarytenoid (V), cricothyroid (C) and lateral cricoarytenoid (L).

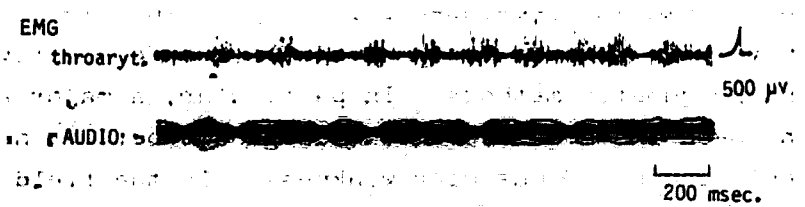

Fig. 3 Raw EMG trace of the thyroarytenoid in a case of spastic dysphonia recorded during sustained vowel phonation.

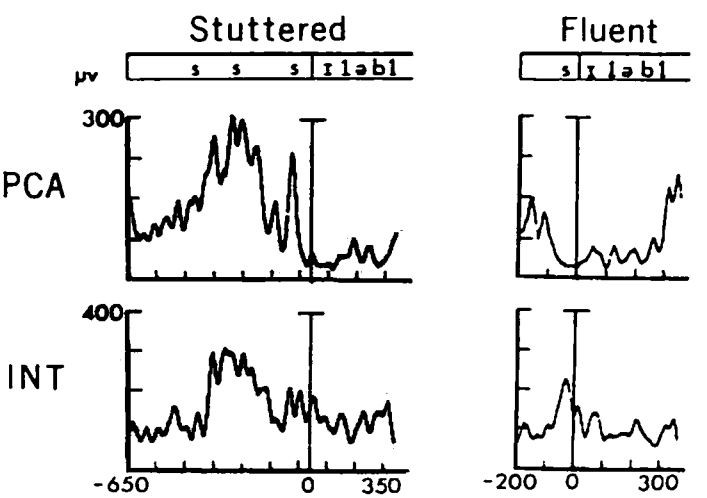

Fig. 4 Integrated EMG patterns of the posterior cricoarytenoid (PCA) and the interarytenoid (INT) in both stuttered and fluent conditions (From Freeman and Ushijima, 1976).

anterior commisure. To avoid bringing the needle too close to the mucosal surface, the subject is asked to phonate during insertion so as to bring the vocal folds to the midline. For the cricothyroid, insertion is made at the level of the lower edge of the cricoid ring and 5 mm lateral to the midline. The needle is advanced postero-laterally and slightly upward aiming toward the inferior tuberculum of the thyroid cartilage. Penetration of the fascia is often felt when the tip of the needle reaches the muscle.

Pathological kinesiology in spastic dysphonia can be well documented in EMG terms. Figure 3 exemplifies the EMG pattern of the thyroarytenoid muscle obtained in a typical case of spastic dysphonia. The pattern of grouping voltage in the adductors seems compatible with the glottal behavior of intermittent overconstriction and can be taken as a peripheral manifestation of abnormal laryngeal control in this disorder. As will be discussed below, EMG can also be used for evaluation of the effect of surgical treatment of spastic dysphonia.

EMG examination of the abductor laryngeal muscle in spastic dysphonia has not been performed to date. However, EMG study of stuttering (Freeman and Ushijima, 1976) revealed evidence of abnormal laryngeal kinesiology regarding discoordination of adduction-abduction function of the laryngeal muscles in stuttering. Freeman and Ushijima noted the following as characteristic of stuttering larynx: (1) a high level of muscle activity, and (2) a disruption of adductor-abductor reciprocity which is important for normal voicing. Figure 4 clearly illustrates these two points. From a kinesiological viewpoint, the latter finding is particularly interesting, and this pattern of disruption of coordination can also be found in spastic dysphonia, since the two diseases have clinically similar patterns, particularly in the initiation of voiced sounds. Further studies are needed to explore this point in spastic dysphonia.

4. Recent trends in the treatment of spastic dysphonia and their physiological basis

There is unanimous agreement that all existing forms of treatment for spastic dysphonia give poor results. The patient often secludes himself from social relationships with others and resists therapeutic approaches such as psychotherapy and voice training.

Recently, Dedo (1976) proposed that deliberate section of the unilateral recurrent laryngeal nerve would relieve the condition of the overconstriction of the glottis thus improving the vocal quality of patients with spastic dysphonia. He has performed this nerve section surgery on more than 200 cases and claims that overall results are quite promising. Dedo also found that approximately 25% of the patients who underwent this type of surgery needed some speech therapy postoperatively because they developed too breathy voice due to an open glottis.

Similar attempts have been made by other authors, and Som (1976), for example, crushed the recurrent nerve (RLN) and obtained satisfactory results as well.

It is conceivable that the sacrifice of the hemilaryngeal function by cutting or crushing the unilateral RLN may promise improvement in spasticity but at the same time it can produce iatrogenic laryngeal problems including breathy hoarseness and weakened power of vocal energy as well as intermittent aspiration.

In addition to these surgical side effects, it is also conceivable that with the unilaterally functioning larynx the vocal fold may tend to overadduct postoperatively beyond the midline to meet the paralyzed fold on phonation, thus causing reappearance of spastic speech mode in the long run.

Iwamura and Hirose recently reported that selected section of the thyroarytenoid muscle branch (TA branch) of the recurrent nerve could provide relief for the strained quality of voice in spastic dysphonia (Iwamura and Hirose, 1979). Since the remaining recurrent nerve branches innervating the lateral cricoarytenoid and the interarytenoid are kept intact, the adduction function of the severed side is maintained although it becomes somewhat weakened. As a result, the degree of postoperative disability seems less than the above mentioned techniques of total elimination of the unilateral recurrent nerve function. In the following, basic notions of and clinical experience with selected section of TA branch will be presented.

i. Rationale for selective division of a thyroarytenoid (TA) branch of the RLN

Experienced laryngologists and speech pathologists have often noted that whenever a patient with spastic dysphonia raises his or her speaking pitch, the spastic pattern tends to decrease or even

disappear as long as the patient maintains higher pitches while talking. This characteristic temporary disappearance of spasticity is also present in laughing, singing and humming which patients do with elevation of voice pitch. The characteristic phenomenon of spontaneous disappearance of spasticity can be explained by laryngeal muscle function. In normal phonation, the vocal cords adduct due to activation of the adductor muscles and relaxation of the abductor muscles. At the same time, the cricothyroid muscle (CT) also becomes active to give appropriate tension to the vocal fold.

It has been found that the CT is the primary pitch raiser of voice and is antagonistic to the thyroarytenoid muscle in respect to its effect on the length and thickness of the vocal cord (Hirano, Vennard and Ohala, 1970). When the CT contracts for higher pitches the vocal fold tends to move from the midline to the paramedian position (Arnold, 1961; Van den Berg and Tan, 1959). It is reasonable to assume that the effort of raising the pitch of voice is accomplished by increasing CT activity resulting at the same time in lesser degree of vocal fold adduction.

In spastic dysphonia, overconstriction of the glottis exists secondary to abnormal activation of the adductors, while in higher pitch phonation activation of CT can overcome adduction forces and normal muscle balance between CT and adductors, TA in particular, is obtained.

The selective section of TA branch may enhance this mechanism of "muscle balance" in addition to causing substantial decrease in the force of adduction due to TA inactivation. From this consideration, postoperative voice therapy is mandatory.

ii. Experimental studies

To examine the effect of sectioning selected branches of the RLN, animal experimentation was undertaken in two different programs; (1) selective division of the unilateral TA branch of the RLN in 8 dogs and (2) selective division of the unilateral LCA branch of the RLN in 4 dogs. Two to four month follow-up postoperatively revealed that unilateral selective TA branch section provided apparently normal voice quality, while unilateral selective LCA branch section produced incomplete closure of the glottis with resultant breathy hoarseness. These experimental results suggest the possibility of clinical application of unilateral

selective TA division for patients with spastic dysphonia, because this particular surgical technique can provide not only weakened adduction on phonation but also full appreciation of basic laryngeal functions.

iii. Procaine block of the RLN for selection of cases

One percent solution of Procaine was injected slowly into the lower neck where the RLN runs between the trachea and the esophagus. A needle must be in place outside the thyroid gland to avoid trauma which may eventually develop into hematoma. Approximately 5 to 10 ml of one percent solution of Procaine can successfully block the RLN on the injected side with significant reduction of spastic mode of voice sound and relatively breathy hoarseness for a while. Marked reduction in vocal fold mobility on the injected side is evident on mirror examination. The choice of a candidate with spastic dysphonia for selective division of a TA branch of the RLN on one side can be based on clinical confirmation of the significant effect of Procaine on decrease in degree of vocal spasticity.

EMG examination can reveal the change in spastic pattern after Procaine block. Figure 5-a illustrates that both TA muscles show a pattern of grouping voltage which is indicative of spasticity during phonation before Procaine block. As can be seen in the audioenvelop, phonation is interrupted due to spasticity. After Procaine block of the right RLN, it is obvious that spastic pattern is absent even in the EMG trace of the left TA, and the pattern of vowel phonation also appears to be very stable (Figure 5-b).

iv. Surgical techniques

An oblique incision of approximately 4 cm is made along the lateral margin of the thyroid ala on the right side under local anesthesia with or without neuroleptanalgesia. The lateral parts of the sternohyoid muscle and the thyroid gland can be visualized and retracted medially to expose the thyroid ala. Sharp detachment of thyropharyngeal muscle from the thyroid ala is done to prepare to elevate the posterior edge of the thyroid cartilage anteriorly. With elevation of the posterior edge of the thyroid ala by two prongs of a double hook, attention should be directed to the muscular process of the arytenoid cartilage. Identification of the muscular process can be made quite easily by close

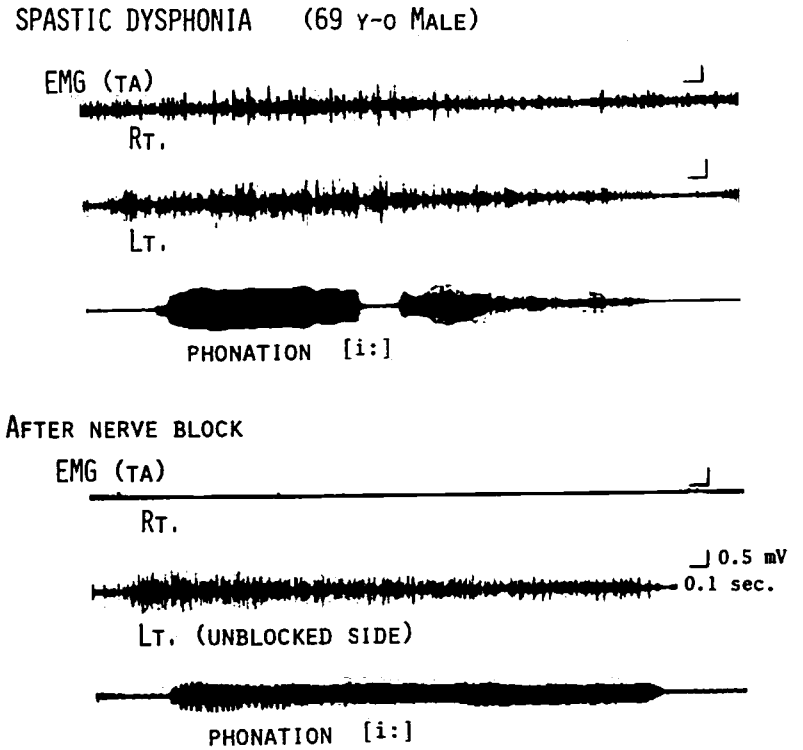

Fig. 5-a Raw EMG trace of the thyroarytenoid muscles in a case of spastic dysphonia before Procaine block.
5-b Some recordings after Procaine block.
(Cal. 5 mV for Rt. and 2 mV for Lt.)

observation of its alternative upward and downward movements in correspondence with phonatory and inspiratory phases. Just below the muscular process there is a thin nerve running horizontally to innervate solely the TA muscle. Meticulous separation of this portion of the nerve from the underlying structure is made to allow the placement of a pair of stimulation electrodes. Synchronous contraction of the TA muscle is visible with electrical stimuli on this particular nerve, and it can be distinguished whether it is a motor or a sensory nerve (Galen's anastomosis).

The nerve is then tied at two different sites and cut at the mid point. Electrical coagulation is applied to the central cut end of the TA branch to prevent spontaneous regeneration.

Immediately after completion of the selective division of the TA branch, the patient is asked to phonate and, in successful cases, one can immediately note marked decrease in spasticity.

v. Postoperative vocal rehabilitation

Postoperative vocal rehabilitation is mandatory. Rehabilitation must be directed towards raising speaking pitch, which should be effective for preventing overconstriction of the glottis. Falsetto phonation may be encouraged at the beginning and, on accommodate themselves to speaking with slightly high pitch sounds including humming, addressing, short sentences, etc.

vi. Results

1) So far, selective division of a TA branch of the RLN on the right side has been performed in four cases with spastic dysphonia. All four have shown significant improvement in communicative speech. These cases have been followed up one to six months postoperatively.

2) EMG recording obtained preoperatively from both CT and TA muscles revealed spasmodic contraction synchronously with spastic vocalization. Postoperative EMG traces, however, demonstrated electrical silence of the TA on the operated side with a decreased tendency in spasmodic pattern of bilateral CT as well as TA on the non-operated side.

vii. Comment

Dedo's paper in 1976 reporting successful results of simple section of the unilateral RLN in the neck for spastic dysphonia encouraged us to develop a more specific surgical technique, i.e., selective division of a TA branch of the RLN. This procedure not only results in significant improvement in vocal spasticity but also preserves the primary laryngeal functions in terms of adduction and abduction of bilateral vocal cords postoperatively.

In this regard, selective division of a TA branch of the RLN may be preferable to simple section of the unilateral RLN. Another point we would like to strongly emphasize is that postoperative vocal rehabilitation is very important to maintain the improvement in spastic speech pattern; continuous training to raise the speaking pitch is essential. For this purpose both laryngologists and speech pathologists should conduct continuous postoperative follow-up of patients over a long period of time. We continue to interview patient once very other week in our hospital.

As stated above, the goal of the present technique is to provide not only decrease in adduction force but also relative ease in achieving a well-balanced pattern of muscle activation in phonation. Since the apparent adduction-abduction function of both vocal folds is essentially preserved even postoperatively, recurrence of spasticity is a possibility. To avoid this sequela and for complete success, the importance of postoperative voice therapy cannot be underestimated.

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