The Cricothyroid Muscle Does Not Influence Vocal Fold Position in Laryngeal Paralysis

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The status of the cricothyroid muscle, which is innervated by the superior laryngeal nerve, is believed to influence the vocal fold position in laryngeal paralysis. It is believed that isolated lesions of the recurrent laryngeal nerve generally result in the paralyzed vocal fold assuming a paramedian position but that with lesions of both the superior and recurrent laryngeal nerves, a more lateral (intermediate or cadaveric) vocal fold position can be expected.

Twenty-six consecutive patients with unilateral vocal fold paralysis underwent transnasal fiberoptic laryngoscopy (TFL) and laryngeal electromyography (LEMG). By TFL, the vocal fold positions were paramedian in 8 patients, intermediate in 7, and lateral in 11. By LEMG, 13 patients had isolated recurrent laryngeal nerve lesions and 13 patients had combined (superior and recurrent laryngeal nerve) lesions.

There was no correlation between the vocal fold position and the status of the cricothyroid muscle, *i.e.*, the status of the cricothyroid muscle by LEMG did not predict the vocal fold position nor did the vocal fold position by TFL predict the site of lesion. In addition, we investigated the possibility that the degree of thyroarytenoid muscle recruitment (tone) might correlate with vocal fold position, but no relation was found. We conclude that *1.* the cricothyroid muscle does not predictably influence the position of the vocal fold in unilateral paralysis; *2.* thyroarytenoid muscle recruitment (tone) does not appear to influence vocal fold position; and *3.* still unidentified and unknown factors may be responsible for determining vocal fold position in laryngeal paralysis.

INTRODUCTION

The status of the cricothyroid (CT) muscle, which is innervated by the superior laryngeal nerve, is believed to influence the vocal fold position in recurrent laryngeal nerve paralysis.[1–4] The belief is that isolated lesions of the recurrent nerve generally result in the paralyzed vocal fold assuming a paramedian position; combined lesions of both the superior and recurrent laryngeal nerves are expected to be associated with lateralization of the affected vocal fold. Thus, historically, vocal fold position has been considered by clinicians to reflect the neural site of lesion.[4]

Recent experimental and clinical reports have contradicted that assumption.[5–7] In addition, laryngeal electromyographic (LEMG) data indicate that at least partial reinnervation of the affected laryngeal muscles follow paralysis.[5–11] Because LEMG provides reliable site-of-lesion information, we used it to reevaluate the relation between lesion location and vocal fold position.

MATERIALS AND METHODS

A retrospective review of the medical records of 26 consecutive patients with previously untreated unilateral vocal fold paralysis was carried out; all were initially seen at our Voice Center in 1993. Half of the patients were men and half were women. Eighteen (70%) of the 26 patients had paralysis of the left vocal fold and 8 (30%) had paralysis of the right vocal fold. Twelve (46%) of the patients had had vocal fold paralysis for less than 1 year, and 14 (54%) for 1 or more years.

Ten (38%) of the 26 cases were associated with an iatrogenic cause; among them, 2 were paralyzed after endotracheal intubation and 1 after resection of a vagal schwannoma. Eight (31%) had a neoplastic cause for the paralysis, of which 5 had lung carcinoma, 2 had esophageal carcinoma, and 1 had a skull-base meningioma. In 1 of the remaining 8 patients, herpes zoster infection was the cause of the paralysis, and in the other 7 (27% of the entire group), the cause was considered idiopathic.

Each patient was evaluated by transnasal fiberoptic laryngoscopy (TFL) and LEMG. Diagnostic studies were interpreted independently, *i.e.*, the vocal fold position by a laryngologist (J.A.K.) and the LEMG findings by a neurologist (F.O.W.). The positions of the paralyzed vocal fold were classified as follows:

Paramedian was defined as a glottic chink of less than 1.5 mm with good vocal process–to–vocal process contact, *i.e.*, the posterior commissure was not open.

Intermediate was defined as a glottic chink of 1.5 to 2.5

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mm with the posterior commissure open or closed.

Lateral (lateralized or cadaveric) was defined as a glottic chink of more than 2.5 mm with the posterior commissure open.

Technique and Interpretation of LEMG

LEMG for patients with unilateral vocal fold paralysis was performed with a standard protocol carried out by both the otolaryngologist and the neurologist. The otolaryngologist inserted the needle electrodes into the larynx, and the neurologist operated the EMG equipment and interpreted the findings. We routinely used a Nicolet Viking Electromyograph (Nicolet Biomedical Inc., Madison, Wis.) and disposable monopolar needle electrodes (cat. no. 902-DMF37, Teca Corp., Pleasantville, N.Y.). We recorded two simultaneous channels of signals: the first channel was EMG (low-frequency filter setting = 20 Hz, high-frequency filter = 10,000 Hz); the second channel was a voice recording made with an accelerometer (Rochester Electromedical Inc., Tampa, Fla.) taped in place over the skin of the neck in the submandibular triangle (low-frequency filter = 200 Hz, high-frequency filter = 3000 Hz).

For assessment of needle placement during electrode insertion, both channels were run at slow sweep speeds (500 ms/cm), and gain was usually kept at 500 µV-1 Mv/cm. These speeds allow the best visual assessments of the relations of EMG and voice maneuvers. Audio features of the signal, specifically the presence of high-frequency or crisp motor unit potential (MUP) discharges, can be used to ensure the localization of the needle to the target muscle during appropriate vocal maneuvers.

MUP morphology is analyzed at sweep speeds of 10 ms/cm. At this sweep, individual motor units are isolated to permit analysis of their complexity, duration, and amplitude. Gain settings for MUP analysis are usually 500 µV-1 Mv/cm. Laryngeal MUPs are typically shorter in duration and smaller in amplitude than those of most other muscles. Due to the poor muscle relaxation in the larynx, difficulties in eradicating distant motor unit noise, and the small size of laryngeal MUPs, longer sweep speeds and higher gains sometimes help to screen out unwanted noise.

Signs of insertional activity (fibrillations) were searched for last. They usually can be recognized by their characteristic auditory features, so identification commonly occurs even when slow sweep speeds are used. These wave forms can be specifically analyzed at faster sweep speeds (10 ms/cm) and low gain settings (100 µ/cm), if indicated, particularly if hard copy prints are desired. Other discharges, fasciculations, myokymia, or complex repetitive discharges also may be identified readily. Fibrillation potentials were graded as follows: 1+ = only 1 or 2 fibrillation potentials in an entire muscle; 2+ = at least 3 distinct fibrillation potentials (positive sharp waves or fibrillation negative spikes) that last for at least 3 seconds each; 3+ = sustained fibrillation potentials on every needle insertion; 4+ = fibrillations so dense as to obscure the EMG baseline.

LEMG was performed with the patient in the supine position with the neck slightly extended and no local or topical anesthesia. The sequence of muscles tested was *1.* the CT muscle on the clinically uninvolved side; *2.* the CT muscle on the paralyzed side; *3.* the thyroarytenoid (TA) muscle on the clinically uninvolved side; and *4.* the TA muscle on the paralyzed side.

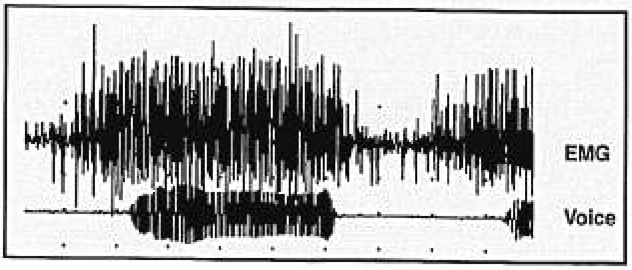

Fig. 1. Normal laryngeal EMG tracing of the TA muscle (4+ recruitment). The upper tracing (EMG) shows many MUPs; the lower tracing (voice) shows the voice recording (500 ms/div; top tracing, 500 µV/div; bottom tracing, 2 µV/div).

The clinically uninvolved CT muscle was identified in the CT membrane by advancing the needle electrode through the muscular layer (approximately 1 cm lateral to the midline) and then withdrawing the needle electrode until crisp action potentials were elicited during sustained, high-pitched phonation of the vowel /i/. The patient was asked to flex the neck during the examination to be certain that the needle electrode was not in one of the strap muscles. After examination of the normal side was completed, the affected (paralyzed) side was examined in a similar fashion.

The TA muscles were also examined by a percutaneous technique through the CT membrane; an attempt was made to keep the electrode beneath the mucosa (submucosal). The TA muscle is located at a level halfway between the CT membrane and the thyroid notch; the needle electrode is angled laterally, approximately 1 cm medial to the posterior edge of the ipsilateral thyroid lamina. (The laryngeal anatomy is variable, and use of fixed angles for distances to find the TA muscles is not recommended. The technique outlined was derived from experience doing medialization laryngoplasty. The operator must visualize the internal anatomy of the larynx as it relates to the external framework.) As with the CT muscles, the clinically normal side was done first. Multiple areas of each muscle were sampled during both phonation of a sustained vowel /i/ and with sniffing maneuvers (to look for synkinesis).

To quantify recruitment and examine wave form morphology, the EMG of each muscle was examined by means of several different sweeps—we routinely use 500-ms/cm, 200-ms/cm, 50-ms/cm, and 10-ms/cm sweeps. The two slower sweeps (500 ms/cm and 200 ms/cm) are used so that recruitment of MUPs can be quantified and to establish the correlation of EMG with phonation as recorded by the accelerometer. The two faster sweeps (at 50 ms/cm and 10 ms/cm) are generally used to examine wave form morphology and to search for spontaneous activity.

In EMG, the term *recruitment* is defined as "the orderly activation of the same and new motor units with increasing strength of voluntary muscle contraction."[12] In LEMG, recruitment describes the relative number of MUPs during graded activation of the vocal cords. Normally, multiple different MUPs fire during maximal activation; however, this number is reduced in patients with loss of nerve fibers. Recruitment analysis, then, provides real-time information regarding the degree of innervation of laryngeal muscles. Figure 1 shows an example of full recruitment in the TA muscle (upper tracing) and the voice recording (lower tracing), and

TABLE I.
Patient Information, Vocal Fold Position, and Electromyographic Data.

Series	Patient	Age	Sex	Side	Duration of Paralysis (Years)	Etiology of Paralysis	Vocal Fold Position*	CT Muscle Status†	TA Muscle Status‡
1	CP	66	M	L	2.0	Idiopathic	Lat	N	3+
2	WS	44	F	R	0.5	Idiopathic	Lat	N	1+
3	VF	50	F	L	0.5	Idiopathic	Para	N	1+
4	RT	33	M	R	5.0	Idiopathic	Int	N	0
5	CM	84	M	R	0.5	Neoplastic (ca esophagus)	Lat	N	0
6	MW	66	F	L	2.0	Neoplastic (ca lung)	Int	N	3+
7	EY	73	F	L	0.5	Neoplastic (ca lung)	Int	N	2+
8	JO	63	M	R	1.0	Neoplastic (ca esophagus)	Int	N	3+
9	WH	72	M	L	0.5	Neoplastic (ca lung)	Para	N	0
10	GH	49	M	L	1.0	Iatrogenic (s/p thyroidectomy)	Para	N	3+
11	SL	16	F	L	2.5	Iatrogenic (s/p intubation)	Lat	N	3+
12	ML	31	M	L	1.0	Iatrogenic (s/p thoracotomy)	Lat	N	2+
13	LC	68	F	L	0.5	Iatrogenic (s/p thyroidectomy)	Para	N	2+
14	JC	47	F	L	2.0	Idiopathic	Int	2+	1+
15	LH	61	F	L	0.5	Idiopathic	Lat	2+	3+
16	AS	43	M	L	2.0	Idiopathic	Lat	3+	2+
17	JR	33	M	L	0.1	Iatrogenic (s/p vagal tumor)	Int	0	0
18	DP	65	M	R	1.0	Neoplastic (ca lung)	Para	3+	2+
19	EM	67	M	L	0.2	Neoplastic (ca lung)	Para	2+	0
20	VW	37	F	R	0.5	Neoplastic (meningioma)	Para	2+	1+
21	ES	56	M	L	0.4	Iatrogenic (s/p thoracotomy)	Lat	3+	0
22	MB	61	F	R	0.5	Iatrogenic (s/p thyroidectomy)	Para	2+	1+
23	VB	62	F	L	32.0	Iatrogenic (s/p thyroidectomy)	Int	2+	2+
24	DS	35	F	R	1.0	Iatrogenic (s/p cervical disk)	Lat	2+	1+
25	CB	67	M	L	4.0	Iatrogenic (s/p intubation)	Lat	3+	2+
26	MF	44	F	L	2.0	Herpes zoster	Int	2+	2+

*Paramedian (gap less than 1.5 mm); intermediate (gap 1.5 to 2.5 mm); lateral (gap greater than 2.5 mm).
†Recruitment by EMG (graded 0–4+); supplied by the superior laryngeal nerve.
‡Recruitment by EMG (graded 0–4+); supplied by the recurrent laryngeal nerve.
L = left; Lat = lateral; N = normal; R = right; Para = paramedian; Int = intermediate; ca = cancer; s/p = status post.

Figure 2 shows an example of 0 recruitment in the TA muscle (upper tracing) and the voice recording (lower tracing).

We quantify recruitment from 0 to 4+ in accordance with standard EMG practice[12] as follows: 0 = no MUPs; 1+ = 1 to 2 MUPs; 2+ = 3 to 5 MUPs; 3+ = not full recruitment—diminished number of MUPs and baseline EMG appears between successive MUPs; 4+ = full recruitment—neither isolated MUPs nor baseline segments of EMG are detected in the EMG recording.

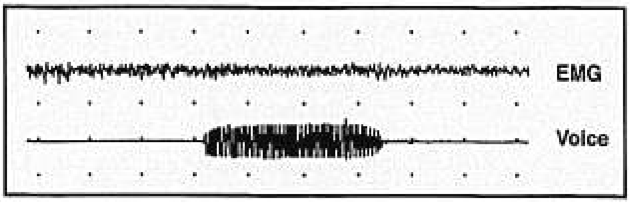

Fig. 2. Abnormal laryngeal EMG tracing of the TA muscle (0 recruitment). The upper tracing (EMG) shows no MUPs, and the lower tracing (voice) shows the voice recording (200 ms/div; top tracing, 500 μV/div; bottom tracing, 2 μV/div).

For the experienced electromyographer, auditory features of the EMG recording are often more informative than the oscilloscopic record. The ear is a much better discriminator of frequency patterns than the eye. With 4+ recruitment, all repeating MUPs blend together in a white noise–like cacophony, whereas with 3+ recruitment the distinctive audio characteristics of individual MUPs can be appreciated by the ear.

Most important in addressing the central issue of this article was a rather simple question: In each of the study subjects was the LEMG of the CT muscle normal or abnormal? We assumed that a normal LEMG implies intact neural function and that an abnormal LEMG implies neural dysfunction. It is important to note that patients who demonstrated no recruitment of voluntary MUPs demonstrated spontaneous activity, thus indicating that the needle electrodes were in muscle.

RESULTS

The findings in the 26 patients in the study are shown in Table I. By TFL, 8 patients had a paramedian paralysis, 7 had an intermediate paralysis, and 11

TABLE II.
Relation Between Site of Lesion and Vocal Fold Position in Laryngeal Paralysis (n = 26).

Site of Lesion*	Vocal Fold Position†		
	Paramedian	Intermediate	Lateralized
Recurrent laryngeal nerve lesion only (n = 13)	4/13 (31%)	4/13 (31%)	5/13 (38%)
Combined lesions (superior and recurrent laryngeal nerves) (n = 13)	4/13 (31%)	3/13 (23%)	6/13 (46%)

*By LEMG.
†By TFL.

TABLE III.
Relation Between Vocal Fold Position and Electromyographic Grading of Thyroarytenoid Recruitment (n = 26).

Position*	Laryngeal EMG (Grading of Recruitment)			
	0	1+	2+	3+
Paramedian (n = 8)	25%	37%	25%	13%
Intermediate (n = 7)	28%	16%	28%	28%
Lateralized (n = 11)	18%	36%	18%	28%

*By TFL.

had a lateral paralysis. By LEMG, 13 (50%) of the 26 patients had isolated lesions of the recurrent laryngeal nerve, and 13 (50%) had combined (superior and recurrent laryngeal nerve) lesions. Of the recurrent nerve lesion group, paramedian, intermediate, and lateral positions were found almost equally, *i.e.*, 4 (31%), 4 (31%), and 5 (38%), respectively. Of the combined nerve lesion group, the results were quite similar: paramedian 4 (31%), intermediate 3 (23%), and lateral 6 (46%). There was no significant difference in the vocal fold positions of either group (Table II).

Because CT muscle function did not appear to influence vocal fold position, we investigated the possibility that TA muscle tone, as a function of recruitment of MUPs, might correlate with position. We reasoned that patients with active MUPs (good tone) would have medial placement of the vocal fold and that poor tone would be associated with a more lateralized position. Table III shows the TA muscle recruitment data for each of the three vocal fold positions; again, no difference among any of the groups was seen.

One trend was noted: the lowest levels of MUP recruitment were usually seen early after onset of paralysis, and recruitment appeared to increase with time. Six patients had 0 recruitment (no MUPs at all) in the TA muscle; in each case, spontaneous activity was present, indicating that the needle electrode was indeed in the TA muscle. Five of the six patients were tested 6 (or fewer) months after onset of the paralysis (patients 5, 9, 17, 19, 21; Table I); however, only one long-term patient had 0 recruitment in the TA muscle 5 years after paralysis (patient 4; Table I). These findings support the clinical observation that electrical silence and vocal fold atrophy after paralysis are uncommon.

DISCUSSION

The Wagner-Grossman hypothesis maintains that when paralysis of the recurrent laryngeal nerve occurs, the vocal fold is maintained in a paramedian position (near the midline) by the action of the ipsilateral CT muscle.[1,4] In 1970, Dedo[4] reported the vocal fold positions and LEMG results of 52 patients with vocal fold paralysis and reaffirmed the Wagner-Grossman hypothesis. It is interesting to note, however, that a review of Dedo's data reveals that other factors (such as posttraumatic fixation) may have been responsible for the vocal fold immobility in some cases; in addition, in other cases the vocal fold behaved in a manner opposite to that predicted, violating the Wagner-Grossman hypothesis.

Recently, Woodson[6] reported 14 cases of unilateral laryngeal paralysis, 7 with recurrent laryngeal nerve lesions and 7 with vagal lesions (diagnosed by LEMG); she found no difference in the positions of the vocal folds in the two groups. In addition, Woodson[7] performed animal experiments and concluded that *1.* vocal fold position in laryngeal paralysis is not determined by the status of the CT muscle; and *2.* LEMG showed that some degree of return of voluntary MUP activity invariably follows paralysis. Our LEMG data confirm the impressions of Woodson, namely that the Wagner-Grossman theory is incorrect and that some degree of spontaneous reinnervation is common in laryngeal paralysis.

Paradoxical Recruitment

The LEMG data suggest that spontaneous regeneration usually occurs after laryngeal paralysis, *i.e.*, muscle tone returns. Why, then, don't these vocal folds recover normal movement? Six patients in the series reported had 3+ recruitment of MUPs in the TA muscles (Table I), and similar findings have been documented in other studies.[9–11]

In other muscles of the body, this degree of electrical activity would be expected to be associated with function (movement) of those muscles. The explanation for this finding is not cricoarytenoid fixation. Many of the patients reported have undergone arytenoid adduction procedures, and in none was cricoarytenoid ankylosis found.

The recurrent laryngeal nerve has nerve fibers that supply opposing (abductor and adductor) muscles. As an analogy, it is as if the biceps and the triceps of the arm had the same motor nerve supplying both muscles. Presumably, the theory that abductor fibers reinnervate adductor muscle and vice versa might explain the persistent immobility of a reinnervated vocal fold. Such synkinesis has been elegantly demonstrated to occur in an animal model.[8] In addition, we have found synkinesis (TA muscle MUPs with sniffing) in some patients with paralysis undergoing LEMG, *i.e.*,

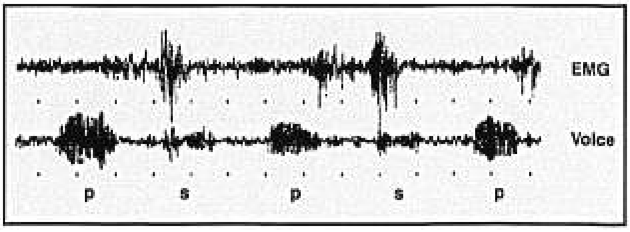

Fig. 3. Synkinesis from an abnormal laryngeal EMG tracing of the TA muscle. There is no recruitment of MUPs with phonation (p), but MUPs are seen with sniffs (s). Normally, the TA muscle does not fire with sniffing, but the posterior cricoarytenoid muscle, an abductor, does. The upper tracing is the EMG, and the lower tracing is voice and sniffs (200 ms/div; top tracing, 500 μV/div; bottom tracing 1 μV/div).

paradoxical recruitment. Figure 3, which shows laryngeal synkinesis, is an LEMG tracing from the patient shown in Figure 2; however, the tracing in Figure 3 was obtained several months after that in Figure 2, indicating that the return of tone was primarily due to aberrant regeneration.

It seems likely that different patterns of spontaneous aberrant regeneration might be the explanation for the different vocal fold positions after laryngeal paralysis. In other words, variations in aberrant regeneration (synkinesis) could, in part, explain vocal fold position, although to speculate on a specific pattern or mechanism to account for each of the positions would be conjectural at this point. New studies with multiple-hooked wire electrodes that simultaneously monitor the CT, TA, and posterior cricoarytenoid muscles could shed light on this hypothesis.

CONCLUSIONS

1. On the basis of LEMG data, it appears that the CT muscle does not predictably influence the position of the vocal fold in unilateral paralysis.

2. In addition, TA muscle recruitment (tone) does not appear to influence vocal fold position.

3. The mechanism(s) responsible for the position of a vocal fold after paralysis remains unknown.

4. Vocal fold atrophy after paralysis appears to be uncommon, perhaps because some degree of spontaneous electrical reinnervation eventually returns in most cases, even when laryngeal nerves appear to have been interrupted unequivocally.

5. Vocal fold paralysis appears to be different from paralysis in other areas of the body. Since substantial LEMG activity returns (*i.e.*, many MUPs) more frequently than vocal fold mobility, it is likely that synkinesis occurs in which abductor fibers regenerate into adductor muscle and vice versa.

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