LACK OF EVOKED LARYNGEAL ELECTROMYOGRAPHY RESPONSE IN PATIENTS WITH A CLINICAL DIAGNOSIS OF VOCAL CORD PARALYSIS

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There has been recent debate about whether patients with vocal cord immobility have a neurologic paralysis or whether synkinesis, the misdirection of axons to competing laryngeal muscles, is responsible for the lack of voluntary vocal cord motion. This issue was studied in 15 patients with vocal cord paralysis who underwent laryngeal reinnervation. Evoked electromyography was performed with a surface electrode endotracheal tube. The recurrent laryngeal nerve (RLN) was identified and stimulated with constant current. Of the 15 patients, only 1 produced a compound muscle action potential upon nerve stimulation. The remaining 14 patients had no evoked response during RLN stimulation. A control group of 8 patients with normal vocal cord mobility was studied, and each had a normal evoked electromyography response after RLN stimulation. These results support the assertion that patients who require treatment for vocal cord paralysis do not have synkinesis produced by RLN reinnervation.

KEY WORDS — electromyography, larynx, paralysis, synkinesis, vocal cord.

INTRODUCTION

Vocal cord paralysis may be secondary to surgical trauma, invasion of the recurrent laryngeal nerve (RLN) by cancer, or penetrating trauma to the neck or chest. In most cases, it is idiopathic. The adopted position of the vocal cord has been a long-standing matter of debate and is thought to reflect the underlying cause of the paralysis.[1] Wagner[2] and Grossmann[3] proposed that isolated RLN paralysis caused the vocal cord to adopt a paramedian position. Combined superior laryngeal nerve (SLN) and RLN paralysis produced a more lateralized position, presumably secondary to the loss of adductory function by the cricothyroid muscle.[2,3] In contrast, Rosenbach[4] and Semon[5] proposed that vocal cord paralysis represented a progressive dysfunction of abductory nerve fibers first, followed later by adductory nerve fibers.

It has been postulated more recently that many patients presenting with a diagnosis of unilateral vocal cord paralysis may actually be demonstrating synkinesis, rather than a truly paralyzed vocal cord.[6] Synkinesis is the misdirected and inappropriate reinnervation of the laryngeal muscles by nerve fibers of the RLN, which results in neuromuscular discoordination and simultaneous contraction of abductor and adductor muscles.[6] An examining otolaryngologist may therefore mistake a cord fixed in position by the simultaneous contraction of its abductors and adductors for a paralyzed vocal cord.

Fifteen patients with a diagnosis of vocal cord paralysis were referred to UCLA Medical Center for evaluation and treatment. Patients with unilateral vocal cord paralysis underwent arytenoid adduction and simultaneous anastomosis of the RLN to a branch of the ansa cervicalis. Patients with bilateral vocal cord paralysis underwent unilateral transection of the thyroarytenoid muscle nerve branch and reinnervation of the posterior cricoarytenoid muscle. Before reinnervation, each patient underwent evoked electromyography (EMG). This article summarizes the results. Only 1 patient with a diagnosis of vocal cord paralysis demonstrated evoked EMG activity with stimulation of the RLN. Evoked EMG in the remaining patients showed no activity in the musculature on the paralyzed side.

PATIENTS AND METHODS

Fifteen patients presented to UCLA Medical Center with a diagnosis of vocal cord paralysis: 13 with unilateral vocal cord paralysis and 2 with bilateral vocal cord paralysis (Table 1). The duration of paralysis ranged from 2 months to 17 years (mean, 3 years 4 months; median, 1 year 2 months). There were 7 men and 8 women. Three patients had received collagen injection previously. One patient had undergone previous type I thyroplasty. All 13 patients with unilateral vocal cord paralysis complained of a hoarse, breathy voice and fatigue with prolonged voice use. Both patients with bilateral vocal cord paralysis com-

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TABLE 1. SUMMARY OF PATIENTS PRESENTING WITH VOCAL CORD PARALYSIS

Patient	*Age (y)*	*Sex*	*Side*	*Duration*	*Cause*	*Prior Therapy*
1	42	M	Left	1 y 1 mo	Aortic laceration	Collagen injection ×1
2	47	M	Left	8 y 7 mo	Idiopathic	Collagen injection ×2
3	29	F	Both	2 y 7 mo	Total thyroidectomy	
4	70	M	Left	8 mo	Total thyroidectomy	
5	44	F	Left	2 y	Idiopathic	
6	35	M	Right	2 y	Idiopathic	
7	25	F	Left	8 y	Idiopathic	
8	36	F	Left	1 y 9 mo	Craniotomy	
9	49	F	Left	1 y	Cervical fusion	
10	52	F	Right	17 y	Thoracotomy	Thyroplasty
11	64	M	Left	8 mo	Carotid endarterectomy	
12	44	F	Both	10 y	Total thyroidectomy	
13	60	M	Right	2 y	Sarcoidosis	Collagen injection ×3
14	55	F	Left	9 mo	Left thyroidectomy	
15	51	M	Left	2 mo	Dissecting aortic aneurysm	

plained of hoarseness and dyspnea with exertion, and were found to have mild inspiratory stridor. Ten of the 15 patients underwent a preoperative phonatory function evaluation that included evaluation of vocal cord waveform, pitch range, subglottic pressure, transglottic airflow, acoustic analysis, percent jitter, mean shimmer, glottic resistance, and harmonics-to-noise ratio.

All patients were intubated transorally with a custom-made endotracheal tube outfitted with surface electrodes capable of recording evoked laryngeal EMG responses. This technique has been reported previously.[7] After the induction of general anesthesia, the larynx was exposed through a transcervical incision with subsequent identification of the SLN and RLN. Separate stimulations of the SLN and RLN on the paralyzed side were performed with 0.1-ms square wave pulses with an intensity of 0.5 to 3 mA.

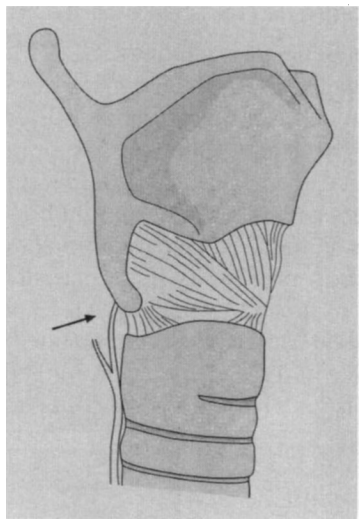

Fig 1. Relationship of recurrent laryngeal nerve to cricothyroid articulation. Nerve enters larynx 1 cm posterior and 1 cm inferior to joint (arrow).

All patients with unilateral vocal cord paralysis underwent arytenoid adduction and reinnervation of the paralyzed vocal cord with anastomosis of the RLN to a branch of the ipsilateral ansa cervicalis. This technique has been described previously.[8] Patient 3, with bilateral vocal cord paralysis, underwent transection of the thyroarytenoid muscle branch through a window made in the ipsilateral lamina of the thyroid cartilage with subsequent anastomosis of the RLN to the ipsilateral ansa cervicalis. This was done to selectively reinnervate the posterior cricoarytenoid muscle and improve her respiratory symptoms. Patient 12, also with bilateral vocal cord paralysis, also underwent transection of the thyroarytenoid muscle branch and reinnervation of the posterior cricoarytenoid muscle with an ipsilateral cricothyroid muscle pedicle. In all patients, the anatomic identity of the RLN was confirmed by following the RLN to its entrance intralaryngeally at the cricothyroid joint. In all patients, the nerve was identified at a point 1 cm posterior and 1 cm inferior to the cricothyroid articulation (Fig 1).

In 4 patients, 28-gauge bipolar concentric needle electrodes (Neurosupplies, Waterford, Connecticut) were placed into the ipsilateral thyroarytenoid muscle through the thyroid cartilage window and the ipsilateral RLN was stimulated. This step was done to confirm the identity of the thyroarytenoid muscle and also to determine whether stimulation of the RLN would activate motoneuron units within the muscle.

All patients underwent identification and stimulation of the SLN on the operative side, and the subsequent EMG waveforms were recorded. This step was done to confirm that the endotracheal tube was in place and that the surface recording electrodes were functional. In addition, a control group of 8 patients who underwent hemithyroidectomy or total thyroidectomy were also studied. All patients had normal

TABLE 2. ABNORMALITIES FOUND ON PHONATORY FUNCTION EVALUATION

Patient	Mucosal Waveform	Pitch Range	Subglottic Pressure (cm H2O)	Transglottic Airflow (LPS)	Acoustic Analysis	Mean Jitter (%)	Mean Shimmer (dB)	Laryngeal Resistance (cm H2O/ LPS)	Harmonics-to-Noise Ratio (dB)
1	↓	↓	↑	↑	Too aperiodic for interpretation				
2	↓	↓	WNL	↑		↑	WNL	↓	↑
3	↓	↓	↑	↑	Too aperiodic for interpretation			↓	
7			↓	WNL				↓	
8			WNL	↑	Too aperiodic for interpretation			↓	
9	Absent		WNL	WNL				WNL	
10			WNL	WNL		↑	↑	WNL	↓
11			↑	↑				WNL	
13				↑		↑	WNL	↓	WNL
15	↓	↓	↓	↑		↑	WNL	↓	WNL

Blank space indicates no data.

LPS — liters per second; ↓ — decreased; ↑ — increased; WNL — within normal limits (ie, subglottic pressure 5.5 to 11.3 cm H2O, transglottic airflow 0.128 to 0.254 LPS, mean jitter 0.324% to 0.641%, mean shimmer 0.189 to 0.495 dB, laryngeal resistance 29.6 to 54.4 cm H2O/LPS, harmonics-to-noise ratio 22.14 to 30.98 dB).

vocal cord motion before and after the operation. In all patients, 1 or both RLNs were identified and stimulated, and the resulting evoked EMG waveforms were compared to those of the patients with unilateral vocal cord paralysis.

RESULTS

All 10 patients who underwent preoperative phonatory function evaluation showed multiple abnormalities (Table 2). Most commonly, unilateral vocal cord paralysis produced decreased glottic resistance, which resulted in a compensatory increase in subglottic pressure and transglottic airflow.

All 8 patients who underwent hemithyroidectomy or total thyroidectomy had normal intraoperative evoked EMG waveforms with RLN stimulation (Fig 2A). In contrast, of the 15 patients with vocal cord paralysis, only 1 patient (No. 6) showed a normal evoked EMG waveform with RLN stimulation. This patient subsequently underwent ipsilateral arytenoid adduction only.

The remaining 12 patients with unilateral vocal cord paralysis showed no evoked EMG waveform with RLN stimulation (Fig 2B). Neither patient with a diagnosis of bilateral vocal cord paralysis showed evoked EMG activity in the vocal cord on the operative side. In all 15 patients, stimulation of the SLN on the operative side produced ipsilateral contraction of the cricothyroid muscle that could be seen visually and that demonstrated a characteristic EMG

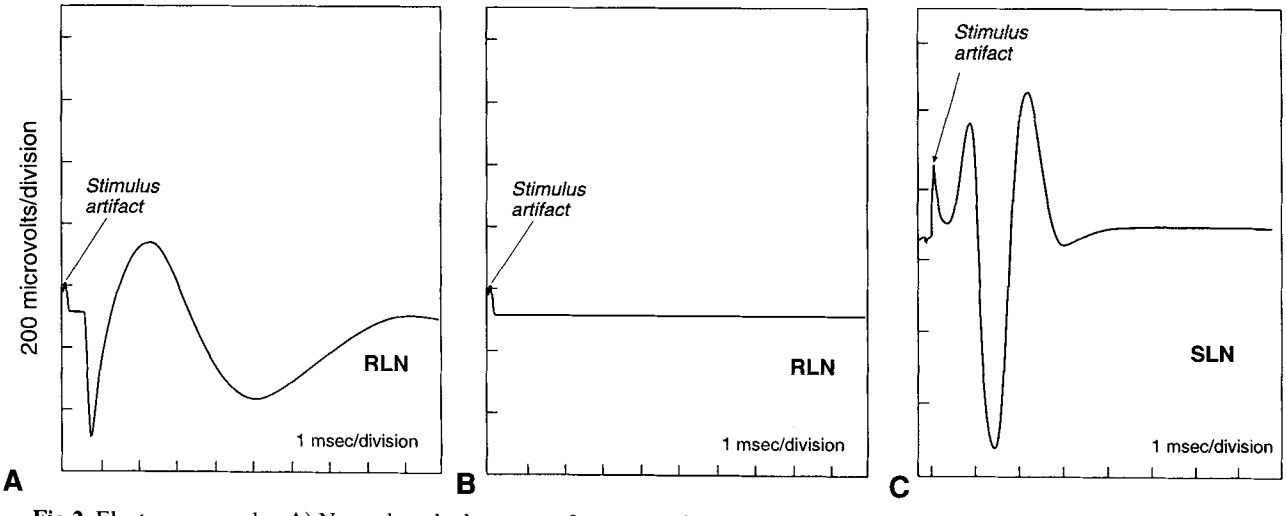

Fig 2. Electromyography. **A)** Normal evoked response from normal vocal cord after stimulation of ipsilateral recurrent laryngeal nerve (RLN). **B)** Lack of evoked response from paralyzed vocal cord after stimulation of ipsilateral RLN. **C)** Normal evoked response after stimulation of ipsilateral superior laryngeal nerve (SLN).

waveform, as well (Fig 2C). This waveform has been described previously.[7] This finding served as confirmation that the endotracheal tube was in place and functional.

Of the 4 patients in whom EMG activity in the thyroarytenoid muscle was measured with intramuscular bipolar concentric needle electrodes, no patient showed evoked EMG activity with stimulation of the ipsilateral RLN. Two patients showed electrical silence after insertion of the needle electrodes, and 2 patients showed fibrillation potentials.

DISCUSSION

Unilateral vocal cord paralysis is uncommon. The most common causes are trauma, thyroidectomy, and nonlaryngeal malignancies, and these constitute the bulk of referrals to otolaryngologists. Approximately one third of patients with idiopathic vocal cord paralysis improve, one third remain the same, and one third require intervention.

In evaluation of an immobile vocal cord, a distinction between true paralysis and immobility resulting from other factors must be made. Paralysis implies a disruption of neuromuscular innervation, whereas immobility may be caused by cricoarytenoid joint fixation, synkinesis, or myopathy.[9] Laryngeal EMG may be useful in distinguishing between these various causes and in assessing the prognosis of vocal cord paralysis.[10-12] It is thought that the phenomenon of synkinesis complicates the functional reinnervation of the paralyzed hemilarynx and that most patients may be demonstrating synkinesis, rather than true paralysis.[6]

Interestingly, despite widely varying causes of vocal cord immobility, none of these 14 patients showed evoked EMG activity with RLN stimulation or evidence of synkinesis. This finding implies either that motoneurons have not regenerated along the RLN or that they have not made functional connections to the motor end plates. In contrast to other series[10-12] that have investigated EMG patterns in the paralyzed vocal cord, the group of patients in this series underwent stimulation of the RLN under direct visualization. Stimulation of the RLN failed to produce movement or EMG activity in the ipsilateral vocal cord. Synkinesis caused by aberrant reinnervation from the RLN did not occur in these 14 patients and therefore could not account for the observed vocal cord paralysis.

The 4 patients who underwent EMG recording from the thyroarytenoid muscle with bipolar concentric needle electrodes also showed no evidence of motoneuron regeneration from the RLN. This finding also served as confirmation that the surface recording electrodes from the endotracheal tube were functioning. Because the needle electrode recordings were simply confirming our observations made with the surface recording electrodes, no further needle electrode recordings were made in the remaining patients.

The 1 patient (No. 6) who was found to have an EMG waveform with RLN stimulation had had paralysis for 3 years. Of interest is that in reviewing the videostroboscopy images obtained before operation, we saw that the right cord was actually paretic rather than paralyzed. The innervation of the cord must have been sufficient to provide a normal EMG waveform but a minimum of physiological motion.

Of all patients in this series who underwent surgery and who subsequently developed a vocal cord paralysis, only 2 (patients 1 and 15) were known to have transections of the RLN. The RLN in the remaining surgical patients was presumed to have been stretched or compressed but left intact. Thus, these patients who sustained trauma to the RLN had the same functional outcome as those patients whose vocal cord paralysis was idiopathic or atraumatic. This finding implies that there may be a common underlying pathophysiologic mechanism at work regardless of the cause of vocal cord paralysis. For example, there may be a physical obstruction somewhere along the length of the RLN or at the junction of its takeoff from the vagus nerve, perhaps from neuroma formation or more extensive scarring, that prevents motor axons from regenerating all the way back to the denervated hemilarynx.

In the evaluation of vocal cord paralysis, EMG may be a useful tool in the differentiation of synkinesis from other disorders.[10-12] For example, with synkinesis, intramuscular EMG recording from laryngeal adductor muscles may demonstrate activity during inspiration and expiration, suggesting aberrant innervation by abductory nerve fibers.[6] Patients who exhibit vocal cord paralysis after traumatic intubation may demonstrate normal EMG activity suggesting arytenoid dislocation.[13] Characteristic evoked EMG patterns may be seen with denervation injury and subsequent reinnervation, depending on the time from the onset of injury.[14] Fibrillation potentials usually denote denervation, with polyphasic potentials indicating reinnervation.

Electromyographic activity may be measured with needle or surface electrodes. Needle electrodes permit measurement of spontaneous and evoked potentials, important in the evaluation of denervation and reinnervation, whereas surface electrodes measure evoked potentials, actually, the summation potential produced by the coordinated contraction of fibers in the muscle under investigation.[15]

Prior investigations in dogs have demonstrated a motor branch from the external division of the SLN to the thyroarytenoid muscle.[16] Wu et al[17] have also confirmed the presence of this branch in 44% of human larynges. The existence of such a branch in a patient may represent an alternative route of motor reinnervation to the paralyzed thyroarytenoid muscle and may mislead the clinician in interpreting EMG data. For example, the detection of polyphasic potentials in a paralyzed thyroarytenoid muscle, although indicative of reinnervation,[14] may actually represent reinnervation from the SLN, rather than the RLN. In addition, adjacent laryngeal muscles may sprout motor branches to their denervated neighbors. This phenomenon of intralaryngeal muscular neurotization has been observed for some time.[18-20] Therefore, polyphasic reinnervation potentials may not be indicative of a pending return of physiological vocal cord function, and represent a potential pitfall in the interpretation of EMG data.

Future investigations should include recordings of evoked and spontaneous EMG activity in the thyroarytenoid muscle with bipolar concentric needle electrodes. There may be scattered motoneuron units within the thyroarytenoid muscle that receive axons either from the regenerating RLN or from the external branch of the SLN, but that collectively fail to produce electrical activity that can be measured with a surface recording electrode. Superior laryngeal nerve and RLN stimulation with intramuscular recording made from bipolar concentric needle electrodes could detect these motoneuron units.

In addition, recording of the RLN compound action potential (CAP) at a site proximal to the RLN's entry into the larynx might elucidate whether motoneuron axons have regenerated in the RLN. A stimulating electrode could be placed on the ipsilateral vagus nerve, and a recording electrode could be placed on the RLN just proximal to its entry into the larynx. After vagal stimulation, the absence of a recordable CAP would imply that motoneuron axons have failed to regenerate along the RLN between the stimulating and recording electrodes. This finding would confirm failure of the motoneuron axons to reach motor end plates, or true paralysis. Alternatively, the presence of a normal CAP would imply that although motoneuron axons have regenerated, they have not made functional connections to the motor end plate. The presence of a diminished CAP would imply incomplete regeneration of motoneuron axons.

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