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The Impact of Cricothyroid Involvement on Adductor Recovery in Unilateral Vocal Fold Paralysis

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Objectives/Hypothesis: Wide variation in postinjury functional recovery is a hallmark of unilateral vocal fold paralysis (UVFP), ranging from zero to full recovery. The present study examined the impact of cricothyroid (CT) muscle involvement on recovery using quantitative laryngeal electromyography (LEMG) of the thyroarytenoid–lateral cricoarytenoid (TA-LCA) muscle complex at multiple times postinjury.

Study Design: Prospective cohort study in a medical center.

Methods: Eighty-one patients with UVFP (37 males and 44 females) received an initial assessment of quantitative LEMG, stroboscope, acoustic voice analysis and 36-Item Short Form Survey quality-of-life questionnaire at 3 to 6 months after UVFP onset and a follow-up assessment at 12 months after UVFP onset.

Results: The initial and follow-up assessments were performed at 4.3 ± 1.9 and 12.5 ± 1.3 months after UVFP onset, respectively. The peak turn frequency of the TA-LCA muscle complex on the lesion side was improved at the follow-up (470 ± 294 Hz) compared with the initial assessment (300 ± 204 Hz) ($P < .001$). Patients were also divided into two groups with (n = 27) and without (n = 54) CT involvement, respectively. TA-LCA muscle complex turn frequency improved in patients without CT involvement (from 277 ± 198 to 511 ± 301 Hz; $P < .001$), but not in those with CT involvement (from 345 ± 211 to 386 ± 265 Hz; $P = .46$). Seventy-one of all patients received early intervention with intracordal hyaluronate injection, showing similar therapeutic effects in those with and without CT involvement.

Conclusions: Acute UVFP with combined TA-LCA muscle complex and CT muscle involvement has a poor prognosis, with poorer recovery of TA-LCA muscle complex recruitment. Early interventions should be considered in patients with UVFP with CT involvement.

Key Words: Unilateral vocal fold paralysis, injection laryngoplasty, electromyography, reinnervation, recurrent laryngeal nerve, superior laryngeal nerve.

Level of Evidence: 2

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INTRODUCTION

Spontaneous recovery of vocal fold motion occurs in approximately 30% of patients with acute unilateral vocal fold paralysis (UVFP),[1,2] but the natural course varies widely among patients. Subclinical regeneration of the recurrent laryngeal nerve (RLN) has been reported in both human and animal histologic studies[3,4] by showing a recovery of recruitment when examined by laryngeal electromyography (LEMG) as well.

Regeneration of the RLN might be influenced by a variety of factors such as the mechanism and severity of injuries.[5] Limited reinnervation of the vocalis muscles will cause vocal fold atrophy and impede voice recovery in UVFP. Reinnervation to the thyroarytenoid–lateral cricoarytenoid (TA-LCA) muscle complex produces contractive tone during phonation and improves voice quality gradually.[6] Spontaneous reinnervation after RLN injuries has been reported in animal models.[7,8] Woodson[4] found that spontaneous reinnervation to the TA was more robust in RLN injury than in vagal injury in a cat model. The findings from Woodson indicated that the location of the injury may determine the prognosis. However, no in vivo studies have reported on the influence of concurrent cricothyroid (CT) involvement on recovery from UVFP. LEMG that can infer the locations of injury by analyzing the involved muscles could then shed light on this issue.

The superior laryngeal nerve (SLN) originates from the vagus nerve, which arises from the nucleus ambiguus and leaves the skull base through the jugular foramen.[9] Theoretically, the CT muscle is innervated by the

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external branch of the SLN, and increases the tension of the vocal folds during phonation. Patients with high-level injury, such as lesions above the bifurcation of the RLN and SLN, or dual laryngeal nerve injury, such as injuries in both the RLN and SLN, will manifest with denervation changes in the CT muscle. UVFP patients with CT muscle involvement have been shown to have poorer voice and quality of life compared with patients without CT muscle involvement.[10]

LEMG has been used to assess the degree of motor unit recruitment for several decades. Serial assessment of the intrinsic laryngeal muscle by LEMG can reflect changes in status with time, indicating the degree of recovery. Quantitative LEMG that measures the turn frequency has recently provided additional information on laryngeal neuromuscular activity.[10–12] Evaluating TA-LCA muscle complex activity using quantitative LEMG at multiple time points can objectively evaluate the recovery.[13]

Spontaneous reinnervation after UVFP can be observed clinically,[4,5] but it remains unclear what type of UVFP is manifested with better spontaneous reinnervation. The present study aims to investigate the role of CT muscle involvement, which indicates concomitant SLN injury, on the spontaneous reinnervation in the TA-LCA muscle complex. Based on previous animal studies,[4,7,14] we hypothesized that the spontaneous reinnervation on the TA-LCA muscle complex can be predicted by their CT muscle status.

MATERIALS AND METHODS

Patients

This prospective cohort study enrolled patients diagnosed with UVFP who presented with voice and swallowing problems within 6 months, confirmed by both laryngoscopy (immobility of one vocal fold) and LEMG (denervation changes in the TA-LCA muscle complex in the paralysis side), from September 2011 to January 2016. The research was approved by the institutional review board of Chang Gung Medical Foundation, and written informed consent was obtained from each participant prior to recruitment. Patients were divided into two groups according the initial LEMG findings: non-CT (without CT muscle involvement) or CT (with CT muscle involvement) groups.

Assessments

Patients received an initial evaluation at 3 to 6 months after symptom onset and a follow-up evaluation at around 12 months from symptom onset. Each assessment involved LEMG, videolaryngostroboscopy, Voice Outcome Survey (VOS), laboratory voice analysis, and 36-Item Short Form Health Survey (SF-36) quality-of-life questionnaire. The enrollment and follow-up flowchart are shown in Figure 1.

LEMG examination. LEMG and quantitative LEMG were performed as described previously.[10] A Nicolet Viking Select (Cardinal Health, Dublin, OH) was used with its band-pass filter set between 20 Hz and 10 kHz. Electric signals were obtained using a concentric needle electrode with the surface ground electrode on the forehead. The neck areas of the TA-LCA muscle complexes and CT muscles were cleaned and sterilized before the test. The protocols were as follows: 1) For the TA-LCA muscle complex, the patient was asked to produce three series of /iii/ sounds at three different intensities (low, moderate, and highest possible),

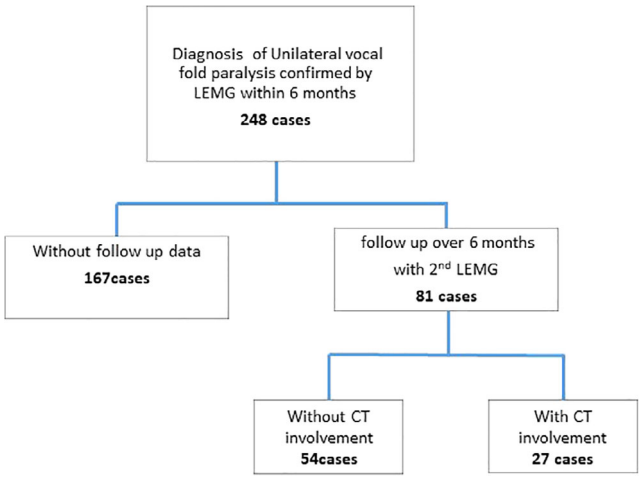

Fig. 1. Flowchart of patient recruitment and group assignment. CT = cricothyroid; LEMG = laryngeal electromyography. [Color figure can be viewed in the online issue, which is available at www.laryngoscope.com.]

with each /i/ lasting at least 400 ms and each inter-/i/ interval lasting about 200 ms.[10] 2) CT function was tested by recording a glissando upward /iii/ at normal loudness three times.

Insertional and spontaneous activities were examined at the initial stage using each test. Then, we performed semiquantitative motor unit and recruitment analyses, specifically when the rise time of a motor unit action potential was <0.6 ms, indicating a close proximity to the recorded motor unit.

The definition of an abnormal LEMG test was the existence of spontaneous activities (i.e., fibrillation, positive sharp wave, or complex repetitive discharge), >30% polyphasic potentials, or decreased interference pattern (reduced, discrete, or no interference pattern). Motor unit recruitment tracings were recorded with sweep speeds of 10 ms per division and a gain of 200 μV per division.

Quantitative LEMG analysis. The raw LEMG data were binned in each 200-ms epoch.[10] An automatic algorithm was used to localize the timing and amplitude of each turn. Specifically, a turn was defined by a change in polarity with an amplitude of at least 100 μV before and after the change, to exclude peaks inherent to noise. Turn frequency was computed for each epoch as the number of turns divided by epoch duration. For the TA-LCA muscle complex, turn frequencies were first computed for each epoch. The peak turn frequency was the mean among epochs with the highest three turn frequencies. The turn ratio is the ratio of peak turn frequency between the lesion and normal sides as shown in Equation 1:

$$\text{Turn ratio} = \text{peak turn frequency in lesion side} / \text{peak turn frequency in normal side} \quad (1)$$

Laryngeal configuration: normalized glottal gap area. Glottis movie images were recorded by videolaryngoscopy while the patient was producing /eee/ at modal pitch and comfortable loudness, and glottal conformations were recorded for several phonatory cycles. The definition of normalized glottal gap area (NGGA), as described in Equation 2, was analyzed using image-processing software (ImageJ; National Institutes of Health, Bethesda, MD). The traced glottal gap area was expressed in square pixels.[15]

TABLE I.
Patient Characteristics.

Demographic Data	Total, N = 81	Non-CT Group, N = 54	CT Group, N = 27	*P* Value
Age, yr	51.1 ± 14.6	49.9 ± 13.7	53.6 ± 16.1	.281
Sex, male/female	37/44	26/28	11/16	.638
Palsy side, left/right	49/32	36/18	13/14	.148
Full recovery, yes/no*	9/72	8/46	1/26	.259
Lesion side TA-LCA peak turn frequency, Hz†		276.8 ± 197.5	345.4 ± 211.2	.154
Normal side TA-LCA peak turn frequency, Hz†		897.2 ± 323.8	762.7 ± 355.3	.221
Etiology				
Nonsurgery				
Idiopathic	14	5	9	
Thyroid tumor	1	0	1	
Herpes infection	1	0	1	
Iatrogenic				
Thyroidectomy	33	19	14	
Esophagectomy	16	16	0	
Lung surgery	8	8	0	
Brain or skull base surgery	3	1	2	
Cervical spine surgery	1	1	0	
Heart surgery	3	3	0	
Mediastinal surgery	1	1	0	

*Recovery to full range of vocal fold motion at follow-up assessment.
†Quantitative laryngeal electromyography assessment at initial assessment.
CT = cricothyroid muscle; F = female; M = male; TA-LCA = thyroarytenoid–lateral cricoarytenoid muscle complex.

$$\mathrm{NGGA} = \left(\text{narrowest glottal-gap area}/\text{membranous vocal fold length}^2\right) \times 100\,\text{units} \quad (2)$$

Voice outcome survey. The VOS voice-related quality-of-life assessment is a five-item survey that evaluates the physical and social problems associated with UVFP.[16] The VOS was translated into Mandarin Chinese by the present research team, following a standard survey-validation process.[17] The survey items and total scores were normalized on a scale of 0 (worst) to 100 (best), based on published algorithms.

Laboratory voice analysis. The procedure was described in previous reports.[17–19] The voice sample was analyzed using voice-analysis software (Computerized Speech Lab model 4300B, version 5.05; Kay Elemetrics Corp., Lincoln Park, IL) with a sampling rate of 25.6 kHz and 16-bit quantization. Fundamental frequency, jitter (perturbation of frequency), shimmer (perturbation of amplitude), and harmonic-to-noise ratio were tabulated from the recorded voice. The maximal phonation time was determined as the duration for which a patient could sustain a vowel /a/. The S/Z ratio, which is the ratio of the duration of an /s/ to a /z/, represented vocal fold control, with the ideal value close to 1.

Health-related quality of life

Health-related quality of life was evaluated using the SF-36 questionnaire. Each score was tabulated according to the published algorithms on a scale of 0 (maximal disability) to 100 (no disability). The validated SF-36 Assessment Standard Taiwan version 1.0 with its Taiwanese norm was adopted.[20,21]

TABLE II.
The Change of Peak Turn Frequency in the CT and Non-CT Groups.

Parameters	Group	Initial Assessment	Follow-up Assessment	*P* Value	*P* Value Group Time
Normal side of TA-LCA, Hz	Non-CT	897.2 ± 323.8	869.4 ± 323.6	.001*	.828
	CT	762.7 ± 355.3	713.6 ± 273.5	.493	
Lesion side of TA-LCA, Hz	Non-CT	276.8 ± 197.5	511.2 ± 301.2	<.001†	<.01*
	CT	345.4 ± 211.2	385.9 ± 264.8	.461	
Turn ratio of TA-LCA	Non-CT	0.31 ± 0.27	0.65 ± 0.41	<.001†	<.05‡
	CT	0.43 ± 0.30	0.50 ± 0.37	.473	

Turn ratio of TA-LCA = lesion side of TA-LCA/normal side of TA-LCA.
*$P < .01$.
†$P < .001$.
‡$P < .05$.
CT = cricothyroid muscle; TA-LCA = thyroarytenoid–lateral cricoarytenoid muscle complex.

TABLE III.
Distribution of the Change Thyroarytenoid–Lateral Cricoarytenoid Peak Turn Frequency.

Group	Not Increased	Increased				
		Subtotal	Chang of Peak Turn Frequency, Hz			
			0–200	201–400	401–600	>600
Non-CT group, n = 54	11	43	15	14	6	8
CT group, n = 27	14	13	6	5	1	1
Total	25	56	21	19	7	9

CT = cricothyroid muscle.

Hyaluronate Injection Laryngoplasty

The study patients were free to choose the treatment options as observation or underwent hyaluronate injection laryngoplasty (HIL) for their initial symptoms, as described previously.[13,19] For HIL, patients were seated upright without sedation, and lidocaine spray was administered into their mouth and nose until numbness was experienced. A fiberoptic laryngoscope with distal chip (laryngoscope: ENF TYPE V2; platform: EVIS Exera II; Olympus Optical Co., Ltd., Tokyo, Japan) was inserted through the patient's nostril to observe the glottal area. A needle was the entered through the CT muscle junction. After passing through the CT muscle membrane, an adequate amount of hyaluronate (Restylane/Juvederm) was pushed slowly into the vocal fold.[13]

Statistical Analysis

The data were analyzed using SPSS (IBM, Armonk, NY). Comparisons of baseline assessments between the two groups were performed using Student t tests for parametric data and χ^2 tests for categorical data. Comparisons of changes in parameters over time between the two groups were conducted using repeated measures analysis of variance (ANOVA). The α value was defined as .05.

RESULTS

A total of 248 UVFP patients received LEMG within 6 months of symptom onset during the study period. Eighty-one patients with acute UVFP (37 males and 44 females, mean age 51.1 ± 14.6 years) were recruited. Their initial and follow-up assessments were carried out 4.3 ± 1.9 and 12.5 ± 1.3 months after symptom onset, respectively. Fifty-four (67%) and 27 (33%) patients were assigned to the non-CT (without CT muscle involvement) and CT (with CT muscle involvement) groups, respectively (Fig. 1). Regarding the etiology, UVFP was surgery-related in 67 (83%) patients and caused by other etiologies in the remaining 14 patients (17%). There were no significant differences in sex distribution, age, side of paralysis, etiology, and initial peak turn frequency of the TA-LCA muscle complex between the non-CT and CT groups (Table I).

Among all patients, the peak turn frequency of the paralyzed TA-LCA muscle complex increased significantly in the follow-up LEMG assessment (from 299.7 ± 203.5 Hz to 469.5 ± 294.0 Hz; $P < .001$) and the turn ratio between paralysis and healthy sides increased from 35% to 60% ($P < .001$). However, the increase differed according to the CT muscle involvement, with a significant increase in the non-CT group (from 277 ± 198 to 511 ± 301 Hz; $P < .001$) but not in the CT group (from 345 ± 211 to 386 ± 265 Hz; $P = .461$) (Table II). At the end of follow-up, nine of the 81 cases (11.11%) showed recovery of full vocal fold motion. Eight of them were from the non-CT group, with only one with CT involvement initially. There was no difference between the two groups in terms of the probability of achieving recovery of full vocal fold motion ($P = .26$) (Table I).

We further analyzed the distributions of patients who showed increases in TA-LCA muscle complex turn frequency at follow-up. Forty-three of the 54 (79.6%) patients in the non-CT group showed increased adductor turn frequency, compared with only 13 out of 27 (48.1%) in the CT group ($P < .01$). The case numbers of specific grade of peak turn frequency changes are shown in Table III.

Seventy-one patients opted to receive HIL. HIL resulted in comparable improvements in patients in both the CT and non-CT groups, reflected by videolaryngostroboscopy, voice analysis, and quality-of-life measurements (all $P > .05$) (Table IV).

DISCUSSION

The major implication for this study is the prognostic value of LEMG. For UVFP patients with unknown nerve segments of injury, such as those induced by thyroidectomy, skull base surgery, or intubation, LEMG is valuable as the involvement of the CT muscle indicates poor prognosis of reinnervation in adductor muscles. In patients of UVFP, the related symptoms can be attributed to the residual and regenerative innervation to the intrinsic laryngeal muscle.[4] Prognostic factors for vocal motion returning in UVFP has been reported,[1,11,22,23] but that for spontaneous reinnervation is still lacking. Damrose et al.[24] suggested that spontaneous reinnervation might be mediated by the SLN or by sprouting from adjacent nonparalyzed laryngeal muscles, a hypothesis that is supported by a lack of evoked LEMG activity in the paralyzed musculature when electrically stimulated at the RLN.[24] Our results showed that involvement of the CT muscle may predict a poor grade of reinnervation in TA-LCA muscle complex, supporting the assistance role of SLN in spontaneous reinnervation following UVFP.

Traditionally, the TA-LCA muscle complex and CT muscles are known to be innervated by the RLN and SLN, respectively. In cases of vocal fold paralysis, CT muscle involvement may represent a high-level injury above the bifurcation of the RLN and SLN or dual

TABLE IV.
Comparison Between the Non-CT and CT Groups for the Improvement Following HIL.

	Group	Baseline	6 Months Post-HIL	Paired *t* Test *P* Value	*P* Value Group × Time
Stroboscope					
Closed-phase NGGA	Non-CT	9.67 ± 10.47	2.55 ± 4.72	<.001*	.515
	CT	8.55 ± 8.91	3.03 ± 5.54	.003†	
Open-phase NGGA	Non-CT	19.7 ± 10.97	12.84 ± 7.99	<.001*	.596
	CT	17.14 ± 8.5	11.85 ± 6.95	.006†	
Voice laboratory analysis					
Maximum phonation time, sec	Non-CT	5.45 ± 4	9.88 ± 5.19	<.001*	.133
	CT	5.3 ± 3.44	8.05 ± 3.66	.008†	
S/Z ratio	Non-CT	2.25 ± 1.42	1.33 ± 0.61	<.001*	.098
	CT	2.09 ± 1.29	1.73 ± 1.38	.170	
Fundamental frequency, Hz	Non-CT	190.57 ± 75.76	169.89 ± 45.65	.027‡	.132
	CT	174.41 ± 59.3	176.97 ± 38.07	.763	
Jitter, %	Non-CT	5.02 ± 5.51	2.28 ± 1.81	.002†	.343
	CT	3.57 ± 2.63	2.12 ± 1.78	.024‡	
Shimmer, dB	Non-CT	0.97 ± 1.06	0.7 ± 1.13	.254	.328
	CT	1.14 ± 1.3	0.46 ± 0.48	.052	
Harmonic-to-noise ratio	Non-CT	5.94 ± 3.26	8.41 ± 3.35	<.001*	.382
	CT	6.01 ± 3.5	7.5 ± 2.29	.068	
Voice outcome survey	Non-CT	32.5 ± 16.18	58 ± 21.05	<.001*	.351
	CT	72.9 ± 22.39	76.3 ± 21.59	<.001	
SF-36 quality of life					
Physical functioning	Non-CT	83.25 ± 18.3	86.75 ± 18.37	.235	.984
	CT	29 ± 42.96	55 ± 47.38	.314	
Role limitation due to physical health	Non-CT	55 ± 47.02	65 ± 45.45	.001†	.233
	CT	46.62 ± 45.2	68.68 ± 42.82	.297	
Role limitation due to emotional problem	Non-CT	60 ± 42.76	70 ± 44.47	.002†	.326
	CT	50 ± 19.43	60.7 ± 17.08	.268	
Vitality	Non-CT	56.25 ± 20.96	63.75 ± 22.88	<.001*	.488
	CT	61.68 ± 18.48	67.2 ± 17.12	.048‡	
Mental health	Non-CT	65.4 ± 22.11	73 ± 17.21	.051	.688
	CT	52.66 ± 25.8	75.52 ± 23.31	.098	
Social functioning	Non-CT	55.7 ± 29.83	73.25 ± 27.38	<.001*	.488
	CT	72.72 ± 24.15	82.38 ± 20.49	.030‡	
Bodily pain	Non-CT	86.5 ± 19.58	87.8 ± 16.27	.007†	.192
	CT	50.1 ± 22.12	57.3 ± 20.73	.807	
General health perceptions	Non-CT	58.5 ± 25.19	59 ± 26.98	.008†	.199
	CT	30.7 ± 13.78	61.7 ± 20.22	.920	

CT group N = 27.
*$P < .001$.
†$P < .01$.
‡$P < .05$.
CT = cricothyroid muscle; HIL = hyaluronate injection laryngoplasty; NGGA = normalized glottal gap area; SF-36 = 36-Item Short Form Health Survey.

injuries involving the RLN and the external branch of the SLN. Branches between these nerves, known as Galen's anastomosis, have been reported to form a communicating nerve in human subjects.[25–28] A human larynx dissection study performed by Maranillo et al.[29] showed the existence of communication branches between the RLN and SLN in 85% of cases, and half of these gave off collateral branches to the TA-LCA muscle complex. The existence of such communication branches supports the hypothesis of alternative motor reinnervation to the TA-LCA muscle complex in UVFP in addition to direct RLN regeneration.

Spontaneous reinnervation has been reported to be affected by the anatomical location of the injury.[4,7,14] Woodson noted spontaneous reinnervation in isolated peripheral RLN injury but not after vagotomy in a cat model,[4] whereas collateral reinnervation from the SLN was shown to aid reinnervation after RLN injury in a rat model as well.[7,14] To the best of our knowledge, the present study is the first to report that CT muscle activity

can predict spontaneous reinnervation of the adductors in patients with UVFP. The finding implies that a patent SLN provides the neuronal substrate for reinnervation.

It is possible that patients with less denervation would expect a better recovery. To control this factor, in the initial evaluation, we performed quantitative EMG for the TA-LCA muscle complex, which measures neuromuscular recruitment from the remaining motor axons in the injured nerve. The results did not reveal a difference between the non-CT and CT groups, and thus the better recovery in the non-CT group is not a result of differences in their initial recruitment levels.

Although the level of reinnervation differed between UVFP patients with and without CT involvement, the effects of HIL were similar in both groups. The development of the distal chip laryngoscope has increased the popularity of injection laryngoplasty with temporary substances during the early phase of UVFP, and the conventional concept of delaying surgical treatment is being challenged by preferable short- and long-term treatment results.[13,30–32] LEMG was suggested as a practical means of stratifying patients with UVFP.[1,18,22,33–36] Finally, given that UVFP with CT muscle involvement has a poorer prognosis, in addition to voice rehabilitation, we suggest that these patients should receive early temporary injection laryngoplasty, rather than following a wait-and-see strategy.

Munin et al.[1] reported that LEMG offered fair-to-good negative prediction accuracy of the return of vocal fold motion. LEMG findings without spontaneous activities (such as fibrillation, positive sharp wave) or synkinesis indicated a higher chance of recovering vocal fold mobility,[23,36] whereas the application of quantitative LEMG further increases the predictive accuracy for vocal fold motion recovery.[11] Voice quality may also improve with time even in patients without vocal motion recovery. From the experiences of reinnervation surgery by ansa cervicalis, the TA muscle tone might be the factor that accounts for their voice recovery.[37,38] Increased recruitment in LEMG might improve muscle tone in laryngeal muscles, further helping restore voice function even in patients without full recovery of vocal fold motion.[6,39] Quantitative LEMG measurements of turn frequency in the TA-LCA muscle complex can reflect the severity of laryngeal nerve injury,[12] whereas improvements in turn frequency between the initial and follow-up assessments can provide an objective quantification of reinnervation. In the present study, although there were only nine patients (11.1%) with motion recovery, around 70% of UVFP cases showed improvements in peak turn frequency in the TA-LCA muscle complex with average turn ratio achieving 60%, a finding that is important for patient consultation for determining the management for acute UVFP. CT muscle involvement in UVFP measured by LEMG predicted less reinnervation in the TA-LCA muscle complex, a condition that may reduce the recovery of muscle tone and voice. Future study needs to correlate the improvement of voice quality and the level of spontaneous reinnervation.

The present study had several limitations. First, we were unable to determine if CT muscle involvement represented injury at the high vagal level or dual SLN and RLN injury, though this would greatly affect the source of the reinnervating axons. Second, the study lacked the power to analyze the correlation between the recovery of quantitative LEMG and voice function. Nevertheless, the evidence is robust that CT muscle involvement by LEMG study indicates a poorer spontaneous recovery, thus providing important information for clinical practice.

CONCLUSION

This study showed substantial improvement of recruitment of the TA-LCA muscle complex in most UVFP patients, with less robust improvement in patients with combined CT muscle involvement. However, early HIL produced similar improvements in UVFP with and without CT muscle involvement.

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