Influence of Superior Laryngeal Nerve Injury on Glottal Configuration/Function of Thyroidectomy-Induced Unilateral Vocal Fold Paralysis

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Abstract

Objective. Recurrent laryngeal nerve (RLN) injury may induce unilateral vocal fold paralysis (UVFP). During thyroidectomy, the most common cause of UVFP, the superior laryngeal nerve (SLN), is also at risk of injury. In the literature, the influence of SLN injury on glottal configuration and function in patients with UVFP remains controversial. The present study investigates SLN injury influence on glottal configuration and function in patients with UVFP after thyroidectomy.

Study Design. Prospective controlled study.

Setting. Tertiary medical center.

Subjects and Methods. The SLN and RLN function of 34 patients with UVFP after thyroidectomy was determined by laryngeal electromyography. The subjects were dichotomized into the isolated RLN injury group (n = 26) or the concurrent SLN/RLN injury group (n = 8). We evaluated glottal angle and paralyzed vocal fold shape during inspiration, normalized glottal gap area, and glottal shape during phonation. The glottal function measurements included voice acoustic and aerodynamic analyses and the Voice Handicap Index. The aforementioned parameters of the RLN and concurrent SLN/RLN injury groups were compared.

Results. There were no statistical differences in glottal configuration such as glottal angle, paralyzed vocal fold shape, normalized glottal gap area, and glottal shape between the RLN and concurrent SLN/RLN injury groups. There were also no significant differences in other glottal function analyses including fundamental frequency, mean airflow rate, phonation quotient, maximal phonation time, and Voice Handicap Index.

Conclusion. In the present study, we did not find any evidence that SLN injury could significantly influence the glottal configuration and function in patients with UVFP.

Keywords

cricothyroid muscle, glottal configuration, superior laryngeal nerve, thyroidectomy, vocal fold paralysis

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In healthy people, the vocal folds abduct for breathing and adduct for phonation. The function of the larynx is controlled by the recurrent laryngeal nerve (RLN) and the superior laryngeal nerve (SLN), which are branches of the 10th cranial nerve, or vagus nerve. The movement of vocal folds may be impaired by various etiologies, and vocal fold paralysis can occur when the RLN is injured. Nowadays, thyroidectomy is considered to be the most common cause of unilateral vocal fold paralysis (UVFP).\(^1\)

The SLN innervates the cricothyroid muscle, which mainly controls the longitudinal tension of the vocal folds and the pitch of the voice.\(^2\)

In patients with UVFP, RLN injury and loss of vocal fold adduction could cause insufficient glottal closure and then hoarseness. However, the effect of the cricothyroid muscle on UVFP is not well understood. According to the Wagner-Grossman theory,\(^3,4\) in RLN paralysis, the

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paralyzed vocal fold is maintained near the midline (paramedian position; Figure 1) by action of the cricothyroid muscle. This theory was based on observations that bilateral RLN nerve paralysis causes airway obstruction, which is relieved by paralysis or removal of the cricothyroid muscle. The validity of this theory has been disputed by other investigations. Woodson and Koufman et al found that vocal fold position in UVFP is not determined by the status of the SLN or the cricothyroid muscle. Because of the discrepancy between studies, the influence of SLN injury on glottal configuration and function of patients with UVFP remains controversial.

In an animal study, Chhetri et al observed that SLN activation increased vocal fold closure speed and consistent phase asymmetry toward the vocal fold with higher SLN activation. In a report of 3 cases of isolated unilateral SLN paralysis/paralysis by Tsai et al, the videostroboscopic findings included ipsilateral vocal fold bowing and shortening. However, in UVFP cases, Woodson indicated that different degrees of RLN injury may cause changes in vocal fold position. Therefore, previous findings of isolated SLN injury could be obscured. Clarification of the distinction between isolated RLN paralysis and concomitant SLN/RLN paralysis could be helpful in clinical practice. If glottal configuration in patients with concurrent SLN/RLN injury was proved to be significantly different from that of isolated RLN injury, some adjuvant surgical procedures that simulate cricothyroid muscle contraction, such as cricothyroid subluxation, could be considered in the treatment plan depending on the laryngoscopic findings. The aim of the present study is to investigate whether SLN injury has a significant influence on glottal configuration and function in patients with thyroidectomy-induced UVFP.

![Image](https://example.com/image.png)

**Figure 1.** Schematic representation of the larynx during inspiration showing the position of the vocal cord. (a) Median. (b) Paramedian. (c) Intermediate. (d) Fully abducted.

### Subjects and Methods

The present study was approved by the Institutional Review Board of Taichung Veterans General Hospital, Taichung City, Taiwan (IRB TCVGH Nos. C07083 and C08208). All patients gave written informed consent for participation in the study. The laryngeal electromyography (LEMG) punctures were performed by the same laryngologist (C.-C.W.), and LEMG signals were interpreted by the same neurologist (M.-H.C.) from the same apparatus (Cadwell Sierra 6200A; Cadwell Laboratories, Inc, Kennewick, Washington). The LEMG procedure and interpretation were described in detail in our previously published article. Briefly, both the cricothyroid muscle and thyroarytenoid muscle were approached transcutaneously. The LEMG signals obtained from the cricothyroid and thyroarytenoid muscles represented the nerve function of the SLN and RLN, respectively. The SLN and RLN injuries were defined as significant recruitment reduction and/or presence of spontaneous activity in the LEMG examination. Only patients with RLN paralysis after thyroidectomy from April 2008 to January 2013 were included for analysis, and the data of 34 patients were used for the study. According to the LEMG findings, we divided the patients into 2 groups: (1) the isolated RLN injury group and (2) the concurrent SLN/RLN injury group.

All study subjects received videostroboscopy for recording of the vocal fold motion and glottal configuration during inspiration and phonation. Through videostroboscopy, we evaluated the glottal angle and paralyzed vocal fold shape (Figure 2) during inspiration and the normalized glottal gap area and glottal shape (Figure 3) during phonation. The digitized stroboscopic images of the glottis at maximal glottal opening during inspiration were captured for analysis. The glottal angle was calculated using Image J 1.42q software (Wayne Rasband, National Institutes of Health, Bethesda, Maryland), and the paralyzed vocal fold shape was classified as bowed or straight, as depicted in Figure 2. The glottal angle was measured during inspiration because the cricothyroid muscle is activated in this respiratory phase, and its action reflects the SLN status. The glottal angle was used in the study by Woodson to reflect the lateralization of immobile vocal fold. According to the Wagner-Grossman theory, if SLN paralysis has a lateralizing effect, the glottal angle may become larger. In addition, according to the observation of Tsai et al., isolated SLN paralysis may manifest as ipsilateral vocal fold bowing and shortening. Therefore, the shape of the paralyzed vocal fold was also recorded in our study for analysis. In addition, the digitized stroboscopic images of maximal glottal closure during phonation were also captured for normalized glottal gap area measurement and glottal shape classification. According to the report by Omori et al., the normalized glottal gap area can be defined and measured as the glottal gap area (pixels × pixels)/(unaffected side membrane vocal fold length)²(pixels × pixels) × 100 (Figure 3A, B). The glottal shape was classified as triangular (Figure 3A) or...
fusiform (Figure 3B). Considering again the Wagner-Grossman theory,3,4 the arytenoid may be more lateralized in concurrent SLN/RLN paralysis in UVFP. Therefore, patients with concurrent SLN/RLN paralysis may have a larger normalized glottal gap area or a triangular glottal shape.

The patients’ glottal functions were recorded via acoustic and aerodynamic analyses in our voice lab. Each patient’s habitual comfortable voice was audiotaped, and parameters including fundamental frequency when counting from 1 to 10, phonation of the vowel /a/, maximal phonation time, mean airflow rate, and phonation quotient were recorded by a commercially available system (Aerophone II; Kay Elemetrics Corp, Lincoln Park, New Jersey). The data of 15 women aged 41 to 49 years without RLN or SLN/RLN injury were retrieved from our previously published studies in which acoustic and aerodynamic data of reference Taiwanese adults were analyzed.14,15 These people were used as a control group to compare their glottal function with that of the RLN injury and concurrent SLN/RLN injury groups in the present study. The Voice Handicap Index (VHI), a self-administered questionnaire developed by Jacobson et al,16 was completed by all patients with UVFP to grade the severity of their voice handicap.

The comparisons of data were performed using the Mann-Whitney U test, Kruskal-Wallis test, or Fisher exact test. Data were analyzed using SPSS software, version 10.0.7 (SPSS, Inc, Chicago, Illinois), and statistical significance was defined as $P < .05$.

**Results**

Of our 34 patients, 26 (76.5%) had RLN injury and 8 (23.5%) had concurrent SLN/RLN injury (Table 1). The median examination time after symptom onset, the median thyroarytenoid muscle recruitment reduction, and the percentage of fibrillation potentials detected in thyroarytenoid muscle showed no significant difference between the 2 groups. The SLN injuries were confirmed by LEMG in the concurrent SLN/RLN injury group, and the median cricothyroid muscle recruitment reduction was 80%. In the

![Figure 2. Glottal angle in inspiration is shown as (o) between paralyzed (p) and normal (n) vocal folds. The paralyzed vocal fold shapes are bowed (A) and straight (B), respectively.](image)

![Figure 3. The normalized glottal gap area was defined as the glottal gap area (*)/normal(n) vocal fold length$^2$ x 100. The glottal shapes are triangular (A) and fusiform (B), respectively.](image)
isolated RLN injury group, 22 patients (85%) were female and 4 were male. All concurrent patients in the SLN/RLN injury group were women. There were no significant differences in gender or ages between the 2 groups (Table 1).

The results of the glottal configuration analysis are summarized in Table 2. There were no statistically significant differences in glottal angle, paralyzed vocal fold shape, normalized glottal gap area, or glottal shape between the 2 groups. The glottal functions, including voice acoustic/aerodynamic data analysis and VHI scores, of the RLN injury group (excluding the data of the 4 male patients), the concurrent SLN/RLN injury group, and controls are summarized in Table 3. There were no significant differences between the 2 diseased groups in all parameters (a vs b). When we compared the data of the 2 diseased groups with the data of controls, we observed no statistical differences in fundamental frequency. Except for maximal phonation time, mean airflow rate, and phonation quotient, the differences were statistically significant (Table 3). Because the control group had no voice problem, they did not need to complete the self-administered VHI, and the VHI score was deemed to be zero.

**Discussion**

In patients with UVFP, RLN injury could cause insufficient glottal closure and then husky voice. According to the study by Rosenthal et al.¹⁷ iatrogenic injuries during surgery and malignancy are the major causes of UVFP. Multiple series¹⁸-²⁰ reported that iatrogenic injuries are more frequent, and thyroidectomy remains the most common surgery associated with iatrogenic UVFP. The surgical importance of the RLN and SLN is due to their close anatomical relationship with the thyroid gland.²¹ The incidence of thyroidectomy-induced RLN damage ranges from 0.2% to 13.2%.²²-²⁴ Although SLN is at minimal risk during thyroidectomy, in some circumstances, the intimate anatomical relationship of SLN and tumors or impossible visual identification of SLN that is located deeply in the fascia of the inferior constrictor muscle may produce an SLN injury.²⁵,²⁶ Chhetri et al.⁸ and Tsai et al.⁹ described their findings of isolated unilateral SLN injury. However, in most cases, SLN injury can be difficult to identify intraoperatively and is difficult to detect during routine postoperative laryngoscopy.²⁷ When SLN paralysis occurs concurrently with RLN paralysis, the additional impact of SLN paralysis on the glottal configuration or function in patients with UVFP remains controversial.

At the end of the 19th century, Wagner³ and Grossmann⁴ proposed that a paralyzed vocal fold in the paramedian position should be considered as a pure unilateral RLN paralysis and that a paralyzed vocal fold in the intermediate position should be considered as a combined paralysis of the RLN and SLN. This theory was supported in a 1970 study conducted by Dedo²⁸, he demonstrated that after an RLN section in dogs, the ipsilateral vocal fold was paralyzed in the paramedian position and that a further section of the SLN...
Table 3. Comparison of Glottal Function of Women in the RLN Injury Group (n = 22), Concurrent SLN/RLN Injury Group (n = 8), and Controls (n = 15).a

<table>
<thead>
<tr>
<th>Group</th>
<th>Counting F0, Hz, Median (IQR)</th>
<th>/a/ F0, Hz, Median (IQR)</th>
<th>MPT, s, Median (IQR)</th>
<th>MAFR, mL/s, Median (IQR)</th>
<th>PQ, mL/s, Median (IQR)</th>
<th>VHI, Median (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RLN Injury (a)</td>
<td>201 (186-212)</td>
<td>206 (189-221)</td>
<td>3.73 (2.2-5.2)</td>
<td>365 (206-480)</td>
<td>550 (388-653)</td>
<td>71 (56-81)</td>
</tr>
<tr>
<td>SLN/RLN Injury (b)</td>
<td>197 (155-217)</td>
<td>193 (152-207)</td>
<td>3.5 (3-5.8)</td>
<td>332 (127-426)</td>
<td>576 (303-698)</td>
<td>66 (31-98)</td>
</tr>
<tr>
<td>Controls (c)</td>
<td>195 (180-209)</td>
<td>207 (184-223)</td>
<td>23 (21-28)</td>
<td>75 (50-122)</td>
<td>119 (99-146)</td>
<td>NA</td>
</tr>
</tbody>
</table>

Abbreviations: F0, fundamental frequency; IQR, interquartile range; MAFR, mean airflow rate; MPT, maximal phonation time; NA, data not available because the Voice Handicap Index was not administered and should be zero in controls; PQ, phonation quotient.

*P values are bolded when statistically significant.

drove the vocal fold into the intermediate position. Dedo then concluded that the cricothyroid muscle has tensile and also adductive action on vocal folds. However, there were some exceptions to this observation, and the intermediate vocal fold position was also observed in some patients with isolated RLN paralysis. Besides, the categorization of paralyzed vocal fold positions is completely subjective without any quantitative measurement of the glottal angle.

Woodson5,6 in 1993 disagreed with the Wagner-Grossmann theory. In a study of 14 subjects with UVFP, Woodson found that there was no statistically significant difference between the RLN injury group and the concurrent SLN/RLN injury group in terms of glottal configuration and glottal angle.5 The main differences between Woodson’s study5 and the present one are the etiologies of UVFP for recruited patients, application of LEMG, and evaluated parameters. In Woodson’s study,5 UVFP patients had various etiologies. The degree of RLN injuries was not evaluated by LEMG, and the severity of RLN injuries in different groups might be considered a confounding factor. In our study, thyroarytenoid muscle recruitment reduction reflects the degree of RLN injury, and there was no significant difference between the 2 diseased groups. Therefore, the bias of RLN injury difference was minimal, and we might reduce the potential confounding effects from heterogeneous etiologies or asymmetry of RLN injury severity. Koufman et al7 also disagreed with the Wagner-Grossmann theory. In their study, SLN paralysis was confirmed by LEMG, but the vocal fold position was subjectively categorized only to paramedian, intermediate, and lateral positions. In addition, we evaluated voice parameters that were not used in the aforementioned 2 studies. From different research methods, we believe we have provided further information on the issue under discussion.

Between the RLN injury group and the concurrent SLN/RLN injury group, there was no significant difference in glottal angle in our study (Table 2). Our results are in accordance with those of Woodson,5 indicating that loss of unilateral SLN function does not significantly influence the position of the paralyzed vocal fold during inspiration. Regarding the mechanics of the laryngeal skeleton, contraction of one cricothyroid joint by the healthy-side cricothyroid muscle should affect both vocal folds equally in unilateral SLN paralysis. Even if physiologic contraction of both cricothyroid muscles could exert a mediating force on a paralyzed vocal fold, loss of one cricothyroid muscle would not completely abolish that force.29 Similarly, there was no significant difference in normalized glottal gap area (Table 2). This finding further indicates that the loss of SLN function does not change the glottal area significantly either in inspiration or phonation.

Aforementioned in the introduction section, SLN paralysis may make the vocal folds look bowed, but there was no significant difference in paralyzed vocal fold shape between the 2 groups (Table 2). Actually, 46% of RLN injury patients also had bowed vocal folds. This may be due to common anterior displacement and slackening of the paralyzed vocal fold with loss of the opposing contraction by the posterior cricoarytenoid muscle that is innervated by the RLN.5 During phonation, there was no significant difference in glottal shape either (Table 2). Glottal configuration both at rest and during phonation is a complex phenomenon that cannot be attributed solely to the action of adductor and abductor muscles. In the various laryngeal movements, it is possible to recognize the contribution of all the muscles of the larynx.30 Since the RLN controls all of the intrinsic muscles of the larynx except the cricothyroid muscle, we would expect that the contribution of a single cricothyroid muscle to SLN function may have a limited effect on glottal configuration.

In glottal function analysis (Table 3), no statistical differences were found between the RLN injury group, the concurrent SLN/RLN injury group, or controls when we compared counting from 1 to 10 and vowel /a/ fundamental frequency. Because a large variation of fundamental frequency is usually found in controls, the influence of RLN or
SLN injury may not be significant. However, when we analyzed the aerodynamic data, we found there were statistically significant differences between both diseased groups and the control group because of the glottal closure insufficiency during phonation (Figure 3). The maximal phonation time was significantly shorter because the mean airflow rate or phonation quotient was higher in patients with UVFP. However, the aerodynamic data were not significantly different between the RLN injury and concurrent SLN/RLN injury groups. Again, it shows that SLN injury is not an important factor in the aerodynamics of patients with UVFP. Therefore, the vHI scores of the RLN injury and concurrent SLN/RLN injury groups did not differ significantly.

There were some limitations of our study. First, the sample size was small. From a power analysis (80% power, 5% α value) of our sample, in order to exclude type 1 or type 2 errors, we would need a sample size ranging from 41 to 750,805 patients (179 for the glottal angle evaluation, 41 for the paralyzed vocal fold shape, 134 for the normalized glottal gap area, 467 for the glottal shape, 285 for counting the fundamental frequency, 66 for the vowel /a/ fundamental frequency, 519 for the maximal phonation time, 256 for the mean airflow rate, 750,805 for the phonation quotient, and 994 for the VHI). However, studies with a similar research purpose that we cited in this article, including those by Woodson and by Koufman et al., also had small sample sizes. This reflects the reality that this study topic is clinically hard to investigate. In our opinion, it is very difficult to obtain a sample that is large enough to achieve adequate power for every measured parameter in a single medical center. The limitation could probably be minimized by conducting a multicenter study in the future. Currently, we have tried our best to reduce other possible biases by recruiting patients prospectively with homogenous etiology and quantitatively measuring RLN function and glottal configuration/function. However, the level of medical evidence of this study still remains level IIIb (individual case-control study with very limited population).  

Second, there is no standard methodology for evaluating SLN function in patients with UVFP. To make a more accurate evaluation of vocal fold position, Woodson and we, in the present study, used glottal angle as a parameter that could be gauged by a protractor. Woodson also evaluated other parameters such as the vocal fold angle at the tip of the vocal process of the arytenoid cartilage, and she used the vocal fold ratio to compare the length of paralyzed and mobile vocal fold membranes. She found that the arytenoid cartilage was tipped anteriorly in every patient with UVFP. Sometimes the vocal fold membrane was obscured by the overhanging arytenoid and the aforementioned 2 parameters could only be estimated. Because we also had similar findings in our study, we did not use the 2 parameters for analysis. However, we applied other videolaryngoscopic and functional parameters listed in Tables 2 and 3. Unfortunately, the highest tones produced by a task that may challenge the cricothyroid muscle were not recorded for every patient because it was hard for some patients with glottal insufficiency to follow the instructions and elevate the pitch. Therefore, the analysis of maximal pitch change was discontinued. However, according to the literature, there were large variations of maximal pitch in normal people in several studies. If we used the highest pitch or pitch range as a parameter, it probably would not have made any significant difference. Although it would be desirable to collect pre- and postoperative glottal morphological and functional data for comparison, this would be very difficult to do since most patients are referred from other health care centers and preoperative laryngoscopic image recording is not a routine procedure in clinical practice. Finally, according to our results, concurrent SLN/RLN injury cannot be distinguished from a pure RLN injury by laryngeal imaging alone. Further investigation is needed to devise other measures that could help differentiate concurrent SLN/RLN injury from pure RLN injury and define the clinical impact of this differentiation.

Conclusion
Concurrent SLN/RLN injury is not uncommon in thyroidectomy-induced UVFP. In our study of 34 patients with UVFP, we did not find any evidence that SLN injury could significantly affect the glottal configuration, acoustic/aerodynamic functions, or subjective VHI in patients with RLN paralysis. However, a study with a larger scale remains necessary to further clarify this specific topic.

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Author Contributions
Armando De Virgilio, study concept, statistical analysis, draft of the manuscript; Ming-Hong Chang, acquisition of data and analysis, article revision for important intellectual content; Rong-San Jiang, acquisition of data and analysis, article revision for important intellectual content; Ching-Ping Wang, acquisition of data and analysis, article revision for important intellectual content; Shih-An Liu, study design, data interpretation, article revision for important intellectual content; Chen-Chi Wang, study concept, data collection, draft of the manuscript.

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