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Superior laryngeal nerve injury: effects, clinical findings, prognosis, and management options

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Abstract

Purpose of review—The superior laryngeal nerve (SLN) provides motor innervation to the cricothyroid muscle. However, the functions of this muscle and the anatomic variations of the nerve that supplies it are not fully understood. SLN paresis and paralysis (SLNp) is difficult to diagnose because of a lack of consistent laryngeal findings, and its effects on the voice likely goes beyond simple pitch elevation control.

Recent findings—Although SLNp has traditionally been thought to lead to voice pitch limitation, recent research findings reveal multiple roles for this nerve in voice and speech. Cricothyroid muscles are the primary controls of fundamental frequency of voice. SLNp can lead to significant contraction of pitch range, vocal fold vibratory phase asymmetry, and acoustic aperiodicity, thus leading to an overall poor vocal quality. In addition, cricothyroid muscles may also play a role in pitch lowering and shifting from voiced to unvoiced sounds during speech.

Summary—Subtle signs, symptoms, and diagnostic findings associated with SLNp make this disorder difficult to characterize clinically. Lack of treatment methodologies to restore the dynamic action of the cricothyroid muscles poses difficulties in treating patients with this condition. A more thorough understanding of the effects of SLNp will improve diagnosis and treatment.

Keywords

cricothyroid muscle; superior laryngeal nerve; voice disorder

INTRODUCTION

The superior laryngeal nerve (SLN) and its functions, although physiologically important, may still be one of the least understood in the head and neck region. Most of the research and clinical interest in the field of neurolaryngology is focused on the recurrent laryngeal

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Conflicts of interest

The views expressed in this article are those of the author and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the United States Government. There are no other conflicts of interest.

nerve (RLN). This is likely because RLN injury is clinically more important as well as easier to identify and treat.

The prevalence of SLN injury and dysfunction is unknown as many cases go undiagnosed. Injury to the external branch of the SLN in thyroidectomies is reported to be as high as 58% [1,2]. However, isolated injury to the external branch can be difficult to detect because of subtle symptoms and variability in the clinical signs and symptoms. Injury to the common trunk of the SLN can also occur, primarily during skull base surgery or with lateral approaches to the pharynx, as a result of direct injury to the vagus nerve. Injury to the internal branch results in lack of sensation from the supraglottic larynx and contributes to dysphagia.

Anatomy

The SLN branches from the vagus nerve near the inferior half of the nodose ganglion, about 36 mm below the jugular foramen and 40 mm above the carotid bifurcation [2,3]. It then travels 1.5 cm caudally in which it divides into the internal and external branches [1]. The internal branch, which pierces the thyrohyoid membrane, provides sensory innervation to the supraglottic larynx, whereas the external branch, carrying motor innervation, travels to and innervates the cricothyroid muscle. There is some variation within the course of the nerve, specifically with relation to the superior thyroid vessels as originally described by Cernea [2]. In addition, its relationship with the superior constrictor may be highly variable, as it can run entirely superficial to the muscle, completely within the muscle or partially within the muscle as first described by Friedman *et al.* [4]. In addition, there are reports of a connection from the external SLN branch to the midportion of the thyroarytenoid muscle, described as the human communication nerve [2,5]. According to Wu *et al.* [5], this nerve was present in less than half of the specimens and may assist in adduction of the vocal fold. However, whether the SLN provides motor innervation to the thyroarytenoid remains to be investigated.

The major muscle supplied by the SLN, the cricothyroid muscle, consists of two parts: the pars recta and the pars oblique. Both parts originate at the inferior margin of the thyroid cartilage with the pars recta originating from the midline to the thyroid tubercle and the pars oblique from the thyroid tubercle to the inferior cornu. These then insert on the anterior and lateral portions of the cricoid cartilage. A third muscle belly has also been described, which originates from the inner perichondrium of the thyroid cartilage [3,6]. It is generally accepted that the thyroid cartilage remains fixed because of its attachments to the hyoid and sternum via the strap muscles, thus cricothyroid contraction primarily moves the cricoid cartilage. Paired cricothyroid joints and ligaments allow two main types of motion: first, rotation around the common frontal axis of both; and second, sliding horizontal motion in the sagittal direction [6]. Symmetric contraction of the cricothyroid muscles raises the anterior cricoid cartilage, resulting in narrowing of the cricothyroid space, posterior and inferior motion of the arytenoids, and lengthening and thinning of the vocal folds. When the cricothyroid joint is contracted to its limits of motion, the sliding motion is not possible because of distension of the cricothyroid ligaments [6]. It is believed that with unilateral contraction, particularly with the par oblique, this sliding motion may occur resulting in the

rotation of the larynx sometimes seen with unilateral paralysis, although this may not occur in all larynges [3].

EFFECTS

Very little has been written about SLN paresis and paralysis (SLNp) regarding specific effects on the voice [3]. It is commonly felt that SLNp primarily affects professional voice users as it hampers their ability to produce higher vocal registers and decreases vocal projection [2,3]. In an excellent study evaluating phonatory effects of SLN paralysis, Roy *et al.* [7] performed selective blockade of the cricothyroid muscle and external branch of the SLN with lidocaine in 10 patients. The most common effect on the voice was reduced fundamental frequency range and reduction in the highest obtainable fundamental frequency. Interestingly, patients also had contraction of fundamental frequency at the lower range as well. This would be compatible with studies showing role of cricothyroid muscles in fundamental frequency lowering and devoicing during speech [8■,9]. Voicing and devoicing are important phenomena in which sounds are shifted between voiced and unvoiced, depending on the phonologic environment. Roy *et al.* [7] also noted a decrease in mean airflow with normal phonation and overall increase in subglottic pressures as well as increased phonatory instability characterized by increased jitter. These changes are possibly secondary to increased compensatory thyroarytenoid activation. Thyroarytenoid activation can also increase fundamental frequency in certain circumstances [10■■]. This would also explain overall increased laryngeal resistance and increased subglottic pressure.

Roy *et al.* [11] also evaluated the effects of unilateral external branch SLN block on laryngeal position and vibration. They did not find vibratory phase asymmetry, length asymmetry, or vocal fold height mismatch. Instead, they reported deviation of the petiole toward the side of the weakness in 60% of patients, particularly with glissando up maneuver. They also reported improved glottic closure with high-pitched voice tasks and axial rotation of the posterior commissure toward the normal side in 50% of patients with rapidly alternating ‘eee’ and sniff maneuver. Chhetri *et al.* [12■] showed, in a canine model, that simulated unilateral SLNp resulted in a significant vibratory phase asymmetry, with the side with the intact SLN opening earlier. Although not seen in the study by Roy *et al.* [11], there are several case reports/case series that support the concept of phase asymmetry resulting from SLNp [13-15]. Phase asymmetry has been demonstrated in physical models, canine models, and humans diagnosed with SLNp using laryngeal electromyography (LEMG). We believe that a phase asymmetry is present with SLNp. Unfortunately, phase asymmetry is not specific for SLNp and can be seen with other causes of tension asymmetry such as RLN paresis and paralysis [16].

Current literature provides no consensus for diagnosis of SLNp based on laryngoscopy alone. In our opinion, there are two main factors resulting in variability of examination. The first is variability in the cricothyroid joint horizontal sliding motion, which may add to vocal fold stiffness changes in the transverse plane [12■]. If no horizontal sliding was present and the joint only moved along the frontal axis, minimal changes to the voice would be expected with unilateral cricothyroid paralysis. It is likely that the degree of sliding permitted correlates with more severe voice changes [3,6]. The second is the compensatory action of

the intrinsic muscles of the larynx, particularly the thyroarytenoid and possibly the lateral cricoarytenoid muscles, which affects the overall balance of vocal fold tissue stiffness and thus laryngeal posture and vibration. In addition, this compensation may change significantly over time.

DIAGNOSTIC TEST

LEMG has been used as the gold standard in diagnosis of SLNp because of its ability to detect signs of denervation [3]. Although LEMG may be more reliable than laryngoscopy, there is very little research specifically addressing its role in the diagnosis or treatment of SLN injury. There have been no studies comparing LEMG finding and vocal quality. Jansson *et al.* [1] showed, using preoperative and postoperative EMG after thyroidectomy, that the majority of patients with EMG-diagnosed SLNp had a reduced pitch and poorer quality voice. However, they also noted that several patients with preoperative SLNp had no vocal abnormalities at the time of testing. Dursun *et al.* [17] recommended performing LEMG combined with physical exam findings to confirm the diagnosis. There are several limitations of LEMG. First, interpretation of LEMG findings is subjective and accuracy of findings is dependent on the experience of the person performing and interpreting the test. Second, some technical issues may remain such as sampling of adjacent nontarget muscle, such as the strap muscles [18]. Others have reported the possibility of sampling error examining partially denervated muscle or longstanding paralysis with variability in reinnervation. Repeated EMG needle insertions leading to muscle hemorrhage or transient damage may also lead to false positive results [18]. Finally, patients with voice problems, especially following surgery, are hesitant to undergo procedures perceived to be invasive, especially if it is unclear whether it would change the recommended treatment, thus limiting the usefulness of LEMG [19].

PROGNOSIS

There are no studies specifically addressing the prognosis relating to a SLN injury, regardless of cause. Idiopathic SLNp, which appears to be fairly rare, has been assumed to be due to viral neuropathy and is expected to behave in a manner similar to RLN paralysis [12■]. Because of the difficulty in reliably diagnosing SLN injury, determining a rate of recovery is difficult. For SLNp after thyroidectomy, spontaneous recovery is variable [2]. Many report the outcomes as ‘poor’ or ‘worse’ than RLN injury and recommend early voice therapy to prevent poor voice outcomes. However, these recommendations are uniformly based on anecdotal observations. It is likely that these cases with poor outcomes are simply cases severe enough to be diagnosed with the paralysis in the first place, leading to a high level of observational bias [13]. It is our opinion that cricothyroid dysfunction following thyroid surgery can also result from direct injury to the cricothyroid and may occur more commonly from that injury to the SLN. This may occur during dissection of the isthmus, pyramidal lobe, or delphian node [3].

TREATMENT

A wide variety of treatment options have been previously reported, ranging from steroids for acute potential viral injury, voice therapy, and a variety of surgical procedures including

medialization laryngoplasty, modified type 4 thyroplasty, variations on cricothyroid approximation, and reinnervation using nerve muscle pedicle technique [3,20-22]. Because of difficulty in accurately diagnosing SLN injury, discussion of treatment options is limited to anecdotal evidence.

Voice therapy is the most commonly prescribed therapy for long-standing isolated SLN paralysis, although it seems that the improvement in voice is variable. Therapy is targeted at building cricothyroid muscle strength using activities such as glissando maneuvers [17]. The variability in the effectiveness of voice therapy likely stems from both the difficulty in diagnosing SLN paralysis via clinical means and variable effects of SLN paralysis on different patients. There are no studies adequately evaluating the effects of voice therapy on SLN paralysis.

Although not commonly performed, there are several small case series describing surgical treatment for SLN injury. One study by Shaw *et al.* [21] used carefully applied unilateral cricothyroid approximation (type 4 thyroplasty) in a series of 10 patients with reportedly excellent improvement in the quality of the voice and vocal range. A longterm follow-up for a similar procedure, however, showed persistent quality improvement but a decrease in range over time, likely due to decreased tension on the suture over time. Nasseri and Maragos [20] performed both type 1 and type 4 thyroplasty in nine patients. Again, good results were reported. The addition of the type 1 thyroplasty was added, because of the ‘decreased’ power noted with SLN paralysis. The study by Yin and Zhang [10■■] would support this concept. The addition of medialization likely further enhances the compensatory activity of the thyroarytenoid muscle, increases stiffness of the vocal fold, and allows for more efficient increase in subglottic pressures, leading to improvement in vocal quality and range. However, more research needs to be performed to prove that these surgeries are effective.

Another surgical procedure has been described in a small series of patients using a ‘muscle nerve muscle’ pedicle using a nerve cable graft. In this procedure, a nerve cable is attached to the normal cricothyroid muscle on one side; it is then grafted into paralyzed cricothyroid muscle on the contralateral side [22]. Although the authors describe good results with this technique, it would seem that there is a lot of room for failure when using a cable graft just buried into muscle bellies. However, further research into reinnervation techniques is warranted to restore the dynamic role of the cricothyroid muscle during phonation.

CONCLUSION

The major factor impeding progress in determining adequate treatment for SLN paralysis has been the general lack of diagnostic criteria, likely leading to many undiagnosed or misdiagnosed cases. New research into voice changes associated with this condition will likely improve diagnostic accuracy and hopefully remove the idea that the SLN only contributes to pitch elevation. Further research is needed to determine improved diagnosis on examination and to improve the index of suspicion in patients with poor voice quality and reduced vocal range. Once there is improved diagnostic accuracy, further work can be performed on treatment of this complex disorder.

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■ of special interest

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KEY POINTS

- SLN paralysis has a complex effect on voice, which accounts for more than just increases in pitch.
- Vibratory phase asymmetry is likely the most common finding on laryngoscopy but is not specific to SLN paralysis.
- LEMG is currently the gold standard in diagnosis, although this has not been well studied in terms of outcomes.
- Voice therapy is the most commonly prescribed initial treatment.
- Combined medialization laryngoplasty and modified type 4 thyroplasty appears to be one of the most promising surgical treatment options.