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Superior laryngeal nerve preservation in peri-apical surgery by mobilization of the viscerovertebral angle

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Abstract
Iatrogenic lesions of the superior laryngeal nerve (SLN) are much more common than is generally recognized. Since injury to this nerve may cause transient or even persistent changes either in quality of voice or in deglutition, an attempt should be made to localize and identify the nerve during surgery. This study included 74 patients who underwent surgical dissection near the thyroid apex in the neck. Effective prevention of SLN injury during surgery was achieved by anatomical localization of the nerve in the viscerovertebral angle and its functional identification with the nerve stimulator. Post-operative analysis consisted of subjective interview, rigid laryngoscopy, acoustic analysis, laryngeal videostroboscopy and cricothyroid electromyography. Four patients complained of post-operative voice changes; two were diagnosed as SLN injury (2.4 per cent), one as reflux laryngitis and the fourth as intubation granuloma. Surgical access to the SLN in the periapical area may be achieved through mobilization of the viscero-vertebral angle. The use of a nerve stimulator during difficult situations should keep SLN injury at a minimum.

Key words: Laryngeal nerves; Thyroidectomy; Laryngectomy; Laryngoscopy; Electromyography

Introduction

The superior laryngeal nerve (SLN) and its external branch are at risk during surgical dissection around the thyroid apex, and several techniques have been described for their intra-operative identification to minimize potential damage. The importance of preserving the external laryngeal nerve (ELN) was dramatically demonstrated in 1935 when the famous opera soprano, Amelita Galli-Curci, sustained injury to that nerve during thyroidectomy for an enlarging goitre. This complication ended her glorious career.[1] The most recommended procedure for avoiding injury to the nerve is anatomical localization of the nerve during dissection.[2] Accurate localization of the SLN may be achieved by surgical mobilization of the viscerovertebral angle.

Surgical anatomy

The viscerovertebral angle is an anatomico-surgical abstraction describing the pathway from the base of the skull to the mediastinum, through which pass the major neurovascular structures of the neck (Figure 1). The viscero-vertebral angle is bounded by: (1) the myovertebral column including the prevertebral muscles, the vertebrae and the spinal cord, (2) the cervicovisceral column including the pharynx, the oesophagus, the larynx, the trachea, the thyroid and the parathyroid, and (3) the great neurovascular conduit including the carotid arteries, the internal jugular vein, the vagus nerve, the sympathetic chain and the descendens hypoglossi nerve.[3] The hyoid bone is of central importance to mobilization of the viscero-vertebral angle because it provides attachment to most of the cervical musculature especially the digastric and stylohyoid muscles by which it is attached to the base of the skull, and the omohyoid muscle by which it is attached to the scapula (Figure 2). The digastric and stylohyoid muscles

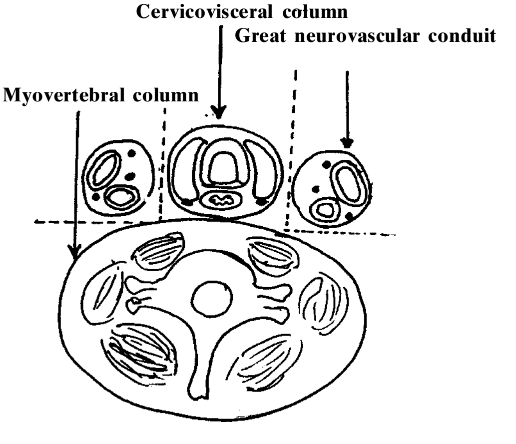

Fig. 1
Viscero-vertebral angle and the cluster of colonnades.

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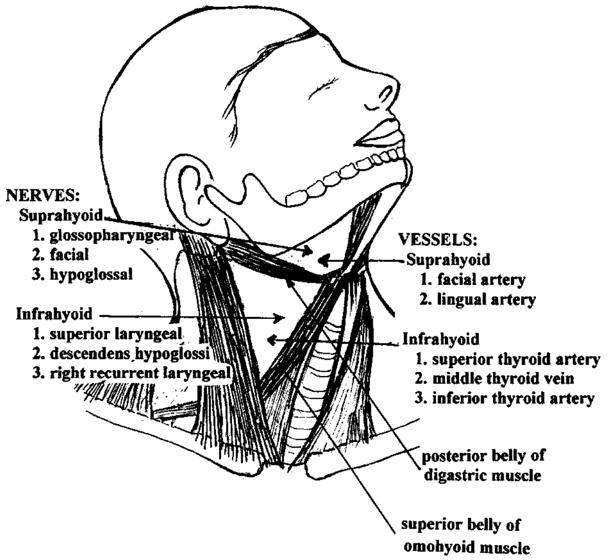

FIG. 2

The viscero-vertebral angle bisectors: nerves and vessels, and the hyoid suspenders.

are important for access to the viscero-vertebral angle above the hyoid bone, and the omohyoid muscle is important for access below the hyoid bone.[4] The glossopharyngeal, facial and hypoglossal nerves cross the viscerovertebral angle in the suprahyoid area, whereas the superior laryngeal, descendens hypoglossi and the right recurrent laryngeal nerves cross it in the infrahyoid area. The SLN descends on the sidewall of the pharynx deep to the internal carotid artery where it divides into internal sensory and external motor branches. The internal laryngeal nerve (ILN) descends with the superior laryngeal artery to pierce the thyrohyoid membrane at a point one cm anterior to the superior cornu and above the thyroid ala. The ELN descends parallel and deep to the superior thyroid artery on the inferior constrictor muscle to which it sends a branch. It passes deep to the sternothyroid to wind around the inferior thyroid tubercle to reach the cricothyroid muscle. There are five significant vessels that cross the viscero-vertebral angle, two above (the facial and the lingual arteries) and three below the hyoid level (the superior thyroid artery, the middle thyroid vein and the inferior thyroid artery). Thus, a certain logic to surgery in the cervical area can be formulated to assist the operator in attaining the goals of the surgical procedure by taking advantage of the anatomy and its developmental sequences, rather than being intimidated by it.[5]

It was our intent in this paper to present and evaluate a surgical technique for identification and preservation of the SLN during surgical dissection in the neck near the thyroid apex.

Patients and methods

During the period from March 3, 1995 to November 23, 1998, 78 patients underwent surgical dissection near the thyroid apex for various indications (Table I), in which the SLN and/or its branches were considered at risk during surgery. No subject had had previous neck surgery or irradiation; all had had normal pre-operative indirect laryngoscopy.

TABLE I

INDICATIONS AND TECHNIQUES OF NECK EXPLORATION

n	Indication	Technique of surgery
51	Benign thyroid nodule	Lobectomy and isthmectomy
11	Malignant thyroid disease	Total thyroidectomy
2	Supraglottic carcinoma (Stage II)	Supraglottic partial laryngectomy + functional neck dissection
2	Carotid paraganglioma	Transcervical subadventitial removal
2	External laryngocele	Transcervical removal
1	Lateral saccular cyst	Transcervical removal
3	UES dysphagia	UES myotomy
2	Pharyngeal pouch	UES myotomy + pouch surgery

UES = upper oesophageal sphincter.

After premedication with midazolam (1–2 mg intravenously), the patients were taken to the operating room, where they were monitored with an electrocardiogram, pulse oximetry, and non-invasive blood pressure measurement. General anaesthesia was induced with sodium thiopental (4–5 mg/kg) and fentanyl (3–5 μg/kg). Succinyl choline (1 mg/kg) or mivacurium (0.25 mg/kg) was used to facilitate endotracheal intubation. Anaesthesia was maintained with nitrous oxide/oxygen and isoflurane. No additional neuromuscular blocking agents were used. The lungs were ventilated mechanically, keeping the end-expired carbon dioxide in the range of 28–32 mmHg.

Exploration was carried out through a transverse cervical incision following a natural skin crease; the depth of the incision was subplatysmal where it remains superficial to the anterior jugular veins. The cutaneoplatysmal flaps were elevated; the topographical boundaries of the surgical field were the lateral extent of the hyoid bone above, the sternal notch below, and the lateral margins of the tendinous sternal origin of the sternocleidomastoid muscle laterally.

The medial margin of the sternocleidomastoid muscle was mobilized from its tendinous origin to the level of hyoid bone. The superior belly of the omohyoid muscle was mobilized from its tendon to its hyoid insertion, exposing the great neurovascular complex of the internal jugular vein, carotid arteries, vagus nerve and descendens hypoglossi nerve. The sternohyoid and sternothyroid muscles were also mobilized from their manubrial origins to their respective insertions into the hyoid bone and thyroid cartilage, while preserving their innervation from the descendens hypoglossi nerve, providing access to the cervical viscera composed of the larynx, trachea, pharynx, oesophagus, thyroid and parathyroid glands. Lateral retraction of the great neurovascular bundle and medial rotation of the cervical viscera opened the viscero-vertebral angle, aided by transection and haemostatic control of the middle thyroid vein. Crossing the angle are the superior

TABLE II
OPERATIVE COMPLICATIONS AMONG 74 PATIENTS UNDERGOING MOBILIZATION OF VISCEROVERTEBRAL ANGLE

Complication	n
Deaths	0
Returned to operating room for bleeding	1
Hypocalcaemia	
Transient	2
Permanent	0
Recurrent laryngeal nerve injury	0
Seroma	1
Post-operative atelectasis	1
Post-operative gastric bleeding ulcer	0
Post-operative voice change	4

thyroid vessels, the SLN with its internal and external branches, the middle thyroid vein, the inferior thyroid vessels, the right recurrent laryngeal nerve and the sternohyoid and sternothyroid branches of the descendens hypoglossi nerve. The surgeon then approached the dissection recognizing that what is to be resected depends largely upon what is found. During peri-apical surgical dissection, every effort was made for identification and preservation of the superior laryngeal nerve and its branches. The nerve was identified as it crossed the viscerovertebral angle medial to carotid artery bifurcation. Use of the nerve stimulator was not routine; in situations such as upper-pole enlargement, distortion by tumour or gland hypertrophy, enlarged pyramidal lobe and bleeding, a Hilger facial nerve stimulator, Model 2R (WR Medical Electronics Co. St. Paul, Minnesota) was used. Intra-operative identification of the SLN or its external branch was verified by watching the cricothyroid muscle contract after stimulation with the nerve stimulator probe. Stimulation intensities above 0.5 mA were avoided.[1] The remaining steps of the operation were performed in a routine way, always keeping the SLN and its branches under direct vision. At the end of the operation, the integrity of the nerve was verified with the nerve stimulator. Drains were used only in a large dead space, extensive neck dissection, large goitres and substernal goitre. Closure was performed in three layers.

Post-operatively, all patients were monitored in the recovery room before they were transferred to their hospital room; all patients were evaluated for haemorrhage, hypocalcaemia and airway distress. Patients were discharged from the hospital when their oral intake had stabilized and were evaluated for hypocalcaemia, seroma, wound infection, wound healing, recurrent nerve palsy and voice change.

Subjects were evaluated for superior laryngeal nerve injury three months post-operatively using a subjective interview, rigid laryngoscopy and acoustic analysis. Doubtful cases were referred to an outside institution for laryngeal videostroboscopy and electomyography of the cricothyroid muscle. Each subject was interviewed to assess for the presence of symptoms, including voice change, hoarseness, voice fatigue or pitch change. Data were stored in a computer database and analysed. Four patients were lost to follow-up and excluded from the study.

TABLE III
PRE-OPERATIVE AND POST-OPERATIVE GROUP MEANS FOR VOICE FUNDAMENTAL FREQUENCY (FO), VOCAL RANGE (VR), JITTER PERCENTAGE AND SHIMMER PERCENTAGE

	Pre-operative	Post-operative	*P* value
FO (Hz)	217	221	0.34
VR (Hz)	373	387	0.28
Jitter (%)	0.41	0.44	0.19
Shimmer (%)	1.21	1.07	0.23

Results

The study included 74 patients who underwent a neck exploration in which the superior laryngeal nerve and/or its branches were at risk during surgical dissection. The indications and techniques of surgery were shown in (Table I). The male-to-female ratio was 2:3. The age of the patients ranged from 26 to 67 years, with a mean of 43 years.

Eighty-five SLNs were investigated and all were identified during operation: 36 nerves with and 49 without the aid of the nerve stimulator. There were no operative deaths. One patient was returned to the operating room because of bleeding. Two patients developed transient hypocalcaemia requiring cholecalciferol and calcium supplementation for a period not greater than four months. One patient developed seroma three weeks post-operatively and required three fine-needle aspirations in the office before resolution. No permanent parathyroid or recurrent laryngeal nerve impairment was noted (Table II).

In the follow-up period, four patients complained of post-operative voice change. Two of them noted a persistent, mildly weak voice with decreased upper pitch range. Their acoustic analysis showed a deep fall in fundamental frequency (F0) and a marked reduction in vocal range (VR); statistical analysis of the pre-operative and post-operative vocal characteristics of the whole group, however, showed no significant differences (Table III). The two patients had had a papillary carcinoma that required total thyroidectomy. Rigid laryngoscopy demonstrated bowing of the affected vocal fold with ipsilateral rotation of the posterior glottis. Laryngeal videostroboscopy at an outside institution showed a bowed appearance and inferior displacement of the affected fold, and asymmetry of the travelling mucosal wave. Electromyography of the affected cricothyroid muscle demonstrated low-amplitude fibrillation potentials at rest and absent interference pattern with phonation of a high-pitched /i/. The other two had no objective evidence of nerve injury; one was diagnosed as reflux laryngitis and improved with a proton pump inhibitor and the other had intubation granuloma.

TABLE IV
POST-THYROIDECTOMY SUPERIOR LARYNGEAL NERVE INJURY IN THE LITERATURE

Authors	Surgical technique	Evaluation method	Results	Incidence
Reeve *et al.*[18]	Expose nerve to prevent damage	Laryngoscopy Voice assessment	Total: 100 patients with 157 nerves; 92 nerves were identified. Three patients with voice changes.	3%
Kark *et al.*[19]	Identify and preserve the nerve	Questionnaire Oscilloscopy Laryngoscopy	Total: 38 patients with 56 nerves; 47 nerves were identified. Two patients with permanent voice changes.	5%
Lekacos *et al.*[20]	Separate ligation of superior pole vessels; no exposure of the nerve	Indirect laryngoscopy	Total: 122 patients with 22 nerves. None with nerve injury.	0%
Lennquist *et al.*[10]	Visualize the nerve whenever possible	Indirect laryngoscopy	Total: 38 patients with 50 nerves; 36 nerves were identified. One patient with permanent voice change.	2.6%
Cernea *et al.*[21]	Identify the nerve with nerve stimulator near the pole.	Indirect laryngoscopy EMG Acoustic analysis	Total: 26 patients, nerve was identified in 24. No patient with nerve injury.	0%
Teitelbaum and Wenig[6]	Separate ligation of superior pole vessels. Searching the nerve is not routine.	Subjective interview EMG Videostroboscopy	Total: 20 patients with 26 nerves. One patient with nerve injury.	5%
Loré *et al.*[7]	Separate ligation of superior pole vessels. Not necessary to expose the nerve.	Subjective interview Laryngoscopy	Total: 66 patients with 96 nerves. Five patients with permanent voice change.	7.5%
This study	Identify and preserve the nerve with nerve stimulator in difficult situations	Subjective interview, rigid laryngoscopy, acoustic analysis, EMG, laryngeal videostroboscopy.	Total: 74 patients with 85 nerves; 36 nerves were identified with the aid of the nerve stimulator and 49 without. Two patients with permanent voice change.	2.4%

EMG = electromyelography.

Discussion

Injury to the recurrent laryngeal nerve is a well recognized complication of thyroid surgery. When it occurs, it is easily detected by indirect laryngoscopy. As a result, the incidence of this injury is well documented, ranging from 0.3 to 13 per cent.[6] On the other hand, injury to the SLN is difficult to detect, and may be frequently overlooked. Consequently, the incidence of injury to the SLN during surgery of the thyroid is poorly documented in the literature (Table IV). To enhance the diagnostic ability of the otolaryngologist with respect to SLN injury, laryngoscopic examination was combined in this study with detailed subjective voice evaluation and acoustic analysis. To indict the superior laryngeal nerve, all other causes of voice changes must be eliminated, for example, recurrent laryngeal nerve injury, endotracheal intubation (e.g. mucosal lesions, arytenoid dislocations) as well as any local mucosal disease of the vocal folds including that caused by hypothyroidism, ageing or gastroesophageal reflex.[7] Patients with positive voice changes were evaluated by laryngeal videostroboscopy and cricothyroid electromyography. Only two patients (two nerves out of 85 SLNs) showed an asymmetrical mucosal travelling wave, inferior displacement and bowing of the affected vocal fold and ipsilateral posterior glottic rotation on laryngeal videostroboscopy, suggesting SLN injury.[8] The same two patients also demonstrated low-amplitude fibrillation potentials at rest and an absent interference pattern with phonation of a high pitched /i/ on electromyography of the affected cricothyroid muscle. These findings were also suggestive of cricothyroid muscle paralysis.[9] The remaining two cases were found to be due to intubation granuloma and gastroesophageal reflux, and improved after appropriate therapy.

Because there is no known effective treatment for this injury, prevention is extremely important.[6] Methods that have been advocated to preserve the SLN and its branches include skeletonization and individual ligation of the superior thyroid pole vessels adjacent to the capsule of the gland,[7] identification of the nerve prior to ligation at the superior thyroid pole,[10] use of a nerve stimulator,[11,12] or an intra-operative nerve monitor.[1,2] Most of the previous studies emphasized identification of the nerve at the upper pole of the thyroid gland, where the topography of the nerve shows much more variability in its relationship to the superior thyroid artery. Therefore, it seems easier to identify the nerve further cranially.[13] In our study, the nerve was identified in all cases as it crosses the viscerovertebral angle in the infrahyoid area, below the posterior belly of the digastric muscle. Moreover, mobilization of the angle displayed a wide surgical field and exposed structures for ease of access to targeted surgical objective.[5]

To our knowledge, this is the first study to assess the presence of SLN injury in a group of patients who had undergone surgical dissection in the neck near the thyroid apex for various indications. It is the intimate anatomical relationship of the ELN with the superior thyroid artery which predisposes the nerve to iatrogenic injury during thyroid surgery. Sixty-two patients underwent neck exploration for manage-

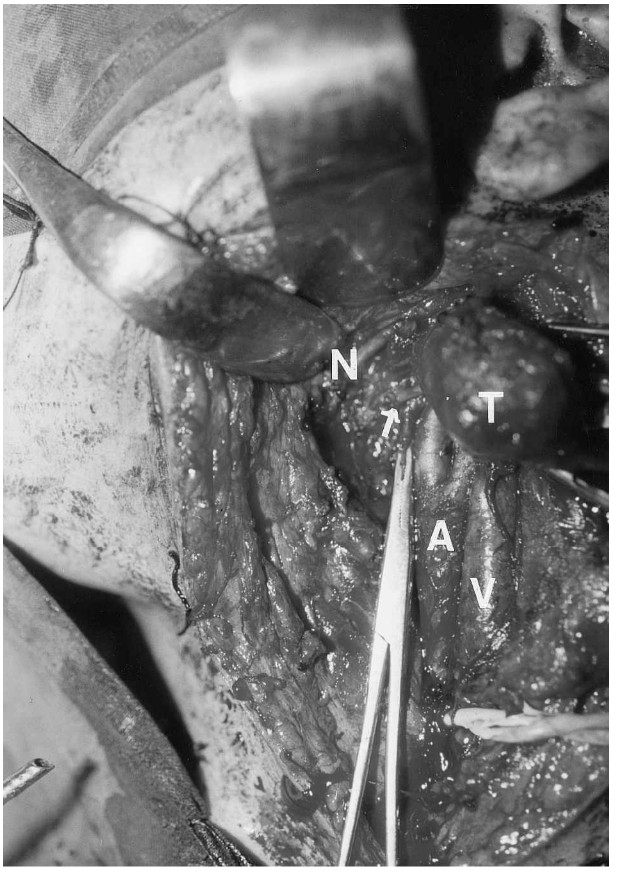

FIG. 3

A photograph demonstrating dissection exposure of the superior laryngeal nerve (arrow) during removal of a left carotid body tumour (T). N = hypoglossal nerve, A = common carotid artery, V = internal jugular vein.

ment of thyroid lesions; 51 with benign thyroid nodules were treated with total lobectomy and isthmectomy, and 11 with thyroid malignancy were managed by total thyroidectomy. The high points of the surgical technique were the superior pole exposure being the last step in thyroidectomy, and the vessels being separately ligated and divided as close to the thyroid capsule as feasible. Clearing of the vessels in the critical area was assisted by the nerve stimulator to identify and expose the nerve, especially in upper pole enlargement, distortion by a tumour and bleeding. ELN injury was encountered in two patients with malignant thyroid who underwent total thyroidectomy. The incidence (2.4 per cent) compares favourably with those obtained in other series (Table III).

Conservation laryngeal surgery effectively controls laryngeal carcinoma. The success of conservation laryngeal surgery is judged by the patient's ability to achieve physiological respiration, phonation and deglutition.[14] A mobile arytenoid is necessary for adequate speech and swallowing after partial laryngectomy. The mobility of the arytenoid was assured by avoiding injury to RLN during disarticulation of the cricothyroid joint. The arytenoid and adjacent pyriform sinus mucosa should be also preserved with its sensory innervation, the posterior descending branch of ILN, to avoid postoperative aspiration and dysphagia.[15] We routinely preserved the main trunk and posterior descending branch of the internal division of the SLN in supraglottic laryngectomy by identification of the main trunk in the viscero-vertebral angle medial to the carotid bifurcation. The posterior descending branch of the ILN was preserved during tumour resection by leaving it attached to the previously elevated, preserved pyriform sinus mucosa. Bilateral preservation of SLN is an important factor in the successful swallowing rehabilitation after partial laryngectomies, because the ability to sense the food bolus with the preserved mucosa improves deglutition. The SLN may also provide some motor fibres to the cricoarytenoid unit, and therefore its preservation may have an impact on airway closure and speech, as well as sensation.[14]

Three patients underwent a lateral cervical approach to the thyrohyoid membrane to remove an external laryngocele (two cases) and a lateral saccular cyst (one case). The saccular cysts are similar to laryngoceles in that they represent an abnormal dilatation or herniation of the saccule; however, they are distinct from laryngoceles in that there is no opening to the ventricle of the larynx and they are mucus-filled.[16] During operation, the sternocleidomastoid muscle and the major neurovascular bundle were retracted to mobilize the viscerovertebral angle. The sac was identified and dissected, with care taken at this point to avoid injury to the ILN. At the close of the operation, the laryngeal sensation was tested by directly touching the laryngeal mucosa to elicit a cough reflex. All three cases had an intact nerve.

The presenting symptom of the carotid body tumour was a neck mass located at the bifurcation of the common carotid artery. Contrast-enhanced CT clearly demonstrated a mass expanding the carotid bifurcation. Arteriography displayed the characteristic findings of a carotid paraganglioma: a vascular mass in the bifurcation of the carotid artery, and bowing and displacement ('lyre' sign) of the internal and external carotid arteries. The operation was performed by a joint team including an otolaryngologist and a vascular surgeon. Surgical excision required good exposure through mobilization of the viscero-vertebral angle with proximal and distal control of the cartoid artery. Every effort was made to identify and preserve the vagus, hypoglossal, spinal accessory and superior laryngeal nerves (Figure 3). No patient developed cranial nerve palsy, baroreceptor failure or first-bite pain.

Five patients underwent myotomy of the upper oesophageal sphincter (UES). The myotomy was indicated because the UES was not sufficiently relaxed during deglutition, resulting in dysphagia. In two of these patients, the myotomy was associated with pouch surgery. The viscerovertebral space was reached and the SLN was identified and preserved. In order not to damage the pharyngeal and oesophageal nerve supply, a dorsomedial myotomy was performed. The divided muscle border was sutured on itself or to the longus colli muscle to prevent recurrence.[17] No patient developed SLN injury or fistula.

Conclusion

The use of consistent anatomical landmarks and the mobilization of the viscero-vertebral angle facilitate localization of the superior laryngeal nerve. This straightforward approach provides a reproducible methodology to improve the chances of identifying and saving the nerve during head and neck surgery. The use of a nerve stimulator during difficult situations should keep SLN injury at a minimum. Moreover, this approach displays a wide surgical field and exposes structures for ease of access to targeted surgical objective.

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