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Recurrent Laryngeal Nerve Damage and Phonetic Modifications after Total Thyroidectomy: Surgical Malpractice Only or Predictable Sequence?

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Abstract. Modifications of phonation occurring after total thyroidectomy (TT) are usually attributed to surgical malpractice, but other causes of voice impairment even in nonoperated subjects should also be taken into account. This study analyzes 208 patients who underwent TT from January 1, 1999 through December 31, 2001. Follow-up ended on December 31, 2003. Only cases in which the surgeon ruled out the possibility of operative damage to the laryngeal nerves were included. All patients underwent pre- and postoperative clinical and instrumental nose and throat examination (NTE). Preoperatively, 86 patients (41%) showed hoarseness or dysphagia: 4 (2%) monoplegia and 12 (6%) hypomobility of the vocal cords due to impaired function of the recurrent laryngeal nerve (RLN); 6 (3%) cord hypotonia due to impairment of the superior laryngeal nerve (SLN); 34 (16%) dysphagia: and 30 (14%) hoarseness due to other causes. At follow-up 1 month after surgery, 71 patients (34%) had an onset of previously absent signs and symptoms: 8 (4%) had palsy of one vocal cord (2% permanent); 6 (3%) had cord hypomobility (all temporary); 12 (6%) had cord hypotonia due to disease of the SLN, 4 of which (2%) were permanent; 44 patients (21%) had symptoms due to scarring and adhesions between the laryngotracheal axis and the prethyroid muscles and between these and the skin. One patient (0.5%) had a nodular cord lesion that occurred after 3 months. Overall, more than one-third of the patients had preoperative voice modifications or swallowing impairment, around one-third had these problems after TT, and less than one-third were free of pre- and postoperative complications. The surgeon's care to avoid damage to the anatomica integrity of the of laryngeal nerves does not exclude functional problems of the nerves and of laryngeal dynamics. In fact, such problems could be referred to outcomes linked to the operation itself (hematoma, edema, scarring adhesion) or to events that only temporarily follow surgery but must be considered as an unavoidable sequel (e.g., neuritis, viral neuritis, myopathy). The patient should undergo a careful clinical and instrumental NTE to detect conditions prior to surgery, and the information provided by the surgeons should be thorough to allow the patient to be aware of all possible sequels and consequences.

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Total thyroidectomy (TT), a surgical procedure that must be performed with particular care, requires skillful surgical experience. Such experience is necessary but not sufficient to avoid complications that, though limited, may affect the social life of the patient when they involve temporary or definitive injuries of the laryngeal nerves and phonation modifications.

The patient often relates voice impairment temporally with the thyroidectomy procedure as a complication that is unavoidably due to surgical malpractice and rarely, instead, as a possible result of the operation. We have carried out a study to evaluate the preoperative and postoperative incidence of pathology of the laryngeal nerves, enabling us also to recognize causes other than surgically induced anatomic damage.

Methods

A series of 208 patients who underwent total thyroidectomy from January 1, 1999 through December 31, 2001 by a single surgeon of skilled experience in the field of endocrine surgery were assessed. The only selection criteria evaluated by the operating surgeon was the absence of iatrogenic injuries to the anatomic integrity of the recurrent laryngeal nerve (RLN) detectable during surgery. The correctness of the surgical indication, the surgical technique employed, and the postoperative course were examined by two independent, skilled surgeons with full access to the clinical documentation. The 208 evaluated cases were subdivided as follow.

- 193 (93%) patients suffering from benignant pathologies: diffuse in 11 (5%) cases and nodular in 182 cases (95%)
- 15 (7%) people suffering from malignant pathology: differentiated in 12 (88%) cases, medullary in 1 (6%), and undifferentiated in 1 (6%).
- 33 (16%) retrosternal goiters (when the neck was stretched the goiter was plunged into the chest to a depth of at least four transverse fingers below the sternal notch)

There were 175 (84%) females and 33 (16%) males, with a female/male ratio of 5.3:1.0. The mean age was 53 years, and the mean hospital stay was 3.5 days.

All patients underwent extracapsular TT; and in two cases of papillary neoplasia with monolateral lymphnode metastases, we combined it with modified neck dissection (MND). The RLN was always exposed as far as its inlet below the cricopharyngeal muscle, leaving the posterior part of it adherent to the connective tissue that protects and vascularizes it. The sternohyoid muscles were dissected but never resected, whereas the sternothyroid and the thyrohyoid muscles were mobilized from the thyroid to allow ligation of the superior gland pedicle, with special care taken to not injure the external branch of the superior laryngeal nerve (SLN).

At enrollment we obtained the medical history mainly aimed at phonation and respiratory and swallowing functions. Thereafter we submitted all patients before surgery and again 1 month later to the following examinations.

1. Nose and throat clinical examination (NTE) was completed as well as basal laryngeal video-endoscopy (eventually sensitized by means of a stain (e.g., methylene blue mint syrup) in case of swallowing troubles).
2. Logopedic examination and acoustic analysis of the voice by means of Speech Viewer II (SPV2)—IBM Independence Series. SPV2 exploits high-resolution graphic and the high reproduction sound capability of computers, allowing an audio-video representation of the characteristics of sound and verbal production. In detail we monitored the tone pitch, sound intensity, sound emission, timing, and spectrum. Voice examination by means of SPV2 was preferred to video-stroboscopy because it is better borne by patients and because it is the interactive instrument that we employ to assist logopedic reeducation in case of damage.

Clinically symptomatic patients underwent the NTE evaluation immediately after surgery to establish, when needed, early logopedic rehabilitation to order to prevent inadequate spontaneous compensation.

For each patient we had video and audio recordings available and a data file that pointed out following.

- Presence of possible respiratory pathology and bone-muscle deformities
- Previous operations on the neck or the laryngeal and tracheal structures
- Preoperative and postoperative nose and throat examination
- Voice and swallowing impairment before and after TT surgery, related (or not) to disease of the inferior and superior laryngeal nerve
- Notes concerning the surgical procedure
- Postoperative complications

The follow-up was ended on December 31, 2003.

Results

During the prehospitalization period all 208 patients underwent nose and throat control, logopedic examination, and acoustic voice analysis by means of SPV2. The results showed the following (Fig. 1). There were 28 (13%) patients who were symptomatic for phonation impairment, 8 of whom had a negative

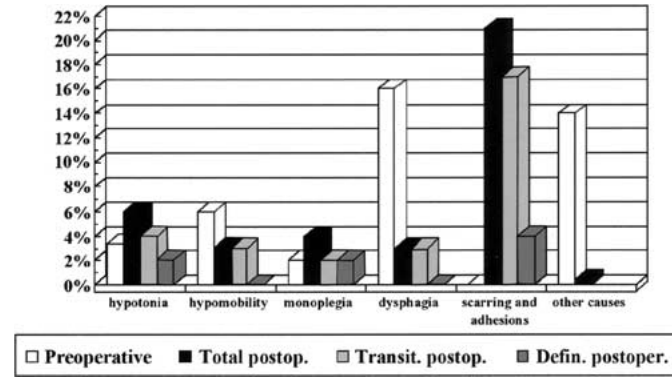

Fig. 1. Preoperatively and postoperatively detected vocal alterations. Postop: postoperative; Transit:transitional; Defin.:definitive.

NTE (symptoms were attributed to pulmonary deficit or to functional compressive-like laryngotracheal factors), the other 20 had an abnormal NTE report: 6 cases were cord, myxedema or Reinke's edema, 2 cord nodules, 2 cases of incoordination, 4 (2%) cord hypotonia, 4 (2%) cord hypomobility, and 2 (1%) monoplegia. Of the 180 patients who were asymptomatic for phonation impairment, 24 (12%) showed NTE pathologic objectivity; 2 cases of cord hyperemia with gastroesophageal reflux, 4 cases of cord myxedema or Reinke's edema, 4 chronic laryngitis, 2 cases of incoordination, 2 cord hypotonia, 8 cord hypomobility, and 2 monoplegia that were well compensated by the contralateral cord. Another 34 (16%) patients also had a swallowing impairment, such as a feeling of bolus block and occasional inhalation. In 19 cases the ear-nose-throat (ENT) examination, video-endoscopic investigation, and voice analysis were all negative, whereas in 16 they were combined: 2 cases of cord hyperemia with gastrosophageal reflux disease, 4 cord hypotonia, 2 incoordination, 4 cord hypomobility and 4 monoplegia.

Approximately 1 month after TT, all 208 subjects underwent, as outpatients, a nose and throat check with combined logopedic examination and acoustic voice analysis by means of SPV2. Among them, 71 (34%) showed an onset of swallowing and voice impairment that were previously absent, including hoarse voice, whispery voice, diplophonic voice, falsetto or changes of timbre, tone, extension, intensity, vocal fatigue, and difficulty with the singing voice as well.

We have noted (Fig. 1) eight patients (4%) with vocal cord monoplegia: 6 at the right (75%) and 2 at the left (25%). Four (2%) were temporary (two of the right cord and two of the left one) and were cured within 90 days with complete remission of the dysphonic symptoms. Four (2%) were definitive (they had not recovered a year after detection). When follow-up was completed, two had had a complete remission of the dysphonic symptoms within a year with good compensation of the contralateral cord, even though the palsy persisted, whereas in two (1%) dysphonia resulted in permanent changes of the timbre and intensity.

We also found six patients 4 (3%) with cord hypomobility, which completely receded within 90 days. There were 12 cases (6%) of cord hypotonia due to SLN impairment with voice modification, easy fatigue during phonation, and difficulty with high pitch and singing voice. Eight (4%) of the twelve had a spontaneous recovery within 40 days and four (2%) had permanent dysfunction because the problem did not recede after 1 year (Fig. 2).

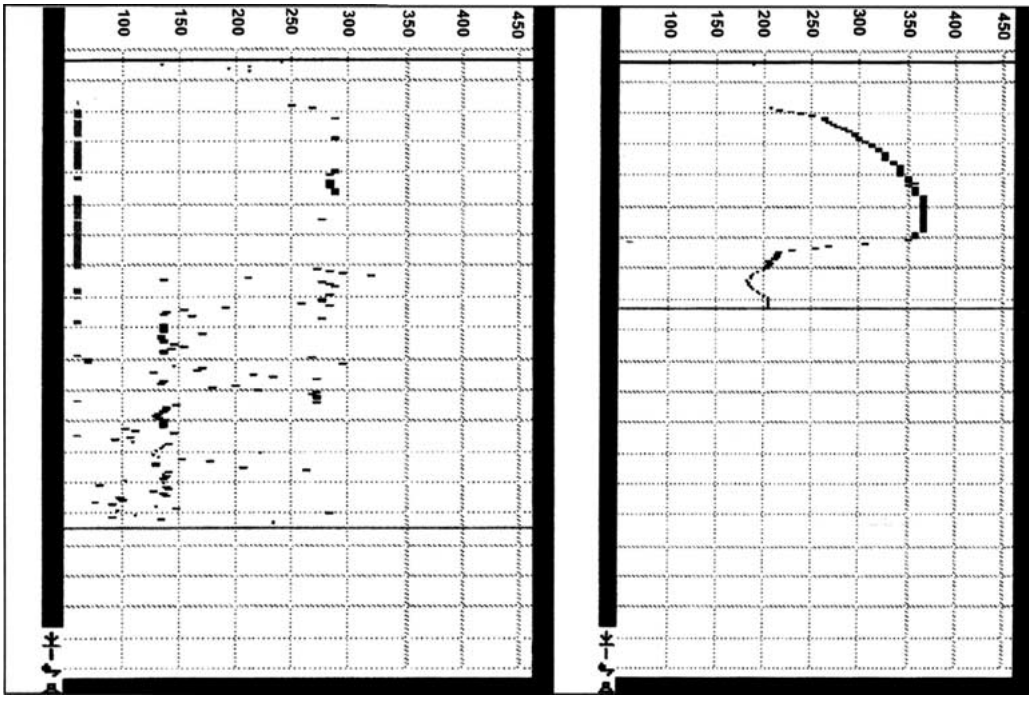

Fig. 2. Superior laryngeal nerve (SLN) deficit: acoustic analysis. **A**. Vocalization difficulty that starts almost normally but shows a precocious decrease. **B.** Impossible to vocalize.

In get another patient a nodular lesion appeared after 3 months. Finally, 44 patients (21%) had voice impairment due to adhesions between the laryngotracheal axis and the prethyroidal muscles plus scarring between these and the skin with partial impairment of laryngeal vertical movement. This situation had manifested as a feeling of obstructed swallowing and changes in laryngeal dynamics with modification of the vocal tone and timbre without detection of cord alterations. Of the 44 patients, 36 recovered within 90 days and 8 (4%) had a permanent disability. In these cases, the clinical phonation symptoms almost overlapped the signs and symptoms attributed to SLN damage but with a different outline on the acoustic analysis.

All patients with monoplegia, hypomobility and cordal hypotonia underwent early speech therapy. Six of the eihgt patients with cordal monoplegia (3%) complained of swallowing impairment, which receded within 30 days. We did not observe any patients with cord diplegia. Of the 34 patients with preoperative swallowing impairment, 10 reported symptom remission within 2 months after surgery, whereas 24 reported no not record any variation. Of the 28 patients who showed preoperative phonation hindrance, 8 had symptom remission within 60 days after surgery, whereas 20 reported no improvement.

Disussion

Surgical therapy deeply worries patients suffering from thyroid disease because of possible persisting voice alteration (ranging from moderate and almost imperceptible timbre and tone changes to severe dysphonia) and the fear of tracheotomy. Both permanent and transient lesions of the RLN have an important impact on the patient's social life, and such lesions have been reported by various authors with rates that range from an unlikely 0% [1, 2] to a perhaps exaggerated 10% [3–6]. An Italian multicenter study [7] reported that after TT the total incidence of RLN lesions was 4.3%, with 2.4% transient and 1.3% permanent. Altogether, 0.6% were bilateral lesions, with half of them (0.3%) undergoing tracheotomy. In contrast, lesions of the external branch of the SLN are more difficult to evaluate and are often undervalued, but their reported incidence is around 10% [8, 9].

It is almost impossible to perform a meta-analysis of the lesions of the laryngeal nerves after TT because of the difficulty of comparing figures from different studies. As a matter of fact, most studies do not include preoperative investigations; preoperative instrumental examinations are not performed as a routine; and only recently has attention been focused on SLN injuries. Furthermore, there is often some misunderstanding regarding the terminology applied to impaired motility; therefore hypotonia, hypomobility, and damage are frequently not taken into consideration.

Our study emphasizes that (Fig. 3) preoperativery more than one-third of patients (86/208) show phonation and deglutition problems; 8% are due to damage to the RLN (6% cord hypomobility, 2% monoplegia), 3% are due to deficit of the SLN (hypotonia), and 16%, are due to swallowing problems.

After TT, around one-third (71/208) of patients complain about the onset of phonation and swallowing problems that were previously absent: 7% are due to RLN damage (3% cord hypomobility, 4% monoplegia), 6% are due to a deficit of the SLN (hypotonia), and 21% are due to scarring and adhesion formation (caused by modifications to and laryngeal dynamics and the changed anatomic conditions). Finally, fewer than one-third of patients are free from preoperative and postoperative complications.

The transient (2%) or definitive (2%) injuries to the RLN after TT occurred even if the surgeon had technically exposed the nerve and followed it carefully as far as its inlet below the pharyngeal constrictor muscle. Mainly, he had not noted any intraoperative complication that could aronse the suspicion of an

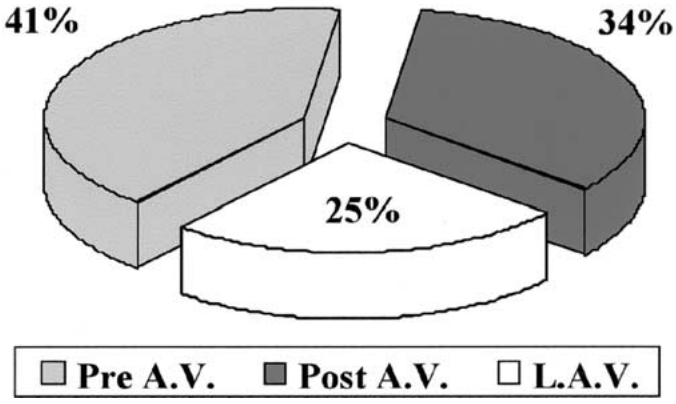

Fig. 3. Vocal alterations, Pre A.V.: preoperatively detected vocal alterations; Post A.V.: postoperative detected vocal alterations; L.A.V.: lack of vocal alterations.

injury to the nerve's anatomic integrity. All cord hypo mobility was transient, and 75% of the monoplegia cases (six cases) affected the right RLN.

Knowledge of the surgical anatomy of the neck, thyroid illnesses, and required surgical treatments is essential to keep the complication rate within reasonable limits [10]. The surgeon must strictly comply with some technical rules:

(1) The accessory laryngeal musculature must not be interrupted unless absolutely necessary. This reduces adhesions with the pharynx and the trachea [11], and an undamaged musculature plays an important role in cases of logopedic rehabilitation [12]. (2) The RLN must be identified and exposed [13] not only in its beginning tract in the mediastinum but along its entire course up to the inlet of the lower pharyngeal constrictor muscle (crico pharyngeal muscle). In fact, this represents the most critical site where the anatomic integrity of the nerve might be damaged as it frequently divides into thin branches, only one of which provides innervation to the vocal cord [14]. The nerve must not be detached on its posterior part from the connective tissue that protects and vascularizes it. (3) to avoid lesions to the external branch of the SLN it is necessary to stretch the superior pole of the thyroid laterally, opening a space between it and the aerial axis and ligating the derivation branches of the superior thyroid artery behind the gland tissue [10]. Persisting in an anatomic search might result in damage of the nerve [15]. (4) Use of a monopolar electrocoagulator as well as bipolar, radiofrequency, and ultrasound (Harmonic Scalpel) electrocoagulators must be banned behind the laryngeal nerves. (5) To avoid dangerous haemostatic manereuvms it is preferable to ligate the small retroneural arterial vessel near the nerve entry under the cricopharyngeal muscle. (6) Care must be exerted, especially on the right side, to avoid a nonrecurrent inferior laryngeal nerve, which is present in about 1% of the cases [16].

These technical indications that we have described are mandatory, although voice alteration might still arise even with a perfectly performed TT procedure. Such alteration might be linked (or not) to injury to the RLN nerve or the external branch of the SLN. It can sometimes be detected by means of a thorough NTE examination and may be not be due to iatrogenic injuries, even though it may be transiently linked to the surgical procedure itself [17].

Table 1. Causes of RLN damage.

Surgical causes linked to direct lesions for technical mistakes
Damage to the anatomic nerve integrity
Thermal lesions by diathermy
Surgical causes linked to particular anatomopathologic conditions
Excessive nerve skeletization (the nerve adheres to the thyroid capsule)
Axon damage caused by excessive strain (voluminous cervicomediastinal goiter; nerve adhering to the thyroid capsule)
Hematoma
Edema
Nonsurgical causes linked to intubation maneuvers
After difficult orotracheal intubation paralysis
Nonsurgical causes linked to operation
Neuritis (caused by scar tissue, myelin lesion)
"a frigore" or "a calore" paralysis
Viral neuritis
Myopathy

RLN: recurrent laryngeal nerve.

Table 2. Causes of damage of the SLN external branch

Surgical causes linked to direct lesions for technical mistakes
Damage to the anatomic nerve integrity
Thermal lesions due to diathermy
Surgical causes linked to particular anatomopathologic conditions
Excessive strain for retraction of the superior vascular pedincle after section (when the external branch is among the dissection branches of the superior thyroid artery)
Axon damage caused by the superior pedicle adhering to the larynx
Hematoma
Edema
Nonsurgical causes linked to intubation maneuvers
After difficult orotracheal intubation paralysis
Nonsurgical causes linked to operation
Neuritis (caused by scar tissue, myelin lesion)
"a frigore" or "a calore" paralysis
Viral neuritis
Myopathy

SLN: superior laryngeal nerve

A remarkable percentage of patients (46% [18], and in our study 41%) complain of laryngeal disease, with NTE assessment of severe voice complications, without having undergone any neck surgery. This shows that there are many factors that may cause alteration of vocal cord motility: laryngeal disease combined with gastroesophageal reflux disease (GERD); chronic laryngitis; functional dysphonia, cord edema; nodular pathology; and pathology that involves laryngeal nerves (neuritis, viral neuritis, myopathy) [9, 17, 18]. To safeguard the anatomic integrity of the laryngeal nerves does not exclude the onset of functional troubles linked to pathologic outcomes that frequently occur spontaneously and that might originate from or be induced by surgical manipulation of the thyroid cavity. In addition, so far as operated patients are concerned, we must take into consideration not only the causes that are directly linked to surgical treatment, such as traumatic lesions of the laryngeal nerves, hematoma, and edema, but those related to difficult intubation [19] (Tables 1, 2).

Whenever voice alterations occur in a patient who has undergone TT, especially if they are linked to altered motility of the vocal cords, they are said to be caused surgical malpractice, even if they are transient or arise months or years after surgery. On the other hand, once the operation is ended, it becomes practically impossible for the surgeon to demonstrate the anatomic integrity of the nerves or to cite any other cause that cannot be proven

eventhough it is possible or likely. Some intraoperative devices have been suggested for demonstrating the integrity of the laryngeal nerves: RLN intraoperative monitoring by an electrode inserted in the endotracheal tube [20], indirect stimulation via the vagal nerve, direct RLN [21] computer-assisted evoked electromyography with stimulating surgical instruments [22]. At present, this seems more suitable for experimental use than for clinical employment.

To avoid not diagnosisng preoperative injuries that might be attributed later to the surgical procedure, it is crucial that all patients who undergo surgery for thyroid or parathyroid pathology be submitted to careful clinical and instrumental nose and throat examination [23] with optical fibroscopy of the vocal cords and audio and video taping of eventual recorded changes.

Finally, we must stress that in our study 18 patients who had preoperative problems (21%) no longer had those complications after surgery, and that in 44 of 71 (62%) patients who had had symptoms after surgery the symptoms were due to modifications of the laryngeal dynamics and to adhesions between the laryngotracheal axis and the prethyroid muscles and between these and the skin.

The importance of phonation complications and the ensuing controversies must force the surgeon to inform the patient thoroughly about the surgical indications, therapeutic choices, the benefits expected after surgical therapy, and the procedure's risks, including transient and permanent injuries to the vocal cords and the possibility of permanent voice alteration [12, 24]. The surgeon must properly explain these factors, and the patient must understand that they are not exceptional events linked solely to the technical result of the procedure. They must be considered as possible, predictable consequences.

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