RLN (e.g. crush, stretch or ischemic injuries) can damage the myelin sheath (axonotomesis) which can still recover spontaneously but can lead to misdirected fiber regrowth and vocal cord muscular contractions that are poorly coordinated with altered glottic function (termed [illegible]).[6] More complete trauma to RLN (e.g. laceration or severe crush or stretch injuries) leads to interruption of the endoneurial, perineurial and/or epineurial sheaths (neurotmesis), which is followed by incomplete or absent nerve regrowth and permanent vocal cord paralysis (Table 1).[7]

Unilateral vocal cord (VC) paralysis symptoms vary between patients and within a given patient over time. On fiberoptic laryngoscopy, the vocal fold usually remains in the paramedian position initially, allowing for a fairly normal voice with vocal fatigue and minor pitch changes. Despite VC paralysis voice changes may be initially offset by VC edema brought on by intubation and may not present definitively for several days. With time, the paralyzed vocal fold will atrophy causing the voice to worsen. Over time the vocal cord position can evolve to either more lateral (with voice worsening) or more medial (with vocal improvement). Another reason for symptomatic variability overtime is the amount of contralateral VC compensation which also varies between patients and in the same patient over time. A final variable in VC position is the degree of injury and the timeframe of recovery if this occurs. The glottal air leak during phonation typically causes a breathy-weakened voice rather than vocal coarseness generically termed hoarseness.

Bilateral vocal fold paralysis usually presents acutely after extubation. Both vocal folds remain in the paramedian position causing partial airway obstruction. The patient may have biphasic stridor and be in respiratory distress. Occasionally, a patient will not have airway symptoms in the immediate postoperative period (because the airway is sufficient despite the paralyzed vocal folds) and the patient may present at a follow-up visit complaining of shortness of breath or stridor with exertion as the vocal cord position evolves to its final paralytic position.

EXTERNAL BRANCH OF SUPERIOR LARYNGEAL NERVE INJURY

The external branch of the superior laryngeal nerve (EBSLN) provides motor function to the cricothyroid muscle, whose contraction slides the upper laryngeal cartilage (i.e. thyroid cartilage) forward relative to the lower laryngeal cartilage (i.e. the cricoid cartilage) and is important in adjusting the vocal fold length and tension. As the EBSLN is intimately related to the superior thyroid artery at the upper pole, one ligates the terminal branches of the superior thyroid artery as close to the thyroid capsule as possible to avoid damaging this nerve.

Despite the fact that most thyroid surgeons are familiar with the story of the famous opera singer Amelita Galla-Curci and her career-ending voice compromise after thyroid surgery, survey made in different professional organizations suggest few surgeons make an effort to identify this nerve.

Trauma to EBSLN results in an inability to lengthen the vocal fold, and therefore, to create a higher pitched sound and vocal projection. The clinical presentation may be quite subtle in many patients with increased tendency for vocal fatigue and decreased pitch range being the most common symptoms. For the singer or the professional voice user however, paralysis of the EBSLN may be career threatening by the loss of the upper register of the voice.

It is very difficult to diagnose EBSLN injury on indirect or fiberoptic laryngoscopy. Posterior glottic rotation toward the paretic side, bowing of the vocal fold on the weak side and inferior displacement of the affected cord can be very subtle and easily missed. The diagnosis EBSLN paralysis is facilitated by the use of videostroboscopy and most confirmed through laryngeal EMG of the cricothyroid muscle (available only in specialized voice laboratories). Due to these diagnostic difficulties limitations, the reported incidence of injury to the EBSLN varies widely from 0-25%.[8]

VOICE AND THE GLOTTIC EXAM: AN OVERVIEW

It is centrally important to recognize that in both preoperative and postoperative settings, VC paralysis may be present without significant vocal symptoms. This basic fact is the rationale for the inclusion of glottic exam in all patients both preoperative and postoperative.

The divergence of glottic function on laryngeal exam and voice symptoms is due to several factors including variable remaining cordal function, variability in paralytic cord position and variability of contralateral cord compensation. It is common to observe improvement in symptoms due to better contralateral cord compensation which could be falsely interpreted by the less experienced observer as an indication that VC paralysis has resolved. Without glottic exam this scenario may result in a sober re-evaluation of glottic function if contralateral surgery goes wrong at some point in the future. The correlate is also true that hoarseness may derive from many sources and does not necessarily imply VC paralysis. It is only the laryngeal exam that can accurately indentify VC paralysis.

REPORTED INCIDENCE OF RLN PARALYSIS AND OF LARYNGEAL EXAM

The reported incidence of RLN injuries varies widely between 0 and 30% (reviewed in).[9] An important step forward in understanding the real incidence of voice symptoms and RLN palsy rate has been the development of nation-wide audit databases. The Scandinavian quality register reported 2359 thyroid operations performed within one year lead to an immediate (i.e. within 6 weeks) RLN palsy in 3.1% of nerves at risk and this rate dropped to 1.7% at 6-months.[10] The [illegible] ' audit recorded 55 patients with new RLN palsies from 2454 cases of first-time thyroid surgery (2.1%) while 155 patients experienced voice changes (5.6%). After 344 cases of [illegible] thyroid surgery

the reported rate of RLN palsy was 3.9% and voice changes occurred in 8.1%.[11] It is important to emphasise that both the Scandinavian and British quality registers derive from surgeon-reported cases without routine postoperative laryngeal exam. Furthermore, many of those who report these cases use postoperative laryngoscopy only in patients with persistent/ severe voice changes. In this context, the rates of temporary and permanent RLN palsy are deemed to be severely underestimated. This view was confirmed by a recent review of 27 articles and 25,000 patients, in which the RLNP rate varied 10 fold according to the method of examining the larynx and ranged from 26 to 2.3%.[12]

VOICE CHANGES IN PATIENTS WITH INTACT VOCAL FOLD MOTILITY

Over a decade ago, several groups reported that postoperative voice changes can occur in patients with intact vocal fold motility. Typical symptoms are often transient and consist of voice fatigue during phonation and difficulty with high pitch and singing voice due to a decrease in the speaking fundamental frequency and vocal range.[13] The speech becomes more monotone and vocal pitch can be more than two semitones lower[14] but there is progressive normalization within three months postoperatively.[15]

In a prospective nonrandomized study of 100 patients, subjective voice changes occurred in one third of patients all of whom had normal VC motion postoperatively.[16] Similarly, during a prospective single-arm study of 54 patients, 30% of patients reported early subjective voice changes and 14% reported late (3-month) changes.[17] A higher incidence of 50% was found in a larger study of 400 patients but such problems resolved within 6 months in 85% of patients and within 1 year in 98% of them.[18] In two large series of post-thyroidecotmy patients without VC paralysis studied with modern laryngeal lab testing have shown that nearly 80% have subjective voice complaints and alterations in objective voice analysis (e.g. fundamental freq/pitch changed and decrease in maximum sound pressure).[19] Thus, subjective voice symptoms as well as objective laryngeal abnormalities seen in voice laboratory are common after thyroid surgery and may occur without vocal cord paralysis. Some of these changes may in fact be long lasting. One retrospective analysis at a mean of 4 years after uncomplicated thyroidectomy in a cohort of 60 patients identified a high prevalence of nonspecific voice changes (28%) and impaired swallowing (15%).[20]

The proposed mechanisms for voice alteration despite grossly normal RLN function include partial/ subclinical RLN dysfunction, unrecognized, partial/subclinical EBSLN dysfunction. Also included is endotracheal tube associated vocal cord/laryngeal injury, regional surgical effects such as strap muscle denervation and regional scarring as well as coincident voice change. An initial algorithm for organization of thought about such patients is outlined in Table 1.

In addition to voice changes, subjective nonspecific upper aerodigestive symptoms including dysphagia are common after thyroidectomy but are infrequently recorded or reported. A [illegible] was developed as a subjective self-evaluation questionnaire[19] and it demonstrated that swallowing problems occur in the first week in over half of patients and can last for more than three months. Possible discrete injuries to the extrinsic perithyroidal neural plexus including afferent fibers innervating the pharyngeal and laryngeal structures might explain such symptoms.

VOCAL CORD PARALYSIS WITHOUT VOICE SYMPTOMS

As noted above early VC edema may offset VC paralysis symptoms in the early postoperative period. In the later postoperative period VC paralysis can be asymptomatic due to variable remaining cordal function, variablity in paralytic cord position and variability of contralateral cord compensation. It is common place for example that with a permanent VC paralysis the symptoms improve due to contralateral VC compensation suggesting falsely that the VC paralysis has resolved.[21,22] In a recent study of 98 patients with VC paralysis, voice was normal in 20% and improved to normal in an additional 8%, hence nearly 1/3 were or became asymptomatic.[23] The fact that the surgical mishap is asymptomatic does not lessen its significance as a potential source of learning for the surgeon relating the surgical conduct of that case to a postoperative outcome. There is also significance of asymptomatic VC paralysis in terms of swallowing and increased morbidity if the contral nerve is operated on in the future.

PREOPERATIVE VOCAL CORD ASSESSMENT: THE RATIONALE

A long-standing debate has focused on the need for indirect laryngoscopy in all patients prior to thyroid surgery. There are several reasons in favour of its routine use.

Firstly, as noted above up to 1/3 or more of patients with unilateral RLN paralysis are asymptomatic and voice changes cannot be relied on as predictor of vocal cord function. Preoperative voice symptoms or lack of such symptoms are not a reliable indicator of RLN function. In a study of 340 patients, preoperative RLN palsy was identified in 22 patients and 55 patients had preoperative voice changes, allowing the authors to calculate a sensitivity of 68% and a positive predictive value of only 38% if one would rely on voice symptoms to predict RLN palsy.[24] The authors own experience is that voice changes as a screening test for RLN palsy have a sensitivity of 33% and specificity 75%, as demonstrated in an analysis of 365 patients.[22] In the same study, RLN palsy had an excellent predictive value for invasive thyroid disease (sensitivity 76%, specificity 100%).[22] The importance of knowing there is invasive, i.e. T4 disease preoperative cannot be overstated and allows for more appropriately aggressive surgical planning (as it relates to the airway and regional nodal basins), detailed preoperative imaging and more specific preoperative patient counseling. Aside from its significance in detecting invasive disease many studies

document a low but significant number of patients undergoing surgery even with benign disease with preoperative idiopathic VC paralysis.[25] Preoperative nerve injuries not detected preoperatively will likely be considered as resulting for the ensuing thyroid surgery on postoperative review.

Secondly, preoperative recognition of VC paralysis is essential in planning the procedure since management of the RLN found invaded at surgery is based on knowledge of its preoperative function.[22] The surgeon is empowered in the operating room by knowledge of preoperative VC function.

Finally if one is committed to accurate quality assessment through postoperative VC exam, then the rational interpretation of the postoperative assessment implicitly requires preoperative examination.

The 2003 *BAES* audit showed that preoperative laryngoscopy was done in 70% of patients undergoing thyroidectomy.[11] Although the UK guidelines suggest that all patients undergoing reoperative thyroid surgery should have preoperative laryngoscopy, only 75% of patients did so. These figures have remained unchanged in the 2005/2006 BAES audit. Current NCCN guidelines (National Comprehensive Cancer Network, USA) recommend VC examination in all patients undergoing thyroid cancer surgery (http://www.nccn.org).

In a large US and German thyroid cancer study group report of 5583 patients while preoperative vocal abnormality was found in 8.2% preoperative laryngology was performed on only 6.1%. One of the study's main recommendations was that laryngoscopy be performed preoperatively more frequency.[26] The Scandinavian Quality register for thyroid and parathyroid surgery in reviewing thyroid procedure in their 2008 report from 40 endocrine surgical units form Sweden/Denmark, note that preoperatively only 54% of patients undergoing thyroid surgery in these specialized endocrine units underwent preoperative laryngoscopy.[10]

POSTOPERATIVE VOCAL CORD ASSESSMENT: THE RATIONALE

Despite the acknowledged importance of postoperative laryngeal exam, the 2003 *BAES* audit showed that this was performed only in 4.1% of patients after first-time operations and in 7% of patients undergoing reoperative surgery.[11] The Scandinavian Quality register reported from 27 participating endocrine surgical departments in 3682 thyroid and parathyroid surgeries that only 45% patients were examined routinely at 1-6 weeks after surgery with 5.8% rate of RLN paralysis at those evaluations. Interestingly this rate seen on routine laryngeal postoperative exam fell to only 2.9% when the patient's larynx was examined based only on symptoms. These data suggest that routine laryngeal postoperative exam results in significantly higher rates of vocal cord paralysis than expected and significantly higher than if only patients who are symptomatic are evaluated. Basically the postoperative rate of VC paralysis doubles with routine laryngeal exam as compared with rates of postoperative hoarseness.[10]

CONCLUDING REMARKS

Traditionally the risk for recurrent laryngeal nerve injury after thyroidectomy quoted to patients derives from limited expert series and likely does not reflect the current true incidence in many clinical settings. This is especially true when as in the US the vast majority of thyroid surgery is done by surgeons not specializing in thyroid surgery and who perform low volume thyroid surgery.

The true rate of neural injury in various clinical settings can not be known unless each surgeon in their surgical practice with their unique practice mix examines the larynx on all patients pre and postoperatively as a standard quality assessment routine. Voice changes are in fact quite common and cannot be distinguished from nerve injury without routine glottic exam pre and postoperative laryngeal exam.

We propose four basic etiologic elements be considered with post-thyroidectomy vocal change (Table 1). Essential in the diagnosis is the laryngeal exam. It is understood that RLN and SLN injury may occur postoperatively and yet not have significant vocal symptoms. Preoperative and postoperative laryngoscopy should become part of the current quality assessment of all patients undergoing thyroidectomy since this is the only reliable way of accurate determination of the real

Table 1: Overview of etiologic factors that may be related to postoperative thyroidectomy voice changes

I. Neural Injury: A. RLN B. External branch SLN	Neuropraxia, Axonotomesis, Neurotomesis
II. Endotracheal tube associated laryngeal injury	a. Vocal cord injury/edema b. Arytenoid dislocation c. Paralysis or paresis (rare)
III. Regional nonneural effects	a. Strap muscle injury or denervation b. Global perilaryngeal plexus neural injury (non-RLN, non-SLN) c. Regional scarring/laryngeal fixation d. Direct cricothyroid muscle myositis
IV. Coincident voice change due to nonsurgical factors	a. Viral b. Other

incidence of RLN injury. This is especially true in this era of optimal quality assessment of surgical patients, a goal endorsed by virtually all surgical organizations. We owe this to our patients.

If we are to know exactly how well we are doing our surgery we must examine the larynx in all patients pre and postoperatively. The larynx is central in modern thyroid surgical practice.

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