Vocal cord paralysis is one of the feared iatrogenic complications of thyroidectomy. It can be resulted from transient insult to the recurrent laryngeal nerve (RLN) due to traction, stimulation, perineural bleeding or permanent damage due to section of the nerve intraoperatively. The transient injury is more common to occur, with the figure doubled to the permanent palsies. Visual misidentifications such as anatomical variations of the RLN for example in cases with extra-laryngeal branches, intertwining branches of the RLN with the inferior thyroid artery, distorted course of RLN and in non-recurrent laryngeal nerve has made the risk to be increased [1]. With the advancement of nerve identification techniques for example using intraoperative neuromonitoring, the rate can be expected to be further reduced.

Despite of iatrogenic cause of nerve injury leading to the voice change post operatively, some degree of alteration of vocal cord function may already present before the thyroid surgery. Alteration of physiological vocal cord function can be either by direct compression to the airway or RLN nerve compression. The thyroid lesion itself can affect the vocal cord function due to direct compression to the airway especially from a long standing mega-goiters that may compress the malacic trachea hence compromise the volume of air in the lower tract. This condition may impair the adequate subglottic pressure that is the prerequisite for the normal mucosal wave generation to result a normal voice production. On the other hand, a relatively small lesion that directly compresses the RLN can also cause neuropraxia causing voice change. Reversal in these conditions can be expected after thyroidectomy, lobectomy or even after aspiration of the thyroid cyst is performed [2,3].

In some circumstances, the resultant vocal cord paralysis secondary to the RLN compression by benign thyroid mass or more commonly RLN invasion by malignant counterpart is not clinically detectable or noticeable. It is true in cases whereby the unilateral nerve is affected with a small phonation gap. The patient may notice the slight voice change; either hoarse, breathy, weak or dysphonic for transient duration. Later on, the voice may ‘normalize’ with time as the insult is usually gradual and attributed to the compensatory mechanism of the opposite normal-functioning vocal cord. The moving vocal cord will compensate with hyper-adduction; crossing the midline to overcome the glottis gap induced by the palsied side. Thus, the dysphonia will disappear and the patient and the family members will appreciate the ‘normal’ voice despite of abnormal vocal cord function.

For patient who is considered for thyroid surgery, subjective or symptomatic voice assessments alone are insufficient [4]. In these cases, the role of laryngoscopy before surgery is of paramount importance. The traditional examination using indirect laryngoscopy (IDL) (looking into the mirror image of the larynx) should be replaced with more advanced phoniatric tests in order to provide superior diagnostic sensitivity [5]. Techniques and instruments for laryngeal examination which had evolved with time have allowed more detailed and precise evaluation of the larynx in general and the vocal cords in specific.

Rigid or flexible laryngoscopy allows the observation of larynx especially glottis closure in different tasks given to patient, patients were instructed to perform calm breathing, single vowel phonation, coughing and sniffing (maneuver to force abduct the vocal fold) [6]. The glottis closure not only contributed by the position of the vocal fold, it is also attributed to the bulk of the vocal fold. In a denervated vocal fold, the vocalis muscle will undergo a spectrum of degree of atrophy which will cause reduction in vocal fold bulk that eventually increases the degree of the phonation gap. Laryngoscopy is also used to document compensatory gestures that commonly occur in vocal cord paralysis for example supraglottic squeezing, ventricular phonation or pharyngeal constriction [6].

Compared to a normal light laryngoscopy, a stroboscopic examination has the advantage in slowing down the vocal fold vibration hence permits the observation of the detail of the mucosal wave. Vocal fold paralysis in stroboscopic examination may show mid-cord gap, posterior gap and level difference between the vocal fold, furthermore it can detect reduction in the amplitude of the mucosal wave [6].

There are cases whereby the patient presented with persistent hoarse voice after thyroidectomy with normal looking postoperative IDL. This presentation-finding inconsistency necessitates a more detailed examination than an IDL. The IDL image appreciation of minute changes attributed by external branch of superior laryngeal nerve (EBSLN) paralysis for example, is very operator-dependant and one may easily miss the subtle changes induced. The vocal cord will show gross symmetrical movement however, the configuration of the free edge which is bowed is hardly appreciated. EBSLN supplies the cricothyroid muscle which is responsible as the sole tensor of the vocal cord. Thus the lesion to the EBSLN will make the vocal cord lose its tension ability. One may appreciate these changes with either a 70 degree or 90 degree laryngoscopy or a stroboscopy for better delineation of subtle changes.

References

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