Recurrent Laryngeal Nerve Injury — Causes and Surgery

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The right recurrent laryngeal nerve arises from the right vagus nerve at the level of the innominate artery, where it loops around the subclavian artery. The nerve then travels along the pleura to the tracheoesophageal groove between the trachea and esophagus. The left recurrent laryngeal nerve has a longer course arising from the left vagus nerve and loops around the aortic arch and ligamentum arteriosum to ascend parallel to the left tracheoesophageal groove.

Anatomy and Function of the Recurrent Laryngeal Nerve
The recurrent laryngeal nerve (RLN) receives sensory innervation from the trachea, esophagus, and pyriform sinus before it enters the larynx deep into the inferior constrictor muscle and posterior to the cricothyroid articulation. The inferior thyroid artery and its branch, the inferior laryngeal artery, are responsible for blood supply to the RLN, which may pass anteriorly, posteriorly, or between the branches of the inferior thyroid artery.

Motor fibers of the RLN supply the lateral and posterior cricoarytenoid and thyroarytenoid muscles, as well as the oblique and transverse interarytenoid muscles. The posterior cricoarytenoid muscle is responsible for the abduction of vocal cords, while the thyroarytenoid, interarytenoid, and lateral cricoarytenoid muscles are responsible for the adduction of the vocal folds. The RLN supplies motor innervation to the inferior constrictor and cricopharyngeus muscles and provides sensory innervation to the subglottis and vocal-fold mucosa.

Anatomy of the vocal folds

There are two pairs of vocal folds; the true folds consist of muscles, ligaments, and lining mucosa. The false folds are present superior to the true folds. The laryngeal ventricle is a recess that separates true folds from the false. Vocal folds extend from the arytenoid cartilage posteriorly to the midline anteriorly. Both vocal folds approximate only during Valsalva and cough. True vocal folds divide the larynx into supraglottic, glottic, and subglottic compartments.
The laryngeal muscles can be subdivided into **intrinsic and extrinsic muscles**. The former is responsible for the mobility of vocal folds and phonation, and includes the posterior and lateral cricoarytenoid, interarytenoid, cricothyroid, and thyroarytenoid muscles. During inspiration, true vocal folds are abducted laterally, while during phonation, they move medially towards the midline.

All intrinsic muscles of the larynx are **innervated by the RLN except the cricothyroid muscle**, which is innervated by the external branch of the superior laryngeal nerve. The RLN is responsible for sensory innervation of laryngeal mucosa in the region inferior to the vocal folds.

**Pathology of Recurrent Laryngeal Nerve Injury**

Recurrent laryngeal nerve paralysis can involve the left, right, or both RLNs. The left RLN, being more superficial and longer running from the chest up through the neck, is more susceptible to injury than the right nerve. Injury can be due to **surgery, trauma, bacterial or viral infections, neurotoxic drugs, or tumors**.

**Causes of Recurrent Laryngeal Nerve Injury**

**Right RLN injury arises due to the following:**
- Neck trauma
- Benign or malignant thyroid disease
- Carcinoma of the esophagus
- Surgical trauma
- Subclavian artery aneurysm
- Idiopathic causes (mainly viral neuritis)
- Cervical lymphadenopathy

**Left RLN injury is likely to arise due to the following:**
- Trauma
- Thyroid diseases
- Thyroid and esophageal carcinomas
- Cervical lymphadenopathy
- Bronchogenic carcinoma
- Aortic aneurysm
- Enlarged left auricle
- Intrathoracic surgery
- Idiopathic

Bronchogenic carcinoma is an important cause that must always be ruled out in the case of left RLN injury. The most common cause is **non-thyroid cervical surgery**. Paralysis of the RLN can occur due to central causes affecting the nucleus ambiguus and its associated vagus nerve, and also result from conditions such as bulbar and pseudobulbar palsy, jugular foramen syndrome, and parotid tumors. Other lesions can be due to demyelinating diseases, skull base tumors, and cerebrovascular accidents.

RLN injury in the neck is due to thyroid tumors or surgery, cervical spine surgery, esophageal tumors, and deep penetrating wounds to the neck.

RLN injury in the chest may occur due to cardiac surgery, lung cancer, pulmonary tuberculosis, oesophageal cancer, mitral stenosis, and thoracic aortic aneurysm.
Bilateral RLN paralysis can be fatal. It is mostly caused during thyroid and cervical surgeries, trauma, endotracheal intubation, central brain disorders, diabetic neuropathy, organophosphorus poisoning, myasthenia gravis, and neurodegenerative disorders such as poliomyelitis and amyotrophic lateral sclerosis.

Clinical Manifestations of Recurrent Laryngeal Nerve Injury

The RLN is responsible for motor innervation of the laryngeal muscles. Injury to the nerve leads to the loss of adduction and abduction of vocal folds and results in its subsequent dysfunction during phonation, breathing, and deglutition.

In unilateral nerve injury, the paralyzed vocal fold is situated in the paramedian or partially lateral position and does not affect the airway patency, although phonation and deglutition are affected. However, compensation by the contralateral vocal folds may help in phonation. Therefore, unilateral RLN injury leads to hoarseness of voice and dysphagia that may improve and lead to asymptomatic presentations.

The airway is patent (without obstruction) due to abduction of the vocal folds. During phonation, a weak voice results due to the escaping air from the partially closed glottis. Deglutition, especially that of fluids, is impaired in glottal incompetence. Additionally, central lesions of the vagus nerve may cause sensory loss.

In bilateral injury that is mostly sustained during surgery, serious manifestations may be witnessed as soon as the patient is extubated.

Bilateral paralysis of the vocal folds can lead to stridor, difficulty in breathing, and aspiration. Breathing can be mildly distressed or severely impaired with biphasic stridor. The positioning of the denervated vocal folds is close to the midline and the glottis shows a narrow opening. Phonation may still be preserved in bilateral paralysis, but with inadequate intensity. Preoperative assessment of vocal-fold function is mandatory for legal purposes. Postoperative assessment is required for early detection of a malfunction, even if the patient is asymptomatic.

Diagnosis of Recurrent Laryngeal Nerve Injury

Patient history may often exclude factors such as heavy-metal neurotoxicity caused by lead and mercury, neurodegenerative disorders, alcoholism, diabetes, and neurotoxic drugs including phenytoin and isoniazid. Therefore, accurate medical history is essential to evaluate the possible etiology. MRI/CT of the head, neck, and chest, and esophagoscopy may be helpful in diagnosing neoplastic lesions affecting the nerve.

Vocal folds can be examined using indirect or fiberoptic laryngoscopy. Rigid laryngoscopy is helpful in differentiating between neurogenic paralysis of the vocal folds and cricoarytenoid arthritis secondary to prolonged endotracheal intubation or rheumatoid arthritis.

Management of Recurrent Laryngeal Nerve Injury

Unilateral nerve injury can be managed conservatively for up to 6 months to allow for spontaneous healing in the case of neurapraxia. For total nerve transection during surgery, corrective surgery should be considered as early as possible. Surgical
intervention includes **augmentation, medicalization, and reinnervation to improve voice quality**. Electromyography is considered to assess spontaneous recovery or determine the need for corrective surgery. Nimodipine has been suggested for the treatment of idiopathic and iatrogenic unilateral and bilateral vocal-fold paralysis without total transection, as it helps with neuronal regeneration and recovery.

Vocal fold augmentation (injection laryngoplasty) involves transoral or transcervical injection of collagen, hyaluronic acid, and autologous fat into the paralyzed cord. This results in bringing the paralyzed cord closer to the midline to prevent aspiration and cough, and improves phonation.

Medialization thyroplasty is performed by inserting an implant lateral to the paralyzed cord to shift it medially. Silicone or Gore-Tex sheets are implanted using a transcervical approach through the thyroid cartilage; these sheets are adjustable and allow for tuning of the voice.

Reinnervation of the vocal folds can be achieved using microsurgical techniques, but are met with limited success.

In the case of bilateral nerve injury, patients need **breathing assistance and airway reconstruction**. Permanent or temporary tracheostomy, endotracheal intubation, and reinnervation are emergency procedures that can be performed if required. Posterior establishment of the airway through the glottis using laser cordectomy is ideal to improve both breathing and quality of phonation. This is achieved via partial transection of the cords and arytenoid to establish an airway through the glottis. Some patients can benefit from lateralization of the vocal cords after arytenoidectomy; however, the outcome of this procedure on the quality of phonation is not satisfactory.

References

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