

Paparella: Volume III: Head and Neck

Section 2: Disorders of the Head and Neck

Part 5: The Larynx, Trachea, and Esophagus

Chapter 31: Vocal Cord Paralysis

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History

Since the time of Hippocrates (460 to 370 BC), denervation of the larynx has been known to lead to gross disturbance of the larynx's ability to protect the tracheobronchial tree with resultant life-threatening aspiration. Galen of Pergamon (129 to 199 AD) was the first anatomist to describe the recurrent laryngeal nerve as a branch of the vagus nerve, which he also described. He carried out experiments on live pigs, showing that division of the recurrent laryngeal nerve stopped the pig from squealing. He concluded that the voice originated from the larynx; previously it had been thought that the heart was the origin of the voice. Willis (1621 to 1675) was the first to give the description of the vagal and recurrent laryngeal nerves as found in most modern textbooks.

Although bilateral abductor paralysis was discussed in 1836 by Trusseau, it was not until after the advent of indirect laryngoscopy in the mid-1850s that knowledge of the in vivo appearance of vocal cord paralysis was possible, enabling Gerhardt to become the first to diagnose the condition clinically. The development of techniques to allow examination of the larynx in vivo coincided with the development of surgery of the thyroid gland, which had the direct effect of greatly increasing the number of patients presenting with disturbances of laryngeal innervation.

Anatomy

The cortical center for the formulation of speech is Broca's area. This area is an extension of the primary somatomotor cortex into the inferior frontal gyrus of the frontal lobe. Most information as to its functional significance is derived from the results of ablation or stimulation in experimental animals and humans (Penfield and Roberts, 1959). Ablation of Broca's area in the left or "dominant" hemisphere abolishes vocalization and usually produces a motor or expressive aphasia or paralysis of speech in humans. In some individuals, however, the leading speech area is on the right, and in some persons there is no such dominance. Stimulation in conscious patients can lead to acts of vocalization such as the utterance of a vowel sound. Little is known of the anatomic connections of Broca's area, although subjacent to it are large concentrations of association fibers effecting interconnections among many other parts of the cortex, and in recent years the pulvinar of the thalamus has been implicated as a subcortical center associated with the speech centers (Ojemann et al, 1968). Damage to the motor speech area does not lead to paralysis of the vocal musculature, all of which is in any case amenable to stimulation by appropriate parts of the primary motor area in the precentral gyrus.

The muscles of the pharynx and larynx are innervated by special visceral efferent branchiomotor fibers via the glossopharyngeal and vagus nerves and the cranial part of the accessory nerve. These fibers arise from the nucleus ambiguus. Each nucleus is composed of a column of cells approximately 2 cm in length lying in the reticular formation of the medulla, halfway between the spinal nucleus of the trigeminal nerve and the inferior olive. Each nucleus is connected to the corticonuclear tracts of both sides. The posterior inferior cerebellar artery supplies the posteroinferior portion of the cerebellum and the lateral portion of the medulla. Occlusion may lead to Wallenberg's syndrome (ipsilateral vocal cord paralysis with contralateral hemiparesis and analgesia) and ipsilateral ataxia, ipsilateral facial analgesia, and ipsilateral Horner's syndrome.

Like many other cranial nerve motor nuclei, the nucleus ambiguus can be divided into several subnuclei. The glossopharyngeal fibers arise from the cranial group of cells, whereas the individual laryngeal muscles are innervated by relatively discrete subnuclei in more caudal zones; most caudally, the cells of the nucleus ambiguus send axons into the cranial part of the accessory nerve.

Only a few special visceral efferent fibers destined for the vagus nerve pass from the nucleus ambiguus and join fibers from the dorsal nucleus of the vagus nerve to emerge through the ventral part of the spinal tract of the trigeminal nerve. Most fibers from the nucleus ambiguus run in the cranial root of the accessory nerve and join the vagus nerve below the skull.

The vagus nerve leaves the retro-olivary sulcus of the medulla oblongata by a series of rootlets in line with those of the glossopharyngeal nerve above and the cranial root of the accessory nerve below. The vagal rootlets unite to form a single trunk that runs through the subarachnoid space of the posterior cranial fossa. It passes below the glossopharyngeal nerve and above the accessory nerve and crosses the basiocciput behind the jugular tubercle. The vagus nerve enters the transverse slit of the middle compartment of the jugular foramen in company with the accessory nerve and contained in the same sheath of dura mater and arachnoid as the accessory nerve. A fibrous septum separates the vagus nerve and accessory nerves from the glossopharyngeal nerve and inferior petrosal sinus, which lie in the anterior compartment. The large posterior compartment is occupied by the termination of the sigmoid sinus. On emerging from the jugular foramen, the vagus nerve perforates the arachnoid and dura mater.

At its exit from the skull, the vagus nerve has two ganglionic enlargements, which are the sites of cell bodies of its afferent fibers. The small superior ganglion has bodies for the meningeal and auricular branches and lies in the jugular foramen. The much larger inferior ganglion, which lodges cell bodies of all the other sensory fibers in the vagus nerve, distends the vagus nerve just below the skull base.

The cranial fibers of the accessory nerve pass over the inferior vagal ganglion, but are attached to it by fibrous tissue only. Beyond the inferior ganglion, the cranial root of the accessory nerve blends with the vagus nerve and gives the vagus nerve all its nucleus ambiguus fibers to be distributed in appropriate branches to the pharyngeal and laryngeal muscles.

After leaving the skull, the vagus nerve lies in the carotid sheath between the internal jugular vein behind and the internal carotid artery in front, but as the vagus nerve descends vertically through the neck, its relationship to the great vessels changes and gradually it comes to lie behind and between the internal jugular vein and the common carotid artery.

The larynx is supplied by the superior and recurrent laryngeal nerves, which are the nerves supplying the fourth and sixth arches, that is, the superior laryngeal and recurrent laryngeal nerves. The nerve to the fifth arch does not persist in the adult. On the left side, the recurrent laryngeal nerve passes posterior to the ductus arteriosus, which is the sixth arch artery. Each branchial arch receives a branch from the nerve to that arch, this branch being the post-trematic nerve of the arch, since it lies posterior to the gill slit (trema = a slit). Further, each arch receives a branch of the nerve of the succeeding arch; this branch is called pretrematic, since it passes above and under the gill slit. The artery of each arch lies caudal to or behind the main post-trematic nerve at the arch. Therefore, since the recurrent laryngeal nerve is caudal to the ductus arteriosus (the sixth arch artery), it follows that the recurrent laryngeal nerve is probably not a nerve of an arch but may be a postbranchial nerve.

The superior laryngeal nerve leaves the vagus nerve just below the nodose ganglion and passes inferomedially deep to both carotid arteries toward the thyroid cartilage. It has an external and an internal branch. The external branch descends medial to the carotid sheath on the inferior constrictor muscle of the pharynx. It supplies branches to that muscle and ends in the cricothyroid muscle. The sensory internal branch descends to the thyrohyoid membrane, pierces this membrane along with the superior laryngeal artery, and supplies the mucous membrane of the pharynx, the epiglottis, the vallecula, the vestibule of the larynx, the pyriform recess, the aryepiglottic folds, and the mucous membrane on the back of the arytenoid cartilage. It also supplies one or two purely sensory branches to the interarytenoid muscle (Rueger, 1972); these branches unite with twigs from the recurrent laryngeal nerve to the same muscle.

The right vagus nerve passes in front of the origin of the right subclavian artery in the root of the neck and gives off the right recurrent laryngeal nerve, which loops below the artery and ascends behind the carotid sheath between the trachea and the esophagus. The left recurrent nerve arises from the left vagus nerve as the latter crosses the aortic arch and loops below the ligamentum arteriosum and ascends behind the aorta to reach the groove between the trachea and esophagus on the left side.

As each nerve ascends, it is closely related to the corresponding inferior thyroid artery and may be damaged during operations on the thyroid gland. At the level of the cricoid cartilage, each nerve passes beneath the lower border of the inferior constrictor muscle in company with the laryngeal branch of the inferior thyroid artery and then enters the larynx behind the cricothyroid joint. It supplies all the intrinsic muscles of the larynx except the cricothyroid muscle and distributes sensory branches to the laryngeal mucous membrane below the level of the vocal cords. Before entering the larynx close to the cricothyroid joint, it usually divides into a motor and sensory branch, not into adductor and abductor rami as has sometimes been asserted (Williams, 1954).

The variations in the relationships of the recurrent laryngeal nerves as they approach the larynx are especially important in surgery of the thyroid gland (Bowden, 1955; Doyle et

al, 1967). The nerve does not always lie in a protected position in the tracheoesophageal groove but may lie a little in front of it (slightly more frequent on the right side of the neck), and it may occasionally be some distance lateral to the trachea at the level of the lower part of the lobe of the thyroid gland. On the right side, there is an almost equal chance of finding the nerve anterior to, posterior to, or intermingled with the terminal branches of the inferothyroid artery, whereas on the left side, the nerve is most likely to be posterior to the artery and is least likely to be anterior.

Physiology

The muscles of the larynx may be divided into extrinsic (which attach the larynx to neighboring structures) and intrinsic, which consist of two separate groups - those that control the operation of the inlet (the aryepiglottic and thyroepiglottic muscles) and those that move the vocal folds. The names of the muscles are easy to remember, as they are determined by the cartilages to which they are attached. The posterior cricoarytenoid muscle is the only dilator muscle of the rima glottidis, producing abduction of the vocal folds. The lateral cricoarytenoid, the interarytenoid, and the lateral thyroarytenoid muscles adduct the vocal folds and contribute to the sphincteric functions of the larynx.

Each arytenoid cartilage articulates with the cricoid cartilage by means of a synovial joint. The cricoid facet for the arytenoid cartilage is oblong and convex and is located at the posterolateral extremity of the cricoid rim. The arytenoid cartilage can rotate on the cricoid cartilage around an axis passing not quite vertically through the arytenoid cartilage. Contraction of the lateral cricoarytenoid muscle rotates the arytenoid cartilage in the opposite direction and abducts the vocal cords. The arytenoid cartilage may also glide laterally and downward following the slope of the cricoid facet, or medially toward the other arytenoid cartilage so that contraction of the unpaired interarytenoid muscle brings the arytenoid cartilages together. The two movements usually occur together; thus the lateral fibers of the thyroarytenoid muscles both draw the arytenoid cartilages forward and rotate these cartilages medially.

The tension of the vocal folds is increased by the contraction of the cricothyroid muscles and the medial fibers of the thyroarytenoid muscles. The cricothyroid muscles raise the anterior arch of the cricoid cartilage upward, reduce the cricothyroid gap, and tilt the posterior lamina of the cricoid cartilage backward. The arytenoid cartilages superimposed on the cricoid lamina are then carried backward, thus elongating and stretching the cords and increasing their tension.

The vocal cords of all mammalian larynges move simultaneously and symmetrically. In humans, the left nerve is 43 cm long, and the right nerve is 32 cm long. Other factors being equal, this variation in the conduction pathway from nucleus ambiguus to intralaryngeal musculature should result in a discernible lag of the left cord behind the right cord. Harrison (1981) investigated the possible anatomic variations between the two recurrent laryngeal nerves in humans and in the giraffe looking for a realistic explanation for differences in conduction time. His findings indicate that in both humans and the giraffe, the left recurrent nerve does contain more larger, fast-conducting fibers.

The functions of the larynx may be broadly classified as follows:

1. Respiratory air channel and air flow regulation. The larynx is part of the tubular system of airways for the passage of air to and from the lungs in breathing. At the glottis, the cross sectional area of the respiratory airway is smaller than at any other level. It thus plays a role in determining the resistance to air flow, which is related inversely to diameter. During normal respiration, the cords are relaxed and halfway between adduction and full abduction. The vocal folds of some individuals abduct slightly in inspiration and adduct slightly during expiration. Thus, the lumen of the larynx at this point narrows in expiration and widens in inspiration. This movement of the vocal folds is usually very slight in the resting subject but increases in amplitude with increasing depth of breathing. With very deep breathing, as in violent exercise, the folds may be abducted almost flush with the lateral wall so that the resistance to air flow is reduced to a minimum.

2. Sphincteric functions. The primary sphincteric action of the larynx is to protect the tracheobronchial tree during swallowing and vomiting. The larynx also acts as a check valve, first, to permit the intrathoracic pressure to rise for explosive release in reflex coughing and sneezing and second, to permit the intra-abdominal pressure to rise in defecation, micturition, parturition, and straining, as in lifting very heavy weights.

The larynx can be divided functionally into three successive physiologic sphincters at different anatomic levels. These sphincters, in cephalocaudal order, are the aryepiglottic folds, the vestibular folds, and the vocal folds. The muscular component of all these sphincters is derived from the internal muscles of the larynx. The sphincters may contract together or independently. All three may act together (eg, during deglutition). The vestibular and vocal folds may appose without closure of the aryepiglottic sphincter, as can be observed during laryngoscopy. The vestibular folds cannot be closed independently of the vocal folds. Conversely, the vocal folds adduct without closure of the other two sphincters during phonation. Mechanical valvular mechanisms are present at the vestibular folds and the vocal folds but not at the aryepiglottic sphincter level. This mechanical action provides a more resistant and powerful valve than does the contraction of the sphincter muscle alone, but muscle contraction is essential for effective operation of the folds as a mechanical valve. At the level of the vestibular folds, the effect of the mechanical valve is to prevent outflow of air. The greater the outflow pressure applied to the valve the greater its resistance. These folds have no useful mechanical action in impeding inflow. At the level of the vocal folds, the mechanical principle acts in the opposite direction, preventing inflow. The unidirectional nature of these folds have flat lower surfaces, and pressure on this surface is uniformly distributed. Their upper surfaces slope obliquely. Pressure applied from above to this surface is maximally deflected onto the free margins, which are therefore readily forced apart. The vocal folds have flat upper surfaces, whereas the lower surfaces are curved and sloping and thus not designed to resist pressures from below.

3. Receptive field for reflexes, for example, the cough reflex. The reflex is initiated by chemical or mechanical irritation of the larynx. Transmission through the vagus nerve results in stimulation of the cough reflex center in the medulla of the brain.

4. Phonation and speech. In the larynx, the pitch of the voice is regulated by the tension, length, and shape of the vocal folds, dependent on the action of the laryngeal

musculature, and the pressure of the intraglottic column of air being forced through the vibratory folds. The effective force setting the folds into vibration and producing sound is the pressure of the intraglottic air column. As the column of air passes up from the trachea it passes between the vocal cords. Here, the cross sectional area of the airway is smallest, and consequently the air is moving quickest. According to Bernoulli's principle, in a tube the total energy that is the sum of kinetic energy and the pressure energy is constant. At the vocal folds, the kinetic energy will be maximal; therefore, the pressure drops and pulls in the mucosa to fill the small gap. This complete closure causes a sharp rise in the subglottic air pressure, which increases until a column of air pushes between the mucosal folds, causing them to open again; thereafter, the cycle of closing and opening repeats itself. This is the accepted aerodynamic or tonic theory of vocal fold vibration. It is strongly supported by findings that refute the neuromuscular theory, which states that the vocal fold vibration is believed to be due to active laryngeal muscle contractions at the frequency of the laryngeal tone produced.

Pathophysiology

The physiologic function of the larynx is adversely affected by vocal cord paralysis. Interference with protection of the tracheobronchial tree and respiration are the more serious consequences and may be life-threatening. Interference with voice production, though not a threat to physical survival, may affect social and economic survival.

The vocal folds can adopt a number of positions from full abduction to complete adduction. The classification of the positions of the vocal cords has been the subject of much disagreement. Six positions are described (Clerf and Suehs, 1941; Negus, 1947). Since the cords do not assume the "cadaveric position" after death, it has been suggested that this term be changed to *intermediate*. Median and paramedian positions are relatively indistinguishable, so it is best to use only the term *paramedian*. The position of the vocal cord cannot be recorded precisely. In particular, the paramedian and cadaveric positions are approximately 2 to 3 mm apart and are often difficult to record with accuracy (Maran, 1979).

The difficulty in recording vocal cord position may account for the incorrect theories and inconsistencies in other theories, which seek to explain the variety of positions of the vocal cord in vocal cord paralysis. The following sections discuss some of the theories put forward.

Semon's Law

The first theory depended on the concept that abductor fibers in the recurrent laryngeal nerves are more susceptible to pressure than are adductor fibers. The theory was proposed by Rosenbach (1880) and Semon (1881), but it is usually known as Semon's law. After several amendments it stated that "in the course of a gradually advancing organic lesion of a recurrent nerve or its fibers in the peripheral trunk of the recurrent nerve, three stages can be observed. In the first stage, only the abductor fibers are damaged; the vocal folds approximate in the midline and adduction is still possible. In the second stage the additional contracture of the adductors occurs so that the vocal folds are immobilized in the median position. In the third stage, the adductors become paralyzed and the vocal fold assumes the cadaveric position." The theory has, however, clinical and experimental inconsistencies. The vocal fold is

paralyzed in the paramedian position with either gradual pressure or complete division of the recurrent laryngeal nerve (Arnold, 1961). There is no evidence to prove that progressive pressure on the recurrent nerve first causes paralysis of the posterior cricoarytenoid and then the adductor muscles (Maran, 1979).

Differential Innervation Theory

Another theory of that time was based on the anatomic fact that the recurrent nerve often branches outside the larynx. If some branches were going to the adductor muscles and others to the abductor muscles, injury to the individual branches would cause paralysis of specific muscles. However, it is now known that extralaryngeal branching produces a motor and a sensory branch. The motor portion of the recurrent laryngeal nerve branches deep to the thyroid cartilage, and the branches are only a few millimeters long, thus making selective damage to individual motor branches improbable.

Changes in the Cricoarytenoid Joint and Paralyzed Muscles

Changes in the cricoarytenoid joint and paralyzed laryngeal muscles have been proposed to explain the position of the cord (Ellis, 1954). Degeneration in the joint has been found (Kirchner, 1966). This may explain changes in position of the folds with time, but there is no evidence to show that a paralyzed fold contracts (Maran, 1979), and there has been no anatomic proof that vocal folds ending in the intermediate position are scarred or different in size from those ending in the paramedian position.

Interarytenoid Muscle Contraction

Another theory is that the paramedian position in unilateral recurrent paralysis may be the result of contraction of the interarytenoid muscle, which receives bilateral innervation. However, although interarytenoid muscle contraction approximates the arytenoid cartilages and closes the posterior glottis when both recurrent nerves are intact, partial movement when one nerve is intact does not appreciably close the space between the membranous portions of the fold.

Disturbance of Autonomic Supply

It has been proposed, without experimental evidence, that the intrinsic laryngeal muscle tone and the position of the vocal cords is determined by contractions resulting from disturbance of the vascular and sympathetic nerve supply.

Wagner and Grossman Theory

The most popular and widely accepted theory is that of Wagner (1890) and Grossman (1897), which states that complete paralysis of the recurrent laryngeal nerve results in the cord being in a paramedian position because the intact cricothyroid muscle adducts the cords. If the superior laryngeal nerve is also paralyzed, the cord will be in the intermediate position because of the loss of this adductive force. This theory has been confirmed by visual and electromyographic evaluation of dog larynges during division of the recurrent nerve, the superior laryngeal nerve, and both nerves (Dedo, 1970).

According to the theory, chest lesions should cause recurrent laryngeal paralysis alone, but in many patients with lung cancer the cord is in the intermediate position. It has been proposed that retrograde atrophy of the vagus nerve occurs up to the nucleus ambiguus and stretching of the recurrent nerve by an enlarged heart or aortic aneurysm pulls the vagus nerve down from the base of the skull, injuring the superior laryngeal nerve (Maran, 1979).

Paralyzed vocal folds may show some movement. It has been postulated that attempts at adduction by a paralyzed vocal fold may be due to contraction of the bilaterally innervated interarytenoid muscle as well as the intact cricothyroid muscle. The cricothyroid muscle has been shown to be the major component of this movement. Adduction is seen in bilateral recurrent nerve paralysis in the paramedian position with electromyographically proven interarytenoid muscle paralysis and is abolished when the superior laryngeal nerve is divided in experimental animals (Dedo, 1970).

Pathogenesis

Vocal cord paralysis is a sign of disease and not a diagnosis. It may be due to a lesion at any point from the cerebral cortex to the neuromuscular junction.

As the corticobulbar fibers decussate at the upper border of the medulla, generally only a bilateral symmetric lesion of the cortex produces a laryngeal palsy. Rarely, a unilateral cortical lesion may produce isolated palsies so variable is the decussation. Because of the sheer size of the nucleus ambiguus, small lesions in it may produce isolated laryngeal and pharyngeal motor losses. Lesions of the nucleus ambiguus produce a bilateral paralysis more often than a unilateral paralysis. Peripheral damage to the laryngeal innervation is of three major types: (1) to the vagal trunk above the nodose ganglion, the origin of the superior laryngeal nerve, (2) to the vagus nerve below that level or to the recurrent laryngeal nerves, and finally (3) to the superior laryngeal nerve alone.

Vocal cord paralysis may be congenital or acquired.

Congenital Paralysis

Many infants with stridor in infancy have congenital paralysis of one or both vocal cords. Holinger and Brown (1967) found that vocal cord paralysis composed 10 per cent of congenital laryngeal abnormalities. Congenital vocal cord paralysis can occur with or without associated anomalies, which include neurologic, laryngeal, and cardiac defects, but the most common associated abnormality is hydrocephalus, which may or may not be associated with the Arnold-Chiari malformation. The precise mechanism of vocal cord paralysis in children remains unclear, but various theories have been proposed, including brain stem dysgenesis, mechanical compression, or stretching of the vagus nerve, with or without resultant neuronal degeneration and circulatory impairment of the brain stem (Holinger et al, 1978). Many cases are idiopathic and may follow a complicated labor and delivery in which there may have been head trauma, hypoxia, or shoulder dystocia (Gentile et al, 1986).

Acquired Paralysis

Table 1 shows the incidence of recurrent laryngeal paralysis from various causes as derived from our series over the last 20 years. The incidence of the various causes is similar to those of other recent large series (Parnell and Brandenburg, 1970; Stenborg, 1973; Maisel and Ogura, 1964; Titcher, 1976). Because of its longer path, the left recurrent laryngeal nerve is affected in 78 per cent of cases, the right recurrent laryngeal nerve is affected in 16 per cent of cases, and both nerves are affected in 6 per cent of cases. Since the most common cause is malignant disease, the maximal age incidence is in the seventh decade of life. Men are affected eight to ten times more often than are women (Stell and Maran, 1978). The following sections discuss the various causes.

Table 1. Percentage Incidence of Causes of Vocal Cord Paralysis

Malignant disease	31
Surgical trauma	29
Idiopathic	24
Nonsurgical trauma	7
Inflammatory	4
Neurologic	1
Miscellaneous	4

Malignant Disease. One in three recurrent laryngeal nerve paralysees is due to cancer, and of these 50 per cent are due to lung cancer, 20 per cent to esophageal cancer, and 10 per cent to thyroid cancer. Other causes include temporal bone neoplasms; posterior fossa tumors; nasopharyngeal tumors; paragangliomas of the vagal, jugular, and carotid bodies; metastatic cancer; and lymphomas.

Surgical Trauma. Surgery to the thyroid gland is the most common cause of surgical trauma. Esophageal and lung resection for carcinoma, operations on the carotid artery and cervical spine, radical neck dissection, mediastinoscopy, surgery on a pharyngeal pouch, partial laryngectomy, and cardiac surgery (including pacemaker insertion) may all cause nerve damage.

Nonsurgical Trauma. Neck injuries resulting from automobile accidents, skull fractures, and penetrating injuries of the neck may all cause damage to the nerve. Stretching of the nerve by the enlarged left side of the heart in congestive cardiac failure, aneurysm of the aortic arch, and a dilated pulmonary artery in mitral stenosis have been quoted as causing paralysis. Paralysis has been described in nonmalignant thyroid disease (Worgan et al, 1974) and as being caused by a benign parathyroid adenoma (Love, 1972). Minuck (1976) observed that unilateral vocal cord paralysis may follow intubation with an endotracheal tube without the presence of any local lesion. Ellis and Pallister (1975) reported four similar cases and by cadaveric dissection showed that the inflated cuff of an endotracheal tube compresses the anterior branch of the recurrent laryngeal nerve between the cuff and the interior surface of the thyroid lamina. Just such a mechanism had been postulated by Hahn and colleagues in 1970 when they hypothesized that a defective, irregularly inflating cuff placed in the larynx could exert unduly high pressure localized to the intralaryngeal course of the recurrent laryngeal nerve. It was a full 6 months before vocal cord movement began to be restored in

Minuck's patient.

Inflammatory Causes. By far the most common cause (95 per cent) in this group is pulmonary tuberculosis, caused by either apical or mediastinal scarring or involvement of mediastinal glands (Maran, 1979). Other inflammatory causes include jugular thrombophlebitis of aural origin, subacute thyroiditis (Langevitz and Cabili, 1983), meningitis, influenza, diphtheria, typhoid fever, and a range of granulomas at the skull base, including tuberculosis.

Neurologic Causes. Central causes of acquired paralysis include cerebrovascular disease and brain stem ischemia, epilepsy, Parkinson's disease, multiple sclerosis, syringobulbia, amyotrophic lateral sclerosis, poliomyelitis, and head injuries. Neuropathies can be caused by alcohol or diabetes, and paralysis has also been described in the Guillan-Barré and Charcot-Marie-Tooth syndromes. Neuritis from vinblastine therapy can also occur.

Miscellaneous Causes. Hemolytic anemia, thrombosis of the subclavian vein, syphilis, collagen diseases, myasthenia gravis, and lead and arsenic ingestion are miscellaneous causes of acquired paralysis.

Idiopathic Causes. No cause is ever found for many laryngeal nerve lesions. Because of the high incidence of malignant disease in the lung, the cause of a vocal cord paralysis should not be regarded as unknown until at least 18 months after the first diagnosis. Many idiopathic paralyzes are due to viral disease, but actually proving a viral cause is difficult. Paralysis can be due to infectious mononucleosis (Stell and Maran, 1978). Many cases were described in the influenza epidemic of A2 Hong Kong 1/68 virus in Europe, and some were associated with a phrenic nerve paralysis. Only 20 per cent of the vocal cord paralyzes resolved within 18 months (Fex and Elmquist, 1973).

Immobility of the vocal cord may be due to cricoarytenoid ankylosis, which may follow severe laryngeal infections such as syphilis or tuberculosis, often with consequent perichondritis or chondral necrosis, or may follow various kinds of trauma. In 1976, Goodman and associates reported fixation of the cartilage in gout, with uric acid deposits in the joint space. Wolman and co-workers (1965) studied the larynx postmortem in eight patients with advanced rheumatoid arthritis; all but one had complained of dyspnea and stridor. Changes were found not only in the cricoarytenoid joints but also in the laryngeal muscles. The joint changes consisted of marked thickening of the synovial membrane resulting from infiltration by chronic inflammatory cells and erosion of the adjoining articular surfaces of both cricoid and arytenoid cartilages. However, there was no ankylosis, and although movement was restricted, the arytenoid cartilages remained mobile in all cases. The myopathy was considered to be the result of a rheumatoid neuropathy and secondary muscular atrophy. The essential lesion was thought to be arteritis of the vasa nervorum. In 1966, Montgomery reported 26 cases of immobility of the arytenoid cartilage due to rheumatic inflammation. Histologic investigation of the cricoarytenoid joint showed a connective tissue ankylosis in only two patients. Among a large number of ankyloses of the arytenoid joint reported by Rethi (1955), recurrent laryngeal nerve paralysis was the only cause in 16 cases. Fixation of the arytenoid cartilage is more common in bilateral than in unilateral vocal cord paralysis. Tucker and Rusnov (1981) report that one third of all patients who had bilateral vocal cord paralysis for longer than 6 months also experienced ankylosis of the cricoarytenoid joint.

Assessment of the Patient With Vocal Cord Paralysis

Symptoms and Signs

Vocal cord paralysis can present with hoarseness, stridor, poor cough, aspiration, or dysphagia. In acute paralysis of the recurrent laryngeal nerve, the voice is extremely breathy, phonation time is limited, and the ability to cough is impaired. The paralyzed cord lies in the paramedian position. When the cricothyroid muscle is also paralyzed, the voice is hoarser and the cord lies in the intermediate position. If the sensory internal branch of the superior laryngeal nerve is also affected, normal secretions and saliva tend to accumulate on the flaccid cord and spill over unexpectedly, causing sudden minor coughing episodes or a need to clear the throat.

Since it is often difficult to record accurately the position of the cord, a better indication of vocal cord position is the width of the membranous glottic chink in phonation. In the paramedian position, not only will the gap be greater but also the tip of the vocal process will be directed medially as a definite prominence. Classically, when there is combined recurrent and superior laryngeal paralysis, the larynx is said to be askew, with the thyroid cartilages rotated over the cricoid ring, the affected cord at a lower level, and supraglottic anesthesia. These latter signs, however, are neither consistent nor always obvious enough to be of use in clinical diagnosis.

Observation of Wrisberg's cartilage in the aryepiglottic fold may help in differentiating paralysis from fixation of the cricoarytenoid joint. In fixation of the cricoarytenoid joint, the cartilage retains its normal upright position, braced by the action of the posterior cricoarytenoid muscle. In recurrent laryngeal nerve paralysis, this action fails and the cartilage droops forward at the posterior end of the glottis, making it appear shorter than its fellow. Again these signs are not always obvious on indirect laryngoscopy.

In bilateral abductor paralysis, voice quality may be normal and there may be no aspiration. Breathing, though, may be turbulent. With the passage of time over a year, the paralysis may cause sufficient bilateral cord atrophy to result in less turbulent laryngeal air flow. However, as the atrophic vocal cords assume more lateral positions, vocal quality suffers and occasional aspiration may ensue.

Patients with either unilateral or bilateral vocal cord paralysis may experience transient dysphagia. During the acute phase, the loss of the protective mechanism of the supraglottic sphincter makes swallowing difficult, but complete compensation usually develops later. However, vagal paralysis above the inferior ganglion, whether unilateral or bilateral, can be catastrophic. Not only is the larynx paralyzed but the pharyngeal muscles are also denervated. Because high vagal paralysis affects both the superior and recurrent laryngeal nerves, a slightly abducted paralyzed cord results. The open position of the vocal cord invites aspiration. Meanwhile, simultaneous pharyngeal paralysis produces ballooning of the atonic constrictor muscles, resulting in a reservoir of secretions capable of contaminating an incompetent larynx and soiling the lungs. More important, the combined lack of sensory cues from the pharynx, larynx, and trachea produces total impairment of the pharyngeal stage of swallowing, resulting in further life-threatening aspiration pneumonitis.

Complete interruption of the intracranial portion of one vagus nerve results in a characteristic paralysis. The soft palate droops on the ipsilateral side and does not rise in phonation. There is loss of the gag reflex on the affected side as well as of the curtain movement of the lateral wall of the pharynx, in which the faucial pillars move medially as the palate rises in saying "ah". There may also be a loss of sensation in the external auditory canal and behind the pinna.

To identify the cause of vocal cord paralysis, it is essential to ask about symptoms such as a sore throat, otalgia, dysphagia, cough, hemoptysis, weight loss, and any noticed lumps. It is also necessary to ask about the patient's smoking habits or any recent illness, especially a viral one. Paralysis caused by surgical trauma is usually seen shortly after the operation, but it is often diagnosed later. For example, it is possible for a bilateral abductor palsy to be caused by thyroidectomy and for the patient to have few or no symptoms immediately afterward. Only several years later, if the cricoarytenoid joint becomes fixed or the vocal cords become edematous from an upper respiratory infection, does the patient experience stridor. Hence, it is important to ask about all previous surgery to the head, neck, and chest.

Consideration of the causes of vocal cord paralysis indicates the range of additional physical signs to be found on general examination and a mandatory head and neck examination. Particular note should be made of other neurologic deficits, which will help to determine the site of the lesion. If the lesion is intramedullary, there are usually ipsilateral cerebellar signs, loss of pain and temperature sensation over the ipsilateral face and contralateral arm and leg, an ipsilateral Bernard-Horner syndrome, and involvement of the glossopharyngeal and accessory cranial nerves. If the lesion is extramedullary, within or at the exit of the skull, or high in the neck, again the glossopharyngeal and accessory cranial nerves are frequently involved. In the past, such combinations of lower cranial nerve palsies have been given a variety of eponymic designations. In lesions of the lower neck, possible associated neural deficits are seen in the accessory and phrenic nerves. In the mediastinum, usually only the recurrent laryngeal nerve is affected. In children, unlike adults, unilateral vocal cord paralysis may give rise to stridor on physical effort. Thus, breathing is stridulous on crying, coughing, or straining at the stool, but the child sleeps quietly. Babies with bilateral paralysis, in contrast, are in more or less constant distress. Bilateral cord paralysis in children may be confirmed by passing an endotracheal tube between the vocal cords at which time the symptoms will be relieved. All children with laryngeal paralysis have feeding difficulties, with overspill and infection of the tracheobronchial tree. For this reason, these babies tend to be weak and fail to gain weight.

Investigations

Radiology. A chest radiograph should always be performed, both as part of the general assessment and to demonstrate any lung disease, particularly carcinoma. Tomograms of the left upper lobe and left hilum, or a computed tomography (CT) scan, are often required. A barium swallow should also be performed if the cause remains undetected after a chest radiograph; it demonstrates not only esophageal neoplasms but also compression by thyroid masses and an enlarged left atrium. An intermediate position of the vocal cord indicates a likely cause above the level of the larynx; thus, radiology of the skull base, petrous bone, and nasopharynx is indicated. If an intracranial pathologic site is suspected, CT is required.

Laboratory Analysis. Every patient should have full blood indices measured. The differential white count, film, and erythrocyte sedimentation rate help in the diagnosis of lymphomas or tuberculosis. If infectious mononucleosis is suspected, an appropriate blood test will be necessary. Serologic examination for prior infection with syphilis should be undertaken. Neuropathy secondary to diabetes occurs only in well-established, previously diagnosed diabetes, so a glucose tolerance test is not needed. Viral serologic examination may be performed but needs to be repeated weeks later to note a change in antibody titers.

Miscellaneous Studies. If a thyroid gland mass is palpable, a thyroid isotope scan and ultrasound scan of the thyroid gland will be required, whereas if nothing is palpable it is unlikely that anything will be demonstrated by a scan.

Endoscopy. The preceding investigations may reveal a cause that requires endoscopic confirmation and biopsy. Moreover, if all the appropriate investigations produce normal results, panendoscopy is necessary. This procedure comprises examination of the postnasal space, direct laryngoscopy, tracheoscopy, bronchoscopy and esophagoscopy. Direct laryngoscopy should demonstrate the mobility of the cricoarytenoid joint. A suction tip or spatula is used to passively displace the arytenoid cartilage laterally and exclude fixation of the cricoarytenoid joint. Direct laryngoscopy can also demonstrate a previously unsuspected subglottic carcinoma. If panendoscopy gives normal results, a biopsy of the ipsilateral fossa of Rosenmüller is required, particularly if the vocal cord is in an intermediate position. Similarly, a carinal biopsy is particularly indicated if the vocal cord is in a paramedian position.

General anesthesia should be employed in almost all situations in which diagnosis or treatment of lesions of the pediatric larynx is contemplated. However, the flexible fiberoptic laryngoscope allows a prolonged view of the larynx in an atraumatic setting under topical anesthesia. It is particularly useful for examining laryngeal function and allows a panoramic view of the supraglottic and glottic areas without interfering with laryngeal movement. It is impossible to assess the movements of the vocal cords accurately with the patient under general anesthesia. The main stimulus for movement of the vocal cords is phonation, and very few patients talk under general anesthesia! Furthermore, a patient recovering from anesthesia makes mass involuntary movements of the pharynx and larynx, and these can simulate movement of the cords.

Respiratory Function Tests. The functional assessment of the severity and character of an upper airway obstruction is important for diagnosis, management, and evaluation of the efficacy of medical and surgical treatment. In vocal cord paralysis, the site of airway obstruction is extrathoracic and the airway wall is mobile; inspiratory flow (Brookes and Fairfax, 1982). Simple spirometric pulmonary function tests are commonly assessed by analysis of a forced expiration from total lung capacity and therefore are concerned with expiratory events. A more sensitive objective functional assessment of upper airway obstruction is made by measuring air flow rates at the mouth throughout both phases of respiration and enables a flow-volume loop to be constructed. Flow-volume loop analysis not only is more sensitive at detecting airway obstruction but also can specifically define the major site and character of obstruction. It is in defining the latter two features that conventional spirometry is most limited. The characteristics of the flow-volume loop categorize upper airway obstruction into one of three types: (1) a variable extrathoracic

obstruction, such as vocal cord paralysis, which characteristically leads to a selective reduction in inspiratory flow rates; (2) a variable intrathoracic obstruction, which leads to a selective reduction in expiratory flow rates; (3) a fixed obstruction, such as a mass or infiltrative lesion, which causes a nonyielding or constant reduction in the cross sectional area of the airway and results in a flow rate limited in expiration and inspiration (Kashima, 1984).

Voice Assessment. Tape recording of vocal function before and after treatment is often invaluable in evaluating results (Lewy, 1976). Procedures for assessment of voice disorders comprise tests that elicit information about the process of voice production and the nature of the sound generated.

Electromyography of the laryngeal muscles evaluate some of the parameters that regulate the vibratory pattern of the vocal folds. Electromyography is particularly useful in vocal cord paralysis. It is helpful in differentiating vocal cord paralysis from immobility of the vocal cords caused by mechanical fixation. It also gives information about the degree and the extent of paralysis. It is useful in determining the side to subject to lateral fixation of the vocal cord in cases of bilateral vocal cord paralysis, and it has been reported as being helpful in determining the prognosis for recovery. A favorable prognosis is indicated by the presence of action potentials induced by voluntary activity (Hirano et al, 1974). One aerodynamic test is the mean air flow rate. In recurrent laryngeal nerve paralysis it is generally greater than normal. It is a good indicator of phonatory function and can be used to monitor treatment (Shigemori, 1977). Stroboscopy, ultra-high speed cinematography and glottography can be used to examine the vibratory pattern of the vocal folds. Stroboscopy is the most practical technique for use in clinical practice.

Acoustic analysis of the voice signal has a promising future as a diagnostic tool in the management of voice disorders, as it is noninvasive and provides objective and quantitative data. Conversely, experienced voice clinicians are frequently able to determine the causative pathologic conditions on the basis of the psychoacoustic impression of abnormal voices. The nature of the pathologic voice has been classified and described, but the definition of the terms used has often been controversial, and standardization of the terminology is required. Rontal and colleagues (1975) state that there are three characteristics identifiable to spectrographic analysis that are related to a paralyzed vocal cord: (1) excessive breathiness, (2) aperiodicity of vocal cord movement, and (3) a breakdown in formant structure. The length of time for which patients can sustain phonation is frequently abnormally short in recurrent laryngeal nerve paralysis (Shigemori, 1977), and the range of frequencies that a patient can cover is reduced (Sawashima, 1968). Lewy (1976) has recorded cough sonograms before and after treatment of vocal cord paralysis to demonstrate improvement in cough effectiveness.

Treatment

Prophylactic Treatment

Thyroid gland surgery techniques can be improved to reduce the rate of irreversible nerve damage. Better preoperative control of thyrotoxic glands and earlier diagnosis of malignancy makes operative conditions much better. Thyroidectomy without exposing the recurrent laryngeal nerves causes a 5.4 per cent rate of paralysis, half of which is not

reversible (Gisselsson, 1950). Although routine exposure of the nerves causes an 8 per cent rate of paralysis, only 1.3 per cent is irreversible (Widstrom, 1973). Thus, routine exposure of the nerve at thyroidectomy is advised. Early attempts to detect recurrent laryngeal nerve damage by electrical stimulation were unsuccessful (Holl-Allen, 1967), but more recently techniques have been devised that will help identify the recurrent laryngeal nerve at operation and thereby reduce the likelihood of nerve damage. Hvidegaard and colleagues (1983) describe a method in which electrical stimulation of the recurrent nerve causes adduction of the vocal cord and is registered by means of a balloon placed between the vocal cords.

Definitive Treatment

A procedure similar to that for assessment of paralysis of the facial nerve and the facial muscles using electrophysiologic methods is not generally routinely available for the recurrent laryngeal nerve. The chance of spontaneous return of function therefore cannot be assessed, and there is thus no early objective criteria on which to decide whether an operation is required for a recurrent laryngeal nerve paralysis. Recovery of the recurrent laryngeal nerve and return of full movement to the paralyzed vocal cord is possible for up to 12 months. Since an ankylosis of the cricoarytenoid joint resulting from the paralysis has not been demonstrated within 6 months, an expectant policy is advised for at least 6 to 12 months before definitive treatment of the larynx.

Early Exploration, Decompression, and Primary Neuroorrhaphy

When paralysis follows surgery to the neck, however, the question of early exploration of the neck arises. Although some patients have recovered vocal cord function after exploration, some have recovered without surgery; it is therefore difficult to assess the results of surgery. Occasionally, a ligature is found around a nerve and removed. Ogura (1961) described decompression by subperichondrial removal of the posteroinferior part of the thyroid cartilage with the inferior cornu for an idiopathic nerve paralysis. So far evidence of its efficacy is lacking. In patients with anterior cervical trauma, decompression of the recurrent laryngeal nerve has been advocated but only when spontaneous recovery has not occurred after several months.

In 1909, Horsley reported the first successful repair of the recurrent laryngeal nerve. He performed neuroorrhaphy in a 40-year-old woman whose recurrent laryngeal nerve had been severed by a bullet from a pistol. In 1928, Lahey reported a similar case, but he admitted later (Lahey and Hoover, 1938) that he had failed to demonstrate return of vocal cord function.

Frazier and Mosser (1926), Colledge (1925), and Ballance (1928) all reported repairs of the recurrent laryngeal nerve. In 1927, Colledge and Ballance first noted the problem of paradoxical movement of the cord, that is, abduction of the cord on expiration and adduction of the cord on inspiration, following simple suture anastomosis of the nerve. Following introduction and refinement of electromyography, Siribodhi (1963) was able to help explain these paradoxical movements that were seen after recurrent laryngeal nerve repair: adductor muscles had been reinnervated by both adductor and abductor nerve fibers and the abductor muscle had been reinnervated by both abductor and adductor nerve fibers. This misdirected reinnervation results in muscular competition for control of the arytenoid cartilage by adductors and the abductor in both inspiration and expiration. The clinical results of

anastomosis of the recurrent laryngeal nerve is therefore a vocal cord that neither abducts nor adducts but bulges medially during inspiration. If both recurrent laryngeal nerves have been repaired and both vocal cords so afflicted, airway compromise results. The only positive advantage of neuroorrhaphy is that if only one cord is paralyzed and reinnervated randomly as previously described, the voice may be better than if the cord had remained paralyzed, but this is debatable. Further, in 1982, Salassa and colleagues described partial airway obstruction resulting from medial bulging of a unilateral reinnervated vocal cord. This airway obstruction may represent one of the major deterrents to anastomosis of this nerve as a means of providing vocal cord tone for phonation following unilateral paralysis. At present, therefore, primary neurography is rarely used in the treatment of unilateral vocal cord paralysis.

Unilateral Paralysis

Unless the nerve is mechanically interrupted, recovery from vocal cord paralysis is the rule rather than the exception, but most recoveries take up to a year. In most patients who do not recover recurrent nerve function, the contralateral vocal cord compensates by moving across the midline to achieve a satisfactory glottic closure, restoring a reasonable voice, preventing aspiration, and producing an adequate cough. Hence, the policy of 6 months of watching and waiting for recovery or compensation, accompanied by speech therapy, should be followed. However, if the paralysis is the result of a carcinoma, especially of the lung, or from a cause from which one would not expect complete or adequate recovery, the distress of a weak voice and inefficient cough should be alleviated earlier. For the professional voice user, it might again be wise to consider early intervention to allow the patient a quick return to employment.

When recovery or compensation does not occur, the patient can be offered further treatment and may compensate if the free margin of the paralyzed cord can be displaced medially enough for the opposite cord to oppose it during swallowing and speech.

Intracordal injection of polytetrafluoroethylene (Teflon) paste has largely replaced the laryngofissure approaches of the past in which slivers of cartilage were inserted into the anterior end of the vocal cord at open operation via a median thyrotomy approach (Meurmann, 1952). Various materials have been injected into the paralyzed vocal cord in an attempt to move it from a paramedian to a midline position. In 1911, Bruning successfully treated patients who had unilateral vocal cord paralysis by injecting paraffin. This gave good but short-lived results. Embolization, drifting of the paraffin, and paraffinoma led to abandonment of this procedure. Glycerine, tantalum powder, cartilage, and ground bone have all been tried, but their use was discontinued after the introduction of polytetrafluoroethylene paste and silicone injections. Silicone, although initially a promising material, sets up no local reaction and, therefore, does not remain in place beyond a few months. As a result, the initial improvement to the voice quality is lost. A polytetrafluoroethylene implant goes through a number of changes in the weeks following its injection. The glycerine in which the polytetrafluoroethylene shreds are suspended is absorbed, a marked granulomatous reaction takes place, and the final depot of polytetrafluoroethylene granules is of slightly smaller volume than the original implant. Polytetrafluoroethylene granules, from 7 to 100 microns in diameter, do not migrate far from their original site of implantation. After more than 2 decades of use there is no evidence that polytetrafluoroethylene implantation is carcinogenic. Absorbable gelatin sponge (Gelfoam) paste may be used instead of polytetrafluoroethylene

if the paralysis is judged to be temporary. Lewy (1964), Rubin (1965), Siegler (1967), and Dedo and colleagues (1973) have all reported successful results with intracordal injection of polytetrafluoroethylene in large numbers of patients.

Polytetrafluoroethylene injection is best performed under local anesthesia because it allows the patient to phonate on command. Changes in the cord size, position, and relationships are observed, and voice tone may be monitored. Voice quality improvement during the procedure is an important and reliable guide and protects against excess injection. The anterior floor of the mouth, the pharynx, and the larynx are anesthetized with 5 per cent cocaine. To anesthetize the trachea (to stop coughing), 2 per cent cocaine is dropped between the cords onto the mucosa of the trachea. A Brünings syringe is charged with polytetrafluoroethylene paste. The syringe has a very long needle and is connected by a pistol grip with a ratchet handle, so designed that closure of one notch of the handle delivers 0.1 mL of polytetrafluoroethylene paste. The larynx is exposed by direct laryngoscopy. An anterior commissure laryngoscope is used because it fixes the vocal cords, making it easier to inject the paste into the larynx. On the paralyzed side, the upper surface of the vocal cord can be exposed by retracting the false vocal cord laterally with the end of the needle. The needle is then inserted into the soft tissues lateral to the vocal cord, midway between the anterior commissure and the vocal process of the arytenoid cartilage, and the handle is closed one notch. The paste extrudes slowly and the needle must be left in place several seconds during which the vocal cord can be seen to move slowly toward the midline. Once the position becomes stationary, the patient is asked to say "E", and if the mobile cord does not approximate the paralyzed cord, another 0.1 mL is injected. It should be necessary to repeat this procedure at the most three times; once the cords are approximated, the procedure is finished. Paste should not be injected into the subglottic space or into the edge of the vocal cord or too far laterally, as shown in the diagram. Some authors advocate the use of a needle guard to prevent advancing the needle too far. The ideal depth in any particular case, however, will vary with the size of the larynx. Also, the needle guard may give a false sense of security in that injections beyond the ideal depth can result as the needle guard indents the mucous membrane of the laryngeal ventricle. The patient can eat and talk on the same day and can leave the hospital the following day. Pain is usual but mild and can be treated with simple analgesics. Occasionally, infection occurs and should be treated with antibiotics. Laryngeal obstruction can occur if too much polytetrafluoroethylene has been injected, particularly if infection supervenes. The obstruction is treated with antibiotics, steroids, and humidification and, if necessary, a temporary tracheostomy. Because of the danger of laryngeal obstruction, it is better to inject too little than too much, because it is always possible to come back later and give a further injection.

Injection of a cord palsied by loss of the external and recurrent laryngeal nerves is much less satisfactory than in the case of isolated recurrent nerve palsy. In most cases, the volume is restored to a varying degree, but the timbre of vocal cord palsy remains. The bulked, rigid, implanted cord probably still requires the cricothyroid muscle to tense it for the functioning vocal cord to occlude adequately and produce improved phonation.

If the paralysis is due to laryngeal trauma, the injection of polytetrafluoroethylene will not be successful because of the replacement of the thyroarytenoid muscle with scar tissue. The best alternative is to attempt to add bulk laterally and push the scarred glottis medially. This can be done by inserting a piece of thyroid cartilage between the thyroid lamina and its

internal perichondrium (Smith, 1972).

Unilateral vagal paralysis above the nodose ganglion results not only in paralysis of the larynx but also paralysis of the pharyngeal muscles. The overriding therapeutic concern in such a patient is the prevention of aspiration. A cuffed tracheostomy tube and a nasogastric tube are essential. Vocal cord augmentation by polytetrafluoroethylene injection aids glottic closure but is never completely effective in preventing aspiration. A cricopharyngeal myotomy facilitates swallowing and minimizes stasis of saliva. Salivary production may also be reduced by dividing the chorda tympani and tympanic nerves. A number of these measures in combination usually succeed in rehabilitating the patient and allow the tracheostomy and nasogastric tubes to be removed.

Large series have shown the technique of injection of polytetrafluoroethylene to be a safe, well-tolerated, inexpensive, easily performed, and expeditious treatment for unilateral vocal cord paralysis. Nonetheless, several investigators have described operative procedures designed to reinnervate the paralyzed laryngeal muscles rather than rely on the replacement of muscle bulk with polytetrafluoroethylene. Indeed, the phonatory function of the vocal cords is best served by two vocal cords with equal muscle tension and mass and with symmetric positions. High-speed glottic motion pictures reveal that normal vocal cords beat and undulate symmetrically and synchronously; a paralyzed vocal cord injected with a bolus of polytetrafluoroethylene is not capable of this behavior. Thus, reinnervation techniques are being pursued to improve the voices of patients with unilateral vocal cord paralysis. Tucker (1977) has described unilateral reinnervation using a nerve-muscle pedicle. The nerve-muscle pedicle consists of a branch of the ansa hypoglossi, which is traced distally to a 2-mm to 3-mm block of omohyoid muscle. The nerve-muscle pedicle is then isolated for a sufficient length to reach the larynx and is passed through a window cut in the lamina of the thyroid cartilage, at which point it is approximated to the lateral thyroarytenoid muscle. Takenouchi and Okamoto (1973) have also used this technique. These authors report an improved voice and resolution of aspiration in some cases.

As previously described, recurrent laryngeal nerve neurotomy is not a workable option. Misdirected regeneration results in vocal cord spasm and medial inspiratory bulging, and the vocal cord does not regain normal phonatory capabilities, despite reinnervation. Experimental nerve transfers have been reported since the 1920s (Colledge and Ballance, 1927). Crumley and Izdebski (1986) recently described a patient with unilateral vocal cord paralysis following proximal transection of the vagus nerve in whom the ipsilateral ansa hypoglossi nerve was anastomosed to the recurrent laryngeal nerve and the contralateral ansa hypoglossi was implanted into the ipsilateral cricothyroid muscle. The patient gained an excellent voice that sounded normal to several experienced listeners, with a nearly normal voice print oscillographic analysis. Such procedures for unilateral paralysis are to a certain degree experimental and should be performed by clinicians experienced in treating laryngeal paralysis. At this time, nerve transfers in such cases should be left to experienced investigators and the time-tested technique of polytetrafluoroethylene injection should be used for the routine management of symptomatic, persistent, unilateral vocal cord paralysis.

Bilateral Abductor Paralysis

The most common cause of bilateral abductor paralysis is thyroidectomy. If the injury is to the recurrent laryngeal nerves only, the vocal cords assume a paramedian position with a good voice but an inadequate airway. Sooner or later, all patients with bilateral abductor paralysis have stridor. This may be present immediately after operation or may be precipitated later by an upper respiratory tract infection. The immediate need is a satisfactory airway, so a tracheostomy is required.

If the paralysis results from surgery and if the status of the nerve is unknown, one should wait at least 6 to 12 months before attempting therapy. The same is true in the rare case of bilateral idiopathic paralysis. It is advisable to await recovery, allowing the patient to be extubated. If further operation is needed, one can then assess which side recovers most, because an operation will then be done on the other cord.

The form of therapy used to provide an adequate airway depends upon the patient's individual needs and preferences. There are those patients, particularly professional voice users, who will want to maintain a permanent tracheostomy, and a normal voice. There are others who will prefer decannulation by widening of the airway. However, if the widening is passive, there will be some loss of voice. The airway may be passively enlarged at the constricted glottis by unilateral displacement of a vocal cord laterally or by removing part of one vocal cord. The greater the lateralization achieved, the greater the chance of aspiration and the greater the degree of hoarseness. For the same reason, bilateral arytenoidectomy is contraindicated because aspiration can be catastrophic.

Corpectomy. Early attempts to relieve airway obstruction secondary to vocal cord paralysis included open or endoscopic resection of one or both vocal cords; they usually failed because granulation and scar tissue later narrowed the airway. In 1922, Chevalier Jackson described the operation of ventriculocorpectomy, in which he left the arytenoid cartilage in situ. Kirchner (1979) has described a debulking procedure in which the thyroarytenoid muscle is removed endoscopically using microcautery. The preserved mucosa of the cord is held in place by sutures passed transcutaneously with spinal needles. The arytenoid cartilage is left in place, preventing the occasional posterior web secondary to arytenoidectomy. Corpectomy, with or without arytenoidectomy, has returned to fashion with the advent of the laser; it is relatively easy to perform and is without serious complications.

Lysis of the External Branch of the Superior Laryngeal Nerve. Tschiasny (1957) described denervation or removal of the cricothyroid muscle. The vocal cord assumes a more lateral position provided that the arytenoid cartilage is mobile and the cricothyroid muscle functions before the procedure. The voice quality was not discussed, but the operation means loss of the last tensor of the vocal cord.

Laterofixation of the Cord. In 1939, King used a lateral approach to gain access to the larynx via the inferior constrictor muscle, suturing the arytenoid cartilage to the severed omohyoid muscle. This was intended to provide a dynamic muscle sling but he also described suturing the arytenoid cartilage to the thyroid ala to counter the effects of scar tissue on the cricoarytenoid joint and was therefore the first surgeon to lateralize and fix the arytenoid cartilage.

Subsequent modifications of this operation took place until in 1946 Woodman described a lateral laryngeal approach to perform an arytenoidectomy. He removed the body of the arytenoid cartilage and fixed its vocal process laterally to the thyroid cartilage. In 1953, Woodman reported a collected series of 521 cases operated by 90 different surgeons, with a better than 90 per cent success rate of decannulation when the operations were done on patients with no previous history of attempts at lateralizing surgery. This operation remains a popular procedure for managing bilateral vocal cord paralysis, and it produces a satisfactory airway when the posterior commissure is between 4 and 6 mm, but when it is greater than 6 mm, a poor voice results. The patient always has a tracheostomy, so a general anesthetic is given by a cuffed endotracheal tube. A horizontal skin incision on one side of the neck is made at the level of the lower border of the thyroid cartilage. The larynx is mobilized by dissecting medial to the sternomastoid muscle and the carotid sheath. The posterior border of the thyroid cartilage is cut down on through the fibers of the inferior constrictor muscle. At the level of the lower border of the thyroid cartilage, the space between the upper end of the trachea and the upper end of the esophagus is opened up, and the posterior surface of the cricoid cartilage is exposed. This surface is followed upward as far as the cricoarytenoid joint. Using a small elevator, the arytenoid cartilage is dissected free of all its connections. Particular care is taken when dissecting on the medial part of the cartilage not to damage the laryngeal mucosa. The vocal process of the cartilage is divided, and the main body of the cartilage is removed. A 2-0 silk suture on a small round-bodied needle is placed through the vocal cord in the region of the vocal process and then through the posterior part of the lamina of the thyroid cartilage; one turn of a tie is then thrown. It is important to assess the state of the laryngeal airway at this stage, so the larynx is examined with a direct laryngoscope from above. The gap between the vocal cords at the posterior end should be about 4 mm. If this is so, after changing gowns and gloves and placing a fresh towel over the head of the patient, the tie already made is completed and the wound is closed. If the gap is not big enough, the suture is tied tighter, and after doing this, it is again checked by direct laryngoscopy. The wound is closed in the usual way with suction drainage.

A new method of laterofixation was described in 1984 by Ejnell and colleagues. A nylon thread is passed through hypodermic needles inserted through the thyroid cartilage. The needles are withdrawn and the nylon thread is tightened to form a permanent loop around the vocal cord. The advantages claimed are that no perioperative tracheostomy is needed, the influence on the voice is adjustable, and if needed there remains the possibility of performing a more extensive surgical procedure.

Arytenoidectomy and Arytenoidopexy. Arytenoidectomy and arytenoidopexy attempt the compromise of constructing an airway posteriorly while the cord is in passive apposition anteriorly for phonation. In 1949, Thornell described endolaryngeal arytenoidectomy by a submucosal resection of the arytenoid cartilage. The deep tissues were cauterized with the intention of causing retraction of the vocal cord laterally. In 1968, Downey and Kennon suggested laryngofissure as an approach through which arytenoidectomy could be performed. Montgomery (1961) has described the operation of arytenoidopexy in which the arytenoid cartilage is fixed laterally with a pin to the cricoid cartilage. In 1984, Ossoff and co-workers reported successful restoration of the airway and decannulation in 10 of 11 patients in whom arytenoidectomy was performed by vaporization with a CO₂ laser.

Reinnervation. The restoration of phasic vocal cord abduction by selective reinnervation of the paralyzed posterior cricoarytenoid muscle remains controversial. Two basic types of reinnervation procedures have been described. One is the nerve-muscle pedicle first described as a procedure to restore abduction in the paralyzed larynx (Tucker et al, 1970). In 1976, Tucker presented a series of seven patients who had undergone the procedure: he stated that in only five of them had sufficient time elapsed to evaluate the results. He noted return of spontaneous abduction of the reinnervated vocal cords during inspiration in all five patients. A segment of the omohyoid muscle with its innervation from the ansa hypoglossi is sutured into the posterior cricoarytenoid muscle. Sadly his results have not been reproduced by other workers for whom the nerve-muscle pedicle has always failed to produce motion of the vocal cords despite numerous attempts (Rice, 1984). Irrefutable proof of its efficacy in humans is not available. Only three institutions have reported limited subjective success, although there are experimental data suggesting that reinnervation may not be occurring but rather that it is lateral scarring from the operative procedure that widens the airway and stabilizes the arytenoid cartilage. Fixation of the arytenoid cartilage then allows the pull of the sternothyroid muscle on the thyroid cartilage to become transferred to the arytenoid cartilage, thereby enhancing the effect of the sternothyroid muscle on glottic enlargement. Arytenoid cartilage stabilization provides the cricothyroid muscle with a more physiologic fulcrum and allows it once again to lengthen the vocal cord. In Crumley's opinion, these appear to be the methods by which the neuromuscular pedicle technique produces airway improvement and are in keeping with the rapid postoperative success reported.

In the second technique, either the phrenic (Rice, 1982; Crumley, 1982) or the ansa cervicalis nerve (Rice and Burstein, 1983) is anastomosed to the abductor branch of the recurrent laryngeal nerve. Although the technique has been successful in animals, only Rice has a patient who appears to show reinnervated abduction.

In addition to reinnervation techniques, other methods of re-energizing the paralyzed muscle have been investigated. Investigations have shown experimentally that electrical stimuli can trigger the laryngeal muscles. Zelear and Dedo (1977) described the successful repetitive stimulation of a unilaterally paralyzed cricoarytenoid muscle triggered by the opposite muscle's contraction. However, clinical trials with such devices have been limited by delays in developing satisfactory implantable devices.

Today a conservative traditional approach is appropriate in the management of bilateral abductor paralysis. The patient should be allowed to choose between a tracheostomy and a lateralization procedure, largely depending on how important the quality of voice is.

Complete Laryngeal Paralysis

Complete laryngeal paralysis occurs when both superior and recurrent laryngeal innervation is interrupted. The cords lie in intermediate positions and invite chronic uncontrollable aspiration that will, if untreated, result in the death of the patient. Short-term management includes use of a cuffed tracheostomy tube, for protection and toilet of the tracheobronchial tree, and a nasogastric tube. Although vocal cord augmentation and cricopharyngeal myotomy may help, the respiratory and alimentary tracts usually require separation by surgical means. If the neurologic condition is not progressive, a total

laryngectomy should be seriously considered. However, an alternative is surgical closure of the larynx, which has the advantage that the operation is reversible if the patient should gain sufficient return of neuromuscular function. The epiglottic flap operation of Habal and Murray (1972), Lindeman's diversion procedure (1975), and Montgomery's glottic closure procedure (1975) have all been successful in the prevention of aspiration. Habal described sealing the supraglottic larynx by suturing the epiglottis as a flap over the arytenoid and aryepiglottic folds. The airway is protected, but laryngeal function is difficult to monitor. This approach through the vallecula endangers the superior laryngeal nerves, which could further decrease the possibility of rehabilitation of the larynx.

Lindeman severs the trachea, forms a tracheal stoma from the lower end, and anastomoses the upper end to the esophagus so that aspirated materials are diverted back into the esophagus. The procedure was successfully performed and reversed in dogs. One patient has undergone the procedure, but the reversal procedure had not been performed at the time of the report. The advantage of the procedure is the ability to monitor the function of the larynx by indirect laryngoscopy. A disadvantage, however, is that the amount of surgery performed near the recurrent nerves plus the subsequent scarring puts the nerves in jeopardy.

Montgomery reported successful closure of the larynx in 12 patients. Mucosa was removed from the free edges of the vocal folds, the posterior commissure, and the false vocal cords via a midline thyrotomy. The areas were approximated by deep sutures running most of the length of the vocal cords, which were anchored to the external surface of the opposite thyroid lamina. Although described as reversible, none of his patients had recovered sufficiently to warrant reversal. Monitoring return of function would be difficult; electromyography would probably be required. An epiglottopexy operation has recently been described by Brookes and McKelvie (1983). The epiglottis is laterally divided and sutured to a similarly prepared aryepiglottic fold, leaving a small posterior central opening 2 to 3 mm in size. This provides adequate protection of the tracheobronchial tree, and with a valved tracheostomy tube in situ it allows adequate protection of the tracheobronchial tree and adequate respiration and phonation. Again, this procedure is reversible.

Treatment of Vocal Cord Paralysis in Children

Initial therapy is conservative because 50 per cent of cases of vocal cord paralysis in children resolve (Gentile et al, 1986; Swift and Rogers, 1987). It is possible that the other 50 per cent of patients form the so-called idiopathic lesions seen in adult life. However, these babies have stormy infancies with repeated respiratory infections and very slow weight gain. Since the swallowing of liquids presents difficulties for these children, an improvement in the general condition can often be brought about by the introduction of a semisolid weaning diet at an earlier age than usual. In some children with hydrocephalus, cerebrospinal fluid shunting results in resolution of the laryngeal paralysis (Bluestone et al, 1972), and the results of posterior fossa decompression in treating children with hindbrain malformations and vocal cord paralysis are promising (Wickramasinghe et al, 1968; Hoffman et al, 1975). Some children with bilateral vocal cord paralysis but without hydrocephalus may be steered through childhood without recourse to tracheostomy. If a tracheostomy has to be performed, it must be maintained until the age of 14 to 16 years when further surgery such as arytenoidectomy can be considered. At this age, the child can take an intelligent part in deciding whether or not to exchange voice for airway, after a lengthy opportunity to assess how well tracheostomy

can be incorporated into daily living.