K. J. Lee: Essential Otolaryngology and Head and Neck Surgery (IIIrd Ed)

Chapter 15: The Larynx

Embryology of the Larynx (see Chap. 11, pages 306-310)

Anatomy

Anatomy

The larynx consists of a framework of cartilages, held in position by an intrinsic and extrinsic musculature, and lined by mucous membrane which is arranged in characteristic folds.

The larynx is situated in front of the fourth, fifth, and sixth cervical vertebrae. The upper portion of the larynx, which is continuous with the pharynx above, is almost triangular in shape; the lower portion leading into trachea presents a circular appearance.

Laryngeal Cartilages

The laryngeal cartilages form the main framework of the larynx and consist of:

1. Thyroid cartilage (unpaired).
2. Cricoid cartilage (unpaired).
3. Epiglottis (unpaired).
4. Arytenoid cartilage (paired).
5. Corniculate cartilage (paired).
6. Cuneiform cartilage (paired).

Thyroid Cartilage

The thyroid cartilage (hyaline cartilage) is the largest and encloses the larynx anteriorly and laterally, thus shielding it from all but the most forceful blows. This cartilage is composed of two alae which meet anteriorly, dipping down from above to form the thyroid notch before meeting at the protuberance of the Adam's apple. Posteriorly, each wing has a superior cornu, extending upward about 2 cm, and a much shorter inferior cornu which articulates with the cricoid cartilage below. This is the only direct articulation of the thyroid cartilage, all other relationships with contiguous structures being maintained by muscles or ligaments.

Cricoid Cartilage

The cricoid cartilage (hyaline cartilage) lies directly below the thyroid cartilage. It is the strongest of the laryngeal cartilages, and is shaped like a signet ring. The flat portion of the ring or lamina is located posteriorly and extends upward to form the posterior border of the larynx. Since the cricoid cartilage forms the only complete annular support of the laryngeal skeleton, its preservation is essential for the maintenance of the enclosed airway.

In the adult, the cricoid cartilage is at the level of C-6, C-7 and in the child at the level of C-3, C-4.

Posterolaterally, the cricoid articulates with the inferior cornua of the thyroid cartilage with which it shares true synovial joints. These joints permit a rocking action of the cricoid cartilage on the thyroid cartilage and also a slight anteroposterior sliding motion. Also through synovial joints, the cricoid cartilage on its posterosuperior aspect supports the two arytenoid cartilages.

Epiglottis

The epiglottis (fibroelastic cartilage) is a leaf-shaped structure attached to the inside of the thyroid cartilage anteriorly and projecting upward and backward above the laryngeal opening. The petiole is the small, narrow portion of the epiglottis that is attached to the thyroid cartilage.

Arytenoid Cartilages

The arytenoid cartilages (mostly hyaline cartilages) are much smaller in size, yet they are primarily responsible for the opening and closing of the larynx. Roughly pyramidal in shape, they rest on the upper edge of the cricoid lamina at the posterior border of the larynx. The anterior projection of each arytenoid, or vocal process, receives the attachment of the posterior or mobile end of each vocal cord. The lateral prominence of each arytenoid cartilage is known as the muscular process because of the insertion of numerous muscles. The arytenoids articulate with the cricoid cartilage at the cricoarytenoid joint, which permits a wide range of motion in three directions.

Corniculate Cartilages

The corniculate cartilages (fibroelastic cartilages), also called cartilages of Santorini, are small cartilages above the arytenoid and in the aryepiglottic folds.

Cuneiform Cartilages

The cuneiform cartilages (fibroelastic cartilages), also called cartilages of Wrisberg, are elongated pieces of small yellow elastic cartilages in the aryepiglotic folds.

Triticeous Cartilage

The triticeous cartilage (cartilage triticea) is a small elastic cartilage in the lateral thyrohyoid ligament. When calcified, it could be mistaken for a foreign body on the soft tissue x-ray film.

Ossification

1. Thyroid cartilage ossifies at 20-30 years of age. Ossification begins in the inferior margin and progresses cranially.

2. Cricoid cartilage ossifies after the thyroid cartilage. The first part to be calcified is the superior portion and could be mistaken for a foreign body. Calcification progresses caudally.

3. Arytenoid cartilages calcify at the third decade.

4. The hyoid ossifies from six centers shortly after birth and is completed by 2 years of age.

Laryngeal Ligaments and Membrane

Extrinsic

The extrinsic ligaments of the larynx bind the cartilages to the adjoining structures and to one another, and round out the laryngeal framework.

1. Thyrohyoid membrane and ligaments attach the thyroid cartilage to the hyoid bone. The thyrohyoid membrane is pierced on each side by: (a) superior laryngeal vessels; (b) internal branch of superior laryngeal nerve. Median thyrohyoid ligament is the thickened median portion of the thyrohyoid membrane. Lateral thyrohyoid ligament forms the thickened posterior border of the thyrohyoid membrane on each side, and the cartilago triticea is often found in this ligament.

2. Cricothyroid membrane and ligaments connect the thyroid and cricoid cartilages. This ligament may be pierced for emergency tracheotomy (cricothyrotomy) with little fear of bleeding. However, because of the proximity of the vocal cords, this space should not be used for prolonged tracheal intubation since scar tissue may be produced to interfere with the mobility of the cords.

3. The cricotracheal ligament attaches the cricoid cartilage to the first tracheal ring.

4. The epiglottis is suspended in position by membranous connections to the hyoid bone, the thyroid cartilage, and the base of the tongue.

Intrinsic

The intrinsic ligaments unite the cartilages of the larynx and perform an important role in the closure of this organ.

1. The elastic membrane of larynx is the fibrous framework of the larynx. It lies beneath the laryngeal mucosa and is divided into upper and lower parts by the ventricle of the larynx.

2. The quadrangular membrane is the upper part of the elastic membrane of the larynx, extending from the lateral margin of the epiglottis to the arytenoid and corniculate cartilages, and inferiorly to the false cord. It forms part of the wall between the upper pyriform sinus and the laryngeal vestibule. The quadrangular membrane and the conus elasticus are separated by the ventricle of Morgagni.

3. Conus elasticus (cricovocal membrane) is the name given to the lower part of the elastic membrane of the larynx. It is composed mainly of yellow elastic tissue. It is attached to:

a. Inferiorly: Superior border of the cricoid cartilage.
b. Superoanteriorly: Deep surface of angle of the thyroid cartilage.
c. Superoposteriorly: Vocal process of the arytenoid cartilage.

4. The median cricothyroid ligament is formed by the thickened bottom part of the conus elasticus.

5. The vocal ligament which forms the framework of the vocal cord is the free upper edge (the strongest part) of the conus elasticus.

6. The thyroepiglottic ligament attaches the epiglottis to the thyroid cartilage.

Cavity of the Larynx

1. The cavity of the larynx is divided into three parts:

a. Vestibule.
b. Ventricle.
c. Subglottic space

by two folds of mucous membrane:

a. False cords.
b. True cords.

2. The vestibule lies between the inlet and the edges of the false cords and is bordered by:

Anteriorly: Posterior surface of the epiglottis.
Posteriorly: Interval between the arytenoid cartilages.
Laterally: Inner surface of the aryepiglottic folds and upper surfaces of the false cord.

3. The ventricle of the larynx (ventricle of Morgagni) is a deep, spindle-shaped recess between the false and true cords, and lined by a mucous membrane which is covered externally by the thyroarytenoid muscle.

4. The saccule is a conical pouch which ascends from the anterior part of the ventricle. It lies between the inner surface of the thyroid cartilage and the false cord. Numerous mucous glands open on to the surface of its lining mucosa for lubricating the vocal cords.

5. The glottis (rima glottidis) is the space between the free margin of the true vocal cords. This space is wide and triangular in shape when the vocal cords are abducted (as in respiration), but assumes a slitlike appearance during adduction of the cords (in phonation).

The posterior glottic chink in the adult is 18-19 mm. In the newborn it is 4 mm. The total glottic chink in a newborn is 14 mm.

6. The subglottic space lies between the true vocal cords and the lower border of the cricoid cartilage.

7. The preepiglottic space is a wedge-shaped space lying in front of the epiglottis and bounded by:

Anteriorly: Thyrohyoid membrane.
Anterosuperiorly: Hyoid.
Superiorly: Vallecula.
Posteriorly: Part of the epiglottis.
Laterally: Hyoepiglottic ligament.

8. The false cords (ventricular bands) are the upper set of two horizontal folds on each side of the laryngeal cavity and extend from the angle of the thyroid cartilage anteriorly to the bodies of the arytenoid cartilages posteriorly, and have a primitive constricting function.

9. The true cords (the lower set) are directly concerned with the production of voice and with the protection of the lower respiratory passages. These folds stretch from the angle of the thyroid cartilage anteriorly to the vocal processes of the arytenoid cartilages posteriorly. They enclose the vocal ligament and a major portion of the vocalis muscle. The covering epithelium is closely bound down to the underlying vocal ligament, and the blood supply is poor. Hence the pearly white appearance of the vocal cords in life.

Laryngeal Joints

Cricothyroid Joint

The cricothyroid joint is a synovial joint with a capsular ligament between the inferior cornu of the thyroid cartilage and the facet on the cricoid cartilage at the junction of arch and lamina. Two movements occur:

1. Rotation: Through a transverse axis.
2. Gliding: Slightly.

Cricoarytenoid Joint

The cricoarytenoid joint is a synovial joint with a capsular ligament between the base of the arytenoid cartilage and the facet on the upper border of the lamina of the cricoid cartilage. Two movements occur:

1. Rotation: Of the arytenoid, on a vertical axis. The vocal process moves medially or laterally.
2. Gliding: The arytenoids move toward or away from each other. A strong posteior cricoarytenoid ligament prevents excessive movements of the arytenoid on the cricoid.

Laryngeal Muscles

1. The extrinsic muscles of the larynx are concerned with the movement and fixation of the larynx as a whole and consist of levator and depressor groups.

The depressor group consists of:

1. Sternohyoid (C-2, C-3).
2. Thyrohyoid (C-1).
3. Omohyoid (C-2, C-3).

The elevator group consists of:

1. Geniohyoid (C-1).
2. Digastrics (anterior, cranial nerve V and posterior VII).
3. Mylohyoid (V).
4. Stylohyoid (VII).

2. The middle constrictor muscle is attached to the greater cornua of the hyoid bone. The inferior constrictor muscle is attached to the oblique lines of the thyroid cartilage and to the cricoid cartilage. These muscles influence the position of the larynx during phonation.

3. The intrinsic muscles of the larynx are directly concerned with its protective and phonatory functions, and consist of one unpaired muscle, the transverse arytenoid, and four paired muscles which act on the cricoarytenoid and cricothyroid joints, respectively.

1. Interarytenoid muscle (unpaired).

a. Transverse.
b. Oblique.

2. Posterior cricoarytenoid muscle (paired).

3. Lateral cricoarytenoid muscle (paired).

4. Thyroarytenoid muscle (paired).

5. Cricothyroid muscle (paired).

4. The interarytenoid muscle is unpaired and consists of transverse and oblique fibers connecting the bodies of two arytenoid cartilages. This muscle is innervated bilaterally by the recurrent laryngeal nerve and therefore is not paralyzed by unilateral recurrent nerve disease. Action: Approximation of arytenoids and closure of glottis.

5. The posterior cricoarytenoid muscle passes from the posterior surface of the cricoid lamina to the muscular process of the arytenoid cartilage. Action: Lateral rotation of arytenoids and abduction of vocal cords (main abductor).

6. The lateral cricoarytenoid muscle passes from the cricoid arch to the muscular process of the arytenoid cartilage. Action: Medial rotation of arytenoids and adduction of vocal cords.

7. The thyroarytenoid or vocalis muscle arises from the inner aspect of the thyroid angle anteriorly and inserts into the vocal ligament and into the arytenoid cartilage. Action: Fine control of the vocal cords - relaxation of cord, firming of edge, changing of mass, etc.

8. The cricothyroid muscle arises from the arch of the cricoid anteriorly, and inserts into the inferior horn and body of the thyroid cartilage above. It elevates the arch of the cricoid cartilage as a lever, and thus tilts the lamina with the attached arytenoid cartilages posteriorly. Action: Elongation and tension of the cords (chief tensor).

Recent data suggest this muscle plays a major role in the overall regulation of breathing by its control of expiratory resistance and flow. Cricothyroid contraction in expiration results in vocal elongation increasing glottic size. Such a mechanical effect reduces airway resistance and shortens expiratory duration.

9. Laryngeal movements:

Abduction: Posterior cricoarytenoid.

Adduction:

a. Lateral cricoarytenoid.
b. Transverse portion of interarytenoid.
c. Thyroarytenoid.

Tension:

a. Cricothyroid (chief tensor).
b. Thyrarytenoid or vocalis (internal tensor).

Mucous Membrane of the Larynx

1. Stratified squamous epithelium is found over:

a. Vocal cords.
b. Upper part of vestibule of larynx.

2. Ciliated columnar epithelium lines the remainder of the cavity.

3. Mucous glands are found in:

a. Ventricles and sacculi.
b. Posterior surface of epiglottis.
c. Margins of aryepiglottic folds. There are none on the free edges of the vocal cords.

4. Reinke's layer of connective tissue lies immediately under the epithelium of the larynx and superficial to the elastic layer. There are no glands beneath it and no lymph vessels in it.

Nerve Supply

1. The larynx is supplied by two branches of vagus nerve: superior laryngeal and inferior (recurrent) laryngeal nerves.

2. The superior laryngeal nerve (SLN) divides extralaryngeal into:

a. Internal branch (sensory).
b. External branch (motor and sensory).

The larger internal branch supplies sensory innervation to those areas of the larynx above the glottis. The smaller external branch gives motor innervation to the cricothyroid muscle and sensory supply to the anterior infraglottic larynx at the level of the cricothyroid membrane.

3. The recurrent (or inferior) laryngeal nerve (RLN) supplies motor innervation to all the intrinsic laryngeal muscles of the same side except for the cricothyroid and to the interarytenoid muscle of both sides. It also supplies sensory innervation to those portions of the larynx below the glottis.

4. The recurrent laryngeal nerve has a much longer course on the left side than on the right. On the left side it turns around the arch of the aorta. On the right side it turns around the subclavian artery. In the neck it lies between the trachea and esophagus as it approaches the larynx. Its terminal part passes upwards, under cover of the ala of the thyroid cartilage, immediately behind the inferior cricothyroid joint.

5. Each nucleus ambiguus is the somatic motor nucleus of the 9th, 10th, and 11th nerves.

6. The nucleus ambiguus is supplied by the posterior inferior cerebellar artery (branch of the vertebral) and the anterior inferior cerebellar artery (branch of the basilar).

Blood Supply

Upper Larynx

1. External carotid artery.
2. Superior thyroid artery.
3. Superior laryngeal artery.

Lower Larynx

1. Subclavian artery.
2. Thyrocervical artery.
3. Inferior thyroid artery.
4. Inferior laryngeal artery.

Venous Drainage

Upper Larynx

1. Superior laryngeal vein.
2. Superior thyroid vein.
3. Internal jugular vein.

Lower Larynx

1. Inferior laryngeal vein.
2. Inferior thyroid vein.
3. Innominate vein.

Lymphatic Drainage

1. The lymphatic drainage from the larynx drain mainly into the deep cervical group of lymph nodes. It is of great clinical importance that the vocal cords themselves contain scarcely any lymphatic channels.

2. The lymphatic network of the supraglottic structures is extensive. The channels collect in a pedicle at the anteriro end of the aryepiglottic fold, pass laterally, anterior to the anterior wall of the pyriform fossa, and leave the larynx with the neurovascular bundle through the thyrohyoid membrane. Almost all (98%) of the channels end in the upper deep cervical nodes between the digastric tendon and the omohyoid muscle. The remainder pass to the lower cervical chain or the spinal accessory chain.

3. The lymphatics of the infraglottic area have a more variable drainage pattern than those of the supraglottic network. The channels leave the area in three pedicles. The anterior pedicle passes through the cricothyroid membrane and many vessels end in the prelaryngeal (Delphian) nodes in the region of the thyroid isthmus. Channels then leave these nodes with the remaining anterior channels to travel to the deep inferior cervical nodes. The two posterolateral pedicles leave the larynx through the cricotracheal membrane with some

channels going to leave through the cricotracheal membrane to the paratracheal chain of nodes, while others pass to the inferior jugular chain.

4. Generally, lymphatic drainage from each half of the larynx is quite separate and little crossover or mixing occurs.

5. There is evidence that lymphatic channels do cross the midline in the supra- and infraglottic areas. Contralateral drainage is more likely to occur spontaneously from the infraglottic areas, thus lesions of this area may be associated with less consistent patterns of metastases.

Physiology of the Larynx

Basic Funtions

Three basic functions of the larynx in order of importance are: protection, respiration, and phonation.

1. Protection by the larynx as a sphincter prevents the entrance of anything but air into the lung.

a. Closure of the laryngeal inlet.
b. Closure of the glottis.
c. Cessation of respiration.
d. Cough reflex, expulsion of secretions and foreign bodies.

2. Respiration governed by active muscular dilatation of the laryngeal aperture assists in the regulation of gaseous exchange with the lung and in the maintenance of acid-base balance.

3. Phonation is voice produced by the vibration of the vocal cords.

4. Other functions: Fixation of the chest is also a function of the larynx. Closure of glottis will help to increase intrathoracic and intra-abdominal pressure and aid in lifting, digging, defecation, vomiting, urination, or childbirth.

The protective function of the adult human larynx is admittedly precarious by virtue of its low position in the neck.

The human newborn exhibits a nasolaryngeal connection by approximation of the epiglottis with the posterior surface of the palate, thereby ensuring against aspiration by forming a continuous upper and lower airway. Obligate nasal breathing in the newborn period (1-6 months) is related to this anatomic configuration.

The epiglottis in the adult serves as a laryngeal shield to direct swallowed food laterally into the pyriform fossae and away from the midline laryngeal aperture. This protective function is enhanced by elevation of the larynx toward the nasal cavity during the

height of deglutition. The corniculate and cuneiform cartilages are contained in the aryepiglottic folds to provide stiffness and support to these structures.

Due to their structural configuration the false cords prevent the egress of air from the lungs (provides an expectorative function) whereas the true cords with their upturned margins are capable of impeding its ingress (protective function).

Neuromuscular Physiology

Afferent System

The density of sensory innervation is greatest in the laryngeal inlet and especially the laryngeal surface of the epiglottis, an observation consistent with its protective function of the distal respiratory tract. Afferent impulses are delivered through the ganglion nodosum to the brain stem tractus solitarius.

Efferent System

Motor distribution to the intrinsic laryngeal musculature originates in the medullary nucleus ambiguus. Each RLN innervates all muscles except for the cricothyroideus which is supplied by the external division of the SLN. The interarytenoid muscles receive bilateral motor innervation from both RLN.

The sole abductor is the posterior cricoarytenoid which is a muscle extending from the posterior aspect of the cricoid plate to the muscular process of the arytenoid. The major laryngeal adductors are the thyroarytenoid and lateral cricoarytenoid muscles. The cricothyroid muscle adducts and tenses the vocal cord, passively lengthening it by 30%. The interarytenoid muscles close the posterior gap in the glottis.

Paralysis of the SLN leads to denervation of the ipsilateral cricothyroid muscle, resulting in rotation of the posterior commissure toward the inactive side from unopposed contraction of the contralateral cricothyroid muscle. RLN injury results in a paramedian vocal cord position because of the adductor action of the intact SLN contracting the ipsilateral cricothyroid muscle.

Neurophysiology of Protective Function

Man does not possess a crossed adductor reflex, i.e. stimulation of one SLN does not produce simultaneous activation of the contralateral adductor musculature. Therefore, unilateral SLN paralysis may lead to aspiration (failure of ipsilateral cord closure) despite the integrity of both RLN.

There are three sphincteric tiers of airway protection:

1. Contraction of the superior division of the thyroarytenoid muscles contained in the aryepiglottic folds.

2. Contraction of middle thyroarytenoid fibers in the false cords.

3. Contraction of inferior division of the thyroarytenoid at the level of the true cord. This is the most significant barrier to aspiration owing to the upturned border of the cord margin.

Stimulation of the SLN, as well as all major cranial afferent nerves, special sensory, and spinal somatic sensory nerves, produces strong laryngeal adductor responses, emphasizing the primitive role of respiratory protection from a wide variety of potentially noxious stimuli. Laryngeal spasm is solely mediated by the SLN. SLN stimulates also produces inhibition of laryngeal abductor activity, resulting in various degrees of reflex apnoea.

Neurophysiology of Respiratory Function

Widening of the glottis occurs with rhythmic bursts of activity in the RLN. The glottis opens a fraction of a second before air is drawn in by the descent of the diaphragm. Electromyogram studies show that phasic inspiratory abduction, via muscular contraction of the posterior cricoarytenoids, is synchronous with respiration. The degree of abductor activity varies directly with the degree of ventilatory resistance (i.e. decreases with tracheotomy). Phasic inspiratory contraction of the cricothyroid muscle (vocal cord adductor and isotonic tensor) increases the anteroposterior diameter of the glottic chink. Therefore, both the posterior cricoarytenoid and cricothyroid muscles are driven by the medullary respiratory center.

Neurophysiology of Phonation

Speech results from the production of a fundamental tone produced at the larynx and is modified by resonating chambers of the upper aerodigestive tract. The vocal folds are positioned near the midline by isotonic tensing of the cricothyroid muscles. The thyroarytenoid muscles provide finer isometric modifications.

As pitch increases the true cords lengthen and tense isotonically through the action of the cricothyroid muscles. Cord thinning is produced by thyroarytenoid action which increases internal tension of the true cord. Extrinsic laryngeal muscles also may affect pitch (i.e. sternothyroid).

Laryngeal Voice Production

Myoelastic-Aerodynamic Theory. During expiration the air current flowing through the glottis is unidirectional and the vocal cords vibrate in an alternating mode. The sequence of events is as follows: The laryngeal muscles first position the vocal cords (various degrees of adduction) and place them under the appropriate longitudinal tension. Next muscular and passive forces of exhalation cause the subglottic air pressure to increase. Subglottic air pressure reaches a point where it exceeds muscular opposition and the glottic chink is forced open. When the vocal cords start opening from complete closure, they open in a posterior to anterior direction. Thus the posterior portion of the glottis is the firs to open, first to reach maximum excursion and first to recontact each other at the end of a vibratory cycle. After the release of the puff of air there is a reduction of subglottic pressure and the vocal cords approximate each other again. The myoelastic forces of the vocal cords exceed the aerodynamic forces. The myoelastic forces are enhanced because air current flowing through

a narrow channel exerts a negative pressure upon the channel walls (Bernoulli's effect). The vocal cords are thus sucked back together in an adducted state until the subglottic air pressure can overcome the myoelastic forces of the reapproximated cords, and the cycle is thereby repeated.

The resulting waveform of the vocal cords is not sinusoidal but sawtooth in type, and can be classified as a relaxation oscillator. An unvoiced output (glottis opened) is essentially a noise.

Neuromuscular or Neurochronaxic Theory. This theory, now disproved, suggested that each new vibratory cycle was initiated by central neuronal impulses via the vagus nerve to the appropriate laryngeal muscles. According to this theory the rate of impulses delivered to the larynx will determine the frequency of vocal cord vibration. Physiologic and audiometric analysis have led us to believe that this theory is untrue (i.e. voice is still produced in a patient with bilateral vocal cord palsy).

Components of Human Vocal Mechanism

1. Activator: Energy produced by the expiratory phase of the breathing apparatus.
2. Generator: Glottis vibrates at different frequencies.
3. Resonator: Sound modulation occurring in supraglottis, nasopharynx, oropharynx, and nasal chambers.
4. Articulators: Precise movements of palate, tongue, teet, and lips used to mold different sounds.

Glottic Closure Reflex - Control Mechanisms

Reflex laryngeal closure is produced by rapid contraction of the thyroarytenoid (TA) muscle in response to SNL stimulation. Exaggerated reflex glottis closure leads to laryngospasm which is maintained well beyond the cessation of mucosal irrigation. Obstructive apnea secondary to prolonged laryngospasm may produce death by acute hypoxia and hypercapnia. The body's fail-safe mechanism in dealing with this phenomenon is that laryngospasm is inhibited by (1) increased arterial PCO_2, (2) decreased arterial PO_2, (3) positive intrathoracic pressure, and (4) inspiratory phase of respiration. The most common causes of laryngospasm are inhaled irritants, manipulation of the upper aerodigestive tract (i.e. extubation), foreign bodies, and mucus or blood in the glottic chink.

Arrhythmia, bradycardia, and occasionally cardiac arrest may result from stimulating the larynx. The mechanism appears to be related to stimulation of nerve fibers which arise in aortic baroreceptors, and in some individuals, travel to the central nervous system by way of the recurrent laryngeal nerve, ramus communicans, and superior laryngeal nerve. These can result from light anesthesia, prolonged laryngoscopy, repeated attempts at intubation, respiratory obstruction, and tracheal irritation. The reflex cardiac effects can be controlled by atropine and enhanced by morphine.

Selected Disorders of the Larynx

Inflammatory Diseases

Acute Epiglottitis

Acute epiglottitis is a special form of rapidly progressing acute laryngitis in which the inflammatory changes primarily involve the epiglottis. It occurs mainly in children aged 2-7 years, although infants, older children, and even adults may be affected.

Etiology: Haemophilus influenzae type B

Symptoms:

1. Rapidly progressive dyspnea, especially in children. May be fatal within a few hours of onset unless immediately diagnosed and treated. This is a medical emergency!

2. Dysphagia starts with sore throat, difficulty in swallowing, then refusal of oral feedings.

3. Dehydration, fever, tachycardia, restlessness, exhaustion with respiratory and circulatory collapse.

4. The voice usually is not hoarse but may present with a "hot potato voice".

5. Patient prefers upright position and leaning slightly forward. Do not place the patient in a recumber position.

6. The most important clinical feature is the swollen, bright red epiglottis obstructing the pharynx at the base of the tongue.

7. A patient who is already in extreme respiratory distress may develop a total airway obstruction when an attempt is made to visualize the epiglottis. A quick look at the epiglottis in the emergency room with a tongue blade is to be condemned. Lateral x-ray of the neck is preferable to direct examination and will show an enlarged epiglottis.

8. Blood culture usually shows H. influenzae type B.

Treatment:

In the past decade, changes have occurred in the management of acute epiglottitis. Recent data suggest that nasotracheal intubation is the preferred method of treatment for acute epiglottitis over tracheotomy. The following steps of management are recommended:

1. The patient suspected of acute epiglottitis should be evaluated in the emergency room by a team consisting of a pediatrician, anesthesiologist, and otolaryngologist.

2. A lateral extended neck x-ray is obtained with a physician in attendance. Oral examination with a tongue depressor is not advised.

3. The patient is immediately taken to the operating room if the x-ray is diagnostic or progressive epiglottitis is suspected.

4. Orotracheal intubation is performed with both an anesthesiologist and an otolaryngologist present and prepared for immediate bronochoscopy and/or tracheotomy.

5. The otolaryngologist examines the epiglottis by direct laryngoscopy and the epiglottis and blood are cultured. The endotracheal tube is then replaced by a nasotracheal tube which is firmly secured by a tape and a string around the neck.

6. The patient is transferred to the ICU for close observation. Restraints to prevent accidental self-extubation may be needed.

7. Ampicillin 200-400 mg/kg and chloramphenicol, 50-100 mg/kg daily are administered intravenously. Hydration is managed by intravenous fluids. Frequent nasotracheal suction is needed.

8. Steroids may be of value in limiting the progression of inflammation and edema.

9. Extubation is done 24-48 hours after intubation if direct laryngoscopy at bedside shows a decrease in the supraglottic edema.

10. The patient is observed for an additional 24-48 hours and discharged with appropriate antibiotic therapy for a total of 10 days.

11. It is important to emphasize that the skilled nursing staff familiar with the management of acute cardiorespiratory problems is necessary following nasotracheal intubation, and that the anesthesiologist and otolaryngologist should always be available in the even of self-extubation. In an institution where the special 24-hour pediatric ICU is not available, it is safer to rely on the time-tested tracheotomy.

12. Since complete obstruction can occur with alarming suddenness, nasotracheal intubation or tracheotomy should be performed as soon as the diagnosis of acute epiglottitis is made.

Acute Laryngotracheobronchitis

Laryngotracheobronchitis is an acute infection of the lower respiratory passages, extending from the larynx down into the smalled subdivisions of the bronchial tree. It is endemic throughout the year, but may react in epidemic proportions in any locality during the winter season.

Etiology: Probably a virus. Parainfluenza types 1-4 have been isolated frequently. Haemophilus influenzae, streptococcus, staphylococcus, and pneumococcus are commonly cultured. The disease occurs in children, especially between the ages of 1-3 years.

Pathology: A descending inflammation of the mucous membrane lining the lower respiratory tract, followed by congestion, edema, and exudation of a thick tenacious secretion. Anatomically, the conus elasticus is the most involved site.

Symptoms:

1. At the onset, the disease is like an ordinary cold except for the early presence of a croupy cough.
2. Hoarseness is noted shortly thereafter.
3. As the swelling increases, inspiratory stridor develops.
4. Retractions then occur.
5. Circumoral pallor and cyanosis usually precede a decrease in breath sounds that, in turn, is an indication that death may be imminent. Immediate establishment of an airway is mandatory.
6. In addition to these symptoms of respiratory embarrassment, anorexia and fever are common in the early stages while restlessness, dehydration, and exhaustion may be noted later.
7. Agitation, increased pulse (to 140), and respiration rate (to 80) are signs of increasing levels of CO_2.

Treatment:

1. Hospitalization and close observation.
2. Ultrasonic humidification (a most important treatment).
3. Antibiotics (ampicillin 150 mg/kg/day).
4. Racemic epinephrine via intermittent positive pressure breathing (IPPB).
5. Corticosteroids (100 mg hydrocortisone IM on admission) may be helpful.
6. Sedation is contraindicated since it may compromise the airway.
7. Parenteral fluids.
8. Oxygen.
9. Timely nasotracheal intubation or tracheotomy. When in doubt, do it. Progressive retraction associated with agitation, cyanosis, lethargy, increased pulse (to 140), respiration rate (to 80), and CO_2 level is an indication for immediate nasotracheal intubation or tracheotomy.

Tuberculous Laryngitis

Etiology: Almost always secondary to active pulmonary tuberculosis.

Pathology:

1. Cellular infiltration.
2. Proliferation and nodular function.
3. Granulation tissue in the interarytenoid fold.
4. Perichondritis, cartilage necrosis.

a. Multiple small superficial ulcers in the interarytenoid fold, false and true cords, and the epiglottis.

b. Perichondritis and cartilage necrosis.

Symptoms:

1. Hoarseness.
2. Cough, late, with production of blood streaked sputum.
3. Pain and referred earache are fairly common.
4. In advanced cases, dyspnea from edema of the larynx and scar contraction or destruction of underlying cartilages.
5. Biopsy is essential for diagnosis to rule out malignancy. The tuberculous granuloma similar to that of pemphigoid is subepithelial. The pemphigus involvement is intraepithelial.
6. The most common site of tuberculosis of the larynx is the posterior larynx (interarytenoid fold). The next most common site is the laryngeal surface of the epiglottis.

Prognosis: If diagnosed and treated early, prognosis is good. If the local manifestations include cartilaginous involvement, the prognosis is more serious, since irreparable harm may have been inflicted upon the framework or soft tissues of the larynx.

Treatment:

1. Streptomycin and para-aminosalicylic acid.
2. Treatment of pulmonary lesion.
3. Voice rest.
4. Narcotics for pain.
5. Injection of the superior laryngeal nerve with procaine (Novocaine) or alcohol for relief of pain.
6. Tracheotomy for obstruction.
7. Surgery for secondary stenosis, if indicated.

Syphilitis Laryngitis

Etiology: Treponema pallidum. Extremely rare in the congenital form and now very rare also in the acquired form of the disease.

Pathology: The larynx is never affected in the primary stage of the disease. During the secondary stage, infection and mild edema of the larynx are common and mucous patches may be observed. These lesions are temporary and disappear with the resolution of this phase. The gummata are characteristic of laryngeal involvement in the tertiary stage. Ultimate breakdown of these lesions results in the development of ulcerations, perichondritis, and fibrosis.

Symptoms:

1. A mild hoarseness is often the only symptom. Gummata and ulcerations may lead to varying degrees of hoarseness.
2. There is no pain.
3. As the swelling increases or fibrosis develops, symptoms of respiratory embarrassment may occur.
4. The diagnosis is confirmed by serologic tests and biopsy.

Prognosis: In early lesions, the prognosis is quite favorable, but destruction of cartilages cause permanent changes.

Treatment:

1. Penicillin.
2. Supportive measures.
3. Tracheotomy for respiratory obstruction.
4. Reconstructive operation for severe laryngeal disease.

Sarcoidosis

Sarcoidosis is a systemic granulomatous disease of unknown etiology. It affects primarily the lung and mediastinal nodes but laryngeal involvement may occur in 1-5% of all patients with sarcoidosis.

Pathology: The lesion is characterized by noncaseating granuloma. The lack of caseation distinguishes sarcoid from tuberculosis, pathologically. Since the disease occurs submucosally, ulcerations are rare.

Symptoms:

1. Hoarseness is the prominent feature.
2. Pain usually is not present.
3. If the lesion is large, dyspnea may be present.
4. The epiglottis is most often involved, typically with small nodules at the free margin, which can become a confluent, indurated swelling. Other areas commonly affectged are the aryepiglottic folds, arytenoids, false cords, and subglottic areas. The true vocal cords are rarely involved.
5. Both diffuse edema and exophytic masses may cause airway obstruction.
6. Biopsy is essential for diagnosis.

Prognosis: Permanent clinical remission usually occurs.

Treatment:

1. Systemic and/or local injection of steroids may relieve obstruction by decreasing edema.
2. Radiation is not effective.
3. Tracheotomy, with surgical removal of bulky obstructing lesions, is a safe, conservative approach.

Etiology: Klebsiella rhinoscleromatis (von Frisch bacillus). Rare in the USA.

Symptoms:

1. Hoarseness, cough, and increasing dyspnea.
2. The most common site of scleroma of the larynx is the subglottic region. Pale pinkish swelling may be seen below the vocal cords.

Treatment:

1. Streptomycin.
2. Steroids.
3. Tracheotomy (often needed).

Perichondritis of the Larynx

Etiology:

1. Infection (tubercluosis, syphilis, septic laryngitis, etc).
2. Trauma.
3. High tracheotomy.
4. Radiotherapy.
5. Neoplasm with secondary infection.

Pathology:

1. Perichondritis leads to subperichondrial abscess, necrosis of cartilage, and later, stenosis.

2. Perichondritis of the thyroid cartilage is more common than perichondritis of the epiglottis. This is because the epiglottis is fibroelastic cartilage where the perichondrium is adhered to the cartilage.

Symptoms:

1. May be insidious or of sudden onset.
2. Fever and malaise (acute form).
3. Local pain and tenderness.
4. Enlargement of laryngeal framework, swelling of the neck.
5. Abscess and fistula.
6. Hoarseness, cough, dysphagia, and dyspnea.

Diagnosis:

1. Syphilis and malignant disease should be ruled out.
2. Rule out unsuspected foreign body.

Treatment:

1. Hospitalization.
2. Systemic antibiotics.
3. Tracheotomy.
4. I and D if indicated.
5. Dilatation for stenosis.
6. Laryngofissure.
7. Laryngectomy for extensive necrosis of a cartilage.

Glanders

Glanders is a serious infectious disease marked by the occurrence of multiple granulomatous abscesses throughout the body, caused by Pseudomonas mallei (Actinobacillus mallei). Perichondritis and cartilage destruction may complicate the laryngeal disease.

Leprosy of the Larynx

Leprosy of the larynx is rare. It affects the larynx in 10% of the cases. It is caused by Mycobacterium leprae or Hansen's bacillus.

Treatment:

1. DDS (Diaminophenylsulfone; dapsone) (for 1-4 years).
2. Steroids.
3. Tracheotomy.

Diphtheric Laryngitis

Diphtheric laryngitis is rare.

Etiology: Corynebacterium diphtheriae.

Symptoms:

1. Onset insidious.
2. Hoarse, croupy cough is the first symptom.
3. Grayish white membrane on the larynx. Its removal is followed by bleeding.

Treatment:

1. Antitoxin.
2. Penicillin.
3. Tracheotomy.

Mycotic Infection of the Larynx

Fungal infections of the larynx are rare. Blastomycosis, histoplasmosis, and candidiasis are the most commonly encountered.

Blastomycosis. Blastomycosis is seen in endemic proportions in North America and is mainly a disease of the skin and lungs. However, primary involvement of the larynx does occur. It is caused by Blastomyces dermatitidis and characterized by diffuse nodular infiltration of the larynx, vocal cord fixation, ulcer, and stenosis.

The epithelium undergoes marked hyperplasia of the pseudoepithelial type and may be mistaken for carcinoma. Microabscesses contains the organisms, giant cells, and mononuclear cells occur in the epidermis and dermis and are characteristic of this disease.

Symptoms:

1. Hoarseness and cough occur early.
2. Dyspnea and dysphagia are late symptoms.
3. In the early stage, the laryngeal mucosa is difusely inflamed and granular.
4. Tiny miliary nodules may be seen on the vocal cords.
5. In advanced stage, mucosal ulceration, covered with foul smelling, greenish exudate, under which is a bright red granular bed.
6. Later, fibrosis, fixation of arytenoids, or stenosis develops.

Treatment: Amphotericin B.

Histoplasmosis. Histoplasmosis is caused by Histoplasma capsulatum and usually associated with pulmonary histoplasmosis.

Treatment:

1. Amphotericin B.
2. Sulfonamide.

Candidiasis (moniliasis). This is caused by Candida albicans, and is almost always the result of chemotherapeutic suppression of normal bacterial flora. It is characterized by white patches on a bright red mucosa.

Treatment: Nystatin (Mycostatin).

Actinomycosis. This disease, caused by Actinomyces bovis is characterized by a yellowish granulomatous infiltration which suppurates. It involves the neck and perilaryngeal structures.

Treatment: Penicillin or tetracycline.

Coccidiomycosis. Caused by Coccidioides immutus, this disease is endemic in the San Joaquin Valley area of California. It is more often seen in colored races. The lesion consists of nodular masses of granulomatous tissue.

Benign Tumors and Cysts of the Larynx

Benign Tumors

Benign tumors of the larynx are relatively uncommon. They occur in the following order of frequency: papilloma, chondroma, neurofibroma, leiomyoma, angiofibroma, myoma, hemangioma and chemodectoma.

Papilloma. Papilloma is the most common benign tumor of the larynx, and occurs in patients of all ages.

Etiology:

1. The causative agent is thought to be a virus.
2. Seems to be related to hormonal changes. Papillomas usually regress during puberty.

Pathology:

1. Papillary epithelial tumor usually involving the true cords, but may affect supraglottic and subglottic regions.
2. May also involve the trachea and bronchus.
3. Papilloma in juveniles is more often multiple, and recurs more frequently than that in adults.
4. Papillomas in adults are usually single, but may undergo malignant change.

Symptoms:

1. Aphonia or weak cry is usually the first sign in infants.
2. Dyspnea.
3. Patients with papilloma have low serum magnesium.

Treatment:

1. Repeated laryngoscopic removal to maintain an adequate airway is currently the standard treatment.
2. Tracheotomy is occasionally necessary.
3. Cryosurgery.
4. Autogenous vaccine.
5. Exogenous leukocyte interferon therapy has been proposed by Haglund.
6. Ultrasound therapy.
7. In view of a high incidence of recurrence, thyrotomy and pharyngotomy are not indicated.
8. Irradiation is contraindicated because of its carcinogenic effects.
9. Laser surgery.

Chondroma. Chondroma is a slow growing lesion, composed mainly of hyaline cartilage. It affects males more often than females (10:1).

The most frequent site of origin is the internal aspect of the posterior plate of the cricoid cartilage, followed by the thyroid, arytenoid, and epiglottis.

Symptoms:

1. Hoarseness, dyspnea, and dysphagia (in that order) are the presenting symptoms.
2. A full sensation within the throat may be present.
3. The symptoms are insidious.
4. Dyspnea and hoarseness are prominent with a subglottic mass arising from the internal aspect of the cricoid.
5. The dysphagia is more common in lesions arising from the posterior aspect of the cricoid.
6. Hoarseness is due to restriction of cord mobility by the mass.
7. A mirror examination shows a smooth, firm, round, or nodular, fixed tumor covered by normal mucosa.
8. Chondroma of the thyroid, cricoid, or tracheal cartilages may present as a hard neck mass.
9. A soft tissue film, laminogram, and laryngogram will delineate the extent and site of lesion.
10. Calcification is commonly seen on X-ray.

Treatment:

1. Excision: The site of origin determines the approach.
2. Thyrotomy for tumors of the anterior aspect of the cricoid.
3. A lateral external approach, with or without pharyngotomy, for chondromas of the thyroid, posterior aspect of cricoid, or arytenoid.
4. Recurrence is common if the tumor is not removed completely. Peroral removal is not advised.
5. Total laryngectomy may be necessary for treatment of recurrences.
6. Reconstruction of cricoid cartilage defect by suturing the inferior cornu of the thyroid cartilage to the first tracheal ring may obviate the need for total laryngectomy in selected cases.

Neurofibroma. This is a rare tumor arising from the Schwann cells. The tumor most commonly arises from the aryepiglottic fold. The incidence favors females 2:1.

Granular Cell Myoblastoma. These tumors are thought to be of neurogenic origin. They occur in any age group and preponderantly affect males. The lesion usually occurs at the posterior aspect of the true cords or arytenoids. The lesion is small, sessile, and gray. Hoarseness is often the only symptom. The mucosa may show pseudoepitheliomatous hyperplasia.

Treatment: Excision by direct laryngoscopy.

Adenoma. This tumor is rare. It arises from the mucous glands. The most common site is the false cord or ventricle.

Treatment: Excision perorally or by thyrotomy.

Chemodectoma. Chemodectomas arise from paraganglion tissue. They usually are seen in the false cord and aryepiglottic fold, and are smooth, cystic, and red. Biopsy may be associated with bleeding.

Treatment: Lateral pharyngotomy.

Lipoma. Lipoma is a rare, pedunculated or submucosal tumor which usually arises from the aryepiglottic fold, epiglottis, true cord, and pharyngeal wall.

Treatment:

1. Excision via laryngoscope for pedunculated lesion.
2. Lateral pharyngotomy for submucous tumor.

Hemangioma. Hemangioma in adults are more common than in children. They occur on vocal cords, subglottic regions, and pyriform sinus. Hemangioma in children is discussed under Congenital Anomalies.

Treatment: Excision is best handled by suspension laryngoscopy (for a small angioma) or by lateral pharyngotomy (for a large angioma).

Pseudoepithelial Hyperplasia. This is a benign epithelial change that may resemble carcinoma. It can be caused by:

1. Tuberculosis.
2. Syphilis.
3. Granular cell myoblastoma.
4. Blastomycosis.
5. Pachyderma laryngis.
6. Radiation.
7. Papillary keratosis (premalignant).

When a diagnosis of pseudoepithelial hyperplasia is made, further biopsy or studies may be necessary to rule out blastomycosis, granuloma cell myoblastoma, etc.

Cysts and Tumorlike Lesions of the Larynx

Retention Cyst. Retention cyst occur most often where mucous glands are abundant. The false cord, ventricle, epiglottis, aryepiglottic fold may be the sites.

Treatment:

1. Laryngoscopic removal.
2. Maruspialization.

Prolapse of the Ventricle. Ventricular prolapse is protrusion of ventricular mucosa between the true and false cords. It is frequently associated with chronic bronchitis, and the presenting symptom is hoarseness. A sessile pink mass arising between the false and true cord is seen.

Treatment: Laryngoscopic removal with a forceps.

Laryngocele: This is an air-filled dilation of the appendix of the ventricle. There are two types:

1. External laryngocele, the more common form in which the sac protrudes above the thyroid cartilage and the thyrohyoid membrane and presents as a mass in the neck.
2. Internal laryngocele, less common, in which the sac remains within the thyroid cartilage.

3. The combined type may be present.

Etiology: Unknown.

Symptoms:

1. External laryngocele presents as a swelling in the neck which increases in size with increased intralaryngeal pressure.
2. Internal laryngocele presents with hoarseness and dyspnea.
3. Indirect laryngoscopy may show a smooth dilation at the false cord level involving the false cord and aryepiglottic fold.
4. Diagnosis may be confirmed by a tomogram of the neck showing air within the sac.

Treatment:

1. Laryngoscopic decompression for small lesions.
2. Lateral external approach for larger lesions.

Contact Ulcer of the Larynx

Etiology:

Contact ulcers and granulomas appear to be caused by vocal abuse or nonlinguistic laryngeal trauma, such as repeated harsh coughing or persistent throat clearing. Allergic rhinosinusitis causing postnasal drip also may be a factor. Many patients have a hiatal hernia or peptic esophagitis, predisposing to reflux of gastric contents and acid into the pharynx, with resulting coughing, laryngospasm, and harsh throat clearing. Contact ulcers and

granulomas heal when the hiatal hernia, gastric reflux, and peptic esophagitis are treated adequately.

Pathology: The most common site is vocal process of the arytenoid.

Symptoms:

1. Hoarseness.
2. Pain on exertion.
3. Typical ulceration.

Treatment:

1. Absolute voice rest.
2. Vocal reeducation.
3. Avoidance of irritants.
4. Broad-spectrum antibiotics and steroids may be of help.

Vocal Nodules (Singer's Nodules)

This may be considered a localized traumatic laryngitis.

Etiology:

1. Vocal overuse: Screaming in children, harsh talking in adults, and faulty techniques in singers.
2. Predisposing factors: Ectomorphic and athletic body type, vociferous and aggressive personalities.
3. Precipitating factors: Allergy, thyroid and emotional imbalance, URI, sinusitis.
4. Aggravating factors: Cigarette smoking and alcohol.

Pathology:

There are two types:

1. Acute or fresh type (soft, reddish, vascular, edematous).
2. Chronic or mature type (hard, white, thickened, fibrosed).

Clinical Features:

1. Found more often in women, children (more often in boys), professional singers, lecturers, etc.
2. Causes hoarseness.
3. The most common site is at the junction of the anterior and middle third, usually bilateral (see Table 15-1). The middle part of the membranous vocal cord has the greatest amplitude of vibration, and hence is most likely to develop singer's nodule.

Treatment:

For children:

1. Parent counselling.
2. Psychotherapeutic rehabilitation.
3. Vocal reeducation.
4. Microlaryngoscopic laser excision in rare cases unresponsive to voice therapy.

Table 15-1. The Most Common Site of Benign Laryngeal Lesions

Contact ulcer	**Vocal process of arytenoid**	**Uni- or bilateral**
Laryngeal polyp	**Junction of anterior and middle third**	**Usually unilateral**
Vocal cord nodule	**Junction of anterior and middle third**	**Bilateral**
Postintubation	**Vocal process of the arytenoid**	**About 50% bilater.**

For adults:

1. Voice rest.
2. Voice therapy.
3. Microlaryngoscopic excision or laser vaporization followed by voice therapy.

Intubation Granuloma

Etiology: Endotracheal intubation.

Age and sex: All adults. The incidence is higher in females (4:1) because the tube falls more to the posterior commissure, and because the mucosa is thinner.

Site: Invariably on the vocal process of the arytenoid.

Treatment:

1. Excision when pedunculated.
2. Attempts of removal during the sessile stage should be avoided as recurrence is likely.
3. Microlaryngoscopic laser excision appears to be more effective than surgical excision.

Chronic Nonspecific Disease of the Larynx

Pachyderma Laryngis

Pachyderma laryngis is a specific entity in which the posterior commissure and the posterior thrid of the true cords are the site of a localized hyperplastic and keratinized process. Histologically, acanthosis, parakeratosis, keratosis, and hyperkeratotic papilloma are noted. There is no dyskeratosis. It is not premalignant. Diagnosis is made by biopsy.

Treatment: Nonspecific.

Keratosis of the Larynx

This is a term used to denote a group of premalignant epithelial lesions in which an abnormality of growth and/or maturation has occurred.

Etiology:

1. Smoking, vocal abuse, chronic laryngitis, vitamin deficiencies.
2. Exact cause unknown.

Symptoms:

1. Hoarseness is the only symptom.
2. A raised reddish area of mucosal irregularity overlying a portion of one or both cords with chronic inflammation.

Treatment:

1. Cessation of smoking and other causative agents.
2. Direct laryngoscopy with excision.
3. Periodic examination.

Leukoplakia of the Larynx

This is a pathologic premalignant process characterized by a thick whittish layer of hyperkeratotic epithelial cells.

Etiology: Vocal abuse, excessive smoking and intake of alcohol, iritative environment.

Symptoms:

1. Hoarseness.
2. White patches on vocal cords.

Treatment:

1. Laryngoscopic excision.
2. Removal of causative factors.
3. Periodic examination.

Arthritis of the Cricoarytenoid Joint

Etiology:

1. Rheumatoid arthritis is by far the most common cause (about 25% of cases of rheumatoid arthritis).
2. Gout, collagen diseases (lupus erythematosus).
3. Gonorrhoea, tuberculosis, syphilis, rare.
4. Trauma.

Symptoms:

1. Lump in throat.
2. Throat pain aggravated by swallowing or speaking.
3. Referred ear pain.
4. Hoarseness, stridor, and dyspnea.
5. Striking, bright red swelling over the arytenoid.
6. Palpation of the arytenoid produces severe pain.
7. Vocal cord may be fixed in the paramedian or intermediate position. Direct laryngoscopy and palpation of the arytenoids is necessary to differentiate fixation from paralysis.
8. Other signs of rheumatoid arthritis (sedimentation rate, C-reactive protein, and gamma globulin, abnormal; positive test for rheumatoid factor).

Treatment:

1. Control systemic rheumatoid arthritis.
2. Salicylates.
3. Steroids.
4. Tracheotomy.
5. Arytenoidectomy or arytenoidopexy for midline fixation of both vocal cords. Unilateral fixation rarely requires therapy.
6. Teflon injection.

Acquired Stenosis of the Larynx

Injury to the larynx leading to acquired stenosis can involve the supraglottis, glottis, or subglottis, or any combination of these.

Clinical features and evaluation:

1. Careful history taking.
2. Thorough physical examination of ENT areas with special attention to the neck, larynx, and pharynx.
3. Radiologic examination should include x-rays of the chest and lateral neck, and laminogram of the larynx. Xeroradiography and/or contrast laryngography may be helpful in delineating abnormalities. A CT scan has been found to be of great value in evaluation of laryngeal trauma.

4. Endoscopy.

a. Fiberoptic flexible nasopharyngolaryngoscope (Machida or Olympus). This is a very useful atraumatic diagnostic tool for evaluation of laryngeal trauma. This also can be used to evaluate the subglottic region via the tracheotomy stoma and may eliminate the need for direct laryngoscopy in some cases.

b. Rigid telescope with right-angle lens (excellent tool for photographic documentation).

c. Direct laryngoscopy.

d. Esophagoscopy.

e. Tracheoscopy.

f. Bronchoscopy.

Supraglottic Stenosis

Etiology:

1. External crushing trauma in the region of the hyoid bone inflicted during an automobile accident.

2. Penetrating wound, caustic ingestion, severe infection.

Clinical features:

1. In supraglottic injuries, the blow often leads to fractures of the thyroid ala transversely with detachment of the epiglottis and false cords from the anterior commissure.

2. The most common injury seen is rupture of the thyroepiglottic ligament with superior retraction of the epiglottis and herniation of the soft tissues of the preepiglottic space into the laryngeal lumen.

3. There often is an associated tear in the posterior pharyngeal wall. The arytenoid cartilages may be dislocated. The epiglottis is easily seen on tongue depression in these cases. This is a useful clinical sign.

4. Direct laryngoscopy reveals that the false cords are splayed apart and necrotic granulation tissue is present between the true and false cords.

Treatment:

1. Tracheotomy may be a lifesaving measure. Intubation prior to tracheotomy should be avoided.

2. The transhyoid or thyrotomy approach should be used for repair of lacerations and for replacing various structures to their normal position. Epiglottidectomy and arytenoidectomy may be necessary.

Glottic Stenosis

Classification: Glottic stenosis may be classified into three varieties:

1. Anterior stenosis (web):

a. With laryngeal function.
b. With bilateral paralysis.

2. Posterior stenosis.

3. Complete stenosis.

a. With laryngeal function.
b. With bilateral laryngeal paralysis.

Anterior Glottic Stenosis

Two types:

1. Anterior web results from traumatic endoscopy, lye burns, infections, or foreign body.
2. More extensive (thick) stenosis usually results from external trauma.

Clinical features:

1. Symptoms from anterior webs may be minimal.
2. Hoarseness.
3. Varying degrees of respiratory distress.

Treatment:

For thin webs:

1. Endoscopic section followed by dilatations.

For thick stenosis:

a. When vocal cords are not paralyzed:

1) Endoscopic section with insertion of a keel without thyrotomy as described by Dedo.

2) External thyrotomy approach with insertion of a keel.

b. When bilateral paralysis complicates the glottic stenosis: External thyrotomy approach with arytenoidectomy with lateralization of the vocal cord and insertion of a conforming silicone stent (instead of a keel).

Posterior Glottic Stenosis

Etiology:

1. External trauma.
2. Internal trauma.
3. Infection (tuberculosis, diphtheria).

Clinical features:

1. Diagnosis is made by indirect, fiberoptic, or direct laryngoscopy.
2. Dyspnea on exertion and hoarseness.

Treatment:

1. Resection of posterior web via thyrotomy approach.
2. Montgomery recommends use of a superiorly based mucous membrane flap from the interarytenoid space.

Complete Glottic Stenosis

Etiology: External laryngeal trauma.

Treatment:

1. Anterior midline vertical thyrotomy approach with incision of stenotic area in the midline.
2. Use of local mucosal flap, buccal or nasal septal mucosa grafts, or split-thickness skin grafts.
3. Insertion of a conforming intralaryngeal stent.
4. The stent is left in position for 4-8 weeks.
5. When bilateral vocal cord paralysis accompanies complete glottic paralysis, arytenoidectomy is necessary.

Subglottic Stenosis

Etiology:

1. Long-term endotracheal intubation (most frequent). The incidence reported by different authors varies between 0.9-3.0%. This problem occurs most frequently in children, since in them the subglottis is the narrowest part of the upper airway. Consequently, pressure from an endotracheal tube inflicts the most damage to this area.
2. External trauma (penetrating wound).
3. Internal trauma (high tracheotomy, traumatic endoscopy, foreign body).
4. Neoplasm (chondroma, fibroma, and carcinoma are most common in this area).
5. Radiation.
6. Severe infection.
7. Congenital stenosis.

Clinical features:

1. Dyspnea on exertion.
2. Wheezing is common and is often misinterpreted as asthma or a chronic tracheobronchial infection.
3. Nonproductive cough and voice change.
4. The diagnosis can be made by indirect laryngoscopy. However, it is very difficult to determine the exact level of the stenosis with the laryngeal mirror examination. A lateral neck x-ray resolves this problem by indicating more exactly the size of the lesion. The lesion is even more accurately outlined in a laminagram, laryngogram, or CT scan. The inferior extent may be evaluated by a flexible fiberoptic endoscope passed retrogradely via a tracheotomy stoma.

Treatment:

1. Dilatation may be helpful, but also may be harmful since it may denude the mucosa thereby worsening stenosis.
2. Local steroid injection may be of benefit in some cases.
3. A low tracheotomy may be used.
4. Rethi's procedure: Rethi described a procedure in which the anterior and posterior rings of the cricoid are splin in a sagittal plane. This enlarges the diameter of the subglottic lumen without disturbing the esophageal mucosa. A Montgomery's stent is inserted with or without a mucosal graft to ensure maintenance of anterior and posterior diastasis of the cartilage fragments. Although Rethi did not originally describe keel insertion to prevent anterior commissure webbing, this approach is currently considered preferable. The keel is inserted at the time the internal stent is removed through a laryngofissure. The keel is then removed 2 weeks later.
5. Hyoid arch transposition: In 1975, Druck et al described a method of treatment for subglottic stenosis in which the anterior arch of the cricoid is removed, and an autogenous bone graft from the midportion of the hyoid is interposed between the cut edges of the cricoid, thereby externally widening the subglottic diameter.
6. Cricoid excision with thyrotracheal anastomosis: Conley introduced the concept of segmental resection of the stenotic subglottis with end-to-end anastomosis, and it has subsequently been reported on by several authors (Gerwat and Bryce; Carcassone et al; Pearson et al).
7. Laryngotracheoplasty: In 1974, Evans and Todd reported use of a surgical approach for relief of subglottic stenosis in children. In this procedure, the laryngofissure is modified in a "stepped-off" fashion, producing cartilaginous interdigitations of the cricoid and upper tracheal rings on each side. These interdigitations are distracted, and the open position is maintained by suturing the cartilage externally and stenting internally with rolled Silastic sheeting. The internal stent is then removed after 6 weeks by the endoscopic approach. Good results have been reported with use of this method.
8. Anterior cartilage splitting with autogenous costal cartilage graft: Doig et al first described the use of anterior cricoid cartilage splitting with an autogenous costal cartilage graft inserted to widen the cricoid ring. A similar procedure has been advocated by Fearon and Cotton for treatment of subglottic stenosis in pediatric patients. Excellent results have been reported.

Congenital Anomalies of the Larynx

Congenital Web

A congenital web develops as a band which extends over part (web) or all (atresia) of the glottis. However, the anterior two-thirds of the glottis is the most susceptible site.

Symptoms:

1. Depend on degree of glottic closure.
2. Atresia present as severe dyspnea at birth. Death may follow if not promptly recognized and treated.
3. Small web may be asymptomatic.
4. Mainly weak or hoarse cry and cough.

Treatment:

1. Immediate insertion of a bronchoscope or a tracheostomy tube for atresia.
2. Thyrotomy and insertion of a McKnaught's tantalum plate between the vocal folds.

Congenital Laryngeal Cyst

This cyst occurs most commonly in the supraglottic area (lateral wall of supraglottis or on the epiglottis) or associated with a laryngocele, producing inspiratory stridor and weak cry. Diagnosis is made by direct laryngoscopy.

Treatment:

1. Emergency treatment by aspiration.
2. Endoscopic excision later.

Congenital Subglottic Stenosis

The subglottic region 2-3 mm below the true cord is the site of predilection.

Symptoms:

1. Severe barking stridor.
2. Expiratory stridor if subglottic.

Treatment:

1. 40-50% need tracheotomy.
2. Dilatation.

Vocal Cord Paralysis

Etiology:

1. Trauma at birth (unilateral).
2. Platybasia (bilateral).
3. Arnold-Chiari syndrome (bilateral).
4. Left vocal cord paralysis may result from stretching of the left recurrent nerve due to a congenital cardiovascular lesion.

Symptoms:

Unilateral: Weak cry.
Bilateral: Crowing inspiration, severe stridor.

Treatment:

Unilateral: No treatment.
Bilateral: (1) Tracheotomy; (2) Arytenoidectomy or arytenoidopexy best delayed until age 5 or 6.

Subglottic Hemangioma

Subglottic hemangioma is a rare anomaly of early infancy which may be associated with a skin hemangioma (50% of cases). The anterior subglottic area is the most susceptible site.

Symptoms:

1. Inspiratory stridor is noted at birth or soon thereafter.
2. There may be a history of repeated episodes of croup.
3. Hoarseness is not a common symptom.
4. Direct laryngoscopy reveals a pink to blue, easily compressible, subglottic tumor.
5. A biopsy is never done, since the ensuing hemorrhage may be fatal.
6. The tumor can be seen on a lateral neck x-ray.

Treatment:

1. Tracheotomy for respiratory obstruction.
2. Systemic administration of steroids (Cohen): A course of prednisone (20 mg) daily for 1 month and repeated 1-2 times as indicated. This may be the least hazardous therapy.
3. Intralesional injection of steroid.
4. Use of the carbon dioxide laser excision appears to be a safe, effective treatment for subglottic capillary hemangiomas and recommended by Healy et al. It is not used for cavernous-type lesions. Intense humidification is necessary in the immediate postoperative period to prevent crust formation.
5. Radiation therapy: Because of the possibility of injury to developing laryngeal cartilage and the risk of thyroid carcinoma exists, radiation therapy is not recommended.

6. Surgical excision via a midline tracheal incision and an intraluminal stent.

Laryngomalacia

This is the most common laryngeal abnormality of the newborn and is due to unusual flaccidity of the laryngeal tissues, especially the epiglottis.

Symptoms:

1. Inspiratory stridor and noisy respiration noted soon after birth, usually worse with the infants on their backs compared with the infants on their stomachs.
2. Diagnosis requires direct laryngoscopy which reveals a flaccid, curled epiglottis which is drawn over the glottis on inspiration.
3. The vocalcords are normal in appearance and motility.
4. Rule out lower respiratory tract anomalies by bronchoscopy.

Treatment:

1. Observation (stridor usually disappears by 12-16 months of age).
2. Tracheotomy in rare cases.

Cri-du-Chat Syndrome

Recently a new nosologic entity called the cri-du-chat syndrome was described (Ward et al) in which the larynx has the same appearance as that seen in laryngeal chondromalacia. The cri-du-chat syndrome is caused by partial deletion of a number 5, group B chromosome and is characterized by a weak, wailing cry, like that of a kitten. Other accompanying features of the syndrome include severe mental retardation, a rounded facies, a beaklike profile, microcephaly, hypotonia, hypertelorism, antimongoloid palpebral fissures, epicanthal folds, strabismus, and a variety of visceral abnormalities.

Laryngeal Clefts

Laryngeal clefts are very rare. Irregular and incomplete fusion of the laryngotracheal septum results in a tracheoesophageal fistula or cleft of the larynx. These anomalies are manifest very soon after birth.

Symptoms:

1. Cyanosis with feeding.
2. Stridor.
3. Abnormal cry.
4. Pneumonia.
5. Usually fatal unless diagnosed early and corrected early.
6. Diagnosis made by direct laryngoscopy.

Laryngeal Atresia

Laryngeal atresia results from a failure of the developing larynx to recanalize after the normal epithelial fusion that takes place toward the end of the third month of gestation. Atresia may be at the supraglottic, glottic, or subglottic levels either separately or combined.

Clinical features:

1. Aphonia if atresia is complete.
2. Hoarse stridorous if atresia is incomplete.

Treatment:

1. Immediate tracheotomy.
2. When the laryngeal skeleton is preserved: Partial resection of the atretic portion in the transverse plane with reanastomosis of the distal portion of the laryngotracheal tube to the proximal part.
3. When the entire laryngeal lumen is filled with scar: A hollowed groove for the future respiratory tube is created, lined with a free skin graft, and later implanted with cartilage.

Congenital Supraglottic Webs

If they are relatively thin and membranous, supraglottic webs may be treated by direct incision of the web through the laryngoscope. Thicker webs may require subsequent dilatation to maintain an adequate lumen.

Congenital Glottic Stenosis (Webs)

Glottic stenosis occurs in three different forms (Holinger): (1) A thin, transparent, membranous sheet covering the superior aspect of the true vocal cords and the anterior part of the intervening glottis; (2) a web of variable thickness lying between portions of the membranous cords; and (3) fusion of the anterior half of the true vocal cords.

Treatment:

For thin web:

1. Endoscopic section followed by dilatations.

For thick web:

1. Endoscopic section with insertion of a tantalum or silicone keel. Tracheotomy required.
2. External thyrotomy approach with insertion of silicone keel. The keel left for 2 weeks.

Acute Laryngeal Trauma

Blunt trauma to the neck is being seen with increasing frequency. Severe laryngeal injury may occur without open neck injuries. The undiagnosed laryngeal trauma case may succumb early from laryngeal obstruction or develop late laryngeal stenosis that requires the permanent wearing of a tracheotomy tube.

Three poor prognostic features in acute blunt laryngeal injuries include: early airway obstruction requiring tracheotomy, the presence of bare cartilage in the laryngeal lumen, and fracture and collapse of the cricoid.

Clinical manifestations:

In any patient who has sustained a possible laryngeal injury, the following symptoms are indicative of some derangement of laryngeal structure:

1. Increasing airway obstruction with dyspnea and stridor.
2. Dysphonia or aphonia.
3. Cough.
4. Hemoptysis.
5. Neck pain.
6. Dysphagia and odynophagia.

Distinctive clinical signs indicative of laryngeal injuries are:

1. Deformities of the neck including alteration in contour and swelling.
2. Subcutaneous emphysema.
3. Laryngeal tenderness.
4. Crepitus over the laryngeal framework.

Diagnosis:

1. Indirect and direct laryngoscopy: Direct laryngoscopy may precipitate airway obstruction in a patient with an acute laryngeal injury. In cases not requiring tracheotomy, indirect mirror laryngoscopy or flexible fiberoptic laryngoscopy gives a comprehensive and undisturbed view of the larynx.
2. Roentgenograms of the neck and chest must be taken to detect laryngeal fractures, tracheal injuries, and pneumothorax.
3. The CT scan is an excellent method of diagnosing hyoid fractures, fracture dislocation of thyroid and cricoid cartilages, and distortion of laryngeal structures.

Treatment:

1. Conservative management is recommended when blunt trauma has resulted in soft tissue injuries, such as small lacerations, ecchymoses, or submucosal hematomas without evidence of laryngeal fracture or a compromised airway. Conservative management includes voice rest, humidification, bed rest, and systemic steroids.

2. If immediate repair of the larynx cannot be done for any reason, it may be helpful to administer steroids, so that granulation tissue and collagen deposition would be reduced, thereby facilitating an anatomic repair.

3. For establishment of an airway tracheotomy is the preferred means of management in these patients (avoid high tracheotomy). Premature intubation can obscure important diagnostic considerations as well as precipitate life-threatening airway obstruction.

4. Cricothyrotomy may be indicated in a dire emergency. In such cases, remove the tracheotomy as soon as possible and convert it to a standard tracheotomy.

5. Many of these patients have multiple injuries; yet suspicion and recognition of the acute laryngeal injury is imperative.

6. Surgical exploration is indicated in any neck injury with symptoms of stridor, voice change, cartilage disruption, and cervical emphysema.

7. Exploration of laryngeal structure is best performed through a horizontal incision to minimize scarring of the anterior neck.

8. Tears of the pyriform sinuses, hypopharynx, posterior larynx, thyroarytenoid ligament and muscle are repaired.

9. When repairing laryngeal injuries all mucosa must be sutured carefully. Local flaps or free mucosal grafts taken from the epiglottis or buccal mucosa should be used to close defects. All cartilaginous and submucosal tissues must be covered with epithelium.

10. If an arytenoid cartilage is completely avulsed and displaced, it is better to remove it than attempt to reposition it. Partial arytenoid disruption can be treated with repositioning and mucosal repair.

11. Laryngeal cartilage fractures, like any other fractures, must be reduced and immobilized. Repair should be done within 7-10 days of the time of injury.

12. Splint a laryngeal fracture by means of a mold or stent in the laryngeal lumen. A rubber finger-cot, filled with Ivalon sponge may be used.

13. The stent is usually inserted through a thyrotomy or infrahyoid laryngotomy and is fixed above and below by stainless steel sutures passes through the skin.

14. The stent is fixed in a position so that the upper end is at the level of the aryepiglottic folds and the lower end is just above the tracheotomy site. It should be left for 4-8 weeks.

Foreign Bodies in the Larynx and Tracheobronchial Tree

1. Choking on foods causes 2500-3000 deaths per year in the USA. It is the sixth most common cause of accidental death. Fifty-five percent of aspirated foreign bodies involve the respiratory tract in children 6 months to 4 years of age. The accident is neither observed nor suspected in over one-third of these cases.

2. All techniques which are used for aiding the obstructed patient in an emergency such as pounding on the back, Heimlich maneuvers, and finger probing of the throat are dangerous and should be discouraged unless the airway obstruction is unrelieved by the patient's own reflexes. These techniques may result in further impaction and the possibility of a total obstruction not present before these attempts.

3. In the series of Cohen et al, nuts were the most common type of foreign body aspirated (55), followed by food particles (20%), and metallic objects (16.7%). Forty-three percent were found in the left bronchial tree (contrary to the common belief based on anatomic details), 38% in the right bronchial tree, and 4% in the larynx.

4. General anesthesia is recommended. Oxygen standby without any anesthesia may have to be used in cases where the airway obstruction is severe and the airway cannot be adequately controlled.

5. The most common postoperative problem is subglottic laryngeal edema, treated with humidification and systemic steroids. Bronchitis, pneumonia (impaction with long-standing foreign bodies), and laryngotracheobronchitis may be seen. Complications of foreign bodies include bronchial suppuration, bronchial ulceration, granulation tissue, bronchial stenosis, peribronchial and peritracheal lymphadenopathy with compression of bronchi, pneumonitis, atelectasis, obstructive emphysema, pneumomediastinum, and pneumothorax.

6. Encapsulated dry vegetable substances can swell in the presence of moisture and may have to be broken into smaller pieces to avoid total obstruction of the trachea during removal.

7. A serious intraoperative complication can result from the removal of a large obstructive foreign body from the bronchus and having it get stuck at the level of the larynx. Attempts should be made to remove it from the glottis but if this is not possible, the foreign object should be pushed back down into one of the bronchi so that ventilation can proceed with the unobstructed lung.

8. Anesthesia methods: Preanesthesia medications - narcotics and sedatives - are contraindicated because they may depress respirations from anesthesia. The apneic relaxant technique allows sufficient time for the atraumatic manipulations of the bronchoscope. This consists of a light plane of anesthesia accompanied by a muscle relaxant.

9. Intravenous dexamethasone phosphate (4-8 mg) is given before endoscopy to minimize subglottic edema.

Paralysis of the Larynx

1. The larynx is supplied by two branches of vagus nerve: superior laryngeal and inferior (recurrent) laryngeal nerves.

The superior laryngeal nerve divides extralaryngeally into: (a) the internal branch which supplies sensory innervation to the laryngeal cavity above the glottis, and (b) the external branch (motor) which supplies the cricothyroid muscle.

The recurrent or inferior laryngeal nerve supplies motor innervation to all the intrinsic laryngeal muscles of the same side except for the cricothyroid and to the interarytenoid muscle of both bodies. It also supplies sensory innervation to those portions of the larynx below the glottis.

2. Paralysis of the laryngeal muscles originates in one of two areas: the central nervous system or the peripheral motor nerves. In most cases (90%) laryngeal paralysis is the result of peripheral nerve involvement. Arising from the ambiguus nucleus in the midbrain, motor impulses to the intrinsic laryngeal muscles travel via the vagus nerve into the chest, where they enter the recurrent laryngeal nerve. On its passage back to the larynx the right recurrent laryngeal nerve crosses the right subclavian artery, while the left recurrent laryngeal nerve winds awounds the arch of the aorta in close relation to the heart. The ascent of the recurrent laryngeal nerve occurs in a groove between the trachea and the esophagus in close relation to the mediastinal lymph nodes, thyroid gland, and esophagus. Thus a swelling of any of these structures may cause pressure on one of the recurrent laryngeal nerves and obstruct muscles on the involved side. Depending upon the nerve fibers involved and the muscles they supply, many different types of paralysis may occur.

3. Paralyzed vocal cords are best described by their position - median, paramedian, intermediate, extreme abduction (lateral). In the median position, the paralyzed cord remains in the midline. This is a frequent position of a paralyzed vocal cord since the abductor muscles are weaker and more vulnerable than the adductor fibers. The intermediate position, often called cadaveric, is midway between the midline and position of complete abduction. The paramedian is between the median and intermediate.

4. Regardless of the type of paralysis it is hard to predict the permanent position of the vocal cord because of:

a. Continued function of remaining musclers.
b. Muscle fibrosis.
c. Tone of the autonomic system.
d. Tension of the conus elasticus.

5. Unilateral midline paralysis is the most frequent; the left more than the right. The paralyzed vocal cord usually lies lower than the normal cord.

6. Diagnostic evaluation:

a. Careful history and physical examination.

b. Inspection. Indirect mirror examination of the larynx is the time-honored standard for evaluating the larynx. Flexible fiberoptic scopes passed through the nose and nasopharynx, or a rigid lens system introduced through the oral cavity, provide a better view of laryngeal movement and allow prolonged study of the larynx during phonation. These laryngoscopes are adaptable to high-speed cinematography or the TV camera, giving permanent records of the patient's laryngeal function.

Laryngostroboscopy augments the routine indirect mirror examination in studying vocal cord mobility. In this technique a contact microphone is placed on the neck of the patient to pick up his voice and transmit it to an analyzer that determines the fundamental vocal frequency. The fundamental frequency is transmitted electronically to a xenon lamp which flashes an intermittent beam of light at the same frequency. The light is then reflected - using a standard head mirror - to the laryngeal mirror and then to the larynx. The strobe light gives the laryngologist the optical illusion that the vocal cords are moving slowly or not at all. This allows the vocal cords to be studied throughout their vibratory cycle. It is an expensive device of interest primarily to the researcher of vocal function. It does not yet replace the standard indirect laryngoscopy for the practicing otolaryngologist.

c. Electromyography: With the patient awake under topical anesthesia, EMG can be done with hook needle electrodes through an external approach. This technique gives a precise indication of the function of the intrinsic muscles of the larynx, but requires experience and a special skill in the placement of these electrodes. Hirano et al claim that it can be done without affecting normal speech and articulation and that muscle activity can be recorded during normal speech. Although this has a great deal to offer in exactly delineating the extent of laryngeal nerve paralysis, it is still a research tool in most institutions at the present time.

d. Laboratory studies:

1) CBC, ESR, urinalysis.
2) Chest x-ray, PA and lateral.
3) Skull series, particularly base view to rule out erosion of jugular foramen.
4) Cervical spine films.
5) Barium swallow.
6) Thyroid scan.
7) VDRL, FTA.
8) Glucose tolerance test.
9) Lumbar puncture.

e. Endoscopy: Endoscopy should be done as the last step in evaluating vocal cord paralysis. This should include nasopharyngeal examination to rule out neoplasm, direct laryngoscopy to palpate the arytenoid to differentiate vocal cord paralysis from fixation of the cricoarytenoid joint, and bronchoscopy and esophagoscopy to rule out a mass lesion or occult neoplasm even if routine x-ray is normal.

f. Radiological evaluation: Laryngeal tomograms and contrast laryngograms can be of value in the detection and evaluation of vocal cord paralysis. In recurrent laryngeal nerve paralysis, four distinctive x-ray findings have been reported: (1) The vocal cord takes a triangular shape. (2) The laryngeal ventricle is dilated. (3) The cross-sectional area of the vocal cord is diminished. (4) The normal subglottic shoulder is replaced by a straight line. In addition, a loss of abduction can be seen on inspiration. A CT scan of the larynx also will be of help in evaluation of laryngeal paralysis.

Unilateral Paralysis

Etiology: Unilateral paralysis of the vagus or recurrent laryngeal nerve can be the result of one of the following:

1. Tumor in the thyroid gland, mediastinum, or esophagus.
2. Surgical trauma. The most commoncause is thyroidectomy.
3. Pressure on the left recurrent laryngeal nerve by a hypertrophied heart or an aortic aneurysm.
4. Toxic neuritis following influenza, or alcohol, lead, or arsenic poisoning.
5. Rarely by a central lesion.
6. Unknown cause (about 20%).

Finding:

1. Involvement of the nerve may be partial, with weakness and decreased motility of the ipsilateral cord, or may be total with a resulting unilateral paralysis.

2. The paralyzed vocal cord usually is fixed in the paramedian position (incomplete paralysis).

3. Involvement of the vagus nerve may lead to fixation in an intermediate or lateral position (complete paralysis). Depending on the cause, the paralysis may be either temporary or permanent.

Symptoms: Hoarseness is usually the only symptom of unilateral laryngeal paralysis. Even this symptom often gradually disappears as the healthy cord increases its excursion beyond the median line. Functional apposition of the voice may persist for a somewhat longer period. The feebleness of the cough mechanism parallels the degree of hoarseness.

Treatment:

Nonsurgical:

1. Voice therapy.

Surgical:

1. Medialization technique: One of the early attempts to correct the breathiness of unilateral vocal cord paralysis by the technique of medialization of the paralyzed vocal cord

was described by Meurman who placed a piece of autogenous costal cartilage between the thyroid cartilage and the inner perichondrium just lateral to the paralyzed cord through the midline thyrotomy approach. A wedge of thyroid cartilage has been used Copheim and others.

2. Arytenoidopexy: The abducted paralyzed cord can be brought to the midline and held there by direct suture (reverse King's procedure) or by pin fixation (Montgomery). The reverse King's procedure is routinely done during supraglottic laryngectomy when the cord is paretic or the arytenoid is sacrificed. Montgomery's arytenoidopexy is used in laryngeal reconstruction following trauma to restore glottic competence.

3. Intracordal injection of the vocal cord was first performed by Brunings - in 1911. He used paraffin for injection but because it resulted in paraffin granulomas the technique has been discontinued.

Arnold using Teflon mixed with glycerin, revived injection of the vocal cord for treatment of paralysis. Largely through his work and that of Lewy, Teflon injection has become the mainstay of treatment for hoarseness, aspiration, and loss of cough seen in vocal cord paralysis.

Gelfoam paste may be injected initially to judge whether or not vocal augmentation will be effective.

4. Nerve-muscle transposition: The most recent procedure suggested for treatment of the intermediate vocal cord paralysis was introduced in 1977 by Tucker. He reported his preliminary experience with the nerve-muscle technique to restore adduction of the unilaterally paralyzed vocal cord. He states that there are two major advantages to his technique: (a) avoidance of the need to section and/or anastomose nerves, and (b) selective reinnervation of only those necessary muscle groups. To this date, the nerve-muscle pedicle technique has been applied in nine patients with unilateral vocal cord paralysis. This procedure is reserved for patients with unilateral vocal cord paralysis showing evidence of involvement of the superior and recurrent laryngeal nerves, a posterior glottic defect greater than 3-4 mm, and a bowed vocal cord. In addition, it is recommended that 6 months be allowed to lapse to permit possible spontaneous reinnervation of compensation to occur before performing this surgical procedure. Through a horizontal incision, the main loop of the ansa hypoglossi over the jugular vein is identified and traced distally until the branch to the anterior belly of the omohyoid muscle is seen. The nerve-muscle pedicle is designed by mobilizing the nerve branch with its blood supply. The nerve will travel a few millimeters between the muscle bundles before branching. The portion of muscle that should be excised lies just past the actual point of nerve entry to preserve the motor end plates. Sutures are then placed around a 3 mm^2 block of muscle adjacent to the nerve. The block is then cut from the omohyoid muscle. The technique for unilateral intermediate vocal cord paralysis differs at this point in that a window of thyroid cartilage is carefully removed, maintaining the inner perichondrial layer. This is incised and reflected, exposing the outermost fibers of the lateral thyroarytenoid muscle. The previously prepared nerve-muscle pedicle is sutured into place using the stay sutures. The outer perichondrium is then sutured back into place and the wound is closed.

Bilateral Abductor Paralysis

Bilateral abductor paralysis is the most common form of bilateral motor paralysis, and is of great clinical importance.

Etiology: In almost all instances it is by extensive thyroid surgery, with injury of both recurrent laryngeal nerves.

Findings: Bilateral abductor paralysis of the vocal cords is manifested by a paralysis of both vocal cords near the median line.

Symptoms:

1. Destruction of both recurrent laryngeal nerves or injury of these nerves is usually followed by a history of transient hoarseness.
2. Weakness of voice is usually prolonged.
3. Cough mechanism is less forceful.
4. As the vocal cords approach the median line, respiratory embarrassment may become increasingly severe requiring an immediate establishment of adequate airway.

Treatment:

1. A tracheotomy should be performed for respiratory difficulty.
2. The long-term management of this condition requires lateralization of one of the paralyzed vocal cords to an appropriate position so that the airway will be adequate for removal of the tracheotomy and the voice will not be weakened excessively. A 5 mm glottic chink provides an adequate airway, but may result in a weak voice. Four millimeters seems to be ideal if the treated paralyzed cord is at a lower level than the nonlateralized cord. There are three major techniques:

Endolaryngeal arytenoidectomy: In 1948 Thornell first described the oral approach to arytenoidectomy for lateralization of one or both vocal cords. A contraindication to this procedure is severe cardiopulmonary disease where adequate respiratory function requires a normal glottic airway of a tracheotomy tube. This procedure has been regarded as being technically difficult. However, use of the surgical microscope and CO_2 laser has made this procedure easier today.

Extralaryngeal arytenoidectomy:

a. Posterior approach: King first described an extralaryngeal approach to improve respiratory obstruction and to maintain the voice. Today, the Woodman's procedure, or a modification thereof, is the most frequently employed surgical approach for the treatment of respiratory obstruction due to bilateral vocal cord paralysis.

Through the external approach, the arytenoid is exposed. The body of the arytenoid is cut from the vocal process and removed. The suture is then tied around the inferior cornu of the thyroid cartilage while the surgeon, using a laryngoscope, directs the securing of the suture to allow lateralization of the vocal cord 4-5 mm and a downward positioning of 1-2

mm below the opposite cord level. A 5 mm posterior glottic chink is needed for an adequate airway in an adult, but with a 5 mm chink the voice is poor. Therefore it is suggested that the glottic chink be made 4 mm, but to place one vocal cord lower than the other. The success rate of decannulation is 75-95%.

b. Thyrotomy approach: This is the procedure of choice of many laryngologists for bilateral abductor vocal cord paralysis. It offers direct microsurgical visualization and exact placement of the vocal cord.

3. Nerve-muscle transposition: The technique of nerve-muscle pedicle transposition was described by Tucker. The advantages stated by Tucker are as follows:

a. It restores an adequate airway without further loss of voice.
b. There is a return of function within 6-12 weeks.
c. There is selective reinnervation of abductor muscles only that avoids crossed or inappropriate return of function.
d. This procedure does not interfere with potential spontaneous reinnervation.
e. This is technically less difficult than nerve anastomosis. The procedure is based on the assumption that the strap muscles are accessory muscles of respiration and contract during deep inspiration.

By transporting a nerve-muscle pedicle into the posterior cricoarytenoid muscle, one would expect contraction of this muscle and resultant abduction of the vocal cords during deep inspiration.

The indication for this operation is bilateral vocal cord paralysis with airway obstruction. Contraindications to this procedure are preexisting traumatic or neurologic loss of the branch of the ansa hypoglossi to the anterior belly of the omohyoid muscles, and fixation of the cricoarytenoid joints.

It is preferable to employ the reinnervation technique first for voice rehabilitation and if this fails, to use Teflon injection to supplement the results, especially in patients who rely on their voice for their livelihood.

Superior Laryngeal Nerve Paralysis

Etiology: Usually secondary to thyroidectomy or supraglottic laryngectomy.

Symptoms:

1. Lowered voice.
2. Posterior commissure deviates to the paralyzed side.
3. Paralyzed side has a vocal cord that is bowed, flabby, and lower. Guttman's test: Frontal pressure on the thyroid cartilage in the normal subject lowers the voice while lateral pressure raises his voice. In paralysis of the cricothyroid muscle, the opposite is true.

Treatment:

1. As a rule no therapy is necessary.
2. Vocal therapy.
3. A surgical procedure to narrow the cricothyroid space may be of benefit if symptoms are severe. Arnold described suturing the thyroid to the cricoid cartilage to elevate the cartilage during phonation.

Dysphonia

Dysplastic Dysphonia

Dysplastic dysphonia refers to chronic hoarseness due to structural malformation of the larynx (i.e. asymmetry of larynx, congenital webbing, vocal cord sulcus).

Habitual Dysphonia

1. Vocal nodules and polyps: Unilateral polyps cause different vibratory patterns of the two vocal cords leading to diplophonia. Polypoid degeneration of the entire cord is known as Reinke's edema. Speech therapy and surgical ablation (CO_2 laser is especially useful here) are the modalities of treatment.

2. Chronic hypertrophic laryngitis is the end result of chronic laryngeal irritation secondary to vocal abuse, smoking, excessive alcohol, allergies, or nasal obstruction.

3. Dysphonia plicae ventricularis is faulty participation of the false cord in phonation. This disorder can be the end stage of chronic hyperkinetic dysphonia from continuous vocal abuse and also can be seen in some cases of laryngeal paralysis and CNS disorders.

Psychogenic Dysphonia of Emotional Origin

1. Hypokinetic and hyperkinetic psychogenic dysphonia.
2. Spastic dysphonia.

Endocrine Dysphonia

1. Gonadic disorders: Eunuchoid voice, laryngopathia gravidarum.
2. Thyroid and parathyroid disorders: Cretinism, myxedema, hyperthyroidism, calcium imbalances affecting speech musculature.
3. Adrenal disorders: Addison's disease leads to progressive aphonia secondary to muscular weakness. Adrenocortical hyperfunction leads to vocal virilization, particularly in women.
4. Pituitary disorders: Acromegaly leads to vocal virilization. Pituitary hypogenitalism results in the eunuchoid voice of dwarfism.

Paralytic Dysphonia: See Laryngeal Paralysis

Dysarthric Dysphonia of Central Origin

This may result from cerebral palsy, parkinsonism, chorea, multiple sclerosis, cerebellar disease, or bulbar paralysis.

Myopathic Dysphonia from Muscular Disease

Sternothyroid muscle paralysis secondary to injury of the ansa hypoglossi, and myasthenia gravis pseudoparalytica can result in myopathic dysphonia.

Neurovegetative Dysphonia from Autonomic Imbalance

1. Vasomotor monocorditis: Reddening, and edema of one vocal cord. Believed to be of vasomotor origin. It must be distinguished from laryngeal tuberculosis, syphilis, and early vocal cord cancer.

2. Contact ulcers: Associated with granuloma on the other vocal cord. Seen in singers and in "type A" personalities.

Traumatic Dysphonia After Laryngeal Injury

Hematomas, joint articulation injuries, and lesions of the extrinsic laryngeal muscles are the cause of traumatic dysphonia.

Spastic Dysphonia

1. Spastic dysphonia is a discrete vocal disorder characterized by strained, choked vocal attacks (laryngeal stuttering) and is associated with increased tension of the entire phonatory system. The voice characteristics in spastic dysphonia include glottic stammering, hoarseness, monopitch voice, and reduced volume. The secondary characteristics associated with this disorder include facial and neck grimacing, fatigue of chest musculature, ticlike contractions of upper torso and face, and facial flushing. The onset usually follows some stressful period in middle-life and there is no female/male predominance. The etiology is unknown but it is thought to be a vocal expression of psychoneurotic behavior. Other authors have felt it to be secondary to a CNS or proprioceptive disorder of the larynx.

2. Laryngeal findings are those of hyperadduction of the vocal cords with the patient attempting to phonate against a closed glottis.

3. Treatment modalities:

a. Psychotherapy and speech therapy reported to have uniformly poor results after long periods of intervention.

b. Surgical therapy first proposed by Dedo was to deliberately section the RLN to prevent hyperadduction of the vocal cords. This was done after temporary paralysis with

lidocaine (Xylocaine) and showed significant improvement in voice quality. Sectioning of the RLN in spastic dysphonia patients has resuled in greater ease and improved quality of phonation, with reduction or elimination of facial and neck grimaces in most patients. Some authors advocate crushing of the RLN as opposed to section so that permanent paralysis would not occur (Biller and Som). Other authors suggest selective sectioning of the adductor branch of the recurrent laryngeal nerve (Carpenter et al).

c. One recent study by Aaronson showed a high rate of recurrence of spastic dysphonia symptoms (39%) 1-1.5 years after RLN sectioning. This can be treated in some case by deliberate paralysis of the ipsilateral superior laryngeal nerve.

Intractable Aspiration

Intractable aspiration resulting from loss of protective laryngeal function is seen with severe brain stem or cranial nerve IX, X, ad XII deficits.

Aspiration pneumonia can be a devastating and fatal complication of an otherwise debilitating but nonfatal illness, often a neurologic disorder.

Numerous nonsurgical and surgical techniques have been proposed to prevent this intractable life-threatening condition:

1. Nonsurgical techniques:

a. Nasogastric tube supplemented by IV feeding provides a temporary solution for obtaining nutrition. Long-term use of the tube may result in rhinosinusitis, postcricoid ulceration, and chondritis of the larynx.

2. Surgical techniques:

a. Cuffed tracheotomy tube for temporary relief from aspiration. Long-term cuffed tubes may result in stomal infection, tracheal stenosis, esophageal erosion, and innominate artery fistulization.

b. Vocal cord augmentation with Teflon injection. This is never completely effective in correcting aspiration caused by high vagal paralysis.

c. Cricopharyngeal myotomy to facilitate passage of food and minimize stasis of secretions.

d. Bilateral chorda tympani and tympanic nerve sections to reduce saliva production.

e. Gastrostomy (better tolerated than nasogastric tubes on a long-term basis).

f. Separation of the larynx and trachea by creation of a tracheostoma and closure of the larynx at the level of the first tracheal ring.

g. Use of epiglottic flap to arytenoids.

h. Linderman's diversion procedure with tracheoesophageal anastomosis and creation of a tracheostome. In this technique, aspirated saliva and food are diverted into the esophagus. This procedure may be reversible.

i. Montgomery's glottic closure procedure. Both vocal cords are sutured together via a laryngofissure; theoretically reversible. Sasaki and coworkers described a modified method of laryngeal closure.

j. Total laryngectomy.

Voice Restoration After Laryngectomy

1. In 1931 Guttman described a technique which restored voice in several patients. He created a fistula between the trachea and the pharynx using an electrocautery needle. Ever since, numerous methods of surgical restoration of voice after laryngectomy have been described which include Asai's laryngoplasty, air bypass voice prosthesis (1975), pseudoglottis procedure (Staffieri, Sisson and Goldman).

2. Singer-Blom tracheoesophageal puncture (TEP) technique: In 1979 Singer and Blom described a technique of voice restoration that created a fistula between the top of the tracheal stoma and the tube. A soft Silastic device was inserted in the fistula to maintain its patency and to allow shunting of air from the trachea into the hypopharynx without aspiration.

In 1981, Singer and Blom described 129 patients, 88% of whom had achieved fluent voices. They noted that esophageal voice is profoundly affected by the residual function of the pharyngeal constrictor musculature. Selective division of these muscles will enhance voice acquisition in most cases of failed esophageal speakers.

3. Candidates for the Singer-Blom procedure include any motivated patient who, after total laryngectomy, has an adequate-sized tracheostoma; a pharyngoesophageal lumen adequate to permit passage of an esophagoscope; sufficient hand-eye coordination to handle and take care of the prosthesis on a daily basis, or a family member who is willing to do this for the patient; and the ability to cover the stoma with the thumb or finger to divert airflow from the lungs through the device into the esophagus. Prior radiation therapy or partial or total pharyngectomy is not a contraindication.

4. In spite of some of the postoperative problems, it appears that at the present time this is the surgical procedure of choice to vocally rehabilitate the total laryngectomy patient.

Tracheotomy

General Considerations

1. Tracheotomy is done to form a temporary opening in the trachea. Tracheostomy, in which the trachea is brought to the skin and sewed in place, provides a permanent opening. Tracheostomy is usually done in connection with laryngectomy.

2. Tracheotomy is indicated for two groups of patients.

a. Those who have an obstruction at or above the level of the larynx (mechanical obstruction).

b. Those who have an actual obstruction to the airway but cannot raise secretions (secretional obstruction).

Tracheotomy in the second group is becoming increasingly common.

3. Elective tracheotomy may be necessary when respiratory problems are anticipated in the postoperative period in patients being subjected to major head and neck thoracic operations or in patients with chronic pulmonary insufficiency.

Therapeutic tracheotomy is indicated in any case of respiratory insufficiency due to alveolar hypoventilation in order to bypass obstruction, to remove secretions, or to provide for the use of mechanical artificial respiration.

4. Clinical signs of upper airway obstruction include:

a. Retraction (suprasternal, supraclavicular, intercostal).
b. Inspiratory stridor.
c. Restlessness, apprehension, disorientation, leading to coma.
d. Rising pulse and respiratory rates.
e. Pallor (earlier sign) and cyanosis (late danger sign).
f. Fatigue and exhaustion due to excessive efforts to breathe through an obstructed airway.

The patient's exhaustion must not be regarded as a sign of improvement, but rather a danger sign. It is a mistake to wait for late clinical signs of obstruction to appear before performing a tracheotomy. The time to proceed with the operation is whenever the possible need was first considered. Surgical manipulation in hypoxaemic patients often is associated with cardiac arrest.

5. Function of a tracheotomy: In addition to the bypass of an upper airway obstruction, tracheotomy has several other physiologic functions which include:

a. Decreasing the amount of dead space in the tracheobronchial tree, usually 70-100 mL. The decrease in dead space may vary from 10-50%, depending on the individual's physiologic dead space.

b. Reduction of resistance to airflow which, in turn, reduces the force required to move air. This will result in increased total compliance and more effective alveolar ventilation, provided the tracheotomy opening is large enough.

c. Protection against aspiration.

d. Enables swallowing without reflex apnea which is important in respiratory patients.

e. Access to the trachea for cleaning.

f. Pathway to deliver medication and humidification to the tracheobronchial tree, with or without intermittent positive pressure breathing.

g. Decreasing the power of the cough and thereby preventing peripheral displacement of secretions by the high negative intrathoracic pressure associated with the inspiratory phase normal cough.

6. Tracheotomy in infants and children should always be done after a bronchoscope, endotracheal tube, or catheter has been inserted to provide an airway and some rigidity to the trachea. This will convert an emergency tracheotomy to an orderly one. It is easy, in these small patients, to carry dissection too deeply and laterally to the trachea with resulting damage to the recurrent laryngeal nerve, the common carotid artery, apex of the pleura, or the cervical esophagus. Caution must be used when incising the tracheal wall not to insert the knife too deeply and lacerate the posterior wall. When the head of a child is turned or keeps moving, a trachea may be entered too laterally. A bronchoscope or endotracheal tube in the trachea will help eliminate these complications.

Indications

Indications for tracheotomy may be summarized as below:

A. Mechanical obstruction.

1. Obstructive tumors involving the larynx, pharynx, upper trachea and esophagus, thyroid gland.

a. When in advanced stage.
b. Edema from radiotherapy.
c. As adjunct to surgery.

2. Inflammation of larynx, trachea, tongue, and pharynx.

a. Acute epiglottitis.
b. Viral croup.
c. Ludwig's angina, etc.

3. Congenital anomalies obstructing larynx or trachea.

a. Laryngeal web or atresia.
b. Tracheoesophageal anomalies, etc.

4. Trauma of larynx and trachea.

a. Cartilaginous framework and soft tissues.
b. Inhalation of steam or fumes (burn).

5. Maxillofacial trauma with extensive bony and soft tissue damage.

a. Le Fort II, III, multiple fractures of mandible and maxilla, etc.
b. Hemorrhage.
c. As adjunct to surgery.

6. Bilateral vocal cord paralysis.

7. Foreign bodies.

8. Sleep apnea syndrome.

B. Secretional obstruction.

1. Retained secretions and inadequate cough.

a. Thoracic and abdominal surgery.
b. Bronchopneumonia.
c. Burns aboute face, neck, and respiratory tree.
d. Conditions producing coma, i.e. diabetes mellitus, uremia, septicemia, and liver failure.

2. Alveolar hypoventilation.

a. Drug intoxication and poisoning.
b. Flail chest, fractured ribs, and surgical emphysema.
c. Paralysis of chest wall.
d. Chronic obstructive pulmonary disease, i.e. emphysema, chronic bronchitis, atelectasis, bronchiectasis, and asthma.

3. Both retained secretions and alveolar hypoventilation.

a. Central nervous system disease, i.e. stroke, encephalitis, Guillan-Barre syndrome, poliomyelitis, and tetanus.
b. Eclampsia.
c. Massive head and chest injuries.
d. Neurosurgical postoperative coma.
e. Air and fat embolism.

f. Several of the conditions noted in (a) and (b) may include both alveolar hypoventilation and retained secretions.

Technique

Refer to standard textbooks.

Postoperative Considerations

1. Immediate postoperative chest x-rays (AP and lateral) are important to ascertain the length and position of the tracheotomy tube and to rule out complications of tracheotomy such as pneumomediastinum or pneumothorax.

2. The inner cannula of the tracheotomy tube should be removed and cleaned every 1-2 hours for the first 2 or 3 days to prevent obstruction by dried mucus. This is especially important in infants.

3. A tracheotomy tube in a fresh tracheotomy should be left in place 2-3 days before it is changed. By this time a permanent tract exists and there is little danger of being unable to reinsert the tube. Changing a tube before this time may result in loss of the tracheal opening into the neck wound with possible fatality.

4. A string around the neck should never be loosened "for comfort". The tube may slip out of the fresh tracheotomy wound.

5. Suctioning must be done often, especially during the first few days after tracheotomy because of the increase in tracheobronchial secretions secondary to tracheal irritation.

6. A tracheotomy should be left in place no longer than necessary, especially in children. Removal as soon as it is expedient will help reduce the incidence of tracheobronchitis, tracheal ulceration, tracheal stenosis, tracheomalacia, and persistent tracheocutaneous fistula.

Complications

Complications of tracheotomy may be summarized as follows:

A. Immediate.

1. Apnea.
2. Hemorrhage.
3. Pneumothorax and pneumomediastinum.
4. Subcutaneous emphysema.
5. Malpositioned tube.
6. Tracheoesophageal fistula.
7. Recurrent laryngeal nerve paralysis.
8. High tracheotomy (injury to the cricoid cartilage).

9. Aerophagia.
10. Aspiration of gastric contents.

B. Delayed.

1. Delayed hemorrhage.
2. Tracheoesophageal fistula.
3. Tracheocutaneous fistula.
4. Displacement or obstruction of a tube and a cuff.
5. Atelectasis and pulmonary infection.
6. Tracheomalacia.
7. Dysphagia.
8. Difficult decannulation.
9. Problems with neck scar.
10. Tracheal stenosis.

a. Apnea: When tracheotomy is performed on a patient with a history of chronic hypoxia, the patient may take one or two breaths right after the procedure and then suddenly become apneic. This is due to physiologic denervation of the peripheral chemoreceptors by the sudden increase of PO_2, and because hypoxia may be largely responsible for respiratory drive in these patients, apnea results. Some form of respiratory assistance is necessary until enough CO_2 is removed to allow a return of sensitivity of central chemoreceptors. The patient should never be left unattended after an emergency tracheotomy.

b. Hemorrhage: This may occur if hemostasis is not secured at operation.

c. Pneumothorax and pneumomediastinum: Pneumothorax may be caused by injury to the cupula of the pleura which rises into the neck in infants and young children and is subject to injury during the operative procedure. This usually occurs when the tracheotomy is done without prior establishment of an airway by a bronchoscope or an endotracheal tube.

Pneumomediastinum may result from air being sucked through the wound in a child severely obstructed and having violent respiratory movements, or it may result from excessive coughing which forces air into the deep tissue planes of the neck, which then dissects into the mediastinum. Should the parietal pleura rupture, pneumothorax will result. Pneumomediastinum may require no surgical therapy, but pneumothorax often requires the placement of chest tubes with an underwater seal.

d. Malpositioned tube: This is a frequent complication. Careful preoperative selection of a tube followed by postoperative roentgenographic evaluation will prevent this complication. Tubes of excessive length may impinge on the anterior wall of the trachea or the carina producing partial tracheal obstruction as well as ulceration and possible rupture of the innominate artery. The tube may extend down one bronchus with resultant atelectasis of the opposite lung. Too short a tube may predispose to displacement of the tube out of the trachea, especially when the neck is flexed in obese individuals or small children.

e. Tracheoesophageal fistula: This results from penetration through the muscular posterior tracheal wall into the esophagus, or approaching the trachea from the side. Recurrent

laryngeal nerve injury rarely occurs. These complications can be avoided by dissecting the midline of the neck and by inserting a rigid endotracheal airway.

f. Aerophagia: Aerophagia is seen most often in infants and young children and should be recognized as a cause of persistent dyspnea. It is treated with nasogastric tube decompression of the swallowed air. Death of an infant secondary to aerophagia with respiratory compromise has been reported.

g. Delayed hemorrhage: This is most often due to erosion of a major vessel by pressure necrosis from the cuff, or occasionally, the tip of the tracheotomy tube. Any bleeding occurring 4-5 days postoperatively should be given careful and immediate attention because of the threat that it may represent erosion into a major vessel. The innominate artery is the vessel most commonly involved with the common carotid, inferior and superior thyroid arteries, aortic arch, or occasionally, the innominate vein.

Mathog et al proposed possible preventative measures, which include the following: (1) adequate skin incision to allow visualization of palpation of abnormal vessels; (2) avoidance of a "low" tracheotomy, i.e. minimal extension of the head, gentle traction with a tracheal hook, and the stoma placed in the second and third tracheal rings; (3) elimination of metal tubes, with the use of plastic or silicone rubber tubes without a cuff and roentgenographic guidance to be certain the position and length of tubes are appropriate; (4) high humidity and aseptic care of the tracheotomy.

h. Delayed tracheoesophageal fistula: This is usually fatal and results from severe pressure necrosis from an overinflated cuff or from the tip of a malpositioned tube, the erosion occurring through the posterior tracheal wall and the anterior wall of the esophagus. Typically, the aspiration through the fistula results in severe pneumonitis.

i. Difficult decannulation: This is a frequent complication in children. A tracheotomy tube should be decannulated within 8-10 days (or sooner) whenever possible. If not, decannulation becomes difficult because (1) the child gets used to less resistance and less effort (tracheotomy decreases the dead space), (2) the child forgets the apneic reflex during deglutition, and (3) tracheal collapse develops.

j. Neck scar: The use of a vertical skin incision is the most frequent cause of unsightly scar formation. The duration of tracheotomy is also important in scarring which is lessened by early removal of the tube. Vertical contracture and widening of a hypertrophic scar will require a Z-plasty for repair.

k. Tracheal stenosis: Stenosis of the larynx follows injury and perichondritis of the cricoid cartilage which is the only circular tracheal support. Tracheal stenosis is most common in children and thought to result from excision of cartilage from the anterior tracheal wall. Exuberant granulations may develop on the anterior tracheal wall due to delayed epithelialization when there is a large defect in the anterior tracheal wall, and may cause obstruction and bleeding.

Tracheotomy related subglottic stenosis may be related to bacteriologic pathogenesis via tracheostomal contamination. Therefore the ability to control stomal contamination with

topical and/or systemic antibiotics may play a role in the prevention of the wound infection leading to cicatrical scar and stricture.

Endotracheal Intubation

1. Nasotracheal intubation in acute epiglottitis has been recommended. A smooth polyvinylchloride tube of somewhat smaller caliber than that corresponding to the patient's age is used. After intubation, children are placed in a cool-oxygen tent. The tube is tolerated well. Duration of intubation is usually 24-48 hours. Because most children with acute epiglottitis must be intubated to be tracheotomized, and because the critical perior for airway obstruction is at most 48 hours, nasotracheal intubation may be preferable to acute tracheotomy in a severely ill child. However, for those who are responsible for the occasional case, it is safer to rely on the time-tested tracheotomy. Endotracheal intubation should be done only by an experienced anesthesiologist or otolaryngologist.

2. There has been renewed interest in prolonged endotracheal intubation. Autopsy study showed:

a. Total damage and laryngeal ulceration in the intubated larynx and trachea were statistically related to duration of intubation but not to age or sex of the patient.

b. Significant ulcerations were confined to the posterior half of the larynx and the anterior and lateral aspects of the trachea between the third and tenth rings.

c. Intubation beyond 48 hours was associated with significant laryngeal ulceration, increasingly severe vocal process perichondritis, and frequent infection by microorganisms.

d. Intubation beyond 96 hours was associated with severe damage to the vocal processes and the subglottis, and a higher incidence of inferior vocal fold ulceration.

e. Tracheal damage in patients with multiple intubations or extubation before death was similar to that in the continuously intubated patients, but inflammation was more widespread and deeper.

f. Orotracheal intubation of adults for more than 96 hours may cause permanent damage.

3. Causes of inadequate ventilation with intubation include:

a. Mucus or clot obstruction.
b. Herniation of the cuff down over the end of the tube.
c. Collapse of the beveled end of the tube so as to block the lumen.
d. Lodging of the open portion of the beveled tube against the tracheal wall so as to occlude its lumen.
e. Kinking of the proximal unarmored end of the tube at its attachment to the adaptor.
f. Collapse of the endotracheal tube lumen by the inflated cuff.

Tracheotomy and Laryngeal Function

Prolonged tracheotomy may result in aspiration due to a weakened, ill-coordinated adductor reflex response resulting from behavioural alterations of medullary adductor motor neurons. Experimental evidence has show that phasic abductor activity in the posterior cricoarytenoid muscles diminishes as ventilatory resistance decreases. When airflow is shunted through a tracheotomy, abductor activity not only gradually diminishes, but completely disappears. The longer the duration of decreased ventilatory resistance, the more difficult it is to reestablish the abductor function once it is lost. This helps to explain the difficulty encountered in tracheotomy decannulation and fusion of denuded vocal cords (after endotracheal intubation injuries) when laryngeal abductor activity is lost resulting in the absence of phasic inspiratory abduction.

Neurologically Intact Larynx Before Tracheotomy. Vocal cords abduct and elongate on inspiration secondary to activation of the PCA (producing abduction) and CT muscle (producing elongation). During expiration the cords close to the median position (CT muscle adduction).

Neurologically Intact Larynx After Tracheotomy. Vocal cords remain in the intermediate position in both phases of the respiratory cycle. Decreased airway resistance produced by tracheotomy abolishes the physiologic activity of both the PCA and CT muscles. Therefore tracheotomy can and will cause lateralization of a paralyzed cord (in acute low vagal or RLN paralysis) and this should be considered in determining the neuroanatomic site of injury.

Sleep Apnoea

The sleep apnea syndrome (SAS) is now recognized as an important clinical entity caused by repetitive episodes of upper airway tract obstruction during sleep. If unrecognized and untreated this condition may lead to death.

1. Apnea is the cessation of airflow at the level of the nares and mouth, and is classified into three different types: (1) central, (2) obstructive or peripheral, and (c) mixed.

2. Central apnea occurs when the respiratory center fails to initiate a respiratory effort. Obstructive or peripheral apnea is the cessation of nasal and oral airflow with persistence of respiratory efforts. Respiratory efforts are manifested by a rocking movement of the chest and abdomen, and retraction of the intercostal and suprasternal spaces. Mixed apnea is a combination of central and obstructive apnea.

3. The duration of pathologic apnea episodes vary with age. Apnea episodes longer than 10 seconds for adults, 15 seconds for infants (up to 6 months old), and 20 seconds for premature infants are considered pathologic.

4. The sleep apnea syndrome is characterized by the presence of at least 30 true apneic episodes in both REM (rapid eye movement) and non-REM sleep, during 7 hours of unsedated nocturnal sleep. In the adult sleep apnea syndrome, the number of apneic episodes is usually several hundred per night.

5. The incidence of sleep apnea syndrome is unknown. However, its occurrence is probably underestimated.

6. The spectrum of this syndrome encompasses all age groups, being particularly prominent among the following:

a. In infancy where it may take the form of the sudden infant death syndrome (SIDS).

b. In early childhoo where it is associated with inflammatory diseases and hypertrophy of nasal and oropharyngeal structures.

c. In adults where it is commonly seen in obese white males in the fifth decade of life. Adults usually are affected by a central or mixed type of apnea, whereas children exhibit obstructive apneic spells in the majority of cases. There is a striking male preponderance in most reports of sleep apnea syndrome (20:1 to 60:1).

7. Central (medullary) chemoreceptors, peripheral (carotid body) chemoreceptors, and mechanoreceptors play a significant role in the physiology of respiratory control. Ventilation is increased upon stimulation of the medullary chemoreceptors by an increase in PCO_2. Carotid body chemoreceptors, located at the bifurcation of the common carotid arteries are responsive to PCO_2, H^+, and PO_2, and transmit their neural messages to the brain stem respiratory centers via the carotid sinus nerve (branch of the glossopharyngeal nerve). Mechanoreceptors consist of bronchopulmonary and chest wall subgroups. The former transmits neural activity to the brain stem respiratory centers via cervical vagal fibers.

8. The precise etiology of the sleep apnea syndrome is unknown. However, it is felt that a disarrangement of the neurologic control of the pharyngeal airway is an essential factor. The pharyngeal airway is the most compliant part of the upper airway due to its lack of skeletal support. Patency of the pharyngeal airway during sleep is maintained by the involuntary adjustment of muscular tone via the central nervous system. During inspiration the pharyngeal muscles must increase their tone to overcome the tendency of the pharyngeal airway to collapse. Patients with the sleep apnea syndrome may exhibit an aberration of this control mechanism. Fiberoptic studies of patients with obstructive SAS demonstrate recurrent closure of the velopharyngeal sphincter with opposition of the lateral pharyngeal walls at the level of the superior constrictor muscle.

9. Obstructive causes of SAS include:

a. Adenotonsillar hypertrophy. Adenoidal hypertrophy alone (normal tonsils) can cause cor pulmonale, systemic and pulmonary hypertension.

b. Nasal obstruction (septal deformity).

c. Nasopharyngeal abnormalities (stenosis, pharyngeal flap repair of palatal clefts or velopharyngeal insufficiency).

d. Pharyngeal obstruction (vallecular cysts, lingual tonsils).

e. Laryngeal obstruction (congenital webs, vocal cord paralysis, obstructive polyps and cysts, etc).

f. Craniofacial anomalies with posterior displacement of the hypomandibular complex (Pierre Robin syndrome, Treacher Collins' syndrome).

10. Approximately 80% of adults with obstructive sleep apnea present with daytime hypersomnolence. Other daytime symptoms include early morning headaches, easy fatigability, personality change, intellectual deterioration, poor memory, behavior problems, poor job and school performances. Nighttime symptoms include loud snoring, restlessness, somnambulance, nocturnal enuresis, insomnia, and nightmares.

One-third of sleep apnea patients are overweight and 25-60% are hypertensive. Digital clubbing, nasal polyps, facial and thoracic abnormalities, and endocrine disorders all serve as supportive evidence of SAS.

11. Arrhythmias are found in almost all patients with sleep apnea syndrome (up to 96%). These included sinus bradycardia, asystole, sinus arrhythmias, ventricular and sinus tachycardias, and 2° AV block.

12. There is increasing evidence of an association between sudden infant death syndrome and apnea during sleep. The incidence of SIDS is 2-3% per 1000 live births, with a definite male predominance. Ninety-one percent of the deaths occur before 6 months of age. Postmortem examinations of these infants have revealed pulmonary vasculature hypertrophy suggestive of chronic hypoxia prior to death. Pulmonary petechial hemorrhages also are found, suggestive of marked negative intrathoracic pressure (i.e. caused by trying to breathe against an obstruction).

13. The diagnostic evaluation of patients with sleep apnea syndrome usually involves polysomnographic monitoring since the physical examination is often benign. Polysomnographic monitoring consists of a graphic recording of extraocular movements, EEG, ECG, oral and nasal thermistors, and an abdominal strain gauge. This evaluation documents the relationship between the respiratory irregularity and the disturbed EEG pattern. The percentage of total sleep time spent in apnea is calculated, and further studies are undertaken (i.e. videofluoroscopy, nasopharyngoscopy) if the apnea is of the obstructive type and of significant duration (greater than 1% of total sleep time). Continuous skin surface electrodes provide and alternate method of documenting alveolar hypoventilation in a noninvasive manner.

14. Management modalities for central apnea include the following: methyl xanthines for infantile apnea, progesterone, theophyline, diaphragm pacing, acetazolamide, and tricyclid antidepressants have been used for adults.

Management of obstructive sleep apnea syndrome includes the following: specific reconstruction for orofacial anomalies, T and A for adenotonsillar hypertrophy, nasal surgery for nasal airway obstruction, oral and laryngoscopic surgery for hypopharyngeal and laryngeal obstruction, weight reduction, and endocrinologic evaluation and treatment. Obstructive areas not amenable to surgery must be bypassed with either nighttime nasopharyngeal airway tubes or tracheotomy.

Laser in Otolaryngology

Carbon Dioxide Laser

Biophysics

1. The word LASER is an acronym derived from the first letters of the words light amplification by stimulated emission of radiation.

2. A laser is a device for converting some form of "pumping" energy, such as heat, light, or electricity, into radiant energy of a special kind at one or more discrete wavelengths.

3. When the wavelength of radiant energy lies within the visible portion of the electromagnetic spectrum, it is called light, with which we are all familiar. However, not all lasers emit their radiant energy as light, and even those which do emit light produce a kind not found in natural phenomena.

4. The radiation emitted by all lasers has three special qualities:

a. It is coherent.
b. It is highly collimated.
c. It is monochromatic.

Coherent means that all the waves are exactly in phase (step) with each other in both space and time. Collimated means that the rays are parallel to each other. Monochromatic means that all the waves have exactly the same wavelength (or color, if they are visible).

5. The first lasers were made in the later 1950s and early 1960s. These early lasers utilized gems, such as rubies, through which radiant energy was passes or pumped through. Atoms in the ruby were stimulated or "excited" by the energy to cause them to emit coherent, collimated, and monochromatic radiation. This radiation could be focused with lenses and mirrors into a beam of enormously concentrated energy.

6. Early lasers were not very efficient, converting only about 1% of the "pumping energy" into laser energy. Lasers that utilized gases such as argon, neon, and carbon dioxide turned out to be much more efficient, converting as much as 15% of the incoming energy into laser energy. Also gas lasers can produce a continuous beam while a ruby laser produces a beam only in short pulses.

7. About one decade ago, the first practical gas lasers were developed. The most efficient of these is the carbon dioxide (CO_2) laser, converting about 15% of its pumping energy into coherent output radiation. The primary radiant output of the CO_2 laser occurs at a wavelength of 10.6 microm (a micron is one-millionth of a meter). This is in the infrared portion of the spectrum, where it is invisible to the human eye. Because of its relatively high efficiency and the fact that the lasing medium is a gas, so that cooling the laser is easy, it can be operated as a continuous-wave (not pulsed) device at power outputs up to 500 W or more.

8. The radiant waves from the CO_2 laser at 10.6 microm are ideal for surgical removal of tissue because:

a. They are strongly absorbed by all solids (except metals and certain metallic compounds), and by liquids, notably water, which is the major constituent of most living cells. In water, nearly all the radiant energy is absorbed within a depth of 100 microm from the irradiated surface.

b. They are not significantly scattered laterally from the target point.

c. Their absorption is not dependent on the color of the tissue.

d. They do not cause genetic mutation of the cells' nuclei (which may occur with gamma rays and x-rays). The physical effect of the waves from the CO_2 laser in any absorbing material is conversion of their radiant energy to heat.

e. They destroy cells and their nuclei (including those of bacteria) by flash boiling of the cellular water, which takes place at 100°C.

f. Because they are continuous waves, not pulsed, their average power is equal or comparable to their peak power. Thus there is no risk of causing a blast effect around the point where the laser beam strikes the tissue (a problem which makes pulsed lasers hazardous for many surgical uses).

g. They can be efficiently reflected from mirrors made of highly polished metals like stainless steel, which steer the laser beam under the manual control of the user.

h. They can be focused to very small spots (2 mm or less) by low-loss lenses made of special metallic compounds covered with ultrathin, antireflective coatings.

i. When deliberately defocused, they can be used to coagulate blood and seal small vessels in vascular tissue without vaporization of cells.

j. The CO_2 laser system delivering enough power for surgery is compact and operable from an ordinary electric wall outlet.

Laser Surgery in Otolaryngology

It has been found that the carbon dioxide laser provides a practical method of tissue ablation or excision which can be carried out with excellent control. The laser beam is directed with a hand piece in the oral cavity, with the surgical microscopic laser attachment (micromanipulator) in the nose, pharynx, and larynx, and with the endoscope-bronchoscope assembly in the tracheobronchial tree.

Laser surgery appears to be most valuable when precise surgery is needed to preserve function.

The laser beam can be used to vaporize predetermined volumes of tissue in a precisely controlled fashion by using an appropriate amount of energy. A control knob sets the power level of the beam and a foot-switch-controlled interval times operates the shutter allowing the beam to impact on the target area for an appropriate period. For precise microsurgery, a power setting of 15 W is commonly combined with a time exposure of 0.20 sec; for gross dissection 15-25 W of power may be used continuously in the manual mode, bypassing the timer, using the foot switch to start and stop the dissection. In contrast to pulsed lasers, the continuous-wave carbon dioxide laser has comparatively little impact shock effect, so that it has minimal tendency to scatter soft tissue.

Clinical Application

Otolaryngologic application of CO_2 laser includes:

1. Excision of benign lesions of:

a. Larynx (polyps, nodules, granuloma, papilloma, hemangioma, leukoplakia, web, subglottic stenosis, etc).
b. Trachea and bronchus.
c. Nose (papilloma, polyps, granuloma, hereditary telangiectasia, rinophyma, etc).
d. Oral cavity (lesions of tongue, palate, valleculae, hypopharynx, tonsil fossae).
e. Nasopharynx (with stainless steel mirror; recurrent adenoids, other inaccessible lesions).

2. Excision and palliation of malignant lesions of the oral cavity, hypopharynx, larynx, trachea, and bronchus.

a. Early cancer in these regions can be cured by laser excision.
b. Use of laser for malignant lesions is for diagnosis (biopsy), debulking, improved airway, and cure.

3. Surgical procedures such as:

a. Excision and repair of choanal atresia (Healy).
b. Partial turbinectomy.
c. Intranasal antrostomy.
d. Palatine tonsillectomy.
e. Lingual tonsillectomy.
f. Arytenoidectomy.
g. Partial epiglottidectomy.

Advantages

1. Precise excision.
2. Rapid tissue destruction.
3. Reach inaccessible sites.
4. Excellent hemostasis.
5. Minimal postoperative edema, pain, and scarring.
6. Avoid tracheotomy.
7. Minimize hospitalization.

Disadvantages

1. Costly.
2. Time consuming.
3. May be harmful to patients and operating room personnel if proper precautions are not taken.

Precautions

1. Protect eyes.
2. Protect adjacent tissues.
3. Avoid flammable anesthetics.
4. Protect endotracheal tubes.
5. Keep protective gauze wet.

Management of Complications

1. Complications of CO_2 laser microlaryngoscopy are generally associated with striking unprotected materials in the airway or unprotected tissues of the patient or operating room personnel. To reduce the risk of ocular damage the patient's eyes should be covered by moistened eye pads and all operating personnel should wear plastic protective glasses. The radiation emitted by the CO_2 laser is predominantly absorbed in the cornea, not the retina, and gross corneal opacification can result from thermal denaturation and coagulation of proteins.

2. Precautions should be taken to avoid endotracheal tube ignition during CO_2 laser surgery. This involves wrapping the tube with reflective aluminium tape and placing moist neurologic cottonoids to cover the entire balloon.

3. Results of irradiating polyvinyl chloride and red rubber tubes indicate that red rubber is less flammable and therefore safer to use in conjunction with the CO_2 laser.

4. Management of endotracheal tube ignition involves the following:

a. Remove the damaged tube while the patient is paralyzed to facilitate reintubation without laryngospasm.

b. Intravenous steroids and antibiotics are given as if treating a tracheal or pulmonary burn.

c. Intraoperative bronchoscopy is performed to remove any charred debris and assess the extend of damage.

d. Delayed extubation with reexamination of the subglottic and trachea may be needed to assess the extent of further airway compromise.

Argon vs. CO_2 Laser

1. The CO_2 laser is an ideal for incising, excising, vaporizing, or debulking soft tissue masses with precision and good visibility. Because the argon wavelength (0.488-0.515 microm) is shorter than that of the CO_2 (16.6 microm), it is possible to generate a smaller spot with the argon under similar working conditions. As a visible beam, the argon wabelength is easily transportable by fiber optic light carriers. These two factors make the argon laser more suitable for precise otologic microsurgery. As a soft tissue laser the preferential absorption of argon radiation by red tissue makes it possible for the first time to eliminate hemangiomatous lesions without physical contact and with minimal disturbance of the overlying skin.

2. The CO_2, which has the infrared wavelength (10.6 microm) is efficiently absorbed by water and since biologic tissues contain 80% water, it is an ideal medium for absorbing infrared radiation. The argon wavelength (0.48 microm) is in the visible blue green spectrum and preferentially absorbed by a red medium.

3. The primary effect of CO_2 laser is vaporization while that produced by the argon is photocoagulation.

4. Characteristics of argon and CO_2 lasers are shown in Table 15.2.

Table 15-2. Comparison of CO_2 and Argon Lasers

Characteristics	Argon	CO_2
Energy	Thermal	Thermal
Wavelength	Visible (0.48 microm)	Nonvisible (10.6 microm)
Color	Blue green	Infrared
Intensity	1-10 W	10-100 W
Spot size	0.1 mm	1 mm
Cooling system	Open	Closed
Fiberoptic	Yes	No.

Applications of Argon Laser in Otology

1. Myringotomy.
2. Ear canal osteoma.
3. Tympanoplasty.
4. Stapedectomy (Perkins 1980).
5. Hemostasis.
6. Lysis of adhesions.
7. Acoustic neuroma surgery (Glasscock 1981).

Because of the nature of the CO_2 beam, it is necessary to mount the laser itself onto the operating microscope, thereby making it difficult to adapt to otologic microscurgery. The ability of the argon laser to be transferred through a fiberoptic bundle has made it possible to introduce a whole new concept to microsurgery of the ear and adjacent structures.