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laryngeal position in newborns and young infants. This arrangement prevents the mixing of ingested food and inhaled air, thereby enabling the baby to breathe and swallow liquids almost simultaneously in a manner similar to that of monkeys. Thus, the baby can breathe through the nose with only minimal, if any, cessations as liquid flows from the oral cavity around the larynx into the esophagus (Fig 2–4A). Because of this high laryngeal position, newborns are essentially, if not obligatorily, nose breathers.[49,50] Indeed, studies of infants that have had deviation of the epiglottis/larynx due to ankyloglossia, and thus an anterior displacement of the larynx dislodging it from the palate and nasopharynx, demonstrated both suckling difficulties and unstable and low arterial oxygen percent saturation levels (SaO_2). Correction of the ankyloglossia allowed the larynx to regain its normal position with epiglottic/palatal overlap in the nasopharynx with concomitant improvement of deficits.[51] As with nonhuman primates, the connection between the epiglottis and the soft palate is usually constant, but may be interrupted during the swallowing of a particularly large or dense bolus of food or liquid, during vocalization or crying, or because of disease as noted above.

Although the high position of the larynx in a human newborn or young infant effectuates the

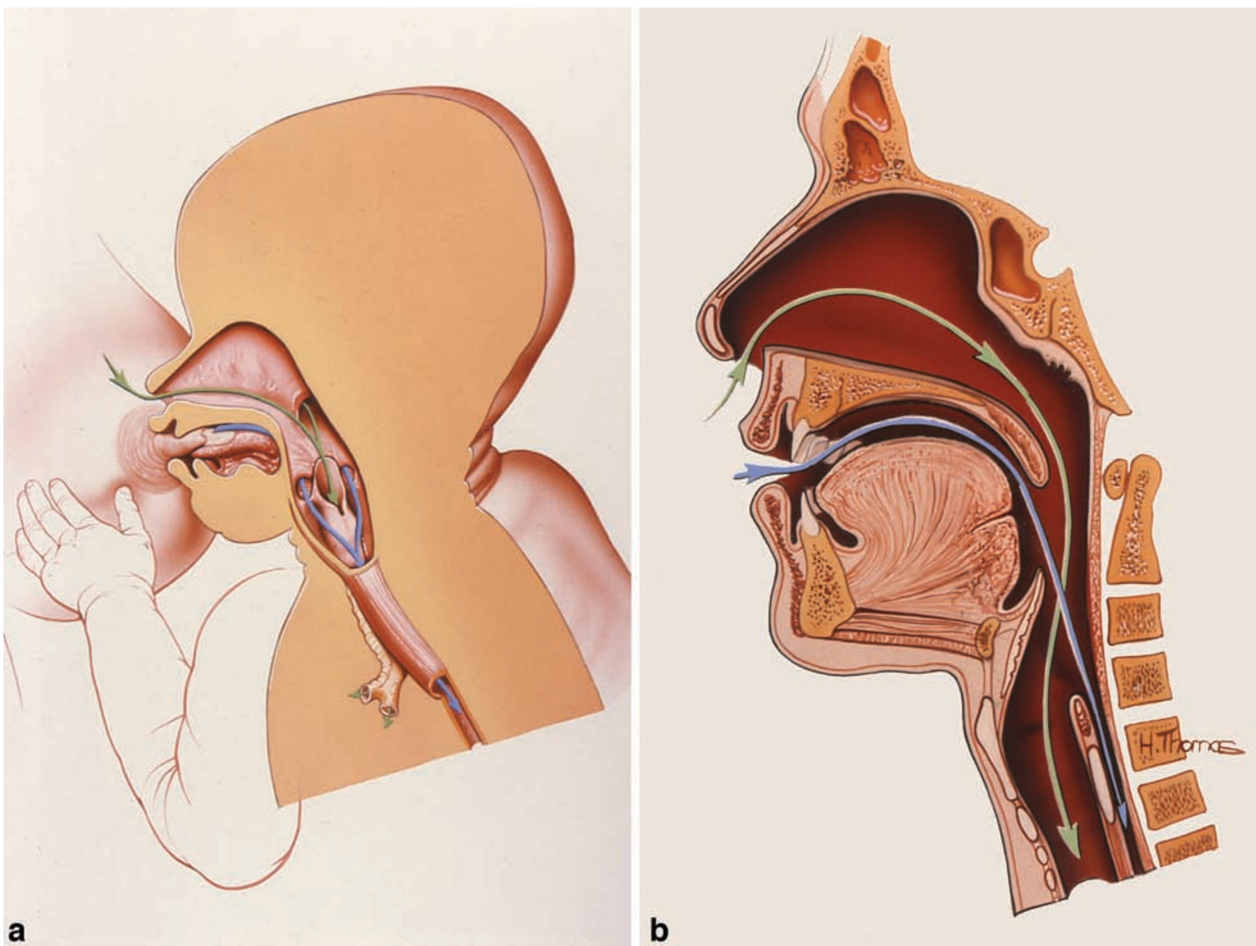


Fig 2–4. Drawings depicting: **A.** the aerodigestive tract of a newborn human during suckling and **B.** the aerodigestive region in an adult human. Green arrows = respiratory route, blue arrows = digestive route. Note that the high laryngeal position in the infant effectuates largely distinct pathways whereas the lowered position of the larynx and tongue in the adult mandates the crossing of pathways.

Fig 2–5. Reconstruction of the head and neck anatomy of *Australopithecus africanus*, an early human ancestor, during quiet nasal respiration (based upon the fossil Sts 5 from Sterkfontein, South Africa; for discussion see text and reference 80). As with living monkeys and apes, we hypothesize that the earliest hominids would exhibit a highly positioned, intranarial larynx during nasal breathing, as well as during ingestion of some foods. The high larynx would also have limited the supralaryngeal area and thus the ability to modify laryngeal sounds compared to living humans.

respiratory and upper digestive maladies probably evolved along with our laryngeal shifts.[6,60,62–63]

What force or forces could have caused this change? Although the answer is probably a matrix of factors, the prime generator may be based in our ancestor's need to feed the respiratory system's requirement for increased air intake and oxygen. Such need could have been instigated by a series of evolutionary events that seminally affected our ancestors at this time. Prominent among these would have been the marked increase in brain size and—arguably complexity—with early members of *Homo*[82,83] that concomitantly could have increased oxygen demands on the system. Increased brain size may also have structurally affected the cranial base, arguably causing internal flexion affecting laryngeal position. In addition, the necessity of short burst, or endurance, running on the African savannas to escape fast predators or chase equally fast prey may have become increasingly important. Indeed, recent studies have suggested that the human body plan is specifically designed to maximize endurance running, and that this ability likely evolved at this time as well.[84] Rather than redesign our nasal complex in order to capture additional oxygen—evolution could have endowed us with

Fig 3–20. Stage 19. 18-mm crown-rump length embryo.

Fig 3–21. Sagittal section approximately in the median plane, at stage 19 (18 mm). The vertebral centra can be seen on the right, and the base of the skull (containing a part of the notochord) can be seen at the top of the photograph. Below the epiglottis, the cartilaginous hyoid condensation is visible. (From Tucker JA, O'Rahilly R. Observations on the embryology of the human larynx. *Ann. Otol* 1972; 81:520–523.)

Figure 3–23 shows the Carnegie stage 19 embryo. The laryngeal cecum, which originates as a triangular lumen extending along the ventral aspect of the arytenoid swellings, continues its caudal descent until it reaches the level of the glottic region. The epithelial lamina completely separates the ventral laryngeal cecum from the dorsal pharyngoglottic duct.

Stage 20

The embryo is approximately 51 days of age and 18 to 22 mm in length. The cartilaginous hyoid is visible below the epiglottis. Perichondrial development (appositional growth) is evident. Cartilaginous appositional growth also represents a shifting of the embryonic center from interstitial to appositional growth (Figs 3–24 and 3–25).

Stage 21

The embryo is approximately 52 days of age and 22 to 44 mm in length. The thyroid gland is identifiable, inferolateral to the cricoid cartilage (Fig 3–26). The coronal section through the laryngeal region demonstrates the triangular appearance of the infraglottic lumen (Fig 3–27). The epithelial lamina completely separates the laryngeal cecum from the infraglottic lumen.

Stage 22

The embryo is approximately 54 days of age and 23 to 28 mm in length. Maturation of the anlagen of the intrinsic and extrinsic muscular tissue as well as the laryngeal cartilages continues.

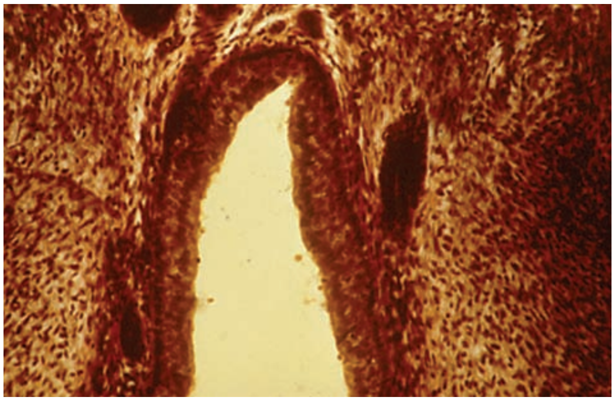


Fig 3–44. Tucker Fetal Collection (20 weeks). Membranous anterior commissure of the vocal fold.

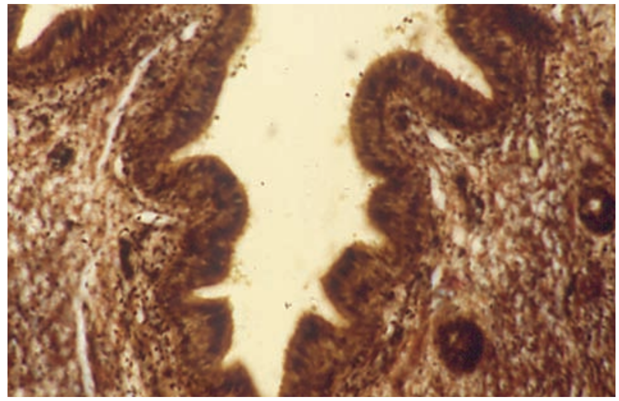


Fig 3–46. Tucker Fetal Collection (5 months). High power view of the posterior glottis respiratory epithelium.

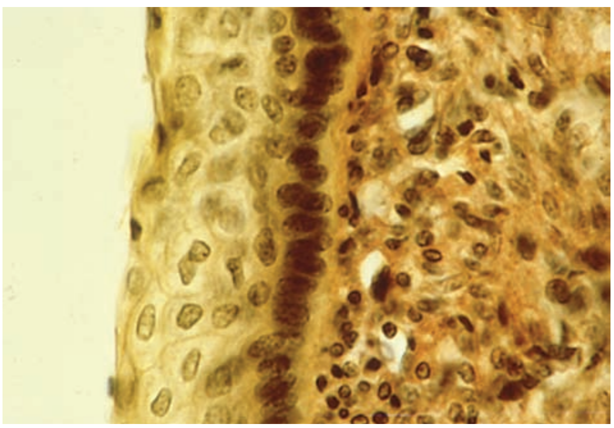


Fig 3–45. Tucker Fetal Collection (5 months). High power view of the epithelium of the anterior vocal fold with first seen squamous epithelium.

noid facets are well defined, smooth, and symmetrical. Each arytenoid articulates with an elliptical facet on the posterior superior margin of the cricoid ring. The cricoid facet is approximately 6 mm long and has a cylindrical shape.[19] Most cricoarytenoid motion is rocking; however, along the long axis of the cricoid facet, gliding also occurs.[20] The cricoarytenoid joint is an arthrodial joint, supported by a capsule lined with synovium and supported posteriorly by the cricoarytenoid ligament.[21] The cricoarytenoid joint controls abduction and adduction of the true vocal folds, thereby facilitating respiration, phonation and protection of the airway.

Fig 3–47. Sagittal section of a 5th-month fetus, Tucker Fetal Collection No. 411, 140 mm in length. Fibroelastic cartilage is present in the epiglottis (*E*). (From Tucker JA, Tucker and Tucker.[2])

Fig 4–16. After the administration of topical anesthesia, the child is positioned on the parent's lap with the arms and head gently secured. The infant safely tolerates this examination with minimal discomfort.

Table 4–1. Recommendations for Selection of Endotracheal Tubes and Bronchoscopes for Pediatric Patients

	Endotracheal Tube		Bronchoscope		
Patient Age	ID (mm)	OD (mm)	Size	ID (mm)	OD (mm)
Premature	2.0–3.0	2.8–4.3	2.5	3.5	4.2
0–3 MO	3.5	5.0	3.0	4.3	5.0
3–9 MO	4.0	5.6	3.5	5.0	5.7
9 MO–2 YR	4.5	6.2	3.7	5.7	6.4
2–3 YR	5.0	7.0	4.0	6.0	6.7
4–5 YR	5.5	7.6	4.5	6.6	7.3
6–7 YR	6.0	8.2	5.0	7.1	7.8

Note: These endotracheal tube sizes refer to uncuffed tubes.

In preparation for a pediatric tracheotomy, several points bear mention. The hyoid bone represents the most obvious palpable landmark. The thyroid cartilage often cannot be felt and the cricoid, although it has some definition, may feel like a large 1st tracheal ring. The thyrohyoid and cricothyroid membranes cannot be felt easily but may be inferred from the positions of the hyoid bone and cricoid cartilage.

In addition to anatomic considerations, the growth of the larynx has an effect on common pediatric laryngeal pathologies. The most commonly encountered laryngeal problems are (1) laryngomalacia, (2) vocal fold paralysis/paresis, and (3) subglottic

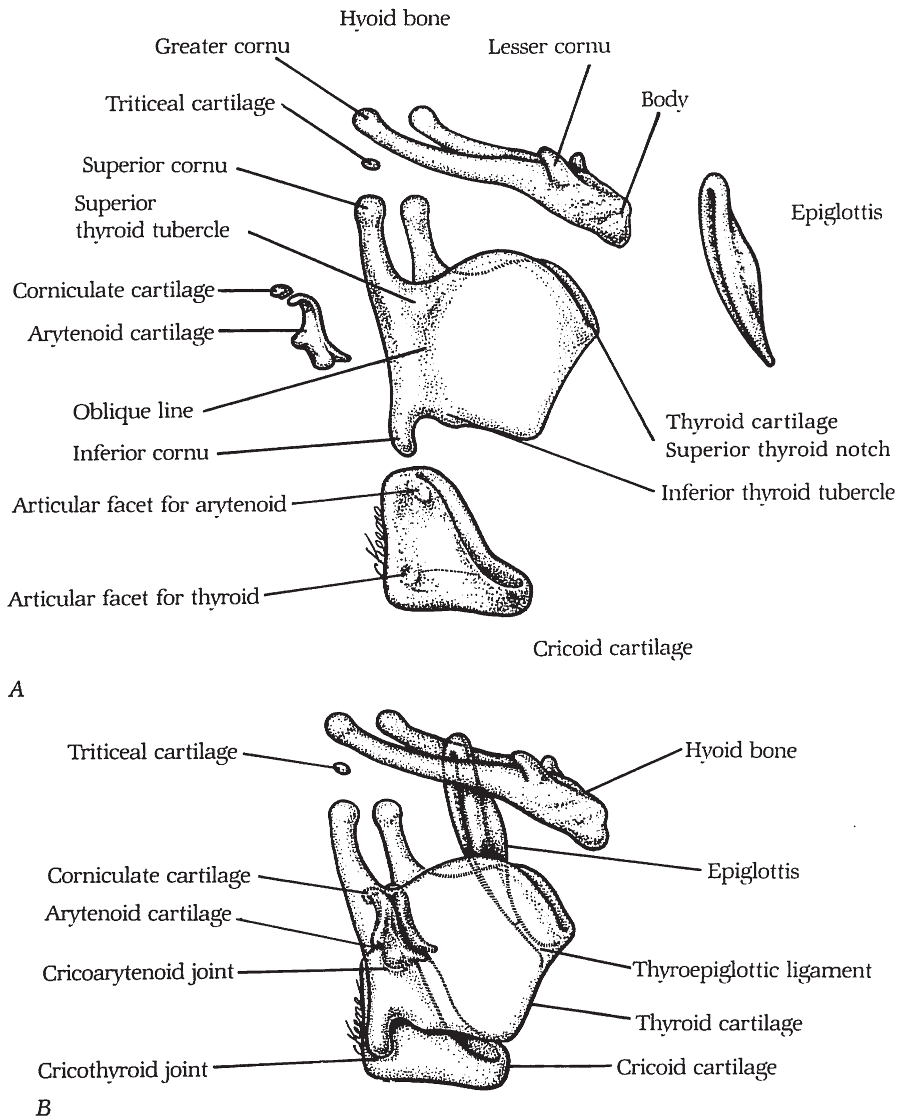


Fig 5–3. Disarticulated larynx (**A**) and in normal approximation (**B**).

of the arytenoid cartilages are composed of elastic fibrocartilage and do not undergo ossification.

The male and female larynges differ little in size up to puberty. Thereafter, while the female larynx grows slightly, the male larynx enlarges in all dimensions, reaching the following average proportions.[3]

	Male	Female
Length	44 mm	36 mm
Transverse diameter	43 mm	41 mm
Anteroposterior diameter	36 mm	26 mm

The thyroid cartilage (*thyrus* = "shield," Greek) is the largest laryngeal cartilage, and it shields the opening to the airway and supports most of the soft tissue folds in the larynx. The angle between the laminae of the thyroid cartilage exhibits sexual dimorphism with a 90° angle in men and a 120° angle in women. This angle is similar in both sexes before puberty. The laminae fuse at the midline symphysis with a narrow strip of cartilage, the intrathyroid cartilage, after birth, but it is not unusual to find residual intrathyroid cartilage in the midline in the infant. In men, the isthmus forms a downward

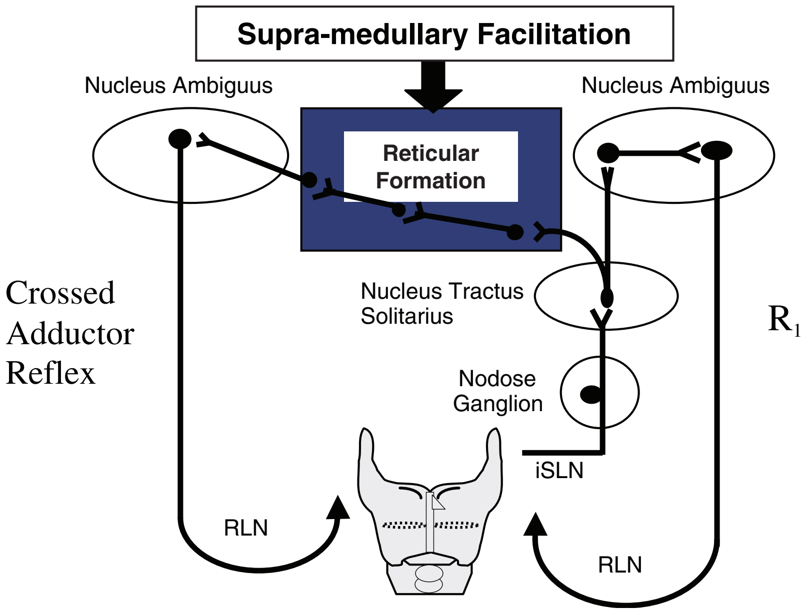


Fig 6–3. Organizational model of the ipsilateral and crossed adductor reflex pathway in man.

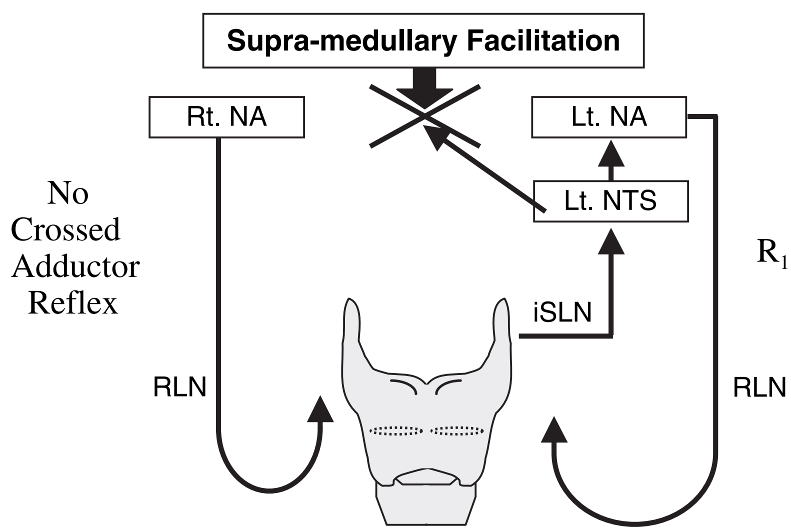


Fig 6–4. Organizational model demonstrating the loss of contralateral R_1 under deep anesthesia.

cartilages in the posterior gap, completes the first of the 3 sphincteric tiers of protection. The second tier of protection occurs at the level of the false cords, consisting of bilateral folds forming the roof of each laryngeal ventricle. The third tier of protection occurs at the level of the true vocal cords, which in man are shelflike with a slightly upturned free border. The inferior division of the thyroarytenoid muscle forms the bulk of this shelf, and with the passive valvular effect of the upturned border or the true cord margin, the true vocal cord perhaps is the most significant of the 3 barriers to aspiration.

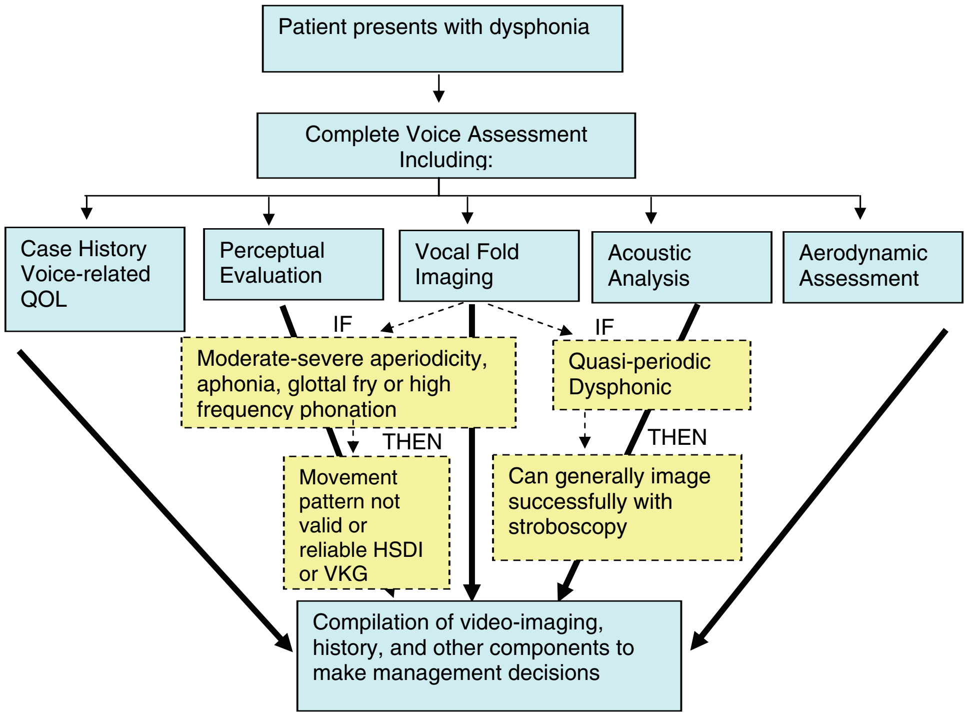


Fig 11–12. Flow diagram illustrating the role of stroboscopy and how it might be used to help management decisions and to determine the need for additional visualization techniques.

Tests of vocal function, including all forms of laryngeal imaging, have come a long way from their inception in the latter part of the 19th century, and as we move forward in this 21st century, we may be edging toward more science and less art.[39,49,51,53,64,69] Group data related to age, gender, phoneme produced, and vocal training are increasingly available.[17,30,67,70,91–93]

Instruments designed specifically to quantify voice production are increasingly available. Clinical studies have documented the effectiveness of these instrumental measures for monitoring treatment effectiveness. Although voice testing has not taken on the aura or routine nature of the audiogram, it has matured to the point that it no longer seems acceptable to describe the voice by merely listening to the patient phonate or inspecting the larynx with a mirror and standard light source. The evolution of voice testing is in its infancy. Future development is likely to unfold similarly to audiometric testing, with normative data and standards for testing and routine measures likely to be developed and imposed on voice clinicians, to ensure quality of service of the patient. International standards committees have already been formed and have presented preliminary recommendations. Data collected from voice instruments are likely to improve understanding of the physiologic and anatomic aspects of voice production as well as the pathogenesis of various laryngeal diseases.

In this century, with rising health care costs and moves to contain them, clinicians are likely to be faced with tough choices concerning equipment selection. They would do well to be guided by a few simple questions: Does the instrument cover the entire range of production seen in children and adults with normal and disordered voice; does the instrument provide simultaneous measures of at least 2 parameters of vocal function; do the resultant data have a unique value that has direct clinical application; are the derived measures both valid and reliable; are the derived measures necessary to further

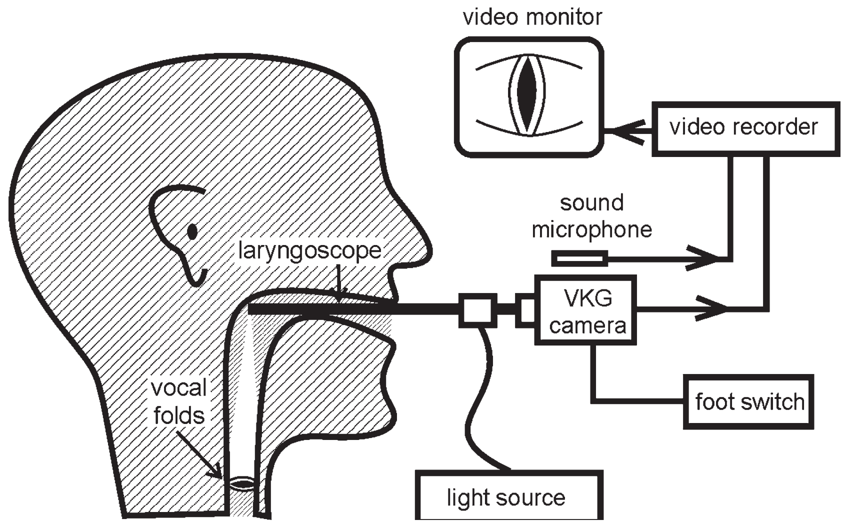


Fig 15–1. Equipment setup in videokymography.

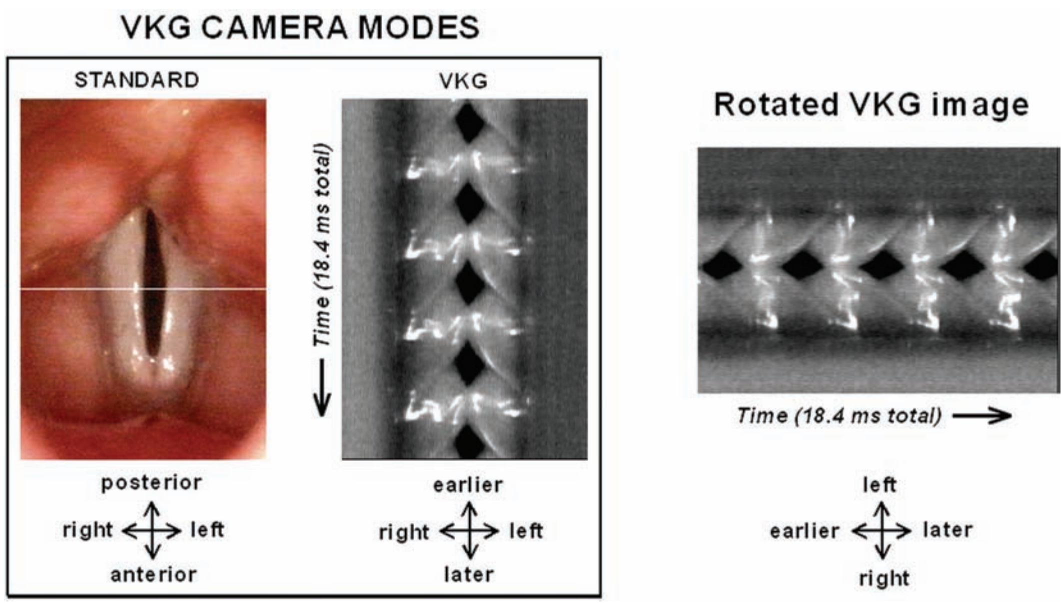
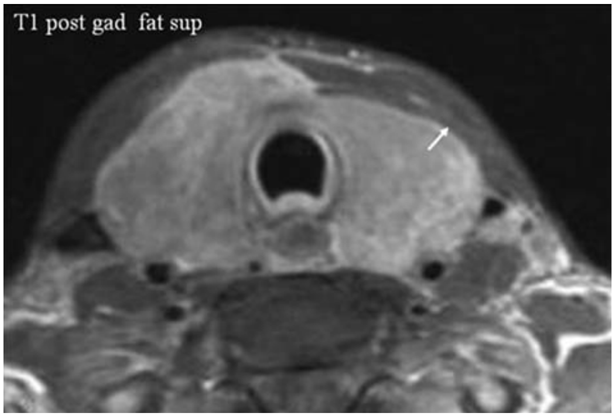
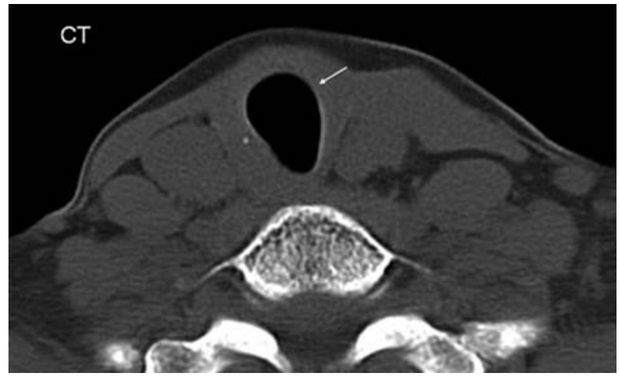


Fig 15–2. Two modes of the videokymographic camera. The measuring position for the VKG image is marked by the line in the standard image; the videokymogram displays the vibratory pattern of the middle part of the vocal folds. On the very right, the VKG image is rotated to show the time along horizontal axis. The investigated subject was a female with normal vocal folds phonating with moderate effort at the frequency of about 250 Hz.

delivers kymographic images. These are obtained by putting together many (almost 150) successive images from the same line recorded at a high-speed rate (almost 8000 line images per second). These kymographic images, which are composed and delivered by the videokymographic camera in real time, are called "videokymographic images" or simply "videokymograms." During phonation,



a



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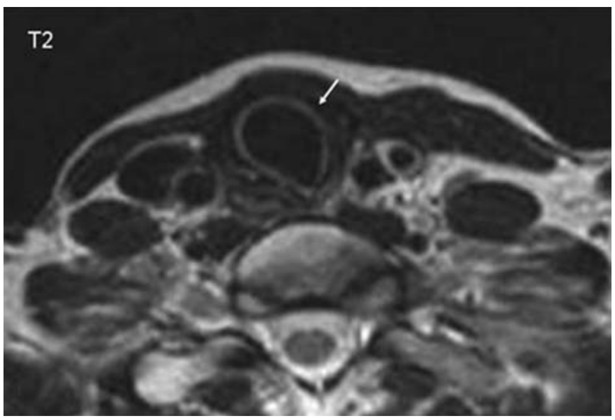

c

Fig 16–8. MRI soft tissue resolution: advantageous for cartilage visualization. (a) Patient with large thyroid carcinoma surrounding trachea and cricoid cartilages. (a) Postgadolinium T1 fat suppressed image identifies cartilaginous tissue as well as accentuates tumor enhancement and its interface with adjacent tissues (*arrow*). (b, d) Different patient, post-thyroidectomy with residual tracheomalacia. (b) axial unenhanced CT incompletely identifies tracheal cartilage (*arrow*) (c) Axial T2 image better displays cartilage structure, its thickness and continuity, and relationship to the tracheal lumen and the thyroid bed tissues.

and the arytenoids can be visualized superior to the cricoid cartilage.

Coronal views are ideal for assessing the superior to inferior extent of neoplasms. In particular, the mucosal and submucosal extent of disease not apparent clinically may be assessed, which is particularly useful for assessment of subglottic extension of disease. The high signal of the fatty tissue in the normal paraglottic space offers an excellent anatomic landmark on coronal and axial T1W sequences. If the ventricle is not seen well, the false cord can be differentiated from the true cord on T1W images by the fatty signal of the false cord versus the intermediate density thyroarytenoid muscle signal of the true cord.

Axial views are best for evaluating laryngeal cartilage invasion but the appearance is quite variable depending on the degree of ossification. Ossified cartilage, which has medullary fat, will have a bright signal on T1W and FSE T2 images and a lower (darker) signal on conventional spin echo (CSE)T2W images and is excellent indicator for the lack of tumor involvement of that cartilage. Nonossified cartilage is usually of low signal on both T1W and T2W sequences. Ossified cartilage cortex looks black (hypointense or low signal) on both sequences. The cervical lymph nodes can be assessed on either sequence, but are more easily visualized on the T2 or post gadolinium T1 sequences.

COMPUTED TOMOGRAPHY VERSUS MAGNETIC RESONANCE IMAGING[36,37,43,49,50] (See Fig 16–9)

The advantages of CT, in general, include greater availability or accessibility, greater patient acceptance or compliance, lower cost, faster image acquisition seen in Figure 27–4, other causes of supraglottic stenosis are related to infections of the supraglottic and epiglottic tissue and cartilage complicated by the trauma of intubation.

Acquired glottic stenosis occurs in the anterior commissure or the posterior glottis in the intra-arytenoid region. Anterior commissure stenosis is usually the result of an iatrogenic injury such as operating on both sides of the anterior commissure at the same time leading to scarring. As seen in Figure 27–5, in the pediatric patient population, the likely cause is overly aggressive removal of laryngeal papilloma in the anterior commissure region.

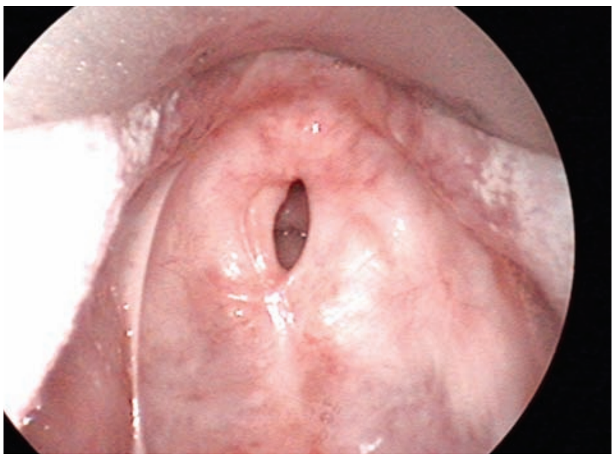

Fig 27–4. Supraglottic stenosis involving the epiglottis, intra-arytenoid and aryepiglottic fold region in a child following a bacterial infection of the epiglottis.

The etiology of posterior glottic stenosis in children is usually attributed to prolonged intubation[27–29] as seen in Figure 27–6. Although posterior-glottic stenosis is often seen in conjunction with subglottic stenosis, it also occurs as an isolated condition. Other causes of posterior glottic stenosis are related infection, inflammatory conditions, neoplasms, and trauma and are outlined in Table 27–2. Transglottic stenosis is rare and unfortunate. Etiology is similar to posterior glottic stenosis.

EVALUATION AND MANAGEMENT

Symptoms of glottic and subglottic stenosis relate to airway, voice, and swallowing. The evaluation of a child with suspected glottic or subglottic stenosis varies based on the acuity and status of the airway, and depends on whether the child is intubated, unintubated, or already has a tracheotomy. An acutely obstructed child is approached much differently than a stable child with a tracheotomy or an intubated child. Stridor is the most common symptom

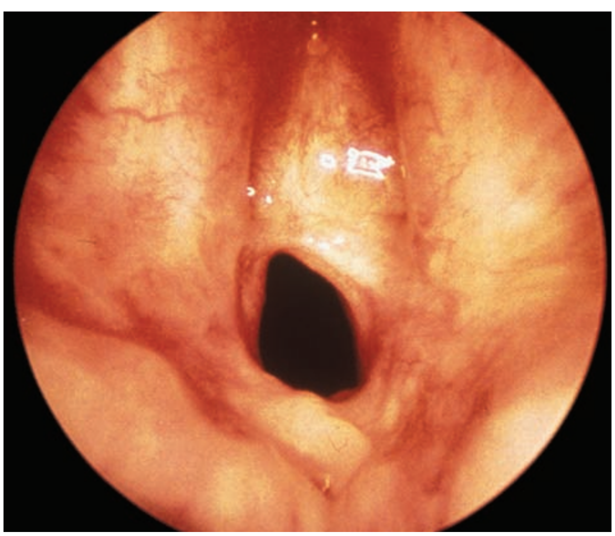

Fig 27–5. Anterior glottic web formation after overly aggressive laryngeal papilloma surgery.

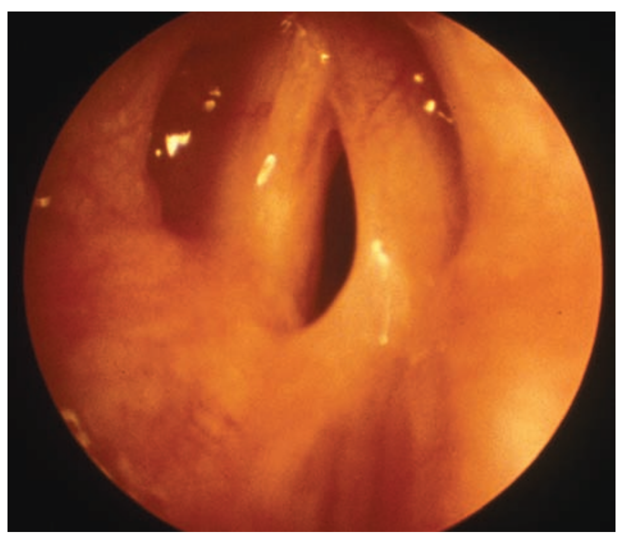

Fig 27–6. Posterior glottic stenosis from prolonged intubation in a patient with pathologic GERD.

describes a ratchetlike sensation felt during passive flexion extension of the upper limb or neck. Bradykinesia refers to slowness of voluntary movement. When this affects the muscles of facial expression, it yields a characteristic lack of facial affect described in the medical literature as "masklike." The integrity of postural reflexes can be easily evaluated by asking the patient to walk 3 or 4 paces and turn 180 degrees. Affected patients will not be able to pivot, but instead will take several steps to rotate their body.

The underlying pathology is neuronal loss in the substantia nigra and consequent loss of dopamine in the basal ganglia. The mainstay of treatment remains dopamine replacement, augmented by agents to boost transit of levodopa across the blood-brain barrier, and other dopamine receptor agonists that have been developed in recent years. The aim is symptomatic relief, and the underlying disease remains relentlessly progressive. Stimulation of deep brain nuclei by means of a surgically implanted electrode is gaining popularity in incapacitating cases of parkinsonism that resist medical management.

Parkinson Hypophonia

The characteristic low-intensity monotone voice of patients with PD has been termed Parkinson hypophonia. The laryngoscopic correlates of Parkinson hypophonia include vocal fold bowing (spindle-shaped glottic insufficiency) (Fig 34–2), vocal fold bradykinesia, and tremor.[6–8] These laryngeal abnormalities usually coexist with oral-motor articulatory difficulties and poor respiratory function and coordination, both reflections of underlying rigidity and bradykinesia. These are not infrequently accompanied by cognitive dysfunction.[9–13] In addition, underestimation of own-speech volume is a perceptual anomaly that has been found consistently in patients with Parkinson hypophonia, and may be a critical derangement in this type of dysphonia.[14,15]

Standard medical treatment with levodopa generally improves speech and voice difficulties in PD.[16–18] Early enthusiasm for vocal fold augmentation to remedy the observed glottic insufficiency[19,20] has been tempered by an understanding for the broader range of abnormalities involved in Parkinson hypophonia; only in the carefully selected patient without severe respiratory or articulatory dysfunction is augmentation likely to offer appreciable benefit. Deep brain stimulation (DBS) has been shown to be most effective in tremor suppression, but evidence for practical voice and speech benefit from this procedure remains equivocal.

The most effective intervention, apart from systemic medication, may be behavioral therapy. Lee Silverman Voice Therapy (LSVT) is an intensive course of behavioral therapy that aims to increase phonatory effort to overcome the impaired self-perception of loudness and appears to result in

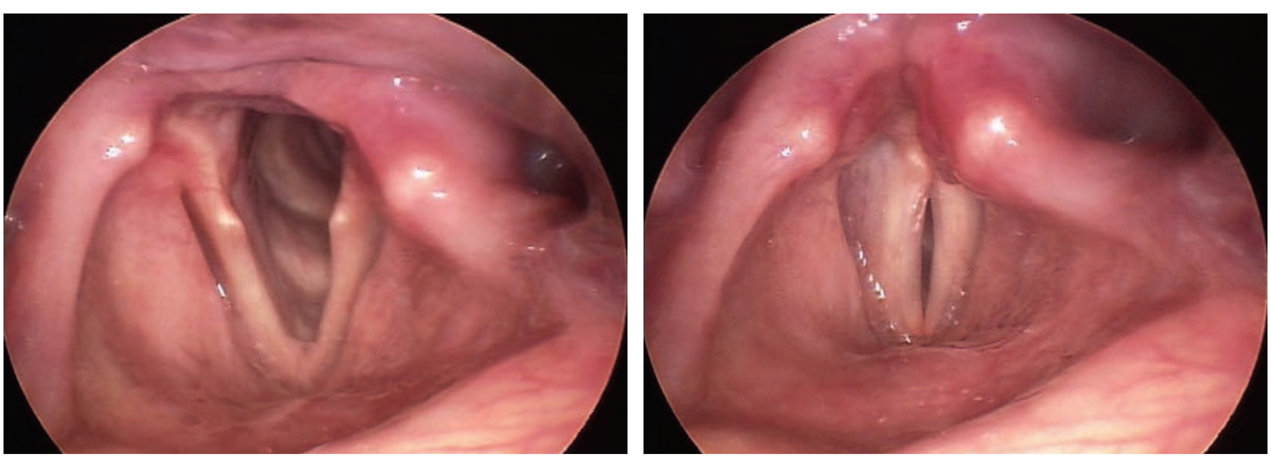

Fig 34–2. Best phonatory glottal closure is shown at right in this 56-year-old attorney with Parkinson's disease. Glottic insufficiency is exacerbated by early vocal fold atrophy. Symptoms were typical of Parkinson hypophonia, and the patient improved with voice therapy.

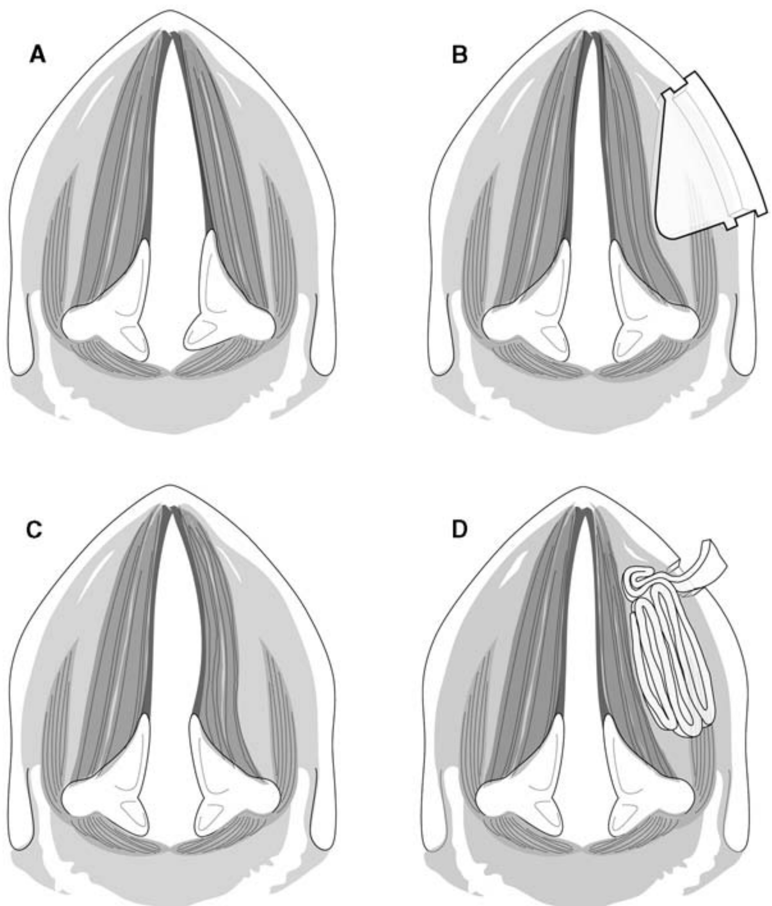

Fig 40–3. Two prototypical approaches to window placement are shown. This figure illustrates the use of a prosthesis such as the Montgomery or some hand-carved Silastic patterns. Correction of the glottic insufficiency (**A**) includes some medialization of the vocal process when the shim is placed (**B**). The use of Gore-Tex is represented in a similar sequence (**C**, **D**) demonstrating correction of vocal fold bowing.

windows, regardless of the type of implant or formula one chooses to follow. The most critical landmark is a horizontally oriented line extending from the midway point of the thyroid cartilage ventrally and extending dorsally parallel to the inferior plane of the cartilage. We first measure the distance from the thyroid notch to the inferior margin (approximately 20 mm in males and 15 mm in females). The site of the anterior commissure is estimated to be half this distance and there is often a small depression in the cartilage at this point. The plane of the superior edge of the vocal fold corresponds to the horizontal line projected posteriorly from this point (Fig 40–4). When landmarks are obscured by surgery or trauma it is easy to confirm this level by inserting a needle and observing intraluminal placement with the endoscope[19] or using a lacrimal probe through a small pilot hole.[20] For the typical Silastic carved or preformed prosthesis, a rectangular window is marked out on the surface of the thyroid cartilage such that the superior edge is at the projected line of the vocal fold (Fig 40–4). It is important to keep the window as inferior as possible while preserving at least 3 mm of cartilage along the inferior rim for support. The anterior edge should be 6 to 10 mm posterior to the ante-

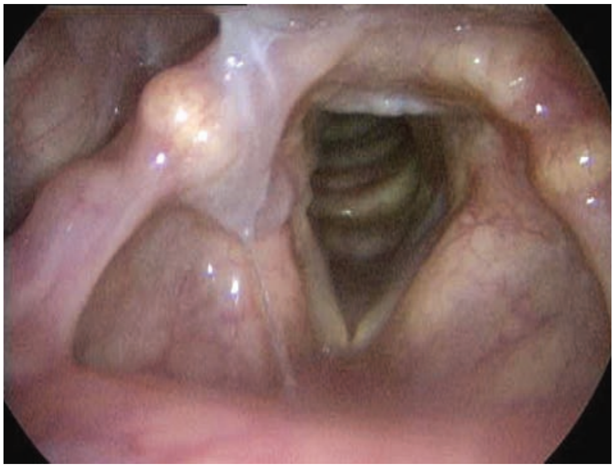

Fig 43–2. Bacterial mucopus from sinobronchial sources draping into the larynx.

Fig 43–3. Bacterial superinfection on chronic laryngitis sicca.

therapeutic vocal fold injection, and other laryngeal trauma.[12,43]

Chronic laryngitis sicca with superinfection is another challenging problem to treat. These patients tend more often to be elderly with multiple medical problems, and consequently multiple medications that may result in mucosal drying. As a result, epithelial defenses are compromised and low-grade bacterial superinfection may take place, particularly in association with stagnant secretions overlying the dry mucosa (Fig 43–3). The normal function of the larynx with mucus lubrication, mucosal protection, and the rate of airflow traversing the larynx is compromised in such individuals. Consequently, the conditions are more favorable for the creation of biofilms, which may explain why these patients are more often refractory to treatment.

Common and uncommon bacterial pathogens causing laryngitis are listed in Table 43–2. The following discussion focuses on acute epiglottitis/supraglottitis which still may be encountered as a life-threatening entity, as well as a synopsis of other uncommon bacterial infections of the larynx.

Table 43–2. Bacterial Infections in the Larynx

Common pathogens	*Streptococcus pneumonia* *Streptococcus pyogenes* *Haemophilus influenzae* *Klebsiella pneumoniae* *Neisseria gonorrhoeae* *Branhamella catarrhalis* Fusobacterium and other anerobes *Staphylococcus aureus*
Less common pathogens	*Corynebacterium diphtheriae* *Francisella tularensis* *Mycobacterium tuberculosis* *Mycobacterium leprae (leprosy)* *Treponema pallidum (syphilis)* Actinomycoses *Salmonella typhii* *Proteus spp.* *Pseudomonas aeruginosa* *Klebsiella rhinoscleromatis* *Pseudomonas mallei* *Nocardia*

Acute Epiglottitis

Epiglottitis has been traditionally felt to be a sequel to infection of Waldeyer's ring or to a traumatic event, supported by case reports that describe a history of preceding pharyngitis or upper respiratory symptoms.[44] The organism most commonly associated with epiglottitis is *Haemophilus influ-*

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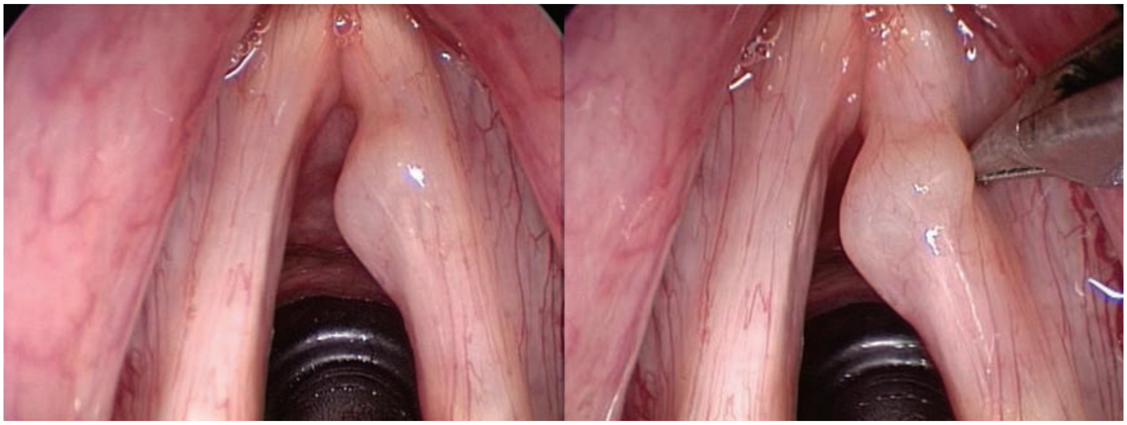

Fig 13–8. Gentle pressure on the lateral aspect of the vocal fold can evert the vocal fold and expose the caudal portion of the lesion to view.

endeavor is that, although primarily concerned with restoration of mucosal pliability, it is carried out on the static vocal fold, without any means of assessing vibratory function intraoperatively. A new instrument to accomplish this task has been proposed,[36] but its practical utility remains to be determined.

Subepithelial Injection

Kass et al[37] have formally described the useful technique of subepithelial injection into the vocal fold. Such an injection facilitates subepithelial dissection by expanding tissue planes, and, when a hemostatic agent like epinephrine is used, by aiding in hemostasis. It also serves to better delineate pathology in cases where the lamina propria is obliterated, as in scar sulcus, and tumor invasion. Infiltration is easily performed with specially designed needles, or by means of a trimmed, 25-gauge or smaller butterfly needle.

Surgical Approach

The surgical approach in a given case is determined by the relation of the lesion to the layered vocal fold microanatomy, and informed by the appearance of the lesion at stroboscopy. The surgeon should always be guided by the principal of maximal preservation of normal tissue, and its corollary of minimal handling and disruption of uninvolved tissue. There is certainly no place for the removal of uninvolved epithelium as "margins" in nonmalignant lesions of the vocal fold. In certain circumstances, for example, when there are mild to moderate reactive changes contralateral to the primary lesion, there is a good case to be made for leaving small irregularities undisturbed.

Most benign lesions of the vocal fold arise within the superficial lamina propria. Microflap surgery, the dominant surgical paradigm in endolaryngeal microsurgery, offers subepithelial access for removal of these lesions and preserves overlying tissue. Since the widespread adoption of microflap approaches, it has become common to speak of the superficial lamina propria as the natural plane of dissection in microlaryngoscopic surgery. Unfortunately, this has created the impression that disruption of this layer is somehow free of consequences. In fact, the contrary is probably true; it is the single most important layer for phonatory vibration. Its loose structure causes it to cleave readily, a tendency that offers the surgeon a path of low resistance. Preventing undue separation of this layer beyond the minimal requirements for lesion excision is a challenge of endolaryngeal microsurgery. Epithelial incisions, once made quite laterally to avoid trauma to the epithelium of the vibratory margin,[38,39] have tended to be placed closer to the lesion to minimize the amount of superficial lamina propria which must be disturbed to reach the pathology.[40]

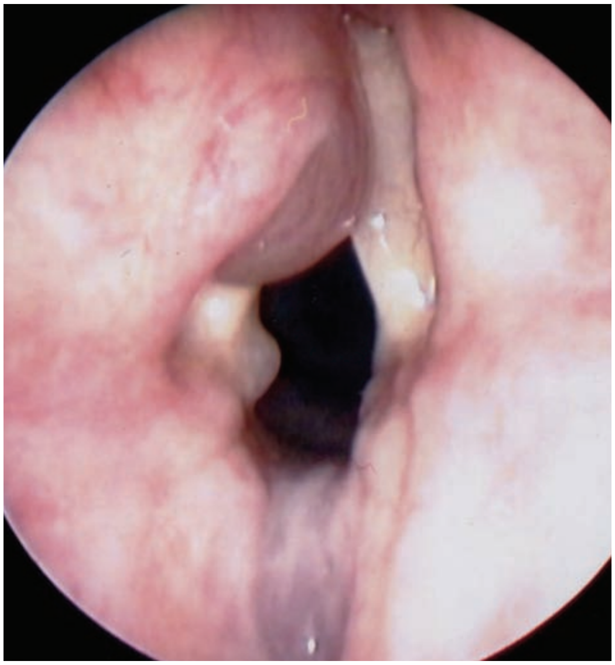

Fig 18–10. Saccular cyst.

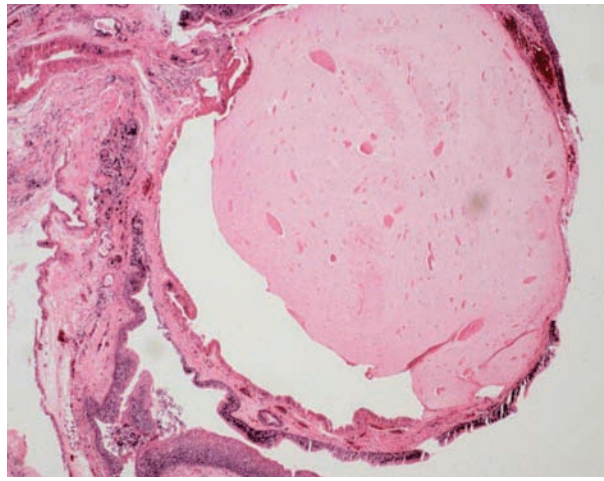

Fig 18–11. Epiglottic cyst, the result of cystic dilation of a duct.

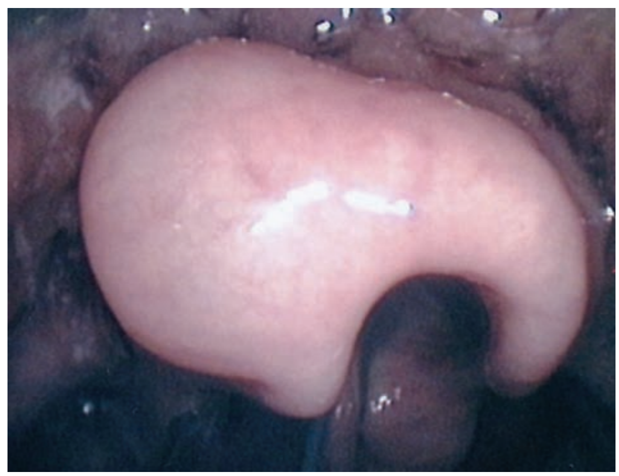

Fig 18–12. A ductal cyst.

lateral, and herniates through the thyrohyoid membrane. The differential diagnosis of saccular cysts may also include a branchial cleft cyst. The anatomic location of the duct or tract will also aid in the distinction. The tract of a branchial cleft cyst will not lead through the thyrohyoid membrane, but will continue superiorly along the anterior border of the sternocleidomastoid muscle and may end at the angle of the mandible or in the tonsillar bed.

Ductal Cysts

Squamous, "tonsillar" and oncocytic laryngeal cysts can be the result of blockage of a minor salivary gland duct. As mentioned, this is the most commonly encountered type of laryngeal cyst. The cyst lining is actually the dilated ductal epithelium; it may be squamous, oncocytic (see subsequent discussion on salivary lesions) or squamous with surrounding lymphoid stroma—referred to as "tonsillar" cysts (Fig 18–11). Squamous cysts and oncocytic cysts have a predisposition for the ventricular bands, ventricle, aryepiglottic folds, and epiglottis. Tonsillar cysts have a predisposition for the vallecula—an area with tonsillar remnants. Simple conservative excision is curative.

Other Laryngeal Cysts and Sinuses

Epidermal inclusion cysts, dermoid cysts, and branchial cleft cysts may occur in the endolarynx. An epidermoid cyst, a keratin-filled cyst lined by stratified squamous mucosa, may be the result of a traumatic mucosal inclusion, or a congenital rest. Rarer still are dermoid cysts, which contain skin adnexal structures and are purely mature benign growths

Treatment and Prognosis

Conservative endoscopic removal will be curative for most cases. GCT have a very low rate (8%) of recurrence, even after incomplete excision. Recurrent tumors or frankly malignant tumors require resection with free margins. Twelve of 20 patients reported in the literature with metastatic malignant GCT ultimately died of disease.[65] We have seen a large (4.7-cm) hypopharyngeal GCT in a 29-year-old woman, which was ultimately fatal after locoregional metastasis. We have also seen a recurrent, non-metastazing laryngeal GCT, with atypical features (nuclear pleomorphism, spindling of cells, Pagetoid spread into overlying mucosa), that we classified as an atypical GCT.[66] Chiang et al[67] recently reported a patient with malignant laryngeal GCT and lung metastases.

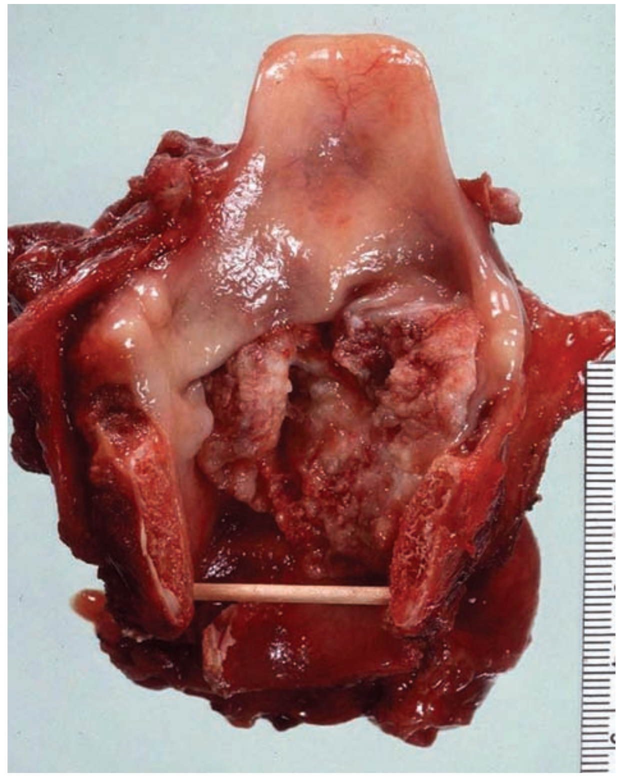

Fig 18–17. Transglottic squamous carcinoma.

Squamous Cell Carcinoma

Clinical Features

The exact laryngeal site for a tumor may determine or influence (1) the type of presenting symptoms, (2) stage at presentation, (3) surgical options, and (4) prognosis. Glottic tumors present with changes in voice quality, that is, hoarseness; patients tend to seek medical care when these tumors are relatively small. Large glottic tumors or bilateral glottic tumors may present with worsening upper airway obstruction and stridor (Fig 18–17). Supraglottic tumors may be larger than glottic tumors before becoming symptomatic (Fig 18–18). Epiglottic tumors may cause a change in vocal quality (a muffled or "hot potato voice"). Tumors at the base of the epiglottis may be asymptomatic and escape visualization at indirect laryngoscopy ("winklekarzinom" or cancer in the corner"). Primary ventricular carcinomas are rare, and the majority of tumors encountered here result from the direct spread of glottic primaries. Primary ventricular carcinomas are noteworthy in that they remain hidden from the observer on laryngeal examination, merely forming a bulge under the intact vestibular fold mucosa. Most "infraglottic" tumors actually arise from the undersurface of the vocal fold; they are considered and staged as glottic

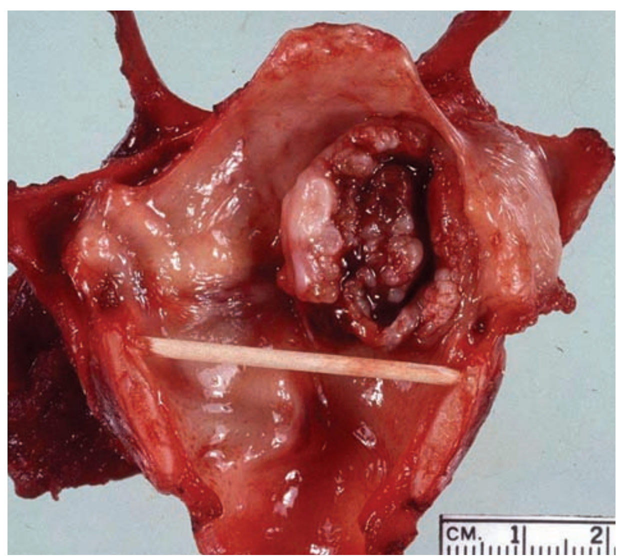

Fig 18–18. Supraglottic squamous carcinoma.

tumors. Infraglottic carcinomas may also present with hoarseness. Direct microlaryngoscopy may reveal their presence only after the vocal cords are

Table 19–2. Primary Tumor Categories (T)

TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
Supraglottis	
T1	Tumor limited to one site of the supraglottis with normal vocal cord mobility
T2	Tumor invades mucosa of more than one adjacent subsite of the supraglottis or glottis or region outside the supraglottis (eg, mucosa of base of tongue, vallecula, medial wall of pyriform sinus) without fixation of the larynx
T3	Tumor limited to larynx with vocal cord fixation and/or invades any of the following: postcricoid area, preepiglottic tissues, paraglottic space, and/or minor thyroid cartilage erosion (eg. Inner cortex)
T4a	Tumor invades through the thyroid cartilage and/or invades tissues beyond the larynx (eg. trachea, soft tissues of neck including deep extrinsic muscle of the tongue, strap muscles, thyroid, or esophagus)
T4b	Tumor invades prevertebral space, encases carotid artery or invades mediastinal structures
Glottis	
T1	Tumor limited to the vocal cord(s), which may involve the anterior or posterior commissures, with normal vocal cord mobility
	T1a Tumor limited to one vocal cord
	T1b Tumor involves both vocal cords
T2	Tumor extends to the supraglottis and/or subglottis or with impaired vocal cord mobility
T3	Tumor limited to the larynx with vocal cord fixation
T4a	Tumor invades cricoid or thyroid cartilage and/or invades tissues beyond the larynx (eg. trachea, soft tissues of neck including deep extrinsic muscle of the tongue, strap muscles, thyroid, or esophagus)
T4b	Tumor invades prevertebral space, encases carotid artery or invades mediastinal structures
Subglottis	
T1	Tumor limited to the subglottis
T2	Tumor extends to the vocal cord(s) with normal or impaired vocal cord mobility
T3	Tumor limited to the larynx with vocal cord fixation
T4a	Tumor invades cricoid or thyroid cartilage and/or invades tissues beyond the larynx (eg. trachea, soft tissues of neck including deep extrinsic muscle of the tongue, strap muscles, thyroid, or esophagus)
T4b	Tumor invades prevertebral space, encases carotid artery or involves mediastinal structures

histopathologic grade (G), using the Broder's classification (Table 19–5),[74] be recorded. Other tumors of the head and neck may originate from tissues of glandular epithelium, odontogenic, lymphoid, various soft tissue, or bone and cartilage origin. Only laryngeal tumors of squamous cell origin are included in the AJCC TNM cancer staging system. Because tumors of squamous cell origin always display histopathologic differentiation, grades of G1, G2, or G3 are always used. Tumors containing areas of undifferentiation adjacent to areas of squamous differentiation are classified as poorly differentiated.

Table 19–3. Regional Lymph Node Categories

NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single ipsilateral lymph node 3 cm or less in greatest dimension
N2	Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension; or in bilateral or contralateral lymph nodes none more than 6cm in greatest dimension
N2a	Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension
N2b	Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension
N2c	Metastasis in bilateral or contralateral lymph nodes none more than 6 cm in greatest dimension
N3	Metastasis in a lymph node more than 6 cm in greatest dimension

Table 19–4. Distant Metastasis Categories (M)

MX	Presence of distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Table 19–5. Histopathologic Grade (G)

GX	Grade cannot be assessed
G1	Well differentiated
G2	Moderately differentiated
G3	Poorly differentiated
G4	Undifferentiated

Optional Descriptors. Table 19–6 shows optional descriptors but no specific recommendation for their recording is made by the AJCC or UICC.

Table 19–6. Optional Descriptors

Lymphatic Invasion (L)	
LX	Lymphatic invasion cannot be assessed
L0	No lymphatic invasion
L1	Lymphatic invasion
Venous Invasion (V)*	
VX	Venous invasion cannot be assessed
V0	No venous invasion
V1	Microscopic venous invasion
V2	Macroscopic venous invasion
Residual Tumor (R) Classification	
The absence or presence of residual tumor after treatment is described by the symbol R.	
RX	Presence of residual tumor cannot be assessed
R0	No residual tumor
R2	Microscopic residual tumor
R3	Macroscopic residual tumor

Note: Macroscopic involvement of the wall of veins (with no tumor within the veins) is classified as V2.

TNM System for Unified Stage Groupings

For each cancer, the individual T, N, and M category ratings are combined in tandem to form expressions, such as T2N1M0 or T3N2M1. Because 6 categories of T, 4 categories of N, and 2 categories of M create 48 possible combinations for the TNM expressions, stage groupings (*I*, *II*, *III*, and *IV*) are created to ease statistical analyses (Table 19–7).[1,75] The various combinations were selected based on the observation that patients with localized tumors had higher survival rates than patients with widespread tumors.

When there is no nodal involvement, the stage is determined by the extent of primary tumor. Thus, T1 = Stage I; T2 = Stage II; T3 = Stage III; T4a = Stage IVA, and T4b = Stage IVB. With nodal spread, stage is essentially determined by extent of nodal involvement. Thus, N1 is classified as Stage III for T1-3 and Stage IVA when T = 4a. When N is greater than N_2 and M = 0 then stage is Stage IVB. If M = 1, then stage is Stage IVC.

from research at Washington University to demonstrate how the TNM system could be expanded with the inclusion of symptom severity and prognostic comorbidity. These 3 variables are combined, using conjunctive consolidation, to create the CS Staging System.

The goal of the CS staging system project was twofold: (1) to demonstrate the prognostic importance of symptom severity and comorbidity and (2) to demonstrate that a composite CS staging, created by the addition of symptom severity and comorbidity to the TNM system, could substantially improve the prognostic precision of laryngeal cancer staging. The 1st step in the creation of the CS system was the creation of a functional severity (FS) system. The term functional severity is used for the conjunction of symptom severity and comorbidity as this represents the functional aspects of the cancer. As seen in Table 19–16, the conjoined impact of symptom severity and comorbidity is shown. Within each category of symptom stage, the survival rates were substantially lowered when prognostic comorbidity was present. For example, patients with local (Stage 1) symptoms had a 76% (181/237) 5-year survival rate without prognostic comorbidity, but this was reduced to 41% (9/22) when prognostic comorbidity was present. Because both symptom severity and prognostic comorbidity were independently important, categories of each variable were combined using the conjunctive consolidation techniques described above. Thus the 8 groups generated by the conjunction between symptom stage and prognostic comorbidity, were *consolidated* into 3 composite, FS stages, labeled as *alpha, beta,* and *gamma*.

Next, the prognostic impact of FS was examined within each TNM stage. As shown in Table 19–17, within each vertical column of TNM anatomic stage, the FS staging system defined important and consistent prognostic gradients. The 5-year survival rates in TNM Stage I ranged from 81% to 40%, 77% to 12% in TNM Stage II, 59% to 38% in TNM Stage III, and from 56% to 8% in TNM Stage IV based on FS Stage.

These results demonstrate the profound prognostic heterogeneity that can exist among patients who are in the same TNM stage. The 12 categories created by the conjunction of FS and TNM stage

Table 19–16. Five-Year Survival Rates in Conjunction of Symptom Severity and Prognostic Comorbidity Stages

Symptom Stage	Prognostic Comorbidity Stage				Total
		Absent		Present	
1	Alpha	181/237 (76%)	Gamma	9/22 (41%)	190/259 (73%)
2		53/73 (73%)		2/8 (25%)	55/81 (68%)
3	Beta	96/173 (55%)		8/33 (24%)	104/206 (50%)
4		21/53 (40%)		1/10 (10%)	22/63 (35%)
Total		351/536 (65%)		20/76 (27%)	371/609 (61%)

Symptom stage: 1 = local, 2 = peri-local, 3 = systemic, and 4 = distant.

Alpha, Beta, and Gamma refer to the names of the three categories of the new composite FS Staging System.

hemilaryngectomy or SCPL alter the sphincteric function of the larynx and are therefore considered to be high-risk procedures in elderly patients. Conservation surgery has been performed for malignant and benign laryngeal tumors in children[32,33] for whom the same oncologic and functional principles apply.

The patient's use of voice professionally, and in general, must be evaluated preoperatively. Neolaryngeal voices (with 1 or both vocal folds resected) are not *normal* voices. The degree of dysphonia that is *acceptable* to the patient must be determined preoperatively. The degree of dysphonia is not always predictable and can vary from patient to patient, even with the same surgical technique.

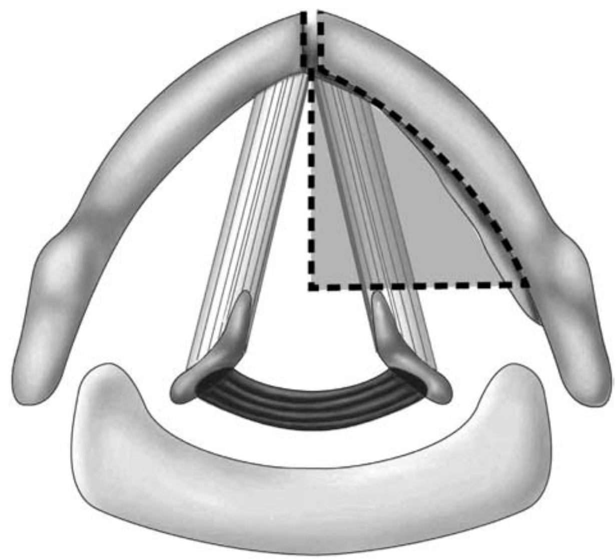

Fig 24–3. External cordectomy, superior view. Reprinted with permission from Amplifon, France.

REVIEW OF SURGICAL TECHNIQUES FOR GLOTTIC CANCER

Vertical Partial Laryngectomies

The vertical partial laryngectomies are referred to as such because tumor resection is carried out through a vertical thyrotomy, with or without resection of part of the thyroid cartilage. These techniques include cordectomies, frontolateral laryngectomies, hemilaryngectomies, and frontal anterior laryngectomies.

Laryngofissure with Cordectomy

Principles and Indications. One of the earliest techniques (1st described in the early 19th century), the external cordectomy involves removal of 1 vocal fold via an anterior midline thyrotomy, with preservation of the laryngeal framework (Figs 24–3 and 24–4). It is indicated for T1a carcinoma, limited to the middle 3rd of the vocal fold with normal laryngeal mobility and without extension to the anterior commissure. This external approach for cordectomy has largely been supplanted by transoral laser cordectomy, the oncologic results being the same for both therapeutic options for tumors classified as T1a.[34–36] Cordectomies via thyrotomy are now reserved for surgical indications when microlaryngoscopic exposure of the larynx is impossible due to the patient's anatomic configuration. Radiation therapy can also be indicated for T1a glottic carcinoma, with excellent local control.[37–39] Radiation therapy is thought to result in better voice quality. Surgery performed for recurrence or a 2nd primary tumor after initial radiation therapy has decreased local control rates.[40]

Technique. Under general anesthesia with orotracheal intubation, a limited apron incision is made at the lower aspect of the neck, to avoid aligning the skin incision with the thyrotomy and to provide adequate exposure for the section of the thyroid isthmus and exposure of the trachea if tracheotomy is deemed necessary during the procedure or in the postoperative course. The skin flap is elevated up to the upper border of the thyroid cartilage and the strap muscles divided at the midline. The prelaryngeal tissue (level VI or the Delphian node) is resected completely, exposing the cricothyroid membrane, and systematically sent for pathologic examination. The cricothyroid membrane is incised vertically on the midline, taking care not to perforate the cuff of

Since than many different silicone-made prostheses have been developed (Table 27–1). The non-self-retaining prostheses (Bivona, Blom-Singer Duckbill and low-pressure devices, Herrmann) are designed for secondary placement some time following laryngectomy. The patient should be able to remove and replace the device for maintenance. The disadvantages of these non-self-retaining devices are the attachment of the prosthesis to the skin with glue, regular removal for maintenance, reinsertion problems with spontaneous closure of the fistula, irritation of the tracheoesophageal shunt, extrusion of the prosthesis, and shunt migration.[53] The self-retaining prostheses (Blom-Singer indwelling, Groningen, Nijdam, Provox, Traissac, Voice Master) need daily maintenance without removal. During voice

Table 27–1. Overview of Different Types of Tracheoesophageal Voice Prostheses with (non) Self-Retaining Capacities and Their Specific Method of Insertion (anterograde, retrograde, or bidirectional).

Voice Prostheses	Types	Insertion	Self-Retaining
Algaba		Anterograde	Yes
Bivona	Duckbill	Anterograde	No
	Low resistance	Anterograde	No
	Ultralow resistanc	Anterograde	No
	Bivona-Colorado	Anterograde	No
Blom-Singer	Duckbill	Anterograde	No
	Low pressure	Anterograde	No
	Indwelling low pressure	Anterograde	Yes
	Advantage indwelling	Anterograde	Yes
Bonelli valve		Anterograde	Yes
Groningen	Standard button	Bidirectional	Yes
	Low resistance	Bidirectional	Yes
	Ultralow resistance	Bidirectional	Yes
Henley-Cohn		Anterograde	Yes
Herrmann ESKA		Anterograde	No
Nijdam	Valveless	Retrograde	Yes
Panje voice button		Retrograde	Yes
Provox	Provox® Type I	Retrograde	Yes
	Provox® Type 2	Bidirectional	Yes
	Provox® ActiValve	Bidirectional	Yes
Staffieri		Retrograde	Yes
Mozolewski	Supratracheal	Anterograde	No
Traissac		Retrograde	Yes
Voicemaster	Primo	Anterograde	Yes
	Standard	Anterograde	Yes

4, 1998, a team led by the senior author performed a total laryngeal transplantation in a man who had sustained severe laryngeal trauma in a motor vehicle accident.[8]

THE FIRST SUCCESSFUL COMPOSITE HUMAN LARYNGEAL TRANSPLANT

The recipient was a 40-year-old man who had suffered a crush injury to his larynx and pharynx during a motorcycle accident 20 years earlier. Despite multiple attempts at another institution to reconstruct his larynx, he remained aphonic and tracheotomy dependent. The patient underwent extensive pretransplant counseling including psychiatric evaluation, speech pathology testing, and 4 interviews with members of the surgical team. All the people involved agreed that the patient understood the risks and his motivation was appropriate. The procedure was approved by the Institutional Review Board of the Cleveland Clinic Foundation. After a 6-month search, a 40-year-old man who was brain dead from a ruptured cerebral aneurysm was identified as a suitable donor. He met all the predetermined criteria for acceptance in regard to HLA matching (4 of 5) and serum virology.

During the donor organ harvest, the entire pharyngolaryngeal complex, including 6 tracheal rings and the thyroid and parathyroid glands was removed (Fig 40–1). The organ complex was stored in University of Wisconsin solution during transport until revascularization 10 hours later. Prior to surgery, the recipient patient received cyclosporine, azathioprine, and methylprednisolone. After surgical exposure of the patient's severely deformed laryngeal structures but prior to their removal, perfusion to the donor organ was re-established. The donor's right superior thyroid artery was anastomosed to that of the patient, whereas the proximal end of the donor's right internal jugular vein was

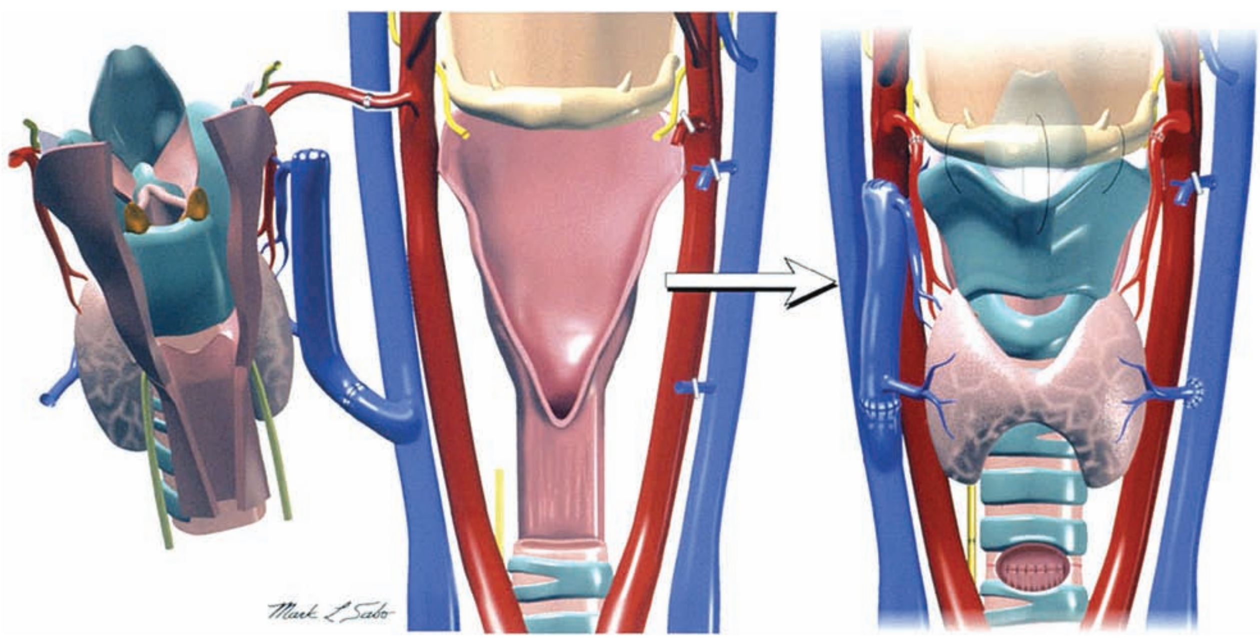

Fig 40–1. The 1998 surgical technique of the first successful composite laryngeal transplantation. Anastomoses included the donor right internal jugular vein to recipient right facial vein, donor superior thyroid arteries to recipient superior thyroid arteries, and donor left middle thyroid vein to recipient left internal jugular vein. Note that both superior laryngeal nerves were anastomosed, whereas only the patient's right recurrent laryngeal nerve could be located for anastomosis to the donor organ's right recurrent laryngeal nerve.