SURGICAL MANAGEMENT OF SLEEP APNEA AND SNORING

EDITED BY
DAVID J. TERRIS AND RICHARD L. GOODE

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Preface

The young field of sleep-disordered breathing continues to mature, with regard to both diagnostic accuracy and treatment alternatives. Besides the nonsurgical management having evolved considerably over the past decade, procedural options have also changed, justifying a fresh and comprehensive review of this surgical discipline.

Leading practitioners of surgical treatment of sleep-disordered breathing have spent a considerable amount of time in detailing thought-provoking and educative coverage of a multitude of time-honored and new procedures in this text, with emphasis on the latest path-breaking discoveries and glimpses into what the future may hold. The atlas-like figures will make this a helpful reference for anyone embarking upon novel procedures. The expert review of the diagnostic tools and nonsurgical treatment will serve to fortify the knowledge base requisite for practitioners managing patients with sleep disorders. Finally, the thought-provoking chapters on evidence-based medicine and the ''ideal procedure'' should stimulate individuals to join the growing army of investigators who together will answer many of the remaining questions in this field.

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Upper Airway Surgical Anatomy

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1. INTRODUCTION

Obstructive sleep disordered breathing (OSDB) results from anatomical upper airway abnormalities and changes in neural activation mechanisms intrinsic to sleep causing hypotonia of the pharyngeal dilator muscles (1). Surgical correction of the obstruction requires accurate identification of the obstructive process and a thorough knowledge of the anatomy and function of the site to allow the surgeon to create a physiologic airway when medical therapy has failed. The purpose of this chapter is to discuss the anatomy of these structures as they relate to surgical treatment of upper airway obstruction during sleep. As our knowledge of the anatomic and physiologic functions of the upper airway expands, so will the treatment options to produce minimally invasive procedures to obtain functional and long-lasting results.

2. NASAL AIRWAY

The primary function of the nasal passages is to work as a resistor. By matching impedance of the upper and lower airways, the nasal passages control breathing frequency and expiratory length (2). The pertinent parts of nasal airway anatomy are the nasal valves, lateral nasal walls, nasal septum, and nasal mucosa.

The nasal valve is the narrowest portion of the nasal passage and is composed of an internal and an external portion (Fig. 1). The *internal valve* is the primary nasal airflow regulator in leptorrhine (Caucasian) noses (3). In contrast, the inferior turbinates are the primary nasal airflow regulators in platyrrhine (Asian and African) noses (3). The internal nasal valve is the area between the caudal end of the upper lateral cartilage and the septal cartilage. The posteroinferior limit is the anterior part of the inferior turbinate and the soft tissue at the pyriform aperture. The angle between the caudal end of the upper lateral cartilage and the septal cartilage is the valve angle. This angle is normally 10–15° in the leptorrhine nose and is more obtuse in platyrrhine noses. The *external valve* comprises the mobile alar wall and cutaneous skeletal support (Fig. 1). It is described as the region caudal to the internal valve,

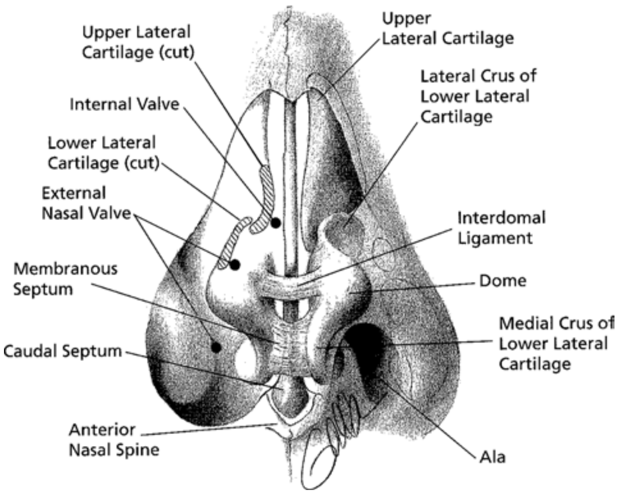

Figure 1 Internal and external nasal valve. (Modified from Ref. 3.)

bounded laterally by the nasal alae containing fibro-fatty tissue and bony pyriform aperture, medially by the septum and columella, and inferiorly by the nasal floor. Both internal and external valves function as Starling resistors. The transmural pressure increases at these narrow sites according to the Bernoulli principle leading to collapse and a decrease in airflow. Weak valves deform at low transmural pressure and lead to premature collapse and airway obstruction.

The lateral nasal wall contains the inferior, middle, superior, and, if present, a supreme turbinate (Fig. 2). These tissue ridges are composed of scrolls of bone covered with a thick mucous membrane. The bony portions of the middle and superior turbinates are extensions of the ethmoid bone. The inferior turbinate is an independent bone which articulates with the nasal surface of the maxilla, the perpendicular plate of the palatine bone, and the ethmoid and lacrimal bones. The main bulk of the inferior turbinate is the lamina propria, which is built of loose connective tissue and superficially harbors an inflammatory cell infiltrate (Fig. 3). The medial mucosal layer (MML) is thicker than the bone and the lateral mucosa. There are more glands and goblet cells in the lateral mucosa layer (LML). The extent of venous sinusoids varies significantly, with the greatest difference in the inferior mucosa layer (IML). Decreased proportion of glands and an increase in venous sinusoids are associated with advanced age. The cancellous central bony layer is made of interwoven trabeculae and houses the major arterial supply of the turbinate. Both MML and IML are ideal targets for surgical reduction in cases of obstruction caused by turbinate dysfunction. The superior turbinate has a thinner and less vascular mucosa and is the only turbinate to contain olfactory epithelium, housing the olfactory nerve sensory cells in its mucous membrane. Turbinate enlargement produced by "overstimulation" of the parasympathetic system or "understimulation" by the sympathetic innervation can produce obstruction. Resistance or turbulence produced by the turbinate optimizes air contact with the mucous membrane and produces a sensation of normal airflow. If it is too high or too low, a sensation of obstruction may occur. The "nasal cycle" is a cyclic alteration of constriction and dilation of the inferior turbinate and occurs approximately every 3–6 hr. Abnormalities in cycling, vasoconstrictive medications, infections, allergic disease, and compensatory hypertrophy may

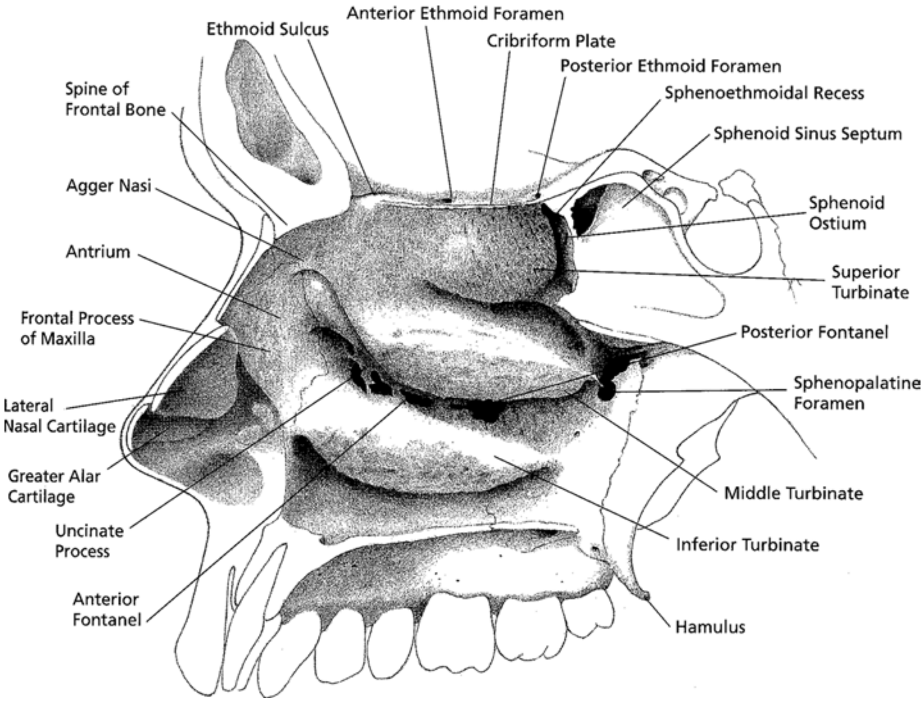

Figure 2 Lateral nasal wall with inferior, middle, and superior turbinates. (Adapted from Ref. 3.)

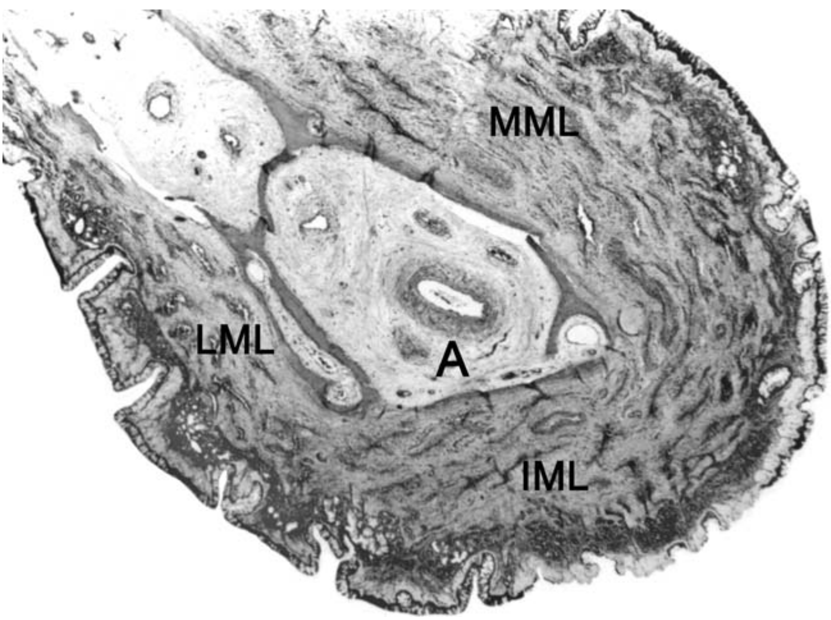

Figure 3 Inferior turbinate, thick MML, vascular IML, thin LML, and central artery (A). (Adapted from Ref. 5.)

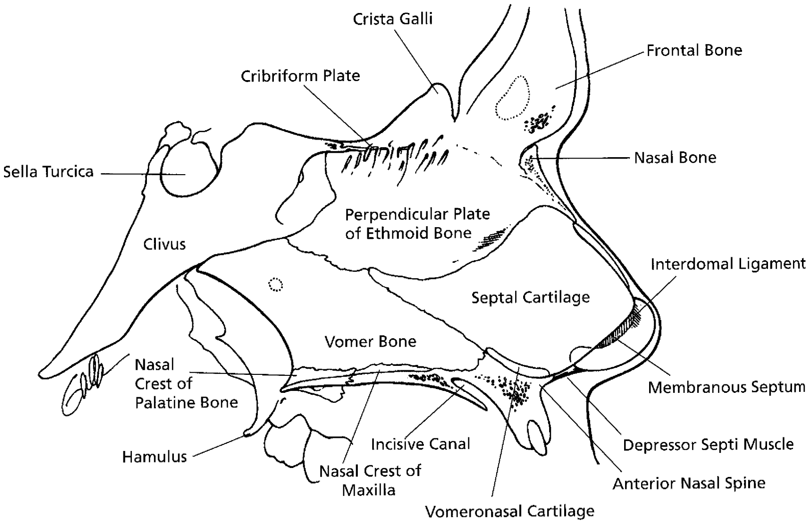

Figure 4 Components of the nasal septum, membranous, cartilaginous, and bony portions made up of the perpendicular plate of the ethmoid and vomer bones. (Adapted from Ref. 3.)

cause anatomic obstruction. Concha bullosa is turbinate enlargement caused by the pneumatization of the turbinate portion of the anterior ethmoid bone. Concha bullosa often refers to aeration of the middle turbinate but can occur in the superior and inferior turbinate and can be a source of nasal airway obstruction.

The nasal septum divides the nasal cavity in the midline and consists of membranous, cartilaginous, and bony portions (Fig. 4). The membranous septum is a skin-covered membrane extending from the columella to the caudal edge of the quadrangular cartilage. The cartilaginous and bony septal portions are covered by perichondrium and periosteum, respectively. A thin mucous membrane covers these layers. The cartilaginous septum is wider at its base, at its junction with the upper lateral cartilages, and at the anterior septal body. The septal body corresponds to a widened area of septal cartilage just anterior to the middle turbinate (1). The major bony portion of the septum is formed by the perpendicular plate of the ethmoid bone (superiorly) and the vomer (inferiorly). The perpendicular plate of the ethmoid makes up a portion of the anterior skull base. Surgical manipulation of this bone during septoplasty can fracture the cribiform plate and cause cerebral spinal fluid leakage. The vomer articulates with the palatine and maxillary bones inferiorly and clivis posteriorly (Fig. 4). The nasal spine of the frontal bone and the nasal crest of the maxillary and palatine bones also provide a small contribution to the bony septum (Fig. 4).

General sensory innervation to the nasal mucosa is primarily by the trigeminal nerve (V_1, V_2) and the greater petrosal branch of the facial nerve. The nasal septum is supplied anteriorly by the nasopalatine nerve (V_2) and the anterior ethmoidal nerve (V_1) and posteroinferiorly by the nasal branches of the greater palatine nerve (V_2). The lateral nasal wall is supplied by the nasal branch of the maxillary nerve (V_2), the greater palatine (V_2), and anterior ethmoidal nerves (V_1).

Blood supply to the nasal cavity is by the internal and external carotid artery systems. The sphenopalatine branch of the internal maxillary artery supplies the posterior nose and can be a significant source of postoperative bleeding in turbinate surgery. This branch exits the pterygopalatine fossa through the sphenopalatine foramen and one to three branches course within the inferior turbinate (Fig. 3). The anterosuperior nose is supplied by the anterior and posterior ethmoid arteries from the internal carotid by the ophthalmic artery. Retraction of these vessels into the orbit during nasal surgery can produce intraorbital hemorrhage. The nasal vestibule is supplied by branches of the facial artery. The septum is supplied by the anterior and posterior ethmoidal arteries, greater palatine artery, sphenopalatine branches, as well as the septal branch of the superior labial artery from the facial artery. These vessels anastomose in the anterior septal cartilage mucosa as Kiesselbach's plexus.

3. ORAL AIRWAY

3.1 Tongue

The tongue is divided into oral tongue (anterior two-thirds) and pharyngeal tongue (posterior one-third) by the sulcus terminalis. This sulcus is a V-shaped groove behind the circumvallate papillae. The apex of the V is posterior and represents the foramen cecum. The oral tongue surface is covered by filiform, fungiform, and circumvallate papillae. Papillae are replaced on the pharyngeal tongue surface by lingual tonsils. The lingual tonsil is the inferior portion of Waldeyer's ring and is made up of discrete lymphoid tissue masses with overlying lenticular papillae (6). Hypertrophy of lingual and palatine tonsils and adenoid tissues is common and often significant. These anatomic obstructions of the oral and nasal airways require careful evaluation during the patients work-up for OSDB.

The tongue muscle consists of intrinsic and extrinsic muscles. The four intrinsic muscles are located entirely within the tongue and are divided by a median fibrous septum fixed to the hyoid bone (Fig. 5). The intrinsic muscles consist of superior

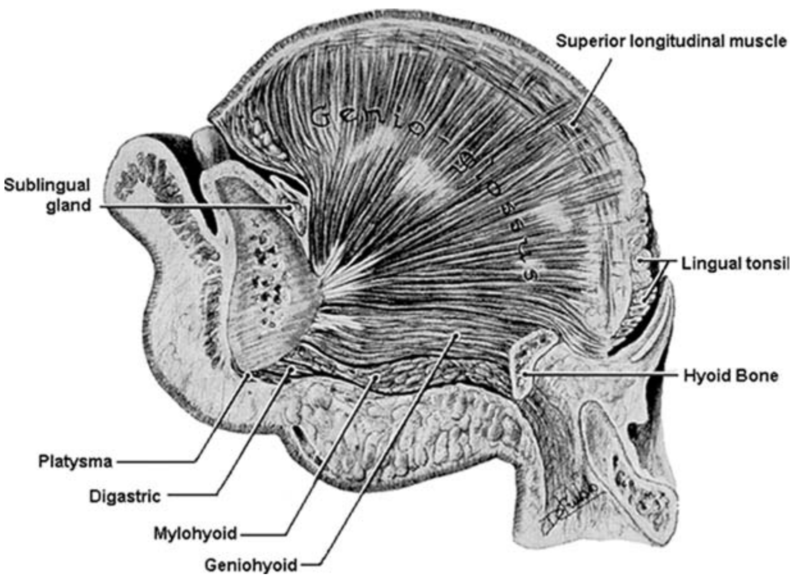

Figure 5 Genioglossus and hyoid bone on median section. (Modified from *Grant's Atlas of Anatomy*, 10th ed. Baltimore, MD: Williams and Wilkins, 1999.)

and inferior longitudinal muscles and, to a lesser extent, the transverse and vertical muscles. The longitudinal muscles modify the shape of the tongue during speaking and swallowing. The transverse muscles narrow and elongate the tongue whereas the vertical muscles flatten and broaden the tongue.

Four extrinsic muscles, the genioglossus, hyoglossus, styloglossus, and palatoglossus, attach outside the tongue (Table 1). The larger genioglossus and hyoglossus extend into the tongue from the sublingual region while the styloglossus and palatoglossus enter the tongue superiorly and laterally (Fig. 6) The genioglossus protrudes and depresses the tongue. It originates on the lingual surface of the anterior mandible at the superior genial tubercle just above the geniohyoid muscle attachment (Figs. 5 and 6). This tubercle can be palpated and serves as an important landmark in tongue advancement surgery for avoiding incisor root damage and preserving muscle attachment. The distance from the genial tubercle superior border to the central root apex can be less than 5 mm (8). The genioglossus opens the retroglossal air space and is considered the most important muscle in keeping the upper airway patent. Contraction is phasic with inspiration; this activity decreases with sleep, becomes almost non-existent during rapid eye movement sleep, ceases in patients with obstructive sleep apnea (OSA) at the onset of an apnea, and increases at the termination of an obstruction (9). Mechanoreceptors control genioglossus activity and are critical in maintaining upper airway patency in patients with OSDB. Overworking of the genioglossus during the day can result in both muscle hypertrophy with airway impingement and loss of muscle contraction (10). The hyoglossus muscle (Fig. 6) arises from the lateral body and greater horn of the hyoid, travels vertically, and passes lateral to the posterior portion of the genioglossus. It courses between the styloglossus laterally and the inferior longitudinal muscle medially to insert into the side of the tongue. It depresses and retracts the tongue. Surgical advancement of the hyoid-hyoglossus complex can advance the tongue base to open the posterior airway space (PAS). The styloglossus extends from the styloid process and ligament to insert on the side and inferior aspect of the tongue (Fig. 6). Its action is to pull the tongue upward and backward. The palatoglossus travels from the palatine aponeurosis inferiorly toward the tonsil and forms the palatoglossal arch (anterior pillar). It inserts into the side of the tongue and is responsible for elevating the posterior tongue (Table 1). Mathur (11) suggests that a similar mechanoreceptor reflex that activates the genioglossus activates the palatoglossus muscle to dilate the pharynx under negative upper airway pressure.

The mylohyoid and digastric muscles are associated with tongue position and movement (Fig. 6). The mylohyoid muscle extends from the mylohyoid line on the mandibular inner surface to the midline where posterior fibers insert onto the hyoid bone and anterior fibers meet contralateral mylohyoid fibers at the median raphe. It elevates the hyoid bone and consequently the floor of the mouth and tongue base. The geniohyoid muscle and tongue musculature lie above the mylohyoid muscle and the anterior belly of the digastric muscle and a portion of the submandibular gland lie below. The digastric muscle consists of anterior and posterior bellies. From its origin on the medial mastoid process, the posterior belly travels toward the hyoid bone. Its fibers transition into a tendon that then pierces the stylohyoid muscle, passes through the hyoid fascial sling, and continues anteriorly as the anterior belly. The digastric muscles, and likely the mylohyoid and geniohyoid muscles, aid in mouth opening. Because these muscles are attached to the mandible and hyoid, they play a role in opening the PAS with mandibular advancement surgery.

Table 1 Extrinsic Tongue Muscles

Muscle	Origin	Insertion	Innervation	Action
Genioglossus*	Upper part of mental spine of mandible	Ventral surface oftongue; anterior surface of body of hyoid bone	CN XII	Draws forward and posterior part protrudes the apex of the tongue
Palatoglossus*	Oral surface of the palatine aponeurosis	Side and dorsum of the tongue	CN XI via the pharyngeal branch of the CN X and pharyngeal plexus.	Elevates posterior part of tongue
Styloglossus	Styloid process and stylohyoid ligament	Side and inferior aspect of tongue	CN XII	Elevation and retraction of the tongue, creating a trough for swallowing
Hyoglossus	Body and greater horn of hyoid bone	Into the inferior side of the tongue	CN XII	Depresses and retracts tongue

Note: The asterisks denote pharyngeal dilators.
Source: From Ref. 27.

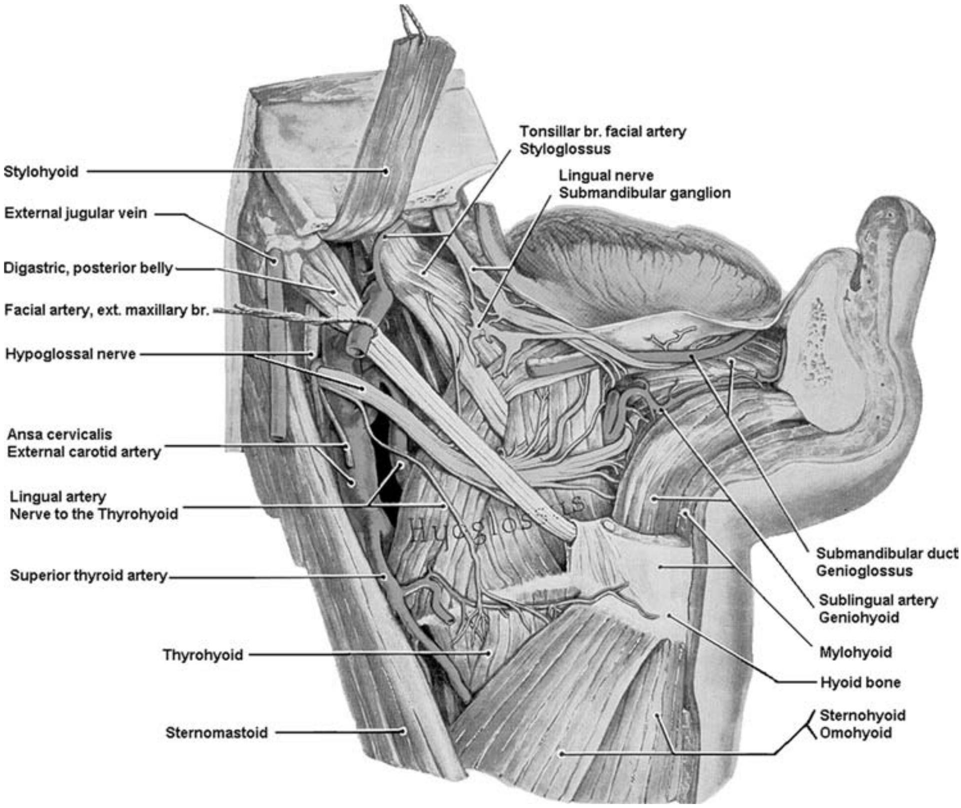

Figure 6 Extrinsic tongue muscles, genioglossus, hyoglossus, styloglossus, and palatoglossus, attached to the outside of the tongue. (Modified from *Grant's Atlas of Anatomy*, 10th ed. Baltimore, MD: Williams and Wilkins, 1999.)

The hyoid bone has no osseous articulation like other species. It is a freely mobile bone that participates in swallowing and phonation (Figs. 5 and 6). In humans it descends in the neck as the oral pharynx elongates. The lengthening of the oral pharynx may be responsible for the laxity at the tongue base that produces obstruction in recumbent sleep (12). This length is measured by the MP-H distance between the mandibular plane (MP) and the hyoid bone (H) on lateral cephalometric films (Fig. 9). The reference range is 11–19 mm. The longer this distance, the higher the possibility of the patient having OSA. The hyoid bone is derived from the second and third arches. The second (hyoid) arch assists in forming the side and front of the neck. From the hyoid cartilage are developed the styloid process, stylohyoid ligament, and lesser cornu of the hyoid bone. The third arch cartilage gives rise to the greater cornu of the hyoid bone. The ventral ends of the second and third arches unite with those of the opposite side to form the body of the hyoid bone and the posterior tongue. The middle constrictor and pharynx attach to the hyoid laterally. During the pharyngeal phase of swallowing, the hyoid (and attached thyroid and cricoid cartilage) is raised by the suprahyoid muscles (mylohyoid, anterior belly digastric, geniohyoid, stylohyoid, hyoglossus, genioglossus, styloglossus, and palatoglossus), which elevate the larynx. The infrahyoid muscles (thyrohyoid, omohyoid, and sternohyoid) depress and stabilize the hyoid bone and with the geniohyoid stabilize and dilate the retroepiglottic laryngopharynx by tensing the hyoepiglottic ligament (9). These muscles are phasic with inspiration and factors that affect hyoid position can adversely affect the muscles and cause narrowing of the laryngopharynx. Surgical immobilization of the hyoid bone during suspension procedures may cause dysphagia (13).

Blood supply to the tongue is primarily from the two lingual arteries that branch from the external carotid at the level of the greater horn of the hyoid bone (Fig. 6). The lingual artery gives off a suprahyoid branch at the posterior edge of the hyoglossus muscle, passes deep to this muscle, and gives two dorsal lingual branches that supply the pharyngeal tongue and palatine tonsil. Near the anterior hyoglossus muscle border, it divides into the deep lingual and sublingual arteries. The deep lingual artery (s. artery ranine) is in the lateral tongue and can be identified and controlled intraoperatively on the lateral surface of the genioglossus if injured during midline tongue reduction or thermal volumetric reduction. The sublingual artery supplies the sublingual gland and tongue musculature.

Motor innervation to both intrinsic and extrinsic tongue musculature, excluding the palatoglossus, is by the hypoglossal nerve (XII) (Fig. 6). The medial branch of the hypoglossal nerve innervates the tongue protruders (genioglossus and geniohyoid). The lateral branch of the hypoglossal nerve innervates the tongue retractors (styloglossus and hyoglossus). Selective electrical stimulation of these nerves can stiffen and dilate the PAS during sleep (14). The lingual nerve (V_3) is a branch of the inferior alveolar nerve and supplies general sensory innervation for touch, pain, and temperature sensation to the oral tongue and lingual gingiva. It also contains post-ganglionic special visceral afferents (taste) via the chorda tympani nerve (VII) from the geniculate ganglion and parasympathetic nerves for the submandibular and sublingual glands. The glossopharyngeal nerve (IX) supplies taste and general sensation to the pharyngeal tongue and vallecula. The internal branch of the superior laryngeal nerve (X) supplies sensory fibers to the base of the tongue and epiglottis. Although the circumvallate papillae are part of the oral tongue, they are innervated by the general sensation and taste fibers of the glossopharyngeal nerve (IX).

Airway assessment of the anatomic tongue-palate position was described in a pre-anesthesia airway assessment by Mallampati et al. (15). Fujita (16) and Friedman (17) have described pre-surgical assessments of the palate and tongue base to predict surgical results. The Mallampati test is performed with the patient in the sitting position, head held neutral, mouth wide open, and tongue protruding to the maximum. Class I is visualization of the soft palate, fauces, uvula, anterior and posterior pillars. Class II is visualization of the soft palate, fauces, and uvula. Class III is visualization of the soft palate and the base of the uvula. Class IV is soft palate that is not visible at all. Dempsey and Woodson have found that clinical airway assessments using Mallampati and Mueller tests did not correlate with apnea–hypopnea index (AHI) and body mass index (BMI) (18). Although surgeons do not have a perfect clinical tool for evaluation of the anatomic site of lesion evaluation, imaging studies, sleep endoscopy, and newer methods of airway assessment and classification are ongoing and will, in time, account for more accurate diagnosis and surgical outcomes.

3.2 Maxilla and Mandible

The bony support of the oral airway is provided by the maxilla and mandible. The maxilla develops from membranous bony plates and has five components: the zygomatic and frontal extensions, the palatine bone, the alveolar processes, and an aerated maxillary sinus. The palatine bones articulate with each other to form the majority of the hard palate. The maxilla articulates with the vomer, lacrimal, sphenoid, and palatine bones. The palate consists of an anterior, bony, hard palate and a posterior, mobile, fibromuscular soft palate. The anterior portion of the hard palate is composed of the maxillary palatine processes and the palatine horizontal plates. Posteriorly, the hard and soft palates are continuous. Posterior to the maxillary central incisor teeth, the incisive canal transmits the nasopalatine nerve and greater palatine artery, which provide sensation and vascularity to the anterior hard palate. The greater palatine foramen is medial to the third molar teeth and transmits the greater palatine nerve and vessels supplying sensation and vascularity to the posterior hard and soft palate. The greater palatine foramen can be used to access the maxillary branch of the trigeminal nerve (V_2) in the pterygopalatine fossa with local anesthesia. The posterior hard palate can be surgically reduced or advanced to mobilize the intact soft palate away from the posterior retropalatal pharynx in palatal advancement surgery for OSDB (19). This method retains the natural anatomy and function of the soft palate and uvula.

The mandible develops from Meckel's cartilage and consists of the horizontal body, the tooth-bearing alveolar portion, and two vertical rami that extend superiorly from the mandibular angle (Fig. 8). The mandibular ramus contains the anterior coronoid process and posterior condylar process, which are separated by the mandibular notch. The genial tubercles, which may be fused in the midline, project from the lingual aspect of the mandibular body as superior and inferior mental spines (Fig. 9). The genioglossus and geniohyoid muscles attach to the superior and inferior spines, respectively. The sublingual fovea lies lateral to each genial tubercle and contains a sublingual gland. Posterior to the sublingual fovea is the attachment of the mylohyoid muscle. Just inferior to the genial tubercle is the attachment of the anterior belly of the digastric muscle in the digastric fossa. The mandibular canal containing the inferior alveolar nerve lies posterosuperior to the mylohyoid line. The lingula (Fig. 9) is a sharp bony edge that overhangs the mandibular canal and serves as the inferior surgical landmark for the medial horizontal cut of the bilateral sagittal split osteotomy (BSSO). The lingula-to-mandibular-notch distance varies from 10.5 to 15 mm (3). The mandibular foramen is 15–20 mm inferior to the mandibular notch. The inferior alveolar nerve is in close association with the mandible for about 4–5 mm above the lingula. The horizontal ramus osteotomy is best placed 7.5 mm below the mandibular notch (3). The inferior alveolar nerve (V_3) descends with the lingual nerve between the medial and lateral pterygoid muscles. Its mylohyoid branch lies in the mylohyoid groove and provides a motor branch to the mylohyoid muscle and anterior belly of the digastric muscle. It then enters the mandibular foramen to innervate mandibular dentition. The inferior alveolar nerve and associated vessels travel through the mandibular canal; it exits through the mental foramen as the mental nerve and vessels. The mental nerve innervates the skin of the lower lip and chin, the lip mucosa, and adjacent gingiva. At the mental foramen, the neurovascular bundle is susceptible to injury during genioglossus muscle advancement procedures. The mental foramen is located on the lateral mandibular body at or near the vertical body midline and between the first and second premolar root apices (Fig. 8). It can be posterior to the second premolar in about 24% of cases or anterior to the first premolar in 1–2% of cases (20).

The masseter and medial pterygoid muscles attach laterally and medially to the ramus and angle and serve as the blood supply to the proximal mandible, which assists in proximal bone survival in BSSO procedures. The blood supply to the distal mandible is by the inferior alveolar artery. It travels through the mandibular canal and supplies dentition, bone, and the surrounding soft tissue. The mandible is also supplemented anteriorly by vessels in the geniohyoid, genioglossus, and digastric (anterior belly) muscles (21). At the level of the first premolar, the inferior alveolar artery branches into the incisive and mental branches. The incisive branch supplies the anterior teeth and supporting structures. The mental branch joins the inferior labial and submental vessels and supplies the chin. Vessels of the temporal mandibular joint capsule and lateral pterygoid muscle supply the condyle. The temporalis muscle inserts on the coronoid process and provides blood supply to this aspect of the ramus.

The *dental relationship* was described by Angle in 1899 as the relationship of the maxillary and mandibular teeth. In class I occlusion, the mesial buccal cusp of the maxillary first molar contacts the mesial buccal groove of the mandibular first molar. In class II occlusion or retrognathia, the mesial buccal cusp is mesial to the groove and in class III occlusion or prognathic occlusion, the cusp is distal to the groove.

The *skeletal relationship* ultimately determines the soft tissue relationships of the airway; it refers to the position of the maxilla and mandible in relation to the skull base and is measured by the lateral cephalometric radiograph (Fig. 7). The SNA (sella, nasion, subspinale) and SNB (sella, nasion, supramentale) angles determine the relative differences and deficiencies in facial growth. SNA is 82° (greater denotes maxillary hyperplasia, and less, maxillary hypoplasia). SNB is 80° (greater denotes prognathism and less, retrognathism). Mandibular retrognathia is associated with a posterior tongue position and pharyngeal airway obstruction. Riley in 1989 suggested that an SNB $< 78\%$ should undergo genioglossus advancement to open the PAS. A long MH distance (MP-H), mentioned earlier, and a narrow PAS measurement are helpful in determining site of lesion and treatment options (22). In a meta-analysis evaluating retrognathic mandibles, narrow PAS, enlarged tongues and soft palates, inferiorly positioned hyoid bones, and retrognathic maxillas, Miles et al. (23) found that only one cephalometric variable, mandibular body length (Go-Gn: gonion-gnathion), demonstrated a clinically significant association in patients with OSA. Dempsey found that the single most important cephalometric variable in predicting AHI severity was the horizontal dimension of the maxilla. A horizontal distance from the porion vertical to the A point (PV-A: porion vertical-supradentale) or the PV-A distance correlated well with the BMI for an increasing amount of the variance in AHI as the severity of AHI increased (Fig. 7) (18). Cephalometric studies are limited to two dimensions; they are inaccurate for cross-sectional or volumetric data and miss soft tissue structures such as the lateral pharyngeal walls or parapharyngeal fat. Schwab and Goldberg (24) describe the usefulness of the CT and MRI scans to provide direct volumetric acquisitions of images of bony support and adipose tissue. Studies by Shepard and Thawley (25) and Trudo et al. (26) have shown the most common site of pharyngeal collapse identified with CT and MRI scans and suggest the retropalatal pharynx as the most common site of obstruction and not retroglossal (25,26). These data suggest that patients undergoing UPPP without scanning would predictably have good results while those with retroglossal obstruction may not show improvement.

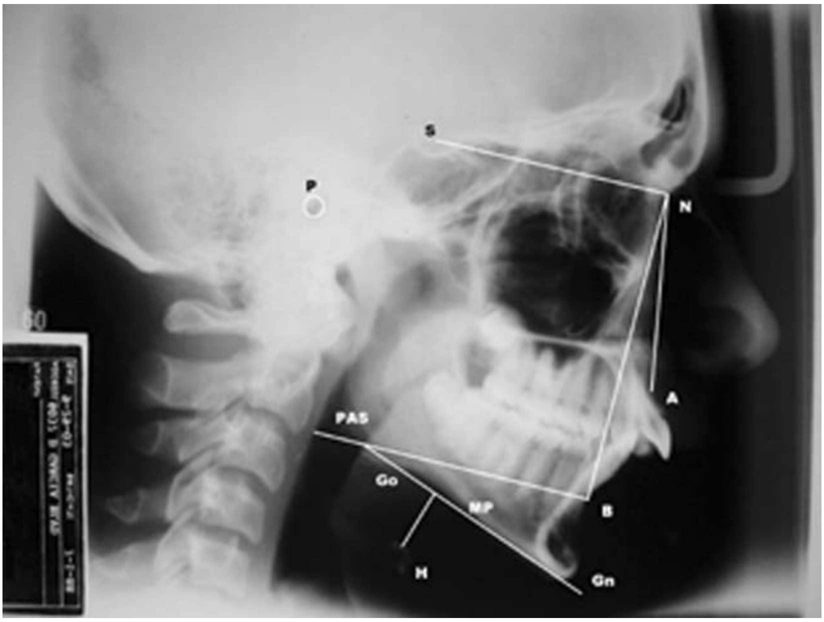

Figure 7 Lateral cephalometric radiograph: SNA, sella-nasion-subspinale (82±2) where *A* is the maximal concavity on the anterior surface of the maxilla; SNB, sella-nasion-supramentale is the mandibular-cranial base angle (80±2), *B* is the maximal concavity on the anterior surface of the mandible; mandibular plane (MP) from the gnathion (Gn, most inferior point on the mandibular symphysis) through the gonion (Go, point of the jaw angle intersection between the ramal and mandibular lines); hyoid bone (H) and MH distance (11–19); PAS, posterior airway space (10–16) between the posterior tongue dorsum and posterior pharyngeal wall on a line between the gonion and the *B* point; P, porion is the central point on the upper margin of the external auditory meatus.

4. PHARYNGEAL AIRWAY

Based on standard anatomic definitions (*Gray's Anatomy*) and others (7,27), the pharyngeal airway is anatomically divided into three regions: the nasopharynx, defined as the area behind the nose and above the soft palate; the oropharynx, defined as the area from the soft palate to the upper border of the epiglottis; and the laryngopharynx, defined as the area from the upper border of the epiglottis to the inferior border of the cricoid cartilage (28). Accurate evaluation of the obstructing airway behind the soft palate and tongue has subdivided the oropharynx into the retropalatal pharynx (or velopharynx) and the retroglossal pharynx, respectively. The obstructing portion behind the epiglottis in the laryngopharynx (or hypopharynx) is referred to as the retroepiglottic pharynx. Consistency in this terminology can accurately localize the anteroposterior site of collapse and will improve data collection, surgical training, and, most importantly, surgical outcome.

Pharyngeal patency during wakefulness is attributable to continuous neuromuscular control, which supervises the activity of the dilator capacity of the pharyngeal musculature. Diaphragmatic contraction creates airflow and subatmospheric intra-airway pressure, which narrows the collapsible segments (9). Dilator muscles

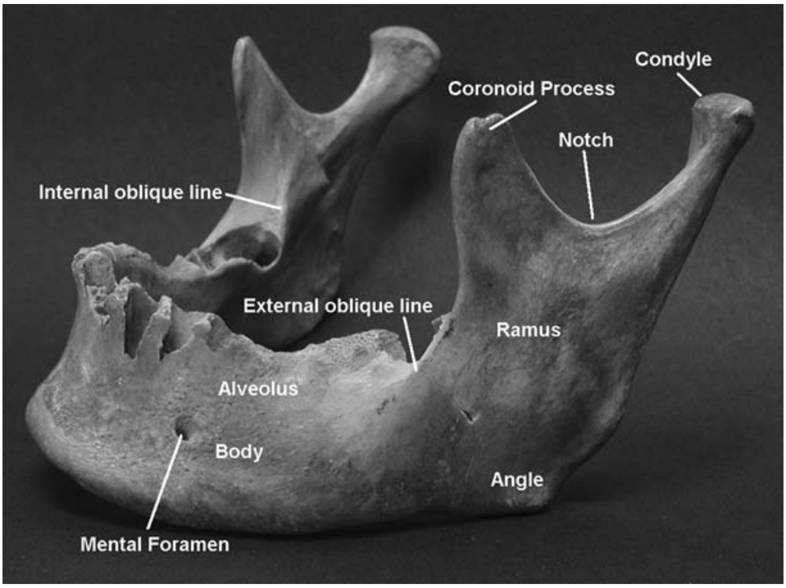

Figure 8 Mandible, lateral aspect of body, alveolus, ramus, condyle, and coronoid process.

stiffen the airway and are discussed later and in Tables 1–3. Pharyngeal patency is a function of the transmural pressure across the pharyngeal wall as well as the compliance of the pharyngeal wall (28). Rowley et al. feel that the intrinsic properties of the airway wall determine retroglossal compliance independent of changes in the neuromuscular activity associated with changes in the sleep state (30). Huang and Williams (31) suggest that rigid tissue is required to maintain airway patency, low tissue

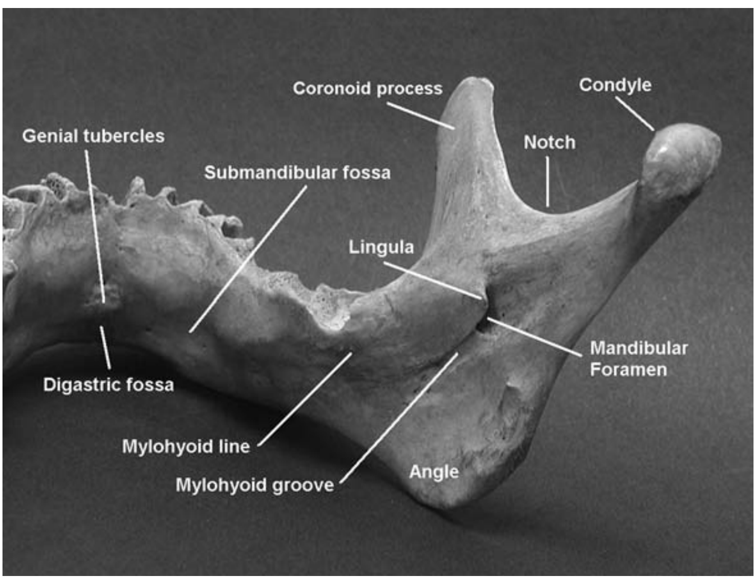

Figure 9 Mandible, lingual aspect, genial tubercles, mylohyoid line, mandibular foramen, and coronoid notch.

Table 2 Muscles of the Palate

Muscle	Origin	Insertion	Innervation	Action
Tensor veli palatini*	Scaphoid fossa of medial pterygoid plate, spine of sphenoid, and auditory tube cartilage	Tendon around hamulus to insert in the palatine aponeurosis	CN V medial pterygoid n. via otic ganglion	Tenses soft palate and opens mouth of auditory tube during swallowing and yawning
Palatoglossus*	Palatine aponeurosis	Side of tongue	CN XI through pharyngeal branch of vagus via pharyngeal plexus	Elevates posterior part of tongue and draws soft palate into tongue
Palato-pharyngeus*	Posterior boarder hard palate and palatine aponeurosis	Lateral wall of pharynx and posterior boarder thyroid cartilage	CN XI through pharyngeal branch of vagus via pharyngeal plexus	Tenses soft palate and pulls walls of pharynx superoanteriorly and medially during swallowing
Musculus uvulae	Posterior nasal spine and palatine aponeurosis	Mucosa of uvula	CN XI through pharyngeal branch of vagus via pharyngeal plexus	Shortens uvula and pulls it superiorly
Levator veli palatini	Cartilage of auditory tube and petrous part of the temporal bone	Palatine aponeurosis	CN XI through pharyngeal branch of vagus via pharyngeal plexus	Elevates soft palate during swallowing and yawning

Note: The asterisks denote pharyngeal dilators.
Source: From Ref. 27.

Table 3 Pharyngeal Muscles

Muscle	Origin	Insertion	Innervation	Action
Stylo-pharyngeus*	Medial styloid process of temporal bone	Posterior and superior boarders of thyroid cartilage with palatopharyngeus	CN IX	Dilate retropalatal oropharynx at rim of soft palate
Salpingo-pharyngeus	Inferior cartilaginous portion of auditory tube	Blends with palatopharyngeus	Cranial root of XI via pharyngeal br. of X and pharyngeal plexus	Elevates (shortens and widens) pharynx
Palato-pharyngeus	Posterior portion of hard palate and palatine aponeurosis	Posterior boarder of lamina of thyroid cartilage and side of pharynx and esophagus	Cranial root of XI via pharyngeal br. of X and pharyngeal plexus	Elevates (shortens and widens) pharynx
Superior constrictor	Ptergo-mandibular raphe, ptergoid plate, ptergoid hamulus, mandible	Median raphe of pharynx and tubercle of the occipital bone	Cranial root of XI via pharyngeal br. of X and pharyngeal plexus	Constricts pharyngeal wall during swallowing
Middle constrictor	Greater and lesser horn hyoid bone	Median raphe of pharynx	Cranial root of XI via pharyngeal br. of X and pharyngeal plexus	Constricts pharyngeal wall during swallowing
Inferior constrictor	Oblique line thyroid lamina and side of cricoid cartilage	Median raphe of pharynx	Cranial root of XI via pharyngeal br. of X and pharyngeal plexus and recurrent laryngeal	Constricts pharyngeal wall during swallowing

Note: The asterisk denotes pharyngeal dilator.
Source: From Ref. 27.

(structural) stiffness will cause initial narrowing, and the Bernoulli effect will lower effective stiffness. Neuromuscular function reaction to negative pressure is delayed during sleep and is powerless to stop the airway from collapsing. The adult human is the only mammal that suffers from OSA because the oropharyngeal complex lacks support; in other mammals the tip of the uvula touches the tip of the epiglottis and the hyoid bone supports this segment by its articulation with the cervical spine (32). The anatomic or structural changes within each pharyngeal segment are affected by neuronal tone and by the position of the patient (33). Studies suggest that the male predisposition to pharyngeal collapse is anatomically based, primarily as a result of the increased length of the vulnerable airway as well as increased soft palate size (12).

4.1 Nasopharynx

The nasopharynx is posterior to the nasal cavity beginning at the paired posterior nasal choanae just superior to the soft palate. The roof and posterior wall of the nasopharynx form a continuous surface that lies inferior to the body of the sphenoid bone and the basilar part of the occipital bone. The pharyngeal tonsil (adenoid) is in the mucous membrane of the roof and posterior wall of the nasopharynx and is often the site of obstruction in adenoid hyperplasia. The lateral wall of the nasopharynx contains the auditory tube and cartilage, the tensor and levator palate, and salpingopharyngeus muscles. Posterior to the auditory tube and salpingopharyngeal fold is the pharyngeal recess.

4.2 Oropharynx

The oropharynx extends from the soft palate to the tip of the epiglottis and is bounded laterally by the palatoglossal and palatopharyngeal arches. The collapsible segment posterior to the soft palate is referred to as the retropalatal pharynx. Inferior to that, the collapsible tongue base segment of the oropharynx, which is posterior to the tongue from the tip of the uvula to the tip of the epiglottis, is the retroglossal pharynx. The soft palate is connected to the hard palate by a tensor aponeurosis of connective tissue, which extends posterior-inferiorly from the margin of the hard palate. It serves as a point of attachment for much of the soft palate musculature and is continuous with the lateral pharyngobasilar fascia and tensor veli palatine muscle tendons (Fig. 10) The soft palate contains five muscles, four paired slings and one midline muscle, listed in Table 2. The palatopharyngeus muscle (posterior pillar) is the most superficial of the soft palate musculature and has anterior and posterior extensions that blend with the musculus uvula and levator veli palatini (Fig. 10). The palatopharyngeus originates on the hard palate and palatine aponeurosis and inserts into the lateral pharyngeal wall. During swallowing, it tenses the soft palate and pulls the pharyngeal walls superiorly, anteriorly, and medially. The uvular muscle (musculus uvulae) lies just deep to the posterior (upper) layer of the palatopharyngeus muscle (Fig. 10). It extends inferiorly in the soft palate midline and pulls the uvula superiorly and anteriorly. The palatoglossus muscle (anterior pillar) originates from the palatine aponeurosis and inserts into the side of the tongue. It draws the posterior tongue and soft palate together. It is a soft palate muscle because it receives vagus innervation like other soft palate musculature, and will be discussed in the section on external tongue musculature. The tensor veli palatini muscle originates from the lateral wall of the Eustachian tube cartilage, sphenoid spine, and scaphoid fossa of the medial pterygoid plate. As it passes inferiorly, it

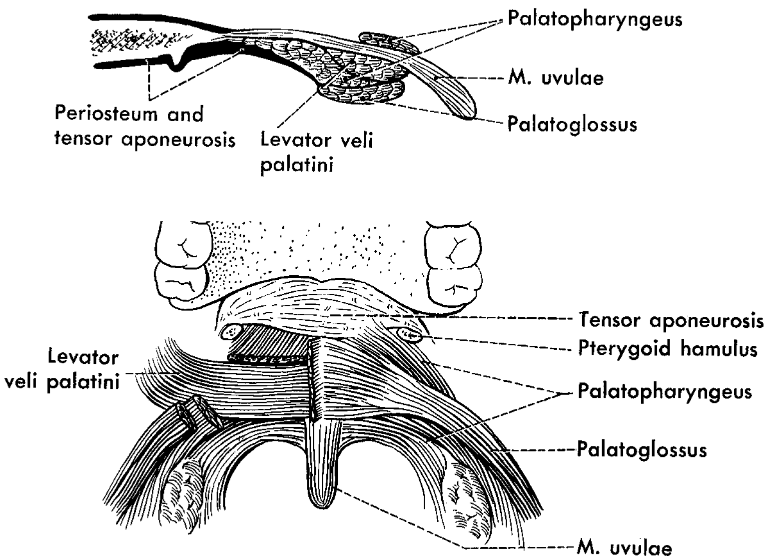

Figure 10 Diagram of the aponeuroticomuscular structure of the soft palate in longitudinal section above and muscles of the soft palate from below. (From Hollinshead WH. *Anatomy for Surgeons*: Volume 1, The Head and Neck. 2nd ed. Hagerston, MD: Harper and Row, 1968:386.)

hooks around the pteygoid hamulus and inserts into the palatine aponeurosis. The tensor veli palatini opens the Eustachian tube as well as tenses and retracts the soft palate away from the posterior pharyngeal wall (Table 2). The decrease in tonic activity of the tensor palatini during sleep correlates with increased upper airway resistance (9). The levator veli palatini muscle originates on the inferior surface of the petrous temporal bone and inferomedial Eustachian tube cartilage, travels inferoanteriorly between the musculus uvula and palatopharyngeus, and inserts into the soft palate. It contributes to soft palate bulk, opens the Eustachian tube, and retracts the soft palate to bring it in contact with the posterior pharyngeal wall. Together the levator palatini and palatopharyngeus work with the superior constrictor to close the retropalatal pharynx, an event important in speech and swallowing (Table 2).

Motor innervation to the soft palate is by the branches of the ascending pharyngeal plexus (X); however, the tensor veli palatini muscle associated with the Eustachian tube, is innervated by a branch of the mandibular nerve (V_3). The blood supply to the palate is primarily from the descending palatine artery, a branch of the maxillary artery. The greater palatine artery branches from the descending palatine artery, then travels with the greater palatine nerve anteriorly on the junction of the hard palate and alveolar process. It enters the incisive canal and ultimately anastamoses with septal branches of the sphenopalatine artery in the nasal cavity. The lesser palatine arteries anastomose with the ascending pharyngeal (palatine branch), facial (ascending palatine branch) and dorsal lingual (tonsillar branch) arteries on the soft palate. These accessory vessels are an important source of blood supply to the palate in maxillary advancement surgery, when the greater palatine vessels are sectioned in the posterior cuts of the Lefort osteotomy.

Pharyngeal lymphatic tissue predominates in an incomplete ring located in the pharyngeal (adenoid), tubal, palatine, and lingual tonsil regions known as

Waldeyer's ring (Fig. 11). The palatine tonsils are located in the fossa between the palatoglossal and palatopharyngeal arches. The pharyngobasilar fascia overlies the palatopharyngeus and superior constrictor muscles to create the tonsil bed. The dominant tonsillar blood supply is the tonsillar branch of the facial artery (Figs. 12 and 13). It travels through the superior constrictor muscle and enters the inferior tonsillar pole. Additional palatine tonsil blood supply includes tonsil branches of the ascending and descending palatine, lingual, and ascending pharyngeal arteries. The glossopharyngeal, vagus, and pharyngeal plexus branches innervate the tonsils and pharyngeal arch (Fig. 13).

4.3 Laryngopharynx

The laryngopharynx or hypopharynx lies posterior to the larynx (Fig. 11) and extends from the tip of the epiglottis and pharyngoepiglottic folds to the inferior border of the cricoid cartilage, where it narrows and becomes continuous with the eso-

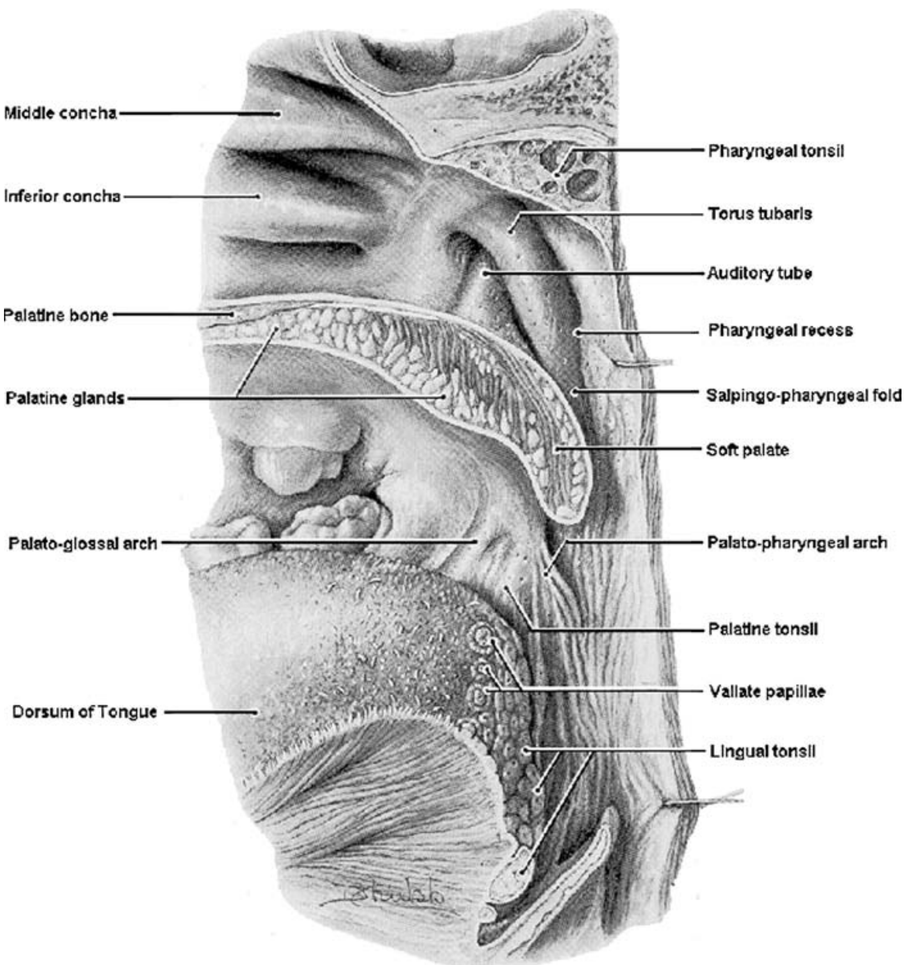

Figure 11 Interior of the pharynx, side view. (Modified from *Grant's Atlas of Anatomy*, 10th ed. Baltimore, MD: Williams and Wilkins, 1999.)

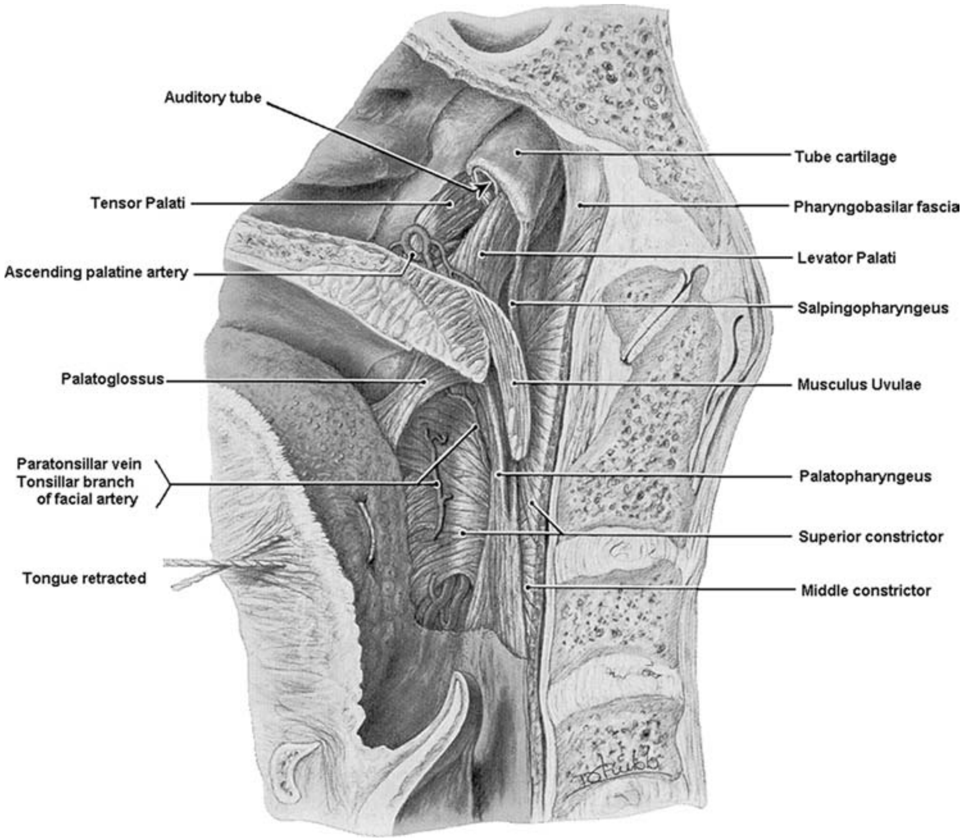

Figure 12 Interior of the pharynx dissected, side view. (Modified *Grant's Atlas of Anatomy*, 10th ed. Baltimore, MD: Williams and Wilkins, 1999.)

phagus. The segment capable of collapsing is referred to as the retroepiglottic pharynx, which lies just posterior to the epiglottis. Posteriorly, the laryngopharynx is related to the bodies of C4–6 vertebrae. Its posterior and lateral walls are formed by the middle and inferior constrictor muscles. Anterior movement of the hyoid bone by muscle pull or by surgical intervention will dilate the retroepiglottic pharynx through traction on the hyoepiglottic ligament (Fig. 5).

4.4 The Pharynx

The respiratory and alimentary passages become common in the middle part of the pharynx where the collapsing nature of the alimentary portion potentially compromises the respiratory passage during sleep, specifically, anterior obstruction from the palate (retropalatal), tongue (retroglossal), and epiglottis (retroepiglottic) and lateral pharyngeal wall collapse. Anatomically, the pharynx lies in front of the cervical vertebral column and prevertebral fascia and behind the nasal cavity, oral cavity, and larynx (Figs. 11 and 12). This fibromuscular tube has deficiencies in the walls at the nasal choanae, the oral cavity (oropharyngeal isthmus), and the laryngeal inlet. The lateral walls are complete except for the Eustachian tube openings in the nasopharynx. The mucosa of the nasopharynx is purely respiratory having respiratory

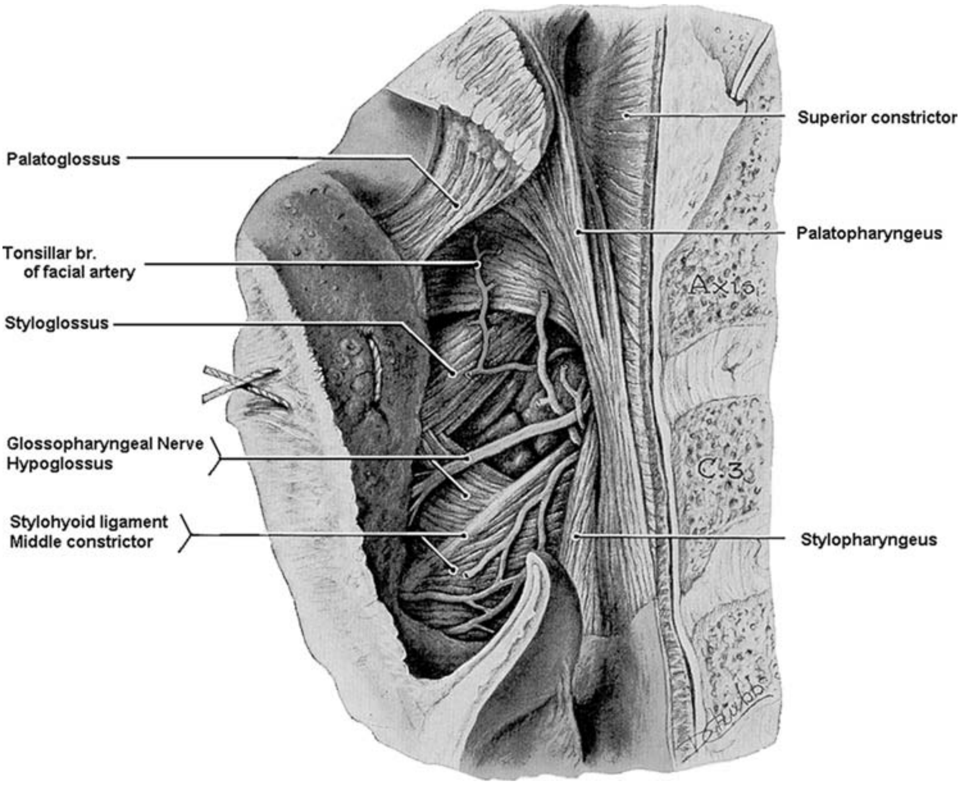

Figure 13 Interior of the pharynx deep dissection, side view. (Modified from *Grant's Atlas of Anatomy*, 10th ed. Baltimore, MD: Williams and Wilkins, 1999.)

epithelium, the oropharynx mucosa is both respiratory and alimentary, and the laryngopharynx is alimentary having stratified squamous epithelium. Outside the mucosa is the pharyngobasilar fascia, which is the submucosa of the pharyngeal wall and is visible superiorly since there is no muscular layer external to it (Fig. 12). This fascia separates the epithelium from the pharyngeal constrictor muscles. The constrictor muscles are surrounded by a thin buccopharyngeal fascia, which allows the pharynx expansion and mobility and contains the pharyngeal plexus of nerves (7).

The muscular wall of the pharynx is composed of two layers of three muscles each. The external layer is composed of circular constrictor muscles (superior, middle, and inferior), which contract serially to push a bolus down to the esophagus. The internal layer is composed of longitudinal muscles (palatopharyngeus, stylopharyngeus, and salpingopharyngeus), which elevate and dilate the pharynx to accommodate a bolus during swallowing (Figs. 12 and 13).

The superior constrictor has a continuous anterior attachment to the medial pterygoid plate, pterygoid hamulus, pterygomandibular raphe, and posterior end of the mylohyoid line. From here it sweeps posteriorly to attach in the midline to the pharyngeal tubercle of the occipital bone (skull base) superiorly and to the pharyngeal raphe inferiorly. The middle constrictor has a relatively smaller anterior attachment from the lower portion of the stylohyoid ligament and lesser and greater cornu of the hyoid bone. It fans posteriorly to attach at the pharyngeal raphe enclosing the superior constrictor above and extending to the level of the larynx below. The stylopharyngeus muscle and glossopharyngeal nerve pass through the gap between the superior and middle constrictors. The inferior constrictor arises from the oblique line on the lamina of the thyroid and cricoid cartilage. The fibers arch upward overlapping the middle constrictor; inferior fibers from the cricoid cartilage run horizontally and constitute the cricopharyngeus muscle. The three constrictors are supplied by the branches of the pharyngeal plexus IX, X, IX (cranial root), and sympathetic plexus. The cricopharyngeus muscle is supplied by the external branch of the superior laryngeal nerve (X). The action of the pharyngeal constrictors is elevation and compression of the pharynx (7).

The pharyngeus muscles (stylo-, salpingo-, and palatopharyngeus) dilate the pharynx and elevate the larynx in phonation and swallowing (Table 3). The stylopharyngeus muscle arises from the medial aspect of the styloid process and descends close to the superior pharyngeal constrictor and passes through the gap between the superior constrictor and middle constrictor muscles before attaching to the posterior boarder of the thyroid cartilage. It elevates the larynx and pharynx and is supplied by the glossopharyngeal nerve (IX). The salpingopharyngeus muscle arises from the cartilage of the Eustachian tube, descending inside the superior constrictor muscle to attach like the stylopharyngeus to the thyroid cartilage. It lies within the muscular wall of the pharynx, adding an additional fold to the pharynx below the Eustachian tube. The salpingopharyngeus elevates the larynx and pharynx and opens the Eustachian tube. It is innervated by the pharyngeal plexus. The palatopharyngeus muscle originates from the palatine aponerosis and inserts on the posterior pharyngeal wall and thyroid cartilage. It elevates the larynx and pharynx while depressing the palate and is innervated by the pharyngeal plexus. The most important dilator of the pharynx is the genioglossus muscle and, to a minor extent, the tensor veli palatini (Tables 1 and 2). The tensor palatini may require palatopharyngeal muscle coordinated activation to adequately influence upper airway collapse (34). The geniohyoid and sternohyoid muscles are considered as dilators of the pharynx. The infrahyoid muscles (thyrohyoid, omohyoid, and sternohyoid) work in conjunction with the geniohyoid at the hyoid bone to enlarge the retroepiglottic laryngopharynx by tensing the hyoepiglottic ligament. According to Benumof (9), "The inspiratory patency of the retropalatal, retroglossal, and retroepiglottic pharynx is caused by contraction of the tensor palatini, the genioglossus, and the hyoid bone muscles, respectively."

Sources for anatomic obstruction of pharyngeal airway include inflammatory disorders that cause diffuse enlargement of lymphatic tissues, hypertrophy of tongue and pharyngeal muscles, extra-pharyngeal fat compression, and structural deformities. Neoplastic disease (benign and malignant), metabolic and traumatic abnormalities are rare and should be considered in the evaluation of the obstructed airway. Patients with craniofacial abnormalities and sleep apnea compared with normal controls have small retroposed mandibles, narrow PAS, enlarged tongues and soft palates, inferiorly positioned hyoid bones, and retroposition of their maxillas (24,26,36). Patients with mandibular retrognathia (SNB $< 74^\circ$) and morbid obesity (BMI $> 33\ \mathrm{kg/m^2}$) are poor candidates for UPPP because it will not address the underlying process involving the lateral walls and retroglossal pharynx (37). Lymphoid hypertrophy causing airway obstruction may result in mouth breathing and deformities of the hard palate and upper maxillary dentition. The palatine tonsils cause obstruction of the lateral pharynx and displacement of palatoglossus and palatopharyngeus muscles. Lingual tonsil hypertrophy can obstruct the retroglossal and retroepiglottic airway in the midline. Both palatine and lingual tonsil hypertrophy respond well to a variety of surgical options (38). The lateral nasopharynx may have wall enlargement from tubal elevations (tori tubarii) from superior and posterior cartilaginous projections. In addition, muscle hypertrophy and lateral wall fold enlargement by the salpingopharyngeus muscle and palatopharyngeus muscles within the muscular wall may decrease the lateral airway space below the tubal elevations (Figs. 11–13). "The deposition of fat in the pharynx of OSA patients appears to be predominantly into the lateral walls of the pharynx and the volume of fat in the lateral pharyngeal walls correlates well with the severity of OSA. The converse is also true; weight loss improves the pharyngeal and glottic function of OSA patients. Fat deposition in the lateral walls not only narrows the airway but also changes the shape of the pharynx in obese patients from a long transverse (lateral) ellipse to a short transverse and long anterior-posterior axis. Pharyngeal dilator muscles that increase the upper airway size are located anterior to the pharynx and are less efficient with a long anterior-posterior elliptical axis" (9).

The pharynx is innervated through the pharyngeal plexus. The pharyngeal plexus is a group of fine, ramifying nerve fibers on the posterior aspect of the pharynx which lie over the middle constrictor muscles. Motor innervation is derived from the vagus nerve (X), which supplies efferent motor fibers from the cranial part of the accessory nerve which originate within the nucleus ambiguous and supply the pharyngeal constrictor muscles, levator palate, salpingopharyngeus, palatopharyngeus, and palatoglossus (all striated muscles derived from brachial arches). The exceptions are stylopharyngeus supplied by the glossopharyngeal nerve (IX) and the tensor veli palatini by the trigeminal nerve (V_3) (7). The vagus also supplies parasympathetic motor to the glands of the mucosa. Recent studies indicate that the glossopharyngeal nerve also supplies the levator veli palatini, pharyngeal constrictors, and cricopharyngeus muscles via its inputs to the pharyngeal plexus. Glossopharyngeal nerve activity has dilatory effects on the pharyngeal airway (14). Kuna (39) showed that stimulation of the glossopharyngeal nerve resulted in a greater increase in the lateral diameter of the retropalatal and oropharyngeal levels, whereas stimulation of the pharyngeal branch of the vagus decreased the retropalatal and retroglossal airways. Surgical treatment of the lateral wall to reduce soft tissue volume or increase tissue stiffening could potentially injure the glossopharyngeal nerve and potentially affect the dilator function of the stylopharyngeus muscle, possibly injuring the stylopharyngeus directly (Fig. 13).

Sensory innervation of the pharynx is by the vagus nerve (X) from afferent general somatic fibers originating in the sensory nucleus of the trigeminal nerve via the maxillary nerve (pterygopalatine ganglion) to the nasopharynx. The glossopharyngeal nerve (IX) supplies afferent fibers from the pharyngeal mucosa of the mucous membrane below the nasopharynx. Sympathetic fibers from the superior cervical ganglion supply the blood vessels and are responsible for vasoconstriction within the pharynx.

The blood supply of the pharynx is by multiple sources, most are branches of the external carotid artery and include the ascending pharyngeal, ascending palatine, facial, lingual, laryngeal, and superior and inferior thyroid arteries. Venous plexuses on the external surface of the larynx drain into the internal jugular vein. Lymph from the pharynx passes to the retropharyngeal lymph nodes and drains to the deep cervical nodes. The carotid and facial arteries are lateral to the middle and inferior constrictor muscles of the oropharynx and laryngopharynx (Fig. 13). These vessels would also be at risk of injury in surgical procedures treating the lateral pharynx at this level. Currently, surgical treatment of the obstructing pharynx is limited to "anterior wall therapy," treating the soft palate, advancing the tongue and hyoid bone, and advancing the maxilla and mandible. Superior and lateral wall therapy is limited to adenoid and tonsil removal. The lateral pharynx probably plays a significant role in some cases of OSDB; however, this potential is not easily quantified or often studied by current methods. Although a consideration for surgical therapy, one should proceed with caution as the potential to damage the pharyngeal dilator nerves and muscles and carotid artery is significant. For this reason and the fact that the posterior pharyngeal wall is not at this time an option for airway enlargement therapy, continuous positive airway pressure and surgical procedures to treat anteroposterior collapse of the palate, tongue, and epiglottis will continue as standard therapy for OSDB. Surgeons are encouraged to use universal nomenclature for naming the obstructing pharyngeal segments in their studies to improve research and surgical outcome. We must continue to pursue accurate site of lesion testing to identify the obstructing level(s) that accurately depict the obstructing segment during sleep. By understanding the anatomy and physiologic mechanisms which support the open upper airway during sleep, we can better expand our role in the diagnosis and management of OSDB and continue to pursue surgical treatment procedures that produce safe, functional, and lasting results.

REFERENCES

1. Horner RL. The neuropharmacology of upper airway motor control in the awake and asleep states: implications for obstructive sleep apnoea. Respir Res 2001; 2:286–294.
2. Mirza N, Lanza DC. The nasal airway and obstructed breathing during sleep. Otolaryn Clin North Am 1999; 32(2):243–262.
3. Janfaza P, Montgomery WW, Salman SD. Surgical Anatomy of the Head and Neck. Philadelphia, PA: Williams & Wilkins, 2001:259–272.
4. Kern EB. Rhinomanometry. Otolaryn Clin North Am 1973; 6:863.
5. Berger G, Balum-Azim M, Ophir D. The normal inferior turbinate: histomorphometric analysis and clinical implications. Laryngoscope 2003; 113(7):1192–1198.
6. Paff GH. Anatomy of the head and neck. Philadelphia, PA: W.B. Saunders Company, 1973:179.
7. Moore KL, Dalley AF. Clinically Oriented Anatomy. 4th ed. 4th. Philadelphia, PA: Lippincott Williams & Wilkins, 1999:1049–1059.
8. Mintz SM, Ettinger AC, Geist JR. Anatomic relationship of the genial tubercles to the dentition as determined by cross sectional tomography. J Oral Maxillofac Surg 1995; 53:1324.
9. Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. Anesthesiol Clin North Am 2002; 20(4):789–811.
10. Yang C, Woodson BT. Upper airway physiology and obstructive sleep-disordered breathing. Otolaryngol Clin North Am 2003; 36(3):409–421.
11. Mathur R, Mortimer IL, Jan MA, Douglas NJ. Effect of breathing pressure and posture on palatoglosal and genioglossal tone. Clin Sci (Colch.) 1995; 89:441–445.
12. Malhotra A, Huang Y, Fogel RB, et al. The male predisposition to pharyngeal collapse importance of airway length. Am J Respir Crit Care Med 2002; 166:1388–1395.
13. Li KK, Riley RW, Powell NB, Troell R, Guilleminault C. Overview of phase I surgery for obstructive sleep apnea syndrome. Ear Nose Throat J 1999; 78(11):836,837,841–845.
14. Kuna S, Remmers JE. Anatomy and physiology of upper airway obstruction. In: In: Principles and Practice of Sleep Medicine. 3rd ed. 3rd. Philadelphia, PA: W.B. Saunders Company, 2000:840–858.
15. Mallampati SR, Gatt SP, Gugino LD, Desai SP, et al. A clinical sign to predict difficult tracheal intubation: a prospective study. Can Anaesth Soc J 1985; 32(4):429–434.

16. Fujita S. Obstructive sleep apnea syndrome: pathophysiology, upper airway evaluation and surgical treatment. Ear Nose Throat J 1993; 72(1):67–72,75–76.
17. Friedman M, Ibrahim H, Bass L. Clinical staging for sleep-disordered breathing. Otolaryngol Head Neck Surg 2002; 127(1):13–21.
18. Dempsey JA, Skatrud JB, Jacques AJ, et al. Anatomic determinants of sleep-disordered breathing across the spectrum of clinical and nonclinical male subjects. Chest 2002; 122:840–851.
19. Woodson BT, Toohill RJ. Transpalatal advancement pharyngoplasty for obstructive sleep apnea. Laryngoscope 1993; 103(3):269–276.
20. Tebo HG, Telford IR. An analysis of the relative positions of the mental foramina. Anat Res 1950; 106:254 (abstract).
21. Castelli W. Vascular architecture of the human adult mandible. J Dent Res 1963; 42:786.
22. Riley R, Powell N, Guilleminault C. Inferior mandibular osteotomy and hyoid myotomy suspension for obstructive sleep apnea: a review of 555 patients. J Oral Maxillofac Surg 1989; 47:159–164.
23. Miles PG, Vig PS, Weyant RJ, et al. Craniofacial structure and obstructive sleep apnea syndrome—a qualitative analysis and meta-analysis of the literature. Am J Orthod Dentofacial Orthop 1996; 109:163–172.
24. Schwab RJ, Goldberg AN. Upper airway assessment: radiographic and other imaging techniques. Otolaryngol Clin North Am 1998; 31(6):931–968.
25. Shepard JW, Thawley SE. Evaluation of the upper airway by computerized tomography in patients undergoing uvulopalatopharyngoplasty for obstructive sleep apnea. Am Rev Respir Dis 1989; 140(3):711–716.
26. Trudo FJ, Gefter WB, Welch KC, et al. State-related changes in upper airway caliber and surrounding soft-tissue structures in normal subjects. Am J Respir Crit Care Med 1998; 158(4):1259–1270.
27. Williams PL, Warwick R, eds. Gray's Anatomy. 37th ed. London: Churchill Livingston, 1989:1323–1330.
28. Rama AN, Tekwani SH, Kushida CA. Sites of obstruction in obstructive sleep apnea. Chest 2002; 122:1139–1147.
29. Badr MS. Pathogenesis of obstructive sleep apnea. Prog Cardiovasc Dis 1999; 41:323–330.
30. Rowley JA, Sanders CS, Zahn BR, Badr MS. Effect of REM sleep on retroglossal cross-sectional area and compliance in normal subjects. J Appl Physiol 2001; 91:239–248.
31. Haung L, Williams JE. Neuromechanical interaction in human snoring and upper airway obstruction. J Appl Physiol 1999; 86(6):1759–1763.
32. Barsh CI. The origin of pharyngeal obstruction during sleep. Sleep Breath 1999; 3(1):17–21.
33. Isono S, Tanaka A, Nishino T. Lateral position decreases collapsibility of the passive pharynx in patients with obstructive sleep apnea. Anesthesiology 2002; 97(4):780–785.
34. McWhorter AJ, Rowley JA, Eisele DW, et al. The effect of tensor veli palatini stimulation on upper airway patency. Arch Otolaryngol Head Neck Surg 1999; 125:937–940.
35. Yu X, Fujimoto K, Urushibata K, Matsuzawa Y, Kubo K. Cephalometric analysis in obese and nonobese patients with obstructive sleep apnea syndrome. Chest 2003; 124(1):212–218.
36. Watanabe T, Isono S, Tanaka A, et al. Contribution of body habitus and craniofacial characteristics to segmental closing pressures of the passive pharynx in patients with sleep-disordered breathing. Am J Respir Crit Care Med 2002; 165(2):260–515.
37. Riley RW, Powell NB, Guilleminault C. Maxillary, mandibular, and hyoid advancement for treatment of obstructive sleep apnea: a review of 40 patients. J Oral Maxillofac Surg 1990; 48:20–22.
38. Suzuki K, Kawakatsu K, Hattori C, et al. Application of lingual tonsillectomy to sleep apnea syndrome involving lingual tonsils. Acta Otolaryngol Suppl 2003; 550:65–71.
39. Kuna ST. Effects of pharyngeal muscle activation on airway size and configuration. Am J Respir Crit Care Med 2001; 164:1236–1241.

2

The Anatomy of Sleep Disordered Breathing: An Evolutionary Perspective

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Nothing in biology makes sense except in the light of evolution.
—*Theodosius Dobzhansky*

1. INTRODUCTION

Anatomy is the foundation on which the house of surgery is designed and built. Sleep disordered breathing (SDB) is an anatomic abnormality. In spite of our physical examinations, surgical procedures, and clinical studies, we have failed to fully define the anatomy of SDB and find a simple, successful operation to correct this morbid, mortal condition.

It is possible that an evolutionary viewpoint answers the question of why anatomically modern *Homo sapiens* (*H. sapiens*) experience this disease and brings perspective to the anatomy. To elucidate the evolutionary concept, this chapter will describe the anatomic changes that have occurred in man's evolution from our closest living primate relative, the chimpanzee, to modern man.

The anatomic changes discussed herein are well described in the literature and are listed in Table 1. Table 2 describes several key terms. There are multiple theories on the cause of these modifications in man's anatomy, including bipedalism, binocular vision, and expansion of the brain. A fourth view, possibly the most important, is that the changes were driven by man's development of an upper respiratory tract to facilitate speech.

Man is arguably the only animal that experiences SDB (2,3). Man is inarguably the only animal with complex speech. Man is also the only animal with a true oropharynx, the principal site of SDB.

In order to speak and articulate vowels, the supralaryngeal vocal cord tract (SVT) requires a horizontal ratio (pharynx to lips) equal to the vertical ratio (vocal cords to pharynx) or $SVT_H:SVT_V=1:1$ (4,5). The SVT begins at the glottis, and

Table 1 Anatomic Requirements and SVT Changes for Modern Speech

Requirements	Changes
1:1 ratio SVT_V to SVT_H	Klinorynchy, laryngeal descent
Buccal speech	Laryngeal descent, shortened soft palate, loss of epiglottic–soft palate lock-up
Narrow, distensible, angulated SVT	Klinorynchy, anterior migration of foramen magnum, oropharyngeal tongue, acute craniobase angulation

Source: From Ref. 1.

includes the oropharynx and oral cavity. Figure 1 shows the 1:1 ratio in man, as compared to *Pan troglodytes*, the common chimpanzee.

The 1:1 SVT required descent of the larynx and shortening of the face. As the larynx was left unprotected in the new, elongated oropharynx, the tongue, which is flat in most animals and generally restricted to the oral cavity, became rounded and rotated posteriorly into the oropharynx. This facilitates speech and protects the larynx during swallowing, but when man falls asleep and the muscles relax, the tongue falls into the oropharynx and obstructs the airway. Sleep apnea then ensues (6).

Figure 2 is a drawing of the midsaggital section of the head of a goat, *Capra hircus*. Note the long mouth, small flat tongue, and high larynx. Figure 3(a) is an MRI of an adult *H. sapiens*. Note the short face, the large oropharyngeal tongue and the low larynx as compared to *C. hircus*. Figure 3(b) is a midsagittal MRI of the upper respiratory tract of an intubated, prone Rhesus monkey, *Macaca mulatto*, with the head extended. Comparisons to *H. sapiens* and *C. hircus* show the marked changes that have occurred to *H. sapiens*.

This chapter will describe the upper aero-digestive tract of modern mammals using the common chimpanzee, our closest relative, review the anatomic evolutionary changes from chimpanzee to modern man, and last, explore current scientific work showing that these changes are more pronounced in those with increasing SDB severity.

2. ANATOMY

P. troglodytes, the common chimpanzee, is an obligate nose breather. Air passes through the nose to the nasopharynx. The larynx sits high, adjacent to C2. The

Table 2 Key Terms

Primates: Diverse order of mammals ranging from lemurs to humans. Man belongs to the family Pongidae, which includes the great apes, the genera *Pongo* (orangutans), *Pan* (chimpanzees), *Gorilla* (gorillas) and *Homo* (man)

Homo sapiens (abbreviated *H. sapiens*): Genus and species of man. Anatomically modern man (abbreviated *H. sapiens*) separates this group from various taxa of "archaic" *H. sapiens*

Pan troglodytes (abbreviated *P. troglodytes*): The common chimpanzee

Supra vocal cord tract (abbreviated SVT): The voice passage from vocal cords to oral lips including the supraglottis, oropharynx, and oral cavity. The vertical segment, SVT_V, extends from the vocal cords to the top of the oropharynx. The horizontal segment, SVT_H, extends from the oropharynx to the lips

Source: From Ref. 1.

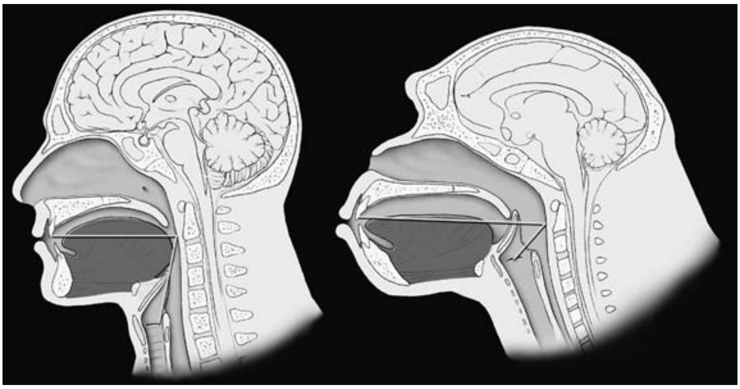

Figure 1 Ratios of distances from incisor to pharynx and pharynx to larynx in *H. sapiens* and *P. troglodytes*. The 1:1 SVT_V to SVT_H ratio is shown on the left. For comparison, the same ratio for the common chimpanzee is shown on the right. (From Ref. 1.)

epiglottis locks behind the long soft palate. The oropharynx, meaning soft palate to hyoid, is essentially nonexistent, so air passes from the nasopharynx directly into the larynx, i.e., it does not pass through an oropharynx.

The mandible and maxilla are long and robust, primarily to hold the teeth. Using the formula of incisors (I), canine (C), premolars (P), and molars (M), nonhominid primates have 44 teeth designated:

$$\begin{matrix} \text{Maxilla} & \text{3I-1C-4P-3M} \\ \text{Mandible} & \text{3I-1C-4P-3M} \end{matrix} = 44$$

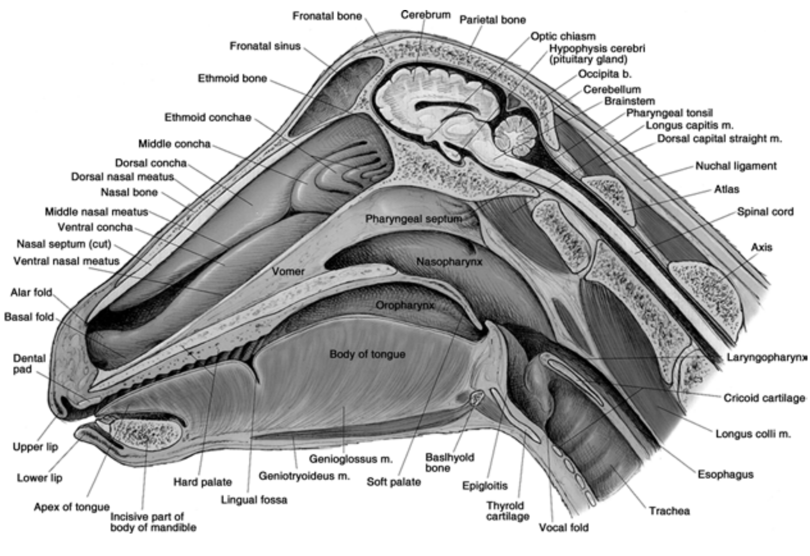

Figure 2 Midsagittal view of *C. hircus*. Note the high position of the larynx relative to the descended position in man, the relationship of epiglottis to soft palate (epiglottic–soft palate lock-up), the facial projection, the long maxilla and mandible, the length of the sphenoid bone, the obtuse craniobase angle, the long palate to foramen magnum distance, and the small, flat tongue, which resides exclusively in the oral cavity. (From Ref. 7.)

The tongue of nonhominid, modern mammals, primates included, is long, flat, and confined to the oral cavity. The tongue's primary function is to bolus food to the hypopharynx for swallowing.

The soft palate is long and drapes like a curtain in front of the epiglottis. Food is therefore shuttled under the soft palate, around the epiglottis, and directly into the hypopharynx, which is large in diameter. Prehominid mammals can typically breathe

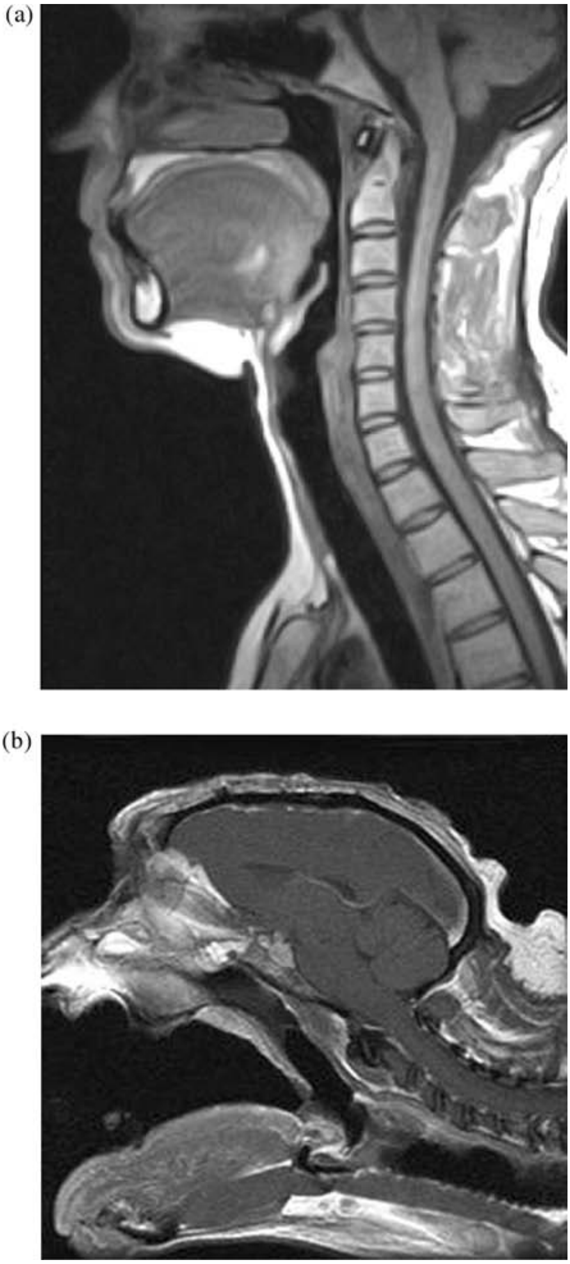

Figure 3 (*Caption on facing page*)

and swallow at the same time, advantageous both for respiration and for olfaction. As previously mentioned, the larynx is high, typically adjacent to C1–C2. Aspiration of food is uncommon.

In *H. sapiens*, the nose is intended as the primary respiratory tract. However, humans with anatomic and inflammatory nasal problems use the oral cavity to assist in respiration. Most humans use their oral cavity for respiration during heavy exertion. Many suffer partial to total nasal obstruction when asleep and the oral cavity becomes a major part of the upper respiratory tract.

Man's nasopharynx is held open, as it is in animals, by a bony skeleton including the pterygoid bones laterally, the basisphenoid above, and the occipital bone and cervical spine posteriorly.

Air must pass from the nasopharynx through the oropharynx to the laryngeal introitus. The oropharynx is a floppy, distensible tube and is partially supported by bone. This area can be filled with soft tissue including the tonsils, tongue, and soft palate. The top of the oropharynx is variably defined as beginning at the posterior hard palate or at the posterior soft palate. The bottom of the oropharynx is at the level of the hyoid. The valleculae lie at the level of the hyoid and may be used to define the inferior border of the oropharynx when viewed from above by direct or endoscopic inspection. The larynx is low, lying at the level of the bottom of C4. Once air enters the larynx, the respiratory tube is well supported by cartilage.

Sleep apnea is a disorder almost exclusively of the oropharynx. This 2–3 cm portion of the pharynx is the only piece of the respiratory tract not fully supported by skeleton. Man is really the only animal with an oropharynx. Other modern mammals have an oropharynx in theory, but in most animals, the distal nasopharynx connects directly to the proximal laryngopharynx (hypopharynx), so the oropharynx for all intents and purposes is a nonexistent structure. Even if one argues that the oropharynx extends from hard palate to hyoid and is an existing portion of the pharynx in animals, it is wide and short, and as the epiglottis protrudes superiorly, the respiratory tract is fully protected from the digestive tract and from the collapse of the surrounding soft tissue during sleep.

While man's oropharynx is easily distended, such as when exhaling against pursed lips, it is easily collapsed, as readily seen during a Mueller maneuver.

Figure 3 (*Facing page*) Midsagittal MRI of *H. sapiens* and *Macaca mulatta*. (a) T1 midsagittal MRI of an adult male *H. sapiens*. Note in comparison to Figs. 2 and 3(b), the narrow oropharynx, the short face, the low-lying larynx adjacent to the bottom of C4, and the large globular tongue. A portion of the tongue falls in the oropharynx. (b) T1 midsagittal MRI of the upper respiratory tract of a Rhesus monkey, *M. mulatta*. The anesthetized patient is sternal recumbent and the endotracheal catheter is placed transorally. As the animal is anesthetized, the head is extended. Compared to Fig. 3(a), note the long soft palate, the craniobase extension, the wide oropharynx, the high larynx with vocal cords adjacent to the bottom of C1, and the relatively small, flat tongue confined to the oral cavity. As the animal is intubated, the epiglottis has been pressed anteriorly, but in the nonintubated patient, would lock behind the soft palate. If one defines the superior end of the oropharynx as beginning at the level of the posterior end of the soft palate, then the upper and lower boundaries of the oropharynx essentially overlap and there is no oropharynx. Conversely, if one defines the oropharynx as beginning at the posterior border of the hard palate, then an oropharynx exists, but as can be seen, is quite wide. (Courtesy of Erik Wisner, D.V.M., University of California, Davis, School of Veterinary Medicine.)

As for deglutition, man's oral cavity is short, barely holding 32 teeth, which are designated:

$$\begin{array}{ll} \text{Maxilla} & \text{2I-1C-2P-3M} \\ \text{Mandible} & \text{2I-1C-2P-3M} \end{array} = 32$$

This includes the wisdom teeth, i.e., the third molars, for which there is often little room. Man's teeth are also significantly smaller than the teeth of similar-sized animals (8). They are reportedly 10% smaller than those in a similar-sized primate (9). There is little to no room for the four third molars and orthodontists often have the four premolars extracted to make room for orthodonture. This would leave man with 24 teeth designated:

$$\begin{array}{ll} \text{Maxilla} & \text{2I-1C-1P-2M} \\ \text{Mandible} & \text{2I-1C-1P-2M} \end{array} = 24$$

Other mammals do not have impacted molars, even though they have more teeth, all of which are larger.

In some humans, the oral cavity is particularly short in an anterior posterior direction and narrow in a coronal dimension. The soft palate is short compared to other animals. The oropharynx transcends a distance of 2–3 cm. Food is bolused from the oral cavity to the oropharynx. During deglutition, food passes directly over the laryngeal introitus before passing into the hypopharynx.

One hundred and forty-five years ago, Charles Darwin noted "... the strange fact that every particle of food and drink we swallow has to pass over the orifice of the trachea with some risk of falling into the lungs" (10). To protect the airway, the supraglottis must close and the oropharyngeal tongue must press the bolus through the oropharynx, over the larynx, and into the hypopharynx. It is unclear whether the epiglottis plays any role in deglutition as its resection rarely results in aspiration in contrast to the aspiration difficulties which occur following oropharyngeal tongue resection or supraglottectomy.

In summary, the upper respiratory tract in nonhominid mammals is well supported throughout its length and clearly separated from the alimentary tract. The two tracts cross at the level of the epiglottis, but as the epiglottis sits high and locks behind the soft palate, and as the soft palate is long, food passes on either side of the airway.

In man, the respiratory and alimentary tracts must share the floppy oropharynx. At one moment the oropharynx serves the respiratory tract and at another moment the oropharynx serves the alimentary tract.

3. EVOLUTIONARY ANATOMIC CHANGES

Seen from an evolutionary perspective, the anatomy of man's upper respiratory tract is particularly interesting. The driving force for recent evolutionary nonhominid change is the positive selection for speech (11). The changes seen are predicated on the fact that in order to pronounce vowels, the supralaryngeal vocal cord tract requires that $SVT_H:SVT_V=1:1$ (4,5). Conversely, other theories for recent evolutionary change, i.e., bigger brain, binocular vision, or bipedalism, would not have required the changes described in this chapter.

There are several ways to view evolutionary change. The first is to choose some distant relative and examine the evolved differences. Man's closest living relative is

P. troglodytes, the common chimpanzee. This of course assumes that our mutually common ancestor, approximately 5 million years ago, looked more like *P. troglodytes* than *H. sapiens*.

A second perspective is to assume that ontogeny recapitulates phylogeny or that development from embryo to adult in some manner reflects our phylogenetic development. This approach has been taught and held to be true by scientific students of evolution.

A third way to view evolution is to examine fossils of more recent ancestors and to deduce from their skeletons what changes have occurred most recently.

A fourth viewpoint is examination of the spectrum of variation in modern man to determine whether there is a correlation with anatomic changes, in this case, a correlation of anatomic change with increasing apnea–hypopnea index (AHI).

All four approaches will be used to describe the evolutionary changes of the SVT that predispose man to SDB.

3.1 Klinorynchy

Klinorynchy is the rotation of the splanchnocranium under the neurocranium (2). Figure 4 is an artist's depiction of the evolution from *P. troglodytes* to

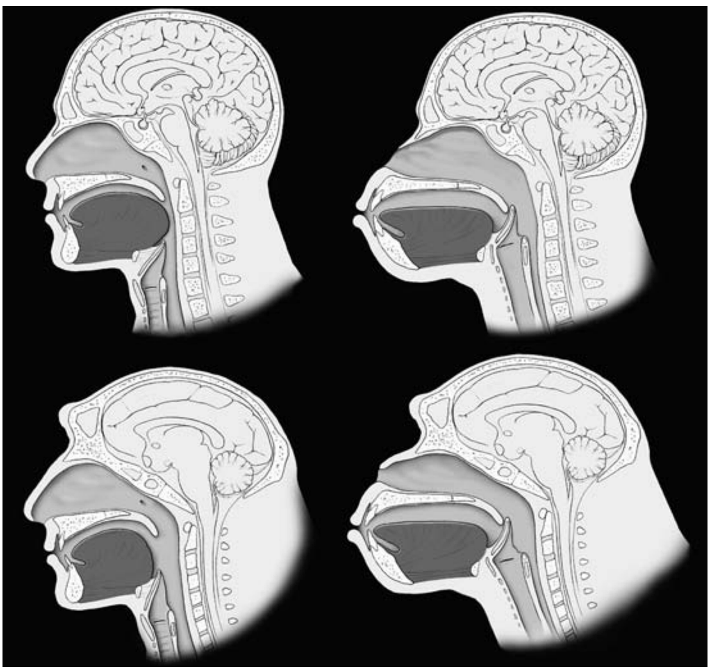

Figure 4 Klinorynchy as demonstrated by the evolution from *P. troglodytes* to *H. sapiens*. The lower right figure is a midsagittal view of *P. troglodytes*. The upper left figure is a midsagittal view of *H. sapiens*, with the tongue drawn in the awake position with the tongue base pulled forward. The upper right figure shows the splanchnocranium of *P. troglodytes* combined with the neurocranium of *H. sapiens*. The lower left shows the neurocranium of *P. troglodytes* combined with the splanchnocranium of *H. sapiens*. The key changes have not been driven by the expansion of the neurocranium over the midface, but rather the retrusion of modern man's mid- and lower faces. (From Ref. 1.)

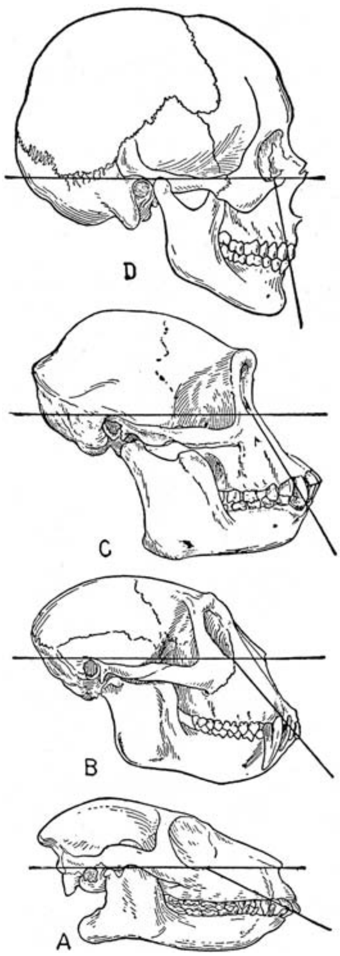

Figure 5 Side view of skulls of primates, showing progressive shortening of the muzzle, downward bending of the face below the eyes and forward growth of the chin. A. Eocene lemuroid; B. Old World monkey; C. Female chimpanzee, D. Man. (B and C after Elliot.) (From Ref. 12.)*

H. sapiens. One might think the frontal lobes of the brain increased in size and pushed the forehead over the face. While the brain did expand anteriorly, the majority of the change comes from shortening of the maxillary bones, palatine bones, ethmoid bones, and mandible. Numerous sources document this shortening of the splanchnocranium (4,12). Figure 5, from the anthropology book, *Our Face from Fish to Man*, originally printed in 1929, shows klinorynchy from lemur to man.

The change in dentition from 44 teeth in *P. troglodytes* to 32 teeth in *H. sapiens* further documents the shortening of the facial bones, specifically those bones surrounding the oral cavity, namely maxillary, palatine, and mandibular bones.

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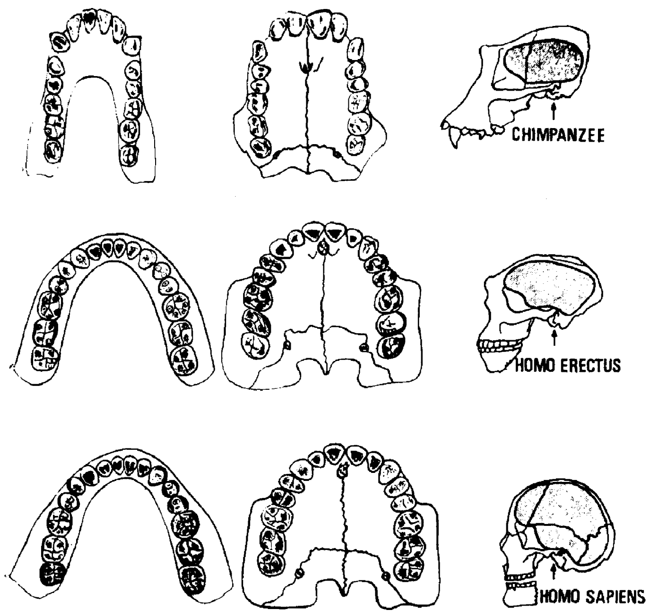

Figure 6 Maxillae of *P. troglodytes*, *Homo erectus*, and *H. sapiens*. The maxilla of *H. sapiens* is short and wide. The teeth are crowded. The shortening of the maxilla is depicted in the lateral views. The arrows on the figure's right depict the anterior rim of the foramen magnum and serve as a reference point for the posterior pharynx. Note the narrowing of the pharynx as depicted by the distance from the posterior maxilla to the anterior foramen magnum. (From Ref. 9.)

3.2 Anterior Migration of the Foramen Magnum

The second change that shortened the SVT_H was the anterior migration of the foramen magnum and the cervical spine. This is shown in Fig. 6. Examination of primate skulls shows that the foramen magnum is located more anteriorly the closer one gets to modern man (13). While one could opine that this helped balance the forward enlarging cranium and favored bipedalism, one can also opine that this further shortened the horizontal segment of the SVT, thereby improving speech. Keep in mind that the further the larynx descends, the longer the oropharynx extends, the more the respiratory and alimentary tracts overlap, and the more at risk is man of aspiration and asphyxiation.

3.3 Laryngeal Descent and Loss of Epiglottic-Soft Palate Lock-Up

The next and perhaps most important change that occurred in man's anatomy is laryngeal descent. Negus describes descent of the larynx in the classic text, *The Comparative Anatomy and Physiology of the Larynx*. Negus writes that the larynx and epiglottis in all animals reside superior to the oropharynx (please note that if the larynx resides superior to the oropharynx, there is no oropharynx). In many mammals, including dolphins, bears, and dogs, the larynx sits at the skull base. The monkey's larynx is between the skull base and the first cervical vertebra. The cat and the squirrel have the lowest-lying larynx, which resides at the top of the first cervical vertebra (14). Only man has a descended larynx, which is located between the third and fourth

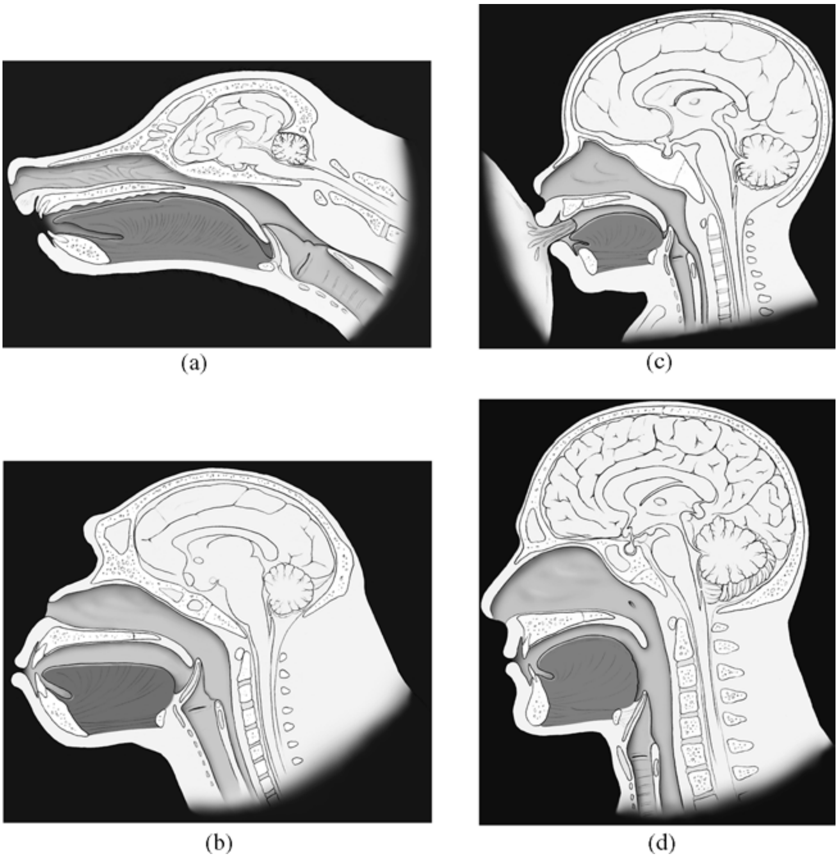

Figure 7 The epiglottic–soft palate relationship and the descent of the larynx. (a) In the dog, *Canis familiaris*, the tongue resides exclusively in the oral cavity, the epiglottis and soft palate are locked up, and the larynx resides high in the neck. (b) In the common chimpanzee, *P. troglodytes*, the tongue resides exclusively in the oral cavity, the epiglottic–soft palate relationship persists, and the larynx is high. (c) In the infant *H. sapiens*, the epiglottic–soft palate lock-up persists (ontogeny recapitulates phylogeny), the larynx is high, and the tongue is primarily in the oral cavity. As the juvenile matures, the larynx descends and the tongue falls into the pharynx. (d) In the adult *H. sapiens*, the epiglottic–soft palate lock-up is lost. The larynx is descended. The tongue protrudes into the pharynx. (From Ref. 1.)

cervical vertebrae in the human newborn and is located at the bottom of the fourth cervical vertebrae in the human adult. Figure 7(a–d) shows these relationships in the dog, the chimpanzee, the infant human, and the adult human.

Negus' view of the evolution of speech is summarized as follows. As primates assumed an upright position, they began to rely more on vision than on olfaction. This permitted the degeneration of the sense of smell and liberated the soft palate (14).

The degeneration of the sense of smell liberated the soft palate from the necessity of contact with the epiglottis and allowed a gap to be interposed between the two. After separation had occurred, it became easy for laryngeal sounds to escape from the mouth and for the oral cavity, lips, and oral tongue to form vowel and consonant sounds, i.e., buccal speech.