Anatomy and Physiology of Obstructive Sleep Apnea

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KEYWORDS

• Obstructive sleep apnea • Pathophysiology • Anatomy • Hypoglossal nerve • Pcrit • Ventilator control • Pharyngeal • Risk factors

KEY POINTS

- The oropharynx is the most common site of airway collapse in obstructive sleep apnea (OSA), and enlarged parapharyngeal fat pad, thicker lateral pharyngeal walls, and increased tongue volume play key roles.
- Pharyngeal closing pressure is influenced by craniofacial abnormalities, soft tissue crowding from obesity, caudal traction, and lung volumes.
- Reversible heightened arousal threshold in apneic patients combined with high loop gain ventilator response to arousals contribute to ventilatory instability.
- Obesity, sex, and age are pathophysiologic factors that promote OSA.

ANATOMY

This article reviews how the upper airway anatomy influences the pathophysiology of obstructive sleep apnea (OSA), and the pertinent anatomy.

The upper airway is a common passageway for digestive, respiratory and phonatory systems. Traditionally it is divided to 3 sections: the nasopharynx, oropharynx, and hypopharynx.

The nasopharynx extends from the posterior margin of the nasal turbinates; it sits above the soft palate and continues inferiorly with the oropharynx. The posterior wall is occupied by adenoids, which when inflamed can partially obstruct the upper airway. The soft palate, a nearly vertical flap, extends from the posterior edge of hard palate and terminates in the uvula. All the muscles of the soft palate are innervated by the pharyngeal branch of vagus nerve except the tensor veli palatini, which is innervated by the medial pterygoid nerve. A posterior elevation of the soft palate toward the posterior pharyngeal wall can cause enlargement of the oral cavity during swallowing and produce narrowing of the nasopharynx. Adenotonsillar disease can lead to sleep-disordered breathing. Polysomnography in children with allergic rhinitis and adenoidal hypertrophy found that 66% have mild apnea.[1] In fact in children, tonsillectomy and/or adenoidectomy is the first therapeutic modality to be considered for the treatment of OSA.[2]

Humans naturally breathe through the nose, particularly during sleep, when the daily oral fraction of breathing, estimated at 7%, drops to 4% during sleep.[3] Although during wakefulness both nasal and oral resistances are equal, nasal resistance is lower than the oral at night,[4] but increases in the supine position.[5] Hippocrates[6] first mentioned the connection between the nose and breathing in sleep when he described a role of

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Sleep Med Clin 8 (2013) 425–431
http://dx.doi.org/10.1016/j.jsmc.2013.07.016
1556-407X/13/$ – see front matter

nasal polyps in restless sleep. Nasal obstruction can occur secondary to deviated septum, chronic rhinosinusitis, and nasal polyps. Nasal congestion has been associated with a 3-fold increase in the incidence of snoring and daytime sleepiness.[7] Earlier studies indicate that acute nasal obstruction could increase the apnea-hypopnea index (AHI), prolong rapid eye movement (REM) latency, and increase non-REM (NREM) sleep.[8] However, nasal obstruction alone is not thought to cause any moderate or severe OSA.[6,9]

The hard and the soft palate form the roof of the oral cavity, and the lingual mucosa covers the floor. The lateral part of the oral cavity is covered by buccal mucosa and anterior pillars of palatine tonsils, which define the junction with oropharynx. The tongue, which occupies the major part of the oral cavity, has both extrinsic and intrinsic muscle groups. The 4 extrinsic tongue muscles are the genioglossus, hyoglossus, palatoglossus, and styloglossus. The genioglossus is the largest and most-studied pharyngeal dilator muscle. All of these muscles are innervated by the hypoglossal nerve except the palatoglossus, which is innervated by the vagus nerve. The intrinsic muscles of the tongue (superior and inferior longitudinal, transverse, and vertical muscles) are confined to the tongue. The anterior two-thirds of the tongue is innervated by the facial nerve, whereas the posterior one-third is innervated by cranial nerve IX. The size of the tongue is an important risk factor for OSA.[10]

An increase in the size of type II muscle fibers is seen in OSA compared with normal subjects, which could represent a response to vibratory strain or perhaps neuronal activity.[11,12]

The hypoglossal nerve is a critical component in the motor control of upper airway dilatation. The muscle fibers in the posterior part of the tongue are fatigue-resistant, thereby sustaining the forward tongue position and preventing its collapse into the retroglossal area. Using this mechanism, the therapeutic effect of proximal hypoglossal nerve stimulation can be used to treat OSA.[13]

The oropharynx extends from the soft palate to the epiglottis. The anterior part of oropharynx is formed by the posterior part of the tongue and the soft palate, whereas the posterior part is formed by the pharyngeal constrictor muscles. The lateral pharyngeal walls are formed by the pharyngeal constrictors, muscles of the extrinsic tongue, muscles of the soft palate, and the larynx. Other structures that contribute to upper airway lumen, located in the retropalatal area, are the palatine tonsils and parapharyngeal fat pads.

A magnetic resonance imaging (MRI) study revealed a smaller minimum airway area in patients with OSA in the retropalatal region, and particularly in the lateral dimension, compared with individuals with normal breathing.[14] The volume of the tongue and lateral walls have been shown to independently increase the risk of OSA.[15]

The oropharynx is the most common site of airway collapse in patients with OSA,[16] which is more likely to occur during REM sleep.[17] A recent study evaluating the role of parapharyngeal fat in the predisposition to OSA used MRI to examine pharyngeal anatomy. Patients with retropalatal airway closure had a higher percentage of parapharyngeal and soft palate fat, whereas patients with retroglossal airway closure had an increased volume of the tongue and parapharyngeal fat pad (**Fig. 1**).[18]

The caudal portion of the upper airway is the hypopharynx, which extends from the superior border of the epiglottis to the inferior border of the cricoid cartilage. It is formed anteriorly by the base of the tongue and the epiglottis, and

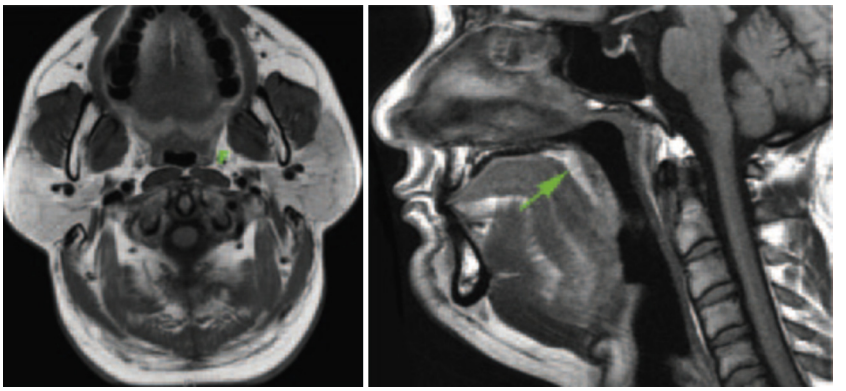

Fig. 1. Parapharyngeal fat pad and fat tissues in the soft palate (*green arrow*). (*From* Li Y, Lin N, Ye J, et al. Upper airway fat tissue distribution in subjects with obstructive sleep apnea and its effect on retropalatal mechanical loads. Respir Care 2012;57(7):1100; with permission.)