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Control of the Pharyngeal Musculature During Wakefulness and Sleep: Implications in Normal Controls and Sleep Apnea

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Abstract

Respiration involves the complex coordination of several pump and upper airway/pharyngeal muscles. From a respiratory perspective, the major function of the pharyngeal muscles is to keep the airway patent allowing for airflow in and out of the lung with minimal work by the respiratory pump muscles. The activity of each of the pharyngeal muscles varies depending on its function, but many reduce their activity during sleep. In healthy individuals, these muscles can respond to respiratory stimuli during sleep in order to prevent airway collapse. However, in individuals with an anatomically small airway, the muscles cannot always compensate for the increased mechanical load. Thus, a vulnerable situation in which the airway is prone to collapse may occur with the development of obstructive sleep apnea (OSA). The present article describes the current understanding regarding the control of the pharyngeal musculature during wakefulness and sleep, as well as the implications for OSA.

Keywords

Pharyngeal muscle control; breathing; wake; sleep; obstructive sleep apnea

Introduction

The human pharyngeal airway is a complicated structure comprising a number of muscles whose functional integration is essential for several complex tasks including swallowing, speech and breathing. From the respiratory perspective, the primary goal of these pharyngeal muscles is to keep the airway open allowing for the flow of air in and out of the lung with minimal work by the respiratory pump muscles. While the pharyngeal muscles manage this task with relative ease during wakefulness, their activity is often diminished at sleep onset^(1–3) causing the airway to become narrower, increasing the resistance and thus the propensity for collapse. In healthy individuals, the pharyngeal muscles are able to adequately compensate for the increase in airway resistance to maintain a patent airway. However, individuals that have a narrow upper airway, either due to obesity or bony structures crowding the airway, are at an increased risk of pharyngeal collapse during sleep⁽⁴⁾. This is the case in most patients with obstructive sleep apnea (OSA), a disorder characterized by repetitive collapse of the upper airway during sleep. In this article we review the current understanding of the control of the pharyngeal musculature in healthy individuals, the impact that sleep has on their control, as well as the implications that this has for the pathophysiology of OSA.

Pharyngeal muscle groups and their respiratory modulation

The human pharyngeal airway extends from the nasal septum to the epiglottis and despite the posterior pharyngeal wall, has relatively little bony or rigid support (Figure 1). The airway is therefore largely dependent on the activity of the pharyngeal muscles to maintain patency. Based on their function and anatomy, the pharyngeal muscles are divided into four primary groups. Table 1 summarizes the muscles that make up each of these four groups, where they originate and insert, nerve they are innervated by, and the major action they perform when activated. Although each of the muscle groups is described below, it is noteworthy that some pharyngeal muscles have been better characterized than others. We describe the current understanding regarding respiratory modulation of these muscles; i.e. whether they are active predominately during inspiration or expiration and whether their activity occurs in bursts (phasic) or is continuous (tonic). Furthermore, we address the responsiveness of the pharyngeal muscles to common respiratory stimuli such as hypercapnia, hypoxia as well as increases in pharyngeal negative pressure.

Extrinsic Muscles of the Tongue

The extrinsic muscles of the tongue consist of the genioglossus, hyoglossus, and styloglossus and are important for the protrusion and retraction of the tongue. The genioglossus is by far the most extensively studied of all the pharyngeal airway muscles both in humans and animals and as such will be a large focus of our discussion. The genioglossus is the primary protruder muscle of the tongue with its contraction playing a seminal role in keeping the pharyngeal airway open, mainly by widening the oropharynx in the anterior-posterior dimension⁽⁵⁾. The genioglossus receives input from respiratory pattern generating neurons in the brainstem, negative pressure receptors within the upper airway, and the neurons that regulate state (wakefulness versus sleep). As such, it is not surprising that the genioglossus demonstrates respiratory modulation as almost all investigations indicate that this muscle has a phasic pattern of activation during inspiration with tonic activity present during expiration^(6, 7). The tonic activity is considered to be important in determining the baseline size and stiffness of the airway, with the phasic activity during inspiration counteracting the negative pressures generated by the diaphragm⁽⁸⁾. Evidence that the genioglossus receives direct input from the respiratory central pattern generator (CPG) comes from studies demonstrating the muscle is activated approximately 50–100 msec prior to the diaphragm or the start of inspiratory flow⁽⁹⁾. This suggests that the CPG is preparing the airway for forthcoming inspiratory effort. Furthermore, both hypoxia and hypercapnia^(10, 11) lead to increases in both the tonic and phasic activity of the muscle⁽¹⁰⁾. Importantly, the genioglossus also responds to negative airway pressure^(12, 13). Negative pressure in the airway activates mechanoreceptors located in the larynx which, through the superior laryngeal nerve, lead to reflex muscle stimulation⁽¹³⁾. It is this reflex that allows the muscle to react to threats to airway patency with increased contraction, thereby assisting in maintaining or increasing the size of the airway.

Most studies addressing genioglossal activation described to date, have utilized multiunit recording techniques which describe the global activity of many single motor units that comprise the muscle. More recently, protocols recording from single motor units have demonstrated considerably greater complexity in the control of this muscle than was previously thought⁽¹⁴⁾. Saboisky et al⁽¹⁵⁾ described six different patterns of firing for single motor units in the genioglossus (see Figure 2); four had clear respiratory modulation (inspiratory phasic, inspiratory tonic, expiratory phasic and expiratory tonic) while two fired with little relationship to the respiratory cycle (tonic and tonic other). In their study, Saboisky et al⁽¹⁵⁾ showed that approximately 50% of the 110 motor units recorded displayed inspiratory modulation, 16% were active during expiration, and 34% showed no respiratory modulation. Importantly, these motor units generally have a higher firing frequency than is

commonly observed in other respiratory muscles and increase their activity primarily by recruitment of new motor units as opposed to increased firing frequency⁽¹⁶⁾. However, it remains interesting that a single muscle can display the differential firing patterns (inspiratory, expiratory and tonic) seen in the genioglossus. As such, considerable future investigation will be needed to fully understand the control of this and other upper airway dilator muscles.

The other two extrinsic tongue muscles, hyoglossus and styloglossus, are involved in the retraction of the tongue⁽¹⁷⁾. Little is known about whether these muscles show respiratory modulation in humans, but available data in animals suggest that they are inactive under baseline conditions but can respond to hypoxia and hypercapnia in an inspiratory phasic manner^(18, 19). Furthermore, isolated stimulation of either retractor muscles in humans does not seem to improve pharyngeal patency whereas stimulation of the genioglossus does reduce airway collapsibility⁽²⁰⁾. Interestingly, there is some evidence to suggest that all three of the extrinsic muscles can be co-activated simultaneously in response to respiratory stimuli. Such co-activation results in a general stiffening of tongue position which can reduce airway collapsibility⁽¹⁰⁾, indicating that the airway size is related to the balanced neural drive to the tongue protruders and retractors.

Palatal Muscles

There are five palatal muscles; levator palatini, tensor palatini, palatoglossus, palatopharyngeus, and musculus uvula, and their actions are heavily dependent on the whether the breathing route is oral or nasal. The levator palatini, whose activation closes the nasal airway thus promoting oral respiration, has been shown to have both inspiratory and expiratory phasic activity with background tonic activity depending on the study^(21–23). A common finding is that the activity of the levator palatini is more pronounced during oral compared to nasal breathing. The musculus uvula has a similar respiratory modulation and its activation influences the shape of the soft palate and thereby may function to improve closure of the retropalatal oropharynx during such actions as oral breathing and swallowing. However there are no available data on whether the musculus uvula is modulated by negative airway pressure or alterations in blood gas tensions whereas the levator palatini has been shown to increase its activity in response to both stimuli^(24–26).

In contrast, the palatoglossus and palatopharyngeus both act to open the nasal airway while also pulling the palate onto the base of the tongue, which tends to constrict or close the oral airway. Animal and human studies both suggest that these muscles have predominantly inspiratory phasic activity that is enhanced during nasal versus oral breathing^(27, 28). Both muscles rapidly increase their firing in response to brief pulses of negative pressure or inspiratory resistive loading⁽²⁶⁾, and one study suggests that the palatoglossus increases its phasic activity in response to hypercapnia⁽²⁸⁾. Finally, the tensor palatini, which is probably the second most studied upper airway muscle, acts to stiffen the palate also facilitating nasal ventilation. However, available evidence regarding its respiratory modulation is highly variable and differs between human and animal investigations. In humans, multiunit studies have shown that the tensor palatini is tonically activated and is not modulated with respiration⁽²⁹⁾, although some data suggest that the muscle may be phasically active during inspiration^(1, 30). In contrast, studies in the dog suggest both an inspiratory and expiratory phasic pattern⁽²²⁾. Nevertheless, the tensor palatini has been shown to increase its activity in response to negative pressure pulses, while the responses to hypercapnia and hypoxia remain inconsistent.

Muscles Controlling Hyoid Bone Position

There are six muscles that attach to the hyoid bone and are responsible for controlling its position. Working together, the function of these muscles is to pull the hyoid bone anteriorly and caudally, thereby dilating the airway. More specifically, activity of the geniohyoid, mylohyoid, and digastric muscles pulls the hyoid bone in the anterior direction, stylohyoid activity pulls the bone posteriorly whereas the sternohyoid, omohyoid, and thyrohyoid exert their force in the inferior direction. The geniohyoid is the best characterized of all these muscles in terms of its modulation during respiration. Apart from one recent report⁽³¹⁾, most human and animal studies have shown that the geniohyoid demonstrates strong inspiratory phasic activity (with tonic expiratory activity) during wakefulness^(32–35). This activity increases in response to hypercapnia and negative pressure⁽³⁵⁾. Furthermore, increases in the volume of the upper airway are associated with a shortening of this muscle suggesting it is minimally involved in respiration but can influence the size and patency of the upper airway⁽³⁶⁾. Unfortunately, there is a paucity of data on the respiratory activity of the other five muscles controlling hyoid position during wakefulness. The available evidence suggests that these muscles display similar inspiratory phasic activity that increases in response to negative pressure and CO₂ as reported in the geniohyoid.

Pharyngeal Constrictor Muscles

There are three pharyngeal constrictor muscles, the superior, middle, and inferior, which all act to constrict the pharyngeal airway primarily to assist with swallowing. However, there are limited data available addressing their respiratory modulation under resting conditions. The available data either suggest that there is no modulation or, if present, that the activity is primarily during the expiratory phase of the respiratory cycle⁽²³⁾. Although little is known about whether the pharyngeal constrictor muscles are stimulated by negative pressure, stimulation by hypoxia or hypercapnia results in the emergence of strong phasic expiratory activity in both human and animal studies^(23, 37–40). Interestingly, experimental data in animals suggest that the activity of these muscles constricts the airway and reduces its diameter at normal and high low volumes. However, at very low lung volumes they may actually open the airway^(38, 41).

Sleep Effects on Pharyngeal Dilator Muscle Activity/Control

Sleep is divided into two states; NREM (non-rapid eye movement) and REM (rapid eye movement) sleep, with NREM sleep being further subdivided into stages N1, N2 and N3 corresponding to increasing depth of sleep. Importantly, sleep onset is met with a reduction in respiratory drive and a concomitant increase in resistance of the upper airway^(42–44) which thus increases the propensity towards upper airway collapse. Much of the work investigating the influence of sleep on pharyngeal muscle control and patency in humans has focused primarily on the genioglossus muscle and to a lesser extent the tensor palatini, with the majority of this work being completed in NREM sleep. This is likely due to the fact that these two muscles are relatively easy to access when compared to other pharyngeal muscles and both are considered to be important in maintaining pharyngeal patency. As such, this section will focus specifically on the effect of NREM sleep on these two muscles.

There is a multitude of evidence from multiunit recordings showing that genioglossal muscle activity falls shortly after sleep onset⁽³⁾. In addition, evidence from single motor unit recordings suggest that the decrement in activity is primarily the product of a complete drop out of inspiratory phasic and inspiratory tonic units, either immediately at sleep onset or shortly thereafter⁽²⁾. Interestingly, it is the inspiratory phasic motor units that are primarily recruited at arousal from sleep⁽⁴⁵⁾. However, tonically active motor units tend to maintain their firing frequency at sleep onset illustrating that the control of the genioglossus

is quite complex. The mechanism underlying the initial reduction in activity at sleep onset has been somewhat difficult to elucidate as there are a number of variables that change simultaneously. Firstly, one explanation may be the loss of the 'wakefulness' drive as the genioglossus muscle is influenced by a number of sleep-sensitive neural systems including the serotonergic (raphe) and noradrenergic (locus coeruleus) neurons^(46, 47). Both of these neuromodulators are excitatory to the muscle and the cells decrease their firing frequency during sleep. Second, sleep onset is associated with a decrease in drive to the respiratory pump muscles⁽¹⁾, which may consequently reduce drive to the muscle. Thirdly, such a reduction in respiratory drive would also though lower intra-pharyngeal negative pressure swings, and thereby diminish the reflex input to the genioglossus. However, when respiratory drive is completely abolished by allowing an individual to fall asleep during non-invasive mechanical ventilation, there is still a decrease in genioglossus activity⁽⁴⁸⁾. This evidence strongly suggests that wakefulness *per se*, independent of any respiratory or mechanical stimuli, has a major influence of the activity of the genioglossus.

Several studies using multiunit recordings have shown that the ability of the genioglossus muscle to respond to negative pressure and hypercapnia are somewhat diminished during sleep when compared to wakefulness^(9, 49, 50). However, the muscle does not completely lose its ability to react to both airway pressure and rising CO₂. Following sleep onset, the activity of the genioglossus begins to rise again often reaching activity levels similar to that during wakefulness when stable sleep is reached⁽¹⁾. This increase in activity is attributed to a compensatory response to the combined increase in pharyngeal resistance (generating larger negative pressure swings in the airway) and elevations in CO₂ that occur following during sleep, rather than the effect of sleep itself. This neuro-compensatory response is consistent with the idea that genioglossus activity is important in an attempt to maintain a patent airway during sleep⁽⁵¹⁾.

Multiunit recordings from the tensor palatini and a number of other pharyngeal muscles, demonstrate a substantial reduction in activity at sleep-onset and this decrement is often associated with an increase in pharyngeal airflow resistance. However, in contrast to the genioglossus, the fall in activity that occurs in the tensor palatini transpires gradually as sleep progresses from stage N1 through to N3^(29, 51). A similar sleep-related decline in the level of tonic muscle activity has also been described in the geniohyoid⁽³⁴⁾, although inspiratory activity remained preserved. Furthermore, although the tensor palatini can increase its activity in response to respiratory stimuli during sleep, it does not seem to be as sensitive as the genioglossus. As a result, tensor palatini activity is commonly quite low during sleep despite elevations in both upper airway resistance and PCO₂. Overall, the available data suggest that the drive to several pharyngeal muscles is reduced during sleep as well as the responsiveness to both chemical and mechanical stimuli.

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is a condition present in at least 4% of adult men and 2% of adult women⁽⁵²⁾ and is characterized by repetitive collapse (apnea) or partial collapse (hypopnea) of the pharyngeal airway during sleep^(53–56). Such obstructions often lead to increasing respiratory efforts until the airway re-opens and breathing is restored, often producing respiratory effort-related arousals from sleep (see Figure 3). These transient events are often associated with intermittent hypoxia and hypercapnia, large swings in intrathoracic pressure as well as surges in sympathetic activation. As a result, patients with OSA are often sleepy, have decreased quality of life, and may have an increased risk of adverse cardiovascular events including stroke, heart attack, heart failure, and even death.

Although the exact mechanisms responsible for OSA are incompletely understood, an anatomically small pharyngeal airway is a common characteristic of most patients with OSA⁽⁵⁷⁾. While obesity in individuals with OSA is a common factor responsible for small calibre airways, bony structures that crowd and reduce airway size, or in the case of children, large tonsils and adenoids may also contribute⁽⁴⁾. However, most individuals with OSA have little problems breathing while awake, almost always maintaining ventilatory rates, volumes and blood gas tensions within a normal range. This suggests that there are mechanism(s) that compensate for the poor pharyngeal anatomy during wakefulness. Indeed, the increased upper airway resistance caused by an anatomically small airway leads to reflex activation of negative pressure mechanoreceptors located in the larynx, ultimately resulting in increased activity in a number of upper airway muscles including the genioglossus and tensor palatini^(58, 59). Furthermore, the elevated genioglossus activity can be reduced by splinting of the upper airway open with CPAP, thereby removing some of the resistance^(3, 60). Thus, the available evidence suggests that during wakefulness, there is a neuromuscular compensation to the pharyngeal dilator muscles of OSA patients, primarily because their collapsible airways require increased levels of muscle activation in to maintain a patent airway. Unfortunately the basic neural mechanisms driving this compensation are not well understood.

At sleep onset, the activity of the pharyngeal dilator muscles is reduced in OSA patients^(3, 61). This fall in muscle activity also coincides with a marked increase in airway resistance that often results in a reduction or complete loss of patency in the pharyngeal airway yielding a hypopnea or apnea. As a consequence, PO_2 begins to fall and PCO_2 rises which both act to stimulate respiratory drive. Although during this time, the activity of the genioglossus increases in response to the increasing negative airway pressure and changing blood gas tensions, it is often insufficient to reopen the airway⁽⁶²⁾. In general, arousal from sleep secondary to the augmented ventilatory drive is required to reopen the airway⁽⁶³⁾. Once the airway patency is restored and the blood gas disturbances are restored, the individual falls back asleep and the process repeats itself.

If the mechanisms described above were the total explanation for apnea pathogenesis, then variability in airway anatomy should explain apnea severity. The most common way that anatomy is assessed is by measuring the 'passive' critical pharyngeal closing pressure (P_{crit}), of the upper airway which is illustrated in Figure 4A. A value above 0 cm H_2O indicates very poor anatomy with airway closure above atmospheric pressure whereas a value below -4 or -5 cm H_2O indicates that the individual should not have sleep apnea. A P_{crit} between 0 and -5 cm H_2O represents a grey zone with some patients having obstructive apneas and others not. Thus, apnea pathophysiology must be more complex⁽⁴⁾ than a simple anatomic abnormality with anatomy of the upper airway only accounting for a small proportion (see Figure 4B) of the observed variability in apnea severity⁽⁶⁴⁾.

To date, a number of other variables have been identified as important contributors to the pathogenesis of sleep apnea. First, the ability of the upper airway muscles to respond to a respiratory challenge and reopen the airway is one such factor. Current data suggests that some individuals are more sensitive to the negative pressure reflex control of pharyngeal dilator muscle activity during sleep than others and are thus better able to compensate for their deficient anatomy during sleep⁽⁶²⁾. Furthermore, using a variation of the P_{crit} technique which assesses the dynamic neuromuscular control of the upper airway, Patil et al⁽⁶⁵⁾ found that the ability of the upper airway muscles to adequately compensate for increased mechanical loads and stabilize airway patency during sleep is attenuated in OSA patients when compared to healthy controls. Second, some individuals are able to stay asleep longer in the face of increasing respiratory stimulation (often referred to as a high respiratory arousal threshold). A high arousal threshold allows sufficient time for the

pharyngeal muscles to be recruited and effectively compensate for a deficient anatomy, whereas a low arousal threshold would not. Third, the stability of the ventilatory control system (often described by loop gain; LG) is also an important contributor to the pathophysiology of obstructive sleep apnea (OSA) in some patients^(66, 67). A high LG can cause large fluctuations in respiratory drive which are associated with parallel swings in neural drive to the respiratory and pharyngeal muscles. Such fluctuations in drive increase the propensity to collapse at the nadir of respiratory drive. Furthermore, a high LG will also lead to large increases in respiratory drive during an apnea or hypopnea often promoting respiratory effort-related arousals from sleep. Finally reductions in lung volume that occur at sleep onset and throughout the night may play a role in OSA^(68, 69), as such decrements can reduce the longitudinal tension on the upper airway increasing its collapsibility. Importantly, a major factor limiting our current understanding of OSA pathogenesis is that no study to date has measured all these variables in a single individual or a group of individuals with and without sleep apnea. Thus, the aim of future research is to better understand how these traits interact to produce OSA in individual patients, and how this knowledge may allow for individualization of care.

Summary and Future Directions

In summary, all four groups of pharyngeal muscles can work together in a highly coordinated fashion to maintain and protect the patency of the airway thereby allowing unencumbered respiration during wakefulness. Electromyographic recordings have demonstrated that many of the pharyngeal muscles demonstrate respiratory modulation, most commonly by increased firing during inspiration while maintaining tonic activity during expiration. Many of these muscles can also respond to respiratory (rising PCO₂ or falling PO₂) and local stimuli (negative pressure). Single motor unit recordings from these muscles indicate multiple and complex activation patterns, with motor unit recruitment instead of increased firing frequency, being the primary response to stimulation. During sleep, the activity of the pharyngeal muscles is generally reduced, decreasing the size of the airway. In response to the associated increasing negative pressure swings and CO₂ these muscles are stimulated and restore airway patency in healthy individuals. However, this does not always occur in obstructive apnea patients. Although there are many factors that contribute to the development of obstructive sleep apnea, the variable ability of these muscles to reopen the airway is certainly one. Others important factors include a poor pharyngeal anatomy, a low respiratory arousal threshold, a high loop gain and a low lung volume. Future research is needed to develop methods to reliably measure each of these variables or traits to understand their role in apnea pathogenesis in the individual patient.

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Figure 1. The anatomy and musculature of the human pharyngeal airway

A simplified diagram illustrating several of the important pharyngeal muscles involved in maintaining a patent airway. Panel A is a mid-sagittal view of the human upper airway and illustrates the 4 distinct anatomical subdivisions: the nasopharynx, retropalatal oropharynx (originally termed velopharynx), retroglossal oropharynx (originally termed oropharynx), and the hypopharynx. Panel B is a parasagittal view. From Kryger et al⁽⁷⁰⁾; reprinted with permission.

Figure 2. Single motor unit discharge patterns in the genioglossus

Examples of the six discharge patterns found in the genioglossus (panel A–F). Each panel shows the raw electromyograph (EMG), the instantaneous discharge frequency plot for the single motor unit and the tidal volume (in litres) for two breaths. From Saboisky et al. (2006) *Journal of Neurophysiology*, Am Physiol Soc, used with permission⁽¹⁵⁾.

Figure 3. Example of Obstructive Sleep Apnea

An example taken from a clinical polysomnogram in a patient with severe OSA. Note that despite the repeated respiratory efforts to breathe (thoracic and abdominal bands), there is no nasal airflow indicating the airway has become obstructed. Such obstructions are often associated with repeated oxygen desaturations and arousals from sleep. Abbreviations: EEG, electroencephalogram; EMG, electromyogram; EKG, electrocardiogram; SaO₂, arterial blood oxygen saturation.

Figure 4. Determination of Pcrit and its relationship to AHI

A) Illustrative example of how the passive critical pharyngeal closing pressure (Pcrit) is determined. The Pcrit is determined by intermittently lowering CPAP from the subject's therapeutic pressure to progressively lower levels until zero flow occurred. The peak flow from the 3rd–5th flow-limited breaths after a pressure drop are then plotted against mask pressure and are fit with a straight line; the x intercept of this line (zero flow crossing) was taken as the Pcrit. In this example the Pcrit is 0.5cm H₂O. B) Data illustrating the poor relationship between Pcrit and AHI. From Sforza et al⁽⁶⁴⁾; Reprinted with permission of the American Thoracic Society. Copyright © American Thoracic Society.

Table 1

Summary table of the locations, innervations and actions of the pharyngeal muscles

Muscle group	Muscle	Origin	Insertion	Efferent Nerve Innervation	Primary action
Extrinsic Muscles of the tongue	Genioglossus	Mental spine of the mandible	Base of the tongue	Hypoglossal (medial branch)	Protrudes (and depresses) the tongue
	Styloglossus	Styloid process of the temporal bone and stylomandibular ligament	Base of the tongue	Hypoglossal (lateral branch)	Raises and retracts the tongue
	Hyoglossus	Greater horn of the hyoid bone	Base of the tongue	Hypoglossal (lateral branch)	Depresses and retracts the tongue
Muscles controlling palatal shape and position	Tensor palatini	Variable combination of the cranial base and auditory tube cartilage	Palatal aponeurosis	Trigeminal (mandibular branch)	Stiffens the soft palate
	Levator Palatini	Temporal bone of the skull	Palatal aponeurosis	Vagus (pharyngeal branch)	Raises the soft palate
	Palatoglossus	Palatal aponeurosis	Base of the tongue	Vagus (pharyngeal branch)	Elevates tongue and pulls palate down onto the posterior tongue
	Palatopharyngeus	Palatal aponeurosis	Lateral pharyngeal wall	Vagus (pharyngeal branch)	Lifts the pharyngeal wall and may move the soft palate anteriorly
	Muscular Uvula	Soft palate	uvula	Vagus (pharyngeal branch)	Raises uvula
Muscles influencing hyoid bone position	Geniohyoid	Genial tubercle of mandible	Body of the hyoid bone	Hypoglossal	Elevates and draws the hyoid forward
	Mylohyoid	Mylohyoid line of mandible	Body of hyoid bone and median raphe	Trigeminal (mandibular branch)	Elevates hyoid bone and supports the floor of the mouth
	Thyrohyoid	Lamina of thyroid cartilage	Greater cornu of hyoid bone	Cervical (first branch)	Raises and changes the form of the larynx
	Stemohyoid	Manubrium and clavicle	Body of hyoid bone	Ansa cervicalis	Depresses hyoid bone and larynx
	Stylohyoid	Styloid process	Body of hyoid bone	Facial nerve	Draws the hyoid and tongue superiorly and posteriorly
	Omoxyoid	Upper border of the scapula	Body of hyoid bone	Ansa cervicalis	Depresses/stabilizes hyoid bone
Pharyngeal constrictor muscles	Superior	All of these muscles originate from an	Variety of structures in the anterior pharynx including the tongue	Pharyngeal plexus from both the vagus and glossopharyngeal nerve	Similar effect in all three muscles: Thought to close the airway as part of swallowing. Also has a mechanical effect that depends on lung volume. At high lung volumes they act as
	Middle			Pharyngeal plexus from both the vagus and glossopharyngeal nerve	
	Inferior			Pharyngeal plexus from both the vagus and glossopharyngeal nerve	

Muscle group	Muscle	Origin	Insertion	Efferent Nerve Innervation	Primary action
		aponeurosis in the posterior midline			constrictors whereas at low lung volumes they act as dilators.