The Interplay between Tongue Tissue Volume, Hyoid Position, and Airway Patency

Commentary on Genta et al. Upper airway collapsibility is associated with obesity and hyoid position. SLEEP 2014;37:1673-1678.

Jason P. Kirkness, PhD; Mudiaga Sowho, MD, MPH; Emi Murano, MD, PhD

1Johns Hopkins Sleep Disorders Center, Division of Pulmonary and Critical Care Medicine, Johns Hopkins School of Medicine, Baltimore, MD; 2Department of Otolaryngology–Head and Neck Surgery, Johns Hopkins School of Medicine, Baltimore, MD

In this issue of SLEEP, Genta and colleagues report the results of a study examining the association between the factors attributed to obesity and hyoid bone position with the collapsibility of the upper airway.1 Their paper provides novel metrics that are linked to the upper airways anatomic predisposition for collapse, highlighting the interplay between tongue tissue volume, hyoid position, and airway patency. The unresolved issues are whether the static upper airway volume has a functional association with the collapsibility of the airway during sleep and whether treatments aimed to alter hyoid position are indicated.

Genta et al. demonstrate a direct association between pharyngeal length and upper airway collapsibility,2 providing a mechanistic explanation for the relationship between pharyngeal length and severity of sleep apnea.3-4 The propensity for collapse may vary between individuals, since the upper airway extends from the nares and oral cavity to the glottis at the entrance of the larynx, divided into four anatomical regions, each with distinct function: (i) the nasopharynx, (ii) velopharynx, (iii) oropharynx, and (iv) hypopharynx. An intricate arrangement of soft (24 muscles and other soft tissues), cartilaginous (thyroid cartilage, epiglottis), and bony (hard palate, mandible, and hyoid bone) tissues are required to accommodate functions that include heating and humidification of air, respiration, speech, and swallowing.5 The posterior vertebral column is the only fixed source of bony or cartilaginous support for the upper airway, making it highly deformable to enable swallowing and speech. The range of upper airway motion, pharyngeal patency, tongue movement, and swallowing are all enhanced by attachment of the anterior, posterior, and inferior muscles of the ventral neck region to the hyoid bone.6 The main function of the hyoid bone is to suspend and provide longitudinal traction to tracheal, laryngeal, and upper esophageal structures, and provide a central anchor point for the lingual and pharyngeal musculature.8 The flexibility of the upper airway comes at the expense of a high propensity for collapse during sleep, which may lead to sleep disordered breathing.9,10

The hyoid positioning in relation to the mandibular plane and the pharyngeal length in the featured article by Genta provide us a unique insight into the coupling between the hyoid bone topology and the passive mechanical collapsibility of the upper airway. Pharyngeal airway dimensions are the result of the interaction between bony and soft tissues, while obesity, a major risk factor for OSA, may lead to enlargement of upper airway soft tissues, particularly the tongue.11-13 The tongue may enlarge antero-posteriorly and decrease upper airway diameter due to the fixed bony structure. The tongue may enlarge antero-posteriorly decreasing upper airway diameter, and may expand caudally displacing the hyoid downward, consequently increasing pharyngeal length and mandibular plane to hyoid distance (MPH).14-15 The MPH and pharyngeal morphological measurements may be or become valuable OSA biomarkers, as mandibular surgical advancement or prosthesis mandibular repositioning had suggested changes in pharyngeal patency with distinct soft tissue shape. There are demonstrable changes in the structure of the upper airway in patients who develop sleep apnea compared to normal subjects. In general, the changes occur in the soft tissue and bony structures that translate to a smaller airway as well as alterations in shape.16,17 Nevertheless, it should be noted that a smaller airway does not necessarily translate into a more collapsible airway during sleep.18

A number of changes to the upper airway at sleep onset predispose it to collapse and can result in sleep disordered breathing in some individuals. During wakefulness, the upper airway is under both volitional and non-volitional control.19 However, during sleep or anesthesia, the entire upper airway is essentially under non-volitional control.19 At the onset of sleep, there is a fall in upper airway muscle activity,4 which results in decreased upper airway compliance, smaller upper airway lumen size, and increased upper airway resistance.21-23 Furthermore, the pharyngeal neuro-compensatory reflexes, which are triggered by negative intraluminal pressure and ventilatory motor output, due to loss of the wakefulness stimuli, are reduced.24-26 During sleep, it is often difficult to precisely identify whether collapse is due primarily to altered neural or mechanical control or a combination of both factors. However, biomechanical11,17,27 and imaging28 models have aided in determining the various factors that lead to collapse as well as provide the means to dissect the relative contribution of neuromuscular activity to sleep apnea. High-resolution real-time imaging during natural sleep would aid in determining precisely where and how the upper airway collapses.

The primary treatment mode for obstructive sleep apnea is still positive airway pressure.29 Measures available to those who poorly adapt to CPAP include oral appliances that enlarge the airway by way of advancing the mandible, and surgical options such as septoplasty, adenotonsillectomy, uvulopalatopharyngoplasty, mandibular advancement,30 tongue reduction,11 bariatric procedures,32 and most recently hypoglossal pacemakers.33 Hyoid bone suspension procedures have also been described.
and are thought to support lower pharyngeal and supraglottic tissues without the need of major skeletal reconstruction. As with other surgical procedures, success has been variable, and there is controversy as to whether the surgical morbidity is acceptable, since it is usually used in combination with another procedure, such as uvulopalatopharyngoplasty. Since surgical procedures permanently alter the upper airway size and shape, a multidisciplinary assessment should be mandated to determine potential impacts on other upper airway functions such as speech, swallowing, and collapsibility. Genta and colleagues make a case for assessing static upper airway size during sleep as a way to characterize the collapsibility of the upper airway.

If the combination of tongue tissue volume factors and hyoid position are key to determining the passive anatomic characteristic that predisposes to obstructive sleep apnea, it remains to be determined: (a) if hyoid displacement during sleep is associated with increased prevalence of OSA; (b) if active neuromuscular compensatory responses to obstruction are associated with hyoid displacement; (c) if treatment success could be predicted based on hyoid position or displacement; (d) if there are longitudinal effects of weight loss on hyoid displacement relative to upper airway function during sleep; (e) how to best assess the upper airway function relative to the disease state in order to determine treatment methods; and (f) if it is possible to establish a forum to engage the broader community with multidisciplinary expertise in upper airway function.

**CITATION**

**DISCLOSURE STATEMENT**
The authors have indicated no financial conflicts of interest.

**REFERENCES**