EDITORIAL

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[1]; Mudiaga Sowho, MD, MPH[1]; Emi Murano, MD, PhD[2]

[1]*Johns Hopkins Sleep Disorders Center, Division of Pulmonary and Critical Care Medicine, Johns Hopkins School of Medicine, Baltimore, MD;* [2]*Department 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,[1] providing a mechanistic explanation for the relationship between pharyngeal length and severity of sleep apnea.[2-4] 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[5] are required to accommodate functions that include heating and humidification of air, respiration, speech, and swallowing.[6] 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.[7] 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,[8] 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, biomechanical[11,17,27] and imaging[28] 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, adenotonsilectomy, uvulopalatopharyngoplasty, mandibular advancement,[30] tongue reduction,[31] bariatric procedures,[32] and most recently hypoglossal pacemakers.[33] Hyoid bone suspension procedures have also been described,

Submitted for publication August, 2014
Accepted for publication August, 2014
Address correspondence to: Jason P. Kirkness, PhD, Johns Hopkins Sleep Disorders Center, 5501 Hopkins Bayview Circle, Level 4, Asthma & Allergy Building, Baltimore, MD 21224; Tel: (410) 550-0574; Fax: (410) 550-3374; E-mail: jason.kirkness@jhmi.edu

and are thought to support lower pharyngeal and supraglottic tissues without the need of major skeletal reconstruction.[34,35] 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.[36] 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 predispose 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

Kirkness JP, Sowho M, Murano E. The interplay between tongue tissue volume, hyoid position, and airway patency. *SLEEP* 2014;37(10):1585-1586.

DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.

REFERENCES

1. Genta PR, Schorr F, Eckert DJ, et al. Upper airway collapsibility is associated with obesity and hyoid position. Sleep 2014;37:1673-8.
2. Carlisle T, Carthy ER, Glasser M, et al. Upper airway factors that protect against obstructive sleep apnoea in healthy older males. Eur Respir J 2014 May 15. [Epub ahead of print].
3. Segal Y, Malhotra A, Pillar G. Upper airway length may be associated with the severity of obstructive sleep apnea syndrome. Sleep Breath 2008;12:311-16.
4. Shigeta Y, Ogawa T, Tomoko I, Clark GT, Enciso R. Soft palate length and upper airway relationship in OSA and non-OSA subjects. Tex Dent J 2013;130:203-11.
5. Strohl KP. Upper airway muscles of respiration. Am Rev Respir Dis 1981;124:211-13.
6. Bogaerts M, Deggoujf N, Huart C, et al. Physiology of the mouth and pharynx, Waldeyer's ring, taste and smell. B-ENT 2012;8 Suppl 19:13-20.
7. Kim Y, McCullough GH. Maximum hyoid displacement in normal swallowing. Dysphagia 2008;23:274-9.
8. Bogart BI, Ort VH. Elsevier's integrated anatomy and embryology. Mosby, 2007.
9. Brown EC, Cheng S, McKenzie DK, Butler JE, Gandevia SC, Bilston LE. Respiratory movement of upper airway tissue in obstructive sleep apnea. Sleep 2013;36:1069-76.
10. Kirkness JP, Peterson LA, Squier SB, et al. Performance characteristics of upper airway critical collapsing pressure measurements during sleep. Sleep 2011;34:459-67.
11. Shapiro SD, Chin CH, Kirkness JP, et al. Leptin and the control of pharyngeal patency during sleep in severe obesity. J Appl Physiol 2014;116:1334-41.
12. Sutherland K, Lee RW, Phillips CL, et al. Effect of weight loss on upper airway size and facial fat in men with obstructive sleep apnoea. Thorax 2011;66:797-803.
13. Kirkness JP, Schwartz AR, Schneider H, et al. Contribution of male sex, age, and obesity to mechanical instability of the upper airway during sleep. J Appl Physiol 2008;104:1618-24.
14. Chi L, Comyn FL, Mitra N, et al. Identification of craniofacial risk factors for obstructive sleep apnoea using three-dimensional MRI. Eur Respir J 2011;38:348-58.
15. Brown EC, Cheng S, McKenzie DK, Butler JE, Gandevia SC, Bilston LE. Tongue and lateral upper airway movement with mandibular advancement. Sleep 2013;36:397-404.
16. Schwab RJ, Pasirstein M, Pierson R, et al. Identification of upper airway anatomic risk factors for obstructive sleep apnea with volumetric magnetic resonance imaging. Am J Respir Crit Care Med 2003;168:522-30.
17. Schwartz AR, Patil SP, Squier S, Schneider H, Kirkness JP, Smith PL. Obesity and upper airway control during sleep. J Appl Physiol 2010;108:430-5.
18. Mohsenin V. Effects of gender on upper airway collapsibility and severity of obstructive sleep apnea. Sleep Med 2003;4:523-9.
19. Horner RL. Neural control of the upper airway: integrative physiological mechanisms and relevance for sleep disordered breathing. Compr Physiol 2012;2:479-535.
20. Fogel RB, Trinder J, White DP, et al. The effect of sleep onset on upper airway muscle activity in patients with sleep apnoea versus controls. J Physiol 2005;564:549-62.
21. Ayappa I, Rapoport DM. The upper airway in sleep: physiology of the pharynx. Sleep Med Rev 2003;7:9-33.
22. Finkelstein Y, Wolf L, Nachmani A, et al. Velopharyngeal anatomy in patients with obstructive sleep apnea versus normal subjects. J Oral Maxillofac Surg 2012;72:1350-72.
23. Strohl KP, Butler JP, Malhotra A. Mechanical properties of the upper airway. Compr Physiol 2012;2:1853-72.
24. McGinley BM, Schwartz AR, Schneider H, Kirkness JP, Smith PL, Patil SP. Upper airway neuromuscular compensation during sleep is defective in obstructive sleep apnea. J Appl Physiol 2008:105:197-205.
25. Tomori Z, Donic V, Benacka R, Kuchta M, Koval S, Jakus J. [Regulation of respiration and its sleep-related disorders]. Sb Lek 2002;103:65-71.
26. Bilston LE, Gandevia SC. Biomechanical properties of the human upper airway and their effect on its behavior during breathing and in obstructive sleep apnea. J Appl Physiol 2014;116:314-24.
27. Hoshino Y, Ayuse T, Kobayashi M, et al. The effects of hormonal status on upper airway patency in normal female subjects during propofol anesthesia. J Clin Anesth 2011:23:527-33.
28. Sutherland K, Schwab RJ, Maislin G, et al. Facial phenotyping by quantitative photography reflects craniofacial morphology measured on magnetic resonance imaging in Icelandic sleep apnea patients. Sleep 2014;37:959-68.
29. Sowho MO, Woods MJ, Biselli P, McGinley BM, Buenaver LF, Kirkness JP. Nasal insufflation treatment adherence in obstructive sleep apnea. Sleep Breath 2014 Jul 12. [Epub ahead of print].
30. White DP, Shafazand S. Mandibular advancement device vs. CPAP in the treatment of obstructive sleep apnea: are they equally effective in short term health outcomes? J Clin Sleep Med 2013;9:971-2.
31. Sorrenti G, Piccin O, Scaramuzzino G, Mondini S, Cirignotta F, Ceroni AR. Tongue base reduction with hyoepiglottoplasty for the treatment of severe OSA. Acta Otorhinolaryngol Ital 2004;24:204-10.
32. Ravesloot MJ, Hilgevoord AA, Van Wagensveld BA, DeVries N. Assessment of the effect of bariatric surgery on obstructive sleep apnea at two postoperative intervals. Obes Surg 2014;24:22-31.
33. Eastwood PR, Barnes M, Walsh JH, et al. Treating obstructive sleep apnea with hypoglossal nerve stimulation. Sleep 2011;34:1479-86.
34. Cillo JE Jr, Dalton PS, Dattilo DJ. Combined elliptical window genioglossus advancement, hyoid bone suspension, and uvulopalatopharyngoplasty decrease apnea hypopnea index and subjective daytime sleepiness in obstructive sleep apnea. J Oral Maxillofac Surg 2013;71:1729-32.
35. Randerath WJ, Verbraecken J, Andreas S, et al. Non-CPAP therapies in obstructive sleep apnoea. Eur Respir J 2011;37:1000-28.
36. Karatayli-Ozgursoy S, Demireller A. Hyoid suspension surgery with UPPP for the treatment of hypopharyngeal airway obstruction in obstructive sleep apnea. Ear Nose Throat J 2012;91:358-64.