THE PHARYNGEAL AIRWAY IS A complicated structure combining soft tissue and boney elements that make it prone to collapse when neuromuscular activity wanes during sleep. Anatomic structures form a pliable conduit capable of changing airway patency dynamically to support its respiratory, alimentary, and vocal functions. Respiratory function is supported by active neuromuscular and passive structures that tether and stretch the airway open. In the current issue of the Journal of Applied Physiology, Owens et al. (5) propose novel methods for determining how structures that dilate and lengthen the pharynx contribute to the maintenance of patency during inspiration.

Early efforts to model upper airway collapsibility concentrated primarily on factors regulating its transmural pressure, which is defined by the pressure difference across the airway lumen. Initially, intraluminal "suction" pressures generated during inspiration were thought to play a critical role in the pathogenesis of pharyngeal collapse and occlusion (6). Subsequently, investigators demonstrated that downstream suction pressure merely caused the pharynx to flow-limit during inspiration but not occlude (9). Rather, occlusion could be induced by lowering the upstream pressure at the nose, which occurred as the transmural pressure fell to zero. In fact, the nasal pressure required to occlude the pharynx could be determined experimentally in sleeping subjects. This pressure, also known as the pharyngeal critical pressure (Pcrit), was taken to represent the pressure exerted on the pharyngeal lumen by surrounding tissues (10). The anatomic determinants of variations in tissue pressure, however, remained largely undefined.

The effects of airway anatomy on peripharyngeal tissue pressure have been modeled to consider the effect of boney structures that enclose the airway in a box (16). As extraluminal tissue pressure is increased within the boney enclosure, the pharyngeal transmural pressure would decrease and the airway collapsibility (Pcrit) would increase accordingly (see Figure 1) (8). In theory, such increases could result from decreases in the size of the "box" due to anatomic alterations such as microagnathia, a high arched hard palate, or mid-face hypoplasia, which increase airway collapsibility. Alternatively, collapsibility can increase by "stuffing" more soft tissue inside the box, as might be the case when macroglossia, adenotonsillar hypertrophy, or central adiposity are present. According to this view, airway patency and the surrounding tissue pressure are determined by the sum of radial forces around the pharyngeal lumen exerted by boney and soft tissue structures.

However, pharyngeal collapsibility (Pcrit) may not simply be determined by surrounding tissue pressures. In early studies in isolated upper airway preparations, Van de Graff (14) demonstrated that caudal tracheal traction decreased pharyngeal airflow resistance markedly. This effect was evident even after cervical linkage with thoracic structure was severed, suggesting that longitudinal tension within the airway "wall" served to stiffen the airway and prevent its collapse. Thut et al. further quantified effects of caudal traction and established that pharyngeal collapsibility (Pcrit) decreased markedly with minimal increases in caudal airway traction (13). Roweley et al. (7) further explored the interaction between radial and longitudinal forces on the airway and determined that caudal traction augmented responses to radial traction, leading to speculation that elongating the airway increased its "wall" tension. Stiffening of the airway wall increased its ability to withstand the compressive effects of surrounding tissues. More recent evidence from Karaitis et al. (4) suggested that caudal tracheal traction can also compress the surrounding tissues, effectively removing tissue from the "box." Thus evidence from animal models suggests that caudal traction can decrease pharyngeal collapsibility by increasing wall tension and decreasing surrounding tissue pressures (Fig. 1).

Understanding the impact of longitudinal tension on airway collapsibility has clinical implications, which stem from early observations that lung inflation increases caudal traction on upper airway structures (15). Increases in lung volume can dilate the pharynx (1), decrease its collapsibility (11), and ameliorate obstructive sleep apnea (2). Effects of lung volume on pharyngeal mechanics have been gleaned from measurements of pharyngeal collapsibility during sleep at different levels of end-expiratory lung volume. In these experiments, end-expiratory lung volume was varied by manipulating the pressure at the body surface with subjects sleeping in an "iron lung" (Portalung). A 1-liter decrease in lung volume was associated with an ~2-cmH2O increases in Pcrit per liter (11) without alterations in pharyngeal compliance (12). This effect may mediate the well-recognized increase in sleep apnea susceptibility in obesity, which generally decreases functional residual capacity (3). The physiologic basis for this lung volume response, however, remains largely unexplored. Conducting studies on fully instrumented subjects sleeping in a Portalung is especially challenging and significantly limits the numbers of participants available for study.

In the current issue of the Journal of Applied Physiology, Owens et al. (5) offer a clever alternative to testing sleeping subjects in the Portalung. These investigators reasoned that alterations in lung volume can confound measurements of Pcrit, which is derived by measuring airflow at various levels of nasal pressure. Instead, they devised a new method for holding end-expiratory lung volume constant during the measurement of Pcrit. They varied nasal pressure during inspiration and expiration separately with a bilevel ventilator and maintained a constant level of end-expiratory pressure and lung volume. They found that breath by breath variability in Pcrit during step reductions in nasal pressure were abolished by eliminating concomitant decreases in end-expiratory lung volume. Their method provides definitive evidence for the influence of lung volume on measurements of upper airway collapsibility and offers the potential for characterizing lung volume responses in airway collapsibility across a spectrum of sleep apnea risk factors and disease state.

Methodologic and physiologic insights from Owens et al. will enable investigators to characterize effects of radial and longitudi-
Fig. 1. Effects of longitudinal airway traction on pharyngeal surrounding pressures within the boney enclosure (see box, dashed lines). Caudal traction decreases compressive forces (in left compared with right panel). [Adapted from Schwab et al. (Ref. 8, an official journal of The American Thoracic Society) and reprinted with permission of the American Thoracic Society (c) 2003 American Thoracic Society.]

Invited Editorial

dinal tension on airway collapsibility in humans. Their streamlined methods confine the subject’s exposure to nasal pressure challenges for only five breaths. Their method will facilitate throughput in protocols investigating whether alterations in lung volume mediate increases in sleep apnea susceptibility with increased age and weight and in men and postmenopausal women. Finally, the method could be used to determine whether lung volume responses are blunted in sleep apnea compared to matched normal subjects. Studies such as these would add immeasurably to our ability to model and elucidate the mechanical factors controlling pharyngeal collapsibility during sleep.

Having maintained a constant lung volume over a series of breaths, Owens et al. have utilized their method to enhance our understanding of negative effort dependence, a phenomenon characterized by a roll-off in flow with increasing degrees of inspiratory effort. When present, airflow obstruction can worsen progressively during a single inspiration and across of sequence of flow-limited breaths, leading to further decreases in ventilation and respiratory instability during sleep. Utilizing their method, Owens et al. established that negative effort dependence cannot be abolished by holding lung volume constant. It is still unclear whether observed decreases in airflow are related to increases in tracheal tug that accompany increases in inspiratory effort. Further work will be required to determine the precise mechanism for this response.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


REFERENCES


