Upper airway surface tension: is it a significant cause of airflow obstruction during sleep?

Obstructive sleep apnea is characterized by repetitive episodes of upper airway obstruction during sleep, leading to oxyhemoglobin desaturation, recurrent arousals, and long-term neurocognitive, metabolic, and cardiovascular dysfunction. It is widely recognized that this disorder is due to alterations in the control of upper airway patency during sleep (16). Among the factors leading to upper airway collapse and airflow obstruction, alterations in airway structures and/or disturbances in neuromuscular control are thought to play major roles (6, 12). Recent work by Kirkness and others (8–11) suggests that surface tension plays a role in modulating upper airway patency and may also be a marker for disturbances in airway neuromuscular control.

A number of structural alterations are thought to compromise airway patency and predispose to increasing degrees of airflow obstruction during sleep. Bulk changes in soft tissue mass, anatomic narrowing by bony structures, and decreases in axial tension have all been associated with reductions in upper airway patency in animals and humans (17, 19). Each of these factors leads to a loss in forces that either tether upper airway structures open or stretch the luminal surface longitudinally, making it more resistant to collapse (17). Structural defects caused by microagnathia, fatty deposits, soft tissue infiltration, and the loss of caudal trachea traction have all been demonstrated to increase upper airway collapsibility, resistance, compliance, or luminal cross-sectional area (1).

Recent studies by Kirkness and coworkers (8–11) have now added surface tension in upper airway lining fluid to the list of structural factors that influence airway patency. In an extensive series of experiments, these investigators have systematically explored the effects of surface-acting forces on upper airway function in animals and humans. Their work was prompted by earlier observations that surfactant might help restore upper airway patency and reduce collapsibility in animals (13) and awake humans (18). Kirkness and coworkers (8) initially developed an elegant technique for measuring surface tension in scant amounts of upper airway lining fluid and demonstrated a strong correlation between changes in upper airway collapsibility and surface tension induced by surfactant and saline instillation. In studies in anesthetized rabbits and humans, they demonstrated that surfactant improved upper airway patency, as reflected by decreases in critical opening pressures and upstream resistance, and that it decreased the hysteresis between upper airway opening and closing pressures (9, 10).

The authors’ most recent study, which appears in this issue of the Journal (see Ref. 11), extends their work in anesthetized rabbits and humans to the sleeping human with intact airway neuromuscular control mechanisms. Examining the effect of surfactant instillation, they demonstrated significant decreases in the surface tension of pharyngeal lining fluid, upper airway collapsibility, and sleep apnea severity (respiratory disturbance index). Observed improvements in sleep apnea severity were primarily related to decreases in hypopneas in their patient group, which correlated with reductions in surface tension. This study provides firm evidence that surface tension plays a role in the regulation of airway patency and may influence sleep apnea severity.

An intriguing finding was that improvements in the sleep apnea severity correlated with the decrease in surface tension after surfactant instillation. This relationship is consistent with the notion that surface tension may vary substantially between individuals and may either predispose to or protect against sleep apnea. In fact, surface tension can vary over a relatively wide range, from 50 to 90 mN/m among study subjects (10). Although the mechanism for variability in surface tension is unclear, differences in salivary flow, route of breathing, and/or pharyngeal mucosal properties may play a role. Recurrent mucosal trauma from continuous snoring and inspiratory flow limitation during sleep may dry the mucosa excessively and adversely elevate surface tension. Thus a vicious circle of decreasing surface tension leading to worsening airflow obstruction, mucosal trauma, and further increases in sleep apnea severity may result. Moreover, dessication of the pharyngeal lining may account for alterations in local sensory receptors, which have recently been demonstrated to play a role in maintaining airway patency (7).

Is there a role for measurements of surface tension and surfactant instillation in the management of sleep apnea? The effects of surfactant can be gauged from critical pressure measurements pre- and postinstillation. The findings of Kirkness et al. (11) demonstrate that critical pressures can decrease by 2–3 cmH2O after surfactant administration. Relatively modest reductions in critical pressure of this magnitude would be expected to decrease hypopnea frequencies but have little effect on apnea frequencies (5), as confirmed by these authors. Decreases in critical pressure up to 3 cmH2O are comparable to those previously observed with changes in body position (2, 14) and might represent an adjunct to therapy for sleep apnea patients. Moreover, decreases in critical pressure of this magnitude should be effective in relieving upper airway resistance syndrome and/or diminishing snoring intensity in those without sleep-disordered breathing (3, 4).

Finally, measurements of upper airway surface tension might guide clinicians in deploying surfactant therapy and might be used as a marker for mucosal injury and
sensory receptor dysfunction in this disorder (15). Further work is required to correlate surface tension in upper airway lining fluid with altered structural changes and sensorineural defects in upper airway control.

REFERENCES


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