Can we breathe and swallow at the same time?

The article by Samson et al. (7) in this issue of the Journal of Applied Physiology raises new issues concerning the regulation of nonnutritive swallowing (NNS) and breathing during sleep. The past 10 years have seen a large increase in interest in this field (2, 3). Clearly, NNS is essential for survival. It has been estimated that the combined volume of oral and nasal secretions is well in excess of 2 liters each day. Furthermore, clinical experience has shown that, in the absence of competent NNS, the lungs rapidly fill with these secretions, producing death within a few days.

It has been acknowledged that neural mechanisms regulating precise coordination of swallowing and breathing are critically important considering that the pharynx serves as a conduit for air going to and from the lungs and for fluids going to the stomach. This dual pharyngeal function is the basis for the frequency encountered pulmonary aspiration syndromes in clinical medicine (5, 8). Coordination of breathing and swallowing is especially important during sleep when volitional swallowing is absent. It is noteworthy that the NNS mechanism is often frequently imperfect because most of healthy individuals experience nightly aspiration of pharyngeal secretions during sleep (4).

In mechanical terms, the function of NNS is analogous to that of a basement sump pump. As pharyngeal fluids accumulate a critical volume is eventually reached such that a regulating mechanism must initiate a swallow to avoid flooding the lungs. It now is clear that the laryngeal chemoreflex is mediated by neural receptors concentrated in the interarytenoid notch. Furthermore, the laryngeal chemoreflex appears to be the principal mechanism that initiates a NNS (6). Again, analogous to a sump pump, it has been suggested that accumulating secretions in the piriform fossae periodically reach a critical volume (6). When this volume is reached, the pooled secretions come into contact with the inner aryteroid receptors, a NNS occurs, and the fossae are emptied. Although each of the various mechanisms envisioned by Samson et al. (7) are potentially relevant, it would seem that the most straightforward explanation for the effect of positive nasal pressures in decreasing NNS would be an increased pharyngeal storage capacity for secretions resulting from dilation of the piriform fossae. Assuming the rate of secretion production remained constant, this would result in decrease swallowing frequency.

What of the integration of swallowing and breathing? Samson et al. (7) have provided new information here. They have shown that, unlike the adult, neonatal NNS can occur at any time during inspiration or expiration. This finding also true of human infants, in which case the diaphragm continues to contract during a swallow (9). Therefore, except for a slight prolongation of inspiration and expiration during a swallow, infants, at least, do not appear to coordinate timing of breathing and swallowing at all. This makes physiological sense because the critical volume for a NNS is likely not dependent on respiratory phase, and so aspiration must be avoided during inspiration since inspiration from time to time will coincide with the need to swallow. In this case adequate protection from aspiration is ensured by an obligatory glottic closure during the swallow (1, 9).

Also of interest is the observation of Samson et al. (7) of an excessive accumulation of gastric air during positive pressure breathing. This is potentially relevant to another aspect of the coordination of NNS and breathing. A brief diaphragmatic contraction has been noted to occur just before the onset of the pharyngeal muscle contraction that propels a pharyngeal fluid bolus into the esophagus (9). At this time, both the superior nasopharynx and inferior pharyngeal valves and the superior esophageal sphincter are closed. It has been suggested that the physiological function of this brief inspiratory effort (the “swallow breath”) is to inhale pharyngeal air and thereby prevent air swallowing (9). Conceivably, positive nasal pressure could interfere with elevation of the soft palate, which is essential for closure of the superior pharyngeal valve. Valve closure is essential for the perceived air swallowing preventive function of the swallow breath.

Finally, Samson et al. (7) discuss the clinical relevance of their findings, suggesting that when patients are treated with positive nasal pressure, decreased NNS might compromise upper airway protection. Accordingly, they suggest that further studies are warranted. Clearly there are numerous clinically relevant ramifications of inadequate coordination of swallowing and breathing. For example, how is the upper airway protected during episodes of obstructive sleep apnea (OSA)? If collapse of the pharyngeal airway during an OSA episode is associated with reduced piriform fossae volume, the pooled secretions there in might reach the level of the glottis. If so, aspiration could occur with the sudden airway reopening that terminates the OSA episode. Does aspiration in fact occur during OSA episodes or is swallowing sufficient to prevent this? These are also questions deserving further study.

REFERENCES


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