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The Effects of Superior Laryngeal Nerve lesion on Swallowing Kinematics and Airway Protection

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Abstract

The superior laryngeal nerve (SLN) carries sensory information from the mucosal tissues of the pharynx superior to the vocal folds, and carries motor signals to the cricothyroid muscles. It also provides partial innervation to the thyroarytenoid and posterior cricoarytenoid muscles. Finally, the SLN initiates the swallow. When a food or liquid bolus is swallowed, the epiglottis and the false and true vocal folds work together to seal off the airway and allow the bolus to pass through the esophagus. If the SLN is damaged, it usually leads to dysphasia in which food or liquid enters the airway.

We hypothesized that a lesion in the SLN would (1) cause an increase in liquid bolus entering the airway, and (2) the coordination between the soft palate, hyoid bone, thyroid cartilage, and epiglottis would be decreased. We surgically transected the right SLN in four 2-3 week old infant pigs. Under anesthesia, radio-opaque markers were injected into the soft palate, or sutured into the tissues superficial to the hyoid bone and thyroid cartilage. A metal clip was placed onto the tip of the epiglottis to facilitate visualization. Videofluoroscopy was used to assess the airway protection. We digitized the markers to assess movement of the pharynx and larynx during swallows. We found that the unilateral lesion of the SLN did not significantly affect the airway safety. However, it appears that sensory deficits have a considerable impact on neuromuscular coordination. Without the sensory information, the brainstem sends poor, uncoordinated instructions to the muscles controlling the soft tissues of the pharynx.

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