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Gastroesophageal and Laryngopharyngeal Reflux.

Gastroesophageal reflux is noted in 65–100% of infants with laryngomalacia [Thompson 2010]. The airway obstruction of laryngomalacia generates negative intrathoracic pressure which promotes gastric acid reflux onto the laryngopharyngeal tissues leading to laryngopharyngeal reflux. The laryngeal tissues are sensitive to the acid exposure and become edematous as a response. Increased supraglottic edema results in further collapsing of these tissues into the airway and further obstructive symptoms. A vicious cycle of increased obstruction, GERD, and edema then ensues. Prolonged acid exposure also blunts laryngeal sensation which decreases the motor response to swallow in response to secretions. Decreased laryngeal sensation explains the coughing and choking during feedings which are commonly seen with laryngomalacia. The vagal reflex responsible for laryngeal tone is also responsible for lower esophageal sphincter tone and esophageal motility [Thompson 2007]. Decreased lower esophageal tone and esophageal dysmotility are known risk factors for GERD and could be a factor in the GERD seen in laryngomalacia patients.

GERD should be treated in all patients with laryngomalacia and feeding symptoms. Upright positioning during feeding and bottles that minimize aerophagia may decrease the number of reflux events. Acid suppression therapy improves symptoms and may shorten the duration of the natural course. A combination of daytime proton pump inhibitor therapy and nighttime histamine type-2 receptor antagonist therapy is used. Most infants are kept on acid suppression therapy for an average of 9 months [Thompson 2010].

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