Laryngeal Reflexes

Exploring Terra Incognita

THE most honest of ancient cartographers referred to areas of the map beyond human knowledge as simply terra incognita. Despite their obvious clinical signi-

ance,1 reflexes that involve the function of the upper airway largely remain terra incognita for the anesthesiologist. Since the classic description of human laryngospasm by Fink in 1956,2 the anesthesia literature has been largely barren of clinical investigation into this important topic. Basic information regarding each component of these reflexes, especially in anesthetized humans, is simply not available. There are several possible reasons for this ignorance, but many probably relate to the difficulty in assessing laryngeal function in vivo. In this issue of ANESTHESIOLOGY, Tagaito et al.3 introduce a technique that may prove useful to explore this unknown territory.


Anesthesiology 1998; 88:1433–4
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Lippincott–Raven Publishers

Accepted for publication March 21, 1998. Key words: Airway; fentanyl; intravenous anesthetics; opioids; propofol; reflex.
These authors used a fiberoptic endoscope inserted through the laryngeal mask airway to directly visualize laryngeal behavior in propofol-anesthetized subjects. Simultaneous measurements of airflow permitted correlation of laryngeal structure with function (i.e., gas flow). Responses to stimulation of upper airway receptors were evoked by spraying the vocal cords with water. Using this technique, they describe a variety of laryngeal responses to this stimulation, including expiration, panting, coughing, and apnea with laryngospasm. Laryngospasm itself proved to be a complex response, consisting of two phases: an initial phase involving closure of the false vocal cords, and a later, more prolonged phase characterized by apposition of the true vocal cords. These reflexes were attenuated in a dose-dependent fashion by fentanyl, an effect that could not be explained by concurrent increases in arterial carbon dioxide. These results have direct clinical relevance. Laryngeal reflexes are a double-edged sword in the patient treated with a mask airway (laryngeal or otherwise). The presence of brisk laryngeal reflexes during propofol anesthesia is beneficial from the standpoint of protection from aspiration. However, it also means that patients are vulnerable to laryngospasm from other causes, such as surgical stimulation. Adjuvant drugs, such as fentanyl, can reduce laryngeal responsiveness, although responses still were evident in some subjects even after administration of 200 µg of fentanyl. The intriguing aspect of this work, as is the case with most good articles, is the additional questions that are raised. How can patients tolerate the presence of the laryngeal mask airway in the hypopharynx and supraglottic regions, yet maintain responsiveness to stimulation of the vocal cords? Do receptors or other parts of the reflex arc adapt? This idea may be supported by the fact that induction doses of propofol or other hypnotic agents are needed to insert the laryngeal mask airway, yet during emergence the device generally is tolerated until the patient is nearly awake. Alternatively, are there differences in the laryngeal responses elicited by stimulation of different sites of the upper airway (e.g., sites with afferent activity mediated via the superior laryngeal v. s. recurrent laryngeal nerves) or different receptors? Do laryngeal responses to stimulation applied at the airway (such as water sprayed on the vocal cords) differ from responses that can be elicited from stimulation of sites distant from the larynx (such stimulation of distal esophageal afferents by acid refflux or surgical stimulation)? If so, is there any combination of anesthetic drugs or adjuvants that could preserve laryngeal responses to local stimulation (such as provided by regurgitated gastric contents) yet attenuate laryngeal responses to surgical stimulation? Is laryngeal responsiveness a function of simply of anesthetic depth, or are there significant differences among anesthetic agents in their laryngeal effects? Why is laryngeal responsiveness, as measured by the propensity to develop laryngospasm, seemingly heightened during "light" stages of anesthesia? Are there patient factors that predispose to laryngospasm?

Ultimately the answers to these and other questions will require a detailed knowledge of the physiology of human laryngeal responses, including receptors, peripheral neural transmission and central integration, and effectors (i.e., laryngeal and pharyngeal muscles). The article by Tagaito et al. is a significant contribution that ultimately may result in a more complete map of this clinically important territory.

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References