Recurrent Laryngeal Nerve Afferents and Their Role in Laryngospasm

Joel A. Sercarz, MD, Sina Nasri, MD, Bruce R. Gerratt, Ph.D, Steven T. Fyfe, MD, and Gerald S. Berke, MD

Airway protection is a critical and phylogenetically old function of the larynx. Therefore, the sensory supply to the larynx is extensive, including afferent nerve endings in the mucosa, muscles, and joints. The majority of afferent fibers are in the distribution of superior laryngeal nerve (SLN). The afferents of the recurrent laryngeal nerve (RLN) have been infrequently studied in humans.

Laryngospasm is defined as a prolonged occlusion of the glottis caused by contraction of the intrinsic laryngeal muscles. It is a well-known potential hazard of any type of laryngeal or pharyngeal surgery. Laryngospasm is more likely in the presence of excessive secretions or in patients who are extubated in an inappropriate plane of anesthesia. Laryngospasm has been observed in 0.87% of all patients undergoing general anesthesia, and in 1.74% of children 0 to 9 years of age.1 Laryngospasm is more common when the larynx is instrumented during a procedure.

Laryngospasm can be differentiated from reflex laryngeal closure, which is shorter in duration, and can be elicited by a wider variety of stimuli. According to Suzuki and Sasaki,2 laryngospasm is elicited only by repetitive suprathreshold stimulation of SLN afferents.

We recently obtained evidence for the presence of RLN afferents and showed their possible role in laryngospasm. Two cases form the basis of this clinical report. In both patients, laryngeal surgery for vocal fold medialization was performed and was complicated by postoperative laryngospasm, which resolved after the injection of the ipsilateral RLN with lidocaine.

CASE REPORTS

Case 1

The patient is a 61-year-old woman who presented with a 9-year history of dysphonia caused by an idiopathic left true vocal cord paralysis. Her previous evaluation included a computed tomography (CT) scan from the skull base to the mediastinum and a barium swallow, both of which were normal. She complained of breathy vocal quality and lack of vocal strength. She denied a history of laryngeal trauma, aspiration, dysphagia, thyroid surgery, neck mass, odynophagia, smoking, or alcohol use. Her past history was significant for asthma.

On examination, the voice was breathy and hoarse. Laryngostroboscopy showed incomplete glottal closure, diminished excursion of the left vocal fold, and absence of a left mucosal wave. This was consistent with an idiopathic left recurrent laryngeal nerve paralysis.

The patient was admitted in July 1990 for a left-sided arytenoid adduction. The procedure was uncomplicated, but in the recovery room shortly thereafter, she developed intractable laryngospasm. Her arterial oxygen saturation decreased to 80%.

The laryngospasm and airway distress were persistent and an emergency tracheostomy was required. After the tracheostomy, the larynx was video recorded, showing continued laryngospasm. The RLN was injected in the tracheoesophageal groove on the operated (left) side, 1 cm inferior to the cricoid cartilage. The laryngospasm was broken after the injection; the findings were recorded on a videotape (Figs 1 and 2). The patient tolerated...
plugging of the tracheostomy tube, and has not had a recurrence of laryngospasm after decannulation.

**Case 2**

The patient was a 38-year-old man when he first presented to UCLA in 1987 for the evaluation of hoarseness. He was found to have a T₂ squamous-cell carcinoma of the left larynx. He underwent vertical partial laryngectomy in February, 1987. The pathological analysis indicated that the surgical margins were free of tumor.

After surgery, the patient developed a breathy dysphonia that reduced his ability to generate adequate vocal strength. Videolaryngoscopy showed an adequate airway but significant glottic chink with inadequate projection of the pseudocord. Therefore, he underwent transcutaneous Teflon (Polytefl, Mentor Inc., Norwell, MA) injection of the pseudocord in March 1988.

He continued to have a breathy voice despite the augmentation, so a second transcutaneous injection was performed in August 1990. Immediately after the operation, he developed severe airway distress. Indirect laryngoscopy showed laryngospasm. He received 8 mg of dexamethasone and an injection of 1% lidocaine to the left RLN. This strategy was used because of its success with the patient described in case no. 1. The patient's laryngospasm was broken and his airway distress resolved without further intervention.

**DISCUSSION**

Although the human RLN is known to have afferent fibers, they have been studied primarily in laboratory animals. Basic research in the cat model documented the presence of RLN sensory fibers in the infraglottic larynx similar to the SLN fibers in the supraglottis. Significantly, research by Kirchner and Wyke³-⁶ in the 1960s showed that the RLN has afferent innervation of the laryngeal joints. The afferent fibers played a role in laryngeal closure when electrically stimulated.

The airway has numerous receptors involved with airway protection and reflex laryngeal closure. These include chemoreceptors, which are involved in laryngeal protection and the laryngeal chemoreflex: if water is placed into the larynx, then apnea, swallowing, and cardiovascular changes occur.³ Receptors in the larynx are also responsible for proprioception, pressure, tactile, and sensitivity to water and many chemical stimuli. In addition, there are mechanoreceptors in the laryngeal muscles and joints that also have significant influence on laryngeal reflexes.

Attempts to arrest laryngospasm experi-
mentally have included electrical stimulation of the PCA muscle to promote abduction. Other investigators have attempted to prevent laryngospasm with topical lidocaine placed into the airway. In the experimental setting, laryngospasm has been blocked in canines with blocking electrodes. No previous attempt to arrest laryngospasm with lidocaine injection has been reported.

A canine study by Sant’ambrogio et al discovered that the abundant receptors for temperature in the supraglottis (SLN) are not present in the area supplied by the RLN, a pattern similar to the RLN supply in the trachea. Pressure receptors were found in both SLN and RLN afferents. Probing the subglottis was found to be the most effective stimulus for RLN afferents. The same investigators detected the presence of rapidly adapting mechanoreceptors in RLN nerve endings.

Kirchner and Wyke studied laryngeal articular reflexes in a series of studies published in the 1960s. They dissected the cricothyroid and cricoarytenoid joints of cats and directly stimulated the articular nerves with bipolar electrodes. They showed that afferent impulses in the laryngeal articular nerves were able to stimulate contraction of intrinsic laryngeal muscles. The articular nerves appeared to be similar to afferents present in the joints of the limbs. It was also shown that the cricothyroid joint contains afferents destined for the RLN.

In both of the clinical cases presented, laryngospasm occurred after a laryngeal procedure and was arrested by lidocaine injection of the RLN on the side of the surgery. The effectiveness of this treatment is unexpected in case no. 1, because the efferent fibers of the RLN were paralyzed. Nonetheless, it is likely that afferent nerve fibers in the RLN were responsible for the reflex laryngospasm. One possibility is that mechanoreceptors in the cricothyroid joint could have produced the afferent stimulus for the reflex spasm of the contralateral cord. The vagus nerve was most likely not injected, given the technique used for administration. In case no. 2, the injection effectively arrested the laryngospasm, despite resection of some of the laryngeal musculature and the mucosa on the side of the injection.

These cases suggest that RLN afferents, in addition to the more commonly implicated SLN afferents, may be responsible for laryngospasm in the human. The musculature involved in laryngospasm may be either ipsilateral or contralateral to the stimulus. Finally, the RLN afferents may persist despite idiopathic laryngeal paralysis or resection of a portion of the larynx, as with vertical partial laryngectomy.

In the majority of cases, laryngospasm is most likely caused by SLN afferents. However, in some cases, RLN afferents may be responsible. In the case presented, injection of lidocaine into the RLN arrested the laryngospasm. This conclusion is in disagreement with other reports, that defined laryngospasm as a response to SLN stimulation only. Future basic research will help to verify these preliminary observations in the human larynx and further elucidate the pathophysiology of laryngospasm.

REFERENCES