Severe Dysphonia after Use of a Laryngeal Mask Airway

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COMPLICATIONS that occur after laryngeal mask airway (LMA) use are usually minor and include an incidence of sore throat occurring in 7–16% of patients.1,2 More severe complications are anecdotal (i.e., lingual artery compression,3 transient hypoglossal nerve paralysis4 and transient bilateral vocal cord paralysis).5–8 We report three cases of severe dysphonia after anesthesia during which an LMA was used.

Case Reports

Case 1

A 19-yr-old man, weighing 67 kg, underwent surgery for unilateral inguinal hernia. Preoperative assessment disclosed no significant medical problems. After induction with 150 μg fentanyl and 2.5 mg kg−1 propofol, a fully deflated, size 3 LMA was easily inserted. The cuff was inflated with 20 ml air. Anesthesia was maintained with isoflurane and 50% N2O in oxygen, and his lungs were ventilated using positive pressure ventilation. The procedure lasted 90 min. The inflated LMA was gently rejected by the awake patient during recovery. A few hours after surgery, the patient complained of pharyngeal paresthesia, sore throat, and dysphonia. On the following days, the dysphonia progressively worsened until the patient was aphonic, and a laryngeal incompetence with fluid aspiration occurred. A direct laryngoscopy showed an immobilized right vocal cord. A stroboscopic examination confirmed a right recurrent nerve palsy. Two months later, the patient recovered vocal cord mobility.

Case 2

A 54-yr-old woman, weighing 52 kg, underwent a dilatation and curettage and a breast biopsy. Preoperative assessment did not disclose any relevant medical history. After induction with 100 μg fentanyl and 5 mg kg−1 propofol, a fully deflated, size 3 LMA, lubricated with silicone spray, was easily inserted. The cuff was inflated with 30 ml air. Anesthesia was maintained with propofol, and the patient spontaneously breathed 60% N2O in oxygen. The procedure lasted 60 min. During recovery, the LMA was gently expelled by the patient. A few hours later, the patient complained of sore throat and dysphagia. The next day, hoarseness occurred. On the third day, the patient

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was aphonic and exhibited severe dysphagia, with laryngeal incompetence. A direct laryngoscopy showed an immobilized right vocal cord. The electromyogram confirmed a right recurrent nerve palsy. Dysphagia lasted 4 months, and dysphonia lasted 6 months. Six months after surgery, a stroboscopic examination showed partial recovery of vocal cord mobility.

**Case 3**

A 68-yr-old man, weighing 71 kg, was scheduled to undergo colonoscopy. Preoperative assessment did not disclose any relevant medical history except a colectomy for cancer 1 yr before. After induction with 3 mg·kg⁻¹ propofol, a fully deflated, size 4 LMA was easily inserted. The cuff was inflated until no leak occurred during manually assisted ventilation; the total volume injected was not noted. Anesthesia was maintained with propofol, and the patient spontaneously breathed 50% N₂O in oxygen. The procedure lasted 30 min. The LMA was gently removed by the anesthetic nurse without deflating the cuff. After recovery, the patient complained of sore throat and laryngeal paresthesia. The next day, the patient experienced severe dysphonia and a severe sore throat. Laryngoscopy and electromyography of the vocal cord allowed a diagnosis of a dislocation of the left arytenoid with an immobilized left vocal cord. The voice was partially restored by speech therapy.

**Discussion**

Case 3 is the first report of arytenoid dislocation after the use of an LMA. This rare complication is, in most cases, due to a direct trauma during tracheal intubation. In this case, several mechanisms may be discussed. Direct trauma during placement can be hypothesized either by the leading edge of the cuff, if the LMA is directed toward the laryngeal vestibule instead of the hypopharynx, or by the tip of the mask bowl if the soft deflated rim of the mask, folded back during insertion, lifts the arytenoid. If this occurs, the tip of the mask bowl may strike the arytenoid instead of passing behind it. Also, if the LMA is inserted against the laryngeal inlet, inflation of the cuff may result in backward displacement of the vocal process, and dislocation of the arytenoid may occur during swallowing, removal of the LMA with the inflated cuff, forced traction, or twisting of the LMA, which may cause a rotation of the larynx. Another possible mechanism is direct trauma to the arytenoid cartilage against the LMA during recovery, if the patient swallows while the LMA is still in situ. During swallowing, the larynx moves upward and forward, and the superior constrictor muscles of the pharynx contract. This may cause the superior aspect of the arytenoid to be forced against the LMA, especially if the lower part of the cuff is so high that it is inflated immediately posterior to the arytenoids.

In case 3, the LMA was inserted apparently easily in the correct position and removed without deflating the cuff, when the patient was awake. This favors the hypothesis of a trauma to the arytenoid against the LMA during recovery or removal of the LMA. However, because we cannot be sure that LMA placement was adequate, a misplacement may have occurred, with a resulting direct trauma to the left arytenoid.

Four case reports of vocal cord paralysis after LMA insertion have been published. Several mechanisms of injury have been proposed and discussed: misplacement of the LMA tip between the false cords might cause pressure on the vocal folds and lead to paresis; hyperextension of the neck could result in stretching of the vagus nerve; local diffusion of the viscous lidocaine jelly applied to the LMA cuff; reaction to products used for cleaning; pressure neuropaxia by the overinflated cuff due to diffusion of nitrous oxide. All authors agree that the likely mechanism is a pressure neuropaxia by the cuff of the LMA. The recurrent laryngeal nerve passes medial and dorsal to the inferior cornu of the thyroid cartilage and lies lateral to the broad expanse of the posterior lateral aspect of the cricoid, before passing anterior on the superior aspect of the posterior cricoarytenoid muscle. The likely point of injury to the recurrent nerve by the LMA is in the cricoid region, at the lower part of the pyriform fossae, because it is not protected from pressure exerted by the cuff. It should be pointed out that, when perfectly positioned, the LMA lies with the tip resting against the upper esophageal sphincter, with the sides facing the pyriform fossae.

In cases 1 and 2, a misplacement of the LMA can be excluded because no sign of partially obstructed breathing pattern occurred when the LMA was in situ (i.e., 90 min and 60 min, respectively). In addition, the LMA appeared to be positioned correctly (i.e., black line facing cranially; bulge of thyroid and cricoid cartilages on inflation of cuff, short tubing protruding from mouth, low inspiratory pressure, and easy manual ventilation). No lidocaine jelly was used to lubricate the bowl. The diagnosis of neuropaxia can be substantiated by the ears, nose, and throat examination findings and the resolution of the palsy within a few months. However, immobilization of the vocal fold may also be due to a local pressure-induced inflammatory process, like cricoarytenoid joint arthritis or ankylosis, subsequently leading to recurrent nerve involvement. Progressive worsening of dysphonia and dysphagia and the duration of symptoms are in favor of an ischemic in-
Inflammatory reaction. In addition, an inflammatory reaction of the cricoesophageal muscle, which is responsible for the opening of the upper sphincter of the esophagus and is located in the region possibly compressed by the cuff, may have worsened the dysphagia. In case 2, the overinflation of the cuff (30 ml air) and the severity of dysphagia favor this hypothesis.

Pharyngeal morbidity is usually low after LMA use, and the incidence of sore throat is approximately 10%, mild hoarseness 0–12%, and mild dysphagia 4%.1,2,3 Symptoms last from a few hours to no more than 2 days.4 However, more severe complications have been reported and thought to be due to pressure exerted by the cuff against the surrounding pharyngeal structures.14 This pressure can exceed the capillary perfusion pressure even when the cuff is inflated with the recommended inflation volume.15,16 In addition, due to intra-cuff nitrous oxide diffusion, the cuff pressure increases during anesthesia.15–17 To limit this pressure, partial cuff deflation, intra-cuff pressure monitoring, or limiting to the pressure at which the seal occurs have been proposed.15,16,17 Two recent articles demonstrate that intra-cuff pressure can be decreased to 22 mmHg in spontaneously ventilating patients18 and to a pressure less than 43 cm H2O in ventilated patients,13 with minimal ventilatory effects. Brimacombe and Berry17 recommend choosing as large an LMA size as possible to decrease the intra-cuff pressure needed to preclude any leaks ("just seal pressure") and therefore limit pharyngeal morbidity. Recently, Brain18 pointed out that the published inflation values represent maximum volumes, and recommended not inflating the cuff above a pressure of 60 cm H2O.

In summary, we report three cases of severe, long-term dysphonia and glottic incompetence after use of an LMA. We speculate that these complications were due either to pressure exerted by the cuff on the pharyngeal structures or to misplacement of the LMA. We suggest limiting cuff pressure to the "just seal pressure" and routinely using the insertion technique described by Brain that has led to less misplacement.12

References

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