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Vocal fold palsy after use of the laryngeal mask airway

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Abstract
We report two cases of left vocal fold palsy following use of the laryngeal mask airway. In both cases anaesthesia was uneventful with a duration of about 60 minutes. It is proposed that high intra-cuff pressures induced during anaesthesia resulted in distension of the hypopharynx and subsequent neuropraxia of the motor branches of the recurrent laryngeal nerve and the external branch of the superior laryngeal nerve.

Key words: Vocal fold paresis; Anaesthesia, inhalation; Laryngeal mask airway, complications

Introduction

The laryngeal mask airway (LMA) is regarded to be a safe and convenient method of providing an airway for general anaesthesia avoiding some of the complications of endotracheal intubation. However, with increasing usage the number of reported complications is rising. Local complications at the site of placement of the mask are often the result of high cuff pressures that arise during general anaesthesia. The increase in volume and subsequent pressure of the cuff leads to distension of the hypopharynx. We report two cases of vocal fold palsy following the use of the LMA. The anatomy of the nerve supply to the larynx and a possible mechanism of injury is described.

Case reports

Case 1

A 63-year-old male who sustained a subcapital fracture of the right femur after tripping over a paving stone was prepared for a closed reduction and fixation of the fracture. His pre-operative anaesthetic assessment was unremarkable and he was graded as American Society of Anaesthesiologists (ASA) class 1. In particular, no abnormality of his voice was noted pre-operatively. Induction of anaesthesia was carried out with propofol, midazolam and morphine. A size 4 LMA was inserted without difficulty. Anaesthesia was maintained with 67 per cent nitrous oxide in oxygen with isoflurane for a duration of 55 minutes. Both removal of the LMA and the early post-operative recovery phase was uneventful.

The next day, however, he complained of a sore throat and hoarseness and was subsequently referred for an otolaryngology opinion. Examination of his larynx confirmed an immobile left vocal fold. A chest X-ray was requested to exclude any pathology affecting the recurrent laryngeal nerve and this was normal. Speech therapy was recommended, but as his voice gradually improved over the next six weeks he did not wish to pursue this.

Case 2

A 64-year-old female was scheduled for an elective vaginal hysterectomy. She was in good general health classed as ASA 1. After an oral premedication of temazepam and metaclopramide, anaesthesia was induced with propofol and fentanyl. A size 3 LMA was inserted without difficulty and anaesthesia maintained with nitrous oxide and isoflurane in oxygen and increments of fentanyl for a duration of 60 minutes. There were no reported problems with either the operative or anaesthetic course. Removal of the LMA and the early post-operative recovery period were uneventful.

Two days later the patient complained of hoarseness. As this did not improve she sought a specialist opinion from her general practitioner who duly referred her. When she was seen in the department of otolaryngology two months after her surgery, she was found to have an immobile left vocal fold with the fold lying in the paramedian position. A CT scan from the skull base to the mediastinum was requested to exclude any pathology that may have affected the recurrent laryngeal nerve, and this was found be normal.

She did not wish to pursue speech therapy as her voice gradually improved over the next three months. Nasendoscopy performed at this interval of three months confirmed that there was an improvement in the mobility of the fold.

Discussion

The LMA has become a popular method of providing an airway for general anaesthesia. It has also been demonstrated that use of the LMA leads to less change in vocal function compared to endotracheal intubation with some authors recommending its use in voice professionals (Harris *et al.*, 1990; Lee *et al.*, 1993). It was therefore surprising to see two cases with a significant change in vocal function secondary to vocal fold palsy after use of a LMA.

There have been isolated reports of hypoglossal nerve damage after the use of the LMA and these have been suggested to be due to pressure exerted by the cuff on the nerve as it passes superior to the greater cornu of the hyoid bone (King and Street, 1994; Nagai *et al.*, 1994). It is well known that the cuff pressures in the LMA increase during

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general anaesthesia due to diffusion of nitrous oxide into the cuff. This has been elegantly demonstrated by Marjot who also showed that these pressures may exceed the capillary perfusion pressure of the adjacent pharyngeal mucosa (Marjot, 1993; O'Kelly *et al.*, 1993). It is proposed that a similar mechanism of injury took place in these two cases causing a neuropraxia of the recurrent laryngeal and external branch of the superior laryngeal nerves. These nerves provide the motor supply to the intrinsic muscles of the larynx with the recurrent laryngeal nerve supplying all the muscles except for the cricothyroid which is supplied by the external branch of the superior laryngeal nerve.

Anatomy

The superior laryngeal nerve leaves the vagus at the nodose ganglion just below the skull base and passes inferomedially deep to the carotid artery. The external branch descends on the inferior constrictor muscle on the lateral wall of the pyriform fossa and ends in the cricothyroid muscle passing below the apex of the pyriform fossa.

The recurrent laryngeal nerve branches from the vagus under the subclavian artery on the right and the arch of the aorta on the left. Both recurrent laryngeal nerves ascend in the tracheo-oesophageal groove and reach the larynx by passing below the lower border of the inferior constrictor muscle (the apex of the pyriform fossa) (Bowden, 1955; Durham and Harrison, 1964).

When the LMA is inserted, its distal rim lies wedged in the hypopharynx and in some cases the oesophagus may be seen inside the distal rim (Benumof, 1992). It is therefore proposed that when the intracuff pressure rises due to nitrous oxide diffusion, stretching and direct pressure on the recurrent laryngeal and external branch of the superior laryngeal nerves occurs, and in these two cases led to neuropraxia.

The diagnosis of neuropraxia is supported by the onset of the injury, the laryngoscopic findings and the resolution of the palsy within a few months. The possibility of a dislocated arytenoid cartilage was also entertained but this was not supported by the clinical findings, in particular, the gradual improvement in voice. The observation of an immobile vocal fold in the paramedian position precludes the possibility of only the external branch of the superior laryngeal nerve being affected and it is therefore presumed that neuropraxia of either the recurrent laryngeal nerve alone, or in combination with the external branch of the superior laryngeal nerve, was the cause of the vocal fold palsy (Dedo, 1970). The authors intend to carry out further study on the effect of the LMA on laryngeal nerve function using electromyography to determine accurately which nerves are affected.

This report questions the recommended advice given to voice professionals to choose the LMA instead of endotracheal intubation. Whilst the LMA may still be preferable to tracheal intubation these two cases have shown that its use is not without risk to the laryngeal nerve supply. We recommend that this information should be included in counselling voice professionals prior to general anaesthesia.

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