A case of near fatal laryngospasm

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A 46 year old man was admitted to intensive care with a diagnosis of encephalitis (Glasgow coma scale 12/15) and bronchopneumonia. He had a past history of obesity (BMI 34) and sleep apnoea. He was reviewed for mechanical ventilation but it was requested that the physiotherapy staff continue to manage the chest, including insertion of a nasopharyngeal or Guedels airway if necessary.

The patient then proceeded to have intermittent periods of desaturation to SpO₂ 89–92%. A junior physiotherapist was paged urgently. On examination, the patient was slumped in supine with nasal prongs in situ (2 l/min), respiratory rate 15 breaths/min, SpO₂ 94%, and with widespread audible transmitted sounds. He did not cough on command.

It was decided to insert a nasopharyngeal airway and suction the patient. A size 7 Portex™ was selected and size checked between the patient’s nares and tragus. Due to the patient’s body habitus and difficulty in finding a wardsperson, the procedure was completed in a slumped supine position. The airway was lubricated and inserted without difficulty. Suction through the nasopharyngeal airway was applied and a large amount of purulent secretions aspirated. The patient’s SpO₂ improved initially to 96–97% and the chest was clearer on auscultation. However, after three minutes, the patient developed a tracheal tug, SpO₂ fell to 85%, and respiratory rate rose to 56 breaths/min. Emergency assistance was summoned. The patient continued to deteriorate, and by the time the intubation trolley arrived SpO₂ had fallen to 39% and the patient was deeply cyanosed.

The patient was given bag and mask ventilation, paralysed with suxamethonium chloride 1.0 mg/kg intravenously, intubated, and ventilated on synchronised intermittent mandatory ventilation (volume cycled). On intubation the vocal cords were noted to be extremely swollen and oedematous and he was noted to have pulmonary oedema.

So what occurred with this patient? It was hypothesised that the extremely swollen vocal cords and subglottic oedema noted on intubation were due to laryngospasm. This patient had not been intubated previously and had been suctioned only once. Therefore, there was no other reason for the swollen cords.

As the patient exhibited pulmonary oedema on intubation this also supported the diagnosis of laryngospasm, as negative pressure involved in obstruction of the airway can precipitate pulmonary oedema. It was fortunate that this patient was in intensive care at the time of the incident. Any further delay in intubation may have proven fatal.

Why did laryngospasm occur? Laryngospasm is most commonly precipitated by direct airway stimulation (airway manipulation, blood or secretions in the pharynx) (Visvanathan et al 2005). Therefore the insertion of a nasopharyngeal airway or suction catheter may have initiated the laryngospasm.

In this patient, the incorrect sizing of the nasopharyngeal airway may have increased the amount of direct airway stimulation. When inserted correctly, the nasopharyngeal airway should lie approximately 10 mm above the epiglottis.

If the airway is too short it will fail to separate the soft palate from the posterior wall of pharynx. If the airway is too long it can pass into the larynx or into the vallecula (a blind-ended pouch at the root of the tongue (Stoneham 1993), or it can cause airway obstruction if it is pressed against soft tissues.

Selection of the correct size of nasopharyngeal airway is controversial (Roberts et al 2005). Current methods taught include matching the nasopharyngeal airway length with the distance between the nose and ear (nares-tragus); correlating the size of the nostril with a patient’s little finger width and matching a patient’s height with the length of the nasopharyngeal airway. The former two methods were used with this patient. However, no correlation has been found between nares-epiglottis and nares-tragus distance in adults (Stoneham 1993) and measurements of little finger width frequently over estimate nasopharyngeal airway size (Roberts and Porter 2003). Use of these methods may have increased the likelihood of direct airway stimulation.

A number of other factors may have also been responsible for an inaccurate estimation of the nasopharyngeal airway size:

• A slumped supine position may have shortened the nares-epiglottis distance.
• Patients who are obese with a ‘bull neck’ are recognised as having a short ‘chin to larynx distance’ (Frerk et al 1996). Anaesthetic guidelines for determining airway anatomy recommend estimating ‘thyromental distance’ ie the space between the thyroid cartilage and floor of mouth (Magboul 2005). If this distance is less than two fingers it indicates the patient has an unusually short distance between the mouth and larynx.
• Patients with established obstructive sleep apnoea are at higher risk of laryngospasm and pulmonary oedema on insertion of definitive airways ie naso/endo tracheal tubes (Lorch and Sahn 2008).

So what should be done differently? Nasopharyngeal and oropharyngeal airways are usually inserted temporarily as a mechanism of halting progressive respiratory deterioration and avoiding intubation and the subsequent complications; but they can potentiate serious problems. It is important that clinicians are aware of factors that determine the appropriate sizing of an airway, which patients are more likely to constitute a difficult airway, and the potential complications related to airway insertion and suctioning. This should be incorporated into current undergraduate teaching.

The length, not the diameter of a nasopharyngeal airway is the most important measurement when choosing the correct airway. The correct estimation of nares-epiglottis distance is best based upon a patient’s height, rather than nares-tragus or little finger width measurements (Roberts and Porter 2003, Stoneham 1993). Additional factors may impact on this estimation (eg patient position, airway anatomy). In this case study, the patient’s position could have been improved prior to suction, but was difficult at the time.

Laryngospasm is a recognised risk of airway suctioning due to direct airway stimulation. The risk may be decreased by selecting an accurate nasopharyngeal airway size and.
where possible, inserting suction catheters until a cough is stimulated and not through or beyond the larynx.

It is important for clinicians to be aware of the signs and symptoms of laryngospasm and the management required. The type of laryngospasm that occurred in this incident involved subglottic oedema. Laryngospasm of this type is more dangerous and is different to the usual post extubation stridor we often observe in intensive care. Subglottic oedema is quieter and the airway is obstructed more quickly. Treatment for this type of laryngospasm requires immediate paralysis, bag mask ventilation, and intubation.

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References