

right hemidiaphragm. She most likely had some gut and urinary smooth muscle dysfunction. A cardiology assessment showed no cardiomyopathy. Her oxygen saturation was 98% on room air preoperatively. Her chest was clear.

Anaesthesia was induced with sevoflurane in oxygen supplemented with propofol. Rocuronium 50 mg was used for muscle relaxation and intubation was achieved uneventfully. Anaesthesia was maintained with desflurane in oxygen enriched air, with a small dose of fentanyl (50 µg). The procedure took an hour and a half. Active warming was used. There was no residual paralysis on a train-of-four monitor for at least 20 minutes prior to extubation. She was given sugammadex 200 mg in order to avoid the need for neostigmine and also to ensure that there was absolutely no residual neuromuscular block on extubation. She was extubated uneventfully, with no postoperative respiratory dysfunction and did not require a high dependence unit bed. She was discharged later the same day.

She presented again six months later for another urological procedure. At her pre-admission assessment she was noted to have a resting oxygen saturation of 90%, rising to 98% only after a series of deep inspirations. Paralysis of her right hemidiaphragm was confirmed.

During this second anaesthetic, under the care of another anaesthetist, she received rocuronium 50 mg, and then two hours later cisatracurium 4 mg. One hour later, after confirming no residual paralysis on the train-of-four monitor, she was given neostigmine 2.5 mg and glycopyrrolate 0.4 mg. She had had a total of 200 µg of fentanyl over the three-hour case. Unfortunately, despite a strong train-of-four, her tidal volumes and oxygen saturations were low and she could not be extubated. She was agitated and clearly stressed by her inability to ventilate adequately. She was sedated and required three hours of ventilatory support in our ICU before being extubated. She developed a postoperative lower respiratory infection and required inpatient admission for four days. While the cause of the failed extubation was likely to have been multifactorial, it is possible that the use of neostigmine may have been a significant factor. Neostigmine has been implicated in worsening neuromuscular block in patients with myotonic dystrophy<sup>2</sup> and in worsening myotonia<sup>2,3</sup>. Also as summarised by Owen and Chu<sup>1</sup>, there have been cases where large doses of neostigmine have failed to reverse neuromuscular block, while in other cases neostigmine has been used effectively and safely.

In view of the different postoperative courses in this patient when sugammadex versus neostigmine

were used, I consider that reversal of rocuronium with sugammadex appears to be the safer choice in patients with myotonic dystrophy.

J. PETROVSKI

*Sydney, New South Wales*

#### References

1. Owen PM, Chu C. Emergency caesarean section in a patient with myotonic dystrophy: a case of failed postoperative extubation in a patient with mild disease. *Anaesth Intensive Care* 2011; 39:293-298.
2. Buzello W, Kreig N, Schlickewei A. Hazards of neostigmine in patients with neuromuscular disorders. Report of two cases. *Br J Anaesth* 1982; 54:529-534.
3. Aldridge LM. Anaesthetic problems in myotonic dystrophy. A case report and review of the Aberdeen experience comprising 48 general anaesthetics in a further 16 patients. *Br J Anaesth* 1985; 57:1119-1130.

#### **Delayed airway compromise following extubation of adult patients who required surgical drainage of Ludwig's angina: comment on three coronial cases**

The American Society of Anesthesiologists Task Force on the Management of the Difficult Airway<sup>1</sup> states "the literature does not provide a sufficient basis for evaluating the benefits of an extubation strategy for the difficult airway. The Task Force regards the concept of an extubation strategy as a logical extension of the intubation strategy".

Unfortunately, there is little information about detection of delayed airway compromise in adult patients with Ludwig's angina (a bacterial infection of the floor of the mouth causing swelling and pain) following extubation in the operating theatre. Airway management of this condition is problematic and typically focuses on the difficulties of tracheal intubation prior to surgical drainage<sup>2</sup> of a dental abscess and Ludwig's angina. Postoperative airway monitoring is usually limited to the post-anaesthesia care and intensive care units.

The general literature concerning diagnosis of the acute obstructed airway frequently provides a list of symptoms or signs including dyspnoea, stridor, voice changes and drooling. They are rarely prioritised and may lead to confusion amongst junior personnel over what symptoms and signs are important acute airway management indices<sup>3</sup>. Focus is usually on immediate care with reference to devices (such as exchange catheters and bougies) or tests to identify patients who are suitable for extubation (e.g. radiological imaging, direct laryngoscopy before extubation or cuff leak tests). Unfortunately, these tests take only a 'snap shot' of the airway patency and this may

change rapidly. Laryngeal oedema may reform within the patient's airway following removal of a tracheal tube. Follow-up fiberoptic nasal endoscopy has been advocated for assessment of acute epiglottitis<sup>4</sup>. In the postoperative phase of Ludwig's angina, this requires a level of expertise that may not be available outside the high-dependency unit.

Previously, one of the authors (KBG) reviewed the coronial inquest of the death of a patient from acute airway obstruction from Ludwig's angina<sup>5</sup>. We wish to highlight some points that might assist in the management of similar cases.

The authors collated data from the National Coroners Information System ([www.ncis.org.au](http://www.ncis.org.au)) during the period 2000 to 2008 (Queensland 2001 onwards). Keywords used to search the database included hypoxia (excluding hanging, drowning, strangulation), oesophageal intubation, failed intubation and airway obstruction.

A total of 23 cases which involved deaths from acute upper airway obstruction in the postoperative period were identified, of which three occurred following extubation after surgical drainage of dental abscess and Ludwig's angina. All three were male patients aged between 27 and 39 years and all three died between January 2001 and December 2002. One patient died in the post-anaesthesia care unit

on the day of surgery and the other two in a general surgical ward following surgery. One of these two patients died on the day of operation. The other patient was left intubated and ventilated overnight after surgery. He was extubated on the following day but died after transfer to the general ward.

Signs known to indicate respiratory distress including poor voice (soft, hoarse or weak), poor cough and pain and/or difficulty in swallowing (drooling) were reported in each case. Voice changes and poor cough are secondary to oedema of the glottis and difficulty in adducting the vocal cords. The cause of drooling may occur during the oral and pharyngeal phases of the swallow. Initially, secretions are normally pushed posteriorly towards the pharynx with an anterior-to-posterior tongue elevation. Ludwig's angina displaces the tongue and interferes with this mechanism. In the pharyngeal phase, the larynx prevents material from entering the trachea by closing the true vocal cords, false vocal folds and aryepiglottic folds respectively. Displacement of the tongue and glottic and supraglottic oedema impede this function.

Stridor, respiratory distress and cyanosis are late signs of impending airway obstruction – often too late for controlled re-intubation of the trachea. The coronial inquests suggest that the junior medical

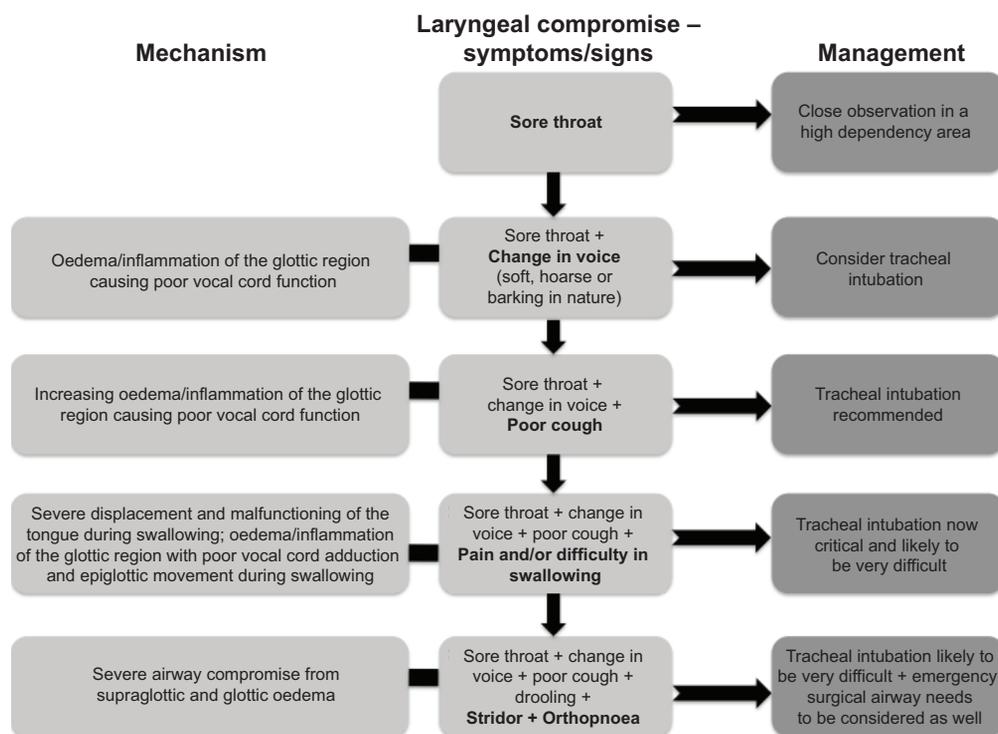


FIGURE 1: Signs and symptoms of laryngeal compromise with suggested clinical management.

staff and nurses involved had problems making a diagnosis and arranging emergency tracheal intubation. In all three cases consultant specialists became involved late in the management, either just prior to or when airway obstruction occurred. Unfortunately, emphasis was placed on pulse oximetry and detection of acute respiratory distress as a way of detecting the requirement for emergency tracheal reintubation. In all of these cases, nurses, junior medical staff and anaesthetic trainees apparently failed to appreciate the importance of the more subtle clinical features of laryngeal compromise. Figure 1 outlines the main features their implications for management.

Lessons learned from these coronial inquests support the management the first author (KBG) suggested in a recent case report<sup>5</sup>. First, some patients may complain of a very painful throat which may require opiates. Following this, the patients experience deterioration in their voice quality (e.g. attaining a soft, hoarse or barking in nature). Therefore, the voice quality of patients with suspected airway compromise should be assessed by asking them to phonate. Poor voice quality should be considered an important sign prompting immediate involvement of senior medical staff, because it most likely indicates oedema and malfunction of the voice production function of the glottis. The next sign of laryngeal compromise appears to be a poor cough. Like a poor voice, this sign may indicate oedema and malfunction of the glottis. A poor cough is a major concern and urgent tracheal re-intubation should be considered. Finally, painful and/or difficult swallowing followed by stridor and respiratory distress are hallmarks of impending airway obstruction. Urgent attention by an anaesthetist or intensive care specialist is important to reassess and arrange immediate tracheal intubation. While the order of the early clinical signs may vary among patients, the loss of voice, cough or swallow should mandate urgent review by experienced medical staff and patient relocation to a high dependence area.

Another concern is the use of opioids to manage the severe postoperative pain in patients with Ludwig's angina. This may lead to patient somnolence, with a reduction in the likelihood of detecting changes in voice quality or cough. Regular paracetamol and use of non-steroidal anti-inflammatory drugs may help reduce opioid use and assist with clinical monitoring.

We accept that only three cases provide a limited scientific foundation for our recommendations. Moreover, clinical pathways and algorithms are no substitute for clinical judgment based on experience.

Nevertheless, these three cases suggest that more attention should be paid to the early prodromal signs of airway compromise in patients with Ludwig's angina, and that monitoring should continue well after extubation. Every anaesthetist, surgeon and intensive care physician should be aware of the key clinical signs to monitor in these patients and have a clear understanding about when airway intervention is required. These signs should also be clearly explained to ward nurses, junior medical staff and anaesthetic trainees outside the high dependency units who are called on to manage patients with Ludwig's angina.

**Acknowledgement:** The authors would like to thank Ms Marde Hoy from the National Coroners Information System for her assistance with system database search.

K. B. GREENLAND

C. ACOTT

R. SEGAL

R. H. RILEY

A. F. MERRY

*Brisbane, Queensland*

#### References

1. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report. *Anesthesiology* 2003; 98:1269-1277.
2. Ovassapian A, Tuncbilek M, Weitzel EK, Joshi CW. Airway management in adult patients with deep neck infections: a case series and review of the literature. *Anesth Analg* 2005; 100:585-589.
3. Awad Z, Pothier DD. Management of surgical airway emergencies by junior ENT staff: a telephone survey. *J Laryngol Otol* 2007; 121:57-60.
4. Neff SP, Merry AF, Anderson B. Airway management in Ludwig's angina. *Anaesth Intensive Care* 1999; 27:659-661.
5. Greenland KB. Death following upper airway obstruction after surgical drainage of Ludwig's angina – case report. *ANZCA Bulletin* 2011; March 54.

#### **Hypocoagulation state in severe systemic inflammatory response syndrome/sepsis is consistent with endogenous heparinoid effect as detected by thromboelastography**

A hypocoagulation state may occur in severe systemic inflammatory response syndrome or in sepsis, which may be caused in part by endogenous heparinoids<sup>1</sup>.

We report the thromboelastography (TEG, Haemoscope, IL, USA) findings of a patient who developed a perioperative myocardial infarction and developed a severe systemic inflammatory response syndrome 48 hours after radical cystectomy. The blood sample was collected from an artery