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Iatrogenic airway injury

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Key points

- Knowledge of airway injuries aids prevention and allows timely diagnosis and correct treatment.
- Consideration should be given to cuff pressure measurement in tracheal tubes and supraglottic airway devices.
- Tracheal stenosis should be suspected in patients with dyspnoea after prolonged intubation.
- Excessive force with introducers, bougies, and stylets can lead to perforation of the aerodigestive tract.
- The use of video laryngoscopes is associated with airway injury.

While most anaesthetists use airway devices on a daily basis without apparent complication, they should always be aware of the possibility of injury. Knowledge of these injuries aids prevention and allows timely diagnosis and correct treatment. In this article, we summarize injuries reported from common airway interventions with particular focus on post-intubation tracheal stenosis (PITS).

Face masks

Allergic and irritant type reactions are much less common with single-use face masks than with reusable masks, especially where chemical disinfectants are used in the cleaning process. Soft tissue injuries such as abrasions and ulcers are possible in anaesthetic practice. However, now that prolonged face mask anaesthesia is uncommon, they are more likely to be observed in patients receiving non-invasive ventilation or continuous positive airway pressure. The eye is particularly vulnerable to contact with a face mask and corneal abrasions are possible unless a correctly sized mask is used and continued care is taken. Serious harm from the use of face masks is uncommon and appears to be caused mainly by excessive pressure. This can result either from the face mask itself or from the anaesthetist's hands when performing a jaw thrust manoeuvre and can result in sensory change to the face from pressure to the mental nerve. Likewise pressure to the mandibular branch of the facial nerve can result in paralysis of the muscles of the lower lip and chin. These examples of neuropraxia are rare, occurring only in occasional case reports, and appear to be transient, typically resolving over the course of weeks to months. Central retinal artery occlusion resulting in temporary blindness has been reported in relation to prolonged face mask use.

Supraglottic airway devices

The laryngeal mask airway (LMA™) has been in use for more than 25 yr and has an excellent safety profile in terms of iatrogenic injury. The manufacturer estimates that there have been more than 300 million LMA™ insertions, yet serious injuries are only described in a few case reports. Most harm from supraglottic airways results from inappropriate selection, for example, in those patients at risk from aspiration of gastric contents. Physiological complications of airway manipulation are beyond the scope of this article, but readers are referred to the supraglottic airway section of the RCOA NAP 4 audit for more information.[1]

The most common complication from supraglottic airway device (SAD) insertion is transient sore throat and although the incidence may be as high as 17.5%, this is still significantly lower than that seen with tracheal tubes (reported as high as 90%). More serious injury results from neuropraxia associated with pressure on cranial nerves from the tube (lingual nerve), or cuff (hypoglossal and recurrent laryngeal nerves) of the SAD have been reported in small case series. Injury to the lingual nerve usually presents as loss of taste and sensation to the tip of the tongue, hypoglossal nerve as dysphagia, and recurrent laryngeal nerve as altered voice and rarely, stridor. Symptoms typically present within 48 h and appear to resolve spontaneously over the course of weeks to months. Identified risk factors for the development of these injuries are the use of nitrous oxide and selection of a SAD that is too small, both of which result in over-inflation of the cuff and excessive pressure on adjacent structures. The manufacturer of the LMA™ recommends a *maximum* inflation pressure of 60 cm H_2O, and often a gas-tight seal will be found at much lower pressure. It has been proposed that the incidence of neuropraxia (and sore throat) will be decreased by routine measurement of cuff pressures;[2] however, guidelines are lacking on the subject. Oedema of the uvula, epiglottis, and posterior pharyngeal wall has also been reported with SADs. Whether any of these injuries occur less frequency in second-generation devices with non-inflatable cuffs remains uncertain.

Tracheal tubes

Post-intubation tracheal stenosis

The first *cuffed* tracheal tube was described by Arthur Guedel in 1927 and was designed to prevent contamination of the airway and provide a gas-tight seal. The early cuffed tracheal tubes could be described as 'low volume high pressure', and required high cuff pressures to overcome the compliance of the elastic cuff material. When the cuff was inflated, the measured pressure would bear little relation to the contact pressure between the cuff and the tracheal mucosa. In addition, the circular shape of the cuff could result in points of high contact pressure in the D-shaped trachea. Ischaemia of the tracheal mucosa was identified as the causative factor in the development of tracheal stenosis in patients who had been intubated. A significant contribution to the underlying pathophysiology and surgical treatment of PITS was made by American thoracic surgeon Hermes Grillo[3] and prompted the development of 'high volume low pressure' (HVLP) cuffs in the 1970s.

Ischaemia of the tracheal mucosa occurs when the cuff or any other section of the tube (such as the tip or entry point of a tracheostomy tube) provide a contact pressure greater than capillary perfusion pressure. The cartilaginous tracheal rings also receive their blood supply from the overlying submucosa and are also susceptible to damage from high cuff pressures. This results in perichondritis and healing takes place by granulation with strictures typically developing over 3–6 weeks. The pattern of injury is displayed in Figure 1.

The critical contact pressure that causes ischaemia has been estimated at 30 cm H_2O, and therefore, it is recommended that HVLP cuffs are operated at pressures below this.[4] Continuous monitoring and adjustment of cuff pressure of tracheal tubes reduces the incidence of postoperative sore throat and decreases the incidence of microaspiration, and ventilator-acquired

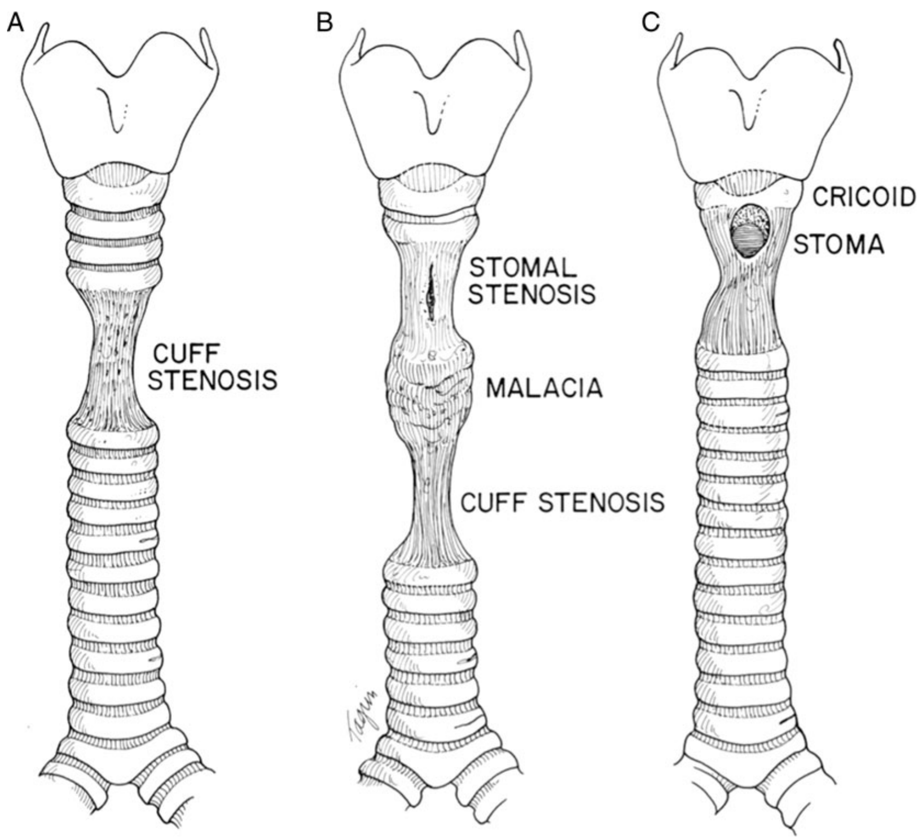

Fig 1 Spectrum of post-intubation lesions. (A) Cuff stenosis from the cuff of a tracheal or tracheostomy tube. (B) Cuff stenosis from a tracheostomy tube in conjunction with a stenosis at the level of the tracheal stoma and an intervening segment of malacic airway. (C) Laryngotracheal stenosis induced by a tracheostomy tube placed too high in the trachea. A cricothyroidostomy tube or a shallow tracheal intubation with an overinflated cuff can create a similar lesion. Reproduced with permission from Elsevier.

pneumonia, in intensive care patients. Despite this, several studies have shown that routine measurement of cuff pressure is not widely or reliably performed in either anaesthesia or intensive care, despite recommendations.[5] As with pressure monitoring of LMA™ cuffs, there is a lack of guidelines on the subject.

Despite decreased incidence with the introduction of HVLP cuffs, PITS continues to present. It occurs in roughly equal proportions between patients who have only been intubated with a tracheal tube and in those who are intubated and subsequently undergo tracheostomy. Stenosis can occur both at the stoma site and at the site of the cuff in patients post-tracheostomy. It has been estimated that the adult tracheal lumen needs to be reduced by 75% before the stenosis becomes symptomatic. Surprisingly, the incidence of PITS after prolonged tracheal intubation is poorly understood as follow-up is limited and only patients with severe stenoses are likely to present. However, case series indicate that ~80% of acquired stenosis follows tracheal intubation or tracheostomy. The overall population incidence in the UK has been estimated at 4.9 cases per million per year.[6]

The biggest risk factor appears to be prolonged intubation and there is a strong correlation between duration of intubation and the development of PITS. Our experience is that many patients who are diagnosed with 'idiopathic' tracheal stenosis will, on careful questioning, give a history of prior intubation.

The diagnosis of PITS may be made when respiratory distress follows a period of prolonged invasive ventilation, but patients can also present many months afterwards with dyspnoea and wheeze. Patients are often misdiagnosed with asthma. A history of dyspnoea after prolonged ventilation should prompt a clinician to consider PITS and the diagnosis can be confirmed by flexible or rigid endoscopy (shown in Fig. 2), spirometry, and CT scanning. Spirometry, in the form of flow–volume loops, is particularly useful as it provides a dynamic, quantifiable assessment of a fixed obstruction. Spirometry patterns seen in asthma and tracheal stenosis are displayed in Figure 3.

There is a window of opportunity, usually under 3 months, for stenotic lesions when they can be successfully treated with balloon dilatation. More mature stenosis may need linear grooves cut in the affected segment before balloon dilatation. Open cricotracheal resection of the affected tracheal section can also be performed in severe cases. In the presence of significant stenosis, all these procedures present surgical and airway management challenges, and as such, they require careful planning and delivery, including referral to specialist centres for more complex procedures. Patients will typically undergo multiple treatment episodes over an extended period of time and quality of life can be severely impaired.

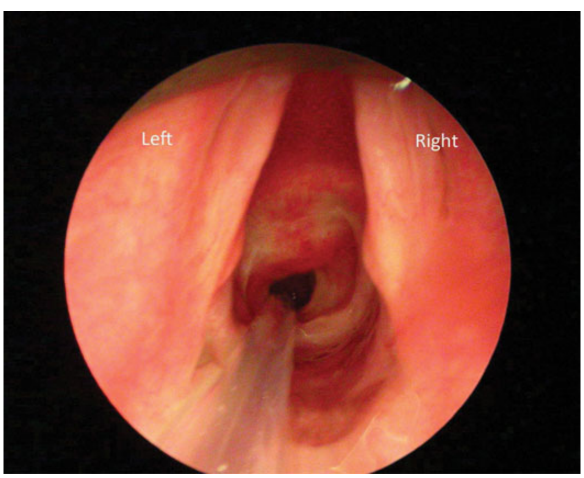

Fig 2 Four millimetre subglottic stenosis at distal edge of cricoid cartilage. A high-frequency jet ventilation catheter can be seen passing through the stenosis.

Laryngeal injury

Intubation of the trachea can result in direct injury to the larynx. This can occur either from traumatic passage of a tracheal tube on intubation and extubation or from intraoperative pressure and irritation from the rigid portion of the tube. Patients usually present with hoarseness or altered voice immediately after extubation, but in some cases, the voice is only affected months later. Vocal cord haematoma is the most common lesion seen on laryngoscopy after presentation to an ENT specialist. Mucosal thickening with oedema is the next most common, followed by mucosal laceration (laryngoscopic view of a mucosal laceration is displayed in Fig. 4), laceration of the vocalis muscle, and arytenoid subluxation.[7] Contact ulcers to the larynx can also occur after intubation. These normally heal by re-epithelialization, but in some cases, the ulcers result in granuloma formation (seen in Fig. 5) in a mechanism similar to that described above with PITS. Some cases of apparent unilateral and bilateral recurrent laryngeal nerve palsy may actually be due to fixation of the cricoarytenoid joint from scarring as a result of the chronic inflammatory process. The intralaryngeal portion of the recurrent laryngeal nerve (which innervates the intrinsic muscles of the larynx except cricothyroid) is also vulnerable to compression from a tracheal cuff positioned just below the vocal cords. Unilateral vocal cord palsy (seen in Fig. 6) should be considered as a cause of altered voice in patients intubated for only a short time. Bilateral recurrent nerve palsy is very rare but can result in stridor and airway obstruction.

Anaesthetic risk factors identified for laryngeal injury include increased tracheal tube size, increased cuff pressure, use of an introducer, intubation without muscle relaxation (leading to poor intubating conditions), and duration of intubation. In addition, patient-specific factors such as female sex (possibly because of a smaller glottic opening), a history of cigarette smoking, and gastrooesophageal reflux disease have also been shown to increase the risk. Patients presenting with altered voice or stridor should be referred to an ENT specialist and initial diagnosis is made by fibreoptic nasendoscopy. Minor lacerations will heal over the course of a few days, but major lacerations and vocal cord haematoma may take several months to resolve and result in severe laryngeal dysfunction.

Dental injuries

Dental injuries account for 11% of all medicolegal claims against anaesthetists in the UK.[8] The incidence of damage has been estimated from both retrospective and prospective studies at about 0.1%. Between a half and three-quarters occur with laryngoscopy and intubation of the trachea, and up to one-fifth on extubation. The remaining damage is caused by oral airways, LMAs, and suction devices. The most commonly damaged teeth are the maxillary incisors; they are thinner and have single roots compared with the more posterior teeth. The left maxillary incisor appears particularly vulnerable with its proximity to the largyngoscope blade on direct laryngoscopy. The risk factors for dental damage are tracheal intubation, poor dental condition, dental implants such as caps or crowns, and difficult intubation. Strategies to minimize dental damage should be considered in these patients

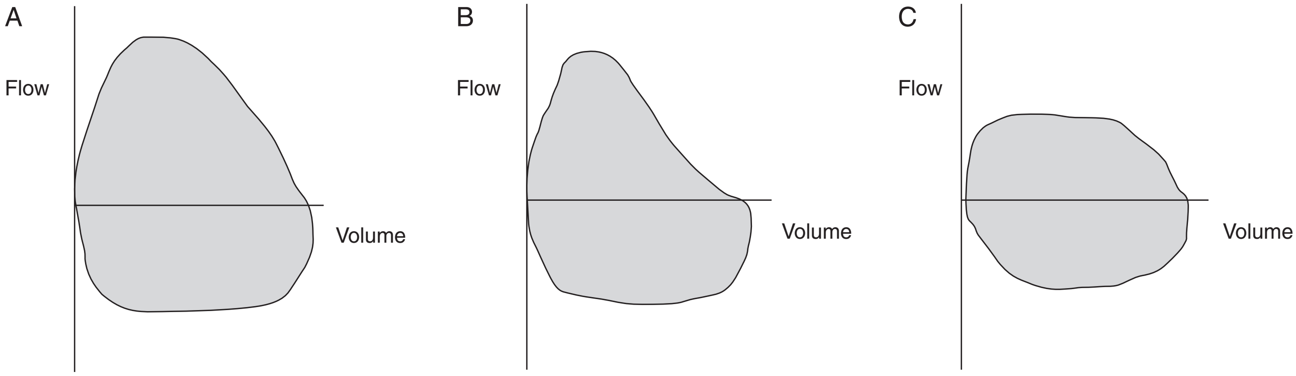

Fig 3 Demonstration of various flow–volume loops. (A) Normal flow–volume loop. A continuous curve of flow is plotted against volume with expiration above the *x*-axis and inspiration below. (B) Small airway obstruction. This is seen in asthmatic patients and the loop shows a characteristic scooped out appearance after the point of maximum expiratory flow caused by partial small airway collapse. (C) Fixed extrathoracic obstruction. This is seen in patients with tracheal stenosis. Both maximal inspiratory and maximal expiratory flow are decreased, giving a squashed appearance.

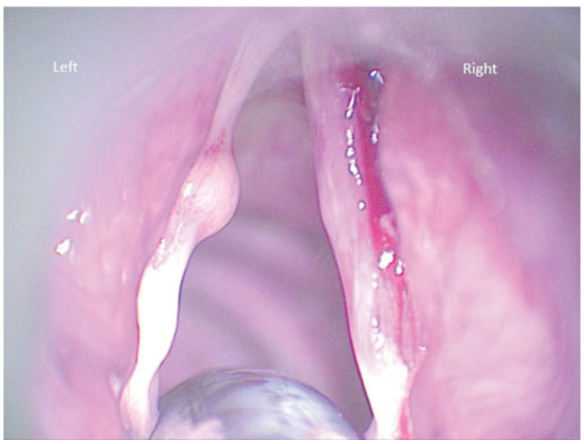

Fig 4 This patient was intubated for a microlaryngoscopy for excision of a left vocal fold intracordal cyst. The patient sustained a linear tear of the healthy right vocal cord from the tip of the microlaryngoscopy tube. This iatrogenic damage potentially worsens the difficulties in voice production in the postoperative period.

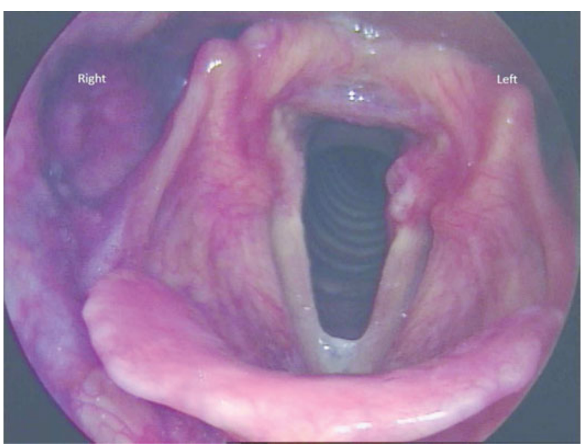

Fig 5 Arytenoid granuloma over vocal process of the left vocal cord.

and may include the use of gum shields and tracheal intubation without direct laryngoscopy (e.g. video laryngoscopy or fibreoptic intubation). In the event of damage, a dental opinion should be

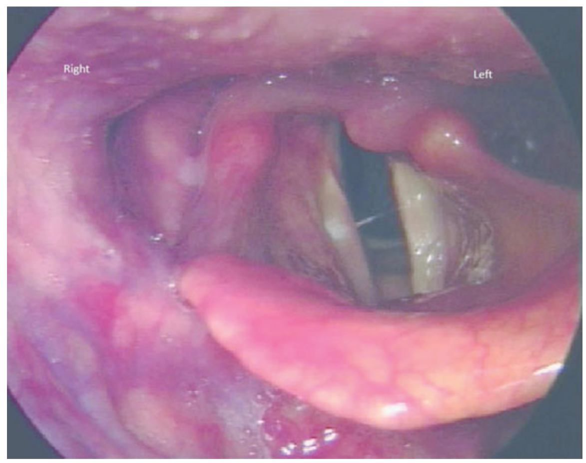

Fig 6 Left vocal cord palsy. Note the left vocal cord is in the paramedian position and shows a degree of bowing.

urgently sort. Tooth avulsion can be immediately treated by reimplantation of the tooth into its socket once the patient has recovered from anaesthesia. Absent teeth, noticed by the anaesthetist, surgeon, or patient, should be accounted for and a chest radiograph may help to exclude aspiration. Teeth in the digestive tract usually do not require intervention but if located in the airway, they must be retrieved (usually by rigid bronchoscopy). Patients with less severe damage should be referred for early dental consultation.

Nasal intubation

Epistaxis has been shown to occur in nearly 50% of nasotracheal intubations, but it is usually self-limiting and of little clinical significance. Particular care to avoid epistaxis is required in the context of antiplatelet and anticoagulant medication, and where difficulty with tracheal intubation is expected. Strategies widely accepted to reduce the incidence include the routine use of a topical vasoconstrictor, lubrication, thermosoftening, and use of Jacques catheters (a catheter passed into the nose and out of the mouth to guide the tube and dilate the nasal passage). Simple inspection of the nares and attempts to identify a more patent nasal passage by occlusion and sniffing are misleading. If in doubt, nasendoscopy is preferred.

Nasal tubes can cause infection to the paranasal air sinuses from direct obstruction of their ostia, but sinusitis is usually only a problem in longer term nasotracheal intubation on intensive care units. Ninety-five per cent of patients will show radiographic evidence suggesting infection after 1 week of nasal intubation. Other injuries are rare but have been described, and range from damage and avulsion of turbinates to pharyngeal perforation. Intracranial placement of a nasotracheal tube after transphenoidal surgery and traumatic base of skull fracture have both been described, and these conditions should represent relative contraindications to nasal intubation.

Bougies, introducers, and airway exchange catheters

These airway adjuncts have an important role in assisting tracheal intubation, especially in emergency situations. As with all of the adjuncts described in this article, there is potential for serious injury associated with their use.

Perforation of the digestive tract

This is a rare and life-threatening complication of tracheal intubation that is most commonly caused by bougies and other relatively thin introducers, but has also been reported with laryngoscope blades and tracheal tubes. It appears to be associated with repeated intubation attempts, and as such, difficult intubation is a risk factor, as is age over 60 and female sex. The cardinal signs and symptoms include neck pain, subcutaneous emphysema, dysphagia, and pyrexia. Early diagnosis and surgical referral is extremely important as risk of death from mediastinitis and subsequent sepsis increases if treatment is delayed over 24 h.

Tracheobronchial injury

Minor airway injury resulting in blood on bronchial aspiration has been reported in 5% of bougie-assisted intubations. Major haemorrhage, mucosal injury, and tracheal perforation leading to pneumothorax have all been reported and were causes of serious morbidity identified in the RCOA NAP 4 audit.[1] It is recommended that when using any of these devices, caution and the minimum force required is used to avoid injury. Additionally it has been suggested that the maximum depth that a bougie or airway exchange catheter should be inserted is 26 cm to avoid damage from entering the distal tracheobronchial tree. The design and materials used in different devices affect the stiffness and may influence the likelihood of injury. Clinicians should have a low clinical threshold for requesting a chest radiograph, to assist with diagnosis of pneumothorax and pneumomediastinum, especially after prolonged or multiple attempts at tracheal intubation. As with suspected oesophageal or pharyngeal perforation, early referral of tracheobronchial injury to a thoracic, ENT, or upper GI surgical service is important.

Video laryngoscopes

Video laryngoscopes (VLs) have been available for more than 10 yr and as clinical experience develops, their relative safety profile compared with conventional techniques can be assessed. It has been shown that less force is exerted on the maxillary incisors when using VLs compared with Macintosh blades.[9] It is tempting to believe that VLs will have a reduced propensity to cause dental damage as they do not rely on line of sight and studies large enough to prove this are awaited.

Unfortunately, serious complications have been described with VLs. There are several case reports of pharyngeal injuries with tracheal tubes loaded onto rigid stylets used to guide the tracheal tube through the glottis. It has been suggested that this may be because when using most systems, the operator often does not directly observe passage of the tube through the oropharynx, instead focusing on the video image. In one series of 2004 routine VL intubations, 0.3% of patients suffered major injuries including vocal cord trauma, tracheal injury, trauma to the hypopharynx, tonsillar perforation, and dental injury.[10] Training in VL should emphasize the importance of introducing laryngoscopes and tubes/stylettes under direct vision until the camera confirms position of the tip of the tube before tracheal intubation.

Tracheostomy

Tracheostomy tubes are usually inserted either by intensive care specialists via a percutaneous approach or by surgeons after tissue dissection. Anaesthetists should have a sound knowledge of their complications as they may be involved with insertion in theatre or called upon to assist with emergency treatment of their complications. These complications can be divided into early (<1 week) and late (over 1 week). Early complications of both insertion techniques that require intervention include major haemorrhage (damage to the innominate artery is well recognized and commonly fatal), pneumothorax, and infection. The procedure has an overall mortality of ~0.5%. A more accurate estimate of morbidity and mortality will be available with the publication of the NCEPOD tracheostomy report in June 2014. Specific airway injuries associated with tracheostomy often present late, once the patient has been decannulated. The most common airway injury is tracheal stenosis, which has been described in detail above. Other injuries include creation of false passage, tracheal cartilage fracture, tracheo-oesophageal fistula, and vocal cord paralysis.

Conclusion

Minor airway injury is common after instrumentation and has the potential to cause significant distress for patients. Anaesthetists should be aware of this possibility and use strategies to minimize risk to patients, especially when learning new techniques. In management of the difficult airway, securing a safe airway is a clear priority; however, in these situations, anaesthetists should be aware that the risk of airway injury is increased and should be actively considered.

Declaration of interest

None declared.

MCQs

The associated MCQs (to support CME/CPD activity) can be accessed at www.access.oxfordjournals.org by subscribers to *BJA Education*.

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