PROBLEMS ASSOCIATED WITH TRACHEAL EXTUBATION

M. HARTLEY AND R. S. VAUGHAN

All anaesthetists will, at some stage, have experienced problems after tracheal extubation. Indeed, the frequency of such problems probably exceeds those relating to tracheal intubation. Whereas a large volume of the anaesthetic literature addresses problems associated with tracheal intubation, it is unusual to find discussion concerning those following extubation. This review attempts to redress this imbalance.

DIFFICULT EXTUBATION

Difficulty in removing a tracheal tube at the end of a procedure is a rare, but dangerous and occasionally fatal complication of tracheal intubation. Blanc and Tremblay, in a review of the complications of tracheal intubation, reported that difficult extubation could be attributed to one of three basic mechanisms [7]: failure to deflate the tracheal tube cuff; an excessively large cuff catching on the vocal cords; adhesion of the tube to the tracheal wall because of an absence of lubricant. In addition, the tube may be transfixed by a suture or a wire to an adjacent structure.

In the majority of cases, however, the cause is the failure to deflate the cuff. This may occur inadvertently when a patient awakens suddenly and makes vigorous attempts to remove the tracheal tube. On other occasions, the cause is a failure of the cuff deflating mechanism, for example, when the cuff inflating tube has been clipped by artery forceps [39]. The walls subsequently remain crimped together and the cuff remains inflated, making extubation difficult. A similar outcome has been reported after the inflating port of a plastic tube was kinked by the retaining bandage attached to the tracheal tube [76].

Tavakoli and Corssen have reported an incident in which the inflating port became separated inadvertently at the point of insertion through the tracheal tube [78]. The cuff remained inflated and extubation was not possible. Direct laryngoscopy was performed and the tracheal tube withdrawn until the cuff rested against the vocal cords. It was then possible, under direct vision, to deflate the cuff by inserting a plastic cannula. The tracheal tube was removedatraumatically. These authors also suggested that, if attempts to remove a tube with a persistently inflated cuff fail, deflation of the cuff could be achieved by puncturing the cuff with a needle passed through the cricothyroid membrane when the cuff is at this level.

Brock-Utne and colleagues have recently reported an attempt to deflate the tracheal tube cuff rapidly, whereupon the pilot balloon and valve assembly were detached from the pilot tube [8]. The pilot tube was stretched, occluding the stump of the tube which was still attached to the inflated cuff, so that the tracheal tube could not be removed. Deflation of the cuff was accomplished eventually using a syringe and needle inserted into the stump of the pilot tube. These authors discourage the increasingly common practice of pulling off the pilot balloon and valve assembly in order to deflate the tracheal cuff, not only to avoid this complication, but also because it is important to have available a functional tube of the correct size, if reintubation suddenly becomes necessary.

Difficult tracheal extubation may follow forceful intubation with an inappropriately large tracheal tube [77], or may follow uneventful intubation with an appropriately sized tracheal tube in patients with a laryngeal abnormality [73]. In such circumstances, it is recommended that rotation of the tracheal tube [77] or manipulation of the larynx and tracheal tube under direct vision [73] may allow successful extubation.

Several instances of difficult extubation have been recorded as a result of sleeve formation by the cuffs of red rubber [36, 46, 49] and plastic [58] tracheal tubes. The sleeve is formed after the tracheal tube cuff is deflated and folds on itself during attempted extubation. The increase in the external diameter becomes too great for easy passage through the vocal cords. Should this occur, it is recommended that reinsertion, rotation and traction on the tracheal tube may allow successful extubation. If not, reininsertion and inflation of the cuff may smooth out the fold in the cuff and allow extubation after further deflation [49]. Other techniques using skin hooks [36] or forceps [46] to reduce the cuff fold have also been described.

The majority of reports of difficult extubation after fixation of the tracheal tube to adjacent structures have occurred in association with orofacial surgery. Surgical perforation and fixation of a nasotracheal tube has been described with both surgical wires [4, 52] and surgical screws [50]. It has

KEY WORDS
been recommended that, whenever the possibility exists of fixation of the tube during surgery, the tube should be moved up and down slightly to ensure that it has not been accidentally fixed [52]. Others have suggested the routine passage of a suction catheter down the tracheal tube when blindly directed wires or screws have been used [4]. More recently, the use of the flexible fiberoptic bronchoscope has been recommended to verify the integrity of the tracheal tube [50]; nevertheless, a patient has been reported from whom the nasotracheal tube could not be removed, despite these recommendations and manoeuvres [41]. The tube was removed subsequently without difficulty after the cuff inflating port was cut. After extubation, it was discovered that a Kirschner wire had passed between the nasotracheal tube and the cuff inflating port, thus preventing extubation. A similar incident has been recorded in which a nasogastric tube became entangled with the same port in the nasopharynx [28]. Extubation was prevented by tightening of a loop which wedged the inflating tube adaptor between the walls of the nose and the nasotracheal tube, and became possible only after the inflating port was cut just behind the adaptor.

Another case report described a nasotracheal tube being partially severed during a maxillary osteotomy procedure [71]. The partially cut tube formed a "barb" that caught on the posterior aspect of the hard palate when extubation was attempted. Tracheal extubation was possible only after the removal of the surgical sutures and wires.

Endobronchial tubes may also be fixed by the surgical procedure during thoracic surgery. Dryden reported a case of fatal circulatory collapse after the removal of a Carlens double-lumen tube which had been accidentally sutured to the pulmonary artery [23] and recommended that when endobronchial extubation proved to be difficult, fiberoptic bronchoscopy should be performed to inspect the tube for a tethering suture. A similar case presented as an impossible extubation of a left-sided double-lumen tube [1]. Fiberoptic bronchoscopy revealed adequate cuff deflation and no apparent cause of tube fixation, although the presence of blood in the left lumen was noted. A more vigorous attempt at extubation resulted in fresh bleeding from the left lumen and surgical fixation of the tube was strongly suspected. The surgical wound was reopened and removal of some sutures freed the tube. Subsequent endobronchial extubation was uneventful.

**CARdiovascular RESPONSE TO EXTUBATION**

Tracheal extubation is performed usually with the patient in a light stage of anaesthesia and produces significant increases in heart rate and arterial pressure which persist into the recovery period [5, 6, 25]. In 10 ASA I and II patients who were not receiving cardiovascular or antihypertensive medication, in whom extubation was performed when they were able to breathe spontaneously and open their eyes to command, Dyson and colleagues demonstrated increases of 20% or more in both heart rate and systolic arterial pressure in 70% of the patients [24]. The exact mechanism of these cardiovascular responses in man is unknown, but it is believed to be associated with the release of catecholamines causing increases in heart rate, myocardial contractility and systemic vascular resistance [66]. Recently, Lowrie and colleagues demonstrated a significant increase in the plasma concentration of adrenaline, but not noradrenaline, after tracheal extubation in a small group of patients who had undergone major elective surgery [56].

Wohlner and colleagues studied the haemodynamic responses to tracheal extubation in patients after coronary artery surgery and a period of 18–24 h mechanical ventilation [89]. They demonstrated significant increases in heart rate, mean arterial pressure, cardiac index and systemic vascular resistance index, beginning at 1 min and continuing until 10 min after extubation. They also demonstrated similar significant increases in mean pulmonary artery pressure, pulmonary artery occlusion pressure and pulmonary vascular resistance index. Other studies in similar groups of patients failed to demonstrate significant changes in some or all of these variables [26, 63]. However, the residual actions of preoperative cardiovascular medication and anaesthetic or analgesic agents may have attenuated the ability of the cardiovascular system to respond to reflex stimulation in these patients.

**Clinical significance**

The majority of patients are able to tolerate the haemodynamic response to tracheal extubation without any significant consequences. However, patients with co-existing disease may be unable to tolerate this response, whilst others will demonstrate an exaggerated response which itself is poorly tolerated.

In patients with coronary artery disease, the haemodynamic response to extubation may upset the balance between myocardial oxygen supply and demand, resulting in myocardial ischaemia. An ischaemic myocardial metabolic response to tracheal extubation has also been demonstrated in some patients after coronary artery surgery [26, 88]. Slogoff and Keats have shown that perioperative myocardial ischaemia is related significantly to episodes of tachycardia, but not to episodes of hypertension [72]. They have also established that the occurrence of perioperative myocardial ischaemia is associated with subsequent development of postoperative myocardial infarction and postulated a causal relationship. However, it has been suggested that a more likely explanation of the association between perioperative ischaemia and infarction is that these are two unrelated manifestations of severe underlying coronary artery disease [80]. Nevertheless, although suppression of the haemodynamic response to extubation is unlikely to influence the outcome in the majority of patients with stable coronary artery disease, there remains a small number of patients in whom a single hyperdynamic episode may produce a clinical catastrophe. Thus it seems reasonable to attempt to minimize the haemodynamic response to tracheal extubation in all patients with coronary artery disease.

In both treated and untreated hypertensive
patients, emergence from anaesthesia followed by tracheal extubation is associated with significant increases in heart rate and arterial pressure. These increases mimic in size those associated with laryngoscopy and tracheal intubation in the same groups of patients [74]. It is also established that laryngoscopy and tracheal intubation in patients with hypertension, whether treated or not, produces greater increases in arterial pressure than occur in normotensive patients [66]. Thus it is reasonable to expect that hypertensive patients may exhibit an exaggerated hypertensive response to awakening and tracheal extubation compared with that seen in normotensive patients. Such hypertensive crises may result in cardiac decompensation, pulmonary oedema or cerebral haemorrhage.

The evidence of Hodgkinson and colleagues [42] suggests that, in parturients with severe pre-eclampsia, the cardiovascular response to tracheal extubation is as dramatic as the response to tracheal intubation. They demonstrated mean maximal increases of 45 mm Hg in mean systemic arterial pressure, 20 mm Hg in mean pulmonary arterial pressure and 20 mm Hg in pulmonary arterial occlusion pressure. These variables remained at values greater than the pre-extubation baseline for at least 10 min after extubation. They also reported pulmonary oedema with tracheal extubation in three patients with severe pre-eclampsia. In the Report on Confidential Enquiries into Maternal Deaths in the United Kingdom 1985–87 [21], hypertensive disorders of pregnancy were responsible for the second largest number of deaths (27 deaths). In the majority of these, the immediate cause of death was either pulmonary oedema (10 deaths) or cerebral haemorrhage (11 deaths). As these occurrences are likely to relate to episodes of severe hypertension, it is important to be aware of the potential of these and other patients with hypertensive disorders to show an exaggerated haemodynamic response to tracheal extubation and to attempt to minimize this response by pharmacological means.

In patients who have undergone neurosurgical operations, the postoperative period is frequently complicated by hypertension. A recent study has demonstrated that, after intracranial surgery, 91% of patients became hypertensive in the period beginning when the volatile anaesthetic was discontinued and ending after the trachea was extubated [59]. In such patients, autoregulation of cerebral blood flow may be disturbed and a sudden increase in arterial pressure may lead to increases in both cerebral blood flow and intracranial pressure. These increases may result in either herniation of brain contents or decrease in cerebral perfusion pressure, leading to cerebral ischaemia. It seems reasonable, therefore, to attempt to prevent or suppress the haemodynamic response to extubation in such patients.

Prevention

Exubation of the trachea with the patient in a deep plane of anaesthesia, achieved by inhalation or i.v. anaesthetic agents, opioid analgesics, or both, avoids cardiovascular stimulation. However, such actions may produce depression of the respiratory and cardiovascular systems and may, occasionally, result in difficulty in the management of the upper airway. A difficult mask airway, previous difficult tracheal intubation or the risk of aspiration of gastric contents therefore contraindicates such an approach.

Lignocaine has been used to suppress the cardiovascular response to extubation in patients in a light stage of anaesthesia. Bidwai, Stanley and Bidwai demonstrated that the tracheal administration of lignocaine 60 mg 3–5 min before extubation and lignocaine 40 mg at extubation prevented increases in heart rate and arterial pressure during and after tracheal extubation [6]. They subsequently demonstrated that i.v. administration of lignocaine 1 mg kg\(^{-1}\) 2 min before tracheal extubation also prevented increases in heart rate and arterial pressure during and after extubation [5]. Wallin and colleagues observed that an i.v. infusion of lignocaine 2 mg \(\text{min}^{-1}\) throughout the perioperative period suppressed extubation-induced tachycardia and hypertension [85]. However, evidence from other studies has not always supported the ability of lignocaine to suppress the haemodynamic response to tracheal extubation, by either route of administration [25, 89].

Recently, the effects of pharmacological agents acting directly on the cardiovascular system have been investigated. The cardioselective beta-adrenoceptor antagonist, esmolol, given in a dose of 1.5 mg kg\(^{-1}\) i.v. 2–5 min before tracheal extubation, has been shown to attenuate the increases in heart rate and arterial pressure that occur in healthy patients at extubation without producing the decreases in heart rate and arterial pressure which may occur with larger doses [24]. The rapid onset and short duration of action (\(T_1/2 = 9\) min) of esmolol make it an ideal agent to prevent acute increases in heart rate and arterial pressure which occur at extubation [35].

In neurosurgical patients, the use of agents that relax vascular smooth muscle to control the increase in arterial pressure at extubation should be avoided, as the resultant cerebral vasodilatation may increase cerebral blood volume and thus intracranial pressure.

The use of alpha- and beta-adrenergic receptor antagonists has been advocated in such patients [83]. The combined alpha- and beta-adrenoceptor antagonist, labetalol, has been compared with esmolol to treat patients after craniotomy who exhibited significant hypertension after tracheal extubation [59]. Both esmolol and labetalol were effective in treating hypertension, but decreases in heart rate were more frequent in the immediate postoperative period in patients given labetalol.

COMPLICATIONS AFTER TRACHEAL EXTUBATION

Trauma

Trauma to any of the structures forming the upper and lower airways is possible at tracheal extubation, but it has rarely been reported. Trauma to the larynx and vocal cords is particularly likely after a difficult extubation and it has been recommended that direct
laryngoscopy should always be performed immediately after extubation to assess any laryngeal damage if attempts to remove a tracheal tube have been forceful and repeated [49]. In addition, damage to the mobile structures in the upper airway as a result of excessive suction at the time of extubation should always be considered.

The sequelae of trauma to the laryngeal structures may only become apparent at or soon after extubation, although the trauma may have occurred at an earlier stage of the intubation process. Dislocation of the arytenoid cartilages has been described after a difficult intubation [81], but has also followed uncomplicated tracheal intubation [31]. The usual presentation is painful swallowing and voice change, but the condition may also present as acute respiratory failure because of upper airway obstruction immediately after extubation [11, 14, 81]. This injury should be considered in all patients who experience signs of upper airway obstruction in the early period after extubation. It has been suggested also that arytenoid dislocation may have been the cause of some of the cases described previously as "glottic oedema" occurring after tracheal extubation [11]. Management requires immediate reintubation and the subsequent treatment involves either early reduction of the arytenoids by application of gentle pressure with a laryngeal spatula or prolonged tracheal intubation, or even tracheotomy, to prevent arytenoid movement and enable healing of the dislocated joint [11].

A recent case report described a lung laceration, with subsequent pneumothorax and lung abscess, after tracheal extubation over a tracheal tube changer during which the changer was advanced until a firm resistance was felt [55], yet it has been suggested that a gum elastic bougie be left in the trachea after tracheal extubation of patients with abnormal airway anatomy to assist if emergency reintubation is required [82]. The recommended technique for ascertaining tracheobronchial placement of the bougie is the detection of resistance to further passage of the stylet [47]. The authors of this case report disagreed with this recommendation and suggested that measurement and use of markings to limit the depth of insertion are warranted.

**Tracheal collapse**

Tracheomalacia may be primary or secondary (usually to thyroid pathology), but may produce upper airway obstruction only after tracheal extubation. Blanc and Tremblay described a case of tracheomalacia in a 14-yr-old boy, secondary to Pott's disease of the cervical spine [7]. Respiratory obstruction occurred after retraction of the tracheal tube 4–5 cm from the carina and was corrected only by reinserting the tube to within 1–2 cm of the carina. They recommended that, in such patients, tracheal extubation should be performed slowly. If ventilatory obstruction should occur, the tracheal tube should be repositioned and retained until surgical correction of the lesion is possible.

Tracheomalacia may result from prolonged compression by an expanding thyroid goitre, particularly within the confines of the thoracic inlet. The cartilaginous rings supporting the trachea may be weakened or even destroyed, compromising the structural integrity of the upper airway after the extrinsic compression is relieved. Tracheal collapse usually occurs after extubation and requires emergency reintubation. The subsequent options include surgical resection of the affected segment of the trachea, internal or external tracheal support, or airway diversion below the affected trachea through a tracheostomy. These options have recently been reviewed by Geelhoed [32].

**Airway obstruction**

Laryngospasm, laryngeal oedema and vocal cord paralysis are important causes of upper airway obstruction occurring immediately after extubation. They will be discussed separately. It is also important to remember causes of airway obstruction relating to foreign bodies such as throat packs, dentures and blood clots. All require immediate removal to relieve the obstruction.

In some subjects, airway obstruction occurs as a consequence of the surgical procedure—for example, haemorrhage complicating thyroid surgery [84] or other procedures in and around the neck [9, 18]. It has been suggested that the upper airway obstruction in such patients may result from laryngeal and pharyngeal oedema secondary to venous and lymphatic congestion, rather than compression of the trachea by haematoma [38]. In the case of postthyroidectomy bleeding, immediate release of the wound sutures may produce dramatic improvement, although definitive treatment requires tracheal intubation followed by surgical decompensation and control of the source of bleeding.

**Laryngospasm**

Laryngospasm has been defined as an occlusion of the glottis by the action of the intrinsic laryngeal muscles [68]. The structural and functional basis of the laryngospasm reflex has been reviewed in detail by Rex [68]; it is essentially a protective reflex, mediated by the vagus nerves, which acts to prevent foreign material entering the tracheobronchial tree. Laryngospasm is the most common cause of upper airway obstruction after tracheal extubation. It is particularly frequent in children after upper airway surgery, for example after adenotonsillectomy, with which the incidence is approximately 20% [2, 54]. It is precipitated by local irritation of the vocal cords by secretions or blood, when the plane of anaesthesia is insufficient to prevent the laryngospasm reflex, but too deep to allow co-ordinated cough. Thus it is most likely to occur after tracheal extubation of a patient in a plane of anaesthesia somewhere between an awake and a deeply anaesthetized state.

The evidence relating frequency of laryngospasm and other airway-related complications, including arterial oxygen desaturation, to tracheal extubation performed in awake compared with deeply anaesthetized patients is conflicting. Patel and colleagues did not demonstrate any significant difference in the incidence of such complications between tracheal extubations undertaken in subjects awake or anaesthetized after either adenosillectomy or strabismus.
surgery, in children who had been anaesthetised with nitrous oxide and halothane [62]. In contrast, the study of Pounder, Blackstock and Steward suggested that, in children undergoing minor surgery not involving the airway, awake extubation after either halothane or isoflurane anaesthesia was associated with a greater likelihood of arterial oxygen desaturation than occurred with tracheal extubation of deeply anaesthetized subjects [65]. In addition, while there was no significant difference in the incidence of airway-related complications between the two agents if the trachea was extubated while the children were deeply anaesthetized, isoflurane was associated with more airway-related complications than halothane in children undergoing tracheal extubation whilst awake. This study must not be interpreted to imply that the trachea should always be extubated while children are deeply anaesthetized. There are many patients, such as those with a full stomach or those whose airway is difficult to manage, who should be awake and able to maintain and protect the airway immediately after extubation.

Lignocaine has been used to prevent extubation laryngospasm after tonsillectomy. Baraka studied 40 children after tonsillectomy and showed that none of the 20 children who had received an i.v. injection of lignocaine 2 mg kg⁻¹ 1 min before extubation developed laryngospasm after tracheal extubation. In the control group, four of 20 children developed severe laryngospasm after extubation of the trachea [2]. In a similar study in adults after tonsillectomy, no patient in either the lignocaine or the control group developed laryngospasm. However, lignocaine 2 mg kg⁻¹ i.v. 2 min before removal of the tracheal tube did significantly reduce the incidence of coughing after extubation [33].

Leicht, Wisborg and Chraemmer-Jorgensen questioned this prophylactic effect of lignocaine [54]. They studied 100 children after tonsillectomy in whom lignocaine 1.5 mg kg⁻¹ was administered i.v. before tracheal extubation, which took place at a consistent depth of anaesthesia, namely when there were signs of swallowing. There was no difference in the incidence or severity of laryngospasm between this group and a control group using these dosages and timing schedules. The results of this study suggest that the beneficial effects of lignocaine demonstrated by Baraka may have been attributable to a central increase in the depth of anaesthesia. Therefore, to derive maximum benefit from the central nervous system depression produced by lignocaine, tracheal extubation must be performed before signs of swallowing activity occur.

Laryngospasm presents as a spectrum ranging from mild inspiratory stridor to complete upper airway obstruction. Although the former is not life-threatening, it may progress rapidly to the latter if managed incorrectly. Complete upper airway obstruction results in the loss of inspiratory stridor and it is of paramount importance that this "silence" is not mistaken for an improvement in the clinical state.

Management of laryngospasm after tracheal extubation may follow one of two lines of treatment. After any precipitating cause has been identified and removed by direct laryngoscopy and suctioning of the laryngo-pharynx:

1. 100% oxygen must always be administered, if necessary by positive pressure ventilation until, as the patient awakens, laryngospasm disappears.

2. The plane of anaesthesia may be deepened using i.v. or inhalation anaesthetic agents until laryngospasm and other reflexes are abolished.

However, as these methods are not always successful, it is sometimes necessary to administer the short acting neuromuscular blocking agent, suxamethonium chloride, to enable oxygenation of the patient and, if required, tracheal intubation [17]. Some anaesthetists also give i.v. atropine before suxamethonium, particularly for repeated doses or if there is hypoxia. The threshold to establish muscle paralysis and ventilation should be determined by the arterial oxyhaemoglobin saturation as indicated by pulse oximetry. Thus, faced with a patient who develops laryngospasm after tracheal extubation and an arterial oxyhaemoglobin saturation that decreases to less than 85%, it is reasonable to suggest these further measures.

Older textbooks of anaesthesia recommended the use of atropine or other anticholinergic agents as a treatment for laryngospasm. In a review of this practice, Rosen argued that atropine is of no value as either prophylaxis or treatment of reflex laryngospasm [69]. However, anticholinergic agents may be useful to remove some of the predisposing causes of laryngospasm by suppressing the secretion of saliva and mucus which may stimulate laryngospasm should they enter the upper respiratory tract.

Other measures have been recommended to treat laryngospasm after extubation. Baraka administered lignocaine 2 mg kg⁻¹ i.v. to the four children in his control group who developed severe laryngospasm following extubation; this was successful in these children within 30–90 s [2]. Owen reported the successful use of an i.v. injection of doxapram 1.5 mg kg⁻¹ in five patients who had developed laryngospasm after extubation and noted that cessation of laryngospasm was immediate in all patients [61].

Laryngeal oedema

Laryngeal oedema is an important cause of upper airway obstruction after tracheal extubation in children and, more particularly, in neonates and infants. The oedema may be localized to the supraglottic, retroarytenoid or subglottic regions [7].

Supraglottic oedema. Oedema occurs in the loose connective tissue on the anterior surface of the epiglottis and on the aryepiglottic folds. The epiglottis may be displaced posteriorly by the swelling, blocking the glottic aperture on inspiration and resulting in severe acute upper airway obstruction.

Retroarytenoidal oedema. Oedema occurs in the loose connective tissue just below the vocal cords and behind the arytenoid cartilages. Movement of the arytenoid cartilages is thus restricted and this limits abduction of the vocal cords on inspiration.

Subglottic oedema. The subglottic region has fragile respiratory epithelium with loose submucosal...
connective tissue that is easily traumatized and prone to oedema. The non-expandable cricoid cartilage, which encircles the subglottic region, is the narrowest part of the airway in children and limits the outwards expansion of the oedema.

Subglottic oedema is a particular problem in neonates and infants, in whom a minor degree of oedema may produce a significant reduction in the internal laryngeal cross-sectional area. This area in the newborn is no greater than 14 mm², and a 1-mm thick layer of oedema in the subglottic region reduces this area to 5 mm² (35% of normal) [75]. In an adult, this degree of oedema would not cause significant obstruction.

The incidence of oedema after tracheal extubation of children and the factors contributing to its development were established in a prospective study by Koka and colleagues [48]. They found an overall incidence of 1% in children younger than 17 yr, with those younger than 4 yr particularly susceptible. Factors that showed a significant positive correlation with the development of laryngeal oedema were the use of a tight-fitting tracheal tube, occurrence of trauma at tracheal intubation, a duration of intubation greater than 1 h, coughing on the tracheal tube or a change in position of the patient’s head and neck during surgery. Surprisingly, the presence of upper respiratory tract infection was not a significant contributory factor.

Laryngeal oedema presents as inspiratory stridor, usually within 6 h of extubation. In such patients, movement of respired gases must be confirmed repeatedly, as diminishing stridor may represent impending complete upper airway obstruction rather than resolving oedema.

**Management of laryngeal oedema after extubation** of the trachea depends upon its severity. Mild cases may respond to conservative measures, with inhalation of a humidified and warmed, oxygen-enriched gas mixture. Nebulized adrenaline 1:1000 (0.5 ml kg⁻¹ up to 5 ml) has been recommended in the management of infective croup in children [67] and has also been used successfully in the management of laryngeal oedema after tracheal extubation [15, 16, 37]. Relief is dramatic, but short-lived, and additional nebulized adrenaline may be required at regular intervals.

The value of systemic steroids is less clear. I.V. dexamethasone 0.25 mg kg⁻¹ immediately, followed by 0.1 mg kg⁻¹ 6 hourly for 24 h has been recommended [40], although recent studies in both children and adults indicated that dexamethasone was ineffective in the prevention of laryngeal oedema after tracheal extubation [20, 29, 79].

More severe cases of oedema after extubation, or those which fail to respond to the above measures, may require a period of reintubation with a smaller tracheal tube.

**Vocal cord paralysis**

Vocal cord paralysis resulting from trauma to the vagus nerves or their branches is an important, although rare, cause of upper airway obstruction after extubation. The rarity of the condition was confirmed in a prospective study by Walts, Calcaterra and Cohen, who visualized the vocal cords of 100 patients after tracheal extubation, none of whom showed any evidence of vocal cord paralysis [86].

Unilateral vocal cord paralysis is usually a benign condition which presents as hoarseness in the early postoperative period followed by recovery over several weeks. Bilateral vocal cord paralysis is a more serious condition which may present as upper airway obstruction immediately after extubation. The usual methods of relieving upper airway obstruction are not effective, although assisted ventilation with a facemask may overcome the upper airway obstruction. Laryngoscopy reveals motionless vocal cords which lie adducted with a very narrow glottic aperture. Immediate insertion of a tracheal tube eliminates the upper airway obstruction. Recovery is usual but often delayed, and a tracheostomy may be required temporarily.

Vocal cord paralysis is described usually after surgical procedures involving the head and neck, the thyroid gland or the thoracic cavity. It has also been described in association with increased intracranial pressure, when it may present as upper airway obstruction after tracheal extubation [13, 64]. Vocal cord paralysis has also occurred unexpectedly after surgery remote from the head and neck [34, 43, 60]. It has been suggested that tracheal intubation itself may result in peripheral nerve damage leading to vocal cord paralysis. Cavo has listed approximately 30 reports of true vocal cord paralysis in which tracheal intubation appeared to be the only explanation for the peripheral nerve damage [12]. He has also confirmed the cadaver study of Ellis and Pallister [27], who determined the course of the recurrent laryngeal nerves which, after leaving the thorax, travel in the oesophagotracheal groove towards the larynx where they divide into anterior and posterior branches. The anterior branches were found to be susceptible to compression by the tracheal tube cuff where they lie beneath the mucosa and immediately medial to the lamina of the thyroid cartilage.

**Pulmonary oedema associated with upper airway obstruction**

The development of pulmonary oedema after an episode of acute upper airway obstruction is a phenomenon which is being described more frequently. There are numerous reports, in both children and adults, of pulmonary oedema complicating upper airway obstruction after tracheal extubation [3, 19, 22, 30, 44, 45, 53, 57, 70, 87].

The clinical presentation, management and pathogenesis of pulmonary oedema associated with upper airway obstruction have been reviewed recently by Lang and colleagues [51]. The onset of the pulmonary oedema is usually within minutes of either development of acute upper airway obstruction or after relief of obstruction. Resolution usually occurs spontaneously over a period of a few hours. The essentials of management are maintenance of the airway by tracheal intubation, administration of supplementary oxygen and, if necessary, institution of positive pressure ventilation until the condition...
resolves. The majority of patients do not require either aggressive haemodynamic monitoring or drug therapy. The pathogenesis of the pulmonary oedema is multifactorial, although the markedly negative intrathoracic pressure generated during an episode of acute upper airway obstruction is probably the dominant pathophysiologic mechanism.

Laryngeal incompetence

Aspiration of gastric contents or foreign bodies at tracheal extubation may occur in patients whose protective laryngeal reflexes are obtunded by the residual effects of local or general anesthetic agents. In addition, the work of Burgess and colleagues suggests that laryngeal function is disturbed for at least 4 h after tracheal extubation, even in alert postoperative patients [10]. The mechanism of this laryngeal incompetence after extubation of the trachea is thought to be inability of the larynx to sense foreign material. The best protection against pulmonary aspiration is obtained by pharyngeal suction under direct vision, followed by extubation in the lateral position with head-down tilt.

NOTE ADDED IN PROOF: Since this review was accepted for publication, another treatment for laryngospasm has been recommended. I.v. diazepam has been used successfully to abolish the spasm, although the dose per kg is not stated [Gilbertson AA. Laryngeal spasm. British Journal of Anaesthesia 1993; 71: 168–169].

REFERENCES


