Tracheal extubation

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Key points

Problems associated with extubation, recovery, and emergence are more common than problems at intubation; many aspects are controversial with no clear guidelines or protocols.

The key to management of all post-extubation airway problems is rapid and effective administration of oxygen.

Intra-cuff local anaesthetic or spray can be used for a smooth emergence.

Laryngospasm is the commonest cause of post-extubation airway obstruction and can be life threatening.

Patients at high risk post-extubation require specific preformulated strategies.

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Respiratory complications after tracheal extubation are three times more common than complications occurring during tracheal intubation and induction of anaesthesia (4.6% *vs* 12.6%).[1] A closed claims analysis of the American Society of Anesthesiologists database revealed that death or brain damage with induction of anaesthesia decreased from 62% of perioperative claims in 1985–1992 to 35% in 1993–1999. This may reflect widespread adoption of difficult airway guidelines which predominantly address induction of anaesthesia. In contrast, the claims for death or brain damage associated with maintenance, extubation, and recovery remained almost the same.[2] Development of specific airway management plans addressing these periods of risk should improve patient safety. This article reviews the controversies and problems related to emergence and extubation after anaesthesia.

Tracheal extubation: awake or anaesthetized?

When deciding when to extubate, two main considerations should be taken care: (i) was there any previous difficulty in controlling the airway?; (ii) what is the risk of pulmonary aspiration? As a general rule, patients should be extubated awake. A small number of studies involving children show a greater incidence of upper airway complications with awake extubation as a result of increased airway reactivity.[1] However, the endpoint for wakefulness in these studies was taken as swallowing; the incidence of respiratory complications dramatically decreased when extubation was performed when eyes were open, with spontaneous ventilation.[3]

Extubation under deep anaesthesia decreases cardiovascular stimulation and reduces the incidence of coughing and straining on the tube. However, the incidence of respiratory complications has been found to be greater after extubation under deep anaesthesia, regardless of the type of operation.[1] A systematic approach to extubation is suggested in Figure 1.

Patient position

Despite advances in anaesthesia, the reported incidence of pulmonary aspiration in the perioperative period has not decreased in the last three decades; it varies from 2.9 to 10.2 per 10 000 anaesthetics.[4] Mortality rates of patients who have aspirated vary from 0 to 4.6%.[4] The traditional practice of extubating in the left lateral, head-down position maintains airway patency by positioning the tongue away from the posterior pharyngeal wall and also protects the airway from aspiration. Laryngoscopy and reintubation may be favourable in this position for experienced anaesthetists.[5]

The practice of extubation in the supine, sitting up position is controversial. The suggested rationale for its use in starved patients after anaesthesia utilizing short acting agents is not, as yet, associated with evidence to show that it is as safe as traditional methods. However, the relative ease of reintubation in a supine position does merit its use in a semi-upright position in patients who are expected to be difficult to be intubated, obese or have chronic respiratory disease.[5, 6] In these patients, and in those who have undergone upper airway surgery, the supine semi-upright position also facilitates spontaneous respiration and diaphragmatic expansion, aids an effective cough reflex, increases functional residual capacity (FRC) and encourages lymphatic drainage and reduction of airway oedema. Recent practice guidelines for patients with obstructive sleep apnoea recommend a semi-upright, lateral or any non-supine position for extubation and recovery.

Extubation in the prone position may be necessary after spinal surgery. A technique has been described where, after reversal of neuromuscular block, spontaneous sustained regular ventilation was achieved with the patient still anesthetized. Anaesthesia was then discontinued and the patient allowed waking up without stimulation; extubation was performed with eyes open or purposeful movements.[7]

In children, extubation in the recovery position while still anaesthetized is still common practice.[3]

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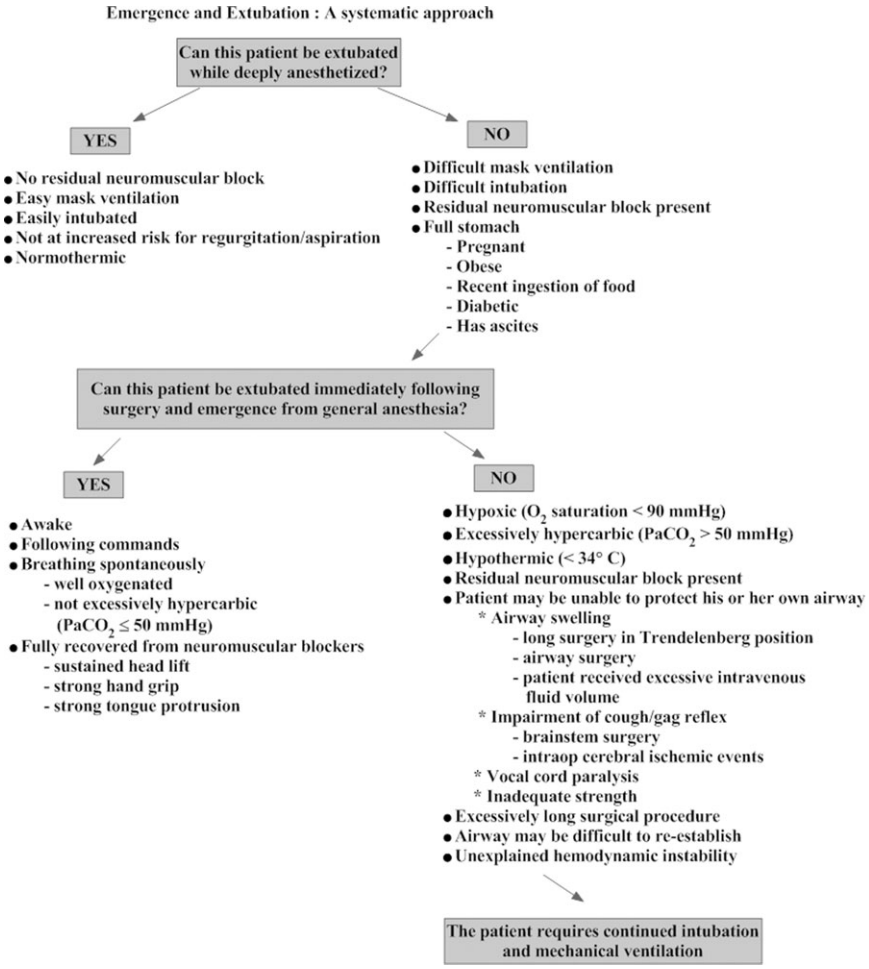

Fig 1 An approach to extubation[14] (reproduced with permission from Lippincotts, Williams, and Wilkins).

Timing of extubation

Ikari and Sasaki demonstrated that the firing threshold of the laryngeal adductor neurons involved in laryngospasm varies in a sinusoidal manner during spontaneous ventilation.[8] The mean threshold for glottic closure is increased during inspiration. Thus, extubation is usually carried out at end-inspiration when the glottis is fully open to prevent trauma and laryngospasm. Direct laryngoscopy, suctioning of the posterior pharynx, administration of 100% oxygen, ventilation to aid washout of inhalation agents, and positive pressure breath at extubation to prevent atelectasis are routine manoeuvres before extubation.

Double-lumen tubes

Removal of double-lumen tubes has been associated with tears of the posterior membranous trachea, bronchial rupture, and failure to extubate after long procedures. Double-lumen tubes are more difficult to remove during emergence as they are stiffer, bulkier, and longer; they may cause tracheobronchial trauma. A single-lumen tube is often substituted under anaesthesia at the end of the procedure for postoperative ventilation or delayed extubation. This can be facilitated by the use of hollow tracheal tube exchange catheters allowing oxygen insufflation, or fibreoptic scopes with ports for oxygen insufflation. Tubular fibreoptic laryngoscopes [WuScope (Pentax Precision Instruments, Orangeburg, NY)] have also been used for placement and tube change under direct vision.[9]

Biting the tracheal tube during emergence

A Guedel airway, inserted as a bite-block before starting to lighten anaesthesia, often ensures a secure airway through the tracheal tube during emergence. Alternatively, the tracheal tube is left *in situ* with cuff deflated, so that patients can breathe around it if the tube is bitten on during emergence. Loose teeth, caps and bridges are a hazard and should be carefully watched and accounted for if a patient is biting on the tube or airway. A nasal tracheal tube withdrawn to the nasophrynx can be used as a nasal airway during emergence.

Where should extubation be performed?

There remains some controversy about the ideal location for extubation, and transfer practices on the way to the recovery room. In the UK, 65–91% of extubations are undertaken in theatre.[6] The Anaesthetic Incident Monitoring Study (8372 reports) revealed recovery room incidents in 5% of patients; half of these occurring in ASA I–II patients and 43% of problems were related to the airway. Although many hospitals now use recovery area staff trained to perform extubations, the ultimate responsibility for the patient remains with the anaesthetist.[5]

Several studies have shown a decrease in oxygen saturations during transfer from the operating theatre, even when oxygen is administered.[1] On arrival in the recovery unit, up to 20% of patients may have oxygen saturation $<92\%$. Despite the use of 40% oxygen by face mask, 15% of patients will have an oxygen saturation of $<92\%$ for >30 s. Children are particularly susceptible to hypoxaemia during transfer; 50% have a saturation of $<95\%$ on arrival in the post-anaesthesia care unit (PACU).[9] However, one study revealed that only 63% of anaesthetists always use oxygen enriched air for transferring patients to the recovery area.[4] It is recommended that 100% oxygen is administered before leaving theatre and high inspired oxygen given during transfer.

Problems associated with extubation

Mechanical causes of difficult extubation

Possible causes of inability to remove the tracheal tube are failure to deflate the cuff caused by a damaged pilot tube, trauma to the larynx, cuff herniation, adhesion to the tracheal wall and surgical fixation of the tube to adjacent structures. Sequelae can vary from aspiration to fatal haemorrhage if undue force is applied. The problem is usually solved by puncturing the cuff transtracheally or using a needle inserted into the stump of the pilot tube; rotation and traction of the tube; using a fibreoptic scope for diagnosis; and surgical removal of tethering sutures.

Cardiovascular response

Tracheal extubation is associated with a 10–30% increase in arterial pressure and heart rate lasting 5–15 min.[8] Patients with coronary artery disease experience a 40–50% decrease in ejection fraction. The response may be attenuated by pharmacological interventions including: esmolol (1.5 mg kg^{-1} i.v. 2–5 min before extubation), glyceryl trinitrate, magnesium, propofol infusion, remifentanil/alfentanil infusion, i.v. lidocaine (1 mg kg^{-1} over 2 min), topical lidocaine 10% and perioperative oral nimodipine with labetalol.[7, 8] Alternatively, tracheal intubation can be converted to a laryngeal mask before extubation (see later).

Respiratory complications

The incidence of coughing and sore throat at emergence ranges from 38 to 96%. Specific techniques have been used to minimize this. Filling the tracheal tube cuff with liquid avoids overinflation as a result of an increase in temperature or N_2O diffusion. Lidocaine 2% with $NaHCO_3$ 1.4 or 8.4% has an excellent diffusion profile across the PVC cuff (45–65% in 6 h), is safe and less irritant in case of cuff rupture, especially if 1.4% $NaHCO_3$ is used (more physiological pH). This technique significantly reduces the incidence of sore throat in the 24 h postoperative period, coughing, bucking, restlessness, and hoarseness during emergence, without suppressing the swallowing reflex;[10] these findings merit consideration for patients with cardiovascular disease, increased intraocular or intracranial pressure and pulmonary hyper-reactivity. The laryngotracheal instillation of Topical Anaesthetic (LITA™, Sheridan, Hudson RCI) tracheal tube has an additional pilot tube through which local anaesthetic can be instilled into the larynx via 10 small holes above and below the cuff. Instillation of lidocaine through this device has been shown to reduce coughing before extubation and decrease sedation requirements in the intensive care unit (ICU).

Early postoperative hypoxaemia may be caused by inadequate minute ventilation, airway obstruction, increased ventilation perfusion mismatch, diffusion hypoxia, post-hyperventilation hypoventilation, shivering, inhibition of hypoxic pulmonary vasoconstriction, mucociliary dysfunction, and a decrease in cardiac output. In a large study involving >24 000 patients, 0.9% had a hypoxic event ($SpO_2<90\%$) in the PACU requiring intervention other than supplemental oxygen. Sustained mild hypoxaemia (>5 min) or severe desaturation ($<80\%$) in the elderly (>80 yr old) is associated with greater risk of silent myocardial ischaemia, ECG abnormalities or delirium. In a mathematical model, it has been shown that desaturation time to $<85\%$ is most rapid in postoperative adults (23 s) when compared with apnoeic children (46 s) and standard adults (84 s).[9] Another study involving >24 000 patients in a paediatric hospital found hypoxaemia to be the commonest respiratory adverse event (0.34%) in the PACU in children <8 yr of age. Preoxygenation (100% oxygen) before extubation and administration of high inspired oxygen during transfer with continuous positive airway pressure can decrease the incidence of early hypoxaemia post-extubation.

Active and passive heavy smokers, patients suffering from chronic obstructive pulmonary disease and children with mild to moderate upper respiratory tract infections have a high incidence of bronchospasm at extubation.

Residual neuromuscular block may be an absolute or contributory cause of post-extubation hypoxaemia, even after reversal and use of short acting agents.

Airway obstruction

A differential diagnosis of post-extubation upper airway obstruction (UAO) includes laryngospasm, laryngeal oedema, haemorrhage, trauma and vocal cord paralysis/dysfunction.

Laryngospasm is the most common cause of post-extubation UAO. An analysis of 4000 cases reported by the Australian Incident Monitoring Study revealed a 5% incidence of perioperative

laryngospasm (almost always after extubation in intubated patients). This may present as mild inspiratory stridor or complete airway obstruction.[11] It is more common in children undergoing upper airway surgery. Laryngospasm is most frequently caused by local irritation by blood or saliva and is likely to occur in patients during light planes of anaesthesia, when they are neither able to prevent this reflex nor generate an adequate cough. In children, the incidence can be reduced if they are left undisturbed in the lateral recovery position until they wake up.[3] I.V. magnesium (15 mg kg^{-1} over 20 min) and lidocaine (1.5 mg kg^{-1}) have been used to prevent laryngospasm. Table 1 describes a management plan for established laryngospasm.

Laryngeal oedema is an important cause of UAO in neonates and infants and presents as inspiratory stridor within 6 h of extubation.[8] Supraglottic oedema may displace the epiglottis posteriorly blocking the glottis on inspiration. Retroarytenoidal oedema below the vocal cords limits abduction of the vocal cords on inspiration. Subglottic oedema of 1 mm in neonates can reduce the laryngeal cross-section by 35%. Associated risk factors include a tight fitting tube, trauma at intubation, duration of intubation >1 h, coughing on the tube and change of head and neck position during surgery. It is also common in adults after prolonged translaryngeal intubation in the critically ill. Management includes: (i) warm, humidified, oxygen enriched air mixture; (ii) nebulized epinephrine 1:1000 (0.5 ml kg^{-1} up to 5 ml); (iii) dexamethasone 0.25 mg kg^{-1} followed by 0.1 mg kg^{-1} six hourly for 24 h; (iv) Heliox (60:40 or 80:20) temporarily stabilizes respiration giving other modalities time for effect; (v) reintubation with a smaller tube in severe cases.

UAO can be caused by direct compression or as a result of severe laryngeal and pharyngeal oedema secondary to venous and lymphatic congestion resulting from a haematoma. Immediate release of wound sutures and reintubation with definitive control of haemorrhage is usually required.

Trauma (e.g. excessive suctioning, traumatic intubation or extubation) may damage the airway and cause UAO. Arytenoid cartilage dislocation can present acutely as UAO or later cause voice change and painful swallowing. Immediate reintubation followed by gentle reduction of arytenoids or prolonged tracheal intubation is required.

Table 1 Structured approach to the management of laryngospasm[7] (the main aim is to rapidly oxygenate the patient)

Think of
Airway irritation/obstruction
Blood/secretions
Light anaesthesia
Regurgitation
Management
100% oxygen
Visualize and clear pharynx/airway
Jaw thrust with bilateral digital pressure behind temperomandibular joint, oral/nasal airway
Mask CPAP/IPPV
Deepen anaesthesia with propofol (20% induction dose)
Succinylcholine 0.5 mg/kg to relieve laryngospasm (1.0–1.5 mg/kg i.v. or 4.0 mg/kg i.m. for intubation). Be aware of contraindications, for example, neuromuscular problems
Intubate and ventilate

Vocal cord paralysis is a rare cause of UAO. This usually results from trauma to the vagus nerve after surgery involving the head and neck or thoracic cavity or direct trauma or pressure from intubation itself. Unilateral paralysis, which presents with hoarseness of voice in the early postoperative period, can usually be managed conservatively and, depending on aetiology, may recover over several weeks. Bilateral vocal cord paralysis may present as acute post-extubation UAO, requiring immediate reintubation.

Vocal cord dysfunction is an uncommon cause of UAO, usually occurring in young females with recent upper respiratory tract infection and emotional stress. It presents with laryngeal stridor or wheezing similar to asthma but unresponsive to bronchodilator therapy. Reintubation and a surgical airway may be necessary. Definitive diagnosis is by actual visualization of the paradoxical adduction of vocal cords during inspiration.[8]

Post-obstructive pulmonary oedema

The incidence of post-obstructive pulmonary oedema is ~1:1000 anaesthetics; most patients are children or young fit adults. The common pattern is an episode of airway obstruction at emergence followed by rapid onset of respiratory distress, haemoptysis, and bilateral radiological changes consistent with pulmonary oedema. Both clinical and radiological features usually resolve within 24 h with no sequelae, although delayed presentation of up to 24 h, and progression to acute lung injury and death have also been reported. The pathophysiology is uncertain. It is thought to be a result of Starling forces resulting from negative intra-alveolar pressure, increased cardiac filling, and haemorrhage from disruption of pulmonary vessels. Other implied factors are effects of hypoxaemia and catecholamine release on alveolar capillary permeability and impairment of alveolar fluid clearance by volatile anaesthetic agents. Treatment includes prompt application of positive airway pressure and oxygenation. One differential diagnosis is neurogenic pulmonary oedema, which has a similar (but more severe) clinical presentation; it characteristically presents within minutes to hours of a severe central nervous system insult.

Tracheomalacia

Softening or erosion of the tracheal rings leading to tracheal collapse and UAO may be primary or secondary to a prolonged insult by a retrosternal thyroid or other tumours, enlarged thymus, vascular malformations, and prolonged intubation. Failed extubation, complicated by inspiratory stridor or expiratory wheezing, may be the first signs of the condition. Techniques for extubation include deep extubation to avoid coughing and maintenance of continuous positive airway pressure (CPAP) to maintain airway patency.

Pulmonary aspiration

One-third of cases of pulmonary aspiration occur after extubation. A multicentre, prospective study of ~200 000 operations in France from 1978 to 1982 found that 14 of 27 clinically significant

aspirations occurred after operation. The swallowing reflex is obtunded by anaesthetic agents and laryngeal function may be disturbed, with an inability to sense foreign material for at least 4 h, even in apparently alert postoperative patients.

Recognizing the high risk patient

Such patients include those likely to be difficult to extubate and to reintubate. Patients with severe cardiopulmonary disease, congenital or acquired airway pathology, morbid obesity, obstructive sleep apnoea,[2] severe gastro-oesophageal reflux, and patients who needed multiple attempts at intubation may have problems at extubation. Contributing surgical factors include: recurrent laryngeal nerve damage (10.6% in malignant thyroids); haematoma (0.1–1.1% post-laryngeal/thyroid surgery); oedema and distortion of anatomy after head and neck surgery; posterior fossa surgery; inter-maxillary fixation; and drainage of deep neck and dental abscesses.[12]

Strategies for difficult extubation

Substituting a laryngeal mask for a tracheal tube while the patient is still anaesthetized and paralysed

A laryngeal mask airway (LMA) is inserted while the patient is in a deeper plane of anaesthesia after tracheal extubation. Muscle relaxation is then antagonized and the LMA removed when spontaneous breathing resumes and commands are obeyed. This avoids coughing and a pressor response to extubation and there is lesser need for airway manipulation compared with deep tracheal extubation with insertion of Guedel airway.[8, 12]

Extubation over a flexible bronchoscope

This can be considered in suspected laryngeal paralysis, tracheomalacia or tube entrapment. An LMA is substituted as mentioned earlier and the patient is allowed to resume spontaneous ventilation while still anaesthetized. A flexible bronchoscope is then advanced through the LMA. This enables visualization of the anatomy and assessment of laryngeal function. If required, reintubation can be facilitated using an Aintree intubation catheter which jackets the flexible bronchoscope. The latter is then removed along with the LMA and the patient is reintubated over the catheter.[12]

Use of a tracheal tube exchange catheter (reversible tracheal extubation)

This strategy is especially useful for patients expected to be difficult to reintubate. Tracheal tube exchangers (e.g. the Cook airway exchange catheter) are long hollow catheters with connectors for jet and/or manual ventilation and respiratory monitoring; most have depth and radio opaque markers, and end or distal side holes. They can be introduced through a tracheal tube permitting extubation. Spontaneous breathing, talking and coughing may take place around the device and they are tolerated well enough to be left in place until it is deemed that reintubation will not be required (up to 72 h). Figure 2 shows an algorithm for difficult extubation, which complies with the recommendations of the ASA task force on management of the difficult airway.[8, 12]

Extubation in the intensive care unit

Extubations in ICU are either protocol directed or unplanned. A significant proportion of accidental extubations may not require intubation. Reintubation after planned extubation occurs in up to 20% of patients within 24–72 h of extubation. Mortality of patients who fail a trial of extubation is 2.5–10 times than among patients who are successfully extubated. Outcome improves if reintubation occurs within 12 h of extubation.[13]

Predicting unsuccessful extubation

Classical weaning criteria are not accurate in predicting extubation outcome. Bedside tests have been used: for example, the gag reflex, strong cough to stimulation by a suction catheter, absence of excess secretions. Recent research has shown that inability to open the eyes, follow with the eyes, grasp the hand, and stick out the tongue make unsuccessful extubation four times more likely. The spontaneous breath trial (SBT) using a T-piece or continuous positive airway pressure of 5 cm of H_2O or pressure support of 3–14 cm of H_2O in adults has 80–95% sensitivity for predicting successful extubation. After completion of the SBT, a prospective study showed that a weak cough (grade 0–2) or those patients who could not cough onto a white card held 1–2 cm from the tracheal tube were three to four times more likely to have unsuccessful extubations.

The cuff leak test is used to test laryngeal patency. The average difference between inspiratory and expiratory volume after cuff deflation, recorded for six consecutive breaths, is determined. A volume $<$10–12% of delivered tidal volume implies upper airway oedema.

Post-extubation stridor

Post-extubation stridor (PES) occurs after 2–16% of extubations in the ICU. A randomized prospective multicentre double-blind trial of 700 patients showed that the incidence of laryngotracheal oedema leading to upper airway narrowing was higher in female patients, especially those intubated for $>$36 h. Other risk factors for PES include: mobile and large tracheal tubes; excess cuff pressure; tracheal infection; patients fighting the ventilator; aggressive tracheal suctioning; and the presence of a nasogastric tube. An audible leak or quantitative assessment of cuff leak volume may be used to predict risk of PES. The same principles also apply after general anaesthesia. Children who have an absent air leak at 25 cm of H_2O have a 2.8 times greater incidence of adverse respiratory events (laryngospasm, UAO, and oedema). Laryngeal ultrasound is being evaluated to predict PES. Prophylactic dexamethasone given to susceptible children decreases the prevalence

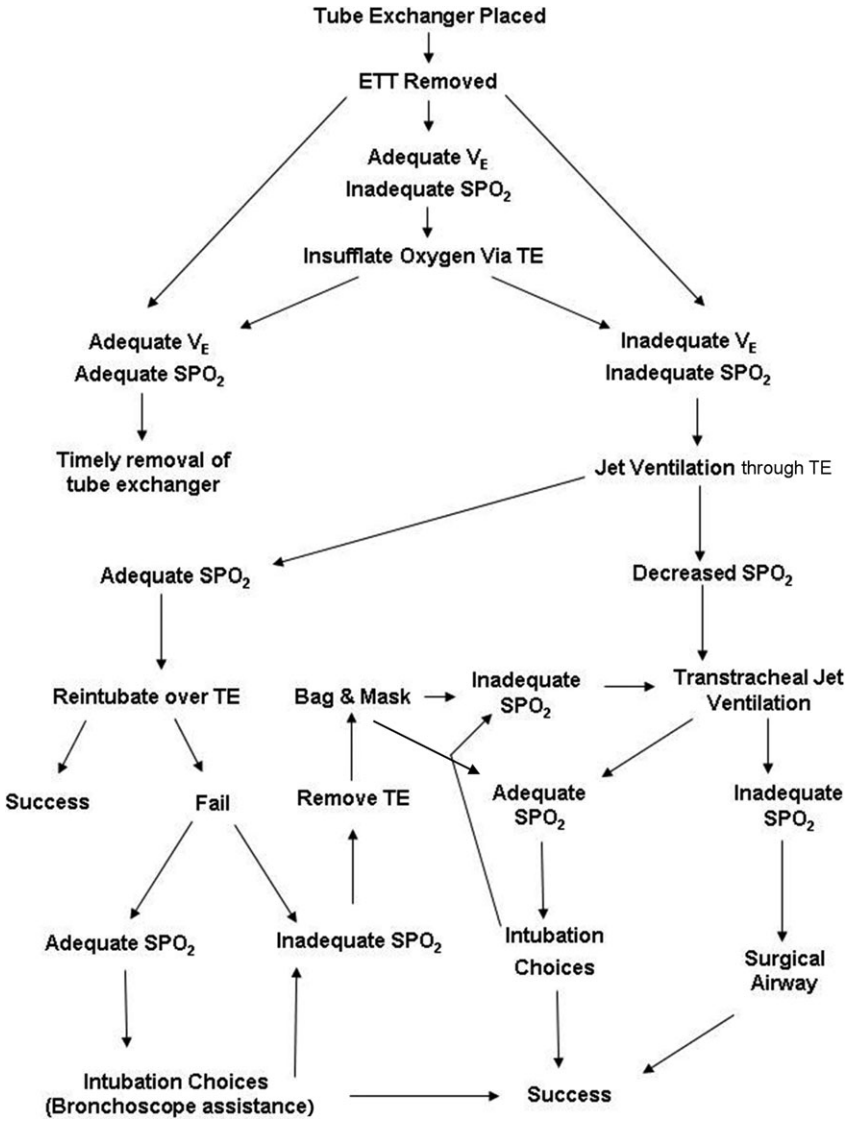

Fig 2 An algorithm for extubation of the difficult airway (reproduced with permission from Bailey P[8]).

of PES by 40%. A single methyl prednisolone injection in adults significantly decreases oedema in 6–7 h.

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Please see multiple choice questions 14–18