Critical incidents: the respiratory system

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Abstract

Respiratory complications are expensive, not just in terms of the overall litigation burden faced by anaesthetists but also, and more importantly, the mortality and morbidity burden faced by our patients. Critical incidents arising in the respiratory system can cause rapid deterioration if left unchecked: trauma to airway structures can be debilitating or even life threatening; hypoxaemia may result in damage to other organ systems, most notably the brain. Each patient carries their own risk profile, as well as unique ideas, concerns and expectations of their anaesthetist. An understanding of the potential critical incidents that may befall the respiratory system, a patient-centred approach to discussing these risks, and familiarity with the procedures for mitigating harm are all necessary components of safe, effective practice in anaesthesia.

Keywords Airway failure; airway injury; airway obstruction; aspiration; cannot intubate; cannot oxygenate; lung-protective ventilation; pneumothorax; respiratory complications

Royal College of Anaesthetists CPD Matrix: 1B02, 1C01, 1C02, 1F01, 2A01, 2A06, 3A01.

Learning objectives

After reading this article, you should be able to:

- Describe the critical incidents which may impact the respiratory system
- Summarize and quantify the risks of respiratory complications to a non-medical (patient) audience
- Formulate a management plan to reduce the risk of respiratory complications during anaesthesia

Critical incidents affecting the respiratory system can rapidly precipitate deterioration in other areas of the body; they represent a significant source of morbidity and mortality in anaesthesia. In the UK, a review of cases logged with the National Health Service Litigation Authority (NHSLA) between 1995 and 2007 found adverse airway and respiratory system events accounted for 12% of anaesthesia-related claims, and 53% of deaths. A thorough understanding of the possible respiratory system complications which may arise during anaesthesia is fundamental to its safe delivery.

The Montgomery ruling in the UK Supreme Court highlights the need for clinicians to better tailor discussions of potential complications to the individual patients with whom they consult. We can no longer rely on the Bolam test (whether an esteemed body of medical opinion would uphold a decision) when weighing-up which information to share in the consenting process; clinicians must include information about any and all risks which their patients could consider material to informing their choices when consenting for medical investigations and treatments. The aim of this review is to describe the potential respiratory system complications of anaesthesia, illustrate how these can be mitigated and, where possible, provide quantitative information regarding the risk of occurrence as a reference for clinicians.

Registries of medical litigation claims can teach us the value placed on events by the legal system, but for numerous reasons offer little insight into the true epidemiology of such incidents. Large investigations, such as the Fourth National Audit Project (NAP4) of the Royal College of Anaesthetists (RCoA) and Difficult Airway Society (DAS), provide a useful resource for understanding the rates of more serious complications. However, less serious, more frequent complications may also be notable and of importance to patients despite being considered 'common-place' by many anaesthetists.

Oropharyngeal injury

Cuts or bruising to the lips or tongue occurs in approximately 5% of anaesthetics; however, it is likely that there is significant under-reporting of such events. The risk of damage to a tooth is approximately 1:4500 cases. Laryngoscopy is the most common cause: careful, detailed preoperative assessment and documentation of the teeth, coupled with care during airway management, reduces the risk of dental damage. Rolled swabs placed between the lateral molars as a 'bite-block' can provide protection from biting and grinding during emergence. Conversely, an oropharyngeal airway adjunct may prevent occlusion of an airway from biting but poses a risk to teeth.

The estimated frequency of sore throat after anaesthesia is 20% where a supraglottic airway (SGA) is used, increasing to 40% with a cuffed tracheal tube (TT). Insertion of gastric tubes, temperature probes, suction apparatus and devices such as oesophageal Doppler or transoesophageal echo probes also risk trauma. In most cases, symptoms are mild and short-lived, but they may prove a source of complaint or litigation in the context of extended severity and chronicity, or where the patient uses their voice professionally.

Lubrication and correct size selection of airway devices, including laryngoscopes, can reduce shearing forces applied to the tissues with which they come into contact; a meticulous approach to correct placement and inflation pressures is also paramount. High volume, low pressure cuffs are to be preferred

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for TTs and pressure gauges should be used to appropriately titrate the air in SGA cuffs ($\leq$60–70 cmH_2O) and perhaps in TTs (<30 cmH_2O).

There have been case reports of damage to the laryngeal, glossopharyngeal and hypoglossal nerves caused by direct pressure from SGAs compressing the structures of the oropharynx. The i-gel™ is associated with a lower incidence of sore throat than cuffed devices, but its bulk may increase airway trauma. Clumsy laryngoscopy, especially with curved blades and stylets, can cause pharyngeal injury or perforation. Forces applied during laryngoscopy are reduced by use of videolaryngoscopy; a 2016 Cochrane review demonstrates a reduced rate of airway trauma and sore throat when videolaryngoscopy is used.

Upper airway and oesophageal injury

Laryngeal injury

The vocal cords and posterior laryngeal structures may be injured in up to 1% of intubations, affecting mostly young, healthy patients during routine uncomplicated intubation and accounting for around one-third of airway trauma claims against anaesthetists. Injuries include laryngeal granuloma, nerve palsy, arytenoid dislocation and fracture; subglottic stenoses and granulomata are rarer. Harm may be temporary or permanent and may interfere with speech, swallowing and occasionally with breathing. Injury to the posterior laryngeal components from an SGA is less common than intubation related injury. Use of excessively large tubes or inflation of a cuff across the laryngeal inlet are prime causes of injury. Solutions include avoiding intubation where it is not indicated, using videolaryngoscopy where possible to avoid blind (bougie) techniques, selecting smaller TTs and optimally managing and monitoring cuff pressure and position during use.

Tracheal and oesophageal injury

These injuries are rare, but convey significant mortality owing to mediastinitis. Oesophageal intubation with a TT can be enough to cause perforation; both oesophagus and trachea may be injured by (mis)use of stylets, TTs and bougies. Avoidance of oesophageal intubation, scrupulous use of airway adjuncts and early detection of symptoms (e.g. chest pain, surgical emphysema) and treatment after potentially injurious events reduces harm.

Lower airway injury

Barotrauma to the lower airway may result from mismanagement of the upper airway, in particular from inappropriate methods of oxygen delivery. Microscopic barotrauma occurs when positive pressure ventilation is applied with too large tidal volumes creating or exacerbating acute lung injury (see below). Macroscopic barotrauma (air leak, pneumothorax, pneumomediastinum, surgical emphysema) can occur when a high-pressure source is applied to the airway without a route for egress.

High-pressure sources include gas cylinders (400 kPa), wall oxygen (400–600 kPa) and 'jet ventilation' equipment (e.g. Sanders injectors and Manujets, 100–400 kPa). In contrast, an anaesthetic machine's fresh gas output supplies at approximately 2–6 kPa, increasing to 30–40 kPa on 'flush' mode (although this varies between machines). While it is raised gas pressures which *cause* injury, damage is hastened and more severe with increased flow rates.

Danger is greatest when a long hollow device is placed deep in the airway and oxygen is administered *via* a high pressure source. Examples are the Aintree intubation catheter (AIC) and hollow bougies used in airway exchange, or an airway exchange catheter (AEC) left in situ after extubation. If the distal portion of such a device becomes wedged, there is no escape path for delivered gas and barotrauma will result. Ensuring the tip remains above the carina (approximately 23 cm from the lips) will protect against 'wedging' and subsequent injury.

On balance AECs, AICs and hollow bougies should be reserved for airway exchange; on the rare occasions when oxygen delivery *via* these devices becomes necessary, care must be taken to maintain the tip above the carina and low-pressure gas sources must be used.

Lung injury as a consequence of controlled ventilation

Positive pressure ventilation may cause injury by effects of volume (volutrauma), pressure (barotrauma) and repeated inflation/deflation (shear/atelectrauma). Controlled ventilation leads to migration of inflammatory cells into the alveolae and release of cytokines. There is also a decrease in surfactant, an increase in production of mucus and a decrease in the efficiency of the mucociliary transport mechanisms. Already injured lungs are at particular risk of further injury. Emerging evidence suggests that 'lung protective ventilation' may have a role in the operating theatre and in elective major surgery, as well as in the intensive care unit (ICU).

Lung protective ventilation involves: low volumes (6–7 ml/kg ideal body weight); relatively high positive end expiratory pressure (PEEP) to maintain a position on the steep part of the compliance curve; avoidance of high mean airway pressures through use of pressure controlled ventilation; avoidance of high inspired oxygen concentrations; reduced periods of disconnection; and use of recruitment manoeuvres such as 'sighs' or periods of continuous positive airway pressure (CPAP) at approximately 30-minute intervals. Such protocols are associated with a lower incidence of postoperative pulmonary complications and reduced length of stay. Which components of the 'package' are most important, and how long after extubation protection persists, remains unclear.

Hypoxaemia

The NAP4 point estimate for the risk of death or brain damage from failure of oxygenation during anaesthesia was approximately 1:151,000 anaesthetics, increasing to 1:110,000 when a TT was used. The corresponding rate for SGAs was 1:202,000, and for face mask (FM) anaesthesia 1:154,000. The duration and severity of hypoxaemia required to cause death or organ failure is dependent on many factors. In NAP4 some patients came to harm from relatively short periods of apparently modest hypoxaemia while others survived prolonged and severe events (e.g. up to 30 minutes of SpO_2 <70%).

Hypoxaemia will most likely first be detected by a fall in the oxygen saturations in the presence of an otherwise adequate pulse plethysmogram. More occult presenting signs include

pallor with cyanosis, tachycardia with hypertension (early in adults), bradycardia (in young children, or late in adults), other arrhythmias and electrocardiograph (ECG) disturbances or, in extreme cases, cardiac arrest. Causes of hypoxaemia include difficulties with airway management, hypoxic inspired gas mixtures or anaesthetic circuit disconnection, inadequate ventilation, increased shunt, significant pneumothorax, pulmonary oedema, or equipment failure. Haemoglobinopathies and the use of intravenous dyes can produce spuriously low saturation readings (84–86% in methaemoglobinaemia) or reassuringly high readings when hypoxaemia is in fact present (as high as 98% with 10% carboxyhaemoglobin levels).

On detection of hypoxaemia, 100% oxygen should be administered at high flow rates; oxygen supply should be confirmed by checking pressure gauges and oxygen analysers in the breathing circuit. Mechanically ventilated patients should be temporarily switched to manual ventilation, confirming adequate volumes are being delivered and enabling assessment of equipment for defects, such as obstructions or leaks. Use of a self-inflating bag enables rapid discrimination between gas delivery/anaesthetic circuitry faults and problems with the airway device/patient. Manual ventilation also facilitates assessment of the patient's airways, which may have become obstructed by mucous, blood or gastric aspirate, and their lung dynamics, which may have been adversely affected by bronchospasm, oedema, chest wall deformity, pneumothorax or surgical intrusion. Inspection and auscultation of the chest during manual ventilation may facilitate assessment of the cause of hypoxaemia and direct subsequent actions to remedy it. Waveform capnography is critically useful to distinguish hypoxaemia from failure of ventilation (flat or very abnormal trace) from pulmonary causes (normal or nearly normal trace).

Airway failure

The majority of airway failures are unanticipated (>80% for FM techniques, >90% for intubation). When one element of airway management fails, the risk of other techniques failing is increased many-fold. This 'composite failure' is underappreciated but the likely cause of many airway disasters. Mask ventilation is difficult in 1–5% of cases and impossible in approximately 1:500. Failure rates for SGAs are less well described, ranging from 1:500 to 1:10 in different studies. In practice, an SGA may need replacing in 3–5% of cases and may fail totally in 1%.

Difficulty with tracheal intubation occurs in approximately 5–10% of anaesthetics. Failed tracheal intubation occurs in 1:1000–2000 routine anaesthetics, 1:250–300 rapid sequence inductions (RSIs), and 1:100 emergency department (ED) and ICU intubations. Difficult, failed, or delayed intubation was the primary cause in approximately half of events reported to NAP4. Importantly, failed intubation likely occurs at some point in all airway disasters. When direct laryngoscopy fails, further attempts with the same technique have a failure rate of approximately 80%. Videolaryngoscopy after failed direct laryngoscopy carries an approximate 90% success rate.

Each modality of airway management has its own risk factors, but obesity, being male, poor mouth opening (Mallampati 3–4), poor neck extension, and increasing age likely contribute across modalities.

Early identification of airway failure is key to mitigating its sequelae; guidelines in the UK mandate waveform capnography be used in clinical environments where child and adult patients undergo, or are dependent on, ventilation via an artificial airway – including anaesthesia, critical care and emergency departments. The failure to detect improper placement of a TT, owing to absent or misinterpreted waveform capnography, has been identified as a future 'Never Event' by NHS Improvement. In response, the RCoA and DAS have launched the 'No Trace = Wrong Place' safety campaign, aimed at facilitating early recognition of failed intubation. A crucial message of this campaign, given the high rates of intubation failure amongst critically ill patients, is that waveform capnography persists (albeit in attenuated form) throughout cardiac arrest and resuscitation. There is controversy as to whether the never event should also apply to neonates. There are practical challenges to use of waveform capnography in neonatal practice; it is usually readily achieved during anaesthesia, and often used during neonatal transfer, but only rarely in neonatal critical care.

The term 'cannot intubate, cannot oxygenate' (CICO) describes the situation in which progressive modalities of airway management have failed and oxygen delivery is critically compromised. The estimated CICO event rate in NAP4 was 1:50,000 anaesthetics, which is similar to other case series. The death rate from CICO was 1:479,000 anaesthetics.

Risks for CICO include surgery of the head and neck, although an increased incidence is also observed in obstetric anaesthesia and in obese patients. Rates are higher in ED and ICU settings, owing to both technical factors related to the case mixes encountered and non-technical factors related to carrying out a complex task in a setting, and as part of a team, where it may not be routinely practised.

When CICO arises, it is important to maintain a structured approach to rescuing the situation, avoiding task fixation and multiple attempts at laryngoscopy worsening an already precarious state. In the UK, the DAS publish guidelines for the structured management of unanticipated difficult intubation, encouraging a stepwise progression through the different airway management modalities. Perhaps complimentary to the DAS guidelines, the 'vortex' model describes a more fluid approach in which adequate FM ventilation, SGA and TT placement can be attempted in any order, but with no more than three attempts (or one optimal attempt) at each. The final common pathway to these approaches, in the context of ongoing airway failure – with or without hypoxaemia, is progression to establishing an emergency front of neck airway (FONA) for oxygenation.

NAP4 highlighted cases where FONA was undertaken without an attempt at oxygenation with SGA and some patients in whom FONA was established without prior administration of appropriate muscle relaxant. This has been confirmed in recent registry studies. Adequate neuromuscular blockade and attempts at oxygenation *via* an SGA are likely to rescue most CICO events and should both be undertaken before proceeding to FONA. Waking the patient should always be considered but is not always feasible.

Where FONA is attempted, NAP4 demonstrated low success rates of needle-based techniques in the hands of anaesthetists. The DAS 2015 guidelines have subsequently recommended the scalpel-bougie FONA as the rescue technique of choice.

Conversely, needle FONA techniques are favoured by some. The main pitfalls of these are difficulty accessing the airway in obese patients, equipment failure in crisis situations and complications of the high pressure source ventilation needed to ventilate via a narrow cannula. While the choice of technique remains highly controversial, the DAS guidelines rightly emphasize that the most important factor is familiarity with the chosen technique and consistency (and avoidance of choice) within an organization. Regular drills in the chosen technique, including the use of higher-fidelity 'obese' training manikins, should be undertaken in an effort to ensure procedural familiarity.

Aspiration of gastric contents

Prior to the introduction of the laryngeal mask airway (LMA), when TTs and FMs predominated, the risk of pulmonary aspiration of gastric contents ('aspiration') was estimated as 1:4000 for elective cases, and 1:900 for emergencies. Approximately 1:5000–10,000 elective anaesthetics undertaken with an LMA are complicated by aspiration; figures for more advanced (second generation) SGAs are not readily available and likely differ.

Aspiration was the most common cause of anaesthesia-related mortality in NAP4, which reported a death or brain damage rate of 1:287,000 anaesthetics. Many cases were associated with junior anaesthetists, operating without direct supervision, failing to recognize and adequately control for extant risk factors (Table 1). The majority of cases occurred during maintenance of anaesthesia with a first-generation SGA, where aspiration may be occult. Second-generation SGAs, such as the ProSeal™ or Supreme™ LMAs or i-gel™ may play some part in reducing the risk, as well as facilitating earlier detection of regurgitation.

Risk periods for aspiration include induction, before or during airway manipulation, and at the end of anaesthesia. Sensible (but not prolonged) preoperative starvation guidelines, perioperative decompression of the full stomach and premedication with antacids (e.g. sodium citrate), proton pump inhibitors (e.g. omeprazole), H_2-receptor blockers (e.g. ranitidine) and pro-kinetics (e.g. metoclopramide), may help reduce the risk or mitigate the sequelae of its occurrence.

Rapid sequence induction (RSI), and in particular the use of cricoid force, has incited much discussion and controversy. A recent large randomized controlled study compared cricoid to sham force, reporting the latter to be non-inferior in preventing pulmonary aspiration and the former to be associated with increased grade of laryngeal view. However, the aspiration event rate was lower than expected, limiting the trial's power; the study population were likely low-risk for aspiration. Moreover, the applied force may have been sub-therapeutic and the clinical significance of the worsened view with cricoid force is not clear.

The controversy surrounding the use of cricoid force is by no means settled and is likely set to continue for some time to come. Nevertheless, we know that *correctly applied* cricoid force will occlude the oesophagus or hypopharynx in many patients, and does not necessarily impede either laryngoscopy or gentle mask ventilation. Where the risk of aspiration is high and cricoid force is not used, in the UK at least, the risk of successful litigation is likely increased.

When aspiration does occur, the priority is to provide adequate oxygenation while reducing the risk of further harm. The latter includes preventing further aspiration and minimizing acute lung injury, while the former is most securely achieved through tracheal intubation. The traditional approach is to place the patient head-down, in the left lateral position, with the aim of facilitating passive drainage of pharyngeal fluid away from the larynx during laryngoscopy. While gravitationally sensible, it should be noted that such a manoeuvre adds to the technical complexity of rapidly intubating, and more conventional positioning may be preferable. The suction assisted laryngoscopic airway decontamination (SALAD) technique has recently been described as an alternative approach which maintains the patient in an optimal position for successful intubation. A suction catheter is used to displace the tongue and clear a path for laryngoscopy, before being 'parked' in the hypopharynx, held there in the left-hand groove of the laryngoscope. It has been demonstrated in simulation manikins but is as yet unproven in real patients and their anaesthetists.

Post-intubation tracheal suctioning prior to positive pressure ventilation, ideally performed during flexible optical bronchoscope (FOB) inspection, may reduce the distal spread of particulate and liquid material, associated with atelectasis and infection in peripheral lung segments. Where risk of aspiration is controlled for at intubation (and in some other circumstances) a strategy is needed to protect against aspiration at emergence; the stomach should be emptied where possible, patients should be fully reversed of neuromuscular blockade (confirmed with quantitative monitoring), placed in a safe position, and only extubated when fully awake and in possession of their protective reflexes.

Many aspiration events require only simple observation; after 2 hours, if the patient is asymptomatic, normally oxygenated and with a normal radiograph, complications are rare. Following significant events, prolonged ventilation may be required; the patient's course may be complicated by pneumonitis from exposure to low pH aspirate, pneumonia caused by gastric flora, or acute respiratory distress syndrome (ARDS). Antibiotics

Risk factors for aspiration

Patient	Surgical
Full stomach	Emergency surgery
Pregnancy: from 16/40 gestation until 24–48 hours postpartum	Upper gastrointestinal surgery
Gastrointestinal obstruction	Lithotomy position
Raised intra-abdominal pressure	Laparoscopy
Previous gastro-oesophageal surgery	**Anaesthetic**
Hiatus hernia	Junior anaesthetist
Recent trauma	Light planes of anaesthesia
Opioid use	Supraglottic (esp. first generation) airways
Raised intracranial pressure	Positive pressure ventilation
Gastro-oesophageal reflux disease	Difficult airway management (difficult intubation, difficult FM ventilation, poorly placed SGA)
Morbid obesity	Prolonged intubation attempts
	Oesophageal intubation
	Long operation times

Table 1

should not be given prophylactically, but remain in reserve should pneumonia develop. There is no survival or morbidity benefit derived from the routine use of steroids. Death following severe aspiration occurs either early from airway obstruction, or late from multi-organ failure secondary to ARDS.

Aspiration of blood

Where the airway has been exposed to blood from surgical or anaesthetic interventions, the risk of its aspiration should always be considered. Blood in the airway was a significant cause of complications reported to NAP4, including obstruction and fatalities during emergence, accounting for approximately 20% of reported cases. This is a particular risk in the context of head, neck and dental surgery.

When large volumes of whole blood are aspirated, the resultant clots can cause prolonged obstruction within the trachea and bronchial tree, leading to catastrophic, prolonged failure of oxygenation. Aspiration of blood can be occult, often occurring after the removal of a secured airway, or in the recovery area. Awareness of the risk, coupled with a meticulous and gentle approach to suctioning under direct vision following at risk surgeries, protects against inhalation of the 'coroner's clot' which can collect in the nasopharynx of the anaesthetized, supine patient. If a flat capnography trace develops after the airway has been exposed to blood, the possibility of clot aspiration should be actively excluded through direct laryngoscopy, suctioning, airway exchange, and direct examination of the trachea with FOB or rigid bronchoscopy.

Laryngospasm

Direct stimulation from above, or surgical manipulation of distant sites, can result in the forced closure of the vocal cords by the intrinsic muscles of the larynx. Laryngospasm is reported to occur to some degree in up to 1% of anaesthetics, with markedly increased incidence in paediatric practice, particularly in infants. Adult patient risk factors for laryngospasm are asthma, smoking, concurrent upper respiratory tract infection, obesity and reflux. It can also be precipitated by surgeries involving a shared airway, dissection close to the superior laryngeal nerve and surgical stimulus, especially cervical or anal dilatation. Induction and emergence are particular risk periods for laryngospasm. Low doses of intravenous or vaporized anaesthetic agents during maintenance of anaesthesia with an SGA can also be a cause, as can thiopentone (which accentuates airway reflexes) or irritant vapours (e.g. desflurane).

Initial management of laryngospasm is with 100% oxygen at high flow rates via a tightly fitting mask, using two hands if necessary to produce a jaw thrust and adequate seal. Vigorous attempts at ventilation will only serve to inflate the stomach, increasing the risk of aspiration; CPAP should instead be maintained. During emergence, CPAP, jaw thrust and patience may be enough to break the spasm, as may anterior pressure behind the ramus of the mandible (Larsen's point). Where practical, the airway should be suctioned to remove debris from around the larynx. If these techniques fail, or if laryngospasm occurs on induction, the next step is to deepen anaesthesia with propofol in 0.5 mg/kg increments. Refractory spasm may require (re)paralysis and (re)intubation in order to (re)gain control. Very small doses of intravenous succinylcholine — e.g. 0.1 mg/kg — may be sufficient to manage severe laryngospasm. In paediatric practice when laryngospasm occurs during gaseous induction intramuscular succinylcholine 4 mg/kg is rapidly effective.

Once stabilized, treatable causative factors should be addressed. Following prolonged episodes, patients should be monitored for evidence of post-obstructive pulmonary oedema (POPO), which may require an extended period of ventilation and diuretics on ICU, before extubation is re-attempted.

Bronchospasm

Bronchospasm occurs when smooth muscle in the conducting airways contracts, as a result of irritability (smokers, asthmatics, volatile anaesthetics), or light anaesthesia. In anaphylaxis, asthma and lung disease, it is also associated with airways inflammation and oedema, increased mucus secretion and airway plugging. Bronchospasm occurs in approximately 0.2% of all general anaesthetics, with the risk tenfold greater in those with reactive airways disease. Ventilation pressures will rise and widespread expiratory wheeze may be heard on auscultation or within the breathing circuit. Expiratory time will lengthen and carbon dioxide elimination will be slowed, resulting in a characteristic 'ramped' or 'sharks-fin' capnography waveform. A misplaced SGA, endobronchial intubation, tracheal tube tip/wall apposition, and mucous plugging can all mimic bronchospasm and should be excluded.

Where bronchospasm is suspected, high concentration oxygen should be delivered and anaesthesia deepened using a non-irritant volatile (sevoflurane, halothane) or intravenous anaesthetic. Salbutamol via the TT or intravenously, nebulized ipratropium bromide, and intravenous aminophylline, ketamine and magnesium all produce bronchodilation, as does adrenaline (intravenously or nebulized) in extreme cases. Intravenous corticosteroids act more slowly to suppress the underlying inflammatory processes and may be appropriate depending on the cause. Ventilation using reduced tidal volumes may be required to protect against barotrauma. Respiratory acidosis above pH 7.2 may be tolerated to allow for extended expiratory times, preventing 'gas trapping' which raises intrathoracic pressures, limits venous return, and reduces cardiac output.

Pneumothorax and tension pneumothorax

Simple pneumothorax is uncommon during anaesthesia. It may develop from pre-existing airways disease (e.g. asthma, emphysema), injury (e.g. rib fractures, blunt or penetrating thoracic injury), surgery (e.g. neck, chest, diaphragm, laparoscopy), or anaesthetic procedures (e.g. supraclavicular nerve block, subclavian or internal jugular central venous line, poorly managed controlled ventilation). During positive pressure ventilation or use of nitrous oxide, a simple pneumothorax — which may cause a minor degree of respiratory compromise — may progress to a tension pneumothorax.

Tension pneumothorax is characterized by reduced chest movement and increased expansion on the affected side. Percussion of the affected hemithorax may demonstrate hyperresonance and auscultation may reveal reduced or absent breath sounds. Ultrasonography will reveal loss of normal 'lung sliding' and 'comet tails' and the presence of a 'bar code sign'. A late clinical sign is deviation of the trachea away from the side with the pathology. As the volume of the pneumothorax

increases, mediastinal shift results in kinking of the vena cava and engorgement of the neck veins. Failure of venous return causes a fall in cardiac output followed quickly, if untreated, by cardiac arrest and death.

On diagnosis, application of 100% oxygen is classically followed by large-bore needle decompression thoracostomy (second intercostal space, mid-clavicular line), prior to definitive intercostal drain insertion. In prehospital and major trauma settings, open thoracostomy in the mid-axillary line is increasingly practised as the first-line decompression strategy for positive-pressure ventilated patients. Large bronchopleural connections may require multiple drains in parallel to prevent re-accumulation, or lung isolation using double-lumen ETTs.

Pulmonary embolus

When embolic material from the periphery lodges in the vessels of the pulmonary vasculature, an area of dead-space is produced, leading to immediate reactive changes in gas exchange, causing hypoxia and normocapnoea or hypocapnoea. There is an immediate fall in compliance, an increase in pulmonary vascular resistance and a reduction in cardiac output. The typical sequence of events is a fall in $etCO_2$, a rise in airway pressures (or fall in tidal volume depending on ventilatory mode), tachycardia, hypotension and then hypoxaemia. During massive or widespread pulmonary embolus (PE), right heart failure can occur, followed by cardiac arrest. Large blockages by fat (long-bone procedures and cementation), gas (neurosurgical or laparoscopic procedures, open central lines) or amniotic fluid (caesarean section) are most likely during anaesthesia, although embolus of septic vegetations and foreign bodies may also occur.

Although rarely implicated perioperatively, postoperative venous thromboembolism (VTE) complicates approximately 1% of general surgical and many multiples greater orthopaedic and trauma procedures. As a member of the perioperative team, the anaesthetist shares in the responsibility of risk assessment and prophylaxis against VTE by ensuring the correct application of mechanical and pharmacological measures tailored appropriately to the individual's risk profile.

Large PEs can trigger widespread inflammatory states and consumptive coagulopathies, which further complicate the situation. Management of PE is mainly supportive, with high-flow oxygen to reduce pulmonary hypoxic vasoconstriction, off-loading the right ventricle, and vasopressors to increase systemic vascular resistance, maintaining coronary perfusion. Where the source can be established, this is treated as appropriate (e.g. anticoagulation or thrombolysis for blood clot, cessation of surgery for fat embolus, flooding the operative site and raising venous pressure for air embolus).

Pulmonary oedema

Sudden onset respiratory distress, particularly in the recently extubated patient, may indicate the onset of acute pulmonary oedema. Causes may be: cardiogenic, such as infarction, ventricular failure, or valvular pathology; or hydrostatic, in the context of excessive perioperative fluid therapy or increased porosity of pulmonary capillary beds owing to inflammatory processes (anaphylaxis, inhalational injury, sepsis, ARDS) or neurogenic causes (subarachnoid haemorrhage, brain injury, seizures, eclampsia).

Post obstructive pulmonary oedema (inaccurately also termed 'negative pressure' pulmonary oedema) is a specific anaesthesia complication that may occur after forced efforts at spontaneous ventilation against a closed (e.g. laryngospasm) or occluded (e.g. foreign body, bitten down on) airway. It can follow extubation if upper airway obstruction (obesity, obstructive sleep apnoea) is not recognized. It can be followed by severe hypoxaemia and oedema lasting considerably longer than the precipitating event and requiring standard treatment, including admission to ICU.

Management of pulmonary oedema involves sitting the patient up and applying high-flow oxygen with CPAP if awake, or PEEP if intubated and ventilated. Diuretics and nitrates reduce preload, shifting a failing heart leftwards on Starling's curve and improving pump function. Awake patients will experience significant distress for which low-dose opiates are traditionally employed. Twelve-lead ECG, bedside echocardiography and serial troponin assays are useful in determining the underlying cause. ◆

FURTHER READING

Birenbaum A, Hajage D, Roche S, et al. Effect of cricoid pressure compared with a sham procedure in the rapid sequence induction of anesthesia: the IRIS Randomized Clinical Trial. *JAMA Surg* 2019; **154:** 9–17.

Cook TM. Airway complications – strategies for prevention. *Anaesthesia* 2018; **73:** 93–111.

Cook TM, Woodall N, Frerk C, eds. 4th national Audit Project of the royal College of anaesthetists and the difficult airway society 2011. London: Royal College of Anaesthetists. ISBN 978-1-9000936-03-3 accessed 3rd July 2019, http://bit.ly/NAP-4.

Desciak MC, Martin DE. Perioperative pulmonary embolism: diagnosis and anesthetic management. *J Clin Anesth* 2011; **23:** 153–65.

Frerk C, Mitchell VS, McNarry AF, et al. Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. *Br J Anaesth* 2015; **115:** 827–48.

Higgs A, McGrath BA, Goddard C, et al. Guidelines for the management of tracheal intubation in critically ill adults. *Br J Anaesth* 2018; **120:** 323–52.

Lewis SR, Butler AR, Parker J, Cook TM, Smith AF. Videolaryngoscopy versus direct laryngoscopy for adult patients requiring tracheal intubation. *Cochrane Database Syst Rev* 2016; **11:** CD011136.

Neuberger Hale, Kerr Clarke, Wilson Reed, Hodge. JUDGMENT Montgomery (appellant) v lanarkshire health board (respondent) (Scotland) Lady Hale, Deputy President. London: The Supreme Court, 2015.

Royal College of Anaesthetists. Risks associated with your anaesthetic. Available from:. 2017. London: Royal College of Anaesthetists. accessed 3rd July 2019 https://bit.ly/RCoA-Risks.

Warner MA, Warner ME, Weber JG. Clinical significance of pulmonary aspiration during the perioperative period. *Anesthesiology* 1993; **78:** 56–62.