ELSEVIER

Revalidation FOR ANAESTHETISTS RCA Revalidation matrix
Matrix codes: 1H02, 1I03, 2A03, 2G01, 2G04, 3I00

BJA Education, 18(10): 310–316 (2018)

doi: 10.1016/j.bjae.2018.06.003
Advance Access Publication Date: 28 August 2018

Physical injuries during anaesthesia

D.W. Hewson and J.G. Hardman*

Anaesthesia & Critical Care, University of Nottingham, Nottingham, UK

*Corresponding author: j.hardman@nottingham.ac.uk

Learning objectives

By reading this article, you should be able to:

- Discuss the main risks of physical injury to the oral cavity and airway arising from general anaesthesia.
- Describe the risk factors, aetiology, and management of injury to the eye during anaesthesia.
- Explain the causes, presentation, and management of nerve injury arising from anaesthetic practice including those sustained during regional or general anaesthesia.

Key points

- Most physical injuries during anaesthesia arise from human error, equipment failure or improper use, or both.
- Dental injury is common and anaesthetists should be familiar with the management of dental fracture and avulsion.
- Suspected corneal abrasions warrant immediate ophthalmology review. Most abrasions heal with no long-term effects on vision.
- Prone positioning for major spinal surgery carries a significant risk of visual loss, but direct external compression on the globe is not necessary for this devastating complication to occur.
- Meticulous attention to patient positioning reduces the risk of pressure ulcers, nerve injury, and compartment syndrome.

Physical injury may arise in anaesthetic practice as a result of the actions (or inactions) of the anaesthetist, and typically arises as a result of extrinsic force applied to the patient, usually while they are unconscious or have altered sensation. Patients entrust their safety to anaesthetists whilst under their care, and it is the anaesthetist's duty to minimise the risk of physical injury to their patients.

The spectrum of injury during anaesthesia ranges from the relatively innocuous, to the rare and potentially fatal; physical injury during anaesthesia is a common cause for civil litigation. While the potential for harm through adverse drug reaction or equipment failure may not always be mitigated, many physical injuries occur as a result of human error. These injuries can be reduced in frequency and severity through education, training, and quality assurance. An understanding of the physical injuries that can arise in anaesthetic practice is essential in order to inform patients during the process of obtaining consent.

Causative factors

Human factors

Inadequacies in decision-making, situational awareness, workload management, team communication, and clinical leadership are common contributors to physical injury arising during anaesthesia. Poor working relationships, varying levels of training amongst staff, and challenging working conditions make failure more likely. Team training and simulation-based training are effective in reducing the incidence of this type of error. Insufficient training, inadequate experience, and poor preparation of the patient, environment or equipment, make physical injury in the presence of negative human factors more likely.[1]

David Hewson BSc (Hons) PGCert FHEA FRCA *is a consultant anaesthetist at Nottingham University Hospitals NHS Trust and clinical assistant professor at the University of Nottingham.*

Jonathan Hardman BMedSci (Hons) FRCA FANZCA DM *is head of anaesthesia and critical care research, University of Nottingham, consultant anaesthetist at Nottingham University Hospitals NHS Trust and associate editor-in-chief of the British Journal of Anaesthesia. He has published extensively on the medicolegal implications of anaesthesia.*

Accepted: 26 July 2018

When injury commences, effective monitoring and vigilance allow for a greater period for action before the injury becomes severe. During this period, when the injury, or potential for injury, is apparent but has not yet caused significant damage, the anaesthetist must act with precision, according to rehearsed protocols or drills.

Equipment failure

Failures of anaesthetic equipment, particularly gas supplies, breathing systems, airway devices, and mechanical infusion pumps, may result in significant injury to the patient. Meticulous checking of equipment before use is mandatory.

Patient factors

The occurrence of certain physical injuries is more common in the presence of specific patient comorbidities, which should be identified at preoperative assessment. For example, the incidence of dental damage during direct laryngoscopy is significantly higher in patients with pre-existing poor dentition or airway abnormalities.[2] Consent for anaesthesia should include appropriate estimation of the risk of injury in the context of the identified comorbidities and an outline of the steps available to mitigate those risks. It is an anaesthetist's duty to disclose material risks of physical injury as part of the consent process, the test of materiality being whether the particular patient (or a reasonable person in the patient's position) would be likely to attach significance to the risk.

Injury to the airway

Injury to the oropharyngeal cavity, teeth, larynx, or trachea is a well-recognised complication of general anaesthesia. Multiple incautious attempts at airway instrumentation and manipulation are a recurrent theme in cases involving traumatic airway injury, hypoxia, and death. Planned limits on the number of attempts at airway instrumentation are vital, especially in the context of difficult tracheal intubation.

Soft tissue injury

Soft tissue injury (e.g. mucosal abrasions) to the lips, tongue, or oropharyngeal mucosa can be identified after operation in approximately 50% of patients after general anaesthesia with direct laryngoscopy.[3] These injuries are almost all transient, responding well to simple ointments and oral hygiene. Although usually minor in the context of the overall postoperative recovery period, these injuries can be distressing, and a straightforward explanation of how they have occurred, and their likely healing time is useful for patients.

Approximately 40% of patients report a sore throat after tracheal intubation and 20% after laryngeal mask insertion.[4,5] Sore throat is not usually associated with identifiable physical injury and resolves within 48 h with simple analgesia and hydration. Symptoms persisting longer than this should trigger further investigation by the anaesthetist with support from ear, nose, and throat specialist colleagues to exclude laryngopharyngeal haematoma, perforation, granuloma, or nerve damage. Larger tracheal tubes, poor insertion technique, high cuff pressures, use of non-humidified breathing systems, duration of anaesthesia, and use of nasogastric tubes appear to increase the risk of postoperative sore throat. Recurrent laryngeal nerve palsy has been described after laryngeal mask insertion.[6]

Major soft tissue injury to the airway sustained during airway instrumentation includes perforating trauma to the pharyngeal, laryngeal, oesophageal, or tracheal walls. Such major injuries are rare [4th National Audit Project of the Royal College of Anaesthetists (NAP4) reported one case of life-threatening tracheal trauma as a result of the use of a bougie], but the risk of death after such injuries is 15–20%. Typically, perforation of the trachea is recognised within a few hours by the development of mediastinal and subcutaneous emphysema. Bronchoscopy and oesophagoscopy should be performed to confirm the diagnosis and exclude a concomitant anterior oesophageal perforation. Reported sites of oesophagopharyngeal perforation are the pyriform fossa, the hypopharynx posterior to cricopharyngeus muscle, and the posterior wall of the cervical oesophagus. The symptoms of such perforations are non-specific and include cough and pain, progressing to fever, dysphagia, and dyspnoea. Clinical findings include surgical emphysema, pneumothorax, pneumomediastinum, and signs of sepsis. The risk of death is reduced by early diagnosis and initiation of treatment; therefore, immediate surgical referral is required.

Dental injury

Fracture, dislocation, or avulsion of one or more teeth occurs in approximately 1% of general anaesthetics. In almost 90% of cases, injury is sustained to the (single-rooted) upper incisors. This is typically during tracheal intubation or extubation, often in patients with pre-existing poor-quality dentition (or fragile dental prostheses such as bridges or crowns), and more frequently in association with difficult intubation. Purpose-designed bite blocks inserted pre-emptively between the premolars reduce the risk of dental injury secondary to masseter contraction at the time of emergence from general anaesthesia. Oropharyngeal airways are often used for this purpose, but are themselves associated with dental injury.[7] The use of protective plastic guards for the teeth during anaesthesia is uncommon and is not evidence-based. Teeth may also be injured during the removal of tracheal tubes and supraglottic airways. These should never be withdrawn while the patient bites on the airway.

In a patient at high risk of dental injury (anticipated difficult intubation, poor dentition) a modification to the anaesthetic (e.g. the use of regional rather than general anaesthesia, or a supraglottic airway device rather than tracheal intubation) may be possible. Suitable non-urgent patients can be referred before operation for dental review.

In the event of dental fracture sustained during anaesthesia, the loose fragment must be identified and removed from the patient's airway and the patient counselled after operation on the event and the need for dental review. The fragment should be stored in saline or milk as it may be suitable for bonding after operation. Many hospitals have local policies to manage such events, including referral pathways. If a tooth is avulsed during anaesthesia, it should be immediately relocated into its socket, provided the socket is healthy and the patient is not immunocompromised, and firm pressure applied for several minutes. The patient's trachea should be extubated with the utmost caution given that further avulsion during extubation carries the risk of aspiration of the tooth into the trachea or oesophagus. An alternative is to store the avulsed tooth in saline or milk. In either case, the patient should be offered a clear explanation and an apology, and should see a dentist as soon as practical for consideration of dental reimplantation and splinting.

Injury to the eye

Injuries to the eye after non-ocular surgery occur in <0.1% of anaesthetics. Despite only accounting for 2% of claims in the ASA Closed Claims Project, eye injuries are associated with significantly larger financial settlements.

Independent risk factors for ocular injury include the lateral and prone positions, prolonged surgery, and surgery to the head or neck.

Corneal abrasion

Symptomatic corneal abrasions account for more than 50% of eye injuries associated with anaesthesia. Abrasions are caused by direct trauma, inadvertent chemical irritation, or exposure keratopathy. During general anaesthesia, tear production is reduced with a concomitant reduction in tear-film stability. This causes corneal epithelial drying and exposes the cornea to direct trauma. The inner surface of the upper eyelid becomes more adherent to the cornea, which can cause injury when the eye is re-opened. General anaesthesia also results in lagophthalmos from relaxation of the orbicularis oculi muscle, further increasing the vulnerability of the cornea.

Physical measures to prevent corneal abrasion include the apposition and taping of the upper and lower eyelids, instillation of ointment (e.g. methylcellulose or viscous gels), or bio-occlusive dressings. The only antiseptic skin preparation that is non-toxic to the eye is povidone-iodine 10% solution, and this should be used when the face is prepared for surgery. Chemical irritation of the cornea by passively regurgitated gastric contents while in a steep Trendelenburg position can be mitigated by premedication with proton pump inhibitors and meticulous taping of the eyes.

Symptomatic corneal abrasions cause pain, a sensation of grit in the eye, tearing, redness, and photophobia. Central abrasions may also result in a reduction in vision. The diagnosis is confirmed using fluorescein staining of the cornea under direct ophthalmoscopy or slit lamp examination.

If the eye has been exposed to irritant chemicals, it should be irrigated thoroughly with saline. Patients with suspected abrasions should be seen urgently by an ophthalmologist to perform an examination of the eye to assess the damage sustained, institute treatment, and organise follow-up. Treatment usually comprises topical antibiotics, lubricants, and occasionally patching of the eye to reduce pain on blinking. The most severe abrasions may be managed by a bandage contact lens. The majority of abrasions heal with no long-term effects on vision.

Visual loss

Postoperative visual loss (POVL) persisting for more than 30 days occurs in approximately 0.0008% (1 in 125,000) of patients undergoing non-cardiac surgery and 0.09% (1 in 1100) patients undergoing cardiac surgery. Of the POVL sustained during non-cardiac surgery, patients undergoing spinal fusion surgery in the prone position are particularly vulnerable, with an estimated risk of approximately 0.06% (1 in 1800).[8] Patients undergoing spinal surgery resulting in blood loss greater than 1000 ml and an anaesthetic duration greater than 6 h accounted for 96% of the cases of POVL reported after spinal surgery to the ASA POVL Registry.[9]

POVL is usually the result of ischaemic optic neuropathy (ION), retinal vascular occlusion, or rarely, cortical blindness. Patients at particular risk of ION after surgery include those of male sex, aged greater than 50 yrs, with coexisting anaemia.[8] The aetiology of postoperative ION is not yet clearly defined but is likely to be multifactorial and determined, at least in part, by normal variations in eye anatomy, and risk factors such as diabetes mellitus or atherosclerosis. In one registry analysis, 20% of the patients who developed POVL because of ION after spinal surgery were positioned in Mayfield pins, which suggests that direct compression of the globe is not a necessary precondition for ION to develop. Nevertheless, the horseshoe headrest has been implicated in many cases of direct eye pressure damage in the prone position. Its use in the prone position is therefore inadvisable.

POVL typically presents as painless loss of vision. Central retinal artery occlusion may reveal a pale retina with 'cherry red' spot on fundoscopic examination. Urgent referral to an ophthalmologist is required. The goal of treatment in neuropathic lesions is to reduce optic nerve fibre oedema with corticosteroids or osmotic diuretics, while maintaining a normal haemoglobin concentration and arterial blood pressure. Treatment options for retinal artery occlusion may include vasodilator therapy, increasing perfusion pressure, thrombolysis, reduction in red blood cell viscosity, and corticosteroids.

Injury to nerves

Nerve injury can occur during anaesthesia when a nerve is subjected to stretch, compression, hypoperfusion, trauma, or neurotoxic material. Injury to the nervous system is commonly ascribed to incautious patient positioning, or as a complication of regional anaesthesia; however, direct surgical incision and retraction are responsible for a significant proportion of the nerve injuries that occur while a patient undergoes surgery.[10]

Nerve injury after peripheral nerve blockade

Mechanisms of nerve injury after peripheral nerve blockade will be discussed in more detail in a separate forthcoming article in *BJA Education*. Intraneural injections are not recommended, but if intraneural injection does occur, then intrafascicular intraneural injection is far more damaging than extrafascicular intraneural injection.

It is difficult to accurately estimate the incidence of nerve injury because of the methodological heterogeneity of studies examining this rare complication. When consenting patients for peripheral nerve blockade, it is reasonable to explain that 0–2.2% of patients at 3 months, 0–0.8% of patients at 6 months, and 0–0.2% of patients at 1 yr will have symptoms suggestive of nerve injury.[11] The consent process should be individualised for specific patients. For example, patients with existing peripheral neuropathy should be counselled that postoperative nerve injury may be more common in this group.

There are several potentially modifiable anaesthetic factors that may influence the likelihood of nerve injury arising after regional anaesthesia. These are discussed in more detail elsewhere in this journal and summarised briefly below.

Nerve localisation technique

No particular technique of identifying the proximity of needle-tip to nervous tissue (elicited paraesthesia, nerve

stimulation, or ultrasound-guidance) has been found to reduce the incidence of nerve injury in clinical trials.[12]

Timing of peripheral nerve block

International guidance in adults is to perform both peripheral and central neuraxial blockade in awake patients, although there are circumstances when it may be safer to perform the block under deep sedation or general anaesthesia (e.g. paediatric practice, patients with movement disorders).[13] Awake patients may report paraesthesia and also early symptoms of local anaesthetic toxicity. If paraesthesia occurs, then needle advancement should stop and the needle repositioned.

Needle design

Short bevel, non-cutting (i.e. blunt-tipped) needles produce less frequent fascicular damage than long, sharp-tipped needles. Larger gauge needles produce more damage than smaller needles.

Injection pressure

High injection pressure is associated with intrafascicular intraneural injection. In-line manometer devices exist and are more accurate than subjective estimations of pressure injection, but whilst sensitive, these devices lack specificity and there is little evidence as yet for their use.

Local anaesthetic choice

Local anaesthetic applied directly to a nerve results in microscopic nerve fibre injury, oedema, and reduced neural blood flow resulting in neural injury. This effect is worsened by the addition of adrenaline (norepinephrine), which therefore should be used with caution in patients with pre-existing nerve injury or those at higher than normal risk of injury.

Nerve injury after neuraxial techniques

Life-changing and occasionally fatal injury can result from neuraxial blockade. Some 2.0–4.2 cases of permanent harm per 100,000 neuraxial blocks were reported by the 3rd National Audit Project of the Royal College of Anaesthetists (NAP3). The clear majority of such cases involve neuraxial blocks sited in the perioperative period (as opposed to pain management or obstetric settings).[14] When communicating the risk and benefits of neuraxial blockade to patients, the specific clinical circumstances must influence the conversation. Coagulopathy, pre-existing spinal disease, suspected or actual underlying infection, acute physiological status, immunosuppression, and the presence or absence of specific comorbidities (e.g. aortic stenosis), all have a major influence on the risk/benefit profile of the proposed technique.

Vertebral canal haematoma

The accumulation of blood in the intrathecal, subdural, or epidural space can mechanically impinge the spinal cord or nerve roots and cause permanent injury. Coagulopathy, pre-existing spinal canal abnormality, or traumatic needle insertion are implicated in many cases and national guidance should be consulted before undertaking neuraxial techniques in patients with abnormalities of coagulation.[15,16]

Radicular back pain, unexpected weakness, paraesthesia, or bowel or bladder dysfunction should alert to the possibility of vertebral canal haematoma. Prolonged loss of motor power or sensory blockade (particularly extending beyond the expected dermatomal distribution or that appears after initial block resolution) should trigger a high index of suspicion. Any infusions of local anaesthetic should be stopped, and a senior clinician should perform and document a full neurological examination every 30 min for no more than 4 h. If the neurological deficit does not resolve in that time, then the patient should undergo immediate whole-spine MRI. Emergency decompressive surgery within a 6–8 h period from symptoms to onset may result in recovery of neurological function.

Epidural abscess

Epidural abscess is a rare complication of neuraxial blockade. NAP3 reported 20 cases of epidural abscess out of 707,455 neuraxial blocks performed. Prolonged catheter placement, poor aseptic technique, underlying immunocompromise, or patient coagulopathy place patients at risk of abscess formation. Untreated active systemic or local infections are absolute contraindications to neuraxial techniques.

Radicular back pain (often with localised tenderness and presenting over a period of days to weeks), malaise, fever, sensory deficit, weakness, and abnormal bowel or bladder function after neuraxial needling, all suggest epidural abscess formation. Immediate MRI scanning is required to identify epidural pathology, but patients should be systemically resuscitated, relevant sites cultured, and systemic antibiotics commenced according to local microbiology advice. Staphylococcus (often methicillin-resistant) is the most commonly isolated pathogen. Surgical decompression and drainage, followed by a lengthy course of antibiotics, is routine in confirmed cases.

Chemical damage

Adhesive arachnoiditis (characterised by fibrous collagen band formation, inflammation and hyperaemia of the meninges, altered blood flow, abnormal CSF circulation, and syringomyelia) has been cited as a consequence of chlorhexidine contamination of the epidural or subarachnoid spaces. This has a poor prognosis and few effective treatments.[17,18]

National guidelines formulated in response to case reports conclude that '0.5% [alcoholic chlorhexidine] solution should be preferred over a 2% solution for skin asepsis before central neuraxial blockade'.[19] The isolation of chlorhexidine from neuraxial needling equipment, and the drying of antiseptic solution before needling, is also recommended. The use of 'open systems' (such as gallipots) containing potentially injectable solutions should be avoided during neuraxial blockade.[20]

Nerve injury cause by patient positioning

Nerve injury after incorrect patient positioning is an avoidable harm and can complicate cases involving sedation, regional, or general anaesthesia. Patient positioning requires meticulous planning and communication between surgical and anaesthetic teams. Sometimes compromise is required between the demands of surgical access and the requirement to minimise patient harm from incautious positioning. Vigilance is needed during positioning at the start of a case, but also during the course of surgery, as deliberate or inadvertent movement of the patient intraoperatively can result in nerve injury.

The preoperative consent process should disclose the risks of position-related nerve injury. If factors are present that lead to a significant risk of nerve injury (e.g. pre-existing mononeuropathy), or if the patient would be particularly affected by

an injury should it occur (e.g. a lower limb nerve injury in a professional football player) these should be specifically addressed. If peripheral neuropathy is present before operation, this should be carefully documented and the steps taken to ensure safe positioning explained in the medical record.

There are reports of injury to many nerves as a result of patient positioning, but the commonest injuries are to the ulnar nerve, common peroneal nerve, and brachial plexus.

Ulnar nerve injury

Ulnar nerve damage complicates 0.3–0.5% (1 in 215 and 1 in 385) of anaesthetics.[21] Many injuries occur in patients with preoperative subclinical abnormalities of ulnar nerve conduction. An important component of the postoperative assessment is therefore contralateral ulnar nerve neurophysiological testing, as an underlying subclinical ulnar nerve conduction abnormality may be identified. To reduce pressure on the cubital tunnel during anaesthesia:

(i) Avoid elbow extension with forearm pronation.
(ii) Avoid >90° elbow flexion and >90° shoulder abduction.
(iii) Ensure the non-invasive blood pressure cuff does not overly the cubital tunnel.
(iv) Provide soft external padding at the cubital tunnel.
(v) Maintain neutral forearm position with shoulders adducted.

Ulnar nerve injury presents with pain, or motor or sensory deficit in the distribution of the ulnar nerve. In approximately 50% of patients, there is some recovery of nerve function in the short term; however, the remaining 50% of patients are likely to continue to experience ulnar nerve abnormalities at 2 yr.[22]

Common peroneal nerve injury

Common peroneal nerve injury results in weak ankle dorsiflexion, weak foot eversion, and paraesthesia or pain in the dermatomal distribution of the nerve. Classically described as a complication of lithotomy positioning (where the nerve is compressed against the head of the fibula while patient's legs are held in stirrups), this nerve injury has also been reported as a consequence of knee arthroplasty. The incidence of common peroneal nerve injury is approximately 0.02% (1 in 4615) of cases undertaken in the lithotomy position.[23] To reduce the risk of nerve injury (and compartment syndrome) during lithotomy positioning:

(i) Ensure there is no external pressure on the common peroneal nerve as it travels in proximity to the proximal fibula.
(ii) Check that leg supports themselves do not compress the calves.
(iii) Lower the patient's legs (for a minimum of 15 min) at least every 3 h.

Brachial plexus injury

The inferior trunk of the plexus is at highest risk if shoulder abduction exceeds >90°, if the arms are positioned below the level of the torso when supine, or if the head is rotated to the contralateral side.

Superior and middle trunk injury occurs after external compression against the shoulder (e.g. by shoulder braces in the Trendelenburg position) exacerbated by contralateral neck flexion. Such injuries have been seen after surgery in the supine, lateral decubitus, Trendelenburg, and prone positions, and have an overall incidence of approximately 0.05% (1 in 2000).

During all cases of anaesthesia it is prudent to ensure that shoulder abduction does not exceed 90°, that head rotation and neck lateral flexion are not excessive, and that arm boards are level with the patient.

Injury to muscles and skin

Pressure ulcers

Sustained external pressure on any area of the body can cause reduced skin perfusion, ischaemia, and necrosis. Pressure exerted on bony prominences and in patients who are elderly, poorly-nourished, immobile, incontinent, or suffering from chronic disease is especially likely to cause pressure ulceration. Pressure ulceration has also been described in the obstetric setting, where multiple factors (relative immobility, body weight, incontinence) may heighten risk. The development of a pressure ulcer in the postoperative period may be secondary to events that occurred during the intraoperative phase; therefore it is essential, when positioning the patient, to dissipate pressure over as large an area as possible, use appropriate padding, and maintain vigilance over pressure points, particularly during prolonged surgery. Non-blanching redness is the first sign that skin has been poorly perfused; these suspect areas must be documented and observed closely in the postoperative period.

Compartment syndrome

Compartment syndrome complicates approximately 0.002% (1 in 50,000) of anaesthetics. The lower limbs are predominantly affected, and injury is associated with prolonged (>4 h) or incorrect leg placement in the Lloyd Davies or lithotomy positions. There are reports of compartment syndrome developing in the dependent upper limbs of patients placed lateral decubitus. Other causes are extravasation of fluid from misplaced or displaced i.v. or intraosseous cannulae, arterial tourniquets, and extrinsic compression from surgical instruments or team members.

The clinical diagnosis of compartment syndrome depends on a suitable index of suspicion in high-risk patients and recognition of the clinical signs and symptoms in the postoperative period (the cardinal sign being severe pain aggravated by passive stretching). Urgent orthopaedic referral is required in all suspected cases. Further management may involve osseofascial compartment pressure monitoring, or decompressive fasciotomy.

Tourniquet injuries

These injuries occur as a result of tissue compression and ischaemia. Underlying skin, nerve, muscle and blood vessels may all be affected.

Approximately 50% of injuries involve the skin, and typically include bruising and blistering of the skin at the distal edge of the cuff. Pressure necrosis or friction burns are produced by movement of poorly applied tourniquets. In addition, chemical burns have been reported when antiseptic solutions seep beneath tourniquets and are held against the skin under pressure.

With regard to nerve injury arising from tourniquet use, the radial nerve is most frequently affected in the upper limb, and the sciatic nerve most commonly in the lower limb.

Table 1 Measures to reduce the incidence of fire during surgery on the airway.

Avoidance of alcohol-based skin preparations
Use of laser-resistant tracheal tubes with cuffs filled with saline rather than air
Use of low inspired oxygen concentrations and avoidance of nitrous oxide
Minimal exposure of the airway to electrosurgical or laser techniques
Close liaison between anaesthetist and surgeon should high inspired oxygen be necessary

Longer inflation times and higher tourniquet pressures play a role in the development of pressure-related nerve injury.

The majority of tourniquet-related injuries are the result of equipment failure or improper use. Anaesthetists should be aware that faulty aneroid gauges can permit excessive pressures to be generated by pneumatic tourniquets. These devices should therefore be tested and calibrated at regular intervals.

Tourniquet cuffs should exceed the circumference of the limb by one third, and be applied to the widest part of the limb. The skin beneath the cuff should be well padded and free of folds. Once applied, the cuff should not be rotated into a new position and the distal edge of the cuff should be sealed to avoid seepage of skin antiseptics beneath the cuff. Tourniquets should be inflated to pressures of 40–80 mm Hg above the point at which an arterial pulse distal to the tourniquet is lost, and should be deflated for at least 10 min after a maximum of 120 min of inflation in healthy patients. In patients with chronic disease or acute physiological disturbance, the inflation time should be reduced. The presence of sickle cell disease, peripheral neuropathy, infection in the limb, and peripheral vascular disease or previous deep vein thrombosis are relative contraindications to the use of tourniquets.

Burn injuries

Most intraoperative burns are usually temporary and non-disabling; they are associated with warming devices, electrocautery, alcohol-based skin preparation, or a combination of these factors.

If the current pathway of monopolar diathermy is interrupted by incorrect placement of the dispersive plate, any points of contact between tissue and metal (e.g. ECG electrodes) may provide an alternate return pathway, potentially resulting in burns. The dispersive plate must therefore be in secure contact with dry, shaved skin, away from bony prominences, scar tissue, and metal implants. The use of protective quivers, audible signals from the electrocautery equipment when in use, and alarms to indicate a malfunctioning dispersive plate, are further protective measures. Electrocautery, especially monopolar diathermy, may alter the functioning of implanted medical devices (pacemakers, defibrillators, nerve stimulators) and the metallic components of these devices may themselves serve as return pathways for current, causing localised tissue damage.

However, burns to the face, airway, or both, are frequently disabling and are occasionally fatal; they therefore require special consideration. Such burns are usually associated with cautery or laser fires in the context of high fractions of supplemental oxygen, alcohol-based skin preparation solutions, or both. Recommendations for the prevention of fires during surgery on or near the airway are listed in Table 1.

Teams undertaking airway surgery should undergo regular training in the prevention and management of an airway fire. In the event of fire, ventilation should be paused and the oxygen supply to the airway disconnected. The fire should be extinguished using water or saline. Ventilation should resume using as low an inspired oxygen concentration as possible. A damaged tracheal tube should be replaced (and consideration should be given to replacing undamaged tubes) with a tube of lumen diameter 8 mm or greater to facilitate bronchoscopy to assess the extent of the airway burn and perform pulmonary toilet and bronchial lavage. If a significant burn has been sustained to the oropharynx or face, an uncut tube may be required to accommodate post-injury swelling.

Fire arising from medical oxygen cylinders and causing injury to patients, staff, or both, has been reported in the literature.[24] It is recommended that oxygen cylinder valves should be opened slowly to select the desired flow before being placed close to patients, and that the use of designated cylinder holders is preferable to placement directly onto mattresses or other combustible material.

Declaration of interest

JGH receives fees for civil, criminal and coronial medicolegal work, and is associate editor-in-chief of the BJA. DWH declares no conflict of interest.

MCQs

The associated MCQs (to support CME/CPD activity) will be accessible at www.bjaed.org/cme/home by subscribers to *BJA Education*.

References

1. Cook TM, MacDougall-Davis SR. Complications and failure of airway management. *Br J Anaesth* 2012; **109**: i68–85
2. Warner M, Benenfeld S, Warner M, Schroeder D, Maxson P. Perianesthetic dental injuries frequency, outcomes, and risk factors. *Anaesthesiology* 1999; **90**: 1302–5
3. Mourão J, Moreira J, Barbosa J, Carvalho J, Tavares J. Soft tissue injuries after direct laryngoscopy. *J Clin Anesth* 2015; **27**: 668–71
4. Biro P, Seifert B, Pasch T. Complaints of sore throat after tracheal intubation. *Eur J Anaesthesiol* 2005; **22**: 307–11
5. Higgins PP, Chung F, Mezei G. Postoperative sore throat after ambulatory surgery. *Br J Anaesth* 2002; **88**: 582–4
6. Jones FL, Hegab A. Recurrent laryngeal nerve palsy after laryngeal mask airway insertion. *Anaesthesia* 1996; **51**: 171–2
7. Falzon D, Foye R, Jefferson P, Ball DR. Extubation guidelines: Guedel oropharyngeal airways should not be used as bite blocks. *Anaesthesia* 2012; **67**: 919
8. Shen Y, Drum M, Roth S. The Prevalence of perioperative visual loss in the United States: a 10-year study from 1996 to 2005 of spinal, orthopedic, cardiac, and general surgery. *Anesth Analg* 2009; **109**: 1534–45
9. Lee L, Roth S, Posner K *et al.* The American Society of Anesthesiologists postoperative visual loss registry analysis of 93 spine surgery cases with postoperative visual loss. *Anaesthesiology* 2006; **105**: 652–9
10. Dwyer T, Drexler M, Chan VWS, Whelan DB, Brull R. Neurological complications related to elective orthopedic surgery. *Reg Anesth Pain Med* 2015; **40**: 443–54

11. Neal JM, Barrington MJ, Brull R *et al*. The second ASRA practice Advisory on neurologic complications associated with regional anesthesia and pain medicine. *Reg Anesth Pain Med* 2015; **40**: 401–30
12. Neal JM. Ultrasound-guided regional anesthesia and patient safety. *Reg Anesth Pain Med* 2010; **35**: S59–67
13. Bernards CM, Hadzic A, Suresh S, Neal JM. Regional anesthesia in anesthetized or heavily sedated patients. *Reg Anesth Pain Med* 2008; **33**: 449–60
14. Cook T, Counsell D. Major complications of central neuraxial block: report on the third national Audit Project of the Royal College of anaesthetists. *Br J Anaesth* 2009; **102**: 179–90
15. Horlocker TT, Wedel DJ, Rowlingson JC *et al*. Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy. *Reg Anesth Pain Med* 2010; **35**: 64–101
16. Harrop-Griffiths W, Cook T, Gill H *et al*. Regional anaesthesia and patients with abnormalities of coagulation. *Anaesthesia* 2013; **68**: 966–72
17. Miaki K, Matsui H, Nakano M, Tsuji H. Nutritional supply to the cauda equina in lumbar adhesive arachnoiditis in rats. *Eur Spine J* 1999; **8**: 310–6
18. Killeen T, Kamat A, Walsh D, Parker A, Aliashkevich A. Severe adhesive arachnoiditis resulting in progressive paraplegia following obstetric spinal anaesthesia: a case report and review. *Anaesthesia* 2012; **67**: 1386–94
19. Campbell J, Plaat F, Checketts M, Bogod D. Safety guideline: skin antisepsis for central neuraxial blockade. *Anaesthesia* 2014; **69**: 1279–86
20. NHS Improvement. *Restricted use of open systems for injectable medication*. 2016. Available from: https://improvement.nhs.uk/news-alerts/restricted-use-open-systems-injectable-medication/. [Accessed 5 June 2017]
21. Alvine F, Schurrer M. Postoperative ulnar-nerve palsy. Are there predisposing factors? *J Bone Jt Surg Am* 1987; **1**: 255–9
22. Warner M, Warner D, Matsumoto J, Harper M, Schroeder D, Maxson P. Ulnar neuropathy in surgical patients. *Anesthesiology* 1999; **90**: 54–9
23. Warner M, Martin J, Schroeder D, Offord K. Lower-extremity motor neuropathy associated with surgery performed on patients in a lithotomy position. *Anesthesiology* 1994; **81**: 6–12
24. Kelly FE, Hardy R, Hall EA *et al*. Fire on an intensive care unit caused by an oxygen cylinder. *Anaesthesia* 2013; **68**: 102–4