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REVIEW

Emergency care in facial trauma—a maxillofacial and ophthalmic perspective

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Vision threatening injury

Summary Facial trauma, with or without life- and sight-threatening complications, may arise following isolated injury, or it may be associated with significant injuries elsewhere. Assessment needs to be both systematic and repeated, with the establishment of clearly stated priorities in overall care. Although the American College of Surgeons Advanced Trauma Life Support (ATLS) system of care is generally accepted as the gold standard in trauma care, it has potential pitfalls when managing maxillofacial injuries, which are discussed. Management of facial trauma can arguably be regarded as "facial orthopaedics", as both specialities share common management principles. This review outlines a working approach to the identification and management of life- and sight-threatening conditions following significant facial trauma.

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"The boy lay for dead a while, and dozed longer. It appeared a strange sight at first to me, his face being beaten in, and the lower jaw sticking out ... there I saw the Os palati and the uvula beaten so close backwards .... Upon which I got up behind the uvula; then raising it a little upward, pulled it forward with the bone into its former place very easily". R. Wiseman (1622–1676) *Several Chirurgical Treatises*

Introduction

The aim of this review is to consider life- and sight-threatening conditions that may occur following trauma to the face. Although head injuries are commonly associated, primary and secondary brain injury is not included in this review, as this is a subject already extensively covered in the published literature, with well established, and recently updated, care pathways.[89]

In order to support this review, a National Library of Medicine's Medline database search was performed to identify the English language literature relevant to this topic. Key words and phrases used included "facial trauma", "facial injury", "airway obstruction", "life-threatening haemorrhage", "epistaxis", "emergency surgery", "emergency airway", "embolisation", "blindness" and "vision threatening injury". Whilst not an exhaustive review, we have endeavoured to make it representative of the literature. Where appropriate, we have also supplemented it with our own collective experiences of trauma management in our unit.

Facial trauma, with or without life- and sight-threatening complications, may arise following isolated injury, or it may be associated with significant injuries elsewhere.[3,121] Life- and sight-threatening complications may also occur following apparently trivial injuries, which may not immediately be evident on arrival in the resuscitation or emergency setting. Assessment needs to be both systematic *and repeated*, with the establishment of clearly stated priorities in the patient's overall care. However, these priorities may rapidly change as injuries, or events, evolve and become clinically apparent (for instance vomiting in the supine patient, or the development of shock in a patient with panfacial injuries).

In many countries, the American College of Surgeons Advanced Trauma Life Support (ATLS) system of care[5] is now generally accepted as the gold standard in the management of the injured patient. In patients with significant facial injuries, the ATLS approach has potential pitfalls and careful consideration of these is necessary. These dilemmas will be outlined further. True maxillofacial and ophthalmic "emergencies", as defined later, are uncommon. It is, nevertheless, important to be aware of their possible occurrence, particularly when known risk factors are identified, (for instance, patients on Warfarin therapy), of early warning signs and to appreciate how they can impact on the patient's overall management. If ever in doubt, reassess the patient.

In many respects, parallels can be drawn with orthopaedic surgery. Management of facial trauma can arguably be regarded as "facial orthopaedics", as both specialities share common management principles, notably an appreciation of the significance of associated soft tissue injury.[19,100,105,120]

What is an "emergency"?

Terminology can be confusing when defining clinical urgency. Interventions may be considered as "resuscitation", "emergency", "urgent" or "routine", although a degree of overlap may occur between some. In this review, we have focused on any clinical problem that requires *immediate identification and management to preserve life, or sight*. In this context, interventions may be either resuscitative or emergent, but not necessarily definitive treatments.

Following facial trauma, many conditions may be considered clinically "urgent" (e.g. contaminated wounds and open fractures), but these can be left until the patient has been fully stabilised, with little, or no, increase in mortality or morbidity.[124] With limb injuries, treatment of urgent conditions should be carried out within 6 h, with facial injuries this can be delayed further, if necessary. In the multiply injured patient, facial injuries need to be prioritised accordingly, taking into account other life- or limb-threatening injuries.[22]

In this review, "emergency" care effectively means airway care, control of profuse bleeding and the management of vision-threatening injuries (VTI). As previously mentioned, brain injury is not included in this review. In the absence of these complications, facial injuries can wait at least for a short while, during which the entire patient is assessed. Failure to recognise and manage these conditions rapidly can result in loss of life, or sight.

Facial injuries resulting in life threatening conditions include:

1. Facial injuries resulting in airway compromise (e.g. panfacial fractures with gross displacement, mobility, or swelling; comminuted fractures of the mandible; gunshot wounds; profuse bleeding; foreign bodies);
2. With such injuries it is important to remember that the following injuries may be associated;
3. Anterior neck injuries, resulting in airway compromise (e.g. penetrating injuries, laryngeal or tracheal injuries);
4. Injuries resulting in profuse blood loss (e.g. penetrating neck, facial fractures, however rare[21]).

ATLS and the maxillofacial region

All clinicians involved in trauma care should be competent in carrying out a primary survey and initiating resuscitative procedures. When managing facial injuries, this involves assessment and maintenance of the airway and control of obvious bleeding. Early consideration of vision-threatening injuries (for instance retrobulbar haemorrhage, or loss of eyelid integrity) is also important, but should not distract from the initial assessment and resuscitation. Although the aim of the primary survey is to identify and treat life-threatening problems, the early identification of a sight-threatening condition may be possible during "D" = disability (once "A", "B" and "C" have been addressed and the pupils are assessed). Although the primary reason for examining the pupils at this stage, (together with the Glasgow coma scale (GCS)), is to assess any neurological disability, associated ocular findings must also be noted to help identify "visual disability". This is a convenient time rapidly to assess the visual pathway, although it is not comprehensive. Recognition of vision-threatening injuries, based simply on the history, mechanism of injury, a high index of suspicion and gross clinical findings, is all that is required at this stage, rather than detailed evaluation, which will need to be undertaken later.

In the alert patient, it takes only a few seconds for a member of the trauma team to ask the patient if he/she can see clearly with each eye (in turn) during assessment of the GCS, and carefully to palpate the globes through closed eyelids. Early identification of a potential vision-threatening problem enables early referral to an appropriate specialist and initiation of treatment where necessary. When dealing with acute sight-threatening conditions, "appropriate" may mean an on-site speciality with expertise in periorbital trauma care, depending on the condition (e.g. retrobulbar haemorrhage).

Airway obstruction, bleeding and sight-threatening conditions may, at first, be subtle and may not become apparent until the secondary survey is under way. This reminds us of the two well-established principles in trauma care:

(i) The need for a high index of suspicion (often based on the mechanism of injury and known patterns of injury);
(ii) The need for frequent re-assessment of the patient.

Airway with control of cervical spine

In each trauma patient, the first priority is to assess the airway thoroughly, while at the same time protecting the cervical spine. In many cases, initial assessment simply requires a verbal response from the patient, often to questions like "what happened?" or "how do you feel?" Even in those patients who give an appropriate response, this should still be followed by direct inspection of the mouth and pharynx for loose, or foreign, bodies (after all, most of us can still talk with food or even chewing gum in our mouths), and signs of continuing bleeding. Even significant midface bleeding may not be obvious in the supine patient, if he/she is sufficiently conscious to swallow the blood. This will only be picked up by direct inspection of the pharynx — take a good look. Retropharyngeal haematoma, secondary to cervical spine injury, can also occasionally result in airway obstruction,[72] and its presence should be alert the examining clinician to the possibility of a cervical spine injury. In the conscious patient, if suction is required, this should be carried out carefully, as stimulation of the soft palate and pharynx can trigger vomiting.

The cervical spine should be immobilised, using either manual in-line techniques, or a hard collar, blocks and straps, unless the patient is agitated and extremely restless. In such circumstances, combative patients may only tolerate a hard collar. Too rigid immobilisation of the head in a struggling patient may simply create a fulcrum and, thereby, increased leverage on the neck as the rest of the body moves. If a patient fails to settle promptly despite adequate oxygenation, correction of severe hypovolaemia and appropriate pain relief, and does not allow essential investigations to be carried out, formal anaesthesia with intubation and ventilation must be considered. This is usually safer than sedating the patient in the absence of definitive airway control.

It is important to bear in mind that the "Airway" is not just the mouth, and that obstruction may occur at any point from the face to the carina. Many factors can contribute to airway compromise,[67] notably loss of consciousness. This is most commonly associated with alcohol overdose and brain injury. Obstruction may also arise from foreign bodies (chewing gum, sweets, dentures, teeth, blood and secretions), or displaced and/or swollen tissues.[6] The most common obstructing materials that threaten the airway in facial injuries are blood and vomit. Trauma to the front of the neck (bicycle injuries, automobile crashes, falls, sports injuries, clothesline injuries and hanging)[14,49] can also result in direct injury to the upper airway and an expanding haematoma due to arterial bleeding.

The potential for obstruction is present in almost all patients with significant facial injuries, due to pooling of blood and secretions in the pharynx, especially when supine. In most conscious patients this is simply swallowed — but will collect in the stomach. However, with midface fractures, particularly fractures of the mandible, swallowing may be painful and less effective in clearing the airway.[90] Early signs of obstruction may easily be missed, particularly when managing patients who are intoxicated, or who have an associated brain injury. Not only are these patients at risk of vomiting, but any reduction in consciousness impairs protective airway reflexes and care must be taken if these patients are positioned supine.

It is therefore important to identify oral, or nasal, bleeding even in the alert patient. If not, swallowed blood will accumulate in the stomach, resulting in nausea and a risk of unexpected vomiting (perhaps later on in the patient's care, when the patient is less well supervised by staff). Alcohol intoxication, commonly associated with facial trauma, is well known to result in both loss of consciousness and vomiting.

In the patient with significant facial injuries, who cannot sit up, two difficult decisions become necessary:

(1) Does the airway need securing? (i.e. using formal anaesthesia and intubation.)
(2) If so, how urgently?

Not all patients vomit, and once they are intubated further evaluation becomes difficult, often resulting in the need for a cerebral CT. On the other hand, supine patients can vomit unexpectedly and need to be kept under close observation, with the appropriate skills, and a clear plan of how, to manage vomiting. Early notification, by pre-hospital personnel, of a patient's impending arrival allows an appropriate team to assemble in advance. A senior experienced anaesthetist, or other clinician trained in advanced emergency airway management, should also be present during the assessment of these potentially problematic patients. A "difficult intubation trolley" should also be readily available in the resuscitation room.

Can I sit up?

If a patient has sustained major facial injuries, it is important to recognise the potential implications of repeated requests, or attempts, by the patient to sit up. This may indicate a desire to vomit, or unrecognised partial airway obstruction from swelling, loss of tongue support, or bleeding. Patients may try to sit forwards and drool, thereby allowing blood and secretions to drain from the mouth (Fig. 1). This position is at variance with ATLS teaching — "Proper immobilisation is achieved with the patient in the neutral position, i.e. supine, without rotating or bending the spinal column" ... "Cervical spine injury requires continuous immobilisation of the entire patient with a semi-rigid cervical collar, backboard, or tape and straps, before and during trans-

Figure 1 Patient with facial injury being allowed to sit up in order to drain blood and secretions. Struggling obstructed patients can come to more harm by being restrained than allowed to sit up if they insist.

fer to a definitive-care facility". Furthermore, sitting up will load the spine axially if the head is unsupported.

Careful assessment and judgement are again required in those patients with an apparently isolated, significant facial injury and careful log-rolling may be a useful solution. On occasions, allowing the patient to sit up may be appropriate, depending on the mechanism of injury and concern for other injuries, notably spinal. This decision is based on a risk/benefit analysis, i.e. the risk/benefit of keeping the patient supine with potential airway obstruction, versus the risk/benefit of axial loading of a potential spinal injury. The head still needs to be supported in order to minimise axial loads and, if possible, a hard collar should be applied. When multi-system injury is obvious, or suspected, attempts to sit up are even more problematic and if the patient is combative, despite adequate oxygenation, correction of severe hypovolaemia and appropriate pain relief, as outlined previously, early intubation and ventilation may be necessary to secure the airway. A senior anaesthetist, or other clinician trained in advanced emergency airway management, should be present. Whatever the circumstances, all efforts should be made to protect the cervical spine as best possible, using at least manual in-line immobilisation, even if this does not mean using a collar, or blocks and straps. However, patients should never be forced, or restrained, onto their backs — this is more likely to compromise both the airway and any occult spinal injury. Those who refuse to lie down may have to be managed, as best possible, on their sides (other injuries permitting), or sitting up. This is not easy but is achievable. Although the spine is not immobilised in a conventional position, the main consideration here is to maintain the airway without doing further harm, such as could occur with attempts to restrain the patient.

The significance of fractures and soft tissue swelling

Loss of tongue support and significant swelling may occur with bilateral ("bucket handle"), or comminuted, anterior mandibular fractures.[100] In the conscious patient, airway control may be possible, even if nursed supine. However, it is not secure. It is in the head-injured, or intoxicated, patient that loss of tongue control and other protective reflexes may rapidly become a problem. Comminuted (therefore, high energy) fractures of the mandible carry a greater risk, as there is very poor tongue support. Significant soft tissue swelling and intraoral bleeding may also occur in such injuries. Simple anterior mobile mandibular fractures may temporarily be reduced and stabilised by passing a "bridle wire" around one, or two, healthy teeth on either side of the fracture and tightening it.[100] This can be done using local anaesthetic. It reduces bleeding from the torn mucosa and, by immobilising the fracture, enables the patient to swallow more effectively.

Occasionally, displaced midface fractures may cause airway obstruction. The face can be regarded as a "crumple zone",[100] due to the presence of the sinuses. High energy impacts to the fragile middle third of the facial skeleton may therefore result in multi-fragmentary fractures that collapse backwards and downwards, along the inclined surface of the relatively resistant skull base. In some respects, the midface can be regarded as an airbag, absorbing much of the energy, which otherwise would have been transferred to the brain, although this concept has been challenged.[84] This crumpling and displacement can result in impaction of the posterior structures, notably the soft palate, which then swells, into the pharynx. Combined mandibular and middle third facial fractures are indicative of significant underlying injury, with a high risk of airway problems. Furthermore, such patients may have an associated brain injury, compounding the problem; they often bleed profusely, the blood being swallowed if awake, and they may develop gross soft tissue swelling. This type of injury emphasises the need for regular and repeated assessments, as airway obstruction, unexpected vomiting and hypovolaemic shock from unrecognised bleeding are all common consequences, which may not readily be apparent on initial presentation.

Significant soft tissue swelling usually occurs with major "panfacial" injuries, often necessitating prolonged intubation, or planned elective tracheostomy (Fig. 2). Airway-threatening swelling can occasionally occur in the absence of fractures, particularly in patients taking anticoagulants, or those with coagulopathies.[36,110] Posterior pharyngeal swelling may indicate an underlying cervical spine injury and can also lead to airway obstruction. Soft tissue injuries to the neck may also accompany facial injuries, and thereby contribute further to pharyngeal oedema and bleeding. Fractures of the hyoid bone,[66] usually visible on the lateral cervical spine film, should be regarded as a surrogate "marker" of significant injury (rather like fractures of the first and second ribs) and indicative of the risk of airway obstruction. It is important to remember that swelling, from whatever cause, can take several hours to develop. Clinicians need to be wary and regularly to re-examine the patient. Stridor is a particularly worrying sign and often necessitates urgent intubation. The alleged beneficial effects of steroids in the acute management of facial trauma have not been proven.

Figure 2 Patient with major maxillo-facial injury on ICU with tracheostomy, bite blocks, Foley catheters and packs (note facial swelling).

The anterior neck

This is often a forgotten site, particularly when the patient is in a hard collar, and requires careful and regular examination. If the patient arrives with a collar, this should be unfastened and the anterior neck examined, while maintaining in-line manual immobilisation of the cervical spine. In terms of clinical findings, the anterior neck can be regarded as a watershed between "Airway" and "Breathing" during the primary ATLS survey, as life-threatening problems in either can manifest clinical signs here. Pouiselle's law[1] indicates that even a small change in the radius of a tube can result in a significant change in flow through it. Although strictly applicable to fluid dynamics, his formula nevertheless highlights the potential for problems secondary to swelling within the larynx and trachea. Although unusual, fractures of the larynx and hyoid do occur and may lead to substantial swelling of the glottis, after a variable time.[53] A history of wearing a motorcycle helmet, of strangulation, or of contact sport injury are important pointers. A hoarse voice, haemoptysis, surgical emphysema, or fracture crepitus in the neck are highly suggestive of such injuries and should actively be sought. Carefully palpate the great vessels, hyoid and larynx for signs of injury and look for external swelling, which may reflect internal swelling.

The cervical spine

In the UK, there is much variation in how spinal injury is excluded, particularly in patients with concomitant brain injury. A comprehensive review of the literature and guidelines for the initial management and assessment of spinal injury has been published by a Working Party for the British Trauma Society.[91] The American College of Surgeons also teaches that "trauma occurring above the clavicle should raise a high suspicion for a potential cervical spine injury". For any mechanism of injury capable of causing cervical spinal injury, the safest policy is

[1] Pouiselle's law states that fluid flow through a tube (F) is proportional to the pressure gradient (DP/L = change in pressure over length), the radius (r) and the fluid viscosity. Flow in a tube can be increased by either increasing the tube's radius, decreasing the viscosity, and/or increasing the factor DP/L. The latter can be increased by either increasing the perfusion pressure, and/or decreasing the vessel resistance.

to assume an injury to be present, until it can be excluded according to accepted protocols.

Cervical spine injuries have been associated with maxillofacial injuries,[129] especially following high velocity trauma, with reported incidences between 1 and 6%[12,31,55,114] although this concept has been challenged.[56,92,126] Nevertheless, the confident exclusion of cervical spine injuries remains difficult,[33,45] Several patterns of cervical spine injury following facial trauma have been reported. Mandibular fractures may be associated with upper cervical spine injuries, while mid facial injuries may be associated with lower cervical spine injuries.[79] This may be related to the patterns flexion or extension of the neck at the moment of impact. Although interesting, this is of little practical importance in the resuscitation room and the best policy is to assume that spinal injury is present, until proven otherwise by clinical and radiological examination (Fig. 3). It is also important to remember that an unstable ligamentous injury can still occur despite normal radiographic appearances.[34]

In all trauma patients hard collars should fit properly; this is particularly important in patients with mandibular fractures, in order to prevent undue pressure on, and displacement of, the bony fragments. Furthermore, it has been suggested that hard collars may exacerbate intracranial hypertension in patients with severe head injury.[58] Although the clinical significance of this is yet to be established, the detrimental effects of increased intracranial pressure (ICP) in head-injured patients are well understood. For these reasons, the cervical spine needs to be "cleared" as soon as is practically possible, particularly if anaesthesia is required to repair any facial injuries. This is difficult in the presence of alcohol intoxication, brain injury, opiate administration, or distracting injuries.

Figure 3 Control of profuse midface bleeding, using bite blocks, foley catheters and nasal packs.

Airway maintenance techniques

All trauma patients should receive oxygen. Spontaneous control of the airway can quickly be lost and the airway may be very difficult to secure following facial trauma. With severe facial injuries, early involvement of an experienced anaesthetist is essential and should be anticipated well in advance of signs of impending obstruction. Occasionally, a surgical airway is required, notably when there is gross swelling from panfacial injuries, or inadequate mouth opening during attempts to intubate the patient. Members of the trauma team should be competent in performing this.

Several techniques exist for maintaining an airway.

- Suction;
- Jaw thrust;
- Chin lift;
- Oro- or/naso-pharyngeal airways;
- Tongue suture;
- Laryngeal mask.

It is important to appreciate that maintaining an airway is not the same as securing it and airway patency can still be lost. High volume suction, using a wide bore soft plastic sucker, should be readily available to clear the mouth, nose and pharynx of blood and secretions, taking care not to induce vomiting. Loss of the protective gag reflex indicates the need for endotracheal intubation.

The jaw thrust and chin lift are commonly used techniques, but may be difficult, although not impossible, to carry out in the presence of severely comminuted mandibular fractures. Following blunt trauma, if a chin lift is performed it is important not to extend the head into the "sniffing position". Care must also be taken not to distract the fractures as not only this is painful, but results in further blood loss and can tear mucosa. Both the chin lift and jaw thrust have been shown to produce movement of the cervical spine[38] and need to be performed with counter-support of the head to prevent this. If the patient is unconscious and has sustained a high impact anterior mandibular fracture, where tongue support has been lost completely, the use of a tongue suture, or pointed towel clip, may still be warranted to distract the tongue, facilitating suction and intubation. However, this technique is likely to cause bleeding.

Posteriorly displaced, middle third fractures may be reduced manually to improve the airway. Grasping the maxilla and pulling it anteriorly achieves this (Fig. 4). Disimpaction does not require much force, but it must be undertaken with protection of the cervical spine. Once reduced, it may be necessary to provide support with a mouth prop, as long as the patient has an intact lower jaw. Reduction has the additional benefit of controlling haemorrhage from middle third fractures.

Before 1990, the choice of airway device was essentially limited to the facemask, or an endotracheal tube. Since then, a number of airway devices has become available. The laryngeal mask airway (LMA) was introduced in United Kingdom in the late 1980s and has found widespread use in elective anaesthetic practice. Although cuffed to help maintain its position, it should be regarded as little more than an oro-pharyngeal tube in terms of its ability to protect the airway. Use requires specific training and it is not without complications. It can induce vomiting and placement can produce movement of the neck.[18]

None of these adjuncts provides a definitive and secure airway. The use of LMAs has been discouraged by the American College of Surgeons Committee on Trauma. Naso-pharyngeal airway, and naso-gastric, or naso-tracheal tubes, are generally regarded as contra-indicated in mid face injuries, or in suspected skull base fractures, following reports of accidental intracranial positioning.[5,29,48,64,107,108] The risk of this is said probably to be low, although this has been challenged.[50] Skull base fractures should be suspected in all midface injuries, particularly if there is periorbital ecchymosis (raccoon eyes), retro-auricular ecchymosis (Battle's sign), VIIth nerve palsy, or CSF leaks.

Figure 4 Traction on a depressed mid-face segment to establish airway patency.

Vomiting following facial injuries (before spinal clearance)

This is a particular problem in the management of facial injuries, and one that can occur with very little warning. It poses an obvious threat to the unprotected airway, especially in the presence of a possible cervical spine injury. Predisposing factors include not just recently ingested food and blood in the stomach, but also alcohol intoxication and brain injuries, both of which are common in facial trauma. Swallowed blood, which may go unrecognised in the conscious supine patient, also seems to be a potent trigger to nausea and vomiting.

Early warning signs may include repeated requests or attempts by the patient to sit up. These should not be interpreted simply as non-compliant behaviour secondary to drugs, alcohol, or brain injury. The difficulty arises in deciding which patients are at high risk of pulmonary aspiration after vomiting and therefore need to be intubated to secure the airway. This decision is even more critical if transfer, or imaging (notably CT), outside the resuscitation room is necessary. Anaesthesia and intubation are not without risks and continued sedation limits further clinical evaluation of other body regions, especially the abdomen, central nervous system and muscle compartments. Most patients with minor or moderate facial injuries do not vomit and the indication to secure the airway is usually not pressing. Senior anaesthetic assistance is required to evaluate the risk/benefits of intubation.

A clear and agreed plan of action is necessary when dealing with vomiting in the supine patient. If the patient has only just arrived and the head, chest, pelvis and legs are still securely strapped to a spine board, tilting the board head-down while clearing the airway is probably the safest way to secure the airway and maintain spinal protection. Tilting the board laterally is often suggested, but is awkward and puts the spine at risk if the straps are loose, or have already been released. After the straps have been removed, whether or not the patient is still on the spine board, tilting head down is still preferable to log rolling in response to vomiting. Log rolling is a *coordinated* technique requiring at least four individuals. When warnings signs are recognised and time allows this may be possible. However, in view of its unpredictable nature, vomiting can occur at any time and often well after the primary survey, by which time the trauma team may have dispersed. In our experience, vomiting is best managed by lowering the head of the trolley approximately 15–30 cms and applying high flow suction. This is a procedure that any clinician can do safely and single handedly, rather than struggle to roll the patient, or delay whilst waiting for help to arrive. Although somewhat messy and distressing to the patient, it is effective in clearing the airway and maintains spinal immobilisation. Patients who are still supine and in head-blocks should have an experienced nurse escort and suction with them at all times, particularly when they are taken out of the resuscitation room. They should also be observed at all times until the cervical spine is "cleared" and the blocks removed.

Definitive airway

A definitive airway is usually a *cuffed tube in the trachea* and may be required if there is any doubt about the patient's ability to protect his/her own airway, either immediately, or in the near future. The choice of definitive airway includes oro-tracheal intubation, naso-tracheal intubation and surgical cricothyroidotomy. All are relatively safe in experienced hands,[59] even in the presence of an unstable cervical spine injury, provided that the technique is one with which the clinician is skilled and confident.[81]

Oro-tracheal intubation with in-line cervical immobilisation is the technique of choice in the majority of cases. In-line cervical immobilisation has been demonstrated to be safe in the presence of an unstable cervical spine injury, although studies have shown that some movement of the cervical spine still occurs.[77,83] The choice of instrumentation may also be important in this respect.[43] The Bullard laryngoscope is a rigid, fibroptic device that minimises movement of the neck, but it takes longer to intubate than with the standard Mackintosh laryngoscope. Surprisingly, intubation is sometimes easier than anticipated in panfacial injuries, as the mobile facial bones can be displaced gently by the laryngoscope, providing an adequate view of the vocal cords. Difficult visualisation of the vocal cords occurs when there is continued bleeding and swelling of the pharyngeal walls. Despite this observation, it is prudent to be prepared for a surgical airway, in case airway control is not possible, and also to have a "difficult intubation trolley" to hand.[35] In the absence of midfacial, or craniofacial, fractures, alternative definitive airway techniques include blind naso-tracheal intubation, or fibre-optic assisted oro- and naso-tracheal intubation. Together with surgical airways, these techniques have been shown to be associated with less manipulation of the injured cervical spine.[44] However, they require extensive training and the use of fibre-optic assistance is often limited, as the view may be obscured by blood. Fibre-optic intubation in the conscious patient, although useful in spinal injuries, is not without risk,[85] particularly in an emergency setting. Naso-tracheal intubation is potentially dangerous in the presence of anterior cranial base fractures,[10,51] although this assumption has been challenged.[7] Retrograde intubation has also been described and shown to minimise cervical spine manipulation, but its use is not well established in the trauma setting.[11,61,115,125]

The only indication for creating a surgical airway is failure to secure the airway, within a safe time limit, by any other means. Surgical airways include needle cricothyroidotomy and surgical cricothyroidotomy. Surgical cricothyroidotomy is now advocated by the American College of Surgeons (ACS) Committee on Trauma as an appropriate alternative for emergency airway control, if endotracheal intubation is not possible. Tracheostomy is generally regarded as obsolete in the acute trauma setting as it is too time-consuming to perform and is potentially unsafe.[78] The need to convert cricothyroidotomy to a tracheostomy at a later date has also been questioned.[128] The key factor in performing a needle, or surgical, cricothyroidotomy is identification of the cricothyroid membrane, which should be possible, provided the anterior neck is not too swollen. Several surgical techniques are described and in principle these are straightforward, even for the inexperienced,[20,32,37,39] although not without complications.

Needle cricothyroidotomy may be used to provide some oxygenation while preparing for a surgical cricothyroidotomy. It is not a secure airway but may be used in extremis while a definitive (surgical cricothyroidotomy) airway is prepared. In conventional ATLS management, 15 l/min of O_2 is delivered by a Y-connector, or three-way tap device, with 1 s inspiration and 4 s expiration. This will only deliver 250 ml into the trachea during inspiration, some of which will pass up into the upper airway rather than into the lungs. Under these circumstances CO_2 control cannot be maintained. Expired gases pass via the patient's upper airway only, the dimensions of the cannula precluding any significant expiration through it. If there is total upper airway obstruction, it is necessary to reduce the oxygen supply to 2 l/min insufflation to avoid progressive hyperinflation. A jet injector device attached to the same cannula is an alternative way to maintain adequate ventilation. It is essential to check the cannula position carefully before attaching the device. If the cannula tip lies outside the tracheal lumen, the high driving pressure will cause massive surgical emphysema in the tissues which will make subsequent airway control impossible.

Breathing

In the context of isolated maxillofacial injuries, breathing problems may occur following aspiration of teeth, dentures, vomit and other foreign materials. Of course, if the patient has sustained multiple injuries, then other life-threatening ventilation or "B" problems should also be sought. Ventilation may also be impaired with high neurological injuries, secondary to spinal injuries or associated brain injury. This may necessitate intubation and ventilation. If teeth or dentures have been lost and their whereabouts unknown, a chest X-ray *and soft tissue view of the neck* should be taken to exclude their presence, either in the pharynx or lower airway.[15,119] Unfortunately, acrylic, from which "plastic" dentures are made, is not very obvious on a radiograph and a careful search is necessary. All foreign bodies need to be removed.

Circulation

Advanced Trauma Life Support teaches us that "any cold and tachycardic patient should be considered to be in hypovolaemic shock until proven otherwise". When hypovolaemic shock is present, facial injuries are unlikely to be the sole cause[21] and a careful search made elsewhere for occult bleeding (consider chest, abdomen, pelvis, retroperitoneum, limbs and on the floor). However, "severe" facial haemorrhage has been reported to occur in approximately 1 in 10 serious facial injuries.[41] Blood loss from the scalp, face and neck can be profuse and is usually obvious. Blood loss from midface fractures may not be recognised and can be difficult to control due to the extensive collateral blood supply, derived bilaterally from both the internal and external carotid arteries.[76]

Bleeding following facial trauma may be either revealed or concealed. Actively bleeding wounds, such as the scalp, can simply be closed with any strong suture to hand. A continuous technique is both quick and effective in haemostasis. In the scalp, full thickness bites are taken to close the aponeurosis, the layer on which the vessels predominantly run.[74,68] This is *not* a definitive closure, but simply an adjunct to "C" — control of haemorrhage. When significant bleeding is from the depths of a puncture wound (usually in the root of the neck) placing the tip of a urinary catheter into the wound and gently inflating the balloon has been suggested.[103] Care is required not to damage adjacent deeper structures.

Bleeding from comminuted fractures and soft tissue injuries can contribute to hypovolaemia and should be considered in all facial fractures. On occasion, what appears to be a simple broken nose can nevertheless continue to bleed and remain unrecognised in the supine patient. Any displaced fracture will bleed and in this respect fractures involving the tooth-bearing part of the mandible may also contribute to continued blood loss. Very often this is not torrential haemorrhage, but rather a constant trickle, which because the patient keeps swallowing the blood, is not immediately apparent (until he or she vomits!). Subsequent reassessments of the airway should therefore include a look for fresh blood in the pharynx and active bleeding from any oral wounds.

It is in the major midface and in panfacial injuries that blood loss can quickly become significant.[111] In these patients, blood loss is usually from multiple sites along the fracture planes and from associated soft tissues, rather than from a named vessel: this makes control of bleeding difficult. Significant *concealed* bleeding may occur in the supine patient, and should be remembered in cases of persisting shock. These patients often develop considerable soft tissue swelling and the airway may need to be secured early. If endotracheal intubation is required, blood loss may then become more apparent, as this is no longer swallowed and overspills from the mouth and nose. In addition to repeated clinical examination, arterial blood gas evaluations are particularly useful in the early detection of haemorrhagic shock. A significant base deficit often represents lactic acidosis and is an indication of tissue hypoperfusion. Many blood gas machines now measure lactate, in addition to base deficit. This is useful as large infusions of saline-based fluids can produce hyperchloraemic acidosis, which can result in a significant base deficit.

Shock management

Once "Airway" and "Breathing" have been addressed, the next priorities are to stop obvious and significant blood loss, and to establish wide-bore intravenous access, through which fluids may be given rapidly. *How much fluid* and *what type of fluid* are currently two areas of controversy in the literature, as the "permissive hypotension", "damage limitation surgery" and the "crystalloid versus colloid" debates continue. Guidelines for fluid therapy in the multiply injured patient have changed recently and current evidence seems to suggest that vigorous fluid administration in the presence of *uncontrolled* bleeding may be harmful. Uncontrolled haemorrhage generally refers to significant bleeding that requires immediate surgical, or radiological, intervention. In practical terms, this refers to massive haemothorax, mediastinal bleeding, and continued blood loss into the abdomen and pelvis. Haemorrhage from open wounds and limb fractures can quickly be brought under control in the emergency department with appropriate pressure and/or splinting. Instead of 1–2 l as an initial fluid bolus, smaller aliquots (250 ml in adults) with frequent re-evaluation have been recommended. Following blunt trauma, the aim is to maintain a minimal systolic blood pressure of 80 mmHg, or a palpable radial pulse, until bleeding is controlled. However, this approach is complicated in the presence of associated brain injury, where hypotension is harmful and an adequate cerebral perfusion pressure needs to be maintained.

Direct pressure, clips and sutures may all be used to control obvious external bleeding, as described previously for the scalp. When displaced midface fractures are present, manual reduction not only improves the airway, but is frequently effective in controlling blood loss from the fracture sites, although it may be difficult to achieve anatomical reduction in comminuted fractures. Once reduced, a mouth prop helps to maintain reduction. This technique is comparable in limiting haemorrhage to the reduction of a displaced femoral fracture, or closing an "open-book" pelvic fracture. Oral bleeding may be controlled with local gauze packs and manual reduction of any displaced fractures in the dental arches, using the teeth as a guide (if firmly attached). Once reduced, a bridle wire can then be placed as previously described. The amount of blood loss may be over estimated, as patients often salivate profusely.

Epistaxis, either in isolation or associated with midface fractures (once these have been reduced and supported by a mouth prop), may be controlled using a variety of nasal balloons, or packs. If the source of nasal bleeding is in the posterior nasopharynx, urinary catheters can be passed via both nostrils into the pharynx (under direct vision), inflated with saline and then gently withdrawn until the balloons wedge into the post-nasal space. Judgement is again required with pan facial injuries due to the risk of cranial intubation. However, in those patients with profuse haemorrhage a risk/benefit analysis is needed, as gentle passage of a soft catheter safely, under direct vision, is often possible. When unstable midfacial fractures are present, inflation of the balloons may displace the fractures further, thereby increasing blood loss. Temporary stabilisation of the reduced fractures using a mouth prop is therefore necessary *before* inflation is attempted. If the mandible is also fractured, this needs additional stabilisation. Light anterior nasal packs, or nasal tampons, can then be placed.

These manoeuvres should be regarded solely as resuscitative measures, as analogous to wrapping a sheet around a reduced open book pelvis. The fractures are not anatomically reduced and nasal packs are not without risk.[60] Sinusitis, meningitis and brain abscess are all potential complications, although the role of antibiotic prophylaxis is not clear. Blindness has even been reported.[47]

Surgical intervention

In the presence of persistent haemorrhage, despite appropriate interventions, remember to consider coagulation abnormalities, either preexisting (e.g. haemophilia, chronic liver disease, Warfarin therapy), or acquired (e.g. dilutional coagulopathy from blood loss, or DIC). Emergency surgical intervention may be required as part of the primary survey. If so, it is important to remember that the secondary survey has not yet been performed (although some aspects will have been covered in the immediate search for the source of blood loss) and this must be documented and communicated to the receiving teams. Depending on the overall clinical picture, the patient's haemodynamic status, degree of continuing blood loss and index of suspicion for other injuries, further investigations may be required immediately prior to, during, or following surgery (for instance chest X-ray, pelvic X-ray, CT of the head, or CT/FAST ultrasound of the abdomen).

Following induction of anaesthesia and intubation, manual reduction of facial fractures can be carried out more readily and effectively, if not already accomplished, as previously described. At all times, the cervical spine must be carefully immobilised. The hard collar will have been removed to facilitate intubation and manual in line immobilisation must be continued until the collar, blocks and straps have been replaced. If facial fracture reduction proves to be effective in controlling bleeding, maintaining it manually for a short period provides the anaesthetist with time to "catch up" with fluid administration, if necessary.

The optimal time definitively to repair facial fractures is not known, although it has been suggested that better outcomes may be possible with earlier, or immediate, repair. This need has to be balanced against the patient's overall condition. If not undertaken immediately, repair can be deferred safely, which, depending on the clinical picture and need for further investigations, may be up to several weeks. If this is felt appropriate, a tracheostomy may be required at the end of any surgery, depending on the degree of anticipated swelling. This allows the patient to be woken up for further assessment, with a secure airway. In a multiply injured, or unstable patient, facial fracture repair does not need to be undertaken immediately and rapid temporary reduction and stabilisation of mobile, bleeding fractures, supplemented where necessary by nasal and oral packs, is effectively a form of ''damage limitation surgery''. This avoids the risks of prolonged anaesthesia and surgery in a sick patient and allows earlier transfer to an intensive care unit for further resuscitation. However, if definitive care is deferred in a very sick patient, the development of severe organ failure may preclude later facial injury repair surgery within the optimal time frame. This may have to be accepted. Prolonging immediate surgery may increase the risk of multi-organ failure.

In conventional damage control surgery for abdominal injuries, planned secondary and definitive surgery is undertaken at 48 h. With major facial injuries, a longer delay may be necessary before definitive repair. This may be necessary to allow swelling to resolve, further imaging, investigations and assessment (for instance visual pathways), planning and informed consent to be undertaken.

There are various ways to stabilise facial fractures temporarily, using wires, splints or plating techniques if fracture sites are exposed, or using the patient's occlusion if one jaw is uninjured — intermaxillary fixation. External fixation of facial injuries, although not as frequently used as it was 30 years ago, is also very effective in providing rapid 'first aid' stabilisation in the multiply injured patient, or where there are limited facilities, prior to transfer to a definitive care centre. With gunshot wounds, or other types of contamination, this method also provides good 'long-term' temporary fixation, until the contaminated wounds have healed. External fixators are also particularly useful in maintaining space and orientation in continuity defects.[124]

If bleeding continues despite reduction of facial fractures and packing, further interventions may involve ligation of the external carotid, and often the ethmoidal, arteries, via the neck and orbit, respectively.[115,122] Most of the literature on this subject relates to isolated nasal epistaxis, rather than facial trauma. Because of the extensive collateral supply ligation may be necessary on both sides.[130] Alternatively endoscopic techniques, such as transantral and intranasal approaches, have been described.[40,99,101,127] These are of limited use in panfacial fractures, where multiple bleeding points may be present, both in bone and soft tissues. These techniques are best used in localised nasal injuries resulting in uncontrollable epistaxis.

Supra-selective embolisation

This is increasingly being reported as an effective alternative to surgical ligation in life-threatening facial haemorrhage. The use of supra-selective embolisation in trauma remains controversial, but has been reported to be very successful, with certain obvious advantages over surgery. It is increasingly used in extremity trauma and bleeding secondary to pelvic fractures,[1,28,117] and is now well documented as a successful treatment method in penetrating injuries,[16] blunt injuries and intractable epistaxis.[82,88] Catheter-guided angiography is used, first to identify and then to occlude the bleeding point, or points. Embolisation involves the use of balloons, stents, coils, or chemicals.[69] Supra-selective embolisation can be performed without the need for a general anaesthetic and, in experienced hands, is relatively quick. Its value, therefore, is seen in the unstable patient. Multiple bleeding points can be identified precisely and the technique is repeatable. However, immediate access to facilities and on site expertise are essential. Complications include iodine sensitivity and, following extensive embolisation, end organ ischaemia and subsequent necrosis. Stroke and blindness have also been reported.

Vision-threatening injuries (VTI)

These include:

- Retrobulbar haemorrhage;
- Traumatic optic neuropathy;
- Open and Closed globe injuries;
- Loss of eyelid integrity;
- Chemical injury.

Trauma accounts for more than a million people world-wide who have been blinded bilaterally, and unilateral blinding injuries have an estimated annual incidence of 500,000 cases. This makes trauma one of the leading causes of unilateral loss of sight.[27,94,102] In the United States alone, the cost of ocular trauma is estimated at $200 million per annum, with 900,000 reported cases of occupation-related eye injuries alone.[75] Eighty percent of injuries occur in men, with a median age of 27 years. Although the majority of eye injuries are accidental, there is a worrying trend in assaults' accounting for more than 20% of cases, many of which are drug and alcohol related.[65]

Loss of sight following blunt facial trauma may be crudely considered to be due to the following mechanisms.

- Direct injury to the globe;
- Direct injury to the optic nerve, e.g. bony impingement;
- Indirect injury to the optic nerve, e.g. deceleration injury resulting in shearing, stretching forces;
- As a result of a generalised or regional fall in tissue perfusion (anterior ischaemic optic neuropathy, retrobulbar haemorrhage, nutrient vessel disruption);
- Loss of eyelid integrity.

The most common presentation is blindness immediately post injury, although delayed visual loss is also well documented.[30,46] All patients with craniofacial, or midfacial, injuries should be started on regular, specific eye observations, in addition to any head injury observations. Ideally, all patients with craniofacial trauma and suspected eye injury should be reviewed by an ophthalmologist.[95]

Visual acuity testing and colour perception are said to be the most appropriate and useful clinical tests to recognise and document any loss of vision.[2] However, these require a patient who is fully awake and co-operative. Visual assessment in the unconscious patient is extremely difficult. It is in these patients that early and possibly treatable threats to sight may be missed. Clinical assessment usually falls initially to the assessment of pupillary size, reaction to light and globe tension on gentle palpation. The presence of a relative afferent pupillary defect (RAPD) is regarded as a sensitive clinical indication of visual impairment. Initial fundoscopy is difficult to perform without dilating the pupil and may be misleadingly normal as the optic nerve takes time to atrophy. However, it may be possible to detect intra-ocular haemorrhage, retinal oedema, retinal detachment, or swelling of the optic disc. The role of visual evoked potentials has been reported as a useful adjunct in early detection,[42,63] although the authors have no experience of this.

Ocular injuries are common following facial trauma,[17] notably with injuries sustained to the upper face and forehead. The bony orbit is deficient anteriorly and to a variable degree laterally and offers the eye little protection from trauma to the face. However, a number of additional factors are said to protect the globe from trauma, which may explain why blindness following facial injuries is uncommon. These include the prominence of the periorbital bones, the globes' resilient structure and patients' mechanisms of self-protection (such as turning the head, raising the hands and reflex eye closure).[131] Injury to the optic nerve itself is uncommon. As the nerve enters the orbit, the optic foramen is a dense ring of bone. This is believed to offer some protection by encouraging fractures in this region to propagate *around* the foramen, rather than into it.[8] The nerve itself is relatively slack within the orbit, but is tethered as it passes through the foramen, a site where shearing forces may come into play.[80]

Ocular injuries range from simple corneal abrasions to devastating injuries resulting in total and irreversible loss of sight. Injuring forces necessary to damage the globe may leave the periorbital tissues relatively unscathed, and unless specific attention is directed to the eye, sight-threatening injuries can easily be missed. Penetrating injuries may occur from small, high velocity missiles at the time of the incident. This must be considered when the history suggests the presence of broken glass, wood, or metal fragments at the scene, or examination of the patient reveals eyelid, or periorbital, lacerations.

Ruptured globes and perforations with retained intra-ocular foreign bodies must be considered in all craniofacial injuries. Because of the close relation between the structures within the anterior and middle cranial fossae and the orbit, (separated only by some of the thinnest bones in the body), intracranial injury must also be considered in all penetrating orbital injuries, even if not immediately apparent. Suspicion of penetrating brain and eye injuries is based on the history, mechanism of injury and clinical findings. These should be considered in all high velocity penetrating orbital injuries.

All ocular injuries require immediate ophthalmic referral. Visual loss is the next priority, once life and limb-threatening problems have been addressed. Arguably, vision-threatening injuries are just as important as limb-threatening problems, especially if they are present bilaterally. Both impede rehabilitation and dramatically reduce the quality of life.

Retrobulbar haemorrhage

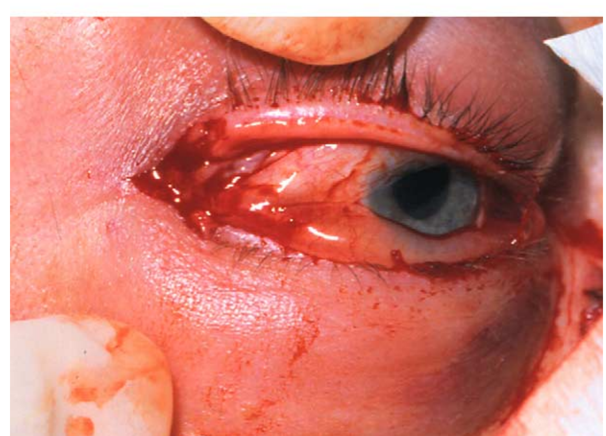
Retrobulbar haemorrhage (RBH) is usually a clinical diagnosis, and needs to be treated as soon as possible. It is effectively a compartment syndrome within the orbit and, as such, should be managed with the same degree of urgency (if not more so) as muscle compartment syndromes elsewhere (e.g. the lower limb). Irreversible damage has been estimated to occur following only 60 min of ischaemia.[4,9,57,106] Raised intra-orbital pressure is caused by bleeding and associated oedema that is contained behind the relatively unyielding orbital septum. Bleeding can occur within, or outside, the ''cone'' formed by the recti muscles, an intra-conal bleed being more severe. As the pressure rises, it compresses the ophthalmic and retinal vessels, resulting in retinal ischaemia. Untreated retrobulbar haemorrhage can result rapidly in blindness and ophthalmoplegia. In most cases, symptoms develop within a few hours after injury, but can occur much later.[113] Regular eye observations should be continued, although for how long they should be observed is not known. Pragmatically speaking, if the patient is unable to report symptoms following craniofacial trauma, due to anaesthesia or brain injury, eye observations should be continued until the patient can communicate. Retrobulbar haemorrhage is also known to occur following repair of mid-face fractures.

Retrobulbar haemorrhage must be treated as soon as possible, but its presence should *not* distract from the initial assessment and resuscitation of the entire patient. A convenient time rapidly to assess the eyes is when the pupils are assessed as part of the Glasgow Coma Scale. Pain, proptosis, loss of vision and the presence of an afferent pupillary defect are the principal features for which to look. Other clinical findings are eyelid oedema, chemosis (conjunctival oedema) and ophthalmoplegia. In unconscious, or agitated, patients it may not be possible to assess visual acuity and they may not complain of pain. A tense, proptosed globe and a dilated pupil may be the only clues to the presence of a retrobulbar haemorrhage. Careful interpretation of a dilated pupil is required, as this may also represent significant brain, *or* ocular, injury, *or both*. The Glasgow Coma Scale should also be recorded, as this will usually help to indicate significant brain injury. CT scanning of the brain and the orbits should be carried out in uncertain cases. This will demonstrate severe proptosis, stretching of the optic nerve and a tented posterior sclera in retrobulbar haemorrhage.

In those patients in whom visual loss may be reversible, and who are well enough, the management of retrobulbar haemorrhage is surgical. The aim of treatment is to decompress the orbit, thereby restoring retinal perfusion. Medical treatments and a lateral canthotomy may ''buy time'', while preparing the patient for surgery. High-dose intravenous steroids, acetazolamide (250—500 mg) and mannitol (1 g/kg) are started before surgery and continued after surgery until the globe pressure is seen to be falling.

A lateral canthotomy, with lateral canthal tendon division, can be performed under local anaesthesia in the emergency setting. Lignocaine 1%, with adrenaline (1 in 200,000), is injected into the lateral canthal area of the affected eye, the lateral canthus incised to the orbital rim and the canthal tendon identified and cut. The lower eyelid is then pulled forward and its lateral attachment to the orbital rim divided.[112] This allows the globe to translate forward, partially relieving the pressure by increasing the retrobulbar volume (Fig. 5). If necessary, the same procedure can also be applied to the upper eyelid laterally. Formal decompression is then carried out under a general anaesthesia. The orbital and intra-conal space is entered, allowing the blood and oedema fluid to escape via a drain, which is left in situ. Various approaches are possible, the infra orbital approach being the most commonly used.

(a)



(b)

Figure 5 (a, b) Lateral canthotomy to decompress retrobulbar tension.

Traumatic optic neuropathy

Traumatic optic neuropathy occurs in between 0.5 and 5% of closed head injuries, and visual loss is permanent in approximately half of these patients.[118] It occurs when injuring forces, transferred to the optic canal, result in damage to the optic nerve. Stretching, contusion, or shearing forces can injure the nerve as it passes through the relatively thick and unyielding canal, into the orbit. These initial insults also initiate a cascade of molecular and chemical mediators, which cause secondary vasospasm and vaso-occlusion, oedema, and necrosis. This can result in intraneural compression, vaso-occlusion and a local form of compartment syndrome, which is initially reversible. However, this progresses to arterial obstruction and irreversible infarction.[116,123]

Deceleration injuries and blunt trauma to the face and head are common causes of traumatic optic neuropathy and motor vehicle collisions, falls and assaults account for the majority. Displaced cranio-orbital fractures and associated oedema can also compress the nerve directly, compromising its vascular supply. Less commonly, nerve sheath haematoma, or complete transaction, can occur. When complete transaction has occurred, no treatment is possible, but with all other injury mechanisms early treatment may result in a degree of visual sparing or recovery. Clinically, the different types of injury cannot be distinguished and CT scanning is usually necessary.

Initial diagnosis of traumatic optic neuropathy is clinical. Loss of consciousness following a significant head injury is a common association. Visual loss is usually profound and almost instantaneous, but it can be partial and delayed. Clinical findings that suggest an optic nerve injury include decreased visual acuity and a relative afferent pupillary defect. Bruising, or oedema of the eyelids can make examination difficult, and with a swollen, visually impaired eye other causes of reduced vision, such as retrobulbar haemorrhage, or open and closed globe injuries, should also be considered. When the eye appears normal, but there is reduced vision and an afferent pupillary defect, injury to the nerve near the optic canal should be suspected. Optic nerve avulsion, or nerve compression resulting in nerve head swelling or central arterial and venous occlusion are readily recognisable on fundoscopy. Visual fields and colour vision testing are usually not possible, as vision is so poor. Although visual evoked potentials have been shown to be helpful in making the diagnosis, this is not widely available. CT imaging will demonstrate optic canal fractures and MRI can show soft tissue swelling and sheath haematoma.

Traumatic optic neuropathy needs immediate ophthalmic referral and treatment must be initiated as soon as it is recognised. Treatment is controversial and may be medical, or surgical. Medical treatment aims to reduce the oedema and inflammation that contributes to nerve ischaemia. Better outcomes have been shown if steroids are given within 8 h of the injury. Intravenous methylprednisolone, at a dosage of 30 mg/kg over 30 min, followed by 15 mg/kg 6 hourly over 2 days, is one treatment regimen. If there is clinical improvement, then a reducing dose of oral prednisolone, 80 mg → 60 mg → 40 mg → 20 mg (each dose for 3 days) is started, if the patient is able to take oral drugs. Stress ulcer prophylaxis should be considered. The role of surgical decompression is controversial, but is generally reserved for patients who fail to respond to steroid treatment, in whom visual recovery is felt possible.[117,119] Surgical approaches include trans-ethmoidal, transcranial, or via a lateral orbitotomy, depending on the surgeon's preferences, expertise and resources available, and the individual circumstances of the patient.

Open and closed globe injuries

International standardisation of the terminology and classifications used in ocular trauma has been developed to help to overcome problems in interpreting research from different centres.[71,97] "Open" globe injury refers to a full thickness wound in the corneo-scleral wall of the eye. This may be caused by blunt trauma (globe rupture), or by a sharp object (laceration, or penetrating and perforating injury, with or without a retained intra-ocular foreign body). A "closed" globe injury does not have a full thickness wound in the eye wall and includes lamellar lacerations, superficial foreign bodies and contusion of the globe.

Four separate variables are important in classifying globe injuries.

- Mechanism of injury.
- Grade of injury. This is determined by the visual acuity as measured by the Snellen chart.
- The presence or absence of a relative afferent pupillary defect in the injured eye.
- Which "zone" of the eye involved in the injury. (Zone I is the cornea up to the corneal limbus. Zone II is the area extending back from the limbus for 5 mm and includes injury to the iris, lens and the ciliary body. Zone III is all structures posterior to zone II including retina, optic nerve, choroid and the presence of a vitreous haemorrhage.)

Generally speaking, a poor initial visual acuity, presence of a relative afferent pupillary defect and posterior involvement of the eye all carry a bad prognosis. This holds true for both closed and open globe injuries.[23,24,98,118]

Vision-threatening globe injuries may not be obvious and a high index of suspicion is required. Lid laceration, subconjunctival haemorrhage, bruising and oedema are all commonly associated. Blood-stained tears may indicate the possibility of an open globe injury. With an open globe injury, the eye looks collapsed and uveal tissue (Fig. 6), retina and the vitreous gel may be seen prolapsing out of the eye. A hyphaema (Fig. 7) and vitreous haemorrhage are usually present and the lens may be damaged and cataractous. The intra-ocular pressure is low and aqueous fluid may be seen to leak from the wound when fluorescein drops are instilled. In cases of small high velocity objects (metal and glass fragments) the eye may appear intact and a small entry wound easily overlooked (Fig. 8a—c). The history is therefore important in indicating the possibility of an intra-ocular foreign body (IOFB). IOFBs may be visible if the view of the fundus is clear. Ultrasound scan has been shown to be useful in detecting globe rupture, IOFBs, retinal tears and retinal detachment, if the view of the fundus is poor.[93,104] Care must be taken not to apply pressure to the eye during examination, as this can expel further ocular contents in an open globe injury.

With closed globe injuries, the eyelid injuries and subconjunctival haemorrhage can be similar to those of open globe injuries. However, the globe looks formed and the intra-ocular pressure is usually high, due to blood blocking of the trabecular meshwork in the drainage angle. Iris sphincter muscle tears, iris dialysis, hyphaema and a displaced lens may be present. Vitreous haemorrhage, choroidal ruptures, retinal commotio and tears leading to a retinal detachment may be visible, if the view of the fundus is clear.

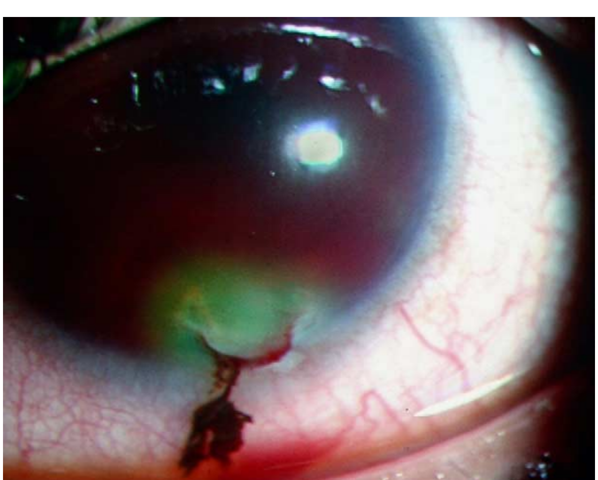


Figure 6 Uveal prolapse following penetrating injury of the globe.

Figure 7 Hyphaema — a blood level in the anterior chamber of the eye.

Management of globe injuries depends of whether the injury is open or closed. Analgesia and anti-emetics should be administered and the patient's tetanus status checked. A hard plastic shield should be taped over the eye to stop rubbing in open globe injuries, especially in children. Primary surgical repair of the open globe should be performed under general anaesthesia, as soon as possible and no later than within 24 h after trauma. Depolarising agents can result in tetanic extraocular muscle contractions, which can expel ocular contents, and should be avoided. During repair, the full posterior extent of the wound should be explored and closed using non-absorbable sutures.[26,87] Foreign bodies are usually removed at the same time. Intravenous ciprofloxacin, or vancomycin and ceftazidime combination, are thought to reduce the risk of endophthalmitis. A later vitrectomy is often required to clear vitreous haemorrhage, in order to prevent proliferative vitreo-retinopathy and consequent tractional retinal detachment. Post-operative management aims to control inflammation, infection, pain and intra-ocular pressure, while the eye settles. Closed globe injuries are managed with steroid, antibiotic, cycloplegic and anti-hypertensive eye drops, also aiming to control the same factors.

Careful follow-up is required, as endophthalmitis, retinal detachment, glaucoma, cataract and retinal membrane formation can all occur. The prognosis depends on the initial degree of damage to the globe and whether any of these complications arises. Corneal scarring and irregular astigmatism lead to poor vision.

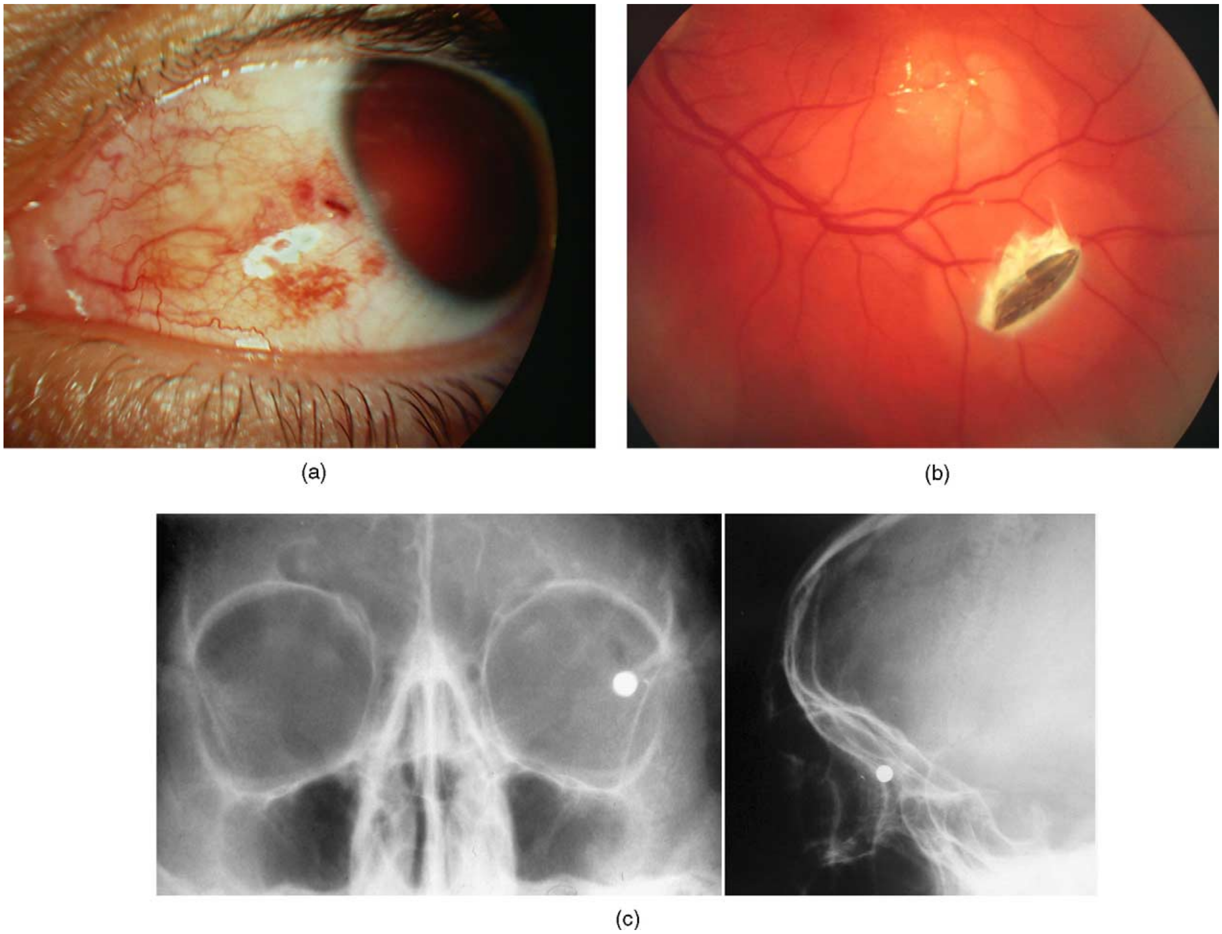


Figure 8 (a) Minute entry wound belies the severity of this open globe injury. (b) An intra-ocular foreign body. (c) X-rays of an intra-ocular foreign body.

Sympathetic ophthalmitis is a rare complication occurring in 0.1% of ocular perforations. It is characterised by uveitis in the healthy eye, developing more than 14 days after trauma, and can lead to loss of vision. Early repair of the injured eye has reduced the incidence of this complication and the role of early enucleation remains controversial. Most surgeons aim to preserve the globe after trauma for both the visual potential and better cosmesis.

Loss of eyelid integrity

Inability effectively to close the eyelids rapidly results in desiccation of the cornea, ulceration and potentially loss of sight. Even relatively minor eyelid lacerations may predispose to this and are easily overlooked. Avulsion of the eyelids is a rare, but devastating injury and extremely difficult to reconstruct. Furthermore, eyelid lacerations may indicate serious underlying ocular injury.

Orbital and ocular adnexal injuries are present in approximately one quarter of open globe injuries and are associated with a worse prognosis for visual acuity.[54] In the presence of eyelid lacerations, assessment and management of the underlying globe is more important than that of the eyelid. Closed globe injuries, globe rupture and blow out fractures of the orbit are likely, following blunt trauma. Penetrating ocular, orbital and brain injuries, with or without retained foreign bodies, must be excluded if there is a history of sharp, or high velocity missile, injury. Bite injuries are usually associated with loss of tissue and are contaminated.[13]

Visual acuity, visual fields, colour vision, ocular movement, the pupil and the fundus should be examined in all patients with eyelid lacerations. The position, length and depth of the wound(s) should then be documented. Medially sited eyelid injuries can damage the lachrymal drainage system and require special attention. Upper lid injuries may affect the levator muscle and its function should be noted. Full neurological examination is required if penetrating brain injury is suspected, or in the presence of altered consciousness. A small lid laceration may be the entry wound for a significant penetrating globe, orbital or cranial injury and may conceal a large retained foreign body. Failure to detect damage to the underlying structures is the main source of error when evaluating lid lacerations. Plain orbital X-rays may reveal fractures and retained foreign bodies, but CT scan is the investigation of choice if the history suggests a significant risk of the above.

The timing of surgery depends on the general condition of the patient and the presence of other injuries. Repair of lid lacerations can safely be deferred for up to 48 h, so long as the eye is protected, if other injuries take precedence. However, if unprotected, the cornea can dry very quickly, resulting in an epithelial defect, ulceration and loss of vision. This is especially important in the unconscious patient. Under these circumstances, until the defect is repaired, eyelid remnants should be pulled over to provide corneal cover, if necessary using a traction suture. Liberal application of chloramphenicol ointment, or artificial tears should be administered and the whole area covered with a wet sterile gauze swab. If a delay in repair is expected, the wound should be cleaned and superficial foreign bodies removed. Copious amounts of saline irrigation under pressure (using a 20 ml syringe and 18-gauge cannula) can be used to wash out foreign bodies and reduce microbial load. Intravenous antibiotic cover (e.g. Co-amoxiclav 500 mg tds) is needed for all bite injuries and contaminated wounds. As indicated above, the patient's tetanus status should be checked.

Most simple lacerations can be explored and cleansed under local anaesthesia and then closed in layers. Care must be taken to ensure that suture ends do not rub the cornea and cause abrasions. Many shallow cuts can appose without the use of sutures. They scab over and heal extremely well, as the lid is very vascular. Complex lacerations, including any involving the lid margin, lateral and medial canthal regions, medial third of the lids and levator muscle, must be referred for specialised repair. These lacerations can disrupt the lachrymal drainage system and functional integrity of the lid, and require detailed understanding of the functional and cosmetic anatomy of the region. Full details of surgical techniques are covered elsewhere.[25,52,73,86] As the eyelids are very vascular, even necrotic looking and avulsed tissue can survive and, therefore, no tissue should be excised.[62] Adequate cosmetic and functional results can be achieved, but it may require further operations.

Chemical injury

Chemical injuries account for about a tenth of all ocular injuries.[70] Mostly, they are mild, with no significant long-term effects, but a small proportion lead to blindness. Chemicals that have a pH different from that of the eye (which is pH 7.4) can cause a burn. Of these, alkalis cause more damage than acids, as they break down lipid membranes and penetrate deeper. Alkalis also account for the majority of chemical injuries.[96] The greater the pH difference, the more concentrated the solution and the longer the contact time, the more damage is caused. The type of chemical involved is also important. Ammonium hydroxide causes more damage than sodium or calcium hydroxide, as it penetrates faster. Solid particles (lime and cement) can lodge in the conjunctival fornices and have a prolonged contact time. Damage to the conjunctival vascular endothelium leads to ischaemia and necrosis of the ocular surface, with loss of epithelial stem and goblet cells. Domestic and industrial accidents, and assault are the commonest causes of chemical injury. Many household cleaning detergents contain sodium hydroxide.

Patients present with severe pain, blepharospasm, watering and variable reduction in vision. A corneal abrasion, opacity and limbal ischaemia are present if a significant injury has occurred. All eyes must receive local anaesthetic drops, pH evaluation and irrigation with copious amounts of Ringer lactate (at least 2 l), started immediately. Water, although widely available, is hypotonic to the cornea and therefore movement of fluid into the stroma by osmosis risks deeper penetration of the chemical. Ringer lactate is better that saline, as it also buffers the solution rather than just diluting it; furthermore, saline, although isotonic, has a supraphysiological sodium level, and its contact with all tissues should be avoided, if possible. Try to obtain the pH of the offending chemical and establish the baseline pH of both eyes. Irrigation must continue until the pH is normal. All efforts must be made to look for, and to remove, particulate matter from the conjunctival sac. First aid lavage of the eye is the most significant factor in the prognosis for the outcome of the chemical eye insult.[109]

Immediate referral to an ophthalmologist should be made, once the first aid measures have been started. Further management with intensive, topical potassium ascorbate, antibiotics, steroids, cycloplegia and oral Vitamin C usually requires admission under specialist ophthalmic care. The clinical grading of such burns centres around the degree of limbal ischaemia affecting the eye. The prognosis for vision is good if less than a third of the limbal circumference is ischaemic. Results are poorest if more than three-quarters of the limbus is lost. In such an event, loss of vision results from severely dessicated eyes, corneal scarring and vascularisation, cataract, glaucoma and uveitis. In the long term, limbus stem cell transplantation and replacing the ocular moisture is essential for visual rehabilitation. A corneal transplant can then be performed if the ocular surface environment is adequate to maintain its clarity, following the above measures.

Conclusions

Fortunately, life- and vision-threatening maxillofacial emergencies are uncommon. However, they do occur in well-defined high risk groups and, as such, it is important that clinicians maintain a high index of suspicion and treat these emergencies accordingly. The best outcome for these traumatised patients is associated with treatment by a multi-disciplinary trauma team, which includes a maxillofacial surgeon who has experience of these conditions.

References

1. Agolini, Shah SF, Kamalesh, Jaffe J, Newcomb J, Rhodes M, Reed JF. III, Arterial embolization is a rapid and effective technique for controlling pelvic fracture hemorrhage. J Trauma: Inj Infect Crit Care September 1997;43(3):395—99.
2. Al-Qurainy A, Stassen LFA, Dutton GN, Moos KF, El-Atta A. The characteristics of midfacial fractures and the association with ocular injury: a prospective study. Br J Oral Maxillofac Surg 1991;29:291—301.
3. Alvi A, Doherty T, Lewen G. Facial fractures and concomitant injuries in trauma patients. Laryngoscope 2003;113(1):102—6.
4. Amagasaki, Tsuji, Nagaseki. Visual recovery following immediate decompression of traumatic retrobulbar haemorrhage via transcranial approach. Neurol Med Chir (Tokyo) 1998;38:221—4.
5. American College of Surgeons. Advanced Trauma Life Support Program for Doctors: ATLS. 6th ed. Chicago, IL: American College of Surgeons; 1997.
6. Ardekian L, Rosen D, Klein Y, et al. Life-threatening complications and irreversible damage following maxillofacial trauma. Injury 1998;29(4):253—6.
7. Arrowsmith JE, Robertshaw HJ, Boyd JD. Nasotracheal intubation in the presence of frontobasal skull fracture. Can J Anaesth 1998;45(1):71—5.
8. Babajews A, Williams JLl. Blindness after trauma insufficient to cause bony injury: case report and review. Br J Oral Maxillofac Surg 1986;24:7—11.
9. Bailey, Kuo, Evans. Diagnosis and treatment of retrobulbar haemorrhage. J Oral Maxillofac Surg 1993;51:780—2.
10. Bahr W, Stoll P. Nasal intubation in the presence of frontobasal fractures: a retrospective study. J Oral Maxillofac Surg 1992;50:445.
11. Barriot P, Riou B. Retrograde technique for tracheal intubation in trauma patients. Crit Care Med 1988;16(7):712—3.
12. Beirne JC, Butler PE, Brady FA. Cervical spine injuries in patients with facial fractures: a 1-year prospective study. Int J Oral Maxillofac Surg 1995;24:26—9.
13. Beadles KA, Lessner AM. Management of traumatic eyelid injuries. Semin Ophthalmol 1994;9(3):145—51.
14. Bent III JP, Silver JR, Porubusky ES. Acute laryngeal trauma: a review of 77 patients. Otolaryngol Head Neck Surg 1993; 109:441—9.
15. Blaschke U, Cheng EY. Foreign body in upper airway. Unsuspected cause of obstruction. Postgrad Med 1989;86(3): 235—7.
16. Borsa JJ, Fontaine AB, Eskridge JM, et al. Transcatheter arterial embolization for intractable epistaxis secondary to gunshot wounds. J Vasc Interv Radiol 1999;10(3):297—302.
17. Brandt MT, Haug RH. Traumatic Hyphema: a comprehensive view. J Oral Maxillofac Surg 2001;59:12.
18. Brimacombe J, Keller C, Kunzel KH, et al. Cervical spine motion during airway management: a cinefluoroscopic study of the posteriorly destabilized third cervical vertebrae in human cadavers. Anesth Analg 2000;91(5):1274—8.
19. Brinker, Bailey MR, Daniel Jr E. Fracture healing in tibia fractures with an associated vascular injury. J Trauma: Inj Infect Crit Care January 1997;42(1):11—9.
20. Brofeldt BT, Panacek EA, Richards JR. An easy cricothyrotomy approach: the rapid four-step technique. Acad Emerg Med 1996;3(11):1060—3.
21. Bynoe RP, Kerwin AJ, Parker 3rd HH, et al. Maxillofacial injuries and life-threatening hemorrhage: treatment with transcatheter arterial embolization. J Trauma 2003;55(1): 74—9.
22. Cannell H, Dyer PV, Paterson A. Maxillofacial injuries in the multiply injured. Eur J Emerg Med 1996;3(1):43—7.
23. Cascairo MA, Mazow ML, Prager TC. Paediatric ocular trauma: a retrospective survey. J Pediatr Ophthalmol Strabismus 1994;31:312—7.
24. Casson RJ, Walker JC, Newland HS. Four-year review of open eye injuries at the Royal Adelaide Hospital. Clin Exp Ophthalmol 2002;30:15—8.
25. Chang EL, Rubin PA. Management of complex eyelid lacerations. Int Ophthalmol Clin 2002;42(3):187—201.
26. Colby K. Management of open globe injuries. Int Ophthalmol Clin 1999;39(1):59—69.
27. Congdon NG, Friedman DS, Lietman T. Important causes of visual impairment in the world. JAMA 2003;290(15):2057—60.
28. Cook RE, Keating JF, Gillespie I. The role of angiography in the management of haemorrhage from major fractures of the pelvis. J Bone Joint Surg Br 2002;84(2):178—82.
29. Cornett MS, Paris Jr A, Huant TY. Case report: intracranial penetration of a nasogastric tube (letter). Am J Emerg Med 1993;11:94—6.
30. Cullinane, Reddy, Bass, et al. Anterior ischaemic neuropathy. J Trauma July 1999;47(1):210.
31. Davidson JS, Birdsell DC. Cervical spine injury in patient with facial skeletal trauma. J Trauma 1989;29:1276—8.
32. Davis DP, Bramwell KJ, Vilke GM, et al. Cricothyrotomy technique: standard versus the rapid four-step technique. J Emerg Med 1999;17(1):17—21.
33. Davis JW, Phreaner DL, Hoyt DB, Mackersie RC. The etiology of missed cervical spine injuries. J Trauma 1993;34:342—6.
34. Demetriades D, Charalambides K, Chahwan S, et al. Nonskeletal cervical spine injuries: epidemiology and diagnostic pitfalls. J Trauma: Inj Infect Crit Care April 2000;48(4):724—7.
35. Diaz JH. The difficult intubation kit. Anesthesiol Rev 1990; 17(5):49—56.

36. Diecidue R, Richard J, Spera J, et al. Post-traumatic haemorrhage in a patient with previously undiagnosed Von Willebrand's disease. J Oral Maxillofac surg 2000;58:3.
37. DiGiacomo C, Neshat KK, Angus LD, et al. Emergency cricothyrotomy. Mil Med 2003;168(7):541—4.
38. Donaldson 3rd WF, Heil BV, Donaldson VP, Silvaggio VJ. The effect of airway maneuvers on the unstable C1—C2 segment. A cadaver study. Spine 1997;22(11):1215—8.
39. Eisenburger P, Laczika K, List M, et al. Comparison of conventional surgical versus Seldinger technique emergency cricothyrotomy performed by inexperienced clinicians. Anesthesiology 2000;92(3):687—90.
40. El-Guindy A. Endoscopic transantral sphenopalatine artery ligation for intractable posterior epistaxis. Ann Otol Rhinol Laryngol 1998;107:1033—7.
41. Frable MA, El-Roman N, Lenis A, Hung JP. Hemorrhagic complications of facial fractures. Laryngoscope 1974;84: 2051—7.
42. Gellrich NC, Zerfowski M, Eufinger H, et al. A new diagnostic schedule and reliable treatment plan for traumatic optic nerve lesions. Mund Kiefer Gesichtschir 1998;2(Suppl 1): 107—12.
43. Gerling MC, Davis DP, Hamilton RS, et al. Effects of cervical spine immobilization technique and laryngoscope blade selection on an unstable cervical spine in a cadaver model of intubation. Ann Emerg Med 2000;36(4):293—300.
44. Gerling MC, Davis DP, Hamilton RS, et al. Effect of surgical cricothyrotomy on the unstable cervical spine in a cadaver model of intubation. J Emerg Med 2001;20:1—5.
45. Gerrelts BD, Petersen EU, Mabry J, Petersen SR. Delayed diagnosis of cervical spine injuries. J Trauma 1991;31:1622—6.
46. Ghufoor, Sandhu, Sutccliffe. Delayed onset of retrobulbar haemorrhage following severe head injury: a case report and review. Injury 1998;29:139—41.
47. Giammanco P, Binns M. Temporary blindness and ophthalmoplegia from nasal packing. J Laryngol Otol 1970;84: 631—3.
48. Gianelli Castiglione A, Bruzzone E, Burrello C, et al. Intracranial insertion of a nasogastric tube in a case of homicidal head trauma. Am J Forensic Med Pathol 1998;19:329—34.
49. Gold SM, Gerber ME, Shott SR, et al. Blunt laryngotracheal trauma in children. Arch Otolaryngol Head Neck Surg 1997;123:83—7.
50. Goodisson, Shaw DW, Snape GM, Leslie. Intracranial intubation in patients with maxillofacial injuries associated with base of skull fractures? J Trauma: Inj Infect Crit Care February 2001;50(2):363—6.
51. Hall DB. Nasotracheal intubation with facial fractures. JAMA 1989;261:1198.
52. Harstein ME, Fink SR. Traumatic eyelid injuries. Int Ophthalmol Clin 2002;42(2):123—34.
53. Hartmann PK, Mintz G, Verne D, Timen S. Diagnosis and primary management of laryngeal trauma. Oral Surg Oral Med Oral Pathol 1985;60(3):252—7.
54. Hatton MP, Thakker MM, Ray S. Orbital and adnexal trauma associated with open-globe injuries. Ophthal Plast Reconstr Surg 2002;18(6):458—61.
55. Haug RH, Wible RT, Likavec MJ, Conforti PJ. Cervical spine fractures and maxillofacial trauma. J Oral Maxillofac Surg 1991;49:725—9.
56. Hills MW, Deane SA. Head injury and facial injury: is there an increased risk of cervical spine injury? J Trauma 1993; 34(4):549—53. discussion 553—4.
57. Hislop, Dutton, Douglas. Treatment of retrobulbar haemorrhage in accident and emergency departments. Br J Oral Maxillofac Surg 1996;34:289—92.
58. Ho AM, Fung KY, Joynt GM, et al. Rigid cervical collar and intracranial pressure of patients with severe head injury. J Trauma Inj Infect Crit Care 2002;53(6):1185—8.
59. Holley J, Jorden R. Airway management in patients with unstable cervical spine fractures. Ann Emerg Med 1989;18(11):1237—9.
60. Holmes S, Coghlan K, McAllinden P, Hardee P, Chan O. Complications with use of the Epistat in the arrest of midfacial haemorrhage. Injury 2003;34:901—7.
61. Hung OR, al-Qatari M. Light-guided retrograde intubation. Can J Anaesth 1997;44(8):877—82.
62. Hurwitz JJ, Kratzky V. Dog and human bites of the eyelid respond with retrieved autogenous tissue. Can J Ophthalmol 1991;26:334—7.
63. Ikejiri M, Adachi-Usami E, Mizota A, et al. Potentials in traumatic optic neuropathy. Ophthalmologica 2002;216(6): 415—9.
64. Junsanto T, Chira T. Perimortem intracranial orogastric tube insertion in a pediatric trauma patient with a basilar skull fracture. J Trauma 1997;42:746—7.
65. Katz J, Tielsch JM. Lifetime prevalence of ocular injuries from the Baltimore Eye Survey. Arch Ophthalmol 1993;111: 1564—8.
66. Kaufman HJ, Ciraulo DL, Burns RP. Traumatic fracture of the hyoid bone: three case presentations of cardiorespiratory compromise secondary to missed diagnosis. Am Surg 1999; 65(9):877—80.
67. King HK. Airway managements of patients with maxillofacial trauma. Acta Anaesthesiol Sin 1996;34(4):213—20.
68. Kirolles S, Haikal FA, Saadeh FA, et al. Fascial layers of the scalp. A study of 48 cadaveric dissections. Surg Radiol Anat 1992;14(4):331—3.
69. Komiyama M, Nishikawa M, Kan M, et al. Endovascular treatment of intractable oronasal bleeding associated with severe craniofacial injury. J Trauma 1998;44(2): 330—4.
70. Kuckelkorn R, Schrage N, Keller G, et al. Emergency treatment of chemical and thermal eye burns. Acta Ophthalmol Scand 2002;80(1):4—10.
71. Kuhn F, Witherspoon D, Heimann k, et al. A standardized classification of ocular trauma. Ophthalmology 1996;103: 203—4.
72. Kuhn JE, Graziano GP. Airway compromise as a result of retropharyngeal hematoma following cervical spine injury. J Spinal Disord 1991;4(3):264—9.
73. Kulwin DR, Kersten RC. Eyelid laceration repair. In: Tse DT, editor. Colour atlas of ophthalmic surgery: oculoplastic surgery. Philadelphia, PA: JB Lippincott; 1992. p. 27—34.
74. Last RJ. Anatomy, regional and applied, 6th ed. Churchill Livingston; 1978.
75. Leads from MMWR: leading work-related diseases and injuries-United States. JAMA 1990;251:2503—4.
76. Leiugh F, Neil-Dwyer G, Rowe EF. Primary care. In: Rowe NL, Williams JL, editors. Maxillofacial injuries. New York: Churchill Livingstone; 1994. p. 65—92.
77. Lennarson PJ, Smith D, Todd MM, et al. Segmental cervical spine motion during orotracheal intubation of the intact and injured spine with and without external stabilization. J Neurosurg 2000;92(Suppl 2):201—6.
78. Lewis RJ. Tracheostomies: indications, timing and complications. Clin Chest Med 1992;13:137.
79. Lewis VL, Manson PN, Cerullo Lj, Meyer PR. Facial injuries associated with cervical fractures: recognition, patterns and management. J Trauma 1985;25:90—3.
80. Lipkin AF, Woodson GE, Miller RH. Visual loss due to orbital fracture: the role of early reduction. Arch Otolaryngol Head Neck Surg 1987;113:81—3.

81. Lord SA, Boswell WC, Williams JS, et al. Airway control in trauma patients with cervical spine fractures. Prehosp Disaster Med 1994;9(1):44—9.
82. Mahmood S, Lowe T. Management of epistaxis in the oral and maxillofacial surgery setting: An update on current practice. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2003;95:23—9.
83. Majernick TG, Bieniek R, Houston JB, Hughes HG. Cervical spine movement during orotracheal intubation. Ann Emerg Med 1986;15(4):417—20.
84. Martin 2nd RC, Spain DA, Richardson JD. Do facial fractures protect the brain or are they a marker for severe head injury? Am Surg 2002;68(5):477—81.
85. McGuire G, el-Beheiry H. Complete upper airway obstruction during awake fibreoptic intubation in patients with unstable cervical spine fractures. Can J Anaesth 1999;46(2):176—8.
86. McNab AA, Collin JRO. Eyelid and canthal lacerations. In: Linberg JV, editor. Oculoplastic and orbital Emergencies. Norwalk, CT: Appleton & Lange; 1990. p. 1—13.
87. Mittra RA, Mieler WF. Contraversies in the management of open-globe injuries involving the posterior segment. Surv Ophthalmol 44:215—25.
88. Moreau S, DeRugy MG, Babin E, et al. Supraselective embolisation in intractable epistaxis: review of 45 cases. Laryngoscope 1998;108:887—8.
89. National Institute of Clinical Excellence http://www.nice.org.uk/.
90. Ng M, Saadat D, Sinha UK. Managing the emergency airway in Le Fort fractures. J Craniomaxillofac Trauma 1998;4(4):38—43.
91. Oakley P, Brohi K, Wilson A, et al. Guidelines for initial management and assessment of spinal injury. Working Party for the British Trauma Society Injury. Int J Care Injured 2003;405—25.
92. Oller DW, Meredith JW, Rutledge R, et al. The relationship between face or skull fractures and cervical spine and spinal cord injuries: a review of 13,834 patients. Accid Anal Prev 1992;24(2):187—92.
93. Özdal MPC. Mansour M, Deschens J. Ultrasound biomicroscopy evaluation of the traumatized eyes. Eye 2003;17:467—72.
94. Parver LM, Dannenberg AL, Blacklow B, et al. Characteristics and causes of penetrating eye injuries reported to the National Eye Trauma System Registry, 1985—1991. Public Health Rep 1993;108(5):625—32.
95. Pelletier CR, Jordan DR, Braga R, McDonald H. Assessment of ocular trauma associated with head and neck injuries. J Trauma Inj Infect Crit Care 1998;44:350—4.
96. Pfister R. Chemical injuries of the eye. Ophthalmology 1983;90:1246—53.
97. Pieramici DJ, Sternberg P, Aaberg TM, et al. A system for classifying mechanical injuries of the eye (globe). Am J Ophthalmol 1997;123:820—31.
98. Pieramici DJ, Eong KG, Sternberg Jr P, et al. The prognostic significance of a system for classifying mechanical injuries of the eye (globe) in open-globe injuries. J Tauma 2003;54(4):750—4.
99. Pritikin JB, Caldarelli DD, Panje WR. Endoscopic ligation of the internal maxillary artery for treatment of intractable posterior epistaxis. Ann Otol Rhinol Laryngol 1998;107:85—91.
100. Rowe W. Maxillofacial injuries. 2nd ed. Churchill Livingston; 1994.
101. Rockey JG, Anand R. A critical audit of the surgical management of intractable epistaxis using sphenopalatine artery ligation/diathermy. Rhinology 2002;40(3):147—9.
102. Roodhoof JMJ. Leading causes of blindness worldwide. Bull Soc Belg Ophtalmol 2002;283:19—25;
Sclafani AP, Salvatore JA. Angiography and transcatheter arterial embolization of vascular injuries of the face and neck. Laryngoscope 1996, February;106(2):168—73.
103. Royal College of Surgeons of England, Definitive Surgical Trauma Skills Course and Manual; 2002.
104. Rubsamen PE, Cousins SW, Winward KE, et al. Diagnostic ultrasound and pars plana vitrectomy in penetrating ocular trauma. Ophthalmology 1994;101:809—14.
105. Sanders R, Swiontkowski M, Nunley J, et al. The management of fracture with soft-tissue disruptions. J Bone Joint Surg 1993;75:778.
106. Saussez, Choufani, Brutus, et al. Lateral canthotomy; a simple and safe procedure for orbital haemorrhage secondary to endoscopic sinus surgery. Rhinology 1998;36:37—9.
107. Schade K, Borzotta A, Michaels A. Intracranial malposition of nasopharyngeal airway. J Trauma Inj Infect Crit Care 2000;49(5):967—8.
108. Schell RE, Mathern GW. Complication of nasogastric tube placement after a gunshot wound to the face. Oral Surg Oral Med Oral Pathol 1990;70:525—6.
109. Schrage NF, Langfeld S, Zschocke J, et al. Eye burns: an emergency and continuing problem. Burns 2000;26(8):689—99.
110. Shaw R, Mcnaughton G. Emergency airway management in a case of lingual haematoma. Emerg Med J 2001;18:408—9.
111. Shimoyama T, Kaneko T, Horie N. Initial management of massive oral bleeding after midfacial fracture. J Trauma: Inj Infect Crit Care February 2003;54(2):332—6.
112. Spoor TC. An atlas of ophthalmic trauma. Martin Duntz Publishing; 1997.
113. Srinivasan V, Sherman IW, O'Sullivan G. Surgical management of intractable epistaxis: audit of results. J Laryngol Otol 2000;114(9):697—700.
114. Sinclair D, Schwartz M, Gruss J, McLellan B. A retrospective review of the relationship between facial fractures, head injuries and cervical spine injuries. J Emerg Med 1988;6:109—12.
115. Slots P, Vegger PB, Bettger H, Reinstrup P. Retrograde intubation with a Mini-Trach II kit. Acta Anaesthesiol Scand 2003;47(3):274—7.
116. Sobaci G, Mutlu FM, Bayer A, et al. Deadly weapon-related open-globe injuries: outcome assessment by the ocular trauma classification system. Am J Ophthalmol 2000;129:47—53.
117. Sriussadaporn S, Sirichindakul B, Pak-Art R, Tharavej C. Pelvic fractures: experience in management of 170 cases at a university hospital in Thailand. J Med Assoc Thai 2002;85(2):200—6.
118. Steinsapir KD, Goldberg RA. Traumatic optic neuropathy. Surv Ophthalmol 1994;38:487—518.
119. Tay AB. Hazard: denture clasps embedded in oral tissues—case reports. Singapore Dent J 1997;22(1):18—21.
120. Tscherne H, Gotzen L. Fractures with soft tissue injuries. Berlin, Germany: Springer-Verlag, 1984. p. 5—9.
121. Tung T-C, Tseng W-S, Chen C-T, et al. Acute life threatening injuries in facial fracture patients: a review of 1,025 patients. J Trauma 2000;49(3):420—4.
122. Waldron J, Stafford N. Ligation of the external carotid artery for severe epistaxis. J Otolaryngol 1992;21(4):249—51.
123. Walsh FB. Pathological—clinical correlations. I: Indirect trauma to the optic nerves and chiasm. II: Certain cerebral involvements associated with defective blood supply. Investig Ophthalmol 1966;5:433—49.
124. Ward BP, Schendel SA, Hausamen JE. Maxillofacial surgery. Churchill Livingston; 1999.

125. Weksler N, Klein M, Weksler D, et al. Retrograde tracheal intubation: beyond fibreoptic endotracheal intubation. Acta Anaesthesiol Scand 2004;48(4):412—6.
126. Williams J, Jehle D, Cottington E, Shufflebarger C. Head, facial, and clavicular trauma as a predictor of cervical-spine injury. Ann Emerg Med 1992;21(6):719—22.
127. Winstead W. Sphenopalatine artery ligation: an alternative to internal maxillary artery ligation for intractable posterior epistaxis. Laryngoscope 1996;106:667—9.
128. Wright MJ, Greenberg DE, Hunt JP, et al. Surgical cricothyroidotomy in trauma patients. South Med J 2003;6(5):465—7.
129. Wolfgang H, Hausberger K, Sailer R, et al. Prevelance of cervical spine injuries in patients with facial trauma. Oral Surg Oral Med Oral Pathol Oral Rad Endod 2001;92: 370—6.
130. Zachariades N, Rallis G, Papademetriou G, et al. Embolization for the treatment of pseudoaneurysm and the transection of facial vessels. Oral Surg Oral Med Oral Radiol Endod 2001;92:491—4.
131. Zacharides N, Papavassiliou D, Christopoulos P. Blindness after facial trauma. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996;81:34—7.