Abstract: The aim of this review is to provide the reader with the most commonly accepted principles for the management of head trauma patients. The initial clinical evaluation and resuscitation, radiological evaluation, monitoring, intracranial pressure and cerebral perfusion pressure management, brain protection, associated organ dysfunctions and complications, anaesthetic management and the singularities of paediatric head trauma patients are described, either for the acute phase and the secondary phase of management.

Key words: Head trauma; management; review.

Optimal management of head trauma patients is challenging. Good knowledge of brain physiology and traumatic brain injury physiopathology is essential to successfully manage patients with head trauma. Success or failure depends on several factors including the initial severity of brain and associated lesions and their adequate clinical evaluation, efficient and non harmful early resuscitation, availability of a multidisciplinary neuro-trauma centre, and prevention and early detection of complications. In this paper, the currently accepted general principles governing the management of head trauma patients are reviewed, either for the acute phase and for the secondary phase of management. However, many of the frequently proposed therapies are not supported by class I evidence in the literature. Their benefit still need to be demonstrated by large randomized controlled trials (1). The initial evaluation, making decision regarding orientation towards a neuro-trauma centre, early resuscitation, radiological evaluation, monitoring modalities, intracranial pressure (ICP) and cerebral perfusion pressure (CPP) management, brain protection, non-neurological organ dysfunction of central origin, indications for surgery, anaesthetic management, and paediatric head trauma patient management will be described.

INITIAL CLINICAL EVALUATION AT THE ACUTE PHASE OF MANAGEMENT

When taking care of head trauma patients on the scene of the accident, the very first step consists of a rapid and efficient evaluation of the severity of brain aggression and associated lesions. This initial evaluation is of utmost importance for guiding patient orientation and future therapeutic decisions. The most widely used clinical neurological evaluation is the Glasgow Coma Score (GCS) associated to the determination of pupil size and reactivity to light (2). It is based on the best eye (1 = no eye opening, 2 = opening to pain, 3 = opening to verbal command, 4 = spontaneous opening), verbal (1 = no verbal response, 2 = incomprehensible sounds, 3 = inappropriate words, 4 = confused response, 5 = orientated response) and motor (1 = no motor response, 2 = extension to pain, 3 = flexion to pain, 4 = withdrawal from pain, 5 = orientated response to pain, 6 = obeys to command) responses of the patient to stimulation (3). Over a maximal score of 15, mild brain injury corresponds to a GCS ≥ 13, moderate to a GCS between 9 and 12 whereas severe brain injury is defined as a GCS ≤ 8. The initial GCS has a prognostic value and serves as a reference for subsequent evaluation. It must be evaluated after the initial correction of vital functions. The motor part of the score is the most pertinent. Several other neurological symptoms may also help in evaluating the patient. A brainstem lesion can be suspected in the absence of fronto-orbicular, oculo-cephalic, oculo-vestibular and oculo-cardiac reflexes (4). Caution is advised when looking for those reflexes. Moving the head can be
dangerous in case of associated cervical spine lesion. Pupil size and reactivity to light anomalies may not be easy to interpret, particularly when the patient is intoxicated with alcohol, cocaine, amphetamines or other psychotropic substances, or in case of orbital trauma.

Special attention should be paid to concomitant lesions to other parts of the body, namely the face, the cervical spine, the thorax, the abdomen, as well as the upper and lower limbs. Lesions to the face and mouth may render tracheal intubation difficult. Concomitant lesions of the cervical spine are very common in head trauma patients. Management of such lesions will not be detailed here and interested readers are referred to our previously published review paper (5). Lesions to the thorax and the abdomen can be life threatening by compromising ventilation (pneumo-hemothorax, costal fractures), and haemodynamic stability (heart failure caused by myocardial contusion, tamponade, aortic isthmus rupture, silent intra-abdominal bleeding). Trauma to the limbs and wounds to the scalp can also cause severe bleeding.

In each case, the initial evaluation of the head trauma patient must be fast and efficient in order to avoid delaying early resuscitation. Early resuscitation and adequate decision on the need for hospital admission is essential. Minimal criteria for non-delayed hospital admission include transient or persisting loss of consciousness, post-traumatic amnesia, persisting nausea and vomiting, or difficulty with assessment (alcohol ...) (6).

**EARLY RESUSCITATION AND MANAGEMENT ON THE SCENE OF TRAUMA**

The goal here is the restoration of vital functions. Early resuscitation always start with the so-called ABC’s. There is a consensus to say that all severe brain trauma patients (GCS ≤ 8) must be mechanically ventilated, as well as those with associated facial injuries (7). Management of the airway can reveal difficult, particularly in patients with associated cervical spine and facial injuries, and careful sedation is often required. A rapid sequence induction is mandatory and necessitates skilled care providers (8). Stability of the cervical spine must always be insured and cricoid pressure will be applied using a two-hand technique (5).

The choice of anaesthetic medication will depend on their potential side effects and on the experience of the practitioner. It must always be kept in mind that, outside the hospital and before ICP can be monitored, cerebral perfusion pressure must be maintained at all costs. It means that any comatose patient will be considered as a patient with raised ICP and, therefore, a need for maintaining adequate CPP.

Possible medications to induce anaesthesia include propofol, barbiturates, etomidate, ketamine or benzodiazepines. Propofol and barbiturates reduce the cerebral metabolic rate, and hence ICP. They also have neuroprotective properties (9). However, they may induce profound hypotension in hypovolaemic patients. Their use will therefore be limited to those patients whose volaemia is supposed to be adequate or after adequate fluid resuscitation. The same is true, although to a lesser extent, for benzodiazepines. Etomidate is a good first choice, as it has brain protective effects and does not induce hypotension. However, it can not be used for maintenance of anaesthesia due to its depressing effects on endogenous adrenal secretions. Ketamine is brain protective, has strong antinociceptive properties and helps maintaining haemodynamic stability. However, it increases the cerebral metabolic rate and increases ICP. Caution should therefore be paid in patients suspect of increased ICP. However, this effect is strongly attenuated by the concomitant use of propofol or barbiturates.

Analgesia should be insured using opioid derivatives. It is legitimate to use either alfentanil, sufentanil or fentanyl. However, one must keep in mind that intermittent neurological examination will be necessary during the next hours. Opioid derivatives poorly alter the motor response to stimulation, but may alter consciousness at high doses. Choosing a medication with unfavourable pharmacokinetic properties may impede those evaluations. It is also the reason why remifentanil may be preferred in some instances, although the haemodynamic consequences can be more marked when using this medication than when using other opioids (9).

Neuromuscular blocking agents are useful to facilitate tracheal intubation. Although it induces muscular fasciculation and increases ICP, succinylcholine may be of great help when intubation is expected to be difficult. The alternative is the use of a high dose of rocuronium (0.9 mg kg\(^{-1}\)). In that case, prolonged muscle relaxation may be dangerous if tracheal intubation is impossible. Furthermore, it may delay reliable neurological evaluation. The upcoming availability of the new steroid neuromuscular blocking agents antagonist, cyclodextrin, may soon overcome the problem of prolonged
However, studies are still needed to determine the effect of cyclodextrin-induced neuromuscular blockade antagonism on ICP.

Other agents may help reducing the sympathetic response to tracheal intubation, and hence the raise in ICP at that time. In that respect, lidocaine at the dose of 1.5 mg kg\(^{-1}\) has been shown to attenuate the cardiovascular, coughing and ICP responses to intubation (11).

Maintenance of sedation to allow for transportation to the trauma care centre can be performed using a continuous infusion of propofol or midazolam and sufentanil or remifentanil, once haemodynamic stability is achieved. Halogenated inhaled anaesthetic agents are not easy to use outside the operating theatre. Some of them have vasodilating properties on the cerebral vasculature and hence raise ICP. For those reasons, they are not frequently used for sedation of head trauma patients. Due to its effects on ICP and its potential neurotoxic effects, nitrous oxide should always be avoided (9). Intermittent muscle relaxation may be necessary, particularly if coughing on the tracheal tube cannot be alleviated by the intravenous anaesthetic regimen. Non-depolarising intermediate duration agents such as atracurium, cis-atracurium or rocuronium will be used. Continuous muscle relaxation is not recommended, as it impede neurological evaluation. Long term muscle relaxation may also have neuromuscular trophic consequences.

Beside clinical signs of brain herniation such as anisocoria and Cushing’s reaction (bradycardia and hypertension), no real signs of intracranial hypertension can be recognised in a ventilated and sedated patient. The clinician will therefore be mainly guided by the initial GCS. Moderate hyperventilation to a target end-tidal CO\(_2\) partial pressure between 30 and 35 mmHg will be the rule until brain imaging and/or direct measurement can rule out increased ICP. Indeed, prolonged hypocapnia has a limited duration of action on ICP and can be deleterious for the brain as it can favour brain ischaemia through excessive vasoconstriction (12). Mean blood pressure will be maintained at least above 70 mmHg, ideally 90 mmHg, through fluid administration and, perhaps, the use of vasopressors. At least isotonic fluids should be used, as hyponatremia can be very detrimental for the injured brain (13). Only dramatic situations of brain herniation will require the infusion of mannitol (0.25 to 1 g kg\(^{-1}\)), hypertonic saline, furosemide and/or barbiturates, while preserving perfusion pressure. Prone position of the head to 30° is beneficial to reduce ICP. Peripheral saturation in oxygen will be maintained in the normal range, at least above 95% and the stomach will be emptied using a gastric tube. Caution is necessary when inserting this probe if a skull base fracture is suspected (“sunglasses” haematoma). In that case, insertion through the mouth, under laryngoscope-directed vision is preferable.

**BRAIN LESION IMAGING STRATEGY**

Upon arrival in the trauma centre, it is necessary to assess the severity of lesions. Beside imaging of concomitant lesions to other parts of the body (cervical spine, thorax, abdomen, limbs), a brain CT scan must be performed as soon as possible when GCS is low, in case of neurological deficits, epileptic seizure, skull fracture and cerebrospinal fluid (CSF) leak (6). It will allow identification of intracranial lesions and may indicate the need for surgical intervention (extra-dural, sub-dural or intra-cerebral haematoma, brain contusion, acute hydrocephaly, depressed skull fracture, open fracture and penetrating lesions). Intracranial hypertension can be detected through visualisation of brain oedema, mass lesion, reduced cistern size and midline shift. The early brain CT scan has a strong prognostic value (2).

**MONITORING DURING THE ACUTE AND THE SECONDARY PHASE OF MANAGEMENT**

**Basic monitoring**

Basic monitoring including electrocardiogram, non-invasive blood pressure and peripheral oxygen saturation must be started immediately. End-tidal CO\(_2\) monitoring can be of great help to adjust ventilation in mechanically ventilated patients, allowing the indirect control of arterial CO\(_2\) concentration. Invasive arterial blood pressure monitoring will often be started as soon as possible, that is once the patient is admitted to the emergency care unit. This monitoring will allow faster detection of episodes of hypo- or hypertension, tight control of mean blood pressure (and hence of cerebral perfusion pressure), guiding fluid administration and the use of vasoactive medications, and performing blood gas analyses. Temperature and urine output monitoring are also recommended.

Advanced monitoring techniques can be used during the secondary phase of trauma patient care,
either to guide therapeutic measures or to help in evaluating prognosis.

ICP monitoring

Decision to monitor ICP depends in part on CT results (14). Generally speaking, ICP monitoring is required in patients with an initial GCS ≤ 8. However, it is also recommended in patients with a GCS ≥ 8 and severe brain oedema, reduced cistern size, severe temporal lobe contusion, midline shift and obliteration of the third ventricle. Conversely, patients with a GCS ≤ 8 but a normal CT will require ICP monitoring if they are more than 40 years of age, they display motor deficits or a systolic blood pressure lower than 90 mmHg (15). In many instances, neurosurgeons favour ICP measurement through an intra-ventricular catheter. This method is accurate, allows CSF drainage, and recalibrations. However, placement of those catheters necessitates the environment of an operating theatre and may be associated with infectious complications or lesions of brain parenchyma (16). It has been known for a long time that the number of high ICP episodes strongly determines the outcome of head trauma patients (17). Combined to mean blood pressure monitoring, it allows controlling the CPP. A detailed description of the goals to achieve regarding ICP and CPP management is provided hereafter. Other ICP monitoring devices are available such as the subarachnoid bolts and intraparenchymal fiberoptic devices. Subarachnoid bolts allow CSF drainage, but are less accurate. Fiberoptic devices do not allow CSF drainage and may also damage brain tissue.

Jugular venous oxygen saturation

Jugular venous oxygen saturation (SjO2) monitoring can be instituted in severe cases to help for therapeutic decisions. It necessitates the retrograde insertion of a catheter into the jugular bulb. Usually, this is not feasible in the emergency care unit and will only be performed once the patient is admitted to the intensive care unit. SjO2 reflects the global balance between O2 delivery to the brain and its metabolic needs. Normal SjO2 values range between 55 to 71% (18). As a sustained SjO2 value below 50% is associated to cerebral ischaemia, the practitioner will adapt CPP. O2 delivery (cardiac output, haemoglobin, arterial O2 partial pressure) and cerebral metabolic rate to maintain SjO2 into the normal range. This monitoring will also help detecting vasospasm and deleterious effects of hyperventilation. Noteworthy, excessive and prolonged hyperventilation can be harmful to localised brain regions, and, as SjO2 reflects the global situation of the brain, this deleterious effect will not be detected by SjO2 monitoring (19).

Cerebral blood flow monitoring

Global or regional cerebral blood flow (CBF) can be measured intermittently using several techniques such as the Kety-Schmidt method, xenon dilution (20), jugular thermodilution, single-photon emission scan, xenon-enhanced scan, perfusion scan, magnetic resonance angiography, and positron emission tomography. These techniques are not widely used in daily practice, essentially because they are too complex. A step further, the study of regional CBF distribution using positron emission tomography may have prognostic value in severe brain-injured patients such as vegetative or minimally conscious patients (21). Transcranial doppler (TCD) does not allow measuring CBF and is essentially helpful to appreciate vasospasm, the consequences of elevated ICP/low CPP and carotid dissection. Through detection of cerebral circulatory arrest, it is also useful for the diagnosis of brain death (18).

Electrophysiological monitoring

Continuous monitoring of the electroencephalogram (EEG) is necessary to evaluate barbiturate-induced coma in severely elevated ICP patients. In that case, barbiturate infusion is target-ed to obtain a flat or quasi-flat EEG. EEG monitoring also allows detecting non-convulsive seizure activity, which requires immediate therapeutic intervention. Other electrophysiological monitoring modalities can also be used. For example, the Bispectral Index (BIS) allows individual titration of sedation, detection of seizure activity and guidance of barbiturate coma (22, 23). It can predict the probability of return to consciousness once sedation has been withdrawn (24). Somatosensory-evoked potentials are good predictors of outcome (25).

Brain oxygenation and metabolism

Brain oxygenation and metabolism, either global or regional, can be assessed through several monitoring modalities. Near infrared spectroscopy has been proposed in that respect but seems to be less reliable than other techniques (26). The invasive cerebral tissue oxygen monitoring, although
more invasive, offers a promising alternative and would be able to detect ischemic or hyperaemic episodes (27-29). Cerebral microdialysis measures metabolic substrates (glucose, lactate, pyruvate, adenosin or xanthin), neurotransmitters (glutamate, aspartate, GABA), cellular death witnesses (potassium, glycerol), and exogenous substances (medications). So far, evidence has not granted this technique as a clinical tool (30, 31).

**Intracranial Pressure and Cerebral Perfusion Pressure**

CPP maintenance is a determinant factor of outcome in severely brain-injured patients (17). CPP is the difference between mean arterial blood pressure (MABP) and ICP. Normal values range between 70 and 85 mmHg. In the normal brain, the ischemic threshold of CCP is considered to be 50 mmHg. When pressure autoregulation of CBF is altered, this threshold may shift to higher or lower values. Intense debate has occurred on determining the ideal CPP level to be targeted (32), and it is not easy to define an optimal CPP threshold. Maintaining CPP at too high levels (> 70 mmHg) exposes to the risk of hyperaemia and raised ICP, while maintaining it at too low levels to the risk of ischemia. The optimal threshold will therefore be defined on an individual basis, using estimates of the adequacy of O2 delivery to the brain such as SjO2, near infrared spectroscopy, invasive brain tissue oxygenation monitoring or microdialysis.

CPP can be controlled through modifications of the two components of its equation, namely ICP and MABP. MABP is easily modified using fluid infusion and/or vasoactive medications (e.g. levoirenine, dobutamine). Caution is advised concerning the administered amount of fluids, as excess fluid may lead to pulmonary oedema or acute respiratory distress syndrome. Indeed, brain trauma patients are prone to develop such problems (33). According to the Monro-Kellie principle (any raise in the content of the rigid skull box is associated to a pressure increase), the reduction of ICP can be achieved through reducing brain size (osmotherapy), the surgical removal of a mass, reducing CSF volume (drainage), opening the rigid box (decompressive craniectomy), or opening the rigid box (decompressive craniectomy). The therapeutic gradation can be schematized as follows: moderate hyperventilation (PaCO2 at 35 mmHg first, 30 if not sufficient), CSF drainage, 0.25 to 0.5 g kg⁻¹ mannitol (up to 2 g kg⁻¹) (34) or hypertonic saline (35) (150 mg kg⁻¹) (intermittent boluses every 4 hours, monitor natremia, plasma osmolarity, and renal function), furosemide (20 mg boluses, maintain volaemia), EEG-guided barbiturate administration, decompressive craniectomy and hypothermia (2). Decompressive craniectomy seems to improve outcome in severe head trauma patients with raised ICP that is refractory to other treatments, although prospective studies are still needed to confirm the role of this therapeutic measure and its indications (36, 37). Pentobarbital-induced coma should be started using a loading dose of 5-10 mg kg⁻¹ administered over 30 minutes. Maintenance can be achieved using a continuous infusion at the rate of 1-3 mg kg⁻¹ h⁻¹, and titration should be performed to obtain EEG-burst suppression and serum pentobarbital levels between 3 and 4 mg % (22). The maintenance of haemodynamic stability is mandatory.

**Prevention of Secondary Brain Damage and Brain Protection**

The practitioner in charge of traumatic brain-injured patients must always keep in mind the need for preventing aggravation of the initial brain lesions. The prevention of secondary brain damage and brain protection occurs through two main guiding principles: the prevention of secondary brain damage of systemic origin (secondary brain aggression of systemic origin, SBASO), often referred to as passive neuroprotection, and the instauration of brain protecting therapies, or active neuroprotection. The prevention of SBASO relies on the maintenance of homeostasis. Any episode of hypoxia (ventilation problems, pulmonary oedema, ...), anaemia (concomitant haemorrhage), hypo- or excessive hypertension, hyperglycaemia (> 8.33 mmol l⁻¹), hypo- or hypernatremia and hyperthermia should be avoided or treated as fast as possible (38). Seizure activity is common and prophylaxis is recommended (phenytoin, valproate).

Direct brain protecting therapies are scarce, although numerous laboratory investigations have evidenced several possible therapeutic measures. Direct proofs of the efficiency of those measures for improving outcome in humans are not easy to obtain. Several anaesthetic agents such as barbiturates, propofol, halogenated compounds, xenon, ketamine, magnesium and lidocaine have theoretical protective effects on the injured brain through their action at various levels of the secondary neuronal damage cascade, including apoptosis for
some of them (38). However, the question of the best agent to use is not resolved. Beside the anaesthetic armamentarium, most of the promising drugs evidenced by laboratory and animal experimentation have revealed disappointing in clinical practice. Research is still in progress for the most promising ones such as magnesium, calcium channel blockers, NMDA antagonists, anti-inflammatory medications, anti-proteases, free radical scavengers, immunomodulators and neurotrophic factors. Other tools include preconditioning, hyperoxia and hypothermia. Preconditioning consists in exposing the brain to minor insults (ischaemia, hypoxia, hyperoxia ...) or to medications (sevoflurane, erythropoietin, ...) that induce an increased tolerance to further aggression. Although promising, clinical evidence is still too poor to recommend their use routinely. This is also true for eucaric hyperoxia (39). Finally, the National Acute Brain Injury Study on hypothermia (NABISH) (40) has not permitted to conclude that hypothermia is beneficial following a traumatic brain injury, although it is recommended not to rewarm TBI patients that are hypothermic on admission. Noteworthy, hypothermia remains beneficial following cardiac arrest (41) and may be interesting to control for intractable raised ICP. It is now commonly admitted that steroids have no place for brain protection in traumatic brain injury (2).

NEUROENDOCRINE AND NON-NEUROLOGICAL ORGAN DYSFUNCTIONS OF CENTRAL ORIGIN

Beside direct effect on cerebral function, brain trauma may induce several neuroendocrine and non-neurological organ dysfunctions. Among them, inappropriate anti-diuretic hormone secretion syndrome (IADHS), diabetes insipidus, hypopituitarism, neurogenic pulmonary oedema, and the dysautonomic storm syndrome are the most common.

Inappropriate antidiuretic hormone secretion syndrome

IADHS is mainly characterised by the occurrence of hyponatremia that cannot be attributed to another cause (hypotonic fluid infusions, renal salt wasting syndrome, excessive osmotic or diuretic-enhanced diuresis) and may be treated using fluid intake restriction and, eventually, arginin vasoressin-receptor antagonists (42). Hypertonic saline infusion to correct hyponatremia may be dangerous, particularly in more than 48 hours hyponatremic patients, as it can induce central demyelination.

Diabetes insipidus

Diabetes insipidus is suspected in patients presenting high volume hypotonic diuresis (urine density < 1010) that do not respond to fluid restriction. Treatment consists of desmopressin subcutaneous administration (2-4 µg every 12 hours).

Hypopituitarism

The incidence of various degrees of hypopituitarism following TBI may be as high as 50%, and this pathology may concern any of the hypothalamo-hypophyso-peripheral hormonal axis (43). Systematic screening of pituitary function is recommended for all patients with moderate to severe TBI (44). Treatment consists of hormonal replacement.

Neurogenic pulmonary oedema and dysautonomic syndrome

The origin of the neurogenic pulmonary oedema is not known with precision and may be due to excessive adrenergic activity and/or cardiac failure, and would therefore be a consequence of the dysautonomic storm syndrome (45). This syndrome is characterised by several neurological symptoms, hypertension, hyperthermia, and tachycardia. These elements may have cardiac and neurological consequences (myocardial infarct, cardiac failure, raised ICP). The treatment is symptomatic and may require pulmonary artery catheterism, as well as echocardiographic monitoring.

ADDITIONAL CONCERNS OF THE SECONDARY PHASE OF MANAGEMENT

Among the other concerns related to the management of head trauma patients, sepsis, hypercatabolism, stress ulcer, and thromboembolic events are the most common. TBI patients are immunocompromised and sepsis is frequent, particularly of respiratory origin (46). Increased caloric and protein requirements of head trauma patients should be met without delay, using the following formula : RME = 152 – 14(GCS score) + 0.4(HR) + 7(DSI), where RME is the resting metabolic expenditure, GCS is the Glasgow coma scale, HR is the heart rate and DSI is the number of days since the injury (47). A stress ulcer prophylaxis should be immediately started using either type II histaminic
receptors antagonists or hydrogen ion pump inhibitors. Antithrombotic prophylaxis should also be started using stocking or intermittent calf compression, as well as low molecular weight heparins. Caution should be paid to the risk of intracranial bleeding in case of haemorrhagic traumatic lesions (48, 49).

ANAESTHETIC MANAGEMENT WHEN SURGERY IS REQUIRED

Anaesthetic management, either for intracranial or peripheral surgery, is again governed by the prevention of SBASO and the protection of the brain as much as possible (34). Surgical priorities will be determined on a case by case basis, the control of menacing bleeding often being the first concern. Large venous access is mandatory in those conditions. A central venous catheter may be of help to guide fluid administration but is not mandatory, and must not delay surgery. Beside classical anaesthetic monitoring, invasive arterial blood pressure monitoring will often be necessary. Bladder catheterisation and temperature monitoring will be instituted. The management of ICP and CPP during surgery will be easier if ICP can be directly monitored, but the placement of the ICP monitoring device is not always possible before or at the beginning of surgery. Throughout the procedure, normoxia, normothermia, and normoglycaemia will be maintained, and end-tidal PCO₂ will be in the range of 30-35 mmHg. Haemoglobin concentration and/or haematocrit will be checked at regular intervals. If an ICP monitoring device is available, the management of arterial blood pressure will target a CPP of 70 mmHg through adjustments of MABP (fluids, levoirenine) or ICP (drainage, mannitol, lasix, hypertonic saline). Otherwise, MABP should be maintained at 90 mmHg as much as possible if raised ICP is suspected. If the patient is not already intubated and sedated upon arrival in the operating theatre, a rapid sequence induction is the rule, using the same technique as the one described above. The same rules also apply for the choice of anaesthetic agents used to maintain anaesthesia. Caution should be paid to the adequate positioning of the head, alleviating jugular compression and favouring a 30° prone position.

PAEDIATRIC BRAIN TRAUMA

Specific concerns relate to the management of paediatric head trauma patients. The volume of the head compared to the volume of the body is proportionally more important in children than in adults. The physical mechanism responsible for brain lesions in children is therefore often related to high energy deceleration. Diffuse axonal lesions and generalised oedema are more common than intracerebral haematoma and contusion, with the consequence that children will present more frequently with an altered state of consciousness and seizures rather than focal deficits (50). It is worth to note that this decelerative mechanism of injury will often be responsible for cervical spinal cord injuries without radiological abnormalities (SCIWORA). The skull of children is also immature and is more prone to fractures than the adult. It is also more compliant when sutures and fontanels are not closed. The neurosurgical emergency to be fear of in children is the extradural haematoma, which necessitates immediate surgical drainage. It is frequently associated to parietal skull fracture, but not in all cases. It must be suspected in children whose neurological status deteriorates rapidly : an aggravating cephalalgia, nausea and vomiting and progressive stupor must warn the clinician. Hemiparesia and anisocoria are signs of temporal herniation, which jeopardizes the immediate vital prognosis. Those extradural haematomas may also manifest in the form of a hypovolaemic shock in very young children. Traumatic subdural haematomas are frequent in 5 month children (shaken babies), in which the subdural spaces are large and the brain more mobile. The management principles of head trauma in children are the same as those described for the adult (51), except that the target CPP to be maintained is lower and ranges between 40 and 50 mmHg instead of 70 (50).

CONCLUSIONS

The management of head trauma patients is challenging and requires tight collaboration between emergency, intensive care, radiology, anaesthesiology and neurosurgery practitioners. Careful initial evaluation, efficient early resuscitation, targeted imaging, rapid surgical interventions, and purposeful advanced life support are determinant factors of the outcome.

References