28 Traumatic brain injury

CLASSIFICATION OF HEAD INJURY

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Concussion is defined as the alteration of consciousness as a result of closed head injury but is generally used to describe consciousness at the time of injury is not a prerequisite. Key features include confusion and amnesia. The patient may be lethargic, easily distractable, forgetful, slo w to interact or emotionally labile. Gait disturbance and incoordination may be seen. While symptomatic following a head injury , patients may exhibit disordered cerebral autoregulation, making them especially vulnerable to repeat impacts. Second impact syndrome following an apparently trivial repeat injury comprises malignant brain swelling that can quickly progress to coma and death. Although the existence of the syndrome is disputed, and it is certainly rare, it should be considered in advice to individuals engaged in sports or activities carrying a risk of further injury: symptomatic players should not return to play . Postconcussive syndrome is a loosely defined constellation of symptoms persisting for a prolonged period after injury . Patients may report somatic features such as headac he, dizzi - ness and disorders of hearing and vision. They may also suffer - a variety of neurocognitive and neuropsychological distur - bances, including difficulty with concentration and recall, insomnia, emotional lability , fatigue, depression and personal - ity c hange. Some patients may exaggerate symptoms, seeking y of secondary gain (compensation). - Summary box 28.2 Minor and mild head injury /uni25CF /uni25CF /uni25CF

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Examination primary survey

Examination: primary survey

ATLS guidelines address a fundamental priority: ensuring uninterrupted perfusion of the brain with oxygenated blood. This is especially important after a head injury , given the disturbance to intracranial autoregulation and the sensitivity of the primary injured brain tissue to further insult. Bleeding from scalp lacerations may require management as part of the primary survey as the blood loss can be substantial and ongoing. Check the responsiveness of the pupils and conscious level and check for any gross focal neurological deficits. The blood glucose level should also be measured as early as possible as hypoglycaemia is very dangerous and easily reversible. Pupils The pupil size should be recorded in millimetres and the reactiv ity documented as present, sluggish or absent. Uncal herniation Sir Gordon Morgon Holmes , 1876–1965, physician, National Hospital for Nervous Diseases, London, UK. William John Adie , 1886–1935, physician, National Hospital for Nervous Diseases, London, UK. parasympathetic supply to the pupil. Unopposed sympathetic activity produces a sluggish enlarged pupil, progressing to fixed and dilated under continued compression. Established pupil changes ma y reflect pathology anywhere in the eye or the reflex loop made up by the optic nerve, the oculomotor nerve and the brainstem. Direct ocular trauma or nerve injury in association with a skull base fracture can cause mydriasis (dilated pupil) to be present from the time of injury . Pre-existing discrepancy in the pupil size (anisocoria), as a result of Holmes–Adie pupil or cataracts for example, may also complicate assessment. - Glasgow Coma Scale score The GCS score is the sum of scores on three components, as detailed in Table 28.4 . The breakdown of the GCS score into eye opening, verbal and motor components should always be recorded and used when communicating the status to other doctors. Remember that the score represents the best perfor - mance elicited, so a patient flexing in response to a painful stimulus on the left and localising on the right scores ‘M5’. A /uni00A0 sternal or supraorbital rub or trapezius squeeze represents an appropriate painful stimulus. Neurological deficit Gross focal neurological deficits, such as paraplegia, may be evident at the primary survey , and an assessment to exclude such a deficit should be carried out, especially if the patient is to be intubated so that subsequent examination will be impossible. Detailed neurological examination is included in the secondary survey . -

TABLE 28.4 Glasgow Coma Scale score for head injury. Eyes open Spontaneously 4 To verbal command 3 To painful stimulus 2 Do not open 1 Verbal Normal 5 Confused 4 Inappropriate/words only 3 Sounds only 2 No sounds 1 Intubated patient T Motor Obeys commands 6 Localises to pain 5 Withdrawal/ flexion 4 Abnormal flexion 3 Extension 2 No motor response 1

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A full secondary survey will be required. Particular attention must be paid to the head, neck and spine. Head Examination of the head should include inspection and palpa tion of the scalp for evidence of subgaleal haematoma and scalp lacerations, which may bleed profusely and potentially overlie fractures. Examine the face f or evidence of fractures, especially to the orbital rim, zygoma and maxilla. Clinical evidence of a skull base fracture may include Battle’s sign (Figure 28.3) and ‘racoon’ or ‘panda’ eyes (bilateral periorbital bruising). Haemotympanum, or overt bleeding from the ear if the tympanic membrane has ruptured, and CSF rhinorrhoea or otorrhoea are also highly suggestive of a fracture of the base of the skull. A complete examination of the cranial nerves will reveal, for example, facial or vestibulocochlear nerve damage asso ciated with skull base fracture. Midbrain or brainstem dys function may produce gaze par esis (inability of the eye to look across beyond the midline), dysconjugate gaze (inability of the William Henry Battle , 1855–1936, surgeon, St Thomas’s Hospital, London, UK. conjunctiva and cornea of the eyes, and the retina using an ophthalmoscope, looking for hyphaema (blood in the anterior chamber of the eye), papilloedema or retinal detachment. Blood in the mouth may be due to tongue-biting at seizure. The GCS score and pupil status, assessed as part of the pri - mary survey , require re-evaluation at the secondary survey and regularly thereafter. Neck and spine Studies have demonstrated an incidence of cervical fracture of up to 10% in association with moderate and severe TBI. Cervical spine injury must be presumed in the context of head injury until actively excluded. In a high-energy mechanism such as a road tra ffi c accident or fall from a height, thoracic and lumbar spine injuries must also be excluded. Plain radio - graphs are of limited value in excluding significant cervical spine injury . Even CT imaging does not exclude the possibility - of significant ligamentous injury . Therefore, whenever feasible, these patients should be managed in a hard collar until the neck can be cleared clinically . A peripheral nerve examination with documentation of limb tone, power, reflexes and sensation needs to be performed early to identify spinal pathology . This is especially important in patients who may subsequently be intubated and ventilated when this assessment will no longer be possible. Obtunded patients should move all four limbs in response to an appropri - ate painful stimulus. The patient will need to be log-rolled to palpate for thoracic or lumbar deformity , and any cervical collar should be removed - at this stage to allow palpation of the cervical spine before it - is then replaced again. If there is associated spinal injury , a thoracic sensory level is much more easily established by sen - sory examination on the back. A per rectum examination is also performed at log-roll, assessing for anal tone, sensation in the awake patient and anal wink (sphincter seen to contract in response to a pinprick stimulus). Priapism is a strong predictor of sever e cord injury even in intubated patients. Summary box 28.5 Secondary survey /uni25CF /uni25CF /uni25CF

Figure 28.3 Battle’s sign. A skull base fracture may be associated with bruising over the mastoid process. Battle’s sign, periorbital bruising and blood in the ears/nose/ mouth may point to a base of skull fracture Cervical spine fractures are common and must be actively excluded Log-roll to check the whole spine for steps and tenderness and for a per rectum examination

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History

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Mechanism In moderate and severe TBI, a history must be obtained from witnesses and paramedics. High-energy mechanisms of injury , including a fall from a height or a high-speed road accident, will require careful clinical and radiological exclusion of associated multisystem and spinal injury (see Chapters 27 and 30). In the case of road traffic accidents in particular, extraction time and evidence of hypoxia or haemodynamic instability at the scene is important information to obtain from the paramedics. Falls such as myocardial infarction, hypoglycaemia or subarachnoid haemorrhage, with crucial implications for management. Neurological progression A specific check should be made for any loss of consciousness at the time of injury and its duration. The GCS score and pupil responses should be recorded at the scene, during transfer, at admission and regularly thereafter. A deterioration in the GCS score is an important index of developing a potentially reversible secondary injury . It is also useful to assess the extent of amnesia, retrograde (events prior to the injury) and antero grade (events afterwards). If the patient was intubated at the scene of the accident, it is valuable to know whether the patient was moving all four limbs before this. Past medical history Obtain details of the patient’s medical background, including allergies and normal medications. Of particular note here are antiplatelet agents, potentially requiring platelet transfusion especially if surgery is required, and anticoagulants, which may need reversal. Summary box 28.3 History /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

Bystanders and paramedics may give vital information on the: Preinjury state (fits, alcohol, chest pain) Mechanism and energy involved in the injury (speed of vehicles, height fallen) Conscious state and haemodynamic stability of the patient after the accident Length of time taken for extrication Check the medication history, especially anticoagulants and antiplatelet agents

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INTRACRANIAL PRESSURE Intracranial pressure and ce

INTRACRANIAL PRESSURE Intracranial pressure and cerebral blood flow

The brain depends on continuous perfusion for oxygen and glucose delivery , and hence survival. Normal cerebral blood flow (CBF) is about 55 /uni00A0 mL/min for every 100 /uni00A0 g of brain tissue. Ischaemia results when this rate drops below 20 /uni00A0 mL/min, and even lower levels will result in infarction unless promptly corrected. † The learning objectives of this chapter are aligned with the Intercollegiate Surgical Curriculum Programme (ISCP) ST3 Neurosurgery Knowledge Requirements in Cranial Trauma and comprise: • the resuscitation, assessment, investigation and continuing care of head-injured patients • the prevention and detection of secondary intracranial and systemic insults. Alexander Monr o (secundus) , 1733–1817, Scottish anatomist, physician and medical educator. George Kellie , 1770–1829, Scottish surgeon and pupil of Alexander Monro (secundus). Flow depends on cerebral perfusion pressure (CPP), which is the di ff erence between the mean arterial pressure (MAP) and the intracranial pressure (ICP): CPP (75–105 /uni00A0 mmHg) = MAP (90–110 /uni00A0 mmHg) – ICP (5–15 /uni00A0 mmHg) Typical normal values are given in parentheses. In fact, in - the normal brain, variations in vascular tone maintain a con - stant CBF across a range of MAP between 50 and 150 /uni00A0 mmHg (or higher in the setting of chronic hypertension) and a cor - responding range of CPP – the process of cerebral auto- regulation . Autoregulation can be impaired in the context of trauma, so that MAP and ICP must be actively regulated in these patients to maintain proper perfusion. The Monro–Kellie doctrine and herniation syndromes Alexander Monro observed in 1783 that the cranium is a ‘rigid box’ containing a ‘nearly incompressible brain’. Any expansion in the contents, especially haematoma and brain swelling, may be initially accommodated by exclusion of fluid components, venous blood and cerebrospinal fluid (CSF). Further expansion is associated with an exponential rise in ICP (Figure 28.1). Uncontrolled increases in ICP result in cerebral herniation (Figure 28.2). Typically , herniation of the uncus of the tem - poral lobe over the tentorium results in pupil abnormalities (see Pupils), usually occurring first on the side of any expand - ing haematoma. Cerebellar tonsillar herniation through the foramen magnum compresses medullary vasomotor and

The resuscitation, assessment, investigation and • continuing care of head-injured patients The prevention and detection of secondary intracranial • and systemic insults

(b) respiratory centres, classically producing Cushing’s triad hypertension, bradycardia and irregular respiration. The patient is then said to be ‘ coning ’, and brainstem death will result without immediate intervention. Summary box 28.1 Intracranial pressure /uni25CF /uni25CF Harvey Williams Cushing , 1869–1939, Professor of Surgery , Harvard University Medical School, Boston, MA, USA, considered the founding father of modern neurosurgery .

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torium cerebelli, where it can compress the third nerve. (4) Central herniation and (5) tonsillar herniation result in brainstem compromise, manifesting as Cushing’s triad.

INTRACRANIAL PRESSURE Intracranial pressure and cerebral blood flow

The brain depends on continuous perfusion for oxygen and glucose delivery , and hence survival. Normal cerebral blood flow (CBF) is about 55 /uni00A0 mL/min for every 100 /uni00A0 g of brain tissue. Ischaemia results when this rate drops below 20 /uni00A0 mL/min, and even lower levels will result in infarction unless promptly corrected. † The learning objectives of this chapter are aligned with the Intercollegiate Surgical Curriculum Programme (ISCP) ST3 Neurosurgery Knowledge Requirements in Cranial Trauma and comprise: • the resuscitation, assessment, investigation and continuing care of head-injured patients • the prevention and detection of secondary intracranial and systemic insults. Alexander Monr o (secundus) , 1733–1817, Scottish anatomist, physician and medical educator. George Kellie , 1770–1829, Scottish surgeon and pupil of Alexander Monro (secundus). Flow depends on cerebral perfusion pressure (CPP), which is the difference between the mean arterial pressure (MAP) and the intracranial pressure (ICP): CPP (75–105 /uni00A0 mmHg) = MAP (90–110 /uni00A0 mmHg) – ICP (5–15 /uni00A0 mmHg) Typical normal values are given in parentheses. In fact, in - the normal brain, variations in vascular tone maintain a con - stant CBF across a range of MAP between 50 and 150 /uni00A0 mmHg (or higher in the setting of chronic hypertension) and a cor - responding range of CPP – the process of cerebral auto- regulation . Autoregulation can be impaired in the context of trauma, so that MAP and ICP must be actively regulated in these patients to maintain proper perfusion. The Monro–Kellie doctrine and herniation syndromes Alexander Monro observed in 1783 that the cranium is a ‘rigid box’ containing a ‘nearly incompressible brain’. Any expansion in the contents, especially haematoma and brain swelling, may be initially accommodated by exclusion of fluid components, venous blood and cerebrospinal fluid (CSF). Further expansion is associated with an exponential rise in ICP (Figure 28.1). Uncontrolled increases in ICP result in cerebral herniation (Figure 28.2). Typically , herniation of the uncus of the tem - poral lobe over the tentorium results in pupil abnormalities (see Pupils), usually occurring first on the side of any expand - ing haematoma. Cerebellar tonsillar herniation through the foramen magnum compresses medullary vasomotor and

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Introduction

†

INTRODUCTION

Head injury accounts for 3–4% of emergency department attendances, with around 1500 cases per 100 /uni00A0 000 population per year in the UK. Annual mortality attributable to head injury is estimated at 9 per 100 /uni00A0 000, and it remains the leading cause of death and disability from childhood to early middle age, with an estimated 2% of the US population suffering long term disability as a result of head injury . Road traffic accidents are the leading cause of head injury , responsible for up to 50% of cases. Other common mechanisms of injury include falls and assault. There is significant geographical variation, for example firearms are the third leading cause in the USA. Traumatic brain injury (TBI) can be considered as the combination of primary injury sustained on impact, and hence not medically modifiable, and secondary injury developing in the following hours and days. Understanding the importance of intracranial pressure (ICP) and related parameters is key to minimising secondary injury and improving outcomes.

Learning objectives

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To be familiar with: The physiology of cerebral blood /f_l ow and the • pathophysiology of raised intracranial pressure Learning objectives

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MINOR AND MILD HEAD INJURY

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After exclusion of associated cervical spine injury , it is import - ant to consider the possibility of a ‘lucid interval’ that may precede delayed deterioration due to an expanding intracranial haematoma. In general, patients with isolated head injuries and without ongoing deficits can be safely discharged fr om the emergency department, provided they meet suitable criteria, Health and Care Excellence (NICE) (Table 28.2). /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Patients who do not meet all the discharge criteria will need admission for a further period of observation and/or brain imaging. Early computed tomography (CT) imaging is desir able in patients with a persistent reduced conscious level, focal deficits, suspected fractures or risk factors for intracranial bleed (Table 28.3). Significant clinical or radiological abnormalities should be discussed with the neurosurgical service. Man these patients will struggle with features of concussion for a period after their injury , with headaches and somnolence typ ical. Follow-up by a head injury specialist nurse or equivalent is therefore desirable. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

TABLE 28.2 UK National Institute for Health and Care Excellence discharge criteria in minor and mild head injury. GCS 15/15 with no focal de /f_i cits Normal CT brain if indicated (see Table 28.3) Patient not under the in /f_l uence of alcohol or drugs Patient accompanied by a responsible adult Verbal and written head injury advice: seek medical attention if: Persistent/worsening headache despite analgesia Persistent vomiting Drowsiness Visual disturbance Limb weakness or numbness CT, computed tomography; GCS, Glasgow Coma Scale score. TABLE 28.3 UK National Institute for Health and Care Excellence (NICE) guidelines for computed tomography (CT) in head injury. Indications for CT imaging in head injury within 1 hour GCS <13 at any point GCS <15 at 2 hours Focal neurological de /f_i cit Suspected open, depressed or basal skull fracture More than one episode of vomiting Post-traumatic seizure Indications for CT imaging within 8 hours Age >65 Coagulopathy (e.g. aspirin, warfarin or rivaroxaban use) Dangerous mechanism of injury (e.g. fall from a height, RTA) Retrograde amnesia >30 minutes GCS, Glasgow Coma Scale score; RTA, road traf /f_i c accident.

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Resuscitation is performed according to Advanced Trauma Life Support (ATLS) guidelines, beginning with management of the airway with cervical spine control and proceeding to assess and manage breathing and circulation. The history obtained in parallel is key to shaping ongoing management. MODERATE AND SEVERE TRAUMATIC BRAIN INJURY Resuscitation and evaluation

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Medical management

Medical management

From initial resuscitation, through surgical intervention and into the subsequent phase of ICU management, medical management strategies aim to minimise secondary brain injury through avoidance of hypoxia and hypotension and control of ICP . Unchecked, secondary injury leads to a further cycle of deterioration (Figures 28.2 and 28.9). - Early discussion of patients and imaging with the regional neurosurgical service is advisable. UK trauma audit and research network data show higher mortality in patients with severe TBI managed in non-neurosurgical centres, and this is reflected in NICE guidelines, which recommend early transfer irrespective of the need for surgery . Control of intracranial pressure Intubation and ventilation are required early in the manage - ment of severe brain injury for airway control. They are often required in moderate brain injury to facilitate the safe management and transfer of unstable and frequently agitated patients and in order to control ICP . A bolus of mannitol or hypertonic saline may be administered to temporise ICP , for - example while scanning and transferring the patient. Management of the intubated patient, following evac - uation of any focal haematomas, is guided by ICP monitoring using a bolt ICP monitor or else an external ventricular drain inserted into the lateral ventricle, which can also contribute to ICP control by permitting CSF drainage. A sustained ele - vated ICP ex ceeding 20–25 /uni00A0 mmHg is associated with a poor outcome, and maintenance of a CPP of at least 60 /uni00A0 mmHg is important in preventing secondary injury . ICP can be controlled by simple measures, including raising the head of the bed and loosening the collar to impro ve venous drainage. Seizures and pyrexia should be actively controlled. Medical management titrated to ICP includes escalating doses of sedatives , analgesics and ultimately muscle relaxants. Target ventilatory and circulatory parameters are set out in Table 28.5 . /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Where these measures fail, neurointensivists may seek to control brain swelling using mannitol or hypertonic saline infu - sions. Where autoregulation is preserved, inducing high CPP may reduce ICP through vasoconstriction. A range of further interventions are e ff ective in controlling ICP , but evidence for long-term outcome benefit is limited or absent. These inter - v entions include induction of therapeutic hypothermia or thiopentone coma and surgical decompressive craniectomy . Pituitary dysfunction Electrolyte imbalance is common in TBI and contributes to brain swelling and to causing seizures. Diverse mechanisms

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of excretory dysregulation in association with brain insult, leads to volume depletion and hyponatraemia. The syndrome of inappropriate antidiuretic hormone (SIADH) leads to water retention and hyponatraemia in the context of pituitary damage. This is of particular concern in head injury since low serum osmotic pressure can contribute to brain swelling, so hypotonic fluids are avoided in this setting. Conversely antidiuretic hormone secretion may be compromised in the context of trauma, producing diabetes insipidus, resulting in hypernatraemia. All aspects of pituitary function may be compromised in the setting of TBI. Routine screening of pituitary hormone levels and liaison with endocrinology are important aspects of optimal medical management. Note that routine, rather than directed, administra tion of corticosteroids in severe head injury is associated with increased mortality and is not recom mended. Seizures Seizures may occur early (within 7 days) or late. The cumu lative probability is between 2% (mild TBI) and 60% (severe TBI with exacerbating features). Risk factors include injury severity , especially the presence of intracerebral haemorrhage, depressed skull fractures and tears of the dura. Antiepileptics, typically phenytoin, are administered prophylactically to patients at high risk of seizures. Nutrition Enteral nutrition is preferred to intravenous parenteral nutri tion on the grounds of cost and associated complications, and should be commenced within 72 hours of injury . Prokinetics (e.g. metoclopramide, erythromycin) can be administered to promote absorption. Outcomes and sequelae The long-term sequelae of moderate and severe TBI include headache and memory and cognitive impairments, contributing to the postconcussive syndrome described above. Rehabilitation represents a complex and prolonged multidisciplinary challenge. The Glasgow Outcome Scale score is used to quantify the degree of recovery achieved after head injury , especially for research purposes, and is detailed in Table 28.6 . Good recovery implies independence and potential to return to work rather than a full return to previous capacity . Medical management of head injury /uni25CF /uni25CF /uni25CF /uni25CF

TABLE 28.6 Glasgow Outcome Scale. Good recovery 5 Moderate disability 4 Severe disability 3 Persistent vegetative state 2 Dead 1 First line ICP control involves optimising sedation, ventilation and serum sodium levels Paralysis and external ventricular CSF drainage are important adjuncts There is little evidence for benefit with therapeutic hypothermia, barbiturate coma or decompressive craniectomy Check pituitary function, consider seizure prophylaxis, commence enteral nutrition within 72 hours

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of excretory dysregulation in association with brain insult, leads to volume depletion and hyponatraemia. The syndrome of inappropriate antidiuretic hormone (SIADH) leads to water retention and hyponatraemia in the context of pituitary damage. This is of particular concern in head injury since low serum osmotic pressure can contribute to brain swelling, so hypotonic fluids are avoided in this setting. Conversely antidiuretic hormone secretion may be compromised in the context of trauma, producing diabetes insipidus, resulting in hypernatraemia. All aspects of pituitary function may be compromised in the setting of TBI. Routine screening of pituitary hormone levels and liaison with endocrinology are important aspects of optimal medical management. Note that routine, rather than directed, administra tion of corticosteroids in severe head injury is associated with increased mortality and is not recom mended. Seizures Seizures may occur early (within 7 days) or late. The cumu lative probability is between 2% (mild TBI) and 60%

(severe TBI with exacerbating features). Risk factors include injury severity , especially the presence of intracerebral haemorrhage, depressed skull fractures and tears of the dura. Antiepileptics, typically phenytoin, are administered prophylactically to patients at high risk of seizures. Nutrition Enteral nutrition is preferred to intravenous parenteral nutri tion on the grounds of cost and associated complications, and should be commenced within 72 hours of injury . Prokinetics (e.g. metoclopramide, erythromycin) can be administered to promote absorption. Outcomes and sequelae The long-term sequelae of moderate and severe TBI include headache and memory and cognitive impairments, contributing to the postconcussive syndrome described above. Rehabilitation represents a complex and prolonged multidisciplinary challenge. The Glasgow Outcome Scale score is used to quantify the degree of recovery achieved after head injury , especially for research purposes, and is detailed in Table 28.6 . Good recovery implies independence and potential to return to work rather than a full return to previous capacity . Medical management of head injury /uni25CF /uni25CF /uni25CF /uni25CF

TABLE 28.6 Glasgow Outcome Scale. Good recovery 5 Moderate disability 4 Severe disability 3 Persistent vegetative state 2 Dead 1 First line ICP control involves optimising sedation, ventilation and serum sodium levels Paralysis and external ventricular CSF drainage are important adjuncts There is little evidence for benefit with therapeutic hypothermia, barbiturate coma or decompressive craniectomy Check pituitary function, consider seizure prophylaxis, commence enteral nutrition within 72 hours

Non-accidental injury

Non-accidental injury

Head injury in children and vulnerable adults may be due to abuse. Significant findings include delayed presentation, injuries of disparate age, retinal haemorrhages, bilateral chronic subdural haematomas, multiple skull fractures and neurological injury without external signs of trauma. Non-accidental injury

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TRAUMATIC BRAIN INJURY IN THE CHILD

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- Head injury in children is common and presents specific chal - lenges relating to physiology , assessment, management and safeguarding. Children have large heads compared with the rest of their bodies, predisposing to both head and neck injury . - In the case of minor head injur y , good assessment depends on winning the trust of child and parent, while identifying risk factors requiring further admission for observation or CT scan (Table 28.7). Non-accidental injury should always be considered; for example, it is key to ensure that the reported mechanism of injury is in keeping with the child's develop - mental stage and to examine for injuries outside the normal distribution for childhood accidents. The Paediatric Glasgow Coma Scale is applied in the under-twos (Table 28.8). - Moderate and severe head injury should be managed b y a trauma team in a resuscitation room, using paediatric ATLS protocols directed at optimising physiology to prevent secondary brain injury , and with intensive car e unit (ICU) involvement for airway management as appropriate. Children with open sutures can lose substantial blood volumes into the head. Palpating the fontanelle allows direct assessment of ICP , and in all cases head and neck CT imaging are key to guiding definitive management. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

TABLE 28.7 UK National Institute for Health and Care Excellence criteria for computed tomography scan in children following head injury. Suspicion of NAI First seizure GCS <14 or <15 in under-ones GCS <15 2 hours post injury Signs of fracture of the base of skull Focal neurological de /f_i cit Bruise/swelling/laceration >5 /uni00A0 cm in under-ones More than one of: Loss of consciousness >5 minutes Abnormal drowsiness Four or more episodes of vomiting Dangerous mechanism Amnesia >5 minutes GCS, Glasgow Coma Scale score; NAI, non-accidental injury.

Greenberg MS. Handbook of neurosurgery , 9th edn. Stuttgart: Thieme Medical Publishers, 2019. Samandouras G (ed.). The neurosurgeon's handbook. Oxford: Oxford University Press, 2010.

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TRAUMATIC BRAIN INJURY IN THE CHILD

- Head injury in children is common and presents specific chal - lenges relating to physiology , assessment, management and safeguarding. Children have large heads compared with the rest of their bodies, predisposing to both head and neck injury . - In the case of minor head injur y , good assessment depends on winning the trust of child and parent, while identifying risk factors requiring further admission for observation or CT scan (Table 28.7). Non-accidental injury should always be considered; for example, it is key to ensure that the reported mechanism of injury is in keeping with the child’s develop - mental stage and to examine for injuries outside the normal distribution for childhood accidents. The Paediatric Glasgow Coma Scale is applied in the under-twos (Table 28.8). - Moderate and severe head injury should be managed b y a trauma team in a resuscitation room, using paediatric ATLS protocols directed at optimising physiology to prevent secondary brain injury , and with intensive car e unit (ICU) involvement for airway management as appropriate. Children with open sutures can lose substantial blood volumes into the head. Palpating the fontanelle allows direct assessment of ICP , and in all cases head and neck CT imaging are key to guiding definitive management. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

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