

Medical management

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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 management 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 evacuation 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 elevated ICP exceeding 20–25 mmHg is associated with a poor outcome, and maintenance of a CPP of at least 60 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 improve 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. Where these measures fail, neurointensivists may seek to control brain swelling using mannitol or hypertonic saline infusions. Where autoregulation is preserved, inducing high CPP may reduce ICP through vasoconstriction. A range of further interventions are effective in controlling ICP, but evidence for long-term outcome benefit is limited or absent. These interventions 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

Raised intracranial pressure Deranged Necrosis autoregulation Inflammation Hypotension Reduced Secondary cerebral brain injury perfusion Hypoxia Increased metabolic requirements – seizure, pyrexia, inflammation Figure 28.9 Brain swelling and mass lesions contribute to raised intracranial pressure, which compromises perfusion, leading to secondary brain injury and further swelling. TABLE 28.5 Key parameters to maintain in head-injured patients in neurointensive care. PaCO = 4.5–5.0 kPa 2 PaO

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[Na] >140 /uni00A0 mmol/L + [K]
>4 /uni00A0 mmol/L + + [K],
plasma potassium concentration;
[Na], plasma sodium
concentration; CPP , cerebral
perfusion pressure; ICP ,
intracranial pressure; MAP , mean
arterial pressure; PaCO , partial
pressure of 2 carbon dioxide in
arterial blood; PaO , partial
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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, administration of corticosteroids in severe head injury is associated with increased mortality and is not recommended. Seizures may occur early (within 7 days) or late. The cumulative 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 nutrition 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

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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