

Intensive care for traumatic brain injury

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A brief review of the causes, results, treatment and possible consequences of traumatic brain injury are presented by a world leader in the field.

Epidemiology

Traumatic brain injury (TBI) has often been termed a silent epidemic [1]. Precise data is scarce, but its incidence has been estimated in Europe at around 235 cases/year/100,000 inhabitants [2], with much worse figures in Asia and South America, connected with the increasing amount of road traffic [3]. After road traffic accidents, falls are an important cause of TBI. They occur more frequently in people aged 70 or older. Many factors contribute to the increased risk of falls in elderly people: walking impairment, dizziness, previous stroke, cognitive impairment, postural hypotension, poor visual acuity and multiple medication.

Interpersonal violence is involved in 2–15% of cases. Most TBIs are the result of blunt trauma, but in some countries penetrating injuries are more common, e.g. in the United States where gunshot wounds are the major cause and account for 40% of all head injury deaths, while 34% are secondary to road traffic accidents. TBI not only remains a leading cause of death in young males but in survivors leads to different degrees of disability, with personal discomfort, family strain and a high cost to society.

TBI ranges in severity from mild to fatal. It has a tendency to evolve over time so that even injuries that may seem mild or moderate at the time of accident can worsen in minutes/hours. The usual ratio of mild/moderate/severe is around 22:1.5:1 or for every severely injured patient there are about 1.5 moderately injured and 22 mildly injured patients [2].

Pathophysiology of traumatic brain damage

Head injury is a syndrome rather than a

single disease, comprising a wide range of anatomical damage (such as diffuse axonal injury, contusions and space-occupying intradural and extradural haematomas) in different combinations. Following the damage caused by the initial impact, called primary damage, additional tissue damage (named secondary brain damage) follows, due to a cascade of biochemical and biomechanical events. A typical example is a vascular injury, with initial damage to an artery of the skull, evolving into the accumulation of blood compressing the brain, as in epidural haematoma. Secondary brain damage expands the immediate tissue disruption, and is frequently exacerbated by systemic insults such as hypoxia and/or arterial hypotension.

Insults are generally events that produce an imbalance between the supply of substrates to the cerebral tissue and the metabolic needs of the tissue itself. Typically, insults cause ischaemia, either because they cause deterioration of the oxygen transport (as in case of anaemia, hypo-oxygenation or arterial hypotension), or because they cause an increase in the energy requirements of the tissues, such as in epileptic attacks or during hyperthermia. Mortality can increase threefold in people suffering arterial hypotension and hypoxia, compared to people with normal oxygenation and haemodynamics [4]. These data have been recently confirmed in the study from McHugh et al [5].

TBI is often associated with extracranial injuries, and sometimes the severity of those extracerebral lesions requires immediate surgery, as in case of spleen or hepatic haemorrhages. Under ideal conditions simultaneous surgical treatment of extracranial and intracranial

lesions can be devised, but in practice life-threatening haemorrhages should be treated first.

The combination of primary and secondary damage, and the deleterious role of systemic insults, has three consequences:

- qualified assistance, capable of preventing respiratory and circulatory imbalance, is essential at the scene of the accident
- every TBI has to be taken seriously and the patient watched carefully to exclude secondary worsening. CT scans, looking for intracranial masses that may require urgent surgical operation, are essential in people at risk [6]
- definitive treatment of severe TBI requires hospitals where several departments can cooperate: neuroradiology and emergency laboratory support are necessary for the diagnosis, neurosurgery for haematoma evacuation, trauma surgery for limb fractures or abdominal bleeds, intensive care for monitoring and care of severe injuries, etc.

Intracranial pressure in TBI

The cranial cavity is a rigid container surrounding delicate and incompressible elements: brain tissue, cerebrospinal fluid (CSF) and blood. The intracranial volume totals approximately 1,500–1,900 mL in adults; 80% of this volume is brain, 10% is blood and 10% CSF. When these volumes are in equilibrium the resulting intracranial pressure (ICP), is around 10 mmHg in the adult. This pressure can be measured in the lumbar sac by lumbar puncture, or at different points of the intracranial system (such as in the subdural space, directly in the parenchyma, or in the lateral ventricles) using special probes [7].

When additional volume is added to the cavity it is initially accommodated by displacement of CSF into the lumbar sac and/or by compression of the cerebral veins, with corresponding reduction of the amount of blood contained in the system. Most of the accommodation is by CSF displacement into the spinal cord. As soon as these buffering mechanisms are exhausted the ICP rises, along an exponential curve. Intracranial hypertension is defined as an ICP >20 mmHg.

The most effective way of preventing increased ICP is surgical removal of intracranial masses, such as haematomas or large contusions.

A common cause of raised ICP during the first hours after TBI is an enlarging haematoma, while different mechanisms are responsible for intracranial hypertension in the following days. Water increase in the cells or in the extracellular space (brain oedema), vessel dilatation, enlargement of contusions, CSF accumulation (hydrocephalus) are among the potential causes, often interacting with each other [8]. Intracranial hypertension has two main consequences:

- it may compress vital parts of the brain tissue and vasculature, with direct damage to the underlying structures
- it may cause a reduction in cerebral perfusion, leading to brain ischaemia. Cerebral perfusion pressure (CPP) is estimated by subtracting ICP from the mean arterial pressure. In normal adults CPP is around 60 mmHg. A clear relationship between low CPP and unfavourable outcome has been demonstrated in a large series of TBI patients [9].

Intensive care for TBI

Intensive care after TBI has two main goals: to maintain physiological values in all organs, as in all ICU patients, with special attention to preserving cerebral homeostasis.

Accurate control of systemic haemodynamic and respiratory function is therefore essential, coupled with maintenance of fluid balance, serum osmolality and adequate nutrition. Invasive arterial blood pressure monitoring is recommended with the reference point set at the same level as ICP measurement to allow accurate calculation of the cerebral perfusion pressure. Patients with severe TBI are routinely intubated and artificially ventilated to ensure adequate

oxygenation and to prevent aspiration of saliva or vomit into the airways, since comatose patients can not properly defend themselves.

Neurological examination, with routine clinical checks of motor response to pain, pupil size and reactivity to light, are important but often difficult because of sedation and pharmacological paralysis. Monitoring of ICP and CPP is recommended both by the European and North American guidelines in all patients with severe head injury with abnormalities on the initial CT scan [10, 11]. In patients with a normal CT scan the incidence of intracranial hypertension is lower, but is significant in the presence of two or more of the following risk factors: age over 40, unilateral or bilateral motor posturing, systolic blood pressure \leq 90 mmHg.

The most effective way of preventing increased ICP is surgical removal of intracranial masses, such as haematomas or large contusions. Medical treatment is based on the combination of sedation, analgesia, mannitol, CSF withdrawal and hyperventilation [12]. More aggressive treatments, such as extensive decompressive craniectomy or hypothermia, are currently being investigated in clinical trials.

Complications of ICP monitoring are haemorrhage due to introduction of the catheter and infection. The risk of infection is highest in case of ventricular monitoring and the rate of infection has been shown to be proportional to duration of monitoring. ICP and CPP monitoring provide information regarding the intracranial system as a whole. New techniques for the in-depth exploration of small volumes of the brain are available. Debate still exists whether such techniques should be employed in relatively undamaged parts of the brain, considered representative of global oxygenation and metabolism, or should be employed in the penumbra zone of contusions, with the purpose of rescuing potentially viable regions. These new tools allow tissue oxygenation, local blood flow and neurochemistry to be measured [13].

Outcome after TBI

Outcome after TBI depends on the severity of injury, the biological response to trauma (which is age dependent and is likely to have a relevant genetic component), quality of care from the scene of the accident to long-term rehabilitation, including social factors such as family support, financial resources, etc. Modern treatment of TBI has improved outcome considerably, with a mortality rate around 25% for severe cases; half of patients return to previous life with good recovery or moderate disability. Consequences of TBI are never negligible however, since even moderate injuries may result in disability in a high proportion of survivors [14].

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