THE MANAGEMENT OF HEAD INJURIES IN THE ICU

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A long, long time ago the god Shiva was returning home from a small war and, as he had won (again), he was in a fairly good mood. Unfortunately his wife, Parvati, was not expecting him and, wishing to bathe in peace, had fashioned a man out of clay, brought him to life and named him Ganesh. She instructed him to prevent anyone entering the house whilst she washed. Shiva finding entry to his own home barred by a stranger, briskly lost his temper and, a short while later, Ganesh equally abruptly lost his head. Parvati, disturbed by the commotion, rushed outside to find the decapitated body of Ganesh lying on the ground. A domestic incident ensued. In an attempt to make amends, Shiva promised to replace Ganesh's head with that of the first animal he found resting on the ground and facing east, which turned out to be an elephant. Shiva removed the elephant's head and placed it on Ganesh's body, thereby restoring not only Ganesh to life but also peace and harmony to the family home.

Ganesh

Unfortunately, head transplants have not gone so well since then. The management of head injuries in the intensive care unit (ICU) has traditionally rested on the belief that the patient should be sedated for a day or two, mildly hyperventilated and not given too much in the way of intravenous fluids, to reduce the chance of potentially damaging rises in the intracranial pressure (ICP).

The evidence to support this practice is missing. In fact, there has been increasing appreciation that this management is not only illogical but may actually be directly harmful to the patient, increasing the probability of poor cerebral perfusion and worsening patient outcome^(1,2). Nevertheless, trainees continue to learn from this ancient text, and patients continue to suffer because of it. Why should this be?

The crux of managing critically ill patients in the ICU depends on monitoring the patient's overall condition, focusing particularly on the damaged organ systems and trying to correct or reverse abnormalities in those systems if they should occur. If an organ does fail it may be possible to do without it entirely, or, if it is essential, replace it with a new one. Except if it's a head. In most DGHs, patients with significant head injury are managed by presumption – the damaged organ is not monitored, so we don't know what's going on inside the head and therefore can't make any attempt to correct problems. Instead, we look after the patient according to fairly general universal guidelines, with no ability to adapt those guidelines to an individual patient's needs. We hope for the best. And, heck, if they do badly, we know that there's nothing more that we could have done.

PRIMARY BRAIN INJURY

Apart from avoiding motorbikes and bungee jumping there is little that can be done to reduce the incidence of primary acute head injury (AHI). We are, therefore, limited to trying to minimise the consequences of the head injury once it has happened: surgical treatment where indicated and trying to avoid the development of secondary brain injury when surgery has nothing to offer. In fact, immediate access to neurosurgical intervention is so important the Royal College of Surgeons has stated that arrangements should be in place to ensure that patients who require emergency decompressive surgery are able to undergo it within four hours of the time of injury⁽³⁾. This is a laudable aim, but not one that is realised frequently enough. Anyone who has ever tried to arrange the interhospital transfer of a patient with an acute head injury will know how hard it can be to find a bed.

There is little to suggest that this is likely to change. On average at RLI we admit 10-12 head-injured patients annually to the ICU, and a further five or so who have not suffered an acute injury but would benefit from the management guidelines developed for head-injured patients. These numbers are too large to overlook.

SECONDARY BRAIN INJURY

Once the primary head injury has occurred, patients are at particular risk of secondary injury which is defined as anything which may worsen the initial cerebral insult.

Secondary head injuries may occur in up to 80% of patients with a severe head injury⁽⁴⁾ and are the cause of considerable long-term morbidity⁽⁵⁾. Of the possible secondary insults, **hypotension** and **hypoxia** are the two most powerful predictors of a poor outcome⁽⁶⁾. The injured brain not only loses its ability to match regional blood flow to regional metabolic need⁽⁷⁾ but also, in most cases, displays an increase in the cerebral metabolic rate⁽⁸⁾. The combination of these processes with systemic hypotension places the brain at exceptional risk of significant secondary ischaemia. Since an episode of hypotension can double the probability of a poor outcome, the most immediate management of AHI must be the detection and treatment of these physiological insults 'with the urgency of a cardiac arrest'⁽⁶⁾, on the presumption that raising the systemic blood pressure will lead to a proportionate rise in cerebral perfusion pressure (CPP) (the difference between the mean arterial pressure and the intracranial pressure). There is even some impressive evidence that raising the CPP from the normal value of 50mmHg to over 75mmHg, using either inotropes or vasopressors, may improve the outcome in patients with AHI⁽⁹⁾. Raising the target CPP is a response to the observation that in head-injured patients the loss of cerebral vascular autoregulation and the onset of 'pressure-passive' cerebral blood flow occurs at a higher CPP than in normal individuals. The cerebral blood flow is usually fairly constant between perfusion pressures of 50 and 150mmHg (Figure 1), but falls in proportion to the fall in perfusion pressure when it slips below 50mmHg. In the head-injured patient, assuming we can treat a heterogeneous population as a homogenous group, the onset of pressure-passive flow occurs at roughly 70-80mmHg. Since the ICP is generally also higher in patients with an AHI, the mean arterial pressure (the diastolic blood pressure plus one third of the difference between the diastolic and systolic values) necessary to preserve an adequate CPP in these patients needs to be at least 30 to 40mmHg higher than in the uninjured. Sedating and ventilating is simply not enough.

Figure 1 Normal cerebral autoregulation (solid line) and two possible variations following head injury (dotted lines). Cerebral blood vessel diameter is shown as open circles above. The figure of 80 mmHg is illustrative only.

The most powerful influence on the diameter of the cerebral blood vessels is the arterial carbon dioxide (CO_2) content. A rise in CO_2 content leads to cerebral vasodilation and increased cerebral blood flow, though since the blood vessels take up more room when they are dilated than when they are constricted it might also lead to an increase in ICP. In the presence of an already raised ICP it might therefore make

sense to hyperventilate the patient, thereby driving down the CO_2 , constricting the blood vessels and reducing the ICP. Though this, of course, also reduces the blood flow. It might, in fact, be the first example of treating an injured organ by cutting off its blood supply. The role of hyperventilation in the routine management of the head-injured patient is therefore somewhat dubious, unless there is some way of measuring the effect it has on the cerebral circulation⁽¹⁰⁾. Which of course there is.

MEASUREMENT OF CEREBRAL PERFUSION

The easiest way to measure whether there is adequate cerebral blood flow is to measure the oxygen saturation of the blood in the jugular vein and, by comparing it to the oxygen saturation in the systemic arterial blood, using ordinary arterial blood gases, work out what proportion of the oxygen delivered to the brain in the arterial blood has been removed during its passage through the brain⁽¹¹⁾. This can be done by inserting a jugular central line so that it goes upwards, until its tip lies within the skull, which can be easily checked on x-ray. It is then possible to calculate the jugular oxygen extraction ratio and, by seeing how this changes with greater or lesser degrees of hyperventilation, determine how much hyperventilation is compatible with an adequate cerebral blood flow. A high oxygen extraction ratio implies that the brain is being undersupplied with oxygenated blood, so the CO_2 should be allowed to rise to increase the vessel diameter and cerebral blood flow – and oxygen delivery – to the brain. A very low extraction ratio, by contrast, implies that the oxygen requirements of the brain are far below that which is being delivered, which may be either because a normal brain is receiving far more oxygenated blood than it needs or that there are so few remaining viable brain cells that almost none of the oxygen delivered is being used. Therefore, using the jugular oxygen extraction ratio, it is finally possible (and quite simple) to make sense of a mode of management that previously relied on wishful thinking. It is not, however, enough.

INTRACRANIAL PRESSURE

The ultimate disaster in the head-injured patient is an untreatable, or untreated, steady rise in the intracranial pressure. Even when being aggressively managed, the likelihood of a good outcome is inversely proportional to the maximum ICP, as well as the percentage of the time the ICP is above 20mmHg⁽¹²⁾. To some extent, the brain can compensate for rises in ICP due to cerebral oedema or haemorrhage by a corresponding decrease in intracerebral interstitial or cerebrospinal fluid (CSF). As the ICP rises, however, this compensatory mechanism becomes exhausted and further small rises in intracerebral volume may lead to a rapid and dramatic rise in the ICP, causing compression of the blood vessels within the brain or pressure damage to other intracranial structures. There are a number of ways in which a rising ICP may be treated and disaster averted, or possibly simply delayed, but to embark on these sometimes hazardous forms of treatment, such as hyperventilation, we need to know that the ICP is rising. Waiting for the development of clinical signs, such as a deteriorating Glasgow Coma Score or changes in the pupil size, simply allows us to make the diagnosis when it is too late to do anything about it. What we need to be able to do is to measure the ICP directly.

Monitoring the ICP in head-injured patients requires drilling through the skull and inserting a measuring device of some sort into, or beside, the brain. Until recently this has been seen as the pursuit of the neurosurgeon, who is able to deal with the possible bleeding that may result from the placement of a monitoring device, by performing a craniotomy or drilling a burr hole.

Is it practical to insert intracranial devices in a DGH? Most assuredly yes.

MEASUREMENT OF INTRACRANIAL PRESSURE

The development of intracranial pressure measurement devices has made monitoring the ICP progressively easier, so that many DGHs now routinely use ICP monitoring kits, in particular the Codman microtransducer, on their head-injured patient⁽¹³⁾, (Figure 2). To date, there are no significant reports of complications. The Codman system is a portable intraparenchymal device – that is, the tip of the pressure transducer is placed into the brain tissue via a small hole drilled through the skull. This allows for reliable measurement of the ICP over many days, with minimal signal

Figure 3 Camino intraparenchymal device in situ, showing pressure sensor within parenchyma of brain, securing screw within skull and portable monitor

drift (which is crucial since devices cannot be recalibrated once inserted) and the facility to transfer the patient, with the device in-situ, within and between hospitals. Other methods of monitoring the ICP, such as near-infrared spectroscopy, or the transcranial Doppler measurement of the arterial waveform in the middle cerebral artery, are possible and may be less invasive or, in the case of cerebral ventricular catheters, allow more direct measurement and treatment of a raised ICP. None provides the simplicity and robustness of the intraparenchymal sensor^(12,14). Furthermore, neither the training required nor the start-up costs incurred (about £5,000) should provide a significant barrier to the introduction of this technique into the average DGH. It might

even be argued that if a DGH is not able to provide appropriate care to head-injured patients they should all be transferred to a hospital that can. Indeed, within the northwest region, a neurosurgeon from each neurosurgical centre has provided training in ICP bolt placement for DGH intensivists to encourage the development of the service. Unfortunately, whilst their support is very welcome, it has not always been given with the unanimous support of their colleagues.

DO THESE MEASUREMENTS IMPROVE OUTCOME?

Is there evidence that manipulating the ICP, or optimising the CPP, improves outcome? Unfortunately not. Since clinical equipoise does not exist – that is, no-one really doubts that managing patients to reduce their ICP helps their long-term outcome – there is no serious prospect of such a study ever being done in man. What is known is that the death rate associated with traumatic brain injury is dropping, though whether this is due to improved patient management or improvements in the design of cars, helmets and passenger restraints is unclear⁽¹⁵⁾. Whilst it may not be possible to identify which components of patient care are of the most individual benefit it is, thankfully, true that programmes of aggressive management of severe head injuries in critical care lead to improved patient outcome. In the context of head injury this does not necessarily mean fewer deaths but rather fewer survivors with severe or moderate persisting disability⁽¹⁶⁾.

OTHER FACTORS

However attractive it may be to measure the ICP in patients with AHI, such measurement it is of no use unless it is part of some coherent plan for patient management. There is a somewhat regrettable pressure building for the imposition of guidelines for the management of particular conditions, which threatens to constrain the intuition and practice of individual clinicians. Nevertheless, where there is a clear reason to adopt particular clinical practices, and where traditional management promotes an illogical antithesis of those practices, there is a potential place for guidelines. At the infirmary we have, therefore, developed guidelines for the management of head-injured patients in ICU, which are both fairly didactic and, we think, appropriate. We have subsequently presented these guidelines to the Association of Northwest Intensive Care Units (ANWICU), who have accepted them as the standard of care across the region. Our guidelines deal not only with the points mentioned above, but also matters such as

- the early control of post-head injury pyrexia (which is virtually universal, increases CO₂ production and may worsen hypoxia)
- appropriate methods of ventilation
- fluid resuscitation
- electrolyte balance
- nutrition
- the sensible use of mannitol.

We must also remember all the other diverse matters which are necessary for the care of the head-injured patient. As more is known about the complex biochemical and

pathological processes involved in the process of injury – the potential protective role of magnesium supplements or calcium-channel blockers, or the place of deliberate hypothermia, for example – our guidelines will incorporate them and, we hope, we will continue to be able to provide the level of care our patients deserve.

The outcome from AHI is often disappointing, even in patients in whom the initial injury was deemed to be only mild. In fact, a recent study demonstrated that one year after head injury about 47% of survivors had persisting moderate to severe disability, irrespective of whether they initially presented with mild, moderate or severe head injury⁽¹⁶⁾. These are depressing statistics, and emphasise that we should be doing whatever we can to improve outcome. The correct approach must surely involve the meticulous control of possible secondary insults, early monitoring and immediate management of an elevated ICP and rapid delivery to the neurosurgeons when appropriate. Sedation, ventilation and prayer may help, but they will no longer suffice.

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