Acute Head Injuries: Anaesthetic Considerations

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In the first part of this article I describe a personal approach, in the second I will elaborate on the controversial aspects of the management of these patients.

Traumatic head injuries produce two types of brain injury. The primary injury occurs in the first few milliseconds of the trauma and consists of the biomechanical effect of forces applied to the brain. These are manifest by contusions and lacerations of the brain, and diffuse vascular injuries with petechial haemorrhages. Potential also exists for direct damage from intracerebral haemorrhages. There is no treatment, at this stage, for these injuries.

The secondary injury occurs minutes to hours after the trauma and represents the complicating processes initiated by the primary injury. A common feature of this is cerebral ischaemia both focal and general. Hypoxia, hypotension, arterial/venous hypertension, hyperglycaemia, and raised intra-cranial pressure (ICP) (haemorrhage, brain swelling, brain oedema, hypercapnia, hypoxia) may contribute. The secondary injury is amenable to treatment and this is where we must currently focus our attention.

An Approach to management:

The key to management is to deal with these patients as emergencies. This relies on early recognition of those patients with potential intracranial injuries, immediate correction of factors leading to secondary injuries, rapid diagnosis of intracranial pathology (CT scan) and then immediate delivery of the definitive management (often surgical).

Any patient who is not able to fully cooperate to have a CT scan must be intubated and have controlled ventilation. No patient with a head injury should be sedated for a scan. Studies suggest a 6-8% prevalence of cervical spine fractures in head injured patients, and if lateral spine views (including C7) are not available the prudent course is to assume a cervical spine fracture exists. It is worth bearing in mind that simple lateral views do not fully rule out a fracture, for this one needs a full cervical spine series. Unless a difficult intubation is anticipated, the intubation technique should use a standard rapid sequence induction. I use an appropriate dose of thiopentone (not propofol as it causes more hypotension), Suxamethonium (there are no important increases in ICP if adequate doses of thiopentone are used, you need to use a nerve stimulator and wait till the twitches have disappeared or wait about 20 seconds AFTER the fasciculations have finished to ensure they are fully paralysed) and cricoid pressure (with strict neck immobilisation, minimising extension unless a cervical spine fracture has been excluded. If you have Alfentanil available then that is a useful adjuvant to intubation in a dose of between 5-30μg/kg (depending on age, other disease states and cardiovascular stability). Fentanyl is no use as it onset time is too slow to be a worthwhile adjuvant to intubation in a rapid sequence induction.

It is sensible to remove a neck collar, including those with openings, prior to doing this as they make intubation more difficult. It can then be replaced after intubation is successful. If a spinal fracture is suspected then placing a hand behind the neck, to prevent posterior displacement of the spine when cricoid pressure is applied, is also useful. Once the trachea is intubated the patient should be given a long acting muscle relaxant (which has not cardiovascular side effects, eg Rocuronium 50mg) to prevent coughing and hyperventilated with oxygen.

I would ventilate these patients on 100% oxygen until the patient is transferred to ICU/Theatres in which case the $Fi0_2$ can be reduced to the level needed to maintain a SpO_2 of 98%.

If there are no overt signs of raised ICP (dilated pupils) then I would try to use mild hyperventilation only. If you have transport capnography, then I would aim for an $ETCO_2$ of about 30. If there is clinical evidence of coning or markedly elevated ICP on the CT scan then it is justifiable to go lower than this ($ETCO_2$ of 20-25mmHg).

Fluid resuscitation must be with isotonic fluids only, e.g. normal saline. Hartmann's solution is hypotonic and should not be used. The only place for colloids is when there is a need to deliver large volumes of fluid and blood is not available (but only because on a ml for ml basis colloids are better at volume restoration than crystalloids). Glucose containing fluids are usually hypotonic, lead to hyperglycaemia and should be avoided. If, after 2 units of crystalloids and/or colloids, the blood pressure is not rapidly restored then blood should be given if blood loss is assumed to be the cause of the hypotension; either 0-ve or group specific. Anaemia should be corrected and some evidence exists to suggest that the optimal haemoglobin is 100gms/l.

If the patient is unconscious and haemodynamically stable then I would give a large dose of mannitol (1.5gm/kg) over 20 minutes. Frusemide 0.3mg/kg will augment this effect. A urinary catheter should be placed.

Anaesthesia consists of unrousable unconsciousness and reflex depression, both motor and autonomic. These patients are usually unconscious or at the very least likely to be amnesic but reflex depression is always needed. Paralysis provides motor reflex depression but transporting patients around a hospital can be very stimulating and extreme hypertension may occur if nothing else is used. I use 100μg doses of fentanyl ($\leq\approx$500μg) to help prevent this. Acute episodes of hypertension are best managed by boluses of thiopentone or Propofol as they have a more rapid onset than opioids. Midazolam is not ideal in this setting as it is not a good reflex depressant and has a slower onset.

An alternative, but only if all the equipment is immediately available, is to use a Propofol infusion at about 2-8mg/kg/hr or for TCI between 1-4μg/ml. The actual dose depends on the blood pressure responses.

Half of **t**his patient group have other major injuries, the management of which may take precedence over their head injury. One must treat the most life threatening condition first but treatment of head injuries comes before treatment of other non-life threatening conditions. If these patients need urgent non-neurosurgical procedures then one should use the same anaesthetic principles outlined below. The placement of an ICP monitor allows optimal management.

The question of the ideal blood pressure for these patients is controversial (see later). I aim for a mean blood pressure around 80-100mmHg or 120-150mmHg Systolic in adults and only control it with fentanyl or thiopentone/propofol. Thiopentone and Propofol depress the cerebral metabolic rate (CMR) and cerebral blood flow (CBF) and so will reduce the ICP. Vasodilators such as diazoxide, hydralazine, nitroglycerine, or nitroprusside may be catastrophic in this situation. Experience suggests that paralysis; mild hyperventilation, thiopentone, and fentanyl are almost always sufficient to control the blood pressure. I transport these patients with $\approx$15% head up to further decrease their ICP.

One also needs to avoid simple things that increase ICP eg head position, ties obstructing neck veins, coughing (all patients should be fully paralysed after intubation until they are delivered to the ICU), PEEP or excessive ventilation.

Assuming the patient is stable enough for a CT scan it is imperative that no anaesthetic drugs that are cerebral vasodilators be used. This means no nitrous oxide, inhalational agents, or ketamine. The patient should remain paralysed to prevent coughing and to minimise the intrathoracic pressure. Once the CT scan is done, a definitive plan of management will then be made.

Invasive monitoring will be needed in most of these patients but time should not be wasted while this is instituted. They can be placed during any delays in the Emergency department or while the head is shaved in theatre.

If the patient is going to theatre for a craniotomy then my technique is to run a propofol infusion (a cerebral vasoconstrictor), ideally using TCI. I start at 2μg/ml and increase as necessary but do not go below 2 (it is preferable to keep it above 3 especially after the brain is decompressed). If this is not available then use ≈7mg/kg/hr.

I then add fentanyl (<10μg/kg) in divided doses (50-100μg boluses). The fentanyl dose depends on the blood pressure response and little may be able to be given prior to skin incision. Transiently increasing the TCI to 6μg/ml for 60 seconds prior to the Mayfield pins (that fix the head) and continuing for 1-2 minutes after will usually control the BP response to pin placement. The TCI should then be reduced to the pre-pin level. The skin incision line is infiltrated with 0.5% bupivacaine and 1/200,00 adrenaline. I aim for an endtidal CO_2 of ≈30mmHg (≈25 if there is evidence of conning).

If you have time, equipment and the experience you can use a Remifentanil infusion instead of Fentanyl however time is of the essence and you should not do this unless you really do not how to use it (this is not the time to experiment if you have never used it before). You should only use a programmable syringe pump for this and start the infusion rate at about 5μg/kg/hr, it can then be increased or decreased as necessary. If I do this, then I would not go above 4μg/ml of propofol and use the Remifentanil to control the BP. Boluses of Remifentanil (0.5μg/kg) can be used to prevent the BP increase with Pins. Acute hypertension later in case can be controlled with smaller boluses (starting at 0.25μg/kg). If you have a TCI Remifentanil pump then use "Effect Site" targeting with starting concentrations of 2mg/ml and then between 1-6ng/ml during the case.. Transiently increasing the Remifentanil concentration to about 4-6ng/ml will cover the stimulus of Mayfield pin insertion (or intubation). Assuming the haemodynamics are stable a value of 6ng/ml is usually about right but you must avoid hypotension so erring slightly on the low side is best. It will then need to be reduced to pre-pin levels once it is clear the BP is not continuing to rise.

If hypotension occurs then one should decrease the Remifentanil (if used) and Propofol. I would treat the hypotension in the interim with IV fluids and vasopressors, eg phenylephrine or metaraminol. Remember hypotension is catastrophic in these patients. I would aim to keep the MAP at or above 80mmHg.

If you have time and can find a place on the skull to put the electrodes then either BIS or Entropy monitoring can be used to guide the administration of the anaesthetic agents. Typically one aims to get the value below 60. Given the likely sub-optimal placement of the electrode I would not go below a Propofol TCI of 2μg/ml.

Once the brain is decompressed (when the bone flap is elevated in an extra-dural haematoma or, more commonly, when the dura is opened with a subdural haematoma) you may see a quite marked fall in BP. This relates to the fall in sympathetic tone when the previously raised ICP (which has produced a Cushing reflex) is normalised. The fall in pressure is more marked if the patient is hypovolaemic. Kawaguchi and co-workers performed a retrospective study on acute SDH to determine preoperative risk factors for sudden MAP decrease on dural opening. Low Glasgow Coma Scale score, absence of the mesencephalic cistern on CT scan, and bilaterally dilated pupils were all found to be predictors of a MAP reduction of more than 20% on dural opening. It is said to be more common in sub-dural rather than extra-dural lesions.

You need to prepare for this by ensuring the patient is well hydrated and having a vasopressor handy. Should the BP fall below a mean of 70mmHg you should treat this with vasopressors, fluids and reducing the anaesthetic agents. The patient should be able to tolerate a lower BP at this stage, as the ICP should be substantially reduced.

Further management depends on whether the patient should be extubated at the end of the operation. Peak brain oedema can take 24-72 hours to occur so it is usually prudent to leave the trachea intubated (ensure that an adequate amount of fentanyl is given to prevent hypertension during transport to ICU). An exception is the patient with an acute extradural who was initially well after the trauma and then suddenly deteriorates. Such patients may awaken once the brain is decompressed.

Patients with acute traumatic subdurals will usually have underlying brain trauma (the sub-dural is due cortical vessel damage rather than bridging vein damage as occurs with chronic subdurals) and so one can expect a degree of brain swelling.

Occasionally the brain will have such marked cerebral oedema that the brain will bulge out of the craniotomy wound. Doing wide, sometimes bilateral, decompressive craniotomies may be needed.

Decompressive craniotomies have been practiced for millennium. With the practice of "trephination" being described by the ancient Greeks and more recently by Kocher about 100 years ago. The practice has come back into favour fairly recently for those patients with markedly elevated ICP despite maximal medical therapy and in the absence of intracranial mass lesions.

The technique clearly reduces ICP and there is some evidence of improved outcome (but not level 1 or 2) when the procedure is done early (within 24-48 hours). The potential complications are not well described and the patient will have to have further procedures to replace the bone flaps or fashion new prosthetics to repair the bony deficit. There are a number of randomised trials proposed or started that will help delineate the role of Decompressive craniectomy in traumatic brain injury.

If it is planned to leave the trachea intubated then I continue with the anaesthetic as outlined except that I would return the ETCO2 to ≈35torr. If extubation is planned for the conclusion of surgery (uncommon) then one can either continue with the TIVA technique or reduce the Propofol and add a <1mac of Sevoflurane or even use 65% nitrous. Allow the endtidal CO2 to increase to the about 40torr so that you can see if the brain will be "tight" at the level of PaCO2 that is likely to occur soon after extubation. Should the addition of inhaled anaesthetic agents make the brain to become swollen they should be withdrawn. Unless the swelling resolves rapidly the patient should remain intubated post-operatively.

I will give no more fentanyl (if used), use propofol (and Remifentanil if used) to control the blood pressure Cease propofol about 10 minutes before surgery ends (if an inhaled agent is added) or reduce the amount if TIVA is being used. Continue or increase the Remifentanil to control the BP or Entropy/BIS (if used). Use metoprolol to help control BP if >10μg/kg/hr Remifentanil is needed to control the blood pressure during this final ten minutes. The patient can be extubated provided they are conscious, able to maintain and protect their airway, can breath adequately, clear their secretions, well oxygenated and cardiovascularly stable.

If a patient with a suspected head injury needs other more non-cranial urgent surgery then placement of an ICP monitor whilst that procedure is underway allows optimal management of the patient's intra-cranial state until such time as definitive investigations and management are possible. In this case, the anaesthetic technique is the same as for a craniotomy except that in a cardiovascularly unstable patient I would tend to use more propofol (which has a short duration of effect) rather than the relatively more long lasting fentanyl. If Remifentanil is used this allows for far more rapid changes in the degree of reflex suppression.

If a ventricular catheter is placed (which will usually be the case with patients needing urgent Decompressive surgery), then drainage of CSF can be used to control the brain swelling/ICP. Patients with slit ventricles may be difficult to place a ventricular catheter.

Occasionally a patient with traumatic brain injury will develop DIC. This cascade is thought to be initiated by exposure of the subendothelial collagen and release of brain tissue thromboplastin, which activate both the intrinsic and extrinsic coagulation pathways. If this occurs replacement of the coagulation factors will be needed. The more severe the brain injury the more likely this is to occur.

Overall, don't waste time, and treat head injuries as emergencies. Try to treat the physiological changes that cause secondary injuries before they cause damage, have a low threshold for doing a CT scan, try to reduce ICP (and brain shift) and deliver the definitive management as quickly as possible.

Controversies

Hyperventilation

This used to be the mainstay of management of raised ICP, however it is now recognised that it may cause cerebral ischaemia in these patients even with relatively small reductions. In normal people, hypocarbia does not cause cerebral ischaemia until the arterial CO2 tension is reduced below 20mmHg. The other problem is that its effects are only short lived because CBF will have returned to baseline by twenty-four hours. This means any subsequent slight increase in PaCO2 (from the previously low level) leads to increased CBF. Hypercarbia on the other hand can lead to brainstem herniation. In patients with luxury perfusion short-term hypocarbia is justifiable but luxury perfusion cannot be determined clinically.

The general consensus is for normocarbia or slight hypocarbia ($PaCO_2$ of 35mmHg).

In patients with evidence of conning (eg unequal pupils) or marked increases in ICP (>20mmHg) hyperventilation is justifiable as an emergent treatment. In the absence of such signs and prior to surgery I would aim for mild hypocarbia ($ETCO_2$ ≈30). As we are often manually ventilating patients without CO_2 monitoring during transports I would err on the side of mild hyperventilation. Once the brain is decompressed or the ICP is found not to be elevated, $PaCO_2$ should be returned to normal.

The only outcome study (Muizelaar 91) was comparing chronic ICU hypocarbia v normocarbia and showed worse outcomes at 3/12 and 6/12. There are no outcome studies looking at the use of acute hypocarbia.

Fluids

Much has been written about the use of either crystalloids or colloids in the resuscitation of trauma patients either with or without head injuries and it is indisputable that for equivalent volumes colloids will produce a greater expansion of intravascular volume. However, many of the studies that suggest an increase in cerebral oedema are flawed because they used hypotonic crystalloids.

The blood brain barrier is normally impervious to most ions, transport across being actively controlled. This means that total osmolality rather than the colloid osmotic pressure (COP is ≈ 1.5mosmoles/kg) is the important osmotic factor (unlike at other capillary beds). Many studies that have looked at the cerebral effects of crystalloids have used Ringer's Lactate or Hartmann's solution as the crystalloid and stated that this is isotonic. Hartmann's solution has 280 mmol of dissolved ions present but due to incomplete dissociation there are not 280 mmol of particles. The actual osmolality is 265mosmole/kg (normal plasma osmolality is ≈285) that is quite hypotonic. Normal saline (308mmols of ions) has an osmolality of 285 and is isotonic.

Most Studies that have kept total osmolality constant and reduced COP have not shown an increase in cerebral oedema. However there is one paper by Drummond in 1998 that looks at colloid

oncotic pressure in an animal model where the severity of the brain injury was just sufficient to begin to cause BBB dysfunction (as assessed by Evan's blue leakage into the brain parenchyma). This was a "mild" brain injury. They then compared fluid resuscitation with blood, Hetastarch 6% in normal saline, saline whilst keep plasma osmolality constant. In the saline group COP was reduced to about half normal (representing fairly aggressive saline administration). They also had a group with half-strength saline. The blood and Hetastarch solutions did not differ in oedema formation (they did not reduce either total osmolality or COP). Half-strength saline had the greatest increase in oedema.

6% Hetastarch is not available in Australia and is limited to 20ml/kg/d due to concerns about coagulation.

Volvulen, a new 6% Pentastarch (Hydroxyethyl Starch - HES 130/0.4) has normal saline as it's crystalloid base and has no significant coagulation issues and can used in volumes up to 33mls/kg/day (this is the Australian TGA approved limit, it has been used in volumes up to 70mls/kg). It would, theoretically, be the ideal colloid to give for head injury resuscitation.

Many of the older "colloids" available in Australia (all the Albuminex products) are actually slightly hypotonic. Recent analysis of the "SAFE" has shown that head injured patients have a worse outcome if Albuminex is used instead of Normal Saline for ICU resuscitation.

From the above-mentioned studies we can postulate that, depending on the severity of the BBB damage, we will have brain areas where the osmotic/oncotic gradient is totally effective (normal BBB), areas where only the colloid oncotic gradient is effective (mild opening of the BBB, with pore size similar to the periphery), and areas where there is no osmotic/oncotic gradient effect (BBB breakdown).

The message is to avoid and/or correct, in patients with brain or spinal cord injury, a decrease in ''both'' serum osmolality and COP. This message, however, is part of the ''common clinical sense.''

Other than situations where the rate of administration is a problem there is no advantage in using colloids with regard to cerebral oedema. Overall then, normal saline should be the crystalloid fluid used at least in the acute stages of the patient's management.

Hyperglycaemia has been shown to cause a worse neurological deficit for a given degree of cerebral ischaemia and given that ischaemia is extremely common in this group of patients this needs to be avoided. 5% dextrose, once the dextrose is metabolised, is pure water, which will lower plasma osmolality that may worsen cerebral oedema.

One of the independent factors in a worse outcome from head injury is anaemia (Hb <100gms/l). There are some animal studies suggesting that a haematocrit of 30% provides optimal oxygen delivery in cerebral ischaemia but an haematocrit less than this is worse than higher haematocrits. Overall, one should start blood early in multi-trauma resuscitation to prevent anaemia.

Blood Pressure

The cerebral haemodynamics of acute head injuries have been extensively studied in both animal models and humans. One of the major problems has been the difficulty of measuring CBF in early hospital management, especially if operative intervention is necessary. Recently the advent of Stable Xenon enhanced CT scanning has meant that regional and global CBF/CBV have been able to be measured at the time of the initial CT scan. This has only been done in adults.

These studies show that there is a high prevalence of ischaemia (31%) at this time (about 3 hours after injury). Unless there was evidence of brain stem herniation (unilateral pupil changes) then

ischaemia was more marked supratentorially. If there were unilateral pupil changes then ischaemia was more marked in the brain stem. In patients with a subdural haematoma this ischaemia was reversed following decompression. In patients with a global decrease in CBF this was reduced for the first two days and then, on average, returned to normal. This was not however a uniform finding. There are some patients (≈10%) who have hyperaemia that contributes to their raised ICP, while others have normal flows and some have low flows. If metabolism is not appropriately reduced (as it is in many patients) then this may lead to ischaemia. Unless jugular venous oxygen content is measured (and from that cerebral oxygen consumption calculated) it is impossible to determine if flow is appropriate (coupled).

Hypotension (systolic BP <90mmHg in an adult) at any stage of the hospital stay (arrival, resuscitation, in theatre or in the ICU) has been shown to dramatically decrease the patient's likelihood of a good outcome. However just maintaining SBP > 90mmHg may be insufficient to prevent cerebral ischaemia, as animal and human studies have also shown that autoregulation is impaired with head injuries and that the lower limit of autoregulation is shifted to the right. cerebral perfusion pressures (CPP) of less than 70mmHg commonly produces decreased CBF (cf 50mmHg in normal people). The accepted aim has been to maintain CPP ≥70mmHg. Some patients may even need higher CPP than this. Rosner et al 1990 has demonstrated that in some patients a higher CPP will lead to a reduced ICP (autoregulatory vasoconstriction). This is only the case where autoregulation is still intact, unfortunately in only about 50% of head injured patients is this the case.

On the other hand hypertension might be undesirable if autoregulation is impaired leading to increased CBF (which occurs in some patients) or if it worsens bleeding (leading to greater mass effect).

Claudia Robertson in 2001 reviewed the literature on CPP control and showed that there was no advantage to elevating the CPP > 70mmHg and an increased risk of ARDS (which had an increased mortality). She concluded that, as a general rule, one should aim for a CPP of 60mmHg.

There may be however individual patients who benefit from a CPP greater than this. Only with specific monitoring of cerebral ischaemia is it possible to determine in which patients this will occur.

The "Lund" approach has been promoted recently which suggests the opposite! They emphasises the need to avoid ongoing progressive brain oedema in an attempt to ensure adequate regional perfusion. They use low CPPs (down to 50) in order to reduce capillary hydrostatic pressure, reduction of Cerebral Blood Volume, with maintenance of colloid osmotic pressure. This relates to ICU management and the two may not be inconsistent with differing management required at different phases of the patient's management (or for differing pathophysiology).

The absolute level of ICP is also of some concern independently of its effects on CPP. It has been well described in studies done in the 1950s (and in patients with benign intracranial hypertension) that uniform increases in ICP of ≤100mmHg produce minimal effects. Similarly we know that induced hypotension to 50mmHg produces no ill effects in the majority of normal patients. Non-uniform increases in ICP, which cause brain shift and herniation, on the other hand cause problems at a much lower ICP. Evoked potential changes occur with supratentorial ICP >30mmHg and we know clinically that head injured patients deteriorate with ICP at this level (before CPP may be compromised).

Overall, in the acute setting at least, I aim for a systolic blood pressure around high "normal" (≈150mmHg in adults). Ideally the CPP should be optimised to the patient's ICP and SJVO2 but this is not practical in the emergent situation. Systolic blood pressures > about 160mmHg I would try to reduce with Fentanyl/Thiopentone/Propofol. Fentanyl does not act quickly and should not be used for control of rapid increases in blood pressure.

Remember to take into account the effect of head elevation if you are measuring the BP with a cuff (manual or NIBP) or to position the transducer at the level of the head if one is using invasive monitoring.

Head Elevation

Whilst not all authors agree the majority of studies indicate that 15-30°head elevation reduces ICP without compromising CPP or cerebral oxygenation. Marked degrees of head elevation >30° may actually increase ICP in some patients by causing autoregulatory vasodilatation. Again ideally, head elevation should be optimised to CPP. I usually use about 20° head elevation in the acute management of these patients.

Diuretics

Mannitol is extremely effective in reducing ICP. It does this by its osmotic action causing brain shrinkage and by producing vasoconstriction secondary to a reduction in viscosity. The effects start within 10 minutes and peak at about 60 minutes. Given the extreme importance of improving the CBF to prevent cerebral ischaemia, and given the minimal side effects of mannitol I feel that any unconscious head injured patient should be given mannitol 1.5gm/kg as soon as is practical. Despite what is commonly said, mannitol, given rapidly, may produce profound hypotension (not hypertension) and should be given over 20 minutes. The other important side effect of rapid administration of mannitol is transient hyperkalaemia (≤2mmol/l).

Cruz has shown that giving doses of mannitol of 1.5gm/kg (cf 0.7mg/kg) when signs of brain stem compression are seen (pupillary widening) improves outcome in traumatic non-missile acute intraparenchymal haemorrhage, acute subdurals and diffuse brain swelling.

Anticonvulsants

Long-term administration of anticonvulsants has not been shown to be necessary in head injured patients however ≈10% of severe head injured patients who do not have anticonvulsants have seizures in the first week of hospital stay. The consequences of seizures are a severe increase in CMR, CBF and CBV that lead to marked increases in ICP that may be catastrophic. The risks of short-term phenytoin are small and I would suggest that all head injured patients requiring surgery should be loaded with phenytoin (15mg/kg). One needs to give this slowly as it causes hypotension. Whilst it is clear that short term used of phenytoin reduces the risk of early seizures meta-analysis does not reveal a long-term benefit however.

Steroids

The Cochrane collaboration performed a meta-analysis on steroids in head injury and showed only a 1.3% benefit overall. When the best studies only were included however, there was no difference. Despite this they concluded the matter needed further study. The follow-up CRASH study was published in 2004 and looked at the use of Methylprednisolone. It was stopped early (after 12 or 20,000 planned patients were recruited) when interim analysis showed that not only were steroids not of benefit but the overall mortality was higher in the steroid group compared with control. The guidelines of the American Society of Neurological Surgeons recommend not using them.

Hypothermia

There were a number of early studies that showed mild hypothermia (32-34°C) is beneficial in head injuries. It reduces CMR, CBF, ICP and improves outcome. A recent large multi-centre trial on the use of hypothermia in head injuries failed to show any benefit. It may be that the reason for this was that in the earlier studies the "normothermic" group included patients patient who were

hyperthermic. Hyperthermia, by as little as 1°C, has been shown to worsen outcome in a variety of cerebral ischaemia settings. It is generally accepted that one must avoid hyperthermia when cerebral injuries are present. Usually with multi-trauma, avoiding hyperthermia is not difficult as the patient will almost always get cold during their initial hospital care. I would try and warm these patients but probably aim for 35-36°C only to avoid them becoming hyperthermic. Hypothermia is one of the modalities that can reduce ICP and if brain swelling is a problem I would not warm past 34-5°C.

CSF Drainage

The placement of a ventricular catheter allows for both monitoring of ICP and a means to control ICP by removing CSF. There is a slightly greater incidence of infection for ventricular catheters compared with intra-parenchymal catheters or sub-arachnoid bolts.

Surgery for non-life Threatening Injuries

The timing of surgery for non-life or limb threatening orthopaedic surgery is controversial. The risk is hypotension that may be associated with this surgery. Hypotension in the first 24-48hrs of head injury is associated with a worse outcome.

Ideally the minimum should be done in the first 48-72hrs to allow the patient to stabilise. If surgery is necessary within this time frame then it should proceed under ICP control with careful maintenance of CPP, oxygenation, $PaCO_2$ and avoidance of anaemia.

References

Admides et al. Current Controversies in the management of patients with severe Traumatic Brain Injury. ANZ. . Surg. 2006: 76: 163-174

Bayir et al. Promising Strategies to Minimise secondary brain injury after head trauma. Crit Care Med 2003 (3) 1 Supplement S112-117.

Beddell et al. Anesthetic Management of Traumatic Brain Injury An Clin N. America 20 (2002) 417-439

Bouma, G.J., et al: Blood pressure and intracranial pressure-volume dynamics in severe head injury: relationship with cerebral blood flow. Journal Neurosurgery. 1992. 77: 15 - 19.

Bouma, G.J., et al: Evaluation of Regional Cerebral Blood Flow in Acute Head Injury by Stable Xenon-Enhanced Computerised Tomography. Acta Neurochir (Suppl). 1993; 59: 34 - 40.

Bouma, G.J., et al: Ultra-early evaluation of regional cerebral blood flow in severely head-injured patients using xenon-enhanced computerised tomography. Journal of Neurosurgery. 1992; 77: 360 - 368.

Bouma, Gerrit J., et al: Cerebral Circulation and Metabolism after Severe Traumatic Brain Injury: The Elusive Role of Ischemia. J. Neurosurg, 1991; 75: 685 - 693.

Chesnut, R.M., et al: early and Late Systemic Hypotension as a Frequent and Fundamental Source of Cerebral Ischemia Following Severe Brain Injury in the Traumatic Coma Data Bank. Acta Neurochir (suppl). 1993; 59: 121 - 125.

Chestnut RM, Prough DS, Critical Care of Severe Head Injury (Symposium). New Horizons, 3 (3) 1995

Clifton GL, et al: Lack of effect of induction of hypethermia after acute brain injury. NEJM 2001; 344: 556-63

Cold, G. E. Measurements of CO2 Reactivity and Barbituate Reactivity in Patients with Severe Head Injury. Acta Neurochir. 1989; 154 - 163.

Cold, G.E. Does Acute Hyperventilation Provoke Cerebral Oligaemia in Comatose Patients After Acute Head Injury? Acta Neurochir. 1989; 100 - 106.

Cold, G.E., Cerebral Blood Flow in Acute Head Injury. The Regulation of Cerebral Blood Flow and Metabolism during the Acute Phase of Head Injury, and its Significance for Therapy. Acta Neurochir. 1990; Suppl-49: 1 - 64.

Cold, G.E., Does Acute Hyperventilation Provoke Cerebral Oligeamia in Comatose Patients after Acute Head Injury ? Acta Neurochir. 1989; 96: 100 - 106.

Cold. G.E., et al: The Effects of Paco2 Reduction on Regional Cerebral Blood Flow in the Acute Phase of Brain Injury. Acta Anaesthesia. Scanda. 1977: 21: 359 - 367.

Coles et al. Effect of hyperventilation on cerebral blood flow in traumatic head injury: clinical relevance and monitoring correlates. Crit Care Med 2002; 30(9): 1950-9

Coles et al. Hyperventilation following head injury: Effect on ischemic burden and cerebral oxidative metabolism. Crit Care Med 2007; 35 (2): 568-578

Coles et al. Incidence and Mechanisms of cerebral ischemia in early clinical head injury. J. Cerebral Blood Flow Metabolism 2004; 24: 202-11

Cottrell JE, Smith DS. Anesthesia and Neurosurgery IIIed 1995 Mosby

Cruz, J. Traumatic Brain Ischemia during Neuro Intensive Care: Myth rather than Fact? Arq Neuopsiquiatr. 2001, 59 (3-A) 479-482

Cruz C, Minoja G, Okuchi K. Improving clinical outcomes from acute subdural hematomas with emergency preoperative administration of high doses of mannitol: a randomized trial. Neurosurgery 2001;49(4):864-71.

Cruz C, Minoja G, Okuchi K. Major clinical and physiological benefits of early high doses of mannitol for intraparenchymal temporal lobe hemorrhages with abnormal pupilary widening. Neurosurgery 2002;51(3):628-38.

Cruz J, Minoja G, Okuchi K, Facco E. Successful use of the new high-dose mannitol treatment in patients with Glasgow Coma Scores of 3 and bilateral abnormal pupillary widening: a randomized trial. Journal of Neurosurgery 2004;100(3):376-83.

DeWitt et al. Should Pressors be used to augment cerebral blood flow after Traumatic Brain Injury? Crit Care Med 2000: 28(12): 3933-5

Doberstein, C.E, Curtis. E., et al: Clinical Considerations in the Reduction of Secondary Brain Injury. Neurotrauma / Collective Review. Annals of Emergency Medicine.22(6):993-7. 1993

Doppenberg et al. Clinical Trials in Traumatic Brain Injury: Lessons for the future. J. Neurosurg Anesthesiol. 2004; 16 (1) 87-94

Drummond et al. The effect of the reduction of colloid oncotic pressure, with and without reduction of osmolality, on Post Traumatic Cerebral Edema. Anesthesiology 1998; 88(4): 993-1002

Durward, Q.J., et al: The Influence of Systemic Arterial Pressure and Intracranial Pressure on the Development of a Cerebral Vasogenic Edema. J. Neurosurg. 1983; 59: 803 - 809.

Dutton et al. Traumatic Brain Injury. Current Opin Crit Care 9:503-9. 2003

Eker C, et al: Improved outcome after severe head injury with a new therapy based on principles for brain volume regulation and preserved microcirculation. Crit Care Med 1998; 26:1881-6

Feldman, Z., et al: Effect of Head Evaluation on Intracranial Pressure, Cerebral Perfusion Pressure, and Cerebral Blood Flow in Head Injured Patients. J. Neurosurg, February 1992; 76 No: 2: 207 - 211.

Forster et al. Managing Elevated Intracranial Pressure 2004. Current Opinion in Anaesthesiology 2004; 17: 371-376

Ghajar, J. Traumatic Brain Injury. Lancet, 2000 (356) 923-929

Graham, D. et al. Recent Advances in Neurotrauma. J. Neuropath & Exp Neurology. 2000 (8) 641-651.

Grande et al. Volume targeted therapy of increased intracranial pressure: the Lund concept unifies surgical and non-surgical treatments. Acta Anaesthesiol Scan 2002; 46: 929-941

Guha, A. Management of Traumatic Brain Injury: some current evidence and applications. Postgrad Med. J. 2004:80:650-3

Guidelines for the management of sever Traumatic Brain Injury. Cerebral Perfusion Pressure. Brain Trauma Foundation Guidelines 2003

Guohua et al. Mechanisms of Brain Injury after intra-cerebral heaemorrhage. Neurology 2006 53-63

Huang et al. Clinical Outcome of severe head injury using three different ICP and CPP protocol driven outcomes. J. Clinical Neuroscience 2006: 13: 818-22

Hutchinson et al. Decompressive craniectomy in Traumatic Brain Injury – time for randomised trials? (editorial). Acta Neurochir (wein) 2005; 147: 1-3

Lam A., Anaesthetic Management of Acute Head Injury. 1995 McGraw Hill

Lee et al. Perioperative Head Injury Management in the Multiple Injured Trauma Patient. Int. Anesth. Clinics. 40(3) 2002

Lewelt, W., et al: Autoregulation of Cerebral Blood Flow after Experimental Fluid Percussion Injury of the Brain. J. Neurosurg, 1980; 53: 500 - 511.

Mackenzie, C. Threats and Opportunities in Pre-Hospital Management of Traumatic Brain Injury. Neurosurg Anesthesiol 2004: 16, 70-74

Marik et al. Management of Head Trauma. Chest 2002:122; 699-711

Marioin, D.W., et al: The use of Stable Xenon-enhanced Computed Tomographic Studies of Cerebral Blood Flow to Define Changes in Cerebral Carbon Dioxide Vasoresponsivity caused by a Severe Head Injury. Neurosurgery. 1991; 29: 869 - 873.

Marion, D.W., et al: Acute Regional Cerebral Blood Flow changes caused by Severe Head Injuries. J. Neurosurg, 1991; 74: 40714.

Marion, D.W., et al: The use of Moderate Therapeutic Hypothermia for Patients with Severe Head Injuries: A Preliminary Report. Journal of Neurosurgery. 1993; 79: 354 - 362.

Marshal, R. The Functional relevance of cerebral hemodynamics: why blood flow matters to the injured and recovering brain. Current Opinion in Neurology 2004: 17: 705-9

Menon, D. Procrustes, the Traumatic Penumbra, and Perfusion Pressure Targets in Closed Head Injury (editorial). Anesthesiology 2003; 98: 805-7

Miller, J. Douglas., Head Injury and Brain Ischaemia - Implications for Therapy. Br. J. Anaesth, 1985; 57: 120 - 129.

Miller, J. Douglas., et al: Chapter 15: Raised Intracranial Pressure and its Effect on Brain Function. The Scientific Basis of Clinical Practice, 1985; 266 - 278.

Miller, J.D. Head Injury - Neurological Emergency. Journal of Neurosurgery, Neurology and Psychiatry. 1993; 56: 440 - 447.

Miller, J.D., et al: Severe Intracranial Hypertension. Int Anesthesiol Clin. 1979; 17: 19 - 75.

Miller, J.D., et al: Management of Intracranial Hypertension in Head Injury: Matching Treatment with Cause. Acta Neurochir. 1993; 57: 152 - 159.

Muizelaar, J.P., et al: Adverse Effects of Prolonged Hyperventilation in Patients with Severe Head Injury: A randomized Clinical Trial. J. Neurosurg, 1991; 75: 731.

Muizelaar, J.P., et al: Cerebral Blood Flow and Metabolism in Severely Head Injured Children. Part 1: Relationship with GCS Score, Outcome, ICP, and PVI. J. Neurosurg, 1989; 71: 63 - 71.

Muizelaar, J.P., et al: Cerebral Blood Flow and Metabolism in Severely Head Injured Children. Part 2: Autoregulation. J. Neurosurg, 1989; 71: 72 - 76

Muizelaar, J.P., et al: Cerebral Blood Flow and Metabolism in Severely Head-Injured Children: Relationship with GCS Score, ICP and PVL. J. Neurosurg, 1989; 71: 63.

Muizelaar, J.P., et al: Effect of Mannitol on ICP and CBF and Correlation with Pressure Autoregulation in Severely head Injured Patients. J. Neurosurg, 1984; 61: 700 - 706.

Muizelaar, J.P., et al: Overview of Monitoring of Cerebral Blood Flow and Metabolism after Severe Head Injury. LE JOURNAL CANADIAN DES SCIENCES NEUROLOGIQUES. 1994; 21: S6 - S11.

Muizelaar, J.P., et al: Pial Arteriolar Vessel Diameter and CO2 Reactivity during Prolonged Hyperventilation in the Rabbit. Journal of Neurosurgery. 1988; 69: 923 - 927.

Nordstrom et al. Assessment of the lower limit for cerebral perfusion pressure in severe head injuries by bedside monitoring of regional energy metabolism. Anesthesiology 2003; 98: 809-14

Nortje et al. Traumatic Brain Injury: physiology, mechanisms, and outcome. Current Opinion in Neurology 2005, 17:711-8

Odgen et al. Hyperosmolar agents in Neurosurgical Practice: The Evolving role of hypertonic saline. Neurosurgery 2005; 57: 207-215.

Pasternak et al. Neuroanesthsiology Review 2005 J. Neurosurg. Anesth 2006:18:93-105

Prabhu et al. Anaesthesia for extra-cranial surgery in patients with traumatic brain injury. Anaesth. Critical Care & Pain. 2004. 156-9

Ramani, R. Hypothermia for brain protection and resuscitation. Current Opinion in Anesthiol 2006; 19: 487-91

Robertson, C. Management of cerebral perfusion pressure after Traumatic Brain Injury. Anesthesiology 2001; 95: 1513-17.

Rosner, M.J., et al: Cerebral Perfusion Pressure, Intracranial Pressure, and Head Elevation. Journal of Neurosurgery. 1986; 65: 636 - 641.

Rosner. M.J., et al: Cerebral Perfusion Pressure Management in Head Injury. The Journal of Trauma. August 1990; 30: No: 8; 933 - 941.

Schrader, H., et al: Influence of Blood Pressure on Tolerance to an Intracranial Expanding Mass. Acta Neurol Scand, 1985; 71: 114 - 126.

Schrader, H., et al: Regional Cerebral Blood Flow and CSF pressures during Cushing Response induced by a Supratentorial Expanding Mass. In manuscript , 1984.

Schroder, M.L., et al: Focal ischemia due to traumatic contusions documented by stable xenon-CT and unltrstructural studies. Journal of Neurosurgery. 1995; 82: 966 - 971.

Schroder, M.L., et al: Documented reversal of global ischemia immediately after removal of an acute subdural hematoma. Journal of Neurosurgery. 1994; 80: 324 - 327.

Seelig, J.M., et al: Traumatic Acute Subdural Hematoma: Major Mortality Reduction in Comatose Patients treated under 4 Hours. N. Engl J. Med, 1981; 304: 1511 - 1518.

Shiozaki, T., et al: Effect of mild hypothermia on uncontrollable intracranial hypertension after severe head injury. Journal of Neurosurgery. 1993; 79: 363 - 368.

Siesjo, B.K., Basic Mechanisms of Traumatic Brian Damage. Neurotrauma / Collective Review - Annals of Emergency Medicine, June 1993; 22: 6: 959/11 - 969/21

Skoglund et al. Aspects on Decompressive Craniectomy in Patients with Traumatic Brain Injury. Journal of Neurotrauma 2006; 23(10): 1502-1509.

Steiner et al. The effects of Large-dose Propofol on Cerebrovascular Pressure Autoregulation in Head-Injured Patients. Anesth Analg 2003; 97:572-6

Steiner et al. Predicting the response of intracranial pressure to moderate hyperventilation. Acta Neurochir (wien) 2005; 147: 477-483

Stocchetti et al. Hyperventilation in Head Injury: A Review. Chest 2005; 127: 1812-27

Tecoult et al. Influed of Anesthesia in Experimental Traumatic Brain Injury. J. Neurosurgical Anesth. 2000 (3), 255-261.

Vespa, P. The implications of cerebral ischemia and metabolic dysfunction for treatment strategies in neurointensive care. Current Opinion in Crit Care 2006; 12: 119-23

Vincent et al. Primer on Medical Management of severe brain injury. Crit Car Med 2005 (6). 1392-99.

Wakai et al. Mannitol for acute Traumatic Brain Injury. Cochrane Review. 2006

Wang et al. Neuroprotection targets after Traumatic Brain Injury. Current Opinion in Neurology 2006; 19: 514-9

Zornow, M.H., et al: Acute Cerebral Effects of Isotonic Crystalloid and Colloid Solutions Following Cryogenic Brain Injury in the Rabbit. Anesthesiology. 1988; 69: 180 - 184.

Zornow, M.H., et al: Effect of a Hypertonic Lactated Ringer's Solution on Intracranial Pressure and Cerebral Water Content in a Model of Traumatic Brain Injury. J. Trauma. 1989; 29: 484 - 488.