# MANAGEMENT OF A HEAD INJURY - Case Report

Dr. Frank J.M. Walters FRCA, Consultant Anaesthetist, Frenchay Hospital, Bristol BS16 1LE, UK, Frank_Walters@Compuserve.com and Dr. Ray Towey FRCA, Consultant Anaesthetist, Bugando Medical Centre, Mwanza, Tanzania, emach@africaonline.co.tz

This is a report of a patient who has suffered a head injury. The purpose is to illustrate the practical application of the basic physiological and pharmacological principles explained before (Neurophysiology-intracranial pressure and cerebral blood flow, Update in Anaesthesia 1998;8:18-23 and Neuropharmacology-Intracranial pressure and cerebral blood flow. Update in Anaesthesia 1998;9:29-37). The problem is presented with the management and a range of anaesthetic techniques.

## The Case

Cycling to work in the morning, a fit 30 year old man has an accident which causes severe damage to his head. Initially he is conscious but confused, and is taken to the local Accident Department. When he is admitted it is found that he has become unconscious.

## Initial Management

An initial assessment is performed urgently, in the sequence described below.

**A** Airway control including cervical spine immobilisation with a stiff collar.

**B** Breathing

**C** Circulation

**D** Dysfunction or Disability

**E** External Examination

His **airway** is clear. He is **breathing** adequately. His blood pressure is 180/90mmHg and he has a regular pulse with a rate of 55 bpm. There was no report of blood loss at the scene. He is warm and well perfused. Thus his **circulation** is adequate.

**Neurological dysfunction** is assessed by looking at **conscious level**, **pupils** and **posture**. Conscious state is assessed using the **G**lasgow **C**oma **S**core (**GCS** score Table 1) or the **AVPU** system. Glasgow Coma score range is 3-15, if it is less than 8, the patient has serious damage with raised intracranial pressure (ICP) more than 20 mmHg (normal 5-13 mmHg).

The **AVPU** is simple to carry out and offers a rapid method of assessment.

| | |
|---|---|
| **A**lert | Yes/No |
| **V**erbal - response to verbal command | Yes/No |
| **P**ain - response to painful stimulus | Yes/No |
| **U**nresponsive | Yes/No |

Patients who are not alert and are not responding to command (P or worse) are equivalent to a GCS of around 8 which indicates a severe injury.

On examination of the pupils the right pupil is found to be fixed and dilated, the left pupil is small and reacting. He is unresponsive to pain (GCS less 8, AVPU less than P). This is a neurological emergency where delay may result in a fatal outcome or major disability. A rapid secondary survey is carried out to exclude other life threatening injuries.

Although the risk of neck injury is low, it cannot be excluded and therefore the neck is kept stabilised with a semi-rigid collar and sand bags or blocks joined with tape with straps across the forehead (figure 1). An IV infusion is started with normal saline (0.9%).

**Table 1: The Glasgow Coma Scale**

| **Glasgow Coma Scale for Assessment of Level of Consciousness** | |
|---|---|
| *Eye Opening:* | |
| Spontaneous | 4 |
| To speech (not necessarily a request for eye opening) | 3 |
| To pain (stimulus should not be applied to face) | 2 |
| None | 1 |
| *Best Motor Response:* | |
| Obeys commands | 6 |
| Localise (purposeful movement towards the stimulus) | 5 |
| Normal flexion (withdraws from painful stimulus) | 4 |
| Abnormal flexion (decorticate posture) | 3 |
| Extension (decerebrate posture) | 2 |
| No movement | 1 |
| *Verbal Response:* | |
| Oriented (knows name, age) | 5 |
| Confused (still answers questions) | 4 |
| Inappropriate words (recognisable words produced) | 3 |
| Incomprehensible sounds (grunts/groans, no actual words) | 2 |
| None | 1 |
| **TOTAL SCORE** | **/15** |

## COMMENT

*As soon as the patient is admitted to hospital the basic ABCDE sequence described above is rapidly carried out to detect any problems such as airway obstruction or respiratory arrest which will rapidly cause death unless treated. With head injuries, respiratory obstruction will cause hypoxia and raised carbon dioxide and will lead to increased intracranial pressure causing severe secondary damage. An adequate blood pressure is vital in a patient with raised intracranial pressure.*

*The patient has suffered a primary head injury from trauma. The signs indicate a rapidly rising intracranial pressure, with coning of the temporal lobe probably due, in this case to an extradural haematoma. This is a rapidly fatal condition if it is not treated urgently. When treated quickly, a good recovery may result. It is essential that further brain damage from ischaemia (due to low blood pressure and cerebral swelling) resulting from factors such as hypoxia, high carbon dioxide levels and venous congestion does not occur.* ***Hypotension and hypoxia will increase mortality of a patient with a severe head injury by 70-80%.***

### Anaesthetic Management

Whenever possible the patient should be reviewed by an anaesthetist in the receiving room or accident department. As the patient has low AVPU and GCS scores, he needs to be intubated and ventilated to ensure a clear airway, full oxygenation and low normal carbon dioxide levels before going to the CT scanner. Since there is still concern regarding a possible neck injury, further movement of the neck during intubation could cause injury to the cervical cord. Therefore intubation is carried out with the head in the neutral position and **manual in-line** traction to prevent neck movement (figure 2). To perform this manoeuvre, an assistant grasps the mastoid processes and the front part of the collar is removed to allow adequate mouth opening. Do not apply excessive traction as this can cause further damage to the cervical spine.

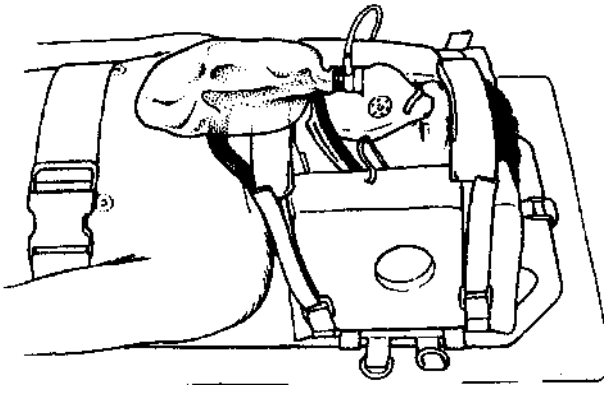

*Figure 1: Cervical spine immobilisation with a long spine board, rigid collar, lateral blocks and straps.*

The mouth is clear, but as he may have eaten breakfast recently, a rapid sequence induction is necessary. He is pre-oxygenated and given an intravenous narcotic fentanyl 150 mcg, (pethidine 50 mg or morphine 5 mg would be reasonable alternatives) followed by a slightly reduced dose of thiopentone 150-200 mg to ensure anaesthesia,

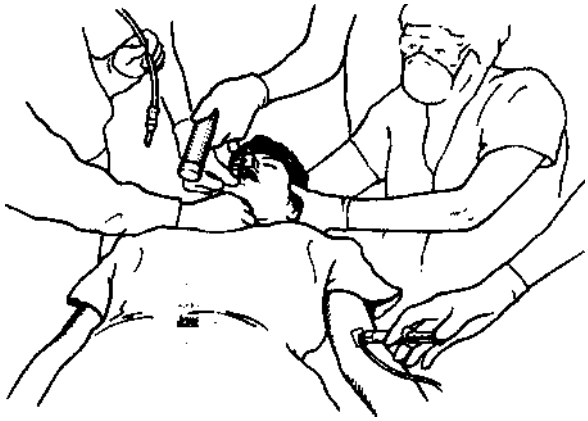

*Figure 2: Rapid sequence induction of anaesthesia and manual in-line cervical traction in an acute trauma patient*

without causing hypotension. Many places today are now using a reduced dose of propofol 90 - 100 mg, for induction and continuing anaesthesia with a propofol infusion. Cricoid pressure is applied, he is paralysed with 100mg of suxamethonium, and intubated once fasciculation has stopped.

**It is important to maintain intermittent positive pressure ventilation for neurosurgical patients to produce a moderately low normal arterial $CO_2$ ($PaCO_2$ 35 mmHg - 4.7 kPa) which will help to reduce cerebral swelling and hence intracranial pressure.**

After tying in the endotracheal tube the blood pressure is measured again. It has fallen to 80/55 mmHg. 500mls of normal saline (0.9%) is rapidly given, and a 6mg dose of ephedrine is administered IV which restores the blood pressure to 180/90 mmHg.

**IV fluid therapy**

When anaesthesia is started, the cardiovascular system is depressed, particularly the ability to compensate for a reduction in blood volume (Cardiovascular Physiology Update in Anaesthesia 1999;10:2-8). Therefore part of the treatment of a fall in blood pressure is a rapid infusion of fluid intravenously.

**Anaesthesia is now maintained** using a further dose of fentanyl 150 mcg or iv morphine 10mg slowly, vecuronium 10mg and the patient ventilated with oxygen enriched air. He is monitored with a pulse oximeter, an ECG and BP. An infusion of 150mls 20% mannitol (0.25 - 0.5 g/kg) is commenced, followed by a litre of 0.9% saline, and a urinary catheter inserted.

He is transported to the CT scanner which shows a large extradural haematoma (figure 3) and therefore is immediately taken to the operating theatre for craniotomy.

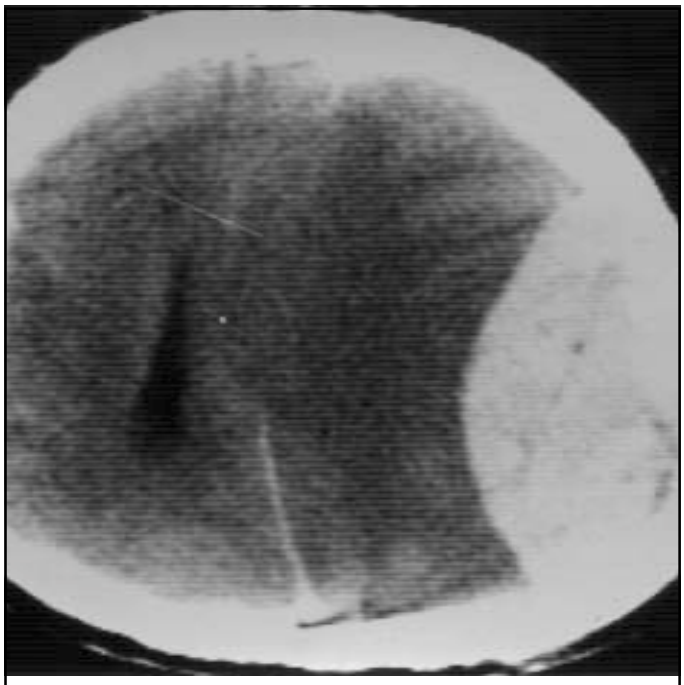

*Figure 3: CT scan of a patient with an acute extradural haematoma*

**Teaching Point**

*Hypotension following induction*

Reasons for concern - hypotension will reduce perfusion pressure in the brain. This has two adverse effects:

1 Blood flow through the brain will fall dramatically, reducing oxygenation of the brain and cause ischaemia. The brain tolerates this badly and will suffer major neurological damage (stroke, paralysis, death).

2 The arteries in the brain will sense a reduction in flow and will try to compensate (**autoregulation**). They will dilate in an attempt to reduce their resistance and thus increase flow. Dilatation will increase their volume and cause a further increase in brain volume and intracranial pressure, making the situation worse.

*Saline vs Colloid vs Dextrose*

The brain is surrounded by a membrane separating it from the vascular space - the **blood-brain barrier.** This membrane will only allow water to pass through it. **Therefore only fluid with the same concentration of sodium as plasma should be given intravenously.** Otherwise, the plasma will become more dilute and water will pass from it into the brain, making the brain swell, and thus increase pressure further.

Normal Saline (0.9%) has a similar concentration of sodium and therefore is the fluid of choice for the brain. Colloid can be given if required to treat hypovolaemia due by major blood loss.

When Dextrose solutions in water (5% Dextrose, Dextrose 4%-Saline 0.18%) are given, the dextrose is metabolised leaving just the water or a very dilute saline solution. This "dilutes" the blood, reducing the concentration of sodium in the plasma. The water then passes into the brain where the concentration of sodium is higher. The brain then swells, and intracranial pressure will rise.

**Vasopressors** As explained, the blood pressure must be raised quickly. Therefore a small dose of cardiovascular stimulant drug can also be given intravenously to raise the blood pressure while the fluid is being run in. Suitable drugs include ephedrine, 3-6 mg, methoxamine 1-2 mg, or adrenaline 25-100 mcg.

**Teaching point**

Different anaesthetic options during transfer to theatre and in the CT scanner

The aim of the anaesthetist is the maintenance of the patient in a physiologically stable state so that no further harm to the damaged brain occurs. This means full oxygenation with slight hypocapnia and without coughing or straining (avoiding cerebral venous congestion). As an alternative to the technique described, many centres now use a propofol infusion, 2-6 mg/kg/h, by syringe pump which can be started in the accident department and continued into theatre. A thiopentone infusion can be used, but is much more difficult to manage because thiopentone is not rapidly metabolised. Therefore it accumulates and can take days to reverse.

**Importance of monitoring**

It is easy for the brain to be damaged during this period. Unnoticed hypotension, hypoxia or coughing, which can occur unexpectedly and suddenly, can cause irreversible damage. Therefore close clinical monitoring of the patient is crucial.

**CT scanner**

The CT scanner is used to confirm the diagnosis and to guide the surgeon to where the bone flap should be raised. A short emergency scan is carried out causing the minimum of delay to the start of surgery (figure 3). CT scanning is not available in many centres, and therefore the patient would be transferred directly to theatre by the anaesthetist for initial burr holes to be carried out in the temporal region on the side of the dilated pupil.

## Theatre

There is a wide range of anaesthetic drugs and hence techniques available. Anaesthesia may be maintained with an inhalational agent - isoflurane would be the first choice and halothane the second choice. If only ether is available then use ether. If available, increments of morphine, pethidine or preferably fentanyl should be used as narcotics reduce the risk of coughing and the concentration of inhalational agent required. Neuromuscular blockade is maintained with non-depolarising muscle relaxants, to avoid coughing and straining with the minimum concentration of the inhalational agent required. However, if no long acting muscle relaxant is available then continue with intermittent positive pressure ventilation using the inhalational agent to suppress normal ventilation. In some units where propofol is available, anaesthesia can be maintained with a propofol infusion (2-6 mg/kg/hour), oxygen enriched air, and small increments of narcotics. The infusion rate is adjusted to ensure that the blood pressure does not fall.

The circulation is monitored by observing the peripheral circulation, pulse rate, blood pressure and urine output. Blood pressure should be monitored either invasively or frequently with a cuff. Continuous measurement of the patient's blood pressure is very helpful as it allows blood pressure changes to be treated accurately and efficiently. Do not delay surgery to insert an arterial line. However there is usually time during preparation of the patient to attempt radial artery cannulation. If this proves difficult then a cannula can be put into the femoral artery to provide monitoring for the duration of surgery.

**Teaching point**

***Hypertension in theatre***

A systolic blood pressure of 180 mmHg, may appear to be high for a 30 year old man, but it is vital until the clot has been removed. This is because the body has raised the blood pressure to overcome the high intracranial pressure. Therefore do not allow the blood pressure to fall below this level.

In contrast, if the blood pressure rises to more than 200 mmHg systolic, this indicates insufficient depth of anaesthesia. Treat this with a small increase in concentration of inhalational agent or propofol infusion rate and a further dose of narcotic until the mean arterial blood pressure falls to 140 mmHg (corresponding approximately to a systolic arterial pressure 180 mmHg).

***Hypotension in theatre***

Note that high concentrations of inhalational agents cause cerebral vasodilatation, increasing cerebral blood volume and thus cerebral swelling. This worsens the situation by causing a further rise in intracranial pressure. In addition higher concentrations may cause a fall in blood pressure. The combination of high ICP and low BP would severely reduce cerebral perfusion and should be treated quickly with intravenous fluids, vasopressors and a reduction in the concentration of volatile agent.

**Operation**

At operation, a large clot is found under high pressure. Once released, the blood pressure falls to normal levels. The bleeding point is identified and secured. The brain which was compressed, is pulsating with each heart beat and respiration. If an Intensive Care (ICU) bed is available and it is decided to ventilate the patient for a period postoperatively, an ICP monitor is inserted. As described below, a catheter is inserted subdurally and connected to an arterial transducer.

**Postoperative care**

The patient is now taken to ICU to allow the anaesthetic drugs to wear off with an intracranial pressure monitor in situ. After some hours he is opening his eyes, coughing on the ET tube and breathing well. His ICP is 12 mmHg and he is allowed to wake up and is extubated.

**An inexpensive ICP monitor**

If an arterial pressure transducer is available this can be done simply with a neonatal umbilical catheter. The catheter is inserted either subdurally or intraventricularly and filled with saline by the surgeon. A 1-2 cm subcutaneous tunnel is formed to reduce the risk of the catheter being pulled out. Care must be taken to avoid kinking the catheter. Under aseptic conditions it is connected to a transducer without the usual pressure heparin flush system. A dampened looking pressure trace is seen with pressure fluctuations with each cardiac cycle. It will rise with coughing and will be flat if it is blocked or ICP is very high. It does not often block, but if this happens only the surgical team should attempt to unblock it. Intraventricular catheters are less likely to block but have greater risk of infection, their use being limited to 5 days

**Conclusion**

1 The initial assessment and resuscitation is vital and must be carried out: a scheme is described.

2 A method of anaesthesia is described based on simple physiological and pharmacological principles which, when used, will reduce the risk of added damage.

**An unconscious patient with an extradural haematoma will die or be permanently severely damaged unless treated quickly and correctly. Urgent surgical decompression is required. It ALONE is not enough. Attention to basic simple clinical details will prevent additional irreversible damage occurring before the intracranial pressure can be reduced by releasing the haematoma.**

**Teaching Point**

At the end of surgery the anaesthetist should achieve the following aims

1 Prevent further damage from physiological factors which will cause brain swelling.

2 Observe the patient to detect any deterioration.

3 Provide adequate analgesia.

Deterioration postoperatively may occur from brain swelling or accumulation of a further haematoma. The best method of detecting any deterioration in neurological status postoperatively is observation of the conscious patient, looking for a change in conscious level and new or changed neurological signs. Therefore when a simple haematoma without bruising or contusion to the brain is removed early from an otherwise fit patient who has no other trauma, as in this case, it is better to wake the patient up immediately after surgery. Anaesthesia is stopped and the patient extubated. In contrast, when there is significant brain contusion or other systems are damaged by the accident, waking and extubation are delayed if an ICU bed is available.

**ICU available**

The patient is taken to the ITU where ventilation and sedation are continued. BP, pulse and ICP are monitored. When the ICP remains normal, the patient is allowed to wake up and be extubated.

**ICU not available**

Anaesthesia is stopped in theatre and the patient allowed to wake up. Neuromuscular block is reversed and spontaneous respiration allowed to start. Extubation should be carried out when the endotracheal tube is seen to be irritating the trachea. Initially the patient lies in the recovery (lateral) position until airway reflexes have returned. The patient is then sat up at about 30° to reduce cerebral venous congestion. The patient should be given the best nursing care that the hospital can provide with Glasgow Coma Score or AVPU recordings started, recorded and repeated at 15 minute intervals. Any deterioration in conscious level or appearance of new neurological signs is reported to the surgical team immediately.