THE SEVERE HEAD INJURY: METHODS OF ASSESSMENT
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Cecil Armstrong Calvert has been regarded as one of the most outstanding men the Belfast Medical School has produced, graduating with First Class Honours in 1922. He was a clinician and surgeon of international reputation.

He was the founder and Director of the Department of Neurosurgery in this, the Royal Victoria Hospital, until his untimely death in 1956, and he tirelessly devoted his time and energy to the treatment of his patients and the training of his staff.

During the war he established, with Sir Hugh Cairns, a Head Injury Centre at Oxford—the first of its kind in Britain.

His most significant contributions to medical knowledge were in the field of head injuries: he developed methods of treatment for compound fractures of the base of the skull which are standard treatment to-day. This present research would have been of special interest to him.

INTRODUCTION

TRAUMA is the epidemic of this age and takes a heavy toll where tuberculosis and plague were once the giants. It, too, carries away the young and those in the prime of life, for well over half the victims are less than 30 years of age (Gögler, 1965).

Head injury is responsible for 70 per cent of the deaths which follow trauma, but the unpredictability of the condition makes the prognosis uncertain and leads to doubts about the benefit of different treatments. In coronary artery disease the value of objective measurements and continuous monitoring of cardiac function have been clearly demonstrated (Adjej et al, 1971). In head injury care, where we deal chiefly with young patients, it is now appreciated that monitoring of cerebral function should accompany clinical assessment. The investigations to be described were an attempt to enlarge our understanding of the natural history of this condition and to improve the evaluation of treatment.

HISTORY

The study of head injuries is one of the earliest projects undertaken in medical history dating from 3000 B.C. In excavations in Peru, skulls have been found with evidence of healed trepanation beside fracture lines—showing that the procedure was carried out during life and with a degree of success. Indeed, of the 400 or so skulls found, some 60 per cent showed signs of healing. (Tello, 1913). The earliest written accounts of head injuries are in the Edwin Smith papyrus of 1400 B.C. At that time doctors plainly had decided which patients had a good prognosis.
In this, the first text book of medicine, 19 patients with head injuries are described, and clear instructions on treatment are given. Patients fell into three categories:

(a) Those who would recover without treatment;
(b) Those who might recover and therefore could be treated;
(c) Those who were not to be treated because of a hopeless prognosis.

The penalty for not following this advice ranged from a fine of 10 shekels of silver to amputation of the right hand (Horax, 1952). This advice is still applicable, but the determination of these categories — those who will improve and those who will die — has exercised medical minds for centuries. Hippocrates said: “No head injury was too trivial that it should be ignored, or too severe that it be despaired of”. But, 2,400 years later, Penfield had little further to add to this broad generalization saying: “It must be decided initially whether or not the prognosis is hopeless, but the criteria which may be said to mark a case as ‘hopeless’ are few” (Penfield, 1941).

In the last few decades the electrical activity of the brain, as detected by EEG, has been used in the study of severe head injuries. Changes accompany improvement in clinical condition; an absolutely flat record usually indicates brain death; but in this field the test’s value has been limited. In recent years other parameters such as intracranial pressure, cerebral blood flow and biochemical changes in the cerebrospinal fluid have been studied. They will be described in a later section.

Pathophysiology

As has already been suggested, the severe head injury is a dangerous condition with a mortality of between 50-70 per cent. Surprisingly few patients who die from head injury have large intracranial haematomata. Naked-eye autopsy examination of the brain usually reveals very little abnormality. Microscopically, however, Strich (1956) and Nevin (1967), identified diffuse areas of damage in the brain. They describe groups of damaged axons in the white matter and areas with neuronal death surrounded by zones of oedematous cells. Normal brain tissue lies between the damaged areas. The changes resulting from the initial injury are serious enough but, as the skull is a closed compartment, a vicious circle of rising pressure becomes established. Trauma results in oedema which raises intracranial pressure, oedema reduces the blood and oxygen supply to sick cells, the resulting ischaemia promotes further oedema. As cerebral oedema is an undesirable consequence of the head injury, the object of treatment is a reduction of intracranial pressure and improvement in the blood supply to the tissues most requiring it. This basic pathophysiology leads us to investigations in patients with severe head injury.

1. Intracranial pressure monitoring. Control of cerebral oedema reduces intracranial pressure. A low pressure therefore becomes the aim of treatment.

2. Biochemical studies of cerebrospinal fluid (C.S.F). CSF is the extracellular fluid of the brain cells. Abnormal levels of metabolites indicate the extent of cellular damage and have a bearing on prognosis.

3. Cerebral blood flow. Cerebral oedema reduces the blood flow; changes in flow provide an indication of the severity of cerebral oedema, and to some extent show the effect of treatment.
METHODS

As intracranial pressure, nutrition of brain cells and cerebral blood flow are so closely related, it seemed reasonable to measure all three simultaneously in severe head injuries.

1. Intracranial Pressure (ICP)

Isolated pressure measurement by lumbar puncture was used initially but was unreliable (Quinke, 1891). A small, intracranial pressure device has now been developed which allows continuous pressure recording. The model used is a silicon beam planar resistor type of strain gauge, (Ferranti & Akers). The device is placed in the subdural space through a burr hole. It is quite small and does not damage the cerebral cortex. The pressure transducer is connected to a battery-operated amplifier and a slow running chart recorder. The transducer can be left inside the head for 10 days and is easily removed by cutting out the retaining stitch. This equipment has recorded intracranial pressures as high as 100 mgHg which is 10 times normal, (or the equivalent of five lumbar puncture manometers placed on top of each other). The rate of change in intracranial pressure is perhaps, more important than the actual levels. Breath-holding or coughing can raise the pressure to 25-30 mmHg, (that is, two to five times normal), and similar changes can accompany induction of anaesthesia and endotracheal intubation. The effects of different forms of treatment, to be described later, can be assessed by these pressure changes.

2. CSF Biochemistry

The vicious circle of oedema and raised intracranial pressure increases the damage to injured brain cells. Normal cells require glucose and oxygen which are metabolized to carbon dioxide and water. Injured and ischaemic brain cells lack oxygen; their deranged metabolism leads to the accumulation of lactic acid. This metabolite accumulates in the CSF in amounts which depend to some extent on the number of injured cells. A correlation between CSF lactic acid level and prognosis in head injuries has been suggested by Kurze et al, (1966). But the actual levels and the length of time the levels were raised has not been evaluated.

In 38 severe head injuries, serial samples of CSF were obtained by lumbar puncture. If intracranial haematoma was suspected, lumbar puncture followed operation. The CSF sample obtained was divided into three parts: one for lactic and pyruvic acid estimation, the second for Astrup estimation, and the last for protein and red cell content. The pressure was also measured and compared with the intracranial pressure values. Samples were obtained daily or, in some cases, twice daily. The values obtained in 18 of these are shown in Fig. 1. The values of lactate rapidly fall after the injury, in fact, the higher the level, the more rapid the decline. Normal values were 10-15 mg. per cent in our studies, while some patients had values up to 12 times normal levels. In all the samples studied, (with the exception of those with gunshot wounds), no patient has survived with a lactate level above 55 mg. per cent and no patient died of his head injury with a lactate value of less than 25 mg. per cent. So from this study it is felt that the levels of lactate in the CSF provide a valuable prognostic test (Crockard & Taylor, 1971).

The graph in Fig. 1 shows the importance of obtaining the CSF sample within 24 hours of injury. Levels between 30 mg. per cent and 55 mg. per cent are of
FIG. 1. Serial CSF lactate values following severe head injury, showing the rapid decline in concentration over 48 hours; the results stress the importance of an early sample in prognosis.
uncertain prognostic value. When a patient with lactate levels in this range dies, he has usually presented with decerebrate rigidity, respiratory difficulty and hyperpyrexia. The relatively low lactate level suggests brain damage limited to a small, but vital, area.

Taking the highest results obtained from all 38 cases, there is a marked difference in the CSF of those who died and the survivors, (Table 1). The lactate level in those who survived was significantly lower, and the lactate/pyruvate ratio was half that obtained from fatal cases. The CSF was also much more acid in those who died; in survivors the CSF pH remained about normal. The increased CSF acidity may explain changes in respiration and cerebral blood flow, as pH is important in the regulation of both. The rapid fall in CSF lactate levels is not unexpected; as the damaged cells which produce lactate die or recover, the metabolite enters the blood stream where it is metabolized. But, if the brain damage by increasing cerebral oedema continues, further anaerobic glycolysis produces more lactate. In three patients with massive intracranial haematoma, the lactate levels rose to over 55 mg. per cent just before death.

Serial study of CSF lactate levels thus provides useful prognostic data. Abnormal metabolism caused by increasing cerebral oedema can be detected; a level over 55 mg. per cent is usually incompatible with survival.

|          | Lactate | Pyruvate | L/P | pH  | Bicarbonate |
|----------|---------|----------|-----|-----|-------------|
| Alive    | 2.98    | 0.16     | 33.8| 7.31| 21.36       |
|          | (26.8mg%) | (1.42mg%) |     |     |             |
| Dead     | 6.34    | 0.18     | 61.1| 7.17| 16.5        |
|          | (57.1mg%) | (1.61mg%) |     |     |             |

3. Cerebral blood flow

The techniques for measuring cerebral blood flow have grown more and more sophisticated, and now the classical method of Lassen & Ingvar (1961) involves an intracarotid injection of Xenon, monitored by 32 computer-linked collimators. Because of the difficulty, the potential hazards, and the limitations of the procedure, in Belfast we have concentrated on the less exact but simple, atraumatic and easily repeatable technique of Radiocirculography (RCG). The method was developed by Oldendorf in 1962 and pioneered by Taylor (1966). Basically the method is as follows:

Into an antecubital vein a very small quantity of a radioisotope (Technetium 99) is injected below an inflated cuff. This is rapidly released and the passage of the radioactive bolus, or "slug", is monitored over the praecordium and then the head.
The radiation is detected by collimators over the praecordium and two situated to look at the cerebral hemispheres individually. As the isotope passes through the heart and lungs, the radioactivity is measured and expressed graphically (Fig. 2). As it passes through the head another curve is obtained. The time taken for the

**Radiocirculography**

500 μCi Tc in 1 - 2 ml saline
Antecubital Vein
Rapid Cuff Release

![Diagram of radiocirculography](image)

**Fig. 2.** Radiocirculography (RCG). The head is placed on the angled collimators prior to injection of the Technetium (Tc 99m). As the radioisotope “slug” passes through the heart and head, a primary curve is obtained. The length of this curve provides a Transit Time (TT) for circulation; its derivative a Mean Circulation Time (MCT).

The isotope to pass through the head is known as the Transit Time (TT). A differentiated curve obtained from this primary curve provides an average circulation time through the brain, or Mean Circulation Time (MCT). In simple terms, the better the blood flow, the shorter will be the Transit Time and the higher will be the amplitude of the curve. Conversely, a low amplitude and a long TT denote a poor cerebral circulation. Obviously cardiovascular disease will influence cerebral circulation and the praecordial monitor allows a correction factor to be used.

As the skull is a rigid, closed box, very high intracranial pressure adversely affects blood flow. After reduction of the pressure, blood flow improves. This, in theory, is what happens when an extradural haematoma compresses the brain. Several patients have been studied serially before and after operation; the predicted improvement in blood flow followed removal of the haematoma (Fig. 3). With the two collimators scanning each cerebral hemisphere separately, similar changes have been noted in hemispheric blood flow after removal of a subdural or intracerebral haematoma and aspiration of abscess or cystic tumour.
EXTRADURAL HAEMATOMA

![Diagram of extradural haematoma blood flow changes over time](attachment://image.png)

**Fig. 3.**
Blood flow changes following removal of a large extradural haematoma showing its rapid improvement.

The RCG becomes a useful guide to the patient’s progress. If, for example, the flow curves obtained from each cerebral hemisphere do not improve after treatment, we conclude that either the ICP has not been sufficiently reduced, or that there is extensive underlying brain damage previously unrecognized.

As with the other tests described, signs associated with a grave prognosis have been found with RCG. Basically there are two types of curve which bode ill: the first is a flattening of the curve until it is difficult to see if any isotope enters the head (Fig. 4); presumably if the pressure is too high inside the head the blood flow will cease. The second characteristic curve which preceded death in six cases is more difficult to explain. After a flattening of the curve, the amplitude suddenly increased and the TT decreased to become identical in shape with the cardiac curve.
EFFECT OF CARBON DIOXIDE ON HEAD INJURIES

Reference has already been made to the vicious circle of trauma-cerebral oedema-raised-intracranial-pressure. The cerebral oedema results from direct damage to the cerebral capillaries and subsequent swelling of the ischaemic cells. The normal nutrition of cells is interrupted and acid metabolites such as lactic acid will accumulate. This leads to the dilatation of small vessels in the damaged area. A major factor in the control of cerebral blood flow is the amount of carbon dioxide in the blood—an increase acts directly on normal cerebral vessels and they dilate. But what happens in a pocket of damaged brain tissue? Blood vessels in this area are already maximally dilated and unresponsive as a result of trauma and the accumulation of acid metabolites. If the carbon dioxide in blood rises, only the normal vessels dilate; the intracranial volume and hence pressure will rise, causing further ischaemia in the damaged area. To make matters worse, the dilated vessels in normal brain shunt the blood from the ischaemic areas, a process termed the intracerebral steal phenomenon (Lassen, 1966; Symon, 1969).

That these changes occur in practice and not just in theory will be demonstrated by the following example (Fig. 5), which shows a decrease in cerebral blood flow due to increased $\text{CO}_2$ in the blood. In the normal brain, blood flow would speed up with an increasing $\text{CO}_2$ level, but in damaged brain there is a paradoxical response. As can be seen, the blood flow has deteriorated with the addition of $\text{CO}_2$ to the gas mixture breathed by the patient. From a theoretical point of view it can be seen that anything which increased blood levels in head injuries will produce further damage. The commonest way for this to occur is by lack of attention to the airway in injured patients, allowing the tongue to fall back into the pharynx with the patient lying on his back or by failure to establish an adequate airway.

TREATMENT BY HYPERVENTILATION

If $\text{CO}_2$ accumulation is bad for the patient with brain injury, what would be the result of deliberately removing $\text{CO}_2$ by hyperventilation? If the $\text{CO}_2$ level falls, vessels which can react will constrict and thus reduce the intracranial pressure. The shunt (or steal) reverses and the nutrition of the damaged area improves. This, then is the theoretical basis for a new method of treating the severe head injury—namely, hyperventilation by a mechanical respirator.
Here is the effect of deliberately lowering the CO$_2$ levels by mechanical "over-breathing", i.e., intermittent positive pressure ventilation, (IPPV). The patient (RC), was involved in a brawl and sustained a head injury with generalised brain swelling (Fig. 6). The top curve was obtained before treatment began. The high level of CO$_2$, (57 mmHg) can be seen, (normal range 35-39 mmHg). The patient was paralyzed with a curare-like drug, intubated and mechanically ventilated to reduce the CO$_2$ levels. The effect on the cerebral circulation was dramatic and the improvement persisted after assisted ventilation had ceased. This illustrates the point made earlier, namely, the importance of breaking the vicious circle of oedema-raised intracranial pressure-increasing damage. A total of 26 patients have been hyper-ventilated, keeping the pCO$_2$ between 25-30 mmHg. After ventilation for an initial period of 48 hours, the curare drugs are reversed to allow clinical assessment. If the clinical condition is improved and the blood flow and intracranial pressure remain at satisfactory levels, hyperventilation is not resumed. Longer periods of hyperventilation — sometimes as long as two weeks — have been tried; the best results, however, are evident in a few days. The aim of treatment is to improve the quality of life in survivors, not merely the prolongation of vegetative life. The prognostic tests already listed (intracranial pressure measurement, CSF lactate levels and cerebral blood flow), provide helpful guidance in carrying out this treatment.

In successful cases, the blood flow improved, shown by a decreased TT, and the intracranial pressure fell. In those who did not respond to treatment, the pressure...
Fig. 6. Improvement of blood flow with hyperventilation in a successful case. There was a corresponding fall in intracranial pressure.

rose above 70mmHg and the RCG showed no shortening of TT. In fact, in some cases it was shown that the cerebral vasculature no longer reacted to blood CO$_2$ levels, presumably because of the extent of damage incurred at the time of injury and immediately following it. If there was no reaction by the vessels to alterations in CO$_2$ levels, then, on theoretical grounds, it was predictable that hyperventilation would not be successful.

Of course there are hazards with mechanical ventilation of patients with serious head injuries. As they are paralyzed with curare-like drugs it is difficult to detect the development of intracranial haematoma; before and during treatment all possible measures are taken to exclude such a lesion. If the ventilation is excessive (for instance, if the pCO$_2$ falls below 20 mmHg), normal brain cells are rendered ischaemic (Zwetnow, 1968). During treatment it is important to make frequent measurements of blood gases and CSF chemistry.
CONCLUSION

This research investigation has not reached any new conclusions, rather it has re-stated the vital role of objective measurements in a dynamic condition such as head injury. It is by this method only that the treatment of serious head injuries can advance. Factors associated with a poor prognosis are given in Table 2 and, using these measurements with clinical judgment, it is possible that the quality of life of survivors can be improved and the prolongation of vegetative life avoided.

| Table II |
|-----------------------------------------------|
| **Results associated with a poor prognosis** |
| ▲ Intracranial pressure (unresponsive to treatment) |
| ▲ C.S.F. lactate 50 mg% |
| RCG — Long Transit Time (unresponsive to treatment) |
| — Very Short Transit Time (vasoparalysis) |

Hyperventilation is a new approach to the problem of intracranial hypertension resulting from cerebral oedema and, while it is too early to be dogmatic, initial results have been encouraging.

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