SALVAGING "HOPELESS" POST HEAD INJURY PATIENTS

A.E. Delikan & M. Namazie

INTRODUCTION:

THE MANAGEMENT of head injuries has always been a matter of serious concern for those involved in the care of such critically ill patients. The neurosurgeon, the anaesthesiologist and sometimes the neurologist, form a closely-knit team in the management of such patients who are ideally cared for in intensive care or critical care units.

With the increase in motor traffic and the number of motorised vehicles on Malaysia roads we are seeing an increase in the number of head injured patients. The number of head injured patients has risen to alarming figures in the more industrialized countries. In a study by Jennet (1975) it was found that over 33% of admissions to accident units were due to head injuries. Very often these patients belong to younger age groups or to the most useful working ages. In 1973 in England and Wales, 16% of all fatalities in males between the ages of 15 and 40 were attributable to head injuries (Registrar General, 1975). It is indeed a tragic loss of human life in their prime. It is, therefore, not surprising that more attention is being paid to the care of these patients both in the acute phase and in the prolonged care.

Early teachings on management of head injuries emphasised the fact that when a brain lesion was not amenable to surgical treatment, especially when there were severe clinical symptoms, the prognosis was poor. Further management was mostly of routine nursing care and sedation when indicated. Care of the airway and respiration were haphazard due to lack of suitable trained personnel and the absence of intensive care units in the country. Even with the advent of intensive care units in Malaysia, initiation of laborious and personnel-requiring series of treatment are withheld in patients in whom the outlook is considered "hopeless". It is, therefore, not surprising that the mortality or a permanent morbidity is high in such patients.

In this paper we report 17 cases of severely head injured patients in the younger age group age who were managed in the multi-disciplinary Intensive Care Unit of the University Hospital, Kuala Lumpur, over a 2 year period.

MATERIAL AND METHOD:

17 young patients (age range: 3 - 14 years) with head injury (sub-dural haematoma and intra-cerebral injury or brain stem injury with generalised cerebral edema) and showing signs of decerebration had exploratory craniotomy with evacuation of any haematoma found and haemostasis.

Post-operatively, these patients were managed in the Intensive Care Unit and the regime of management consisted of mechanical intermittent positive pressure ventilation (IPPV), regular administration of Dexamethasone intravenously, mild hypothermia to maintain temperature at 34 $\pm$ 2°C and regular timed administration of Diazepam intravenously. All patients were paralysed with a non-depolarising muscle relaxant. Nasotracheal intubation with PVC endotracheal tube was preferred in the first two weeks of management. The regime was followed for a period of 4 - 5 days.

RESULTS:

11 (64.7%) of the 17 patients died; 4 of them died during the regime and the other 7 succumbed later. The latter patients had prolonged unconsciousness and were tracheostomised to assist in the spontaneous respiration and for tracheo-bronchial clearance of secretions.

6 patients (35.3%) regained consciousness within 1 - 14 days after cessation of the regime and their recovery is graded as shown in Table 1. 3 were of category A, while there were one each of the remaining categories B, C and D.

Neurological deficits observed were altered personality, limb weakness, unsteady gait and speech defects.

A.E. Delikan, M.B.B.S. (S'pore), F.F.A.R.C.S.
F.F.A.R.A.C.S., A.M. (Malaysia).
Professor and Head, Dept. of Anaesthesiology,
and Intensive Care,
University of Malaya,
Kuala Lumpur,
MALAYSIA.

M. Namazie, M.B.B.S. (Madras), F.F.A.R.C.S.
Lecturer,
Dept. of Anaesthesiology,
University of Malaya,
Kuala Lumpur,
MALAYSIA.

TABLE 1: CLASSIFICATION OF SURVIVING PATIENTS

CATEGORY A: (3 Patients)	i) No neurological deficits. ii) Back to normal activity within 3 months.
CATEGORY B: (1 Patient)	i) Neurological deficits persisting for 3-6 months. ii) Back to normal activity within 3-6 months.
CATEGORY C: (1 Patient)	i) Neurological deficits persisting for 6-12 months. ii) Back to normal activity within 6-12 months.
CATEGORY D:	i) Neurological deficit persisting for more than 12 months.

DISCUSSION:

The management of head injured patients should ideally be carried out by a team consisting of the neurosurgeons, anaesthesiologist and sometimes the neurologist. Involvement of the anaesthesiologist has stemmed from the observations that early attention to maintenance of adequate airway has improved the prognosis of these patients. Echols *et al.* (1950) recommended tracheostomy on every unconscious head injured patient if it was considered that the coma would last longer than 24 hours. Huang *et al.* (1963) and Rossanda *et al.* (1966) have shown that this is the only way of ensuring a clear airway of comatose patients. There is now no doubt as to the importance of maintaining a free airway in the management of head injured patients. However, we do not tracheostomise our patients immediately. With the use of PVC endotracheal tubes with soft cuffs it is now possible to keep these tubes in the trachea for about 2 weeks without causing any serious damage to trachea. Tracheostomy is done if the coma lasts for more than 10 days.

A spontaneous over ventilation is often observed on head injured patients after establishing a clear airway. This leads to respiratory alkalosis and a raised arterial pH. (Frowein, 1970; Brown, 1970). Severely head injured patients may over ventilate to a stage of exhaustion which is further augmented by severe restlessness and decerebrate cramps. Sedatives, when used, calms the patients but always results in deterioration of the clinical condition.

The brain lesion creates intracerebral metabolic acidosis which is believed to initiate spontaneous hyperventilation (Kaasik *et al.* 1969). Experimental evidence shows that small shifts of as little as 0.1 unit from normal CSF pH are sufficient to cause changes in respiratory rate. (Pappenheimer, 1967). Zupping et al (1971) have shown close correlations between the severity of intracerebral acidosis, brain injury and the degree of hyperventilation.

Spontaneous hyperventilation is an attempt to restore the intracerebral pH to normal levels by rapid elimination of carbon dioxide. A normal cerebral pH is of fundamental requirement for maintenance of normal vasomotor tone, decrease of brain edema and intracranial pressure (ICP). Restitution of blood supply, resulting from normal ICP and vasomotor tone, to certain parts of the hypoxic brain diminishes the area of brain which might otherwise suffer permanent damage.

Though spontaneous hyperventilation is a useful protective mechanism it has several disadvantages. Heavy burden on the heart resulting from extra work of breathing, restlessness and decerebrate cramps soon leads to myocardial insufficiency and sometimes cardiac arrest. The patients may become exhausted and reduced pulmonary gas exchange may cause hypoxia and hypercarbia with catastrophic effects on cerebral blood flow and intracranial pressures. This may also be produced by sedative drugs used to control restlessness.

Controlled hyperventilation, on the other hand, allows adequate sedation, relieves muscular overwork, maintains adequate pulmonary gas exchange and allows administration of higher concentration of oxygen when indicated. Finer control of $PaCO_2$ levels is possible with controlled ventilation. $PaCO_2$ plays a major role in the control of cerebral blood flow and ICP. Controlled hyperventilation is known to decrease ICP and it may also partially compensate for the low intracerebral pH (Gordon and Rossanda, 1970).

Role of Steroids in Reducing ICP

Steroids are commonly used in the treatment of brain edema. Dramatic decrease in ICP is seen when the brain edema is due to brain tumors. The use of steroids in head injured patients is still controversial. Hoyt *et al.* (1972), showed no beneficial effect of dexamethasone when compared with a placebo in a double blind trial. Ransohoff (1972), in a double-blind study of patients with acute head injuries showed a tendency to an improved survival rate and a better quality of survival in the steriod treated group. However, this was not statistically significant because of the small number of patients. An increased survival rate in head injured patients treated with methyl prednisolone was re-

ported by Sparacio *et al.* (1965). However, Tornheim *et al.* (1978) in reviewing several clinical trials as well as experimental studies concluded that the controversial data that are presently available suggest that steroids are not as effective with traumatic brain edema as they are with edema stemming from other intracranial pathologies. In our patients we are not able to say whether the use of steroids influenced the final outcome since we had no control.

Hypothermia in Head Injured Patients:

Hypothermia decreases the metabolism and the oxygen requirement and therefore protects the neurones from ischaemic damage. Experimental study by Rosomoff (1959, 1961) showed that the beneficial effect of hypothermia occured only when the body temperature was reduced before the injury had taken place. Some effect may be observed if hypothermia is induced within three hours of injury and no effect could be demonstrated if the treatment is initiated after seven hours.

Hyperthermia, on the other hand, has highly damaging effects on neurones and must be prevented. Temperature should not be allowed to rise above normal. It is for this reason we maintain the temperature of our head injured patients at 34 $\pm$ 2°C.

All the 17 cases we managed showed signs of decerebration. The mortality of 64.7% is similar to that reported by Bricolo *et al.* (1977) in such patients. In decerebrate patients age does not affect mortality rate whereas in non-decerebrate patients the mortality increases significantly with age.

5 out of the 6 patients who survived in our series were back to normal activity within 12 months. The results obtained in this limited series have influenced us to give such patients the maximum possible chance for survival. Based on this preliminary small series we are now managing severely head injured patients with the aid of continuous I.C.P. monitoring to guide us rationally in controlling the I.C.P.

The quality of survival following severe head injury is of paramount importance to justify the great amount of money and human resources spent in the management of such patients. In our series 29% of our patients recovered sufficiently to resume their former activity within 12 months and, therefore, abandoning these patients appears to be unacceptable. However, with aggressive management of severely head injured patients it is possible to salvage a patient in a "persistent vegetative state" (Jennet and Plum, 1972). It is virtually impossible to differentiate in the early stages those who would recover fully from those who whould be in a persistent vegetative state. The uncertainty of prognosis therefore leaves no option but to aggressively treat all head injuries. Survival of some patients with permanent physical or mental handicaps is a price that must be paid to salvage some patients who would recover sufficiently to lead a normal life.

SUMMARY:

17 patients with severe head injuries were managed in the multidisciplinary Intensive Care Unit after craniotomy. The post-operative management consisted of Intermittent positive pressure ventilation, regular intravenous administration of dexamethasone, mild hypothermia and regular intravenous diazepam. Of the 17 patients, 6 patients survived. 5 of the surviving patients were back to their normal activities within 12 months. Based on this study it is felt that all severely head injury patients must be aggressively treated, survival of a few patients in a persistent vegetative state is inevitable if such a course of management is pursued.

ACKNOWLEDGEMENT:

We thank Mrs. F.S. Wong for the secretarial assistance rendered.

REFERENCES:

Bricolo, A., Turazzi, S., Alexandre, A., *et al.* (1977). Decerebrate rigidity in acute injury. *J. Neurosurg.*, **48:** 680-698.

Brown, A.S. (1970). Intermittent positive pressure ventilation in the management of severe head injury. In *Head Injury Symposium,* Churchill-Livingstone, Edinburgh, pp. 266-271.

Echols, D.H., Llewellyn, R., Kirgis, H.D., *et al.* (1950). Tracheostomy in the management of severe head injuries. *Surgery,* **28:** 801-811.

Frowein, R.A. (1970). Organization of head injury care in the hospital. In *Head Injury Symposium*, Churchill-Livingstone, Edinburgh, pp. 156-158.

Gordon, E., and Rossanda, M. (1970). Further studies on cerebrospinal fluid acid-base status in patients with brain lesions. *Acta Anaesth Scand.*, **14:** 97-109.

Hoyt, H.J., Goldstein, F.P., Reigel, D.H., *et al.* (1972). Clinic²/₃ evaluation of highly water-soluble steriods in the treatment of cerebral edema of traumatic origin. *Clin. Pharmacol Ther.*, **13:** 141 (Abstract).

Huang, C.T., Cook, A.W., and Lyons, H.A. (1963). Severe craniocerebral trauma and respiratory abnormalities. *Arch. Neurol.*, **9:** 545-554.

Jennet, B., and Plum, F. (1972). Persistent vegetative state after brain damage. *Lancet*, **1:** 734-737.

Jennet, B. (1975). Who cares for head injuries? *Br. Med. J.*, **3:** 267-270.

Kaasik, A.E. and Zupping, H. (1969). Correlations between gas exchange and CSF asid-base status in patients with cerebral haemorrhage. In Brock, M., Fieschi, C., Ingvar, D.H. *et al.* (eds), *Cerebral Blood Flow*, Springer, Berlin, pp. 129-131.

Pappenheimer, J.R. (1967). The ionic composition of cerebral extracellular fluids and its relation to control of breathing. *Harvey Lect.*, **61:** 71-94.

Ransohoff, J. (1972). The effects of steroids on brain edema in man. In Reulen, H.J., Schurmann, K., eds. *Steroids and Brain Edema,* Springer-Verlag, New York, pp. 211-218.

Registrar General (1975). *Statistical Review of England and Wales for the Year 1973*, H.M.S.O., London.

Rosomoff, H.L. (1959). Experimental brain injury during hypothermia. *J. Neurosurg.*, **16:** 177-185.

Rosomoff, H.L (1961). Effects of hypothermia and hypertonic urea on the distribution of intracranial contents. *J. Neurosurg.*, **18:** 753-759.

Rossanda, M., Di Guigno, G., Corona, S. *et al.* (1966). Oxygen supply to the brain and respirator treatment in severe comatose states. *Acta Anaesth. Scand.*, Supp. **23:** 766-774.

Sparacio, R.R., Lin, T.H., and Cook, A.W. (1965). Methyl prednisolone sodium succinate in acute cranio-cerebral trauma. *Surg. Gynaecol. Obstet.*, **121:** 513-516.

Tornheim, P.A. and McLaurin, R.L., (1978). Effect of dexa methasone of cerebral edema from cranial impact in the cat. *J. Neurosurg.*, **48:** 220-227.

Zupping, R., Kaasik, A.E. and Raudam, E. (1971). Cerebrospinal fluid metabolic acidosis and brain oxygen supply. Studies in patients with brain infarction. *Arch. Neurol.*, **25:** 33-38.