Outcome of Patients with Traumatic Brain Injury Managed on a Standardised Head Injury Protocol

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Abstract

A standardisedprotocol in the management of severe head injury in our hospital enables pre-determined critical care-paths and consistent treatment regimes to be instituted. In Singapore there has been no previously reported data on the outcome of severely head injured patients.

Over a 6-month period, 48 consecutive patients who were enrolled in our severe head injury protocol were prospectively studied. In addition to demographic and outcome data, physiologic measurements obtained from a computerisedpatient information system (Carevue Hewlett-Packard 9000) were analysed to determine the mean cerebral perfusion pressure (CPP) and intracranial pressure (ICP) achieved throughout the protocol period.

Median Glasgow Coma Score for all patients on admission to the protocol was 6 (range 4 to 8). The mean age was 34.46 ±15.03 years with a male to female ratio of 43:5. The average duration of treatment on the protocol was 110.73 hours. Initial ICP measured was 25.5 ±19.68 mmHg. Outcome was measured at 6 months post-injury using the Glasgow Outcome Score. Favourable outcome (GOS 4-5) was seen in 29 of 48patients (60.4%) while 12 out of 48 (25%) had an unfavourable outcome. There was a mortality of 14.6% (7 of 48 patients). Patients who survived had a higher mean CPP (P = 0.00005), a lower initial ICP and a mean ICP (P = 0.007 and 0.0009).

The use of a protocol with standardised treatment goals in the management of traumatic brain injury allows for the optimal use of limited resources and provides consistency in treatment. Good outcome is related to early aggressive resuscitation to prevent hypotension and hypoxia, prompt evacuation of surgical mass lesions and the maintenence of an adequate cerebral perfusion pressure. Our results are comparable with that reported in other established neurotrauma systems.

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Key words: Cerebral perfusion pressure, Glasgow Outcome Score, Intracranial pressure, Severe head injury

Introduction

Trauma is the fifth commonest cause of death in Singapore.' In 1996, trauma contributed to 5.4% of mortality. Severe head injuries are estimated to account for over half of trauma mortality, and a further unquantified proportion of severe and permanently disabled survivors. Patients tend to be young, economically productive, and result in a multiplied financial and emotional burden to previous dependents, subsequently rendered caregivers. As a tertiary neurosurgical and neurointensive care centre, our Departments treat approximately 1500 cases of head injury a year, including 200 craniotomies for patients with severe head injuries.

As basic and clinical research throw more light into the pathophysiology of traumatic brain injury (TBI), and the efficacy of specific regimes in its treatment, an increase in the intensity and complexity of clinical management follows. Fundamental concepts such as maintenance of an adequate cerebral perfusion pressure (CPP) and cerebral oxygenation, multi-modality monitoring of cerebral compliance (e.g. intracranial pressure monitoring); metabolism [e.g. xenon computerized tomography (CT), microdialysis, electroencephalography]; blood flow (e.g. transcranial Doppler and cortical laser flowmetry probe) and oxygen supply-demand relationships (e.g. jugular venous oxygen saturation) are widely accepted and practised, even if few of such modalities have been subjected to the gold standard of comparison in randomised-controlled studies.

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Nevertheless, uniformity of TBI treatment remains elusive, even in countries with highly developed medical systems such as the United States. Gajar et al[2] reported tremendous variability in the level of treatment and monitoring from respondents across 277 neurosurgical centres. Within a centre, adoption of a uniform treatment protocol is beneficial, since it compels practitioners to determine, at the onset, sound and evidence-based practice regimes, and further requires the centre to specify and streamline clinical care-paths, practices, and inter-departmental physician responsibilities, leading to more efficient and ultimately efficacious therapy.

In our centre, a critical care-path for severe TBI patients, from the point of admission to definitive care, has been established since early 1996. Also established has been a comprehensive treatment protocol in the intensive care unit. This paper describes salient aspects of the treatment regime, characteristics, and outcome measures of 48 consecutive patients admitted into the treatment protocol over a 6-month period.

Description of Treatment Protocol

All patients admitted to our hospital's Emergency Department with polytrauma undergo a resuscitation protocol based on priorities of management as outlined in the Advanced Trauma Life Support (ATLS) System. Patients with concomitant depressed conscious level are treated as for head injury. Immediate airway control, endotracheal intubation using pharmacological adjuncts, assumption of cervical spine injury until proven otherwise, and mechanical ventilation, are performed for patients who fulfil the criteria for severe head injury with a Glasgow Coma Scale score of 8 or less. Reversible causes of hypoxia, particularly due to mechanical impediment to adequate ventilation and oxygenation are aggressively treated. The patient is reviewed in the trauma room of the Emergency Department by the in-house neurosurgical registrar or senior registrar. Hypotension, in particular, is aggressively treated with isotonic crystalloid, colloids or blood products to maintain normotension. Patients suspected of raised intracranial pressure are given an empiric dose of mannitol. Pulmonary hyperventilation is not routinely initiated unless there is evidence of impending transtentorial herniation, and as a bridge to immediate craniotomy. Once stabilised, and in the absence of immediate life-threatening haemorrhage necessitating immediate non-neurosurgical intervention, the patient is sent for a CT scan. The presence of an intracranial mass lesion with significant mass effect is an indication for emergency craniotomy and evacuation of the mass. This decision is made at the CT scan room and the patient is transferred immediately to the operating room. In the absence of a surgically evacuable lesion, or after surgery, the patient is re-assessed in the neurosurgical intensive care unit (NICU) and recruited into the severe head injury treatment protocol based on the inclusion and exclusion criteria as shown in Table I. All patients in the treatment protocol are placed on continuous multi-modality monitoring of physiological parameters. These include general parameters as follows: continuous electrocardiograph, invasive intra-arterial pressure, continuous pulse oximetry, end-tidal CO_2, continuous transduced central venous pressure or pulmonary artery catheter, hourly urinary output, and core temperature. All data are continuously monitored and entered automatically at 15 minute intervals into a clinical information system (CIS, Hewlett-Packard Carevue 9000). Specific neurological parameters monitored include: 1) intracranial pressure using a strain gauge ICP monitor (Microsensor skull bolt kit Codman #82-6632 or Microsensor ventricular kit Codman #82-6633) in the intra-parenchymal, subdural, epidural or intraventicular space; 2) Jugular bulb venous oxygen saturation monitoring ($SjvO_2$), via the left or right internal jugular vein using a 7 F single lumen catheter (Arrow-Howes Single Lumen Central Venous Catheter) in the absence of contraindications e.g. coagulopathy, known cervical injury; 3) Cerebral perfusion pressure (CPP) computed as the mean arterial pressure (MAP) minus the ICI'; 4) Transcranial Doppler ultrasonography at least daily for patients with traumatic sub-arachnoid hemorrhage or to monitor middle cerebral arterial flow velocities in patients on induced barbiturate coma; 5) Processed or raw electroencephalograph (EEG) for patients on induced barbiturate coma; 6) Hourly clinical neurological examination; 7) repeat intracranial CT scan examination as indicated. The following investigations are performed on admission, at regular intervals, and as required: full serum electrolytes panel, serum osmolarity, full blood count, prothrombin time and partial thromboplastin test, arterial blood gases, chest radiograph, lower-extremity doppler ultrasonography. A cerebral perfusion pressure of 70 mmHg is targeted. This is

TABLE I: INCLUSION AND EXCLUSION CRITERIA FOR THE INSTITUTION OF THE HI PROTOCOL

Inclusion Criteria (one or more of the following:)
. Post resuscitation GCS of 8 or less, admitted to the ICU with severe TBI and/or CT evidence of severe TBI.
. Post craniotomy(for traumatic mass lesions) with potential for brain swelling.
. Intracranial mass lesions with space-occupying effects on CT scan and/or difficulty in assessing clinical neurological state.
Exclusion Criteria
. Uncorrectable haemodynamic instability with or without polytrauma, and unlikely to survive with or without surgery.
. Associated terminal illness for which no further medical intervention is desired.
. Evidence of brainstem death with CT evidence of irreversible brainstem injury.

achieved by maintaining normovolaemia with fluid boluses if the central venous pressure (CVP) is less than 8 to 12 mmHg, followed by infusion of vasopressors or inotropes (noradrenaline, adrenaline, or dopamine) to achieve an adequate MAP. Extra-cranial causes of hypotension are always sought for and the appropriate treatment instituted. In selected cases, a pulmonary artery catheter is used to guide fluid and electrolyte therapy, particularly in the elderly, or with intercurrent cardiogenic or septic shock. Prevention of raised ICI' includes the following routine measures: Adequate systemic and cerebral oxygenation. Sedation with continuous intravenous morphine infusion at 2 to 5 mg/h or propofol at 10 to 80 mg/h, titrated to effect. Endotracheal suctioning for bronchial toilet is performed after intra-tracheal instillation of lignocaine 4% 1 to 2 ml or succinylcholine 25 to 50 mg to reduce coughing. Head positioning is maintained in the neutral, in-line position to prevent jugular venous occlusion. Patients may be nursed up to 30 degrees in the reversed Tredelenburg position if MAP is not adversely affected. Normothermia is maintained with aggressive treatment of hyperthermia with anti-pyretics, anti-inflammatory agents, and surface cooling blankets. A source of infection contributing to hyper-pyrexia is sought for and treated aggressively. A sustained or upward trend in ICP above 20 mmHg is an indication to repeat an intracranial CT scan to re-evaluate brain parenchyma and exclude surgically treatable lesions. In the absence of a surgically amenable lesion, the MAP is raised to maintain the target CPP; sedation is deepened if restlessness or noxious stimulation is a potential cause; intermittent or continuous external ventricular drainage of cerebral spinal fluid (CSF) catheter against a pressure head of 20 mmHg; skeletal muscle paralysis with vecuronium bromide if ICI' elevation is attributable to raised intrathoracic pressure during mechanical ventilation. Subsequent treatment of raised ICP is based on mixed jugular venous oxygen saturation ($SjVO_2$). If $SjVO_2$ is indicative of cerebral hypaeremia (70% to 85%), minute ventilation is increased by 10% increments till ICP is reduced; if $SjVO_2$ less than 55%, systemic arterial hypoxaemia is excluded or treated, following which intravenous mannitol 25 gm bolus given over 20 minutes and observed for effect. Where brain compliance permits, minute ventilation is reduced by 10% increments until $SjvO_2$ is restored above ischaemic threshold of 55%. Mannitol infusions may be repeated to effect depending on the response to the initial dose. Barbiturate coma is induced if the above measures fail to control raised ICP below 30 mmHg and if CPP cannot be kept above 70 mmHg despite moderate intracranial hypertension. A bolus dose of IV thiopentone 500 to1000 mg is given, followed by 250 to 500 mg infusion hourly to achieve 3 to 5 EEG burst suppressions per minute. Barbiturates are withdrawn after 24 to 48 h of restoration of ICP <20 mmHg. Protocols and treatment guidelines for conventional ICU management, including early enteral nutrition, infectious disease surveillance, deep venous thrombosis surveillance and prophylaxis, and ventilatory management and weaning support the management of these head-injured patients.

Patients are weaned gradually from all aspects of the head injury protocol, on clinical neurological improvement and improvement of radiological and neurophysiological parameters.

Materials and Methods

All demographic and physiological data captured on the NICU patient database in 48 consecutive patients admitted into the Treatment Protocol from May 1996 to October 1996 were analysed. CT scan findings were categorised according to the Traumatic Coma Data Bank Classification of severity of head trauma Table IIa.[3] Outcome assessment at 6 months post-injury was performed by an occupational therapist blinded to the initial treatment and graded according to the Glasgow Outcome Score[4] (GOS), viz, favourable (GOS 5 & 4), unfavourable (GOS 3 & 2), or death (GOS 1). Data relating to all aspect of treatment, including duration of CPP management, age, post-resuscitation Glasgow Coma Scale, mean cerebral perfusion pressure, mean intracranial pressure were correlated with outcome.

Results

Patients Characteristics

Forty-three males and 5 females with an age range of 12 to 77 years and a mean of 34.46 ± 15.03 (SD) years were studied. The racial distribution is shown in Table III. Road traffic accidents accounted for the majority of the head injuries, occurring in 27 of the 48 patients (56%); of which motorcyclists accounted for 70.3%, pedestrians in 6 patients (22.2%), 1 car driver and 1 cyclist. Falls occurred in 14 patients (29%) and assaults in 2 patients (4%). In 5 patients the mechanism was unknown. Multiple injuries were seen in 22 of 48 patients (45.8%). The CT scan characteristics of the patient population are shown in Table IIb.

The initial post-resuscitation Glasgow Coma Scale (GCS) ranged from4 to 8. Those with a GCS of more than 8 were only admitted to the protocol when their GCS deteriorated to 8 (7 of 48 patients, 15.9%). The median post-resuscitation GCS was 6. Only one patient had documented hypotension on arrival at the emergency room and this was quickly corrected with aggressive fluid resuscitation. Twenty-seven of the 48 patients (56%) underwent emergency craniotomy to evacuate intracranial haematoma. ICP monitors were inserted in all patients and the mean initial ICP was 25.5 ± 19.68 mmHg (mean ± SD). The mean duration that the patients were placed on the protocol was 110.73 ± 73.93 hours with a

TABLE IIa: TRAUMATIC COMA DATABANK CLASSIFICATION OF CT SCAN FINDINGS

Category	Definition
Diffuse Injury I	No visible injury
Diffuse Injury II	Cisterns are present with midline shift of O-5 mm and/or Lesion densities are present No high or mixed density lesion of volume >25 ml Bone fragments and foreign bodies may be present
Diffuse Injury III	Cisterns are compressed or absent with midline shift of 0 to 5 mm; no high or mixed density lesion of volume >25 ml present
Diffuse Injury IV	Midline shift >5mm, no high or mixed density lesion of volume >25 ml
Evacuated mass lesion	Any surgical lesion removed
Non evacuated mass lesion	High or mixed density lesion of volume >25 ml not surgically removed.

TABLE IIb: DIAGNOSTIC CATEGORIES OF TYPES OF CT SCAN ABNORMALITIES AND NUMBER OF PATIENTS IN THE SERIES

Categories	No of Patients
Diffuse Injury I	0/48
Diffuse Injury II	2/48
Diffuse Injury III	8/48
Diffuse Injury IV	2/48
Surgically evacuated mass lesion	27/48
Mass lesion not evacuated surgically	9/48

range of 5 to 360 hours. The mean ICP was 25.2 ± 14.85 mmHg and the mean CPP was 70.52 mmHg ± 18.78 mmHg.

Outcome

Outcome of patients following severe head injury was assessed using the Glasgow Outcome Scale (GOS) measured at 6 months post-injury. The definition of GOS is shown in Table IV. Our results for patient outcome are shown in Figure 1. Favourable outcome (GOS 4 & 5) was seen in 29 of 48 patients (60.40%), unfavourable outcome (GOS 2 & 3) was seen in 12 of 48 patients (25%), and there was a mortality of 14.6% (7/48). Statistical analysis by ANOVA revealed that the differences in initial ICI', mean ICP and mean CPP amongst the 3 groups were statistically significant. Post-hoc tests using least significance difference, Student-Newman-Keuls tests revealed that the difference was mainly between patients who died and those who survived with respect to mean ICP, initial ICP and mean CPP (Table V). The relationships of

TABLE III: CHARACTERISTICS OF THE 48 PATIENTS WITH SEVERE HEAD INJURY

Age (y)	34.46 ± 15.03	
Sex ratio M:F	43:5	
Racial Distribution		
Chinese	24	(50%)
Malay	3	(7.1%)
Indian	13	(27.1%)
Others	8	(16.7%)
Length of protocol	110.73 ± 73.93	(range 5 to 360 h)
Median post-resuscitation GCS	6	(range 3 to 15)
Initial no. of patients with GCS <8	41	(85.4%)
Hypotension and hypoxia	1/48	(2.1%)
Initial ICP mmHg	25.5 ± 19.68	(range 2 to 75)
Mean ICP mmHg	25.2 ± 14.85	(range 1.8 to 73.8)
Mean CPP mmHg	70.52 ± 18.78	(range 2.7 to 109.5)
Craniotomy for haematoma	27/48	(56.25%)

TABLE IV: DEFINITION OF GLASGOW OUTCOME SCORE[4]

GOS	Definition
5	Good recovery. Resumption of normal life. May have minor neurologic and psychological deficits
4	Moderate disability (disabled but independent). Ability to travel by public transport and can work in a sheltered environment. Disabilities include varying degrees of dysphasia, hemiparesis, ataxias, intellectual and memory deficits and personality change.
3	Severe disability (conscious but disabled). Dependent for daily support due to mental or physical disabilities or both. Includes patients only able to maintain self care within the room and house.
2	Persistent vegetative state
1	Death

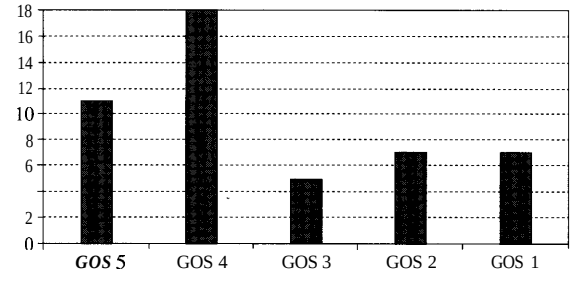
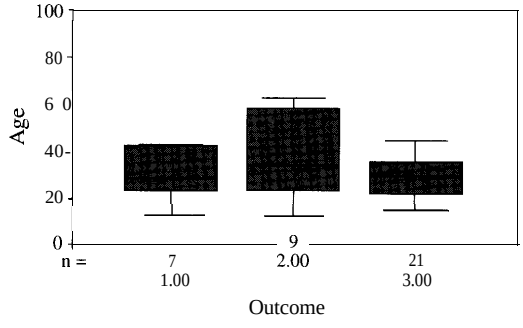
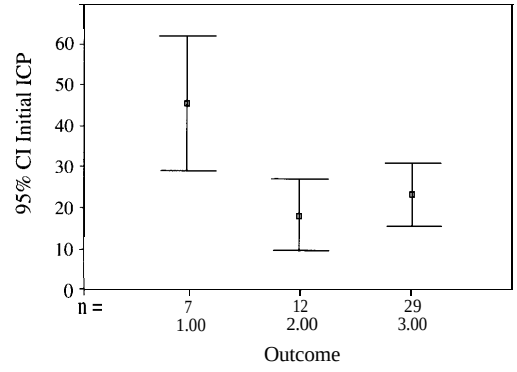


Fig. 1. Outcome of patients.

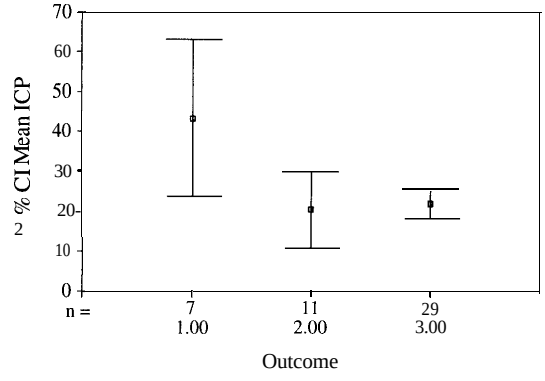
outcome are shown in the respective bar charts and scatterplots (Figs. 2 to 7). There was a significant relationship between the length of protocol and the post-resuscitation GCS (coefficient -0.3847, $P = 0.013$, Fig. 7). Patients who were admitted with a better initial post-resuscitation GCS tended to be on the protocol for a shorter period of time. Patients admitted with a poor



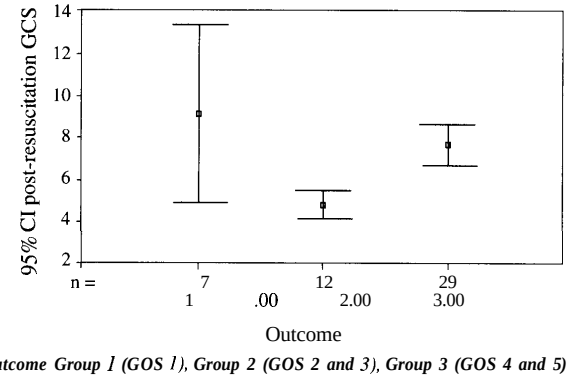
Outcome Group 1 (GOS I), Group 2 (GOS 2 and 3), Group 3 (GOS 4 and 5)
Fig. 2. Error Bar Chart of age and outcome.



Outcome Group 1 (GOS I), Group 2 (GOS 2 and 3), Group 3 (GOS 4 and 5)
Fig. 3. Error Bar Chart of initial ICP and outcome.



Outcome Group 1 (GOS 1), Group 2 (GOS 2 and 3), Group 3 (4 and 5)
Fig. 4. Error Bar Chart of mean ICP and outcome.



Outcome Group 1 (GOS 1), Group 2 (GOS 2 and 3), Group 3 (GOS 4 and 5)
Fig. 5. Error Bar Chart of post-resuscitation GCS and outcome.

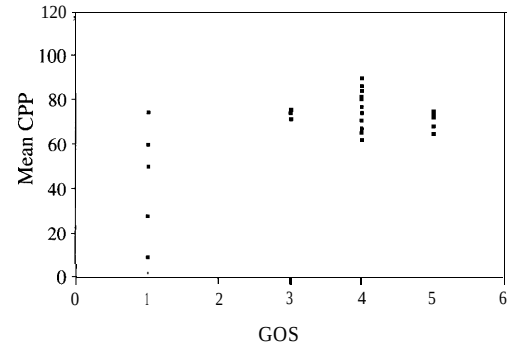


Fig. 6. Scatter plot of mean CPP versus Glasgow Outcome Score.

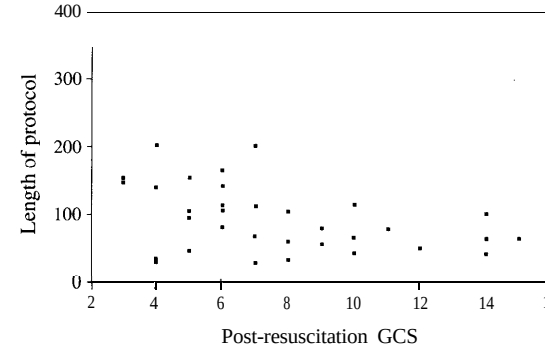


Fig. 7.** Scatterplot **of length of protocol and post-resuscitation GCS.

GCS generally were on the protocol longer if they ultimately survived, those with a poor GCS but had only had a short period of monitoring probably did not survive the head injury. Operative cases (emergency craniotomies) when compared with non-operated cases revealed no significant difference between these 2 groups ($P = 0.6029$) (Table VI).

Pharmacological intervention was also reviewed. Intravenous morphine and propofol was used in37 (82.2%) and 30 (68%) patients respectively. Mannitol was us ed in 40 (88.9%) patients for control of intracranial hypertension. Inotropes were used to improve CPP in 31 patients (70%). Six patients (12.5%) had intractable intracranial hypertension that necessitated the use of barbiturate coma.

Our outcome results were compared to recently published series in the literature (Table VII).

Discussion

Prognostic factors governing outcome after severe head injury have been well characterised by many large studies.[5-7] They include age, duration between traumatic event and definitive treatment (including craniotomy), documented hypotension and hypoxia, initial post-resuscitation GCS, pupillary abnormality, the initial intracranial pressure, and more recently the adequacy of cerebral perfusion pressure. Improvements in pre-hospital stabilisation and transfer to the Accident and Emer-

TABLE V: CORRELATION BETWEEN OUTCOME AND MEAN VARIABLES

	Favourable	Unfavourable	Dead	F ratio	F probability
Age	32.97 ±12.22	37.25 ± 18.28	35.86 ± 20.79	0.37	0.69
Length of protocol	112.17 ± 86.6	133 ± 50	74 ± 39.4	1.34	0.27
Initial ICP	23.48 ± 19.52	18.5 ± 13.59	45.86 ± 17.79	5.56	0.007
Mean ICP	22.5 ± 9.9	20.7 ± 13.9	43.5 ± 21.3	8.3	0.0009
Mean CPP	76.0 ± 11.55	74.87 ± 8.57	40.86 ± 27.48	17.68	.0000

TABLE VI: OUTCOME CORRELATED WITH SURGERY VERSUS NO SURGERY

	Surgery	No surgery
Favourable outcome	17	12
Unfavourable outcome	5	7
Dead	4	3

Fischer's exact test: not significant $P = 0.6092$

TABLE VII: OUTCOME DATA PUBLISHED IN THE LITERATURE

GOS	Ng I et al 1998 n = 178 %	Cruz[35] 1998 n = 158 %	Rosner[18] 1995 n = 746 %	TCDB[6] 1991 %
n = 48				
4-5	60.4	74	59	25.2
3	10.4	14	9	28
l-2	29.2	12	31	46.5
Mortality	14.6	9	29	32.5

gency Department have presented us with the opportunity to enable a larger group of patients to benefit from early aggressive resuscitation, surgical management and intensive care monitoring and manipulation of intracranial pressure and cerebral perfusion pressure. The use of protocols is now well accepted and implemented in most neurotrauma centres around the world as a means to such ends. The recent joint Brain Trauma Foundation publication of "Guidelines in the management of severe head injury" has summarised currently accepted treatment standards for severely head injured patients.[8] Triage and patient selection are still deemed necessary to exclude unsalvageable patients from a protracted and expensive treatment protocol, and to conserve limited ICU resources for patients with the best likelihood for meaningful survival. In our series we have excluded patients from the protocol if they were deemed to be unsalvageable due to the severity of their injuries. We also excluded patients from the series who were admitted to the NICU with a head injury but had a GCS that was more than 8 from this reported series. However our inclusion criteria err on the side of aggressive pre-emptive monitoring and treatment of moderate head injured patients with a potential for deterioration in addition to those who are severely head injured on admission even though they were not ultimately included into this series unless there was secondary deterioration in the NICU. Such "Talk and Die" patients have been recognised by various workers in the past for the significant contribution to unfavourable outcome from in-hospital secondary brain insults.[9]

A review of the current literature shows that our treatment protocol is based on sound clinical evidence. Very early (<6 hours) after injury, global reduction in cerebral blood flow (CBF) to ischaemic levels (<18 mL/100 g/min) occurs in a small but significant proportion of patients. Outcome is significantly correlated with the degree and duration of ischaemia. Conversely, early decreased CBF return to normal values in patients who recover from their injuries. Cerebral metabolism is co-related with outcome: in patients who regain consciousness, there is a corresponding increase in CMRO, as the functional status improves. This is also related to an increase in the cerebral blood flow.[10-14] A strategy to maintain an adequate cerebral perfusion pressure in order to improve cerebral blood flow to the ischaemic areas of the brain is borne out by evidence of an improve ment and stability in $SjvO_2$ and reduction and stability in pulsitility index, using the transcranial Doppler, as cerebral perfusion pressure is optimally increased.[15] A CPP paradigm in head injury management has been shown to correlate with outcome in studies previously reported by us and others.[16-18] There is however potential for the development of vasogenic oedema in the setting of very high cerebral perfusion pressures in cerebral vessel beds in which pressure autoregulation is lost, resulting in a pressure passive increase in cerebral blood flow.[19,20]

The ability to individualise optimisation of CPP remains a worthwhile goal that can be achieved by a variety of multi-modality monitors, including arterio-jugular venous oxygen difference, transcranial doppler (TCD) and regional cerebral blood flow measurements. In our protocol, a combination of ICI', mean arterial pressure (MAP) and $SjvO_2$ measurements serve to provide the basic parameters monitored to determine adequacy of cerebral perfusion pressure, and concomitant evidence of ischaemic or hypaeremic blood flows. Jugular venous saturation reference values (<55%; >75%), rather than $A\text{-}jVO_2$ differences, is used to negate possible confounding influence of haemoglobin concentra-

tion in the determining an ischaemic state. In states of ischaemic $SjvO_2$ <55%, systemic hypoxia is excluded, followed by a search for possible focal deficit or mass lesions as a cause for cerebral ischaemia.[21] In their absence, an elevation in $PaCO_2$ is justified to reduce global cerebral vasoconstriction provided this does not result in rebound intracranial hypertension.[22,23] An alternative pathway in the treatment algorithm is to administer mannitol to reduce blood viscosity, improve cerebral blood flow, and reduce cerebral blood volume, particularly in vessel beds with intact flow autoregulation.[24] Kirkpatrick et al[25] have recently shown that mannitol infusion improves $SjvO_2$, reduce ICP even in vessel beds that do not exhibit intact autoregulation as evidenced by increase in TCD velocities. Muizelaar et al[26] had previously studied the effects of mannitol in severe head injury and concluded that its efficacy in reducing CBV is significant only in patients with intact autoregulation. Mannitol infusions, if efficacious, may be continued intermittently to effect, with frequent serum osmolality and osmolar gap monitoring. The role of empiric mannitol in patients with high ICP and CT evidence of low-density areas of "cerebral oedema" has also been advocated although this is not practised in our centre if there is no evidence of global ischaemia and ICP does not exceed 30 mmHg in the presence of an adequate CPP >70 mmHg.[27] Cerebral hypaeremia in the absence of intracranial hypertension is not an indication for treatment, and may passively signify improved CBF commensurate with improved clinical status over time, as described above.

The clinical efficacy of mild hypothermia in severe head injury has been shown experimentally to result in suppression of excitatory amino acids and dopamine in the brain's extracellular space.[28] Two recent small prospective studies have shown evidence of improved outcome at 6 months,[29,30] prompting a larger multi-centre trial that is currently underway. The application of mild hypothermia is not without its complications, particularly in causing peripheral vasoconstriction and shivering that necessitates careful haemodynamic management with induced vasodilatation to reduce peripheral vascular resistance and pharmacological paralysis. There has however been no evidence that a hypocoagulable state is induced at temperatures of 34°C in head injured patients studied.[29] Supportive therapies in the form of optimal sedation management, ventilatory management, early enteral nutritional support (including replacement of early hypozincemia and hyperzincuria) and prevention of gastroparesis, deep venous thrombosis prophylaxis, and a rational infection prevention regime probably play a more significant role in reducing morbidity and improving outcome than is presently recognised.[31-34]

Outcome at 6 months in our study was not correlated with age, but was strongly correlated with the initial ICI', mean ICP and CPP. That the lack of correlation with age is surprising, since many reports have shown a direct association, but a correlation of initial and mean ICP is to be expected, since a more severe primary head injury reflected by a higher initial ICP should expectedly have a poorer outcome.

Mean ICI' was significantly associated with outcome, and this agreed with the numerous papers on intracranial pressure. Analysis of mean CPP achieved shows that survivors generally had a CPP of more than 70 mmHg, as opposed to those who died (mean 40.86, P <0.00005). This seems to be in agreement with data previously described by Rosner et al.[17,18]

We also attempted to compare our results with that of the literature on the outcome following head injury. The Traumatic Coma Databank (TCDB) results published in 1991[6] shows outcome based on reduction of ICP. With increased understanding of the pathophysiology of traumatic brain injury, several workers have published their results based on CPP maintenance[17,18] and optimised hyperventilation[35] with improved results. Our results which reflect the same management philosophy show comparable results in outcome.

It is difficult to determine if the implementation of the protocol has improved outcome of these group of patients in Singapore. However, the acceptance of the staff involved in the day to day management of these patients are high, and a general reduction of the number of brain dead patients with stable haemodynamic parameters in the ICU may be indicative of our improving efforts at detecting and treating secondary brain insults.

Conclusions

The use a standardised head injury protocol in the management of traumatic brain injury patients allows us the maximal use of our available health resources, and also provides consistency in the treatment process. An aggressive approach in the prevention of hypotension and hypoxia, prompt extirpation of a surgical mass lesion and the maintenance of an adequate cerebral perfusion pressure and control of intracranial hypertension using judicious use of optimised hyperventilation and osmotic diuretics in these patients are essential in ensuring good outcome in this group of patients.

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