SECONDARY INJURY IN TRAUMATIC BRAIN INJURY PATIENTS — A PROSPECTIVE STUDY

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Objective. Secondary insults of hypotension and hypoxia significantly impact on outcome in patients with traumatic brain injury (TBI). More than 4 hours' delay in evacuation of intracranial haematomas has been demonstrated to have an additional impact on outcome. The objective of this study was to document the incidence of these preventable secondary insults in patients admitted with moderate or severe brain injury.

Methodology. All moderate and severe head injury patients admitted to Groote Schuur Hospital over a 3-month period were studied prospectively. Data were obtained from ambulance dockets, referral letters, patient charts and attending medical staff. Preventable secondary insults (hypotension, hypoxia) and time delay to assessment and surgery were documented. Outcome was assessed using the Glasgow outcome scale (GOS) at discharge or outpatient follow-up.

Results. Ninety-six patients were studied. Forty-nine patients experienced at least one recorded preventable event of hypoxia or hypotension. Seventeen had an intracranial haematoma requiring evacuation. The mean time interval between injury and surgery was 455 minutes. No haematoma was evacuated within 4 hours of injury. Patients referred via a primary or secondary care facility experienced a mean additional delay of 70 minutes.

These results demonstrated a significant incidence of secondary injury and delay to assessment and surgery. We believe that education and a raised awareness of the impact of secondary insults may have a positive impact on TBI outcome in our referral area.

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Traumatic brain injury (TBI) is a significant cause of morbidity and mortality in South Africa. The nature and degree of the primary injury are powerful determinants of outcome following TBI. No therapeutic intervention has been shown to alter the effect of the primary injury. The injured brain is vulnerable to secondary insults in the immediate post-injury period. Ischaemia commonly occurs in association with severe TBI.[1] There is strong evidence that secondary ischaemic insults to the brain worsen outcome.[2] Even brief periods of systolic hypotension or hypoxia have been associated with dramatically increased mortality.[2]

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In patients with TBI, those with acute intracranial haematomas have been shown to have a particularly poor outcome. The mortality rate reported in unconscious patients with acute subdural haematomas has been as high as 60 - 90%. This dismal prognosis may be improved with rapid evacuation of intracranial haematomas. Seelig *et al.*[3] reported a 30% mortality for patients with acute subdurals evacuated within 4 hours of injury and a three-fold increase in mortality if the surgery was performed more than 8 hours after injury. The case for rapid evacuation of extradural haematomas is even more compelling.[4]

There seems little point in pursuing expensive and unproven therapies for TBI until the incidence of preventable secondary insults has been markedly reduced. We therefore studied the incidence of hypoxia, hypotension and delayed evacuation of intracranial haematomas in patients with TBI referred to a tertiary care hospital.

METHOD

All TBI patients with a Glasgow Coma Scale (GCS) score less than 13 admitted to Groote Schuur Hospital (GSH) from 1 December 1999 to 29 February 2000 were entered into the study. Data were entered prospectively from the time of admission. Data were obtained from referral letters, ambulance dockets and patient charts. Initial assessment of vital signs and airway management were entered by the admitting trauma unit doctors. An arterial blood gas measurement was performed on admission. Neurological assessment was undertaken by the admitting neurosurgeon. Patient records were examined daily for evidence of recorded secondary insults. Hypotension was defined as systolic blood pressure less than 90 mmHg and hypoxia as pulse oximetry oxygen saturation of less than 90% or arterial partial pressure of oxygen less than 8 kPa. Events other than the strictly defined hypoxia and hypotension were also recorded. These were recorded as 'other' and included airway obstruction, pulmonary aspiration and seizures.

Computed tomography (CT) scans were reviewed by one person (DGW) and patients were assigned to one category each according to the findings. In this categorisation intracranial haematomas and other focal pathology took precedence over diffuse injury. CT scans with evidence of gliding contusions, small white matter haemorrhages or cerebral swelling were labelled 'diffuse injury'.

The following times were recorded: time of injury, arrival at

GSH, assessment by a neurosurgeon and start of anaesthetic. Times were obtained from ambulance dockets and patient records. If time of injury was unknown or not recorded then ambulance dispatch time was taken as time of injury.

Outcome was assessed using the Glasgow Outcome Scale (GOS)[5] at discharge or at follow-up outpatient visits. Follow-up times were all less than 6 weeks. Fisher's exact one-tailed test was used for this statistical analysis.

RESULTS

Demography

A total of 96 patients were entered into the trial — 83 men and 13 women. The mean age was 31.5 years, with a range of 13 - 79 years.

Table I. Mechanism of injury

Mechanism of injury	Number of patients
MVA occupant	20
MVA pedestrian	26
Assault (blunt instrument)	20
Assault (sharp instrument)	7
Gunshot	8
Fall	9
Other/unknown	6
Total	96

MVA = motor vehicle accident.

In addition to head injury, 51 patients suffered other non-neurological injuries, including 28 patients with long bone fractures, 21 with thoracic injuries, 10 with abdominal injuries and 26 with maxillofacial or cervical spine injuries.

Secondary insults

During the pre-admission period (from injury to the GSH trauma unit) a total of 36 patients suffered recorded secondary insults. Of these, 21 experienced hypoxia, hypotension or both.

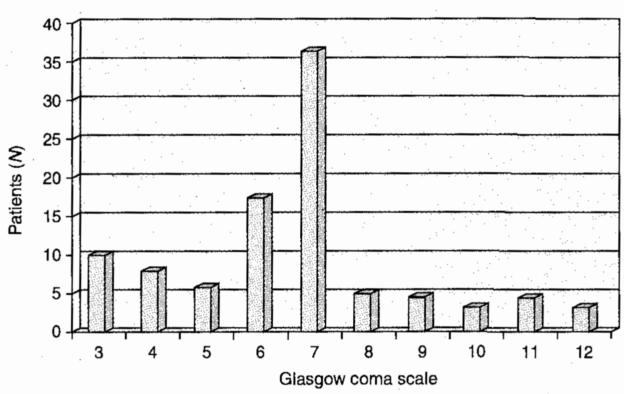

Fig. 1. Glasgow Coma Scale on admission.

Table II. Computed tomography (CT) scan findings

CT scan findings	Number of patients
Diffuse injury	24
Focal contusion	19
Subarachnoid haemorrhage	13
Subdural haematoma	22
Extradural haematoma	7
Normal CT scan	9
CT not done	2
Total	96

Table III. Number of patients recorded as suffering at least one secondary insult at each care stage

Care stage	Hypotension	Hypoxia	Both	Other*
Pre-admission (N = 36)	8	7	6	15
Emergency room (N = 8)	6	2	0	0
Hospital ward (N = 34)	13	11	7	3

* Includes airway obstruction, pulmonary aspiration and seizures.

A total of 8 patients suffered either hypoxia or hypotension in the trauma unit emergency room.

A total of 34 patients suffered at least one episode of recorded secondary insult in the hospital ward. Of these, 13 experienced hypotension, 11 hypoxia and 7 both, while 3 patients experienced airway obstruction or seizure.

Overall, 49 patients experienced at least one recorded episode of hypotension, hypoxia or airway obstruction from the time of injury to 72 hours post injury.

Recorded delay

The following times were recorded: time of injury, time of admission to GSH, time of assessment by the neurosurgeon and time of start of the anaesthetic. Overall, the mean time between injury and neurosurgical assessment was 354 minutes. In patients with intracranial haematomas requiring surgical

Table IV. Mean time intervals from injury to GSH admission, assessment and operation

	N*	Mean time (minutes)
Injury — GSH	76	141 (SD = 146)
GSH — neurosurgeon	84	225 (SD = 163)
Neurosurgeon — operation	17	113 (SD = 70)

* Number of patients, at each stage, where accurate times were available.
SD = standard deviations.

evacuation (N = 17), the mean time from injury to theatre was 455 (standard deviation (SD) 236) minutes. No haematoma was evacuated within 4 hours of injury and 3 patients underwent surgery more than 8 hours after the injury.

Patient referral patterns showed that of the 96 patients, 39 were transferred from the scene of the injury directly to GSH. Forty-two patients were initially assessed at a secondary-level hospital, 13 initially at a primary-level hospital and 2 patients at both primary and secondary level before transfer to GSH. The mean time between injury and neurosurgical assessment was 304 (SD 152) minutes for patients transferred directly to GSH and 374 (SD 234) minutes for patients transferred via another hospital.

Outcome

Outcome data were available for 74 patients.

Of the patients who experienced secondary insults, 15 had a good outcome (GOS 4 and 5) and 10 had a poor outcome (GOS 1 - 3). In the group with no recorded secondary insults 23 patients had a good outcome and 25 a poor outcome. There was no significant difference (P = 0.23) between the two groups.

Table V. Glasgow outcome scales (GOS)

GOS *	All patients (N)	Patients GCS ≤ 8 (N)
1	29	29
2	2	2
3	4	2
4	24	19
5	15	9
Total	74	61

*GOS: 1 = dead; 2 = vegetative state; 3 = severe disability; 4 = minor disability; 5 = no disability.

Discussion

The demographic characteristics of the patients in this study are typical of those in other studies of head injury, with the majority of patients being young males. The mechanism of injury was mainly motor vehicle accident and interpersonal violence.

The majority of patients (82:96) suffered severe head injuries (GCS ≤ 8). A possible reason for this is that patients with initial higher GCS scores were observed at primary- and secondary-level hospitals and only referred to GSH if they deteriorated or failed to improve. The large number of patients (N = 36) with a GCS of 7 is partly due to the practice of sedation and endotracheal intubation of restless patients in order to facilitate CT scanning. Intubated patients were assigned a verbal GCS score of 1.

With regard to the CT scan diagnosis, one-third of patients had diffuse injuries. Another one-third had intracranial extra-axial haematomas. Not all patients with intracranial haematomas proceeded to surgery. This is because either the haematomas were considered too small to require decompression or the patient had no motor response and surgery was considered futile. Two patients died before a CT scan could be performed.

More than half of the patients suffered injuries other than head injuries. Among these patients facial (27.8%) and chest (21.8%) injuries were relatively common, increasing the risk of airway obstruction and hypoxic events.

Neurological deficit following TBI has long-term personal, social and economic implications for the patient and society. Any intervention demonstrated to improve outcome in this group of patients could be very cost effective, especially if the intervention itself was inexpensive.

Using data from the traumatic coma data bank, Chesnut *et al.*[2] demonstrated the significant impact of even brief periods of hypotension or hypoxia in patients with acute TBI. Patients who experienced neither hypotension nor hypoxia had a mortality of 27% which increased to 64% in patients with even a single episode of hypotension. Hypoxia alone was associated with a smaller but still significant increase in mortality to 50%. Hypotension and hypoxia in combination led to a threefold increase in mortality to 75%. These data have been supported by work demonstrating that both early episodes of hypoxia and/or hypotension, and also later insults during subsequent intensive care, were associated with worse outcome.[6,7]

In our study more than half the patients (49:96) experienced at least one recorded episode of hypoxia, hypotension or airway obstruction. It must be stressed that this is the minimum rate of secondary insult, as many events may have escaped recognition or not have been recorded. One-third of the patients experienced secondary insults in the pre-admission period. This includes the immediate post-injury period, initial stabilisation by ambulance personnel, transport and primary or secondary hospital assessment/resuscitation.

Recorded secondary insults in the GSH trauma unit emergency room were relatively few (8:96). We believe this is an accurate figure as patients are monitored continuously and vital signs are documented half-hourly.

Twenty-one patients suffered secondary insults in the GSH hospital wards. This includes patients in the neurosurgical intensive care unit, trauma high care unit and general wards. It is in these locations that patients will have spent relatively the longest period of time and a proportional increase in the number of recorded secondary insults would be expected.

These results demonstrate that a significant opportunity exists to improve the outcome from TBI in our patients by the diligent application of the basic principles of resuscitation.

Particular attention needs to be paid to patients with suspected TBI so that they are assured optimal cardiovascular resuscitation and oxygenation. Awareness needs to be raised about the particular implications of failure to ensure oxygenation of the injured brain.

Intracranial haematomas resulting in raised intracranial pressure are an important cause of secondary injury in TBI. Prompt evacuation of intracranial haematomas may reduce mortality significantly. Seelig *et al.*[3] published findings on a series of 82 consecutive patients with acute subdural haematomas. If the haematoma was evacuated within 4 hours of injury, mortality was 30%. Mortality increased to 90% if the haematoma was evacuated more than 8 hours after injury (P = 0.0001).

In our study no intracranial haematoma was evacuated within 4 hours of injury and at least 3 were evacuated more than 8 hours post-injury.

The mean time from injury to GSH was 221 minutes. We believe that the times recorded for this period are an underestimate. For those patients referred from another hospital the time of injury was seldom available. For these patients time of injury was taken as the time the ambulance was despatched to transfer the patient to GSH. More than half of the patients (59:96) were assessed at another hospital before transfer to GSH. This was associated with a mean additional delay of 70 minutes before assessment by a neurosurgeon.

The mean time from admission to assessment by a neurosurgeon was 225 minutes. This time includes initial assessment and resuscitation by trauma unit staff, transport to and from the CT scanner and neurosurgical assessment. That this period amounts to more than 3.5 hours reflects both the lack of resources available to deal with these cases and probably an underestimation of the urgency required.

The mean time between neurosurgical assessment and start of anaesthesia was 113 minutes. This delay of almost 2 hours is a reflection of both a lack of available theatre time and that TBI cases have to compete for theatre time with other urgent surgical cases.

The evidence suggests that delayed evacuation of intracranial haematomas has a significant impact on mortality. Our study identified time to haematoma evacuation as an area where every patient experienced unacceptable delay. Even patients with a relatively high initial GCS may have a high risk of harbouring an intracranial haematoma.[8] Surgical outcome is much better if the haematoma is diagnosed and evacuated before the patient becomes deeply comatose.[4] We believe that in a city such as Cape Town there is little to be gained by admitting patients with significant head injuries to primary and secondary care hospitals where there is no diagnostic CT scan capability or neurosurgical expertise. This can only lead to delays in the provision of definitive neurosurgical care, with potential negative effect on outcome.

These results support the institution of a policy decision to transfer all patients with suspected moderate or severe TBI directly to a hospital where diagnostic CT capability exists and is supported by a neurosurgical service. Within this service there needs to be an understanding by all practitioners of the critical impact that delay can have on outcome and that patients need to have their diagnostic and definitive therapeutic procedures expedited.

These results show no significant difference in outcome between the patients with recorded secondary insults and those without recorded insults. The incidence of secondary insults is so high that it is probable that many secondary insults in both groups went unrecorded. This makes the outcome data difficult to interpret and would require a larger study with an improved ability to detect insults.

In conclusion, more than half the patients with TBI admitted to GSH experienced potentially avoidable secondary insults and all patients requiring surgery for intracranial haematomas suffered unacceptable delay. We need to target these areas in an attempt to limit the impact of TBI before we consider more expensive and less efficacious approaches.

References

1. Graham DI, Ford I, Adams JH, *et al.* Ischaemic brain damage is still common in fatal non-missile head injury. *J Neurol Neurosurg Psychiatry* 1989; **52:** 346-350.
2. Chesnut RM, Marshall LF, Klauber MR, *et al.* The role of secondary brain injury in determining outcome from severe head injury. *J Trauma* 1993; **34:** 216-222.
3. Seelig JM, Becker DP, Miller JD, Greenberg RP, Ward JD, Choi SC. Traumatic acute subdural haematoma: a major mortality reduction in comatose patients treated within four hours. *N Engl J Med* 1981; **304:** 1511-1518.
4. Seelig JM, Marshall LF, Toutant SM, *et al.* Traumatic epidural haematoma: unrecognized high lethality in comatose patients. *Neurosurgery* 1984; **15:** 617-620.
5. Jennett B, Bond M. Assessment of outcome after severe brain damage. *Lancet* 1975: **1:** 480-484.
6. Jones PA, Andrews PJD, Midgley S, *et al.* Measuring the burden of secondary insults in head-injured patients during intensive care. *J Neurosurg Anesthesiol* 1994; **6:** 4-14.
7. Robertson CS, Contant CF, Gokaslan ZL, Narayan RL, Grossman RG. Cerebral blood flow, arteriovenous oxygen difference and outcome in head injured patients. *J Neurol Neurosurg Psychiatry* 1992; **55:** 594-603.
8. Teasdale GM, Murray G, Anderson E, *et al.* Risks of traumatic intracranial haematoma in children and adults, implications for managing head injuries. *BMJ* 1990; **300:** 363-367.

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