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A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension

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Abstract *Object:* The object of our study was to determine, in children with traumatic brain injury and sustained intracranial hypertension, whether very early decompressive craniectomy improves control of intracranial hypertension and long-term function and quality of life. *Methods:* All children were managed from admission onward according to a standardized protocol for head injury management. Children with raised intracranial pressure (ICP) were randomized to standardized management alone or standardized management plus cerebral decompression. A decompressive bitemporal craniectomy was performed at a median of 19.2 h (range 7.3–29.3 h) from the time of injury. ICP was recorded hourly via an intraventricular catheter. Compared with the ICP before randomization, the mean ICP was 3.69 mmHg lower in the 48 h after randomization in the control group, and 8.98 mmHg lower in the 48 hours after craniectomy in the decompression group (P=0.057). Outcome was assessed 6 months after injury using a modification of the Glasgow Outcome Score (GOS) and the Health State Utility Index (Mark 1). Two (14%) of the 14 children in the control group were normal or had a mild disability after 6 months, compared with 7 (54%) of the 13 children in the decompression group. Our conclusion was that when children with traumatic brain injury and sustained intracranial hypertension are treated with a combination of very early decompressive craniectomy and conventional medical management, it is more likely that ICP will be reduced, fewer episodes of intracranial hypertension will occur, and functional outcome and quality of life may be better than in children treated with medical management alone (P=0.046; owing to multiple significance testing P <0.0221 is required for statistical significance). This pilot study suggests that very early decompressive craniectomy may be indicated in the treatment of traumatic brain injury.

Keywords Decompressive craniectomy · Traumatic brain injury · Intracranial pressure · Outcome

Introduction

The maintenance of cerebral perfusion pressure is an important aspect of the management of traumatic brain injury (TBI). Despite advances in invasive and noninvasive cerebral monitoring, and improvements in the pharmacological management and understanding of cerebral oedema, the mortality and morbidity of patients with severe TBI remains high [1, 6, 28). This reflects the extent of both the primary injury and any secondary insults caused by inadequate cerebral perfusion [10, 23, 37]. Raised intracranial pressure (ICP), and particularly high peak pressure, is associated with a poor outcome from head injury [6, 27, 31, 32, 36]. There is no controlled tri-

al showing that ICP monitoring and lowering of ICP improves long-term outcome in head injury, but significant reductions in mortality and morbidity can be achieved in patients with severe head injury by using intensive management protocols [6, 27, 31). In both animal and human studies, surgical decompression has been found to lower ICP by increasing intracranial volume [2, 5, 20, 21, 22]; however, a number of studies have found that decompression may cause worsening of cerebral oedema [11, 16], haemorrhage [33], and shift of the brain with variable degrees of necrosis [24, 33, 4].

Decompressive craniectomy has been well described in the management of encephalopathy of Reye's syndrome [3], ischaemic stroke with brain infarction [2, 13, 14, 15, 25], cerebral tumour [40], severe encephalitis [38] and TBI [2, 12, 17, 18, 19, 21, 24, 25, 35, 43, 44, 45]. These study reports suggest that decompressive craniectomy lowers ICP [12, 18, 21, 35, 43], that the timing of decompressive craniectomy in relation to injury is relevant [12, 13, 17, 35, 45] and that the functional outcome of patients is improved [12, 15, 17, 19, 24, 34, 35, 43, 45]; unfortunately, the studies used historical controls, nonrandomized controls or no controls.

We therefore performed a prospective randomized control trial of early decompressive craniectomy. The principal outcome was a functional assessment of outcome at 6 months after injury; a secondary outcome was the control of ICP.

Methods

All children over 12 months of age who were admitted to a 16-bed multi-disciplinary paediatric intensive care unit were eligible for the study if they had sustained a TBI and had a functioning intraventricular catheter. All children were treated from admission onward according to a standardized protocol for head injury management (Appendix A), the major goal being to maintain ICP <20 mmHg with an adequate cerebral perfusion pressure. This was defined initially as a cerebral perfusion pressure >50 mmHg. In 1993, parameters for an adequate cerebral perfusion pressure were then adjusted for age; ≥35 mmHg (1–4 years), ≥40 mmHg (5–8 years), ≥45 mmHg (9–12 years) and ≥50 mmHg (>12 years). A final definition for adequate cerebral perfusion pressure was then implemented in 1997: ≥50 mmHg (1–4 years), ≥60 mmHg (5–8 years) and ≥70 mmHg (>8 years). Children who had sustained intracranial hypertension during the first day after admission (ICP 20–24 mmHg for 30 min, 25–29 mmHg for 10 min, 30 mmHg or more for 1 min) or had evidence of herniation (dilatation of one pupil or the presence of bradycardia) were eligible for randomization. Children were randomized to conventional medical management (control group) or decompressive craniectomy plus conventional medical management (decompression group). Randomization was performed with groups blocked at four. We used the Zelen method of randomization [46], with informed consent for surgery requested from a parent or guardian. Ethics approval for the study was obtained from the Hospital's Ethics in Human Research Committee.

We aimed to perform surgery within 6 h of randomization. A bitemporal craniotomy was performed in each patient via a bilateral vertical incision in the mid-temporal region. A disc of temporal bone measuring 3–4 cm was then removed from beneath the temporalis muscle on each side with an additional craniectomy extending the opening down to the level of the floor of the middle cranial fossa. The dura was left intact and, in a few cases, was scarified in a crisscross pattern using a 15 scalpel blade. This scarification resulted in minimal expansion of the dura and was therefore not practised as a routine. If and when the children recovered sufficiently, the cryo-preserved bone flaps were replaced.

Data collected consisted of age, the best motor response, Glasgow Coma Score (GCS), and pupil reactivity before paralysis. Brain injury was classified by a senior radiologist from computerized tomography (CT) radiographs according to the following criteria [29]: diffuse injury I – no visible pathology; diffuse injury II – cisterns are present, shift <5 mm and/or lesion densities present, no high- or mixed-density lesion >25 ml; may include bone fragments and foreign bodies; diffuse injury III (swelling) – cisterns compressed or absent, midline shift is 0–5 mm, no high- or mixed-density lesion >25 ml; diffuse injury IV (shift) – midline shift >5 mm, no high- or mixed-density lesion >25 ml; evacuated mass lesion (EML) – any lesion surgically evacuated; nonevacuated mass lesion (NEML)– high- or mixed-density lesion >25 ml, not surgically evacuated.

ICP and cerebral perfusion pressure were measured via a standard intraventricular catheter connected to a pressure transducer with measurements recorded hourly in the intensive care unit. The duration of stay in both the intensive care unit and the hospital was recorded at discharge. Using a standardized questionnaire (Appendix B), outcome status was evaluated 6 months after injury either in a telephone interview with each child's parent or guardian, or by chart review and questioning of the treating physician by a research assistant experienced in outcome evaluation (A.T.). We used a modified Glasgow Outcome Score (GOS) to obtain a functional outcome [8] and a Health State Utility (HSU) index (Mark 1) to obtain an assessment of quality of life [41]. Outcome categories for the modified GOS were defined as normal; functionally normal (both intellectually and physically) but requiring medication or medical supervision; mildly disabled but likely to lead an independent existence; moderately disabled and dependent on care; severely disabled and totally dependent on care (including children in a persistent vegetative state); and death. Children who were normal or functionally normal or had a mild disability were defined as having a favourable outcome; children who had a moderate or severe disability or had died were defined as having an unfavourable outcome. The nature of disability was classified as motor, cognitive or behavioural. The HSU index consisted of four categories, each representing possible levels of functioning in respect to physical function (mobility and physical activity); role function (self-care and role activity); social and emotional function (emotional and social activity); and health problems. All levels within each category are assigned a numerical value from which an overall health state utility value is calculated. All possible health states lie within a range of 1.00 to – 0.21, where 1.00 is healthy, 0 is dead, and negative values reflect a health state considered "worse than death" [41]. Four outcome categories were defined: good (HSUV 1.00–0.7); moderate (HSUV 0.69–0.3); poor (HSUV 0.29–0); and very poor (HSUV less than 0). Good was considered a favourable outcome, while categories moderate, poor and very poor were considered unfavourable.

The cause of death was classified as cerebral herniation or withdrawal of treatment for brain death or poor prognosis. Brain death was diagnosed according to standard criteria [4]. Poor prognosis was defined as the presence of two or more of the following confirmed by a neurologist, or in the case of a CT scan a radiologist, blinded to randomization: bilaterally absent somatosensory evoked potentials (SEP); diffuse hypodensity of the brain on CT scan ("black brain") and electroencephalographic (EEG) abnormalities known to be associated with poor prognosis, such as burst suppression, alpha coma or electrocortical silence in the presence of low anaesthetic drug levels (thiopentone, midazolam and diazepam). Statistical analysis was performed using StatXact 3.0 and Stata 5.0.

Results

Twenty-seven patients admitted to the intensive care unit between November 1991 and December 1998 were randomized. Fourteen children were randomized to medical management alone, and 13 children to decompressive craniectomy and medical management. They had a median age of 120.9 months (range 13.6–176.4 months).

In the control group, prior to paralysis there was a median GCS of 5 (range 4–9), a best motor response of nil response in 1 child, extension in 2 children, flexion in 10 children and localization to pain in 1 child. The pupils were reactive (plus or minus equal) in 11 children and fixed and dilated in 3 children. Brain injury was classified as diffuse injury II in 2 children, diffuse injury III in 6 children, diffuse injury IV in 1 child, and nonevacuated mass lesion in 5 children. In the decompression group, there was a median GCS of 6 (range 3–11). The 1 child who presented with a GCS of 11 had a large extradural haematoma and required ICP monitoring for extensive orthopaedic surgery 6 h after admission. Following this procedure, sustained intracranial hypertension was evident. The best motor response in the decompression group was nil response in 2 children, extension in 2 children, flexion in 6 children and localization to pain in 3 children. Pupils were reactive (plus or minus equal) in 12 children and fixed and dilated in 1 child. Brain injury was classified as diffuse injury II in 3 children, diffuse injury III in 8, and nonevacuated mass lesion in 2 children. The mean ICP in the control group, calculated from the last three pre-randomisation values, was 25.6 mmHg (SD 8.1, range 15–44), as against 26.4 mmHg (SD 7.9, range 16–41) in the decompression group.

Randomization occurred at a median of 16 h (range 3–29 h) after injury. In the control group the median time for randomization was 17.2 h (range 3–29 h). The median time for randomization in the decompression group was 15.0 h (range 6.3–23.2 h). Surgery was performed at a median of 17.3 h (range 6.5–27.5 h) from admission and 19.2 h (range 7.3–29.3 h) after injury. ICP following randomization in the control group, or following surgery in the decompression group, was calculated for 48 h in 11 of the 14 children in the control group and 11 of the 13 children in the decompression group. In 3 children in the control group and 1 child in the decompression group ICP monitoring was not possible for so long owing to their deaths before the 48-h period ended. ICP monitoring was discontinued in 1 further child in the decompression group because of intraventricular catheter malfunction. Individual ICP values for each group are displayed in Fig. 1. The results of ICP control for each group are shown in Table 1.

A two-sample *t*-test detected a mean difference between mean pre- and postoperative ICP of 8.98 mmHg (SD 6.60), 95% CI 4.987 – 12.968, in the decompression group and of 3.69 mmHg (SD 7.14), 95% CI –0.435–7.807, in the control group. The difference between the two groups was –5.29 [95% CI –10.75–0.170 (P = 0.057)]. When a two-sample *t*-test was performed following log transformation of the data a *P*-value of 0.083 was found. In the control group, ICP decreased after randomization in 10 children (71%), increased in 3 (21%), and remained unchanged in 1 (7%). Following log transformation, ICP decreased marginally in this child. In the decompression group, ICP decreased after

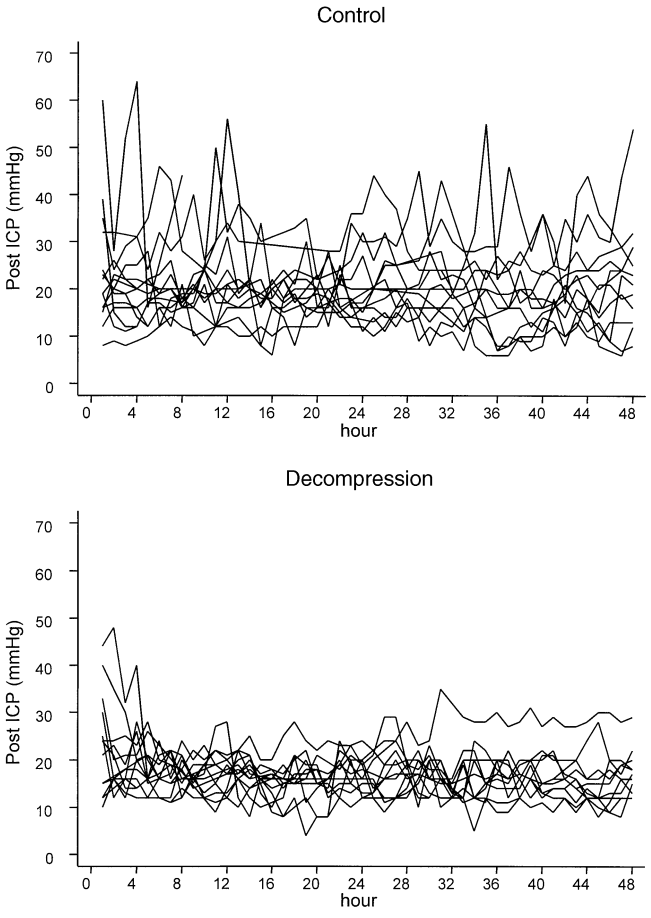

Fig. 1 Individual intracranial pressure curves for members of the control group and of the decompression group

Table 1 Intracranial pressure (*ICP*) control for 48 h after randomization in control group and 48 h after decompressive craniectomy in decompression group

	Control (n=14)	Decompression (n=13)
ICP (mmHg) mean	21.9 (SD 8.5)	17.4 (SD 3.4)
Range (mmHg)	11–44	11–25
ICP >20 mmHg Number of episodes	223	107
ICP >30 mmHg Number of episodes	59	9

Table 2 Outcome 6 months after injury

	Control (*n*=14)	Decompression (*n*=13)
Glasgow Outcome Score		
Favourable	2	7
Unfavourable	12	6
Health State Utility Index		
Favourable	1	6
Unfavourable	13	7

surgery in 12 children (92%) and increased in 1 child (8%). In children in whom ICP decreased, the mean reduction in ICP in the control group was 7.0 mmHg (SD 5.3), as opposed to 9.8 mmHg (SD 6.2) in the decompression group (two-sample *t*-test: *P*=0.26).

Kaplan-Meier survival analysis showed a trend towards a shorter time in intensive care in the decompression group (median of 9.6 days in intensive care, range 1.7–31.2 days) than in the control group (median 12.8 days, range 1.0–14.8 days), but the difference was not significant (Breslow-Gehan log rank test, *P*=0.12). The median stay in hospital was 26.8 days (range 13.8–73.3 days) in the decompression group and 47.7 days (range 21.9–73.1 days) in the control group (*P*=0.33).

The outcome status of the surviving children was evaluated a median of 5.9 months (range 4–16 months) after injury. One child in the decompression group was evaluated at 16 months, as telephone contact was not possible and the child failed to arrive to keep any follow-up appointments with the treating physician. The maximum time for outcome assessment in the remaining 26 children was 7 months in the control group and 7.5 months in the decompression group. The GOS and HSU scores are shown in Table 2. Two (14%) of the 14 children in the control group had a favourable outcome and 12 (86%) had an unfavourable outcome. Seven (54%) of the 13 children in the decompression group had a favourable outcome and 6 (46%) had an unfavourable outcome (two-tailed Fisher's exact test *P*=0.046; the *P*-value required for significance following repeated significance testing was <0.0221 [30]). Two children in the control group had a pre-existing disability: 1 child with mild motor and cognitive disabilities was found to have moderate disabilities of the same nature after injury, and the other child had mild cognitive and behavioural disabilities, which remained unchanged after the injury. One child from each group was classified as having a mild disability on the GOS but had a moderate outcome when the HSU was used. In the control group, 6 children had more than one disability. The nature of disability was motor on 7 occasions, cognitive on 6 occasions and behavioural on 3 occasions. In the decompression group, 5 children had more than one disability: motor on 4 occasions, cognitive on 6 occasions and behavioural on 1 occasion. Treatment was withdrawn in the control group following confirmation of brain death in 2 children and because of poor prognosis in 3 children; 1 child died as a result of cerebral herniation. In the decompression group, 3 children died as a result of withdrawal of treatment for poor prognosis. As a cumulative total, death did not occur less than 24 h after the injury in any child; it occurred less than 48 h after injury in 3 children and less than 1 week after injury in all of the 9 children who died.

Discussion

The concept of wide bone removal for the treatment of intracranial hypertension has been recognized since the nineteenth century [40], and a variety of surgical techniques have been described, including circular decompression and unilateral and bilateral craniectomy using a subtemporal or frontal approach. We chose the bitemporal craniectomy to promote decompression of the temporal lobes and achieve ICP control while reducing the degree of transtentorial herniation and upper brain stem compression. The dura was not opened, to avoid gross cerebral herniation and further injury to the brain. Historically, decompressive craniectomy has been employed as a final attempt to control intractable intracranial hypertension unresponsive to conventional therapy [17, 35]. We chose to perform surgical decompression early in the treatment phase of sustained intracranial hypertension, and to monitor its effects on both ICP control and outcome.

The results of this trial show that when children with TBI and sustained intracranial hypertension are treated with a combination of early decompressive craniectomy and conventional medical management ICP is reduced and fewer episodes of intracranial hypertension occur. The results suggest that with craniectomy functional outcome and quality of life may be improved over those obtained in children treated with medical management alone. These encouraging results indicate that even earlier decompressive craniectomy may be advantageous.

However, there were a number of problems with this study: the sample for the trial was small, the trial ran over 7 years, outcome evaluation was performed early in the recovery phase following traumatic brain injury, the process of outcome evaluation did not include a face-to-face interview with the children and their families, and a statistical analysis was performed on the outcome data twice during the last 6 months of the trial.

Over the last decade improved road safety measures, including speed cameras, random breath testing, compulsory bike helmets, lowering of freeway speed limits and improved road systems have facilitated a reduction in road fatality and serious injury in our region [42]. A reduction in the number of children admitted to our intensive care unit with severe TBI reduced the number of

children eligible for the study; for this reason it took 7 years to achieve a sample size of 27.

A number of changes were made in our management of children with TBI during this trial, including changes to the definition of adequate cerebral perfusion pressure, less aggressive hyperventilation, and changes to hypothermia regimens and fluid management. Although these changes were not anticipated in the design phase of the trial, randomization in blocks of 4 was used to reduce the effect of any change to medical management if and when it occurred.

Evaluation of outcome at 6 months after injury provided a global picture of outcome status; however, a change in outcome over time in children with traumatic and nontraumatic brain injury has been noted in another study [9], where a difference in outcome between 1 and 5 years after injury was noted in 42.5% of children. Eight of the 17 children surviving after TBI improved, 2 deteriorated, and 7 remained unchanged. In our study outcome was evaluated 6 months after injury, and it is likely that outcome status will be different at 5 years.

We used either telephone interview or chart review and questioning of the treating physician to evaluate outcome; children were not assessed in person. The reliability of outcome assessment through telephone interview is well established [26], and our intensive care unit has performed over 3,000 outcome evaluations using this method over the last 10 years.

As further changes in the management protocol now seem necessary, with the possible use of transcranial doppler, hypothermia and jugular bulb oximetry, it seemed important for the trial to be completed. This prompted us to perform a statistical analysis on the outcome data twice during the last 6 months of the trial prior to the final analysis. An adjusted *P*-value <0.0221 was then needed for the results to be statistically significant. The results of this study therefore have to be regarded as only preliminary findings. There is a need for a large multi-centre randomized control trial to assess the effect of decompressive craniectomy following TBI.

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Appendix A: Protocol for the management of head injury [7, 39][1]

A. Ventilation

1) Instituted for all patients who present with:
   a) Flaccid or extensor (decerebrate) or flexor (decorticate) posturing
   b) Or deteriorating conscious state
   c) Or respiratory failure
2) Ventilation settings should aim to:
   a) Maintain a P_aCO_2 of 35–40 mmHg
   b) And maintain a P_aO_2>80 (preferably 100) mmHg
   c) And provide PEEP of 3–5 cmH_2O

B. Paralysis / sedation / analgesia

Following initial neurological assessment:

1) Paralysis
   a) All patients until verification of head injury status is obtained through CT scan
   b) All patients immediately following ICP (intracranial pressure) catheter insertion for evaluation of ICP
   c) All patients with sustained intracranial hypertension for 24 hours from the time of injury
   d) Use pancuronium: 0.1–0.15 mg/kg p.r.n.
2) Sedation
   a) Diazepam 0.1 mg/kg i.v. 4-hourly
   b) Give extra i.v. boluses of diazepam (0.05 mg/kg) if there are autonomic signs of response to stimuli eg. tachycardia, increased blood pressure or lacrimation on passive movement of limbs
3) Analgesia
   a) Morphine by i.v. infusion at 40–80 mcg/kg/h
   b) Morphine bolus of 50 mcg/kg i.v. before painful procedures e.g. turning a child with fractures

C. Fluids

1) Correct hypovolaemia: i.v. colloid (5% albumin in normal saline), bolus dose of 5–10 ml/kg p.r.n.
2) Provide maintenance hydration:
   a) Intravenous fluid requirements: give normal saline with KCl 40 mmol/l at the following rate (assuming a total of 5 ml/h for morphine + IA + inotrope + dextrose infusions).

Wt (kg)	4	6	8	10	12	14	16	20	30	40	50	60	70
ml/h	5	9	13	17	20	23	27	30	34	40	44	47	50

   b) Maintain urine output at ≥1.0 ml/kg/h
3) Maintenance of biochemistry
   a) Serum sodium 140–150 mmol/l.
   Give 3 ml/kg of 3% saline i.v. over one hour, then 0.5 ml/kg/h if sodium <140 mmol/l
   b) Normoglycaemia: start 50% dextrose at 0.5 ml/kg/h and adjust to maintain blood glucose at 4–8 mmol/l. Avoid hyperglycaemia (blood glucose >8 mmol/l).

D. ICP monitoring (via intraventricular catheter)

1) Instituted for all patients who have:
   a) A Glasgow Coma Score (GCS) <9
   b) Or extensor or flexor posturing or flaccidity
   c) Or a swollen brain at craniotomy
   d) Or nonpurposeful movements, posturing or flaccidity and require a prolonged surgical procedure, e.g. laparotomy or an orthopaedic procedure
2) Device set-up/diagram:
   see Procedure Manual – Intracranial Pressure Monitoring

[1] Protocol developed 1 August 1998 by the Departments of Intensive Care and Neurosurgery

3) General principles of management:

a) The arterial pressure transducer and ICP transducer are zeroed at the level of the external acoustic meatus to allow ICP and CPP (cerebral perfusion pressure) to be measured from the same baseline.
b) Venting of cerebrospinal fluid (CSF) is regulated by the height of attachment of the drainage burette (at the level of the drip point) above the level of the external acoustic meatus. This level is determined by the ICP level prescribed for venting (mmHg) × 1.36 = the height of the burette in centimeters above the external acoustic meatus, e.g. 20 mmHg × 1.36=27.2. Thus, the top of the burette (at the level of the drip point) is secured 27.2 cm above the external acoustic meatus to drain at 20 mmHg.
c) When continuously venting, the ICP should only be measured after the venting has been turned off at the distal 3-way tap for 20 seconds.
d) Daily culture of CSF:
See Procedure Manual – Intracranial Pressure Monitoring – for procedure guidelines.

4) Treatment of raised ICP

The primary objective is maintenance of an adequate CPP = mean arterial pressure minus mean ICP. Adequate CPP is defined as:
Neonate: >30 mmHg
1 month – 1 year: >40 mmHg
1 year – 4 years: >50 mmHg
5 years – 8 years: >60 mmHg
8 years and over: >70 mmH
Maintenance of adequate cerebral perfusion is achieved through:
a) Reducing ICP:
Treatment aim: ICP ≤20 mmHg.
Treatment regimen:
i) Moderate hyperventilation via hand bagging for no longer than 2 minutes only if there is cerebral herniation or an ICP >40 mmHg
ii) Mannitol 0.25–0.5 g/kg/dose i.v. (2–4ml/kg of 12.5%, 1.25–2.5 ml/kg of 20%) Precautions: ensure serum osmolality is no greater than 320 mosmol to prevent risk of renal failure, and do not give more than three doses of mannitol per 24 hours due to the risk of cerebral accumulation of mannitol and potentiation of cerebral oedema.
iii) Intermittently vent CSF for 1 minute
iv) Continuously vent CSF (inform neurosurgical team)
v) Thiopentone. Avoid hypotension. Slow bolus dose of 1–5 mg/kg i.v. (maximum of 1 mg/kg/min with not more than five doses recommended, depending upon blood pressure response) – then intravenous infusion: 1–5mg/kg/h via central venous catheter. Level: 150–200 ummol/l (× 0.24 = mcg/ml); Biochemistry Department to be notified of request for thiopentone levels before blood sampling.
b) Increasing mean arterial pressure:
a) Ensure normovolaemia
b) Commence dopamine at 5–15 mcg/kg/min i.v. (especially if a thiopentone i.v. infusion is in progress or if cardiac echo demonstrates decreased ventricular function)
c) Add noradrenaline 0.05–0.5mcg/kg/min i.v. if required.

E. Cervical collar and use of sand bags

1) Use sand bags beside the head and a cervical collar on all patients who:

a) Fail to move all limbs spontaneously.
b) Have a normal lateral cervical spine X-ray but the whole spine from occiput to T1 is not seen.
c) Have an abnormal lateral cervical spine X-ray including fracture, subluxation or soft tissue abnormality.
d) Complain of neck pain.

2) The collar should be changed after 24 hours from a “Stifneck” to a “Philadelphia” variety. These are available on request from Orthotics.
3) Collar and sand bags may be removed from patients who have a normal lateral cervical spine X-ray visualizing C-1 to T-1. Neurosurgeon’s consent is required before removal of collar.

F. Induced hypothermia

1) Commenced on all patients from admission who present with flaccidity, extensor or flexor posturing.
2) Patients are ventilated, paralysed and cooled to 33 degrees celsius by means of a cooling blanket.
3) Hypothermia is maintained for 24–36 hours only
4) Rewarming is achieved gradually via a servo-controlled warming/cooling blanket. The patient’s core temperature should be allowed to rise 1 degree every three hours up to 37 degrees.

G. Prophylactic antibiotics

1) Penicillin 30 mg/kg/dose 4- to 6-hourly i.v. for all patients who present with a compound skull fracture
2) Antibiotics are NOT required in the following instances unless requested by the neurosurgeon:

a) Children with a fractured base of skull opening into the nose, middle ear or paranasal sinus
b) Children with an externally draining intraventricular catheter

H. Anticonvulsants

1) The drug of choice is phenytoin (Dilantin).
a) Loading dose: 15–20 mg/kg (maximum 1.5 g) i.v. over one hour.
b) Maintenance dose:
1st week of life – 4 mg/kg/dose 12-hourly i.v.
2nd week of life – 4 mg/kg/dose 8-hourly i.v.
3 weeks to 5 years – 4 mg/kg/dose 6-hourly i.v.
5 years to 9 years – 4 mg/kg/dose 8-hourly i.v.
>12 years – 2 mg/kg/dose 6- to 12-hourly i.v. (maximum 100 mg).
c) Level 40–80 µmol/l (×0.25 = mcg/ml)
Can be performed any morning by Biochemistry Department

2) Monitor for and document all suspected seizure activity.

Bi-temporal EEG monitoring should be performed on all paralysed patients.

I. Nutrition

1) Enteral feeding should be commenced only when hypothermia is stopped.
2) Naso-jejunal (NJ) feeding is the preferred mode of feeding.
3) Recommended preparations for NJ feeding:

a) Begin with an elemental (pre-digested) formula:
Under 2 years: Neocate (iso-osmolar 15% concentration)
Over 2 years: Paediatric Vivonex (iso-osmolar 19% concentration)

b) Once tolerated a whole-protein formula can be introduced:
Under 2 years: maintenance infant formula
Over 2 years: Osmolite (iso-osmolar)

4) Recommended preparations for NG feeding:
Under 2 years: maintenance infant formula
Over 2 years: Osmolite (iso-osmolar)
Ensure Plus (hyperosmolar) if higher caloric intake required

5) If diarrhoea occurs investigate all potential causes before stopping feeds.

J. Positioning

All patients should be:

1) Nursed with head elevated 10 degrees.
2) Maintained with their head in a neutral position with all flexion, extension, lateral flexion and rotation avoided during turning.
3) Log-rolled from side to side every 2–4 hours and maintained at a 45 degree angle. The patient's head and shoulders should be supported throughout the turn.
4) Placed on an air mattress at the time of admission. Cooling blankets should be placed under the trunk only so that pressure relief is available to the head at all times.

K. Specific diagnostic measures

1) **EEG** performed on day 2 post injury.
2) **CT** scan of the head performed at the time of admission, upon subsequent deterioration in neurological condition and at the discretion of the intensivist or neurosurgeon.
3) **SEP** (somatosensory evoked potentials) performed on day 2 post injury.

Appendix B: Outcome evaluation

1. Mobility/physical activity

a) Does your child have any limitations in regard to walking/running/jumping? **Yes/No**. If yes, please specify.
b) Does your child experience any unexpected breathlessness/tiredness when playing with other children or exercising? **Yes/No**. If yes, please specify.
c) Is help required from other people, or from mechanical aids (wheelchair, frame) for your child to move around? **Yes/No**. If yes, please specify.
d) Parents' perceptions of child's physical abilities in relation to other children of a similar age. Please specify.

2. Self-care/role activity

a) How much help does your child need to eat/dress/bathe/toilet (as age appropriate).
Please circle the most appropriate.
1. No help
2. A small amount of help
3. A moderate amount of help
4. Is totally dependent upon help from another person.

b) Which of the following does your child attend
1. Kindergarten
2. School: year _____
3. None

c) Does your child need any of the following support
1. Integration aide
2. Special needs school
3. Rehabilitation (including physiotherapy, occupational therapy, speech therapy)
4. None

d) If your child attends school, how much school would your child miss throughout the school year?
1. Less than one week
2. Between one and two weeks
3. Between two and four weeks
4. More than a month

e) Parents' perceptions of child's ability to cope with school level.

3. Social/emotional function

a) During an average day is your child generally
1. Happy
2. Anxious
3. Depressed
4. Aggressive

b) Does your child have any problems in making and maintaining friendships ? **Yes/No**.
c) Parents' concerns with respect to child's behaviour. Please specify.

4. Health problems

a) Has your child developed any new health problems since your admission to hospital in ——? (Royal Children's Hospital, Melbourne). **Yes/No**
If yes, please specify.
b) Has there been any change in the management, or the severity of, those health problems that your child experienced prior to admission to hospital in ——? **Yes/No**
If yes, please specify.
c) Does your child require regular follow-up by a specialist doctor? **Yes/No**
d) Does your child require regular medication? **Yes/No**
If yes, please specify.
e) Does your child experience any pain/discomfort on a regular basis? **Yes/No**
Specify location and frequency.
f) Does your child have any vision problems? **Yes/No**
Does your child need to wear glasses? **Yes/No**
g) Does your child have any hearing problems? **Yes/No**
Does your child need to wear a hearing aide or communicate using sign? **Yes/No**
h) Does your child have any scars that have healed poorly, or have any other physical problems that cause your child, or you, concern? Specify.
i) Additional comments.

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