

Severe traumatic brain injury in children – are the results improving?

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Summary

Questions under study: Traumatic brain injury (TBI) remains an important cause of mortality and morbidity in children. Medical management is constantly being refined, and thus results should improve. The aim of the present study was to analyse our data of recent years and to compare them with previous series (1978–83 and 1988–92).

Patients and methods: The data of 51 children (1 month to 16 years old) with severe blunt TBI treated in our unit from 1994 to 1998 were analyzed retrospectively. Severe TBI was defined by immediate loss of consciousness and an admission Glasgow coma scale (GCS) <8. Outcome was classified by using the Glasgow outcome scale (GOS) 6 to 12 months after injury.

Results: 35 patients (69%) showed a good outcome (GOS 4 and 5), 14 died (GOS 1), one survived in a permanent vegetative state (GOS 2), and another was severely disabled (GOS 3) (GOS 1–3 = bad outcome, 31%). Bad outcome was associated with low GCS (i.e. 3 and 4), fixed and dilated pupils at admission, invisible basal cisterns on first com-

puterized tomography, and presence of coagulopathy. Moderate to severe intracranial hypertension was also significantly related to bad outcome in the 26 patients with intracranial pressure monitoring. Compared to our first series severity of TBI was unchanged, and the incidence of multiple injury and consumption coagulopathy was less frequent. Intubation rate prior to admission to the centre increased from 35% to 94%. Intensive care measures (duration of mechanical ventilation, use of hypothermia, mannitol, thiopentone etc.) were less aggressive. The rate of good outcome remained unchanged (69% vs. 60%).

Conclusions: Despite changing management policies, results were comparable with those of our former series. This fact underlines the importance of primary injury and the secondary role of intensive care management on final outcome.

Key words: head injury; multiple injury; computerized tomography; consumption coagulopathy; intracranial pressure

Introduction

Traumatic brain injury (TBI) remains a major cause of mortality and morbidity in children. In the USA, children up to 15 years make up more than half of all new TBI cases with an annual incidence rate of 185 per 100 000 children [1]. For Europe

and Switzerland corresponding data are lacking. Although the majority of TBI (85.5%) was classified as mild, there was a case fatality rate of 6%. Twenty-one (52.5%) deaths occurred at the scene or before hospital admission and 19 in the hospital [1]. After primary injury, surgical management aims to prevent secondary brain ischemia and herniation by compressive hematomas. However, debates continue about best and necessary monitoring and medical treatment modalities, and therefore protocols might change over time [2]. The aim of the present study was therefore to gain data of recent years (1994–98) and to compare them with the former periods of 1978–83 and 1988–92 [3, 4].

List of abbreviations used:

CT	Computerized tomography
GCS	Glasgow coma scale
GOS	Glasgow outcome scale
ICP	Intracranial pressure
ICU	Intensive care unit
TBI	Traumatic brain injury

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Patients and methods

All patients with severe blunt TBI admitted to the intensive care unit (ICU) of the University Children's Hospital at Bern, Switzerland, were reviewed for the time period 1.1.1994–31.12.1998. Inclusion criteria for the present report were: 1. age 1 month to 16 years; 2. severe blunt TBI with immediate loss of consciousness and persistence of coma (Glasgow coma scale (GCS)<8) at least until and during admission to our hospital; and 3. admission to our medical centre within 6 h after injury.

Management: In all patients tracheal intubation had been performed at the scene of the accident or during hospital admission, and all were on mechanical ventilation. After initial cardio-respiratory stabilisation in the emergency department, computerized tomography (CT) of the skull was performed and relevant space occupying lesions were evacuated immediately. Placement of an intracranial pressure (ICP)-monitoring device (Gaeltec epidural transducer or Camino intraparenchymal transducer) was decided by the neurosurgeon in charge. In general, a GCS<6 and/or evidence of raised ICP were considered as an indication. All patients had arterial catheters placed for monitoring arterial blood pressure and blood gases. Main emphasis during intensive care was placed on respiratory care (slight to moderate hypocapnia, moderate hyperoxemia), haemodynamic support in order to achieve sufficient cerebral perfusion pressure (>50 mm Hg in children <5 years of age, and >70 mm Hg in older children), anti-convulsive treatment, and hydro-electrolytic and thermal maintenance. Initial intravenous maintenance fluid was administered in form of 0.9% saline, amounting to 80% of normal maintenance. Means to reduce intracranial hypertension, after exclusion of surgically correctable space

occupying lesions by repeated CT, included diuretics, hypertonic saline, hypocapnia, and barbiturate coma [5, 6]. The goal was to keep ICP <20–25 mm Hg, and, if the jugular bulb was catheterised, a jugular venous oxygen saturation of 55–75% was considered to be ideal [7].

Data analysis: The records of all patients fulfilling study criteria were analysed for age, cause of TBI, presence of multiple injury, cardiovascular instability, and GCS (15 points scale) at time of admission to hospital. Multiple injury was defined by TBI and life threatening injury of viscera, pelvis or major long bones, as previously described [8]. Pupillary reactions at admission were classified as bilaterally fixed and dilated vs. all other possibilities. Initial CT scans were analysed for presence of haematomas (epidural, subdural or intraparenchymal; operated or not operated), haemorrhages, and visibility of basal cisterns. Coagulation data were classified as normal/slightly abnormal (prothrombin time $\geq 60\%$ of normal, partial thromboplastin time <60 s, fibrinogen $\geq 1\text{g/l}$), or moderate to severe consumption coagulopathy (i.e. worse coagulation profile without or with abnormal bleeding tendency) [3]. ICP was analysed with regard to "maximum mean sustained ICP" which had to be on the same level for at least 30 min. Outcome was judged according to the Glasgow outcome scale (GOS) 6 to 12 months after injury: GOS 1 (dead), GOS 2 (persistent vegetative state) and GOS 3 (severely disabled) were summarised as bad outcome, as opposed to GOS 4 (moderately disabled) and GOS 5 (good recovery) which were summarised as good outcome [9]. For statistical analysis Fisher's exact probability, Chi-square, and Student's t-tests were used. P-values <0.05 were considered to be significant.

Results

During the 5 years under review 2451 patients aged 1 month–16 years were admitted to ICU, 243 suffering from blunt TBI. Fifty-one children fulfilled inclusion criteria (32 boys, 19 girls). Mean accident-admission interval was 2 hours (range 0.3–4.8 h). Mean age was 8.1, median 7.0, and range 0.2–16.1 years. Seventeen children were ≤ 5

years of age with 10 traffic accidents, 6 falls, and 1 child abuse as causes of TBI. Thirty-four patients were >5–16 years of age with 22 traffic accidents, 7 falls, and 5 sport accidents as causes. Distribution of bad outcomes with regard to cause of TBI was as follows: traffic accidents, 11; falls, 4; sport accident, 0; child abuse, 1. Tracheal intubation was

Table 1

Clinical and computerized tomography findings and outcome (n = 51).

	Good outcome (n = 35)	Bad outcome (n = 16)	P-value
Age ≤ 5 years	11	6	
>5 years	24	10	n.s.
Multiple injury	4	2	n.s.
Cardiovascular instability	3	5	n.s.
Glasgow coma scale 3 + 4	8	10	
5–7	27	6	p <0.01
Both pupils fixed and dilated	0	8	p <0.001
Epidural haematoma operated / not operated	3 / 0	2 / 0	n.s.
Subdural haematoma operated / not operated	2 / 7	2 / 3	n.s.
Intraventricular, intraparenchymal and subarachnoid haemorrhage	26	10	n.s.
Perimesencephalic cisterns not visible in CT	1	8	p <0.001

performed in 28 cases at the scene of the accident, in 20 at the referral hospital, and in 3 in our emergency department. Multiple injury was present in 6 cases, all traffic accidents. The following distribution was found with regard to GCS: GCS 3 in 15 patients, GCS 4 in 3, GCS 5 in 13, GCS 6 in 10, and GCS 7 in 10. Six patients showed a combination of GCS 3 and bilaterally fixed mydriasis, 4 of these suffered in addition from cardiovascular instability. Only in 3 (6%) instances initial CT was interpreted as normal; in the vast majority (48 scans or 94%) one or more abnormalities were found. Main admission data including CT findings and outcomes are summarized in table 1.

Admission coagulation data were available in 50 cases, results are summarized in table 2.

Table 2
Admission coagulation data (n = 50).

	Patients with good outcome (n = 35)	Patients with bad outcome (n = 15)	P-value
Coagulation normal/slightly abnormal	32	6	
Moderate consumption coagulopathy	3	2	p < 0.001
Severe consumption coagulopathy	0	7	

ICP was monitored in 26 (51%) patients (for results see below). In 5 cases with GCS 3 and fixed and dilated pupils, ICP monitoring was considered to be futile and therefore not done. In a 2.5 years old girl and a 12 years old boy, both with fatal course, ICP was not monitored because a large decompressive craniectomy had been performed to evacuate epi- and subdural haematomas. Jugular bulb catheterisation was performed in 17 (33%) patients. Maximum mean sustained ICP was as follows in patients with good outcome: 8 patients remained ≤ 19 mm Hg, 8 were between 20 to 40 mm Hg (moderate intracranial hypertension), and 1 was ≥ 41 mm Hg (severe intracranial hypertension); whereas 7 children with bad outcome stayed between 20–40 mm Hg and 2 above 40 mm Hg. Mean sustained ICP ≥ 20 mm Hg was significantly associated with bad outcome (p < 0.05).

Main data of intensive care are shown in table 3. In no case hypothermia or decompressive craniectomy were performed in order to control ICP. Diabetes insipidus was diagnosed in 5 patients (GOS 1, 4 patients; GOS 4, 1 patient; p < 0.05). Syndrome of inappropriate ADH secretion occurred 3 times (GOS 1, 1 patient; GOS 4 and 5, 2 patients). Good outcome was observed in 35 patients (GOS 5, 17 patients; GOS 4, 18 patients),

Table 3
Intensive care data (n = 51).

	Patients with good outcome (n = 35)	Patients with bad outcome (n = 16)	P-value
Mean (range) duration of mechanical ventilation (d)	3.5 (0.1–22.3)	2.1 (0.1–7.7)	n.s.
Mean (range) duration of ICU stay (d)	5.6 (0.7–30.8)	2.4 (0.1–8.9)	p < 0.01
Catecholamines	8	13	p < 0.001
Mannitol	8	5	n.s.
NaCl 2.5%	2	1	n.s.
Thiopentone	2	1	n.s.

Table 4
Comparison of 3 periods of neurointensive care.

	1978-83	1988-92	1994-98	P-value
Nr. of cases of TBI admitted	187	236	243	
Nr. of severe TBI (GCS < 8)	40	57	51	n.s.
Mean age (years)	7.5	6.6	8.1	n.s.
Traffic accidents (%)	75	54	63	n.s.
Multiple injury (%)	45	21	12	< 0.001
Moderate to severe coagulopathy (%)	67	51	24	< 0.001
Admission GCS 3+4 (%)	43	28	36	n.s.
Pupils fixed and dilated at admission (%)	20	16	16	n.s.
Tracheal intubation before admission (%)	35	77	94	< 0.001
Intracranial haematoma operated (%)	22	18	18	n.s.
Invisible basal cisterns on first CT (%)	28	17	18	n.s.
Mean duration of mechanical ventilation (days)	8.9	4.3	3.1	< 0.001
Intracranial pressure monitoring (%)	95	53	51	< 0.001
Thiopentone infusion (%)	60	35	6	< 0.001
Good outcome overall (%)	60	63	69	n.s.

CT = Computerized tomography; GCS = Glasgow coma scale; TBI = traumatic brain injury. Statistical analysis compares 1978–83 vs. 1994–98.

whereas a bad outcome occurred in 16 cases (GOS 3, 1 patient; GOS 2, 1 patient; GOS 1, 14 patients). The later patients all died due to severe brain injury with fatal brain swelling. Formal criteria of brain death were fulfilled in 10 cases, whereas in 4 cases supportive treatment was withdrawn (all with bilateral loss of N₂₀-components of somatosensory evoked responses). A 7 year-old boy in whom supportive care was also stopped, survived in a permanent vegetative state.

A comparison of the present series with our

former one is presented in table 4. Severity of TBI judged by the proportion of patients with GCS <5, bilaterally fixed and dilated pupils, and initial CT findings remained unchanged. Since the first period of 1978–83 the incidence of multiple injury and moderate to severe coagulopathy have decreased significantly, the rate of tracheal intubation before admission to the medical center has nearly tripled, and neurointensive care measures were significantly less aggressive. Outcome remained unchanged.

Discussion

With the present third series we now have studied 148 children with severe blunt TBI, accounting constantly for 20–25% of all cases of head injuries admitted to our ICU. Over the years admission criteria have remained the same, however, the retrieval area may have been enlarged mainly towards the French speaking part of Switzerland. Although management aspects have changed significantly over time, results have remained surprisingly constant, with one third of patients showing a bad outcome. Since our results have not improved despite our presumably better care, the two major possibilities are: 1. better care at the scene has allowed more severe cases to enter the hospital (see below), or 2. (more likely) the complexity of traumatic and hypoxic-ischemic lesions are not amenable to the refinements of intensive care medicine. In fact, we know from experimental animals that ischemia-reperfusion events and cell death are much more complex than expected. Thus, even the best intensive care support adds little to the biomolecular events postinjury [10]. However, beside these fundamental thoughts detailed aspects are to be considered too. Several factors are known to influence outcome after TBI including age, mechanism of injury, severity of mechanical impact, presence of multiple injury, and concomitant hypoxic or ischemic insults. The impact of medical and surgical management is less clear [2, 5]. Hypoxic-ischemic events might be reduced, but aggressive interventions tend also to increase medical complications [11].

In our first series age ≤5 years was associated with a worse outcome, but this was not confirmed by the third series. In a recent study of 477 paediatric head trauma patients (not confined to severe TBI) age ≤1 year but not ≤5 years was associated with increased mortality rates, most likely due to a high proportion of child abuse as mechanism of TBI in the infant age group [12]. In all our series child abuse played only a very minor role.

Severity degrees of TBI were most likely equally distributed in our three periods. The proportion of patients admitted with GCS <5, bilaterally fixed and dilated pupils, invisible perimesencephalic cisterns in first CT, and space occupy-

ing haematomas remained unchanged over time. However, some contradiction may arise from our coagulation data. In the last series less patients suffered from coagulopathy. This may suggest that TBIs were less severe in this series. It has been speculated that the coagulation cascade is triggered by myelin basic protein which has been shown to be severely elevated in patients with fatal TBI [13, 14]. Furthermore the value of coagulation studies for outcome prediction after severe TBI has been shown in all our 3 series, and also by other authors studying coagulatory function after severe isolated TBI [15, 16]. On the other hand, decreased incidence of multiple injury may be the reason for less frequent coagulopathy in the last series; or better prehospital care (early intubation, use of 0.9% saline) has stopped the vicious circle of cerebral hypoxia and sustained entry of myelin basic protein into the bloodstream.

In all our series multiple injury was nearly exclusively found in victims of traffic accidents. Speed limits and other traffic safety measures help to reduce incidence and severity of road accidents and thus multiple injury. Despite an enormous increase in traffic density the number of deaths due to traffic accidents has decreased in Switzerland from 1268 in 1978 to 597 in 1998 (Federal Office for Statistics). The role of multiple trauma for outcome after severe TBI is controversial: whereas Chestnut et al. found a poorer outcome in patients with severe TBI and multiple trauma [17], Sarrafzadeh et al. were able to show that after initial stabilisation in the ICU outcome was not influenced by multiple injury [18].

The devastating effects of hypoxia/ischemia after TBI are well known, and considerable efforts have been undertaken in the prehospital phase to avoid such complications. This is documented by the ever increasing intubation rate of children transported to our centre. As shown by Suominen et al., early and especially “field” intubation may improve results after severe paediatric TBI [19]. Together with improved prehospital respiratory care, there has also been a change in policy of fluid administration: in the last years all patients have been admitted with infusions of 0.9% sodium

chloride, compared to less sodium-rich solutions (Ringer's lactate etc.) in previous years [20].

Management of severe TBI in our ICU has changed considerably over the past two decades and moved to less aggressive intensive care. Steroids, severe hypocapnia, and hypothermia have been abandoned, and a tenfold decrease in thiopentone infusion has been realized. ICP was monitored less frequently, and duration of mechanical ventilation was much shorter in the last series. The use of mannitol has decreased from around 40% in the first two series to 25% in the third, however, in the third series 6% of patients received 2.5% sodium chloride in order to improve cerebral perfusion pressure and jugular bulb venous saturation. These changes in management over time are representative of our uncertainty about best treatment modalities. The lack of evidence based data is also expressed by Segal et al. who surveyed recently ICP monitoring and neurointensive care practices in the United Kingdom and were able to show wide variations in management policies [21].

Injury to admission time intervals and in-/exclusion criteria have a major impact on reported results. Death rate falls nearly exponentially after trauma [1, 3, 22], therefore series with big time in-

tervals between accident and admission to the medical centre will help to select the good cases [23]. In all our series that time interval has remained constantly short. Another important aspect is the in- or exclusion of patients who are admitted in an obviously brain dead state (i.e. GCS 3 with bilaterally fixed and dilated pupils). In our series we have always included these patients, because our criterion was admission to ICU. Would these patients have been excluded good outcome would have reached 78% in the last period.

In conclusion, we are left with the sobering fact that despite constant refinements in management one third of children shows a bad outcome after severe TBI. Therefore we have to realize that our interventions directed against secondary hypoxic-ischemic injuries have most likely a limited impact on final outcome, but that the primary injury plays the crucial role.

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