Outcome from severe head injury in children and adolescents

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A consecutive series of 37 children (17 years old and under) with severe head injury is presented. The data confirm that morbidity and mortality are lower in children than in adults: 51% of these young patients had a good recovery or moderate disability at 6 months. The mortality rate in this series (33%) is higher than in some reports, but probably more closely approximates the death rate from these injuries in an unselected pediatric population than do statistics from tertiary care hospitals. There was no significant relationship between age and outcome in this age group, but mass lesions and uncontrolled intracranial hypertension adversely affected outcome. Diffuse cerebral swelling was commonly seen on computerized tomography scans, and generally was associated with a satisfactory outcome (75%). Two of 13 deaths were considered preventable, emphasizing the narrow therapeutic safety margin and extreme care required in treating these patients.

KEY WORDS • brain injury • intracranial pressure • hematoma • hyperemia • coma • children

Previous reports have shown that outcome after severe head injury is better in children than in adults, with mortality rates as low as 6% in one pediatric series. However, mortality rates may be affected by inadvertent patient selection that occurs when unknown or inconstant referral patterns preclude treatment of some patients in a given population or region. For example, critically injured patients who are transported to the nearest hospital for stabilization and die before reaching the appropriate trauma center would not be included in the patient group from which outcome statistics are derived. In San Francisco, some of these selection problems are avoided because its geographical area is small and almost all major trauma victims are brought rapidly to San Francisco General Hospital Medical Center (SFGH). This unusual centralization of trauma care allows us to examine outcome in virtually all severely head-injured children and adolescents in a well defined metropolitan area.

Clinical Material and Methods

We reviewed all cases of severe head injury in patients younger than 18 years of age who were treated at SFGH between 1977 and 1982. Data were recorded prospectively using a standardized format for subsequent computer analysis. Patients who remained comatose — defined as a Glasgow Coma Scale (GCS) score of 8 or less — for at least 6 hours were included. Outcome was determined at least 6 months after injury and included the following categories: good recovery (resumption of preinjury activities with no or minimal neurological deficit or apparent change in personality or school performance); moderate disability (able to function independently at a reduced level because of personality, intellectual, or physical differences compared with preinjury status); severe disability (unable to function independently and requiring substantial care at home or in an institution as a result of intellectual or physical impairment); vegetative state; and death.

The neurological data reported here were obtained at assessment in the emergency room after resuscitation. The GCS score was derived from the patient’s best response in each of three categories (eye-opening, motor, and verbal responses), which were scored and summed; a total score of 3 represents the most severe injury and 15 the least severe. Until March, 1979, plain skull x-ray films, ventriculography, and angiography were used as indicated to evaluate pediatric head-injury patients. Since then, these diagnostic procedures have been replaced by computerized tomography (CT).

All patients were managed according to a standard
Severe head injury in children and adolescents

TABLE 1

<table>
<thead>
<tr>
<th>Pediatric head-injury protocol*</th>
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<tr>
<td>early CT scan (since 1979)</td>
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<tr>
<td>early surgery for mass lesions causing a midline shift &gt; 5 mm</td>
</tr>
<tr>
<td>intensive care management (as required):</td>
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<tr>
<td>- intubation &amp; controlled ventilation to obtain PaCO2 &lt; 25 mm Hg</td>
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<tr>
<td>- nasogastric suction &amp; antacids</td>
</tr>
<tr>
<td>- IV maintenance fluids at ⅔ normal amount</td>
</tr>
<tr>
<td>- ICP monitoring (intraventricular or subdural catheter, or subarachnoid screw)</td>
</tr>
<tr>
<td>- mannitol, furosemide administration</td>
</tr>
<tr>
<td>- sedation, paralysis induced</td>
</tr>
<tr>
<td>- barbiturate-induced coma not used</td>
</tr>
<tr>
<td>- dexamethasone before 1981, none since</td>
</tr>
<tr>
<td>- oral feedings beginning 3 to 5 days after injury</td>
</tr>
<tr>
<td>- electrolyte &amp; urine output monitoring twice daily for 7 days, then once daily</td>
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</tbody>
</table>

* CT = computerized tomography; IV = intravenous; ICP = intracranial pressure.

pediatric head-injury protocol (Table 1). Significant intracranial hematomas were promptly evacuated in all patients. Compound skull fractures were debrided and irrigated with bacteriocidal solutions, and depressed fragments were elevated and wired in place. Skull fragments depressed less than 5 mm were generally treated without surgery.

Intensive care procedures included endotracheal intubation to protect the airway and to allow controlled ventilation. Intracranial pressure (ICP) was monitored with an intraventricular or subdural catheter or with a subarachnoid screw device. Hyperventilation to achieve a PaCO2 of 20 to 30 mm Hg was instituted when ICP rose toward 20 mm Hg. If ICP remained above 20 mm Hg for 3 to 5 minutes, mannitol (0.5 to 2 mg/kg) was administered alone or with furosemide (0.1 to 0.3 mg/kg). Sedation (morphine sulfate, 0.5 to 1 mg intravenously) and muscle relaxation (pancuronium, 0.1 mg/kg intravenously) were used when ICP became elevated because of agitation. We did not induce barbiturate coma in this series of patients. Before 1981, we gave dexamethasone in low (< 20 mg/day) or high (> 20 mg/day) doses, but have not used corticosteroids since then. Serum electrolytes and urine output were measured twice daily for the first 7 days after injury, and once daily thereafter. Antacids were given routinely by nasogastric tube. Two-thirds of the normal amount of maintenance fluids was given intravenously until oral feedings were begun, 3 to 5 days after admission, and the amount of intravenous fluids was reduced.

Results

Thirty-eight children were in traumatic coma for 6 hours or longer; one patient was lost to follow-up review before 6 months. Most of the patients were injured in motor-vehicle accidents (5%), as pedestrians (38%), in domestic accidents (21%), or during sports activities (11%).

The admission GCS scores of the remaining 37 pa-

TABLE 2

<table>
<thead>
<tr>
<th>Glasgow Coma Scale (GCS) score at admission</th>
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<tr>
<td>GCS Score</td>
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<tr>
<td>-----------</td>
</tr>
<tr>
<td>3-4</td>
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<tr>
<td>5-8</td>
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<tr>
<td>&gt; 8</td>
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<tr>
<td>total</td>
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TABLE 3

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<tr>
<th>Summary of best motor response</th>
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<tbody>
<tr>
<td>Response</td>
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<tr>
<td>----------</td>
</tr>
<tr>
<td>obeys</td>
</tr>
<tr>
<td>localizes</td>
</tr>
<tr>
<td>normal flexion</td>
</tr>
<tr>
<td>abnormal flexion</td>
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<tr>
<td>abnormal extension</td>
</tr>
<tr>
<td>flaccid</td>
</tr>
<tr>
<td>total</td>
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TABLE 4

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<tr>
<th>Type of mass lesions found in 17 cases</th>
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<tr>
<td>Type of Hematoma*</td>
</tr>
<tr>
<td>SDH</td>
</tr>
<tr>
<td>EDH</td>
</tr>
<tr>
<td>ICH</td>
</tr>
<tr>
<td>SDH/EDH</td>
</tr>
<tr>
<td>SDH/ICH</td>
</tr>
<tr>
<td>SDH/EDH/ICH</td>
</tr>
<tr>
<td>total cases</td>
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* SDH = subdural hematoma; EDH = epidural hematoma; ICH = intracerebral hematoma.

tients are summarized in Table 2. Ten children were not comatose on admission, but lapsed into a coma subsequently. Flaccidity or posturing was present at admission in 17 patients (46%) (Table 3). Twenty-two patients (59%) had pupillary dysfunction, including bilaterally unreactive pupils in 11.

Computerized tomography scanning was performed in 18 patients, eight of whom fulfilled the criteria of diffuse cerebral swelling with slit-like ventricles, obliterated perimesencephalic cisterns, and flattened cortical gyri without a mass lesion or hemorrhagic contusion. The remaining 10 patients had either an intra- or extraxial hematoma or a focal parenchymal contusion.

Seventeen patients (46%) had intracranial hematomas (Table 4), and six had more than one type of mass lesion. Depressed skull fractures were identified in eight children, six of whom had compound fractures and dural laceration.

Intracranial pressure monitoring was begun soon after injury in 31 patients and after the 1st day in two
additional patients (Table 5). About half the patients being monitored at any given time had an elevated ICP (20 mm Hg or greater).

The outcome at 6 months is listed in Table 6. Nineteen patients (51%) achieved a good recovery or had a moderate disability. Six patients (16%) were severely disabled or vegetative, and 12 (33%) died. Half of the deaths occurred within 72 hours of the trauma; in all of these patients, ICP was persistently greater than 40 mm Hg despite attempts to control intracranial hypertension. Five patients died after 28 days. In four, ICP had been elevated for at least 24 hours at some time during their hospital course. The remaining patient, whose ICP was in the 20 to 40 mm Hg range for 3 consecutive weeks, remained in a vegetative state until death 18 weeks after injury.

Only one patient died of immediate extracerebral causes; namely, cardiac contusion resulting in hemodynamic collapse. Two patients died from iatrogenic causes. Inappropriate antidiuretic hormone secretion went unnoticed in the first child, who was improving 9 days after injury. This resulted in abrupt massive cerebral edema that led to uncontrollable ICP elevation and uncal herniation. The other patient suffered fatal anoxic encephalopathy after an unrecognized respirator disconnection while paralyzed during angiography.

Table 7 shows the effect of age on outcome. Although mortality and severe morbidity rates were greater in older children (11 to 17 years), the relationship between age and outcome was not statistically significant (p = 0.055, Kendall's exact t-test). A higher percentage of younger patients (5 years and less) achieved an independent outcome (good recovery or moderate disability) than did older children. The mortality rate was not higher in patients who were comatose on admission (33%) than in those who lapsed into coma after admission (30%).

Although the three worst motor response scores were usually associated with death or poor outcome, 41% of children who were flaccid or showed abnormal extensor or flexor posturing at admission had a satisfactory outcome (Table 8). Similarly, 25% of the patients with bilaterally unreactive pupils at admission, usually a fatal sign in comatose adults, had a satisfactory outcome.

Good and poor outcomes were almost equally distributed among patients with mass lesions. Eight of 15 patients (53%) who required surgical evacuation of an intracranial hematoma had a good recovery or a moderate disability. Six of eight patients (75%) in whom the CT scan showed diffuse cerebral swelling had a satisfactory outcome (Table 9). Uncontrolled intracranial hypertension was unusual in this group of patients; only one patient with diffuse cerebral swelling had an ICP greater than 40 mm Hg.

Discussion

Despite early assessment and aggressive therapy, the mortality rate in children with severe head injury
Severe head injury in children and adolescents
treated at our institution is higher than that reported by
others. Factors that might account for this include
differences in patient population (for example, we in-
cluded patients with mass lesions, whereas some authors
have not14), in the frequency of intracranial hema-
toma,1,13 and in treatment. Inadvertent patient selection
differences also may be involved. In the series from
Children's Hospital of Philadelphia (CHOP),9 at least
one-quarter of the children were referred from outside
hospitals 24 hours after injury; thus, an unknown num-
ber of patients with unknown outcomes were excluded,
including those who died at referring institutions.
Because of the centralized trauma system in San Fran-
cisco, such exclusions were avoided in our series.

Although in some series10,12,35,36 outcome was better
in patients younger than 10 years of age, we did not
find a significant relationship between age and out-
come. Braakman, et al., and Bruce, et al., also found
no relationship between age and outcome; Hendrick
and colleagues18,19 reported a higher mortality in infants
than in older children.

In our series, as in others,8,26,30,33,38 persistently ele-
vated ICP correlated with poor outcome. Patients whose
ICP remained above 40 mm Hg either died or were left
with severe deficits. Half of the patients who died
within 72 hours of injury had an ICP greater than 40
mm Hg until the time of death. Only one patient who
died had a normal ICP throughout the hospital course.
Most of the published data concerning the effect of ICP
on outcome are from adult patients, and must be ap-
plied cautiously to pediatric patients. Humphreys, et al.,19
reported that ICP alone is not a good prognostic
index in children.

Mass lesions are more common in adult head-injury
patients and have a higher mortality rate than diffuse
injuries,5,11,13,30,32,36 which are more common in chil-
dren.14 In the CHOP series,9 a mass lesion did not
influence outcome. In contrast, nearly 50% of our
patients with mass lesions, regardless of type, died or
were severely disabled. Mayer, et al.,28 and Jennett, et al.,
also found that mass lesions adversely affected
outcome in pediatric patients, and others have reported
that the presence of hematoma adversely affects
outcome in adults.15

Areas of increased density in brain parenchyma on
CT scans after severe head injury may represent
increased cerebral blood volume.6,34,41 Cerebral blood flow
is increased after trauma in children; in three patients
with initially increased cerebral blood flow, CT scans
showed resolution of parenchymal "swelling" after flow
returned toward normal.8 The CT density of the brain
was reported to be increased soon after injury when
brain swelling was present, and returned toward normal
when brain swelling resolved. The authors interpreted
the increased density as hyperemia, which may also
have accounted for the reported increases in cerebral
blood flow. In another study of head-injury patients,
cerebral blood volume was diminished, but three of
four children had been hyperventilated or sedated be-
fore it was measured. In the remaining patient, cerebral
blood volume was elevated soon after trauma.25

Diffuse cerebral swelling was seen in 44% of our
patients undergoing CT scans. Early posttraumatic re-
active hyperemia may be present in nearly one-half of
severely head-injured children.7 Bruce and co-workers7
reported that mannitol may elevate ICP by increasing
the cerebral blood volume, but in our patients ICP rose
immediately after infusion of mannitol only once, even
though it was administered many times. Despite their
apparently grave condition and the poor outcome re-
ported by Humphreys, et al.,19 children with diffuse
cerebral swelling often do quite well.7 In our series, 75% of
such patients had a satisfactory outcome.

The adverse influence of systemic insults on outcome
is well documented.1,2,22,30,31,39 Extracranial insults,
which are reported to occur less frequently in children
than in adults,20 had little effect on outcome in our
patients. One child with severe cardiac contusion died
from hypotension, hypoxemia, hypercoagulable states,
sepsis, and shock can usually be averted by prompt and
aggressive management. However, the two preventable
deaths in our series attest to both the small safety margin
in children and the importance of scrupulous attention
to treatable problems.

Barbiturate-induced coma has been advocated to
control intracranial hypertension when other measures
fail.29-40 Pentobarbital in serum concentrations of 3 to
4 mg% sometimes lowers elevated ICP that is uncon-
trolled by standard means.27 Marshall, et al.,28 have
claimed that high-dose barbiturates improved outcome
after severe head injury; their series included some
patients in whom a mass lesion had been evacuated
before the onset of rising ICP. However, in analyzing
their data, Miller29 concluded that the overall outcome
was not greatly influenced by barbiturate therapy. Yano
and colleagues42 also found no effect of barbiturates on
outcome from head injury in adults. In pediatric pa-

tients without mass lesions, Shapiro and Marmarou41
found a greater improvement in the pressure-volume
index in patients who received barbiturates and man-
nitol than in those who received mannitol alone. None-
thless, barbiturate-induced coma carries a risk of car-
diac rhythm depression and pulmonary complica-
tions,29,39 and was not used in our patients.

There is no evidence that glucocorticoids have a
beneficial effect in patients with head trauma. Most
studies have shown that steroids, regardless of the dose
or type used, do not affect ICP, brain compliance, or
survival statistics.4,12,15,16,26,32 although in one uncon-
trolled study, high-dose dexamethasone reduced ICP
and improved outcome in children with severe head
injury.14 Because of the apparent lack of efficacy and
the possibility of complications, we do not use corti-
costeroids in the management of pediatric or adult head
injury.

In summary, we agree that the outcome after severe
head injury is more favorable in children than in adults.
The mortality rate of 33% in our series of unselected pediatric patients with traumatic coma coincides with results from other pediatric studies.3,17,19,22,35,36 Severe neurological dysfunction at admission does not preclude a satisfactory outcome in children and adolescents. Posttraumatic reactive hyperemia is associated with a good outcome in most patients except in rare instances when ICP cannot be adequately controlled. Persistently elevated ICP (greater than 40 mm Hg) correlated with an adverse outcome in younger patients with and without surgical lesions. Barbital coma should be considered when standard measures fail to control intracranial hypertension. Although we do not agree that the “mortality rate for children being hospitalized with severe head injury should not exceed 10%,” the outcome is certainly better in children than in adults and justifies aggressive management of all children who have head injuries, regardless of the initial ominous presentation.

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References

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