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The head-injured child who "talks and dies" *

A report of 4 cases

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Abstract. The phenomenon of "talking and deteriorating" after closed head injury exists in children. A variety of causes have been identified, few of which are operatively remedial. Four cases of children with head trauma are reported, in each of whom there was an interval during which the child verbalized to some degree. Rapid neurological decline then occurred approximately 30–50 h postinjury in each child, who subsequently died from their trauma. In all instances the children were injured in motor vehicle accidents or falls, had initial Glasgow Coma Scale ratings of 9 or better, and demonstrated irritability and restlessness just prior to their deterioration. In no circumstance was a space-occupying intracranial hematoma present. Post-mortem brain examinations in two of the children showed in common multiple cerebral contusions, brain edema with herniation phenomena and hypoxic-ischemic encephalopathy.

Key words: Pediatric head injury – Delayed coma – Coma scale

It is implied that a patient who has suffered head trauma and has a lucid interval and who subsequently shows neurological deterioration has probably not suffered an overwhelming injury to the brain [9]. Traditional teaching urges the recognition of the treatable surface hematoma in such a patient, where the removal of the clot (characteristically an extradural hematoma) will salvage life. Other factors that may account for similar deterioration include systemic hypotension with resultant cerebral ischemia, hypoxia and seizures and, post-traumatic cerebral edema.

The phenomenon described as "talking and deteriorating" also exists in children, where the explanations for it appear to differ from adults. Children who show neurological deterioration after a lucid interval usually do not have an expanding intracranial hematoma. A number of other causes for the deterioration have been cited. These include acute cerebral edema, convulsions, a migrainous event, functional disturbance of the rostral brainstem, bacterial meningitis or a concomitant viral meningoencephalitis [11]. Obviously, the problem, however infrequent, appears to be poorly explained when it does occur in children.

The entry criteria for the child's admission to the intensive care unit (ICU) were examined in the earlier reviews of severely head-injured children at the Hospital for Sick Children (HSC) in Toronto [3, 5]. The head-injury protocol mandated that all children with an initial Glasgow Coma Scale (GCS) of 7 or less be placed on protocol in the unit. However, there was a small number of patients who had initial HSC GCS ratings of 8 or better and who were admitted to the neurosurgical ward, only to deteriorate subsequently and require transfer to the ICU for vigorous resuscitative therapy. This paper will consider four such cases, all of whom have in common an initial GCS of 8 or better, all of whom "talked" and then deteriorated, and all of whom eventually perished from their head trauma. As the intent is to identify preventable factors in this phenomenon, there will be no consideration of another small group of patients who also showed a lucid interval followed by deterioration and then recovery. This group usually suffered an early post-traumatic convulsion, and this matter has been addressed previously [3, 5, 11].

Patients and methods

The major HSC review of severely head-injured children examined the patients treated between January 1979 and June 1985 [4]. The patients to be studied in the present paper were in part extracted from that earlier study, but also include other relevant cases who received head-injury treatment up to 30 June 1988.

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Case summaries

Case 1

This 10-year-old boy was riding his bicycle when it came in collision with an automobile. He suffered head trauma and was taken to a primary hospital where his GCS was rated at 12. It was reported that he was conscious and alert. Within 6 h of his injury, he was transferred to HSC and given a GCS of 11. The verbal component was 4, to the extent that he would sit up and exclaim "leave me alone." His eyes were closed, but he moved his limbs spontaneously and purposely. A superficial laceration was present in the right temporal region, but the neurological examination was otherwise normal. Skull radiographs were normal. His initial electrolytes were normal and the subsequent fluid administration was appropriate for weight, although his urine output was one quarter of the intake. Throughout the initial period of testing, his GCS was always 10 or greater.

Approximately 30 h post-admission, the child was difficult to arouse and his pupils were dilated and fixed. Shortly after that, he had a grand mal seizure and cardiac arrest. His vital signs returned, although there was subsequent difficulty maintaining systolic blood pressure. It was noted during the resuscitation that the serum sodium had dropped from 137 mmol to 121 mmol over a period of 4.5 h. After transfer to the ICU and placement on mechanical ventilation, the intracranial pressure (ICP) was monitored and readings obtained between 2 and 10 torr. Immediate burr holes showed the brain on the right side to be "slack" and the left side to be extremely "tight." A "thin smear of subdural hematoma" was found on the left. Subsequently, the computed tomography (CT) scan (his first) showed gross cerebral edema but no mass lesions. The ICP was elevated, and the child at the 8th day fulfilled all clinical and laboratory evidence of brain death.

The case was investigated by the provincial coroner and no post-mortem examination was ordered.

Case 2

A 4-year-old child sustained injuries to the head, face and abdomen as an unrestrained passenger in a motor vehicle that came in collision. Perhaps his ultimate condition was signalled by initial decerebrate posturing. However, when assessed at 2.5 h post-trauma [by which time had been given mannitol and dexamethasone (Decadron), which brought about "improvement"], his GCS was 9 [2, 3, 5]. The left pupil was larger, he had retinal hemorrhages, and he withdrew from noxious stimuli. Skull radiographs were unremarkable and a CT scan was not done until 36 h post-admission.

During the interval, the child's vital signs remained stable except for his pupils. Because the left pupil dilated further and reacted very sluggishly, the CT scan was completed and was normal except that the "basal cisterns were not well visualized."

He was transferred to the ICU for continued heat-injury management, which included the placement of an ICP monitor. It was noted that in the course of 27 h his serum sodium had falled from 141 to 129. The ICP, which was initially normal, began to escalate and at 55 h post-trauma, the child had a cardiac arrest. He was resuscitated from that, begun on a barbiturate coma program, and thereafter to his demise 7 days post-admission, had difficulty with bilateral pneumothoraces, intracranial hypertension, hypothermia, and eventually vasomotor collapse. Brain death was declared.

The coroner's post-mortem examination revealed occipital skull fracture, bilateral frontal, temporal and occipital cerebral contusions, acute left subdural hematoma, laceration of the septum pellucidum, and extensive cerebral edema with hypoxic-ischemic encephalopathy, hippocampal uncal and cerebellar tonsillar herniation, and thrombosis of the left internal jugular vein.

Case 3

This 14-year-old boy was said to have fallen 10 ft. (about 3 m) while sleepwalking and to have struck his right temporal region on the corner of a piano. He was found unconscious, and when first assessed in the referring hospital, was given a GCS of 7. His initial HSC evaluation was done 7 h post-trauma, when the GCS was 13 [4, 5]. He had a small abrasion on the right temporal region, was intermittently quiet and agitated, would resist examination, and he struggled to sit up. He opened his eyes spontaneously and would hold up fingers to command. His speech was occasionally intelligible. He had no apparent asymmetric motor movements, seemed to be purposeful on occasion, and had bilateral extensor plantar responses. The skull radiographs were unremarkable.

During the next 24 h, his condition remained unchanged. He was intermittently restless and agitated. His first CT scan done at that time showed small intracerebral hematomata in the right frontal and parietal regions. There was associated right hemispheric edema. During this study, he had several apneic episodes, so that he was intubated and mechanically ventilated. An ICP monitor was placed, and it initially recorded normal pressure. However, that became elevated about 4 h post-ICU admission and proved to be intractable to the usual maneuvers to diminish pressure. The boy's condition continued to deteriorate and he died 72 h post-admission. It is specifically noted that his electrolytes and fluid balance were maintained at normal levels until 24 h prior to demise, when he developed marked hypernatremia.

The coroner's post-mortem examination revealed extensive bilateral frontal and temporal lobe cerebral contusions. Intracerebral hemorrhage involved the right frontal lobe and the temporal lobes bilaterally. A diffuse shear injury involving the cervical cord with mechanical disruption, swollen axons and petechial hemorrhages was also noted. Hypoxic-ischemic encephalopathy was noted along with cerebellar tonsillar herniation and bilateral uncal herniation.

Case 4

This 10-year-old boy fell through a carport roof, approximately 12 ft. (3.5 m), landing on his head. He was described as being unconscious at the scene, and when assessed first at the primary hospital had a tested GCS of 7–8. His condition was stabilized at the primary hospital, and the child was transferred to HSC.

By that time, he was restless, but beginning to move his extremities in a purposeful fashion. He did not moan or speak. At 6 h post-trauma his clinical condition was upgraded to a GCS rating of 10 [2–4]. The CT scan showed a left temporo-parietal linear skull fracture extending into the petrous bone. There was mild, diffuse brain swelling with shift. The perimesencephalic cistern was slightly effaced.

The child's clinical condition remained stable and may have improved over the next 22 h. For example, his GCS improved to 11–12, when it became apparent the child was engaging in short sentence conversation with his mother, asking about various inserted tubes and lines. He remained somewhat restless and agitated. Approximately 26 h after admission, he had the first of two brief grand mal seizures. In each instance, they stopped spontaneously, but the child was started on anti-convulsant medication after the first such event. He aroused quickly after these events, but remained irritable and restless and was endeavoring to pull out his various lines.

Approximately 4 h after the second seizure, the child developed respiratory deterioration and had a full cardio-pulmonary arrest. Resuscitation was prolonged and consciousness was never recovered. Follow-up CT scan outlined severe brain edema with evidence of herniation. At the time of the arrest, the child's serum sodium was 127, having been recorded at 140 approximately 30 h earlier.

At the request of the family, the child was maintained on life-support equipment for the week after his arrest, and was eventually pronounced brain dead 10 days post-trauma. The coroner's office was notified, but a post-mortem was not obtained.

Discussion

The possible explanations for a child's deterioration after a lucid interval which follows head injury have recently been examined [1, 11]. It is acknowledged at the outset that a surgeon's primary responsibility in such a circumstance is to diagnose and treat an expanding intracranial hematoma, but it is noted that in children who have received a "severe" head injury, such a hematoma is not a constant factor, occurring in no more than 35% of such patients [1, 5]. In none of the cases reported here was an immediate or delayed intracranial hematoma of sufficient volume found responsible for the child's deterioration.

The phenomenon of "talk and deteriorate" or "talk and die" after head injury begs certain definitions. The review of Marshall et al. analyzed 34 "severely" head injured patients, although all must have had an initial GCS of better than 8, to which level (or worse) they subsequently deteriorated [7]. The patients studied had initial verbal scores of 3 or better. The pediatric review of Snoek et al. addressed "deterioration" after "mild" head injury and included patients who had suffered no immediate loss of consciousness, no amnesia, hematoma or skull fracture (or a linear fracture only) [11]. To be fair, the HSC patients were likely more seriously injured with GCS scores of 9, 10, 11 and 13, and verbal scores of 4, 3, 4, and 4, respectively. Common to all the children in our series was the fact that they were in a state of restless agitation, and although the speech delivery was clear and meaningful, it was nevertheless protective and indicative of the patient's agitated state.

Ninety-four percent of children with severe head trauma are injured in one of two ways – road traffic accidents (as vehicle passengers, pedestrians, or cyclists) and falls [5]. Such events would seem to be easily confirmed, but Bruce when addressing delayed deterioration of consciousness after minor head injury in children cautions that the history of trauma may be disparate with the clinical picture [1]. In each of the four cases reported here was the history substantiated; two children suffered falls and two were injured in road traffic accidents. On the basis of the history alone, each of the four children sustained injuries by mechanisms capable of producing "severe" head trauma, notwithstanding their initial GCS scorings, which indicated that three of them had suffered "moderate" and one had suffered "minor" head injury. There was incidentally some variation in the GCS scores taken immediately after trauma, and those which were recorded at the obligatory time of 6 h post-trauma by HSC examiners.

Does the entity of "talk and deteriorate" or more particularly "talk and die" occur in children? Snoek et al. reviewed 42 children who deteriorated after head injury, and only one of whom had a treatable intracranial hematoma [11]. Thirteen other children had early post-traumatic convulsion; all recovered. There were 29 "non-convulsive" cases who showed either "rapid" (within 36 h) or "slow" deterioration. There were 3 deaths, 1 in the former group and 2 in the latter ("slow") group, all apparently due to severe and uncontrollable unilateral or diffuse brain swelling. The necropsy review of Reilly et al. identifies 3 children with head injuries who talked and died, and all 3 were described as eventually having "status epilepticus" [8]. It is not clear whether the seizures were the cause or result of the deterioration, especially as 2 of the patients were infants. The study of Reilly et al. was of 66 patients (mostly adults), 25% of whom did not have intracranial hematoma at autopsy. Most of these had raised intracranial pressure and the commonest finding was local swelling related to brain contusions. Almost half of the non-hematoma cases had ischemic or hypoxic brain damage usually without contusions.

The matter of the child with a head injury who "talks and deteriorates" was alluded to in an earlier paper from HSC [3]. In that study, 10 of 138 children were admitted to the ICU for treatment because of an earlier post-traumatic convulsion. Such may be witnessed, but in other instances it could occur completely unobserved. We were previously reassured that most of the children with this innocent complication had an ultimate GCS of 8 or better and showed improvement within 16–24 h after injury. This group of children would correspond to the first group reported by Snoek et al. [11].

However, more alarming is the smaller group of patients who not only "talk and deteriorate," but as represented by the four patients reported here, proceed on to die, after suffering a sudden and relatively unexpected combination of cardiorespiratory arrest, convulsions and markedly raised intracranial pressure. It is noted, first of all, that although the initial GCS scorings of these four patients categorized them as sustaining "moderate" (3 children) or "mild" (one patient) head injury, all sustained their injuries by mechanisms usually responsible for "severe" head injury (two in motor vehicle accidents, two in falls). Only one of our four patients was reported as being conscious at the scene of trauma; the other three were initially unconscious at least, and one of them showed decerebrate posturing. Yet, all four patients had improved GCS ratings at the obligatory 6-h testing point. By that time, and for a short while thereafter, all had verbal responses which were characterized at best by "confused" speech, or in one instance by "inappropriate words." It was after these bursts of verbal expression that the patients deteriorated and perished.

At the time of their deterioration, three of the four children were shown to be hyponatremic, where in fact their electrolyte values had been normal 4.5–30 h earlier. Urine and serum osmolarity values were not obtained post-resuscitation, as either the sodium was very quickly corrected or the patient expired from the event bringing about the deterioration. Perhaps as testimony to the otherwise benign appearance of the initial head injury, only one of the four patients had an immediate post-admission CT scan (which was reported as within normal limits). CT scans were obtained on a delayed basis in the other three patients, two of whom were studied at the time of their deterioration, and the fourth child at a point when his clinical condition was stable. The scans in each of these three patients were abnormal, although none outlined a lesion requiring operative correction.

The post-mortem examination of the brains of two of the children showed in common multiple cerebral contu-

sions, brain edema with traditional herniation phenomena and hypoxic-ischemic encephalopathy. Although focal hemorrhages were noted within parenchyma, none of these would require traditional operative release. It is thus postulated that each child's condition deteriorated because of a delayed cerebral hyperemic state.

It is apparent from previous reports as well as this paper that a small number of children who have received head trauma will not only "talk and deteriorate," but some will proceed on to die of their injuries [8, 11]. This observation is in contrast to the comment by Luerssen et al. who in a review of 1,906 children less than 14 years of age noted that no child with GCS score of 11 or more died [6]. It is apparent from our earlier study, as well as the present paper, that as reliable as it is, GCS of 8 or greater is not always associated with a satisfactory outcome [5]. The question therefore is how we are best able to predict those children who are at risk for such deterioration and disastrous outcome?

It is recommended, first of all, that the history of injury be borne in mind when the decision for the child's post-admission placement is made. In that regard, Sainsbury and Siebert have addressed this matter in children, but again only specific to the development of complicating intracranial hemorrhage [10]. They noted that all 35 children who subsequently developed intracranial hematoma had "symptoms or signs clearly needing continued observation or treatment six hours after the injury." The paper, however, did not address the issue of children who deteriorated for other reasons, and who might die of their injury. Thus, we would suggest that all children who have sustained violence as a result of a motor vehicle accident or a fall, and who have an initial GCS scoring of 9–12, be admitted to a "step-down" intensive care facility. It is assumed that all children who have a GCS of 8 or less will be admitted on protocol to an intensive care unit, and it may not always be practical or possible to admit children with better scores to that same unit.

Early CT scanning is recommended for these same patients (i.e., GCS 9–12), as is close neurological watch testing. The clinician should be alert to coincident restlessness in the child with closed head trauma. The usual covert causes of such should be sought and, in particular, attention given to the possibility of cerebral hypoxia. Intravenous fluid balance and monitoring of electrolytes with urinary output measurements should be performed at least through the first 48 h. Notwithstanding a recent report of Hahn et al., it is unclear whether the early administration of prophylactic anticonvulsant medication would have aborted the seizure occurrences in two of our children [2]. Finally, consideration may have to be given to current head injury protocols and whether children with GCS of 9 or better should enter a protocol for ICP recording and assisted ventilation.

References

1. Bruce DA (1984) Delayed deterioration of consciousness after trivial head injury in childhood. Br Med J 289:715–716
2. Hahn YS, Fuchs S, Flannery AM, Barthel MJ, McLone DG (1988) Factors influencing post-traumatic seizures in children. Neurosurgery 22:864–867
3. Humphreys RP (1983) Outcome of severe head injury in children. Concepts Pediatr Neurosurg 3:191–201
4. Humphreys RP (1989) Patterns of pediatric brain injury. In: Miner ME, Wagner KA (eds) Neurotrauma: treatment, rehabilitation and related issues, no 3. Butterworths, Boston, pp 115–126
5. Humphreys RP, Jaimovich R, Hendrick EB, Hoffman HJ (1983) Severe head injuries in children. Concepts Pediatr Neurosurg 4:230–242
6. Luerssen TG, Klauber MR, Marshall LF (1988) Outcome from head injury related to patient's age. A longitudinal prospective study of adult and pediatric head injury. J Neurosurg 68:409–416
7. Marshall LF, Toole BM, Bowers SA (1983) The National Traumatic Data Bank, part 2: Patients who talk and deteriorate. Implications for treatment. J Neurosurg 59:285–288
8. Reilly PL, Adams JH, Graham DI, Jennett B (1975) Patients with head injury who talk and die. Lancet II:375–377
9. Rockswold GL, Leonard PR, Nagib MG (1987) Analysis of management in thirty-three closed head injury patients who "talked and deteriorated." Neurosurgery 21:51–55
10. Sainsbury CPQ, Siebert JR (1984) How long do we need to observe head injuries in hospital? Arch Dis Child 59:856–859
11. Snoek JW, Minderhoud JM, Wilmink JT (1984) Delayed deterioration following mild head injury in children. Brain 107:15–36