

Fatal head injury in children

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SUMMARY A comprehensive neuropathological study was undertaken on 87 children aged between 2 and 15 years with fatal head injuries to identify those features which occurred at the time of head injury (fractured skull, contusions, intracranial haematoma and diffuse axonal injury) and those which were subsequently produced by complicating processes (hypoxic brain damage, raised intracranial pressure, infection and brain swelling). The types of brain damage identified were remarkably similar to those seen in adults. The only difference was the prevalence of diffuse brain swelling in children.

Trauma is a major cause of death in children,¹ and in school children it is the most common single cause. Of all accidental deaths, the greatest number are due to head injury.²⁻⁴

In our previous studies on fatal non-missile head injury we already drew attention to the prevalence of diffuse brain swelling in children and adolescents⁵ but did not study in depth all the types of brain damage that occur in children. There may, therefore, be other differences between the types of brain damage that occur in children and in adults which so far have been overlooked, a knowledge of which might contribute to the management of head injury in children. There are no very good reasons for assuming that there are many differences in the types of brain damage sustained^{6,7} as the biomechanics of the injury are similar in all age groups other than in infancy and non-accidental injury, but the time seemed right to analyse in detail brain damage in fatal head injury in children as recorded in our comprehensive database.

Material and methods

Between 1968 and 1982 full necropsies were performed on 635 cases of fatal non-missile head injuries which had been managed by the department of neurosurgery at this institute. There were 497 males and 138 females with an age range of 9 weeks to 89 years (median 36 years) and a duration of survival from one hour to 14 years three months (median two to three days). The brains and spinal cords were fixed in 10% formol saline for at least three weeks before dissection. The cerebral hemispheres were cut in a standard manner⁸ in the coronal plane, the cerebellum at right angles to

the folia, and the brain stem horizontally. Comprehensive histological studies, including the examination of large celloidin sections of brain, were carried out on 434 of the 635 cases. Macroscopic and histological abnormalities were recorded on a series of line diagrams of the cerebral hemispheres, the cerebellum, and the brain stem. All abnormalities were then transferred to a proforma and the data stored and analysed on the University of Glasgow's mainframe computer.

The present analysis is based on 87 children aged between 2 and 15 years on whom comprehensive histological studies were undertaken. Cases less than 2 years of age were excluded as before this age the brain, skull base, and calvaria differ from those of the adult.⁹ The clinical records of the children were assessed to establish whether they had been able to talk immediately after their injury, even if only in a confused manner, on the basis that if an individual who sustains a head injury is able to talk, brain damage occurring at the moment of injury cannot have been overwhelming.¹⁰

The criterion of pressure necrosis in one or both parahippocampal gyri was used as evidence that the intracranial pressure had been high during life as a result of a supratentorial expanding lesion.¹¹ The total contusion index¹² is a quantitative index that takes into account the depth and extent of contusions in various parts of the brain: 0 means that there were no contusions, a contusion index in the 20's is indicative of moderately severe contusions, while one of more than 37 is indicative of severe contusions.

Results

There were 58 boys and 29 girls with an age range between 2 and 15 years (median 9 years). Survival

ranged from two and a half hours to three years (median less than 48 hours). Most injuries were sustained as a result of a road traffic accident (n = 62, 71%) a fall (n = 18, 21%), or from assaults (n = 2, 2%). Of the remaining five cases, two were crush injuries, while the precise circumstances were unknown in three. Fewer patients (n = 14, 16%) talked after their injury than those who did not (84%), suggesting that children die most often as a result of damage sustained at the moment of injury, as a third of our total database of 635 cases talked after their injury.

There was a fracture of the skull in 63 (72%) cases. The total contusion index ranged from 0 to 47 (median 16.0): there were seven cases (8%) without contusions: the mean total contusion index in the remaining cases was (mean 17.61 (SD) 13.02). Large intracranial haematomas were present in 30 (34%) cases of which 26 were supratentorial and four infratentorial. Of the 26 supratentorial haematomas, three were extradural, four pure subdural, 14 pure intracerebral and in five there was a "burst" frontal or temporal lobe. Of the four infratentorial haematomas, two comprised a "burst" lobe, one a pure intracerebellar haematoma, and one pure subdural haematoma.

Diffuse axonal injury¹¹ was identified in 19 (22%) cases. Severe or moderately severe ischaemic damage in the neocortex was present in 53 (61%) cases. Of these 53 cases, there were 25 with diffuse necrosis; 12 in which the ischaemic damage was centred on the boundary zones between the major arterial territories, particularly between the anterior and middle cerebral arterial territories; three in which the ischaemic damage was centred on the middle or anterior cerebral arterial territories; and 13 cases in which there was a mixed pattern of ischaemic damage in the cortex. In 70 of the 87 cases there was ischaemic damage in the hippocampus (bilateral in 56) and in 54 ischaemic lesions in the basal ganglia or thalamus. In the remaining 34 of the 87 cases, there were 25 in which the ischaemic damage was mild (less than five small ischaemic lesions in the brain), leaving only nine cases in which there was no ischaemic damage.

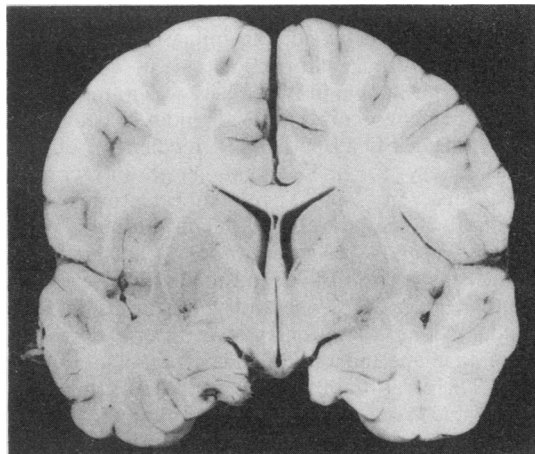


Figure Diffuse swelling of both cerebral hemispheres: lateral ventricles are small and there are bilateral tentorial herniae (arrows).

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There was swelling of the brain in 61 (70%) cases (table). In 45 of these it was bilateral: in 18 of these there was no apparent cause (figure), in 14 it was attributable to ischaemic damage, in eight to contusions, in two to intracranial haematomas, and in three to a combination of these factors. There was no association with diffuse axonal injury. In 16 cases with unilateral hemispheric swelling it was attributable to an intracranial haematoma in nine, to ischaemic brain damage in four, to contusions in two, and to a combination of these in one.

Morphological evidence that the intracranial pressure had been high during life was found in 72 (83%) cases. In four cases there was acute bacterial meningitis while cerebral fat embolism was identified in one case.

Thirteen (15%) of the 87 cases were able to talk immediately after their injury: they were aged between 2 and 15 years (median 9 years) with a survival of

Table Cause and prevalence of brain swelling in 86 of 87 children aged between 2 and 15 years

Left	Right		Intracranial haematoma	Ischaemic brain damage	Combination	Not known	Total
	Absent	Contusions					
Absent	25	1	7	2	0	0	35
Contusions	1	8	0	0	0	0	9
Intracranial haematoma	2	0	2	0	0	0	4
Ischaemic brain damage	2	0	0	14	0	0	16
Combination	1	0	0	0	3	0	4
Not known	0	0	0	0	0	18	18
Total	31	9	9	16	3	18	86

between eight hours and 14 days (median 48 hours). Of the 13 cases, six sustained their injury as a result of a fall and five from a road traffic accident. The precise cause was not known in two. There was a fracture of the skull in eight, a total contusion index of between 0 and 38 (mean SD 11.69 (11.72); median 9), diffuse axonal injury in one, and a large intracranial haematoma in six (five supratentorial—two extradural, two subdural, and one intracerebral; and one infratentorial—a “burst” lobe). Ischaemic brain damage was severe in five, and moderately severe in five: there was diffuse necrosis of neurons in five, boundary zone infarction in two, arterial territory infarction in one and a combined pattern of infarction in two. There was brain swelling in nine cases (bilateral in eight) due to ischaemia in four, of unknown cause in three and to intracranial haematoma in one; in one case there was unilateral hemispheric swelling due to a combination of factors. There was evidence of raised intracranial pressure in nine cases.

Discussion

As there are few studies that detail the neuropathology of head injury in childhood⁶ the principal purpose of this study was to analyse the findings in the 87 cases to identify those features which occurred at the time of injury (primary damage, such as fracture of the skull, contusions and lacerations, and diffuse, axonal injury) and those produced by complicating processes which did not present clinically for a period of time after injury (secondary damage, such as intracranial haemorrhage, brain swelling, hypoxic brain damage, infection and raised intracranial pressure).^{5,14}

Although morbid anatomical studies are sparse, it is possible to compare the current series of patients with the information derived from computed tomography scans, although case selection might account for any differences. One such study is that of Zimmerman and Bilaniuk,¹⁵ who reported the principal computed tomography findings in 262 children with acute head injuries, 30% of whom had minimal to no disturbance of consciousness, 31% minimal to moderate disturbance of consciousness, 25% of whom had clinically important disturbance of consciousness and 14% of whom were in coma. In contrast, in the present series there was a larger number of more severely injured patients as 84% did not experience a lucid interval. Head injury due to falls is said to be much more common in childhood than in adults,¹⁶ although in the present series of cases most of the injuries were sustained as a result of a road traffic accident (71%) rather than falls (21%).

The presence of a fracture of the skull indicates that the impact has had considerable force, but many patients with a fracture have no evidence of brain

damage and make a smooth and uneventful recovery.⁵ The incidence of fracture of the skull varies with the clinical severity of the head injury, ranging from 8% of children seen in the accident and emergency room to 27% of those requiring admission to hospital.¹⁷ In the present group there was a fracture of the skull in 62% of the children who had been lucid following injury and 72% of the cases that had not been lucid.

Cerebral contusions are common in non-missile head injury and have been regarded as their hallmark; indeed, they were present in some 90% of the present series. The finding of a median contusion index of 16.0 in this series is therefore unexpected in that reports suggest that contusions are less common in head injured children than in adults.¹⁵ Our own studies suggest that these figures of 35% in adults and 16% of children¹⁵ underestimate quite considerably the true incidence of contusions when quantitative methods are used in the assessment of their incidence and severity.¹²

Recent clinical¹⁸ and pathological¹² studies have emphasised the importance of diffuse brain damage in head injury. According to Zimmerman and Bilaniuk,¹⁵ computed tomography evidence of this type of brain damage, referred to by these authors as “shearing injury”, is less common in children (4%) than in adults (7%), most of the patients having been passengers in car accidents: 73% of the children with “shearing injury” died, a figure not too dissimilar from adults.¹⁹ In the present series of cases, diffuse axonal injury was found in one of the 14 children who had talked after injury and in 18 of those who had not.

The complication most commonly associated with deterioration after a head injury is intracranial haematoma.¹⁰ In our study of 434 consecutive cases of fatal head injury there were 152 (35%) with a sufficiently large supratentorial intracranial haematoma to warrant evacuation.²⁰ This figure is similar to that found in the present series of children (30%). Our study would therefore suggest that in severe head injuries the incidence of supratentorial intracranial haematomas is similar in adults and children.

Nevertheless, intracranial haematomas are said to be a less common complication than in adults,¹⁵ even though more occur without fracture.^{21,22} The incidence of extradural haematoma in children is said to range from between 2.6% of head injury admissions under the age of 15^{23,24} to 6.5%.²⁵ In our series, of the 26 children with supratentorial haematomas there were three (12%) with extradural haematomas. Acute subdural haematomas are also said to be less common in children—about 8% of cases with severe injury—than in adults.^{15,23} In our series pure acute subdural haematoma comprised four of the 26 supratentorial haematomas (15%) and one of the four infratentorial

haematomas (25%). Perhaps not unexpectedly, the largest incidence of intracranial haematomas was found in the group of patients who talked and died (six of 13, 46%).

In our major series of 434 cases of fatal head injury, we found moderate or severe ischaemic brain damage in 50%.²⁰ In the present series it was present in 61%. The patterns of ischaemic brain damage were similar, with many examples of both diffuse necrosis of neurons and focal damage, either in the neocortex or subcortical structures. In many instances the pathogenesis of the ischaemic lesions can be adduced from the pattern of the brain damage.²⁶ It is quite clear from the present series that the incidence of ischaemic brain damage in children with fatal head injury is high and its pathogenesis is similar to that occurring in adults.

Focal cerebral oedema due to disruption of the blood-brain barrier with the extravasation of protein rich fluid into the extracellular space of the brain was seen in many of the cases of this series in relation to contusions, or intracerebral haemorrhage, or a combination. There is also an entity of diffuse cerebral swelling which has been found by computed tomography scanning in over 40% of comatose children after head injury.²⁷ The principal computed tomography findings are those of small ventricles and compression of the basal cisterns and the principal pathological features are enlargement of the cerebral hemispheres and obliteration of the cerebrospinal fluid spaces. Where intracranial pressure monitoring has been carried out pressures in excess of 20 mm Hg have been found in over 80% of cases.²⁸ Increased density of the cerebral parenchyma on computed tomography scans soon after injury has been interpreted as evidence that cerebral hyperaemia is a cause of diffuse cerebral swelling,²⁷ and in some children increased cerebral blood volume has been documented using technicium labelled red cells.²⁹ In the present series of children swelling of the brain was found in 61 cases, in 45 of which it was bilateral. This is in contrast to an incidence of 17% in the cases aged more than 15 years in the main series. In 27 of the 45 cases the swelling was attributable to ischaemic damage, contusions or intracranial haematomas, or a combination of these factors. In the remaining 18, however, no underlying cause could be identified—that is, they were examples of idiopathic brain swelling resulting from head injury in children. In three cases the patients had talked after their injury. There were also examples of unilateral hemispheric swelling attributable to ischaemic damage, contusion, and intracranial haematoma.

Morphological evidence of a high intracranial pressure was present in some 70% of our main series of 425 cases.²⁰ In the present series it was present in 83%.

This analysis of brain damage in head injury in children has established that it is not strikingly

dissimilar from that seen in adults. Hypoxic brain damage and evidence of a high intracranial pressure are common in children but the only really striking difference is the occurrence of diffuse brain swelling in children. Its causation and management remain a problem.

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