Diffuse axonal injury caused by assault

D I Graham, J C Clark, J H Adams, T A Gennarelli

Abstract
The case reports of 50 fatal head injuries caused by assault and managed at the Institute of Neurological Sciences, Glasgow, were reviewed. Fifteen cases had diffuse axonal injury.

Diffuse axonal injury is a well recognised type of brain damage brought about by a head injury, usually as a result of a road traffic accident or fall from a height. It does not seem to be widely appreciated that it may also occur as a result of an assault. This has important medicolegal implications.

Diffuse brain damage and diffuse axonal injury (DAI) are largely attributable to acceleration or deceleration of the type and magnitude associated with road traffic accidents and less frequently with falls from a height. This study is a review of the Department’s experience of DAI resulting from an assault.

Methods
Full necropsies were performed on the 50 fatal head injuries due to assault in our database. All had been managed by the Department of Neurosurgery at the Institute of Neurological Sciences, Glasgow, between 1968 and 1990. In 15 of the 50 cases there was diffuse axonal injury. There were 13 men and two women with an age range between 17 and 83 years (average 38.3 years). Survival ranged from 26 hours to 21 months (average 87 days). The cause of injury was determined as far as possible from the medicolegal records (table). Eleven of the cases were in coma from the time of injury and four experienced a partial lucid interval they were able to talk in a confused manner a short time after the injury. Comprehensive histological studies were carried out, as described previously.

Results
These are summarised in the table.

Of the 15 cases in this study, DAI was grade 3 in 10, grade 2 in one, and grade 1 in 4. In four of the 15 cases DAI was identified only by microscopic examination. There was a fracture of the skull in five cases; it was depressed in three. The contusion index ranged from zero to 61 (average 9.9); there were five cases without contusions, five in which they were minimal (TCI 1–10), three in which they were mild (TCI 11–20), one in which they were moderate (TCI 21–30) and one in which they were severe. Large intracranial haematomas (TCI of > 30) were present in four cases, all of which were supratentorial: of these one was pure subdural, one pure extradural, one pure intracerebral and in one there was a “burst” temporal lobe.

Ischaemic damage was present in 11 of the cases: it was mild in seven, moderately severe in two, and severe in two. There were four cases in which there was no ischaemic damage.

There was swelling of the brain in four cases: in three it was unilateral and in one bilateral. Evidence that the intracranial pressure had been high during life was found in seven cases. In one case acute bacterial meningitis was found.

Discussion
The finding of 15 cases with DAI as a result of an assault was rather surprising as previous studies have suggested that it was largely caused by injuries to the head of the type associated with either road traffic accidents or a fall from a height, and rarely after an assault. As in other cases of DAI a gradient of severity of injury was also found in the current series of cases following assault.

Particular attention was paid to details surrounding the assault. Most of the cases in this study were involved in a brawl that resulted in an exchange of punches, which in some was also associated with additional blows to the head by kicking or heavy objects. In some instances, as a result of punching, the victim had an accelerated fall striking his head on the ground, kerb, or pavement. In some instances there was no punching, blows to the head being delivered by heavy objects made either of wood or metal. Unfortunately, precise details are not known and it is perhaps not surprising, therefore, that it has not been possible to identify a type of assault that predisposes to a particular pattern of damage and to identify a causal association between the mechanism of injury in those cases either with or without diffuse axonal injury.

Diffuse axonal injury has not been described in any of the review articles reporting the neurological sequelae of boxing. Acute neurological sequelae of boxing are largely due to the formation of either subdural or intracerebral haematomata. The best known chronic sequel of boxing is the development of dementia pugilistica the neuropathology of which includes changes in the septum pellucidum, scarring of the cerebellar tonsils, depigmentation of the substantia nigra, numerous
neurofibrillary tangles, and more recently with the multifocal deposition of \( \beta \) protein as diffuse plaques.

Although this study has established that all grades of DAI occur in fatal head injuries due to assault, the mechanism of its causation is unclear. The lack of full information surrounding the assault clearly leaves open the possibility that the victims sustained injuries other than those simply due to punching. Therefore, this study cannot determine whether DAI was produced by the assault itself, by the ensuing fall to the ground, or by a combination of the two. In any case the combination of head motions that resulted clearly exceeded the acceleration conditions that are necessary to produce diffuse axonal injury.

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<table>
<thead>
<tr>
<th>Patients</th>
<th>Sex</th>
<th>Age</th>
<th>Lucid interval</th>
<th>Survival</th>
<th>Cause of injury</th>
<th>Fracture of skull</th>
<th>Large intracerebral haematoma</th>
<th>Total concussion index</th>
<th>Severity of ischaemic brain damage</th>
<th>Diffuse swelling</th>
<th>Evidence of high intracranial pressure</th>
<th>Grade of DAI</th>
<th>Other pathologies</th>
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<tbody>
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<td>1</td>
<td>M</td>
<td>47</td>
<td>None</td>
<td>2 m</td>
<td>Punch</td>
<td>-</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>18</td>
<td>None</td>
<td>21 m</td>
<td>Punch</td>
<td>-</td>
<td>-</td>
<td>6</td>
<td>Mild</td>
<td>+</td>
<td>3</td>
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<td>59</td>
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<td>-</td>
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<td>2</td>
<td>-</td>
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<td>4</td>
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<td>25</td>
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<td>4 w</td>
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<td>-</td>
<td>13</td>
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<td>-</td>
<td>-</td>
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<td>M</td>
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<td>M</td>
<td>83</td>
<td>None</td>
<td>6 d</td>
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<td>+</td>
<td>-</td>
<td>16</td>
<td>Mild</td>
<td>-</td>
<td>-</td>
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<tr>
<td>7</td>
<td>F</td>
<td>18</td>
<td>Partial</td>
<td>26 h</td>
<td>Blows from metal crowbar</td>
<td>D+</td>
<td>R ICH</td>
<td>25</td>
<td>Mild</td>
<td>R</td>
<td>+</td>
<td>1</td>
<td>Acute meningitis</td>
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<td>-</td>
<td>5</td>
<td>Mild</td>
<td>-</td>
<td>-</td>
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<tr>
<td>9</td>
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<td>17</td>
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<td>6 d</td>
<td>Sticks and blows from bricks and punch</td>
<td>D+</td>
<td>R SDH</td>
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<td>Moderate</td>
<td>R</td>
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<tr>
<td>10</td>
<td>M</td>
<td>62</td>
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<td>5 d</td>
<td>Blows from hammer</td>
<td>D+</td>
<td>L ICH SDH</td>
<td>61</td>
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<td>B</td>
<td>+</td>
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<td>Partial</td>
<td>5 m</td>
<td>Punch and blow from baseball bat</td>
<td>-</td>
<td>L EDH</td>
<td>3</td>
<td>Mild</td>
<td>-</td>
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<td>F</td>
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<td>None</td>
<td>11 d</td>
<td>Punch and blow from unknown object</td>
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<td>-</td>
<td>0</td>
<td>Mild</td>
<td>-</td>
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<td>43</td>
<td>None</td>
<td>10 m</td>
<td>Punch and fall</td>
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<td>-</td>
<td>0</td>
<td>Severe</td>
<td>-</td>
<td>-</td>
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<td>29</td>
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<td>12 d</td>
<td>Punch and blow from metal pipe</td>
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<td>-</td>
<td>0</td>
<td>Severe</td>
<td>R</td>
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<td>Punch and fall</td>
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<td>0</td>
<td>Severe</td>
<td>R</td>
<td>+</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

\( d = \text{days}; w = \text{weeks}; m = \text{months}; h = \text{hours}; D+ = \text{depressed fracture}; R = \text{Right}; L = \text{Left}; B = \text{Bilateral}; \) ICH = Intracerebral haematoma; EDH = Extradural haematoma.

For definitions of large intracerebral haematoma, total concussion index, severity of ischaemic brain damage, evidence of high intracranial pressure and grade of DAI see Adams.
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