BRAIN INJURIES IN CAR OCCUPANTS: A CORRELATION OF IMPACT DATA WITH NEUROPATHOLOGICAL FINDINGS

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ABSTRACT

This study is based on 26 car occupants who were fatally injured or who sustained a severe head injury in crashes near Adelaide, South Australia. Crash reconstruction techniques were used to estimate the acceleration of the head in 17 cases. These accelerations were correlated with the severity and distribution of brain injury as determined by neuropathological and/or radiological examination. Estimated linear accelerations ranged from 1000 m/s^2 to 6600 m/s^2. There was a positive association between linear acceleration and the extent of haemorrhagic brain injury, in lateral, but not frontal, impacts to the head. The estimated angular accelerations ranged from 6000 rad/s^2 to 50,000 rad/s^2 and showed a similar positive association with the extent of brain injury in lateral impacts. For the entire group of cases there were 15 frontal impacts to the head, 7 lateral impacts, three occipital impacts and in one case the location impact was not known. Lateral impacts were more likely to be associated with death than were frontal impacts at similar levels of acceleration.

1 INTRODUCTION

Since 1982, the NHMRC Road Accident Research Unit at the University of Adelaide has been attempting to refine head impact tolerance data by analysis of real world pedestrian - vehicle collisions utilizing detailed injury, vehicle and site data for each case. The method has been described in detail by Ryan et al [1].

The present paper reports on the application of this method to the study of head impacts to car occupants.

2 METHODOLOGY

2.1 Case Selection

We included all cases of severe head injury or death (from any cause) occurring to car occupants in crashes in and around Adelaide. We defined severe head injury as a Glasgow Coma Score of less than 8 when measured 6 hours after injury, or 7 when the 14 point version of the scale was used [2,3,4]. For logistic reasons, the study area was limited to a radius of about 100 km from Adelaide. Data collection ran for a six month period from August 1989 to January 1990 inclusive.

2.2 Case Investigation Protocol

The South Australian Coroner requires that an autopsy be carried out in all deaths due to traffic crashes occurring in South Australia. Whenever a fatal car crash occurred in the study

area, a Unit staff member attended the victim's post-mortem examination, and recorded the presence of internal and external injuries and various body measurements.

Non-fatal cases were identified through regular contact with the staff of the State's principal neurosurgical centre at the Royal Adelaide Hospital (RAH), to which most severely head-injured adults are admitted, and with the associated Adelaide Children's Hospital (ACH), to which most severely head-injured children (0-14 years) are admitted. Those severe head injuries managed at other metropolitan hospitals usually also came to our notice because RAH staff provide neurosurgical cover for these hospitals. Whenever a patient was admitted with a severe head injury, as defined above, after a car crash, careful note was made of external body marks and other injuries in the same manner as at autopsy for the fatally injured cases. Brain injury was documented according to clinical, computerised tomographic (CT) and magnetic resonance imaging (MRI) findings.

The vehicles involved in the crash were examined in detail. Special note was made of signs of occupant contact with the vehicle interior. The external damage was measured and recorded in sufficient detail to enable use of the CRASH 3 program to calculate the change in velocity occurring to each vehicle in the crash [5]. The crash sites were visited and mapped. A careful record of skid marks, pavement scrapes and vehicle final rest positions was made.

2.3 Case Analysis

Detailed biomechanical analysis was carried out in those cases in which the vehicle impact velocity was known, the site of head contact on the vehicle was identified and the location of the impact on the head was established.

The velocity of the vehicle at impact was estimated by various means including estimations based on the radius of curvature of centrifugal pre-impact skids, the length of braking skid marks and pre- and post-impact trajectories. Eye witnesses provided further information in some cases. The CRASH 3 program was used to calculate the change in velocity or delta V of each vehicle. From this body of information, the victim's head impact velocity was estimated. This estimate was based on physical review of the events in the crash, including the timing of the head contact, the likely trajectory of the head, restraint usage and the extent of intrusion into the occupant compartment. The surface contacted by the occupant's head could often be identified by the presence of a dent and/or by adherent hair, fat or blood. In most cases the surface was one of the interior structures (B-pillar, header or roof side rail). There were some cases where external structures were contacted, eg bonnet of the striking car or an intruding tree or pole.

A combined stiffness for the skull and struck surface was estimated using a procedure similar to that described in an earlier paper [1]. For example, the interior surface of the B-pillar was classed as "hard" (600,000 N/m) and the inner surface of a door was classed as "medium" (360,000 N/m). Side window glass was classed as "soft" (160,000 N/m) because the very high force required for fracture of the glass acts for only a very short time and the effect was assumed to be equivalent to that of a less stiff metal surface.

The location of the impact point on the head was determined from the external markings on the scalp (lacerations, abrasions and bruises) and on the location of subgaleal bruising sometimes found on reflection of the scalp. Radiological findings (scalp swelling, fractures) were often helpful. The impact location was coded according to the coordinate system devised by Ryan et al [1] .

The head impact velocity, stiffness of the contacting surface, and impact location on the head were then used to calculate linear and angular acceleration of the head, according to the equations given in Figure 1 in a manner similar to that used in our pedestrian impact study [1]. It was difficult to estimate the head impact velocity relative to the object struck because often the latter was itself being deformed in the crash. However it is thought that the resulting acceleration estimates are likely to be conservative. It was assumed in most cases that the impact of the head was normal to the surface of the vehicle interior or other object. The exceptions were one or two cases where the impact was obviously tangential, as in contact with the lower edge of the header area above the windscreen. In these cases the resultant force normal to the skull was estimated and used in the subseqent calculations.

2.4 Neuropathological Examination

The extent of the victim's head injury was established either from autopsy or by clinical investigation. In fatal cases, the brain was removed as a whole, fixed in formalin and examined according to our established neuropathological protocol [6,7]. Briefly, this entails transecting the upper midbrain and slicing the cerebral hemispheres in the coronal plane at 10 mm intervals for macroscopic examination and photography. The hemisphere and brainstem slices are then embedded in paraffin and sectioned for staining with Weil's method for myelin and haematoxylin-eosin; the cerebellar hemispheres are sectioned in the parasagittal plane, and stained in the same manner. Representative transverse sections of the brainstem and corpus callosum are also stained with the Palmgren method.

In the present study, the brains were studied by one or more of three pathologists (PCB, GS, DAS) and lesions were recorded on standard coronal diagrams of the cerebral hemispheres (usually 11 diagrams) brainstem (6 diagrams) and cerebellum (2 diagrams). In cases with evidence of localized traumatic lesions, the neuropathological findings were further recorded on summary diagrams giving a three-dimensional presentation of the cerebral hemispheres (Figure 2) to permit coding of lesion location for storage in a computer file. These summary diagrams have a total of 105 sectors over the 11 coronal sections of the cerebral hemispheres. The present configuration of the sectors differs somewhat from that presented in 1989 [1]. The changes have been made to allow for more specific identification of traumatic lesions in the corpus callosum and basal ganglia and to conform more closely to the vascular beds.

A measure of the extent of injury to the cerebral hemispheres is obtained by counting the number of sectors in which there is evidence of one or more haemorrhagic lesions. This number, which has a maximum value of 105 (the total number of sectors), we have called the Haemorrhagic Lesion Score (HLS). Note that it is a measure of the extent of injury, not necessarily the severity, although in most cases the extent and severity are likely to be closely correlated.

In cases surviving long enough to be admitted to hospital, clinical details were recorded for the purpose of this study. Copies of CT scans (obtained with General Electric 8800 scanners) and MRI scans (obtained with a Siemens 1 Tesla Magnetron) were examined and, where relevant, traumatic abnormalities were recorded on the above diagrams. The increased or decreased density and signal intensity associated with each abnormality was noted.

In the fatal cases diffuse axonal injury and anoxic cerebral damage were recorded but attention was focused on focal lesions, notably haemorrhagic lesions and lacerations. To enable reasonable comparisons to be made between brain injury patterns in surviving and fatal cases only full thickness haemorrhagic lesions were coded on the summary diagrams for the fatal cases. This was done on the assumption that such lesions could be expected to be evident, in most cases, on CT and/or MRI scans. The validity of this assumption is being tested in a separate study.

3 RESULTS

3.1 Data Collection

Data collection ran over a six month period from 1 August 1989 to 31 January 1990. Twenty-two crashes were studied over the period. Seventeen of these crashes entered the study on the basis of a fatal injury to at least one vehicle occupant, whilst the remainder included a seriously head injured occupant who survived. There were 19 fatal and 7 seriously head injured occupants in the 22 crashes. The fatalities were all adults; of the seven who survived, five were adults and two were children. There were 26 occupants receiving other types of injuries and 14 non-injured occupants.

During the study period 116 road traffic crash deaths occurred in South Australia as a whole. Sixty-nine of these road users were vehicle occupants, but 45 of them died in crashes outside the study area or were occupants of heavy vehicles. Twenty-four died as car occupants in crashes in the study area and 19 of these were investigated. Thus the series represented 79% of eligible fatal cases occurring during the study period. It was more difficult to assess the completeness of the sampling of serious head injury cases, there being no complete register of these. It was thought, however, that most eligible surviving cases were included in the study.

3.2 Types of Crash

Geographically, the 22 crashes were equally divided: 11 occurred in metropolitan and 11 in rural locations. Table 1 relates seating position to type of impact. Nearly all cases were front seat occupants: 13 were drivers, 10 were front seat passengers and 3 were rear seat occupants. There were 10 frontal impacts, (counting as two impacts a crash in which both cars contained index cases), 12 side-impacts, and one rollover with ejection.

Eight crashes occurred at an intersection; in six of these the case occupant's vehicle was struck in the side by another car or truck. In the other two, the case occupant was unrestrained and died when the front of his vehicle struck the side of another car.

Five crashes resulted from loss of control on a straight section of road. In two crashes the car left the road travelling sideways and struck a utility pole, in another two the car struck a tree or pole head on, and in the fifth crash the case car was struck on the side by an oncoming car. Four crashes involved loss of control after running wide onto a dirt shoulder whilst cornering. One resulted in rollover, one a side impact with a tree, one a side impact with a roadside object and one a frontal collision with an oncoming car. Three crashes resulted where the car ran wide on a bend and crashed head on into a tree or pole, and one when a car continued on at a T-junction, striking an earth bank. The remaining case involved a collision between a car and a truck which turned across its path.

3.3 Clinical Findings

There were 20 males and 6 females. It is noteworthy that the surviving cases were all aged less than 30 years, compared with 6 of 19 fatal cases. Three of the latter were aged over 50 years.Of the 19 deaths 12 were either dead when found at the crash site or on arrival at hospital, and 7 died in hospital at intervals of from one to four days. Of these fatally injured persons, seven had injuries to the other body regions sufficient to cause death (AIS $\geq$ 5) [8], and one had traumatic amputations of an arm and a leg.

Seven patients survived after varying periods in coma and have recovered either with no disabilities, or with disabilities which do not preclude independent life. All underwent CT scans, and three also underwent MRI scans. These investigations showed evidences of primary brain injury in six cases; the seventh had no CT evidence of cerebral injury and although in coma when admitted, his recovery was so rapid and complete that his cerebral injury was clearly not of great severity; this case is of questionable relevance in the study but is included here because the lowest recorded coma score was 7/14.

3.4 Distribution of Injury and Location of Impact

The location of the impact sites on the head was correlated with the neuropathological findings for all 26 cases; a more detailed study was made of the 17 cases in which impact accelerations were estimated. The position of the impact on the head in the horizontal plane was recorded on a clockface. Impacts at 11, 12 and 1 o'clock were classed as frontal impacts, at 2, 3, 4 and 8, 9, 10 o'clock were classed as lateral, and impacts from 5, 6 and 7 o'clock were classed as occipital.

In the 26 cases there were 15 frontal, seven lateral and three occipital impacts. In one case it was not possible to determine the orientation of the impact. Of the 15 frontal impacts, four showed no significant brain damage, and two showed only non-focal anoxic change. Nine showed contusional brain damage: in six this was localized to the frontal or frontal and temporal lobes and in three there was more diffuse haemorrhagic injury, defined as damage in five or more cerebral lobes. Of the seven lateral impacts, one showed no significant cerebral injury, two showed localised frontal or temporal lesions and four showed diffuse haemorrhagic injury. Of the three occipital impacts, two showed multifocal contusional lesions and one frontotemporal lesions.

Cases with estimated head acceleration due to impact

In 17 cases it was possible to estimate linear and angular accelerations for the impact to the head. Eleven of these cases were fatal, six were non-fatal. There were 9 frontal impacts to the head (5 non-fatal), 6 lateral impacts (1 non-fatal) and 2 occipital impacts (both fatal) (Table 2). The estimated linear acceleration ranged from 1000 m/s^2 to 5800 m/s^2in non-fatal cases, and from 1800 m/s^2 to 6600 m/s^2 in fatal cases. Estimates of angular acceleration ranged from 14000 rad/s^2 to 43000 rad/s^2 in non-fatal cases, and from 6000 rad/s^2 to 50000 rad/s^2 in fatal cases.

It can be seen from Table 2 that lateral and occipital impacts were associated with fatal outcomes to a much greater extent than frontal impacts, although the range of accelerations experienced was very similar.

Site of head impacts

The objects struck by the head are set out in Table 3. It is obvious, and to be expected, that those parts of the vehicle at about head height for a seated occupant were most frequently the sites of impacts. The roof side rail, B-pillar and header above the windscreen and the door, including glass, were the most frequent objects struck. The impact with the steering wheel hub occurred when the steering wheel and column intruded upwards in a frontal impact, and the hub was struck by the driver's face, she was restrained by a lap sash seat belt (Case 11.1). In another case, the head of a front seat passenger struck the bonnet of the striking car through the side window during the impact on the passenger's side door. The head impact with the steel and concrete pole occurred in an offset frontal impact with extreme intrusion. Four impacts with the roof side rail were fatal, as were two of the impacts on B-pillars. Estimated head impact velocity ranged from 25 to 60 km/h with estimated linear accelerations ranging from 1900 m/s^2 to 4800 m/s^2.

Frontal impacts to the head

Of the 9 frontal impacts, injury to the brain was observed in three non-fatal and one fatal case. Injuries were found in the frontal region (two cases) and anterior vertex (one case) of the non-fatal cases and in the frontal, inferior and temporal brain regions of the fatal case (Figures 3, 4, 5). Estimated linear accelerations experienced in these impacts ranged from 1000 m/s^2 to almost 6000 m/s^2. There was no clear association between the presence or extent of cerebral injury and the estimates of linear or angular acceleration for these frontal impact cases.

Lateral impacts

Of the six lateral impacts, brain injury was observed in four cases (one non-fatal, three fatal). In the non-fatal case injury was seen in the frontal region. In the fatal cases injury was widespread, all throughout the cerebral hemispheres (Figure 6). There was a tendency for the presence and extent of cerebral injury to be associated with higher levels of acceleration (Figures 7 and 8).

Occipital impacts

Both occipital impacts were fatal; brain injury was observed to be remote from the occipital region in each case (Figure 9). The linear accelerations were estimated to be about 3000 m/s^2 and the angular about 20000 rad/s^2 (Figures 10 and 11).

Skull fractures and brain injury

Brain injury can occur immediately below a fracture due to the in-bending of the skull at the time of impact and the movements of the bone edges on either side of the fracture line. Fractures occurred in 7 of the 17 cases in which accelerations were estimated. In one non-fatal case (08.1) a frontal fracture was associated with frontal injury over three brain sectors. The remaining 6 fractures occurred in fatal cases, one frontal, four lateral and one occipital. In only

two of the four lateral cases were the fractures obviously associated with brain injury. In case 15.1 extensive fracturing of the base and vault were judged to have affected 32 brain sectors. In case 05.2 a fracture was associated with injury in 9 sectors. Therefore, in the three cases (08.1, 15.1, and 05.2) injury judged to be associated with a fracture amounted to 3 of 9 injured sectors, 32 of 101, and 9 of 56, respectively. In the remaining four cases of skull fracture there was no association with brain injury. In two cases there was no injury to the brain at all (12.1, 19.1) and in two cases the fracture was remote from the brain injury (18.1, 22.1).

It appears that fracture of the skull is not invariably associated with brain injury. It also appears that, in this series, skull fractures were more prevalent in lateral impacts (4/6) than in frontal impacts of similar severity (2/9).

4 DISCUSSION

This pilot investigation of head injury in car occupants has used the multidisciplinary methods which have been applied previously by the NHMRC Road Accident Research Unit to the study of head injuries in fatal pedestrian cases [1]. These methods are similar in some respects to those used by Ommaya and Digges [9].

In this study we have included non-fatal head injury cases. This has meant that in these cases we have had to rely on the brain injury information which is provided by CT and MRI scans rather than the neuropathological data available in fatal cases. These radiological processes appear to detect only the larger traumatic lesions, although they can be located with reasonable accuracy and these locations represented on the standard brain section diagrams. As noted above, in this paper we have included only the more severe haemorrhagic lesions in the fatal cases in an attempt to ensure reasonable comparability between the sensitivity of detection of lesions in the non-fatal and fatal cases. The validity of this approach is currently being examined by comparing the injury information available from radiological and neuropathological sources in a small group of head injury cases who died after admission to hospital.

Although the number of cases in this study is small, it is clear that frontal impacts to the head form a much higher proportion of all head impacts among car occupants than among pedestrians, with a correspondingly smaller proportion of occipital impacts. However, the frontal impacts were not associated with very extensive injury to the brain, regardless of the severity of the impact within the range of severities studied (up to an estimated 6000 m/s^2). Unlike the frontal impact cases, there was some indication of a positive correlation between the estimated linear and angular accelerations and the extent of brain injury in lateral impacts.

The most injurious impacts were to the sides of the head; they were more likely to be fatal and to result in fractures of the skull than were frontal or occipital impacts. Although brain injury in the immediate proximity of a skull fracture may account for one-third to one-half of the brain lesions found in perhaps half of the cases observed this does not adequately explain the higher severity of injury in these lateral impact cases. The higher case fatality rate in the lateral head impact group could also not be attributed to fatal injuries to other body regions nor to higher estimated accelerations of the head. The roof side rail and the B-pillar were associated with most of the fatal lateral impacts, with estimated head impact velocities, relative to the struck area, ranging up to 60 km/h. If these findings can be confirmed by further investigations, which are underway in our Unit, then a strong case will exist for the improvement of head protection during impacts with the sides of the interior of the passenger compartment.

There was no detectable brain injury in five of the 11 fatal cases for which a head acceleration was estimated. In two of these cases there was a fatal injury to another body region but even in these two cases the estimated linear acceleration of the head was substantial (1900 and 4400 m/s^2). In the other three cases the estimated linear accelerations ranged from 1800 to 2900 m/s^2. Three of the five cases involved frontal impacts to the head, including the one which resulted in an acceleration of 4400 m/s^2. The other two were lateral impacts, both of which were associated with skull fractures. This absence of injury is not based solely on the detection of severe lesions, as with one exception (case 11.1, in which there were minor petechial lesions and anoxic damage) the brain sections were normal. Therefore at this stage we have no explanation to offer for the lack of detectable brain injury in these cases.

5 CONCLUSION

The results of this pilot study of head injury to car occupants shows that the location of the impact on the head is clearly important, the outcome after frontal impact being in general more favourable than after impact to the side of the head. This may, subject to confirmation in further investigations, be found to indicate a greater need for head impact protection on the sides rather than the front of the passenger compartment.

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TABLE 1: Seating position and type of impact for car occupants

Type of Occupant	Type of Impact				
	Frontal	Side Impact Same	Side Impact Opposite	Rollover	Total
Driver	7	2	3	1	13
Left Front Passenger	2	7	1	0	10
Rear Passenger	1	2	0	0	3
Total	10	11	4	1	26

TABLE 2: Head impact location, accelerations and brain injury for fatal[1] and non-fatal cases

	NON FATAL				FATAL			
Impact Location	Case No	Acceleration Linear m/s^2	Acceleration Angular rad/s^2	HLS[2]	Case No	Acceleration Linear m/s^2	Acceleration Angular rad/s^2	HLS
FRONTAL	08.1	1000	15000	9 +	17.2	1800	14000	0
	17.1	1800	14000	0	11.1	2900	22000	0
	19.2	2300	18000	0	22.1	3900	19000	11 +
	23.1	4300	30000	6	06.1	4400	33000	0 x
	09.1	5800	43000	5				
LATERAL	03.1	2800	21000	7	12.1	1900	19000	0 + x
					19.1	2000	6000	0 +
					07.1	3000	23000	55
					15.1	4800	24000	101 +
					05.2	6600	50000	56 +
OCCIPITAL					18.1	2900	22000	9 + x
					05.1	3300	16000	20

1 Death not necessarily due to head injury in all cases.
2 Haemorrhagic Lesion Score.
\+ Skull fracture.
x Fatal injury to other body region.

TABLE 3: Objects struck in 17 head impacts

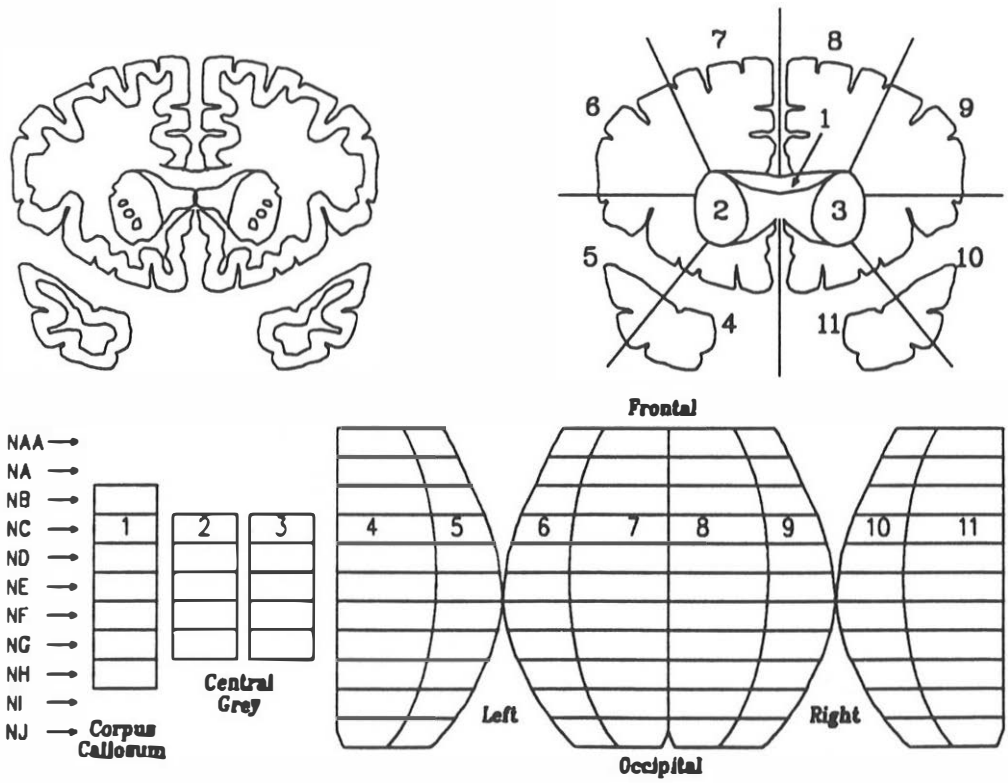
Object	N
Roof side rail	5
Door and/or glass	3
B-pillar	3
Header	3
Steering wheel hub	1
Steel and concrete pole	1
Bonnet of striking car	1

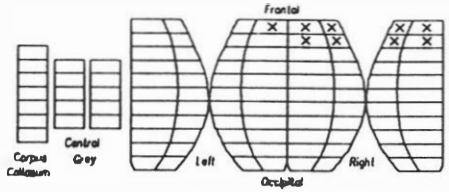
FIGURE 1: Summary of head impact calculations

(1)	a	=	$v\sqrt{K/M}$	m/s^2	
(2)	F	=	Ma	N	
(3)	α	=	$\frac{Fx}{I}$	rad/s^2	
(a)	M	=	0.0306 (T) + 2.46	kg	
(b)	I	=	0.02	kg m^2	
(c)	K	=	600,000	N/m	(hard surface)
			350,000	N/m	(medium surface)
			140,000	N/m	(soft surface)

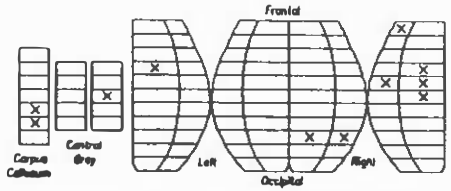
a - Maximum linear acceleration of head due to impact
F - Maximum force on head due to impact
α - Maximum angular acceleration of head due to impact
v - Velocity of head at impact
K - Combined stiffness of the head and impacted surface
M - Mass of the head
x - Offset of force vector from head centre of gravity
I - Moment of Inertia of the head
T - Total body mass of the occupant

FIGURE 2: Coding of brain injury

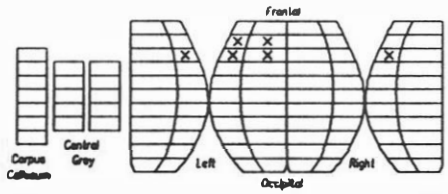




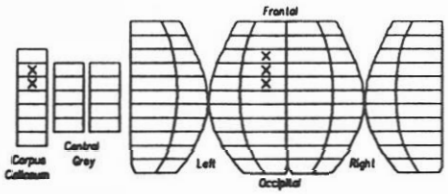
Case 08.1 Non fatal, 1000 m/s², 15000 rad/s²



Case 22.1 Fatal, 3900 m/s², 19000 rad/s²



Case 23.1 Non fatal, 4300 m/s², 30000 rad/s²



Case 09.1 Non fatal, 5800 m/s², 43000 rad/s²

FIGURE 3: Frontal impacts: distribution of haemorrhagic lesions

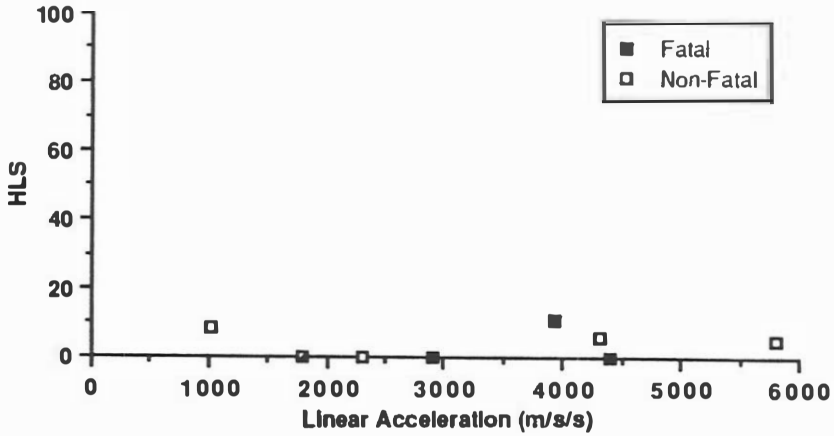


FIGURE 4: Haemorrhagic Lesion Score (HLS) by linear acceleration: frontal impacts

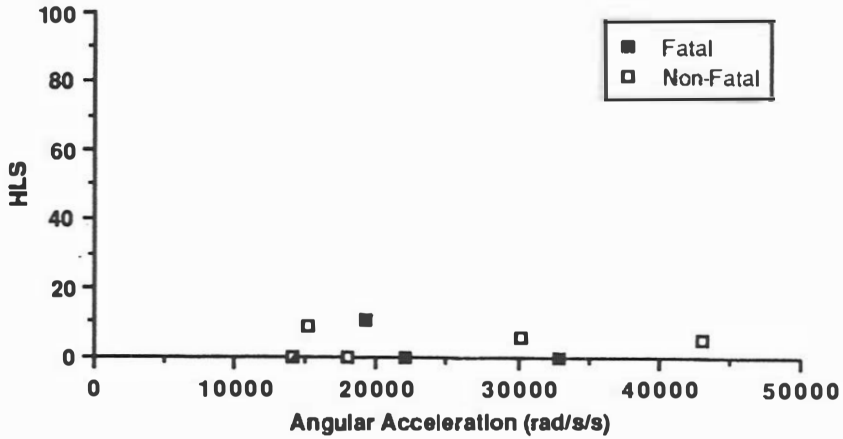
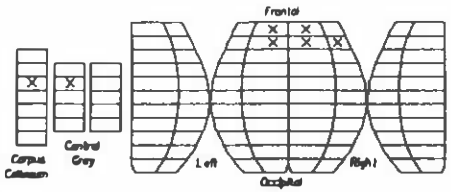
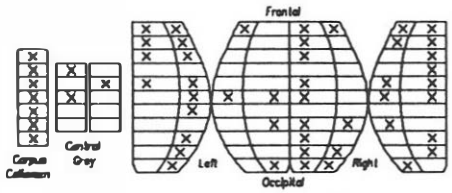


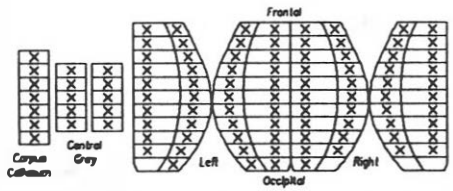
FIGURE 5: Haemorrhagic Lesion Score (HLS) by angular acceleration: frontal impacts



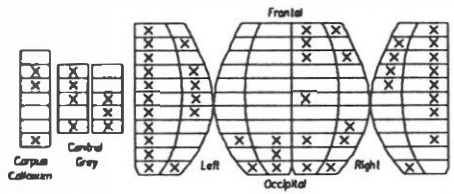
Case 03.1 Right impact, non fatal, 2800 m/s², 21000 rad/s²



Case 07.1 Left impact, fatal, 3000 m/s², 23000 rad/s²



Case 15.1 Right impact, fatal, 4800 m/s², 24000 rad/s²



Case 05.2 Left impact, 6600 m/s², 50000 rad/s²

FIGURE 6: Lateral impacts: distribution of haemorrhagic lesions

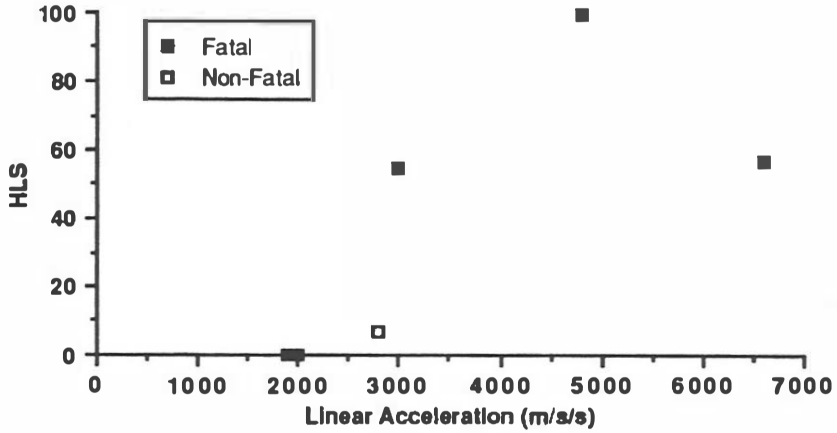


FIGURE 7: Haemorrhagic Lesion Score (HLS) by linear acceleration: lateral impacts

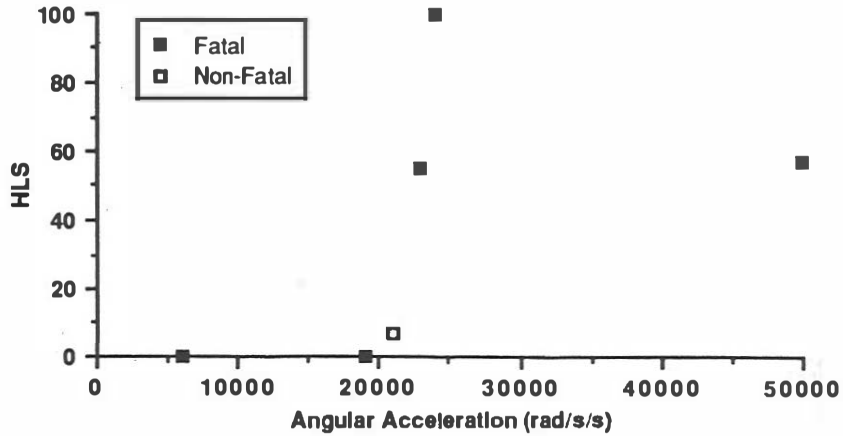
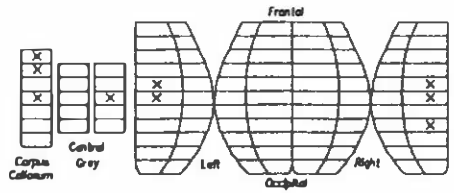
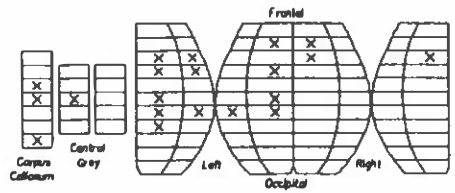


FIGURE 8: Haemorrhagic Lesion Score (HLS) by angular acceleration: lateral impacts



Case 18.1 Fatal, 2900 m/s², 22000 rad/s²



Case 05.1 Fatal, 3300 m/s², 16000 rad/s²

FIGURE 9: Occipital impacts: distribution of haemorrhagic lesions

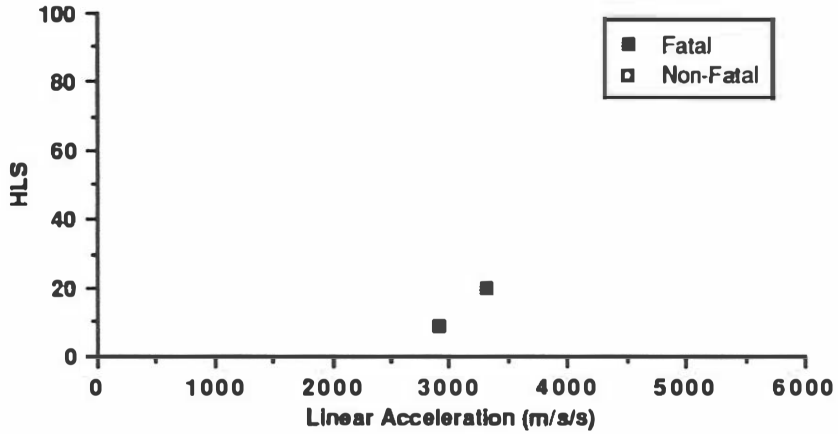


FIGURE 10: Haemorrhagic Lesion Score (HLS) by linear acceleration: occipital impacts

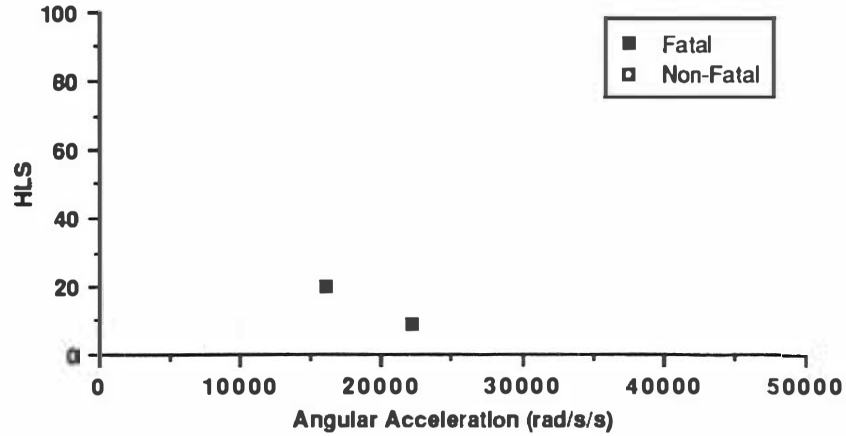


FIGURE 11: Haemorrhagic Lesion Score (HLS) by angular acceleration: occipital impacts