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HEAD INJURY IN CAR OCCUPANTS: REPORT ON A PILOT STUDY

BR Paix[1], PC Blumbergs[2], CN Kloeden[3], AJ McLean[3]

GA Ryan[3], G Scott[2] and DA Simpson[3]

1 NHMRC Road Accident Research Unit, University of Adelaide (Now at Accident and Emergency Department, Modbury Hospital, Adelaide).

2 Head Injury Laboratory, Division of Tissue Pathology, Institute of Medical and Veterinary Science, Adelaide.

3 NHMRC Road Accident Research Unit, University of Adelaide, Adelaide, South Australia.

INFORMATION RETRIEVAL

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ABSTRACT: The neuroradiological and neuropathological data from 26 killed or severely injured car occupants have been quantitated using a novel system of diagrammatic representation suitable for storage in a computer file. These data have been correlated with the biomechanical investigation of the 22 road crashes in which these persons were injured. In 17 cases it was possible to estimate head impact accelerations; linear accelerations were in the range 1000-6600 m/sec^2, and angular accelerations in the range 14 000-50 000 rads/sec^2. A positive correlation between incidence of haemorrhagic brain injury and head impact acceleration appeared to be present when impact sites were in the lateral and occipital parts of the head, but not after frontal impacts, which in general were clinically less injurious. The injuries were reviewed from the preventive viewpoint: there was little to suggest that better clinical management would have improved the outcome, but there were possibilities for injury mitigation by improved head protection within the passenger compartment.

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CONTENTS

1 INTRODUCTION

Head injury is acknowledged to be one of the most important causes of morbidity and death in traffic accidents. In Australia, as in other motorized countries, deaths from traffic accidents involving the drivers and passengers of cars outnumber all other road deaths, and Selecki et al. (1981) showed that head injuries accounted for about 60% of all deaths of car occupants.

Car occupants may suffer head injuries from impact by the road surface, if they are ejected; they may suffer impacts in the interior of the car, against passenger compartment pillars, the windscreen, the steering wheel, and other structures, or they may be injured by impact with some object intruding into the interior of the car, such as another car. Numerous efforts have been made to establish the tolerance of the brain, in order to design appropriate head protection.

1.1 Human tolerance to head injury impact

There are many difficulties in making quantitative estimates of impact severity and in relating these to brain injury. Humans are quite variable. There is a considerable range of "normal" values for virtually any measurable biological parameter. Some of these, such as age, head mass, scalp thickness and skull strength are likely to influence the degree of injury produced by a given insult. Brain injury includes a spectrum of different processes, and is not a single measurable entity. Impact to the head can produce direct local damage, such as cerebral laceration or contusion. It can also produce remote "inertial" damage such as diffuse vascular injury (DVI) or diffuse axonal injury (DAI). There may also be secondary damage due to anoxia, bleeding or oedema following the initial trauma. There may be multiple mechanisms of injury resulting from a single impact. Rotational and linear acceleration may both be generated by a single impact and may each tend to produce different types of brain injury.

Ethical and practical considerations place major restrictions on the design of studies of impact tolerance. It is usually necessary to try to interpret naturally occurring experiments on living humans (i.e. crash victims), or to extrapolate from other injury models. Such extrapolations from surrogates for the living human head introduce their own uncertainties.

Cadavers are reasonably similar anatomically to living humans but cadaver tests can produce only "anatomical" results, such as skull fractures. They cannot be assessed for "physiological" deficits such as duration of unconsciousness nor can their injuries be accentuated or alleviated by secondary physiological processes. They also lack the muscle tone and protective reflexes of living subjects.

Mechanical dummies or "Anthropomorphic Test Devices" (ATDs) are only approximations of living subjects and cannot experience physiological changes in response to impact. Subhuman primates are living systems but are only approximations to human anatomy: there are anatomical differences and scaling considerations due to factors such as body mass and brain size.

Human volunteer experiments must be performed at the lower end of the tolerance scale and thus are not direct representations of the results of higher severity impacts.

Indeed, the problems of surrogate experiments and real world analysis are opposed. Surrogate experiments generally involve applying a known trauma to a surrogate head, and then extrapolating the observed damage to produce a damage prediction for the human system. Real world analysis begins with detailed damage data and attempts retrospectively to estimate the trauma necessary to produce the damage.

1.2 Established estimates of head impact tolerance

The Wayne State Tolerance curve was an early scale relating impact severity and head injury (Lissner et al. 1960; Gurdjian 1972). It principally related skull fracture to linear acceleration of the cadaver head in the anteroposterior (AP) direction. From it has arisen a number of mathematical equations for head injury. Perhaps the most widely used is the Head Injury Criterion (HIC) which is measured for regulatory purposes in US barrier crash tests. It should be noted that it is based on linear acceleration of the head in the AP direction. In practice, its correlation with real life injury outcome appears to be poor (Newman 1986; Goldsmith 1989), though it has recently been defended by Hopes & Chinn (1989) on pragmatic grounds.

A more recent scale, the Mean Strain Criterion (MSC) was produced by extrapolating injuries produced in primate experiments to produce human tolerance levels (Stalnaker et al. 1971, 1987). It is as yet unknown whether this scale provides better correlation with real life tolerance than the HIC.

1.3 In-depth analysis of head injury mechanisms in fatally injured pedestrians

Since 1982, the NHMRC Road Accident Research Unit has been attempting to refine head injury tolerance data by in-depth analysis of real world pedestrian accidents. Such a strategy avoids the limitations of cadaver, dummy and primate experiments, but faces difficulties of its own. The principal source of uncertainty is error in estimation of head impact forces. The basic strategy of this pedestrian study was to collect detailed injury, vehicle and site data for each pedestrian accident and to use these data to reconstruct the accident biomechanics. The method has already been described in detail by Ryan et al. (1989).

1.4 Estimation of head impact severity in car occupants

It was decided that our pedestrian accident study protocol could be modified and applied to car occupant cases. We proposed to use the same basic strategy of in-depth evaluation of data from the vehicle(s), the accident site and the victim(s). We now report on a pilot study which endeavours to assess the feasibility of the proposal.

2 METHODOLOGY

2.1 Case selection

We aimed to include all cases of severe head injury or of death (from any cause) occurring to car occupants in crashes in and around Adelaide. We defined severe head injury as the record of 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 (Jennett & Teasdale 1981; Reilly et al. 1987; Teasdale & Jennett 1974). For logistic reasons, the study was confined to an area having a radius of approximately 100 km from Adelaide. Data collection ran for a six month period from August 1989 to January 1990.

2.2 Case investigation protocol

Daily telephone contact was made between a Unit staff member and the Office of the South Australian Coroner. The Coroner requires that an autopsy is 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.

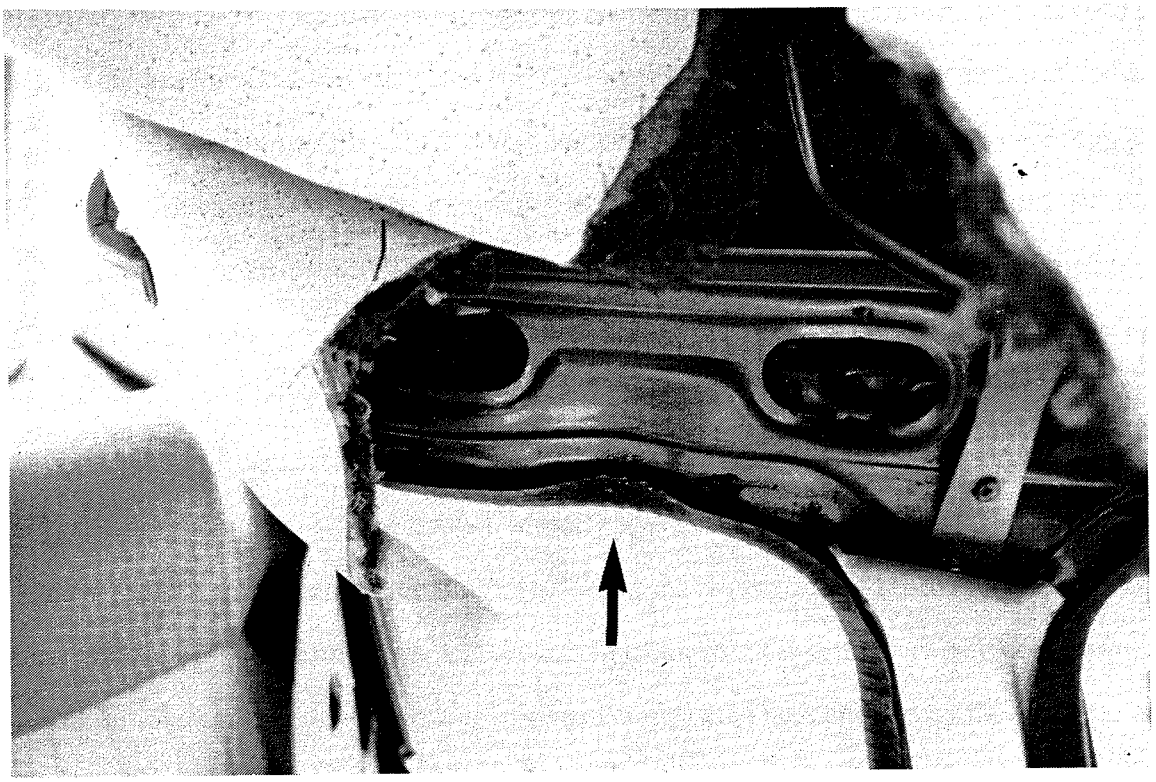
Regular contact was maintained with the staff of the State's principal neurosurgical centre, the Royal Adelaide Hospital, to which most severely head-injured adults are admitted, and with the associated Adelaide Children's Hospital, to which most severely head-injured children (0-14 years) are admitted. The few severe head injuries managed at other metropolitan hospitals usually also came to our notice because RAH staff provide neurosurgical cover for these hospitals also. Whenever the victim of a car crash was admitted with a severe head injury, as defined above, careful note was made of external body marks in the same manner as at autopsy for the fatally injured cases. Brain injury was documented according to clinical, computed tomography (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. In particular, evidence of head contact with steering assembly, roof pillars, instrument panel, bonnet, header area or elsewhere was sought (Figure 1a, b). The external damage was measured in sufficient detail to enable use of a simple computer reconstruction of the crash.

The crash sites were visited and mapped. Careful records of skid marks, pavement scrapes and vehicle final rest positions were made.

FIGURE 1: Car crash investigation (Case N023).

(a) Car which left road and struck a tree on the left side (indicated by arrow).



(b) The driver's head was struck by the intruding roof rail (indicated by arrow), in the left frontal region.

2.3 Case analysis

Detailed biomechanical analysis was carried out for those cases in which sufficient information was available: this required that the vehicle impact velocity was known or estimated, the head contact site identified and the location of the impact on the head established.

The velocity at impact for vehicles was estimated by whatever means were available, and these varied from case to case. The methods used included estimations based on the radius of curvature of centrifugal pre-impact skids, the distance travelled in a fall from a height, 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 (delta V) of each vehicle (CRASH 3 user's guide and manual, 1986). 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 occupants 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 expected interior structures (A- or B-pillar, header or steering assembly), but there were several contacts with intruding external structures (eg bonnet of the other car) and several ejections with ground contact (see Table 4). A combined stiffness for skull and struck surface was estimated. The values used were those used for the pedestrian impact study (Ryan et al, 1989). For example, the interior surfaces of the A and B pillars were 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, namely lacerations, abrasions and bruises, and on the location of subgaleal bruising on reflection of the scalp. Radiological findings (scalp swelling, fractures) were helpful. The impact location was coded according to a modified form of the coordinate system devised by Ryan et al (1989). The impact velocity, stiffness of the contacting surface, and impact location were then used to calculate linear and angular acceleration of the head, according to the equations given in Figure 2, in a manner similar to that used in our pedestrian impact study. 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 (possibly two) cases where the impact was apparently 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 subsequent calculations.

FIGURE 2

a: Calculation of linear acceleration after impact:

$$a = v\sqrt{K/M}$$

where

a = maximum linear acceleration of head due to impact (m/sec^2)
v = velocity of head at impact (m/sec)
K = combined stiffness of head and impacted surface (N/m)
M = mass of head (kg) [calculated from total body mass (T):M = 0.0306(T) + 2.46]

FIGURE 2

b: Calculation of angular acceleration after impact:

$$\alpha = \frac{Fx}{I}$$

where

α = maximum angular acceleration of head due to impact (rad/s^2)
F = maximum force on head due to impact (N)
x = offset of force vector from head centre of gravity (m)
I = moment of inertia of head (0.02 kg m^2)

2.4 Neuropathological examination

The extent of the victims head injury was established from autopsy or clinical investigation. In fatal cases, the brain was removed as a whole, fixed in formalin and examined according to our established neuropathological protocol (Gibson et al, 1985; Blumbergs et al, 1989). 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 wax and sectioned; embedded coronal blocks are cut on the LKB motorized sledge microtome and smaller blocks are cut on a Leitz rotary microtome. Coronal sections are cut at 10 μ thickness and stained with Weil's method for myelin and with 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 cerebral lesions, the neuropathological findings were further recorded on antero-posterior diagrams giving a three-dimensional presentation of the cerebral hemispheres (see Appendix I: Mark III diagram) to permit coding of lesion location for storage in a computer file. This system of graphic coding distinguishes superficial and deep zones in the cerebral hemispheres in an arbitrary manner, and divides each coronal section into sectors. The superficial zones include the cerebral cortex and the subjacent white matter and are divided into 8 sectors, while the deep zones constitute three: one which includes the corpus callosum, and two which represent the right and left halves of the supratentorial central grey matter (basal ganglia and diencephalon). Since the corpus callosum and central grey nuclei are not present in the frontal and occipital poles, the representative sectors appear respectively in only seven and five coronal slices. Thus, the total number of supratentorial sectors represented is $(11 \times 8) + 7 + (5 \times 2) = 105$. This method of graphic presentation of lesion distribution differs somewhat from that presented by Ryan et al (1989), which is shown in Figure 17: changes have been

made to allow more specific identification of traumatic lesions in the corpus callosum and basal ganglia, and to conform more closely with cerebral vascular anatomy. The successive forms of graphic presentation are described and compared in Appendix I. The latest (Mark IV) version of the diagram is used in Figure 3, which includes 10 sectors identifying infratentorial lesions; these were recorded but not discussed in the present report.

The severity of contusions in the cerebral cortex was recorded in three grades:

(1) haemorrhagic contusions not involving the full cortical thickness but showing more than 3 separate haemorrhages per low power field;

(2) haemorrhagic contusions involving the full thickness of the cerebral cortex;

(3) gross loss of cortex at least down to the white matter (cortical laceration).

Lesions (chiefly haemorrhagic) in the white matter and/or basal ganglia were also recorded in three grades:

(1) lesions only visible microscopically;

(2) lesions seen with the naked eye (> 5 mm diameter) in Weil stained sections and/or unstained slices;

(3) gross loss of white matter.

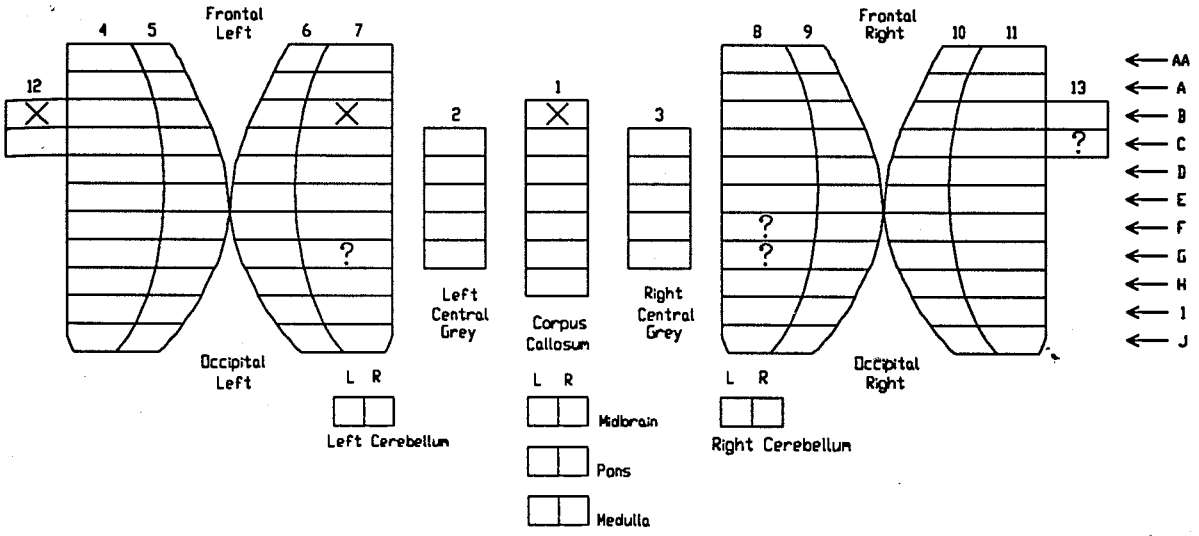


FIGURE 3a: Case 007: haemorrhagic lesions identified in CT scan on day of death

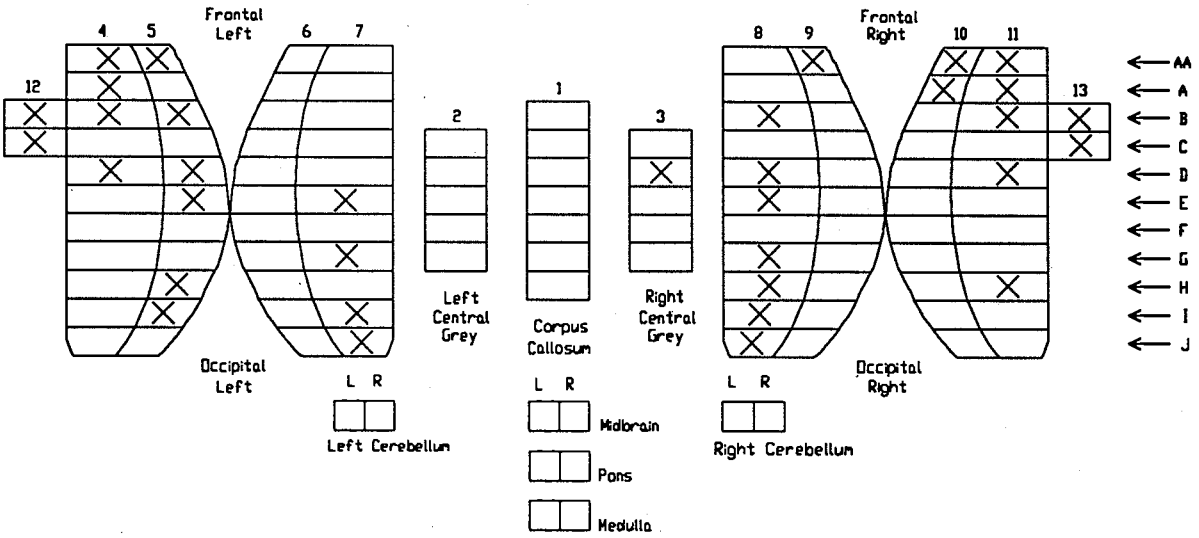


FIGURE 3b: Case 007: haemorrhagic lesions identified by autopsy

In this study, chief attention was given to vascular lesions, notably haemorrhagic contusions, and to lacerations; diffuse axonal injury and anoxic cerebral damage were recorded, but were not considered in correlations of lesion sites and impact forces.

In cases surviving long enough to be admitted to hospital, clinical details were recorded. Copies of CT scans (obtained with General Electric 8800 scanners) and MRI scans (obtained with a Siemens 1 Tesla Magnetom) were studied, and abnormalities were recorded on similar diagrams, using coloured symbols to identify increased or decreased density and signal intensity.

To obtain a quantitative measure of injury severity, we considered only haemorrhagic lesions, and to enable us to make reasonable comparisons between brain injury patterns in fatal and in surviving cases, we coded only autopsy lesions greater than 5 mm in diameter. This was done in the assumption that such lesions could usually be seen in CT and/or MRI scans: this assumption is being tested in a separate study. A cumulative measure of total brain injury was obtained by counting the number of sectors in which haemorrhagic lesions were recorded. The total of these affected sectors has been termed the Haemorrhagic Lesion Score (HLS), and since there are 105 sectors in the diagram, the maximum HLS is 105.

Thus, in autopsied cases, the HLS was determined by the systematic neuropathological examination described above; in non-fatal cases haemorrhagic lesions diagnosed by CT (± MRI) scans were enumerated in the same way, but there is under-detection of lesions by this method, as was evident in four cases in which both CT and autopsy data could be compared (Figure 3).

3 RESULTS

3.1 Data collection

Data collection ran over a six month period from the 1st August 1989 to 31st January 1990. Twenty-two crashes were included over the period. During the study period there were 116 road accident deaths 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 accidents occurring during the study period.

During the study period, we also identified five adults and two children who were car occupants in road crashes and who survived severe head injuries as defined in our protocol. It was more difficult to assess the completeness of this sampling of serious head injury cases, there being no complete register of these. It was felt, however, that most eligible surviving cases were included in the study.

3.2 Types of crash (see Appendix II)

Geographically, the crashes were equally divided: of the 22 crashes, 11 occurred in metropolitan and 11 in rural locations. 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. There were 26 occupants receiving other types of injury and 14 non-injured occupants.

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

	Type of Impact				
Type of Occupant	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

15

Table 1 relates crash type to seating position and type of crash. 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 a crash in which both cars contained index cases), 15 side impacts, and one rollover with ejection.

TABLE 2: Type of crash and nature of subsequent impacts

Type of Crash		Nature of Impact
Right angle collision at intersection	8	5 side impact with car 1 side impact with truck 2 frontal impact with car
Loss of direction control:		
- on straight road	5	2 side impact with pole 2 frontal impact with pole or tree 1 side impact with car
- on dirt shoulder in cornering	4	1 rollover 1 frontal impact with car 1 side impact with roadside obstacle 1 side impact with tree
- on curve	3	3 frontal impact with tree or pole
Other	2	1 frontal impact with embankment at T-junction 1 frontal impact with truck turning across path

Table 2 compares the type of crash and the resultant impact. 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 of these resulted in two index cases (cases 13.1 and 13.2). 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. In three crashes, a car ran wide on a bend and crashed head on into a tree or pole. The remaining cases involved a collision between a car and a truck which turned across its path, and a crash when a car continued on at a T-junction, striking an earth bank.

Twelve victims are believed to have worn seatbelts; one was ejected when the side of the car was destroyed. Thirteen are believed to have been unrestrained, and three of these were ejected. In one case, belt status was uncertain.

3.3 Role of alcohol

Data on alcohol consumption are available for only 18 of the 26 index cases: in 10 of these, levels in excess of 0.10 g/100 ml were recorded in the drivers supposedly responsible for the crashes. In two other cases, drivers had alcohol blood levels below .05 g/100 ml, but it is unclear whether this was a contributory factor. There were no record of levels in the range 0.05 - 0.10 g/100 ml.

3.4 Clinical findings

There were 20 males and 6 females. The age distribution is given in Table 3. 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) (Abbreviated Injury Scale 1990) and one had traumatic amputations of an arm and a leg. Of the cases dying in hospital, 5 underwent CT scanning and 4 of these have been reviewed and correlated with the autopsy findings.

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.

TABLE 3: Age distribution in head injured car occupants

Age (years)	Survived	Died	Total No. Cases
0-9	1	-	1
10-19	3	5	8
20-29	3	5	8
30-39	-	4	4
40-49	-	1	1
50-59	-	1	1
60-69	-	1	1
70-79	-	1	1
Uncertain	-	1	1
TOTAL	7	19	26

3.5 Pathology (see Appendix III)

Neuropathological findings

Of the 12 cases found dead at the accident site or on admission to hospital, six had major (AIS ≥ 5) thoracic and/or abdominal visceral injuries, and it is likely that these were the immediate causes of death; in one of these (017.1), the brain showed no neuropathological evidence of injury, and in two elderly victims (012 and 021) there were only minor haemorrhagic lesions. Of the seven cases dying in hospital, one (006) had a lethal cardiac injury and virtually no microscopic signs of cerebral injury. Thus, in four fatal cases, there was no neuropathological evidence of significant brain injury, though this is not to say that such injury did not occur, since submicroscopic lesions are not excluded. In three other cases it is likely that extracranial injuries were responsible for death, but there were nevertheless noteworthy neuropathological abnormalities.

Thirteen fatal cases showed multiple haemorrhagic lesions, ranging from a massive intracerebral haematoma (Figure 4a) to multiple small petechial haemorrhages (Figure 4b).

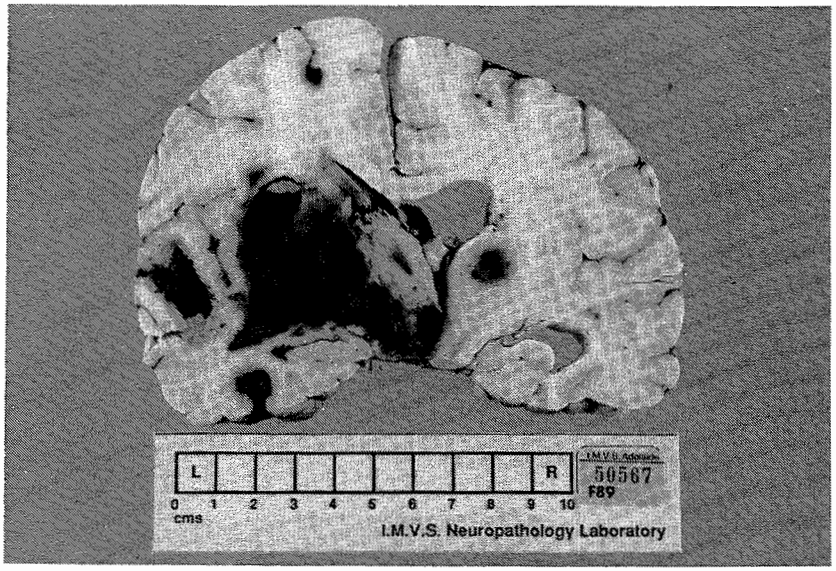


FIGURE 4a: Massive intracerebral haemorrhages in a 36 year old male car driver, who died 19 and half hours after a frontal impact (probably head contact with steering wheel).

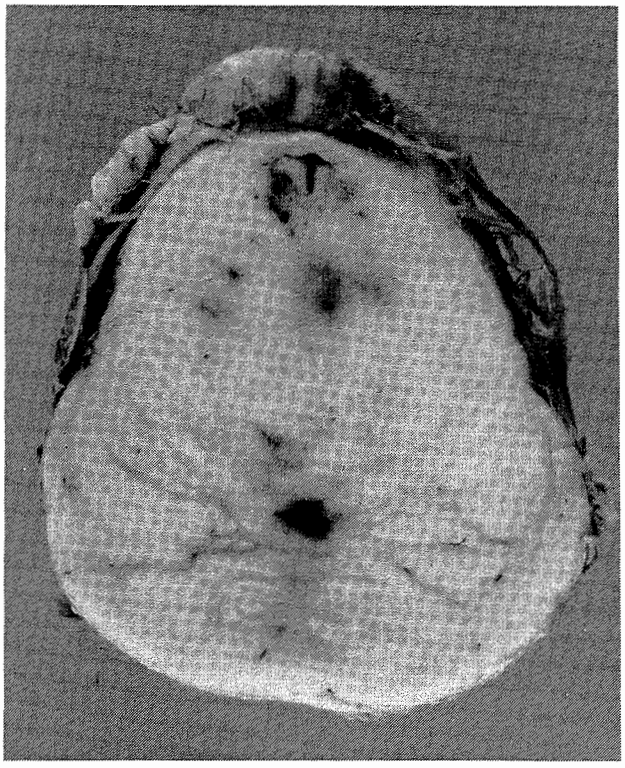


FIGURE 4(b): Scattered haemorrhages in basis pontis, pontine tegmentum and around aquaduct (same case)

If DVI is defined by a finding of small or large haemorrhages in the brainstem, corpus callosum and at least five of the eight lobes of the cerebral hemispheres, then there were 12 such cases; in 11 of these there were also haemorrhages in the basal ganglia. The impression of overwhelming cerebral damage was supported by the finding of gross lacerations in five cases: these included complete (001) (Figure 5) or partial (010) pontomedullary tears in two and a gross laceration of the midbrain and cerebellum in one (005.2).

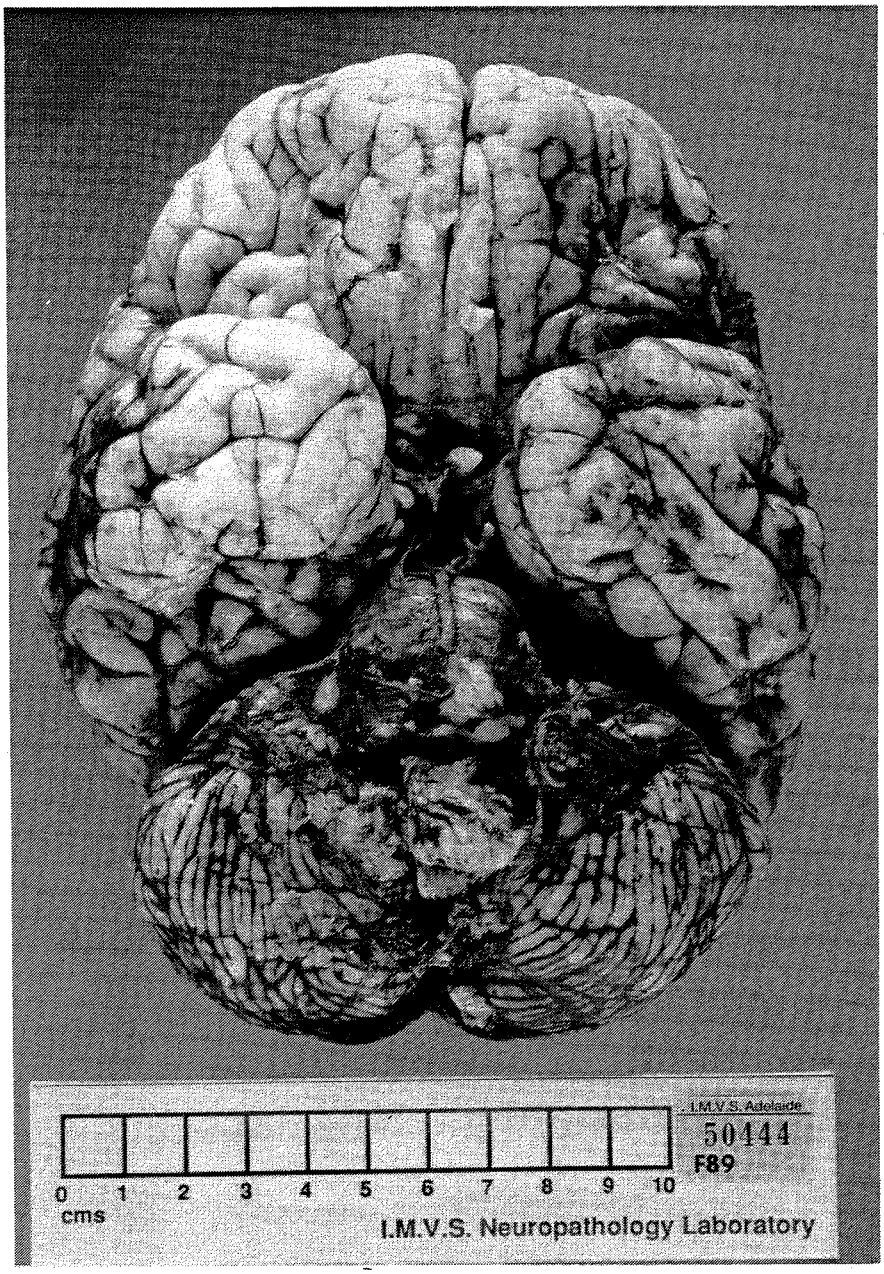


FIGURE 5: Complete avulsion of medulla in a 23 year old male car driver (case 001), who hit a tree: it is thought that he suffered an occipital impact from the head of an unrestrained rear passenger.

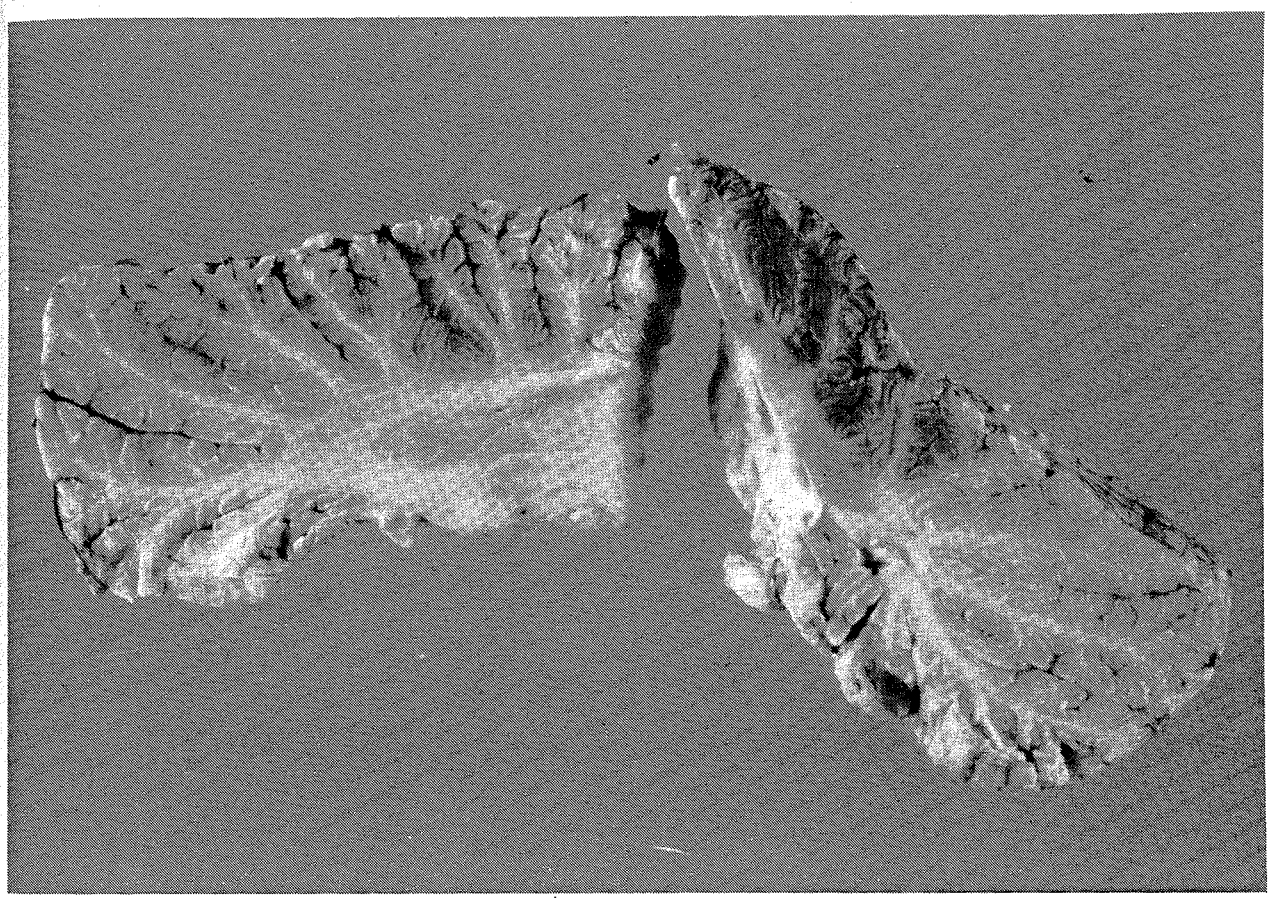
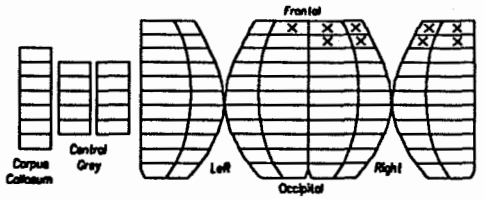
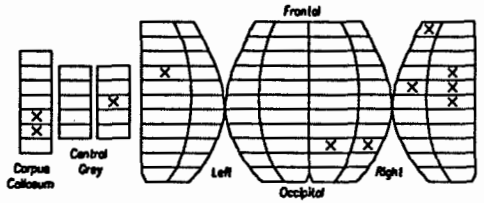


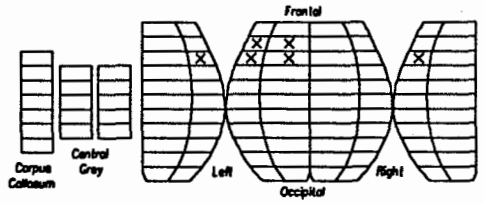
FIGURE 6: Haemorrhagic infarction of superior vermis of the cerebellum, most marked on the right, associated with hydrocephalus and clinical deterioration after a possibly survivable injury (Case 011).



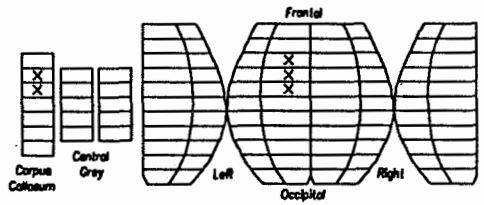
Case 08.1 Non fatal, 1000 m/s^2, 12000 rad/s^2



Case 22.1 Fatal, 3900 m/s^2, 19000 rad/s^2



Case 23.1 Non fatal, 4300 m/s^2, 30000 rad/s^2



Case 09.1 Non fatal, 5800 m/s^2, 43000 rad/s^2

FIGURE 7: Frontal impacts: distribution of haemorrhagic lesions

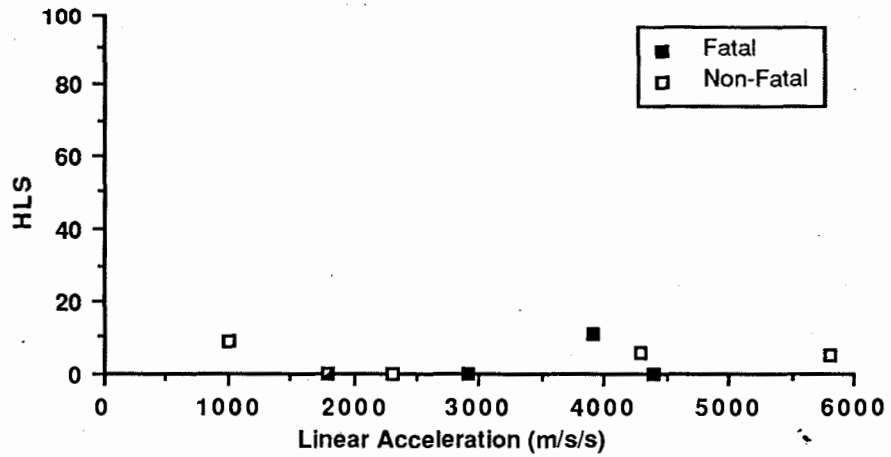


FIGURE 8: Haemorrhagic Lesion Score (HLS) by linear acceleration: frontal impacts

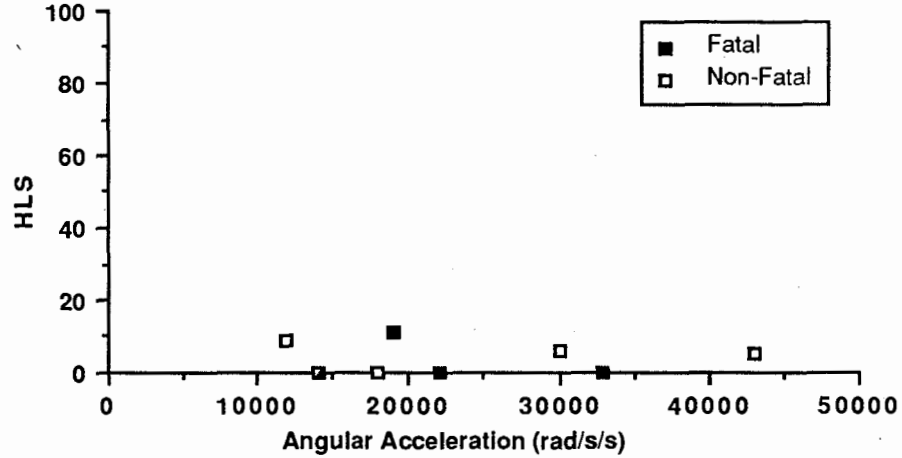
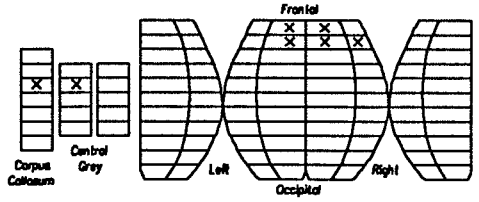
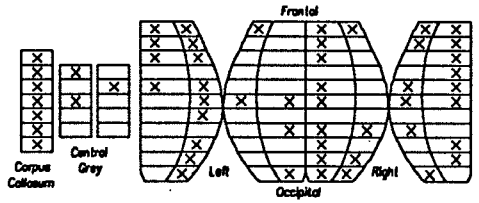


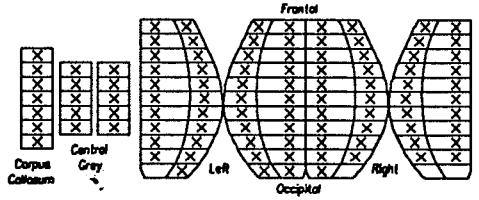
FIGURE 9: Haemorrhagic Lesion Score (HLS) by angular acceleration: frontal impacts



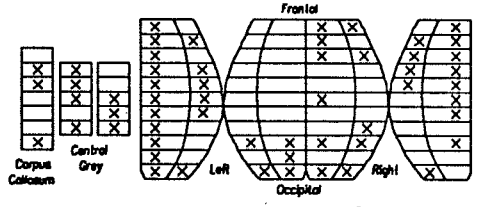
Case 03.1 Right impact, non fatal, 2800 m/s², 21000 rad/s²



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, fatal, 6600 m/s², 50000 rad/s²

FIGURE 10: Lateral impacts: distribution of haemorrhagic lesions

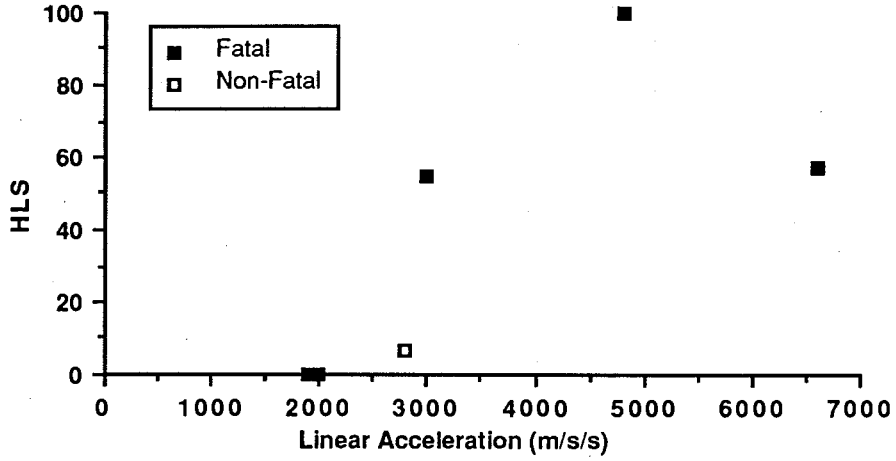


FIGURE 11: Haemorrhagic Lesion Score (HLS) by linear acceleration: lateral impacts

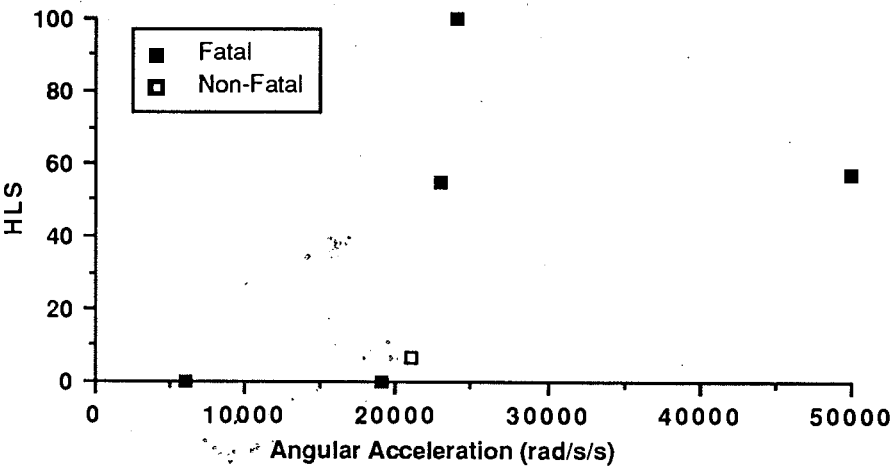
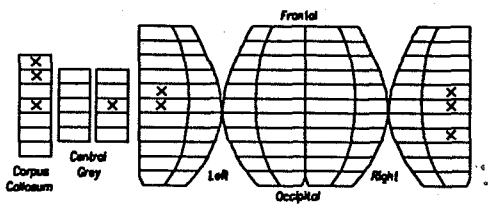
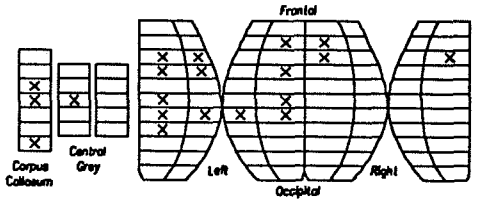


FIGURE 12: Haemorrhagic Lesion Score (HLS) by angular acceleration: lateral impacts



Case 18.1 Fatal, 2900 m/s^2, 22000 rad/s^2



Case 05.1 Fatal, 3300 m/s^2, 16000 rad/s^2

FIGURE 13: Occipital impacts: distribution of haemorrhagic lesions

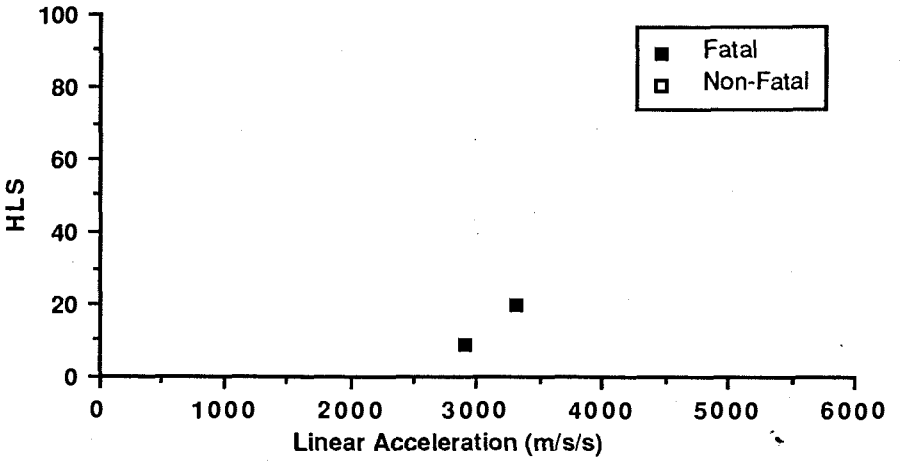


FIGURE 14: Haemorrhagic Lesion Score (HLS) by linear acceleration: occipital impacts

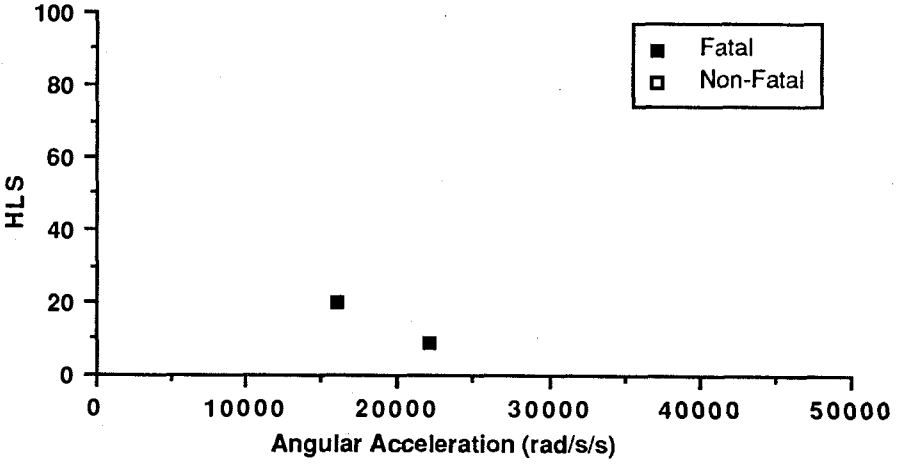


FIGURE 15: Haemorrhagic Lesion Score (HLS) by angular acceleration: occipital impacts

Brainstem haemorrhages were present in 13 cases: midbrain haemorrhages were present in all, pontine haemorrhages in 12, and medullary haemorrhages in eight: two of the later were associated with pontomedullary lacerations. The medullary haemorrhages, when present, were smaller and less striking than in the rostral brainstem.

Two patients (011 and 019.1) showed predominantly anoxic brain damage. One (019.1) survived for some 15 hours; she had had a seemingly severe head injury, with skull fracturing, but suffered an early cardiac arrest and appeared to have died from pulmonary oedema. The other survived 63 hours and can be regarded as a talk-and-die victim (Reilly et al, 1975), though it seems unlikely that she was ever really lucid: she deteriorated after a laparotomy for splenic rupture, and was then found by CT scan to have hydrocephalus which did not respond to ventricular drainage. Autopsy showed extensive anoxic damage with marked cerebellar swelling and reverse transtentorial herniation (Figure 6). The case records were incomplete, and the exact sequence of the deterioration was unclear.

Apart from these patients with possibly preventable anoxic damage, there were no autopsied cases showing significant remediable lesions. Two (001 and 005.1) had acute subdural haematomas (ASDH) and one had a massive intracerebral haemorrhage (Figure 4), but neither the clinical nor the autopsy data suggest that operation for these would have been fruitful.

Evidences of DAI were very meagre: this can be attributed to the short survival times, which did not allow progressive degeneration of injured axons to become evident with the staining methods used.

In the seven surviving cases, neuropathological data were derived from CT scans obtained soon after admission and in 3 also from MRI scans, obtained some months later. One case (019.2) had a relatively less severe head injury with no conclusive CT abnormalities. Two cases (003 and 023) showed prolonged loss of consciousness associated with CT evidence of bilateral predominantly frontal damage: these very probably suffered DAI, though the MRI findings were not so diffuse or so specific as to give this diagnosis unequivocal support. One case (009) had an acute surgically treated ASDH; CT showed only ipsilateral haemorrhagic

lesions and swelling, but MRI later showed widespread abnormalities. Three cases (004, 008 and 015.2) had localised frontolateral impacts causing depressed skull fractures: these cases showed CT evidence of frontal and/or temporal contusions clearly related to the impact site.

These were present in 10 of the 19 autopsied cases and in at least three of the seven survivors. One of the autopsied cases had a compound depressed fracture in the parietal region; three of the survivors and one autopsied case had depressed frontal fractures. The fracture patterns conformed to those usually seen after severe blunt head impacts at various sites: in eight cases the fractures were confined to the skull base, in three there was also vault involvement, and in two the fractures were in the frontal vault. Correlations of fracture sites and cerebral injury were carried out, using the schematized diagrams of lesion incidence and severity. Cortical lesions could be related to the sites of fractures in 3 of 4 lacerations, and in most of the cases with contusions. In both cases of pontomedullary tear there were basal fractures and in one of these there was also an atlanto-occipital dislocation. However, in three autopsied cases there were no signs of cerebral injury in spatial relation to the fractures.

Spinal injuries

These were recorded in three cases; in one, an atlanto-occipital dislocation, there was a pontomedullary avulsion (Figure 5).

Extracranial injuries

These were numerous. As noted above, seven of the 19 fatalities were attributed to critical (AIS $\geq$ 5) or lethal (AIS $\geq$ 6) visceral injuries; serious visceral injuries (AIS $\geq$ 3) were recorded in eight others, and in only four were there no clinically significant injuries outside the head. Similarly, only one of the seven surviving cases had no major extracranial injuries. Blood loss and pulmonary oedema were contributory causes of death in the two cases of anoxic cerebral damage, and probably so in a third case with contusion injury of the brain.

3.6 Impacts

The impacting agents are set out in Table 4.

TABLE 4: Impacting object

Category	Object Striking Head	No Cases
Car interior:		
- fixtures	side door and window	2
	pillars: A	1
	B	3
	roof and roof fixtures	5
	steering wheel	2
	seat and dashboard	1
	windscreen (not ejected)	1
- mobile objects	passenger's head	1
Intruding objects	utility pole	2
	car bonnet	1
Ejection	window and uncertain object	2
	side glass of second vehicle	1
Uncertain		4
TOTAL		26

The head sites believed to have been the points of the chief impact are set out in Table 5. To classify the sites, a clock face was used: frontal impacts are at positions 11, 12 and 1 o'clock, lateral at 2-4 and 8-10 o'clock and occipital at 5-7 o'clock.

TABLE 5: Sites of head impact

Site	No. of Cases
Frontal	15
Lateral	7
Occipital	3
Multiple or uncertain	1
TOTAL	26

We endeavoured to correlate the impact sites with the neuropathological findings, giving special attention to those cases in which impact accelerations were estimated (see below). Of the 15 frontal impacts, four showed no significant brain damage and two showed severe but non-focal anoxic change. The other nine cases showed contusional brain damage: in six this was localized to the frontal or frontal and temporal lobes and in three there was DVI. Of the seven lateral impacts, one showed no significant cerebral injury, one showed localised frontal or temporal lesions and five showed DVI. Of the three occipital impacts, two showed multifocal contusional lesions (with pontomedullary avulsion in one case) and one had frontotemporal lesions. The case (016) in which the impact site/sites were not certain showed very severe DVI.

3.7 Estimates of 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 6). The estimated linear acceleration ranged from 1000 m/s^2 to 5800 m/s^2 in 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 6 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.

TABLE 6: Impact Location, Accelerations and Brain Injury for Fatal and Non-fatal Cases

	NON FATAL				FATAL			
	Acceleration				Acceleration			
Impact Location	Case No	Linear m/s^2	Angular rads/s^2	HLS*	Case No	Linear m/s^2	Angular rads/s^2	HLS*
FRONTAL	008	1000	15000	9 +	017.2	1800	14000	0
	017.1	1800	14000	0	011	2900	22000	0
	019.2	2300	18000	0	022	3900	19000	11 +
	023	4300	30000	6	006	4400	33000	0 x
	009	5800	43000	5				
LATERAL	003	2800	21000	10	012	1900	19000	0 + x
					019	2000	6000	0 +
					007	3000	23000	55
					015.1	4800	24000	101 +
					005.2	6600	50000	56 +
OCCIPITAL					18.1	2900	22000	9 + x
					05.1	3300	16000	20

* Haemorrhagic Lesion Score
\+ Skull fracture
x Fatal injury to other body region

(Case 017.2 suffered minor injuries and is not included elsewhere in the series.)

Frontal impacts

Of the 9 frontal impacts for which accelerations could be estimated, 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. Linear accelerations experienced in these impacts ranged from 1000 m/s^2 to almost 5800 m/s^2. There was no clear association between the presence (Figure 7), or the extent, of cerebral injury and the estimates of linear (Figure 8) or angular (Figure 9) 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, throughout the cerebral hemispheres (Figure 10). There was a tendency for the presence and extent of cerebral injury to be associated with higher levels of acceleration (Figures 11 and 12).

Occipital impacts

Both occipital impacts were fatal; brain injury was found in both. The acceleration was estimated to be just above 3000 m/s^2 in one and just below in the other. Brain injury was remote from the occipital region in both cases (Figure 13). The linear accelerations were estimated to be about 3000 m/s^2 (Figure 14) and the angular accelerations about 20 000 rad/s^2 (Figure 15).

3.8 Site of brain injury and location of impact

It appears from the above observations that brain injury appears in the frontal and/or temporal regions in frontal, lateral and occipital impacts, with sparing of the occipital region. This was particularly evident in frontal impacts, and to a lesser extent in lateral impacts. There was a tendency for the frontal region to be injured, regardless of position of impact, and for frontal impacts there was no clear association between severity of brain injury (HLS) and angular acceleration.

3.9 Acceleration and severity of brain injury in fatal cases

It appeared that the threshold for haemorrhagic brain injury in fatalities was a linear acceleration of about 2000 m/sec^2. A similar calculation for angular acceleration gave a thresh hold of about 10,000 rad/sec^2.

3.10 Preventable causes of bad outcome

The clinical and autopsy data were scrutinized to see whether better outcomes might have been achieved by different management at any stage. Nothing of much significance was noted. It was thought unlikely that the three fatal causes of intracranial bleeding (001, 005.1 and 015.2) would have benefited by operative intervention. One of the patients with anoxic brain damage (019.1) had a severe chest injury, with pulmonary oedema, but it seems unlikely that this was remediable. Case 011, who died on the fourth day after an initial period of partial responsiveness and capacity to talk, developed fatal anoxic brain swelling in association with the treatment of a ruptured spleen: unfortunately the clinical data (from another hospital) did not allow us to say whether avoidable hypotension was a factor in this unusual complication (Figure 6).

4 DISCUSSION

This pilot investigation has applied to the study of head injury in car occupants the multi-disciplinary methods used by this Unit in investigating head injuries in fatal pedestrian cases. It is similar in some respects to the method used by Ommaya and Digges (1985). It has proved possible to study cases surviving head impacts by using CT and MRI scans to provide information on the presence and position of brain injuries. As noted above, we have in this study considered only the more severe (> 5 mm diameter) haemorrhagic lesions found at autopsy, in an attempt to ensure reasonable comparability with the sensitivity of detection by CT and/or MRI scanning. We have carried out further studies (to be published) to calibrate these methods of investigation against each other, in a series of six cases examined before death by CT, by MRI of the cadaveric head, and by the neuropathological methods described above. It is evident that CT and MRI findings are less sensitive than autopsy examination as indicators of lesion location, but MRI is much more sensitive than CT. However CT is under some circumstances better able to identify the nature of a haemorrhagic lesion.

Although the number of cases in this study of car occupants is small, it is clear that frontal impacts to the head form a much higher proportion of all head injuries in car occupants than in pedestrians, with correspondingly fewer 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).

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. 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. 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 under way 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 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, as was one of the frontal cases. This absence of injury is not based solely on the detection of severe lesions. With one exception (case 012, in which there were minor petechial lesions) the brain sections were normal, apart from anoxic damage in the listed case. Therefore at this stage we have no explanation to offer for the lack of detectable brain injury in these cases.

Holbourn (1943, 1945) argued that shear strains induced by rotation of the skull were the cause of brain injury and demonstrated with the use of a gelatin model that strains were greatest in the frontal and temporal regions and the vertex of the brain, following impacts to the occiput. He also pointed out that impacts on the occipital and frontal regions produced identical strain patterns. These observations have been corroborated by Gennarelli et al in primates (1982) and Thibault et al in models (1987). Our observations of injury distribution in this study and in our earlier study of fatal injuries in pedestrians (Ryan et al, 1989) support these conclusions based on theoretical and laboratory investigations. Certainly, our small sample suggests that a proportion of brain injuries are directly associated with skull fractures, when it is likely that the impacting force causes destructive deformation of the brain in the vicinity of the fracture. But numerous brain injuries must be attributed to other mechanisms, of which shear strain due to rotation seems the most likely. Our data suggest a correlation between severity of haemorrhagic brain injury and both linear and angular acceleration from lateral impacts: however the number of cases is still too small for this correlation to be considered as conclusive.

Perhaps the most intriguing finding in this study has been the absence of any correlation between haemorrhagic lesion severity and estimated acceleration in frontal impacts. It is also noteworthy that frontal impacts had a better outcome. We believe this observation deserves further study. So, interestingly, did the London surgeon Percival Pott (1779), who made the same observation:

> "I think that I have seen more patients get well, whose injuries have been in or under the frontal bone, than any other bones of the cranium. If this should be found to be generally true, may not the reason be worth enquiring into? "

It was hoped that the study would contribute information of value in reducing the incidence of preventable causes of bad outcome after head injury. In two cases, the finding of

anoxic brain damage did raise the possibility of avoidable cardiopulmonary or hypotensive complications, but the data did not either substantiate or disprove this suspicion. The study brought out one issue of immediate practical concern: the possibility of providing better head protection for car occupants. We found that in more than half the cases, the head hit a fixture inside the car, and in most of these the site of impact was at or above head level. This finding, if confirmed, is relevant in the current debate on the value of airbags contained in the steering wheel: it is likely that these would not have affected the outcome in some of our cases of lateral impact. Other means of reducing the impact force should be considered, such as helmets, energy absorbing pillars and roofrails, or airbags in different sites.

5 CONCLUSIONS

1. In a pilot study of 26 car occupants who suffered fatal or severe head injuries, it was possible to estimate the impact-induced head acceleration in 17 cases from data obtained by accident investigations. This relatively high percentage (65.4%) justifies extending the study to obtain a larger case sample.

2. In the pilot study, frontal head impacts were more numerous, but less injurious than lateral and occipital impacts.

3. The neuropathological and radiological (CT and MRI) findings in these cases have been quantitated with respect to extent and severity, and standard diagrams have been devised to allow these data to be coded in a computer file.

4. Preliminary comparisons suggest that CT and MRI examinations underestimate the severity of haemorrhagic brain lesions; a further study has been carried out to confirm this impression.

5. There appeared to be a positive correlation between injury severity (measured by the number of brain areas showing haemorrhagic lesions) and head impact acceleration for lateral and occipital impacts, but this was not evident with frontal impacts, which appear to be less injurious than comparable lateral and occipital impacts. Possible reasons for this difference in vulnerability are being explored.

6. The study gave an unselected sample of car crashes causing death and/or serious head injury in metropolitan Adelaide and in the surrounding (100 km radius) rural areas. The epidemiological findings were generally in accordance with expectations, as were the findings from crash examination; alcohol consumption and failure to use seatbelts were identified as important factors in crash/injury precipitation respectively.

7. The structures causing head impacts were identified in 22/26 (84.6%) cases; side pillars, side doors and windows, and roof fixtures were incrimated in 11 (42.3%) cases. If this incidence is confirmed in the forthcoming larger series it will justify improving the head

protection of car occupants. This could be done by wearing helmets and/or by making the car interior structures more effective in absorbing energy.

8. The injuries were considered retrospectively from the viewpoint of preventable errors in management, both of the accident site and in the hospital. Two cases showed signs of anoxic brain damage, and in one of these the history of a period of earlier clinical responsiveness did raise the possibility of a remediable complication, but the available case records were incomplete and no conclusion was possible.

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The chief findings made in this study were reported to the International Research Council on Biokinetics of Impacts in Potsdam Germany, on 11-13th September 1991, and have been published in the proceedings of that meeting.

APPENDIX I: DIAGRAMMATIC RECORDING OF BRAIN LESIONS

Since the inception of our neuropathological studies on head injury mechanisms in road crashes, we have routinely recorded findings on conventional brain diagrams representing coronal slices of the cerebral hemispheres (10-11 diagrams), transverse slices of the brainstem (usually 6 diagrams), and parasagittal slices of the cerebellum (usually 2 diagrams). A uniform colour-coded notation has been employed:

Haemorrhages and haemorrhagic lesions: RED

Cortical: dots - contusion not extending the full thickness of the cortex but with ≥ 3 collections of red blood cells per high power field.

: oblique hatching = petechial haemorrhages in full cortical thickness and/or confluent haematoma.

: solid colour = loss of tissue and/or laceration.

White matter and basal ganglia:

: dots = lesions < 5mm across and/or visible only microscopically.

: oblique hatching = lesions > 5mm across and/or visible in gross inspection.

: solid colour = loss of tissue and/or laceration.

Subarachnoid blood is recorded by dashes, and subdural blood by oblique hatching, over the surface of the cortex.

Axonal lesions: BLACK

All areas: dots = scattered DAI.

: hatching = extensive DAI.

Ischaemic and hypoxic lesions: GREEN

: hatching = extensive lesions.

Non-specific gliosis: BLUE

In 1988 it was decided to record haemorrhagic lesions on diagrams representing the cerebral cortex as if unfolded into a two-dimensional format and divided by transverse and sagittal/parasagittal lines into sectors. Each coronal line corresponded with a coronal slice, and it was thus possible to record the presence (and if necessary the severity) of a lesion recorded

in coronal diagrams as involvement of a specific sector. The number of affected sectors, and their cerebral locations, could then be recorded in a computer file. Since this method of recording was introduced, it has been progressively modified and elaborated. Four versions (now termed Marks I-IV) have been used, and these are in some important respects different; they are therefore described and illustrated, with indications of their use in publications by NHMRC Road Accident Research Unit staff.

Mark I diagram (Figure 16). The cerebral hemispheres are reconstructed from 10 coronal slices, each of which is represented by 4 cortical radial segments; a single basal segment represents the deep white matter and diencephalon. There are thus 10 x (8 + 1) = 90 sectors. The Mark I diagram was not used in any publication.

Mark II diagram (Figure 17). In this, 8 sectors are added to represent the temporal lobes: each temporal lobe slice is divided into superomedial and inferolateral sectors. There are thus 98 sectors. This diagram was used by Ryan et al. (1989).

Mark III diagram (Figure 18). This embodies 11 coronal slices, as in practice this has usually been the number examined. Each slice is divided by a radial line into 8 segments, but the temporal lobes are now included with the related parts of the frontal lobes. The deep structures are now separately represented as the corpus callosum (supposed to be visible in 7 coronal slices) and the right and left basal ganglionic regions (supposed to be visible in 5 coronal slices). There are thus (11 x 8) + 7 + (5 x 2) = 105 sectors. This diagram was used by Manavis et al. (1991) and by Simpson et al. (1991).

Mark IV diagram (Figure 19). This adds to the above representation 2 sectors for each temporal lobe, 1 sector for each half of the cerebellum, and 6 segments for the brain stem. There are thus 105 + (2 x 2) = 109 supratentorial sectors and 8 infratentorial sectors. The Mark IV diagram is at present being used in a comprehensive study of pedestrians and car occupants; it appears in Figure 3 in the present report.

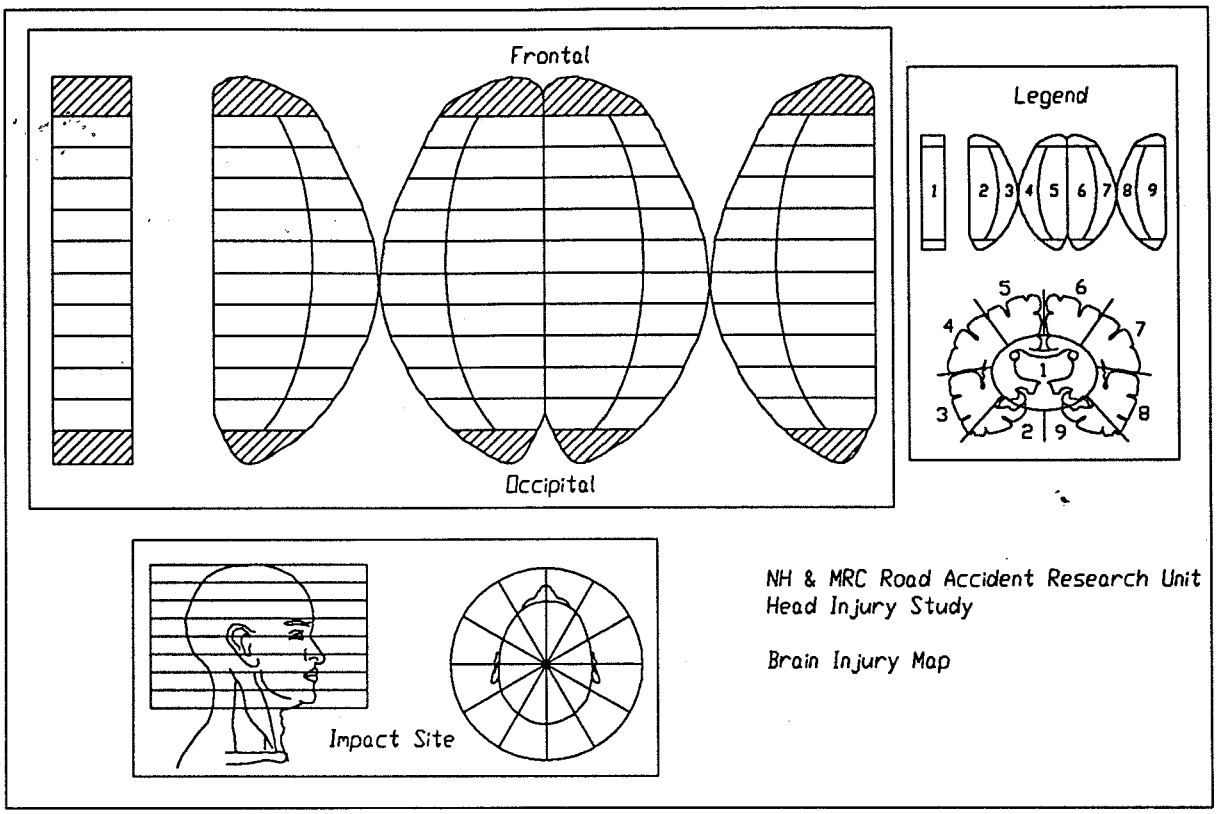

FIGURE 16: Mark I: as used in preliminary studies on pedestrian injuries: 90 supratentorial sectors

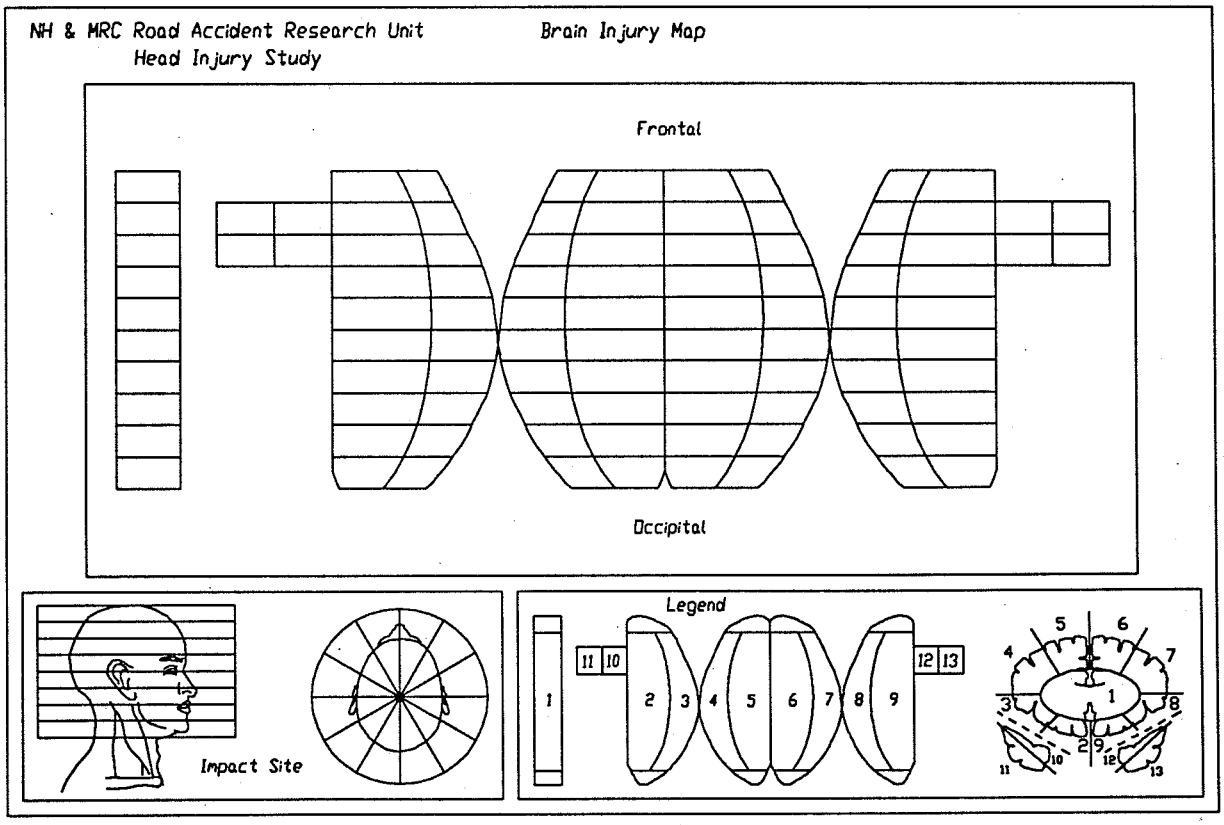

FIGURE 17: Mark II: as used by Ryan et al (1989): 98 sectors in supratentorial region

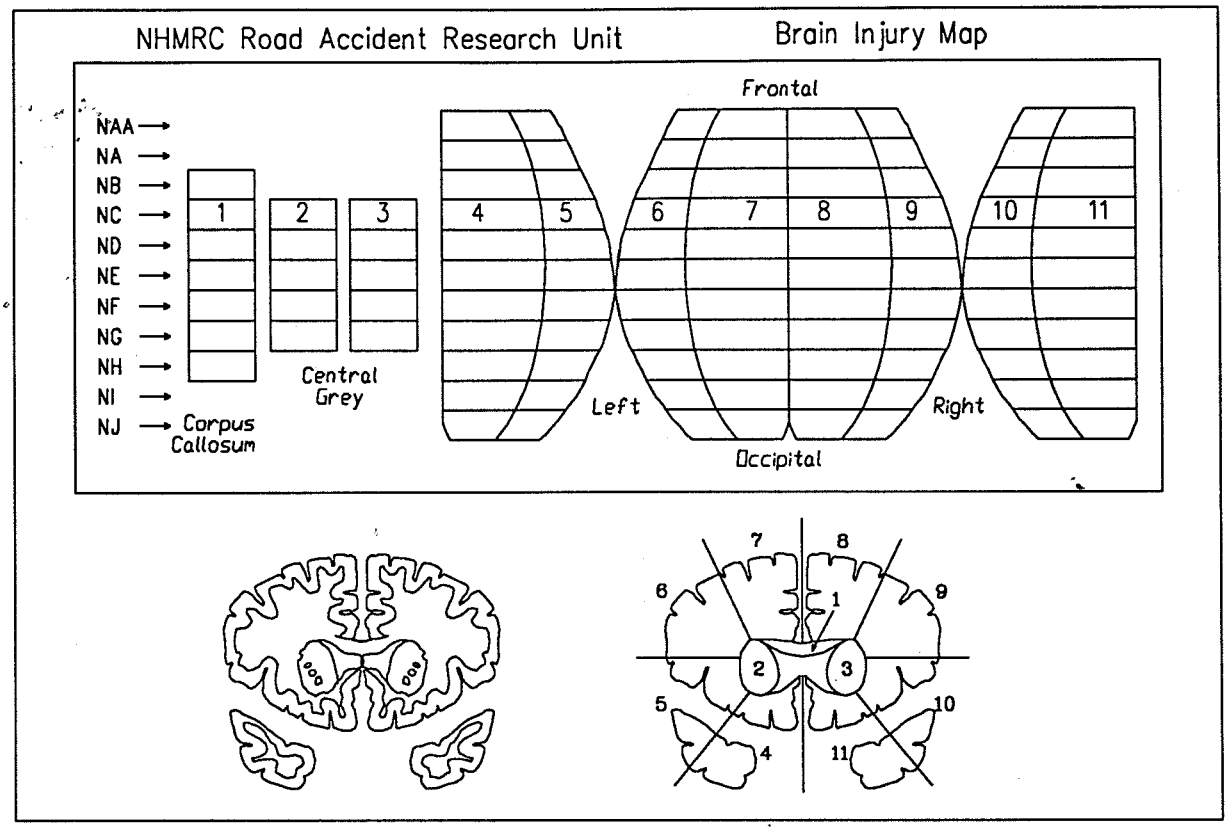

FIGURE 18: Mark III: as used by Simpson et al (1991) in study of car occupant injuries: 105 sectors in supratentorial region

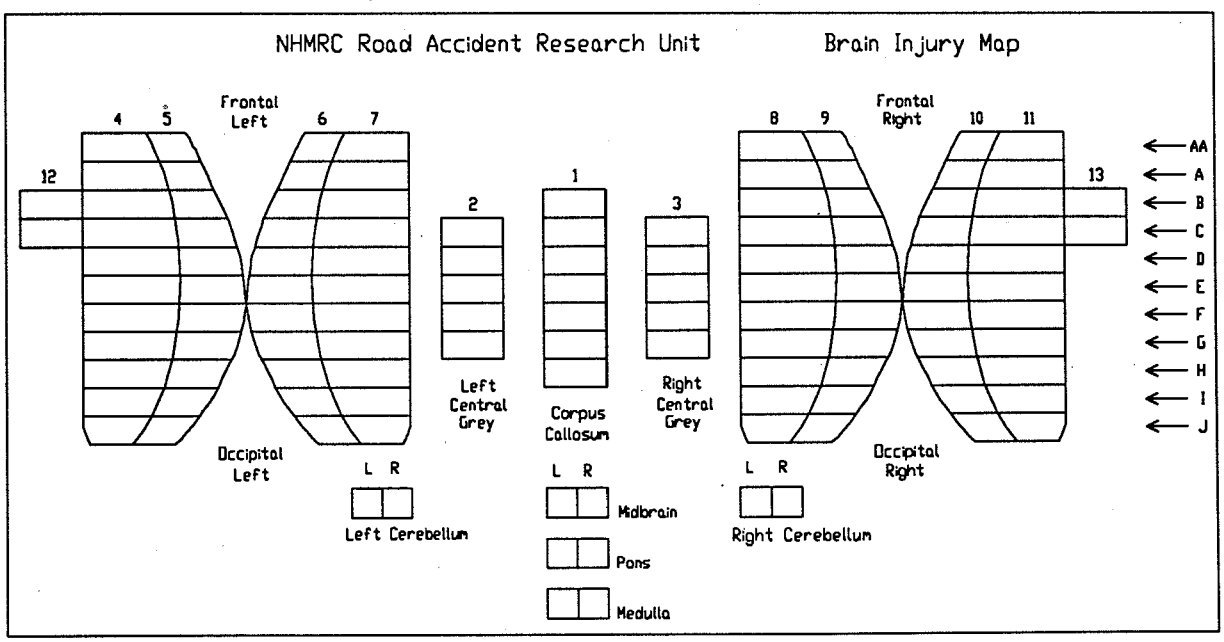

FIGURE 19: Mark IV: comprehensive brain injury map: 109 sectors in supratentorial region and 10 sectors in infratentorial region

APPENDIX II: TABULATION OF CRASH DATA

Case No.	Occupant Status	Body Style Vehicle	Event Causing Crash	Object Causing Injury	Estimated Car Impact Speed
001	Driver	Sedan	Collision tree frontal	(1) passengers head? (2) mirror and header	(?) > 70 km/hr
002	(Omitted)				
003	Passenger Right rear (struck side)	Sedan	Collision car right side	Intruding door	V1 - 60 km/hr V2 - 40 km/hr *(Head impact speed 30 km/hr)*
004	Driver	Sedan	Collision truck frontal	Intruding A-pillar	Not known
005.1	Driver (far side)	Sedan	Collision pole left side	? Intruding pole	70 km/hr
005.2	Passenger left front (struck side)	Same vehicle	Collision pole left side	? Steering wheel	70 km/hr
006	Passenger left front (far side)	Sedan	Collision pole right	? Window (ejected)	50-60 km/hr
007	Passenger left front (struck side)	Sedan	Collision car left side	Intruding car	70 km/hr (calculated) *(Head impact speed 65 km/hr)*

Case No.	Occupant Status	Body Style Vehicle	Event Causing Crash	Object Causing Injury	Estimated Car Impact Speed
008	Passenger right rear (struck side)	Sedan	Collision car right	? Pavement ? road (ejected)	20 km/hr
009	Driver (far side)	Sedan	Collision car left	Intruding B-pillar	60 km/hr
010	Driver	Sedan	Rollover	(1) Window (2) Ground (ejected)	90 km/hr
011	Driver	Sedan	Collision tree	Steering assembly	60 km/hr
012	Passenger left front (struck side)	Sedan	Collision car left side	Intruding B-pillar	(Calculated)
013.1	Driver	Sedan	Collision car frontal	Intruding windscreen	V1 - 140 km/hr
013.2	Driver	Sedan	Collision car frontal	Steering wheel	V2 - 90 km/hr
014	Passenger ? site	Sedan	Collision truck left side	? A-pillar	Not known
015.1	Driver	Station wagon	Collision object (old cart) right side	Header rail	115 km/hr *(Head impact speed 50 km/hr)*
015.2	Passenger left front (far side)	Same vehicle	Collision object (old cart) right side	Uncertain (mobile in cabin)	115 km/hr

Case No.	Occupant Status	Body Style Vehicle	Event Causing Crash	Object Causing Injury	Estimated Car Impact Speed
016	Driver	Utility (tray top)	Collision car frontal	Mobile object in vehicle	Not known
017.1	Passenger left front	Sedan	Collision bank and culvert frontal	Roof and sunvisor	70 km/hr? *(Head impact speed 25 km/hr)*
017.2	Driver	Same vehicle	Collision bank and culvert frontal	Uncertain	70 km/hr *(Head impact speed km/hr)*
018	Driver (struck side)	Sedan	Collision car right side	Roof and radio speaker	V1 - 70 km/hr V2 - 70 km/hr *(Head impact speed 30 km/hr)*
019.1	Passenger right rear	Sedan	Collision pole frontal	Drivers seat	70 km/hr *(Head impact speed 23 km/hr)*
019.2	Passenger left front	Same vehicle	Collision pole frontal		70 km/hr *(Head impact speed 70 km/hr)*
020	Passenger left front (struck side)	Sedan	Collision car left side	? Window (ejected)	Not known
021	Driver	Sedan	Collision tree frontal		20 km/hr
022	Passenger left front	Station wagon	Collision pole frontal	? Intruding pole	60 km/hr *(Head impact speed 40 km/hr)*
023	Driver (far side)	Hatchback	Collision tree left side	Intruding roof rail	43-50 km/hr

APPENDIX III: TABULATION OF PATHOLOGICAL FINDINGS

(a) Fatal Cases

Case	Impact Site	Cerebral Lesions	Subdural Haemorrhage	Skull Fracture	Other Injuries AIS ≥ 5	Outcome (GOS)
001	R parieto-occipital	DVI; PM tear	+ L side	Base	AOD	Dead at site
005.1	Occipital	DVI maximal L side	+ L side	-		Died in hospital day 1
005.2	R frontal	L frontal and L temporal lacerations; peduncle lacerated; DVI	-	Base and vault	-	Dead at site
006	R frontal	-	-	-	Heart	Died in hospital day 1
007	L lateral	DVI	-	-	-	Died in hospital day 1
010	Frontal	DVI	-	Base	-	Died at site
011	R lateral and frontal	Anoxia; cerebellar swelling	-	-	-	Died in hospital day 4
012	L lateral	(Minor lesions)	-	Base	Multiple chest	Died at site

Case	Impact Site	Cerebral Lesions	Subdural Haemorrhage	Skull Fracture	Other Injuries AIS ≥ 5	Outcome (GOS)
013.1	Frontal	DVI	-	-	Heart	Died at site
013.2	Frontal	DVI maximal L side; ICH	-	Facial	-	Died in hospital day 1
014	L lateral	Lacerations and contusions; DVI	-	Cpd; L parietal depressed + base	-	Dead on arrival
015.1	R lateral	Lacerations, DVI	-	R temporal + base	-	Died at site
016	L lateral	Severe DVI	-	-	Liver	Dead on arrival
017	Frontal	-	-	-	Heart	Died at site
018	(Frontal) R occipital	Bitemporal contusions; DVI	-	Base	-	Dead on arrival
019.1	R lateral	Anoxia	-	R temporal + base	-	Died in hospital day 1
020	Frontal	DVI	-	Base (small)	Aorta	Dead on arrival
021	? L frontal	(Minor lesions)	-	-	Heart	Died at site
022	L frontal	DVI	-	Base + L parietal	(Limb amputations)	Died in hospital day 1

(b) Survivors

Case	Impact Site	Imaging	Cerebral Lesions	Subdural Haemorrhage	Skull Fracture	Outcome (GOS)
003	R lateral	CT	Multiple haemorrhages	-	-	Motor disabilities (Moderate)
004	R frontal	CT	Ipsilateral frontal & temporal haemorrhage	-	Base	Behavioural disabilities (Good recovery)
008	R frontal	CT	Ipsilateral frontal	-	Depressed R frontal	Intellectual disabilities (Outcome otherwise uncertain)
009	L fronto-lateral	CT/MRI	Contralateral haemorrhage; bilateral white matter damage	Ipsilateral (large)	-	Motor & intellectual disabilities (Moderate)
015.2	R frontal	CT	Ipsilateral temporal haemorrhage (small)	-	Comminuted fronto-basal	Outcome uncertain (? Good recovery)
019.2	R fronto-	CT	-	Dubious inter-hemispheric haemorrhage	-	(Good recovery)
023	R frontal	CT/MRI	Bilateral white matter damage	-	-	Motor & intellectual disabilities (Moderate)

Abbreviations for Appendix III

DVI = diffuse vascular injury + macroscopic contusions; PM = pontomedullary tear; ICH = intracerebral haemorrhage;
AOD = atlanto-occipital dislocation; Cpd = compound