MECHANISMS OF BRAIN INJURY RELATED TO MATHEMATICAL MODELLING AND EPIDEMIOLOGICAL DATA

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ABSTRACT

Measurements of the impulse frequency response of head impact points on the exterior and the interior of a car were used to calculate the modal mass and stiffness for each point. These points were then arranged in an hierarchy of increasing stiffness and grouped into three classes. Thirty two head impact cases (13 pedestrian, 4 car occupants and 15 fall victims) in which the distribution of injury to the brain had been recorded in detail were grouped according to the stiffness of the object struck and by the location of the impact on the head. The distribution of the brain injury lesions in the anterior, middle and posterior regions of the brain were determined for each class of stiffness (soft, medium or hard) and location of impact (occipital or lateral). Distinctive patterns of brain injury distribution were noted for each class of stiffness and each location of impact. Three probable mechanisms of brain injury were distinguished. They were: relative motion between the brain and the skull, local bone deformation and intra-cerebral strains. Each mechanism was related to a range of stiffness and natural frequency of the structure impacted. Hence these theories of brain injury mechanisms are consistent with observed epidemiological data and with conclusions drawn from mathematical modelling.

INTRODUCTION

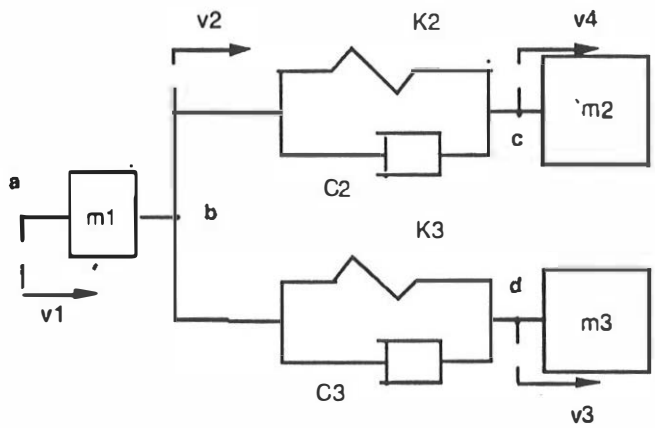
This paper is the result of collaboration between the Laboratoire des Chocs et de Biomecanique, Institut National de Recherche sur les Transports et leur Securite (INRETS), Bron, the Laboratoire des Systemes Biomecaniques of the Institut de Mecanique des Fluides (IMF) at the Universite Louis Pasteur, Strasbourg, and the National Health and Medical Research Council Road Accident Research Unit (RARU), University of Adelaide, Adelaide, South Australia. The information on brain injury distributions gathered through a combination of crash investigation and neuropathological studies by RARU is compared with the model of mechanisms of injury to the brain recently proposed by INRETS and IMF (1,2).

First, the model of the head is presented, together with the possible mechanisms of injury derived from this model. We then present the method used to estimate the stiffness of the structures struck by the head, as identified in the crash investigations. Following this, the brain injury data are presented, arranged by

stiffness classification. Finally, the relationship between the injury distributions and the impact stiffness is then examined in the light of the proposed theoretical models.

MODEL AND PROPOSED MECHANISMS

In a previous study, we recorded the mechanical impedance of physical models (box + brain + water) and of heads in vitro and in vivo (1,2). These experiments were performed by means of a hammer and an accelerometer and clearly demonstrated the existence of a natural frequency at 120 Hz due to the brain mass. The modal parameters related to this frequency prove that the brain is brought to resonance relative to the rest of the head. This work allowed us to show that a relative brain-skull movement appears at frequencies beyond 100 to 200 Hz and led us to propose a new mass - spring model distinguishing the brain mass from the other head elements (Fig. 1).



head	m1 kg	m2 kg	m3 kg	K2 N/m	K3 N/m	C2 Nm/s	C3 Nm/s
vivo	0.4	1.6	2.2	0.7E6	10E6	200	1500
vitro	0.8	1.7	3.6	0.7E6	25E6	600	70

Figure 1. The lumped head model with its modal parameters

The governing equation of the model allowed us to identify the relative brain-skull speed by means of Fourier's transform. The damped model impedance is :

$$Z = Z_1 + \frac{Z_2 Z_4}{Z_2 + Z_4} + \frac{Z_3 Z_5}{Z_3 + Z_5} \qquad (1)$$

Where:

mt : overall head mass

m1 : frontal bone mass

m2 : brain mass

m3 : mt - m1 - m2

K2(3) ; C2(3) : m2 (and m3) rigidity and damping

$Z1 = j\,\omega\,m1$

$Z2 = C2 + K2 / j\,\omega$

$Z3 = C3 + K3 / j\,\omega$

$Z4 = j\,\omega\,m2$

$Z5 = j\omega\,m3$

Kirchhoff's laws applied to the points a, b, c, and d, express the balance at each node and permit us to calculate the relative displacement, velocity and acceleration of the brain and skull as a function of head impedance and the excitation force (2).

This vibration approach to the behaviour of the head under impact conditions led us to suspect that the mechanism of injury to the brain may be related to the duration of the impact or, put another way, to the maximum value of the frequency spectrum of the impact. We proposed two mechanisms:-

In the first, when the maximum frequency contained in the impact spectrum is less than about 150 Hz, the brain moves with the skull, thus producing shear strains in the deeper parts of the brain. This mechanism results in the diffuse brain injuries observed in long duration impacts - for example an impact to a subject wearing a helmet.

The other mechanism is related to the relative motion between the skull and the brain which occurs when the impact energy is concentrated in the 100-800 Hz frequency range. This mechanism leads to subdural haematomas and to focal contusions located in the cortex and the periphery of the brain. It is typically observed in short duration impacts - for example an impact of a subject without a helmet on concrete.

In this joint study we compare the proposed mechanisms with observed epidemiological data.

METHOD

Determination of dynamic stiffness

The stiffness of a structure is usually determined by measuring the amount of deformation as a function of the force applied by an impactor. This method does not provide sufficient information to fully understand the dynamic behaviour of an isolated structure of high rigidity. An alternative method is to derive the stiffness through the use of a mathematical model of the dynamic behaviour of the structure following an impact. In order to construct a mathematical model of the impacted structure we can determine the apparent mass at the impact point from the frequency response to an impulse.

The test procedure consisted of striking the point of interest with a hammer which was instrumented to record the impact force. An accelerometer was placed close to the impact point to record the response of the structure. The two signals were then Fourier transformed and the transfer function (ie the apparent mass) was then determined from the ratio of the force Fourier transform to the acceleration Fourier transform. The apparent mass was then plotted in dB as a function of the frequency logarithm.

As a first approximation, the impact structure can be expressed as a lumped model with one mass, one spring and one damper in series. This is a good approximation, given that the amplitude curve of the apparent mass is a horizontal line (describing mass behaviour), followed by a first natural frequency and a straight line with a negative slope (describing spring behaviour) (Figure 2). In some

cases a second or third natural frequency exists leading to more complicated models. This aspect will be considered in the future.

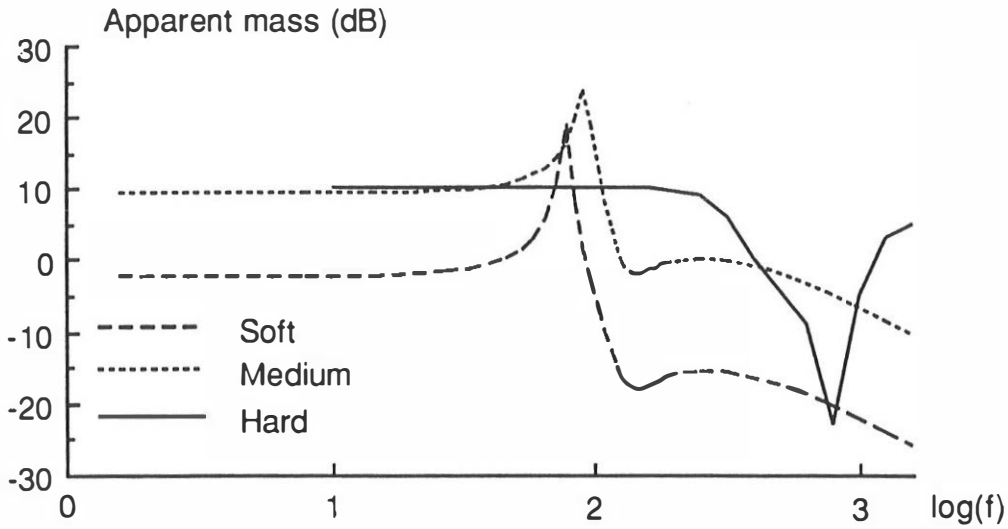


Figure 2. Apparent mass and impact stiffness

The main difference between the impact points lies in their natural frequencies rather than in their modal masses. The apparent mass curves are not all defined in the same frequency range. At relatively soft points for example, the Fourier transform of the force signal contains little high frequency energy, leading to a transfer function defined only below a given frequency (eg 800 Hz). At the opposite extreme, very rigid points have a poor response (at the acceleration Fourier transform level) at low frequency, so the apparent mass is only defined in a given frequency range (eg 100-1500 Hz).

Table 1 presents the characteristics of selected impact points on the exterior and interior of an Australian 1976 model XC Ford Fairmont sedan. These points represent the common head impact points for fatally injured pedestrians and car occupants as observed in a large series of cases studied by RARU (3,4,5). Points 1 to 11 are exterior pedestrian head impacts and points 21 to 27 are interior car occupant head impact points. Points 28 and 29 refer to head impacts on concrete and similar surfaces as a result of falls. For each impact point this table gives the first natural frequency, the modal mass at low frequency and the modal stiffness. The impact points were classified into three groups based on the following criteria: an impact point with a low stiffness (less than 100 x 10^4 N/m) was classed as "soft". A "medium" point was one with a stiffness in the range 100 x 10^4 to 200 x 10^4 N/m, while "hard" points had a stiffness greater than 200 x 10^4 N/m. Figure 3 illustrates this classification. It can be seen that the first natural frequencies of the medium stiffness group partially overlap those of the soft and hard groups.

Table 1. Impact point characteristics

Point N°	Structure	f1 (Hz)	m (kg)	K 10^4 (N/m)	Classification
1	wing	77	0,20	4,7	soft
3	bonnet (1/3)	47	0,87	7,6	soft
6	bonnet-grille	126	0,75	47	soft
8	roof	100	0,55	21	soft
10	bonnet (middle)	28	0,33	1	soft
26	middle st. wheel	134	0,20	24	soft
27	st. wheel (rim)	67	0,20	4	soft
5	bonnet (side)	200	1,13	178	medium
21	roof rail no lining	126	2,90	182	medium
22	roof rail lining	121	3,00	173	medium
25	roof / windscr.	126	0,77	164	medium
4	wing (side)	245	1,90	450	hard
7	top A-pillar	794	3,70	9200	hard
9	windsceen	473	0,35	309	hard
11	A-pillar (ext.)	500	0,37	365	hard
23	B-pillar (int.)	645	1,02	1675	hard
24	A-pillar (int.)	407	3,90	2550	hard
28	concrete	2000	78	> 10000	hard
29	other hard	-	-	> 10000	hard

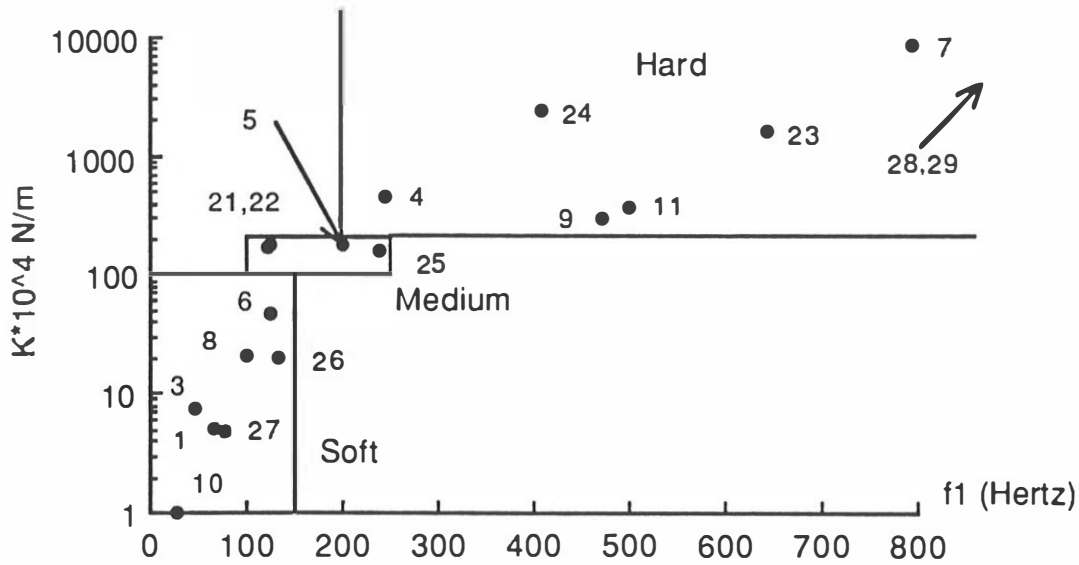


Figure 3. Classification of impact stiffness and first natural frequency

Brain injury data

For each case, the injuries to the brain were recorded on a diagram following neuropathological examination. The brain was divided into 11 coronal sections labelled AA, A, B, C.....J at 10mm intervals from front to rear. Each section was divided into sectors numbered from 1 to 11 and the presence of injury, as indicated

by haemorrhage or laceration within each sector, was recorded. It was thus possible to record the distribution of the injury within the brain in three dimensions.

As can be seen from the diagram (Figure 4) sectors 1, 2 and 3 refer to the central part of the brain and include the corpus callosum (sector 1) and the left (sector 2) and right (sector 3) central nuclei. Sectors 4, 5 and 10, 11 are the inferior portions of the left and right hemispheres respectively, and sectors 6, 7 and 8, 9 refer to the superior parts of the left and right hemispheres respectively. For the purposes of this study the brain was divided into three regions: the anterior region (A) consisted of sections AA, A and B; the middle region (M) consisted of sections C,D,E,F and G; and the posterior region (P) comprised sections H,I and J. Sector 1, representing the corpus callosum, extends from section B to section H, while sectors 2 and 3 extend from sections C to G. These central three sectors are therefore almost entirely confined to the middle region.

In order to calculate a measure of lesion frequency for a given regional sector (for example M-7) the total number of lesions recorded in this sector for a specific impact condition were divided by the number of sections in the given region and the number of cases involved in the impact under consideration, ie the frequency of lesions was standardised to take into account the differing number of sectors and cases in each group of impact conditions.

The velocity or the acceleration developed in each impact have not been considered in this study, because, according to the model under consideration the velocity of impact will determine the amplitude but not the frequency spectrum of the response, and we believe that it is the latter which largely determines the distribution of injury in the brain.

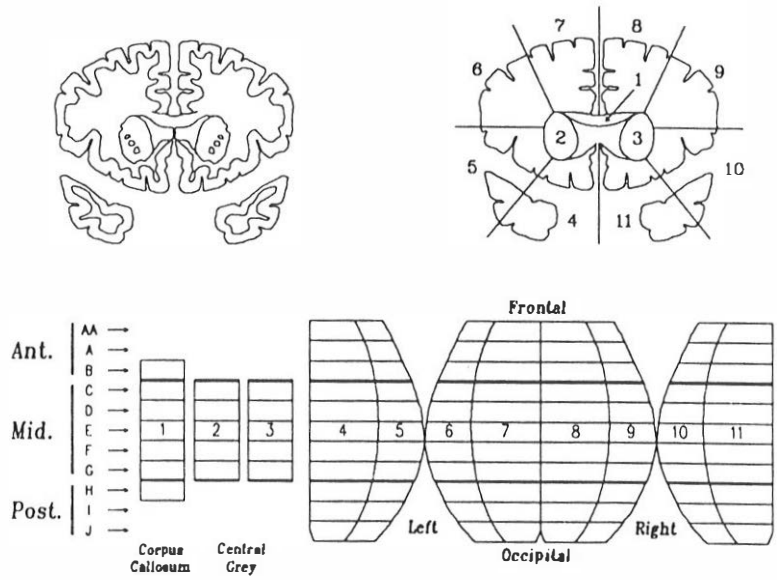


Figure 4. Diagrams used for recording injury to each section of the brain

RESULTS

Brain Injury Cases

The relationship between the distribution of brain injury and the location and severity of the impact to the head in cases of pedestrian and fall fatalities, and fatal and severe head injuries in car occupants has been presented previously (3,4,5). In this study we classified these cases as a function of the stiffness of the structure impacted, regardless of whether the case was a pedestrian, car occupant or suffered a fall. The characteristics of each are set out in Table 2. We excluded from the analysis 11 cases in which injury was present in all brain regions, because it was felt that they did not contribute to the detailed study of specific lesions as a function of impact characteristics. We have only considered cases with impacts on the occipital and lateral regions.

The 32 cases (13 pedestrian, 4 car occupants and 15 falls) were classified by stiffness of impact and position of impact on the head. The injuries for the lateral impacts were all coded as if the impacts were on the left. The distribution of the 32 cases was as follows:

Stiffness of impacted surface	Location of impact on head	
	Occipital	Lateral
Hard	10	9
Medium	3	3
Soft	3	4

The association between the stiffness of the impacted structure and the distribution of injury will be considered for occipital and lateral impacts separately. The histograms presenting the standardised frequency of lesions in the anterior, middle and posterior regions for hard, medium and soft impact stiffnesses and for occipital and lateral impacts to the head are shown in Figures 5 to10.

Occipital impacts

For hard impacts (Figure 5, N=10), injuries primarily occurred in all sectors of the anterior region. There were few injuries in the middle region and none in the posterior region. Acute subdural haemorrhage (ASDH) occurred in six cases.

In medium impacts (Figure 6, N=3), injuries occurred in the middle and anterior regions, in the inferior and para-sagittal sectors. Injuries were observed in the central sectors of the middle region. ASDH was observed in one case.

For soft impacts (Figure 7, N=3), injuries were found in all three regions, primarily in the inferior and the central sectors, but no ASDH was seen in these cases. Inspection of the histograms shows that for occipital impacts, as the impacted structure decreased in stiffness, the region of the brain showing injury spread posteriorly and the central sectors of the brain were more affected.

Lateral impacts

In hard lateral impacts (Figure 8, N=9), injury occurred in all three regions of the brain, primarily in the inferior sectors and the corpus callosum (sector 1). Acute subdural haemorrhage was observed in 7 of the 9 cases.

In medium impacts (Figure 9, N=3), injuries were recorded in the anterior and middle, but only a few in the posterior, regions. The para-sagittal sectors, together with the central brain, were the most affected. ASDH was observed in one case.

In soft impacts (Figure 10, N=4), the anterior and middle regions were the most predominantly affected, particularly the central, inferior and para-sagittal sectors, with ASDH occurring in three cases.

In the hard lateral impacts all three regions of the brain were injured, however there were very few injuries to the posterior region in the softer impacts, that is, the anterior and middle regions were injured in all impacts. The central sectors of the brain were injured with all degrees of stiffness. ASDH occurred in both hard and soft impacts.

DISCUSSION

Occipital impacts

The distribution of injuries resulting from impacts on hard structures suggests that the mechanism of injury was not local bone deformation because there were no lesions close to the position of the impact, that is, in the posterior parts of the brain. Also, the mechanism was not due to intra-cerebral shear strains because there were very few lesions in the central parts of the brain. The mechanism of brain injury may be related to relative movement between brain and skull. This is more likely to occur in impacts with hard structures, where there is a large high frequency component. The natural frequency of the brain is much lower than that of the skull; as a result the brain does not respond in the same way to impact. The brain becomes de-coupled from the skull and moves independently. It is therefore more likely to be injured by direct contact with the skull particularly in the anterior region which has a much more complicated topography than the posterior (6). This would explain why lesions were observed much more frequently in the anterior region of the brain rather than in the posterior. The high rate of ASDH observed in hard impacts would be in accord with this theory of relative movement between brain and skull (7,8).

For impacts on structures with medium stiffness the lesion mechanism, once again, was unlikely to be related to local bone deformation or displacement because there were no lesions near the location of the impact to the head. In these impacts the impact energy is distributed between high and low frequencies. Therefore some stresses can be transmitted to the brain, causing central lesions, before it becomes de-coupled from the skull. It is probable that there may be two mechanisms at work in medium hardness impacts. In one, the brain moves with the skull causing intra-cerebral strains and thus, central lesions and in the other, relative movement between the brain and the skull causes the peripheral lesions.

For impacts with soft structures the main mechanism operating appears to be intra-cerebral stress as indicated by the lesions observed in the central part of the brain. The lesions observed in the posterior region suggest the operation of local occipital bone deformation or displacement at or near the point of impact. The brain-skull relative motion mechanism may be less important in these cases.

Lateral impacts

Lateral impacts on hard structures also involve high frequencies and therefore a similar mechanism of relative movement between the brain and skull as with occipital impacts, but in this instance the movement is in the coronal plane. This relative movement may cause the lesions in the inferior sectors of the cerebral hemispheres in the middle and anterior cranial fossae, as well as the posterior parts of the brain. This is well illustrated by the symmetrical nature of the histograms relating to the middle region. The cases of ASDH may be caused by the same mechanism. The brain injury in these cases is probably not related to local parietal bone displacement, which would cause asymmetric lesions in the middle region.

In lateral impacts with structures of medium stiffness there were relatively more lesions in the central areas of the brain, compared with hard impacts, suggesting a mechanism involving cerebral stresses during the impact. The peripheral lesions, particularly in the para-sagittal and inferior sectors could be due to the relative movement between the brain, the skull and the falx cerebri, in the coronal plane. That is, as in similar occipital impacts, there may be two mechanisms of brain injury operating.

For lateral impacts with soft objects the distribution of lesions is much more asymmetric than for the other lateral impacts. This suggests a mechanism involving parietal bone deformation or displacement at least in the middle region. The frequency of central injuries suggests that the role of intra-cerebral shear strains is an important mechanism in these impacts, particularly as it was observed that the frequency of injury observed in the central sectors of the brain increased with increasing "softness" of the impacted structure.

The relatively high incidence of ASDH in soft lateral impacts, compared with soft occipital impacts, suggests that there was another mechanism, perhaps a more local effect, in operation. With regard to the 11 cases which were excluded from this study because of the extensive brain injury suffered, 10 impacted a hard structure and one impacted a structure of medium stiffness. All were high energy impacts. Theoretically, impacts on hard structures contain energy at low frequencies as well as high frequencies. On the other hand, soft impacts contain energy only in the low frequency domain. Therefore, high energy hard impacts can simultaneously produce the effects of a hard and a soft impact, that is, injury in all regions, as seen in these 11 cases.

CONCLUSIONS

During impacts with hard structures, where the impact energy is contained in the high frequency range, the brain tends not to move, leading to relative motion between the brain and skull with the consequent potential for injury. This mechanism may be responsible for most peripheral lesions, especially those located in the inferior sectors of the brain adjacent to the anterior and middle cranial fossae that is, the regions of the skull where there is great anatomical complexity. About two thirds of these cases were associated with ASDH, most likely due to the rupture of veins bridging the gap between the brain and the skull. This mechanism would also tend to explain the difference in distribution of injury between occipital and lateral impacts. For lateral impacts, in general, the sectors with maximal frequency of injury were found inferiorly and temporally while for occipital impacts the sectors most often injured were in the anterior region.

For impacts with structures of medium stiffness the lesions observed suggest that, as in hard impacts, relative motion between the brain and the skull is an important mechanism. The presence of more central lesions in comparison with hard impacts suggests that intra-cerebral shear strains play a more important part in medium impacts.

For impacts with structures of relatively soft stiffness the brain tends to move with the skull because of the predominantly low frequencies involved in these impacts. These movements result in intra-cerebral strains which can produce lesions in the central parts of the brain. The asymmetric nature of the more peripheral lesions is probably due to bone deformation or displacement at the time of the impact. Figures 7 and 10 show only relatively minor differences in injury distribution between occipital and lateral impacts, suggesting that intra-cranial geometry is unlikely to play a role in the mechanism involved. This leads to the conclusion that the peripheral lesions in these soft impacts were not due to relative brain-skull motions.

From this study we can conclude that the distributions of brain injury found in the epidemiological studies can largely be explained by the previously proposed mechanisms of brain injury:

i) Relative motion between the brain and the skull. This occurs in impacts of hard or medium stiffness (natural frequency >250 Hz). These are often accompanied by ASDH, and lesions in the frontal, lateral and inferior regions of the brain.

ii) Intra-cerebral shear strains. These occur when the natural frequency of the impact is less than about 250 Hz, with the result that the outer parts of the brain move with the skull causing shear strains in the cerebral tissue as the inner parts of the brain lag behind the outer.

iii) Local bone deformation or global displacement. In soft or medium impacts the local deformation or displacement of the bone at the site of impact produces injuries around the area of the impact.

The relative importance of these three mechanisms of brain injury in any one impact will depend on the local conditions at the time of impact, particularly the stiffness characteristics of the impacted structure. The theoretical model suggests that the natural frequencies of the head and the structure are the characteristics with most importance in determining the distribution of injury in the brain for a particular location of impact on the head.

Acknowledgements : We wish to thank the French Ministry of Research and Technology and the National Health and Medical Research Council of Australia for their support for this project. We also wish to acknowledge the contribution to this research of Dr PC Blumbergs and Dr G Scott, of the Institute of Medical and Veterinary Science in Adelaide, who performed the neuropathological examinations.

Table 2. Summary of Impact Data

Case (1)	Impacted structure	Impact point N° and classification	Head impact (2)	Age	ASDH (3,4)	fracture (4)
P47-86	bonnet-grille	06 soft	O	68	0	1
P03-88	bonnet (1/3)	03 soft	O	17	0	1
P12-87	bonnet (1/3)	03 soft	O	20	0	0
P24-87	bonnet (1/3)	03 soft	L	08	1	1
P53-87	bonnet (1/3)	03 soft	L	54	1	0
P70-85	bonnet-grille	06 soft	L	14	1	1
P28-85	bonnet-grille	06 soft	L	32	0	1
C18-01	roof rail	22 medium	O	46	-	1
C05-01	roof rail	22 medium	O	16	1	0
C23-01	roof rail	22 medium	O	16	-	0
C03-01	roof rail	22 medium	L	06	-	0
P15-88	bonnet (side)	05 medium	L	12	0	0
P37-87	bonnet (side)	05 medium	L	64	1	0
P29-86	windscreen	09 hard	O	39	1	1
P32-86	windscreen	09 hard	O	81	0	0
F2	concrete	28 hard	O	43	1	1
F3	concrete	28 hard	O	37	1	1
F4	concrete	28 hard	O	79	0	0
F5	ceramic floor	29 hard	O	77	0	1
F6	concrete	28 hard	O	84	0	1
F7	concrete	28 hard	O	42	1	1
F8	wooden door	29 hard	O	80	1	0
F9	concrete	28 hard	O	77	1	0
P18-85	A-pillar (ext)	11 hard	L	55	1	1
P51-87	windsceen	09 hard	L	17	1	0
F11	timber logs	29 hard	L	58	1	1
F12	concrete	28 hard	L	53	1	1
F13	concrete	28 hard	L	71	1	1
F15	concrete	28 hard	L	66	0	1
F16	concrete	28 hard	L	76	1	1
F17	wooden dresser	29 hard	L	74	1	0
F18	iron fence	29 hard	L	71	0	0

(1) P = pedestrian, C = car occupant, F = fall
(2) O = occipital, L = lateral
(3) ASDH = acute subdural haematoma
(4) 0 = absent, 1 = present

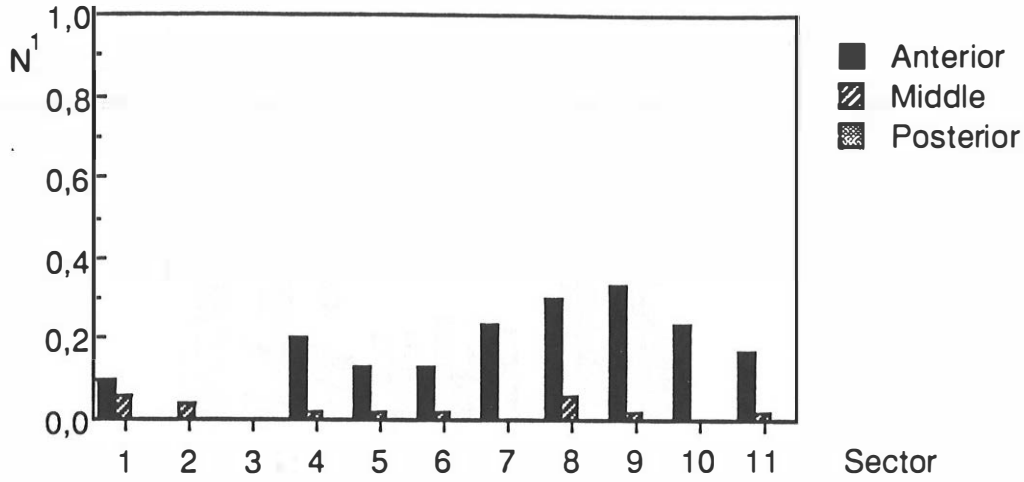


Figure 5. Hard occipital impacts (10 cases)

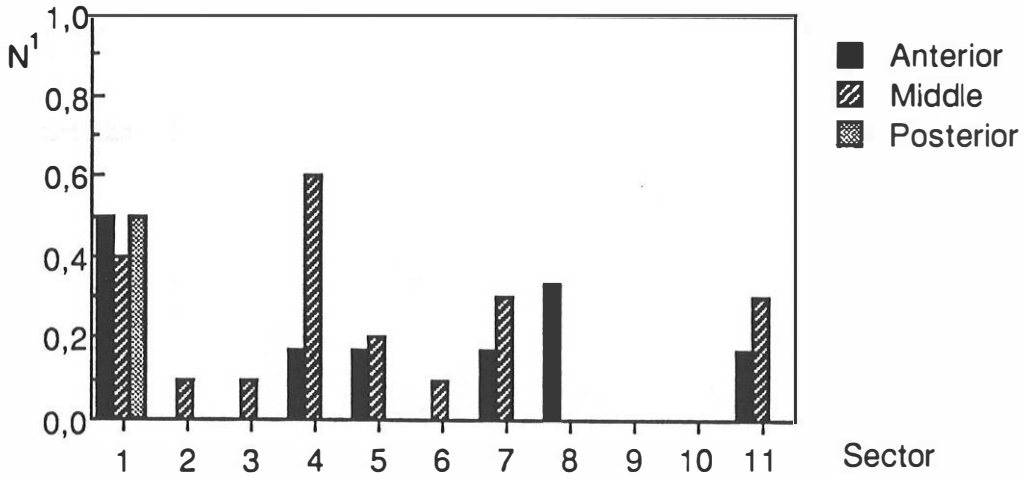


Figure 6. Medium occipital impacts (3 cases)

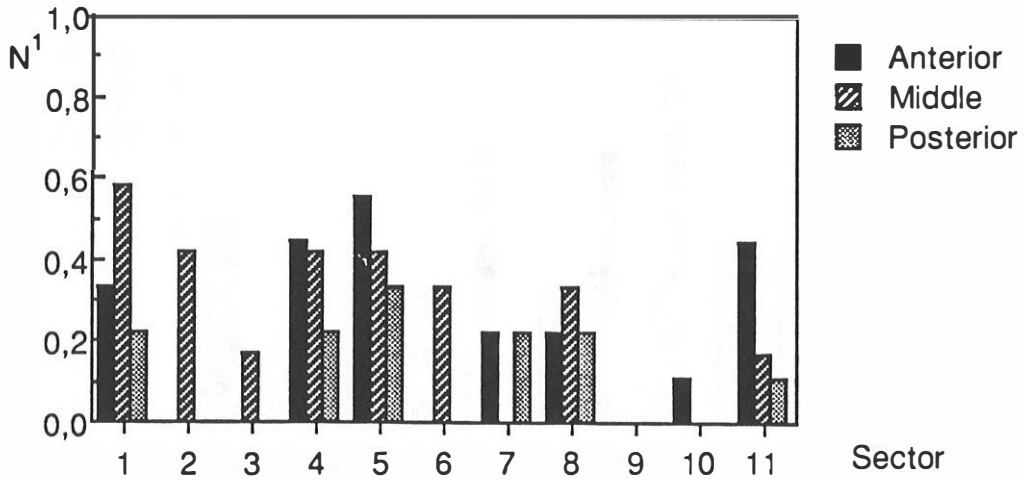


Figure 7. Soft occipital impacts (3 cases)

[1] N = Standardised number of sectors with injury

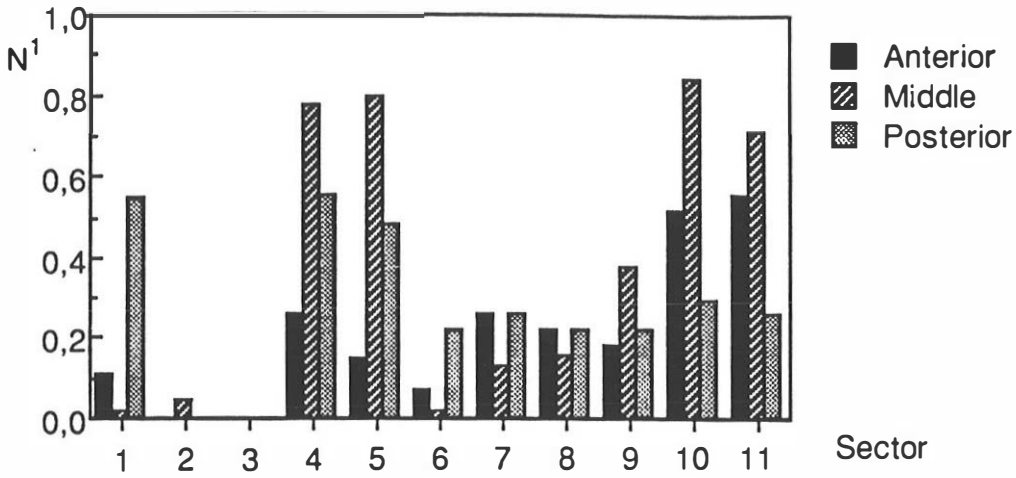


Figure 8. Hard lateral impacts (9 cases)

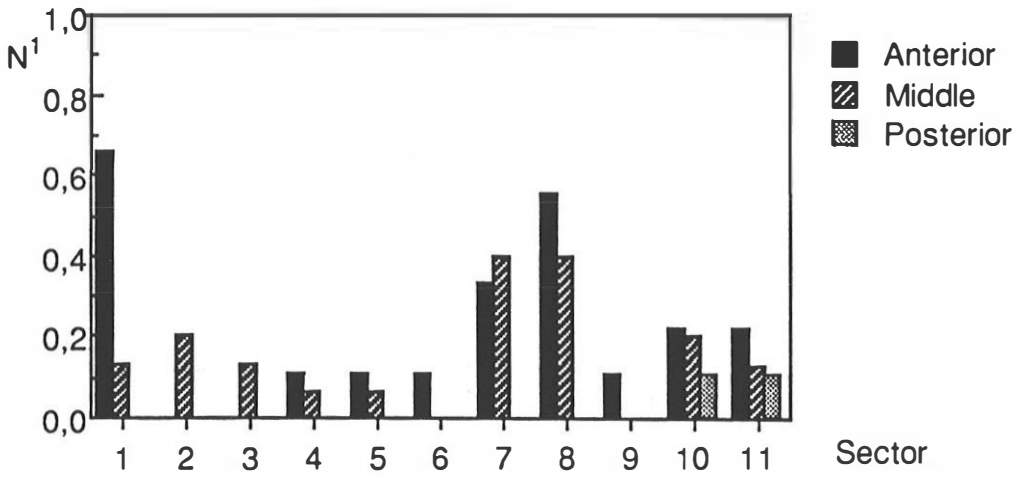


Figure 9. Medium lateral impacts (3 cases)

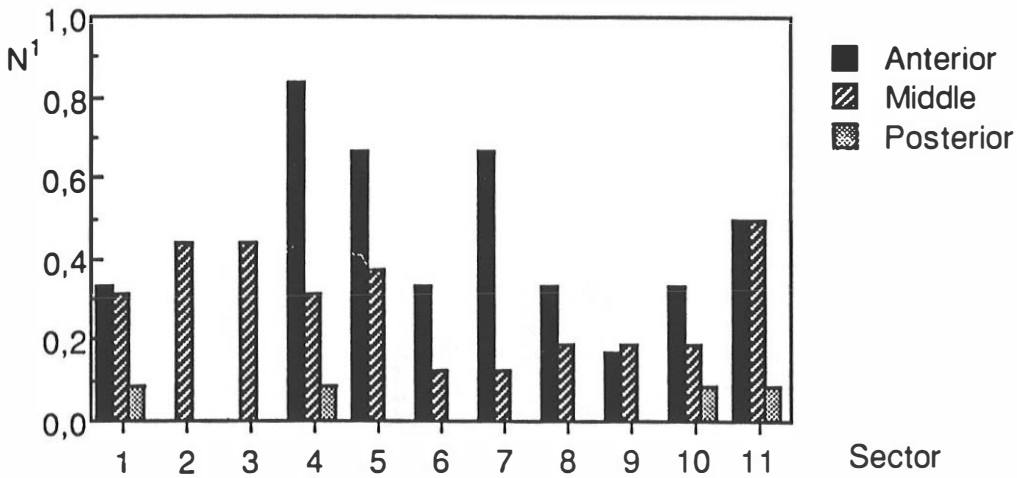


Figure 10. Soft lateral impacts (4 cases)

[1] N = Standardised number of sectors with injury

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