

BRAIN INJURY PATTERNS IN FALLS CAUSING DEATH

J Manavis¹, PC Blumbergs¹, G Scott¹, JB North²

DA Simpson³, GA Ryan³ and AJ McLean³

¹Head Injury Laboratory,
Division of Tissue Pathology,
Institute of Medical & Veterinary Science;

²Department of Neurosurgery,
Royal Adelaide Hospital;

³NHMRC Road Accident Research Unit,
University of Adelaide.

ABSTRACT

The NHMRC Road Accident Research Unit at the University of Adelaide has developed procedures for studying the brain injury patterns in fatal head injury cases and relating these to estimates of the acceleration of the head. Information obtained from clinical records and police accident investigation reports were correlated with autopsy data and detailed neuropathological examination of the fixed brain for each case.

The brain injury patterns resulting from impacts to the head in a series of 8 cases of falls from greater than the subjects' height are compared with 10 cases of falls from the subject's own height and related to estimates of the magnitude of the impacts.

Important factors associated with the pattern of brain injuries are the magnitude of the acceleration of the head, the location of the impact on the head, and the type of surface struck.

INTRODUCTION

Since 1981 the NHMRC Road Accident Research Unit has studied traumatic brain injury mechanisms operating in road accidents involving pedestrians (1), car occupants (2) and motorcyclists (3). In this paper we present the pattern of injury to the brain due to falls which resulted in death following admission to hospital.

METHODS

Case Selection

There were 22 patients who sustained a fatal head injury as a result of a fall and were admitted to the Neurosurgery Unit of the Royal Adelaide Hospital during the period 1983-1990; all 22 died in hospital. In four cases there were no adequate neuropathological data or information on the fall environment, such as height fallen and type of surface impacted. This left 18 cases which were suitable for detailed study. Eight were falls from greater than the victim's own height and 10 were falls from their own height.

Investigative Procedures

The steps in working up a case of fatal head injury resulting from a fall were as follows:

1. Data relating to the circumstances of the fall were obtained from the victim (if sufficiently lucid), and from eyewitness reports, police reports and the case notes of the Royal Adelaide Hospital.
2. The location of the impact point on the head was determined at autopsy from abrasions, bruises and skull fractures. Impact locations were classified as occipital, lateral or frontal (Fig. 1). Other information obtained from the post-mortem included a description of other injuries and the victim's height and body weight.
3. After removal at autopsy, the brain was placed in concentrated formalin and after a minimum fixation period of two weeks was coronally sectioned at 10mm intervals and examined macroscopically. Each section was photographed in black and white. Standard neuropathological sampling was undertaken on all cases. In addition, histological sections of each consecutive slice of the whole cerebral hemispheres were examined in 10 cases. This type of sampling gave at least 11 coronal sections at 10mm intervals through the cerebral hemispheres, at least 6 transverse sections of the brainstem and at least 4 sections of the cerebellum in the parasagittal plane.
4. Lesions were recorded using line diagrams of the 11 standard coronal cross sections of the cerebral hemispheres which we have developed for this purpose (Fig. 2). The first format used was described at a previous IRCOB conference (1). The present format conforms more closely to areas supplied by different vascular beds. There are now up to 3 central and 8 radial sectors, totalling 105 sectors for the cerebral hemispheres. The central sectors consist of the left and right central grey matter and the corpus callosum. Haemorrhage (microscopic and macroscopic) was the main marker for vascular injury.

Head Impact Calculations

Head impact velocities and head accelerations were estimated from the assumed fall height and the nature of the surface struck by the head. With few exceptions, the struck surface was effectively rigid and so the combined stiffness of the head/struck surface has been taken as the stiffness of the head, which we have estimated to be 1,300 kN/m (1). The resulting acceleration of

the head on impact was calculated using the formula $a = v \sqrt{\frac{K}{m}}$ where v is the estimated head impact velocity, K is the stiffness and m is the mass of the head.

Brain Injury Severity

Lesions which were evident on macroscopic or microscopic examination of each brain were recorded on the line diagrams of the 11 coronal sections. Primary impact lesions such as haemorrhages (macroscopic or microscopic), which act as markers of vascular injury, and lacerations or areas of tissue disruption were carefully recorded. Secondary brain lesions such as hypoxic-ischaemic damage and changes consequent to raised intracranial pressure were ignored. The presence of diffuse axonal injury will be the subject of a separate report.

The following injuries to the cortex or white matter of each sector of each cross section (Fig. 2) were recorded: small haemorrhages (less than full cortical thickness in the cortical mantle or less than 5mm diameter in the white matter), confluent haemorrhages (full cortical thickness in the cortical mantle and greater than 5mm in diameter in the white matter) and loss of tissue. Lesions in the 3 central sectors (central grey matter and corpus callosum) were also recorded in a similar manner.

The distribution of injury for each brain was then displayed in the diagram shown in Figure 2. The Haemorrhagic Lesion Score (HLS) was calculated by summing the number of sectors of

the brain over all cross sections in which such lesions were present; no weighting was given to lesions of greater or lesser magnitude.

RESULTS

The characteristics of the 18 cases studied are summarised in Tables 1-3. Ages ranged from 28 to 84 years; there were 13 males and 5 females. There were 9 occipital impacts, 8 lateral impacts and one frontal impact. The impacts resulted in linear accelerations of the head which we estimated to range from about 2000 to 7000 m/s². In 13 cases the struck surface was a concrete floor. The other surfaces included timber objects (3 cases), a bitumen road and a wrought iron fence. The HLS ranged from 0 to 63.

Severity of Impact and Extent of Injury

The relationship between HLS and severity of impact as measured by the estimated acceleration of the head at impact is shown separately for occipital and lateral impacts in Figures 3 and 4. For occipital impacts (Fig. 3) linear accelerations ranged from about 3000 to 7000 m/s²; the HLS generally increased with increasing acceleration.

For lateral impacts (Fig. 4) linear accelerations ranged from 2000 m/s² to 6000 m/s² and there was a trend for HLS to increase with increasing linear acceleration. However beyond an acceleration level of about 3000 m/s² there was no clear evidence of any further increase in HLS.

Distribution of Injury

We have examined the effects of increasing levels of acceleration on the distribution of vascular injury for occipital and lateral impacts.

Occipital Impacts

Figures 5 to 8 show the number of brains with one or more haemorrhagic lesions in any given sector. Figure 5 shows that for occipital impacts resulting from falls from the victim's own height (estimated linear acceleration of the head ≤ 3000 m/s²) the injuries were concentrated in the inferior frontal and temporal lobes of the cerebral hemispheres except in one case where there were occipital lacerations and contusions beneath a left occipital fracture. Two cases showed only acute subdural haematomas (ASDH) without any parenchymal damage. There was no injury to the central region of the brain.

Figure 6 shows the pattern of injury in occipital impacts from falls from greater than victim's own height (estimated linear acceleration > 3000 m/s²). The injuries once again involved the inferior frontal and temporal lobes. One case also showed damage to the occipital lobes directly underlying an extensive left occipital fracture. Injury to the central region was also present.

Lateral Impacts

Figure 7 refers to lateral impacts in falls from the victim's own height (estimated linear acceleration ≤ 3000 m/s²). The injury patterns are presented in Figure 7 as though the impacts were all on the right side of the head. This is done on the assumption that the head and brain are symmetrical about the longitudinal axis. There was no injury to the central region in this group. One of the cases sustained a pure bridging vein rupture resulting in an acute subdural haematoma without any parenchymal damage. Case 18 (Table 3) showed parenchymal damage on the side of the impact in association with an ASDH and no evidence of contralateral parenchymal damage.

Figure 8 refers to lateral impacts in falls from greater than the victim's own height (> 3000 m/s²). Once again the injury distributions of these cases are presented in Figure 8 as though the

impacts were all on the right side of the head. All four cases were associated with skull fractures and massive acute subdural haematomas. In these lateral impacts the parenchymal damage was concentrated beneath the impact site and in the opposite hemisphere. There was also evidence of injury to the central region in this group.

DISCUSSION

Acute Subdural Haematoma

Acute subdural haematoma (ASDH) is found in about 30% of severe head injuries from all causes and it has been estimated that 60% of patients with ASDH will die (4). ASDH may arise by several different mechanisms (5) of which tearing of the bridging veins that traverse the subdural space from the brain's surface to the various dural sinuses is an especially important type. In most of these fatal falls the head impacted a very hard surface (usually concrete) which itself deforms very little. Thus there is usually a short impact duration and abrupt deceleration which creates the conditions which Gennarelli and Thibault believe to result in rupture of bridging veins (4).

ASDH occurred in 14/18 of our falls (7/8 lateral impacts, 1/1 frontal impact and 6/9 occipital impacts). These results are similar to those described in a previous study of traumatic ASDH where 72% of cases of ASDH attributed to rupture of the bridging veins were the result of falls or assaults and only 24% were due to motor vehicle related accidents (4). The authors of that study showed that three factors were crucial for rupture of the bridging veins, 1) the magnitude of the acceleration of the head, 2) the rate of acceleration onset and 3) the duration of the acceleration. There was also a high incidence of ASDH in the series of falls reported by Seelig et al. (6).

In our material the four patients with ASDH and no other primary traumatic brain damage or skull fracture, (ie bridging vein rupture type of ASDH) had the lowest estimated head accelerations (2500 m/s² to 3000 m/s²). See Tables 1-3.

Coup and Contrecoup Contusions

Lindenberg (7 & 8) reported that contusions caused by falls in which the head impacts an object much more massive than itself tended to occur opposite the site of the impact. Such lesions are often called contralateral (contrecoup) contusions. They occur with far greater frequency at the frontal and temporal poles and on the inferior surfaces of the frontal and temporal lobes than they do elsewhere in the brain. Contralateral (contrecoup) contusions are rare at the occipital poles. However this may largely be a consequence of a higher frequency of occipital rather than frontal impacts in falls and a greater susceptibility to injury of the frontal and temporal regions of the brain. It has been generally accepted that contusions are very rarely sustained beneath the point of cranial impact in falls, i.e. no ipsilateral or coup contusions, provided there has been no fracture of the skull (9).

We observed that, in fact, fractures of the skull occurred in 12 of the 18 cases and six of these 12 showed associated ipsilateral (coup) fracture contusions. All 12 cases associated with skull fractures showed the expected contralateral (contrecoup) contusion pattern of damage, which is contrary to a hypothesis derived from experimental evidence (10) in which skull fracture is thought to reduce the probability of contralateral injury. In four of the six cases without skull fractures there was massive ASDH but no evidence of parenchymal damage. One case showed parenchymal damage on the same side as the impact in association with ASDH (case 18), and case 4 was the only case with contralateral (contrecoup) parenchymal damage (Tables 1-3).

Thus in this series only one of the 18 cases of fatal head injury resulting from falls showed a pure pattern of contralateral (contrecoup) damage of the type discussed by Dawson et al. (9).

Occipital Impacts

In seven of the nine occipital impacts (Table 1), contusions of varying severity involved the frontal and temporal lobes, i.e. in the contrecoup position. The other two cases of occipital impact

both sustained ASDH secondary to rupture of a bridging vein without any other skull or brain damage. One of these patients was receiving anticoagulant medication before the fall which is known to predispose to unwanted bleeding.

Three of the 9 occipital impacts resulted in contusions at the site of the impact of the head and these were all associated with fractures (fracture contusions).

Lateral Impacts

Contusions of varying severity involved the frontal and temporal lobes in six of the eight lateral impacts. The other two cases of ASDH attributed to bridging vein rupture had no other type of primary traumatic damage and were associated with the lowest head accelerations in the series (2000 and 2500 m/s²) (see Table 3). Three of the eight lateral impacts showed ipsilateral (coup) contusions associated with fractures. In this series of 8 cases there were no cases of the contralateral (contrecoup) pattern of damage occurring as the sole type of traumatic brain damage.

Frontal Impacts

Only one of the 18 cases sustained a frontal impact on the head. This resulted in a bridging vein rupture type of ASDH (Table 2). There were no contralateral (contrecoup) occipital contusions in this case.

CONCLUSIONS

1. An acute subdural haematoma (ASDH) was present in 14 out of 18 (78%) cases admitted to hospital with ultimately fatal head injury due to a fall. In 10 of the 14 cases with ASDH there were other types of traumatic brain and skull damage present as well. Four cases of ASDH attributed to bridging vein rupture had no other primary brain lesion, and were associated with the lowest head accelerations.
2. Skull fractures were present in 12 of the 18 cases (66%). Six of these 12 cases were associated with contusions adjacent to the impact and all 12 showed contralateral contusions.
3. Thirteen of the 18 cases showed haemorrhagic parenchymal lesions contralateral to the site of impact, but only one showed contralateral parenchymal damage as the sole type of brain damage.
4. Lesions in the central sectors of the brain were only present in victims who had fallen from a distance greater than their own height.
5. Wide variations in our measure of the extent of brain injury (Haemorrhagic Lesion Score) were observed in cases of similar head impact severity.

ACKNOWLEDGEMENTS

The authors wish to thank the staff of the Accident Investigation and Prevention Section, South Australian Police, the State Coroner for their willing cooperation, Craig Kloeden for the computer generated figures, Chris Snook from the University of Adelaide Engineering Department, for assistance in estimates of head velocity and acceleration, and Rae Tyler for the typing of the manuscript. This work was supported by the Australian National Health and Medical Research Council.

REFERENCES

1. Ryan GA, McLean AJ, Vilenius ATS, Kloeden CN, Simpson DA, Blumbergs PC. Head impacts and brain injury in fatally injured pedestrians. In: Proceedings of International IRCOBI Conference on the biomechanics of impacts. Bron (France): International Research Council on Biokinetics of Impacts, 1989; 276-37.
2. Simpson, Ryan GA, DA Paix BR, Blumbergs PC, Scott G, Kloeden CN, McLean AJ. Brain injuries in car occupants: a correlation of impact data with neuropathological findings (1991 IRCOBI conference).
3. Cooter RD, David DJ, McLean AJ, Simpson DA. Motorcyclist helmet-induced fatal skull base fracture. *Lancet* 1988; 1: 84-85.
4. Gennarelli TA, Thibault LE. Biomechanics of acute subdural haematoma. *Trauma* 1982; 22: 680-686
5. Jones NR, Blumbergs PC, North JB. Acute subdural haematomas: aetiology, pathology and outcome. *Aust NZ J Surg* 1986; 56: 907-913.
6. Seelig JM, Becker DP, Miller JD, Greenberg RP, Ward JD, Choi SC. Traumatic acute subdural haematoma: Major mortality reduction in comatose patients treated within four hours. *N.Eng.J.Med.* 1981; 304: 1511-1518
7. Lindenberg R. Mechanical injuries of brain and meninges. In: WV Spiz and RS Fisher (eds), *Medicolegal investigation of death*. Springfield: Charles C Thomas, 1973: 447-450.
8. Lindenberg R, Freytag E. The mechanism of cerebral contusions - A pathologic anatomic study. *Am Med Assoc Arch Path* 1960; 69: 440-469.
9. Dawson SL, Hirsch CS, Lucas FV, Sebok BA. The contrecoup phenomenon: reappraisal of a classic problem. *Human Pathology* 1980; 3: 155-166.
10. Yanagida Y, Fujiwara S, Mizoi Y. Differences in the intracranial pressure caused by a "blow" and/or a "fall" - an experimental study using physical models of the head and neck. *Forensic Science International* 1989; 41: 135-145.

Figure 1: System specifying the location of the impact to the head

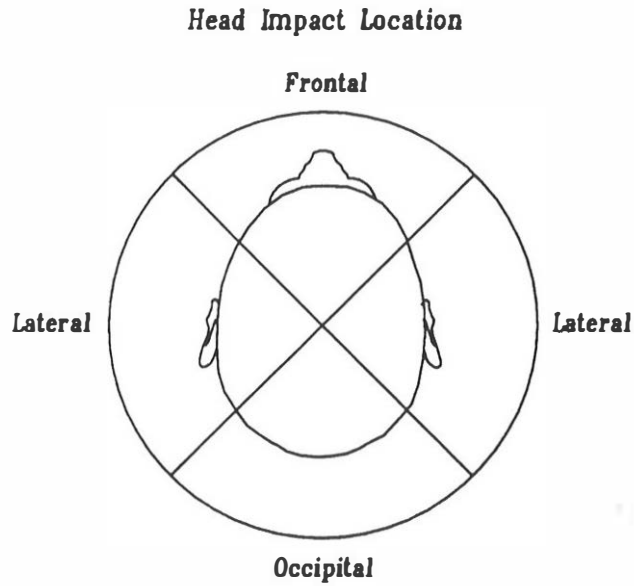
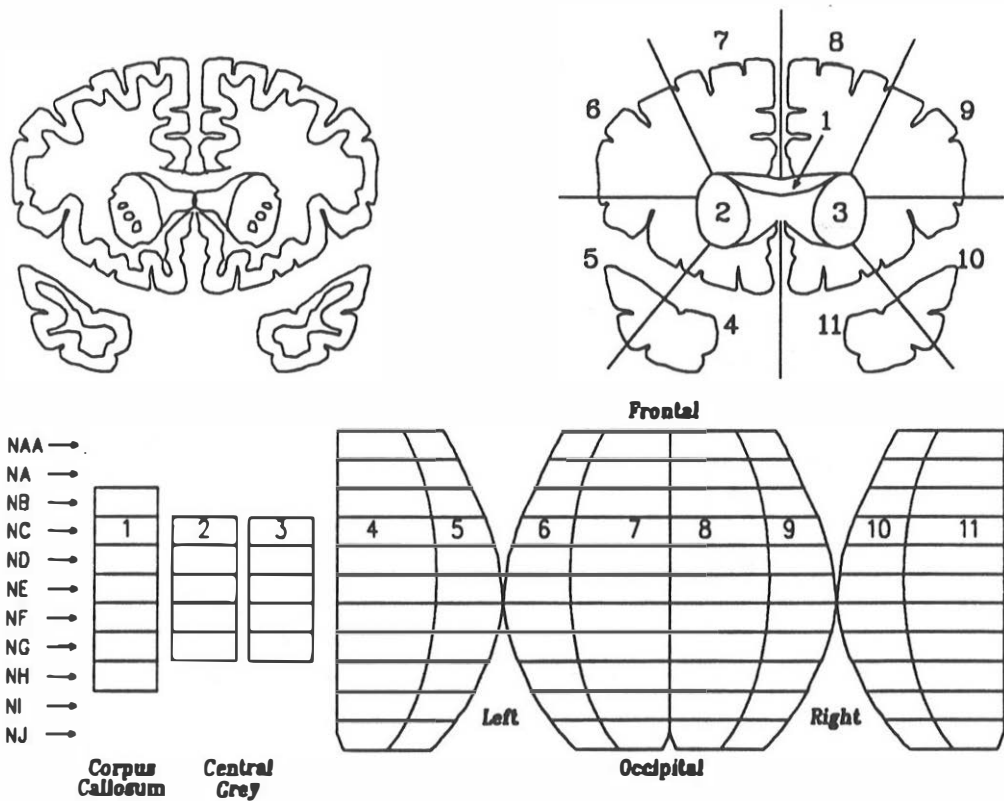
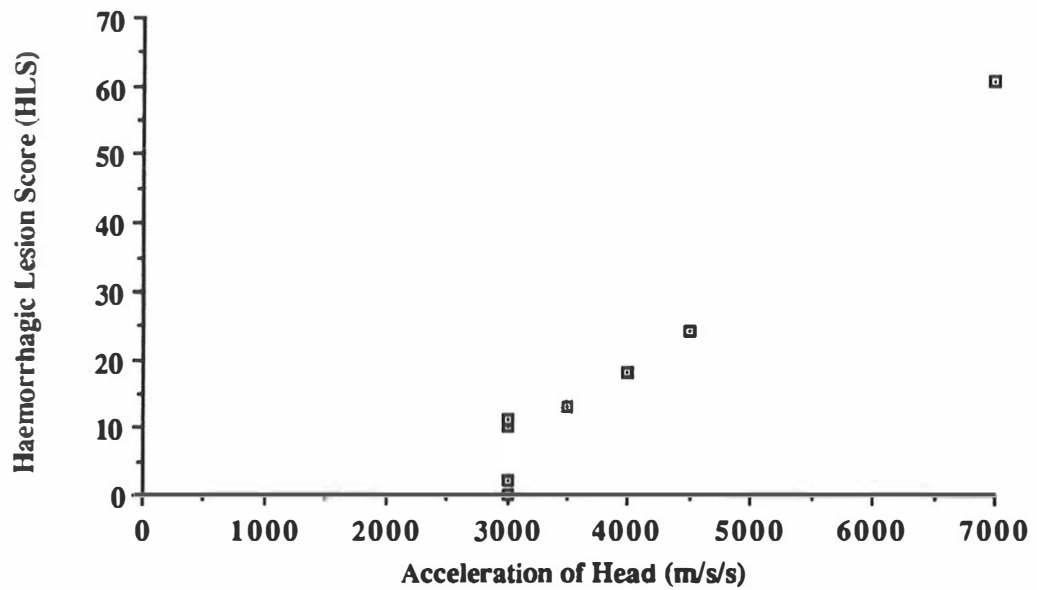


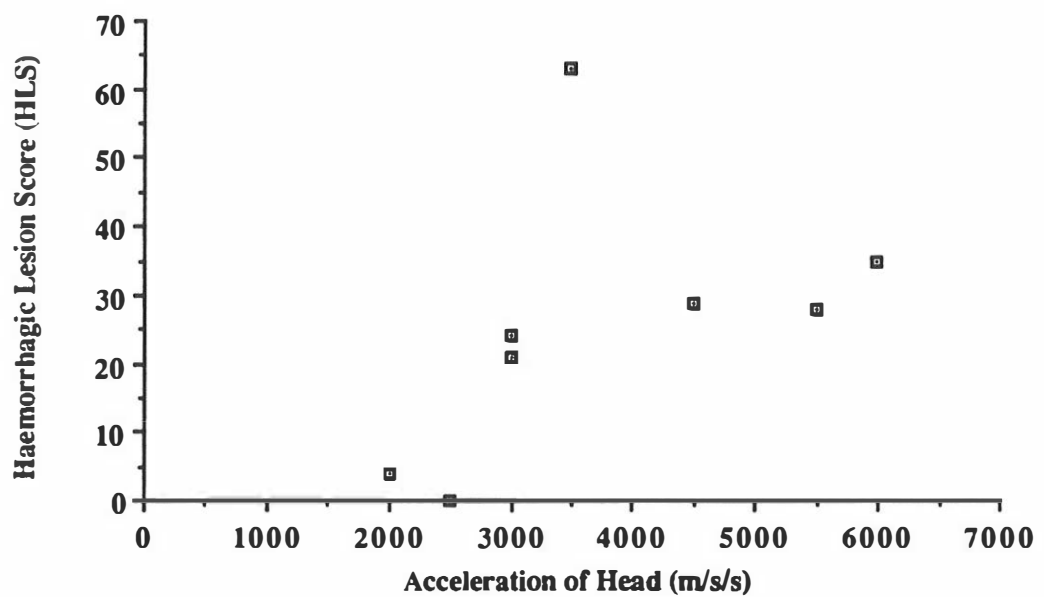
Figure 2: Diagrams used for recording injury to each section of the brain



**Figure 3: Haemorrhagic Lesion
Score by linear acceleration of the head : Occipital Impacts**



**Figure 4: Haemorrhagic Lesion
Score by linear acceleration of the head : Lateral Impacts**

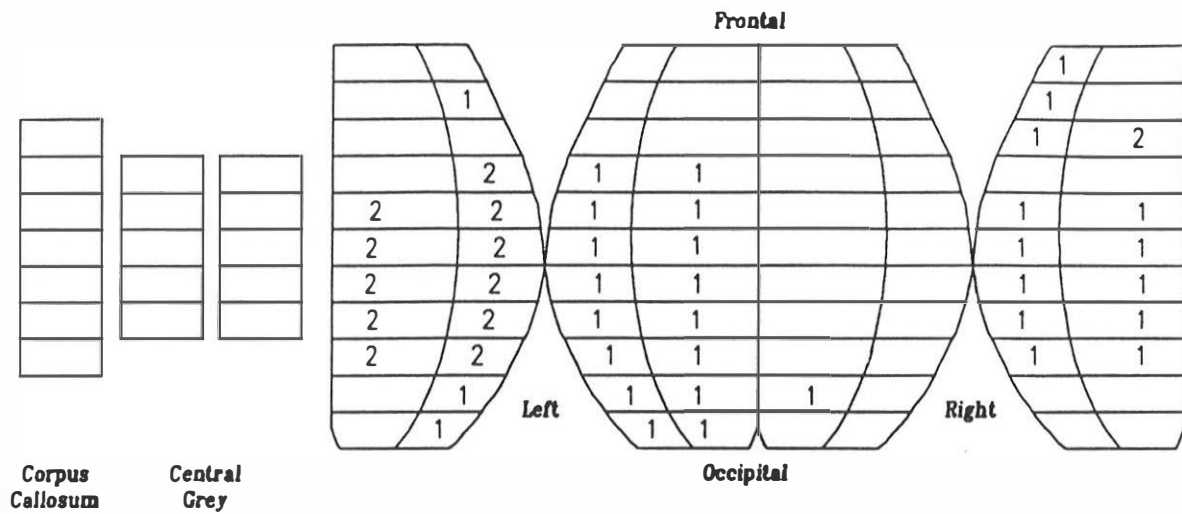


The diagram illustrates the distribution of 1000 voxels across a human brain. The brain is divided into four main regions: Frontal, Occipital, Left, and Right. The Corpus Callosum and Central Grey matter are also labeled. The distribution of voxels is as follows:

Region	Voxel Count
Frontal	1000
Occipital	100
Left	100
Right	100
Corpus Callosum	100
Central Grey	100

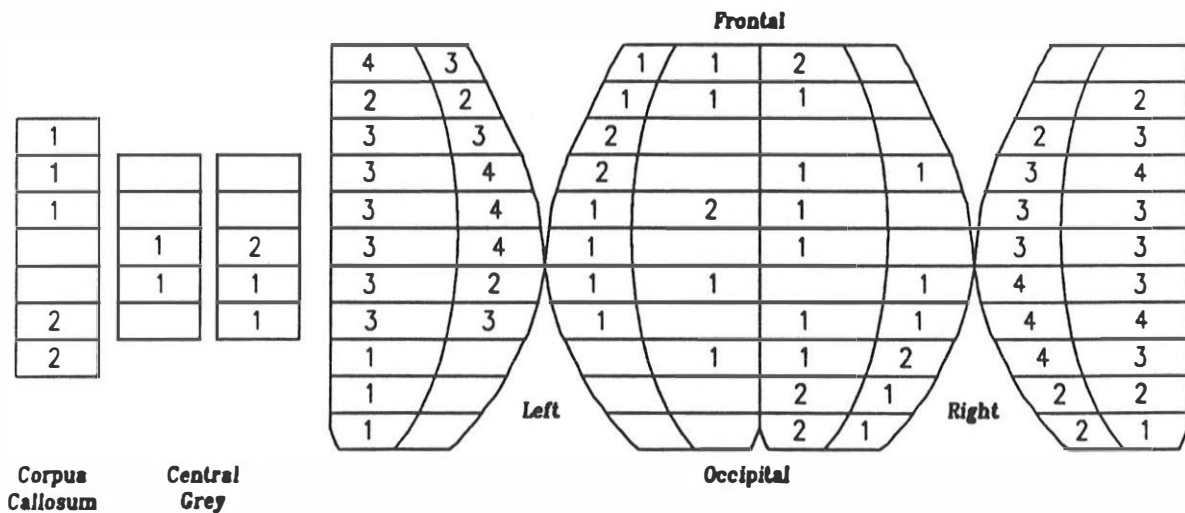
[illegible]

**Figure 7: Frequency Distribution of Haemorrhagic Lesions
for 3 Lateral Impacts $\leq$ Own Height ($\leq 3000 \text{ m/s}^2$)**



(Lesions plotted as though all impacts were on the right side of the head)

**Figure 8: Frequency Distribution of Haemorrhagic Lesions
for 4 Lateral Impacts $>$ Own Height ($> 3000 \text{ m/s}^2$)**



(Lesions plotted as though all impacts were on the right side of the head)

TABLE 1: OCCIPITAL IMPACTS

Case Number	Age	Sex	Survival Period	Fall Height (m)	Impact Surface	Estimated Impact Velocity (m/s)	Estimated Linear Acceleration (m/s/s)	HL Score	Acute Subdural Haematoma	Skull Fracture (+/-)	Posterior (Ipsilateral)	Haemorrhagic Lesions Anterior (Contralateral)
1	28	M	1 day	9.40	Concrete	14	7000	61	Left	+	Cerebellum & Occipital lobes	Inferior Frontal & Temporal lobes
2	43	M	10 days	3.80	Concrete	8.6	4500	24	Right	+	Cerebellum	Burst R frontal
3	37	M	1 day	3.30	Concrete	8.0	4000	18	Right	+	-	Inferior Frontal & Temporal lobes
4	79	M	22 days	2.15	Concrete	6.5	3500	13	-	-	-	R frontal lobe
5	77	M	2 days	1.87	Ceramic floor	6.1	3000	10	-	+	-	L Frontal & Temporal lobes
6	84	F	1 day	1.85	Linoleum on Concrete floor	6.0	3000	2	-	+	-	R Frontal & L Temporal lobes
7	42	M	5 hours	1.80	Concrete	5.9	3000	11	Left	+	L Occipital lobe with lacerations	L Frontal & L Temporal
8	80	F	3 days	1.70	Heavy Wooden door	5.8	3000	0	Left	-	-	-
9	77	F	2 days	1.67	Concrete	5.7	3000	0	Left	-	-	-

TABLE 2: FRONTAL IMPACTS

Case Number	Age	Sex	Survival Period	Fall Height (m)	Impact Surface	Estimated Impact Velocity (m/s)	Estimated Linear Acceleration (m/s/s)	HL Score	Acute Subdural Haematoma	Skull Fracture (+/-)	Ipsilateral Haemorrhagic Lesions	Contralateral
10	68	M	13 hours	1.73	Linoleum on Concrete	5.8	3000	0	Right	-	-	-

TABLE 3: LATERAL IMPACTS

Case Number	Age	Sex	Survival Period	Fall Height (m)	Head Impact Site	Impact Surface	Estimated Impact Velocity (m/s)	Estimated Linear Acceleration (m/s/s)	HL Score	Acute Subdural Haematoma	Skull Fracture (L/R)	Ipsilateral Haemorrhagic Lesions	Contralateral
11	58	M	5 days	7.90	L	Timber logs	12	6000	35	Right	Left	-	R frontal
12	53	M	6 days	5.85	R	Concrete	11	5500	28	L>R	Right	R Temporal	L Temporal
13	71	M	9 days	3.75	R	Concrete	8.6	4500	29	Right	Right	-	L frontal L Temporal
14	57	M	4 hours	2.40	R	Bitumen road	6.9	3500	63	Left	Right	R Frontal	L Frontal & Temporal
15	66	M	12 days	1.78	L	Concrete	5.9	3000	21	-	Left	L Temporal	R Temporal
16	76	F	5 days	1.73	L	Concrete	5.8	3000	24	Right	Left	-	R Burst lobes
17	74	M	2 days	1.15	R	Wood dresser	4.8	2500	0	Right	-	-	-
18	71	F	22 days	.73	R	Iron fence	3.8	2000	4	Right	-	R Frontal	-