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Abstract The report discusses the characteristics of head and neck injuries sustained by occupants of passenger cars. The epidemiology and biomechanics of such injuries are discussed. The extent and prognosis of treatment is described. Both existing countermeasures and current research into new or improved countermeasures are evaluated. Recommendations are made with respect to measures to reduce head injury in passenger vehicles.

KEYWORDS:

NOTES:

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HEAD AND NECK INJURIES IN PASSENGER CARS: A Review of the Literature

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1. INTRODUCTION

The following review of the nature and causes of head and neck injuries sustained by the occupants of passenger cars involved in a crash has been conducted to assess the potential for the reduction of the frequency and severity of these injuries in Australia.

The review commences with a discussion of the characteristics of head and neck injuries of the type occurring in passenger cars. These sections are followed by a review of the epidemiology of head and neck injuries sustained by car occupants and a discussion of the biomechanics of such injuries. The extent to which head and neck injuries can be treated successfully is then described. The development and effectiveness of existing countermeasures and current research into new or improved methods of either preventing or ameliorating the severity of head and neck injuries completes the review of the literature.

No attempt has been made to review the literature on crash prevention, even though prevention of the crash obviously eliminates the possibility of crash injury. The emphasis in this review is on injury control, given that the occupant is in a car which is involved in a crash.

The prospects for recovery are considered in the chapters on treatment of head and neck injuries. The review does not deal extensively with the long term outcome for a person who has sustained a head injury.

The report concludes with recommendations for action and for further studies.

An bibliography of the relevant literature is presented in an appendix.

2. CHARACTERISTICS OF HEAD INJURIES

2.1 INTRODUCTION

Considered as an anatomical region, the head includes the brain, the organs of sight, hearing, smell and taste, the upper digestive tract (mouth, jaws, teeth and tongue), and the upper airway (nose, mouth, upper pharynx). These structures are all liable to injury, either singly or in combinations, in car crashes.

In studying traffic crashes, it is usual to classify head injuries in two groups:

(1) Skull and brain

(2) Ear, eye and face

This subdivision is used in the Abbreviated Injury Scale (AIS) and will be followed here (Gennarelli et al., 1985). The AIS codes injuries in six grades of severity, from 1 (minimal injury) to 6 (injury usually incompatible with life). These grades are designed solely to quantify the severity of the initial injury, and to describe the immediate threat to life: the AIS does not categorise types of injury, and should not be used as a guide to outcome, since this may be affected by the quality of treatment.

Table 2.1 shows in simplified form how the different pathological types of brain injury relate to AIS grades. It is seen that injuries which are very different in their causes, management, and likely outcome (e.g. surface clot and brain laceration) receive the same grade of severity. In the same way, injuries of the eye and ear of very different significance are subsumed under a single numeral. Many of the reports reviewed by us use the AIS advantageously to categorise injury severity; however, in a study of the pathology of the brain injuries sustained by car occupants, it

is necessary to consider the various clinico-pathological categories of injury, and in many otherwise informative studies of crashes these are not identified. Conversely, many neuropathological reports do not identify the causes of injury, beyond listing vehicular crashes as a single group. This has made this aspect of the literature review in some respects unsatisfactory.

For the clinician and for the neuropathologist, it is important to distinguish between open injuries, in which the scalp and skull are penetrated, with risk of infection and likelihood of local brain damage, and closed injuries, in which violence - usually deceleration - is transmitted through the intact skull to the enclosed brain (Fig. 1). Closed head injuries are characterised by widespread damage to nerve fibres (axons) and blood vessels, and by bruising (contusion) of localised areas of brain. These two fundamental types of primary brain injury can occur separately or together. Both are seen in car occupants. Both may be complicated by secondary causes of brain injury, such as lack of oxygen (hypoxia), or compression by bleeding within the skull. Secondary processes are of great clinical importance since they are potentially curable if recognised soon enough. This is especially true of intracranial bleeding over the surface of the brain (extradural and subdural haemorrhage: see Fig. 2).

The AIS also codes injuries of the ear (1-2), eye (1-3) and facial skeleton (1-4). Again, the clinico-pathological categories of injury are more important than their severity: however, the pathology of these injuries is much less complex than the pathology of brain injuries, and most published reports describe both the injury categories and the causes of injury. Hearing may be impaired either by injury to the small bones (ossicles) of the middle ear, which can be rectified, or by damage to the

inner ear or auditory nerve - a cause of irreparable deafness. Vision may be impaired by open (penetrating) or closed violence to the globe of the eye, or by damage to the optic nerve - a cause of irreparable blindness. Facial injuries are classified according to the anatomy of the damage, and especially the skeletal damage: facial fractures are subdivided into three regions - upper third (forehead and root of nose), middle third (nose, upper jaw and cheekbones), and lower third (mandible or lower jaw).

2.2 REVIEW OF LITERATURE

2.2.1 SKULL AND BRAIN

Car occupants often suffer injuries of the skull and brain, and these injuries cause the majority of all road deaths. Selecki et al. (1981) found that in NSW in 1977, neurotrauma (head and spinal injury) accounted for 68.0% of 518 deaths of drivers and 64.9% of 342 deaths of passengers. Non fatal brain injuries are much more numerous. In a U.K. study of 14019 car occupants, Rutherford et al. (1985) found that 1721 suffered minor (AIS 1, 2) brain injuries and 139 suffered major (AIS$\geq$3) brain injuries, giving an incidence of 13.3% among car occupants admitted to hospital: the study excluded deaths occurring before admission.

Another recent U.K. study, by Bradford et al. (1986) gave a more complete picture: of 1603 injured car occupants, 182 sustained minor (AIS 1, 2) head injuries, 31 severe (AIS $\geq$ 3) non fatal injuries, and 58 died with head injuries, though in only 30 deaths was it found that the head injury was the most severe or only injury. Thus, in this series of car occupants, head injury was recorded in 16.9% of all cases, but in 65% of all deaths.

Understanding of the basic mechanisms of brain injury has advanced greatly during the last decade. Neuropathological studies, ably reviewed

by Adams et al. (1985), have shown that in closed head injury due to acceleration, there are two main forms of brain damage:

(1) diffuse, where nerve fibre (axon) systems and blood vesels are torn by shearing stresses (Strich, 1956); Adams et al. (1977) have described the damage to nerve fibres as diffuse axonal injury (DAI).

(2) focal, where specific parts of the brain, notably the frontal and temporal lobes, are impacted against the interior surface of the skull and bruised or torn (contusions).

Gennarelli and Thibault (1982) have shown in animal (primate) experiments that angular acceleration is especially detrimental. These workers have also shown that the pathological effects of experimentally induced acceleration vary with the direction and the rate of acceleration. High rates of angular acceleration ($> 1 \times 10^5$ rads/sec^2) operating for a short period (< 5 millisecs) are likely to tear veins which then bleed, resulting in potentially lethal compression of the brain by surface (subdural) clot formation. Similar angular acceleration over a longer period is likely to cause widespread tearing of nerve fibres (DAI). In the discussion that followed this important paper, it was argued that these findings could be related directly to the brain injuries of car occupants, and that better impact attenuation might only produce more cases of survival with DAI. Whatever the deductions from Gennarelli's data may be, it would be unwise to assume that these experiments exactly mimic the injuries of car occupants, especially with respect to subdural clots. Jones et al. (1986) considered that the relative rarity of acute subdural clots among car occupants could relate to accelerations of longer pulse duration, but this relative rarity was not seen in the large NSW series reported by Stening et al. (1986), in which 23% were car occupants. DAI,

cerebral contusions, and acute subdural haemorrhage are now recognised as the major pathological effects of closed head injury, and DAI is especially important as a cause of permanent disability: head injured patients who survive in the vegetative state, or with severe disabilities after prolonged periods of unconsciousness, are now seen as likely to have sustained irreversible DAI at the time of impact (Strich, 1956; Jennett and Plum, 1972; Adams et al., 1977). However focal primary injury, especially frontal and temporal contusions, may be the basis of other disabilities, especially disturbances of personality (Walsh, 1985).

Pathological studies, especially those of the Glasgow school (Graham et al., 1978; Adams et al., 1985) have also emphasized the importance of secondary causes of brain damage. Local or general impairment of oxygen supply (hypoxia) causes impaired function in nerve cells, which indeed die after only a few minutes of total oxygen deprivation. Raised pressure within the skull, whether due to a surface clot or to swelling of the injured brain (Tornheim et al., 1984), is also harmful and can cause death or permanent disability. It is especially in the prevention or treatment of the secondary pathological complications of head injury that better logistic and clinical management may give better results: our studies of preventable causes of death after head injury provide support for this hope (Simpson et al., 1984; Selecki et al., 1986).

Computerized tomography (CT scanning) makes it possible to visualise in the living patient most of the pathological processes resulting from head injury (Bartlett & Neil-Dwyer, 1979), with great clinical benefit (Teasdale et al., 1982). Most contusions, surface clots, and most forms of brain swelling can be seen in CT scans, and sequential scanning over time allows clinical progress to be studied. CT scanning became widely available in Australia in 1976, and is now regarded as virtually

indispensable in head injury management (Neurotrauma in Australia, 1986). It must however be pointed out that CT scanning will not visualise all forms of brain injury; in particular, DAI is not shown, though its presence may be sometimes inferred from the finding of haemorrhage in significant sites (Zimmermann et al., 1978; Lobato et al., 1986).

Another area in which there has been an important advance is the understanding of the pathology of minor head injuries (AIS 1, 2), often termed concussions, a term that is probably now best discarded (Simpson 1979) since it has been given different meanings in different lay, medical, and medicolegal contexts. Minor head injuries are characterised clinically by brief loss of consciousness and rapid return to apparent normality. In the past it has been said that such injuries, by definition, are completely reversible; as a corollary, those who complain of persisting symptoms after minor head injury have been considered to be neurotic (Miller, 1961). Workers in Auckland NZ have however clearly shown that such injuries may cause prolonged and possibly permanent impairment of memory (Gronwall and Wrightson, 1974, 1975; Wrightson and Gronwall, 1981). There is little doubt that these disturbances of memory result from brain injury, albeit sometimes magnified by neurosis or a lust for compensation. Oppenheimer (1968) reported microscopic changes in the brain after apparently trivial injury, and Povlishock et at (1983) found neuronal damage in cats subjected to injuries considered to be comparatively slight. It is suggested (Adams et al., 1977) that less severe acceleration injuries inflict axonal damage comparable with the DAI seen after major head injury, though of much less magnitude and perhaps localised to a vulnerable part of the brain, e.g. the brainstem reticular formation. The subject of minor head injury is reviewed at length by Alves and Jane (1985); these authors do not identify the minor head injuries of car occupants specifically, but it is known that

car occupants do suffer such injuries very frequently: as noted earlier, Rutherford et al. (1985) reported a high incidence of such injuries (AIS 1 & 2), with reductions of 34.2% in drivers and 58.1% in front seat passengers after the introduction of legislation enforcing seat belt wearing (see section 8.2.1).

The pathology of open head injuries sustained by car occupants has received much less attention, doubtless because they are much less common than closed head injuries. The literature, and personal experience, confirm that such injuries do occur. Windscreen injuries of the brain have attracted some attention: Rushworth and Toakley (1969) reported five such cases, all inflicted by toughened glass. Laminated windscreens appear to be less likely to inflict such injuries. Penetrating injuries from projecting objects within cars, or sharp objects struck by ejected car occupants, are sometimes mentioned in reports on large series of cases; however, as stated earlier, they are not separately identified in the AIS system. Road crashes figure in large series of compound depressed skull fractures, and children are especially likely to suffer such injuries, since their skulls are thin and easily shattered or penetrated. Braakman and Jennett (1975) reported on nearly 1000 depressed skull fractures from centres in the U.K. and Holland: 440 (47%) of these were due to traffic crashes, and half of these were under 16 years old. Unfortunately, this paper does not distinguish injured car occupants from other road users. Personal experience suggests that open head wounds are not rare among injured car occupants, but do not often entail deep penetration. Surface brain damage, involving the cerebral cortex, may result and can lead to epilepsy; in the large series of depressed skull fractures cited above, Jennett (1975) found an incidence of delayed epilepsy in 20% of adults and 9% of children, rising significantly when there was penetration with tearing the dura mater.

AIS NUMBER	INTERNAL ORGANS: ANATOMICAL INJURY	LEVEL OF CONSCIOUSNESS	SKELETAL INJURY
1	-	no loss of consciousness but headache and/or dizziness	-
2	injury to cranial nerve	amnesia loss of consciousness < 1 hour "concussion"	simple linear fracture
3	brain contusion	as above, with neurological deficit, or - loss of consciousness 1-6 hours	fracture skull base compound skull vault fracture
4	brain laceration surface clot < 100ml intracerebral clot	as above, with neurological deficit, or - loss of consciousness 6-24hrs. coma with appropriate movements on pain	fracture skull base + tear of dura with tissue loss massive compound skull fracture
5	brainstem contusion, large (> 100ml) surface clot, diffuse fibre injury (DAI), penetrating brain wound	as above, with neurological deficit, or - loss of consciousness > 24 hrs. deep coma with inappropriate movements	-
6	brainstem crush laceration, open wound of brainstem, massive head crush	-	-

TABLE 2.1: AIS (1985) CODING OF INJURIES TO SKULL AND BRAIN (Condensed).

2.2.2 EAR, EYE AND FACE

Car occupants often suffer injuries in this anatomical region: in the U.S. National Crash Severity Study, a third of all injuries were in the facial area (Huelke & Compton, 1983). Injury of the external ear is not common, and of no special importance. Injuries to the middle ear, inner ear, or auditory nerve are important causes of deafness; they are usually associated with closed head injuries, and may not be identified in road trauma studies. Brodie Hughes (1964) reported a 7.3% incidence of middle ear deafness and a 1.5% incidence of auditory nerve deafness in a personal series of 1800 head injuries: his series does not identify causes of injury, and may have been selected. Early recognition of middle ear deafness is important, as dislocation of the auditory ossicles may be rectified by operation.

Eye injuries are more numerous and better documented. In their large U.K. series of injured car occupants, Rutherford et al. (1985) reported eye injuries of all types in 159 drivers, 85 front seat passengers and 30 rear seat passengers: this represented 2.0% of all injuries needing admission. Of these, only 23 suffered penetrating wounds of the eyeball (AIS 2: laceration of sclera or cornea).

The pathology of penetrating eye injuries has been discussed by several writers concerned to show the risks to eyesight from shattered toughened glass windscreens (Hass and Chapman-Smith, 1976; Keightley, 1983; Blake, 1983). Blunt injuries to the eyeball and closed damage to the optic nerves are known complications of car crashes. They are especially likely to follow frontal impacts. Elisevich et al. (1984) reported 24 cases of severe visual loss associated with multiple injuries; 14 of these were due to motor vehicle crashes of unspecified type. This article gives a good account of the pathological mechanisms of visual

impairment in closed head injuries, with a review of the literature. Flaherty et al. (1983) reported three instructive cases of bilateral eye injury; all victims were drunk and none wore a seatbelt. They emphasize the crippling nature of these injuries as well as the value of microsurgical treatment (see section 6.2).

Injuries to the soft tissues of the face and to the facial skeleton are still more numerous: in the U.K. series of injuries to car occupants, Rutherford et al. (1985) reported 441 facial fractures, representing 3.1% of their large sample of U.K. injuries. Facial wounds were even more numerous. Facial injuries are not ordinarily a threat to life, except when there is associated obstruction of the airway and/or inhalation of blood; Arajarvi et al. (1986) reported 20 deaths of this type in a series of 84 road traffic crashes causing maxillofacial injuries. These were collected in Finland (pop. 4.8 million) during an eleven year nationwide study of road crash fatalities. Facial injuries are much more often the cause of disfigurement and loss of self-esteem, chronic pain, and dental disability due to loss or malocclusion of the teeth.

The pathology of facial skeletal injuries is relatively simple and is well set out in the AIS system; however this does not distinguish compound and simple (closed) fractures. The AIS recognises four grades of severity for facial injuries, and classifies the skeletal injuries along conventional clinical lines.

Fractures of the mandible (lower jaw) are often sustained by car occupants: in the large U.K. series (Rutherford et al., 1985), these fractures constituted 17.5% of all facial skeletal injuries, though very few were classed as major (AIS 3) fractures with comminution, displacement, and/or external wound. Bochlogyrus (1985) recently reviewed a German series of 1521 mandibular fractures, the causes of which in some 570 cases

"involved" automobiles (no further details given). Complications were reported in 184 (21.5%) of all cases: these included infection (6.0%), malocclusion (4.2%), and nerve injury (7.2%). The author regarded the outcome of treatment as highly satisfactory; however, the list of pathological complications shows that the injury has to be given careful attention.

Fractures of the maxilla (upper jaw) involve the middle third of the face, and such fractures were listed in 49 (11.1%) of Rutherford's U.K. series: 36 were minor (AIS 1, 2) and 13 (AIS 3, 4) major injuries. Maxillary fractures are classified according to their pathology, in three types defined by the French surgeon Le Fort (1901) on anatomical grounds. Le Fort fractures of type III extend to the skull, and can lead to serious complications, including meningitis: this fracture scores 4 in the AIS. Mid-facial fractures are often associated with visual disturbances, sometimes of serious nature; Holt et al. (1983) reported a 76% incidence of visual injury in 436 patients with mid-face fractures (all causes).

Nasal fractures are the commonest facial skeletal injury sustained by car occupants: there were 255 cases, all save two classed as minor injuries, in the U.K. series cited: they constituted 57.8% of the total number of facial skeletal injuries. Nasal fractures are usually considered to be relatively minor injuries, and readily treated by simple means; however, Illum (1986) found that after 3-4 years, as many as 16% of his patients were aware of narrowing of the nasal airway, and half of these found the sensation disagreeable; there was also cosmetic impairment in 18%.

Fractures of the zygomatic or malar bone (cheekbone) occurred in 51 (11.6%) cases in the U.K. series (Rutherford et al., 1985). These injuries were recently discussed by Ellis et al. (1985) from Glasgow. They are

serious only when they involve the orbit (eye socket), when there may be double vision or nerve damage. The orbit may also be injured in other ways: its walls may buckle, or rupture into one of the adjacent air sinuses, and double vision may be caused by this.

Injuries to the upper third of the face (forehead, eyebrows, root of nose) are not separately listed in the AIS, and may be considered either as frontal craniocerebral injuries or as orbital injuries. They are relatively less common, but can be serious, especially if there is injury to the brain or eyes. Ioannides et al. (1984) reviewed a series of 23 cases from Nijmegen, mostly due to traffic crashes. These represented 5.6% of all facial injuries. In 32%, there was some long term visual impairment, and psychiatric sequelae were also recorded.

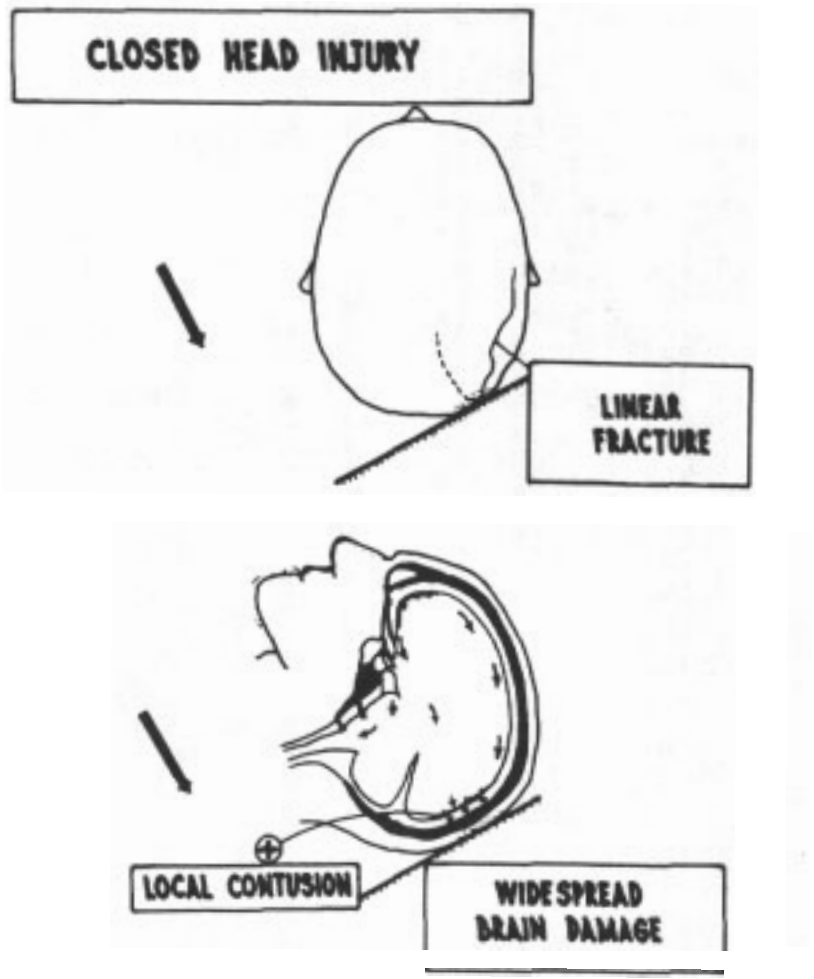
2.3 CONCLUSIONS

This review of the pathology of the head injuries sustained by car occupants has shown a surprising paucity of data relating to the pathogenesis of brain injuries. Road crash investigators have documented the severity of these injuries and have related them to crash speeds, impacting objects, etc., but have not defined the pathological diagnoses. Neuropathologists have identified some important diagnostic entities, but do not as a rule discuss the causes of injury. The series of studies commissioned by the Neurosurgical Society of Australasia (Selecki et al., 1981; Neurotrauma in Australia 1986) endeavoured to provide better data, and these studies give useful information on the mortality and morbidity of intracranial haemorrhages sustained by car drivers and passengers, but they are dependent on the accuracy of the diagnoses in a wide variety of hospitals and these are not always reliable. We believe that there is a need for more detailed studies that correlate vehicle crash studies, clinical

outcome, and neuropathological examination. We are endeavouring to carry out such studies. There is a need to define more accurately the limits of tolerance of the human brain in different types of car crash and at different ages: existing safety standards are based chiefly on simulated impacts using dummies or cadavers, and these cannot reproduce the complexity of deceleration injuries of the brain (see section 5).

By comparison, the pathology of facial and ocular injuries is more easily understood. The limits of tolerance of the facial skeleton were established some time ago (Hodgson, 1967), and the relations of injury and impact have been well documented in studies by craniofacial surgeons, oral surgeons and ophthalmologists. From our review of the literature, the most striking finding has been the high incidence of disability, usually not of crippling severity, but nevertheless of considerable personal and economic significance.

FIGURE 2.1

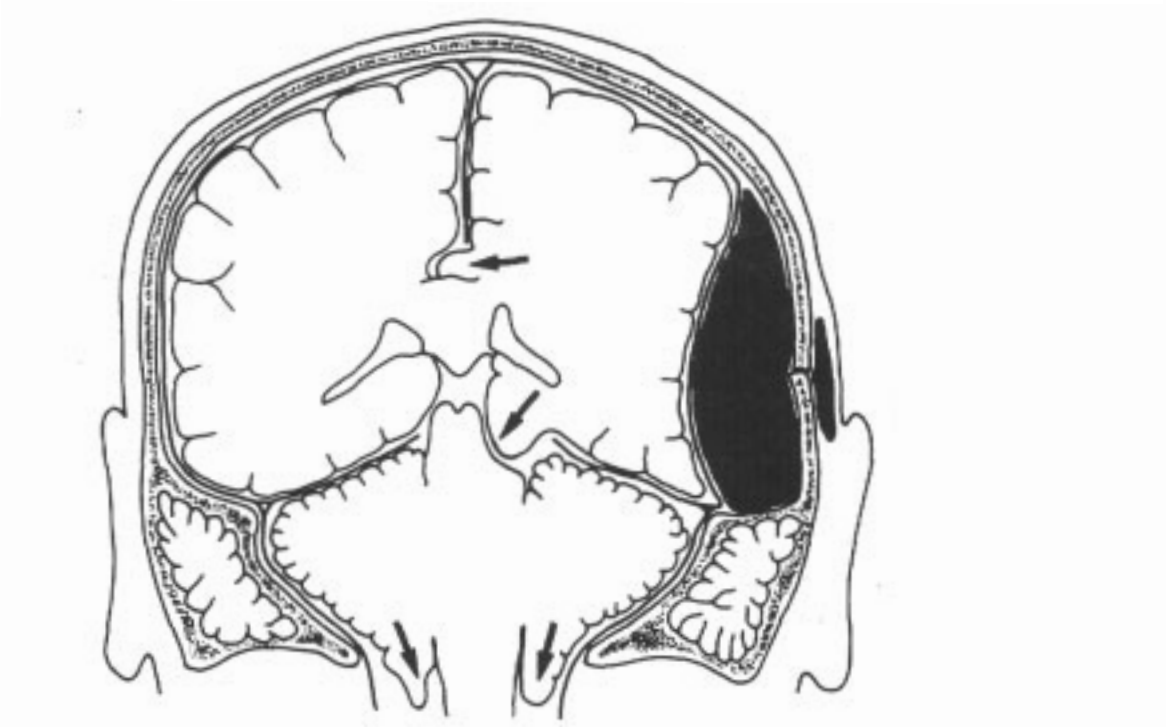


Pathology of closed head injuries.

(a) The head strikes a flat surface: the skull is deformed, and linear fracture(s) radiate from the joint of impact.

(b) Local deformation of the skull causes contusion (bruising) at the point of impact. More importantly, the brain (diagrammed in longitudinal section) is violently decelerated: linear and angular deceleration (shown by small arrows) sets up shearing stresses, which may tear nerve fibres and small blood vessels within the brain.

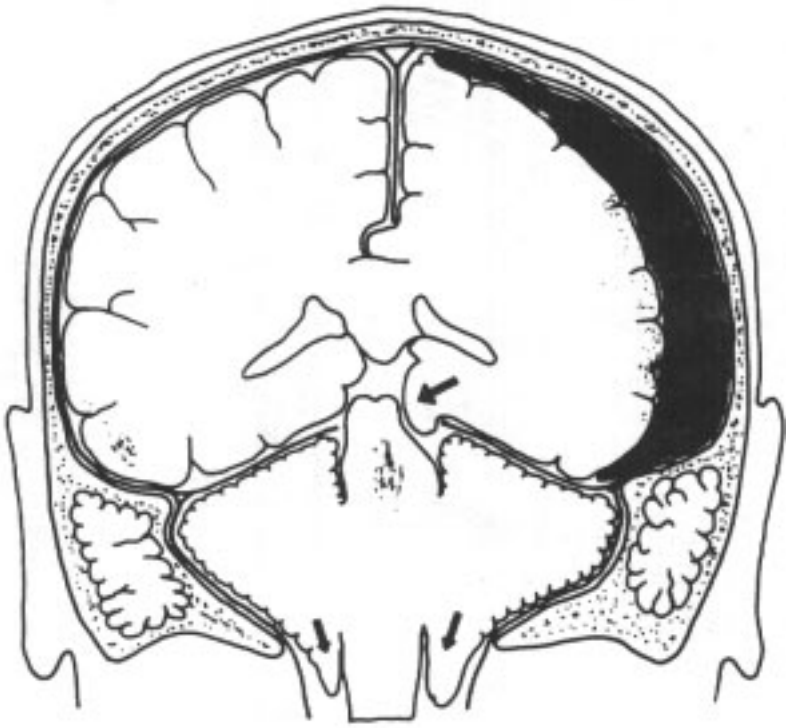
FIGURE 2.2 Surface haemorrhages causing compression of the brain, shown in vertical sections at ear level.



(a) Extradural haemorrhage: a fracture has torn a small artery (not shown), resulting in bleeding and formation of a clot (solid black) between the skull and the dura mater (black line). Brain displacements (large arrows) may result in compression of vital nerve centres.

Urgent operation is lifesaving in most instances (Bricolo and Pasut 1984).

FIGURE 2.2 (continued)



(b) Subdural haemorrhage: violence (e.g. deceleration) to the brain has torn an artery or vein, resulting in bleeding and clot formation (solid black) directly on the brain, deep to the dura mater (black line). Unlike the extradural clot, the subdural clot spreads rapidly over the surface of the brain causing severe brain compression and displacement (large arrows).

Worsening is very rapid and the mortality is high: however operation within four hours of injury saves some lives (Seelig et al 1981).

3. CHARACTERISTICS OF CERVICAL SPINAL INJURIES

3.1 INTRODUCTION

As an anatomical region the neck includes muscles concerned with the support and directional control of the head, the upper parts of the respiratory and digestive tracts, i.e. the trachea (wind pipe), larynx (voice box) pharynx (cavity behind the nose and mouth leading to the trachea and oesophagus) and oesophagus (gullet, a muscular tube connecting mouth to stomach). Also in the anterior part of the neck are thyroid and parathyroid glands, nerves, supportive tissue and major blood vessels which supply the brain and facial structures with nutrients. Towards the back of the neck are the seven cervical vertebrae which support the head and protect the spinal cord contained within the spinal canal.

All of the above mentioned structures are liable to injury in passenger vehicle crashes. Injuries to structures in the anterior portion of the neck are usually minor (AIS < 3); few are life threatening (Rutherford et al., 1985). Gikas (1983) gives examples of injury mechanisms and concludes that almost all can be eliminated through prevention of penetration of vehicle structures into the passenger compartment (e.g., windscreen or posterior edge of bonnet), and the provision of adequate restraints for the occupant in the vehicle. Injuries to these structures will not be considered further.

Injury to the cervical spinal column, its supportive ligamentous structures and contained spinal cord, is a common and too often disabling consequence of automobile accidents. Severity of injury may range from a minor strain with no long term disability to tetraplegia (paralysis of all four limbs), or even death.

The susceptibility of the neck to road trauma will be discussed further in the chapter on the biomechanics of the head and neck. It

relates to the inherent flexibility of the cervical region and lack of support unlike that provided to the thoracic region by the ribs and muscles of the chest and back, or to the lumbar region by the abdominal and the lumbar back muscles (Fife, 1987).

The role of the neck is to support the head, to provide sufficient movement to enable complete surveillance of the surrounding environment, and to protect the vulnerable structures passing from head to trunk and limbs, (i.e., spinal cord and nerve roots) and major vessels passing from heart to head. In fulfilling these roles the normal relationship between vertebrae must be maintained under physiologic loads so that neither damage nor irritation of spinal cord or nerve roots occurs. Bony, ligamentous and muscular elements combine to fulfill this role.

Bony Elements (see Fig. 1):

There are seven cervical vertebrae. The first, C1, or atlas, vertebra, articulates with the occipital condyles of the skull (Fig. 1.B). Movements of this joint are limited to flexion and extension. The seventh, (C7) articulates with the relatively fixed first thoracic vertebra, (T1). The intervening vertebrae, intervertebral discs and joints permit a normal range of movement unmatched over the same distance elsewhere in the spine.

The articulation between the first (C1) and second (C2) cervical vertebrae is unique in that there is no intervertebral disc, and it is responsible for approximately 45 per cent of all rotary movement of the head about the axis of the spine (Panjabi and White, 1978).

Ligamentous and Fibrous Elements (See Fig. 2):

Between all but the upper two vertebrae there exists an inter-vertebral disc (see Fig. 1.A). This is made up of an outer tough fibro elastic tissue called the annulus fibrosus, which surrounds an inner, gelatinous, nuclear material (nucleus pulposus) which becomes less pliable

with increasing age. The outer circular fibres around the nucleus provide considerable support for the spine at rest but are insufficient to maintain alignment when subject to moderate shearing forces (Fig. 3.B illustrates this type of force).

As well as intervertebral discs there are seven major ligaments which combine to preserve vertebral alignment at rest and in movement (see Fig. 2.D). These ligaments extend throughout the length of the spine and are reinforced in the upper cervical segments by additional ligaments (illustrated in Fig. 2.A) which maintain the relationship of the odontoid process of C2 (see Figs. 1.C and 2.B) with the anterior arch of C1. Damage to any of these structures may result in displacement of bony elements and damage to the spinal cord.

Muscular Elements:

The posterior or dorsal musculature of the neck is of considerable mass and has both the strength and stamina required to support the head throughout normal daily activities. The anterior musculature is of lesser mass and primarily concerned with directional control of head and neck movements. Both play a minor role in maintenance of vertebral alignment and prevention of spinal injury. Melvin and Weber (1985) concluded that in a completely surprise impact the time for maximal reflex muscle contraction force is of the order of 130 to 170ms, probably too long to prevent injury at speeds likely to produce significant neck injury.

Several studies however, including that by Larder (1985), have reported an increased incidence of minor neck injuries in women involved in vehicle crashes, postulated to be the result of lesser cervical muscle mass. Unfortunately, this observation failed to reach statistical significance and requires further assessment.

A large number of the articles reviewed use the Abreviated Injury Scale (AIS) to indicate severity of injury. This scale groups injuries into six grades from AIS 1 (minimal injury) to AIS 6 (usually not compatible with life). Table 1 outlines a simplified AIS scale as it applies to the cervical spine. The AIS classification does not indicate possible outcome, intensity, duration, cost of treatment required, or residual disability.

3.2 LITERATURE REVIEW

Our aim was to identify the incidence and severity of various types of cervical injury, both fatal and non-fatal, due to passenger vehicle crashes on the road. Several difficulties were encountered in reviewing the literature and are listed below.

(1) In a study of the characteristics of spinal injury one needs to consider specific clinico pathological entities (i.e., the type of injury) together with the event(s) producing them. In many otherwise informative accident reports this information is not included. For example, many reports fail to identify the cause of injury beyond listing road crashes as a single group, and many more fail to differentiate the type of injury associated with this group from others (e.g., sporting injuries, falls, etc.).

(2) The widely used AIS groups injuries with a wide range of possible outcomes. For example, an atlanto occipital dislocation without initial neurological defect is given an AIS rating of 2, the same as a fracture to a spinous process or laceration to a cervical nerve root. An atlanto-occipital dislocation, however, is unstable by definition and if incorrectly managed or missed, may result in a complete cord lesion with an AIS rating of 6.

(3) In the crash research literature the type and level of cervical injury is seldom reported and severity is only indicated by the AIS classification.

(4) Studies are often limited to either survivors or fatalities of road crashes, and comparisons between the two groups are brief, if present at all. Studies using hospital admissions and discharges will miss victims who do not present to hospital or, who are managed as outpatients. This biases results to the more severe end of the scale and distorts the overall perspective of cervical injuries.

(5) The method of data collection will affect the accuracy of information reported. For example, studies of fatal cervical injuries based on routine post-mortem examination alone, will probably miss 50 per cent of fatal cervical injuries (Alker, 1978; Bucholz, 1979). Davis et al. (1971) concluded that where violent trauma occurred death is often attributed to head injury without consideration of the possible role of cervical cord involvement.

(6) In the absence of an objective method of identification of minor soft tissue injury to the cervical spine, it is possible that the incidence of minor injury (AIS < 3) may be over-estimated.

Injuries to the cervical spine can be evaluated in several ways. Of greatest significance is the identification of those associated with damage to the spinal cord, or the potential to produce damage to the cord if managed incorrectly. Damage to the cord may result from abnormal alignment of the canal due to anterior compression fracture (a flexion compression injury). Alignment of the spinal cord may also be altered through fractures to the body, lamina, or pedicles of the vertebrae (see Fig. 2.D) or disruption of supporting ligamentous structures. Displacement of a bone fragment or disc material into the spinal canal may result in a cord

lesion. Abnormal movement of an intervertebral joint may result in irritation of the cord or of a nerve root. Bleeding, swelling or bruising within or around the cord may result in secondary cord damage through limitation of its blood supply.

The mechanism and nature of the above injuries are well reported in a number of articles (Maiman et al., 1983; Moffat et al., 1978; Hodgson and Thomas, 1980; Partnoy et al., 1979).

The characteristics of various cervical injuries are outlined below indicating their range of severity and incidence, evident from a review of the literature. Table 2 sets out associations of gross neurological patterns and broad injury mechanisms as reported by Manar (1974).

Juhl (1981) reported a series of 601 road traffic crash victims in Denmark who had reported injuries to the neck. 434 were occupants of passenger vehicles. 91.8 per cent had an injury level of AIS < 3 and 87.1 per cent were graded as having an injury of AIS 1 (i.e., acute neck strain). He also reported that these minor injuries may result in long standing symptoms and disability.

Larder (1985) reported the findings of a UK study following introduction of mandatory seat belt legislation, and with a significant increase in belt usage found a major reduction in car occupant deaths and serious injuries of around 25 per cent. A concurrent hospital based study indicated a trend towards a relative increase in the incidence of neck strains of around 18 per cent. Rutherford et al. (1985) found a statistically significiant increase in the incidence of neck injury in seat belt wearing occupants of vehicles involved in crashes since the introduction of seat belt legislation. 544 occupants of passenger vehicles involved in crashes were identified of whom 82 (15 per cent) reported a neck injury which had been recorded in medical records. There were 8 fatal

cervical injuries, but the great majority (92.7 per cent) were AIS level 1, or injuries not satisfying the minimum requirements for classification under the AIS system (i.e., acute neck strain without fracture or dislocation and neurological defect).

In 22.6 per cent pain was not evident for some time after the crash, and therefore not reported at initial post injury medical examination. 37 per cent reported persistent pain for greater than 4 months. Larder felt these injuries were genuinely reported and not exaggerated by malingering or compensation hunting individuals as the great majority of injuries reported in this study would not result in civil claims for damages.

States (1985) in reviewing Larder's paper reported that in his experience nearly half the patients with acute cervical strain do not develop pain, stiffness or headache for 24 hours, and onset may be delayed for up to 48 hours. He also felt permanent disability occurs in a significant number of patients with this type of injury and related his experience to that reported by Hohl (1974), who found 43 per cent of 146 patients with acute cervical strains sustained in vehicle crashes followed for more than 5 years had significant limitations of previously normal activity. Norris and Watt (1983) from a U.K. study reported similar morbidity.

Macnab (1964) and Gates (1966) report that cervical sprain or "whiplash" is associated with persistent morbidity of up to 74 per cent ranging from mild to severe limitation of activity and pain.

The above figures indicate the predominance of this relative minor neck injury but also its significance in terms of residual disability, pain, time lost from work, and compensation.

Injury to the spinal cord is relatively uncommon but. It may occur with or without a radiological (X-ray) abnormality. Where X-ray evidence

of injury is lacking transient subluxation (or displacement) of vertebrae may have occurred at the moment of the injury, a situation more often seen in children as reported by Burke (1974). Acute rupture of an intervertebral disc may also result in neurological defect without plain X-ray abnormality.

If a neurological defect is evident it may be either complete, i.e. without evidence of spinal cord function below the level of the injury, or incomplete, where some cord function remains below the level of the injury. It is of paramount importance to make this classification early, as, if a complete lesion is evident from the outset recovery is far less likely (Swain et al., 1985).

Both types of cord lesions are seen in victims of passenger vehicle crashes. No accident or illness, from whatever cause, is more devastating than tetraplegia (defined by Griffin, 1985, as impairment or loss of motor and/or sensory function in cervical neurologic segments due to damage of neural elements within the cord). A previously active individual, retaining a keen and alert mind, finds him or herself paralysed and dependent on others for bodily needs, which is associated often with considerable loss of self esteem. Damage extends beyond that of the injured individual, to include family, friends, the community and society in general. The economic costs are staggering (Krause, 1985). Intensive and expensive, medical, nursing and rehabilitation therapy is required over long periods. Griffin (1985) reported a median of 2 months inpatient treatment from his study with often little achieved in terms of patient independence. A small number of patients surviving high cervical cord injuries may not only be dependent on other individuals for bodily needs but also on a mechanical respirator to keep them breathing. The independence achieved will vary according to the level and completeness of the injury.

Griffin (1985) points out that where a cervical lesion has resulted in early death, the cord lesion is predominantly C1-C3. Individuals with injuries to the mid cervical segments (C4) often survive to hospitalisation but may perish from complications developing during the period of initial hospitalisation. Lesions below this level are certainly compatible with survival and a moderate degree of independence even if the lesion is complete. Selecki (1986) from his series, found 65 per cent of lesions to the cord above the level of C5 were fatal, and of those below, 30 per cent were fatal.

In Griffin's study of 154 traumatic spinal cord injuries spanning a 47 year period, 58 (37.7 per cent) died prior to hospitalisation, and in 67 per cent of these individuals, all of whom underwent autopsy examinations, the cervical injury was felt to be significant as the cause of death. 18 (11.5 per cent) died during their first period of hospitalisation.

Selecki et al. (1986) in his report of 202 cases of traumatic spinal cord injury (all levels) from several hospitals in NSW during 1977 and 1978. Thirty six per cent of the total died as a result of their injuries, 49 per cent were left with severe disability, 9% had a moderate disability and 6 per cent made a good recovery (outcome reported on the basis of the Glasgow Outcome Scale). There were 133 survivors of whom, 78 had cervical spinal injuries, with 25 being completely, and 28 incompletely, tetraplegic. This study identified 67 (51 per cent) of the 132 persons injured in motor vehicle crashes as car drivers or passengers, 25 of whom died.

Burke (1985) reported details of 352 patients with spinal injuries treated at the Austin Hospital, Victoria, from July 1978 to December 1981. 154 (52 per cent) sustained their injuries in road crashes. Only 33 per

cent of crashes occurred in capital cities where 70 per cent of the population resides, 27.5 per cent occurred in country towns, and 44.4 per cent in rural areas. 170 (52 per cent) of the spinal cord injuries were cervical. Of the 154 who were injured in road crashes, 59 were left with major disability, 26 were completely, and 33 incompletely, tetraplegic (comparable figures to those of Selecki, 1986).

Kraus (1985) reviewed US National Health Survey figures from 1977, which indicated a prevalence of complete or partial spinal cord lesions living at home as 90/100,000 population. Earlier figures from the US National Head and Spinal Cord Injury Survey of 1974 showed a prevalence of these types of cord lesions, in institutions of 13/100,000 population. These figures of course include spinal cord injuries from all causes, but as road crash injury accounts for the largest single group (Sutton, 1973, Australia: 50 per cent; Burke, 1977, Australia: 52 per cent; and Kraus et al., 1975, Northern California: 56 per cent) the prevalance of tetraplegia due to motor vehicle crashes is of the order of 50/100,000 population.

Unfortunately, few articles report details of bony and soft tissue injury. Juhl (1981) reported details of the type of cervical injury in his series of road crash victims in Denmark. 66.4 per cent had fractures of the cervical spine, 17.7 per cent an acute strain, 16.6% medullary or cord lesions, 14.3 per cent dislocations, 1.4% nerve root injuries and 0.5 per cent an intervertebral disc rupture. The level of the injury, clinical outcome or residual disability was not reported.

Bucholz (1979), reporting on a post-mortem study of 112 road crash-fatalities found 26 (23 per cent) had cervical injuries (type of road user not specified). All had a fracture, dislocation or both. Nine cases were at the atlanto-occipital joint, threewere of C1, five were injuries of the

odontoid process of C2 (see Fig. 1.C), four were fractures of the body, lamina, or pedicle of C2, there were two fractures of C3, one of C5, and two of C6.

Langwieder (1981) related neck injury to the position in the vehicle at the time of impact, and reported an incidence of neck injury in front seat occupants of between 15 per cent and 21 per cent, and of 5-7 per cent for rear seat occupants. The variation was attributed to the younger average age and shorter stature of rear seat occupants, and the relatively higher back rest. One-third of neck injuries resulted from frontal, 40 per cent from rear, and 21 per cent from lateral impacts. In all groups of impacts, 90 per cent had injuries of AIS < 3 and almost 100 per cent of those injuries from rear impacts had an AIS rating of 1. Frontal, lateral and rollover crashes were found to cause the most severe injuries.

Hodgson (1980), in reviewing mechanisms of injury, to the spine concludes that rotational and shearing forces (see Fig. 3.B) tend to produce dislocations, and compressive forces, fractures. Partnoy et al. (1979), Melvin and Webber (1985) and Panjabi and White (1978) support his views, and the concept is generally accepted. Similarly, it is accepted that injuries to the cervical spine are the result of complex processes in which the external load alone is not a good predictor of failure of bony or ligamentous structures and the resulting injury to the spinal cord. The nature of the injury will depend on the configuration of the head and neck on the trunk at the time of impact, and the direction and magnitude of both internal and external forces experienced. The outcome will depend on the level of the injury, involvement of neural structures and the completeness of any injury to the cord.

3.3 CONCLUSION

This review of the literature relating to the characteristics of spinal cord injury has been disappointing. It has been difficult to compare like to like: studies of vehicle crash injury patterns, characteristics, and mechanisms, take many different forms and information is often lacking in at least one area of interest, in particular, the pathological nature of any lesions present.

Many reports relate injuries to dummy and cadaver tests where the complexity of physiological cervical movements are not reliably reproduced.

It is obvious that more work and ongoing study is required to relate the injury and its outcome to the mechanism, with the aim of identification of measures which may reduce the incidence of these expensive and often tragic injuries.

TABLE 1: Abbreviated Injury Scale (AIS) 1985. Coding of injuries to the cervical spine and its neural contents (condensed).

AIS Number	Neural Element Injury	Spinal Injury	Clinical Defect
1.	-	Acute strain. No fracture or dislocation.	No neurological defect. Pain and discomfort only.
2.	Nerve root or brachial plexus laceration or avulsion.	-	Sensory and/or motor to part of the upper limb. Complete or partial, permanent or transcient.
		Dislocation without cord contusion or laceration. Includes atlanto occipital dislocation.	No neurological defect. May have sensation of instability. Will have pain and discomfort.
		Minor vertebral compression.	Pain without neurological defect.
3.	Cord contusion/compression	Without fracture/dislocation or with either or both.	Depending on level transcient motor/sensory signs to respiratory/cardiac arrest.
	-	Fracture to part of vertebrae.	Pain and possibly sensation of instability.
	Nerve root damage (Radiculopathy)	Disc herniation or rupture.	Pain, may be weakness or sensory loss in part of upper limbs.

TABLE 1: (cont)

4.	Incomplete cord syndrome due to compression or contusion e.g., lateral, central, anterior cord.	Without fracture/dislocation or with either or both.	May be dissociated sensory loss, hemiplegia etc., depending on site in cord of pathology.
5.	Complete cord syndromes, level C4 or below. Incomplete cord syndrome due to cord laceration	Without fracture/dislocation or with either or both. "	Quadraplegic or paraplegia with no sensation. "
6.	Complete cord syndrome level C3 or above.	Without fracture/dislocation with either or both.	Not compatible with life.

TABLE 2: Associations of gross neurological patterns and broad injury mechanisms in the cervical spine (Marar, B.C. 1974)

	Neurologic Damage	Injury
Group I	Total motor and sensory loss to all four limbs. Total transection of the cord. No recovery occurred.	Burst fracture or bilateral, facet dislocation. Flexion compression injury.
Group II	Motor loss of varying degrees, either in all four extremities or in the upper imbs only. Sometimes there was segmental or patchy transcient sensory loss associated. (Central spinal cord damage).	Hyperextension injury.
Group III	Complete motor loss in the extremities with hypoesthesia and hypologesia to the level of the lesion. No loss of position or viboratory sense. (Anterior spinal cord damage).	Vertical compression, bursting injury "tear drop" fracture dislocation possibly some associated flexion or extension.
Group IV	Motor power in all four limbs or the upper extremities alone with no sensory loss.	Unilateral facet dislocation, fractured arch of atlas and a variety of injuries.
Group V	Brown-Sequard syndrome. (Lateral spinal cord damage).	Unilateral facet dislocation or a burst fracture.

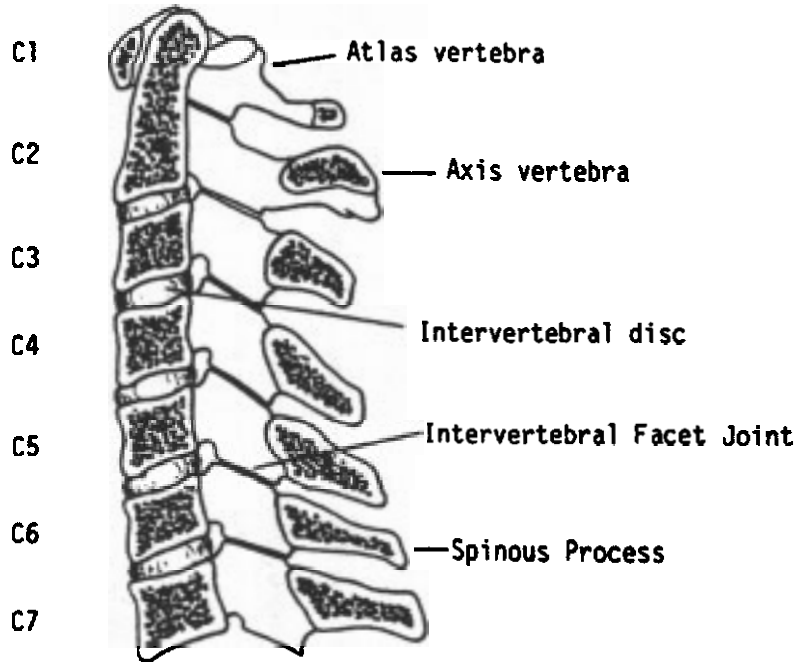


Fig 1.A: The cervical spinal column, showing the relationship of vertebrae and the intervertebral discs.

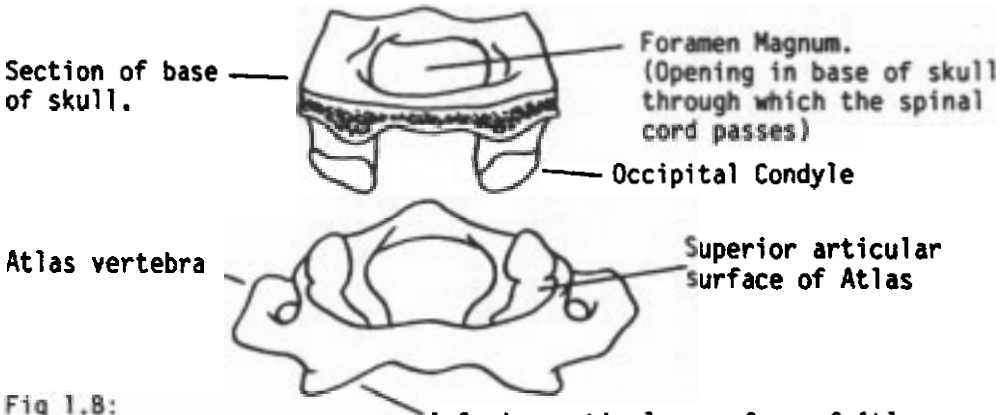


Fig 1.B:

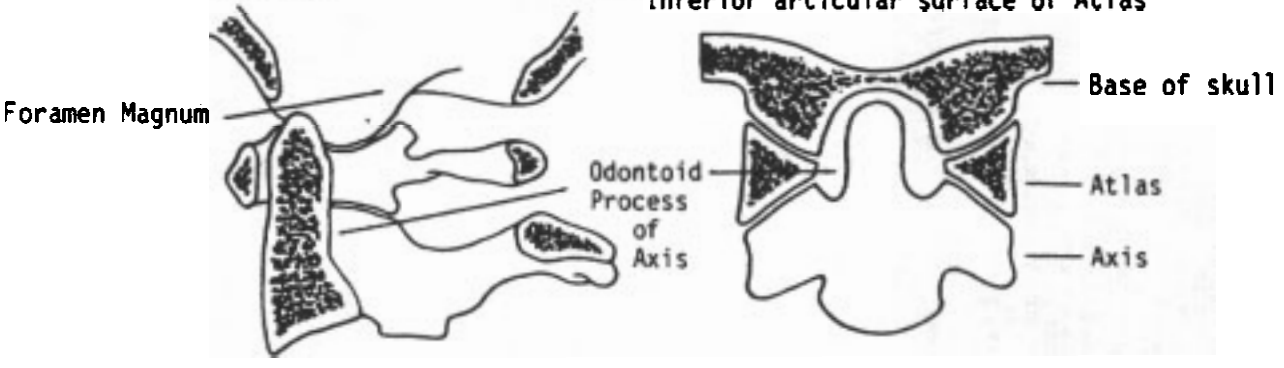


Fig 1.C:

Fig 1.B: The Atlanto Occipital articulation drawn distracted to demonstrate the articular surfaces, which allow predominantly flexion and extension movements.

Fig 1.C: Cross-section of Occipito Atlanto Axial articulation. Shows relationship of bony structures in both a frontal cross-section (side to side) and anterior-posterior cross-section (front to back)

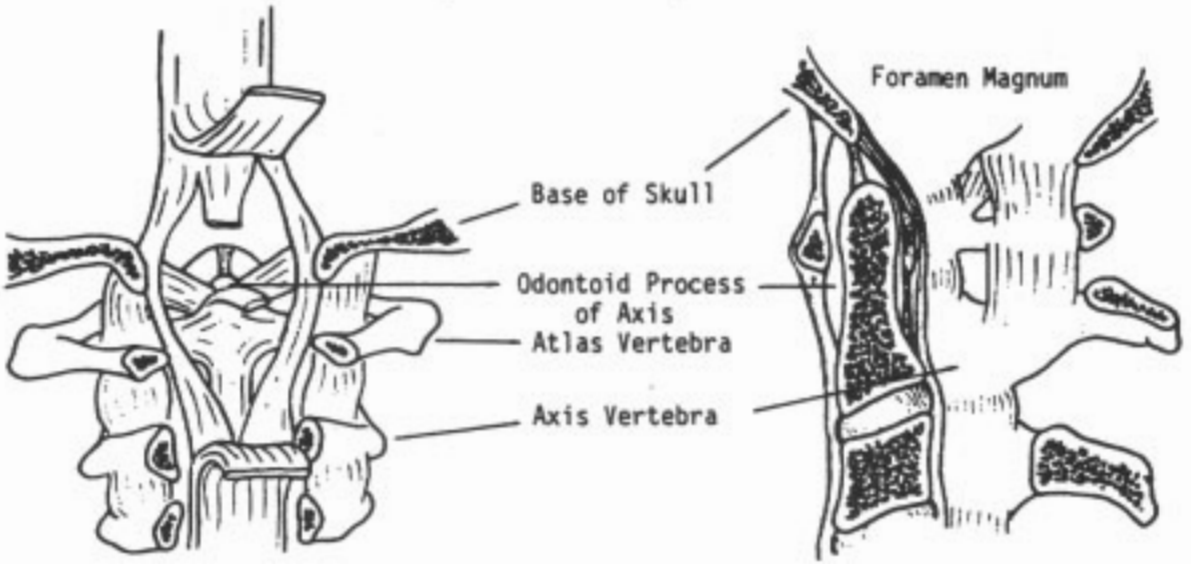


Fig. 2.A: Frontal cross-section (left) and anterior-posterior cross-section of the upper cervical spine. Shows the ligamentous supports to the bony elements shown in Fig. 1.

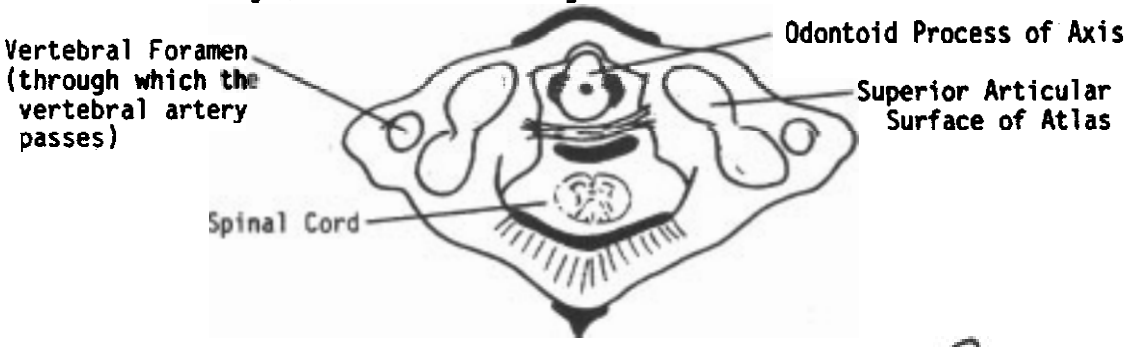


Fig. 2.B.

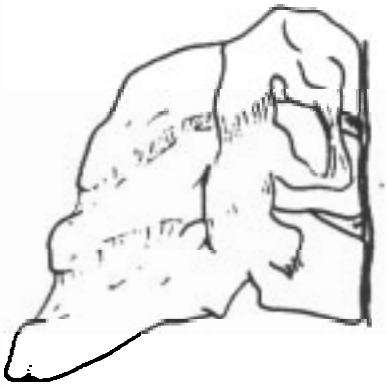


Fig. 2.C:

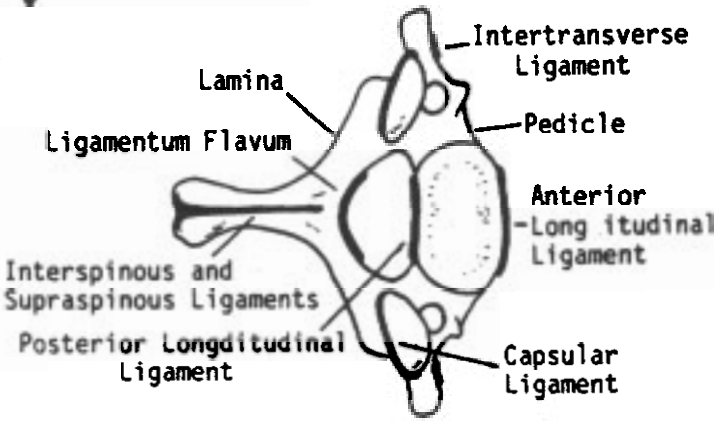
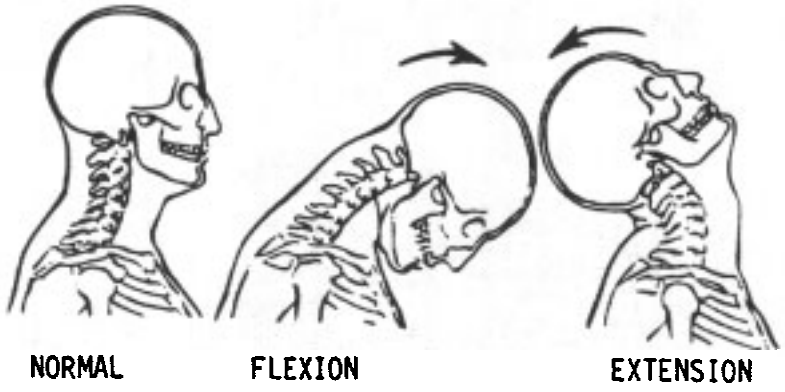


Fig. 2.D:

Fig. 2.B: The Atlas vertebra viewed from above showing the ligaments which help maintain the alignment of the upper cervical spine.

Fig. 2.C: Shows a section of the lower cervical spine from the side. Again ligamentous supports are shown.

Fig. 2.D: Shows a lower cervical vertebra viewed from above with the seven ligaments mentioned in the text identified.



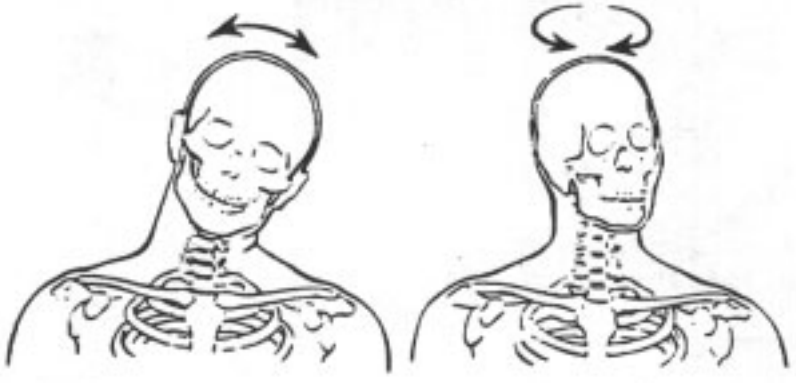
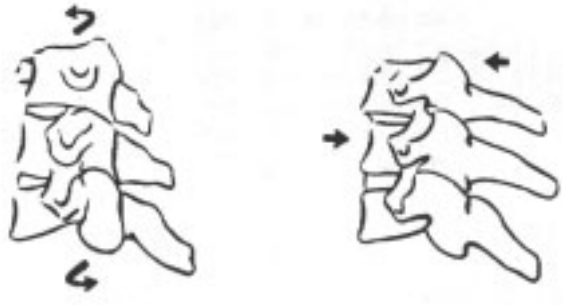


Fig. 3.A: Direction of movement of the head and neck on the torso and the terms used to describe this movement.



4. EPIDEMIOLOGY OF HEAD AND NECK INJURY

4.1 INTRODUCTION

Head and neck injuries are almost certainly the leading cause of death for the occupants of passenger cars who are fatally injured when the vehicle is involved in a crash. Selecki et al. (1981) estimated that two-thirds of all deaths to vehicle occupants in New South Wales in 1977 were due to neurotrauma. However, it is difficult to go beyond a broad general statement of this type because there is very little detailed information available on the incidence and nature of the injuries to these body regions among the occupants of passenger cars.

There is much routinely available information on deaths and injuries to drivers and passengers (e.g. Australian Bureau of Statistics, Catalogue Nos. 9405.0 and 9403.0) although it is often difficult to separate the occupants of passenger cars from the occupants of other vehicles. Nevertheless, routinely available data can yield valuable insights into the factors which correlate with injury to vehicle occupants, as illustrated by Baker et al. (1984). They present United States data on the differences in death and injury rates for passenger vehicle occupants which are associated with many factors, including age and sex, geographic, seasonal and temporal differences, type of crash, vehicle type and size, type of road and speed limit, and alcohol intoxication. Comparisons such as these tell us much about the descriptive epidemiology of injury to the occupants of motor vehicles but they give no direct indication of the frequency, type or severity of injury to the head and neck.

Unfortunately, a similar type of problem is found in studies which are primarily concerned with the characteristics of the injury. There is often very little information on the circumstances in which the injury was sustained, beyond broad categories such as "road traffic accident". This

deficiency is particularly evident in the literature on the neuropathology of head injuries (see 2.2.1).

For the above reasons the preferred criteria for the selection of the papers reviewed in this Chapter were that there be at least some information on both the body region injured and the type of road user involved. Even these criteria were found to be restrictive, however, and so reference is occasionally made to other papers to illustrate particular topics and to indicate more clearly the range of data which is available on head and neck injuries to the occupants of passenger cars.

4.2 REVIEW OF THE LITERATURE

4.2.1 Head Injury

Several attempts have been made since the early 1970's to describe the incidence of head injury (from all causes) in defined populations (Simpson et al., 1981 and Selecki et al., 1981, in Australia; and Jennett et al., 1981, in the U.K.). Frankowski et al. (1985) have reviewed the seven studies conducted up to that time on the descriptive epidemiology of head injury in defined populations in the the United States: Annegers et al. (1980), Anderson et al. (1980), Klauber et al. (1978), Cooper et al. (1983), Whitman et al. (1984), Jagger et al. (1984), and Kraus et al. (1984). Frankowski notes that it is difficult to compare the results of these seven studies because of methodological issues such as differences in the definition of what constitutes a "head injury" and in the sources and completeness of case ascertainment.

Luchter (1986) made an estimate of the current number of traffic related brain injuries in the United States based largely on the data in the reports reviewed by Frankowski. He concluded that the total number of such injuries is in the range of about 110,000 to about 300,000 per year,

with a median estimate of about 167,000 (an annual rate of 70 cases per 100,000 population). The number of moderate and severe cases is in the range of 30,000 to 118,000 with a median estimate of about 58,000, or a rate of about 25 cases per 100,000 population per year. The rate of head injury fatalities was thought to be about 10 per 100,000. These estimates refer to all traffic related brain injuries, including those sustained by the occupants of passenger cars.

The head injury death rate from traffic and transport crashes in San Diego County in 1980 was about 12 per 100,000 population (Frankowski et al., 1985). This estimate, and that by Luchter of 10 per 100,000 for the United States, can be compared with an estimate of 19 deaths per 100,000 population from head injuries sustained in road traffic crashes in New South Wales in 1977 (Selecki, 1981). Similarly, the estimate of abut 70 cases of head injury due to road crashes per 100,000 population in the United States (Luchter, 1986) is less than estimates of 137 and 179 for city and country areas in South Australia (Woodward et al., 1984). However, Frankowski's comment on the need to use an agreed set of definitions of both cases and type and severity of injury is probably even more relevant to international comparisons than to those studies conducted in the United Kingdom.

The above studies have, with some exceptions, either been based on specified populations or on road users of all types. None of the studies referred to causal factors beyond these general categories. The first large scale survey of injuries to car occupants, and the causes of those injuries, was initiated by Hugh De Haven in the Department of Public Health and Preventive Medicine of Cornell University Medical College in New York City in 1952. This research programme, commonly referred to as ACIR, was a national sample survey of injury-producing automobile crashes. The data

collection centred on State Highway Patrol officers and local medical practitioners who were trained by ACIR field staff.

One of the first publications from this project described the injuries sustained by 2,253 persons in 1,000 cars involved in injury producing crashes in selected States across the United States during the years 1952 through 1955 (Braunstein, 1957). Head injury was found to be by far the most frequent type of injury. Of all persons injured, 3.0 per cent received a head injury classified as dangerous, and 4 per cent were fatal. It was estimated that in the United States at that time, approximately 30,000 persons injured in automobile crashes required neurosurgical care and that many of these injuries occurred far from the immediate vicinity of trained neurosurgeons. It was noted that the head was injured most frequently alone but nevertheless very often in combination with other body areas. Fractures of one or more facial bones were observed in 7.2 per cent of the head injured occupants.

Kihlberg (1965) examined the ACIR data files to assess the frequency, severity and cause of injuries to the head. The data files by then comprised 71,453 occupants of crash-involved passenger cars, of whom 53,725 were injured in one way or another. The number of persons who sustained a head injury was 37,613 of which 6,847 were thrown out of the car. Of all of the fatalities in the ACIR files, 61.6 per cent were ascribed to head injury. (Kihlberg estimated that motor vehicle crashes of all types in the United States caused annually three million head injuries of which 30,000 were fatal, a rate of about 16 per 100,000 population in 1963-64). The leading causes of head injury to car occupants were the windscreen glass (19 per cent), windscreen surround (14 per cent), steering assembly (15 per cent), ejection (11 per cent), instrument panel (5 per cent), top of the passenger compartment (4 per cent), broken windows (4 per

cent), back rest of front seat (4 per cent) and the rear view mirror (3 per cent). The degree of head injury is indicated in Table 4.1, which also illustrates the positive association between the severity of head injury and ejection from the car.

TABLE 4.1: DEGREE OF HEAD INJURY

Degree of Head Injury	Per Cent of Head Injuries		
	All Occupants	Non-Ejected Occupants	Ejected Occupants
Minor	66.2	69.3	52.0
Nondangerous	24.0	24.0	23.7
Dangerous	4.9	3.6	10.7
Fatal	5.0	3.0	13.5
Total	100.0	100.00	100.0

(Kihlberg, 1965)

The ACIR data files were particularly well suited to the investigation of the multiplicity of injuries sustained by many severely injured car occupants. Using the ACIR classification of six body regions (head, neck, upper torso, lower torso, upper limbs and lower limbs), Kihlberg (1970) presented information on the pattern of injury among 57,597 injured car occupants who had sustained a total of 130,525 "injuries" (meaning injured body regions). The head was injured in 70.8 per cent of the cases (injured occupants) and the neck in 10.8 per cent. Seventy per cent of the cases were injured in at least two body regions and 38 per cent in three or more.

The relative frequency of "dangerous" or "fatal" overall injury ranged from 5.9 per cent for cases with one body region injured to 59.2 per cent for those with an injury to all six body regions. This close association between severe and fatal injury and the multiplicity of injury poses major difficulties in attempts to attribute death to any one injury for many fatally injured car occupants because there will usually be more lethal injuries than fatally injured occupants.

The multiplicity of injury has been noted by many other investigators, including Nelson (1974) in his report on the pattern of injury survey conducted by the Royal Australasian College of Surgeons. While much of the information in this report does not identify the type of road user among the 36,077 injured persons studied, there are data on the probability of severe (meaning AIS > 1) injury to the head and face for drivers and passengers. Table 4.2, which summarizes these data, is based on Tables 11 (c) and (d) from the report on the survey. It is notable that, among injured occupants, the probability of "severe" injury to the head or face was reduced for both drivers and front seat passengers by the use of seat belts. At the time of this survey (1971-1973) static three-point belts would have been fitted to almost all of these seating positions.

TABLE 4.2: PROBABILITY OF SEVERE* INJURY TO HEAD OR FACE (IF INJURED)

Seating Position	Body Region	Seat Belt	
		Worn	Not Worn
Driver	Head	5.9%	14.5%
	Face	6.1	9.2
Front passenger	Head	5.5%	11.2%
	Face	5.6	8.1

* approximately AIS > 1 (Nelson, 1974)

Nygren (1984) reported on injuries to the occupants of 339,675 private cars insured with the Folksam Insurance Group in Sweden during a five year period from 1976. There were 8,592 drivers and 5,469 passengers who were injured or killed in these vehicles. One of the aims of Nygren's study was to examine the effect of the weight of the injured occupant's car on the relative frequency of different types of injury. He was able to show that the relative frequency of "skull/brain" injury to surviving injured drivers who were wearing seat belts in large cars (46 per cent) was about half that of similar drivers in small cars (88 per cent). Small cars were defined as those weighing less than 950 kg, and large cars as those of 1,250 kg or more.

Seat belt usage was unknown in 55 per cent of the total sample of injured occupants. However the association between belt usage and the frequency of injury to the "skull/brain" is shown in Table 4.3 for surviving injured drivers and front seat passengers in medium sized cars. Elsewhere in his report Nygren notes that seat belts protected car occupants from severe "skull/brain" injuries and decreased the fatality risk. However facial injuries were relatively high among belted drivers, supposedly caused by the steering wheel.

TABLE 4.3: PERCENTAGE OF SURVIVING INJURED OCCUPANTS[1] WITH A HEAD OR FACE INJURY OF ANY SEVERITY BY BELT USAGE

Seating Position	Body Region	Seat Belt	
		Worn	Not Worn
Driver	Head	24.0%	45.2%
	Face	18.1	31.8
		1516[2]	217
Front passenger	Head	21.9%	39.2%
	Face	14.6	34.4
		776	125

1 Medium weight cars (Nygren, 1984)

2 Number of drivers/passengers

The degree of medical disability was routinely assessed about five years after the accident by the insurance company. Data on the incidence of permanent medical disability of 10 per cent or more resulting from "skull/brain" injuries among the surviving injured occupants is presented by Nygren for two twelve-month periods. These data indicate that the frequency of such a disability increased with age and was greater for males than for females, even when controlling for seating position in the car.

Rutherford et al. (1985) compared the injuries sustained by 14,019 car-occupants who presented at one of 14 selected hospitals in the United Kingdom during the year before, and the year after, the introduction of compulsory seat belt wearing legislation at the end of January 1983. They reported a 15 per cent reduction in patients brought to hospital and a 25 per cent reduction in those admitted. There were fewer injuries to the

face, eye and brain after the introduction of the legislation but the incidence of major brain injuries and some facial fractures increased among drivers, possibly due to contact between the driver's head and the steering wheel. There was little change in the number of brain injuries among fatally injured drivers in a study run concurrently with the hospital-based one. However, there was a reduction of about one-third in the number of these injuries among fatally-injured front seat passengers.

From the data presented by Rutherford et al. (1985) it is possible to calculate the number of major (AIS 3+) injuries to the head and face and to present these injuries as a percentage of all major injuries. (Table 4.4). There was an increase from 14.7 per cent before the introduction of the compulsory seat belt wearing law to 18.3 per cent afterwards.

TABLE 4.4: MAJOR INJURIES TO THE HEAD AND FACE AS A PERCENTAGE OF ALL MAJOR[1] INJURIES

Body Region Injured	Compulsory Seat Belt Wearing	
	Before	After
Head and face	14.7%	18.3%
Other body regions	85.3%	81.7%
All major injuries[2]	648 (100%)	535 (100%)

(from Rutherford et al., 1985)

[1] Major = AIS 3+.

[2] Major injuries to car occupants, all seating positions.

The National Highway Traffic Safety Administration (NHTSA) in the United States instituted a National Accident Sampling System (NASS) in an attempt to provide population-based data on both road crashes and injuries.

The development of the programme commenced in 1976, and it became fully operational at 50 sites across the USA in 1982. Based on road crashes reported to the police, NASS is intended to provide more detailed and representative data than would otherwise be available (National Center for Statistics and Analysis, 1978).

Luchter (1986) presents data extracted from the NASS files by Partyka for the years 1982 through 1984. Partyka estimated that 39, 467 or 20.0 per cent, of the annual average of 197,087 injuries rated AIS 3+ were to the head and face for car occupants. This figure is higher than the 14.7 per cent for car occupants in the United Kingdom before the introduction of the mandatory seat belt wearing law (Table 4.4). However, as Engert (1986) has noted, the national estimates based on the NASS data are imprecise. The 95 per cent confidence interval for estimated number of major injuries to the head and face would be approximately plus or minus 30 per cent.

Information on the objects struck by the head for car occupants comes from detailed studies of the crashed vehicles with prior knowledge of the nature of the injury to the head. In some instances evidence of head contact will be found in the absence of any reported head injury. There have been many studies of this type, but relatively few have been based on a representative sample of crashes (see, for example, McLean et al., 1981). However, one of the characteristics of studies conducted in this manner is that it is very difficult, for reasons of logistics and cost, to investigate a large number of cases. This means that care must be taken to note the particular circumstances of the cases of interest before extrapolating to a wider population. Nevertheless, the examination of crashed vehicles provides information on the specific causes of head injuries which can be obtained in no other way.

With the increased usage of seat belts, often as a consequence of mandatory wearing laws, attention has focused on the objects struck by the head of the belted occupant. McLean (1981) compared the experience of belted and unbelted occupants in this regard in the cases covered by the Adelaide in-depth accident study ten years ago. The reliance placed on physical evidence of belt wearing in that study meant that there was some bias towards more severe crashes among the restrained, compared to the unrestrained, occupants. A recent study in England has demonstrated the almost overwhelming importance of the steering wheel as a cause of injury to the head and face (Harms et al., 1987).

The National Accident Sampling System (NASS) in the United States is the most ambitious attempt to obtain detailed data on crashes in a statistically valid manner so that national estimates can reasonably be derived from the sample. Monk et al. (1987) identified the objects in the upper interior of the passenger compartment which, when contacted by an occupant in a crash, produced an injury to the head or face rated AIS 3 or greater. This study was based on 1981-84 data from NASS and from the earlier National Crash Severity Study. The cases selected were those for which an estimate of delta V (the change of velocity in the crash) was available. It was found that A-pillar contacts accounted for 57 per cent of the head or face injuries caused by the upper interior (which excludes the steering assembly); the sun visor/front header rail, 26 per cent; the side header rail, 13 per cent and the roof, 5 per cent. No severe head or face injuries were associated with contact with the B-pillar.

This study by Monk et al. (1987) provides an indication of the objects struck by the head and face, and hence the relative need for padding of objects in the upper interior of the passenger compartment. There are, however, three aspects of this work which should be noted. The

first is that the cases selected were unrestrained occupants. The second is that, as noted above, they were also cases for which delta V was known. This meant that the analysis eliminated crashes such as single vehicle rollovers, because the calculation of delta V is not practicable in such cases. These two aspects of the study would have an obvious effect on the relative frequency with which various parts of the upper interior were struck. The third observation is that searching the NCSS and NASS data files yielded only 66 cases which met the criteria for this study, indicating the now well-recognized deficiency in NASS in that it does not cover many severe crashes because of the representative nature of its sampling structure (this has led to plans for changes in 1988 to ensure that more cases of severe crashes are investigated).

4.2.2 Neck injury

The availability of population-based data on neck injury is such that it is extremely difficult to estimate even the incidence of fatal injury. This is partly because of a lack of adequate studies, compounded by a failure to separate injury to the neck from other spinal injury and by the difficulty involved in identifying injury to the cervical spine when there are more obvious fatal injuries. However, some estimates are available (Selecki et al., 1981; Kraus et al., 1984).

Whereas fatal injuries to the neck can be diagnosed, the far more common soft-tissue injuries, often termed "whiplash injury" generally are not amenable to objective assessment. This may be one reason why whiplash injury accounts for such a large proportion of the total cost of claims for compensation for personal injury. (The South Australian third party personal injury insurance scheme pays out about 50 million dollars a year for whiplash injury, almost half of the total cost of all claims). Consequently, studies based on insurance data may over-estimate the true magnitude of the incidence of whiplash injury.

There is some evidence, based on self-reporting of neck injury, that females are much more susceptible to whiplash injury than are males. In a study conducted in North Carolina, McLean (1973) found that female occupants were 50 per cent more likely to report a neck injury than were males in cars which had been hit from the rear. This difference existed after allowance had been made for differences in height and seating position.

4.3 CONCLUSION

While the information available on the incidence and severity of head and neck injury is sufficient to indicate that it is a major problem it is not adequate as a basis for the development of more effective countermeasures or even the evaluation of existing ones.

5 BIOMECHANICS

5.1 INTRODUCTION

Research into head and neck biomechanics is designed to examine the effect of impact on these two body regions with a view to understanding the mechanism of injury and, ultimately, to establish limits of tolerance to impact, presuming that such limits exist. Limits of tolerance can then be used in the further development of safety systems in motor vehicles and in the design of crash helmets, thus reducing the incidence of brain injury in the exposed population. Of necessity, if tolerance limits are to be implemented, there must be the successful development of an anthropometric test device (ATD) which models not only the anthropometry of the human frame, but also its response to impact. Two pre-requisites must therefore be accepted as achievable in impact biomechanics research: that tolerance limits exist and can be ascertained; and that ATDs can be improved to the point where they accurately represent the kinematics and biodynamic response of humans.

Although crash victim simulation (CVS) computer programs may be useful in research into tolerance limits because full scale reconstruction is not, and should not, be acceptable with living human subjects, the actual safety device must be tested under crash circumstances and therefore with an ATD. Research into impact tolerance and ATD development must therefore proceed concurrently.

The following discussion of head and neck impact biomechanics examines the history of research in this area and, in particular, kinematic parameters suggested as possible indicators of brain injury and neck injury. This discussion concentrates on the development of, not only, tolerance limits for impact, but also on the development of anthropometric devices for the measurement of such limits.

5.2 HEAD INJURY BIOMECHANICS

The basic premise behind all research into the biomechanics of head impact is that brain injury is related to the kinematics of the head and therefore kinematic parameters describing the head movement at impact can be used to define tolerance limits of brain injury. Goldsmith (1966) provided a detailed theoretical analysis of the physical processes occurring as a result of a head impact. According to Goldsmith, when two bodies collide, two distinct effects are produced in each body: the propagation of stress waves through each body; and disturbances at or near the point of contact which he termed "contact phenomena". In his review of previous attempts at describing brain injury mechanisms he cited a number of different mechanisms: vibration of the entire skull; localised large deformations or distortions of the skull; brain displacement and/or separation at the point opposite to impact; establishment of large pressure gradients, including negative amplitudes; propagation of steep-fronted waves in the cranium; rotation of the cerebral mass; and neurovascular friction.

A number of these are basically "inertial effects", mechanisms where the brain is said to lag behind the motion of the skull and thus producing injury due to shearing or tearing of brain tissue. Others are primarily concerned with explaining the contre-coup injuries and therefore are based on theories of wave propagation and cavitation, ignoring rotational effects.

It is interesting that two schools of thought can be seen in the above discussion - those who believe in what are essentially linear effects (injury due to skull deformation at impact and cavitation) and those who believe in rotational effects (shearing and tearing of brain tissue) as being largely responsible for injury. It would seem likely that both

effects are important, yet the history of head injury biomechanics reveals periods where either the linear or rotational view was dominant. The development of the Head Injury Criterion (HIC), enshrined in U.S. Government regulations for automotive testing, occurred during a period when linear effects were believed to be the major cause of brain injury. The recent history of head injury research now shows a resurgence in the rotationalist view.

5.2.1 Translational models

The development of the so-called "Wayne State Curve" in 1960 set translational acceleration as the main kinematic parameter for describing head injury. Basically the curve was derived from data obtained in tests on embalmed cadavers, animals and human volunteers, in which the acceleration and time pulse were plotted. Essentially, the relationship suggested that to produce injury required short pulses of high acceleration or long pulses of low acceleration. As pointed out by Hess et al. (1980), this was the first graphical representation of a critical injury threshold based on impact conditions. From this curve the Gadd Severity Index evolved (Gadd, 1966) and eventually the Head Injury Criterion, HIC, (Versace, 1971). The Head Injury Criterion is not, however, a good tool for assessing injurious levels of impact to the head. In fact, it seems to bear little relationship to the severity of head injury (Patrick et al. 1974, Cesari et al. 1975, Cesari et al. 1979). This is hardly surprising, since the Wayne State data were collected from test subjects that were not representative of the population at risk; the acceleration, in the case of the cadaver tests, was poorly measured; and some of the data were either incorrectly plotted or not plotted at all (Newman, 1980).

Hess et al. (1980), in their historical review of the Head Injury Criterion, concluded that although it had its weaknesses, it was unlikely

to be replaced in the near future by another injury criterion. Newman (1980) simply declared that HIC was invalid and the implication from this is, of course, that any research into injury prevention based on HIC may also be invalid. Lockett (1985) takes exception to Newman's view of HIC and, by mathematical analysis of a deformable object under time-dependent loading, he derived a criterion which was similar in form to HIC. He concluded from this analysis that HIC was, therefore, a plausible approximation to a fundamentally correct criterion. This, however, does not negate Newman's arguments against HIC. A criterion for translational acceleration may exist and be in a form similar to HIC, as Lockett suggests, but knowing this does not increase the worth of HIC. Ultimately the value of HIC must be assessed on its ability to discern injury-producing from non-injury-producing head impact. Clearly HIC cannot do this. Grosch (1985) finds HIC incapable of distinguishing between hard contact (A-pillar, steering wheel), soft contact (airbag) and non-contact. This is due to the variable time interval used to calculate HIC. Grosch suggests using the time period during which the acceleration is above 60g (a tolerance level originally suggested by the originators of the Wayne State Curve). With this modification, HIC deals more appropriately with both hard and soft contacts.

The manner in which the Wayne State Curve developed into the Head Injury Criterion is another example of what Mackay (1984) described as the 'extraordinarily cavalier' manner in which crash protection measures have been introduced. Mackay was referring to the introduction of protective devices such as head restraints, energy-absorbing steering columns etc., and the failure of authorities to evaluate their performance and, if necessary, improve their design. This argument holds equally well for the development and acceptance of the Head Injury Criterion. HIC derived from

an experiment that was not well-controlled and yet it has dominated research into head protection.

It is appropriate, before commencing a discussion of rotational injury mechanisms, to discuss work carried out by Muccardi et al. (1977), who investigated both translational and rotational kinematic parameters. In a series of tests conducted with 26 monkeys the authors measured a number of kinematic waveforms from which 34 kinematic parameters were calculated. These parameters were then used in an Adaptive Learning Network to model 3 brain injury outcomes: overall AIS (Abbreviated Injury Scale), unconsciousness AIS and the duration of unconsciousness. It appears from this study that the overall AIS was associated with translational velocity and acceleration; unconsciousness AIS with the early occurrence of maximum angular acceleration in the presence of high translational acceleration; and time of unconsciousness appeared to increase with increasing components of angular acceleration relative to translational acceleration. Although this study was only based on 26 monkey experiments it is, nevertheless, illuminating and suggests that by examining a large number of kinematic parameters a greater understanding of the injury process can be achieved.

5.2.2 Rotational motion

Holbourn (1943) was a very early proponent of rotational motion as the chief cause of brain injury. He concluded that there were two main mechanisms for brain injury: deformation of the skull with or without brain injury; and sudden rotation of the head, producing contre-coup injuries, intracranial haemorrhages and probably concussion.

Holbourn noted that the assumption of a mechanics of head injury implied that head injury could be determined by the physical properties of the skull and brain, and Newton's Laws. Holbourn concluded that brain injury was due to shear-strains produced in the brain. Given this,

deformation of the skull would produce localised shear-strains and, hence, localised injury, whereas a blow to the head would produce linear and rotational velocity changes in the brain. It was Holbourn's view that the rotational component produced the shear-strains and hence the brain injury. He did not accept that compression and rarefaction, the product of translational acceleration, were significant injury-producing mechanisms because of the brain's virtual incompressibility to hydrostatic loading.

Dissatisfaction with injury criteria based solely on translational head motion led to a resurgence of research work into injury causation via rotational head motions. Hirsch and Ommaya (1970) reported a series of tests conducted on Rhesus monkeys. The researchers found that the placing of a cervical collar around the neck of the animal prior to testing increased the tolerance of the animal to head impulse loading. The cervical collar had the effect of reducing the rotational response of the animal's head, but not the translational response. The authors concluded that the increased tolerance of the monkeys to impulsive loading of the head was due to the inhibition of rotational motion.

Further work on cerebral concussion and rotational acceleration was conducted by Ommaya and Hirsch (1971). A criterion for concussion was based on the following:

1) loss of coordinate response to external stimuli;
2) apnoea greater than 3 sec.;
3) brachycardia (rate decreased by 20-30 beats/sec);
4) loss of corneal and palpebral reflexes;
5) loss of voluntary movement;
6) pupillary dilation greater than 15 sec.

They conducted a series of tests on monkeys in which rotational acceleration was generated by direct impact to the occipital bone or by

impact to the base of the chair carrying the animal. Using scaling techniques, a tolerance limit of 1,800 rad/sec/sec was suggested for concussion in humans.

Gennarelli et al. (1972) further examined the differences between translational and rotational head motions and, in particular, the differences in brain injury produced by pure translational motion of the head and that which was largely rotational. Twenty-five squirrel monkeys were used, twelve of which were subjected to peak head acceleration levels of 665-1230g and the remaining thirteen primarily to rotational motion, the peak tangential acceleration levels being in the range of 348 to 1025g. Contact phenomena were minimised by the design of the apparatus producing the head acceleration. None of the animals receiving the primarily translational impulse were concussed, whereas all of the thirteen animals receiving the rotational acceleration were. The latter group of animals also showed a high incidence of subdural haematoma, subarachnoid haemorrhage and intracerebral petechial haemorrhage.

Ommaya and Gennarelli (1974), on the basis of their experimental work, developed the hypothesis that clinically observed damage to the brain would always be found at the surfaces of the brain in mild cases of concussion and would extend inwards as the severity of trauma increased. Pathological data did not exist which adequately described injury to the brain at the three levels of severity: reversible deficit, irreversible deficit with survival, and irreversible deficit plus death. Although data could not be found to support the hypothesis, what data did exist did not refute the hypothesis.

Rotational acceleration was further investigated as a mechanism for the production of acute subdural haematoma (ASDH) by Gennarelli and Thibault (1982). ASDH are most commonly produced by tearing of the bridging

veins which travel from the brain's surface to the various dural sinuses. Using an apparatus similar to that used previously in the investigation of cerebral concussion, the animal's head was rotated through an angle of 60° in times varying from 5 to 25 milliseconds. Because the animal's head was encased inside a helmet, contact phenomena were minimized. The results indicated that the bridging veins were particularly sensitive to the rate of onset of acceleration. This would explain why ASDH are more often seen in falls rather than in automobile accidents. The former usually result in head impact onto a hard surface (e.g. the ground) producing a sudden deceleration of the head at impact, whereas in the latter the head impact may often be against surfaces of the automobile which are comparatively soft (e.g., dashboard, steering wheel), thus producing a less rapid onset of head deceleration.

Newman (1986) investigated brain injury in two cadaver tests with reference to his generalised acceleration model for brain injury threshold (GAMBIT). One of the cadavers sustained a brain injury whereas the other had no brain injury. HIC did not discriminate between the two head impacts sustained by the cadavers and was calculated as 1,073 and 1,063 for the brain-injured and non-brain-injured cadavers respectively. GAMBIT, on the other hand, indicated for the brain-injured cadaver higher levels of rotational acceleration in the presence of high translational acceleration than were seen in the non-brain-injured cadaver.

Impacts of the facial skeleton are not generally considered to be as serious as impacts directly to the skull. An impact to the chin, however, can produce head rotation and, in certain circumstances, a fracture of the base of the skull (Tarriere et al. 1976). This type of injury is important because it is fatal and not uncommon. Mergnargues et al. (1975) reported five cases of circular based skull fracture where this type of impact could be implicated.

5.3 NECK INJURY BIOMECHANICS

Huelke and Nusholtz (1986) provided an extensive review of the literature on cervical spine injury biomechanics. From this review a number of observations can be made. Many spinal injury mechanisms are hypothesised from clinical observation of the injury itself, rather than from any experimental investigation. Experimental work that has been done has been largely in the form of static loading of the cervical spine or its components and this does not provide any information on the tolerance of the cervical spine to dynamic loading, nor do they necessarily reflect the type of loading which occurs to an individual during impact.

White and Panjabi (1978b) reviewed a number of specific cervical spine injuries describing the nature of each injury, and the proposed or accepted mechanism of injury. The injury mechanism is described using what the authors refer to as the major injury vector (MIV) which is supposed to represent the most dominant force and/or moment operating at the vertebrae and responsible for causing the injury. The MIV is described relative to a coordinate system outlined in White et al. (1975). This coordinate system differs from that normally used in that the y-axis rather than the z-axis is the vertical axis. Not all the cervical spine injuries reviewed by White and Panjabi (1978b) are traumatic in nature. Those that are include:

Atlanto-occipital dislocation. An often-fatal injury, the mechanism of which is described as a large magnitude force acting in the +z direction, causing a shear force at the atlanto-occipital joint, rupturing the articular capsule and detaching the head from the spinal column.

Fractures of the posterior arch of C1. A fracture which occurs behind the lateral masses of the atlas where the ring is grooved by the vertebral artery. This is a weak point in the ring and the MIV is

considered to be in the -y direction with some extension producing a force of vertical compression on the posterior arch of the atlas.

Comminuted fracture of the ring of C1. This fracture is also called a Jefferson fracture after its discoverer, an English neurosurgeon. The MIV is similar to that previously discussed for the simple fracture of C1, but in this case the magnitude of the force would be greater and the direction would be aligned through the centre of the ring producing a force which acts through the occipital condyles and tends to burst the ring apart.

Fracture of the dens or odontoid process. This can be a fracture in the odontoid process, in the junction of the odontoid and the body of C2, or at the base of the odontoid in the body of C2. The MIV is considered to be a force acting in the -z direction (anterior to posterior) possibly during hyperextension and acting via the anterior ring of C1. It could also occur in hyperflexion (+z) where the force would be applied to the odontoid by the transverse ligament.

Atlanto-axial dislocations and subluxation. Either an anterior or posterior displacement of C1 on C2 or, possibly, rotary subluxation of C1 on C2. The MIV is considered to be similar to that for the dens fracture but is of sufficient magnitude to produce a dislocation. For the rotary subluxation the MIV is considered to be a torque produced by impact to the head such that the force of impact is directed through the centre of mass of the head.

Traumatic spondylolisthesis of the axis. This type of fracture is generally known as a Hangman's Fracture. It is a fracture of C2 resulting in the separation of the anterior and posterior elements of the vertebra. Fracture of the spinous process of C3 may also occur. A

large extension force causes a bending moment at the odontoid resulting in rotation in the sagittal plane. This bending moment is opposed by ligamentous forces in the anterior portion of C2, and by compressive forces generated between the facet joints of C2 and C3. The net result of this interaction of forces is the production of maximum bending in a region of C2 where the cross-section of the bone is smallest.

Cervical compression fracture. This denotes a large group of fractures of cervical vertebrae ranging from simple compression fractures through to comminuted or "tear drop" fracture dislocations. The MIV is exerted downwards (-y direction) and primarily in the region of the anterior elements. The extent of fracture depends on the magnitude of the force and the physical properties of anatomic structures adjacent to the vertebra.

Unilateral facet dislocation. This type of injury involves abnormal displacement of the articular facets on one side of the involved vertebra. Normal movement at the facets of adjacent vertebrae allows axial rotation and lateral bending but under conditions of trauma, when these normal motions may be exaggerated, dislocation of one facet can occur.

Bilateral facet dislocation. Unlike the previous injury, this type involves dislocation of both facets. The MIV is presumed to be a flexion bending moment in the sagittal plane with very little axial rotation, lateral bending or compression present.

Whiplash. Generally considered to be a hyperextension injury, it is by far the most common cervical injury in motor vehicle crash. Macnab (1964) considered hypertension to be much more likely to produce soft tissue damage than flexion or lateral bending. In a series of

tests performed by Macnab, monkeys were placed on horizontal platforms so that the head and neck protruded over the edge. The platforms were then dropped from a variety of heights and hyperextension was produced when the platform was brought to an abrupt halt. A number of soft tissue injuries were noted ranging in severity from minor tears of the sternocleidomastoid to partial avulsion of the longus colli. Tears in the longus colli were associated with retropharyngeal haematoma and, invariably, with damage to the cervical sympathetic nerves.

The use of the MIV to describe the mechanism of neck injury is of limited use since it gives no indication of the magnitude of force required to produce injury. Although it is more descriptive than the overused terms 'hyperextension' and 'hyperflexion', White and Panjabi (1978b) provide little information as to how the MIV is determined, nor does Ommaya (1984). Although it is an improvement on the method described by Roaf (1972), it would appear that it is still derived from clinical investigation of the injuries rather than from experimental data. Patrick (1970) conducted a series of tests on volunteers and cadavers. The volunteers were subjected to static loading and dynamic loading below injurious levels. The cadavers were subjected to dynamic loads similar to those experienced by the volunteers, and to dynamic loading in excess of these levels. The author used a severity index defined as the ratio of the dynamic reaction to the maximum voluntary static reaction. The severity index had been suggested earlier by Mertz & Patrick (1967). A severity index was calculated for neck torque, neck shear force and neck axial force. The index for neck torque was larger than that for either axial force or shear force and Patrick concluded that neck torque was the limiting injury factor. Subsequent sled tests on human volunteers and cadavers were carried out by Mertz and

Patrick (1971). From these tests the authors were able to calculate non-injurious neck response corridors for the neck in flexion and extension. Based on these data, critical limits of 57 Nm in extension, and 190 Nm in flexion, were suggested as values below which no ligamentous damage would occur. Goldsmith and Ommaya (1984), in their discussion of neck injury tolerance limits, also described the loading of the neck produced by head impact. The loading was said to produce an axial force along the spinal column, a shear force perpendicular to the cervical column, and a torque about the occipital condyles. The tolerance limits referred to by Goldsmith and Ommaya came from the study conducted by Mertz and Patrick (1971).

Dynamic testing has been carried out by Nusholtz et al. (1981) using 12 cadavers subjected to an impact to the top of the head. Further tests conducted on 8 cadavers, also impacted on the top of the head, were reported by Nusholtz et al. (1983). The purpose of these tests was to investigate fracture or fracture-dislocation injuries which are normally attributed to extension/compression or flexion/compression mechanisms. These tests found that the orientation of the head, cervical spine and thoracic spine were critical factors in influencing the injury outcome of the cervical spine. The inadequacy of the terms 'hyperextension' and 'hyperflexion' in describing cervical spine injury mechanisms, was also highlighted. Essentially so-called 'flexion' injury was found when extension had occurred and vice versa. It also appeared that most cervical injury occurred during compression and not during the resultant extension or flexion movement.

5.4 ANTHROPOMETRIC TEST DEVICES

A plethora of anthropometric test devices (ARDs) or, more simply, test dummies, have been created since the first generation of dummies was

developed by the U.S. Air Force to test pilot ejection seats. The Alderson ATD (Naab, 1966), the Sierra ATD (Mate and Popp, 1970), "Repeatable Pete" (McElhaney et al, 1973), the TRRL side impact ATD (Harris, 1976) and the Hybrid I, Hybrid II, ATD 502 and Hybrid III ATD (Foster et al. 1977) are just a few that have been developed since the mid-sixties. Each design has brought improvements in geometry and kinematics, yet it remains a fact that ATDs do not behave like human beings and they do not respond to impact in the same way as human beings. How well ATDs model human behavior under impact cannot be estimated accurately because of the lack of good data on the dynamic response of humans.

The performance of the head-neck structure, in particular its response to acceleration which produces flexion or extension, has been questioned for some time. Mertz et al. (1973) conducted tests on a number of ATDs where the moment and angular displacement of the head in extension and flexion was determined and then compared to the response corridors developed by Mertz and Patrick (1971). None of the ATDs, which were all commercially available or commonly used, provided neck responses which fitted the response corridors. The authors concluded that additional effort was required in order to produce a neck which performed adequately. Realistic simulation of the neck response was vitally important if the dynamic response of the head to impact was to be modelled accurately.

Wismans and Spenny (1983) used data from dynamic lateral flexion tests to produce a performance requirement for mechanical necks. These tests were conducted on volunteers by the Naval Biodynamics Laboratory. The authors concluded that a two pivot system with three degrees of freedom was sufficient to model the human neck in dynamic lateral flexion. The torque-rotation characteristics for such a two pivot system were given. The authors repeated this study for the head and neck in flexion (Wismans and

Spenny, 1984) and found that the two pivot system with two degrees of freedom modelled the neck response.

The location of the upper pivot was described as being "near the occipital condyles" because the authors could not estimate the precise location on the volunteers being used. It is debatable, however, whether or not the occipital condyles are in fact the best location for modelling head-neck rotation. While it may be the anatomical centre of rotation, it is not, as Frish et al. (1976) point out, the mathematical centre. The mathematical centre of rotation of the head-neck is above the occipital condyles, as noted by White and Panjabi (1978a).

5.5 DISCUSSION

It would appear from this review of the literature that there is general agreement concerning the three principal mechanisms of brain injury: brain injury which is localised at the point of head impact and produced by mechanisms referred to as contact phenomena; brain injury, such as contre-coup contusion, produced by translational motion of the head; and brain injuries such as diffuse axonal injury and acute subdural haematoma, produced by rotational motion of the head.

Although there has been much work carried out in the field of head impact biomechanics, meaningful tolerance criteria have proved elusive. The Head Injury Criterion (HIC), the only tolerance criterion currently specified in vehicle safety standards, does not correlate well with injury severity. Given that it derives from a very limited set of data, this is not surprising. HIC is also based on only one kinetic parameter, translational acceleration. The work of Gennarelli and others on the effect of rotational acceleration on brain injury has been far more methodical and successful than any of the work which went into the

development of HIC, yet it has not led to the development or use of a tolerance criterion for rotational acceleration in any safety regulation.

Any future work in the development of brain injury tolerance criteria needs to examine the extent to which all three principal injury mechanisms act, and interact, in the production of neurotrauma.

With regard to neck injury, the tolerance corridors developed by Mertz and Patrick for the neck in flexion and extension, have been influential in the design of mechanical necks for test dummies. These tolerance criteria were established for whiplash injury and are based on a small number of volunteer and cadaver tests. They are only valid, if they are valid at all, when there is no head impact. It would appear that the large number of more serious cervical spine injuries (dislocations and fractures) are produced by the combination of axial forces, shear forces and torques acting on the cervical spine and occurring at levels in excess of those produced in a simple whiplash event. These more serious cervical injuries are obviously influenced by the relative orientation and motion of head, neck and thoracic spine prior to head impact, and also by the characteristics of the head impact itself because it will produce sudden changes in the axial forces, shear forces and torques acting on the cervical spine. Head impact is, therefore, an important factor in spinal injury biomechanics.

Work on head injury biomechanics should not then proceed in isolation; the development of tolerance criteria for head impact should take into account those factors which are known to produce serious spinal injury.