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Head and neck impact, burn and noise injury criteria — A Guide for CEN helmet standards committees

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National foreword

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Head and neck impact, burn and noise injury criteria - A Guide for CEN helmet standards committees

Critères relatifs au traumatisme cervico-facial et aux lésions dues aux brûlures et au bruit - Guide destiné aux comités des normes sur les casques de protection du CEN

Kriterien für Verletzungen durch Einwirkung auf Kopf und Hals, Verbrennungen und Lärmverletzungen - Leitfaden für Arbeitsgruppen, die europäische Helmnormen erarbeiten

This Technical Report was approved by CEN on 27 December 2010. It has been drawn up by the Technical Committee CEN/TC 158.

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Foreword

This document (CEN/TR 16148:2011) has been prepared by Technical Committee CEN/TC 158 “Head protection”, the secretariat of which is held by BSI.

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Introduction

Members of helmet Standards committees frequently need to define limits for test procedures. Such limits relate to test values that indicate the potential for injury and yet it is often difficult for members to know the type and severity of injury that is represented by a given test value. Over the years, criteria have been developed for different body regions and usually these have been derived from a combination of accident and casualty data, and tests on cadavers, cadaver body parts, animals and human volunteers. However, such criteria are often used by the automotive industry as pass/fail values without a clear understanding of human tolerance to injurious forces. This sometimes leads to the mistaken belief that any value below the stated limit implies uninjured and all values above imply a serious or fatal injury.

This misconception gives very little freedom to choose values that are different from the often inappropriate automotive value. This is particularly true for head injury criteria for which values for a helmeted head may be different to those for the unhelmeted head. Many accidents to wearers of helmets, which cover a wide range of activities from horse riding to downhill skiing, result in a closed head injury. This is when the brain is damaged without any skull or external tissue damage. Conversely, head injuries in automotive accidents are much more frequently open head injuries with skull fracture and soft tissue lesions.

Other misconceptions arise because of the failure to understand that human response to a given dose or injurious parameter varies across a range of the population. The dose response curve tends to be "S" (sigmoid) shaped such that as the magnitude of the injurious parameter increases so does the percent of the population that sustains an injury of a given severity. Thus, a family of "S" curves can be generated for a range of injury severity such as AIS and a measurement or criterion such as HIC, the Head Injury Criterion. Unfortunately, the data for such an analysis is generally difficult to obtain because measurements generated by test apparatus do not relate directly to injury severity because a headform for example does not respond in an impact like a human head. Hence, it is necessary to find a relationship between these test measurements and injury severity.

This paper is designed to provide information to convenors that will help in choosing test limits in relation to a particular injury type and severity. It is worth noting that accident investigators use a scale known as the Abbreviated Injury Scale, AIS (AAAM). This was developed (in the USA) so that injury severity could be recorded in databases regardless of the body region and type of injury thus avoiding lengthy medical terms that were unfamiliar and difficult to interpret. This paper begins by reviewing the AIS scale and its application to head and neck injuries and burn injuries. Thereafter, each measurement type is reviewed and the severity of injury for given values is identified where possible. A section on burn injuries and fatigue related to heat exposure has been included to assist with Standards for equipment to protect firefighters. The Appendix describes the skin structure and the category and consequence of burn injuries.

Premature deafness because of high noise levels and the converse problem of over attenuation of auditory warnings was also considered. Suggested levels have been included with details of test methods in Annex A.

1 Abbreviated injury scale, AIS

This is a scale that extends from 0 to 6 where 0 is uninjured and 6 is unsurviveable. Each level can be applied to any body region according to a coding manual developed by the Association for the Advancement of Automotive Medicine (AAAM). Tables 1 and 2 give the scale and injury severity and an indication of the head and neck injuries that would be classified at each level. Table 3 gives similar information for burn injuries by degree, surface area and region of the body.

Table 1 — AIS scale with head injury severity

	AIS 0 uninjured	AIS 1 minor	AIS 2 slight	AIS 3 moderate	AIS 4 serious	AIS 5 severe	AIS 6 unsurviveable
Scalp							
superficial abrasions, contusions, lacerations		X					
major laceration or minor blood loss			X				
blood loss >20% or total scalp loss				X			
Intracranial vessels (arteries)							
laceration					X	X	
Cranial nerves							
contusion, laceration, loss of function			X				
Brain							
swelling, contusions, haemorrhage				X			
haematoma, large >15cc contusion					X		
massive >30cc contusions, diffuse axonal injury, large haematoma						X	
crush, penetrating injury							X
Loss of consciousness							
< 1 hour			X				
1 - 6 hours or < 1 hour with neurological deficit				X			
6 – 24 hours, or 1-6 hours with neurological deficit					X		
> 24 hours, or 6-24 hours with neurological deficit						X	
Skull Fracture							
simple			X				
compound				X			
complex, open, loss of brain tissue					X		

Table 2 — AIS scale with neck injury severity

	AIS 0 uninjured	AIS 1 minor	AIS 2 slight	AIS 3 moderate	AIS 4 serious	AIS 5 severe	AIS 6 unsurviveable
Whole area							
Skin							
superficial abrasions, contusions, lacerations		X					
major laceration or minor blood loss			X				
blood loss >20%				X			
Decapitation							X
Vessels (arteries)							
carotid, jugular and vertebral laceration minor			X				
carotid jugular and vertebral laceration major				X			
Nerves							
vagus injury		X					
phrenic injury			X				
Spine							
hyoid fracture			X				
cord contusion				X			
incomplete cord syndrome					X		
complete cord syndrome or laceration C-4 or below						X	
complete cord syndrome or laceration C-3 or above							X
disc injury without nerve root damage			X				
disc injury with nerve root damage				X			

Table 3 — AIS scale with burn injury severity

	AIS 0 uninjured	AIS 1 minor	AIS2 slight	AIS 3 moderate	AIS 4 serious	AIS 5 severe	AIS 6 unsurviveable
1st degree							
unspecified		X					
2nd degree							
< 10% TBS (Total Body Surface)		X					
3rd degree							
< 10% TBS			X				
< 10% TBS with face, hand or genitalia involvement				X			
2nd or 3rd degree							
10% to 19% TBS			X				
10% to 19% TBS with face, hand or genitalia involvement				X			
20% to 29% TBS				X			
20% to 29% TBS with face, hand or genitalia involvement					X		
30% to 39% TBS					X		
30% to 39% TBS with face, hand or genitalia involvement						X	
40% to 89% TBS						X	
≥ 90%							X
TBS = Total Body Surface							

2 Peak linear acceleration (A.3.1 & A.4)

This is the most frequently used parameter in helmet testing and is derived usually from a tri-axial accelerometer mounted in the headform unless the headform is rigidly supported and then the source is a single axis accelerometer. In both types, the helmet is mounted onto the headform and then the apparatus allowed to fall unimpeded onto a rigid anvil.

Table 4 is a scale published by Newman (1980) and is supported by research that is more recent.

Table 4 — Peak acceleration and typical AIS Equivalent

Peak Acceleration	AIS
< 50 g	AIS 0
50 g – 100 g	AIS 1
100 g – 150 g	AIS 2
150 g – 200 g	AIS 3
200 g – 250 g	AIS 4
250 g – 300 g	AIS 5
> 300 g	AIS 6

Although not specifically stated in the original research paper it should be considered that the above values represent 50th percentile, which means that 50 percent of the population would sustain an injury of a given AIS severity for the corresponding range of acceleration. It is interesting to note that historically, values have been set which correspond to AIS 5 and that this has resulted in helmets that have given reasonable protection.

In some standards, the helmet is mounted onto a fixed headform and then a mass is dropped onto the helmet. Values given in Table 4 may be used with caution provided the falling mass is approximately 5 kg and the headform is attached to an appropriate neck. Replacing the fixed headform test by a falling headform, guided or free-fall, should be considered.

3 Head injury criterion HIC (A.4)

Annex A gives details of the derivation of HIC and the formula is given below.

$$HIC = \left[\left(\frac{1}{t_2 - t_1} \cdot \int_{t_1}^{t_2} a_{res} dt \right)^{2,5} \cdot (t_2 - t_1) \right]_{\max}$$

The benefit of HIC over peak linear acceleration is that HIC is related to time and it is known that pulses with the same peak value but different duration can give a different injury outcome. Unfortunately, HIC and AIS values have never been satisfactorily correlated. Although, recent research (COST 327) has provided tentative values for AIS 2 and AIS 3, see below. Nevertheless, researchers have provided an assessment of the probability of death for HIC ranges. A summary of the various findings is given in Table 5.

Table 5 — Probability of death for HIC ranges

HIC	Probability of death	AIS (where known)
1000	10 % - 15 %	2
1500	35 %	3
2000	35 % - 50 %	
3000	55 %	
4000	60 % - 65 %	

It should be noted that where a range is given, this is indicative of more than one source. It should also be noted that HIC is derived from the GSI (Gadd Severity Index) (see A.4.2) used in some Standards. GSI and HIC are potentially interchangeable but only for regular pulse shapes. Therefore, it is recommended that GSI be replaced by HIC.

4 Rotational motion (A.2.6, A.3.2 & A.4.3)

4.1 Peak Rotational Acceleration

This is a parameter that is known to contribute substantially to brain injury but the relationship with injury is difficult to quantify. It is also a parameter that is considered by test authorities to be difficult to measure because it requires a nine-accelerometer array in the headform and complex interpretation. Nevertheless, the research shows, Table 6, that concussion AIS 1-2 can occur at 5 000 rad/sec^2 and fatal injury AIS 5-6 can potentially occur at 10 000 rad/sec^2. This correlates with data that indicates that there is a 35 % risk of a brain injury of AIS 3 - 6 at 10 000 rad/sec^2.

Table 6 — Probability of brain injury

Peak rotational acceleration	AIS	Probability of injury
5 000 rad/s^2	1 - 2	
10 000 rad/s^2	3 - 6	35 %

4.2 Tangential force at the helmet surface

This parameter is measured in motorcycle helmet Standards BS 6658 and Regulation.22-05. It is not directly related to rotational acceleration at the centre of a headform but is a function of helmet geometry. Thus, the following information in Table 7 which was obtained from motorcycle accident reconstruction data needs to be interpreted with care.

Table 7 — Injury related to peak tangential force on a motorcycle helmet

Peak tangential force	AIS	Probability of injury
1 000 N	1	-
2 000 N	2	-
3 500 N (Reg 22-05)	3	< 50 %
4 000 N	3	50 %
7 000 N	4	-

5 Skull crushing and penetration force (A.2.2 & A.3.3)

5.1 Crushing force

Resistance to crushing and a means of measuring the crush force transmitted to the skull are frequently discussed in helmet Standards committees. Below in Table 8 is a dynamic force that is typically required to fracture the facial bones and the skull. The information is from different sources hence the range and suggested tolerance values.

Table 8 — Typical fracture forces

Bone	Fracture Force Range (N)	Mean Tolerance Value (N)
Zygoma	614 - 3 470	1 000
Zygomatic arch	925 - 2 110	1 500
Maxilla	623 - 1 980	900
Mandible centre	1 890 - 4 110	3 000
Mandible lateral	818 - 3 405	1 900
Mandible midbody	1 290 - 1 445	NA
Skull tempero parietal (rigid impactor) dynamic tests	4 670 - 14 950	8 500
Skull tempero parietal (rigid impactor) static tests	3 430 - 7 830	5 070
Frontal static rigid	3 910 - 11 790	6 360
Frontal dynamic rigid	5 420 - 7 870	6 400
Frontal dynamic padded	5 340 - 15 100	11 260

The above results show that the force required to crush a human skull varies. However, analysis indicated that when the skull was crushed between two rigid plates the value was typically 5 000 N, and 10 000 N when the plates were lined with energy absorbing material such as is used for helmet liners. Hence a limit of 10 000 N is suggested for a helmet type fitted with an energy absorbing liner and 5 000 N for a helmet type that comprises a hard shell without an energy absorbing liner.

5.2 Penetration force

Assessment of resistance to penetration is included in a number of Standards. The following values are based upon an impactor with a flat circular plate 25,4 mm diameter. This may not be representative of a typical object causing a penetration injury but it is helpful information.

Skull region	Fracture force range (N)	Mean tolerance value (N)
Tempero parietal	2 500 – 10 000	5 200

6 Neck injury

Neck injury potential is assessed against shear, tension and extension (and sometimes compression) measured using transducers in a dummy neck. Cumulative plots of these values against time are required because injury potential is also a function of time. The following values (except compression) represent a

greater than 50 % probability of a neck injury of AIS ≥ 3 (Hobbs *et al* 1999). Compression is based upon values given by Yamada (1970).

Shear	3,1 kN	1,5 kN for 25 ms to 35 ms	1,1kN for 45 ms or greater
Tension	3,3 kN	2,9 kN for 35 ms to 59 ms	1,1 kN for 60 ms or greater
Extension	57 Nm		
Compression	3,7 kN		

7 Noise (Appendix section A5.0)

Details of potential test methods to evaluate helmet noise attenuation to prevent premature deafness without over attenuation of auditory warning signals are given in Annex A, A 5.

For the purposes of the European Directive 2003/10/EC the exposure limit values and exposure action values in respect of daily noise exposure levels and peak sound pressures are fixed at:

a) exposure limit values: $L_{EX.8heq}$, = 87dB(A) and P_{peak} = 200 Pa;

b) upper exposure action values: $L_{EX.8heq}$, = 85dB(A) and P_{peak} = 140 Pa;

c) lower exposure action values: $L_{EX.8heq}$, = 80dB(A) and P_{peak} = 112 Pa.

Table 9 gives values for maximum exposure times as recommended by the UK Royal Aerospace Establishment.

Table 9 — Maximum exposure time for different noise levels

Noise level dB(A)	Maximum continuous exposure period (hours)
95	3
96	2
99	1
103	0,33
109	0,08 (5 min)
113	0,033 (2 min)
140	0

8 Heat: burns and fatigue (A.6)

8.1 Burns

This section was included for the benefit of those defining Standards for firefighting equipment although it may be used for other applications. Studies have recommended that the range of environment in which firefighters operate is categorised by three levels as follows:

— Level 1: Routine conditions - air temperatures up to approximately 100 °C and a radiant heat source of up to approximately 1,25 kW/m^2.

— Level 2: Hazardous conditions - air temperatures up to approximately 250 °C and a radiant heat source of up to approximately 8 kW/m^2.

— Level 3: Emergency conditions - air temperatures up to and above 800 °C and radiant heat sources from 80 kW/m^2 up.

Table 10 gives the burn injury degree for a range of temperature and exposure time for air and surface contact, and water. The table is compiled from a range of sources with full details in A.6.4. Table 11 gives the burn injury degree for thermal energy and time, and radiation.

Table 10 — Burn injury degree for a range of temperature and exposure time for air and surface contact, and water

Temp.°C	Time s – second m - minute	Contact substance					
		Air or surface contact			Water		
		Burn injury degree			Burn injury degree		
		1st	2nd	3rd	1st	2nd	3rd
100	15 s		X				X
82	30 s		X				X
71	60 s		X				X
68	1 s	NK			X		
64	2 s	NK			X		
60	5 s	X1					X
56	15 s	NK			X		
53	1 m	NK			X		
51	3 m	NK			X		
49	5 m	X					X
38	NS	safe			safe		
<0	NS				≥ X		

NS = not specified; NK = not known; 1= time not specified for air/surface contact

Table 11 — Burn injury degree for thermal energy and time, and radiation

Thermal Energy/unit area cal/cm2 (kJ/m^2) (average for a range of subjects)	Time sec	Radiation kW/m2 (average for a range of subjects)	Injury
3,2 (7,6)	0,5	3,8	1st Degree burn
3,13 (7,5)	0,5	4,0	2nd Degree burn
4,15 (9,9)	0,5	4,5	3rd Degree burn

8.2 Heat fatigue

Equipment to protect from heat may be available but the greater the temperature against which it is designed to protect the more of an encumbrance it becomes and the greater the fatigue when it is worn. This dichotomy

needs to be considered when writing Standards for such equipment, for firefighters, for example, and the following information is presented to help with this.

Table 12 — Maximum exposure for unimpaired mental performance

Temp. (°C)	Max. exposure for unimpaired mental performance.min. (hr)
40	23
34	60 (1)
32	120 (2)
31	180 (3)
<30	Not limited

The information given in Table 12 is taken from graph given in A.6.5. It is intended as a guide for health and safety at work but is given here to assist with the specification of protective equipment. The graph appears to be exponential hence, the rapid decrease in the maximum exposure for a given temperature increment.

9 References

The Abreviated Injury Scale (AIS). 1990 Revision; *Association for the Advancement of Automotive Medecine (AAAM)*, 2340 Des Plaines River Road Suite 106, Des Plaines, Illinois 60018, USA

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Hobbs, C.A. *et al* (1999). *European New Car Assessment Programme (Euro NCAP). Assessment Protocol and Biomechanical Limits (Version 2)*

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Annex A

Biomechanics of head injury from impact, noise and heat

A.1 General

In accidents, the human head is exposed to loads greatly exceeding the capacity of its natural protection. This explains why, despite the extensive research on head injury during the past 50 years and the continuous improvement of head protection devices, head injury is still by far the most common cause of fatal injury in accidents. The consequences of severe head injuries are often fatal or long lasting and not fully recoverable.

Head injuries can be divided into two categories: primary injuries, which are a direct consequence of the physical loading of the head and appear at the time of the accident and secondary injuries, which are directly related to the severity of the primary injuries and can appear up to several days after the accident. Primary injuries can result in several physiological changes, such as necrosis, post-traumatic oedema, increased intracranial pressure, hypoxia, ischemia, intracranial hypertension or other vascular changes. Secondary injuries are directly related to primary injuries, therefore, decreasing the severity of the primary injuries will automatically decrease the severity of the subsequent secondary injuries.

A clear understanding of the types of head injury occurring in various types of accident and the injury mechanisms causing these injuries is important to improvements in protective devices. Extensive medical research has led to substantial information on the characteristics of head injuries, the locations in the head and the likelihood of occurrence in the various types of accidents. The characteristics of the most common head injuries occurring in accidents is indicated in Clause 3.

Mechanisms causing head injury are still not clearly understood. Traditionally, head injuries have been related to impacts and accelerations of the head and research has concentrated upon the effects of these two types of loading. Originally, impact and acceleration were studied for their ability to cause only a few particular head injuries. However, in most accidents, impact and acceleration are inseparable and a wide range of head injuries occurs. It is shown in this report that the resulting kinematics of the head and the behaviour of the brain inside the head, rather than accelerations, should be considered as the causes of head injury.

Nevertheless, it is acceleration that is usually measured in helmet impact tests. Thus, the committee members responsible for establishing the requirements for Standards need to be aware of the relationship between acceleration and the implied kinematics of the brain and skull and, in turn, the potential for injury.

Although the causes of head injury are not fully understood, various head injury criteria have been proposed through the years and some of these criteria (Head Injury Criterion, HIC; Gadd Severity Index, GSI) are used in Standards for head protective devices. Such criteria are reviewed and discussed.

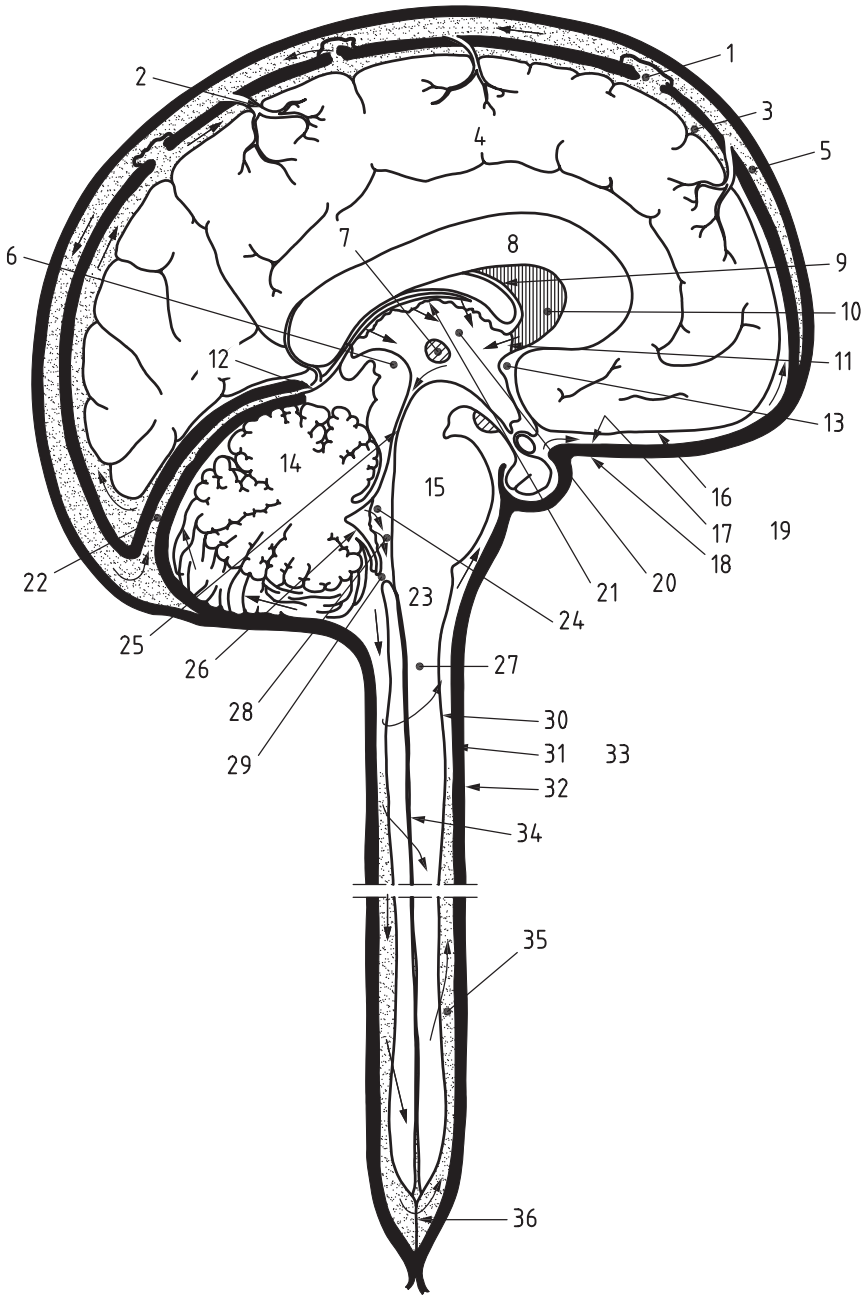
Premature deafness because of high noise levels can also be classed as an injury. This and the and the converse problem of over attenuation of auditory warnings was also considered. Suggested levels have been included with details of test methods.

A section on burn injuries and fatigue related to heat exposure has also been included to assist with writing Standards for equipment to protect firefighters. The skin structure and the category and consequence of burn injuries is described and discussed as is the exposure related to temperature, time and radiation to cause a burn of a given degree.

A.2 Head injuries

A.2.1 Head Anatomy

The term head injury comprises various kinds of trauma to the skull and its contents. Often, several different types of head injury occur simultaneously in an accident. The anatomical location of the lesions and their severity determine the physiological consequences. In this section, a review of the information found in the literature on the most commonly reported head injuries in accidents is given. The injuries are divided into cranial injuries, skull fractures, and intracranial injuries. The intracranial injuries are further subdivided into injuries to vascular and neurological tissues. This review is intended to provide the reader with sufficient information on the characteristics of these injuries, their common locations of occurrence in the head, and their relative importance in terms of severity of outcome and frequency of occurrence. This will give the reader the required background information for the discussion of the injury mechanisms in Clause 3. Figure A.1 shows the main anatomical structures of the head and their locations inside the head.



Key

1	Arachnoid villus	2	Superior cerebral vein	3	Subarachnoid space of brain
4	Cerebrum	5	Superior sagittal sinus	6	Posterior commissure
7	Intermediate mass	8	Corpus callosum	9	Choroid plexus of lateral ventricle
10	Lateral ventricle	11	Interventricular foramen	12	Great cerebral vein
13	Anterior commissure	14	Cerebellum	15	Pons
16	Pia mater	17	Arachnoid	18	Dura mater
19	Cranial meninges	20	Third ventricle	21	Choroid plexus of third ventricle
22	Straight sinus	23	Medulla oblongata	24	Lateral aperture (of Luschka)
25	Cerebral aqueduct	26	Choroid plexus of fourth ventricle	27	Spinal cord
28	Fourth ventricle	29	Median aperture (of Magendie)	30	Pia mater
31	Arachnoid	32	Dura mater	33	Spinal meninges
34	Central canal	35	Subarachnoid space of spinal cord	36	Filum terminale

Figure A.1 — Main anatomical structures of the head and the locations inside the head

A.2.2 Skull Fracture

Skull fracture can occur with or without brain damage, but is in itself not necessarily an important cause of neurological injury (Gennarelli 1985; Prasad *et al*. 1985). Skull fracture can be either open or closed. A closed fracture is a break in the bone, but with no break in the overlying skin. An open fracture, on the other hand, is a contiguous break in both the skin and underlying bone and is more serious than a closed fracture because of the accompanying risk for infections. Usually, skull fractures are subdivided according to their location of occurrence. A distinction is thus made between fractures in the face, in the vault of the skull and in the base of the skull. Even though fractures to the face are very painful and inconvenient for the patient, they do not constitute a threat to life nor cause serious neurological damage. Fractures to the vault can cause meningeal and cortical injury when fragments of fractured bone enter the cranial cavity. However, the most threatening form of skull fracture is a basilar or basal skull fracture. This part of the skull contains passages for the blood vessels, providing the blood supply to the entire brain, and to passages for the neurological connections between the brain and the rest of the body. Fractures of the skull base around the cavities where the blood vessels and nerves pass can lead to damage to these vital connections.

Within the above-mentioned anatomical areas of the skull, skull fractures are usually further subdivided into linear, depressed and comminuted fractures (Thomas *et al*. 1973). In a linear skull fracture, skull penetration does not occur and the contact effects are confined to the contact area. The skull is only cracked in linear skull fractures. Usually, the crack has the form of a single line running from the area of impact and may involve either the inner or outer table or both (Douglass *et al*. 1968). A variant of the linear fracture is the stellate fracture, which is a group of star shaped cracks radiating from the central impact point. The dural arteries lie close to the inner skull table and are, therefore, sensitive to skull deformation. Linear fractures perpendicular to the path of a dural artery may rupture the artery and cause an extradural haematoma (see section Intracranial Haematoma), which can compress the underlying brain (Douglass *et al*. 1968). Thomas *et al*. (1973) stated that approximately 80 % of skull fractures are linear. According to Bakay and Glasauer (1980), about 50 % of the linear fractures occur in the mid portion of the skull and extend toward the base of the middle fossa. The remaining half of the linear skull fractures are equally divided between the frontal and occipital regions

Miller and Jennett (1968) defined depressed skull fracture as a depression of a bone fragment of at least the thickness of the skull. Depressed skull fractures are similar to linear skull fractures, only the impact surface is smaller. This causes concentrated contact effects, resulting in skull penetration. The skull fragments that are driven into the cranial cavity can lead to trauma to the underlying brain and blood vessels (Figure A.2). According to Bakay and Glasauer (1980), half of the depressed skull fractures occur in the frontal area of the skull and the remainder are divided between the parietal and posterior regions. About half of the depressed fractures are associated with dural lacerations, often without clinical evidence of injury to the underlying brain (Thomas *et al*. 1973). In comminuted skull fractures, the fractured part of the skull is broken into more than two pieces. An example of a depressed comminuted fracture is given in Figure A.3.

Thomas *et al*. (1973) noted that approximately 50 % of all skull fractures occur in the mid portion of the skull and extend toward the middle fossa. In skull base fractures the middle fossa is most frequently involved, followed by the anterior fossa and the posterior fossa (Luna *et al*. 1981). Simpson *et al.* (1989) found numerous instances of basilar fractures in vehicular accident victims in Australia. In these cases, transverse fractures of the middle fossa were most frequent and Simpson *et al* (1989) and Luna *et al* (1981) attributed these injuries to facial impacts. A characteristic of motorcycle accident victims is that fractures of the vault are rare among helmeted riders, but that basilar skull fractures are frequently encountered, both in helmeted and unhelmeted riders (Hurt *et al.* 1986; Thom and Hurt 1993).

Luna *et al.* (1981) studied motorcycle accidents and found that skull fractures are rarely restricted to one area of the skull. Usually, the fractures are multiple, often including the skull base. In skull base fractures the middle fossa is most frequently involved, followed by the anterior fossa and the posterior fossa.

Two common types of basilar fracture are the hinge fracture and the ring fracture. The hinge fracture is a bilateral fracture of the middle fossa. After this fracture occurs, the anterior and posterior portions of the skull are free to hinge about the fracture line (Huelke *et al.* 1988; Thom and Hurt 1993). Ring fractures completely encircle the foramen magnum. Got *et al.* (1983) found ring fractures to be associated with laceration of the brain stem. Smith and Dehner (1969) observed in their studies of military motorcycle fatalities that fractures of the posterior fossa would typically curve around the foramen magnum.

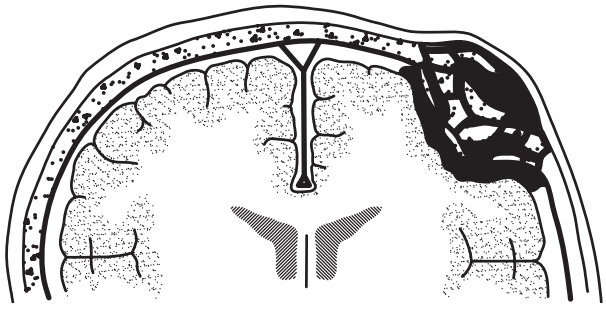


Figure A.2 — Depressed skull fracture of the vault, with dural laceration and cortical contusion (Bakay and Glasauer 1980)

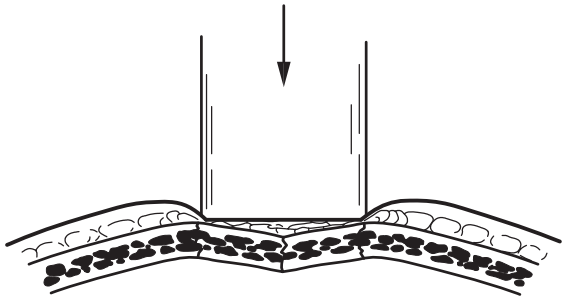


Figure A.3 — Comminuted depressed skull fracture (Melvin and Evans 1971)

A.2.3 Cerebral Contusion

Cerebral contusions are bruises of the brain caused by haemorrhages of small blood vessels. Contusions are the most frequently found type of brain injury (Prasad *et al.* 1985), crest of gyri being common places for them to occur. In these cases, the contusions are wedge-shaped with the apex extending into the white matter (Lindenberg and Freytag 1960). Contusions are most often multiple and are frequently associated with other lesions such as cerebral haemorrhage, subdural haematoma and extradural haematoma (Prasad *et al.* 1985). Clinical findings indicate that most cerebral contusions occur at the frontal and temporal lobes (Gurdjian 1966), regardless of whether the patients had experienced frontal or occipital impacts (Adams *et al.* 1982a). This suggests that the inner geometry of the skull may contribute to contusions.

It has long been considered that contusions and head injury are synonymous. Yet, observations in patients and in experimental subhuman primates have shown that severe and fatal damage to the brain resulting from head injury can be sustained without visible contusions. In addition, patients usually make a good recovery, despite having sustained severe cerebral contusions (Adams *et al.* 1982a).

Contusions have been studied extensively by various pathologists. Many pathologists subdivide contusions in coup contusions, occurring at the point of impact, and contrecoup contusion, occurring at remote sites from the impact (Cooper 1982; Prasad *et al.* 1985). Some use an even more extensive subdivision, in which contusions are classified as coup, contrecoup, intermediate, and so-called 'gliding' contusions (Lindenberg and Freytag 1957, 1960; Voigt and Löwenhielm 1974; Adams *et al.* 1982a; Gennarelli *et al.* 1982b). In this classification, coup contusions are defined as contusions occurring at the point of impact, while contrecoup contusions occur contra-lateral to the site of impact. The intermediate contusions are vascular disruptions on brain surfaces that are not adjacent to the skull, while the gliding contusions occur along the superior margin of the cerebral hemispheres.

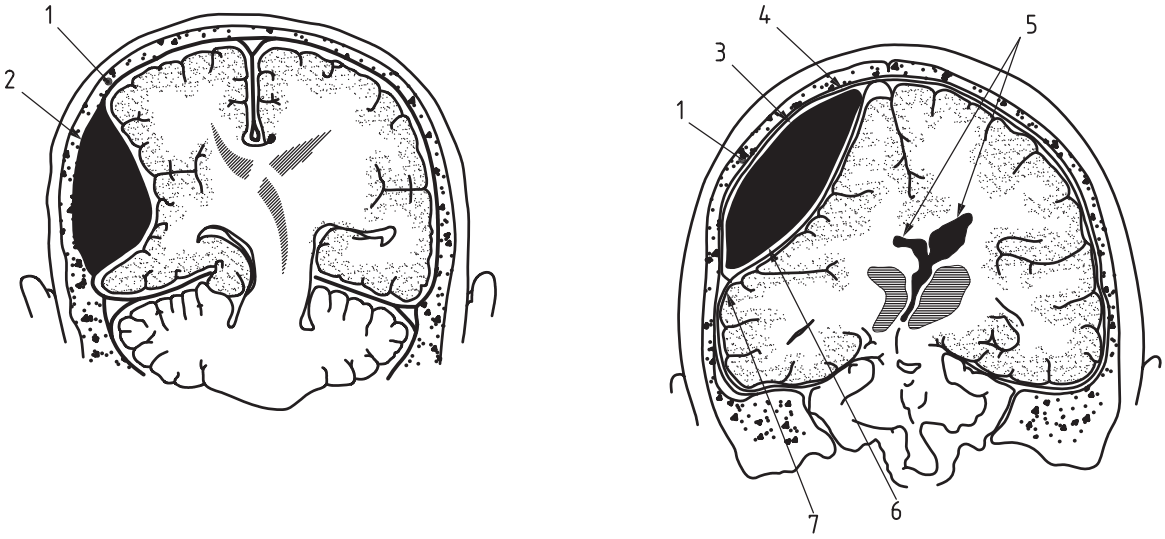
The above classifications for contusions may give the impression that the different types of contusion are caused by different injury mechanisms. However, the only difference between the different contusions in these classifications is their location inside the head. As will be shown later, the different types of contusion are all caused by contact of the brain with more rigid intracranial surfaces (skull, meninges). This single mechanism can cause contusions anywhere inside the skull at an interface between the brain and these rigid surfaces.

Therefore, the different contusion classifications bring more confusion than clarification to the issue of brain injury and are better avoided.

A.2.4 Intracranial Haematoma

An intracranial haematoma is an accumulation of blood inside the head caused by a haemorrhage. The colour of the haematoma indicates the kind of blood vessel that has been ruptured: arterial blood is bright red and venous blood is dark red. The type of blood vessel that is damaged will determine the size of the haematoma and its effect on the surrounding tissue. Arterial blood is under high pressure, causing blood to emerge in spurts when the artery is damaged. Since the blood pressure in veins is much lower, blood flows steadily out of a damaged vein at lower pressure. Damage to minor vessels produces only an oozing of blood. Medical observations show that at the first instances of a contusion the bruises appear dark in colour, indicating venous rupture. Although the bleeding inside the head may not be the main cause of injury, as a result of the bleeding the haematoma may expand causing compression and shifting of the brain and an increase in intracranial pressure. This expansion of the haematoma determines the amount and severity of the resulting neurological injury. The effects of two common types of haematomas, extra(epi)dural and subdural haematoma, are shown in Figure A.4.

The intracranial haematomas are named after the location in the head in which they occur (Figure A.5). Extradural haematomas (EDH) are caused by rupture of blood vessels lying on or above the dura mater (Gennarelli 1985). Almost always a meningeal artery is torn, most commonly a branch of the middle meningeal artery (Adams *et al.* 1982a). As a result of the rupture of a meningeal artery, blood accumulates between the vault and the dura mater. The outer layer of the dura is closely attached to the inner surface of the cranial bones, especially at the sutures and at the base of the skull. In order to be able to expand, the extradural haematoma has to separate the dura from the skull. Therefore, it will often take several hours before the haematoma attains a sufficiently large size to compress the surrounding neurological tissues substantially. According to Cooper (1982), 50 % - 68 % of the patients with EDH had no substantial intracranial pathology. The remainder of the patients had subdural haematoma and cerebral concussions associated with the EDH. These associated lesions influence the outcome of the extradural haematoma. Extradural haematomas are usually secondary to skull fracture and the outcome is worse when they are accompanied by skull fractures (Chapon *et al.* 1985).

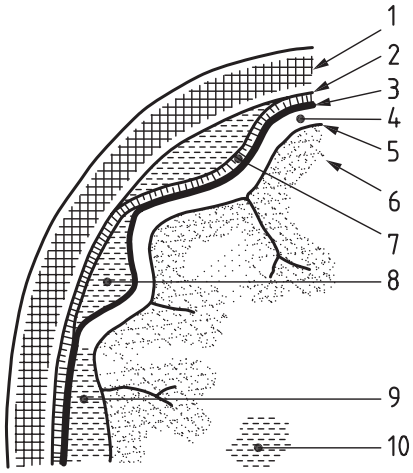


a) Extra (epi) dural haematoma **b) Subdural haematoma**

Key

1	Dura	2	Epidural hematoma	3	Subdural hematoma
4	Thick outer membrane	5	Compressed ventricular system	6	Thin inner membrane
7	Arachnoid				

Figure A.4 — Effects of two types of haematoma: Compression of the cortex and other intra cerebral structures as well as shiftingand bending of brain stem are clearly visible (Bakay and Glasauer 1980)



Key

1	Skull	2	Dura mater	3	Arachnoidal membrane	4	Subarachnoidal space
5	Pia mater	6	Brain	7	E.D.H	8	S.D.H
9	S.A.H	10	I.C.H				

Figure A.5 — Types of intracranial haematomas See text for meaning of the abbreviations (Chapon *et al.* 1983)

A subdural haematoma (SDH) is an accumulation of blood in the space between the dura and the arachnoid: the subdural space. According to Gennarelli and Thibault (1982), acute subdural haematoma (ASDH) was the most important cause of death in patients with severe head injuries. This was due to three factors: high incidence (30 %), high mortality (60 %) and high head injury severity (two-thirds have Glasgow Coma Scale of 3, 4, or 5) (Gennarelli *et al.* 1982a).

Clinically, three different kinds of ASDH can be identified:

1) penetrating injuries resulting in direct laceration of cortical arteries and veins;

2) large contusions causing bleeding into the adjacent subdural space; contusions of the temporal and frontal lobes are common causes of this type of ASDH;

3) the most common type of ASDH results from tearing of veins that bridge the subdural space between the brain's surface and the various dural sinuses.

Gennarelli and Thibault (1982) found that in angular accelerated primates ASDH always overlays the ruptured veins, its size being related to the number of disrupted veins. The subdural haematoma was in these cases always frontally predominant and frequently extended into the hemispheric fissure.

In subarachnoid haematomas (SAH), bleeding occurs in the space between the arachnoid and the pia mater. Subarachnoid haemorrhage seems by far the most common haemorrhage in patients who sustain closed head trauma but is in itself rarely lethal (Blumbergs 1985).

Intra cerebral haematomas (ICH) are well defined homogeneous collections of blood within the cerebral parenchyma (mainly in the frontal and temporal lobes), caused by rupture of large vessels that are located deep inside the brain (Prasad *et al.* 1985). Large traumatic intra cerebral haematomas are often associated with extensive cortical contusions (Gennarelli 1985). The haemorrhages begin superficially and extend deeply into the white matter. In one third of the cases, they extend as far as the lateral ventricle. Some cases of ICH extending into the corpus callosum and the brainstem have been reported (Cooper 1982).

The main significance of a traumatic intracranial haematoma is that it acts as an intracranial expanding lesion, ultimately leading to tentorial haematein and compression of the brainstem. Haematein of the brain through the tentorial notch - the hole in the tentorium through which the midbrain passes - is especially common as a cause of injury (Gardner *et al.* 1986). However, since many haematomas seem to expand slowly and progressively, there is often a delay before the clinical features of a high intracranial pressure and brain shift present themselves (Adams *et al.* 1982a). This delay leaves sufficient time for intervening medical care.

A.2.5 Cerebral Concussion

Concussion was one of the first head injuries to be scientifically examined and its characteristics have been studied intensively ever since. For a long time head injury accompanied by loss of consciousness was directly associated with concussion. Trotter (1924) was one of the first to clearly define concussion as "an essentially transient state due to head injury which is of instantaneous onset, manifests widespread symptoms of purely paralytic kind, does not as such comprise any evidence of structural cerebral injury, and is always followed by amnesia for the actual moment of the accident". This definition clarified the distinction between concussion and other types of head injury.

Several other researchers (Denny-Brown 1945; Symonds 1962; Ommaya 1966) have since contributed their personal interpretations and revisions of his definition. Nevertheless, the basic phenomena in all these definitions are impairment of consciousness, decrease in responsiveness and post traumatic amnesia. These effects are maximal immediately following injury, lessen progressively thereafter and are considered to be transient and fully reversible. Since the brainstem plays an important role in the functions that are impaired during concussion, many researchers have focused on brainstem injury as a cause of concussion.

Detailed information on the sequela of concussion in humans is rare since this type of head injury is never further examined after the first diagnosis. Most data on concussion has been obtained from animal and cadaver testing. However, two of the main characteristics of human concussion, duration of unconsciousness and amnesia, cannot be measured in experiments with animals (the level of unconsciousness in animals is hard to define due to the necessity for anaesthesia, and amnesia has no animal counterpart) and are not defined in cadaver tests. Therefore, in animal tests, other mainly physiological effects accompanying concussion are measured. The physiological effects occurring with concussion are typical changes in respiration, blood pressure and electrocardiogram (ECG) and electroencephalogram (EEG) signals, as well as certain typical reflexes (Ommaya 1966). These physiological reactions are reversible up to a certain magnitude of loading, but irreversible in more severe blows (Gurdjian *et al.* 1955; Olsson *et al.* 1971).

Ommaya (1966) raised the extremely important issue of doubt in transferring concussion data from lower species to humans. He stated that the smaller brains of lower animals tolerate injury better because they are more compact. The less compact, looser structure of larger brains deforms more readily when energy is applied. The similarity between the primate brain-cord geometry and that of humans would perhaps make such extrapolations more permissible. Supporting this argument, are the markedly opposite type of blood pressure change obtained in monkeys as compared with that in cats and dogs.

A.2.6 Diffuse and Axonal Injury

Experimental data indicates that the consequences of diffuse damage can cause injuries that vary from concussion, without apparent neurological sequelae, to prolonged traumatic coma with long-term, usually irreversible neurological outcome commonly found in severely head injured patients. According to Gennarelli (1987), diffuse damage is responsible for 40 % of all hospitalized head injury patients and for one-third of all fatal head injuries. It is also the main cause of permanent neurological disability in survivors.

Denny-Brown and Russell (1941) and Denny-Brown (1945) pioneered the research on diffuse injury. They found a gradation in the effects of cerebral concussion and claimed that concussion was a result of widespread deformation of neural tissue. Nevertheless, the term concussion was used for the entire range of injuries. Strich (1956) was one of the first to define, clearly, diffuse brain damage as a remarkable uniform pattern of abnormalities in the brain, characterized by focal, macroscopically identifiable lesions (tearing) in the rostral brainstem and in the corpus callosum, and microscopical evidence of diffuse damage to the white matter. Since then, numerous findings of diffuse abnormalities in white matter in patients with head injuries have been reported: (Strich 1961, 1969, 1970; Grecevik and Jacob 1965; Nevin 1967; Peerless and Rewcastle 1967; Oppenheimer 1968; Tomlinson 1970; Clark 1974; Adams 1975; Adams *et al.* 1977, 1980, 1982a, 1982b; Zimmerman and Bilaniuk 1978; Komatsu *et al.* 1979).

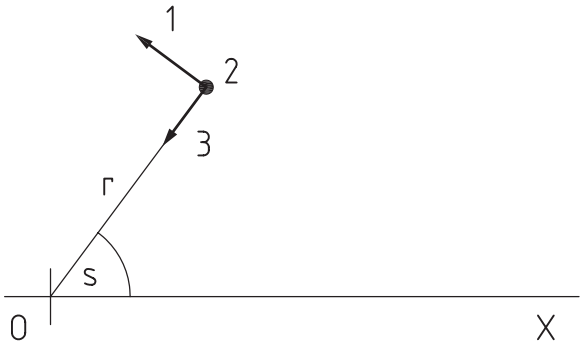
Adams *et al.* (1977) observed that the lesions in the rostral brainstem occurred in the dorsolateral quadrant in the region of the superior cerebellar peduncle. This indicates that these lesions might be caused by impact with the tentorium. The lesions were both macroscopic (mainly haemorrhages) and microscopic (rarefaction of tissue and axonal retraction balls). Lesions with essentially similar morphology were also found in the corpus callosum. The size of these lesions appeared to be inversely proportional to the duration of survival of the patient (Adams *et al.* 1977). Widespread damage to axons, another characteristic of diffuse brain damage, could only be identified microscopically and manifested itself in the form of axonal retraction balls, microglia stars and degeneration of fibre tracts throughout the white matter of the brain. Adams and Doyle *et al.* (1986) found that deeply seated intra-cerebral haematomas and contusions were associated substantially with diffuse white matter (diffuse axonal injury). It was Adams and Mitchell *et al.* 1977 who first used the term Diffuse Axonal Injury (DAI) to describe, perhaps more accurately, the cause of death associated with diffuse white matter damage. Since then the term has been used more widely, and perhaps wrongly, to describe many types of injury associated with axonal damage.

Gennarelli *et al.* (1982b) were able to reproduce all the characteristics of diffuse brain injury in their experiments with subhuman primates. They found a good correlation between the clinical state of the animals and the severity of the structural abnormalities in their brains. Animals with macroscopic lesions in the corpus callosum and rostral brainstem, and with numerous axonal retraction balls, either failed to regain consciousness or remained severely disabled after their injury. The retraction balls were most frequently seen in the parasagittal white matter in the cerebral hemispheres and in the midbrain and rostral pons. In less disabled animals, retraction balls were less numerous and the lesions in the corpus callosum and in the rostral brainstem could only be identified microscopically. The animals that made a good recovery from the injury showed no identifiable structural damage in the brain. Gennarelli *et al.* concluded that the severity of injury measured either by duration of unconsciousness or by outcome was associated with increasing incidence and severity of DAI and that neurological changes were highly correlated to the amount and distribution of axonal damage within the brain. However, it should be noted that not all lesions can be directly attributed to the immediate injury. Especially in long survival cases, secondary effects may have a definite influence on the observed changes in neuronal tissue. These secondary effects are induced by the immediate injury.

A.3 Head injury mechanisms

A.3.1 The dynamics of impact

Injury is caused when one particle of the body moves relative to the adjacent particle such that the elastic limit of the joining material is exceeded and damage occurs; the brain is no exception. Relative movement can occur only if adjacent particles are differently accelerated oevr time and although an impact to the head results in an acceleration (deceleration is simply negative acceleration) injury will not occur if the acceleration is evenly applied to all particles of the brain. In practice injury does occur and to understand why, it is important to consider the motion of a particle in space and for simplicity this will be confined to a plane.



Key

1 Transverse motion 2 Particle P 3 Radial motion

Figure A.6 — Representation of a particle in space using polar coordinates

With O as the pole, let OX be the initial line, let the polar coordinates of particle P be r, length of vector r, and θ being the angle between r and OX, positive in the anticlockwise sense. Thus $\dot{\theta}$ is defined as the angular velocity of P about O. The direction of the position vector of P is the radial direction and perpendicular to this, with θ increasing, is the transverse direction.

It can be shown using conventional vector analysis that the radial and transverse acceleration components are as follows:

radial component $r\ \ddot{\theta} + 2\ \dot{r}\ \dot{\theta}$

transverse component is $\ddot{r} \bullet r\ \dot{\theta}^2$

If the motion is circular about O with radius a and the particle passes through the position P with speed v then the tangential component becomes $a\ \ddot{\theta}$ and the normal component becomes $\frac{v^2}{a}$.

Thus this has illustrated mathematically that when a body rotates with uniform angular velocity there is a force toward the centre of rotation proportional to the distance from this centre and the mass. Hence an acceleration can be defined acting along a line passing through the centre. If the angular velocity changes then an angular acceleration is induced and this, in turn, gives rise to a linear acceleration normal to the line through the centre and proportional to the distance from the centre. It is this gradation of linear acceleration that gives rise to tangential sheer forces within the brain causing severe injury as is described below.

Rotation about a point other than the centre of gravity is a cyclic combination of linear and rotational motion because the average movement of the centre of gravity is zero over a period of time substantially greater than

the period of the cycle. This type of motion describes that of the head in a traffic accident and any other impact because of the rotation about the atlanto-occipital joint (neck), which can be considered to be a damped spring. The general equations for a point in space, as above, would be needed to calculate the total acceleration of a particle which would be a combination of linear acceleration generated by linear and rotational motion.

A.3.2 Injury caused by movement of the brain relative to the skull

Figure A.7 gives some of the possible effects of brain movement relative to the skull. Such large relative movements have been observed in experiments (Shelden *et al.* 1944; Pudenz and Shelden 1946; Gurdjian and Lissner 1961; Gosch *et al.* 1969, 1970; Ommaya *et al.* 1969; Shatsky *et al.* 1974a, b). In most of these experiments the whole upper half of the skull had been replaced by a lucite calvarium and because the dura had been removed, the effects of the tethering the brain at the vault could not be obtained.

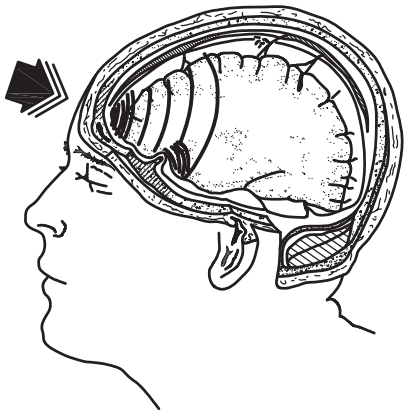


Figure A.7 — Some of the possible effects of relative brain movement (Viano 1988)

The skull is smooth at the vertex, but highly irregular at the base. Therefore, sliding of the brain against the internal surface of the skull is facilitated at the vertex, but is impeded at the skull base where it can lead to high shear strains in the meningeal and cortical tissues. Sufficient shearing of these tissues will cause lacerations, contusions and haematomas in the cortex.

The shape of the base of the skull is much more irregular in the frontal and temporal regions than in the occipital region. This explains why most cerebral contusions occur at the frontal and temporal lobes (Courville 1942; Gurdjian 1966), regardless of whether the site of impact is frontal or occipital (Gurdjian *et al*. 1955). Holbourn (1943) found in his gelatin model of the brain that in rotational motion the highest shear strains occurred in the anterior part of the temporal lobe near the base of the skull.

Rotation of the skull relative to the brain presses the highly irregular skull base towards the brain (Figure A.8). This leads to a combined compression and shearing of the meningeal and cortical tissues in this area, which increases the effects of the sliding of the brain over the skull base (Viano 1995). The effects of this relative rotation are most severe when the head is subjected to a rapid forward or rearward motion relative to the torso as shown in Figure A.8.

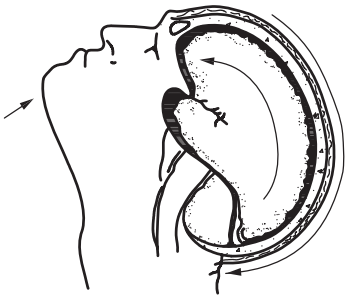
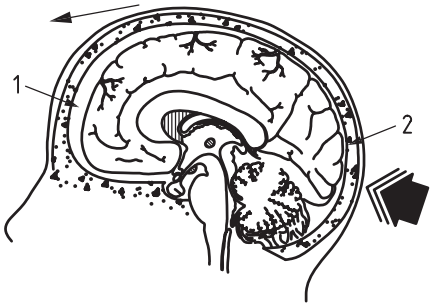


Figure A.8 — Rotation of the skull towards the brain (Sellier and Unterharnscheidt 1963)

The main functions of the connections between the skull and the brain at the vertex are to tether the brain to prevent excessive movements and to protect the blood supply to the cortical tissues. The functions of the tissues crossing the skull-brain interface at the skull base are much more vital. All of main blood vessels supplying the entire brain and the neurological connections between the brain and the rest of the body pass through this location.

The relative movement between the skull and the brain is always toward the site of impact. Because of this, intracranial tissue is compressed at the site of impact and strained at the contra lateral site. This leads to an increase in pressure at the site of impact and a reduction in pressure at the opposite site (Figure A.9).

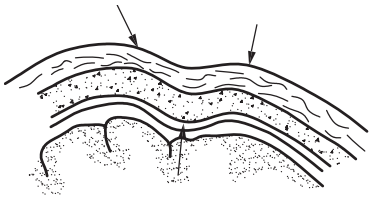


Key

1 Negative pressure 2 Positive pressure

Figure A.9 — Intracranial pressure changes due to relative movement between brain and skull (Douglass et al. 1968)

This effect was clearly visible in Nahum et al's experiments (1977), where pressurized cadaver heads were subjected to frontal impact (Figure A.10). The highest positive peak pressures were found beneath the impact site in the frontal region. The pressure magnitudes decreased and eventually became negative in the area opposite the site of the impact. The greatest negative pressures were generated at the posterior fossa which, due to the inclination of the skull, was the point opposite the impact site. Brain material is virtually incompressible and therefore the volume cannot change. The relative movement of the brain toward the site of impact compresses the brain in that direction. To balance this volume change, the brain expands sideways, which leads to increased pressure at the temporal lobes. This was seen in Nahum et al's tests. Since the surface area in the frontal region is much smaller than in the occipital region, relative movement between the brain and the skull leads to higher pressures in the frontal region than in the occipital region. The frontal base is also more irregular in shape and thus the frontal region of the skull is the most likely location for cortical injuries caused by relative movement between the brain and the skull.



The arrows denote the sites under tension resulting from skull bending. These sites are the most likely place for fractures to start (Douglass et al. 1968).

Figure A.10 — Skull bending caused by impact

It is interesting to see in Nahum *et al*'s findings that the pressure changes at the different locations inside the skull started at the same time and reach their peak values simultaneously. This indicates that the pressure changes are caused by rigid body displacements of the brain inside the skull and not by wave propagation effects. If the pressure changes had been caused by wave propagation effects, the changes would have started at the frontal lobe and gradually propagated to the back. Since this was not the case, it can be concluded that wave propagation effects have no serious influence on pressure changes inside the skull.

A.3.3 Cranial and related injuries

A.3.3.1 Skull fracture

According to Gurdjian et al. (1950) and Thomas et al. (1973), skull bending is the cause of linear skull fractures. As a result of an impact, the skull bends inwards at the site of the impact and bends outward at some distance from the impact site (Figure A.10). When the skull is deformed beyond its loading capacity, it fractures. Since bone is weaker in tension than in compression, cracks will appear at the skull's outer table in the regions where the skull bends outwards and on the inner table in regions in which the skull bends inwards. These cracks will propagate along lines of least resistance. The length of the fracture and its direction depend on the local material properties and skull thickness. Gurdjian et al. (1950) showed with their stress coat technique that outward bending, of the skull may occur at a considerable distance from the site of impact. They observed that at sufficiently high impact loads the inward bending area immediately around the point of impact was fragmented by radial fracture lines extending from the point of impact. This type of fracture has been defined in the previous section as a stellate fracture. The radial fractures are caused by the tensile stresses on the internal surface of the skull. On the outer surface of the skull, circular or curvilinear fractures occur at the junction of the indented and not-indented skull. The distance of these fractures from the site of impact denotes the extend of the area of indenting.

In an extensive survey of the literature on basilar skull fracture by Huelke et al. (1988), several mechanisms were proposed as the cause of these fractures. Originally, it was thought that basilar fracture results from cranial vault impacts, causing deformations remote from the impact site (Gurdjian et al. 1949, 1953; Walker 1973). Ring fractures about the foramen magnum were shown to be likely to occur in impacts to the crown of the head, driving the skull downward onto the cervical spine (Voight and Skold 1974; Skold and Voight 1977). Voight and Skold (1974) noted that once a ring fracture occurred in the middle fossa, the skull became unstable and deformed dramatically. This deformation caused further propagation of the original fracture into other parts of the skull. Thom and Hurt (1993) found that axial loading of the neck was significantly associated with basilar fracture for unhelmeted motorcyclists.

In addition to impacts of the vault, many authors emphasize the strength of the atlanto-occipital ligaments as a cause of basilar skull fractures. The strength of these ligaments between the skull and the cervical spine will prevent separation of the cervical spine and the skull base in cases where the skull is pulled away from the spine. As a result of this, the skull base will fail, leading to ring fractures at the occipital skull base, instead of tearing of the ligaments (Thom and Hurt 1993). This mechanism was thought to be the cause of ring fractures resulting from hyper motions of the head and experimental data has substantiated this theory. In 1973, Schmidt showed that basilar ring fractures can be caused by hyperextension. In his tests, the basilar ring fracture segment showed slight outward separation from the main portion of the skull, indicating a distraction type of fracture. Voight and Skold (1974) found that frontal impact causing hyperextension was a cause of incomplete ring fractures. The strength of the atlanto-occipital ligaments was also thought to be the cause of

ring fractures in hyperflexion of the neck resulting from occipital impacts. This mechanism might also explain the findings of Clemens and Burow (1972), where ring fractures of the occipital bones were found when human cadaver torsos were accelerated. Thom and Hurt (1993) found that hyperextension was associated with fractures to the anterior and middle fossae of the skull base. Surprisingly, cervical torsion and lateral motion were not found to be related to basilar skull fracture in this study.

Apart from the previous two injury mechanisms, there are indications that basilar skull fractures can also be caused by impacts to the face and especially the mandible (Hodgson *et al.* 1970; Thomas et al. 1973; Brit *et al.* 1980; Harvey and Jones 1980; Lau *et al.* 1987). Hurt *et al.* (1981) studied 900 motorcycle accidents in Los Angeles and noted that in severe impacts to the mandible, the transmission of the force through the mandible could produce a basilar skull fracture with laceration of the base of the brain. The possible ways of force transmission from mandibular impact to the skull base are shown in Figure A.11. Huelke *et al.* (1988) concluded that basilar fractures, particularly the hinge type, were produced by facial impact, especially impact to the anterior mandible. From the above it can thus be concluded that basilar skull fractures are related to impacts to the head, forces acting on the head and neck, and hyper motions caused by these forces.

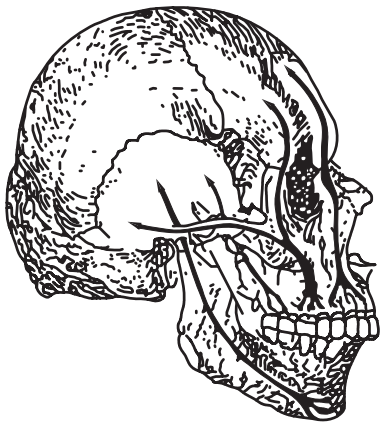


Figure A.11 — Possible ways of force transmission to different parts of the skull following mandibular impact (Chapman 1985)

It is shown consistently in the literature that basilar skull fractures are linear fractures and are generally aligned with or parallel to the direction of force application, e.g., rear impact caused posterior fossa fracture (Thom and Hurt 1993). Important exceptions to this rule were the cases of hyperflexion from rear impacts, which caused partial or complete ring fracture, and hyperextension from frontal impacts, which caused hinge fracture. Regardless of their location, all these fractures were brittle tensile fractures due to skull bending. While the energy and forces causing fractures of the cranial vault have been quantified experimentally, the complex nature of the base of the skull has prevented equivalent quantification of fracture forces for this basilar region (Huelke *et al.* 1988). However, it is generally acknowledged that forces causing basilar skull fracture are less than those causing fracture of the vault (Thom and Hurt 1993).

A remarkable finding in research on basilar skull fractures is that of Alem *et al.* (1984). They impacted the crowns of heads of unembalmed cadavers and results showed a rigid impacting surface with sufficient impact energy to cause fractures at the impact site. However, if under the same conditions the impact site was padded, the fractures appeared at the base of the skull. Increasing the thickness of the padding prevented skull fractures, but the fractures then occurred in the cervical spine. Gurdjian *et al.* (1949) had already found that a relatively thin layer of soft tissue, e.g., the scalp, considerably increased the amount of energy required for fracture. Nahum *et al.* (1968) also found that the thickness of soft tissues and particularly its presence or absence were critical. Further research may provide a better understanding in the high incidence of basilar skull fractures in both unhelmeted and helmeted motorcyclists (Hurt *et al.* 1986; Thom and Hurt 1993).

A.3.3.2 Intracranial injuries related to skull fracture

Fragments of bone resulting from skull fracture or skull penetration has been shown to cause damage to underlying meningeal and cortical tissues. The dura is adherent to the inner aspect of the cranial bones, particularly at the sutures and at the base of the skull, and contains several blood vessels. Skull deformation

or skull fracture can easily cause rupture of these blood vessels, leading to an extradural haematoma (Adams *et al.* 1980; Cooper 1982; Chapon *et al.* 1985). Acute subdural haematomas can be caused by direct laceration of the bridging veins or the cortical veins and arteries by penetration wounds resulting from impacts to the head (Gennarelli 1985). Large cortical contusions resulting from skull deformation or skull penetration can lead to subdural haematomas and/or subarachnoid haematomas (Gennarelli 1985).

Several researchers have addressed skull denting as a cause of cortical contusions (Holbourn 1943, 1945; Gurdjian and Gurdjian 1976; Nusholtz *et al.* 1984; Gennarelli 1985). According to Gennarelli (1985), contusions beneath the impact site are caused either by skull denting or by the rapid snapping back of a dented area of the skull. In the first case, the contusion is caused by compressional loading of meningeal and/or cortical tissue. In the second case, the brain surface is subjected to tensile forces. However, Viano (1995). questions whether the skull is capable of denting sufficiently to actually cause these injuries. As will be shown later, it is more likely that contusions are caused by contact of the brain with more rigid intracranial surfaces due to inertial loading of the brain after the head is set into motion. One way to check which of the two injury mechanisms mentioned above is correct is to look at the results of impacts to constrained heads. According to the skull bending mechanism, contusions beneath the impact site would still occur in this case. According to the relative skull-brain movement mechanism, the brain will not move relative to the skull and no (or far fewer) contusions would occur. Unfortunately, publications of such experiments have not been found. Further, it is generally acknowledged that contusions are caused by compression of a tissue. Therefore, Gennarelli's theory that cortical contusions can be caused by tension loading does not comply with this general definition of contusion. Either, another injury mechanism causes these cortical contusions, or the observed injuries are not contusions, but another type of vascular injury.

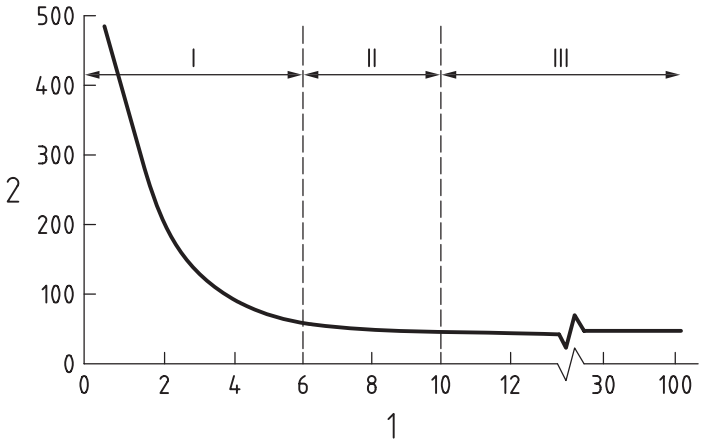
Gurdjian (1975) and Gennarelli (1985) claimed that shock waves resulting from contact phenomena can cause injury remote from the impact site. Although it is a fact that impact will inevitably lead to wave propagation, the extent of wave propagation effects inside the head are still not clear and, from evidence in the previous section, it seems very unlikely that wave propagation can actually cause brain injury.

A.4 Head injury criteria

A.4.1 Wayne State tolerance curve

The Wayne State Tolerance Curve is considered to be the foundation of research on human head injury criteria. This curve evolved from the work of Lissner *et al.* (1960), Gurdjian *et al.* (1953a, 1961) and Patrick *et al.* (1963), and gives the tolerable average acceleration in A-P direction (Anterior-Posterior) as function of the pulse duration. The curve is given in Figure A.15. Slight cerebral concussion without any permanent effects was considered to be within human tolerance. Only translational accelerations were used in the development of the curve which was obtained from different experiments with cadavers, animals and volunteers. That substantial acceleration causes injury over short durations, while smaller accelerations require longer duration to cause injury is an assumption fundamental to the curve formulation.

The short duration part of the curve (*2<t<6 ms*) was derived from cadaver tests in which skull fracture was chosen as injury criterion. Cadaver and animal tests were used for the intermediate pulse durations (*6<t<10 ms*). For this part of the curve, intracranial pressure was used as the injury criterion in the cadaver tests and concussion was chosen as the injury criterion in the animal tests. The long duration part of the curve (t>*10 ms*) was obtained from volunteer tests. There was no head impact in these tests and no injuries were observed. By assembling all these tests in one curve it was assumed that skull fracture and concussion correlate. Lissner et al. maintained that for a given duration, accelerations above the curve lead to injury (survival hazards), while accelerations below the curve are tolerable and cause, at most, cerebral concussion without permanent effects. Except for the long duration accelerations, the WST-curve has never been validated for living human beings.



Key

1 Impulse duration t (ms) 2 Acceleration (g)

Figure A.12 — Wayne State Tolerance Curve

The figure is divided into 3 parts:

1) short duration area, obtained from cadaver experiments;

2) intermediate duration area, obtained from cadaver and animal experiments;

3) long duration area, obtained from volunteer tests.

Accelerations are given in terms of the gravitational acceleration [g]. At a given duration, accelerations above the curve give injury, while accelerations below the curve do not lead to injury (Beusenberg 1991).

A.4.2 Head injury criterion, HIC

The Wayne State curve as described above led to the development of the Gadd Severity index (GSI) which was expressed in the form:

$$GSI = \int_T a(t)^{2.5}\, dt$$

where

T = the total pulse duration, and

$\alpha(t)$ = acceleration at the centre of mass of the head, as a function of time.

This was described as the weighted impulse criteria for which a value of 1 000 was considered unsafe. However, it can be shown that for irregular pulse shapes, there may exist within the pulse envelope which has a value greater than that for the whole pulse. Thus, it was decided that the maximum value within the pulse should be assumed to be the criterion for head injury.

This became the Head Injury Criteria, HIC, which is given below:

$$HIC = \left[\left(\frac{1}{t_2 - t_1} \cdot \int_{t_1}^{t_2} a_{res}\, dt \right)^{2,5} \cdot (t_2 - t_1) \right]_{\max}$$

with:

t_1 and t_2 [ms] any two points in time during any interval in the impact;

a = resultant acceleration of the centre of mass of the head. After much discussion over many years, t_1 and t_2 were defined to be any two times during the entire impact duration for which HIC is a maximum value. Hodgson and Thomas (1972) suggested that the critical HIC interval should be less than 15 ms, even if the HIC value exceeded the threshold of 1 000 over a longer interval. His finding was based on examination of events where the concussive outcomes were known or could be determined. The threshold of 1 000 is still under discussion, because head injuries were found at HIC values of 500, while HIC values of 3 000 were sustained without major injury.

A.4.3 Rotational acceleration — Published research

There is no criterion related specifically to rotational acceleration. However, there has been research to determine what values of rotational acceleration are likely to cause injury and this is reviewed below.

Ryan et al (1989) attempted a more complex prediction of linear and rotational acceleration based on 26 fatal pedestrian impacts with cars. The linear acceleration was estimated from impact velocity, head mass and the combined stiffness of the head and vehicle panel. For occipital impacts death was reported to occur at linear accelerations of about 200 g upwards, and it seemed to be the case that increasing linear acceleration, up to about 700 g, was accompanied by increasing angular acceleration ranging from 6 000 rad/s^2 to 15 000 rad/s^2. For lateral impacts there was no clear relation between brain injury density, BID, and linear acceleration, which had a similar range to that for occipital impacts (200 g – 700 g), but the range of angular acceleration was higher, at 8 000 rad/s^2 to 50 000 rad/s^2. The estimated values for linear acceleration indicate that fatal injuries can occur at 200 g and above. This is fairly consistent with Newman's proposed scale (1986) which assigns AIS 4 to a range of peak accelerations of 200 g – 250 g, but, as noted above, it is also consistent with HIC levels of the order of 1 000 or more.

The information above is based on samples of mainly fatally injured road accident victims, but it also includes some very seriously injured victims. However, if a device to protect the head is to be successful, then data on those who have received blows, but remain largely uninjured, needs to be considered and used in the design of the device. This is necessary to avoid biasing the efficacy of a device to the advantage of a "weak" sector of the population which may result in a disadvantage for a "strong" sector. This is particularly relevant to a device such as a helmet where the thickness of the energy absorber is strictly limited and needs to be optimised for a representative spread of that population most likely to use the helmet. Thus it is important to consider research such as that by Pincemaille et al (1989) who measured head accelerations sustained by volunteer boxers. The peak linear accelerations recorded ranged from 20 g to 159 g, but were of very short duration so that the corresponding HIC values ranged from 5 - 348. However, the peak angular accelerations were as high as 16 234 rad/s^2, while 3 500 rad/s^2 was the minimum recorded peak for any blow, of which a total of over 80 were analysed. In only one case was any impairment noticed and this was considered very slight and was temporary. These values for rotational acceleration contrast sharply with the value of 4 500 rad/s^2 reported by Löwenhielm (1975) to have caused fatal bridging vein rupture in a 10 year old girl who fell to the ground from an armchair.

A.5 Noise

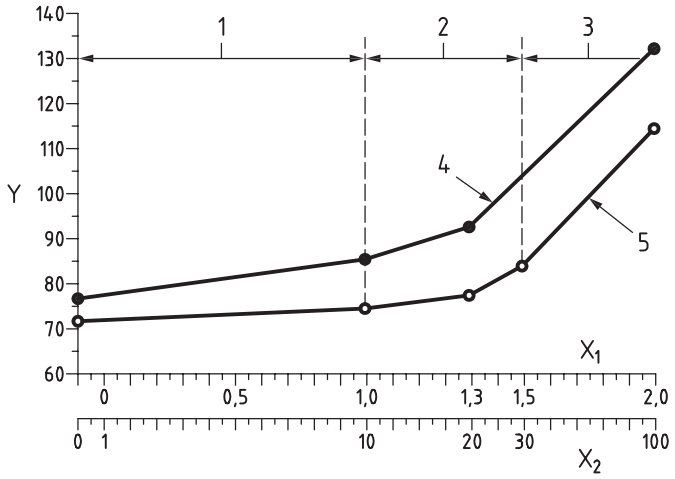
A.5.1 Introduction

Certain levels of noise for periods in excess of defined limits can cause premature deafness. Equipment to protect from noise is available but under some circumstances the characteristics of a helmet can exacerbate the ambient noise levels or conversely reduce the level of important auditory signals such that the safety of the wearer is reduced. Tests for noise need to be established and helmets evaluated against such tests where the wearer is potentially at risk. The following information relates to motorcycle helmets but the principles embodied may be applied to any helmet or ear defender.

A.5.2 Motorcycle aerodynamic noise

It is low-frequency aerodynamic noise, generally centred around 500 Hz, created by the turbulent airflow over the rider's helmet that has been shown to be the major contributory factor to hearing problems. Previous research has demonstrated that both short-term and long-term hearing damage can occur as a result of exposure to aerodynamic noise. At speeds greater than 40 mile/h (64 km/h) approximately 90 dB(A) of low-frequency wind noise is produced at the rider's ear, with this level rising to around 120 dB(A) at 100 mile/h (160 km/h).

At 55 mile/h a motorcyclist experiences approximately 96 dB(A) of aerodynamic noise and an exposure of more than two hours would exceed the second action level of the Health and Safety at Work Act, Great Britain where the wearing of hearing protection equipment becomes compulsory. Therefore, noise attenuation, especially at low frequencies is an area in of helmet design that has the potential for considerable improvement and would confer substantial benefits to motorcycle riders.



Key

1 Vehicle noise	2 Transition	3 Wind noise	4 Barehead
5 Helmet	Y Sound level dB(A)	X_1 Log $_{10}$ Speed	X_2 Speed (mph)

Figure A.13 — Aerodynamic noise generated at different motorcycle speeds

Exposure to this excessive low-frequency noise has the potential to cause hearing damage. Figure A.13 shows that the levels of helmet noise rise to decibel values which, depending on the exposure time and the frequency of exposure, are capable of hearing damage at relatively low speeds.

A.5.3 Recommended test methods for measuring helmet noise

A.5.3.1 General

The problems associated with helmet designs and the effect on rider hearing have been outlined in the previous sections. It is recommended that where appropriate helmet Standards include a test for the helmet sound attenuation capabilities. There are two main areas that must be tested to ensure that aural damage is minimised. These are the aerodynamic design of the helmet, if appropriate, and the degree to which the helmet attenuates the noise over a range of frequencies-necessary to ensure that auditory warnings are not over attenuated.

Two tests are proposed which will allow the noise qualities of different types helmet to be compared. Test A aims to identify the noise dose at the wearer's ear, whereas test B provides a more detailed breakdown of the sound attenuation capabilities of the helmet over a range of frequencies. The latter test therefore enables the

performance of the helmet over the important frequency ranges to be assessed and ensures that warning sounds are audible.

A.5.3.2 Test A – Assessment of aerodynamic noise- summary

A.5.3.2.1 General

Assessment of the 'equivalent continuous noise level' over a period of [5] minutes (LAeq,[5] minutes) for a range of airflow speeds. The test limits have yet to be defined, but would be expressed as a maximum for each airflow speed. It is suggested that at 70 mile/h (112 km/h) the maximum be set at a L_{Aeq},[5] minutes of 90 dB(A).

A.5.3.2.2 Equipment requirements

The following equipment is necessary:

- a suitable sub-sonic wind tunnel, capable of airflow velocities up to 100 mile/h, in which a helmeted headform may be positioned far enough away from the sides to avoid interference;
- a suitable dummy headform, either modified such that it has a representative human ear structure or an appropriate proprietary dummy supplied with the necessary microphone equipment within the ear;
- an appropriate miniature microphone (IEC 651 type 0 or 1);
- appropriate analysis equipment of equivalent certified accuracy as that of the microphone capable of recording L_{Aeq} dB(A) and peak dB.

A.5.3.2.3 Test procedure

The miniature microphone shall be secured within the 'ear' of the headform and the helmeted headform placed inside the sub-sonic wind tunnel in a position such that the airflow around the helmet is unaffected by any structure other than the helmet under test.

The dB(A) over a period of [5] minutes and the peak dB level shall be recorded at every airflow velocity from 40 mile/h to 100 mile/h, with airflow speed increasing in 10 mile/h increments.

A.5.3.2.4 Test criteria and limits

The criteria of the test are L_{Aeq},[5minutes] and peak dB (not A-weighted). The latter value is not A-weighted in order to allow compatibility with the European Directive 2003/10/EC.

The limits of these values is more difficult to define and require further research and testing before any figures are set. However, sensible limits based on what might be achievable may be that L_{Aeq}, [5]minutes does not exceed 90 dB(A) at 70 mile/h (112 km/h) and that at no speed must the peak dB level exceed 140 dB (A).

For the purposes of the European Directive 2003/10/EC the exposure limit values and exposure action values in respect of daily noise exposure levels and peak sound pressures are fixed at:

a) exposure limit values: $L_{EX.8heq}$, = 87dB(A) and P_{peak} = 200 Pa;

b) upper exposure action values: $L_{EX.8heq}$, = 85dB(A) and P_{peak} = 140 Pa;

c) lower exposure action values: $L_{EX.8heq}$, = 80dB(A) and P_{peak} = 112 Pa.

Since the exposure of a motorcyclist to noise is likely to be less than an average working day, it seems sensible to set the noise level for the maximum permissible speed limit (70 mile/h UK) at the exposure limit values: $L_{EX.8heq}$, = 87 dB(A) and P_{peak} = 200 Pa. In addition, the draft code of practice for noise exposure at

discotheques (Noise Advisory Council, 1986) stated that the maximum permissible noise exposure level should be a L_{Aeq}, 5minutes of 100dB (A) within an area directly adjacent to the speakers. Helmets for motorsport however require separate limits to be set since at high speeds even 100 dB(A) will be exceeded. However, it is unlikely that the noise level may be retained below a fully 'safe' threshold even with the use of technologies such as Active Noise Reduction (ANR). Therefore, a limit based on what may be achieved by current helmets would encourage manufacturers to design helmets with noise qualities in mind. Equivalent continuous noise levels (L_{Aeq}, [5]minutes) limits for speeds of 40 mile/h, 50 mile/h, 60 mile/h, 70 mile/h, 80 mile/h, 90 mile/h and 100 mile/h should be defined so that a helmet would protect the rider sufficiently at a range of speeds. It is suggested that a ranking system is used to compare helmet performance. This would provide a simple and clear indication of the noise-reducing qualities of the helmet.

A.5.3.3 Test B Assessment of sound attenuation by frequency- summary

A.5.3.3.1 General

This test is an assessment of noise attenuation by sound frequency. This would enable determination of an audiogram for the entire frequency spectrum. The test criteria would be expressed in dB(A) and the attenuation limits are yet to be defined and would vary depending on the sound frequency.

A.5.3.3.2 Equipment requirements

The following equipment is necessary in order that this test may be performed.

- suitable anechoic chamber conforming to BS 6655;
- suitable dummy headform, either one modified such that it has a representative human ear structure or an appropriate proprietary dummy which is supplied with the necessary microphone equipment within the ear;
- appropriate miniature microphone (IEC 651 type 0 or 1);
- appropriate analysis equipment of equivalent certified accuracy as that of the microphone capable of recording dB(A);
- appropriate speaker system capable of emitting pure tones of a specified frequency.

A.5.3.3.3 Test procedure

The miniature microphone shall be secured within the 'ear' of the headform and the helmeted headform placed inside the anechoic chamber.

A series of pure tones between 100 Hz and 6 000 Hz shall be emitted from the speaker positioned exactly 1 metre from and at right-angles to, the in-ear microphone. The sound level dB(A) shall be recorded with and without the test helmet fitted and an audiogram determined to assess the noise attenuation qualities of the helmet over the entire frequency spectrum.

A.5.3.3.4 Test criteria and limits

The criteria of the test are proposed to be dB(A). The attenuation requirements are difficult to define. However tests with ANR fitted to a motorcycle helmet reliably achieved noise reductions of 3-8 dB(A) and up to 11 dB(A) under optimum conditions. Furthermore, past testing involving ANR fitted into flying helmets have resulted in reductions of up to 14 dB(A), indicating that such reductions for motorcycle helmets are feasible. Even without ANR technology, improvements in visor fit, shell aerodynamics, ear padding, and the fit of the helmet at the neck have the potential to deliver significant noise reductions in the range 5-8 dB(A). It is important that an optimum attenuation audiogram is identified so that the limits for noise attenuation over the frequency range influenced by aerodynamic noise can be identified without affecting safety by impairing

hazard detection. Once this has been determined, appropriate attenuation limits for each frequency range may be proposed.

However, limits such that attenuation of at least [6]dB(A) are achieved is suggested, since such a reduction in the range 500 Hz – 1 000 Hz would have the effect of reducing the sound pressure level reaching the ear by 75 %.

A.6 Heat: burns and fatigue

A.6.1 Introduction

Temperatures above certain values will cause burn injuries the extent of which will depend on the exposure. Equipment to protect from heat is available but the greater the temperature against which it is designed to protect the more of an encumbrance it becomes and the greater the fatigue when it is worn. This dichotomy needs to be considered when writing Standards for such equipment, for firefighters, for example, and the follwing information is presented to help with this. The information is given in A.5.2.

A.6.2 Anatomy of the skin

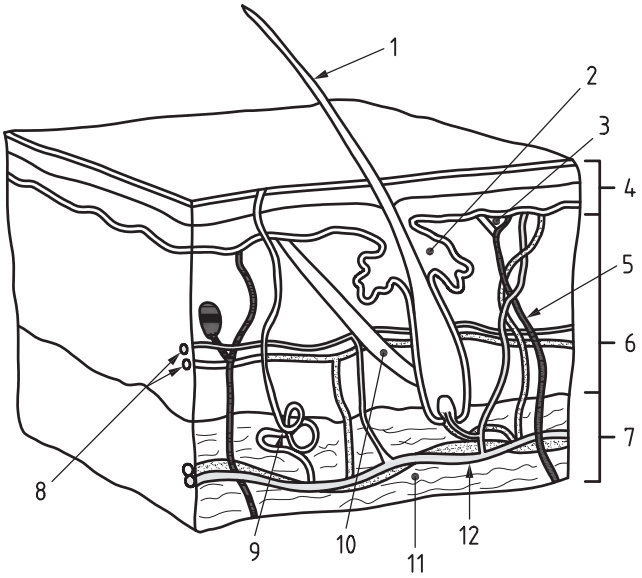
The skin is the largest organ of the body and covers the entire body. In addition to protecting against heat, light, injury and infection, the skin also: regulates body temperature, stores water and fat, is a sensory organ, prevents water loss and prevents entry of bacteria.

Throughout the body, the characteristics of the skin, such as thickness, colour and texture vary. For example, the head contains more hair follicles than any other body region, while the soles of the feet contain none. In addition, the soles of the feet and the palms of the hands are much thicker.

The skin is made up of the following layers, with each layer performing specific functions:

- epidermis;
- dermis;
- subcutaneous fat layer.

Figure A.14 below illustrates the composition of the skin.



Key

1	Hair	2	Sebaceous gland	3	Sensory nerve ending	4	Epidermis
5	Nerve	6	Dermis	7	Subcutaneous tissue	8	Capillaries
9	Sweat gland	10	Muscle	11	Fat, collagen, fibroblasts	12	Arteriole

Figure A.14 — The structure of the skin and the relative position of the layers

The epidermis is the thin outer layer of the skin, which consists of three parts:

— stratum corneum (horny layer);

— keratinocytes (squamous cells);

— basal layer.

The stratum corneum comprises contain fibrous proteins (keratins).and the outermost layer is continuously shed. The stratum corneum prevents the entry of most foreign substances as well as the loss of fluid from the body.

The keratinocytes layer, just beneath the stratum corneum, contains living keratinocytes (squamous cells), which mature and form the stratum corneum. The basal layer is the deepest layer of the epidermis, containing basal cells. Basal cells continually divide, forming new keratinocytes, replacing the old ones that are shed from the surfae of the skin. The epidermis also contains melanocytes, which are cells that produce melanin (skin pigment).

The dermis is the middle layer of the skin. and contains the following:

— blood vessels;

— lymph vessels;

— hair follicles;

— sweat glands;

— collagen bundles;

— fibroblasts;

— nerves.

The dermis is held together by a protein called collagen, made by fibroblasts. This layer also contains pain and touch receptors.

The subcutis (subcutaneous fat layer) is the deepest layer of skin and consists of a network of collagen and fat cells, which help conserve the heat of the body and protects the body from injury by acting as a "shock absorber."

A.6.3 Burns and the consequences

A burn is one of the most painful of all injuries. When a burn occurs to the skin, nerve endings are damaged causing intense feelings of pain. Serious burns are complex injuries and in addition to the burn injury, a number of other functions may be affected. Burn injuries can affect muscles, bones, nerves and blood vessels. The respiratory system can be damaged, with possible airway obstruction, respiratory failure and respiratory arrest. Burns injure the skin and, therefore, impair the body's normal fluid/electrolyte balance, body temperature, body thermal regulation, joint function, manual dexterity, and physical appearance. In addition to the physical damage caused by burns, casualties may also suffer emotional and psychological problems that begin at the accident site and can last a long time.

Burns are classified in two ways, by method and by degree.

Methods include:

— Thermal - including flame, radiation, or excessive heat from fire, steam, and hot liquids and hot objects;

— Chemical - including various acids, bases, and caustics;

— Electrical - including electrical current and lightning;

— Light - burns caused by intense light sources or ultraviolet light, which includes sunlight;

— Radiation - such as from nuclear sources and ultraviolet light can also be a source of radiation burns.

Degree is defined as follows:

— **First degree burns** are superficial injuries that involve only the epidermis or outer layer of skin. They are the most common and the most minor of all burns. The skin is reddened and extremely painful. The burn will heal on its own without scarring within two to five days. There may be peeling of the skin and some temporary discoloration.

— **Second degree burns** occur when the first layer of skin is burned through and the second layer, the dermal layer, is damaged but the burn does not pass through to underlying tissues. The skin appears moist and there will be deep intense pain, reddening, blisters and a mottled appearance to the skin. Second degree burns are considered minor if they involve less than 15 percent of the body surface in adults and less than 10 percent in children. When treated with reasonable care, second degree burns will heal themselves and produce very little scarring. Healing is usually complete within three weeks.

— **Third degree burns** involve all the layers of the skin. They are referred to as full thickness burns and are the most serious of all burns. These are usually charred black and include areas that are dry and white. While a third-degree burn may be very painful, some patients feel little or no pain because the nerve endings have been destroyed. This type of burn may require skin grafting. As third degree burns heal, dense scars form.

Knowledge of the consequences of different sources and the effect on different body regions is important when designing protection. A minor burn caused by nuclear radiation is more severe than a burn caused by thermal sources whereas chemical burns are dangerous because the chemical may remain on the skin.

Burns to the face are considered the most severe because breathing and eyesight can be affected although burns to hands and feet are also of special concern because movement of fingers and toes could be impeded.

Degree (depth) of the burn is important because it could cause infection of exposed tissues and permit invasion of the circulatory system. Thereafter, area, the amount of the skin surface involved in the burn is critical. The adult body is divided into regions, each of which represents nine percent of the total body surface. These regions are the head and neck, each upper limb, the chest, the abdomen, the upper back, the lower back and buttocks, the front of each lower limb, and the back of each lower limb. This constitutes 99 percent of the human body; the remaining one percent is the genital area. With an infant or small child, more emphasis is placed on the head and trunk.

A.6.4 Burn severity and heat exposure

The temperature to which the skin is exposed and the length of time of the exposure determines the severity of the burn. The following is a Table A1 produced by the Burn Foundation, Philadelphia, PA. (www.burnfoundation.org) and the American Burn Association, Chicago, IL. (www.Ameriburn.org). It gives the water temperature and the time required for a 3rd degree burn.

Table A.1 — Water temperature and time for 3rd a degree burn

Water temperature ºF (ºC)	Time required for a 3rd degree burn s (second), m (minute)
155 (68)	1 s
148 (64)	2 s
140 (60)	5 s
133 (56)	15 s
127 (53)	1 m
124 (51)	3 m
120 (49)	5 m (10)
100 (38)	safe

Research by RF Chaillet *et al.* (1965) has identified the effects of surface contact at different temperatures and his conclusions are given in Table A.2.

Table A.2 — Surface temperature and time for injury

Surface temperature ºF (ºC)	Time second	Injury or sensation
212 (100)	15	2nd Degree burn
180 (82)	30	2nd Degree burn
160 (71)	60	2nd Degree burn
140 (60)	NS	Pain; tissue damage
120 (49)	NS	Pain
93 (34)	NS	Warm; neutral effect
54 (12)	NS	Cool
37 (3)	NS	Cool; uncomfortable
32 (0)	NS	Pain
<32 (<0)	NS	Pain; tissue damage
NS = not specified		

Information from the New York Fire Department (F.D.N.Y) (2003) gives an indication of injury for exposure to various air temperatures as indicated in Table A.3.

Table A.3 — Air temperature, time for injury

Air temperature ºF (ºC)	Time second	Injury
212 (100)	15	2nd Degree burn
180 (82)	30	2nd Degree burn
160 (71)	60	2nd Degree burn

These figures are identical to those in Table A.2 and suggest that contact with air and solid surfaces requires longer times for a given injury than contact with water at a similar temperature. This is to be expected and relates to the thermal capacity of the substance and the fact that water has one of the highest known values and, furthermore, steam condensing to water emits large quantities of heat (latent heat of evaporation).

White and Bowen (1959) have provided an indication of thermal energy per unit area (radiation) related to injury as follows:

Table A.4 — Thermal energy and time for injury

Thermal Energy/unit area cal/cm^2 (kJ/m^2)		Time (sec)	Radiation kW/m^2	Injury	
White Subjects	Black Subjects		White Black Subjects	Ave	
3.2 (7.6)	N/A	0,5	3.8[8] N/A	3.8_8	1st Degree burn
3.9 (9.3)	1.8-2.9 (4.3-6.9)	0,5	4.65_8 2.65-3.95[8]	4.0[8]	2nd Degree burn
4.8 (11.4)	3.3-3.7 (7.9-8.8)	0,5	5.7[8] 3.95-4.4[8]	4.5[8]	3nd Degree burn
Values in () and [8] calculated by the author.					

This indicates that black people are more susceptible to burns than white people, which is perhaps counterintuitive and should be noted. The metric MKS values in brackets and the radiation values were calculated by the author.

Paper CEN/TC 159 N283 (2002) by the joint working group on firefighters protective equipment has stated that "human skin is subject to bums at a skin temperature of 44 °C and the rate of injury is roughly trebled for each °C increase above the threshold tissue temperature. For example, the damage rate at 50 °C is about 100 times that at 44 °C - 45 °C."

However, this is difficult to relate to conventional scientific data such as is given in Tables A.1 to A.4 because it does not quantify the damage rate. If damage rate is assumed to be proportional to time then the increment from 71 ºC to 82 ºC (11 ºC) should be 1/3[11] which equals 5,6 x 10^{-6} whereas the factor in Table A.2 for this temperature increment is 0,5. It is suggested that the committee defines what is meant by damage rate.

If persons, for example firefighters, are to be protected in hot environments then it is important to define such environments and ensure that the helmet and protective clothing is designed to minimise the extent of burn injury by reference to the data given in Tables A.1 to A.4. Paper CEN/TC 159 N283 (2002) has categorised environments as follows:

- Level 1: Routine conditions - air temperatures up to approximately 100 °C and a radiant heat source of up to approximately 1,25 kW/m^2.
- Level 2: Hazardous conditions - air temperatures up to approximately 250 °C and a radiant heat source of up to approximately 8 kW/m^2.
- Level 3: Emergency conditions - air temperatures up to and above 800 °C and radiant heat sources from 80 kW/m^2 up.

Research by the joint working group on radiant heat measured the distance from heat source to sample and reported thus: "for heat fluxes at various intensities from 10 to 80 kW/m". At a source temperature of 1 000 °C, a temperature of 600 °C was calculated 1,6 m from source; of 500 °C, 2,25 m from source; of 400 °C, 2,9 m from source and of 300 °C, 4,9 m from source. These figures must be taken with caution as they take into account only radiation and assume no convection of air - which is an environment which could not exist in a real life.

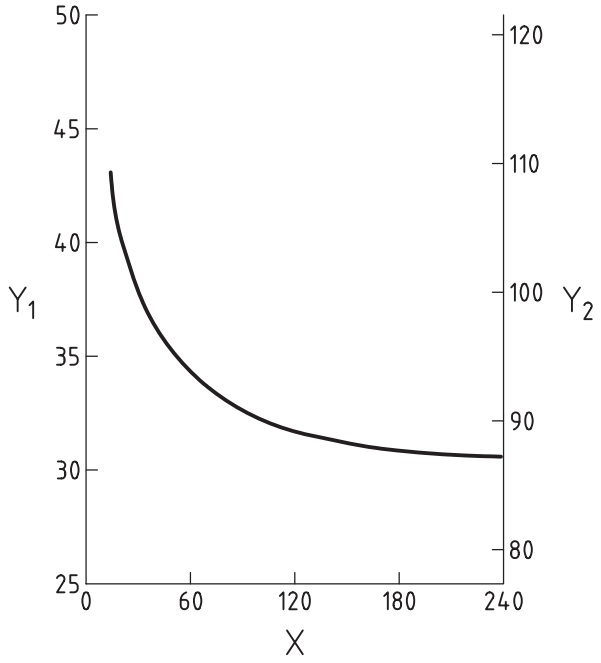
Further testing was carried out with a Subject- not a trained Firefighter - dressed in Firefighters' Personal Protective Equipment to EN Standards (Helmet with visor, clothing, gloves, boots - but no Self Contained Breathing Apparatus) over undergarments of a short sleeved cotton T shirt; standard cotton based working rig

trousers and woollen socks. A trained Firefighter was not used for this test so that there would not be any influence of "conditioning" to radiant heat exposures.

The Subject stood at various distances from a severe radiant heat source - estimated at or above level 3: emergency conditions i.e, temperature in excess of 800 °C and radiant heat source more than 80 kW/e. Reports were taken from the Subject as to feelings of discomfort at various distances - after 20 s at 10 m from the fire; after 20 s at 5 m from the fire and after 20 s at 3 m from the fire. No discomfort at all was felt at 10 m and at 5 m, but at 3 m the Subject reported a certain amount of discomfort from heat being felt through the helmet visor (this was a long visor covering the full face). "This indicates the extent of protection given by current firefigfhting equipment."

A.6.5 Fatigue and heat exposure

That equipment designed to protect against heat also causes fatigue because of the bulk and insulation properties is of concern particularly because firefighters rarely encounter the very high temperatures seen in emergency conditions as defined in 6.3. Consequently firefighters consider that for some 90 % of the time in the working environment the equipment supplied, which is designed to protect against emergency conditions, adds to rather then relieves from the stress of the environment. It was not possible to find information that readily correlated stress levels with environmental conditions. However, Figure A.15 shows the recommended maximum exposure time from 25 °C to 43 °C for unimpaired mental performance. This would suggest that if the temperature within the firefighting protective equipment, helmet for example, rises to about 40 °C, then mental performance will be impaired after about 20 min.



Key

X Exposure time, (min) Y_1 WBGT (^{o}C) Y_2 WBGT (^{o}F)

Figure A.15 — Temperature vs maximum exposure time for unimpaired mental performance (Prof. J. W. Sutherland. Michigan Inst. Technology, 2002, lecture on Industrial Safety)

A.7 Conclusions

The purpose of this review is to provide the evidence to support the information given in the main report. Thus, a series of conclusions was considered unnecessary.

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