

Chapter 2

Basics of the Biomechanics of Brain Injury

2.1 Introduction

Brain injury is a major public health problem and is commonly seen in falls and automotive crashes as well as in other environments, such as contact sports, military action, and assaults. Although the brain is protected by the skull, it can be injured in relatively low-speed impacts, such as in American football. Statistics on traumatic brain injury (TBI) provided by the National Center for Injury Prevention and Control reveal that, in 2010, there were over 50,000 deaths due to TBI and that TBI was diagnosed in more than 280,000 hospitalizations and in 2.2 million emergency room visits. Falls are the leading cause of TBI, especially among the youngest and oldest age groups, while motor vehicle crashes were the third leading cause of TBI (14 %). The various causes are shown in Fig. 2.1.

Because of the fact that effective treatment of TBI, even mild TBI (mTBI), is generally not available, prevention of TBI should be a top priority, and biomechanics can play a leading role in this effort. Biomechanical research on TBI has been carried out in the USA for over 75 years (see Chap. 1), and there is some information about the response of the brain to impact and its tolerance. However, there is still a divided opinion on the causes of TBI because it is not clear whether linear acceleration or angular acceleration/velocity is the principal cause of TBI. It is more than likely that both forms of acceleration play a role in causing TBI since in a head impact both forms of acceleration are present and linear acceleration increases monotonically with angular acceleration.

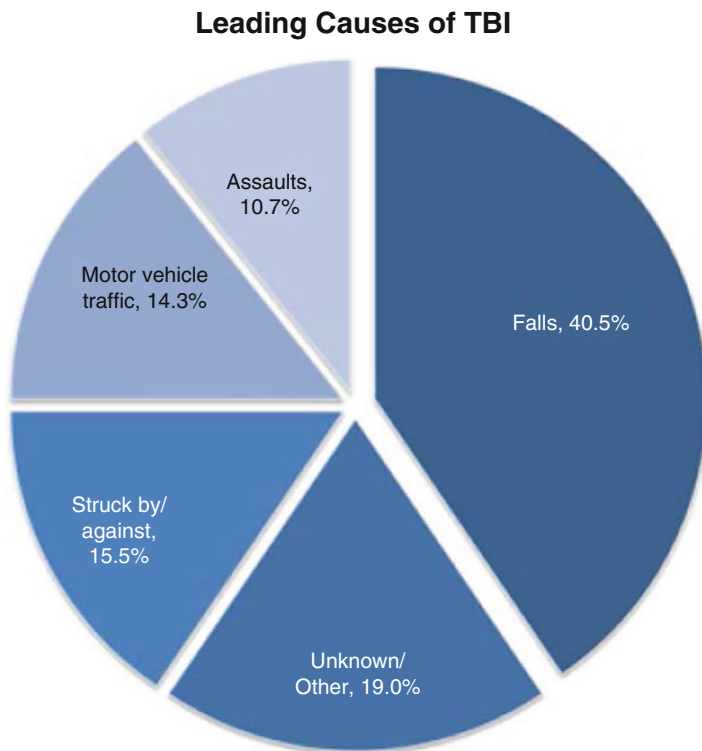


Fig. 2.1 Various causes of traumatic brain injury in 2010 (source: National Center for Injury Prevention and Control, http://www.cdc.gov/traumaticbraininjury/get_the_facts.html)

2.2 Anatomy of the Head and Brain

No biomechanical discussion is complete without some understanding of the anatomical structures involved. The focus in this chapter is on the head and brain. Even though the brain is part of the central nervous system which encompasses the spinal cord as well, the anatomy of the spinal cord is deferred to a later chapter. For the head, it is particularly important that reader has some basic information of its complex anatomy, especially that of the brain. The head consists of the scalp, skull, face, meninges, and brain.

The scalp is the outer covering of the head and is composed of the hair, skin, fascia, muscles, and periosteum. Its thickness is from 5 to 7 mm and it moves as a single layer. An embalmed scalp is thicker because some of the embalming fluid is retained in it.

The skull is the strong box or vault protecting the brain. It consists of eight bones that are fused into a single shell in the mature adult. Its thickness varies from 4 to 7 mm, and it is composed of three layers—cortical bone in the inner and outer layers sandwiching a layer of spongy or trabecular bone in the center. This spongy

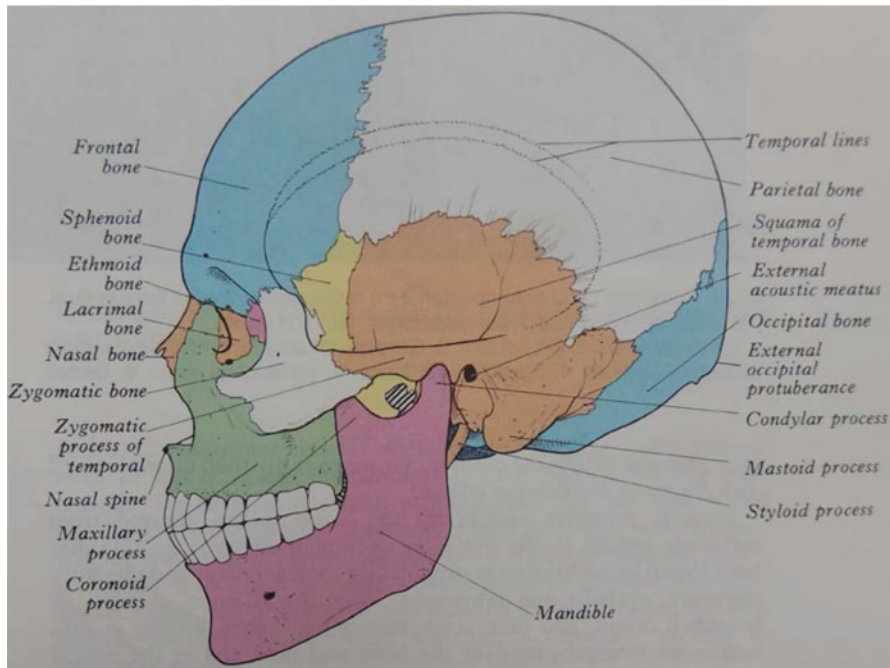


Fig. 2.2 Bones of the skull and face (taken from Gray (1995)). Reprinted from Gray's Anatomy: The Anatomical Basis of Medicine and Surgery, 38th edn. by Gray, (Churchill Livingstone), 1995, with permission from Elsevier

bone layer is also known as the diploe. The facial bones make up the rest of the skull and are fused to the skull. There are 14 facial bones, 13 of which are fused or attached to the skull to form the face which is defined as an area of the head between the forehead and the mandible or lower jaw. The mandible is free to move and articulates about the temporal mandibular joint (TMJ) to enable chewing. At the base of the skull, there is a thick cylindrical bone around the foramen magnum, the opening for the spinal cord to pass through. Figure 2.2 is a drawing of the skull, showing all the bones of the skull and some of the bones of the face. The borders separating the skull bones are called sutures which do not close until about the age of 30. Younger skulls have some cartilage on either side of the sutures.

Under the skull are three meninges or membranes that cover the brain. The outermost layer is a double-layered membrane called the dura mater or simply the dura. It is the thickest and toughest of the three meninges. The outer layer of the dura is adherent to the inside surface of the skull in the adult, while the inner layer is mostly fused to the outer layer except down the midline of the skull where the two layers are separated to form the superior sagittal sinus—a cavity that collects venous blood from the brain for drainage back into the heart. The meninges are shown in Fig. 2.3 which also shows the bridging veins that transport the blood from the brain into the superior sagittal sinus. According to Haines et al. (1994), below the dura are two thin layers of cells called border cells. The outer layer is called the dural border cell and the inner layer is called the arachnoid border cell.

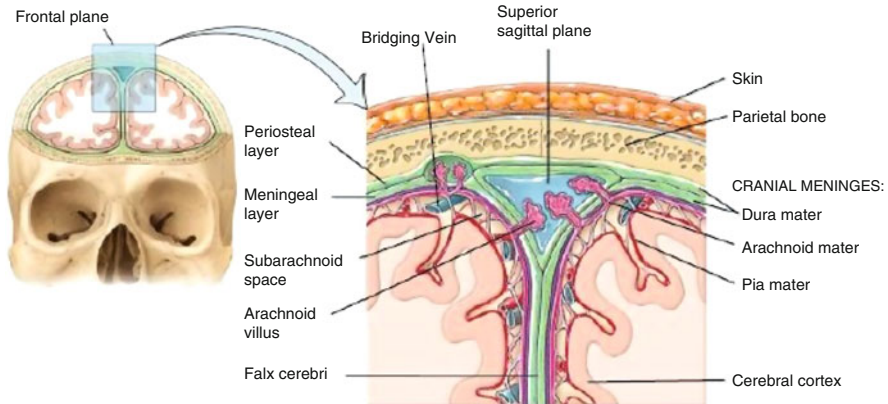


Fig. 2.3 The cerebral meninges, the superior sagittal sinus and bridging veins that bridge the CSF layer and transport the blood from the brain into the superior sagittal (taken from Tortora and Nielsen (2013)). Republished with permission of John Wiley and Sons Inc., from G.J. Tortora, M.T. Nielsen, *Principles of Human Anatomy*, 13th edn. Chapter 18, 2013, permission conveyed through Copyright Clearance Center, Inc.

Under the border cells is the second membrane, the arachnoid, which is a very thin layer but which contains a basement membrane that is impervious to water. Below the arachnoid is a layer of cerebral spinal fluid (CSF) in the subarachnoid space (SAS) which varies in thickness depending on age. There are trabeculae (collagenous soft tissue) which tether the arachnoid to the pia, the third membranous cover of the brain. The pia is extremely thin and is almost transparent. So the surface of the brain is visible through the pia which appears to wrap tightly around the brain, investing itself into the sulci of the brain. There is also a basement membrane associated with the pia. The only region that allows the brain to move relative to the skull is the CSF layer because the border cells between the arachnoid and dura prevent sliding in those layers. A more detailed diagram depicting the three meninges is provided by Fig. 2.4.

Another aspect of brain mobility in the CSF region is the role of the trabeculae within it. Older mathematical models of the brain simulated the CSF as a layer of fluid with no shear resistance, ignoring the presence of the trabeculae. Jin et al. (2006, 2007) studied the mechanical properties of the pia-arachnoid complex (PAC) and found that the trabeculae were capable of resisting tension applied in the normal direction to the membranes and of resisting shear in the plane of the membranes. That is, the amount of sliding in the CSF is governed by the trabeculae and not by the CSF, and brain models should simulate the CSF layer as a low shear modulus solid.

2.2.1 *Anatomy of the Brain*

As mentioned above, the brain is (a major) part of the central nervous system (CNS). It consists of a network of neurons and supporting tissue that form the

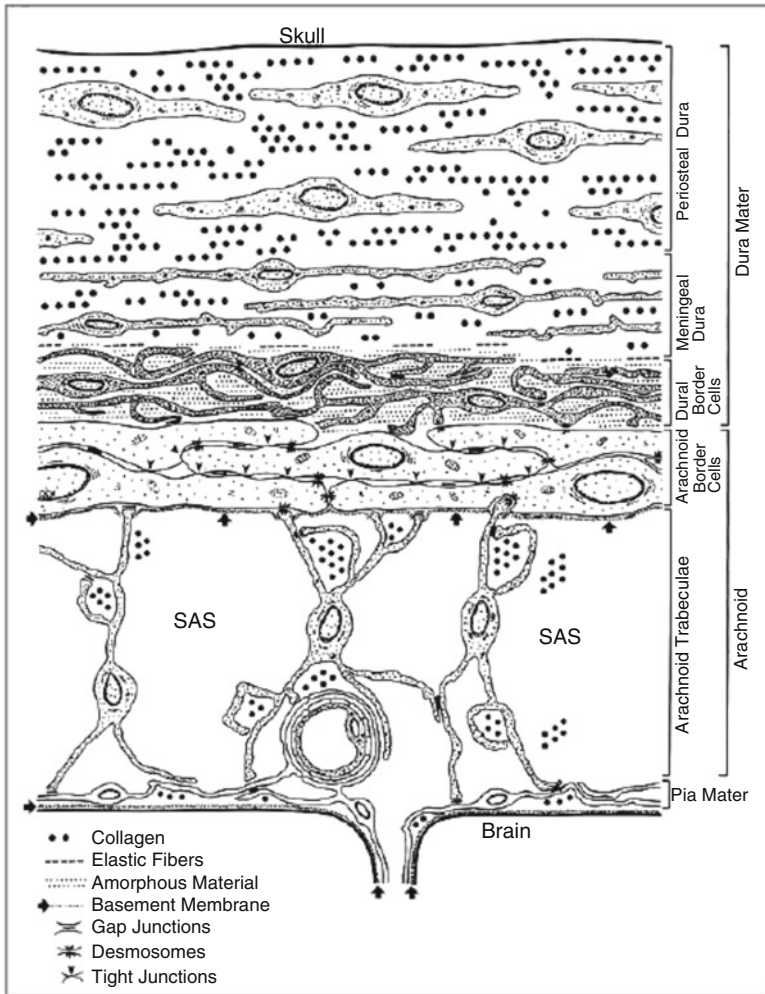
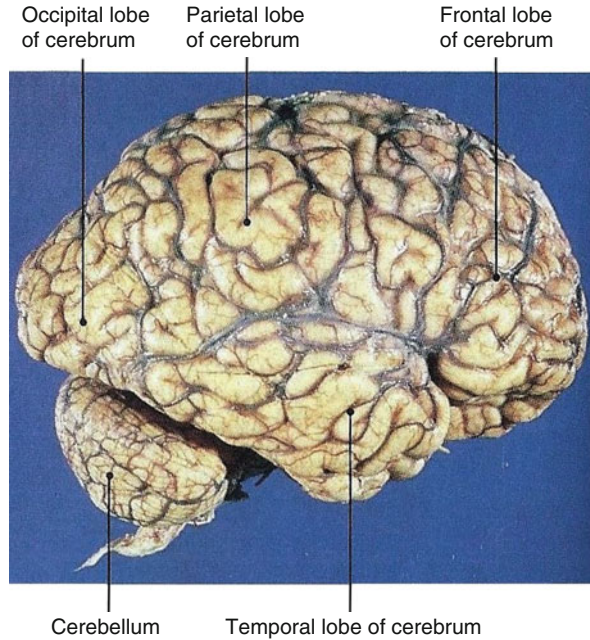


Fig. 2.4 Details of the three cerebral meninges, based on a study by Haines (1991). Republished with permission of John Wiley and Sons Inc., from D.E. Haines, *On the question of subdural space*, *The Anatomical Record: Advances in Integrative Anatomy and Evolutionary Biology*, 1991, permission conveyed through Copyright Clearance Center, Inc.

control center for the body. Functionally, it is divided into gray and white matter. The cells of the CNS are neurons which make up the gray matter, and the extension of the neurons or axons makes up the white matter. The gray matter is found mostly on the outside of the brain. The main components of the brain are the forebrain or cerebrum, the midbrain, and the hindbrain. The cerebrum occupies a large volume of the skull and consists of two hemispheres that are separated by a membrane called the falx cerebri which is actually an extension of the dura. Each half of the cerebrum can be divided into four lobes as shown in Fig. 2.5. The frontal lobe is

Fig. 2.5 The brain. The cerebrum and the hindbrain are visible. Approximate locations of the lobes of the cerebrum are identified (taken from Carola et al. (1992)). Republished with permission of McGraw-Hill Education, from R. Carola, J.P. Harley, C.R. Noback (eds.), *Human Anatomy & Physiology*, 2nd edn., 1992; permission conveyed through Copyright Clearance Center, Inc.



right behind the frontal bone (forehead) and below it is the temporal lobe. The parietal lobe is behind the frontal lobe, and the occipital lobe is behind the parietal lobe. The brain weighs about 1.36 kg (3 lb) or constitutes about 2 % of body weight. It is 165 mm long and 140 mm wide. The center of gravity of the head is located just above the horizontal Frankfort plane and just anterior to the auditory meatus. In Fig. 2.6, the x -axis is at the level of the Frankfort plane, and the origin is at the center of the auditory meatus (ear canal).

There is a cavity in each hemisphere called the lateral ventricle. It is filled with CSF and communicates with the third and fourth ventricles. The midbrain is composed of the various parts of the thalamus, including the hypothalamus. The hindbrain is made up of the cerebellum, pons, and medulla oblongata, the most caudal part of the brain stem. The cerebellum is covered by a membrane called the falx cerebelli, a part of the dura that separates it from the occipital lobe. It is required for fine movement, motor corrections, and reflex modifications. The third ventricle is a small cavity in the midline of the forebrain beneath the lateral ventricles. It connects with the fourth ventricle which is located behind the pons. The walls of the ventricles are lined with ependymal cells that produce CSF which circulates from the lateral ventricles to the third and fourth ventricles to the CSF space between the cerebral meninges. The CSF also surrounds the spinal cord, extending down to the lumbar level.

The brain requires a constant and ample supply of oxygen. As a result, there are numerous blood vessels in the brain. The blood vessels shown in Fig. 2.7 are the cerebral arteries without the veins. The preparation was made possible by injecting

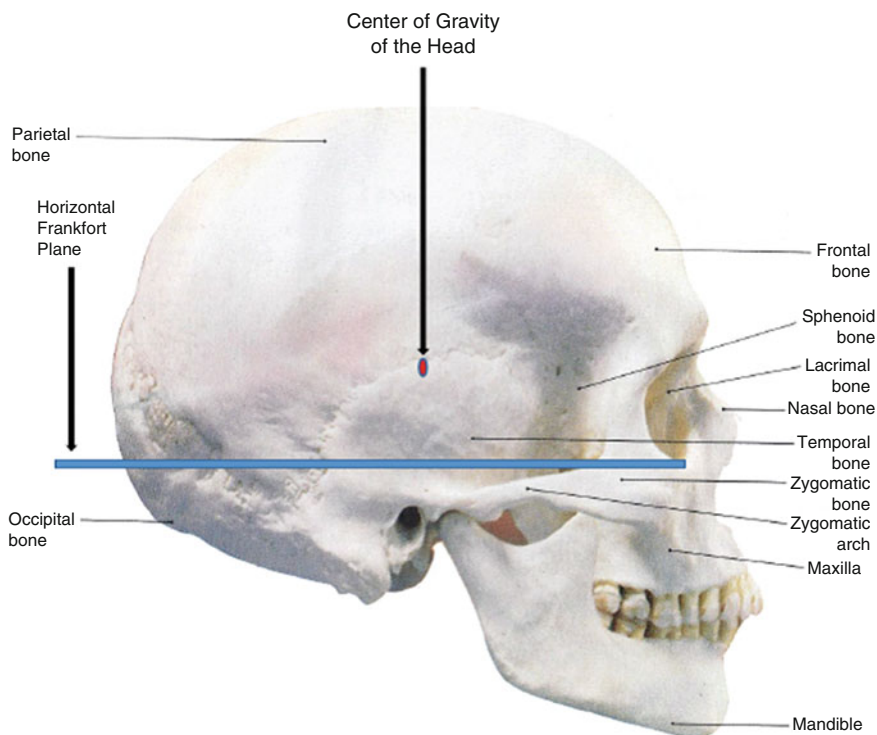


Fig. 2.6 The approximate location of the center of gravity (cg) of the head is in the midsagittal plane slightly anterior to the auditory meatus and about 3 cm above the Frankfort plane which is at the level of the inferior border of the orbit or eye socket. The illustration of the skull was taken from Carola et al. (Eds.), 1992, *Human Anatomy & Physiology*. Republished with permission of McGraw-Hill Education, from R. Carola, J.P. Harley, C.R. Noback (eds.), *Human Anatomy & Physiology*, 2nd edn., 1992; permission conveyed through Copyright Clearance Center, Inc.

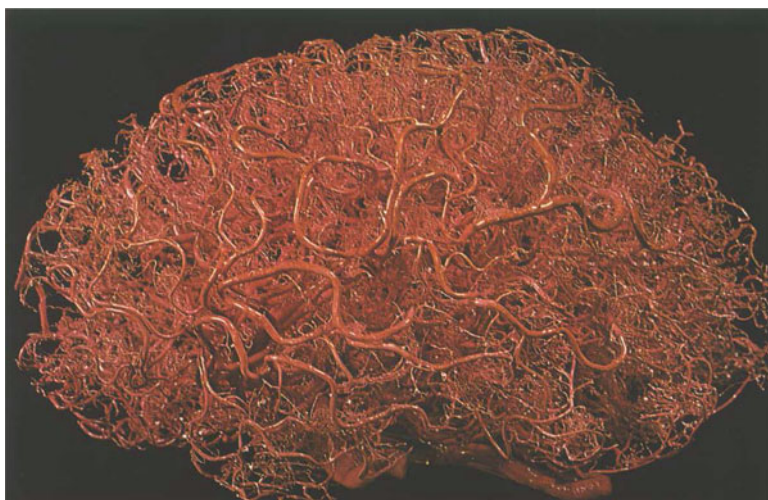


Fig. 2.7 Arteries of the human brain (taken from McMinn and Hutchings (1977)). Reprinted from R.M.H. McMinn, R.T. Hutchings, *Color Atlas of Human Anatomy* (Year Book Medical Publishers), 1977, with permission from Elsevier

a rubberized solution into the cerebral arteries and allowing it to set. After which, the rest of brain is dissolved in an acid solution. Thus, the vessels shown are slightly smaller than the actual vessels because the arterial walls have also been dissolved. The stiffness of the brain is due largely to that of the arteries and veins because brain material is extremely soft and weak.

2.2.2 Histology of Brain Cells

The CNS is made up of neurons and neuroglia (supporting cells). Neurons or nerve cells are the functional cells of the CNS with the ability to sense external input as well as activate muscle cells. They encode information and transmit it rapidly to other neurons or non-neuronal cells, in some cases, over large distances, using minute electrochemical signals. There are many types of neurons, as shown in Fig. 2.8, but they all have in common a soma or cell body containing a nucleus, a single axon for the transmission of signals, and one or more dendrites that are neuronal receptors. Figure 2.9 shows a typical neuron and its components.

Fig. 2.8 Various types of neurons. Legend: cb stands for cell body and ax stands for axon

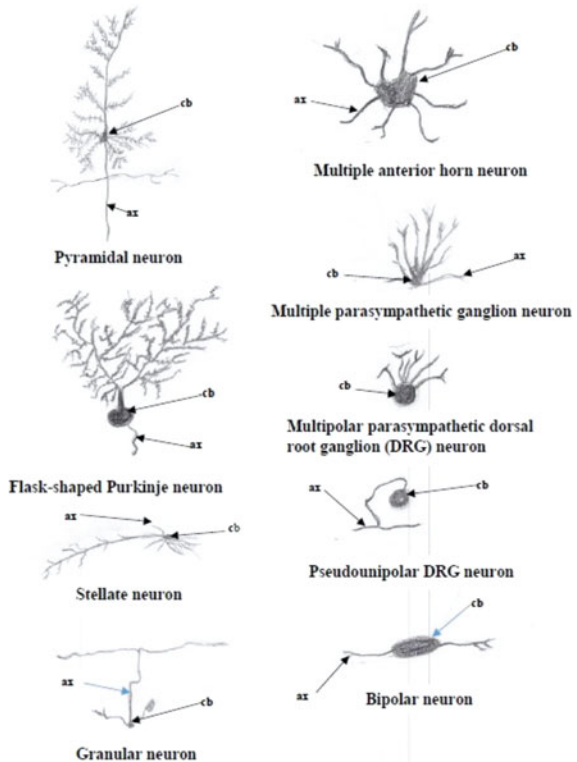


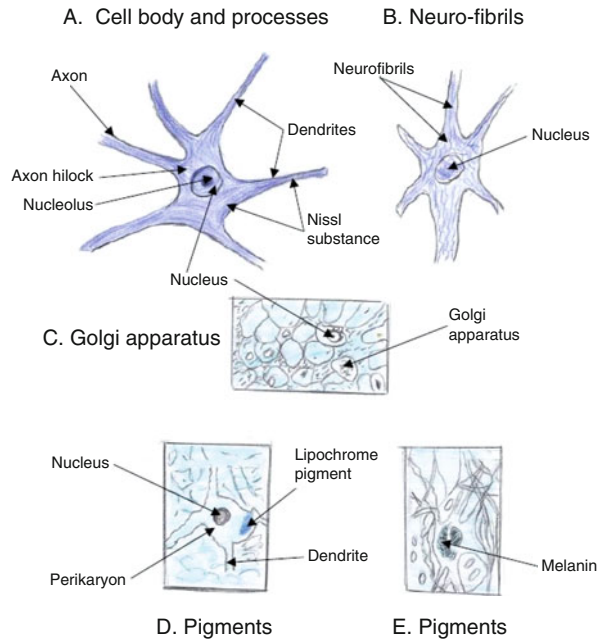
Fig. 2.9 A typical neuron and its components

Figure 2.9A refers to Nissl substance or Nissl bodies which are membrane-bound ribonucleoproteins that are distinctive in shape and abundant in the cell body and dendrites. Their main function is protein synthesis for the cell. Axons do not contain Nissl bodies. As shown in Fig. 2.9B, neurofibrils are found in all neurons. They are delicate threads running in every direction through the cytoplasm of a neuron and extend into the axon and dendrites. They consist of neurofilament bundles and neurofibrils. The subunits of neurofibrils are neurofilaments which are 7.5–10 nm in diameter. There are also neurotubules and microtubules (25 nm in external diameter) that provide rapid transport of protein molecules synthesized in the cell body via the dendrites and axon. Figure 2.10 shows the microstructure of microtubules which are made up of tubulin molecules. Since protein synthesis is not an axonal function, the axon has no Nissl bodies. The axon is uniformly cylindrical, and its axoplasm contains many organelles, such as mitochondria, microtubules, microfilaments (6 nm in diameter), and neurofilaments. The microfilaments are paired helical chains of actin which can contract, providing a means for intra-axonal transport of protein molecules. The neurofilaments provide the axon with mechanical strength and outnumber microtubules by a considerable amount. Some axons are myelinated, that is, covered by a myelin sheath which is a lipid protein that wraps around the axon in multiple layers. The sheath is discontinuous and the area of discontinuity is called the node of Ranvier (Fig. 2.11). Biomechanically, the node may be a weak spot for the axon where it may break under tension, resulting in diffuse axonal injury (DAI). This injury will be discussed in detail later on in the chapter. Dendrites are processes extending

Fig. 2.10 (A–D)
Microstructure of a
microtubule (taken from
Alberts et al. (1994)).
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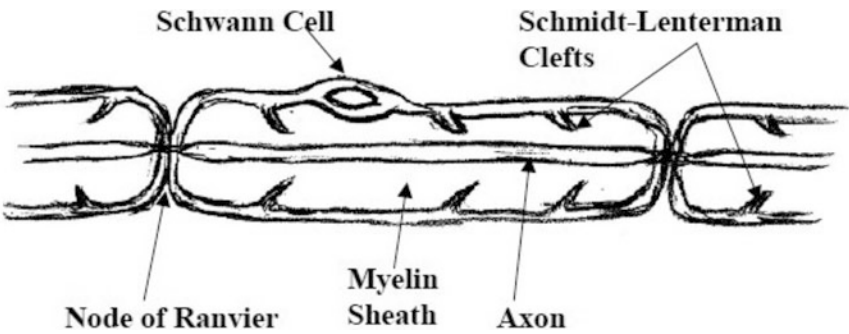
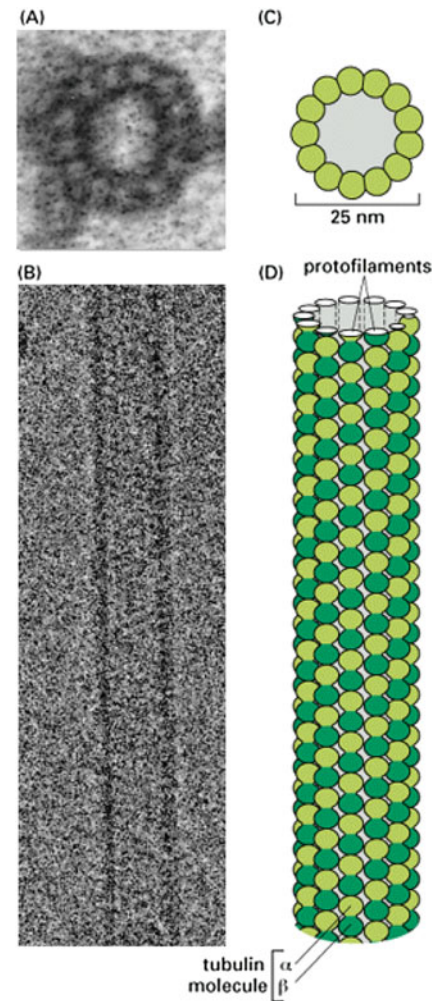


Fig. 2.11 The node of Ranvier of a myelinated axon

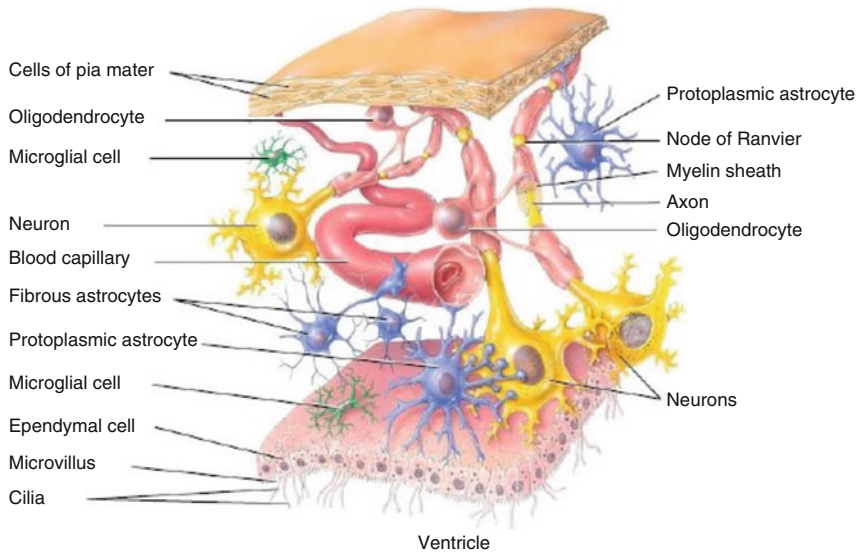


Fig. 2.12 The four main types of neuroglia which are supporting cells for the CNS (taken from Tortora and Nielsen (2013)). Republished with permission of John Wiley and Sons Inc., from G.J. Tortora, M.T. Nielsen, *Principles of Human Anatomy*, 13th edn. Chapter 16, 2013, permission conveyed through Copyright Clearance Center, Inc.

from the cell bodies to increase its receptive surface area. Thus, dendrites, together with the soma, can receive excitatory or inhibitory signals from other axons to set off an action potential for the neuron or prevent it from occurring.

Neuroglia are supporting cells of CNS neurons and are found between these neurons. The four main types of neuroglia are astrocytes, oligodendroglia, ependymal cells, and microglia. Figure 2.12 is an illustration of these cells and how they support the neurons. Astrocytes are branched stellate cells and are the largest of the neuroglia. They surround the capillaries of the brain and form part of the blood-brain barrier. The cytoplasm of astrocytes contains glial fibrillary acidic protein (GFAP), the concentration of which increases if there is a proliferation of astrocytes due to brain injury. Figure 2.13 is a schematic of the blood-brain barrier and of the role of astrocytes. Oligodendrocytes produce myelin for the axon, while the ependymal cells produce and monitor the CSF in the CNS. Microglial cells are small, have little cytoplasm, and have a few processes. In the presence of lesions or infection, they enlarge, become mobile, and take on the role of scavenger cells (phagocytes).

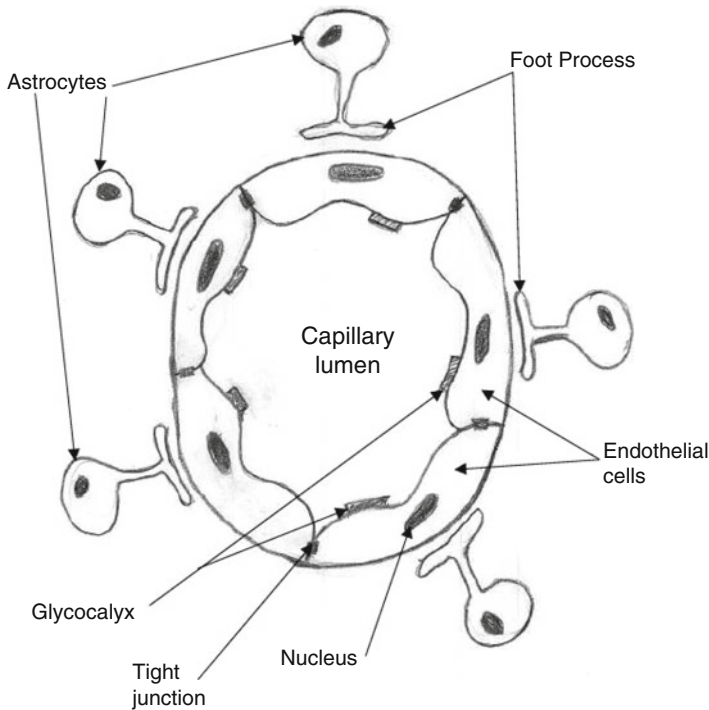


Fig. 2.13 The role of astrocytes in the blood-brain barrier. Orthogonal arrays of particles in the foot process of the astrocytes along with the tight junctions in the endothelial cell layer may play a role in the prevention of diffusion of molecules from the capillaries into the brain

2.3 Types of Head Injury

According to Ommaya (1985), there are four main types of head injury:

1. Injuries to the scalp
2. Injuries to the skull
3. Extracerebral bleeding (focal or diffuse)
4. Brain tissue damage (neural and/or vascular)

Scalp lacerations are considered minor injuries, but there can be a lot of bleeding because the dense subcutaneous tissue of the scalp prevents constriction and retraction of the arteries. There are many types of skull fractures varying from linear fractures to penetrating fractures. Gurdjian et al. (1950) described the extensive research done in the 1940s investigating the mechanism of skull fracture. Since bone is weak in tension, the fractures are explained by the development of tensile stresses in the skull as it is being hit. The impacted site bends inward and the outer table is in compression, while the adjacent areas of the skull bend outward. Thus, the outer table fractures at some distance from the site of impact in the outbended

skull. During rebound, the impact site bends outward and the outer table is in tension. The result is that a linear fracture would start at a site remote from the impact site but would travel toward the impact site at the end of the impact. Higher-energy impacts cause stellate patterns of fracture as well as depressed fractures and crushing of the skull. Extracerebral bleeding is due to arterial rupture under the skull but above the dura. When an artery, such as the middle meningeal artery, is ruptured under the skull, the epidural hematoma that forms separates the dura from the skull and rapidly increases intracranial pressure. If not treated promptly, death will result. Inbending of the skull at the location where the artery is running under it and above the dura is the suspected cause.

2.3.1 Brain Tissue Damage

Brain tissue damage can be broadly divided into four major categories:

1. Concussion or mild traumatic brain injury (mTBI)
2. Contusion or bruising of the brain
3. Intracerebral hemorrhage or intracranial bleeding
4. Brain laceration or tearing of the brain

2.3.1.1 Concussion

Concussion is sometime defined as a mild form of brain injury (mTBI) characterized by a temporary loss of brain function. There does not have to be a loss of consciousness although concussion severity is graded by the length of unconsciousness. However, in more severe forms of brain injury, concussion is also one of the leading symptoms. Other measures of concussive severity are post-traumatic amnesia and confusion. There are many signs and symptoms of concussion which occur right after the injury. Physical signs include headache, dizziness, vomiting, and nausea as well as visual disturbances, tinnitus (ringing in the ears), sleep disturbance, and occasionally seizures. Cognitive symptoms include disorientation, inability to focus, and slowed reaction time. Psychological symptoms include irritability, moodiness, memory problems, and lethargy. In mTBI, physical concussive symptoms generally resolve in a matter of weeks, while other symptoms may persist.

In most civilian situations, concussion is brought about by rotational motion of the head, such as a punch in boxing, where the linear acceleration is relatively low, while the angular acceleration is relatively high. In the military, blast waves from explosions can cause concussion, and it is suspected that the mTBI is due to the pressure wave passing through the brain. This mechanism has yet to be confirmed. In both cases, there is diffuse axonal injury due to the disruption of axons, even in mTBI. Tensile axonal loads break the axon and the microtubules inside it. The flow

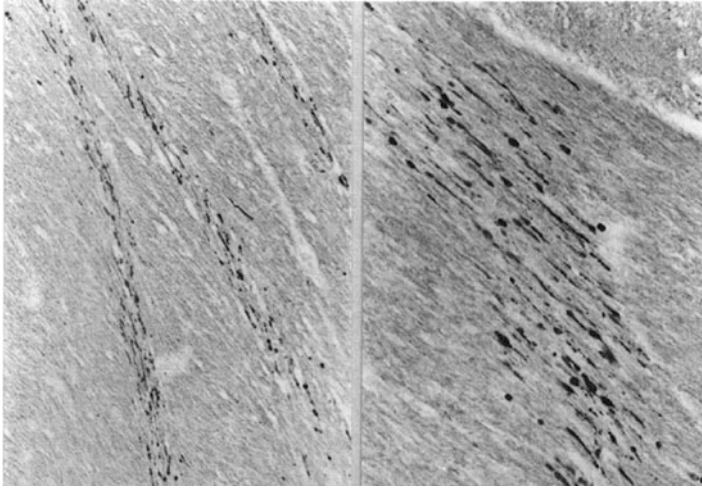


Fig. 2.14 Diffuse axonal injury in the human corpus callosum. Dark lines are swollen axons, and black circles are retraction balls, made visible by means of β -APP staining (taken from Gentleman et al. (1993)). Reprinted from S.M. Gentleman, M.J. Nash, C.J. Sweeting, D.I. Graham, G.W. Roberts, β -Amyloid precursor protein (β APP) as a marker for axonal injury after head injury. *Neuroscience Letters*, 160, 139–144, 1993, with permission from Elsevier

of axonal transport of protein products is stopped. By the use of antibody staining techniques, the location of the tear can be identified by the bulb of axonal transport material at the end of the broken axon. This is known as a retraction bulb or ball and is the hallmark of DAI (Strich 1956). It should be noted that DAI is seen over a large area of white matter and is also seen in more severe forms of brain injury. Figure 2.14 is a micrograph of DAI found in the corpus callosum of a human brain. Note that DAI does not appear in the axon immediately after the injury as it takes time for the retraction balls to form and for the axons to swell. Although it is assumed that this may take a day or so, there is evidence provided by Hortobagyi et al. (2007) that DAI can be seen in brains of trauma victims who survive for 35 min after the injury. This has been confirmed by Morrison and MacKenzie (2008). It is also obvious that, in the human, a diagnosis of DAI can only be made after death.

2.3.1.2 Contusion

Bruising of the brain is called a cerebral contusion which is a focal injury. It occurs under the site of impact (the coup site) as well as on the side opposite to the impact (the contrecoup site). The capillaries in the cerebral cortex (gray matter) are broken either by the pressures generated by the impact or by relative motion of the brain with respect to the uneven surfaces of the interior of the skull, particularly on the surfaces of the frontal and temporal lobes. The pia is not torn in contusive injuries.

The pressure mechanism is used to explain the coup-contrecoup phenomenon. Positive pressure at the site of impact and negative pressure on the side opposite to

the impact are the mechanisms causing the observed contusive injury. The symptoms are similar to a concussive injury and their severity is dependent on the extent of the contusion.

2.3.1.3 Intracerebral Hemorrhage

Intracerebral hemorrhage or intracranial bleeding is due to the rupture of blood vessels inside the brain. In the absence of impact injury, the most common cause is a hemorrhagic stroke. In an impact situation, weak blood vessels rupture due to the impact, resulting in intracerebral hemorrhage. Arterial ruptures can rapidly increase intracranial pressure and cause death if there is no surgical intervention.

2.3.1.4 Brain Laceration

Brain lacerations occur in severe head impacts which cause the brain to be mechanically torn apart. Usually, the pia and arachnoid are torn at the injury site and lacerations can be thought of as severe form of brain contusion. Skull fractures are commonly associated with this injury, and blood vessels are usually ruptured, resulting in intracerebral hemorrhage and the risk of increased intracranial pressure. The collection of blood in the brain or on the surface of the brain is known as a mass effect or a hematoma. It is detected on computer tomography scans which frequently show a midline shift of the brain as the hematoma pushes the brain to the opposite side.

2.4 Theories of Brain Injury Mechanisms

Based on the years of research on brain injury, dating back to 1766, the following mechanisms have been proposed (Pudenz and Shelden 1946):

1. Positive pressure mechanism
2. Negative pressure mechanism
3. Pressure gradient mechanism
4. Rotational mechanism

The work of Pudenz and Shelden (1946) was aimed at demonstrating brain motion during head impact, but the authors went into a long discussion of the above mechanisms before describing the brain motion they documented with high-speed film for impacts to monkey heads that had their skull caps removed and replaced by a Lucite calvarium (Shelden et al. 1944). The biomechanical explanations provided in this early paper on brain injury may not be totally accurate because the membranes of the brain were no longer intact, but it is of historic interest.

Brain injury due to pressure mechanisms is attributed to translational motion of the brain, i.e., linear acceleration. When there is an impact to the head, the in-bending of the skull and the acceleration of the head due to the impact both

Table 2.1 Average impulse (in psi-s) for different degrees of concussion in dogs for all 72 tests (Gurdjian et al. 1954)

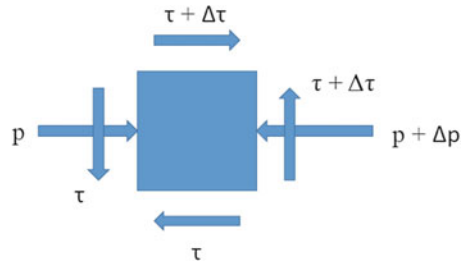
	No concussion	Threshold concussion	Mild concussion	Severe concussion
	0.038	0.153	0.085	1.060

contribute to the development of compression or a positive pressure at the impact site. This pressure travels as a wave across the brain and is reflected off the skull on the contrecoup site, creating a negative pressure there. The pressure mechanism of concussion was demonstrated by Gurdjian et al. (1954) who developed a method of applying pressure to the brain of an anesthetized animal (dog) without having to strike it on the head with an impactor. These authors were roundly criticized by the press for cruelty when Gurdjian was shown in the papers with a dog's head in one hand and a hammer in the other. They invented the fluid percussion method which applied a pressure pulse of air to a small area of the dura using a specially designed valve or dropping a weight onto a column of water in contact with the dura. He achieved a range of impact durations of less than 1 ms to as high as 120 ms. The peak pressures ranged from 4 to 74 psi (27.5 to 509.5 kPa). The severity of the injury was divided into four groups: no concussion, threshold concussion, mild concussion, and severe concussion. They found that concussion occurred for impacts with high pressures and short duration or with low pressures and long durations. We can take the data and go a step further by defining impulse as the product of peak pressure and pulse duration and calculating it for all 72 tests performed by Gurdjian et al. (1954). The averaged results are shown in Table 2.1 for the four conditions. The threshold impulse turned out to be the highest, and if that is put aside, there is an increase in impulse with injury severity – 0.038 psi-s for no concussion, 0.085 psi-s for moderate concussion, and 1.060 psi-s for severe concussion. The passage of pressure waves across the brain has been measured, and the coup-contrecoup phenomenon causing concussion is accepted by the medical community. The implication of this hypothesis is that pressure damages the neurons in some way and causes dysfunction described above in Sect. 2.3.1.1, in the absence of head rotational motion. Exposure of the head to blast overpressure due to explosions, such as the detonation of improvised explosive devices (IED), is a major cause of mTBI sustained by returning US veterans from the Middle East. The mechanism of injury due to a pressure wave at the cellular level needs to be found before effective preventative measures can be implemented to protect our soldiers.

When there is a pressure wave moving through the brain, a pressure gradient exists. In Fig. 2.15, the pressure on the low-pressure side of a small element of the brain (left side) is p , while that on the high-pressure side is $p + \Delta p$. This unbalance in normal force on the element results in the generation of a shear stress $\Delta \tau$ above whatever shear stress, τ , that might exist on the faces of the element. This shear tends to distort the element and cause injury to the brain.

Of course, if the brain undergoes rotational motion, then large shear stresses develop as a result of the rotation. This is the basis for the rotational mechanism of brain injury, originally proposed by Holbourn (1943) who developed a simple

Fig. 2.15 Pressure gradient produces shear stress



physical model of the brain, using gel, to explain the rotational mechanism of injury. Holbourn's argument that rotation causes the brain to deform in shear and thus become injured is sound, but the argument that pressure cannot cause injury, based on the fact that the nerves continue to conduct signals under immense hydrostatic pressures, is flawed. In an impact, the pressure wave applies the load at very high rates and acts as a shock to the brain. This shock effect is quite different from hydrostatic pressure. However, severe rotational motion in the absence of a direct impact can result in concussion, as shown by Gennarelli and Thibault (1982). Angular accelerations of the order of $100,000 \text{ rad/s}^2$ were applied to the heads of rhesus monkeys, causing the development of DAI as well as acute subdural hematoma in their brains.

Ommaya and Hirsch (1971) proposed a scaling law for head acceleration between the human and different species of experimental animals. This law is given by Eq. (2.1) below:

$$\alpha_h/\alpha_r = (m_r/m_h)^{2/3} \quad (2.1)$$

where α_h is the human head angular acceleration, α_r is the rhesus head angular acceleration, m_h is the human brain mass, and m_r is the rhesus brain mass.

Then, for $m_h = 1360 \text{ g}$ (g), $\alpha_r = 100,000 \text{ rad/s}^2$, and $m_r = 70\text{--}100 \text{ g}$, the equivalent human head angular acceleration would be between 13,800 and 17,500 rad/s^2 . This level of angular acceleration is difficult to achieve in a human for a purely rotational (non-contact) head impact without some serious injury to the neck or the head-neck junction. This is not to say that the study was not worthwhile. Instead, it points to the fact that both linear and angular acceleration are responsible for concussion. Note that when the head impacts a surface of any kind, such as a windshield, there is not only linear deceleration but also a very high concomitant angular acceleration that does not involve whipping of the head or the involvement of the neck. Figure 2.16 shows the relationship of linear and angular acceleration of a helmeted dummy head subjected to a frontal impact against a foam-covered rigid surface (King et al. 2003).

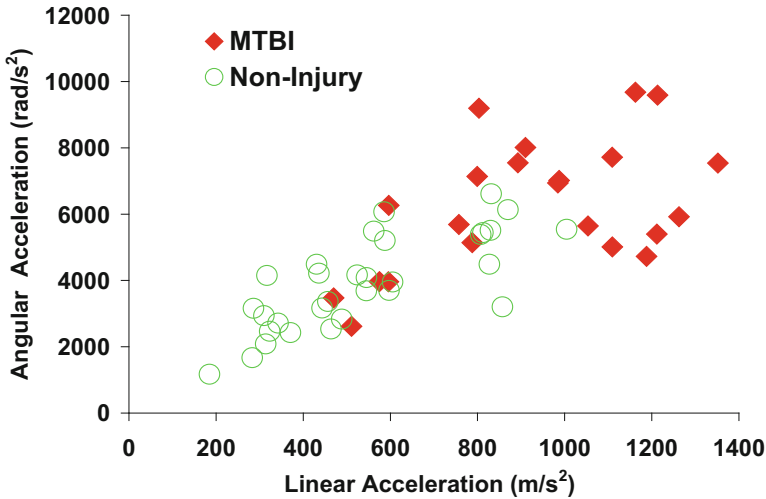
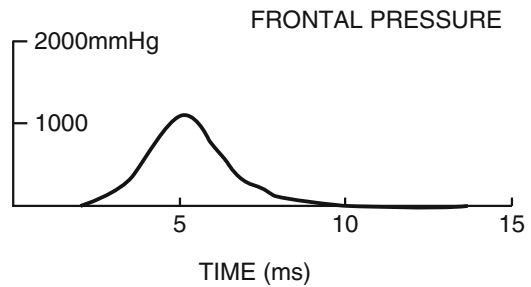


Fig. 2.16 In head impacts, linear and angular acceleration usually increase monotonically

Fig. 2.17 Intracranial pressure data from a frontal impact to a cadaver head (Nahum et al. 1977)



2.5 Mechanical Response of the Head and Brain

Early researchers in head injury measured linear head acceleration to define the impact response of the head. Accelerometers were relatively light and small and could be attached conveniently to the skull, either directly to the skull or using a mount that is held to the skull with bone screws. Pressure sensors were available to measure intracranial pressure in living animals but were not useful in cadaveric brains which were embalmed. In unembalmed or fresh cadavers, brain tissue degrades quickly after death, and the measured pressures would be meaningful only if the cadavers were tested soon after death. The general rule of thumb is that the cadaver should be kept in a 4 °C cooler while under preparation and tested no more than two weeks after death and that the total time outside the cooler should be less than 24 h. Intracranial pressure data were provided by Nahum et al. (1977), as shown in Fig. 2.17. Attempts were made in the twentieth century to visualize brain and skull response during impact (Shatsky et al. 1974; Nusholtz et al. 1984), but

they were largely unsuccessful or provided little useful information on brain motion. Thus, the response of the head was defined by its acceleration response to impact. The response is dependent on the stiffness and the geometry of the surface the head impacts, and a large volume of published information is available. One of the aims of determining mechanical response is to build a surrogate or dummy that can be used in the design of automobiles, helmets, and other protective devices. In this case, data derived from impacts against padded surfaces are not useful because the stiffness and other characteristics of the padding are difficult to specify or may change with repeated impacts and the same padding is not necessarily available to all researchers. For this reason, skull response to impacts against a rigid surface provides the “standard” data that can be used by all researchers. Figure 2.18 shows cadaveric head impact data of the forehead against a rigid surface and the response of the Hybrid III head to a frontal impact (Mertz 1985).

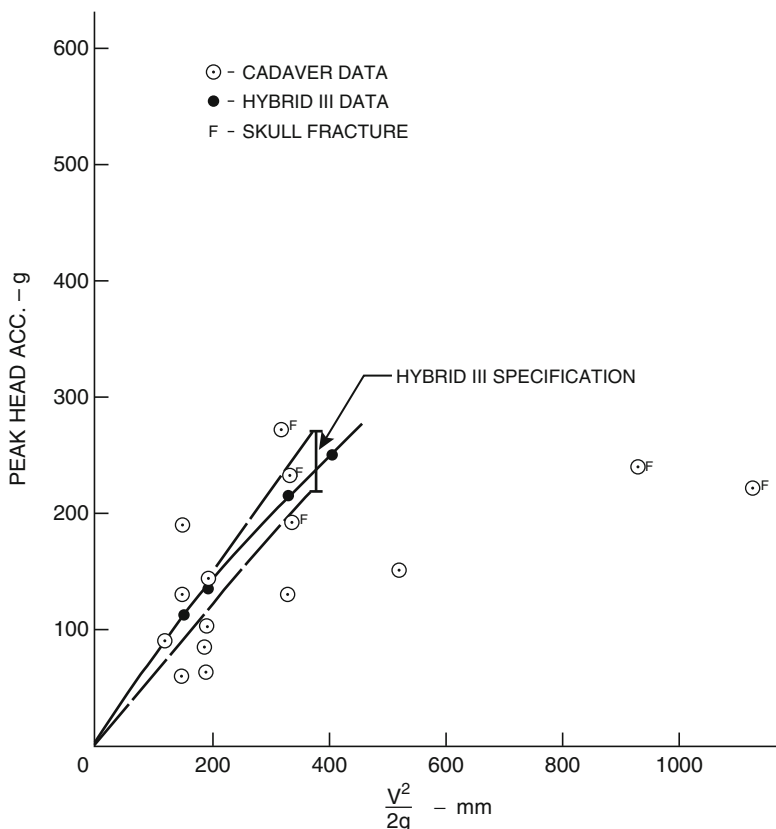
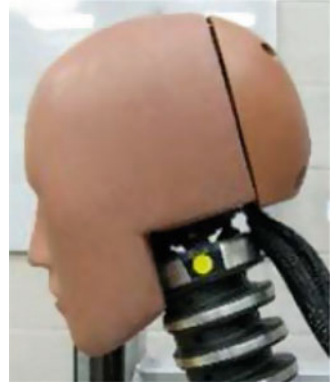


Fig. 2.18 Cadaver head impact data used to design the Hybrid III head. The data were from cadaveric forehead impacts to a rigid surface. The letter F adjacent to a data point indicates that there was skull fracture. The abscissa, $V^2/2g$, is an equivalent free fall drop height (taken from Mertz (1985)). Reprinted with permission Copyright © 2017 SAE International. Further distribution of this material is not permitted without prior permission from SAE

Fig. 2.19 Side view of a 50th percentile Hybrid III head



The cadaver data were obtained by Hodgson and Thomas (1971, 1975) who attached cadavers to a pallet that was hinged at the level of the floor and with the head overhanging the pallet. In this way, when the pallet was dropped from a known height, the forehead impacted a rigid surface in the form of a steel plate. However, the velocity of impact was mistakenly assumed to be equal to the square root of the product of $2gh$ where g is gravitational acceleration and h is the height of the drop. It was later discovered that the actual velocity of impact was greater than the free-fall velocity. Thus, the abscissa in Fig. 2.18 is the corrected free-fall height in terms of its free-fall velocity. The Hybrid III dummy is currently used as the human surrogate in the automotive industry. It consists of an aluminum head form covered by a vinyl “scalp” that was tuned to simulate the response of head impact against a rigid surface (see Hybrid III specification in Fig. 2.18). A photograph of the Hybrid III head is shown in Fig. 2.19. It is representative of a 50th percentile male in both size and weight. It should be noted that, strictly speaking, the Hybrid III head simulates human response for impacts of its forehead against a rigid surface. However, Mertz (1985) showed correlation of Hybrid III padded impacts to cadaver data.

2.5.1 Visualization of Brain Response

As mentioned in Sect. 2.4 above, attempts at direct visualization of brain motion by optical means during a head impact were made by Pudenz and Shelden (1946) and subsequently by several others. The use of a Lucite calvarium enabled the visualization of the motion of the surface of the brain. However, because a portion of the skull, including the dura, was removed, it is not clear if the observed motions could have been exaggerated because the normal constraints provided by the meninges no longer existed and there was air/gas between the brain and the Lucite. It was a valiant attempt, but the results may not be realistic and no quantitative data were provided. The use of roentgenography to visualize brain motion was mentioned

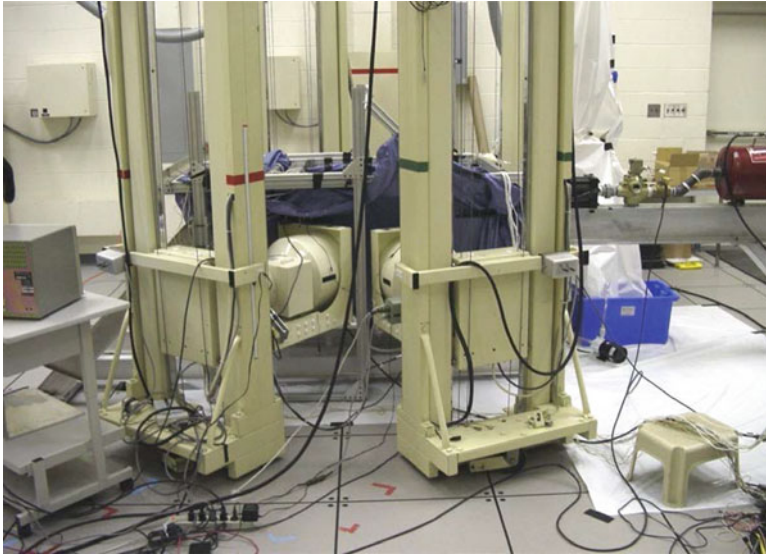


Fig. 2.20 Photograph of the biplanar X-ray setup (courtesy of Dr. Warren Hardy)

above in Sect. 2.5. Shatsky et al. (1974) used flash X-ray cinematography to capture head and brain motion during impact. A pulsed X-ray source with a duration of 30 ns was used to obtain X-ray images of a rhesus monkey head as it impacted a rigid wall. The images were enhanced by an image intensifier. There was also a study by Nusholtz et al. (1984) using a “high-speed” X-ray system, but the resolution was poor and the X-ray was only able to detect motion of blood vessels filled with a radiopaque dye.

Accurate visualization of brain motion in an intact skull was first reported by Hardy et al. (2001). A biplanar high-speed X-ray unit was used to obtain three-dimensional motion characteristics of cadaveric brain relative to the skull. A photograph of the system is shown in Fig. 2.20. The gantries in the foreground are supporting the image intensifiers. The X-ray sources are in the background and are hidden by the curtains surrounding the test specimen. On the right, the red tank contains compressed air that is used to accelerate the head before it hits a rigid surface. The experimental setup is shown diagrammatically in Fig. 2.21. The X-ray sources run continuously, and the X-ray images are captured and intensified by image intensifiers before they are recorded on video cameras. Initially, the recording speed was 250 frames/s, but eventually it was increased to 1000 frames/s. Movement of specimens placed in the crosshatched area can be measured in 3-D with an accuracy of ± 0.1 mm, using stereophotogrammetric methods. There are many details in the reduction of the data, such as computation of the motion in 3-D and correction for parallax, and the reader is referred to Hardy et al. (2001) for these procedures.

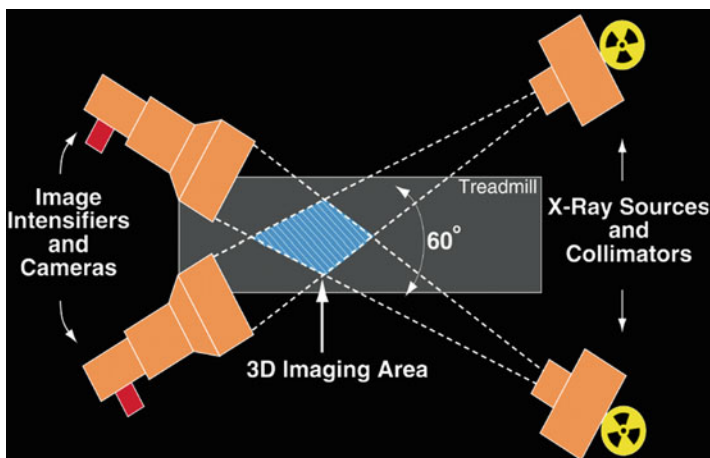


Fig. 2.21 Schematic of a biplanar high-speed X-ray system. The 3-D imaging area is in light blue ($45 \times 30 \times 25$ cm). The 3-D accuracy is ± 0.1 mm. This system is located on the main campus of Henry Ford Hospital, Detroit, MI

Freshly dead unembalmed cadaveric heads were used. To be able to visualize brain motion using the X-ray unit, tiny radiopaque targets in the form of tin spheres, 1.9 mm in diameter, were encased in plastic tubing to reduce its density to approximate that of the brain. Tin was used in place of dense metals such as gold or lead to reduce the mass of the target without a loss of contrast in the X-ray image. The targets were called neutral density targets (NDTs), and they did not cut through the brain material but would instead move with the brain during an impact. The NDTs are shown in Fig. 2.22. A large spinal needle was used as a guide for the placement of the targets. It was inserted into the brain through a small hole in the skull to a known depth, and targets were dropped into the needle and pushed into the brain at specified intervals to form a column of six or seven targets. In a typical test series for a sagittal plane impact, two columns of targets were inserted, one anteriorly, called the anterior column (AC), and the other posteriorly, called the posterior column (PC), as shown in Fig. 2.23. The head was decapitated at the level of T4 and was suspended upside down so that any air that might have entered the cranial cavity could be flushed out with artificial CSF that was used to pressurize the brain to simulate a living brain. Other instrumentation consisted of a 9-accelerator package attached to the skull and arranged in 3-2-2-2 configuration to measure the linear and angular acceleration of the head. A discussion of the method can be found in Chap. 5. The head assembly was suspended on a carriage that could slide smoothly on two horizontal rails, as shown in Fig. 2.24 enabling it to be accelerated by a piston driven by the compressed air. The moving head and carriage were arrested when the front of the head impacted a block of Lucite. The head stopped in the zone where the X-ray beams crossed and where it was possible to compute the 3-D location of the targets, using stereophotogrammetric methods. That zone is the light blue area shown in Fig. 2.22. Alternately, the

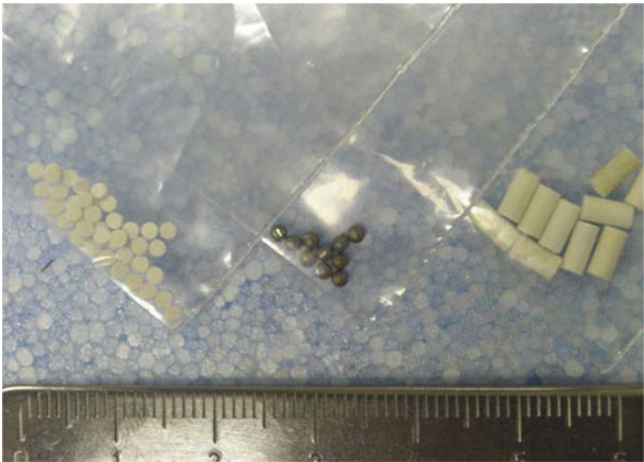
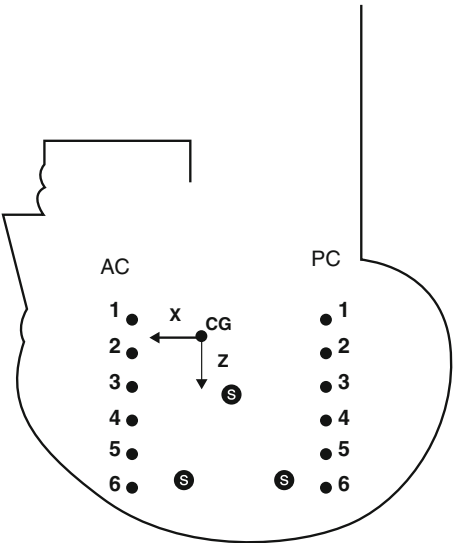


Fig. 2.22 Neutral density targets made from tin spheres encased in a plastic tube to reduce its density to approximately that of the brain. The tin spheres are in the center of the photograph. On the right are the plastic tubes and on the left are end caps to keep the sphere in the tube (taken from Hardy et al. (2001))

Fig. 2.23 Location of neutral density targets in a cadaveric brain for a sagittal plane impact. AC stands for anterior column and PC stands for posterior column (taken from Hardy et al. (2001))



air-driven impactor hit the back of the head directly while it was in the stereoscopic zone. The approximate location of the center of gravity (cg) of the head, as described above, was identified, and the motion of the targets relative to this hypothetical point was computed. These targets moved in a figure eight pattern, as shown in Fig. 2.25. Reduction of the data from the video cameras attached to the biplanar X-ray unit to the form shown in Fig. 2.25 is, to say the least, not a simple

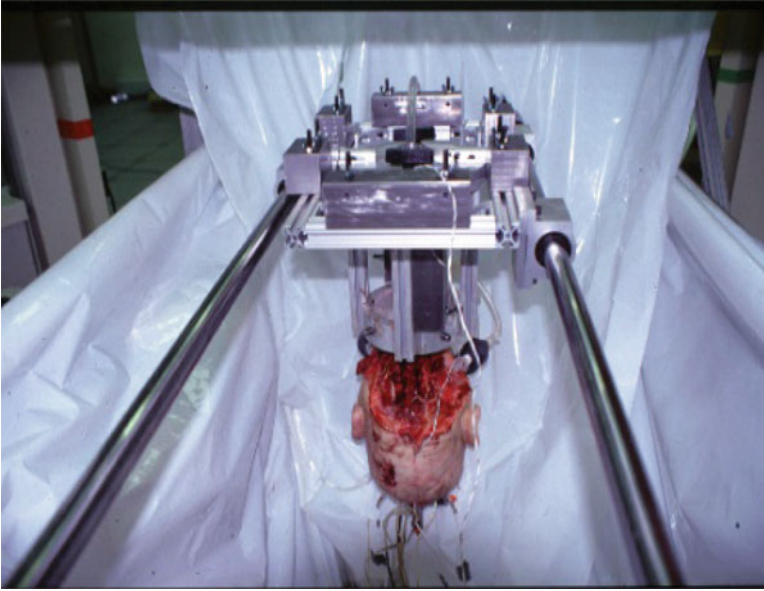


Fig. 2.24 Cadaveric head specimen suspended from a carriage used to accelerate the head into a Lucite block (taken from Hardy et al. (2001))

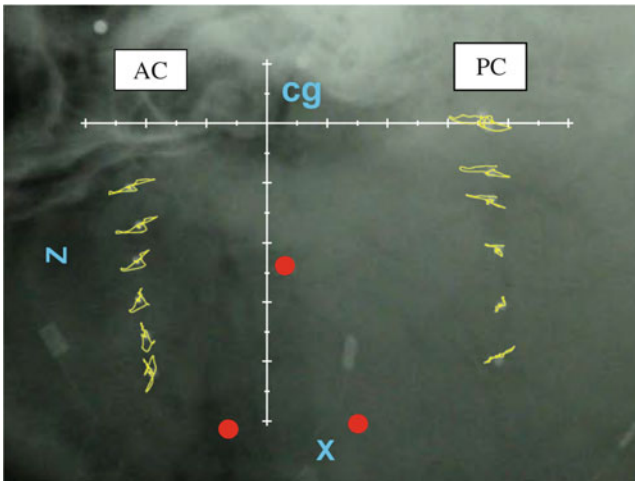


Fig. 2.25 The brain traces out a figure eight pattern during impact relative to the center of gravity of the head. The motion appears to decrease near the skull. The data were derived from a frontal impact against a Lucite block with a resultant deceleration of 62 g and a peak angular acceleration of 2529 rad/s^2 . AC stands for anterior column and PC stands for posterior column (taken from Hardy et al. (2001))

Table 2.2 Summary of head kinematics measured based on Hardy (2007) tests

Head kinematics	Range
Linear speed (m/s)	3.5 ± 0.3
Linear acceleration (g)	29–190
Resultant acceleration (g)	38–291
HIC ₁₅	87–959
Angular acceleration (rad/s ²)	2,370–24,206
Angular speed (rad/s)	20.3 ± 5.7

process. An automated image enhancement and target tracking algorithm were set up to process the digital video data from two cameras that recorded target motion obliquely. Distortion of the images needed to be corrected and the field of view was calibrated using a multi-point calibration cube after each test. Calculations were performed to transform the target motion data into a set of anatomical coordinates with the origin located at the presumed cg of the head. Additionally, the target data were synchronized in time with the measured acceleration data. Details of the procedure to produce the data as presented were described by Hardy (2007). Repeated tests were performed on each specimen because of the long preparation time needed to set up the experiment. Thus, the impact levels were generally kept low to avoid skull fracture. Table 2.2 is a summary of the head response parameters for the 30 tests performed on seven cadaveric specimens.

Note that in Fig. 2.25, the crown of the head is toward the bottom of the figure and that the excursions of the targets closer to the skull appear to decrease in the direction of the skull. The maximum excursion occurs near the center of the brain which is limited to ± 5 mm for a wide range of angular accelerations. Hardy (2007) also found that during linear acceleration, there is very little brain motion, of the order of ± 1 mm or less.

The combination of a biplanar X-ray system and the use of neutral density radiopaque targets produced unique three-dimensional motion data of the brain relative to the skull. The motion was largely due to head rotation and tended to follow a looping pattern with excursions limited to about ± 5 mm. A lot more data can be found in Hardy (2007).

2.5.2 Mechanical Properties of the Pia-Arachnoid Complex

Since the cerebral meninges are sandwiched between the skull and the brain, they are expected to play a significant role in any head impact. In particular, the pia-arachnoid complex (PAC) constitutes the mobile part of the meninges and participates in brain-skull interaction during a head impact. The PAC is compressed at the coup site and stretched or sheared at the contrecoup site. In compression, the CSF in the PAC is expected to transmit the compressive load to the brain, but, in tension or shear, the trabeculae take over the role of transmitting the load from the dura to the brain. For this reason, the properties of the PAC should be quantified



Fig. 2.26 Diagram of the pia-arachnoid complex, showing a blood vessel in the subarachnoid space (taken from Alcolado et al. (1988)). Republished with permission of John Wiley and Sons Inc., from R. Alcolado, R. Weller, E. Parrish, D. Garrod, The cranial arachnoid and pia mater in man: anatomical and ultrastructural observations. *Neuropathology and Applied Neurobiology* 14, 1–17, 1988, permission conveyed through Copyright Clearance Center, Inc.

both in terms of its response to normal traction and in shear. Basically, we are interested in the tensile and shear properties of the trabeculae in the CSF layer.

2.5.2.1 Response of the PAC to Normal Traction

Jin et al. (2007) studied the response of the PAC under normal traction, using fresh bovine specimens taken from different parts of the brain and at four different strain rates ($0.36\text{--}116.3\text{ s}^{-1}$). A diagram of the ultrastructure of the PAC is shown in Fig. 2.26. The bovine brain was selected for this study because of its relatively large size and the availability of fresh material from the slaughter house. The brains were harvested from calves aged 17–20 weeks immediately after they were slaughtered. Forty specimens, taken from four bovine brains, were tested within 48 h after death. After the skull was sawed open, the dura was carefully cut open so as not to damage the underlying structures, and the brain was taken out of the skull. Pieces of PAC about $20 \times 20\text{ mm}$ in size were dissected from the cortex, with about 2–5 mm of brain attached, as shown in Fig. 2.27A. The attached brain tissue was carefully removed from the pia, using a scalpel, and the PAC was placed on a plastic sheet with the pia facing up (Fig. 2.27B). A cubic polyethylene block, 127 mm in size, was attached to the pia with cyanoacrylate glue (Elmer's Krazy Glue), as shown in Fig. 2.27C. The arachnoid surface was thoroughly washed with an artificial CSF solution before it was glued to another polyethylene block of the same size and carefully aligned with the first block. After the glue had set, the excess tissue around the polyethylene blocks was trimmed off (Fig. 2.27D). This procedure ensured that there was no glue between the blocks. It was also important to ensure that there was no air bubble trapped between the blocks and the specimen to ensure that bond between the specimen and the blocks was stronger than the tensile resistance of the PAC. Since the blocks were transparent, trapped air bubbles could be spotted easily,

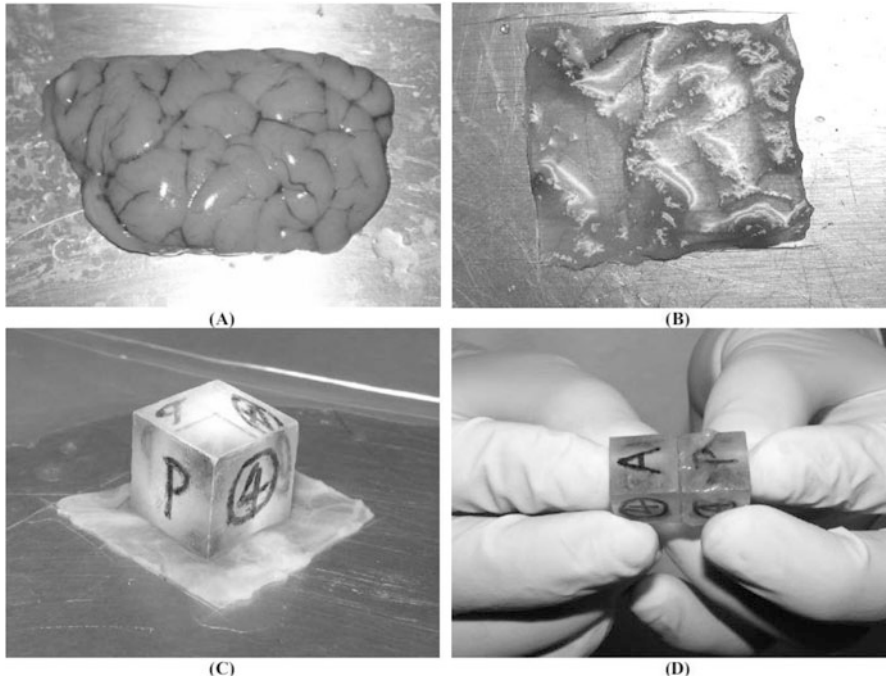


Fig. 2.27 This figure describes the specimen preparation procedure. (A) The cortex of the brain with the PAC attached. (B) PAC with the underlying brain removed and the pia facing up. (C) A polyethylene block (marked P for pia) was glued to the pia side of the PAC. (D) A second block (marked A for arachnoid) was glued to the opposite side of the PAC and the excess tissue was trimmed away (taken from Jin et al. (2007))

and defectively glued specimens were not tested. The specimen was tested in a Model 1321 Instron materials testing machine, again using cyanoacrylate glue to attach one end to the loading head of the machine and the other to its base. Both the applied tensile force and the loading head displacement were measured. The specimens were tested at strain rates of 0.36, 2.0, 20.5, and 116.3 s^{-1} . It was estimated by King et al. (2003) that the brain can sustain strain rates of $30\text{--}80 \text{ s}^{-1}$ in NFL mTBI cases and by Franklyn et al. (2005) in traumatic axonal injury cases in vehicular crashes. Specimens were taken from the frontal ($n = 14$), occipital ($n = 15$), and parietal ($n = 11$) regions of the brain in order to determine regional differences in mechanical properties of PAC, if any. In order to calculate the strain on the PAC in normal traction, an additional 65 PAC specimens were stained with hematoxylin, and the thickness of the specimen was measured under a microscope. Details of the procedure to measure PAC thickness are provided in Jin et al. (2006). The PAC thickness was $23.6 \pm 5.8 \text{ }\mu\text{m}$.

In terms of results, it was found that there was no regional difference in the response of the PAC for the four regions of the brain. It was also found that the mechanical response of the PAC was rate sensitive and that its elastic modulus was significantly higher at 116.3 s^{-1} than at the other 3 strain rates. Similarly, the ultimate stress and ultimate strain are rate sensitive. These data are shown in

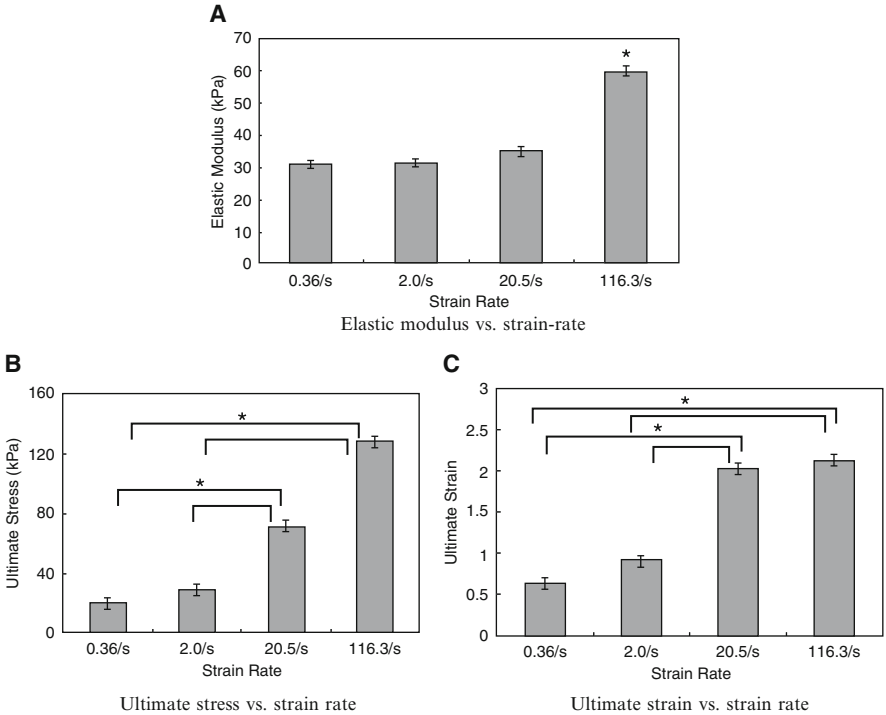


Fig. 2.28 Strain rate dependency of the PAC due to normal traction, as demonstrated by its elastic modulus (A), ultimate stress (B), and ultimate strain (C) (taken from Jin et al. (2007))

Fig. 2.28. Note that for most viscoelastic materials, the ultimate strain decreases with increasing strain rate, but for the PAC, the opposite is true. The PAC may be responding more like a structure than a single material, and more detailed study is required to explain this phenomenon.

2.5.2.2 Response of the PAC to Shear

Jin et al. (2011) conducted a similar study of the PAC to elicit its response to shear loading. This study has important implication with regard to the modeling of the PAC in finite element models of the brain. The CSF layer obviously cannot be modeled as a pure fluid because it contains trabeculae which are collagenous in nature. However, it would be difficult to assign a shear modulus to the PAC because no data were available. This study was designed to provide these data over a range of strain rates. The specimens used and the procedure for their preparation are identical to that described by Jin et al. (2007) with the exception that the blocks were glued together at the time of testing rather than ahead of time. A mini Instron materials testing machine was used along with a fixture that was designed to apply a

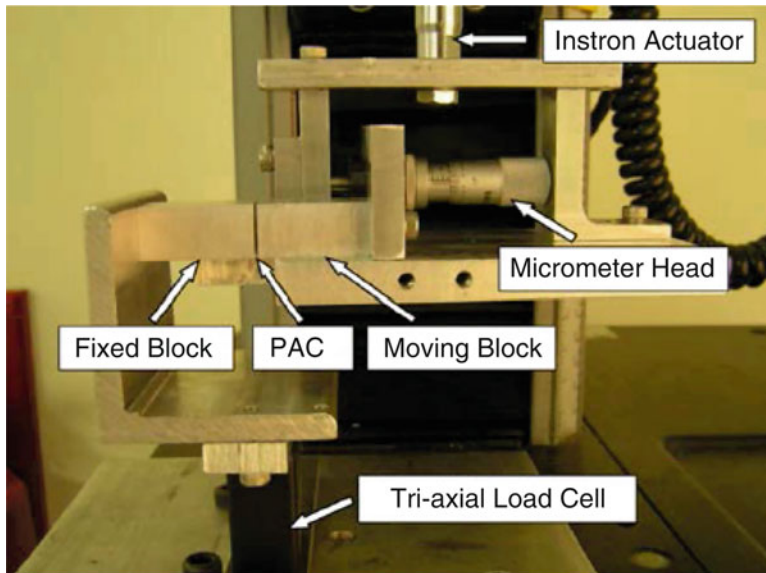


Fig. 2.29 Loading fixture to test the PAC in shear (taken from Jin et al. (2011)). Reprinted from X. Jin, K.H. Yang, A.I. King, Mechanical properties of bovine pia-arachnoid complex in shear. *Journal of Biomechanics*. 44(3), 467–474, 2011, with permission from Elsevier

pure shear load on the PAC specimen. It is shown in Fig. 2.29. The PAC is glued to the fixed block, and cyanoacrylate glue is sprayed onto the PAC surface as well as on the surface of the movable block. Then the movable block is moved by the micrometer toward the fixed block to glue the movable block to the PAC. Compression is maintained until the glue sets. At this point, the micrometer is turned in the opposite direction to relieve the compression before testing begins. A shear force is applied to the PAC when the loading head of the Instron moves downward. This shear load and the displacement of the loading head are recorded. Since the measured displacement is very small, it was necessary to take into account the deformation of the test fixture to obtain the true displacement experienced by the PAC. Forty-three PAC specimens from the frontal, parietal, and occipital regions of the brain were tested at 0.84 , 7.3 , and 72 s^{-1} .

In terms of results, there is again no regional difference in shear modulus, ultimate stress, and ultimate strain for the three groups. The strain rate effect is again seen in shear. Figure 2.30 shows the increase in modulus, ultimate stress, and ultimate strain as a function of strain rate. Significant differences at $p < 0.05$ and $p < 0.001$ are identified by asterisk(s) (*). Both the shear modulus and ultimate stress are rate sensitive. Ultimate strain does not appear to be very sensitive to strain rate. The takeaway message is that the CSF layer can resist shear and any brain model that assumes it to be a pure fluid will probably not predict the correct motion of the brain.

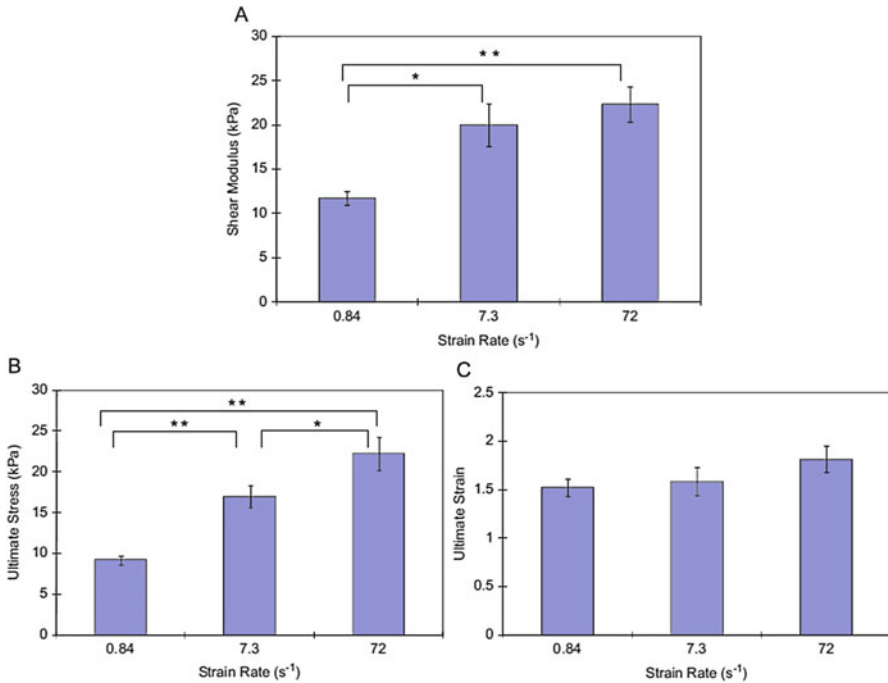
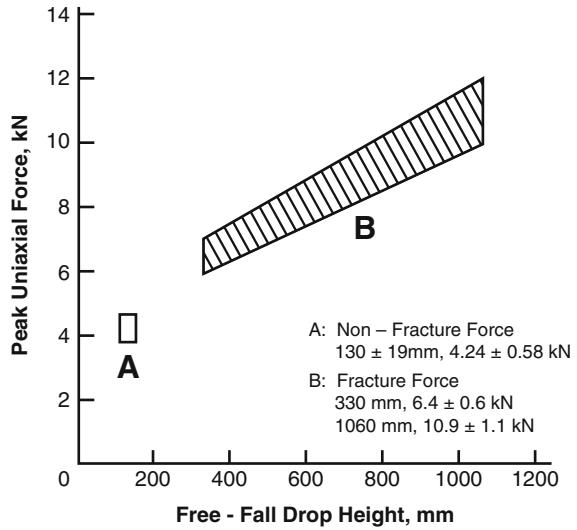


Fig. 2.30 Strain rate dependency of the PAC due to shear loading, as demonstrated by its shear modulus (A), ultimate stress (B), and ultimate strain (C) (taken from Jin et al. (2011)). Reprinted from X. Jin, K.H. Yang, A.I. King, Mechanical properties of bovine pia-arachnoid complex in shear. *Journal of Biomechanics*. 44(3), 467–474, 2011, with permission from Elsevier

2.6 Tolerance of the Head and Brain to Blunt Impact

Unintentional injuries to the head and brain are usually caused by blunt impacts as opposed to penetrating impacts that are encountered in intentional injuries. Tolerance of the head refers to the tolerance of the skull to fracture which has a bearing on the tolerance of the brain to impact, but skull fracture is not a precise measure of brain injury. Lissner et al. (1949) initiated brain injury research by studying the energy required to cause skull fracture because the presence of skull fracture is an index of the severity of the blow, with a history of unconsciousness and severe brain damage (Gurdjian et al. 1963). The energy required to fracture a skull is highly variable, depending on the location of the impact on the skull or its thickness, the thickness of the scalp, and the shape of the impactor. It can vary from 400 in-lb to over 1000 in-lb or 58 to over 146 N-m (Lissner et al. 1949). Also, brain damage can occur with or without skull fracture. Thus, it is necessary to separate the tolerance of the skull to fracture from the tolerance of the brain to the many forms of brain injury discussed in Sect. 2.3.1.

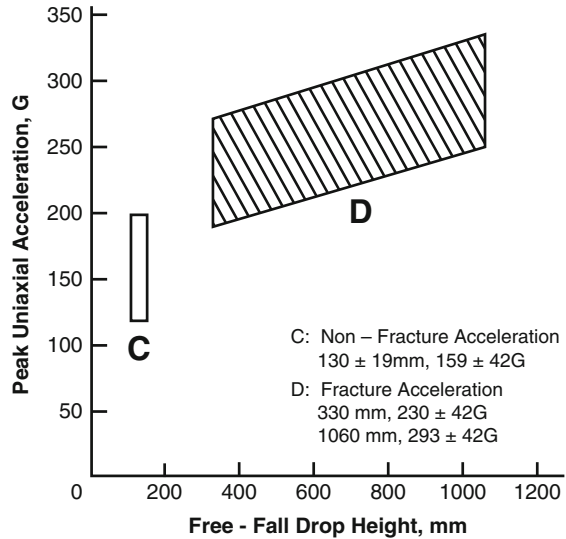
Fig. 2.31 Tolerance of the human skull to impact with a rigid surface in terms of peak impact force (taken from Prasad et al. (1985))



2.6.1 Tolerance of the Skull to Fracture

Hodgson and Thomas (1971, 1975) dropped embalmed cadaver heads onto a rigid (steel) surface. The whole cadaver was placed on a pallet hinged at the floor level with the head extending beyond the top edge of the pallet. The velocity of impact was originally calculated as the square root of twice the product of the height of the head above the impacted surface (h) and the acceleration due to gravity (g) ($\sqrt{2gh}$) (see Prasad et al. 1985, p. 12). It was eventually corrected by measuring the velocity of the head of a dummy placed on the pallet. The free-fall drop height was based on the measured velocity. The head acceleration was measured by an accelerometer placed on the skull on the opposite side of the impact, and the force of impact was measured by a load cell on the floor. There were frontal, side, and occipital (rear) impacts. However, there was a large amount of scatter in the data, and it was not possible to draw regression curves for each direction of impact. If all the data from the three directions of impact were grouped together, it was possible to draw rectangles around the data to separate the fractured cases from the non-fracture cases. The fracture force as a function of the corrected free-fall drop height is shown in Fig. 2.31. It is seen that the skull can be fractured from a drop height of 330 mm or approximately 13 in. In terms of head acceleration, the tolerance of the skull is shown in Fig. 2.32. The lower limit for fracture is in the 200 g range. It should be noted that the embalmed cadaver has a thicker scalp because it retains embalming fluid and it is reasonable to expect the tolerance to fracture to be somewhat lower.

Fig. 2.32 Tolerance of the human skull to impact with a rigid surface in terms of peak head acceleration (taken from Prasad et al. (1985))



2.6.2 Tolerance of the Brain to Blunt Impact

Blunt impacts to the brain can cause a variety of brain injuries at different severities. If the AIS manual is consulted, there are injuries to the brain from AIS 1 (mild concussion) to AIS 6 (crushed skull), with a large number of injuries at each AIS level. If we are to look at brain injury from the viewpoint of automotive safety, the original standard was set for AIS 4+ injuries because airbags were not available and seat belt use was not popular. Currently, the tolerance level has been lowered to reflect the protection afforded by the airbag. In sports, the issue of mTBI is a major concern because athletes can experience multiple head impacts. Thus, the exposure level for football players is much lower than that for automotive occupants.

The history of the search for human tolerance to blunt impact goes back to the work of early researchers. Some of the tolerance data were deduced from the head accelerations required to cause a skull fracture or to cause blood vessel damage in cadaveric brains which were perfused with India ink. Other data were based on an extrapolation of live animal concussion data to the human level.

The process took about two decades of research to result in a head injury tolerance curve, currently known as the Wayne State Tolerance Curve (WSTC), which was first published in preliminary form by Lissner et al. (1960) as a fracture tolerance curve shown in Fig. 2.33. Animal data were added to this curve along with the single data point from the famous sled ride by Col. Stapp resulted in the original WSTC. It is a plot of the “effective” acceleration of the head vs. the duration of impact, as shown in Fig. 1.5. The term “effective acceleration” was not defined but assumed to be less than the peak acceleration but larger than the average acceleration. Eventually, effective acceleration became synonymous with average acceleration. What the WSTC basically says is that the brain

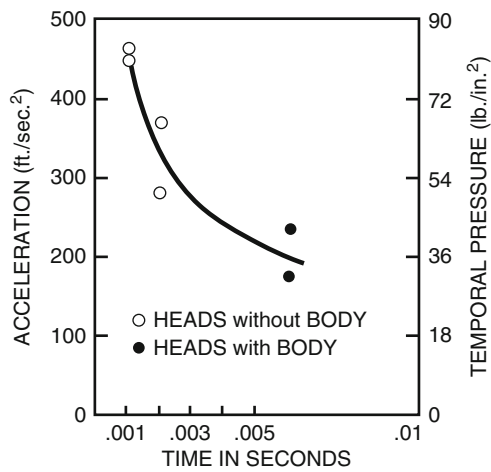
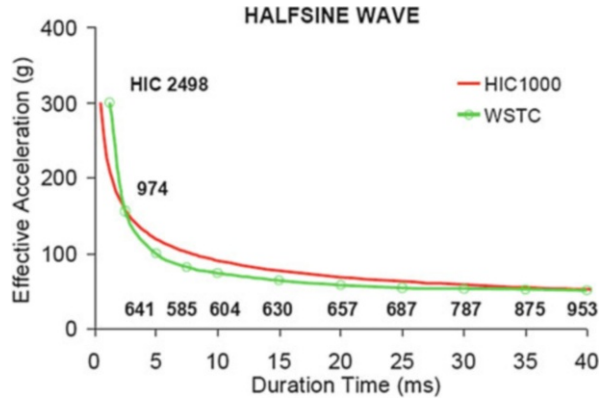


Fig. 2.33 Tolerance of the skull to fracture in terms of acceleration and pulse duration. Clinically, a simple skull fracture is frequently associated with a mild concussion. Thus, this curve can be regarded as a tolerance curve for brain concussion. It is the forerunner of the Wayne State Tolerance Curve shown in the next figure. (Note: The units for acceleration along the ordinate should be g's instead of ft/s²) (taken from Lissner et al. (1960)). Reprinted from H. Lissner, M. Lebow, F. Evans, Experimental studies on the relation between acceleration and intracranial pressure changes in man. *Surgery, Gynecology & Obstetrics* 111, 329–338, 1960, with permission from Elsevier

Fig. 2.34 Comparison of HIC of about 1000 for a half-sine wave with the WSTC



can tolerate higher impact accelerations at shorter durations. According to Patrick et al. (1963), the curve was based on a reversible concussion with no aftereffects, but it turned out that it marks the boundary for a severe head injury.

In terms of the data, the short-duration data (10 ms or less) were generally associated with cadaveric skull fracture data, while those between 10 and 40 ms were from animal data and the asymptote was based on the 42 or 45 g peak chest acceleration sustained by Col. John Paul Stapp who rode the rocket sled in Alamo-gordo, NM, in 1949 and suffered an injury to his right eye. This level was eventually raised to 80 g because at 42–45 g, a brain injury is not likely.

Gadd (1962) plotted the WSTC on log-log paper and discovered that the curve approximated a straight line with a slope of -2.5 . He proposed a severity index (SI) which became known as the Gadd Severity Index (GSI) in the form:

$$\text{GSI} = \int a^{2.5} dt \leq 1000 \quad (2.2)$$

where $a(t)$ is the resultant head acceleration and t is the time

The integration is to extend over the entire duration of the pulse. The rulemakers at the National Highway Traffic Safety Administration (NHTSA) proposed that Eq. (2.2) should be used as a head injury criterion for automotive crash safety. It was a good suggestion in that the measured head acceleration in dummy tests could be used conveniently to calculate the GSI and used to determine if the head impact was acceptable. It soon became evident that this criterion was difficult to meet, and Versace (1971) proposed an alternate criterion which became known as the Head Injury Criterion or HIC. The equation for HIC is as follows:

$$\text{HIC} = (t_2 - t_1) \left[\int_{t_1}^{t_2} a(t) dt / (t_2 - t_1) \right]^{2.5} \Big|_{\max} \quad (2.3)$$

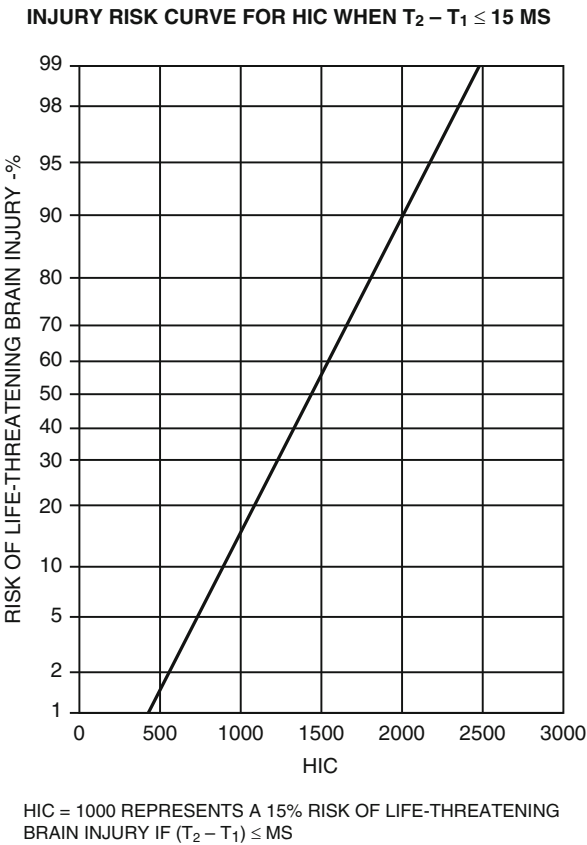
where $a(t)$ is the resultant head acceleration and t_1 and t_2 are the limits of integration over time selected so as to maximize the value of HIC.

The time interval would obviously have to be within the pulse duration of the impact. This time interval should not exceed 15 ms because most impact durations of head contact last 15 ms or less. With this limitation, the calculated HIC is designated as HIC_{15} . Although field accident data indicate that, in the automotive environment, if there was no head contact with vehicular structures during a crash, there were no cases of head injury, it would still be possible to calculate a HIC value for the measured head acceleration (Prasad and Mertz 1985). In this case, the maximum interval needs to be extended to 36 ms and the calculated HIC is designated as HIC_{36} . The process for determining the value of HIC is embodied in a software program that selects the time duration t_1 to t_2 that maximizes the value of HIC, using the interval for numerical integration as 1 ms.

Obviously, HIC is dependent on the pulse shape, and it is not possible to compare HIC to the WSTC for all pulse shapes. However, if we assume a half-sine wave for the head acceleration, the calculated HIC values for durations between 1 and 40 ms were found to be comparable to the WSTC, as shown in Fig. 2.34. The red curve is for a HIC of 1000, while the HIC values for the WSTC (green) curve range from 585 to 2498.

Prasad and Mertz (1985) also did a detailed analysis of the human head impact data collected from a variety of sources. They found that, for the most part, skull fracture and brain damage (arterial rupture) occurred at comparable HIC values and decided that since these were cadaveric data, it was more reliable to use skull fracture data as a basis for brain damage than data related arterial rupture.

Fig. 2.35 Injury risk curve in terms of HIC based on the WSTC (based on Prasad and Mertz (1985))



There were 54 tests with 27 skull fractures. A logistic curve was drawn to determine the probability of skull fracture (or brain damage) as a function of HIC. Using the logarithmic scale for injury probability (of AIS 4+), the S-curve becomes a straight line, as shown in Fig. 2.35. It is seen that at a HIC of 1000, the risk of an AIS 4+ injury is 15 %. The current US safety standard calls for a limit of $HIC \leq 700$ or an injury risk of approximately 5 %.

It should be noted that neither the WSTC nor GSI or HIC can provide detailed information on the damage sustained by the brain. For example, it is not possible to predict from HIC whether there was DAI, concussion, subdural hematoma, brain stem injury, or even skull fracture. HIC is useful for assessing the degree of protection provided by safety features in a car and is thus a suitable criterion for rulemaking. To study brain injury in greater detail, we need to look into specific research studies and to use computer models to predict outcome.

Currently, there is no accepted tolerance value for the brain to angular acceleration. Research involving the use of concussion data derived from on-field helmet-to-helmet impacts revealed that the angular acceleration for a 50 % probability of a mild concussion is between 5500 and 6400 rad/s^2 (see Table 6.4, Chap. 6).

Questions for Chapter 2

2.1. Select the statement that is valid, as it relates to brain injury:

- ☐ (i) To generate high shear strains in the brain, it is necessary to subject the head to linear accelerations
- ☐ (ii) Mild traumatic brain injury cannot occur unless the victim was unconscious for a short time
- ☐ (iii) A noncontact head impact resulting in a rotational acceleration of 6000 rad/s^2 can cause a severe brain injury
- ☐ (iv) Bridging vein ruptures occur as a result of high angular accelerations in the mid-sagittal plane
- ☐ (v) All of the above

2.2. Select the statement that is valid, as it relates to brain injury:

- ☐ (i) The pia and arachnoid are separated by cerebral spinal fluid and the two membranes are not connected by any soft tissue
- ☐ (ii) The shear resistance between the pia and arachnoid is due solely to the presence of the cerebral spinal fluid
- ☐ (iii) It is valid to model the space between the pia and the arachnoid as a Newtonian fluid
- ☐ (iv) The space between the pia and the arachnoid is devoid of blood vessels
- ☐ (v) None of the above

2.3. Understanding the mechanical response of the pia-arachnoid complex (PAC) is important for modeling of the brain. Which of the following statements is true:

- ☐ (i) There is no cerebral spinal fluid between the pia and the arachnoid
- ☐ (ii) The trabeculae offer no tensile resistance when the arachnoid is pulled away from the pia
- ☐ (iii) The border cells are between the pia and the arachnoid
- ☐ (iv) The PAC has been tested in both plane tension and normal traction
- ☐ (v) None of the above

2.4. Select the statement that is valid, as it relates to brain injury:

- ☐ (i) Diffuse axonal injury (DAI) occurs several hours or days after head impact
- ☐ (ii) Brain motion within an intact human skull during an impact is more sensitive to linear acceleration than to angular acceleration
- ☐ (iii) DAI can only occur in the gray matter of the central nervous system (CNS)
- ☐ (iv) The tolerance of the brain to angular acceleration is 16000 rad/s^2
- ☐ (v) If HIC is under 1000, there can still be brain injury

2.5. Wearing a helmet helps to prevent or minimize brain injury by

- ☐ (i) Decreasing angular acceleration
- ☐ (ii) Decreasing linear acceleration
- ☐ (iii) Preventing relative brain motion with respect to the skull
- ☐ (iv) Causing the skull to deform more
- ☐ (v) None of the above

2.6. Select the statement that is valid, as it relates to brain injury:

- ☐ (i) Diffuse axonal injury (DAI) is actually a breakdown of the microtubules within axons
- ☐ (ii) DAI cannot be produced by pure linear acceleration
- ☐ (iii) DAI can only be caused by angular acceleration
- ☐ (iv) DAI can be diagnosed from a CT scan of the brain
- ☐ (v) DAI severity is not related to the duration of coma

2.7. The diploe of the skull is

- ☐ (i) The outer covering of the skull bone
- ☐ (ii) The outer layer of bone of the skull
- ☐ (iii) The middle layer of bone of the skull
- ☐ (iv) The inner layer of bone of the skull
- ☐ (v) The inner covering of the skull bone

2.8. Which of the following statements is incorrect:

- ☐ (i) The dura is a double-layered covering above the brain surface, next to the skull
- ☐ (ii) The dura is a thick and tough membrane which is attached to the skull
- ☐ (iii) The dura forms a sinus in the midline of the skull for drainage of venous blood
- ☐ (iv) The dura is not in contact with the cerebral spinal fluid surrounding the brain
- ☐ (v) The dura is a thin membrane between the arachnoid and the pia

2.9. The following statements relate to normal cerebral spinal fluid (CSF). Which one is incorrect?

- ☐ (i) CSF contains water and proteins
- ☐ (ii) CSF has the approximate mass density of water
- ☐ (iii) CSF contains a substantial number of red as well as white blood cells
- ☐ (iv) CSF is contained within the dural sac of the central nervous system
- ☐ (v) CSF is found in the ventricles of the brain

2.10. The central nervous system

- ☐ (i) Consists entirely of white matter
- ☐ (ii) Consists entirely of gray matter
- ☐ (iii) Consists of both neurons and axons

- ☐ (iv) Ends at the junction of the head and neck
- ☐ (v) None of the above

2.11. Which of the following statements is incorrect?

- ☐ (i) The white matter consists mainly of axons
- ☐ (ii) The brain of a normal person weighs approximately 6 lb
- ☐ (iii) The medulla oblongata is the brain stem
- ☐ (iv) The cerebellum is larger than the cerebrum
- ☐ (v) All of the above are correct

2.12. There are several types of brain injury due to blunt impact. Which one of the following is incorrect?

- ☐ (i) Focal injuries
- ☐ (ii) Mass lesions
- ☐ (iii) Lacerative injuries
- ☐ (iv) Diffuse injuries
- ☐ (v) None of the above

2.13. Diffuse axonal injury

- ☐ (i) Can only occur in the gray matter
- ☐ (ii) Is not caused at the instant of impact
- ☐ (iii) Can only be caused by angular acceleration
- ☐ (iv) Can only occur in the white matter
- ☐ (v) Is not associated with loss of consciousness

2.14. Several mechanisms for brain injury have been proposed. Which of the following is not commonly accepted as being valid?

- ☐ (i) Positive pressure at the coup site
- ☐ (ii) Negative pressure at the contrecoup site
- ☐ (iii) Pressure gradients causing development of shear stresses
- ☐ (iv) Change in volume of the skull
- ☐ (v) Rotational effects causing DAI

2.15. The following statements refer to blunt impact to the head. Which one is incorrect?

- ☐ (i) High-speed X-ray data on brain motion are available
- ☐ (ii) High-speed X-ray data on brain motion are available for low severity impacts
- ☐ (iii) High-speed X-ray data on brain motion are not available from living human subjects
- ☐ (iv) High-speed X-ray data on brain motion have not been published
- ☐ (v) High speed X-ray data on brain motion can be acquired at 1000 frames/s

2.16. Brain motion within the skull due to a low speed impact (less than 5 m/s):

- ☐ (i) Cannot be measured or observed
- ☐ (ii) Is very large, exceeding 20 mm in most cases
- ☐ (iii) Is more sensitive to linear acceleration than angular acceleration
- ☐ (iv) Is relatively small, not exceeding 10 mm in most cases
- ☐ (v) None of the above

2.17. The human skull can be fractured at the following levels of impact:

- ☐ (i) Drop heights in excess of 0.5 m
- ☐ (ii) Force levels in excess of 6 kN
- ☐ (iii) Peak accelerations below 100 g
- ☐ (iv) (i) and (iii) above
- ☐ (v) (i) and (ii) above

2.18. Tolerance of the brain to blunt impact:

- ☐ (i) Can be expressed in terms of $HIC < 200$
- ☐ (ii) Is limited in angular acceleration to 5000 rad/s^2
- ☐ (iii) Is due entirely to linear acceleration
- ☐ (iv) Is due entirely to angular acceleration
- ☐ (v) None of the above

2.19. Brain motion relative to the skull is

- ☐ (i) Larger than $\pm 10 \text{ mm}$ at the periphery of the brain
- ☐ (ii) Occurs between the dura mater and the skull
- ☐ (iii) Higher due to linear acceleration than angular acceleration
- ☐ (iv) Limited to $\pm 5 \text{ mm}$ regardless of the severity of impact
- ☐ (v) None of the above

2.20. The following statements relate to normal cerebral spinal fluid (CSF). Which one is incorrect?

- ☐ (i) CSF contains water, blood cells, and proteins
- ☐ (ii) CSF has the approximate mass density of water
- ☐ (iii) CSF is found in the brain, both between the pia and the arachnoid and in the ventricles
- ☐ (iv) CSF is contained within the dural sac of the central nervous system
- ☐ (v) CSF is found in the ventricles of the brain

Answers to Problems by Chapter

Prob	Ans
1	(iv)
2	(v)

(continued)

Prob	Ans
3	(iv)
4	(v)
5	(ii)
6	(i)
7	(iii)
8	(v)
9	(iii)
10	(iii)
11	(ii)
12	(v)
13	(iv)
14	(iv)
15	(iv)
16	(iv)
17	(v)
18	(v)
19	(iv)
20	(i)

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