

# SECTION A

## Cranial Trauma

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### Biomechanics of Head Injury

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How mechanical energy injures the head has been of interest to physicians for centuries. Gradually a picture has emerged that now permits a comprehensive understanding of the causes of many types of head injury. In this chapter we will review the state-of-the-art knowledge of the mechanical events that result in the numerous varieties of head injury that we encounter clinically.

To understand how an individual mechanical input to the head results in a particular type of head injury, one must consider multiple factors. First, the nature, the severity, and the site and direction of mechanical input to the head are important. The manner in which the head responds to that input will determine what structures are injured and to what extent they are injured. Finally, the total injury produced by mechanical trauma depends not only on the primary mechanical damage but also on the complex interaction of pathophysiological events that follow. This chapter will concern only the primary mechanical events.

In general terms, primary head injuries include three distinct varieties, each having a unique set of mechanical etiology (Table 188-1). *Skull fracture* can occur with or without damage to the brain, but is itself usually not an important cause of neurological disability. Injuries to the vascular or neural elements of the brain and its coverings cause neurological dysfunction and can be readily divided into two categories, each of which has, for the most part, different mechanistic causes. *Focal injuries* result from localized damage and account for approximately 50 percent of all hospital admissions for head injury.<sup>1,3a</sup> The cortical contusions, subdural hematomas, epidural hematomas, and intracerebral hematomas that constitute focal injuries are responsible for

two-thirds of head injury associated deaths.<sup>4</sup> *Diffuse brain injuries* are associated with widespread brain damage. This damage may be principally functional, as in the case of concussive injuries, or may be structural, as seen in prolonged traumatic coma unassociated with mass lesions, a condition recently termed *diffuse axonal injury*.<sup>2,3a,6</sup> Diffuse brain injuries account for approximately 40 percent of hospitalized head-injured patients and for one-third of the deaths, and are the most serious cause of persisting neurological disability in survivors.

#### Mechanisms of Injury

The types of mechanical loading of the head are numerous and complex.<sup>9</sup> Input can be either slow (static loading) or, as more commonly occurs, rapid (dynamic loading). Static loading implies that the injury forces are applied gradually, usually over 200 ms or longer. This is comparable to a slow squeezing effect, and if sufficient force is applied, serious multiple skull fractures result. After the skull has absorbed as much energy as it can withstand, it begins to crush, the brain itself becomes compressed and distorted, and serious or fatal brain injury occurs. Static loading is so uncommon that it will not be discussed in further detail.

The most frequent type of mechanical input is dynamic input. Here the injury forces act in less than 200 ms, and in most cases in less than 50 ms. As will be seen later, the duration of the input is a critical factor in determining which type of head injury occurs. Dynamic loading can be of two types, impulsive loading and impact loading. *Impulsive loading* occurs when the head is set into motion (or when the moving head is stopped) without the head being struck. These conditions occur not infrequently, as when a child is shaken by the shoulders or when a blow occurs to the body in such a manner that the head moves violently (blows to the

TABLE 188-1 The Primary Head Injuries

Skull Fractures	Focal Injuries	Diffuse Injuries
Linear	Contusions	Concussion
Depressed	Coup	Mild
Basilar	Contrecoup	Classical
	Intermediate	Diffuse axonal injury
	Hematomas	Mild
	Extradural	Moderate
	Subdural	Severe
	Intracerebral	

face or impact to the chest or thorax). In these circumstances, there is no impact to the cranium, and the resulting head injuries are caused solely by the inertial forces that result from head acceleration or deceleration.

*Impact loading* is the more frequent type of dynamic loading and usually causes acceleration of the head (inertial effects), as well as many regionalized effects known as contact phenomena. The inertial effects can be minimal in certain impact conditions if the head is prevented from moving when it is struck. The contact phenomena are a complex group of mechanical events that occur both near and distant from the point of impact. The magnitude and importance of these contact phenomena vary with the size of the impacting device and with the magnitude of force of the impact. Immediately beneath the point of impact there is localized skull deformation, with inbending of the skull surrounded by outbending of the skull peripheral to the impact site. If the degree of local skull deformation is significant, penetration, perforation, or fracture of the skull occurs. Additionally, shock waves that travel at the speed of sound propagate throughout the skull from the point of impact, as well as directly through the brain substance. The shock waves cause local changes in tissue pressure, and if these result in sufficient brain distortion, brain damage results.

The strains induced by the inertial (acceleration-deceleration) or by the contact (skull bending, shock waves) loading are the ultimate and proximate causes of injury. Three types of strain can occur: compression, tension, and shear. The type of injury that results from a particular circumstance is determined by the type and location of the induced strains and by the tissue's ability to withstand those particular strains. Strain is best understood as the amount of deformation that the tissue undergoes as a result of mechanical loading. Tensile strain, for example, is the amount of elongation that occurs when a material is stretched. If a column of rubber 10 cm in length becomes 11 cm long when stretched, it has undergone a 10 percent tensile strain. A glass column under the same load may become only 10.1 cm long, a 1 percent strain. The inherent properties of these two materials are different, not only in how much strain occurs under given loading conditions but also with regard to how much strain is necessary to cause failure (breakage) of the material. Thus the rubber column may tolerate a 20 percent strain before it breaks, while the glass column may break at 0.5 percent strain. In this example the glass rod would have broken, but the rubber column would not have, even though the rubber underwent a greater strain. In addition, biological tissues are viscoelastic; that is, their tolerance to strain changes with the rate at which the mechanical load is applied. Characteristically, biological tissues withstand strain better if they are deformed slowly rather than quickly; that is, they become more brittle and will break at lower strain levels under rapidly applied loads.

The three principal tissues involved in head injury vary considerably in their tolerances to deformation; bone, for example, is considerably stronger than vascular or brain tissue. It therefore requires more force to obtain injurious levels of strain. However, bone shares with vascular and brain tissue the common property of being more able to withstand compressive strains than shear strains, with a tensile strain tolerance somewhere in between. For bone there is proportion-

ately less difference between the three strain tolerances, whereas for brain there is a considerable difference in its ability to withstand compression and shear. Since brain is virtually incompressible and since it has a very low tolerance to tensile and shear strain, the latter two types of strain are the usual causes of brain damage. The same is true for vascular tissue. Whether vascular or brain tissue damage occurs depends on the exact properties of these two tissues. As we will see later, vascular tissue tends to fail under more rapidly applied loading conditions than does brain tissue, and situations exist that, depending on the type of input to the head, can cause relatively pure injury to the vascular elements or to the neural elements within the head.

## Mechanistic Causes of Head Injuries

Most head injuries are due to one of two basic mechanisms, contact or acceleration. Contact injuries require that the head strike or be struck, irrespective of whether the blow causes the head to move afterward. Acceleration injuries result from violent head motion, irrespective of whether the head moves because of a direct blow or not (Table 188-2).

### Contact Injuries

Contact injuries, in general, are caused by forces that occur during impact. These injuries result solely from contact phenomena and have nothing whatever to do with head motion or head acceleration or deceleration. Since most impacts also set the head into motion, these injuries rarely occur clinically in pure form; more frequently, contact injuries have superimposed acceleration (inertial) injuries. Many times, however, the injuries received by a patient are predominantly contact-related. Contact injuries can therefore be viewed as injuries that would occur if the head were prevented from moving.

Contact forces are twofold in nature: effects that occur locally at or near the impact and effects that occur remote from impact. In both instances, contact injuries cause focal injuries; contact forces do not cause diffuse brain injury (concussion or primary traumatic unconsciousness).

TABLE 188-2 Mechanistic Types of Head Injury

Contact Injuries*	Acceleration Injuries†
Skull deformation injuries	Surface strains
Local:	Subdural hematoma
Skull fracture (linear, depressed)	Contrecoup contusion
Extradural hematoma	Intermediate coup contusion
Coup contusions	Deep strains
Remote:	Concussion syndromes
Vault and basilar fractures	Diffuse axonal injury
Shock wave injuries	
Contrecoup contusion	
Intracerebral hematoma	

\*Blow to head necessary; head motion not necessary.

†Direct blow to vault not necessary; head motion necessary.

### Local Contact Effects

Injuries due to the local effects of contact forces comprise most linear and depressed skull fractures, epidural hematomas, and coup contusions.

The occurrence of skull fracture depends on the material properties of the skull, the magnitude and the direction of impact, the size of the impact area, and the thickness of the skull in various areas.<sup>8</sup> When an object strikes the head, local skull inbending occurs, and the resulting skull deformation causes a compression strain on the outer table and a tensile strain on the inner table. Since bone is weaker in tension than in compression, sufficient inbending causes a fracture to begin in the inner table. It will then propagate along lines of least resistance from the impact site. The length of the fracture and its direction depend on the thickness of the skull at the impact site. A sufficiently small impactor will focus the impact energy at the impact site and is more prone to cause a depressed fracture or skull perforation, whereas a larger object distributes the impact force over a wider area and is less likely to cause fracture.

The epidural hematoma can be viewed as a complicated variety of skull fracture in which the dural vessels are torn. The mechanical failure of these vessels can occur as a fracture propagates across the vessel, or the vessel may be injured without fracture if there is sufficient skull bending to tear it.

Coup contusions occur beneath the site of impact under certain conditions. Such contusions are due to either direct injury to the brain beneath an area of skull inbending or negative pressures that develop when an area of skull inbending rapidly snaps back into place. The former mechanism causes highly focused compressive strains, whereas the latter subjects the brain surface to very high tensile strains; in either case these strains are sufficient to cause tissue failure of the pial and cortical vessels and of the brain tissue itself. The localized contusion that results is therefore a mixture of vascular and brain disruption. Brain laceration is an extension of the same phenomena, where skull inbending is sufficient to perforate the brain tissue.

### Remote Contact Effects

Contact phenomena can cause injury remote from the impact site by two mechanisms: skull distortion and shock waves. Both contribute to vault fractures that occur away from the impact site, to basilar skull fractures, and to contrecoup and the so-called intermediate coup contusions.

Remote vault fracture can occur if the impact occurs over a thick portion of the skull. Local inbending has little effect, but the skull bends out around the impact zone, putting the outer table under tension and the inner table under a compressive strain. If this area of outbending is in a thin area of the skull, a fracture begins in the outer table some distance from the point of impact. The fracture will again propagate along the line of least resistance, usually, but not always, toward the impact site. Often the line of least resistance is not over the vault, and the various characteristic types of basilar skull fracture occur.

Shock waves begin from the point of impact and travel rapidly in all directions from it. Those that spread through

the skull contribute to skull distortion and the ensuing basilar and remote vault fractures. Other shock waves spread through the brain in several microseconds and, like waves in water, may reflect from the opposite side of the head and reverberate within the brain. If the shock waves are amplified by this reverberation, the resulting strains may be sufficient to exceed the brain's tolerance. Damage to vascular or brain tissue at sites of strain concentration would result. Although this thesis has been used to explain the genesis of intermediate and contrecoup contusions, the role of shock waves in causing local brain damage has been a matter of debate. It has been argued that because these waves travel exceedingly rapidly through the brain, their effects are so quickly dissipated that they are not an important injury mechanism.

### Acceleration (Inertial) Injuries

Inertial loading of the head, whether caused by impact or by impulsive loading, accelerates or decelerates the head. From the mechanical point of view, acceleration and deceleration are the same physical phenomenon and differ only in direction. Thus the effects of accelerating the head in the sagittal plane from posterior to anterior are exactly the same as the effects of decelerating the head from anterior to posterior.

Head acceleration results in compressive, tensile, and shear strains that cause structural damage by one of two mechanisms. First, acceleration damage can be due to differential acceleration of the skull and the brain. This well-known phenomenon occurs because the brain is free to move within the skull and because it lags behind the skull for a brief moment after acceleration begins. The result is that the brain moves relative to the skull and the dura, causing strain on the subdural bridging veins. This is the mechanism that causes most subdural hematomas. Furthermore the movement of the brain away from the skull creates regions of low pressure (tensile strain) which if sufficiently intense cause contrecoup contusions. The second way that acceleration is injurious is that it produces strains within the brain itself. This is the mechanism for diffuse brain injuries (the concussion syndromes and diffuse axonal injury) and the so-called intermediate coup contusions. In diffuse brain injuries that are associated with structural damage, the acceleration loading causes sufficient strains within the brain so that the brain tissue itself is injured; in the case of intermediate coup contusions, the vascular tissue tolerances are exceeded.

The type of acceleration damage that occurs depends on the type of acceleration, the amount of acceleration, the direction of head motion, and the duration of the acceleration load. Three types of acceleration can occur: (1) translational acceleration occurs when the brain's center of gravity (roughly the pineal gland) moves in a straight line, (2) rotational acceleration occurs when there is movement about the center of gravity without the center of gravity itself moving, and (3) angular acceleration occurs when components of translational and rotational acceleration are combined. Here there is movement of the center of gravity in an angular manner. Rotational acceleration is a virtual impossibility in clinical situations since it requires that the head must pivot around an axis that goes through the pineal region. This

would mean that the whole body swings around the head. However, rotational acceleration may occur, namely with motion in a horizontal plane. Similarly, purely translational acceleration is uncommon, since this type of movement is very unphysiological, except perhaps in the case of acceleration in the superoinferior direction that results from a pure vertex impact. Because of the head-neck anatomy, angular acceleration is the most common type encountered clinically, the center of angulation most often being in the lower cervical spine. The exact location of the center of angulation determines the proportion of translation and rotation that the brain undergoes. As the center of angulation moves higher up the cervical spine, there is a greater rotational component, and when the center of angulation moves lower, there is proportionately more translational acceleration. Knowing the type of acceleration is important since it has been shown that concussive injuries do not occur if the head is not accelerated<sup>3</sup> or if it undergoes a purely translational acceleration,<sup>9</sup> but that concussion readily occurs when the head experiences angular acceleration.<sup>9</sup> Although it does not produce concussion, translational acceleration is injurious and can produce various focal injuries including cortical contusion, intracerebral hematoma, and subdural hematoma. It is not surprising, therefore, that situations exist where substantial brain damage has occurred without loss of consciousness.

However, considering the three types of head acceleration, angular acceleration is not only the most frequent, but the most injurious. Except for skull fracture and epidural hematoma, virtually every known type of head injury can be produced by angular acceleration.<sup>1</sup>

The amount of acceleration damage also depends on the magnitude of the acceleration loading. However, because of the viscoelastic nature of biological tissue, the response of the tissue is determined not only by the acceleration magnitude, but also by the rate at which the acceleration occurs. The acceleration magnitude can be viewed as proportional to the amount of strain delivered to the brain and the acceleration rate to the strain rate. The rate of acceleration varies inversely with the duration for which the acceleration is applied if the acceleration magnitude is constant, and varies directly with the acceleration magnitude if the acceleration duration is constant.

Three zones of interest are encountered as acceleration duration increases at a constant amount of acceleration. First, at very high strain rates (short acceleration durations) the properties of the brain are such that much of the acceleration effects are damped, and as a consequence, the brain actually experiences very little strain. Therefore, extremely high accelerations are required to produce injury. The second zone begins as the acceleration duration is increased slightly. Less damping occurs, and therefore it requires less acceleration to produce injurious strains within the head. However, the strain that occurs under these conditions is confined to the surface, since the acceleration is present for such a short time that the strains cannot penetrate deeply. The types of injury that can be produced in these circumstances are those at the brain surface, notably subdural hematoma. As the duration of acceleration increases further, the third zone begins; less of the inertial effects are damped, and the resulting strains are able to propagate deeper into

the brain. This can cause axonal injury and result in prolonged traumatic coma.

Strain rate also increases if, for constant acceleration duration, the acceleration magnitude is increased. In the first zone described above, the strain rate is already so high that increasing it further adds little to the injury pattern. In the second zone, vascular tissues at the brain surface are already jeopardized, and so increasing the strain rate further can exceed the vascular tissue tolerance and cause damage where none existed before or can increase the number of disrupted vessels. In the third zone, where strain rate produces damage to brain tissue but is insufficient to produce vascular damage, increasing the acceleration magnitude can increase the strain rate sufficiently to cause vascular damage.

Therefore, acceleration damage to the brain can be of several types, depending on the amount of acceleration, the duration of acceleration, and the rate at which acceleration is applied to the head. *Structural damage* to superficial vascular tissue, especially to bridging veins and pial vessels, occurs in high strain rate conditions (short acceleration duration), whereas brain tissue, principally axonal, damage occurs in lower strain rate, longer pulse duration circumstances. An intermediate zone exists in which both vascular damage and axonal damage occur. *Functional damage* without gross tissue disruption occurs at lower levels of strain, as is seen in cerebral concussion.

## Injuries and Their Mechanisms

This section summarizes the injury mechanisms for each of the several types of clinically important head injuries.

### **Skull Fracture**

*Linear Fracture* Linear fractures occur solely because of the contact effects due to impact. Acceleration (inertial) effects play no role. A linear fracture is caused by an impacting surface that is of intermediate size; it has to be sufficiently large so that skull penetration does not occur and sufficiently small so that the contact phenomena are not distributed widely over the head. Acceleration injuries may be superimposed, if substantial head motion occurs after impact.

*Depressed Fracture* Depressed fractures are similar to linear fractures except that the impacting surface is smaller. The contact phenomena are more focused, allowing skull perforation.

*Basilar Fracture* Basilar fractures are due to the remote effects of skull impact. Stress waves that propagate from the impact point or changes in skull shape due to impact cause them.

### **Epidural Hematoma**

Epidural hematoma, like skull fracture, is not related to head acceleration. Vascular disruption occurs because of contact-related skull fracture or contact-related skull deformation.

### Contusions

**Coup Contusions** Contusion beneath the site of impact is caused by local tissue strains that arise from local skull inbending. In order for such localized effects to occur, the impacting surface must be relatively small and hard. The failure of pial vessels most likely occurs because of high tensile strains that are produced when the focally depressed skull rapidly returns to its normal configuration.

**Contrecoup Contusions** Superficial focal areas of vascular disruption remote from the site of impact occur principally because of acceleration (inertial) effects. Brain motion toward the impact site causes tensile strains to occur at an area opposite from impact; if the tensile strains that result are larger than the vascular tolerance, contusion results. However, unlike coup contusions, impact is not necessary for contrecoup contusion to occur; the term *coup* is therefore a misnomer, since the critical mechanism is acceleration (or deceleration) and not impact. In situations where the head undergoes impulsive loading, contrecoup lesions occur solely because of the acceleration effects. If impact causes considerable skull distortion (due to stress waves), tensile strains can also occur remote from impact and cause contusional damage, but the predominant mechanism for contrecoup contusions is head acceleration.

The relative proportion of coup versus contrecoup contusions depends on the response of the head to impact, that is, on how much of the impact energy is converted into head motion. A hard, small impact surface (hammer blow) tends to produce focal skull deformation with underlying coup contusion, but since much of the energy is dissipated at the impact site, there is little head acceleration and consequently little or very small contrecoup contusion. On the other hand, a softer, larger impact surface (as seen in falls) results in less focal injury beneath the point of impact; more energy is converted into setting the head into motion. In this case a large contrecoup contusion results, the coup lesion being smaller or not present. It has been stated that coup lesions predominate if the head is accelerated, and contrecoup contusions predominate if the head is decelerated. This may be a useful clinical saw, but it is true only to the extent that *most* clinical situations in which the head is accelerated are injuries from small, hard impactors (assaults), and *most* deceleration injuries occur against broad surfaces (falls) or against softer surfaces (padded auto interiors). Thus most "acceleration" injuries have a greater proportion of contact phenomena and less acceleration than do "deceleration" injuries, where the proportion is usually reversed.

**Intermediate Coup Contusions** This name has been given to vascular disruptions on brain surfaces that are not adjacent to the skull. Although the mechanism of these lesions has not been extensively studied, it is likely that the lesions are due to strain concentrations that result from impact-generated stress waves. In some instances, however, brain movement due to acceleration effects may cause focal loading against internal bony or dural elements. This may be the mechanism for inferomedial temporal lobe (against the tentorium or petrous ridge) or cingulate (against the falx) intermediate coup contusions.

### Intracerebral Hemorrhage/Hematoma

Large traumatic intracerebral hematomas are often associated with extensive cortical contusions; these can be viewed as contusions in which larger, deeper vessels have been disrupted. Smaller hematomas that are not associated with contusion probably occur because of stress wave concentration due to impact or because of acceleration-induced tissue strains (tensile and shear) deep within the brain. Deep, medial, or paracentral small hemorrhagic lesions seen in prolonged coma are undoubtedly due to tissue and vascular tears at areas of strain concentration due to acceleration effects.

### Subdural Hematoma

Three varieties of acute subdural hematoma (SDH) are found clinically. The first two can be called complicated SDH—SDH associated with contusion and SDH associated with cortical laceration. These result from contact or acceleration effects that cause the primary lesion. The third type of SDH is most frequent and is due to disruption of surface vessels, usually bridging veins, and results entirely from inertial, and not from contact, forces. Because of the sensitivity of the bridging veins to high strain rate conditions and because of their superficial location, SDH results from head accelerations that produce short-duration, high-strain rate loading.<sup>5</sup> These conditions are best met in falls where the head strikes a broad surface; there is a distributed loading of the head so that little of the energy is dissipated by focal skull injury, and the deceleration causes tensile and shear strains of high strain rate to occur at the brain-skull interface. Not infrequently, subdural hematoma is associated with diffuse axonal injury (DAI; see below) because the mechanism of these two injuries is similar and often the two coexist. This explains those cases where SDH is small but the underlying brain damage is great.

### Cerebral Concussion

All gradations of concussion (transient neurological dysfunction due to trauma) are produced entirely by inertial forces. That is, brain motion causes these injuries, not the contact forces associated with impact. Therefore, for concussion to occur as an isolated injury, contact forces must be minimal. Often this is not the case, and the contact forces then may compound the concussion with contact injuries, most commonly skull fracture and contusion.

### Diffuse Axonal Injury

Axonal damage appears to be the pathological substrate of prolonged traumatic coma and, like cerebral concussion, is solely due to inertial effects and not to contact phenomena.<sup>6</sup> The amount and location of axonal damage probably determine the severity (duration and depth) of injury and depend on the magnitude, duration, and rate of onset of acceleration. DAI is produced by longer acceleration loading and loading with more gradual onset than loading which produces acute SDH. Thus DAI is most common when the

head is impulsively loaded or where impact occurs with relatively soft broad objects, such as occurs in accidents involving motor vehicle occupants. In fact, although both DAI and SDH are acceleration injuries, there is a marked difference in their causes. Almost all cases of DAI, especially its severe form, arise from vehicular injury (impact to padded dashboards, resilient windshields, energy-absorbing steering columns, etc.) where acceleration duration is long. Conversely, most cases of acute SDH occur because of falls or assaults where the impact duration is short and the deceleration abrupt.<sup>3a</sup>

Characteristic patterns of injury have been described and document areas where brain tissue tears or microscopic damage to axons occur.<sup>2,7</sup> The location of the damaged axons in DAI determines the specific neurological sequela of the injury and is very dependent on the direction in which the head moves during injury. Current data suggest that coronal plane head movement is much more injurious than sagittal plane movement.<sup>6</sup>

## References

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## Pathophysiology of Head Injury

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Craniocerebral trauma may produce direct impact injury to the brain with parenchymal contusion and laceration or with shearing of myelinated pathways in the white matter of the cerebral hemispheres and brain stem. These primary injurious processes may set in motion a train of secondary alterations in brain metabolism, intracranial hemodynamics, and brain water compartmentation which evolve during the hours following head injury. A satisfactory outcome for the head-injured patient requires recognition and successful treatment of these derangements. In addition, these evolv-

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ing pathophysiological processes can produce changes in the intracranial pressure-volume relationship with resulting intracranial hypertension and transtentorial and subfalcine brain herniation. Despite the apparent diversity of these derangements in brain physiology, the magnitude of the insult to the brain parenchyma is ultimately dependent on the degree of hypoxic/ischemic injury sustained by the neurons therein.

The skein of problems cited above may be further complicated by the systemic manifestations of head trauma which include abnormalities of water balance and hormonal function that follow hypothalamic or hypophyseal injuries. In this chapter we will present an overview of cerebral and systemic pathophysiological changes following head trauma; the dynamic interaction between altered intracranial hemodynamics, cerebral metabolism, brain oxygen tension, traumatic brain edema, and increased intracranial pressure is emphasized. The other chapters on cerebral edema, increased intracranial pressure, cerebral blood flow, and normal cerebral metabolism serve as useful background for this chapter.

## Cerebral Metabolism and Head Injury

The final common pathway of neuronal injury after head trauma is impairment of the delivery of oxygen and cellular metabolic substrates, especially glucose, to cellular sites.