INTRODUCTION

"No head injury is so serious that it should be despaired of, nor so trivial that it can be ignored".

Hippocrates

This statement concerning the uncertainty that prevails about the likely course of events after head injury is as true today as Hippocrates wrote it more than 2000 years ago (Kalayanaraman 1980). With increasing industrialization and more rapid methods of transport, the incidence and severity of head injuries are increasing. Trauma is a major health and social problem. It is the number one cause of death of persons from birth and 38 years in the United States and the number three killer overall. For every death, there are at least two permanent disabilities (Trunkey 1985).

In 1970, there were 114,000 deaths due to accidental injuries in the United States. There were 4.91 deaths per 100 million vehicular miles (Gurdjian 1972).

In 1976, there were 65,428,000 persons injured in United States. Of these, 7,560,000 had sustained head injuries (Caveness 1979). In 1977, "head injuries", including laceration and contusions of the face and head in the United States (Cooper 1982). Trauma accounted for 3,800,000 annual loss in working years of life, attributable to premature deaths due to injuries. In India, 800 persons are killed in accidents/100,000 vehicles (Sambasivam 1984).

Because trauma affects primarily young people and is associated with so many disabilities, the cost to society is staggering - 83.5 billion dollars per year or 227 million dollars per day in United States (Trunkey 1985).
Not until the last three decades, have large series of civilian head injuries been studied to identify factors of prognostic significance on a statistical basis (Tamas et al 1985). With the birth of modern neurosurgery and spurred on by two world wars, more refined attempts at managing head injuries were initiated (Dharmarajan 1987).

The uncertainty after mild injuries is different from that after severe damage. After mild injury, the problem is to know the likelihood of complications developing that can transform the case from a trivial to a life-threatening situation; the commonest complications are intracranial haematoma, infection and epilepsy. After severe injuries, the question is whether or not the patient will survive, and if he does what the likelihood is of persistent disability (Jennett et al 1975). The consequence of this uncertainty is that the management of head injuries depends more on intuition than on logically-based decisions. Thousands of mild injuries are admitted briefly to hospital every year, although the chances of a complication are very small in most of them. With severe injuries, an undue proportion of effort is liable to be expended on patients who do not survive or who are left so severely disabled that even members of the victim's family question whether it would not have been better if the patient had died soon after the accident. The price of intensive care, which saves the lives of many who would previously have died, is accumulation in the society of young people with permanent disablement. It is the lack of reliable statistics by which to predict the outcome after severe injury that leaves the doctor feeling that he must treat all patients as though they had potential for recovery and indeed many patients do make good recovery. The problem is whether these patients can be recognised soon after injury and maximum effort devoted to their care. Doctors dealing with patients with head injuries have to make a series of management decisions. Early decisions include whether or not to admit mildly injured patients to hospital, to which
department the more severely injured should go and whether or not expensive and elaborate investigations and treatment should be instituted or continued. While such decisions are common to the whole of medicine, they have unusual significance after head injury, because of the ultimate outcome varying from complete recovery to permanent crippling. If such decisions are to be made on logical grounds, they should depend on the clinician's estimate of the benefit likely to result from alternative courses of action.

The ability to predict the outcome of illness has always been an important aspect of the doctor's role in society; in previous times, when he could do little to influence the course of disease, his skill was largely judged by the accuracy of his forecasts. However, the availability in modern times of a wide range of therapeutic regimes in fact makes it all the more important to have accurate information about the natural history of disease (Jennett 1972).

Prognosis is of particular importance after severe head injury, because the outcome can vary so dramatically. Survival is now commonplace, but this reduction in mortality has been achieved only at the cost of expensive intensive care and at the cost of increasing number of disabled survivors, many of whom never work again and some of whom remain hopelessly crippled both mentally and physically.

What is a remarkable recovery? One must approach the definition of outcome after head injury in a dispassionate manner; this is difficult, because it is concerned not so much with an absolute state of health as with a comparison with the patient's previous state. But which previous state? A different view of recovery may be taken by the patient himself, who knows only how well he was before injury; by the doctor, who knows only how bad he was after injury; and by the family, who know something of both. Indeed, the terms used by doctors to describe recovery can be classified as optimistic or pessimistic. It seems
inescapable that those concerned with the care of severe head injuries must sooner or later accept it as a duty, both on humanitarian and on economic grounds to try to identify those patients who have a reasonable prospect of recovery and to concentrate available resources on the treatment and rehabilitation of those patients. Such an approach implies that management decisions such as 1) how vigorously to treat and 2) how long to continue vigorous treatment should be based on a prediction of the ultimate outcome.

It has been considered difficult to predict, soon after injury, what a patient’s ultimate outcome will be. The progress that has been made to resolve this problem in the last decade is partly the result of applying more standardised methods to assess the initial severity of brain damage and the degree of recovery. It also reflects the introduction of computers as tools to store data about large number of patients, to which statistical techniques can then be applied to calculate prognostic probabilities on the basis of recorded past experience (Jennett 1972, Teasdale and Jennett 1981).

Prognosis may concern itself with different kinds of prediction, expressed as a probability. The earlier a prediction is made, the less likely it is to be accurate, but the longer it is delayed the less useful it will be. Clearly what is required is an early prediction which attempts only to forecast large outcome classes.

To be practical, a predictive system has to be based on a relatively limited number of items of information about the patient (Jennett 1979). A patient’s course and outcome following a severe head injury can be influenced, and often irrevocably altered, by several non-neurological factors, such as associated somatic injuries and medical complications. The unpredictability of these influences renders even the best prognostic system imperfect. Outcome predictions altered by such complications always err toward over-optimism. Although undesirable, such over-optimistic predictions can be accounted for and do not
have adverse implications with regard to patient management. However, far more serious are errors of undue pessimism. These are not related to unexpected complications and represent an inherent weakness of the prognostic system. The optimal prognostic indicant or combination of indicants would, therefore, predict outcome with a high degree of accuracy and reliability and err only toward over-optimism (Narayan et al 1981).
MECHANISMS OF HEAD INJURY

The development and application of sophisticated techniques of physics, chemistry and biomedical engineering have helped to elucidate many problems in the mechanics of head injury.

Experimental studies

The modern application of physics to head injuries was started by Holbourn (Kalyanaraman 1980) and within two or three decades biomedical engineers have become interested in this field. Several experimental models have been used to study the mechanisms of injury to the brain. Sheldon and Pudenz and Sheldon used a lucite calvarium for the direct observation of the movement of the brain during cranial trauma. Ommaya et al. and Gosch et al. described the lexan calvarium as an improved method for direct observation of the brain. Hodgson et al. studied distortions occurring at various instants during the impact using intravascular contrast media or lead tags embedded in the brain, with flash X-ray exposures of the order of nano seconds. The use of pressure transducers, accelerometers and high-speed cinematography has helped to elucidate many of the phenomena occurring during acute head injury. The use of plastic containers filled with 1 percent solution of millin gyellow which can be seen with the polariscope to become doubly refractile in areas of stress has helped the understanding of shear stresses. High speed photographic instrumentation taking 240,000 pictures per second was used to study a simplified model of a skull and brain under dynamic loads. Extensive research is being carried on by electrophysiological, electron microscopic and neurochemical techniques to elucidate the mechanisms concerned in brain injury. Synaptic transmission may be affected
in head injury due to the failure of astrocytes to maintain a constant extracellular environment in the brain. A striking accumulation of glycogen in astrocytes has been shown to occur following an acute oxygen and glucose deprivation.

Trauma to the head may result when the moving head hits against an object or when a moving object impinges on the head. The speed and mass of the moving object and the direction of the force of impact and its spread may vary greatly resulting in injuries that are trivial or disasters. The force of injury is taken up by the movement of the head or the neck where possible, by movement of the scalp, by the absorption of the force by the skull and by movement of the dura and all intracranial structures including the brain and blood vessels along the direction of the force of the impact. This is followed by cessation of movement of the dura and the intracranial structures. In addition to the direction and force of the injury, the physical properties of the skull, the dura, the dural partitions, the brain and its blood vessels determine the type of injury suffered (Kalyanaraman 1980).

To understand how an individual mechanical input to the head results in a particular type of head injury, one must consider multiple factors. 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. 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 (Gennarelli and Thibault 1985).

Static and Dynamic head injuries

The head may be hit while it is stationary and unable to move because of a rigid support eg. a brick falling on the head of the man sleeping on the floor, the wheel of a car
passing over the head of a pedestrian fallen on the road. Under these conditions, there is no relative movement of the brain within the skull and no force of acceleration and deceleration acting on the brain. This type of injury is static head injury.

The vast majority of head injury belong to the group referred to as dynamic head injury. Here the stationary head is brought into motion either because of the movement of the whole body (e.g. falls from heights, being thrown off a scooter, etc.) or by an object hitting the head directly (acceleration injury). There is often a rotating movement of the head in relation to the neck (Lindgren et al 1973). More often, the injury is due to the head being brought to rest by hitting the ground or another object, as in traffic accidents (deceleration injury) (Gurdjian and Gurdjian 1978). Several forces are brought into play during such violent movements (Kalyanaraman 1980). Thus input can be either slow (static loading) or rapid (dynamic loading). In static loading, the injury forces are applied gradually, usually over 200 milliseconds or longer. In the most frequent dynamic loading, the injury forces act in less than 200 milliseconds. Dynamic loading can be of two types, namely 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. 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 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.
Effect on the skull

Immediately beneath the point of impact, there is localised skull deformation, with inbending of the skull surrounded by outbending of the skull peripheral to the impact site (Gurdjian et al 1966). If the degree of local skull deformation is significant, penetration, perforation or fracture of the skull occurs. Although the skull of adults is less elastic than that of children, it will bend when a force of sufficient magnitude is applied. The bone will break when the degree of bending reaches the limit of its tensile strength. The table of the bone which is on the convexity of a bend is subjected to the maximum stretch and will fracture first. A local deformation of the skull occurs when a small object hits the skull with sufficient energy. The bone is bent inwards as a cone and at the apex of the cone, the inner table is stretched and breaks. At the circular base of the cone, the outer table is bent maximally and breaks. Thus a typical depressed fracture is produced. If the impinging force is large and acts over a small area, the fractured pieces of bone may get detached and be carried inside the brain. In addition to local deformation, fractures are also produced by general deformation. Even though a force tends to travel in the direction in which it was initiated, it also tends to follow the lines of least resistance and splits into components avoiding stout barriers of bone. Fractures of the base of skull may be produced by forces applied directly at the level of the base, by general distortion of the skull, by extension of a fracture from the vault and by a force applied to the base through the spinal column or face. Rarely, a tangential injury to the skull by a sharp object may slice off a part of the vertex resulting in an elevated fracture (Kalyanaraman 1980). Patients with upper facial fractures are at greatest risk for serious closed head injury (Lee et al 1987).
Effects on intracranial structures

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 (Lindenberg 1966). Three types of strain can occur: compression, tension 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.

Mechanistic causes of head injuries

The local contact effects of head injury produce skull fracture (linear or depressed), extradural haematoma and coup contusions. The remote contact effects produce skull distortion and shock waves; both contribute to vault fractures, basilar skull fractures, contrecoup injuries and intermediate coup contusions (Gurdjian 1972).

Acceleration injuries produce damage by surface strains or deep strains. Surface strains result in subdural haematoma, contrecoup contusion and intermediate contusion. Deep strains produce concussion and diffuse axonal injury. (Gennarelli and Thibault 1985).

Parts of the brain may move in relation to each other and in relation to the anchoring blood vessels. The moving surfaces of the brain come into forceful contact with the parts of the skull. The frontal and temporal lobes are often injured due to their impact against the irregular bony surface of the anterior and middle cranial fossa and the sphenoidal ridges. The dural septa resist the movements of the brain and thus the falx and
the tentorium play a significant part in the development of primary brain contusions. Sudden suction effects due to acceleration and deceleration produce negative pressure in the region opposite to that of the moving force. When the negative pressure exceeds one atmosphere, small areas of cavitation with old foci of destruction and haemorrhage occur. Stretching and shearing forces cause movements of different layers of the brain in relation to one another resulting in rupture of the fibre tracts. Pressure gradients develop particularly at the craniospinal junction, resulting in extrusion effects and movement of the brain in relation to the spinal cord. Injury to blood vessels may lead to their rupture, spasm or thrombosis. Venous obstruction aggravates oozing and oedema while arterial obstruction causes ischaemia and infarction.

Pathophysiology of head injury

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 haemodynamics and brain water compartmentation which evolve during the hours following head injury.

Cerebral metabolism and Head injury

The final common pathway of neuronal injury after head injury is impairment of the delivery of oxygen and cellular metabolic substrates, especially glucose to cellular sites. Cellular hypoxia results when there is a deficient amount of oxygen necessary for maintaining aerobic glycolysis. In this state of deficient oxygen delivery and tissue hypoxia,
anaerobic glycolysis predominates. Tissue oxygen deprivation severely limits the generation of ATP which is necessary for maintenance of brain energy requirements.

Cerebral \( \text{O}_2 \) and \( \text{CO}_2 \) tension

The brain is totally dependent on continued supplies of oxygen and glucose. As an organ, the brain represents approximately 2 percent of adult body weight; it receives approximately 20 percent of the cardiac output and utilizes approximately 15 to 20 percent of all oxygen and glucose consumed by the entire body per unit time. Neurons with an established vulnerability to hypoxia/ischaemic insult are localised to the following sites: cerebral cortical pyramidal cells in layers 3, 5 and 6; hippocampal pyramidal cells in areas h1, h3, h4 and h5; cerebellar purkinje cells and focal scattered cells in the basal ganglia and thalamus. Furthermore, regional free radical formation and acidosis may add further to focal membrane insult. Oxygen reaches the neuron by the process of simple diffusion. Carbon dioxide and hydrogen ions have a significant influence on \( \text{O}_2 \) availability to cerebral cortical tissue. Increased blood flow under conditions of hypercarbia and tissue acidosis is altered to a variable extent in injured brain.

Intracranial haemodynamics following head injury

Cerebral blood flow (CBF) measurements taken early after human head injury have demonstrated a wide range of values from 20 ml per 100 g brain per min to 65 ml per 100 g brain per min. The prognosis of head injured patient with low or high CBF is poor (O'Connor 1983). Acutely, head trauma produces impairment of cerebrovascular autoregulation either locally with isolated injury or diffusely with more severe injury and may result in diffuse vasodilation and an increase in intracranial blood volume, particularly
in children. A relatively normal range of arterial blood pressure is required for autoregulation of CBF. The system does not work properly when the perfusion pressure falls below 60 mm Hg (torr). When the pressure rises above 150 mm Hg in an otherwise normotensive subject, the arterioles become dilated beyond their autoregulatory constrictive capacity. Autoregulation is easily abolished by trauma. Dissociation between cerebrovascular autoregulation and CO$_2$ response is common. Injury to brain frequently traumatises its vasculature. Small vessels may be directly injured. Vascular spasm and microthrombi may form, resulting in the "no-reflow" phenomenon. Cerebral vasodilator stimuli tend to increase intracranial blood volume, which in turn raises intracranial pressure (ICP). In severe trauma, increased flow caused by the dilatation of small arteries and arterioles lasts longer than in minor trauma. The CBF in such instances can be two to five times greater than that of normal gray matter. It actually represents a luxury perfusion. Severe injury to the brain stem results in marked luxury perfusion, yet cerebral O$_2$ consumption and cortical electrical activity are greatly reduced. Later on the vasopressor mechanism begins to fail. This is accompanied by profound cerebral ischaemia and diminished CBF. Eventually the difference between the arterial and intracranial pressures declines. In the final stage of cerebrovascular decompensation, intracranial and arterial pressures are inseparable and CBF is non-existent. The patient passes into shock and respiratory arrest occurs. (Bakay and Glasauer 1980).

**Traumatic brain oedema**

Brain oedema is a frequent concomitant of serious head injury. Klatzo divided brain oedema into two types: vasogenic and cytotoxic (Long 1983). Vasogenic oedema refers to transvascular leakage of solutes and water into the extracellular space and cytotoxic oedema
refers to intracellular fluid expansion in response to cellular toxins or injury. The vasogenic oedema originates from abnormalities of the injured vasculature. Following head injury, an alteration in blood brain barrier occurs and protein-rich vasogenic oedema fluid results; this oedema fluid appears to collect preferentially in the white matter (Tandon 1984). In cytotoxic oedema, there is cellular swelling secondary to exposure to toxic substances. The effects of brain oedema may be manifested in different forms after head injury. This may produce a worsening in a neurological condition or the occurrence of a herniation syndrome. Differential pressures within the brain secondary to the mass effect of oedema can produce herniation of brain tissue beneath dural folds that compartmentalise the intracranial cavity. Herniation appears to be an attempt to compensate for the development of significant pressure gradients within a swollen brain limited by a cranial compartment of fixed size. Once brain tissue has herniated, the circulation of the tissue is compromised and the degree of swelling due to ischaemia increases and perpetuates the ongoing pathological processes. Posttraumatic brain oedema is one of the commonest and more important sequelae of head injury (Misra et al 1984).

Intracranial pressure

The intracranial compartment in adults is of fixed volume. As compensatory mechanisms which dampen initial increases in intracranial pressure (ICP) fail, intracranial compliance decreases and elastance increases. During this phase, the baseline ICP may only be slightly increased or normal. As ICP continues to increase, compliance decreases further and treatment modalities are less successful in controlling further elevations in ICP. Episodic increases of ICP lasting for fractions of an hour may occur on an already elevated base; these plateau waves are correlated with deterioration of neurological status. The
final stage in the decompensation of protective mechanism occurs when complete vasomotor paralysis of the cerebral vasculature develops. In this phase, there is no responsiveness to treatment modalities and the elevation of ICP is sustained. The development of vasomotor paralysis is ominous. (Popp and Bourke, 1985).

Primary traumatic brain lesions

Concussion

Impact acceleration of the head sets up forces which result in direct brain deformation and in cerebral pressure gradients producing shear stresses and indirect tissue distortion. These forces exert their predominant effect on brainstem sites. Mechanical stresses produce a sudden neuronal depolarisation or excitation, followed by a period of nerve cell paralysis and transmission failure. If the initially high posttraumatic cerebral metabolic demands are met, neurological symptoms will be mild and of brief duration. Secondary complications of hypoxia and reduced cerebral blood flow, however may result in net energy failure and the production of more severe or long lasting deficits. Considerably more force is needed to produce concussion. (Shelton and Demakis 1979, Ward Jr. 1966, Parkinson 1983).

Contusion

Contusions are bruises of the neural parenchyma. Most commonly involving the crown of a gyrus, they tend to be wedge shaped. They represent extravasation of erythrocytes about small lacerated vessels within the neural parenchyma. Contusions are classified as coup, contrecoup, intermediate coup, gliding herniation and fracture contusions.
Laceration

Brain lacerations are physical disruptions of the brain integrity at the macroscopic level.

Intraparenchymal haemorrhages

They occur most commonly in the cerebral hemispheres. Cortical contusions, lacerations or subdural haematoma may be present along with the intraparenchymal haemorrhage. (McCormick).

Extracerebral haemorrhages

Epidural (extradural) haematoma, subdural haematomas and subarachnoid haemorrhage may occur. Acute subdural haematomas are very often associated with focal traumatic brain lesions.

Direct brain stem injury

Contusions and lacerations of the brain-stem occur most commonly in association with traumatic lesions elsewhere in the brain. However they can occur as essentially solitary lesions. Common sites are pons and pontomedullary junction (Tandon 1964, Britt et al 1980).

Diffuse axonal injury

In acceleration/deceleration injuries of the head, when these forces are large, there is widespread destruction of axons in the white matter in the cerebral hemispheres and
brain stem accompanied by prolonged coma. Haemorrhagic lesions in midline structures, particularly in corpus callosum are common. Additional haemorrhagic areas in internal capsule, peri-third ventricular gray matter and dorsolateral upper brain stem can occur. The haemorrhages are asymmetrical in the two hemispheres. (Langfitt et al 1983).

Secondary traumatic brain lesions

The brains of victims of fatal closed head trauma typically confirm a variety of vascular/ischaemic lesions that are secondary to increased ICP, herniation induced vascular occlusions or systemic hypotension. The major types of secondary posttraumatic brain damage are: medial temporal (parahippocampal) necrosis, focal infarction due to specific artery compression eg. posterior cerebral artery, border zone infarcts and focal hypoxic damage, diffuse ischaemic necrosis of neurons and secondary brain stem (Duret) haemorrhages.

Herniation

Primary injuries of brain are frequently localised and produce swelling and bleeding. The mass effect of such lesions predisposes the damaged brain tissue to herniate from one cranial compartment to another (Hardman 1979). These brain hernias abut against the free margins of the falx cerebri and tentorium cerebelli or impact foramen magnum and dorsum sellae. Subfalcial hernias may produce shift of anterior cerebral arteries. In transtentorial herniation, oculomotor nerves are tethered against the posterior cerebral arteries and dislocates midline penetrating arterial branches supplying the tegmental gray matter of the midbrain and rostral pons. Shifts of the midbrain may push the cerebral peduncle opposite the uncal hernia against the tentorial margin and produce Kernohan’s
notch. Herniation of the cerebellar tonsils into the foramen magnum compresses the adjacent medulla oblongata. Upward herniation of upper part of vermis of cerebellum through the tentorium can occur.
C.T. APPEARANCES IN HEAD INJURIES

Computed tomography has revolutionised the diagnostic evaluation of intracranial disorders in general and traumatic intracranial lesions in particular. CT is noninvasive, rapidly provides accurate information regarding the presence, extent and nature of intracranial lesions resulting from trauma. It enables identification of collections of blood in a manner previously not possible (Peyster and Hooover 1985). The unique ability of CT to detect subtle differences in tissue density in a noninvasive manner in a short period of time has proved valuable in the detection of various traumatic intracranial lesions, especially haematomas, (Kalyanaraman 1981, Kishore et al 1987).

A. Extracerebral injury

1. Epidural haematoma

Acute epidural haematoma (EDH) appear in the CT scan as biconvex, rarely planoconvex zones of increased density adjacent to the calvarium. Haematomas which still contain fresh uncoagulated blood at the time of examination demonstrate areas of diminished or normal tissue density within the haematoma itself. In very rare cases of disturbed haemostasis, the haematoma is differentiated from surrounding brain tissue by a delicate filmy contor. The displaced dura is otherwise sharply delineated against brain tissue (Lanksch et al 1979).

2. Acute subdural haematoma

Acute subdural haematoma (SDH) appears as sickle shaped zones of increased density over large portions of a hemisphere. Displacement of midline structures and associated parenchymal brain injury may be present.
3. Subdural hygroma

Subdural hygroma appears as CSF dense zones under the skull vault. Associated cerebral contusion may be present.

B. Traumatic brain lesions

1. Cerebral contusion

Contusion appears in CT scan as nonhomogeneous high density zones with attenuation values of 50 to 60 HU. This appearance is due to the presence of multiple small areas of haemorrhage within the brain substance interspersed with areas of oedema and tissue necrosis.

2. Intracerebral haematoma (ICH)

This is seen as a well-circumscribed homogeneous high-density zone with an attenuation of 70 - 90 HU, usually surrounded by areas of low density due to oedema. (Kishore et al 1987).

3. Subarachnoid haemorrhage

Subarachnoid haemorrhage (SAH) appears as curvilinear areas of increased density conforming to the basal cisterns, convexity sulci and interhemispheric fissure or as a layer of increased density on the tentorium (Peyster and Hoover 1982).

4. Intraventricular haemorrhage

With CT, intraventricular haemorrhage (IVH) is readily recognised as a collection of increased density conforming to the ventricular system.

5. Brain Swelling

Brain swelling may be due to brain oedema or due to posttraumatic vascular engorgement. CT scan may demonstrate diffuse or irregular areas of decreased absorption values or slightly increased absorption values. Generalised cerebral swelling can grossly
appear almost normal, except for compression of ventricle, cisterns and convexity sulci. With diffuse oedema, cerebral density may be slightly lower than usual. Vascular engorgement may manifest itself by overall increased cerebral density.

6. Diffuse axonal injury

Diffuse axonal injury (diffuse white substance injury) is characterised by small focal areas of haemorrhage in the corpus callosum, adjacent to third ventricle and deep in the cerebral hemisphere (Perini et al 1984).

C. Cerebral herniations

The major forms of herniations of brain are descending transtentorial, ascending transtentorial, tonsillar, subfalcine and transcalvarial herniations (Dharmarajan 1989, Greenberg 1984).

1. Uncal descending transtentorial herniation

The CT findings are distortion of the lateral aspect of the suprasellar cistern, displacement of the brain stem toward the contralateral side with increase in the width of subarachnoid space between the mass and ipsilateral free tentorial edge, medial stretching of the ipsilateral posterior cerebral artery seen on the enhanced CT scan, occipital hypodense lesions, representing calcarine infarctions, brain stem hyperdense lesions representing haemorrhage, distortion of the elongated U shaped tentorial incisura and contralateral temporal horn widening (Osborn 1977 and Stovring 1977).

2. Ascending transtentorial herniation

Upward herniation of the superior vermis and accompanying blood vessels through the tentorial notch causes midbrain compression. Compression of the quadrigeminal plate cistern by the superior vermis causes the cistern to appear flattened with posterior
indentation. In most advanced stage, the following changes are noted; effacement of the superior cerebellar and quadrigeminal cistern, flattening or reversal of the curvature of the posterior third ventricle and obstructive hydrocephalus (Osborn et al 1978).

3. **Tonsillar herniation**

   This is accompanied by obstructive hydrocephalus.

4. **Subfalcine herniation**

   The CT findings are compression rotation of frontal horn and displacement across the midline, effacement of the anterior portion of the suprasellar cistern, contralateral shift of the anterior cerebral artery, cingulate gyrus hypodensity, stretching and bowing of the falx, frontal hypodensity consistent with anterior cerebral artery hypodensity.