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**Hair Analysis** *See* **DNA:** Hair Analysis; **Substance Misuse:** Hair Analysis

# HEAD TRAUMA

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**Pediatric and Adult, Clinical Aspects**
**Neuropathology**

## Pediatric and Adult, Clinical Aspects

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### Introduction

This article considers the clinical and pathological features of head injury in adults and children.

Globally head injury is a major problem for health services, not only in the industrialized but also in the developing world. Every year in the UK, about 1 million patients are admitted to hospitals with head injuries of varying severity. Sixty-three percent of adults who sustain a moderate head injury and 85% who sustain severe head injuries will be disabled 1 year afterwards. Patients are frequently left with significant psychological and physical problems, which have major social and economic ramifications. A large proportion of patients who sustain head injuries of significance will not be able to reintegrate into their former lives and occupations and will remain unemployed in perpetuity. The cost of head injury to society at large is enormous and a considerable burden is placed not only upon the acute hospital services but also upon rehabilitation facilities and social services.

### Epidemiology

There are of course many ways in which head injuries can occur, but in the UK 41% of injuries are attributable to falls, 30% to assaults, and 13% occur as a result of road traffic accidents (Figure 1). In the UK, head injuries account for 1% of all annual deaths, but importantly this figure represents 15–20% of all deaths occurring in the 5–35-year age group.

### Classification of Head Injuries

A variety of systems and methods have been devised for the classification of head injury. One of the most logical and straightforward is that devised by The Working Party on the Management of Head Injuries proposed by the Royal College of Surgeons of England in 1999. Three categories are outlined:

1. Minor head injuries are defined as those when patients are admitted to hospital for less than 48 h.
2. Intermediate head injuries are defined as those when patients are admitted to hospital for more than 48 h but do not require intensive care or cranial surgery.
3. Severe head injuries are defined as those patients who require intensive care or operative neurosurgical interventions.

This classification is clinical and based on the state of the patient at presentation but does not take into account the eventual outcome.

**Figure 1** Road traffic accidents, especially involving motor cyclists, are common causes of head injury.

## The Pathology of Head Injury

Brain damage following head injury is conventionally divided into primary damage, which occurs at the moment of impact, and secondary damage, which results from processes that are initiated at the time of impact.

Primary damage includes scalp lacerations, skull fractures, contusions and lacerations of the brain, diffuse axonal injury, and intracranial hemorrhage. Secondary damage includes brain swelling, raised intracranial pressure, ischemia and hypoxia, infection, and epilepsy.

Another important distinction to make is between closed nonmissile head injury and penetrating head injuries.

### Primary Damage

**Scalp wounds** The scalp is very vascular and it should be appreciated that wounds may bleed profusely and this can result in massive external blood loss. Scalp wounds are important as they may indicate the site of injury. Their size and location should be recorded carefully. They may overlie a depressed fracture of the skull, thereby making the injury compound.

**Skull fractures** Skull fractures are present in 3% of people attending an accident and emergency department. Many studies in adults have shown that the more severe a head injury, the more likely it is to be associated with a fracture. Thus, 65% of patients who are admitted to neurosurgical departments have skull fractures and 80% of fatal head injuries will have evidence of a fracture. Fractures occur in 62% of patients with severe head injuries and extend into the skull base in 77% of those patients. Isolated fractures of the skull base occur in 5% of patients who have sustained a severe head injury. By definition, a depressed fracture is said to have occurred if the fragments of the inner table of the skull are depressed by at least the thickness of the diploe. A patient who sustains a head injury that is associated with a skull fracture has a greater chance of having an intracranial hematoma than one who has not sustained a skull fracture.

A depressed fracture is compound if there is an overlying scalp laceration. Such injuries are said to be "penetrating," if there is in addition a breach in the dura mater. It should be remembered that basal skull fractures involving the paranasal air sinuses or middle ear cleft are technically compound and such injuries may be associated with cerebrospinal fluid (CSF) rhinorrhea and otorrhea, respectively. Fractures of the middle cranial fossa can be associated with CSF rhinorrhea rather than otorrhea, if CSF passes through the eustachian tube into the nasopharynx.

**Contusions and lacerations of the brain** Contusions represent areas of focal brain damage and are essentially areas of bruising of the brain. Contusions occur when the brain impacts against the bony protuberances that make up the skull base and, to a lesser extent, areas within the skull vault (Figure 2). A number of studies have shown that, no matter where the point of impact is on the skull, contusions tend to predominate in the frontal and temporal lobes. A contusion typically involves the crest of a gyrus, which will appear hemorrhagic and swollen on macroscopic examination. Old contusions in head-injury survivors appear as shrunken, yellowish-brown areas known as "plaques jaunes." The significance of contusions is their capacity to excite cerebral edema in the adjacent brain and by so doing cause a rise in intracranial pressure (Figure 3). A coup contusion occurs at the site of impact, whereas a

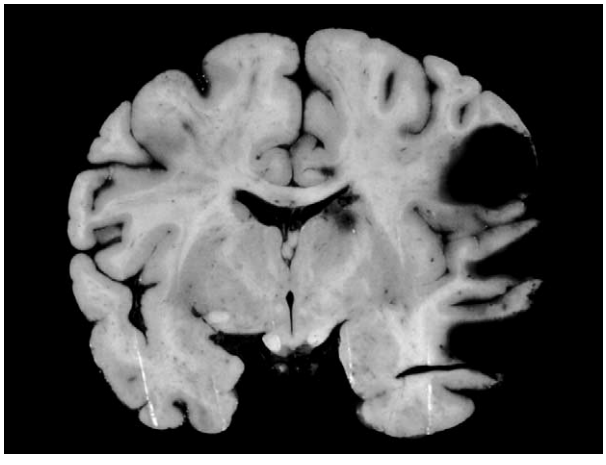

**Figure 2** This man fell downstairs one night after coming back from a bar and was found dead the following morning. This coronal section shows frontal and temporal contusions.

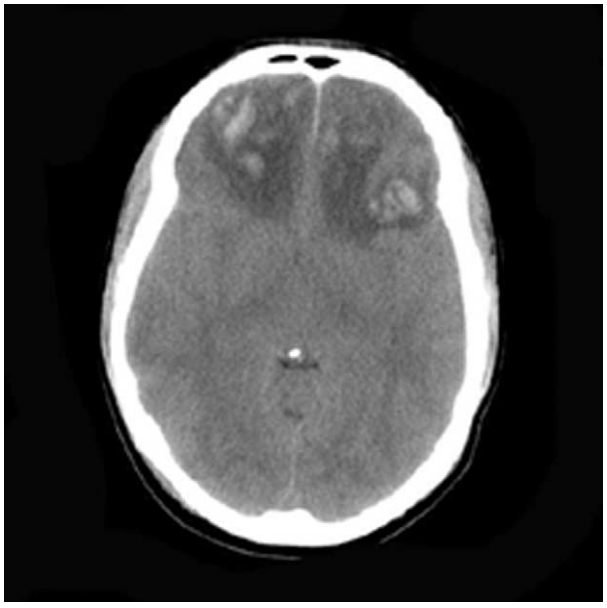

**Figure 3** This computed tomography head scan shows bilateral frontal contusions. Note the edema surrounding the contusions as well as effacement of the normal sulcal pattern and basal cisterns.

contrecoup contusion occurs at a point diametrically opposite to the point of impact. Typically with contusions, the leptomeninges remain intact but if they are torn, a laceration of the brain will occur. An area of confluent contusions in which the leptomeninges have been torn and bleeding has occurred into the subdural space is often referred to as a "burst lobe." Given the known predominance of contusions in the frontal and temporal lobes of the brain, burst frontal and temporal lobes tend to predominate.

Various attempts have been made to classify contusions, some of which are useful in interpreting the pattern of injury, but others tend to be obfuscating or noncontributory. Useful concepts include fracture contusions that occur at the site of a fracture, and herniation contusions that occur along the medial aspect of the temporal lobes or on the cerebellar tonsils. One of the more confusing terms that may be encountered is the "gliding contusion," which occurs as a result of rotational injury and typically involves the superior surfaces of the cerebral hemispheres. The distinction between a gliding contusion and diffuse axonal injury can sometimes be difficult to make.

**Diffuse axonal injury** Sabina Strich, who was working in Oxford in the 1950s, was the first to describe the pathological features of what is now referred to as diffuse axonal injury (DAI). This term was coined by Hume Adams in Glasgow and is now universally accepted. Sabina Strich studied a cohort of patients who had sustained severe head injuries, many of whom had remained in a persistent vegetative state until death or were certainly severely disabled. At autopsy, she observed a characteristic concatenation of macroscopic and microscopic lesions, the precise appearances of which depended upon the interval between the injury and death. Lesions of DAI injury typically occur in two stereotyped anatomical sites:

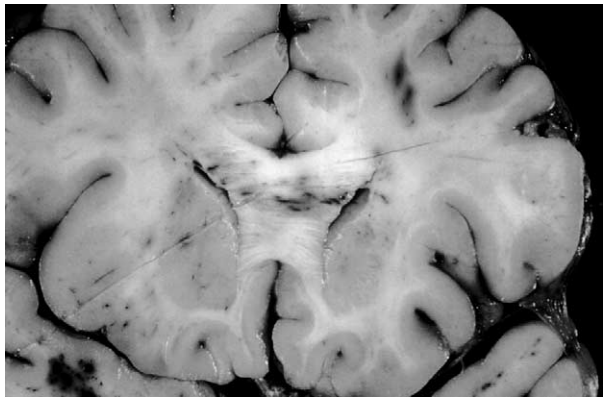

**Figure 4** This young woman was involved in a high-speed road traffic accident. She lost consciousness at the scene and never regained it. At autopsy she had typical features of diffuse axonal injury. Note the typical area of hemorrhage within the corpus callosum.

1. Within the corpus callosum, typically to one side of the midline and extending over a variable anteroposterior distance (Figure 4)
2. Within the dorsolateral quadrant of the rostral brainstem, typically in the vicinity of the superior cerebellar peduncles (Figure 5). If a head-injured patient who has sustained a DAI survives for only a few days, lesions in the anatomical locations described above usually have a hemorrhagic appearance, but with the passage of time all that may be visible macroscopically is a shrunken area of scarring. As is implicit in the name, DAI is "diffuse" and will not merely be confined to the dorsolateral quadrant of the brainstem and the corpus callosum.

Microscopically, evidence of diffuse damage may be noted throughout the brain. Histologically, the appearances of DAI again vary according to the length of survival following head injury. If death occurs within a few days of injury, the characteristic appearance of retraction balls will be seen in silver-stained preparations (Figure 6). These balls represent an extravasation of axoplasm from torn axons. If the patient survives for 2–4 weeks, microglia will infiltrate the areas involved, forming what is often referred to as microglial stars. Astrocytes and lipid-filled macrophages will also be seen amongst these clusters of microglia. In patients who survive for a

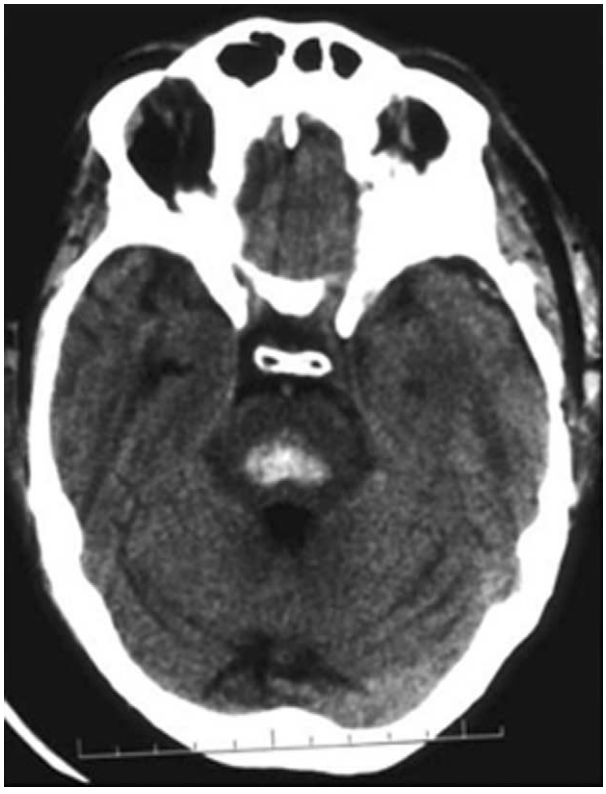

**Figure 5** An area of hemorrhage is seen in the midbrain that is typical of diffuse axonal injury. This young motorcyclist remained in a persistent vegetative state after a head injury.

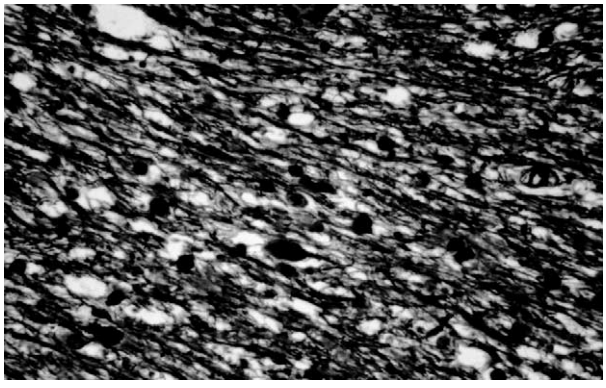

**Figure 6** This photomicrograph shows a section through the corpus callosum stained with a silver preparation. Retraction ball formation is well demonstrated. Original magnification ×400.

prolonged time, stains for myelin will show demyelination of the involved tracts. In severe cases of diffuse axonal injury, ventricular dilatation is often pronounced due to loss of adjacent white matter. Patients who have sustained diffuse axonal injuries form a distinct clinicopathological group. They tend to be unconscious from the time of impact and remain so and have a low incidence of skull fracture, contusion, and intracerebral hematoma. Moreover, the likelihood that such patients will have raised intracranial pressure is significantly lower than in those who sustain contusional injuries.

### Secondary Brain Damage

It must be accepted that other than preventive measures, nothing can be done to influence the extent of primary brain injury. The goal of all clinicians treating patients who sustain head injuries should be to prevent or minimize secondary brain damage by appropriate and timely intervention. It is convenient to classify the etiology of secondary events that cause brain damage into intracranial and extracranial insults.

## Extracranial Insults

### Hypoxemia

This may be attributable to damage to the brainstem which results in decreased respiratory drive or centrally mediated pulmonary edema. Furthermore, pulmonary complications such as pneumothorax, aspiration pneumonia, hemopneumothorax, or rib fractures, particularly when associated with a flail segment of the chest wall, can all contribute to hypoxemia. This often leads to a decrease in the patient's level of consciousness and moreover can in itself contribute further to cerebral edema. Head injuries are often associated with multiple trauma, which is frequently associated with serious internal or external blood loss. This can result in the development of hypotension leading to decreased cerebral function. The combination of hypoxemia and hypotension is particularly lethal and must be recognized and treated promptly if the patient is to survive. Hypotension can result in neuronal necrosis or in areas of boundaries zone infarction in the watershed areas of profusion between the main arterial territories.

## Intracranial Insults

### Intracranial Hematoma

These should be regarded as secondary phenomena even though the initiating event that results in their development occurs at the time of injury. The accumulation of a hematoma takes time and the injurious effects of such lesions are attributable to their capacity to produce raised intracranial pressure and internal herniations of the brain, e.g., subfalcine, transtentorial, and cerebellar tonsil herniation.

### Extradural Hematoma

Classically these arise from damage to the middle meningeal artery (Figure 7). In fact, approximately

70% of extradural hematomas are due to arterial bleeding and the remainder are due to damage and resultant bleeding from the venous sinuses of the dura mater. Extradural hematomas are typically situated in the temporoparietal region and are associated with a fracture of the temporal bone with resultant damage to the middle meningeal artery. They may, however, be bilateral or occur within the posterior cranial fossa. A skull fracture is present in 85% of cases in adults.

Extradural hematomas are often associated with minimal primary brain damage and consequently with timely surgical intervention, they are associated with a good prognosis. Unfortunately, the overall mortality from extradural hematomas is in the region of 30% and this relates to delays in diagnosis and transfer to neurosurgical facilities as well as their development in those patients who do have more severe primary brain damage.

One of the important points for the pathologist to note at the time of autopsy is that artefactual extradural hematomas can occur in fire-related deaths where the head has been subject to intense heat. The effect of such heat is to produce fissure fractures of the skull and, as a result of this, blood may extravasate into the extradural space following contraction of the meninges. The macroscopic appearances of such a hematoma are distinct from those of extradural hematomas of traumatic origin. In those extradural hematomas that are associated with fire damage, the hematoma tends to be pink and spongy, compared with the dark-red appearance of lesions of traumatic origin. Despite this, interpretation of extradural hematomas in the presence of fire damage can be difficult and may obviously raise the possibility of criminal activity, where it becomes important to distinguish the possibility of the victim having sustained a blow to the head, which was followed by an attempt to destroy the body by incineration.

### Subdural Hematomas

These are classified as intradural lesions along with intracerebral hematomas of traumatic origin. Subdural hematomas are more common than extradural hematomas and typically occur when veins that traverse the subdural space en route to the venous sinuses of the dura mater are disrupted (Figure 8). Subdural hematomas are in general associated with a far greater degree of primary brain damage than extradural hematomas. In 13% of cases, however, subdural hematoma is "pure," and in these circumstances there is little evidence of other primary brain damage. Macroscopically, the hematoma tends to envelop the entire hemisphere and is therefore more extensive than its extradural counterpart. Acute subdural hematomas may also develop from hemorrhage from a brain laceration or torn cortical artery.

The overall mortality of acute subdural hematoma is considerably greater than that of extradural

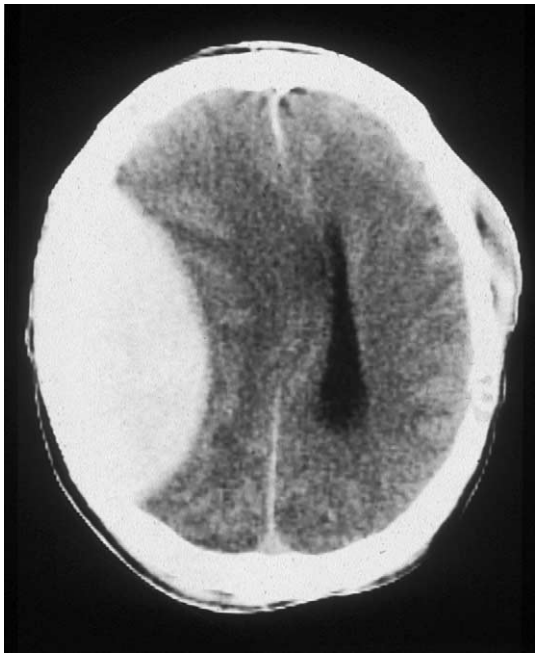

**Figure 7** A computed tomography scan showing a large right-sided extradural hematoma. There is midline shift with subfalcine herniation. Note also the left-sided extracranial soft-tissue swelling.

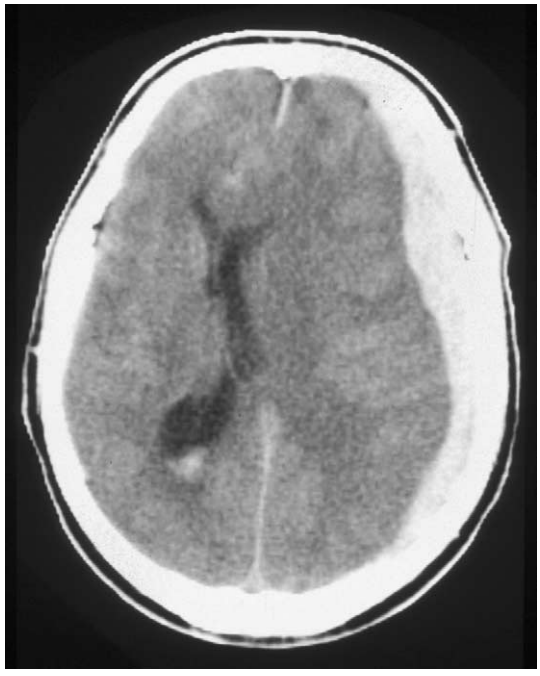

**Figure 8** This man fell off some scaffolding at work. His Glasgow Coma Scale on arrival to hospital was 6. This computed tomography scan shows a left acute subdural hematoma causing midline shift and subfalcine herniation.

**Figure 9** An operative photograph of the man shown in Figure 8. The dura is tense and blue due to the underlying acute subdural hematoma, which can be seen to extrude through a tear in the dura.

hematomas and this is largely a reflection of the greater degree of primary brain damage with which the hematoma is associated. Surgical evacuation at craniotomy is the treatment of choice (Figure 9).

It is important to distinguish a chronic subdural hematoma from its acute subdural counterpart. They tend to form a distinct clinical and pathological entity and are often associated with a minor degree of head trauma, which is often forgotten by the patient. In many instances, loss of consciousness did not take place (Figure 10).

Any condition that results in an increase in the disparity between the volume of the brain and the volume of the cranial cavity can predispose to the development of a chronic subdural hematoma. Conditions such as chronic alcoholism, degenerative brain disease leading to dementia, and senescence may all be associated with the development of a chronic subdural hematoma. Moreover, chronic epileptics who have cerebral atrophy are also predisposed to developing chronic subdural hematomas not only due to the increased disparity between the volume of the brain and the intracranial cavity but also because of the possibility of repeated head trauma that may occur during seizures.

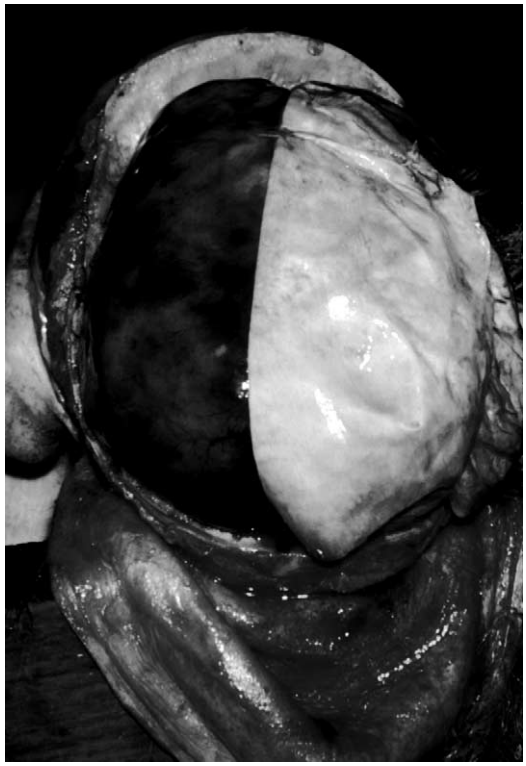

**Figure 10** This elderly woman was found dead at home. She had been complaining of headache and was noted to drag her right leg when last seen alive. Six weeks earlier she attended hospital because she had banged her head on an open cupboard door. This photograph shows the typical features of a chronic subdural hematoma.

### Intracerebral Hematomas

Hemorrhages that form discrete hematomas within the brain substance as opposed to contusions tend to predominate in the frontal and temporal lobes and

need to be considered separately. They tend to be multiple and may also occur in the basal ganglia. It is well recognized that traumatic intracerebral hematoma may develop several days after the initial injury and, in these circumstances, the lesion is referred to as a delayed intracerebral hematoma, or the apoplexy of Bollinger. From a medicolegal standpoint, if a patient develops such a hematoma and subsequently dies, important legal consequences may flow from the initial injury or assault.

## Brain Damage Secondary to Raised Intracranial Pressure

Such damage is a common sequel to nonmissile head injury. Internal herniation of the brain may occur. In subfalcine herniation, an expanding lesion involving one hemisphere causes the cingulate gyrus on the medial aspect of that hemisphere to herniate beneath the free edge of the falx cerebri. In transtentorial herniation, the parahippocampal gyrus on the medial aspect of the temporal lobe is forced through the tentorial hiatus and this causes compression of three important structures, namely the brainstem, the oculomotor nerve, and the posterior cerebral vessels. Compression of the brainstem will result in distortion of the reticular activating system and a depression in the level of consciousness. Compression of the corticospinal tract within the brainstem will result clinically in a contralateral hemiparesis as the pyramidal tract has not decussated at that stage. Compression of the oculomotor nerve will result in ipsilateral pupillary dilatation, due to the now unopposed sympathetic innervation of the pupil. When the posterior cerebral vessels are compressed, a situation that is seen in agonal cases, infarction of the calcarine area of the occipital lobe will take place.

Masses within the posterior cranial fossa will result in herniation of the cerebellar tonsils through the foramen magnum.

The effect of such internal herniation is to cause obliteration of the normal basal cisterns and the development of pressure gradients between one intracranial compartment and another. Vascular damage to the herniated areas of the brain can be seen. The finding of necrosis within the parahippocampal gyri will allow the pathologist to express a view as to whether the intracranial pressure was raised during life. In fatal cases, secondary hemorrhages or areas of infarction may be seen within the brainstem and these are known as Duret hemorrhages.

## Brain Swelling

This is a well-recognized phenomenon following head injury. The significance of brain swelling is that it may contribute to a rise in intracranial pressure as described elsewhere. Brain swelling may be produced by three different mechanisms:

1. Swelling of the white matter adjacent to contusions, which is considered to be due to leakage of fluid from damaged vessels and loss of local arteriolar tone.
2. Diffuse swelling of the hemisphere or of the whole brain. Unilateral hemispheric swelling may occur in association with an overlying acute subdural hematoma, and following its evacuation the brain can sometimes swell very rapidly and dramatically into the space formerly occupied by the hematoma.
3. Swelling of the entire brain may take place in children. The mechanism by which this occurs is uncertain but the immaturity of the blood–brain barrier may be important. It is extremely important to appreciate that, whatever the mechanism producing brain swelling, it can be exacerbated by extracranial events such as hypotension and hypoxia. A vicious circle can then occur, leading to an increase in brain swelling, which then contributes further to a rise in intracranial pressure.

## Hydrocephalus

This may occur after head injury and can either be communicating or noncommunicating. Communicating hydrocephalus is more frequently observed following head injury and results from the presence of blood within the subarachnoid spaces, which leads to a derangement of flow and absorption of cerebrospinal fluid (CSF). Characteristically, this complication may develop some 10–14 days following injury and will be clinically apparent when there is a failure to improve after initial progress has been made or where frank deterioration occurs.

Noncommunicating hydrocephalus may develop acutely secondary to a posterior fossa hematoma that causes compression and obstruction of the cerebral aqueduct of Sylvius or the fourth ventricle itself.

Treatment may involve the insertion of a ventriculoperitoneal shunt or similar CSF-diverting procedure.

## Penetrating Head Injuries

These are classified into two types.

1. Missile injuries from bullets and shrapnel wounds
2. Stab injuries that may result from domestic and industrial accidents, self-inflicted wounds, and criminal assault.

In the USA eight per 100 000 persons die as a result of penetrating head injuries. Half of all homicides in persons under the age of 45 occur as a result of brain injury and 35% of these are attributable to gunshot wounds. In the UK the incidence of gunshot wounds is significantly less.

### Mechanisms of Injury

Projectiles traveling at low velocities and stab wounds cause damage that is confined to the tract that they produce within the brain. High-velocity missile injuries on the other hand cause diffuse damage which extends beyond the tract. The explanation of this phenomenon lies in simple Newtonian mechanics. The kinetic energy (KE) of a projectile is given by $KE = 1/2\,mv^2$, so it will be readily appreciated that the kinetic energy of a projectile is chiefly influenced by its velocity, that depends on a square function. The distance the projectile travels and the energy it has expended in doing so will determine the amount of energy that is transferred to biological tissue. Wounds sustained at point-blank range will invariably be more destructive than those sustained 1.5 km (1 mile) from the muzzle of a firearm. Conventionally, projectiles are classified as being either low or high velocity. The speed of sound in air is $333\,m\,s^{-1}$ and this is the dividing line between low and high velocity projectiles. Pistols and revolvers have a muzzle velocity of $250\,m\,s^{-1}$, whereas most high-power military rifles have a muzzle velocity in the region of $750\,m\,s^{-1}$. Shrapnel injuries are classified as being high-velocity, as projectiles from bombs or grenades are usually traveling faster than the speed of sound.

### Missile Injuries to the Cranium

A projectile will pass through the cranium in a path determined by its velocity, trajectory, and the structures it encounters. A projectile may lodge in the brain parenchyma or may ricochet off the inner table of the skull, causing damage on its secondary or sometimes tertiary paths. If the missile has sufficient kinetic energy, it may pass through the brain and produce an exit wound. Such wounds are invariably more irregular and disruptive to tissue than the entry wound. Damage to the scalp is caused by a laceration from the projectile, which may be compounded by pressure waves and powder burns if the weapon is discharged at close quarters. A common misconception is that projectiles are sterile, having been fired from a rifle or revolver. This is completely fallacious and bacteria will invariably pass into the cranial cavity as a result of the wound. The interaction between the projectile and the skull produces a comminuted fracture and the in-driven bone may act as secondary projectiles, producing further damage in their own right. Projectiles produce damage to the brain parenchyma in three ways:

1. Penetration, in which damage is produced along the tract, extends only to a short distance on either side of the tract. This type of injury is typically associated with low-velocity projectiles.
2. Shock waves. These longitudinal waves travel in front of the missile at speeds in excess of $333\,m\,s^{-1}$. This type of energy produces structural damage beyond the macroscopic tract of the projectile and explains the functional damage remote from the macroscopic pathway of the bullet.
3. Cavitation. This is a particularly disruptive force, the size of the cavity being proportional to the kinetic energy of the missile. Tissue damage is attributable to the brain parenchyma being pushed centrifugally from the surface of the missile. Tissues are damaged by the blast effect and the compression against unyielding dural and bony structures. As the cavity is formed, the pressure within it falls below atmospheric pressure and as a result debris, including bacteria, is drawn into the wound. Following this, the pressure within the cavity can rise and in certain circumstances can result in total destruction of the skull.

The damage produced by the missile is a potent source of brain swelling and edema, which can cause intracranial pressure to rise very rapidly. High-velocity missiles tend to produce a higher and more rapid rise in intracranial pressure than low-velocity projectiles.

## Posttraumatic Epilepsy

This is characteristically divided into early posttraumatic seizures, which occur within 7 days of head injury, and late posttraumatic epilepsy, which occurs at any point thereafter. In addition, some authorities also recognize a third category, entitled immediate posttraumatic epilepsy, which occurs within 1 min to 1 h after head injury.

### Early Posttraumatic Epilepsy

There is a 30% incidence in severe head injury and approximately 1% incidence in mild to moderate injury. In pediatric practice, 2.6% of children under the age of 15 who sustain a head injury which causes a brief loss of consciousness or amnesia will experience an early posttraumatic fit.

Early posttraumatic epilepsy may be associated with the development of adverse events such as a rise in intracranial pressure, alterations in blood

**Table 1** Risk factors for the development of posttraumatic epilepsy

| | |
|---|---|
| 1 | Penetrating head injuries |
| 2 | Depressed skull fractures |
| 3 | Dural tears |
| 4 | Early posttraumatic epilepsy |
| 5 | Presence of intracranial hematoma and/or structural brain damage |
| 6 | Long duration of posttraumatic amnesia |

pressure, and the release of excessive neurotransmitter substances.

### Late Posttraumatic Epilepsy

By definition, this occurs more than 7 days after a head injury. It has been estimated that the incidence of late posttraumatic epilepsy overall is somewhere between 10% and 13% within 2 years of significant head trauma. The incidence of late posttraumatic epilepsy is higher following severe head injury than with moderate or mild trauma. Although the incidence of early posttraumatic epilepsy is higher in children, the development of late seizures is less frequently observed in children.

Penetrating cranial trauma is associated with a higher incidence of posttraumatic epilepsy than closed-head injury. The overall incidence is 15% in patients who are followed up for a period of 15 years. The majority of patients who have not had a seizure within 3 years will not go on to develop fits. Table 1 outlines the major risk factors for developing posttraumatic epilepsy.

Anticonvulsant medication can be used to prevent early posttraumatic seizures in those considered to be at higher risk. Prophylactic use of anticonvulsant medication does not reduce the incidence of late posttraumatic fits however.

## Outcome and Prognosis

Age is a major factor that determines the degree of recovery following head injury. In broad terms, infants recover better than children, and children better than adults. A number of factors have been shown to be associated with a poor prognosis following head trauma. These include:

1. A persistent rise in intracranial pressure of more than 20 mmHg despite hyperventilation
2. Increasing age
3. Impaired or absent pupillary responses or eye movements
4. Hypotension
5. Hypercapnia
6. Hypoxemia or anemia
7. The presence of a mass lesion requiring surgical removal
8. Raised intracranial pressure during the first 24 h after injury.

It has also been shown that the presence or absence of the basal cisterns as visualized on the presenting computed tomography scan is an important prognostic indicator. In general, effacement of the basal cisterns is associated with a poor outlook. The Glasgow Outcome Scale (GOS) is often used to assess the outcome following a head injury and the categories of this scale are shown in Table 2.

One of the questions that is invariably asked by patients, their relatives, and lawyers acting in personal injury claims, is over what period of time can improvement be expected to take place? It is generally expected that natural recovery following a head injury will take place for up to 2 years and any problems that remain thereafter can be regarded as being fixed or permanent. Moreover, the maximum rate of recovery tends to take place within the first 6 months and, after this, recovery occurs at a much slower pace. Furthermore, if a patient has scored 4 on the GOS at 6 months it is most unlikely he/she will score 5 at the end of the ensuing 18 months.

## Postconcussional Syndrome

This is a characteristic condition that follows head injury. It is characterized by a variety of symptoms that can be divided into somatic, cognitive, and psychosocial problems. Paradoxically, it is often the milder head injuries that are associated with the greater severity of symptomatology. Symptoms such as headache, dizziness, blurring of vision, tinnitus, poor concentration, impairment of short-term

**Table 2** Glasgow Outcome Scale

| | |
|---|---|
| 1 | Dead |
| 2 | Persistent vegetative state |
| 3 | Severely disabled |
| 4 | Moderately disabled |
| 5 | Good recovery |

Notes: A persistent vegetative state denotes a patient who remains unresponsive, mute, and in whom there is no psychological meaningful response due to inactivity of the cerebral cortex. Subcortical and brainstem centers still function to some degree and sleep/wake cycles are evident.
Severely disabled survivors: this denotes patients who are reliant on the help of others for one or more of the activities of daily life.
With moderate disability, patients are independent for the activities of daily living but are unable to return to their previous occupation or level of activity.
With a good recovery, patients are able to return to their premorbid level of functioning and occupation.

memory, loss of libido, alteration of the sleep/wake cycle, and intolerance of noise are frequently described. The treatment of postconcussional syndrome is difficult but a number of studies have shown that a full explanation of the problems the patient is likely to have at the time of discharge from hospital can go a long way towards reducing the duration of these symptoms.

## The Role of the Clinical Forensic Physician in the Assessment of Nonfatal Head Injuries

The role of the clinical forensic physician or medical examiner falls into three categories:

1. The acute assessment of the head-injured patient
2. The assessment of an accused who may have sustained a head injury and who has also taken alcohol or illicit drugs
3. The preparation of medical reports as directed by the court or insurance companies.

In the emergency setting, the standard principles of maintaining the airway, ensuring that breathing is occurring and the circulation is maintained are of paramount importance and cannot be overemphasized. Attention should be paid to these factors while the emergency services are arriving. The details of such resuscitative measures are well known and are essentially beyond the scope of this article but the interested reader is referred to the ATLS manual for further information.

In practice, the initial neurological assessment of a head-injured patient is of extreme importance not only because it enables the severity of the injury to be gauged but also because it provides a baseline from which improvement or deterioration can be measured.

Three baseline parameters should be recorded:

1. Glasgow Coma Scale (GCS) (Table 3)
2. Pulse rate and blood pressure
3. Presence or absence of focal neurological deficit.

When called to see a detainee in custody who is unconscious or drowsy, the clinical forensic physician should have a high index of suspicion that the individual may have sustained a head injury. Assurances from the police that the accused has taken a large amount of alcohol or illicit drugs should increase the index of suspicion rather than decrease it. Many prisoners meet an untimely end when persisting drowsiness or unconsciousness is attributed to the ingestion of alcohol or drugs, when in fact an extradural or subdural hematoma is present but only diagnosed correctly at the time of autopsy. If in doubt, assume that the patient has sustained a head injury and arrange for immediate transfer to hospital, where appropriate assessment by a neurosurgeon can take place. Table 4 shows the criteria for immediate hospital transfer irrespective of GCS. Table 5 outlines the criteria for transfer and admission to hospital after a recent head injury.

**Table 3** The Glasgow Coma Scale

| | *Score* |
|---|---|
| *Eye opening* | |
| Spontaneous | 4 |
| To speech | 3 |
| To painful stimulus | 2 |
| None | 1 |
| *Best motor response* | |
| Obeys commands | 6 |
| Localizes painful stimulus | 5 |
| Withdraws (normal flexion) | 4 |
| Flexes abnormally | 3 |
| Extension | 2 |
| No response | 1 |
| *Best verbal response* | |
| Oriented | 5 |
| Confused | 4 |
| Says inappropriate words | 3 |
| Makes incomprehensible sounds | 2 |
| No verbal response | 1 |

**Table 4** Criteria for immediate hospital transfer irrespective of Glasgow Coma Scale score

- Unequal pupils
- Unequal motor examination
- An open-head injury with leaking cerebrospinal fluid or exposed brain tissue
- Neurological deterioration
- Depressed skull fracture

It is also vitally important to assume that all patients who have sustained a head injury have also sustained an injury to the cervical spine until proven otherwise. Practically, this means that the neck should be immobilized prior to any movement or transferred by the application of a collar, placement of sandbags adjacent to the neck, and taping the forehead to the stretcher. Appropriate imaging can then confirm or refute the presence of a cervical spine injury. It is important to appreciate that an injury to the cervical spine has not been excluded unless the C7/T1 junction has been adequately demonstrated.

The clinical forensic physician is often called upon to write medical reports for the court on patients who have been examined. Many books and courses are devoted to this important area of practice, but when reporting on head-injured patients the following factors should be taken into consideration:

**Table 5** Criteria for hospital admission after a recent head injury

- Confusion or other depression of the level of consciousness at the time of examination
- Skull fracture or clinical suspicion if X-rays are not available
- Neurological signs or headache or vomiting
- Difficulty in assessing the patient, e.g., alcohol, drugs, the young, epilepsy
- Other medical conditions, e.g., diabetes mellitus, hemophilia
- The patient's social condition or lack of a responsible adult to continue observation

Posttraumatic amnesia with full recovery is not an indication for admission. If a person remains in police custody, written instructions about possible complications and the action to be taken should be left with the custody officer.

1. Date and time of the injury
2. Mechanism of the injury
3. Whether the injury resulted in loss of consciousness
4. The presence of other injuries
5. Neurological assessment, including the presenting GCS
6. Whether alcohol or illicit drugs were taken
7. Whether the patient had a fit
8. Significant past medical history, including other closed-head injuries and the presence of preexisting epilepsy
9. The treatment the patient received, including the period of hospitalization
10. An estimation of the period of posttraumatic amnesia.

If supplementary medical reports are required to deal with the condition and prognosis of the patient, the continuing symptoms that are attributable to the injury should be recorded in detail.

It may be difficult to give a definitive prognosis, but the period over which natural recovery can take place should be emphasized. Where specialist information is required, from a neurosurgeon, neuropsychologist, or other practitioner, a recommendation that the patient should be seen by such experts before a final opinion can be offered is often helpful to the instructing lawyer or insurance agency.

## Pediatric Head Injury

Although many of the principles governing the pathology and management of head injury in adults can be applied with some modification to childhood head injury, there are important differences. From the epidemiological standpoint, it should be appreciated that children often have milder head injuries than adults. Despite this, 75% of children hospitalized through trauma will have a head injury. In the pediatric age group, the overall mortality for all severities of head injury requiring hospitalization has been reported as being between 10% and 13% but it is important to distinguish that with severe head injuries in children who are presenting with decerebrate posturing, the mortality can be as high as 71%.

The type of injury that children sustain is also distinct in some cases from those that affect adults. Birth injuries occur with skull fractures, cephal hematoma, subdural and extradural hematomas. The possibility of child abuse should not be forgotten in the pediatric age group and the peculiar phenomenon of growing skull fractures or leptomeningeal cysts should also be considered.

In addition, the response to injury can be somewhat different in children. The main differences are that posttraumatic seizures are more likely to occur within the first 24 h in children than in adults. In addition, malignant cerebral edema following head injury is a well-recognized phenomenon in young children who have sustained cranial trauma. The degree of swelling may be disproportionate to the apparent severity of the injury. The likely mechanism of such diffuse cerebral swelling, which is often refractory to all forms of treatment, is expected to be hyperemia.

As far as outcome is concerned, children in general do better following head injury than adults, although there is some evidence that very young children who are of preschool age do not fare as well as schoolchildren.

## Head Injury and Child Abuse

Approximately 10% of children under the age of 10 years who attend hospital following alleged accidents are in fact the victims of nonaccidental injury. It is interesting to note that most children who are assaulted are below the age of 3, an age group in which accidental head injury has a low incidence. The attending clinician should have a high index of suspicion but obviously needs to proceed with the utmost tact. Factors that should alert a clinician to the possibility of child abuse include:

1. Retinal hemorrhages
2. Other injuries of differing ages
3. Bilateral chronic subdural hematomas in children less than 2 years of age
4. Significant neurological damage with minimal signs of external trauma. Infants and children who are shaken sustain acceleration and deceleration injuries to the head. Forces which are proportionally larger in children due to the large size of the head relative to the body tend to occur and this problem is exacerbated by the relative

underdevelopment of the cervical musculature. The characteristic features of the shaken-baby syndrome include retinal hemorrhages, subdural hematomas, traumatic subarachnoid hemorrhage, and very little in the way of signs of external trauma. The attending clinician should look for finger marks on the chest and multiple rib fractures where the child's torso may have been held during the shaking process. In fatal cases death is usually attributable to raised intracranial pressure which is refractory to ventilation and osmotic diuretics.

## See Also

**Children:** Physical Abuse; **Coma, Definitions and Differential Diagnoses:** Pediatric; Adult; **Falls from Height, Physical Findings:** In Adults; **Head Trauma:** Neuropathology; **Injury, Fatal and Nonfatal:** Sharp and Cutting-Edge Wounds

## Further Reading

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# Neuropathology

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## Introduction

Head injuries account for a significant portion of the cases examined by the medical examiner and account for approximately half of all trauma deaths. Head injuries result most often from vehicular accidents, gunshot wounds, falls, assaults, and child abuse. Head-injury rates are greatest in urban areas, where rates are as high as 32/100 000; 50% of head injuries result from traffic accidents, 20–40% from gunshot wounds, 10% from falls, and 5–10% from assaults. Certain procedures for the examination of the central nervous system are discussed in this article because these are essential to document injuries and evaluate the forensic issues. Forensic issues associated with head injuries include mechanisms of injury, timing of injury, onset of symptoms, and survival after injury. Pediatric head injury, particularly abusive injury, is considered in a separate section.

## Examination Procedures

It is essential that the head and brain be examined in a planned and consistently uniform manner so that injuries can be recognized and thoroughly documented. Examination of the brain usually follows a systematic examination of the face, scalp, and neck with documentation of any abnormalities. The pathologist must be present when the cranial cavity is opened to make observations about the presence or absence of epidural or subdural blood.

## External Examination

The examination of the nervous system begins with the external appearance of the face, scalp, and neck. Observation and documentation of all injuries to the face, scalp, and neck should be made in a systematic manner. Injuries in the scalp should be examined after shaving the adjacent hair.

Certain external features may be indicative of internal injury, particularly basilar skull fracture, and should direct attention to those sites:

- Periorbital ecchymosis is the blue or purple discoloration of the periorbital soft tissues caused by fracture of the overlying orbital plate of the anterior cranial fossa (Figure 1).
- Mastoid ecchymosis or the battle sign is blue or purple discoloration over the mastoid area caused by fracture of the petrous portion of the temporal bone.
- Blood running from the ear is also caused by fracture of the petrous portion of the temporal bone.

### Blunt Trauma of the Head

Blunt-force trauma is frequent about the face, neck, and scalp and should be thoroughly documented. Abrasions are areas in which the skin surface has been scraped away and are especially seen over the prominences of the forehead, nose, cheeks, and chin.

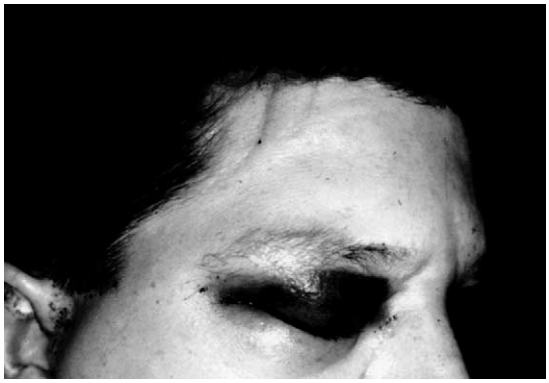

**Figure 1** The right periorbital region in a case of gunshot wound from the right to the left temporal area where there were bilateral periorbital ecchymoses.

Certain patterns of abrasion may indicate a mechanism for the injury. Dicing abrasions are small edged or rectangular abrasions caused by fragments of the tempered glass of the side windows of automobile doors.

Contusions are bruises in which blunt force has damaged underlying small vessels with bleeding into the soft tissue, producing an area of discoloration at the skin surface. Contusions also point to sites of blunt force that may be related to underlying intracranial injury and are important to describe and document. Blunt trauma to the scalp frequently does not produce an externally evident contusion due to the multilayered nature of the scalp and its ability to absorb energy. At autopsy, however, impact sites can be readily noted as hemorrhage within the galea of the reflected scalp. To age these injuries, sections should be taken for microscopic examination.

Lacerations are blunt-force injuries in which the skin surface is torn open, the wound edges are abraded, and the depth of the wound contains tissue bridges. A variety of patterns of lacerations can be recognized, such as those that result from linear objects and hammers. Care should be taken to distinguish sharp wounds from lacerations.

## Internal Examination

### Examination of the Scalp

An incision across the top of the head from mastoid to mastoid area allows the scalp to be reflected forward and backward so that the undersurface of the galea is able to be closely inspected. Any injury evident from the outside can be even more closely viewed from the inside.

### Examination of the Skull

Impact injury to the head frequently produces bleeding into the periosteum of the calvarium. In order to examine the skull properly, the periosteum should be peeled away, along with the temporalis muscles. Fractures of the skull can then be fully visualized, described, and documented. When the calvarium is opened, the presence of epidural or subdural blood should be noted and documented. Description of the location, amount, and condition of the blood is pertinent. Note should be made of the fit of the brain within the cranial cavity: does it fit tightly due to brain swelling or loosely due to atrophy? After removing the brain, the dura must be gently removed from the cranial fossae so those bones can be examined for fracture.

### Skull Fractures

Fracture of the skull denotes that sufficient force has been applied to the head to exceed the ability of the bone to bend without breaking. Fracture may or may not be accompanied by intracranial injury, but in the more common circumstances it does indicate that the head has received an impact. In describing fractures, anatomic location, length, additional fracture lines, and features such as depression should be included.

### Linear Fracture

Linear fractures are the most common fractures at all ages and account for approximately 70% of all fractures. They occur in both the calvarium and the skull base. They are caused by broad-based forces striking the head over a wide area and are common in accidents, such as traffic accidents and falls. A linear fracture is a simple crack and may have additional extensions of stellate form, in which case it should be considered a complex linear fracture (Figure 2).

### Depressed Fracture

Depressed fractures are those in which the bone is displaced inward and may impinge upon the dura and brain, creating complications from cortical contusion and laceration. Depressed fractures are caused by a forceful impact striking over a small area and occur in circumstances such as falls on to a protruding object and impact with instruments such as hammers.

### Comminuted Fracture

A comminuted fracture is one in which the bone fractures into fragments and is caused by a significant force striking over a broad area. Comminuted fractures occur in crushing head injuries and from repeated blows to the head by a blunt object.

### Diastatic Fracture

A diastatic fracture results when a suture is forcefully opened, and it requires significant force. These

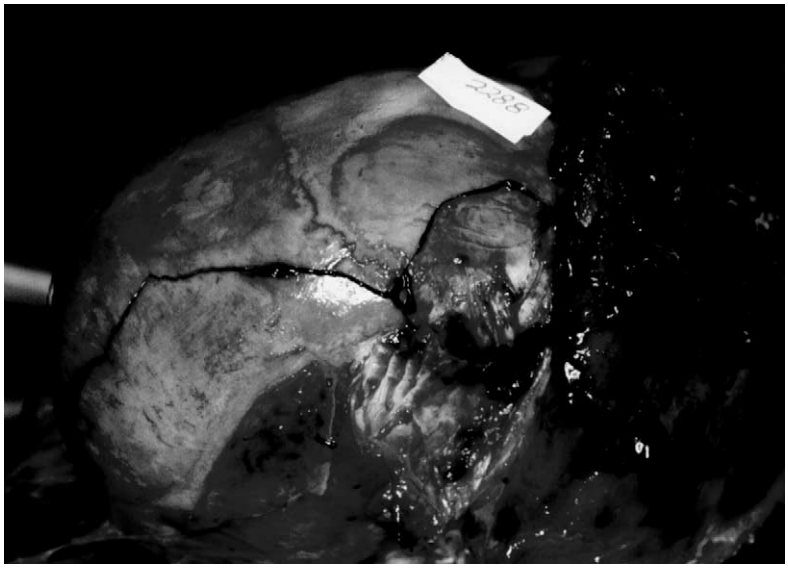

**Figure 2** Linear fracture of right frontoparietal calvarium with overlying subgaleal hemorrhage sustained in a vehicular accident.

fractures usually occur early in life but may be seen at any time and are particularly common in traffic accidents. Marked brain swelling frequently causes sutural separation in young children before the sutures are fused, and these separations are not fractures.

### Compound Fracture

Compound fractures describe fractures underlying laceration of the surface soft tissues and create the possibility of complications from contamination of these wounds.

### Basilar Skull Fractures

Fractures can originate in the base or the calvarium and extend from one to the other. Fractures of the skull base are prone to a variety of complications due to the possibility of lacerating vessels or nerves leaving the brain. Several recognizable patterns of basilar fracture are common in forensic practice. Fractures of the orbital plates associated with periorbital hemorrhages are common and are caused by gunshot wounds to the head, falls on to the back of the head, and vehicular accidents. These fractures are sometimes called contrecoup fractures and do not indicate additional trauma at the site of fracture. The hinge fracture is a fracture extending across the middle cranial fossae through the petrous portions of the temporal bones, so bleeding is to be expected from the ear(s) (Figure 3). Hinge fractures result from very forceful impacts and are most common in vehicular accidents. The ring or circle fracture is a fracture around the foramen magnum through the middle and posterior fossae. Ring fractures are most commonly caused by severe hyperextension of the head on the neck during a traffic accident. A less common mechanism is landing on the feet from a high fall or jump so that the vertebral column is driven upward into the skull base.

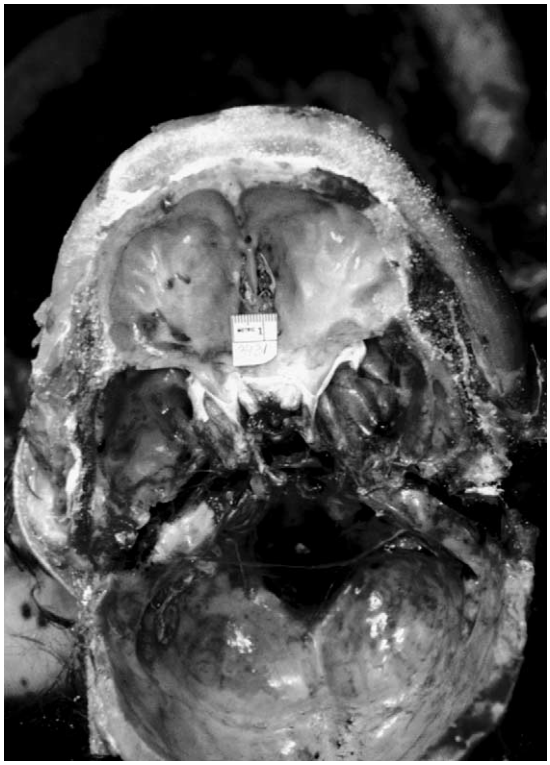

**Figure 3** Hinge fracture through petrous portions of temporal bones separating the front and posterior parts of the skull base caused by a vehicular accident.

## Immediate-Impact Brain Injuries

### Contusions

Brain contusions are bruises of the cortical surface that damage the surface from the outside inward, producing disruption of tissue and vessels. The resulting lesion is a wedge-shaped, devitalized area of punctate and streak hemorrhages extending through a variable depth of the cortex. With greater forces or in patients with bleeding problems, hemorrhage may extend into the adjacent subcortical white matter (Figure 4). Subarachnoid bleeding in the adjacent leptomeninges always accompanies contusion. In surviving patients, contusions undergo changes related to the inflammatory response, with removal of necrotic tissues resulting in a cavitated scar with surrounding gliosis and hemosiderin staining of the leptomeninges. Contusions may be classified into a number of useful categories based on the mechanism of the injury.

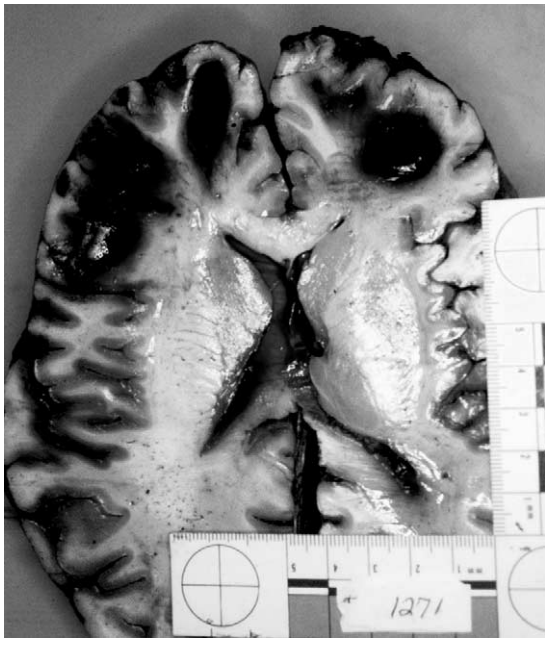


**Figure 4** Massive deep contusions of frontal lobes with extension of contusion hemorrhages into the subcortical white matter.

### Fracture Contusion

Contusions that occur at the site of skull fracture should be called fracture contusions (Figure 5). If the fracture lacerates the brain, the lesion is a fracture laceration.

### Coup Contusion

A coup contusion is caused by a blow to the stationary head with an intact skull at the site of impact. The contusion occurs beneath the point of impact on the convex and lateral surfaces of the brain. They tend to be much less severe than contrecoup contusions.

### Contrecoup Contusion

A moving head impacting a surface in certain falls will result in a common pattern of contusion called the contrecoup contusion. The circumstances of such falls include an adult abruptly losing balance and falling from standing height backwards or to the side, falling downstairs, or jumping from a moving vehicle. Falls from heights greater than 5–6 m typically do not demonstrate this pattern of contusion. Children younger than 4 or 5 years of age do not demonstrate this pattern of contusion. The mechanism of injury is not certain but is most easily understood by Holbourn's rotational shear force

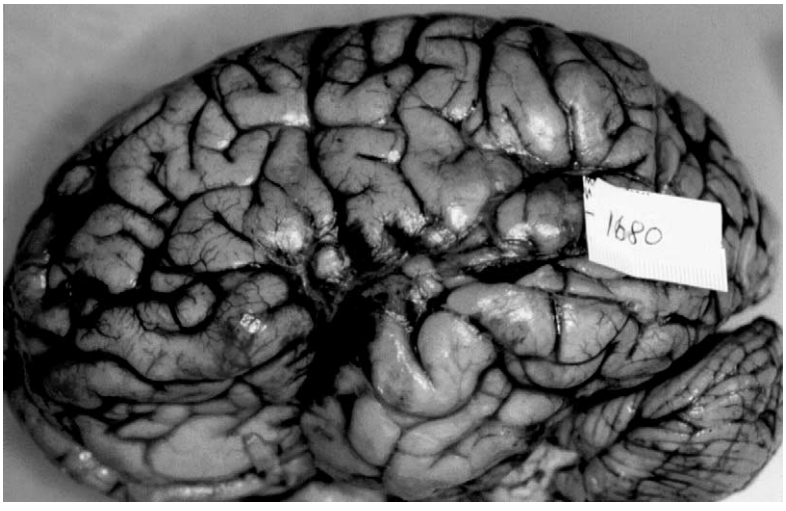


**Figure 5** Fracture contusion of left inferior frontal and superior temporal gyri with overlying subarachnoid hemorrhage.

theory developed from studies of brain movement in relationship to head movement. Movements of the head that cause the brain to impact the rigid shelves of bone of the anterior and middle cranial fossae create contusions over the orbital surfaces of the frontal lobes, the frontal poles, the temporal poles, and the lateral surfaces of the temporal lobes (Figure 6). Remarkably, the greatest extent of contusion is found opposite the point of impact to the head. Contrecoup contusions are frequently massive deep contusions and may cause hemorrhage to burst through the cortex and leptomeninges into the subdural space, resulting in overlying subdural hemorrhage or the "burst lobe."

### Herniation Contusion

Herniation contusions result from massive forces thrusting the brain against the skull base or a dural boundary and are frequently the result of gunshot wounds at a distance from the contusion. These contusions are seen over the hippocampal, parahippocampal, and occipitotemporal gyri.

### Crushing Head Injury

A crushing head injury refers to a stationary head impacted one or more times by massive forces causing comminuted fractures of the skull with fragments of bone contusing and lacerating the brain. The resulting contusions should be described as fracture contusions. Examples of these injuries include homicidal assaults with weapons such as choppers or cleavers or crushing of the head by an automobile wheel or industrial apparatus. Because the head is stationary, consciousness may be maintained during these injuries until brain swelling causes significant increased intracranial pressure.

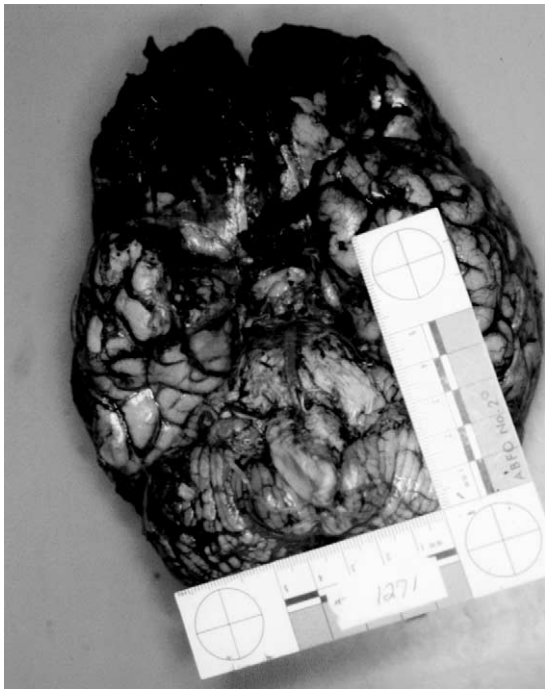

**Figure 6** Contrecoup contusions of orbital surfaces greater on the right than on the left with bursting of blood through arachnoid membrane into subdural space.

### Falls from a Great Height

Falls from a height greater than 5–6 m may be considered a "great height" because it is at this point that falling head injuries lose the contrecoup pattern of contusion and the injury consists of multiple skull fractures with fracture contusions and lacerations of the brain. These injuries look much like the crushed head injury.

### Diffuse Axonal Injury

Diffuse axonal injury (DAI) refers to diffuse damage to the axonal processes and may result from either traumatic injury or hypoxic–ischemic injury. Traumatic DAI (TDAI) is an injury caused by forces that move the head and result in inertial damage to axonal processes and the vascular adnexae. Such forces may be either impact to the head or impulse shaking mechanisms in which the head is abruptly accelerated and decelerated. These mechanisms cause the brain to move abruptly within the cranial cavity somewhat differently than the more rigid skull moves. Differential movement of the brain causes disruption of axonal processes from the periphery of the brain inward, particularly at sites where the adjoining tissues are nonuniform. As axonal processes are torn, nearby small vessels also tear and these vascular injuries produce the grossly recognizable lesions seen in TDAI. Streak hemorrhages occur diffusely within the brain, most notably in the subcortical white matter, corpus callosum, deep gray structures of the basal ganglia, and the lateral extensions of the rostral brainstem. As axonal processes are disrupted at deeper levels, clinical symptoms reflect this damage to neuronal connections. When significant axonal damage occurs at the level of the deep gray structures and upper brainstem, the clinical appearance is immediate unconsciousness (traumatic unconsciousness). The inertial movement of the brain that tears the axonal processes also tears the vascular adnexae of the brain. These adnexae are the bridging veins that, when torn, result in subdural bleeding. In most cases of TDAI, there are at least small amounts of blood in the subdural space. TDAI most commonly occurs in victims of traffic accidents where severe acceleration–deceleration forces are encountered. Less commonly, TDAI is seen in other types of incidents, such as falls and assaults.

### Brainstem Avulsion

Brainstem avulsions are tears at the pontomedullary or cervicomedullary junction, usually caused by extreme hyperextension of the head in a vehicular accident. The disruption may be partial or complete, and the survival period depends on the completeness of the tear. These injuries are associated with overlying subarachnoid hemorrhage. These injuries occur in conjunction with TDAI.

## Penetrating Injuries

### Gunshot Wounds of the Head

Gunshot wounds of the head are very common injuries. Penetrating wounds are those in which the missile enters the head and does not exit. Perforating wounds are ones in which the missile enters the head, passes through, and exits the head. The behavior of a missile passing through the head depends on a number of factors, including the type of weapon, the caliber of the missile, the bullet construction, and the range of fire. For shotgun wounds of the head, the behavior depends primarily on the range of fire; at close and contact ranges, these wounds will usually be perforating and devastating. Gunshot wounds to the head caused by high-velocity missiles at close and contact range also tend to be perforating. Of gunshot wounds to the head caused by handguns, at close and contact range, approximately 30% will perforate the head and about 30% will perforate the skull. Many low-velocity missiles ricochet within the head to create secondary pathways, whereas others come to rest beneath the skull or the scalp.

Bullets passing through bone of the skull typically do so with a widening cone of fracture of the bone. An entrance wound will thus demonstrate a wider bevel on the inner table and an exit wound will demonstrate a wider bevel on the outer table. An exception is the tangential wound, which demonstrates a combination of the two patterns, reflecting the fact that a fragment entered and a fragment exited – the keyhole defect.

Low-velocity bullet wounds of the brain consist of fracture contusions surrounding the entrance and exit defects of the brain, and these wounds cannot be distinguished from each other (Figure 7). Bullet wounds tend to show subarachnoid hemorrhage over most of the brain. The pathway through the brain consists of macerated brain mixed with blood and possibly bone fragments. The bone fragments will be closer to the entrance wound. High-velocity wounds are very damaging at any distance. These wounds often result in the head tearing open with extensive fractures, and at close range they may cause the brain to be extruded. Shotgun wounds at close or contact range are also very damaging and result in massive injuries, often with extrusion of the brain.

### Stab Wounds of the Head

Stab wounds of the head are much less common than gunshot wounds and are more likely to penetrate in the thinner portions of bone, such as the squamous portion of temporal bone or the orbits. The effects of a stab wound to the head are related to the direct damage from severing tissues as well as resulting hemorrhage and later infection (Figure 8).

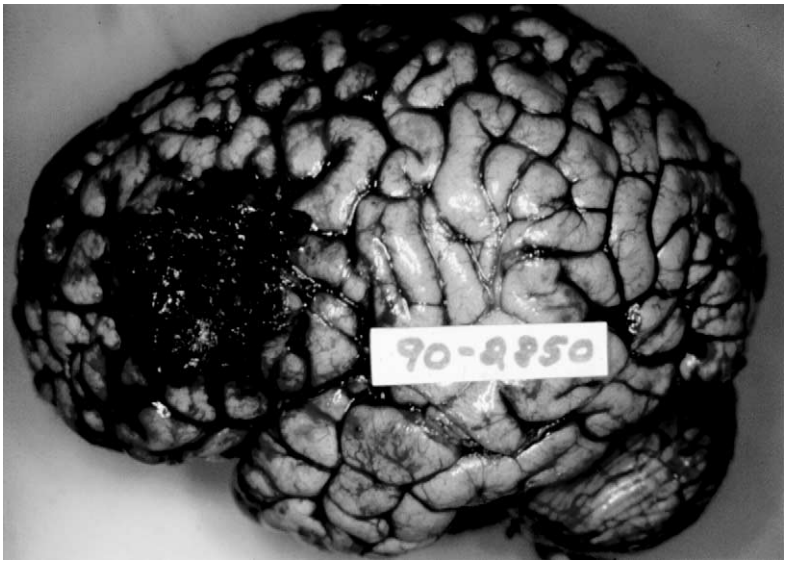


**Figure 7** Low-velocity gunshot wound entering the left middle frontal gyrus with surrounding fracture contusions and subarachnoid hemorrhage.

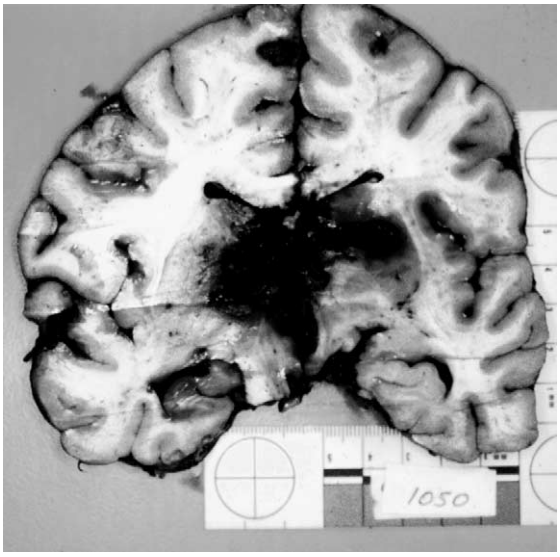


**Figure 8** Stab wound that passed through the right eye into the right basal ganglia producing hemorrhage of both basal ganglia.

## Intracranial Hemorrhage

### Epidural Hemorrhage

Epidural hemorrhage is bleeding that occurs beneath the skull and over the dura. It is always related to impact to the head, and in approximately 85% of cases there is fracture of the skull. In younger skulls, impact without fracture may produce cranial distortion and strip the dura off the bone with epidural bleeding. Bleeding is usually from a branch of the middle meningeal artery but may be of venous origin from a sinus or diploic vein. Fewer epidural hemorrhages are seen in the very young and the elderly because the dura is so firmly adherent to the bone and difficult to strip off. Epidural hemorrhage tends to lie over the cerebral convexities but may also occur at the base. Because epidural hemorrhages lie above the dura, they tend to take on a lens-like configuration and produce a sharply outlined impression on the underlying brain, with the gyri being markedly flattened out. Brain beneath an epidural hemorrhage frequently demonstrates fracture contusions caused by the accompanying fracture.

Bleeding in an epidural hemorrhage varies from small insignificant amounts to large amounts that may produce a space-occupying mass. The amount of epidural hemorrhage needed to be significant as a mass lesion depends on the location of the bleeding. In an adult, epidural blood over the cerebral convexity greater than 100 ml is significant, whereas much less blood in the posterior fossa causes a mass effect.

Epidural hemorrhage as an isolated injury should have little morbidity or mortality because it can be easily removed surgically if it causes increased intracranial pressure and, if small, may resolve on its own. Epidural hemorrhages associated with other brain injuries have a much worse prognosis.

### Subdural Hemorrhage

Subdural hemorrhage is bleeding beneath the dura and over the surface of the arachnoid membrane. Nontraumatic causes of subdural bleeding are ruptured berry aneurysms and hypertensive hemorrhages that rupture through the subarachnoid membrane into the subdural space. Traumatic subdural hemorrhage is very common and may result from tearing of a venous sinus or an arachnoid artery but most often results from tearing of bridging veins. Subdural hemorrhage does not require an impact to the head; it requires only that the head be accelerated or decelerated so that the brain moves abruptly within the intracranial compartment and puts strain on the bridging veins to the point of tearing.

Subdural bleeding lies over the cerebral convexities as well as the base and tends to be unilateral in adults (Figure 9). The amount of blood required to produce a mass effect depends on the age of the individual and state of development of the brain. In infants and young children, very small amounts of subdural blood may be significant as a marker of diffuse brain injury. Young children may develop increased intracranial pressure


**Figure 9** Thin film of acute subdural blood over both cerebral convexities and lying within the skull cap.

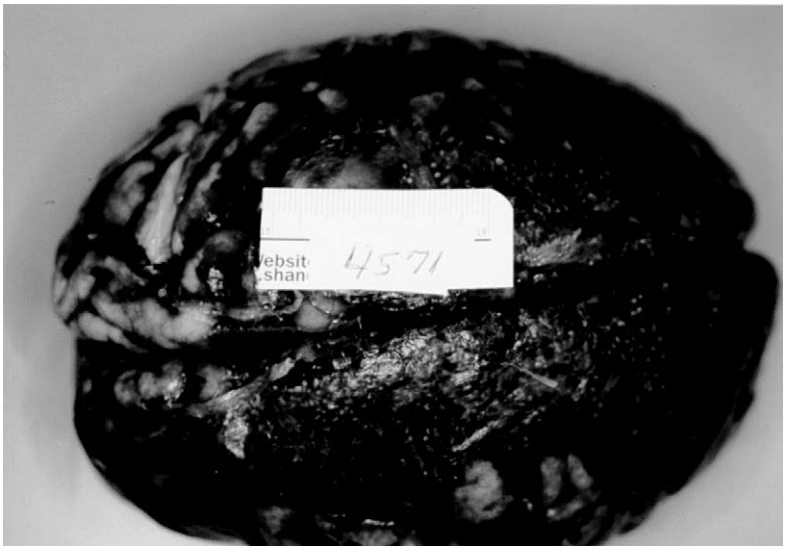

**Figure 10** Thick layer of fresh subarachnoid hemorrhage over both cerebral convexities caused by chopper blows to the head.

when the amount of blood reaches 30–50 ml. In adults, 100–150 ml of subdural blood produces mass effect. Individuals with brain atrophy from age or alcoholism may require greater amounts of blood.

The cortical surface beneath a layer of subdural blood frequently demonstrates foci of subarachnoid hemorrhage because the torn bridging veins are invested with a layer of arachnoid and, when torn, small amounts of subarachnoid bleeding also occur. The brain surface may be flattened in a crescent shape beneath a collection of subdural blood but will not be as uniformly flattened as seen with epidural hemorrhage.

Blood that remains in the subdural space over time may transform into a chronic membrane in certain circumstances, but not in all cases. Most subdural blood in a previously normal brain will resolve rather rapidly or be rapidly organized. The classic chronic subdural membrane develops in certain patients who have a low intracranial pressure as a result of brain atrophy (elderly or alcoholic patients) or hydrocephalic patients who have been surgically shunted. In these patients, small amounts of bleeding into the subdural space induce an ingrowth of granulation tissue from the dural side. Minor trauma may result in microbleeding in the fragile microcapillaries of the granulation tissue. A repeated cycle of induced growth and bleeding may result in a thick classic membrane in this group of individuals.

### Subarachnoid Hemorrhage

Subarachnoid hemorrhage is bleeding into the subarachnoid space usually occupied by the cerebrospinal fluid. Natural causes of subarachnoid hemorrhage are ruptured berry aneurysms and vascular malformations. Trauma frequently causes subarachnoid bleeding,

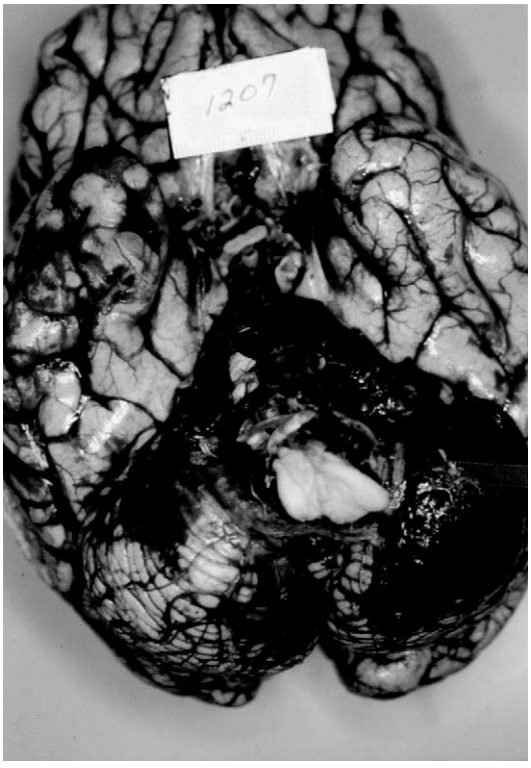

**Figure 11** Thick layer of fresh traumatic basilar subarachnoid hemorrhage over the ventral surface of the brainstem and cerebellum.

which is seen in association with contusions, lacerations, and gunshot wounds (Figure 10).

Traumatic basilar subarachnoid hemorrhage is a rare injury, most often caused by damage to a vertebral artery at C1 and related to lateral movement of the atlas if the head or neck is struck firmly. Tearing of the vertebral artery or dissection of the vascular

lamina can result in blood tracking upward to lie in a thick layer over the ventral surface of the brainstem and cerebellum (Figure 11).

## See Also

**Deaths:** Trauma, Head and Spine

## Further Reading

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