

CHAPTER 12

HEAD TRAUMA

Head trauma from motor vehicle accidents, industrial mishaps, falls, and physical assault has become a significant part of medical practice. Even though specialized care by a neurosurgeon is required in many cases, the general physician must understand when neurosurgical consultation is required and what should be done until the surgeon arrives. In addition, all physicians must be aware of the sequelae of head trauma and their assessment and management.

In most cases of head trauma, the mechanism of injury is a blunt force to the head that is of sufficient force to cause a concussion. The term concussion can be most simply defined as an immediate impairment of neural function (ionic, metabolic, neurotransmitter, vascular) secondary to a mechanical impact to the head. The impaired consciousness is the most consistent symptom; however, less commonly there can be impaired vegetative function (respiratory slowing, bradycardia, hypotension) and motor impairment including hypotonia, areflexia, and Babinski signs. There is usually full recovery from a simple uncomplicated concussion, but the patient can have residual amnesia for the events of the injury. The neurodiagnostic studies show no structural macroscopic pathologic lesions in patients with cerebral concussion, but there may be microscopic diffuse axonal shearing injury.

The severity of the concussion can be defined by the length of posttraumatic amnesia experienced by the patient or by the length of coma (Table 12-1). The length of posttraumatic amnesia, though difficult to assess at times, seems to give the most valid prediction of eventual outcome. The length of unconsciousness is usually easier to determine and therefore often used. Posttraumatic amnesia is measured from the last memory before the trauma to the return of full continuous memory after the trauma. The amnesic period can be divided into two sections: one, the period before the trauma (retrograde amnesia), and two, the period after the trauma (anterograde amnesia). In the more severe cases of head trauma in which the coma period is greater than 24 hours, the assessment of the changing level of consciousness using the Glasgow coma scale (see Chapter 9, Stupor and Coma) is important in predicting eventual outcome. A prognosis based on examination in the first 48 hours is not particularly valid but improves steadily thereafter. Certain patients suffer a blow to the head without loss of consciousness or awareness and no period of amnesia. There is unlikely to be any traumatic brain injury and this is defined as trivial head injury.

Table 12-1 Severity of Concussion Defined by Length of Posttraumatic Amnesia	
Severity of Concussion	Length of Amnesia
Slight	0-5 minutes
Mild	5-60 minutes
Moderate	1-24 hours
Severe	1-7 days
Very severe	>7 days

PATHOPHYSIOLOGY

A blow to the head causes its injury basically by putting excessive strain on the brain and supporting tissues. The scalp can be lacerated, the skull can be fractured, or a hematoma can form from torn blood vessels. These can occur in the scalp, under the galeal aponeurosis (subgaleal), between the dura and skull (epidural), between the dura and arachnoid (subdural), or in the brain (intracerebral). Contusions (bruises) can occur on the cortical surfaces. Such focal damage can occur at the point of impact or frequently on the opposite pole of the brain along the force line (contrecoup). The contrecoup injury is most frequently seen in deceleration injuries where the moving head strikes a stationary object (e.g., when a patient falls and the head hits the floor). Subarachnoid blood is usually present, and widespread damage can also occur throughout the white matter. These white matter lesions are due to the shearing forces initiated by the brain's rapid acceleration (when the head is struck) or deceleration (when the moving head hits a stationary surface). The major damage to the brain seems to be caused by the rather free rotational and lateral motion of the brain within the skull. This motion allows the brain to move against rough surfaces of the base of the skull and also to twist and distort itself. It is these distortional forces and movement that produces the strain and stretching of axons and vessels.

CLINICAL HISTORY AND EXAMINATION

In head trauma cases, it is important to know the exact mechanism of the injury. A blow to the head with a hammer will produce a much different pattern and extent of injury (focal damage to underlying brain) than would a fall from a motorcycle at 45 mph. The latter accident will likely cause injuries to the head, neck, long bones, chest, and so on. Remember that the head bone is connected to the neck bone; therefore following head injury there is frequently injury to the cervical spine. Cervical injuries (whiplash) can also cause brain trauma as a result of the rapid acceleration and rotation of the head.

The physician should inquire whether the patient had been drinking or taking illicit or sedative drugs. Such substances can depress the level of consciousness and make the effects of the trauma appear more significant than they actually are. The examiner should ascertain if the patient experienced a period of unconsciousness and, if so for how long. If the patient is still in a coma on arrival at the emergency department, witnesses or ambulance attendants can be asked whether conscious was regained after the trauma. Some patients are initially rendered unconscious by a head injury, then slowly awaken to the point of being able to carry on a conversation (lucid interval) only to slip into coma again. A history of a lucid interval such as

this is crucial when present for it frequently indicates that the patient is rapidly developing a life-threatening epidural hematoma. The lucid interval is not pathognomonic for epidural hematomas and can sometimes be seen in cerebral contusion, subdural hematoma, and intracerebral hematoma; however, its presence adds further urgency to evaluation and treatment and is most common in epidural hematoma.

In the alert patient, it is necessary to establish the length of posttraumatic amnesia and to question the patient as to the presence of any neurologic symptoms (e.g., headache, dizziness, confused feeling, and diplopia). Is the patient oriented to place and time? Sometimes the only sign of significant cerebral dysfunction is disorientation.

In the seriously injured patient who is still comatose, the adequacy of the airway and circulation must be assessed immediately. The clinician should auscultate the chest and then establish adequate ventilation. An important word of caution: almost all head trauma involves extension, flexion, or rotational injury to the cervical spine with possible fracture. *Be very careful not to move the neck any more than is absolutely necessary!* An unstable cervical spine can easily damage the spinal cord if moved. After cervical spine is evaluated and patient judged stable, protect the airway in patients with depressed level of consciousness and this may involve endotracheal intubation.

The adequacy of circulation must next be evaluated and shock treated if diagnosed. Administer fluids as isotonic (normal) saline or lactated Ringer's solution to avoid worsening cerebral edema with hypotonic fluids. Avoid and treat hyperthermia with acetaminophen or cooling blanket. Another word of caution: shock cannot be due to intracranial bleeding except in young infants. It can be secondary to blood loss from a large scalp laceration, but it is usually due to injuries elsewhere in the body (e.g., spleen, liver, kidney, lungs, thigh muscles). When taking the pulse the examiner should make note of bradycardia. In some patients with a rapidly expanding intracranial lesion the pulse will drop, respiration will slow, and blood pressure often rises. This is called the Cushing reflex due to tonsillar herniation.

The head should be examined for obvious signs of trauma. All deep scalp lacerations should be clean and palpated. A fracture line or depression under a laceration indicates that there is a possible communication from the scalp to the intracranial compartment. In such cases the risk of intracranial infection is great. The examiner should look for evidence of basilar skull fracture: blood behind the eardrum, cerebrospinal fluid leaking from either the nose (rhinorrhea) or the ear (otorrhea), or slowly developing ecchymosis around the eyes (raccoon eyes) or over the mastoid process (Battle's sign).

After the patient is stable and has adequate ventilation and circulation, a rapid screening neurologic examination should be performed. Special attention should be directed toward the level of consciousness, alterations in mental status, papillary symmetry and reactivity, and symmetry of reflexes and motor control.

In patients with head and facial traumatic injury and suspected traumatic brain injury, CT should be the initial study (but initially make certain there is no cervical spine injury as neck is extended in CT scan) and should include bone windows (to delineate skull fracture). MRI is indicated

when CT does not adequately explain the clinical findings of the patient. MRI is superior to delineate corpus callosal pathology in white matter diffuse axonal shearing injury. If CT and MRI show no significant injury in patients with suspected traumatic brain injury consider these possibilities 1. non-traumatic primary brain pathology e.g. stroke which caused the trauma, 2. drug or alcohol overdose 3. hypoxic ischemic injury 4. seizures with postictal state or nonconvulsive status.

CLINICAL SYNDROMES

There are many clinical management problems presented by patients with head trauma. These problems have two phases; the management of the acute trauma and the appreciation and treatment of the sequelae of the injury.

CONCUSSION

The loss of consciousness is the most consistent defining feature of concussion; however, less commonly there can be impaired vegetative function (respiratory slowing, bradycardia, hypotension), mental confusion, and motor disturbances including weakness, hypotonia, areflexia, and Babinski sign. A knockout in boxing is an example of concussion. In concussion, the period of impaired consciousness rarely lasts longer than 30 minutes, and there is usually full recovery; however, the patient usually remains amnesic for the episode. It is important to determine the duration of loss of consciousness as well as how long the patient is dazed and confused. Because there is full recovery the pathophysiologic substrate of concussion is not clearly known; however, it is probably due to the acute distortion of the brain stem ascending activating system (reticular formation). If trauma-related abnormality is found on CT or MRI, the disorder is no longer considered to be a simple concussion. It is believed that patients with concussion have suffered a reversible pathophysiologic disturbance. There is impaired brain function (caused by reversible electrical conduction disturbance) without evidence of pathologic injury to brain tissue. It is possible that a mild form of white matter shearing injury (micro and macroscopic axonal shearing injury) has occurred, but it is insufficient to produce abnormal findings of the neurologic examination or imaging studies. The patient who has had a simple concussion but is awake on arrival at the emergency department should have full physical and neurologic examinations and probably a plain skull x-ray to check for a linear fracture.

Because neck injury is often associated with head injury, cervical spine roentgenography can be considered. If the skull and cervical spine roentgenograms are negative and neurologic examination including mental status is clear, the patient can usually be discharged to the care of a responsible person, although hospital admission for 24 hours of neurological observation could be justified as delayed neurological deterioration may occur. If released, the patient should be carefully observed for 24 to 48 hours at home. Any signs of decreased arousability, confused mental status, pupillary changes, or unilateral weakness should prompt immediate return to the hospital for further diagnostic tests, especially brain CT scan.

There is often a question as to which patient is to be admitted. The usual guidelines in considering admission are listed in Box 12-1.

The CT/MRI scan has revolutionized the evaluation of head trauma patients. CT scans can readily identify the presence of most intracranial injuries including hematomas, contusions, tissue ischemia, mass effect, edema, and hydrocephalus. CT scanning will not demonstrate white matter shearing injuries, which are seen more clearly with MRI.

BOX 12-1

1. Patients still unconscious on arrival in the emergency department.
2. Patients with positive (focal) neurologic signs.
3. Lethargic or confusional patients
4. Patients with evidence of basilar skull fracture on examination
5. Patients with skull fracture on roentgenography
6. Patients with severe headaches
7. Patients with nausea and vomiting
8. Patients under the influence of alcohol or drugs (in such patients, trauma-related alterations in the level of alertness and mentation cannot be accurately assessed)
9. Persons living alone or at a great distance from the hospital

Management issues and severity assessment.

Risk stratification can be done on the basis of following features:

1. Low risk – no concussion, normal exam, no drug or alcohol intoxication, no systemic injury, none or minimal scalp laceration or hematoma, Glasgow score of 14 to 15; patient may complain of headache, visual blurring or dizziness
2. Moderate risk – concussion, amnesia, vomiting, seizure, signs of skull fracture, alcohol or drug intoxication, unreliable or unknown history of trauma, Glasgow score of 9 to 14.
3. Severe – Glasgow score of 8 or less, decreased mental state, neurological deficit, skull fracture.

Patients in low risk group probably do not require neuro-imaging studies and can be discharged but need follow-up by responsible adult for 12 to 24 hours and need to return to medical facility if neurological symptoms develop. In patients who have suffered concussion and CT shows no abnormality and there is no evidence of drug or alcohol intoxication, they may be sent home but require follow-up for 24 hours by responsible adult. If not available, hospital observation for 24 hours is prudent management. All others require hospital admit and observation.

PATHOLOGICAL TRAUMATIC INJURIES

CONTUSION

A contusion represents damaged neuronal tissue on the cortical surface, basically a bruise with evidence of tissue necrosis, edema, and extravasated blood. These are most common at superficial cortical regions at the impact point or where the moving brain rubs or comes to a stop along the inner surface of the skull. They are frequently seen directly underneath depressed skull fractures. The most common locations for contusion are on the inferior surface of the frontal and temporal lobes. With blows to the occipital region there can be an occipital contusion (coup injury) and frontal contusion (contrecoup lesion) as a result of the vacuum phenomenon as the brain accelerates from front to back within the skull. If there is a contusion with actual discontinuity of brain tissue, this is called a brain laceration. Contusions can be multiple and small and can resolve spontaneously; however, larger confluent contusions can form large focal lesions with intracerebral hematomas. These lesions can cause mass effect and result in focal neurologic deficits and impaired consciousness; at times surgical evacuation can be indicated. The diagnosis of cerebral contusions can be established by CT.

DIFFUSE WHITE MATTER SHEARING INJURY

The brain usually shows no surface cortical contusions; however, hemorrhagic contusions do occur in the white matter, most commonly in the corpus callosum and cerebellar peduncles. There are multiple micro and macroscopic axonal injuries which usually occur in midline structures – corpus callosum, hypothalamus, midbrain, pons. There is often intraventricular blood and ventricular dilatation. As the process evolves, the hemorrhage resolves. There can be atrophy of white matter with microscopic evidence of wallerian axonal degeneration. Clinically, these patients with CT/MRI evidence of white matter lesions are usually comatose with bilateral motor impairment and Babinski signs. CT scanning can show hemorrhages in the white matter and ventricles; however, MRI is more effective in demonstrating the nonhemorrhagic white matter shearing injuries. This injury may be associated with persistent disability and these patients may remain in chronic vegetative state.

POSTTRAUMATIC INCREASED INTRACRANIAL PRESSURE

In patients who have suffered head injury elevated intracranial pressure can result from structural brain injury (e.g., contusions, hematomas, edema). With increased intracranial pressure there can be reduced brain perfusion. This results in cerebral ischemia and further elevation of intracranial pressure. When intracranial pressure is due to a focal lesion there can be a significant shift of intracranial structures and herniation syndromes. With supratentorial mass lesions there is transtentorial descending herniation with distortion of the midbrain. This causes ipsilateral dilated pupil (due to oculomotor nerve compression) and contralateral hemiparesis (due to corticospinal tract compression.) With infratentorial mass lesions, Cushing reflex (arterial hypertension, bradycardia, respiratory slowing and irregularity) develops due to tonsillar compression of medulla (tonsillar herniation). In some patients severe diffuse brain swelling and edema can occur after trauma in the absence of contusions and hematomas. The mechanism of this diffuse edema is not understood, but it can be a very serious, often fatal,

complication of closed head injury. Treatment of increased intracranial pressure includes the following:

- ❖ Hyperventilation to reduce PCO_2 and induce cerebral vasoconstriction to reduce blood volume
- ❖ Mannitol (1 gm/kg intravenously in adults or 0.25 gm/kg as an initial dose in children)
- ❖ Dexamethasone (10 mg intravenously initially and 4 mg every 4 hours)
- ❖ Surgical evacuation of the hematoma

If the patient is combative, agitated and confused, intubation and sedation may be required. Utilize propofol (infuse 0.1 to 0.5 mg/kg per minute). This permits utilization of endotracheal intubation and mechanical ventilation. Propofol may also be effective in reducing elevated intracranial pressure. Repeated doses of mannitol should be avoided as it may have opposite effect by accumulating fluid in portion of brain with intact blood brain barrier. Intubation with controlled ventilation should control intracranial hypertension. Positive end-expiratory pressure improves tissue oxygenation, however, this may increase intracranial pressure. Adequate brain perfusion pressure and cerebral blood flow must be maintained, but the blood brain barrier and cerebral autoregulation are frequently impaired in patients with traumatic brain injury. If there is reduced perfusion pressure, this results in reduced cerebral blood flow and resultant ischemia; this can be treated by increasing systolic blood pressure and reducing CSF pressure (achieved by external drainage or mannitol). If there is increased cerebral perfusion pressure, there is vasogenic edema and this is treated with hyperventilation, mannitol or corticosteroids. Also, to prevent intracranial hypertension, avoid neck rotation (causes jugular vein compression), and avoid stimulation of palate and pharynx which induces gag and laugh reflex.

INTRACRANIAL HEMATOMAS

Small blood vessels in the brain and meninges are often torn when patients sustain a blow to the head. When this occurs, the resultant leakage of blood will form a hematoma that will expand at a rate commensurate with the degree of the vessel tear. The contents of the skull (brain, CSF, and vessels) have only limited ability to accommodate to the expanding clot, and very soon signs of increased intracranial pressure and herniation develop. In the unconscious patient deterioration because of an expanding clot can be manifest solely by the appearance of decerebrate posturing or sluggish pupils. In the alert patient the first sign of a developing clot is frequently a subtle change in the level of consciousness or mental state. Any deterioration in the patient's neurologic status subsequent to head trauma should alert the physician to the possibility of a developing intracranial clot, and an emergency CT scan should be obtained. Most patients who become alert after their initial concussion, then subsequently deteriorate and die, have an intracranial hematoma. Failure to recognize an intracranial hematoma is one of the major avoidable factors when patients die after head trauma.

EPIDURAL HEMATOMA

The epidural hematoma accounts for 15% to 30% of all intracranial clots. These clots develop between the dura and inner table of the skull. Seen more commonly in young patients (10 to 30 years old) the lesion is associated with a linear skull fracture which crosses the middle meningeal artery in more than 90% of the cases. The fracture is usually in the temporal or

temporoparietal area. In more than half of the cases the fracture crosses the middle meningeal artery groove, and that artery or its companion vein is the major source of the bleeding. In approximately 30% of the cases, however, bleeding can be traced to one of the venous sinuses. Bilateral lesions are distinctly rare, and most epidurals are in the temporoparietal area (70%).

The hematoma – if of arterial origin – grows very rapidly and can cause deterioration and death within minutes; therefore early recognition is critical. Clinically, 30% to 50% of the patients have a history of a lucid interval after trauma. On the other hand, 15% give no history of an initial loss of consciousness with the trauma. Epidural hematoma occurs most commonly in young patients; it is rarely seen in older patients because dura becomes adherent to skull in older patients. The clinical course of epidural hematoma evolves from immediate loss of consciousness from which patient recovers (concussion) to lucid interval to delayed deterioration (altered consciousness, contralateral hemiplegia, ipsilateral papillary dilatation).

Early features can be headaches and vomiting, but these findings are not as significant as the appearance of abnormal neurologic signs (e.g., decreased level of consciousness or signs of uncal herniation). Seizures are rare unless there is accompanying cerebral contusion or hemorrhage. In some patients the pulse may drop, respirations may slow, and blood pressure may rise because of increased intracranial pressure (Cushing reflex). If there are signs and symptoms of raised intracranial pressure, mannitol is administered initially. Dexamethasone is also useful. Corticosteroids can take 6 to 24 hours to be effective, and mannitol or hyperventilation (to cause vasoconstriction) is initially needed in emergency situations.

Prompt surgical evacuation of the clot can be very rewarding if done early enough because 60% of the patients will not have other associated brain damage and recovery can be complete. The presence of hemiparesis and papillary dilatation indicate impending brain stem compression due to transtentorial herniation. If treatment is delayed, however, mortality rates are high: 17% when one pupil is dilated and fixed at the time of surgery, almost 50% when both pupils are fixed and 80% when decerebrate posturing is present.

SUBDURAL HEMATOMA

The subdural hematoma forms between dura and arachnoid. The source of bleeding in chronic subdural hematomas is usually the small veins that bridge the arachnoid space and enter dural sinuses. Acute subdural hematomas can have mixed arterial and venous bleeding. Subdural hematomas occur commonly in persons 40 years of age and older, particularly in the elderly, in whom mild shrinking of the brain causes stretching of the bridging veins. Documented trauma is implicated in about 50% of cases, but its incidence decreases with patient age. Bilateral lesions are found in 20% of adult patients but is 50% of pediatric patients. Alcoholics are particularly vulnerable because they are often victims of head trauma and also have blood clotting abnormalities (Figures 12-1 through 12-3). Subdural hematomas in children are often the result of unreported trauma (battering).

Clinical symptomatology is similar to that of epidural hemorrhage in many respects; however, seizures (associated with underlying cerebral contusion or hemorrhage) are more common in subdural hemorrhage, and signs of uncal herniation are less frequent. Skull fracture occurs in less than 50% of cases and is more frequent in the young. Unlike patients with epidural

hematoma, in whom the fracture is almost always on the side of the clot, 50% of patients with subdural hematomas have the fracture on the opposite side. A subdural hematoma can be an acute, subacute, or chronic lesion; each has its own clinical and prognostic significance.

Acute. An acute lesion develops in less than 3 days, is usually related to severe trauma, and is therefore often associated with intracerebral and epidural clots. This lesion is more common in young patients; frequently the patient does not regain consciousness from the trauma, and a lucid interval is present in less than 30% of the patients. If surgery is performed in the first 4 hours mortality is low, but if surgical treatment is delayed mortality may be high. The prognosis is very closely related to the condition of the patient at the time of surgery. The hematoma can be anywhere in the cranial vault and is often associated with a contused and lacerated brain. Acute subdural hematomas can also occur in the rupture of berry aneurysms or in blood dyscrasias.

Subacute. Lesions can develop in 3 days to 3 weeks, and deterioration can be due as much to swelling of underlying hemisphere as to the enlargement of the hematoma collection itself; hemiparesis is present in 50% of the patients contralateral to the hematoma, but in 25% it is ipsilateral. Mortality is 25%.

Chronic. A lesion can manifest 3 weeks or more after trauma. Researchers disagree as to pathophysiologic mechanism governing the gradual increase in size of these lesions, but intermittent rebleeding is probably the major factor. Accumulation of fluid caused by an osmotic gradient in the subdural membrane has also been postulated but seems less likely to be the mechanism. Clinically the chronic subdural hematoma can appear as personality or other mental change, signs of focal neurologic dysfunction, increased intracranial pressure, or focal seizures. The diagnosis is established by CT/MRI. A fluctuating level of consciousness can occur but is not a prominent feature. Surgery is required, and the mortality rate is 15%. Some hematomas are not diagnosed clinically and can resorb spontaneously or become calcified or even ossified; detection is often made on a scan done for an unrelated condition.

INTRACEREBRAL HEMORRHAGE

By definition intracerebral hemorrhages represent collections of blood of more than 5 ml. They are most commonly found in either the temporal or frontal lobes, although occasionally they can be present in the occipital lobe or cerebellum. With blow to the head (coup lesions), there may be injury to the opposite brain region as brain strikes the inner table of the skull (contrecoup lesions). Contusions and hemorrhages may develop and enlarge one to two days following the head injury. Rotational injuries may cause shearing of blood vessels within brain parenchyma to cause intracerebral hemorrhage. Intracerebral hemorrhages are usually located within deep white matter and contusions are more superficial cortical in location. These lesions are usually associated with severe trauma and clinically appear as expanding mass lesions with significant focal neurologic deficit. Traumatic intracerebral hemorrhage usually occurs in superficial locations. It is important to differentiate traumatic intracerebral hemorrhage from other types of nontraumatic intracerebral hemorrhage (e.g., hypertension, amyloid angiopathy, rupture angioma, aneurysm) that resulted in a neurologic deficit (e.g., seizure, hemiplegia) and that may have caused the patient to sustain head trauma either by a fall or auto accident.

SKULL FRACTURES

These are classified as linear depressed and comminuted. Compound fractures have scalp laceration directly over the fracture line. Skull fractures are indicators of potentially serious

traumatic brain injury and these patients need to have CT. Linear fractures are most common where skull is thin – temporal-parietal region. These linear fractures do not require surgery and heal spontaneously. In depressed skull fracture, bone is displaced inward to tear the dura and contuse the underlying superficial brain. In comminuted fracture, there are multiple broken bone fragments which are usually but not necessarily inwardly displaced. If dura is torn, CSF leak may occur and meningitis may develop and underlying brain is contused or lacerated. Also, injury to venous dural sinus may occur to result in venous sinus thrombosis. Depressed and comminuted fractures usually require surgical intervention to elevate bone fragments, debride and decompress contused tissue, repair dural injury.

BASILAR SKULL FRACTURE OR FRACTURES ACROSS PARANASAL SINUSES

Any fracture that crosses a paranasal sinus or results in a dural tear of the cribriform plate (causing cerebrospinal fluid rhinorrhea) or temporal bone (causing otorrhea) produces a communication between the subarachnoid space and the environment. The possibility of meningitis occurring in this situation is very real and demands special observation and treatment.

Basilar fractures are difficult to diagnose by roentgenography (less than 50% on routine skull films and CT with “bone windows”), so they must be inferred from clinical signs. Special base-of-skull tomograms can help identify these fractures. Fracture of petrous temporal bone may cause hemotympanium, tympanic perforation, tinnitus, vertigo, hearing loss, lower motor neuron facial weakness, ecchymosis of scalp in region of mastoid process (Battle sign) and otorrhea. With frontal fracture, look for anosmia, bilateral periorbital ecchymosis (raccoon eyes, rhinorrhea). If the fracture is through a sinus or cribriform plate, air can often be seen intracranially on either the skull film or, even more reliably, CT scan. CSF can sometimes be seen in the paranasal sinuses. Chronic CSF rhinorrhea can usually be accurately diagnosed and the location of the leak identified by introducing a radioactive isotope into the CSF and performing a nuclide cisternogram. Water-soluble contrast material can also be used and CT scanning employed to identify the site of the leak.

Treatment of a CSF fluid leak consists of bed rest for 7 to 10 days. The use of prophylactic antibiotics is controversial. If antibiotics are used, they should be continued at least 1 week after the leak closes spontaneously or is closed surgically. Unfortunately meningitis can develop many months or even years after the trauma and even with adequate treatment can recur. Most CSF leaks stop spontaneously in the first week; this is particularly true of otorrhea. If the leak does not spontaneously seal with 10 days, surgical exploration is necessary. If the fracture and fistula involve the frontal or sphenoidal sinus, the likelihood of spontaneous healing is less, necessitating surgical intervention sooner.

Basilar fracture can also damage facial or acoustic nerves, so assessment of cranial nerve function is necessary in each case.

DEPRESSED SKULL FRACTURES

A skull fracture is considered to be compound if there is a scalp laceration. If the dura is torn, this is a penetrating injury. Depressed fractures can result from motor vehicle (including motorcycle) accidents or from the head being struck by objects (work-related injuries, assault attacks), or they can be caused by missile injuries. Diagnosis of the fracture is established by a skull roentgenogram, and evidence of an underlying focal contusion is established by CT/MRI scanning. The major complication of depressed fracture is meningitis. Treatment of a depressed fracture consists of surgical debridement, elevation of the fracture, repair of the dura (if it has been torn), and antibiotics.

SEQUELAE OF HEAD INJURY

The most significant sequelae occur in the cases of severe injury, but there are some very important problems that can occur after mild trauma. In general, recovery from any head trauma is better in young patients. Improvement proceeds slowly over many weeks or months

DIFFUSE DAMAGE (Posttraumatic Encephalopathy)

The most severe form of diffuse damage is the chronic vegetative state in which the patient has multiple neurologic abnormalities and is so severely damaged that, even though the eyes are open, meaningful contact with the environment is minimal or nonexistent. All grades of recovery occur in diffuse damage, and varying degrees of neurologic impairment are demonstrated. In general the prognosis is directly related to the length of posttraumatic amnesia or coma and inversely related to the age of the patient.

One common sequela of blunt head trauma that is often not fully appreciated until the major motor disabilities have cleared is the behavioral effect of bilateral frontal and temporal damage. Because of the contour of the inner surface of the skull, the orbital (inferomedial) surface of the frontal lobes and the medial temporal lobes are often contused or otherwise damaged during the injury. In moderate concussions other neurologic deficits can be mild. The results of frontal or temporal trauma are twofold. From the temporal damage there develops a recent memory or new learning deficit. This is usually subtle and reversible in slight or mild concussion but can be a significant problem in more severe injuries. Such a deficit can greatly hinder job performance, particularly if the patient must learn a new vocation after the accident. The memory problem can improve slowly over years, but improvement is most rapid in the first 12 months after the trauma. Frontal lesions can cause a very distressing change in personality or temperament. This is characterized by a lack of motivation, apathy, at times euphoric disinterest, a lack of social restraint, and a distressing lack of goal direction. Pursuant to these and other intellectual deficits it is suggested that all patients who suffer significant head trauma should have a full neuropsychologic evaluation before returning to work or entering a vocational rehabilitation program.

FOCAL DAMAGE

Focal contusions or hematomas will often leave residual focal neurologic deficits (e.g., aphasia, hemiplegia, hemiparesis, visual field defect).

HYDROCEPHALUS

After trauma there is occasionally sufficient blood or fibrous adhesions in the subarachnoid space at the base of the brain to obstruct the flow of CSF. In such instances a communicating hydrocephalus can develop. This diagnostic possibility should always be considered in any patients with head trauma in whom deterioration subsequently occurs. This can be during the acute phase (mostly from blood) or many weeks or months later. The clinical signs in these cases will be gait unsteadiness, incontinence, behavioral changes often simulating a frontal lobe syndrome, or intermittent confusion.

CRANIAL NERVE ABNORMALITIES

The cranial nerves are anchored to the skull as they exit through the various foramina; because of this they are often damaged when the skull is fractured or the brain is thrown about during acceleration/deceleration injury. The effects injury can have on the first, second sixth, seventh, and eighth cranial systems are listed in Box 12-2.

Box 12-2

1. First (olfactory). This is the most commonly damaged nerve, especially with basilar skull fractures. In patients with cerebrospinal fluid rhinorrhea, anosmia (loss of sense of smell) is extremely common. Recovery is slow and related to the severity of the trauma. In general less than 50% of the patients recover full function. On occasion there is recovery for some odors and not others, and sometimes the sense of smell is distorted. To some persons anosmia is a minor inconvenience, but to others it can be a major disability, for instance, for a restaurant chef or wine producer.
2. Second (optic). Direct injury to either the second cranial nerve or chiasm occurs. Often the resultant loss of vision is transient.
3. Sixth (abducens). This is the most commonly affected nerve of the extraocular muscles and injury may result in diplopia.
4. Seventh (facial). With fracture through the temporal bone, the seventh nerve is often interrupted acutely. In some patients without fracture, delayed (usually 2-3 days) facial paralysis can manifest.
5. Eighth (acoustic). In basilar fractures in which otorrhea or hematomotympanum is present, hearing can be affected as well as vestibular function.

SEIZURES AND ELECTROENCEPHALOGRAPHIC CHANGES

After acute head injury the EEG can show slowing of background rhythm, suppression of voltage, or paroxysms (bursts) of slow activity. This is also true in a high percentage of patients with severe whiplash. The incidence of such abnormalities is highest in children. As acute effects of the injury dissipate (2 to 3 weeks) the EEG will either return to normal or show evidence of focal residual damage or dysfunction. Focal slowing is the most common finding, but focal or even generalized epileptic activity is occasionally seen. If an EEG is to be ordered at all, it is best to obtain it in the recovery phase rather than acutely. The most important information to be gained from the EEG is the probability of a patient developing posttraumatic

epilepsy. If the EEG show abnormal bursts, the patient has a 20% to 25% chance of developing seizures and therefore should be treated with anticonvulsants. This is particularly true if there is any family history of epilepsy.

There is a definite risk of developing a chronic, recurring seizure disorder after significant head traumas. The four factors listed in Box 12-3 significantly raise the probability of developing posttraumatic epilepsy.

In simple concussions in which there is only a fleeting loss of consciousness, the probability of developing a seizure disorder is no greater than that in the general population. Immediately following blow to the head, impact seizure may occur. This does not usually recur and does not require treatment.

Seizures indicate injury to brain parenchyma and may occur in the early post-traumatic period due to hemorrhage as blood is epileptogenic. As blood resolves seizures may stop and anti-epileptic medication discontinued. Seizures can first appear many years after an accident, and prophylactic treatment does not seem to be efficacious in preventing their appearance. These seizures are probably due to the presence of gliotic scar tissue which forms in the injured brain region. Most posttraumatic seizures are focal and are more difficult to treat than idiopathic epilepsy. Posttraumatic epilepsy is a chronic disorder,, with only a 40% cure rate after 5 years.

Box 12-3

1. Depressed fractures particularly with dural tear have a very high incidence of seizures (>50%)
2. Penetrating wounds or grossly destructive lesions have high incidence (>50%)
3. Amnesia (>24 hours)
4. Time after injury that the first seizure occurs:
 - a. Immediate (little chance of chronic seizures)
 - b. First week (35%)
 - c. First to eighth week (70%)

POSTTRAUMATIC SYNDROME

Posttraumatic syndrome is the most common and probably most complex sequela of head trauma. The basic elements of the syndromes are headache, dizziness, visual blurring (due to convergence insufficiency), difficulty concentrating, and a host of vague behavioral symptoms such as anxiety, depression, and nervous instability. This syndrome is curiously more common in trivial or slight trauma than it is in serious injury. It is logical that some of these symptoms should occur after an acute injury because a blow to the head will have a number of organic effects such as the following; injury to the soft tissues of the skull or meninges will cause headache; labyrinthine dysfunction has been well documented to explain the dizziness,

convergence insufficiency may cause visual blurring when reading, and trauma to the ascending activating system as well as limbic system and cortex will lead to difficulty with concentration, memory, and emotional stability.

The syndrome usually lasts a few weeks to a few months. The symptoms gradually disappear but can be exacerbated by strenuous physical exercise, emotional stress, or the use of alcohol. Patients in laboring jobs or highly stressful jobs should be warned that symptoms might recur when they return to work. Employers must also know this and be sympathetic. Rest and symptomatic treatment are usually all that is required.

A major problem arises when the symptoms do not abate in a reasonable length of time. Such patients often become depressed and angry, which are factors that can cause the symptoms to increase. Because many head injuries occur in vehicle or work-related accidents, the problems of fault and compensation arise. When a person feels that someone else is to blame for his or her injury, anger and desire for recompense can consciously or unconsciously perpetuate the symptoms. It has been repeatedly shown that head injuries incurred during sporting events have a much lower incidence of posttraumatic symptomatology than injuries in which fault is an issue. Many, but not necessarily all, of the patients with symptoms lasting more than 6 to 12 months probably have considerable psychologic, legal, financial, or other social factors responsible for the perpetuation of their symptoms. The physician should maintain a sympathetic interest in helping the patient over the posttraumatic period but must also be aware of these other often significant factors. Simple symptomatic treatment is best. Narcotic medications should not be given for more than a few weeks after the accident, except in rare cases.

In some patients trauma to the scalp and branches of the external carotid artery produces migrainelike headaches. These headaches should be treated in the same fashion as other migraines, but the success of treatment is not as satisfactory.

SUMMARY

Head trauma is a common medical problem. Most head injuries are mild, but some patients develop serious complications such as intracranial hematomas (e.g., subdural or epidural). Neurosurgical consultation is the most important aspect of the management of significant head trauma, but the evaluation and management of mild head trauma can be adequately handled by any physician. CT scanning has been a great comfort in screening head trauma patients but is not necessary in all cases. The posttraumatic syndrome is one of the complex problems from head injury; this condition is usually managed by the primary care physician.

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Figure 12-1. CT scan (nonenhanced) of a large subdural hematoma.

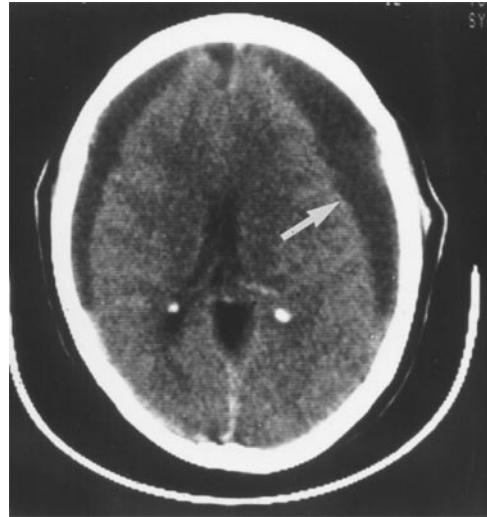


Figure 12-2. CT scan demonstrating bilateral hypodense subdural hematomas. The one on the right (*arrow*) is chronic; note lens shape. The one on the left is subacute; note how it follows the contour of the convexity.

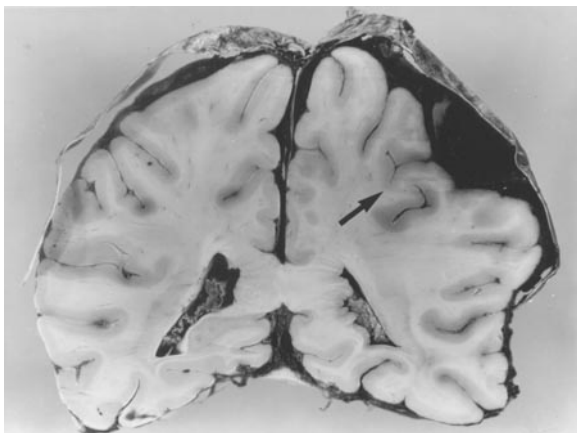


Figure 12-3. Pathologic specimen corresponding to the scan in Figure 12-2.

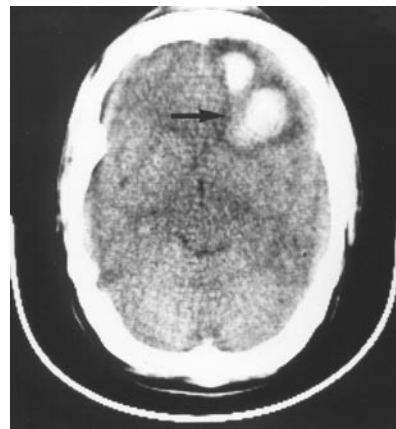


Figure 12-4. Traumatic intracerebral hematoma in the right lobe (*arrow*).