457e Concussion and Other Traumatic Brain Injuries

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Almost 10 million head injuries occur annually in the United States, about 20% of which are serious enough to cause brain damage. Among men <35 years, accidents, usually motor vehicle collisions, are the chief cause of death and >70% of these involve head injury. Furthermore, minor head injuries are so common that almost all physicians will be called upon to provide immediate care or to see patients who are suffering from various sequelae.

Medical personnel caring for head injury patients should be aware that (1) spinal injury often accompanies head injury, and care must be taken in handling the patient to prevent compression of the spinal cord due to instability of the spinal column; (2) intoxication is frequently associated with traumatic brain injury, and thus testing for drugs and alcohol should be carried out when appropriate; and (3) additional injuries, including rupture of abdominal organs, may produce vascular collapse, shock, or respiratory distress that requires immediate attention.

TYPES OF HEAD INJURIES

CONCUSSION

This form of minor head injury had in the past referred to an immediate and transient loss of consciousness that was associated with a short period of amnesia. Many patients, however, do not lose consciousness after a minor head injury but instead are dazed or confused, or feel stunned or "star struck," and the term *concussion* is now applied to all such cognitive and perceptual changes experienced after a blow to the head. Severe concussion may precipitate a brief convulsion or autonomic signs such as facial pallor, bradycardia, faintness with mild hypotension, or sluggish pupillary reaction, but most patients quickly return to a neurologically normal state.

The mechanics of a typical concussion involve sudden deceleration of the head when hitting a blunt stationary object. This creates an anterior-posterior movement of the brain within the skull due to inertia and rotation of the cerebral hemispheres on the fulcrum of the relatively fixed upper brainstem. Loss of consciousness in concussion is believed to result from a transient electrophysiologic dysfunction of the reticular activating system in the upper midbrain that is at the site of rotation (Chap. 328). The transmission of a wave of kinetic energy throughout the brain is an alternative explanation for the disruption in consciousness.

Gross and light-microscopic changes in the brain are usually absent following concussion, but biochemical and ultrastructural changes, such as mitochondrial ATP depletion and local disruption of the blood-brain barrier, may be transient abnormalities. Computed tomography (CT) and magnetic resonance imaging (MRI) scans are usually normal; however, a small number of patients will be found to have a skull fracture, an intracranial hemorrhage, or a brain contusion.

A brief period of both retrograde and anterograde amnesia is characteristic of concussion, and it recedes rapidly in alert patients. Memory loss spans the moments before impact but may encompass the previous days or weeks (rarely months). With severe injuries, the extent of retrograde amnesia roughly correlates with the severity of injury. Memory is regained erratically from the most distant to more recent memories, with islands of amnesia occasionally remaining. The mechanism of amnesia is not known. Hysterical posttraumatic amnesia is not uncommon after head injury and should be suspected when inexplicable behavioral abnormalities occur, such as recounting events that cannot be recalled on later testing, a bizarre affect, forgetting one's own name, or a persistent anterograde deficit that is excessive in comparison with the degree of injury. Amnesia is discussed in Chap. 36.

A single, uncomplicated concussion only infrequently produces permanent neurobehavioral changes in patients who are free of preexisting psychiatric and neurologic problems. Nonetheless, residual problems in memory and concentration may have an anatomic correlate in microscopic cerebral lesions (see below).

The mechanisms by which a blast injury affects the brain and causes symptoms that are associated with concussion, a problem mainly in military medicine, are not known. The energy of a blast wave can enter the cranium through the openings of the orbits, auditory canals, and foramen magnum. There are not consistent changes in cerebral imaging studies but more subtle indications of tissue disruption have been found, comparable to those of mild concussion. It has been difficult to separate the direct effects of the blast from the consequences of being thrown against fixed objects or injured by flying debris.

CONTUSION, BRAIN HEMORRHAGE, AND AXONAL SHEARING LESIONS

These pathologic changes are the result of severe cranial trauma. A surface bruise of the brain, or *contusion*, consists of varying degrees of petechial hemorrhage, edema, and tissue destruction. Contusions and deeper hemorrhages result from mechanical forces that displace and compress the hemispheres forcefully and by deceleration of the brain against the inner skull, either under a point of impact (coup lesion) or, as the brain swings back, in the antipolar area (contrecoup lesion). Trauma sufficient to cause prolonged unconsciousness usually produces some degree of contusion. Blunt deceleration impact, as occurs against an automobile dashboard or from falling forward onto a hard surface, causes contusions on the orbital surfaces of the frontal lobes and the anterior and basal portions of the temporal lobes. With lateral forces, as from impact on an automobile door frame, contusions are situated on the lateral convexity of the hemisphere. The clinical signs of contusion are determined by the location and size of the lesion; often, there are no focal neurologic abnormalities, but these injured regions are later the sites of gliotic scars that may produce seizures. A hemiparesis or gaze preference is fairly typical of moderately sized contusions. Large bilateral contusions produce stupor with extensor posturing, while those limited to the frontal lobes cause a taciturn state. Contusions in the temporal lobe may cause delirium or an aggressive, combative syndrome.

Acute contusions are easily visible on CT and MRI scans, appearing as inhomogeneous hyperdensities on CT and as hyperintensities on T2 and fluid-attenuated inversion recovery (FLAIR) MRI sequences; there is usually surrounding localized brain edema (Fig. 457e-1) and some subarachnoid bleeding. Blood in the cerebrospinal fluid (CSF) due to trauma may provoke a mild inflammatory reaction. Over a few days, contusions acquire a surrounding contrast enhancement and edema that may be mistaken for tumor or abscess. Glial and macrophage reactions result in chronic, scarred, hemosiderin-stained depressions on the cortex (*plaques jaunes*) that are the main source of posttraumatic epilepsy.

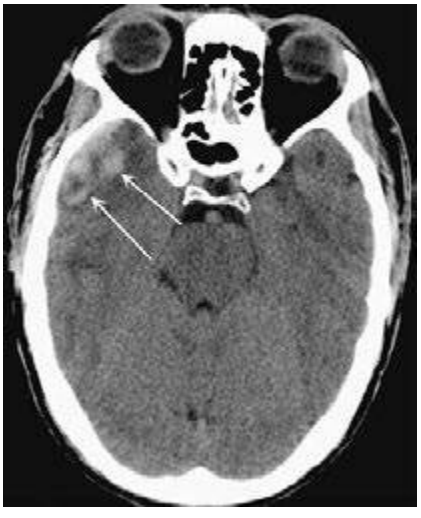

FIGURE 457e-1 Traumatic cerebral contusion. Noncontrast computed tomography scan demonstrating a hyperdense hemorrhagic region in the anterior temporal lobe.