trauma may or may not be elicited in relation to chronic subdural hematoma; the injury may have been trivial and forgotten, particularly in the elderly and those with clotting disorders. Headache is common but not invariable. Additional features that may appear weeks later include slowed thinking, vague change in personality, seizure, or a mild hemiparesis. Headache fluctuates in severity, sometimes with changes in head position.

Bilateral chronic subdural hematomas produce perplexing clinical syndromes, and the initial clinical impression may be of a stroke, brain tumor, drug intoxication, depression, or a dementing illness. Drowsiness, inattentiveness, and incoherence of thought are generally more prominent than focal signs such as hemiparesis. Rarely, chronic hematomas cause brief episodes of hemiparesis or aphasia that are indistinguishable from transient ischemic attacks. Patients with undetected bilateral subdural hematomas have a low tolerance for surgery, anesthesia, and drugs that depress the nervous system; drowsiness or confusion persists for long periods postoperatively.

CT without contrast initially shows a low-density mass over the convexity of the hemisphere (Fig. 457e-5). Between 2 and 6 weeks after the initial bleeding, the clot becomes isodense compared to adjacent brain and may be inapparent. Many subdural hematomas that are several weeks in age contain areas of blood and intermixed serous fluid. Bilateral chronic hematomas may fail to be detected because of the absence of lateral tissue shifts; this circumstance in an older patient is suggested by a "hypernormal" CT scan with fullness of the cortical sulci and small ventricles. Infusion of contrast material demonstrates enhancement of the vascular fibrous capsule surrounding the collection. MRI reliably identifies subacute and chronic hematomas.

Clinical observation coupled with serial imaging is a reasonable approach to patients with few symptoms, such as headache alone, and in those with small chronic subdural collections. Treatment of minimally symptomatic chronic subdural hematoma with glucocorticoids is favored by some clinicians, but surgical evacuation is more often successful. The fibrous membranes that grow from the dura and encapsulate the collection require removal to prevent recurrent fluid accumulation. Small hematomas are resorbed, leaving only the organizing membranes. On imaging studies, very chronic subdural hematomas are difficult to distinguish from hygromas, which are collections of CSF from a rent in the arachnoid membrane.

CLINICAL SYNDROMES AND TREATMENT OF HEAD INJURY

MINOR INJURY

The patient who has briefly lost consciousness or been stunned after a minor head injury usually becomes fully alert and attentive within minutes but may complain of headache, dizziness, faintness, nausea, a single episode of emesis, difficulty with concentration, a brief amnestic period, or slight blurring of vision. This typical concussion syndrome has a good prognosis with little risk of subsequent deterioration. Children are particularly prone to drowsiness, vomiting, and irritability, symptoms that are sometimes delayed for several hours after apparently minor injuries. Vasovagal syncope that follows injury may cause undue concern. Generalized or frontal headache is common in the following days. It may be migrainous (throbbing and hemicranial) in nature or aching and bilateral. After several hours of observation, patients with minor injury may be accompanied home and observed for a day by a family member or friend, with written instructions to return if symptoms worsen.

Persistent severe headache and repeated vomiting in the context of normal alertness and no focal neurologic signs is usually benign, but CT should be obtained and a longer period of observation is appropriate. The decision to perform imaging tests also depends on clinical signs that indicate that the impact was severe (e.g., persistent confusion, periorbital or mastoid hematoma, repeated vomiting, palpable skull fracture), on the seriousness of other bodily injuries, and on the degree of surveillance that can be anticipated after discharge. Two studies have indicated that older age, two or more episodes of vomiting, >30 min of retrograde or persistent anterograde amnesia, seizure, and concurrent drug or alcohol intoxication are sensitive (but not specific) indicators of intracranial hemorrhage that justify CT scanning. It may be appropriate to be more liberal in obtaining CT scans in children because a small number, even without loss of consciousness, will have intracranial traumatic lesions but this exposes the child to radiation.

Concussion in Sports In the current absence of adequate data, a common sense approach to athletic concussion has been to remove the individual from play immediately and avoid contact sports for at least several days after a mild injury and for a longer period if there are more severe injuries or if there are protracted neurologic symptoms such as headache and difficulty concentrating. No individual should return to play unless all symptoms have resolved and an assessment has been made by a health care professional who has experience with treatment of concussion. Once cleared, the individual can then begin a graduated program of increasing activity. Younger athletes are particularly likely to experience protracted concussive symptoms, and a slower return to play in this age group may be reasonable. These guidelines are designed in part to avoid a perpetuation of symptoms but also to prevent the rare *second impact syndrome*, in which diffuse and fatal cerebral swelling follows a second minor head injury.

In the past, mental decline in boxers late in their careers had been called *dementia pugilistica*. There is some evidence that repeated concussions from other sports are associated with a similar delayed and progressive cognitive disorder that is due mainly to the deposition of tau protein in cortical neurons. The brains of these patients display deposition of tau protein in the superficial cortical layers, and particularly in the depths of sulci within the frontal cortices, a pattern named *chronic traumatic encephalopathy* (CTE) that is quite unlike other degenerative conditions. CTE is an intensively studied and provocative entity. Its contribution, if any, to late-life dementia and parkinsonism in former athletes, soldiers, or others who have sustained repeated concussive injuries is unknown. **CTE is also discussed in Chap. 444e.**

INJURY OF INTERMEDIATE SEVERITY

Patients who are not fully alert or have persistent confusion, behavioral changes, extreme dizziness, or focal neurologic signs such as hemiparesis should be admitted to the hospital and have cerebral imaging. A cerebral contusion or hematoma will usually be found. Common syndromes include: (1) delirium with a disinclination to be examined or moved, expletive speech, and resistance if disturbed (anterior temporal lobe contusions); (2) a quiet, disinterested, slowed mental state (abulia) alternating with irascibility (inferior frontal and frontopolar contusions); (3) a focal deficit such as aphasia or mild hemiparesis (due to subdural hematoma or convexity contusion or, less often, carotid artery dissection); (4) confusion and inattention, poor performance on simple mental tasks, and fluctuating orientation (associated with several types of injuries, including those described above, and with medial frontal contusions and interhemispheric subdural hematoma); (5) repetitive vomiting, nystagmus, drowsiness, and unsteadiness (labyrinthine concussion, but occasionally due to a posterior fossa subdural hematoma or vertebral artery dissection); and (6) diabetes insipidus (damage to the median eminence or pituitary stalk). Injuries of this degree are often complicated by drug or alcohol intoxication, and clinically inapparent cervical spine injury may be present. Blast injuries are often accompanied by rupture of the tympanic membranes.

After surgical removal of hematomas, most patients in this category improve over weeks. During the first week, the state of alertness, memory, and other cognitive functions often fluctuate, and agitation and somnolence are common. Behavioral changes tend to be worse at night, as with many other encephalopathies, and may be treated with small doses of antipsychotic medications. Subtle abnormalities of attention, intellect, spontaneity, and memory return toward normal weeks or months after the injury, sometimes abruptly. Persistent cognitive problems are discussed below.

SEVERE INJURY

Patients who are comatose from the moment of injury require immediate neurologic attention and resuscitation. After intubation, with care taken to immobilize the cervical spine, the depth of coma,