risk of future seizures. It has been estimated that 17% of individuals with brain contusion, subdural hematoma, or prolonged loss of consciousness will develop a seizure disorder and that this risk extends for an indefinite period of time, whereas the risk is ≤2% after mild injury. The majority of convulsions in the latter group occur within 5 years of injury but may be delayed for decades. Penetrating injuries have a much higher rate of subsequent epilepsy.

SUBDURAL AND EPIDURAL HEMATOMAS

Hemorrhages beneath the dura (subdural) or between the dura and skull (epidural) have characteristic clinical and imaging features. They are sometimes associated with underlying contusions and other injuries, often making it difficult to determine the relative contribution of each component to the clinical state. The mass effect and raised ICP caused by these hematomas can be life threatening, making it imperative to identify them rapidly by CT or MRI scan and to remove them when appropriate.

Acute Subdural Hematoma (Fig. 457e-3) Direct cranial trauma may be minor and is not required for acute subdural hemorrhage to occur, especially in the elderly and those taking anticoagulant medications. Acceleration forces alone, as from whiplash, are sometimes sufficient to produce subdural hematoma. Up to one-third of patients have a lucid interval lasting minutes to hours before coma supervenes, but most are drowsy or comatose from the moment of injury. A unilateral headache and slightly enlarged pupil on the side of the hematoma are frequently, but not invariably, present. Stupor or coma, hemiparesis, and unilateral pupillary enlargement are signs of larger hematomas. In an acutely deteriorating patient, burr (drainage) holes or an emergency craniotomy are required. Small subdural hematomas may be asymptomatic and usually do not require evacuation if they do not enlarge.

A subacutely evolving syndrome due to subdural hematoma occurs days or weeks after injury with drowsiness, headache, confusion, or mild hemiparesis, usually in alcoholics and in the elderly and often after only minor trauma. On imaging studies, subdural hematomas appear as crescentic collections over the convexity of one or both hemispheres, most commonly in the frontotemporal region, and less often in the inferior middle fossa or over the occipital poles (Fig. 457e-3). Interhemispheric, posterior fossa, or bilateral convexity hematomas are less frequent and are difficult to diagnose clinically, although drowsiness and the neurologic signs expected from damage in each region can usually be detected. The bleeding that causes larger hematomas is primarily venous in origin, although additional arterial bleeding sites are sometimes found at operation, and a few large hematomas have a purely arterial origin.

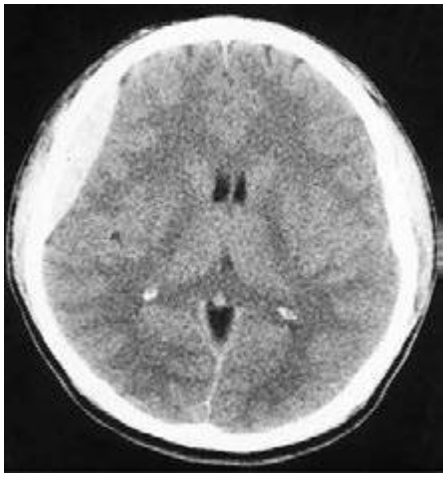

FIGURE 457e-4 Acute epidural hematoma. The tightly attached dura is stripped from the inner table of the skull, producing a characteristic lenticular-shaped hemorrhage on noncontrast computed tomography scan. Epidural hematomas are usually caused by tearing of the middle meningeal artery following fracture of the temporal bone.

Epidural Hematoma (Fig. 457e-4) These usually evolve more rapidly than subdural hematomas and are correspondingly more treacherous. They occur in up to 10% of cases of severe head injury but are associated with underlying cortical damage less often than for subdural hematomas. Most patients are unconscious when first seen. A "lucid interval" of several minutes to hours before coma supervenes is most characteristic of epidural hemorrhage, but it is still uncommon, and epidural hemorrhage is not the only cause of this temporal sequence. Rapid surgical evacuation and ligation or cautery of the damaged vessel is indicated, usually the middle meningeal artery that has been lacerated by an overlying skull fracture.

Chronic Subdural Hematoma (Fig. 457e-5) A subacutely evolving syndrome due to subdural hematoma occurs days or weeks after injury with drowsiness, headache, confusion, or mild hemiparesis, usually in alcoholics and in the elderly and often after only minor or unnoticed trauma. On imaging studies, chronic subdural hematomas appear as crescentic clots over the convexity of one or both hemispheres, most commonly in the frontotemporal region (Fig. 457e-3). A history of

FIGURE 457e-3 Acute subdural hematoma. Noncontrast computed tomography scan reveals a hyperdense clot that has an irregular border with the brain and causes more horizontal displacement (mass effect) than might be expected from its thickness. The disproportionate mass effect is the result of the large rostral-caudal extent of these hematomas. Compare to Fig. 457e-4.

FIGURE 457e-5 Computed tomography scan of chronic bilateral subdural hematomas of different ages. The collections began as acute hematomas and have become hypodense in comparison to the adjacent brain after a period during which they were isodense and difficult to appreciate. Some areas of resolving blood are contained on the more recently formed collection on the left (*arrows*).