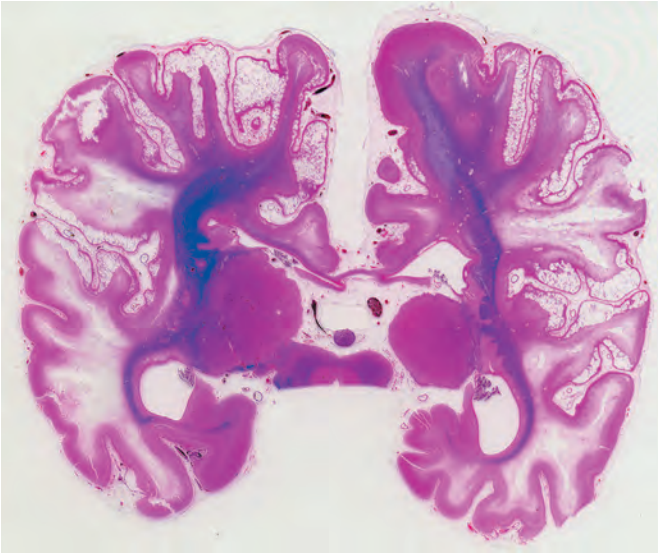

Figure 28-8 Multicystic leukoencephalopathy. Numerous cystic spaces representing the consequences of widespread ischemic injury are present.

spasticity, dystonia, ataxia/athetosis, and paresis, attributable to insults occurring during the prenatal and perinatal periods. Signs and symptoms may not be apparent at birth and only declare themselves later, as development proceeds. Postmortem examinations of children with cerebral palsy have shown a wide range of neuropathologic findings, including destructive lesions traced to remote events that may have caused hemorrhage and infarction.

In premature infants there is an increased risk of *intraparenchymal hemorrhage* within the germinal matrix, often near the junction between the developing thalamus and caudate nucleus. Hemorrhages may remain localized or extend into the ventricular system and thence to the subarachnoid space, sometimes leading to hydrocephalus.

Infarcts may occur in the supratentorial periventricular white matter *(periventricular leukomalacia)*, especially in premature infants. These take the form of chalky yellow plaques consisting of discrete regions of white matter necrosis and calcification. When both gray and white matter are involved by extensive ischemic damage, large destructive cystic lesions develop throughout the hemispheres; this condition is termed *multicystic encephalopathy* (Fig. 28-8).

In perinatal ischemic lesions of the cerebral cortex, the depths of sulci bear the brunt of injury and result in thinned-out, gliotic gyri *(ulegyria)*. The basal ganglia and thalamus may also suffer ischemic injury, with patchy neuronal loss and reactive gliosis. Later, aberrant and irregular myelinization gives rise to a marble-like appearance of the deep nuclei (*status marmoratus*). Because the lesions are in the caudate, putamen, and thalamus, movement disorders such as choreoathetosis are common clinical sequelae.

Trauma

The anatomic location of the lesion and the limited capacity of the brain for functional repair are major determinants of the consequences of CNS trauma. Injury of several cubic centimeters of brain parenchyma may be clinically silent (if in the frontal lobe), severely disabling (if in the spinal cord), or fatal (if in the brainstem).

The physical forces associated with head injury may result in *skull fractures, parenchymal injury,* and *vascular injury;* all three can coexist. The magnitude and distribution of a traumatic brain lesions depend on the shape of the object causing the trauma, the force of impact, and whether the head is in motion at the time of injury. A blow to the head may be *penetrating* or *blunt*; it may cause either an *open* or a *closed injury*.

Skull Fractures

A fracture in which bone is displaced into the cranial cavity by a distance greater than the thickness of the bone is called a *displaced skull fracture*. The thickness of the cranial bones varies; therefore, their resistance to fracture differs greatly. Also, the relative incidence of fractures among skull bones is related to the pattern of falls. When an individual falls while awake, such as might occur when stepping off a ladder, the site of impact is often the occipital portion of the skull; in contrast, a fall that follows loss of consciousness, as might follow a syncopal attack, commonly results in a frontal impact. Symptoms referable to the lower cranial nerves or the cervicomedullary region, and the presence of orbital or mastoid hematomas distant from the point of impact, raise the suspicion of a basal skull fracture, which typically follows impact to the occiput or sides of the head. CSF discharge from the nose or ear and infection (meningitis) may follow. The kinetic energy that causes a fracture is dissipated at a fused suture; fractures that cross sutures are termed *diastatic*. With multiple points of impact or repeated blows to the head, the fracture lines of subsequent injuries do not extend across fracture lines of prior injury.

Parenchymal Injuries

Concussion

Concussion is a clinical syndrome of altered consciousness secondary to head injury typically brought about by a change in the momentum of the head (when a moving head is suddenly arrested by impact on a rigid surface). The characteristic neurologic picture includes instantaneous onset of transient neurologic dysfunction, including loss of consciousness, temporary respiratory arrest, and loss of reflexes. Although neurologic recovery is complete, amnesia for the event often persists. The pathogenesis of the sudden disruption of neurologic function is unknown; it probably involves dysregulation of the reticular activating system in the brainstem. Post-concussive neuropsychiatric syndromes, typically associated with repetitive injuries, are well recognized and there is increasing evidence that significant cognitive impairment can emerge along with distinct pathologic findings terms chronic traumatic encephalopathy (discussed later).

Direct Parenchymal Injury

Contusions and *lacerations* are brain injuries caused by transmission of kinetic energy to the brain. A contusion is analogous to the familiar bruise caused by blunt trauma, while a laceration is an injury caused by penetration of an