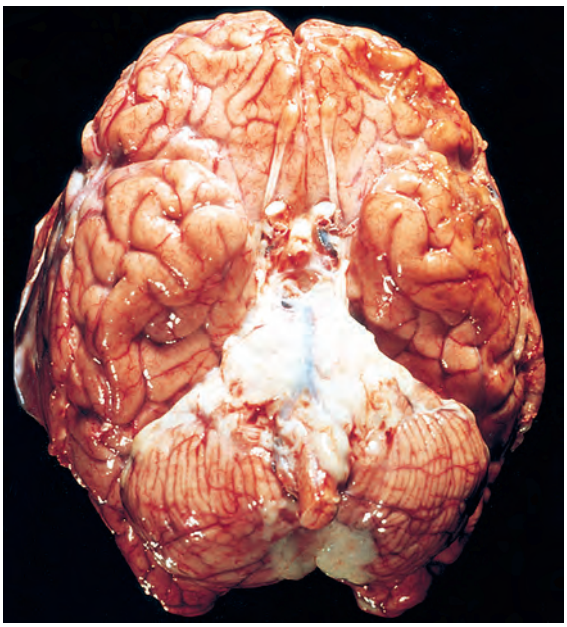

Figure 28-21 Pyogenic meningitis. A thick layer of suppurative exudate covers the brainstem and cerebellum and thickens the leptomeninges. (From Golden JA, Louis DN: Images in clinical medicine: acute bacterial meningitis. N Engl J Med 333:364, 1994.)

MORPHOLOGY

In acute meningitis, an exudate is evident within the leptomeninges over the surface of the brain (Fig. 28-21). The meningeal vessels are engorged and stand out prominently. The anatomic distribution of the exudate varies; in *H. influenzae* meningitis, for example, it is usually basal, whereas in pneumococcal meningitis it is often densest over the cerebral convexities near the sagittal sinus. From the areas of greatest accumulation, tracts of pus follow along blood vessels on the surface of the brain. When the meningitis is fulminant, the inflammation may extend to the ventricles, producing ventriculitis.

On microscopic examination, neutrophils fill the subarachnoid space in severely affected areas and are found predominantly around the leptomeningeal blood vessels in less severe cases. Particularly in untreated meningitis, Gram stain reveals variable numbers of bacteria. In fulminant meningitis, the inflammatory cells infiltrate the walls of the leptomeningeal veins and may extend focally into the substance of the brain (cerebritis). Phlebitis may lead to venous thrombosis and hemorrhagic infarction of the underlying brain.

Leptomeningeal fibrosis may follow pyogenic meningitis and cause hydrocephalus. Particularly in pneumococcal meningitis, large quantities of the capsular polysaccharide of the organism produce a gelatinous exudate that promotes arachnoid fibrosis, a condition referred to as **chronic adhesive arachnoiditis**.

bacterial culture in a patient with manifestations of meningitis, including meningeal irritation, fever, and alterations of consciousness of relatively acute onset. The disease is generally of viral etiology (in about 80% of cases enteroviruses), but may be bacterial, rickettsial, or autoimmune in origin. The clinical course is less fulminant than that of pyogenic meningitis, and the CSF findings also differ; in aseptic meningitis there is a lymphocytic pleocytosis, the protein elevation is only moderate, and the glucose content is nearly always normal. The viral aseptic meningitides are usually self-limited and are treated symptomatically. Remarkably, even with molecular methods for detection of pathogens, the etiologic agent is identified in only a minority of cases. When pathogens are identified, enteroviruses are the most common etiology, accounting for 80% of the cases. The spectrum of pathogens varies seasonally and geographically. An aseptic meningitis-like picture may also develop subsequent to rupture of an epidermoid cyst into the subarachnoid space or the introduction of a chemical irritant (chemical meningitis). In these cases, the CSF is sterile and there is pleocytosis with neutrophils and an increased protein concentration, but the sugar content is usually normal.

Acute Focal Suppurative Infections

Brain Abscess

A brain abscess is a localized focus of necrosis of brain tissue with accompanying inflammation, usually caused by a bacterial infection. Brain abscesses may arise by direct implantation of organisms, local extension from adjacent foci (mastoiditis, paranasal sinusitis), or hematogenous spread (usually from a primary site in the heart, lungs, or bones of the extremities, or after tooth extraction). Predisposing conditions include acute bacterial endocarditis, which may give rise to multiple brain abscesses; congenital heart disease with right-to-left shunting and loss of pulmonary filtration of organisms; chronic pulmonary sepsis, as in bronchiectasis; and systemic disease with immunosuppression. Streptococci and staphylococci are the most common offending organisms identified in non-immunosuppressed patients.

MORPHOLOGY

Abscesses are discrete lesions with central liquefactive necrosis surrounded by brain swelling (Fig. 28-22). At the outer margin of the necrotic lesion there is exuberant granulation tissue with neovascularization around the necrosis. The newly formed vessels are abnormally permeable, accounting for marked vasogenic edema in the adjacent brain tissue. In well-established lesions, a collagenous capsule is produced by fibroblasts derived from the walls of blood vessels. Outside the fibrous capsule is a zone of reactive gliosis containing numerous gemistocytic astrocytes.

Cerebral abscesses are destructive lesions and patients often present with progressive focal neurologic deficits; signs and symptoms related to increased intracranial pressure may also develop. Typically, the CSF has a high white cell count and an increased protein concentration, but the glucose content is normal. The source of infection may be apparent or may be traced to a small distant focus that is not symptomatic. The increased intracranial pressure can lead to fatal herniation. Other complications include abscess rupture with ventriculitis or meningitis, and venous sinus thrombosis. With surgery and antibiotic