Figure 28-17 Lacunar infarcts in the caudate and putamen *(arrows)*.

hemorrhages. In time these hemorrhages resorb, leaving behind a slitlike cavity *(slit hemorrhage)* surrounded by brownish discoloration; on microscopic examination, slit hemorrhages show focal tissue destruction, pigment-laden macrophages, and gliosis.

Hypertensive Encephalopathy

Acute hypertensive encephalopathy is a clinicopathologic syndrome arising in the setting of malignant hypertension, and is characterized by diffuse cerebral dysfunction, including headaches, confusion, vomiting, and convulsions, sometimes leading to coma. Rapid therapeutic intervention to reduce the accompanying increased intracranial pressure is required, since the syndrome often does not remit spontaneously. At postmortem examination such individuals may show an edematous brain with or without transtentorial or tonsillar herniation. Petechiae and fibrinoid necrosis of arterioles in the gray and white matter may be seen microscopically.

Individuals who, over the course of many months and years, suffer multiple, bilateral, gray matter (cortex, thalamus, basal ganglia) and white matter (centrum semiovale) infarcts may develop a distinctive clinical syndrome characterized by dementia, gait abnormalities, and pseudobulbar signs, often with superimposed focal neurologic deficits. The syndrome, generally referred to as *vascular (multi-infarct) dementia*, is caused by multifocal vascular disease of several types, including (1) cerebral atherosclerosis, (2) vessel thrombosis or embolization from carotid vessels or from the heart, and (3) cerebral arteriolar sclerosis from chronic hypertension. When the pattern of injury preferentially involves large areas of the subcortical white matter with myelin and axon loss, the disorder is referred to as *Binswanger disease*; this distribution of vascular white-matter injury must be distinguished clinically and radiologically from other diseases that affect the hemispheral white matter. In addition, many individuals with neurodegenerative diseases resulting in cognitive impairment or dementia also have evidence of cerebrovascular disease. The presence of significant cerebrovascular disease increases risk of neurologic impairment for a given level of lesions associated with the degenerative diseases, suggesting that it is an independent contributing factor to disruption of normal brain function.

Intracranial Hemorrhage

Hemorrhages may occur at any site within the CNS—outside the brain or within it (intraparenchymal). Hemorrhages in the epidural or subdural space are typically associated with trauma and were discussed earlier. Hemorrhages within the brain parenchyma and in the subarachnoid space, in contrast, are more often a manifestation of underlying cerebrovascular disease.

Intraparenchymal Hemorrhage

Rupture of a small intraparenchymal vessel can result in a hemorrhage within the brain, often associated with sudden onset of neurologic symptoms (stroke). Spontaneous (nontraumatic) intraparenchymal hemorrhages occur most commonly in middle to late adult life, with a peak incidence at about age 60 years. Hemorrhages in the basal ganglia and thalamus are commonly designated "ganglionic hemorrhages," whereas those that occur in the lobes of the cerebral hemispheres are called "lobar hemorrhages." The two major causes of these patterns of hemorrhage are hypertension and cerebral amyloid angiopathy, respectively. In addition, other local and systemic factors may cause or contribute to nontraumatic hemorrhage, including systemic coagulation disorders, neoplasms, vasculitis, aneurysms, and vascular malformations.

Hypertension is the risk factor most commonly associated with deep brain parenchymal hemorrhages, accounting for more than 50% of clinically significant hemorrhages and for roughly 15% of deaths among individuals with chronic hypertension. Hemorrhages associated with hypertension are typically in deep white matter or deep gray structures, followed by the brainstem and cerebellum. Hypertension leads to a number of vessel wall abnormalities, including accelerated atherosclerosis in larger arteries, hyaline arteriolosclerosis in smaller arteries, and (in severe cases) proliferative changes and frank necrosis of arterioles. Arteriolar walls affected by hyaline change are presumably weaker than are normal vessels and are therefore vulnerable to rupture. In some instances chronic hypertension is associated with the development of minute aneurysms, termed *Charcot-Bouchard microaneurysms*, which may be the site of rupture. Charcot-Bouchard aneurysms, not to be confused with saccular aneurysms of larger intracranial vessels in the subarachnoid space, occur in vessels that are less than 300 μm in diameter, most commonly within the basal ganglia.

MORPHOLOGY

Hypertensive intraparenchymal hemorrhage may originate in the putamen (50% to 60% of cases), thalamus, pons, cerebellar hemispheres (rarely), and other regions of the brain (Fig. 28-18*A*). Acute hemorrhages, independent of etiology, are characterized by extravasation of blood with compression of the adjacent parenchyma. Old hemorrhages show an area of cavitary destruction of brain with a rim of brownish discoloration. The early lesions consist of a central core of clotted blood surrounded by a rim of brain tissue showing anoxic neuronal and glial changes as well as edema. Eventually the edema resolves, hemosiderin- and lipid-laden macrophages appear, and proliferation of reactive astrocytes is seen at the periphery of the lesion. The cellular events then follow the same time course that is observed after cerebral infarction.

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