Cerebral Small-Vessel Disease Diseases of the brain's small vessels **(Chap. 427)** can also cause symptomatic ischemic or hemorrhagic stroke but are more often clinically asymptomatic and recognized only during evaluation for cognitive decline or other symptoms. The two common age-related cerebral small-vessel pathologies are arteriolosclerosis and cerebral amyloid angiopathy. *Arteriolosclerosis* represents thickening of arterioles due to infiltration of plasma proteins into the vessel wall. The primary risk factors for this process are age, hypertension, and diabetes mellitus. Cerebrovascular arteriolosclerosis can present as a cause of ischemic or hemorrhagic symptomatic stroke, both most commonly centered in territories supplied by deep penetrating vessels such as thalamus, basal ganglia, or brainstem. *Cerebral amyloid angiopathy* is defined by deposition of the β-amyloid peptide in the walls of small cerebral arteries, arterioles, and capillaries, with consequent loss of normal wall structure. Its primary risk factor is advancing age. Cerebral amyloid angiopathy is most often recognized symptomatically as a cause of intracerebral hemorrhage **(Chap. 428)**, commonly located in cerebral cortex, subcortical white matter (collectively known as lobar hemorrhages), or the cerebral convexity subarachnoid space. The distinction between the deep penetrating territories most commonly affected by arteriolosclerosis and superficial lobar brain regions affected by cerebral amyloid angiopathy often allows the two small-vessel diseases to be radiographically distinguished.

Despite differences in their underlying pathogenic mechanisms, the two cerebral small-vessel diseases produce a similar range of ischemic and hemorrhagic brain lesions detectable by histopathology at autopsy or MRI scan during life **(Fig. 433-1)**. *Small (lacunar) infarcts* are a common feature of arteriolosclerosis and less commonly of cerebral amyloid angiopathy. Chronic lacunar infarcts can appear on MRI fluid-attenuated inversion recovery (FLAIR) sequences as a hyperintense rim surrounding a hypointense cavitated core with diameters typically 3–15 mm **(Fig. 433-1*A*)**, but this characteristic appearance evolves in only a subset of small infarctions, and many cannot be readily identified in the chronic stage. *Microinfarcts* <3 mm are characteristic of both small-vessel