

involved in small-vessel strokes. In the anterior circulation, small penetrating arteries called *lenticulostriates* arise from the proximal portion of the anterior and middle cerebral arteries and supply deep subcortical structures (**upper panels**). In the posterior circulation, similar arteries arise directly from the vertebral and basilar arteries to supply the brainstem (**lower panels**). Occlusion of a single penetrating artery gives rise to a discrete area of infarct (pathologically termed a “lacune,” or lake). Note that these vessels are too small to be visualized on CT angiography.

Clinical Manifestations The most common small-vessel stroke syndromes are the following: (1) *pure motor hemiparesis* from an infarct in the posterior limb of the internal capsule or the pons; the face, arm, and leg are almost always involved; (2) *pure sensory stroke* from an infarct in the ventral thalamus; (3) *ataxic hemiparesis* from an infarct in the ventral pons or internal capsule; (4) and *dysarthria and a clumsy hand* or arm due to infarction in the ventral pons or in the genu of the internal capsule.

Transient symptoms (small-vessel TIAs) may herald a small-vessel infarct; they may occur several times a day and last only a few minutes. Recovery from small-vessel strokes tends to be more rapid and complete than recovery from large-vessel strokes; in some cases, however, there is severe permanent disability.

A large-vessel source (either thrombosis or embolism) may manifest initially as a small-vessel infarction. Therefore, the search for embolic sources (carotid and heart) should not be completely abandoned in the evaluation of these patients. Secondary prevention of small-vessel stroke involves risk factor modification, specifically reduction in blood pressure (see “Treatment: Primary and Secondary Prevention of Stroke and TIA,” below).

■ LESS COMMON CAUSES OF STROKE

(Table 427-2) *Hypercoagulable disorders* (**Chap. 65**) primarily increase the risk of cortical vein or cerebral venous sinus thrombosis. Systemic lupus erythematosus with Libman-Sacks endocarditis can be a cause of embolic stroke. These conditions overlap with the antiphospholipid syndrome (**Chap. 357**), which probably requires long-term anticoagulation to prevent further stroke. Homocysteinemia may cause arterial thromboses as well; this disorder is caused by various mutations in the homocysteine