

# Lacunar stroke

Occlusions of the [lenticulostriate artery](#) or penetrating branches of the [Circle of Willis](#) or vertebral or basilar arteries are referred to as lacunar strokes. About 20% of all strokes are lacunar and have a high incidence in patients with chronic hypertension.

Small (3–7 mm) lacunes may be due to lipohyalinosis (vasculopathy due to HTN) of arteries <200 microns (may also be cause of many ICHs); this vasculopathy is indicative of small vessel disease, unlikely to be prevented by [carotid endarterectomy](#).

## Clinical features

The cells distal to the occlusion die, but since these areas are very small often only minor deficits are seen. When the infarction is critically located, however, more severe manifestations may develop, including paralysis and sensory loss.

Within a few months of the infarction, the necrotic brain cells are reabsorbed by macrophage activity, leaving a very small cavity referred to as a lake (or lacune in French).

Is one of a pair of blood vessels that supply oxygenated blood to the posterior aspect of the brain (occipital lobe) in human anatomy. It arises near the intersection of the posterior communicating artery and the basilar artery and connects with the ipsilateral middle cerebral artery (MCA) and internal carotid artery via the posterior communicating artery (PCommA).

Lacunar infarcts may have serious functional consequences if they involve motor or sensory fibers in the [internal capsule](#), but may be 'silent' if they involve other small regions of white matter or the basal ganglia.

## CT

Small infarcts in deep noncortical cerebrum or brainstem resulting from occlusion of penetrating branches of cerebral arteries. Size of infarcts ranges from 3–20 mm (CT detects larger ones; better sensitivity in white matter).

In the elderly, CT scanning shows signs of infarction in only approximately half of the most of the common form of lacunar stroke (pure motor stroke),

## MRI

MRI has increased the yield: the probability that CT or MRI will be positive is generally a function of the severity of the deficit [Mohr JP and Sacco RL, 1992].

1) Stroke/Brain Attack reporter's Handbook. Englewood, Colo: National Stroke Association, 1995

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