palsies. In some cases their deficits may be more extensive and mimic those of middle cerebral artery infarctions. Posterior cerebral artery infarcts lead to visual loss, often without any motor deficit. With involvement of the medial temporal lobes supplied by the PCAs, there may also be behavioral disturbances, including delirium and amnesia.

Brainstem infarcts cause specific syndromes due to the affected neural pathways and nuclei. Midbrain infarcts often produce vertical gaze deficits and impaired consciousness if the reticular activating system is involved.

Many cerebral infarctions do not cause weakness, such as fluent (or Wernicke's) aphasia, cortical visual loss, and Wallenberg syndrome. Because the inferior division of the MCA supplies the lateral temporal lobe and parietal lobes, including Wernicke's area, occlusion of that vessel may cause a prosodic, fluent speech with multiple paraphasic errors and poor comprehension, while sparing the motor strip in the frontal lobe. Emboli traveling up the basilar artery may cause significant infarction in the territory of both posterior cerebral arteries, causing complete blindness, sometimes without awareness of the deficit on the part of the patient, due to infarction of both occipital lobes (the "top of the basilar syndrome"). Behavioral abnormalities, memory loss, and eye movement abnormalities may also occur, due to the involvement of the medial temporal lobe structures and the midbrain eye movement centers. Small emboli to branches of the superior division of the MCA may cause focal weakness of the hand, particularly fine finger movements, simulating a peripheral compression neuropathy.

In patients presenting with dizziness, it is particularly difficult to distinguish stroke from vestibular neuronitis or Ménière's disease (see Chapter 113). The presence of a normal head-thrust test, skew deviation, or direction-changing nystagmus are all signs of stroke, rather than a peripheral cause. Patients should be followed until they can walk without imbalance; patients with nausea and vomiting due to cerebellar infarction may develop fatal brainstem compression due to swelling.

The signs and symptoms of *subarachnoid hemorrhage* differ from other stroke types due to the absence of focal deficits. Instead, patients present with abrupt onset of severe headache (i.e., "the worst headache of my life"), vomiting, altered consciousness, and sometimes coma, typically without localizing signs.

*Thrombosis of cerebral veins* or the larger draining dural sinuses present with a combination of headache due to elevated intracranial pressure, seizures, and focal deficits due to hemorrhage. Rarely the syndrome of *thunderclap headache*, or sudden severe headache without any focal signs similar to that occurring in SAH, may be due to venous thrombosis. Occlusion of the cerebral venous sinuses may occur in association with a hyperviscosity or hypercoagulable state, including pregnancy or hormonal contraceptive use. Imaging findings include bilateral hemorrhagic infarctions in a parasagittal distribution and extensive white matter edema. Contrast-enhanced CT may demonstrate the *empty delta* sign, indicating a filling defect in the sagittal sinus. Magnetic resonance venography (MRV) and T1 weighted MRI images confirm the presence of thrombus; cerebral angiography is seldom needed to confirm the diagnosis.

## DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The benefit of thrombolytic therapy within 3 hours of onset of acute ischemic stroke requires urgent differentiation of ischemic stroke from hemorrhage and other causes of sudden neurological symptoms. Headache, vomiting, seizures, and coma, are more common in hemorrhagic stroke, though these are never reliable enough to preclude imaging. The distinction is straightforward in most cases once a head CT is performed. The hyperdense signal of blood in the parenchyma on CT almost invariably distinguishes hemorrhage from ischemia. In exceptional cases the typical hyperdensity of ICH is absent owing to severe anemia or to its subacute state, during which blood may be indistinguishable from brain tissue. Certain imaging findings on initial CT further support a presumed diagnosis of infarction, such as a hyperdense vessel sign indicative of thrombus in the vessel, or loss of the gray-white junction and sulci in the cortex, and loss of the demarcation of the insular cortex and deep gray nuclei, both of which are early indicators of ischemia and edema (Fig. 116-4). CT angiography often identifies the site of vascular occlusion.

Imaging in the setting of suspected acute ischemia does not definitively diagnose ischemia, but rather excludes hemorrhage; if clinical symptoms are consistent with cerebral ischemia, then thrombolytic treatment is indicated. Primary stroke centers must perform and interpret CT scans within 30 minutes of the arrival of a patient with suspected stroke. MRI can also effectively exclude acute hemorrhage, and diffusion-weighted imaging sequences are more sensitive to the earliest changes of ischemia (Fig. 116-5), but the speed and availability of CT make it the initial imaging modality of choice at most centers. MRI scanning may then be used to provide additional information. Specific MRI sequences have greater sensitivity to blood than CT, and many identify hemorrhagic infarction missed by CT.

Clinical features at stroke onset may suggest a subtype of cerebral infarction but require confirmatory laboratory data. Cerebral embolism is suggested by sudden onset and a syndrome of circumscribed focal signs attributable to cerebral surface infarction, such as pure aphasia or pure hemianopia. Unless the source of embolization is obvious on hospital admission, blood cultures, electrocardiographic monitoring, and echocardiography are indicated.

A diagnosis of atherosclerotic infarction is suggested if there were previous TIAs, particularly when the symptoms are stereotypical. Doppler ultrasonography or MRA can usually identify the stenosis. In equivocal cases, CT angiography or conventional angiography may be needed. Small penetrating vessel infarcts, *lacunar infarcts*, usually spare cortical functions, such as language and cognition, but cause loss of elementary neurologic function, such as strength, sensation, and coordination. Up to 25% of patients with lacunar infarcts have large-vessel disease or a cardioembolic source, so it is important to carry out a complete etiologic evaluation in all stroke patients.

Up to 50% of patients with transient deficits lasting less than 24 hours have evidence of infarction on imaging, and the risk of stroke and other vascular events is as high after TIA as after completed stroke. In the acute setting, when decisions about thrombolysis must be made, it is virtually impossible to know which