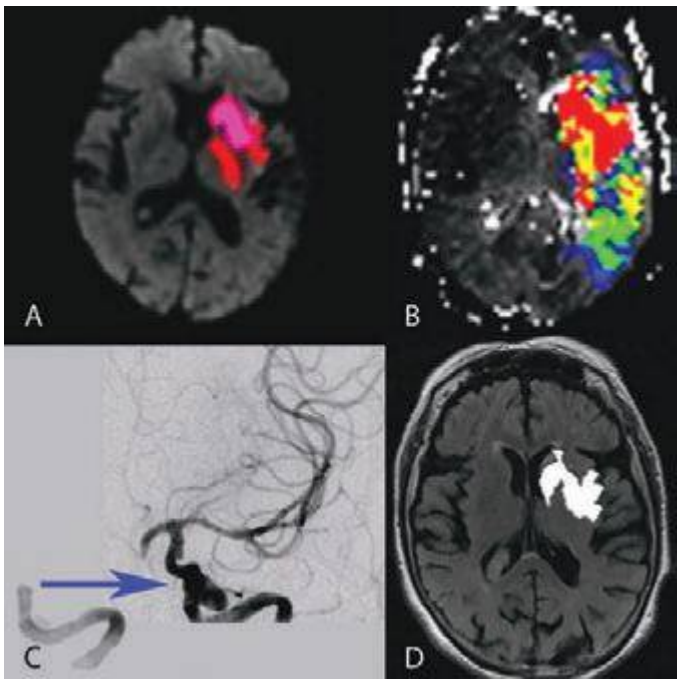

FIGURE 446-16 Magnetic resonance imaging (MRI) of acute stroke. ***A.*** MRI diffusion-weighted image (DWI) of an 82-year-old woman 2.5 h after onset of right-sided weakness and aphasia reveals restricted diffusion within the left basal ganglia and internal capsule (*colored regions*). ***B.*** Perfusion defect within the left hemisphere (*colored signal*) imaged after administration of an IV bolus of gadolinium contrast. The discrepancy between the region of poor perfusion shown in ***B*** and the diffusion deficit shown in ***A*** is called *diffusion-perfusion mismatch* and provides an estimate of the ischemic penumbra. Without specific therapy, the region of infarction will expand into much or all of the perfusion deficit. ***C.*** Cerebral angiogram of the left internal carotid artery in this patient before (*left*) and after (*right*) successful endovascular embolectomy. The occlusion is within the carotid terminus. ***D.*** Fluid-attenuated inversion recovery image obtained 3 days later showing a region of infarction (*coded as white*) that corresponds to the initial DWI image in ***A***, but not the entire area at risk shown in ***B***, suggesting that successful embolectomy saved a large region of brain tissue from infarction. *(Courtesy of Gregory Albers, MD, Stanford University; with permission.)*

principally produced by trauma. SAH results from trauma or the rupture of an intracranial aneurysm or arteriovenous malformation (AVM) (Chap. 330). Intracerebral and intraventricular hemorrhage will be considered here.

DIAGNOSIS

Intracranial hemorrhage is often discovered on noncontrast CT imaging of the brain during the acute evaluation of stroke. Because CT is more widely available and may be logistically easier, CT imaging is the preferred method for acute stroke evaluation (Fig. 446-1). The location of the hemorrhage narrows the differential diagnosis to a few entities. Table 446-6 lists the causes and anatomic spaces involved in hemorrhages.

EMERGENCY MANAGEMENT

Close attention should be paid to airway management because a reduction in the level of consciousness is common and often progressive. The initial blood pressure should be maintained until the results of the CT scan are reviewed and demonstrate an intracerebral hemorrhage (ICH). In theory, a higher blood pressure should promote hematoma expansion, but it remains unclear if lowering of blood pressure reduces hematoma growth. Recent clinical trials have shown that systolic blood pressure (SBP) can be safely lowered acutely and rapidly to <140 mmHg in patients with spontaneous ICH whose initial SBP was 150–220 mmHg. The INTERACT2 trial is the only large phase 3 clinical trial to address the effect of acute blood pressure lowering on ICH functional outcome. INTERACT2 randomized patients with spontaneous ICH within 6 h of onset and a baseline SBP of 150–220 mmHg to two different SBP targets (<140 mmHg and <180 mmHg). In those with the target SBP <140 mmHg, 52% had an outcome of death or major disability at 90 days compared with 55.6% of those with a target SBP <180 mmHg (p = .06). There was a significant shift to improved outcomes in the lower blood pressure arm, whereas both groups had a similar mortality. This study shows that it is not harmful, and may be modestly beneficial, to lower blood pressure in acute ICH. Thus, it is reasonable to target an SBP <140 mmHg initially in this group of patients. In patients who have higher SBP on presentation or who are deeply comatose with possible elevated ICP, it is unclear whether the INTERACT2 results apply. In patients who have ICP monitors in place, current recommendations are to maintain the cerebral perfusion pressure (mean arterial pressure [MAP] minus ICP) above 60 mmHg. Blood pressure should be lowered with nonvasodilating IV drugs such as nicardipine, labetalol, or esmolol. Patients with cerebellar hemorrhages or with depressed mental status and radiographic evidence of hydrocephalus should undergo urgent neurosurgical evaluation; these patients require close monitoring because they can deteriorate rapidly. Based on the clinical examination and CT findings, further imaging studies may be necessary, including MRI or conventional x-ray angiography. Stuporous or comatose patients with clinical and imaging signs of herniation are generally treated presumptively for elevated ICP, with tracheal intubation, administration of osmotic diuretics such as mannitol or hypertonic saline, and elevation of the head of the bed while surgical consultation is obtained (Chap. 330). Reversal of coagulopathy and consideration of surgical evacuation of the hematoma (detailed below) are two other principal aspects of initial emergency management.

INTRACEREBRAL HEMORRHAGE

ICH accounts for ~10% of all strokes, and about 35–45% of patients die within the first month. Incidence rates are particularly high in Asians and blacks. Hypertension, coagulopathy, sympathomimetic drugs (cocaine, methamphetamine), and cerebral amyloid angiopathy cause the majority of these hemorrhages. Advanced age and heavy alcohol consumption increase the risk, and cocaine and methamphetamine use is one of the most important causes in the young.

Hypertensive Intracerebral Hemorrhage • ***PATHOPHYSIOLOGY*** Hypertensive ICH usually results from spontaneous rupture of a small penetrating artery deep in the brain. The most common sites are the basal ganglia (especially the putamen), thalamus, cerebellum, and pons. The small arteries in these areas seem most prone to hypertension-induced vascular injury. When hemorrhages occur in other brain areas or in nonhypertensive patients, greater consideration should be given to other causes such as hemorrhagic disorders, neoplasms, vascular malformations, and cerebral amyloid angiopathy. The hemorrhage may be small, or a large clot may form and compress adjacent tissue, causing herniation and death. Blood may also dissect into the ventricular space, which substantially increases morbidity and may cause hydrocephalus.

Most hypertensive ICHs initially develop over 30–90 min, whereas those associated with anticoagulant therapy may evolve for as long as 24–48 h. However, it is now recognized that about a third of patients even with no coagulopathy may have significant hematoma expansion with the first day. Within 48 h, macrophages begin to phagocytize the hemorrhage at its outer surface. After 1–6 months, the hemorrhage is generally resolved to a slitlike orange cavity lined with glial scar and hemosiderin-laden macrophages.

CLINICAL MANIFESTATIONS ICH generally presents as the abrupt onset of a focal neurologic deficit. Seizures are uncommon. Although clinical symptoms may be maximal at onset, commonly the focal deficit worsens over 30–90 min and is associated with a diminishing level of consciousness and signs of increased ICP such as headache and vomiting.

The putamen is the most common site for hypertensive hemorrhage, and the adjacent internal capsule is usually damaged (Fig. 446-17). Contralateral hemiparesis is therefore the sentinel sign.