

Intracranial Hemorrhage

Hemorrhages may occur at any site within the cranium—outside the brain or within it (intraparenchymal).

Hemorrhages in the epidural or subdural space are typically associated with trauma and were discussed earlier; hemorrhages within the brain parenchyma and in the subarachnoid space, in contrast, are more often a manifestation of underlying cerebrovascular disease and are discussed in the following sections

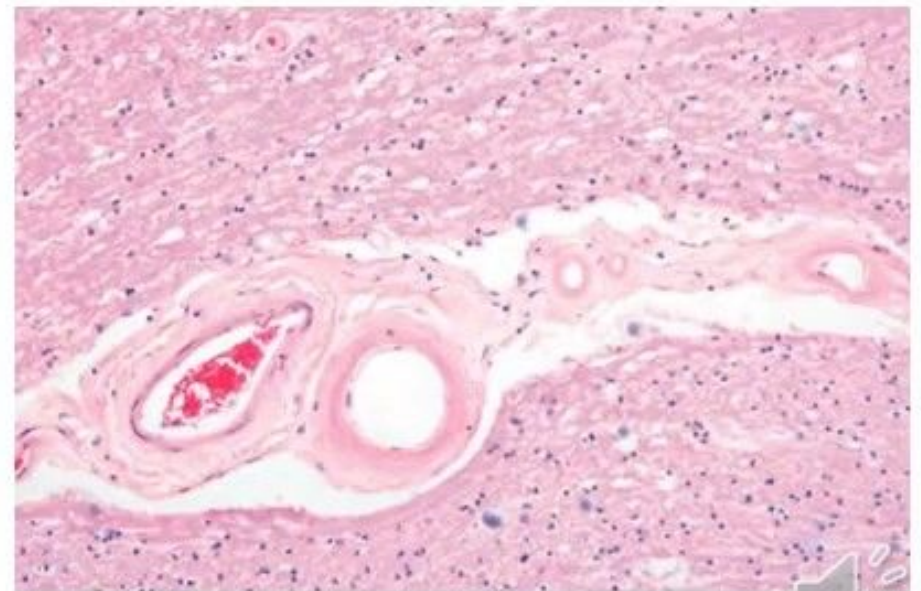
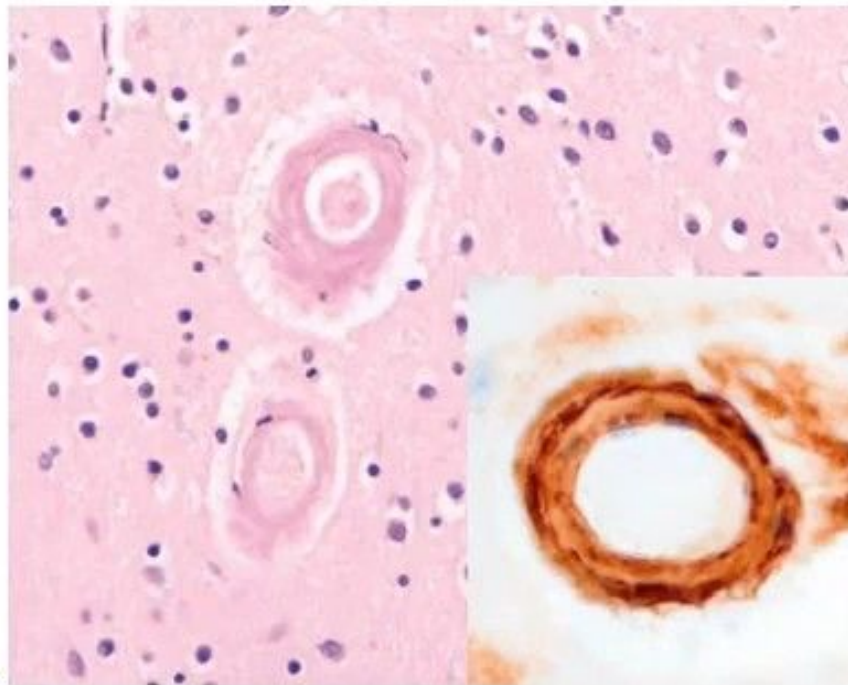
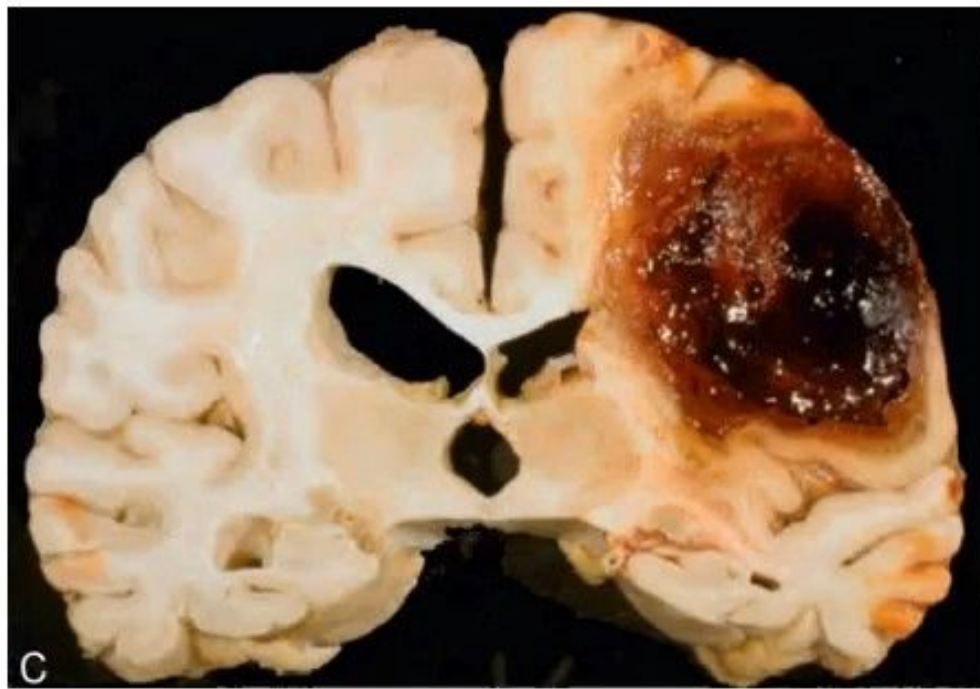


Intraparenchymal Hemorrhage

Rupture of a small intraparenchymal vessel can result in a primary hemorrhage within the brain, often associated with sudden onset of neurologic symptoms (stroke); this should not be confused with the secondary hemorrhagic transformation of an occlusive infarct (described earlier).

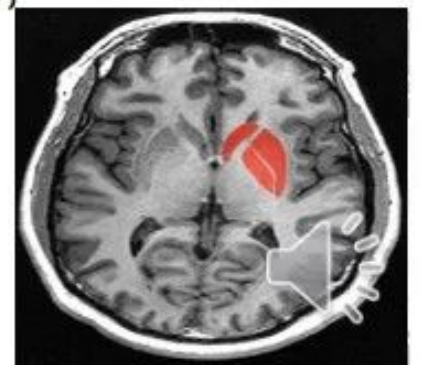
Spontaneous (nontraumatic) intraparenchymal hemorrhages occur most commonly in middle to late adult life, with a peak incidence at about 60 years of age. Hemorrhages in the basal ganglia and thalamus are commonly designated “ganglionic hemorrhages,” whereas those that occur in the lobes of the cerebral hemispheres are called “lobar hemorrhages”; the two major causes of these patterns of hemorrhage are hypertension and cerebral amyloid angiopathy, respectively. In addition, other local and systemic factors may cause or contribute to nontraumatic hemorrhage, including systemic coagulation disorders, neoplasms, vasculitis, aneurysms, and vascular malformations.





Hypertension is the risk factor most commonly associated with deep brain parenchymal hemorrhages, accounting for more than 50% of clinically significant hemorrhages and for roughly 15% of deaths among individuals with chronic hypertension. Hypertensive intraparenchymal hemorrhage may originate in the putamen (50% to 60% of cases), thalamus, pons, cerebellar hemispheres (rarely), and other regions of the brain . Hypertension leads to a number of vessel wall abnormalities, including accelerated atherosclerosis in larger arteries, hyaline arteriolosclerosis in smaller arteries, and (in severe cases) proliferative changes and frank necrosis of arterioles.

As described earlier, if small arteries affected by hyaline arteriolosclerosis do not rupture but are occluded, the result is lacunar infarction



Cerebral amyloid angiopathy (CAA) is the risk factor most commonly associated with lobar hemorrhages

MORPHOLOGY Acute primary intraparenchymal hemorrhages are characterized by a central core of clotted blood that compresses the adjacent parenchyma; this compression leads to secondary infarction of the affected brain tissue, with anoxic neuronal and glial changes as well as edema. Eventually the edema resolves, hemosiderin- and lipid-laden macrophages appear, and proliferation of reactive astrocytes is seen at the periphery of the lesion; the cellular events then follow the same time course that is observed after cerebral infarction. Old hemorrhages show areas of parenchymal cavitory destruction with a rim of brownish discoloration.



Clinical Features Intracerebral hemorrhage can be clinically devastating if it involves a large part of the brain or extends into the ventricular system. When hemorrhage affects smaller regions, it is either clinically silent or evolves like an infarct; over weeks or months, there is a gradual removal of the hematoma, sometimes with considerable clinical improvement. Again, the location of the hemorrhage determines the clinical manifestations.



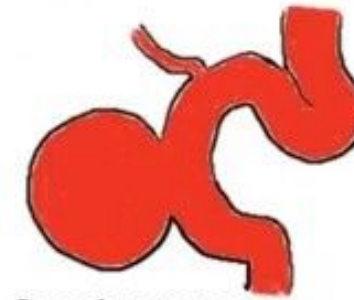
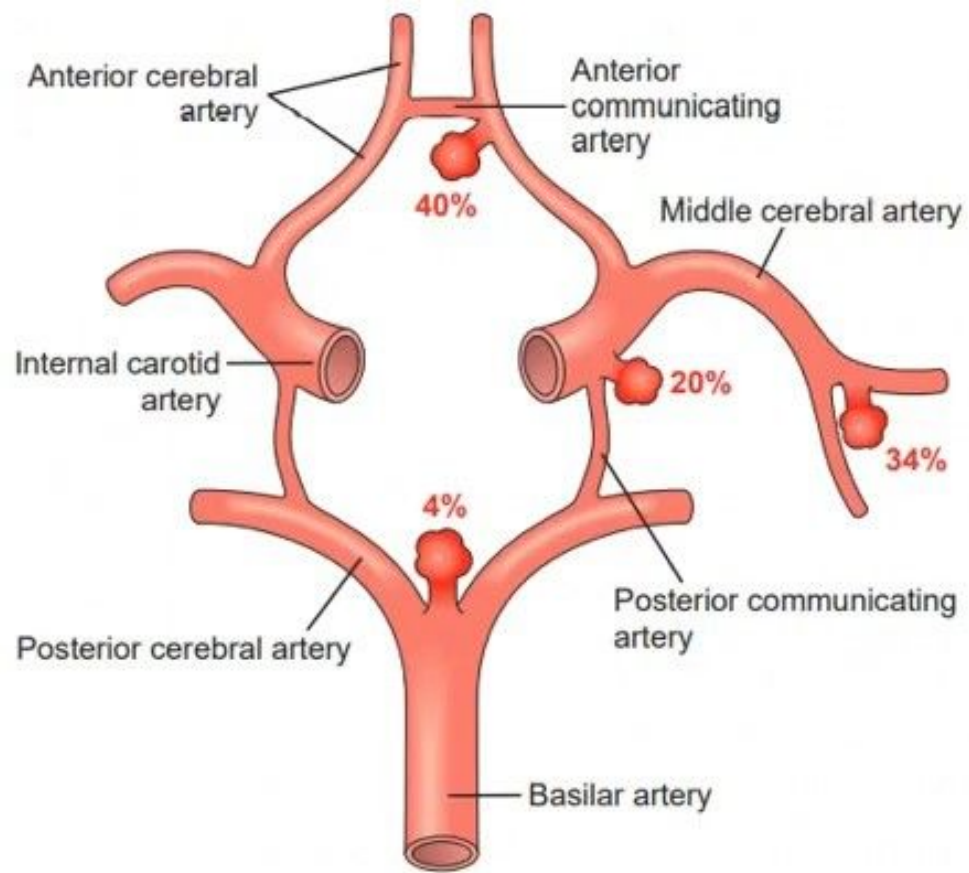
Subarachnoid Hemorrhage and Ruptured Saccular Aneurysm
The most frequent cause of spontaneous subarachnoid hemorrhage is rupture of a saccular (“berry”) aneurysm in a cerebral artery. (As noted earlier, brain trauma is the most common cause of subarachnoid hemorrhage overall.) Nontraumatic subarachnoid hemorrhage may also result from rupture of a primary intracerebral hemorrhage into the ventricular system, vascular malformation, hematologic disturbances, and tumors.



Saccular aneurysm is the most common type of intracranial aneurysm; other aneurysm types include atherosclerotic (fusiform; mostly of the basilar artery), mycotic, traumatic, and dissecting. These latter three, like saccular aneurysms, are most often found in the anterior circulation, but more often cause cerebral infarction rather than subarachnoid hemorrhage.

Saccular aneurysms are found in about 2% of the population according to recent data from community-based radiologic studies. About 90% of saccular aneurysms are found near major arterial branch points in the anterior circulation; multiple aneurysms exist in 20% to 30% of cases based on autopsy series.

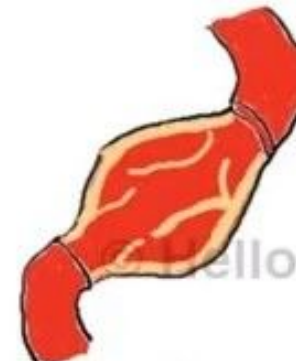




Saccular aneurysm



Atherosclerotic aneurysm



Mycotic aneurysm



Traumatic aneurysm

Pathogenesis

Although the etiology of saccular aneurysms remains obscure, the structural abnormality of the involved vessel (absence of smooth muscle and intimal elastic lamina) suggests that they are developmental anomalies. The majority occur sporadically, but genetic factors may be important in their pathogenesis because there is an increased incidence of aneurysms in first-degree relatives of those affected. Although they are sometimes referred to as “congenital,” the aneurysms are not present at birth but develop over time because of an underlying defect in the media of the vessel wall.



MORPHOLOGY

An unruptured saccular aneurysm is a thin-walled outpouching, usually at an arterial branch point along the circle of Willis or a major vessel just beyond. Saccular aneurysms measure from a few millimeters to 2 or 3 cm in diameter and have a bright red, shiny surface and a thin, translucent wall. Atheromatous plaques, calcification, or thrombi may be found in the wall or lumen of the aneurysm.



Vascular Malformations

Vascular malformations of the brain are classified into four principal groups: arteriovenous malformations, cavernous malformations, capillary telangiectasias, and venous angiomas. Of these, the first two are the types associated with risk of hemorrhage and development of neurologic symptoms. Although lacking morphologic features that typify neoplasms, arteriovenous malformations are frequently associated with activating somatic mutations in the KRAS oncogene within the endothelial cells that line the malformed vessels, suggesting that dysregulated RAS signaling has a central role in their pathogenesis.



MORPHOLOGY

Arteriovenous malformations (tangled networks of wormlike vascular channels with high blood flow due to prominent, pulsatile arteriovenous shunting) may involve vessels in the subarachnoid space, the brain, or both . They are composed of greatly enlarged blood vessels separated by gliotic tissue, often with evidence of prior hemorrhage.

Some vessels can be recognized as arteries with duplication and fragmentation of the internal elastic lamina, while others show marked thickening or partial replacement of the media by hyalinized connective tissue.



Cavernous malformations consist of distended, loosely organized vascular channels arranged back to back with collagenized walls of variable thickness; there is usually no brain parenchyma between vessels in this type of malformation. They occur most often in the cerebellum, pons, and subcortical regions. Foci of old hemorrhage, infarction, and calcification frequently surround the abnormal vessels.



Clinical Features

Arteriovenous malformations are the most common clinically significant vascular malformation. Males are affected twice as frequently as females. The lesion often presents between 10 and 30 years of age as a seizure disorder, an intracerebral hemorrhage, or a subarachnoid hemorrhage. The most common site is the territory of the middle cerebral artery, particularly its posterior branches



Vascular Dementia

Individuals who, over the course of many months and years, suffer multiple, bilateral, gray matter (cortex, thalamus, basal ganglia) and white matter . infarcts may develop a distinctive clinical syndrome characterized by dementia, gait abnormalities, and pseudobulbar signs, often with superimposed focal neurologic deficits. The syndrome, generally referred to as vascular dementia, is caused by multifocal vascular disease of several types, including (1) cerebral atherosclerosis, (2) vessel thrombosis or embolization from carotid vessels or from the heart, and (3) cerebral arteriolosclerosis from chronic hypertension

