infarction of the lateral medulla (Wallenberg syndrome), characterized by vertigo, imbalance, Horner syndrome, dysphagia, and sensory loss.

After originating as the union of the right and left vertebral arteries, the basilar artery travels up the ventral pons. Paramedian and circumferential penetrating arteries exit the basilar to dive into the pontine parenchyma. Proximally, the basilar gives off the paired anterior inferior cerebellar arteries (AICA), and more distally the superior cerebellar arteries (SCA); these perfuse the ventrolateral aspect of the cerebellar cortex. An internal auditory (labyrinthine) artery arises either directly from the basilar or from the anterior cerebellar artery to supply the cochlea, labyrinth, and part of the facial nerve. Ischemia in the basilar territory may, therefore, cause hearing loss and vertigo, sometimes as an isolated symptom.

The basilar artery terminates in the right and left posterior cerebral arteries (PCAs). A series of penetrators arise from the posterior communicating and posterior cerebral arteries to supply the hypothalamus, dorsolateral midbrain, lateral geniculate, and thalamus. The posterior cerebral artery supplies the inferior temporal lobe, and the medial and inferior surfaces of the occipital lobe. In some patients a single large penetrating vessel at the midline of the terminal basilar artery may supply medial aspects of both thalami (the artery of Percheron); emboli occluding this vessel may, therefore, cause bilateral thalamic infarcts, with a decrease in alertness and vertical gaze abnormalities, without significant motor deficit.

The brain's anastomotic network includes not only the connections through the Circle of Willis, but also intercommunicating systems extracranially and more distal connections intracranially through meningeal anastomoses that cover the cortical and cerebellar surfaces (pial-pial collaterals). These networks all protect the brain from ischemia by providing alternate routes to circumvent obstructions in the main arteries.

Venous anatomy is more variable than arterial. Superficial veins drain into the transverse, superior sagittal, and cavernous sinuses. The deep venous drainage is via the great vein of Galen, which drains into the straight sinus, and in turn drains into the torcula along with the sagittal sinus. Blood drains from the torcula to the transverse sinus, then to the sigmoid sinus, and thereafter the jugular vein. Anterior venous drainage is via the cavernous sinus, which communicates with the contralateral cavernous sinus, the transverse sinus via the superior petrosal sinus, and the inferior petrosal sinus, which drains directly into the jugular bulb.

Vascular Pathogenesis

There are multiple mechanisms leading to brain ischemia. Hemodynamic infarction occurs as a result of reduced perfusion, usually in the setting of arterial stenosis due to atherosclerosis. In some cases, stenosis may be due to arterial dissection, vasculitis, fibromuscular dysplasia, or other arteriopathies. Embolism occurs when a thrombus originating from a more proximal source (e.g., arterial or cardiac) travels through the arteries and occludes a cerebral artery. Paradoxical embolism occurs when a thrombus crosses from the venous circulation to the left side of the heart through a patent foramen ovale or, less commonly, an intrapulmonary arteriovenous shunt. Other particles that may embolize include neoplasm, fat, air, or other foreign substances. Air emboli can follow injuries or procedures involving the lungs, the dural sinuses, or jugular veins. Fat embolism usually results from a bone fracture. Septic emboli arise from bacterial endocarditis.

Intracranial hemorrhage results from the rupture of a vessel anywhere within the cranial cavity. Intracranial hemorrhages may be classified by location (e.g., extradural, subdural, subarachnoid, intracerebral, intraventricular), by the nature of the ruptured vessel (e.g., arterial, capillary, venous), or by cause (e.g., primary, secondary). Trauma is often involved in the generation of extradural hematoma from laceration of the middle meningeal artery or vein, and subdural hematomas from traumatic rupture of veins that traverse the subdural space.

Intracerebral hemorrhage is characterized by bleeding into the substance of the brain, usually originating from a small penetrating artery. Hypertension has been implicated as the cause of weakening in the walls of arterioles and the formation of microaneurysms (i.e., Charcot-Bouchard aneurysms). The most common sites for hypertensive arterial hemorrhage are the putamen, pons, cerebellum, and thalamus. Blood under arterial pressures destroys or displaces brain tissue. Amyloid angiopathy, due to the vascular deposition of β-amyloid protein similar to that seen in Alzheimer disease has been implicated as an important cause of lobar hemorrhage in elderly patients. Other causes of hemorrhage include arteriovenous malformations, aneurysms, moyamoya disease, bleeding disorders or anticoagulation, trauma, tumors, cavernous angiomas, and illicit drug abuse.

Subarachnoid hemorrhage occurs when blood is localized to the surrounding membranes and cerebrospinal fluid. It is most frequently caused by leakage of blood from a cerebral aneurysm. The combination of congenital and acquired factors leads to a degeneration of the arterial wall and the release of blood, under arterial pressures, into the subarachnoid space and cerebrospinal fluid. Aneurysms may be distributed at different sites throughout the base of the brain, particularly at the origin or bifurcations of arteries of the circle of Willis. Other secondary causes that may lead to SAH include trauma, arteriovenous malformations, bleeding disorders or anticoagulation, amyloid angiopathy, or cerebral sinus thrombosis.

The most common intrinsic disorder of the cerebral blood vessels is atherosclerosis, which shares similarities in pathology with atherosclerosis throughout the body. Arteriosclerotic plaques may develop at any point along the carotid artery and the vertebrobasilar system, but the most common sites are the bifurcation of the common carotid artery, the origins of the MCAs and ACAs, and the origins of the vertebral from the subclavian arteries (Fig. 116-2). In the past it was thought that intracranial atherosclerotic disease required significant stenosis (>50%) to cause symptoms. However, recent pathological and radiological studies provide evidence that substenotic lesions can also cause strokes due to plaque rupture and acute thrombosis, as is the case elsewhere in the body.

Small-vessel disease refers to the occlusion of a penetrant branch of a larger artery, usually due to microatheroma or to lipohyalinosis, a degenerative disorder of the vessel characterized by deposition of fatty and proteinaceous material. Hematological disorders and coagulopathies, including leukemia, Waldenstrom macroglobulinemia, polycythemia, primary and secondary