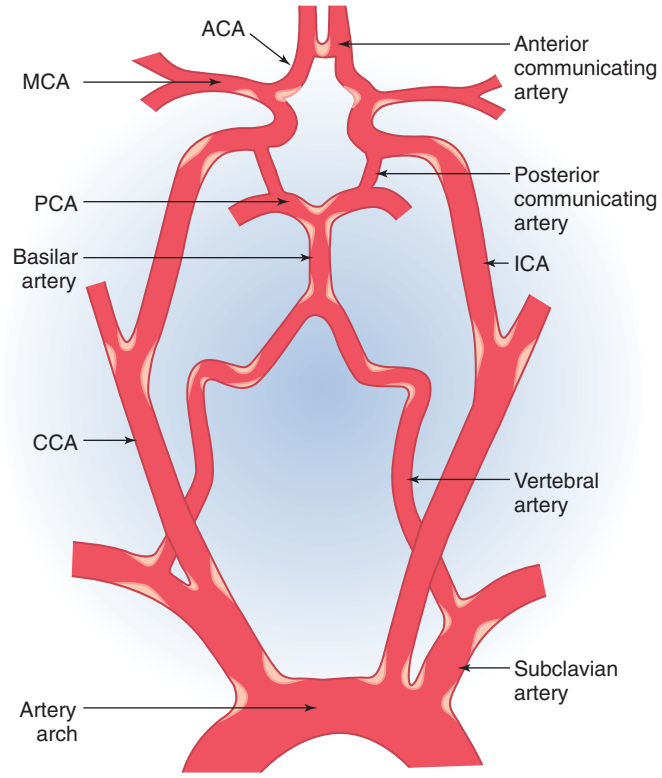

FIGURE 116-2 Sites of predilection for atheromatous plaque. ACA, Anterior cerebral artery; CCA, common carotid artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery. (From Caplan LR: Stroke: a clinical approach, ed 2, Boston, 1993 Butterworth-Heinemann.)

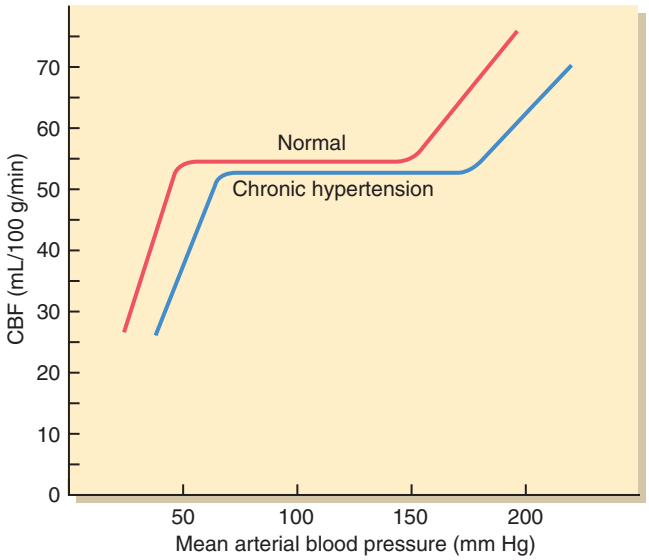

FIGURE 116-3 Autoregulatory cerebral blood flow (CBF) response to changes in mean arterial pressure in normotensive and chronically hypertensive persons. Note the shift of the curve toward higher mean pressures with chronic hypertension. (From Pulsinelli WA: Cerebrovascular diseases-principles. In Goldman L, Bennett JC, editors: Cecil textbook of medicine, ed 21, Philadelphia, 2000, Saunders, p 2097.)

antiphospholipid antibody syndrome, and genetic defects of the coagulation cascade, may also lead to occlusive thrombi and emboli.

The cerebral circulation differs from the systemic circulation. The brain is protected by the anastomoses described above. In addition, *cerebral autoregulation* maintains a constant cerebral perfusion pressure over a range of systemic blood pressures (Fig. 116-3). Cerebral arterioles have a well-developed muscular coat that allows constriction in response to increased blood pressure, and dilation with hypotension. The arterioles are also exquisitely sensitive to changes in peripheral arterial concentrations of carbon dioxide ($PaCO_2$) and oxygen (PaO_2). When the partial pressure of CO_2 decreases, such as after hyperventilation, the arterioles constrict and blood flow is reduced. In healthy individuals, cerebral autoregulation maintains a constant cerebral blood flow over mean arterial pressures of 60 to 140 mm Hg. In patients with chronic hypertension, the autoregulatory curve is shifted to the right, so that even minor reductions in blood pressure levels may not be tolerated. At blood pressures above these limits, moreover, as in severe hypertension, autoregulatory capacity may be overwhelmed, leading to breakthrough edema and hemorrhage. In the setting of infarction or hemorrhage, cerebral autoregulation is also impaired, resulting in cerebral dependence on systemic blood pressure to maintain adequate perfusion. Thus, decreasing the blood pressure in the setting of acute ischemia is hazardous.

Specific disorders may originate from autoregulatory dysfunction: posterior reversible encephalopathy syndrome (PRES) and the reversible cerebral vasoconstriction syndrome (RCVS). In posterior reversible encephalopathy syndrome, there is loss of autoregulatory control with leakage of fluid across the blood-brain barrier, primarily in the posterior regions of the brain. Patients present with elevated blood pressures, headaches, seizures, and loss of visual function. Reversible cerebral vasoconstriction syndrome, a recently recognized syndrome, remains incompletely characterized, and shares features with posterior reversible encephalopathy syndrome. The two disorders overlap in 10% or more of cases. Patients with reversible cerebral vasoconstriction syndrome are typically young women who present with acute, severe headache, have minimal or no neurological deficits, and may have evidence of non-aneurysmal, superficial SAH as well as vasospasm of the cerebral arteries. Sympathetic innervation of the vessels is also less in the posterior circulation than anteriorly, leading to a reduced ability of the posterior circulation to adapt to changes in blood pressure, and may contribute to the propensity for edema to form in the occipital lobes during hypertensive crises.

In addition, focal cerebral activity, such as occurs when activating brain regions responsible for moving a limb, is accompanied by accelerated metabolism in the appropriate region, and is accommodated by slight increases in local blood flow and oxygen delivery. Exploitation of this increased local energy demand and delivery is what allows imaging of functional brain activity using MRI, which can detect subtle changes in regional cerebral blood flow.

Intracerebral capillaries also lack adventitia, with astrocytes serving as the vascular component of the neurovascular unit. Tight junctions at the capillary level play an important role in the blood-brain barrier, which limits permeability between the vascular compartment and the brain tissue.