portion of the media of large arteries with oxygen and nutrients.

As already alluded to, arteries are divided into three types based on their size and structural features: (1) large or *elastic arteries*, including the aorta, the major branches of the aorta (the innominate, subclavian, common carotid, and iliac arteries), and the pulmonary arteries; (2) medium-sized or *muscular arteries*, comprising smaller branches of the aorta (e.g., the coronary and renal arteries); and (3) small arteries (≤2 mm in diameter) and *arterioles* (20 to 100 μm in diameter), within tissues and organs.

Capillaries are approximately the diameter of a red cell (7 to 8 μm); they have an endothelial cell lining but no media, although variable numbers of *pericytes*, cells that resemble smooth muscle cells, typically lie just deep to the the endothelium. Collectively, capillaries have a huge cross-sectional area, and also have a relative low flow rate. The combination of thin walls and slow flow make capillaries ideally suited for the exchange of diffusible substances between blood and tissues. Because functionally useful oxygen diffusion in tissues is limited to a distance of only approximately 100 μm, the capillary network of most tissues is very rich. Tissues with high metabolic rates, such as myocardium and brain, have the highest density of capillaries.

Blood from capillary beds flows into postcapillary venules and then sequentially through collecting venules and small, medium, and large veins. In most inflammatory reactions, vascular leakage and leukocyte exudation occur preferentially from postcapillary venules (Chapter 3).

Relative to arteries at the same level of branching, veins have larger diameters, larger lumens, and thinner and less organized walls (Fig. 11-1). These structural features augment the capacity of the venous side of the circulation, which on average contains about two-thirds of total blood volume. Less rigid walls means that *veins are subject to dilation and compression, as well as infiltration by tumors and inflammatory processes*. Reverse flow (due to gravity) is prevented in the extremities by venous valves.

Lymphatics are thin-walled channels lined by specialized endothelium; they provide conduits to return interstitial tissue fluid and inflammatory cells to the bloodstream. *Lymphatics can also transport microbes and tumor cells, thus constituting an important potential pathway for disease dissemination*.

KEY CONCEPTS

Vascular Structure and Function

- All vessels except capillaries share a three-layered architecture consisting of an endothelium lined intima, a surrounding smooth muscle media, and supportive adventitia, admixed with extracellular matrix.
- The smooth muscle cell and matrix content of arteries, veins, and capillaries vary according to hemodynamic demands (e.g., pressure, pulsatility) and functional requirements.
- The specific composition of the vessel wall at any given site within the vascular tree influences the nature and consequences of pathologic injuries.

Vascular Anomalies

Although rarely symptomatic, anatomic variants of the usual vascular supply are important for treating physicians to recognize, as the failure to do so may lead to surgical complications and impede attempted therapeutic interventions (e.g., placement of coronary artery stents). Among congenital vascular anomalies, three are of particular medical significance:

- *Developmental* or *berry aneurysms* occur in cerebral vessels; when ruptured, these can be causes of fatal intracerebral hemorrhage (Chapter 28).
- *Arteriovenous fistulas* are direct connections (usually small) between arteries and veins that bypass the intervening capillary bed. They occur most commonly as developmental defects but can also result from rupture of an arterial aneurysm into the adjacent vein, from penetrating injuries that pierce arteries and veins, or from inflammatory necrosis of adjacent vessels; surgically generated arteriovenous fistulas provide vascular access for chronic hemodialysis. Like berry aneurysms, arteriovenous fistulas may rupture, leading to intracerebral hemorrhage. Large or multiple arteriovenous fistulas can produce clinically significant effects by shunting blood from the arterial to the venous circulations, forcing the heart to pump additional volume and leading to high-output cardiac failure.
- *Fibromuscular dysplasia* is a focal irregular thickening in medium and large muscular arteries, including renal, carotid, splanchnic, and vertebral vessels. The cause is unknown, but is probably developmental; first-degree relatives of affected individuals have an increased incidence. Segments of the vessel wall are focally thickened by a combination of medial and intimal hyperplasia and fibrosis, resulting in luminal stenosis; in the renal arteries, this can be a cause of renovascular hypertension (Chapter 20). Immediately adjacent vessel segments can have markedly attenuated media (on angiography, the vessels are said to have a "string of beads" appearance), leading to vascular outpouchings (*aneurysms*) that can rupture. Fibromuscular dysplasia can manifest at any age; although it is seen most frequently in young women, there is no association with oral contraceptives or increased estrogen expression.

Vascular Wall Response to Injury

The integrated functioning of endothelial cells and smooth muscle cells impacts vessel development as well as the physiologic and pathophysiologic responses to hemodynamic and biochemical stimuli. Their function (and dysfunction) are described briefly, followed by discussion of specific vascular disorders.

Endothelial cells form a specialized lining for blood vessels. Although endothelial cells throughout the vascular tree share many attributes, populations that line different portions of the vascular tree (large vessels vs. capillaries, arteries vs. veins) have distinct gene expression profiles, behaviors, and morphologic appearances. Thus, endothelial cells in liver sinusoids or in renal glomeruli are