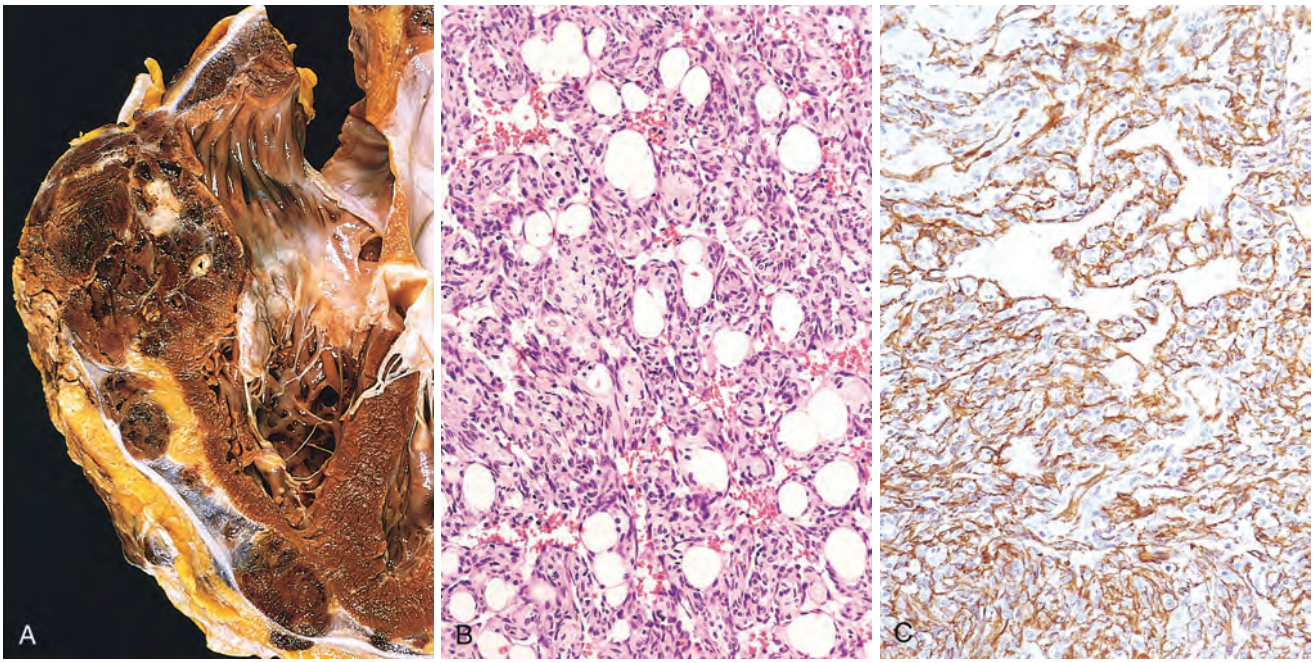

Figure 11-33 Angiosarcoma. **A,** Angiosarcoma involving the right ventricle. **B,** Moderately differentiated angiosarcoma with dense clumps of atypical cells lining distinct vascular lumens. **C,** Immunohistochemical staining for the endothelial cell marker CD31, demonstrating the endothelial nature of the tumor cells.

Hemangiopericytoma. Hemangiopericytomas have been considered tumors that arise from pericytes, the myofibroblast-like cells associated with capillaries and venules. Recent studies suggest that tumors of pericytes are very rare and the vast majority of those previously assigned to this group are derived from other cells such as fibroblasts and are classified as such. One example is the solitary fibrous tumor that arises on the pleura.

KEY CONCEPTS

Vascular Tumors

- Vascular ectasias are not neoplasms, but rather dilations of existing vessels.
- Vessel neoplasms can derive from either blood vessels or lymphatics, and can be composed of endothelial cells (hemangioma, lymphangioma, angiosarcoma) or other components of vascular wall cells
- Most vascular tumors are benign (e.g., hemangiomas), some have an intermediate, locally aggressive behavior (e.g., Kaposi sarcoma), and others are highly malignant (e.g., angiosarcoma).
- Benign tumors typically form obvious vascular channels lined by normal-appearing endothelial cells. Malignant tumors are more often solid and cellular, exhibit cytologic atypia, and lack well-defined vessels.

Pathology of Vascular Intervention

The morphologic changes that occur in vessels following therapeutic intervention (e.g., stenting or bypass surgery) largely recapitulate the changes that occur in the setting of any vascular insult. Local trauma or thrombosis (e.g., due to a stent), or abnormal mechanical forces (e.g., a saphenous vein inserted into the arterial circulation as a coronary artery bypass graft), all induce the same stereotypical healing responses. Analogous to the various insults that drive atherosclerosis, any therapeutic intervention that injures the endothelium also tends to induce intimal thickening by recruiting smooth muscle cells and promoting extracellular matrice deposition.

Endovascular Stenting

Arterial stenoses (especially those in coronary arteries) can be dilated by transiently inflating a balloon catheter to pressures sufficient to rupture the occluding plaque (*balloon angioplasty*); in doing so, a (hopefully) limited *arterial dissection* is also induced. Although most patients improve symptomatically following angioplasty alone, a*brupt reclosure* can occur as a result of compression of the lumen by an extensive circumferential or longitudinal dissection, by vessel wall spasm, or by thrombosis. Thus, more than 90% of endovascular coronary procedures now involve both angioplasty and concurrent *coronary stent* placement.

Coronary stents are expandable tubes of metallic mesh. They provide a larger and more regular lumen, "tack down" the intimal flaps and dissections that occur during angioplasty, and mechanically limit vascular spasm. Nevertheless, due to endothelial injury, *thrombosis* is an important immediate post-stenting complication, and patients must receive potent antithrombotic agents (primarily platelet antagonists) to prevent acute catastrophic thrombotic occlusions. The *long-term* success of angioplasty is limited by the development of *proliferative in-stent restenosis*. This intimal thickening is due to smooth muscle cell ingrowth, proliferation, and matrix synthesis, all driven by the initial vascular wall injury; it causes clinically