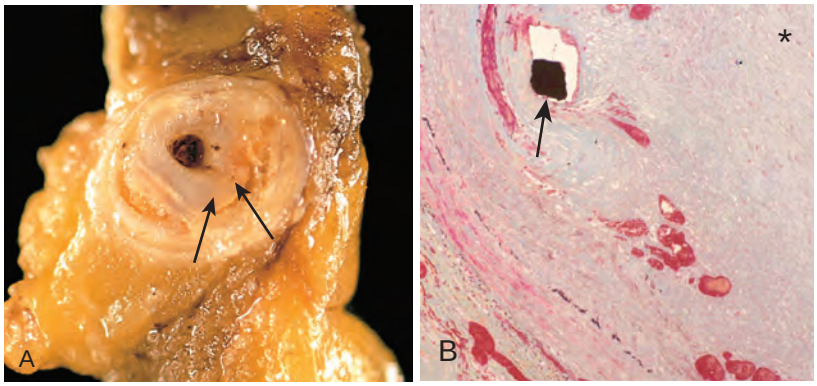

Figure 11-34 Restenosis after angioplasty and stenting. **A,** Gross view demonstrating residual yellow atherosclerotic plaque (*arrows*) and a new, tan-white concentric intimal lesion inside of that plaque. **B,** Histologic view shows a thickened neointima separating and overlying the stent wires (the black diamond indicated by the *arrow*), which encroaches on the lumen (indicated by the *asterisk*); Movat stain with matrix staining gray-green. (***B,*** Reproduced from Schoen FJ, Edwards WD. Pathology of cardiovascular interventions, including endovascular therapies, revascularization, vascular replacement, cardiac assist/replacement, arrhythmia control, and repaired congenital heart disease. In Silver MD, Gotlieb AI, Schoen FJ (eds): Cardiovascular Pathology, 3rd ed. Philadelphia, Churchill Livingstone, 2001.)

significant luminal occlusion in up to a third of patients within 6 to 12 months of stenting (Fig. 11-34).

The newest generation of *drug-eluting stents* is designed to avoid this complication by leaching antiproliferative drugs (e.g., paclitaxel or sirolimus) into the adjacent vessel wall to block smooth muscle cell activation. Although the duration of drug elution is short (days to weeks), these drug-eluting stents nevertheless reduce the incidence of restenosis at 1 year by 50% to 80%. However, because of the antiproliferative effect of the drug-eluting stents, the time to reendothelialization is prolonged and patients require extended courses of anticoagulation to prevent stent thrombosis.

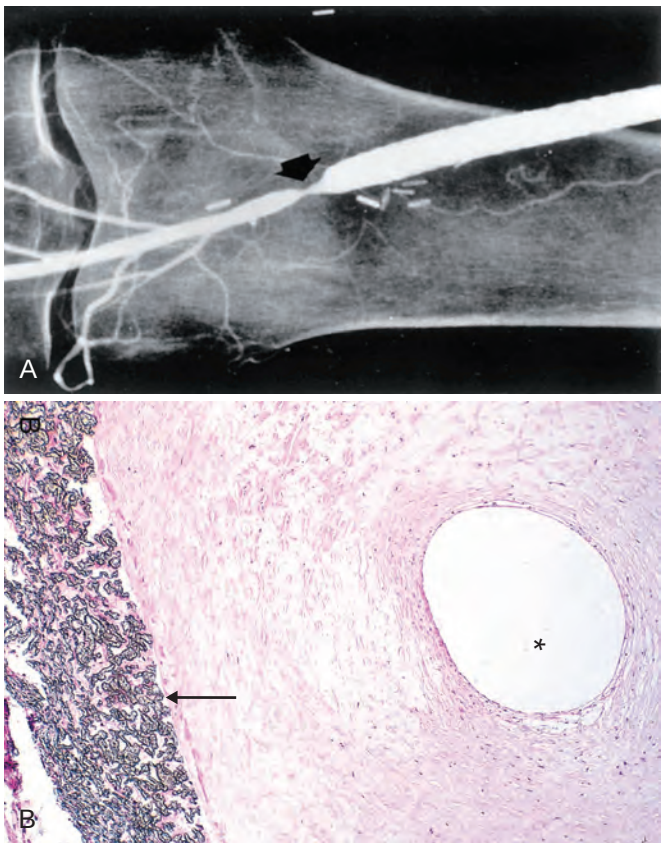

Figure 11-35 Intimal hyperplasia at the distal anastomosis of a synthetic femoral-popliteal graft. **A,** Angiogram demonstrating constriction *(arrow).* **B,** Photomicrograph demonstrating Gore-Tex graft *(arrow)* with prominent intimal proliferation and very small residual lumen *(asterisk).* (**A,** Courtesy Anthony D. Whittemore, MD, Brigham and Women's Hospital, Boston, Mass.)

Vascular Replacement

Synthetic or autologous vascular grafts are increasingly used to replace damaged vessels or bypass diseased arteries. Large-bore (12- to 18-mm) synthetic conduits function well in high-flow locations such as the aorta; unfortunately, small-diameter artificial grafts (≤8 mm in diameter) generally fail as a result of early thrombosis or late intimal hyperplasia, the latter at the junction of the graft with the native vasculature (Fig. 11-35).

Consequently, when small-bore vessel replacement is required (e.g., in coronary bypass surgeries), the grafts are fashioned from saphenous veins (taken from the patient's own leg) or left internal mammary arteries. The long-term patency of saphenous vein grafts is only 50% at 10 years; grafts occlude due to thrombosis (typically early), intimal thickening (months to years postoperatively), and vein graft atherosclerosis—sometimes with superimposed plaque rupture, thrombi, or aneurysms (usually more than 2 to 3 years). By contrast, 90% or more of internal mammary artery grafts are patent at 10 years. With the advent of stenting (and better control of risk factors such as hyperlipidemia), the frequency of coronary arterial bypass surgery has decreased in recent years.

SUGGESTED READINGS

Vascular Structure and Function

Monahan-Earley R, Dvorak AM, Aird WC: Evolutionary origins of the blood vascular system and endothelium. *J Thromb Haemost* 11(Suppl 1):46, 2013. *[Interesting discussion of the evolutionary basis for vascular development, including cogent explanations for endothelial heterogeneity.]*

Semenza G: Vasculogenesis, angiogenesis, and arteriogenesis: mechanisms of blood vessel formation and remodeling. *J Cell Biochem* 102:840, 2007. *[Good overview of physiologic and developmental blood vessel formation and remodeling.]*

Vascular Wall Response to Injury

Gimbrone MA Jr, Garcia-Cardeña G: Vascular endothelium, hemodynamics, and the pathobiology of atherosclerosis. *Cardiovasc Pathol* 22:9, 2013. *[Well-written review on endothelial responses to mechanical forces from one of the leading groups in the field.]*

Pober JS, Min W, Bradley JR: Mechanisms of endothelial dysfunction, injury, and death. *Annu Rev Pathol Mech Dis* 4:71, 2009. *[Well-written and scholarly review of the etiology and outcomes of endothelial injury.]*