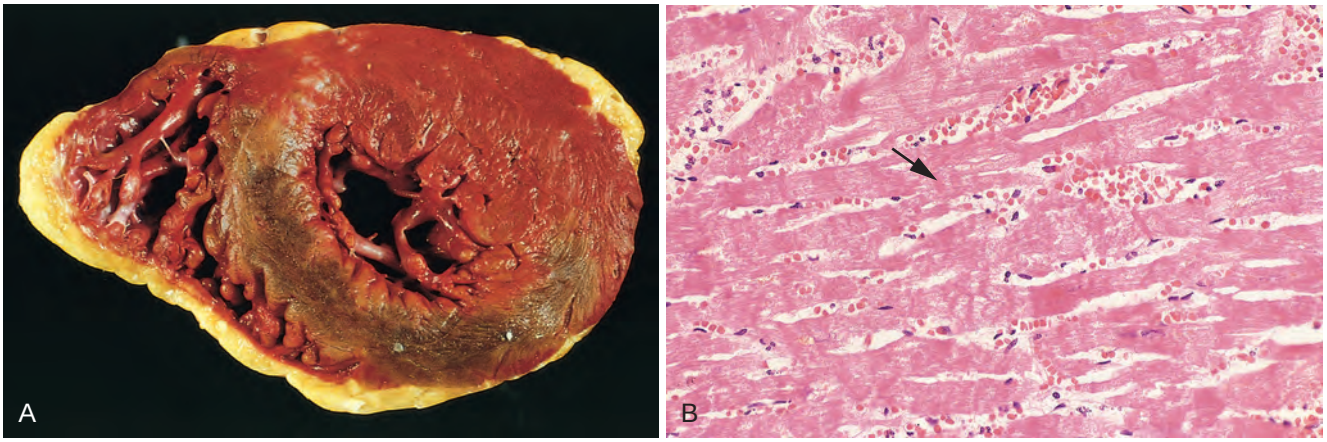

Figure 12-15 Consequences of myocardial ischemia followed by reperfusion. Gross **(A)** and microscopic **(B)** appearance of myocardium modified by reperfusion. **A,** Large, densely hemorrhagic, anterior wall acute myocardial infarction in a patient with left anterior descending artery thrombus treated with streptokinase, a fibrinolytic agent (triphenyl tetrazolium chloride-stained heart slice). Specimen oriented with posterior wall at top. **B,** Myocardial necrosis with hemorrhage and contraction bands, visible as dark bands spanning some myofibers *(arrow)*.

coronary blood flow (the first 3 to 4 hours following obstruction are critical) and (2) the extent of restoration of blood flow and correction of the underlying causal lesion. Indeed, thrombolysis can remove a thrombus occluding a coronary artery, but does not alter the underlying atherosclerotic plaque that initiated it. In contrast, percutaneous transluminal coronary angioplasty (PTCA) with stent placement not only eliminates a thrombotic occlusion but also relieves some of the original obstruction and instability caused by the underlying disrupted plaque. CABG provides a new conduit for flow bypassing the area of vessel blockage.

The typical appearance of reperfused myocardium is illustrated in Figure 12-15. Reperfused infarcts are usually hemorrhagic because the vasculature is injured during ischemia and there is bleeding after flow is restored. Microscopic examination reveals that irreversibly injured myocytes exhibit *contraction bands,* intensely eosinophilic intracellular "stripes" composed of closely packed sarcomeres. These result from the exaggerated contraction of sarcomeres when perfusion is reestablished, at which time the interior of cells with damaged membranes is exposed to a high concentration of calcium ions from the plasma. Thus, *reperfusion not only salvages reversibly injured cells but also alters the morphology of lethally injured cells.*

The effects of reperfusion on myocardial viability and function are discussed later and summarized in Figure 12-16. Although clearly beneficial, reperfusion can trigger deleterious complications, including arrhythmias as well as damage superimposed on the original ischemia, so-called *reperfusion injury.* This term encompasses various forms of damage that can occur after restoration of flow to "vulnerable" myocardium that is ischemic but not yet irreversibly damaged (Fig. 12-16*B*). As discussed in Chapter 2, reperfusion injury may be mediated by oxidative stress, calcium overload, and inflammatory cells recruited after tissue reperfusion. Reperfusion-induced microvascular injury not only results in hemorrhage but can also cause

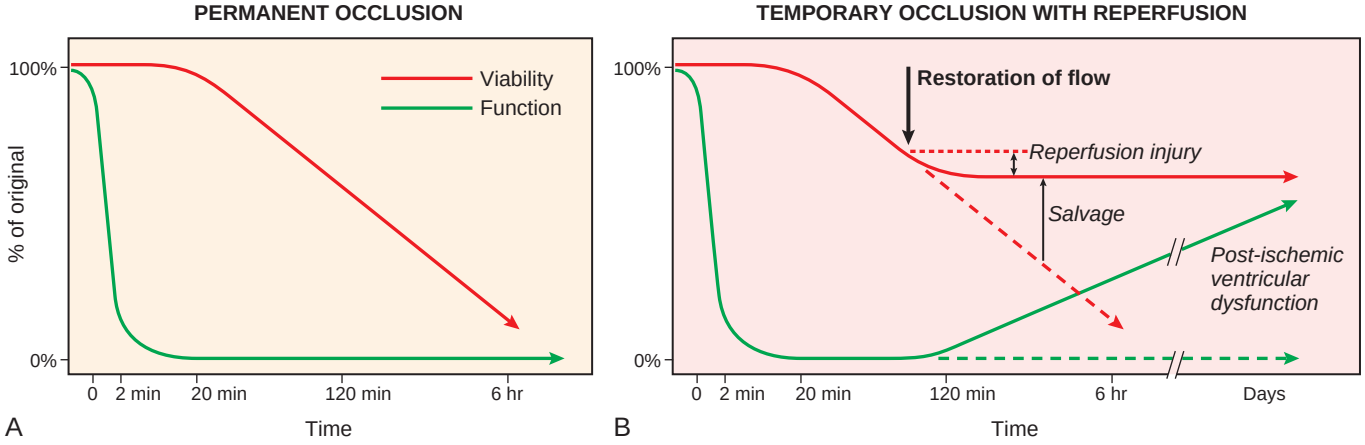

Figure 12-16 Effects of reperfusion on myocardial viability and function. Following coronary occlusion, contractile function is lost within 2 minutes and viability begins to diminish after approximately 20 minutes. If perfusion is not restored (**A**), then nearly all myocardium in the affected region suffers death. **B,** If flow is restored, then some necrosis is prevented, myocardium is salvaged, and at least some function can return. The earlier reperfusion occurs, the greater the degree of salvage. However, the process of reperfusion itself may induce some damage *(reperfusion injury)*, and return of function of salvaged myocardium may be delayed for hours to days *(postischemic ventricular dysfunction* or *stunning)*.