the in-hospital death rate has declined from around 30% to approximately 5% in patients receiving timely therapy. Factors associated with a poorer prognosis include advanced age, female gender, diabetes mellitus, and—due to the cumulative loss of functional myocardium—previous MI. Half of the deaths associated with acute MI occur within 1 hour of onset, most commonly due to a fatal arrhythmia; most of these individuals never reach the hospital. MI therapeutic interventions include:

- Morphine to relieve pain and improve dyspneic symptoms
- Prompt reperfusion to salvage myocardium
- Antiplatelet agents such as aspirin, P2Y12 receptor inhibitors, and GPIIb/IIIa inhibitors
- Anticoagulant therapy with unfractionated heparin, low-molecular-weight heparin, direct thrombin inhibitors, and/or factor Xa inhibitors to prevent coronary artery clot propagation
- Nitrates to induce vasodilation and reverse vasospasm
- Beta blockers to decrease myocardial oxygen demand and to reduce risk of arrhythmias
- Antiarrhythmics to manage arrhythmias
- Angiotensin-converting enzyme (ACE) inhibitors to limit ventricular dilation
- Oxygen supplementation to improve blood oxygen saturation

Despite these interventions, many patients have one or more complications following acute MI (Fig. 12-18):

- *Contractile dysfunction.* Myocardial infarcts produce abnormalities in left ventricular function roughly proportional to their size. There is usually some degree of left ventricular failure with hypotension, pulmonary vascular congestion, and interstitial pulmonary transudates, which can progress to pulmonary edema and respiratory impairment. Severe "pump failure" *(cardiogenic shock)* occurs in 10% to 15% of patients following acute MI, generally with large infarcts involving more than 40% of the left ventricle. Cardiogenic shock has a nearly 70% mortality rate; it accounts for two thirds of in-hospital deaths in those patients admitted for MI.

  Right ventricular infarcts can cause right-sided heart failure associated with pooling of blood in the venous circulation and systemic hypotension.

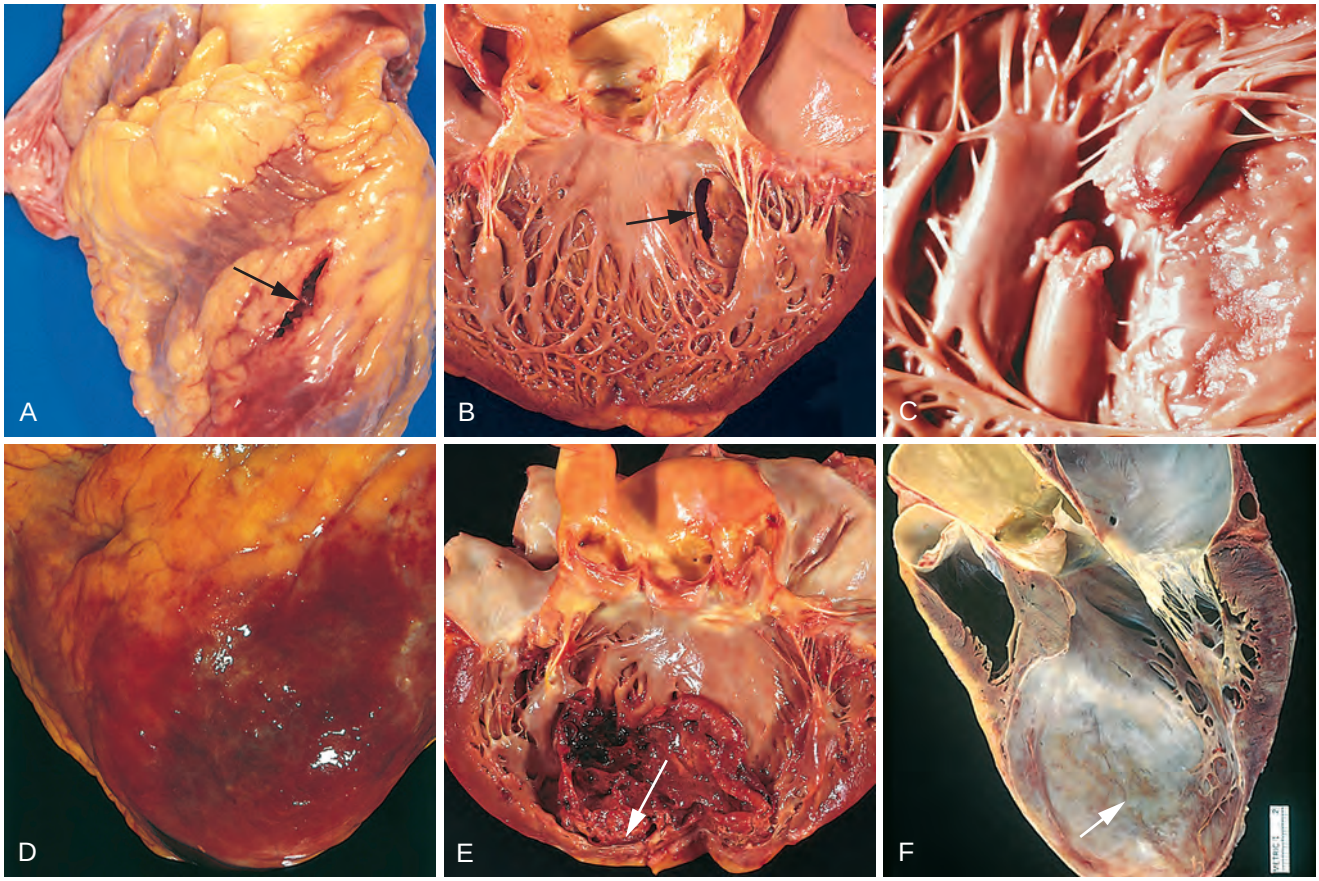

Figure 12-18 Complications of myocardial infarction. **A,** Anterior myocardial rupture in an acute infarct *(arrow).* **B,** Rupture of the ventricular septum *(arrow).* **C,** Complete rupture of a necrotic papillary muscle. **D,** Fibrinous pericarditis, showing a dark, roughened epicardial surface overlying an acute infarct. **E,** Early expansion of anteroapical infarct with wall thinning *(arrow)* and mural thrombus. **F,** Large apical left ventricular aneurysm. The left ventricle is on the right in this apical four-chamber view of the heart. (**A**-**E,** Reproduced with permission from Schoen FJ: Interventional and Surgical Cardiovascular Pathology: Clinical Correlations and Basic Principles. Philadelphia, WB Saunders, 1989; **F,** Courtesy William D. Edwards, MD, Mayo Clinic, Rochester, Minn.)