- *Arrhythmias.* Many patients have *myocardial irritability* and/or conduction disturbances following MI that lead to potentially fatal arrhythmias. MI-associated arrhythmias include sinus bradycardia, atrial fibrillation, heart block, tachycardia, ventricular premature contractions, ventricular tachycardia, and ventricular fibrillation. Because of the location of portions of the atrioventricular conduction system (bundle of His) in the inferoseptal myocardium, infarcts involving this site can also be associated with heart block (see also the discussion concerning arrhythmias).
- *Myocardial rupture.* The various forms of cardiac rupture typically occur when there is transmural necrosis of a ventricle. These include:
  - Rupture of the ventricular free wall (most common), with hemopericardium and cardiac tamponade (Fig. 12-18*A*)
  - Rupture of the ventricular septum (less common), leading to an *acute VSD* and left-to-right shunting (Fig. 12-18*B*)
  - Papillary muscle rupture (least common), resulting in the acute onset of severe mitral regurgitation (Fig. 12-18*C*)

  Free-wall rupture occurs most frequently 2 to 4 days after MI, when coagulative necrosis, neutrophilic infiltration, and lysis of the myocardial connective tissue have appreciably weakened the infarcted myocardium; the anterolateral wall at the mid-ventricular level is the most common site. Risk factors for free-wall rupture include age older than 60, first MI, large, transmural and anterior MI, absence of left ventricular hypertrophy, and preexisting hypertension. Ventricular rupture occurs less frequently in patients with prior MI because associated fibrotic scarring tends to inhibit myocardial tearing. While acute free-wall ruptures are usually rapidly fatal, a fortuitously located pericardial adhesion can abort a rupture and result in a *false aneurysm* (localized hematoma communicating with the ventricular cavity). The wall of a false aneurysm consists only of epicardium and adherent parietal pericardium and thus many still ultimately rupture.
- *Ventricular aneurysm.* In contrast to the *false aneurysms* mentioned earlier, *true aneurysms* of the ventricular wall are bounded by myocardium that has become scarred. Aneurysms of the ventricular wall are a late complication of large transmural infarcts that experience early expansion. The thin scar tissue wall of an aneurysm paradoxically bulges during systole (Fig. 12-18*F*). Complications of ventricular aneurysms include mural thrombus, arrhythmias, and heart failure; rupture of the tough fibrotic wall does not usually occur.
- *Pericarditis.* A fibrinous or fibrinohemorrhagic pericarditis usually develops about the second or third day following a transmural infarct as a result of underlying myocardial inflammation (*Dressler syndrome*; Fig. 12-18*D*).
- Infarct *expansion.* As a result of the weakening of necrotic muscle, there may be disproportionate stretching, thinning, and dilation of the infarct region (especially with anteroseptal infarcts), which is often associated with mural thrombus (Fig. 12-18*E*).
- *Mural thrombus.* With any infarct, the combination of a local abnormality in contractility (causing stasis) and endocardial damage (creating a thrombogenic surface) can foster *mural thrombosis* and potentially *thromboembolism*.
- *Papillary muscle dysfunction.* Although papillary muscle rupture after an MI may certainly result in precipitous onset of mitral (or tricuspid) valve incompetence, most post-infarct regurgitation results from ischemic dysfunction of a papillary muscle (and underlying myocardium), or later from ventricular dilation or from papillary muscle fibrosis and shortening.
- *Progressive late heart failure* (chronic IHD) is discussed later.

The risk of postinfarct complications and the prognosis of the patient depend primarily on the infarct size, location, and fraction of the wall thickness involved (subendocardial or transmural). Thus, large transmural infarcts yield a higher probability of cardiogenic shock, arrhythmias, and late CHF. Patients with anterior transmural infarcts are at greatest risk for free-wall rupture, expansion, mural thrombi, and aneurysm. In contrast, posterior transmural infarcts are more likely to be complicated by conduction blocks, right ventricular involvement, or both; when acute VSDs occur in this area they are more difficult to manage. Moreover, female gender, age older than 70 years, diabetes mellitus and previous MI are poor prognostic factors in patients with ST elevation myocardial infarcts. With subendocardial infarcts, only rarely do pericarditis, rupture, and aneurysms occur.

In addition to the sequence of repair in the infarcted tissues described earlier, the noninfarcted segments of the ventricle undergo hypertrophy and dilation; collectively, these changes are termed *ventricular remodeling*. The compensatory hypertrophy of noninfarcted myocardium is initially hemodynamically beneficial. However, this adaptive effect may be overwhelmed by ventricular dilation (with or without ventricular aneurysm) and increased oxygen demand, which can exacerbate ischemia and depress cardiac function. There may also be changes in ventricular shape and stiffening of the ventricle due to scar formation and hypertrophy that further diminish cardiac output. Some of these deleterious effects appear to be reduced by ACE inhibitors, which lessen the ventricular remodeling that can occurs after infarction.

Long-term prognosis after MI depends on many factors, the most important of which are the residual left ventricular function and the extent of any vascular obstructions in vessels that perfuse the remaining viable myocardium. The overall total mortality within the first year can be as high as 30%; thereafter, each passing year is associated with an additional 3% to 4% mortality among survivors. Infarct prevention (through control of risk factors) in individuals who have never experienced MI *(primary prevention)* and prevention of reinfarction in MI survivors *(secondary prevention)* are important strategies that have received much attention and achieved considerable success.

The relationship of the causes, pathophysiology, and consequences of MI are summarized in Figure 12-19, including the possible outcomes of chronic IHD and sudden death, discussed below.