of rt-PA, although easier to administer, did not further reduce mortality. The major attribute of thrombolytic therapy is its ease of administration, but there is a significant risk (0.5% to 1%) of catastrophic bleeding complications in the form of intracerebral hemorrhage. Age older than 75 years, female gender, hypertension, and concomitant use of heparin increase the risk of this complication. In the case of failed thrombolytic therapy, rescue PCI may be pursued.

Primary PCI has been shown to be superior to thrombolytic therapy based on lower overall mortality rates and reduced risk of recurrent nonfatal MI. It is also associated with higher vessel patency rates and a low risk of intracranial hemorrhage. Primary PCI is frequently performed by mechanical aspiration of thrombus and placement of a coronary stent. Balloon angioplasty may or may not be needed during this procedure. Patients should receive preprocedure thienopyridine (clopidogrel 600 mg or prasugrel 60 mg). Bivalirudin was shown in a clinical trial of primary PCI to be superior to both heparin- and glycoprotein IIb/IIIa–based anticoagulation with lower post-MI mortality and fewer bleeding complications. Centers that are dedicated to primary PCI as the preferred therapy are likely to have the best outcomes when operators are sufficiently skilled and the institution cares for this patient population on a regular basis. Primary PCI is the best option for patients in cardiogenic shock (within 18 hours after onset of shock), for patients with prior CABG (graft occlusion is not amenable to thrombolysis), and for patients older than 70 years of age (conferring a reduced risk of intracerebral hemorrhage compared with thrombolysis).

Complications of Myocardial Infarction

Recurrent Chest Pain

MI is associated with a number of possible problems related to the extent of injury (Table 8-6). Patients can experience post-infarction angina which may reflect re-occlusion of the infarct related vessel. This can occur either in patients who underwent primary PCI with stent placement (stent thrombosis) or thrombolysis. Post-infarction angina usually requires cardiac catheterization for appropriate diagnosis and treatment. Patients with transmural MI are also subject to pericarditis 2 to 4 days after the event. This diagnosis is usually established by the symptom nature and pattern (worse with inspiration or supine position, improved with sitting), which is different from their initial presentation with acute MI. A less common event is the development of pericarditis due to Dressler's syndrome up to 10 weeks after acute MI. This is likely an immune-mediated phenomenon. Pericarditis is treated with aspirin or nonsteroidal anti-inflammatory drugs.

TABLE 8-6 COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

FUNCTIONAL
Left ventricular failure Right ventricular failure Cardiogenic shock
MECHANICAL
Free-wall rupture Ventricular septal defect Papillary muscle rupture with acute mitral regurgitation
ELECTRICAL
Bradyarrhythmias (first-, second-, and third-degree atrioventricular blocks) Tachyarrhythmias (supraventricular, ventricular) Conduction abnormalities (bundle branch and fascicular blocks)

Arrhythmias

The highest risk of life-threatening arrhythmias is during the first 24 to 48 hours after the onset of acute MI. Ischemic myocardium is susceptible to arrhythmia generation, probably based on micro-re-entry associated with ischemic myocardium. The significant mortality risk in the early hours of acute MI is largely attributed to arrhythmias such as VF or VT. The risk of VF is about 3% to 5% in the early hours of MI and diminishes over 24 to 48 hours. One of the benefits of rhythm monitoring during the first 48 hours after presentation is prompt recognition and treatment of life-threatening ventricular arrhythmias.

Accelerated idioventricular rhythm occurs early in the course of MI and may be associated with reperfusion. This arrhythmia is well tolerated and does not require specific therapy.

Ventricular arrhythmias occurring late (>48 hours) after acute MI usually are associated with large underlying MIs and heart failure. Late episodes of VF or VT portend a poor prognosis. Immediate therapy for VF is electrical defibrillation. VT that causes hemodynamic embarrassment is treated with synchronized electrical cardioversion. β-Blocker therapy may help to suppress arrhythmias in patients who are prone to them, as may the use of amiodarone. Correction of residual ischemia may also play a role in controlling VF or VT events. Patients with late VF or hemodynamically significant VT are candidates for an implantable cardioverter defibrillator device (ICD). An ICD can also improve survival in asymptomatic patients with a persistently reduced EF less than 30% at 40 days after their acute MI. ICD therapy is also indicated if the EF is less than 35% at 40 days after MI in a patient with symptomatic heart failure.

Atrial fibrillation (AF) occurs in 10% to 15% of patients after MI. Those more prone to AF include patients with older age, large MI, hypokalemia, hypomagnesemia, hypoxia, or increased sympathetic activity. Rate control with β-blockers (e.g., metoprolol), digoxin, calcium channel blockers (e.g., diltiazem) or some combination of these agents is warranted, as is the use of intravenous heparin to reduce the risk of systemic embolization. Cardioversion is warranted in the face of rapid rates that cause ischemia, heart failure, or hypotension. Amiodarone is sometimes used to help maintain sinus rhythm for the first few months after MI-related AF.

Sinus bradycardia or AV block due to increased vagal tone is common in cases of inferior MI (30% to 40%). Reperfusion of the right coronary artery may be associated with significant bradycardia (Bezold-Jarisch reflex). Atropine (0.5 to 1.5 mg IV) can resolve severe inferior MI–related bradycardia. In contrast, heart block and wide-complex escape rhythms associated with anterior MI suggest an infra–AV node block. This may be worsened by the use of atropine.

Advanced degrees of heart block may require the placement of a permanent pacemaker. Intermittent second-degree or third-degree AV block associated with bundle branch block or