symptomatic AV block are indications for a permanent pacemaker. Type I AV block (Wenckebach) is usually not persistent and rarely causes symptoms that warrant a permanent pacemaker.

Heart Failure and Low-Output States

MI involving 20% to 25% of the left ventricle can result in significant heart failure manifesting with dyspnea due to pulmonary congestion and findings of LV dysfunction such as an S_3 or S_4. Cardiogenic shock is associated with loss of 40% of the myocardium. This condition carries a very high risk of mortality. In the era of widespread use of reperfusion therapy, the incidence of post-MI heart failure or cardiogenic shock has declined. Early use of reperfusion therapies limits infarct size and the risk of complications related to heart failure. When acute heart failure occurs with MI, therapeutic interventions including oxygen, intravenous morphine, and diuretics can help stabilize the patient. Nitroglycerin can also help by reducing the elevated preload. Long-term therapy for heart failure related to reduced EF after acute MI includes the use of ACE inhibitors (or ARBs), appropriate β-blockers, aldosterone receptor antagonists such as eplerenone or spironolactone, and diuretics as needed.

The acutely infarcted ventricle requires an increased filling pressure and volume to optimize its performance. Patients with acute MI may become relatively fluid depleted due to nausea, vomiting, or decreased fluid intake, leading to reduced LV volume and a fall in cardiac output. This can translate into hypotension that is best treated by judicious administration of fluids.

Acute inferior MI is usually associated with a low mortality risk once the early arrhythmia-prone hours have passed. Occlusion of the right coronary artery and a significant acute marginal branch can lead to right ventricular infarction. Approximately 10% to 15% of patients with inferior MI have associated right ventricular infarction. This condition produces a significant increase in mortality risk (in-hospital mortality, 25% to 30% vs. <6%). Hallmarks of right ventricular infarction include elevated jugular venous pressure with Kussmaul sign and hypotension. Right ventricular function frequently recovers, but it may be necessary to administer sufficient volume to maintain right heart output. Short-term inotropic support with dobutamine in sometimes needed, and venodilators and diuretics should be avoided. High-degree AV block, usually transient with inferior MI, may worsen hemodynamics and necessitate temporary AV sequential pacing. AF may not be tolerated and may require cardioversion.

Cardiogenic Shock

Cardiogenic shock is a clinical syndrome associated with extensive loss of myocardium, which leads to a reduced cardiac index (<1.8 L/min/m^2) in the face of elevated LV filling pressures (pulmonary capillary wedge pressure >18 mm Hg), resulting in systemic hypotension and reduced organ perfusion. This shock state is associated with mortality rates in the range of 70% to 80%. Aggressive diagnosis with hemodynamic monitoring and appropriate support with an intra-aortic balloon pump (IABP) and inotropic agents as indicated can help to stabilize the patient. IABP therapy is at best temporizing, and the patient's survival depends on the presence of reversible factors such as ischemia that respond to revascularization or correction of a mechanical complication of MI (e.g., mitral regurgitation or VSD. IABP therapy cannot be used in the face of significant aortic insufficiency and may not be feasible in the presence of significant peripheral vascular disease. Some centers now resort to ventricular assist devices to stabilize the patient with cardiogenic shock, but recovery of LV function is not guaranteed with this approach.

Mechanical Complications

Mechanical complications of acute MI include mitral regurgitation (due to ischemic papillary muscle dysfunction or rupture), VSD, free wall rupture, and LV aneurysm formation. These problems usually occur during the first week after MI, and they account for as much as 15% of MI-related mortality. A new murmur, sudden onset of heart failure, or hemodynamic collapse should also raise suspicion of a mechanical complication of MI. Patients who either were not reperfused or were reperfused late after onset of MI are most at risk for these problems. Echocardiography usually identifies the mechanical problem, and hemodynamic assessment with right heart catheterization can aid the diagnosis. Surgical correction of the defect is usually required.

Papillary muscle rupture or dysfunction leading to acute severe mitral regurgitation results in severe heart failure and up to 75% mortality within 24 hours after onset. Afterload reduction with intravenous nitroprusside and the use of IABP can help to stabilize the patient, but surgical valve repair or replacement will be needed to provide some chance of survival. Surgery is associated with a 25% to 50% mortality risk, but that still is better than the risk with medical or IABP therapy only.

Elderly patients, particularly those with hypertension, are more prone to MI-related VSD. Thrombolytic therapy may also place patients at risk for this complication. Acute VSD with resultant left-to-right shunting can produce severe hemodynamic instability. As with acute mitral regurgitation, afterload reduction and IABP may help to stabilize the patient, but ultimately surgical repair will be required. Moderate to large VSDs are not well tolerated and are associated with significant mortality risk. VSDs related to anterior MI may offer a better opportunity for surgical repair than those resulting from inferior MI. Some patients have been helped by the use of percutaneous closure devices, which can afford an opportunity to delay surgery until there is better tissue healing in the infarct area.

LV free wall rupture is similar to VSD in terms of risk for occurrence and underlying myocardial pathology. Free wall rupture is usually associated with sudden death due to cardiac tamponade. On occasion, a pseudoaneurysm forms and the patient can be treated surgically.

Thromboembolic Complications

In earlier years, thromboembolism in the form of either cardioembolic stroke or pulmonary embolism contributed to 25% of post-MI in-hospital mortality, and clinical events were diagnosed in 10% of patients. The risk of thromboembolism is linked to the presence of LV mural clot, which is more likely to be found in anterior MI with associated apical akinesis and deep venous thrombosis due to prolonged bed-rest. Contemporary methods