TABLE 326-1 ETIOLOGIES OF CARDIOGENIC SHOCK (CS)[a] AND CARDIOGENIC PULMONARY EDEMA

Etiologies of Cardiogenic Shock or Pulmonary Edema
Acute myocardial infarction/ischemia
LV failure
Ventricular septal rupture
Papillary muscle/chordal rupture–severe MR
Ventricular free wall rupture with subacute tamponade
Other conditions complicating large MIs
Hemorrhage
Infection
Excess negative inotropic or vasodilator medications
Prior valvular heart disease
Hyperglycemia/ketoacidosis
Post-cardiac arrest
Post-cardiotomy
Refractory sustained tachyarrhythmias
Acute fulminant myocarditis
End-stage cardiomyopathy
LV apical ballooning
Takotsubo's cardiomyopathy
Hypertrophic cardiomyopathy with severe outflow obstruction
Aortic dissection with aortic insufficiency or tamponade
Severe valvular heart disease
Critical aortic or mitral stenosis
Acute severe aortic regurgitation or mitral regurgitation
Toxic/metabolic
β blocker or calcium channel antagonist overdose
Other Etiologies of Cardiogenic Shock[b]
RV failure due to:
Acute myocardial infarction
Acute cor pulmonale
Refractory sustained bradyarrhythmias
Pericardial tamponade
Toxic/metabolic
Severe acidosis, severe hypoxemia

[a]The etiologies of CS are listed. Most of these can cause pulmonary edema instead of shock or pulmonary edema with CS. [b]These cause CS but not pulmonary edema.

Abbreviations: LV, left ventricular; MI, myocardial infarction; MR, mitral regurgitation; RV, right ventricular; VSR, ventricular septal rupture.

Echocardiography is an invaluable diagnostic tool in patients with suspected CS.

CLINICAL FINDINGS Most patients have dyspnea and appear pale, apprehensive, and diaphoretic, and mental status may be altered. The pulse is typically weak and rapid, often in the range of 90–110 beats/min, or severe bradycardia due to high-grade heart block may be present. Systolic BP is reduced (<90 mmHg or ≥30 mmHg below baseline) with a narrow pulse pressure (<30 mmHg), but occasionally BP may be maintained by very high systemic vascular resistance. Tachypnea, Cheyne-Stokes respirations, and jugular venous distention may be present. There is typically a weak apical pulse and soft S_1, and an S_3 gallop may be audible. Acute, severe MR and VSR usually are associated with characteristic systolic murmurs (Chap. 295). Rales are audible in most patients with LV failure. Oliguria is common.

LABORATORY FINDINGS The white blood cell count is typically elevated with a left shift. Renal function is initially unchanged, but blood urea nitrogen and creatinine rise progressively. Hepatic transaminases may be markedly elevated due to liver hypoperfusion. The lactic acid level is elevated. Arterial blood gases usually demonstrate hypoxemia and anion gap metabolic acidosis, which may be compensated by respiratory alkalosis. Cardiac markers, creatine phosphokinase and its MB fraction, and troponins I and T are typically markedly elevated.

ELECTROCARDIOGRAM In CS due to acute MI with LV failure, Q waves and/or >2-mm ST elevation in multiple leads or left bundle branch block are usually present. More than one-half of all infarcts associated with shock are anterior. Global ischemia due to severe left main stenosis usually is accompanied by severe (e.g., >3 mm) ST depressions in multiple leads.

CHEST ROENTGENOGRAM The chest x-ray typically shows pulmonary vascular congestion and often pulmonary edema, but these findings may be absent in up to a third of patients. The heart size is usually normal when CS results from a first MI but is enlarged when it occurs in a patient with a previous MI.

ECHOCARDIOGRAM A two-dimensional echocardiogram with color-flow Doppler (Chap. 270e) should be obtained promptly in patients with suspected CS to help define its etiology. Doppler mapping demonstrates a left-to-right shunt in patients with VSR and the severity of MR when the latter is present. Proximal aortic dissection with aortic regurgitation or tamponade may be visualized, or evidence for pulmonary embolism may be obtained (Chap. 300).

PULMONARY ARTERY CATHETERIZATION The use of pulmonary artery (Swan-Ganz) catheters in patients with established or suspected CS is controversial (Chaps. 272 and 321). Their use is generally recommended for measurement of filling pressures and cardiac output to confirm the diagnosis and to optimize the use of IV fluids, inotropic agents, and vasopressors in persistent shock (Table 326-2). O_2 saturation measurement from right atrial, RV, and pulmonary arterial blood samples can rule out a left-to-right shunt. In CS, low mixed venous O_2 saturations and elevated arteriovenous (AV) O_2 differences reflect low cardiac index and high fractional O_2 extraction. However, when sepsis accompanies CS, AV O_2 differences may not be elevated (Chap. 324). The PCWP is elevated. Use of sympathomimetic amines may return these measurements and the systemic BP to normal. Systemic vascular resistance may be low, normal, or elevated in CS. Equalization of right- and left-sided filling pressures (right atrial and PCWP) suggests cardiac tamponade as the cause of CS (Chap. 288).

LEFT HEART CATHETERIZATION AND CORONARY ANGIOGRAPHY Measurement of LV pressure and definition of the coronary anatomy provide useful information and are indicated in most patients with CS complicating MI. Cardiac catheterization should be performed when there is a plan and capability for immediate coronary intervention (see below) or when a definitive diagnosis has not been made by other tests.

TREATMENT ACUTE MYOCARDIAL INFARCTION

GENERAL MEASURES

(Fig. 326-2) In addition to the usual treatment of acute MI (Chap. 295), initial therapy is aimed at maintaining adequate systemic and coronary perfusion by raising systemic BP with vasopressors and adjusting volume status to a level that ensures optimum LV filling pressure. There is interpatient variability, but the values that generally are associated with adequate perfusion are systolic BP ~90 mmHg or mean BP >60 mmHg and PCWP >20 mmHg. Hypoxemia and acidosis must be corrected; most patients require ventilatory support (see "Pulmonary Edema," below). Negative inotropic agents should be discontinued and the doses of renally cleared medications adjusted. Hyperglycemia should be controlled with insulin. Bradyarrhythmias may require transvenous pacing. Recurrent ventricular tachycardia or rapid atrial fibrillation may require immediate treatment (Chap. 276).

VASOPRESSORS

Various IV drugs may be used to augment BP and cardiac output in patients with CS. All have important disadvantages, and none has been shown to change the outcome in patients with established shock. *Norepinephrine* is a potent vasoconstrictor and inotropic stimulant that is useful for patients with CS. As first line of therapy norepinephrine was associated with fewer adverse events, including arrhythmias, compared to a dopamine randomized trial of patients