immunosuppressives for entities such as giant cell myocarditis. Refractory CS can be managed with MCS.

### ■ PULMONARY EDEMA

**The etiologies and pathophysiology of pulmonary edema are discussed in Chap. 37.**

**Diagnosis** Acute pulmonary edema usually presents with the rapid onset of dyspnea at rest, tachypnea, tachycardia, and severe hypoxemia. Crackles and wheezing due to alveolar flooding and airway compression from peribronchial cuffing may be audible. Release of endogenous catecholamines often causes hypertension.

It is often difficult to distinguish between cardiogenic and noncardiogenic causes of acute pulmonary edema. *Echocardiography* may identify systolic and diastolic ventricular dysfunction and valvular lesions. ECG ST elevation and evolving Q waves are usually diagnostic of acute MI and should prompt immediate institution of MI protocols and coronary artery revascularization therapy **(Chap. 275)**. Brain natriuretic peptide levels, when substantially elevated, support heart failure as the etiology of acute dyspnea with pulmonary edema **(Chap. 257)**.

The use of a PAC permits measurement of pulmonary capillary wedge pressures (PCWP) and helps differentiate high-pressure (cardiogenic) from normal-pressure (noncardiogenic) causes of pulmonary edema. Pulmonary artery catheterization is indicated when the etiology of the pulmonary edema is uncertain, when edema is refractory to therapy, or when it is accompanied by refractory hypotension. Data derived from use of a PAC often alter the treatment plan, but no impact on mortality rates has been demonstrated.

## TREATMENT

# Pulmonary Edema

The treatment of pulmonary edema depends on the specific etiology. As an acute, life-threatening condition, a number of measures must be applied immediately to support the circulation,