normal ventricular function and a low pulmonary vascular resistance for the procedure to be successful. The modified Fontan procedure separates oxygenated and unoxygenated blood inside the heart and eliminates the excess volume load on the ventricle but does not restore normal anatomy or hemodynamics. This operation is also the final stage in the correction of many complex defects with a functional single ventricle, including HLHS.

Prognosis: Surgical mortality following the Fontan procedure is less than 3% (Park, 2014). The overall survival rate after the Fontan operation was above 95% at follow up of 50 months (Hirsch, Goldberg, Bove, et al, 2008). Postoperative complications include dysrhythmias, systemic venous hypertension, pleural and pericardial effusions, and ventricular dysfunction. Long-term concerns are the development of protein-losing enteropathy, atrial dysrhythmias, late ventricular dysfunction, and developmental delays.

ASD, Atrial septal defect; *CHD,* congenital heart disease; *HF,* heart failure; *HLHS,* hypoplastic left heart syndrome; *PDA,* patent ductus arteriosus; *PS,* pulmonic stenosis; *RVOT,* right ventricular outflow tract; *TGA,* transposition of the great arteries; *VSD,* ventricular septal defect.

Mixed Defects

Many complex cardiac anomalies are classified together in the mixed category (Box 23-4), because survival in the postnatal period depends on mixing of blood from the pulmonary and systemic circulations within the heart chambers. Hemodynamically, fully saturated systemic blood flow mixes with the desaturated pulmonary blood flow, causing a relative desaturation of the systemic blood flow. Pulmonary congestion occurs because the differences in pulmonary artery pressure and aortic pressure favor