**TABLE 272-3 HEMODYNAMIC FINDINGS IN TAMPONADE, CONSTRICTIVE PERICARDITIS, AND RESTRICTIVE CARDIOMYOPATHY**

| | Cardiac Tamponade | Constrictive Pericarditis | Effusive-Constrictive Pericarditis | Restrictive Cardiomyopathy |
|---|---|---|---|---|
| Pericardial pressure | ↑ | ↑ | ↑ | Normal |
| Right atrium pressure | ↑ | ↑ | ↑ (Fails to decrease by 50% or to <10 mmHg after pericardiocentesis) | ↑ |
| Right atrium pressure waveform | Prominent "x" descent | Prominent "x" descent | Prominent "x" descent | Prominent "y" descent |
| | Diminished or absent "y" descent | Prominent "y" descent | "y" descent less prominent than expected | |
| Right ventricle systolic pressure | <50 mmHg | <50 mmHg | <50 mmHg | >60 mmHg |
| Right ventricle end-diastolic pressure | | >1/3 right ventricular systolic pressure | >1/3 right ventricular systolic pressure | <1/3 right ventricular systolic pressure |
| | Equals left ventricular end-diastolic pressure within 5 mmHg | Equals left ventricular end-diastolic pressure within 5 mmHg | Equals left ventricular end-diastolic pressure within 5 mmHg | Less than left ventricular end-diastolic pressure by ≥5 mmHg |
| Right ventricle pressure waveform | | Dip and plateau or "square root" sign | Dip and plateau or "square root" sign | Dip and plateau or "square root" sign |
| Right ventricle–left ventricle systolic pressure relationship with inspiration | Discordant | Discordant | Discordant | Concordant |

uses oxygen as the indicator substance and is based on the principle that the amount of a substance taken up or released by an organ (oxygen consumption) is equal to the product of its blood flow (cardiac output) and the difference in the concentration of the substance in the arterial and venous circulation (arterial-venous oxygen difference). Thus, the formula for calculating the Fick cardiac output is:

Cardiac output (L/min) = (oxygen consumption [mL/min])/(arterial-venous oxygen difference [mL/L])

Oxygen consumption is estimated as 125 mL oxygen/minute × body surface area, and the arterial-venous oxygen difference is determined by first calculating the oxygen carrying capacity of blood (hemoglobin [g/100 mL] × 1.36 [mL oxygen/g hemoglobin] × 10) and multiplying this product by the fractional oxygen saturation. The thermodilution method measures a substance that is injected into and adequately mixes with blood. In contemporary practice, thermodilution cardiac outputs are measured using temperature as the indicator. Measurements are made with a thermistor-tipped catheter that detects temperature deviations in the pulmonary artery after the injection of 10 mL of room-temperature normal saline into the right atrium.

**Vascular Resistance** Resistance across the systemic and pulmonary circulations is calculated by extrapolating from Ohm's law of electrical resistance and is equal to the mean pressure gradient divided by the mean flow (cardiac output). Therefore, systemic vascular resistance is ([mean aortic pressure − mean right atrial pressure]/cardiac output) multiplied by 80 to convert the resistance from Wood units to dyn-s-cm$^{-5}$. Similarly, the pulmonary vascular resistance is ([mean pulmonary artery − mean pulmonary capillary wedge pressure]/cardiac output) × 80. Pulmonary vascular resistance is lowered by oxygen, nitroprusside, calcium channel blockers, prostacyclin infusions, and inhaled nitric oxide; these therapies may be administered during catheterization to determine if increased pulmonary vascular resistance is fixed or reversible.

**Valve Area** Hemodynamic data may also be used to calculate the valve area using the Gorlin formula that equates the area to the flow across the valve divided by the pressure gradient between the cardiac chambers surrounding the valve. The formula for the assessment of valve area is: Area = (cardiac output [cm$^3$/min]/[systolic ejection period or diastolic filling period][heart rate])/44.3 C × square root of the pressure gradient, where C = 1 for aortic valve and 0.85 for the mitral valve. A valve area of <1.0 cm$^2$ and a mean gradient of greater than 40 mmHg indicate severe aortic stenosis, while a valve area of <1.5 cm$^2$ and a mean gradient >5–10 mmHg is consistent with moderate-to-severe mitral stenosis; in symptomatic patients with a mitral valve area >1.5 cm$^2$, a mean gradient >15 mmHg, pulmonary artery pressure >60 mmHg, or a pulmonary artery wedge pressure >25 mmHg after exercise is also considered significant and may warrant intervention. The modified Hakki formula has also been used to estimate aortic valve area. This formula calculates the valve area as the cardiac output (L/min) divided by the square root of the pressure gradient. Aortic valve area calculations based on the Gorlin formula are flow-dependent and, therefore, for patients with low cardiac outputs, it is imperative to determine if a decreased valve area actually reflects a fixed stenosis or is overestimated by a low cardiac output and stroke volume that is insufficient to open the valve leaflets fully. In these instances, cautious hemodynamic manipulation using dobutamine to increase the cardiac output and recalculation of the aortic valve area may be necessary.

**Intracardiac Shunts** In patients with congenital heart disease, detection, localization, and quantification of the intracardiac shunt should be evaluated. A shunt should be suspected when there is unexplained arterial desaturation or increased oxygen saturation of venous blood. A "step up" or increase in oxygen content indicates the presence of a left-to-right shunt while a "step down" indicates a right-to-left shunt. The shunt is localized by detecting a difference in oxygen saturation levels of 5–7% between adjacent cardiac chambers. The severity of the shunt is determined by the ratio of pulmonary blood flow ($Q_p$) to the systemic blood flow ($Q_s$), or $Q_p/Q_s$ = ([systemic arterial oxygen content − mixed venous oxygen content]/pulmonary vein oxygen content − pulmonary artery oxygen content). For an atrial septal defect, a shunt ratio of 1.5 is considered significant and factored with other clinical variables to determine the need for intervention. When a congenital ventricular septal defect is present, a shunt ratio of ≥2.0 with evidence of left ventricular volume overload is a strong indication for surgical correction.

## VENTRICULOGRAPHY AND AORTOGRAPHY

Ventriculography to assess left ventricular function may be performed during cardiac catheterization. A pigtail catheter is advanced retrograde across the aortic valve into the left ventricle and 30–45 mL of contrast is power-injected to visualize the left ventricular chamber during the cardiac cycle. The ventriculogram is usually performed in the right anterior oblique projection to examine wall motion and mitral valve function. Normal wall motion is observed as symmetric contraction of all segments; hypokinetic segments have decreased contraction, akinetic segments do not contract, and dyskinetic segments appear to bulge paradoxically during systole (Fig. 272-3). Ventriculography may also reveal a left ventricular aneurysm, pseudoaneurysm, or diverticulum and can be used to assess mitral valve prolapse and the severity of mitral regurgitation. The degree of mitral regurgitation is estimated