1. A 32-year-old woman is referred for evaluation of rheumatic mitral valve stenosis. No mitral regurgitation was noted. The following values were obtained by Doppler echocardiography:

<table>
<thead>
<tr>
<th>Table 8-1</th>
</tr>
</thead>
<tbody>
<tr>
<td>E wave deceleration time</td>
</tr>
<tr>
<td>Mean diastolic mitral gradient</td>
</tr>
<tr>
<td>Diastolic mitral inflow velocity–time integral</td>
</tr>
<tr>
<td>Heart rate</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Mitral valve area can be calculated by dividing 220 into deceleration time.
B. Stroke volume across the mitral valve is 72 mL per beat.
C. Pressure half-time is 355 ms.
D. Mitral valve area is 0.8 cm².
E. During exertion, her mean gradient is expected to decrease.

2. A 21-year-old man with dyspnea on exertion and enlarged pulmonary artery on chest x-ray underwent transthoracic echocardiography. The study revealed patent ductus arteriosus and the following:

<table>
<thead>
<tr>
<th>Table 8-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular outflow tract (LVOT) diameter</td>
</tr>
<tr>
<td>LVOT velocity–time integral</td>
</tr>
<tr>
<td>Right ventricular outflow tract (RVOT) diameter</td>
</tr>
<tr>
<td>RVOT velocity–time integral</td>
</tr>
<tr>
<td>Heart rate</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Systemic blood flow (Qs) is 7.8 L/min.
B. The ratio of pulmonic to systemic blood flow (Qp:Qs) is less than 1.
C. Stroke volume entering the lungs is 38 mL per beat.
D. Patient is cyanotic in the lower parts of the body.
E. The ratio of stroke volume through the LVOT and the stroke volume through the RVOT is equal to Qp:Qs ratio in this patient.

3. A 39-year-old woman was admitted for severe shortness of breath on exertion. On transthoracic echocardiogram, there was mild pulmonic regurgitation. Continuous wave spectral Doppler tracings of the pulmonic regurgitant jet reveal the following:

<table>
<thead>
<tr>
<th>Table 8-3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early diastolic peak velocity</td>
</tr>
<tr>
<td>End-diastolic velocity</td>
</tr>
</tbody>
</table>
Examination of the inferior vena cava by M-mode echocardiography demonstrated the following:

**Table 8-4**

<table>
<thead>
<tr>
<th>Description</th>
<th>Measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>IVC diameter during expiration</td>
<td>2.6 cm</td>
</tr>
<tr>
<td>IVC diameter during inspiration</td>
<td>2.6 cm</td>
</tr>
</tbody>
</table>

The following statement is TRUE:

A. Right atrial pressure (RAP) is estimated at 6 mm Hg.
B. Pulmonary artery diastolic pressure is greater than 31 mm Hg.
C. Pulmonary artery diastolic pressure is 36 mm Hg minus the right atrial pressure.
D. Pulmonary artery diastolic pressure cannot be assessed if the pulmonic regurgitation is only mild.
E. Pulmonary artery diastolic pressure is normal.

4. A 42-year-old man was admitted to the hospital after a 1-month history of intermittent fever and progressive shortness of breath. Blood cultures grew *Streptococcus viridans*. On transesophageal echocardiogram, perforation of the anterior mitral leaflet and mitral regurgitation were seen. On the color Doppler image, a well-formed flow convergence (PISA) shell was visualized on the ventricular side of the mitral valve in systole. In addition, the following was noted:

**Table 8-5**

<table>
<thead>
<tr>
<th>Description</th>
<th>Measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal mitral regurgitation PISA radius</td>
<td>1.0 cm</td>
</tr>
<tr>
<td>Aliasing velocity at which PISA radius measured</td>
<td>45 cm/s</td>
</tr>
<tr>
<td>Peak velocity of mitral regurgitation jet</td>
<td>500 cm/s</td>
</tr>
<tr>
<td>Velocity–time integral of mitral regurgitation</td>
<td>140 cm</td>
</tr>
</tbody>
</table>

The following statement is TRUE:

A. Vena contracta of the mitral regurgitant flow is expected to be less than 0.3 cm.
B. Effective regurgitant orifice area of mitral regurgitation is approximately 0.6 cm$^2$.
C. Instantaneous flow rate across the mitral valve using the PISA method is 70 mL/s.
D. Mitral regurgitation is moderate (2+).
E. Regurgitant volume is 40 mL per beat.

5. An 84-year-old obese woman with history of hypertension and chronic renal insufficiency became very short of breath at a rehabilitation facility 2 weeks after elective hip replacement. Transthoracic echocardiogram revealed normal left ventricular systolic function, no mitral or aortic valve disease, and the following:

**Table 8-6**

<table>
<thead>
<tr>
<th>Description</th>
<th>Measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak velocity of the mitral E wave</td>
<td>125 cm/s</td>
</tr>
<tr>
<td>Flow propagation velocity of mitral inflow on color M mode</td>
<td>31 cm/s</td>
</tr>
<tr>
<td>Peak velocity of tricuspid regurgitant jet</td>
<td>4 m/s</td>
</tr>
<tr>
<td>Estimated right atrial pressure</td>
<td>15 mm Hg</td>
</tr>
</tbody>
</table>

The following statement is TRUE:

A. Mean pulmonary artery wedge pressure is markedly elevated.
B. On mitral inflow, E to A ratio is expected to be less than 1.
C. Pulmonary artery systolic pressure is 64 mm Hg.
D. The ratio of peak E wave velocity to the peak medial mitral annular tissue Doppler velocity is expected to be less than 8.
E. Flow propagation velocity of mitral inflow on color M mode is normal for her age.

6. A 44-year-old man with trileaflet aortic valve and dilated aortic root measuring 5.5 cm at the level of sinuses of Valsalva is being evaluated for aortic regurgitation. The following statement is TRUE:

A. Regurgitant fraction of 65% would indicate that the aortic regurgitation is severe.
B. Like the size of flow convergence (PISA) radius, the size of vena contracta is strongly influenced by Nyquist limit setting.
C. Vena contracta of at least 0.2 cm would indicate that the aortic regurgitation is severe.
D. Regurgitant volume of 30 mL per beat is consistent with severe aortic regurgitation.
E. Vena contracta obtained by two-dimensional echocardiography can be used to calculate regurgitant volume.
7. A 62-year-old man with history of treated hypertension, chronic atrial fibrillation, and bicuspid aortic valve had a transthoracic echocardiogram done. The study showed the following:

**Table 8-7**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak velocity of mitral regurgitant jet</td>
<td>6.0 m/s</td>
</tr>
<tr>
<td>dP/dt of mitral regurgitant jet</td>
<td>1,900 mm Hg/s</td>
</tr>
<tr>
<td>Ratio of peak mitral E wave to peak velocity of medial mitral annulus (E/e')</td>
<td>16</td>
</tr>
<tr>
<td>Vena contracta of mitral regurgitation</td>
<td>0.2 cm</td>
</tr>
</tbody>
</table>

Systemic blood pressure at the time of study was 120/70 mm Hg. The following statement is TRUE:
A. Peak-to-peak aortic gradient is 90 mm Hg.
B. Patient is in cardiogenic shock due to left ventricular systolic dysfunction.
C. Mean left atrial pressure is approximately 20 mm Hg.
D. The size of vena contracta is diagnostic of severe mitral regurgitation.
E. Left atrial pressure cannot be estimated by E/e' method in patients with atrial fibrillation.

8. A 67-year-old man with aortic regurgitation underwent transthoracic echocardiographic examination. There was no mitral stenosis or regurgitation. The following values were obtained:

**Table 8-8**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak diastolic velocity of aortic regurgitant jet</td>
<td>5.0 m/s</td>
</tr>
<tr>
<td>End-diastolic velocity of aortic regurgitant jet</td>
<td>3.7 m/s</td>
</tr>
<tr>
<td>Pressure half-time of aortic regurgitant jet</td>
<td>656 ms</td>
</tr>
<tr>
<td>Peak aortic antegrade flow velocity</td>
<td>2.2 m/s</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>130/65 mm Hg</td>
</tr>
</tbody>
</table>

Based on the aforementioned data, one can conclude:
A. Patient should be advised against ASD closure because pulmonary hypertension is present.
B. Pulmonary vascular resistance is approximately 16 Wood units.
C. The ratio of pulmonary to systemic blood flow (Qp:Qs) is approximately 2.5:1.
D. Shunt flow is larger than the pulmonic flow (Qp).
E. Patient is cyanotic.

9. A 25-year-old woman is being evaluated for percutaneous closure of her secundum atrial septal defect (ASD). Transthoracic echocardiography demonstrated mild tricuspid regurgitation, no pulmonic stenosis, and the following:

**Table 8-9**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary artery systolic pressure</td>
<td>65 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery diastolic pressure</td>
<td>35 mm Hg</td>
</tr>
<tr>
<td>Left atrial pressure</td>
<td>10 mm Hg</td>
</tr>
<tr>
<td>Right ventricular outflow tract (RVOT) diameter</td>
<td>2.6 cm</td>
</tr>
<tr>
<td>RVOT velocity–time integral</td>
<td>30 cm</td>
</tr>
<tr>
<td>Left ventricular outflow tract (LVOT) diameter</td>
<td>2.0 cm</td>
</tr>
<tr>
<td>LVOT velocity–time integral</td>
<td>20 cm</td>
</tr>
<tr>
<td>Heart rate</td>
<td>75 beats/min</td>
</tr>
</tbody>
</table>

Based on the aforementioned data, one can conclude:
A. Pressure half-time is consistent with severe aortic regurgitation.
B. Aortic valve area can be estimated as 220 divided by pressure half-time.
C. Peak left ventricular systolic pressure (LVSP) is lower than the systolic blood pressure (SBP).
D. Left ventricular end-diastolic pressure (LVEDP) is estimated at 10 mm Hg.
E. Aortic valve area cannot be calculated using continuity equation because there is aortic regurgitation.

10. A 35-year-old woman was noted on clinical examination to have a systolic murmur and was referred for transthoracic echocardiography. The examination
revealed perimembranous ventricular septal defect (VSD), mild tricuspid regurgitation, pulmonic stenosis (PS), intact aortic valve, and the following:

Table 8-10

<table>
<thead>
<tr>
<th>Blood Pressure</th>
<th>120/80 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peak systolic velocity across the VSD</td>
<td>3.0 m/s</td>
</tr>
<tr>
<td>End-diastolic velocity across the VSD</td>
<td>1.0 m/s</td>
</tr>
<tr>
<td>Estimated right atrial pressure</td>
<td>10 mm Hg</td>
</tr>
<tr>
<td>Peak systolic gradient across pulmonic valve</td>
<td>55 mm Hg</td>
</tr>
<tr>
<td>Left-ventricular end-diastolic pressure</td>
<td>12 mm Hg</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Right ventricular systolic pressure (RVSP) is 46 mm Hg.
B. Pulmonary artery systolic pressure (PASP) is 29 mm Hg.
C. RVSP is 84 mm Hg above the right atrial pressure (RAP).
D. PASP is 45 mm Hg higher than the right ventricular systolic pressure.
E. Right ventricular end-diastolic pressure is 28 mm Hg.

11. A 21-year-old college student is noted to have fixed splitting of the second heart sound and right bundle branch block. Real-time three-dimensional transesophageal echocardiogram revealed a 1.2-cm secundum atrial septal defect (ASD) that was circular in shape. On color Doppler image, a well-formed hemispheric flow convergence (PISA) shell is seen on the left atrial side of the ASD. The following data were also obtained:

Table 8-11

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>120/80 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>100 beats/min</td>
</tr>
<tr>
<td>PISA radius</td>
<td>0.7 cm</td>
</tr>
<tr>
<td>Velocity–time integral of left-to-right flow across ASD</td>
<td>80 cm</td>
</tr>
<tr>
<td>Left ventricular outflow tract (LVOT) diameter</td>
<td>2.0 cm</td>
</tr>
<tr>
<td>LVOT velocity–time integral</td>
<td>19 cm</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Ratio of pulmonic to systemic flow (Qp:Qs) is 1.8 to 1.0.
B. Shunt flow (SF) across the ASD is approximately 9.0 L/min.
C. The difference between the pulmonic and systemic stroke volume is 180 mL.
D. Systemic stroke volume is 150 mL.
E. Pulmonic blood flow (Qp) is approximately 7.0 L/min.

12. A 35-year-old woman presents with sudden onset of dyspnea and pulmonary edema. She underwent bedside transthoracic echocardiography, which revealed hyperdynamic left ventricular systolic function, normal aortic valve, and mitral regurgitation. The following data were obtained from the transthoracic echocardiogram:

Table 8-12

<table>
<thead>
<tr>
<th>Blood pressure</th>
<th>95/50 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>120 beats/min</td>
</tr>
<tr>
<td>Peak velocity of mitral regurgitant jet</td>
<td>4.0 m/s</td>
</tr>
<tr>
<td>Time interval from onset of mitral regurgitation to jet velocity of 1 m/s</td>
<td>5 ms</td>
</tr>
<tr>
<td>Time interval from onset of mitral regurgitation to jet velocity of 3 m/s</td>
<td>25 ms</td>
</tr>
<tr>
<td>Vena contracta of mitral regurgitation</td>
<td>0.8 cm</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Peak velocity of the mitral inflow E wave is expected to be low.
B. Left atrial pressure is low.
C. Pulmonary venous flow velocity pattern on spectral Doppler is likely to reveal flow reversal during early diastole.
D. Rate of pressure rise (dP/dt) in the left ventricle is 1,600 mm Hg per second.
E. Left ventricular systolic function is markedly diminished.
13. A 29-year-old Bangladeshi woman with rheumatic mitral stenosis is referred to the cardiac catheterization laboratory for percutaneous mitral balloon valvuloplasty. Upon placement of the pigtail catheter in the left ventricle, the following values were obtained:

**Table 8-13**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular peak systolic pressure</td>
<td>124 mm Hg</td>
</tr>
<tr>
<td>Early left ventricular diastolic pressure</td>
<td>7 mm Hg</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure</td>
<td>10 mm Hg</td>
</tr>
</tbody>
</table>

Transesophageal echocardiogram prior to valvuloplasty revealed the absence of both mitral and aortic regurgitation, as well as the following:

**Table 8-14**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>104 beats/min</td>
</tr>
<tr>
<td>Time–velocity integral of diastolic mitral flow</td>
<td>65 cm</td>
</tr>
<tr>
<td>Mean mitral valve gradient in diastole</td>
<td>21 mm Hg</td>
</tr>
<tr>
<td>Mitral pressure half-time</td>
<td>270 ms</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Mean left atrial pressure is expected to be lower than the mean left ventricular diastolic pressure.
B. Peak velocity of the mitral inflow E wave is expected to be low.
C. Pressure half-time (PHT) may be unreliable in patients prior to valvuloplasty.
D. Mitral valve area (MVA) is 0.6 cm².
E. Mean left atrial pressure (LAP) is approximately 28 mm Hg.

14. An 81-year-old woman with systolic heart murmur was referred for an echocardiography. A heavily calcified aortic valve and normal mitral valve were noted on two-dimensional echocardiographic imaging. Doppler echocardiography of the aortic valve revealed:

**Table 8-15**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular outflow tract (LVOT) diameter</td>
<td>1.9 cm</td>
</tr>
<tr>
<td>Peak velocity across the aortic valve</td>
<td>5.0 m/s</td>
</tr>
<tr>
<td>Peak LVOT velocity</td>
<td>1.0 m/s</td>
</tr>
<tr>
<td>LVOT velocity–time integral (VTI)</td>
<td>20 cm</td>
</tr>
</tbody>
</table>

The following statement is TRUE:
A. Aortic valve area (AVA) cannot be calculated because aortic valve velocity–time integral is not stated.
B. Aortic valve stenosis is subvalvular.
C. Aortic valve area is likely to be less than 1 cm².
D. Left ventricular stroke volume (SV) is 80 mL per beat.
E. Systolic blood pressure is approximately 100 mg Hg above the left ventricular systolic pressure.

15. This continuous wave spectral Doppler tracing of the tricuspid regurgitant jet comes from an 18-year-old woman with pulmonic valve stenosis (Fig. 8-1). The peak pulmonic valve gradient is 24 mm Hg. Right atrial pressure is estimated at 10 mm Hg. The following is TRUE about this patient:
A. Peak pulmonary artery systolic pressure (PASP) is higher than the right ventricular peak systolic pressure.
B. Right ventricular peak systolic pressure is 64 mm Hg above the pulmonary artery peak systolic pressure.
C. Pulmonary artery peak systolic pressure is 50 mm Hg.
D. Right ventricular peak systolic pressure is 24 mm Hg less than the peak pulmonary artery systolic pressure (PASP).
E. Right ventricular peak systolic pressure is 108 mm Hg.
16. An 82-year-old man was referred for evaluation of a systolic ejection murmur. On parasternal long-axis view, the left ventricular outflow tract (LVOT) diameter was measured at 2.0 cm. Above spectral Doppler tracings were obtained in or through the LVOT in the apical 5-chamber view (Fig. 8-2).

The following statement is TRUE:
A. Increased cardiac output alone may explain the elevated gradient across the aortic valve.
B. Marked difference between the subvalvular and valvular velocities in this patient may also be seen in severe aortic regurgitation.
C. Patient has a very severe aortic valve stenosis with a mean gradient of approximately 60 mm Hg.
D. Aortic valve area (AVA) is greater than 1.0 cm$^2$.
E. Patient has hypertrophic obstructive cardiomyopathy (HOCM).

17. This continuous wave spectral Doppler tracing from a 21-year-old woman (Fig. 8-3) represents the flow velocity profile in the main pulmonary artery. Based on this tracing, the following is TRUE about this patient:
A. End-diastolic gradient across the pulmonic valve is high.
B. There is severe pulmonic valve stenosis.
C. Pulmonary artery systolic pressure is 9 mm Hg above the right ventricular pressure.
D. Pulmonic valve regurgitation is severe.
E. The velocity profile is diagnostic of patent ductus arteriosus.
18. The tracings (Fig. 8-4) were obtained from an 82-year-old woman with a normal left ventricular ejection fraction of 65%. **Figure 8-4A** represents blood flow velocity pattern obtained by placing a pulsed Doppler sample volume at the mitral leaflet tips. **Figure 8-4B** represents tissue Doppler of the lateral mitral annulus. Based on these two tracings, the following is TRUE:

A. The patient has excellent exercise capacity.
B. Abnormal left ventricular relaxation alone explains the mitral inflow pattern.
C. Left atrial pressure is elevated.
D. Patient has normal left ventricular diastolic function.
E. Mitral E wave velocity is expected to increase following the Valsalva maneuver.

---

19. **Figure 8-5A,B** were obtained from the same patient at the same heart rate.

The following statement is TRUE:

A. Mitral inflow pattern is diagnostic of restrictive filling.
B. Left ventricular end-diastolic pressure (LVDP) is elevated.
C. The higher the peak velocity of the atrial reversal wave in pulmonary veins, the lower the left ventricular pressure is.
D. The absence of atrial reversal wave in pulmonary vein tracings indicates pulmonary hypertension due to left ventricular dysfunction.
E. Ratio of peak systolic to peak diastolic velocity in pulmonary veins of more than 1 is indicative of elevated left atrial pressure.
**Chapter 8  Doppler and Hemodynamics / 117**

**MITRAL INFLOW**

Mitral A wave duration = 170 ms

*Figure 8-5A*

**PULMONARY VENOUS FLOW**

Atrial reversal wave
Duration = 210 ms; peak velocity 50 cm/s

*Figure 8-5B*

20. Upward deflection in respirometry recordings indicates inspiration, while the downward deflection indicates expiration (*Fig. 8-6*).

The following statement is TRUE:
A. There is no ventricular interdependence.
B. Expiratory increase in diastolic flow reversal in hepatic veins suggests constriction.
C. Abnormal interventricular septal motion is due to right ventricular volume overload.
D. An inspiratory increase in antegrade hepatic vein flow velocities is abnormal.
E. Above M-mode recordings are diagnostic of a large pericardial effusion and tamponade.

**M Mode Recording in Short Axis at Papillary Muscle Level**

*Figure 8-6 A: Hepatic vela pulsed Doppler*

*Figure 8-6B  Hepatic Vein Pulsed Doppler*
21. A 33-year-old man has had a murmur since childhood. The transthoracic spectral Doppler tracings in Figure 8-7 are obtained from the suprasternal view.

Which of the following statements is TRUE:
A. The pattern of diastolic flow is indicative of severe aortic regurgitation.
B. The tracings are diagnostic of aortic coarctation.
C. Quadricuspid aortic valve is the most common cause of aortic stenosis associated with the above flow velocity pattern.
D. The recordings are obtained from the ascending aorta and represent severe aortic stenosis.
E. Patient’s blood pressure in the legs is markedly higher than that in the arms.

---

22. A 91-year-old woman presents with severe shortness of breath. Above two spectral Doppler recordings were obtained from two different valves. Vertical line in each tracing marks the onset of QRS.

The following statement is TRUE:
A. Figure 8-8B represents tricuspid regurgitant jet and the patient has severely elevated right ventricular systolic pressure.
B. Figure 8-8A represents severe aortic stenosis because the jet starts during isovolumic contraction period.
C. The jet with the shorter duration represents aortic stenosis.

D. Peak velocity of 5.0 m/s in Figure 8-8B is not compatible with a tricuspid regurgitant jet.
E. Systolic function of both ventricles is severely diminished.
23. A 55-year-old man with hypertension treated with a beta blocker, and advanced gastric carcinoma, presents with sudden onset of severe shortness of breath. The spectral pulsed Doppler recordings in Figure 8-9 were obtained at the mitral leaflet tips. Upward deflection in respirometry recordings above indicates inspiration, while the downward deflection indicates expiration.

The following statement is TRUE:
A. Respiratory variations in peak velocity of late diastolic flow (A wave) of more than 25% favor constriction over tamponade.
B. A marked decrease in peak E wave velocity seen at the onset of inspiration is consistent with the diagnosis of tamponade.
C. Findings are characteristic of restrictive cardiomyopathy.
D. The ratio of early to late diastolic peak mitral velocity (E/A ratio) of less than 1 favors the diagnosis of constrictive pericarditis.
E. Treatment with diuretics would markedly improve patient’s shortness of breath.

24. A 28-year-old man with liver disease presents with jugular venous distension (Fig. 8-10).

The following statement is TRUE:
A. Right atrial pressure (RAP) rises progressively toward the end of ventricular systole.
B. Right ventricular systolic function is markedly diminished.
C. Peak velocity of 2.2 m/s excludes the diagnosis of pulmonary hypertension.
D. Tricuspid regurgitation is likely mild.
E. There is right ventricular midcavitary gradient during systole.

Peak velocity of tricuspid regurgitant jet = 2.2 m/s

Figure 8-10
25. A 59-year-old man was diagnosed with perimembranous VSD by transthoracic echocardiography. His blood pressure is 90/45 mm Hg and his end-diastolic right atrial pressure is estimated at 10 mm Hg. There was no aortic or tricuspid valve stenosis. The continuous wave spectral Doppler flow pattern (Fig. 8-11) was obtained across his VSD. What is this patient's left ventricular end-diastolic pressure (LVEDP)?
A. 39 mm Hg.
B. 6 mm Hg.
C. 19 mm Hg.
D. 26 mm Hg.
E. 35 mm Hg.

26. A 55-year-old Vietnamese woman presents with progressive dyspnea on exertion, increased abdominal girth, and bilateral pitting edema over the past several months. The mitral annular tissue Doppler tracings were obtained (Fig. 8-12). These tissue Doppler recordings are most consistent with the following diagnosis:
A. Carcinoid heart disease.
B. Amyloidosis.
C. Constrictive pericarditis.
D. Mitral annular calcifications.
E. Flail posterior mitral leaflet.

27. A 29-year-old woman on transesophageal echocardiography in preparation to have a secundum atrial septal defect (ASD) closure was found to have a left-to-right shunt with this continuous wave Doppler flow pattern (Fig. 8-13). Her right atrial pressure was estimated at 8 mm Hg. What is the peak left atrial pressure in this patient?
A. 1 mm Hg.
B. 17 mm Hg.
C. 21 mm Hg.
D. 36 mm Hg.
E. 52 mm Hg.
28. A 49-year-old man was referred for transthoracic echocardiogram for evaluation of a murmur. The continuous wave Doppler tracing across the mitral valve (Fig. 8-14) was obtained from the apical 4-chamber view. In addition, the effective regurgitant orifice area (EROA) of mitral regurgitation is calculated at 0.48 cm², left atrial pressure is estimated at 10 mm Hg, and no aortic stenosis is present. Regarding this patient, which of the following statements is correct:

A. Mitral regurgitation occurs only in early systole.
B. EROA accurately reflects the severity of mitral regurgitation in this patient.
C. Patient is hypotensive at the time of the study.
D. Regurgitant fraction of mitral regurgitation is 60%.
E. Regurgitant volume of this patient is indicative of moderate (2+) mitral regurgitation.

29. A 35-year-old man who grew up in Venezuela reports a history of rheumatic fever in his childhood. He was referred for transthoracic echocardiogram for evaluation of a murmur. The continuous wave spectral Doppler tracing (Fig. 8-15) across the mitral valve was obtained in the apical 4-chamber view. Based on this spectral Doppler tracing, which is the correct diagnosis?

A. Mitral stenosis due to supravalvular ring.
B. Shone complex.
C. Rheumatic mitral stenosis.
D. Cor triatriatum.
E. Subvalvular mitral stenosis.

CASE 1

A 78-year-old obese woman with history of hypertension and poorly controlled diabetes mellitus developed progressive chest pain and shortness of breath for the past 2 days. She had no prior history of coronary revascularization or heart surgery. Her son brought her to the emergency department where she was noted to be diaphoretic and tachypneic.

Electrocardiogram in the emergency department revealed normal sinus rhythm, right bundle branch block, and ST elevations in anteroseptal leads.

Blood pressure 90/50 mm Hg; heart rate 100 beats/min; oral temperature 98.7 degrees. On auscultation of the lungs, rales were noted bilaterally throughout the lung fields. The heart examination revealed prominent S3 and no murmur. Serum troponin was elevated at 40 ng/mL (normal <5 ng/mL). There was marked pulmonary edema on chest x-ray film.

Transthoracic echocardiogram at the time of presentation revealed hypokinesis of six left ventricular segments supplied by the left anterior descending artery; ejection fraction was estimated at 40%. There was mild regurgitation of a structurally normal native mitral valve.

The patient was transferred to the intensive care unit where a Swan–Ganz catheter was placed. Pulmonary artery wedge pressure was 38 mm Hg. Tissue Doppler of the medial mitral annulus and pulsed Doppler recordings with the sample volume at the tips of the mitral valve leaflets were obtained at that time. Patient was in normal sinus rhythm. Peak velocity of the early annular tissue Doppler wave (e′) was 5 cm/s.
30. Which of the following mitral flow velocity patterns is the most likely at this time?
A. Figure 8-16A.
B. Figure 8-16B.
C. Figure 8-16C.
D. Figure 8-16D.
E. Figure 8-16E.

Peak E wave velocity = 45 cm/s
Figure 8-16A

Peak E wave velocity = 60 cm/s
Figure 8-16B

Peak E wave velocity = 150 cm/s
Figure 8-16C

Peak E wave velocity = 200 cm/s
Figure 8-16D
31. From the emergency department, she was taken for coronary angiography, which revealed total occlusion of the proximal left anterior descending artery and diffuse atherosclerosis in the left circumflex artery. Percutaneous coronary intervention was attempted, but the stent could not be deployed in the left anterior descending artery. She was then transferred to the intensive care unit. After appropriate medical therapy, she was discharged home free of symptoms on hospital day 5.

Three days later, she collapsed. Her neighbor called 911 and the patient was intubated in the field for severe hypoxemia. On admission she was afebrile. Laboratory data revealed normal white blood cell count. Chest x-ray film in the emergency department demonstrated massive bilateral pulmonary edema. The following data were obtained by echocardiography the same day (Fig. 8-17 and Video 8-1).

The degree of mitral regurgitation is:
A. Trivial.
B. Mild (1+).
C. Moderate (2+).
D. Moderate to severe (3+).
E. Severe (4+).

32. The most likely etiology of mitral regurgitation in this patient is:
A. Papillary muscle rupture.
B. Bacterial endocarditis.
C. Mitral annular dilatation.
D. Rheumatic heart disease.
E. Mitral valve prolapse.

CASE 2

A 56-year-old man, a recent immigrant from Argentina, has been an avid soccer player since childhood. He reports that over the past year or so, he no longer can run around the soccer field as he used to because of exertional dyspnea. He initially saw a pulmonary specialist who ruled out exercise-induced asthma.

On examination, his blood pressure is 170/70 mm Hg; heart rate 72 beats/min with a regular rhythm; room air oxygen saturation by pulse oxymetry 98%. He has no central or peripheral cyanosis. His lungs are clear. First heart sound (S₁) is normal, while the second heart sound (S₂) is obscured by the continuous, machinery-type murmur best heard in the left upper chest. There is no peripheral edema.

Echocardiography revealed patent ductus arteriosus, normal left ventricular systolic function, no valvular disease, and no hypertrophic cardiomyopathy. Right atrial pressure is estimated at 10 mm Hg.
33. The spectral Doppler tracing in Figure 8-18 represents flow across the patent ductus arteriosus obtained by transthoracic echocardiography. The following statement is TRUE:
A. Pulmonary artery diastolic pressure is 21 mm Hg above the right atrial pressure.
B. The tracing was obtained by pulsed wave Doppler technique.
C. Pulmonary artery pressure is estimated at 26/12 mm Hg.
D. Pulmonary artery systolic pressure is 110 mm Hg.
E. Patent ductus arteriosus is very large because the flow occurs throughout the cardiac cycle.

34. This transthoracic echocardiographic color Doppler image in the parasternal short-axis view at the level of the patent ductus arteriosus comes from the same study as the spectral tracing in previous question (Fig. 8-19).
Using the PISA method, the cross-sectional area of the patent ductus arteriosus at its aortic end during maximum flow is:
A. 0.01 cm².
B. 0.13 cm².
C. 0.22 cm².
D. 1.3 cm².
E. 2.2 cm².

CASE 3
A 24-year-old college athlete collapsed on the basketball court. The coach promptly used the automatic external defibrillator, which delivered an appropriate shock and revived the patient. The patient was then brought to the emergency department.
On physical examination, he was lying comfortably in bed, fully awake, alert, and oriented. Blood pressure 144/72 mm Hg; heart rate 64 beats/min. Lungs were clear on auscultation. Cardiac examination revealed a crescendo-decrescendo systolic ejection murmur along the left sternal border, which increased with Valsalva maneuver. The carotid upstroke was brisk and there was a bisferiens pulse.

35. Transthoracic echocardiogram performed in the emergency department demonstrated hypertrophic cardiomyopathy with asymmetric septal hypertrophy, systolic anterior motion, and normal left ventricular systolic function. Aortic valve was normal. Left atrial pressure was estimated at 10 mm Hg. There was eccentric mitral regurgitation; the spectral Doppler of the mitral regurgitant jet is depicted in Figure 8-20.
The following statement is TRUE:
A. Envelope of the mitral regurgitant jet is not fully recorded because the early systolic portion of the jet is missing.
B. Left ventricular systolic pressure is low.
C. Maximal instantaneous left ventricular outflow gradient is 122 mm Hg.
D. Mitral regurgitation is partly diastolic.
E. Peak left ventricular systolic pressure is 246 mm Hg.

B. Flow velocity pattern of jet #2 is typical of valvular aortic stenosis.
C. Left ventricular outflow gradient has dropped by about 50% compared to the initial echocardiogram.
D. Patient has developed intracavitary gradient as demonstrated by jet #1.
E. Peak left ventricular systolic pressure is now 159 mm Hg minus the left atrial pressure.

**CASE 4**

A 66-year-old man with a long-standing history of ethanol abuse complains of orthopnea, paroxysmal nocturnal dyspnea, and lower extremity edema. He is tachypneic and tachycardic. Blood pressure 90/50 mm Hg, heart rate 110 beats/min; weight 80 kg; height 175 cm; body surface area 2.0 m². Auscultation of the lungs reveals bibasilar rales. Cardiac examination demonstrates an S₃ gallop and no murmur. There is bilateral lower extremity pitting edema pretibially. Transthoracic echocardiogram revealed global left ventricular hypokinesis with an estimated ejection fraction of 25%.
37. To calculate the left atrial volume, the data in Figure 8-22 were obtained:

The left atrial volume index is approximately:
A. 20 mL/m\(^2\).
B. 30 mL/m\(^2\).
C. 40 mL/m\(^2\).
D. 50 mL/m\(^2\).
E. 60 mL/m\(^2\).

38. Mitral inflow and pulmonary venous flow velocity spectral Doppler tracings were obtained on admission and after 5 days of appropriate medical therapy including intravenous diuretics (Fig. 8-23). The following was the result of what medical therapy?

A. Left ventricular preload has increased.
B. Left atrial pressure has decreased.
C. Normal mitral filling pattern was replaced with the pattern of abnormal relaxation.
D. Patient has developed atrial flutter.
E. The change in mitral filling pattern seen in this patient portends grave long-term prognosis.
CASE 5

A 23-year college student came back to the United States from an extended trip to rural areas of the Indian subcontinent, complaining of dyspnea on exertion and chest pain on deep inspiration.

On initial outpatient examination, he was afebrile. His lungs were clear on auscultation. There was a friction rub throughout the precordium. Electrocardiogram was suggestive of pericarditis (Fig. 8-24).

He was prescribed an oral course of a nonsteroidal anti-inflammatory drug (NSAID) and sent home.

Despite taking the NSAID for 2 weeks, there was worsening in his chest pain. Computed tomography of the chest revealed a large pericardial and left pleural effusion with clinical and echocardiographic signs of tamponade. Skin test for tuberculosis (PPD) was positive. Pericardial effusion was drained percutaneously, and the patient was started on appropriate antituberculosis medical therapy.

His chest pain resolved completely. However, his shortness of breath persisted and he started developing bilateral ankle edema. Transthoracic echocardiogram was ordered.

39. Figure 8-25 was also obtained on the echocardiogram.

In the aforementioned recordings, the upstroke of the respirometer curve denotes inspiration, and the downstroke indicates expiration. The following is true:
A. Restrictive cardiomyopathy of the left ventricle is present.
B. Right atrial pressure is low.
C. Left ventricular flow propagation velocity ($V_p$) is abnormal.
D. Patient has constrictive pericarditis.
E. Degree of respiratory variations in the mitral inflow is normal.

40. Video 8-2, obtained in the apical 4-chamber view, demonstrates abnormal septal motion which is due to:
A. Right ventricular pressure overload.
B. Right ventricular volume overload.
C. Left bundle branch block.
D. Ventricular interdependence.
E. Cardiac surgery.
ANSWERS

1. **Answer: D.** Mitral valve area (MVA) can be calculated using the pressure half-time (PHT) method:

   \[ MVA = \frac{220}{PHT} \]  

   In this question, PHT was not given. However, PHT can be calculated from the stated mitral deceleration time (DT) using the following formula:

   \[ PHT = 0.29 \times DT \]

   Thus, in our patient:

   \[ PHT = 0.29 \times DT = 0.29 \times 910 = 264 \text{ ms} \]

   \[ MVA = \frac{220}{264} = 0.8 \text{ cm}^2 \]

   Alternatively, Eqs. 1 and 2 can be combined into the following one:

   \[ MVA = \frac{759}{DT} \]

   In our patient, then:

   \[ MVA = 759/DT = 759/910 = 0.8 \text{ cm}^2 \]

   Therefore, answer D is correct.

   **Answer A** is incorrect because MVA is calculated by dividing 220 into PHT (Eq. 1) and not DT.

   **Answer B** is incorrect because the stroke volume (SV) across the mitral valve in this patient is 53 mL per beat. Once the MVA is calculated, SV and cardiac output (CO) can be derived using the following formulas:

   \[ SV = MVA \times VTI \]

   \[ CO = SV \times HR \]

   where VTI is the mitral velocity–time integral during diastole, and HR is the heart rate.

   In our patient, mitral VTI during diastole was 66 cm and the heart rate was 85 beats/min:

   \[ SV = 0.8 \text{ cm}^2 \times 66 \text{ cm} = 53 \text{ mL} \]

   \[ CO = 53 \text{ mL} \times 85 \text{ beats/min} = 4.5 \text{ L/min} \]

   **Answer C** is incorrect because, as shown previously, PHT in this patient was 264 ms and not 355 ms.

   **Answer E** is incorrect because the resting gradient of mitral stenosis is expected to increase with augmentation of CO such as during exercise, fever, or pregnancy.

2. **Answer: E.** The patient has patent ductus arteriosus (PDA) that is an extracardiac shunt resulting from a communication between the descending thoracic aorta (DTA) and the proximal left pulmonary artery. In utero, the blood that reaches the pulmonary artery from the right ventricle cannot enter the collapsed lungs; instead, it is diverted across the ductus arteriosus into the DTA. Soon after birth, the pressure in the pulmonary artery falls below the pressure in DTA and the blood flow in the ductus arteriosus reverses its direction. It now flows from the DTA into the pulmonary artery. High oxygen content of the ductal blood triggers the closure of ductus arteriosus in most newborns. In rare instances, the communication persists in the post-neonatal period giving rise to PDA.

   In individuals with PDA, the systemic blood flow (Qs) reaches the right-sided heart through systemic veins and continues through the right ventricular outflow tract (RVOT) into the main pulmonary artery. At that level, Qs is joined by the shunt flow (SF) entering the pulmonary artery through the PDA. The sum of Qs and SF represents the amount of blood flow that enters the pulmonary circulation (Qp).

   After passing through the lungs, Qp enters the left-sided heart through the pulmonary veins and exits through the left ventricular outflow tract (LVOT) into the aorta. At the level of the descending aorta, Qp divides into SF, which enters the PDA, and Qs, which continues into the peripheral systemic circulation to ultimately reach the right-sided heart through systemic veins.

   Note that in individuals with PDA, the flow across the RVOT represents Qs and the flow across the LVOT represents Qp. Therefore, the answer E is correct.

   This is in contrast to atrial and ventricular septal defects where LVOT flow represents Qs and the RVOT flow represents Qp. Since in most individuals with PDA, Qp > Qs, it is the left side of the heart and not the right side of the heart that dilates to accommodate the excess blood flow.

   The general echocardiographic formula to calculate volumetric flow (Q) is:

   \[ Q = CSA \times VTI \times HR \]  

   where CSA is the cross-sectional area, VTI is velocity–time integral, and HR is the heart rate.

   One can use right and left ventricular outflow tracts to calculate volumetric flow. Since both tracts are assumed to be circular in shape, the CSA can be expressed in the above equations as follows:

   \[ CSA = \left( \frac{1}{2} \times D \right)^2 \times \pi \]

   where D is the diameter of the outflow tract. Eq. 1, after expressing CSA in terms of Eq. 2, becomes:

   \[ Q = \left( \frac{1}{2} \times D \right)^2 \times \pi \times VTI \times HR \text{ seconds} \]

   Calculations for our patient are summarized in the following table:
where \( V \) is the end-diastolic velocity of the pulmonic regurgitant jet and RAP is right atrial pressure.

RAP can be estimated from the expiratory size of the inferior vena cava (IVC) and the percent decrease in diameter change with inspiration. In our patient, the IVC is dilated (>1.7 cm) and the IVC diameter does not change with inspiration. The estimated RAP is thus greater than 15 mm Hg.

Once RAP is known, we can then calculate PADP:

\[
\text{PADP} > 4 \times (2 \text{ m/s})^2 + 15, \text{ or greater than } 31 \text{ mm Hg}
\]

Therefore, answer B is correct.

Answer A is incorrect because RAP in this patient is >15 mm Hg as demonstrated earlier.

Answer C is incorrect for two reasons: (1) Pressure gradient between PADP and RVDP is 16 mm Hg and not 36 mm Hg and (2) PADP is calculated by adding RAP to the gradient between PADP and RVDP, and not subtracting from it.

Answer D is incorrect because even in mild pulmonic regurgitation, appropriate spectral Doppler tracings of the regurgitant jet can be obtained.

Answer E is incorrect because normal PADP range is typically between 5 and 16 mm Hg.

4. Answer: B. Severe mitral regurgitation (grades 3+ and 4+) is defined by the following criteria:

<table>
<thead>
<tr>
<th>Table 8-16</th>
<th>LVOT</th>
<th>RVOT</th>
<th>Shunt Across PDA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter (cm)</td>
<td>2.0</td>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>Area (cm²)</td>
<td>3.1</td>
<td>4.9</td>
<td></td>
</tr>
<tr>
<td>VTI (cm)</td>
<td>31</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>97</td>
<td>59</td>
<td>97 – 59 = 38</td>
</tr>
<tr>
<td>HR</td>
<td>80</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>Flow (L/min)</td>
<td>7.8</td>
<td>4.7</td>
<td></td>
</tr>
<tr>
<td>Qp:Qs</td>
<td>1.7</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Answer A is incorrect because the flow rate of 7.8 L/min across the LVOT represents Qp and not Qs in patients with PDA.

Answer B is incorrect because Qp:Qs in this patient is greater than 1 (it is 1.7:1).

Answer C is incorrect because the stroke volume that enters the lungs (97 mL per beat) is the sum of the systemic stroke volume (59 mL per beat) that entered the main pulmonary artery through the RVOT and the shunt flow (38 mL per beat) that came into the pulmonary artery through the PDA.

Answer D is incorrect because Qp is much greater than Qs, the SF is in the left to right direction and the patient is unlikely to be cyanotic. In patients with PDA who develop Eisenmenger physiology, there is a right to left shunt. Such patients are cyanotic in the lower parts of the body because the deoxygenated blood from the pulmonary artery crosses the PDA and enters the DTA past the origins of the aortic arch vessels, which supply a fully oxygenated blood to the head and the arms.

3. Answer: B. This patient with severe shortness of breath has elevated pulmonary artery diastolic pressure (PADP). Using the end-diastolic velocity (\( V \)) of the pulmonic regurgitant jet and the \( 4V^2 \) formula, one can calculate the pressure gradient (\( \Delta P \)) between the PADP and the end-diastolic right ventricular pressure (RVDP).

\[
\Delta P = \text{PADP} - \text{RVDP} = 4 \times V^2 \quad \text{(1)}
\]

In the absence of tricuspid stenosis, RVDP is the same as the right atrial pressure (RAP). Thus the pressure gradient can also be expressed as:

\[
\Delta P = \text{PADP} - \text{RAP} = 4 \times V^2 \quad \text{(2)}
\]

Rearranging Eq. 2, PADP can be calculated in the following manner:

\[
\text{PADP} = 4 \times v^2 + \text{RAP} \quad \text{(3)}
\]

Regurgitant orifice area (ROA) can be calculated using the following formula:

\[
\text{ROA}_{\text{MR}} = 2 \times \pi \times r^2 \times \frac{V_{\text{alias}}}{V_{\text{max}}} \quad \text{(1)}
\]

where \( r \) is the PISA radius, \( V_{\text{alias}} \) is the aliasing velocity at which PISA radius is measured, and \( V_{\text{max}} \) is the maximum velocity of the mitral regurgitant (MR) jet on spectral Doppler.

In Eq. 1, the expression \( 2 \times \pi \times r^2 \times V_{\text{alias}} \) represents instantaneous flow rate (IFR):

\[
\text{IFR} = 2 \times \pi \times r^2 \times V_{\text{alias}} \quad \text{(2)}
\]

Now Eq. 1 can be expressed as follows:

\[
\text{ROA}_{\text{MR}} = \frac{\text{IFR}}{V_{\text{max}}} \quad \text{(3)}
\]
In our patient, IFR is calculated as follows:

\[
IFR = 2 \times 3.14 \times (1.0 \text{ cm})^2 \times 45 \text{ cm/s} = 283 \text{ mL/s}
\]

And ROA as:

\[
ROA_{\text{MR}} = \frac{283}{500 \text{ cm/s}} = 0.6 \text{ cm}^2
\]

Therefore, answer B is correct.

Answer A is incorrect because the vena contracta in severe mitral regurgitation is ≥0.7 cm.

Answer C is incorrect because the IFR of the MR jet in this patient is 283 mL per second as calculated as earlier.

Answer D is incorrect because mitral regurgitation is severe since ROA ≥0.4 cm² (it is 0.6 cm²).

Answer E is incorrect because the regurgitant volume (RegV) in this patient is 79 mL per beat. RV can be calculated as follows:

\[
\text{RegV} = ROA_{\text{MR}} \times \text{VTI}_{\text{MR}} (4)
\]

where VTI_{MR} is the velocity–time integral of the MR jet.

In our patient, RV equals 0.6 cm² × 140 cm, or 79 mL per beat. This is again consistent with severe mitral regurgitation (RV ≥60 mL per beat).

**5. Answer: A.** The patient presents with shortness of breath due to elevated pulmonary artery wedge pressure (PAWP). In most instances, PAWP elevation is the result of high left atrial pressure (LAP) elevation.

PAWP can be estimated from the following formula:

\[
\text{PAWP} = 4.6 + 5.27 \times \frac{E}{V_p}
\]

where \( E \) is the peak blood flow velocity of the mitral inflow in cm/s and \( V_p \) is the flow propagation velocity of the mitral inflow (in cm/s) obtained by color M-mode. \( V_p \) recording of this patient is demonstrated in Figure 8-26.

Vp measures the rate at which red blood cells reach the LV apex from the mitral valve level during early diastole. The rate of blood flow from the mitral valve to the LV apex is determined by the rate of LV relaxation during early diastole. Therefore, \( V_p \) is an indirect measure of the rate of LV relaxation; the lower the \( V_p \), the slower the LV relaxation and higher the left ventricular diastolic pressure (LVDP) are.

In our patient:

\[
\text{PAWP} = 4.6 + 5.27 \times \frac{125}{31} = 26
\]

With the value of 26 mm Hg, PAWP is elevated; normal PAWP is ≤12 mm Hg. Therefore, answer A is correct.

Answer B is incorrect because in patients with markedly elevated LAP and PAWP, the peak velocity of the mitral E wave is typically higher than that of the mitral A wave. The patients have either the pseudonormal filling pattern (E/A is between 1.0 and 2.0; E wave deceleration time ≥160 ms) or the restrictive filling pattern (E/A >2 and E wave deceleration time <160 ms).

Answer C is incorrect because the pulmonary artery systolic pressure (PASP) is 64 mm Hg plus the right atrial pressure, or 64 + 15 = 79 mm Hg. In the absence of pulmonic stenosis, PASP is the same as the right ventricular systolic pressure (RVSP). Peak velocity (V) of the tricuspid regurgitant flow can be used to estimate the RV-to-RA pressure gradient (ΔP) at peak systole:

\[
\Delta P = 4 \times V^2 = (4 \text{ m/s})^2 = 64 \text{ mm Hg}
\]

By adding RAP to ΔP, RVSP (and, by extension, PASP) can be calculated as follows:

\[
\text{RVSP} = \text{PASP} = \Delta P + \text{RAP} = 64 + 15 = 79 \text{ mm Hg}
\]

Answer D is incorrect because the ratio of mitral E wave to mitral annular tissue Doppler e’ wave is expected to be greater than 15 in patients with markedly elevated LAP and PAWP.

Answer E is incorrect because the normal \( V_p \) velocity >55 cm/s in young individuals, and >45 cm/s in middle-aged and elderly individuals.

**6. Answer: A.** Severe aortic regurgitation (grades 3+ and 4+) is defined by the following criteria:

<table>
<thead>
<tr>
<th>Table 8-18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe AR</td>
</tr>
<tr>
<td>Regurgitant orifice (cm²) ≥0.3</td>
</tr>
<tr>
<td>Regurgitant fraction ≥50%</td>
</tr>
<tr>
<td>Regurgitant volume (mL) ≥60</td>
</tr>
<tr>
<td>Vena contracta (cm) ≥0.6</td>
</tr>
</tbody>
</table>
Therefore, answer A is correct; the regurgitant fraction of 65% indicates a severe aortic regurgitation.

Answer B is incorrect because vena contracta is not strongly influenced by Nyquist limit color Doppler settings. This is in contrast to PISA radius. By changing the color Doppler Nyquist limit, one also automatically changes the velocity filter. The role of the velocity filter is to prevent color encoding of low velocities. By lowering the color Doppler Nyquist limit, one lowers the velocity filter allowing for inclusion of lower velocities and an increase in the color area. Because vena contracta contains predominantly high velocities, altering the Nyquist limit will not change significantly the size of vena contracta diameter. This is in contrast to PISA radius, which becomes progressively larger with lower Nyquist limits.

The impact of changes in color Doppler Nyquist limit on vena contracta is demonstrated in Figure 8-27.

Answer C is incorrect because in severe aortic regurgitation, vena contracta is ≥0.3 cm.

Answer D is incorrect because in severe aortic regurgitation, regurgitant volume is ≥60 mL per beat.

Answer E is incorrect because the diameter of vena contracta obtained by two-dimensional echocardiography should not be used to calculate the regurgitant volume. Instead, the two-dimensional diameter of vena contracta should be used for semiquantitative assessment of the degree of aortic regurgitation.

7. **Answer: C.** The E/e’ ratio is directly proportional to the left atrial pressure (LAP). The peak velocity of the mitral annular tissue Doppler e’ wave is directly proportional to the rate of LV relaxation during early diastole. The slower the LV relaxation, the higher the left ventricular diastolic pressure (LVEDP) is. Once LVEDP rises, there is a concomitant rise in the LAP and PAWP rise to allow for better filling of a stiff LV. The higher the LAP, the taller the mitral E wave becomes. In summary, as the LV diastolic dysfunction worsens, the peak velocity of the annular tissue e’ wave gets smaller, the mitral E wave gets higher, and the E/e’ ratio becomes progressively larger reflecting the rising LAP and PAWP.

<table>
<thead>
<tr>
<th>Table 8-19</th>
<th>Left Atrial Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>E/e’ using medial e’</td>
<td>&lt;8</td>
</tr>
<tr>
<td>E/e’ using lateral e’</td>
<td>8–12</td>
</tr>
</tbody>
</table>

The E/e’ ratio can be used to estimate LAP in two ways. One approach is to use it semiquantitatively as shown in table below.
Thus, by E/e′ ratio of 16 alone, our patient has an elevated LAP. The other approach is to estimate LAP numerically using the following equation:

\[ \text{LAP} = 1.9 + 1.24 \times \frac{E}{e'} \]  

(1)

In our patient:

\[ \text{LAP} = 1.9 + 1.24 \times 16 = 22 \]

An LAP of 22 mm Hg is significantly elevated; normal LAP is <12 mm Hg.

A simplified form of Eq. 1 is:

\[ \text{LAP} = 4 + \frac{E}{e'} \]  

(2)

In our patient, LAP can be estimated by Eq. 2 as 4 + 16, or 20 mm Hg.

Therefore, answer C is correct.

Answer A is incorrect because the peak-to-peak gradient of aortic stenosis in this patient is 44 mm Hg.

To calculate the peak-to-peak gradient of aortic stenosis, we first need to calculate the peak left ventricular systolic pressure (LVSP), using the following formula:

\[ \text{LVSP} = \Delta P_{\text{MR}} + \text{LAP} \]  

(3)

where \( \Delta P_{\text{MR}} \) is the peak systolic gradient of the mitral regurgitant jet and LAP is the left atrial pressure. After expressing \( \Delta P_{\text{MR}} \) in terms of the peak velocity (V) of the MR jet, Eq. 1 becomes:

\[ \text{LVSP} = 4 \times V^2 + \text{LAP} \]  

(4)

In our patient:

\[ \text{LVSP} = 4 \times (6.0 \text{ m/s})^2 + 20 = 164 \text{ mm Hg} \]

Once LVSP is known, the peak-to-peak aortic gradient (P2P) can be calculated as follows:

\[ \text{P2P} = \text{LVSP} - \text{SBP} \]  

(5)

where SBP is the systolic blood pressure.

In our patient:

\[ \text{P2P} = 164 - 120 = 44 \text{ mm Hg} \]

It is important to emphasize that this pressure gradient, which is commonly measured on cardiac catheterization, is not a physiologic one because it represents a pressure difference at separate points in time as demonstrated in Figure 8-28. P2P is lower than the peak instantaneous gradient obtained by continuous wave Doppler across the aortic valve.

Answer B is incorrect because left ventricular dP/dt is normal. Patients with cardiogenic shock have low dP/dt values. Normal dP/dt = 1,661 + 323 mm Hg per second.

Answer D is incorrect because in severe mitral regurgitation vena contracta of \( \geq 0.7 \) cm.

Answer E is incorrect because either Eq. 1 or Eq. 2 is applicable irrespective of the atrial rhythm (normal sinus rhythm, atrial fibrillation, etc.).

8. Answer: D. Figure 8-29 shows the continuous wave spectral Doppler tracings of our patient.

Using the end-diastolic velocity (V) of the aortic regurgitant jet, one can calculate the pressure gradient (\( \Delta P \)) between the diastolic blood pressure (DBP) and the LVEDP.

\[ \Delta P = \text{DBP} - \text{LVEDP} = 4 \times V^2 \]  

(1)
Rearranging Eq. 1, LVEDP can be calculated in the following manner if the DBP is known:

\[
LVEDP = DBP - 4 \times V^2
\]  

(2)

In our patient:

\[
LVEDP = 65 \text{ mm Hg} - 4 \times (3.7 \text{ m/s})^2 = 10 \text{ mm Hg}
\]

Therefore, answer D is correct.

Answer A is incorrect because in severe aortic regurgitation pressure half-time is < 300 ms.

Answer B is incorrect because the aortic valve (AV) area cannot be calculated by 220 into pressure half-time; that is the formula for calculating the mitral valve area.

Answer C is incorrect because the peak LVSP is always higher than the SBP in patients with aortic stenosis. LVSP becomes progressively higher than SBP as the aortic stenosis becomes more severe. The LVSP-to-SBP pressure gradient is referred to as the peak-to-peak aortic gradient as discussed in answer to Question 8.

Answer E is incorrect because the continuity equation can be used to calculate the AV area in patients with or without aortic regurgitation. The continuity principle states that the stroke volume across the left ventricular outflow tract (LVOT) is the same as the stroke volume across the AV:

\[
\text{LVOT stroke volume} = \text{AV stroke volume}
\]  

(3)

Since stroke volume can be expressed as the product of the cross sectional area (CSA) and the flow velocity integral (VTI), Eq. 5 becomes:

\[
\text{CSA}_{\text{LVOT}} \times \text{VTI}_{\text{LVOT}} = \text{CSA}_{\text{AV}} \times \text{VTI}_{\text{AV}}
\]  

(4)

In patients with aortic regurgitation, there is an increase in antegrade flow from the left ventricle into the aorta due to augmentation of the true left ventricular stroke volume by the aortic regurgitant volume. However, this increase equally affects the flow through the LVOT and the AV in systole. In Eq. 6, this will be reflected in a proportional increase in VTI_{LVOT} and VTI_{AV}.

By continuity equation, aortic valve area (CSA_{AV}) can be calculated as follows:

\[
\text{CSA}_{\text{AV}} = \text{CSA}_{\text{LVOT}} \times \frac{\text{VTI}_{\text{LVOT}}}{\text{VTI}_{\text{AV}}}
\]  

(5)

In aortic regurgitation, there is augmentation of VTI_{LVOT} and VTI_{AV}. However, the ratio of the two VTIs remains the same, and therefore the calculated value of CSA_{AV} is not affected by the presence of aortic regurgitation.

9. Answer: C. Patient has an atrial septal defect (ASD) with a left-to-right shunt. ASD is an intracardiac shunt at the atrial level. Systemic blood flow (Q_s) reaches the right atrium through systemic veins. At the level of the right atrium, it is joined by the shunt flow, which enters the right atrium from the left atrium across the ASD. The sum of Q_s and the shunt flow then passes through the right ventricular outflow tract (RVOT) into the pulmonary circulation. Therefore, the sum of Q_s and the shunt flow represents the pulmonary blood flow (Q_p). This Q_p reaches the left atrium through the pulmonary veins. At the left atrial level, Q_p divides into shunt flow (which traverses ASD to reach the right atrium), and Q_s, which enters the left ventricle. Q_s then passes through the LVOT into the aorta and eventually reaches the right atrium through systemic veins.

In summary, flow through LVOT represents Q_s while the flow through RVOT represents Q_p in patients with ASD.

Shunt calculations for this patient are summarized in the following table:

<table>
<thead>
<tr>
<th>Table 8-20</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>RVOT</strong></td>
</tr>
<tr>
<td>Diameter (cm)</td>
</tr>
<tr>
<td>Area (cm²)</td>
</tr>
<tr>
<td>VTI (cm)</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
</tr>
<tr>
<td>Flow (L/min)</td>
</tr>
<tr>
<td>Pulmonic flow (Q_p)</td>
</tr>
<tr>
<td>Q_p:Q_s</td>
</tr>
</tbody>
</table>
Because $Q_p/Q_s = 2.5:1$, answer C is correct.

Answer A is incorrect because the presence of pulmonary hypertension per se does not preclude ASD closure. It is the degree of pulmonary vascular resistance (PVR) that determines whether a patient is a candidate for ASD closure or not, as discussed later.

Answer B is incorrect because the patient’s PVR is essentially normal. Using the Ohm’s law, PVR can be calculated as follows:

$$PVR = \frac{\Delta P}{Q_p} \quad (1)$$

where $Q_p$ is the pulmonary blood flow (in L/min), and $\Delta P$ is the pressure gradient across the pulmonary circulation. $PVR$ is the difference between the mean pulmonary artery pressure (MPP) and the mean left atrial pressure (LAP). Eq. 1 then becomes:

$$PVR = \frac{MPP - LAP}{Q_p} \quad (2)$$

MPP can be calculated from pulmonary artery systolic pressure (PASP) and the pulmonary artery diastolic pressure (PADP) by using the following equation:

$$MPP = PADP + \frac{1}{2} \times (PASP - PADP) \quad (3)$$

In this patient:

$$MPP = 25 + \frac{1}{2} \times (55 - 25) = 40 \text{ mm Hg}$$

Once MPP is known, we can use Eq. 2 to calculate PVR:

$$PVR = \frac{40 - 10}{11.9} = \frac{30}{11.9} = 3.4 \text{ Wood units}$$

Normal PVR is 1–2 Wood units (80–160 dyne s cm$^{-5}$). In this patient, PVR is only modestly elevated. In principle, ASD closure should not be performed if PVR is 2/3 or more of the systemic vascular resistance (SVR). Since normal SVR is approximately 13 Wood units (range, 11–16 Wood units, or 900–1,300 dyne s cm$^{-5}$), PVR ≥9 Wood units usually precludes ASD closure.

Answer D is incorrect because the shunt flow in this patient is 7.2 L/min. Shunt flow is the difference between $Q_p$ and $Q_s$. In this patient:

$$SF = Q_p - Q_s = 11.9 - 4.7 = 7.2 \text{ L/min}$$

Answer E is incorrect because $Q_p$ is much larger than $Q_s$, the shunt flow is in the left to right direction, and thus the patient is not expected to be cyanotic.

10. **Answer: B.** The presence of VSD allows for calculation of the RVSP and, by extension, the PASP if the systolic blood pressure (SBP) is known.

RVSP in a patient with VSD and no left ventricular outflow obstruction can be calculated as follows:

$$RSVP = SBP - \text{Peak systolic VSD gradient} \quad (1)$$

Using the peak systolic velocity ($V$) across the VSD, peak systolic VSD gradient can be calculated as follows:

$$\text{Peak systolic VSD gradient} = 4 \times V^2 \quad (2)$$

By combining Eqs. 1 and 2, RVSP is then calculated as follows:

$$RVSP = SBP - 4 \times V^2 \quad (3)$$

Thus in this patient,

$$RVSP = 120 - 4 \times (3.0 \text{ m/s})^2 = 84 \text{ mm Hg}$$

When there is no pulmonic stenosis, PASP = RVSP. However, this patient has PS with a PSG of 55 mm Hg across the pulmonic valve. In the presence of PS, the relationship between RVSP and PASP is as follows:

$$PASP = RVSP - \text{Peak PS gradient} \quad (4)$$

In our patient, PASP = 84 - 55 = 29 mm Hg. Therefore, answer B is correct.

Answer A is incorrect because RVSP in this patient is 84 mm Hg as calculated earlier.

Answer C is incorrect because RAP is not required for RVSP estimation using the VSD method.

Answer D is incorrect because PASP is lower than RVSP due to the presence of PS. RVSP exceeds PASP by 55 mm Hg, which is the peak gradient across the stenosed pulmonic valve.

Answer E is incorrect because the right ventricular end-diastolic pressure (RVEDP) in this patient is, if LVEDP is known, RVEDP can be calculated as follows:

$$RVEDP = LVEDP - \text{End – diastolic VSD gradient} \quad (5)$$

Using the end-diastolic velocity ($V$) across the VSD, the end-diastolic VSD gradient can be calculated as follows:

$$\text{End – diastolic VSD gradient} = 4 \times V^2 \quad (6)$$

By combining Eq. 5 and Eq. 6, RVEDP is then calculated as follows:

$$RVEDP = LVEDP - 4 \times V^2 \quad (7)$$
where \( V \) is the end-diastolic velocity across the VSD.

In our patient:

\[
\text{RVEDP} = 12 - 4 \times (1 \text{ m/s})^2 = 12 - 4 = 8 \text{ mm Hg}
\]

11. **Answer: B.** The pulmonic flow (\( Q_p \)) in patients with ASD is the sum of the SF across the ASD and the systemic flow (\( Q_s \)). SF can be calculated either directly or as the difference between \( Q_p \) and \( Q_s \).

One method for direct calculation of SF is the standard echocardiographic formula for determining flow rate through an orifice:

\[
\text{Flow} = \text{CSA} \times \text{VTI} \times \text{HR}
\]

where CSA is the cross-sectional area of the orifice, VTI is the velocity–time integral at the level of the orifice, and HR is the heart rate.

In the first step, we will calculate the CSA of the ASD whose diameter is 1.2 cm. Since the ASD is circular in shape, then ASD area can be calculated as follows:

\[
\text{CSA}_{\text{ASD}} = (\frac{1}{2} \times \text{ASD diameter})^2 \times \pi
\]

In our patient:

\[
\text{CSA}_{\text{ASD}} = \left(\frac{1}{2} \times 1.2 \text{ cm}\right)^2 \times 3.14 = 0.36 \times 3.14 = 1.13 \text{ cm}^2
\]

Next, we can calculate the stroke volume across the ASD as follows:

\[
\text{ASD shunt stroke volume} = \text{CSA}_{\text{ASD}} \times \text{VTI}_{\text{ASD}}
\]

In our patient:

\[
\text{ASD shunt stroke volume} = 1.13 \text{ cm}^2 \times 80 \text{ cm} = 90 \text{ mL per beat.}
\]

In the final step, by multiplying the ASD shunt stroke volume by the heart rate, one can calculate the SF across the ASD. In our patient:

\[
\text{ASD shunt flow} = 90 \text{ mL} \times 100 \text{ beats/min} = 9.0 \text{ L/min.}
\]

Therefore, the answer B is correct.

Answer A is incorrect because the \( Q_p:Q_s \) in this patient is 2.5:1. In this patient, \( Q_s \) is calculated at the level of the left ventricular outflow tract (LVOT) using the following formula:

\[
Q_s = \text{CSA}_{\text{LVOT}} \times \text{VTI}_{\text{LVOT}} \times \text{HR}
\]

where CSA\(_{\text{LVOT}}\) is the CSA of LVOT, VTI\(_{\text{LVOT}}\) is the velocity–time integral at LVOT level, and HR is the heart rate.

In our patient:

\[
Q_s = \left(\frac{1}{2} \times 2.0 \text{ cm}\right)^2 \times \pi \times 19 \text{ cm} = 60 \text{ mL} \times 100 \text{ beats/min} = 6.0 \text{ L/min}
\]

In the next step, we can calculate \( Q_p \) as follows:

\[
Q_p = Q_s + \text{ASD shunt flow}
\]

In our patient:

\[
Q_p = 6.0 \text{ L/min} + 9.0 \text{ L/min} = 15.0 \text{ L/min}
\]

Once \( Q_p \) and \( Q_s \) are known, we can calculate \( Q_p:Q_s \) ratio as follows:

\[
Q_p:Q_s = \frac{15.0 \text{ L/min}}{6.0 \text{ L/min}} = 2.5:1
\]

Answer C is incorrect because the difference between the pulmonic and systemic stroke volumes in this patient is 90 mL/beat. This value represents the ASD shunt stroke volume as calculated earlier.

Answer D is incorrect because the systemic stroke volume in this patient is 60 mL per beat as calculated earlier.

Answer E is incorrect because \( Q_p \) in this patient is 15.0 L/min as calculated earlier.

Calculations related to this question are summarized in this table.

12. **Answer: D.** Continuous Doppler spectral tracing of the mitral regurgitant jet can be used to estimate the rate of pressure rise (\( dP \)) in the left ventricle over

<table>
<thead>
<tr>
<th>Table 8-21</th>
<th>LVOT</th>
<th>ASD</th>
<th>RVOT</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diameter (cm)</td>
<td>2.0</td>
<td>1.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Area (cm(^2))</td>
<td>3.10</td>
<td>1.13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VTI (cm)</td>
<td>19</td>
<td>80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>60</td>
<td>90</td>
<td>150</td>
<td>RVOT stroke volume is the sum of LVOT and ASD stroke volumes.</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>100</td>
<td>100</td>
<td>15.0</td>
<td>Qp is the sum of Qp and ASD shunt flow.</td>
</tr>
<tr>
<td>Flow (L/min)</td>
<td>6.0</td>
<td>9.0</td>
<td>15.0</td>
<td>Qp:Qs = 2.5</td>
</tr>
<tr>
<td>Systemic flow (Qs)</td>
<td>Shunt flow</td>
<td>Pulmonic flow (Qp)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
time (dt), a measure of left ventricular systolic function, using the following formula:

\[ \frac{dP}{dt} = \frac{\Delta P}{RTI} \]  (1)

where RTI is the relative time interval, measured in seconds, between MR jet velocities of 1 m/s \( (V_1) \) and 3 m/s \( (V_2) \). \( \Delta P \) represents the pressure difference between the left ventricular to LAP gradients at \( V_2 \) and \( V_1 \) (Fig. 8-30A).

In the next step, we will calculate RTI in our patient:

\[ RTI = \text{Time at } V_2 - \text{Time at } V_1 = 25 \text{ ms} - 5 \text{ ms} = 20 \text{ ms} \]

Because in Eq. 2, RTI is expressed in seconds, we have to convert our patient RTI from milliseconds to seconds:

\[ RTI = 20 \text{ ms} = 0.02 \text{ s} \]

Once RTI is known, we can calculate \( \frac{dP}{dt} \) in our patient:

\[ \frac{dP}{dt} = \frac{32}{0.02} = 1,600 \text{ mm Hg/s} \]

Therefore, answer D is correct.

Answer A is incorrect because the peak velocity of mitral E wave in severe mitral regurgitation is expected to be high. Peak velocity across an orifice is directly related to flow across that orifice. Since the flow is the product of stroke volume (SV) and heart rate, peak velocity is then a direct function (f) of SV:

\[ \text{E wave velocity} = f(SV) \]  (3)

In mitral regurgitation, SV that crosses the mitral valve in diastole is the sum of the systemic SV \( (SV_{LVOT}) \) and the regurgitant volume \( (\text{RegV}) \). Thus, Eq. 5 can be expressed as follows:

\[ \text{E wave velocity} = f(SV_{LVOT} + \text{RegV}) \]  (4)

The more severe the mitral regurgitation is, the larger the RegV is, and therefore, the higher the peak velocity of the mitral inflow E wave. When native mitral regurgitation is severe (as is the case in this patient as judged by the vena contracta \( \geq 0.7 \text{ cm} \)), peak E velocity is expected to be >1.5 m/s. In severe prosthetic mitral regurgitation, the peak E velocity is usually >2.0 m/s.

Answer B is incorrect because LAP in this patient is elevated. The patient presents with severe mitral regurgitation (vena contracta \( \geq 0.7 \text{ cm} \)) and pulmonary edema due to elevated LAP.

Using the peak velocity \( (V_{\text{max}}) \) of the mitral regurgitant jet, one can calculate the pressure gradient \( (\Delta P) \) between the peak left ventricular systolic pressure \( (\text{LVSP}) \) and the LAP:

\[ \Delta P = 4 \times V_{\text{max}}^2 \]  (5)

In our patient:

\[ \Delta P = 4 \times (4.0 \text{ m/s})^2 = 4 \times 16 = 64 \text{ mm Hg} \]

The sum of this pressure gradient and LAP during systole represents the peak LVSP:

\[ \text{LVSP} = \Delta P + \text{LAP} \]  (6)

By rearranging Eq. 6, we can solve for LAP:

\[ \text{MVA} \frac{220}{270} = 0.8 \quad \text{LAP} = \text{LVSP} - \Delta P \]  (7)
The LAP calculated by this method represents a value on the CV wave portion of the LAP tracing. LVSP is not given in the question. In this patient who does not have aortic stenosis or left ventricular outflow obstruction, LVSP is equal to SBP. Thus we can express Eq. 7 as follows:

\[
\text{LAP} = \text{SBP} - \Delta P
\]  

(8)

In our patient, whose SBP was 95 mm Hg and whose \(\Delta P\) was calculated above at 64 mm Hg, LAP is then calculated as follows:

\[
\text{LAP} = 95 \text{ mm Hg} - 64 \text{ mm Hg} = 31 \text{ mm Hg}
\]

This LAP of 31 mm Hg is highly elevated (normal LAP is \(\leq 12 \text{ mm Hg}\)).

Answer C is incorrect because in severe mitral regurgitation there may be flow reversal in systolic (S) but not diastolic (D) wave on pulmonary venous flow velocity tracings. An example of S wave reversal due to severe mitral regurgitation is shown in Figure 8-30B.

Answer E is incorrect because \(dP/dt\) in this patient is estimated at 1,600 mm Hg/s, which is normal. (Normal \(dP/dt = 1,661 + 323 \text{ mm Hg/s}\).) The value of 800 mm Hg/s would indicate a markedly diminished LV systolic function as seen in cardiogenic shock, for example.

13. Answer: E. In mitral stenosis, there is a pressure gradient between the left atrium and the left ventricle during diastole. In this patient, the mean diastolic pressure gradient is markedly elevated (21 mm Hg). Mean diastolic pressure gradient of \(>10 \text{ mm Hg}\) is consistent with severe mitral stenosis as shown in the table below.

| Table 8-22 |
|---|---|---|
| Mitral valve area (cm\(^2\)) | Mild MS | Moderate MS | Severe MS |
| Mean diastolic gradient (mm Hg) | <1.0 | 1.0–1.5 | >1.5 |
| Mean diastolic gradient (mm Hg) | <5 | 5–10 | >10 |

In this young patient, left ventricular diastolic pressures are normal. Mean LAP can be calculated as follows:

\[
\text{LAP} = \text{Mean mitral gradient in diastole} + \text{Early LV diastolic pressure}
\]

In our patient:

\[
\text{LAP} = 21 \text{ mm Hg} + 7 \text{ mm Hg} = 28 \text{ mm Hg}
\]

Therefore, answer E is correct.

Answer A is incorrect because in mitral stenosis there is an antegrade flow driven by a pressure gradient between the left atrium and the left ventricle in diastole. Therefore, the mean LAP is higher than the mean left ventricular diastolic pressure.

Answer B is incorrect because in mitral stenosis the peak velocity of the mitral E wave is expected to be high. Velocity (V) across an orifice is inversely related to the cross-sectional area (CSA) of the orifice:

\[
V = \frac{1}{\text{CSA}}
\]  

(1)

For mitral stenosis, CSA equals to the MVA and Eq. 1 becomes

\[
V = \frac{1}{\text{MVA}}
\]  

(2)

Therefore, the smaller the MVA (i.e., the more severe the mitral stenosis), the higher the peak velocity of the mitral E wave.

Answer C is incorrect because the PHT method may be unreliable immediately after but not before the mitral valvuloplasty. PHT method assumes that the left ventricular pressure and compliance are normal, and therefore that the deceleration slope of the mitral E wave on spectral Doppler tracings in diastole is the function of the MVA alone.

Immediately after valvuloplasty, there is a sudden increase in the mitral orifice area leading to an increase in the stroke volume delivered to the left ventricle in early diastole. Because the left ventricle compliance cannot change acutely, the left ventricular diastolic pressure increases. With the rise in the left ventricular diastolic pressure, the diastolic gradient between the left atrium and the left ventricle decreases and the mitral PHT shortens above and beyond what would be expected by an increase in the MVA alone after valvuloplasty. Therefore, the PHT method may lead to calculation of an erroneously large MVA.

Answer D is incorrect because the MVA by PHT method in this patient is 0.8 cm\(^2\):

\[
\text{MVA} = \frac{220}{270} = 0.8
\]

14. Answer: C. When velocity–time integrals are not available, AVA can be calculated using the following modified continuity equation:

\[
\text{AVA} = \text{CSA}_{\text{LVOT}} \times \frac{V_{\text{LVOT}}}{V_{\text{AV}}}
\]  

(1)

where CSA\(_{\text{LVOT}}\) is the CSA of the LVOT, \(V_{\text{LVOT}}\) is the peak systolic LVOT velocity, and \(V_{\text{AV}}\) is the peak systolic AV velocity.

The \(V_{\text{LVOT}}/V_{\text{AV}}\) ratio of the two velocities is referred to as the dimensionless index (DI). Thus Eq. 1 can be expressed as follows:

\[
\text{AVA} = \text{CSA}_{\text{LVOT}} \times \text{DI}
\]  

(2)
After expressing the LVOT area in terms of LVOT diameter (D), Eq. 2 becomes:

$$AVA = \left(\frac{1}{2} \times D\right)^2 \times DI$$

(3)

In our patient:

$$AVA = \left(\frac{1}{2} \times [1.9 \text{ cm}]^2\right) \times (1 \text{ m/s/5 m/s})$$

$$AVA = 2.84 \text{ cm}^2 \times 0.2$$

AVA = 0.6 cm²

Therefore, answer C is correct.

As a rule, when the dimensionless index (DI) is ≤0.25, the AVA is <1.0 cm² across the range of LVOT diameters commonly encountered in adults as demonstrated in the following table:

<table>
<thead>
<tr>
<th>LVOT Diameter (cm)</th>
<th>LVOT Area (cm²)</th>
<th>AVA (cm²) if DI = 0.25</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.8</td>
<td>2.54</td>
<td>0.64</td>
</tr>
<tr>
<td>1.9</td>
<td>2.84</td>
<td>0.71</td>
</tr>
<tr>
<td>2.0</td>
<td>3.14</td>
<td>0.79</td>
</tr>
<tr>
<td>2.1</td>
<td>3.46</td>
<td>0.87</td>
</tr>
<tr>
<td>2.2</td>
<td>3.80</td>
<td>0.95</td>
</tr>
</tbody>
</table>

Answer A is incorrect because the modified continuity equation using the DI, as explained earlier, can be used to calculate the AVA when velocity–time integrals are unavailable.

Answer B is incorrect because the subvalvular (LVOT) velocity is normal (1.0 m/s).

Answer D is incorrect because the left ventricular SV in this patient is 57 mL per beat. Left ventricular SV can be calculated as follows:

$$SV = \frac{1}{2} \times (\text{LVOT diameter})^2 \times \text{VTI}_{LVOT}$$

SV = \frac{1}{2} \times (1.9 \text{ cm})^2 \times 20 \text{ cm}

SV = 57 \text{ mL per beat}

Answer E is incorrect because in aortic stenosis, left ventricular peak systolic pressure exceeds the systolic blood pressure. The magnitude of this pressure difference (peak-to-peak gradient) is proportional to the severity of aortic stenosis.

15. Answer: C. Peak RVSP in a patient with or without pulmonic stenosis (PS) can be calculated as follows:

$$\text{RVSP} = \text{Peak RV-to-RA systolic gradient} + \text{RAP}$$

(1)

where RAP is the right atrial pressure. Since RV-to-RA systolic gradient can be estimated from the peak systolic velocity of the tricuspid regurgitant (V), Eq. 1 can be expressed as follows:

$$\text{RVSP} = 4 \times V^2 + \text{RAP}$$

(2)

In the absence of PS, RVSP is equal to PASP. In PS, however, peak RVSP exceeds PASP. The difference between the two pressures represents the peak gradient of PS. Therefore, in patients with PS, PASP is estimated as follows:

$$\text{PASP} = \text{RVSP} – \text{PS gradient}$$

(3)

In our patient:

$$\text{RVSP} = 4 \times (4.0 \text{ m/s})^2 + 10 = 74 \text{ mm Hg}$$

PASP = 74 – 24 = 50 mm Hg

Therefore, answer C is correct.

All calculations are graphically summarized in Figure 8-31 (RVP, right ventricular pressure; RAP, right atrial pressure; PAP, pulmonary artery pressure).

Answer A is incorrect because in the presence of pulmonic valve stenosis, RVSP exceeds PASP as shown in the figure earlier.

Answer B is incorrect because RVSP exceed PASP by 24 mm Hg, the value of the PSG across the pulmonic valve.

Answer D is incorrect because RVSP is 24 mm Hg more than PASP.

Answer E is incorrect because RVSP is 74 mm Hg as calculated earlier.

16. Answer: C. Peak gradient (ΔP_max) of aortic stenosis can be calculated from the peak systolic velocity (V) across the aortic valve obtained by continuous wave Doppler using the modified Bernoulli equation:

$$\Delta P_{\text{max}} = 4 \times V^2$$

(1)

The mean aortic valve gradient (ΔP_mean) is approximately 60% of the peak gradient (ΔP_max):

$$\Delta P_{\text{mean}} = 0.6 \times \Delta P_{\text{max}}$$

(2)
In our patient:

\[
\Delta P_{\text{max}} = 4 \times (5.0 \text{ m/s})^2 = 100 \text{ mm Hg}
\]

\[
\Delta P_{\text{mean}} = 0.6 \times 100 \text{ mm Hg} = 60 \text{ mm Hg}
\]

Therefore, answer C is correct.

Answer A is incorrect because increased cardiac output (as during pregnancy, for instance) leads to a proportional increase in both LVOT and aortic velocities. In this patient, there is a marked difference between the peak systolic LVOT velocity (0.9 m/s) and the peak systolic aortic velocity (5.0 m/s) indicative of aortic stenosis.

Answer B is incorrect because in aortic regurgitation there is a proportional increase in both LVOT and aortic velocities in systole due augmentation of the left ventricular stroke volume by the recirculating regurgitant volume. A wide discrepancy in the peak LVOT and aortic velocities in systole is not expected in severe aortic regurgitation.

Answer D is incorrect because the AVA in this patient is less than 1.0 cm².

\[
\text{AVA} = \text{CSA}_{\text{LVOT}} \times \frac{V_{\text{LVOT}}}{V_{\text{AV}}} \quad (3)
\]

After expressing the LVOT area in terms of LVOT diameter (D), Eq. 3 becomes:

\[
\text{AVA} = \left( \frac{1}{2} \times D \right)^2 \times \frac{V_{\text{LVOT}}}{V_{\text{AV}}} \quad (3)
\]

where CSA_{\text{LVOT}} is the cross-sectional area of the LVOT, \( V_{\text{LVOT}} \) is the LVOT peak systolic velocity, and \( V_{\text{AV}} \) is the peak aortic velocity in systole.

In this patient:

\[
\text{AVA} = \left( \frac{1}{2} \times 2.0 \right)^2 \times \frac{1.2}{5.0} = 0.75 \text{ cm}^2
\]

Answer E is incorrect because the subvalvular (LVOT) velocity of 1.2 m/s is normal.

17. **Answer: D.** The patient has severe pulmonic valve regurgitation, a common long-term complication of tetralogy of Fallot repair.

Because of a large regurgitant orifice, the pressure gradient between pulmonary artery and the right ventricle equalizes rapidly. Equalization is achieved by mid diastole and there is no measurable end-diastolic gradient as demonstrated in **Figure 8-32**.

This rapid deceleration and premature cessation of the pulmonic regurgitant jet are a characteristic finding of severe pulmonic regurgitation. Therefore, answer D is correct.

Answer A is incorrect because the end-diastolic gradient in severe pulmonic regurgitation is approaching zero.

![Figure 8-32](image)

Answer B is incorrect because the peak antegrade velocity across the pulmonic valve in systole is only elevated to about 1.5 m/s (peak systolic gradient \( 4 \times 1.5^2 = 9 \text{ mm Hg} \)). This is consistent with pulmonic regurgitation alone. During systole, stroke volume is augmented by the recirculating regurgitant volume. This flow augmentation leads to higher systolic velocities across the pulmonic valve based on the fundamental equation of fluid dynamics:

\[
V = \frac{Q}{PVA} = \frac{SV \times HR}{PVA}
\]

where \( V \) is the antegrade velocity across the pulmonic valve, \( Q \) is the volumetric flow across the pulmonic valve in systole, \( SV \) is the stroke volume, \( HR \) is the heart rate, and \( PVA \) is the pulmonic valve area. Thus, when the PVA remains constant, any increase in stroke volume leads to elevation in the transvalvular velocity.

Answer C is incorrect, the right ventricular systolic pressure that exceeds pulmonary artery by 9 mm Hg (see the figure above).

Answer E is incorrect because in uncomplicated patent ductus arteriosus, antegrade flow occurs during both systole and diastole. In the patient’s tracing there is antegrade flow in systole and retrograde flows in diastole.

18. **Answer: C.** The tracings were obtained from an elderly woman presenting with acutely decompen-sated heart failure.

Mean left atrial pressure can be estimated semiquantitatively from the ratio of peak flow velocity of mitral E wave and the peak velocity of mitral annular tissue Doppler e’ wave according to the following chart:
inflow A wave, while the retrograde flow into the pulmonary veins is responsible for the atrial reversal (AR) wave.

When the LVDP is elevated at the time of atrial contraction, both the peak velocity and the duration of the AR wave are increased. A peak AR velocity of ≥35 cm/s is indicative of elevated LV end-diastolic pressure.

Elevation of LV end-diastolic pressure can also be inferred when the duration of the AR wave is ≥30 ms more than the duration of the mitral inflow A wave. In our patient, peak velocity of AR was 50 cm/s, and AR outlasted mitral A wave by 40 ms (210 – 170 ms); both are indicative of an elevated LVDP. Therefore, answer B is correct.

For further explanation, the reader is referred to Figure 13 in the Canadian Consensus Recommendations for the Measurement and Reporting of Diastolic Dysfunction by Echocardiography (J Am Soc Echocardiogr. 1996;9:736–760).

Answer A is incorrect because a restrictive filling pattern is characterized by a mitral inflow E/A ratio greater than 2; in this patient, peak E wave velocity is barely higher than the peak A wave velocity.

Answer C is incorrect because the higher the peak velocity of the atrial reversal wave in the pulmonary vein spectral tracing, the higher the LVDP.

Answer D is incorrect because with left ventricular dysfunction, there is an increase in the LVDP leading to secondary pulmonary hypertension. Because of LV diastolic pressure elevation, the pulmonary vein atrial reversal wave is likely to be prominent (as explained earlier) rather than absent. Atrial reversal wave is absent in atrial arrhythmias such as atrial fibrillation.

Answer E is incorrect because with a Valsalva maneuver the peak velocity of the mitral E wave is expected to decrease. Valsalva maneuver decreases preload and leads to a lower early diastolic pressure gradient between the left atrium and left ventricle. This leads to a lower peak velocity of the mitral E wave and a lower mitral E/A ratio.

19. **Answer: B.** In sinus rhythm, the left atrium contracts following the P wave on EKG and the blood is propelled both forward into the left ventricle across the mitral valve, as well as backward into the pulmonary veins, which lack valves. The velocity profile of the forward flow is responsible for the mitral inflow A wave, while the retrograde flow into the pulmonary veins is responsible for the atrial reversal (AR) wave.

In our patient, E/lateral e’ is 142/8 or 18. This ratio is consistent with elevated left atrial pressure. Therefore, answer C is correct.

Answer A is incorrect because the patient is likely to have poor exercise capacity with exertional dyspnea given the elevation of left atrial pressure even at rest. With exertion, left atrial pressure is expected to rise even further.

Answer B is incorrect because the patient’s mitral inflow pattern is a combination of abnormal left ventricular relaxation and elevated left atrial pressure. The mitral E/A ratio that is greater than 2 in conjunction with a rapid E wave deceleration time (<160 ms) indicates a restrictive filling pattern. The features of different filling patterns in individuals older than 60 years are summarized in the table below.

Answer D is incorrect because the patient has a restrictive filling pattern. This is an abnormal finding and consistent with severe left ventricular diastolic dysfunction.

Answer E is incorrect because with a Valsalva maneuver the peak velocity of the mitral E wave is expected to decrease. Valsalva maneuver decreases preload and leads to a lower early diastolic pressure gradient between the left atrium and left ventricle. This leads to a lower peak velocity of the mitral E wave and a lower mitral E/A ratio.

20. **Answer: B.** In constrictive pericarditis, ventricular filling is constrained by an inelastic pericardial sac that envelopes the entire heart except for the cranial portion of the left atrium and the pulmonary
veins. This results in (1) ventricular interdependence and (2) a differential impact of negative intrathoracic pressure that develops during inspiration on pulmonary veins and the heart.

Ventricular interdependence refers to diastolic filling of one ventricle at the expense of the other depending on the respiratory phase. In inspiration, the pressure in the intrathoracic systemic vein decreases. This leads to a larger pressure gradient between extra- and intrathoracic systemic veins, which results in improved RV filling. At the same time, the drop in the intrathoracic pressure with inspiration decreases the pulmonary venous pressure. Because of the thickened rigid pericardium, the drop in the intrathoracic pressure cannot be transmitted to the heart; this results in a decreased pressure gradient between pulmonary veins and the left atrium, and decreased LV filling in diastole.

The net effect of inspiration is such that the right ventricle fills at the expense of the left ventricle, and the interventricular septum moves toward the left ventricle. The opposite occurs in expiration. This is illustrated in the M-mode recordings of our patient. The recordings also demonstrate no pericardial effusion.

With inspiration, the drop in intrathoracic pressure enhances forward flow in the hepatic veins in normal individuals; in constrictive pericarditis, there is an exaggeration of this inspiratory forward flow enhancement. During expiration, the rightward shift of the interventricular septum impedes RV filling; the rise in the RV diastolic pressure then leads to an expiratory increase in hepatic vein flow reversal. Therefore, answer B is correct.

Answer A is incorrect because the presence of marked reciprocal changes in the right and left ventricular filling that are phasic with respiration and indicative of ventricular interdependence.

Answer C is incorrect because the abnormal septal motion due to right ventricular overload (as in atrial septal defect or severe tricuspid regurgitation) is characterized by flattening of the interventricular septum with each diastole rather than being phasic with respiration.

Answer D is incorrect because an inspiratory increase in antegrade velocities is a normal finding. During inspiration, the drop in intrathoracic pressures enhances systemic venous return. This increased flow into the right-sided heart elevates antegrade velocities in the hepatic veins.

Answer E is incorrect because the M-mode reveals no echo lucency posterior to the left ventricle that would be diagnostic of a large pericardial effusion. Instead, it shows pericardial thickening. It is important to emphasize, however, that the abnormal interventricular septal motion phasic with respiration is encountered in both tamponade and constrictive pericarditis.

21. **Answer B.** In a normal descending aorta, antegrade flow occurs only in systole and there is a small flow reversal in early diastole as depicted in the following pulsed wave Doppler tracing in **Figure 8-33A**.

The pulsed-wave Doppler tracings in **Figure 8-7** is abnormal as it demonstrates that antegrade flow is throughout the cardiac cycle. In addition, there is a large peak systolic gradient across the coarctation of almost 60 mm Hg. The presence of a holodiastolic antegrade flow in conjunction with a large systolic gradient is indicative of severe aortic coarctation. Therefore, answer B is correct.

Answer A is incorrect because in severe aortic regurgitation, there is a retrograde flow throughout diastole (holodiastolic flow reversal) as demonstrated in **Figure 8-33B, C**.

Answer C is incorrect because it is the bicuspid and not quadricuspid aortic valve that is typically associated with aortic coarctation. It is estimated that between 25% and 46% of all individuals with coarctation have bicuspid aortic valve.

Answer D is incorrect for two reasons. First, if this were a recording from the ascending aorta, forward velocities would have been recorded above the baseline and not below it. Second, aortic stenosis is not characterized by an antegrade diastolic gradient across the aortic valve.

Answer E is incorrect because coarctation usually occurs distal to the origin of the neck arteries, and the blood pressure in the arms is higher than that in the legs.
Holodiastolic flow reversal in the descending thoracic aorta (arrows) indicative of severe aortic insufficiency is demonstrated by both spectral Doppler (left panel) and color M-mode recordings (right panel).

22. Answer: C. Normal systole consists of isovolumic contraction time, and ejection period. Flow across the aortic valve, whether the valve is normal or stenotic, occurs only during the ejection period of systole. In contrast, tricuspid regurgitant jet extends throughout the systole. Thus, on a spectral Doppler tracing, the aortic stenosis jet is shorter duration and has a later onset compared to the tricuspid regurgitant as demonstrated in figures later. Therefore, answer C is correct.

TRICUSPID REGURGITANT JET
Peak velocity = 4.5 m/sec
Jet duration = 515 msec

AORTIC STENOSIS JET
Peak velocity = 5.0 m/sec
Jet duration = 345 msec

Answer A is incorrect because Figure 8-8B represents the flow velocity pattern across the aortic valve. Note the short time interval (isovolumic contraction time) between the QRS and the onset of flow in the right panel. In contrast, the onset of tricuspid regurgitant jet in Figure 8-8A coincides with the QRS on EKG.

Answer B is incorrect because the aortic jet starts after the isovolumic contraction period.

Answer D is incorrect because a peak velocity of 5 m/s does not exclude a tricuspid regurgitant
jet; such a tricuspid jet velocity can be recorded in a patient with a very severe pulmonary hypertension (pulmonary systolic pressure >100 mm Hg).

Answer E is incorrect because the systolic function of both ventricles appears normal given the rapid rise in velocities from their baseline to their peak values. This rapid flow acceleration is consistent with a normal dP/dt, a measure of systolic function.

23. **Answer: B.** In both tamponade and constrictive pericarditis, there is impairment in ventricular filling during diastole. In tamponade, the impediment is caused by the pericardial fluid around the heart, while in constrictive pericarditis the impediment is caused by a thickened rigid and sometimes calcified pericardium.

In tamponade, the left ventricular filling is impaired from the onset of diastole. On spectral Doppler recordings of mitral inflow, this is manifested by the pattern of abnormal relaxation (peak velocity of the mitral E wave is lower than that of the A wave, and the deceleration time of the E wave is prolonged).

In contrast, in constrictive pericarditis, early diastolic filling is rapid and then abruptly decreases in late diastole when the expanding myocardium reaches the rigid pericardium. This can be demonstrated by either cardiac catheterization or Doppler echocardiography. On cardiac catheterization there is a rapid y descent in right atrial pressure tracings and a dip-and-plateau pattern on right ventricular pressure tracings. On spectral Doppler recordings of mitral inflow, there is a restrictive filling pattern (the ratio of peak E wave to peak A wave velocity >2; deceleration time of E wave <160 ms).

Both in tamponade and constrictive pericarditis, there is ventricular interdependence. Because of ventricular interdependence, there is a marked decrease in left ventricular filling during inspiration. The magnitude of inspiratory drop in early diastolic filling (as measured by peak velocity of mitral E wave) is directly proportional to the severity of either tamponade or constrictive pericarditis. In normal individuals, inspiratory drop in peak E wave velocity with inspiration is small, in tamponade and constrictive pericarditis, the inspiratory drop ≥25%. One uses the following formula to calculate percent respiratory variation in the peak velocity of mitral E wave ($\Delta E$):

$$\Delta E = \frac{E_{\text{Expiration}} - E_{\text{Inpiration}}}{E_{\text{Expiration}}}$$

Bear in mind that marked respiratory variations are not unique to tamponade and constrictive pericarditis; they also occur with labored breathing, asthma, chronic obstructive lung disease, pulmonary embolism, and obesity.

In our patient:

$$\Delta E = \frac{170 - 110}{170} = \frac{60}{170} = 35\%$$

In summary, the combination of the abnormal relaxation mitral inflow pattern and the marked respiratory variations in the peak velocity of the mitral inflow E wave is consistent with the diagnosis of cardiac tamponade. Therefore, answer E is correct.

Answer A is incorrect because in both tamponade and constrictive pericarditis, the respiratory
variations are measured in the peak velocity of the E wave, not the A wave.

Answer C is incorrect for two reasons. First, the mitral inflow filling pattern in this patient demonstrates abnormal relaxation (E/A < 1) rather than restrictive filling (E/A > 2 and E wave deceleration time < 160 ms). Second, there are no significant respiratory variations in mitral inflow in patients with restrictive cardiomyopathy. An additional distinction between restrictive cardiomyopathy and constrictive pericarditis is the peak velocity of the mitral annular tissue Doppler early diastolic e’ wave. The e’ velocity is normal in constrictive pericarditis and diminished in restrictive cardiomyopathy.

Answer D is incorrect because an E/A < 1 favors tamponade over constrictive pericarditis as discussed earlier.

Answer E is incorrect because diuretics should not be administered to patients with tamponade physiology since the decrease in preload caused by diuretics would further impair ventricular filling.

24. Answer: A. The spectral recordings were obtained from a patient with very severe tricuspid regurgitation. When the tricuspid regurgitant orifice is large, there is ventricularization of the RAPs, which results in a very rapid pressure equilibration between right ventricular pressure (RVP) and the RAP as demonstrated in pressure tracings in Figure 8-35A.

The rapid rise in the RAP results in a rapid deceleration slope of the tricuspid regurgitant jet (arrow in the continuous Doppler tracing of the tricuspid regurgitant jet in Figure 8-35B). Therefore, answer A is correct.

Clinically, patients with this type of tricuspid regurgitation typically have a pulsatile liver. An echocardiographic correlate of pulsatile liver is the systolic wave reversal in hepatic vein spectral Doppler tracings as shown in Figure 8-35C.

25. Answer: B. In patients with uncomplicated VSD, there is antegrade flow from the left ventricle to the right ventricle during both systole and diastole. Using the $4 \times V^2$ formula to calculate the peak systolic and end-diastolic pressure gradients from VSD spectral Doppler recordings, one can estimate right ventricular peak systolic pressure (RVSP) and LVEDP, respectively.
As shown in Figure 8-36, this patient’s peak systolic and end-diastolic pressure gradients are as follows:

VSD peak systolic gradient
\[ = 4 \times (3.5 \text{ m/s})^2 = 4 \times 12.25 \text{ mm Hg} = 49 \text{ mm Hg} \]

VSD end-diastolic gradient
\[ = 4 \times (2.0 \text{ m/s})^2 = 4 \times 4 \text{ mm Hg} = 16 \text{ mm Hg} \]

To calculate the LVEDP, we need to subtract the estimate of the RV end-diastolic pressure. In the absence of tricuspid stenosis, RV end-diastolic pressure equals the end-diastolic RA pressure (which is given in the question as 10 mm Hg). Thus:

LVEDP = VSD end-diastolic gradient
- RA end-diastolic pressure
LVEDP = 16 - 10 = 6 mm Hg

In addition, we can calculate the RV peak-systolic pressure in this patient as well by subtracting the VSD peak systolic gradient from the patient’s systolic blood pressure (SBP):

RV peak systolic pressure
\[ = \text{SBP} - \text{VSD peak-systolic gradient} \]
RV peak systolic pressure
\[ = 90 - 49 \text{ mm Hg} = 41 \text{ mm Hg} \]

26. **Answer: C.** Normal mitral annular tissue Doppler recordings demonstrate the following:

- Peak E′ velocity is normally higher in the lateral compared to the medial annulus. In other words, E′ lateral/E′ medial >1.
- E′ velocity is typically higher than the peak velocity of the late diastolic (A′) wave.

In this patient, overall peak E′ velocities are within the normal range. However, the medial E′ velocity is much higher than the lateral E′ velocity. Therefore, in this patient, E′ lateral/E′ medial <1. This phenomenon, when E′ lateral/E′ medial is reversed from what is normally observed, is referred to as *annulus reversus*.

The finding of annulus reversus on mitral annular tissue Doppler is consistent with the diagnosis of constrictive pericarditis. The lateral E′ velocity diminishes relative to the medial E′ velocity because fibrosis and calcifications of the surrounding pericardium restrict the motion of the lateral annulus. The medial E′ velocity is typically not affected in constrictive pericarditis as the septal annulus is not surrounded by the pericardium.

Answer A is incorrect as the left-sided heart valves (mitral and aortic valve) are not typically affected by carcinoid heart disease. Carcinoid disease typically affects the right-sided heart valves rendering the leaflets of the tricuspid and pulmonic valve immobile; this results in both stenosis and regurgitation of the tricuspid and pulmonic valve.

Answer B is incorrect as both medial and lateral annular tissue Doppler velocities are diminished in patients with amyloidosis but the ratio of E′ lateral to E′ medial of >1 is maintained as in normal individuals.

Answer D is incorrect because mitral annular calcification leads to a decrease in both medial and
lateral annular tissue Doppler velocities, but the ratio of E’ lateral to E’ medial of >1 is maintained as in normal individuals.

Answer E is incorrect as patients with mitral regurgitation due to a flail leaflet typically have lateral E’ velocities higher than medial E’ velocities.

27. **Answer:** E. Normally, the peak gradient across an uncomplicated ASD is small and measures only a few mm Hg as the LA pressure is only slightly higher than the RA pressure.

However, in patients with left ventricular dysfunction and/or severe mitral valve disease, the LA pressure may rise to high levels and lead to pulmonary edema. If such a patient has an ASD with a left-to-right shunt, the peak LA pressure can be calculated as the sum of the peak trans-ASD gradient and the RA pressure estimate. In this patient:

\[
\text{Peak trans-ASD gradient} = 4 \times 3.3 \text{ m/s} = 4 \times 11 = 44 \text{ mm Hg}
\]

Since the RA pressure is given as 8 mm Hg, then:

\[
\text{Peak LA pressure} = \text{Peak trans-ASD gradient} + \text{RA pressure} = 44 + 8 = 52 \text{ mm Hg}
\]

28. **Answer:** E. Typically, mitral regurgitation is holosystolic and both EROA and regurgitant volume are accurate measures of mitral regurgitation severity as shown in the table below.

However, when mitral regurgitation is only late systolic (as in many cases of mitral valve prolapse), there is discordance between the EROA and the regurgitant volume.

This patient’s EROA of 0.48 cm\(^2\) would suggest severe mitral regurgitation. However, since the MR jet is only late systolic, its velocity–time integral (VTI) is relatively small and consequently the calculated regurgitant volume is not consistent with severe mitral regurgitation.

In general, regurgitant volume is a product of EROA and the regurgitant jet VTI:

\[
\text{Regurgitant volume} = \text{EROA} \times \text{VTI}
\]

In this patient:

\[
\text{Regurgitant volume} = 0.48 \text{ cm}^2 \times 72 \text{ cm} = 35 \text{ mL}
\]

Thus, the regurgitant volume of 35 mL in this patient is indicative of moderate mitral regurgitation.

In summary, in patients with mitral valve prolapse and late systolic regurgitant jet, EROA overestimates the severity of mitral regurgitation. In such instances, regurgitant volume and fraction are better measures of mitral regurgitation severity compared to EROA.

In contrast, in patients with holosystolic mitral regurgitation, EROA and regurgitant volume are concordant as illustrated in the right panel of Figure 8-37.

In the above patient with annular dilatation, let us assume that EROA is also 0.48 cm\(^2\) as in the patient with mitral valve prolapse. Note, however, that the VTI in the patient with annular dilatation is much higher than that in the patient with mitral valve prolapse (141 cm vs. 72 cm).

The regurgitant volume in the patient with annular dilatation and holosystolic mitral regurgitation can be calculated as follows:

\[
\text{Regurgitant volume} = \text{EROA} \times \text{VTI}
\]

\[
\text{Regurgitant volume} = 0.48 \text{ cm}^2 \times 141 \text{ cm} = 68 \text{ mL}
\]

A regurgitant volume of 68 mL is consistent with severe mitral regurgitation.

Answer A is incorrect because mitral regurgitation in this patient is late systolic and not early systolic.

Answer B is incorrect because EROA overestimates the severity of mitral regurgitation in patients with mitral valve prolapse and late systolic mitral regurgitation as discussed earlier.

Answer C is incorrect because the peak systolic LV to LA gradient based on the mitral regurgitant jet is 135 mm Hg:

\[
\text{Peak LV to LA gradient} = 4 \times (5.8 \text{ m/s})^2 = 135 \text{ mm Hg}
\]

Peak LV systolic pressure can be calculated as follows:

\[
\text{Peak LV systolic pressure} = \text{Peak LV to LA gradient} + \text{LA pressure}
\]

In this patient:

\[
\text{Peak LV systolic pressure} = 135 + 10 = 145 \text{ mm Hg}
\]

**Table 8-26. Grading of Mitral Regurgitation Severity**

<table>
<thead>
<tr>
<th></th>
<th>Mild (1+)</th>
<th>Moderate (2+)</th>
<th>Moderate-severe (3+)</th>
<th>Severe (4+)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EROA (cm(^2))</td>
<td>&lt;0.2</td>
<td>0.20–0.29</td>
<td>0.30–0.39</td>
<td>≥0.4</td>
</tr>
<tr>
<td>Regurgitant fraction</td>
<td>&lt;30%</td>
<td>30–39%</td>
<td>40–49%</td>
<td>≥50%</td>
</tr>
<tr>
<td>Regurgitant volume (mL)</td>
<td>30</td>
<td>30–44</td>
<td>45–59</td>
<td>≥60</td>
</tr>
<tr>
<td>Vena contracta (cm)</td>
<td>&lt;0.3</td>
<td>0.3–0.7</td>
<td>&gt;0.7</td>
<td></td>
</tr>
</tbody>
</table>
In the absence of aortic stenosis:

Systolic blood pressure = Peak LV systolic pressure

Thus, this patient’s systolic blood pressure is approximately 145 mm Hg and the patient is hypertensive rather than hypotensive.

Answer D is incorrect as the regurgitant fraction cannot be calculated from the provided data as the forward stroke volume across a nonregurgitant orifice (such as the LV outflow tract in the absence of aortic regurgitation) is not provided for this patient.

In general:

\[
\text{Regurgitant fraction} = \frac{\text{Regurgitant volume}}{\text{Regurgitant volume} + \text{Forward stroke volume}}
\]

29. **Answer: D.** The spectral Doppler tracing demonstrates antegrade flow in both systole and diastole. Since the patient is in normal sinus rhythm, the flow is triphasic and consists of a systolic (S) wave and two diastolic waves: early diastolic (E) wave and late diastolic (A) wave. The mean gradient of 10 mm Hg in this patient is indicative of significant stenosis.

This triphasic flow is characteristic of cor triatriatum (literally, a heart with three atria). In this congenital malformation, a perforated membrane divides the left atrium into 2 chambers: the posterior left atrium that receives the pulmonary veins, and the anterior left atrium that is connected to the left atrial appendage and is bound by the mitral valve.

Clinical presentation of cor triatriatum is similar to that of mitral stenosis. However, mitral stenosis (whether supravalvular, valvular, or subvalvular) is characterized by an elevated transmitral pressure gradient that is present only during diastole. In contrast, the elevated gradient of cor triatriatum is present in both systole and diastole. This is illustrated in Figure 8-38.

Answers A, C, and D refer to various forms of mitral stenosis (supravalvular mitral stenosis due to supravalvular ring, valvular rheumatic mitral stenosis, subvalvular stenosis). All three answers are incorrect as systolic antegrade flow across the mitral valve is absent in all of them.

Shone complex is a congenital syndrome of sequential obstructions in the left heart and the aorta. Patients with Shone complex may have present with one or more of the following: supravalvular mitral ring, parachute mitral valve (single papillary muscle with maldeveloped chordae resulting in subvalvular mitral stenosis), subaortic membrane, and coarctation of the aorta. Cor triatriatum is not a manifestation of Shone complex.

30. **Answer: C.** The patient initially presents with acutely decompensated heart failure due to acute coronary syndrome (non–ST-elevation myocardial infarction) in the distribution of the left anterior descending coronary artery.

The five mitral inflow patterns presented in Question 30 were as follows:

<table>
<thead>
<tr>
<th>Table 8-27</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
</tr>
<tr>
<td>B</td>
</tr>
<tr>
<td>C</td>
</tr>
<tr>
<td>D</td>
</tr>
<tr>
<td>E</td>
</tr>
</tbody>
</table>
Since the patient has a normal native mitral valve and was in normal sinus rhythm at the time of study, patterns D and E do not belong to this patient.

Using the E/e' ratio concept (discussed in answer to Question 8), we can estimate the mean PAWP for the remaining three patterns:

Of the three remaining patterns, only the restrictive filling (pattern C) predicts a PAWP that is in general agreement with the 38 mm Hg value obtained invasively by Swan–Ganz catheter. Therefore, answer C is correct.

31. Answer: E. The severity of the mitral regurgitation can be assessed using the PISA method to calculate the effective regurgitant orifice area (EROA):

\[
\text{EROA} = 2 \times \pi \times r^2 \times \frac{V_{\text{alias}}}{V_{\text{max}}}
\]

In our patient, radius was 0.9 cm, \(V_{\text{alias}}\) was 69 cm/s, and \(V_{\text{max}}\) was 420 cm/s:

\[
\text{EROA} = 2 \times 3.14 \times (0.9)^2 \times (69/420) = 0.8 \text{ cm}^2
\]

This EROA is very large (see Table 8-26) and indicative of severe mitral regurgitation. Therefore, answer E is correct.

32. Answer: A. Patient presented with severe acute mitral regurgitation 8 days after myocardial infarction in the territory of the left anterior descending artery that resulted in the rupture of the anterolateral papillary muscle. The course of events is consistent with the timeframe in which papillary muscle rupture, a mechanical complication of myocardial infarction, typically occurs.

These additional TEE images in Figure 8-39 further illustrate the case. (See also Video 7-3, ruptured anterolateral papillary muscle; TEE image at 0 degrees.)

Rupture of the anterolateral papillary muscle is less common than the rupture of the posteromedial one. Anterolateral papillary muscle usually has dual blood supply from both left anterior descending and left circumflex arteries. In contrast, posteromedial papillary muscle has solitary blood supply from either right coronary or left circumflex artery. Our patient had total proximal occlusion of the left anterior descending artery and diffuse disease in the left circumflex artery.

Answer B is incorrect because the clinical findings are inconsistent with bacteremia: the patient is afebrile and has a normal white blood cell count. In addition, a vegetation would appear as a shaggy,

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Peak E Velocity (cm/s)</th>
<th>Peak e' Velocity (cm/s)</th>
<th>PAWP = 1.9 + 1.24 × (E/e') (mm Hg)</th>
<th>PAWP = 4 + E/e' (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>45</td>
<td>5</td>
<td>13</td>
<td>13</td>
</tr>
<tr>
<td>B</td>
<td>60</td>
<td>5</td>
<td>17</td>
<td>16</td>
</tr>
<tr>
<td>C</td>
<td>150</td>
<td>5</td>
<td>39</td>
<td>34</td>
</tr>
</tbody>
</table>
independently mobile echo density attached typically to the atrial side of the mitral valve. The mass seen in this patient is attached to the mitral chordae and represents a severed head of the anterolateral papillary muscle.

Answer C is incorrect because mitral annular dilatation typically leads to mitral regurgitation with a central jet. In this patient, the jet is highly eccentric that is consistent with papillary muscle rupture.

Answer D is incorrect because rheumatic mitral valve disease is a chronic disorder that typically begins in childhood and progresses over many years. In our patient, the mitral valve was normal on initial admission and became severely regurgitant only days later. In addition, TEE imaging of the mitral valve in this patient lacks typical findings of rheumatic valve disease such as leaflet thickening and calcification, chordal fusion, and shortening.

Answer E is incorrect because mitral valve prolapse due to myxomatous generation is a chronic valvulopathy that would have been recognized on the initial echocardiogram at the time of first hospitalization. Mitral valve prolapse is characterized by floppy mitral leaflets that protrude into the left atrium above the mitral annular plane in systole due to leaflet and chordal elongation. Papillary muscle rupture is not a typical complication of mitral valve prolapse.

33. **Answer: C.** This patient has a patent ductus arteriosus (PDA) with a left-to-right shunt from the descending thoracic aorta to the left pulmonary artery throughout the cardiac cycle.

Using the spectral Doppler tracings of the PDA flow, one can calculate the peak systolic gradient (PSG) and end-diastolic gradient (EDG) across the PDA.

\[
\text{PSG} = 4 \times \text{PSV}^2 \\
\text{EDG} = 4 \times \text{EDV}^2
\]

where PSV is the peak systolic velocity and EDV is the end-diastolic velocity across the PDA.

In our patient:

\[
\text{PSG} = 4 \times (6.0 \text{ m/s})^2 = 4 \times 36 = 144 \text{ mm Hg} \\
\text{EDG} = 4 \times (3.8 \text{ m/s})^2 = 4 \times 14.4 = 58 \text{ mm Hg}
\]

By subtracting PSG and EDG from systolic and diastolic blood pressure, respectively, one can estimate pulmonary artery systolic blood pressure (PASP) and diastolic blood pressure (PADP).

\[
\text{PASP} = \text{SBP} - \text{PSG} \\
\text{PADP} = \text{DBP} - \text{EDG}
\]

In our patient:

\[
\text{PASP} = 170 - 144 = 26 \text{ mm Hg} \\
\text{PADP} = 70 - 58 = 12 \text{ mm Hg}
\]

Therefore, answer B is correct. This patient’s calculations are summarized in **Table 8-29**.

Answer A is incorrect because the right atrial pressure is not needed to calculate PADP in a patient with PDA when DBP and EDG are known.

Answer B is incorrect because a pulsed wave Doppler technique would not have been able to record such high velocities (including a peak velocity of 6 m/s) without aliasing in an adult.
Answer D is incorrect because PASP in this patient is 30 mm Hg as calculated earlier.

Answer E is incorrect because flow across an uncomplicated PDA occurs throughout the cardiac cycle irrespective of a PDA size. This is because in uncomplicated PDA, the pressures in the descending aorta are higher than the pressures in the pulmonary artery throughout the cardiac cycle.

34. Answer: B. PISA method can be used to estimate the effective orifice area (EOA) of the PDA at its aortic end:

\[
EOA = 2 \times \pi \times r^2 \times \frac{V_{alias}}{V_{max}}
\]

where \(r\) is the PISA radius, \(V_{alias}\) is the PISA aliasing velocity, and \(V_{max}\) is the peak systolic velocity across the PDA.

In our patient:

\[
EOA = 2 \times 3.14 \times (05 \text{ cm})^2 \times 41/500 = 0.13 \text{ cm}^2
\]

Note that the color bar baseline was shifted upward. Of the two Nyquist limits (41 cm/s for antegrade flow and 69 cm/s for retrograde flow), one should use the one in the direction of PDA flow, which is 41 cm/s.

Assuming a circular shape, the PDA orifice in this patient would then have a diameter of approximately 4 mm. The area (A) of a circle is calculated as follows:

\[
A = \left(\frac{d}{2}\right)^2 \times \pi
\]

where \(d\) is the PDA diameter. In our patient:

\[
0.13 = \left(\frac{d}{2}\right)^2 \times 3.14 = \frac{0.13}{3.14} = \left(\frac{d}{2}\right)^2
\]

Solving for diameter (\(d\)):

\[
d = 2 \times \sqrt{\frac{0.13}{3.14}} = 0.4 \text{ cm} = 4 \text{ mm}
\]

The diameter of a PDA usually ranges between 0.9 and 11.2 mm (median = 2.6 mm).

35. Answer: C. This patient has hypertrophic obstructive cardiomyopathy (HOCM) with asymmetric septal hypertrophy. Systolic anterior motion of the mitral leaflets in HOCM leads to (1) dynamic left ventricular outflow tract (LVOT) obstruction and (2) mitral regurgitation. Both the gradient across the LVOT and the gradient across the mitral valve peak late in systole.

One can calculate the peak systolic LVOT gradient from the following three parameters: peak gradient of mitral regurgitant (MR) jet, left atrial pressure, and systolic blood pressure.

Step 1: Calculate the peak systolic LV-to-LA gradient

Using the peak velocity of the MR jet, one can calculate the peak systolic pressure gradient (\(\Delta P_{MR}\)) between the left ventricle (LV) and the left atrium (LA):

\[
\Delta P_{MR} = 4V^2
\]

where \(V\) is the peak velocity of the MR jet.

In our patient:

\[
P_{MR} = 4 \times (8 \text{ m/s})^2 = 4 \times 64 = 256 \text{ mm Hg}
\]

Step 2: Calculate the peak LV systolic pressure (LVSP)

By definition, \(\Delta P_{MR}\) is the difference between the peak LVSP and the LA pressure (LAP):

\[
\Delta P_{MR} = \text{LVSP} – \text{LAP}
\]

Solving for LVSP:

\[
\text{LVSP} = \Delta P_{MR} + \text{LAP}
\]

In our patient:

\[
\text{LVSP} = 256 \text{ mm Hg} + 10 \text{ mm Hg} = 266 \text{ mm Hg}
\]

Step 3: Calculate maximal instantaneous left ventricular outflow gradient (\(\Delta P_{LVOT}\))

\(\Delta P_{LVOT}\) is the pressure difference between the LVSP and the systolic blood pressure (SBP):

\[
\Delta P_{LVOT} = \text{LVSP} – \text{SBP}
\]

In our patient:

\[
\Delta P_{LVOT} = 266 \text{ mm Hg} – 144 \text{ mm Hg} = 122 \text{ mm Hg}
\]

Therefore, answer C is correct. All these calculations are summarized in Figure 8-40A.
Answer A is incorrect because in HOCM, mitral regurgitation increases progressively toward mid to late systole. MR in HOCM is the result of systolic anterior motion (SAM); the anterior leaflet moves progressively toward the interventricular septum and away from the coaptation line with the posterior leaflet. This results in an MR velocity profile that peaks late in systole. Therefore, in our patient, the initial portion of the MR jet is not missing from the Doppler tracing; the Doppler velocity profile is typical for HOCM-related MR.

Answer B is incorrect because LVSP is very high. It is calculated above at 266 mm Hg. Normal LVSP is the same as the normal SBP, which is around 120 mm Hg.

Answer D is incorrect because there is no diastolic MR in this patient. Typically, MR is a systolic phenomenon. In rare instances, MR can start in late diastole (diastolic MR) and continue into systole.

Diastolic MR may occur in severe LV systolic dysfunction or with complete heart block.

Different MR velocity profiles are summarized in Figure 8-40B. Answer E is incorrect because the peak LV systolic pressure in this patient is 266 mm Hg as calculated earlier.

36. Answer: C. In this patient with hypertrophic obstructive cardiomyopathy (HOCM), jet #1 represents the systolic flow velocity pattern across the left ventricular outflow tract (LVOT), and jet #2 represents the flow velocity pattern of the mitral regurgitant (MR) jet.

Jet #1 has a saw tooth appearance because the gradient characteristically peaks late in systole. The systolic anterior motion of the mitral valve in HOCM progressively narrows the LVOT toward the end of systole. This in turn results in ever-increasing systolic blood velocities through the LVOT and the late peaking velocity profile typical of HOCM.

Using the $\Delta P = 4V^2$ formula, we can calculate peak systolic instantaneous gradient ($\Delta P_{LVOT}$) across the LVOT where $V$ represents the peak velocity of jet #1.

$\Delta P_{LVOT} = 4 \times (3.8 \text{ m/s})^2 = 4 \times 14.4 = 58 \text{ mm Hg}$

Since the pretreatment $\Delta P_{LVOT}$ was 120 mm Hg, there was an approximately 50% drop in the gradient on the repeat echocardiogram:

Percent drop in $\Delta P_{LVOT}$

$= (122 - 58)/122 = 64/122 = 50\%$

Therefore, answer C is correct.

Answer A is incorrect because in mitral valve prolapse with click and systolic murmur, mitral regurgitation characteristically does not occur in early systole. The prolapse usually does not create a regurgitant
orifice until mid systole. Once the regurgitant orifice is created, mitral regurgitation continues until the end of systole. The difference in the shape of the MR spectral jet between mitral valve prolapse and HOCM is depicted in Figure 8-41A.

Pulsed wave spectral Doppler tracing of a left ventricular intracavitary gradient.

Figure 8-41B

Answer B is incorrect because jet #2 starts immediately after the QRS complex on the EKG. Therefore, the jet encompasses the isovolumic contraction time. Aortic stenosis flow does not occur in that early portion of systole.

Answer D is incorrect because an intracavitary left ventricular gradient tapers off and peaks even later in systole than the LVOT gradient as shown in Figure 8-41B.

Answer E is incorrect because the peak left ventricular systolic pressure (LVSP) is calculated as follows:

\[
LVSP = \Delta P_{MR} + LAP
\]

where \(\Delta P_{MR}\) is the PSG of the MR jet and LAP is the LAP. After expressing \(\Delta P_{MR}\) in terms of the peak systolic velocity \(V\) of the MR jet, Eq. 1 becomes:

\[
LVSP = 4 \times V^2 + LAP
\]

In our patient:

\[
LVSP = 4 \times (6.3 \text{ m/s})^2 + 10
\]

\[
LVSP = 159 + 10 = 169 \text{ mm Hg}
\]

37. **Answer: D.** Left atrial volume (LAV) can be calculated using the area-length method. The mathematical formula requires three parameters: left atrial area in the apical 4-chamber view \((A1)\), left atrial area in the apical 2-chamber view \((A2)\), and the shorter of the two atrial lengths \((L)\) whether it be in the apical 4- or apical 2-chamber view.

\[
LAV = \frac{8 \times A1 \times A2}{3 \times \pi \times L}
\]

The formula can be simplified by calculating the \(8/3\pi\) ratio as 0.85:

\[
LAV = 0.85 \times \frac{A1 \times A2}{L}
\]

In our patient:

\[
LAV = 0.85 \times \frac{27 \times 26}{5.6} = 107 \text{ mL}
\]

LAV index (LAVI) is calculated by dividing LAV into the body surface area (BSA):

\[
LAVI = \frac{LAV}{BSA}
\]

In our patient:

\[
LAVI = \frac{107 \text{ mL}}{2.1 \text{ m}^2} = 50 \text{ mL/m}^2
\]

Therefore, answer D is correct. This is a severely elevated LAVI (see reference table below).

38. **Answer: B.** The initial echocardiogram, which was obtained at the time of acutely decompensated heart failure, demonstrates a restrictive filling pattern. Because of the high left atrial pressure, the early diastolic gradient across the mitral valve is high. This results in a tall mitral E wave and the ratio of peak mitral E to peak mitral A wave that is usually \(>2\). In addition, the mitral E wave has rapid deceleration (deceleration time \(<160 \text{ ms}\) ). In the pulmonary venous spectral Doppler tracings, the peak of the systolic \((S)\) wave is lower than the peak of the diastolic

| Table 8-30 |
|---|---|
| **LA Volume Index (mL/m²)** | |
| Normal | \(\leq 28\) |
| Mild dilatation | 29–33 |
| Moderate dilatation | 34–39 |
| Severe dilatation | \(\geq 40\) |
(D) wave. The height of the S wave is inversely related to the left atrial pressure. All these findings in mitral and pulmonary vein pulsed wave Doppler tracings are consistent with the restrictive filling pattern.

With appropriate medical treatment, including diuretics, left atrial pressure decreases and the mitral inflow reverts to the pattern of abnormal relaxation common in the patient's age group. The pattern is characterized by an E < A pattern in the mitral inflow and a prolonged deceleration time of the mitral E wave. In the pulmonary veins, the peak velocity of the S wave now exceeds the peak velocity of the D wave (S > D), reflective of lower left atrial pressures. Therefore, answer B is correct. Different mitral and pulmonary vein filling patterns as well as their relationship to mean left atrial pressure are summarized in Figure 8-42.

Answer A is incorrect because the preload has decreased from the initial to subsequent study as judged by the decrease in the mean left atrial pressure. Answer C is incorrect because the initial filling pattern was not normal; it was restrictive. A normal pattern cannot be distinguished from pseudonormal pattern by mitral and pulmonary flow patterns alone. Ancillary data such as the peak velocity of the mitral annular tissue Doppler e' prime wave is required to distinguish normal (e' > 8 cm/s) from pseudonormal pattern (e' < 8 cm/s).

Answer D is incorrect because the presence of a prominent and normally timed A wave in mitral inflow and the S wave in the pulmonary vein argue against atrial arrhythmias such as atrial flutter or atrial fibrillation. In these atrial arrhythmias, the peak velocities of the mitral A wave and the pulmonary vein S wave are greatly diminished.

Answer E is incorrect because it is the persistence of the restrictive pattern despite appropriate medical therapy that portends a grave prognosis, the prognosis is grave with a 2-year mortality estimated at 50% in patients with left ventricular ejection fraction of <40%. In this patient, the change from the restrictive filling to the abnormal relaxation pattern actually portends a better prognosis.

39. **Answer: D.** The three recordings from this patient are consistent with the diagnosis of constrictive pericarditis.

1. **MITRAL INFLOW:** The mitral inflow spectral Doppler tracings demonstrate marked respiratory variations in the mitral E wave velocities. Such a finding would be consistent with either constrictive pericarditis or tamponade, as well as obesity, labored breathing, asthma, chronic obstructive lung disease, etc. However, in each cardiac cycle, the peak velocity of the mitral E wave is larger than that of the mitral A wave (E > A). This indicates that there is no impediment to early mitral filling that would be consistent with constrictive pericarditis. In contrast, tamponade is characterized by impediment in early diastolic filling and an E < A.

2. **COLOR M-MODE:** The flow propagation velocity (Vp) of the early diastolic propagation velocity is normal against atrial arrhythmias such as atrial flutter or atrial fibrillation. In these atrial arrhythmias, the peak velocities of the mitral A wave and the pulmonary vein S wave are greatly diminished.

### Table: Filling Pattern and Typical Mean LA Pressure

<table>
<thead>
<tr>
<th>Filling Pattern</th>
<th>Mitral Inflow</th>
<th>Pulmonary Vein</th>
<th>Typical Mean LA Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal Relaxation</td>
<td>![Image]</td>
<td></td>
<td>8 – 14 mm Hg</td>
</tr>
<tr>
<td>Pseudonormalization</td>
<td>![Image]</td>
<td></td>
<td>15 – 22 mm Hg</td>
</tr>
<tr>
<td>Restrictive Filling</td>
<td>![Image]</td>
<td></td>
<td>&gt; 22 mm Hg</td>
</tr>
</tbody>
</table>

**Figure 8-42**

LWBK1563-c08_p110-155.indd   153 28/06/16   1:12 PM
Normal $V_p$ values are age dependent as shown in the table below.

**Table 8-31**

<table>
<thead>
<tr>
<th>Age</th>
<th>Normal $V_p$ (cm/s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young</td>
<td>&gt;55</td>
</tr>
<tr>
<td>Elderly</td>
<td>&gt;45</td>
</tr>
</tbody>
</table>

$V_p$ measures the rate of left ventricular myocardial relaxation. The faster the rate of myocardial relaxation, the higher the $V_p$ is. Typically, there is no significant myocardial involvement in constrictive pericarditis, and $V_p$ is normal. This is in contrast to restrictive cardiomyopathy that is a myocardial disorder characterized by impaired relaxation and compliance. In restrictive cardiomyopathy, $V_p$ is low.

3. INFERNOR VENA CAVA: In constrictive pericarditis, there is plethora of the IVC as demonstrated by M-mode recordings of the IVC in this patient. The IVC is dilated (2.43 cm in expiration) and collapses less than 50% with inspiration (inspiratory diameter of IVC = 1.97 cm). The finding is indicative of an elevated right atrial pressure (RAP, 11–15 mm Hg). Such a finding is consistent with the diagnosis with constrictive pericarditis. However, IVC plethora is also found in other conditions of elevated RAP such as tricuspid stenosis, severe tricuspid regurgitation, and right ventricular infarct. Therefore, answer D is correct.

Answer A is incorrect because in restrictive cardiomyopathy there are no marked respiratory variations in the mitral E wave velocities. In addition, $V_p$ is low in restrictive cardiomyopathy.

Answer B is incorrect because the IVC plethora is indicative of an elevated RAP.

Answer C is incorrect because $V_p$ in this patient is normal (>55 cm/s).

Answer E is incorrect because there are marked respiratory variations (>30%) in the peak velocity of the mitral E wave.

40. **Answer: D.** The patient has constrictive pericarditis. With each inspiration, the filling of the right ventricle increases and the filling of the left ventricle decreases.

The characteristic movement of the interventricular septum that is phasic with respiration occurs in both tamponade and constrictive pericarditis. The absence of pericardial effusion on the apical 4-chamber view argues against the diagnosis of tamponade.

The abnormal septal motions stated in the remaining four answers are not phasic with respiration. Their characteristics are summarized in the table below.

**Table 8-32**

<table>
<thead>
<tr>
<th>Right ventricular pressure overload</th>
<th>Interventricular septum flattens in systole and diastole. In the short axis, left ventricular contour becomes D shaped rather than circular in both systole and diastole.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right ventricular volume overload</td>
<td>Interventricular septum flattens in diastole. In the short axis, left ventricular contour becomes D shaped rather than circular during diastole.</td>
</tr>
<tr>
<td>Left bundle branch block</td>
<td>Interventricular septum moves posteriorly in the preejection period, and then moves anteriorly (away from the posterior left ventricular wall) during ejection phase of systole.</td>
</tr>
<tr>
<td>Cardiac surgery</td>
<td>Movement of the interventricular septum toward the right ventricle rather than the left ventricle in systole, with normal thickening.</td>
</tr>
</tbody>
</table>

**SUGGESTED READINGS**


AQ1: Please check whether it is OK to change the question to “Which of the following statements is TRUE?”
AQ2: After the placement of Table 8-15, tables have been renumbered. Please verify the renumbering.
AQ3: Please verify the citations of Figures 8-8A and B as per the previous edition.
AQ4: Please verify the figure numbering in the answers.
AQ5: Should the words “Eq. 5” be changed to “Eq. 3” here?
AQ6: Please verify the value of PVR. The answer should be 2.5, not 3.4 Wood units.
AQ7: Some of the equations are in bold face in the manuscript. We have retained these equations as such. Please check if this is OK.
AQ8: The sentence (Answer E is incorrect …) is not complete. Please check.
AQ9: Two equations are given in Eq. 7. Please check.
AQ10: Please check the value 57 here.
AQ11: Please check the value of AVA in the equation. The answer should be 0.24, not 0.75.
AQ12: Please verify the placement of Figure 8-34.
AQ13: Please verify the equation (Peak trans…..) here.
AQ14: Only two tables are cited in the text. Please check if this is OK.
AQ15: Please check the spelling “chordal.”
AQ16: Please cite Figure 8-16 in the text.