CHAPTER 7

Doppler and Hemodynamics

Muhamed Saric and Itzhak Kronzon

1. On echocardiography, the diameter of the inferior vena cava is measured at 1.6 cm during expiration and 0.6 cm after the patient is asked to sniff. The right atrial pressure is estimated at:
A. 0–5 mm Hg.
B. 6–10 mm Hg.
C. 11–15 mm Hg.
D. 16–20 mm Hg.
E. 20 mm Hg.

2. 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 7-1

E-wave deceleration time	910 milliseconds
Mean diastolic mitral gradient	17 mm Hg
Diastolic mitral inflow velocity-time integral	66 cm
Heart rate	85 bpm

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 milliseconds.
D. Mitral valve area is 0.8 cm^2
E. During exertion, her mean gradient is expected to decrease.

3. 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 (PDA) and the following:

TABLE 7-2

Left ventricular outflow tract (LVOT) diameter	2.0 cm
LVOT velocity-time integral	31 cm
Right ventricular outflow tract (RVOT) diameter	2.5 cm
RVOT velocity-time integral	12 cm
Heart rate	80 bpm

The following statement is TRUE:
A. Systemic blood flow (*Q*s) is 7.8 l/minute.
B. The ratio of pulmonic to systemic blood flow (*Q*p:*Q*s) is less than one.
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 left ventricular outflow tract (LVOT) and the stroke volume through the right ventricular outflow tract (RVOT) is equal to the *Q*p:*Q*s ratio in this patient.

4. 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 7-3

Early diastolic peak velocity	3.0 m/sec
End-diastolic velocity	2.0 m/sec

Examination of the inferior vena cava by M-mode echocardiography demonstrated the following:

TABLE 7-4

IVC diameter during expiration	2.6 cm
IVC diameter during inspiration	2.6 cm

The following statement is TRUE:

A. Right atrial pressure 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.

5. 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 color Doppler imaging, a well-formed flow convergence proximal isovelocity surface area (PISA) shell was visualized on the ventricular side of the mitral valve in systole. In addition, the following was noted:

TABLE 7-5

Maximal mitral regurgitation PISA radius	1.0 cm
Aliasing velocity at which PISA radius measured	45 cm/sec
Peak velocity of mitral regurgitation jet	500 cm/sec
Velocity-time integral of mitral regurgitation	140 cm

The following statement is TRUE:

A. Vena contract 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 per second.
D. Mitral regurgitation is moderate (2+).
E. Regurgitant volume is 40 ml/beat.

6. 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 7-6

Peak velocity of the mitral E wave	125 cm/sec
Flow propagation velocity of mitral inflow on color M mode	31 cm/sec
Peak velocity of tricuspid regurgitant jet	4 m/sec
Estimated right atrial pressure	15 mm Hg

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.

7. 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 contract obtained by 2D echocardiography can be used to calculate regurgitant volume.

8. A 62-year-old man with history of treated hypertension, chronic atrial fibrillation, and bicuspid aortic valve had transthoracic echocardiogram done. The study showed the following:

TABLE 7-7

Peak velocity of mitral regurgitant jet	6.0 m/sec
dP/dt of mitral regurgitant jet	1,900 mm Hg/sec
Ratio of peak mitral E wave to peak velocity of medial mitral annulus (E/e')	16
Vena contracta of mitral regurgitation	0.2 cm

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 the E/e' method in patients with atrial fibrillation.

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

TABLE 7-8

Peak diastolic velocity of aortic regurgitant jet	5.0 m/sec
End-diastolic velocity of aortic regurgitant jet	3.7 m/sec
Pressure half-time of aortic regurgitant jet	656 milliseconds
Peak aortic antegrade flow velocity	2.2 m/sec
Blood pressure	130/65 mm Hg

Based on the above 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 is lower than the systolic blood pressure.
D. Left ventricular end-diastolic pressure is estimated at 10 mm Hg.
E. Aortic valve area cannot be calculated using continuity equation because there is aortic regurgitation.

10. 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 7-9

Pulmonary artery systolic pressure	65 mm Hg
Pulmonary artery diastolic pressure	35 mm Hg
Left atrial pressure	10 mm Hg
Right ventricular outflow tract (RVOT) diameter	2.6 cm
RVOT velocity-time integral	30 cm
Left ventricular outflow tract (LVOT) diameter	2.0 cm
LVOT velocity-time integral	20 cm
Heart rate	75 bpm

Based on the above 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.

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

TABLE 7-10

Blood pressure	120/80 mm Hg
Peak systolic velocity across the VSD	3.0 m/sec
End-diastolic velocity across the VSD	1.0 m/sec
Estimated right atrial pressure	10 mm Hg
Peak systolic gradient across pulmonic valve	55 mm Hg
Left ventricular end-diastolic pressure	12 mm Hg

The following statement is true:

A. Right ventricular systolic pressure is 46 mm Hg.
B. Pulmonary artery systolic pressure is 29 mm Hg.
C. Right ventricular systolic pressure is 84 mm Hg above the right atrial pressure.
D. Pulmonary artery systolic pressure is 45 mm Hg higher than the right ventricular systolic pressure.
E. Right ventricular end-diastolic pressure is 28 mm Hg.

12. 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 ASD that was circular in shape. On color Doppler, 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 7-11

Blood pressure	120/80 mm Hg
Heart rate	100 bpm
PISA radius	0.7 cm
Velocity-time integral of left-to-right flow across ASD	80 cm
Left ventricular outflow tract (LVOT) diameter	2.0 cm
LVOT velocity-time integral	19 cm

The following statement is true:

A. Ratio of pulmonic to systemic flow (*Q*p:*Q*s) is 1.8:1.0.
B. Shunt flow across the ASD is approximately 9.0 l/minute.
C. The difference between the pulmonic and systemic stroke volume is 180 ml.
D. Systemic stroke volume is 150 ml.
E. Pulmonic blood flow (*Q*p) is approximately 7.0 l/min.

13. A 35-year-old woman presented 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 at the time of transthoracic echocardiogram:

TABLE 7-12

Blood pressure	95/50 mm Hg
Heart rate	120 bpm
Peak velocity of mitral regurgitant jet	4.0 m/sec
Time interval from onset of mitral regurgitation to jet velocity of 1 m/sec	5 milliseconds
Time interval from onset of mitral regurgitation to jet velocity of 3 m/sec	25 milliseconds
Vena contract of mitral regurgitation	0.8 cm

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.

14. A 29-year-old Bangladeshi woman with rheumatic mitral stenosis is referred to cardiac catheterization lab for percutaneous mitral balloon valvuloplasty. Upon placement of the pigtail catheter in the left ventricle, the following values were obtained:

TABLE 7-13	
Left ventricular peak systolic pressure	124 mm Hg
Early left ventricular diastolic pressure	7 mm Hg
Left ventricular end-diastolic pressure	10 mm Hg

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

TABLE 7-14	
Heart rate	104 bpm
Time-velocity integral of diastolic mitral flow	65 cm
Mean mitral valve gradient in diastole	21 mm Hg
Mitral pressure half-time	270 milliseconds

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 may be unreliable in patients prior to valvuloplasty.
D. Mitral valve area is 0.6 cm^2.
E. Mean left atrial pressure is approximately 28 mm Hg.

15. An 81-year-old woman with systolic heart murmur was referred for echocardiogram. A heavily calcified aortic valve and normal mitral valve were noted on 2D imaging. Doppler echocardiography of the aortic valve revealed:

TABLE 7-15	
Left ventricular outflow tract (LVOT) diameter	1.9 cm
Peak velocity across the aortic valve	5.0 m/sec
Peak LVOT velocity	1.0 m/sec
LVOT velocity-time integral (VTI)	20 cm

The following statement is true:

A. Aortic valve area 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 cm^2.
D. Left ventricular stroke volume is 80 ml per beat.
E. Systolic blood pressure is approximately 100 mg Hg above the left ventricular systolic pressure.

16. This continuous-wave spectral Doppler tracing of the tricuspid regurgitant jet comes from a 18-year-old woman with pulmonic valve stenosis (Fig. 7-1). 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:

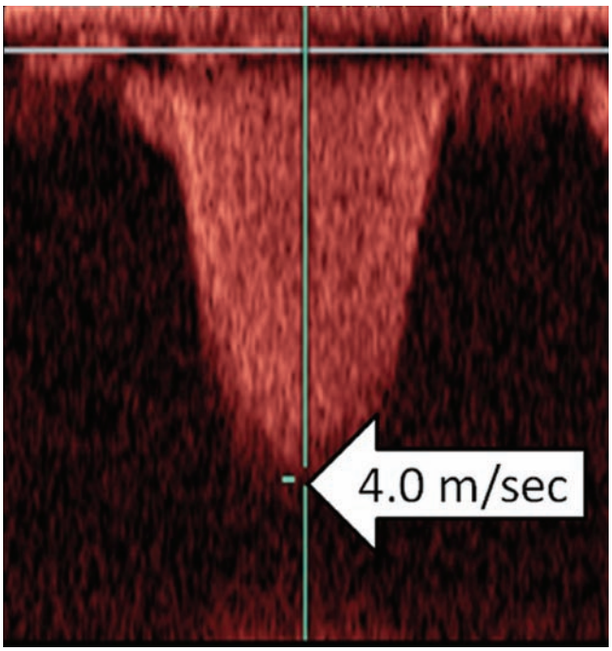


Fig. 7-1

A. Peak pulmonary artery systolic pressure is higher than the right ventricular peak systolic pressure.
B. Right ventricular peak systolic pressure is 64 mm Hg above than 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.
E. Right ventricular peak systolic pressure is 108 mm Hg.

17. An 82-year-old man was referred for evaluation of systolic ejection murmur. On parasternal long-axis view, the left ventricular outflow tract diameter was measured at 2.0 cm.

Spectral Doppler tracings were obtained in or through the left ventricular outflow tract in the apical 5-chamber view (Fig. 7-2).

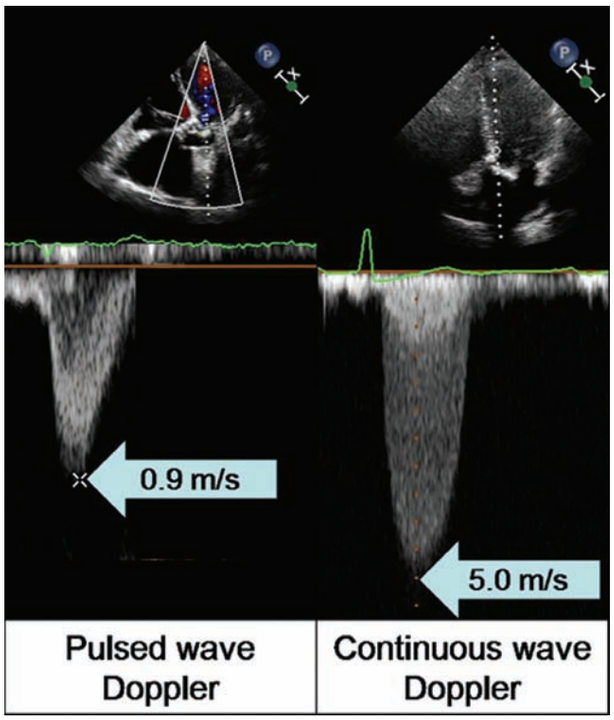


Fig. 7-2

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 is greater than 1.0 cm^2.
E. Patient has hypertrophic obstructive cardiomyopathy (HOCM).

18. The continuous-wave spectral Doppler tracing in Figure 7-3, from a 21-year-old woman represents the flow velocity profile in the main pulmonary artery. Based on this tracing, the following is TRUE about this patient:

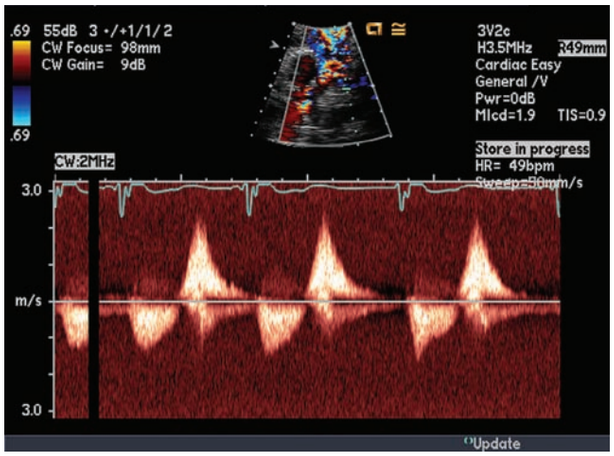
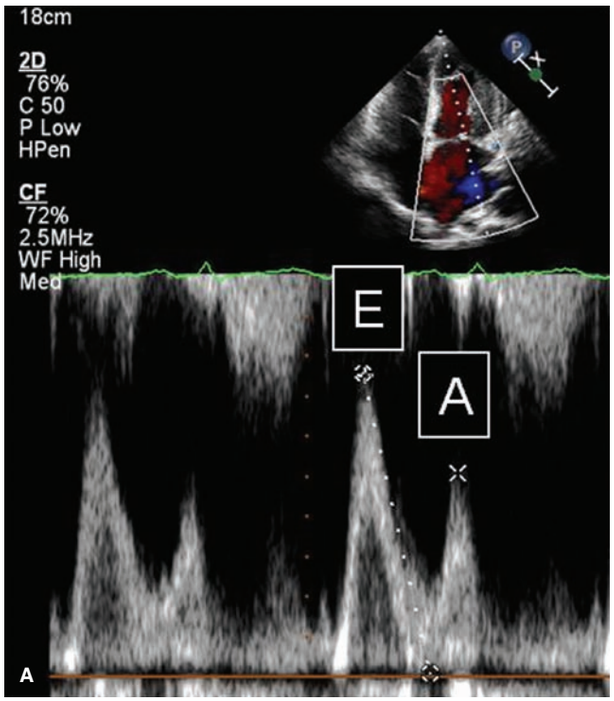


Fig. 7-3

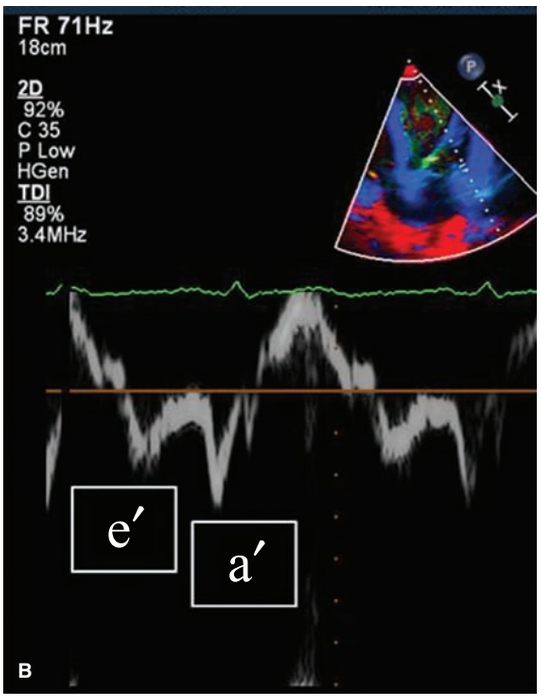
A. End-diastolic gradient across the pulmonic valve is high.
B. There is severe pulmonic valve stenosis.
C. Pulmonary artery systolic pressure is 9 mm Hg above the right ventricular pressure.
D. Pulmonic valve regurgitation is severe.
E. The velocity profile is diagnostic of PDA.

19. The tracings in Figure 7-4 were obtained from an 82-year-old woman with a normal left ventricular ejection fraction of 65%. Figure 7-4A represents



MITRAL INFLOW
Peak E wave velocity = 142 cm/s
E wave deceleration time = 148 msec

Fig. 7-4A



LATERAL MITRAL ANNULUS
Peak e′ velocity = 8 cm/sec
Peak a′ velocity = 10 cm/sec

Fig. 7-4B

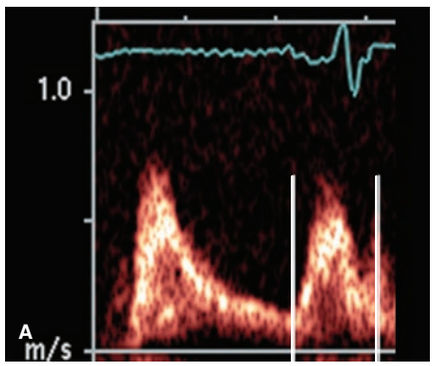
blood flow velocity pattern obtained by placing a pulsed Doppler sample volume at the mitral leaflet tips. Figure 7-4B represents tissue Doppler of the lateral mitral annulus.

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.

20. Figure 7-5A and B were obtained from the same patient at the same heart rate.

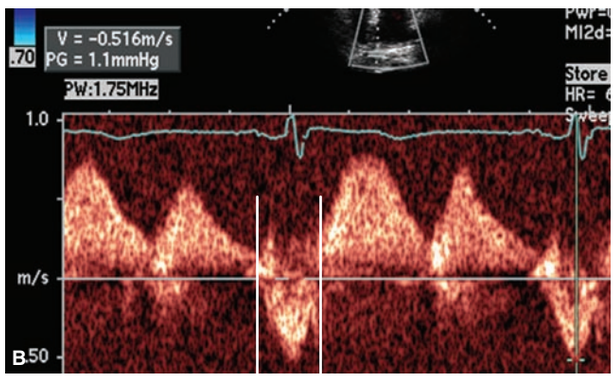
MITRAL INFLOW



Mitral A wave duration = 170 msec

Fig. 7-5A

PULMONARY VENOUS FLOW



Atrial reversal wave
Duration = 210 msec; peak velocity 50 cm/sec

Fig. 7-5B

The following statement is TRUE:

A. Mitral inflow pattern is diagnostic of restrictive filling.
B. Left ventricular end-diastolic pressure 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.

21. Upward deflection in respirometry recordings indicates inspiration while the downward deflection indicates expiration (Fig. 7-6).

M Mode Recording in Short Axis at Papillary Muscle Level

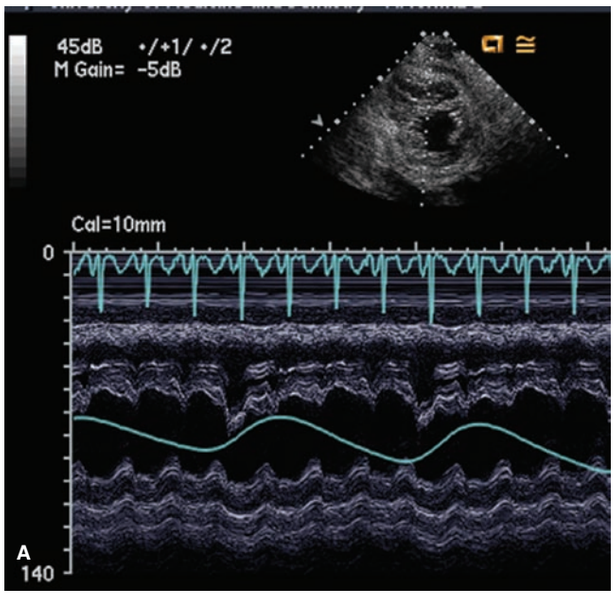


Fig. 7-6A

Hepatic Vein Pulsed Doppler

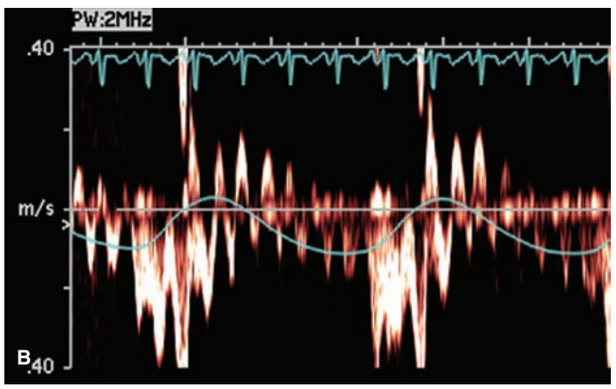
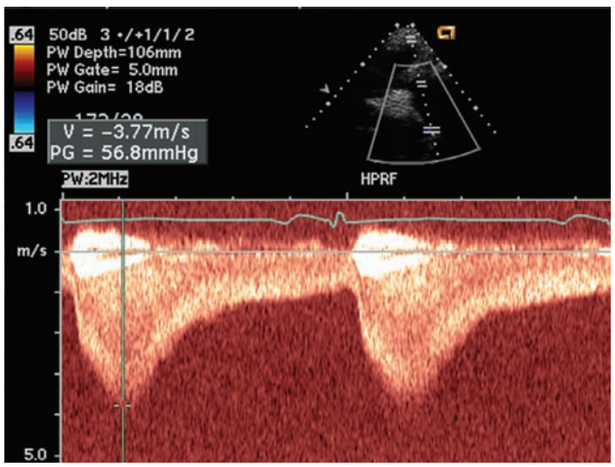


Fig. 7-6B

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. Inspiratory increase in antergrade hepatic vein flow velocities is abnormal.
E. Above M-mode recordings are diagnostic of a large pericardial effusion and tamponade.

22. A 33-year-old man has had murmur since childhood. These transthoracic spectral Doppler tracings in Figure 7-7 are obtained from the suprasternal view.



Peak systolic velocity	3.77 m/sec
End-diastolic velocity	1.0 msec

Fig. 7-7

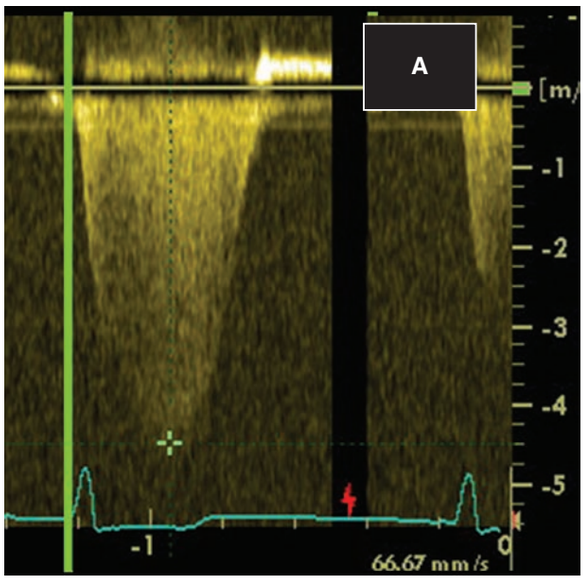
The following statement is TRUE:

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 in the arms.

23. A 91-year-old woman presents with severe shortness of breath. The two spectral Doppler recordings in Figure 7-8 were obtained from two different valves. Vertical line in each tracing marks the onset of QRS.

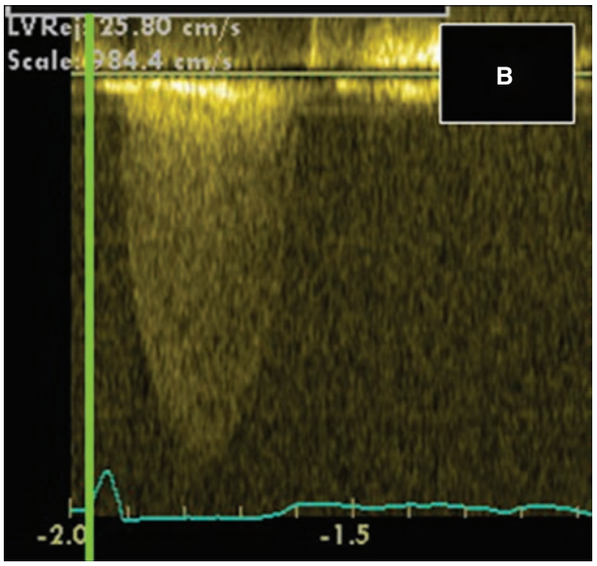
The following statement is TRUE:

A. Figure 7-8B represents tricuspid regurgitant jet and the patient has severely elevated right ventricular systolic pressure.
B. Figure 7-8A represents severe aortic stenosis because the jet starts during isovolumic contraction period.
C. The jet with the shorter duration represents aortic stenosis.



Peak velocity = 4.5 m/sec
Jet duration = 515 msec

Fig. 7-8A



Peak velocity = 5.0 m/sec
Jet duration = 345 msec

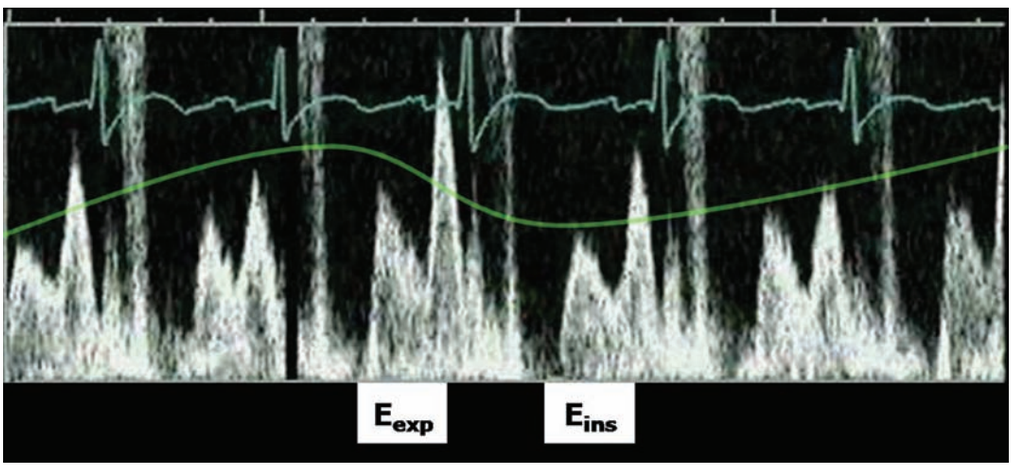
Fig. 7-8B

D. Peak velocity of 5.0 m/sec in Figure 7-8B is not compatible with a tricuspid regurgitant jet.
E. Systolic function of both ventricles is severely diminished.

24. 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. These spectral pulsed Doppler recordings in Figure 7-9 were obtained at mitral leaflet tips. Upward deflection in respirometry recordings above indicates inspiration while the downward deflection indicates expiration.

The following statement is TRUE:

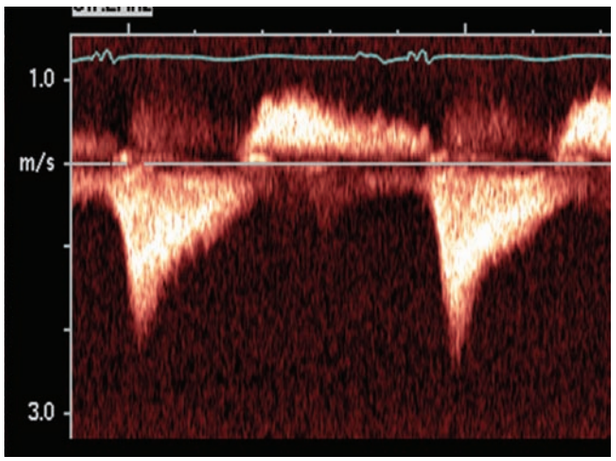
A. Respiratory variations in peak velocity of late diastolic flow (A wave) of more than 25% favor constriction over tamponade.
B. Marked decrease in peak E-wave velocity seen at the onset of inspiration is consistent with the diagnosis of tamponade.
C. Findings are characteristic of restrictive cardiomyopathy.
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.



Expiratory peak E wave velocity (E_{exp}) = 170 cm/sec
Inspiratory peak E wave velocity (E_{ins}) = 110 cm/sec
E wave deceleration time = 260 msec

Fig. 7-9

25. 28-year-old man with liver disease presents with jugular venous distensions (Fig. 7-10).



Peak velocity of tricuspid regurgitant jet = 2.2 m/sec

Fig. 7-10

The following statement is TRUE:

A. Right atrial pressure rises progressively towards the end of ventricular systole.
B. Right ventricular systolic function is markedly diminished.
C. Peak velocity of 2.2 m/sec excludes the diagnosis of pulmonary hypertension.
D. Tricuspid regurgitation is likely mild.
E. There is right ventricular midcavitary gradient during systole.

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.

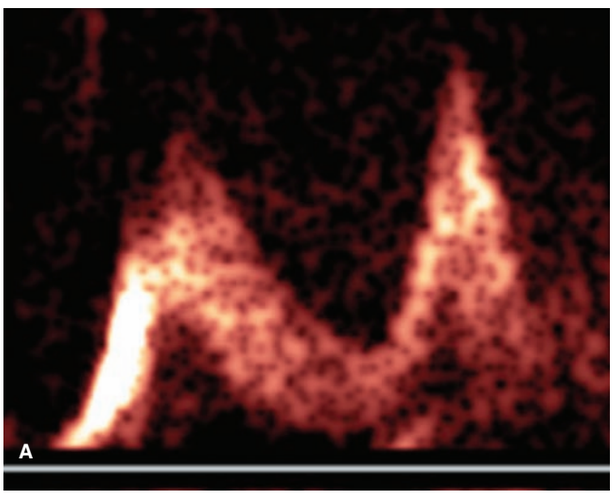
Electrocardiogram in the emergency department revealed normal sinus rhythm, right bundle branch block, and ST elevations in anteroseptal leads.

Blood pressure 90/50 mm Hg; heart rate 100 beats per minute; oral temperature 98.7 degrees. On auscultation of the lungs, rales were noted bilaterally throughout the lung fields. The heart exam 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.

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.

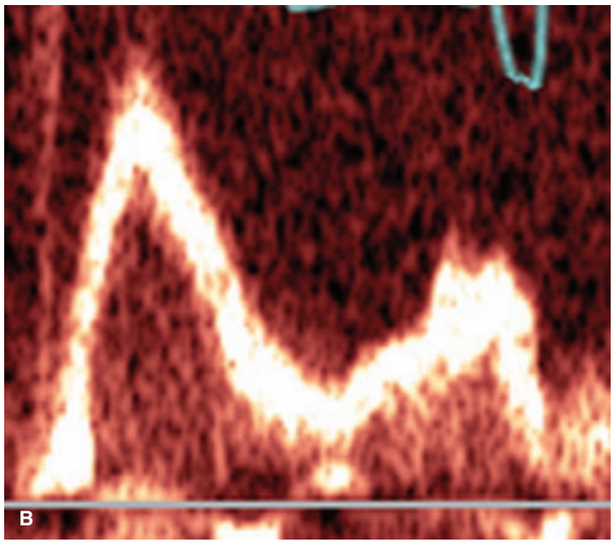
26. The patient was transferred to the intensive care unit where a Swan-Ganz catheter was placed. Pulmonary artery wedge pressure was 38 mm Hg. Tissue Doppler of the medial mitral annulus and pulsed Doppler recordings with the sample volume at the tips of the mitral valve leaflets were obtained at that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

A. Figure 7-11A.
B. Figure 7-11B.
C. Figure 7-11C.
D. Figure 7-11D.
E. Figure 7-11E.



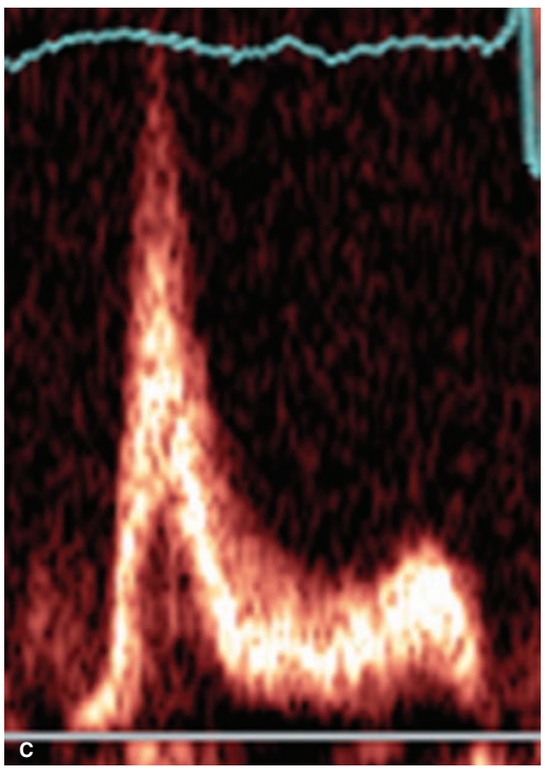
Peak E wave velocity = 45 cm/sec

Fig. 7-11A



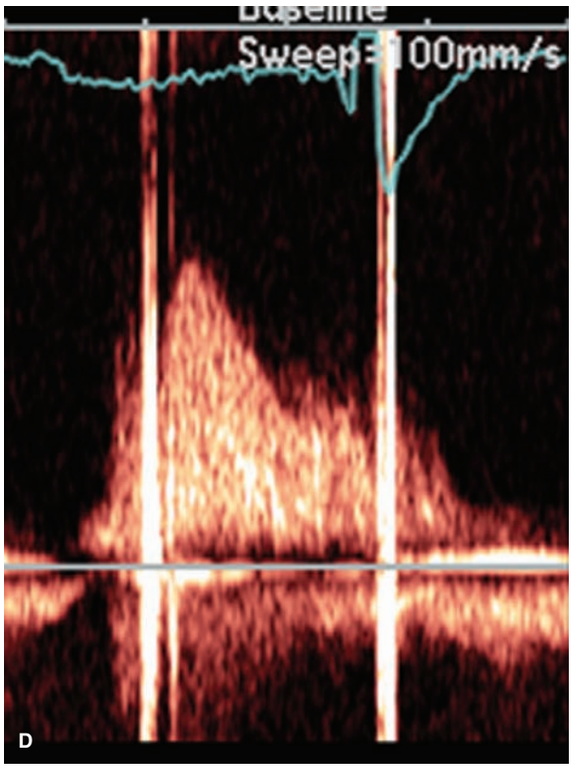
Peak E wave velocity = 60 cm/sec

Fig. 7-11B



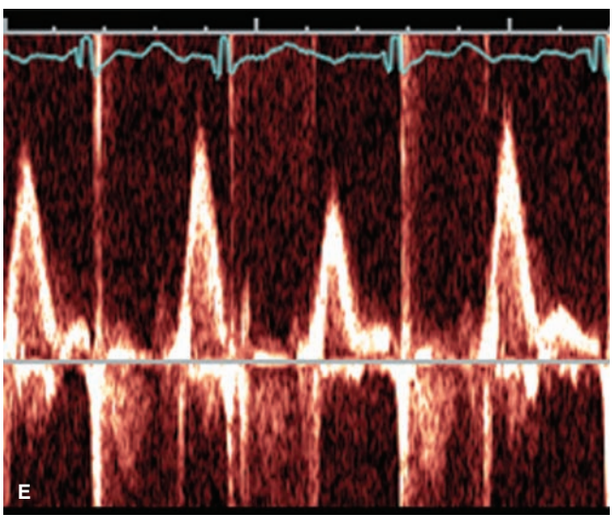
Peak E wave velocity = 150 cm/sec

Fig. 7-11C



Peak E wave velocity = 200 cm/sec

Fig. 7-11D



Peak E wave velocity varies from beat to beat (between 60 and 80 cm/sec)

Fig. 7-11E

27. From the emergency department, she was taken for coronary angiogram which revealed total occlusion of the proximal anterior descending artery and diffuse atherosclerosis in the left circumflex artery. Percutanous 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 five.

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 in the emergency department demonstrated massive bilateral pulmonary edema. The following data were obtained by echocardiography the same day. (See Figure 7-12 and Video 7-1.)

The degree of mitral regurgitation is:

A. Trivial.
B. Mild (1+).
C. Moderate (2+).
D. Moderate to severe (3+).
E. Severe (4+).

28. 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.

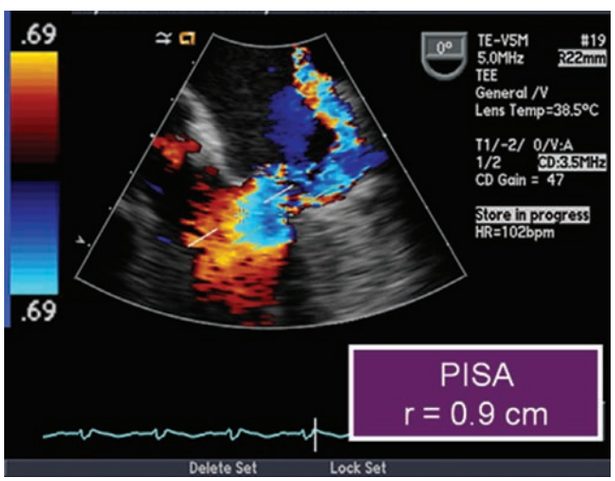
CASE 2:

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 exam, his blood pressure is 170/70 mm Hg; heart rate 72 beats per minute 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 obsured by the continuous, machinery-type murmur best heard in the left upper chest. There is no peripheral edema.

Echocardiography revealed PDA, normal left ventricular systolic function, no valvular disease, and no hypertrophic cardiomyopathy. Right atrial pressure is estimated at 10 mm Hg.

TEE image of mitral valve in systole in midesophageal view



Color Doppler demonstrating PISA on the left ventricular side of the mitral valve; radius is 0.9 cm

Peak systolic velocity of mitral regurgitant jet 4.2 m/sec

Fig. 7-12

29. The spectral Doppler tracing in Figure 7-13 represents flow across the PDA obtained by transthoracic echocardiography.

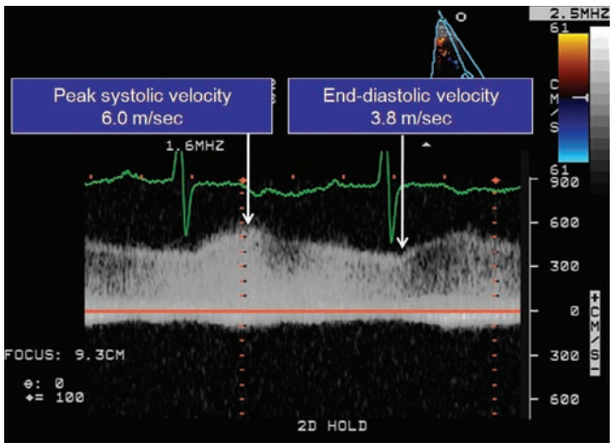


Fig. 7-13

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. PDA is very large because the flow occurs throughout the cardiac cycle.

30. This transthoracic echocardiographic color Doppler image in the parasternal short-axis view at the level of the PDA comes from the same study as the spectral tracing in previous question (Fig. 7-14).

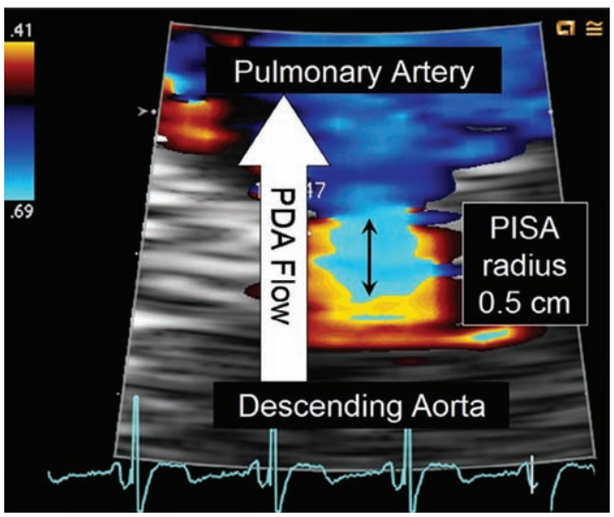


Fig. 7-14

Using the PISA method, the cross-sectional area of the PDA at its aortic end during maximum flow is:

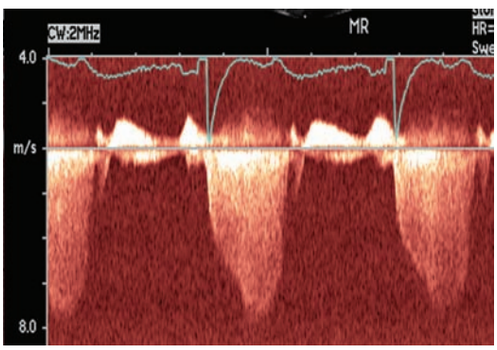
A. cm^2.
B. 0.13 cm^2.
C. 0.22 cm^2.
D. 1.3 cm^2.
E. 2.2 cm^2.

CASE 3:

A 24-year-old college athlete collapsed on the basketball court. The coach promptly used the automatic external defibrillator which delivered an apporopriate 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, and alert and oriented. Blood pressure 144/72 mm Hg; heart rate 64 beats per minute. Lungs were clear on auscultation. Cardiac exam 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 bisferient pulse.

31. 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 7-15.



Peak velocity of mitral regurgitant jet = 8 m/sec

Fig. 7-15

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.

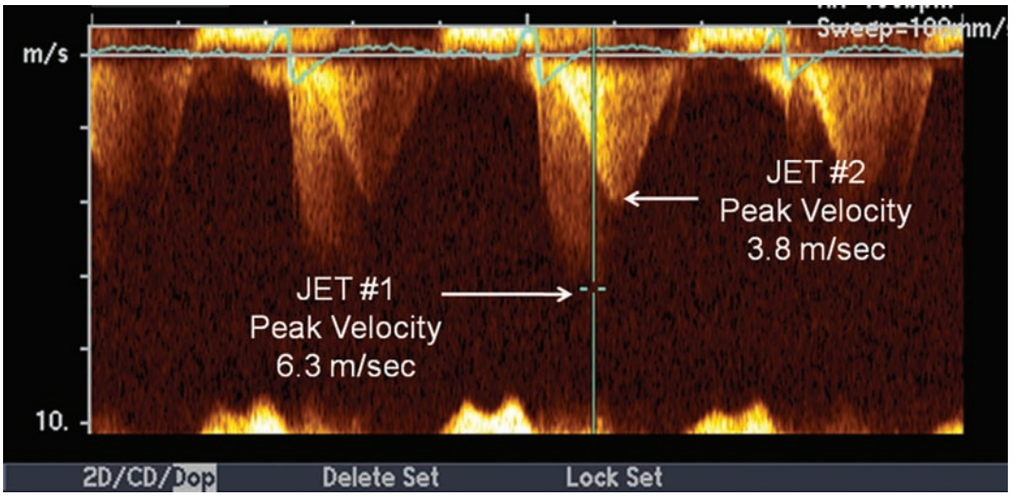


Fig. 7-16

32. The patient was started on oral disopyramide. Repeat echocardiogram was obtained and the spectral tracing in Figure 7-16 was obtained. Left atrial pressure was again estimated at 10 mm Hg. Otherwise, there were no significant changes in his echocardiogram.

The following statement is TRUE:

A. The shape of the mitral regurgitant jet is now suggestive of mitral valve prolapse with click and systolic murmur.
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.

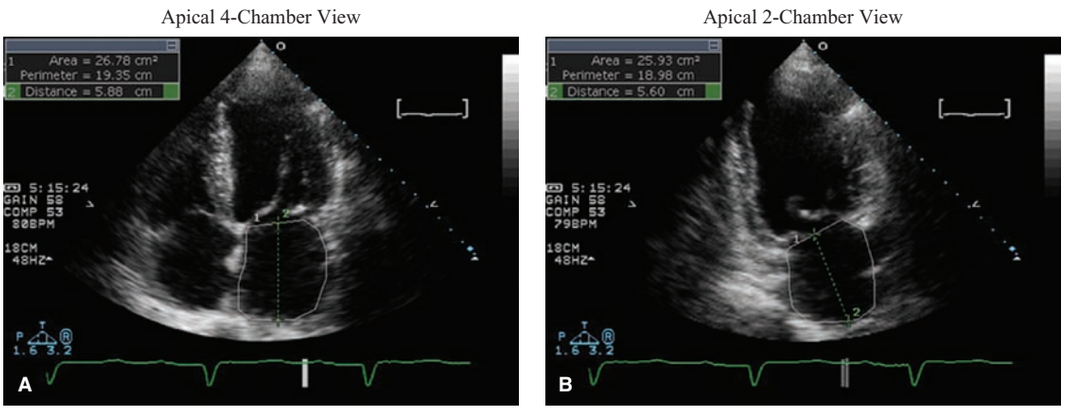
CASE 4:

A 66-year-old man with a longstanding 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 bpm.; weight 80 kg; height 175 cm; body surface area 2.0 m^2. Auscultation of the lungs reveals bibasilar rales. Cardiac exam demonstrate 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%.

33. To calculate the left atrial volume, the data in Figure 7-17 were obtained:



Area (cm^2)	27	26
Length (cm)	5.9	5.6

Fig. 7-17

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.

34. 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. 7-18).

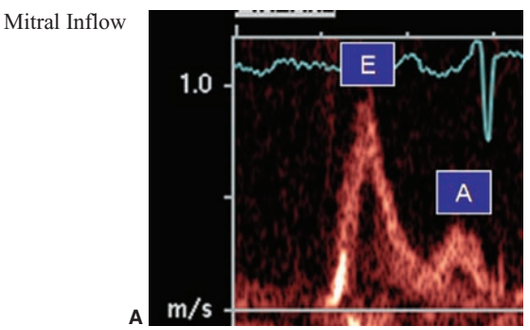
The following was the result of the appropriate medical therapy:

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-old 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.

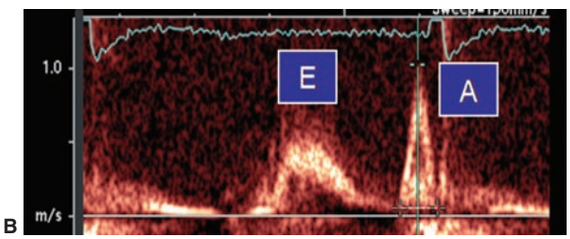
INITIAL STUDY



Mitral E wave deceleration time 140 msec

Fig. 7-18A

FOLLOW-UP STUDY



Mitral E wave deceleration time 270 msec

Fig. 7-18B

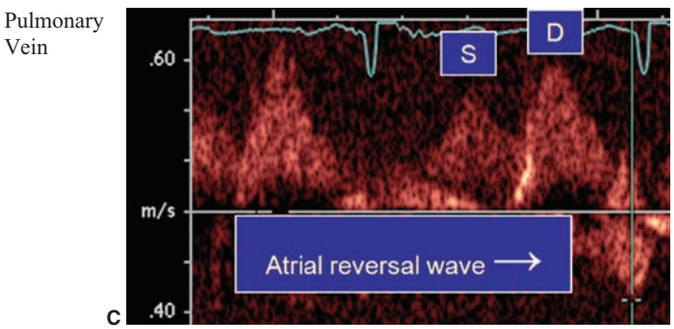


Fig. 7-18C

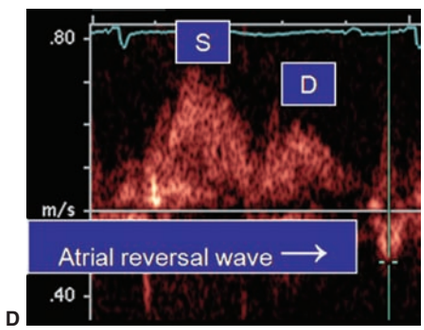


Fig. 7-18D

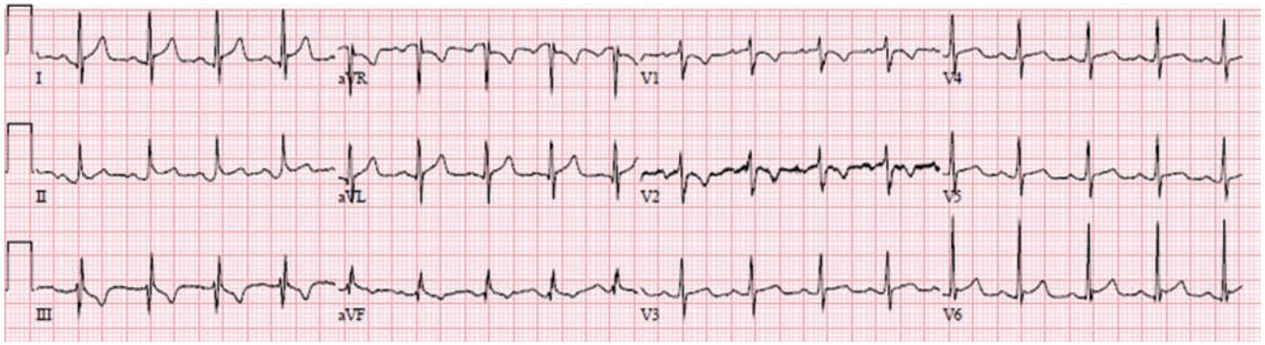


Fig. 7-19

On initial outpatient exam, he was afebrile. His lungs were clear on auscultation. There was friction rub throughout the precordium. Electrocardiogram was suggestive of pericarditis (Fig. 7-19).

He was prescribed an oral course of a nonsteroidal anti-inflammatory agent (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 echocardigraphic signs [AQ1] of tamponade. Skin test for tuberculosis (PPD) was positive. Pericardial effusion was drained percutaneously and the patient was started on appropriate antituberculosis medical therapy.

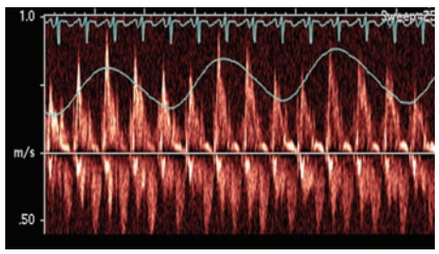
His chest pain resolved completely. However, his shortness of breath persisted and he started developing bilateral ankle edema. Transthoracic echocardiogram was ordered.

35. Figure 7-20 was also obtained on the echocardiogram.

In these recordings, the upstroke of the respirometry 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.

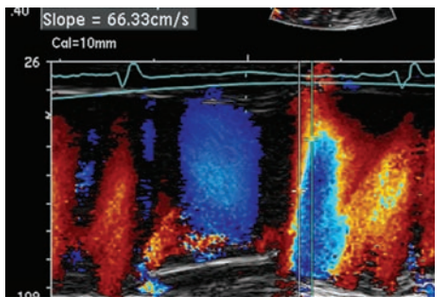
Mitral inflow spectral Doppler



A

Fig. 7-20A

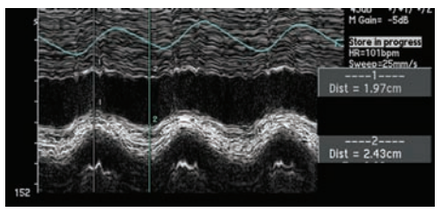
Left ventricular color M mode



B

Fig. 7-20B

Inferior vena cava



C

Fig. 7-20C

36. Video 7-2, 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: A. During expiration, the inferior vena cava (IVC) has a normal diameter of 1.7 cm or less. The measurement should be obtained 1–2 cm proximal to the IVC-right atrial junction and perpendicular to the long axis of the IVC. During spontaneous (negative pressure) inspiration, the diameter of a normal IVC decreases by more than 50%. The patient should be asked to sniff during evaluation for inspiratory diameter change; normal resting inspiration may not be sufficient to induce proper response.

The expiratory diameter of the IVC and the percent diameter decrease during inspiration are dependent on the magnitude of the right atrial pressure (RAP). This table demonstrates how RAP can be estimated from IVC diameter and percent change in diameter during the sniff maneuver.

TABLE 7-16

RAP (mm Hg)	IVC Diameter (cm)	IVC Diameter Change with Inspiration
Volume depletion	<1.2	Total
0–5	1.2–1.7	≥50%
6–10	>1.7	
11–15		≤50%
>15		None

In our patient, the expiratory diameter was 1.6 cm and there was >50% decrease in IVC diameter with inspiration:

Percent change with inspiration = (1.6 cm – 0.6 cm)/1.6 cm = 63%.

It is important to emphasize that the above methodology may not apply to athletes (who have physiologic enlargement of IVC) or intubated patient receiving positive-pressure ventilation.

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

$$\text{MVA} = \frac{220}{\text{PHT}}. \qquad \text{(Eq. 1)}$$

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

$$\text{PHT} = 0.29 \times \text{DT}. \qquad \text{(Eq. 2)}$$

Thus in our patient:

$$\text{PHT} = 0.29 \times \text{DT} = 0.29 \times 910 = 264 \text{ milliseconds},$$

$$\text{MVA} = 220/\text{PHT} = 220/264 = 0.8 \text{ cm}^2.$$

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

$$\text{MVA} = \frac{759}{\text{DT}}. \qquad \text{(Eq. 3)}$$

In our patient then:

$$\text{MVA} = 759/\text{DT} = 759/910 = 0.8 \text{ cm}^2.$$

Therefore, answer (D) is correct.

Answer (A) is incorrect because the 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:

$$\text{SV} = \text{MVA} \times \text{VTI},$$
$$\text{CO} = \text{SV} \times \text{HR},$$

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 bpm:

$$\text{SV} = 0.8 \text{ cm}^2 \times 66 \text{ cm} = 53 \text{ ml},$$
$$\text{CO} = 53 \text{ mL} \times 85 \text{ bpm} = 4.5 \text{ l/min}.$$

Answer (C) is incorrect because as shown above, PHT in this patient was 264 milliseconds and not 355 milliseconds.

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

3. ANSWER: E. Patient has patent ductus arteriosus (PDA), which 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 postneonatal period giving rise to PDA.

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

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

Note that in individuals with PDA, the flow across the RVOT represents *Q*s and the flow across the LVOT represents *Q*p. Therefore, answer (E) is correct.

This is in contrast to atrial and ventricular septal defects where LVOT flow represents *Q*s and the RVOT flow represent *Q*p. Since in most individuals with PDA, *Q*p > *Q*s, it is the left heart and not the right heart that dilates to accommodate the excess blood flow.

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

$$Q = \text{CSA} \times \text{VTI} \times \text{HR}, \qquad \text{(Eq. 1)}$$

where CSA is the cross-sectional area, VTI is the 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:

$$\text{CSA} = \left(\frac{1}{2} \times D\right)^2 \times \pi, \qquad \text{(Eq. 2)}$$

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

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

Calculations for our patient are summarized in this table:

TABLE 7-17

	LVOT	RVOT	Shunt Across PDA
Diameter (cm)	2.0	2.5	
Area (cm^2)	3.1	4.9	
VTI (cm)	31	12	
Stroke Volume (ml)	97	59	97 − 59 = 38
HR	80	80	
	***Q*p**	***Q*s**	
Flow (l/min)	7.8	4.7	
*Q*p:*Q*s	1.7	1	

Answer (A) is incorrect because the flow rate of 7.8 l/minute across the LVOT represents *Q*p and not *Q*s in patients with PDA.

Answer (B) is incorrect because *Q*p:*Q*s 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 *Q*p is much greater than *Q*s, the shunt flow 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 descending thoracic aorta past the origins of the aortic arch vessels, which supply fully oxygenated blood to the head and the arms.

4. 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 4*V*2 formula, one can calculate the pressure gradient (Δ*P*) between the PADP and the end-diastolic right ventricular pressure (RVDP).

$$\Delta P = \text{PADP} - \text{RVDP} = 4 \times V^2. \qquad \text{(Eq. 1)}$$

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

$$\Delta P = \text{PADP} - \text{RAP} = 4 \times V^2. \qquad \text{(Eq. 2)}$$

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

$$\text{PADP} = 4 \times V^2 + \text{RAP}, \qquad \text{(Eq. 3)}$$

where *V* is the end-diastolic velocity of the pulmonic regurgitant jet, and RAP is the right atrial pressure.

As explained in the answer to question 1, RAP can be estimated from the expiratory size of the 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:

PADP > 4 × (2 m/sec)^2 + 15, or greater than 31 mm Hg.

Therefore, answer (B) is correct.

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

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 typically between 5 and 16 mm Hg.

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

TABLE 7-18

	Severe MR
Regurgitant orifice (cm^2)	≥0.4
Regurgitant fraction	≥50%
Regurgitant volume (ml)	≥60
Vena contracta (cm)	≥0.7

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

$$ROA_{MR} = 2 \times \pi \times r^2 \times \frac{Valias}{Vmax}, \qquad \text{(Eq. 1)}$$

where r is the PISA radius, Valias is the aliasing velocity at which PISA radius is measured, and Vmax is the maximum velocity of the mitral regurgitant jet on spectral Doppler.

In Eq. 1, the expression $2 \times \pi \times r^2 \times V$alias represents instantaneous flow rate (IFR):

$$IFR = 2 \times \pi \times r^2 \times Valias. \qquad \text{(Eq. 2)}$$

Now Eq. 1 can be expressed as:

$$ROA_{MR} = \frac{IFR}{Vmax}. \qquad \text{(Eq. 3)}$$

In our patient, IFR is calculated as:

$$IFR = 2 \times 3.14 \times (1.0\ cm)^2 \times 45\ cm/sec = 283\ ml/sec,$$

and ROA as:

$$ROA_{MR} = 283/500\ cm/sec = 0.6\ cm^2.$$

Therefore, answer (B) is correct.

Answer (A) is incorrect because the vena contract in severe mitral regurgitation is > 0.7 cm.

Answer (C) is incorrect because the IFR of the mitral regurgitant jet in this patient is 283 ml per second as calculated above.

Answer (D) is incorrect because mitral regurgitation is severe since ROA > 0.4 cm^2 (it is 0.6 cm^2).

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

$$RegV = ROAMR \times VTIMR, \qquad \text{(Eq. 4)}$$

where VTIMR is the velocity-time integral of the mitral regurgitant jet.

In our patient, RV equals 0.6 $cm^2 \times 140$ cm, or 79 ml per beat. This is again consistent with severe mitral regurgitation (RV > 60 ml per beat).

6. 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:

$$PAWP = 4.6 + 5.27 \times \frac{E}{Vp},$$

where E is the peak blood flow velocity of the mitral inflow in cm/sec, and Vp is the flow propagation velocity of the mitral inflow (in cm/sec) obtained by color M mode. The Vp recording of this patient is demonstrated in Figure 7-21.

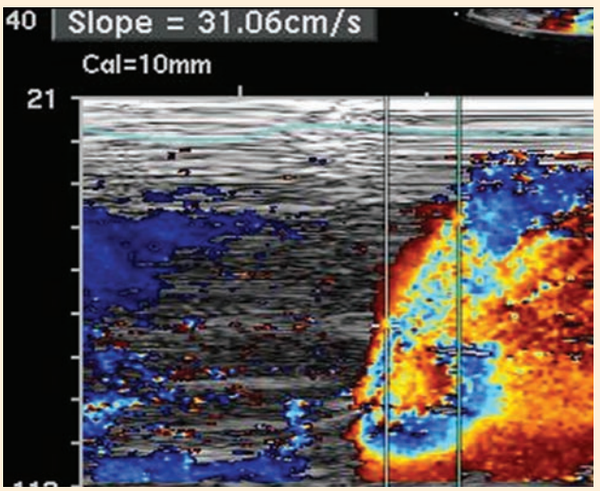


Fig. 7-21

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, Vp is an indirect measure of the rate of LV relaxation; the lower the Vp, the slower the LV relaxation and higher the left ventricular diastolic pressure (LVDP) are.

In our patient:

$$PAWP = 4.6 + 5.27 \times \frac{125}{31} = 26.$$

With the value of 26 mm Hg, PAWP is in this 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 milliseconds) or the restrictive filling pattern (E/A > 2 and E-wave deceleration time < 160 milliseconds).

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 estimated the RV-to-RA pressure gradient (ΔP) at peak systole:

$$\Delta P = 4 \times V^2 = (4 \text{ m/sec})^2 = 64 \text{ mm Hg.}$$

By adding the right atrial pressure (RAP) to ΔP, RVSP (and, by extension, PASP) can be calculated:

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

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. The E/e' ratio is further discussed in the answer to question 8.

Answer (E) is incorrect because the normal *V*p velocity >55 cm/sec in young individuals, and >45 cm/sec in middle-aged and elderly individuals.

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

TABLE 7-19

	Severe AR
Regurgitant orifice (cm^2)	≥0.3
Regurgitant fraction	≥50%
Regurgitant volume (ml)	≥60
Vena contracta (cm)	≥0.6

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 7-22.

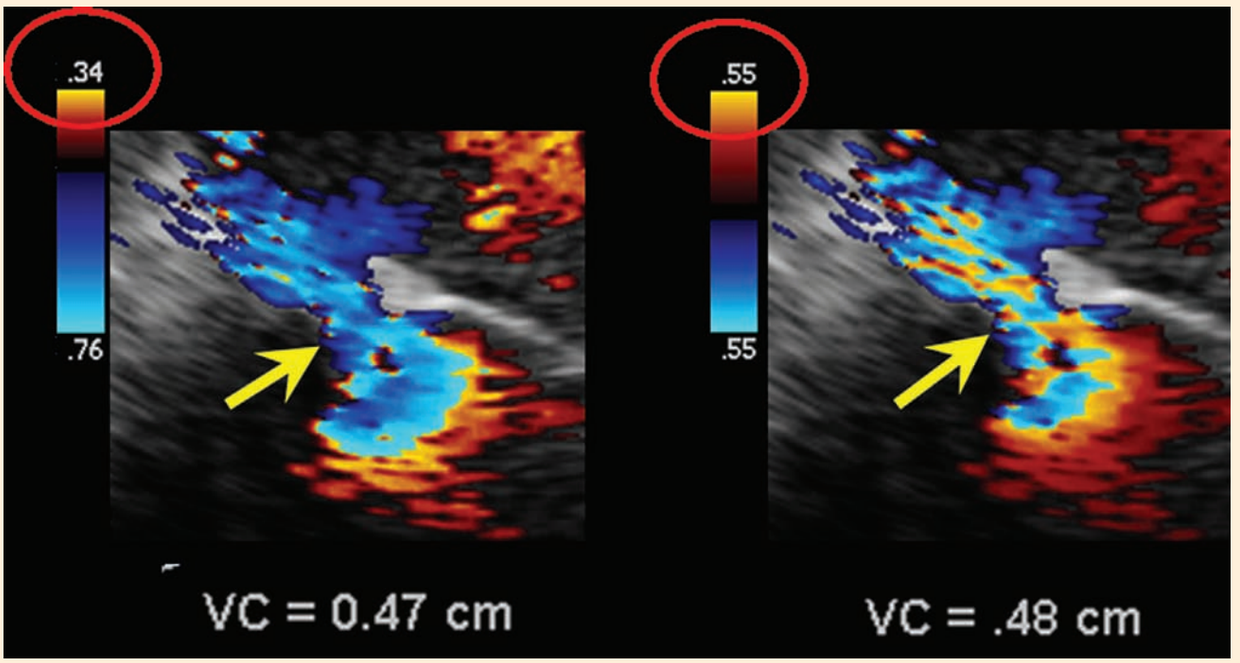


Fig. 7-22

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 2D echocardiography should not be used to calculate the regurgitant volume. Instead, the 2D diameter of vena contracta should be used for semiquantitative assessment of the degree of aortic regurgitation.

8. ANSWER: C. The E/e' ratio is directly proportional to the 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 (LVDP) is. Once LVDP rises, there is a concomitant rise in the LAP and PAWP rises 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.

The E/e' ratio can be used to estimate LAP in two ways. One approach is to use it semiquantitatively as shown in the table.

TABLE 7-20

	Left Atrial Pressure		
	Normal	**Indeterminate**	**Elevated**
E/e' using medial e'	<8	8–15	>15
E/e' using lateral e'		8–12	>12

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:

$$\text{LAP} = 1.9 + 1.24 \times \frac{E}{e'}. \qquad \text{(Eq. 1)}$$

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:

$$\text{LAP} = 4 + \frac{E}{e'}. \qquad \text{(Eq. 2)}$$

A comparison between LAP estimates using Eq. 1 and Eq. 2 is given in Figure 7-23A.

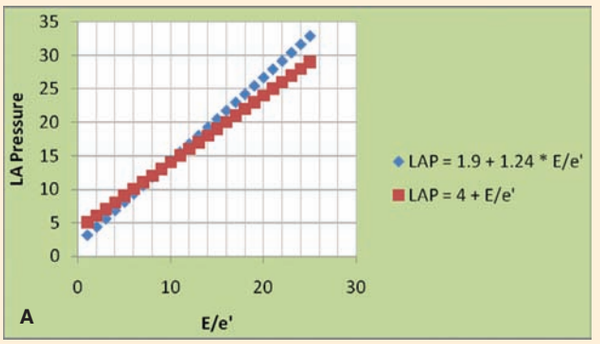


Fig. 7-23A

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:

$$\text{LVSP} = \Delta P_{MR} + \text{LAP}, \qquad \text{(Eq. 3)}$$

where ΔPMR is the peak systolic gradient of the mitral regurgitant jet, and LAP is the left atrial pressure. After expressing ΔPMR in terms of the peak velocity (V) of the mitral regurgitant jet, Eq. 1 becomes:

$$\text{LVSP} = 4 \times V^2 + \text{LAP}. \qquad \text{(Eq. 4)}$$

In our patient:

$$\text{LVSP} = 4 \times (6.0 \text{ m/sec})^2 + 20$$
$$= 164 \text{ mm Hg}.$$

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

$$\text{P2P} = \text{LVSP} - \text{SBP}, \qquad \text{(Eq. 5)}$$

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 7-23B. P2P is lower than the peak instantaneous gradient (PIP) obtained by continuous- wave Doppler across the aortic valve.

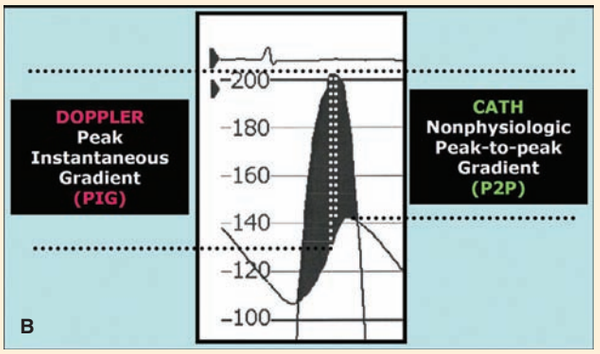


Fig. 7-23B

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/sec. Is it ±1661 mm Hg/sec.

Answer (D) is incorrect because in severe mitral regurgitation vena contracta > 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).

9. ANSWER: D. Figure 7-24 shows the continuous-wave spectral Doppler tracings of our patient.

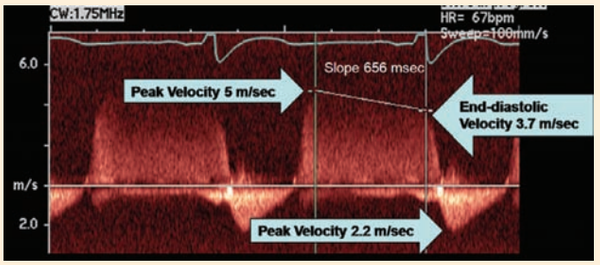


Fig. 7-24

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

$$\Delta P = DBP - LVEDP = 4 \times V^2. \qquad \text{(Eq. 1)}$$

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

$$LVEDP = DBP - 4 \times V^2. \qquad \text{(Eq. 2)}$$

In our patient:

$$LVEDP = 65 \text{ mm Hg} - 4 \times (3.7 \text{ m/sec})^2 = 10 \text{ mm Hg}.$$

Therefore, answer (D) is correct.

Answer (A) is incorrect because in severe aortic regurgitation, pressure half-time is <300 milliseconds.

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

Answer (C) is incorrect because the peak LVSP is always higher than the systolic blood pressure 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 the answer to question 8.

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

$$\text{LVOT Stroke Volume} = \text{AV Stroke Volume} \qquad \text{(Eq. 3)}$$

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

$$CSALVOT \times VTILVOT = CSAAV \times VTIAV \qquad \text{(Eq. 4)}$$

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 left ventricular outflow tract and the aortic valve in systole. In Eq. 6, this will be reflected in a proportional increase in VTILVOT and VTIAV.

By continuity equation, the AV area (CSAAV) can be calculated as follows:

$$CSA_{AV} = CSA_{LVOT} \times \frac{VTI_{LVOT}}{VTI_{AV}}. \qquad \text{(Eq. 5)}$$

In aortic regurgitation, there is augmentation of VTILVOT and VTIAV. However, the ratio of the two VTIs remains the same, and therefore the calculated value of CSAAV is not affected by the presence of aortic regurgitation.

10. ANSWER: C. The patient has an atrial septal defect (ASD) with a left-to-right shunt. An 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 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 7-21

	RVOT	LVOT	Comment
Diameter (cm)	2.6	2.0	
Area (cm^2)	5.3	3.1	Calculated using formula Area = $(0.5 \times \text{Diameter})^2 \times \pi$
VTI (cm)	30	20	
Stroke volume (ml)	159	63	Calculated using formula Stroke volume = Area × VTI
Heart rate (beats per minute)	75	75	Calculated using formula Flow = Stroke volume × Heart rate
Flow (l/min)	11.9	4.7	Shunt flow is the difference between *Q*p and *Q*s, or 7.2 l/min.
	Pulmonic flow (*Q*p)	Systemic flow (*Q*s)	
*Q*p:*Q*s	2.5:1		

Because *Q*p:*Q*s = 2.5:1, the 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 below.

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

$$\text{PVR} = \frac{\Delta P}{Q\text{p}}. \qquad \text{(Eq. 1)}$$

where *Q*p is the pulmonary blood flow (in l/minute), and ΔP is the pressure gradient across the pulmonary circulation. ΔP is the difference between the mean pulmonary artery pressure (MPP) and the mean LAP. Eq. 1 then becomes:

$$\text{PVR} = \frac{\text{MPP} - \text{LAP}}{Q\text{p}}. \qquad \text{(Eq. 2)}$$

MPP can be calculated from PASP and the PADP using the following equation:

$$\text{MPP} = \text{PADP} + \frac{1}{2} \times (\text{PASP} - \text{PADP}). \qquad \text{(Eq. 3)}$$

In this patient:

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

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

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

Normal PVR is 1–2 Wood units (80–160 dyne × sec × 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 900–1300 dyne × sec × 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:

$$\text{SF} = Q\text{p} - Q\text{s} = 11.9 - 4.7 = 7.2 \text{ l/min}.$$

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.

11. ANSWER: B. The presence of ventricular septal defect (VSD) allows for calculation of the RVSP and, by extension, the PASP if the SBP is known.

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

RVSP = SBP – Peak systolic VSD gradient. (Eq. 1)

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

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

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

$$\text{RVSP} = \text{SBP} - 4 \times V^2. \quad \text{(Eq. 3)}$$

Thus, in this patient, RVSP = $120 - 4 \times (3.0 \text{ m/sec})^2$ = 84 mm Hg.

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

$$\text{PASP} = \text{RVSP} - \text{Peak PS Gradient}. \quad \text{(Eq. 4)}$$

In our patient, PASP = 84 − 55 = 29 mm Hg. Therefore, the answer (B) is correct.

Answer (A) is incorrect because RVSP in this patient is 84 mm Hg as calculated above.

Answer (C) is incorrect because the right atrial pressure 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 pulmonic stenosis. 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 the LVEDP is known, the RVEDP can be calculated as:

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

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

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

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

$$\text{RVEDP} = \text{LVEDP} - 4 \times V^2, \quad \text{(Eq. 7)}$$

where V is the end-diastolic velocity across the VSD. In our patient:

$$\text{RVEDP} = 12 - 4 \times (1 \text{ m/sec})^2 = 12 - 4 = 8 \text{ mm Hg}.$$

12. ANSWER: B. The pulmonic flow (Qp) in patients with an atrial septal defect (ASD) is the sum of the shunt flow (SF) across the ASD and the systemic flow (Qs). SF can be calculated either directly or as the difference between Qp and Qs.

One method for direct calculation of SF is the standard echocardiographic formula for determining flow 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 atrial septal defect whose diameter is 1.2 cm. Since the ASD is circular in shape, hence the ASD area can be calculated as:

$$\text{CSAASD} = (\tfrac{1}{2} \times \text{ASD diameter}) \times 2 \times \pi.$$

In our patient:

$$\text{CSAASD} = (\tfrac{1}{2} \times 1.2 \text{ cm}) \times 2 \times 3.14 = 0.36 \times 3.14 = 1.13 \text{ cm}^2.$$

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

$$\text{ASD shunt stroke volume} = \text{CSAASD} \times \text{VTIASD}$$

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 calculated the shunt flow across the ASD. In our patient:

$$\text{ASD shunt flow} = 90 \text{ ml} \times 100 \text{ bpm} = 9.0 \text{ l/min}.$$

Therefore, the answer (B) is correct.

Answer (A) is incorrect because the Qp:Qs in this patient is 2.5:1. In this patient, Qs is calculated at the level of the LVOT using the formula:

$$Q\text{s} = \text{CSALVOT} \times \text{VTILVOT} \times \text{HR}$$

where CSALVOT is the cross-sectional area of LVOT, VTILVOT is the velocity-time integral at the LVOT level, and HR is the heart rate. In our patient:

$$Q\text{s} = (\tfrac{1}{2} \times 2.0 \text{ cm})^2 \times \pi \times 19 \text{ cm} \times 100 \text{ bpm} = 60 \text{ ml} \times 100 \text{ bpm} = 6.0 \text{ l/minute}.$$

In the next step, we can calculate Qp as:

$$Q\text{p} = Q\text{s} + \text{ASD shunt flow}.$$

In our patient:

$$Q\text{p} = 6.0 \text{ l/minute} + 9.0 \text{ l/minute} = 15.0 \text{ l/minute}.$$

Once *Q*p and *Q*s are known, we can calculate the *Q*p:*Q*s ratio:

$$Qp{:}Qs = 15.0 \text{ l/minute} : 6.0 \text{ l/minute} = 2.5{:}1$$

Answer (C) is incorrect because the difference between systemic and stroke volumes in this patient is 90 ml/beat. This value represents the ASD shunt stroke volume calculated above.

Answer (D) is incorrect because the systemic stroke volume in this patient is 60 ml per beat as calculated above.

Answer (E) is incorrect because the *Q*p is this patient is 15.0 l/minute as calculated above. Calculations related to this question are summarized in this table.

TABLE 7-22

	LVOT	ASD	RVOT	Comments
Diameter (cm)	2.0	1.2		
Area (cm^2)	3.10	1.13		
VTI (cm)	19	80		
Stroke volume (ml)	60	90	150	RVOT stroke volume is the sum of LVOT and ASD stroke volumes.
Heart rate (beats per minute)	100	100		
Flow (l/min)	6.0	9.0	15.0	*Q*p is the sum of *Q*p and ASD shunt flow.
	Systemic flow (*Q*s)	Shunt flow	Pulmonic flow (*Q*p)	*Q*p:*Q*s = 2.5

13. 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 time (*dt*), a measure of left ventricular systolic function, using the following formula:

$$dP/dt = \frac{\Delta P}{\text{RTI}}, \qquad \text{(Eq. 1)}$$

where RTI is the relative time interval, measured in seconds, between mitral regurgitant jet velocities of 1 m/sec (*V*1) and 3 m/sec (*V*2). ΔP represents the pressure difference between the left ventricular to left atrial pressure gradients at *V*2 and *V*1 (Figure 7-25A).

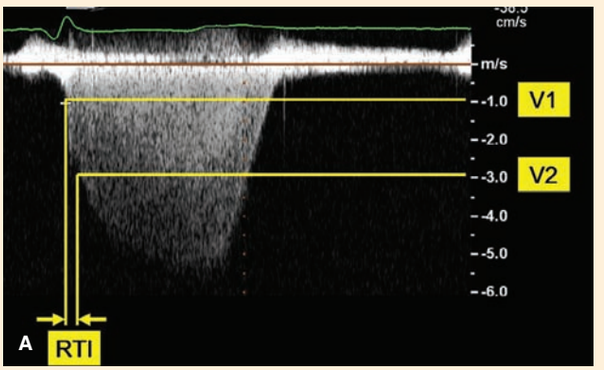


Fig. 7-25A

This pressure difference can be calculated as:

$$\Delta P = (4V22 - 4V12),$$
$$\Delta P = 4 \times (3 \text{ m/sec}) \times 2 - 4 \times (1 \text{ m/sec})^2$$
$$= 4 \times 9 - 4 \times 1 = 36 - 4,$$
$$\Delta P = 32 \text{ mm Hg}.$$

Now, Eq. 1 can be expressed as:

$$dp/dt = \frac{32}{\text{RTI}}. \qquad \text{(Eq. 2)}$$

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

$$\text{RTI} = \text{Time at } V2 - \text{Time at } V1 = 25 \text{ milliseconds} - 5 \text{ milliseconds} = 20 \text{ milliseconds}.$$

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

$$\text{RTI} = 20 \text{ milliseconds} = 0.02 \text{ seconds}.$$

Once RTI is known, we can calculate *dP/dt* in our patient:

$$dP/dt = \text{to } 32/0.02 = 1{,}600 \text{ mm Hg/sec}.$$

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 and heart rate, peak velocity is then a direct function (*f*) of stroke volume:

$$\text{E-wave velocity} = f(\text{SV}). \qquad \text{(Eq. 3)}$$

In mitral regurgitation, SV that crosses the mitral valve in diastole is the sum of the systemic stroke volume (SVLVOT) and the RegV. Thus, Eq. 5 can be expressed as:

$$\text{E-wave velocity} = f(\text{SVLVOT} + \text{RegV}). \qquad \text{(Eq. 4)}$$

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 $>$ 0.7 cm), peak E velocity is expected to be $>$1.5 m/sec. In severe prosthetic mitral regurgitation, the peak E velocity is usually $>$2.0 m/sec.

Answer (B) is incorrect because LAP in this patient is elevated. The patient presents with severe mitral regurgitation (vena contracta $>$ 0.7 cm) and pulmonary edema due to elevated left atrial pressure (LAP).

Using the peak velocity (*V*max) of the mitral regurgitant jet, one can calculate the pressure gradient (ΔP) between the peak left ventricular systolic pressure (LVSP) and the LAP:

$$\Delta P = 4 \times V\,\text{max}^2. \qquad \text{(Eq. 5)}$$

In our patient:

$$\Delta\text{P} = 4 \times (4.0\ \text{m/sec})^2 = 4 \times 16 = 64\ \text{mm Hg}.$$

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

$$\text{LVSP} = \Delta P + \text{LAP}. \qquad \text{(Eq. 6)}$$

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

$$\text{LAP} = \text{LVSP} - \Delta P. \qquad \text{(Eq. 7)}$$

The LAP calculated by this method represents a value on the CV wave portion of the left atrial pressure 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 systolic blood pressure (SBP). Thus, we can express Eq. 7 as:

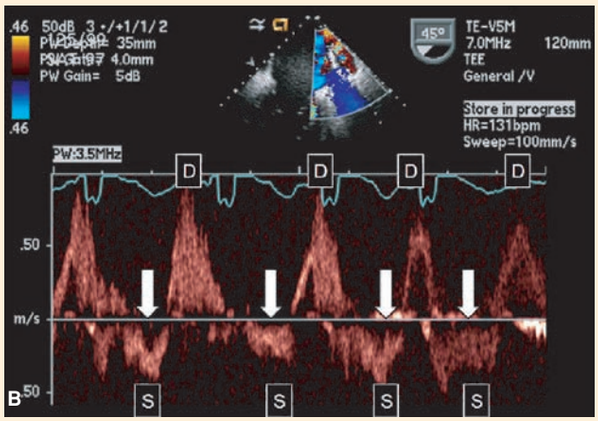
$$\text{LAP} = \text{SBP} - \Delta P. \qquad \text{(Eq. 8)}$$

In our patient, whose SBP was 95 mm Hg and whose ΔP was calculated above at 64 mm Hg, LAP is then calculated as:

$$\text{LAP} = 95\ \text{mm Hg} - 64\ \text{mm Hg} = 31\ \text{mm Hg}.$$

This LAP of 31 mm Hg is highly elevated (normal LAP is $<$12 mm Hg).

Answer (C) is incorrect because in severe mitral regurgitation there may be a 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 7-25B.



Systolic wave reversal (*arrows*) in the left upper pulmonary vein due to severe mitral regurgitation is seen on spectral Doppler recordings on a transesophageal echocardiography. S, systolic wave; D, diastolic wave.

Fig. 7-25B

Answer (E) is incorrect because *dP/dt* in this patient is estimated at 1,600 mm Hg/sec, which is normal. (Normal *dP/dt* = 1661 +/− 323 mm Hg/sec). The value of 800 mm Hg/sec would indicate a markedly diminished LV systolic function as seen in cardiogenic shock, for example.

14. 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 mm Hg is consistent with severe mitral stenosis as shown in this table.

TABLE 7-23

	Mild MS	Moderate MS	Severe MS
Mitral valve area (cm^2)	$<$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:

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

In our patient:

$$\text{LAP is } 21\ \text{mm Hg} + 7\ \text{mm Hg} = 28\ \text{mm Hg}.$$

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 left atrial pressure 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 CSA of the orifice:

$$V \approx \frac{1}{\text{CSA}}. \qquad \text{(Eq. 1)}$$

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

$$V \approx \frac{1}{\text{MVA}}. \qquad \text{(Eq. 2)}$$

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 pressure-half time method may be unreliable immediately after but not before the mitral valvuloplasty. Pressure-half time 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 MLA 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 pressure half-time shortens above and beyond what would be expected by an increase in the mitral valve area alone after valvuloplasty. Therefore, the pressure-half time method may lead to calculation of an erroneously large mitral valve area.

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

$$\text{MVA} = \frac{220}{\text{PHT}} = \frac{220}{270} = 0.8.$$

15. ANSWER: C. When velocity-time integrals are not available, aortic valve area (AVA) can be calculated using the following modified continuity equation:

$$\text{AVA} = \text{CSA}_{\text{LVOT}} \times \frac{V_{\text{LVOT}}}{V_{\text{AV}}}, \qquad \text{(Eq. 1)}$$

where CSALVOT is the CSA of the LVOT, VLVOT is the peak systolic LVOT velocity, and VAV is the peak systolic AV velocity.

The VLVOT/VAV ratio of the two velocities is referred to as the dimensionless index (DI). Thus, Eq. 1 can be expressed as:

$$\text{AVA} = \text{CSA}_{\text{LVOT}} \times \text{DI}. \qquad \text{(Eq. 2)}$$

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

$$\text{AVA} = \left(\frac{1}{2} \times D\right)^2 \times \text{DI}. \qquad \text{(Eq. 3)}$$

In our patient:

$$\text{AVA} = (½ \times [1.9\ \text{cm}]^2) \times (1\ \text{m/sec}/5\ \text{m/sec}),$$
$$\text{AVA} = 2.84\ \text{cm}^2 \times 0.2,$$
$$\text{AVA} = 0.6\ \text{cm}^2.$$

Therefore, answer (C) is correct.

As a rule, when the dimensionless index (DI) is < 0.25, the AVA is $< 1.0\ \text{cm}^2$ across the range of LVOT diameters commonly encountered in adults as demonstrated in this table:

TABLE 7-24

LVOT Diameter (cm)	LVOT Area (cm^2)	AVA (cm^2) if DI = 0.25
1.8	2.54	0.64
1.9	2.84	0.71
2.0	3.14	0.79
2.1	3.46	0.87
2.2	3.80	0.95

Answer (A) is incorrect because the modified continuity equation using the dimensionless index, as explained above, can be used to calculate the aortic valve area when velocity-time integrals are unavailable.

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

Answer (D) is incorrect because the left ventricular stroke volume in this patient is 57 ml per beat. Left ventricular SV can be calculated as follows:

$$\text{SV} = ½ \times (\text{LVOT diameter})^2 \times \text{VTILVOT},$$
$$\text{SV} = ½ \times (1.9\ \text{cm})^2 \times 20\ \text{cm},$$
$$\text{SV} = 57\ \text{ml per beat}.$$

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.

16. ANSWER: C. Peak RVSP in a patient with or without pulmonic stenosis can be calculated as:

$$\text{RVSP} = \text{Peak RV-to-RA systolic gradient} + \text{RAP}, \quad \text{(Eq. 1)}$$

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:

$$\text{RVSP} = 4 \times V2 + \text{RAP}. \quad \text{(Eq. 2)}$$

In the absence of pulmonic stenosis, RVSP is equal to PASP. In pulmonic stenosis, 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:

$$\text{PASP} = \text{RVSP} - \text{PS Gradient}. \quad \text{(Eq. 3)}$$

In our patient:

$$\text{RVSP} = 4 \times (4.0 \text{ m/sec})^2 + 10 = 74 \text{ mm Hg},$$
$$\text{PASP} = 74 - 24 = 50 \text{ mm Hg}.$$

Therefore, answer (C) is correct.

All calculations are graphically summarized in Figure 7-26; RVP, right ventricular pressure; RAP, right atrial pressure; PAP, pulmonary artery pressure.

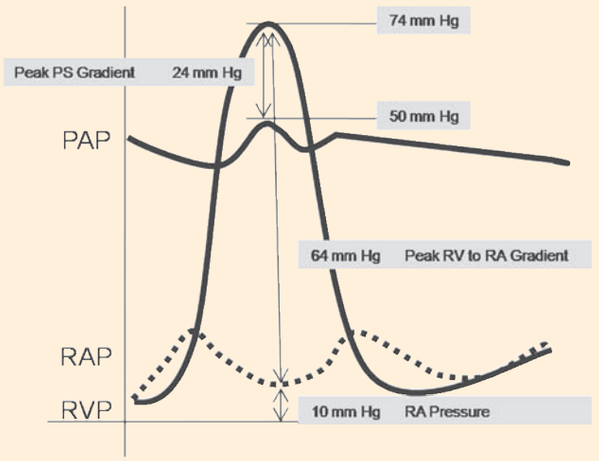


Fig. 7-26

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

Answer (B) is incorrect because RVSP exceed PASP by 24 mm Hg, the value of the peak systolic gradient 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 above.

17. ANSWER: C. Peak gradient (ΔPmax) 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:

$$\Delta P\text{max} = 4 \times \text{V2}. \quad \text{(Eq. 1)}$$

The mean aortic valve gradient (ΔPmean) is approximately 60% of the peak gradient (ΔPmax):

$$\Delta P\text{mean} = 0.6 \times \Delta P\text{max}. \quad \text{(Eq. 2)}$$

In our patient:

$$\Delta P\text{max} = 4 \times (5.0 \text{ m/sec})^2 = 100 \text{ mm Hg},$$
$$\Delta P\text{mean} = 0.6 \times 100 \text{ mm Hg} = 60 \text{ mm Hg}.$$

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/sec) and the peak systolic aortic velocity (5.0 m/sec) 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 to 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 aortic valve area in this patient is less than 1.0 cm^2. AVA in this patient can be estimated using the modified continuity equation:

$$\text{AVA} = \text{CSA}_{\text{LVOT}} \times \frac{V_{\text{LVOT}}}{V_{\text{AV}}}. \quad \text{(Eq. 3)}$$

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

$$\text{AVA} = \left(\frac{1}{2} \times D\right)^2 \times \frac{V_{\text{LVOT}}}{V_{\text{AV}}}, \quad \text{(Eq. 3)}$$

where CSALVOT is the cross-sectional area of the LVOT, VLVOT is the LVOT peak systolic velocity, and VAV 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/sec is normal.

18. 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 7-27.

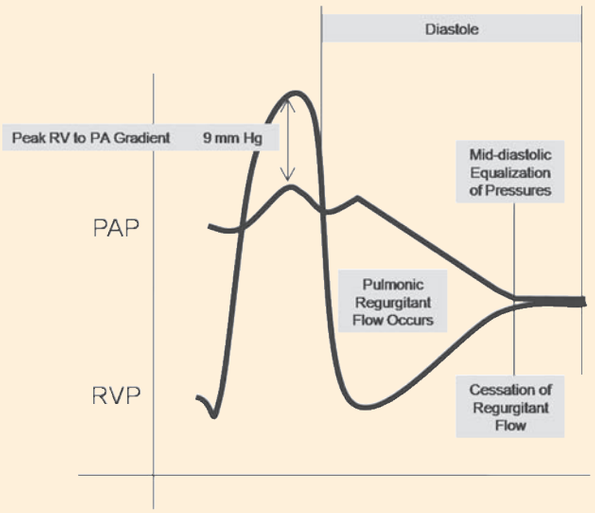


Fig. 7-27

This rapid deceleration and premature cessation of the pulmonic regurgitant jet is 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.

Answer (B) is incorrect because the peak antegrade velocity across the pulmonic valve in systole is only elevated to about 1.5 m/sec (peak systolic gradient = 4 × 1.52 = 9 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:

$$V = \frac{Q}{\text{PVA}} = \frac{\text{SV} \times \text{HR}}{\text{PVA}}$$

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 PVA remains constant, any increase in stroke volume leads to elevation in the transvalvular velocity.

Answer (C) is incorrect as it is the right ventricular systolic pressure that exceeds pulmonary artery by only 9 mm Hg.

Answer (E) is incorrect because in uncomplicated PDA, antegrade flow occurs during both systole and diastole. In the patient's tracing, there is antegrade flow in systole and retrograde flow in diastole.

19. ANSWER: C. The tracings were obtained from an elderly woman presenting with acutely decompensated heart failure.

Mean left atrial pressure can be estimated semi-quantitatively from the ratio of peak flow velocity of mitral E wave and the peak velocity of mitral annular tissue Doppler e' wave according to this chart:

TABLE 7-25

	Left Atrial Pressure		
	Normal	**Indeterminate**	**Elevated**
E/e' using medial e'	< 8	8–15	>15
E/e' using lateral e'		8–12	>12

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 milliseconds) indicates a restrictive filling pattern. The features of different filling patterns in individuals older than 60 years are summarized in this table.

TABLE 7-26

Filling Pattern	Diastolic Dysfunction	Mitral Inflow E/A	E-wave Deceleration Time (milliseconds)	Pulmonary Vein S/D	Mitral Annular e' (cm/sec)
Normal	None	0.6–1.3	≤258	>1	>8
Abnormal relaxation	Mild	<0.8	>258		<8
Pseudonormal	Moderate	0.8–2	160–258	<1	
Restrictive filling	Severe	>2	<160		

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 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 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.

When the left ventricular diastolic pressure 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/sec 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 milliseconds more than the duration of the mitral inflow A wave. In our patient, the peak velocity of AR was 50 cm/sec, and AR outlasted mitral A wave by 40 milliseconds (210–170 milliseconds); both are indicative of an elevated LV diastolic pressure. 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 left ventricular diastolic pressure.

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

Answer (E) is incorrect because when left atrial pressure is elevated in older patients, the peak velocity of the systolic wave (S wave) in the pulmonary vein tracings is generally lower than the peak velocity of the diastolic wave (D wave). The higher the left atrial pressure, the lower the S/D ratio is.

21. ANSWER: B. In constrictive pericarditis, ventricular filling is constrained by an inelastic pericardial sac which 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) differential impact of negative intrathoracic pressure that develops during inspiration on the 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 the 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 impedes RV filling; the rise in the 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 are 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 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 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.

22. ANSWER: B. In a normal descending aorta, antergrade flow occurs only in systole and there is a a small flow reversal in early diastole as depicted in the pulsed wave Doppler tracing in Figure 7-28A:

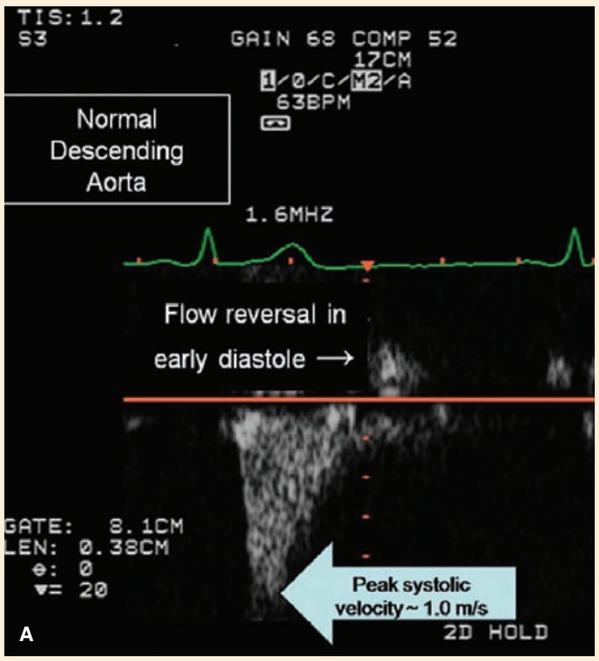


Fig. 7-28A

The pulsed-wave Doppler tracing in Figure 7-7 is abnormal as it demonstrates antegrade flow is through 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 the diastole (holodiastolic flow reversal) as demonstrated in Figures 7-28B and C. [Au2]

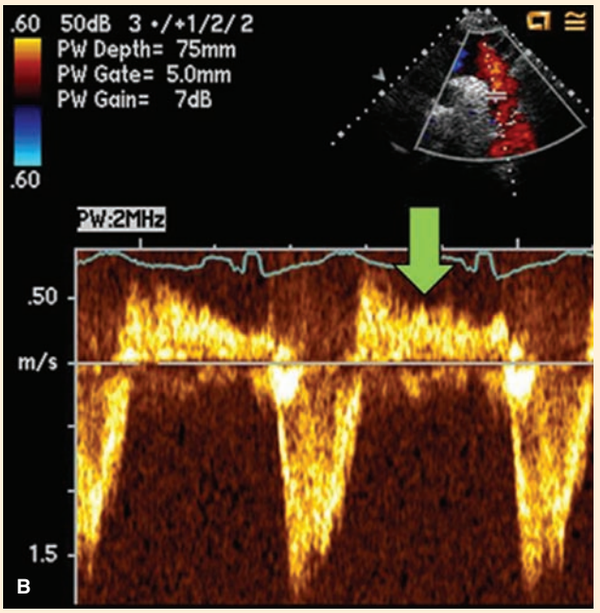


Fig. 7-28B

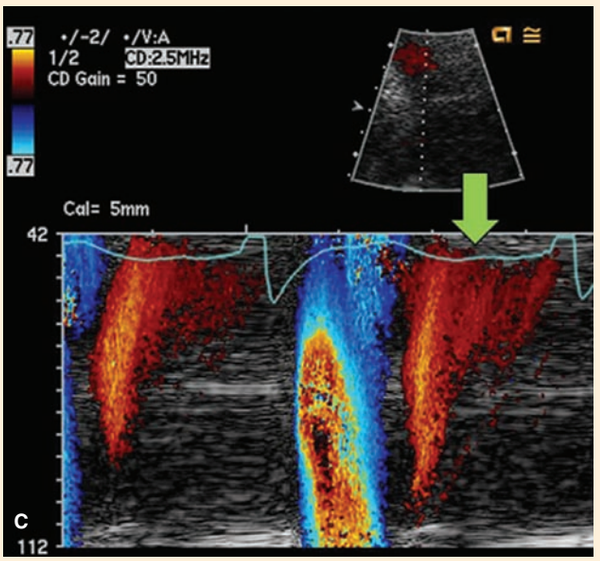


Fig. 7-28C

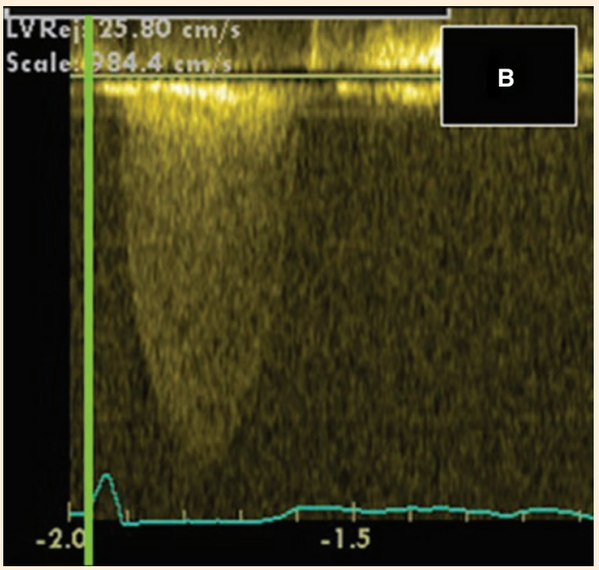
Answer (C) is incorrect because it is the bicuspid and not quadricuspid aortic valve that is typically associated with aortic coarctation. It is estimated that between 25% and 46% of all individuals with coarctation have bicuspid aortic valve.

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 in the legs.

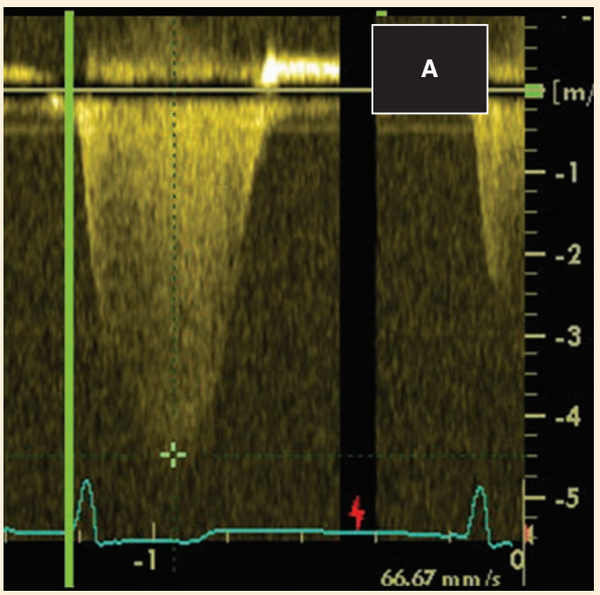
23. ANSWER: C. Normal systole consist of isovolumic contraction time and ejection period. Flow across the aortic valve, whether the valve is normal or stenotic, occurs only during the ejection period of systole. In contrast, tricuspid regurgitant jet extends throughout the systole. Thus, on a spectral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

Answer (A) is incorrect because Figure 7-8B 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 Figure 7-8B. In contrast, the onset of tricuspid regurgitant jet on Figure 7-8A coincides with the QRS on EKG.



Au: Callout missing for this figure.

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



Au: Callout missing for this figure.

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

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/sec 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.

24. ANSWER: B. In both tamponade and constrictive pericarditis, there is impairment in ventricular filling during the 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 tracings 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 but 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 milliseconds).

Both in tamponade and constrictive pericarditis, there is ventricular interdependence, which was discussed in the answer to question 21. Because of ventricular interdependence, there is 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 (ΔE):

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

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 are 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 milliseconds). 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 or increased in constrictive pericarditis and diminished in restrictive cardiomyopathy.

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

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.

25. 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 right atrial pressures (RAP) which results in a very rapid pressure equilibration between right ventricular pressure (RVP) and the RAP as demonstrated in the pressure tracings in Figure 7-29A.

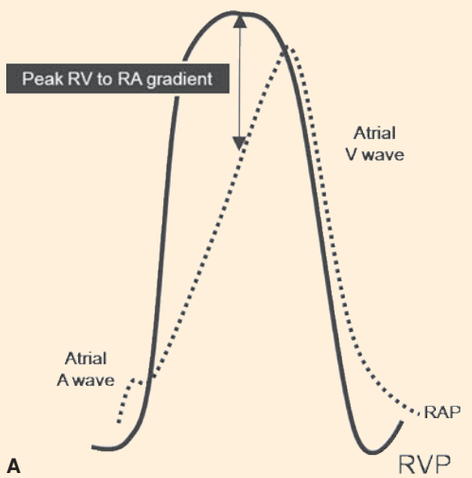
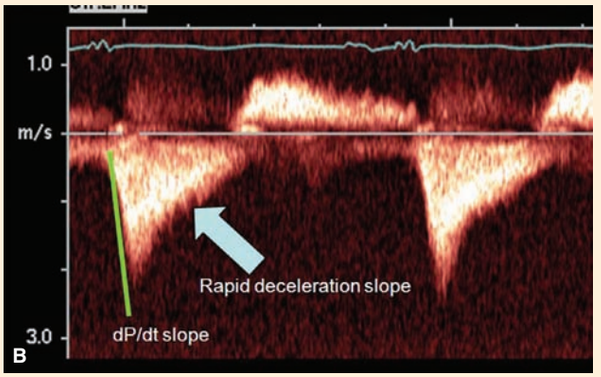


Fig. 7-29A

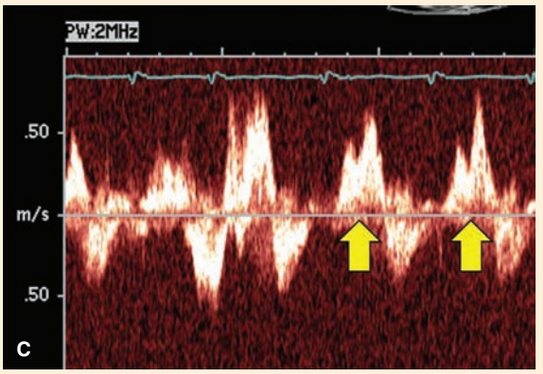
The rapid rise in the right atrial pressure results in rapid deceleration slope of the tricuspid regurgitant jet (arrow in the continuous Doppler tracing of the tricuspid regurgitant jet, Figure 7-29B). Therefore, answer (A) is correct.



Continuous spectral Doppler of tricuspid regurgitant jet

Fig. 7-29B

Clinically, a patient with this type of tricuspid regurgitation typically has a pulsatile liver. An echocardiographic correlate of pulsatile liver is the systolic wave reversal in these hepatic vein spectral Doppler tracings (Fig. 7-29C).



Systolic wave reversal in hepatic vein spectral Doppler tracing indicative of severe tricuspid regurgitation.

Fig. 7-29C

Answer (B) is incorrect because the acceleration rate in tricuspid regurgitant jet velocities from baseline to the peak velocity is fast indicative of a normal *dP/dt* and a normal RV systolic function.

Answer (C) is incorrect because of the rapid pressure equilibration between the RV and RA due to rapid rise in the RA pressure; the peak velocity of the tricuspid regurgitant jet is often low even in the presence of significant pulmonary hypertension.

Answer (D) is incorrect because the flow velocity profile of this patient's tricuspid regurgitant jet is typical of severe tricuspid regurgitation (low peak velocity; rapid deceleration slope due to rapid pressure equilibration between RV and RA).

Answer (E) is incorrect because the spectral Doppler tracing of a midcavitary right ventricular gradient has its peak in late systole. In this patient, the jet peaks in early systole.

26. 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 26 were as follows:

TABLE 7-27

A	Abnormal relaxation pattern (Grade I diastolic dysfunction)
B	Pseudonormal pattern (Grade II diastolic dysfunction)
C	Restrictive filling pattern (Grade III diastolic dysfunction)
D	Mitral inflow in a patient with mechanical mitral valve (note the vertical line artifact due to opening and closing of the prosthetic leaflets).
E	Mitral inflow in a patient with atrial fibrillation.

Since the patient has 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 pulmonary artery wedge pressure (PAWP) for the remaining three patterns:

TABLE 7-28

Pattern	Peak E Velocity (cm/sec)	Peak e' Velocity (cm/sec)	PAWP = 1.9 + 1.24 × (E/e') (mm Hg)	PAWP = 4 + E/e' (mm Hg)
A	45	5	13	13
B	60	5	17	16
C	150	5	39	34

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.

KEY POINTS:

- In normal sinus rhythm, the mitral inflow pattern is characterized by two antegrade waves: E wave in early diastole, and A wave in late diastole following atrial contraction. In atrial fibrillation, the A wave is abolished.
- In elderly patients, the ratio of peak E and A velocities is usually <1 and the deceleration time of the mitral E wave is prolonged.
- Elevation in left atrial pressure in elderly individuals leads to a progressive increase in the E/A ratio and a progressive shortening of the mitral E-wave deceleration time. With progressive increase in left atrial pressure, the abnormal relaxation pattern gradually becomes pseudonormal. With further increase in the left atrial pressures, mitral filling pattern becomes restrictive.

27. ANSWER: E. The severity of 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*alias was 69 cm/sec, and *V*max was 420 cm/sec:

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

This EROA is very large (see this table) and indicative of severe mitral regurgitation.

Therefore, answer (E) is correct.

KEY POINTS:

- Proximal isovelocity surface area (PISA) method can be used to calculate the effective regurgitant orifice (ROA) of mitral regurgitation.
- To calculate ROA, the following three parameters are required: the PISA radius, the aliasing velocity at which PISA radius is measured, and the peak velocity of the mitral regurgitant flow.
- In severe mitral regurgitation, ROA is usually >0.4 cm^2 (>40 mm^2).

28. ANSWER: A. The 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.

The additional TEE images in Figure 7-30 further illustrate the case.

TABLE 7-29

	Mild (1+)	Moderate (2+)	Moderate-Severe (3+)	Severe (4+)
EROA (cm^2)	<0.2	0.20–0.29	0.30–0.39	≥0.4
Regurgitant fraction	<30%	30–39%	40–49%	≥50%
Regurgitant volume (mL)	30	30–44	45–59	≥60
Vena contracta (cm)	<0.3	0.3–0.7	>0.7	

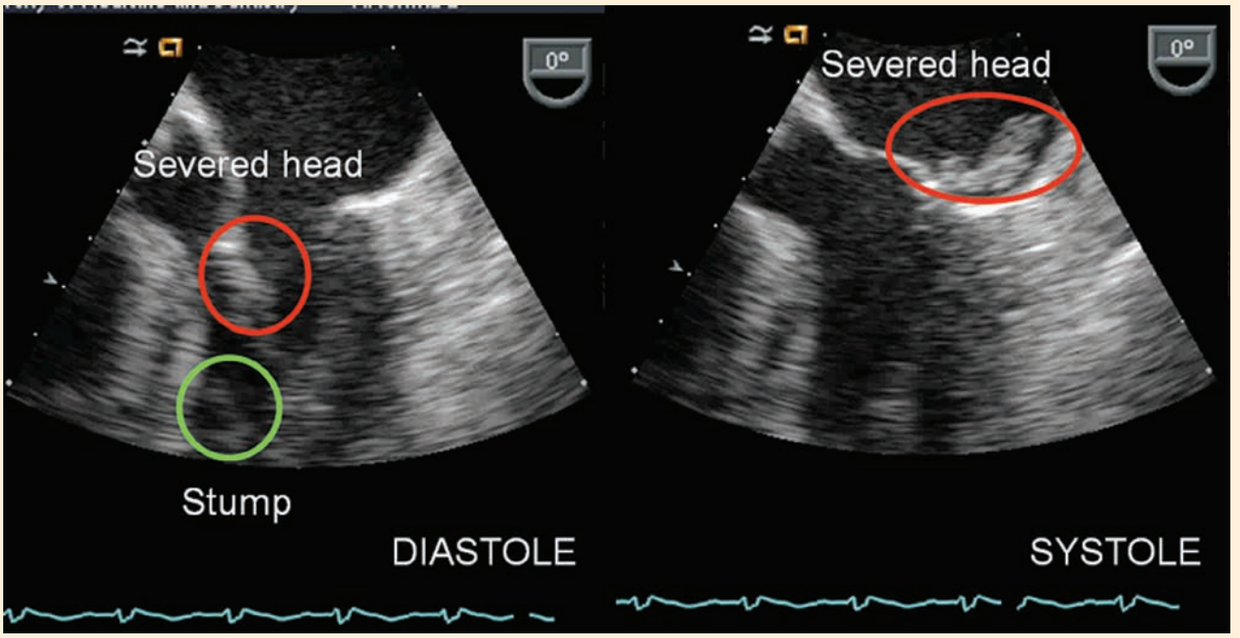


Fig. 7-30

(See also Video 7-3, rupture anterolateral papillary muscle; TEE image at 0 degree.)

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 count. In addition, a vegetation would appear as a shaggy, 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 which 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, 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, etc.

Answer (E) is incorrect because mitral valve prolapse due to myoxamtous 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.

KEY POINTS:

- Common causes of severe acute mitral regurgitation include papillary muscle rupture, myxomatous degeneration of mitral valve (which may lead to prolapsed and flail mitral leaflets), and mitral valve endocarditis.
- Papillary muscle rupture is a subacute complication of a myocardial infarction occurring usually 2–7 days after myocardial infarction.
- Rupture of the posterior papillary muscle is more common than the rupture of the anterior papillary muscle in survivors of myocardial infarction.

29. ANSWER: C. This patient has a 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.

$$PSG = 4 \times PSV2,$$
$$EDG = 4 \times EDV2,$$

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

In our patient:

$$PSG = 4 \times (6.0\ m/sec)^2 = 4 \times 36 = 144\ mm\ Hg,$$
$$EDG = 4 \times (3.8\ m/sec)^2 = 4 \times 14.4 = 58\ mm\ Hg.$$

By subtracting PSG and EDG from systolic and diastolic blood pressures, respectively, one can estimate pulmonary artery systolic blood pressure (SBP) and diastolic blood pressure (DBP)

$$PASP = SBP - PSG,$$
$$PADP = DBP - EDG.$$

In our patient:

$$PASP = 170 - 144 = 26\ mm\ Hg,$$
$$PADP = 70 - 58 = 12\ mm\ Hg.$$

Therefore, answer B is correct. This patient's calculations are summarized in this table.

TABLE 7-30

	Velocity (m/sec)	PDA Gradient (mm Hg)	Blood Pressure (mm Hg)	Estimated Pulmonary Artery Pressure (mm Hg)
Systole	6.0	144	170	26
Diastole	3.8	58	70	12

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/sec) without aliasing in an adult.

Answer (D) is incorrect because PASP in this patient is 30 mm Hg as calculated above.

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.

KEY POINTS:

- In uncomplicated PDA, there is a continuous flow from the descending thoracic aorta into the pulmonary artery.
- Maximum flow velocity of PDA occurs at peak systole while the minimum velocity occurs at end diastole.
- If the blood pressure of a patient with PDA is measured at the time of PDA flow velocity recordings, one can calculate both the pulmonary artery systolic and pulmonary artery diastolic pressures using the peak systolic and end diastolic velocities of PDA flow.

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

$$EOA = 2 \times \pi \times r^2 \times \frac{Valias}{Vmax},$$

where r is the PISA radius, Valias is the PISA aliasing velocity, and Vmax is the peak systolic velocity across the PDA.

In our patient:

$$EOA = 2 \times 3.14 \times (0.5\ cm)^2 \times 41/500 = 0.13\ cm^2.$$

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

Assumting 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:

$$A = \left(\frac{d}{2}\right)^2 \times \pi,$$

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):

$$d = 2 \times \sqrt{\frac{0.13}{3.14}} = 0.4\ cm = 4\ mm.$$

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

KEY POINTS:

- Proximal isovelocity surface area (PISA) method can be used to calculate the size of the aortic orifice of a PDA.
- The following three parameters are needed to calculate the PDA orifice area: PISA radius, aliasing velocity at which PISA is measured, and the peak velocity of the PDA flow.
- The diameter of a PDA usually ranges between 0.9 and 11.2 mm (median 2.6 mm).

31. 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 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 jet, left atrial pressure, and systolic blood pressure.

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

Using the peak velocity of the mitral regurgitant jet, one can calculated the peak systolic pressure gradient (ΔPMR) between the left ventricle (LV) and the left atrium (LA):

$$\Delta PMR = 4\,V2,$$

where V is the peak velocity of the mitral regurgitant jet.

In our patient:

$$\Delta PMR = 4 \times (8\ m/sec)^2 = 4 \times 64 = 256\ mm\ Hg.$$

Step 2: Calculate the peak LVSP.

By definition, ΔPMR is the difference between the peak LVSP and the LA pressure (LAP):

$$\Delta PMR = LVSP - LAP.$$

Solving for LVSP:

$$LVSP = \Delta PMR + LAP.$$

In our patient:

$$LVSP = 256\ mm\ Hg + 10\ mm\ Hg = 266\ mm\ Hg.$$

Step 3: Calculate maximal instantaneous left ventricular outflow gradient (ΔPLVOT)

ΔPLVOT is the pressure difference between the LVSP and the systolic blood pressure (SBP):

$$\Delta PLVOT = LVSP - SBP.$$

In our patient:

$$\Delta PLVOT = 266\ mm\ Hg - 144\ mm\ Hg = 122\ mm\ Hg.$$

Therefore, answer (C) is correct. All these calculations are summarized in Figure 7-31A:

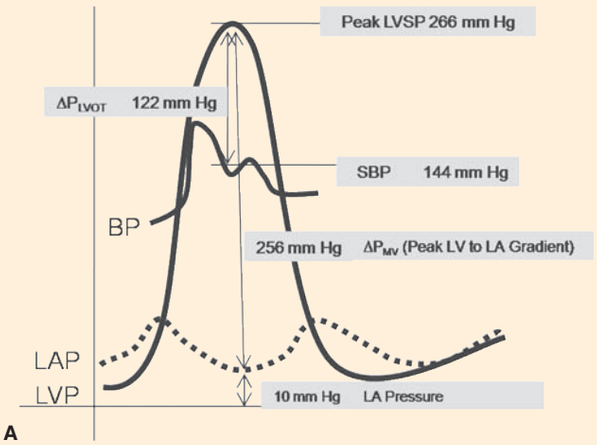


Fig. 7-31A

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 mitral regurgitant 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 7-31B.

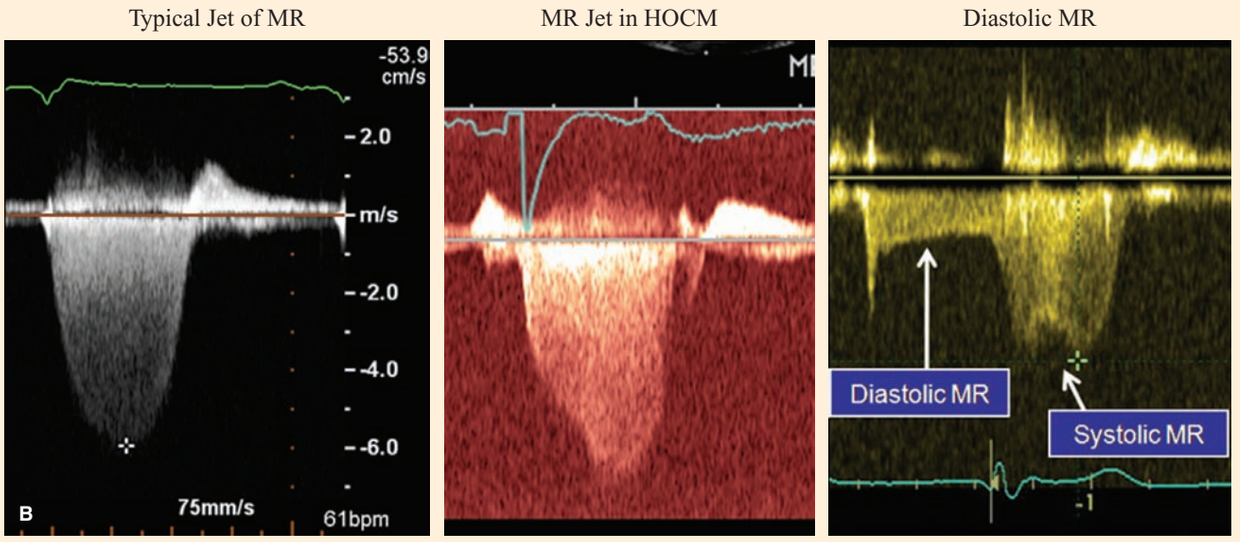


Fig. 7-31B

Answer (E) is incorrect because the peak LV systolic pressure in this patient is 266 mm Hg as calculated above.

KEY POINTS:

- In a patient with hypertrophic cardiomyopathy and a left ventricular outflow gradient, the flow velocity pattern of a mitral regurgitant jet typically peaks late in systole.
- Using the peak velocity of the mitral regurgitant flow and the systolic blood pressure, one can indirectly calculate the peak instantaneous left ventricular outflow gradient.
- Diastolic mitral regurgitation is not typically associated with hypertrophic obstructive cardiomyopathy.

32. ANSWER: C. In this patient with HOCM, jet #1 represents the systolic flow velocity pattern across the LVOT, and jet #2 represents the flow velocity pattern of the mitral regurgitant (MR) jet.

Jet #1 has a sawtooth 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 $\Delta P = 4V2$ formula, we can calculate the peak systolic instantaneous gradient (ΔPLVOT) across the LVOT, where V represents the peak velocity of jet #1.

$$\Delta\text{PLVOT} = 4 \times (3.8 \text{ m/sec})^2 = 4 \times 14.4 = 58 \text{ mm Hg.}$$

Since the pretreatment ΔPLVOT was 120 mm Hg, there was an approximately 50% drop in the gradient on the repeat echocardiogram:

$$\text{Percent drop in } \Delta\text{PLVOT} = (122 - 58)/122 = 64/122 \approx 50\%.$$

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 midsystole. Once the regurgitant orifice is created, mitral regurgitation continues until the end of systole. The difference in the shape of the mitral regurgitant spectral jet between mitral valve prolapse and HOCM is depicted in Figure 7-32A.

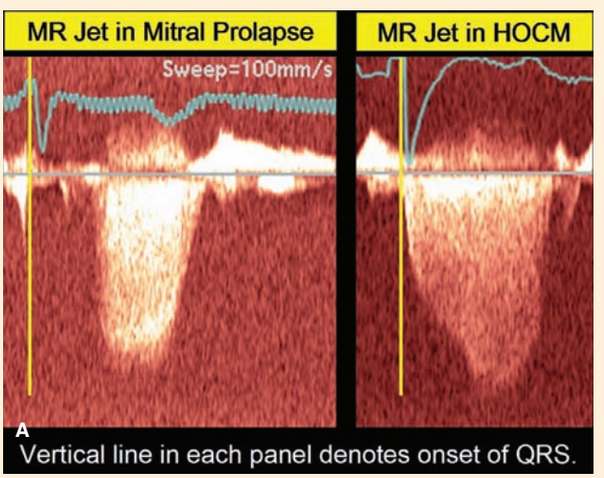
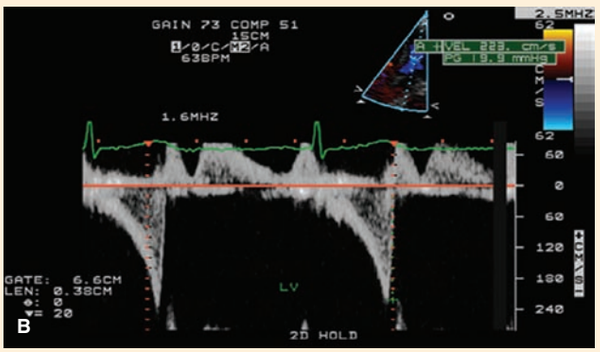


Fig. 7-32A

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. For further discussion of this topic, please see answer to question 23.

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 7-32B.



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

Fig. 7-32B

Answer (E) is incorrect because the peak LVSP is calculated as:

$$\text{LVSP} = \Delta P_{MR} + \text{LAP}, \qquad \text{(Eq. 1)}$$

where ΔPMR is the peak systolic gradient of the mitral regurgitant jet, and LAP is the left atrial pressure. After expressing ΔPMR in terms of the peak systolic velocity (V) of the mitral regurgitant jet, Eq. 1 becomes:

$$\text{LVSP} = 4 \times V^2 + \text{LAP}. \qquad \text{(Eq. 2)}$$

In our patient:

$$\text{LVSP} = 4 \times (6.3 \text{ m/sec})^2 + 10,$$
$$\text{LVSP} = 159 + 10 = 169 \text{ mm Hg}.$$

KEY POINTS:

- In a patient with hypertrophic obstructive cardiomyopathy, the left ventricular outflow gradient characteristically peaks late in systole.
- Using the peak velocity (V) of the flow velocity pattern across the left ventricular gradient, one can calculate the peak instantaneous pressure gradient (ΔP) as $\Delta P = 4V2$.
- Intracavitary left ventricular gradient peaks even later in systole compared to the left ventricular outflow gradient.

33. 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.

$$\text{LAV} = \frac{8 \times A1 \times A2}{3 \times \pi \times L}.$$

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

$$\text{LAV} = 0.85 \times \frac{A1 \times A2}{L}.$$

In our patient:

$$\text{LAV} = 0.85 \times \frac{27 \times 26}{5.6} = 107 \text{ ml}.$$

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

$$\text{LAVI} = \frac{\text{LAV}}{\text{BSA}}.$$

In our patient:

$$\text{LAVI} = 107 \text{ ml}/2.1 \text{ m}^2 \approx 50 \text{ ml/m}^2$$

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

TABLE 7-31

	LA Volume Index (ml/m^2)
Normal	≤28
Mild dilatation	29–33
Moderate dilatation	34–39
Severe dilatation	≥40

KEY POINTS:

- Area-length method is the recommended method for calculating the left atrial volume. Once calculated, the volume should be indexed for patient's body surface area.
- Left atrial volume should be calculated as an average of atrial volumes obtained in the apical 4-chamber and apical 2-chamber views.
- Chronic elevation of left atrial pressure leads to progressive increase in left atrial volume index.

34. ANSWER: B. The initial echocardiogram, which was obtained at the time of acutely decompensated heart failure, demonstrates the restrictive filling pattern. Because of the high left atrial pressures, 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 milliseconds). In the pulmonary venous spectral Doppler tracings, the peak of the systolic (S) wave is lower than the peak of the diastolic (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 7-33.

Answer (A) is incorrect because the preload has decreased from the initial to the 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 are required to distinguish normal (e' > 8 cm/sec) from pseudonormal pattern (e' < 8 cm/sec).

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 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.

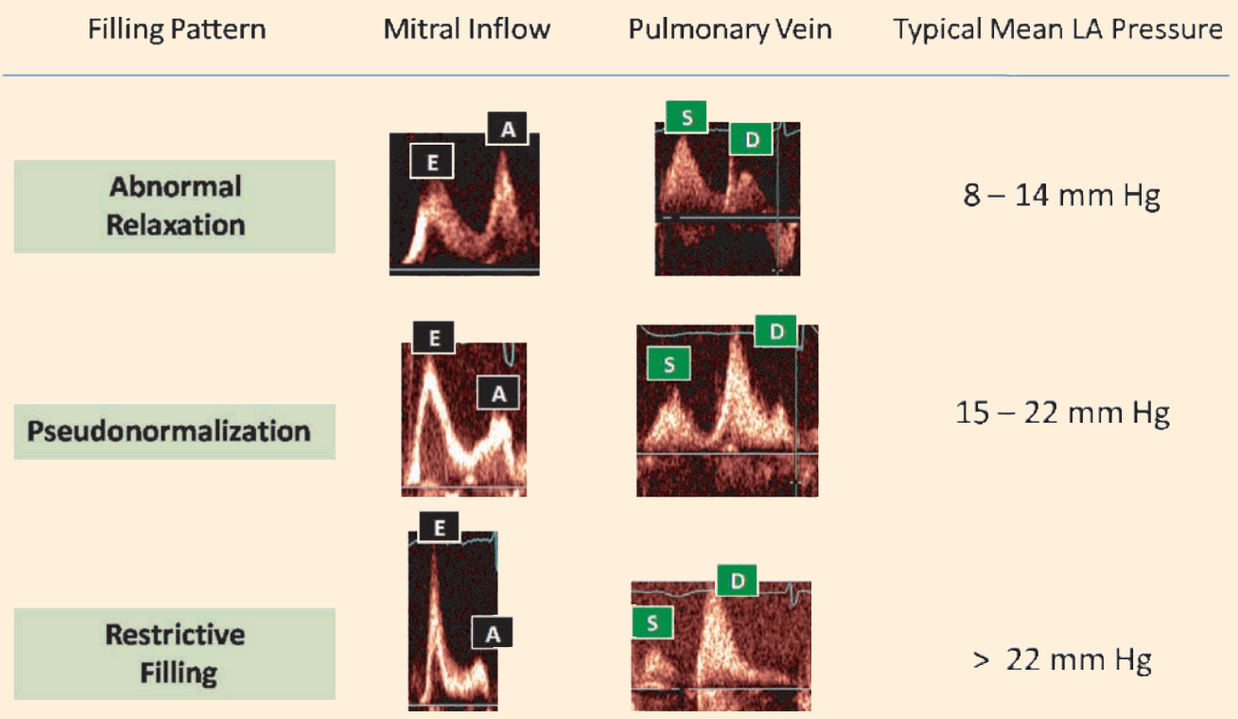


Fig. 7-33

KEY POINTS:

- In a healthy elderly individual, mitral inflow E wave has a lower peak velocity than the A wave (E<A) and there is prolonged E-wave deceleration time. In the pulmonary venous flow velocity recordings, the peak velocity of the systolic (S) wave is typically higher than the peak velocity of the diastolic (D) wave (S>D) in that age group.
- With left atrial pressure elevation, there is a progressive increase in the velocity of the mitral E wave, shortening of its deceleration time, and an increase in the E/A ratio. In addition, there is an inverse relationship between the left atrial pressure and the peak velocity of the pulmonary vein S wave; the higher the left atrial pressure, the lower the peak velocity of the S wave and lower the S/D ratio.
- Therapy with diuretics in patients with elevated left atrial pressure typically leads to lowering of the peak velocity of the mitral E wave and an increase in the peak velocity of the pulmonary vein S wave.

35. ANSWER: D. The three recordings from this patient are consistent with the diagnosis of constrictive pericarditis.

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 which would be consistent with constrictive pericarditis. In contrast, tamponade is characterized by impediment in early diastolic filling and an E<A.

COLOR M MODE—The flow propagation velocity (*V*p) of the early diastolic mitral flow is normal (66 cm/sec). Normal *V*p values are age dependent as shown in this table.

TABLE 7-32

	Normal *V*p (cm/sec)
Young	>55
Elderly	>45

*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, *V*p is normal. This is in contrast to restrictive cardiomyopathy which is a myocardial disorder characterized by impaired relaxation and compliance. In restrictive cardiomyopathy, *V*p is low.

INFERIOR VENA CAVA—In constrictive pericarditis, there is plethora of the inferior vena cava (IVC) as demonstrated by M-mode recordings of 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) as discussed in answer to question 1. Such a finding is consistent with the diagnosis with constrictive pericarditis. However, IVC plethora is also found in outer conditions of elevated right atrial pressure such as tricuspid stenosis, severe tricuspid regurgitation, right ventricular infarct, etc.

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 right atrial pressure.

Answer (C) is incorrect because *V*p in this patient is normal (>55 cm/sec).

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

KEY POINTS:

- In constrictive pericarditis, there is respiratory variation in the peak velocity of the mitral E wave due to ventricular interdependence.
- Flow propagation velocity (*V*p) of the mitral E wave is typically normal in constrictive pericarditis.
- Inferior vena cava is often plethoric in patients with constrictive pericarditis. This plethora is not specific for constrictive pericarditis as it occurs in other conditions that lead to significant elevation in the right atrial pressure (such as tricuspid valve stenosis or right ventricular systolic dysfunction).

36. 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 as explained in the answer to question 21.

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 this table.

TABLE 7-33

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

KEY POINTS:

- Paradoxical interventricular septal motion can occur with each cardiac beat or may be phasic with respiration.
- Examples of paradoxical septal motion with each beat include right ventricular pressure or volume overload, left bundle branch block, and status postpericardiotomy.
- Paradoxical septal motion that is phasic with respiration is encountered in constrictive pericarditis, tamponade, labored breathing, obesity, pulmonary embolism, etc.

SUGGESTED READINGS

1. Lang RM, Bierig M, Devereux RB, et al. Chamber Quantification Writing Group; American Society of Echocardiography's Guidelines and Standards Committee; European Association of Echocardiography. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005;18:1440–1463.
2. Kircher BJ, Himelman RB, Schiller NB. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. *Am J Cardiol.* 1990;66:493–496.
3. Libanoff AJ, Rodbard S. Atrioventricular pressure half-time. Measure of mitral valve orifice area. *Circulation*. 1968;38:144–150.
4. Hatle L, Angelsen B, Tromsdal A. Noninvasive assessment of atrioventricular pressure half-time by Doppler ultrasound. *Circulation.* 1979;60:1096–1104.
5. Nakatani S, Masuyama T, Kodama K, Kitabatake A, Fujii K, Kamada T. Value and limitations of Doppler echocardiography in the quantification of stenotic mitral valve area: comparison of the pressure half-time and the continuity equation methods. *Circulation*. 1988;77:78–85.
6. Bonow RO, Carabello BA, Chatterjee K, et al. ACC/AHA 2006 Guidelines for the Management of Patients with Valvular Heart Disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients with Valvular Heart Disease). *Circulation*. 2006;114:e84–e231.
7. Garcia MJ, Ares MA, Asher C, Rodriguez L, Vandervoort P, Thomas JD. An index of early left ventricular filling that combined with pulsed Doppler peak E velocity may estimate capillary wedge pressure. *J Am Coll Cardiol*. 1997;29:448–454.
8. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quiñones MA. Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. *J Am Coll Cardiol*. 1997;30:1527–1533.
9. Bargiggia GS, Bertucci C, Recusani F, et al. A new method for estimating left ventricular dP/dt by continuous wave Doppler-echocardiography. Validation studies at cardiac catheterization. *Circulation*. 1990;82:316–317.
10. Rossvoll O, Hatle LK. Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound: relation to left ventricular diastolic pressures. *J Am Coll Cardiol*. 1993;21:1687–1696.
11. Abdalla I, Murray RD, Lee JC, et al. Duration of pulmonary venous atrial reversal flow velocity and mitral inflow a wave: new measure of severity of cardiac amyloidosis. *J Am Soc Echocardiogr.* 1998;11:1125–1133. [Au3]
12. Hatle LK, Appleton CP, Popp RL. Differentiation of constrictive pericarditis and restrictive cardiomyopathy by Doppler echocardiography *Circulation*. 1989;79:357–370.
13. von Bibra H, Schober K, Jenni R, Busch R, Sebening H, Blomer H. Diagnosis of constrictive pericarditis by pulsed Doppler echocardiography of the hepatic vein. *Am J Cardiol.* 1989;63:483–488.
14. Pass RH, Hijazi Z, Hsu DT, Lewis V, Hellenbrand WE. Multicenter USA Amplatzer patent ductus arteriosus occlusion device trial. *J Am Coll Cardiol.* 2004;44:513–519.
15. Xie GY, Berk MR, Smith MD, Gurley JC, DeMaria AN. Prognostic value of Doppler transmitral flow patterns in patients with congestive heart failure. *J Am Coll Cardiol.* 1994;24:132–139.
16. Abbasi AS, Eber LM, MacAlpin RN, Kattus AA. Paradoxical motion of interventricular septum in left bundle branch block. *Circulation*. 1974;49:423–427.
17. Reynolds HR, Tunick PA, Grossi EA, Dilmanian H, Colvin SB, Kronzon I. Paradoxical septal motion after cardiac surgery: a review of 3,292 cases. *Clin Cardiol*. 2007;30:621–623.
18. Rakowski H, Appleton C, Chan KL, et al. Canadian consensus recommendations for the measurement and reporting of diastolic dysfunction by echocardiography: from the Investigators of Consensus on Diastolic Dysfunction by Echocardiography. *J Am Soc Echocardiogr.* 1996;9:736–760.
19. Nagueh SF, Appleton CP, Gillebert TC, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. *J Am Soc Echocardiogr.* 2009;22:107–133.

AQ1] Please define “PPD,” if deemed necessary.
[AQ2: Please query au to confirm this is the proper placement for these figures
[AQ3] Pls check the article title in Ref. [11]