History and Physical Examination

1

1.1. A 25-year-old woman presents for routine follow up. She has a 2/6 ejection systolic murmur best heard in the second left intercostal space with normal S1. The S2 is split during inspiration only, and P2 intensity is normal. No apical or parasternal heave. The murmur diminishes during expiration and standing up. What is the likely cause of the murmur?

A. Physiological or normal
B. Atrial septal defect (ASD)
C. Bicuspid aortic valve
D. Hypertrophic obstructive cardiomyopathy (HOCM)

1.2. A 29-year-old pregnant woman was found to have a systolic murmur best heard in the second left intercostal space. It is rough and there was a palpable thrill in the same area and in the suprasternal notch. Patient is asymptomatic and has normal exercise tolerance. What is the likely explanation for the murmur?

A. Pulmonary stenosis (PS)
B. Normal flow murmur due to increased cardiac output
C. Posterior mitral leaflet prolapse causing an anteriorly directed jet
D. Mammary soufflé

1.3. A 22-year-old patient has a hypoplastic radial side of the forearm and fingerized thumb. What might this be associated with?

A. ASD
B. Tetralogy of Fallot
C. Coarctation of aorta
D. Ebstein's anomaly

1.4. A 28-year-old man presented with a history of shortness of breath on exertion. On examination, the pulse rate was 76 bpm and blood pressure (BP) 126/80 mmHg. The left ventricular apex was prominent and forceful. The S1 and S2 were normal, but there was a 2/6 ejection systolic murmur best heard in the third right intercostal space. There was no appreciable variation with respiration, but there was an increase in intensity with the Valsalva maneuver and on standing up. It seemed to be less prominent on squatting. There was no audible click. This patient is likely to have?

A. Valvular aortic stenosis
B. Hypertrophic obstructive cardiomyopathy (HOCM)
C. Mitral valve prolapse (MVP)
D. Innocent murmur

Cardiology Board Review, Second Edition. Ramdas G. Pai and Padmini Varadarajan.

1.5. A 36-year-old asymptomatic woman was found to have a systolic murmur best heard in the apex, but also in the aortic area. It was mid to late systolic and was associated with a sharp systolic sound. What is the likely cause of the murmur?
- **A.** Posterior mitral leaflet prolapse
- **B.** Anterior mitral leaflet prolapse
- **C.** Valvular aortic stenosis
- **D.** Aortic subvalvular membrane

1.6. A 78-year-old man with hypertension and diabetes mellitus presented with exertional shortness of breath of 6months' duration. Examination revealed a 4/6 crescendo-decrescendo or ejection systolic murmur best heard in the second right intercostal space. The first component of the second sound was soft. The murmur was also heard along the right carotid artery. What is this patient likely to have?
- **A.** Mild aortic stenosis
- **B.** Moderate or severe aortic stenosis
- **C.** Pulmonary stenosis
- **D.** MR

1.7. A thrill and a continuous machinery murmur in the left infraclavicular area is indicative of what?
- **A.** Patent ductus arteriosus (PDA)
- **B.** Increased flow due to left arm arteriovenous (AV) fistula for dialysis
- **C.** Venous hum
- **D.** Pulmonary AV fistula

1.8. Which of the following is not a feature of aortic coarctation?
- **A.** A continuous murmur on the back
- **B.** Lower blood pressure in the legs compared with an arm
- **C.** Radiofemoral delay
- **D.** Pistol shot sounds on femoral arteries

1.9. A 22-year-old newly immigrant woman was referred to the high-risk pregnancy clinic because of clubbing and cyanosis. Examination in addition revealed a parasternal heave, 4/6 ejection systolic murmur in the third left intercostal space, normal jugular venous pressure (JVP), and oxygen saturation of 75%. What will you recommend after confirmation of the diagnosis?
- **A.** Continue pregnancy with sodium restriction
- **B.** Continue pregnancy, but deliver at 28 weeks
- **C.** Advise termination of pregnancy
- **D.** Perform percutaneous ASD closure and continue pregnancy

1.10. What is the cause of murmur in ASD?
- **A.** Continuous due to flow across the defect
- **B.** Ejection systolic due to increased flow across the pulmonary valve
- **C.** Mid-diastolic due to increased flow across the tricuspid valve
- **D.** Continuous murmur over lung fields due to increased flow in lungs

1.11. What is a systolic click that disappears on inspiration likely to be due to?
- **A.** Pulmonary valvular stenosis
- **B.** Bicuspid aortic valve

C. MVP
D. Pulmonary hypertension

1.12. A 36-year-old woman presented with an 8-month history of progressive exertional dyspnea. Physical examination revealed a heart rate of 74 bpm, regular, BP 126/78 mmHg, with no pedal edema. JVP and carotid upstroke were normal. Cardiac auscultation revealed normal S1, an accentuated P2 with narrow splitting of S2, an ejection click, and a 2/6 ejection systolic murmur. What is the likely diagnosis?
A. Pulmonary hypertension
B. PS
C. Aortic stenosis
D. ASD

1.13. Causes of prominent "a" wave in jugular venous pulsations include all of the following except which option?
A. PS
B. Pulmonary hypertension
C. Tricuspid stenosis
D. Aortic stenosis
E. ASD

1.14. What is a 6-year-old Amish boy in Pennsylvania with short stature, polydactyly, short limbs, absent upper incisor teeth with dysplasia of other teeth, and a systolic murmur most likely to have?
A. ASD
B. Ventricular septal defect
C. Aortic coarctation
D. PS

1.15. Which of the following describes a ventricular septal defect murmur?
A. Holosystolic
B. Ejection systolic
C. Systolic-diastolic
D. None of the above

1.16. Clubbing and cyanosis in lower limbs, but not upper limbs, is indicative of which of the following?
A. PDA with coarctation of the aorta
B. PDA with pulmonary hypertension
C. Ventricular septal defect Eisenmenger's
D. ASD Eisenmenger's with coarctation of aorta

1.17. A 46-year-old man presented with progressive fatigue and leg swelling. He had no significant past medical history except a front-on collision in a car he was driving. Examination revealed 2+ edema, raised JVP, and an enlarged liver, which seemed to expand during systole. What is the likely diagnosis?
A. Severe tricuspid stenosis
B. Severe tricuspid regurgitation (TR)
C. Constrictive pericarditis
D. Restrictive cardiomyopathy

1.18. A 23-year-old has a mid-diastolic rumble and sharp early diastolic sound. What is the likely explanation?
- **A.** Mitral stenosis
- **B.** Constrictive pericarditis
- **C.** Restrictive cardiomyopathy
- **D.** Bicuspid aortic valve

1.19. A 28-year-old man has history of progressive fatigue and exertional shortness of breath over the previous 6 months. Examination revealed a raised JVP that seemed to increase with inspiration and a sharp precordial sound in early diastole. What is the most likely diagnosis?
- **A.** Right ventricular infarct
- **B.** Tricuspid stenosis
- **C.** Constrictive pericarditis
- **D.** Restrictive cardiomyopathy

1.20. A 66-year-old woman with left breast cancer post mastectomy, radiation, and chemotherapy was admitted with shortness of breath, heart rate of 120 bpm, and BP of 90/60 mmHg. On slow cuff deflation during BP measurement, Korotkoff's sounds started at 90 mmHg during expiration only and throughout the respiratory cycle at a cuff pressure of 70 mmHg. An echocardiogram was obtained. What is this likely to show?
- **A.** Akinesis of the left anterior descending area
- **B.** Thick pericardium
- **C.** Large pericardial effusion
- **D.** Large, globally hypokinetic left ventricle.

1.21. Features of restrictive cardiomyopathy may include all of the following except?
- **A.** Raised JVP
- **B.** Loud S3
- **C.** Kussmaul's sign
- **D.** A diastolic knock in the pulmonary area

1.22. Pulsus paradoxus despite tamponade may not be present in which of the following?
- **A.** ASD
- **B.** Aortic stenosis
- **C.** Mitral stenosis
- **D.** Old age

1.23. Pulsus paradoxus may occur in all of the following except?
- **A.** Tamponade
- **B.** Status asthmaticus
- **C.** Pulmonary embolism
- **D.** Aortic stenosis

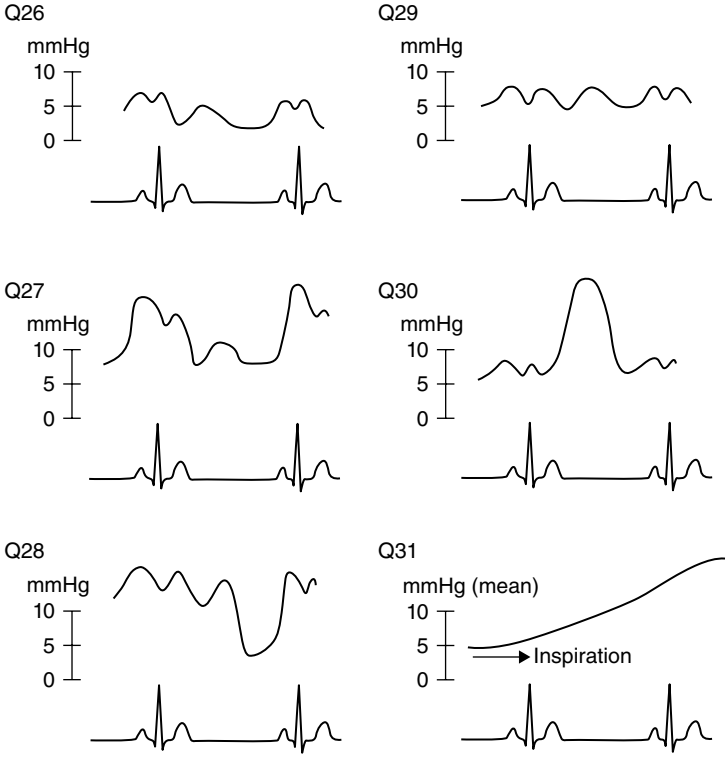
1.24. A Square sign during Valsalva maneuver occurs in which of the following?
- **A.** HOCM
- **B.** MVP
- **C.** Aortic stenosis
- **D.** Congestive heart failure

1.25. An abnormal Schamroth's test may be found in all of the following except?

- **A.** Tetralogy of Fallot
- **B.** Subacute bacterial endocarditis
- **C.** Left atrial myxoma
- **D.** Aortic stenosis

1.26–1.31. For the jugular vein or RA pressure tracings shown in Figures 1.26–1.31, match with an appropriate clinical scenario from the following choices:

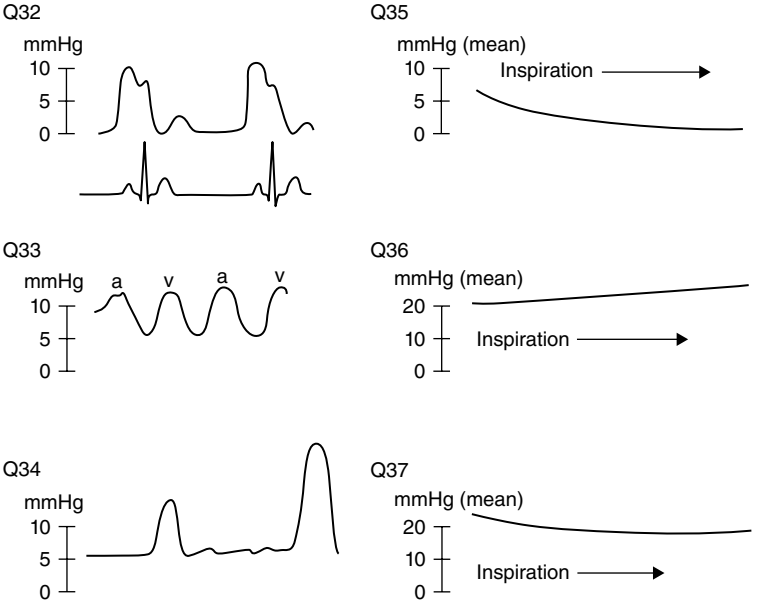
- **A.** Normal
- **B.** Pericardial constriction
- **C.** Restrictive cardiomyopathy
- **D.** ASD
- **E.** Tricuspid stenosis
- **F.** TR
- **G.** Cardiac tamponade
- **H.** Superior vena cava syndrome
- **I.** Heart failure
- **J.** PS



Figures 1.26–1.31

1.132–1.37. For the jugular vein or RA pressure tracings shown in Figures 1.32–1.37, match with an appropriate clinical scenario from the following choices:

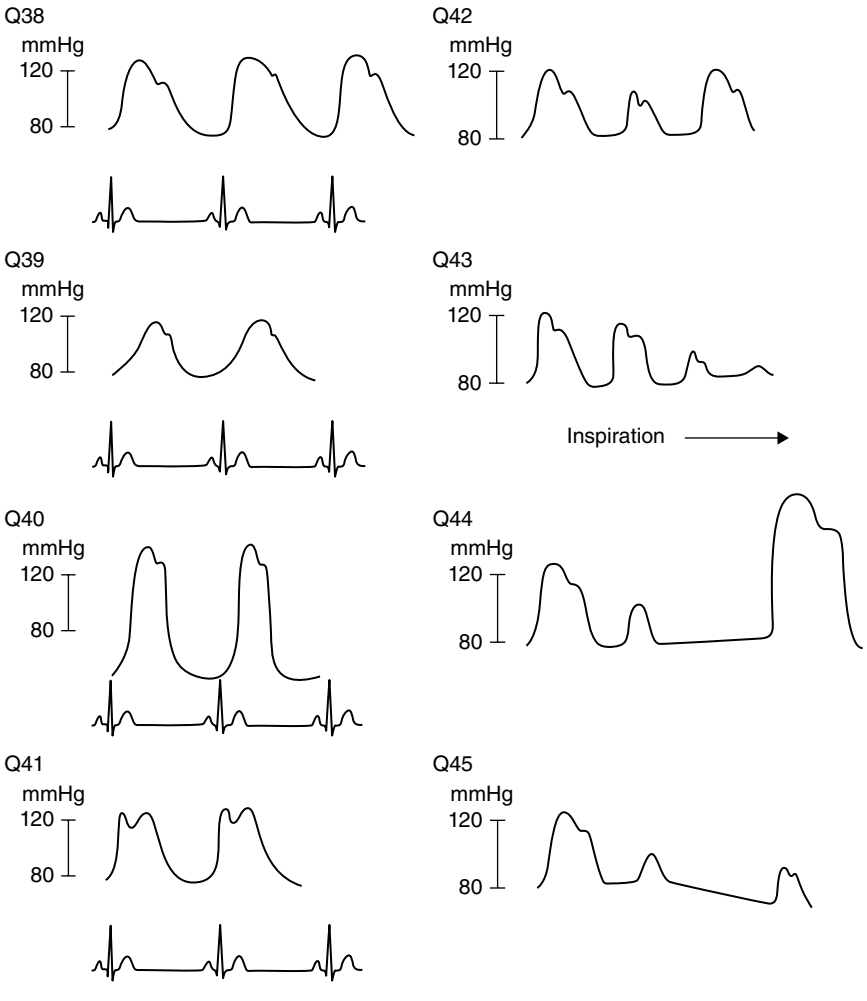
- **A.** Normal
- **B.** Pericardial constriction
- **C.** Restrictive cardiomyopathy
- **D.** ASD
- **E.** Tricuspid stenosis
- **F.** TR
- **G.** Cardiac tamponade
- **H.** Superior vena cava syndrome
- **I.** Heart failure
- **J.** PS
- **K.** Complete heart block



Figures 1.32–1.37

1.138–1.45. For the carotid pulse or arterial pressure tracings shown in Figures 1.38–1.45, match with an appropriate clinical scenario from the following choices:

- **A.** Normal
- **B.** Constriction
- **C.** Aortic stenosis
- **D.** AR
- **E.** Mitral stenosis
- **F.** Mixed aortic stenosis and AR
- **G.** Cardiac tamponade
- **H.** HOCM
- **I.** Heart failure
- **J.** Complete heart block
- **K.** MR
- **L.** Premature ventricular contraction (PVC)



Figures 1.38–1.45

Answers

1.1. A. Physiological or normal.

The murmur is <3/6 in intensity and diminishes with standing, when venous return is less, indicating a flow murmur. S2 split during inspiration only is physiological. During inspiration, increased venous return and pulmonary flow prolongs right ventricular (RV) ejection. This delays P2. The S2 split will be fixed with a similar gap during both inspiration and expiration. Characteristics of a physiological systolic murmur include being 2/6 in intensity or less, diminution during standing and normal physiological split.

1.2. A. Pulmonary stenosis (PS).

A murmur associated with a thrill indicates it is pathological and not just due to increased cardiac output. A thrill in the suprasternal notch is pathognomonic of PS. Valvular PS would be associated with an ejection click which diminishes with inspiration. Left parasternal heave would be indicative of RV hypertrophy (RVH) secondary to pressure overload on the RV imposed by PS. Murmur because

of posterior mitral leaflet prolapse may be heard in the aortic area as the mitral regurgitation jet may hug the ascending aorta and the murmur may be transmitted along that. Mammary soufflé occurs in lactating women because of increased blood flow to the breast and the murmur is continuous.

1.3. A. ASD.

The features are suggestive of Holt-Oram syndrome. They can also have unequal arm length, various other deformities of the hand, VSD and varying degrees of atrioventricular conduction disturbances. Cardiac anomalies are present in 75% of patients with Holt-Oram syndrome which is a genetic defect.

1.4. B. HOCM.

In HOCM, the left ventricular (LV) outflow obstruction is dynamic and is increased by an increase in LV contractility or a reduction in LV size. Standing, Valsalva maneuver, and amyl nitrite inhalation reduce venous return, reduce LV filling, reduce LV size and increase systolic anterior motion of the anterior mitral leaflet (SAM), resulting in increased LV outflow tract (LVOT) obstruction. Squatting kinks the leg arteries, raising peripheral resistance and hence an increase in LV volume through an increase in afterload. This increase in LV size reduces SAM and LV outflow obstruction. Valvular aortic stenosis murmur intensity is flow dependent, and hence reduction in venous return with standing or Valsalva maneuver as well as an increase in peripheral resistance with squatting would diminish the murmur. In mitral valve prolapse (MVP), an increase in LV volume would reduce prolapse, and a reduction in LV volume would lengthen the murmur by producing earlier prolapse. MVP is generally associated with a mid-systolic click.

1.5. A. Posterior mitral leaflet prolapse.

The systolic click and late systolic murmur indicate MVP. In anterior leaflet prolapse, the mitral regurgitation (MR) jet is directed posteriorly and murmur may be conducted to the axilla. In posterior leaflet prolapse, the jet is anterior wall hugging, along the aortic root, which facilitates its conduction to the aortic area.

1.6. B. Moderate or severe aortic stenosis.

This is a classic aortic stenosis murmur with carotid conduction. Soft A2 (first component of S2 indicates significant aortic stenosis). Features of severe aortic stenosis are late-peaking murmur, absent A2, paradoxic splitting of S2, (i.e. split during expiration instead of inspiration), and a slow-rising carotid pulse (pulsus parvus et tardus).

1.7. A. Patent ductus arteriosus (PDA).

Though the other conditions can produce continuous murmurs, they are not associated with a thrill or machinery character. Pulmonary AV fistula results in a continuous murmur over the lung fields. The venous hum is due to flow in the jugular veins and gets less by assuming the supine position. The murmur due to AV fistula created for hemodialysis is heard on the side of the fistula in the clavicular area due to increased venous flow – this can be temporarily silenced by transient pressure over the fistula.

1.8. D. Pistol shot sounds on femoral arteries.

Pistol shot sounds occur in severe aortic regurgitation (AR). Others are features of aortic coarctation. Continuous murmur on the back is due to chest wall arterial collaterals bypassing aortic coarctation. Radiofemoral delay and lower BP in the

leg are due to obstruction in the aorta and collateral dependent flow in the lower extremities. In severe coarctation, the flow in the abdominal aorta would be non-pulsatile and continuous.

1.9. C. Advise termination of pregnancy.

The findings are typical of tetralogy of Fallot, and the murmur is due to PS. Murmur of ASD is due to flow and softer and not associated with clubbing or cyanosis unless associated with Eisenmenger's syndrome. Tetralogy of Fallot is associated with extremely high risk, and the pregnancy should be terminated. Other very high-risk cardiac conditions include severe pulmonary hypertension, severe LV dysfunction or prior history of peripartum cardiomyopathy, and severe left-sided obstructive valvular diseases.

1.10. B. Ejection systolic due to increased flow across the pulmonary valve.

Flow across the defect and the lungs does not produce murmurs. The gradient across ASD is less than 1 mmHg and does not produce turbulence to produce a murmur. It takes a torrential shunt to produce a mid-diastolic flow murmur across the tricuspid valve as the tricuspid aortic valve area of 7–8 cm^2 (compared to 5 cm^2 for the mitral valve and about 3.5 cm^2 for pulmonary and aortic valves). Flow murmurs are generated across smaller valves.

1.11. A. Pulmonary valvular stenosis.

In severe PS, due to low pulmonary artery (PA) pressure, an increase in venous return to right ventricle during inspiration may open the pulmonary valve before systole, eliminating the ejection click.

1.12. A. Pulmonary hypertension.

This is suggested by loud P2. In pulmonary hypertension, P2 moves closer to A2 due to higher pulmonary valve closure pressure, and S2 may be single when PA pressure approaches systemic pressure. Ejection click and soft ejection systolic murmur may occur because of PA dilation. Other features of pulmonary hypertension may include a palpable PA in the second left intercostal space, a palpable P2 or diastolic knock, parasternal heave due to RVH and a prominent "a" wave in jugular venous pulsation due to RVH resulting in accentuated right atrial (RA) systole. PS results in a louder murmur and diminished P2. ASD results in wide, fixed, splitting of S2.

1.13. E. ASD.

A prominent "a" wave occurs due to forceful right atrial systole against some resistance, and this can occur against stenotic tricuspid valve, hypertrophied RV (PS and pulmonary hypertension) or hypertrophied ventricular septum (aortic stenosis, hypertrophic cardiomyopathy, or hypertension). In ASD, a defect in the atrial septum would not allow a prominent "a" wave, even in the presence of pulmonary hypertension, as the right atrium would decompress into the left atrium during atrial systole.

1.14. A. ASD.

ASD, or common atrium as part of Ellis-van Creveld syndrome (EVC) or mesoectodermal or chondroectodermal dysplasia, is an autosomal recessive inheritance disorder that occurs in the old-order Amish population. The *EVC* gene is on chromosome number 4, short arm. It is a form of ciliopathy. Other

ciliopathies that result in abnormal organogenesis include Bardet-Biedl syndrome, polycystic kidney and liver disease, Alstrom syndrome, Meckel-Gruber syndrome, and some forms of retinal degeneration, and so on.

1.15. A. Holosystolic as the pressure gradient between LV and RV is throughout LV isovolumic contraction, ejection and isovolumic relaxation. On echo-Doppler, there may also be a presystolic left-to-right flow associated with left atrial systole.

1.16. B. PDA with pulmonary hypertension.

In PDA Eisenmenger's, the shunt reversal through PDA causes desaturation in the lower part of the body only, resulting in central cyanosis and clubbing in lower extremities and not upper extremities (differential cyanosis). In ASD and VSD, Eisenmengers, cyanosis and clubbing involve both the upper and lower extremities. In PDA without suprasystemic pulmonary hypertension, the flow will be from the aorta to pulmonary artery only and no desaturated blood comes to the aorta.

1.17. B. Severe tricuspid regurgitation (TR).

Liver that is pulsatile (expansile) in systole is indicative of a powerful right atrial "V" wave, and this suggests severe TR. This can also be seen in the jugular venous pulsation. Sternal compression during the motor vehicle accident likely caused a flail tricuspid valve and severe TR. Liver pulsation in tricuspid stenosis is presystolic. Liver pulsations are not seen in constriction or restriction.

1.18. A. Mitral stenosis.

This is typical mitral stenosis with pliable leaflets, which causes opening snap (OS). The A2–OS interval is a good measure of mitral stenosis severity. Normally, 70–100 ms (same as isovolumetric relaxation time). Less than 70 ms indicates high left atrial pressure, suggesting severe mitral stenosis. In constrictive pericarditis, you can get a pericardial knock which is a sharp protodiastolic sound that occurs a little later. Restrictive cardiomyopathy produces S3 due to high left atrial pressure and is generally later and is a dull sound like a thud and best heard with the bell of the stethoscope. S3 is due to rapid deceleration of the early passive filling wave across the mitral valve.

1.19. C. Constrictive pericarditis.

A rise in JVP is paradoxical (Kussmaul's sign), and sharp protodiastolic sound is a pericardial knock – classic features of constriction. Kussmaul's sign occurs due to lack of transmission of negative intrathoracic pressure to the right atrium through the rigid pericardium. Hence, the increased venous return during inspiration causes a rise in RA pressure. In tricuspid stenosis, inspiratory increase in venous return may not readily empty into the right ventricle, causing a paradoxical rise in JVP with inspiration. Kussmaul's sign can occur in an RV infarct but occurs in the setting of inferior myocardial infarction. Restrictive cardiomyopathy may be associated with S3, which is less sharp (a thud), but no venous paradox.

1.20. C. Large pericardial effusion.

This is typical cardiac tamponade with classic paradoxic pulse, hypotension, and tachycardia. Constriction causes venous paradox.

1.21. C. Kussmaul's sign.

As the pericardium is normal in restrictive cardiomyopathy, inspiratory negative intrathoracic pressure is transmitted to the pericardial space and cardiac chambers, and hence the venous paradox does not occur. Diastolic knock is palpable with loud P2; a feature of pulmonary hypertension that it is common in restrictive LV physiology.

1.22. A. ASD.

Pulsus paradoxus may not be present when LV filling is not affected by phase of inspiration due to lack of interventricular dependence, as in ASD, or LV filling from other sources, such as severe AR or MR.

1.23. D. Aortic stenosis.

1.24. D. Congestive heart failure.

See the explanation in Box 1.1 Clinical Pearls. This is because though a Valsalva maneuver reduces left-sided venous return, it does not affect LV stroke volume as it is already well filled and beyond the peak of Starling's curve. An increase in intrathoracic pressure is transmitted to the aorta, causing an increase in aortic pressure.

1.25. D. Aortic stenosis.

This test is named after a South African cardiologist for diagnosis of clubbing. In clubbing, the angle between the nail and nail fold is >165° and when nails of the fingers from both sides are apposed, the gap between them disappears. Clubbing is seen in cardiac conditions (in addition to a variety of pulmonary diseases), in subacute bacterial endocarditis, congenital cyanotic heart diseases, and left atrial myxoma.

1.26. A. Normal.

Note the mean RA pressure of <5 mmHg and the "a" wave due to atrial systole is slightly higher than the "V" wave, which occurs because of RA filling on the closed tricuspid valve. A smaller "V" wave indicates a compliant right atrium. The sharp "C" wave that coincides with the QRS complex of the electrocardiogram is due to a combination of tricuspid valve closure and transmitted carotid impulse.

1.27. E. Tricuspid stenosis.

Note the large "a" wave as the RA contracts against a stenosed tricuspid valve; the gradient is reflected as a large "a" wave. Note the mean RA pressure is also slightly elevated commensurate with trans tricuspid gradient. In conditions causing RVH (pulmonary hypertension, PS, aortic stenosis with septal hypertrophy), the "a" wave may be prominent due to noncompliant RV (RV fourth heart sound), but mean RA pressure may not be high unless there is heart failure.

1.28. B. Pericardial constriction.

Note the elevated RA pressure with rapid "Y" descent.

1.29. D. ASD.

In ASD, the "a" and "V" waves would be of similar height because the defect leads to equilibration of the LA and RA pressures.

1.30. F. TR.

The large "V" wave is due to TR filling up the RA during systole. When the "V" wave pressure is about 25–30 mmHg, it may become palpable to the examining finger and associated with an expansile liver.

1.31. B. Pericardial constriction.

The increasing mean RA pressure with inspiration is the venous paradox or Kussmaul's sign. This is due to dissociation between intrathoracic and intrapericardial pressures because of a thick pericardium. During inspiration, the intrathoracic pressure drops, increasing venous return to the right atrium, but the intrapericardial and intra-RA pressure does not drop and returning blood increases the pressure further. Kussmaul's sign can occur even in acute pericarditis and RV infarcts; the latter invoking pericardial restraint.

1.32. J. PS.

Note a large "a" wave with near-normal mean RA pressure. Contrast this with tricuspid stenosis.

1.33. G. Cardiac tamponade.

Note the raised RA pressure with prominent "X" and "Y" troughs.

1.34. K. Complete heart block.

The intermittent large waves are "cannon a" waves when atria and ventricles happen to contract simultaneously due to AV dissociation.

1.35. A. Normal.

The RA pressure is <5 mmHg and drops with inspiration.

1.36. H. Superior vena cava syndrome.

Note the high JVP which does not drop on inspiration as the superior vena cava is blocked and the jugular vein is not in communication with right atrial hemodynamics or pulsatile changes. In contrast to constriction, the JVP in superior vena cava syndrome is nonpulsatile. It would be high in both.

1.37. I. Heart failure.

High JVP that drops with inspiration.

1.38. A. Normal.

This is the typical normal tracing. Note the fairly rapid upstroke, pulse pressure of about 40 mmHg, dicrotic notch and dicrotic wave.

1.39. C. Aortic stenosis.

Note the very slow rise, attributable to high blood flow velocity across the valve which converts pressure to kinetic energy.

1.40. D. AR.

Note the rapid upstroke, rapid downstroke (water-hammer pulse), wide pulse pressure, and low diastolic pressure due to peripheral vasodilation. This can also occur in PDA and large AV fistulae.

1.41. F. Mixed aortic stenosis and AR.

This is called pulsus bisferiens or double pulse. Can also occur in HOCM.

1.42. I. Heart failure.

This is pulsus alternans. Alternating strong and weak pulse with regular RR interval due to alternating stronger and weaker myocardial contraction with every

other beat attributable to altered calcium handling by contractile proteins. It is a sign of severe systolic dysfunction.

1.43. G. Cardiac tamponade.

A BP drop with inspiration is called pulsus paradoxus. An inspiratory drop of >10 mmHg may indicate tamponade.

1.44. L. Premature ventricular contraction (PVC).

With appropriate increase in pulse pressure in the post PVC beat, because of a combination of increased preload due to a long filling period and increased contractility due to the force-frequency relationship.

1.45. H. HOCM.

In this patient, after PVC, instead of an augmented pulse there is a smaller pulse volume. This is due to the fact that increased contractility in the post-PVC beat increases dynamic LVOT obstruction and reduces stroke volume. This is called Brockenbrough phenomenon on cardiac catheterization. This contrasts with valvular aortic stenosis, where pulse pressure increases after a PVC.

Box 1.1 Clinical Pearls

- Loud S1 occurs when the mitral valve closes forcefully from an open position against the transmitral gradient or prematurely and occurs in mitral stenosis, short pulmonary regurgitation (PR) interval, and hyperdynamic circulation.
- S1 is soft in MR, with a long PR interval, and severe AR. In severe AR, the mitral valve may move toward closure in presystole.
- P2 can be loud in pulmonary hypertension (higher pulmonary valve closure sound) and dilated PA (better P2 transmission).
- S2 may be paradoxically split when aortic valve closure is delayed and comes after P2 as in left bundle branch block, severe aortic stenosis, severe LV dysfunction, and PDA (increased transaortic flow). S2 is split in expiration and not in inspiration when RV filling and ejection time are increased.
- The only right-sided sound/murmur is attenuated by inspiration is pulmonary ejection click of valvular PS as an inspiratory increase in venous return to the right ventricle in the presence of RVH may cause an increase on RV end diastolic pressure high enough to open the pulmonary valve as the PA pressure is lower.
- A Carey Coombs murmur is a mid-diastolic mitral murmur due to mitral valvulitis in rheumatic fever.
- An Austin Flint murmur is a mitral mid-diastolic murmur heard in severe AR. Potential explanations include: AR jet causing an anterior mitral leaflet vibration, or mitral/AR jet interaction or an AR jet causing a partial diastolic closure of anterior mitral leaflet.
- A Graham Steel murmur is an early diastolic murmur of PR that occurs in severe pulmonary hypertension. A higher PA diastolic pressure causes turbulence of the PR jet and the murmur.
- A mid-diastolic murmur of mitral stenosis is best heard with the bell without much pressure as it is a low-frequency murmur and better heard in the left lateral position. Presystolic accentuation indicates atrial contraction.

- A tapping apex typically occurs in mitral stenosis and it is palpable S1.
- Maneuvers are helpful in evaluating murmurs. Inspiration increases right-sided venous return and augments right-sided murmurs. Standing reduces venous return and, after three or four beats, LV filling as well reducing its size. This may augment an HOCM murmur and lengthen an MR murmur of MVP. Squatting kinks limb arteries and increases afterload. This may reduce murmurs of valvular aortic stenosis through reduced stroke volume and an HOCM murmur through an increase in LV volume and shorten a murmur of MVP through increasing LV volume and reducing prolapse.
- The Valsalva maneuver involves expiration against a closed glottis, increasing the intrathoracic pressure. There are four phases of heart rate and BP response. The normal response includes the following:
  - Phase 1. Early during the Valsalva maneuver, pressure on the intrathoracic aorta and pulmonary veins emptying into left heart transiently increases filling, stroke volume, and BP with reflex slowing of heart.
  - Phase 2. With a continued Valsalva maneuver, LV filling diminishes and lowers stroke volume and BP, resulting in compensatory tachycardia. Reflex peripheral vasoconstriction slowly increases BP and lowers the heart rate.
  - Phase 3. With release of the Valsalva maneuver, pressure on the aorta drops, and BP drops with some increase in heart rate.
  - Phase 4. The left ventricle fills, with an increase in stroke volume with continued peripheral vasoconstriction causing BP to overshoot the baseline value. This will reflexively reduce the heart rate below the baseline.

  Phase 2 is used in dynamic auscultation.
- In HOCM, the Valsalva maneuver results in a smaller left ventricle size causing increased LVOT obstruction and an increase in murmur intensity.
- In MVP, reduced LV volume with phase 2 Valsalva results in earlier and greater prolapse and the MR murmur lengthens.
- A heaving apex denotes LV hypertrophy; forceful apical lift signifies LV volume overload, tapping apex loud S1, and bifid apex with presystolic lift LV occurs due to a combination of LVH with a forceful atrial kick causing S4 as it occurs in hypertrophic cardiomyopathy.
- Left parasternal or precordial bulge indicates RVH occurring before the rib cage ossifies (young age), and parasternal lift or heave indicates RVH starting after childhood.
- A downward tug on the larynx held up with fingers after deglutition indicates aortic arch aneurysm. Also called tracheal tug or Oliver's sign, this occurs with every heartbeat.
- A large, sharp systolic wave in jugular venous pulsation during systole is the cannon wave. This occurs when the right atrium contracts over a closed tricuspid valve, as in complete heart block (intermittent) or junctional or idioventricular rhythm with retrograde ventriculoatrial conduction.