BRAUNWALD'S HEART DISEASE

REVIEW AND ASSESSMENT

NINTH EDITION

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PREFACE

Review and Assessment is a comprehensive study guide designed to accompany the ninth edition of *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine*, edited by Dr. Robert O. Bonow, Dr. Douglas L. Mann, Dr. Douglas P. Zipes, and Dr. Peter Libby. It consists of 706 questions that address key topics in the broad field of cardiovascular disease. A detailed answer is provided for each question, often comprising a "mini-review" of the subject matter. Each answer refers to specific pages, tables, and figures in *Braunwald's Heart Disease* and in most cases to additional pertinent citations. Topics of greatest clinical relevance are emphasized, and subjects of particular importance are intentionally reiterated in subsequent questions for reinforcement.

Review and Assessment is intended primarily for cardiology fellows, practicing cardiologists, internists, advanced medical residents, and other professionals wishing to review contemporary cardiovascular medicine in detail. The subject matter and structure are suitable to help prepare for the Subspecialty Examination in Cardiovascular Disease offered by the American Board of Internal Medicine.

All questions and answers in this book were designed specifically for this edition of *Review and Assessment*. I am grateful for the contributions by my colleagues at Brigham and Women's Hospital who expertly authored new questions and updated material carried forward from the previous edition: Dr. Marc Bonaca, Dr. Akshay Desai, Dr. Neal Lakdawala, Dr. Amy Miller, Dr. Fidencio Saldaña, Dr. Garrick Stewart, and Dr. Neil Wimmer. We are very appreciative to Dr. Sara Partington and Dr. Swathy Kolli for submitting many of the new noninvasive images and to the following colleagues who provided additional graphics or support to this edition: Dr. Ron Blankstein, Dr. Sharmila Dorbala, Dr. Eric Green, Dr. Raymond Kwong, and Dr. Saurabh Rohatgi. We also acknowledge and thank the Brigham and Women's Hospital team of cardiac ultrasonographers, led by Jose Rivero, who expertly obtained and alerted us to many of the images that appear in this book.

It has been a pleasure to work with the editorial and production departments of our publisher, Elsevier, Inc. Specifically, I thank Ms. Natasha Andjelkovic, Ms. Dolores Meloni, Ms. Angela Rufino, and Mr. John Casey for their expertise and professionalism in the preparation of this edition of *Review and Assessment*.

Finally, and as always, I am very grateful to each member of my family for their support and patience during the often-long hours required to prepare this text.

On behalf of the contributors, I hope that you find this book a useful guide in your review of cardiovascular medicine.

Leonard S. Lilly, MD
Boston, Massachusetts

CONTENTS

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Section I (Chapters 1 to 23)

FUNDAMENTALS OF CARDIOVASCULAR DISEASE; MOLECULAR BIOLOGY AND GENETICS; EVALUATION OF THE PATIENT

DIRECTIONS: For each question below, select the ONE BEST response.

QUESTION 1

A 54-year-old African-American man with a history of hypertension and hypercholesterolemia undergoes a treadmill exercise test using the standard Bruce protocol. He stops at 11 minutes 14 seconds because of fatigue, at a peak heart rate of 152 beats/min and peak systolic blood pressure of 200 mm Hg. The diastolic blood pressure declines by 5 mm Hg during exercise. During recovery, the systolic blood pressure decreases to 15 mm Hg below his pre-exercise pressure. There are no ischemic changes on the ECG during or after exercise. Which of the following is correct?

A. His peak systolic blood pressure during exercise exceeds that normally observed
B. The change in diastolic blood pressure during exercise is indicative of significant coronary artery disease
C. This test is nondiagnostic owing to an inadequate peak heart rate
D. These results are consistent with a low prognostic risk of a coronary event
E. The postexercise reduction in systolic blood pressure is suggestive of severe coronary artery disease

QUESTION 2

When present, each of the following heart sounds occurs shortly after S_2 EXCEPT:

A. Opening snap
B. Third heart sound
C. Ejection click
D. Tumor plop
E. Pericardial knock

QUESTION 3

A state-of-the-art blood test has been developed for the rapid, noninvasive diagnosis of coronary artery disease. The assay has a sensitivity of 90% and a specificity of 90% for the detection of at least one coronary stenosis of > 70%. In which of the following scenarios is the blood test likely to be of most value to the clinician?

A. A 29-year-old man with exertional chest pain who has no cardiac risk factors
B. A 41-year-old asymptomatic premenopausal woman
C. A 78-year-old diabetic woman with exertional chest pain who underwent two-vessel coronary stenting 6 weeks ago
D. A 62-year-old man with exertional chest pain who has hypertension, dyslipidemia, and a 2 pack-per-day smoking history
E. A 68-year-old man with chest discomfort at rest accompanied by 2 mm of ST-segment depression in the inferior leads on the ECG

QUESTION 4

A murmur is auscultated during routine examination of an 18-year-old asymptomatic college student, at the second left intercostal space, close to the sternum. The murmur is crescendo-decrescendo, is present throughout systole and diastole, and peaks simultaneously with S_2. It does not change with position or rotation of the head. Which of the following best describes this murmur?

A. This is a continuous murmur, most likely a venous hum commonly heard in adolescents
B. This is a continuous murmur resulting from mixed aortic valve disease
C. This is a continuous murmur due to a congenital shunt, likely a patent ductus arteriosus
D. Continuous murmurs of this type can only be congenital; murmurs due to acquired arteriovenous connections are purely systolic
E. This murmur, the result of left subclavian artery stenosis, is not considered continuous, because a continuous murmur can result only from an arteriovenous communication

QUESTION 5

Unequal upper extremity arterial pulsations commonly are found in each of the following disorders EXCEPT:

A. Aortic dissection
B. Takayasu disease

C. Supravalvular aortic stenosis
D. Subclavian artery atherosclerosis
E. Subvalvular aortic stenosis

QUESTION 6

A 58-year-old woman with metastatic breast cancer presents with exertional dyspnea and is found to have a large circumferential pericardial effusion, jugular venous distention, and hypotension. Which of the following echocardiographic signs is likely present?
A. Collapse of the right ventricle throughout systole
B. Exaggerated decrease in tricuspid inflow velocity during inspiration
C. Exaggerated decrease in mitral inflow velocity during inspiration
D. Exaggerated increase in left ventricular outflow tract velocity during inspiration
E. Markedly increased E/A ratio of the transmitral Doppler velocity profile

QUESTION 7

Which of the following statements about pulsus paradoxus is correct?
A. Inspiration in normal individuals results in a decline of systolic arterial pressure of up to 15 mm Hg
B. Accurate determination of pulsus paradoxus requires intra-arterial pressure measurement
C. Pulsus paradoxus in tamponade is typically accompanied by the Kussmaul sign
D. Pulsus paradoxus is unlikely to be present in patients with significant aortic regurgitation, even in the presence of tamponade
E. Pulsus paradoxus is common in patients with hypertrophic cardiomyopathy

QUESTION 8

Which of the following electrocardiographic features is typical of left anterior fascicular block?
A. Q waves in the inferior leads
B. Mean QRS axis between 0 and –30 degrees
C. QRS duration >0.12 millisecond
D. rS pattern in the inferior leads and qR pattern in lateral leads
E. Marked right-axis deviation

QUESTION 9

Each of the following combinations has the potential for significant pharmacologic interaction and drug toxicity EXCEPT:
A. Simvastatin and erythromycin
B. Sildenafil and nitroglycerin
C. Pravastatin and ketoconazole
D. Cyclosporine and St. John's wort
E. Digoxin and verapamil

QUESTION 10

Each of the following conditions is a contraindication to exercise stress testing EXCEPT:
A. Symptomatic hypertrophic obstructive cardiomyopathy
B. Advanced aortic stenosis
C. Acute myocarditis
D. Abdominal aortic aneurysm with transverse diameter of 5.5 cm
E. Unstable angina

QUESTION 11

A 42-year-old woman with hypertension and dyslipidemia underwent a 1-day rest-stress exercise myocardial perfusion single-photon emission computed tomography (SPECT) study with technetium-99m imaging to evaluate symptoms of "atypical" chest pain. Her resting ECG showed left ventricular hypertrophy. She exercised for 12 minutes 30 seconds on the standard Bruce protocol and attained a peak heart rate of 155 beats/min. She developed a brief sharp parasternal chest pain during the test that resolved quickly during recovery. Based on the images in Figure 1-1, which of the following statements is correct?
A. The SPECT myocardial perfusion images are diagnostic of transmural myocardial scar in the distribution of the mid-left anterior descending coronary artery
B. The anterior wall defect on the SPECT images is likely an artifact due to breast tissue attenuation
C. Thallium-201 would have been a better choice of radiotracer to image this patient
D. Gated SPECT imaging cannot differentiate attenuation artifacts from a true perfusion defect
E. A transmural scar is associated with reduced wall motion but normal wall thickening on gated SPECT imaging

QUESTION 12

Which of the following statements regarding the second heart sound (S_2) is TRUE?
A. Earlier closure of the pulmonic valve with inspiration results in physiologic splitting of S_2
B. Right bundle branch block results in widened splitting of S_2
C. Paradoxical splitting of S_2 is the auscultatory hallmark of an ostium secundum atrial septal defect
D. Fixed splitting of S_2 is expected in patients with a right ventricular electronically paced rhythm
E. Severe pulmonic valvular stenosis is associated with a loud P_2

QUESTION 13

A 56-year-old asymptomatic man with a history of hypertension and cigarette smoking is referred for a screening exercise treadmill test. After 7 minutes on the standard Bruce protocol, he is noted to have 1 mm

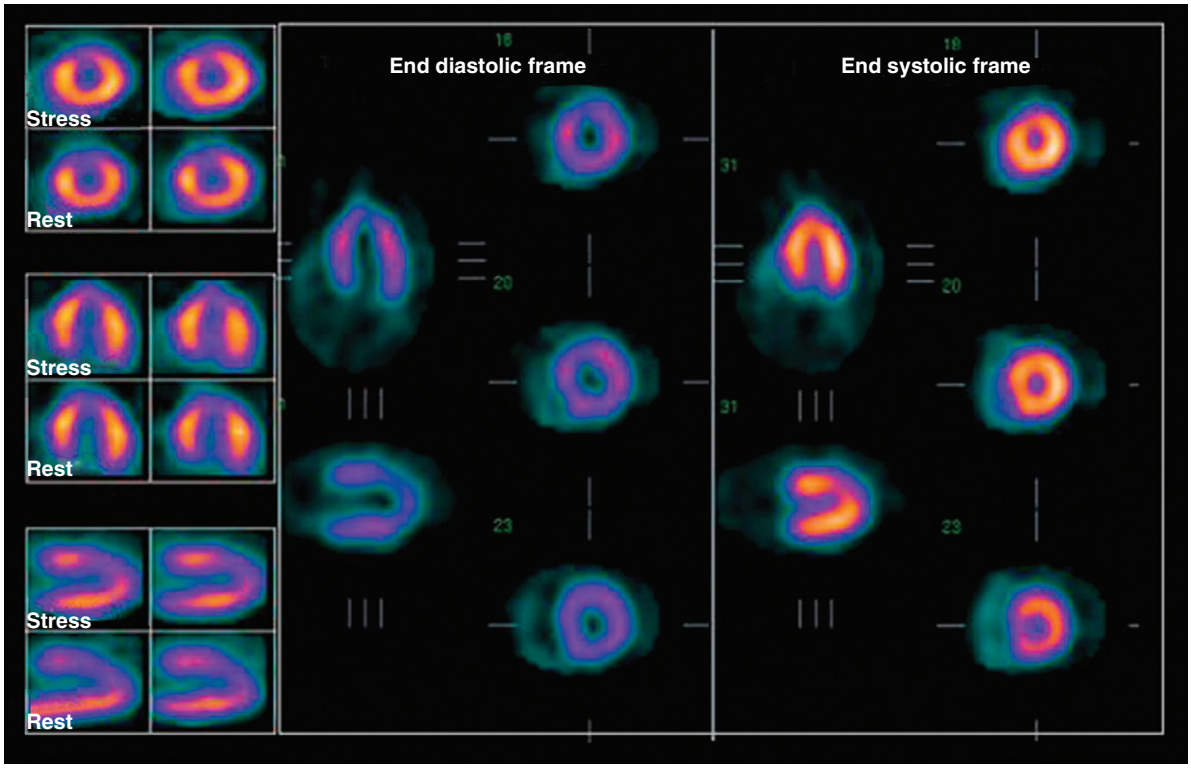

FIGURE 1-1

of flat ST-segment depression in leads II, III, and aVF. He stops exercising at 9 minutes because of leg fatigue and breathlessness. The peak heart rate is 85% of the maximum predicted for his age. The ST segments return to baseline by 1 minute into recovery. Which of the following statements is correct?

A. This test is conclusive for severe stenosis of the proximal right coronary artery
B. His risk of death due to an acute myocardial infarction during the next year is >50%
C. He should proceed directly to coronary angiography
D. The test predicts a 25% risk of cardiac events over the next 5 years, most likely the development of angina
E. This is likely a false-positive test

QUESTION 14

In which of the following clinical scenarios do ST-segment depressions during standard exercise testing increase the diagnostic probability of significant coronary artery disease?

A. A 56-year-old man with left bundle branch block and a family history of premature coronary disease
B. A 45-year-old woman with diabetes and hypertension, with left ventricular hypertrophy on her baseline ECG
C. A 76-year-old woman with new exertional dyspnea, a history of cigarette smoking, and a normal baseline ECG
D. A 28-year-old woman with pleuritic left-sided chest pain after a gymnastics class
E. A 63-year-old man with exertional dyspnea on beta-blocker, digoxin, and nitrate therapies

QUESTION 15

Which of the following statements regarding cardiac catheterization is TRUE?

A. The risk of a major complication from cardiac catheterization is 2.0% to 2.5%
B. The incidence of contrast-induced nephrotoxicity in patients with renal dysfunction is decreased with intravenous administration of mannitol before and after the procedure
C. High osmolar nonionic contrast agents demonstrate a reduced incidence of adverse hemodynamic reactions compared with low osmolar ionic contrast agents
D. One French unit (F) is equivalent to 0.33 mm
E. Retrograde left-sided heart catheterization is generally a safe procedure in patients with tilting-disc prosthetic aortic valves

QUESTION 16

A 75-year-old woman was brought to the cardiac catheterization laboratory in the setting of an acute myocardial infarction. She had presented with chest pain, epigastric discomfort, and nausea. Physical examination was pertinent for diaphoresis, heart rate 52 beats/min, blood pressure 85/50 mm Hg, jugular venous distention, and slight bilateral pulmonary rales. Coronary angiography demonstrated ostial occlusion of a dominant right coronary artery, without significant left-sided coronary artery disease. The presenting ECG likely showed all of the following features EXCEPT:

A. ST-segment elevation in leads II, III, and aVF
B. ST-segment depression in leads V_1 and V_2
C. Sinus bradycardia
D. ST-segment elevation in lead V_4R
E. PR-segment depression

QUESTION 17

Using Doppler echocardiography methods, the following values are obtained in a patient with a restrictive ventricular septal defect (VSD) and mitral regurgitation: systolic transmitral flow velocity = 5.8 m/sec and systolic flow velocity at the site of the VSD = 5.1 m/sec. The patient's blood pressure is 144/78 mm Hg. The estimated right ventricular systolic pressure is (choose the single best answer):
A. 35 mm Hg
B. 40 mm Hg
C. 45 mm Hg
D. 50 mm Hg
E. Not able to be determined from the provided information

QUESTION 18

Which of the following statements regarding left bundle branch block (LBBB) is TRUE?
A. The majority of patients with LBBB do not have structural heart disease
B. In LBBB, the S_2 is widely split with normal respiratory variation
C. The presence of LBBB is associated with significantly reduced long-term survival
D. In LBBB, the T wave vectors are oriented in the same direction as the QRS complex
E. LBBB does not impair myocardial performance

QUESTION 19

Which of the following statements regarding altered electrolytes and electrocardiographic abnormalities is TRUE?
A. Hypocalcemia causes prolongation of the QT interval
B. Hyperkalemia causes QRS narrowing and increased P wave amplitude
C. Hypomagnesemia is associated with monomorphic ventricular tachycardia
D. Hypokalemia causes peaked T waves
E. Severe hypocalcemia has been associated with the presence of a J wave (Osborn wave)

QUESTION 20

For which of the following scenarios is the diagnostic sensitivity of standard exercise testing sufficient to forego additional imaging with either nuclear scintigraphy or echocardiography?
A. A 53-year-old woman with hypertension and left ventricular hypertrophy by echocardiography who has developed exertional chest pressure
B. A 74-year-old man with a history of cardiomyopathy with a normal baseline electrocardiogram on angiotensin-converting enzyme inhibitor, beta-blocker, and digoxin therapies
C. A 37-year-old asymptomatic woman with incidentally detected left bundle branch block
D. A 44-year-old male smoker with Wolff-Parkinson-White syndrome and a family history of coronary artery disease with new exertional chest discomfort
E. A 53-year-old man with hyperlipidemia, a normal baseline ECG, and sharp, fleeting chest pains

QUESTION 21

All of the following statements about the ECG depicted in Figure 1-2 are correct EXCEPT:
A. The basic rhythm is atrial fibrillation
B. The fifth QRS complex on the tracing is likely an example of aberrant conduction
C. The Ashman phenomenon is based on the fact that the refractory period is directly related to the length of the preceding RR interval
D. Right bundle branch block morphology is commonly present in Ashman beats
E. The bundle of His is the likely anatomic location of the conduction delay because it has the longest refractory period

QUESTION 22

The timing of an "innocent" murmur is usually:
A. Early systolic
B. Presystolic
C. Midsystolic
D. Holosystolic
E. Early diastolic

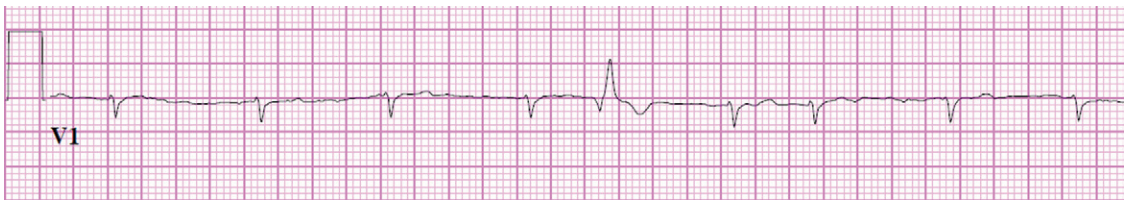

FIGURE 1-2 From Marriott HJL: Rhythm Quizlets: Self Assessment. Philadelphia, Lea & Febiger, 1987, p 14.

QUESTION 23

Which of the following statements about the jugular venous waveform is TRUE?

A. Kussmaul's sign is pathognomonic for constrictive pericarditis
B. The *c* wave is a reflection of ventricular diastole and becomes visible in patients with diastolic dysfunction
C. The *x* descent is less prominent than the *y* descent in cardiac tamponade
D. Phasic declines in venous pressure (the *x* and *y* descents) are typically more prominent to the eye than the positive pressure waves (the *a, c,* and *v* waves)
E. Cannon *a* waves indicate interventricular conduction delay

QUESTION 24

Which of the following statements regarding the measurement of cardiac output is correct?

A. In the thermodilution method, cardiac output is directly related to the area under the thermodilution curve
B. The thermodilution method tends to underestimate cardiac output in low-output states
C. In the presence of tricuspid regurgitation, the thermodilution method is preferred over the Fick technique for measuring cardiac output
D. A limitation of the Fick method is the necessity of measuring oxygen consumption in a steady state
E. Cardiac output is directly proportional to systemic vascular resistance

QUESTION 25

Which of the following conditions is associated with the Doppler transmitral inflow pattern shown in Figure 1-3?

A. Gastrointestinal hemorrhage
B. Constrictive pericarditis
C. Normal aging
D. Restrictive cardiomyopathy
E. Hyperthyroidism

QUESTION 26

A 32-year-old woman, a native of India, is referred by her primary care physician for further evaluation for dyspnea on exertion. On examination, both an opening snap and mid-diastolic rumble are appreciated at the apex. An echocardiogram is obtained. The transmitral Doppler tracing shown in Figure 1-4 permits accurate assessment of each of the following EXCEPT:

A. The presence of mitral stenosis
B. The presence, but not the severity, of mitral regurgitation
C. The transmitral diastolic pressure gradient
D. The etiology of the valvular lesion
E. The mitral valve area

QUESTION 27

A patient with a history of pulmonary embolism undergoes evaluation including noninvasive assessment by Doppler echocardiography. The following values are determined:

Right atrial pressure = 9 mm Hg
Peak systolic velocity across the tricuspid valve = 4 m/sec

What is this patient's right ventricular systolic pressure?

A. 64 mm Hg
B. 73 mm Hg
C. 50 mm Hg
D. 20 mm Hg
E. The information given is insufficient to determine the value

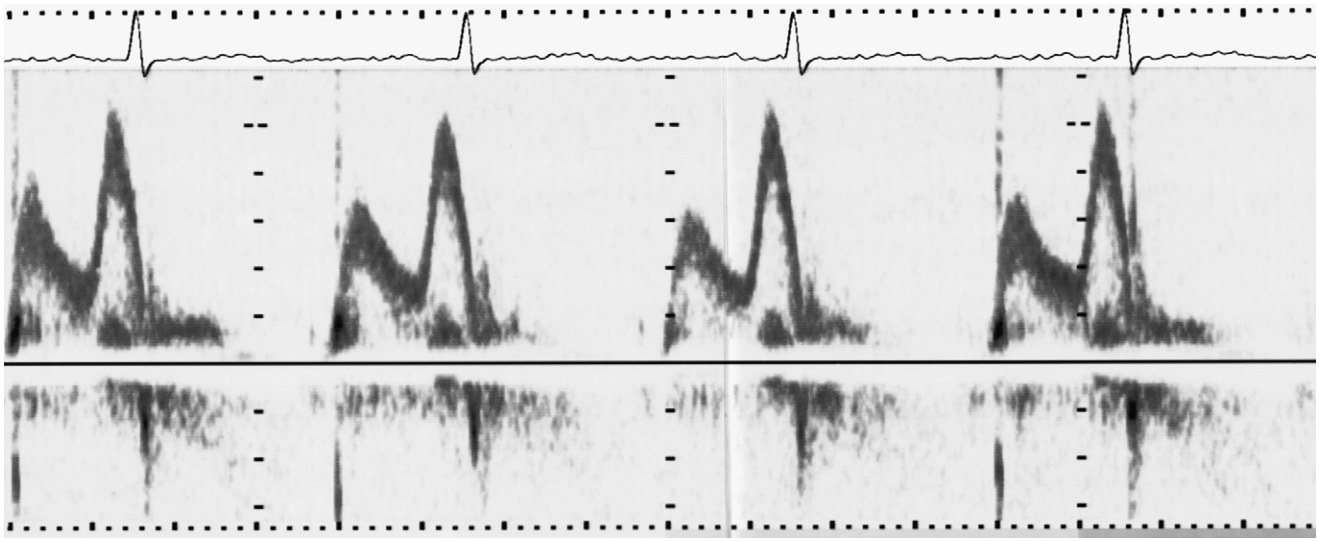

FIGURE 1-3

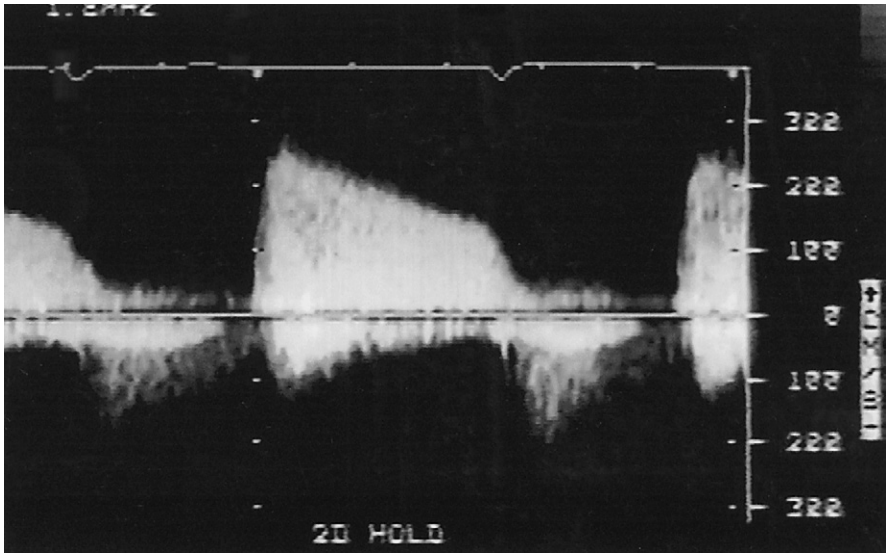

FIGURE 1-4

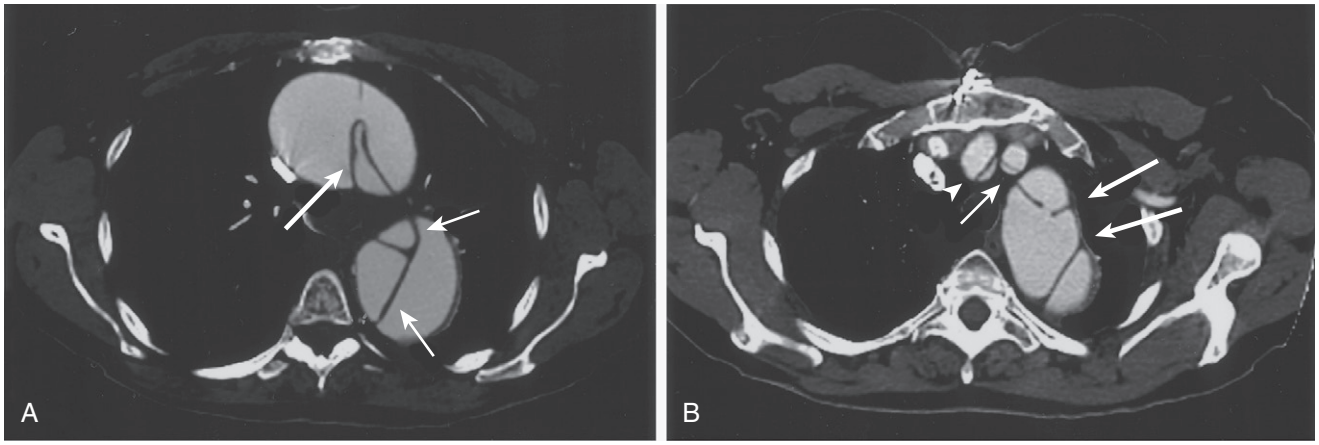

FIGURE 1-5 Courtesy of R. C. Gilkeson, MD, Case Western Reserve University, Cleveland, Ohio.

QUESTION 28

Which of the following statements is TRUE regarding the response to aerobic exercise by healthy older adults?

A. Ventricular stroke volume decreases with age such that there is an age-related fall in cardiac output during exercise
B. Systolic and diastolic blood pressures each rise significantly during aerobic exercise
C. A decline in beta-adrenergic responsiveness contributes to a fall in the maximum heart rate in older individuals
D. A normal adult's cardiac output doubles during maximum aerobic exercise
E. Maximum aerobic capacity does not change significantly with age in sedentary individuals

QUESTION 29

Physiologic states and dynamic maneuvers alter the characteristics of heart murmurs. Which of the following statements is correct?

A. In acute mitral regurgitation, the left atrial pressure rises dramatically so that the murmur is heard only during late systole
B. Rising from a squatting to a standing position causes the murmur of mitral valve prolapse to begin later in systole
C. The diastolic rumble of mitral stenosis becomes more prominent during the strain phase of a Valsalva maneuver
D. The murmur of aortic stenosis, but not mitral regurgitation, becomes louder during the beat after a premature ventricular contraction
E. The murmur of acute aortic regurgitation can usually be heard throughout diastole

QUESTION 30

Which of the following statements regarding the computed tomograms of the chest shown in Figure 1-5 is TRUE?

A. The patient's disorder should be managed medically, with surgical intervention considered only if there is evidence of secondary organ involvement

B. The left common carotid artery is spared by this process
C. The sensitivity of computed tomography for the diagnosis of this condition is >95%
D. Fewer than 50% of patients with this condition will report chest pain
E. Transesophageal echocardiography is necessary to confirm the diagnosis

QUESTION 31

Which of the following statements regarding ST-segment changes during exercise testing is TRUE?
A. The electrocardiographic localization of ST-segment depression predicts the anatomic territory of coronary obstructive disease
B. The J point is the proper isoelectric reference point on the ECG
C. J point depression during exercise is diagnostic for significant cardiac ischemia
D. Persistence of ST-segment depression for 60 to 80 milliseconds after the J point is necessary to interpret the electrocardiographic response as abnormal
E. ST-segment depression must be present both during exercise and in recovery to be interpreted as abnormal

QUESTION 32

An ECG is obtained as part of the routine preoperative evaluation of an asymptomatic 45-year-old man scheduled to undergo wrist surgery. The tracing is shown in Figure 1-6 and is consistent with:
A. Right ventricular hypertrophy
B. Left posterior fascicular block
C. Reversal of limb lead placement
D. Left anterior fascicular block and counterclockwise rotation
E. Dextrocardia with situs inversus

QUESTION 33

Which of the following statements is TRUE regarding exercise test protocols?
A. Regardless of the exercise protocol, the heart rate and systolic and diastolic blood pressures all must increase substantially to achieve a valid test
B. Bicycle, treadmill, and arm ergometry protocols all produce approximately equal heart rate and blood pressure responses
C. The standard Bruce protocol is characterized by only small increases in oxygen consumption between stages
D. A fall in systolic blood pressure during exercise is associated with severe coronary artery disease
E. An optimal graded treadmill exercise test rarely requires more than 5 minutes of exercise on the Bruce protocol

QUESTION 34

Which of the following patients is LEAST likely to have a cardiac cause of his/her recent onset of dyspnea?
A. An active 54-year-old man with a congenitally bicuspid aortic valve who has recently noticed shortness of breath walking his usual 18 holes of golf
B. A 70-year-old woman who sustained an anterior myocardial infarction 1 year ago with a left ventricular ejection fraction of 50% at that time. She has not had recurrent angina but has noted dyspnea during her usual housework over the past 2 months
C. A 46-year-old woman with a history of asymptomatic rheumatic mitral stenosis who recently noticed irregular palpitations and shortness of breath while climbing stairs
D. A 38-year-old woman with a previously asymptomatic ostium secundum atrial septal defect, now 8 months pregnant, who has noted shortness of breath during her usual weekly low-impact aerobics class

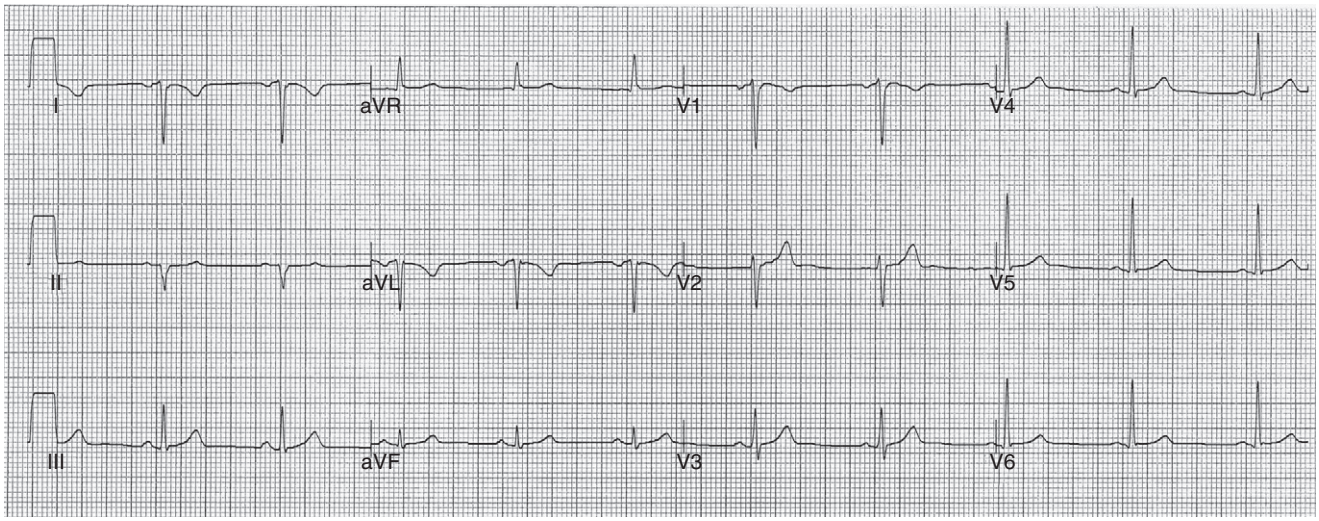

FIGURE 1-6

E. A 22-year-old man with trisomy 21 and a heart murmur who has described shortness of breath carrying grocery bundles over the past 3 months

QUESTION 35

A 68-year-old man with a history of diabetes, hypertension, and hyperlipidemia presents to the emergency department via ambulance, complaining of crushing substernal chest pain. Emergency Medical Services personnel report that anterior ST segments were elevated on the ECG en route. Which of the following electrocardiographic findings is LEAST likely in this patient experiencing an acute anterior ST-segment elevation myocardial infarction?

A. ST-segment elevation in leads V_2 to V_5
B. Shortened QT interval
C. New right bundle branch block
D. ST-segment depression in leads III and aVF
E. Hyperacute T waves in the precordial leads

QUESTION 36

All of the following statements regarding nuclear imaging and acute myocardial infarction (MI) are true EXCEPT:

A. The size of the resting myocardial perfusion defect after acute MI correlates with the patient's prognosis
B. Increased lung uptake of thallium-201 at rest correlates with an unfavorable prognosis
C. Submaximal exercise imaging soon after MI is a better predictor of late complications than adenosine myocardial perfusion imaging
D. Technetium-99m sestamibi imaging can be used to assess the effectiveness of thrombolytic therapy
E. Measuring infarct size by technetium-99m sestamibi imaging before discharge from the hospital is a reliable way to predict subsequent ventricular remodeling

QUESTION 37

Which of the following statements regarding ST-segment elevation during exercise testing is TRUE?

A. ST-segment elevation during exercise testing is a common finding in patients with coronary artery disease
B. ST-segment elevation in a lead that contains a pathologic Q wave at baseline indicates severe myocardial ischemia
C. The electrocardiographic leads that manifest ST-segment elevation during exercise localize the anatomic regions of ischemia
D. ST-segment elevation that develops during exercise is usually a manifestation of benign early repolarization
E. ST-segment elevation during exercise is commonly associated with the development of complete heart block

QUESTION 38

Which of the following statements regarding coronary calcium assessment by electron beam tomography (EBT) is TRUE?

A. The amount of calcium on EBT strongly correlates with the severity of coronary disease detected by angiography
B. Patients who benefit most from screening with EBT are those at a high risk for coronary events based on traditional risk factors
C. The absence of coronary calcium completely excludes the presence of severe obstructive coronary artery stenosis
D. Interpretation of the calcium score is independent of the patient's age and gender
E. A coronary calcium score higher than the median confers an increased risk of myocardial infarction and death

QUESTION 39

Which of the following statements is TRUE regarding prognosis as determined by myocardial perfusion imaging?

A. Patients with normal perfusion in the presence of angiographically documented coronary artery disease have very low rates of cardiac events (<1% per year).
B. Thallium is the preferred isotope for myocardial perfusion imaging in women
C. Transient ischemic dilatation of the left ventricle and lung uptake of the nuclear tracer imply the presence of minor coronary artery disease
D. The combination of clinical and cardiac catheterization data is more predictive of subsequent cardiac events than the combination of clinical and myocardial perfusion data
E. The risk of future cardiac events is unrelated to the number or extent of myocardial perfusion defects

QUESTION 40

A previously healthy 28-year-old man presented to the hospital because of 1 month of progressive exertional dyspnea, weakness, and weight loss. One day before hospitalization he was unable to climb one flight of stairs because of shortness of breath. On examination, he appeared fatigued with mild respiratory distress. His blood pressure was 110/70 mm Hg without pulsus paradoxus. His heart rate was 110 beats/min and regular. The jugular veins were distended without the Kussmaul sign. Pulmonary auscultation revealed scant bibasilar rales. The heart sounds were distant. There was mild bilateral ankle edema. As part of the evaluation during hospitalization, he underwent cardiac magnetic resonance imaging. A short-axis view at the midventricular level is shown in Figure 1-7. Which of the following is the most likely diagnosis?

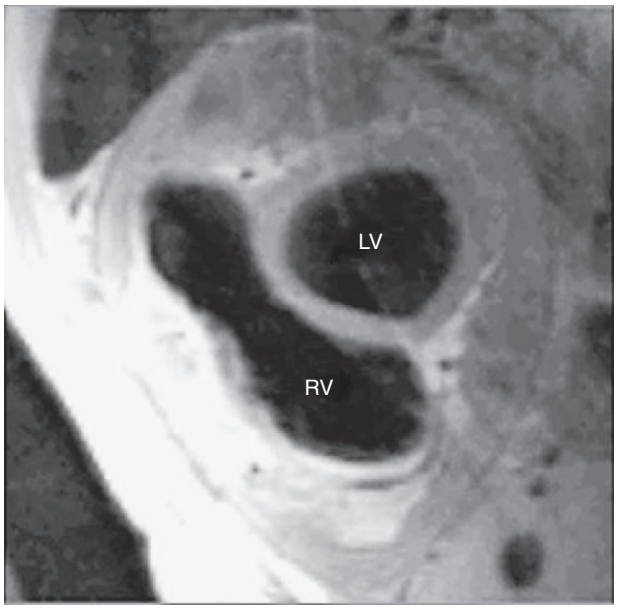

FIGURE 1-7

A. Pericardial malignancy
B. Chronic organized pericardial hematoma
C. Constrictive pericarditis
D. Extracardiac tumor compression of the heart
E. Congenital partial absence of the pericardium with cardiac herniation

QUESTION 41

Each of the following statements regarding intracardiac shunts is true EXCEPT:
A. A left-to-right shunt should be suspected if the difference in oxygen saturation between the superior vena cava (SVC) and the pulmonary artery is 8% or more
B. Oxygen saturation in the inferior vena cava is normally higher than that in the SVC
C. In a suspected atrial septal defect with left-to-right flow, mixed venous O_2 content should be measured at the level of the pulmonary artery
D. A pulmonic-to-systemic blood flow ratio of <1 indicates a net right-to-left shunt
E. Pulmonary artery oxygen saturation exceeding 80% should raise the suspicion of a left-to-right shunt

QUESTION 42

A 46-year-old man with dyspnea on exertion is noted to have a systolic ejection murmur along the left sternal border. An echocardiogram is obtained. Figure 1-8 shows Doppler pulsed-wave interrogation of the left ventricular outflow tract, recorded from the apex. Which of the following initial recommendations would be appropriate?
A. Strict fluid restriction
B. Compression stockings
C. Avoid volume depletion

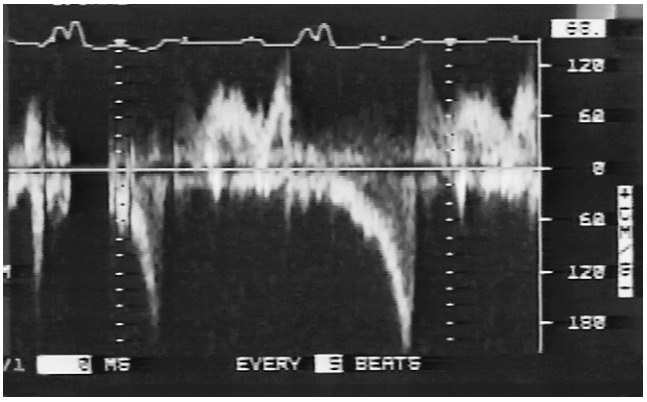

FIGURE 1-8

D. Aortic valve replacement
E. Bed rest

QUESTION 43

Each of the following statements regarding echocardiography in pericardial disease is true EXCEPT:
A. Small pericardial effusions tend to accumulate anterior to the heart
B. Up to 50 mL of pericardial fluid is present in normal individuals
C. In cardiac tamponade, right ventricular diastolic collapse may not occur if pulmonary hypertension is present
D. In the presence of a pericardial effusion, right atrial diastolic indentation is a less specific sign of cardiac tamponade than early diastolic collapse of the right ventricle
E. Chest computed tomography is superior to transthoracic echocardiography as a means to accurately measure pericardial thickness

QUESTION 44

Which of the following statements regarding nuclear imaging in cardiac disease is TRUE?
A. The use of single-photon emission computed tomography (SPECT) with electrocardiographic gating has no impact on the specificity of nuclear testing in women with attenuation artifacts
B. Exercise nuclear stress imaging, rather than pharmacologic stress testing, is the preferred diagnostic modality for patients with left bundle branch block
C. The presence of reversible defects on pharmacologic stress perfusion imaging before noncardiac surgery predicts an increased risk of perioperative cardiac events, but the magnitude of risk is not related to the extent of ischemia
D. Cardiovascular event rates are similar in diabetics compared with nondiabetics for any given myocardial perfusion abnormality
E. Viability of noncontracting myocardium can be accurately evaluated by thallium-201 imaging

ECG SCORING SHEET

General Features
☐ 1. Normal ECG
☐ 2. Borderline normal ECG or normal variant
☐ 3. Incorrect electrode placement
☐ 4. Artifact

P Wave Abnormalities
☐ 5. Right atrial abnormality/enlargement
☐ 6. Left atrial abnormality/enlargement

Atrial Rhythms
☐ 7. Sinus rhythm
☐ 8. Sinus arrhythmia
☐ 9. Sinus bradycardia (<60)
☐ 10. Sinus tachycardia (>100)
☐ 11. Sinus pause or arrest
☐ 12. Sinoatrial exit block
☐ 13. Atrial premature complexes
☐ 14. Atrial parasystole
☐ 15. Atrial tachycardia
☐ 16. Atrial tachycardia, multifocal
☐ 17. Supraventricular tachycardia
☐ 18. Atrial flutter
☐ 19. Atrial fibrillation

AV Junctional Rhythms
☐ 20. AV junctional premature complexes
☐ 21. AV junctional escape complexes
☐ 22. AV junctional rhythm/tachycardia

Ventricular Rhythms
☐ 23. Ventricular premature complex(es)
☐ 24. Ventricular parasystole
☐ 25. Ventricular tachycardia (3 or more consecutive complexes)
☐ 26. Accelerated idioventricular rhythm
☐ 27. Ventricular escape complexes or rhythm
☐ 28. Ventricular fibrillation

AV Conduction
☐ 29. AV block, 1°
☐ 30. AV block, 2°— Mobitz type I (Wenckebach)
☐ 31. AV block, 2°— Mobitz type II
☐ 32. AV block, 2:1
☐ 33. AV block, 3°
☐ 34. Wolff-Parkinson-White pattern
☐ 35. AV dissociation

Abnormalities of QRS Voltage or Axis
☐ 36. Low voltage
☐ 37. Left axis deviation (> –30°)
☐ 38. Right axis deviation (> +100°)
☐ 39. Electrical alternans

Ventricular Hypertrophy
☐ 40. Left ventricular hypertrophy
☐ 41. Right ventricular hypertrophy
☐ 42. Combined ventricular hypertrophy

Intraventricular Conduction
☐ 43. RBBB, complete
☐ 44. RBBB, incomplete
☐ 45. Left anterior fascicular block
☐ 46. Left posterior fascicular block
☐ 47. LBBB, complete
☐ 48. LBBB, incomplete
☐ 49. Intraventricular conduction disturbance, nonspecific type
☐ 50. Functional (rate-related) aberrancy

Q Wave Myocardial Infarction

	AGE RECENT, OR PROBABLY ACUTE	AGE INDETERMINATE, OR PROBABLY OLD
Anterolateral	☐ 51.	☐ 52.
Anterior or anteroseptal	☐ 53.	☐ 54.
Lateral	☐ 55.	☐ 56.
Inferior	☐ 57.	☐ 58.
Posterior	☐ 59.	☐ 60.

ST, T, U Wave Abnormalities
☐ 61. Normal variant, early repolarization
☐ 62. Normal variant, juvenile T waves
☐ 63. Nonspecific ST and/or T wave abnormalities
☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
☐ 65. ST and/or T wave abnormalities suggesting myocardial injury
☐ 66. ST and/or T wave abnormalities suggesting electrolyte disturbances
☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
☐ 68. Prolonged Q-T interval
☐ 69. Prominent U waves

Clinical Disorders
☐ 70. Digitalis effect
☐ 71. Digitalis toxicity
☐ 72. Antiarrhythmic drug effect
☐ 73. Antiarrhythmic drug toxicity
☐ 74. Hyperkalemia
☐ 75. Hypokalemia
☐ 76. Hypercalcemia
☐ 77. Hypocalcemia
☐ 78. Atrial septal defect, secundum
☐ 79. Atrial septal defect, primum
☐ 80. Dextrocardia, mirror image
☐ 81. Chronic lung disease
☐ 82. Acute cor pulmonale including pulmonary embolus
☐ 83. Pericardial effusion
☐ 84. Acute pericarditis
☐ 85. Hypertrophic cardiomyopathy
☐ 86. Central nervous system disorder
☐ 87. Myxedema
☐ 88. Hypothermia
☐ 89. Sick sinus syndrome

Pacemaker Function
☐ 90. Atrial or coronary sinus pacing
☐ 91. Ventricular demand pacemaker (VVI), normally functioning
☐ 92. Dual-chamber pacemaker (DDD), normally functioning
☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

Section I (Chapters 1 to 23)

FUNDAMENTALS OF CARDIOVASCULAR DISEASE; MOLECULAR BIOLOGY AND GENETICS; EVALUATION OF THE PATIENT

ANSWER TO QUESTION 1

D (Braunwald, pp. 168, 177-178)

The normal systolic blood pressure response during exercise is a progressive increase to a peak value between 160 and 200 mm Hg. The higher end of this range is more commonly observed in older patients; in general, black patients tend to have a higher systolic blood pressure response to exercise than white patients. A failure to increase systolic blood pressure to at least 120 mm Hg, or a decline in systolic blood pressure during exercise, is abnormal. Such exertional hypotension occurs in 3% to 9% of patients and is suggestive of underlying multivessel or left main coronary artery disease. Other causes of a decline in systolic blood pressure, or a failure to increase systolic blood pressure with exercise, include cardiomyopathy, vasovagal reactions, ventricular outflow obstruction, hypovolemia, arrhythmias, and prolonged vigorous exercise. Subjects who demonstrate hypotension in the *postexercise* period are much less likely to have advanced underlying coronary artery disease; about 3% of normal subjects younger than 55 years of age demonstrate such a response.

In normal subjects, diastolic blood pressure does not change significantly during exercise. A large change in diastolic blood pressure is uncommon and has not been shown to correlate with underlying coronary artery disease.

The age-related maximum predicted heart rate (MPHR) is estimated from the formula:

$$\text{MPHR} = 220 - \text{age (in years)}$$

which in this patient would be 166 beats/min. The peak heart rate he achieved during the test was 152 beats/min, or 92% of the MPHR (i.e., 152/166 beats/min). An achieved heart rate of ≥85% MPHR is indicative of an adequate diagnostic workload.

Predictors of low prognostic coronary risk in his case include his very good functional capacity (having achieved stage IV of the Bruce protocol) and lack of cardiopulmonary symptoms or ST-segment changes during the test.

REFERENCE

Froelicher VF, Myers J: Exercise and the Heart. 5th ed. Philadelphia, WB Saunders, 2006.

ANSWER TO QUESTION 2

C (Braunwald, pp. 114-117; Fig. 12-8)

S_2 coincides with closure of the aortic and pulmonic valves and marks the onset of diastole at the bedside. Several abnormal heart sounds may follow S_2. The opening snap of mitral stenosis is a high-frequency sound that occurs shortly after S_2. It is generated when the superior bowing of the anterior mitral leaflet during systole rapidly reverses direction toward the left ventricle in early diastole, owing to the high left atrial (LA) pressure. The delay between the aortic component of S_2 and the opening snap corresponds to the left ventricular (LV) isovolumic relaxation time. As mitral stenosis becomes more severe, this phase shortens because of the greater LA pressure, and the interval between S_2 and the opening snap becomes less. Other sounds that occur shortly after S_2 are associated with the rapid filling phase of diastole. These include the third heart sound (S_3), which is a low-frequency sound that is thought to be caused by sudden limitation of LV expansion during brisk early diastolic filling. An S_3 is normal in children and young adults, but the presence of this sound beyond age 40 is abnormal and reflects flow into a dilated ventricle or an increased volume of flow in early diastole, as may occur in mitral regurgitation. A tumor "plop" may be auscultated when an atrial myxoma, attached to the interatrial septum by a long stalk, moves into and obstructs the mitral or tricuspid valve orifice during early diastole. In constrictive pericarditis, a pericardial "knock" may be heard during the rapid filling phase of early diastole as the high-pressure atria rapidly decompress into relatively noncompliant ventricles.

An ejection click is an early systolic sound that represents opening of an abnormal semilunar valve, characteristically a bicuspid aortic valve.

ANSWER TO QUESTION 3

D (Braunwald, pp. 35-36, 178-179; Table 14-3)

In addition to accuracy and reliability, the performance of a diagnostic test depends on its ability to distinguish between the presence and absence of disease. Test

performance depends on its sensitivity and specificity, as well as the prevalence of disease in the population of patients to be studied or the pretest probability of disease in a particular patient. Sensitivity and specificity are characteristics of the diagnostic test that are not altered by disease prevalence or pretest probability. Sensitivity is the percentage of patients with disease who will be correctly identified by the test. Specificity is the percentage of patients without disease who will be correctly identified as disease free by the test. Positive predictive value is the probability that a positive test correctly identifies the presence of disease. Negative predictive value is the probability that a negative test correctly identifies the absence of disease. A perfect diagnostic test has a positive predictive value of 100% (no false-negative results) and a negative predictive value of 100% (no false-positive results).

For a diagnostic test with moderately high sensitivity and specificity, the test will perform best in a population of patients with an intermediate pretest probability of disease (patient D). In patients with a low pretest probability of disease (patients A and B), the positive predictive value of the test is low and there will be a large number of false-positive tests that may prompt unnecessary testing and procedures. In patients with an extremely high pretest probability of disease (patients C and E), the negative predictive value of the test is low and the possibility that a negative result represents a false negative is unacceptably high.

ANSWER TO QUESTION 4

C (Braunwald, pp. 115-118; Table 12-5; Fig. 12-8)

The term *continuous* applies to murmurs that begin in systole and continue without interruption into part or all of diastole. The murmur described here, that of a patent ductus arteriosus, is the classic continuous murmur, peaking in intensity just before or after S_2 then decreasing in intensity during diastole, sometimes disappearing before the subsequent first heart sound. Continuous murmurs may be congenital or acquired and can be caused by (1) an aortopulmonary shunt, such as patent ductus arteriosus; (2) an arteriovenous shunt, including arteriovenous fistulas, coronary artery fistulas, or rupture of an aortic sinus of Valsalva aneurysm into a right heart chamber; (3) constricted arterial vessels (e.g., a femoral arterial atherosclerotic stenosis); (4) turbulence in nonconstricted arteries (e.g., the "mammary souffle," an innocent flow murmur heard during late pregnancy and the puerperium over the lactating breast and augmented by light pressure with the stethoscope); or (5) venous murmurs, such as a cervical venous hum, an often "rough" sounding murmur present in healthy children and young adults. The cervical hum may be accentuated by deforming the internal jugular vein with rotation of the head. It is augmented during pregnancy and in disease states in which there is increased venous flow, such as thyrotoxicosis.

The combined murmurs of aortic stenosis and regurgitation have distinct systolic and diastolic components and do not constitute a continuous murmur.

ANSWER TO QUESTION 5

E (Braunwald, pp. 111-114)

Reduced or unequal arterial pulsations may occur in the arms of patients with atherosclerosis affecting the subclavian arteries, aortic dissection, and unusual arteritides such as Takayasu disease. In supravalvular aortic stenosis there may be selective streaming of the arterial jet toward the innominate artery and right arm, leading to higher pressures in that extremity. This is not the case, however, with subvalvular or valvular aortic stenosis. Valvular aortic stenosis leads to pulsus parvus et tardus, a slowly rising and weak pulse best appreciated by palpation of the carotid arteries. Coarctation of the aorta in adults usually involves the aorta distal to the origin of the left subclavian artery and leads to higher blood pressure in the upper extremities compared with the legs; the arm pulses and pressures are typically equal.

REFERENCE

Lane D, et al: Inter-arm differences in blood pressure: When are they clinically significant? J Hypertens 20:1089, 2002.

ANSWER TO QUESTION 6

C (Braunwald, pp. 252-254, 1655-1665, 1893-1894; Figs. 15-73 and 75-8)

This patient with metastatic breast cancer and a large pericardial effusion has clinical findings consistent with cardiac tamponade.[1] Tamponade physiology results when an accumulation of pericardial effusion causes equilibration of intrapericardial and intracardiac pressures.[2,3] In addition to the presence of an echo-free space surrounding the heart, characteristic echocardiographic and Doppler findings reflect the aberrant pathophysiology of this disorder. Collapse of the right ventricle during early *diastole* occurs because the abnormally elevated pericardial pressure transiently exceeds right ventricular (RV) pressure at that phase of the cardiac cycle. Indentation of the right atrial wall during diastole is a more sensitive marker of increased pericardial pressure but is less specific for tamponade physiology than RV collapse and tends to occur earlier in the course of hemodynamically significant pericardial effusion. Cardiac tamponade is associated with exaggerated ventricular interdependence, a phenomenon manifested at the bedside by pulsus paradoxus. The Doppler correlate of pulsus paradoxus is amplified respirophasic variation of flow across the right- and left-sided cardiac valves. This includes a prominent inspiratory decrease in flow velocity across the mitral and aortic valves (see the transmitral tracing in Figure 1-62), whereas inspiration causes a prominent reciprocal *increase* in flow velocity across the tricuspid and pulmonic valves.

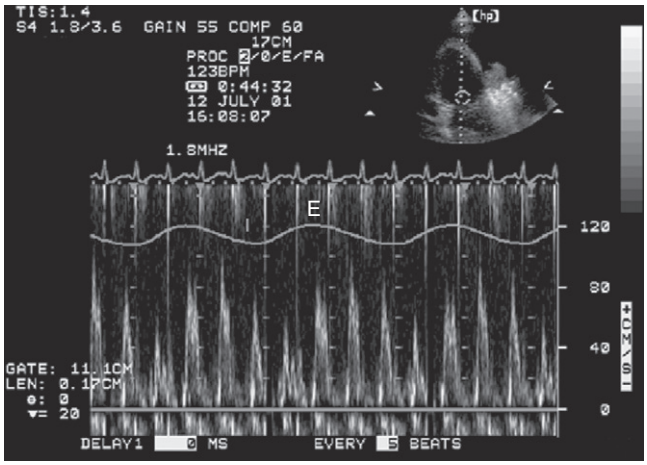

FIGURE 1-62

A marked increase of the E/A ratio of the mitral valve inflow velocity is a finding typical of constrictive pericarditis, not cardiac tamponade.

REFERENCES

1. Chiles C, Woodard PK, Gutierrez FR, Link KM: Metastatic involvement of the heart and pericardium: CT and MRI imaging. RadioGraphics 21:439, 2001.
2. Spodick DH: Acute cardiac tamponade. N Engl J Med 349:684, 2003.
3. Roy CL, Minor MA, Brookheart MA, et al: Does this patient with a pericardial effusion have cardiac tamponade? JAMA 297:1810, 2007.

ANSWER TO QUESTION 7

D (Braunwald, pp. 111-113, 124, 1656)

Pulsus paradoxus is an exaggeration of the normal tendency for arterial pulse strength to fall with inspiration and can be measured easily and accurately at the bedside with a sphygmomanometer. A decline of more than 8 to 10 mm Hg with inspiration is considered abnormal and can be observed in a variety of conditions. Pulsus paradoxus is characteristic of patients with cardiac tamponade, is seen in approximately one third of patients with chronic constrictive pericarditis, and is noted as well in patients with wide intrapleural pressure swings (e.g., bronchial asthma and emphysema), pulmonary embolism, pregnancy, extreme obesity, and hypovolemic shock.

Notably, aortic regurgitation augments left ventricular diastolic pressure and tends to prevent pulsus paradoxus even in the presence of tamponade.

Kussmaul's sign manifests as inappropriate augmentation of the jugular venous pressure during inspiration and implies the presence of constrictive pericarditis, not isolated cardiac tamponade.

ANSWER TO QUESTION 8

D (Braunwald, pp. 144-145; Fig. 13-27; Table 13-6)

Left anterior fascicular block (LAFB) is a common abnormality that can develop in healthy people or in patients with a wide variety of cardiac diseases, including prior anterior myocardial infarction, left ventricular hypertrophy, cardiomyopathies, and degenerative diseases of the conduction system. It results in an alteration of the ventricular activation sequence without prolongation of the QRS complex duration. The characteristic electrocardiographic finding is marked left-axis deviation (–45 to –90 degrees) because of delayed activation of the anterosuperior left ventricular wall. Because inferior and posterior forces are unopposed during early ventricular activation, and anterosuperior forces are unopposed at the termination of activation, the ECG records characteristic rS complexes in the inferior leads and qR complexes in the anterolateral leads. The precordial leads may demonstrate deep S waves in the lateral territory (leads V_4 to V_6), reflecting the late anterosuperior forces.

The development of LAFB can mask the Q waves of a prior inferior myocardial infarction.

REFERENCE

Surawicz B, et al: Recommendations for the standardization and interpretation of the electrocardiogram: III. Intraventricular conduction disturbances. Circulation 119:e235, 2009.

ANSWER TO QUESTION 9

C (Braunwald, pp. 93-94; Tables 10-1 and 10-2)

All drugs prescribed to achieve a particular clinical benefit also have the potential for toxicity. Many factors determine the likelihood of drug toxicity, including the pharmacokinetic and pharmacodynamic properties of the drug and its target, genetic variability in the patient's response to the drug, and drug–drug interactions.

Many medications are metabolized by isoforms of the cytochrome P-450 (CYP) enzyme system, which are expressed in the liver and other tissues. Ketoconazole, erythromycin, and clarithromycin (but not azithromycin) are examples of drugs that inhibit CYP3A4 and 3A5. Because these P-450 isoforms are responsible for metabolism of simvastatin, atorvastatin, and lovastatin, combined therapy with such inhibitors may increase the likelihood of myopathy due to these statins.[1] Pravastatin is not metabolized by the CYP3A system, and thus the risk of myopathy is not increased in the presence of CYP3A inhibitors.

St. John's wort induces activity of CYP3A and results in decreased cyclosporine levels.[2]

Sildenafil, a selective inhibitor of phosphodiesterase type 5 prescribed to treat erectile dysfunction, potentiates the vasodilatory effect of nitrates. Administration of nitrates within 24 hours of sildenafil use has been associated with profound hypotension.

Verapamil inhibits the P-glycoprotein–mediated efflux of digoxin into bile and urine and may contribute to digoxin toxicity.

REFERENCES

1. Thompson PD, Clarkson P, Karas RH: Statin-associated myopathy. JAMA 289:1681, 2003.

2. Zhou S, Gao Y, Jiang W, et al: Interactions of herbs with cytochrome P450. Drug Metab Rev 35:35, 2003.

ANSWER TO QUESTION 10

D (Braunwald, pp. 189-190, 1476; Table 14-5)

Treadmill exercise testing is a safe procedure with an associated mortality of <0.01% and risk of myocardial infarction of 0.04%.[1] The risk of a procedure-related complication is determined by the clinical characteristics of the patient to be studied. Patients with high-grade obstruction of the left ventricular outflow tract, such as those with hypertrophic obstructive cardiomyopathy or critical aortic stenosis, are at an increased risk of a procedural complication owing to the inability of cardiac output to compensate for peripheral vasodilatation during exercise. Patients with unstable angina should not be subjected to the high myocardial oxygen demands of exercise and generally should be referred for coronary angiography instead. Acute myocarditis is associated with an increased risk of exercise-associated sudden death.

Despite the theoretical risk of aortic rupture due to increased wall stress, treadmill exercise testing may be safely performed in patients with an abdominal aortic aneurysm.[2] In contrast, aortic dissection is a contraindication to the stress of exercise testing.

REFERENCES

1. Myers J, Arena R, Franklin B, et al: Recommendations for clinical exercise laboratories: A scientific statement from the American Heart Association. Circulation 119:3144, 2009.
2. Best PJ, Tajik AJ, Gibbons RJ, Pellikka PA: The safety of treadmill exercise stress testing in patients with abdominal aortic aneurysms. Ann Intern Med 129:628, 1998.

ANSWER TO QUESTION 11

B (Braunwald, pp. 302-303, 325, 326; Fig. 17-12)

The images show a fixed defect in the mid and apical anterior wall segments with preserved wall motion and thickening as evident from the end-diastolic and end-systolic frames. These findings are most consistent with imaging artifact due to breast tissue attenuation.

Attenuation artifacts are a common source of error in single-photon emission computed tomography (SPECT). Regional wall motion and wall thickening should be assessed on the electrocardiographic gated SPECT data in any region that shows a fixed perfusion defect. Although a transmural myocardial scar would be associated with reduced wall motion and wall thickening, attenuation artifact is more likely where there is a fixed perfusion defect with normal regional wall motion and wall thickening.

Thallium-201 is a lower-energy radiotracer that results in more attenuation artifacts than technetium-99m imaging, and hence would be an inferior choice in this case.

REFERENCE

Garcia EV, Galt JR, Faber TL, et al: Principles of nuclear cardiology imaging. *In* Dilsizian V, Narula J, Braunwald E (eds): Atlas of Nuclear Cardiology. 3rd ed. Philadelphia, Current Medicine Group, 2009, pp 1-36.

ANSWER TO QUESTION 12

B (Braunwald, pp. 114-117, 121-124)

The normal second heart sound (S_2) consists of two parts, an earlier aortic component and a later pulmonic component. During inspiration, the increased filling of the right ventricle prolongs the ejection phase of the right side of the heart leading to *delayed* closure of the pulmonic valve. This is the predominant factor in normal inspiratory splitting of the S_2. Right bundle branch block delays right ventricular activation and ejection and is therefore associated with *widened* splitting of S_2. Conditions in which left ventricular activation is late, such as left bundle branch block or right ventricular pacing, cause closure of the aortic valve to be delayed. In that setting, the pulmonic valve closure sound actually precedes that of the aortic valve. Then, during inspiration (and prolongation of right ventricular ejection), the delayed closure of the pulmonic valve narrows the timing between the two sounds, a situation known as *paradoxical* splitting.

Fixed splitting of the S_2 is typical of an uncomplicated ostium secundum atrial septal defect. In this condition, closure of the pulmonic valve is delayed because of the increased flow through the right-sided cardiac chambers and an increase in pulmonary vascular capacitance, contributing to a widened split of S_2. On inspiration, augmentation of the systemic venous return is counterbalanced by a reciprocal decrease in the volume of the left-to-right shunt, such that right ventricular filling and the timing of P_2 relative to A_2 do not change, resulting in the fixed splitting.

When valvular stenosis restricts opening of a cardiac valve, the decreased excursion of the leaflets *reduces* the intensity of the closure sound. Thus, in pulmonic stenosis, the pulmonic component of S_2 becomes softer.

ANSWER TO QUESTION 13

D (Braunwald, pp. 172-175, 180)

Electrocardiographic changes during exertion in an asymptomatic patient must be interpreted in light of the pretest likelihood of coronary disease. An exercise-induced ST-segment abnormality is an independent predictor of future cardiac events in men with and without conventional risk factors for coronary disease, although the risk is greatest among the former. However, over 5 years of follow-up, only one in four such patients will actually develop symptoms of cardiac disease, most

commonly angina. Because the patient described in this question is asymptomatic and demonstrates good exercise capacity, there is no need for immediate aggressive intervention such as cardiac catheterization. Appropriate recommendations for this patient with asymptomatic coronary artery disease would include aggressive risk factor modification: smoking cessation, control of hypertension, the addition of aspirin, 81 to 325 mg/d, and treatment of dyslipidemia (e.g., statin therapy to achieve low-density lipoprotein <100 mg/dL).

The distribution of ST-segment depressions during exercise testing correlates poorly with the location of coronary stenoses. Conversely, the location of ST-segment *elevations*, when present, does correlate well with the anatomic lesion causing ischemia.

REFERENCES

Greenland P, Alpert JS, Beller GA, et al: 2010 ACCF/AHA guideline for the assessment of cardiovascular risk in asymptomatic adults: Executive summary. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 122:2748, 2010.

Lauer M, Freolicher ES, Williams M, et al: Exercise testing in asymptomatic adults: A statement from professionals from the American Heart Association Council on Clinical Cardiology, Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention. Circulation 112:771, 2005.

ANSWER TO QUESTION 14

C (Braunwald, pp. 179, 185, 324; Tables 14-3 and 14G-4)

Several clinical situations affect the ST segment and impair the diagnostic utility of the standard exercise ECG. These include the presence of left bundle branch block (LBBB), left ventricular hypertrophy, ventricular preexcitation (Wolff-Parkinson-White syndrome), and digitalis therapy. In these situations, other aspects of the exercise test, such as exercise duration, presence or absence of symptoms, and abnormal blood pressure or heart rate responses, may still provide useful diagnostic information. However, in the presence of these baseline electrocardiographic abnormalities, concurrent imaging (nuclear scintigraphy or echocardiography) is frequently required when more specific diagnostic information is needed. In the case of LBBB, a pharmacologic adenosine stress test with myocardial perfusion imaging helps to avoid artifactual septal perfusion defects compared with exercise protocols.

In patients with a low prior probability of significant coronary artery disease, such as a young woman without significant cardiac risk factors, the development of ST segment depression on exercise testing is more often a nonspecific false-positive result than an indicator of previously undetected coronary artery disease.

REFERENCE

Gibbons RJ, Balady GJ, Bricker JT, et al: ACC/AHA 2002 guideline update for exercise testing: Summary article. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). J Am Coll Cardiol 40:1531, 2002.

ANSWER TO QUESTION 15

D (Braunwald, pp. 383-385, 390, 404, 1937-1940)

Diagnostic cardiac catheterization is a relatively safe procedure, with an overall risk of a major complication of <1%. Mortality rates related to the procedure depend on the population studied and range from 0.08% to 0.75%. The risk of myocardial infarction is approximately 0.05%, and neurologic complications occur in 0.03% to 0.2% of patients. The incidence of acute renal dysfunction in patients with baseline renal insufficiency can be most effectively decreased with intravenous saline administration before and after the procedure. The addition of mannitol or furosemide to saline infusion has been shown to *worsen* renal outcomes in patients receiving an intravenous contrast agent.

Nonionic low osmolar contrast agents reduce the likelihood of adverse hemodynamic and electrophysiologic reactions during angiography. They also reduce the incidence of contrast-induced nephropathy in patients with baseline renal insufficiency, with or without diabetes. Of note, in patients with normal renal function, there is no advantage of low osmolar agents over ionic agents in the prevention of nephrotoxicity.

Cardiac catheters are available in many sizes, shapes, and lengths. The outer diameter of the catheter is specified using French units (F), where 1 F is equal to 0.33 mm.

Patients with tilting-disc prosthetic aortic valves should not undergo retrograde left-sided heart catheterization because of the risk of catheter entrapment, occlusion of the valve, or possible dislodgment of the disc with embolization.

REFERENCE

Smith SC, Feldman TE, Hirshfeld JW, et al: ACC/AHA/SCAI 2005 guideline update for percutaneous coronary intervention: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (ACC/AHA/SCAI Writing Committee to update the 2001 Guidelines for Percutaneous Coronary Intervention). Circulation 113:e166, 2006.

ANSWER TO QUESTION 16

E (Braunwald, pp. 149-156, 1093, 1101, 1153-1156; Figs. 13-35 and 54-4)

Proximal occlusion of a dominant right coronary artery (RCA) leads to infarction of the left ventricular (LV) inferior wall but often also involves the posterior wall, the right ventricle, and portions of the conduction system, which are all supplied by branches of the RCA. ST-segment elevation in leads II, III, and aVF is the sine qua non of transmural infarction of the inferior wall. If the posterior wall is involved, ST-segment depression is usually evident in V_1 and V_2, reflecting a current of injury on the side of the heart opposite those leads (if unipolar leads were placed on the patient's back overlying the posterior wall, ST-segment elevation would be observed instead). Very proximal occlusion of the RCA is often accompanied by right ventricular (RV) infarction because the RV

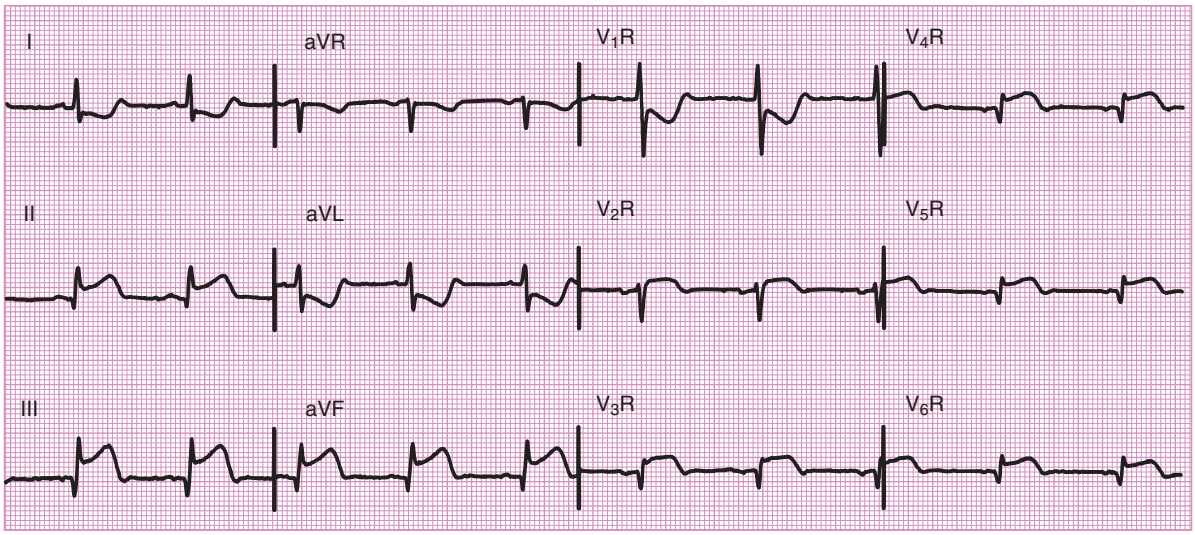

FIGURE 1-63

arterial branch arises near the origin of that vessel. If RV infarction is present, right-sided precordial electrocardiographic leads, particularly V_4R, often demonstrate ST-segment elevation as well (Fig. 1-63). Sinus bradycardia is common in the setting of acute myocardial infarction, especially in inferior or posterior infarction. This arrhythmia, particularly when accompanied by hypotension, may arise from stimulation of cardiac vagal afferent fibers, which are prominent in the inferoposterior left ventricle. Sinus bradycardia may also be a vasovagal response to the severe chest pain in acute myocardial infarction or reflect ischemia of the sinoatrial artery, which arises from the RCA in 60% of the population. Another potential cause of bradycardia in this patient is the development of atrioventricular (AV) block because of either vagal stimulation or ischemia of the AV node. The AV node is supplied by the AV nodal artery, which arises from the RCA 85% of the time.

PR-segment deviations are common in patients with acute pericarditis, not acute myocardial infarction.

ANSWER TO QUESTION 17

B (Braunwald, pp. 230-240)

One of the most clinically important applications of Doppler technology is the estimation of pressure gradients across stenotic orifices or septal defects in the cardiovascular system. The Bernoulli equation relates the pressure difference across a narrowed area to the convective acceleration, flow acceleration, and viscous friction. By modifying the Bernoulli equation, a more clinically useful simplified formula is derived. The simplified Bernoulli equation states that the pressure difference across a flow-limiting orifice = $4V^2$, where V is the peak velocity distal to the obstruction. In this question, we are given the information that the patient's systolic blood pressure is 144 mm Hg, which, in the absence of left ventricular (LV) outflow obstruction, is also the LV systolic pressure. If the ventricular septal defect flow is 5.1 m/sec, then using the modified Bernoulli equation, the pressure gradient across the ventricular septal defect is $4 \times (5.1)^2 = 104$ mm Hg. The right ventricular systolic pressure can then be simply calculated by subtracting that gradient from the LV systolic pressure: 144 – 104, or 40 mm Hg.

If the patient's blood pressure had not been given, it would still be possible to estimate the LV systolic pressure using the mitral regurgitation velocity. If the mitral regurgitation velocity is 5.8 m/sec, then we know, using the modified Bernoulli equation, that the pressure difference across the mitral valve in systole is 135 mm Hg. An estimate of the left atrial pressure is then made using two-dimensional and Doppler parameters, and this value is added to the transmitral systolic gradient to derive an estimate of LV systolic pressure. Thus, if the estimated left atrial pressure were 10 mm Hg, then the LV systolic pressure would be 145 mm Hg.

REFERENCE

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ANSWER TO QUESTION 18

C (Braunwald, pp. 145-146, 746-748)

Left bundle branch block (LBBB) most commonly develops in individuals with underlying structural heart disease. The presence of this conduction abnormality portends a reduced long-term survival, with 10-year mortality rates as high as 50%. In patients with known coronary atherosclerosis, the development of LBBB implies more extensive disease and is often associated with left ventricular (LV) dysfunction. If left-axis deviation accompanies LBBB, more severe clinical disease is likely. The

delayed LV activation in LBBB leads to paradoxical splitting of the second heart sound because pulmonic valve closure abnormally precedes aortic valve closure (see Answer to Question 12).

In LBBB, repolarization of the left ventricle is typically altered, such that the ST segment and T waves are discordant with (i.e., directed opposite to) the QRS complex.

The abnormal ventricular activation in LBBB results in a dyssynchronous pattern of contraction that may impair stroke volume. The duration of the QRS complex in LBBB inversely correlates with the LV ejection fraction.[1] Cardiac resynchronization therapy with biventricular pacing was developed to improve LV function in patients with cardiomyopathy and LBBB and has been shown to improve outcomes in patients with advanced heart failure.[2]

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ANSWER TO QUESTION 19

A (Braunwald, pp. 159-161)

Many electrolyte disturbances result in characteristic electrocardiographic manifestations. Decreased extracellular calcium in hypocalcemia prolongs phase 2 of the action potential (AP), thereby lengthening the AP duration and the QT interval. The long QT interval in hypocalcemia is characteristically flat (i.e., isoelectric), without the concave configuration of many drug-induced prolonged QT states. *Increased* extracellular calcium shortens the ventricular action potential and the duration of the QT interval. The appearance of a J wave (also known as an Osborn wave—see arrow in Figure 1-64), an extra deflection at the junction of the QRS complex, and the ST segment typically observed in cases of severe hypothermia has been reported with severe *hyper*calcemia.[1]

Hyperkalemia causes a specific sequence of electrocardiographic changes depending on its severity. The earliest manifestation is narrow, peaked T waves. The QT interval is usually decreased at that time because of shortened action potential duration. Progressive hyperkalemia reduces the resting membrane potentials in both the atria and ventricles, thus inactivating sodium channels. The net result is to slow depolarization and reduce action potential conduction velocity. The ECG shows widening of the QRS complex with a decrease in P wave amplitude. PR-segment prolongation may also occur. Very marked hyperkalemia leads to slow, undulating ventricular flutter (a "sine wave" appearance) followed by eventual asystole. Hypokalemia, in contrast, manifests primarily as ST-segment depressions with flattened T waves and U wave prominence. Because of prolongation of the QT interval there is a propensity for polymorphic ventricular tachycardia (torsades de pointes).

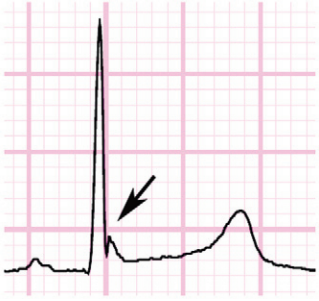

FIGURE 1-64

The effects of magnesium on the surface ECG are not as well characterized. Magnesium deficiency may predispose to prolongation of the QT interval, primarily as a result of a prolonged U wave (QU interval), and torsades de pointes.

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ANSWER TO QUESTION 20

E (Braunwald, pp. 178-179, 185-186, 193-194; Table 14-3; see also Answer to Question 14)

Meta-analyses show that the sensitivity and specificity of the exercise ECG for the detection of coronary artery disease (CAD) are 68% and 77%, respectively. The sensitivity for diagnosis is impaired by resting ST-segment depression or other repolarization abnormalities, including left ventricular hypertrophy, digitalis therapy (even if digitalis effect is not evident on the resting ECG), left bundle branch block, and preexcitation syndromes. In addition, severe hypertension, hyperventilation, and hypokalemia may distort the ST segment, leading to false-positive electrocardiographic interpretations. One of the main diagnostic values of the standard exercise test is to exclude CAD in patients with chest pain with a low pretest likelihood of disease based on age and gender who have a normal resting ECG. If such a patient achieves the maximum heart rate without ST-segment deviations and a normal blood pressure response, it is very unlikely that CAD is the cause of chest pain. For most other populations, the exercise ECG is limited by the suboptimal sensitivity and specificity. Both myocardial perfusion imaging and stress echocardiography increase the sensitivity and specificity of exercise testing in patients with resting electrocardiographic abnormalities. In particular, the negative predictive value of both of these techniques is excellent.

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ANSWER TO QUESTION 21

E (Braunwald, p. 148; Fig. 13-28)

The underlying rhythm is atrial fibrillation. The Ashman phenomenon (as exemplified by the fifth QRS complex in the tracing) represents conduction aberrancy caused by changes in the preceding cycle length. Because the duration of the refractory period is a function of the immediate preceding cycle length, the longer the preceding cycle, the longer the ensuing refractory period and the more likely that the next impulse will be conducted with delay. Normally the refractory periods of the conduction system components are right bundle branch > left bundle branch = atrioventricular node > > His bundle. Therefore, it would be unusual for the bundle of His to be the site of conduction delay and, as is commonly the case, the aberrant beat on this tracing demonstrates right bundle branch block morphology in lead V_1.

ANSWER TO QUESTION 22

C (Braunwald, pp. 115-117; Fig. 12-10)

Innocent (normal) systolic murmurs are related to intracardiac flow rates and are usually loudest in midsystole. They may be caused by normal vibrations of the pulmonary leaflets or exaggeration of normal ejection vibrations within the pulmonary artery or be associated with sclerosis at the base of the aortic valve leaflets in the absence of significant valvular stenosis. The normal mammary souffle, heard over the breasts of women in late pregnancy or during lactation, may be midsystolic in timing or continuous. Careful auscultation usually reveals a time delay between S_1 and onset of this murmur, which is the transit time of flow from the proximal aorta to the mammary arteries.

ANSWER TO QUESTION 23

D (Braunwald, pp. 110-111, 124; Figs. 12-2 and 12-3)

A great deal of information about right-sided heart hemodynamics can be ascertained from the jugular venous pressure waveforms. The *a* wave results from venous distention due to right atrial contraction; the *x* descent reflects atrial relaxation and downward descent of the base of the right atrium during right ventricular (RV) systole. The *c* wave is an inconstant positive deflection in the jugular venous pulse that interrupts the *x* descent and corresponds to ventricular contraction. The *v* wave results from right atrial filling during ventricular systole when the tricuspid valve is closed, and the *y* descent occurs after the tricuspid valve opens and right atrial pressure declines. It is easier for an observer to see the *x* and *y* descents than the positive pressure waves (*a*, *c*, and *v* waves) in the neck because the former produce larger excursions. An elevated jugular venous pressure reflects increased right atrial pressure. During inspiration, the jugular venous pressure normally declines as intrathoracic pressure becomes more negative. Kussmaul's sign is a paradoxical rise in the height of the venous pressure during inspiration. It reflects an inability of the right-sided chambers to accept additional volume, typical of constrictive pericarditis, but may also be observed in patients with right-sided heart failure, severe cor pulmonale, or tricuspid stenosis.

The *a* wave becomes more prominent in conditions that increase the resistance to right atrial contraction, such as RV hypertrophy, pulmonary hypertension, or tricuspid stenosis. Amplified "cannon" *a* waves are evident during any situation that causes atrioventricular dissociation, because the right atrium contracts against a closed tricuspid valve at least intermittently.

In constrictive pericarditis, the *y* descent is rapid and deep because the earliest phase of diastolic RV filling is unimpeded. In contrast, in cardiac tamponade, the *y* descent is blunted and it is the *x* descent that is most prominent.

ANSWER TO QUESTION 24

D (Braunwald, pp. 396-398; Figs. 20-11 and 20-12)

There is no completely accurate method for measuring cardiac output in the cardiac catheterization laboratory. Two commonly used methods are the thermodilution and the Fick techniques. The former involves injection of a bolus of fluid (i.e., saline or dextrose) into the proximal port of a right-sided balloon flotation (e.g., Swan-Ganz) catheter, after which alterations in temperature are measured at the distal end of the catheter. The change in the temperature over time is then plotted to derive the cardiac output, which is *inversely* related to the area under the thermodilution curve (Fig. 1-65). In low cardiac output states there is a larger area under the curve owing to the longer time required for the temperature curve to return to its baseline. However, this technique tends to *overestimate* cardiac output in the setting of low output states, because the dissipation of the cooler temperature to the surrounding cardiac structures results in

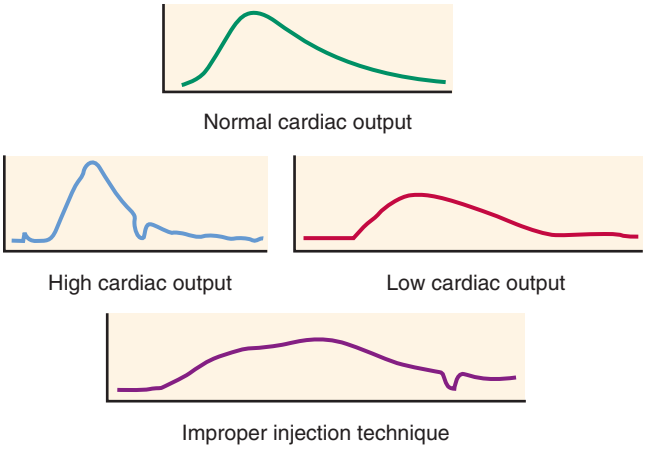

FIGURE 1-65

a reduction in the total area under the curve. In addition, the back and forth flow across the tricuspid valve in patients with severe tricuspid regurgitation also creates significant error in measurement, producing a falsely low cardiac output by this technique.

The Fick technique is based on the principle that cardiac output is equal to the oxygen consumption divided by the difference in oxygen content between arterial and mixed venous blood. That is:

$$\text{Cardiac output} = \frac{O_2 \text{ Consumption}}{\text{A-}VO_2 \text{ Difference}}$$

The Fick technique is more accurate than thermodilution in patients with low cardiac outputs; however, its main limitation is in measuring true oxygen consumption in a steady state. Many laboratories use an "assumed" oxygen consumption by considering the patient's age, gender, and body surface area. Inaccuracy in the oxygen consumption measurement can result in substantial variability in reported cardiac outputs.

Systemic vascular resistance (SVR) is derived by dividing the difference between the mean aortic and right atrial (RA) pressures by the systemic cardiac output (and then multiplying by a constant to convert to the commonly used units of dyne•sec•cm^{-5}):

$$\text{SVR} = 80 \times \frac{\text{Mean Aortic Pressure} - \text{Mean RA Pressure}}{\text{Systemic cardiac output}}$$

The pulmonary vascular resistance (PVR) is obtained by dividing the difference between the mean pulmonary artery (PA) and left atrial (LA) pressures by the pulmonic cardiac output (then multiplying by the same constant). The pulmonary capillary wedge (PCW) pressure is commonly used as a surrogate for left atrial pressure:

$$\text{PVR} = 80 \times \frac{\text{Mean PA Pressure} - \text{Mean LA (or PCW) Pressure}}{\text{Pulmonic cardiac output}}$$

In the absence of intracardiac shunts, the systemic and pulmonic cardiac outputs should be the same.

ANSWER TO QUESTION 25

C (Braunwald, pp. 220-223; Figs. 15-26 and 15-27)

Pulsed Doppler interrogation of mitral valve inflow is useful in identifying disorders of left ventricular (LV) diastolic function. In normal adults, the early (E wave) velocity exceeds the late (A wave) velocity such that the normal E/A ratio is >1.2. The figure accompanying this question illustrates an abnormal pattern of mitral inflow, with an E/A ratio >1.0. Although this pattern may be present in patients who have documented abnormalities of LV relaxation, it can also occur as a result of normal aging.[1] In fact, most people older than age 70 have an E/A ratio <1.0. In addition, the pattern of diastolic mitral inflow is load dependent. If a patient with the illustrated pattern of impaired relaxation were administered intravenous volume, the mitral inflow pattern could change to the "pseudonormalized" form, with an E/A ratio >1.0, without any alteration in the intrinsic relaxation properties of the ventricle. Caution should thus be used when inferring a diastolic or relaxation abnormality from the mitral inflow pattern. Although none of the available echocardiographic indices of diastolic function is infallible, and even stepwise approaches using the mitral inflow pattern, Doppler tissue imaging, and assessment of pulmonary venous inflow can result in internally inconsistent results,[2] echocardiographic measures of diastolic function nonetheless provide clinically important diagnostic and prognostic information.[3]

Constrictive pericarditis and restrictive cardiomyopathy are clinical situations in which the bulk of ventricular filling occurs in early diastole. In these conditions there is a high E/A ratio, often >2.0. Hyperthyroidism and hemorrhage each result in hyperdynamic states in which diastolic relaxation is enhanced, such that reversal of the E/A ratio would be unusual.

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ANSWER TO QUESTION 26

D (Braunwald, pp. 232-240, 1490-1491; Figs. 15-38, 15-39, 15-47, and 66-19)

The figure accompanying this question displays continuous Doppler interrogation of transmitral valvular flow obtained from the apical four-chamber transducer position. During diastole, the deceleration of transmitral inflow is prolonged, consistent with a persistent pressure gradient between the left atrium and left ventricle (i.e., mitral stenosis). In systole, a faint signal of mitral regurgitation (MR) is seen as a downward velocity spectrum. To determine the extent of MR noninvasively, simultaneous two-dimensional echocardiography and color Doppler interrogation would be required. However, the presented Doppler spectrum of mitral inflow is sufficient to determine the severity of mitral stenosis, including the transmitral pressure gradient and the valve area, as described below. The mitral valve area can be calculated noninvasively by three distinct noninvasive echo-Doppler methods. First, if an adequate two-dimensional short-axis image can be obtained in diastole, the valve orifice can be traced and the valve area measured directly using planimetry (see Braunwald, Fig. 66-19). The second method (which can be applied to the provided figure) utilizes the Doppler pressure half-time (PHT), which calculates the time in milliseconds required for the diastolic transmitral pressure gradient to decline to one half

of its peak value (see Braunwald, Figs. 15-39 and 15-47). Because of the relationship between velocity and pressure, this requires determining the time it takes for the peak diastolic velocity to fall to the peak velocity divided by the square root of 2. Most modern echocardiographic machines calculate the PHT automatically once the Doppler profile is traced on the screen, using the equation:

$$\text{Mitral valve area} = 220 \div \text{PHT}$$

This relationship becomes less accurate when there is marked MR or aortic regurgitation, which interferes with the measured pressure gradient across the mitral valve. The third method to calculate the mitral valve area utilizes the continuity equation, which is based on the principle that the volume rate of flow through the heart is constant (see Braunwald, Fig. 15-38). The cause of mitral stenosis in the vast majority of adults is rheumatic heart disease. Occasionally, other causes can be identified, such as congenital abnormalities of the valve or heavy senile calcification that restricts transvalvular flow. The transvalvular Doppler pattern cannot distinguish the etiology of mitral stenosis, but two-dimensional echocardiographic imaging is usually diagnostic in this regard.

ANSWER TO QUESTION 27

B (Braunwald, p. 235)

The general calculation of the pressure gradient across a cardiac value is determined by the equation

$$\Delta \text{ Pressure} = 4 \times \text{velocity}^2$$

In this instance, the pressure gradient is 64 mm Hg (4×4^2). With a right atrial pressure of 9 mm Hg and a pressure gradient of 64 mm Hg, the right ventricular pressure equals 73 mm Hg (64 + 9). Pressure gradients across other cardiac valves and across a ventricular septal defect can be calculated in a similar manner.

ANSWER TO QUESTION 28

C (Braunwald, pp. 168-170, 171, 177-178, 187)

With normal aerobic exercise, the heart rate and systolic blood pressure both rise. Diastolic blood pressure, however, typically does not change significantly. During *isometric* exercise, both systolic and diastolic blood pressures may increase. In normal individuals, the cardiac output rises fourfold to sixfold above basal levels during maximum exercise. The physiologic response to physical activity often becomes attenuated as an individual ages. The heart rate response to exercise is blunted in the elderly, and the predicted maximum heart rate decreases with age (estimated by 220 – age in years). This is due in part to decreased beta-adrenergic responsiveness in older individuals. The average stroke volume is preserved in normal older adults, and the observed decline in maximum cardiac output is due primarily to the blunted heart rate response. Maximal aerobic capacity ($\dot{V}_{O_2}max$) declines 8% to 10% per decade in sedentary individuals, such that there is a 50% fall between ages 30 and 80. Thus, the exercise protocol chosen to test elderly individuals should take into account predicted limitations in exertional capacity.

ANSWER TO QUESTION 29

D (Braunwald, pp. 115-118, 121-124, 1473-1474, 1490-1493, 1499-1504, 1520-1512; Table 12-6)

The intensity of a heart murmur is related to the pressure gradient and rate of flow across the responsible orifice. Physiologic changes or bedside maneuvers that alter the driving pressure gradient or the rate of flow lead to audible changes in murmur intensity. In acute mitral regurgitation (MR), flow is directed backward from the left ventricle into a relatively noncompliant left atrium, leading to a rapid increase in left atrial pressure during systole. Because this abolishes the pressure gradient between the left ventricle and left atrium in late systole, the murmur of acute MR is often present only in early systole. Similarly, in acute aortic regurgitation, the left ventricular (LV) diastolic pressure rises rapidly, leading to cessation of the diastolic murmur in mid to late diastole, as LV and aortic pressures equalize. In patients with mitral valve prolapse, the auscultatory findings vary prominently with physiologic alterations. The valve physically prolapses into the left atrium, and the associated click/murmur commences when the reduction of LV volume during contraction reaches the point at which the mitral leaflets fail to coapt. Maneuvers that decrease LV volume, such as standing from a squatting position, cause the valve prolapse, the click, and the murmur to all occur earlier in systole. In mitral stenosis, the diastolic rumbling murmur increases with any maneuver that augments transvalvular flow and decreases in situations that reduce transmitral flow, such as the strain phase of the Valsalva maneuver.

The systolic murmurs of aortic valvular stenosis and MR are sometimes difficult to distinguish. However, the intensity of aortic stenosis varies from beat to beat when the duration of diastole is not constant, as in atrial fibrillation or after a premature contraction. The murmur of MR is not affected in this manner, because the changes in driving pressure between the left ventricle and the left atrium are smaller.

ANSWER TO QUESTION 30

C (Braunwald, pp. 373, 1319-1335)

This computed tomogram demonstrates a type A aortic dissection involving the ascending (large arrow, A) and descending (small arrows, A) thoracic aorta. In addition, the dissection extends into the aortic arch vessels (large arrows, B), the left common carotid artery (small arrow, B), and the innominate artery (arrowhead, B). Dissection membranes are clearly visualized in the involved

segments. Acute aortic dissection is a rare but often lethal illness. Survival depends on prompt clinical recognition and definitive imaging of the aorta. Stanford type A aortic dissections involve the ascending aorta. Type B aortic dissections do not involve the ascending aorta. The most common symptom of acute aortic dissection is chest or back pain, reported in up to 96% of patients. The pain is typically sudden in onset and severe, often with a "ripping" or "tearing" quality. The patient discussed in this question may have also presented with pulse deficits or neurologic symptoms, given the branch artery involvement demonstrated on the computed tomogram. Consensus guidelines regarding the diagnosis and management of thoracic aortic dissections were published in 2010.[1] Computed tomography (CT) and magnetic resonance imaging (MRI) have outstanding sensitivity for the diagnosis of aortic dissection, on the order of 96% to 100%. Transesophageal echocardiography (TEE) is unnecessary in this case, but it is also an excellent imaging modality for the diagnosis of aortic dissection (sensitivity of ~98% and specificity of 94% to 97%), with the caveat that visualization of the distal ascending aorta and proximal aortic arch may be limited by interference by the trachea and bronchus. Conversely, the sensitivity of standard transthoracic echocardiography for aortic dissection is relatively poor (59% to 85%). Aortography is rarely necessary and has inferior diagnostic capabilities for aortic dissection compared with CT and MRI. Because of its rapid availability in most emergency departments, CT is the initial diagnostic test of choice for suggested aortic dissection in many centers.

All patients with acute aortic dissection should receive immediate parenteral therapy for hypertension, such as intravenous beta blockers to provide rate control, after which vasodilators can be added as needed to control blood pressure. Patients with acute type A aortic dissection should be referred for emergent surgery. Surgery is also the treatment of choice for patients with acute type B aortic dissection complicated by vital organ compromise, rupture or impending aortic rupture, retrograde extension into the ascending aorta, or Marfan syndrome. Patients with uncomplicated acute type B aortic dissections can typically be managed initially with parenteral agents for blood pressure control, monitoring in the coronary care unit, and serial imaging of the aorta to exclude retrograde extension or other indications for surgery and then converted to therapy with oral antihypertensive agents.

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ANSWER TO QUESTION 31

D (Braunwald, pp. 172-177)

ST-segment displacement is the primary means by which ischemia is detected during exercise testing. ST-segment depression is the most common form of abnormal response, whereas ST-segment elevation in a lead without pathologic Q waves is found only in about 1% of patients with obstructive coronary disease. J point depressions with upsloping ST segments are a normal finding during exercise. Patients with ischemic heart disease usually display true ST-segment depression with a horizontal or downsloping configuration. The correct isoelectric reference point for the ST segment is the TP segment. However, because this segment is shortened during exercise, the PQ junction is typically chosen as the reference point instead. In normal individuals who have an early repolarization pattern on the resting ECG, the chronically elevated J point usually returns to baseline during exercise. In this setting, any ST-segment deviations during exercise should be referenced to the PQ junction, not to the original J point position. Ischemic ST-segment depressions may develop only during exercise or may occur during exercise and persist into recovery. In 10% of patients, ischemic changes are observed only during the recovery phase. The onset of ST-segment changes during recovery occurs more commonly in asymptomatic individuals compared with those with symptomatic coronary artery disease. An abnormal ST-segment response is considered to be 0.10 mV (1 mm) or greater of J point depression, with a flat or downsloping ST segment that remains depressed to 0.10 mV or greater at 80 milliseconds after the J point.

Although ST-segment *elevation* is helpful to localize ischemia to particular coronary territories, the location of ST-segment *depression* during exercise does not accurately predict the responsible coronary anatomy.

ANSWER TO QUESTION 32

C (Braunwald, pp. 135, 141-145, 156, 162-163; Tables 13-5 and 13-6)

This ECG demonstrates extreme right-axis deviation of the QRS complex and an abnormally rightward P wave axis (the P waves are inverted in leads I and aVL). Although this pattern might suggest dextrocardia with situs inversus, the normal progression of the R waves in the precordial leads is not consistent with that diagnosis (in dextrocardia R wave progression would be reversed). Rather, the tracing is most consistent with right and left arm lead reversal, a common error of lead placement. In this situation, the recordings from leads aVL and aVR are interchanged and the complexes in lead I are the mirror image of what would be expected in that lead had the limb leads been placed correctly. Figure 1-66 here shows the ECG from the same patient with the leads placed correctly.

ANSWER TO QUESTION 33

D (Braunwald, pp. 168-172, 177-178)

An understanding of the differences between stress testing protocols is required to choose the appropriate study for a specific patient. Six to 12 minutes of progressive exercise, leading to a level of maximal oxygen consumption, provides the greatest diagnostic and prognostic information. An optimal exercise study is characterized by an appropriate rise in both heart rate and systolic

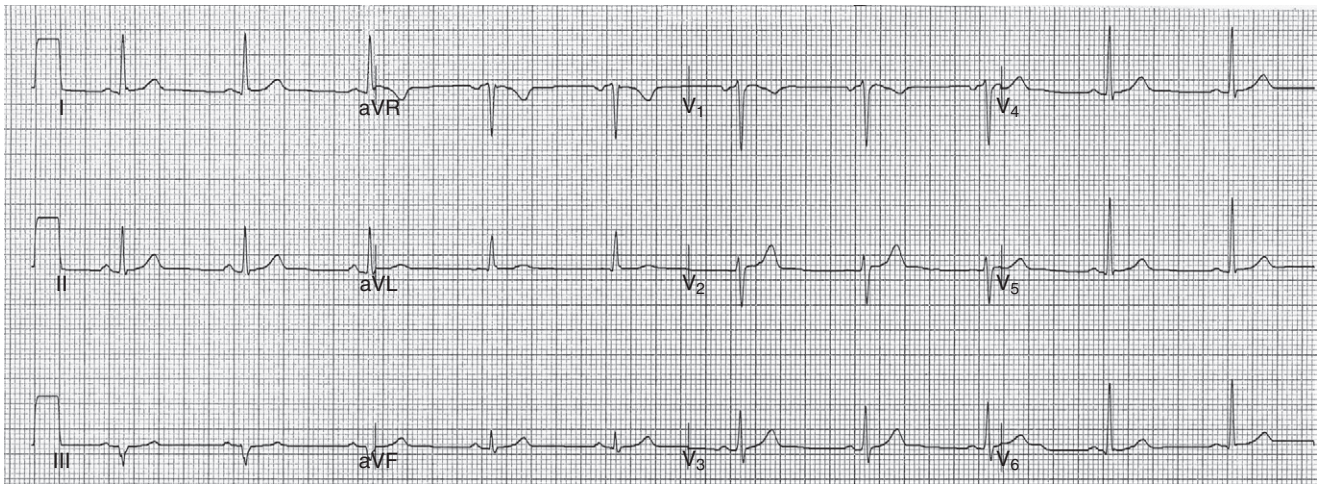

FIGURE 1-66

blood pressure. The diastolic blood pressure may fall, rise, or stay the same, depending on the protocol used and the workload achieved. When systolic blood pressure falls during exercise, it is often indicative of severe underlying coronary artery disease (typically three-vessel or left main disease) or severe left ventricular (LV) contractile dysfunction. Other potential causes include LV outflow obstruction (e.g., advanced aortic stenosis or hypertrophic obstructive cardiomyopathy) and hypovolemia.

Treadmill protocols are the most commonly used form of stress testing and are characterized by achievement of high maximum heart rates and oxygen consumption. Bicycle protocols are sometimes better tolerated by deconditioned patients because of the ramped nature of the test and achieve maximum heart rates similar to, but maximal oxygen consumption less than, treadmill tests. Arm crank ergometry can be useful for patients who cannot perform leg exercise, such as patients with severe peripheral arterial disease. Arm protocols typically produce a higher heart rate and blood pressure response for a given workload than leg exercise protocols; however, the maximum heart rate achieved during arm ergometry testing is typically only about 70% of that achieved during treadmill or bicycle tests. Maximal oxygen consumption and minute ventilation are also lower for arm cycling than for leg exercise. The standard treadmill Bruce protocol is the most commonly used, and a large diagnostic and prognostic database has been accumulated with this regimen. The primary limitation of the Bruce protocol is the large increase in oxygen consumption from one stage to the next, which limits the utility of this protocol in elderly, deconditioned, or ill individuals. In such patients a more gradual regimen, such as the Naughton or Weber protocol, using 1- to 2-minute stages with 1-MET increases per stage, or a ramped bicycle protocol with small increases in workload each minute, is typically better tolerated.

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ANSWER TO QUESTION 34

D (Braunwald, pp. 107-108, 121-124, 1420, 1426-1428, 1472-1473, 1494-1495)

The development of new exertional dyspnea in a patient with cardiac disease may herald progression or a change in the clinical syndrome. Each of the patients in this question has a known, previously asymptomatic cardiac lesion and has recently developed shortness of breath with physical activities. A congenitally bicuspid aortic valve often becomes progressively stenotic with age, and symptoms associated with aortic stenosis (angina, syncope, heart failure) frequently develop in mid to late adulthood. A patient with a history of myocardial infarction who presents with new dyspnea without recurrent angina is likely to have developed left ventricular (LV) dysfunction due to ventricular remodeling. Other potential contributing factors include dyspnea as an anginal equivalent or a superimposed arrhythmia. New irregular palpitations in a patient with a history of rheumatic heart disease may indicate superimposed atrial fibrillation. This is a poorly tolerated complication in patients with mitral stenosis because of the abbreviated LV diastolic filling period during rapid heart rates.

Most young adults with an uncomplicated ostium secundum atrial septal defect (ASD) have normal exercise tolerance. Previously asymptomatic ostium secundum ASDs, in the absence of pulmonary hypertension, are typically well tolerated during pregnancy. Although problems may arise because of paradoxical embolism in the setting of lower extremity venous thrombosis, symptoms of ventricular dysfunction or reversed shunting across the ASD are rare. Exertional dyspnea in the third trimester of pregnancy in such patients is more likely the result of impaired diaphragmatic excursion from the increasing size of the uterus.

The most common causes of morbidity and mortality in trisomy 21 (Down syndrome) are congenital heart defects, which are present in 40% to 50% of patients. The most characteristic abnormalities are endocardial cushion defects, such as ostium primum ASD and "cleft" mitral valve with mitral regurgitation. Approximately one third of the congenital heart lesions are complex defects that are detected early, but simpler cardiac anomalies may remain unnoticed into adulthood. The new onset of exertional dyspnea in a patient with trisomy 21 warrants a full cardiac evaluation, including careful echocardiographic study.

ANSWER TO QUESTION 35

B (Braunwald, pp. 149-159; Fig. 13-35)

Early recognition of myocardial infarction (MI) is critical to take full advantage of emergent percutaneous revascularization or fibrinolytic therapy. The earliest electrocardiographic finding in acute ST-elevation MI is ST-segment elevation and hyperacute (tall, positive) T waves overlying the affected region of myocardium. Reciprocal ST-segment depressions are often noted in leads overlying the opposite cardiac territories. In the absence of reperfusion therapy, T wave inversions become evident in the leads overlying the region of infarction over a matter of hours, accompanied by Q wave development. In the case of an anterior Q wave MI, the early ST- segment deflections become apparent in the anterior precordial leads, whereas ST-segment depressions are often present in the inferior leads. A normal ECG at the time of presentation does not exclude an acute infarction; however, it is unlikely that the ECG would remain normal over hours in the presence of ongoing transmural ischemia. Acute infarction affecting portions of the conduction system may produce a new bundle branch block. The presence of a new right bundle branch block (RBBB) does not obscure the diagnosis of an acute MI, because the ST-segment elevations in the precordium should remain interpretable. However, the ST-segment and T wave changes that accompany a new left bundle branch block (LBBB) typically mask the ST-segment and T wave changes of acute infarction, making the diagnosis more difficult. In a patient with a convincing history of prolonged ischemic chest discomfort, a new LBBB is an acceptable criterion of acute infarction. Interestingly, appearance of a new RBBB during an acute anterior MI has been shown to portend a worse prognosis than a new LBBB in fibrinolytic[1,2] and interventional studies.[3]

A shortened QT interval is typical of hypercalcemia, not acute MI.

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ANSWER TO QUESTION 36

C (Braunwald, pp. 294, 305-306, 326-330)

Myocardial perfusion imaging with either thallium-201– or technetium-99m–labeled compounds (e.g., technetium-99m sestamibi) is useful for the detection of myocardial ischemia and infarction (MI), to characterize infarct size (which predicts future ventricular remodeling), and to determine the effectiveness of acute revascularization. Nuclear imaging is also useful for early risk stratification after an acute MI. The size of the resting myocardial perfusion defect correlates with prognosis: the larger the defect, the worse the outcome.

Certain resting image patterns seen after acute MI, such as increased lung uptake of thallium-201, have been associated with an unfavorable prognosis because they are indicative of impaired left ventricular function. Lastly, pharmacologic stress testing with nuclear imaging after an acute MI has been shown to be safe and to predict in-hospital and late cardiac complications better than submaximal exercise stress imaging. Gated single-photon emission computed tomography (SPECT) myocardial perfusion imaging allows for determination of left ventricular function, which adds additional prognostic information in the management of the postinfarction patient.

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ANSWER TO QUESTION 37

C (Braunwald, p. 175; see also Answer to Question 31)

The development of ST-segment elevation during exercise testing is predictive of the presence of transmural ischemia owing to vasospasm or a high-grade coronary narrowing. It is quite uncommon, occurring in only about 1% of patients with obstructive coronary artery disease. In contrast to ST-segment depression, the development of ST-segment elevation is useful in localizing the anatomic site of ischemia and typically correlates with a perfusion defect on imaging studies. In patients with early repolarization, the normal response is for the elevated J points to return to baseline during exercise.

ST-segment elevation during exercise testing does not have predictive significance when it occurs in leads that contain pathologic Q waves. In that situation, it may represent a region of myocardial scar with a resting wall motion abnormality.

There is no direct association between exercise-induced ST-segment elevation and the development of conduction system abnormalities.

ANSWER TO QUESTION 38

E (Braunwald, pp. 365-368)

Assessment of coronary artery calcification by electron beam tomography (EBT) using electrocardiographic gating is a technique to screen for coronary artery disease (CAD). Although coronary artery calcification is a surrogate marker for coronary atherosclerotic plaque, the correlation between the amount of coronary calcium and the actual angiographic severity of the CAD is weak. The complete absence of coronary calcification on EBT has a strong negative predictive value for high-grade coronary stenosis but does not completely rule out the presence of significant CAD. The Agatston score is the most frequently used system for reporting the severity of coronary artery calcifications, and reference data sets for interpretation are stratified by age and gender. Annual rates of myocardial infarction or cardiovascular death rise with increasing Agatston scores, even in patients with similar Framingham risk scores. Coronary EBT is of most clinical value for patients who are at *intermediate* risk for coronary events based on traditional cardiovascular risk factors and for whom an abnormal scan will have an impact on clinical management. There is currently no role for serial assessment of coronary calcification with EBT in clinical practice.

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ANSWER TO QUESTION 39

A (Braunwald, pp. 294-302, 310-316, 320-329)

Myocardial perfusion imaging provides an important source of prognostic information in patients with coronary artery disease (CAD). Stress myocardial perfusion imaging has been shown to be a powerful predictor of subsequent cardiac events. The combination of clinical and myocardial perfusion data is more predictive than the combination of clinical and cardiac catheterization data. Indeed, even when angiographic CAD is present, a normal myocardial perfusion study confers a very low risk of a subsequent cardiovascular event (<1% per year).

Stress perfusion defects in multiple locations corresponding to multiple vascular territories are suggestive of left main or three-vessel coronary artery disease. Other indicators of high-risk coronary artery disease include large defects, transient pulmonary uptake of tracer, and left ventricular cavity dilatation with exercise. The severity of a myocardial perfusion defect can be assessed in terms of both its size and the extent of its reversibility. A severe defect is one that has little or no uptake with stress imaging, whereas a mild defect may have only a slight reduction in counts with stress. The severity of defects as well as their number and size are important indicators of prognosis. The predictive value of myocardial perfusion imaging is independent of the imaging technique (planar or single-photon emission computed tomography) and the imaging agent used (thallium-201 or technetium-99m sestamibi), with one important exception. There is a decreased likelihood of significant breast attenuation artifact with the use of technetium-99m sestamibi imaging in women, and this imaging agent is preferable in female patients.

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ANSWER TO QUESTION 40

A (Braunwald, pp. 351-352)

Cardiac magnetic resonance (CMR) imaging is an excellent technique to define pericardial abnormalities. It is highly sensitive for the detection of pericardial fluid, masses, and pericardial thickening. The T1-weighted spin-echo CMR image in this case demonstrates diffuse encasement of the heart by a structure within the pericardium that has intermediate signal intensity resembling myocardial tissue (labeled with asterisks in Fig. 1-67). This abnormality is homogeneous in signal intensity such that organized hematoma is unlikely. The features are most consistent with pericardial malignancy.

Biopsy of the affected region demonstrated pericardial angiosarcoma. This is a rare primary pericardial

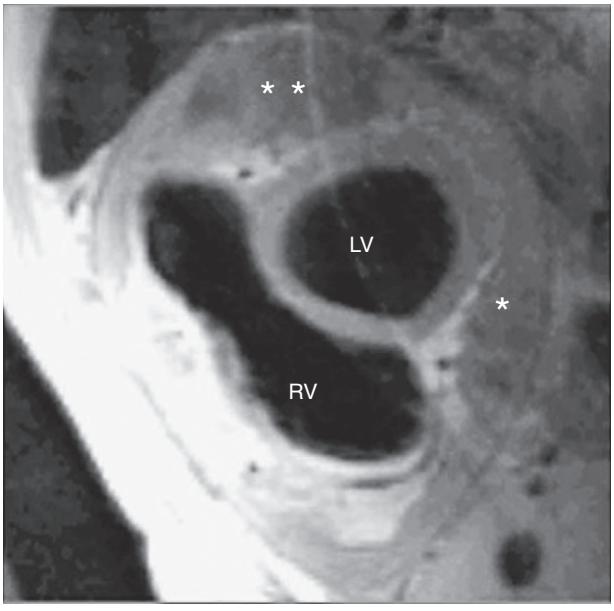

FIGURE 1-67

malignancy that arises from the pericardial vasculature and typically does not metastasize. However, it usually proliferates widely throughout the pericardial cavity and may invade the myocardium. This case was considered inoperable at surgery and the patient died 4 days after presentation.

REFERENCE

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ANSWER TO QUESTION 41

C (Braunwald, pp. 396-398, 400-401)

In a normal individual, systemic and pulmonary cardiac outputs are approximately equal. In the presence of an intracardiac shunt, blood flows abnormally between the pulmonary and systemic circulations. Although many shunts are suspected before cardiac catheterization, certain findings during the procedure may point to unexpected intracardiac communications. For example, suspicion for a left-to-right shunt should be raised if the pulmonary artery oxygen saturation exceeds 80% or if the difference in oxygen saturation between the superior vena cava (SVC) and the pulmonary artery is 8% or more. A right-to-left shunt should be considered if the systemic arterial saturation is < 93% without any other reason.

Normally, the oxygen saturation in the inferior vena cava (IVC) is higher than that in the SVC. Mixed venous saturation is most accurately determined in the pulmonary artery, because complete mixing of venous return has occurred at that level. However, if one is assessing a transatrial shunt with left-to-right flow, the mixed venous saturation must be determined proximal to the shunt and can be estimated by the Flamm formula, measuring oxygen content in the SVC and IVC:

$$\text{Mixed venous oxygen content} = \frac{3(\text{SVC } O_2 \text{ content}) + 1(\text{IVC } O_2 \text{ content})}{4}$$

The ratio of pulmonic to systemic blood flow (Qp/Qs) is used to determine the significance of an intracardiac shunt. A Qp/Qs of <1 indicates net right-to-left shunting. A Qp/Qs of 2.0 or more indicates a large left-to-right shunt that generally requires repair. A Qp/Qs of 1.0 to 1.5 indicates a small left-to-right shunt.

ANSWER TO QUESTION 42

C (Braunwald, pp. 247-250, 1582-1588; Fig. 15-66)

The pulsed-wave Doppler spectrum in the illustration demonstrates late systolic acceleration of flow characteristic of left ventricular outflow tract (LVOT) obstruction, typically seen in patients with hypertrophic cardiomyopathy. This abnormal Doppler signal ("dagger" pattern) is detected in the LVOT and peaks significantly later than that of valvular aortic stenosis, owing to the dynamic nature of the obstruction. Because the disorder is at the subvalvular level, there is no therapeutic role for aortic valve surgery.

The LVOT obstruction may be worsened by any action that decreases left ventricular volume and narrows the distance between the interventricular septum and the anterior mitral leaflet, including volume depletion. Although compression stockings may redistribute volume and increase venous return, such therapy is typically not sufficient to prevent or treat LVOT obstruction. Similarly, bed rest has no specific role in the management of hypertrophic cardiomyopathy.

ANSWER TO QUESTION 43

A (Braunwald, pp. 251-256, 351-352, 1655-1660; Figs. 75-8 and 75-9)

Echocardiography is an excellent technique to detect and grossly quantify the volume of pericardial effusions. The normal pericardial space contains 35 to 50 mL of serous fluid between the visceral and parietal pericardial layers. Small pathologic effusions tend to accumulate posteriorly, external to the left ventricular free wall, because of the effects of gravity. Larger effusions tend to circumscribe the heart. Certain echocardiographic features are good indicators of the presence of cardiac tamponade physiology in patients with pericardial effusion. These include early diastolic collapse of the right ventricle, which indicates the presence of elevated intrapericardial pressure (see Braunwald, Fig. 75-8). However, right ventricular (RV) diastolic collapse may be absent in clinical tamponade if pulmonary hypertension or RV hypertrophy is present, because these forces oppose RV indentation. Diastolic invagination of the right atrium (see Braunwald, Fig. 75-9) is a more sensitive, but less specific, marker of tamponade physiology. Doppler interrogation can also provide clues to the presence of tamponade, including exaggerated respiratory variation of transvalvular velocities (see Answer to Question 6).

Transthoracic echocardiographic imaging is often inadequate in direct assessment of the thickness of the pericardium; transesophageal echocardiography, computed tomography, and magnetic resonance imaging are more accurate techniques for this purpose.

ANSWER TO QUESTION 44

E (Braunwald, pp. 302, 310-311, 316-319, 320-325, 327, 334)

Although nuclear stress testing is commonly used in the evaluation of women with suspected coronary artery disease (CAD), there is the possibility of breast attenuation artifact in female patients. The use of technetium-99m–based agents with single-photon emission computer

tomography imaging and electrocardiographic gating reduce the likelihood of a false-positive study due to such artifact, thereby increasing the specificity of nuclear testing in women. Breast attenuation artifact typically appears as a fixed defect of the anterior or anterolateral wall. If this defect is due to artifact rather than prior infarction, the involved segment(s) will demonstrate normal wall motion on the gated images (see Braunwald, Fig. 17-12, and Answer to Question 11).

Patients with left bundle branch block (LBBB) may demonstrate artifactual exercise-induced perfusion defects, especially in the septal and anteroapical regions. Pharmacologic stress testing with dipyridamole or adenosine minimizes the incidence of such artifacts. Thus it is recommended that patients with complete LBBB on the ECG be evaluated by vasodilatory agents, rather than an exercise protocol, to avoid false-positive results.

Nuclear imaging is a useful modality for preoperative risk stratification before noncardiac surgery, especially for patients at intermediate clinical risk.[1] For example, dipyridamole stress testing with perfusion imaging is predictive of perioperative cardiac events and the magnitude of risk correlates with the extent of ischemia. Patients with diabetes are at increased risk for CAD and its complications, and perfusion defects during testing predict higher event rates in diabetics compared with nondiabetics.[2]

In patients with CAD, assessment of myocardial viability is often of great importance in defining revascularization options. Modalities for the determination of myocardial viability include thallium-201 imaging, magnetic resonance imaging, dobutamine echocardiography, and positron emission tomography.[3]

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ANSWER TO QUESTION 45

B (Braunwald, pp. 343-346)

In this delayed cardiac magnetic resonance (CMR) image, the midseptum and midanterior segments of the left ventricle demonstrate subendocardial late enhancement (the bright area indicated by the white arrows in Fig. 1-68) that involves approximately half of the transmural thickness of the myocardium. This finding, as well as the matching wall motion abnormality by echocardiography, is most consistent with a prior nontransmural myocardial infarction (MI). The presence of MI (acute or old) can be determined accurately using the protocol of late enhancement, which involves injecting intravenous gadolinium and performing CMR after a delay. Gadolinium is an extracellular contrast agent that only minimally

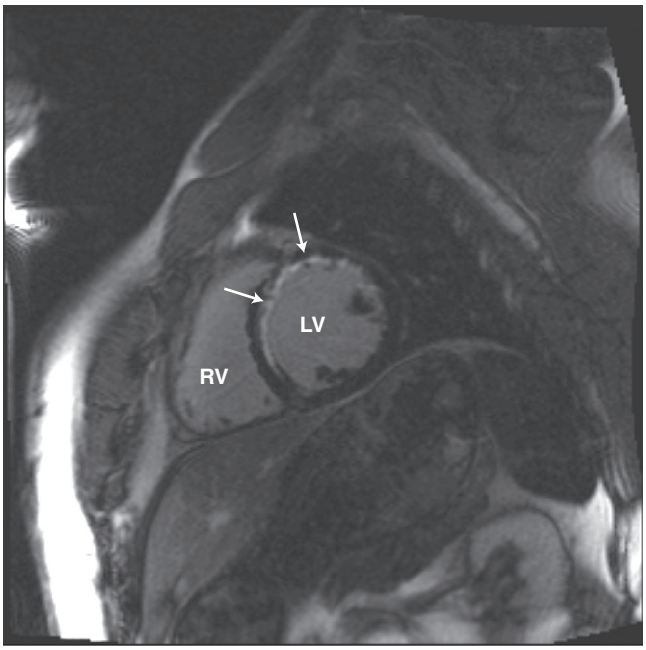

FIGURE 1-68

enters normal myocardial cells. However, disrupted myocardium after an MI allows expansion of the volume of distribution and delayed entry of the contrast agent into the affected region. A subendocardial distribution is indicative of myocardial infarction. Both conventional CMR and late gadolinium enhancement are useful in assessing myocardial viability, to help determine whether a segment of poorly contracting myocardium would benefit from mechanical revascularization. For example, improved wall thickening with low-dose dobutamine CMR correlates well with the presence of viable myocardium. With the late gadolinium enhancement technique, a transmural extent of MI (i.e., region of late enhancement) of <50% is also predictive of functional recovery after revascularization.

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ANSWER TO QUESTION 46

B (Braunwald, pp. 230-231, 234-236, 398, 1474)

As with most stenotic valvular lesions, echocardiographic evaluation of aortic stenosis is accurate and clinically useful. The morphology of the valve can be examined by two-dimensional imaging to assess for congenital or rheumatic abnormalities or age-related changes of valvular architecture. The peak outflow velocity is then measured using continuous-wave Doppler imaging. Important aortic stenosis usually results in outflow velocities of