PRACTICAL CARDIOLOGY REVIEW

A SELF-ASSESSMENT TOOL

EDITED BY

Majid Maleki
Azin Alizadehasl
Majid Haghjoo

Practical Cardiology Review

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A Self-assessment Tool

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PREFACE

Practical Cardiology Review contains more than 400 multi-choice questions and detailed answers regarding various cardiology cases.

This book should appeal to a wide range of colleagues, from practicing professionals to residents and fellows preparing for higher qualifications, thanks to its informative chapters on such mainstays of cardiology as physical examinations, electrocardiography, echocardiography, electrophysiology, intervention, and pharmacological treatment, as well as such commonly confronted issues as cardiomyopathy, heart failure, and cardiac surgery.

Practical Cardiology Review is a learning tool to complement the previously printed books on practical cardiology. The authors each have contributed original questions and meticulous answers in their particular field of expertise.

As cardiologists, we feel a responsibility to be at the cutting edge of the ever-advancing cardiology so as to be able to manage the vast array of cardiac diseases, many of which are life-threatening.

We have had the immense pleasure of collaborating with Cambridge Scholars Publishing in the preparation of this book and wish to express our heartfelt thanks to its editorial and printing departments.

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CHAPTER 1

EVALUATION OF PATIENTS WITH CARDIOVASCULAR PROBLEMS

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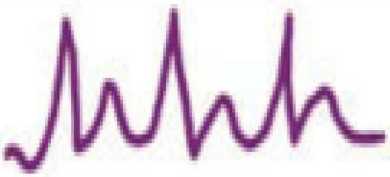
1. What is your diagnosis?



A. Hyperkinetic pulse in a high output state
B. Bisferiens pulse in hypertrophic obstructive cardiomyopathy
C. Dicrotic pulse
D. Pulsus alternans in heart failure

Answer: D

The pulse pressure waveform shows the variation in pressure between the beats in pulsus alternans. Pulsus alternans is a physical finding, with the arterial pulse waveform presenting alternating strong and weak beats. It is almost always indicative of left ventricular systolic impairment, and carries a very poor prognosis.

2. Which phrase is incorrect about V waves?

   A. They start at the end of the systole.
   B. TR produces a prominent V wave that is premature (CV wave) and makes the X descent disappear.
   C. High cardiac output states such as exercise, anemia, and hyperthyroidism may cause increased V wave amplitude.
   D. ASD decreases V wave amplitude.

Answer: D

V wave starts at the end of systole. TR produces a prominent V wave that is premature (CV wave) and makes the X descent disappear. High cardiac output states such as exercise, anemia, and hyperthyroidism may cause increased V wave amplitude. ASD can increase V wave amplitude.

3. The paradoxical bulging of LV impulse is seen in:

   A. Severe MR
   B. IHD
   C. Severe AI
   D. LVH

Answer: B

The paradoxical bulging of LV impulse is seen in myocardial infarction with LV apical aneurysm.

4. The following picture shows a 34-year-old man after coronary artery bypass surgery. What is your diagnosis?

A. Progeria syndrome
B. Turner syndrome
C. Holt-Oram syndrome
D. Friedreich's ataxia

Answer: A

Progeria is an extremely rare autosomal dominant genetic disorder in which symptoms resembling aspects of aging are manifested at a very early age. Progeria is one of several progeroid syndromes.

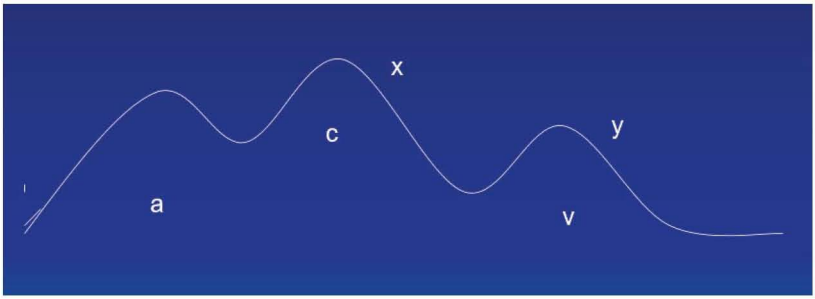
5. Soft S1 is heard in:

A. Pregnancy
B. Arteriovenous fistula
C. Severe MR
D. Atrial myxoma

Answer: C

Soft S1 is heard in calcified mitral valve, first-degree AV block, severe MR, dilated cardiomyopathy and myocarditis.

6. Which of the following processes is not depicted in the diagram (jugular venous pulse)?



A. Atrial contraction (a)
B. Closure of tricuspid valve (c)
C. Ventricle begins to fill (x)
D. Tricuspid valve opening and ventricular filling (y)

Answer: C

Jugular venous pulse waves indicate the following:

a: Atrial contraction
c: Closure of tricuspid valve
x: Atria begins to fill
v: Volume of atria increases
y: Tricuspid valve opening and ventricular filling

7. Which phrase is incorrect about S3?

A. It occurs in early diastole (soon after S2).
B. Blood rushes in just after the mitral valve opens, striking the LV wall (PALPABLE).
C. It is heard only at the left sternal border.
D. It can also be heard in healthy young people.

Answer: C

S3 is heard at apex only.

8. Which phrase is incorrect about aortic valve stenosis murmur?
A. It has a harsh quality.
B. It is best heard in LSB.
C. It is accompanied by a soft S2.
D. It has a diamond-shaped pattern.

Answer: B

Aortic Stenosis features: Harsh murmur at right sternal border, soft S2, diamond-shaped pattern, late peak, delay in carotid pulse, radiates to carotid artery and failure to radiate makes aortic stenosis less likely.

References

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CHAPTER 2

ELECTROCARDIOGRAPHY

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1. In which situation could the right QRS axis deviation not be seen?
   A. Right ventricular hypertrophy
   B. Left posterior fascicular block
   C. Dextrocardia
   D. Primum atrial septal defect

Answer: D

QRS complex: The initial wave (low amplitude and brief duration, less than 30 ms) is positive in leads aVR and V1, and is negative in leads I, aVL, V5, and V6 (representing septal q waves). The QRS pattern in inferior leads may be predominantly upright (qR, rS or RS pattern complexes), and in lead I may present as an isoelectric RS pattern or a predominantly upright qR pattern. The normal mean QRS axis in adults is between −30° and +90° (Table 2.1). Causes of QRS axis deviation are shown in Table 2. In pericardial leads, the transition zone (in this zone, the QRS complex is present as an isoelectric RS pattern) typically occurs in lead V3 or V4. It should be mentioned that men have greater QRS amplitudes and longer QRS durations than women. In contrast to PR interval, the duration of the QRS complex is not influenced by heart rate.

Table 2.1 Axis of QRS complex.

	Range of QRS axis (° degrees)
Normal	−30 to +90
Right axis deviation	> +90
Moderate	+90 to +120
Marked	+120 to +180
Left axis deviation	< −30
Moderate	−30 to −45
Marked	−45 to −90
Superior axis deviation	−80 to −90
Extreme axis deviations (right superior axis deviations)	−90 to −180 (+180 to +270)

Table 2.2 Causes of QRS axis deviation

Right axis deviation	**Left axis deviation**
Normal variation (vertical heart with an axis of 90°)	Normal variation (physiological, often with age)
Mechanical shifts, such as inspiration and emphysema	Mechanical shifts, such as expiration, high diaphragm (pregnancy, ascites, abdominal tumor)
Right ventricular hypertrophy	Left ventricular hypertrophy
Right bundle branch block	Left bundle branch block
Left posterior fascicular block	Left anterior fascicular block
Dextrocardia	Congenital heart disease (primum atrial septal defect, endocardial cushion defect)
Ventricular ectopic rhythms	Emphysema
Pre-excitation syndrome (Wolff-Parkinson-White)	Pre-excitation syndromes (Wolff-Parkinson-White)
Lateral wall myocardial infarction	Inferior wall myocardial infarction
Secundum atrial septal defect	Ventricular ectopic rhythms
	Hyperkalemia

2. In which situation, is the J point elevation abnormal and needs more evaluation?

   A. J point elevation equals 0.2 in lead V4 in a 30-year-old man.
   B. J point elevation equals 0.2 in lead V2 in a 50-year-old man.
   C. J point elevation equals 0.25 in lead V2 in a 30-year-old man.
   D. J point elevation equals 0.15 in lead V2 in a 50-year-old women.

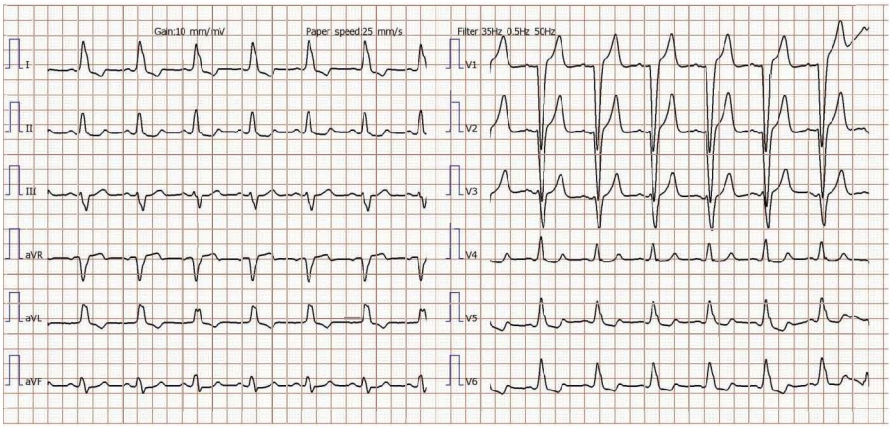
Answer: A

J point: The intersection of the end of the QRS complex and the beginning of the ST segment is termed the J point, and it is normally at or near the isoelectric baseline of the ECG. The greatest amplitude of J point is seen in lead V2 (Table 2.3) (1).

Table 2.3 Upper limits of normal J point elevation based on various conditions.

Leads V2 and V3	
Men ≥ 40 years	0.2 mV
Men < 40 years	0.25 mV
Women	0.15 mV
Leads (except V2 and V3)	0.1 mV

3. Regarding ventricular repolarization interval calculation in this ECG, which method is correct?



A. Corrected QT interval based on Bazett's Formula
B. Corrected QT interval based on Fridericia's formula
C. Corrected QT interval based on a linear model
D. JT interval

Answer: D

QT and JT intervals: JT interval is a more accurate measure of ventricular repolarization, especially in patients with bundle branch block (BBB). The QT interval is rate-dependent, decreasing as heart rate increases.

Formula for measurement of corrected QT interval:

1. Bazett's Formula (QT and RR intervals are measured in seconds): $QTc = QT/\sqrt[2]{RR}$
2. Fridericia's formula (QT and RR intervals are measured in seconds): $QTc = QT/\sqrt[3]{RR}$
3. Formula based on linear model (intervals are measured in milliseconds): QTc = QT +1.75(HR − 60)

Formula for measurement of corrected QT interval in patients with atrial fibrillation:

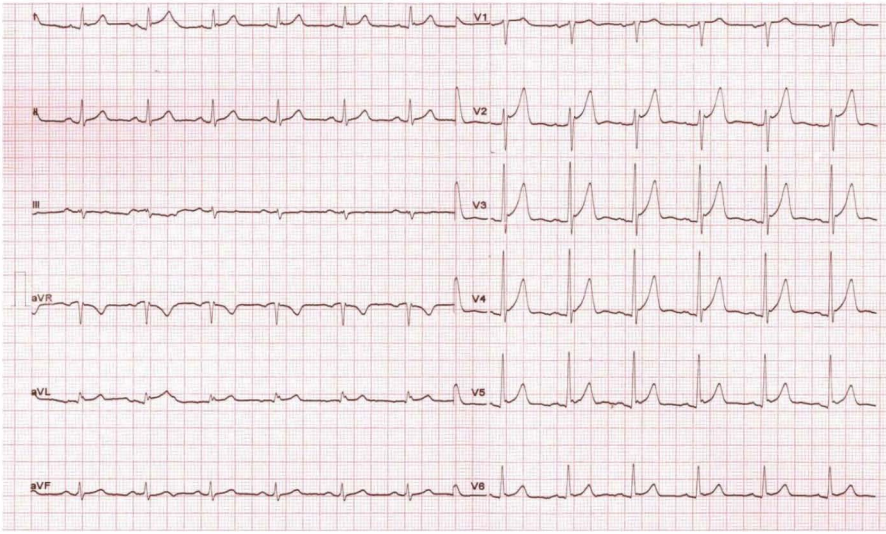
1. $(QTc_{short} + QTc_{long})/2$
2. Onset of R wave to peak of the T wave. (If this was more than half the R-R interval, then the QTc interval is likely to be above the critical threshold of 500 ms)
3. Average of multiple QTc intervals (up to 10)
4. Measure the QT interval in a beat that the R-R interval that is of exactly 1-second duration (because the square root of 1 is 1, so, in this beat, the QTc interval = non-corrected QT interval)

Formula for measurement of corrected JT interval (in the setting of a BBB):

1. Formula based on QT interval: JTc = QTc – QRS
2. JT interval = JT (HR + 100)/518

A JT interval equal to or greater than 112 ms can be identified as a repolarization prolongation.

4. A 47-year-old man presents with atypical chest pain in your emergency department. The CT angiogram shows normal coronary arteries. Which diagnosis is correct?



A. Benign early repolarization
B. Malignant early repolarization
C. Prinzmetal angina
D. Acute pericarditis

Answer: A

Benign early repolarization

The ECG pattern of benign early repolarization (BER) is most commonly seen in young, healthy patients under 50 years of age (Table 4). It produces widespread ST segment elevation that may mimic pericarditis or acute MI. BER can be difficult to differentiate from pericarditis because both conditions are associated with concave ST elevation. These two conditions can be distinguished using the ST segment elevation (from the end of the PR segment to the J point) compared to the amplitude of the T wave in V6. An ST segment / T-wave ratio of more than 0.25 suggests pericarditis, but if this ratio is less than 0.25, it is consistent with BER (Table 2.5, Figure 2.1).

Table 2.4 Criteria for benign early repolarization

Widespread concave ST elevation, most prominent in the mid- to left precordial leads (V2-5).
Concavity of initial upsloping portion of ST segment.
Notching or slurring at the J point.
Prominent, slightly asymmetrical T waves which are concordant with the QRS complexes (pointing in the same direction).
The degree of ST elevation is modest in comparison to the T wave amplitude (less than 25% of the T wave height in V6; against pericarditis).
ST elevation is compatible with the criteria in Table 3.
No reciprocal ST depression to suggest STEMI (except in aVR).
ST changes are relatively stable over time (no progression on serial ECG tracings).
Reduction in ST segment elevation with sympathomimetic factors.

Table 2.5 Criteria for distinguishing BER from pericarditis.

In favor of BER	In favor of pericarditis
ST elevation limited to the precordial leads	Generalized ST elevation
Absence of PR depression	Presence of PR depression
Prominent T waves	Normal T-wave amplitude
ST segment / T-wave ratio < 0.25	ST segment / T-wave ratio > 0.25
Characteristic “fish-hook” appearance in V4	Absence of “fish hook” appearance in V4
ECG changes relatively stable over time	ECG changes evolve over time

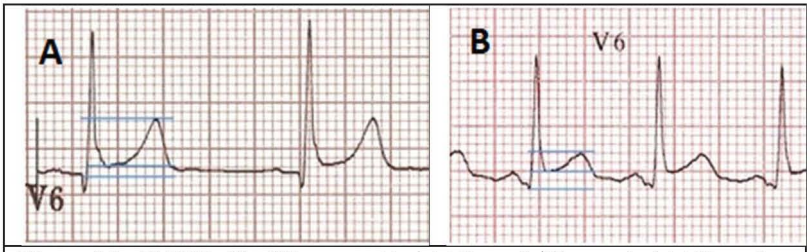


Figure 2.1: A) BER: The ST-segment height (1 mm) / T-wave height (6 mm) ratio is equal 0.16. B) Pericarditis: The ST-segment height (2 mm) / T-wave height (4 mm), ratio is equal (0.5).

5. Which of the following is not compatible with right atrial abnormalities?

A. Rightward shift of mean P wave axis to more than +75 degrees.
B. Increased duration and depth of terminal-negative portion of P wave in lead V1 (P terminal force) such that area subtended by it is >0.04 mm-sec.
C. qR pattern in the right precordial leads without evidence of myocardial infarction (especially with other signs of RV overload)
D. Low-amplitude (<600 μV = 6 mm at usual gain) QRS complexes in lead V1 with a ≥ 3 increase in lead V2

Answer: B

Atrial abnormality

The three general categories of P wave that can alter atrial activation to produce abnormal P wave patterns are: abnormal patterns of activation and conduction, left atrial abnormalities, and right atrial abnormalities. The common diagnostic criteria for left and right atrial abnormalities are listed in Table 6.

Left atrial abnormality: Anatomical abnormalities of the left atrium that alter the P waves include atrial dilation, atrial muscular hypertrophy, and elevated intra-atrial pressures. The most common conditions that lead to left atrial enlargement include hypertension, valvular heart diseases, heart failure and atrial fibrillation.

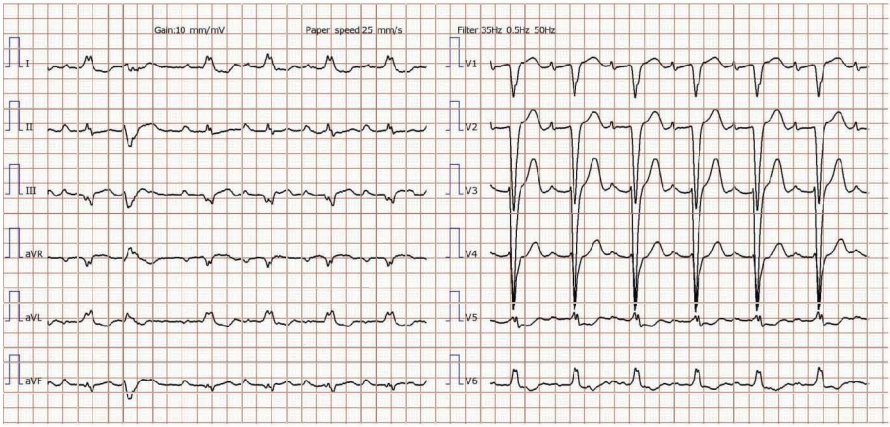
Right atrial abnormality: Right atrial abnormality is a marker of the severity of disease, and also predicts outcome in some disorders including tricuspid regurgitation, pulmonary hypertension, congenital heart disease, and right heart failure. Enlargement of the right atrium may result from right atrial volume or pressure load.

Biatrial abnormality: ECG pattern findings include large biphasic P waves in lead V1 and tall and broad P waves in leads II, III, and aVF.[11]

Table 2.6 Common diagnostic criteria for left and right atrial abnormalities

Left atrial abnormalities	Right atrial abnormalities
• Prolonged P wave duration to >120 ms in lead II • Prominent notching of P wave, usually most obvious in lead II, with an interval between notches of >0.40 ms ("P mitrale") • Ratio between the duration of P wave in lead II and duration of PR segment >1.6 • Increased duration and depth of terminal-negative portion of P wave in lead V1 (P terminal force) so that area subtended by it is >0.04 mm-sec • Leftward shift of mean P wave axis to between −30 and −45 degrees	• Peaked P waves with amplitudes in lead II to >0.25 mV ("P pulmonale") • Prominent initial positivity in lead V1 or V2 >0.15 mV (1.5 mm at usual gain) • Increased area under initial positive portion of P wave in lead V1 to >0.06 mm-sec • Rightward shift of mean P wave axis to more than +75 degrees • qR pattern in the right precordial leads without evidence of myocardial infarction (especially with other signs of RV overload) • low-amplitude (<600 μV = 6 mm at usual gain) QRS complexes in lead V1 with a ≥ 3 increase in lead V2

6. Select the correct option based on this ECG.



A. Left ventricular hypertrophy
B. Hypertrophic cardiomyopathy
C. Dilated cardiomyopathies
D. Biventricular hypertrophy

Answer: C

Dilated cardiomyopathies (DCM): The presence of a relatively low limb voltage (QRS voltage <0.8 mV in each of the limb leads) accomplished with a relatively prominent QRS voltage in the pericordial leads (SV1 or SV2 + RV5 or RV6 >3.5 mV) and poor R wave progression in the pericardial leads suggests DCM. These changes are relatively specific but not a sensitive sign for DCM.[14]

7. In a patient with right ventricular hypertrophy (RVH), which of these options correlates more closely to right ventricular volume overload compared with severe concentric RVH?

A. Tall R waves in anteriorly and rightward-directed leads (leads aVR, V1, and V2)
B. rSr′ pattern in V1
C. Deep S waves and small r waves in leftward-directed leads (I, aVL, and lateral precordial leads)
D. Shift in the frontal plane QRS axis to the right
E. Presence of S waves in leads I, II, and III (the S1S2S3 pattern)

Answer: B

Right ventricular hypertrophy: The common diagnostic criteria for RVH and the ECG criteria for RVH based on its severity are listed in Tables 2.7 and 2.8. Evidence of true RVH in patients with chronic obstructive pulmonary disease includes a positive right axis deviation of more than 110 degrees, deep S waves in the lateral precordial leads, and an S1Q3T3 pattern. Hyperinflation of the lungs in these patients could cause a decrease the amplitude of the QRS complex, right axis deviation, and delayed transition in the precordial leads even in the absence of RVH.

Table 2.7 Common diagnostic criteria for RVH

• R in V1 ≥0.7 mV • QR in V1 • R/S in V1 >1 with R >0.5 mV • R/S in V5 or V6 <1 • S in V5 or V6 >0.7 mV • R in V5 or V6 ≥0.4 mV with S in V1 ≤0.2 mV • Right axis deviation (>90 degrees) • S1Q3 pattern • S1S2S3 pattern • P pulmonale

Table 2.8 ECG criteria for RVH based on severity

Moderate to severe concentric RVH	Less severe hypertrophy (limited to the outflow tract of the right ventricle*)
• Tall R waves in anterior and rightward-directed leads (leads aVR, V1, and V2) • Deep S waves and small r waves in leftward-directed leads (I, aVL, and lateral precordial leads) • A reversal of normal R wave progression in precordial leads • Shift in the frontal plane QRS axis to the right • Presence of S waves in leads I, II, and III (the S1S2S3 pattern).	• rSr′ pattern in V1 • Persistence of s (or S) waves in the left precordial leads
* This pattern is typical of right ventricular volume overload such as that produced by an atrial septal defect.	

8. Which of the following is more often associated with a massive pulmonary embolism?

A. QR or qR pattern in the right ventricular leads
B. S1Q3T3 pattern
C. ST-segment deviation and T-wave inversions in leads V1 to V3
D. Incomplete or complete right bundle branch block (RBBB).
E. Atrial fibrillation
F. ST-segment elevations in the right mid-precordial leads

Answer: F

Pulmonary embolism: ECG changes in patients with pulmonary embolism include:

1. Sinus tachycardia (the most common abnormality)
2. QR or qR pattern in right ventricular leads
3. S1Q3T3 pattern
4. ST-segment deviation and T-wave inversions in leads V1 to V3
5. Incomplete or complete right bundle branch block (RBBB)
6. Right axis deviation
7. Right atrial enlargement (P pulmonale)
8. Atrial tachyarrhythmias (atrial fibrillation, flutter, and tachycardia)
9. ST-segment elevations in the right mid-precordial leads (in massive pulmonary arterial obstruction)

9. Which of the following is not correct in a patient with left bundle branch block?

A. Broad, notched, or slurred R waves in leads I, aVL, V5, and V6
B. Late intrinsicoid (prolonged time to peak R wave) in V1 >50 ms
C. Small or absent initial r waves in right precordial leads (V1 and V2) followed by deep S waves
D. Absent septal q waves in leads I, V5, and V6

Answer: B

Left bundle branch block: The common diagnostic criteria for LBBB are listed in Table 2.9. The mean QRS axis can be normal, deviated to the left, or deviated to the right. In most cases, the ST-T segments are discordant with the QRS complex. LBBB has significant prognostic implications. In patients with or without overt heart disease, LBBB is associated with a higher risk of mortality and morbidity from myocardial infarction, heart failure, and arrhythmias such as high-grade atrioventricular block.

Table 2.9 Common diagnostic criteria for bundle branch blocks

Complete Left Bundle Branch Block	Complete Right Bundle Branch Block
• QRS duration ≥120 ms • Broad, notched, or slurred R waves in leads I, aVL, V5, and V6 • Small or absent initial r waves in right precordial leads (V1 and V2) followed by deep S waves • Absent septal q waves in leads I, V5, and V6 • Late intrinsicoid (prolonged time to peak R wave) in V5 and V6 >60 ms	• QRS duration ≥120 ms • rsr′, rsR′, or rSR′, patterns in leads V1 and V2 • S waves in leads I and V6 ≥40 ms wide • Early intrinsicoid (normal time to peak R wave) in leads V5 and V6 • Late intrinsicoid (Prolonged time to peak R wave) in V1 >50 ms

10. In patients with unstable angina, which of these ECG findings is less correlated with short-term risk for death or nonfatal myocardial ischemia?

A. Angina at rest with transient ST-segment depression of 0.1 mV in leads V1-4
B. New left bundle branch block
C. Sustained ventricular tachycardia
D. T-wave inversion of 0.2 mV in leads V1-4
E. Pathologic Q waves in inferior leads
F. ST-segment depression of 0.05 mV in leads V1-3

Answer: F

Myocardial Infarction: An ECG should be taken within 10 minutes of arrival at the emergency department for patients suspected of acute coronary syndrome. During a symptomatic episode at rest, new persistent or transient ST-segment abnormalities (≥0.05 mV) are strongly suggestive of acute ischemia and severe coronary disease. A normal ECG has a negative predictive value of 80% to 90%; however, a normal ECG does not exclude the possibility of acute coronary syndrome. The risk for acute MI in a patient with a history of CAD and normal ECGs is approximately 4%, and if they do not have a history of CAD, the risk will be 2%. Nonspecific ST-T-wave changes (ST-segment deviation <0.05 mV or T-wave inversion ≤0.2 mV) with or without abnormal Q waves are the most common abnormalities on the ECG in patients with chronic CAD. The presence of a hyperacute T wave or an upright T wave in lead V1 more than lead V6 in a patient with angina pectoralis is highly suggestive of the presence of subendocardial ischemia secondary to coronary artery involvement. However, a symmetrically and deeply inverted T wave suggests transmural ischemia. The likelihood and short-term risks for acute coronary syndrome based on ECG findings are shown in Table 2.10.

Table 2.10 The likelihood and short-term risk for acute coronary syndrome (ACS)

A) Likelihood for ACS based on ECG findings		
High	Intermediate	Low
• New or presumably new transient ST-segment deviation (≥0.1 mV) or T-wave inversion (≥0.2 mV) in multiple precordial leads	• Fixed Q waves • ST-segment depression of 0.05-0.1 mV or T-wave inversion >0.1 mV	• T-wave flattening or inversion <0.1 mV in leads with dominant R waves • Normal ECG
B) Short-term risk of death or nonfatal myocardial ischemia in patients with unstable angina based on ECG findings		
High	Intermediate	Low
• Angina at rest with transient ST-segment changes >0.05 mV • Bundle branch block, new or presumed new • Sustained ventricular tachycardia	• T-wave changes • Pathologic Q waves or resting ST-segment depression <0.1 mV in multiple lead groups (anterior, inferior, lateral)	• Normal or unchanged ECG

11. In which situation, would ST-segment elevation not be seen?

A. Acute pulmonary embolism
B. Brugada pattern
C. Class IB antiarrhythmic drugs
D. Hypercalcemia
E. Hyperkalemia
F. Hypothermia

Answer: C

Electrocardiographic differential diagnosis of ischemia and infarction is shown in Table 2.11.

Table 2.11 Electrocardiographic differential diagnosis of ischemia and infarction

Q wave	ST elevation	Tall R Waves in Leads V1 and V2
Physiologic or Positional Factors	Myocardial ischemia or infarction	**Physiologic and Positional Factors**
Normal variant septal Q waves	Non-infarction, transmural ischemia (e.g. Prinzmetal angina pattern, takotsubo syndrome)	Misplacement of chest leads
Normal variant Q waves in V1-V2, III, and aVF	Acute myocardial infarction (due to obstructive coronary occlusion or other causes)	Normal variants
Left pneumothorax or dextrocardia—loss of lateral R-wave progression	Post-myocardial infarction (ventricular aneurysm pattern)	Displacement of heart toward right side of chest (dextroversion), congenital or acquired
Myocardial Injury or Infiltration	Acute pericarditis	**Myocardial Injury**
Acute processes—myocardial ischemia with or without infarction, myocarditis, hyperkalemia (rare cause of transient Q waves)	Normal variants (including the classic early	Lateral or true posterior myocardial infarction
		Duchenne muscular dystrophy

Chronic myocardial processes—idiopathic cardiomyopathies, myocarditis, amyloid, tumor, sarcoid

Ventricular Hypertrophy or Enlargement

Left ventricular (slow R-wave progression)

Right ventricular (reversed R-wave progression or poor R-wave progression, particularly with chronic obstructive lung disease)

Hypertrophic cardiomyopathy (can simulate anterior, inferior, posterior, or lateral infarcts)

Conduction Abnormalities

LBBB (slow R-wave progression)

WPW patterns

repolarization pattern)

LVH, LBBB (V1-V2 or V3 only)

Acute pulmonary embolism (right to mid-chest leads)

Brugada pattern (RBBB-like pattern and ST-segment elevations in right precordial leads; usually V1 to V2)

Class IC antiarrhythmic drugs (Usually V1 to V2)

Hypercalcemia (Usually V1 to V2)

DC cardioversion (immediately after procedure)

Hyperkalemia (Usually V1 to V2)

Hypothermia (J or Osborn wave)

Intracranial hemorrhage

Myocardial injury (e.g., due to trauma)

Myocarditis (may resemble myocardial infarction or pericarditis)

Tumor invading the left ventricle

Ventricular Enlargement

RVH (usually with right axis deviation)

Hypertrophic cardiomyopathy

Altered Ventricular Depolarization

Right ventricular conduction abnormalities

WPW patterns (caused by posterior or lateral wall preexcitation)

12. A 57-year-old man presented in the emergency department with STEMI. The coronary angiogram showed totally thrombotic LAD cut off from the ostium. Which of the following ECG findings is not expected in this patient?

A. ST elevation in leads aVR and aVL
B. ST depression in leads II and III equals 2mm, and aVF equals 2.5mm
C. ST elevation in lead V1 equals 1mm
D. ST depression in leads V5 and V6

Answer: C

Anterior STEMI: Pinpointing the location of the occlusion site in the LAD territory can be facilitated by identifying different patterns in the ECG (Table 2.12).

Table 2.12 ECG findings for detection of occlusion site of the culprit vessel in anterior wall STEMI (LAD Occlusion)

Proximal to the first septal branch and first diagonal branch ST deviation vector points to the base of heart, causing: • ST elevation in leads aVR and aVL • ST depression in leads II and III (> 1mm), and aVF (> 2mm) • ST elevation in lead V1 (>2 mm) and leads V2 to V4 • ST isoelectric or depressed in leads V5 and V6 NOTE: Acquired intra-Hissal block or RBBB may occur.
Distal to the first septal branch, proximal to the first diagonal branch ST deviation vector points to lead aVL, causing: • ST elevation in leads I and aVL • ST depression in lead III (lead II is isoelectric), but not in leads II or aVF • ST elevation in leads V2 to V6 but not in lead V1
Distal to the first diagonal branch, proximal to the first septal branch ST deviation vector points away from lead aVL and toward lead III, causing: • ST depression in lead aVL and aVR • ST elevation in inferior leads, highest in lead III • ST elevation in leads V1 to V4

Distal LAD
ST deviation vector points apically toward lead III, causing:
- ST depression in leads aVL and aVR
- ST elevation in inferior leads, highest in II
- ST elevation in leads V3 to V6

13. Which of the following does not meet Sgarbossa's criteria in a patient with LBBB and a normal axis?

A. ST-segment elevation ≥1 mm in leads I, aVL
B. ST-segment depression ≥1 mm in leads V1- V3
C. ST-segment elevation ≥5 mm in leads V1- V3
D. ST-segment depression ≥1 mm in leads I, aVL

Answer: D.

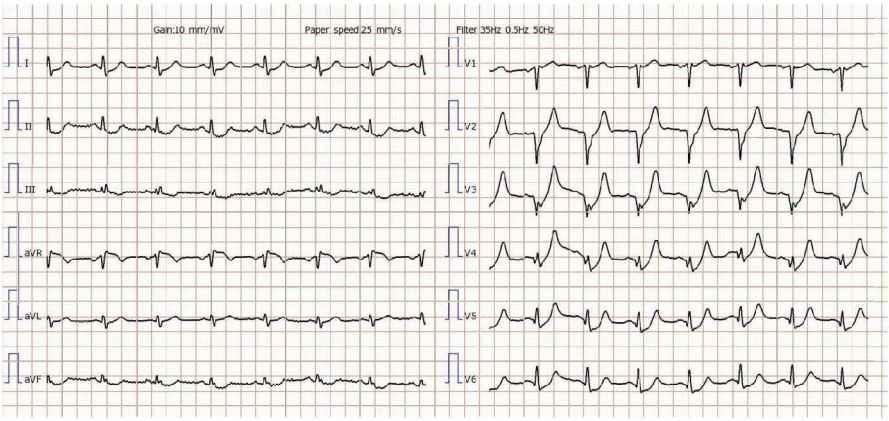
LBBB with Sgarbossa's criteria. Presence of a new LBBB is always pathological and can be a sign of myocardial infarction, although in patients with preexisting LBBB or ventricular paced rhythm, infarct diagnosis is difficult (Table 2.13).

Table 2.13 LBBB with Sgarbossa's criteria

- ST-segment elevation ≥1 mm and concordant with the QRS complex (5 points)
- ST-segment depression ≥1 mm in lead V1, V2, or V3 (3 points)
- ST-segment elevation ≥5 mm and discordant with the QRS complex (2 points)

A score of ≥3 has a specificity of 98% for acute MI

14. A 50-year-old male patient with a history of diabetes mellitus presented with at rest retrosternal angina at 11:00 pm. The ECG below was taken in the emergency department. What is the best strategy for this patient?



A. Primary PCI
B. Initiation of intravenous nitroglycerin; if chest pain and ECG changes are not relived, schedule the patient for angiography and PCI
C. Early invasive PCI
D. Ischemic guided PCI

Answer: A

De Winter pattern: The evidence suggests that this pattern is highly predictive of acute LAD occlusion. Some authors believe that patients with angina pectoralis and this ECG pattern should receive emergency reperfusion therapy with PCI or thrombolysis such as classic STEMI (Table 2.14).

Table 2.14 De Winter pattern

▪ Tall, prominent, symmetric T waves in the precordial leads ▪ Upsloping ST segment depression (1-3 mm) at the J point in the precordial leads ▪ Absence of ST elevation in the precordial leads ▪ ST segment elevation (1-2 mm) in lead aVR

15. Which of the following is not indicative of supraventricular tachycardia (SVT)?

A. Initiation with a premature P wave
B. Tachycardia complexes identical to those in resting rhythm
C. "Short-long" sequence preceding initiation
D. QRS contours consistent with aberrant conduction (V1, V6)
E. Slowing or termination with vagal maneuvers
F. Onset of the QRS to its peak (positive or negative) <50 ms

Answer: C

Tachyarrhythmia: Tachyarrhythmias are broadly divided into supraventricular tachycardia (SVT) and ventricular tachycardia (VT). Distinguishing between SVT and VT is important early in the acute management of a tachyarrhythmia, especially when SVT presents with wide QRS morphology. The most common criteria for distinction between SVT and VT in wide-complex tachycardia are listed in Table 2.15.

Table 2.15 Electrocardiographic distinctions for diagnosis of wide QRS complex tachycardia

Favor SVT	Favor VT
Initiation with a premature P wave	Initiation with a premature QRS complex
Tachycardia complexes identical to those in resting rhythm	Tachycardia beats identical to PVCs during sinus rhythm
"Long-short" sequence preceding initiation	"Short-long" sequence preceding initiation
Changes in the P-P interval preceding changes in the R-R interval	Changes in the R-R interval preceding changes in the P-P interval
QRS contours consistent with aberrant conduction (V1, V6)	QRS contours inconsistent with aberrant conduction (V1, V6)
Slowing or termination with vagal maneuvers	AV dissociation or other non-1:1 AV relationship
Onset of the QRS to its peak (positive or negative) <50 ms	Onset of the QRS to its peak (positive or negative) ≥50 ms
QRS duration ≤0.14 sec	QRS duration >0.14 sec
	Fusion beats, capture beats
	Left axis deviation (especially −90° to 180°)

	Concordant R-wave progression pattern Contralateral bundle branch block pattern from the resting rhythm Initial R, q, or r >40 ms or notched Q in aVR Absence of an "rS" complex in any precordial lead

Supraventricular tachycardia: In this setting, the QRS is generally narrow (duration <120 ms, often referred to as narrow-complex tachycardias); however, in some common scenarios, SVT can produce a wide QRS complex (secondary to BBB, IVCD, or preexcitation). The causes of SVT are listed in Table 12.6. To arrive at the correct diagnosis it is important to carefully search for clues during physical examination, evaluate the ECG during tachycardia, compare with sinus rhythm in the same leads, and evaluate the ECG during carotid sinus massage. RP-PR relationships help differentiate SVTs (Table 2.17). Also, QRS voltage can increase during an SVT.

Table 2.16 Characteristics of SVT

Type of SVT	P wave			QRS		Physical examination				Effect of CSM
	Rate (bpm)	Rhythm	Contour	Rate (bpm)	Rhythm	Pulse	Neck Vein Pulsation	Systolic Blood Pressure	Loudness of First Heart Sound	
Sinus tachycardia	100-180	Regular	May be peaked	100-180	Regular	Regular	Normal	Constant	Constant	Gradual and temporary slowing in heart rate
AV junctional Tachycardia	100-200	Regular	Retrograde	100-200	Regular	Regular	Frog sign	Constant	Variable; Constant if the atria are captured retrogradely	Temporary slowing of ventricular rate (AV block) or no effect
Atrial tachycardia	150-250	Regular; may be irregular	Abnormal	75-200	Generally regular	Generally regular	Normal	Constant	Constant	Paroxysmal: Cessation of tachycardia or no effect
					Irregular with variable conduction	Irregular				Incessant: Temporary slowing of ventricular rate (AV block) or no effect

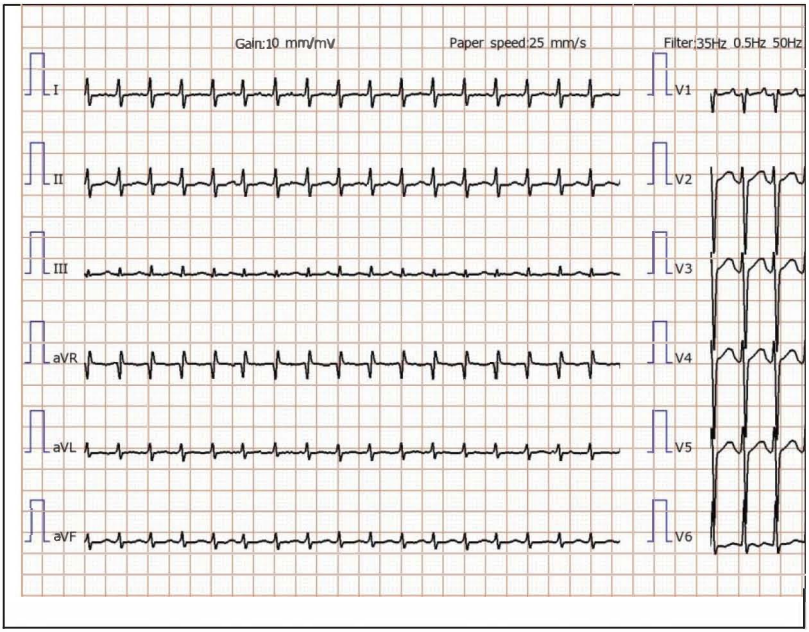
Atrial flutter	250-350	Regular	Sawtooth	75-175	Generally regular	Generally regular	Flutter waves	Constant if regular pulse	Constant if regular pulse	Temporary slowing of ventricular rate (AV block), conversion into atrial fibrillation, or no effect
					Irregular with variable conduction	Irregular		Changing if irregular pulse	Changing if irregular pulse	
Atrial fibrillation	400-600	Grossly irregular	Baseline undulation, no P waves	100-160	Grossly irregular	Irregular	Irregular	Changing	Changing	Temporary slowing of ventricular rate (AV block) or no effect
AV nodal reentry tachycardia	150-250	Very regular except at onset and termination	Retrograde; difficult to see	150-250	Very regular except at onset and termination	Regular	Frog sign	Constant	Constant	Cessation of tachycardia or no effect
AV reciprocating tachycardia	150-250	Very regular except at onset and termination	Retrograde; difficult to see	150-250	Very regular except at onset and termination	Regular	Frog sign	Constant	Constant	Cessation of tachycardia or no effect

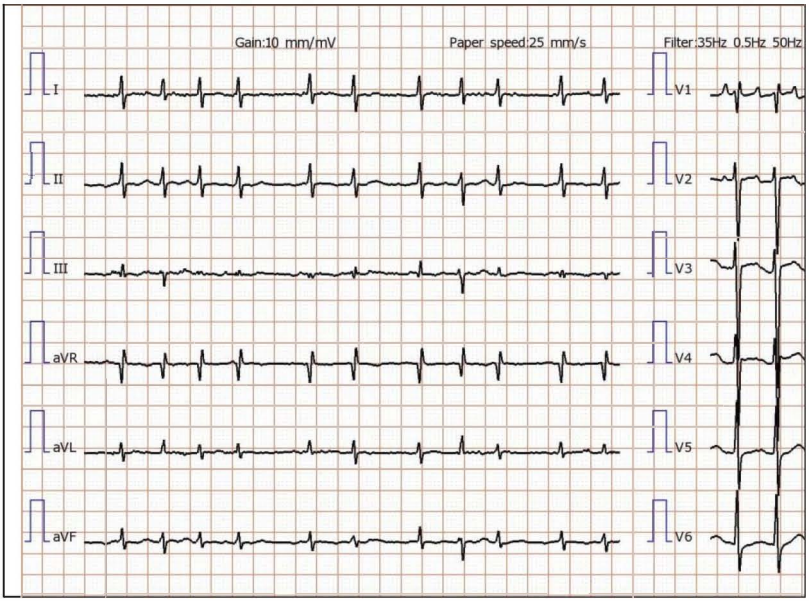
Table 2.17 Supraventricular Tachycardias

Short RP, long PR interval	Short PR, long RP interval
Typical AVNRT	Atrial tachycardia
AVRT	Sinus node re-entry tachycardia
Junctional tachycardia	Atypical AVNRT
Atrial tachycardia with first degree AV block	Junctional tachycardia
	AVRT with a slowly conducting accessory pathway (PJRT)

AVNRT: atrioventricular nodal reentry tachycardia; AVRT: atrioventricular reciprocating tachycardia; PJRT: paroxysmal junctional reciprocating tachycardia.

16. A 50-year-old presented to emergency department with palpitations. The first ECG (upper) was taken. After adenosine administration, the lower ECG was taken. What is your diagnosis?





A. Sinus tachycardia
B. Atrial tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Multifocal Atrial Tachycardia

Answer: B

Upper ECG showed a 1:1 AV association, and after the initiation of rate control, the lower ECG showed a varied AV conduction. In the lower ECG, AV conduction with aberrancy (RBBB morphology, Ashman phenomenon) is seen before the last beat.

Atrial tachycardia**:** During this tachycardia the ectopic P wave precedes the QRS complex. Based on the configuration of the P-wave parameters such as axis and width, the site of origin of an atrial tachycardia can be distinguished. The PR interval depends on the origin site of the P-wave formation and the AV nodal conduction rate. Also, the ratio between P waves and QRS complexes depends on the rate of abnormal atrial impulse formation and the AV nodal transmission characteristics (Table 17). The most common type of atrial tachycardia is paroxysmal (with sudden onset

and offset). The other type of atrial tachycardia is incessant (permanent), which has a persistent nature and is characterized by the inability to control the rate. This form of atrial tachycardia is more prone to develop into dilated cardiomyopathy (tachycardiomyopathy) (Figure 2.2).

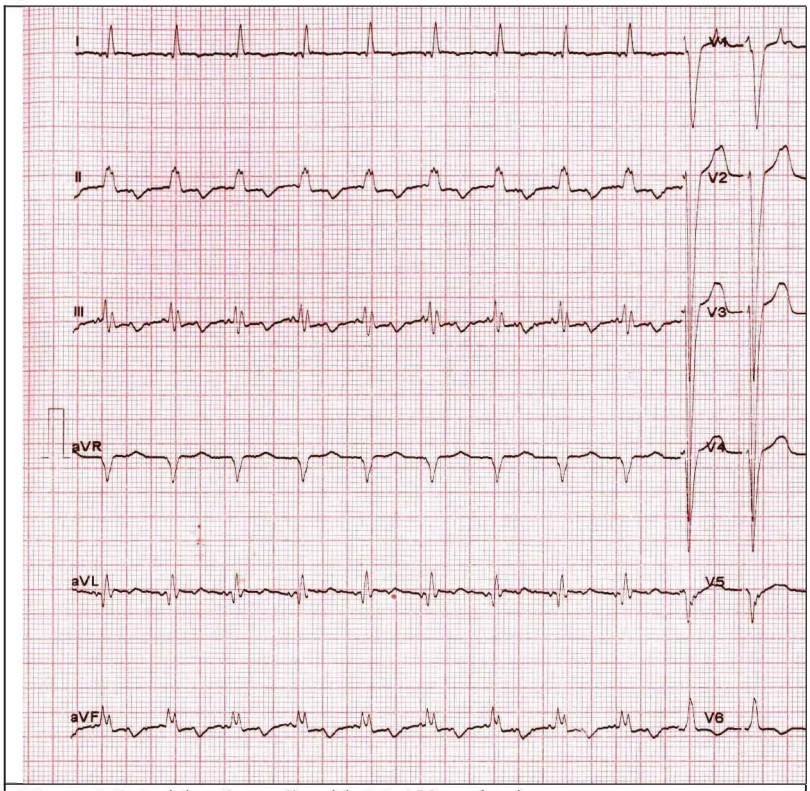


Figure 2.2. Atrial tachycardia with 2:1 AV conduction.

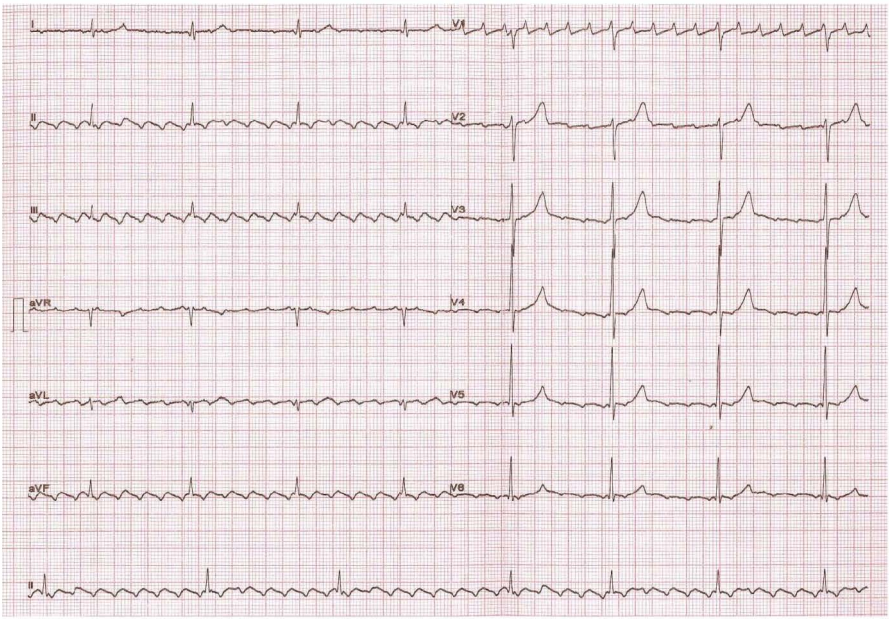
17. A 65-year-old man with a history of chronic obstructive pulmonary disease presented with palpitations. What is your diagnosis?

A. Sinus tachycardia
B. Atrial tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Multifocal Atrial Tachycardia

Answer: E

Chaotic (multifocal) Atrial Tachycardia**:** The main findings of this arrhythmia are: trial rates between 100 and 130 beats/minute, marked variation in P-wave morphology (at least three P-wave contours), totally irregular P-P intervals, and variable PR intervals. This arrhythmia is more common in older patients with chronic obstructive pulmonary disease and congestive heart failure. Some drugs, such as digitalis or theophylline, appear to be an unusual cause of this tachycardia. In this ECG, note that tachycardia is accomplished by more than three P-wave contours, irregular P-P intervals, and variable PR intervals.

18. What is your diagnosis?



A. Sinus tachycardia
B. Atrial tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Multifocal Atrial Tachycardia

Answer: C

Atrial flutter: This arrhythmia is a rapid form of atrial tachycardia sustained by a macroreentrant circuit. Atrial flutter usually occurs in the right atrium but occasionally in the left atrium. During the more common "typical" type, the flutter circulates in a counterclockwise direction around the tricuspid annulus in the frontal plane (counterclockwise flutter); however, during the less common (10%) "atypical" type, the macroreentry circuit rotates in a clockwise direction (down the intraatrial septum and up the right atrial free wall). Other forms of atrial flutter can be observed in the setting of incisional scars from previous atrial surgery, previous atrial ablation, mitral annular flutter, idiopathic fibrosis in areas of the atrium, or other anatomic or functional barriers to conduction in the atria.

In counterclockwise atrial flutter, the flutter waves are negative in leads II, III, aVF, and V6 and upright in V1 (Figures 2.3 and 2.4); whereas in clockwise atrial flutter, the flutter waves are upright in leads II, III, and aVF and often notched (Figure 2.5). In contrast to atrial tachycardia, lack of an isoelectric interval between flutter waves is seen because of evidence of continual electrical activity. The ventricular regularity and rate in this arrhythmia depend on AV transmission characteristics. If the AV conduction ratio is fixed, regular QRS complexes are seen. If irregular QRS complexes are seen, it indicates Wenckebach AV conduction (group beating) or variable AV conduction ratio (irregular with no fixed pattern).

In the ECG at question, note the flutter waves are negative in inferior leads and upright in V1, which is compatible with counterclockwise atrial flutter.

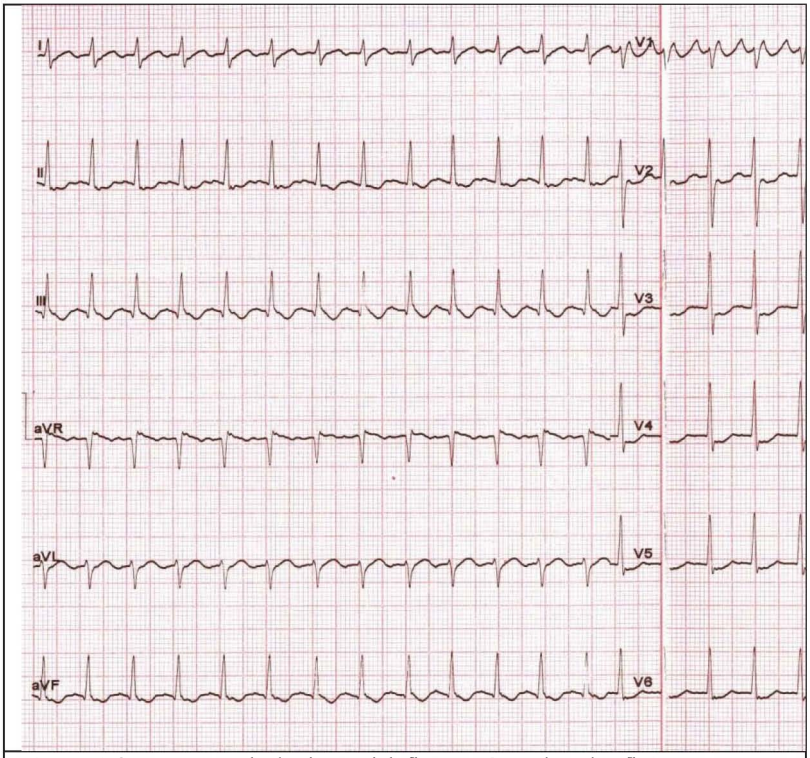


Figure 2.3 A counterclockwise atrial flutter. Note that the flutter waves are negative in inferior leads and upright in V1. The atrial rate is 300 beats/minutes and the ventricular rate is 150 beats/minutes (two-to-one AV conduction).

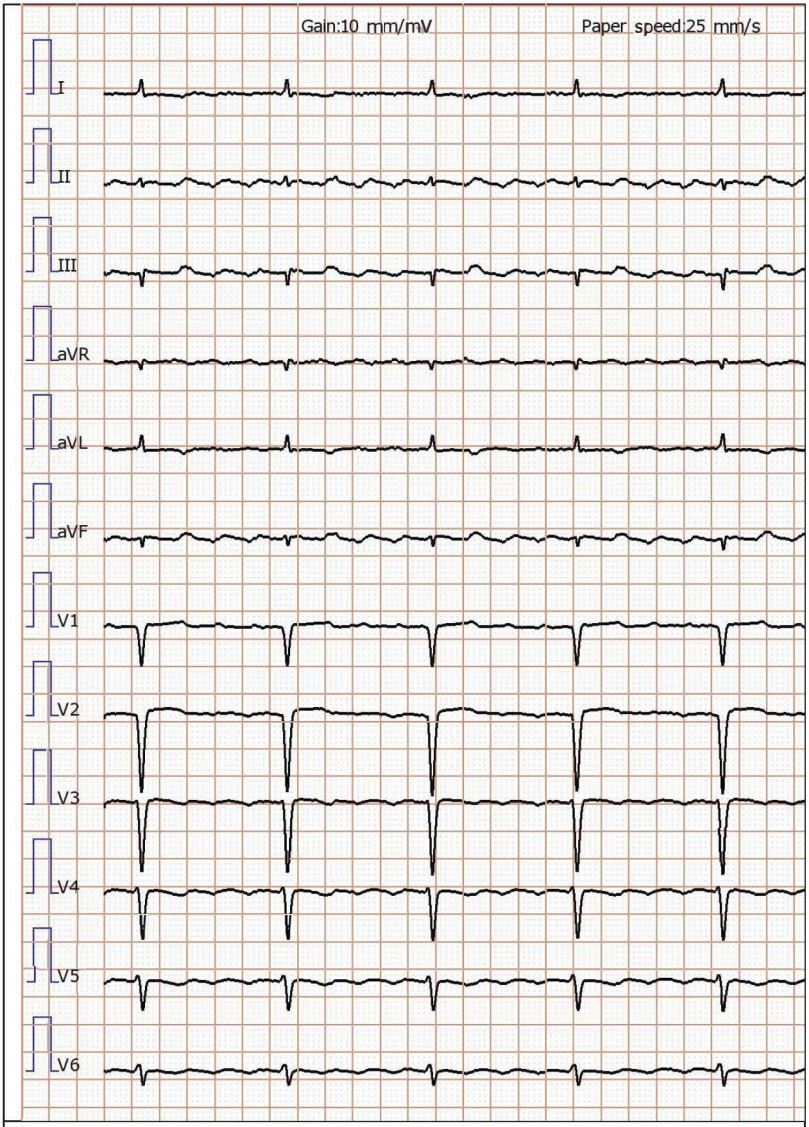


Figure 2.4 A counterclockwise atrial flutter (four-to-one AV conduction). Note that the flutter waves are negative in inferior leads and upright in V1.

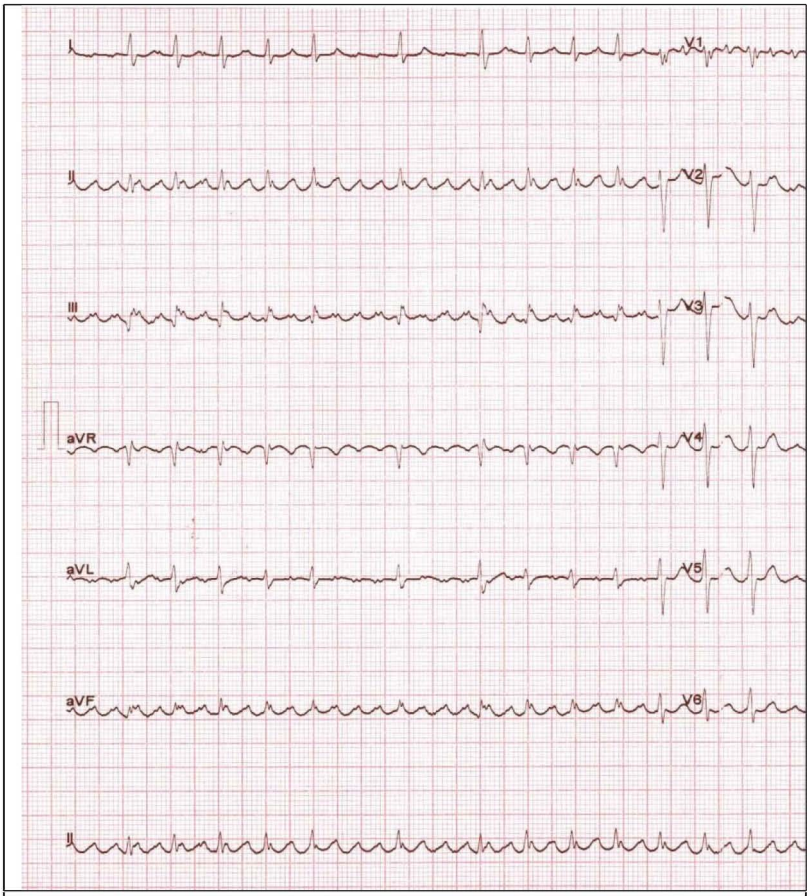
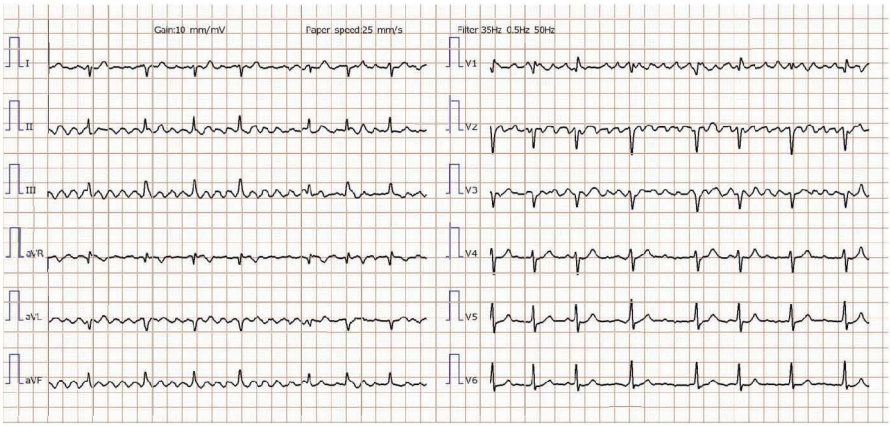


Figure 2.5 A clockwise atrial flutter with varied AV conduction. Note that the flutter waves are positive in inferior leads and negative in V1.

19. What is your diagnosis?



A. Sinus tachycardia
B. Atrial tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. Multifocal Atrial Tachycardia

Answer: D

Atrial fibrillation: This arrhythmia is considered the most common tachyarrhythmia. The fibrillatory or f waves of the atrium are variable in amplitude, shape, and timing, although in lead V1, f waves sometimes appear uniform and can mimic flutter waves (Figure 2.6 and 2.7). The ventricular rhythm is irregular unless complicated by a complete AV block or digitalis toxicity. However, if ventricular responses are very rapid (>170 beats/min), it may appear to be regular.

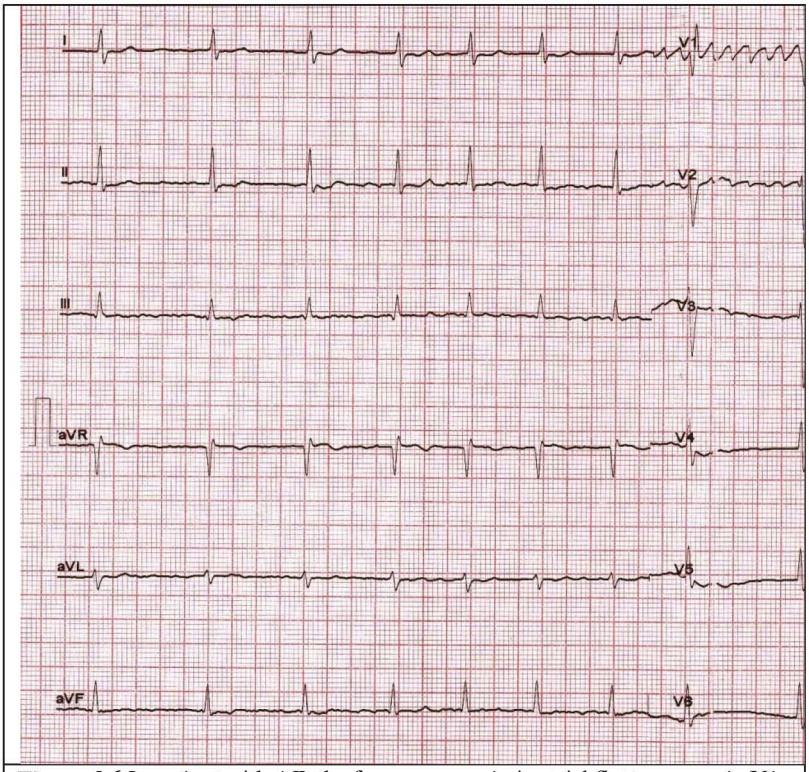


Figure 2.6 In patient with AF, the f waves can mimic atrial flutter waves in V1.

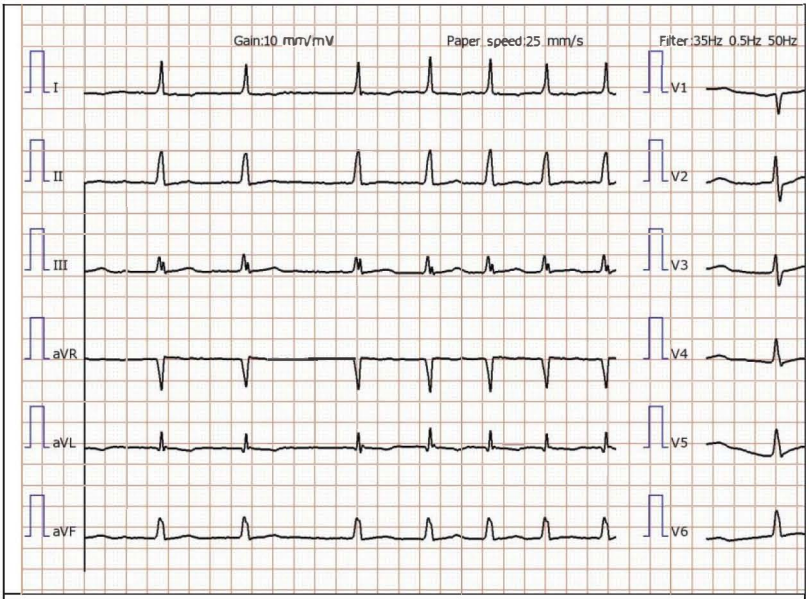
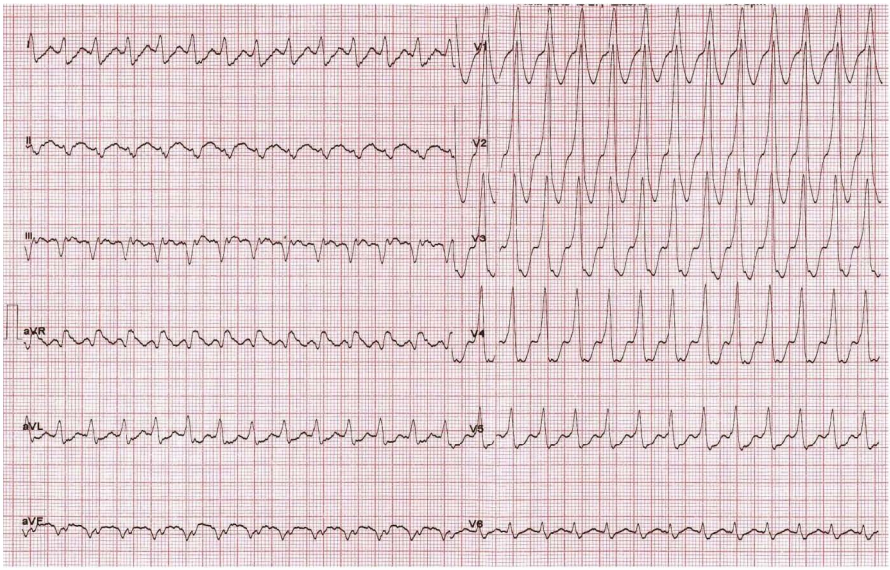


Figure 2.7 Fine f waves in patient with AF.

20. What is your diagnosis?



A. Sinus tachycardia
B. Atrial tachycardia
C. Atrial flutter
D. Atrial fibrillation
E. AV nodal reentry tachycardia
F. AV reciprocating tachycardia

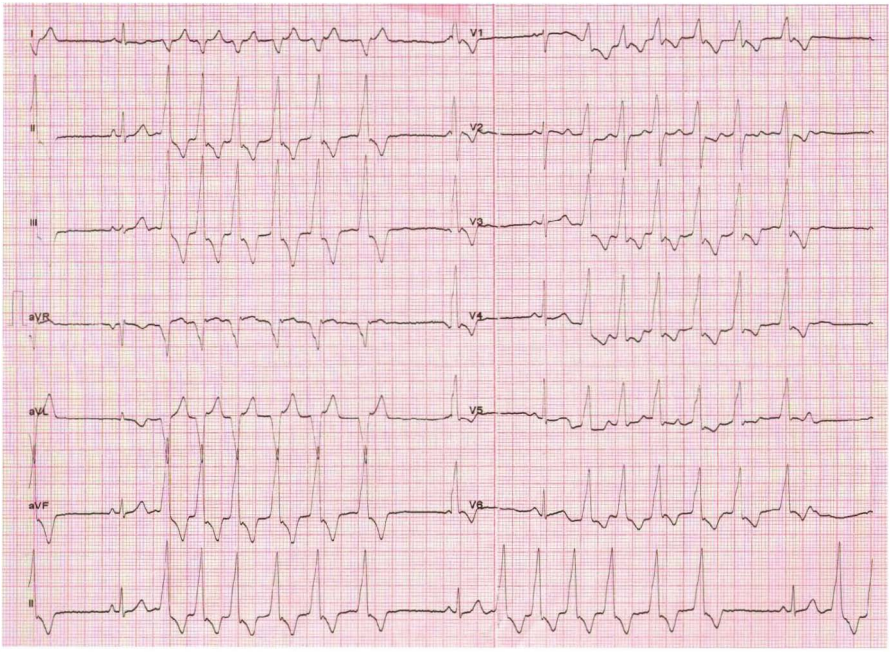
Answer: F

AV reciprocating tachycardia (AVRT): AVRT may result in narrow (orthodromic) or broad (antidromic) QRS tachycardia. The most common tachycardia is orthodromic AVRT characterized by: a normal QRS (<0.12 seconds unless bundle branch block is present), a regular rhythm, ventricular rates of 150 to 250 beats/minute, an RP interval less than the PR interval, and sudden onset and termination. In this tachycardia, the AV conduction is over the AV node and VA conduction is over the accessory pathway (concealed pathway). The morphology of the P wave depends on the location of the AP. The P wave is negative in leads I and aVL if reentry into the atrium is through a left lateral AP, and negative in leads II, III, and aVF if the pathway is posteroseptal. Depression of the ST segment during a

narrow-complex tachycardia generally signifies AVRT using an accessory pathway. Also, QRS alternans are often present, especially at high rates, in contrast to AVNRT.

In the ECG, note the regular wide QRS tachycardia. In differential diagnoses the VT should be considered.

21. What is your diagnosis?



A. Sinus tachycardia with aberrancy
B. Atrial tachycardia with aberrancy
C. Atrial flutter with aberrancy
D. Atrial fibrillation with aberrancy
E. Ventricular tachycardia

Answer: E

Ventricular tachycardia: AV dissociation during a wide QRS tachycardia is a hallmark of VT and present in approximately 60% of VTs. The other 40% show some form of retrograde conduction to the atria. The atrial rate during AV dissociation is usually slower than the ventricular rate, except when atrial tachycardia, atrial flutter, or atrial fibrillation are present. The capture and fusion beats and AV dissociation provide the strongest electrocardiographic evidence for the differentiation of VT from SVT with aberrant conduction. Major features in the differential diagnosis of wide QRS tachycardia are shown in Table 2.18.

In pericordial leads, the presence of a concordant pattern, no R/S pattern, and the onset of R to the nadir of S being longer than 100 ms favor a diagnosis of VT. Negative precordial concordance is almost always indicative of VT, whereas positive precordial concordance may be seen in SVT with AV conduction over an accessory pathway inserting into the posterobasal LV. As a rule, QR complexes during a wide QRS tachycardia are highly suggestive of VT, unless this pattern is present in the same leads during sinus rhythm. QR complexes during VT typically occur in patients with a localized pathology such as a ventricular scar secondary to previous MI, localized infiltrative, or inflammatory myocardial disease. However, a QRS width of more than 140 ms in RBBB and more than 160 ms in LBBB is suggestive of VT, but in some situations these patterns may result from an SVT, such as the presence of BBB with additional intramyocardial conduction delay, AV conduction over an accessory pathway, and consumption of antiarrhythmic drugs that prolong intraventricular conduction (Figure 2.8). If the QRS width during tachycardia is less than during a sinus rhythm, the diagnosis will be VT. In this situation, the VT originates closer to the interventricular septum, resulting in more simultaneous activation of both ventricles and a narrower QRS complex. Other electrocardiographic manifestations are shown in Table 2.18.

In this ECG, note the positive concordance in pericordial leads and the presence of an R/S ratio greater than one in lead V6 in the setting of the RAD, which is compatible with non-sustained VT with RBBB morphology during the sinus rhythm.

Table 2.18 Diagnosis of wide QRS tachycardia

RBBB-Shaped QRS Complex	
Favor SVT	Favor VT
Leads V1 and V6 usually have a triphasic pattern. Lead V1 has an rSR' pattern Lead V6 shows a qRS pattern In lead V6 the R/S ratio is typically greater than one	Lead V1 shows a monophasic or biphasic QRS complex (R, qR, QR, or RS). In lead V6 the presence of a deep S wave with an R/S ratio of less than one when LAD is present In lead V6 the R/S ratio is usually greater than one when RAD is present
LBBB-Shaped GRS Complex	
Favor SVT	Favor VT
If an r wave is present in either lead V1 or V2 it is narrow (less than 0.03 seconds) The downstroke of the S wave is fast, without slurring or notching The distance from the beginning of the QRS to the nadir of the S wave is 0.06 seconds or less.	Lead V1 or V2 has an R wave longer than 0.04 seconds in duration In lead V1 or V2 the downstroke of the S wave is slurred and delayed; the distance from the beginning of the QRS to the nadir of the S wave being more than 0.06 seconds A Q wave in lead V6

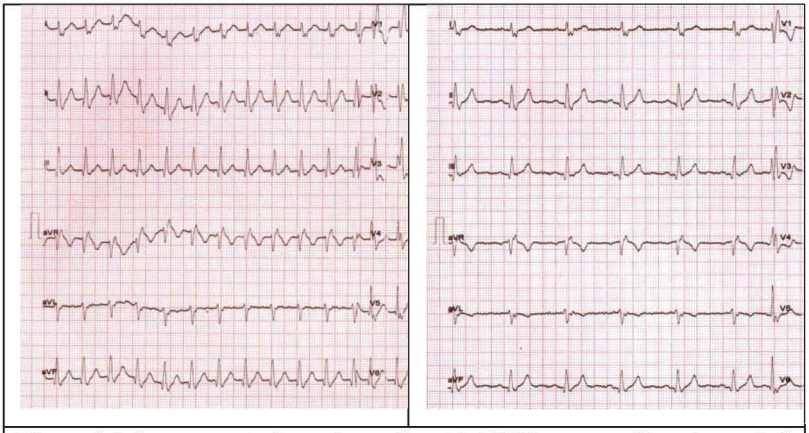
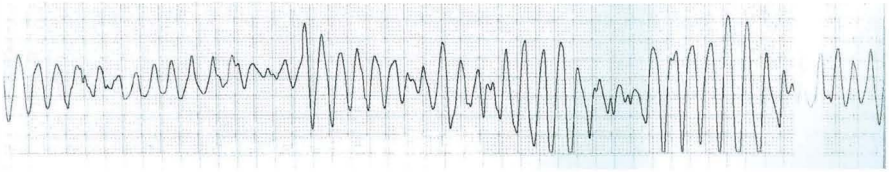


Figure 2.8 Supra-ventricular tachycardia with RBBB pattern. Note the typical RBBB during arrhythmia (left panel), same as during sinus rhythm.

22. What is your diagnosis?

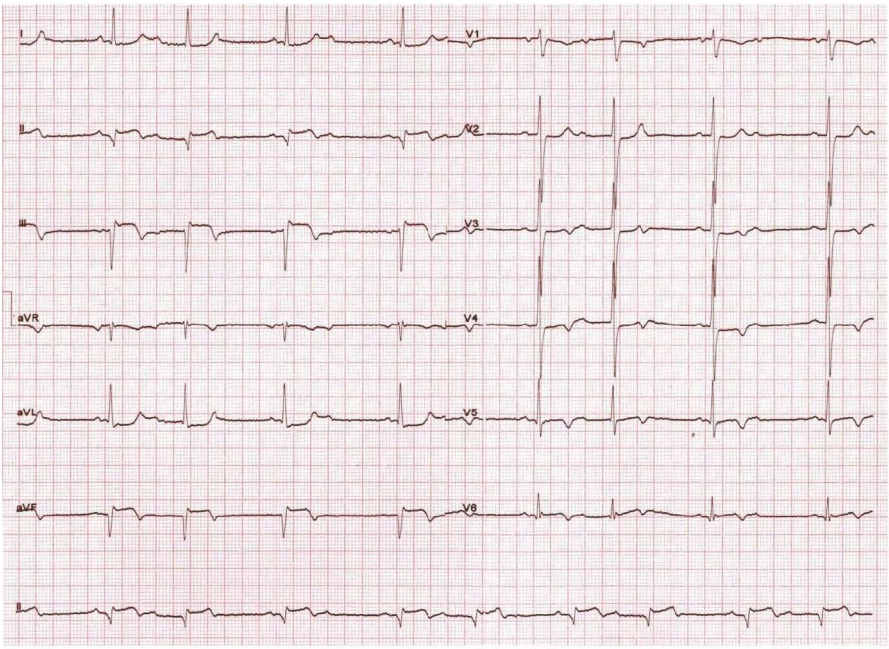


A. Ventricular flutter
B. Ventricular fibrillation
C. Ventricular tachycardia
D. Torsades de pointes

Answer: D

Torsades de pointes: TdP is a special form of polymorphic VT and hallmark characteristics of this rhythm are QRS complexes that rotate about the baseline, deflecting downward and upward for several beats. The rate is 200 to 250 beats/minute, originally described in the setting of a long QT interval, usually with an irregular rhythm, and the QRS complexes are wide with changing amplitude.

23. What is your diagnosis?



A. First-degree AV nodal block
B. Type I second-degree AV block
C. Type II second-degree AV block
D. Third-degree (Complete) AV block

Answer: B

Type I second-degree AV block (AV Wenkebach): The PR intervals progressively lengthen before the dropped beat and the RR intervals shorten. The largest increment in the PR interval is usually between the first and second PR interval and the pauses are less than twice the shortest cycle. Group beating is present and the QRS may be narrow or wide, depending on the presence or absence of bundle branch block (Figure 2.9).

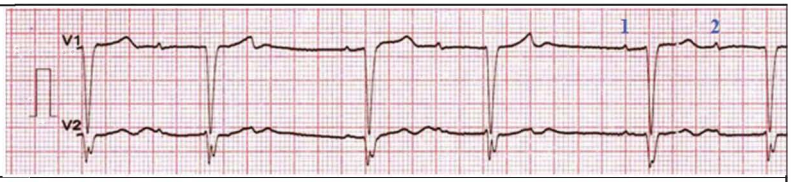


Figure 2.9 Type I second-degree AV block (Mobitz I). The PR intervals progressively lengthen (1st and 2nd P waves) before the dropped beat (3rd P wave) and the RR intervals shorten. Note the group beating.

24. What is your diagnosis?

A. First-degree AV nodal block
B. Type I second-degree AV block
C. Type II second-degree AV block
D. Non-conducted premature atrial complex

Answer: C

Type II second-degree AV block (Mobitz II): The PR interval remains normal or slightly prolonged before and after the blocked P wave. The ventricular rhythm is irregular because of the blocked P wave and the QRS duration is narrow when the lesion is within the His bundle and more than 0.12 seconds when the pathology involves the bundle branches (Figures 2.10, 2.11).

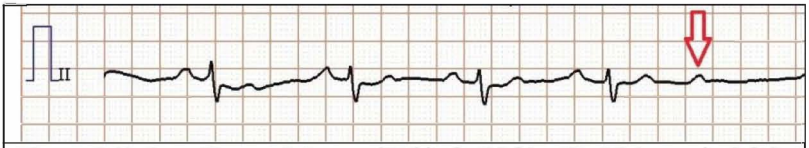


Figure 2.10 Type II second-degree AV block (Mobitz II). PR interval slightly prolonged (240 milliseconds), but remains sustained before and after the non-conducted P wave (red arrow).

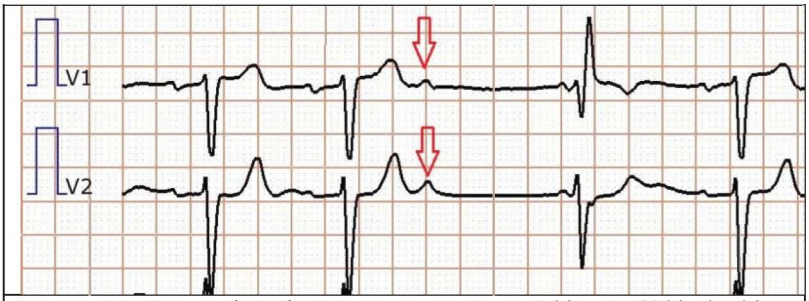
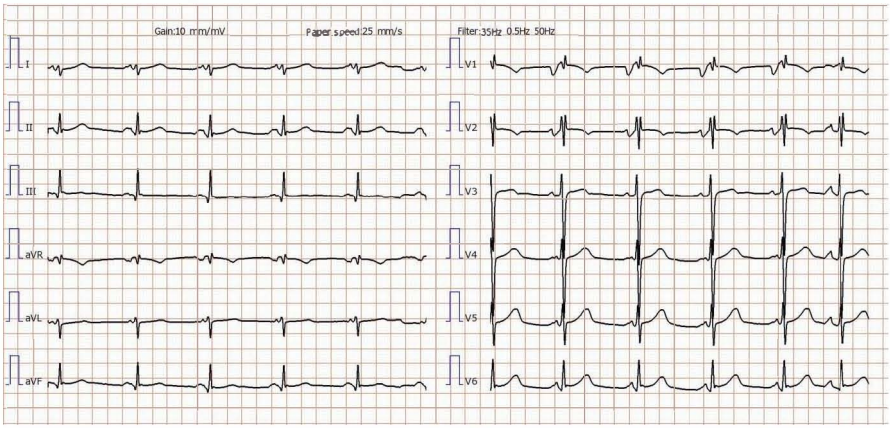


Figure 2.11 Non-conducted PAC. In contrast to a Mobitz II AV block, this P wave is premature and the morphology of this P wave is different from other P waves.

25. What is your diagnosis?



A. Aortic valve stenosis
B. Mitral valve stenosis
C. Tricuspid valve stenosis
D. Pulmonary valve stenosis

Answer: B

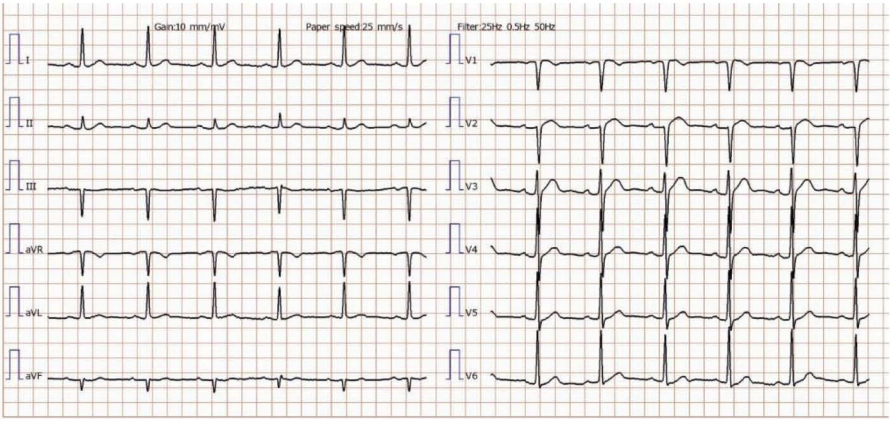
Mitral stenosis: Although the ECG is relatively insensitive for detecting mild MS, it does show characteristic changes in moderate or severe MS. Left atrial enlargement is a principal electrocardiographic feature of MS in patients with sinus rhythm. The left atrial enlargement correlates more closely with left atrial volume than with left atrial pressure. Also, it often regresses after successful percutaneous mitral commissurotomy. The major findings of significant MS are listed in Table 2.19.

In this ECG, note the LA abnormality, right axis QRS deviation, and incomplete RBBB.

Table 2.19 The electrocardiographic findings in moderate to severe Mitral stenosis

ECG change	Interpretation
Left atrial enlargement	In 90% of patients with sinus rhythm
Atrial fibrillation	Common in patients with longstanding MS
RV hypertrophy	Correlates with RV systolic pressure (in about 50% of patients with pressure of 70 to 100 mmHg, and in all patients with pressure greater than 100 mmHg)
Poor R-wave progression	

26. What is your diagnosis?



A. Hypercalcemia
B. Hypocalcemia
C. Hypernatremia
D. Hyponatremia

Answer: A

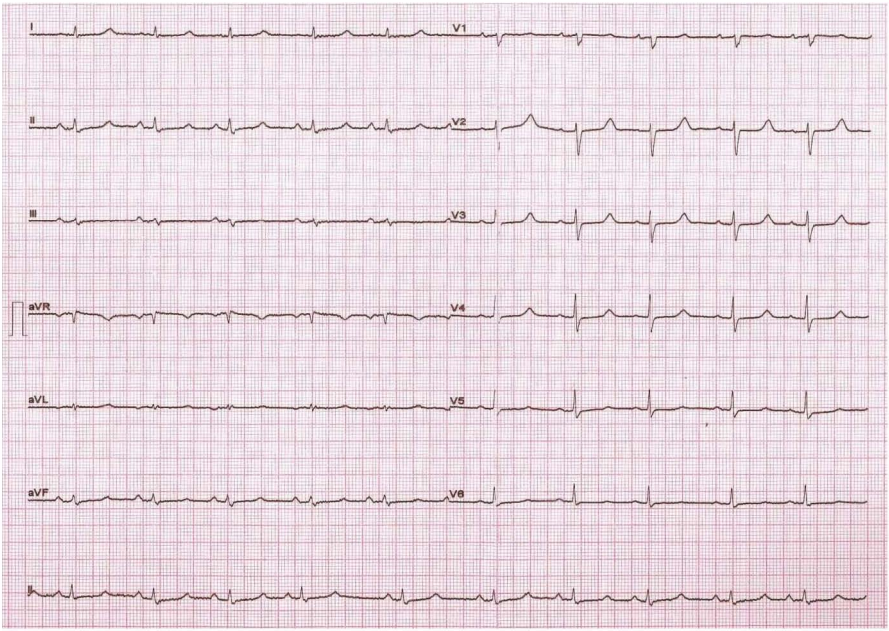
Hypercalcemia and hypocalcemia by causing change in the duration of phase 2 of the action potential could result in changes in the QT interval. Other ECG manifestations are listed in Table 2.20.

The ECG was taken from a patient with hypercalcemia (Ca: 14.5 mg/dl). Note the shortened QT interval and mild ST segment elevation in V1-2.

Table 2.20 The electrocardiographic findings in hypercalcemia or hypocalcemia.[13,27]

Hypercalcemia	Hypocalcemia
Short QT interval	Prolonged QT interval
Decreased T-wave amplitude	Flat ST segment
T-wave notching or inversion	Terminal T-wave inversion
High takeoff of the ST segment in leads V1 and V2 (simulate acute ischemia)	Heart block (in severe cases)
Prolonged P-R interval	Ventricular fibrillation (in severe cases)
Widened QRS complex	
Increased QRS voltage	
Notching of the terminal portion of the QRS complex from a prominent J wave	
AV block progressing to complete heart block and cardiac arrest (especially when serum calcium > 15 to 20 mg/dl)	

27. What is your diagnosis?

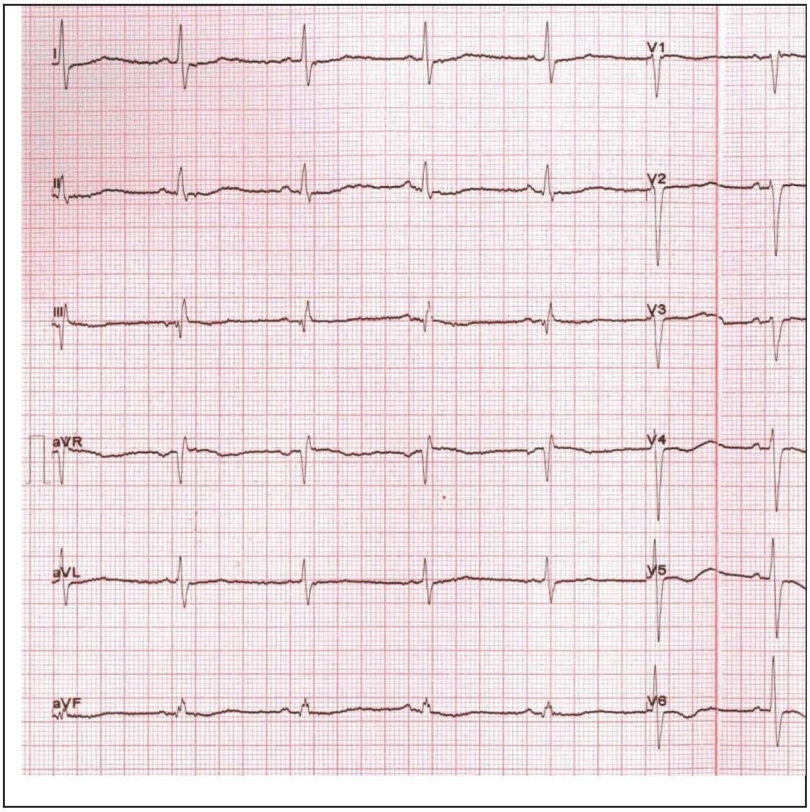


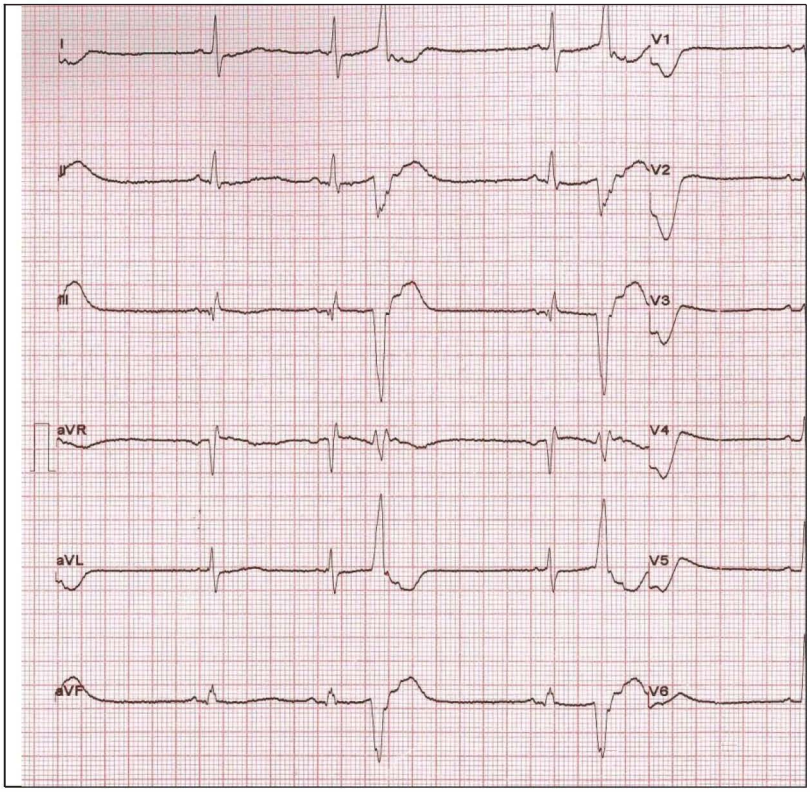
A. Hypercalcemia
B. Hypocalcemia
C. Hypernatremia
D. Hyponatremia

Answer: B

The ECG was taken from a patient with hypocalcemia (Ca: 6.5 mg/dl). Note the prolonged QT interval and flat ST segment.

28. These ECGs were both taken from the same patient: the first ECG is placed above and the second, taken an hour later, below. What is your diagnosis?





A. Hypercalcemia
B. Hypocalcemia
C. Hypokalemia
D. Hyperkalemia

Answer: C

Among the electrolyte disorders, hyperkalemia is the most fatal, and usually the severity of hyperkalemia parallels the changes in the ECG, although in some cases, the ECG findings may not consistently correlate with the level of serum potassium. The earliest effect of hyperkalemia is usually a narrowing and peaking of the T wave. This combination of hyperkalemia and hypocalcemia, which may lead to a shortened QT interval, is commonly seen in patients with chronic renal disease. Other changes are shown in Table 2.21.

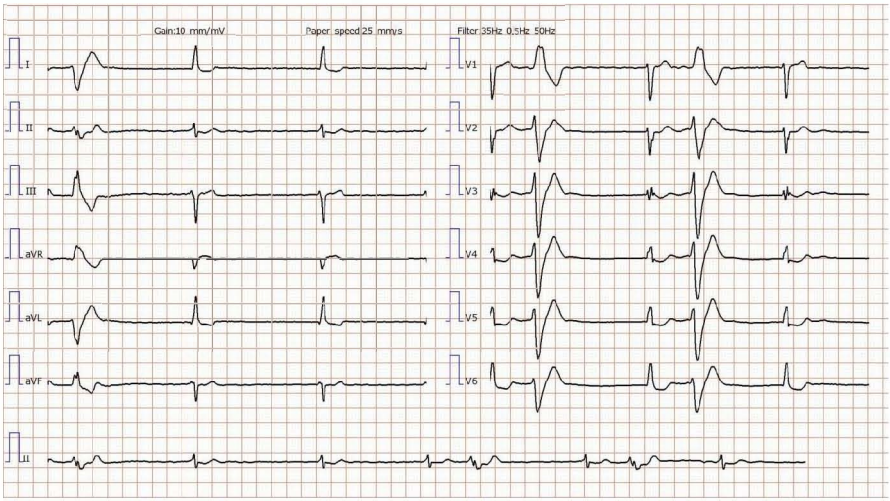
The most important ECG finding in hypokalemia is the presence of prominent U waves. In contrast to normal U waves, in a hypokalemia setting, the U wave becomes much larger than the T wave. When hypokalemia becomes more profound, the amplitude of the T wave lowers as the size of the U wave increases. Other indicators include nonspecific ST depression, prolongation of the QU interval, a slight prolongation in PR interval, and an increase in P-wave amplitude and duration. In severe hypokalemia cases, the increase in QRS amplitude and duration, fusion of the T and U waves, and ventricular arrhythmias, especially torsade de pointes or pulseless electrical activity, may occur.

These ECGs were taken from a patient with hypokalemia (serum level of potassium: 2.4 mg/dl). Note the ST segment depression, prominent U waves, diminished T-wave amplitude, fusion of the T and U waves, and prolonged QT (U) interval (upper ECG). In the lower ECG from the same patient, frequent short coupled PVC can be seen (R on T phenomenon). This patient is at high risk of developing torsade de pointes.

Table 2.21 ECG changes relating to the level of blood potassium.

Mild hyperkalemia (<6.0 mmol/l	Narrow-base T waves Peaking of the T waves (T waves are often taller than the R waves in precordial leads V2 to V4) The QT interval is normal or shortened
Moderate hyperkalemia (6.0 to 7.0 mmol/l)	More pronounced peaking of the T waves QRS complexes widen P waves become broader with diminished amplitude PR interval lengthens (AV block)
Severe hyperkalemia (>7.0 mmol/l)	P waves become unrecognizable Sinoventricular rhythm Further widening of the QRS complex S and T waves merge with a very short ST segment (sinusoidal wave) ST segment elevated in V1-2 Bradycardia Ventricular flutter/fibrillation Asystole

29. Which drug can cause these ECG changes?



A. Digoxin
B. Quinidine
C. Amiodarone
D. Disopyramide

Answer: A

The mechanism of arrhythmias resulting from digitalis toxicity may be attributed to a block in the conduction system (SA or AV nodal regions) or rapid impulse formation in the atrium, AV junction, and ventricular Purkinje system. The electrocardiographic effects of digoxin are shown in Table 2.22. These changes may be seen in normal serum levels of digoxin (Figure 12-13). The arrhythmias resulting from digoxin toxicity are listed in Table 2.23.

Some of the antiarrhythmic drugs affect patients' ECG, depending on excessive dosage and plasma concentrations. A QRS prolongation can be seen in patients who have taken class I antiarrhythmic agents (e.g., quinidine, procainamide, flecainide). Also, a QT(U) prolongation can be observed in those who have taken class IA (e.g., quinidine) and class III agents (e.g., amiodarone, dronedarone, dofetilide, ibutilide and sotalol). In patients receiving antiarrhythmic agents, proarrhythmic events can occur in

as many as 5 to 10%, through such developments as incessant VT, a long-QT syndrome, and torsades de pointes (Table 2.22). In contrast to other antiarrhythmic drugs, although ranolazine mildly increases QT interval, but does not have the electrophysiologic profile that leads to torsades de pointes. Even ranolazine has beneficial effects on the clinical electrophysiologic properties of ventricular and atrial arrhythmias. The electrocardiographical markers of some drugs are listed in Table 2.22.

This ECG was taken from a patient with digoxin toxicity (serum digoxin level: 2.4 ng/ml). Note the AF with slow ventricular response, the digitalis effect or scooped appearance of the ST segment, the ST segment elevation in lead aVR, and the diminished height of the T wave.

Table 2.22 The effects of drugs on ECG[13]

Drug	ECG effects
Digoxin	Concave ST-T depression in many ECG leads, especially leads II, III, aVF, and V4 to V6 (digitalis effect or scooped appearance). Digitalis related ST-T changes can be accentuated by an increased heart rate (false-positive results on stress testing) ST segment elevation in leads aVR and V1 Shortened QT interval. Diminished height of T wave Usually U waves are present.
Class IA agents	QRS prolongation QT(U) prolongation Torsades de pointes
Class IC agents	QRS prolongation QT(U) prolongation Torsades de pointes
Class III agents	QRS prolongation (is seen in Amiodarone and Dronedarone) QT(U) prolongation Torsades de pointes

Tricyclic antidepressants	RAD of the terminal 40 ms frontal plane QRS axis Terminal R wave > 3 mm in aVR R/S ratio > 0.7 in aVR QRS prolongation QT(U) prolongation Torsades de pointes Asystole
Phenothiazines	QRS prolongation QT(U) prolongation Torsades de pointes
Methadone	QT prolongation
Cocaine	Sinus tachycardia Sinus bradycardia Supraventricular tachycardia Bundle branch block Complete heart block QRS prolongation QT(U) prolongation Accelerated idioventricular rhythm Ventricular tachycardia Ventricular fibrillation Asystole Torsades de pointes Brugada pattern (right bundle branch block with ST segment elevation in leads V1, V2, and V3) ST segment elevation

Table 2.23 Arrhythmias resulting from digitalis toxicity[23]

Slow Arrhythmia	
Arrhythmia	Description
Sinus Bradycardia and SA Block	Sinus bradycardia Sinus arrest SA Wenckebach conduction
AV Nodal Block	Prolonged PR interval AV nodal Wenckebach conduction Complete AV nodal block

Rapid Arrhythmia	
Arrhythmia	Description
Atrial Tachycardia with AV Nodal Block	Atrial rate is 130 to 250 beats/min. Two-to-one AV block or AV Wenckebach conduction P axis usually directed inferiorly Ventriculophasic PP alternation often present.
AV Junctional Tachycardia	Ventricular rate is 70 to 140 beats/min. Rhythm is non-paroxysmal. The rate increases with exercise AV dissociation is usually present.
Fascicular VT	Ventricular rate is 90 to 160 beats/min. QRS duration is 0.12 to 0.14 seconds. The QRS usually has an RBBB shape. Right- or left-axis deviation of the QRS is usually present.
Bifascicular (Bidirectional) VT	Ventricular rate is 90 to 160 beats/min. QRS has an RBBB shape. QRS duration is 0.12 to 0.14 seconds. QRS axis alternates from right to left
Ventricular Bigeminy	Bigeminal QRS complexes are polymorphic (in contrast to coronary artery disease, in which the bigeminal QRS complexes would be monomorphic)
Atrial Fibrillation with regular ventricular rhythm	Possible Causes of regular ventricular rhythm: 1. Complete AV block with a junctional escape rhythm 2. Complete AV block with junctional tachycardia 3. Complete AV block with fascicular VT
Atrial Fibrillation with group beating of ventricular rhythm	Possible causes of group beating of ventricular rhythm: AV nodal Wenckebach conduction
Atrial Flutter	Atrial flutter with complete AV block and a junctional escape rhythm Atrial flutter with a junctional tachycardia and AV dissociation Atrial flutter with fascicular VT

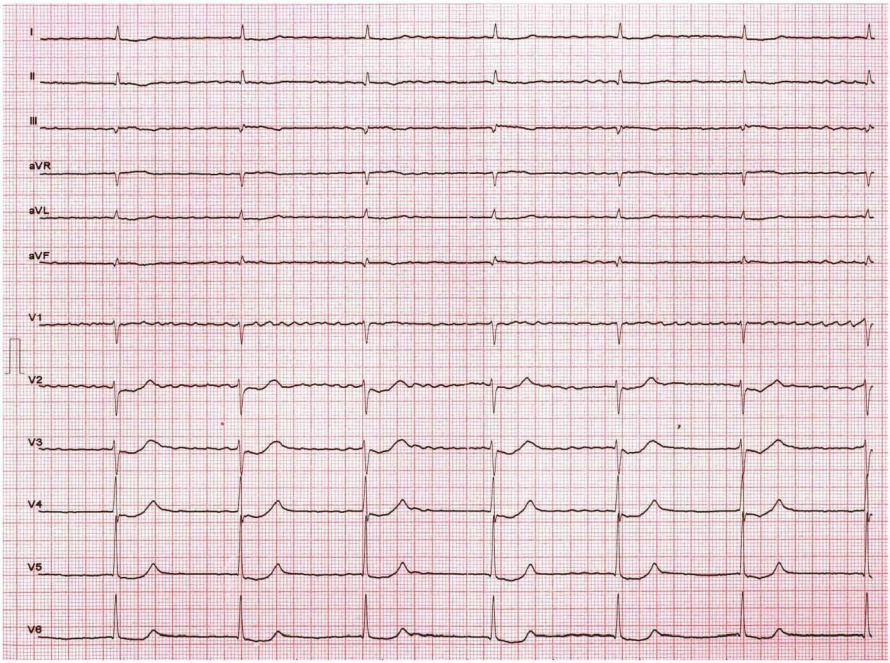


Figure 2.12 ECG of a patient with digoxin toxicity. Note the AF accomplishment with junctional rhythm (complete AV nodal block), digitalis effect or scooped appearance ST segment, mild ST segment elevation in lead aVR, and the diminished height of the T wave.

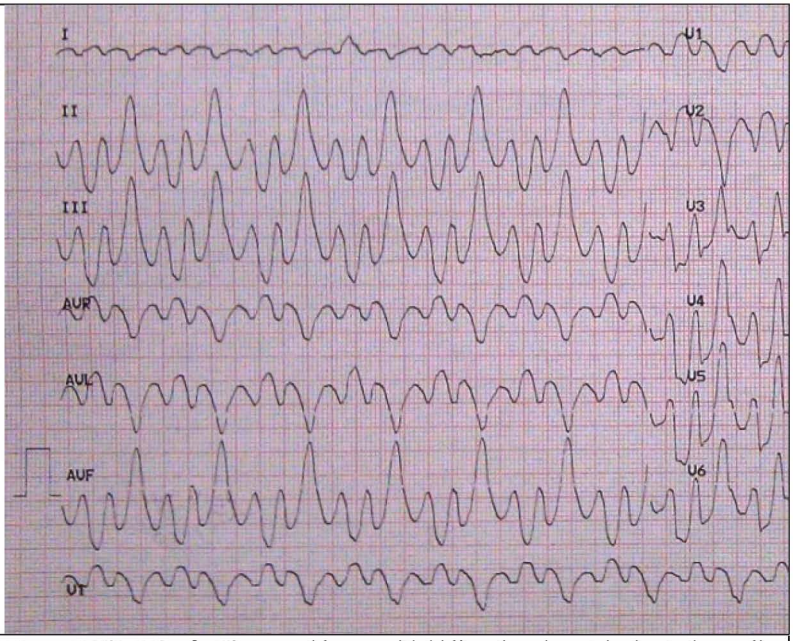
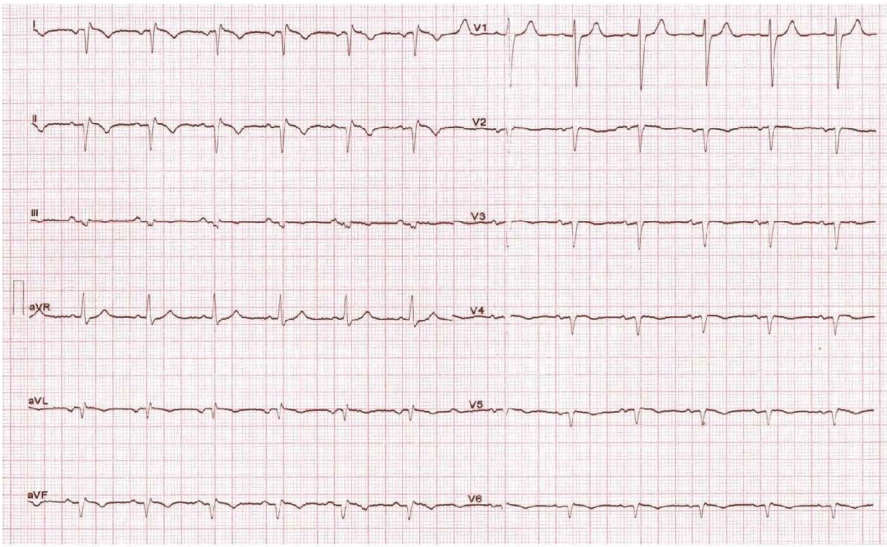


Figure 2.13 ECG of a 59-year-old man with bidirectional ventricular tachycardia in the setting of digoxin toxicity (serum digoxin level: 2.9 ng/ml). Note the regular wide QRS complex tachycardia with alternating LBBB and RBBB pattern.

30. What is your diagnosis?



A. Chest lead reversals
B. Bilateral arm-leg lead reversal
C. Right arm and left leg reversal
D. Dextrocardia

Answer: D

The most common errors during ECG recording are the placement of V1 and V2 electrodes in the 2nd or 3rd rather than in the 4th intercostal space, and the placement of V4 to V6 electrodes too high on the lateral chest. In healthy subjects, the P wave in V1 is positive or biphasic, and if an exclusively negative P wave is seen in V1, it indicates that this lead is recording in the highest placement. Also, chest lead reversals result in inappropriate R wave progression (which is normally seen in patients with dextrocardia).

This ECG was taken from a patient with dextrocardia. Note the right axis deviation, the inversion of all complexes in lead I (negative P, QRS, and T waves), the positive QRS complexes (with upright P and T waves) in aVR, and the reversal progression of the R wave in pericordial leads.

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CHAPTER 3

EXERCISE STRESS TESTING

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1. Exercise testing is appropriate in all the following conditions except:

A. when evaluating response to medical or ablation therapy in patients with known exercise-induced ventricular arrhythmias.
B. in patients with calf pain and borderline ankle-brachial indices.
C. routine screening of asymptomatic diabetic patients with normal resting ECG.
D. when assessing rate-adaptive Pacemaker Function in patients involved in heavy physical activities.
E. In symptomatic patients with preexcitation.

Answer: C

Exercise testing is a useful tool in evaluating the adequacy of response to medical or ablative therapy, particularly arrhythmias induced by exercise, including exercise-induced ventricular tachyarrhythmias (VT) and catecholaminergic polymorphic VT.

In patients with peripheral vascular disease, exercise treadmill tests are recommended to provide objective evidence on the level of functional limitation and to determine the response to therapy. A post-exercise ABI (ankle-brachial index) is also useful, and is performed at rest in both ankles and immediately post-exercise. During leg exercise, due to peripheral vasodilation, systolic blood pressure decreases in the ankles. This leads to a

mild decrease in ABI in healthy patients, with normalization in 1 to 2 minutes in recovery.

In patients with peripheral artery disease (PAD), the pressure drop and decrease in ABI in the ankle is greater, with a longer recovery time. Findings typical of patients with PAD include a post-exercise ABI <0.9, a >5% drop in post-exercise ABI, a >30 mmHg drop in systolic BP at the ankle, and a recovery time of more than three minutes.

Currently there is no adequate evidence for recommending exercise testing for routine screening of asymptomatic diabetic patients. The American Diabetes Association does not recommend routine screenings in asymptomatic patients, even before the start of an exercise training program, because there is no evidence that it would improve outcomes.

Exercise testing could be used in assessing rate-adaptive pacemaker function, especially when exercise intolerance is not resolved after adequate pacemaker adjustments. This is also useful in those who wish to participate in athletic or heavy exercise.

In symptomatic patients with preexcitation, abrupt loss of conduction over a pathway during exercise testing in sinus rhythm is used to identify patients with a low risk of developing rapid conduction and life-threatening ventricular arrhythmias if atrial fibrillation happens.

2. Exercise testing is absolutely contraindicated in patients with which of the following conditions?

A. Known left main coronary artery disease.
B. Acquired complete heart block.
C. Hypertrophic obstructive cardiomyopathy with significant gradient at rest.
D. Active endocarditis.
E. Moderate aortic stenosis with unclear relation to symptoms.

Answer: D

Absolute contraindications of EST include an acute phase of myocardial infarction or unstable angina, uncontrolled arrhythmias, hemodynamic instability, active endocarditis, acute myocarditis or pericarditis, symptomatic

severe aortic stenosis, decompensated heart failure, and acute pulmonary embolism.

Relative contraindications include left main coronary artery disease, moderate aortic stenosis with no clear association with patient's symptoms, an uncontrolled rate of tachyarrhythmias, acquired complete heart block, hypertrophic cardiomyopathy with remarkable resting gradients, and the patient's inability to cooperate.

3. Which sentence best defines One metabolic equivalent (MET)?

A. Total body oxygen uptake during exercise.
B. Peak oxygen uptake achieved during the highest level of dynamic exercise.
C. Resting oxygen expenditure.
D. Maximum arteriovenous oxygen difference.
E. Oxygen expenditure during symptom-limited exercise

Answer: C

Most of the energy needed for muscle work is derived from oxidative pathways to produce adenosine triphosphate; therefore, energy requirements can be estimated by measuring the total body oxygen uptake (VO2). VO2 can be defined in multiples of resting oxygen requirements or metabolic equivalents (METs]. One MET is equal to the amount of oxygen consumed whilst at rest and is approximately 3.5 ml O2/kg body weight x min.

Maximum VO2 (VO2max) is the highest oxygen uptake during the performance of the highest level of dynamic exercise. VO2 peak is the level reached during maximally tolerated symptom-limited exercise and is used during routine exercise testing.

Based on the Fick equation, VO2 is equal to cardiac output multiplied by oxygen extraction (expressed in terms of arteriovenous oxygen difference) and can increase up to three times, with a maximum limit of 15-17ml of oxygen/100ml of blood.

4. All the following factors influence myocardial oxygen demand except:

A. heart rate
B. blood pressure
C. left ventricular contractility
D. left ventricular wall stress
E. hemoglobin levels

Answer: E

Myocardial ischemia is the result of the blood's oxygen supply being inadequate to meet the demands of the myocardium. Myocardial oxygen demand is influenced by heart rate, levels of blood pressure, left ventricular contractility, and left ventricular wall stress. Wall stress is affected by left ventricular cavity size and intra-cavitary pressures, as well as wall thickness. The rate-pressure product (the product of heart rate and systolic blood pressure at each level of exercise) is a good indicator of myocardial demand and is easily measured clinically.

Myocardial oxygen supply is increased during exercise by coronary vasodilation related to neurohormonal stimulations. However, this mechanism becomes impaired in atherosclerotic vessels. Hemoglobin levels also influence the myocardial oxygen supply, with significant or acute anemia impairing the oxygen supply, with ensuing ischemia.

5. Regarding endurance and resistance exercise, all are correct except:

A. During endurance exercise there is a greater increase in cardiac output compared to resistance exercise.
B. Diastolic pressure tends to increase with resistance exercise and fall during endurance training.
C. During endurance exercise there is a greater increase in venous return compared to during resistance exercise.
D. Dynamic arm exercise results in a greater increase in heart rate and blood pressure than dynamic leg exercise.
E. During resistance exercise there is a considerable decrease in peripheral vascular resistance.

Answer: E

In endurance exercise, such as running, cardiac output rises due to increases in sympathetic tone, heart rate, and ventricular contractility. There are also increases in the venous return, leading to increased incidence of stroke. Metabolite accumulation causes vasodilation of muscle arterioles and lowers cardiac afterload which results in better systolic emptying. Systolic blood pressure increases but diastolic blood pressure falls or remains the same because of decreased vascular resistance.

Exercise has different hemodynamic effects on different working muscle groups; for example, dynamic arm exercise results in a greater increase in heart rate and blood pressure than dynamic leg exercise.

During resistance exercise, such as weightlifting, there is an increase in sympathetic activity and heart rate, but due to pressure on working muscle capillaries there is a rise in peripheral vascular resistance and diastolic blood pressure, as well as systolic blood pressure. There is less increase in venous return and cardiac output compared to endurance exercise.

6. Regarding submaximal exercise testing, all are correct except:

A. It is safe to perform early after myocardial infarction before hospital discharge.
B. It is often defined as reaching 70% of the maximal predicted heart rate.
C. It is used to provide prognostic information and guide subsequent therapy.
D. It is utilized to determine allowed levels of daily activity after hospital discharge.
E. The patient is expected to reach a goal of about 10 METs.

Answers: E

A submaximal exercise test is often performed early after myocardial infarction and before the patient is discharged from hospital. It is a safe and reliable method to determine prognosis and optimal therapeutic management. Submax. tests are also useful to determine safe levels of daily activity and plan for a future cardiac rehabilitation program. The endpoint is reaching 70% of maximal predicted heart rate, a peak heart rate of 120 beats/min, or 5METs.

7. Regarding cardiopulmonary exercise testing, all are correct except:

A. It uses ventilatory gas exchange analysis and is more reproducible than routine exercise tests.
B. Cardiopulmonary exercise testing, where available, is preferable and should be performed instead of routine exercise tests.
C. It is useful before cardiac transplantation.
D. It helps to differentiate pulmonary causes of dyspnea from cardiac etiologies.
E. It is useful for evaluating responses to therapy

Answer: A

Cardiopulmonary Exercise Testing (CPX) or exercise testing with ventilator gas exchange analysis provides a more accurate and reproducible method to measure VO2.

Measurements include VO2, VCO2 and minute ventilation. Despite the advantages, there is no need to combine CPX for all patients and in every exercise test. CPX is best used:

i. for heart failure patients to determine functional capacity, prognosis, and response to therapy, or before cardiac transplantation;
ii. to differentiate pulmonary and cardiac causes of limitation in functional capacity;
iii. in clinical situations such as pulmonary hypertension, airway diseases, mitochondrial myopathies, hypertrophic cardiomyopathy, and preoperative evaluations.

8. Exercise testing has a role in evaluating patients with the following congenital heart diseases except:

A. Coarctation of aorta
B. Ebstein anomaly
C. Coronary anomalies
D. Repaired tetralogy of Fallot
E. All the above

Answer: E

Exercise testing is useful in many congenital heart diseases for functional and rhythm disturbance assessments. In patients with coarctation of the aorta and after coarctation repair, an exercise test can be used to reveal exercise-induced hypertension. In patients with Ebstein anomaly and after the repair of tetralogy of Fallot with pulmonary regurgitation, exercise limitation favors surgical correction. Exercise tests also play a role after the correction of coronary anomalies, albeit in combination with imaging methods which offer greater precision. Exercise testing is useful in the assessment of patients with congenital heart disease who wish to participate in athletic activities.

9. Regarding exercise testing in patients with atrial fibrillation, all are correct except:

A. Exercise testing is useful when the initiation of type IC antiarrhythmic drug therapy is considered and myocardial ischemia is suspected.
B. Exercise testing is used for evaluation of the efficacy of the heart rate control during activity in patients with persistent or permanent AF
C. The criterion for adequate heart rate control in patients with atrial fibrillation roughly rates between 90 to 115 beats/min during moderate intensity exercise.
D. Exercise testing can be used to detect exercise-induced atrial fibrillation
E. Exercise testing should not be performed before initiation of medical therapy for heart rate control.

Answer: E

According to atrial fibrillation guidelines, an exercise test should be used before the initiation of type IC antiarrhythmic drugs to rule out underlying myocardial ischemia, to evaluate the efficacy of heart rate control during maximal activity in patients with persistent or permanent AF, and to induce activity related to atrial fibrillation. The criteria for adequate heart rate control varies with the patient's age, but ventricular rates between 90 and 115 beats/min during moderate exercise are generally acceptable.

10. All of the following are associated with high risk exercise test in women except:

A. Exercise capacity of <5 METs.
B. Heart rate recovery of ≤12 beats/min one minute after recovery in upright cool-down protocols.
C. Decrease in systolic blood pressure of >10 mmHg compared to rest.
D. Duke treadmill score of 2.
E. ST-segment elevation ≥2 mm in non-q–wave lead.

Answer: D

In premenopausal women with atypical symptoms and one or no coronary risk factors, the rate of false-positive exercise tests is high. An exercise test is best used for intermediate-risk symptomatic women with a normal resting ECG.

Factors which make an exercise test high risk for the patient include:

i. functional capacity of less than 5 METs or <100% age-predicted METs, calculated as $14.7 - (0.13 \times \text{age})$;
ii. abnormal heart rate recovery;
iii. ST-segment depression ≥2 mm;
iv. ST-segment depression ≥1 mm at <5 METs or >5 min into recovery;
v. ST-segment elevation ≥2 mm (in non q-wave leads but not in aVR);
vi. Duke treadmill score of −11 or less;
vii. a decrease in SBP >10 mmHg from rest in the absence of vasovagal reactions and sustained ventricular tachycardia.

The Duke Treadmill Prognostic Score is a highly validated score for risk determination and is calculated by the following equation:

Duke Treadmill Score = Exercise time − (5 × ST deviation) − (4 × angina index)

The greatest ST deviation in any lead except aVR is considered. The angina index score is calculated as 1 for non-limiting angina and 2 for exercise-limiting angina.

Abnormal heart rate recovery is ≤12 beats/min reduction in heart rate one minute after recovery in upright cool-down protocols, or less than 18 beats/min after one minute when the activity is immediately stopped and less than 22 beats/min after 2 minutes of recovery. It has prognostic significance and is related to increased mortality.

References

1. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.
2. Maleki M et al., Practical Cardiology. Elsevier Health Sciences; 2017.

CHAPTER 4

ECHOCARDIOGRAPHY

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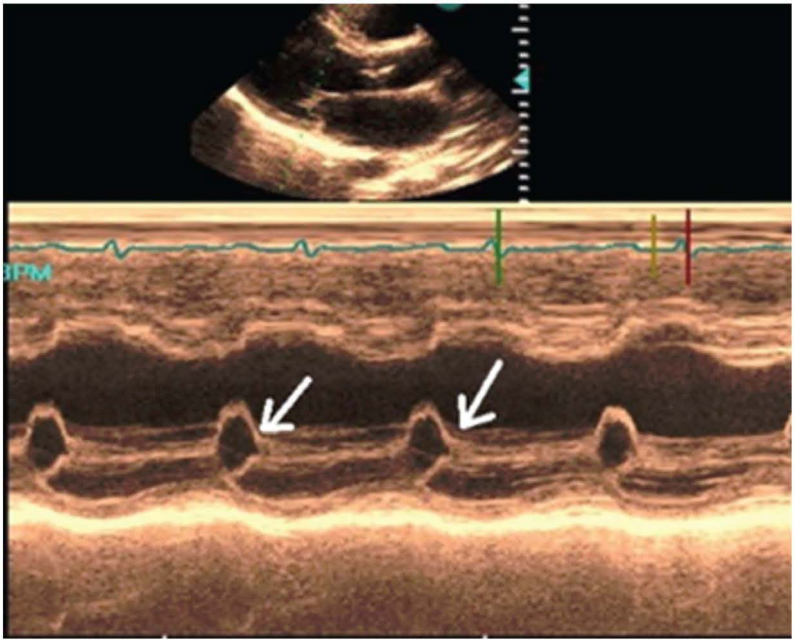
1. Which of the following sentences is not correct regarding myocardial tissue Doppler imaging (TDI)?

A. Tissue Doppler imaging (TDI) is an echocardiography mode that detects high-velocity low-amplitude signals from the myocardium.
B. A sample volume should be placed in the myocardium or valvular annulus.
C. TDI defines the velocity and direction of the myocardium.
D. Doppler-derived strain, and strain rate improve the evaluation of myocardial mechanics.

Answer: A

Tissue Doppler Imaging (TDI) is an echocardiography mode that detects low-velocity, high-amplitude myocardial velocity rather than blood cells that are high velocity and low amplitude. TDI defines the velocity and direction of the myocardium. A sample volume, such as the pulsed wave (PW), is placed in the myocardium or valvular annulus to get a quantifiable spectral contour of myocardial motion. Doppler-derived tissue velocity, strain, and strain rate have been proven to improve the evaluation of myocardial mechanics compared with previous indicators such as wall thickening or motion.

2. Based on the following M-mode study of the mitral valve in a parasternal long axis view in a patient with a history of fever and recent shortness of breath, which of the following diagnoses is most probable?



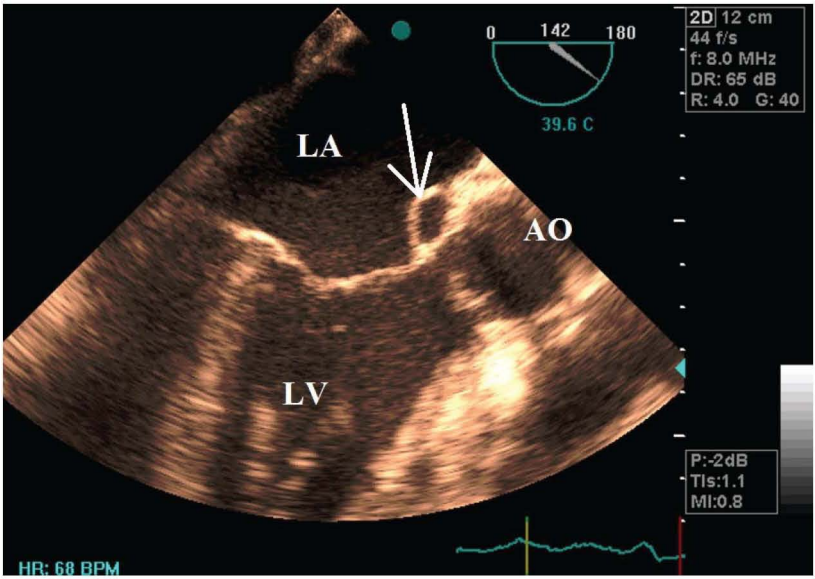
A. Rheumatic mitral stenosis
B. Early closure mitral valve
C. Mass on the mitral valve with thickened leaflets
D. Normal mitral valve M-mode study

Answer: B

The M-mode study shows premature closure of the mitral valve in acute severe aortic regurgitation, most probably due to destructed aortic leaflet and infective endocarditis.

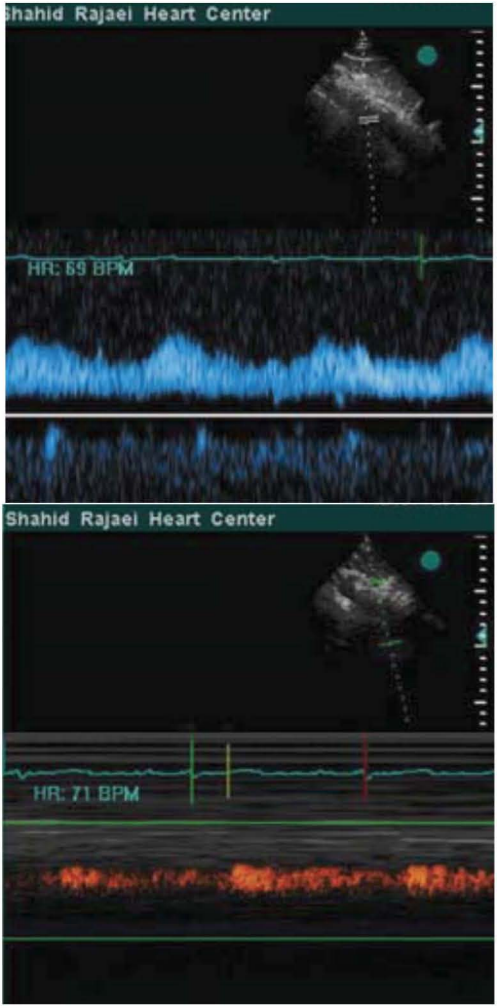
3. The arrow in the following long axis view of a transesophageal echocardiography shows:

A. A cyst in the anterior mitral leaflet
B. Abscess formation in the mitral valve
C. Transverse sinus
D. Pseudoaneurysm of the mitral aortic intervalvular fibrosa



Answer: D

4. The following pulse wave and Color M-mode Doppler studies of the abdominal aorta are suggestive of:

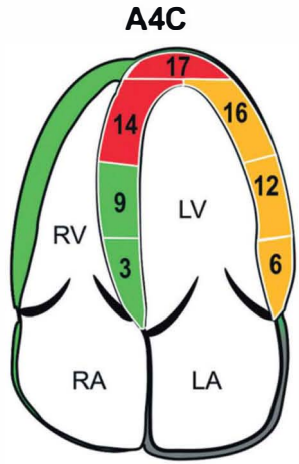


A. PDA
B. Coarctation of the aorta
C. Ruptured sinus of valsalva aneurysm
D. Normal abdominal flow pattern

Answer: B

5. Usually, which coronary artery supplies segments 3 and 6?

A. Segment 3 is supplied by RCA and segment 6 is supplied by LCX
B. Segment 3 is supplied by LCX and segment 6 is supplied by LAD
C. Segment 3 is supplied by LCX and segment 6 is supplied by RCA
D. Segment 3 is supplied by LAD and segment 6 is supplied by RCA



Answer: A

6. A 70-year-old female who has had DOE FC II for a few months ago presents with the following echocardiography data:

MR peak velocity by CW Doppler study = 5 m/sec
Nyquist limit aliasing velocity = 40 cm/sec
PISA radius = 1 cm
The MR severity is:

A. Mild MR (EROA <0.2 cm^2)
B. Moderate MR (EROA = 0.2 – 0.4 cm^2)
C. Severe MR (EROA >0.4 cm^2)
D. Data is insufficient to make a judgement.

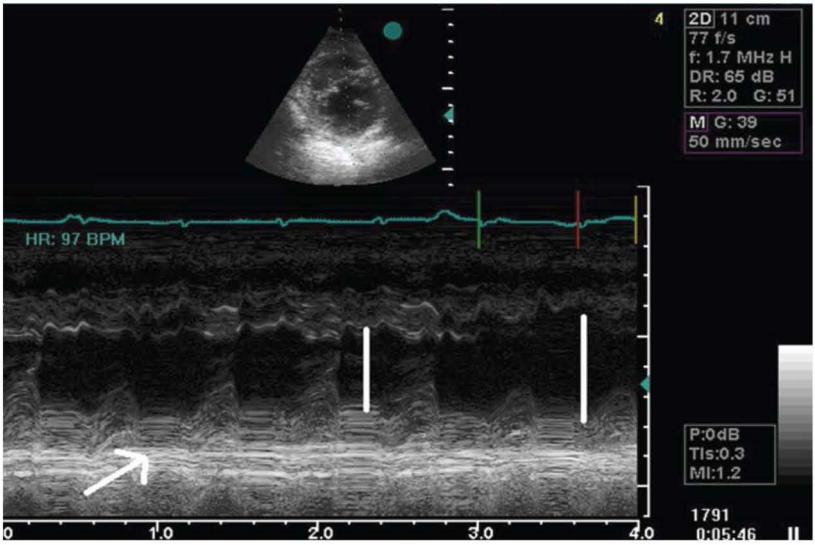
Answer: C

7. In which of the following situations is pressure half-time (PHT) a useful method for estimating the mitral valve area?

A. Severe MR
B. Severe AR
C. Early post-PTMC
D. Reduced LV compliance

Answer: A

8. Based on the following M-mode study, what is your diagnosis?



A. Tamponade
B. Restrictive cardiomyopathy
C. Hypertrophic cardiomyopathy
D. Constrictive pericarditis

Answer: D

The thickened pericardium, flattened left ventricular posterior wall, diastolic septal bounce, and competitive filling of the ventricles in the short-axis view favor a diagnosis of constrictive pericarditis.

9. Which of the following is not a common finding in constrictive pericarditis?

A. Exaggerated respiratory variation in left-right pressure and flow
B. Normal longitudinal strain
C. Bi-atrial enlargement
D. Prominent y descent in venous pressure

Answer: C

Table 3.1 Hemodynamic and echocardiographic features of constrictive pericarditis versus restrictive cardiomyopathy[3]

	Constriction	Restriction
Prominent Y descent in venoua pressure	Present	Variable
Paradoxical pulse	One third of cases	Absent
Pericardial knock	Present	Absent
Equal right- and left-sided filling pressure	Present	Left at least 3-5 mm Hg > right
Filling pressure > 25 mm Hg	Rare	Common
Pulmonary artery systolic pressure > 60 mm Hg	No	Common
"Square root" sign	Present	variable
Respiratory variation in left-right pressure and flow	Exaggerated	Normal
Ventricular wall thickness	Normal	Usually increased
Pericardial thichkness	Increased	Normal
Atrial size	Possible left atrial enlargement	Biatrial enlargement
Septal "bounce"	Present	Absent
Tissue Doppler E' velocity	Increased	Reduced
Speckle tracking	Normal longitudinal; decreased circumferential restoration	Decreased Longitudinal, normal circumferential restoration

10. Which of the following is incorrect regarding the echocardiography of constrictive pericarditis (CP):

A. The propagation velocity of early diastolic mitral inflow usually exceeds 100 cm/s.
B. Mitral valve deceleration time is less than 160 ms.
C. Significant variation can be found in the pulmonary venous Doppler study.
D. Hepatic vein flow Doppler study shows increased inspiratory diastolic flow reversal.

Answer: D

M-mode findings typical of constrictive pericarditis include:

i. a flattened LV posterior wall in the diastole;
ii. abrupt posterior displacement of the septum in early diastole (septal bounce or shudder);
iii. competitive filling of the ventricles (respiratory variation in ventricular size by M-mode study);
iv. the propagation velocity of early diastolic mitral inflow usually exceeds 100 cm/s.

Doppler findings typical of constrictive pericarditis include:

i. Significant respiratory variation in mitral (25–40%) and tricuspid (40–60%) valves by pulsed wave Doppler study for the calculation of the respiratory percentage variation in CP is the same for tamponade: for both the mitral and tricuspid valves, inflow is (expiration-inspiration)/expiration. The result is negative in a tricuspid inflow velocity study.
ii. Mitral valve deceleration time is less than 160 ms (in some but not all patients).
iii. A hepatic vein flow Doppler study shows an expiratory increase in diastolic flow reversal.
iv. Significant variation can be found in the pulmonary venous Doppler study. The Doppler study may show interventricular dependence by increased respiratory variation of mitral and tricuspid inflow velocity as a >25% decrease in mitral E velocity during inspiration.

The maximum drop would be in the first beat after inspiration and there would be a >40% increase of tricuspid E velocity in the first beat after inspiration.

v. An increase of 25% or more in mitral peak E velocity.

However, around 20% of patients with CP do not have typical respiratory variation, mostly because of elevated left atrial pressure or mixed constrictive and restrictive myocardial involvement.

References

1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition. 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 5

CHEST RADIOGRAPHY IN CARDIOVASCULAR DISEASE

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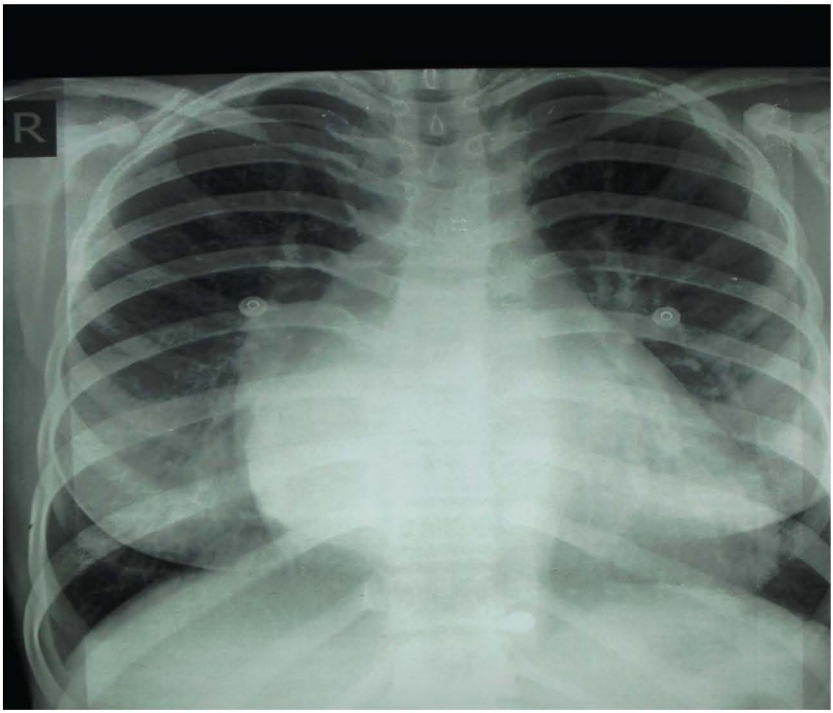
1. What is the most probable diagnosis in this chest x-ray (CXR)?



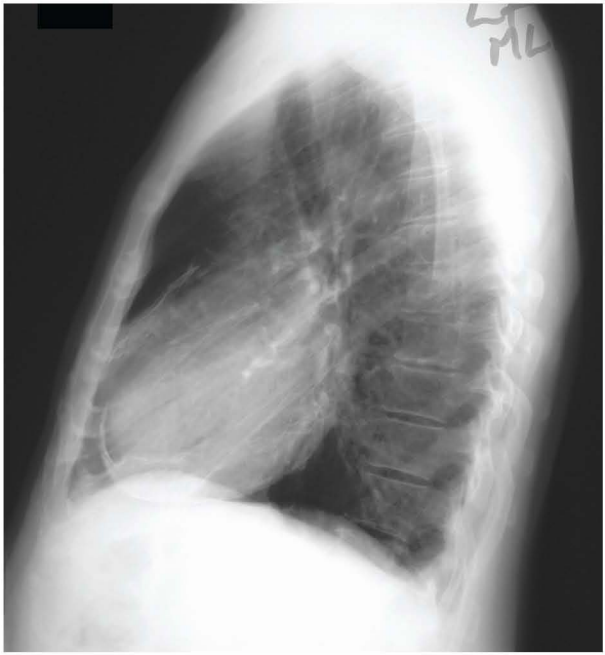
A. Congestive heart failure
B. Mitral valve regurgitation
C. Atrial septal defect
D. Primary pulmonary hypertension

Answer: B

Findings:

i. Increased cardiothoracic ratio
ii. Enlargement of left atrium
iii. Enlargement of left ventricle
iv. Enlargement of left atrial appendage
v. Widening of carina
vi. Pulmonary congestion

2. A 45-year-old male patient was referred to the clinic with the chief complaint of an edema of the bilateral lower extremities. What is the most probable diagnosis of the patient based on the CXR below?



A. Severe aortic valve stenosis
B. Tetralogy of Fallot
C. Mitral valve stenosis
D. Constrictive pericarditis

Answer: D

Findings:

i. Normal cardiothoracic ratio
ii. Thin curvilinear calcification of pericardium
iii. Cephalization of pulmonary vascularity
iv. Mild pleural effusion

3. A 35-year-old male patient was referred to hospital with dyspnea. In a physical exam cyanosis and clubbing were detected. What is the most probably diagnosis for this patient based on the CXR below?

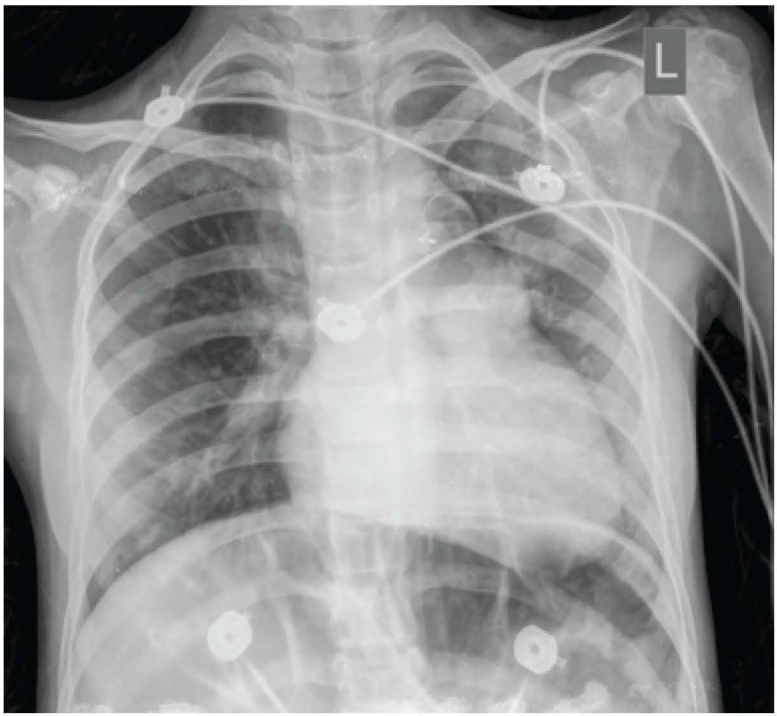
A. ASD and severe PAH (Eisenmenger syndrome)
B. Severe mitral valve stenosis
C. Cardiac tamponade
D. Pulmonary atresia and VSD

Answer: A

Findings:

i. Cardiomegaly
ii. Dilatation of RV
iii. Enlargement of the main pulmonary artery and central branches
iv. Pruning of distal branches of right and left pulmonary arteries
v. Left-sided aortic arch
vi. Normal size of aortic arch

4. A patient was admitted to CCU after an interventional procedure in the cath lab. What is the most probable diagnosis and procedure?



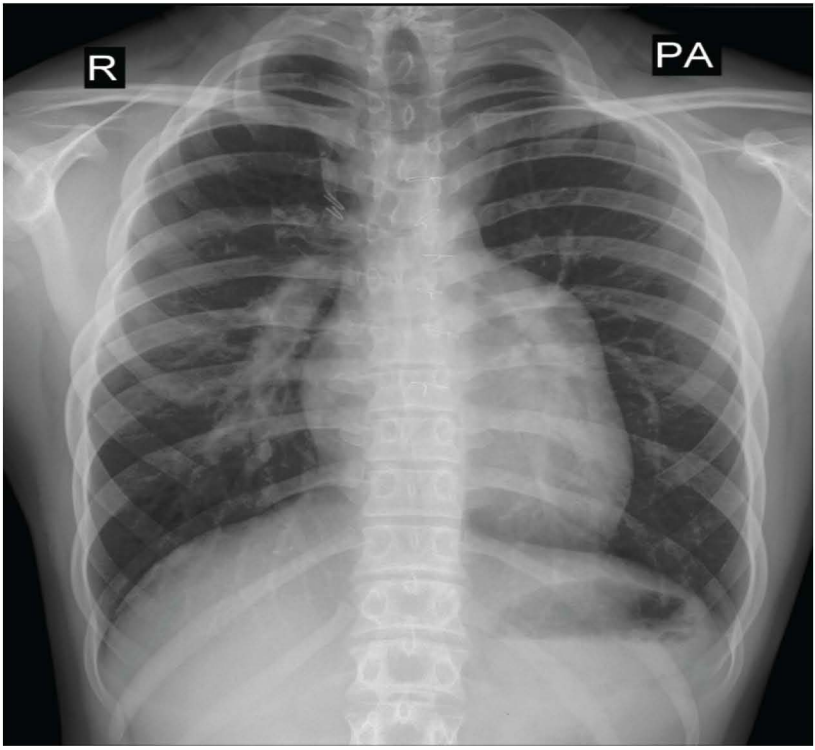
A. Atrial septal defect (ASD) and closure by vascular plug
B. Patent ductus arteriosus (PDA) and closure with Amplatzer device
C. Aortopulmonary collateral artery and closure by coil
D. Coronary fistula and occluded by Amplatzer device

Answer: B

Findings:

i. Cardiomegaly
ii. Dilatation of main pulmonary artery
iii. left-to-right shunt
iv. Dilatation of aortic knob

5. A 30-year- old patient with congenital heart disease was admitted for surgery. What is the most probable diagnosis?



A. Ventricular septal defect
B. Total pulmonary venous drainage
C. Total correction of Tetralogy of Fallot (TFTC)
D. Truncus Arteriosus

Answer: C

Findings:

i. Sutures of sternotomy
ii. Cardiomegaly and dilatation of right ventricle
iii. Aneurismal dilatation of right ventricular outflow
iv. Severe pulmonary valve insufficiency
v. Surgical clips of closure of right modified BT shunt

References

1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition. 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 6

CARDIAC COMPUTED TOMOGRAPHY

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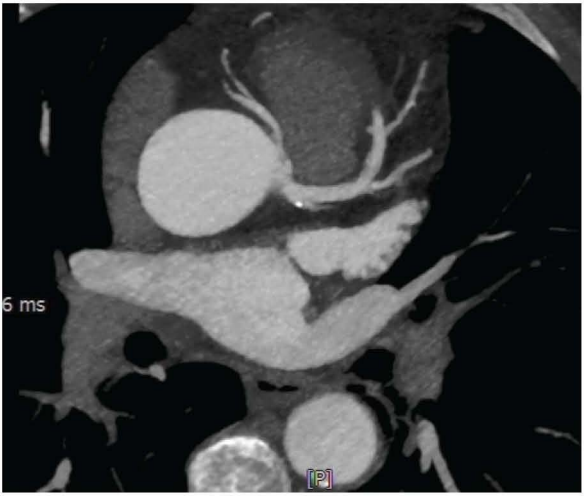
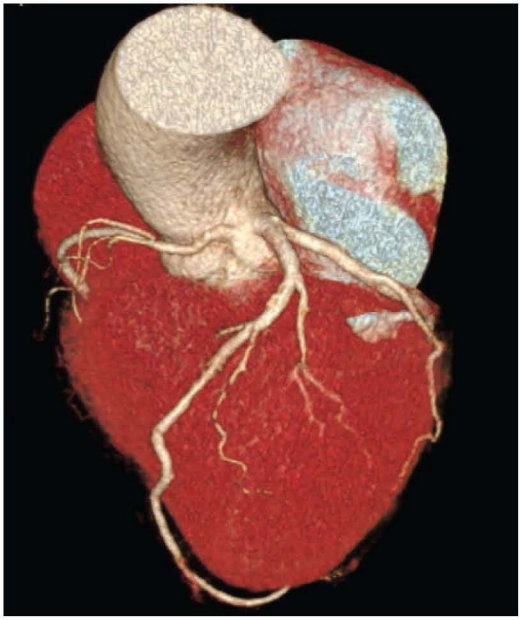
1. A young male patient is referred to the emergency department with atypical chest pain and negative troponin. The patient is sent to the imaging department for coronary CT angiography (CTA). What is the correct diagnosis of the patient according to these images?



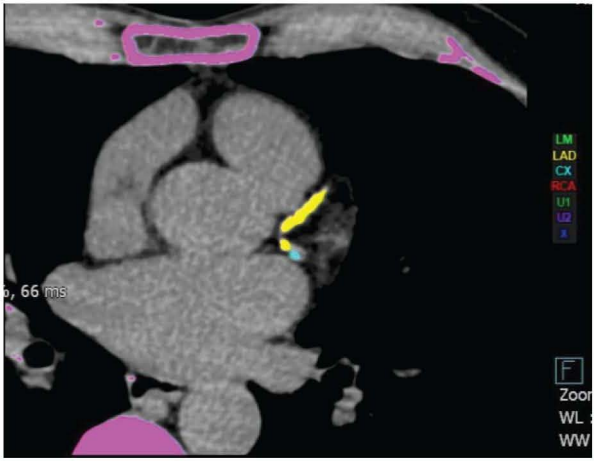


A. Left main arising from right coronary cusp
B. Large conus branch from left coronary cusp
C. Single coronary ostium from right coronary cusp
D. Right coronary artery from left coronary cusp and interarterial course

Answer: D

In this case, the anomalous RCA arises from left sinus valsalva and courses between the aorta and pulmonary artery, with narrowing and compression between them. Patients with this coronary anomaly are predisposed to sudden cardiac death. This is the most common form of a malignant course of the coronary artery.

2. An old man is referred to the imaging department for a coronary CT angiography. The result of the calcium score is attached here. What is your assessment?



Artery	Lesions	Volume / mm³	Equiv. Mass / mg	Score
LM	1	5.9	0.58	4.9
LAD	3	452.8	73.61	585.4
CX	3	123.1	16.20	146.1
RCA	0	0.0	0.00	0.0
Total	**7**	**581.8**	**90.39**	**736.4**
U1	0	0.0	0.00	0.0
U2	0	0.0	0.00	0.0

Settings
Score Type: User defined, Threshold: 130 HU (58.3 mg/cm³ CaHA)
Mass calibration factor: 0.449

A. According to the calcium score, the patient is low risk for coronary atherosclerotic changes (CAD)

B. According to the calcium score, the patient is an inappropriate candidate for a cardiac computed tomography angiography (CCTA)
C. According to the calcium score, the patient is an appropriate candidate for a cardiac computed tomography angiography
D. According to the calcium score, patient is intermediate risk for coronary atherosclerotic changes (CAD)

Answer: B

According to appropriate use criteria for CCTA (2010), a calcium score greater than 400 is not appropriate for cardiac CTA. In addition, the high calcium score in this patient (736) is suggestive of high risk for CAD and major cardiovascular events (MACE). Therefore, other functional studies or conventional angiography should be considered for the patient.

3. A young patient is referred to the emergency department with chest pain. The pretest probability for CAD was low to intermediate. ECG shows nonspecific changes and troponin is negative. What is your opinion about conducting a cardiac CT angiography?

A. Appropriate choice
B. Inappropriate choice
C. Indeterminate choice
D. None of them

Answer: A

According to the guidelines and appropriateness criteria for the performance of cardiac computed tomography, coronary CTA is appropriate for this patient. The coronary CTA shows a high negative predictive value of about 99%.

4. What are the characteristics of vulnerable plaque in coronary CTA?
A. Lipid rich plaque
B. Positive arterial remodeling
C. Napkin sign
D. All of them

Answer: D

Vulnerable plaque, which is thought to be associated with a greater risk for plaque rupture or acute coronary syndrome (ACS), has the following characteristics:

i. Lipid rich plaque (plaque with attenuation values <30 HU)
ii. Positive arterial remodeling (artery diameter ratio of the involved segment to a proximal reference of 1.1 or greater)
iii. Napkin sign
iv. A spotty pattern (<3 mm in size) of calcification

5. A patient is referred to the emergency department with atypical chest pain and a history of CABG. The ECG shows nonspecific change and a slight rise in troponin. What is your preferred choice for evaluation of the bypass grafts?

   A. Exercise tolerance test (ETT)
   B. Echocardiography
   C. Cardiac magnetic resonance (CMR)
   D. Cardiac CTA

Answer: D

In a meta-analysis, the evaluation of coronary bypass graft patency is highly accurate, with sensitivity and specificities of about 100%. Assessing the native coronary arteries is significantly more challenging than evaluating grafts. There is therefore a notable limitation when evaluating native coronary arteries due to severe calcification of coronary arteries and metallic clips. In one study, in evaluable segments, coronary CT angiography had a sensitivity of 86% and specificity of 76% for a significant stenosis.

References

1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition. 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 7

CARDIAC MAGNETIC RESONANCE IMAGING

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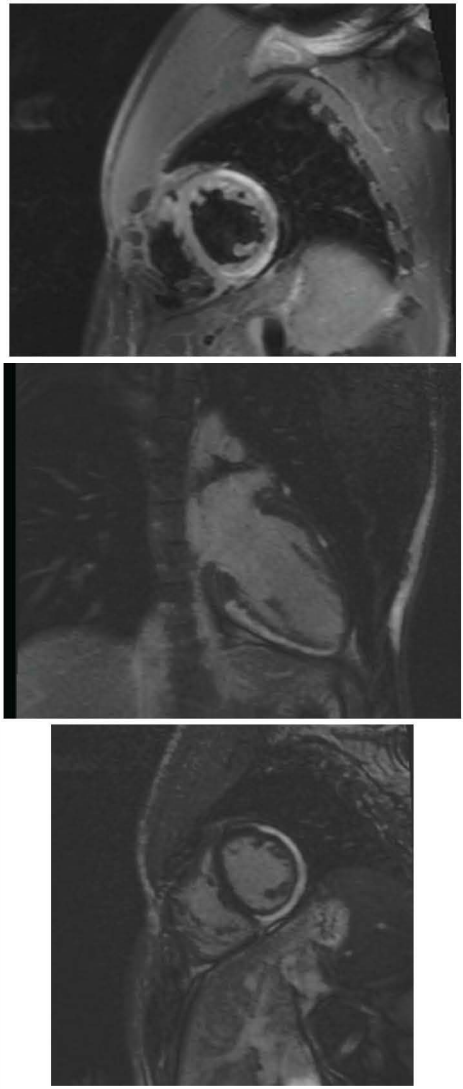
1. A 26-year-old male was referred to the emergency department with chest pain. ST-T changes are seen in all ECG leads. Additionally, a large increase in troponin concentration was observed. A conventional coronary angiography was conducted and the results were normal. The patient was sent to the imaging department and the following CMR (cardiovascular magnetic resonance) scan was taken. Based on the CMR, what is the most probable diagnosis for this patient?



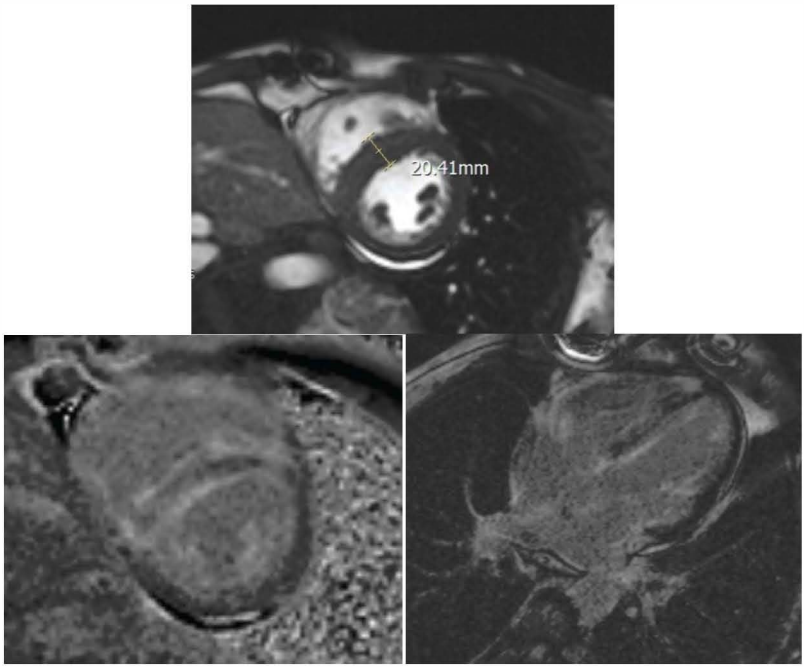
A) Myocarditis
B) Acute myocardial infarction
C) Takatsobo disease
D) Amyloidosis

Answer: A

Findings:

i. Edema in STIR sequence in posterolateral wall of LV.
ii. Late gadolinium enhancement in posterolateral wall of LV.

2. An elderly patient was admitted to hospital with the chief complaint of dyspnea and lower extremity edema. The echocardiographic findings suggest preserved LV systolic function and concentric LV hypertrophy. A CMR scan was done for the patient. What is the most probable diagnosis for this patient, according to attached images?



A. Cardiac sarcoidosis
B. Hypertensive cardiomyopathy
C. Cardiac amyloidosis
D. Hypertrophic cardiomyopathy

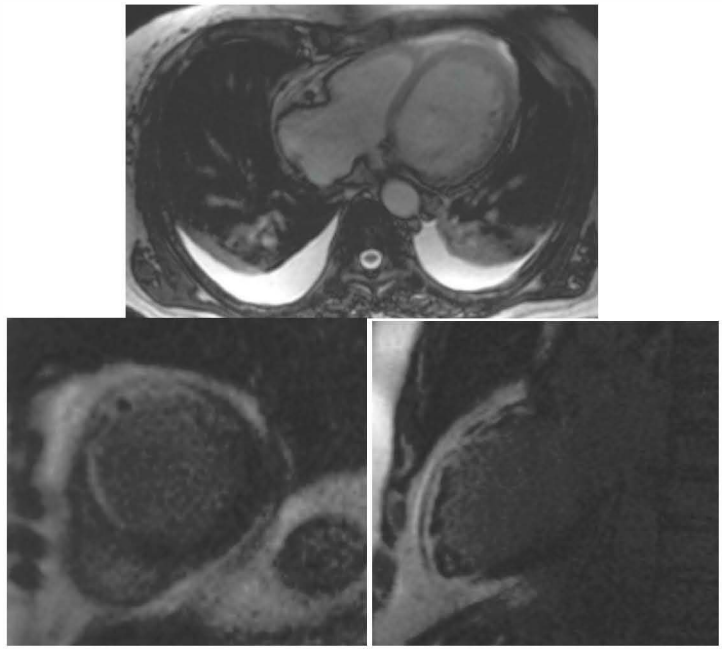
Answer: C

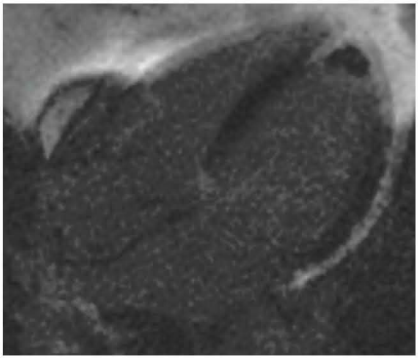
Findings:

i. Concentric hypertrophy of LV
ii. Diffuse subendocardial late gadolinium enhancement (LGE)
iii. Suboptimal nulling of myocardium in the longitudinal relaxation (TI) sequence for LGE

3. A 56-year-old patient with a history of recent myocardial infarction (MI) and three-vessel disease in conventional angiography was sent for CMR.

What is the diagnosis, based on the CMR findings?





A. Non-transmural fibrosis in the LAD territory
B. Transmural fibrosis in the LAD territory
C. Apical clot
D. A, C
E. B, C
F. None of the above

Answer: E

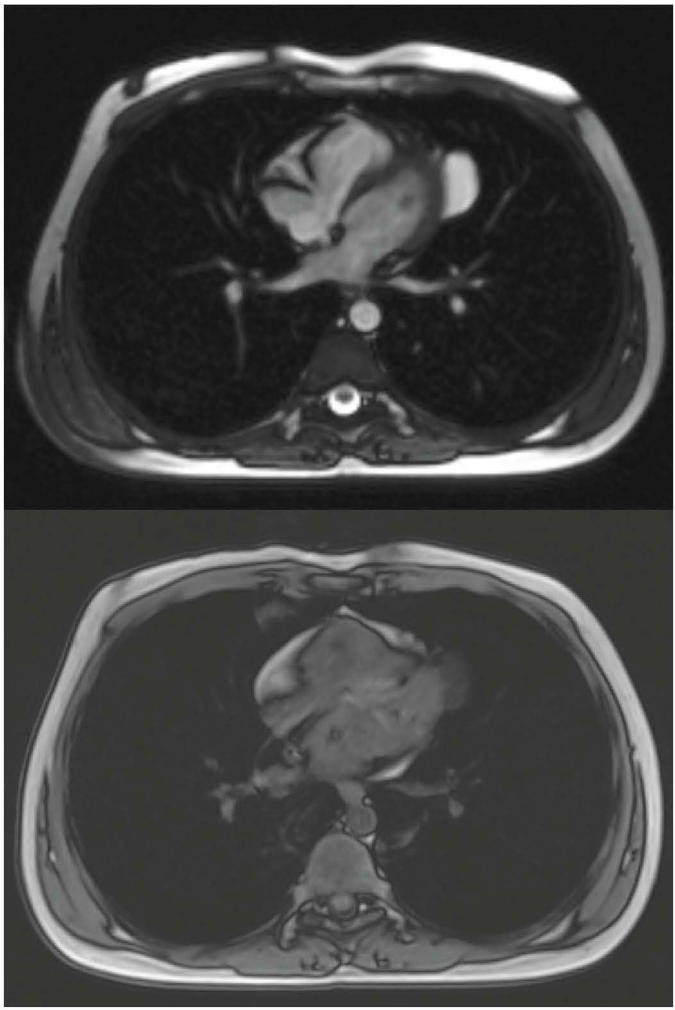
Findings:

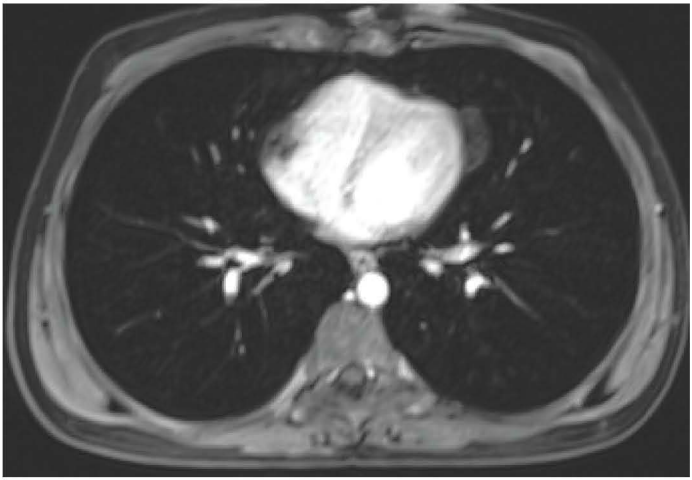
i. Bilateral pleural effusion
ii. Transmural LGE at the mid, apical anteroseptal and anterior segments and apical cap of the LV
iii. Low signal lesion in apex due to clot

These findings are suggestive of significant non-viable tissue in LAD territory.

4. A young patient is sent for a CMR to further evaluate a tumor attached to the left side of apex.

The resultant T1-W and T2-W images are presented below. After a gadolinium injection, no evidence of abnormal enhancement was seen. What is the most probable diagnosis?





A. Cardiac angiosarcoma
B. Myxoma
C. Pericardial cyst
D. Lymphoma

Answer: C

Findings:

i. Low signal lesion in T1-W
ii. Bright signal in T2-W
iii. No evidence of abnormal enhancement after IV contrast administration

5. What is the term used for the time taken for a proton to recover to a longitudinal state?

A. Longitudinal relaxation time or T1 time
B. Transverse relaxation time or T2
C. Time inversion recovery (TI)
D. Proton density

Answer: A

The time it takes for protons get to completely out of phase is called the transverse relaxation time or T2 (Table 7.1).

Table 7.1. Magnetic Resonance Imaging Signal Intensities

	T1-Weighted Image	**T2-Weighted Image**
Liquid (edema)	Dark	Bright
Fat	Bright	Dark
Acute blood	Dark	Intermediate
Subacute blood	Bright	Bright
Cyst	Dark	Bright
Calcium	Dark	Dark

References

1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition; 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 8

NUCLEAR CARDIOLOGY

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1. What is the appropriate stress type for myocardial perfusion imaging (MPI) in patients with left bundle branch block?

A. Exercise
B. Dipyridamole with low-grade exercise
C. Dipyridamole
D. Dobutamine

Answer: C

Reversible perfusion defects may appear in the anteroseptal wall in higher heart rates in patients with left bundle branch block (LBBB) or paced rhythm even in the absence of left anterior descending artery stenosis (LAD) in MPI. Hence, answers A, B and D are wrong. Pharmacologic stress with vasodilators (dipyridamole or adenosine) is the accepted modality of stress in these patients.

2. Which of the following locations should be considered as an abnormal finding in the MPI of a male patient with mild fixed perfusion defect pattern?

A. Basal septum
B. Apex
C. Septum as compared to the lateral wall
D. Anterolateral wall

Answer: D

Shortening of the septum is considered a normal variation in MPI as a result of the membranous septum; it appears as a decreased septal count density. A decreased count density of the apex, secondary to a partial volume effect, is another normal variation that happens in MPI. Mildly less septum-to-lateral wall activity is also considered as a normal variation due to the closer location of the lateral wall to the gamma camera during acquisition. Therefore, answers A, B, and C are wrong. On the other hand, persistent hypoactivity of the anterolateral wall should be considered abnormal in the absence of possible breast attenuation artifacts. Common normal variations and artifacts of MPI SPECT are shown in Table 8.1.

Table 8.1 Common normal variations and artifacts in MPI with SPEC

	Mechanisms	Findings
Normal Variations		
• Shortening of septum	Membranous septum	Decreased count density of basal septum
• Apical thinning	Partial volume effect	Decreased count density of the apex
• Higher lateral-to-septal wall activity	Closer location of lateral wall to gamma camera	Relatively lower count density of septum compared to lateral wall
Artifacts		
• Breast attenuation	Attenuation of photons by overlapping breast	Variable (depending on the breast size, position and density of breast)
• Diaphragmatic attenuation	Attenuation of photons by left hemidiaphragm and right ventricle	Decreased count density of inferior wall
• Patient's motion	Image misregistration in the process of SPECT reconstruction	Variable (depending on the extent, plane and time of motion)

• Subdiaphragmatic activity	Prominent activity in the liver, stomach or intestine	Decreased or increased count density of the adjacent wall (usually inferior wall)
• Technical artifacts	Flood field nonuniformity, Incorrect center of rotation, Inconsistent orientation and alignment of stress and rest studies	Variable

3. What is the severity of stress-induced ischemia and possibly stenosed coronary arteries in figure 8.1?

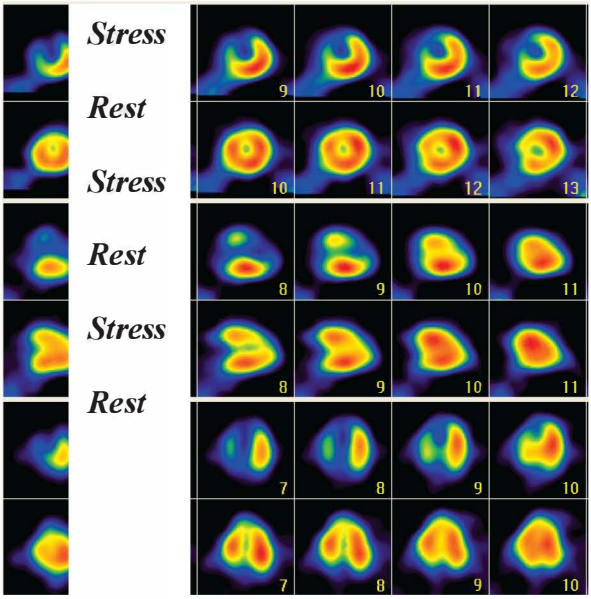

Figure 8.1. Stress-rest MPI images in short axis (upper rows), vertical long axis (middle rows) and horizontal long axis (lower rows).

A. Severe in LAD and mild in LCX
B. Severe in LAD and moderate in RCA
C. Moderate in LAD and mild in LCX and RCA
D. Severe in LAD and moderate in LCX and RCA.

Answer: A

There is almost an absence of activity in the anterior and septal walls on stress images, demonstrating total reversibility on the rest images. Therefore, severe stress-induced ischemia of the LAD territory is evident. The lateral wall shows a mild perfusion defect with total reversibility, compatible with mild stress-induced ischemia of the LCX territory. No distinct perfusion abnormality is noted in the inferior wall.

4. What kind of non-perfusion finding is evident in the previously presented MPI study?

A. Transient ischemic dilation
B. Transient RV visualization
C. Increased lung-to-heart uptake ratio
D. None of the above

Answer: A

The left ventricular cavity appears to be more dilated on stress images than on those from the rest phase, indicating transient ischemic dilation as a result of diffuse subendocardial ischemia. On the other hand, there is no considerable activity either in the right ventricular free wall or in the background to suggest, respectively, a transient RV visualization or an elevated lung-to-heart uptake ratio. Hence, answers B, C, and D are wrong. The most important high-risk non-perfusion markers of MPI SPECT are shown in Table 8.2.

Table 8.2 High-risk non-perfusion markers in MPI

Pattern	Finding	Interpretation
Lung uptake	Increased lung uptake is associated with an elevated LV end-diastolic pressure and can be quantified as lung-to-heart ratio (LHR)	Stress-induced LV dysfunction or severely decreased resting LVEF
Transient LV dilation	Increased LV size after stress as compared to the rest images (even in an otherwise normal MPI)	Most likely indicates subendocardial ischemia and systolic LV dysfunction
Transient right ventricular visualization	More prominent visualization of right ventricle on post-exercise images as compared to the rest images	Stress-induced LV dysfunction associated with severe CAD

5. What do you think the interpreting physician's report was in terms of risk stratification in the previously presented MPI study?

A. Low-risk
B. Intermediate risk
C. High-risk
D. Insufficient data for risk stratification

Answer: C

Transient ischemic dilation, secondary to diffuse subendocardial ischemia, along with severe and extensive stress-induced ischemia stratifies the patient as a high-risk case for subsequent hard cardiac events.

Reference

1. Malek H. Nuclear Cardiology. In: Maleki M, Alizadehasl A, Haghjoo M. Practical Cardiology: Elsevier 2018. p. 167-72.

CHAPTER 9

CATHETERIZATION AND ANGIOGRAPHY

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1. Which of the following is the best marker to get femoral access?

A. A few centimeters lower than the inguinal ligament
B. Maximal femoral pulse
C. Inguinal crease
D. Over the femoral head

Answer: D

In order to puncture the common femoral artery, the femur head can be found using fluoroscopy. Puncturing over the femoral head provides good support for a secure hemostasis. Another way is to palpate the anterior superior iliac spine and symphysis pubis. The line that connects these two anatomic landmarks suggests the location of the inguinal ligament. The common femoral artery is then punctured 2 to 3 cm caudal to this line.

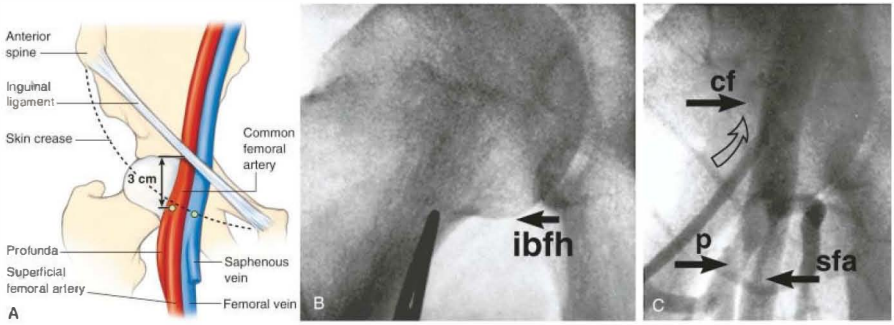

Figure 9.1. Regional anatomy relevant to percutaneous femoral arterial and venous catheterization

A. Schematic diagram showing the right femoral artery and vein coursing underneath the inguinal ligament, which runs from the anterior superior iliac spine to the pubic tubercle. The atrial skin nick should be paced approximately 3 cm below the ligament and directly over the femoral arterial pulsmedially. Also, this level corresponds roughly to the skin crease in most patients. Anatomic localization relative to the inguinal ligament provides a more constant landmark. **B**. Fluoroscopic localization of the skin nick (marked by the tip of the clamp) to the inferior border of the femoral head (ibfh). **C.** A catheter (open arrow) inserted through this skin nick has entered the common femoral artery (cf) above this bifurcation into the superficial femoral artery (sfa) and porofunda (p) branches.

2. Which situation is *not* an indication to do a coronary angiography?

A. Aborted sudden cardiac death
B. Low ejection fraction and no evidence of ischemia in noninvasive tests
C. When noninvasive tests cannot be done
D. Equivocal noninvasive tests

Answer: B

Indications for coronary angiography in patients with stable angina include a history of aborted sudden cardiac death (ASCD), symptoms of heart failure, moderate to high-risk noninvasive tests, and a low ejection fraction with evidence of ischemia in noninvasive tests. Another indication is when noninvasive tests cannot be done or are equivocal.

3. Which of the following is an indication to do coronary angiography before noncardiac surgery?

A. Refractory angina
B. Moderate-risk noninvasive test
C. Bedridden patient
D. Equivocal test before moderate-risk surgery

Answer: A

Indications for angiography before noncardiac surgery are a high-risk noninvasive test, refractory angina, and an equivocal test before high-risk surgery.

4. In which angiographic view is the whole length of the LAD seen well?

A. AP cranial
B. RAO caudal
C. RAO cranial
D. LAO cranial

Answer: C

Suitable left coronary views for evaluation of different coronary segments can be summarized as follows:

i. RAO caudal: LM, proximal LAD, distal LAD, and OMs
ii. AP cranial: Midpart LAD, diagonals, and septals
iii. LAO cranial: Ostium of LM and diagonals, midpart LAD, and distal of dominant LCX
iv. LAO caudal (spider view): LM, proximal LAD, proximal LCX
v. AP caudal: LM, Ostioproximal LAD, proximal LCX
vi. RAO cranial: The whole length of LAD
vii. Left lateral: Midpart LAD, distal LAD

5. A coronary stenosis is considered type C if it is …

A. Eccentric
B. Thrombotic
C. Calcified
D. Diffuse

Answer: D

Features of a type C lesion include its being diffuse (>2cm in length), excessive tortuosity of the proximal segment, extremely angulated segments (≥90°), total occlusion >3 months old, inability to protect major side branches, and degenerated vein grafts with friable lesions.

Table 9.1 Characteristics of Type A, B, and C Coronary Lesions[1,3]

CHARACTERISTIC	DESCRIPTION
Type A Lesions (high success >85%; low risk)	
Discrete (<10 mm)	Little or no calcium
Concentric	Less than totally occlusive
Readily accessible	Not ostial in locations
Nonangulated segment, <45°	No major side branch involvement
Smooth contour	Absence of thrombus
Type B Lesions (moderate success, 60%-85%; moderate risk)	
Tubular (10 to 20 mm in length)	Moderate to heavy calcification
Eccentric	Total occlusions <3 months old
Moderate tortuosity of proximal segment	Ostial in location
Moderately angulated segment, ≥45 degrees, <90°	Bifurcation lesion requiring double guidewire
Irregular contour	Some thrombus present
Type C Lesions (low success, <60%; high risk)	
Diffuse (>2 cm in length)	Total occlusion >3 months old
Excessive tortuosity of proximal segment	Inability to protect major side branches
Extremely angulated segments, ≥90°	Degenerated vein grafts with friable lesions

6. Which coronary anomaly is more benign?

A. Ostial bending of anomalous artery
B. Slit-like coronary ostia
C. Anomalous LCX from RCA
D. Interarterial course of LM from RCA

Answer: C

In some patients, the LCX may originate from the proximal RCA or from the right coronary cusp near the RCA ostium. The course of the LCX is usually retroaortic and benign.

7. Which of the following has no place in coronary muscle bridge treatment?

A. Surgery
B. Beta blockers
C. Calcium blockers
D. Nitrates

Answer: D

In muscle bridge patients, the appropriate approach to treatment is to administer beta-blockers or calcium blockers and avoid nitrates. Stenting or surgery is appropriate in a minority of patients.

8. A distorted bladder shadow in fluoroscopy is a sign of …

A. Pseudoaneurysm
B. AV fistula
C. Retroperitoneal hematoma
D. Subcutaneous hematoma

Answer: C

A distorted bladder shadow in fluoroscopy is evidence for a retroperitoneal hematoma.

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CHAPTER 10

HEMODYNAMIC STUDY

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Table 10.1 Data for right heart catheterization of an advanced heart failure patient

Age	25
BSA	1.75 m^2
Heart rate	108/min
Systemic arterial pressure (mean)	105/75 mmHg
Mean central venous pressure	12 mmHg
Right ventricular pressure	30/5-10 mmHg
Pulmonary arterial pressure (mean)	30/10 mmHg
Pulmonary capillary wedge pressure	14 mmHg
Systemic arterial O2 saturation	94%
Mixed venous O2 saturation	78%
Hemoglobin level	14.5 g/dl

1. What is the estimated cardiac output?

A. 5 Lit/min
B. 7 Lit/min
C. 3.5 Lit/min
D. 4 Lit/min

Answer: B

Calculation of cardiac output by the Fick method is considered the "gold standard" for estimating cardiac output. The blood oxygen content in arterial (CaO2) and venous (CvO2) blood, as well as an estimation of body oxygen consumption (VO2), are required to calculate Fick CO. The arteriovenous oxygen difference is calculated by subtracting the oxygen content of mixed venous blood, usually pulmonary arterial blood in most clinical settings, from pulmonary venous blood, which is estimated by systemic arterial blood. Estimated O2 consumption (ml/min) = 125 × BSA

Fick CO (liter/minute)

$$= \frac{Oxygen\ Consumption\ (mL/min)}{[(Ao\%\ Sat - PA\%sat)/100]\ x\ 1.36\ x\ Hgb\ (mg/dl)\ x\ 10}$$

Considering the mentioned above explanations, the oxygen consumption in this patient will be estimated as 125 × 1.75 = 218.75 ml/min

The arteriovenous oxygen difference will be (0.94-0.78) × 1.36 × 14.5 × 10 = 31.55

So, the estimated cardiac output by Fick method will be 218.75 ÷ 31.55 = ~ 7 lit/min

2. What is the estimated pulmonary vascular resistance by Wood unit?

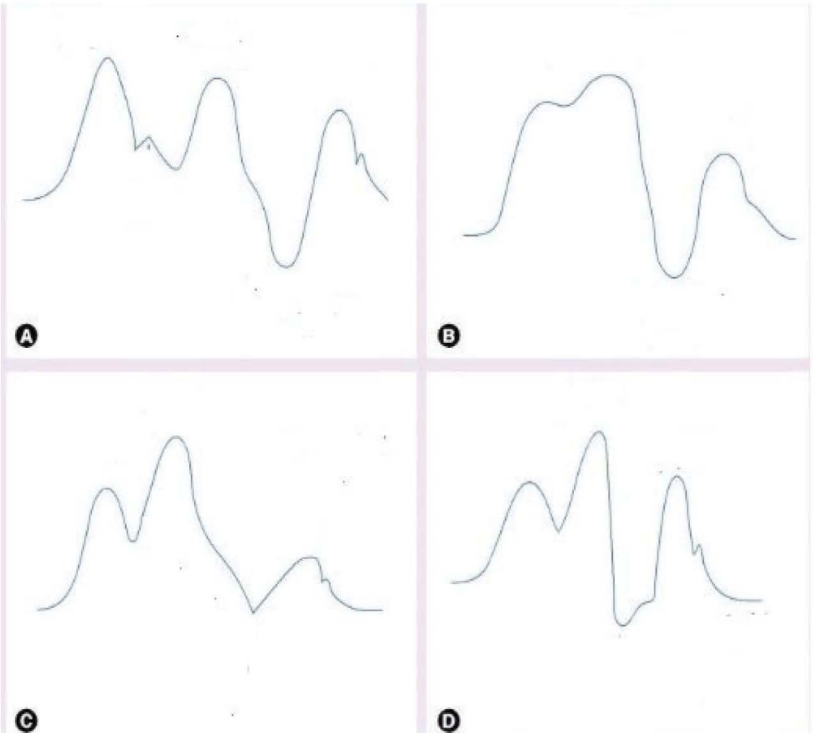
A. 1 Wood
B. 1.5 Wood
C. 0.5 Wood
D. 3 Wood

Answer: C

The estimated PVR by Wood unit can be calculated by following formulae:

PVR (Wood unit or mmHg/L/min) = MPAP – left atrial pressure (PCWP) ÷ Cardiac Output
MPAP = Mean Pulmonary Arterial Pressure = (MPAP – PCWP) ÷ CO
MPAP = 16.7 and PVR = (16.7-14) ÷ 7 = 0.5 Wood

3. Please match the intra-aortic balloon pump waveforms with the appropriate description:



1. Early inflation
2. Late inflation
3. Early deflation
4. Late deflation

Answer:

1. B
2. A
3. D
4. C

When the balloon inflation and deflation is correctly timed, it results in optimal hemodynamic effects. The balloon inflates about 40 milliseconds

before the dicrotic notch. This is timed by the end of the T wave on the ECG. The deflation of the balloon is in time with the R wave of the ECG. The pulse waveform after augmentation shows a reduced aortic systolic and end diastolic pressure.

References

1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition. 2017.
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CHAPTER 11

HEART FAILURE AND PULMONARY HYPERTENSION

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1. Which stage of heart failure can be considered for a newly symptomatic heart failure patient?

A. A
B. B
C. C
D. D

Answer: C

HF syndrome has 4 stages based on structural changes and symptoms. The clinical approach and management strategies are different in each stage. Figure 12-1, chapter 11 of Practical Cardiology shows stages in the development of heart failure and recommended therapy by stage.

2. Which of the following statements is incorrect?

A. Patients who have received diuretics may not present signs of heart failure.
B. The presence of a high BNP level is not necessary for diagnosis of HFPEF (HF with preserved LVEF) or HF mid-range EF.
C. In a non-acute setting, B-type natriuretic peptide (BNP) <35 pg/ml and N-terminal pro-BNP (NT-proBNP) <125 pg/ml are above normal limits.
D. NPs may be low in obese patients.

Answer: B

According to the new definition for heart failure in the setting of HFpEF and HFmrEF, the presence of an elevated NT pro BNP or BNP levels accompanied with evidences of structural heart disease and/or diastolic dysfunction is necessary to diagnosis of HF.

3. Which of the following statements is incorrect?

A. Transesophageal echocardiography (TEE) is not routinely recommended in patients with HF.
B. Using NPs to make a diagnosis of HF is recommended because of the high positive predictive value of their cut points.
C. NPs can be elevated in sepsis and diabetic ketoacidosis.
D. CMR is recommended in HF cases suspected of myocarditis and infiltrative cardiomyopathies.

Answer: B

The negative predictive value of these cut points is higher; therefore, it is recommended to use NPs for ruling out HF, not to make the diagnosis. There are many non-cardiac conditions in which NPs are elevated, including cerebrovascular events, renal failure, advanced age, liver failure, COPD, severe infection and sepsis, anemia, thyrotoxicosis, diabetic ketoacidosis and paraneoplastic syndromes.

4. Which of the following statements is not correct regarding the evaluation of acute heart failure?

A. Patients with first-occurrence heart failure who have acceptable hemodynamic findings do not need hospital admission; outpatient treatment and follow-up would be enough.
B. A routine arterial blood gas measurement is not mandatory in patients with AHF.
C. A ratio of first-measured LDH to ALT greater than 1.5 in patients with AHF is more probably related to the AHF than liver causes of abnormal LFT.
D. The plasma level of NPs should be measured before discharge for prognostic evaluation.

Answer: A

Indications for admission in acute heart failure:
Evidence of severe decompensated heart failure, hypoperfusion and end organ damage, including loss of consciousness, hypotension, pulmonary edema and kidney or liver dysfunction.
Presence of dyspnea at rest accompanied with rapid breathing or arterial oxygen saturation less than 90% at room air.
Arrhythmias with hemodynamic impairment including atrial fibrillation with rapid ventricular response and ventricular tachycardia.
Acute coronary syndrome.
Aggravated edema or presence of severe peripheral congestion even in the absence of dyspnea
Significant electrolyte imbalance.
Presence of other critical conditions such as pneumonia, pulmonary emboli, cerebrovascular accidents, prosthetic valve malfunction or diabetic ketoacidosis.
Frequent ICD firing.
Acute de novo heart failure (first occurrence of heart failure).

5. A 73-year-old woman was admitted to the CCU with an acute anterior myocardial infarction and primary LAD PCI two hours ago. She has drowsiness and a systolic blood pressure of 60 mmHg. A Swan Ganz catheter is placed for her. Considering the following hemodynamic data, which of the following options should be done for the patient?

PCWP = 10 mmHg, RAP = 8 mmHg, PAP = 4 0/20 mmHg

A. Transfer her to the cath lab and repeat the coronary angiography
B. Start norepinephrine
C. Insert an IABP
D. Give her some IV fluid

Answer: D

This hemodynamic study shows that in this patient hypotension is secondary to the hypovolemia and depleted preload. The increase in preload by intravenous fluid will help to increase the stroke volume and blood pressure.

6. A 68-year-old man was admitted to the CCU with a diagnosis of severe systolic heart failure and an LVEF of 15%. A pulmonary artery catheter was placed for him due to developing hypotension. He also has cold extremities and is a little confused. His urinary output has decreased compared to three hours ago. The initial readings are:

BP = 83/56, heart rate = 112, PCWP = 18, PAP = 34/21, RAP = 13, CO = 3.6, CI = 2, SV = 32

Which of the following is the better choice for him?

A. IV fluid
B. Milrinone
C. Increasing furosemide dose
D. Schedule patient for circulatory assist device

Answer: B

As an inotrope, milrinone will be effective in improving the stroke volume. This example demonstrates that the physician should be careful about both filling pressures (preload) and contractility. The low cardiac output is responsible for hypotension and oliguria (low output state) in this scenario, so it is rational to start treatment with an inotrope. Although there may be no change in filling pressures, the improved contractility following treatment with an inotropic agent like milrinone can lead to an improvement in cardiac output.

7. A 62-year-old man is visited in an outpatient clinic. He is a known case of dilated cardiomyopathy and has dyspnea on exertion with an NYHA class of II. His medications are carvedilol, lisinopril, eplerenone, and furosemide. Which of the following would not be helpful in improving his functional class?

A. Starting ivabradine based on his rhythm and heart rate
B. Adding valsartan to his medications, considering his blood pressure
C. Evaluating his candidacy for CRT
D. Substituting lisinopril with ARNI

Answer: B

According to the last heart failure guidelines (figure 12-4, Practical Cardiology), ARBs are recommended in HF patients who cannot tolerate ACE inhibitors because of serious side effects, accompanied with beta blockers and MRAs. ARBs may be considered in combination with ACE inhibitors in symptomatic HF patients who are receiving beta blockers and cannot tolerate MRAs. This combination should be used under strict supervision and the combination of ACE inhibitors plus ARBs plus MRAs should never be started in any heart failure patient.

8. A 55-year-old man with a history of mitral valve replacement presents with signs and symptoms of severe right ventricular failure. He has severe edema, ascitic, oliguria and abnormal liver function tests. Several hours after admission, he develops hypoxemia and loss of consciousness and is placed on mechanical ventilation. Which of the following items is not correct in his management?

A. Use the maximum PEEP setting for mechanical ventilation
B. Use inotropes e.g. norepinephrine
C. Optimize fluid overload based on central venous pressure
D. Undertake renal replacement therapy if required

Answer: A

For protective mechanical lung ventilation in a patient with severe right ventricular failure, the following measures should be kept in mind:

- P-plato ≤ 30 mmHg –
- Tidal volume 4-6 ml/kg/predicted body weight
- Minimize PEEP
- Avoid acidosis, hypercarbia, hypoxemia and auto PEEP

It is recommended that patients with RV failure who are on mechanical ventilation should have daily episodes of spontaneous breathing

9. Which of the following patients should be listed for heart transplantation?

A. An ambulatory HFREF patient with a peak VO2 >14 ml/kg/min
B. A patient who has had an extracorporeal membrane oxygenator (ECMO) due to cardiogenic shock three days ago
C. A patient with asymptomatic restrictive cardiomyopathy
D. A patient with refractory heart failure secondary to crystal abuse

Answer: B

In an AHF setting, heart transplantation should be considered for following patients unless there are contraindications:

- Severe refractory cardiogenic shock requiring high-dose inotropic support or IABP or other short-term mechanical assisted devices and when a long-term assist device cannot be considered for the patient.
- Patients with long-term VAD who have developed VAD-related complications that cannot be resolved or VAD replacement is impossible.

Listing for heart transplantation in a chronic setting should be considered for the following patients, unless there are contraindications:

- Presence of a peak VO2 ≤ 12 ml/kg/min in HF patients receiving beta blockers and ≤ 14 ml/kg/min in HF patients intolerant to beta blockers.

- In women and young patients (<50 years) the percent of predicted peak VO2 (≤ 50%) should also be considered in conjunction with peak VO2.
- Presence of a ventilation equivalent of carbon dioxide (VE/VCO_2) slope of > 35 in the presence of a sub-maximal CPX test (RER < 1.05)
- Patients with an estimated 1-year survival < 80%, calculated by the Seattle Heart Failure Model (SHFM) or a medium/high risk for mortality Heart Failure Survival Score (HFSS).
- Severe or intractable angina chest pain in patients with coronary artery disease who cannot be revascularized.
- Intractable life-threatening arrhythmias unresponsive to medical therapy, catheter ablation, and/or implantation of an ICD.
- Hyper trophic cardiomyopathy, restrictive cardiomyopathy or right-sided cardiomyopathies refractory or unresponsive to medical management.

10. A 60-year-old woman with a history of ischemic cardiomyopathy presents with decompensated heart failure. At the CCU, she is put on intravenous nitrate and a furosemide infusion. After 48 hours, she has oliguria and her creatinine has risen from 1.2 to 2.4. Which of the following actions would be incorrect in the management of this patient?

A. Stopping furosemide as a nephrotoxic agent
B. Adding metolazone
C. Rechecking serum electrolytes and echocardiographic data
D. Performing right heart catheterization

Answer: A

The patient has diuretic resistance and in this condition diuretics such as furosemide should not be stopped and another diuretic such as metolazone should be added to the furosemide to potentiate the diuretic effects in this patient.. Combination diuretic therapy (CDT) should be used only in patients with severe fluid overload refractory to adequate doses of IV furosemide, particularly patients with decompensated advanced heart failure and those who have chronic renal failure. Close monitoring of serum electrolytes is very important. Severe hypokalemia and hypomagnesaemia may develop with CDT. Some

increase in serum creatinine levels may be seen with CDT, but is usually reversible.

11. A 27-year-old woman has been referred for evaluation of dyspnea on exertion. The NYHA functional class is estimated to be about II. The physical exam is unremarkable and she has no significant past medical, drug, or family history. Her electrocardiogram reveals a sinus tachycardia, right axis deviation and P pulmonale. Echocardiography provides the following data:

> Normal LV size, LVEF = 50-55%, flattening of interventricular septum, moderate right ventricular enlargement and mild RV dysfunction, Mild tricuspid regurgitation (TR), TR velocity = 3m/s, right atrial area = 20cm^2, IVC size = 2.2 cm with good collapse and no pericardial effusion or other valvular heart disease.

Which of the following is not among the measures to be taken next?

A. Pulmonary function test/DLCO
B. Chest HRCT
C. ABG
D. Right heart catheterization

Answer: D

This patient has high probability of pulmonary hypertension and the first step should be evaluation of lung problems and left heart disease (Figure 12-7 in Practical Cardiology).

12. Regarding the previous case (question 11), because the findings were unremarkable, the patient is referred to a PH expert center. Further evaluations show a normal V/Q scan, an NT-ProBNP of 1050 ng/L and a 6-minute walk test of 370 meters. Which of the following right heart catheterization data do not have prognostic significance in this patient?

A. Cardiac index
B. Pulmonary vascular resistance
C. Right atrial pressure
D. Mixed venous O2 saturation

Answer: B

In a hemodynamic study of patients with pulmonary hypertension cardiac index, right atrial pressure and mixed venous O2 saturation have prognostic significance.

13. A 36-year-old female patient has a pulmonary arterial pressure of 70 mmHg in right heart catheterization and a PCWP of 5 mmHg. A vasodilator challenge test is performed and the following results are obtained:

Table 11.1

	Baseline	**At the end of test**
PAP	65/35 mmHg	45/25 mmHg
PCWP	5 mmHg	5 mmHg
Cardiac output	5 lit/min	5 lit/min
Heart rate	80	85
Blood pressure	100/70 mmHg	95/65 mmHg
Mixed venous O2 saturation	65%	63%
Systemic arterial saturation	94%	96%

What is the best medical plan for the patient?

A. To initiate Tadalafil
B. To initiate an oral combination of Bosentan and Tadalafil
C. To initiate a calcium channel blocker
D. To initiate intravenous prostaglandin

Answer: C

Considering the result of this vasoreactive challenge test and the decrease in the mean PAP to less than 40 mmHg, the patient is candidate for starting a calcium channel blocker.

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CHAPTER 12

TACHYARRHYTHMIAS

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1. Which kind of supraventricular tachycardia is more likely to present with syncope?

A. Focal atrial tachycardia
B. Atrioventricular nodal reentrant tachycardia
C. Atrial fibrillation
D. Orthodromic atrioventricular reciprocating tachycardia

Answer: C

Syncope is a rare manifestation of supraventricular tachycardias because the heart rate is not so fast as to impair cardiac output. However, atrial fibrillation and flutter may present with syncope caused by a very rapid ventricular rate (>250 bpm).

2. What is the most common paroxysmal supraventricular tachycardia (PSVT) in adults?

A. Atrioventricular nodal reentrant tachycardia
B. Orthodromic atrioventricular reciprocating tachycardia
C. Focal atrial tachycardia
D. Atrial fibrillation

Answer: A

The term PSVT is applied to supraventricular tachycardias with an abrupt onset and offset other than atrial fibrillation, atrial flutter, and multifocal atrial tachycardia. Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common (60%) PSVT in adults. Orthodromic atrioventricular reciprocating tachycardia (o-AVRT) is the second most common (30%) PSVT in adults. Focal atrial tachycardia is the third most common (10%) PSVT in adults.

3. Choose the correct statement on ST-segment and T-wave changes which occur during paroxysmal supraventricular tachycardia.

A. They are more common with atrioventricular nodal reentrant tachycardia.
B. ST-T changes have been reported in 25-50% of patients with orthodromic atrioventricular reciprocating tachycardia.
C. ST-segment depression is mainly related to myocardial ischemia.
D. Memory T wave has been observed in 40% of patients with atrioventricular nodal reentrant tachycardia.

Answer: D

Although it is more common with orthodromic atrioventricular reciprocating tachycardia, ST-T changes are reported in 25-50% of patients with atrioventricular nodal reentrant tachycardia. ST-segment depression is mainly related to a repolarization abnormality and does not indicate myocardial ischemia. Memory T waves are observed in 40% of patients with atrioventricular nodal reentrant tachycardia.

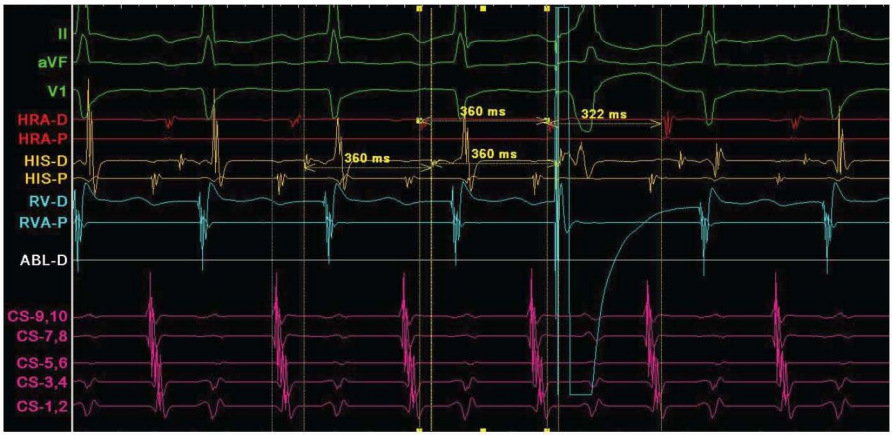
4. Which statement more accurately describes slow-slow atrioventricular nodal reentrant tachycardia?

A. Long AH interval (>180 ms) and the earliest retrograde atrial activation near the His bundle
B. Short AH interval (<180 ms) and the earliest retrograde atrial activation near the coronary sinus ostium
C. Long AH interval (>180 ms) and the earliest retrograde atrial activation near the coronary sinus ostium
D. Short AH interval (<180 ms) and the earliest retrograde atrial activation near His bundle

Answer: C

In slow-slow atrioventricular nodal reentrant tachycardia, both the antegrade and the retrograde limbs of the circuit use slow AV nodal pathways. Therefore, antegrade limb would have a long AH interval and retrograde atrial activation is recorded near the coronary sinus ostium.

5. A 32-year-old woman presented with recurrent palpitations. During an electrophysiology study, the following tracing was recorded. What is the most likely mechanism for her palpitation?

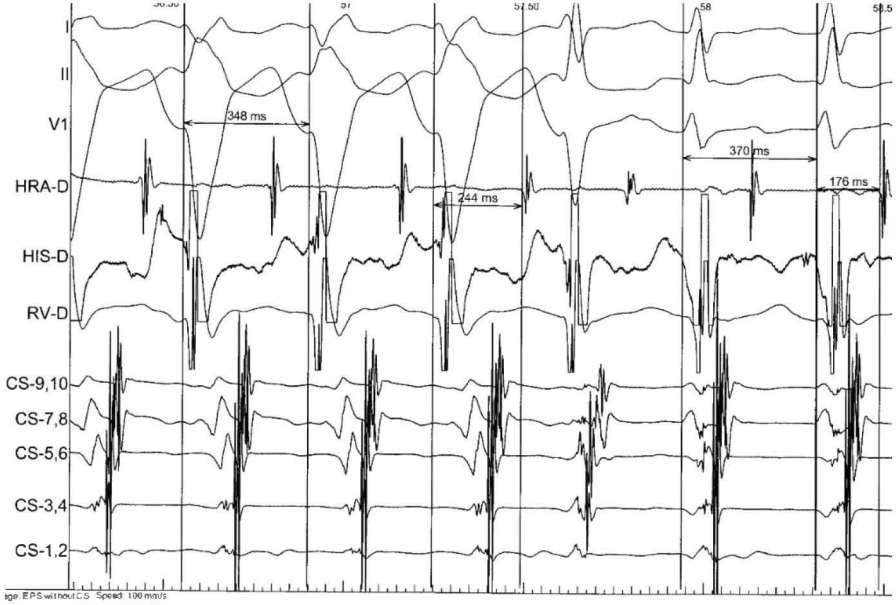


A. Atypical atrioventricular nodal reentrant tachycardia
B. Permanent junctional reciprocating tachycardia
C. Posteroseptal atrial tachycardia
D. Posteroseptal orthodromic atrioventricular reciprocating tachycardia

Answer: B

Atrial advancement by delivery ventricular ectopy during His refractoriness is indicative of a retrogradely conducting accessory pathway. Therefore, only B and D can explain this tracing. However, a long VA interval during tachycardia favors permanent junctional reciprocating tachycardia.

6. A 22-year-old man presented with frequent palpitations. The following tracing was recorded during an electrophysiology study. What is the most likely mechanism?



A. Left free-wall orthodromic atrioventricular reciprocating tachycardia
B. Left variant atrioventricular nodal reentrant tachycardia
C. Left-sided focal atrial tachycardia
D. Left septal orthodromic atrioventricular reciprocating tachycardia

Answer: A

In free-wall accessory pathways, ipsilateral bundle branch block during orthodromic atrioventricular reciprocating tachycardia (AVRT) results in prolongation of the surface VA interval (≥35 ms) because more time is needed for the impulse to travel from the AV node, the His bundle, the contralateral bundle branch, and transseptally to the ipsilateral ventricle to reach the accessory pathway and then activate the atrium. Smaller prolongation (≥25 ms) indicates septal accessory pathway. Aberrancy has

no effect on atrioventricular nodal reentrant tachycardia or focal atrial tachycardia.

7. Which of the following ECG findings is typical for orthodromic atrioventricular reentrant tachycardia?

A. Upsloping ST-segment depression of 2 mm or more at the J-point and lasting for 80 ms in inferior leads
B. Downsloping ST-segment depression of 2 mm or more at the J-point and lasting for 80 ms in precordial leads
C. Downsloping ST-segment elevation of 1 mm at the J-point and lasting for 80 ms in lead aVR
D. RP interval of 60 ms or greater

Answer: B

Characteristic ECG findings in orthodromic atrioventricular reentrant tachycardia are:

i. Horizontal or downsloping ST-segment depression of 2 mm or more at J-point and lasting for 80 ms in inferior and precordial leads;
ii. Horizontal or upsloping ST-segment elevation of 1 mm or greater at J-point
iii. Downsloping ST-segment elevation of 1.5 mm or greater and lasting for 80 ms in lead aVR
iv. An RP interval of 100 ms or more.

8. What is the most common origin for left-sided focal atrial tachycardia?

A. Left atrial appendage
B. Mitral annulus
C. Pulmonary veins
D. Left interatrial septum

Answer: C

Left atrial tachycardias predominantly arise from pulmonary veins.

9. Where do you localize an atrial tachycardia with a negative P wave in the precordial and inferior leads?

A. Posterior tricuspid annulus
B. Right atrial appendage
C. Posterior mitral annulus
D. Inferior crista terminalis

Answer: A

Negative P waves in precordial leads suggest RA appendage or tricuspid annulus. Negative P waves in inferior leads suggest a low atrial origin (CS ostium, posterior mitral or tricuspid annulus). Combination of these P-wave morphologies points to posterior tricuspid annulus.

10. What is the most common cause of atypical left atrial flutter?

A. Surgery for congenital heart disease
B. Coronary artery bypass surgery
C. Incomplete ablation lines related to the Maze procedure or catheter ablation
D. Surgery for valvular heart disease

Answer: C

Atypical left atrial flutters are most commonly caused by incomplete ablation lines related to the Maze procedure or catheter ablation. They are usually seen in the anterior wall, through the roof, or on the septum. Mapping can often be difficult because of low voltages.

11. A 24-year-old woman presented with frequent episodes of palpitation. ECG showed narrow complex tachycardia. Intravenous injection of 12 mg of adenosine resulted in tachycardia termination with a P wave. This rules out:

A. Atrial tachycardia
B. Typical atrioventricular nodal reentrant tachycardia
C. Atypical atrioventricular nodal reentrant tachycardia
D. Orthodromic atrioventricular reciprocating tachycardia

Answer: A

Termination of narrow complex tachycardia with a P wave rules out atrial tachycardia and can be seen in typical or atypical atrioventricular nodal reentrant tachycardia and orthodromic atrioventricular reciprocating tachycardia. However, atrial tachycardia cannot be terminated by an AV block.

12. What is the first-line drug for hemodynamically stable narrow complex tachycardia in the presence of ischemic heart disease?

A. Intravenous adenosine
B. Intravenous verapamil
C. Intravenous esmolol
D. Intravenous digitalis

Answer: B

If intravenous adenosine is contraindicated (patients with bronchial asthma and ischemic heart disease), intravenous verapamil is recommended as the first-line agent.

13. What is the most common cause of wide complex tachycardia?

A. Aberrant supraventricular tachycardia
B. Antidromic atrioventricular reciprocating tachycardia
C. Ventricular tachycardia
D. Pacemaker-mediated tachycardia

Answer: C

Ventricular tachycardia is the most prevalent cause of wide complex tachycardia (about 80%), especially in patients with a history of heart disease (>90%).

14. What is the least useful electrocardiographic sign for the diagnosis of ventricular tachycardia?

A. Negative concordance
B. AV dissociation
C. 2:1 VA association
D. Positive concordance

Answer: D

A definitive ECG diagnosis requires AV dissociation, capture or fusion beat, and a VA association of 2:1 or more. Negative concordance is strongly suggestive of ventricular tachycardia. Positive concordance also suggests ventricular tachycardia, but not as strongly as negative concordance. Antidromic atrioventricular reciprocating tachycardia using the left posterior accessory pathway typically produces positive concordance.

15. What is the most likely cause of polymorphic ventricular tachycardia in the presence of a normal QT interval?

A. Myocardial ischemia
B. Hypertrophic cardiomyopathy
C. Channelopathies
D. Dilated cardiomyopathy

Answer: A

Polymorphic ventricular tachycardia may occur in the setting of a normal or prolonged QT interval. For patients with a normal baseline QT interval, the most likely cause is myocardial ischemia. However, cardiomyopathies (hypertrophic and dilated) and channelopathies (Brugada syndrome and catecholaminergic polymorphic ventricular tachycardia) may present with polymorphic ventricular tachycardia.

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CHAPTER 13

CARDIAC IMPLANTABLE ELECTRONIC DEVICES

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1. What is the NBG (NASPE/BPEG Generic) code for a cardiac resynchronization therapy pacemaker (CRT-P)?

A. VVVRV
B. DDDRD
C. DDDRV
D. VVVRD

Answer: C

In CRT-P, multisite pacing occurs only in the ventricle, therefore, DDDRV is the correct answer.

2. What is the NBD code for a dual-chamber defibrillator with capability for transthoracic impedance measurement?

A. DDED
B. DDHD
C. VVED
D. VVHD

Answer: D

In a dual-chamber defibrillator, the right ventricular lead is responsible for the shock and the anti-tachycardia pacing, but both the atrial and ventricular leads may be involved in the anti-bradycardia pacing. A defibrillator with the capability for hemodynamic measurement in addition to electrogram detection gets an "H" in the third NBD position. Therefore, the correct NBD code in this setting is VVHD.

3. In which of the following conditions is an epicardial pacemaker not indicated?

A. Large ventricular septal defect
B. Large atrial septal defect
C. Persistent left superior vena cava
D. Mechanical tricuspid valve

Answer: C

Although epicardial systems have mainly been replaced by transvenous systems, there are a few situations where one is indicated, including the absence of venous access, congenital cardiac anomalies, intracardiac shunts, tricuspid prosthesis, and for small children. Anomalies such as a persistent left superior vena cava is not an indication for an epicardial pacemaker, although transvenous pacemaker implantation is challenging in this setting.

4. Which of the following conditions is not suitable for subcutaneous ICD implantation?

A. High risk congenital long QT syndrome
B. Pacemaker-dependent patient with history of prior device infection
C. Primary prevention of dilated cardiomyopathy
D. Prosthetic tricuspid valve

Answer: B

Subcutaneous ICD is not indicated in pacemaker-dependent patients, even with a history of prior device infection, because this device has no reliable anti-bradycardia pacing.

5. Which mode change is not possible upon magnet application?

A. AAIR to AOOR
B. VDD to VOO
C. VVI to VOO
D. DDI to DOO

Answer: A

AAI and AAIR modes revert to AOO (not AOOR) upon magnet application. VDD and VVI revert to VOO and DDDI to DOO mode.

6. Total atrial refractory period is equal to:

A. PVAB plus PAV
B. PVAB plus SAV
C. PVARP plus SAV
D. PVARP plus PAV

Answer: C

The total atrial refractory period (TARP) is equal to SAV plus PVARP.

7. Which method is mainly designed to avoid appropriate ICD shock?

A. Programming an algorithm to avoid T-wave oversensing
B. Programming an algorithm to avoid lead noise
C. Programming ATP in the VF zone
D. Programming a longer detection interval

Answer: C

Algorithms designed to avoid oversensitivity to T waves and lead noise are mainly designed to avoid inappropriate ICD therapy. A longer detection interval is mainly applied to avoid inappropriate therapy for nonsustained VT. Therefore, programming ATP in the VF zone is used to convert fast VT in the VF zone by the delivery of ATP rather than a shock.

8. Which cardiac vein is not recommended for left ventricular lead implantation during cardiac resynchronization therapy?

A. Anterolateral cardiac vein
B. Anterior interventricular vein
C. Lateral cardiac vein
D. Posterolateral cardiac vein

Answer: B

Lateral branches of the coronary venous system are the most appropriate locations for LV lead implantation. The middle cardiac vein and anterior interventricular vein are not appropriate targets for LV lead implantation.

9. In a patient who presents with loss of capture within the first week after pacemaker implantation, which condition is the least likely to be responsible for this malfunction?

A. Lead dislodgement
B. Lead maturation
C. Loose connection
D. Electrolyte disturbance

Answer: B

Lead maturation arises because of the trauma to cells surrounding the electrode and this process requires 6-8 weeks. Therefore, it is not an early complication. However, lead dislodgement and loose connection are two of the most commonly observed malfunctions. Electrolyte abnormality may result in loss of capture any time after device implantation.

10. Which drug is most likely to increase the pacing threshold?

A. Amiodarone
B. Mexiletine
C. Dronedarone
D. Flecainide

Answer: D

Class IC antiarrhythmics are the most common drugs that may increase the pacing threshold.

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1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition. 2017.
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CHAPTER 14

BRADYARRHYTHMIAS

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1. Which presentation of sinus node dysfunction is easily missed in clinical practice?

A. Sinus bradycardia
B. Chronotropic incompetence
C. Sinus arrest
D. Sinoatrial exit block

Answer: B

Chronotropic incompetence is often missed in clinical practice, which may partly be related to the absence of universally accepted diagnostic criteria.

2. How can we differentiate a first-degree SA exit block from sinus arrest?

A. Invasive electrophysiology study
B. Ambulatory ECG monitoring
C. Progressive P-P interval shortening before a pause in surface ECG
D. P-P interval surrounding the pause is a multiple of baseline P-P interval in surface ECG

Answer: C

Progressive P-P interval shortening before a pause is the most useful sign to differentiate between a first-degree SA exit block and a sinus arrest.

3. Which kind of sinus node dysfunction is not discoverable from a surface ECG?

A. Sinus arrest
B. First-degree SA exit block
C. Type I second-degree SA exit block
D. Type II second-degree SA exit block

Answer: B

A first-degree SA exit block cannot be recognized from a surface ECG and requires an invasive electrophysiology study.

4. A 60-year-old man presented with easy fatigability during daily activities. During an exercise test, he could achieve a maximal heart rate of 110 beats per minutes. What is your diagnosis?

A. Coronary artery disease
B. Congestive heart failure
C. Chronotropic incompetence
D. Pulmonary disease

Answer: C

A 60-year-old man should be able to achieve a heart rate equivalent to 80% of 220 minus his age, i.e. (220 – 60) x 0.8 = 128. Therefore, he should reach a heart rate of at least 130 beats per minute. Patients with coronary artery disease, heart failure, and pulmonary disease often exhibit exaggerated heart rate responses during exercise tests.

5. What do you recommend for the evaluation of a sick sinus syndrome after surface ECG?

A. Ambulatory ECG monitoring
B. External event recorder
C. Implantable loop recorder
D. Invasive electrophysiology study

Answer: A

After a comprehensive history, physical examination, and a 12-lead ECG, ambulatory ECG monitoring may help to diagnose SSS. If surface ECG and repetitive ambulatory ECG monitoring of 1 to 14 days are incapable of documenting the cause of a patient's symptoms, an external event recorder or an implantable loop recorder should be considered. Invasive electrophysiology study is the last step in diagnostic evaluation of SSS.

6. A 50-year-old woman is referred because of frequent asymptomatic 2-second pauses in ambulatory CG monitoring. What do you recommend?

A. Pacemaker implantation
B. Invasive electrophysiology study
C. Implantable loop recorder
D. Reassurance

Answer: D

An asymptomatic sinus pause, especially of less than three seconds, needs no treatment.

7. In which kind of AV block may invasive electrophysiology study have a role?

A. First-degree AV block
B. Type I second-degree AV block
C. 2:1 AV block
D. Advanced second-degree Av block

Answer: C

Except for 2:1 AV blocks, invasive electrophysiology studies have no significant role in decision-making for patients with AV blocks.

8. A 55-year-old obese woman is referred for episodes of AV block in polysomnography. She had a 3-second complete AV block during an apneic phase of sleep apnea. What do you recommend?

A. CPAP
B. Pacemaker implantation
C. Ambulatory ECG monitoring
D. Invasive electrophysiology study

Answer: A

Episodes of AV block during apneic phases of sleep apnea need no further evaluation or treatment, except for that provided for the sleep apnea.

9. In which scenario is an external event recorder most likely to reveal the underlying cause of a patient's symptoms?

A. Symptoms occurring every year
B. Symptoms occurring every two months
C. Symptoms occurring every six months
D. Symptoms occurring more than once a month

Answer: D

External event recorders are most useful in the context of symptoms that occur more than once a month.

10. What is the most common cause of acquired AV block?

A. Lenegre-lev disease
B. Ischemic heart disease
C. Cardiomyopathies
D. Trauma

Answer: A

Lenegre-lev disease or idiopathic degeneration is the most common cause of acquired AV block.

11. The usual anatomic site for type I second-degree AV block is the ....

A. AV node
B. His bundle
C. Left bundle branch
D. Purkinje system

Answer: A

The usual anatomic site for type I second-degree AV block is the AV node.

12. What is the most common tachycardia in patients with tachy-brady syndrome?

A. Atrial tachycardia
B. Atrioventricular nodal reentrant tachycardia
C. Atrial fibrillation
D. Ventricular tachycardia

Answer: C

A rapid heart rate is mainly caused by atrial fibrillation or flutter in tachy-brady syndrome patients.

13. Which diagnostic test is more useful for the differentiation between SSS and ischemic heart disease?

A. Exercise stress testing
B. Ambulatory ECG monitoring
C. External event recorder
D. Implantable loop recorder

Answer: A

Exercise stress testing is the best test for differentiation between SSS and ischemic heart disease.

14. What is the correct definition for advanced second-degree AV block?

A. Every other P wave is nonconducted
B. At least two consecutive P waves are nonconducted
C. There is progressive PR interval prolongation before nonconducted P waves
D. Occurrence of a single nonconducted P wave without prior PR interval prolongation

Answer: B

Advanced second-degree AV block is characterized by at least two consecutive nonconducted P waves.

15. In which setting would exercise testing and ambulatory ECG monitoring be useful to establish a correlation between symptom and rhythm in patients with an AV block?

A. Persistent first-degree AV block
B. Persistent type I second-degree AV block
C. Intermittent AV block
D. Transient postoperative AV block

Answer: C

In patients with intermittent AV block, exercise testing and ambulatory ECG monitoring are important to establish a correlation between symptoms and rhythm in patients with AV block

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CHAPTER 15

PREVENTIVE CARDIOLOGY

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1. Which statement is not true?

A. According to the 2007 USPSTF guidelines, annual screening of those with an SBP between 120 and 139 mmHg is recommended.
B. Isolated systolic hypertension is more predictive of CV events than isolated diastolic hypertension in youth.
C. The target BP in diabetics is the same as in the general population.
D. All antihypertensive drugs (including beta-blockers) have the same effect on all-cause mortality.

Answer: D.

All four of the recommended classes (CCB, ACE inhibitors, ARB, and diuretics) have a similar effect on HTN, CV events and all-cause mortality, but beta blockers are less efficient for reducing all-cause mortality.

2. Which statement is not true?

A. The predictive value of non-fasting LDL-C is similar to fasting level.
B. In those with moderate CKD (eGFR between 30 and 59 cc/min/1.73m2), the recommended LDL-C and non-HDL-C targets are 100mg/dl and 130 mg/dl respectively.
C. HDL-C level is a predictive factor but not a target one.
D. Statins have more efficacy for secondary prevention than primary prevention.

Answer: D

Statins have similar efficacy on primary and secondary preventions.

3. A 55-year-old obese male patient presenting well-controlled DM without any CV events has been referred to clinic with the following lab tests:

TG = 355mg/dl, LDL-C = 90 mg/dl, HDL-C = 40 mg/dl

His estimated 10-year ASCVD risk score is 6.5%. What is the best management?

A. Rosuvastatin 20 QD
B. Atorvastatin 20 QD
C. Fenofibrate 100 QD
D. No anti-lipid treatment is needed.

Answer: A

According to ACC/AHA 2013 guidelines, intensive statin therapy (Atorvastatin 40-80 or Rosuvastatin 20-40) is recommended for diabetic patients without clinical ASCVD who are between 40 and 75 years old and have LDL-C levels between 70 and 189 mg/dl.

4. According to recent meta-analysis, intensive glucose control in diabetics has more favorable effect on…

A. All-cause mortality
B. CV mortality
C. Stroke
D. Non-fatal MI

Answer: D

Several trials have shown that intensive glucose reduction in diabetics has a favorable effect on microvascular outcomes but not on CV events or all-cause mortality, and even some showed an increase all-cause mortality. However, in recent meta-analyses, more intensive glucose control has been shown to decrease the incidence of non-fatal MI events.

5. A 37-year-old alcoholic man has been referred to the hypertension clinic. His BP is 150/90 mmHg and his BMI is 32 kg/m^2 despite dietary adherence and regular physical activity. Which one is not recommended?

A. Start of antihypertensive agents
B. Stop alcohol drinking
C. Bariatric surgery
D. Start orlistat

Answer: C

Bariatric surgery is recommended for those with a BMI of more than 40 kg/m^2 or those with a BMI of more than 35 kg/m^2 who have comorbidities and have failed to lose weight through behavioral change and pharmacologic therapy.

6. Which statement is true?

A. Fish consumption reduces non-fatal MI.
B. Monounsaturated FAs decrease HDL-C with no effect on CV events.
C. A high-potassium diet reduces stroke rates.
D. Stringent salt reduction decreases all-health risk, especially in heart failure patients.

Answer: C

Eating fish decreases fatal CHD and stroke rates but not total CHD or non-fatal MI. Mono-unsaturated FAs increase HDL-C and it seems that stringent salt reduction may increase all-health risk in heart failure patients.

7. Which one of the following statements about CPR is not true?

A. Baseline hsCRP in asymptomatic people is predictive of long-term all-cause mortality.
B. HsCRP has more predictive value in those with intermediate CVD risk.
C. For CV risk stratification, it is not recommended to measure hsCRP level more than once.
D. After statin initiation, the magnitude of hsCRP reduction is independent of the magnitude of LDL-C reduction.

Answer: C

It is better to measure fasting or non-fasting hsCRP level twice, at least two weeks apart, for CV risk stratification.

8. Which of the following statements about cardiac rehabilitation is true?

A. The rate of mortality and MI reduction is similar in each component of CR.
B. About 70% of eligible patients are referred for CR.
C. Before initiating exercise training, symptom-limited exercise is not a necessary for all patients.
D. The intensity of exercise should begin at 80% of maximal heart rate.

Answer: A

Only 20% of eligible patients are referred for CR. Symptom-limited exercise should be performed before initiating exercise training, and the intensity of exercise should begin at 60-70% of maximal heart rate.

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CHAPTER 16

HYPERTENSION

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1. Regarding the various mechanisms of hypertension, which sentence is correct?

A. Carotid baroreceptors become desensitized and reset by repeated high blood pressure.
B. Sympathetic tone is decreased in obese persons.
C. In sleep apnea syndrome, hypoxic episodes desensitize chemoreceptors.
D. High salt intake in hypertensive patients inhibits the Renin-Angiotensin-Aldosterone (RAS) system.

Answer: A

Carotid baroreceptors become desensitized and reset by repeated high blood pressure. In obese persons, sympathetic tone is increased to burn fat. This results in an elevated SVR. In sleep apnea syndrome, nocturnal hypoxic episodes sensitize chemoreceptors. We expect the inhibition of RAS pathway when someone has a high salt intake, but in some patients, increased sympathetic tone leads to renin release from the kidneys and the persistence of RAS system activity.

2. Which of the following items is a cause of renovascular hypertension?

A. Glomerulonephritis
B. Pyelonephritis
C. Chronic kidney disease
D. Fibromuscular dysplasia

Answer: D

Stenosis of the main stem of the renal arteries or even a branch or accessory renal artery can decrease glomerular perfusion of the affected area. The results are increased renin secretion and high blood pressure. In women younger than 60 years old, the cause is often fibromuscular dysplasia which affects the distal two thirds of the renal arteries with a beading pattern. In older adult patients, especially men, the cause is mostly atherosclerosis.

3. Which of the following items can be recommended for a better control of hypertension?

A. Higher intake of proteins
B. Lower ingestion of magnesium
C. High amounts of beverages
D. Lower intake of dairy products

Answer: A

Dietary Approaches to Stop Hypertension, known as the DASH diet, includes almost every dietary change with confirmed evidence to lower blood pressure:

- High amounts of fruits and vegetables
- Low-fat dairy products
- Fish and poultry
- Whole grains and nuts
- Foods rich in potassium, calcium, magnesium, protein, and fiber
- Lower amounts of red meat, cholesterol, total fat, saturated fat, sweets, and sugar-sweetened beverages

4. In which situation is the use of diltiazem as an antihypertensive drug more appropriate?

A. Asthma
B. Bradycardia
C. HOCM
D. Pregnancy

Answer: C

Non-dihydropyridine calcium channel blockers such as diltiazem and verapamil are weak antihypertensive drugs (verapamil is weaker); however, they are good options for hypertrophic obstructive cardiomyopathy.

5. Which side effect is not a sufficient reason to discontinue the culprit antihypertensive drug?

A. Creatine rise of about 20% above baseline
B. Cough
C. Angioedema
D. Gingival hyperplasia

Answer: A

Gingival hyperplasia is a rare calcium channel blocker side effect; drug discontinuation is necessary in such cases. Creatinine increases of up to 30% above the baseline are acceptable in CKD patients receiving RAS inhibitors and do not require drug discontinuation. Cough and angioedema are among the adverse effects of ACE inhibitors.

6. In truly resistant hypertension, what should be the first step?

A. Maximizing antihypertensive agents' doses
B. Using add-on drugs
C. Renal denervation
D. Searching for possible secondary causes

Answer: D

In resistant hypertension we should encourage life-style changes and search for probable secondary causes of hypertension. Then we need to maximize antihypertensive agents' doses and use add-on drugs.

7. Which of the following is a characteristic finding in gestational hypertension?

A. Proteinuria more than 300 mg/day
B. Renal failure
C. Hypertension after 20 weeks of gestation
D. Doubling of hepatic enzymes

Answer: C

Gestational hypertension is the initiation of hypertension after 20 weeks of gestation without preeclampsia features. Preeclampsia is the same as gestational hypertension, but with one of the following conditions:

- Proteinuria (>300 mg/day)
- Thrombocytopenia (less than 100,000/μL)
- Renal failure
- Liver function impairment (doubling of hepatic enzymes)
- Pulmonary edema
- Visual or cerebral symptoms

8. In which situation is there less urgency to reduce the blood pressure level?

A. Aortic dissection
B. Ischemic stroke
C. Preeclampsia
D. Bleeding from postop suture lines

Answer: B

In some hypertensive emergencies the blood pressure must be lowered much more rapidly. Examples are aortic dissection, preeclampsia, and bleeding from suture lines after surgeries. In acute ischemic strokes, if there is an indication to administer thrombolytics, the blood pressure should be lowered to under 185/110 mmHg. If there is no indication to administer thrombolytics, antihypertensive drugs must be reserved only for those with blood pressures of more than 220/120 mmHg.

9. Which drug is the first choice in hypertension associated with hemorrhagic stroke?

A. Labetalol
B. Hydralazine
C. Nitroglycerine
D. Nitroprusside

Answer: A

In hemorrhagic strokes labetalol or nicardipine are used to lower systolic BP to less than 140 mmHg. It is better to avoid nitroprusside and hydralazine.

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CHAPTER 17

DYSLIPIDEMIA

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1. Microsomal triglyceride transfer protein (MTP) is responsible for production of which lipoprotein?

A. Primordial VLDL
B. Chylomicron
C. LDL
D. Phosphatidylserine

Answer: A

2. Which one of the genetic abnormalities mentioned below produces a different phenotype than others?

A. LDL-receptor mutations
B. Apo B100 mutations
C. PCSK9 gain-of-function mutations
D. APOE mutations

Answer: D

APOE mutations are responsible for dysbetalipoproteinemia, while all the others are associated with familial hypercholesterolemia

3. Which approach is not recommended if myalgia develops after statin initiation?

A. Dose reduction and uptitration
B. Searching for polymyalgia rheumatica and myopathies
C. Thyroid function test request
D. Shifting to fibrates or niacin instead of statins

Answer: D

Dose reduction or temporary discontinuation of statins followed by uptitration is the best approach in benign muscle symptoms related to statins. Fibrates or niacin are not as effective and are associated with more side-effects than statins.

4. In familial hypercholesterolemia, which medication is not recommended as an adjunct to statins?

A. Bile acid sequestrants
B. PCSK9 inhibitors
C. Anacetrapib
D. Ezetimibe

Answer: C

Anacetrapib is a CETP inhibitor and its main effect is on HDL level.

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CHAPTER 18

ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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1. Which of the following is not an absolute contraindicator for fibrinolytic therapy?

A. Suspicious aortic dissection
B. Malignant intracranial neoplasm
C. Dementia
D. Previous intracranial hemorrhage

Answer: C

Table 18.1 Absolute and Relative Contraindications to Fibrinolytic Therapy

Absolute Contraindications	Relative Contraindications
Previous intracranial hemorrhage	History of poorly controlled, chronic, and severe hypertension
Malignant intracranial neoplasm	Significant hypertension at initial evaluation (systolic blood pressure >180 mmHg or diastolic blood pressure >110 mmHg
Known structural cerebrovascular lesion	Ischemic stroke more than 3 months previously
Ischemic stroke within 3 months except for acute ischemic stroke within 4.5 hours	Known intracranial pathology not covered in Absolute Contraindications
Suspicion of aortic dissection	Dementia
Significant facial trauma or closed-head trauma within 3 months	Traumatic or prolonged cardiopulmonary resuscitation (>10 minutes)
Intracranial and intraspinal surgery within 2 months	Major surgery (within 3 weeks)
Severe uncontrolled hypertension unresponsive to emergency therapy	Recent (within 2-4 weeks) internal bleeding
Active bleeding (except for menstrual period) or bleeding diathesis	Non-compressible vascular puncture
For streptokinase, previous treatment more than 5 days and less than previous 6 months	Pregnancy
	Active peptic ulcer
	Current use of anticoagulants

2. Which of the following are predictors of intracranial hemorrhage in patients with ST-segment elevation myocardial infarction?

A. BP lower than 160 mmHg
B. Prothrombin time less than 24 seconds
C. Male sex
D. Aged 75 or older

Answer: D

Predictors of ICH include: aged 75 or older, black ethnicity, female sex, prior history of stroke, systolic blood pressure ≥160 mmHg, weight ≤65 kg for women or ≤80 kg for men, international normalized ratio >4, prothrombin time >24 seconds, and use of alteplase versus other fibrinolytic agents.

3. Which of the following actions is an indicator for IV nitroglycerin therapy in patients treated without reperfusion?

A. Chest pain after three sublingual nitroglycerin tablets
B. Right ventricular infarction
C. Sever aortic stenosis
D. Pulmonary hypertension within the previous 24 hours

Answer: A

In patients suffering from chest pain after three sublingual nitroglycerin tablets, as well as in patients with hypertension or heart failure, intravenous nitroglycerin therapy is valuable. In the case of right ventricular infarction or severe aortic stenosis, nitrates must be used with caution or avoided due to the risk of serious hemodynamic decompensation.

4. Which of the following conduction abnormalities after a myocardial infarction is associated with a higher in-hospital mortality rate?

A. Left bundle branch block
B. Inferior MI because of a high-grade AV block
C. Inferior MI or anterior MI related to a first-degree block
D. Right bundle branch block

Answer: B

Conduction abnormalities include right bundle branch blocks, left bundle branch blocks, first-degree heart blocks, second-degree heart blocks, high-degree blocks, and third-degree (complete) heart blocks. In inferior MI conduction, disturbances may occur acutely or after hours or days. Sinus bradycardia, Mobitz type I, and complete heart block can also be seen, because the sinoatrial node, atrioventricular node, and His bundle are mainly supplied by the right coronary artery.

Conduction abnormalities which happen with anteroseptal MI are less common but more complicated, and the degree of arrhythmic complications is usually directly related to the extent of the MI. A complete heart block with anterior MI may happen in the first 24 hours. It may happen without warning or may be heralded by the occurrence of a right bundle branch block with either a left anterior fascicular block or a left posterior fascicular block. Heart blocks in this situation seem to result from an extensive infarction that involves the bundle branches traveling within the septum. In patients with an anterior or inferior MI, a high (second- or third-) degree atrioventricular block is associated with increased mortality; in inferior MI, a high-grade AV block is usually transient and is associated with higher in-hospital mortality.

5. In the case of a sustained monomorphic VT associated with angina, pulmonary edema or systolic blood pressure <90 mmHg as a complication of an ST-segment elevation myocardial infarction, what is your suggested treatment?

A. Biphasic waveform defibrillator
B. Unsynchronized electrical shock
C. Immediately synchronized electrical cardioversions at an initial energy level of 100 joules
D. Biphasic defibrillator initial shock at 120-200 joules

Answer: C

Sustained polymorphic VT, very rapid VT, and pulseless monomorphic VT should be treated with unsynchronized electrical shock (defibrillation). Using a biphasic waveform defibrillator is optional because the success rate for defibrillation is greater than with a monophasic waveform defibrillator. For a biphasic defibrillator, the initial shock should be at 120 to 200 joules. For monophasic defibrillation, shocks start at 360 joules.

6. Which of the following is not a predictor of tamponade in patients with ST-segment elevation myocardial infarction?

A. Increasing age
B. increased time from symptom onset to therapy
C. Anterior wall MI
D. Male sex

Answer: D

Predictors of tamponade are increasing age, a previous anterior wall MI, female sex, and increased time from symptom onset to therapy. Tamponade is associated with a higher risk of 30-day mortality. If the patient survives for 30 days, there is no increase in their one-year mortality risk.

7. Which of the following statements about cardiogenic shock due to rupture of the interventricular septum in STEMI is true?

A. Age is not associated with the patient's outcomes.
B. Late selective surgery should not be done for patients with heart failure and the absence of cardiogenic shock.
C. A long delay between septal perforation and surgical repair is associated with poor outcomes.
D. Bypassing the associated CAD does not improve long-term survival rates.

Answer: C

Late elective surgery can be done in patients with heart failure in the absence of cardiogenic shock; however, the risk of rapid and unexpected deterioration is always present. Advanced age and a long delay between septal perforation and surgical repair are associated with poor outcomes. Bypassing the associated coronary artery disease can improve long-term survival.

8. Which of following symptoms in peri-infarction pericarditis increases in-hospital and 1-year mortality rates?

A. The appearance of friction rub after the fourth day of MI associated with larger infarct size
B. Lower ejection fraction
C. Less frequent anterior location of MI
D. Inferior MI

Answer: B

Acute pericarditis, diagnosed by a pericardial friction rub with or without chest discomfort, may complicate the MI. Appearance of a friction rub in the first two or three days following an MI is associated with a larger infarct size, a lower ejection fraction, a more frequent anterior location of the MI, and increased in-hospital and one-year mortality.

9. In STEMI, what is the indication of oral beta blockers in patients treated without reperfusion?

A. Heart block
B. Heart failure
C. Low cardiac output
D. Low risk for cardiogenic shock

Answer: D

Oral beta blockers are recommended for all patients with an acute STEMI without any contraindications generally. Oral beta blocker contraindications include heart failure, signs of low output, a high risk of cardiogenic shock, bradycardia, a heart block, or reactive airway disease.

10. Which of following sentences matches the relative contraindications to fibrinolytic therapy presented in Table 18.1 (above)?

A. Internal bleeding 6 weeks ago
B. Known structural cerebrovascular lesion
C. Pregnancy
D. Passive peptic ulcer

Answer: C

For the relative contraindications, see Table 18.1 (above).

References

1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition; 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 19

PERCUTANEOUS CORONARY

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1. Syntax studies show the main difference between coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCR) in long-term follow up of patients with left an LM lesion is:

A. The need for revascularization
B. MI
C. Death
D. Stroke

Answer: A

In a 5-year follow up for patients with LM disease, CABG and PCI gave the same results for MI, stroke, and death.

2. Which is the best way to assess physiological importance in coronary artery lesions?

A. FFR
B. IVUS
C. Conventional angiography
D. OCT

Answer: A

FFR is the main way to assess physiologic importance in coronary stenosis. Other modalities such as IVUS, OCT, and conventional angiography cannot assess the physiology of coronary stenosis.

3. Why is a drug-eluting stent (DES) preferable to a bare-metal stent (BMS)?

A. Lower elastic recoil
B. Lower risk of endothelial hyperplasia
C. Lower risk of early stent thrombosis
D. Lower risk of late stent thrombosis

Answer: B

The main advantage of a DES over a BMS is that it reduces the rate of restenosis by decreasing the likelihood of endothelial hyperplasia.

4. The main reason for performing PCI in chronic stable angina is…

A. Lower risk of death
B. Lower risk of MI
C. Better function class
D. Lower chance of hospital admission

Answer: C

A PCI in chronic stable angina can lead to better function classes. There are currently no studies that provide clarity on its effect on rates of MI, death or hospital admission in comparison with optimal medical treatment.

5. Which of the following items is not a risk factor for stent thrombosis?

A. Long lesion
B. Complex coronary intervention
C. Diabetes mellitus
D. Stent diameter ≥ 3.5 mm

Answer: D

A smaller stent diameter of less than 3 mm can be a risk factor for stent thrombosis

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1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition; 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 20

TRANSCATHETER THERAPIES FOR STRUCTURAL HEART DISEASE

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1. Which of the following is rarely an indication for percutaneous balloon mitral valvuloplasty (BMV) in patients with rheumatismal mitral stenosis (MS)?

A. A mitral valve area (MVA) of ≤1.5 cm^2 in a symptomatic patient with favorable valve anatomy
B. An asymptomatic patient with an MVA of ≤1 cm^2
C. A symptomatic patient with an MVA of ≥1.5 cm^2
D. New atrial fibrillation (AF) in an asymptomatic patient with an MVA of ≤1.5 cm^2

Answer: C

While (A) and (B) are the established indications for BMV, patients with less than severe MS (MVA of ≤1.5 cm^2) are not generally considered for BMV even if they have increased left atrial pressure or pulmonary hypertension (PAH).[1] Even if it is controversial, new AF in an asymptomatic patient with severe MS has been recently reflected as a more accepted indication for BMV.[2]

2. What is the most common indication for balloon aortic valvuloplasty (BAV) in patients with aortic stenosis (AS) in current practice?

A. Poor candidates for surgical or transcatheter aortic valve replacement (AVR)
B. Emergent high-risk noncardiac surgery
C. Bridge to surgical or transcatheter AVR
D. As an adjunctive procedure during transcatheter AVR

Answer: D

BAV has a limited therapeutic role in modern practice because of the unfavorable short- and long-term results and high rate of complications. Recently, with the advent of transcatheter AVR, BAV is increasingly used as an adjunctive procedure for predilatation, balloon sizing and post-dilatation.[3]

3. Which of the following is not a possible indication for closure of a patent foramen ovale (PFO)?

A. Pulmonary hypertension
B. Recurrent paradoxical embolism
C. Platypnea-orthodeoxia syndrome
D. Decompression illness

Answer: A

PFO closure might be considered in patients with recurrent systemic embolism, mainly transient ischemic attack (TIA), a stroke presumed to be the result of a paradoxical embolism, platypnea-orthodeoxia syndrome, and decompression illness. Isolated pulmonary hypertension is not an indication for PFO closure.

4. Which of the following statements about atrial septal defects (ASD) is false?

A. Sinus venosus and septum primum ASDs are not generally amenable to percutaneous closure.
B. Secundum ASDs are usually located in the septum secundum.
C. The presence of symptoms is not essential in the selection of patients for ASD closure.
D. Paradoxical embolism might be an indication for percutaneous ASD closure.

Answer: B

Secundum ASDs are the most common type and are usually located in the primum portion of the atrial septum. Secundum ASDs are generally the only type which are amenable to percutaneous closure. Patients with significant left-to-right shunt (Qp/Qs>1.5:1) and right ventricular volume overload should be considered for ASD closure even in the absence of symptoms. In addition, ASD closure is indicated by the presence of certain symptoms. Symptoms may vary from dyspnea in one extreme to, less frequently, paradoxical embolism in the other extreme. Some patients with PAH might also be considered for the procedure if there is a net left-to-right shunt of at least 1.5:1, or evidence of pulmonary artery reactivity when challenged with a pulmonary vasodilator.

5. Which of the following statements about transcatheter AVR (TAVR) in patients with severe AS is false?

A. TAVR reduces mortality compared to medical therapy and the majority of patients do well during the long-term follow-up.
B. TAVR is a feasible alternative for surgical AVR in patients who are at intermediate risk for surgery.
C. The same indications as for surgical AVR should be considered in selecting patients for TAVR.
D. Non-transfemoral approaches are less desirable and might be associated with a higher rate of complications.

Answer: A

TAVR is superior to medical therapy, but a fairly large number of patients die soon after TAVR and for others it brings no improvement in their quality

of life.[4] Recently, TAVR has been approved for patients at extreme, high, or intermediate risk for surgery.[5]

References

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CHAPTER 21

AORTIC DISORDERS AND THEIR MANAGEMENT

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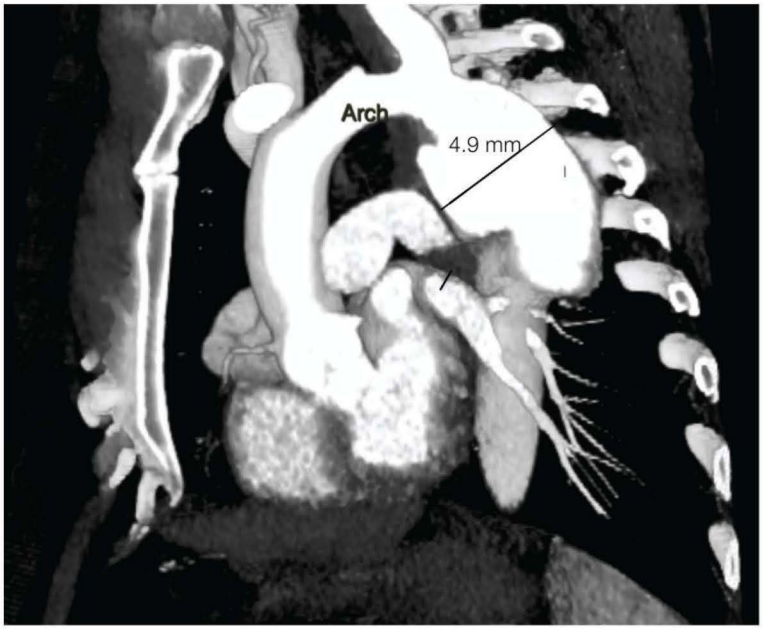
1. A 40-year-old man is referred to the clinic with hoarseness. He has a history of surgical coarctoplasty 15 years ago. His CT scan shows a large saccular aneurysm just after the subclavian artery (see image below). Its measured size is 4.9 cm. What is the best approach for this patient?



A. As the size of the aneurysm is not large enough for intervention, the best approach is imaging surveillance.
B. As the shape of the aneurysm is saccular, the best approach is TEVAR.
C. As it is too close to the subclavian artery, the best approach is surgical arch replacement.
D. Due to the occurrence of this aneurysm at this age, FTAAD highly probable.

Answer: B

A previous history of surgical coarctoplasty and the saccular shape of the aneurysm are both factors that reduce the threshold for any reintervention. Anatomic features, including the arch radius and distance from the left common carotid artery and landing zones, make TEVAR a good and possible option for treatment.

2. A 70-year-old man is referred to the clinic due to a large infra-renal AAA that was found by ultrasound. He has no symptoms in terms of abdominal pain or discomfort. He has a history of diabetes mellitus, systemic hypertension and cigarette smoking for many years. His chief complaint is pain in the distal part of both lower limbs at night and numbness of both lower limbs during the day. His distal pulses are weak. According to his history, what is the *unusual* fact about him?

A. No abdominal symptom is unusual since there is a large AAA
B. Painful distal limbs during night is unusual in this patient
C. History of diabetes mellitus and presence of AAA is unusual
D. His age is unusual, since AAA is diagnosed at lower ages

Answer: C

It has been shown that diabetes mellitus has some protective effect against AAA formation (Table 21.1).

Table 21.1 Risk factors of aneurysm formation of aorta

AAA	TAA
Age>60 The prevalence is 1% between 55-64 years and increases by 2-4% each decade Male sex More than six times more prevalent in men than women Smoking Its association is independent to atherosclerosis, and relates to increase activity of oxidation and proteolysis within the aortic wall. The association is decreased after smoke cessation, although the risk of AAA formation persists for 10 years after cessation. The duration of smoking is more important than the amount smoked. More common in CAD patients HTN May have effects on AAA formation in rats but in human has not been approved yet HLP There are controversial results on the relationship between both higher cholesterol and statin	Cystic Medial Fibrosis (CMF) *HTN:* Aging and HTN lead to degenerative changes within elastic media *Connective tissue disorders:* Marfan syndrome (MFS), Ehlers-Danlos syndrome (EDS), Turner syndrome. MFS is the Fibriline-1 gene mutation, with annuloaortic ectasia. *Bicuspid Aortic Valve (BAV)* is also a congenital condition which is associated with CMF. Annuloaortic ectasia may accompany this disorder. *Familial Thoracic Aortic Aneurysm Syndrome (FTAAS):* It has been shown that many other gene mutations may be involved in TAA formation, apart from MFS or other known syndromes. Although they seem sporadic, further investigations have clarified their familial origin Arteritis *Infective arteritis:* Diseases such as syphilis may be among the

therapy and the risk of AAA Positive family history of AAA This doubles the risk of AAA; it is independent of atherosclerosis and sex Obesity Waist-hip ratio and waist circumference is independently associated with AAA Diabetes mellitus Has protective effect against AAA development	etiologies of TAA. Syphilis, as a cause of TAA, has nowadays faded in importance due to aggressive antibiotic therapy. Infective endocarditis hampers the risk of TAA *Vasculitis:* Large artery vasculitis, Takayasu and giant-cell, are the most important causes. Although ESR rises in giant-cell arteritis, it is not a determinant marker in Takayasu. Chronic Aortic Dissection Imaging surveillance is mandated in uncomplicated type B aortic dissection in order to recognize the progressive dilation of the aorta Atherosclerosis This is not a common cause of TAA. Descending aortic aneurysms have more association with atherosclerosis than ascending and arch aneurisms

3. A 75-year-old man is referred to the emergency department with severe acute chest pain. His vital signs are: PR = 120, RR = 15, BP = 120 mmHg, and O2sat = 96%. His ECG shows ST-elevation of inferior leads and there is a pulse deficit in both upper limbs. Due to suspicion of type A aortic dissection, he has undergone a trans-thoracic echocardiography that reveals small to moderate pericardial effusion and an intimal flap at the ascending aorta with severe aortic regurgitation. Which of the following is correct for the management of this patient?

A. Emergent pericardiocenthesis due to impending tamponade state
B. Maintain BP between 100-110 mmHg with IV Esmolol
C. Emergent surgery should be deferred since the patient is very high risk for surgery
D. TEVAR is the best option since he is high risk for surgery

Answer: B

The patient is high risk for mortality and morbidity during and after surgery; however, he must undergo emergent surgery because it is the only and best therapeutic option for him. To reduce the extension of the dissecting flap, it is best to maintain his BP between 100-110 mmHg with beta blockers.

4. Abdominal aortic aneurysms and distal thoraco-abdominal aneurysms are more frequent in which genetic disorder?

A. MFS: Marfan Syndrome
B. LDS: Loeys-Dietz Syndrome
C. BAV: Bicuspid aortic valve
D. EDS: Ehlers-Danlos syndrome

Answer: D

Ehlers-Danlos Syndrome is divided into three types. In patients with vascular-type EDS, type 3 collagen is abnormal, which can predispose them to aneurysm formation in the abdominal and distal thoracic aorta.

5. A 55-year-old patient is referred to your clinic with a CT angiography. He has a history of type-A aortic dissection, which was treated surgically 5 years ago (Bentall technique). He is anxious and says that the other physician recommended that he should undergo surgery again due to residual intimal flap distal to the repaired ascending aorta. His physical examination is not abnormal and his vital signs are: PR = 70, BP = 140/95 and RR = 10.

His CTA report is as follow: "There is a mechanical prosthetic valve at the aortic position and a graft is seen at [the] ascending aorta without any outpouching at the anastomosis site. After the ascending part there is an intimal flap which extends to the aortic bifurcation and ends up at the right common iliac artery. All arch vessels, as well as the SMA, celiac artery and left renal artery are perfused via the true lumen, but the right renal is perfused via the false lumen but is not hypo-perfused. There is an aneurysm just after the subclavian artery (diameter: 4.5 cm) following another aneurysm at the level of the abdominal branches (diameter: 4.3 cm)."

Which is the best approach for this patient?

A. Imaging follow-up every 6 to 12 months with BP control.
B. The presence of an extensive intimal flap demands immediate surgery or endovascular treatment.
C. The diameter of the aneurysm after subclavian artery and previous history of surgery are indications of TEVAR.
D. Elephant trunk technique is the best for this patient, and should be done as soon as possible.

Answer: A

This is a typical chronic type A aortic dissection which needs imaging surveillance to find aneurysms. The typical sites of aneurysm are i) just after the subclavian artery and ii) at the level of origin of the abdominal branches. The size that needs to be treated accordingly is more than 5.5 cm.

6. Which one of the following statements regarding ultrasound sonography (USG) is not correct?

A. Men over 65 years of age with a history of cigarette smoking benefit from ultrasound assessment for AAA.
B. Women over 65 years of age with a history of cigarette smoking benefit from ultrasound assessment for AAA.
C. Men over 65 years of age without a history of cigarette smoking benefit from ultrasound assessment for AAA.
D. Women over 65 years of age without a history of cigarette smoking benefit from ultrasound assessment for AAA.

Answer: D

To date there is controversy about screening of women for AAA. However, there are some guidelines that recommend ultrasound screening for women over 65 if they have other risk factors for significant atherosclerosis (smoking, CAD or positive familial history) (Table 21.2).

Table 21.2 Guidelines for AA screening

USPSTF guidelines: • Men aged 65-75 who have ever smoked should be screened once for AAA by USG. There is no benefit to repeat screening in men who have negative USG and who are older than 75 years of age. • Men aged 65-75 who have never smoked. There is benefit for routine USG screening in these patients, but the physician can selectively offer screening for AAA to this age group of men. • Women aged 65-75 who have ever smoked. Current evidence is insufficient to assess the balance of benefits and harm from screening women in this age group who have smoked for AAA. • Women who have never smoked. The USPSTF is against routine screening for AAA in women who have never smoked.
ACC/AHA guidelines: • Men aged 65-75 who have ever smoked should undergo a physical examination and a one-time USG.

• Men more than 60 years old who are siblings or the offspring of patients with AAA should undergo a physical examination and a one-time USG.
CSVS guideline: • Men aged 65-75 who are potential candidates for surgery should undergo a USG. • Women older than 65 with multiple risk factors (smoking, family history, cerebrovascular disease) should selectively undergo an USG. • Women older than 65 should not undergo USG routinely.

7. In which of the following scenarios is CTA is mandatory and ultrasound cannot be used as a sole imaging tool?

A. Follow-up of a patient after TEVAR for thoracic aorta aneurysm in the proximal part of the thoracic aorta.
B. Follow-up of a patient with an untreated infra-renal AAA with a diameter of 45 mm.
C. Follow-up of a patient with an untreated thoracic aortic aneurysm just after the left subclavian artery.
D. Follow-up of a post-EVAR patient with no endoleak or size growth in previous imagings.

Answer: B

In cases with borderline sized aneurysms (40-54 mm) and those over 55 mm, CTA is mandated in order to better understand the size and anatomy of the aneurysm. However, in borderline cases (40-54 mm), USG can be used, though not as the sole imaging tool for follow-up. Thoracic aortic aneurysms at the proximal descending aorta can easily be followed up by transesophageal echocardiography (TEE). Even post-TEVAR cases can be followed up in this way (Figure 21.1).

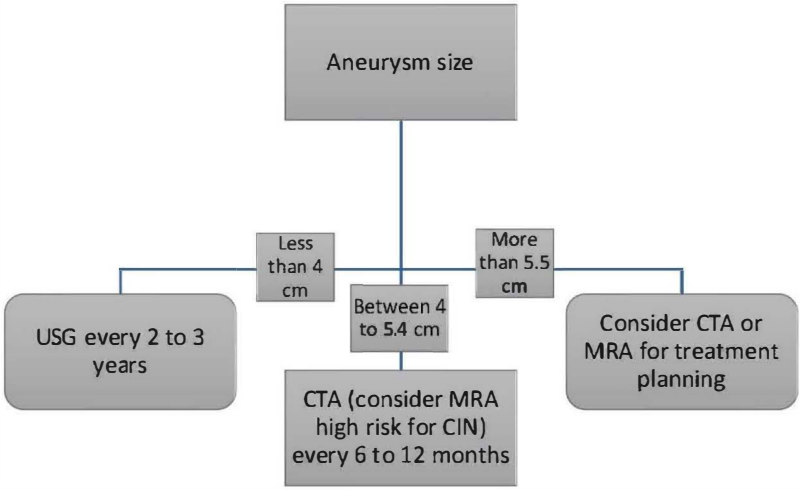


Figure 21.1 Finding an AAA according to one of the screening protocols

8. Which of the following statements about aortic type B dissection variants is *not* true?

A. A penetrating aortic ulcer (PAU) usually causes an extensive dissection and mainly is treated by surgery.
B. Indications of treatment are the same as typical type B aortic dissection.
C. Treatment of intramural hematoma (IMH) is tricky because of extensive aortic involvement that needs to be treated with stent grafts
D. In PAU cases, aortic rupture may occur.

Answer: A

A PAU mostly involves a segment of aorta rather than extensive involvement. Treatment is determined according to factors such as the origin of nearby branches and the type of pathology presented (pseudoaneurysm, perforation, or dissection). Overall the treatment is also segmental. Diagnoses and indications of treatment of type B aortic dissection variants are the same as for typical type B dissections. Like type B aortic dissections, IMH involves a long aortic length. As these cases are

treated by coverage of a considerable length of aorta with stent grafts, treatment is difficult and tends to lead to complications.

9. Which of the following is not an indicator of a ruptured/impending rupture of an AAA?

A. Blue toes in a patient with a known AAA.
B. Hypotension in a patient with an abdominal pulsatile mass without a known AAA.
C. Size of aneurysm more than 60 mm and a pulsatile tender mass in the abdomen.
D. Nausea and vomiting in a case with a history of AAA with a diameter of 50 mm.

Answer: D

AAA is a silent pathology. Once it becomes symptomatic, it means there is high probability of complications and rupture. The main symptoms include sudden abdominal pain and/or distal lower limb embolic events in a case with AAA. It should be noted that other potential causes of these symptoms, such as GI problems or other embolic factors, should be ruled out before marking a patient with an AAA as an "impending rupture". Imaging studies have a very important role in reaching this conclusion. One of the most important markers of a complicated AAA is its size. If the size is over 60 mm, it should be considered the cause of the symptoms even if there are other possible sources, since this size alone designates the AAA as high risk for rupture. The other main factor is the timing of treatment, which is determined by the significance of symptoms and imaging findings.

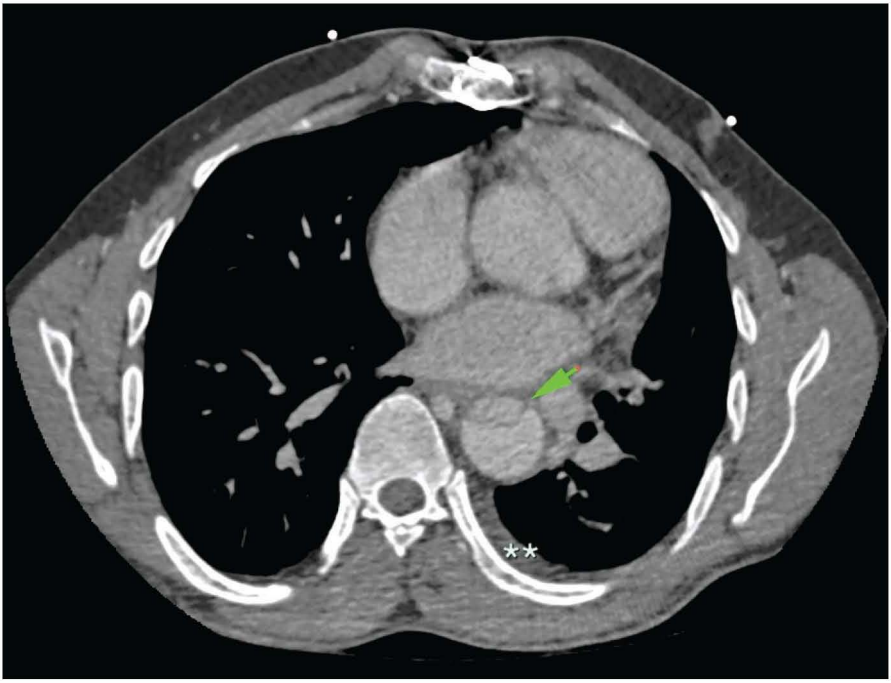
10. Which of the following is *not* an indication for intervention in a patient with acute type B aortic dissection?

A. Uncontrolled severe systemic hypertension
B. Different renal parenchyma brightness in CTA
C. Aortic diameter of 42 mm
D. Small left pleural effusion

Answer: D

A small left pleural effusion (PE) is common in the acute phase of an aortic dissection. This is because of the inflammation process after the dissection.

It should be noted that every patient with a PE must be evaluated for bloody effusion or hemothorax. Using the Hounsfield unit is quite helpful (Figure 21.2).



** marks the pleural effusion.
Arrow marks the dissected descending aorta with intimal flap

Figure 21.2: A case with acute type B aortic dissection and small pleural effusion. The Hounsfield unit and the small nature of the effusion are helpful to rule out a hemothorax.

CHAPTER 22

PERIPHERAL ARTERY DISEASE

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1. The best description for an arterial ulcer is:

A. Shallow, irregularly-shaped
B. Well-demarcated, punched-out
C. Localized tissue necrosis over bony prominences
D. Warm skin necrosis surrounded by callus

Answer: B

Weak pulses, impaired capillary refill, pallor on elevation (dependent rubor), trophic changes, skin discoloration and ulceration or gangrene are some of the manifestations of critical limb ischemia in peripheral artery disease (PAD). Arterial ulcers frequently appear over sites of pressure or trauma or at distal points and characteristically have a well-demarcated "punched-out" appearance with a dry and necrotic wound bed. Venous (A), pressure (C) and neuropathic (D) ulcers are among the differential diagnoses of arterial ulcers.

2. The ankle-brachial index (ABI) is the method of choice to screen for PAD. Which statement about the ABI is not true?

A. An abnormal ABI is associated with higher mortality (more than twice normal).
B. An ABI >1.40 indicates the non-compressibility of the tibial arteries (toe brachial index is indicated for this situation).
C. The sensitivity and specificity of ABI is more than 90%.
D. Advanced age, diabetes and chronic kidney disease may lead to a false positive ABI due to arterial calcification.
E. The severity of PAD and ischemia can be categorized according to the ABI.

Answer: D

The ABI is a diagnostic and prognostic tool for PAD patients which is inexpensive and reproducible. It is the method of choice to screen and establish the diagnosis of lower extremity PAD. An ABI of less than 0.90 is diagnostic with more than 90% sensitivity and specificity. The most important limitation of ABI is arterial calcification and non-compressibility of the tibial arteries, mostly caused by advanced age, diabetes, and chronic kidney disease. The Toe-Brachial Index (TBI) is considered to address this limitation (less than 0.70 is abnormal). Also, ABI is useful to classify PAD as mild (0.70 – 0.90), moderate (0.40 – 0.69) and critical ischemia (less than 0.40).

3. To evaluate and follow the patency of synthetic conduits and stents, which of the following modalities is the best choice?

A. Physical examination
B. Duplex ultrasonography
C. MR angiography
D. CT angiography
E. Digital subtraction angiography (DSA)

Answer: D

DSA is the gold standard for diagnosis of PAD, which is gradually being replaced by noninvasive imaging. Although duplex ultrasonography, CTA, and MRA can all be used to evaluate venous graft patency, duplex ultrasonography is not suitable for patency follow-up of synthetic conduits.

Generally, CTA is superior to MRA for evaluation of stents (metallic or grafted) and is the diagnostic modality of choice.

4. According to AHA/ACC 2016 guidelines on the management of PAD, all of the following patients have increased risk of PAD and need vascular assessment and probably ABI measurement, *except*:

A. A 72-year-old hypertensive woman
B. A 45-year-old diabetic man with a history of smoking
C. A 40-year-old woman with a diagnosis of renal artery stenosis and hypertension without any other risk factors for atherosclerosis
D. A middle-aged male candidate for CABG with a history of premature CAD in his family
E. A 62-year-old man with a family history of PAD

Answer: C

Patients at increased risk of PAD include:

i. Age ≥ 65
ii. Age 50-64 with a risk factor for atherosclerosis or a family history of PAD
iii. Age < 50, with diabetes and one additional risk factor for atherosclerosis
iv. Individuals with known atherosclerotic disease in another vascular bed
v. History and/or physical examination findings suggestive of PAD

5. For PAD patients, symptomatic or asymptomatic, which drug or class of drugs has a class I recommendation for both symptomatic and asymptomatic PAD patients?

A. Aspirin
B. Statins
C. Angiotensin-converting enzyme (ACE) inhibitors
D. Cilostazol
E. Pentoxifylline

Answer: B

According to the updated recommendations of the ACC/AHA guidelines, aspirin in daily doses of 75 to 325 mg has a class I indication in symptomatic PAD individuals and a class IIa indication in asymptomatic patients. Treatment with a high-intensity statin to achieve an LDL level of less than 100 mg/dl for all patients with PAD is supported (class I). ACE inhibitors, irrespective of hypertension, have a class IIa or IIb recommendation for symptomatic and asymptomatic patients with PAD. Cilostazol is a class I recommendation in all symptomatic patients with PAD, but pentoxifylline is not effective for treatment of claudication (class III).

6. For an endovascular approach in vascular occlusions, which levels of arterial or venous disease have the highest risk of fatal bleeding?

A. Aortoiliac, iliocaval
B. Femoropopliteal artery, IVC
C. Femoropopliteal artery, SVC
D. Aortoiliac, IVC
E. Aortoiliac, SVC

Answer: E

If the patient's symptoms and lifestyle fail to improve favorably after first-line treatments, including risk factor modification in addition to exercise and medical therapy, invasive strategies are indicated. An endovascular-first approach is the preferred revascularization technique. Aortoiliac-occlusive disease has the option to be revascularized by covered stents to reduce the restenosis rate (COBEST trial) in addition to minimizing the risk of fatal bleeding. On the other hand, stent fracture, restenosis, and stent thrombosis are the major concerns after SFA intervention. Central vein perforation, although a rare complication in venous interventions, is considered a nightmare if it occurs in the thoracic cage, but behaves benignly in the abdominal or pelvic region (arterial perforation in this territory carries the risk of retroperitoneal hemorrhage or even hemorrhagic shock).

7. In which territory is stenting well established as an effective primary therapy for a nonfocal arterial occlusion?

A. Aortoiliac
B. Common femoral artery (CFA)
C. Superficial femoral artery (SFA)
D. Popliteal artery
E. Tibioperoneal

Answer: A

For treatment of aortoiliac occlusive disease, stenting is an effective primary therapy, especially for nonfocal common iliac occlusions. Stenting of the CFA is generally avoided because of the risk of stent fracture caused by hip movements. In femoropopliteal disease, the efficacy of primary stenting is not well established and only more complex occlusions are treated by primary stenting. In tibioperoneal (below-the-knee) disease, balloon angioplasty alone is the standard of therapy.

8. Cilostazol is an oral phosphodiesterase-3 inhibitor with both vasodilatory effects and a platelet aggregation inhibitory mechanism. All of the below statements about this drug are true, except:

A. Cilostazol improves maximum walking distance by more than 50%.
B. Cilostazol may increase intimal hyperplasia growth.
C. Maximum benefits of cilostazol take 4 months to occur.
D. The adverse effects include tachycardia, diarrhea, dizziness, and an increased tendency to bleed.
E. Its mechanistic similarity to other type III phosphodiesterase inhibitors is the main reason for the contraindication of cilostazol in heart failure patients.

Answer: B

Cilostazol (100 mg twice daily) is a class I recommendation, according to guidelines, to improve symptoms in the absence of heart failure; it should be considered for a primary therapeutic trial in all patients with lifestyle-limiting claudication. Cilostazol improves maximum walking distance by more than 50%. Some data suggest that cilostazol may decrease intimal hyperplasia growth after endovascular therapy and reduce the rate of restenosis.

9. Intervention based on the "angiosome concept" strategy is associated with which arterial territory?

A. Aortic arch
B. Carotid
C. Aortoilaic
D. Femoropopliteal
E. Tibioperoneal

Answer: E

For patients with below-the-knee disease, revascularization is indicated only for limb salvage in the critical limb ischemia (CLI) stage, and claudication rarely mandates an invasive strategy. In patients with CLI with both inflow and outflow occlusions, the inflow level should be treated first. The concept of endovascular intervention in CLI patients is to establish at least a straight-line flow from the hip to the foot ulcer. Intervention according to the feeding vascular territory of the foot ulcer is a relatively new strategy (the angiosome concept) that improves the prognosis of the disease.

10. Commonly, in which arterial territories is an atherectomy device and/or a cutting balloon an option for revascularization?

A. Subclavian and brachial artery
B. Aortoiliac and renal artery
C. Common femoral artery and superior femoral artery
D. Tibioperoneal and popliteal artery
E. Common and internal carotid artery

Answer: C

The main indication of atherectomy and cutting or scoring balloons is severely calcified or non-dilatable occlusions which are resistant to predilation by ordinary balloons. This issue is the primary concern of endovascular therapy, especially in femoropopliteal lesions. The best strategy for common femoral artery occlusions is surgical intervention, especially for young and physically active patients, because of the stent fracture risk caused by hip movements at the CFA region. Adjunctive therapies such as atherectomy devices or cutting balloons may be used to recanalize or to prepare for predilation of femoropopliteal lesions.

CHAPTER 23

CARDIOMYOPATHIES AND MYOCARDITIS

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1. Which statement about peripartum cardiomyopathy (PPCM) is not correct?

A. Nutritional deficiencies have been suggested as the cause of PPCM.
B. Anticoagulation should be avoided in these patients.
C. Vaginal delivery is the preferred method for giving birth.
D. Post-partum use of bromocriptine may be useful in LV recovery.

Answer: B

The exact cause of PPCM is not known, but nutritional deficiency, salt overload, myocarditis, autoimmune processes and genetic causes have all been suggested. The first step in diagnosis is to exclude normal causes of LV systolic dysfunction. Preeclampsia has been proposed as risk factor. For pregnant women with this condition, vaginal delivery is preferred method of birth. Stable patients should be managed medically until spontaneous vaginal delivery. Anticoagulation with heparin should also be instituted because of the risk of thromboembolism in PPCM. Warfarin can be used safely in the third trimester and then switched to heparin before delivery. There is some evidence that post-partum use of bromocriptine and pantoxyphylline could be useful in improving LV systolic function and outcomes in PPCM.

2. A 45-year-old woman has breast cancer and a chemotherapy regimen with doxorubicin has been chosen for her. Which of the following items about her cardiovascular follow-up is *not* correct?

A. Cardiotoxicity is very rare at total doses <450 mg/m^2.
B. Using Dexrazoxane may reduce the risk of cardiotoxicity.
C. Periodic measurements of cardiac biomarkers are not useful in predicting patients who need further cardiac assessment.
D. Aggressive heart failure medical treatment with beta blockers and ACEI should be started for all patients with LV dysfunction following chemotherapy.

Answer: C

Cardiac function should be assessed serially by echocardiography, particularly in those receiving anthracyclines and monoclonal antibodies. Cardiotoxicity is very rare at total doses < 450 mg/m^2 of anthracyclines. Because cardiotoxicity may occur with lower doses, it is highly recommended to monitor cardiovascular function before, during, and after completion of the treatment. It would be useful to have baseline and periodic measurements of biomarker concentrations to identify patients who need further cardiac assessment. Reducing the cumulative dose of anthracyclines, administration of it as infusion rather than a bolus, and the use of dexrazoxane (an EDTA-like chelator) may reduce the risk of anthracycline-induced cardiotoxicity.

3. A 57-year-old man presents with bilateral lower limb pitting edema. In the physical exam he seems pale and has purpura around his eyes, his jugular venous pulse (JVP) has increased, his heart sounds muffled and he has bilateral pitting edema up to his knees. His CXR reveals bilateral pleural effusion. The transthoracic echocardiography is compatible with RCM. Which of the following diagnostic tests would be best for him as a first step?

A. Light chain assay
B. Cardiac MR
C. Endomyocardial biopsy
D. Genetic test

Answer: A

For diagnosis of amyloidosis' clinical manifestations, imaging features, blood and tissue analysis should be considered. About 50% of patients with AL amyloidosis have cardiac involvement. AL cardiac amyloidosis is manifested with rapidly progressive heart failure in which the right sided heart failure signs are more prominent. In AL amyloidosis, monoclonal gammopathy can be detected in the blood and urine by immune electrophoresis methods. A bone marrow biopsy, would show increased plasma cells.

4. Which of the following items is the most useful technique for estimating the myocardial iron deposition in patients suspected of hemochromatosis?

A. Strain imaging echocardiography
B. The evaluation of T2* relaxation time by CMR
C. Endomyocardial biopsy
D. Serum ferritin level

Answer: B

Imaging techniques such as echocardiography and CMR have diagnostic and prognostic significance in hemochromatosis. Conventional echocardiographic measures of systolic and diastolic parameters are insensitive for detecting iron overload of the heart in the early stages. Strain rate imaging is a more sensitive measure to detect the effects of iron overload on cardiac function and allow early detection of systolic and diastolic dysfunction in these patients. The evaluation of the T2* relaxation time by CMR is a useful noninvasive technique for estimating myocardial iron deposition as well as the response to iron chelation therapy. Given the inherent risks of an endomyocardial biopsy, it is not routinely used for the diagnosis of hemochromatosis

5. Which hemodynamic sign is similar in RCM and constrictive cardiomyopathy?

A. The difference between end diastolic left ventricular and right ventricular pressure
B. The respiratory variation in the right atrial pressure waveform
C. The respiratory gradient of pulmonary capillary wedge pressure and left ventricular pressure
D. Dip and plateau (square root sign) of ventricular pressure during diastole

Answer: D

Practical points for differentiating RCM and CP:

- The equalization is more pronounced in CP than RCM. It means the difference between end diastolic left ventricular and right ventricular pressure is ≤ 5 mmHg in CP but > 5 mmHg in RCM.
- Right ventricular end diastolic pressure is more than 1/3 of right ventricular systolic pressure in CP but not in RCM.
- The respiratory variation in right atrial pressure waveform is absent in CP but is present in RCM.
- The respiratory gradient of pulmonary capillary wedge pressure and left ventricle is ≥ 5 mmHg in CP and < 5 mmHg in RCM.
- The pulmonary artery pressure is not elevated in CP (usually < 50 mmHg) but in RCM the PAP is usually more than 50mmHg.

Similarities between CP and RCM:

- Dip and plateau (square root sign) of ventricular pressure during diastole is seen in both CP and RCM.
- In right atrial waveform a prominent "y" descend is seen in both CP and RCM.

6. Which treatment may be harmful in a patient with newly diagnosed HOCM who has a 100 mmHg LVOT pressure gradient at rest and a systolic PAP of 60 mmHg?

A. Verapamil
B. Propranolol
C. Metoprolol
D. Disopyramide

Answer: A

Treatment of Obstructive HCM:

- Dehydration and alcohol should be avoided, so diuretics should be used with caution.
- Vasodilators such as nitrates and phosphodiesterase 3 inhibitors are contraindicated.
- Digoxin should not be used in patients with LVOT obstruction.
- The initial treatment in patients with obstructive HCM is a non-vasodilating beta blocker and titrate it to maximally tolerated dose. If beta blocker therapy fails, disopyramide (maximum dose 400-600 mg/daily) can be used.
- Verapamil can be used when beta blockers are contraindicated. Verapamil should be used with cautious in patients with a severe LVOT gradient (> 100 mmHg) or pulmonary hypertension because it may provoke pulmonary edema.

7. A 37-year-old woman with a history of HOCM, who is treated with 120 mg propranolol daily, presents with AF rhythm and rapid ventricular response. Several hours later, she develops pulmonary edema.

Her vital signs are: SBP = 60/pulse, HR = 120/min (AF Rhythm), RR = 40/min, Afebrile
TTE results are: LVEF = 50%, LVOT pressure gradient = 90 mmHg, Moderate RV dysfunction, sPAP resut: 80mmHg
Lab Data: PH = 7.30, Lactate = 5, Na = 140 mEq/L, K = 4 mEq/L, Creatinine = 1.4 mg/dL

Which is the best management option?

A. IV furosemide and phenylephrine
B. IV furosemide and norepinephrine
C. IV fluid and norepinephrine
D. IV fluid and phenylephrine

Answer: D

Vasodilators and inotropes are contraindicated in HCM patients with severe provocable LVOT obstruction who have developed hypotension and pulmonary edema. The recognition of this condition is very important and the treatment consists of intravenous vasoconstrictors (phenylephrine, metaraminol and norepinephrine) and beta blockers.

8. A young man presents with a severe pleuritic chest pain. He has a history of allergic asthma and a history of a recent common cold. On arrival, his vital signs are stable. His ECG shows sinus tachycardia with frequent PVCs and PACs. Transthoracic echocardiography shows an LVEF of 50% with mild TR. Which of the following is the least helpful diagnostic approach?

A. A panel of viral serology
B. Coronary angiography
C. Cardiac MR
D. Endomyocardial biopsy

Answer: A

The most common etiologies of myocarditis are viral infections and post-viral immune-mediated responses. Myocarditis may be seen in all ages but it is more frequent in younger individuals. Myocarditis should be suspected based on clinical, electrocardiogram, laboratory and imaging findings in the absence of significant coronary artery disease (≥ 50% stenosis) in coronary angiography. Transthoracic echocardiography (TTE) should be performed in all patients with suspected myocarditis. Cardiovascular magnetic resonance findings should be consistent with myocarditis (Lake Louise criteria). CMR can be considered before an endomyocardial biopsy (EMB) in stable patients and EMB should not be replaced by CMR in critically ill patients. Routine viral serology measurement is not recommended. Serum levels of cardiac auto-antibodies should be assessed if possible, depending on the center's expertise.

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1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition; 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 24

VALVULAR HEART DISEASE

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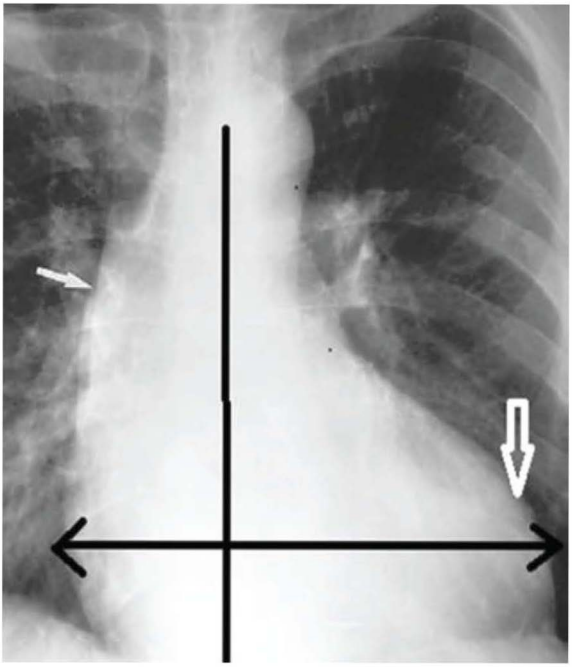
1. Increased LV systolic pressure, increased LV end-diastolic pressure, prolonged systolic ejection time, reduced coronary perfusion pressure, and compression of intra-myocardial coronary arteries are seen in:

A. Severe MR
B. Severe AI
C. Severe AS
D. Severe MS

Answer: C

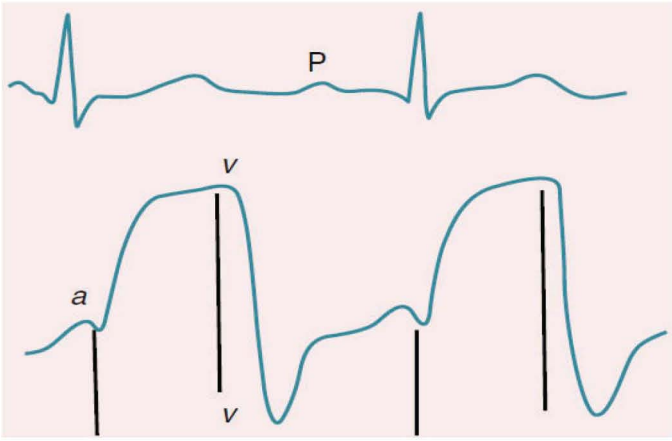
2. What is your diagnosis:



A. Severe AS
B. Severe AI
C. Severe MS
D. Severe TR

Answer: B

3. Based on the following jugular venous waveform, what is your diagnosis?



A. Severe MR
B. Severe MS
C. Severe TS
D. Severe TR

Answer: D

4. The PISA radius in a patient with tricuspid regurgitation by Nyquist's limit of 28 cm/s was 7mm; what is the grade of severity of TR according this parameter?

a. Mild
b. Moderate
c. Severe
d. Indecisive

Answer: C

Table 24.1 Echocardiographic Evaluation of Tricuspid Regurgitation Severity

Tricuapid Regurgitation Severity	**Mild**	**Modarate**	**Severe**
Jet area CFI (cm^2)	<5	5-10	>10
Vena contracta Width	Not defined	0.7 cm	> 0.7 cm
Jet contour and density	Soft and parabolic jet	Variable contour	Dense, triangular, and early peaking
Hepatic vein flow study	Dominant systolic flow	Blunted systolic flow	Systolic flow reversal in the hepatic vein
RA and RV size	Normal RA and RV size	No or mild RA enlargement; normal RV size	RA and RV enlargement
PISA radius with Nyquist's limit 28 cm/s (mm)	<6	6-9	>9

5. With a mitral inflow deceleration time of 758 ms, which of the following is a estimate of the mitral valve area?

A. 0.5 cm^2
B. 1 cm^2
C. 1.5 cm^2
D. 2 cm^2

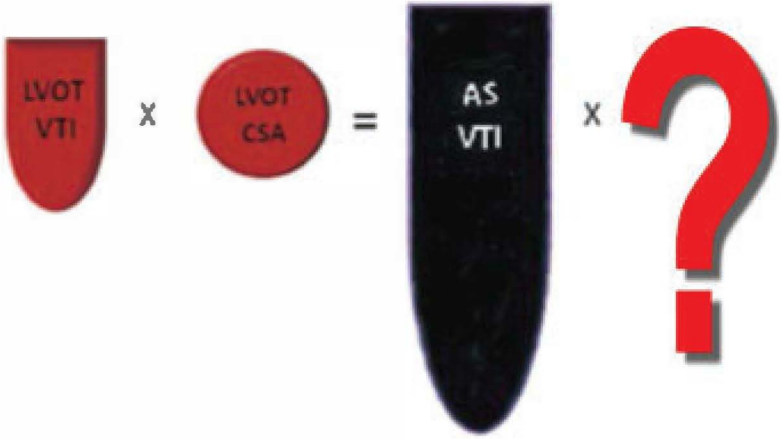
Answer: B

6. With a peak aortic valve velocity of 5 m/s and a peak LVOT velocity of 2 m/s, which is the correct calculation of peak AV gradient?

a. 84 mmHg
b. 116mmHg
c. 100 mmHg
d. 36 mmHg

Answer: A
$\Delta p = 4 \times (V2^2 - V1^2) = 100 - 16 = 84mmHg$

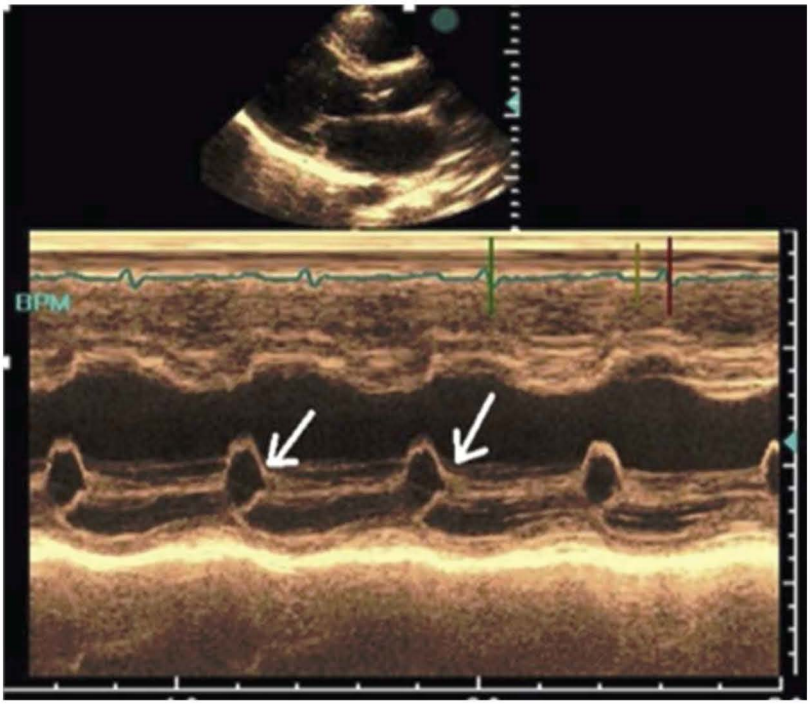
7. What is the **?**



A. Aortic valve area
B. Aortic valve velocity
C. Aortic valve mean gradient
D. LVOT velocity

Answer: A

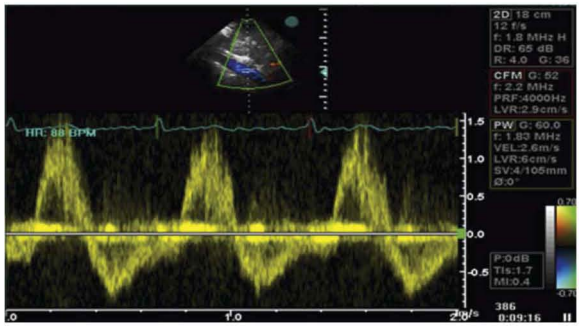
8. What is your diagnosis?



A. Premature closure of the mitral valve in acute severe mitral regurgitation
B. Premature closure of the mitral valve in acute severe aortic regurgitation
C. Premature opening of the mitral valve in acute severe aortic regurgitation
D. Premature opening of the mitral valve in acute severe mitral regurgitation

Answer: B

9. What is your diagnosis based on the following Doppler flow of abdominal aorta?



A. Severe AS
B. Coarctation of Aorta
C. Severe AI
D. Abdominal aorta aneurysm

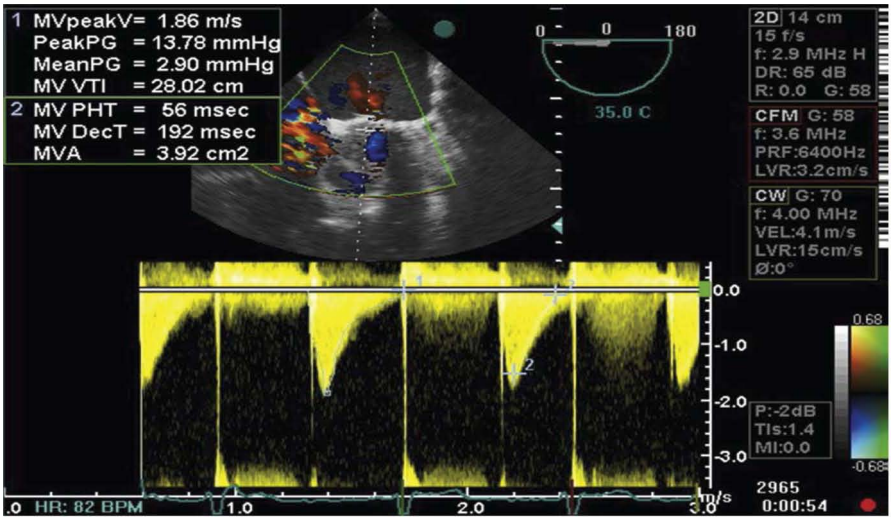
Answer: C

10. Which of the following is the most common mobile tricuspid valve mass?

a. Fibroelastoma
b. Sarcoma
c. Myxoma
d. Carcinoid syndrome

Answer: A

11. What is your diagnosis based on the following the Doppler flow pattern of prosthetic MV?



A. MV Malfunction
B. Significant Pannus ingrowth
C. Normal function
D. Severe para-valvular leakage

Answer: C

12. Which of the following statements about bicuspid aortic valve (BAV) is correct?

A. BAVs seldom have an acceptable function until late in life.
B. It carries an increased risk of aortic dissection (five to nine times higher risk).
C. Most of the patients have severe calcified AS after the age of 60.
D. 50% of patients need AV repair between the ages of 10 to 40 due to significant AI.

Answer: B

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1. Majid Maleki, Azin Alizadehasl, Majid Haghjoo. Practical Cardiology, First Edition; 2017.
2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.

CHAPTER 25

INFECTIVE ENDOCARDITIS

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1. Antibiotic prophylaxis should be considered for all following patients except for:

A. Trans-catheter implanted valves,
B. Any surgically implanted prosthetic valves
C. Any type of CHD repaired with a prosthetic material, lifelong.
D. Patients with a previous episode of IE.

Answer: C

Table 25.1 Cardiac Conditions at Highest Risk of Infective Endocarditis[3]

	Class of Recommendation	Level of Evidence
Antibiotic prophylaxis should be considered for patients at highest risk for IE: 1. Patients with any prosthetic valve, including a transcatheter valve, or those in whom any prosthetic material was used for cardiac valve repair 2. Patients with a previous episode of IE 3. Patients with CHD: a. Any type of cyanotic CHD b. Any type of CHD repaired with a prosthetic material, whether placed surgically or by percutaneous techniques, up to 6 months after the procedure or lifelong if residual shunt or valvular regurgitation remains	IIa	C
Antibiotic prophylaxis is not recommended in other forms of valvular or CHD.	III	C

2. Overall, the incidence of IE has remained stable, but the incidence of IE caused by Staphylococcus aureus has:

A. increased
B. remained unchanged
C. decreased

Answer: A

3. The most common microorganism for culture-negative endocarditis in a patient with pneumonia is:

A. Staphylococcus aureus
B. Aspergillus fumigatus
C. Enterococcus spp.
D. Streptococcus pneumonia

Answer: D

Table 25.2 Epidemiological and Etiological Clues in Culture Negative Endocarditis[4]

Epidemiological	Common Microorganism
IDU	Staphylococcus aureus, including community-acquired oxacillin-resistant strains Coagulase-negative staphylococci β-Hemolytic streptococci Fungi Aerobic gram-negative bacilli, including Pseudomonas aeruginosa Polymicrobial
Indwelling cardiovascular medical devices	S. aureus Coagulase-negative staphylococci Fungi Aerobic gram-negative bacilli Corynebacterium spp.
Genitourinary disorders, infections, and manipulation, including pregnancy, delivery, and abortion	Enterococcus spp. Group B streptococci (Streptococcus agalactiae) Listeria monocytogenes Aerobic gram-negative bacilli Neisseria gonorrhoeae
Chronic skin disorders, including recurrent infections	S. aureus β-Hemolytic streptococci
Poor dental health, dental procedures	VGS Nutritionally variant streptococci Abiotrophia defectiva Granulicatella spp. Gemella spp. HACEK organisms

Alcoholism, cirrhosis	Bartonella spp. Aeromonas spp. Listeria spp. Streptococcus pneumoniae β-Hemolytic streptococci
Burn	S. aureus Aerobic gram-negative bacilli, including P. aeruginosa Fungi
Diabetes mellitus	S. aureus β-Hemolytic streptococci S. pneumoniae
Early (≤ 1 y) prosthetic valve placement	Coagulase-negative staphylococci S. aureus Aerobic gram-negative bacilli Fungi Corynebacterium spp. Legionella spp.
Late (≥ 1 y) prosthetic valve placement	Coagulase-negative staphylococci S. aureus VGS Enterococcus spp. Fungi Corynebacterium spp.

Table 25.2 Epidemiological and Etiological Clues in Culture Negative Endocarditis[5]

Epidemiological	Common Microorganism
Dog or cat exposure	Bartonella spp. Pasteurella spp. Capnocytophaga spp.
Contact with contaminated milk or infected farm animals	Brucella spp. Coxiella burnetii Erysipelothrix spp.
Homeless, body lice	Brucella spp.
AIDS	Salmonella spp. S. aureus S. pneumoniae
Pneumonia, meningitis	S. pneumoniae
Solid organ transplantation	S. aureus Aspergillus fumigatus Enterococcus spp. Candida spp.
Gastrointestinal lesions	Streptococcus gallolyticus (bovis) Enterococcus spp. Clostridium septicum

4. Which sign or symptom is not common in subacute endocarditis?

A. Cerebrovascular accident
B. Osler's node
C. Congestive heart failure
D. Petechiae

Answer: C

5. Which test is seen in infective endocarditis with the lowest probability?

a. Leukocytosis
b. Elevated ESR
c. High C-reactive Protein
d. Anemia

Answer: A

6. Which phrase is incorrect about Duke criteria?

A. Abscess formation in TEE is a major criterion.
B. Increased grade of MR (mitral regurgitation) murmur is a major criterion.
C. The presence of 5 minor criteria definitely indicates the diagnosis of IE.
D. Glomerulonephritis is a minor criterion.

Answer: B

Regarding Duke Criteria, a definite diagnosis of infective endocarditis can be made in the presence of:

- two major criteria;
- one major and three minor criteria;
- five minor criteria.

A possible diagnosis of infective endocarditis can be made in the presence of:

- one major and one minor criteria
- three minor criteria.

Table 25.3 Modified Duke Criteria for the Diagnosis of Infectious Endocarditis[5]

MAJOR CRITERIA
Blood culture positive for IE • Typical microorganisms consistent with IE from two separate blood cultures: • Viridans streptococcus, Streptococcus bovis, HACEK group, Staphylococcus aureus, or community-acquired enterococci in the absence of primary focus • Microorganisms consistent with IE from two persistently positive blood cultures: o At least two positive blood culture of blood samples drawn >12 hours apart or all of three or a majority of four or more separate cultures of blood with first and last samples drawn at least 1 hour apart o Single positive blood culture for Coxiella burnetii or phase I lgG antibody titer>1:800. Evidence of endocardial involvement • Echocardiogram positive for IE (vegetation, abscess, new partial dehiscence of prosthetic valve) • New valvular regurgitation
MINOR CRITERIA
• Predisposition: predisposing heart condition, IDU • Fever temperature > 38°C • Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysms • Intracranial hemorrhages, conjunctival hemorrhages, Janeway lesions • Immunologic phenomena: glomerulonephritis Osler node, Roth spot, rheumatoid factor • Microbiological evidence: positive blood culture but does meet a major criterion or serologic evidence of active infection with organism consistent with IE

7. Which phrase is incorrect?

Table 25.4 Diagnosis of Infective Endocarditis[6]

Diagnosis is Definite in the Presence of	**Diagnosis is Possible in the Presence of**
Two major criteria or One major and three minor criteria or Five minor criteria	One major and one minor criteria or Three minor criteria

A. Early PVE (prosthetic valve endocarditis) pathogens are Staphylococcus aureus and Pseudomonas aeruginosa.
B. Staphylococcus aureus and Pseudomonas aeruginosa are typically highly virulent and invasive.
C. Late PVE more closely resembles ABE (acute bacterial endocarditis) than early PVE.
D. The most common etiologic agents for late PVE are coagulase-negative staphylococci and viridans streptococci.

Answer: C

8. In patients with major ischemic stroke or intracranial hemorrhage, it is reasonable
to delay valve surgery for at least:

A. 1 week
B. 2 weeks
C. 3 weeks
D. 4 weeks

Answer: D

9. The following factors are associated with an increased rate of relapse, except for:

A. Inadequate antibiotic treatment
B. Resistance to conventional antibiotic regimens
C. Empirical antimicrobial therapy for blood culture–negative infective endocarditis
D. Persistence of fever at the fourth postoperative day

Answer: D

Table 25.5 Factors associated with an Increased Rate of Relapse[3]

• Inadequate antibiotic treatment (agent, dose, duration)
• Resistant microorganism: Brucella spp., Legionella spp., Chlamydia spp., Mycoplasma spp., Mycobacterium spp., Bartonella spp., Coxiella burnetii, fungi
• Polymicrobial infection in an IVDA
• Empirical antimicrobial therapy for BCNIE
• Periannular extension
• Prosthetic valve IE
• Persistent metastatic foci of infection (abscesses)
• Resistance to conventional antibiotic regimens
• Positive valve culture
• Persistence of fever at the seventh postoperative day
• Chronic dialysis

10. Consider the following photos, showing tender nodules (left-hand image) and hemorrhagic macules and papules (right-hand image).

Which sentence is correct?

A. Right: Osler nodes; left: Janeway lesions.
B. Right: splinter hemorrhage; left: Janeway lesions
C. Right: Osler nodes; left: splinter hemorrhage
D. Right: Janeway lesions; left: Osler nodes

Answer: D

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CHAPTER 26

PERICARDIAL DISEASE

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1. Which sentence about the functions of the pericardium is incorrect?

A. It has a role in the fixation of the heart and preventing excessive heart motion.
B. It is a mechanical barrier against malignancy.
C. It has a lubricating function between the heart layers and surrounding structures.
D. It contains mechanoreceptors and chemoreceptors that have an autocrine-paracrine role and secrete prostaglandin and prostacyclin.

Answer: B

The pericardium has a significant role as a mechanical barrier against infection, not malignancy.

2. The following drugs can induce inflammatory involvement of pericardium except for:

A. Procainamide
B. Hydralazine
C. Isoniazid
D. Herceptin

Answer: D

The most common drugs in this matter are: Procainamide, Hydralazine, Isoniazid, Cyclosporine

3. ECG changes have four phases. .... of all patients show all of these four stages:

A. Half
B. A third
C. A quarter
D. A fifth

Answer: A

In acute pericarditis, ECG has been suggested as the most important test, with a typical finding of diffuse ST-segment elevation. ECG changes have four phases, but only half of patients show all of the following four stages:

First stage: There is a diffuse ST-segment elevation in most leads except aVR and V1 and it can be associated with PR depression. It occurs within a few hours of the onset of chest pain and lasts hours to days.

Second stage: The ST segments return to an isoelectric line.

Third stage: T-wave inversion occurs. These ECG changes can last weeks to months or even indeterminately in tuberculosis and uremia.

Fourth stage: The ECG may show normalization. Electrocardiographic findings can be mistaken for other diseases such as MI and early repolarization. History and physical examination help to distinguish these.

4. In a patient with pericarditis, in which of the following situations can he or she be managed as outpatient?

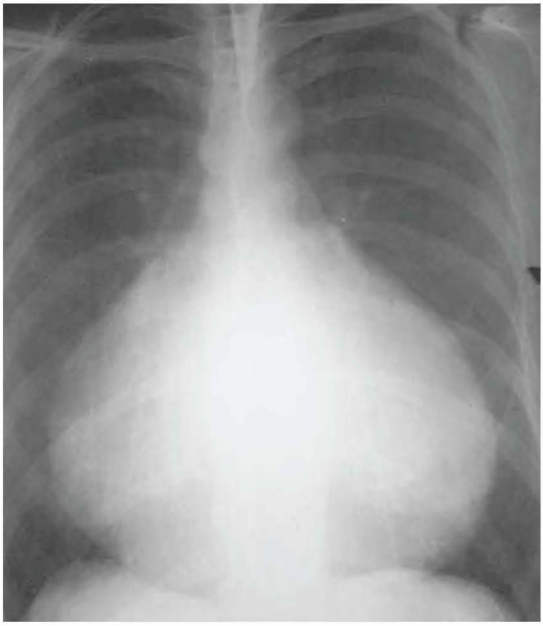
A. Idiopathic causes
B. Temperature >38°C
C. Immunosuppression
D. Myopericarditis

Answer: A

Admission should be considered in the following situations:

i. Moderate- to large-sized effusion
ii. Not responding to the initial treatment
iii. Non-idiopathic causes
iv. Elevated temperature (temperature >38°C)
v. Immunosuppression
vi. Anticoagulation
vii. Myopericarditis
viii. Trauma

5. What is your diagnosis based on the following CXR?



A. Congenitally corrected transposition of great arteries
B. Cardiomegaly in heart failure
C. Significant pericardial effusion
D. Tetralogy of Fallot (ToF)

Answer: C

The diffusely enlarged heart with obscured hilar shadows and no congestion in the lung parenchyma helps to differentiate the condition from cardiomegaly in heart failure; indeed, moderate or more PE on chest radiography may be detected by flask-like cardiomegaly.

6. Which of the following sentences is not correct regarding cardiac catheterization in constrictive pericarditis?

A. Coronary angiography should be done in patients with chest pain who are candidates for pericardiectomy.
B. You would see elevated and equalization of RA, RV, and LV diastolic pressures and pulmonary capillary wedge pressure about 20 mmHg with less than 3 to 5 mmHg differences between them.
C. You would see is elevated pulmonary artery pressure and RV systolic pressure, usually around 55-60 mmHg.
D. You would see a "dip and plateau" or "square root" sign in a ventricular pressure study.

Answer: C

There is a modestly elevated pulmonary artery pressure and RV systolic pressure, usually around 35 to 45 mmHg.

7. Which of the following statements regarding pericardial involvement in thyroid disease is correct?

A. About 50% of patients with severe hypothyroidism have PE.
B. Tamponade is a frequent complication.
C. Treatment of hypothyroidism results in effusion resolution.
D. Both, hyperthyroidism and hypothyroidism are common causes of pericardial effusion.

Answer: C

About 25% to 35% of patients with severe hypothyroidism have pericardial effusion.

8. Which statement about management in CP is incorrect?

A. Tachycardia is a compensatory mechanism, so beta blockers and calcium channel blockers should not be used.
B. High CRP and ESR are useful to identify patients who may respond to anti-inflammatory medication in transient constriction.
C. Digoxin is useful in patients with rapid ventricular response atrial fibrillation and should be given to maintain the heart rate to about 60 beats/min.
D. Hemodynamics and symptoms improve immediately after surgery.

Answer: C
Digoxin is useful in patients with rapid ventricular response atrial fibrillation to maintain the heart rate to about 80 to 90 beats/min.

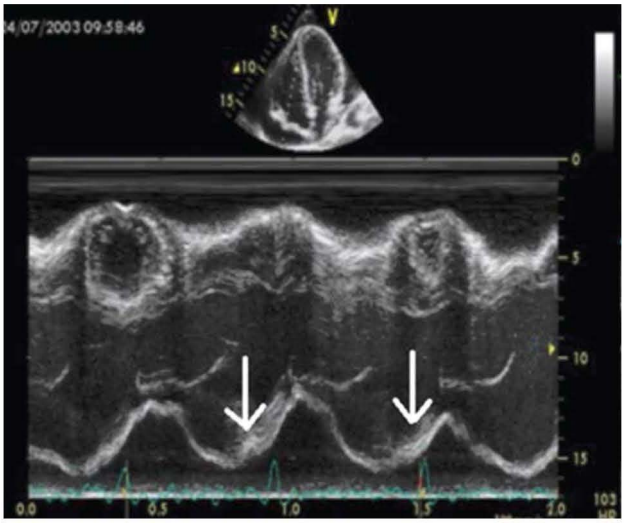
9. The definition of effusive-constrictive pericarditis (ECP) is a failure of the RA pressure to decrease by at least ... %, or to a level of less than ... mmHg after pericardiocentesis and no residual PE.

A. 30, 15
B. 50, 10
C. 30, 10
D. 50, 15

Answer: B

ECP patients are characterized by the failure of their hemodynamic levels to return to normal after pericardiocentesis. Normal is defined as an RA pressure ≥50% or <10 mmHg, with no residual PE.

10. The arrows indicate:



A. Large pericardial effusion with right **atrial** late **diastolic** and early systolic collapse
B. Large pericardial effusion with right **ventricular** late **diastolic** and early systolic collapse
C. Large pericardial effusion with right **atrial** late **systolic** and early diastolic collapse
D. Large pericardial effusion with right **ventricular** late **systolic** and early diastolic collapse

Answer: A

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CHAPTER 27

CONGENITAL HEART DISEASE

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1. An 18-year-old male, with a history of Fontan surgery 12 years ago, is referred to an adult congenital heart disease clinic with severe ascites and shortness of breath. Which of following problems could justify this condition?

A. Inferior vena cava (IVC) conduit stenosis
B. Protein-losing enteropathy (PLE)
C. Systemic ventricle failure
D. A and B

Answer: D

Ascites is a sign of elevated pressure in the IVC and hepatic veins. One of the causes of elevated Fontan circuit pressure is IVC conduit stenosis due to thrombosis or technical surgical problems. Another condition that results in ascites in Fontan patients is protein-losing enteropathy (PLE). PLE is one of the more ominous complications of Fontan patients, with poor response to treatment.

2. A 36-year-old male with a history of atriopulmonary connection (classic Fontan surgery) 26 years ago is referred for frequent episodes of atrial arrhythmia. What is the best clinical approach?

A. Cryoablation
B. Anti-arrhythmic drug therapy
C. Fontan conversion
D. AVN ablation and epicardial pace implantation

Answer: C

One of the common problems in classic Fontan is frequent arrhythmia due to the huge RA.

Ablation and drug therapy are inefficient treatments, but one of the more helpful approaches is Fontan conversion to an intra- or extra-cardiac conduit, so that the RA excluded from circulation, thus helping to control the arrhythmia.

3. A 25-year-old female with a history of tetralogy of Fallot (TOF) repair at the age of two is referred for a follow-up. Which murmur is more commonly heard in clinical examination?

A. Low pitch early diastolic murmur
B. Diastolic rumble in the left sternal border (LLSB)
C. HSM in apex
D. Loud P2

Answer: A

One of the most common consequences in a patient with a history of TOF repair is pulmonary regurgitation; PR in these patients is of low intensity and terminates in the early diastole because of an equalized RVEDP and PADP. Another murmur is SM due to residual VSD. Sometimes SM can be heard due to residual PS. A harsh diastolic murmur is most probably due to aortic regurgitation, which can be seen in patients with TFTC.

4. In a patient with an unrepaired tetralogy of Fallot, which of the following is correct in clinical examination?

A. The degree of cyanosis depends on the size of the VSD.
B. SM in the LSB is decreased if the RVOT obstruction severity increases.
C. In cyanotic spells, systolic thrill intensity is increased.
D. Diastolic murmur in LSB is heard more in patients with an unrepaired TOF.

Answer: B

Cyanosis in TOF depends on the degree of obstruction to the RVOT and increases with RVOT obstruction. The SM of PS depends inversely on RVOT obstruction, because an increase in the degree of stenosis means that more flow crosses from the VSD and the flow through the RVOT is decreased, so in TOF spells, the SM of subvalvular PS is also decreased. In native TOF, DM is not heard except in patients with absent pulmonary valve syndrome; both systolic and diastolic (TO and FRO) murmurs are heard in LSB.

5. Under which of the following circumstances would a patient with TGA and a history of the Rastelli operation 10 years ago be a candidate for redo surgery?

A. Residual VSD without evidence of LV volume overload
B. Severe ischemia in anterior circulation
C. LVOT stenosis shows a gradient of more than 50 mmHg
D. Supra valvular PS shows a gradient of more than 50 mmHg

Answer: C

Sequels of the Rastelli operation in a TGA patient include valve conduit stenosis and degeneration of the valve in conduit. Another sequent and indication for surgery is a residual VSD with a left-to-right shunt ratio of more than 1.5. A further sequel is LVOT obstruction, which is seen especially in patients with a greater distance between the VSD and aorta before surgery, so a larger patch is required for the deviation of the aorta to the LV. In this situation, a gradient of more than 50 mmHg in the LVOT is an indication for redo surgery.

6. What would you recommend for a young woman with history of Senning procedure in childhood who wants to become pregnant?

A. CMR before pregnancy for the evaluation of systemic ventricle function
B. Nothing: pregnancy has no effect on ventricular function
C. Antiarrhythmic treatment during pregnancy
D. HF drug treatment during pregnancy

Answer: A

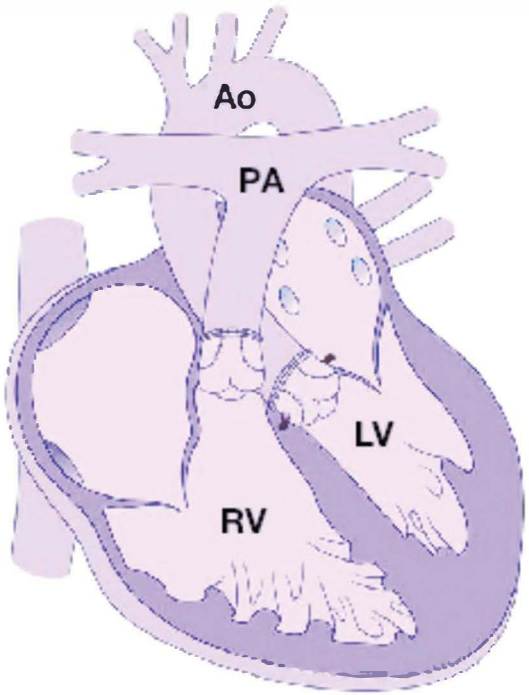
One of the most common complications in Senning procedure is systemic right ventricle dysfunction, but because we cannot precisely evaluate RV function by echo, a CMR is recommended for the precise evaluation of RV function. Before pregnancy, it is important to obtain a good estimation of RV function and to quantify the risk of pregnancy. In patients with an RVEF of less than 45%, the patient should be discouraged against pregnancy. RV function may get worse during pregnancy and this malfunction may persist after delivery.

7. A 16-year-old boy with complete TGA who underwent an arterial switch when 3 months old is referred to echo clinic. What kind of evaluation should be considered in his TTE examination?

A. Exact evaluation for RWMA
B. Evaluation for AI and aortic root dilation
C. Evaluation for supravalvular pulmonary stenosis
D. All of the above

Answer: D

The arterial switch procedure or Janten operation is one of the best surgery choices for TGA patients. During this procedure, the ascending aorta above the coronary arteries is transacted and brought to the posterior; the pulmonary artery is translocated to the anterior and anastomosed to the native aorta and aortic valve; and the coronary arteries are reimplanted on the neo-ascending aorta. The complications of this procedure are proximal coronary artery stenosis, dilation of the neo-aorta and AI, and supra valve pulmonary anastomosis stenosis. Therefore, any echo exam should search for these complications.



8. In a patient with a history of Fontan surgery, if saturation is less than 90% on room air, what can we do to eliminate cyanosis?

A. Fenestration closure
B. Find and close the pulmonary AV fistula
C. Medical treatment for systemic ventricle failure
D. A and B

Answer: D

Cyanosis in Fontan patients has several causes. One of the most common is fenestration between the conduit and the RA which is created by the surgeon. If the patient's condition allows, closure by device is performed to eliminate the cyanosis. Another reason is pulmonary AV fistula and venovenous collaterals that should be sought using CMR or CT

angiography. In such cases, an interventional procedure should be performed to eliminate this pathological condition.

9. In a 3-month-old neonate who was born with a cyanotic heart disorder, the TTE shows both great arteries arising from the RV, no pulmonary obstruction, and a large subpulmonic VSD with bidirectional flow. The aorta is a little anterior to the PA. What kind of surgery should be done?

A. Arterial switch and VSD closure
B. Rastelli procedure
C. Nikaidoh procedure
D. Atrial switch and VSD closure

Answer: A

In this neonate with these echo findings, the diagnosis is a double-outlet right ventricule (DORV) with a Taussig-bing anomaly. In this anomaly, the aorta is a little further anterior to the PA and both arise from the RV, while the VSD is in a subpulmonic position. The preferred surgical approach is an arterial switch and the VSD should be closed.

10. In a 12-year-old boy who complains about infrequent headaches, his physical examination detects high blood pressure in the left arm, but very weak pulses in the right arm and both legs. What would you expect to see in his CXR?

A. Rib notching in the right side
B. Reverse 3 (three) sign in the left border
C. Rib notching in the left side
D. Cardiomegaly

Answer: C

Based on his history and physical examination, this patient has a coarctation of the aorta. The weak right arm pulse suggests he has an aberrant right subclavian artery. In this case, the right subclavian artery has low pressure and the right intercostal artery is not dilated, so rib notching would only be seen in the left side. If both subclavian artery branches are proximal to the coarctation site, rib notching is seen bilaterally. In coarctation, because of pre- and post-stenotic dilation, we can see "3" signs in the left border and "reverse 3" signs are seen in barium esophagography.

11. As a cardiologist in a cardiology-obstetric team, which pregnant patient with a history of congenital heart disease would you allow to continue with her pregnancy under tight supervision?

A. A 25-year-old with a history of Senning procedure and an RVEF of 30%
B. A 32-year-old with a history of Fontan procedure and a systemic EF of 40%
C. A 35-year-old case of BAV with AVA <1 cm^2
D. A 26-year-old case of Marfan syndrome and dilated aortic root (46 mm)

Answer: B

In patients with Eisenmenger syndrome, severe PAH (more than 70% of systemic pressure), severe MS, severe AS, severe systemic ventricle dysfunction (EF<30%), and Marfan syndrome with an aortic root >45 mm, pregnancy is contraindicated. In patients with a history of Fontan, if systemic EF is acceptable, pregnancy under tight supervision could be continued.

12. A 35-year-old male is referred to a cardiology clinic with a history of palpitations. In physical exam, wide fixed S2 splitting and SM in apex are heard, while in ECG findings, rSr' is seen in V1 and LAD. Which condition is less probable?

A. Large atrial septal defect
B. Cleft of the MV and severe MR
C. Moderate-sized VSD
D. Abnormal chorda in the LVOT

Answer: C

Physical examination in this patient showed the existence of an ASD associated with MR, and the ECG findings suggest a partial form of AVSD. Therefore, in the echo findings, a VSD of moderate size is less probable because of no thrill was found in the examination and there was also no evidence of significant PAH. Another finding in patients with partial AVSDs is a cleft on the AMVL and abnormal chorda in the LVOT, along with an elongated LVOT and goose-neck deformity.

13. Which of the following is not an indication for ventricular septal defect (VSD) closure?

A. Recurrent endocarditis
B. Muscular VSD with more than mild aortic regurgitation
C. Left ventricular enlargement
D. Left atrial enlargement
E. Reversible pulmonary hypertension

Answer: B

VSDs that cause chamber enlargement, pulmonary hypertension, ventricular dysfunction or recurrent endocarditis warrant closure. In perimembranous or outlet VSDs, anything more than mild aortic regurgitation favors intervention.

14. Which is not an indication for Ebstein anomaly intervention?

A. Limited functional capacity
B. Systemic arterial desaturation
C. Paradoxical embolism
D. Severe tricuspid regurgitation
E. Supraventricular tachycardia resistant to therapy

Answer: D

Indications for intervention in Ebstein anomaly are: right-sided heart failure, cyanosis, impaired functional capacity, and paradoxical embolism. Relative indications are a cardiothoracic ratio > 60% and drug-refractory supraventricular arrhythmias.

15. An adult patient with a large VSD is referred for hemodynamic evaluation. The following data have been obtained during cardiac catheterization. What is the pulmonary vascular resistance based on Wood units in this patient?

Aortic pressure = 120/80; pulmonary artery = 80/50; left atrial pressure = 12mmHg; right atrial pressure = 10mmHg; patient body surface area = 1.6 m2; hemoglobin = 17mg/dl

O_2 saturations: superior vena cava (SVC) = 65%; inferior vena cava (IVC) = 71%; right atrium = 70%; right ventricle = 75%; pulmonary artery = 75%; aorta = 92%

A. 1.27
B. 3.76
C. 12.7
D. 200
E. 1.18

Answer: C

Cardiac catheterization is used to assess pulmonary (PVR) and systemic vascular resistances (SVR) for the determination of operability and treatment options of patients with CHD.

$$PVR = \frac{meanPAP - meanLAP}{Qp}$$

Qp or pulmonary blood flow (PBF) in liters per minute (l/min) is calculated using the following formula:

$$PBF = \frac{\text{Oxygen consumption (mL/min)}}{(\text{Pulmonary venous O2 sat} - \text{Pulmonary arterial O2sat}) \times 1.36 \times \text{Hgb} \times 10}$$

Oxygen consumption is assumed to be 125 ml/m^2 in many laboratories. Hgb is the hemoglobin concentration (mg/dl). Multiplication by 10 is to convert deciliters to liters, when the hemoglobin is measured as g/dl. When

pulmonary venous saturation is not available, systemic arterial oxygen saturation could be substituted. If systemic arterial saturation is less than 93% due to a right-to-left shunt, pulmonary venous oxygen saturation is calculated as 98%. Thus, in the mentioned patient:

PBF: (125 x 1.6) ÷ [(0.98-0.75) x 1.36 x 17 x 10] = 3.76 l/min

PVR: 60 – 12 ÷ 3.76 = 12.7wood/unit

16. Pulmonary arteriovenous fistulas are complications of which palliative shunt?

A. Central shunt
B. Classic Blalock-Taussig-Thomas (BT) shunt
C. Modified Blalock-Taussig-Thomas shunt
D. Glenn shunt
E. Waterston shunt

Answer: D

Palliative systemic-to-pulmonary (PA) shunts can be classified into arterial and venous types.

Arteria shunts:

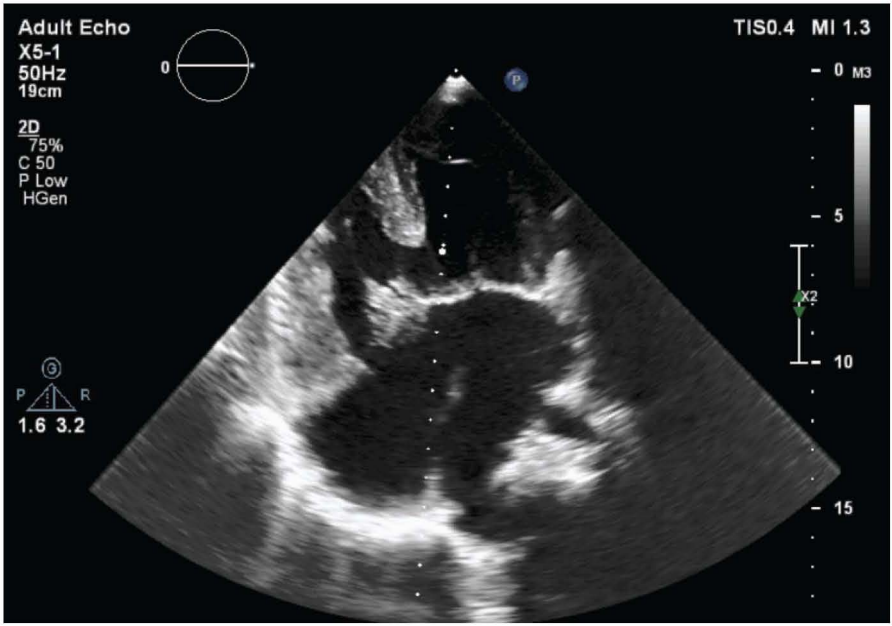
i. Blalock-Taussig-Thomas shunt (subclavian artery to PA); can be classic with end-to-side subclavian artery to PA connection and no ipsilateral arm pulse, or modified with use of surgical grafts and side-to-side anastomosis.
ii. Central shunt (side-to-side aorta to PA by surgical graft)
iii. Potts shunt (descending aorta to LPA)
iv. Waterston shunt (ascending aorta to RPA)

Venous shunt:

Glenn shunt (SVC to PA)

Glenn shunts can increase the pulmonary flow without causing volume overload on the systemic ventricle. However, they can lead to the gradual development of pulmonary arteriovenous fistulas.

17. Which of the following cardiac anomalies are depicted in the image?

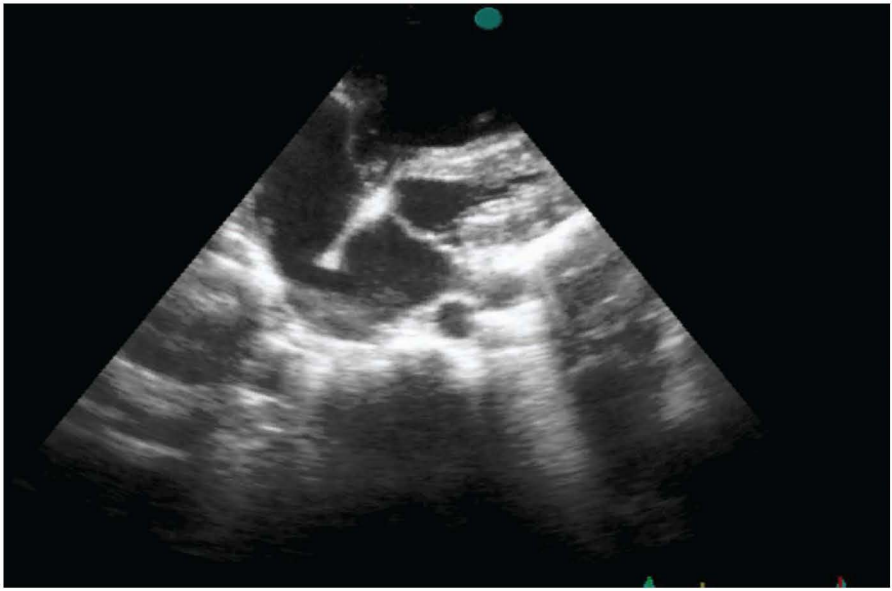


A. Primum ASD
B. Inlet VSD
C. Common atrioventricular valve
D. Complete atrioventricular septal defect (AVSD)
E. All of the above

Answer: E

The image depicts a patient with complete atrioventricular canal defect. The common findings are a common AV junction and absent atrioventricular septum, with the AV valves being at the same level resulting in a primum-type ASD, elongated left ventricular outflow tract, AV valve anomalies with the presence of a cleft, and abnormal lateral posteromedial papillary muscle. An inlet VSD can be large, small, or absent.

18. What is the shown cardiac anomaly?



A. SVC-type sinus venosus atrial septal defect (ASD)
B. IVC-type sinus venosus ASD
C. Muscular VSD
D. AVSD
E. High secundum ASD

Answer: A

There are four types of atrial abnormal communication: (i) ostium primum, (ii) ostium secundum, (iii) sinus venosus, and (iv) coronary sinus defects. Superior vena cava-type sinus venosus defects result from a deficiency between the superior vena cava and, usually, the right upper and middle pulmonary veins. In echocardiography, no rim is seen between the atrial roof and the defect.

19. Which abnormality is uncommon in cyanotic patients?

A. Cerebral emboli in iron-deficient patients
B. Cerebral hemorrhage
C. Brain abscess
D. Gout
E. Atherosclerotic narrowing of the coronary arteries

Answer: E

Cyanosis, or arterial oxygen desaturation, results from the shunting or mixing of systemic venous blood into the arterial blood. Hypoxemia leads to increased production of erythropoietin by the kidneys, which stimulates the production of red blood cells by the bone marrow. Secondary erythrocytosis is therefore seen in all cyanotic patients and could present with hyperviscosity symptoms including dizziness and fatigue. Other abnormalities found in cyanotic patients are hemostatic abnormalities with both thrombotic and bleeding tendencies, neurologic abnormalities (hemorrhage, emboli, brain abscess), and renal and rheumatologic disorders. Patients with central cyanosis often have dilated coronary arteries, while atherosclerotic narrowing is not usual.

20. What physical examination finding is not expected in a patient with an atrial septal defect (ASD)?

A. The a wave is greater than the v wave in jugular venous pressure
B. Mid-diastolic rumble
C. Wide and fixed splitting of S2
D. Systolic ejection murmur
E. Hyperdynamic right ventricular impulse

Answer: A

In ASD, there is a left atrial pattern of jugular venous pressure in which the a wave is equal to the v wave. A wide fixed splitting of S2 is a common clinical finding. A hyperdynamic right ventricular (RV) impulse might be felt at expiration in substernal or left sternal areas and a dilated pulmonary trunk palpated at the second left intercostal space. There could be a mid-diastolic rumble due to increased flow through the tricuspid valve.

21. Which ECG finding is not expected in ASDs:

A. Crochetage(notch) on R waves in inferior leads
B. Atrial flutter
C. Tall R wave in V1
D. Clockwise rotation of the QRS axis in precordial leads
E. Negative P wave in inferior leads

Answer: D

Crochetage or notching in R waves inferior leads is seen in ASDs. Atrial arrhythmias are common in ASDs. With pulmonary hypertension there is a tall R/R' in V1. Right bundle branch block can also be seen. In sinus venosus ASDs, there may be a low atrial rhythm with negative P waves in inferior leads.

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CHAPTER 28

VENOUS THROMBOEMBOLISM

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1. A 35-year-old woman who has used oral contraceptive pills for two years presents with acute-onset dyspnea, chest pain and hemoptysis. She is febrile, her vital signs are BP = 120/65 mmHg, PR = 95 beats/min, T = 39.0 °C, and RR = 20/min. What is your next diagnostic step?

A. D-Dimer
B. Chest CT scan
C. Echocardiography
D. V/Q scan

Answer: A

This patient's Well's score for acute pulmonary embolism is just 1 (hemoptysis). She is considered low-risk for pulmonary embolism, so a D-dimer test is enough as a first step. If it is positive, further study with a more specific test such as a CT-angiography would be appropriate.

Table 28.1 Well's Score for Acute Pulmonary Emboli[1]

Criterion	Score
DVT symptoms or signs	3
An alternative diagnosis is less likely than PE	3
Heart rate >100 beats / min	1.5
Immobilization of surgery within 4 weeks	1.5
Previous DVT or PE	1.5
Hemoptysis	1
Cancer treated within 6 months or metastatic	1

*>4 score points = high probability; ≤4 score points = non-high probability.
DVT, Deep vein thrombosis; PE, pulmonary embolism.

2. A 75-year-old woman is hospitalized for surgery on a tibial fracture resulting from a car crash. Two days after the operation, she develops chest pain and dyspnea. Her blood pressure drops to 80/50 mmHg.
Which pathophysiology has a central role in her situation?

A. Decreased LV preload
B. Decreased L V O_2 supply
C. Increased L V wall tension
D. Increased LV afterload

Answer: A

This patient has most probably developed a massive pulmonary embolism. In this situation, an occlusion of more than 50% of the pulmonary vascular bed results in increased RV (not LV) wall tension and afterload, which in turn decreases the LV preload. A reduced LV O_2 supply is secondary to low blood pressure.

(Practical Cardiology Page 502)
(Fig. 29-3)

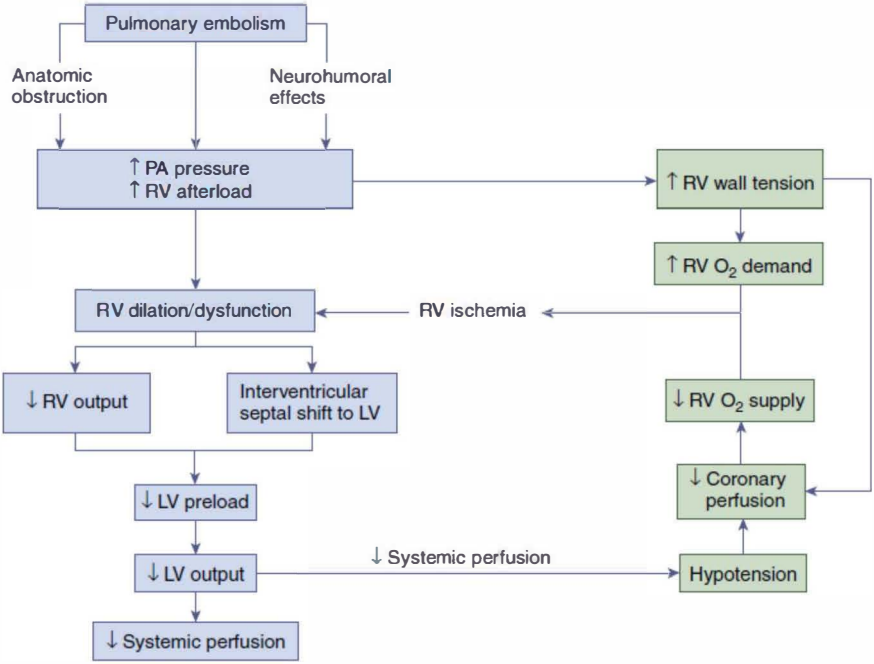

Figure 28.1 Pathophysiology of pulmonary emboli. LV, left ventricular; PA, pulmonary artery; RV, right ventricular[2]

3. A 36-year-old woman who is a known case of ovarian cancer is brought to hospital with dyspnea, palpitations and diaphoresis. Her BP = 80/30 mmHg and HR = 125/min. The hospital lacks imaging or operative facilities other than ECG, duplex and echocardiography. Her echo shows RV overload and dilation. What is the best decision?

A. Send the patient to the nearest hospital that can do a CT-Angiography.
B. Begin immediate thrombolysis together with heparin therapy.
C. Start heparin and send patient to an equipped hospital.
D. Conduct a lower extremity duplex + transesophageal echocardiography to confirm or rule out diagnosis.

Answer: B This patient is in cardiogenic shock and the most probable diagnosis is a pulmonary embolism. According to guidelines, when the patient is in shock and CT-Angiography is not immediately available, echocardiography is useful. If it shows RV overload, we can directly start thrombolysis. Other options are suitable for patients who are more stable/ not so critical.

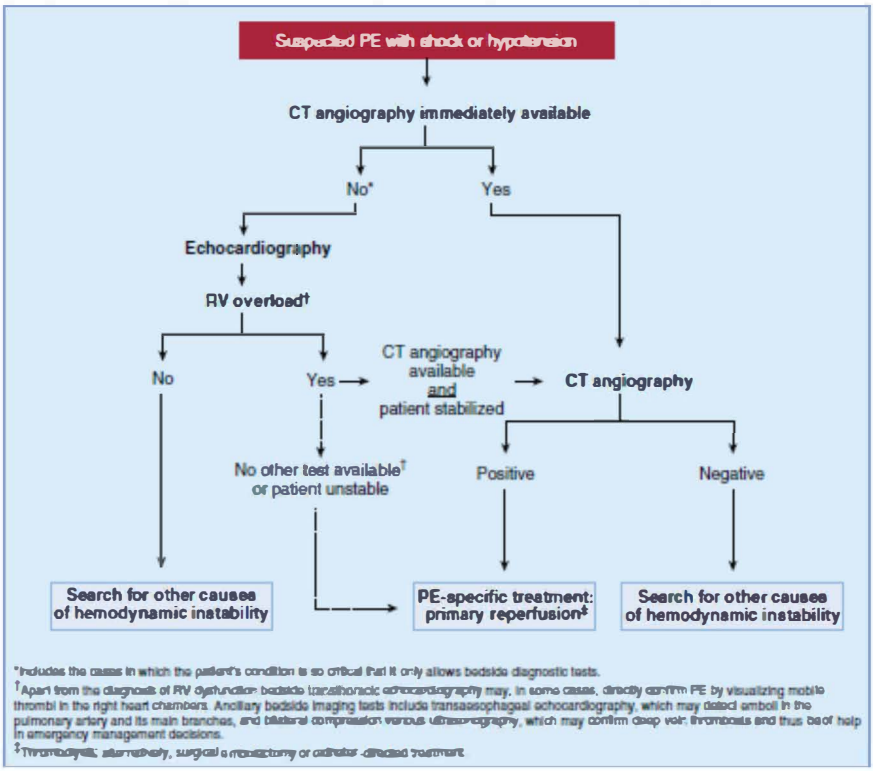

Figure 28.2 Approach to patients with high-risk pulmonary emboli (PE). Computed tomography; RV, right ventricular.[3]

4. Which sentence is not correct regarding treatment of venous thromboembolism (VTE)?

A. In a patient with calf deep vein thrombosis (DVT) after a tibial fracture, three months anticoagulation is enough.
B. If a patient is affected by recurrent VTE on rivaroxaban, switching to warfarin is recommended.
C. It is better not to send intermediate-risk pulmonary embolism (PE) patients for surgical embolectomy even if they have high bleeding risk for thrombolytic therapy.
D. All types of NOACS are contraindicated in patients with severe renal impairments (CrCl<30 ml)

Answer: B

The shortest duration of treatment by anticoagulants is in cases of provoked DVT of the distal leg (three months). According to ACCP guidelines, if a patient develops VTE on Warfarin or NOACs, it is better to switch to LMWH at least temporarily, making B the correct answer. Sending intermediate-risk PE patients for surgical embolectomy is a class IIb action and is best avoided. Using NOACS in patients with severe renal dysfunction is a class III action (contraindicated).

Table 28.2 Guidelines and Highlights on the treatment of Venous Thromboembolism (VTE)[1]

	Level of evidence
Anticoagulation therapy for PE and proximal DVT	ACCP: In patients with proximal DVT or PE, we recommend long-term (3 months) anticoagulant therapy over no such therapy (Grade 1B) ACCP: For patients with DVT of the leg or PE and no cancer who are not treated with dabigatran, rivaroxaban, apixaban, or edoxaban, we suggest VKA therapy over LMWH (Grade 2C) ACCP: In patients with DVT of the leg or PE and no cancer, as long-term (first 3 months) anticoagulant therapy, we suggest dabigatran, rivaroxaban, apixaban, or edoxaban over vitamin K antagonist (VKA) therapy (Grade 2B). ESC: In all patients with acute PE who are planned to be treated with warfarin, parenteral anticoagulation should be started immediately (Class I) ECS: In patient not candidate for systemic thrombolytic or invasive management (CDT or surgical Embolectomy), LMWH or Fondaparinux are preferred over UFH (Class I) ESC: For patients who are candidate for thrombolytic therapy or invasive management (CDT or surgical Embolectomy) intravenous UFH should be started without delay. (Class IC) ESC: Use of VKA and NOAC (Rivoraxaban, Apixaban, Dabigatran, Edoxaban) in in all patients with PE is recommended (Class I) ESC: Use of NOAC in patients with severe renal impairment is not recommended (Class III).
Anticoagulation for distal DVT	ACCP: In patients with an isolated distal DVT of the leg provoked by surgery or by a nonsurgical transient risk factor, we suggest treatment with anticoagulation for 3 months over treatment of a shorter period (Grade 2C)
Anticoagulation in patients with VTE and cancer	ACCP: In patients with DVT of the leg or PE and cancer ("cancer-associated thrombosis"), as long-term (first 3 months) anticoagulant therapy, we suggest LMWH over VKA therapy (Grade 2C), dabigatran (Grade 2C), rivaroxaban (Grade 2C), apixaban (Grade 2C), or edoxaban (Grade 2C).

	ACCP: In patients with DVT of the leg or PE and active cancer ("cancer-associated thrombosis") and who (i) do not have a high bleeding risk, we recommend extended anticoagulant therapy (no scheduled stop date) over 3 months of therapy (Grade 1B), or (ii) have a high bleeding risk, we suggest extended anticoagulant therapy (no scheduled stop date) over 3 months of therapy (Grade 2B).
Provoked vs. unprovoked VTE	ACCP: In patients with a provoked proximal DVT of the leg or PE, we recommend treatment with anticoagulation for 3 months over (i) treatment of a shorter period (Grade 1B), (ii) treatment of a longer time-limited period (eg, 6, 12, or 24 months) (Grade 1B), or (iii) extended therapy (no scheduled stop date) (Grade 1B). ACCP: In patients with an unprovoked DVT of the leg (isolated distal or proximal) or PE, we recommend treatment with anticoagulation for at least 3 months over treatment of a shorter duration (Grade 1B), and we recommend treatment with anticoagulation for 3 months over treatment of a longer time-limited period (eg, 6, 12, or 24 months) (Grade 1B). ACCP: In patients with a first VTE that is an unprovoked proximal DVT of the leg or PE and who have a (i) low or moderate bleeding risk (see text), we suggest extended anticoagulant therapy (no scheduled stop date) over 3 months of therapy (Grade 2B), and (ii) high bleeding risk (see text), we recommend 3 months of anticoagulant therapy over extended therapy (no scheduled stop date)(Grade 1B). ACCP: In patients with a first VTE that is an unprovoked proximal DVT of the leg or PE and who have a (i) low or moderate bleeding risk (see text), we suggest extended anticoagulant therapy (no scheduled stop date) over 3 months of therapy (Grade 2B), and (ii) high bleeding risk (see text), we recommend 3 months of anticoagulant therapy over extended therapy (no scheduled stop date)(Grade 1B) ACCP: In patients with a second unprovoked VTE and who have a (i) low bleeding risk (see text), we recommend extended anticoagulant therapy (no scheduled stop date) over 3 months (Grade 1B); (ii) moderate bleeding risk (see text), we suggest extended anticoagulant therapy over 3 months of therapy (Grade

	2B); or (iii) high bleeding risk (see text), we suggest 3 months of anticoagulant therapy over extended therapy (no scheduled stop date)(Grade 2B)
Systemic thrombolytic therapy	ACCP: In patients with acute PE associated with hypotension (eg, systolic BP <90 mm Hg) who do not have a high bleeding risk, we suggest systemically administered thrombolytic therapy over no such therapy (Grade 2B). ACCP: In most patients with acute PE not associated with hypotension, we recommend against systemically administered thrombolytic therapy (Grade 1B). ACCP: In patients with acute PE who are treated with a thrombolytic agent, we suggest systemic thrombolytic therapy using a peripheral vein over CDT (Grade 2C) ESC: In patients with acute PE and shock or hypotension, systemic thrombolytic therapy is recommended (Class IB). ESC: In patient with acute PE without hypotension and Shock, routine use of thrombolytic therapy is not recommended (Class III). ESC: Thrombolytic therapy should be considered for patient with intermediate-high-risk PE and clinical signs of hemodynamic decompensation (IIa).
Catheter directed Thrombolysis (CDT)	ACCP: In patients with acute proximal DVT of the leg, we suggest anticoagulant therapy alone over CDT (Grade 2C). ACCP: In patients with acute PE associated with hypotension and who have (i) a high bleeding risk, (ii) failed systemic thrombolysis, or (iii) shock that is likely to cause death before systemic thrombolysis can take effect (eg, within hours), if appropriate expertise and resources are available, we suggest catheter-assisted thrombus removal over no such intervention (Grade 2C). ESC: In patients with acute PE and shock or hypotension, CDT should be considered in patients whom systemic thrombolytic is contraindicated or failed (Class IIa). ESC: CDT may be considered in intermediate-high-risk patients if the anticipated risk of bleeding under thrombolytic therapy is high (Class IIb).
Surgical embolectomy for PE	ESC: In patients with acute PE and shock or hypotension, surgical embolectomy is recommended in

	patients whom systemic thrombolytic is contraindicated or failed (Class IC). ESC: Surgical embolectomy may be considered in intermediate-high-risk patients if the anticipated risk of bleeding under thrombolytic therapy is high (Class IIb).
Inferior Vena Cava Filter	ACCP: In patients with acute DVT or PE who are treated with anticoagulants, we recommend against the use of an inferior vena cava (IVC) filter (Grade 1B)
Outpatient management of VTE	ACCP: In patients with low-risk PE and whose home circumstances are adequate, we suggest treatment at home or early discharge over standard discharge (eg, after the first 5 days of treatment) (Grade 2B). ESC: Patients with acute low-risk PE should be considered for early discharge and continuation of treatment at home if proper outpatient care and anticoagulation treatment can be provided (Class IIa)
Recurrent VTE	ACCP: In patients who have recurrent VTE on VKA therapy (in the therapeutic range) or on dabigatran, rivaroxaban, apixaban, or edoxaban (and are believed to be compliant), we suggest switching to treatment with LMWH at least temporarily (Grade 2C) ACCP: In patients who have recurrent VTE on long-term LMWH (and are believed to be compliant), we suggest increasing the dose of LMWH by about one-quarter to one-third (Grade 2C).

5. Which anticoagulant drug has no effect on active factor X (Xa)?

A. Apixaban
B. Fondoparinex
C. LMWH
D. Dabigatran

Answer: D

Both Apixaban & Fondoparinex are factor Xa inhibitors. LMWH inhibits both factor IIa and factor Xa. Dabigatran only inhibits factor IIa and has no effect on factor Xa.

6. A 69-year-old man with liver cirrhosis and hepatic malignancy presents with dyspnea and tachycardia. His vital signs are: BP = 120/100 mmHg, PR = 98 beats/min, R = 15, T = 37.5°C. An echocardiography shows mild RV dilation and dysfunction. Which treatment is best for him?

A. Alteplase + heparin
B. Heparin
C. Apixaban
D. Dabigatran + heparin
E. Alteplase + LMWH
F. LMWH

Answer: F

There is no hemodynamic compromise and only mild RV dysfunction, so alteplase is not indicated. Liver failure also makes the patient susceptible to bleeding, so A and E are incorrect. In both patients with liver failure and malignancy, LMWH is the anticoagulant of choice.

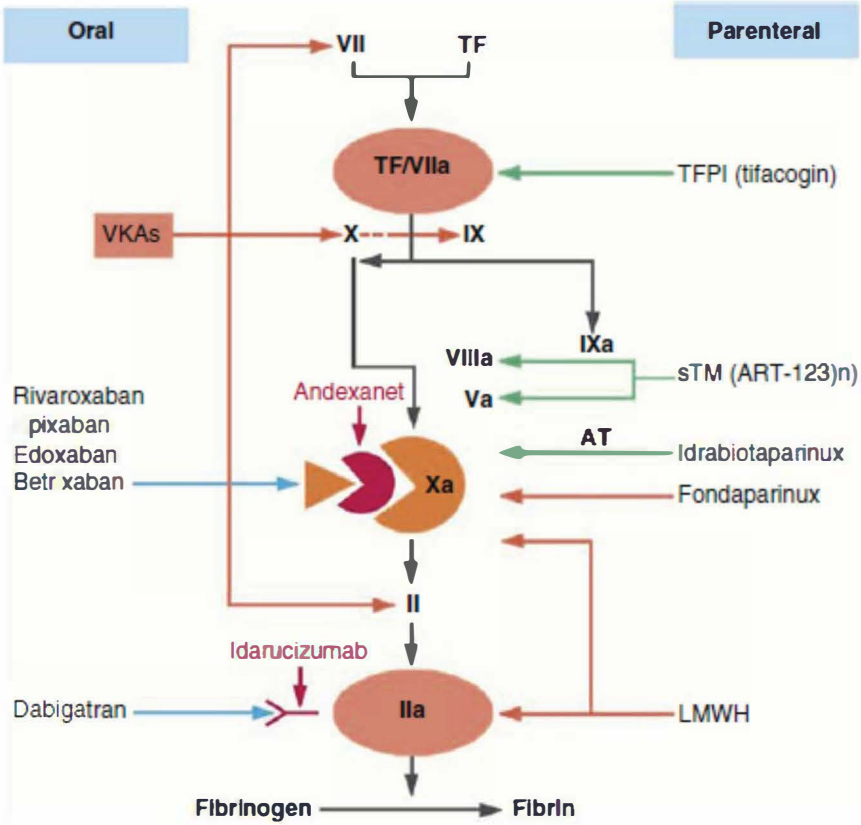

Figure 28.3 Targets of various oral anticoagulation therapies and their antidoes.[4]

7. A 35-year-old active man presents with massive thrombosis of the axillary vein extending to the superior vena cava. Which disorder would *not* be an underlying cause?

A. Protein S deficiency
B. Factor V Leiden
C. Anticardiolipin antibodies
D. Prothrombin gene mutation
E. Factor X deficiency
F. All of the above

Answer: E

Apart from factor X deficiency (hemophilia B or Christmas disease), which is defined by bleeding tendency, all the other choices are coagulation disorders. They make the patient prone to DVTs in unusual sites (as with this patient) as well as recurrent and severe DVTs.[1]

8. Which of the following patients has the greatest risk of recurrent venous thromboembolism events (VTE)?

A. A 65-year-old man with a history of cerebrovascular disease and leg paresis with further complications due to an episode of acute deep venous thrombosis (DVT).
B. A 32-year-old woman who has experienced an episode of pulmonary embolism (PE) during her last pregnancy.
C. A 45-year-old man who had an episode of acute DVT after a car accident.
D. A 55-year-old woman with a history of colon cancer complicated by an acute PE. Her recent colonoscopy after multiple courses of chemotherapy revealed no active cancer.

Answer: A

It should be noted that recurrence is more prevalent in men compared to women and also happens more frequently in patients who have had a spontaneous VTE episode (i.e. unprovoked). Some other risk factors might also have an impact on VTE recurrence; the most widely reported are active cancer, increasing BMI, and neurological diseases with leg paresis. Importantly, an index VTE following pregnancy, fracture, and surgery or trauma have no prognostic implication on VTE recurrence.[1]

9. Which of the following regimens would you <u>not</u> advise for patients with acute PE?

A. Enoxaparin 1mg/kg for a 34-year-old pregnant woman
B. Enoxaparin followed by rivaroxaban for a 32-year-old man
C. Enoxaparin followed by dabigatran for a 65-year-old woman
D. Apixaban for a 42-year-old man

Answer: B

Rivaroxaban and apixaban do not need pretreatment with parenteral anticoagulation. On the other hand, patients on Dabigatran and Edoxaban should be pretreated with parenteral anticoagulation.

10. A 32-year-old man is admitted to the emergency department with recent dyspnea. His initial vital signs are: BP = 120/85 mmHg; HR = 125 bpm. His pulmonary CT angiography reveals bilateral pulmonary emboli affecting both left and right pulmonary arteries. His echocardiography shows moderate right ventricular (RV) enlargement, mild RV dysfunction and a 15 mm thrombus-like mass in transit from the patent foramen oval (PFO). What would be your recommendation for the patient?

A. Surgical embolectomy
B. Anticoagulation only
C. Systemic thrombolysis
D. Catheter-directed thrombolysis

Answer: A

The patient is categorized as intermediate-to-high risk PE. However, the echocardiography exam revealed a clot in transit from the PFO, which is considered an indication for surgical intervention.

11. Which of the following patients would benefit from a hypercoagulable state screening test after an episode of VTE?

A. A 32-year-old man with a submassive PE and a familial history of pulmonary emboli in his father and two of his uncles
B. A 21-year-old woman with a single episode of unprovoked PE
C. A 45-year-old man with a history of renal vein thrombosis
D. A 61-year-old woman with history of warfarin-induced skin thrombosis

Answer: B

A single episode of unprovoked VTE is not an approved indication for a hypercoagulable state test.

12. A 29-year-old man presents at the emergency department with recent dyspnea. BP = 115/65 mmHg, HR = 116 bpm, and O_2 saturation = 89%. A PE has been confirmed CT angiography by. A mild RV dysfunction and a RV/LV diameter ratio of 1 has been reported by the echocardiography exam. Troponin T and ProBNP were reported at 1 ng/dl and 1000 pg/ml respectively. How would you categorize this patient?

A. Low risk
B. Intermediate-to-low risk
C. Intermediate-to-high risk
D. Massive

Answer: C

A patient with a sPESI score of >1 (heart rate >110 bpm, O_2 saturation <90%), RV dysfunction (RV/LV diameter ratio >0.9 and elevated Pro-BNP) and positive cardiac biomarkers (elevated troponin T) is considered an intermediate-to-high-risk patient (Page 507).

13. A 39-year-old man with sudden unilateral pain and swelling of the left leg has been referred to the emergency department from a local day clinic. He states that the edema started three days ago but that he did not pay attention to it. However, the pain has become aggravated since this morning and he cannot bear it anymore. On a physical exam, the leg has a distinct blue discoloration and there is a 5 cm difference between the two legs' circumference. In the day clinic, he underwent an ultrasound exam which revealed a proximal acute DVT from the left iliac vein to the popliteal vein. The arterial pulse is currently very weak. Which treatment strategy would you recommend?

A. Catheter Directed Thrombolysis
B. Surgical Thrombectomy
C. Intensive anticoagulation
D. Both CDT and surgical thrombectomy might be recommended based on the hospital facilities.

Answer: B

The only indication for surgical thrombectomy in acute DVT is when the arterial flow is compromised. The situation varies from phlegmasia alba dolens (which manifests as swollen and white extremities during the early

stage of compromised arterial flow) to the critical phlegmasia cerulea dolens (severe swelling, cyanosis and blue discoloration). Prompt surgical intervention is needed in both situations.[1]

14. Which of the following statements is not correct regarding the clinical risk factors of VTE?

A. Second-generation oral contraceptive (OCP) agents have a lower risk of VTE compared to first- and third-generation OCPs.
B. The third trimester of pregnancy carries the highest risk of VTE.
C. Varicose veins in non-pregnant woman do not increase the risk of acute DVT.
D. May-Thurner syndrome increases the risk of DVT.

Answers: B

Second generation OCP agents have a lower risk of VTE compared to first- and third-generation OCPs. The postpartum period carries the highest risk of VTE during pregnancy. Varicose veins are not considered as a risk factor for VTE. May-Thurner syndrome is among the predisposing factors for DVT; the syndrome is classically defined by a fibrotic and vascular thickening in the left common iliac vein which is the result of compressive force of the right common iliac artery (Page 500).

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CHAPTER 29

CARDIOVASCULAR GENETICS

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1. A 22-year-old female medical student is referred to the emergency department following an episode of syncope during exertion and ongoing palpitations. A twelve-lead ECG shows a wide QRS complex ventricular tachycardia (VT) with LBBB (left bundle branch block morphology). A normal rhythm, left anterior hemi-block and infrequent premature ventricular complexes (PVCs) are also detected in the baseline ECG. She mentions a history of SCD in two first-degree relatives and wants to know about the genetic cause of the disease in her own family. Which one of the following genes would you recommend to test at first step?

A. NKX2.5
B. MYBPC3
C. PKP2
D. KCNQ1

Answer: C

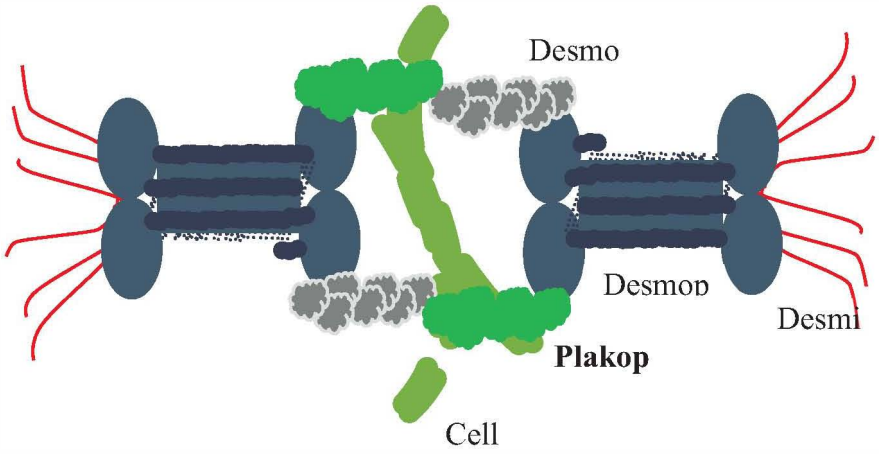

Figure 29.1 The role of plakophilin 2 in desmosome formation

NKX2.5 encodes a transcription factor that has a role in heart development. Mutations of this gene can cause different types of congenital heart disease. The cardiac MyBP-C protein is associated with the basic unit of muscle contraction, i.e. sarcomere. MYBPC3 mutation is one the most common cause of hypertrophic cardiomyopathy. It is also involved in left ventricular noncompaction cardiomyopathy, as well as dilated cardiomyopathy. KCNQ1 encodes a protein belonging to potassium channels in the inner ear and heart muscles. PKP2 encodes plakophilin 2 in the myocardium. This protein has a role in desmosome formation, which is involved in cell-to-cell junctions and signaling (Figure 29.1).

2. Genetic testing was done for the individual mentioned in the previous question. Eight common genes of ARVC/D were sequenced and a heterozygous variant, c.2577+1G>T, was found in the PKP2 gene. What is the meaning of c.2577+1G>T?

A. It is a missense pathogenic variant
B. It is a nonsense variant
C. It is a frameshift variant
D. It is a splice-site variant

Answer: D

Mutations are categorized based on their effects on DNA to base substitutions, deletions, insertions, inversions and translocations. A base substitution may change an amino acid into another amino acid (missense) or change an amino acid codon to a stop codon (nonsense). Insertion or deletion of a number of nucleotides that is not a multiple of three shifts the frame of reading when mRNA is translated (frameshift). An intron has three conserved elements (figure 29.2). Dinucleotides GT and AG at the beginning and end of introns are conserved. These dinucleotides are sites (splice site) for splicing proteins. c.2577+1G>T means the first nucleotide of intron, G changes to T.

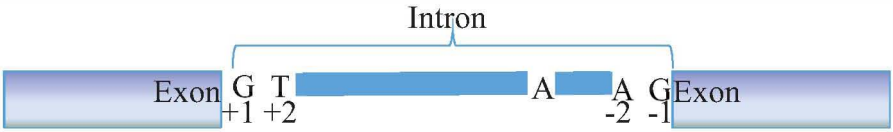

Figure 29.2 The three conserved elements of an intron

3. A chromosomal abnormality causes primary amenorrhoea, congenital heart disease, and skeletal anomaly. What is the name of this abnormality and which technique do you order to detect the underlying genetic mutation?

A. Turner syndrome, karyotyping
B. Noonan syndrome, karyotyping
C. Holt-Oram syndrome, gene sequencing
D. Ellis-van-Creveld syndrome, karyotyping

Answer: A

Karyotyping – ordering and pairing all the human chromosomes – is used to detect chromosomal abnormalities.

4. Genetic testing is usually ordered in the following scenarios except:

A. There is a strong suspicion for LQTS based on clinical examination.
B. For a family member when there is no index case with a known mutation in the family.
C. The patient shows asymptomatic QT prolongation without other clinical conditions that may prolong the QT interval.
D. Asymptomatic patients with QTc values >460 ms (prepuberty) or >480 ms (adults) from a serial 12-lead ECGs.

Answer: B

Genetic testing is usually indicated in following situations: strong suspicion for LQTS based on clinical examination, asymptomatic QT prolongation without other clinical conditions that may prolong the QT interval, and asymptomatic patients with QTc values > 460 ms (prepuberty) or > 480 ms (adults) from a serial 12-lead ECGs.

5. Large deletions in several genes including GATA4, NKX2-5, TBX5, BMP4 and CRELD1 have been reported to cause congenital heart disease (CHD). Which statement do you agree with, if you decide to detect genetic causes of CHD in your patients?

A. Order genetic testing to check these deletions using the MLPA technique.
B. Order whole-exome sequencing.
C. Order a karyotype first, followed by FISH analysis and whole-exome sequencing.
D. There is no way to determine large deletions in CHD patients.

Answer: B

Multiplex ligation-dependent probe amplification (MLPA), a multiplex PCR method, is used for detecting copy number variations.

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CHAPTER 30

RENAL DISORDERS AND CARDIOVASCULAR DISEASE

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1. For a 76-year-old man with a history of severe heart failure and recent pneumonia, a PAC is placed due to oliguria and rise in urea and creatinine following intravenous diuretic therapy for acute heart failure. The following hemodynamic values are measured:

RAP = 5, PAP = 25/12, PCWP = 9, BP = 85/60, HR = 120, CO = 3.6, CI = 2.1

Which approach is the better choice for him?

A. Hemodialysis
B. IV fluid
C. Norepinephrin
D. Increasing furosemide dose

Answer: B

This hemodynamic assessment demonstrates volume depletion as a cause of WRF. Over-diuresis or intercurrent illness may result in volume loss and renal dysfunction in AHF patients. IV fluid therapy should be the first treatment approach and the increasing of filling pressure and CI after fluid therapy shows that volume depletion could be considered a cause of oliguria.

2. A 54-year-old man with a history of ischemic cardiomyopathy and an LVEF of 15% is admitted due to acute decompensated heart failure. His baseline creatinine levels are 1.2 mg/dl. After standard treatments for acute heart failure, his clinical condition improves significantly and the patient has no dyspnea on the second day of his admission, but his creatinine has increased to 1.8 mg/dl at the end of the second day of treatment, returning to the baseline three days later. Which of the following statements about this patient is correct?

A. This condition is called pseudo-worsening renal function.
B. This patient has a very poor outcome due to a transient increase in serum creatinine.
C. His diuretic dose should be reduced.
D. ACEIs should be stopped.

Answer: A

If, during treatment for AHF, the clinical condition of patient improves and serum creatinine increases, this is pseudo-WRF. With continuing treatment and improvement of the congestion, renal function should return to baseline levels. Patients with pseudo-WRF have a good prognosis.

3. A patient has the following arterial blood gas results: pH 7.45; PCO2 = 30 mmHg, and $HCO3^-$ = 20 mEq/l. Which condition is indicated?

A. Metabolic acidosis
B. Metabolic alkalosis
C. Mixed acidosis
D. Respiratory alkalosis

Answer: D

Normal pH is between 7.35 up to 7.45. There is an inverse relationship between pCO2 and pH in a respiratory situation. In this condition, the PCO2 is low but the pH is at the top of the normal range. In alkalosis, pH is high. The results given above show respiratory alkalosis; the kidneys are trying to compensate for this condition through renal excretion of $HCO3^-$. Compensation has been successful, as can be seen by pH levels returning to the normal range.

4. A patient with a nasogastric (NG) tube that is attached to low suction is at risk of which acid-base disorder?

A. Metabolic acidosis
B. Metabolic alkalosis
C. Metabolic acidosis and metabolic alkalosis
D. None

Answer: B

The definition of metabolic alkalosis is a loss or deficit of H^+ ions or acids, or excessive ions of bicarbonate (base). The condition results in either base accumulation or acid loss without compensatory loss of bases in body fluids. This condition can be seen in hypovolemic shock and loss of gastric fluid. Vomiting or sucking gastric fluid via an NG tube, high intake of bicarbonate, whole blood transfusion in massive amounts, and hyperaldosteronism can cause metabolic alkalosis.

5. A patient who is found unresponsive has arterial blood gases drawn. The results are:
pH = 7.12, PCO2 = 90 mmHg, and $HCO3^-$ = 22 mmHg. What is the patient's acid-base disorder?

A. Respiratory acidosis
B. Metabolic acidosis
C. Respiratory acidosis and metabolic acidosis
D. Metabolic alkalosis and respiratory acidosis

Answer: A

The acid-base imbalance is respiratory acidosis but without compensation. Normal pH is 7.35 to 7.45 and normal PCO2 is 35 to 45 mmHg. pH is decreased in respiratory acidosis but PCO2 is elevated. Normal bicarbonate value is 22 to 27 mEq/L. Since the patient's bicarbonate is still in the normal range, the kidneys have not had enough time to adjust this acid-base imbalance. Also, the pH is not in the normal range.

6. A patient with confusion has these results: pH 7.38, pCO_2 = 60 mmHg, HCO_3^- = 31 mmol/l.

What is the patient's acid-base disorder?

A. Acute respiratory acidosis
B. Acute respiratory alkalosis
C. Acute respiratory acidosis and metabolic alkalosis
D. Acute respiratory acidosis and metabolic acidosis

Answer: C

In this acute elevation of pCO_2, the expected HCO_3^- is 24 + 2 = 26 mmol/l. If the actual measured value is higher than this amount, metabolic alkalosis must also be present.

7. A patient with an ileostomy is most at risk for which acid-base disorder?

A. Metabolic alkalosis
B. Metabolic acidosis
C. Respiratory acidosis
D. None

Answer: B

In intestinal secretions there are high levels of bicarbonate which are depleted through ileostomy.

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CHAPTER 31

ENDOCRINE DISORDERS AND THE CARDIOVASCULAR SYSTEM

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1. A 54-year-old woman is admitted with bradycardia, and fatigue. Her ECG shows sinus rhythm and a heart rate of 54 beats/min. In laboratory evaluation, a diagnosis of severe hypothyroidism is confirmed. Which of the following is not expected?

A. Pericardial effusion
B. Decreased CRP
C. Prolonged QT interval
D. Hyperhomocysteinemia

Answer: B

Hyperlipidemia, hyperhomocysteinemia, increased C reactive protein level, and altered coagulation parameters are seen in hypothyroidism.[1] ECG findings include sinus bradycardia, low voltage complexes prolonged PR and QT intervals, and flattened
or inverted T wave.[2] So the second option is wrong: CRP increases rather than decrease in hypothyroidism.

2. A 45-year-old man with growth hormone (GH) deficiency without replacement therapy is referred to your clinic for cardiovascular evaluation regarding the metabolic and anthropometric effects of the GH. Which of the following is more expected in his laboratory data?

A. Decreased LDL, increased TG, decreased waist circumference
B. Increased LDL, increased TG, decreased waist circumference
C. Decreased LDL, decreased TG, decreased waist circumference
D. Increased LDL, increased TG, increased waist circumference

Answer: D

In a patient with growth hormone deficiency, low-density lipoprotein (LDL) and triglycerides increase in both sexes, whereas decreased high-density lipoprotein (HDL) has been observed only in women.[3] The severity and duration of GHD is correlated with an adverse lipid profile, with an inverse association emerging between IGF-I and LDL-cholesterol levels.[4, 5] The prevalence of MetS is increased in GHD patients,[6] and lean individuals with GHD have a larger waist circumference and more abdominal adiposity, with a proportional increase in subcutaneous and visceral tissue with respect to control subjects.[7]

Exposure to large amounts of iodine during amiodarone treatment results in alterations in thyroid function tests (TFT). TSH is the first hormone to be affected. Its concentration rises within 48 hours. Subsequently, serum T4, free T4, and reverse T3 (rT3) concentrations increase. The increase in T4 levels is an expected finding that, in itself, does not constitute evidence for hyperthyroidism.[31] In amiodarone-induced thyrotoxicosis, T4 and T3 are high and TSH is suppressed, so choices A and B are incorrect.

3. A 50-year-old woman is evaluated for exertional chest pain. She notes her hands and feet have been enlarging and her shoe size has increased by two sizes. On physical examination, her blood pressure is 160/90, and laboratory tests show elevated levels of serum IGF1. Based on the exam and lab tests, what would you expect to see in her ECG?

A. Left axis deviation
B. Decreased QT intervals
C. ST-T wave elevation
D. Right axis deviation

Answer: D

Based on the history and lab test, the probable diagnosis is acromegaly. ECG changes at rest in acromegaly are left axis deviation, increased QT intervals, septal Q waves, and ST-T–wave depression.[8] Dysrhythmias that are seen in acromegaly include atrial and ventricular ectopic beats, sick sinus syndrome, and supraventricular and ventricular tachycardia.[9]

4. A 43-year-old man is evaluated for palpitations and dyspnea. He notes about 4 kg weight loss in the last 1 month. On physical examination his skin is warm and wet. His thyroid function test gives the following results: T4 = 15.6 (normal range is 5.5-10.4) TSH = less than 0.01(normal range is 0.5-5). Which clinical or hemodynamic change is more expected for him?

A. Decreased systemic vascular resistance (SVR)
B. Decreased cardiac preload
C. Diastolic hypertension
D. Increased systemic vascular resistance (SVR)

Answer: A

Based on the history and lab test, the diagnosis is thyrotoxicosis. The clinical signs and symptoms of hyperthyroidism include increased left ventricular mass,[10] angina pectoris, systolic murmurs, exercise intolerance, dyspnea on exertion, systolic hypertension.[11] If untreated, in the long-term cardiac hypertrophy and diastolic dysfunction may develop.[11,12,13] The hemodynamic effects of hyperthyroidism are: decreased systemic vascular resistance (SVR), increased heart rate, increased cardiac preload, and increased cardiac output.[14,15]

5. You have been asked to see a 43-year-old man for hypothyroidism and dyslipidemia. A recent workup to evaluate symptoms of fatigue, constipation and dry skin documented Hashimoto thyroiditis. His physician has started him on levothyroxine (for him). On physical examination, he is an apparently healthy man with some puffiness around the eyes.

Height = 188 cm; weight = 97.7 kg; BMI = 27.6 kg/m^2; BP = 137/85 mmHg, HR = 58 beats/min. His thyroid is enlarged and firm on palpation, and no nodules are palpated. The rest of his examination findings are normal. His laboratory test results are:

TSH = 37.0 mIU/l
Total cholesterol = 220 mg/dl
HDL cholesterol = 32 mg/dl
LDL cholesterol = 163 mg/dl
Triglycerides = 160 mg/dl

You do not institute any specific lipid-lowering therapy and plan to repeat a lipid profile in three months. Which of the following patterns of results are you most likely to receive after normalization of thyroid function test?

A. Decreased total cholesterol, increased HDL cholesterol, increased LDL cholesterol and decreased triglycerides
B. No change in total cholesterol, increased HDL cholesterol, no change in LDL cholesterol and decreased triglycerides
C. Decreased total cholesterol, no change in HDL cholesterol, decreased LDL cholesterol and no change in triglycerides
D. Decreased total cholesterol, decreased HDL cholesterol, decreased LDL cholesterol and increased triglycerides

Answer: C

Serum total cholesterol (TC), LDL-C, lipoprotein (a) [Lp(a)], oxi-LDL, ApoB remnants of VLDL, and chylomicron (CM) levels are increased in overt hypothyroidism,[16,17] while serum levels of triglyceride, high-density lipoprotein cholesterol (HDL) and VLDL are normal or slightly increased.[17,18] Changes in serum lipoprotein concentrations in hypothyroidism and during T4 (levothyroxine) therapy are also seen. In some patients, serum lipoprotein (a) concentrations are high and fall during T4 therapy,[19,20,21] whereas other patients have normal values that do not change during therapy.[22,23,24] In this particular case, we would expect LDL and total cholesterol to decrease and TG and HDL to undergo no significant changes.

6. A 39-year-old woman is referred for evaluation of endocrine hypertension. She has been hypertensive for five years and has no known family history of hypertension. She does not smoke cigarettes. She takes 10 mg amlodipine and 50 mg metoprolol daily. On physical examination, her blood pressure is 158/98 mmHg in each arm and her pulse rate is 68 beats/min. Her height is 162.6 cm, and weight is 58.2 k g (BMI = 22 kg/m^2). She is an apparently healthy, non-cushingoid woman without hyperpigmentation or striae. She has no arteriolar narrowing or retinal hemorrhages. Findings on cardiovascular and lung examinations are normal. No abdominal bruits are heard. The lab test results are Na = 136, k = 3.2, FBS = 96. Which complimentary test is necessary in this step?

A. Measurement of 24-hour urine catecholamine
B. Measurement of serum metanephrine
C. Test for serum aldosterone and plasma rennin activity
D. Measurement of 24-hour urine cortisol

Answer: C

Regarding the clinical and lab data, the most probable diagnosis for HTN in a middle-aged woman with hypokalemia is hyperaldosteronism. Therefore, testing for serum aldosterone and plasma rennin activity is the best option at this stage of her evaluation.

Options A and B are for diagnosing pheochromocytoma. Based on the history and clinical data, the pretest probability for pheochromocytoma is low. Option D is for evaluating Cushing syndrome; based on the physical exam, the pretest probability for Cushing syndrome is low.
A diagnosis of primary aldosteronism (PA) is usually made in patients who are in the third to sixth decade of life. The primary screening test for PA is measuring plasma aldosterone and plasma renin activity, and calculating an aldosterone to renin ratio (ARR).[25]

7. A 47-year-old male with palpitations and heat intolerance is referred to your clinic. His ECG shows atrial fibrillation on a physical examination. His pulse is 128 beats/min. He has a goiter and his skin is warm and wet. His lab test results are:

TSH <0.01 mIU/L, T4 = 20.6, FT4 = 3.6 ng/dl (normal: 0.08 - 1.8 ng/dl)

Treatment with methimazole at 20 mg/day and metoprolol at 50 mg/day is started. After 10 days, his symptoms improve and his pulse rate is 90 bpm, but he remains in atrial fibrillation. Which option is indicated at this time?

A. Electrical cardioversion
B. Anticoagulant
C. Amiodarone
D. ASA

Answer: D

The prevalence of atrial fibrillation in those with overt hyperthyroidism is 13.8%. Treatment of atrial fibrillation in the setting of hyperthyroidism includes beta-adrenergic blockade with a beta1-selective or nonselective agent to control the ventricular response.[26] Successful treatment of hyperthyroidism with radioiodine or antithyroid drugs and restoration of normal serum levels of T4 and T3 results in reversion to sinus rhythm in two thirds of patients within two to three months.[27,28] In younger patients with hyperthyroidism and atrial fibrillation in the absence of other heart diseases, hypertension, or other independent risk factors for embolization (CHADS VASc score = 0), the benefits of anticoagulation have not been proven and might be outweighed by the risks.[29] Aspirin provides an alternative for lowering the risk of embolic events in younger individuals and can be used safely.

8. A 33-year-old woman is referred with dyspnea on exertion. She is depressed and has noted 8 kg weight gain in the past 8 months. A physical examination reveals central obesity, hypertension and proximal weakness. Her lab tests show she has diabetes and hyperlipidemia. Which test is indicated for further diagnosis?

A. T4, TSH
B. 24h urine cathecholamine
C. Adrenal CT scan
D. 24-hour urine cortisol

Answer: D

Based on the history and physical examinations, the patient seems to has Cushing syndrome. A diagnosis of Cushing's syndrome can be based on increased cortisol secretion in at least two tests, including 24-hour urinary free cortisol, late-night salivary free cortisol, assessment of midnight plasma cortisol, or a 48-hour dexamethasone suppression test.[30]

9. A 52-year-old man presents with abdominal pain. An abdominal CT scan reveals a 3 cm right adrenal mass. He has noticed spells of palpitation and diaphorase in recent months, and has had 3 hypertension crises in the last month. On physical examination, his blood pressure is 16/100 mmHg, PR = 110. He does not receive any antihypertension medication. Which of the following steps would be incorrect?

A. Check 24h urine catecholamine
B. Begin phenoxybenzamine
C. Begin metoprolol
D. Measure plasma metanephrine

Answer: C

A possible diagnosis for this patient is pheochromocytoma. Diagnosis of pheochromocytoma is done biochemically by measuring cathecholamines and their metabolites in urine and serum. Therefore, answers A and D are correct. Beta-blocking drug therapy should not be initiated before establishing sufficient alpha blockade;[26] prescription of metoprolol before sufficient alpha blocking at this time may aggregate hypertension and predispose the patient to a hypertension crisis.

10. A 72-year-old man with refractory atrial fibrillation is placed on amiodarone. His baseline thyroid function tests are normal. Two months later he comes for follow-up visit. His laboratory test results are:
T4 = 14.2 μg/dl, (normal: 4-11 μg/dl), FT4 = 1.99 ng/ml (normal: .0.8-1.8 ng/ml), TSH = 1.89 (normal: 0.0.46-4.7 mIU/l)

Which of the following explains this lab result?

A. Type 1 amiodarone-induced thyrotoxicosis
B. Type 2 amiodarone-induced thyrotoxicosis
C. Change of thyroid-binding protein
D. Expected change in euthyroid patient on amiodarone

Answer: D
Exposure to large amounts of iodine during amiodarone treatment results in alterations in thyroid function tests (TFT). TSH is the first hormone to be affected. Its concentration rises within 48 hours. Subsequently, serum T4, free T4, and reverse T3 (rT3) concentrations increase. The increase in T4 levels is an expected finding that, in itself, does not constitute evidence for hyperthyroidism.[31] In amiodarone-induced thyrotoxicosis, T4 and T3 are high and TSH is suppressed, so choices A and B are incorrect,.choice c is incorrect because in protein binding change the free T4 is normal and we don't expected to have increased FT4regarding to above explanations the correct choice is D

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CHAPTER 32

HEART AND PULMONARY DISEASES

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1. A 65-year-old male heavy smoker with a history of diabetes mellitus is referred to your clinic with orthopnea and increased shortness of breath during the last week. He had two episodes of myocardial infarction one year ago, and was diagnosed with emphysema based on a high-resolution CT scan taken during a previous admission. He has no cough or sputum, but he has had dyspnea on exertion since that time. Clinical examination shows normal blood pressure; irregular pulse, around 105 beats/min; a respiratory rate of 20 breaths/min, but no respiratory distress; a diffusely enlarged thyroid; increased anterior-posterior diameter of the thorax; bilateral minimal expiratory wheezing; and decreased lung expansion. Prominent jugular venous pulsation, decreased heart sound, and an audible S3 sound on the left sternal border are also observed. Increased liver diameter with mild tenderness, and bilateral edema of lower extremity are obvious on general examination. An available recent chest x-ray shows apparently normal heart size and lung parenchyma, with no apparent evidence of hyperinflation. Lab results show normal WBC and electrolyte count, as well as normal CPK and troponin. Which of the following statements is *not* correct?

A. The risk of pulmonary hypertension is higher in this patient in comparison with the general population.
B. Ischemic heart disease is one of the leading causes of death in such patients.
C. The chance of a sleep breathing disorder in this patient is higher than the general population.

D. The recent exacerbation of his symptoms is most probably due to a recent pulmonary infection.
E. The patient's symptoms might be caused by pulmonary emboli.

2. Which of the following conditions is unlikely to be the cause of exacerbation of this patient's symptoms?

A. New cardiac infarction
B. Pulmonary thrombus embolization
C. Multifocal atrial tachycardia
D. Atrial fibrillation with rapid ventricular response
E. Thyrotoxicosis

3. Which of the following would you not order for this patient as a primary evaluation method?

A. Pulmonary CT angiography
B. Echocardiography and evaluation of cardiac function
C. Thyroid function tests and D-dimer assay
D. ECG
E. Doppler study of lower extremities

4. Which of the following is not appropriate for relieving the patient's symptoms until further evaluation?

A. Start diuretics with a control of blood pressure and electrolytes
B. Start bronchodilator with ipratropium bromide: two puffs, three times a day
C. Reevaluate his previous cardiac medication
D. Start an aminophylline bolus dose and then a maintenance dose if no side effects are noted
E. Start salbutamol at two puffs per symptom

Questions 1 to 4:
1: D, 2: A, 3: A, 4: D

Questions 1 to 4 refer to a usual case of routine practice. The prevalence of COPD and ILD is increasing every year due to industrialization, air pollution, smoking, and occupational exposure. Today we most often encounter patients with chronic respiratory disorders (CRDs) such as COPD and heart failure. Both of these conditions mimic each other's symptoms

and exacerbation of one may worsen the other and may exaggerate or obscure signs and symptoms of the other. The recent exacerbation of this patient's symptoms, along with the with the emphysema, coronary artery disease, and heart failure, may be due to a recent arrhythmia, a pulmonary infection, pulmonary emboli, or even a recent development of thyrotoxicosis. Psychological and environmental exposure are the other causes of exacerbation of symptoms in this patient. The normal-appearing cardiac size in his chest x-ray may be due to the presence of emphysematous hyper-aerated lung and pulmonary congestion due to heart failure and leading to normalization of parenchymal marking. The prevalence of pulmonary hypertension and sleep breathing disorders is higher in such patients in comparison with the general population, both in COPD and in heart failure patients. To go further in evaluating this patient with no obvious risk factors for pulmonary emboli, we need more clues from non-expensive modalities like D-dimer and doppler sonography. Based on the information we currently have, there is no evidence in favor of pulmonary infection.

5. Which of the following statements is not correct regarding sleep apnea and hypoventilation syndrome?

A. Obstructive sleep apnea is associated with heart failure but it remains to be demonstrated whether this association is independent.
B. All patients with heart failure should be assessed for sleep apnea.
C. Patients with obstructive sleep apnea (OSA) and heart failure (HF) may be less sleepy and obese than OSA patients without HF.
D. Continuous positive airway pressure (CPAP) always improves heart failure patients' cardiac function.
E. OSA patients with HF may be less sleepy and obese than OSA patients without HF

Answer: B

Obstructive sleep apnea (OSA) is associated with heart failure but it remains to be demonstrated whether this association is independent. OSA and heart failure (HF) patients may be less sleepy and obese than OSA patients without HF. Continuous positive airway pressure (CPAP) improves heart failure patients' cardiac function, if their polysomnography shows sleep apnea. Patients with heart failure should be assessed for sleep apnea *if* they

have clinical or historical symptoms suggestive of a sleep-related breathing disorder.

6. A 65-year-old male is presented to you with progressive dyspnea and a dry cough, which he has had for two 2 years. He has a history of smoking 14 packs of cigarettes per year. There is clubbing of all fingers, and bilateral Velcro-type crackling is audible in both hemi-thoraxes in the lower field, especially at the posterior. A high-resolution computerized tomographic scan shows bilateral interstitial and septal thickening. Which of the following is not correct about this patient's condition?

A. The prevalence of pulmonary hypertension (PH) is reported as being between 80 and 84%.
B. The prevalence of PH is higher when echocardiography is used as a diagnostic modality.
C. Respiratory failure in such patients eventually leads to right heart failure due to significant hypoxemia and distraction of the microvascular surface.
D. Some patients with this kind of disease may have involvement of myocardium at the same time.
E. None of the diseases like this have tendency to involve the pericardium.

Answer: E

This is a typical presentation of a patient with idiopathic interstitial lung disease (ILD). There are various diseases with systemic effects and lung involvement with autoimmune or idiopathic pathogenesis that present with similar symptoms to this patient. Distraction of the parenchyma and microvasculature plus hypoxia lead to pulmonary hypertension and right heart failure at the terminal phase in ILD patients. Pulmonary hypertension prevalences as high as 84% have been reported, but it is usually less than this. Rheumatological disorders may cause ILD and the involvement of pericardium, myocardium, or endocardium.

7. All of the following statements about the pathophysiology of chronic respiratory disease (CRD) are correct except for:

A. It is uncommon for CRD to lead to right heart failure or dilation without hypoxemia and or hypercapnia.
B. Pulmonary parenchyma and vasculature distraction are among the causes of PH.
C. Most CRD patients have PH that is not proportionate to lung involvement.
D. PH may dramatically improve with the treatment of hypoxemia.
E. Cheyne-Stoke and central sleep apnea are more common in patients with more severe CRDs.

Answer: C

It is uncommon for CRD to lead to right heart failure or dilation without hypoxemia and/or hypercapnia. In addition, some CRDs may cause pulmonary parenchyma and vasculature distraction and lead to increased vascular resistance and pulmonary hypertension. Most CRD patients have pulmonary hypertension that is proportionate to lung involvement, hypoxia and hypercapnia, but a minority have PH that is not proportionate to lung involvement. The latter need management similar to IPAH. Pulmonary hypertension may dramatically improve with the treatment of hypoxemia in the majority of cases and this is therefore the first step of therapy in these patients. Cheyne-Stoke and central sleep apnea are more common in patients with more severe diseases and lower PH (below 38 mmHg).

8. Which of the following statements about CRD patients is not correct?

A. In patients with heart failure due to chronic respiratory disease, the primary focus must be on the treatment of underlying lung diseases.
B. Treatment of hypoxia and hypercapnia are considered if the primary disease is not treatable.
C. Noninvasive treatment is beneficial in patients with respiratory failure.
D. Long-term O_2 does appear to be associated with modest decrease in PAPs.
E. Phosphodiesterase inhibitors and endothelin receptor antagonists have a dramatic effect on the treatment of pulmonary hypertension in these patients

Answer: D

In patients with heart failure due to chronic respiratory disease, the primary focus must be on the treatment of underlying lung diseases. Treatment of hypoxia and hypercapnia are considered if the primary disease is not treatable. Noninvasive treatment is beneficial in patients with respiratory failure. Long-term O_2 does appear to be associated with modest decrease in PAPs. Phosphodiesterase inhibitors and endothelin receptor antagonists have a controversial effect on the treatment of pulmonary hypertension in CRD patients.

9. Regarding patients with lung cancer, all of the following are possible manifestations of cardiac involvement except:

A. The most common cause of heart and pericardium involvement is the remote effects of the lung cancer.
B. Cancer chemotherapy or radiotherapy are both considered a common cause of heart failure and arrhythmia in patients with malignancy.
C. Myocardial necrosis may occur with chemotherapy.
D. Some cardiac arrhythmias are due to the remote effects of a tumor.
E. A local extension of the lung cancer through the pulmonary veins may occur.

Answer: A

Primary or metastatic lung cancer may involve the heart, often with local extension to the pericardium, or sometimes with remote effects. Cardiac involvement through the pulmonary vein and lymphatics is possible. In practice we most often find heart failure, pericarditis and arrhythmia caused by cancer chemotherapy. Chemotherapy for cancer may lead to myocardial necrosis.

10. Which of the following is correct with regards to carcinoid heart disease?

A. One of the pathognomonic findings of carcinoid heart disease is plaque-like deposits of fibrous tissue that most often involve the myocardium.
B. A carcinoid tumor may involve the heart due to its secretions.
C. Myocarditis is a dangerous involvement of the heart that may lead to cardiac arrhythmia and heart failure.
D. Left heart and valves are affected more commonly than right heart chambers by carcinoid tumors.
E. Treatment of carcinoid tumors with high-dose corticosteroids is recommended if there is myocardial involvement.

Answer: B

Carcinoid heart disease is a typical example of cardiac involvement caused by tumor secretions. Pathognomonic plaque-like deposits of fibrous tissue occur on the endocardium of valvular cusps of the right heart, cardiac chambers, and occasionally on the intima of the pulmonary arteries or aorta. Involvement of the right heart is more common than the left side due to the deactivation of carcinoid tumor secretions by lung tissue.

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CHAPTER 33

CARDIOVASCULAR DRUGS AND HEMOSTASIS

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1. Which antibiotics are preferable when a patient is taking warfarin and needs bacterial vaginosis treatment?

A. Metronidazole 500 mg orally twice daily for seven days
B. Clindamycin 300 mg orally twice daily for seven days
C. Azithromycin 1 g po once
D. Erythromycin 500 mg po q6hr for 7 days

Answer: B

Clindamycin does not have any interaction with warfarin.

Table 33.1 Antibiotic choices for patients on Warfarin

Presentation	Recommendations
Community Acquired Pneumonia	• Cefuroxime axetil (Ceftin) 250-500 mg twice daily • Ceftriaxone (500 mg twice daily)*
Urinary Tract Infection	• Nitrofurantoin 100 mg orally twice daily for 5 days • Fosfomycin trometamol 3 g orally in single dose • Cefaclor 250 mg every 8 hours (as capsules or oral suspension)
Otitis Media	• Cefaclor 250 mg every 8 hours (as capsules or oral suspension)
Pharyngitis	• Clindamycin 7 mg/kg orally three times daily (300 mg maximum dose) for 10 days
Bacterial Vaginosis	• Clindamycin cream 2% one full applicator (5 g) intravaginally once daily for 7 days
Cellulitis	• Clindamycin 300-400 mg orally 3 times daily • Linezolid 600 mg orally twice daily (pediatric dose 10 mg/kg orally every 8 hours, maximum 600 mg/dose)

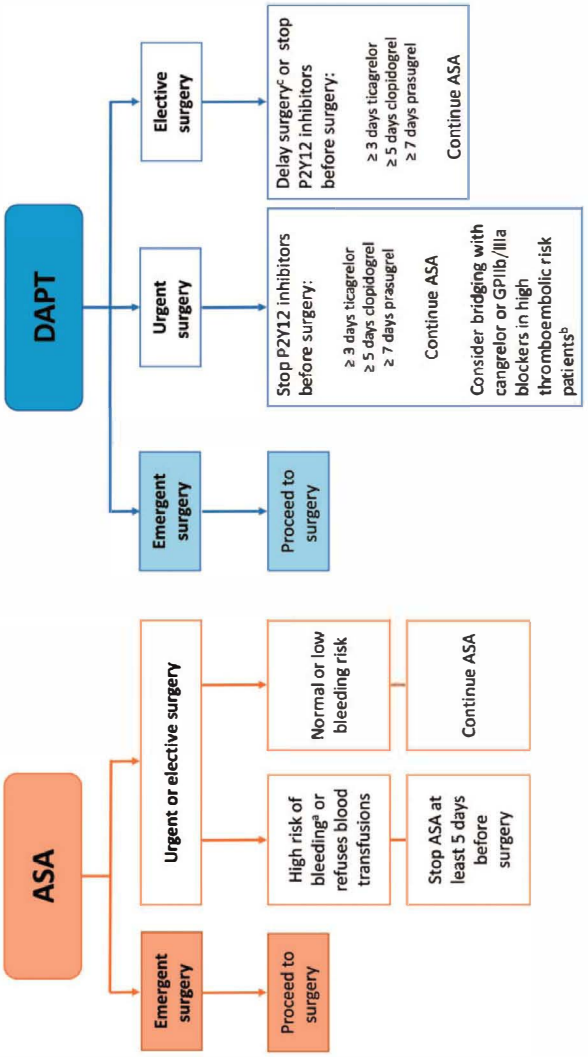
*The elevation of INR due to concomitant warfarin therapy and Ceftriaxone treatment has only been documented in a single case study in 2011, frequent INR monitoring is recommended.

Important factors to consider when reviewing the medication list include the patient's age, the dosage of medications, and his/her usage of antibiotics.

2. Before coronary artery bypass grafting, which of the following approaches is correct for a patient on dual antiplatelet therapy?

A. Stop clopidogrel for 5 days
B. Stop prasugrel for 7 days
C. Aspirin should not be stopped but should be continued at 81–324 mg/day
D. Stop abciximab for 12 hours
E. All of the above

Answer: E



[a] Complex and redo operations, severe renal insufficiency, hematological diseases and hereditary deficiencies in platelet function. [b] Recent stent implantation, recent thromboembolic event and alarming angiographic results. [c] Until the recommended DAPT period is completed. ASA: acetylsalicylic acid; DAPT: dual antiplatelet therapy; GPIIb/IIIa: glycoprotein IIb/IIIa

Figure 33.1 Management of antiplatelet therapy in patients having coronary artery bypass grafting surgery

3. A 76-year-old woman with diabetic nephropathy (serum Cr = 1.1, weight = 60 kg, height = 150 cm) comes to your clinic on Friday in preparation for her scheduled coronary angiogram with contrast on Monday. She currently takes NPH insulin, metformin, lisinopril, furosemide, and aspirin. On physical exam she is euvolemic. Which of these medications should she stop prior to her angiogram?

A. NPH insulin
B. Metformin
C. Lisinopril
D. Furosemide
E. Aspirin

Answer: B

Especially in light of her nephropathy and upcoming contrast administration, she should stop her metformin and remain off the medication 24 to 48 hours post-procedure to limit the potential for developing lactic acidosis.

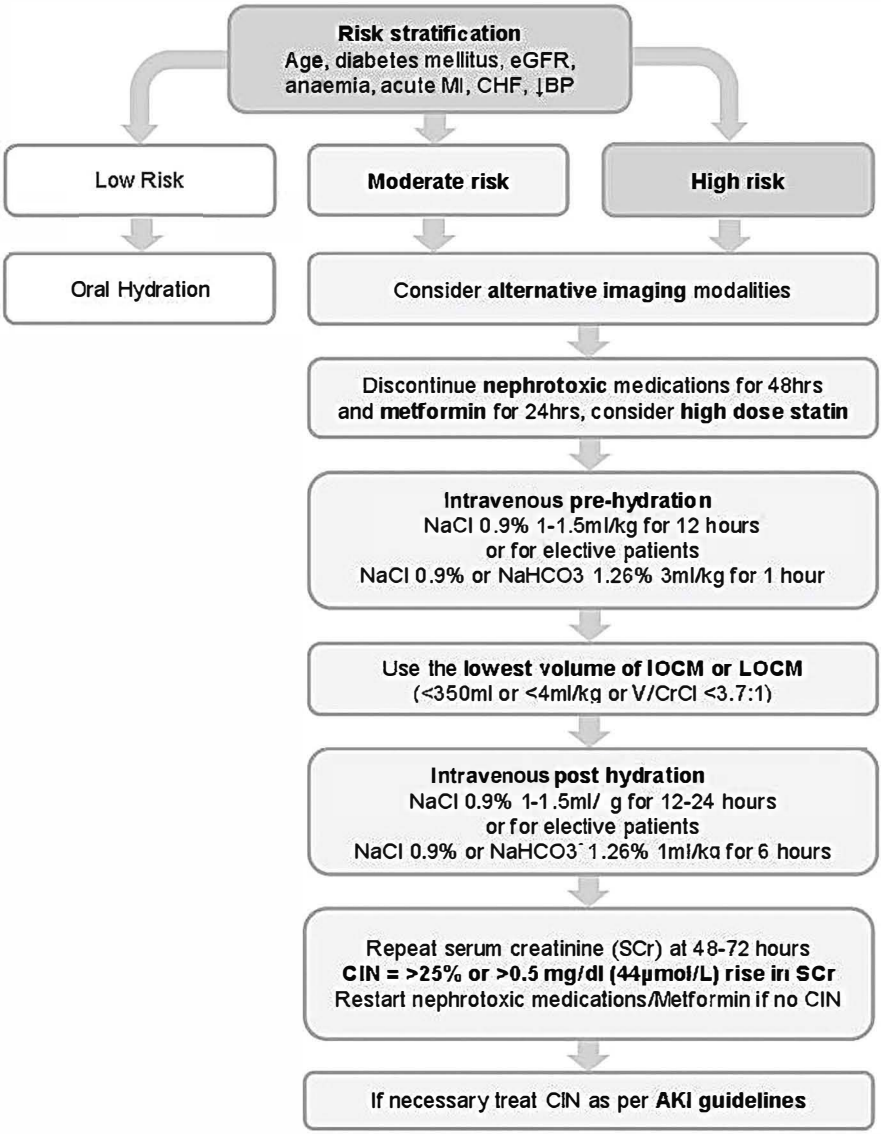


Figure 33.2 Risk stratification scheme for patient scheduled for coronary angiogram

4. Which one is incorrect regarding Amiodarone effects on the thyroid gland?

A. Those undergoing Amiodarone therapy require thyroid test screenings every three months.
B. To relieve the hyperthyroidism caused by Amiodarone, drug discontinuation is enough.
C. Amiodarone's effects on the thyroid are dose dependent.
D. Amiodarone's onset of action has no relationship with the duration of the treatment.

Answer: B

Those undergoing Amiodarone therapy should be screened for thyroid function tests every three months. Amiodarone mechanism of action on thyroid gland has no dose dependency and can appear at any time interval. Since Amiodarone has high lipid solubility and long duration of action, its effects can be present even after a year from its discontinuation.

Table 33.2 Effects of amiodarone on thyroid function tests in euthyroid subjects

Thyroid Hormone	**Acute effects (up to 3 months)**	**Chronic effects (> 3 months)**
Total and free T4	↑ 50%	Remains ↑ 20-40% of baseline
Total and free T3	↓ 15-20%, remains in low-normal range	Remains ↓ 20%, remains in low-normal range
rT3	↑ >200%	Remains ↑ >150%
TSH	↑ 20-50%, transient, generally remains <20 mU/L	Normal

Modified from: Basaria S, Cooper DS, Amiodarone and the thyroid. Am J Med 2005;118: 706-714.

5. Which of the following statements is accurate regarding triple antithrombotic therapy after MI in a patient with AF arrhythmia?

A. The duration should be minimized and proton pump inhibitors should be used.
B. Triple therapy should not be used.
C. Dual therapy is a more logical treatment with a low risk of bleeding.
D. Triple therapy involves one antiplatelet plus two anticoagulant treatments.

Answer: A

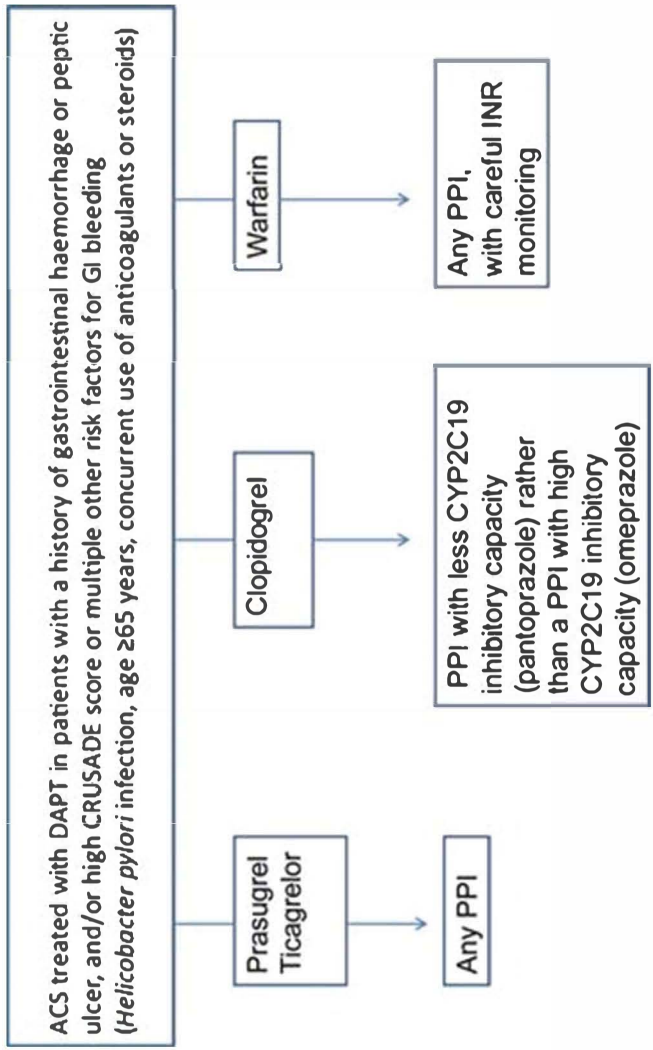


Figure 33.3 Antiplatelets and anticogulants interaction with PPI's

5. JC is a 64-year-old female with a mechanical mitral valve undergoing noncardiac surgery needing interruption of anticoagulation. What would you recommend?

A. Minimize nonanticoagulated period; bridge with heparin or LMWH
B. No bridging needed
C. Bridge with novel anticoagulant
D. Use fresh frozen plasma to cover surgery

Answer: A

Table 33.3 Perioperative management of anticoagulation therapy: Decision to bridge

	High bleeding risk procedures	Low bleeding risk procedures	Minimal bleeding risk procedures
High risk	Warfarin interruption: YES Bridging with LMWH: YES	Warfarin interruption: YES/NO Bridging with LMWH: YES/NO	Warfarin interruption: NO Bridging with LMWH: NO
Moderate risk	Warfarin interruption: YES Bridging with LMWH: YES/NO	Warfarin interruption: YES/NO Bridging with LMWH: YES/NO	Warfarin interruption: NO Bridging with LMWH: NO
Low risk	Warfarin interruption: YES Bridging with LMWH: NO	Warfarin interruption: YES Bridging with LMWH: NO	Warfarin interruption: NO Bridging with LMWH: NO

How to bridge:

i. Stop warfarin for 48 hours
ii. Start lovenox at 1mg/kg SQ BID for 6 doses
iii. Stop lovenox the morning before surgery.

7. Which of the following combinations do not have the potential for drug-drug interactions?

A. Gemfibrozil and atorvastatin
B. Warfarin and fenofibrate
C. Warfarin and voriconazole
D. ASA and metronidazole

Answer: D

Table 33.4 Potential Drug Interactions with NSAIDs

Affected Drugs	Effects of Drug-Drug Reactions	Management Options
Oral anticoagulants	Increased oral warfarin activity Increased risk of bleeding especially GI bleeding	Monitor prothrombin time Monitor for occult blood in stool/urine Monitor concurrent use of aspirin
Lithium	Increase lithium blood concentration Lithium Toxicity	Monitor lithium concentration carefully
Antihypertensive agents	Antihypertensive effects antagonized Hyperkalemia may occur with ACE inhibitors or potassium sparing diuretics	Monitor blood pressure and cardiac function Monitor potassium serum levels
Digoxin	Inhibition of digoxin clearance Adjust digoxin dose as necessary	Monitor digoxin serum concentrations
Phenytoin with ibuprofen or High dose salicylates	Increase phenytoin levels Adjust Phenytoin dose if necessary	Monitor unbounded phenytoin concentration
Methotrexate	Reduced renal clearance of methotrexate	Avoid NSAIDs with high dose of methotrexate

Antacids with salicylates or naproxen sodium	Salicylate concentrations possibly reduced by 25% Aluminum hydroxide decreases naproxen sodium absorption	Monitor patient's clinical status
Probenecid with naproxen Sodium	Reduced clearance of naproxen sodium	Monitor for adverse effects
Aminoglycosides	Inhibits aminoglycoside renal clearance	Monitor antibiotic concentrations
Insulin with salicylates	Possible decrease hypoglycemic effect with large salicylate doses	Monitor blood glucose
Cephalosporins	Possible increased bleeding risk	Avoid concurrent use

Table 33.5 The effects of drugs on INR

	Drugs/ Natural Products	Mechanism of Action	Clinical Implications	Clinical Management	Other Factors that affect INR
Drugs that result in increased INR	Antifungal agents	Reduces warfarin clearance	Bleeding When INR > 3 patient is over-anticoagulated Signs of bleeding: persistent nausea, nose-bleed, dark red/brown urine and blood in bowel movement The longer it takes the blood to clot, the higher the PT and INR	Monitor INR	Lifestyle changes: Increase in alcohol use or binge drinking Decrease in consumption of vitamin K containing foods
	Bactrim			Avoid combination	
	Amiodarone			Give ½ usual dose of Warfarin	
	Acetaminophen			Use doses below 2 g/day	
	Ethanol			Avoid binge drinking	
	H2-Blockers, Statins, & Fibrates			Monitor INR if meds are added, discontinued or altered	
	Pradaxa, Xarelto, low molecular weight Heparin	Interrupts clotting cascade		Monitor, may increase life threatening bleeding	
	Aspirin, Plavix	Inhibits platelet aggregation		Use low ASA or avoid use	
	Vitamin E	Interfere with clotting factors		Monitor Vitamin E above 800 IU per day	

Drugs that result in decreased INR	Barbiturates	Increase or enhances Warfarin clearance	Clotting When INR < 2 there is a risk for blood clot. Patient is (under-anticoagulated).	Monitor INR if medications are added, discontinued or altered	Lifestyle Changes: Decrease in baseline alcohol use increase in consumption of vitamin K containing foods
	Phenytoin				
	Dicloxacillin, Naficillin				
	St. Johns Wort				
	Green tea, Vitamin K	Antagonism of Warfarin's mechanism of action		Instruct patient to keep amount of supplement in diet consistent as possible	
	Spironolactone	Increase concentration of clotting factors		Monitor INR closely when dug is initiated, discontinued or dose is changed	

8. A 56-year-old woman with a history of aortic valve regurgitation and a mechanical aortic valve comes to your office for anticoagulation recommendations prior to dental surgery. She is chronically anticoagulated with warfarin with a goal INR of 2.5. Her EF is 35% by echocardiogram. Which of the following do you recommend?

A. Stop her warfarin 10 days prior to the dental surgery and restart the day after the procedure.
B. Stop her warfarin 3 days prior to the dental surgery and admit her to hospital prior to the procedure for heparinization. She should remain in the hospital until her INR is above 2.0.
C. Stop her warfarin now, begin full-dose aspirin (325 mg) and perform the dental surgery in 7 days.
D. Admit her directly to hospital for 10 mg of vitamin K. Once her INR is 1.7, proceed with the dental surgery. Keep her in hospital until her INR is higher than 2.0.
E. Continue warfarin, but reduce the dose such that the goal INR is now 2.0 and proceed to dental surgery.

Answer: B

According to ACC/AHA guidelines, patients with a bileaflet mechanical AVR with no risk factors (i.e. AF, previous thromboembolism, LV dysfunction, hypercoagulable conditions, older generation thrombogenic valves, mechanical tricuspid valves, or more than one mechanical valve) should stop their warfarin 48 to 72 hours before such a procedure (so that their INR falls to 1.5) and restart within 24 hours after the procedure. Heparin is usually unnecessary. The guidelines are summarized below.

ACC/AHA anticoagulation guideline summary: Antithrombotic therapy in patients with mechanical heart valves

Class I: There is evidence and/or general agreement that antithrombotic therapy is indicated in patients with mechanical heart valves in the following settings:

- Warfarin to achieve a goal INR of 2.0 to 3.0 after:
    1. Aortic valve replacement (AVR) with bileaflet mechanical or Medtronic Hall valves if no risk factors* are present.

- Warfarin to achieve a goal INR of 2.5 to 3.5 after:
    1. AVR with bileaflet mechanical or Medtronic Hall valves if risk factors* are present.
    2. AVR with Starr-Edwards or disc valves other than Medtronic Hall if no risk factors* are present.
    3. Mitral valve replacement (MVR) with any mechanical valve.
- Role of aspirin:
    1. After AVR or MVR in patients who cannot take warfarin, at a dose of 75 to 325 mg/day.
    2. At a dose of 75 to 100 mg/day in addition to warfarin in all patients with mechanical valves and in patients with biological valves who have risk factors*.

Class IIa: The weight of evidence or opinion is in favor of the usefulness of antithrombotic therapy in patients with mechanical heart valves in the following setting:

- In the first three months after AVR, warfarin to achieve a goal INR of 2.5 to 3.5.

Class IIb: The weight of evidence or opinion is less well established for the usefulness of antithrombotic therapy in patients with mechanical heart valves in the following setting:

- In high-risk patients in whom aspirin cannot be used, clopidogrel (75 mg/day) or warfarin to attain a goal INR of 3.5 to 4-5.[5]

9. BH is a 60-year-old male who is transferred to the catheterization laboratory for an emergent PCI following an ST elevation myocardial infarction (STEMI). Two drug-eluting stents are placed. His past medical history includes hypertension (HTN) and diabetes mellitus type 2. The patient's current medications include captopril, atorvastatin, metformin, glibenclamide, and pantoprazole. To prevent stent thrombosis, which of the following antiplatelets would be best to start in combination with 324 mg aspirin?

A. Clopidogrel 300 mg loading dose, then 75 mg PO daily
B. Clopidogrel 600 mg loading dose, then 75 mg PO daily
C. Prasugrel 60 mg loading dose, then 10 mg PO daily
D. Ticagrelor 180 mg loading dose, then 90 mg PO twice daily

Answer: C

Clopidogrel, prasugrel, and ticagrelor may all be used in this patient for dual antiplatelet therapy; however, significant benefits have been seen with prasugrel, particularly in patients with STEMI and diabetes mellitus.

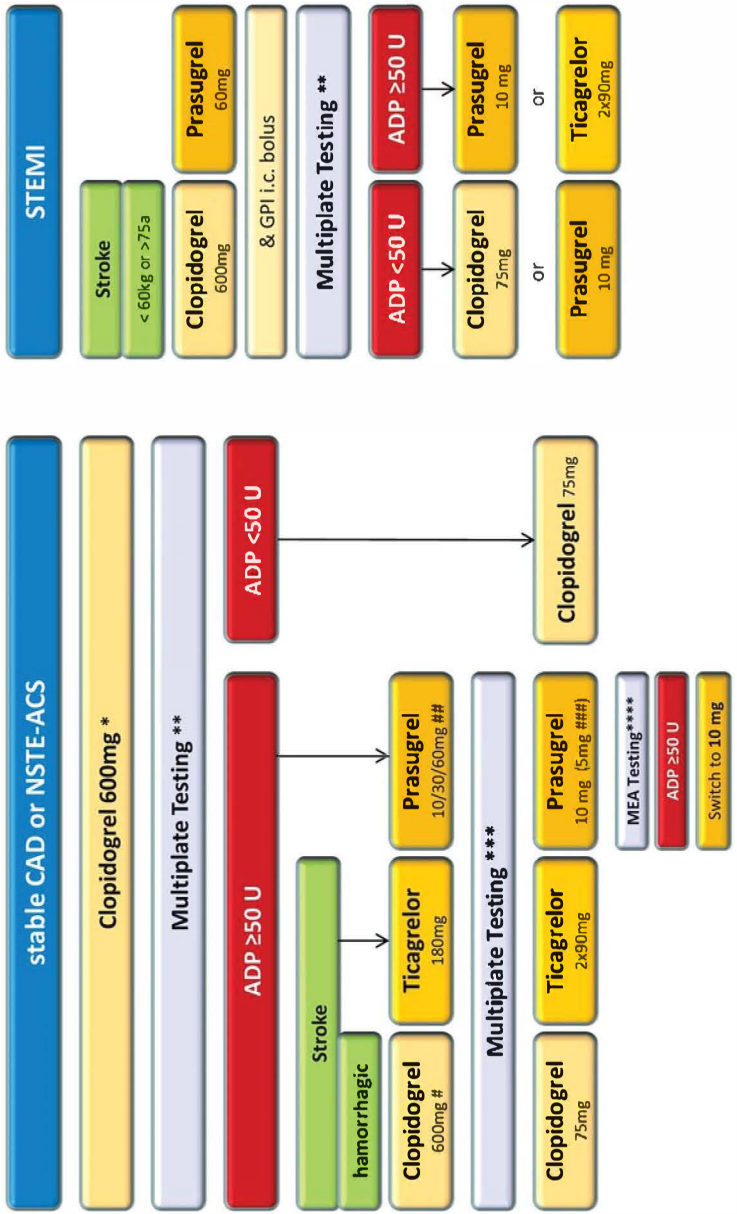


Figure 33.5 Antiplatelets treatment algorithm in STEMI vs NSTEMI

10. Which of the following statements is true?

A. Patients aged 75 or more with ST-segment elevation MI (STEMI) should take a clopidogrel 300 mg loading dose, then 75 mg PO daily for 14 days up to 12 months (if no bleeding).
B. LMWH is indicated for patients with HIT.
C. Alteplase has a fast onset of action with a prolonged duration of action.
D. Higher doses of heparin are typically required in pregnancy.

Answer: D

Due to the presence of more heparin-binding proteins, a greater plasma volume, increased renal clearance, coagulation factors, and heparin degradation in the placenta, heparin dosing is unpredictable in pregnancy, with patients invariably requiring higher doses.

Pregnancy is a hypercoagulable state:

Thrombogenicity:

- Increase in procoagulant factors VII, VIII, X, and fibrinogen, as early as 12 weeks
- Fibrinolytic activity is reduced, progressively increasing PAI-1 and PAI-2.
- Platelet activation and increased production of thromboxane

Anticoagulants

- Protein S reduced by 40-50%, while antithrombin III and protein C remain constant
- Reduced sensitivity to anti-aggregation effects of prostacyclin
- Vasorelaxation

11. For a patient with HIT in the setting of renal failure, the following statements are true regarding treatment, except:

A. Lepirudin is indicated for patients with HIT in the setting of renal failure.
B. Patients with HIT should receive argatroban in the setting of renal failure.
C. Warfarin (Coumadin) monotherapy may induce thrombosis and is contraindicated in acute settings.
D. Platelet transfusions should be avoided in HIT, as they may increase the thrombogenic effect.

Answer: A

Lepirudin is renally excreted and patients with HIT should receive Argatroban in the setting of renal failure.

Table 35.6 Drug treatment guide for patients with HIT

Drug	Intermittent hemodialysis (3 times weekly)		Continuous dialysis	
	Dose	**Monitoring**	**dose**	**Monitoring**
Lepirudin	0.02-0.15 mg/kg bolus at start of dialysis	Monitor to maintain 2-3× baseline aPTT; can also monitor with ECT	Initial bolus 0.01 mg/kg; subsequent boluses 0.005-0.01 mg/kg	Monitor to maintain 1.5-2× baseline aPTT; can also monitor with ECT
Argatroban	250 µg/kg bolus; 2µg/kg/min infusion until 1 h before end of dialysis	Monitor to maintain 1.5-3× baseline aPTT; can also monitor with ECT	0.5-2.0 µg/kg/min	Monitor to maintain 1.5-3× baseline aPTT; can also monitor with ECT
Danaparoid	Before first 2 hemodialysis sessions: 3750 U (2500 U*). For subsequent sessions: 3000 U (2000 U*) if predialysis anti-Xa is <0.3 U/ml; 2500 U (1500 U*) if predialysis anti-Xa is 0.3-0.35 U/ml; 2000 U (1500 U*) if predialysis anti-Xa is 0.35-0.4 U/ml; 0 if predialysis anti-Xa is >0.4 U/ml	Monitor anti-Xa levels to maintain within the range 0.5-0.8 U/ml	Initial bolus 2500 U (2000 U*); 600 U (600 U*) for first 4 h; 400 U (400 U*) for next 4 h; subsequently, 200-600 U (150-400 U*)	Monitor anti-Xa levels to maintain within the range 0.5-0.8 U/ml

*Doses in parentheses for danaparoid use are for patients weighing <55 kg. Abbreviations: aPTT, activated partial thromboplastin time; ECT: ecarin clotting time.

12. Which of the following antiplatelet can be reversed by platelet transfusion?

A. Abciximab
B. Tirofiban
C. Eptifibatide
D. ASA

Answer: A

There is an increased bleeding risk with urgent CABG. Abciximab therapy associated with minimal increase in blood loss with urgent CABG with conventional heparin dosing and platelet Tx transfusions. Patients requiring surgery in the first 12 hours are at highest risk.

13. Which of the following statements about prothrombin complex concentrate is incorrect?

A. PCC is not indicated for urgent reversal of VKA anticoagulation in patients without acute major bleeding.
B. The main benefits of PCC are the speed of administration, its reliable effect, and small treatment volume.
C. Fluid overload or similar cardiac events with PCC are significantly higher than with red blood cell transfusions.
D. PCC contains proteins C and S, as well as factors II, VII, IX, X, and traces of heparin.

Answer: C

The risk of fluid overload or similar cardiac events is significantly lower with prothrombin complex concentrate (3% vs 13%).

Table 33.7 Comparison of PCC, FFP and Factor VIIa when Reversing Warfarin for Emergency Surgery

	Time for Normalizing INR (Includes preparation and Adminis-tration)	**Immune Issues**	**Hemostasis**	**Volume**
PCC	<60 minutes	Virally deactivated No RBCs	Quantified levels of factors	<100 ml
FFP	>2 hours, depending on amount transfused and rate of transfusion	ABO crossmatch TRALI	Factors concentrations are not quantified and may vary	May need 15 to 20 ml/kg
Factor VIIa	<60 minutes	Minimal	Risk of clot/ thromboembolism	<100 ml

PCC = Prothrombin Complex Concentrate; FFP = Fresh Frozen Plasma; INR = international normalized ratio; RBC = red blood cells; TRALI = transfusion related acute lung injury.

14. Which of the following treatments is the most important in terms of reducing the risk of late stent thrombosis with drug-eluting stents (DESs)?

A. Pretreatment with abciximab
B. Optimal stent deployment (complete apposition of stent with vessel wall)
C. The introduction of more potent antiplatelet agents (e.g., ticlopidine, clopidogrel)
D. Aggressive anticoagulation with warfarin and aspirin

Answer: B

With the recent widespread implementation of DESs, late thrombosis data started appearing one year after placement. Patients with thrombosis had longer lesions, longer stents, more stents-per-lesion, and more stent overlap.

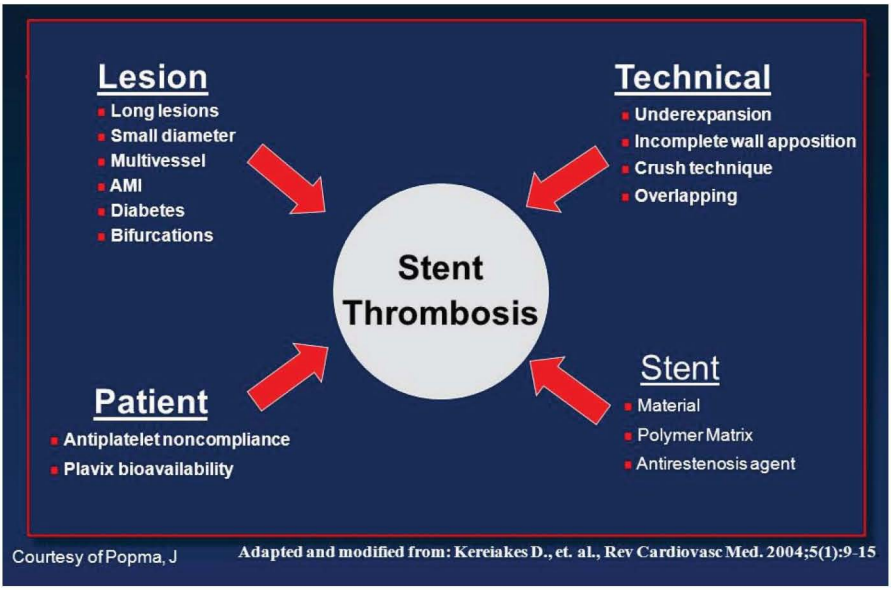


Figure 33.6 Stent Thrombosis: A Multifactorial Problem[3]

15. A 50-year-old female patient is referred to the surgical outpatient department of a tertiary care rural teaching hospital with a complaint of acute pain in the right side of her abdomen since the previous day. To reconfirm the diagnosis, she is advised to undergo a CT scan of her abdomen. For this procedure, she is given an iodinated radio-contrast dye (Urovison-Diatrizoate Meglumine/Diatrizoate sodium injection USP) by an intravenous route. Five minutes after administration of the radio-contrast media, she starts to suffer from shivering, drowsiness, redness and edema on her face and chest, breathlessness, and altered sensorium with cold extremities. Which of the following is incorrect regarding a patient who has had an anaphylactoid reaction to a contrast agent in the past?

A. The patient is at increased risk for a second anaphylactoid reaction if exposed to a contrast agent again.
B. The likelihood of a second anaphylactoid reaction can be reduced by the use of a low osmolar contrast agent.

C. The likelihood of a second anaphylactoid reaction can be reduced by the administration of corticosteroids before the second procedure.
D. The patient may have been exposed to NPH insulin in the past, because NPH insulin increases the likelihood of an anaphylactoid contrast reaction.

Answer: D

The risk of an anaphylactoid reaction to contrast media is increased if a previous reaction has occurred but is not related to use of NPH insulin. Previous NPH use increases the risk of anaphylaxis to protamine (which is used to reverse the effects of heparin).

Table 33.8 Anaphylactoid Reaction Prophylaxis for Emergency Percutaneous Coronary Intervention[4]

Medication	Dosage	Route	Mechanism
Standard Agents			
Methylprednisolone or	80-125 mg	IV	Anti-inflammatory
Dexamethasone or	16 mg		
Solu-Cortef	100 mg		
Cimetidine	300 mg	IV	H2 blockage
Benadryl	25-50 mg	IV or PO	H1 blockage
Not Considered as Standard Therapy			
Montelukast (singulair)	10 mg	PO	Leukotriene inhibitor
Prochlorperazine	10 mg	IV	Decrease nausea

Allergic and anaphylactoid reaction:

- Materials that may cause allergic or anaphylactoid reactions:
  i. Local anesthesia
  ii. Iodinated contrast agent
  iii. Protamine
  iv. Preservatives
- Skin testing with the intended agent at 1:1000 if desired
- Iodinated contrast agent

i. Most common in triggering allergic reactions – up to 1%
ii. Anaphylactoid reaction – involves degranulation of circulating basophils and tissue mast cells by direct complement activation
iii. Other clinical manifestations caused by histamine or other agents – sneezing, urticaria, angioedema, bronchospasm, warm shock.

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CHAPTER 34

STABLE ISCHEMIC HEART DISEASE

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1. Which of the following is true about the primary stages of atherosclerosis formation?

A. Positive remodeling occurs.
B. In this stage, stress testing could suggest the presence of CAD.
C. A coronary angiogram can reveal the presence, extension, and even the content of atherosclerotic plaque.
D. Angina is common in this stage.

Answer: A

In the primary stages of atherosclerosis formation, plaque may extend eccentrically and outward without compromising the lumen (positive remodeling). In this stage, stress testing or angiography may not suggest the presence of CAD. However, imaging modalities such as cardiac computed tomography angiography (CCTA), intravascular ultrasonography (IVUS), or optical coherence tomography (OCT) can reveal the presence, extension, and even content of atherosclerotic plaque. The further progression of atherosclerosis and propagation of the plaque into the lumen can result in hemodynamic obstruction and angina.[1]

2. Which of the following symptoms is not typical of myocardial ischemia and infarction secondary to cocaine usage?

A. Increased myocardial oxygen demand.
B. Coronary vasoconstriction.
C. Accelerated atherosclerosis.
D. Increased platelet activity and aggregation.
E. Elevated levels of fibrinogen and von Willebrand factor.
F. Decreased plasminogen activator inhibitor activity.

Answer: F

Use of cocaine increases myocardial oxygen demand, and coronary vasospasm can lead to myocardial infarction, especially in young individuals. Moreover, long-term cocaine usage can cause premature development of atherosclerosis.[2] The mechanisms of cocaine-Induced myocardial ischemia and infarction are listed below.[3]

Increased myocardial oxygen demand:
i. Heart rate elevation
ii. Increased contractility
iii. BP elevation

Coronary vasoconstriction:
i. Alpha-receptor stimulation
ii. Impaired intracellular calcium handling
iii. Impaired NO production
iv. Increased endothelin-1 levels

Prothrombotic effect:
i. Increased platelet activity and aggregation
ii. Elevated levels of fibrinogen and von Willebrand factor
iii. Increased plasminogen activator inhibitor activity
iv. Accelerated atherosclerosis
v. Small-vessel disease
vi. Plaque erosion
vii. Coronary dissection

3. A 67-year-old man with a history of diabetes mellitus and cigarette smoking is referred to your clinic complaining of chest pain that develops with exertion, but notes that when he continues the exertion at the same or even greater levels after a brief rest period, the symptoms are significantly

reduced or even relived. He has suffered from these symptoms for the last six months. Which of the following is not true?

A. Postprandial angina may be observed in this patient.
B. Nitroglycerine may provide relief for the chest pain.
C. The main mechanism is ischemic preconditioning and collateral recruitment.
D. Searching for an exact etiology other than a cardiovascular one should be considered.

Answer: D

In patients with SIHD, postprandial angina may be assigned as severe CAD, presumably caused by the redistribution of coronary blood flow (steal phenomenon) after a meal.[4] If the duration of angina after rest or nitroglycerin is more than 5 to 10 minutes, this suggests that the pain may be secondary to other causes or severe ischemia, as with acute coronary syndrome (ACS). Response to nitroglycerin may be observed in the setting of esophageal pain and other syndromes.[5] First-effort or warm-up angina are the terms for angina that develops with exertion but symptoms are significantly reduced or completely relieved after continuing the exertion at the same or even greater levels after a brief rest period. The main mechanism of this phenomenon is ischemic preconditioning and collateral recruitment.[6]

4. A 45-year-old woman without any significant past medical history presents in your clinic with typical angina, which she has had for one year. In terms of positive exercise ECG testing, she undergoes a coronary angiography, but all the findings revealed normal coronary arteries. Which of the following sentences is *not* correct about this patient?

A. Myocardial perfusion imaging may reveal reversible stress-induced defects.
B. Generally, the long-term prognosis in this patient is poor.
C. The potential explanations of this complication include endothelial and microvascular dysfunction and impaired coronary flow reserve.
D. Chest discomfort without ischemia in some individuals may be caused by abnormal pain perception or sensitivity.

Answer: B

Some patients, especially women, experience typical anginal symptoms with objective evidence of myocardial ischemia despite normal coronary angiograms (angina without flow-limiting epicardial coronary stenosis, or syndrome X). These patients have ECG changes (ST-segment depression, T-wave inversion, or both) at rest or during the stress test, as well as evidence of reversible stress-induced myocardial perfusion defects on nuclear imaging. Although a benign long-term prognosis is generally emphasized for this syndrome, recently increased risk for adverse outcomes in certain subsets of these patients has been recognized.[7,8] Although the exact pathophysiologic mechanism of this syndrome is still unclear, potential explanations include endothelial and microvascular dysfunction and impaired coronary flow reserve. Other mechanisms include coronary vasospasm and myocardial metabolic abnormalities. However, chest discomfort without ischemia in some individuals may be caused by abnormal pain perception or sensitivity.[1,9]

5. Which of the following statements is not correct regarding a patient with stable ischemic heart disease (SIHD)?

A. Generally, the physical examination findings of patients with SIHD are normal.
B. Examination during an episode of angina may reveal a new mitral regurgitation murmur.
C. Approximately half of patients with SIHD have normal resting ECG findings, even in those with severe CAD.
D. The most common abnormal findings on ECGs in patients with SIHD are significant ST-T–wave inversions with or without abnormal Q waves.
E. The presence of left bundle branch block or left anterior fascicular block in patients with SIHD is often associated with LV dysfunction, multivessel CAD, and a poor prognosis.

Answer: D

Generally, the physical examination findings of patients with SIHD are normal. However, elevated blood pressure, xanthomas, and retinal exudates suggest the presence of CAD risk factors. It is also recommended that the body mass index is calculated and a search done for detection of peripheral vascular disease as well as comorbid conditions such as thyroid disease, renal disease, and diabetes.
Examination during an episode of myocardial ischemia may reveal an

increase in heart rate and blood pressure, new mitral regurgitation murmurs caused by papillary muscle dysfunction or an increase in left ventricular (LV) filling pressure, pulmonary congestion findings (e.g., rales on auscultation) caused by decreased LV compliance, paradoxic S2 splitting caused by delayed relaxation of the LV myocardium and delayed closure of the aortic valve, S4 caused by a decrease in LV compliance, and S3 caused by LV systolic dysfunction. Relief from the ischemia results in a cessation of the symptoms and signs that occurred during the episode.[10-13]

A resting 12-lead ECG should be obtained for all patients with suspected CAD. Approximately half of patients with SIHD have normal resting ECG findings, even in those with severe CAD, and this finding suggests the presence of normal resting LV function and a good long-term prognosis. Patients with previous MI have specific ECG findings, including ST-T–wave changes and Q wave formation. However, the most common abnormal findings on ECGs in patients with SIHD are nonspecific ST-T–wave changes with or without abnormal Q waves. Also, a resting ECG is recommended in all patients during or immediately after an episode of angina. About 50% of patients have new ECG changes during an episode of angina; the most common is ST-segment depression. The presence of ST-T–wave changes, especially during an episode of angina, can correlate with the severity of the CAD and suggest an adverse prognosis.[14]
The presence of conduction disturbances, including left bundle branch block and left anterior fascicular block, is often associated with LV dysfunction, multivessel CAD, and a poor prognosis[15-17] Arrhythmias, especially ventricular premature beats, have low sensitivity and specificity for detecting CAD. ECG findings in favor of LV hypertrophy correlate with systemic hypertension, aortic stenosis, hypertrophic cardiomyopathy, or previous MI with remodeling. Thus, in these patients, echocardiography for further evaluation is recommended.[1]

6. Which of these stress tests provides less prognostic information in a patient with SIHD?

A. Stress ECG
B. Stress echocardiography
C. Stress nuclear test
D. Stress magnetic resonance imaging
E. Stress positron emission tomography

Answer: E
Noninvasive stress (functional) testing to detect inducible ischemia has been

the gold standard technique to diagnose and estimate the severity of CAD in patients with suspected SIHD and to estimate prognosis in patients with known CAD.[38] These tests can provoke ischemia by using exercise or pharmacologic agents either to increase myocardial work (oxygen demand) or to induce vasodilatation-elicited heterogeneity in induced coronary flow. It is important to note that coronary artery narrowing of less than 70% is often undetected by stress testing. Noninvasive stress testing is most valuable when the pretest likelihood of CAD is intermediate. Also, it should be remembered that stress testing should be used only in patients in whom the further information delivered by the testing can alter the planned management strategy.[1] The sensitivity and specificity of various noninvasive tests to diagnose the presence of CAD are shown in Table 33.1.[18]

Table 33.1 Stress tests to diagnose and assess the prognosis in patients suspicious or known SIHD

Test	Sensitivity (%)	Specificity (%)	Provides prognostic information*	Considerations
Exercise stress test				
ECG	45–50	85–90	Yes	Easy to perform; can be used only with normal baseline ECG findings
Echocardio-graphy	80–85	80–88	Yes	Cannot be used in patients with left bundle branch block or right bundle branch block; interpretation may be limited in overweight patients
Nuclear test	73–92	63–87	Yes	Radiation exposure

Pharmacologic stress test				
Dobutamine				
Echocardiography	79–83	82–86	Yes	Limited to patients who cannot exercise; can induce arrhythmias
MRI	79–88	81–91	Yes	Limited use in overweight patients and those with metal implants; can induce arrhythmias
Adenosine				
Echocardiography	72–79	92–95	Yes	Cannot be used in patients with left bundle branch block or right bundle branch block; interpretation may be limited in overweight patients; can cause wheezing and heart block
Nuclear test	90–91	75–84	Yes	Radiation exposure; can cause wheezing and heart block
MRI	67–94	61–85	Yes	Limited use in overweight patients and those with metal implants; can cause wheezing and heart block
PET	81–97	74–91	No	Limited availability; can cause wheezing and heart block

*Most tests evaluate the risk of death, myocardial infarction, or both to assess prognosis. ECG: electrocardiogram; MRI: Magnetic resonance imaging; PET: positron emission tomography.

7. A 50-year-old man presents in your clinic with suspected SIHD since one year ago. In which condition would you not recommend a stress ECG?

A. Presence of complete left bundle branch block
B. Presence of paced ventricular rhythm
C. Presence of preexcitation syndrome
D. Presence of ST-segment depression at rest: 0.5 mm
E. Presence of LV hypertrophy based on ECG
F. Normal ECG findings in a patient who is taking digoxin.

Answer: D

Stress imaging is recommended in SIHD patients with uninterpretable ECGs (e.g., complete left bundle branch block, paced ventricular rhythm, and preexcitation syndrome), ECG changes (ST-segment depression at rest >1 mm, LV hypertrophy), or those taking digoxin.[1,19]

8. Which of these patients with SIHD has a high annual risk of death or myocardial infarction (MI)?

A. A patient with a resting left ventricle ejection fraction (LVEF) of 35% which is not readily explained by non-coronary causes.
B. A patient with resting perfusion abnormalities involving 15% of the myocardium, without a previous known history of MI.
C. A patient with a coronary artery calcium score of 350 Agatston units.
D. A patient with one-vessel CAD with 80% stenosis on his coronary computed tomography angiography (CCTA).

Answer: B

The risk stratification according to noninvasive modalities, including exercise ECG, MPI, and stress echocardiography, is set out below. According to the guidelines, patients with high-risk noninvasive test results, regardless of the severity of symptoms, have a high likelihood of CAD; therefore, coronary arteriography should be done if they have no contraindications to revascularization.[1,20]

Risk stratification according to non-invasive modalities in patients with SIHD:

High risk (>3% annual risk for death or myocardial infarction)

i. Severe resting left ventricular dysfunction (LVEF <35%) not readily explained by noncoronary causes
ii. Resting perfusion abnormalities involving ≥10% of the myocardium without previous known MI
iii. High-risk stress findings on the ECG, including:
   a. ≥2 mm ST-segment depression at low workload or persisting into recovery
   b. Exercise-induced ST-segment elevation
   c. Exercise-induced VT/VF
iv. Severe stress-induced LV dysfunction (peak exercise LVEF <45% or drop in LVEF with stress ≥10%)
v. Stress-induced perfusion abnormalities encumbering ≥10% of the myocardium or stress segmental scores indicating multiple vascular territories with abnormalities
vi. Stress-induced LV dilation
vii. Inducible wall motion abnormality (involving >2 segments or 2 coronary beds)
viii. Wall motion abnormality developing at a low dose of dobutamine (≤10 mg/kg/min) or at a low heart rate (<120 beats/min)
ix. CAC (coronary artery calcium) score >400 Agatston units
x. Multivessel obstructive coronary artery disease (CAD) (≥70% stenosis) or left main stenosis (≥50% stenosis) on CCTA

Intermediate risk (1-3% annual risk for death or myocardial infarction)

i. Mild to moderate resting LV dysfunction (LVEF of 35% to 49%) not readily explained by non-coronary causes
ii. Resting perfusion abnormalities involving 5–9.9% of the myocardium in patients without a history or previous evidence of MI
iii. ≥1 mm ST-segment depression occurring with exertional symptoms
iv. Stress-induced perfusion abnormalities encumbering 5–9.9% of the myocardium or stress segmental scores (in multiple segments) indicating one vascular territory with abnormalities but without LV dilation
v. Small wall motion abnormality involving 1–2 segments and only 1 coronary bed
vi. CAC score of 100–399 Agatston units

vii. 1-vessel CAD with ≥70% stenosis or moderate CAD stenosis (50–69% stenosis) in ≥2 arteries on CCTA

Low risk (<1% annual risk for death or myocardial infarction)

i. Low-risk treadmill score (score ≥5) or no new ST-segment changes or exercise-induced chest pain symptoms when achieving maximal levels of exercise
ii. Normal or small myocardial perfusion defect at rest or with stress encumbering ≥5% of the myocardium*
iii. Normal stress or no change in limited resting wall motion abnormalities during stress
iv. CAC score <100 Agatston units
v. No coronary stenosis >50% on CCTA

Although the published data are limited; patients with these findings will probably not be at low risk in the presence of either a high-risk treadmill score or severe resting LV dysfunction (LVEF <35%).

9. In a patient with SIHD and preserved LV function, which drug could prevent cardiovascular events and therefore reduce mortality and morbidity?

A. Aspirin
B. Clopidogrel (in case of aspirin intolerance)
C. Angiotensin-converting enzyme inhibition
D. Statins
E. Beta-blocking agents

Answer: E

Some drugs have a beneficial effect on atherosclerotic plaque stabilization and, in some cases, plaque regression, which can help to prevent cardiovascular events. Evidence has shown that in patients with SIHD and pre-served LV function, using aspirin (or clopidogrel in case of aspirin intolerance), angiotensin-converting enzyme inhibition, and statins can prevent cardiovascular events and therefore reduce mortality and morbidity. In patients with SIHD and LV dysfunction or those with a history of MI, adding beta-blocking agents reduces both mortality and the risk of a repeat MI. Guidelines recommend that optimal medical treatment includes at least one drug for angina relief plus drugs for event prevention for all patients

with SIHD. Also, it is recommended that patients should be educated about CAD, risk factors, and treatment strategy, and the patient's response should be reviewed soon after starting therapy.[1,12]

10. Which recommendation is not correct regarding risk factor modification in a patient with SIHD?

A. Dietary therapy for all patients should include reduced intake of saturated fats (to <7% of total calories), trans-fatty acids (to <1% of total calories), and cholesterol (to <200 mg/day).
B. In patients with SIHD and a BP of 140/90 mmHg or higher, antihypertensive drug therapy should be instituted in addition to or after a trial of lifestyle modifications.
C. For selected individual patients, such as those with a short duration of diabetes mellitus and a long life expectancy, a goal of HbA1c between 7% and 9% is reasonable.
D. BMI and/or waist circumference should be assessed at every visit, and clinicians should consistently encourage weight maintenance or reduction through an appropriate balance of lifestyle, physical activity, structured exercise, caloric intake, and formal behavioral programs when indicated to maintain or achieve a BMI of between 18.5 and 24.9 kg/m2 and a waist circumference of <102 cm (40 inches) in men and <88 cm (35 inches) in women (less for certain racial groups).
E. Smoking cessation and avoidance of exposure to environmental tobacco smoke at work and home should be encouraged for all patients with SIHD.
F. It is reasonable for patients with SIHD to avoid exposure to increased air pollution to reduce their risk for cardiovascular events.

Answer: C

Guidelines recommend that optimal medical treatment includes at least one drug for angina relief plus drugs for event prevention for all patients with SIHD. Also, it is recommended that patients should be educated about CAD, risk factors, and treatment strategy, and the patient's response should be reviewed soon after starting therapy.[12] Risk factor modifications in patients with SIHD are listed in Table 34.2.

Although the guidelines recommend achieving a target blood pressure of less than 140/90 mmHg in hypertensive SIHD patients,[21] recent data showed that systolic blood pressure of less than 120 mmHg and diastolic blood pressure of less than 70 mmHg were both associated with adverse cardiovascular outcomes and mortality, probably because of inadequate myocardial perfusion (J-curve phenomenon for blood pressure in patients with CAD).[22]

Patients with diabetes are more prone to atherosclerotic plaque formation and cardiovascular events.[23-25] Although there is enough evidence about control of blood glucose in the cardiovascular outcomes of patients with diabetes, the results of intensive therapy in these patients are mixed. A favorable impact of intensive diabetes therapy in patients with type 1 diabetes on the risk of cardiovascular disease has been shown at long-term follow-up;[26] however, this effect has not been shown in patients with type 2 diabetes and has even been associated with adverse outcomes in some cases.[27-29] The long-term follow-up with patients with diabetes has shown that treatment with metformin, compared with insulin or sulfonylurea, leads to a lower incidence of MI and death.[1,30]

Table 34.2 Risk factor modification in patients with SIHD

Indication	Class of recommendation	Level of evidence
Lipid Management		
Lifestyle modifications, including daily physical activity and weight management, are strongly recommended for all patients with SIHD.	I	B
Dietary therapy for all patients should include reduced intake of saturated fats (to <7% of total calories), trans fatty acids (to <1% of total calories), and cholesterol (to <200 mg/day).	I	B
In addition to therapeutic lifestyle changes, a moderate or high dose of a statin should be prescribed in the absence of contraindications or documented adverse effects.	I	A
For patients who do not tolerate statins, LDL cholesterol–lowering therapy with bile acid sequestrants, niacin, or both is reasonable.	IIa	B

Blood Pressure Management		
All patients should be counseled about the need for lifestyle modification: weight control; increased physical activity; alcohol moderation; sodium reduction; and emphasis on increased consumption of fresh fruits, vegetables, and low-fat dairy products.	I	B
In patients with SIHD and a BP of 140/90 mmHg or higher, antihypertensive drug therapy should be instituted in addition to or after a trial of lifestyle modifications.	I	A
The specific medications used for the treatment of high BP should be based on specific patient characteristics and may include ACE inhibitors and/or beta-blocking agents, as well as the addition of other drugs such as thiazide diuretics or calcium channel blocking agents if needed to achieve a goal BP of less than 140/90 mmHg.	I	B
Diabetes Management		
For selected individual patients, such as those with a short duration of diabetes mellitus and a long life expectancy, a goal hemoglobin A1c (HbA1c) of 7% or less is reasonable.	IIa	B
A goal HbA1c between 7% and 9% is reasonable for certain patients according to age, history of hypoglycemia, presence of microvascular or macrovascular complications, or presence of coexisting medical conditions.	IIa	A
Initiation of pharmacotherapy interventions to achieve a target HbA1c might be reasonable.	IIb	A
Therapy with rosiglitazone should not be initiated in patients with SIHD.	III (Harm)	C
Physical Activity		
For all patients, clinicians should encourage 30 to 60 minutes of moderate-intensity aerobic activity at least 5 days and preferably 7 days per week, supplemented by an increase in daily lifestyle activities (e.g., walking breaks at work, gardening,	I	B

household work) to improve cardiorespiratory fitness and move patients out of the least-fit, least-active, high-risk cohort (bottom 20%).		
For all patients, risk assessment with a physical activity history and/or an exercise test is recommended to guide prognosis and prescription.	I	B
Medically supervised programs (cardiac rehabilitation) and physician-directed, home-based programs are recommended for at-risk patients at first diagnosis.	I	A
It is reasonable for clinicians to recommend complementary resistance training at least 2 days per week.	IIa	C
Weight Management		
BMI and/or waist circumference should be assessed at every visit, and clinicians should consistently encourage weight maintenance or reduction through an appropriate balance of lifestyle physical activity, structured exercise, caloric intake, and formal behavioral programs when indicated to maintain or achieve a BMI of between 18.5 and 24.9 kg/m2 and a waist circumference of less than 102 cm (40 inches) in men and less than 88 cm (35 inches) in women (less for certain racial groups)	I	B
The initial goal of weight loss therapy should be to reduce body weight by approximately 5% to 10% from baseline. With success, further weight loss can be attempted if indicated.	I	C
Smoking Cessation		
Smoking cessation and avoidance of exposure to environmental tobacco smoke at work and home should be encouraged for all patients with SIHD. Follow-up, referral to special programs, and pharmacotherapy are recommended, as is a stepwise strategy for smoking cessation (Ask, Advise, Assess, Assist, Arrange, Avoid).	I	B

Management of Psychological Factors		
It is reasonable to consider screening patients with SIHD for depression and to refer or treat when indicated.	IIa	B
Treatment of depression has not been shown to improve cardiovascular disease outcomes but might be reasonable for its other clinical benefits.	IIb	C
Alcohol Consumption		
In patients with SIHD who drink alcohol, it might be reasonable for nonpregnant women to have 1 drink (4 oz of wine, 12 oz of beer, or 1 oz of spirits) a day and for men to have 1 or 2 drinks a day unless alcohol is contraindicated (such as in patients with a history of alcohol abuse or dependence or those with liver disease).	IIb	C
Exposure to Air Pollution		
It is reasonable for patients with SIHD to avoid exposure to increased air pollution to reduce their risk for cardiovascular events.	IIa	C

11. A 50-year-old man with a history of diabetes mellitus and hypertension presents with typical angina, which he has had for one year. After initiation of guideline-directed medical therapy, due to the presence of significant ST-segment depression in leads V4-6 at low workload during exercise stress ECG test, his transthoracic echocardiogram showed an LVEF of 50%. A coronary angiogram revealed 80% stenosis at the mid-part of the left anterior descending artery (LAD) and 70% in the proximal part of the dominant right coronary artery (RCA). The fractional flow reserve (FFR) showed 0.70 in the LAD lesion and 0.85 in the RCA lesion. Which therapeutic option would you recommend for this patient?

A. Medical therapy alone
B. Coronary artery bypass grafting (CABG)
C. Percutaneous coronary intervention (PCI) with new-generation drug-eluting stent in the LAD
D. Percutaneous coronary intervention (PCI) with new-generation drug-eluting stents in the LAD and RCA

Answer: C

Although PCI has a beneficial effect on reduction of the incidence of angina, it did not reduce the risk of death or MI in patients without recent ACS in some studies.[31-33] A recent meta-analysis from three randomized trials (COURAGE [Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation], Nuclear Substudy 0, FAME-2 [Fractional Flow Reserve versus Angiography for Multivessel Evaluation-2], and SWISSI II [Silent Ischemia After Myocardial Infarction II]) suggests that mortality rates may be lower with PCI plus medical therapy versus medical therapy alone in patients with SIHD and significant ischemia based on noninvasive stress imaging or abnormal FFR (≤0.80) (Fig. 34.1).[34]

A large network meta-analysis in 2014 demonstrated that CABG and new-generation drug-eluting stents (DESs) were associated with improved survival rates in patients with SIHD compared with a strategy of initial medical treatment. However, balloon angioplasty, bare-metal stents (BMS), and early-generation DESs did not have such an effect (Fig. 34.2).[35] Based on other evidence, today's new-generation DESs are the standard of care for patients undergoing PCI.[1,36-39] An algorithm for revascularization based on anatomic and clinical status in patients with SIHD is illustrated in Figure 34.3.[40,41]

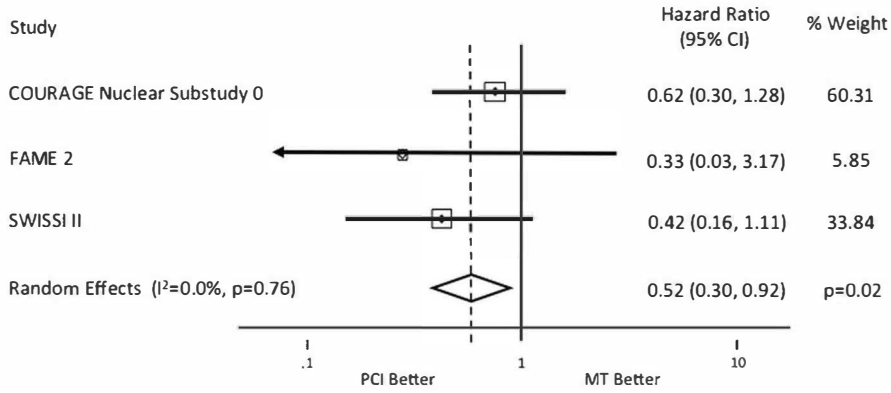
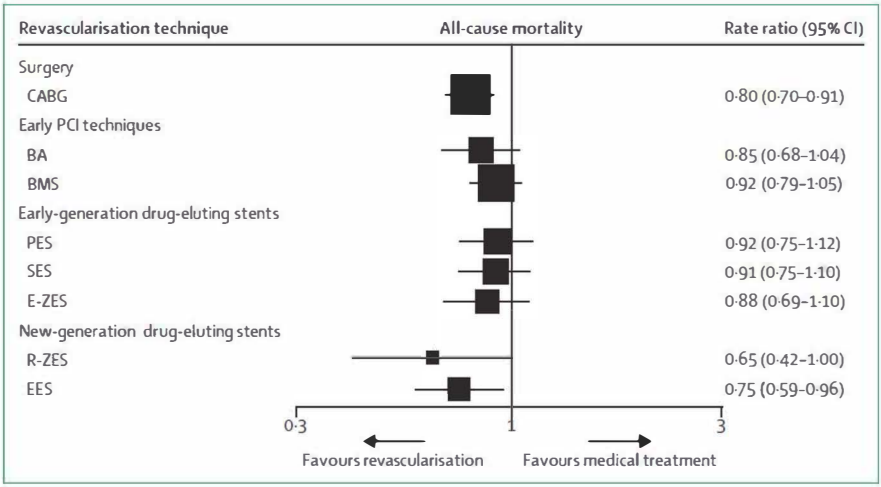
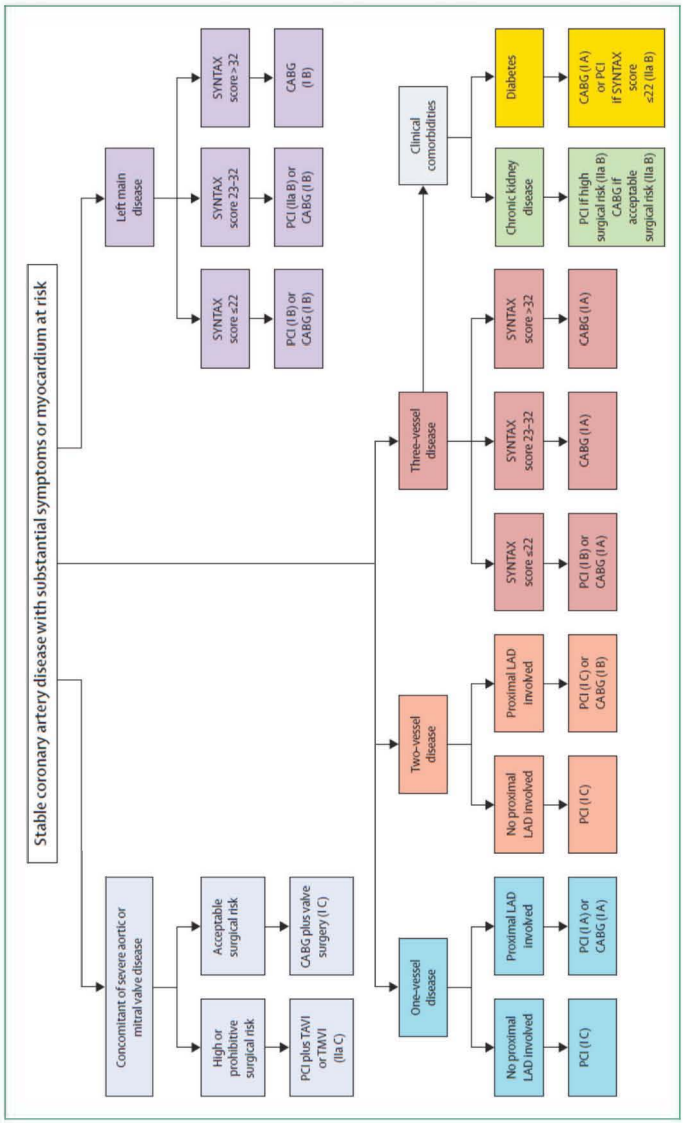


Figure 34.1 The significant mortality benefit with PCI plus medical therapy compared to medical therapy alone in patients with significant ischemia.[34]



CABG: coronary artery bypass grafting; PCI: percutaneous coronary intervention; BA: balloon angioplasty; BMS: bare-metal stents; PES: paclitaxel-eluting stent; SES: sirolimus-eluting stent; E-ZES: endeavor zotarolimus-eluting stent; R-ZES: resolute zotarolimus-eluting stent; EES: everolimus-eluting stent.

Figure **34.2** The all-cause mortality based on different revascularization techniques compared with medical treatment in patients with SIHD[41]



CABG: coronary artery bypass graft; PCI: percutaneous coronary intervention; TAVI: transcatheter aortic valve implantation; TMVI: transcatheter mitral valve; LAD = left anterior descending artery.

Figure 34.3 Algorithm for revascularization based on anatomical and clinical status in patients with SIHD[40,41]

12. Which of the following statements is not correct about patients with SIHD?

A. Compared to young men, young women, despite having a less severe angiographic CAD, have an increased risk of target vessel and target lesion failure 5 years after PCI.
B. The mortality and morbidity of older patients (≥75 years of age) undergoing revascularization are higher compared with younger patients.
C. In patients with diabetes mellitus and multivessel CAD, CABG is more beneficial in long-term clinical outcomes (death and MI) than PCI.
D. Late mortality, MI, and repeat revascularization at long-term follow-up are lower in patients with end-stage renal disease who undergo PCI than with those who undergo CABG.

Answer: D

Revascularization of special groups with SIHD:

Women: Several large studies showed that women had worse outcomes after surgical or percutaneous coronary revascularization compared with men.[42-46] A recent study showed that despite young women having a less severe angiographic CAD, they have an increased risk of target vessel and target lesion failure at 5 years after PCI,[1,46]

Older patients: The mortality and morbidity of older patients (≥75 years of age) undergoing revascularization are higher than with younger patients. This is because of a higher frequency of comorbid conditions such as peripheral vascular and cerebrovascular disease, advanced CAD, renal impairment, and LV dysfunction.[47-50] The results of previous studies that compare revascularization with medical therapy are mixed, although some studies showed that long-term outcomes of older patients were better after revascularization.[1,51-56]

Diabetes mellitus: Evidence shows that in patients with diabetes with multivessel CAD, CABG demonstrates more benefits in long-term clinical outcomes (death and MI) compared with PCI.[57-60] A major analysis of 10 randomized trials showed a worse long-term survival rate in diabetic patients after PCI (balloon angioplasty or BMS implantation) than CABG.[61] The FREEDOM (Future REvascularization Evaluation in patients with

Diabetes mellitus: Optimal management of Multivessel disease) trial compared the outcomes of CABG and PCI with DES in patients with diabetes and multivessel CAD, and found that all-cause mortality and the composite of death and MI was significantly reduced in the CABG group.[1,58]

Renal Insufficiency: Cardiovascular disease is the major cause of death in patients with end-stage renal disease. In these patients, risk factors, including diabetes, hypertension, LV systolic and diastolic dysfunction, abnormal lipid metabolism, anemia, and increased homocysteine levels, are associated with increased CAD and its complications. Also, surgical or percutaneous coronary revascularization is associated with higher complication rates.[62,63] Despite a higher early mortality rate in patients with end-stage renal disease after CABG, these patients have a lower late mortality, MI, and repeat revascularization at long-term follow-up compared with patients who undergo PCI.[1,62,64]

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CHAPTER 35

NON-ST- SEGMENT ELEVATION ACUTE CORONARY SYNDROMES

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1. During recent years, the proportion of acute coronary syndrome (ACS) cases attributed to NSTE-ACS has been increasing, while that associated with STEMI has been declining. Which of the following reasons is not correct?

A. Wider use of preventive measures such as aspirin, statins
B. Aging of the population
C. Broader use of troponin assays with higher sensitivity for myocardial necrosis
D. Increase in smoking

Answer: D

The fraction of ACS attributed to NSTEMI continues to increase, while that for STEMI is declining, for several reasons, including:

i. Wider use of preventive measures such as aspirin, statins, and cessation of smoking.
ii. Aging of the population, with greater prevalence of diabetes and chronic kidney disease, and lower rates of smoking.
iii. Broader use of troponin assays with higher sensitivity for myocardial necrosis, which shifts the diagnosis from UA to NSTE-ACS.[1,2]

2. Which of the following would not be seen in the plaque of a patient with NSTE-ACS secondary to plaque erosion?

A. Lipid rich
B. Proteoglycan and glycosaminoglycan rich
C. Few inflammatory cells
D. Endothelial cell apoptosis
E. High level of triglycerides

Answer: A

Three forms of disruption of coronary artery plaques can precipitate thrombosis: plaque rupture, plaque erosion, and disruptive nodular calcification protruding into the lumen. Plaque rupture remains the most common cause of plaque disruption, although plaque erosion has become responsible for an increasing proportion of acute coronary syndrome events (Table 35.1).[1,3] Superficial erosion highlights the characteristics of lesions associated with fatal erosion that actually contrast quite starkly with those attributed to ruptured plaques. Superficial erosion occurs more commonly in women, individuals with diabetes, and the elderly.

The decrease in prevalence of STEMI and concurrent rise in NSTE-ACS may be due to a shift in the pathology of thrombus formation towards superficial erosion versus ruptures. On the other hand, contemporary optical coherence tomography studies have shown that not only is a growing proportion of ACS due to erosion compared with fibrous cap rupture, but that that erosion associates more frequently with NSTE-ACS than STEMI.[3-5]

Table 35.1 Contrasts between superficial erosion and fibrous cap rupture as causes of arterial thrombosis[3]

Plaque rupture	**Plaque erosion**
Lipid rich	Lipid poor
Collagen poor, thin fibrous cap	Proteoglycan and glycosaminoglycan rich
Interstitial collagen breakdown	Non-fibrillar collagen breakdown
Abundant inflammation	Few inflammatory cells
Smooth muscle cell apoptosis	Endothelial cell apoptosis
Macrophage predominant	Secondary neutrophil involvement
Male predominance	Female predominance
High-, low-density lipoprotein	High triglycerides

3. In the clinical assessment of a patient with NSTE-ACS, which of the following is not correct?

A. Anginal episodes awaking the patient or occurring after meal suggest NSTE-ACS and the possibility of high-risk coronary anatomy.
B. Atypical symptoms are more common in women; older adults; and patients with diabetes, chronic renal insufficiency, or dementia.
C. Atypical symptoms do not influence the patient's prognosis.
D. In younger patients (<50 years of age), cocaine use should always be considered.

Answer: C

Patients with NSTE-ACS often present with anginal chest pain. The quality of angina is similar to stable angina (retrosternal heaviness, tightness, or frank pain) but is often more severe in intensity and lasts longer. It is very usual for the chest pain to occur at rest or after minimal exertion. Anginal episodes awaking the patient or occurring after a meal suggest NSTE-ACS and the possibility of a high-risk coronary anatomy. Atypical symptoms such as isolated dyspnea, epigastric pain, indigestion, unusual locations of pain, altered mental state, and syncope should always be taken into consideration. These atypical symptoms are more common in women; older adults; and patients with diabetes, chronic renal insufficiency, or dementia and are associated with a worse prognosis.[6,7] The presence of risk factors for coronary artery disease (CAD) increases the possibility of NSTE-ACS in a patient with acute chest pain. A history of previous symptomatic CAD, peripheral artery disease, percutaneous coronary intervention (PCI), and coronary artery bypass graft surgery (CABG) is also important. In younger patients (<50 years of age), cocaine use should always be considered.[8]

4. A 56-year-old man presents in the emergency department with an episode of acute retrosternal chest pain from three hours ago. He has a medical history of diabetes mellitus, hypertension, and chronic kidney disease. The first ECG shows diffused ST-segment depression in pericordial leads and was also positive for troponin. The patient is admitted with a diagnosis of NSTE-ACS. Which marker is less helpful for individual prognostication for risk of cardiovascular events in this patient?

A. Elevated troponin
B. Elevated BNP (brain natriuretic peptide)
C. Decreased CrCl (creatinine clearance)
D. Elevated HbA1c
E. Elevated CRP (C-reactive protein)

Answer: D

A number of biomarkers reflecting the diverse causes of NSTE-ACS are useful for prognostication. These include markers of inflammation, myocyte necrosis, acceleration of atherosclerosis, vascular damage, and hemodynamic stress (Table 35.2). For example, detection of cardiac troponin in the blood of patients with NSTE-ACS is not only indicative of myocardial necrosis, but it is also associated with the presence of intracoronary thrombus and distal embolization of platelet microaggregates.[9,10]

In some patients with ACS, elevated levels of BNP (brain natriuretic peptide) directly reflect the degree of left ventricular dysfunction secondary to acute myocardial infarction (MI) and correlate with the risk of adverse events, including death, heart failure, and MI.[1] However, in patients with an acute increase in cardiac troponin without acute MI (e.g., myocarditis), the strong association between levels of BNP and mortality indicate that the level of BNP may reflect the extent or severity of the ischemic insult, even when irreversible injury has not occurred.[11] More importantly, elevation of a baseline BNP identifies patients who are more likely to benefit from more intensive anti-ischemic and lipid-lowering regimens and early coronary revascularization.[1]

C-reactive protein (CRP) is considered a convenient test for detecting low-level systemic inflammation that portends a higher risk of developing atherothrombotic vascular disease and poorer outcomes in patients with ACS. Elevated levels of CRP at presentation identify patients who are at higher mortality risk, irrespective of whether or not there is detectable

elevation of troponin.[10] Moreover, CRP may help to identify those patients with NSTE-ACS who require more intensive management of risk factors, including lipids, glucose, blood pressure, and weight.[1]

The decrease in creatinine clearance is an indicator of the cumulative extent of vascular damage caused by a varied group of insults to the vascular endothelium (e.g., hypertension, dyslipidemia, hyperhomocystinemia, and diabetes).[12] Thus, creatinine clearance (and/or microalbuminuria) may be considered more as direct measure of the end-organ consequences of vascular risk factors than as an assessment of the individual risk factors alone.[10]

Diabetic patients have a two- to eight-fold higher risk of acute MI and a higher risk of death and recurrent ischemic events after presentation with ACS, and they also have worse outcomes after revascularization. HbA1c and blood glucose are obviously markers of this important risk factor.[10]

Multimarker approaches (e.g., simultaneous assessment of troponin, hs-CRP, and BNP) can further improve risk stratification of patients with NSTE-ACS, while measurement of other markers – such as low-density lipoprotein cholesterol, triglycerides, glucose, or HbA1c – are less helpful for individual prognostication.[1]

Table 35.2 Multi-marker approach for risk stratification in ACS[1]

Biomarker	**Independent predictor of risk**	**Useful as a component in a multi-marker strategy**	**Therapeutic implication**
Troponin	+++	++	+++
BNP	+++	++	**0**
Renal dysfunction	++	+	+
Glucose metabolism*	+	**0**	+
CRP	++	++	++

Glucose metabolism* = Hyperglycemia or elevated glycated hemoglobin (HbA1c); BNP: brain natriuretic peptide; CRP: C-reactive protein

5. Which of the following statements about the addition of contrast-enhanced coronary CTA (CCTA) to standard clinical care in patients with suspected angina is incorrect?

A. It helps to clarify the diagnosis of angina secondary to coronary heart disease.
B. It reduces the need for stress testing.
C. It reduces the use of invasive coronary angiography.
D. In centers that can offer high-sensitivity troponin assays, the benefit of CCTA is less clear.

Answer: C

A large multicenter randomized trial comparing standard of care with versus without CCTA in 4,146 patients with suspected angina demonstrated that CCTA helped clarified the diagnosis of angina secondary to coronary heart disease, reduced the need for stress testing, but increased the use of invasive coronary angiography. They found that the addition of CCTA is associated with an apparent reduction in fatal and non-fatal myocardial infarction.[13] A recent randomized multicenter study showed that CCTA, applied early in the work-up of suspected ACS, is safe and associated with less outpatient testing and lowers costs. However, in the era of high-sensitivity troponin assays, CCTA does not identify more patients with significant CAD requiring coronary revascularization, shorten hospital stays, or allow for more direct discharge from the emergency department.[14]

6. A 50-year-old woman presents in the emergency department with chest pain and is suspected of having NSTE-ACS. Which of the following is an appropriate indication for CCTA in this patient?

A. TIMI (thrombolysis in myocardial infarction) score of 5
B. HEART score of 4
C. Atrial fibrillation
D. BMI >39 kg/m^2
E. Equivocal previous functional testing within the previous six months

Answer: E

Coronary CTA in patients with or suspected of having NSTE-ACS can help recognize or exclude the presence of coronary artery disease, identify which

vessels have coronary arthrosclerosis, and assist in risk stratification and prognosis. Previous studies have shown that a negative CCTA (defined as maximal stenosis <50% in all vessels) predicts freedom from 30-day myocardial infarction, coronary revascularization, and cardiovascular death, and that this strategy, compared with a standard evaluation, can lead to a reduction in costs and shortens the length of hospital stays.[15-18] Moreover, a negative coronary CTA is associated with a very low event rate in the longer term. The indications for a coronary CTA in patients with ACS are shown in Table 35.3.[19]

Coronary CTA may not suitable for all low- to intermediate-risk patients who present at the emergency department. Up to one-third may not qualify for imaging because of contrast allergy, renal insufficiency, unresolved tachycardia, or inability to receive a beta-blockade for heart rate control. The ideal patient is not known to be free of disease or to definitely have an underlying coronary disease, but will be considered low- to intermediate-risk without significant contraindications (Table 35.3). Patients with a low (TIMI score of 0–2) or intermediate (3–4) risk, or those with HEART score less than 3 (especially those have had a negative stress test in the past 6 to 12 months) can benefit more from a coronary CTA.[19]

The HEART Score: Classical considerations for risk stratification are History, ECG, Age, Risk factors and Troponin. Each can be scored with zero, one or two points, depending on the extent of the abnormality. The HEART score is the sum of these five considerations (between 0 and 10).

Table 35.3 Appropriate Indications for Coronary CTA in Patients with Acute Chest Pain Syndromes[19]

Appropriate Indications
ECG negative or indeterminate for myocardial ischemia
Low to intermediate pretest likelihood by risk stratification tools
TIMI score of 0–2 (low risk) ideal or TIMI score of 3–4 (intermediate) in some cases
HEART score <3
≥1 Negative troponin values, including point-of-care assays
Equivocal or inadequate previous functional testing during index ED or within previous 6 months
Equivocal indications
High clinical likelihood of ACS by clinical assessment and standard risk criteria (e.g., TIMI score >4)
Previously known CAD
Known calcium score >400
Relative contraindications
History of allergic reaction to iodinated contrast
GFR <60 ml/min/1.73 m^2
Factors likely to lead to nondiagnostic scans; specifics will vary with scanner technology and site capabilities
Heart rate greater than site maximum for reliably diagnostic scans after beta-blockers (usually 70–80 bpm)
Contraindications to beta-blockers and heart rate not controlled
Atrial fibrillation or other markedly irregular rhythm
BMI >39 kg/m^2
Absolute contraindications
Known ACS
GFR <30 ml/min/1.73 m^2 unless on long-term dialysis
Previous anaphylaxis after iodinated contrast administration
Previous episode of contrast allergy after adequate steroid/antihistamine preparation
Pregnancy

ACS, acute coronary syndrome; BMI, body mass index; CAD, coronary artery disease; CTA, computed tomography angiography; ED, emergency department; GFR, glomerular filtration rate; and TIMI, Thrombolysis in Myocardial Infarction.

7. A 70-year-old man has presented in the emergency department with NSTE-ACS. The patient is smoker and his medical history is positive for hypertension, diabetes, and peripheral artery disease. His echocardiogram shows a significant reduction in left ventricular ejection fraction (LVEF: 30%) and the laboratory examination shows a reduced estimated glomerular filtration rate (eGFR: 50 ml/min/1.73m^2). Which of the following has the least effect on long-term prognosis (composite of cardiovascular death, myocardial infarction, or ischemic stroke) in this patient?

A. Age
B. Current smoker
C. Hypertension
D. Diabetes
E. Peripheral artery disease
F. LVEF: 30%
G. eGFR: 50 ml/min/1.73m

Answer: A

Several risk scores that integrate clinical variables and findings on the clinical, ECG, and serum cardiac marker findings have been developed for patients with NSTE-ACS. The TIMI (Thrombolysis In Myocardial Ischemia) risk score for evaluating the relation between seven independent risk factors and short-term outcomes (death, myocardial infarction, or urgent revascularization) is shown in Figure 35.1.[20] This score provides a simple and rapid assessment for detecting high-risk patients who can derive benefit from an early invasive strategy and more intensive antithrombotic therapy. Also, it can predict the severity of angiographic findings such as CAD extension, thrombus burden, and flow impairment.[21,22]

It has been demonstrated that a combination of nine risk factors as a TIMI stable ischemic CAD risk score is a good predictor of long-term prognosis for recurrent atherothrombotic events in patients after ACS (Figure 35.2).[23] Studies have also shown that patients with higher scores benefit from more intensive antithrombotic and lipid-lowering therapies.[23, 24]

Rates of all-cause mortality, myocardial infarction, and severe recurrent ischemia prompting urgent revascularization through 14 days after randomization were calculated for patients with UA/NSTE-ACS based on the number of risk factors. Independent risk factors include age ≥ 65 yrs, ≥ 3 CAD risk factors, known CAD (>50% stenosis), prior aspirin use, ≥ 2

anginal episodes in prior 24 hours, ST-segment deviation ≥ 0.5 mm of initial ECG, and increase in cardiac biomarkers.

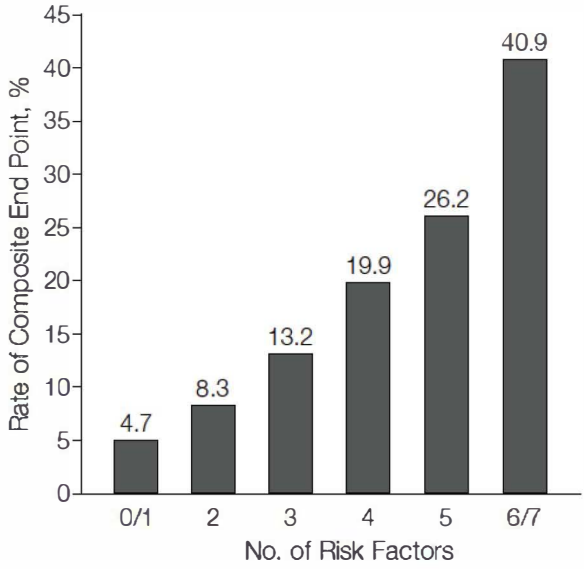
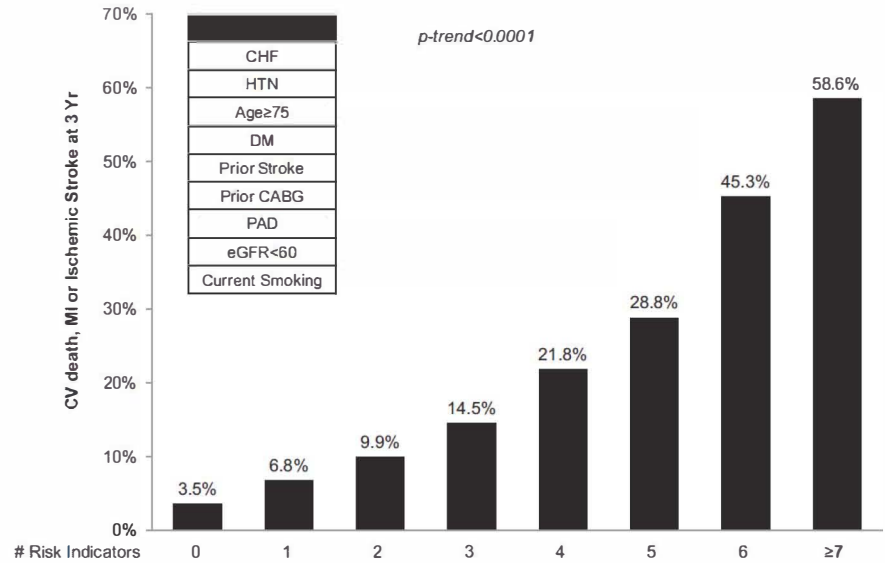


Figure 35.1 TIMI risk score for UA/NSTE-ACS[20]



CABG: coronary artery bypass graft; CHF: congestive heart failure; DM: diabetes mellitus; eGFR: estimated glomerular filtration rate; HTN: hypertension; PAD: peripheral artery disease.

Figure 35.2 Long term stratification after myocardial infarction using TIMI stable ischemic coronary artery disease risk score[23]

8. Which of these anti-ischemic agents can lead to a decrease in mortality in a patient with NSTE-ACS?

A. Metoprolol Succinate
B. Nitroglycerin
C. Verapamil
D. Ranolazine
E. Nicorandil

Answer: A

Guidelines recommend that early use of anti-ischemic therapies by improvement of the balance between oxygen supply and demand can lead to relief of symptoms, prevention of recurrent ischemic events, heart failure, arrhythmia, and in some cases a decrease in mortality (Table 35.4).[25]

Table 35.4 Pharmacologic anti-ischemic agents in NSTE-ACS[25]

Class of Medication	Mechanism of Action	Clinical Effects in NSTE-ACS
Traditional Agents		
Beta blockers	Decrease heart rate, blood pressure, and contractility through antagonism of β1 receptors	Decrease mortality Long-term secondary prevention benefit is less certain in patients without heart failure
Nitrates	Decrease preload through venodilation; vasodilate coronary arteries	No benefit on mortality
Calcium channel blockers	May vasodilate, reduce heart rate, or decrease contractility depending on specific drug	No clear benefit on mortality or reinfarction Increased reinfarction rate when nifedipine is used alone
Newer and Experimental Agents		
Ranolazine	Inhibits late inward sodium current	Decreases recurrent ischemia and arrhythmias
Trimetazidine	Shifts myocardial metabolism from fatty acid to glucose use	Decreases mortality in subset of patients not receiving thrombolysis
Nicorandil	Activates ATP-sensitive K+ channels and dilates arterioles; may have ischemic preconditioning-like effect	Decreases arrhythmias and transient ischemia. No effect on infarct size or ejection fraction
Cyclosporine	Inhibitor of the mitochondrial permeability transition pore involved in reperfusion injury	Reduces infarct size in small studies. Larger clinical trial in progress

9. In which circumstances could nitrates could be used safely in a patient with NSTE-ACS?

A. Use of vardenafil in 28 hours ago
B. Systolic blood pressure = 80 mmHg
C. Severe obstruction to LV outflow
D. Large right ventricular infarction
E. Hemodynamically significant pulmonary embolism

Answer: A

Important contraindications to the use of nitrates include hypotension and recent use of a phosphodiesterase type-5 (PDE-5) inhibitor, sildenafil or vardenafil (within 24 hours), or tadalafil (within 48 hours). Since the catalytic site of PDE-5 normally degrades cyclic guanosine monophosphate, inhibitors of PDE-5 potentiate endogenous levels of cGMP, possibly resulting in exaggerated, prolonged, and dangerous vasodilatory effects of nitrates. Relative contraindications to nitrates include hypotension (SBP <90 mmHg), severe obstruction to LV outflow, a large right ventricular infarction, or a hemodynamically significant pulmonary embolism. In such patients, nitrates should be used with caution.[1]

10. A 65-year-old man is admitted to the emergency department with a diagnosis of NSTE-ACS. Which of the following treatments is not recommended in this patient?

A. If the patient is unable to take aspirin because of hypersensitivity or major gastrointestinal intolerance, a loading dose of clopidogrel 600 mg followed by a daily maintenance dose of 75 mg should be substituted.
B. Ticagrelor may be preferred over clopidogrel as the initial treatment.
C. In patients treated with ticagrelor, the preferred aspirin maintenance dose is 162 mg/day.
D. Use prasugrel only in patients receiving coronary stents.

Answer: C

Table 35.5 2014 Guideline recommendations for antiplatelet agents in patients with NSTE-ACS[1,38]

Non-enteric–coated, chewable aspirin (162 to 325 mg) should be given to all patients without contraindications on presentation, and a maintenance dose of aspirin (81 to 325 mg/day) continued indefinitely.
In patients who are unable to take aspirin because of hypersensitivity or major gastrointestinal intolerance, a loading dose of clopidogrel (300 or 600 mg) followed by a daily maintenance dose of 75 mg should be substituted.
Either clopidogrel or ticagrelor can be used initially with either an early invasive or ischemic guided strategy (COR I, LOE: B).
Ticagrelor may be preferred over clopidogrel as the initial treatment (COR IIa, LOE: B).
In patients treated with ticagrelor, the preferred aspirin maintenance dose is 81 mg/day.
Use prasugrel only in patients receiving coronary stents (COR I, LOE: B).
The use of glycoprotein IIb/IIIa receptor inhibitors is reserved mainly to instances of PCI in high-risk patients who were not adequately pretreated with P2Y12 inhibitors (COR I, LOE: A) or in those patients who were adequately pretreated with P2Y12 inhibitors but have a high-risk profile (COR IIa, LOE: B).
Clopidogrel and ticagrelor should be discontinued at least 5 days (COR I, LOE: B) and prasugrel at least 7 days (COR I, LOE: C) before major surgery.

COR, Class of recommendation; LOE, level of evidence; PCI, percutaneous coronary intervention.

11. Which of the following statements about antiplatelet agents in a patient with NSTE-ACS is not correct?

A. Omeprazole might reduce the antiplatelet effects of clopidogrel.
B. Prasugrel should be considered in those who present with stent thrombosis while receiving clopidogrel.
C. Prasugrel is contraindicated in patients with a low body weight (<65 kg) and older individuals (>75 years).
D. The risk of bleeding complications is unacceptable with prasugrel in case the patient needs an urgent CABG.
E. Ticagrelor is more often associated with increased rates of minor and non-CABG–related bleeding than clopidogrel.
F. Asymptomatic episodes of ventricular pauses have been reported with ticagrelor.

Answer: C.

Clopidogrel: Thienopyridine clopidogrel (300–600 mg loading dose; 75 mg/day as maintenance) is a prodrug, and for it to change to an active metabolite it needs to be oxidized by the hepatic cytochrome P-450 system. The active metabolite irreversibly inhibits the binding of adenosine diphosphate (ADP) to the P2Y12 receptor and, eventually, suppresses platelet activation and aggregation (26). Proton pump inhibitors (PPIs), especially omeprazole, modestly reduce the antiplatelet effect of clopidogrel when assessed by platelet function assays because of competition for metabolism by the CYP3A4 enzyme; however, based on previous studies, a randomized a clinically significant interaction between clopidogrel and PPIs is unlikely.[8,27,28]

Prasugrel: Prasugrel (60 mg loading dose; 10 mg/day as maintenance) is a thienopyridine prodrug requiring hepatic oxidation to form an active metabolite that irreversibly inhibits the platelet P2Y12 receptor. Prasugrel is oxidized very fast, inhibits platelet aggregation almost completely (it is 10 times more potent than clopidogrel), and starts to have an effect after 30 minutes of the loading dose (compared to clopidogrel, which needs 2–6 hours for the effects to start). It has been shown that prasugrel is associated with a lower rate of MI than clopidogrel in NSTE-ACS patients undergoing PCI, especially in patients with diabetes.[29,30] It is also associated with lower rates of stent thrombosis and should be considered in high-risk patients for stent thrombosis or those who present with stent thrombosis despite compliance with clopidogrel therapy.[31] Because of the higher potency, the rate of severe bleeding events, including non-CABG major, spontaneous, and fatal bleeding, are increased by this drug. It should be used with great caution (because of the higher bleeding risk) in patients with low body weight (<65 kg) and older individuals (>75 years) and is contraindicated in patients with a history of stroke or transient ischemic attacks.[29] The risk of bleeding complications is unacceptable with prasugrel in case the patient needs an urgent CABG. Therefore, it is recommended to initiate prasugrel while the patient is on the table, only after the coronary anatomy is clarified and the PCI is planned. For the same reason, patients who initially managed with ischemic-guided medical therapy should receive a P2Y12 inhibitor other than prasugrel. Based on guidelines, if the patient is finally subjected to a PCI, prasugrel can be started over clopidogrel.[8]

Ticagrelor: Ticagrelor (180 mg loading dose; 90 mg twice daily as maintenance) is a nonthienopyridine and reversible P2Y12 inhibitor agent which directly blocks the P2Y12 receptor with a half-life of approximately 12 hours. Ticagrelor reduces the rates of cardiovascular death, MI, stroke,

and stent thrombosis compared with clopidogrel in patients with NSTE-ACS.[32,33] It has been associated with increased rates of minor and non-CABG–related bleeding compared with clopidogrel. Dyspnea without bronchospasm, episodes of ventricular pauses in sinus rates in the first week lasting longer than three seconds, and rises in serum uric acid have been reported with ticagrelor.[34] Compliance to a ticagrelor regime might be suboptimal because its shorter half-life and the need for twice-daily administration reduces adherence among patients.[8]

Based on recent ESC/EACTS guidelines for patients with NSTE-ACS, dual antiplatelet therapy that includes aspirin and a potent P2Y12 receptor inhibitor (prasugrel or ticagrelor) is recommended. Clopidogrel should only be used when prasugrel or ticagrelor are not available or are contraindicated. However, it is not recommended that prasugrel is administered in patients whose coronary anatomy is not known. On the other hand, pretreatment with ticagrelor is associated with early benefits over clopidogrel.[35]

12. A 65-year-old woman admitted is admitted to the emergency department with a diagnosis of NSTE-ACS. Her past medical history is positive for hypertension and diabetes. A physical exam shows BP = 120/80 mmHg, HR = 70 bpm, and SO2 = 93% on room air. The electrocardiogram shows a 2 mm ST-segment depression in anterior leads and a slight increase in serum cardiac troponin levels. Her echocardiogram shows LVEF = 45%, without significant valvular heart disease. After nitrate administration, the chest pain is relieved and the ST-segment returns to the isoelectric baseline. Which therapeutic strategy is the best in this patient?

A. Immediate or urgent invasive strategy
B. Early invasive strategy
C. Delayed invasive strategy
D. Ischemic-guided strategy
E. Conservative strategy

Answer: B

Patients with NSTE-ACS who are intermediate- to high-risk should always be considered for an invasive strategy. Although the appropriate timing of the invasive strategy is a matter of controversy, it seems that an early invasive approach (within 24 hours of admission) reduces recurrent ischemia and hospital stay length (but not death and MI) compared to a delayed invasive strategy, and should be considered in higher-risk patients.

Various therapeutic strategies for patients with NSTE-ACS are summarized in Table 35.6.

Table 35.6 Therapeutic Strategies in Patients with Non-ST–Segment Elevation Acute Coronary Syndrome[8, 35]

	Immediate or Urgent Invasive Strategy	**Early Invasive Strategy**	**Delayed Invasive Strategy**	**Ischemic-Guided or Conservative Strategy**
Time from admission	≤ 2 hrs	≥ 24 hrs	25-72 hr	-
Criteria	• Hemodynamic instability or Cardiogenic shock • Refractory/ ongoing angina refractory to medical treatment • Acute heart failure • New or worsening MR • Mechanical complication of MI • Electrical instability	• GRACE risk score >140 • Elevated cTn levels • Dynamic ST-T changes (symptomatic or silent)	• Diabetes • Renal insufficiency • LVEF <40% • Post-MI angina • Prior PCI/CABG • Prior CABG GRACE risk score of 109–140 • Recurrent symptoms/ischemia on non-invasive testing	• No high-risk criteria • Contra-indication to invasive therapy

CABG: Coronary artery bypass grafting; GRACE: Global Registry of Acute Coronary Events; LVEF: Left ventricular ejection fraction; MI: Myocardial infarction; PCI: Percutaneous coronary intervention.

13. A 75-year-old man presents in the ED with at-rest chest pain and dyspnea NYHA functional class III from six hours ago. The patient was admitted to another center two weeks ago with a diagnosis of NSTE-ACS and discharged with medical treatment. His past medical history is positive for diabetes and CABG (10 years ago). His electrocardiogram shows a 3 mm ST-segment depression in precordial leads. 15 minutes after admission, ventricular tachycardia occurs, which is treated with electrical cardioversion. His echocardiogram reveals a significantly reduced LVEF (35%) without significant valvular heart disease or mechanical complication. After initiation of anti-ischemic agents, the symptoms are relieved and the ST-segment returns to the isoelectric baseline. Laboratory examinations show a significant rise in serum cardiac troponin levels and a decrease in GFR (80 ml/min/1.73 m^2). Which of the following poses the most potent risk of adverse events in this patient?

A. The symptoms
B. ST-segment depression
C. Ventricular tachycardia
D. Diabetes mellitus
E. Early post-infarction angina
F. Prior CABG
G. Rise in serum cardiac troponin level
H. Decrease in GFR (80 ml/min/1.73 m^2)

Answer: C

On presentation, patients who are very high risk should be referred for an immediate invasive coronary angiogram (<2 hr) with intent to perform revascularization (Table 35.7). Centers without a catheterization laboratory or STEMI programs should immediately transfer the patient in to PCI-capable center (36). Selection of NSTE-ACS treatment strategy and timing according to initial risk stratification is shown in Figure 35.3.

Table 35.7. Risk criteria in patients with NSTE-ACS[36]

Very high-risk criteria
Hemodynamic instability or cardiogenic shock Recurrent or ongoing chest pain refractory to medical treatment Life-threatening arrhythmias or cardiac arrest Mechanical complications of MI Acute heart failure Recurrent dynamic ST-T wave changes, particularly with intermittent ST-elevation High-risk criteria
High-risk criteria
Rise or fall in cardiac troponin compatible with MI Dynamic ST- or T-wave changes (symptomatic or silent) GRACE score >140 Intermediate-risk criteria
Intermediate-risk criteria
Diabetes mellitus Renal insufficiency (eGFR < 60 ml/min/1.73 m2) LVEF < 40% or congestive heart failure Early post-infarction angina Prior PCI Prior CABG GRACE risk score >109 and <140
Low-risk criteria
Any characteristics not mentioned above

CABG: coronary artery bypass graft; eGFR: estimated glomerular filtration rate; GRACE: Global Registry of Acute Coronary Events; LVEF: left ventricular ejection fraction; PCI: percutaneous coronary intervention; MI: myocardial infarction.

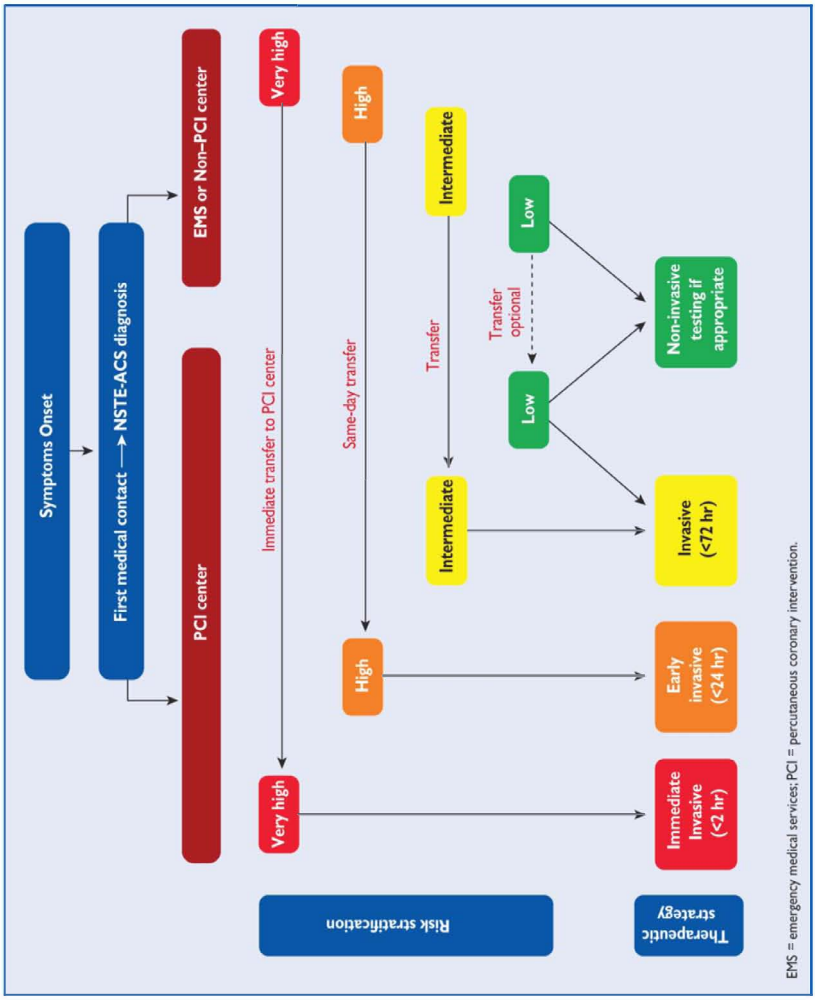


Figure 35.3 Selection of NSTE-ACS treatment strategy and timing according to initial risk stratification[36]

14. A 78-year-old man presents at the emergency department with a diagnosis of NSTE-ACS. The patient is treated with warfarin because of non-valvular atrial fibrillation. He has a medical history of uncontrolled hypertension, diabetes, renal impairment (eGFR: 40 ml/min/1.73 m^2), and ischemic stroke (one year ago). A PCI with DES was done for the patient and discharged after five days of hospitalization. Which strategy is the best for this patient?

A. Triple therapy with warfarin (INR: 2.0–2.5) + aspirin 81 mg/day + clopidogrel 75 mg/day for 4 weeks and then warfarin and clopidogrel 75 mg/day up to 12 months.
B. Triple therapy with warfarin (INR: 2.0–2.5) + aspirin 81 mg/day + clopidogrel 75 mg/day for 3 months and then warfarin and clopidogrel 75 mg/day up to 12 months.
C. Triple therapy with warfarin (INR: 2.0–2.5) + aspirin 81 mg/day + clopidogrel 75 mg/day for 6 months and then warfarin and clopidogrel 75 mg/day up to 12 months.
D. Triple therapy with warfarin (INR: 2.0–3.0) + aspirin 81 mg/day + clopidogrel 75 mg/day for 4 weeks and then warfarin and clopidogrel 75 mg/day up to 12 months.

Answer: A

Consensus recommendations on the management of patients with atrial fibrillation and NSTE-ACS undergoing PCI with stenting are summarized in Table 35.8.[37] Triple therapy may be considered up to 12 months in very select patients at high risk of ischemic events (e.g. prior stent thrombosis on adequate antiplatelet therapy, stenting in the left main or last remaining patent coronary artery, multiple stenting in proximal coronary segments, two stent bifurcation treatments, or diffuse multivessel disease, especially in diabetic patients).[1] Table 35.9 shows the CHA_2DS_2-VASC and HAS-BLED scores.

Table 35.8 Recommended antithrombotic strategies following coronary artery stenting in patients with NSTE-ACS and atrial fibrillation (in whom oral anticoagulation therapy is required)[37]

Hemorrhagic risk	Stroke risk	Recommendations
Low or moderate (HAS-BLED 0–2)	Moderate (CHA_2DS_2-VASC = 1 in males)	6 months: triple therapy of OAC + aspirin 75–100 mg/day + clopidogrel 75 mg/day Up to 12th month: OAC and clopidogrel 75 mg/day (or alternatively, aspirin 75–100 mg/day) Lifelong: OAC[a]
	High (CHA_2DS_2-VASC ≥2)	6 months: triple therapy of OAC + aspirin 75–100 mg/ day + clopidogrel 75 mg/day Up to 12th month: OAC and clopidogrel 75 mg/day (or alternatively, aspirin 75–100 mg/day) Lifelong: OAC[a]
High (HAS-BLED ≥3)	Moderate (CHA_2DS_2-VASC = 1 in males)	4 weeks: triple therapy of OAC + aspirin 75–100 mg/day + clopidogrel 75 mg/day[b] Up to 12th month: OAC and clopidogrel 75 mg/day (or alternatively, aspirin 75–100 mg/day) Lifelong: OAC[a]
	High (CHA_2DS_2-VASC ≥2)	4 weeks: triple therapy of OAC + aspirin 75–100 mg/day + clopidogrel 75 mg/day[b] Up to 12th month: OAC and clopidogrel 75 mg/day (or alternatively, aspirin 75–100 mg/day) Lifelong: OAC[a]

PPIs should be considered in all patients, particularly where aspirin is used. Newer generation drug-eluting stents should be preferred over bare metal stents in patients at low risk for bleeding. New generation drug-eluting stents are generally preferable to bare-metal stents, particularly in patients at low bleeding risk (HAS-BLED 0–2). OAC, oral anticoagulation, either warfarin (INR: 2.0–2.5) or non-VKA oral anticoagulant at the lower tested dose in AF (dabigatran 110 mg b.i.d., rivaroxaban 15 mg o.d. or apixaban 2.5 mg b.i.d.). INR: international normalized ratio; PPIs: proton pump inhibitors.

[a] Alone or combined with single antiplatelet therapy only in very selected cases (e.g. stenting of the left main, proximal bifurcation, recurrent MIs, etc).

[b] Combination of OAC and clopidogrel 75 mg/day may be considered as an alternative.

Table 35.9 CHA_2DS_2-VASC and HAS-BLED scores

CHA_2DS_2-VASC	Score	**HAS-BLED**	Score
Congestive heart failure (or Left ventricular systolic dysfunction)	1	**H**ypertension i.e. uncontrolled blood pressure	1
Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)	1	**A**bnormal renal/liver function	1 or 2
Age ≥75 years	2	**S**troke	1
Diabetes Mellitus	1	**B**leeding tendency or predisposition	1
Stroke or TIA or thromboembolism	2	**L**abile INR	1
Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)	1	**E**lderly (age > 65)	1
Age 65–74 years	1	**D**rug (e.g. concomitant aspirin or NSAIDs) or alcohol	1
Sex **c**ategory (i.e. female sex)	1		

15. A 45-year-old diabetic man with NSTE-ACS underwent a percutaneous coronary intervention (PCI) on the left anterior descending artery (LAD) with a drug-eluting stent. His echocardiogram before discharge showed LVEF = 30% with mild mitral regurgitation. Which strategy for long-term treatment of this patient after discharge is not mandatory?

A. Dual antiplatelet therapy (DAPT) for at least 12 months
B. Beta blocker
C. Angiotensin-converting enzyme inhibitors
D. Aldosterone blockers
E. High-dose statin therapy with therapeutic goal of low-density lipoprotein level less than 70 mg/dl
F. Proton pump inhibitors (PPIs) during DAPT therapy

Answer: F

Patients with NSTE-ACS should receive DAPT for at least 12 months after the index event, regardless of whether they have been managed medically alone, with PCI by drug-eluting stent (DES) or bare-metal stent (BMS), or with CABG. Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers should be started during hospitalization and continued in patients with LV dysfunction, diabetes, hypertension, and stable chronic kidney disease. Aldosterone blockers are indicated in post-MI patients with LV dysfunction, diabetes, or heart failure if not contraindicated.[38] High-dose statin therapy is indicated to be started early after presentation, preferably before PCI, and should be continued after discharge. The therapeutic goal is a low-density lipoprotein level less than 70 mg/dl, but lower levels are not associated with adverse effects, and statin therapy should not be stopped or titrated downward if the patient has tolerated high-dose statins.[39,40] PPIs only need to be administered in patients receiving DAPT who have a history of gastrointestinal bleeding or are at high risk for gastrointestinal bleeding.[8]

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CHAPTER 36

ANESTHESIA AND SEDATION IN CARDIAC PATIENTS

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1. Which laboratory test is useful to evaluate patients who have taken clopidogrel 3-5 days before cardiac surgery?

A. Prothrombin time
B. Platelet aggregometry
C. Partial thromboplastin time
D. Platelet count

Answer: B

Platelet aggregometry is the best test to evaluate platelets function. PT and PTT assess coagulation pathway performance.

2. According to the European system for cardiac operative risk evaluation (Euro SCORE II), which preoperative condition imposes a higher operative risk for a patient?

A. Post infarct ventricular septal rupture
B. Recent myocardial infarct within 90 days
C. Emergency operation
D. Systolic pulmonary pressure >60 mmHg

Answer: A

According to Euro SCORE II, a post-infarct ventricular septal rupture is a serious complication and receives 4 points. A recent MI, pulmonary hypertension, and an emergency operation each get 2 points.

Table 36.1 EuroSCORE: The European System for Cardiac Operative Risk Evaluation Additive Risk Stratification Model[3]

Factor		Points	β
Age	Per 5 yrs or part there of more than 60 yrs	1	0.0666354
Sex	Female	1	0.3304052
Chronic pulmonary diseases	Long-term use of bronchodilators or steroids for lung disease	1	0.4931341
Extracardiac arteriopathy	Any one or more of the following: claudication, carotid occlusion or >50% stenosis, previous or planned intervention on the abdominal aorta, limb arteries, or carotids	2	0.6558917
Neurologic dysfunction	Severely affecting ambulation or day-to-day functioning	2	0.841626
Previous cardiac surgery	Requiring opening of the pericardium	3	1.002625
Serum creatinine	>200 μmol/L^{-1} before operation	2	0.6521653
Active endocarditis	Patient still under antibiotic treatment for endocarditis at the time of surgery	3	1.101265
Critical preoperative state	Any one or more of the following: VT or VF aborted sudden death, preoperative cardiac massage, preoperative ventilation before arrival in the anesthetic room, preoperative inotropic support, IABP, or preoperative actual renal failure (anuria or oliguria<10 mL/hr^{-1}	2	0.9058132
Unstable angina	Rest angina requiring IV nitrates until arrival in the anesthetic room	2	0.5677075
LV dysfunction	Moderate (LVEF 30%-50%)	1	0.4191643
	Poor (LVEF <30%)	3	1.094443

Recent myocardial infarct	Within 90 days	2	0.5460218
Pulmonary hypertension	Systolic pulmonary artery pressure >60 mm Hg	2	0.7676924
Emergency operation	Carried out on referral before the beginning of the next working day	2	0.7127953
Other than isolated CABG	Major cardiac procedure other than or in addition to CABG	2	0-5420364
Surgery on thoracic aorta	For disorder of ascending arch or descending arch	3	1.159787
Postinfarct septal rupture		4	1.462009

CABG, Coronary artery bypass graft; IABP, intraaortic balloon pump; LV, left ventricular; LVEF, left ventricular ejection fraction; VF, ventricular fibrillation; VT, ventricular tachycardia.

3. Which drug group represents the core component of cardiac anesthesia?

A. Benzodiazepines
B. Non-depolarizing muscle relaxants
C. Volatile anesthetics
D. Opioids

Answer: D

The main goal of cardiac anesthesia is to provide balanced anesthesia whilst preserving myocardial function and cardiac output and maintaining sufficient consciousness, pain sensation and neuro-muscular suppression. Opioids are the core components in achieving this goal.

4. Which sedation-analgesic agent should not be used by non-anesthesiologists?

A. Midazolam
B. Morphine
C. Propofol
D. Fentanyl

Answer: C

The frequency of respiratory depression with the administration of propofol is high, and apnea is common with bolus doses, thus the use of this drug must be avoided by those who are unfamiliar with airway management (in general non-anesthesiologists).

5. A 76-year-old female (body weight = 51 kg) was prepared for a TEE exam. After receiving 10 puffs of lidocaine 10%, IV 25 μg fentanyl and 1 mg midazolam was administered. Due to PVCs in her ECG the patient received 100 mg IV lidocaine. After two minutes, she lost consciousness and experienced pallor with BP = 95/56 mmHg HR = 103 /min. What is the most probable cause of this situation?

A. Opioid respiratory depression
B. Lidocaine toxicity
C. Deep sedation due to simultaneous sue of midazolam and fentanyl
D. Cardiogenic shock

Answers: B

The maximum allowable dose of lidocaine is 4.5 mg/kg or 300 mg in normal healthy adults. Each puff of 10% lidocaine spray contains 10 mg lidocaine. Fentanyl 25 mg and midazolam 1 mg lead to mild sedation and are not the cause of loss of consciousness. Thus, lidocaine toxicity is the most probable cause of unconsciousness in this thin, elderly patient.

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CHAPTER 37

PRINCIPLES OF CARDIOVASCULAR SURGERY

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1. Deep hypothermic circulatory arrest includes all the following characteristics except:

A. The patient's body temperature cools down to about 18°C
B. it is usually required in pulmonary thromboendarterectomy
C. It provides a bloodless surgical field and facilitates aortic arch surgery
D. It is mainly used for better myocardial protection during cardiac surgery.

Answer: D

Total circulatory arrest provides a bloodless surgical field in a deep hypothermic state that is mandatory for reconstructive aortic arch surgery. Deep hypothermia (18 °C) provides a longer safe period for stopping the patient's circulation and performing complex aortic arch aneurysm or dissection surgeries. It is also very useful for precise pulmonary thromboendarterectomy in CTEPH patients.

2. Regarding postoperative anticoagulation, all the sentences below are correct except:

A. Mitral valve repair in a patient with normal sinus rhythm needs oral anticoagulation for only 6-8 weeks.
B. Mitral valve replacement with a new-generation mechanical valve needs lifelong oral anticoagulation with an INR therapeutic range of 2.5-3.5.
C. David operation (valve-preserving root replacement) in a patient with NSR needs oral anticoagulation for only 6 months.
D. Pulmonary valve replacement with a new-generation mechanical valve needs lifelong oral anticoagulation with an INR therapeutic range of 3-4.

Answer: C

Antiplatelet medication is usually sufficient for patients who have undergone aortic root replacement with valve-preserving methods such as David or Yacoub techniques.

3. A 57-year-old man with three-vessel coronary artery disease with good coronary anatomy and normal LV function receives four CABGs (coronary artery bypass grafts). Four hours later, his hemodynamics got become unstable, despite the infusion of epinephrine (0.1 micro/kg/min). The patient's vital signs are HR = 102/min, BP = 75/38 mmHg, CVP = 11 mmHg. Arterial blood gas shows pH 7.23, Pao2 = 132, PaCo2 = 28, HCO3 = 11. The volume of postoperative bloody drainage was 200 ml. Hemoglobin is 11.8 g/dl and cardiac troponin serum level is 9 ng/ml. New ST elevation was found in V4 to V6 in his ECG. The postoperative CXR is unremarkable. What is the best way to manage this patient?

A. Perform a TTE or TEE for better evaluation of cardiac tamponade.
B. Transfer the patient to the operation room for graft revision and possible additional graft anastomosis.
C. Infuse a crystalloid solution, add a second ionotropic agent and insert an intra-aortic balloon pump.
D. Transfer the patient to the catheterization room and perform a selective coronary angiography and possible coronary stenting.

Answer: B

The most probable clinical differential diagnosis of the patient status is postoperative MI, based on the unstable hemodynamics despite the inotropic support, acceptable Hgb levels, no excessive postoperative bleeding and suitable CVP measures. New-onset ST elevation in the ECG and the elevated troponin levels support this diagnosis. Since the patient is not hemodynamically stable in the early postoperative phase (only 4 hours) and had good distal run-offs and normal LV function, graft thrombosis is the primary diagnosis. Graft revision and putting an additional graft in may alter the patient's status and prevent Q-wave MI and LV dysfunction.

If the patient had stable hemodynamics, evaluation of graft patency should be done by conventional angiography and coronary stenting undertaken if needed.

4. What is the pathology of mitral regurgitation in Barlow's disease according to the Carpentier classification?

A. Type I
B. Type II
C. Type IIIa
D. Type IIIb

Answer: B

In most degenerative mitral regurgitations, including Barlow disease, the main reason for MR is prolaptic leaflets secondary to the elongated and excessive valve tissue (type II Carpentier classification).

5. A 34-year-old woman presents with dyspnea (FCII). Preoperative imaging studies reveal severe aortic regurgitation, LVEF = 35%, LVEDD = 61mm, aortic annulus = 32 mm, Valsalva sinus diameter = 54 mm, STJ diameter = 52 mm, ascending aorta size = 50 mm, and a normal coronary artery. What is the best approach?

A. Closed medical follow-up, beta blockers, serial TTE
B. AV repair and supra-coronary ascending aortal replacement by a tube graft
C. Valve preserving root reconstruction by David operation
D. Bentall procedure

Answer: D

Symptomatic severe AI in a young woman needs surgical intervention. Since the aortic root diameter is larger than 45 mm in a known case of Marfan syndrome, root reconstruction should be performed. Although valve preservation is the preferred approach to a woman of child-bearing age, an aortic annulus diameter of more than 30 mm is a relative contraindication of David operation due to a high chance of recurrent AI in the long term.

References

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2. Bonow RO, Mann DL, Zipes DP, Lippy P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 10th ed. Philadelphia: Saunders Elsevier; 2015.