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EKGs and CARDIAC STUDIES

Essential Evidence-Based Data for Common Clinical Encounters

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- The perfect "portable brain" for the wards
- Vital facts and figures everyone forgets to remember but must know

ANIL M. PATEL

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Contents

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Preface

Despite the advancement of new technologies the EKG remains an absolute staple of medical practice and education. Clinicians, residents, and students are eager to review sample tracings, as they know the value of a timely EKG test and understand the importance of the test to everyday clinical practice.

This book was written to assist clinicians, interns, residents, medical students, or anyone in the health care profession who is likely to encounter EKGs in clinical practice. While there are many EKG resources available in print, we continually hear from students and residents that there is room for improvement, and we believe none of these resources are as detailed and user-friendly as *Lange Instant Access: EKGs and Cardiac Studies.*

The book includes evidence-based information that is essential in practicing medicine. All the information in the manual was acquired from respected references in the medical literature.

This manual is the final product of two and a half years of hard work and was reviewed by some of the most recognized and respected physicians in cardiology and family medicine. We trust that you will find it helpful in your own educational or clinical activities.

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Acknowledgments

Lange Instant Access: EKGs and Cardiac Studies is dedicated to two individuals. One is my grandmother, who inspired me to reach for the stars and nothing less. The second is the someone special to whom my heart will always belong.

I would like to thank all of my teachers and colleagues for their support throughout my years of education and training. Special thanks go out to my best friends, Ray Glover and Pam Gross.

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EKGs and CARDIAC STUDIES

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Basic

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B Cardiac Action Potential and EKG Tracing	3
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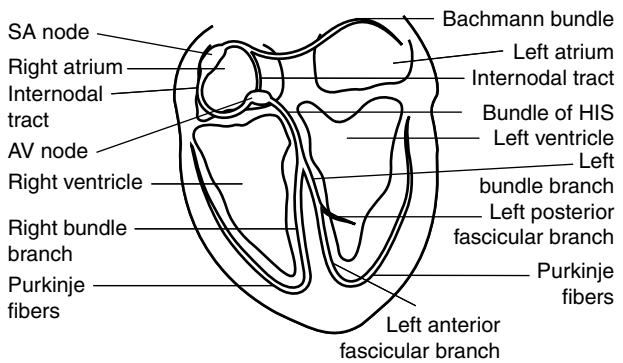
D EKG Tracing	7

A ANATOMY OF CARDIAC CONDUCTION SYSTEM

The normal cardiac conduction pathway is

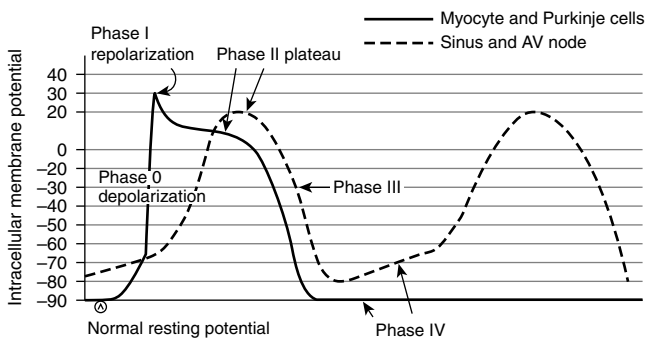
Sinoatrial (SA) node → atrioventricular (AV) node → bundle of HIS → right and left bundle branches → Purkinje system

FIGURE 1–1 Cardiac Conduction System



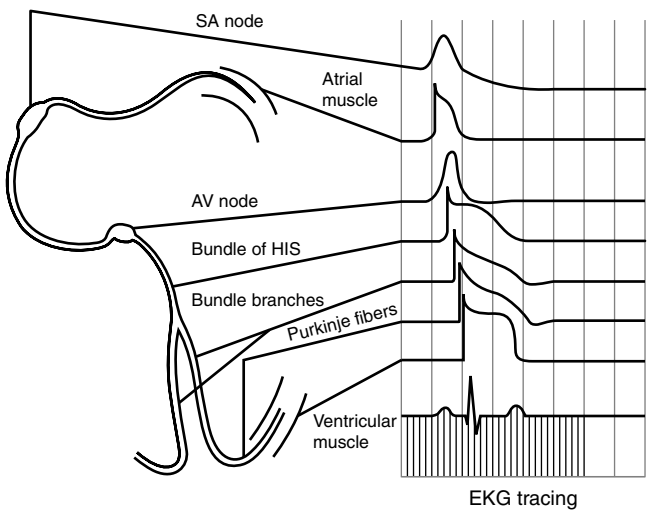
B CARDIAC ACTION POTENTIAL AND EKG TRACING

FIGURE 1–2 Action Potential Generation and Conduction in Myocardium



Phase 0:	Depolarization – Sodium influx in myocyte and Purkinje cells – Calcium influx in sinus and AV node
Phase I:	Initial repolarization
Phase II:	Plateau (sustained calcium influx)
Phase III:	Restoration of membrane resting potential (potassium efflux)
Phase IV:	Restoration of ion gradient by the Na/K pump in myocyte and Purkinje cells Automatic cell depolarization in sinus and AV node

FIGURE 1–3 Cardiac Action Potential



C EKG LEAD PLACEMENT

Precordial Lead Placement

V_1: Right of sternum, fourth intercostal space
V_2: Left of sternum, fourth intercostal space
V_3: Midway between V_2 and V_4
V_4: Midclavicular line, fifth intercostal space
V_5: Midway between V_4 and V_6
V_6: Midaxillary line, fifth intercostal space

FIGURE 1–4

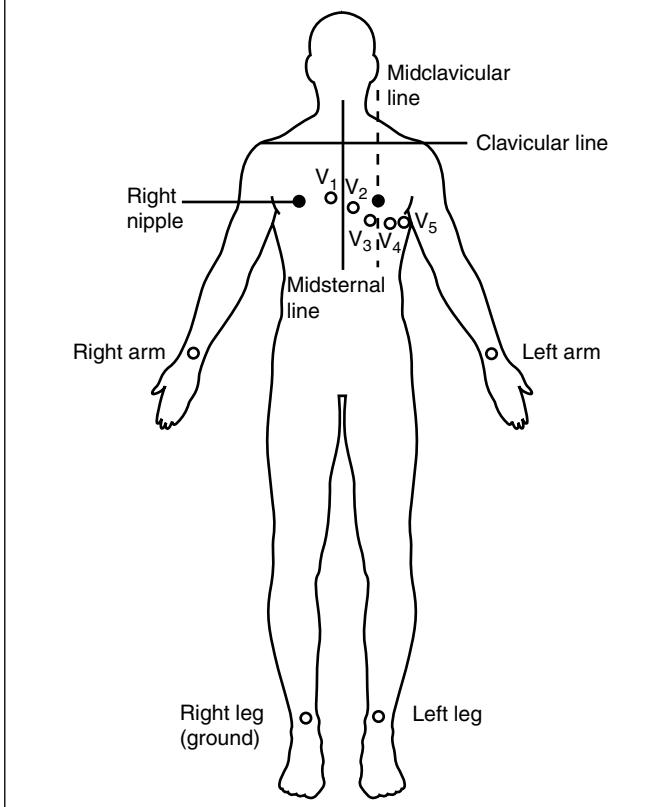
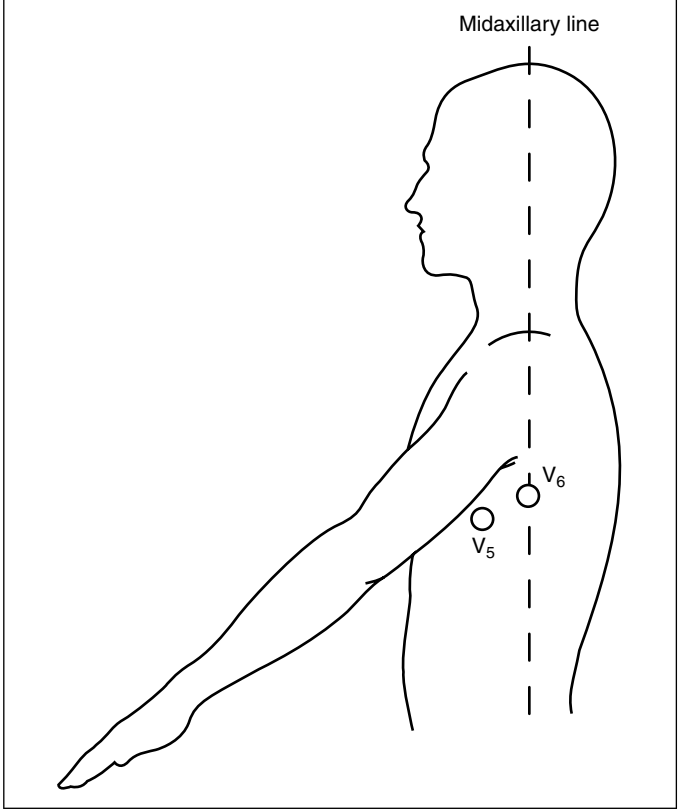
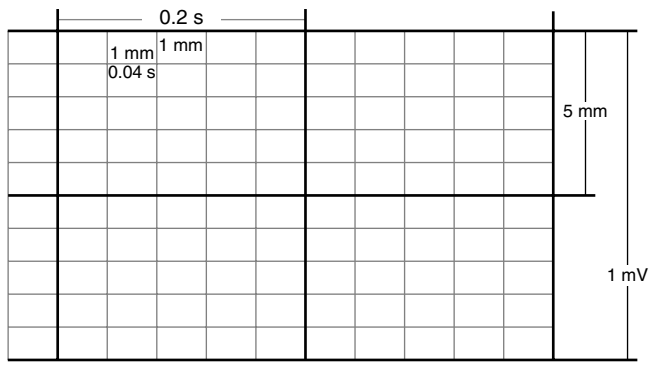

Midclavicular line
Clavicular line
Right nipple
V1
V2
V3
V4
V5
Midsternal line
Right arm
Left arm
Right leg (ground)
Left leg

FIGURE 1–5



D EKG TRACING

FIGURE 1–6 Cardiac Conduction System



i. **Vertical axis:**
- 1 small box = 1 mm
- 1 large box = 5 mm
- 10 mm = 1 mV

ii. **Horizontal axis:**
- 1 small box = 0.04 seconds
- 1 large box = 0.20 seconds
- 5 large boxes = 1 second
- 30 large boxes = 6 seconds

FIGURE 1–7

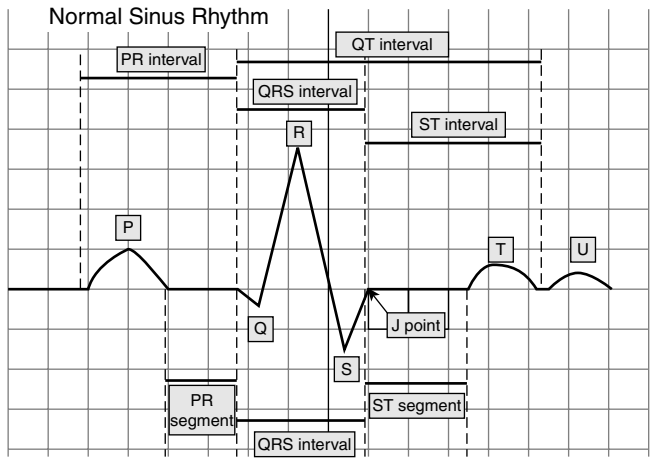

Normal Sinus Rhythm
PR interval
QT interval
QRS interval
ST interval
R
P
T
U
Q
J point
S
PR segment
ST segment
QRS interval

TABLE 1–1: EKG: Waves and Intervals		
■ **P wave** = depolarization of the atria		
■ **QRS** = depolarization of the ventricle		
■ **T wave** = repolarization of the ventricle		
Normal Values		
	Duration (horizontal axis)	**Height (vertical axis)**
P wave	<0.12 s	<2.5 mm
P-R interval	0.12-0.20 s	
QRS interval	0.08-0.10 s	
QT interval	0.35-0.44 s	
QTc interval = QT interval divided by the square root of R-R interval		
Age group	**QTc interval by age**	
0-2 yrs	0.37-0.53	
2-10 yrs	0.39-0.42	
10-14 yrs	0.40-0.42	
>15 yrs	0.35-0.44	

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2
Rate

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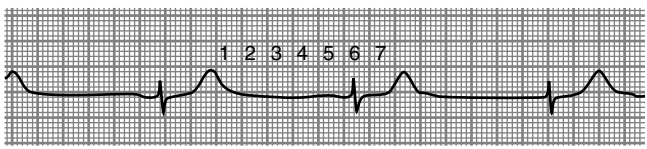
A RATE CALCULATION

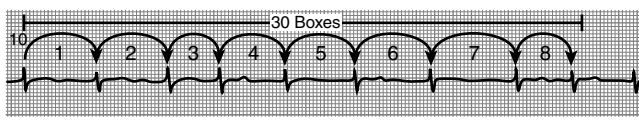
i. Rate is cycles or beats per minute.
ii. Normal rate for the sinoatrial (SA) node is 60 to 100 beats per minute.
iii. Less than 60/minute = sinus bradycardia.
iv. Greater than 100/minute = sinus tachycardia.

There are three well-known methods for calculating the rate.

Count number of large boxes between R-R wave and divide 300 by the number of boxes (300/7 = 42).

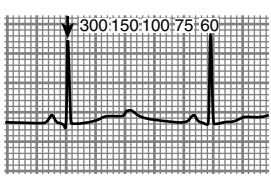


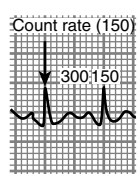
Count number of complete QRS complexes in 6 seconds (30 large boxes) multiplied by 10 ($10 \times 8 = 80$).

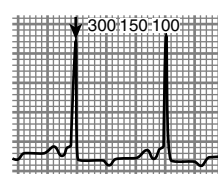


Per big boxes: 300-150-100-75-60.
(Take an R wave on a heavy line or close to heavy line. The next heavy line that is encountered is rate of 300. The next one is 150 followed by 100, 75, and 60 and ending with 50. [See example below].)

Example







3
Rhythm

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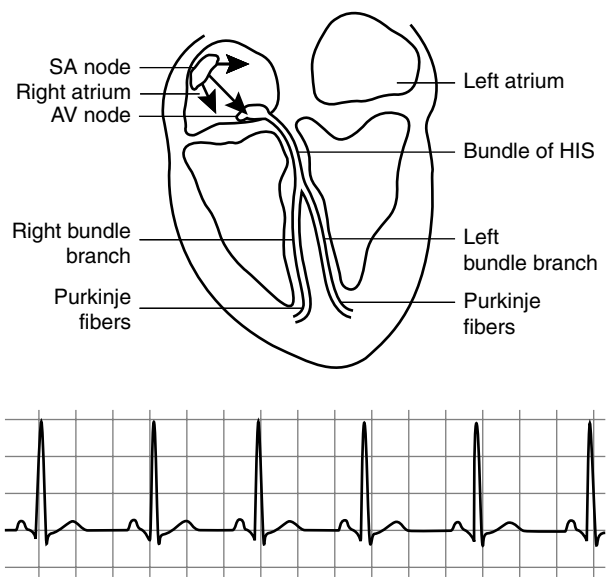
A RHYTHM GUIDELINES

i. Check for a P wave before each QRS (known as sinus rhythm).
ii. Check the rhythm strip for regularity (regular, regularly irregular, and irregularly irregular).
iii. Check PR interval (for atrioventricular [AV] blocks).
iv. Check QRS interval (for block, widening).
v. Check for QT interval prolongation.

FIGURE 3–1 Normal Sinus Rhythm Pathway



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A AXIS AND VECTORS

FIGURE 4–1

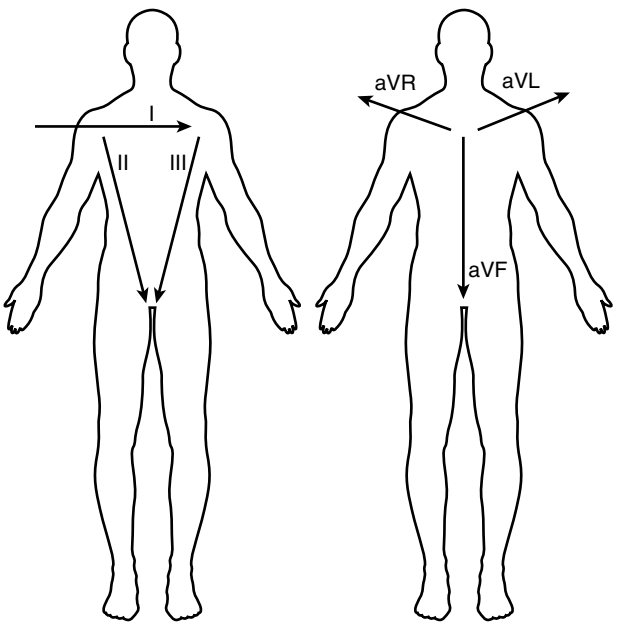
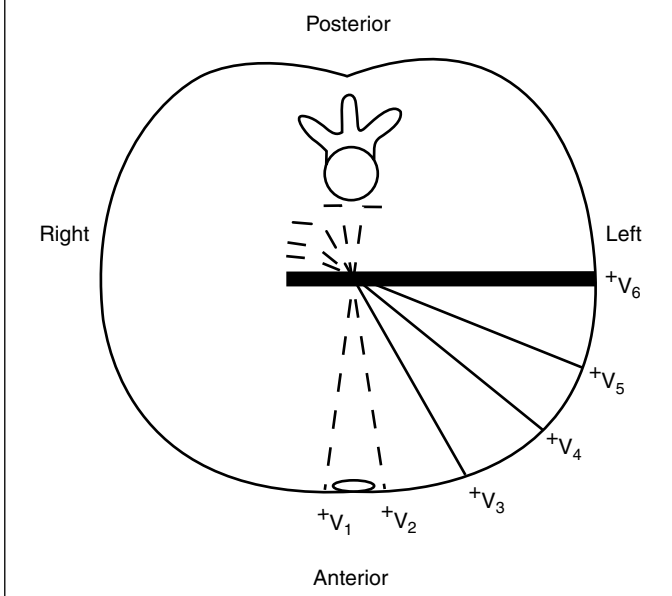


FIGURE 4–2



Direction of depolarization (vector) of the QRS complex:

i. The left ventricle is thicker, so the mean QRS vector is down and to the left. (The origin of the vector is the AV node with the left ventricle being down and to the left of this.)

ii. The vector will point toward hypertrophy (corresponding to electrocardiogram [EKG] deflections above the electrical baseline) and away from the infarct (corresponding to EKG deflections below the electrical baseline).

FIGURE 4–3

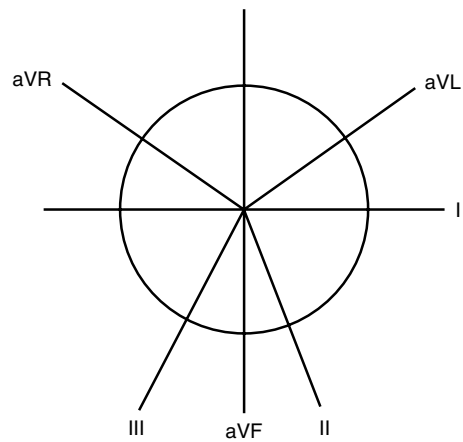


FIGURE 4–4

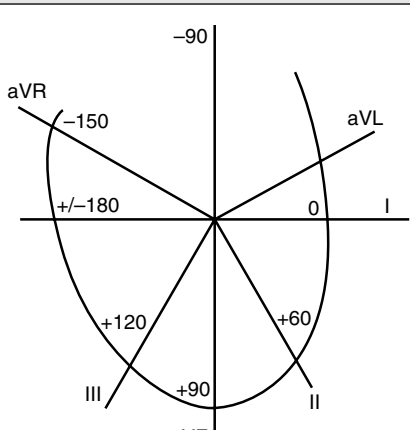
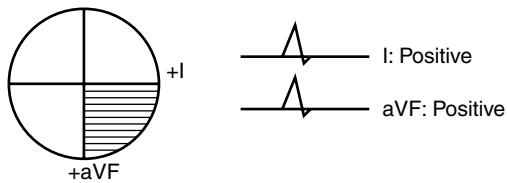


TABLE 4–1 Axis Deviation			
Axis	**Degree (angle)**	**Lead I**	**Lead aVF**
Normal axis	0 to +90	Positive	Positive
Left axis deviation (LAD)	−30 to −90	Positive	Negative
Right axis deviation (RAD)	+90 to +180	Negative	Positive
Indeterminate (extreme) axis deviation	−90 to −180	Negative	Negative

Etiology			
Left axis deviation	LVH	Left anterior fascicular block	Inferior wall MI
Right axis deviation	RVH	Left posterior fascicular block	Lateral wall MI

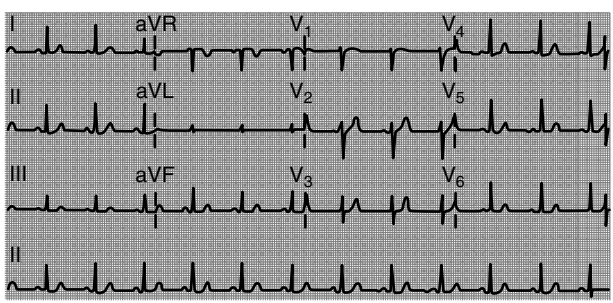
B NORMAL AXIS

FIGURE 4–5 Normal Axis



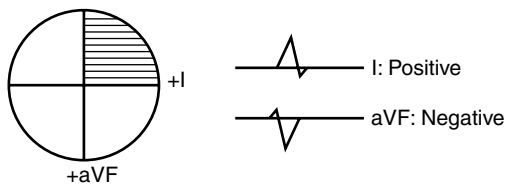
Example

FIGURE 4–6 Normal Axis EKG



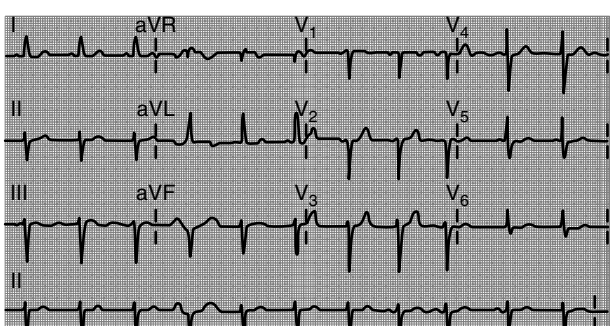
C LEFT AXIS DEVIATION

FIGURE 4–7 Left Axis Deviation



Example

FIGURE 4–8



D RIGHT AXIS DEVIATION

FIGURE 4–9 Right Axis Deviation

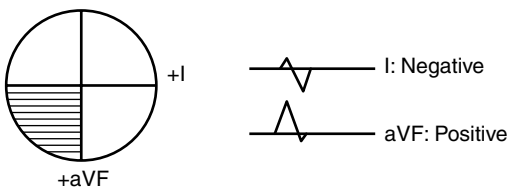
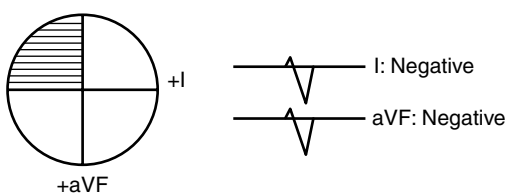
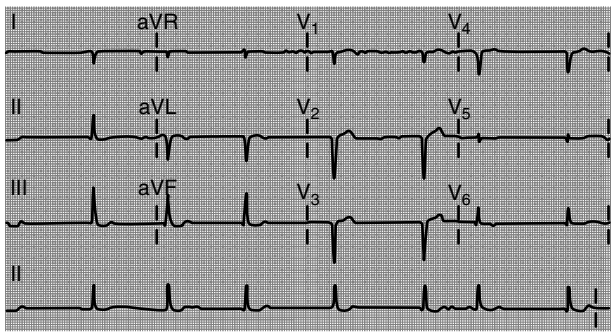


FIGURE 4–10 Extreme Right Axis Deviation



Example

FIGURE 4–11 Right Axis Deviation



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5
Hypertrophy

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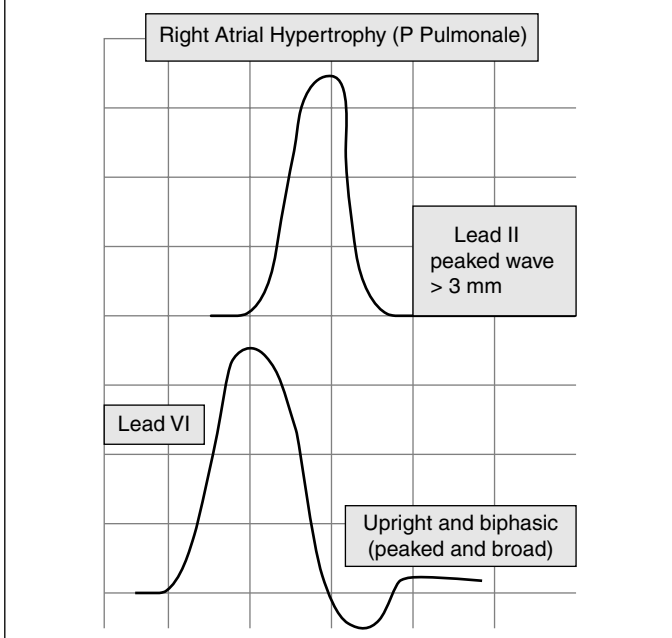
B Ventricular Hypertrophy	29

A ATRIAL HYPERTROPHY

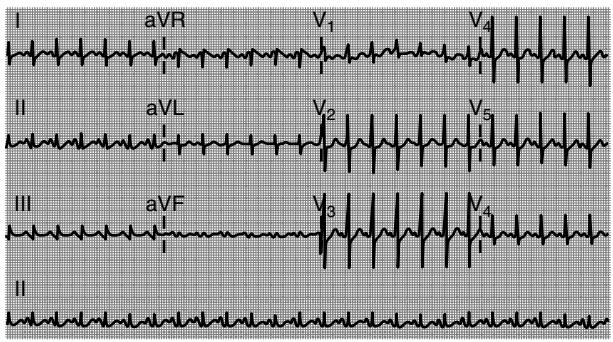
i. **Right atrial hypertrophy**
- Lead II: P wave (>3 mm amplitude)
- Lead V_1: Upright and biphasic P wave

FIGURE 5–1



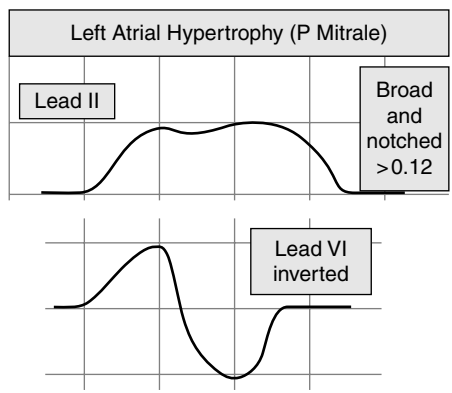
Example

FIGURE 5–2 Right Atrial Enlargement



ii. **Left atrial hypertrophy**
- Lead II: Broad and notched P wave (>0.12 mm)
- Lead V_1: Biphasic P wave with broad negative phase

FIGURE 5–3

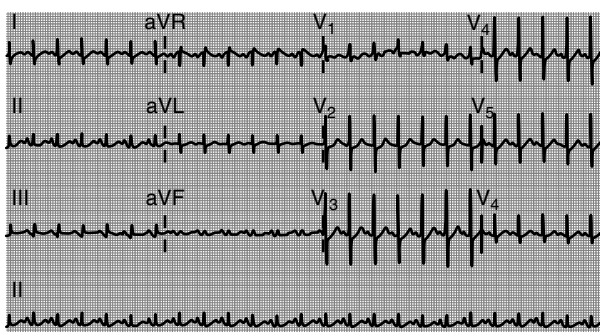


B VENTRICULAR HYPERTROPHY

i. **Right ventricular hypertrophy**
- Right axis deviation
- Possibly a predominant R wave in lead V_1 (in a normal EKG, the S wave is dominant in V_1)
- Deep S in V_6 (in a normal EKG, the QRS complex is predominantly upward in V_6)
- Inverted T waves in leads V_2, V_3
- Peaked P waves may also occur due to right atrial hypertrophy
- QRS < 0.12 second

Example

FIGURE 5–4 Right Ventricular Hypertrophy

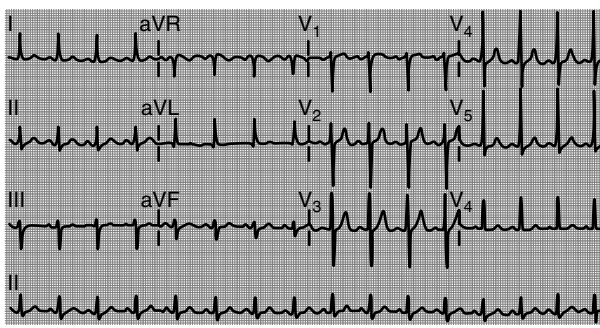


ii. **Left ventricular hypertrophy**

- aVL: R wave > 12 mm
- V_1 or V_2 and V_5 or V_6: S wave in V_1 or V_2 + R wave in V_5 or V_6 → = 35 mm
- V_5 or V_6: R wave > 27 mm

Example

FIGURE 5–5 Left Ventricular Hypertrophy



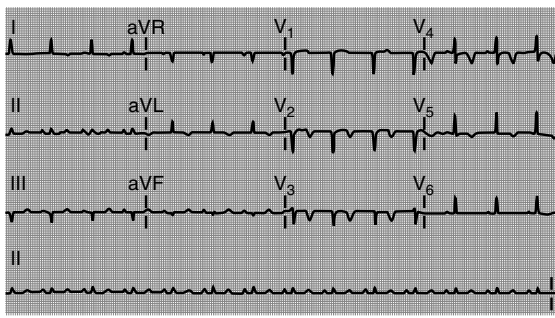
6
Ischemia, Injury, and Infarction

TABLE 6–1 Ischemia, Injury, and Infarct		
Ischemia	Is a relative lack of blood supply	T-wave inversion or ST-segment depression (commonly seen in I, II, V_2–V_6)
Acute injury	Acute damage to myocardium	Elevated ST-segments with or without Q waves
Old infarct	Dead myocardium	Q waves without ST-segment elevation

TABLE 6–2 Leads and Its Location	
V_1–V_2	Anteroseptal wall
V_3–V_4	Anterior wall
V_5–V_6	Anterolateral wall
II, III, aVF	Inferior wall
I, aVL	Lateral wall
V_1–V_2 or V_7–V_9	Posterior wall
V_4R	Right ventricle wall

A ISCHEMIA

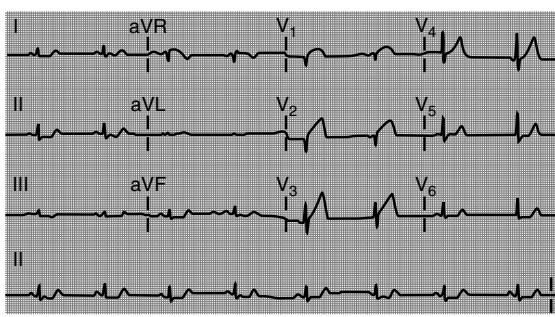
FIGURE 6–1 Ischemia



Note: Symmetric T-Wave Inversions in Leads I, V_2 to V_5

B INJURY

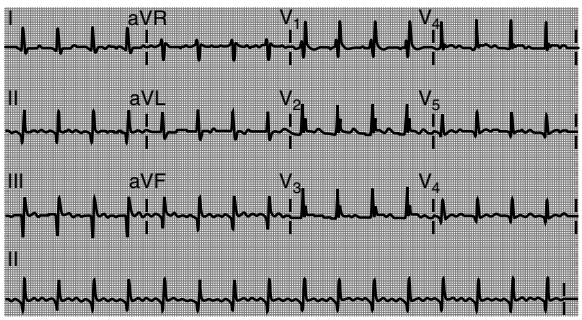
FIGURE 6–2 Injury



Note: ST-Segment Elevation in Leads V_2 to V_3 (Anteroseptal/ Anterior Wall)

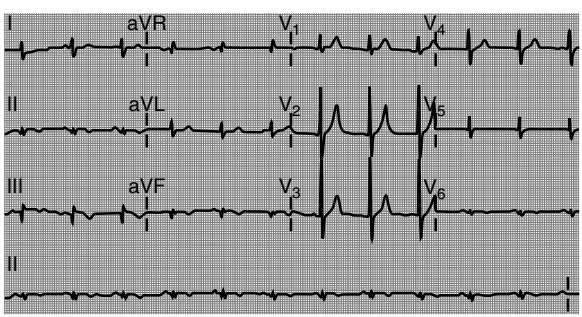
C INFARCT

FIGURE 6–3 Recent Infarct



Note: Q Waves with ST-Segment Elevation in Leads II, III, and aVF (Inferior Wall)

FIGURE 6–4 Inferoposterior Wall Infarct



Note: Tall R wave in V_1 posterior wall infarcts are often associated with inferior wall infarcts (Q waves in II, III, and aVF). Acute posterior wall infarction-related EKG changes can also have tall R waves and ST segment depression in V_1 and V_2.

7
Conduction Blocks

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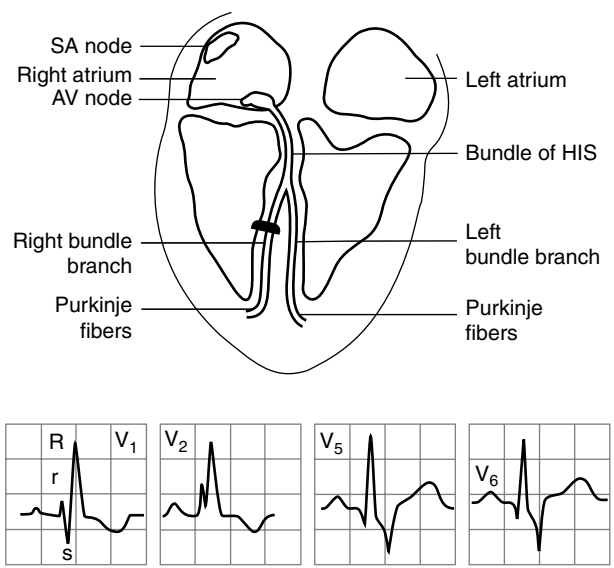
A BUNDLE BRANCH BLOCKS

i. **Complete right bundle branch block**
- QRS complex: ≥0.12 seconds
- S wave: Wide in lead I, wide and slurred in V_5 to V_6
- rsR′: V_1 and V_2
- Secondary ST- and T-wave changes in V_1 and V_2

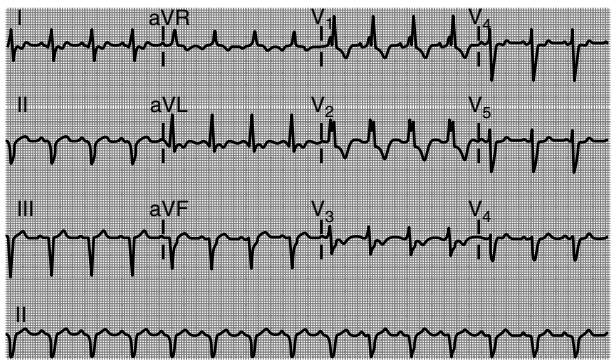
ii. **Incomplete right bundle branch block**
- QRS complex: Between 0.09 to 0.12 seconds
- Axis: May or may not have right axis deviation

FIGURE 7–1 Right Bundle Branch Block (RBBB)



Example: RBBB

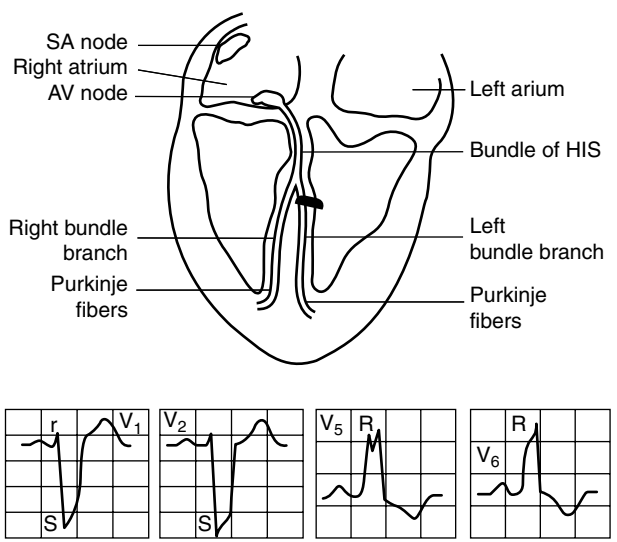
FIGURE 7–2



iii. **Complete left bundle branch block**
- QRS complex: ≥0.12 seconds
- R wave: Wide and slurred in V_5 to V_6
- Leads I, V_5, V_6: ST depression and inverted T wave and lack of Q waves

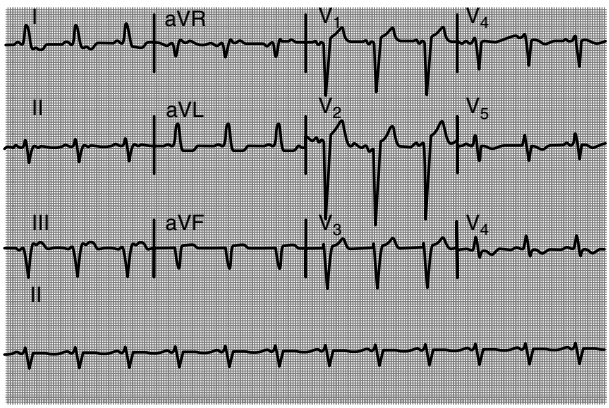
iv. **Incomplete left bundle branch block**
- QRS complex: Between 0.09 and 0.12 seconds
- R wave: Tall R waves in V_5 to V_6
- Lack of Q wave: I, aVL, V_5 to V_6

FIGURE 7–3 Left Bundle Branch Block (LBBB)



Example: LBBB

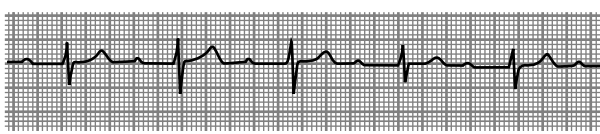
FIGURE 7–4



B FIRST-DEGREE AV BLOCKS

i. **PR intervals:** ≥0.20 seconds or 200 ms
ii. **Etiology:**
- Medications
  - Beta blocker
  - Calcium channel blocker
  - Digitalis
  - Quinidine
- Excessive vagal tone
- Intrinsic disease in the AV junction

FIGURE 7–5 First-Degree AV Block

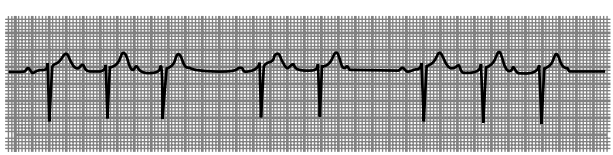


- P wave: P wave prior to QRS wave
- PR interval: >0.20 seconds
- QRS complex: >0.12 seconds
- Rhythm: Normal

C SECOND-DEGREE BLOCKS

i. **Mobitz type I (Wenckebach)**
- Rate: 60 to 100 beats/minute
- Atrial rhythm: Regular
- Ventricular rhythm: Progressive shortening of the R-R interval until the QRS is dropped
- P-wave configuration: Normal
- PR interval: Prolonged with each beat until QRS is dropped
- QRS complex: Normal
- ST segment: Normal
- T wave: Normal
- Etiology: Inferior wall MI, digitalis, beta blocker, calcium channel blocker, rheumatic fever, myocarditis, and excessive vagal tone

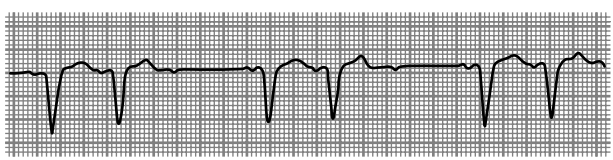
FIGURE 7–6 Second-Degree Type 1 Block



ii. **Mobitz type II (2:1, 3:1 AV block)**

- Rate: Ventricular rate is variable.
- Atrial rhythm: Regular (the P-P interval is constant).
- Ventricular rhythm: Irregular.
- P wave: 2:1, 3:1, or 4:1 conduction with QRS.
- PR interval: Constant (PR intervals are constant until a nonconducted P wave occurs).
- Etiology: Anterior or anteroseptal MI, cardiomyopathy, rheumatic heart disease, coronary artery disease, beta blocker, calcium channel blocker, digitalis.

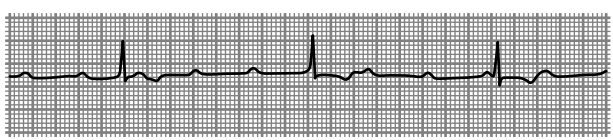
FIGURE 7–7 Second-Degree Type 2 Block



D THIRD-DEGREE AV BLOCKS (COMPLETE HEART BLOCK)

i. There is no relationship with P wave and QRS complex because there is complete AV dissociation.
ii. The dissociation is due to atria and ventricles being controlled by separate foci.
   - Atrial rhythm: Regular
   - P-wave configuration: Normal
   - PR interval: No relationship between P wave and QRS complexes
   - QRS complex: Variable (depends on the intrinsic rhythm)
   - ST segment: Normal
   - T wave: Normal
   - Etiology: Anterior and inferior MI, coronary artery disease, excessive vagal tone, myocarditis, endocarditis, digitalis, beta blocker, calcium channel blocker.

FIGURE 7–8 Third-Degree AV Block

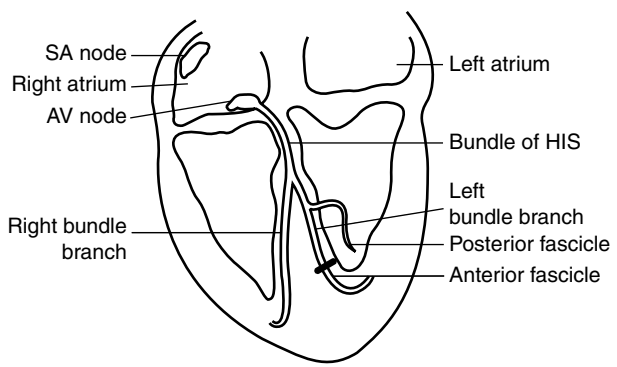


E FASCICULAR BLOCKS

Fascicular blocks are blocks on part of the left bundle, either the posterior or the anterior division.

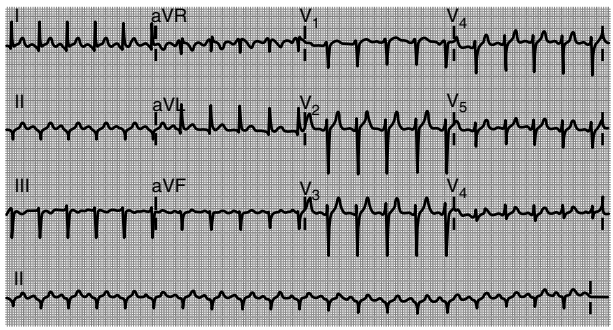
i. **Left anterior fascicular block** (the most common intraventricular conduction defect)
   - Left axis deviation (–30 to –90 degrees).
   - rS complexes in II, III, aVF.
   - Small q in I and/or aVL.
   - The QRS will be slightly prolonged (0.1-0.12 seconds).

FIGURE 7–9 Anterior Fascicular Block



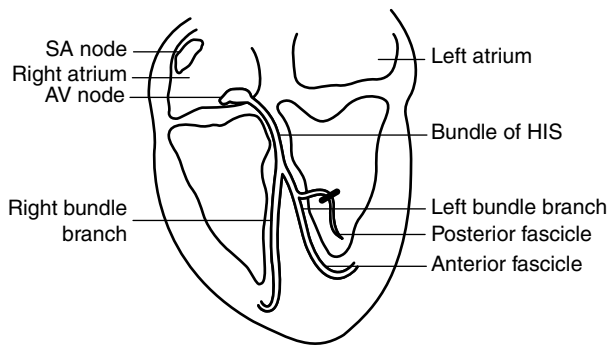
Example

FIGURE 7–10 Anterior Fascicular Block



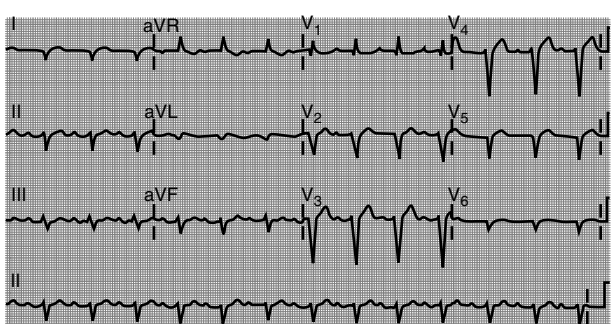
ii. **Left posterior fascicular block** (less common)
- Right axis deviation (usually >+100 degrees)
- rS in lead I
- Q in lead III (S1Q3)
- The QRS will be slightly prolonged (0.1-0.12 seconds)

FIGURE 7–11 Posterior Fascicular Block



Example

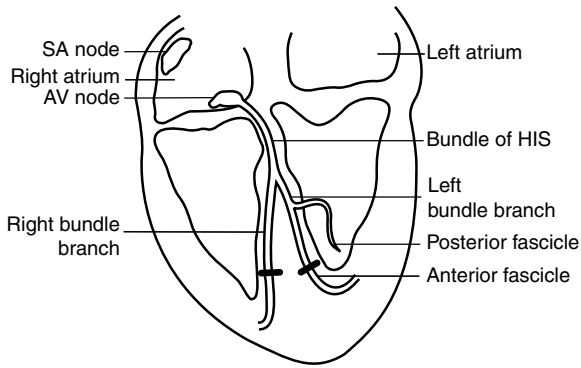
FIGURE 7–12 Posterior Fascicular Block



iii. **Bifascicular block**

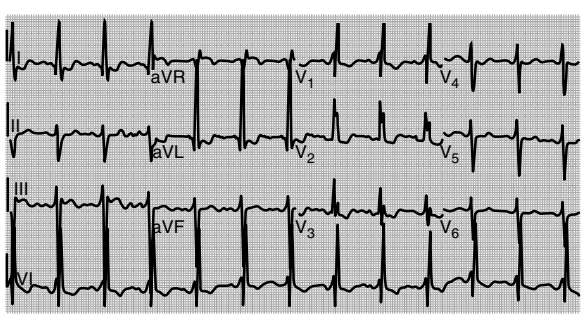
- Represents block of two of the three fascicles.
- The most common of them is RBBB plus left anterior fascicular block (LAFB) or left posterior fascicular block (LPFB).

FIGURE 7–13 Bifascicular Block



Example

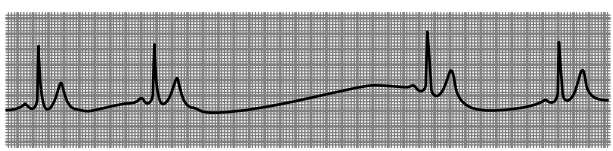
FIGURE 7–14 Right Bundle Branch Block and Left Anterior Fascicular Block



F SINUS PAUSE

i. **Rate:** Variable
ii. **Rhythm:** Sinus
iii. **P wave:** Conducted P wave occurs later in time than expected based on previous sinus rhythm (P-P interval is disturbed)
iv. **PR interval:** 0.12 to 0.20 seconds
v. **QRS complex:** <0.12

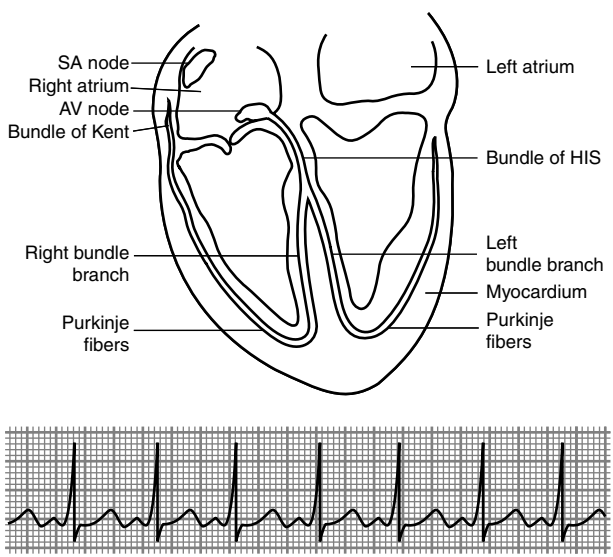
FIGURE 7–15



G WOLFF-PARKINSON-WHITE SYNDROME

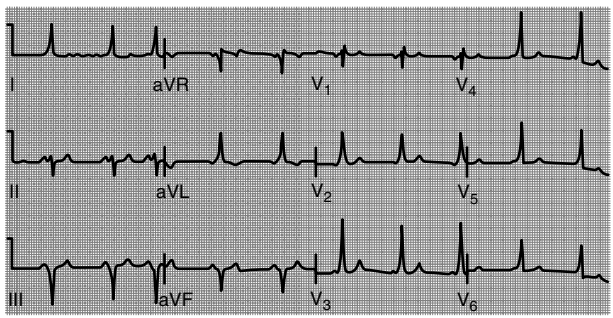
i. **Rhythm:** Sinus
ii. **P wave:** Normal
iii. **P-R interval:** Short (<0.12 second)
iv. **QRS complex:** Slurred (delta wave), prolonged with ST segment and T wave changes

FIGURE 7–16 Wolff-Parkinson-White Syndrome



Example

FIGURE 7–17 WPW Syndrome



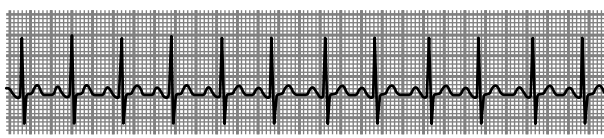
8
Arrhythmias

OUTLINE	
A Supraventricular Arrhythmia	52
B Ventricular Rhythm	73
C Paced Rhythm	83
D Miscellaneous	84

A SUPRAVENTRICULAR ARRHYTHMIA

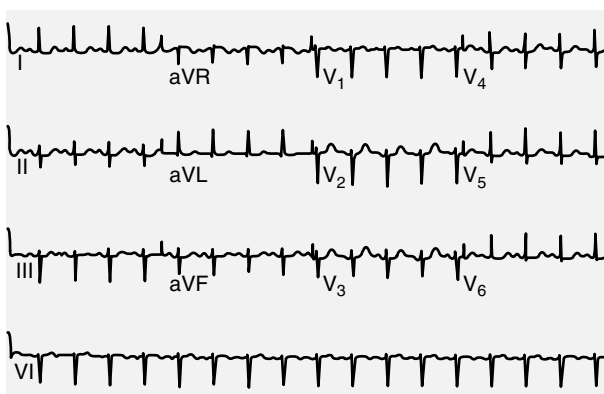
i. **Sinus tachycardia**
- Rate: >100 beats/minute
- Rhythm: Sinus
- P wave: Normal prior to each QRS complex
- PR interval: 0.12 to 0.20 seconds
- QRS complex: <0.12 seconds

FIGURE 8–1



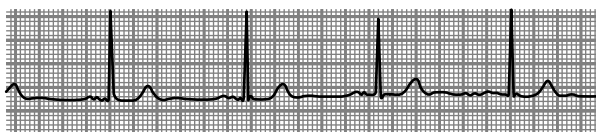
Example

FIGURE 8–2



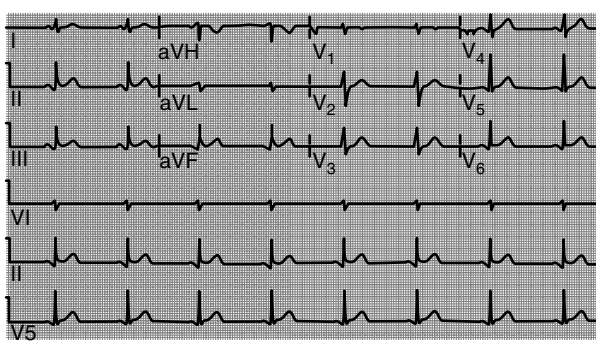
ii. **Sinus bradycardia**
- Rate: <60 beats/minute
- Rhythm: Sinus
- P wave: Normal prior to each QRS complex
- PR interval: 0.12 to 0.20 seconds
- QRS complex: <0.12 seconds

FIGURE 8–3



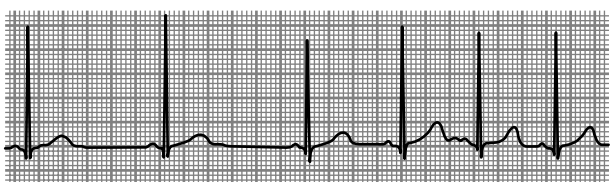
Example

FIGURE 8–4



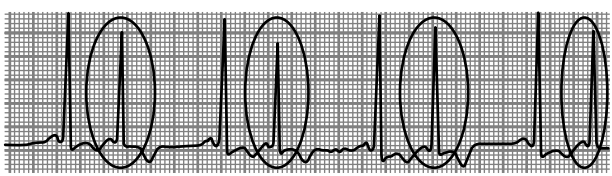
iii. **Sinus arrhythmia**
- Rate: 60 to 100 beats/minute
- Rhythm: Irregular (10% variation in P-P interval)
- P wave: Normal prior to QRS complex
- P-R interval: 0.12 to 0.20 seconds
- QRS complex: <0.12 seconds

FIGURE 8–5



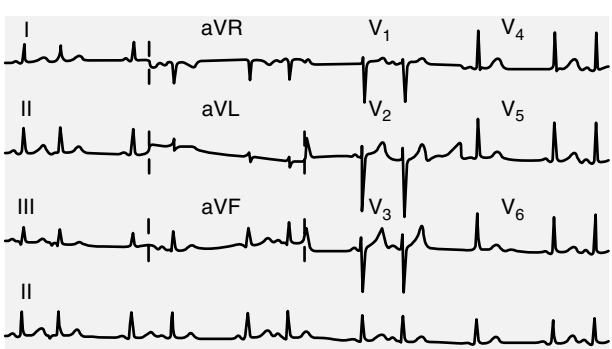
iv. **Atrial bigeminy:** Each sinus beat is followed by an atrial premature beat.
- Rate: N/A
- Rhythm: Irregular
- P wave: Premature and abnormal or hidden
- PR interval: <0.20 seconds
- QRS complex: <0.12 seconds
- Note premature beats below

FIGURE 8–6



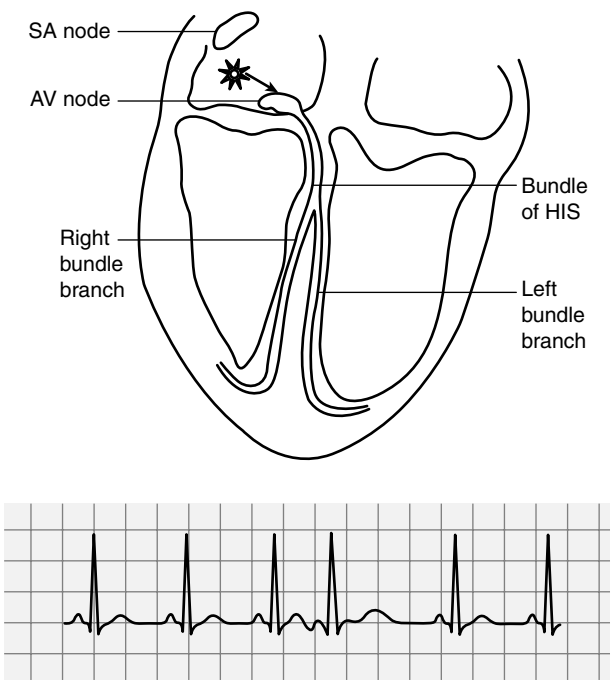
Example

FIGURE 8–7 Atrial Bigeminy



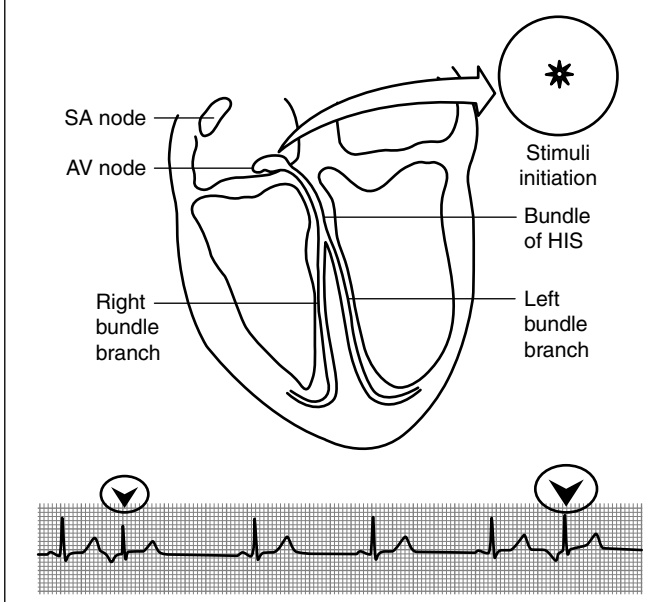
v. **Premature atrial contraction (PAC)**
- Rate: N/A
- Rhythm: Irregular
- P wave: Ectopic
- PR interval: May be normal or >0.20 seconds
- QRS complex: <0.12 seconds
- Normal ventricular complex

FIGURE 8–8 Premature Atrial Contraction



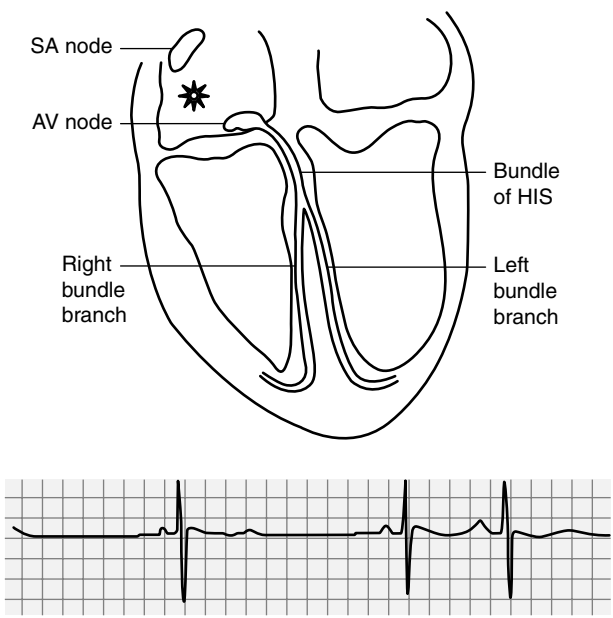
vi. **Premature junctional beat or complex**
- Rate: 60 to 100 beats/minute
- Rhythm: Irregular
- P wave: Can occur prior, during, and after QRS complex
- PR interval: <0.12 seconds
- QRS complex: <0.12 seconds
- Is an ectopic foci in the AV junction

FIGURE 8–9 Premature Junction Beat



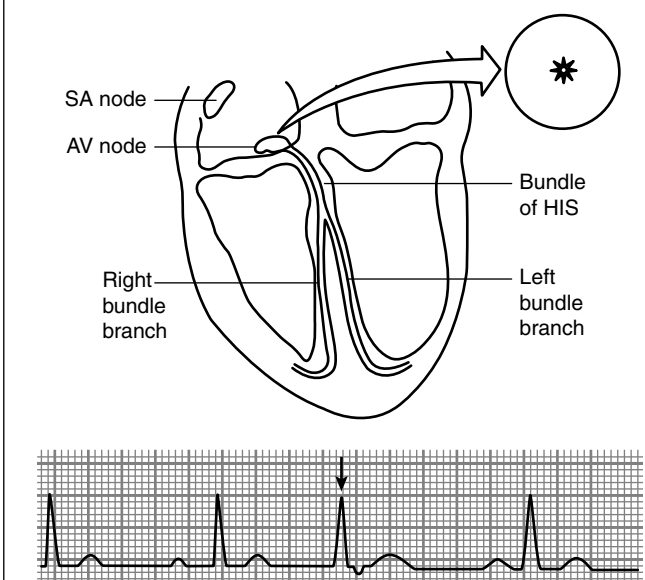
vii. **Atrial escape beat**
Sinus P wave QRS complex that is followed by a P-QRS complex in which the P wave appears later and may have slightly different morphology than the sinus P wave

FIGURE 8–10 Atrial Escape Beat



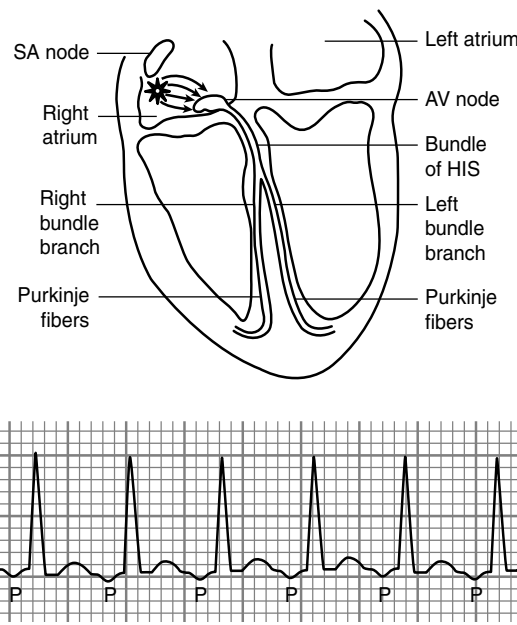
viii. **Junctional escape beat**
- An escape beat that occurs after a pause in the normal sinus rhythm.
- Atrial pacing usually resumes after the junctional beat.
- P wave is missing prior to junctional beat.

FIGURE 8–11 Junctional Escape Beat



ix. **Supraventricular tachycardia (SVT)**
- Regular rhythm
- Rate 140 to 220 beats/minute
- Abnormal P wave (not easily identified)
- Nonspecific ST- and T-wave changes
- QRS complex can be narrow or wide depending on whether there is aberrant conduction

FIGURE 8–12 Supraventricular Tachycardia (SVT)



Example

FIGURE 8–13 SVT

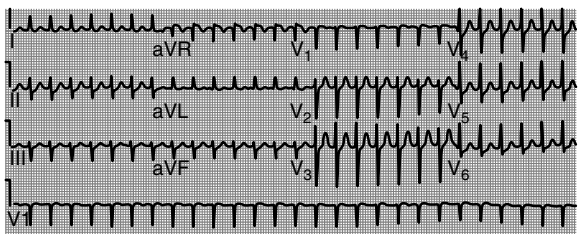
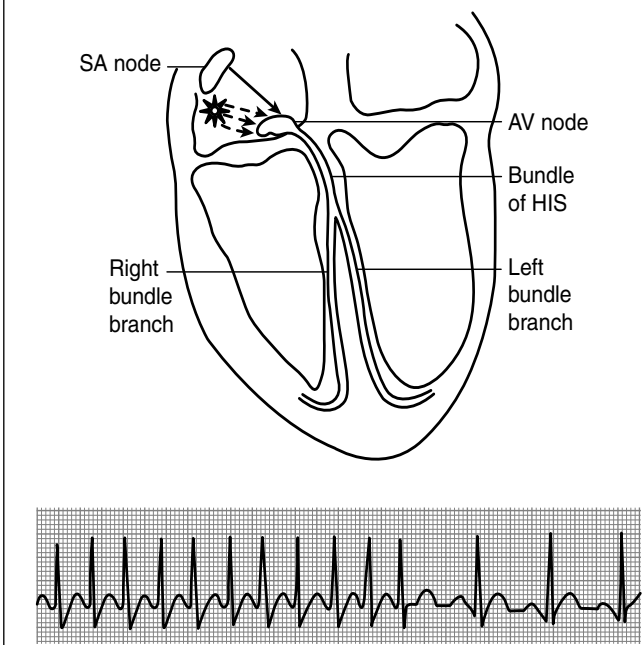
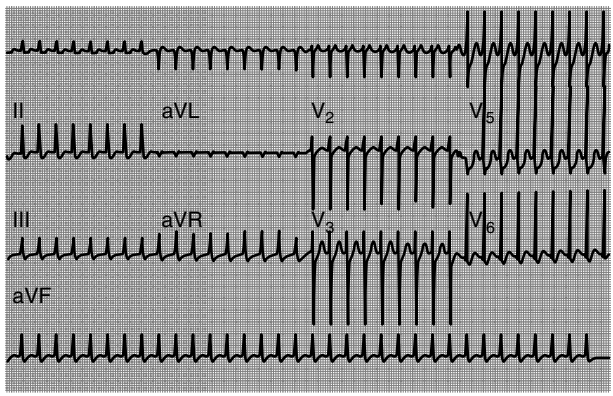


FIGURE 8–14 Paroxysmal Atrial Tachycardia (PAT) or Paroxysmal Supraventricular Tachycardia



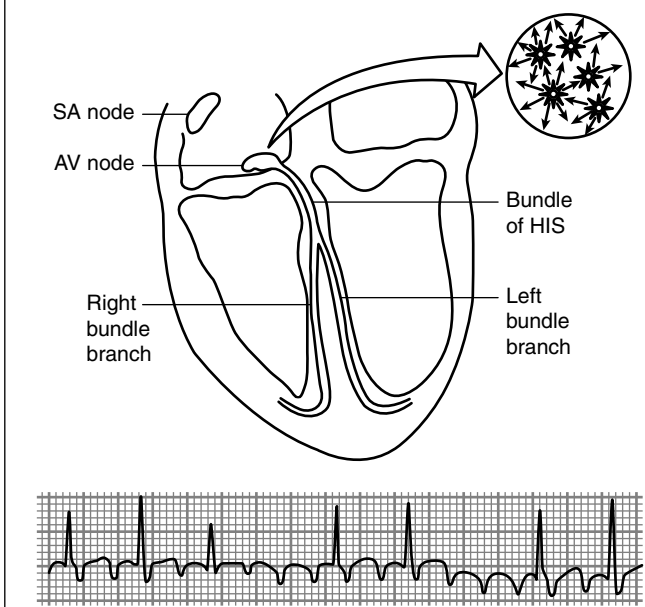
Example

FIGURE 8–15 PAT/PSVT



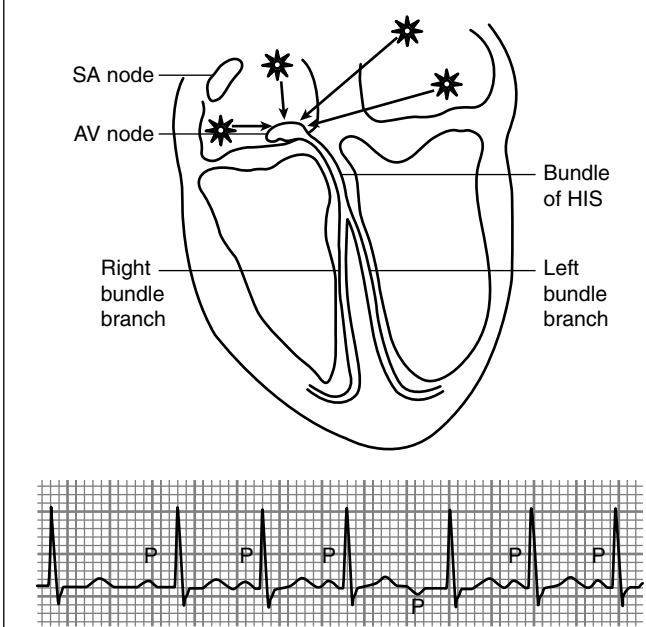
i. Rate exceeds 100 beats/minute
ii. Negative P waves can be seen

FIGURE 8–16 Paroxysmal Junctional Tachycardia



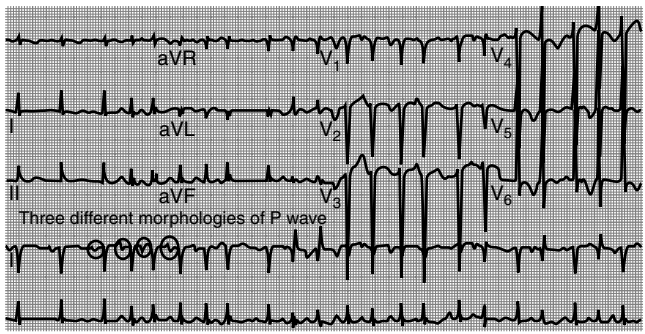
x. **Multifocal atrial tachycardia (MAT)**
- Rate: >100 beats/minute (if <100 beats/minute → it is called multifocal atrial rhythm)
- P wave: At least three different shapes of P waves in one lead
- Rhythm: Irregularly irregular with varying PR, R-R, and PR intervals
- Etiology: COPD or other underlying lung disease

FIGURE 8–17 Multifocal Atrial Tachycardia (MAT)



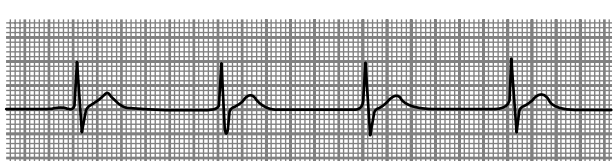
Example

FIGURE 8–18 MAT



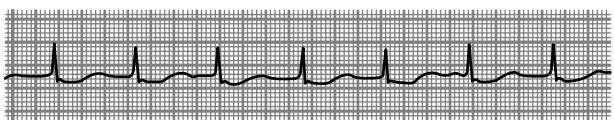
xi. **Junctional rhythm**
- Rate: 40 to 60 beats/minute
- Rhythm: Regular
- P wave: Inverted or absent or after QRS complexes or <0.10 seconds
- PR interval: <0.12 seconds
- QRS complex: <0.12 seconds
- Etiology: Inferior wall MI, hypoxia, electrolyte disturbance, CHF, cardiomyopathy

FIGURE 8–19 Junctional Rhythm



xii. **Accelerated junctional rhythm**
- Rate: 60 to 100 beats/minute
- Rhythm: Regular
- P wave: Inverted or absent or after QRS complexes or <0.10 seconds
- PR interval: <0.12 seconds
- QRS complex: <0.12 seconds

FIGURE 8–20 Accelerated Junctional Rhythm



xiii. **Atrial fibrillation**

- Rate: >350 beats/minute
- Rhythm: Irregularly irregular
- P wave: Absent/fibrillatory waves
- PR interval: N/A
- QRS complex: <0.12 seconds

FIGURE 8–21 Atrial Fibrillation (A.Fib)

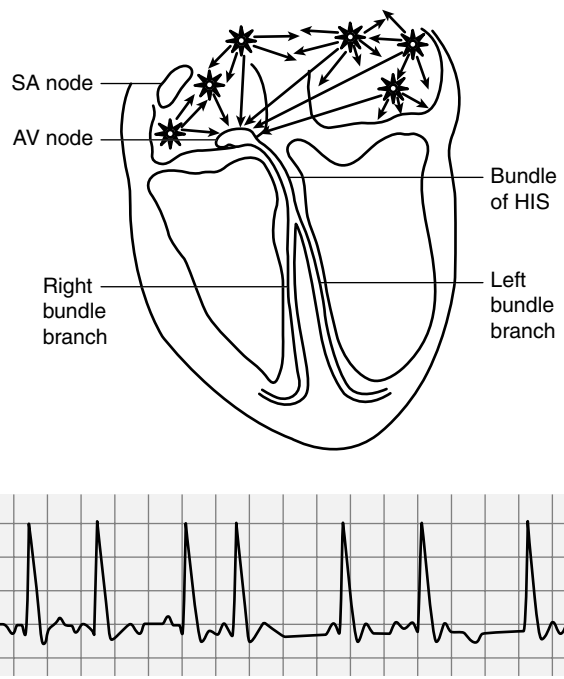
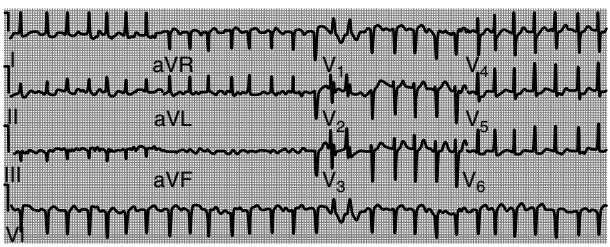


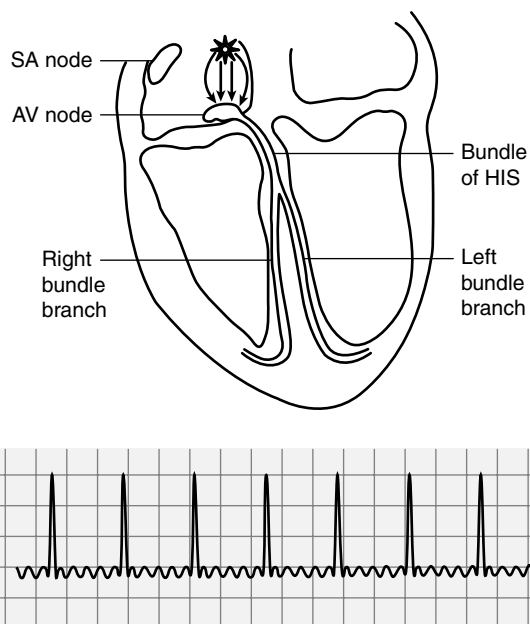
FIGURE 8–22 A-Fib



xiv. **Atrial flutter**

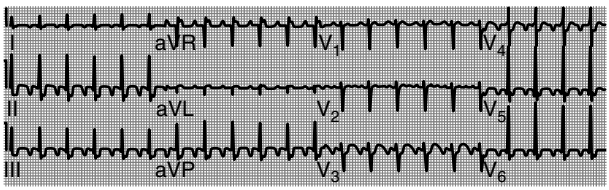
- Rate: 200 to 350 beats/minute (if rate is 150 beats/minute, it may flutter with 2:1 block)
- Rhythm: Regular, sawtooth pattern (best noted in II, III, aVF)
- P wave: Absent/sawtooth pattern
- PR interval: N/A
- QRS complex: <0.12 seconds (conduction ratio—P wave to QRS—may be 2:1, 3:1, etc)

FIGURE 8–23 Atrial Flutter



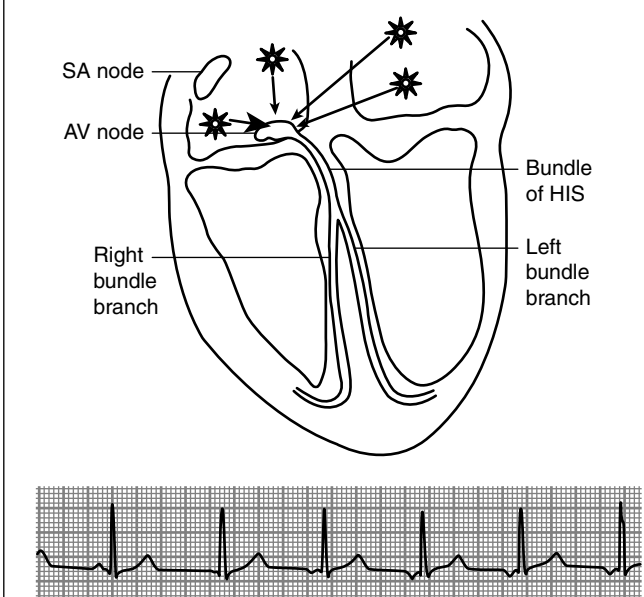
Example

FIGURE 8–24 Atrial Flutter



xv. **Wandering atrial pacemaker**
- Rate: <100 beats/minute
- Rhythm: Irregular
- P wave: ≥3 morphologies
- PR interval: Variable
- QRS complex: <0.12 seconds

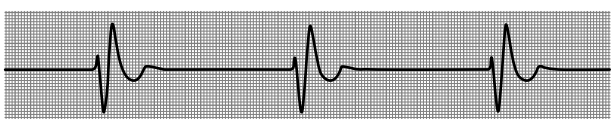
FIGURE 8–25 Wandering Pacemaker



B VENTRICULAR RHYTHM

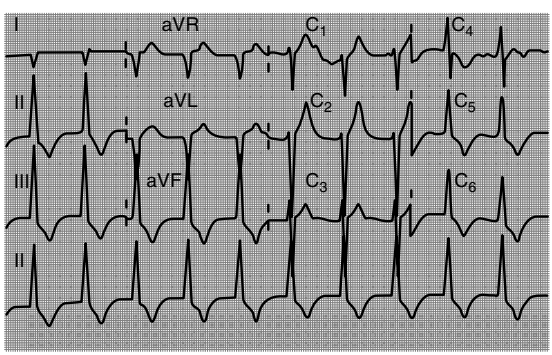
i. **Idioventricular rhythm:** Benign rhythm commonly associated with reperfusion
   - Rate: 30 to 40 beats/minute
   - Accelerated idioventricular rhythm rate (AIVR): 40 to 60 beats/minute
   - Benign and commonly associated with reperfusion in the setting of acute myocardial infarction
   - Rhythm: Regular without P wave or no relationship between P wave and QRS complexes
   - P wave: May be absent
   - QRS complex: Wide (>0.12 seconds)

FIGURE 8–26



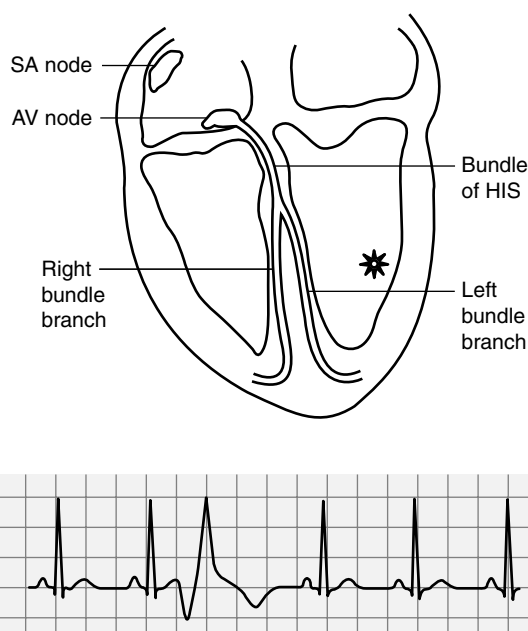
Example

FIGURE 8–27



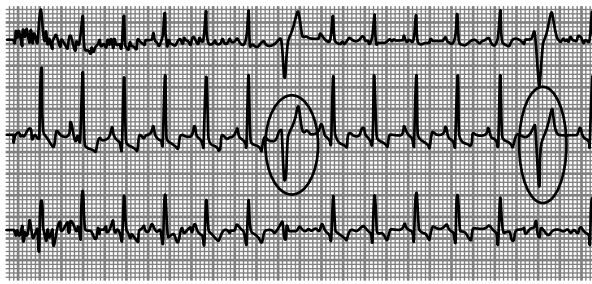
ii. **Premature ventricular contraction (PVC)**
- Is an ectopic foci originating in the ventricles.
- Three PVCs are considered a run of ventricular tachycardia.
- Bigeminy: PVCs that occur in every other beat.
- Trigeminy: PVCs that occur with every third beat.
- Rate: Variable.
- Rhythm: Regular except for the PVC.
- P wave: Absent.
- PR interval: None.
- QRS complex: >0.12 seconds.

FIGURE 8–28 Premature Ventricular Contraction (PVC)



Example

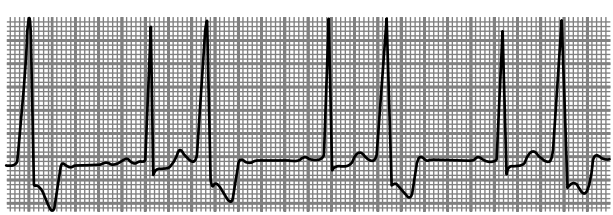
FIGURE 8–29 PVC



iii. **Ventricular bigeminy**

- Bigeminy: PVC followed by a normal QRS complex
  - Rate: 60 to 100 beats/minute
  - Rhythm: Irregular
  - P wave: Normal
  - PR interval: Normal
  - QRS complex: Normal QRS complex followed by a wide QRS complex
  - Etiology: Electrolyte disturbance, hypoxia, medication toxicity, acute MI

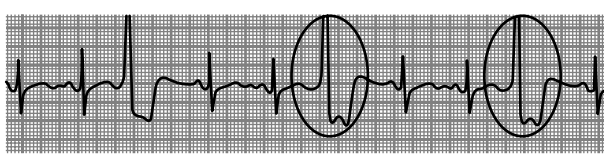
FIGURE 8–30



iv. **Ventricular trigeminy**

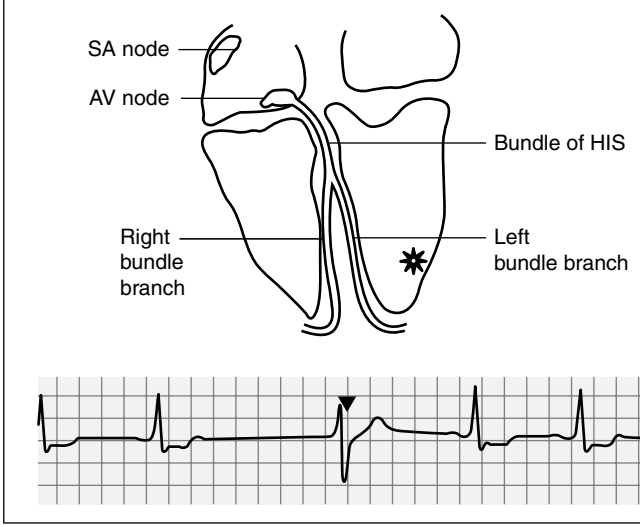
- Trigeminy: PVC followed by a two normal QRS complexes
  - Rate: 60 to 100 beats/minute
  - Rhythm: Irregular
  - P wave: Normal
  - PR interval: Normal
  - QRS complex: Normal QRS complex followed by a wide QRS complex
  - Etiology: Electrolyte disturbance, hypoxia, medication toxicity, acute MI

FIGURE 8–31



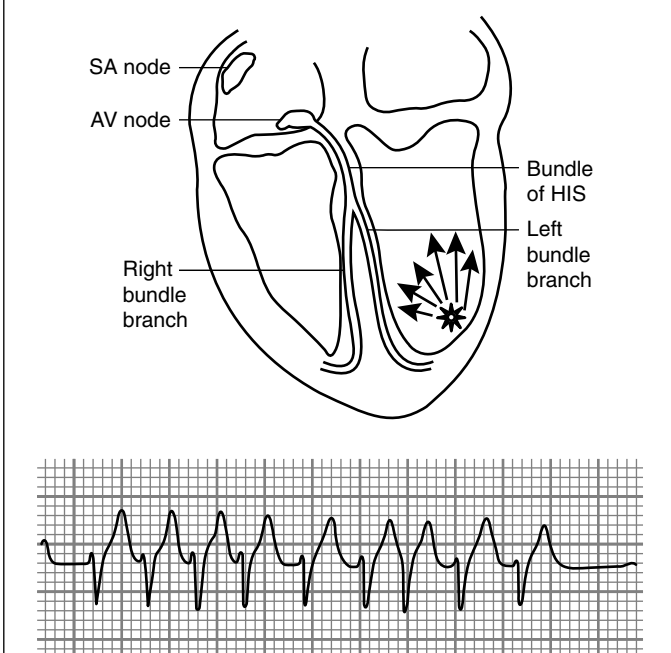
v. **Ventricular escape beat**
   - Rate: N/A.
   - Rhythm: Beat occurs later than expected.
   - P wave: Absent.
   - PR interval: N/A.
   - QRS complex: ≥0.12 seconds.

FIGURE 8–32 Ventricular Escape Beat



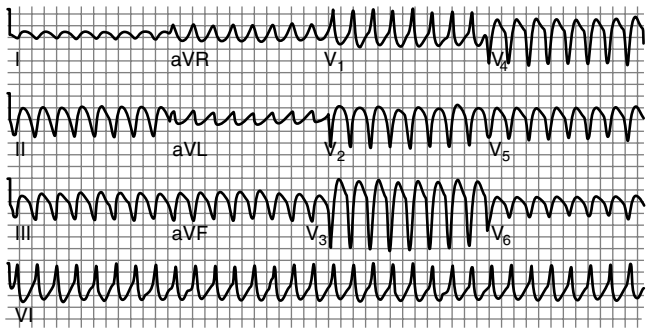
vi. **Ventricular tachycardia (VT)**
- Rate: 150 to 250 beats/minute
- Rhythm: Regular
- P wave: Absent or inverted and is not associated with QRS complex
- PR interval: N/A
- QRS complex: ≥0.12 seconds (wide)
- Etiology: MI, cardiomyopathy, CHF, hypokalemia, hypomagnesemia, medication toxicity, reperfusion following thrombolytic therapy

FIGURE 8–33 Ventricular Tachycardia



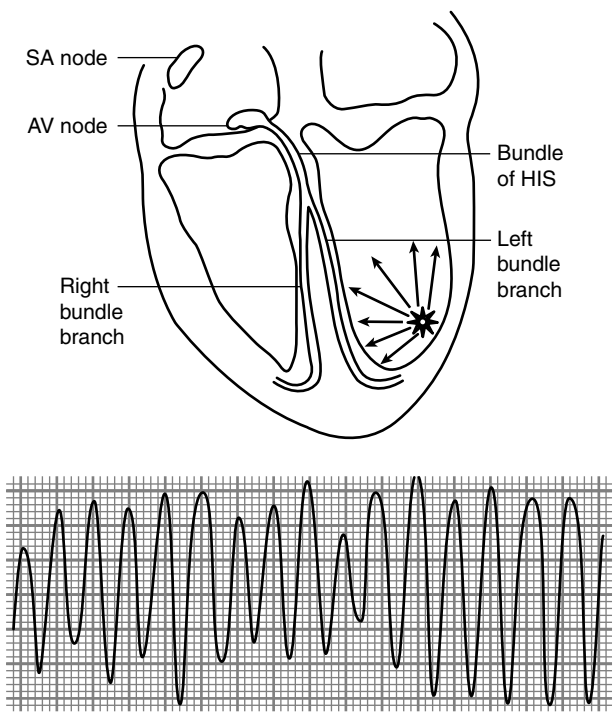
Example

FIGURE 8–34 V. Tach



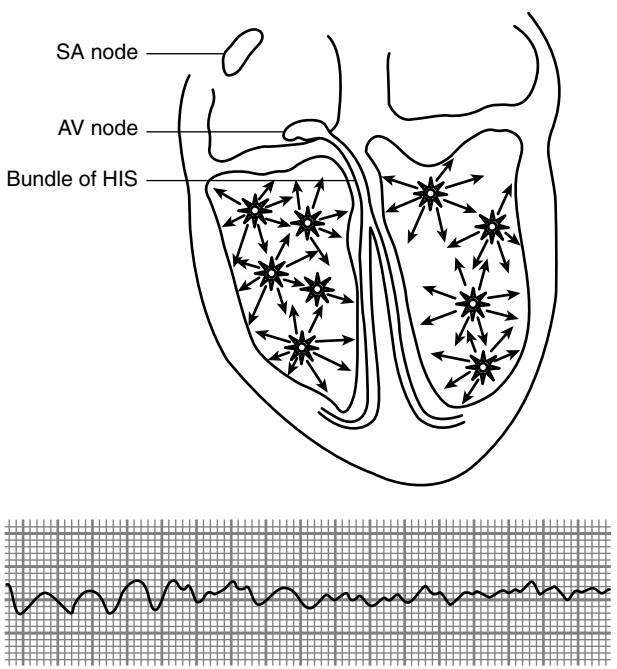
vii. **Ventricular flutter**
- Rate: 250 to 350 beats/minute
- P wave: Absent
- PR interval: N/A

FIGURE 8–35 Ventricular Flutter



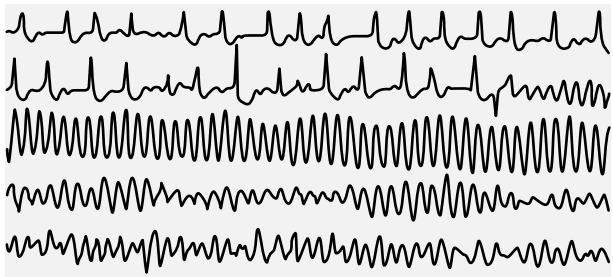
viii. **Ventricular fibrillation (V. Fib)**
- Rate: ≥300 beats/minute
- Rhythm: Irregular
- P wave: Unrecognized
- PR interval: N/A
- QRS complex: Fibrillatory waves
- Etiology: Coronary artery disease, MI, cardiomyopathy, cardiac trauma, medication toxicity, hypoxemia, electrolyte imbalance

FIGURE 8–36 Ventricular Fibrillation (V. Fib)



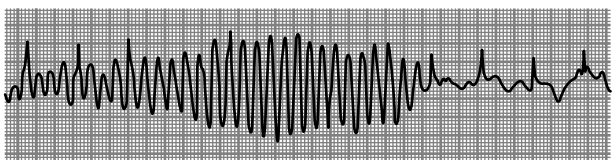
Example

FIGURE 8–37 V. Fib



ix. **Torsades de pointes:** Usually associated with electrolyte abnormalities or medications that may excessively prolong the QT interval

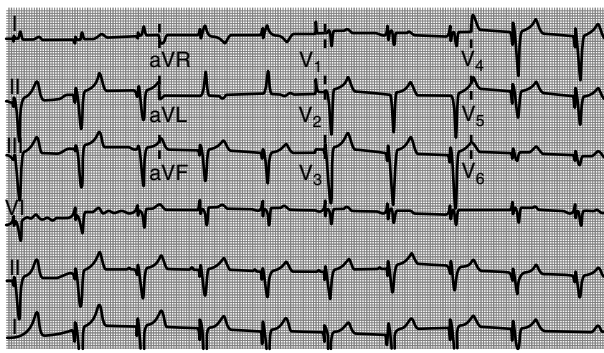
FIGURE 8–38 Torsades de Pointes



C PACED RHYTHM

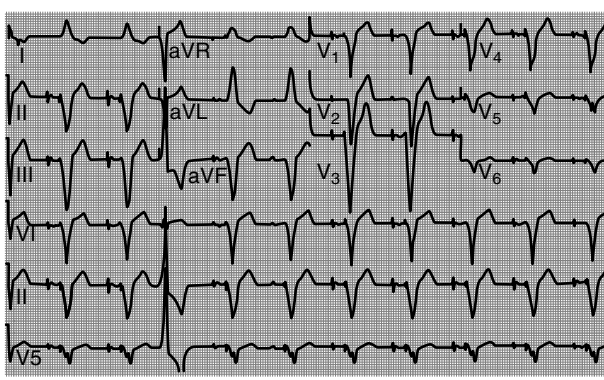
i. **Ventricular demand pacemaker**

FIGURE 8–39



ii. **Dual chamber pacemaker**

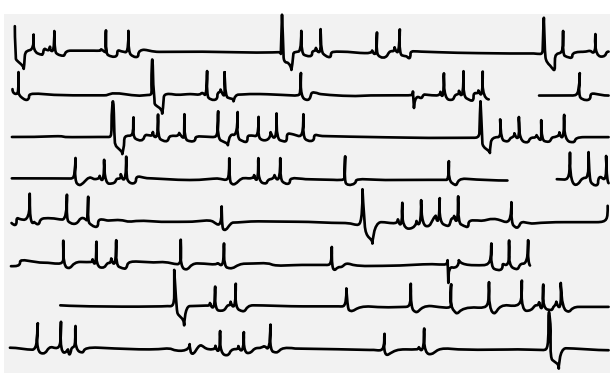
FIGURE 8–40



D MISCELLANEOUS

i. **Sick sinus syndrome (SSS),** also known as tachycardia-bradycardia syndrome
   - Rate: Variable
   - Rhythm: Regular or irregular
   - P wave: Normal
   - PR interval: Normal
   - QRS complex: Normal
   - Etiology: Damage to conduction system
     - Cardiomyopathies, sarcoidosis, amyloidosis, Chagas disease
     - SSS worsened by following medications:
       - Digitalis
       - Calcium channel blocker
       - Beta blocker
       - Sympathomimetics

FIGURE 8–41

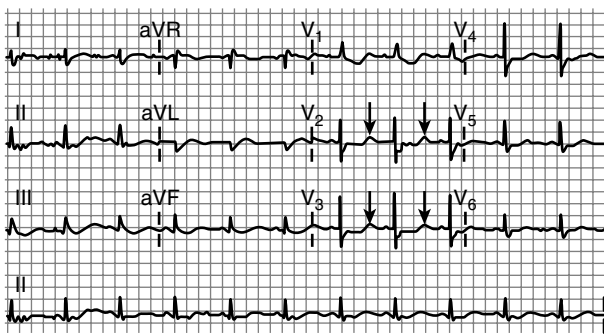


9
Electrolyte and Drug Effects

A HYPOKALEMIA

i. Prolonged PR interval
ii. T-wave flattening
iii. Prominent U waves displayed by arrows

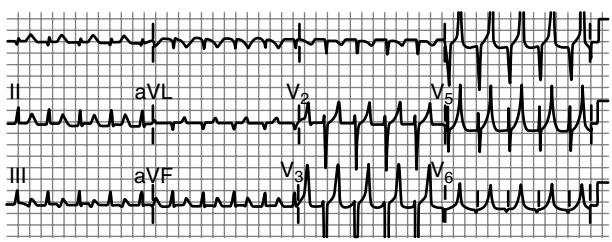
FIGURE 9–1



B HYPERKALEMIA

i. K+ level: 5.5 to 6.5 meq → tall peaked T waves, more prominent in V_3 to V_5
ii. K+ level: 6.5 to 7.5 meq → flattening of P wave and QRS widening
iii. K+ level: >7.5 meq → sinus arrest and possible sine wave pattern due to marked intraventricular conduction delay

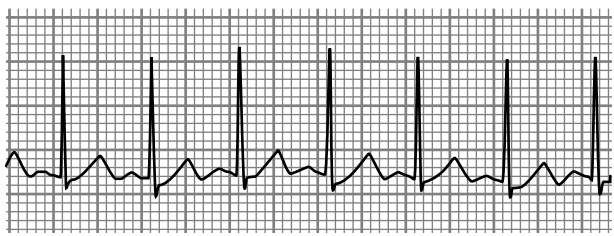
FIGURE 9–2 Hyperkalemia



C HYPOCALCEMIA

i. QT prolongation

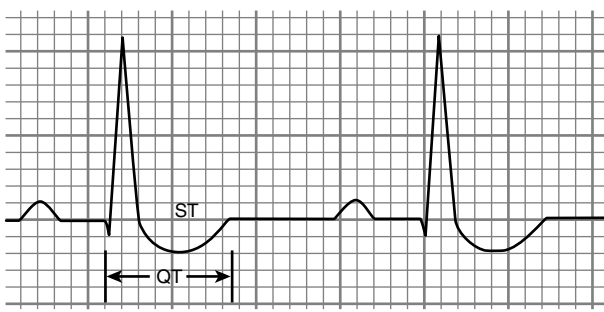
FIGURE 9–3



D DIGITALIS EFFECT

i. Commonly seen in use of digitalis and not with digitalis toxicity
ii. Prolonged PR interval
iii. Depressed and concave (scooped) ST segment: Most prominent in I, II, aVF, and V_2 to V_6

FIGURE 9–4 Digitalis Toxicity



Note: Digitalis toxicity

iv. Induces arrhythmias such as paroxysmal atrial tachycardia (PAT) with block, atrial fibrillation (A. Fib) with complete heart block, accelerated junctional rhythm

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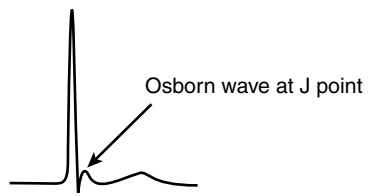
10
Other Conditions

OUTLINE	
A Hypothermia	92
B Pulmonary Embolism	93
C Pericarditis	94
D Pericardial Effusion	95

A HYPOTHERMIA

i. J wave or Osborne wave: Noted immediately after QRS complex, common in lead I.
ii. J wave disappears after warming of body temperature.

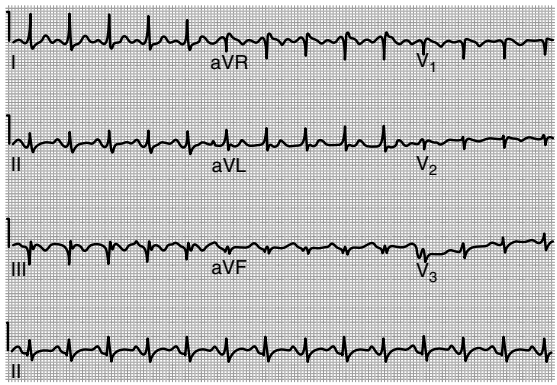
FIGURE 10–1



B PULMONARY EMBOLISM

i. Prominent S wave in lead I
ii. Q wave in lead III
iii. T wave inversion in lead III
iv. Note: Most commonly seen rhythm in pulmonary embolism in sinus tachycardia

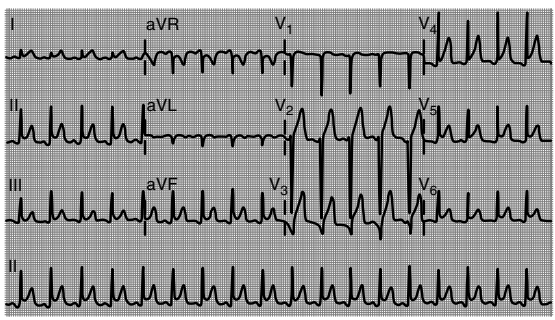
FIGURE 10–2 Pulmonary Embolism



C PERICARDITIS

i. ST segment elevation in leads I, II, aVL, aVF, V_2 to V_6.
ii. A clue that the EKG may be pericarditis is early PR depression and ST segments return to normal before T waves invert.

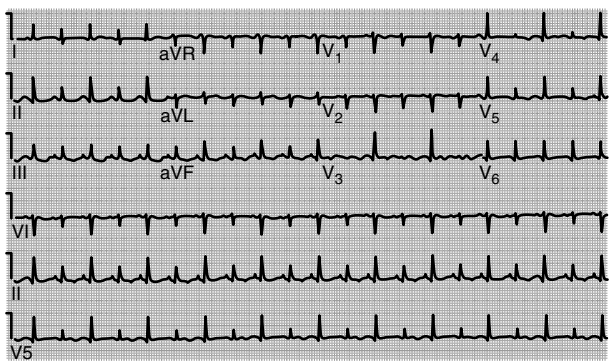
FIGURE 10–3



D PERICARDIAL EFFUSION

i. Electrical alternans noted on EKG.
ii. The amplitude (height) of the R wave alternately varies in every other beat.

FIGURE 10–4 Pericardial Effusion



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11
Cardiac Testing

A CARDIAC STRESS TEST

i. **Pretest probability for coronary artery disease (CAD)**

TABLE 11–1 Pretest Probability for Coronary Artery Disease: Male

Condition	30-39 yrs	40-49 yrs	50-59 yrs	60-69 yrs
Classic angina pectoris	Intermediate	High	High	High
Atypical angina pectoris	Intermediate	Intermediate	Intermediate	Intermediate
Nonanginal chest pain	Low	Intermediate	Intermediate	Intermediate
Asymptomatic	Very low	Low	Low	Low

TABLE 11–2 Pretest Probability for Coronary Artery Disease: Female

Condition	30-39 yrs	40-49 yrs	50-59 yrs	60-69 yrs
Classic angina pectoris	Intermediate	Intermediate	Intermediate	High
Atypical angina pectoris	Very low	Low	Intermediate	Intermediate
Nonanginal chest pain	Very low	Very low	Low	Intermediate
Asymptomatic	Very low	Very low	Very low	Low

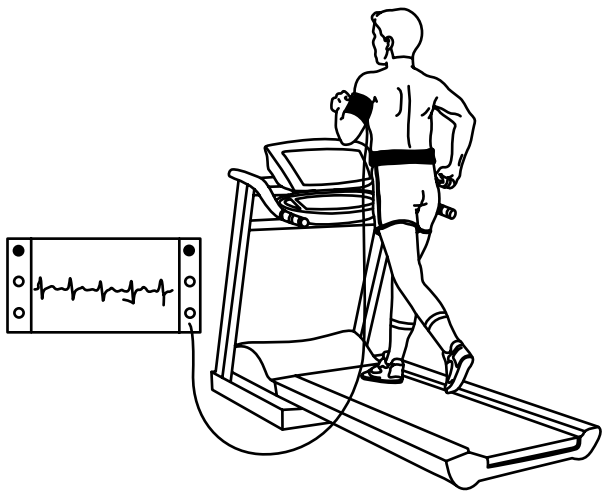
Source: Gibbons RJ, Balady GJ, Beasley JW, et al. ACC/AHA guidelines for exercise testing: Executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). *Circulation*. 1997; 96:345–354.

- High: >90% probability of CAD
- Intermediate: 10% to 90% probability of CAD
- Low: <10% probability of CAD
- Very low: <5% probability of CAD

ii. **Coronary artery disease and cardiac testing modalities**
- Treadmill exercise testing
- Cardiac nuclear imaging
- Cardiac angiography
- Cardiac echo

B EXERCISE STRESS TEST

FIGURE 11–1 Exercise Treadmill Testing (ETT)



i. **Class types: ACC/AHA classification**

Class I: Agreement/evidence of a condition that a procedure or treatment is useful and effective.

Class II: Discrepancy/conflicting evidence of a condition that a procedure or treatment is useful and effective.

- Class IIA: Data/opinion is in support of usefulness/efficacy
- Class IIB: Data/opinion is less well established to support usefulness/efficacy

Class III: Agreement/evidence of a condition that a procedure or treatment is not useful or effective. It may even be harmful.

ii. **Indications for exercise testing for detection of coronary artery disease**

Class I:	Individual with intermediate pretest probability of CAD
Class IIA:	Individual with vasospastic angina
Class IIB:	Individual with high or low pretest probability
Class III:	Individual with ◦ Wolf-Parkinson-White syndrome ◦ Paced ventricular rhythm ◦ >1 mm of resting S-T depression ◦ Complete left bundle branch block

iii. **Indications for ETT for risk stratification in patients with known CAD**

Class I:	Initial testing Change in clinical status post-revascularization (combine with cardiac imaging)
Class IIA:	None
Class IIB:	Stable symptoms and periodic monitoring to guide treatment

iv. **Indications for ETT post-myocardial infarction**

Class I:	Submaximal stress test 4 to 7 days post-uncomplicated myocardial infarction prior to hospital discharge for prognosis, activity prescription, or evaluation of medical treatment
Class II:	Post-revascularization for activity prescription or periodic monitoring of high-risk patients
Class III:	Routine monitoring after revascularization

v. **Indications in asymptomatic patients with no known CAD**

Class I:	None
Class IIB:	Multiple risk factors, diabetes
Class III:	Routine screening

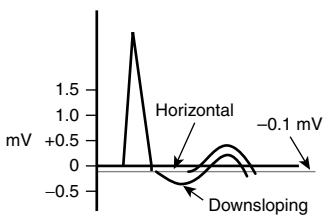
vi. **Disadvantages**

Specificity decreases with

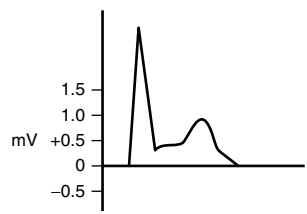
- Marked baseline ST abnormalities
- Use of dioxin
- Left bundle branch block
- Pacemaker

vii. **Baseline EKG changes that may lead to difficulty in interpreting stress test**
- ST depression or elevation (≥1 mm)
- Ventricular strain pattern (secondary from right or left ventricular hypertrophy)
- T-wave inversion (from strain or previous injury)

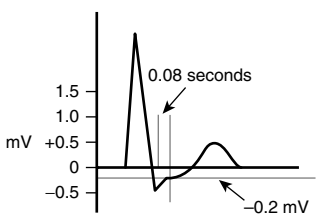
Exercise Stress Test



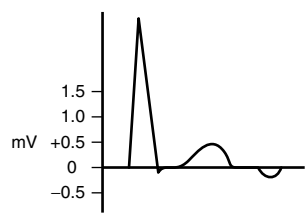
Horizontal downsloping ST-segment depression, ≥ 0.1 mV



ST-segment elevation ≥ 0.1 mV



Upsloping ST-segment depression, ≥ 0.2 mV, 0.08 seconds from the J point



U-wave inversion

(*continued*)

Exercise Stress Test (*Continued*)

Electrocardiographic (EKG) findings suggestive of a positive exercise stress test. In addition to the EKG findings depicted here, the occurrence of frequent premature ventricular contractions (PVCs), multifocal PVCs, or ventricular tachycardia at mild exercise (<70% of maximal heart rate) is suggestive of an exercise stress test positive for myocardial ischemia.

Reproduced with permission from Darrow MD. Ordering and understanding the exercise stress test. *Am Fam Physician.* 1999;59:401-410.

Exercise Testing Contraindications

Absolute

i. Acute aortic dissection
ii. Acute myocardial infarction within 2 days
iii. Acute myocarditis
iv. Acute pericarditis
v. Pulmonary embolism/infarction
vi. Recent lower extremity DVT
vii. Severe symptomatic aortic stenosis
viii. Uncontrolled heart failure
ix. Uncontrolled symptomatic cardiac arrhythmia
x. Unstable angina
xi. Third-degree AV block

Relative

i. Stenotic valvular disease
ii. Electrolyte imbalance
iii. Uncontrolled hypertension
iv. Tachycardia or bradycardia
v. Hypertrophic cardiomyopathy
vi. Inability to exercise
vii. High grade of AV block

Stress Test Protocols

i. There are several protocols in existence for exercise stress testing.
ii. Majority are aimed at obtaining 85% to 100% of age-predicted heart rate.
iii. Maximum predicted heart rate = 220 − age (years).
iv. Metabolic equivalents of tasks (METs) = actual metabolic expense during exercise (resting O_2 consumption [VO_2]) = 3.5 mL/kg/min

Functional capacity in METs

Poor:	<4
Moderate:	4 to 7
Good:	7 to 10
Excellent:	>10

i. **Bruce protocol:** 8 stages, each stage is 3 minutes, substantial increase in incline and speed.
Test is considered correct if ≥6 METs have been obtained. (Note: The test is usually continued even if 6 METs have been reached.)

Certainty of MET levels achieved:

Less than or equal to 5 METs = Poor prognosis in individual of age ≤65 years
10 METs = Good prognosis with medical therapy
Less than or equal to 13 METs = Good prognosis despite abnormal exercise test

ii. **Report of exercise should include the following in its interpretation:**
- Baseline EKG
- EKG changes noted during the test
- Blood pressure during the test
- Arrhythmia or abnormal beats noted during the test
- Symptoms observed during the test
- Approximate exercise capacity of the individual in METs (most important prognostic indicator)
- Whether test was ended prematurely and reason

iii. **Conclusion of the report should include**
- Positive
- Negative
- Equivocal
- Nondiagnostic
- Goal achieved (maximal, submaximal)

iv. **Clinical findings for positive stress test**
- Hypotension
- Angina
- S3, S4, or murmur during exercise

Positive stress test

Less than or equal to 1-mm ST elevation in leads without prior Q waves
Less than or equal to 1-mm horizontal or downsloping ST depression
(Note: All ST segment changes have to occur in at least two consecutive leads and three consecutive beats per lead.)
Less than or equal to 1.5-mm upsloping ST depression
T inversion
U wave
Ventricular tachycardia (VT)

v. **False-positive results on exercise stress testing**
- Coronary artery spasm
- Left bundle branch block
- Cardiomyopathy
- Left ventricular hypertrophy with strain at baseline
- Use of digitalis or antidepressants

C CARDIAC STRESS ECHOCARDIOGRAPHY

i. Is performed to make observation of the wall motion at rest and with stress.
ii. Baseline echocardiography is performed to rule out any abnormalities at rest.

iii. Increases the sensitivity and specificity of the exercise stress testing alone.
iv. If individual is unable to exercise, a pharmacological agent can be useful.

i. **Indications:**
   - Assessment of ventricular function
   - Chamber size
   - Wall thickness
   - Valvular function

ii. **Diagnosis of coronary artery disease in presence of EKG abnormalities that may make stress test uninterruptible such as**
   - Left bundle branch block (Note: Specificity for detecting ischemia decreases in patients with LBBB.)
   - Left ventricular hypertrophy
   - Early repolarization or conduction abnormalities
   - Determine extent and location of ischemia

iii. **Restrictive factors:** Individual with COPD and obesity.
iv. **Disadvantages:** Interpretation is very subjective especially if there is a baseline wall motion abnormality.

D PHARMACOLOGICAL STRESS TEST

i. **Indications:**
   - Individual is unable to exercise.
   - Abnormal baseline EKG such as LBBB (Note: Should be combined with imaging component).

ii. **Disadvantages:** Specificity is reduced in individual with right ventricular pacemaker.

i. **Dobutamine**
   - Useful alternative to adenosine and dipyridamole in individuals with conditions associated with bronchospasm (asthma, COPD, etc)
   - Useful in individual taking Aggrenox or Persantine
   - Useful in individual with severe carotid stenosis

- Increases heart rate and blood pressure
- Enhances contractility
- Half-life is about 2 minutes
  - Contraindications
    - Ventricular tachycardia
    - Myocardial infarction within past 3 days
    - Unstable angina
    - Severe left ventricular outflow obstruction
    - Aortic aneurysm
    - Aortic dissection
    - Systemic hypertension
  - Discontinuation of the dobutamine infusion is as follows:
    - BP: 230/130
    - SBP: <80
    - Patient becomes symptomatic such as chest pain, SOB
    - Less than 2 mm of ST depression from baseline
    - Ventricular tachycardia
    - Supraventricular tachycardia
    - Atrial fibrillation
    - AV block (2:1, complete)
    - Attainment of >85% of predicted heart rate
  - Side effects: Palpitation, chest pain, nausea, anxiety, arrhythmias (SVT/VT/VF), tremors

ii. **Dipyridamole**

- Onset is about 3 minute after infusion for 4 minutes, peaks at approximate 7 to 12 minutes.
- Half-life is >20 minutes.
- Commonly used agent for performing nuclear imaging.
- It may increase the efficacy of antihypertensive medications.
- Individual should avoid caffeine-containing product 24 hours prior to test.
- Individual should avoid theophylline-containing product 72 hours prior to test.
  - Contraindications
    - Myocardial infarction within 72 hours
    - Severe lung disease or asthma

        - Severe left ventricle systolic dysfunction
        - Second- or third-degree heart block
        - Baseline hypotension
    - Side effects: Chest pain, dizziness, headache, shortness of breath, flushing myocardial infarction, stroke
    - Antidote: Aminophylline

iii. **Adenosine**

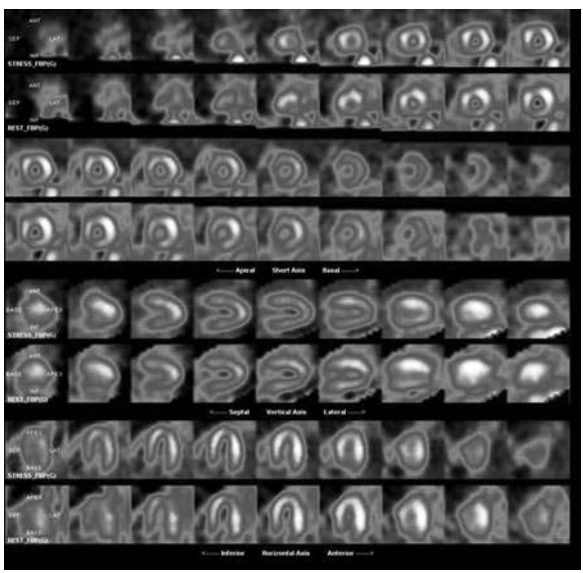
- Rapid onset (seconds)
- Short half-life for elimination (few seconds).
- Most commonly used agent for performing nuclear imaging.
- Very powerful vasodilator of coronaries; requires controlled infusion.
- Individual should avoid caffeine-containing product 24 hours prior to test.
- Individual should avoid theophylline-containing product 72 hours prior to test.
    - Indications
        - Left bundle branch block
        - Paced rhythm
        - Wolff-Parkinson-White syndrome
    - Contraindications
        - High-grade AV block
        - Condition that can cause bronchospasm (asthma, COPD, etc)
        - Sick sinus syndrome
        - Hypotension
        - Individual on Aggrenox/Persantine
        - Caffeine ingestion in past 24 hours (blocks adenosine receptors)
    - Side effects: Chest pain, flushing, shortness of breath, nausea, and headache; arrhythmias (VT/VF)

iv. **Arbutamine**

- Is a strong beta-adrenergic agonist and mild alpha-sympathomimetic agent.
- It increased heart rate and contractility of the myocardium.
- High cost limits its use.

E NUCLEAR IMAGING

FIGURE 11–2 Nuclear Imaging



i. **Indications for nuclear imaging**
- Equivocal stress test or intermediate probability.
- Evaluation of viability of myocardium after myocardial infarction or revascularization.
- Evaluation of preoperative risk.
- Evaluate recurrent symptoms after coronary artery bypass grafting (CABG) or balloon angioplasty (PTCA).
- Chest pain in association with bundle branch block, early repolarization, nonspecific ST changes, post–myocardial infarction, pre-excitation.

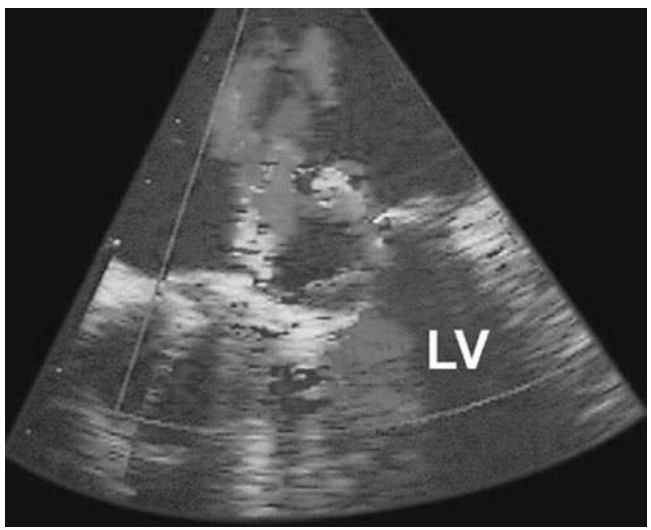
ii. **Radioisotopes**
- Technetium-m99
- Thallium-201

iii. **Pharmaceutical agents**
- Tetrofosmin
- Sestamibi
  - Technetium-m99 sestamibi (Cardiolite)
    - It is deposited into mitochondria and provides enhanced image quality.
    - Superior image acquired in female and obese patient.
  - Thallium
    - Replaces K^+ in the cell
    - Longer half-life than technetium (Note: Areas which are ischemic/necrotic take up less/no blood flow. Thus there is lack of or less uptake of tracer compared to normally perfused areas.)

iv. **Disadvantages:** Artifact may be due to soft tissue like breast.

F CARDIAC ECHOCARDIOGRAPHY

FIGURE 11–3 Ecocardiogram



i. **Echocardiography** is an imaging modality used for assessing cardiac anatomy and function by using high-frequency sound waves.

ii. **There are two routine ways of performing the cardiac echocardiogram.**
- Transthoracic echocardiogram
- Transesophageal echocardiogram

iii. **There are three modalities for the echocardiogram.**
- "M"—mode echocardiography: It provides single-dimension images that allow measurement of the heart chambers. Displays one-dimensional image.

- Two-dimensional echocardiography: It provides cross-sectional slices of the heart. Displays two-dimensional image.
- Color flow echocardiography: It provides visualization of the blood flow across valves and congenital anomalies.

iv. **Indications:**
- Aortic aneurysm
- Atrial function
- Cardiomyopathy
- Congenital heart disease
- Endocarditis
- Great vessel disease
- Heart failure assessment
- Hypotension
- Intracardiac thrombus
- Penetrating/blunt trauma
- Pericarditis
- Unexplained syncope
- Valvular dysfunction/valvular disease
- Ventricular function

v. **Specific clinical indications for transesophageal echocardiogram:**
- Embolism of cardiac source
- Endocarditis
- Prosthetic heart valve function
- Native valvular disease
- Aortic dissection
- Aortic aneurysm
- Intracardiac tumor, mass, or thrombus
- Congenital heart disease

G CARDIAC CATHETERIZATION

i. **Technique:** A catheter is inserted from groin or arm into the heart and then eventually into the coronary arteries and appropriate area.

FIGURE 11–4

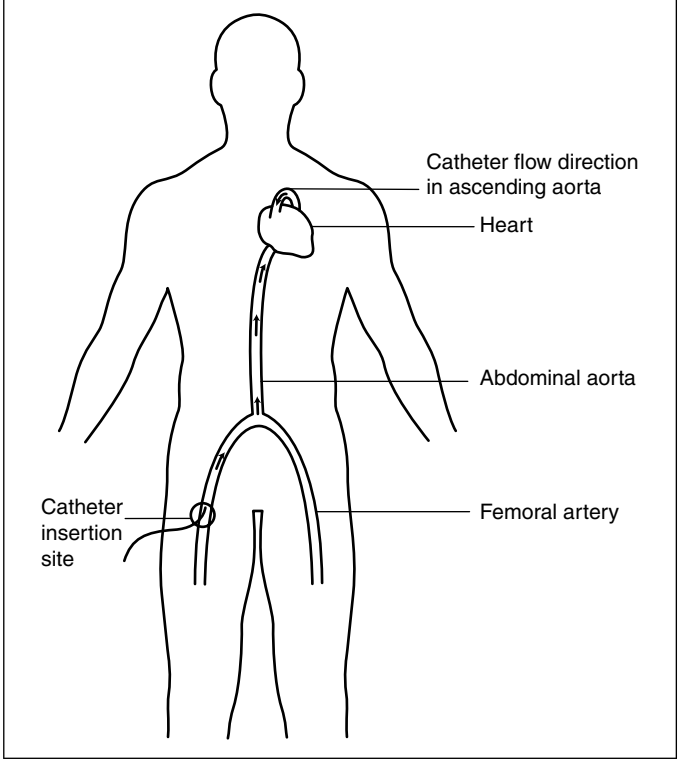
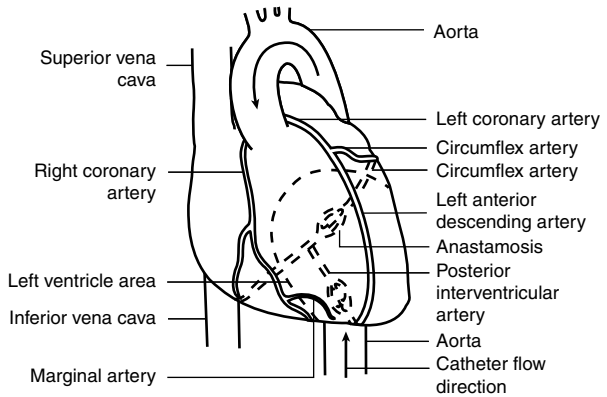


FIGURE 11–5



ii. **Indications:**
- Stenting and dilatation of the coronary arteries in acute myocardial infarction
- Stenting or dilatation of the coronary arteries to relieve symptoms in chronic coronary disease patients
- Diagnosis of coronary artery disease
- Valvuloplasty
- Measure pressure in the heart and aorta
- Cardiac biopsies
- Visualization of the atrium and ventricles
- Electrophysiology which includes ablation of the aberrant pathways

iii. **Complications:**
- Allergic reaction to contrast medium
- Angina
- Myocardial infarction

- Arrhythmia
- Hemorrhage from catheter insertion site
- Pericardial tamponade
- Renal damage from contrast medium
- Stroke

H HOLTER MONITORING

i. It is a device designed to monitor and store electrical activities of the heart (arrhythmia, blocks, etc).
ii. The device can store activities for ≥24 hours.
iii. The Holter device is connected to the chest via series of wires.

I ELECTROPHYSIOLOGIC STUDY

It involves series of tests to help determine the location and type of electrical activity, as well as response to treatment.

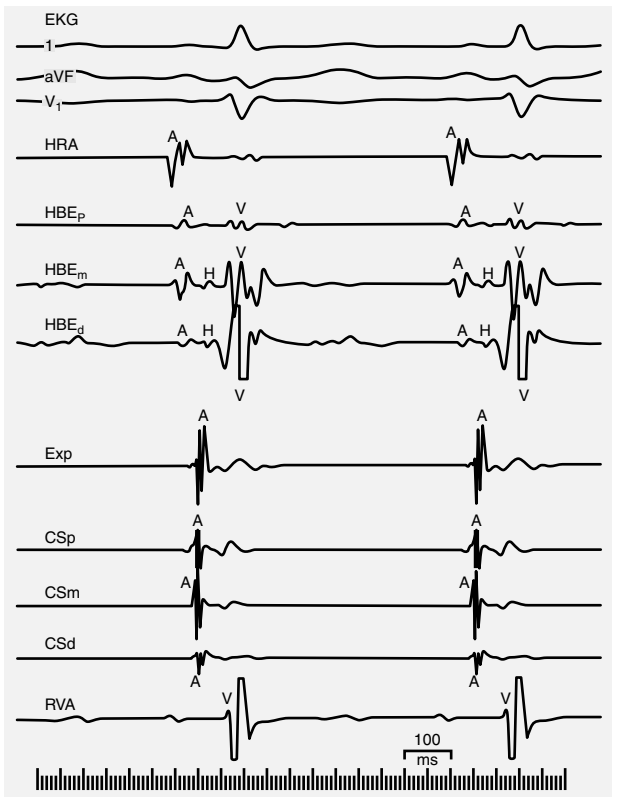
i. **Technique:**
After sedating the patient, "multiple," specialized catheters are inserted via fluoroscopy from groin or neck into specific areas of the heart through which heart rhythm is recorded and the pathways of arrhythmias are determined through small amounts of delivered electricity. The study takes several hours for completion.

ii. **It is a study performed to determine and manage the following conditions:**
- Paroxysmal supraventricular tachycardia
- Ventricular tachycardia
- Atrial flutter
- Risk of cardiac arrest
- Bradycardia
- Syncope
- Effectiveness of medication to control arrhythmia
- Assess the need for an implantable device (pacer, ICD)

Electrophysiologic Study (EPS): Normal Intracardiac Electrograms

Three surface EKG leads: I, aVF, and V_1

FIGURE 11–6



i. **EPS study abbreviations**
- HRA = High right atrium
- A = Atrium
- HBE = Bundle of HIS
- p = Proximal
- m = Mid
- d = Distal
- exp = Exploratory catheter
- CS = Coronary sinus
- RVA = Right ventricular apex
- V = Ventricle

ii. **Complications associated with EPS**
- **Secondary from procedure**
  - Bleeding
  - Infection
  - Pain
  - Allergic reaction
  - Thrombophlebitis
  - Aortic dissection
  - Stroke/TIA
  - Coronary sinus perforation
  - Cardiac tamponade
- **Secondary from programmed cardiac stimulation**
  - Cardiac arrhythmia
  - Myocardiac infarction
  - Bundle branch block
- **Secondary from transcatheter ablation**
  - Third-degree heart block
  - Thromboembolism
  - Cardiac arrhythmia
  - Pericarditis
  - Phrenic nerve paralysis
  - Radiation skin burn
  - Coronary artery thrombosis
  - Myocardial infarction
  - Cardiac perforation from excessive radiation to various cardiac structures

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12
Cardiac Pacemaker

OUTLINE	
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Cardiac pacemaker is a device that supplies electrical stimuli to cause cardiac contractions when there is defect in the intrinsic cardiac electrical activity. It functions by detecting intrinsic cardiac electric potentials. If it senses the potentials are too infrequent or absent, it provides electrical impulses to the heart thus stimulating myocardial contraction.

A INDICATIONS FOR PERMANENT CARDIAC PACEMAKER: ACC/AHA CLASSIFICATION

Class I: Agreement/evidence of a permanent pacing is definitely beneficial, useful, and effective.

Class II: Discrepancy/conflicting evidence of permanent pacing is useful and effective.

- Class IIA: Data/opinion is in support of usefulness/efficacy.
- Class IIB: Data/opinion is less well established to support usefulness/efficacy.

Class III: Agreement/evidence of a permanent pacing is not useful or effective. It may even be harmful.

B INDICATIONS FOR PERMANENT CARDIAC PACEMAKER IMPLANTATION

Class I:

- Symptomatic bradycardia (usually <40 beats per minute).
- Complete AV block (third degree).
- Advanced second-degree AV block.
- Second-degree AV block in the HIS –Purkinje system with bilateral bundle branch block.
- Symptomatic Mobitz I or Mobitz II second-degree AV block.
- Second-degree AV block that is symptomatic and persistent.
- Mobitz II second-degree AV block associated with a wide QRS or chronic bifascicular block regardless of symptoms.

- Transient complex infranodal AV block with associated bundle branch block.
- Second- or third-degree block with associated myotonic muscular dystrophy, peroneal muscular atrophy, Kearns-Sayre syndrome, or Erb dystrophy.
- Individual with neurocardiogenic syncope: Syncope and >3 second of asystole following minimal carotid sinus message.
- Syncope and >3 seconds of asystole or escape rhythm <40 beats per minute in an awake patient.
- Wide QRS escape rhythm.
- Complex ventricular ectopy.
- Sustained ventricular arrhythmia that is pause dependent.
- Less than or equal to 1 year of age: Ventricular rate is <55 beats per minute or <70 beats per minute and has associated congenital heart disease regardless of the symptom.
- Long QT syndrome due to unknown etiology.
- NYHA III or refractory heart failure (EF <35%) after optimization of medical treatment and 3 months post-revascularization, and evidence of ventricular dyssynchrony—indication for biventricular pacemaker implantation.

Class II:

- Sinus bradycardia with no clear association between symptoms and bradycardia (<40 beats per minute).
- Sinus node dysfunction with unknown etiology of syncope.
- Mobitz II second-degree AV block with symptomatic bradycardia.
- First-degree AV block with hemodynamic compromise.
- Asymptomatic second- or third-degree AV block post-MI at the level of AV node.
- Bifascicular or trifascicular block with syncope that can be contributed to transient high-grade AV block.
- Syncope of unknown etiology where major abnormalities of sinus node function are discovered in electrophysiologic (EP) study.
- Recurrent syncope of unknown etiology with abnormal response to carotid sinus message, but syncope is not due to carotid sinus message.

- Recurrent neurocardiogenic syncope with bradycardia (spontaneously or noted during tilt-table testing).
- Symptomatic hypertrophic cardiomyopathy despite optimal medication and significant left ventricular outflow tract obstruction at rest or during exercise.

Class III:

- Syncope of unknown etiology.
- Sinus bradycardia without significant symptoms.
- Sinoatrial block or sinus arrest without significant symptoms.
- Transient ventricular pace.
- Asymptomatic bradycardia in sleep.
- Asymptomatic second-degree Mobitz I AV block (Wenckebach).
- Intermittent AV block.
- Right bundle branch block with left axis deviation without symptom.
- Reversible AV block (secondary for conditions such as sleep apnea, Lyme disease, enhanced vagal tone, post-operative medications (beta blocker, diltiazem, verapamil).
- Long QT due to reversible etiology.
- Torsades de pointes due to reversible etiology.

C TYPES OF PACEMAKERS

i. **Single chamber:** Only one wire is implanted into the atrium or ventricle.
ii. **Dual chamber:** Wires are implanted into two chambers (atrium and ventricle).
iii. **Rate responsive:** Is sensor sensitive to person's physical activity.
iv. **Biventricular pacemaker:** Three leads are implanted. One lead into the atrium, one into the right ventricle, and one into the coronary sinus which stimulates the left ventricle.

D PACING CODES

First letter:	Chamber paced
Second letter:	Chamber sensed
Third letter:	Chamber response to sensing
Fourth letter:	Programmability
Fifth letter:	Antitachycardia function

i. **Chamber paced**
A = Atrium
V = Ventricle
D = Dual (both chambers)
O = None

---

ii. **Chamber sensed**
A = Atrium
V = Ventricle
D = Dual (both chambers)
O = None

---

iii. **Response to sensing**
T = Triggered pacing
I = Inhibited pacing
D = Dual (T + I)
O = None

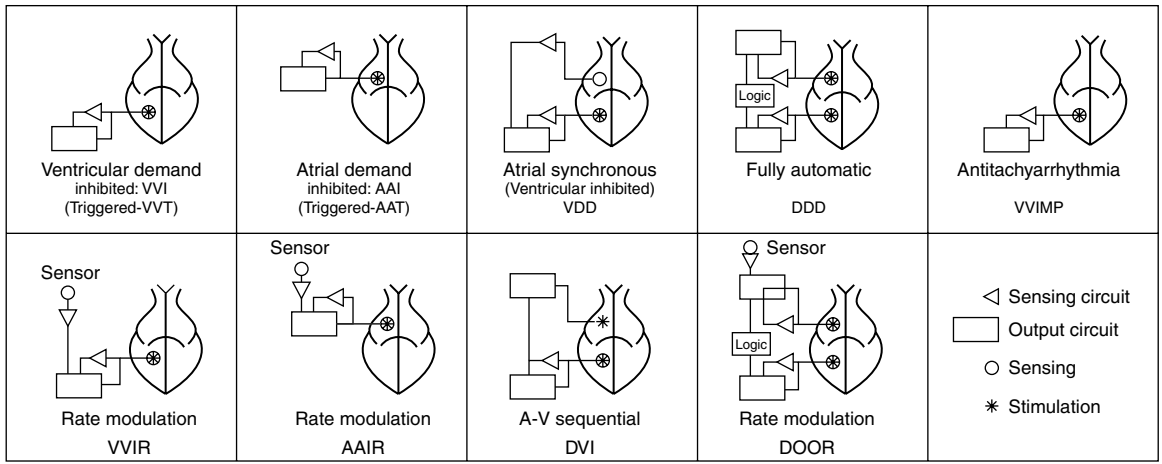
---

iv. **Programmable function**
P = Programmable rate and/or output
M = Multiprogrammability of rate, output, sensitivity, and more
C = Communicating function (telemetry)
R = Rate adaptive
O = None

---

v. **Antitachycardia function**
P = Overdrive pacing
S = Shock
D = Dual
O = None

FIGURE 12–1 Pacer Classification



The NASPE/BPEG genetic pacemaker code for antibradyarrhythm and adaptive-rate pacing and antitachyarrhythmia devices. *PACE.* 1987; 10:794–799.

13
Implantable Cardioverter Defibrillator

OUTLINE	
A Implantable Cardioverter Defibrillator Device	126

A IMPLANTABLE CARDIOVERTER DEFIBRILLATOR DEVICE

The implantable cardioverter defibrillator (ICD) is a device for treatment of cardiac tachyarrhythmia.

Newer ICDs have the functionality to manage bradycardia, tachycardia, low-energy cardioversion, high-energy defibrillation, and electrogram storage. These devices have capacity to multiprogram and respond differently to different rhythm.

i. **ICD device consists of the following four elements:**
- Sensing electrodes
- Defibrillation electrodes
- Pulse generator
- Backup bradycardia pacing in the event of post-defibrillation bradycardia

ii. **Indications:**
- Secondary prevention in an individual with cardiac arrest due to ventricular fibrillation or ventricular tachycardia that is not due to reversible cause
- Secondary prevention of individual with ≥2 episode of spontaneous sustained ventricular tachycardia in the presence of structural heart disease
- Primary prevention in individual with documented MI (at least 30 days post-MI) and impaired left ventricular systolic dysfunction (EF <30%), 1-month post-MI or 3 months post-CABG
- Primary prevention in individual with nonischemic cardiomyopathy, NYHA class II/III heart failure, and left ventricular ejection fraction ≤30%

14
Acute Cardiac Life Support (ACLS) Protocols

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C Sinus bradycardia (symptomatic)	130
D Asystole	131
E Third-degree block (symptomatic)	132
F Second-degree Mobitz type II heart block	132
G Atrial fibrillation/atrial flutter	133
H Narrow complex supraventricular tachycardia	134
I Junctional tachycardia	134
J Ectopic or multifocal atrial tachycardia	134
K Paroxysmal supraventricular tachycardia	134
L Ventricular tachycardia (stable)	135
M Ventricular fibrillation/pulseless ventricular tachycardia	136

A ACUTE CORONARY SYNDROME

i. Maintain airway, breathing, and circulation.
ii. 12-lead EKG.
iii. Intravenous (IV) normal saline to keep (venous infusion saline) open
iv. Administer nitroglycerine 0.3 to 0.4 mg sublingual, repeat in 5 minutes up to three times in total, check blood pressure (BP) between administration; avoid if BP < 100/60 mm Hg.
v. Aspirin 325 mg × 1 dose.
vi. Metoprolol 5 mg IV slow push if heart rate (HR) > 60 and systolic blood pressure (SBP) > 110.
    - May repeat second administration of 5-mg metoprolol IV push in 5 minutes if HR > 60 and SBP > 110.
    - Caution: Consult medical directives if there is evidence of asthma, emphysema, chronic obstructive pulmonary disease (COPD), or other broncho-constricting conditions. Also, if there exist cardiac blocks.
vii. Administer morphine sulfate 2 to 4 mg IV push, IO, or intranasal. May repeat another dose if no relief is obtained (maximum dose: 10 mg); hold if HR < 60 or SBP < 100.
viii. If morphine allergy, administer fentanyl 25 to 50 mcg slow IV push, IM, or intranasal.
ix. Continue monitoring for myocardial infarct and cardiac dysrhythmia.

B STEMI (ST-SEGMENT ELEVATION MYOCARDIAL INFARCT)

i. Maintain airway, breathing, and circulation.
ii. 12-lead EKG.
iii. IV normal saline TKO.
iv. Administer nitroglycerine 0.3 to 0.4-mg sublingual, repeat in 5 minutes up to three times in total, check BP between administration, avoid if BP < 100/60.
v. Aspirin 325 mg × 1 dose.
vi. Metoprolol 5-mg IV slow push if HR > 60 and SBP > 110.
    - May repeat second administration of 5-mg metoprolol IV push in 5 minutes if HR > 60 and SBP > 110.
    - Caution: *Consult medical direction* if there is evidence of asthma, emphysema, COPD, or other broncho-constricting conditions. Also if there exist cardiac blocks.
vii. Administer morphine sulfate 2-to 4-mg IV push, IO, or intranasal. May repeat another dose if no relief is obtained (maximum dose: 10 mg), hold if HR < 60 or SBP < 100; if morphine allergy, administer fentanyl 25-to 50-mcg slow IV push, IM or intranasal.
viii. Continue monitoring for myocardial infarct and cardiac dysrhythmia.
ix. Heparin 50 units/kg (maximum dose: 4000 units) slow IV push, most institutes may have their own heparin protocol.

C SINUS BRADYCARDIA (SYMPTOMATIC)

i. Maintain airway, breathing, and circulation.
ii. 12-lead EKG.
iii. Atropine 0.5-mg IV push every 3 to 5 minutes (maximum dose: 3 mg); children: 0.02 mg /kg IV push, repeat every 5 minute (maximum dose: 0.1 mg).
iv. If not responsive, then consider transcutaneous pacing; IV normal saline TKO.
v. Consider sedation for comfort, such as Versed 2 mg.

D ASYSTOLE

i. Maintain airway, breathing, and circulation,
ii. 12-lead EKG.
iii. IV normal saline TKO.
iv. Consider treating secondary causes.
v. Possible etiologies for asystole.
   - Acidosis
   - Acute myocardial infarct
   - Cardiac tamponade
   - Drug overdose
   - Hyperkalemia
   - Hypovolemia
   - Hypoxemia
   - Pulmonary embolism
   - Tension pneumothorax

vi. Epinephrine 1-mg IV push, repeat every 3 to 5 minutes.
vii. Atropine 1 mg IV, every 3 to 5 minutes (maximum dose: 0.04 mg/kg).
viii. Consider IV fluid bolus 500 cc (NS) if evidence of fluid loss.
ix. Consider bicarbonate 50-mEq IV push or 1 mEq/kg IV.

E THIRD-DEGREE BLOCK (SYMPTOMATIC)

i. Maintain airway, breathing, and circulation.
ii. 12-lead EKG.
iii. Consider transcutaneous pacemaker.

F SECOND-DEGREE MOBITZ TYPE II HEART BLOCK

i. Maintain airway, breathing, and circulation.
ii. 12-lead EKG.
- Atropine 0.5 to 1 mg.
- Transcutaneous pacing.
- Dopamine 5 to 20 μg/kg/min.
- Epinephrine 2 to 10 μg/min.
- Isoproterenol 2 to 10 μg/min.
- Prepare for transvenous pacemaker.

G ATRIAL FIBRILLATION/ATRIAL FLUTTER

i. Management if duration is <48 hours
   - Maintain airway, breathing, and circulation.
   - 12-lead EKG.
   - IV normal saline TKO.
   - Rate control:
     - If ventricular function preserved → diltiazem (or another CCB) or metoprolol (or another beta blocker).
     - If ventricular function is not preserved → diltiazem (only CCB) or digoxin or amiodarone
   - Convert to sinus rhythm
     - DC cardioversion
       or
     - If ventricular function preserved: procainamide, amiodarone, flecainide, or propafenone
     - If ventricular function is not preserved: amiodarone

ii. Management if duration is >48 hours
   - Maintain airway, breathing, and circulation.
   - 12-lead EKG.
   - IV normal saline TKO.
   - Rate control
     - If ventricular function preserved → diltiazem (or another CCB) or metoprolol (or another beta blocker)
     - If ventricular function is not preserved → diltiazem (only CCB) or digoxin or amiodarone
   - Convert to sinus rhythm
     - Start IV heparin infusion.
     - Perform transesophageal echocardiography to exclude atrial clot.
     - Then perform cardioversion within 24 hours and anticoagulate after >4 weeks.

H NARROW COMPLEX SUPRAVENTRICULAR TACHYCARDIA

I JUNCTIONAL TACHYCARDIA

i. Vagal stimulation or Adenosine
ii. EF > 40%
   - Beta blocker or
   - Calcium channel blocker (CCB) or
   - Amiodarone

iii. EF < 40%
   - Amiodarone

J ECTOPIC OR MULTIFOCAL ATRIAL TACHYCARDIA

i. Vagal stimulation or adenosine
ii. EF > 40%
   - Beta blocker or
   - Calcium channel blocker or
   - Amiodarone

iii. EF < 40%
   - Amiodarone

iv. Diltiazem

K PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

i. Vagal stimulation or adenosine
ii. EF > 40%
   - Beta blocker or
   - Calcium channel blocker or
   - Digoxin or
   - Cardioversion or

- Procainamide or
- Amiodarone or
- Sotalol

iii. EF < 40%
- Cardioversion
- Digoxin or
- Amiodarone or
- Diltiazem

L VENTRICULAR TACHYCARDIA (STABLE)

i. **Monomorphic**
- EF > 40%
  - Procainamide or
  - Sotalol or
  - Amiodarone or
  - Lidocaine
- EF < 40%
  - Amiodarone or
  - Lidocaine
  - Synchronized cardioversion

ii. **Polymorphic**
- Normal baseline QT interval
  - Beta blocker or
  - EF > 40%
  - Lidocaine or
  - Amiodarone or
  - Procainamide or
  - Sotalol or
- Normal baseline QT interval
  - Amiodarone or
  - EF < 40%
  - Lidocaine or
  - Synchronized cardioversion

- Prolonged baseline QT interval
  - Magnesium or
  - Override pacing or
  - Isoproterenol or
  - Phenytoin or
  - Lidocaine

M VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA

i. Maintain airway, breathing, and circulation
ii. Defibrillation (maximum three times) (200 J, 200-300 J, and 360 J)
iii. Epinephrine 1-mg IV push (repeat every 3-5 minutes)
or
iv. Vasopressin 40-unit-IV single dose (one time only)
v. Defibrillation × 1 (360 J)
vi. Amiodarone or
lidocaine or
procainamide
vi. Magnesium (if known magnesium deficiency)

15
Summary

OUTLINE	
Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values	138

Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values

Intervals and Lead Areas	
1 small box = 0.04 s or 1 mm	Anteroseptal wall → V_1 and V_2
1 large box = 0.2 s or 5 mm	Anterior wall → V_3 and V_4
P wave → <0.11 s	Anterolateral wall → V_5 and V_6
PR interval → 0.12-0.2 s	High lateral → I and aVL
QRS complex → <0.07-0.10 s	Inferior wall → leads II, III, aVF
QTc interval → 0.33-0.47 s	Lateral leads → I, aVL, $V_{5,}$ and V_6
QTc (corrected QT interval) = QT interval/square root of R-R interval (millisecond)	
A. RATE	
Count number of large boxes between RR and divide 300 by the number of boxes.	
Count number of large boxes between R-R in 10 seconds multiplied by 6.	
Per big boxes: 300-150-100-75-60.	
B. RHYTHM	

P wave followed by QRS → SINUS	Regular	<60 bpm	Sinus bradycardia
		60-100 bpm	Normal sinus rhythm
		>100 bpm	Sinus tachycardia
	Irregular	Sinus arrhythmia	

(*continued*)

Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values (*Continued*)			
No p waves	Irregularly Irregular	Atrial fibrillation	
	Regular	Slow/normal	Junctional/ idioventricular rhythm
		Rapid	SVT/atrial flutter
		Wide complex	Monomorphic-ventricular tachycardia vs SVT with aberrant conduction
			Polymorphic torsade de pointes
PR interval 0.12-0.2 s	First-degree AV block	Constant prolonged PR interval >0.2 (200 ms)	
	Second-degree AV block	Gradual PR prolongation with sudden drop in QRS complex	Mobitz type I Wenckebach
		Constant PR (not prolonged) with sudden drop in QRS complex	Mobitz type II
	Third-degree AV block	QRS does not follow P P-P interval constant R-R interval constant	

(*continued*)

Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values (*Continued*)

C. AXIS

Lead I	Lead aVF	Lead II†	Axis
(+)	(+)	(+)	Normal
(+)	(−)	(−)	Left
(−)	(+)	(+)	Right
(−)	(−)	(−)	Right or indeterminate if aVR+

†Use lead II if a VF is isoelectric
(+) → QRS upward deflection > downward deflection
(−) → QRS downward deflection > upward deflection

D. QRS DURATION

<0.10 s	Normal
0.10-0.12 s	Incomplete BBB or LAFB/LPFB
	LAFB (left anterior fascicular block) = LAD + Q_1S_3
	LPFB (left post-fascicular block) = RAD + Q_3S_1
>0.12 s	Complete RBBB (rSR in V_1)
	LBBB; nonspecific intraventricular conduction delay (qR or q)
	Bifascicular block = RBBB + LAFB

E. HYPERTROPHIES

RAE	Lead II p wave >2.5 mm (also known as "P"-pulmonale)
LAE	V_1 p-wave negative deflection >1 block wide and >1 block deep (also known as P-mitrale)

(*continued*)

Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values (*Continued*)	
LVH	1. R wave in aVL >12 mm 2. (S wave in V_1 or V_2, whichever is larger) + (R wave in V_5 or V_6, whichever is larger) ≥35 mm
RVH	1. R > S in V_1 2. R decreases from V_1 to V_6
RAE = right atrial enlargement	LAE = left atrial enlargement
LVH = left ventricular enlargement	RVH = right ventricular enlargement

F. PROLONGED QTc ETIOLOGIES

QTc (corrected QT interval) = QT interval/Square root of RR interval (millisecond)

Medications	**Miscellaneous Medications**
Antibiotics	Phenylamine
Azithromycin, erythromycin, clarithromycin	Cisapride
Telithromycin	Domperidone
Levofloxacin, moxifloxacin, gatifloxacin	Droperidol
Sparfloxacin	Probucol
Pentamidine	Cocaine
Spiramycin, chloroquine, halofantrine, mefloquine	Terodiline
Antihistamines	Papaverine
Astemizole	Chloral hydrate
Terfenadine	Arsenic

(*continued*)

Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values (*Continued*)	
Medications	**Miscellaneous Medications**
Antiarrhythmics	Cesium chloride
Amiodarone	Levomethadyl
Disopyramide	**Metabolic etiology**
Dofetilide, sematilide, ibutilide, bepridil, mibefradil	Hypokalemia
Procainamide/ *N*-acetylprocainamide	Hypomagnesemia
Quinidine	Hypocalcemia
Sotalol	Hypothyroidism
Psychotropic	Starvation
Butorphanol	**Miscellaneous**
Haloperidol	Idiopathic
Methadone (high dose)	Mitral valve prolapse
Phenothiazine	Myocardial ischemia/ infarction
Risperidone	HIV
SSRI	Hypothermia
TCA	Connective tissue disease
Thioridazine	Jervell–Lange–Nielsen and Romano-Ward syndrome

(*continued*)

Table 15–1 EKG Reading: Normal EKG Intervals and Segment Values (*Continued*)
G. MISCELLANEOUS
COPD pattern: Precordial leads R/S ratio <1
Chronic lung disease: Poor R-wave progression, P-pulmonale, MAT (multifocal atrial tachycardia)
T-wave flattening: Ischemia, hypokalemia, or nonspecific
U wave: Hypokalemia, ischemia
QT shortening: Hypercalcemia
QT prolongation: Hypocalcemia, other metabolic abnormalities
Pulmonary embolism: Tachycardia, T ↓ in V_1-V_4, rarely S in I, Q in III, T inversion in III is seen
WPW: PR shortening, wide QRS, and delta wave

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