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A Comprehensive Study Guide to Improve ECG Interpretation Skills

Third Edition

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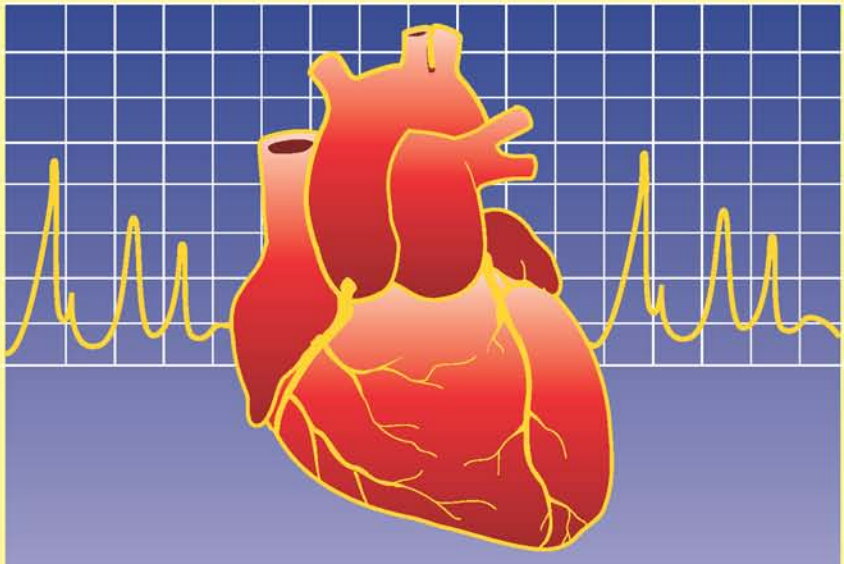

New ECG Cases and Quick Reviews

The Study Guide of Choice for Cardiology Training Programs Around the Nation

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The Complete Guide to ECGs

Third Edition

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Dedication

To my family. My connection from the past, into the future. The foundation that brings joy, meaning and strength to my life.

James O'Keefe

To my wife Karen and sons Noel, Eric, Steve, and Danny — thanks for your patience and support.

Stephen Hammill

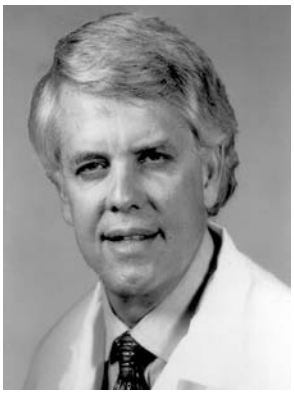
To my mother, father, Ralph, Susie, Bradley, Paulie, Jill, and Josephine, the joys of my life.

Mark Freed

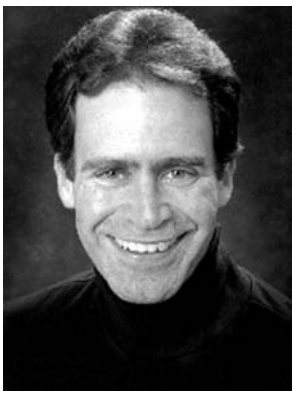
To my wife Cindy, and to my children Leah and Mike, with love; and in memory of my father, Edward.

Steven Pogwizd

James O'Keefe, Jr., MD



Stephen Hammill, MD



Mark Freed, MD

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Preface

The Complete Guide to ECGs has been developed as a unique and practical means for physicians, physicians-in-training, and other medical professionals to improve their ECG interpretation skills. The highly interactive format and comprehensive scope of information are also ideally suited for physicians preparing for the American Board of Internal Medicine (ABIM) Cardiovascular Disease or Internal Medicine Board Exams, the American College of Cardiology ECG proficiency test, and other exams requiring ECG interpretation.

This Third Edition includes many new ECG cases and quizzes and contains more than 1000 questions and answers related to ECG interpretation. Also featured are sections on approach to ECG interpretation and ECG differential diagnosis and an expanded final section on ECG criteria.

We recommend using the answer sheet on many other ECGs in addition to the sample tracings provided. Study groups and regular educational conferences are ideal settings for the presentation of unknown ECGs and discussion of their correct interpretation.

We hope you enjoy reading *The Complete Guide to ECGs* and find it a practical resource for patient care.

James O'Keefe, Jr., M.D.
Stephen Hammill, M.D.
Mark Freed, M.D.
Steven Pogwizd, M.D.

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Abbreviations

APC	*Atrial premature contraction*
AV	*Atrioventricular*
COPD	*Chronic obstructive pulmonary disease*
JPC	*Junctional premature complex*
LAFB	*Left anterior fascicular block*
LBBB	*Left bundle branch block*
LPFB	*Left posterior fascicular block*
LVH	*Left ventricular hypertrophy*
MI	*Myocardial infarction*
RBBB	*Right bundle branch block*
RVH	*Right ventricular hypertrophy*
SA	*Sinoatrial*
SVT	*Supraventricular tachycardia*
VA	*Ventriculoatrial*
VF	*Ventricular fibrillation*
VPC	*Ventricular premature contraction*
VT	*Ventricular tachycardia*
WPW	*Wolff-Parkinson-White*

Nomenclature

The relative amplitudes of the component waves of the QRS complex are described using small (lower case) and large (upper case) letters. For example, an rS complex describes a QRS with a small R wave and a large S wave; a qRs complex describes a QRS with a small Q wave, a large R wave, and a small S wave; and an RSR' complex describes a QRS with a large R wave, a large S wave, and a large secondary R wave (R'). When the QRS complex consists solely of a Q wave, a "QS" designation is used.

Acknowledgments

We wish to acknowledge Monica Crowder Kaufman for her outstanding work in typing and formatting this guide, Norm Lyle for cover art, and the excellent staff at Jones and Bartlett Publishers. We are indebted to these individuals, and hope their efforts are well-received.

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Notice

The ECG interpretations and criteria expressed in this book represent a consensus among the authors based on previously published literature and their own experience and viewpoints. The authors and publisher disclaim responsibility for adverse effects resulting from omissions or undetected errors or adverse results obtained from the use of such information. Readers are encouraged to review other references on ECG interpretation to further expand their knowlege and interpretation skills.

General Instructions

Read each ECG in a thorough and systematic fashion, using the answer sheet to record your findings. Be organized. Be compulsive. Be strict in your application of the ECG criteria. And take your time — even the most experienced electrocardiographers miss important ECG diagnoses when hurrying through an interpretation. Be sure to analyze the following 14 features on each ECG, as outlined here and described in greater detail in Section 1:

01. Heart rate
02. P wave morphology and amplitude
03. Origin of the rhythm
04. PR interval
05. QRS width
06. QT interval
07. QRS axis
08. QRS voltage
09. R wave progression in the precordial leads
10. Abnormal Q waves
11. ST segment
12. T wave
13. U wave
14. Electronic pacemaker

Once these features have been identified, ask yourself the following questions:

1. Is an arrhythmia and/or conduction disturbance present?
2. Is chamber enlargement and/or hypertrophy present?
3. Is ischemia, injury, and/or infarction present?
4. Are any clinical disorders (items 70-89 on answer sheet) likely to be present?

It is important to consider each ECG in the context of the clinical history. For example, diffuse mild ST segment elevation in an asymptomatic patient is likely to represent early repolarization abnormality, whereas the same finding in a patient with chest pain and a friction rub is more likely to represent acute pericarditis.

After coding the ECG on the accompanying answer sheet, study the correct interpretation. If ECG diagnoses were missed or improperly selected, turn to the final section of the book and review the appropriate criteria. The ECG criteria expressed in this book represent a consensus among the authors, based on previously published literature and their own experience and viewpoints.

Answer the multiple choice and fill-in-the-blank questions corresponding to each ECG. Cover the answer column as you work through the “Quick Reviews” and “Pop Quizzes” that follow each case. Place a check mark next to the questions that were answered incorrectly; at the end of each reading session and at the start of each new reading session, return to these questions and be sure they can be answered correctly. Once all the ECGs have been interpreted and the questions answered, review them again until they are mastered.

Common Dilemmas in ECG Interpretation

Questions frequently arise regarding "optimal coding" of ECG tracings, since many specific ECG criteria remain controversial and no single ECG reference standard exists. The following recommendations to some common dilemmas in ECG interpretation represent a consensus among the authors based on previously published literature and their experience and viewpoints.

Problem 1: Q waves are present in leads V_1 and V_2 only. Should a myocardial infarction be coded? Recommendation: No, it is important to follow strict coding criteria when interpreting ECGs. To code an anteroseptal myocardial infarction, Q waves must be present in leads V_1, V_2 *and* V_3. In day-to-day clinical medicine, Q waves in V_1 and V_2 are often referred to as "possible" anteroseptal MI or low anterior forces. While this designation is acceptable in clinical cardiology, Q wave myocardial infarction should not be coded in standardized testing formats.

Problem 2: The ECG shows acute myocardial infarction. Should any other ECG diagnoses be coded? Recommendation: Yes, it is important to code item 65 (ST and/or T abnormalities suggesting myocardial injury) when acute myocardial infarction with typical ST segment elevation is present. Remember to also use this code when ST segment depression is present in leads V_1 and V_2 in the setting of posterior MI.

Problem 3: Left bundle branch block is present. Should acute myocardial infarction ever be coded? Recommendation: No (controversial). Most electrocardiographers are reluctant to diagnose acute myocardial infarction in the setting of LBBB. However, three criteria have independent value for diagnosing acute myocardial injury (item 65):

- ST elevation $\geq$ 1 mm concordant to (same direction as) the major deflection of the QRS
- ST depression $\geq$ 1 mm in lead V_1, V_2, or V_3
- ST elevation $\geq$ 5mm discordant with (opposite direction to) the major deflection of the QRS.

Problem 4: Acute myocardial infarction is present with ST elevation in one portion of the tracing and ST segment depression in another. Is it necessary to code both ST-T changes suggesting myocardial injury and ST-T changes suggesting myocardial ischemia? Recommendation: Many acute myocardial infarctions have ST elevation in some leads and ST depression in others. ST segment depression may be due to reciprocal ECG changes, ischemia adjacent to or remote from the infarct zone, or non-Q-wave myocardial infaction. Item 65 (ST and/or T wave abnormalities suggesting myocardial injury) should be coded in this setting, but not item 64 (ST and/or T wave abnormalities suggesting myocardial ischemia). Remember to code item 59 (posterior MI, age recent or acute) when there is an initial R wave $\geq$ 0.04 seconds in lead V_1 or V_2 with R/S wave amplitude > 1 plus significant (usually $\geq$ 2 mm) ST segment depression, particularly in the setting of acute inferior MI.

Problem 5: Ischemic-looking ST segment elevation is present without pathological Q waves in a patient with chest pain. Should acute myocardial infarction be coded? Recommendation: No, convex upward ST segment elevation without abnormal Q waves in the setting of chest pain should be coded as item 65 (ST and/or T wave abnormalities suggesting myocardial injury). Clinically, this usually represents the early stages of acute infarction (or transient coronary spasm and/or occlusion), and most of these patients need urgent pharmacologic or mechanical intervention to restore coronary blood flow to the jeopardized myocardium. Nevertheless, in the absence of pathological Q waves (or pathological R waves in the case of posterior infarction), acute myocardial infarction should not be coded.

Problem 6: With so many different criteria for the diagnosis of left ventricular hypertrophy (LVH), which should be used as the "gold-standard?" Recommendation: The Cornell criteria (R wave in aVL + S wave in V_3 > 28 mm in males or > 20 mm in females) is probably the most accurate voltage criterion. However, many ECGs meet voltage criteria in one area of the tracing, but not in the others, and all criteria for LVH are relatively insensitive when considered individually. Therefore, it is best to know most or all of the various criteria for LVH (item 40). Remember to code item 67 (ST and/or T wave abnormalities secondary to hypertrophy) if a "strain" pattern is present in association with LVH.

Problem 7: What are the most important criteria for diagnosing right ventricular hypertrophy (RVH)? Recommendation: RVH, like LVH, is difficult to diagnosis due to the numerous different criteria that have been proposed. No single finding is diagnostic of RVH. Important elements include right axis deviation and a dominant R wave with secondary ST and/or T wave changes in leads V_1 and V_2. Right atrial abnormality is also common. If repolarization abnormalities are present, remember to code item 67 (ST and/or T wave abnormalities secondary to hypertrophy).

Problem 8: Second-degree or third-degree AV block is present. Should first-degree AV block also be coded if the PR interval exceeds 0.20 seconds? Recommendation: No. It is not necessary to code first-degree AV block when higher levels of AV block are present.

Problem 9: A junctional or ventricular rhythm is present. Is it necessary to code the underlying atrial rhythm if one is present? Recommendation: Yes. If an atrial rhythm is present in addition to a dominant junctional or ventricular rhythm, the atrial rhythm (and AV block, if present) should also be coded (e.g., ventricular escape rhythm and sinus rhythm with third-degree AV block).

Problem 10: Should left axis deviation be coded when left anterior fascicular block (LAFB) is present? Similarly, should right axis deviation be coded when left posterior fascicular block (LPFB) is present? Recommendation: No. A description of the axis in LAFB or LPFB is redundant.

Problem 11: Wolff-Parkinson-White (WPW) pattern is present. When should myocardial infarction be coded? Recommendation: Acute MI should not be diagnosed in the presence of WPW since most "Q" waves are actually negative delta waves, resulting in a pseudoinfarct pattern.

Problem 12: Atrial fibrillation is present with intermittent episodes of atrial flutter (i.e., "fib/flutter). Should atrial fibrillation or atrial flutter be coded? Recommendation: The best strategy in this setting is to code atrial fibrillation. Atrial flutter should be reserved for tracings that show continuous atrial flutter without interspersed episodes of fibrillation.

Problem 13: Left ventricular hypertrophy with a "strain" pattern (ST depression with T wave inversion) is evident in the lateral leads. Should item 64, "ST and/or T wave abnormalities suggesting myocardial ischemia," be coded? Recommendation: No. When LVH with strain is present, items 40 (left ventricular hypertrophy) and 67 (ST and/or T wave abnormalities secondary to hypertrophy) should be coded.

Problem 14: A narrow QRS tachycardia without P waves is present throughout the ECG tracing. Should item 15 (atrial tachycardia) or item 17 (supraventricular tachycardia, paroxysmal) be coded? Recommendation: Paroxysmal SVT (item 17) should be coded, even if the arrhythmia persists throughout the tracing. Atrial tachycardia (item 15) should be reserved for narrow complex tachycardias with identifiable ectopic P waves; a short PR interval is often but not always present.

Problem 15: A patient with atrial fibrillation or chronic heart failure demonstrates sagging ST segment depression, paroxysmal atrial tachycardia (PAT) with block, or complete heart block with accelerated junctional rhythm on ECG. Should item 70 (digitalis effect) or item 71 (digitalis toxicity) be coded if the clinical history does not specifically state the patient is receiving digoxin. Recommendation: Yes. It is appropriate to code digitalis effect or toxicity for classic findings in a patient likely to be receiving digoxin therapy.

— Section 1 —

APPROACH TO ECG INTERPRETATION

Each ECG should be read in a thorough and systematic fashion. It is important to be organized, compulsive, and strict in your application of the ECG criteria. Analyze the following features on every ECG:

1. Heart rate 5
2. P wave 6
3. Origin of the rhythm 7
4. PR interval 7
5. QRS duration 8
6. QT interval 8
7. QRS axis 9
8. QRS voltage 9
9. Precordial R wave progression 9
10. Abnormal Q waves 10
11. ST segment 10
12. T wave 11
13. U wave 11
14. Electronic pacemaker 11

Once these features have been identified, ask the following questions:

1. Is an arrhythmia or conduction disturbance present?
2. Is chamber enlargement or hypertrophy present?
3. Is ischemia, injury, or infarction present?
4. Is a clinical disorder present (see items 70-89 on answer sheet)?

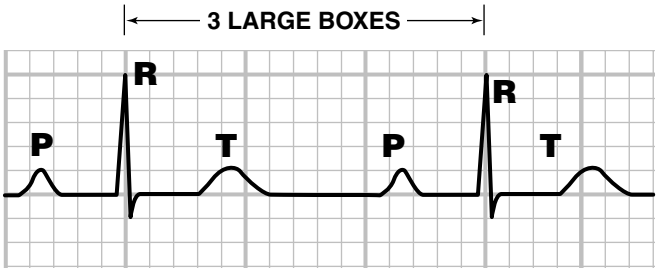
Be sure to consider each ECG in the context of the clinical history. For example, diffuse mild ST segment elevation in a young, asymptomatic patient without previous cardiac history is likely to represent early repolarization abnormality, whereas the same finding in a patient with chest pain and a friction rub is more likely to represent acute pericarditis.

1. Heart Rate

The following method can be used to determine heart rate (assumes a standard paper speed of 25 mm/sec)

Regular Rhythm

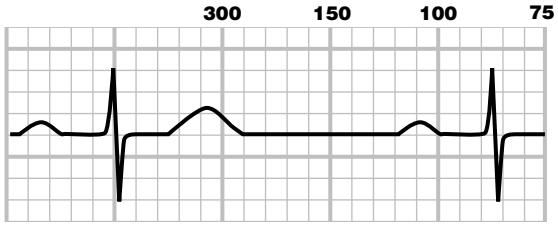
- Count the number of large boxes between P waves (atrial rate), R waves (ventricular rate), or pacer spikes (pacemaker rate)
- Beats per minute = 300 divided by the number of large boxes



Heart Rate = 300 ∏no. large boxes between "R" Waves = 300 ∏3 = 100 bpm

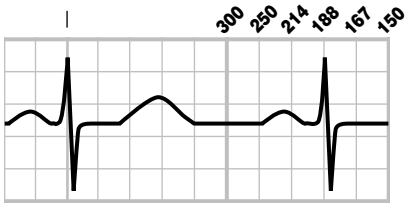
Note: It is easier to memorize the heart rates associated with each of the large boxes, rather than count the number of large boxes (1 ,2, 3, etc) and divide into 300:

Note: If the number of large boxes is not a whole number, either estimate the rate (this is routine practice) or divide 1500 by the number of small boxes between P waves (atrial rate), R waves (ventricular rate), or pacer spikes (pacemaker rate):



ESTIMATED Heart Rate = halfway between 100 and 75 = ~ 87 bpm (or 1500 ÷ 17.5 small boxes)

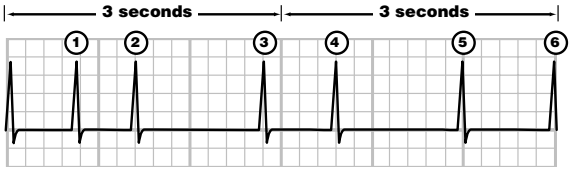
Note: For tachycardias, it is helpful to memorize the rates between 150 and 300 BPM:



Heart Rate = 188 bpm

Slow or Irregular Rhythm

- Identify the 3-second markers at top or bottom of ECG tracing
- Count the number of QRS complexes (or P waves or pacer spikes) that appear in 6 seconds (i.e., two consecutive 3-second markers)
- Multiply by 10 to obtain rate in BPM



ESTIMATED Heart Rate = number of QRS complexes in 6 seconds x 10 = 6 x 10 = 60 bpm

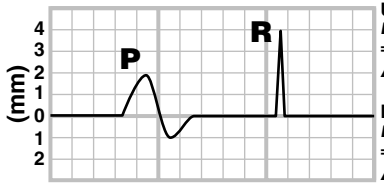
2. P Wave

What It Represents

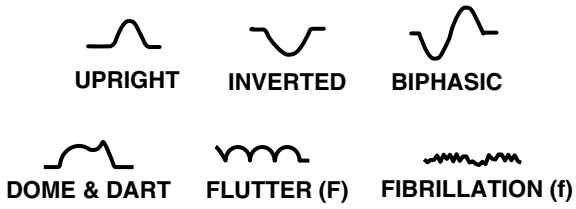
The P wave represents electrical forces generated from atrial activation. The first and second halves of the P wave roughly correspond to right and left atrial activation, respectively.

What to Measure

- Duration (seconds): Measured from the beginning of the P wave to the end of P wave.
- Amplitude (mm): Measured from baseline to top (or bottom) of P wave. Positive and negative deflections are determined separately. One small box = 1 mm on standard scale ECGs (i.e., 10 mm = 1 mV)



- Morphology:



P Wave Characteristics

- Normal P wave duration: 0.08-0.11 seconds
- Normal P wave axis: 0-75°
- Normal P wave morphology: Upright in I, II, aVF; upright or biphasic in III, aVL, V_1, V_2. Small notching may be present
- Normal P wave amplitude: Limb leads: < 2.5 mm; V_1: positive deflection < 1.5 mm and negative deflection < 1 mm

3. Origin of the Rhythm

Rhythm identification is one of the most difficult and complex aspects of ECG interpretation, and one of the most common mistakes made by computer ECG interpretation programs. Proper rhythm interpretation requires integration of heart rate, RR regularity, P wave morphology, PR interval, QRS width, and the P:QRS relationship. No single algorithm can simply describe all the various permutations; however the following rhythm-recognition tables, based initially on the P:QRS relationship and heart rate, provide a useful frame of reference:

— P:QRS Relationships —

P:QRS < 1: Junctional or ventricular premature complexes or rhythms (escape, accelerated, tachycardia)

P:QRS = 1

- **P wave preceeds QRS**: Sinus rhythm; ectopic atrial rhythm; multifocal atrial tachycardia; wandering atrial pacemaker; SVT (sinus node reentry tachycardia, automatic atrial tachycardia); sinoatrial exit block, 2º; conducted APCs with any of the above
- **P wave follows QRS**; SVT (AV nodal reentry tachycardia, orthodromic SVT); junctional / ventricular rhythm with 1:1 retrograde atrial activation

No P Waves: Atrial fibrillation; atrial flutter; sinus arrest with junctional or ventricular escape rhythm; SVT (AV nodal reentry tachycardia, AV reentry tachycardia), junctional tachycardia or VT with P wave buried in QRS; VF

— Heart Rate < 100 BPM —

Narrow QRS (< 0.12 sec) - Regular R-R

- Sinus P; rate 60-100: *Sinus rhythm*
- Sinus P; rate < 60: *Sinus bradycardia*
- Nonsinus P; PR ≥ 0.12: *Ectopic atrial rhythm*
- Nonsinus P; PR < 0.12: *Junctional or low atrial rhythm*
- Sawtooth flutter waves: *Atrial flutter, usually with 4:1 AV block*
- No P; rate < 60: *Junctional rhythm*
- No P; rate 60-100: *Accelerated junctional rhythm*

Narrow QRS - Irregular R-R

- Sinus P, P-P varying > 0.16 seconds: *Sinus arrhythmia*
- Sinus and nonsinus P: *Wandering atrial pacemaker*
- *Any regular rhythm with 2º/ 3º AV block or premature beats*
- Fine or coarse baseline oscillations: *Atrial fibrillation with slow ventricular response*
- Sawtooth flutter waves: *Atrial flutter, usually with variable AV block*
- P:QRS ratio > 1: *2º or 3º AV block or blocked APCs*
- P:QRS ratio < 1: *Junctional or ventricular premature beats or escape rhythm*

Wide QRS (≥ 0.12 seconds)

- Sinus or nonsinus P: *Any supraventricular rhythm with a preexisting IVCD (e.g. bundle branch block) or aberrancy*
- No P†; rate < 60: *Idioventricular rhythm*
- No P†; rate 60-100: *Accelerated idioventricular rhythm*

† AV dissociation may be present

— Heart Rate > 100 BPM —

Narrow QRS (< 0.12 sec) - Regular R-R

- Sinus P: *Sinus tachycardia*
- Flutter waves: *Atrial flutter*
- No P: *AV nodal reentrant tachycardia (AVNRT), junctional tachycardia*
- Short R-P (R-P < 50% of R-R interval): *AVNRT, orthodromic SVT (AVRT), atrial tachycardia with 1º AV block, junctional tachycardia with 1:1 retrograde atrial activation*
- Long R-P (R-P > 50% of R-R interval): *Atrial tachycardia, sinus node reentrant tachycardia, atypical AVNRT, orthodromic SVT with prolonged V-A conduction*

Narrow QRS - Irregular R-R

- Nonsinus P; > 3 morphologies: *Multifocal atrial tachycardia*
- Fine or coarse baseline oscillations: *Atrial fibrillation*
- Flutter waves: *Atrial flutter*
- *Any regular rhythm with 2º/3º AV block or premature beats*

Wide QRS (≥ 0.12 seconds)

- Sinus or nonsinus P: *Any regular or irregular supraventricular rhythm with a preexisting IVCD or aberrancy*
- No P; rate 100-110: *Accelerated idioventricular rhythm*
- No P, rate 110-250: *VT, SVT with aberrancy*
- Irregular, polymorphic, alternating polarity: *Torsade de Pointes*
- Chaotic irregular oscillations; no discrete QRS: *Ventricular fibrillation*

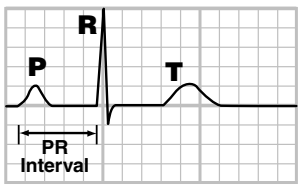
4. PR Interval & Segment

What it Represents

- PR interval represents conduction time from the onset of atrial depolarization to the onset of ventricular repolarization. It does not reflect conduction from the sinus node to the atrium.
- PR segment represents atrial repolarization.

How to Measure

- PR interval (seconds): From the beginning of the P wave to the first deflection of the QRS complex. Measure longest PR seen.



PR INTERVAL = 4 small boxes = 4 x 0.04 = 0.16 sec.

- PR segment (mm): Amount of elevation or depression relative to the TP segment (end of the T wave to the beginning of the P wave).

Definitions

PR Interval

- Normal PR interval: 0.12 - 0.20 seconds
- Prolonged PR interval: > 0.20 seconds
- Short PR interval: < 0.12 seconds

PR Segment

- Normal PR segment: Usually isoelectric. May be displaced in a direction opposite to the P wave. Elevation is usually < 0.5 mm; depression is usually < 0.8 mm
- PR segment elevation: Usually ≥ 0.5 mm
- PR segment depression: Usually ≥ 0.8 mm

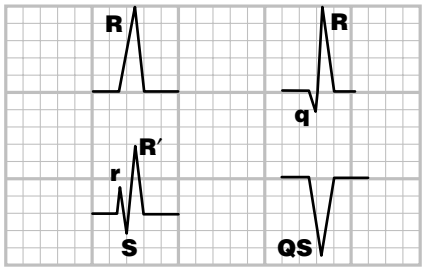
5. QRS Duration

What it Represents

Duration of ventricular activation

How to Measure

In seconds, from the beginning to the end of the QRS (or QS) complex



QRS duration = 1.5 small boxes = 0.06 sec.

Definitions

- Normal QRS duration: < 0.10 seconds
- Increased QRS duration: ≥ 0.10 seconds

  Note: For the purposes of establishing a differential diagnosis, it is often useful to distinguish moderate prolongation of the QRS (0.10 to ≤ 0.12 seconds) from marked prolongation of the QRS (> 0.12 seconds)

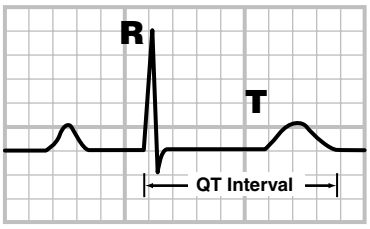
6. QT Interval

What it Represents

Total duration of ventricular systole, i.e., ventricular depolarization (QRS complex) and repolarization (T wave)

How To Measure

- QT interval: In seconds, from the beginning of the QRS (or QS) complex to the end of the T wave. It is best use a lead with a large T wave and distinct termination



QT interval = 8 small boxes = 8 x 0.04 sec. = 0.32 sec.

- Corrected QT interval (QTc): Since the normal QT interval varies inversely with heart rate, the QTc, which corrects for heart rate, is usually determined
  - QTc (sec) = QT interval (sec) divided by the square root of the preceding RR interval (sec). **Example**: For heart rate of 50 BPM, RR interval = 1.2 seconds, and QTc = QT ÷ square root of 1.2 = QT ÷ 1.1
  - Alternative method: Use 0.40 seconds as the normal QT interval for a heart rate of 70 BPM. For every 10 BPM change in heart rate above (or below) 70, subtract (or add) 0.02 seconds. The measured value should be within ± 0.04 seconds of the calculated normal. **Example**: For a heart rate of 100 BPM, the calculated "normal" QT interval = 0.40 seconds — (3 x 0.02 seconds) = 0.34 ± 0.04 seconds. For a heart rate of 50 BPM, the calculated "normal" QT interval = 0.40 seconds + (2 x 0.02 seconds) = 0.44 ± 0.04 seconds.

Definitions

- Normal QTc: 0.35-0.43 seconds for heart rates of 60-100 BPM. The normal QT should be < 50% of the RR interval
- Prolonged QTc: ≥ 0.44 seconds
- Short QTc: < 0.35 seconds for heart rates of 60-100 BPM

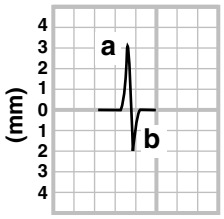
7. QRS Axis

What It Represents

The major vector of ventricular activation

How to Determine

- Determine if "net QRS voltage" (upward minus downward QRS deflection) is positive (> 0) or negative (< 0) in leads I, II, aVF:



NET QRS VOLTAGE = upward – downward deflection (mm) = a – b = 3 – 2 = 1 (positive)

- Determine axis category according to the chart below:

Axis	Net QRS Voltage		
	Lead I	aVF	Lead II
Normal axis (0º to 90º)	+	+	
Normal variant (0º to -30º)	+	-	+
Left axis deviation (-30º to -90º)	+	-	-
Right axis deviation (> 100º)	-	+	
Right superior axis (-90º to +180º)	-	-	
"+" represents positive (> 0) net QRS voltage "–" represents negative (< 0) net QRS voltage			

8. QRS Voltage

How to Measure

In millimeters, from baseline to the peak of the R wave (R wave voltage) or S wave (S wave voltage) (see QRS axis, above)

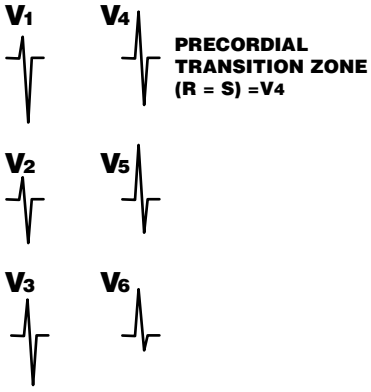
Definitions

- Normal voltage: Amplitude of the QRS has a wide range of normal limits, depending on the lead, age of the individual, and other factors
- Low voltage (from peak of R wave to peak of S wave): Total QRS amplitude (R + S) < 5 mm in all limb leads and < 10 mm in all precordial leads
- Increased voltage: See LVH (item 40, Section 4) and RVH (item 41, Section 4)

9. R Wave Progression

How to Identify

Determine the *precordial transition zone*, i.e., the lead with equal R and S wave voltage (R/S = 1)



Definitions

- Normal R wave progression: Transition zone = V_2-V_4, with increasing R wave amplitude across the precordial leads. (Exception: R wave in V_5 often exceeds R wave in V_6.)
- Poor R wave progression: Transition zone = V_5 or V_6
- Reverse R wave progression: Decreasing R wave amplitude across the precordial leads

10. Q Waves

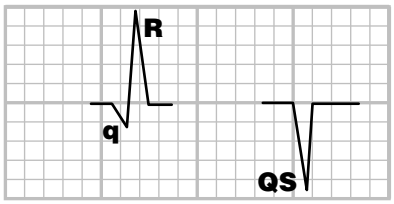
How to Identify

A Q wave is present when the first deflection of the QRS is negative. If the QRS consists exclusively of a negative

deflection, that defection is considered a Q wave, but the complex is referred to as a "QS" complex

What to Measure

Duration, in seconds, from the beginning to the end (i.e., when it returns to baseline) of the Q wave. When the QRS complex consists solely of a Q wave, a "QS" designation is used



Q wave duration = 1 small box = 0.04 seconds

Definitions

- Normal Q waves: Small Q waves (duration < 0.03 seconds) are common in most leads, except aVR, V_1-V_3
- Abnormal Q waves: Any Q wave in leads V_1-V_3. Q wave ≥ 0.03 seconds in leads I, II, aVL, aVF, V_4, V_5, or V_6. Note: For Q-wave myocardial infarction, Q wave changes must be present in at least 2 continguous leads and must be ≥ 1 mm in depth.

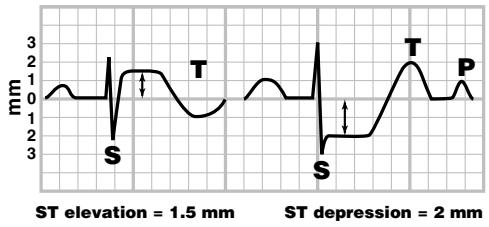
11. ST Segment

What it Represents

The ST segment represents the interval between the end of ventricular depolarization (QRS complex) and the beginning of repolarization (T wave). It is identified as the segment between the end of the QRS complex and the beginning of the T wave.

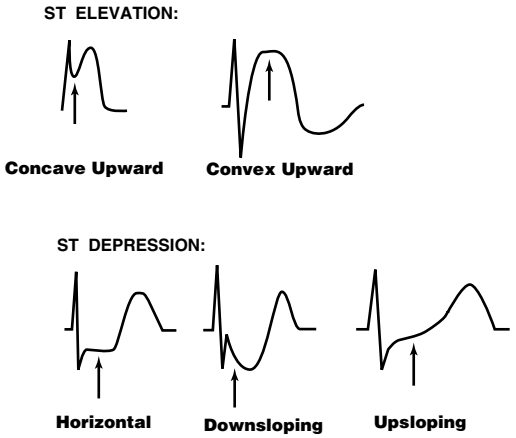
What to Identify

- Amount of elevation or depression, in millimeters, relative to the TP segment (end of the T wave to the beginning of the P wave)



ST elevation = 1.5 mm **ST depression = 2 mm**

- ST segment morphology



Definitions

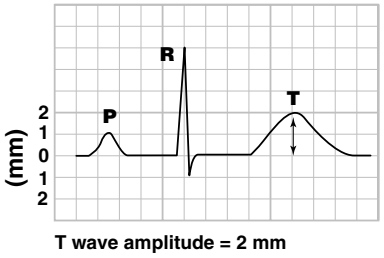
- Normal ST segment: Usually isoelectric, but may vary from 0.5 mm below to 1 mm above baseline in limb leads, and up to 3 mm concave upward elevation may be seen in the precordial leads in early repolarization (see item 61, Section 4).

  Note: While some ST segment depression and elevation can be seen in normals, it may also indicate myocardial infarction, injury, or some other pathological process. It is especially important to consider the clinical presentation and compare it to previous ECGs (if available) when ST segment depression or elevation is identified.
- Nonspecific ST segment: Slight (< 1 mm) ST segment depression or elevation.

12. T Wave

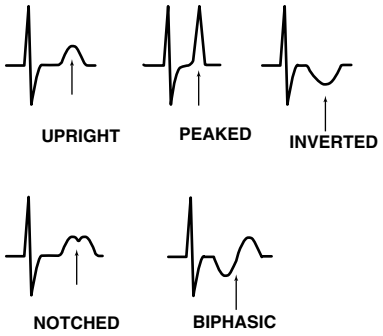
What it Represents
The electrical forces generated from ventricular repolarization

What to Identify
- Amplitude: In millimeters, from baseline to peak or valley of the T wave:



T wave amplitude = 2 mm

- Morphology:



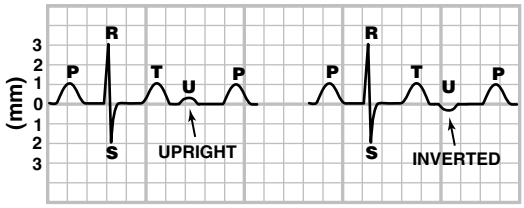
Definitions
- Normal T wave morphology: Upright in I, II, V_3-V_6; inverted in aVR, V_1; may be upright, flat or biphasic in III, aVL, aVF, V_1, V_2. T wave inversion may be present in V_1-V_3 in healthy young adults (juvenile T waves, see item 62, Section 4)
- Normal T wave amplitude: Usually < 6 mm in limb leads and ≤ 10 mm in precordial leads
- Tall T waves: Amplitude > 6 mm in limb leads or > 10 mm in precordial leads
- Nonspecific T waves: Flat or slightly inverted

13. U Wave

What it Represents
Controversial: Afterpotentials of ventricular muscle vs. repolarization of Purkinje fibers.

How to Identify
When present, the U wave manifests as a small (usually positive) deflection following the T wave. At faster heart rates, the U wave may be superimposed on the preceeding T wave.

What to Determine
- Morphology: upright, inverted, or absent
- Height, in millimeters, from baseline to peak or valley



U wave amplitude = 0.3 mm

Definitions
- Normal U wave: Not always present. Morphology is upright in all leads except aVR. Amplitude is 5-25% the height of the T wave (usually < 1.5 mm). U waves are typically most prominent in leads V_2, V_3
- Prominent U wave: Amplitude > 1.5 mm

14. Pacemakers

Overview
Pacemakers are described by a 4 letter code:
- First letter: Refers to the chamber(s) PACED (**A**trial, **V**entricular, or **D**ual)
- Second letter: Refers to the chamber SENSED (**A, V** or **D**)
- Third letter: Refers to the pacemaker MODE (**I**nhibited, **T**riggered, **D**ual).
- Fourth letter: Refers to the presence (**R**) or absence (no letter) of RATE RESPONSIVENESS. Rate-responsive (or rate-adaptive) pacemakers can vary their rate in response to sensed motion or physiologic alterations (e.g., QT interval, temperature) produced by exercise by increasing their rate of

pacing.

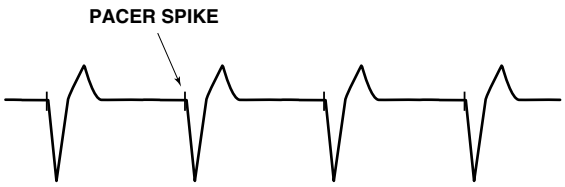
For example, a **VVIR** pacemaker PACES the **V**entricle, SENSES the **V**entricle, is **I**NHIBITED by a sensed QRS complex, and is **R**ate responsive. A DDD pacemaker PACES and SENSES the atria and ventricle; the DUAL MODE indicates that sensed atrial activity will inhibit atrial output and trigger a ventricular output after a designated "AV interval," and that sensed ventricular activity will inhibit ventricular output.

- Typical single chamber pacemakers include VVI and AAI
- Typical dual chamber pacemakers include DVI and DDD

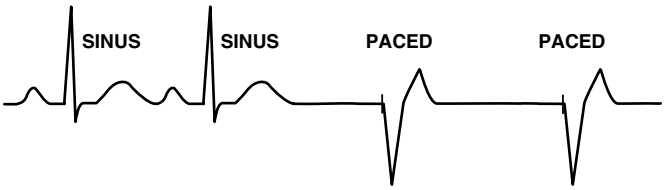
— Approach to Pacemaker Evaluation —

Step 1. Assess underlying rhythm. Determine if the rhythm is 100% paced or whether there is a non-paced intrinsic rhythm with a pacemaker functioning in demand mode.

- 100% ventricular paced



- Ventricular pacing in demand mode (inconstant ventricular pacing from output inhibition by intrinsic sinus rhythm)



Step 2. Determine the chamber(s) PACED

Determine the relationship of pacing spikes to P waves and QRS complexes: A spike preceding the P wave typically represents atrial pacing; a spike preceding the QRS complex typically represents ventricular pacing.

- Atrial (A) paced beat

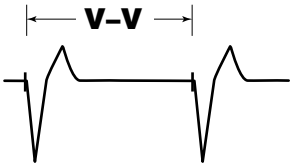
- Ventricular (V) paced beat

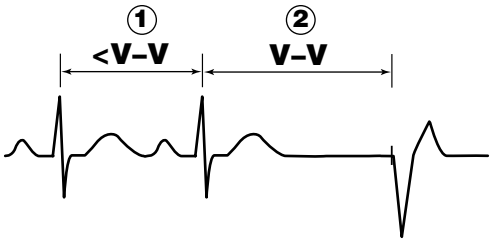
- Atrial (A) and ventricular (V) paced beat

Step 3. Determine timing intervals from 2 consecutively paced beats:

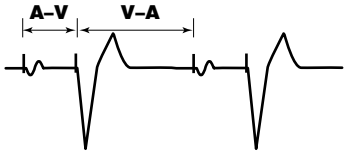
- For atrial pacing, determine the A-A interval

- For ventricular pacing, determine the V-V interval



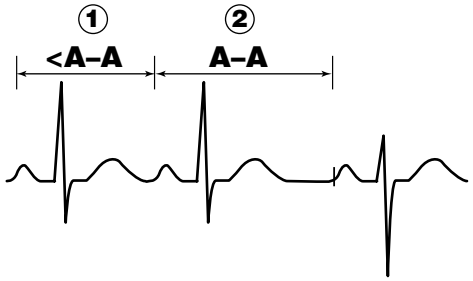


- For dual chamber pacing, determine the A-V and V-A intervals



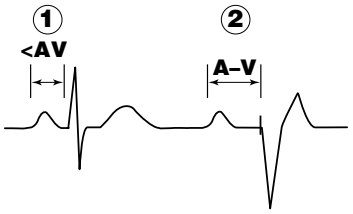
Step 4. Determine the chamber(s) SENSED

- **Atrial pacemaker:** Proper atrial sensing is present when intrinsic atrial activation (native P wave) is followed by: (1) a native P wave that occurs at an interval less than the A-A interval; or (2) an atrial-paced beat that occurs after an interval equal to the A-A interval

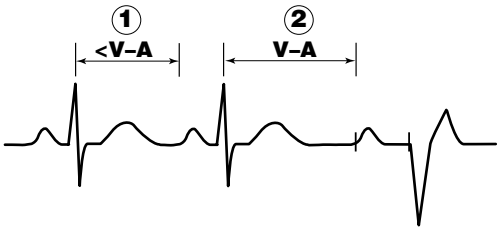


- **Ventricular pacemaker:** Proper ventricular sensing is present when intrinsic ventricular activation (native QRS complex) is always followed by: (1) a native QRS complex that occurs at an interval less than the V-V interval; or (2) a ventricular-paced beat that occurs after an interval equal to the V-V interval

- **Dual chamber pacemaker:**
  - Atrial sensing is evident when intrinsic atrial activation (native P wave) is always followed by: (1) a native QRS complex that occurs at an interval less than the A-V interval; or (2) a ventricular-paced beat that occurs at an interval equal to the A-V interval



  - Ventricular sensing is evident when intrinsic ventricular activation (native QRS complex) is always followed by: (1) a native P wave that occurs at an interval less than the V-A interval; or (2) an atrial-paced beat that occurs at an interval equal to the V-A interval



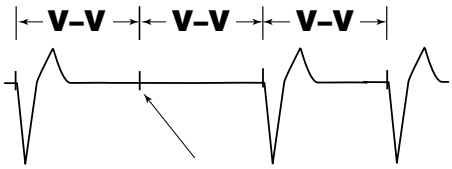
Step 5. Determine the sequence of complexes representing normal pacing function. Keep in mind that single chamber pacing on the surface ECG does not exclude the possibility that a dual chamber pacemaker is present — ventricular-paced beats may be due to a single chamber ventricular pacemaker or a dual chamber pacemaker in which ventricular spikes are timed to follow P waves (DDD pacemaker)

Pacing mode	Atrial pacing spike	Ventricular pacing spike
Atrial pacing	+	-
Ventricular pacing	-	+
Dual-chamber (DDD) pacing	+ + - -	+ - + -

\+ Pacing spike present on surface ECG
\- Pacing spike absent on surface ECG

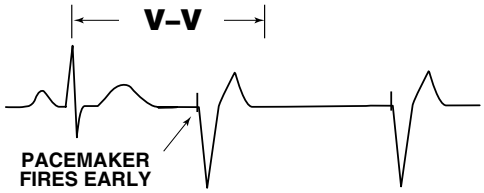
Step 6. Look for pacemaker malfunction

A. Failure to Capture (see item 93, Section 4): Are any pacing spikes not followed by a depolarization?

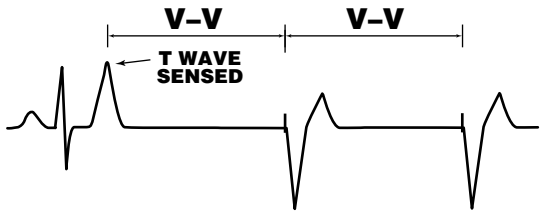


B. Sensing Abnormalities

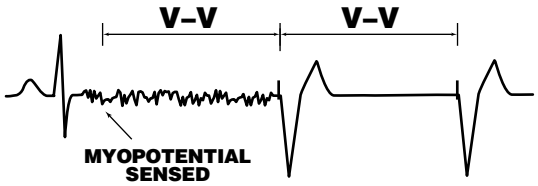
- **Undersensing**: Based on timing intervals, are there pacing spikes that should have been inhibited by a native P wave or QRS complex but were not? This results in a paced beat that appears *earlier* than expected.
  - Example: For ventricular pacing, undersensing is evident when a native QRS complex is followed by a ventricular-paced beat at an interval < V-V interval.



- **Oversensing**: Based on timing intervals, are there pacing spikes that should have been initiated after a native P wave or QRS complex but were not? This results in a paced beat that appears *later* than expected. For ventricular pacing, oversensing occurs when a native QRS is followed by a ventricular-paced beat at an interval much greater than the V-V interval.
  - Oversensing of the T wave, in which the T wave is sensed as (mistaken for) a QRS complex:



  - Oversensing of muscle contractions (myopotential inhibition), in which a myopotential is sensed as (mistaken for) a QRS complex:



C. Other Causes of Pacemaker Malfunction: Less common types of pacemaker malfunction include pacemaker not firing, pacemaker slowing, and pacemaker-mediated tachycardia.

— Section 2 —

ECG DIFFERENTIAL DIAGNOSIS

(Item Numbers in Parentheses Correspond to Criteria in Section 4)

1. P wave 15
2. PR interval 16
3. PR segment 16
4. QRS duration 16
5. QRS amplitude 17
6. QRS axis 17
7. Q wave 17
8. R wave progression 18
9. QRS morphology 18
10. ST segment 18
11. T wave 19
12. QT interval 19
13. U wave 19
14. PP pause 20
15. Group beating 20

P Wave

— LEAD I —

Inverted P wave
- Ectopic atrial premature beat (item 13) or rhythm (items 15, 17)
- AV junctional/ventricular premature complex (items 20, 23) or rhythm with retrograde atrial activation
- Dextrocardia (item 80): Inverted P-QRS-T in leads I and aVL with *reverse* R wave progression in the precordial leads
- Reversal of right and left arm leads (item 03): Inverted P-QRS-T in leads I and aVL with *normal* R wave progression in the precordial leads

— LEAD II —

Tall peaked P wave
- Right atrial abnormality/enlargement (item 05)
- Bi-atrial abnormality

Bifid P wave with peak-to-peak interval < 0.03 sec.
- Normal

Bifid P wave with peak-to-peak interval > 0.03 sec. and P wave duration > 0.12 sec.
- Left atrial abnormality/enlargement (item 06)

Inverted P wave
- Ectopic atrial premature beat (item 13) or rhythm (items 15, 17)
- AV junctional/ventricular premature complex (items 20, 23) or rhythm with retrograde atrial activation

Sawtooth regular P waves
- Atrial flutter (item 18)
- Artifact due to tremor (e.g., Parkinson's disease, shivering) (item 04)

Irregularly irregular baseline
- Atrial fibrillation (item 19)
- Artifact due to tremor (item 04)
- Multifocal atrial tachycardia (item 16)

Multiple P wave morphologies
- Wandering atrial pacemaker (rate < 100 bpm)
- Multifocal atrial tachycardia (rate > 100 bpm) (item 16)
- Sinus or atrial rhythm with multiple atrial premature complexes

— LEAD V_1 —

Tall upright P wave
- Right atrial abnormality/enlargement (item 05)

Deep inverted P waves
- Left atrial abnormality/enlargement (item 06)

Dome and dart P wave
- Ectopic atrial rhythm (item 15)

— NO P WAVES —

P Waves present but hidden
- Ectopic atrial rhythm or APCs (P waves hidden in preceding T wave)
- Junctional rhythm or SVT (P wave buried in QRS)
- Supraventricular rhythm with marked first-degree AV block (P wave hidden in preceding T wave)

P Waves not present
- Sinoventricular conduction due to hyperkalemia (item 74)
- Marked sinoatrial exit block or sinus bradycardia with junctional or ventricular rhythm (escape or accelerated)
- Sinus pause or arrest (item 11)

PR Interval

Prolonged (> 0.20 seconds) PR interval
- First-degree AV block (item 29)
- Complete heart block (item 33): PR interval varies, has no constant relationship to the QRS, and may intermittently exceed 0.20 seconds
- Supraventricular or junctional rhythm with retrograde atrial activation: P wave inverted in lead II
- Atrial premature complex (item 13)

Short (< 0.12 seconds) PR interval
- Short PR with sinus rhythm and normal QRS
- Wolff-Parkinson-White pattern (item 34): Delta wave, wide QRS, ST-T changes in a direction opposite to main deflection of QRS
- Low ectopic atrial rhythm: PR interval usually > 0.11 seconds; P wave inverted in lead II
- Ectopic junctional beat or rhythm with retrograde atrial activation: PR interval usually < 0.11 seconds; P wave inverted in lead II

PR Segment

PR segment depression
- Normals: < 0.8 mm
- Pericarditis (item 84)
- Pseudodepression due to atrial flutter (item 18) or Parkinson's tremor (item 04)
- Atrial infarction: Reciprocal elevation in opposite leads; inferior MI usually evident

PR segment elevation
- Normals: < 0.5 mm
- Pericarditis (item 84): Lead aVR only
- Atrial infarction: Reciprocal depression in opposite leads

QRS Duration

Increased QRS duration 0.10 to < 0.12 seconds
- Left anterior fascicular block (item 45)
- Left posterior fascicular block (item 46)
- Incomplete LBBB (item 48)
- Incomplete RBBB (item 44)
- Nonspecific IVCD (item 49)
- LVH (item 40)
- RVH (item 41)
- Supraventricular beat or rhythm with aberrant intraventricular conduction (item 50)
- Fusion beats
- Wolff-Parkinson-White pattern (item 34)
- VPCs originating near the bundle of His (i.e., high in the interventricular septum)

Increased QRS duration > 0.12 seconds
- RBBB (item 43)
- LBBB (item 47)
- Supraventricular beat or rhythm with aberrant intraventricular conduction (item 50)
- Fusion beats
- Wolff-Parkinson-White pattern (item 34)
- Ventricular premature complexes (item 23)
- Ventricular rhythm (items 24-27)
- Nonspecific IVCD (item 49)
- Paced beat

Section 2. ECG Differential Diagnosis

QRS Amplitude

Low voltage QRS
- Chronic lung disease (item 81)
- Pericardial effusion (item 83)
- Myxedema (item 87)
- Obesity
- Pleural effusion
- Restrictive or infiltrative cardiomyopathy
- Diffuse coronary artery disease

Tall QRS
- LVH (item 40)
- Hypertrophic cardiomyopathy (item 85)
- LBBB (item 47)
- Wolff-Parkinson White pattern (item 34)
- Normal persons with thin body habitus

Prominent R wave in lead V_1
- RVH (item 41)
- Posterior wall MI (items 59, 60)
- Incorrect lead placement: Electrode for lead V_1 placed in 3rd instead of 4th intercostal space
- Skeletal deformities (e.g., pectus excavatum)
- RBBB (item 43)
- Wolff-Parkinson-White pattern (item 34)
- Duchenne's muscular dystrophy

Alternation in QRS amplitude
- Electrical alternans (item 38)

QRS Axis

Left axis deviation
- Left anterior fascicular block (if axis > -45°, item 45)
- Inferior wall MI (items 57, 58)
- LBBB (item 47)
- LVH (item 40)
- Ostium primum ASD (item 79)
- Chronic lung disease (item 81)
- Hyperkalemia (item 74)

Right axis deviation
- RVH (item 41)
- Vertical heart
- Chronic lung disease (item 81)
- Pulmonary embolus (item 82)
- Left posterior fascicular block (item 46)
- Lateral wall MI (items 55, 56)
- Dextrocardia (item 80)
- Lead reversal (item 03)
- Ostium secundum ASD (item 78)

Q Wave

Q wave myocardial infarction (see items 51-60)
- Anterolateral MI: Abnormal Q waves in leads V_4-V_6
- Anterior MI: Abnormal Q waves in at least two consecutive leads in V_2-V_4
- Anteroseptal MI: Abnormal Q waves in leads V_1-V_3 (and sometimes V_4)
- Lateral MI: Abnormal Q waves in leads I and aVL
- Inferior MI: Abnormal Q waves in at least two of leads II, III, and aVF

Pseudoinfarcts (Q waves in absence of MI)
- Wolff-Parkinson-White (item 34): Negative delta waves mimic Q waves
- Hypertrophic cardiomyopathy (item 85): Q waves in I, aVL, V_4-V_6 due to septal hypertrophy
- LVH (item 40): Poor R wave progression, at times with ST elevation in V_1-V_3, can mimic anteroseptal MI. Inferior Q waves may be present and can mimic inferior MI
- LBBB (item 47): QS pattern in V_1-V_4 mimics anteroseptal MI. Less commonly, Q waves in III and aVF mimic inferior MI
- RVH (item 41)
- Left anterior fascicular block (item 46)
- Chronic lung disease (item 81): Q waves appear in inferior and/or right and mid-precordial leads
- Amyloid, sarcoid, and other infiltrative cardiomyopathic diseases: Electrically active tissue replaced by inert substance
- Cardiomyopathy
- Chest deformity (e.g., pectus excavatum)
- Pulmonary embolism (item 82): Q wave in lead III and sometimes aVF, but Q waves in II are rare
- Myocarditis
- Myocardial tumors
- Hyperkalemia (item 74)
- Pneumothorax: QS complex in right precordial leads
- Pancreatitis

- Lead reversal (item 03)
- Corrected transposition
- Muscular dystrophy
- Mitral valve prolapse: Rare Q wave in III and aVF
- Myocardial contusion: Q waves in areas of intramyocardial hemorrhage and edema
- Left/right atrial enlargement: Prominent atrial repolarization wave (Ta) can depress the PR segment and mimic Q waves
- Atrial flutter (item 18): Flutter waves may deform the PR segment and simulate Q waves
- Dextrocardia (item 80)

R Wave Progression (Precordial Leads)

Early R wave progression (tall R wave in V_1, V_2; R/S > 1)

- RVH (item 41)
- Posterior MI (items 59, 60)
- RBBB (item 43)
- Wolff-Parkinson-White pattern (item 34)
- Normals
- Duchenne's muscular dystrophy

Poor R wave progression (first precordial lead where R wave amplitude ≥ S wave amplitude = V_5 or V_6)

- Normals (abnormal lead placement)
- Anterior or anteroseptal MI (items 53, 54)
- Dilated or hypertrophic cardiomyopathy
- LVH (item 40)
- Chronic lung disease (item 81)
- Cor pulmonale (item 82)
- RVH (item 41)
- Left anterior fascicular block (item 45)

Reverse R wave progression (decreasing R wave amplitude across precordial leads)

- Anterior MI (items 53, 54)
- Dextrocardia (item 80)

QRS Morphology

Initial slurring of R wave (delta wave)

- Wolff-Parkinson-White pattern (item 34)

Terminal notching (of R or S wave)

- Hypothermia (Osborne wave; item 88)
- Early repolarization (item 61)
- Pacemaker spike (failure to sense; item 94)
- Atrial flutter (item 18): Flutter waves may be superimposed on QRS

ST Segment

ST segment elevation

- Myocardial injury (item 65): Convex upward ST elevation localized to a *few* leads and terminates with an inverted T (unless hyperacute peaked T wave). Reciprocal ST depression evident in other leads. Q waves frequently present. ST & T wave changes *evolve*, and T wave becomes inverted *before* ST segment returns to baseline
- Acute pericarditis (item 84): Widespread ST elevation (I-III, aVF, V_3-V_6) *without reciprocal* ST depression in other leads except aVR. No Q wave. PR segment depression is sometimes present. ST-T wave changes evolve; T wave often becomes inverted *after* ST segment returns to baseline. Note: Pericarditis (and ST elevation) may be focal
- Ventricular aneurysm: ST elevation usually with deep Q wave or QS in same leads; ST & T wave changes persist and are *stable* over a long period of time
- Early repolarization (item 61): Concave upward ST elevation that ends with an upward T wave, with notching on the downstroke of the R wave. T waves are usually large and symmetrical. ST-T wave changes are *stable* over a long time period
- LVH (item 40)
- Bundle branch block (items 43, 47)
- Central nervous system disease (item 86)
- Apical hypertrophic cardiomyopathy (item 85)
- Hyperkalemia (item 74)
- Acute cor pulmonale (item 82)
- Myocarditis
- Myocardial tumor

ST segment depression

- Myocardial ischemia (item 64): horizontal or downsloping
- Repolarization changes secondary to ventricular hypertrophy (item 67) or bundle branch block (items 43, 47)
- Digitalis effect (item 70)
- "Pseudodepression" due to superimposition of atrial flutter waves or prominent atrial repolarization wave (as seen with atrial enlargement, pericarditis, atrial infarction) on the ST segment
- Central nervous system disorder (item 86)

- Hypokalemia (item 75)
- Antiarrhythmic drug effect (item 72)
- Mitral valve prolapse

Nonspecific ST segment changes

- Organic heart disease
- Drugs (e.g., quinidine)
- Electrolyte disorders (e.g., hypokalemia, item 75)
- Hyperventilation
- Myxedema (item 87)
- Stress
- Pancreatitis
- Pericarditis (item 84)
- Central nervous system disorders (item 86)
- LVH (item 40)
- RVH (item 41)
- Bundle branch block (items 43, 44, 47, 48)
- Healthy adults (normal variant) (item 02)

T Wave

Tall peaked T waves

- Hyperacute MI
- Angina pectoris
- Normal variant (item 02): Usually effects mid-precordial leads
- Hyperkalemia (item 74): More common when the rise in serum potassium is acute
- Intracranial bleeding (item 86)
- LVH (item 40)
- RVH (item 41)
- LBBB (item 47)
- Superimposed P wave from APC, sinus rhythm with marked first-degree AV block, complete heart block, etc.
- Anemia

Deeply inverted T waves

- Myocardial ischemia (item 64)
- LVH (items 40, 67)
- RVH (items 41, 67)
- Central nervous system disorder (item 86)
- Wolff-Parkinson-White pattern (item 34)

Nonspecific T waves

- Persistent juvenile pattern: T wave inversion in V_1-V_3 in young adults
- Organic heart disease
- Drugs (e.g., quinidine)
- Electrolyte disorders (e.g., hypokalemia, item 75)
- Hyperventilation
- Myxedema (item 87)
- Stress
- Pancreatitis
- Pericarditis (item 84)
- Central nervous system disorders (item 86)
- LVH (item 40)
- RVH (item 41)
- Bundle branch block (items 43, 44, 47, 48)
- Healthy adults (normal variant) (item 02)

QT Interval

Long QT interval

- **Acquired conditions**
  - Drugs (quinidine, procainamide, disopyramide, amiodarone, sotalol, dofetilide, azimilide, phenothiazines, tricyclics, lithium)
  - Hypomagnesemia
  - Hypocalcemia (item 77)
  - Marked bradyarrhythmias
  - Intracranial hemorrhage (item 86)
  - Myocarditis
  - Mitral valve prolapse
  - Myxedema (item 87)
  - Hypothermia (item 88)
  - Liquid protein diets
- **Congenital disorders**
  - Romano-Ward syndrome (normal hearing)
  - Jervell and Lange-Nielson syndrome (deafness)

Short QT interval

- Hypercalcemia (item 76)
- Hyperkalemia (item 74)
- Digitalis effect (item 70)
- Acidosis
- Vagal stimulation
- Hyperthyroidism
- Hyperthermia

U Wave

Prominent U wave

- Hypokalemia (item 75)
- Bradyarrhythmias
- Hypothermia (item 88)
- LVH (item 40)
- Coronary artery disease
- Drugs (digitalis, quinidine, amiodarone, isoproterenol)

Inverted U wave

- LVH (item 40)
- Severe RVH (item 41)
- Myocardial ischemia

PP Pause > 2.0 seconds

- Sinus pause or arrest (item 11): Due to transient failure of impulse formation at the SA node. Sinus rhythm resumes at a PP interval that is not a multiple of the basic sinus PP interval
- Sinus arrhythmia (item 08): Phasic gradual change in PP interval
- Second-degree sinoatrial exit block, Mobitz I (Wenckebach) (item 12): Progressive shortening of PP interval until a P wave fails to appear
- Second-degree sinoatrial exit block, Mobitz II (item 12): Pause followed by resumption of sinus rhythm at a PP interval that is a multiple (e.g., 2x, 3x, etc.) of the basic sinus rhythm
- Third-degree sinoatrial exit block (item 12): Complete failure of sinoatrial conduction; cannot be differentiated from complete sinus arrest on surface ECG
- Abrupt change in autonomic tone
- "Pseudo" sinus pause due to nonconducted APCs (item 13): P wave appears to be absent but is actually buried in the T wave — look for subtle deformity of the T wave just preceding the pause to detect nonconducted APCs

Group Beating

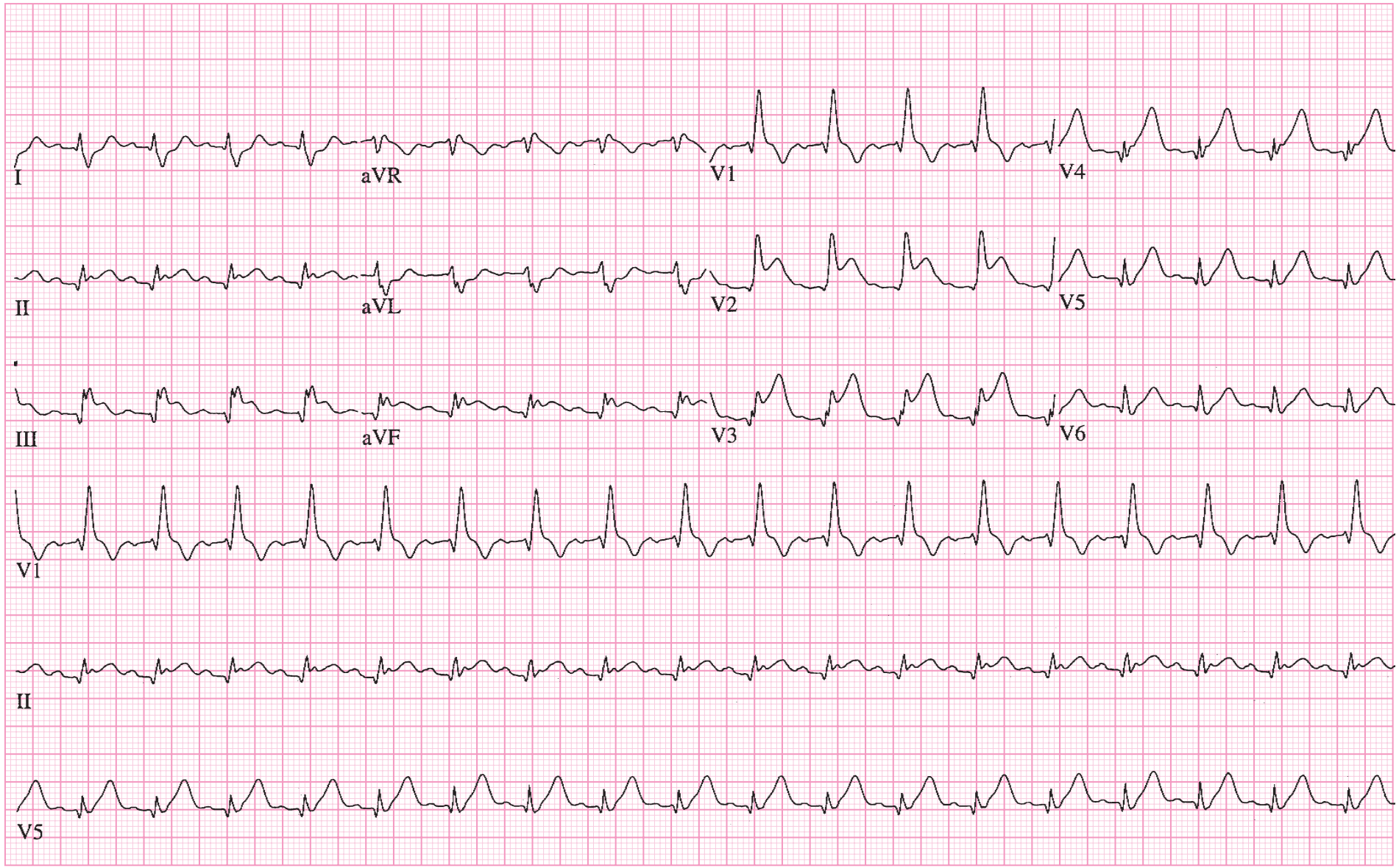
- Mobitz Type I, second-degree AV block (item 30)
- Mobitz Type II, second-degree AV block (item 31)
- Blocked APCs (item 13)
- Concealed His-bundle depolarizations

— Section 3 —

ECG CASES AND QUIZZES

(See p. 555 for index of cases by diagnosis)

ECG 1. 71-year-old male with acute shortness of breath:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

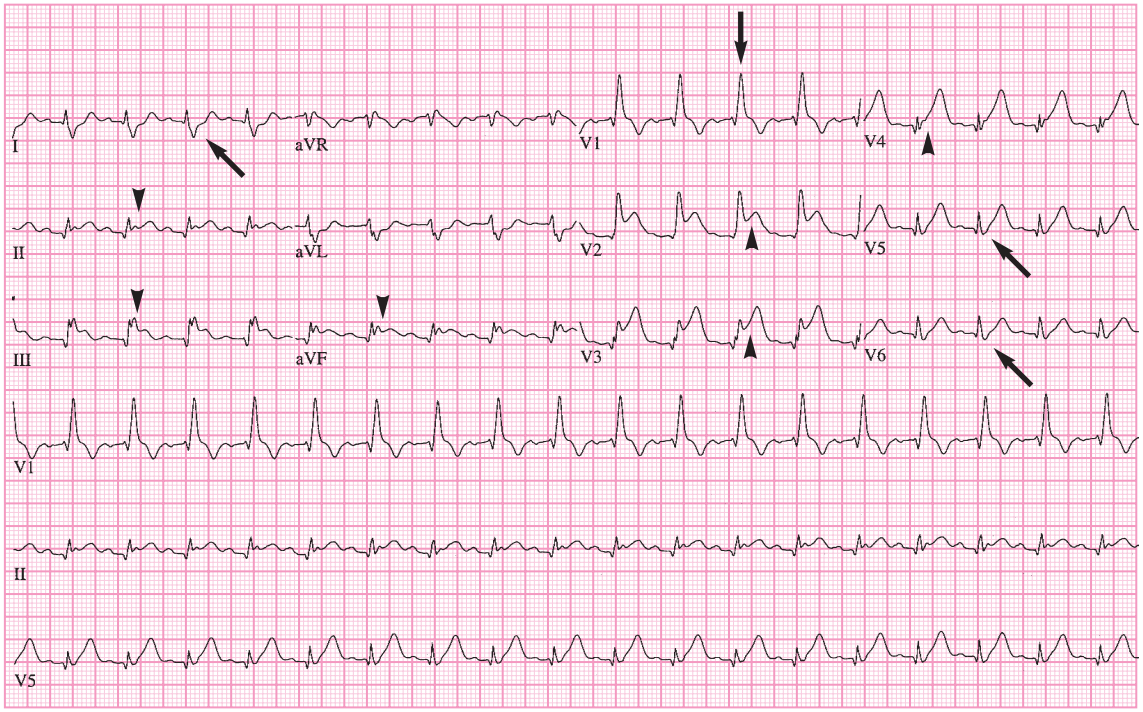
PACED RHYTHMS

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

ECG 1 was obtained from a 71-year-old male with acute shortness of breath immediately upon arrival to the emergency department. The ECG shows sinus tachycardia at 111 beats/minute, RBBB (widened rsR' complex in lead V_1 with wide S waves in leads I, V_5, V_6; arrows), and left posterior fascicular block (axis > +100°). Striking ST segment elevation (arrowheads) with associated Q waves are present in leads V_2-V_4, II, III and aVF, diagnostic of acute anterior myocardial infarction and acute inferior myocardial infarction. Acute occlusion of a large left anterior descending coronary artery that "wraps around" the left ventricular apex and supplies a substantial portion of the inferior wall can produce the appearance of simultaneous acute anterior and inferior infarctions.

Codes:

10	Sinus tachycardia
43	RBBB, complete
46	Left posterior fascicular block
53	Anterior or anteroseptal Q wave MI (age recent or acute)
57	Inferior Q wave MI (age recent or acute)
65	ST and/or T wave abnormalities suggesting myocardial injury



Questions: ECG 1

1. Significant ST segment elevation consistent with myocardial injury or infarction is defined as:

   a. ≥ 1 mm ST elevation in leads V_1, V_2, or V_3
   b. ≥ 2 mm ST elevation in leads V_1, V_2, or V_3
   c. ≥ 2 mm ST elevation in other leads
   d. ≥ 1 mm ST in other leads

2. Repolarization abnormalities that suggest acute or recent myocardial infarction include:

   a. Peaked T waves followed by T wave inversion
   b. ST-elevation followed by peaked T waves
   c. Deeply inverted T waves
   d. Dominant R wave and ST depression in leads V_1 - V_3

3. Match the following types of acute myocardial infarction with their associated ST segment changes:

a. Anterolateral MI	1. ST elevation in I, aVL
b. Lateral MI	2. ST elevation in V_1 - V_3
c. Anterior MI	3. ST elevation in V_4 - V_6
d. Posterior MI	4. ST depression in V_1 - V_3

4. Which parameters on initial ECG obtained independently predict 30-day all-cause mortality in acute myocardial infarction:

   a. Sinus tachycardia
   b. Sum of absolute ST segment deviations (elevation and/or depression)
   c. QRS duration > 100 msec
   d. Rightward axis

Answers: ECG 1

1. Significant ST elevation consistent with myocardial injury/infarction requires the presence of ST elevation at the J-point in two or more contiguous leads, including ≥ 2 mm in leads V_1, V_2, or V_3 , and ≥ 1 mm in other leads. The ST configuration of myocardial injury/infarction is classically described as *convex* upward ("outpouching"). In contrast, the ST configuration of acute pericarditis or normal variant early repolarization is *concave* upward. (Answer: b, d)

2. The repolarization abnormalities of acute injury/infarction occur in a predictable sequence. Hyperacute T waves – tall peaked T waves in the region of the infarct – are seen in the first few minutes of the event. ST-elevation appears next, and generally lasts for several hours or until the infarct artery is opened. The repolarization abnormalities often evolve into inverted T waves in the affected leads within several hours to days. Instead of Q waves and ST elevation, acute posterior MI presents with mirror-image changes in the anterior precordial leads (V_1 - V_3), including dominant R waves (mirror-image of abnormal Q waves) and horizontal ST segment depression (mirror-image of

ST elevation). (Answer: a, c, d)

3. (Answer: a-3, b-1, c-2, d-4)

4. A large study evaluating the initial ECG as a predictor for 30-day all cause mortality in acute myocardial infarction found that sinus tachycardia and the sum of absolute ST segment deviations were the most powerful predictors of outcome. A QRS duration > 100 msec was also shown to be an independent adverse prognostic factor. QRS axis did not affect outcome. (Hathaway, WR, et al. JAMA, 1996, 273:387-391.) (Answer: a, b, c)

— Quick Review 1 —

Sinus tachycardia	
• Rate > ____ per minute	100
• P wave amplitude often (increases/decreases) and PR interval often (increases/decreases) with increasing heart rate	increases decreases
RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ____ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 > ____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ____	I, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
• RBBB (does/does not) interfere with the ECG diagnosis of ventricular hypertrophy or Q wave MI	does not

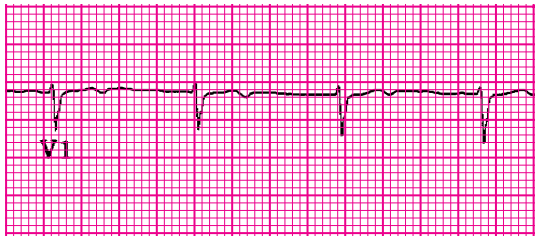
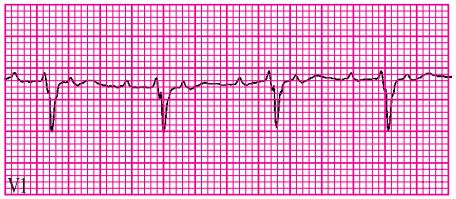
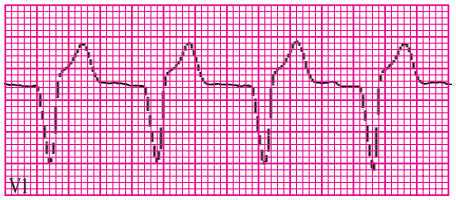
— Quick Review 1 —

Left posterior fascicular block	
• (Left/right) axis deviation with mean QRS axis between ___ and ___ degrees	right 100, 180
• QRS duration between ___ and ___ seconds	0.08, 0.10
• No other factor responsible for ___ axis deviation	right
Anteroseptal MI, probably acute or recent	
• Abnormal Q or QS deflection and ST elevation in leads ____ (and sometimes V_4)	V_1-V_3
• The presence of a Q wave in lead ____ distinguishes anteroseptal from anterior infarction	V_1
Inferior MI, probably acute or recent	
• Abnormal Q waves and ST elevation in at least two of leads ____	II, III, aVF
• Associated ST depression is usually evident in leads I, aVL, V_1-V_3 (true/false)	true

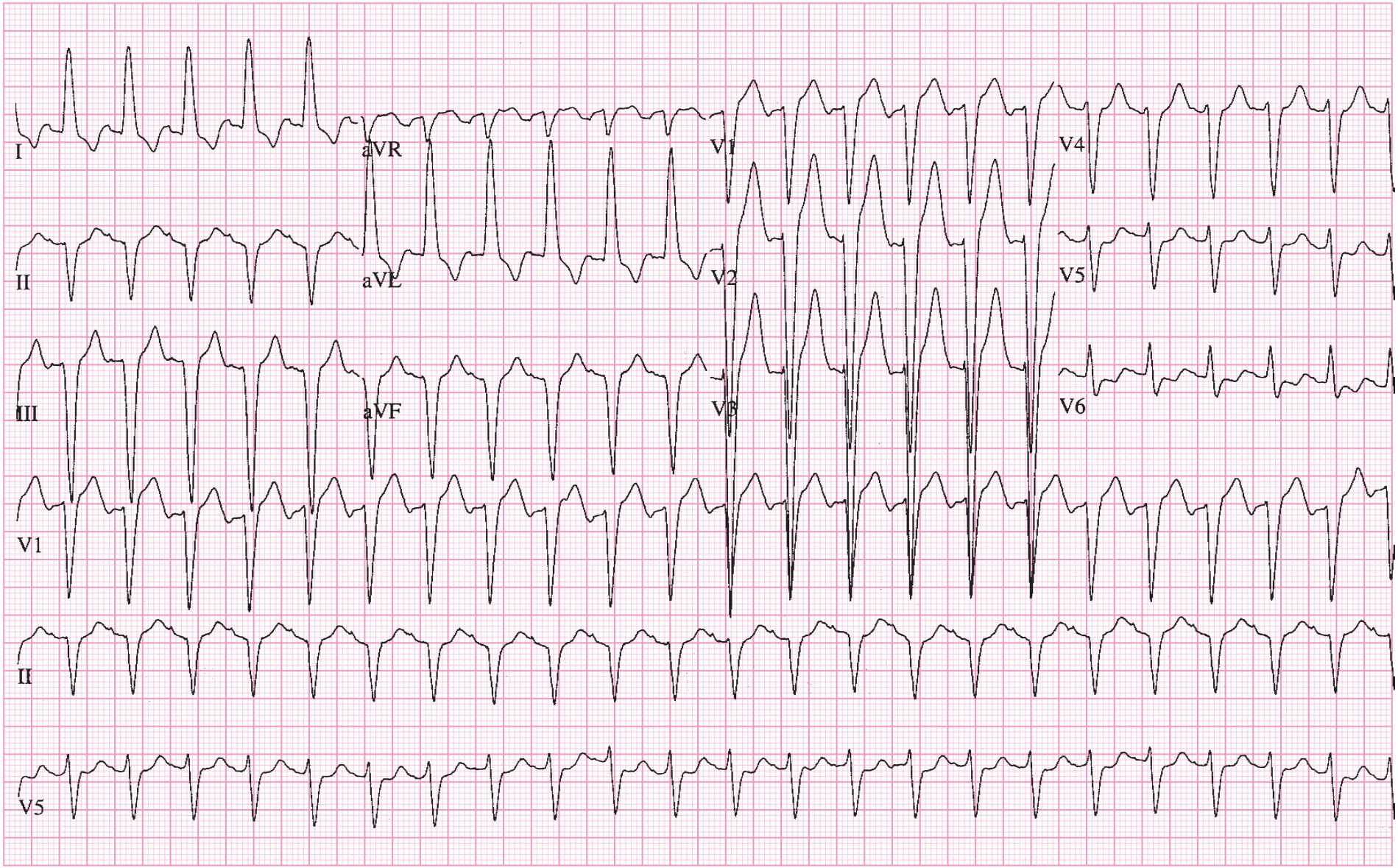
— POP QUIZ —

Rhythm Recognition: HR < 100; Regular RR Interval

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Accelerated AV junctional rhythm. Description: Regular rhythm with junctional QRS complexes (typically narrow) occurring at a rate of > 60 per minute. P waves may precede the QRS by ≤ 0.11 seconds (retrograde atrial activation), may be buried in the QRS (and not visualized), or may follow the QRS complex (look for deformity in ST segment). AV junctional rhythms are often associated with isorhythmic AV dissociation and retrograde atrial activation (not apparent in this ECG).
	Answer: Atrial tachycardia with block. Description: Regular ectopic atrial (nonsinus) rhythm usually at a rate of 150-240 per minute (can be as low as 100 per minute) with occasional nonconducted P waves. Isoelectric intervals are present between all P waves in all leads (unlike atrial flutter). Primary causes include digoxin toxicity (75%) and organic heart disease (25%).
	Answer: Accelerated idioventricular rhythm (AIVR). Description: Regular or slightly irregular ventricular (wide QRS complex) rhythm at a rate of 60-110 per minute. QRS morphology is similar to ventricular premature complexes (VPCs). Competition between normal sinus and ectopic ventricular rhythms often results in AV dissociation, ventricular capture complexes, and fusion beats. AIVR is seen in myocardial ischemia, digitalis toxicity, following coronary reperfusion, and occasionally in normals.

ECG 2. 73-year-old female 24 hours after elective hip surgery:



GENERAL FEATURES

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- ☐ 02. Borderline normal ECG or normal variant
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- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 08. Sinus arrhythmia
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- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 20. AV junctional premature complexes
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- ☐ 34. Wolff-Parkinson-White pattern
- ☐ 35. AV dissociation

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- ☐ 37. Right axis deviation (> +100°)
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- ☐ 39. Low voltage
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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
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- ☐ 72. Antiarrhythmic drug effect
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- ☐ 74. Hyperkalemia
- ☐ 75. Hypokalemia
- ☐ 76. Hypercalcemia
- ☐ 77. Hypocalcemia
- ☐ 78. Atrial septal defect, secundum
- ☐ 79. Atrial septal defect, primum
- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
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- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

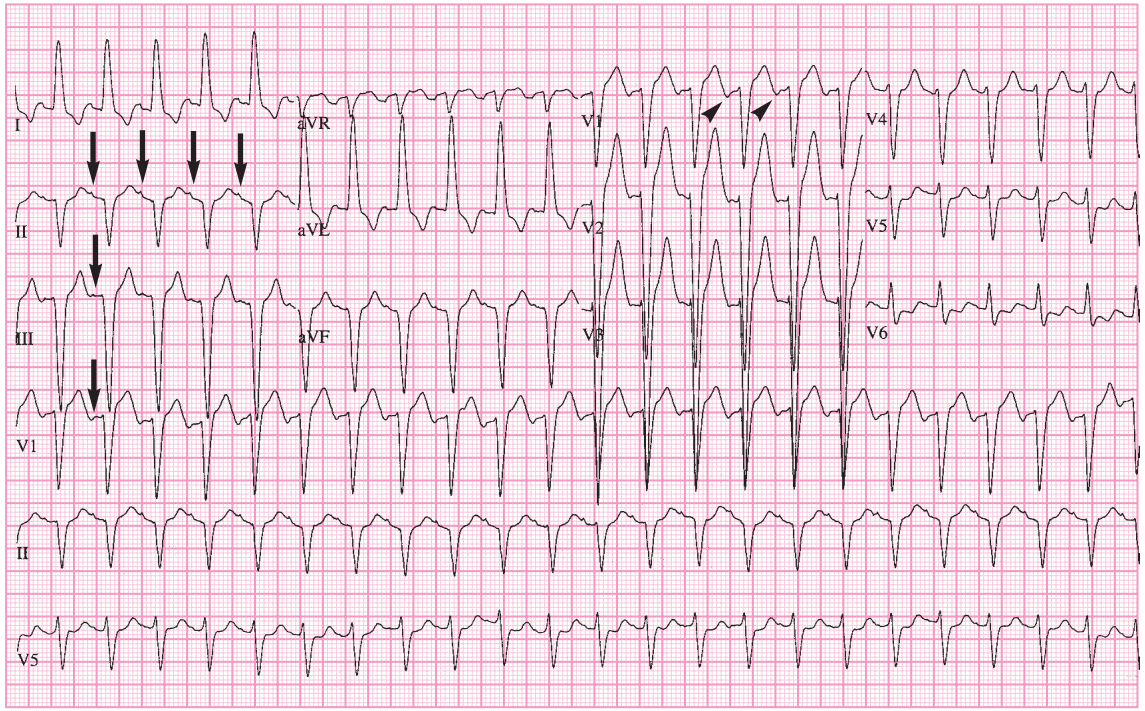
PACED RHYTHMS

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

ECG 2 was obtained in a 73-year-old female 24 hours after elective hip surgery. The ECG shows a rapid wide-complex tachycardia at a rate of 138 beats/minute. On a closer inspection, sinus P waves (arrows) and left bundle branch block (broad monophasic R wave in lead V_1; QRS duration 134 msec) are present. The P waves in lead V_1 (arrowhead) meet criteria for left atrial enlargement. Left axis deviation (QRS axis –54°) is also present.

Codes:

06	Left atrial abnormality/enlargement
10	Sinus tachycardia
36	Left axis deviation (> –30°)
47	LBBB, complete



Questions: ECG 2

1. A QRS duration ≥ ___ seconds is necessary for the diagnosis of complete LBBB:

   a. 0.10
   b. 0.11
   c. 0.12
   d. 0.13

2. LBBB is commonly seen in normal hearts:

   a. True
   b. False

3. Non-voltage related changes often associated with left ventricular hypertrophy include all the following except:

   a. Left atrial enlargement/abnormality
   b. Left axis deviation
   c. Intraventricular conduction disturbance
   d. Prominent U waves
   e. Sinus arrhythmia

4. LBBB interferes with the ECG diagnosis of:

   a. QRS axis
   b. Left ventricular hypertrophy
   c. Right ventricular hypertrophy
   d. Acute MI

Answers: ECG 2

1. Left bundle branch block is diagnosed when the QRS duration is ≥ 0.12 seconds (120 msec) and typical QRS morphology is present. When LBBB morphology is present and the QRS duration measures > 0.10 seconds but < 0.12 seconds, incomplete LBBB should be coded. (Answer: c)

2. LBBB often occurs in various forms of organic heart disease, including ischemic and non-ischemic cardiomyopathy, valvular heart disease, LVH, and congenital heart disease. It is rarely seen in normal hearts. (Answer: False)

3. Non-voltage ECG changes associated with LVH include left atrial abnormality/enlargement, left axis deviation, IVCD, QRS prolongation, abnormal Q waves in leads III and aVF, left axis deviation, prominent U waves, and repolarization abnormalities. Sinus arrhythmia (longest and shortest PP intervals vary by > 0.16 seconds or 10%) is a common finding on normal ECG's that tends to occur in younger and healthier individuals and is not associated with LVH. (Answer: e)

4. LBBB interferes with determination of QRS axis and identification of ventricular hypertrophy and acute MI. Although the formal diagnosis of LVH should not be made in the setting LBBB, echocardiographic and pathological studies show that ~ 80% patients with LBBB have abnormally increased LV mass. (Answer: all)

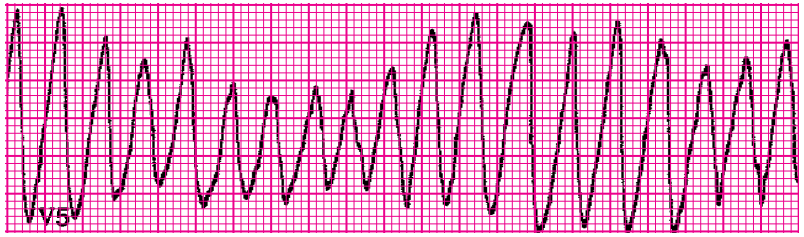
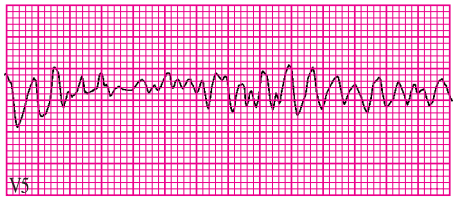
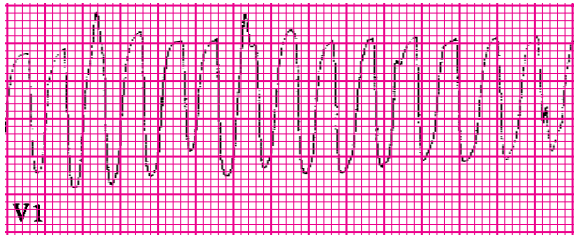
— Quick Review 2 —

Left atrial abnormality	
• Notched P wave with a duration ≥ ____ seconds in leads II, III or aVF, *or*	0.12
• Terminal negative portion of the P wave in lead V_1 ≥ 1 mm deep and ≥ ____ seconds in duration	0.04
Left axis deviation (> –30°)	
• Mean QRS axis between ___ and ___ degrees	-30, -90
LBBB, complete with ST-T waves suggestive of acute myocardial injury or infarction	
• ST elevation ≥ ____ mm concordant to (same direction as) the major deflection of the QRS	1
• ST depression ≥ ____ mm in V_1, V_2, or V_3	1
• ST elevation ≥ ____ mm discordant with (opposite direction to) the major deflection of the QRS	5

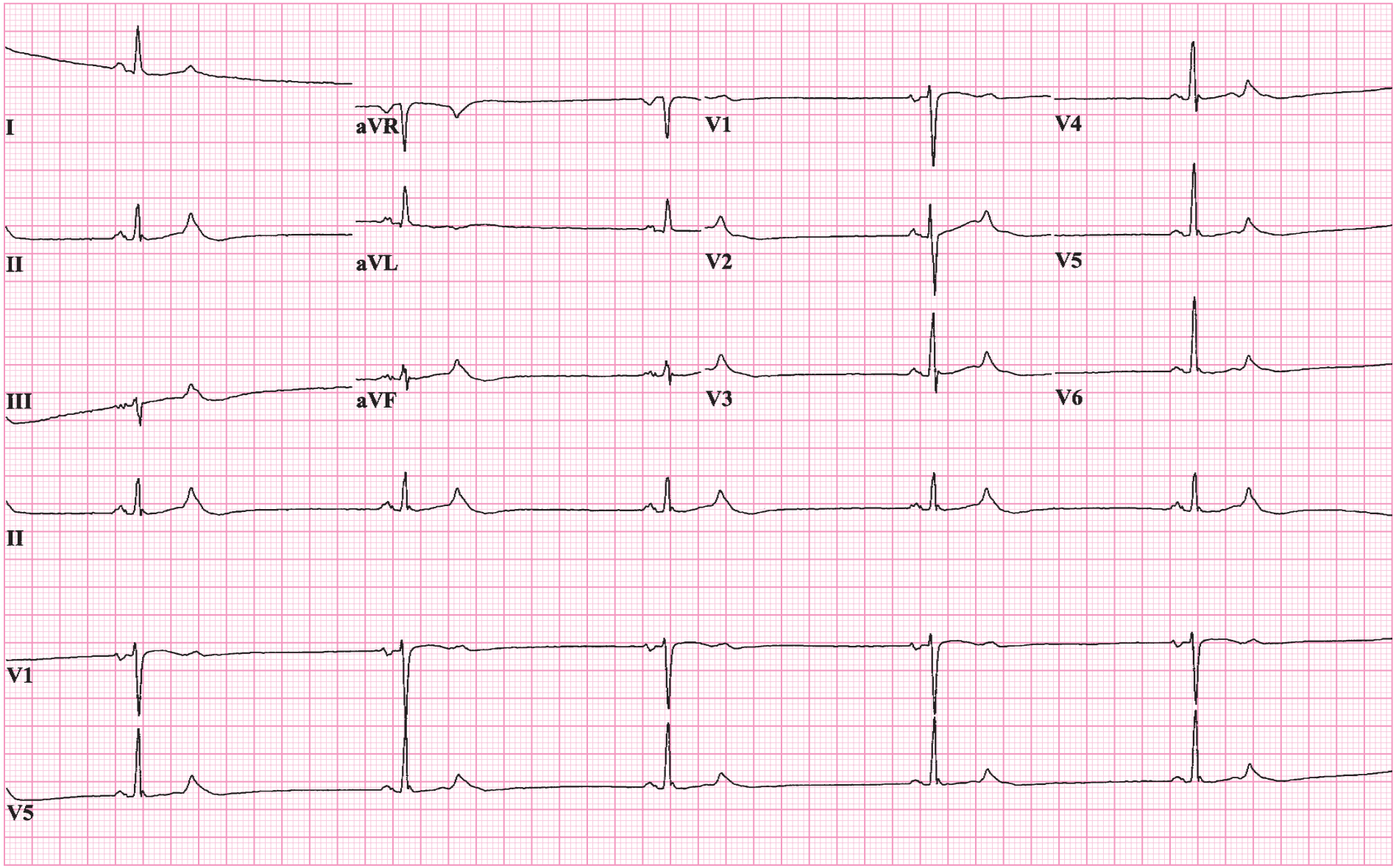
— POP QUIZ —

Rhythm Recognition: Wide QRS Tachycardia

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Torsade de pointes ("twisting of the points"). Description: Polymorphic wide QRS complex tachycardia with cycles of three or more beats occurring with alternating polarity in a sinusoidal pattern. Occurs in the setting of a prolonged QT interval, and is often preceded by long-short R-R cycles. Can degenerate into ventricular fibrillation. Ventricular tachycardia (VT) of similar morphology but without QT prolongation is called "polymorphic VT," not torsade de pointes.
	Answer: Ventricular fibrillation. Description: Extremely rapid and irregular ventricular rhythm demonstrating chaotic, irregular deflections of varying amplitude and contour, without distinct P waves, QRS complexes, or T waves. Lethal rhythm requiring immediate defibrillation.
 Patient brushing teeth; asymptomatic	Answer: Artifact. Description: Rapid arm motion or lead movement (e.g., toothbrushing, hair brushing) can simulate VPCs or ventricular tachycardia, and commonly fools telemetry technicians and sets off monitor alarms. Other causes of artifact include AC electrical interference (60 cycles per second), wandering baseline, skeletal muscle fasciculations/shivering (can simulate atrial fibrillation), tremor (can simulate atrial flutter), electrocautery, and IV infusion pump (can give appearance of rapid P waves).

ECG 3. 61-year-old female with light-headedness:



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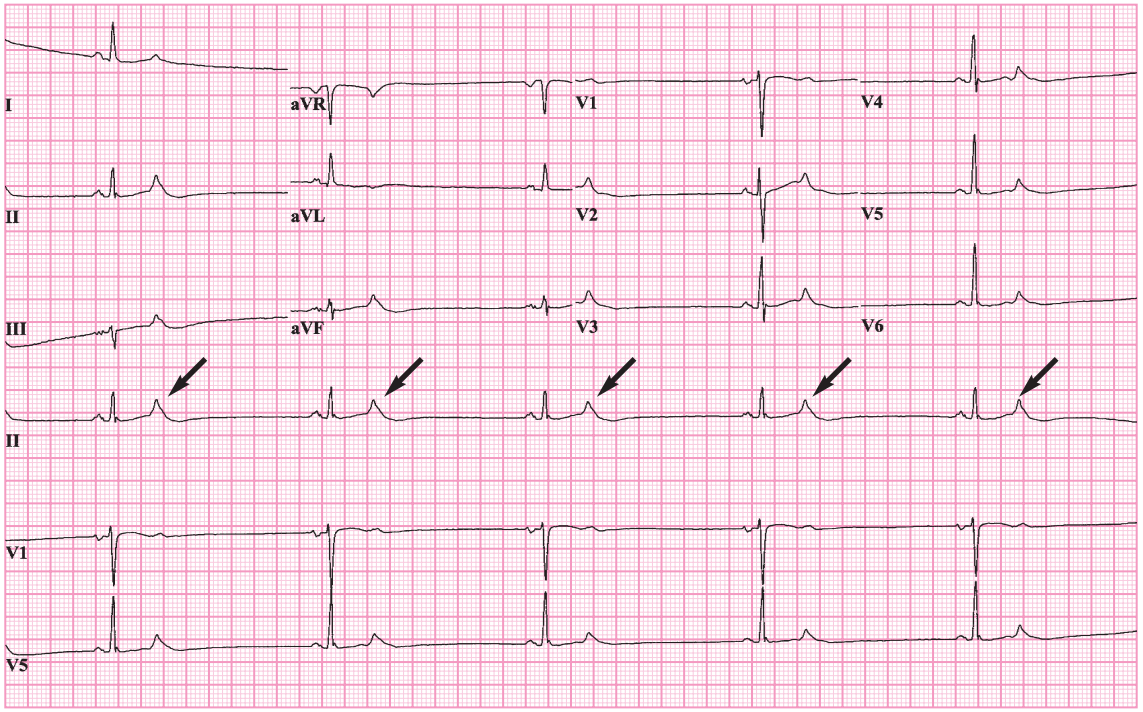
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 3 was obtained in a 61-year-old female with light-headedness. At first glance, the ECG appears to demonstrate extreme sinus bradycardia at a rate of 30 beats/minute. On closer inspection, the unusually-shaped T waves are actually deformed by superimposed P waves (arrows); these atrial premature contractions (APCs), which occur in a bigeminal pattern, are blocked in the AV node and do not conduct to the ventricle. Nonconducted APCs are the most common cause of sinus pauses on the ECG. When a sinus pause is present, it is important to look for a deformed T wave immediately preceding the pause to identify the presence of a nonconducted APC. Sinus bradycardia should not be coded since the slow rhythm is due to the blocked APC's.

Codes:

07	Sinus rhythm
13	Atrial premature complexes



Questions: ECG 3

1. Nonconducted APCs are usually associated with a:

   a. Compensatory pause
   b. Noncompensatory pause

2. The QRS morphology of aberrantly conducted APCs is most often:

   a. Similar to QRS complex during sinus rhythm
   b. RBBB pattern
   c. LBBB pattern

Answers: ECG 3

1. A nonconducted APC manifests as a premature P wave with abnormal morphology that is not followed by a QRS-T complex. It occurs when the APC arrives at an AV node that is refractory to conduction. The P wave is often hidden in the preceding T wave — when you see an RR pause, look for a deformed T wave immediately preceding the pause to identify the presence of a nonconducted APC. The sinus node is usually depolarized and reset so that the next P wave occurs one cycle length after the nonconducted P wave. The resulting "noncompensatory pause" manifests as a premature P wave to subsequent P wave interval equal to one normal PP interval. Uncommonly, a compensatory pause may occur when sinoatrial (SA) "entrance block" is present and the SA node is not reset. (Answer: b)

2. The QRS morphology of aberrantly conducted APC's is most often RBBB pattern, but can manifest as LBBB pattern or variable widening/distortion of the QRS. The longer refractory period of the right bundle (compared to the left bundle) increases the likelihood that an APC will conduct down the left bundle while the right bundle is still refractory. (Answer: b)

— Quick Review 3 —

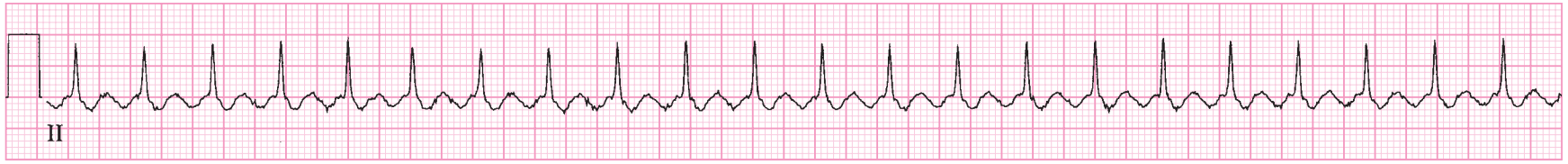
Atrial premature complexes	
• P wave is (normal/abnormal) in configuration	abnormal
• QRS complex is (similar/different) in morphology to the QRS complex present during sinus rhythm	similar
• PR interval may be normal, increased, or decreased (true/false)	true
• The post-extrasystolic pause is usually (compensatory/noncompensatory)	noncompensatory

— POP QUIZ —

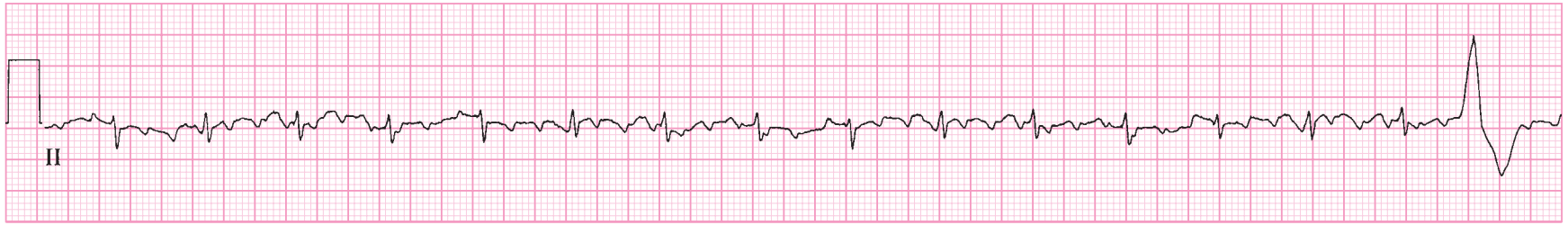
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

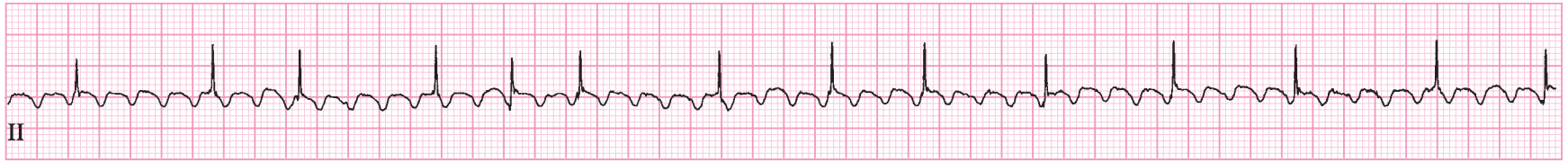


B.



C.

D.



Answer: Tracings A, B, and D show atrial flutter. Tracing A represents atrial flutter with 2:1 AV block, evident from the deeply inverted flutter waves preceding each QRS complex in lead II along with a similar flutter waves superimposed on the ST segments of the preceding beats. The use of calipers will help to verify an atrial flutter rate just less than 300 beats per minute. In tracings B and D, flutter waves are evident between QRS complexes. Tracing C shows sinus tachycardia and is the imposter. While the T wave and the tall sinus P wave suggest atrial flutter, placing calipers to determine the interval between and T wave and P wave and then marching this interval through the tracing fails to show the regularity seen with atrial flutter.

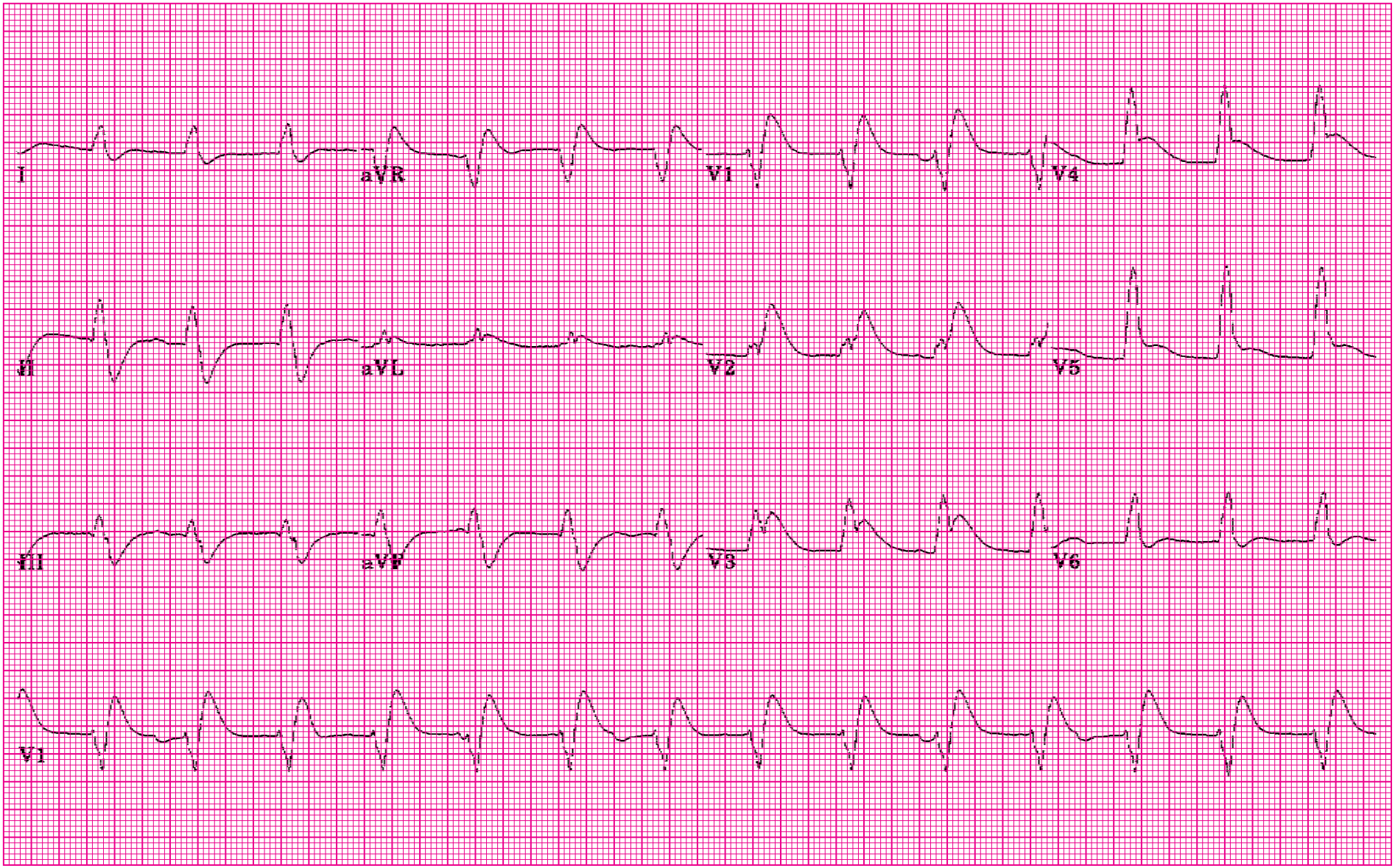
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the ECG diagnosis that best corresponds to the ECG features listed below (see score sheet for options)

ECG Features	Diagnosis
• Non-sinus P wave • Rate < 100 per minute • PR interval > 0.11 seconds	Ectopic atrial rhythm
• Rate < 100 per minute • P waves with ≥ 3 morphologies • PR, RR, and RP intervals vary	Wandering atrial pacemaker
• Resultant ECG mimics dextrocardia with inversion of the P-QRS-T in leads I and aVL	Incorrect lead placement
• Sinus P wave • Longest and shortest PP intervals vary by > 0.16 seconds or 10%	Sinus arrhythmia
• PP interval > 1.6 - 2.0 seconds • Resumption of sinus rhythm at a PP interval that is not a multiple of the basic sinus PP interval	Sinus pause or arrest
• Sinus P wave • Some sinus impulses fail to reach the atria • " Group beating" with: (1) Shortening of the PP interval prior to absent P wave (2) Constant PR interval (3) PP pause less than twice the normal PP interval	Mobitz Type I, second-degree sinoatrial exit block
• Sinus P wave • Some sinus impulses fail to reach the atria • Constant PP interval followed by a pause that is a multiple (2x, 3x, etc.) of the normal PP interval	Mobitz Type II, second-degree sinoatrial exit block

ECG 4. 82-year-old female with chest pain:



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- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

- ☐ 61. Normal variant, early repolarization
- ☐ 62. Normal variant, juvenile T waves
- ☐ 63. Nonspecific ST and/or T wave abnormalities
- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
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- ☐ 70. Digitalis effect
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- ☐ 78. Atrial septal defect, secundum
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- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
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- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

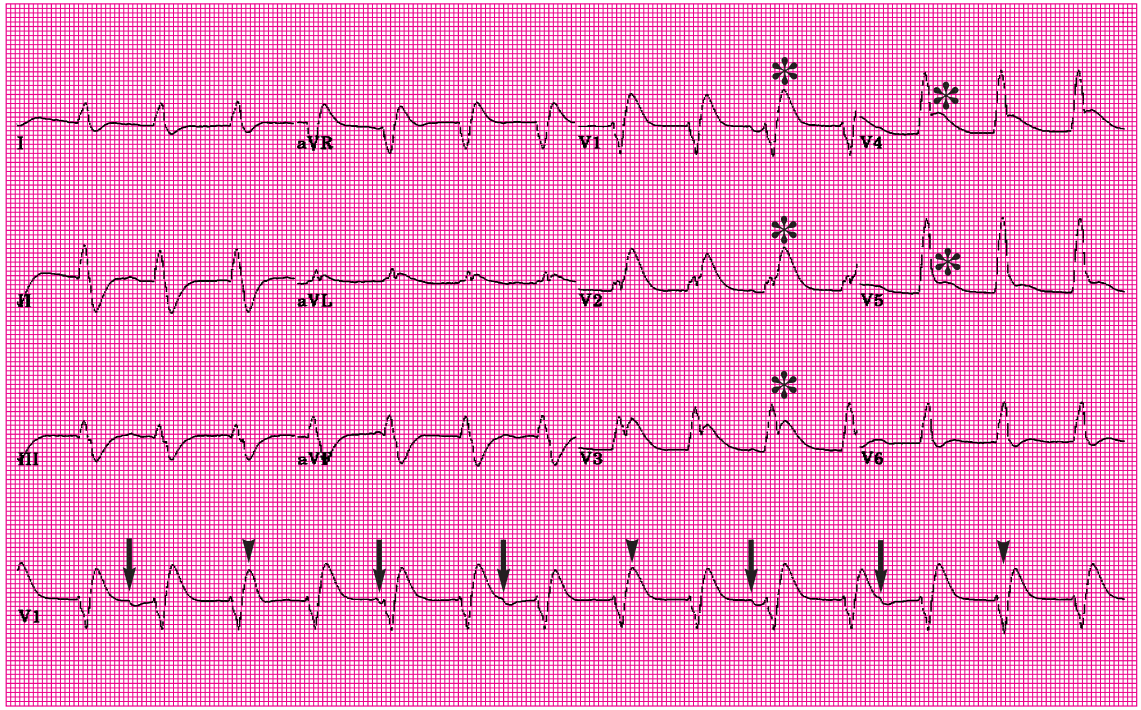
PACED RHYTHMS

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

ECG 4 was obtained from an 82-year-old female with chest pain. The ECG shows a regular, wide QRS complex rhythm at a rate of 94 beats/minute, consistent with accelerated idioventricular rhythm. Sinus bradycardia is also present at a rate of 56 beats/minute (arrows mark P waves, which are sometimes hidden in the QRS complex [arrowheads] but march through the tracing). The sinus rhythm and AIVR are independent of each other, resulting in AV dissociation. LBBB pattern represents electrical activation from the idioventricular rhythm, not true bundle branch block. Marked ST segment elevation is evident in the anterolateral leads (asterisks), consistent with acute myocardial injury; the presence of LBBB pattern makes it difficult to diagnose acute myocardial infarction in a specific location.

Codes:

09	Sinus bradycardia
26	Accelerated idioventricular rhythm
35	AV dissociation
65	ST and/or T wave abnormalities suggesting myocardial injury



Questions: ECG 4

1. Accelerated idioventricular rhythm (AIVR) presents with:

   a. Rates up to but not exceeding 100 BPM
   b. Wide QRS complexes
   c. Occasional fusion beats when there is a competing sinus rhythm

2. ST segment elevation can be due to:

   a. Pericarditis
   b. Acute myocardial infarction
   c. Digitalis
   d. Hyperkalemia
   e. Left ventricular hypertrophy
   f. Intracerebral hemorrhage
   g. Acute cor pulmonale
   h. Hypocalcemia
   i. Early repolarization

Answers: ECG 4

1. AIVR tends to occur at rates between 60-100 BPM, but can occur at rates up to 110 BPM. When AIVR competes with sinus rhythm, fusion beats (QRS complexes intermediate in morphology between the two rhythms) sometimes occur. (Answer: b, c)

2. There are numerous causes of ST segment elevation, including acute myocardial infarction, pericarditis, early repolarization, ventricular aneurysm, myocarditis, LVH, acute cor pulmonale, LBBB, hypertrophic cardiomyopathy, intracerebral hemorrhage, and neoplastic invasion of the heart. Digitalis causes sagging ST segment depression, not ST elevation. Hypocalcemia can lengthen the ST segment, but does not cause ST elevation or depression. (Answer: all except c, h)

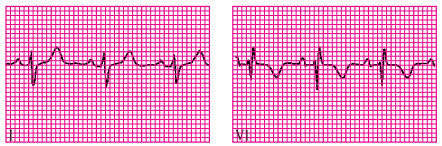
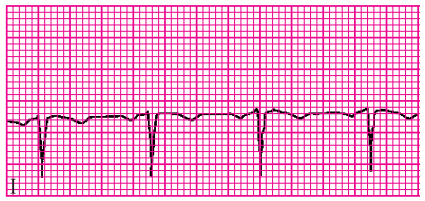
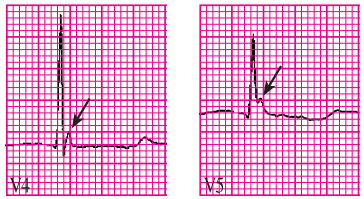
— Quick Review 4 —

Accelerated idioventricular rhythm	
• Highly irregular ventricular rhythm (true/false)	false
• Ventricular rate of ____ per minute	60-120
• QRS morphology is similar to ____	VPCs
• Ventricular ____ complexes, ____ beats, and AV ____ are common	capture, fusion dissociation
AV dissociation	
• Atrial and ventricular rhythms are ____ of each other	independent
• Ventricular rate is (</≥) than the atrial rate	≥

— POP QUIZ —

Pattern Recognition: Clinical Disorders

Instructions: Determine the clinical diagnosis associated with each of the following ECGs.

ECG	Diagnosis	Answer
	a. Atrial septal defect, primum b. Atrial septal defect, secundum c. Dextrocardia d. Intracerebral hemorrhage e. Limb lead reversal f. Precordial lead reversal g. Digitalis effect h. Hypothermia i. Pericardial effusion	*Ostium secundum atrial septal defect (ASD)* represents 70% of all ASDs, and results from deficient tissue in the region of the fossa ovalis. The diagnosis is suggested by an RSR' or rSR' complex with a QRS duration < 0.11 seconds in V_1, incomplete RBBB, right axis deviation ± right ventricular hypertrophy. Right atrial abnormality is present in 30%, and AV block develops in < 20%. (Answer: b)
 Normal R wave progression		*Reversal of right and left arm leads* results in inversion of the P-QRS-T in leads I and aVL (mimicking dextrocardia in limb leads), transposition of leads II and III, and transposition of leads aVR and aVL. Precordial R wave progression can be used to distinguish limb lead reversal from dextrocardia: Limb lead reversal is associated with normal R wave progression; dextrocardia is associated with reverse R wave progression (diminshing R wave amplitude from V_1-V_6). (Answer: e)
		*Hypothermia* results in sinus bradycardia with widening of the QRS, prolongation of PR and QT intervals, and Osborne ("J") waves, which are late upright terminal deflections of the QRS complex ("camel hump" sign). Atrial fibrillation is common, and AV junctional rhythm, ventricular tachycardia, or ventricular fibrillation may also occur. (Note: Shivering sometimes causes baseline artifact mimicking atrial fibrillation, and notching simulating an Osborne wave may be seen in early repolarization.) (Answer: h)

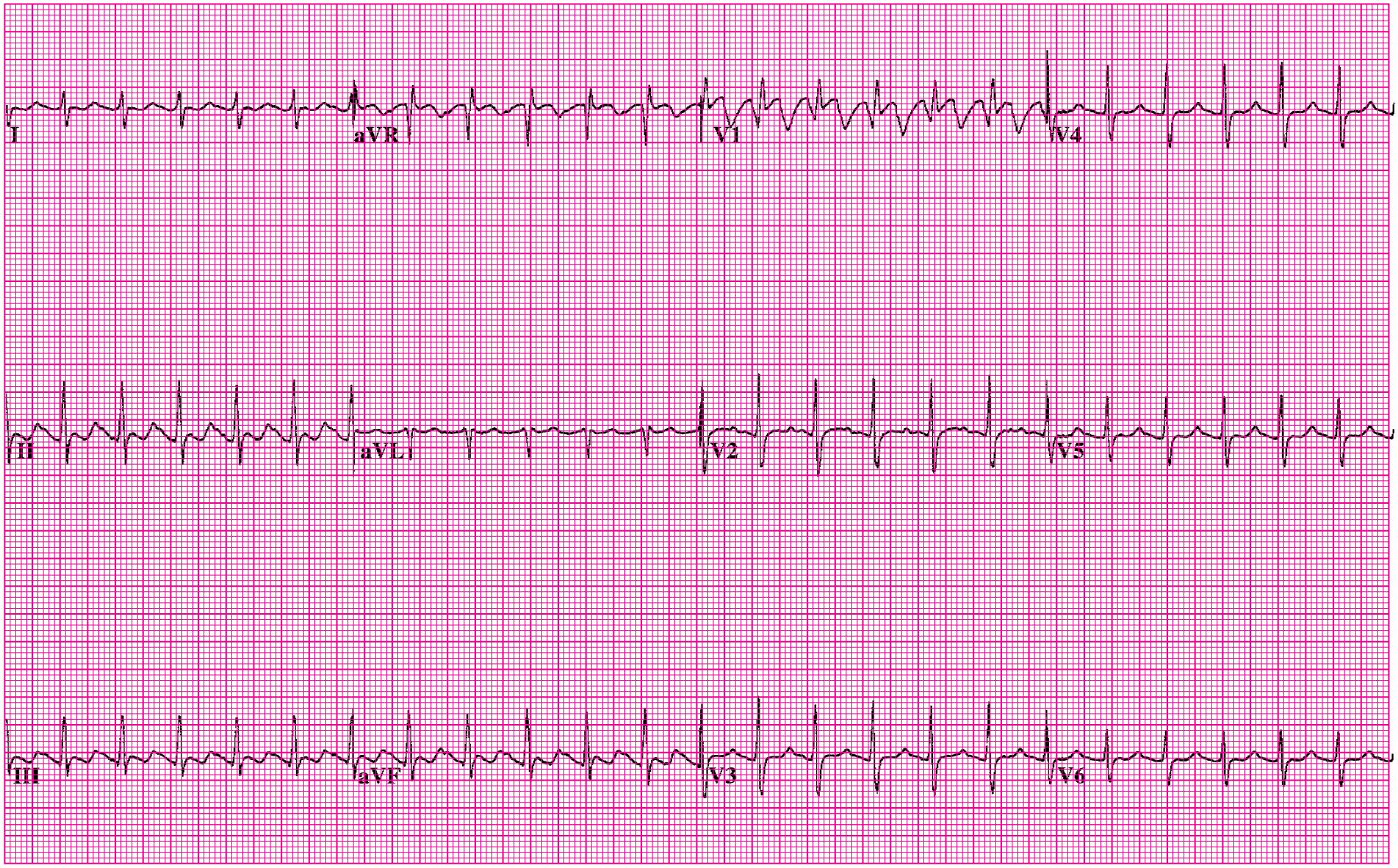
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the ECG diagnosis that best corresponds to the ECG features listed below (see answer sheet for options)

ECG Features	Answer
• Ventricular rate of 30-40 per minute • QRS morphology is similar to VPCs • QRS complex occurs as a secondary phenomenon in response to decreased sinus impulse formation or conduction, or high-degree AV block	Ventricular escape beats or rhythm
• Ventricular rate of 40-60 per minute • QRS morphology similar to sinus/supraventricular impulse • QRS complex occurs in response to decreased sinus impulse formation or conduction, or high-degree AV block; the atrial mechanism may be sinus rhythm, paroxysmal atrial tachycardia, atrial flutter, or atrial fibrillation	AV junctional escape complex
• Ventricular ectopic beats occur at a rate of 30-50 per minute (can range from 20-400 per minute) • VPCs show nonfixed coupling • Fusion complexes may be present • All interectopic intervals are a multiple of the shortest interectopic interval	Ventricular parasystole
• Regular ventricular rhythm at a rate of 60-110 bpm • QRS morphology is similar to VPCs • Ventricular capture complexes, fusion beats, and AV dissociation are common	Accelerated idioventricular rhythm

ECG 5. 46-year-old male four years status-post cardiac transplantation:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

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- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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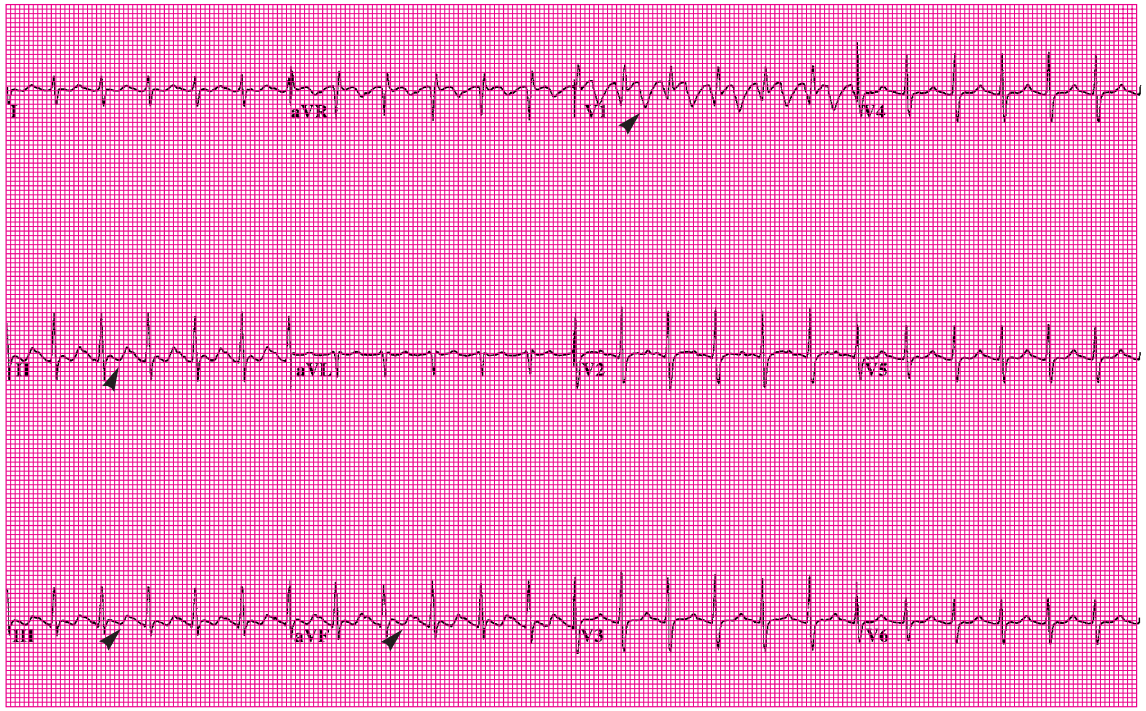
ECG 5 was obtained from a 46-year-old male who is four years status-post cardiac transplantation. The tracing shows a narrow complex tachycardia. Atrial flutter waves (arrowheads) are apparent in the inferior leads and in lead V_1. Every other flutter wave is buried in the QRS complex, so 2:1 AV block should be coded. Flutter waves deform the ST segment in the inferior leads to simulate myocardial ischemia and deform the QRS complex in lead V_1 to simulate an abnormal Q waves. Right axis deviation is also present.

Codes:

18 Atrial flutter

32 AV block, 2:1

37 Right axis deviation (> +100°)



Questions: ECG 5

1. Right axis deviation is associated with all of the following conditions except:

    a. Chronic lung disease
    b. Right ventricular hypertrophy (RVH)
    c. Right bundle branch block (RBBB)
    d. Anterior MI
    e. Lateral MI
    f. Left anterior fascicular block
    g. Left posterior fascicular block
    h. Dextrocardia
    i. Lead reversal

2. The typical response of atrial flutter to carotid sinus massage is:

    a. No effect
    b. Slowing of flutter rate; no change in ventricular response
    c. No change in flutter rate; transient increase in AV block
    d. Conversion to normal sinus rhythm

Answers: ECG 5

1. Right axis deviation is defined by mean QRS axis between 100° and 270°. Among the conditions listed, right axis deviation can be seen in chronic lung disease (e.g., emphysema), RVH, lateral wall MI, left posterior fascicular block, dextrocardia, and limb lead reversal. Other causes include vertical heart, pulmonary embolus, and ostium secundum ASD. Mean QRS axis is usually normal in RBBB and anterior MI, and is leftward in left anterior fascicular block. (Answer: c, d, f)

2. In patients with atrial flutter, carotid sinus massage typically causes a transient increase in AV block and slowing of the ventricular response without a change in flutter rate; less commonly, no effect is seen. When 2:1 AV block is present and atrial flutter is suspected, carotid sinus massage may unmask flutter waves and help confirm the diagnosis. Upon discontinuation of carotid sinus massage, the usual response is return to the original ventricular rate. (Answer: c)

— Quick Review 5 —

Atrial flutter	
• Rapid (regular/irregular) atrial undulations ("F" waves) at a rate of ____ per minute	regular 240-340
• Flutter rate may (increase/decrease) in the presence of Types IA, IC or III antiarrhythmic drugs	decrease
• Flutter waves in leads II, III, AVF are typically (inverted/upright) (with/without) an isoelectric baseline	inverted, without
• Flutter waves in lead V_1 are typically small (positive/negative) deflections (with/without) a distinct isoelectric baseline	positive, with
• QRS complex may be normal or aberrant (true/false)	true
• AV conduction ratio (ratio of flutter waves to QRS complexes) is usually (fixed/variable)	fixed
▸ Conduction ratios of 1:1 and 3:1 are (common/uncommon)	uncommon
▸ In untreated patients, AV block $\geq$ ____ suggests the coexistence of AV conduction disease	4:1
Right axis deviation	
• Mean QRS axis between ____ and ____ degrees	101, 270

Common Dilemmas in ECG Interpretation

Problem

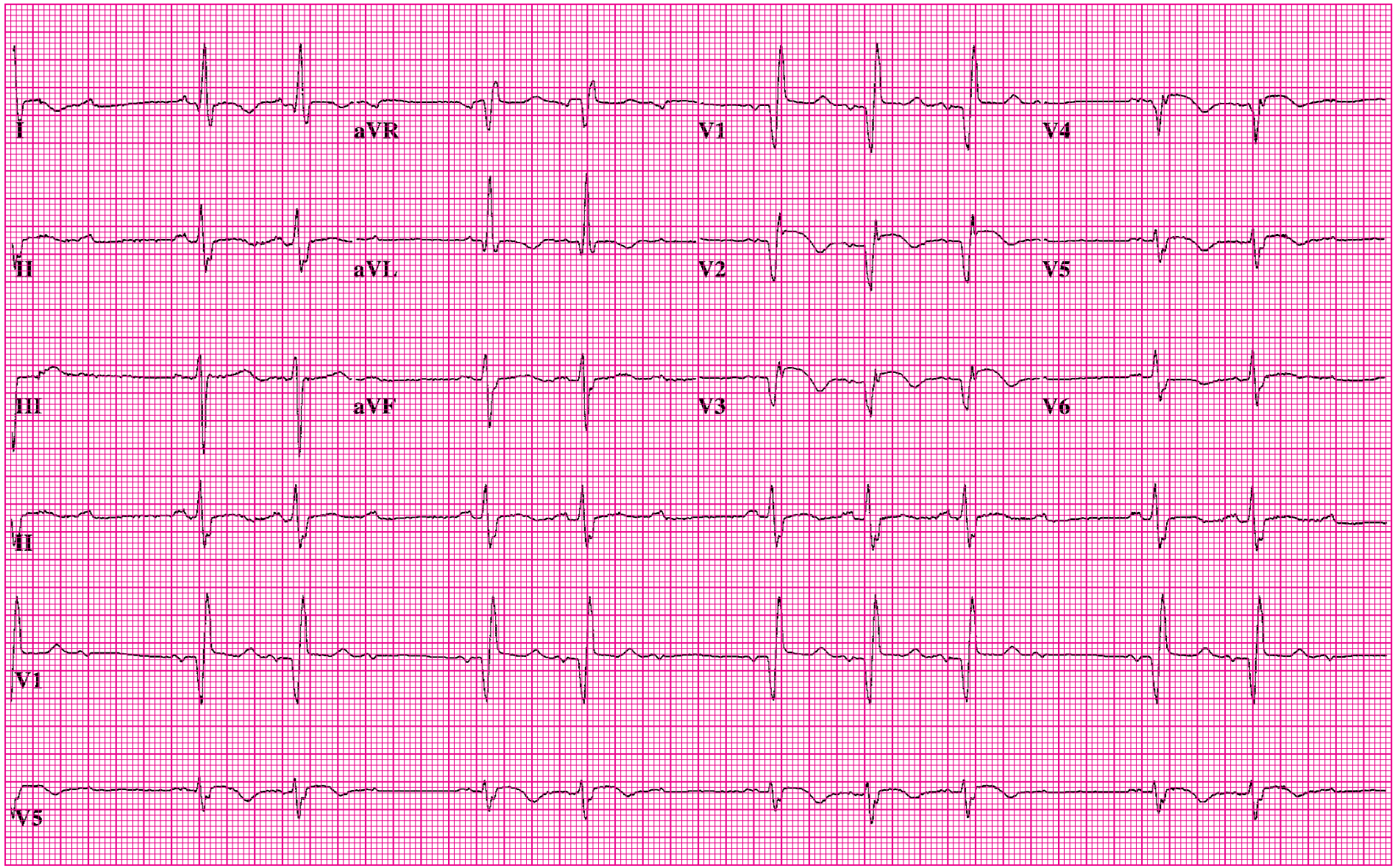
Left bundle branch block is present. Should acute myocardial infarction ever be coded?

Recommendation

No (controversial). Most electrocardiographers are reluctant to diagnose acute myocardial infarction in the setting of LBBB. However, three criteria have independent value for diagnosing acute myocardial injury:

- ST elevation $\geq$ 1 mm concordant to (same direction as) the major deflection of the QRS
- ST depression $\geq$ 1 mm in V_1, V_2, or V_3
- ST elevation $\geq$ 5 mm discordant with (opposite direction to) the major deflection of the QRS

ECG 6. 85-year-old female with recent onset of chest pain:



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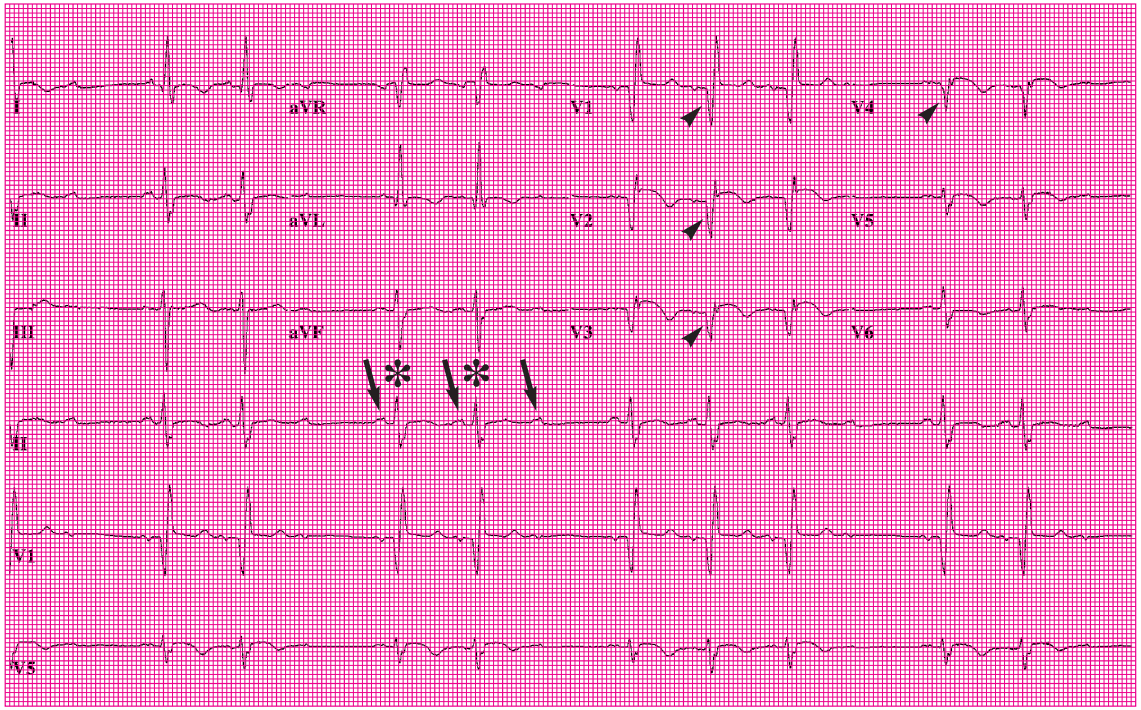
ECG 6 was obtained in an 85-year-old female with recent onset of chest pain. The ECG shows sinus rhythm and Mobitz Type II second-degree AV block with 3:2 AV conduction (3 P waves [arrows] for every 2 QRS complexes [asterisks]). Also noted are right bundle branch block, left atrial enlargement, left ventricular hypertrophy (R wave in aVL $\geq$ 12mm), and acute or recent anteroseptal myocardial infarction (arrowheads) with ST and T wave abnormalities suggesting myocardial injury. The T wave inversions in the lateral leads (I, aVL, V_5, V_6) are consistent with repolarization abnormality secondary to LVH; however, in the setting of evolving myocardial infarction, they are most likely due to myocardial ischemia.

Codes:

06	Left atrial abnormality/enlargement
07	Sinus rhythm
31	AV block, 2° - Mobitz Type II
40	Left ventricular hypertrophy
43	RBBB, complete
53	Anterior or anteroseptal myocardial infarction (age recent or acute)
64	ST and/or T wave abnormalities suggesting myocardial ischemia
65	ST and/or T wave abnormalities suggesting myocardial injury



Questions: ECG 6

1. Features consistent with Mobitz Type II second-degree AV block include all of the following except:

   a. Constant PR interval in the conducted beats
   b. Intermittently nonconducted P waves without evidence of atrial prematurity
   c. RR interval containing the nonconducted P wave is less than two PP intervals
   d. RR interval containing the nonconducted P wave equals two PP intervals

2. Features favoring Mobitz I (Wenckebach) over Mobitz II second-degree AV block in patients with 2:1 AV conduction include:

   a. Classic Mobitz I AV block is present on another part of the ECG
   b. AV conduction improves with exercise
   c. Bifascicular block

Answers: ECG 6

1. This 85-year-old female presenting with an acute anteroseptal myocardial infarction has damaged her AV and His-Purkinje conduction systems, resulting in right bundle branch block and Mobitz II AV block. The diagnosis of Mobitz Type II second-degree AV block requires that the PR interval remains constant in the conducted beats, that there are intermittently nonconducted P waves without evidence of premature atrial complexes, and that the RR interval containing the nonconducted P wave equals two PP intervals. If the RR interval containing the nonconducted P wave is less than two PP intervals, Mobitz Type I second-degree AV block is suggested and evidence for PR interval prolongation should be assessed. (Answer: c)

2. Patients with 2:1 AV block can have either a Mobitz Type I (Wenckebach) or Mobitz Type II mechanism. Maneuvers that increase heart rate and PR conduction (e.g., exercise, atropine) will improve AV conduction and decrease heart block in patients with Mobitz I block at the level of the AV node. In contrast, patients with Mobitz II and block in the His-Purkinje system will often have worsening AV block as heart rate and PR conduction improve. If classic Mobitz I AV block is seen on another part of the ECG, then the episode of 2:1 AV block is most likely based on a Mobitz I mechanism. The presence of bundle branch block or bifascicular block indicates disease in the Purkinje system and suggests that 2:1 AV block is due to a Mobitz II mechanism. (Answer: a, b)

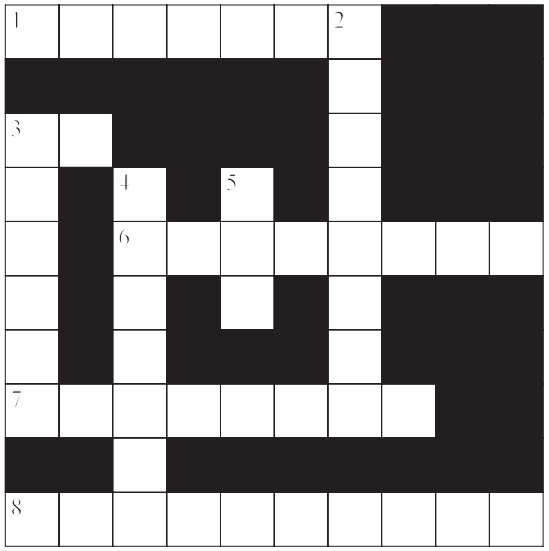
— Quick Review 6 —

AV block, 2° - Mobitz Type II	
• Regular sinus or atrial rhythm with intermittent nonconducted _____ waves (with/without) evidence for atrial prematurity	P, without
• PR interval in the conducted beats is (constant/variable)	constant
• RR interval containing the nonconducted P wave is (less than/equal to/greater than) two PP intervals	equal to
ST and/or T wave abnormalities suggesting myocardial ischemia	
• Abnormally tall, symmetrical, (upright/inverted) T waves	inverted
• Horizontal or ____ ST segments with or without T wave inversion	downsloping
• Associated ECG findings:	
▸ QT interval is usually (normal/prolonged)	prolonged
▸ Reciprocal ____ wave changes may be evident	T
▸ Prominent U waves are often present and may be upright or inverted (true/false)	true

— Quick Review 6 —

Left ventricular hypertrophy	
• **Cornell Criteria** (most accurate): R wave in aVL + S wave in V_3 > ____ mm in males or > ____ mm in females	28, 20
• **Other voltage-based criteria**	
▸ Precordial leads (one or more)	
(1) R wave in V_5 or V_6 + S wave in V_1	
▸ > ____ mm if age > 40 years	35
▸ > ____ mm if age 30-40 years	40
▸ > ____ mm if age 16-30 years	60
(2) Maximum R wave + S wave in precordial leads > ____ mm	45
(3) R wave in V_5 > ____ mm	26
(4) R wave in V_6 > ____ mm	20
▸ Limb leads (one or more)	
(1) R wave in lead I + S wave in lead II ≥ ____ mm	26
(2) R wave in lead I ≥ ____ mm	14
(3) S wave in aVR ≥ ____ mm	15
(4) R wave in aVL ≥ ____ mm	12
(5) R wave in aVF ≥ ____ mm	21
• **Non-voltage related criteria for LVH**	
▸ (Left/right) atrial abnormality	left
▸ (Left/right) axis deviation	left
▸ Onset of intrinsicoid deflection > ____ seconds	0.05
▸ Small or absent R waves in leads ____	V_1-V_3
▸ Absent ____ waves in leads I, V_5, V_6	Q
▸ Abnormal ____ waves in leads II, III, aVF	Q
▸ Prominent ____ waves, especially in leads with large R and T waves	U
▸ R wave amplitude in V_6 (greater than/less than) V_5, provided there are dominant R waves in these leads	greater than

— ECG CROSSWORD PUZZLE —



ACROSS

1. Hyper_____ is associated with QT shortening, a sine wave QRS pattern, and sinoventricular conduction
3. WPW results in a PR interval < 0.12 seconds and a constant _____ interval that is ≤ 0.26 seconds
6. Reversal of right and left arm leads results in P-QRS-T complexes that are _____ in I and aVL
7. Associated findings include low QRS voltage, sinus bradycardia, and pericardial effusion
8. Mobitz Type I _____ exit block demonstrates shortening of the PP interval up to PP pause, a constant PR interval, and a PP pause less than twice the normal PP interval

DOWN

2. Suggested by ST elevation ≥ 1 mm persisting 4 or more weeks after acute MI in leads with abnormal Q waves
3. Type of atrial septal defect associated with left axis deviation
4. _____ toxicity can cause atrial fibrillation with a regular ventricular response due to complete heart block and junctional tachycardia
5. Associated with right axis deviation, a dominant R wave in V_1, secondary ST-T changes, and right atrial abnormality

K	A	L	E	M	I	A			
						N			
P	J					E			
R		D		R		U			
I		I	N	V	E	R	T	E	D
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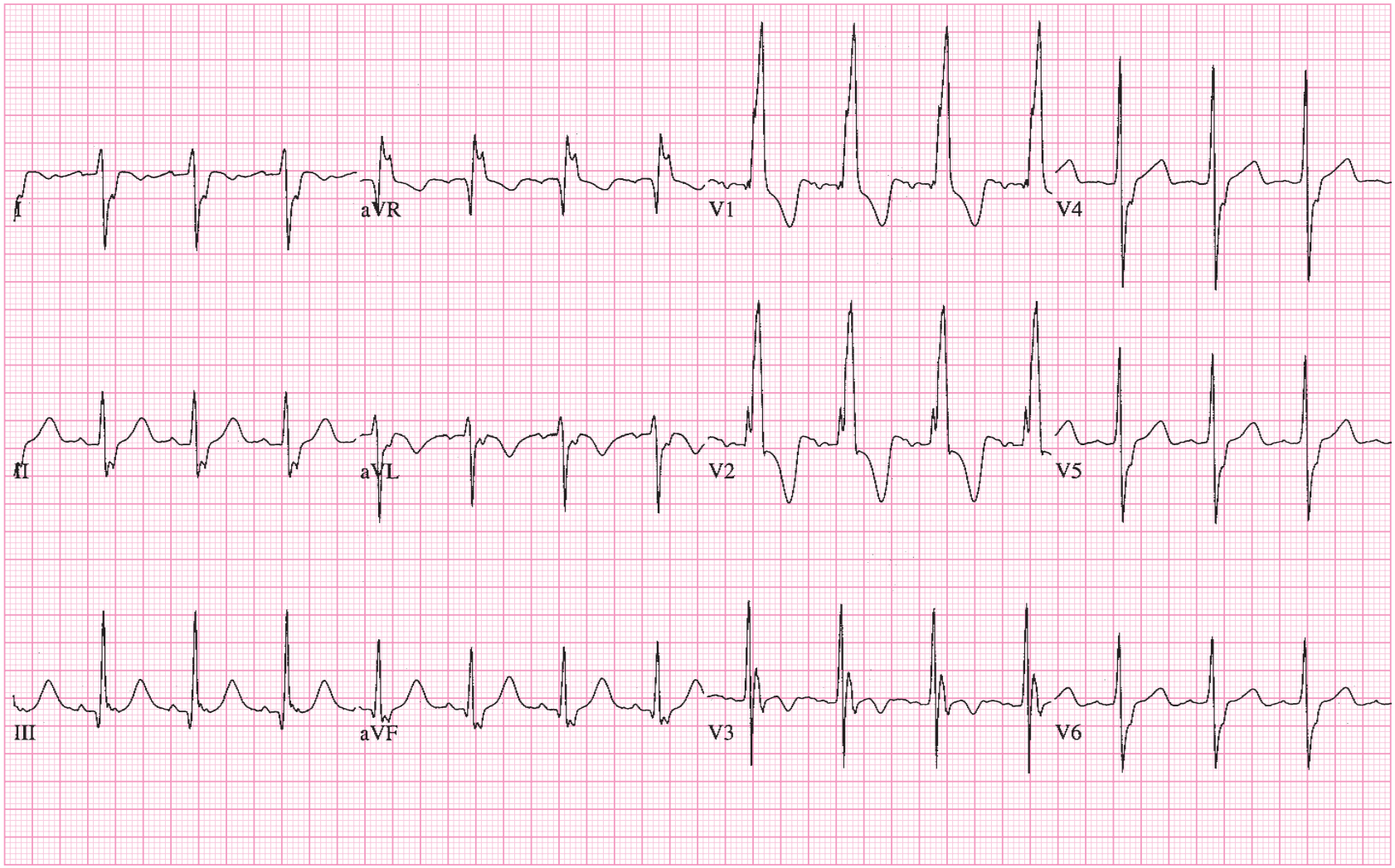
M	Y	X	E	D	E	M	A		
		I							
S	I	N	O	A	T	R	I	A	L

64050_INDD_Overtakes.indd 7

ECG 7. 51-year-old female with shortness of breath:



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GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
- ☐ 11. Sinus pause or arrest
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- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 18. Atrial flutter
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- ☐ 21. AV junctional escape complexes
- ☐ 22. AV junctional rhythm/tachycardia

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- ☐ 23. Ventricular premature complexes
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- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
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- ☐ 29. AV block, 1°
- ☐ 30. AV block, 2°-Mobitz type I (Wenckebach)
- ☐ 31. AV block , 2°-Mobitz type II
- ☐ 32. AV block, 2:1
- ☐ 33. AV block, 3°
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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 51. Anterolateral (age recent or acute)
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- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

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

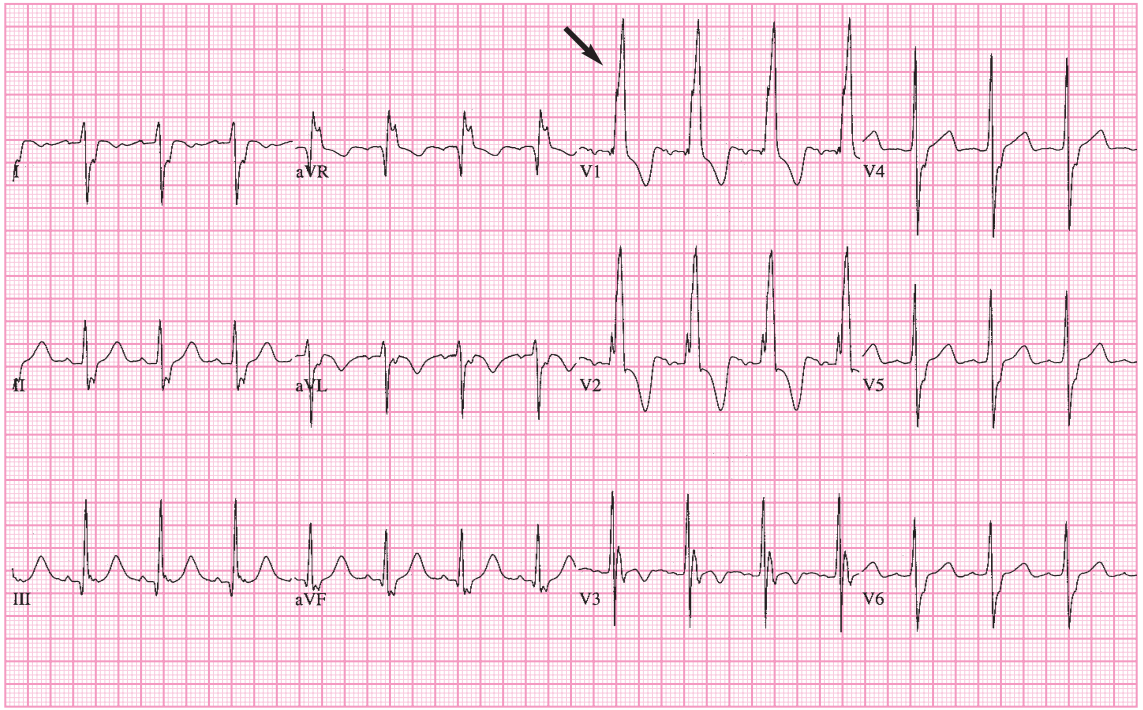
PACED RHYTHMS

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

ECG 7 was obtained from a 51-year-old female with shortness of breath. The ECG shows normal sinus rhythm at a rate of 89 beats/minute, right axis deviation (+130°), and right bundle branch block. The very tall QRS complex in lead V_1 (R wave amplitude = 29 mm)(arrow), particularly in association with right axis deviation, is consistent with right ventricular hypertrophy (RVH). Left posterior fascicular block (LPFB) should not be coded since it requires the absence of other causes of right axis deviation (e.g., RVH).

Codes:

07	Sinus rhythm
37	Right axis deviation (> +100°)
41	Right ventricular hypertrophy
43	RBBB, complete
67	ST and/or T wave abnormalities secondary to hypertrophy



Questions: ECG 7

1. Repolarization abnormalities associated with right ventricular hypertrophy (RVH) are typically most prominent in leads:

   a. V_4-V_6
   b. V_1-V_3
   c. I, aVL
   d. aVR, aVL

2. Conditions that can mimic RVH on ECG include:

   a. Wolff-Parkinson-White pattern
   b. Anterior myocardial infarction
   c. Posterior myocardial infarction
   d. Right bundle branch block (RBBB)

Answers: ECG 7

1. The "strain pattern" of right ventricular hypertrophy manifests shallow T wave inversion with or without downslopping ST segment depression in leads V_1-V_3. When RVH complicates COPD, ST segment depression in the inferior leads is sometimes seen when RVH is caused by chronic lung disease (i.e., pulmonary hypertension). (Answer: b)

2. Many conditions are associated with a tall R wave in V_1 and right axis deviation, and can thus mimic right ventricular hypertrophy. These conditions include Wolff-Parkinson-White syndrome, posterior MI, and right bundle branch block. Anterior MI results in absent or diminished anterior forces. (Answer: a, c, d)

— Quick Review 7 —

Right ventricular hypertrophy	
• Mean QRS axis ≥ ____ degrees	100
• Dominant ____ wave in V_1:	R
▸ R/S ratio in V_1 or V_{3R} (<, =, >) 1, or R/S ratio in V_5 or V_6 (≤, >) 1	> ≤
▸ R wave in V_1 ≥ ____ mm	7
▸ R wave in V_1 + S wave in V_5 or V_6 > ____ mm	10.5 10
▸ rSR' in V_1 with R' > ____ mm	
• Secondary downsloping ST depression & T-wave inversion in the (right/left) precordial leads	right
• (Right/left) atrial abnormality	right
RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ____ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 > ____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ____	I, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
• RBBB (does/does not) interfere with the ECG diagnosis of ventricular hypertrophy or Q wave MI	does not

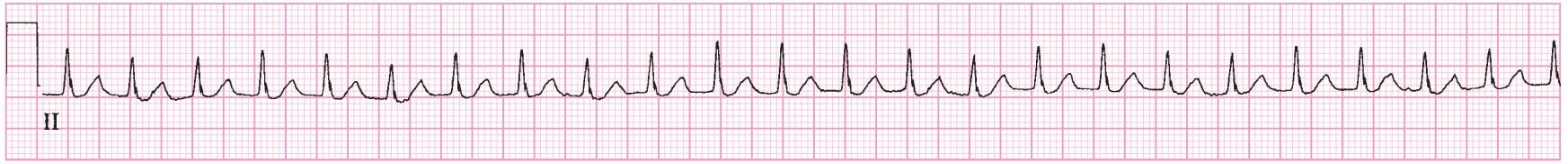
— POP QUIZ —

Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

B.



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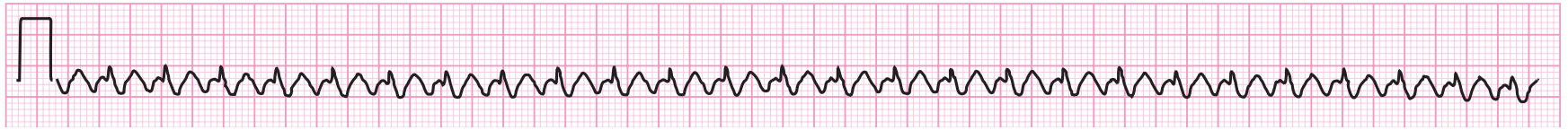

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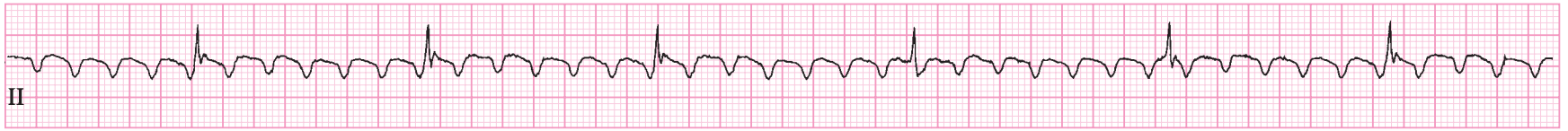


Answer: Tracings A, C and D all show atrial flutter with typical flutter waves. The imposter is tracing B, which shows supraventricular tachycardia, probably AV nodal reentry tachycardia. This is evident from the lack of P waves or any atrial activity.

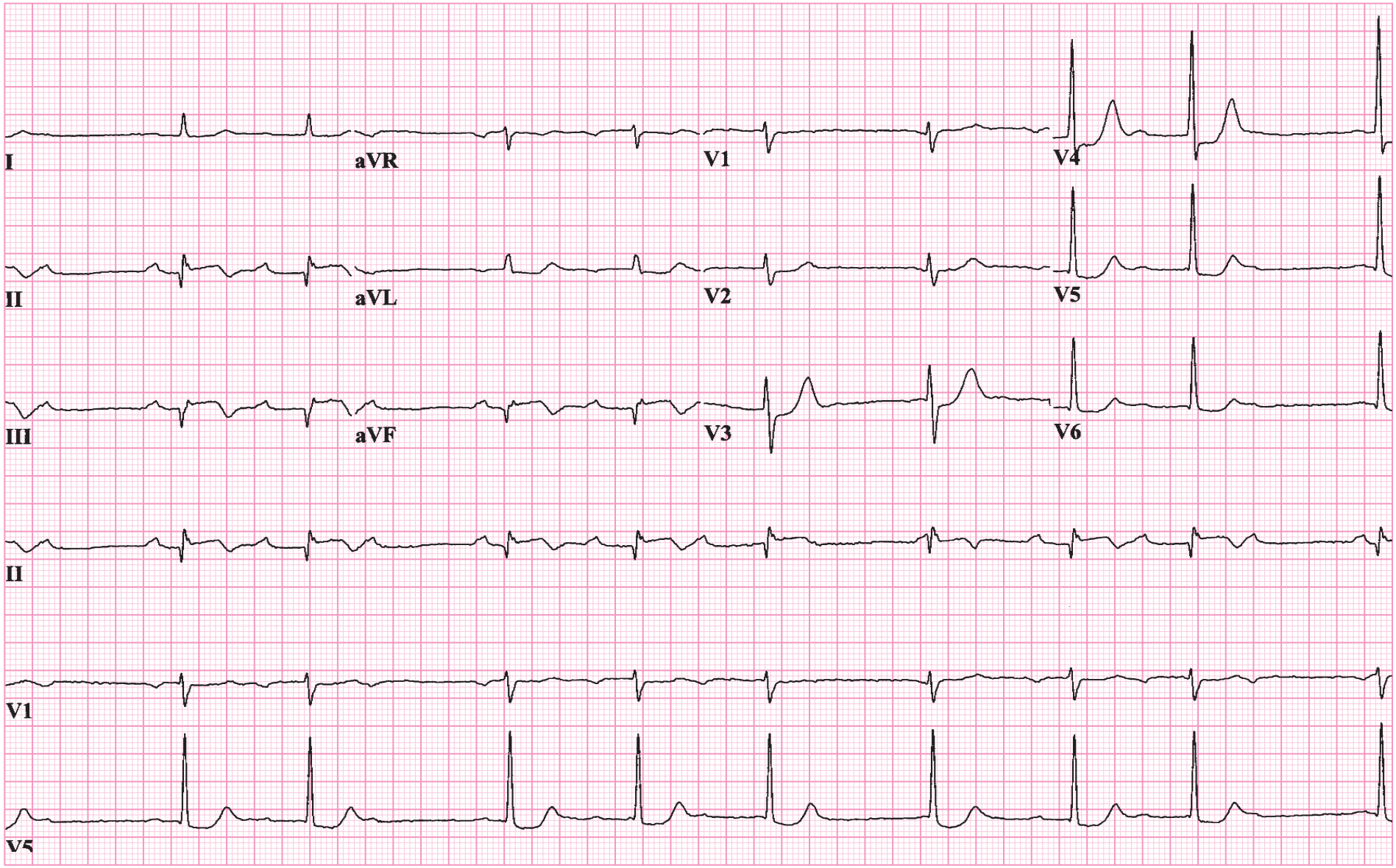
Differential Diagnosis

Peaked T Waves

(T wave amplitude > 6 mm in limb leads or > 10 mm in precordial leads)

- Acute MI (hyperacute phase; transient)
- Normal variant (most common in the mid-precordial leads)
- Hyperkalemia
- Intracranial bleeding
- Left ventricular hypertrophy
- LBBB

ECG 8. 72-year-old male with chest pain and shortness of breath:



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- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
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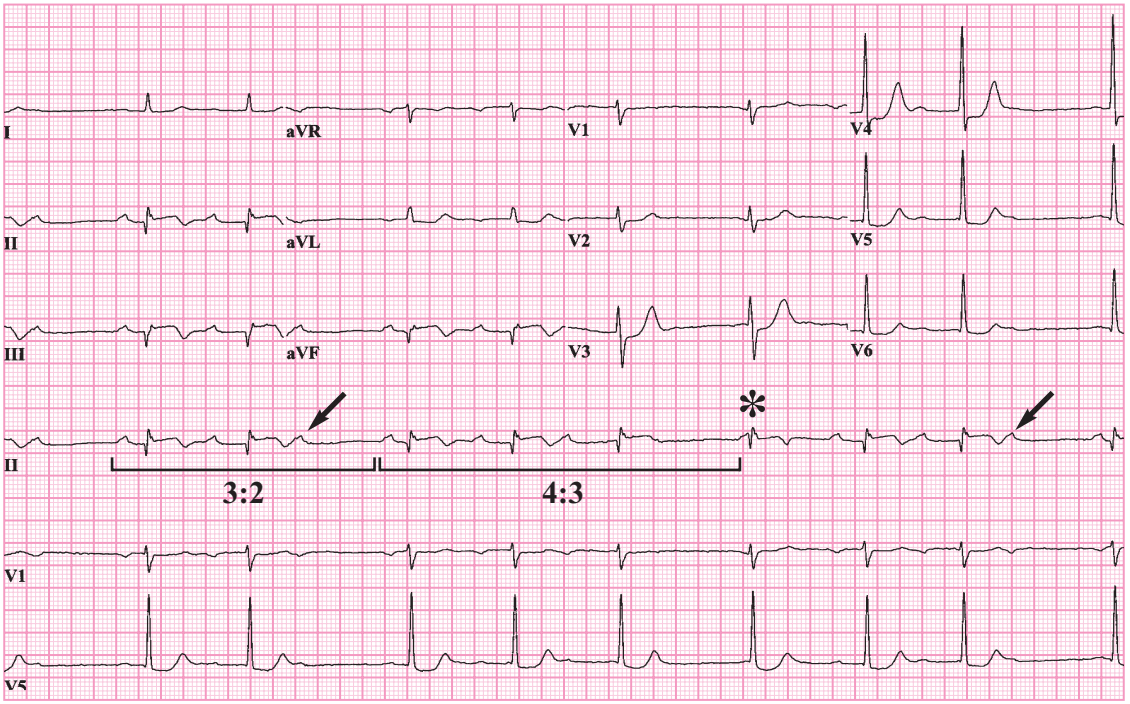
ECG 8 was obtained from a 72-year-old male with chest pain and shortness of breath. The ECG shows abnormal Q waves in II, III, and aVF associated with ST elevation of acute injury, consistent with acute inferior myocardial infarction. In this case, ischemic of the AV node has resulted in an AV Wenckebach pattern; group beating occurs in a 3:2 or 4:3 pattern with progressive PR prolongation prior to a P wave that fails to conduct to the ventricle (arrow). The 6th QRS complex and P wave occur simultaneously (asterisk). This implies that the sinus node had activated the atrium, but before it could conduct through the AV node the ventricle was activated by a junctional escape beat.

Codes:

07	Sinus rhythm
21	AV junctional escape complexes
30	AV block, 2°-Mobitz type I (Wenckebach)
57	Inferior (age recent or acute)



Questions: ECG 8

1. Which of the following types of atrioventricular (AV) block is most likely to occur at the level of the AV node:

   a. Second degree, Mobitz Type I AV block
   b. Second degree, Mobitz Type II AV block
   c. Second degree, high-grade AV block
   d. Complete heart block

d. Which of the following ECG features are consistent with second-degree AV block, Mobitz I (Wenckebach):

   a. Group beating
   b. Progressive shortening of the RR interval until a P wave is blocked
   c. Constant PR intervals immediately before and after nonconducted P waves
   d. Wide QRS complex

Answers: ECG 8

1. Mobitz Type I second-degree atrioventricular block is associated with a Wenckebach pattern on ECG and is almost always due to progressive conduction delay within the AV node. In contrast, Mobitz Type II second-degree AV block is usually due to sudden block in the His-Purkinje system. High-grade AV block and complete heart block can occur within the AV node or the His-Purkinje system, although complete heart block is usually due to block in the His-Purkinje system. (Answer: a)

2. Mobitz Type I second-degree AV block is characterized by progressive prolongation of the PR interval and progressive shortening of the RR interval until a P wave is blocked. Mobitz Type I results in "group" or "pattern beating" due to the presence of nonconducted P waves. Type I block usually occurs at the level of the AV node, resulting in a narrow QRS complex. The presence of a constant PR interval immediately before and after a nonconducted P wave is consistent with Type II block, not Type I block. (Answer: a, b)

— Quick Review 8 —

AV junctional escape complexes	
• QRS complex occurs as a ____ phenomenon in response to decreased sinus impulse formation or conduction, or high-degree AV block	secondary
• Rate is typically ____ per minute	40-60
• Atrial mechanism may be sinus rhythm, paroxysmal atrial tachycardia, atrial flutter, or atrial fibrillation (true/false)	true
• QRS morphology is (similar to/different from) the sinus or supraventricular impulse	similar to
AV block, 2° - Mobitz Type I (Wenckebach)	
• Progressive prolongation of the ____ interval and shortening of the ____ interval until a P wave is blocked	PR RR
• RR interval containing the nonconducted P wave is (less than/equal to/greater than) the sum of two PP intervals	less than
• Results in ____ beating due to the presence of nonconducted P waves	group

— POP QUIZ —

Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

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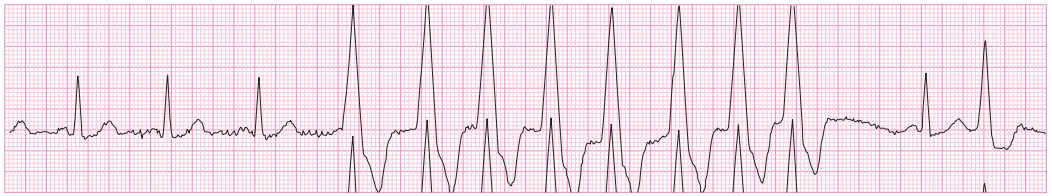
B.

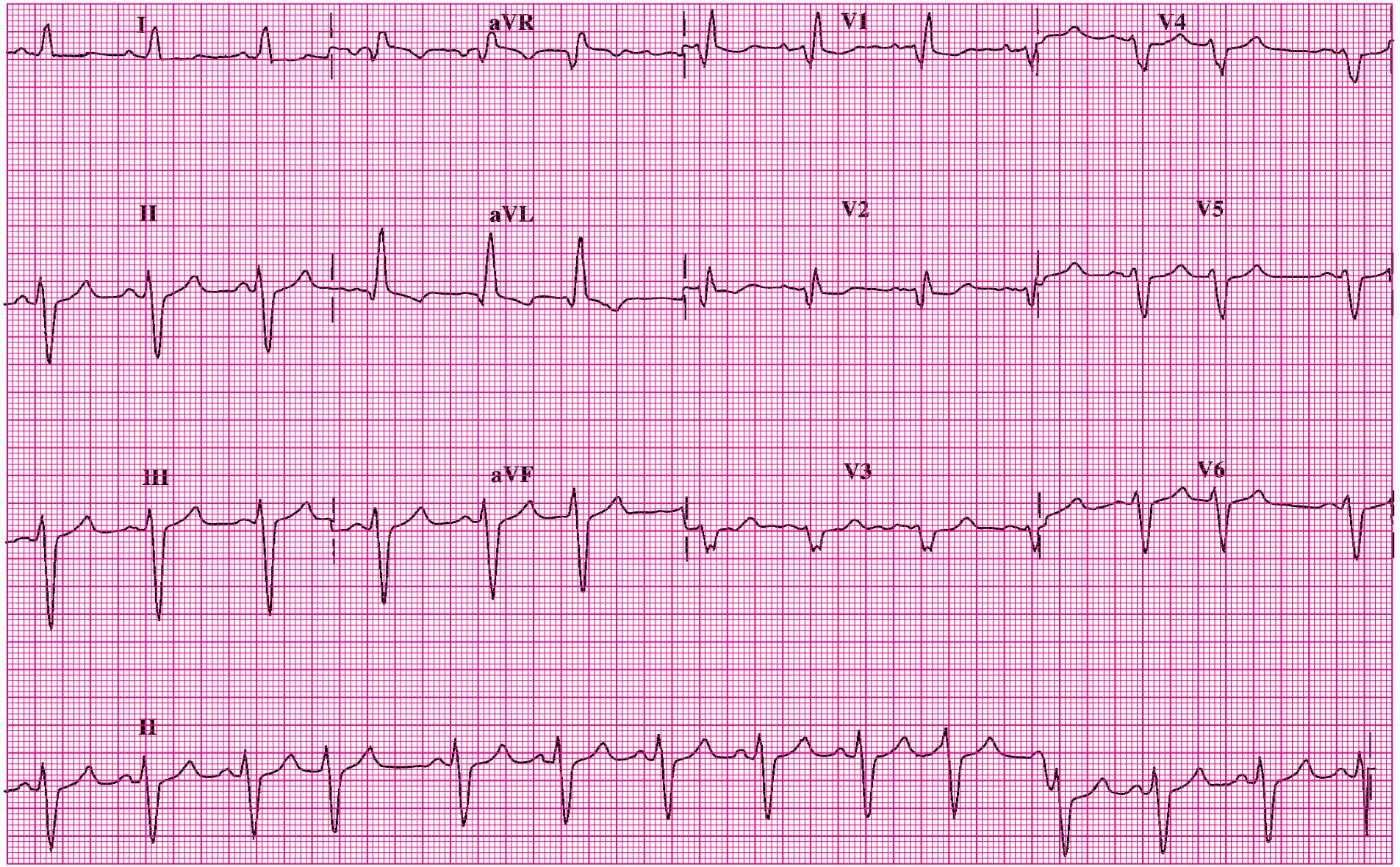
C.



D.

Answer: Tracings B, C and D represent ventricular tachycardia, with a rapid wide QRS complex rhythm. The imposter is tracing A, which shows sinus rhythm with artifact resembling the wide QRS complexes of ventricular tachycardia. The several rapid upstroke deflections throughout the apparent "wide complex rhythm" are sinus beats – they resemble the preceding and subsequent narrow QRS complexes of sinus rhythm and march out regularly across the entire tracing (with the use of calipers).

ECG 9. 69-year-old male with orthopnea and pedal edema:



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- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

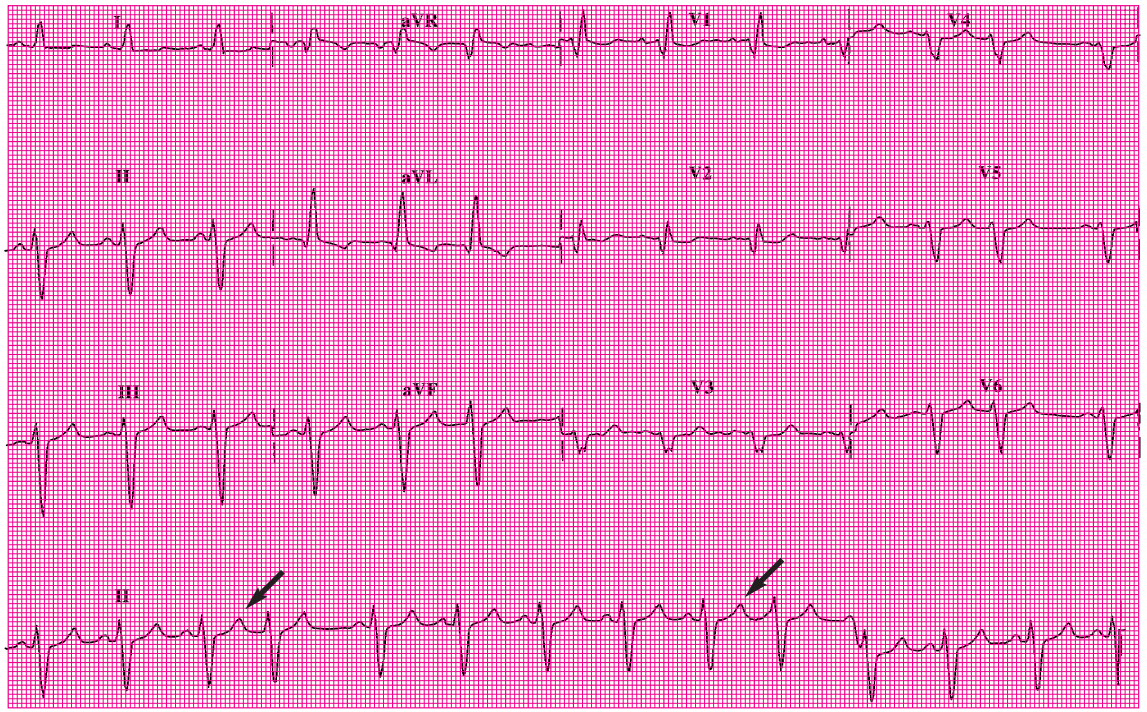
PACED RHYTHMS

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

ECG 9 was obtained from a 69-year-old male with orthopnea and pedal edema. The ECG shows sinus rhythm with normally-conducted APCs (arrows mark the premature P waves, which are superimposed on the preceding T waves). RBBB and left anterior fascicular block are also present. The T wave following the first APC in the rhythm strip is slightly more peaked than the previous T waves, consistent with a post-extrasystolic T wave abnormality.

Codes:

07	Sinus rhythm
13	Atrial premature complexes
43	RBBB, complete
45	Left anterior fascicular block



Questions: ECG 9

1. The tall R wave in aVL is highly specific for LVH:

   a. True
   b. False

2. Findings in this tracing that can be attributed to left anterior fascicular block include:

   a. rS in leads II, III and aVF
   b. qR in leads I and aVL
   c. Large S wave in leads V_4 - V_6
   d. Poor R wave progression

3. The most common cause of right bundle branch block plus left anterior fascicular block (bifascicular block) is:

   a. Cardiomyopathy
   b. Hypertensive heart disease
   c. Coronary artery disease
   d. Lenegre's disease

4. The most common type of myocardial infarction to cause RBBB plus LAFB is:

   a. Inferior MI
   b. Anterior MI
   c. Lateral MI
   d. Posterior MI

5. What is the incidence of complete heart block when bifascicular block occurs during myocardial infarction:

   a. < 5%
   b. 5 - 10%
   c. 10 - 20%
   d. > 20%

6. Did the premature atrial contraction (4th beat in rhythm strip) reset the sinus node:

   a. Yes
   b. No

Answers: ECG 9

1. An R wave in lead aVL $\geq$ 12 mm is highly specific for anatomical LVH. However, specificity is reduced when left anterior fascicular block (LAFB) is present, since LAFB by itself can produce tall R waves in aVL. (Answer: b)

2. ECG manifestations of left anterior fascicular block include left axis deviation, qR in leads I and aVL, and rS in leads II, III, and aVF. Large S waves in V_4 - V_6 and poor R wave progression may also be seen. (Answer: all)

3. Coronary artery disease is the most common cause of bifascicular block (RBBB plus LAFB), and is responsible for up to 50% of cases. Other causes include hypertensive heart

disease, calcific aortic valve disease (with extension of the calcification into the anterior interventricular septum), cardiomyopathy, Lev's disease, Lenegre's disease, surgical trauma, post-cardiac transplant, among others. Complete heart block develops in 5-15% of patients with chronic bifascicular block and in 25-40% of patients with acute bifascicular block secondary to acute MI. (Answer: c)

4. Anterior wall myocardial infarction from occlusion of the proximal left anterior descending coronary artery is the most common cause of acute bifascicular block (RBBB plus LAFB). The right bundle branch and anterior division of the left bundle branch course together in the anterior portion of the interventricular septum, and receive their blood supply from septal perforators of the LAD. (Answer: b)

5. Since progression to complete heart block develops in more than 20% of patients who develop acute bifascicular block during MI, temporary transvenous pacing should be considered. When extensive anterior infarction is evident, mortality remains high despite the presence of a pacemaker; death is often due to pump failure rather than progression to complete heart block. (Answer: d)

6. The PP interval remains constant and the sinus node is undisturbed by the premature atrial contraction in this tracing. (Answer: b)

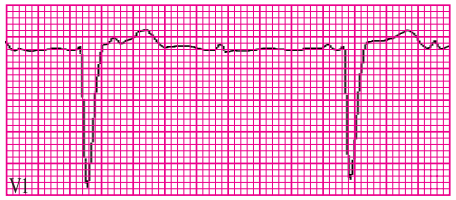
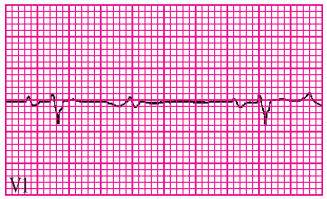
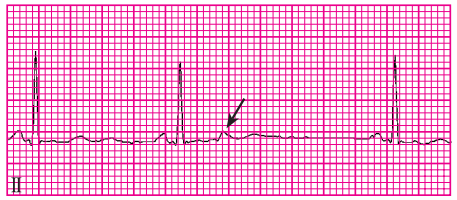
— Quick Review 9 —

Atrial premature complexes, normally conducted	
• P wave is (normal/abnormal) in configuration	abnormal
• QRS complex is (similar/different) in morphology to the QRS complex present during sinus rhythm	similar
• PR interval may be normal, increased, or decreased (true/false)	true
• The post-extrasystolic pause is usually (compensatory/noncompensatory)	noncompensatory
RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ____ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 > ____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ____	I, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
• RBBB (does/does not) interfere with the ECG diagnosis of ventricular hypertrophy or Q wave MI	does not
Left anterior fascicular block	
• ____ axis deviation with a mean QRS axis between ____ and ____ degrees	left -45, -90
• (qR/rS) complex in leads I and aVL	qR
• (qR/rS) complex in lead III	rS
• Normal or slightly prolonged QRS duration (true/false)	true
• No other cause for left axis deviation should be present (true/false)	true
• Poor R wave progression is (common/uncommon)	common

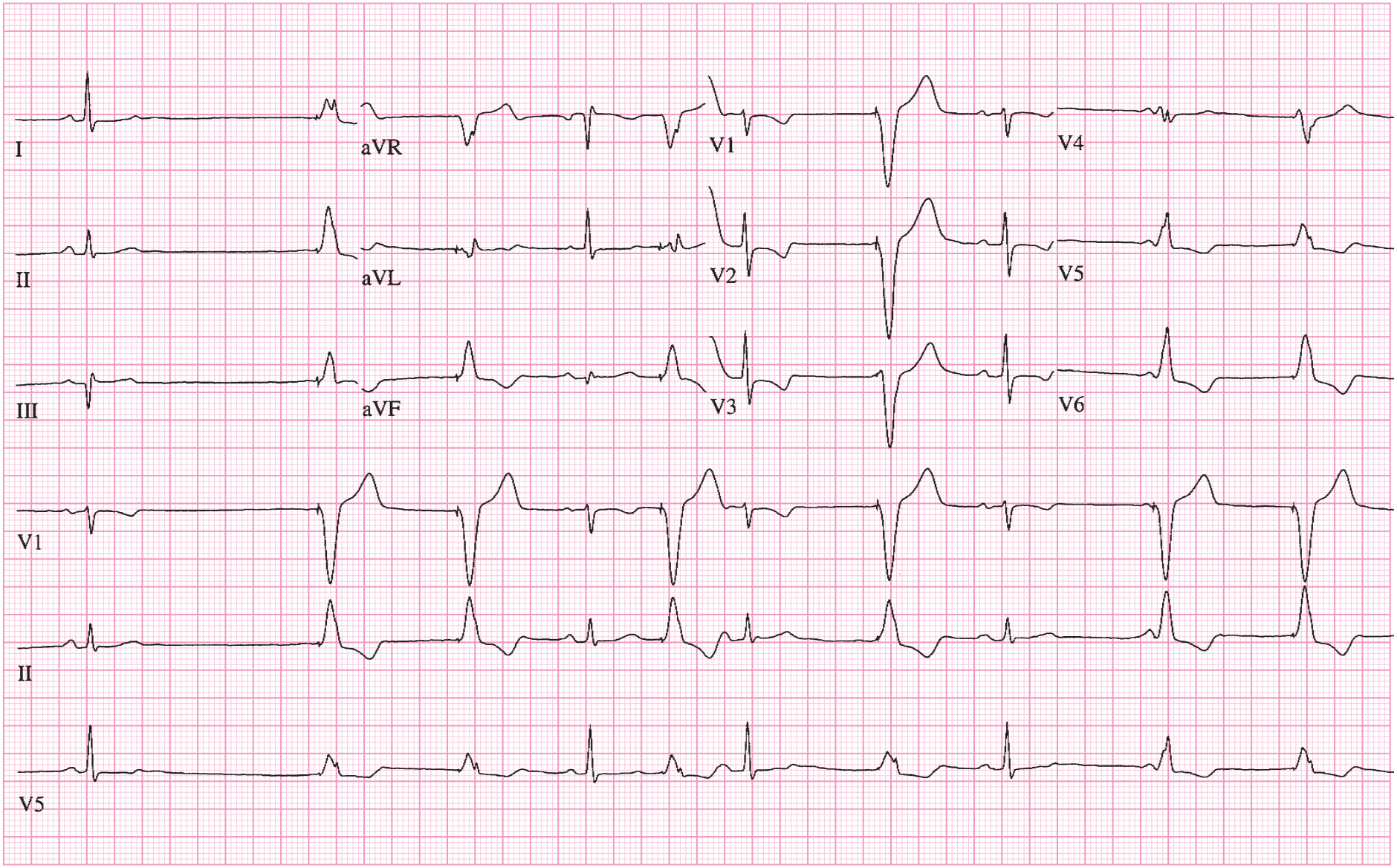
— POP QUIZ —

Pattern Recognition: AV Conduction Abnormalities

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Reflects prolonged conduction from the sinus node to atrial tissue b. 1° AV block c. Can be seen in normal individuals d. 2° AV block, Type I e. Grouped beating due to nonconducted P waves f. Block usually occurs at level of AV node g. More common in inferior MI than anterior MI h. 2° AV block, Type II i. Block may improve with carotid sinus massage and worsen with atropine j. Can be either Mobitz Type I or II k. Atrial and ventricular rhythms are independent of each other l. 3° AV block m. Blocked atrial premature complex n. 2:1 AV block	*Complete (3°) AV block* results in atrial impulses that consistently fail to reach the ventricles. Independent atrial and ventricular rhythms (AV dissociation) result, manifest as variable PR intervals and constant PP and RR intervals (when atrial rate exceeds ventricular rate). Ventricular rhythm is maintained by a junctional or idioventricular escape rhythm or a ventricular pacemaker. (Answer: k, l)
		*2:1 AV block* results in a regular sinus or atrial rhythm in which every other P wave is nonconducted (i.e., two P waves for each QRS complex). AV block can be Mobitz Type I or Type II second-degree AV block, and may require an EP study to distinguish between mechanisms. (Answer: j, n)
		*Nonconducted atrial premature complex (APC)* is identified by a premature ectopic atrial beat (nonsinus P wave) not followed by a QRS-T complex. The P wave is often hidden in the preceding T wave (arrow), resulting in an RR pause that is sometimes mistaken for a sinus pause. (Answer: c, f, m)

ECG 10. 80-year-old male with light-headedness:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 51. Anterolateral (age recent or acute)
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- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

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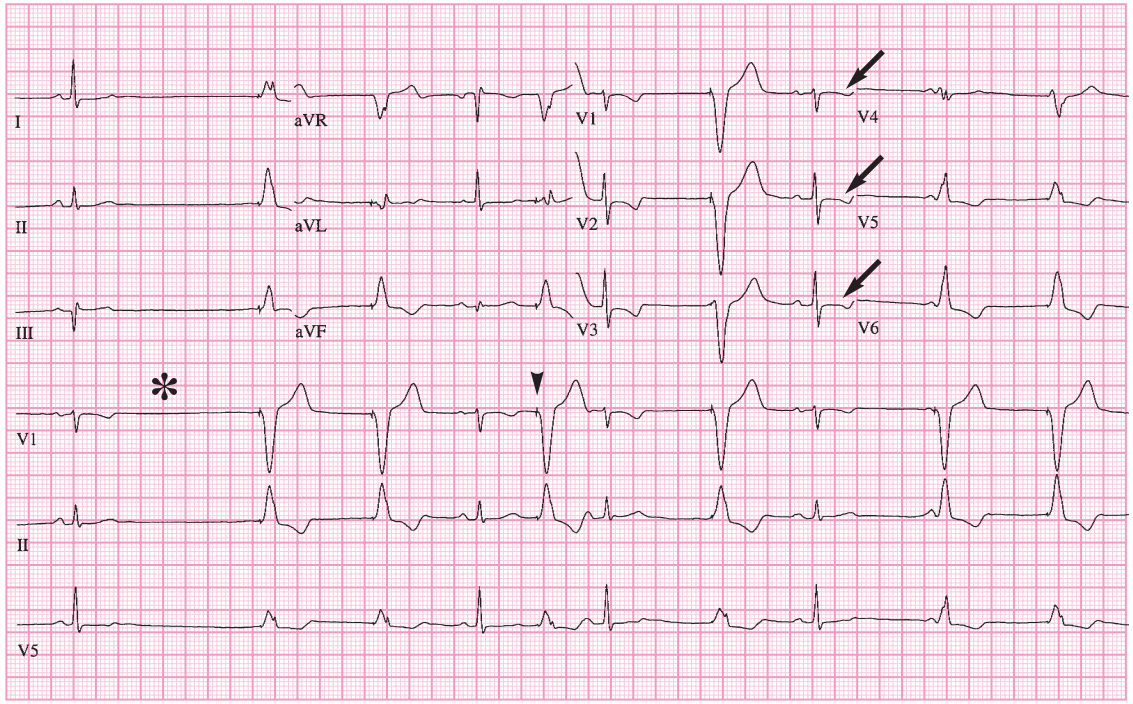
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 10 was obtained in an 80-year-old male with light-headedness. The ECG shows an inconsistent sinus bradycardia at approximately 50 beats/minute. After the first sinus beat, there is a sinus pause of 1.9 seconds that is terminated by a ventricular paced beat. The ventricular pacemaker is pacing at 60 beats/minute but is displaying abnormal sensing function: Oversensing (paced beat occurs late relative to the intrinsic pacemaker rate) is present after the first beat (asterisk), and failure to sense is seen after the fourth beat (paced beat occurs early relative to the intrinsic pacemaker rate) (arrowhead). An old inferior wall myocardial infarction (abnormal Q waves in leads III and aVF) is present, as are repolarization abnormalities suggesting anterior myocardial ischemia (arrow). This constellation of findings — sinus bradycardia and a sinus pause in an elderly patient with a history of previous pacemaker implantation — suggests a diagnosis of sick sinus syndrome.

Codes:

09	Sinus bradycardia (< 60)
11	Sinus pause
58	Inferior Q wave MI (age indeterminate or old)
64	ST and/or T abnormalities suggesting myocardial ischemia
89	Sick sinus syndrome
94	Pacemaker malfunction, not constantly sensing (atrium or ventricle)



Questions: ECG 10

1. Abnormal sensing function of a pacemaker can manifest as all of the following except:

    a. A pacing spike that is not following by an appropriate depolarization
    b. A pause resulting from oversensing of artifact
    c. An early paced beat due to undersensing of an intrinsic depolarization
    d. Failure of a DDD pacemaker to trigger a ventricular depolarization in response to an intrinsic depolarization that failed to reach the ventricle due to AV block

2. ECG features consistent with the diagnosis of sick sinus syndrome include:

    a. Sinus pause or arrest
    b. Tachycardia alternating with bradycardia
    c. SA exit block
    d. Low voltage QRS complexes
    e. Atrial fibrillation with a slow ventricular response
    f. Left atrial abnormality or enlargement

3. A recurring sinus pause that is a multiple of the regular sinus PP interval is consistent with:

    a. Sinus arrhythmia
    b. Blocked atrial premature contraction
    c. Atrial parasystole
    d. SA exit block

4. A sinus pause is defined by a PP interval ≥ ___:

    a. 1.5 seconds
    b. 1.6 seconds
    c. 1.4 seconds
    d. 1.3 seconds

Answers: ECG 10

1. Abnormal pacemaker sensing can cause early paced beats or inappropriately long pauses depending on the type of sensing malfunction. *Oversensing* results in inappropriate inhibition of the pacemaker, usually manifesting as a pause. Oversensing may occur in response to artifact, large T waves, or myopotentials from arm movements (more common with unipolar pacemakers). *Undersensing* occurs when the pacemaker ignores or fails to recognize (e.g., low-amplified VPC) intrinsic depolarizations, and thus, paces prematurely. In the triggered mode, abnormal sensing manifests as failure of the pacemaker to be triggered by an appropriate intrinsic depolarization (e.g., failure to pace the ventricle in response to a nonconducted intrinsic P wave). (Answer: a)

2. Sick sinus syndrome (SSS) is due to sinus node dysfunction, and usually manifests as marked sinus bradycardia with or without episodes of sinus arrest, sinus pauses, or SA exit block. SSS is also commonly referred to as the "tachy-brady" syndrome, due to the frequent occurrence of supraventricular tachycardia alternating with bradycardia. Patients with tachy-brady syndrome may have severe sinus bradycardia or long sinus pauses (i.e., prolonged sinus node recovery time) following an episode of atrial tachyarrhythmia (i.e., SVT, atrial fibrillation). Atrial fibrillation with a slow ventricular response is another

clue to the presence of underlying sinus node dysfunction. (Answers: a, b, c, e)

3. Second-degree sinoatrial (SA) exit block occurs when sinus impulses intermittently fail to capture the atria, resulting in the intermittent absence of a P wave. In type II block, the PP interval is constant and is followed by a pause that is a multiple of the normal PP interval. SA exit block is usually a manifestation of sick sinus syndrome but can also be due to other factors such as use of digitalis or antiarrhythmic drugs. Other causes of SA exit block include hyperkalemia, myocardial infarction, and vagal stimulation. (Answer: d)

4. Sinus pause or arrest is defined as a PP interval ≥ 1.6 seconds. The sinus pause should not be a multiple of the basic PP interval, in which case, SA exit block is suggested. It is also important to distinguish a sinus pause from a nonconducted atrial premature complex, in which case the P wave is typically buried in the repolarization phase of the preceding beat, usually causing a discernable deformity in the ST-segment or T wave of the last complex before sinus pause. Sinus pause or arrest is due to transient failure of impulse formation of the SA node. In contrast, SA exit block results in sinus impulse formation, but conduction to the atrium is either delayed (1° SA exit block) or intermittently fails to capture the atrium (2° SA exit block). 3° SA exit block occurs when there is complete failure of sinoatrial conduction to capture the atrium and cannot be distinguished from complete sinus arrest on the surface ECG. (Answer: b)

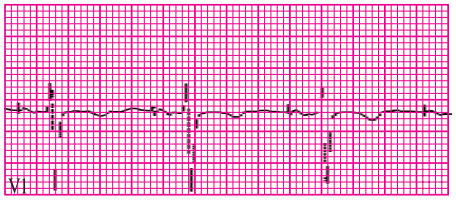
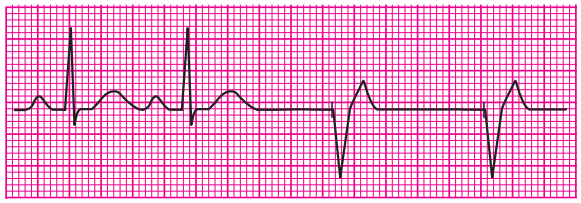
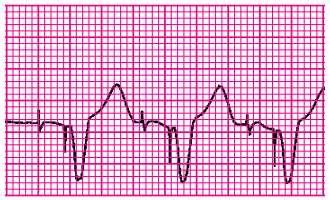
— Quick Review 10 —

Sinus pause or arrest	
• PP interval > _____ seconds	1.6-2.0
• Resumption of sinus rhythm at a PP interval that (is/is not) a multiple of the basic sinus PP interval	is not
• If sinus rhythm resumes at a multiple of the basic PP, consider _____	sinoatrial exit block
Sick sinus syndrome	
• Marked sinus ____	bradycardia
• ____ arrest or ____ exit block	Sinus, sinoatrial
• Bradycardia alternating with ____	tachycardia
• Atrial fibrillation with ____ ventricular response preceded or followed by sinus bradycardia, sinus arrest, or sinoatrial exit block	slow
• Prolonged sinus node ____ time after atrial premature complex or atrial tachyarrhythmias	recovery
• AV junctional ____ rhythm	escape
• Additional conduction system disease is often present, including AV block, IVCD, and/or bundle branch block (true/false)	true
Pacemaker malfunction, not constantly sensing (atrium or ventricle)	
• Pacemakers in the inhibited mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	inhibited
• Pacemakers in the triggered mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	triggered
• Premature depolarizations may not be sensed if they fall within the programmed ____ period of the pacemaker, *or* have insufficient ____ at the sensing electrode site	refractory amplitude

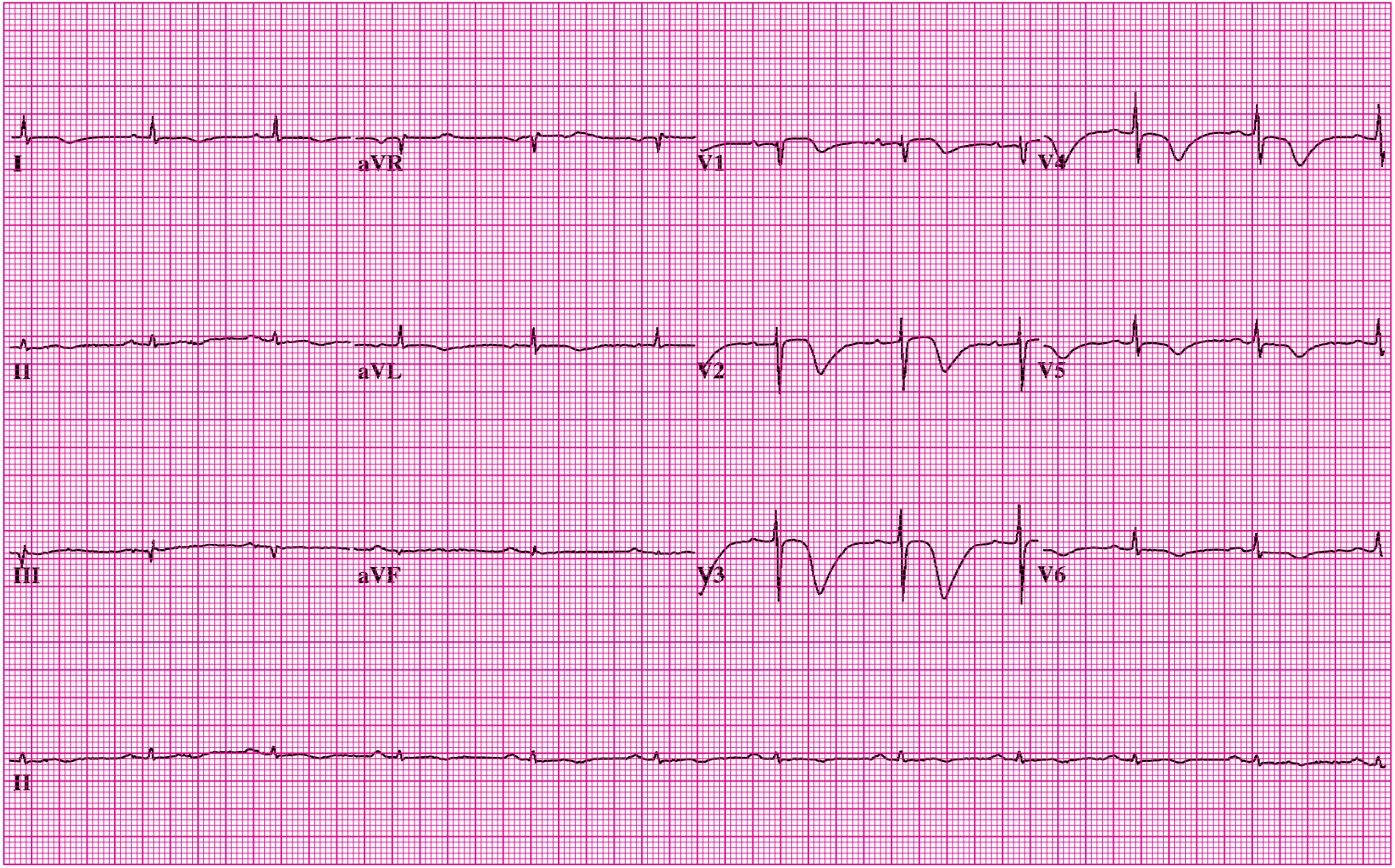
— POP QUIZ —

Pattern Recognition: Pacemakers

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Atrial pacing b. After an interval of time with no sensed atrial activity, an atrial paced beat occurs c. After an interval of time with no sensed atrial activity, a ventricular paced beat occurs d. Ventricular demand pacing e. Asynchronous ventricular pacing f. Interferes with the ECG diagnosis of acute MI and ventricular hypertrophy g. DDD pacing	In *atrial pacing*, each pacemaker stimulus is followed by an atrial depolarization. After an interval of time (A-A interval) with no sensed atrial activity, an atrial paced beat is delivered and a new cycle begins. In response to a native P wave, atrial pacing is inhibited and the pacemaker timing clock is reset. (Answer: a, b)
		In *ventricular demand pacing,* each pacemaker stimulus is followed by a QRS complex of different morphology than the intrinsic QRS. After an interval of time (V-V interval) with no sensed ventricular activity, a ventricular paced beat is delivered and a new cycle begins. In response to a native QRS, ventricular pacing is inhibited and the pacemaker timing clock is reset. A ventricular demand (VVI) pacemaker senses and paces only in the ventricle, and is oblivious to native atrial activity. (Answer: d, f)
		In *dual chamber (DDD) pacing*, if the rate of the intrinsic rhythm is slower than the programmed lower rate limit, atrial (A) and ventricular (V) paced beats will occur (separated by defined A-V and V-A intervals). Following ventricular-sensed activity (either QRS or V-paced beats), the timing clock is reset: If intrinsic atrial activity (P) is sensed prior to the end of the V-A interval, atrial output of the pacemaker will be inhibited; if no intrinsic atrial activity (P) is sensed by the end of the V-A interval, an atrial paced beat will occur. The pacemaker timing clock is also reset following atrial-sensed activity (either intrinsic P-wave or A-paced beats): If intrinsic ventricular activity (QRS) is sensed prior to the end of the A-V interval, ventricular output of the pacemaker will be inhibited; if no intrinsic ventricular activity (QRS) is sensed by the end of the A-V interval, a ventricular paced beat will occur. (Answer: a, b, f, g)

ECG 11. 73-year-old male with new onset neurological deficit:



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- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

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- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
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- ☐ 12. Sinoatrial exit block
- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
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- ☐ 47. LBBB, complete
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- ☐ 49. Nonspecific intraventricular conduction disturbance
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Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
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- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

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- ☐ 70. Digitalis effect
- ☐ 71. Digitalis toxicity
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- ☐ 73. Antiarrhythmic drug toxicity
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- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
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- ☐ 89. Sick sinus syndrome

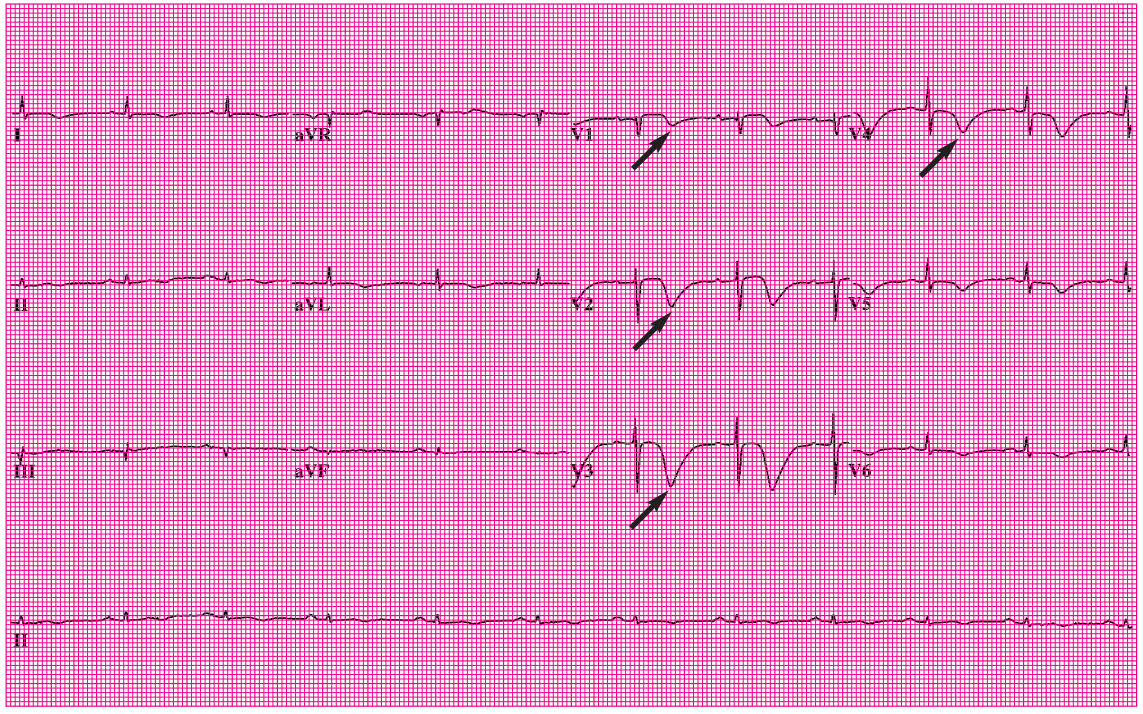
PACED RHYTHMS

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

ECG 11 was obtained in a 73-year-old male with an acute neurological deficit. The ECG shows sinus rhythm, low voltage in the limb leads (but not the precordial leads, so "low voltage" should not be coded), deep anterolateral T wave inversions (arrows), and a prolonged QT interval. Given the clinical presentation, the deeply inverted T waves and prolonged QT interval are likely due to the acute central nervous system event. However, since myocardial ischemia/infarction occurs in 15-20% of patients with stroke and since the anterior T wave changes are worrisome for ischemia, item 64 should also be coded.

Codes:

07	Sinus rhythm
64	ST and/or T wave abnormalities suggesting myocardial ischemia
68	Prolonged QT interval
86	Central nervous system disorder



Questions: ECG 11

1. ECG findings in acute central nervous system (CNS) disorders such as cerebral or subarachnoid hemorrhage include:

   a. Large upright T waves in precordial leads
   b. Increased QRS voltage
   c. Deeply inverted T waves in precordial leads
   d. Prolonged QT interval
   e. Prominent U waves in precordial leads

2. ECG changes associated with acute CNS events can mimic:

   a. Acute myocardial infarction
   b. Left ventricular hypertrophy
   c. Right ventricular hypertrophy
   d. Pericarditis
   e. Antiarrhythmic drug effects

Answers: ECG 11

1. Classic ECG changes of acute central nervous system disorders such as cerebral hemorrhage and subarachnoid hemorrhage usually occur in the precordial leads, and include large upright or deeply inverted T waves, prolonged QT interval, and prominent U waves. Other changes may include T wave notching, loss of T wave amplitude, diffuse ST segment elevation, and abnormal Q waves. Abnormalities of cardiac rhythm include atrial fibrillation, ventricular tachycardia, sinus bradycardia, and sinus tachycardia. Increased QRS voltage is not a feature of acute CNS disorders. (Answer: All except b)

2. ECG changes associated with acute CNS events can mimic acute myocardial infarction (abnormal Q waves, large upright T waves, ST elevation), myocardial ischemia (deep T wave inversion), acute pericarditis (diffuse ST elevation), and antiarrhythmic drug effects (prolonged QT interval, prominent U waves). Increased QRS amplitude mimicking ventricular hypertrophy does not occur. (Answer: a, d, e)

— Quick Review 11 —

Prolonged QT interval

- Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval — 0.44 / RR
- QT interval varies (directly/inversely) with heart rate — inversely
- The normal QT interval should be (less than/greater than) 50% of the RR interval when the ventricular rate is between 65-90. — less than

Central nervous system disorder

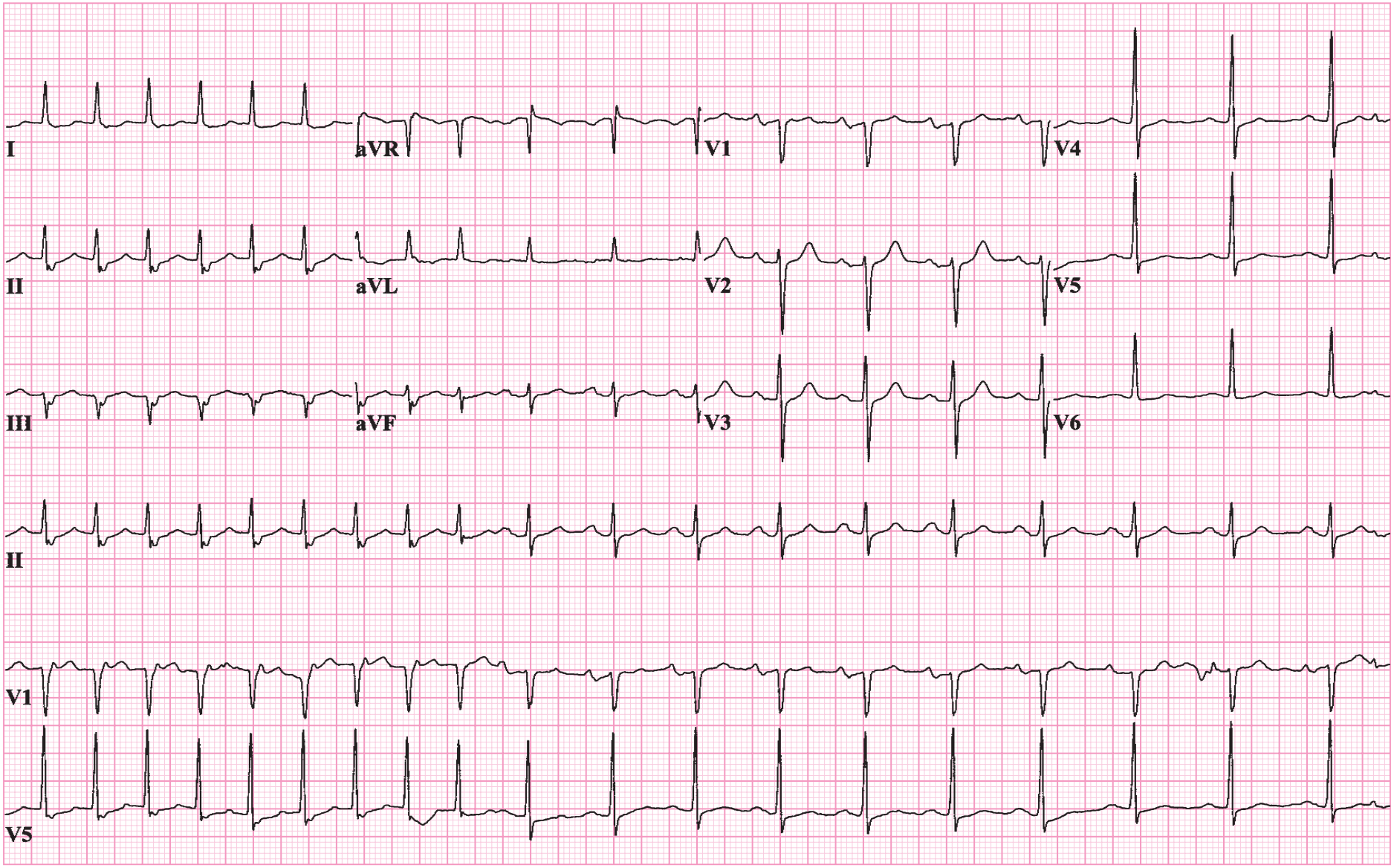
- "Classic changes" usually occur in the (limb/precordial) leads — precordial
  - Large upright or deeply inverted ____ waves — T
  - Prolonged ____ interval (often marked) — QT
  - Prominent ____ waves — U
- Other changes:
  - ST segment changes:
    - Diffuse ST elevation mimicking acute ____ — pericarditis
    - Focal ST elevation mimicking ____ — acute injury
    - ST depression may also occur (true/false) — true
  - Abnormal ____ waves mimicking MI — Q
  - Almost any rhythm abnormality including sinus tachycardia or bradycardia, junctional rhythm, VPCs, ventricular tachycardia, etc. (true/false) — true

Differential Diagnosis

Group Beating

- Mobitz Type I second-degree AV block
- Blocked APCs
- Type II second-degree AV block
- Concealed His-bundle depolarizations

ECG 12. 81-year-old female with palpitations:



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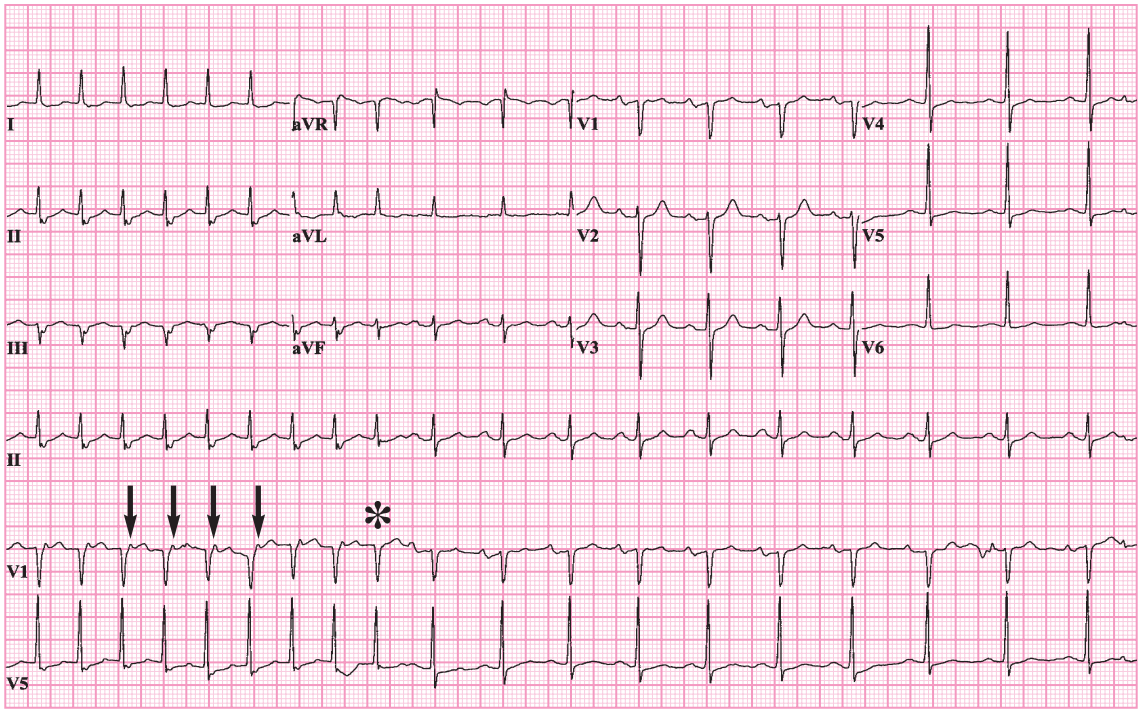
ECG 12 was obtained from an 81-year-old female with palpitations. The ECG shows paroxysmal supraventricular tachycardia (PSVT). The PSVT mechanism is consistent with typical reentry within the AV node (i.e., antegrade conduction down the slow pathway and retrograde conduction up the fast pathway). The lead V_1 rhythm strip initially records SVT with an R' at the end of each QRS complex (arrow). After the 9th QRS complex (asterisk), the rhythm converts to sinus; at the conversion point, the R' is no longer present. The R' represents retrograde conduction through the AV node over the fast pathway with activation of the atrium occurring at the tail end of the QRS complex. This finding strongly suggests that the tachycardia mechanism is typical reentry within the AV node (i.e., AV node reentrant tachycardia, AVNRT).

Codes:

07	Sinus rhythm
17	Supraventricular tachycardia, paroxysmal



Questions: ECG 12

1. Which of the following statements about atrioventricular nodal reentrant tachycardia (AVNRT) are true:

   a. The majority of cases of paroxysmal supraventricular tachycardia (PSVT) are due to reentry within the atrioventricular node
   b. The most common mechanism of AVNRT involves antegrade conduction (from atrium to ventricle) over the fast AV nodal pathway and retrograde conduction over the slow AV nodal pathway (typical P wave inverted in II, III, aVF, and may appear as R' in V_1)
   c. In the present ECG, an accessory pathway is present connecting the atrium and ventricle in the region of the AV node

Answers: ECG 12

1. The most common mechanism of PSVT is reentry within the atrioventricular node. This is termed typical AV node reentry tachycardia and utilizes the slow AV nodal pathway for conduction from the atrium to the ventricle and the fast AV nodal pathway for conduction from the ventricle back to the atrium (see Table). This gives rise to a short RP tachycardia (RP interval < 50% RR interval), in which the retrograde P wave is either buried in the QRS complex or seen at the tail end of the QRS complex, especially in V_1, where it appears as an R' complex. In contrast, the atypical form of AV node reentry tachycardia conducts in the reverse direction — conduction from the atrium to the ventricle occurs over the fast AV nodal pathway, giving rise to a short PR interval, and conduction from the ventricle to the atrium occurs over the slow AV nodal pathway, giving rise to a long RP interval. The slow and fast AV nodal pathways are components of the AV node and are not a separate accessory pathway as in WPW. (Answer: a)

Table. Types of AV Nodal Reentrant Tachycardia (AVNRT)

Type	Reentrant Circuit: Antegrade Conduction	Reentrant Circuit: Retrograde Conduction	ECG
Typical AVNRT (70% of SVT's)	Slow pathway	Fast pathway	Short RP tachycardia (RP interval < 50% of RR interval)
Atypical AVNRT (2-5% of SVT's)	Fast pathway	Slow pathway	Long RP tachycardia (RP interval > 50% of RR interval)

— Quick Review 12—

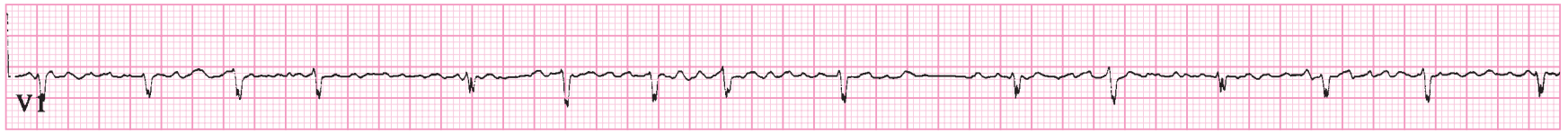
Supraventricular tachycardia, paroxysmal	
• (Regular/irregular) rhythm	Regular
• Rate > ____ per minute	100
• P waves (easily/not easily) identified	not easily
• QRS complex is usually (narrow/wide)	narrow
• If rate is 150 per minute, consider ____	atrial flutter with 2:1 block

— POP QUIZ —

Find The Imposter

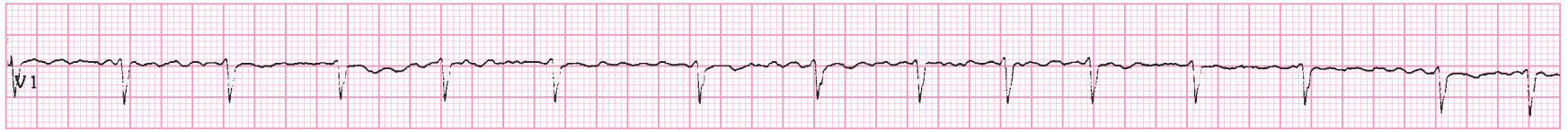
Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

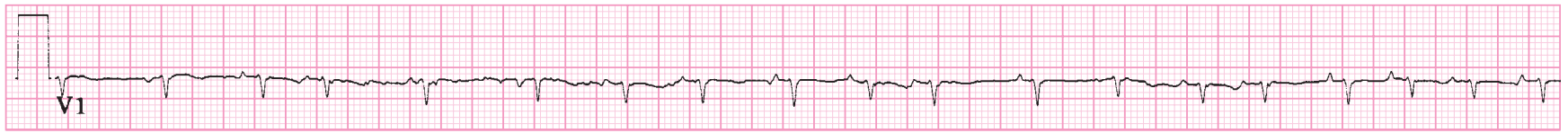


B.

C.



D.

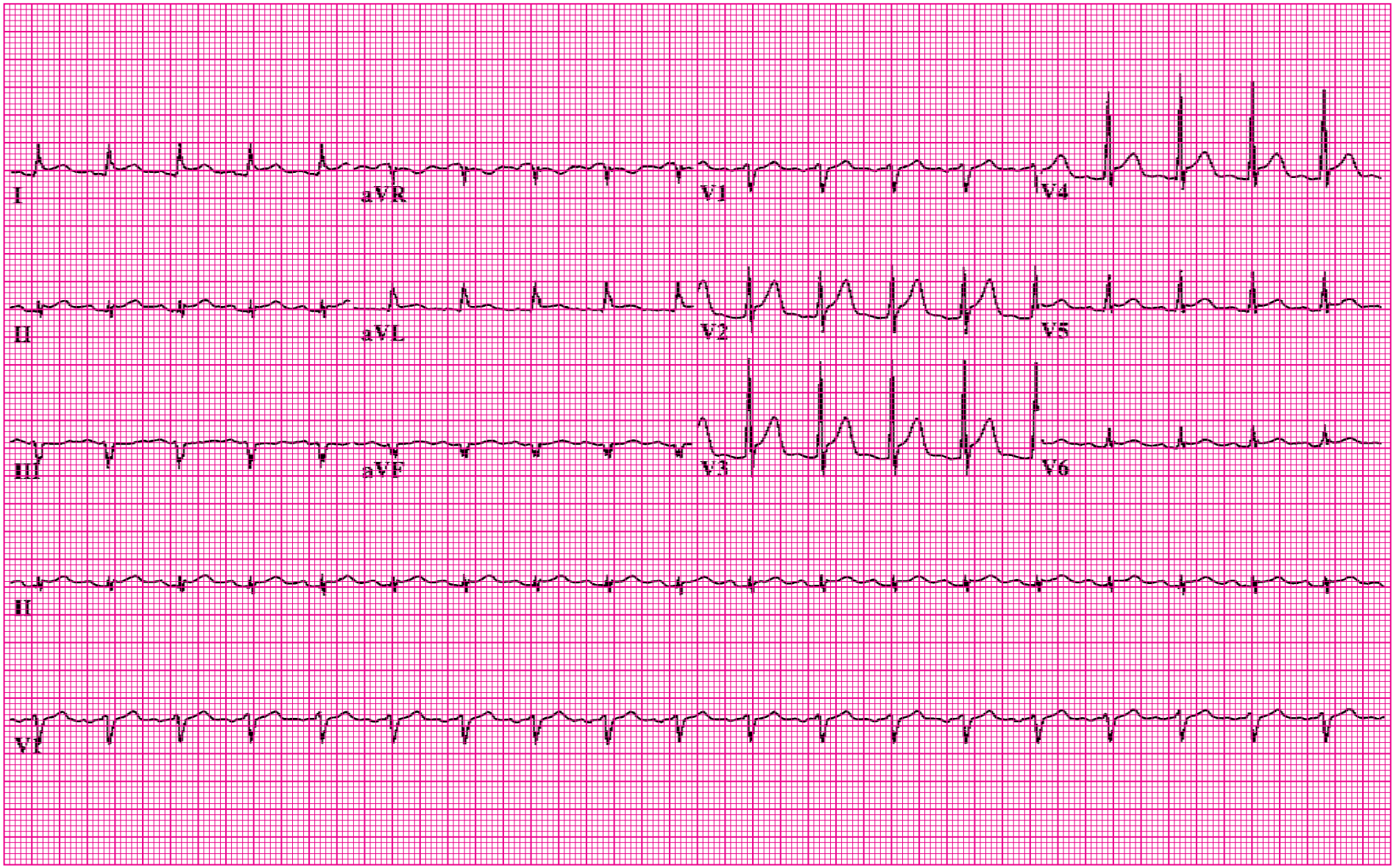


Answer: Tracings A, B, and C demonstrate atrial fibrillation manifest as irregularly irregular rhythms; fibrillatory waves of varying amplitude, duration and morphology; and absent of discrete P waves. Tracing D shows multifocal atrial tachycardia and is the imposter. MAT manifests as an atrial rate > 100 bpm, 3 or more different P wave morphologies (each originating from a separate atrial focus), and varying PP and PR intervals. Multifocal atrial tachycardia, like atrial fibrillation is characterized by an irregular rhythm. However, while definite atrial depolarizations and P waves are noted in tracing D, they are absent the tracings A, B, and C. MAT may also be confused with sinus tachycardia with multifocal APCs.

Don’t Forget!

- Atrial flutter waves can deform QRS complexes, ST segments, and T waves to mimic intraventricular conduction delay or myocardial ischemia
- Think digoxin toxicity when regularization of the QRS is present during atrial fibrillation — this is usually due to complete heart block with junctional tachycardia
- Think Wolff-Parkinson-White syndrome in patients with atrial fibrillation when the ventricular rate exceeds 200 per minute and the QRS is wide (> 0.12 seconds)
- Look for retrograde P waves after ventricular premature complexes and other junctional, ventricular, or low ectopic atrial rhythms
- In junctional premature complexes, the P wave may precede the QRS by ≤ 0.11 seconds (retrograde atrial activation), be buried in the QRS (and not visualized), or follow the QRS complex
- Although multiform VPCs are usually multifocal in origin (i.e., originate from more than one ventricular focus), a single ventricular focus can produce VPCs of varying morphology

ECG 13. 46-year-old female with chest pain:



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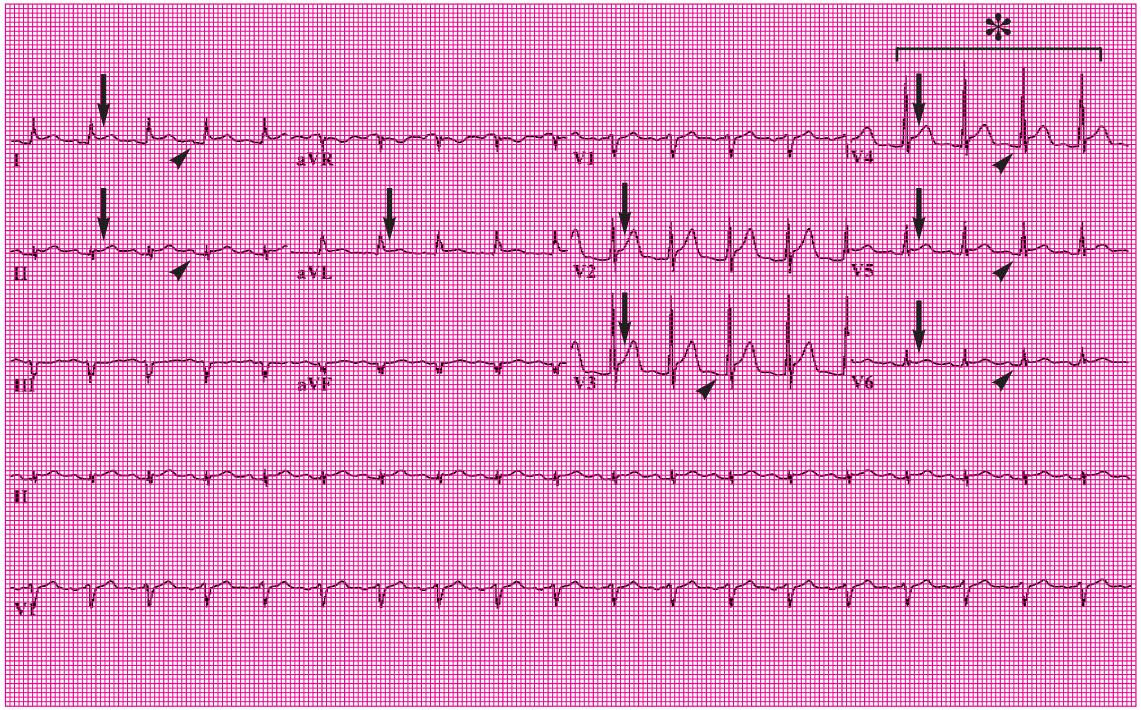
ECG 13 was obtained in a 46-year-old female with chest pain. The ECG shows sinus tachycardia, diffuse ST segment elevation (arrows), mild PR segment depression (relative to the TP segment) (arrowheads), and PR segment elevation in lead aVR. These findings are consistent with acute pericarditis. The diffuse nature of the ST segment elevation in the absence of Q waves, the upwardly concave configuration of the ST segments, and the PR depression suggest pericarditis rather than acute myocardial ischemia. Subtle electrical alternans is noted in lead V_4 (asterisk), which is likely due to pericardial effusion.

Codes:

10	Sinus tachycardia (> 100)
38	Electrical alternans
83	Pericardial effusion
84	Acute pericarditis



Questions: ECG 13

1. The present ECG is consistent with the diagnosis of early repolarization:

   a. True
   b. False

2. Which of the following statements about pericarditis are true:

   a. Electrical alternans is a common and specific finding in pericarditis complicated by pericardial effusion
   b. PR segment depression occurs in all leads
   c. P wave amplitude usually decreases

Answers: ECG 13

1. The diffuse ST segment elevation in this tracing is consistent with early repolarization. (PR segment depression can sometimes be seen on normal ECGs and does not exclude this diagnosis). However, in a patient with pleuritic chest pain, acute pericarditis is the most likely diagnosis. (Answer: a)

2. The typical evolutionary pattern of ST and T wave changes associated with pericarditis include: (1) diffuse ST elevation (except for ST depression in aVR); (2) return of the ST segment to baseline with decreasing T wave amplitude; (3) T wave inversion; and (4) return of the ECG to normal. However, pericarditis may be focal (e.g., post-pericardiotomy) rather than diffuse, resulting in regional rather than diffuse ST elevation. Also, classic ST and T wave changes are more likely to occur in purulent compared to idiopathic, rheumatic, or malignant pericarditis. Pericarditis does not typically affect P wave amplitude or contour, although P wave alternans may occur if pericardial effusion is present. While PR depression is common and often diffuse, it is typically elevated in lead aVR. Electrical alternans is present in a minority of patients with pericardial effusion, and can be seen in several other conditions (e.g., severe LV failure, deep respirations, coronary artery disease). (Answer: none)

— Quick Review 13 —

ST and/or T wave changes suggesting acute pericarditis	
• Classic evolutionary pattern consists of ____ stages	4
▸ Stage 1: Upwardly concave ST segment ____ in almost all leads	elevation
▸ Stage 2: ST junction (J point) returns to baseline and T wave amplitude begins to (increase/decrease)	decrease
▸ Stage 3: T waves (invert/remain upright)	invert
▸ Stage 4: ECG (does/does not) return to normal	does
• Other clues to acute pericarditis:	
▸ Sinus ____	tachycardia
▸ PR ____ early (PR elevation in aVR)	depression
▸ (High/low) voltage QRS	low
▸ Electrical alternans if pericardial ____ is present	effusion
Pericardial effusion	
• (High/low) voltage QRS	Low
• Electrical ____, especially if complicated by cardiac ____	alternans tamponade
• Other features of acute ____ may also be present	pericarditis

Don’t Forget!

Age of myocardial infarction can be approximated from the ECG pattern:

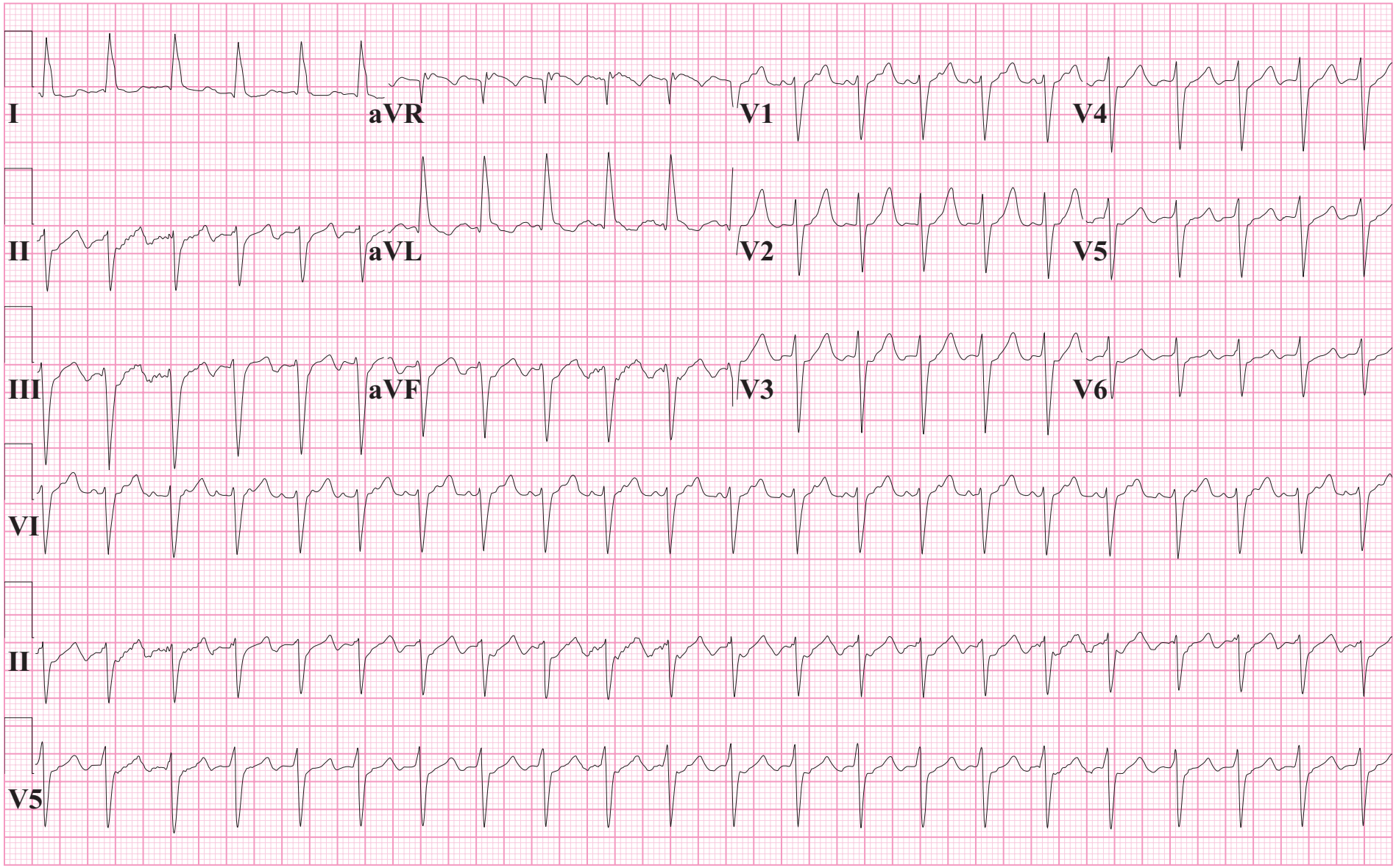
- Acute MI: Abnormal Q waves, ST elevation (associated ST depression is sometimes present in noninfarct leads)
- Recent MI: Abnormal Q waves, isoelectric ST segments, ischemic (usually inverted) T waves
- Old MI: Abnormal Q waves, isoelectric ST segments, nonspecific or normal T waves

MI may be present without Q waves in:

- Anterior MI: May only see low anterior R wave forces with decreasing R wave progression in leads V_2-V_5
- Posterior MI: Dominant R wave with ST depression in leads V_1-V_3

RVH, WPW, and RBBB interfere with the ECG diagnosis of posterior MI

ECG 14. 79-year-old male with “a racing heart”:



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- ☐ 21. AV junctional escape complexes
- ☐ 22. AV junctional rhythm/tachycardia

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- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
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- ☐ 34. Wolff-Parkinson-White pattern
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- ☐ 37. Right axis deviation (> +100°)
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- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
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REPOLARIZATION ABNORMALITIES

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- ☐ 62. Normal variant, juvenile T waves
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- ☐ 76. Hypercalcemia
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- ☐ 79. Atrial septal defect, primum
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- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

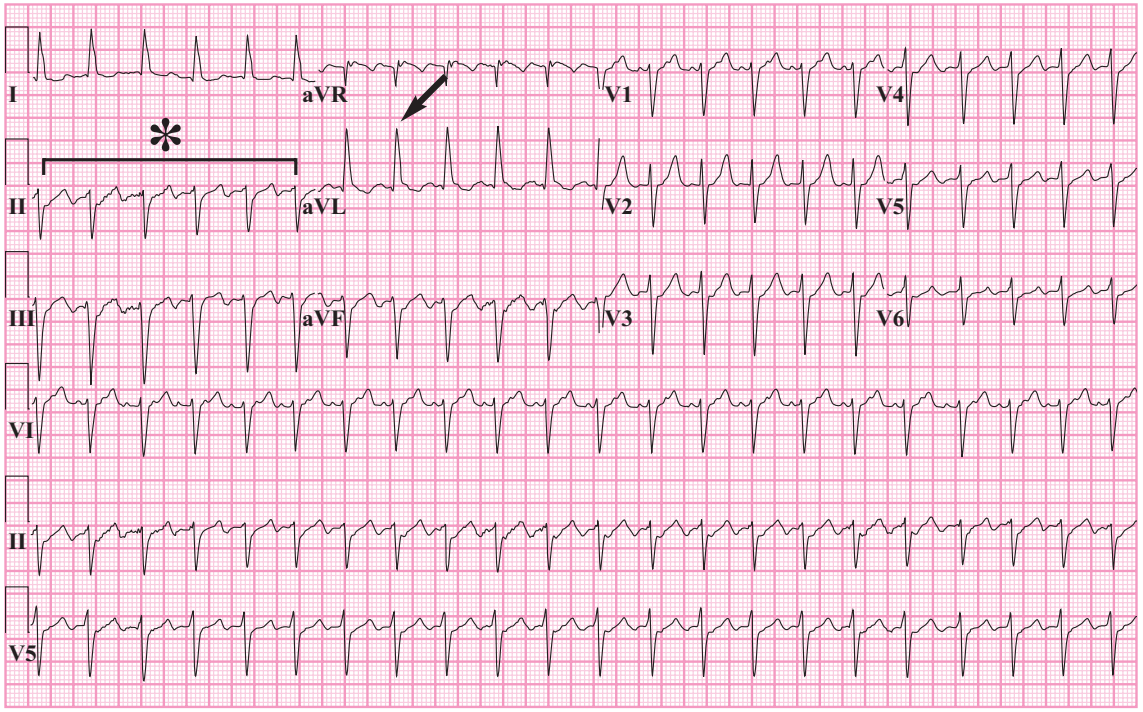
PACED RHYTHMS

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

ECG 14 was obtained from a 79-year-old male while being evaluated for complaints of "a racing heart." The ECG shows a regular, rapid rhythm at 133 beats/minute and left anterior fascicular block (LAFB) (QRS axis – 56°). The sawtooth pattern of the baseline, most obvious in leads II (asterisk), III and aVF, is diagnostic of atrial flutter (atrial rate = 266 beats/minute) with 2:1 AV block, resulting in a ventricular rate of 133 beats/minute. The R wave in aVL measures 14 mm (arrow). Under usual circumstances, an R wave measuring ≥12 mm is a specific finding for left ventricular hypertrophy (LVH). However, LVH should not be diagnosed solely on the basis of aVL voltage in the presence of LAFB, which can artifactually increase the size of the R wave in aVL. The qR complex in leads I and aVL, the rS complex in lead III, and poor R wave progression in the precordial leads are also consistent with LAFB.

Codes:

18	Atrial flutter
32	AV block 2:1
45	Left anterior fascicular block



Questions: ECG 14

1. By definition, left anterior fascicular block is associated with a mean QRS axis of:

   a. – 90° to + 90°
   b. – 30° to – 60°
   c. – 45° to – 90°
   d – 45° to – 100°

2. By definition, left posterior fascicular block is associated with a mean QRS duration of:

   a. +100° to +180°
   b. +90° to +180°
   c. +80° to +180°
   d. +90° to +270°

3. Which of the following findings on ECG precludes the diagnosis of left posterior fascicular block:

   a. LVH
   b. RVH
   c. Lateral MI
   d. Inferior MI

Answers: ECG 14

1. Left anterior fascicular block results in a mean QRS axis between –45° and –90°. LAFB can increase the size of the R wave in leads I and/or aVL, predisposing to a false-positive diagnosis of LVH. LAFB also frequently results in poor R wave progression across precordial leads, sometimes falsely suggesting a diagnosis of old anterior infarction. LAFB is a diagnosis of exclusion and should not be coded if another cause for left axis deviation is present (e.g., LVH, inferior MI, chronic lung disease, LBBB, ostium primum ASD, severe hyperkalemia). LAFB is generally associated with organic heart disease or congenital heart disease and is seen only rarely in normal hearts. (Answer: c)

2. Left posterior fascicular block (LPFB) results in a mean QRS axis of + 100° to + 180°. The QRS duration is generally normal or only slightly prolonged (0.08-0.10 seconds). LPFB is much less prevalent than LBBB, RBBB, or LAFB. Coronary artery disease is most common cause of LPFB, and LPFB is rarely seen in normal hearts. (Answer: a)

3. LPFB is a diagnosis of exclusion and should not be coded if another cause for right axis deviation is present, including RVH, a vertical heart, emphysema, pulmonary embolism, lateral MI, dextrocardia, limb lead reversal, or Wolff-Parkinson-White. (Answer: b, c)

— Quick Review 14 —

Atrial flutter

- Rapid (regular/irregular) atrial undulations ("F" waves) at a rate of ____ per minute — regular, 240-340
- Flutter rate may (increase/decrease) in the presence of Types IA, IC or III antiarrhythmic drugs — decrease
- Flutter waves in leads II, III, AVF are typically (inverted/upright) (with/without) an isoelectric baseline — inverted,without
- Flutter waves in lead V_1 are typically small (positive/negative) deflections (with/without) a distinct isoelectric baseline — positive, with
- QRS complex may be normal or aberrant (true/false) — true
- AV conduction ratio, i.e., ratio of flutter waves to QRS complexes, is usually (fixed/variable) — fixed
  - Conduction ratios of 1:1 and 3:1 are (common/uncommon) — uncommon
  - In untreated patients, AV block ≥ ____ suggests the coexistence of AV conduction disease — 4:1

AV block, 2:1

- Regular sinus or ____ rhythm — ectopic atrial
- 2 ____ waves for every QRS complex — P
- Can be Mobitz type I or type II 2° AV block (true/false) — true
- Progressive prolongation of the ____ interval and shortening of the ____ interval until a P wave is blocked — PR, RR
- RR interval containing the nonconducted P wave is (less than/equal to/greater than) the sum of two PP intervals — less than
- Results in ____ beating due to the presence of nonconducted P waves — group

— Quick Review 14 —

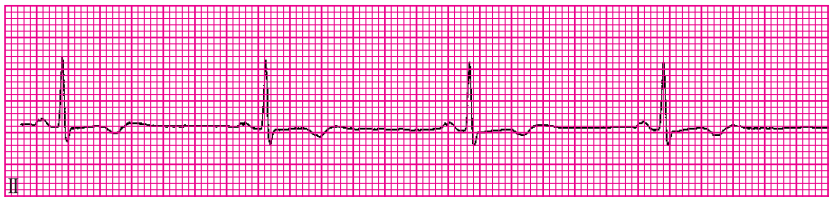
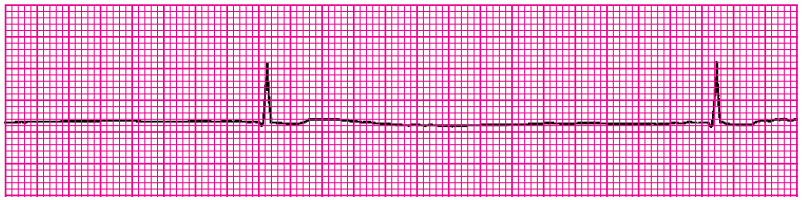
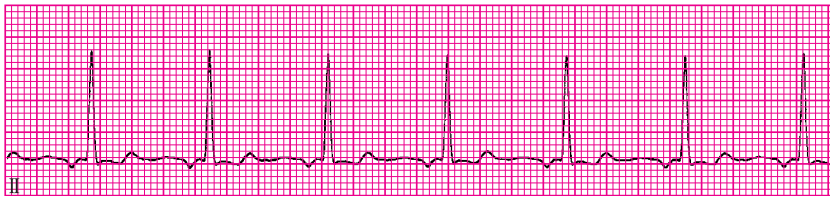
Left anterior fascicular block

- ____ axis deviation with a mean QRS axis between ____ and ____ degrees — left, -45, -90
- (qR/rS) complex in leads I and aVL — qR
- (qR/rS) complex in lead III — rS
- Normal or slightly prolonged QRS duration (true/false) — true
- No other cause for left axis deviation should be present (true/false) — true
- Poor R wave progression is (common/uncommon) — common

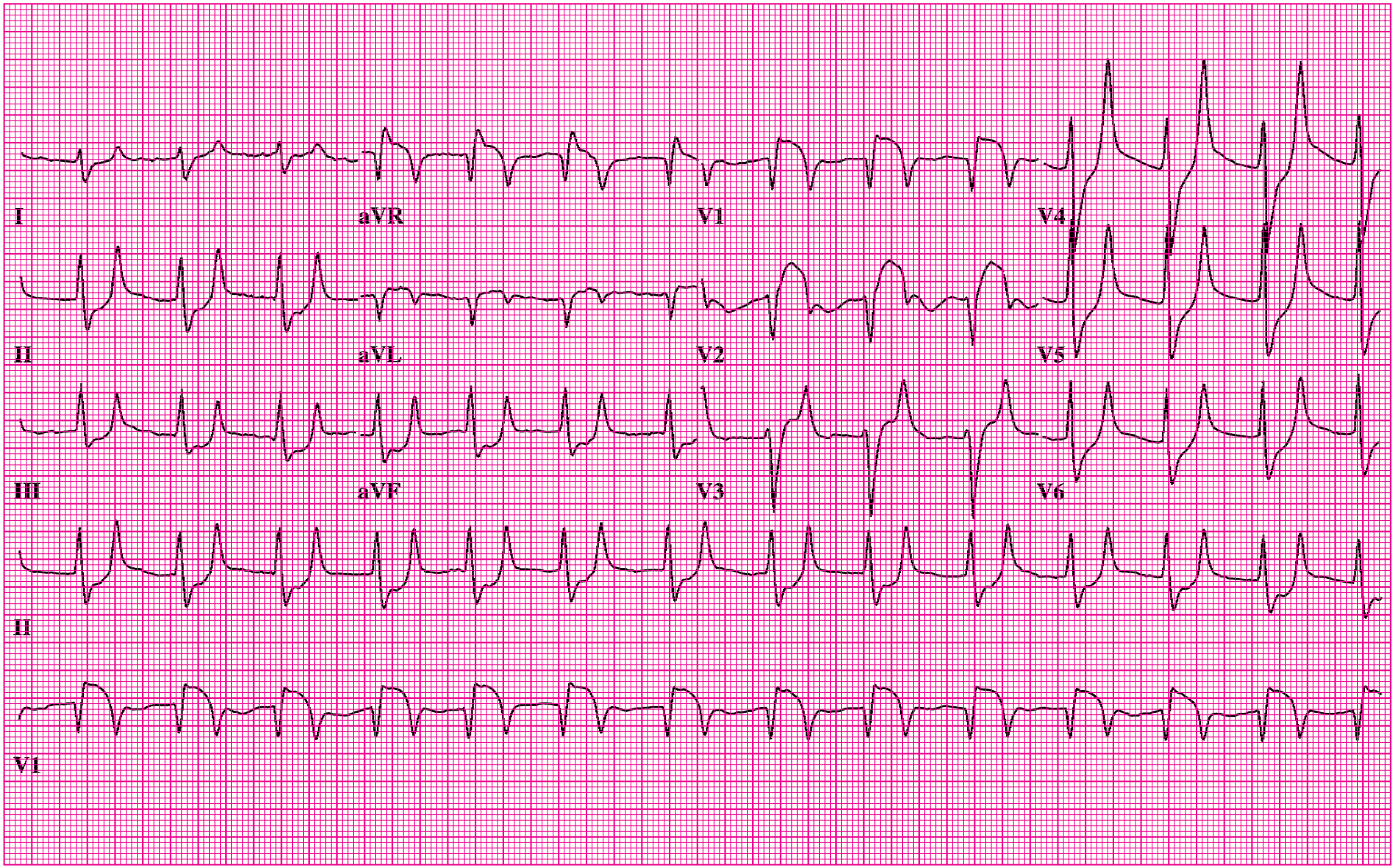
— POP QUIZ —

Rhythm Recognition: HR < 100; Regular RR Interval

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Sinus bradycardia. Description: Regular sinus rhythm (normal P wave axis and morphology) at a rate < 60 per minute. Causes include high vagal tone, myocardial infarction (usually inferior), drugs (beta-blockers, digitalis, amiodarone, verapamil, others), hypothyroidism, hypothermia, obstructive jaundice, hyperkalemia, increased intracranial pressure, and sick sinus syndrome. Note: If the atrial rate is < 40 per minute, consider 2:1 sinoatrial exit block.
	Answer: Sinus pause/arrest with junctional escape complexes. Description: PP interval (pause) > 1.6-2.0 seconds, due to transient failure of impulse formation at the sinoatrial node. Causes include sinus node dysfunction, organic heart disease, drugs, hyperkalemia, vagal stimulation, and MI. Cannot be differentiated from complete failure of sinoatrial conduction (3° sinoatrial exit block) on surface ECG.
	Answer: Ectopic atrial rhythm. Description: Ectopic (nonsinus) P wave at a rate < 100 per minute. P waves can be upright (when the ectopic atrial focus originates near the sinus node) or inverted (when the ectopic focus originates in the lower atrium). PR interval can be prolonged, normal or short, depending on the proximity of the ectopic atrial impulse to the AV node and whether delay is present in the AV conduction system. QRS and QT interval can be normal or prolonged. Note: Inverted P waves in II, III, and aVF suggest either a low atrial rhythm or an AV junctional rhythm with retrograde atrial activation. To distinguish between these mechanisms, measure the PR interval: PR interval > 0.11 seconds suggests a low atrial rhythm; PR interval ≤ 0.11 seconds suggests an AV junctional rhythm.

ECG 15. 66-year-old female with chest pain:



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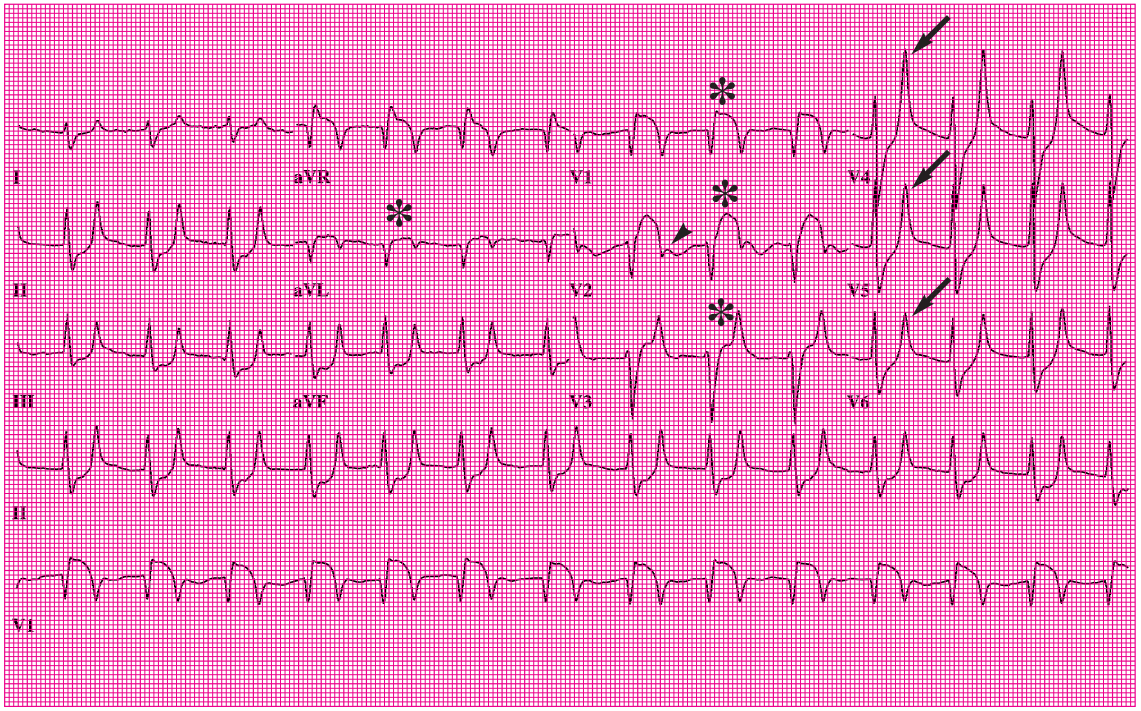
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 15 was obtained in a 66-year-old female with chest pain. The ECG shows sinus rhythm, first-degree AV block, and left posterior fascicular block. The most striking features on this tracing include marked peaking of the T waves (arrows) and a myocardial injury pattern in leads V_1-V_3 and aVL (asterisks). Since a pathological Q wave is present in lead aVL only, acute myocardial infarction should not be coded. This woman was shown to have an occluded left anterior descending (LAD) coronary artery and a potassium level of 8.0 mg/dL.

Codes:

07	Sinus rhythm
29	AV block, 1°
46	Left posterior fascicular block
49	Nonspecific intraventricular conduction disturbance
65	ST and/or T wave abnormalities suggesting myocardial injury
66	ST and/or T wave abnormalities suggesting electrolyte disturbances
74	Hyperkalemia



Questions: ECG 15

1. Peaked T waves can occur with:

   a. Intracranial bleeding
   b. Acute myocardial infarction
   c. Left ventricular hypertrophy (LVH)
   d. Normal variant early repolarization abnormality
   e. Hyperkalemia
   f. Left bundle branch block (LBBB)

2. Hyperkalemia can cause all of the following ECG changes except:

   a. QRS widening
   b. PR prolongation
   c. Prominent U waves
   d. Left anterior fascicular block

Answers: ECG 15

1. Peaked T waves can be seen in hyperkalemia, acute myocardial infarction, intracranial bleeding, and normal variant early repolarization abnormality. Other causes of peaked T waves include marked LVH (usually in right precordial leads) and LBBB. (Answer: all)

2. Hyperkalemia can cause PR prolongation, QRS widening, peaked T waves, and left anterior fascicular block. Hypokalemia, not hyperkalemia, is associated with prominent U waves. (Answer: c)

— Quick Review 15 —

Peaked T waves	
• T wave > ____ mm in the limb leads or > ____ mm in the precordial leads	6, 10
Hyperkalemia	
• ***K^+ = 5.5 - 6.5 mEq/L***	
▸ Tall, peaked, narrow based ____ waves	T
▸ QT interval (shortening/lengthening)	shortening
▸ (Reversible/irreversible) left anterior or posterior fascicular block	Reversible
• ***K^+ = 6.5 - 7.5 mEq/L***	
▸ ____ degree AV block	First
▸ Flattening and widening of the ____ wave	P
▸ ST segment (depression/elevation)	depression
▸ ____widening	QRS
• ***K^+ > 7.5 mEq/L***	
▸ Disappearance of ____ waves	P
▸ LBBB, RBBB, or markedly widened and diffuse intraventricular conduction delay resembling a _____ wave pattern	sine
▸ Arrhythmias and conduction disturbances including VT, VF, idioventricular rhythm, asystole (true/false)	true

— POP QUIZ —

Make The Diagnosis

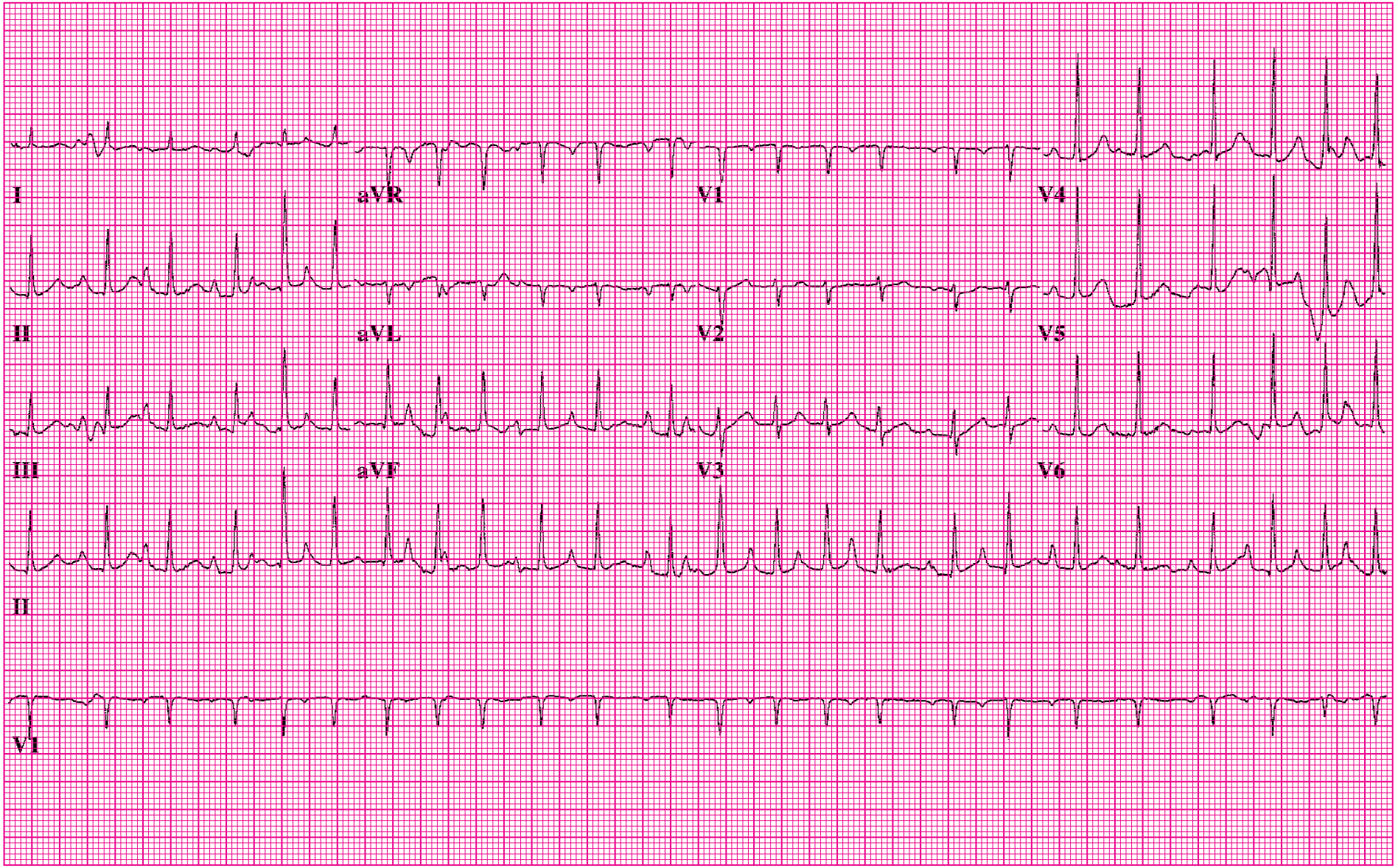
Instructions: Determine the clinical disorder that best corresponds to the ECG features listed below (see items 70-89 on answer sheet for options).

ECG Features	Diagnosis
• QT interval shortening • May see PR prolongation • No effect on P, QRS, and T wave	Hypercalcemia
• Earliest and most common finding is prolonged QT interval from ST segment lengthening • Occasional flattening, peaking, or inversion of T waves	Hypocalcemia
• Tall, peaked, narrow based T waves • QT interval shortening • Reversible left anterior or posterior fascicular block • QRS widening • Disappearance of P waves	Hyperkalemia
• Prominent U waves • ST segment depression • Flattened T waves • Prolonged QT interval • Arrhythmias and conduction disturbances, including paroxysmal atrial tachycardia with block, first-degree AV block, Type I second-degree AV block, AV dissociation, VPCs, ventricular tachycardia, and ventricular fibrillation	Hypokalemia
• Typical RSR' or rSR' complex in lead V_1 with a QRS duration < 0.11 seconds • Incomplete RBBB • Right axis deviation ± right ventricular hypertrophy • Right atrial abnormality in ~ 30% • First-degree AV block in < 20%	Atrial septal defect, secundum

Don’t Forget!

- In RBBB, mean QRS axis is determined by the initial unblocked 0.06-0.08 seconds of QRS, and should be normal unless left anterior or left posterior fascicular block is present
- RBBB does not interfere with the ECG diagnosis of ventricular hypertrophy or Q-wave MI
- LAFB may result in a false-positive diagnosis of LVH based on voltage criteria using leads I or aVL
- Left anterior fascicular block can mask the presence of inferior wall MI
- Left posterior fascicular block can mask the presence of lateral wall MI
- LBBB interferes with QRS axis and the ECG diagnoses of ventricular hypertrophy and acute MI
- Intermittent LBBB is more commonly seen at high rates (tachycardia-dependent), but may be bradycardia-dependent as well
- In up to 30% of cases, P-pulmonale can manifest as left atrial enlargement on ECG. Suspect this possibility when left atrial abnormality is present in lead V_1

ECG 16. 86-year-old male with palpitations and shortness of breath:



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- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 57. Inferior (age recent or acute)
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- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

PACED RHYTHMS

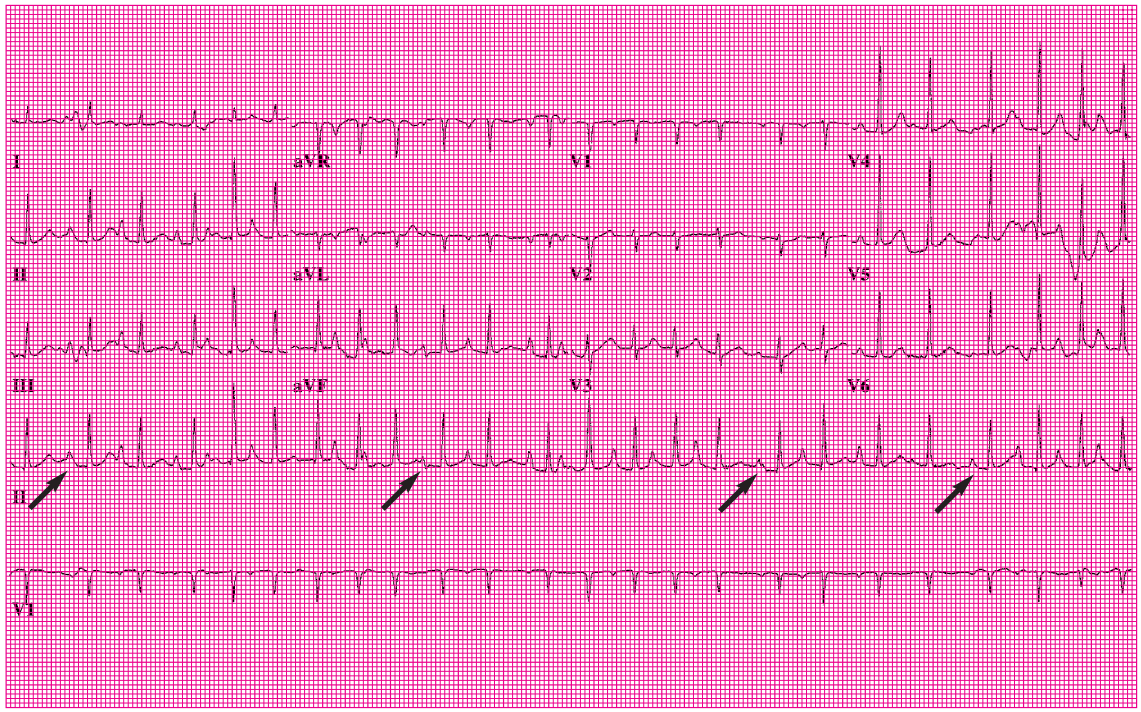
- ☐ 90. Atrial or coronary sinus pacing
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 16 was obtained from an 86-year-old male with palpitations and shortness of breath. The ECG shows multifocal atrial tachycardia and nonspecific ST and/or T wave changes. Several different P wave morphologies are evident in the lead II rhythm strip (arrows).

Codes:

16 Atrial tachycardia, multifocal

63 Nonspecific ST and/or T wave abnormalities



Questions: ECG 16

1. ECG features of multifocal atrial tachycardia include:

    a. Variable PR and RR intervals
    b. Absence of one dominant atrial pacemaker
    c. Atrial rate > 100 per minute
    d. P waves of at least three morphologies
    e. Isoelectric baseline between P waves

2. Irregularly irregular rhythms include:

    a. Sinus tachycardia with frequent atrial premature complexes
    b. Multifocal atrial tachycardia
    c. Ventricular trigeminy
    d. Atrial fibrillation
    e. Atrial flutter with 4:1 AV conduction

Answers: ECG 16

1. Multifocal atrial tachycardia (MAT) is an irregular rhythm with an atrial rate > 100 per minute and at least 3 different P wave morphologies (each originating from a separate atrial focus). The absence of one dominant atrial pacemaker distinguishes MAT from sinus tachycardia with frequent multifocal atrial premature complexes, and the presence of an isoelectric baseline between P waves distinguishes MAT from coarse atrial fibrillation. Rhythm irregularity results in variable PR and RP intervals. MAT does not effect or require AV conduction and can persist during AV block. (Answer: all)

2. Irregularly irregular rhythms include atrial fibrillation, multifocal atrial tachycardia, and sinus tachycardia with frequent APCs. Ventricular trigeminy (two normal QRS complexes followed by a ventricular premature complex in a repeating pattern) results in a regularly irregular rhythm. Atrial flutter with 4:1 AV conduction presents as a regular rhythm. (Answer: a, b, d)

— Quick Review 16 —

Atrial tachycardia, multifocal	
• Atrial rate > _____ per minute	100
• P waves with ≥ _____ morphologies	3
• PR, RR and RP intervals (are constant/vary)	vary
• May be confused with sinus tachycardia with multifocal APCs, or atrial fibrillation/flutter with a rapid ventricular response, but:	
▸ Unlike sinus tachycardia with multifocal APCs, multifocal atrial tachycardia (does/does not) manifest a dominant P wave morphology	does not
▸ Unlike atrial fibrillation/flutter, multifocal atrial tachycardia has a distinct ____ baseline	isoelectric
• P waves may be blocked or conducted with a narrow or aberrant QRS complex (true/false)	true
Nonspecific ST and/or T wave abnormalities	
• Slight ____ segment depression or elevation	ST
• Slightly inverted or flat ____ wave	T

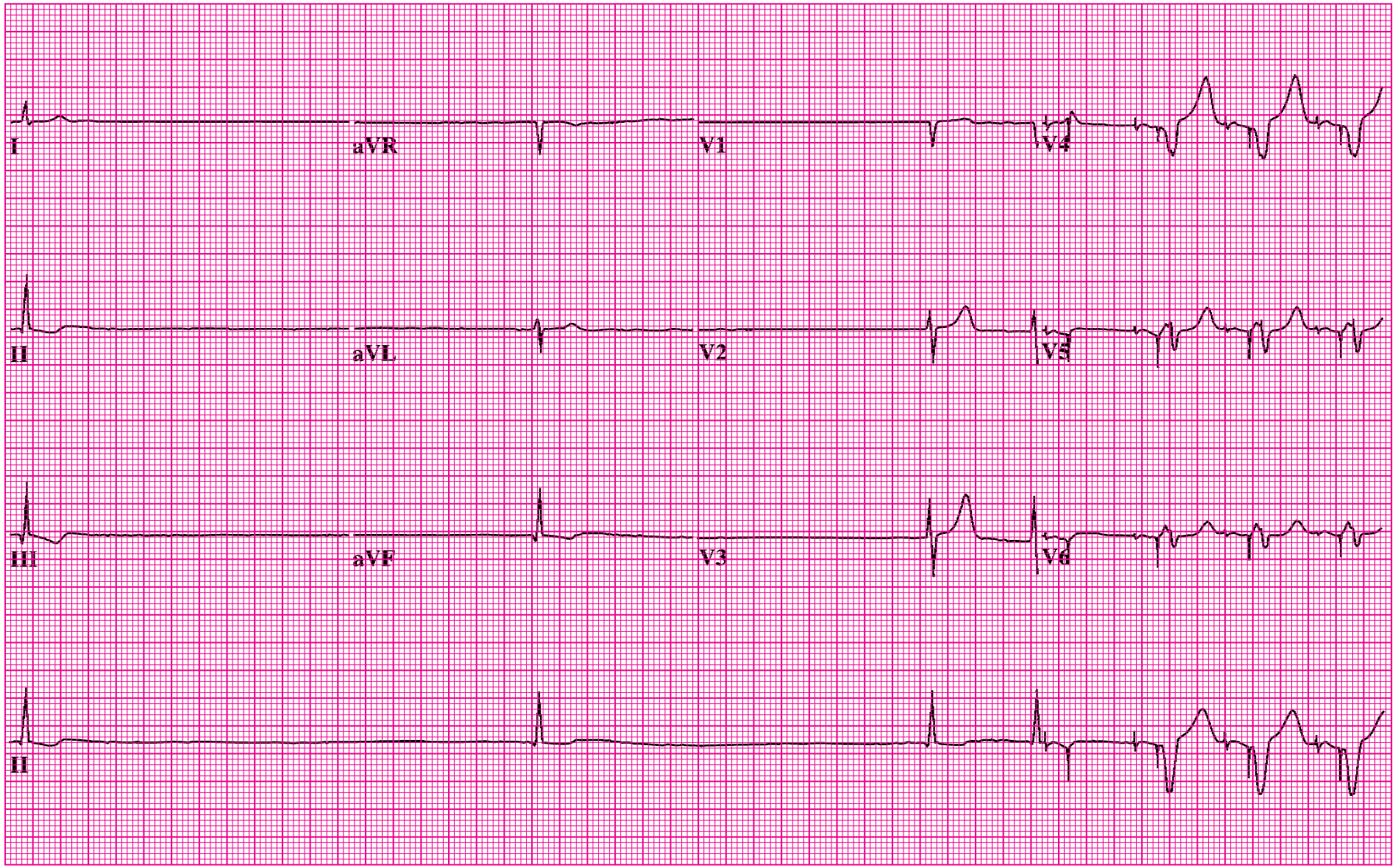
Differential Diagnosis

NONSPECIFIC ST AND/OR T WAVE ABNORMALITIES

Slight (< 1mm) ST depression or elevation
and/or flat or slightly inverted T waves

- Organic heart disease
- Drugs (e.g., quinidine)
- Electrolyte disorders (e.g., hypokalemia)
- Hyperventilation
- Hypothyroidism
- Stress
- Pancreatitis
- Pericarditis
- CNS disorders
- LVH
- RVH
- Bundle branch block
- Healthy adults (normal variant)

ECG 17. 64-year-old female with recurrent syncope:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
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- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 19. Atrial fibrillation

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- ☐ 21. AV junctional escape complexes
- ☐ 22. AV junctional rhythm/tachycardia

VENTRICULAR RHYTHMS

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- ☐ 32. AV block, 2:1
- ☐ 33. AV block, 3°
- ☐ 34. Wolff-Parkinson-White pattern
- ☐ 35. AV dissociation

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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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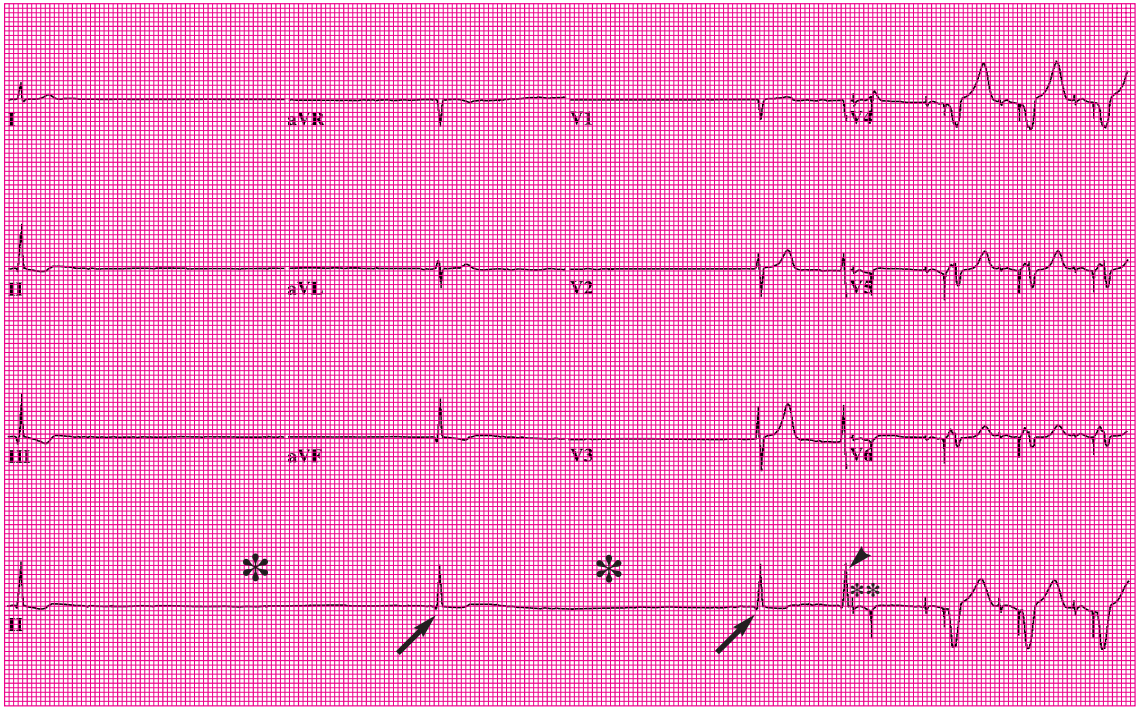
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 17 was obtained in a 64-year-old female with recurrent syncope. The ECG shows sinus arrest (asterisk) with junctional escape complexes (arrows). Near the end of the tracing is a normally conducted junctional premature beat (arrowhead), which is followed immediately by output from an AV sequential pacemaker (double asterisk). The pacemaker fails to sense the premature beat, and fails to fire during sinus arrest. Failure of the pacemaker to capture the ventricle the first time it fires occurs because the myocardium has not yet repolarized; item 93, "pacemaker malfunction, not constantly capturing," should not be coded. The sinus arrest is suggestive of sick sinus syndrome.

Codes:

11	Sinus pause or arrest
20	AV junctional premature complexes
21	AV junctional escape complexes
89	Sick sinus syndrome
92	Dual-chamber pacemaker (DDD)
94	Pacemaker malfunction, not constantly sensing (atrium or ventricle)



Questions: ECG 17

1. Abnormal sensing by a ventricular pacemaker is diagnosed when:

   a. A pacemaker stimulus does not result in appropriate capture
   b. The ventricular pacemaker fails to be inhibited by a QRS complex falling in an appropriate range
   c. A ventricular premature complex falls within the programmed refractory period of the pacemaker
   d. A pacemaker stimulus occurs within the QRS complex

Answers: ECG 17

1. Pacemaker sensing malfunction can involve the atrium and/or ventricle. For a pacemaker in the "inhibited" mode (e.g., VVI), failure to sense manifests as failure of the pacemaker to be inhibited by an appropriate intrinsic depolarization, such as a native QRS. For a pacemaker in the "triggered" mode (e.g., DDD), failure to sense manifests as failure of the pacemaker to trigger appropriately following a native event, such as a P wave. Pacemaker spikes falling within the QRS complex generally do not represent sensing malfunction. Failure to sense results in asynchronous firing of the pacemaker, resulting in a paced rhythm that competes with the intrinsic rhythm. Causes of failure to sense include low amplitude signals (especially VPCs), inappropriate programming of the sensitivity, and all causes of failure to capture. Failure to sense can often be corrected by reprogramming the sensitivity of the pacemaker. (Answer: b)

— Quick Review 17 —

Sinus pause or arrest	
• PP interval > _____ seconds	1.6-2.0
• Resumption of sinus rhythm at a PP interval that (is/is not) a multiple of the basic sinus PP interval	is not
• If sinus rhythm resumes at a multiple of the basic PP, consider _____	sinoatrial exit block
AV sequential pacing	
• Atrial followed by ____ pacing	ventricular
Pacemaker malfunction, not constantly sensing (atrium or ventricle)	
• Pacemakers in the inhibited mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	inhibited
• Pacemakers in the triggered mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	triggered
• Premature depolarizations may not be sensed if they fall within the programmed ____ period of the pacemaker, *or* have insufficient ____ at the sensing electrode site	refractory amplitude

Common Dilemmas in ECG Interpretation

Problem

A dominant junctional or ventricular rhythm is present. Is it necessary to code the underlying atrial rhythm if one is present?

Recommendation

Yes. If an atrial rhythm is present in addition to a dominant junctional or ventricular rhythm, the atrial rhythm (and AV block, if present) should also be coded (e.g., ventricular tachycardia and sinus rhythm with third-degree AV block).

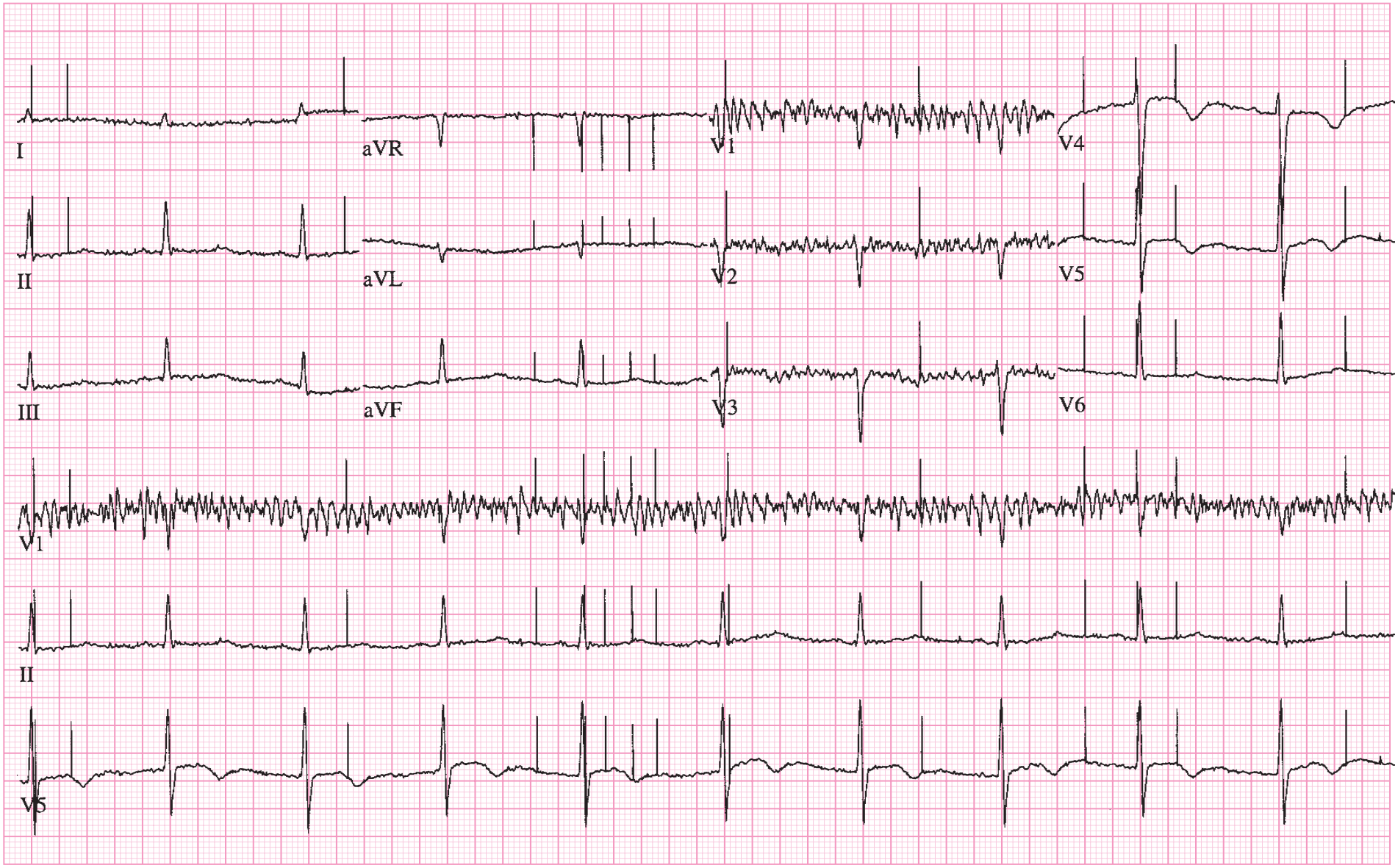
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the clinical disorder that best corresponds to each group of ECG features listed below (see items 70-89 of answer sheet for options)

ECG Features	Diagnosis
• Right atrial abnormality is common • Majority have abnormal QRS complexes: ▸ Large amplitude QRS ▸ Large abnormal Q waves (can give pseudoinfarct pattern in inferior, lateral, and anterior precordial leads) ▸ Tall R wave with inverted T wave in V_1 simulating RVH ▸ Nonspecific ST and/or T wave abnormalities common ▸ Left axis deviation in 20%	Hypertrophic cardiomyopathy
• Low voltage QRS • Electrical alternans and other features of acute pericarditis may be present	Pericardial effusion
• Sinus tachycardia and findings consistent with right ventricular pressure overload: ▸ Right atrial abnormality ▸ Inverted T waves in leads V_1-V_3 ▸ Right axis deviation ▸ S_1Q_3 or S_1Q_3 T_3 pattern ▸ Pseudoinfarct pattern in the inferior leads ▸ Incomplete or complete RBBB ▸ Supraventricular tachyarrhythmias are common • ECG abnormalities are often transient	Acute cor pulmonale, incl. pulmonary embolus

ECG 18. 59-year-old male with shortness of breath:



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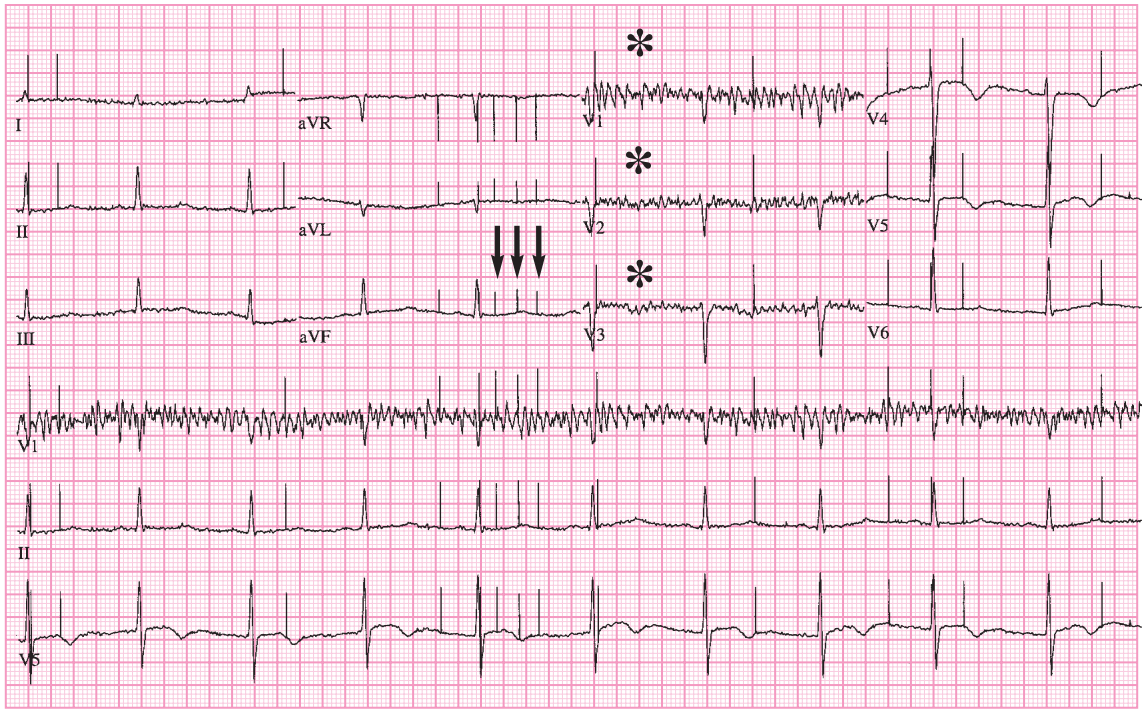
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 18 was obtained from a 59-year-old male with shortness of breath. The ECG shows sinus rhythm at a rate of 66 beats/minute. A number of spikes appear throughout the tracing, raising the possibility of pacemaker malfunction with failure to sense and capture. However, there is no consistent pattern to the spikes, and the multiple spikes that occur after the 5th QRS complex (arrows) are not consistent with any standard pacemaker/ICD pattern. In addition, there are coarse, almost fibrillatory-like oscillations in leads V_1-V_3 (asterisks), corresponding to an atrial rate in excess of 800 beats/minute, much too fast for atrial fibrillation or flutter. The multiple spikes and coarse, irregular oscillations represent baseline artifact (the patient did not have a pacemaker or any other implanted device). Other ECG abnormalities include ST and T wave changes in leads V_4-V_5 concerning for myocardial ischemia and a long QT interval.

Codes:

04	Artifact
07	Sinus rhythm
64	ST and/or T wave abnormalities suggesting myocardial ischemia
68	Prolonged QT interval



Questions: ECG 18

1. Baseline artifact should be suspected when:

   a. There is a regular rhythm in which some leads suggest a very rapid variant of atrial fibrillation other leads clearly show sinus rhythm
   b. There are pacemaker-like spikes with behavior that is not consistent with normal (or even abnormal) pacemaker function
   c. There are runs of wide-complex tachycardia superimposed on a background of narrow complex beats that appear to regularly march through the tracing (from before, until after the wide complex beats)

2. ECG baseline artifact can mimic:

   a. Premature ventricular complexes
   b. Ventricular tachycardia
   c. Ventricular fibrillation
   d. Atrial flutter
   e. Atrial fibrillation

Answers: ECG 18

1. Each situation described above represents an example in which baseline artifact can resemble abnormal heart rhythms. (Answer: all)

2. Baseline artifact can mimic any of the arrhythmias above. Causes of baseline artifact include AC electrical interference (60 cycles per second), tremor (Parkinson's or physiologic), rapid arm motion, skeletal muscle fasciculations (e.g., shivering), electrocautery, and IV infusion pump. (Answer: all)

— Quick Review 18 —

ST and/or T wave abnormalities suggesting myocardial ischemia	
• Abnormally tall, symmetrical, (upright/inverted) T waves	inverted
• Horizontal or ____ ST segments with or without T wave inversion	downsloping
• Associated ECG findings:	
▸ QT interval is usually (normal/prolonged)	prolonged
▸ Reciprocal ____ wave changes may be evident	T
▸ Prominent U waves are often present and may be upright or inverted (true/false)	true
Prolonged QT interval	
• Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval	0.42-0.46 RR
• QT interval varies (directly/inversely) with heart rate	inversely
• The normal QT interval should be (less than/greater than) 50% of the RR interval when the ventricular rate is between 65-90.	less than

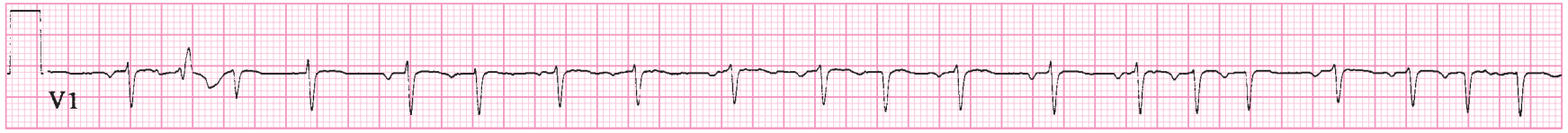
— POP QUIZ —

Find The Imposter

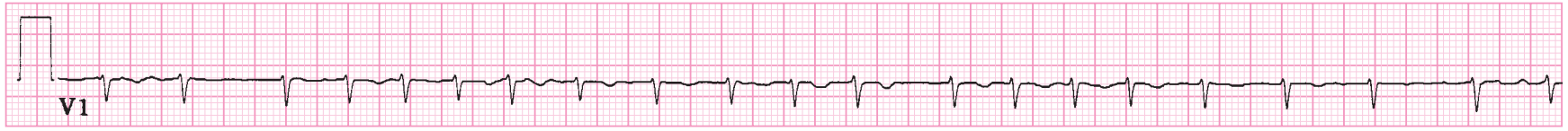
Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

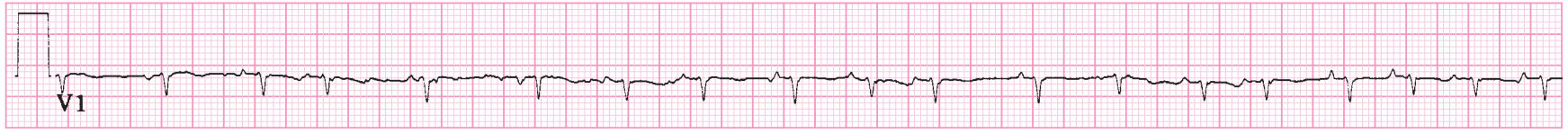
B.



C.



D.



Answer: Tracings A, B, and D show multifocal atrial tachycardia manifest as a narrow complex tachycardia preceded by P waves exhibiting 3 or more different morphologies. Tracing C shows atrial fibrillation with a rapid ventricular response and is the imposter.

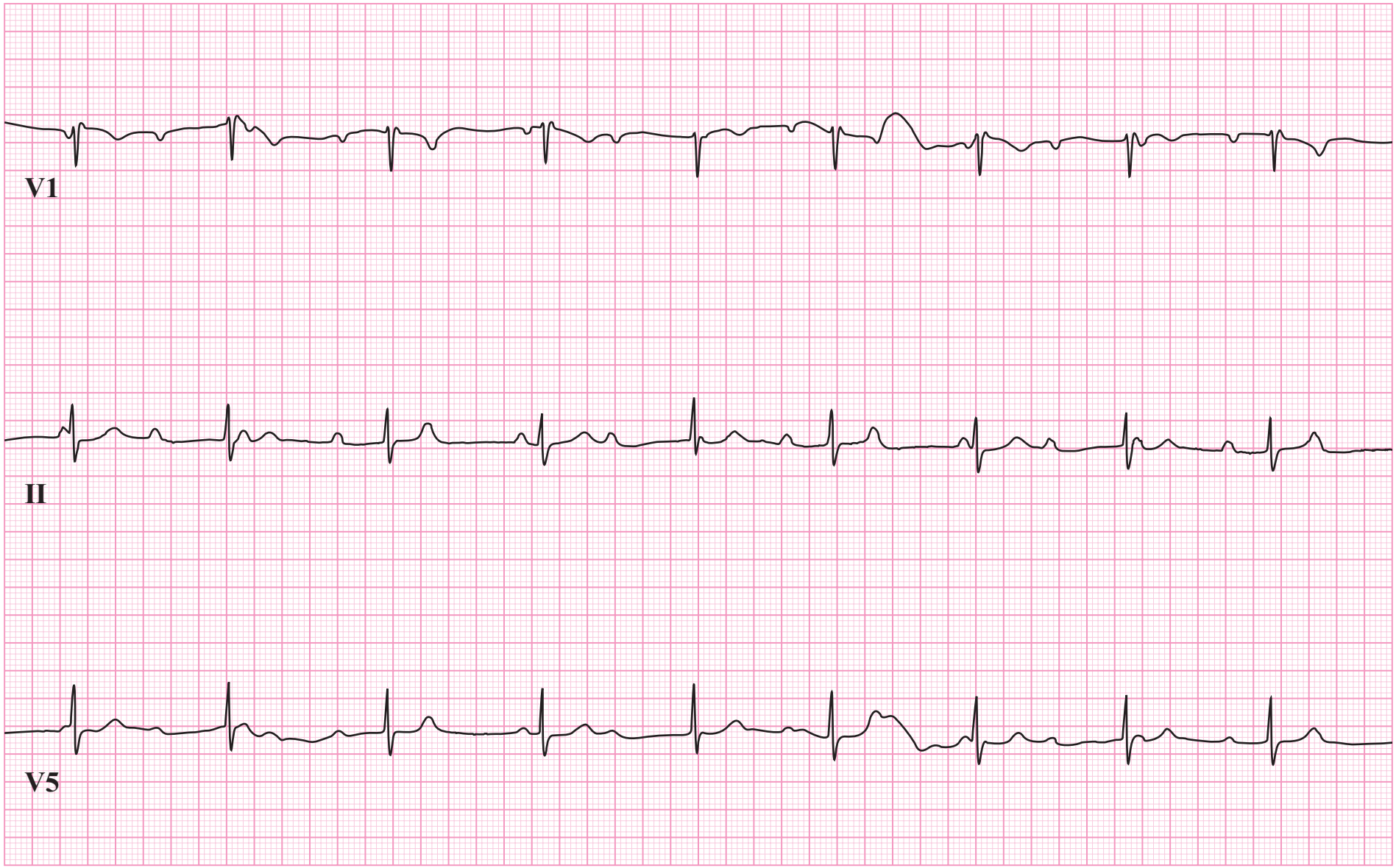
— POP QUIZ —

Find The Mistake

Instructions: Identify the incorrect ECG feature(s) for each of the ECG diagnoses listed below.

ECG Features	Mistake
Wolff-Parkinson-White pattern • Non-sinus P wave • PR interval < 0.12 seconds • Initial slurring of QRS (delta wave) resulting in QRS duration > 0.10 seconds • Secondary ST-T wave changes • PJ interval (beginning of P wave to end of QRS) varies	Sinus P wave is present, and PJ interval is constant (not variable)
2° AV block, Mobitz Type I (Wenkebach) • Progressive prolongation of the PR and RR intervals until a P wave is blocked • RR interval containing the nonconducted P wave is less than the sum of two PP intervals • Results in group beating due to the presence of nonconducted P waves	Progressive shortening (not prolongation) of the RR interval occurs until a P wave is blocked
2° AV block, Mobitz Type II • Regular sinus or atrial rhythm with intermittent nonconducted P waves without evidence for atrial prematurity • PR interval in the conducted beats is constant • RR interval containing the nonconducted P wave is less than or equal to the sum of two PP intervals	RR interval containing nonconducted P wave is equal to (not ≤) two PP intervals

ECG 19. 24-year-old asymptomatic female (rhythm strip):



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P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
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- ☐ 30. AV block, 2°-Mobitz type I (Wenckebach)
- ☐ 31. AV block , 2°-Mobitz type II
- ☐ 32. AV block, 2:1
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- ☐ 34. Wolff-Parkinson-White pattern
- ☐ 35. AV dissociation

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
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Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
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- ☐ 59. Posterior (age recent or acute)
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- ☐ 61. Normal variant, early repolarization
- ☐ 62. Normal variant, juvenile T waves
- ☐ 63. Nonspecific ST and/or T wave abnormalities
- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
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- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

SUGGESTED CLINICAL DISORDERS

- ☐ 70. Digitalis effect
- ☐ 71. Digitalis toxicity
- ☐ 72. Antiarrhythmic drug effect
- ☐ 73. Antiarrhythmic drug toxicity
- ☐ 74. Hyperkalemia
- ☐ 75. Hypokalemia
- ☐ 76. Hypercalcemia
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- ☐ 78. Atrial septal defect, secundum
- ☐ 79. Atrial septal defect, primum
- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
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- ☐ 83. Pericardial effusion
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- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

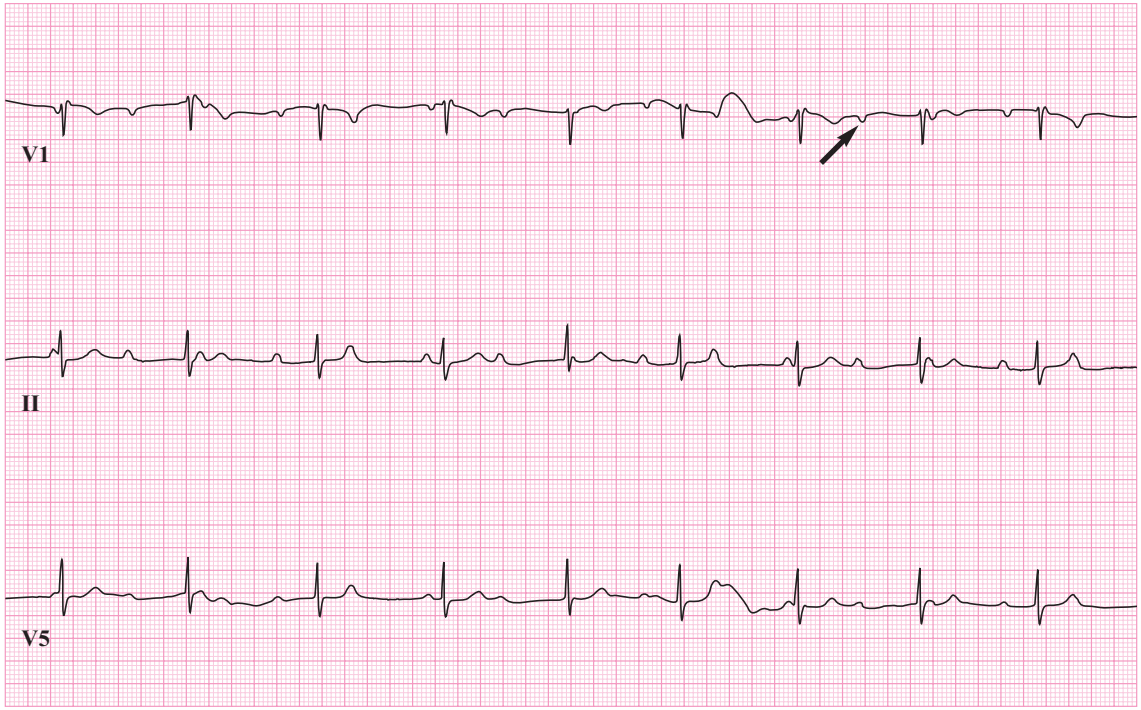
PACED RHYTHMS

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

ECG 19 is a rhythm strip obtained from an asymptomatic 24-year-old female. The ECG shows atrial and ventricular rhythms that are independent of each other (the sinus rate is 99 beats/minute and the ventricular rate is 60 beats/minute). The narrow QRS complexes at a rate of 60 beats/minute suggest a junctional escape rhythm in the setting of complete heart block. Left atrial enlargement (large inverted P wave in V_1) is also present. Because this tracing is a rhythm strip only, a full analysis for QRS axis, presence of MI, etc., is not possible. This woman was diagnosed with congenital complete heart block. In contrast to complete heart block from ischemia or cardiomyopathy, which typically occurs within the His-Purinje system and results in a wide QRS complex (ventricular escape) rhythm, congenital complete heart block usually occurs at the level of the AV node and results in a narrow QRS complex (junctional escape) rhythm.

Codes:

06	Left atrial abnormality/enlargement
07	Sinus rhythm
21	Junctional escape complexes
33	AV block, 3°



Questions: ECG 19

1. Which statement best describes the PP and RR intervals in complete heart block:

   a. Constant PP and RR intervals
   b. Variable PP and RR intervals
   c. Constant PP and variable RR intervals
   d. Variable PP and constant RR intervals

2. The typical heart rate of a ventricular escape rhythm is ___ beats/minute:

   a. 10-20
   b. 20-30
   c. 30-40
   d. 40-50

3. In an AV junctional escape rhythm, which of the following statements about the P wave are true:

   a. P wave can be buried in the QRS
   b. P wave can precede the QRS
   c. P wave can follow the QRS

Answers: ECG 19

1. Although the P waves and QRS complexes are independent of each other in complete heart block, PP and RR intervals are fairly constant (and the atrial rate is usually faster than the ventricular rate). The heart rate in complete heart block is usually maintained by either a junctional escape rhythm (narrow QRS complex), as in the current ECG, or a ventricular escape rhythm (wide QRS complex). (Answer: a)

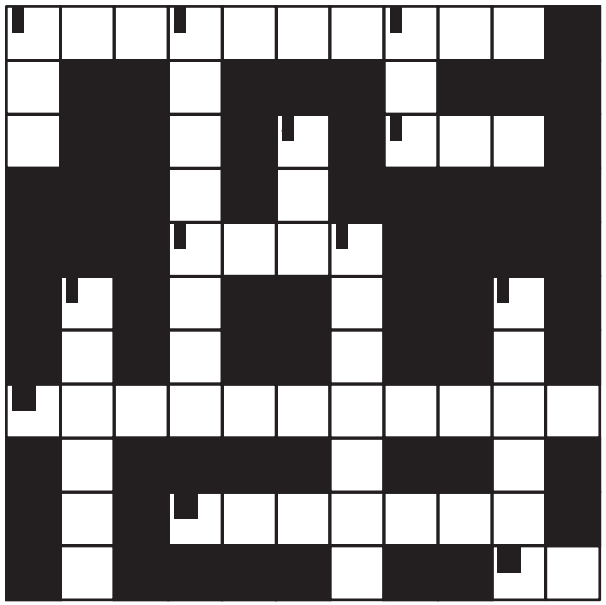
2. The rate of a ventricular escape rhythm is typically 30-40 beats/minute, but can vary from 20-50 beats/minute. The QRS duration is prolonged (> 0.12 sec), and QRS morphology is similar to that of ventricular premature contractions. (Answer: c)

3. The P wave in the junctional escape rhythm tends to be in close proximity to the QRS complex, and may precede it (PR < 0.11 seconds), be buried in it, or follow it. Junctional rhythms usually display a narrow QRS morphology, similar to a sinus or supraventricular impulse. Junctional escape rhythms can occur in the presence of P waves (high-degree AV block) or in the absence of P waves (sinus arrest). The typical rate of a junctional rhythm is 40-60 beats/minute. (Answer: all)

— Quick Review 19 —

AV junctional escape complexes	
• QRS complex occurs as a ____ phenomenon in response to decreased sinus impulse formation or conduction, or high-degree AV block	secondary
• Rate is typically ____ per minute	40-60
• Atrial mechanism may be sinus rhythm, paroxysmal atrial tachycardia, atrial flutter, or atrial fibrillation (true/false)	true
• QRS morphology is (similar to/different from) the sinus or supraventricular impulse	similar to
AV block, 3°	
• Atrial and ventricular rhythms are ____ of each other	independent
• Atrial rate is (faster/slower) than the ventricular rate	faster

— ECG CROSSWORD PUZZLE —

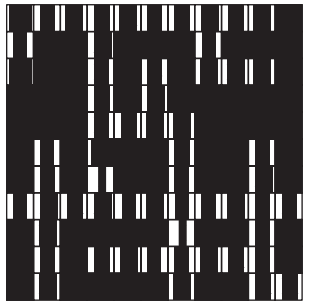


ACROSS

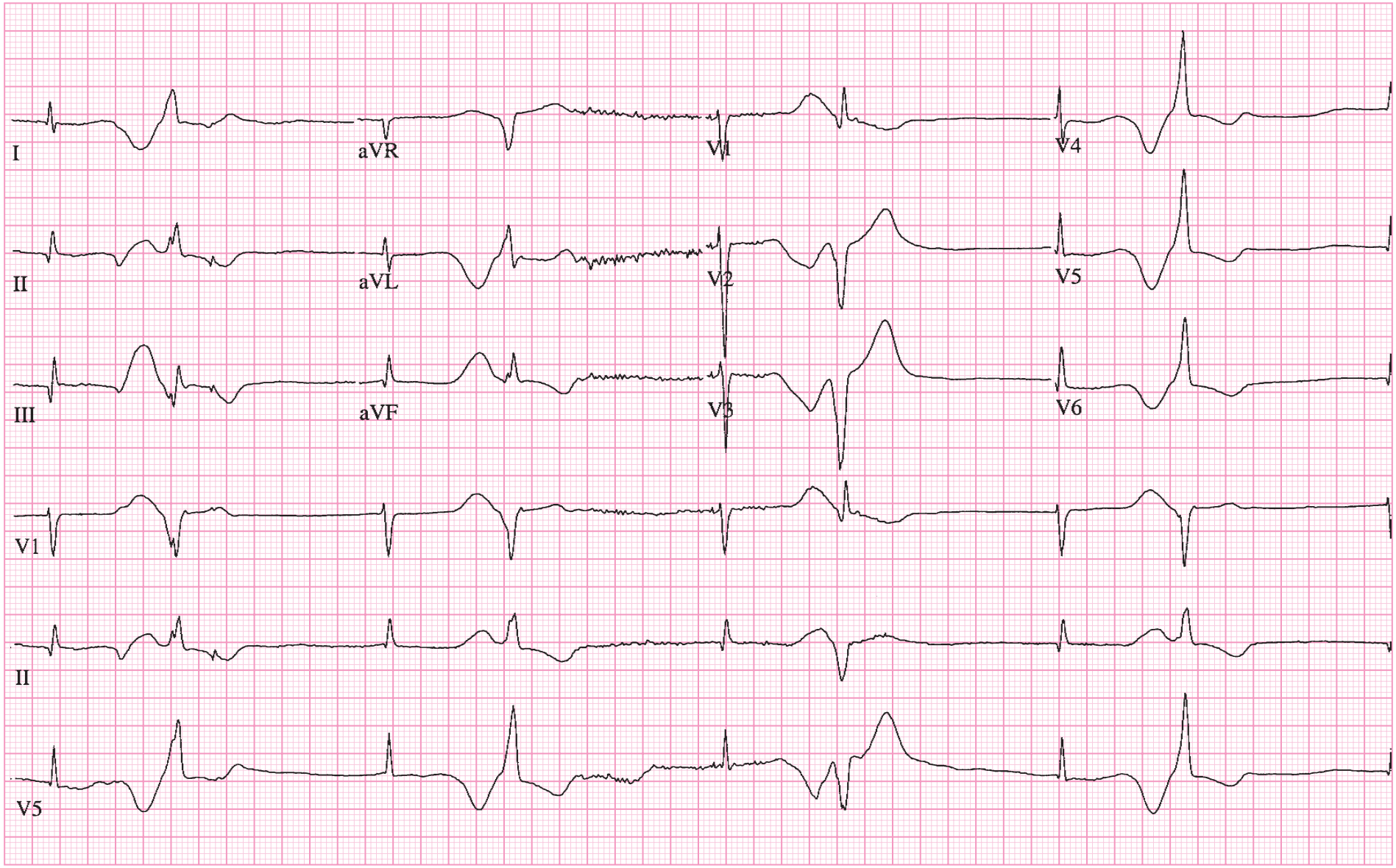
1. "Group beating" can result from this type of AV block
5. A type of AV sequential pacemaker
6. The type of complex that occurs when an impulse activates a chamber, returns to the site of origin, and reactivates the same chamber again
10. Associated with fusion beats and VPCs with nonfixed coupling
11. The type of complex that occurs when an atrial impulse stimulates the ventricle during VT
12. Multifocal atrial tachycardia is associated with varying PR, RP and _____ intervals

DOWN

1. Associated with atrial fibrillation or flutter with a QRS that varies in width (generally wide) and a ventricular response rate > 200 per minute
2. Hypo_____ results in a prolonged QT interval due to ST segment prolongation
3. Associated with an RSR' complex in V_1, left or right axis deviation, and a murmur
4. R wave in aVL > 12 mm
7. Can cause left axis deviation and low voltage
8. _____ T waves can be caused by hyperkalemia, acute MI, intracranial bleeding
9. In the setting of a wide QRS tachycardia, the presence of an R' wave in V_1 that is _____ than the R wave is suggestive of SVT with aberrancy



ECG 20. 62-year-old male with history of treated atrial fibrillation and recent weakness:



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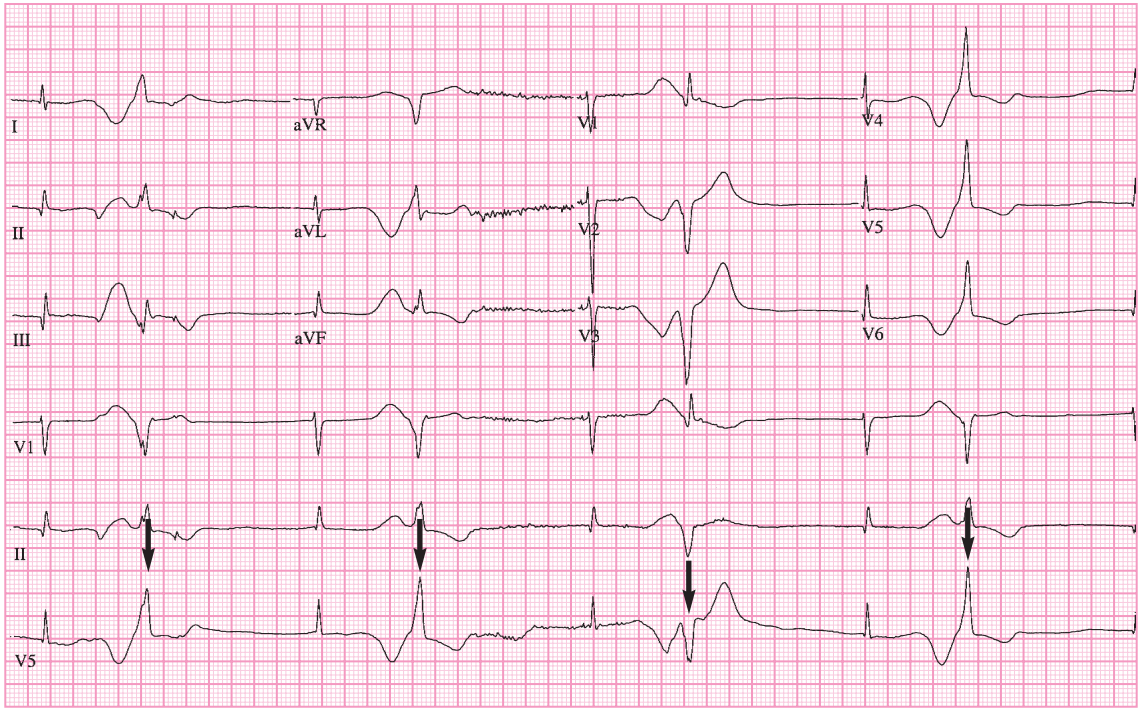
ECG 20 was obtained from a 62-year-old male with a history of treated atrial fibrillation and recent weakness. The rhythm is junctional with occasional ventricular premature complexes (in a bigeminal pattern at a fixed coupling interval) (arrows) and sinus pauses. There is also marked prolongation of the QT interval (QTc interval = 660 msec). Small q waves are present in the inferior leads but do not meet criteria for myocardial infarction. Given the patient's history of treated atrial fibrillation (he is currently on sotalol), the ECG is consistent with antiarrhythmic drug toxicity.

Codes:

11	Sinus pause or arrest
22	AV junctional rhythm/tachycardia
23	Ventricular premature complexes
64	ST and/or T wave abnormalities suggesting myocardial ischemia
68	Prolonged QT interval
73	Antiarrhythmic drug toxicity



Questions: ECG 20

1. Conditions associated with a prolonged QT interval include:

   a. Hyperkalemia
   b. Hypercalcemia
   c. Beta blockers
   d. Myocarditis
   e. Febrile illness

2. Which of the following statements about the QT interval is true:

   a. Prolongs as heart rate slows
   b. Shortens as heart rate slows
   c. Shorter when asleep than awake
   d. Shortens in the beat following a premature ventricular complex

Answers: ECG 20

1. The QT interval represents the time for ventricular depolarization and repolarization to occur. Causes of a prolonged QT interval include low serum magnesium or calcium levels, myocarditis, mitral valve prolapse, hypothyroidism, and hypothermia. Shortening of the QT interval occurs with beta blockers, digitalis, hyperkalemia, hypercalcemia, hyperthyroidism, and hypothermia. (Answer: d)

2. The QT interval varies inversely with heart rate, and lengthens during sleep and in the beat following a ventricular premature complex. (Answer: a)

— Quick Review 20 —

AV junctional rhythm	
• Rate ≤ ____ per minute	60
• QRS complex may be narrow or aberrant (true/false)	true
• Inverted P waves in leads ____ and upright P waves in leads ____ are common	II, III, aVF I, aVL
• RR interval of escape rhythm is usually (constant/variable)	constant
Prolonged QT interval	
• Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval	0.42-0.46 RR
• QT interval varies (directly/inversely) with heart rate	inversely
• The normal QT interval should be (less than/greater than) 50% of the RR interval	less than
Antiarrhythmic drug toxicity	
• Widening of the ____ complex and ____ interval	QRS, QT
• Various degrees of ____ block	AV

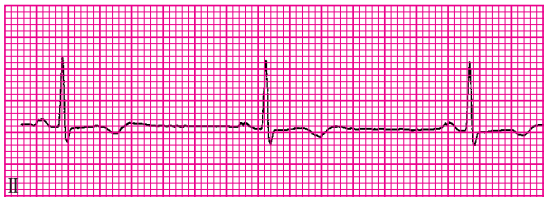
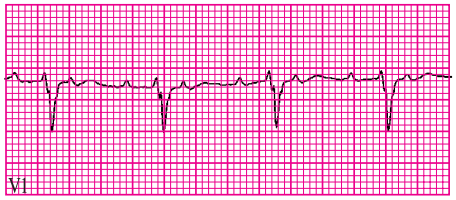
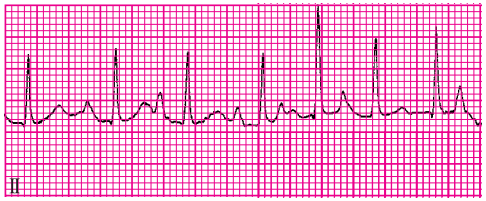
Differential Diagnosis

"PSEUDOINFARCTS" (ECG pattern can mimic Q-wave myocardial infarction)	
• Wolff-Parkinson-White pattern • Hypertrophic cardiomyopathy • Left ventricular hypertrophy • Right ventricular hypertrophy • Left anterior fascicular block • Chronic lung disease • Amyloid heart (or other infiltrative diseases) • Cardiomyopathy • Chest deformity (e.g., pectus excavatum) • Pulmonary embolism • Myocarditis • Myocardial tumors • Hyperkalemia	• Lead reversal • Corrected transposition • Dextrocardia • Left bundle branch block • Pancreatitis • Muscular dystrophy • Mitral valve prolapse • Myocardial contusion • Left/right atrial enlargement: Prominent atrial repolarization wave (Ta) can depress the PR segment to mimic a Q wave • Pneumothorax

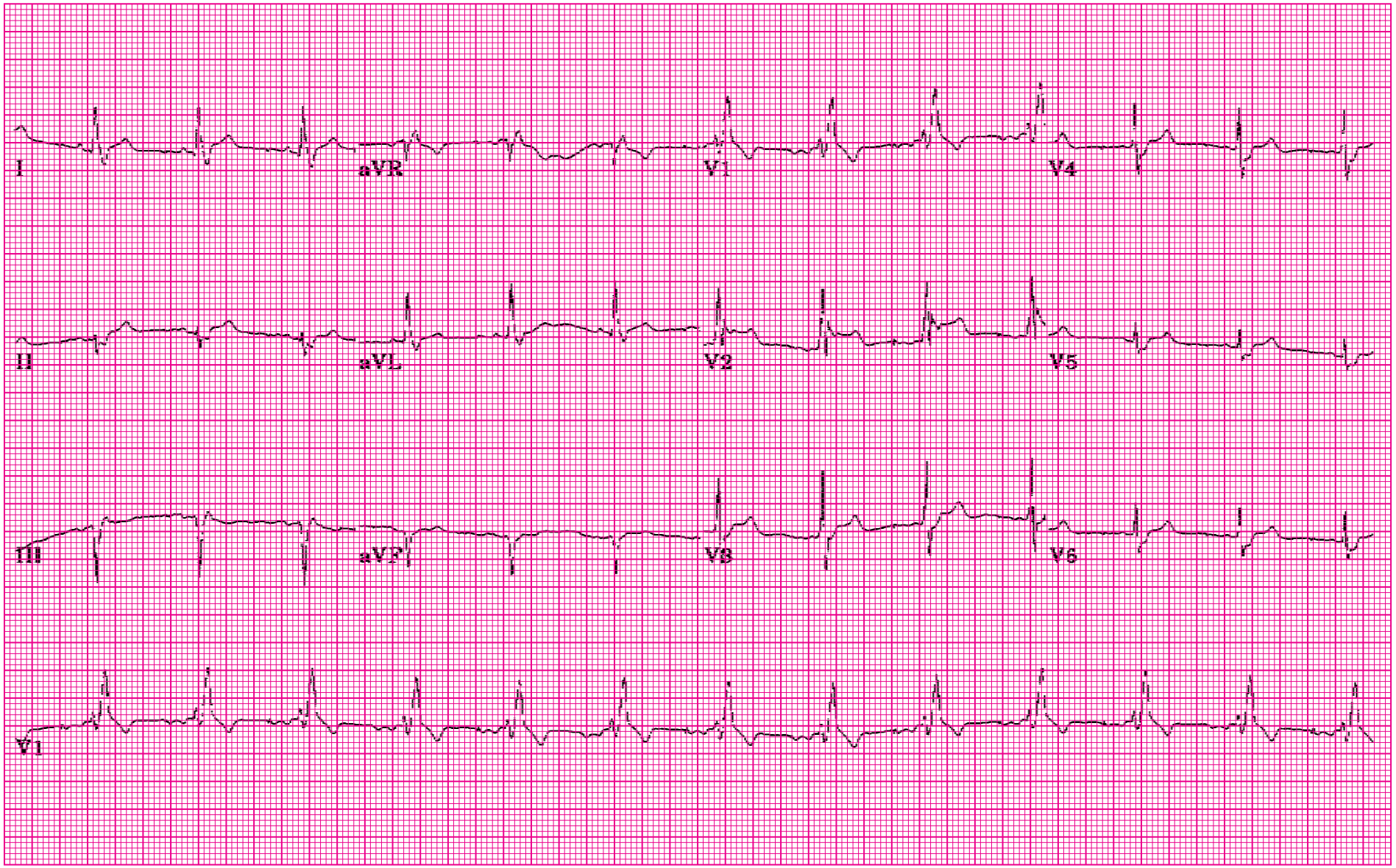
— POP QUIZ —

Pattern Recognition: Drug Effects and Rhythm Disturbances

Instructions: Choose all drugs commonly associated with each of the following rhythm abnormalities.

ECG	Choose All That Apply	Answer
	a. Amiodarone b. Atropine c. Aminophylline d. Digitalis e. Atorvastatin f. Ramipril g. Nitroglycerin h. Metoprolol i. Verapamil	*Sinus bradycardia* results in a regular sinus (upright P waves in lead II) rhythm at a rate < 60 per minute. Common causes include beta-blockers, amiodarone, verapamil, diltiazem, digitalis, Type I antiarrhythmics, clonidine, alpha-methyldopa, reserpine, guanethidine, cimetidine, and lithium. Low-dose atropine may also cause a paradoxical slowing of heart rate. (Answer: a, b [low dose], d, h, i)
		*Paroxysmal atrial tachycardia (PAT) with block* results in nonsinus P waves at a regular atrial rate (usually 150-240 per minute), isoelectric intervals between P waves, and some nonconducted P waves due to 2° AV block. Digoxin toxicity is responsible for 75% of cases and organic heart disease for 25% of cases. Atropine may worsen Type II 2° AV block, but rarely causes this arrhythmia. Note: 2:1 AV block in this ECG may be either Mobitz Type I or Type II. (Answer: d)
		*Multifocal atrial tachycardia* (MAT) results in an irregular atrial rate > 100 per minute with at least three different P wave morphologies (originating from separate atrial foci) and varying PP and PR intervals. MAT is usually associated with some form of lung disease (COPD, cor pulmonade, hypoxia), and can be precipitated by aminophylline. (Answer: c)

ECG 21. 73-year-old male with chest pain:



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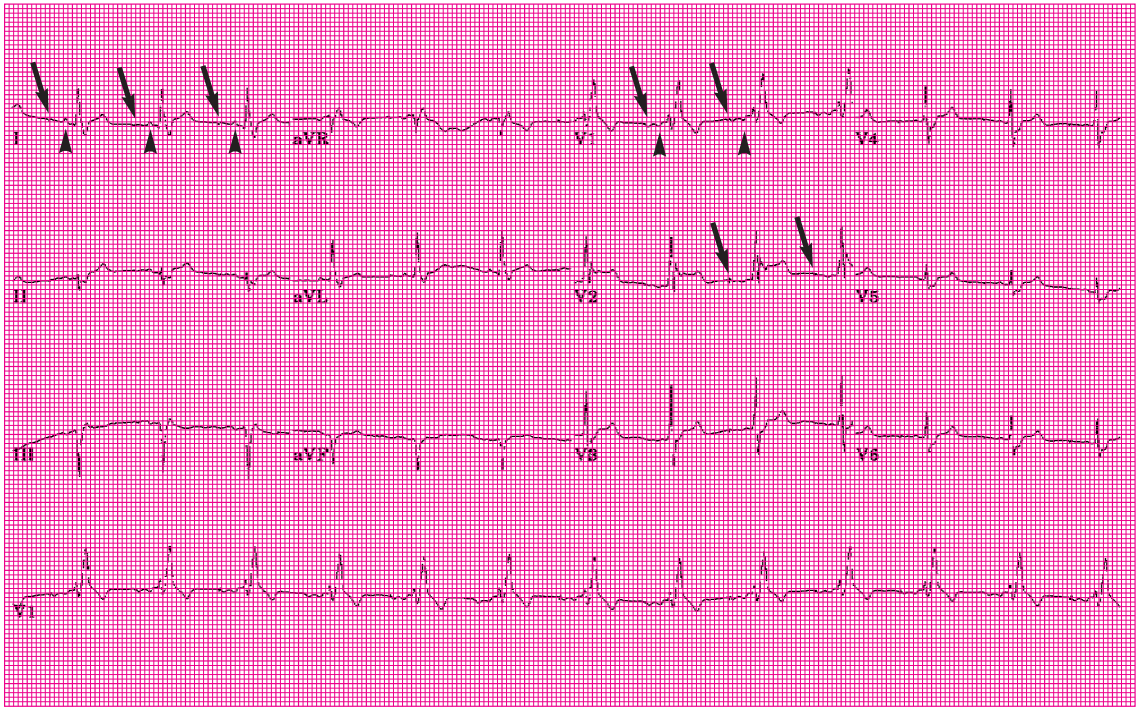
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 21 was obtained from a 73-year-old-male with chest pain. The ECG shows an atrial paced rhythm at a rate of 83 beats/minute (arrows mark atrial pacing spikes; arrowhead marks small atrial depolarization following pacer spike). About 0.21 seconds after each atrial pacemaker spike is a native QRS complex with RBBB morphology. Left anterior fascicular block is present (QRS axis = - 50°), and is responsible for the small R waves in leads II, III and aVF. The ST segment elevation and upright T waves in leads V_1-V_4 are consistent with acute myocardial injury.

Codes:

43	RBBB, complete
45	Left anterior fascicular block
65	ST and/or T wave abnormalities suggesting myocardial injury
90	Atrial or coronary sinus pacing



Questions: ECG 21

1. The presence of atrial pacemaker spikes on a 12-lead ECG without ventricular pacemaker activity rules out dual chamber (DDD) pacing:

   a. True
   b. False

2. The most common cause of ST segment elevation on the resting ECG is:

   a. Left ventricular hypertrophy
   b. Ventricular aneurysm
   c. Pericarditis
   d. Normal variant early repolarization
   e. Acute myocardial infarction

3. ST segment elevation up to ____ mm in leads V_2-V_4 can be considered part of the normal variant early repolarization pattern:

   a. 1 mm
   b. 2 mm
   c. 3 mm
   d. 4 mm

4. What ratio of ST elevation to T wave amplitude in lead V_6 is most helpful in distinguishing pericarditis from normal variant early repolarization:

   a. 15%
   b. 25%
   c. 50%
   d. 75%

Answers: ECG 21

1. In a person with a dual chamber (DDD) pacemaker, atrial pacemaker activity depends on native heart rate, and occurs whenever the native (intrinsic) heart rate falls below the programmed lower rate of the pacemaker. Ventricular pacemaker activity, on the other hand, depends on native AV conduction: If native AV conduction exceeds the programmed AV delay, atrial pacing spikes are followed by ventricular pacemaker activity. If native AV conduction is shorter than the programmed AV delay, ventricular pacemaker output is inhibited, resulting in atrial spikes followed by native QRS complexes. For examination purposes, atrial spikes without ventricular pacemaker activity should be coded for atrial pacing, not dual chamber pacing. (Answer: b).

2. Normal variant early repolarization manifests as concave upward ST segment elevation leading into a symmetrical, upright T wave, which may be relatively large in amplitude.

Some degree of early repolarization ST elevation is present in most of young, healthy individuals. (Answer: d)

3. Concave upward ST segment elevation up to 1 mm in the limb leads and 3 mm in the mid-precordial leads (V_2-V_4) can be seen in normal individuals. However, since ST elevation may be caused by myocardial injury or infarction, acute pericarditis, or ventricular aneurysm, it is important to consider the clinical context and associated ECG findings (e.g., abnormal Q waves, reciprocal ST segment depression) before interpreting ST elevation as "normal." (Answer: c)

4. Both acute pericarditis and early repolarization manifest diffuse, concave upward ST segment elevation. The ratio of ST elevation to T wave amplitude in lead V_6 helps distinguish between these conditions: ST elevation is usually >25% of T wave amplitude in pericarditis, and <25% of T wave amplitude in early repolarization. (Answer: b)

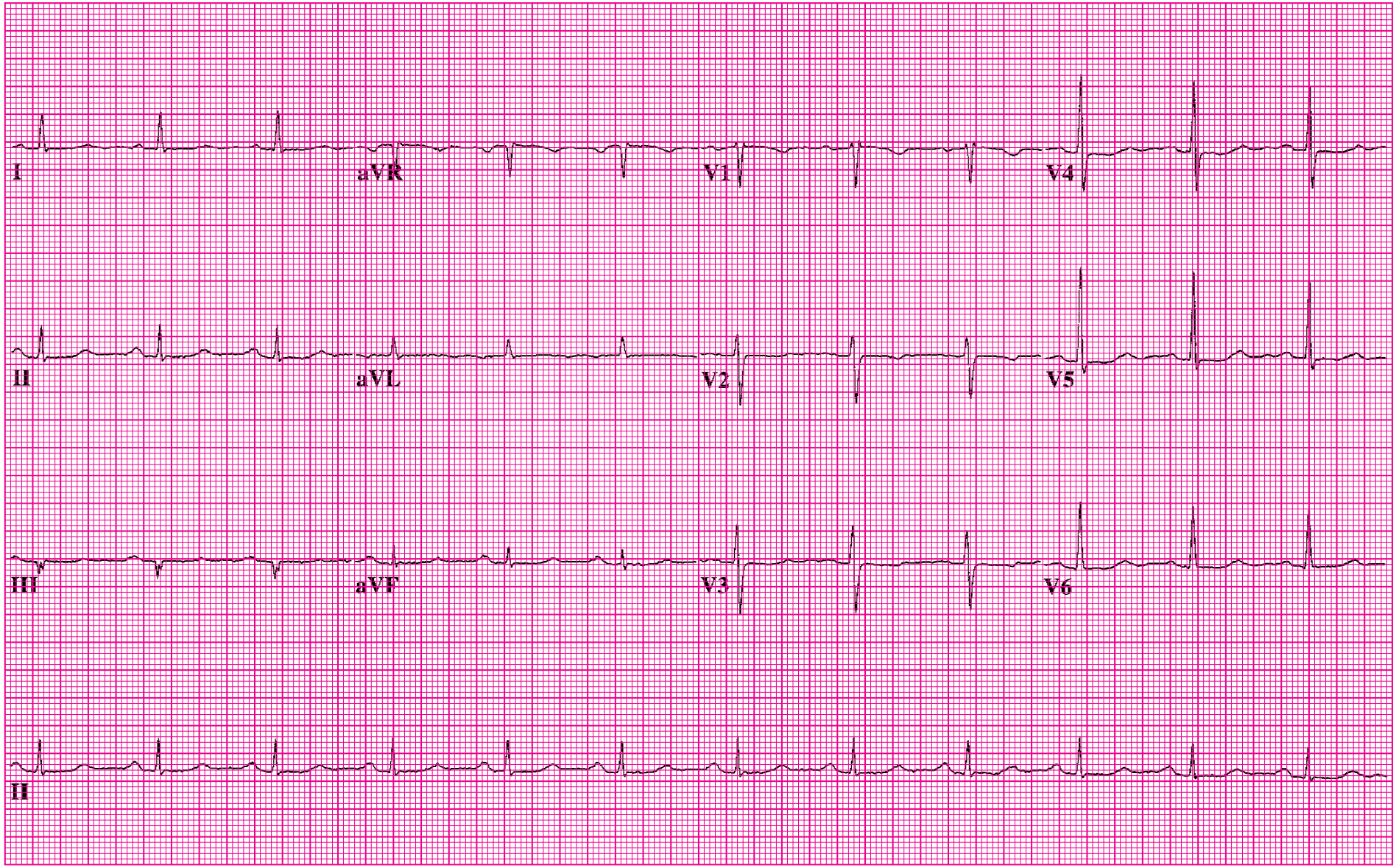
— Quick Review 21 —

RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ____ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 > ____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ____	I, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
• RBBB (does/does not) interfere with the ECG diagnosis of ventricular hypertrophy or Q wave MI	does not
Left anterior fascicular block	
• ____ axis deviation with a mean QRS axis between ____ and ____ degrees	left −45, −90
• (qR/rS) complex in leads I and aVL	qR
• (qR/rS) complex in lead III	rS
• Normal or slightly prolonged QRS duration (true/false)	true
• No other cause for left axis deviation should be present (true/false)	true
• Poor R wave progression is (common/uncommon)	common

Don't Forget!

- Classic evolutionary ECG pattern of acute pericarditis consists of 4 stages (but is not always present):
  - Stage 1: Upwardly concave ST segment elevation in almost all leads except aVR; no reciprocal ST depression in other leads except aVR
  - Stage 2: ST junction (J point) returns to baseline and T wave amplitude begins to decrease
  - Stage 3: T waves invert
  - Stage 4: ECG returns to normal
- Digitalis toxicity can cause almost any type of cardiac dysrhythmia or conduction disturbance except bundle branch block.
- ECG findings in CNS disease can mimic those of:
  - Acute MI
  - Acute pericarditis
  - Drug effect or toxicity

ECG 22. 80-year-old female with chronic renal failure:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
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- ☐ 30. AV block, 2°-Mobitz type I (Wenckebach)
- ☐ 31. AV block , 2°-Mobitz type II
- ☐ 32. AV block, 2:1
- ☐ 33. AV block, 3°
- ☐ 34. Wolff-Parkinson-White pattern
- ☐ 35. AV dissociation

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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
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- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
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- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

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- ☐ 90. Atrial or coronary sinus pacing
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- ☐ 92. Dual-chamber pacemaker (DDD)
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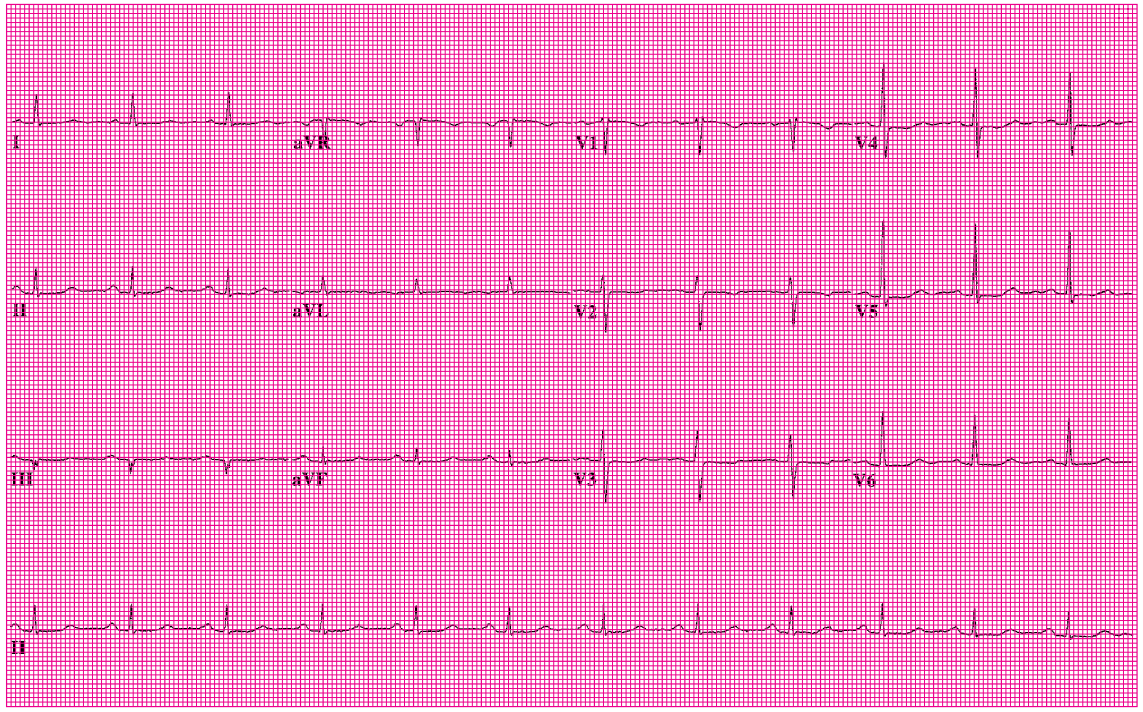
ECG 22 was obtained in 80-year-old female with chronic renal failure. The ECG shows sinus rhythm with first-degree AV block, nonspecific ST-T wave abnormalities, and QT interval prolongation (corrected QT interval measures 0.50 seconds). The long QT interval is primarily due to prolongation of the ST segment (rather than the T wave), which is characteristic of hypocalcemia. This patient was shown to have a serum calcium level of 6.8 mg/dL.

Codes:

07	Sinus rhythm
29	AV block, 1°
63	Nonspecific ST and/or T wave abnormalities
68	Prolonged QT interval
77	Hypocalcemia



Questions: ECG 22

1. Electrolyte abnormalities associated with a prolonged QT interval include:

   a. Hypocalcemia
   b. Hyperkalemia
   c. Hypokalemia
   d. Hypercalcemia
   e. Hypomagnesemia
   f. Hypermagnesemia

2. ECG abnormalities associated with hypocalcemia include all of the following except:

   a. QT prolongation due to ST segment prolongation
   b. Normal T wave duration
   c. Flattened, peaked, or inverted T waves
   d. Notching of the terminal QRS (Osborne wave)

Answers: ECG 22

1. When it comes to electrolyte disorders associated with a prolonged QT interval, think "hypo": hypokalemia, hypocalcemia, hypomagnesemia. (Answer: a, c, e)

2. Hypocalcemia prolongs the QT interval in a very characteristic way — by prolonging the ST segment, but not the T wave. The T wave can be mildly flattened, peaked, or inverted, but has a normal duration. Abnormal notching of the terminal QRS complex (Osborne wave) occurs in hypothermia, not hypocalcemia. (Answer: d)

— Quick Review 22 —

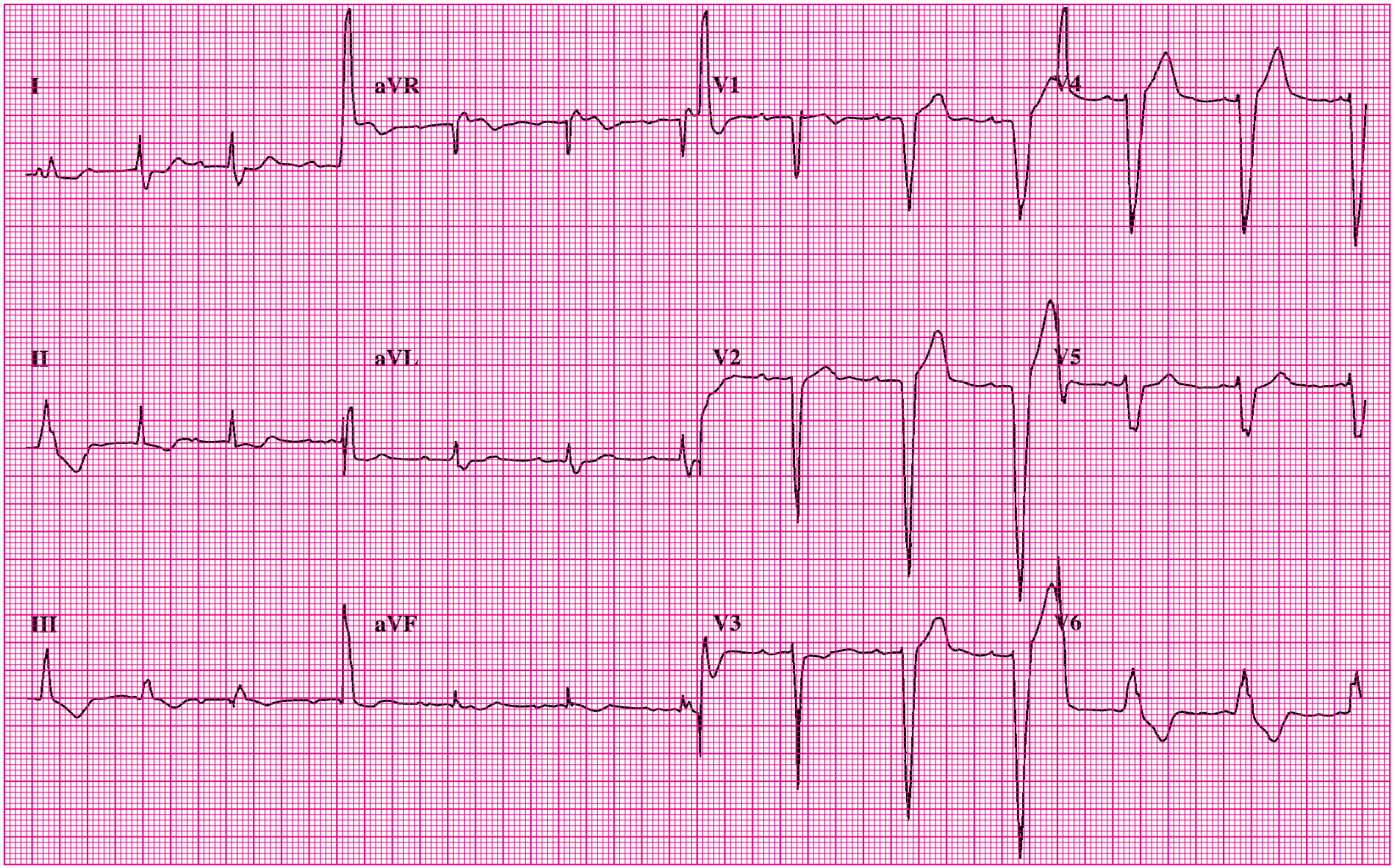
Prolonged QT interval

- Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval — 0.44; RR
- QT interval varies (directly/inversely) with heart rate — inversely
- The normal QT interval should be (less than/greater than) 50% of the RR interval — less than

Hypocalcemia

- Earliest and most common finding is prolonged ____ interval — QT
- Occasional flattening, peaking, or inversion of ____ waves — T

ECG 23. 76-year-old asymptomatic female:



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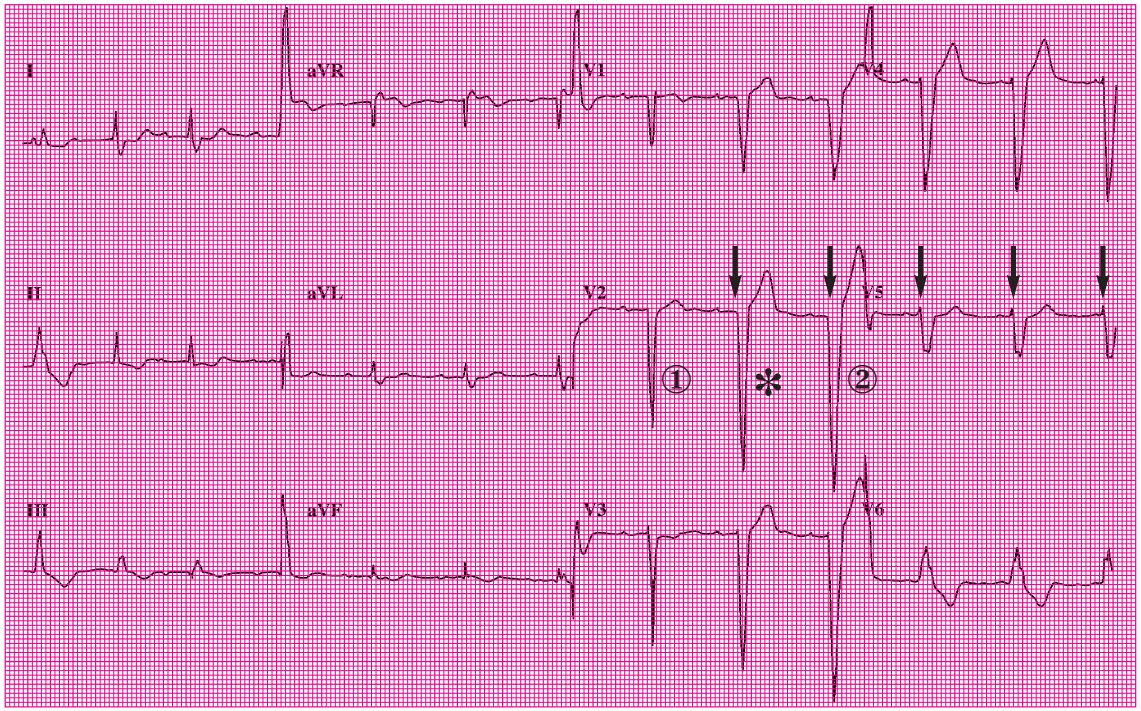
ECG 23 was obtained in a 76-year-old asymptomatic female. During the first half of the ECG, sinus rhythm with first-degree AV block and a nonspecific intraventricular conduction disturbance (QRS in sinus rhythm = 0.11 seconds) are present. This is overridden by an accelerated idioventricular rhythm (arrows), resulting in isorhythmic AV dissociation and fusion complexes (QRS with asterisk is intermediate in morphology between the QRS complexes labeled 1 and 2).

Codes:

07	Sinus rhythm
26	Accelerated idioventricular rhythm
29	AV block, 1°
35	AV dissociation
49	Nonspecific intraventricular conduction disturbance



Questions: ECG 23

1. AV dissociation is characterized by an atrial rate that is usually___ the ventricular rate:

   a. Faster than
   b. Slower than
   c. Equal to

2. By definition, the rate of an accelerated idioventricular rhythm is:

   a. < 30 bpm
   b. 60-110 bpm
   c. 40-60 bpm
   d. 30-50 bpm

Answers: ECG 23

1. AV dissociation occurs when the atrial and ventricular activities are independent of each other, and the atrial rate is ***slower*** than the ventricular rate. This generally occurs in the setting of extreme sinus bradycardia or normal sinus rhythm with a faster (escape or accelerated) junctional or idioventricular rhythm. (Answer: b)

2. Accelerated idioventricular rhythm is a regular rhythm with wide QRS complexes occurring at a rate of 60-110 BPM. AV dissociation, capture complexes, and fusion beats are common during AIVR because of the competition between normal sinus and ectopic ventricular rhythms. AIVR does not have the same adverse impact on prognosis that ventricular tachycardia does. (Answer: b)

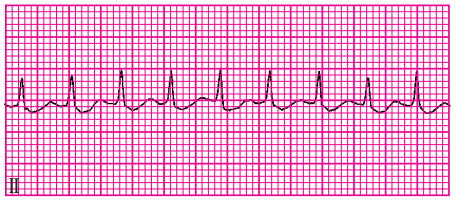
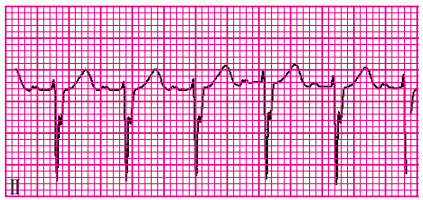
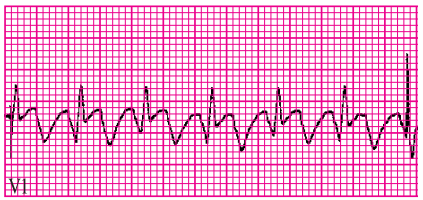
— Quick Review 23 —

Accelerated idioventricular rhythm	
• Highly irregular ventricular rhythm (true/false)	false
• Ventricular rate of ____ per minute	60-110
• QRS morphology is similar to ____	VPCs
• Ventricular ____ complexes, ____ beats, and AV ____ are common	capture, fusion dissociation
AV dissociation	
• Atrial and ventricular rhythms are ____ of each other	independent
• Ventricular rate is (</≥) than the atrial rate	≥

— POP QUIZ —

Rhythm Recognition: HR > 100; Narrow QRS; Regular RR Interval

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: AV node reentrant tachycardia (AVNRT). Description: Narrow complex SVT usually at a rate of 150-250 per minute. There is typically a P wave buried in or immediately following the QRS with a short RP interval (< 0.09 seconds), and an rSr' complex in lead V_1 that is not present during sinus rhythm. Reentry within the AV node occurs as a consequence of antegrade conduction down the slow (α) AV nodal pathway and retrograde conduction up the fast (β) AV nodal pathway. AVNRT is often initiated by an APC, and frequently slows or abruptly terminates in response to carotid sinus massage. AVNRT accounts for 60-70% of SVTs.
	Answer: Sinus tachycardia. Description: Regular sinus rhythm at a rate > 100 per minute. Causes include physiologic response to stress, anemia, fever, drugs (e.g., caffeine, ephedrine, alcohol, nicotine), thyrotoxicosis, myocardial ischemia/infarction, heart failure, myocarditis, hypoxemia, pulmonary embolism, pheochromocytoma, and AV fistula. Cannot be distinguished from sinus node reentrant tachycardia (which has sudden onset and termination) based on surface ECG alone.
	Answer: Atrial flutter. Description: Rapid, regular atrial undulations (flutter or "F" waves) usually at a rate of 240-340 per minute. Flutter waves are typically inverted in leads II, III and aVF, and manifest small positive upright deflections in V_1; "atypical flutter" can show upright F waves in the inferior leads. QRS complexes may be narrow or wide (if underlying aberrancy or bundle branch block). AV conduction ratio (ratio of flutter waves to QRS complexes) is usually a fixed, even number (e.g., 2:1, 4:1), but variable conduction sometimes occurs (e.g., 2:1 and 4:1 in the same tracing). Atrial flutter with 1:1 AV conduction often conducts aberrantly and may be confused with VT. In untreated patients, block ≥ 4:1 suggest coexistent AV conduction disease. Flutter waves sometime deform the QRS, ST, T waves to mimic intraventricular conduction delay or myocardial ischemia/injury.

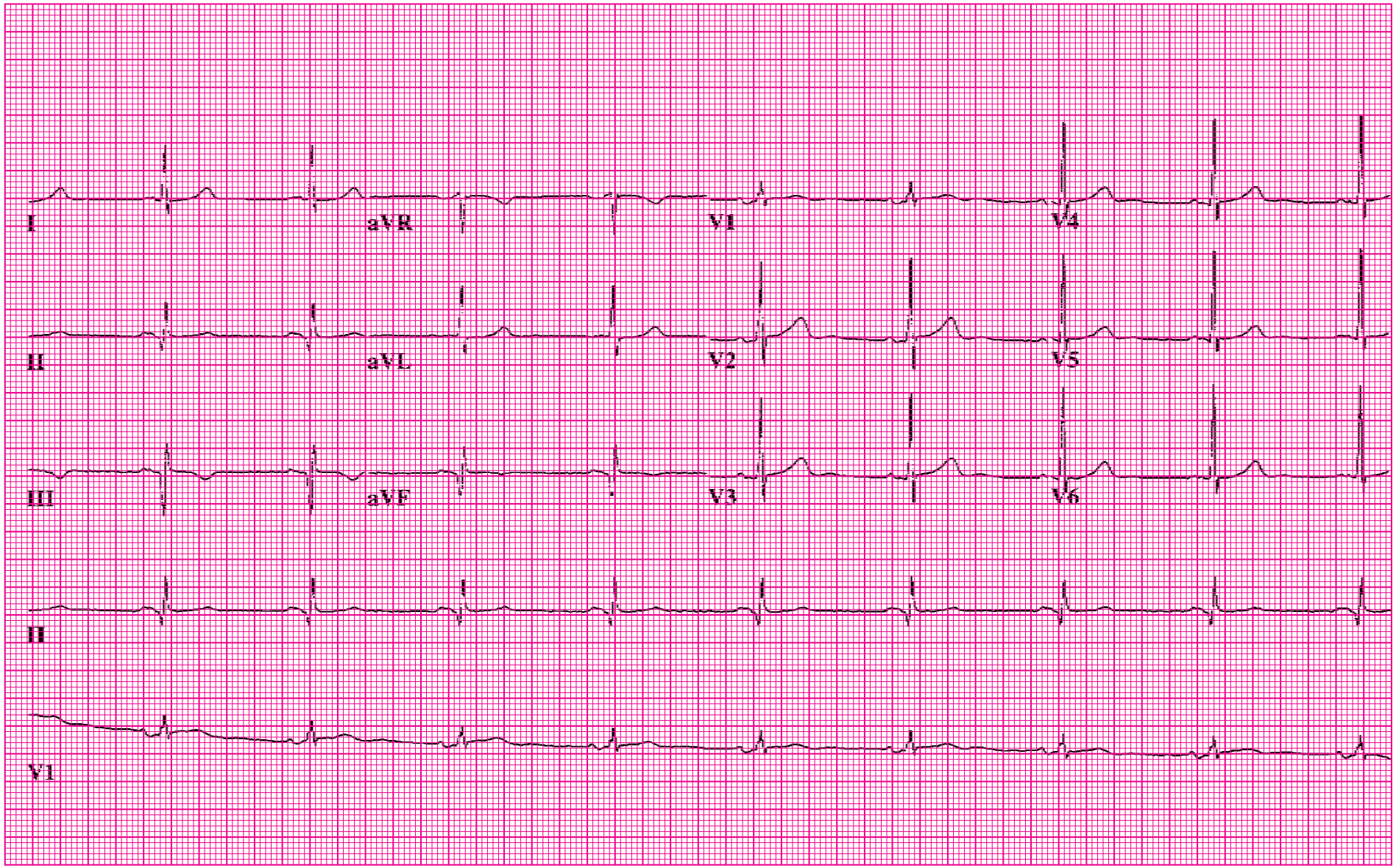
— POP QUIZ —

Differential Diagnosis: P-wave

Instructions: Match the P wave characteristic with all ECG diagnoses that apply.

P Wave Characteristic	Choose All That Apply	Answer
1. Inverted P-QRS-T in lead I; normal precordial R wave progression	a. Ectopic atrial rhythm b. Ventricular rhythm with retrograde atrial activation c. Dextrocardia d. Reversal of right and left arm leads e. Right atrial abnormality f. Left atrial abnormality g. Atrial flutter h. Physiologic tremor i. Multifocal atrial tachycardia j. Sinoventricular conduction 2° to hyperkalemia k. Junctional escape rhythm	1. Reversal of right and left arm leads. Other findings include transposition of leads II and III, and leads aVR and aVL. (Answer: d)
2. Inverted P-QRS-T in lead I; reverse precordial R wave progression		2. Dextrocardia. Normal precordial R wave progression suggests limb lead reversal. (Answer: c)
3. Tall peaked P wave in lead II		3. Right atrial abnormality. (Answer: e)
4. Bifid P wave in lead II with peak-to-peak interval > 0.03 seconds		4. Left atrial abnormality. Bifid P wave in lead II with peak-to-peak interval < 0.03 seconds is a normal variant. (Answer: f)
5. Sawtooth regular P waves at a rate of 300 per minute		5. Atrial flutter. Physiologic tremor occurs at a rate of 5-7 cycles/sec (~ 500 per minute). Parkinson's tremor occurs at a rate of 4-6 cycles/sec (~ 300 per minute). IV infusion pump changes can also mimic P waves. (Answer: g)
6. Multiple P wave morphologies		6. Multiple P wave morphologies can be seen in multifocal atrial tachycardia or sinus/atrial rhythm with multiple APCs. (Answer: i)
7. Tall upright P wave in V_1		7. Right atrial abnormality. (Answer: e)
8. Deeply inverted P wave in V_1 only		8. Left atrial abnormality. (Answer: f)
9. P waves present but hidden		9. Causes include ectopic atrial rhythm or APCs (P wave hidden in preceding T wave), junctional rhythm or SVT (P wave buried in QRS), or supraventricular rhythm with marked first-degree AV block (P wave hidden in preceding T wave). (Answer: a, k)
10. P waves absent		10. Causes include marked sinoatrial exit block, sinus arrest with junctional or ventricular rhythm (escape or accelerated), or sinoventricular conduction 2° to hyperkalemia. Atrial flutter presents with "F" waves, not P waves. (Answer: g, j)

ECG 24. 72-year-old asymptomatic male with previous myocardial infarction:



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- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

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

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

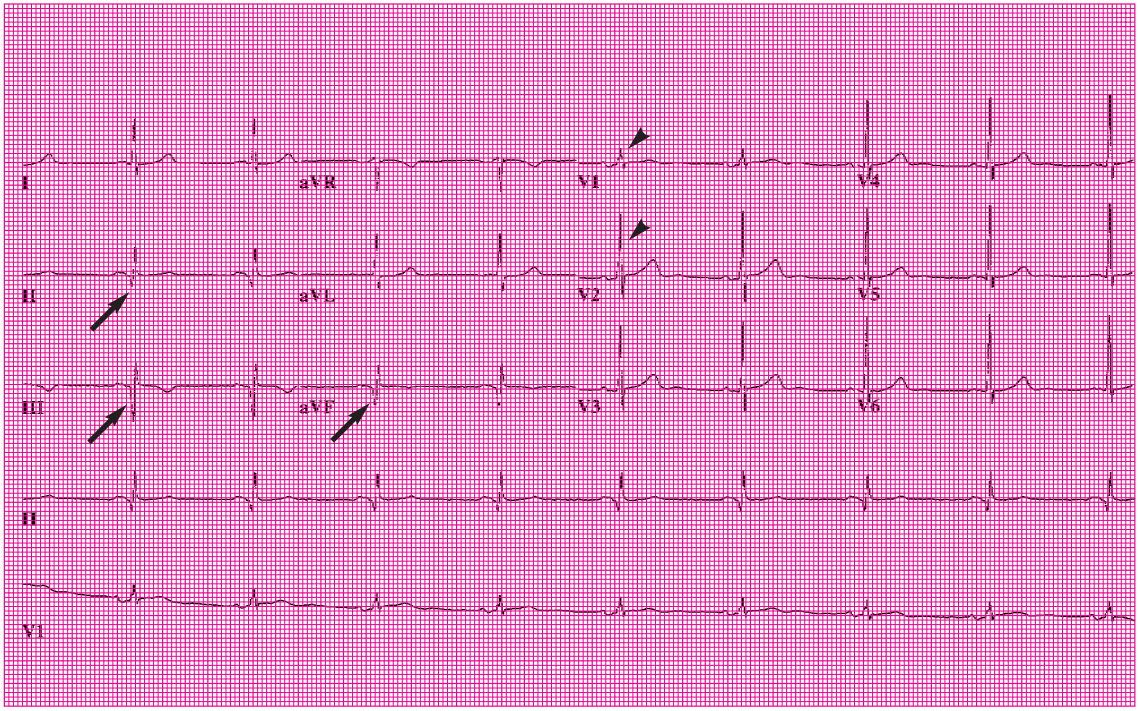
PACED RHYTHMS

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

ECG 24 was obtained in a 72-year-old asymptomatic male with a history of myocardial infarction. The ECG shows sinus bradycardia at 54 beats/minute. The abnormal Q waves in leads II, III, and aVF (arrows) and prominent R waves in leads V_1 and V_2 (arrowheads) are consistent with prior inferoposterior myocardial infarction. Nonspecific ST and T wave changes are present in the inferior leads.

Codes:

09	Sinus bradycardia (< 60)
58	Inferior Q wave MI (age indeterminate or old)
60	Posterior MI (age indeterminate or old)
63	Nonspecific ST and/or T wave abnormalities



Questions: ECG 24

1. Conditions that interfere with the diagnosis of posterior myocardial infarction on ECG include:

   a. Inferior MI
   b. Right ventricular hypertrophy
   c. Wolff-Parkinson-White syndrome
   d. Right bundle branch block

2. The ECG equivalent of a pathological Q wave in posterior myocardial infarction is:

   a. Deep S wave in V_1-V_2
   b. ST depression in V_1-V_2
   c. Tall R wave in V_1-V_2

Answers: ECG 24

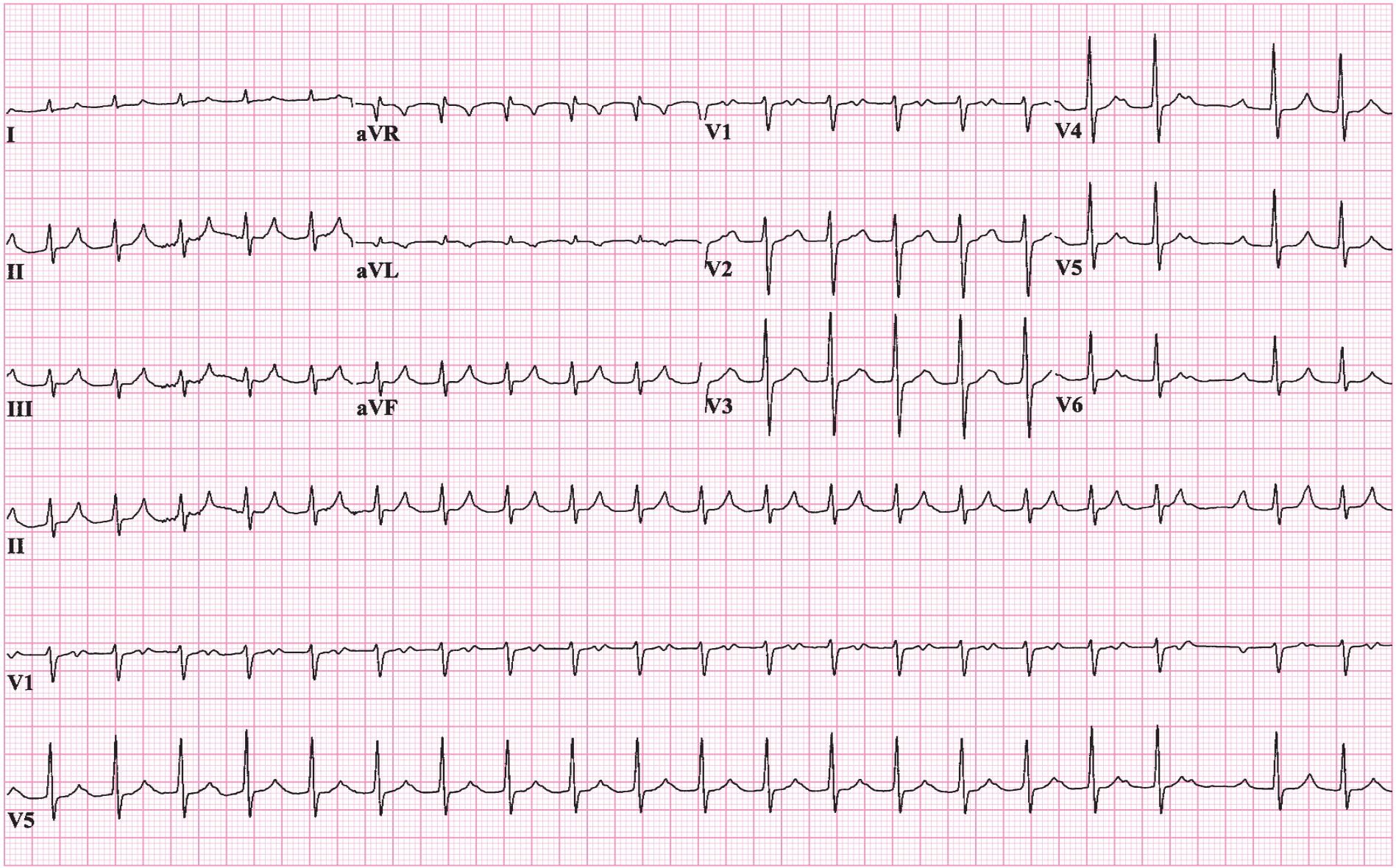
1. Posterior myocardial infarction is diagnosed on ECG in part by the presence of R wave amplitude exceeding S wave amplitude in leads V_1 and V_2. This diagnosis is difficult to make in the setting of RVH, WPW, and RBBB, since these conditions also manifest a dominant R wave in the right precordial leads. Posterior MI typically occurs in the setting of inferior MI, and is often accompanied by pathological Q waves in leads II, III, and aVF. (Answer: b, c, d)

2. The posterior wall of the left ventricular differs from the anterior, inferior, and lateral walls by not having ECG leads directly overlying it. Instead of Q waves and ST elevation, acute posterior MI presents with mirror-image changes in the anterior precordial leads (V_1-V_2), including dominant R waves (the mirror-image of abnormal Q waves) and horizontal ST segment depression (the mirror-image of ST elevation). This can be appreciated by turning the ECG over and looking at leads V_1-V_2 from behind, which will demonstrate the classic appearance of Q waves and ST elevation. (Answer: c)

— Quick Review 24 —

Inferior MI, age indeterminate or probably old	
• Abnormal Q waves (with/without) ST elevation in at least two of leads ____	without II, III, aVF
Posterior MI, age indeterminate or probably old	
• Initial R wave ≥ ____ seconds in leads ____ and ____ with:	0.04, V_1 - V_2
▸ R wave amplitude (greater than/less than) S wave amplitude	greater than

ECG 25. 68-year-old male with palpitations:



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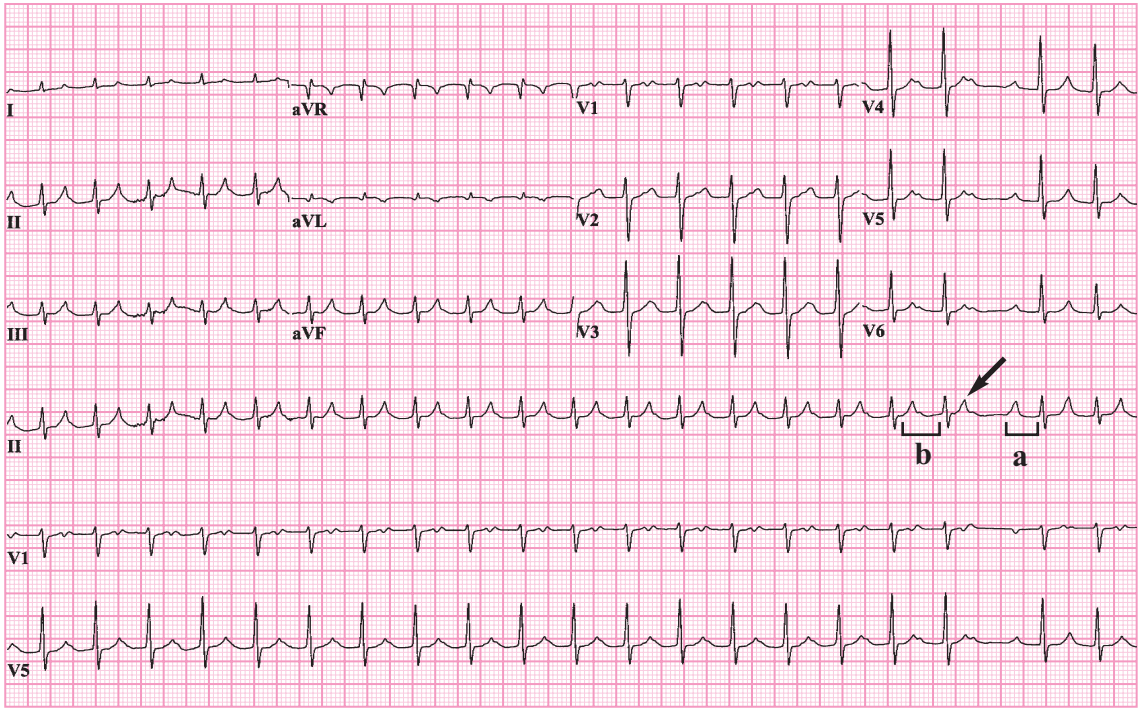
ECG 25 was obtained in a 68-year-old male with palpitations. The ECG shows sinus tachycardia at a rate of 125 beats/minute, with a tall P wave (> 2.5 mm) in lead II consistent with right atrial enlargement. After the 18th QRS complex there is a P wave that fails to conduct to the ventricle (arrow). By comparing the PR interval before (b) and after (a) the nonconducted P wave, it is evident that PR prolongation has occurred prior to the nonconducted P wave followed by shortening of the PR interval. This is consistent with second-degree AV block, Mobitz I (Wenckebach) with an extremely long Wenckebach cycle. With the resumption of conduction following the nonconducted P wave, the PR interval is prolonged, consistent with first-degree AV block.

Codes:

05	Right atrial abnormality/enlargement
10	Sinus tachycardia (> 100)
29	AV block, 1°
30	AV block, 2°-Mobitz type I (Wenckebach)



Questions: ECG 25

1. In Mobitz Type I second-degree AV block, classic Wenkebach periodically is always evident on ECG:

   a. True
   b. False

2. In Mobitz Type I second-degree AV block with infrequent pauses, the PR interval of the beats immediately preceding the blocked P wave may not demonstrate progressive prolongation:

   a. True
   b. False

Answers: ECG 25

1. In Mobitz Type I second-degree AV block, classical Wenckebach periodicity – progressive prolongation of the PR interval and progressive shortening of the RR interval until a P wave is blocked – may not always be evident, especially when sinus arrhythmia is present or an abrupt change in autonomic tone occurs. (Answer: b)

2. In Mobitz Type I second-degree AV block with high conduction ratios (i.e., infrequent pauses), the PR interval of the beats immediately preceding the blocked P wave may be equal to each other, suggesting Type II block. In these situations, it is best to compare the PR intervals immediately before and after the blocked P wave: differences in the PR intervals suggest Type I block, whereas a constant PR interval suggests Type II block. (Answer: a)

— Quick Review 25 —

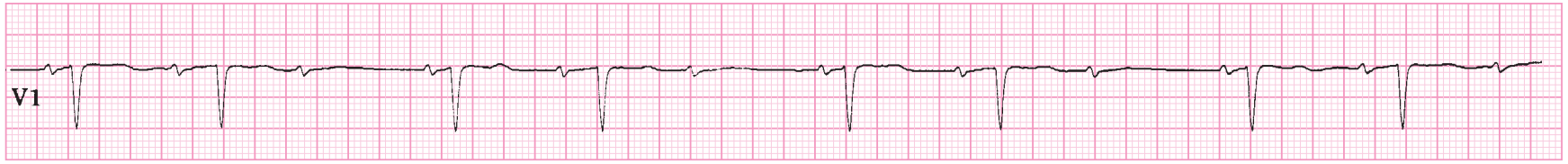
AV block, 1°	
• PR interval ≥ ____ seconds	0.20
AV block, 2° - Mobitz Type I (Wenckebach)	
• Progressive prolongation of the ____ interval and shortening of the ____ interval until a P wave is blocked	PR RR
• RR interval containing the nonconducted P wave is (less than/equal to/greater than) the sum of two PP intervals	less than
• Results in ____ beating due to the presence of nonconducted P waves	group

— POP QUIZ —

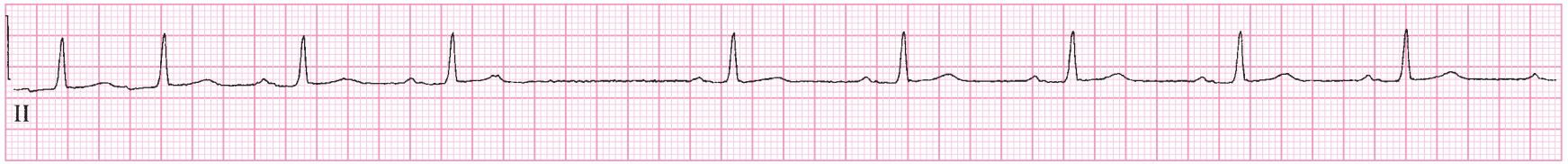
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

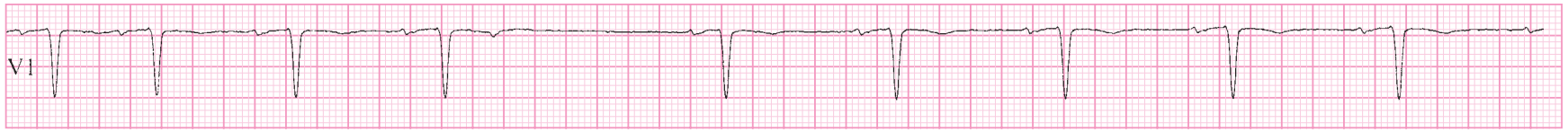


B.



C.

D.



Answer: Tracings B, C, and D show sinus rhythm with blocked APCs. In these tracings, the premature (and blocked) P waves are superimposed on the T waves, giving the impression of a sinus pause. Tracing A shows 2° AV block, Mobitz type 1 (Wenckebach), and is the imposter. In this tracing, there is gradual prolongation of the PR interval leading up to the blocked P wave. The easiest way to confirm the presence of Wenckebach block is to confirm the presence of a longer PR interval in the beat immediately before the non-conducted P wave and a shorter PR interval in the beat immediately after the nonconducted P wave.

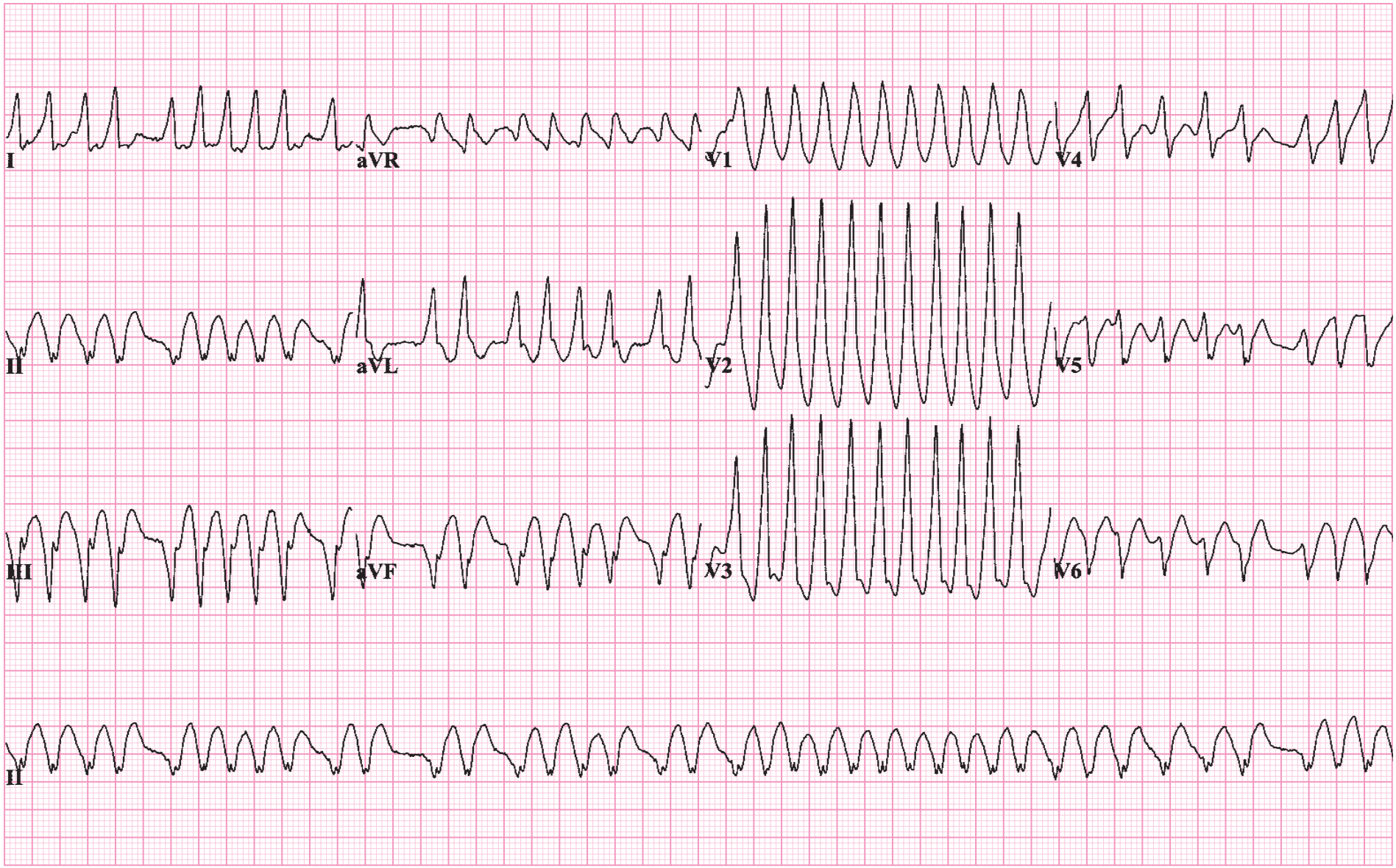
— POP QUIZ —

Differential Diagnosis: U-wave

Instructions: Determine whether the diagnoses below are associated with prominent upright U waves, inverted U waves, or both.

Diagnosis	Answer
Hypokalemia	Prominent upright U waves. ST depression and flattened T waves are common.
Bradycardia	Prominent upright U waves.
Hypothermia	Prominent upright U waves. Osborne (J) waves and prolongation of PR, QRS, and QT are common.
Left ventricular hypertrophy (LVH)	Prominent upright or inverted U waves.
Coronary artery disease	Prominent upright or inverted U waves.
Digoxin	Prominent upright U waves. Sagging ST depression with upward concavity and T wave changes (flat, inverted, or biphasic) are common. QT shortening and PR prolongation may occur.
Antiarrhythmic drugs	Prominent upright U waves (one of earliest findings). Prolonged QT interval and nonspecific ST and T wave changes are common.

ECG 26. 24-year-old male with palpitations and near syncope:



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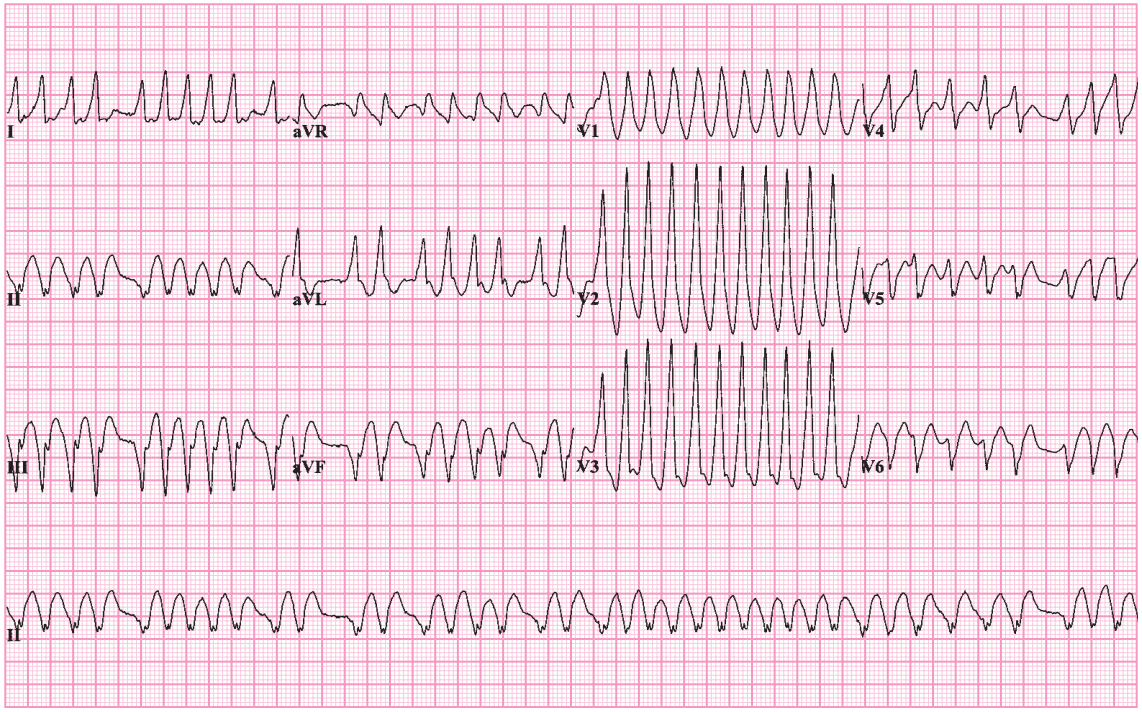
ECG 26 was obtained in an 24-year-old male with palpitations and near syncope. The ECG is consistent with atrial fibrillation with an irregularly-irregular ventricular response and a ventricular rate varying between 150 and 300 beats/minute. The QRS complex is wide with a variable degree of intraventricular conduction delay that does not fit the typical right bundle branch block or left bundle branch block pattern. The variation in the QRS duration coupled with a wide complex tachycardia are consistent with WPW pattern (ventricular pre-excitation). The accessory pathway is connecting the left atrium and left ventricle (indicated by the positive QRS complex in lead V_1) and is located in the posterior portion of the left atrium (indicated by the negative QRS complexes in leads II, III, and aVF). The combination of an irregularly-irregular rhythm with a rapid, wide QRS complex should always bring to mind atrial fibrillation with WPW syndrome and rapid conduction over the accessory AV pathway.

Codes:

19 Atrial fibrillation

34 Wolff-Parkinson-White pattern



Questions: ECG 26

1. The location of the accessory pathway in a patient with WPW and positive delta waves/QRS polarity in leads V_1 and aVF is:

   a. Left lateral
   b. Left posterior
   c. Right posterior
   d. Right lateral

Answers: ECG 26

1. Several algorithms have been published to predict accessory pathway location by assessing the initial polarity of the delta wave and the QRS complex using the 12-lead ECG. Each algorithm has inaccuracies in individuals demonstrating less than maximal preexcitation of the QRS complex. The following table lists a simple algorithm that allows identification of the general accessory pathway location in patients with WPW. The first step for localizing the pathway is to identify the polarity (positive, negative, or isoelectric) of the delta wave and the main portion of the QRS complex in ECG leads aVL, aVF, and V_1. The table below is then used to determine the approximate location of the accessory pathway (Answer: a)

Table. Delta Wave/QRS Polarity and Relationship to Location of the Accessory Pathway

	V_1	aVF	aVL
Left lateral	+	+	–
Left posterior/septal	+	–	+
Right posterior/septal	–	–	+
Right lateral/anterior	–	+	+

— Quick Review 26 —

Atrial fibrillation

- ____ waves are absent — P
- Atrial activity is totally ____ and represented by fibrillatory (f) waves of varying amplitudes, duration and morphology — irregular
- Atrial activity is best seen in the ____ and ____ leads — right precordial, inferior
- Ventricular rhythm is (regularly/irregularly) irregular — irregularly
- ____ toxicity may result in regularization of the RR interval due to complete heart block with junctional tachycardia — Digitalis
- Ventricular rate is usually ____ per minute in the absence of drugs — 100-180
  - ▸ Think ____ if the ventricular rate is > 200 per minute and the QRS is > 0.12 seconds — Wolff-Parkinson-White

Wolff-Parkinson-White pattern

- (Sinus/nonsinus) P wave — sinus
- PR interval < ____ seconds — 0.12
- Initial slurring of QRS (____ wave) resulting in QRS duration > ____ seconds — delta, 0.10
- Secondary ST-T wave changes occur (true/false) — true
- PJ interval, i.e., beginning of P wave to end of QRS, (is constant/varies) — is constant

— POP QUIZ —

Make The Diagnosis

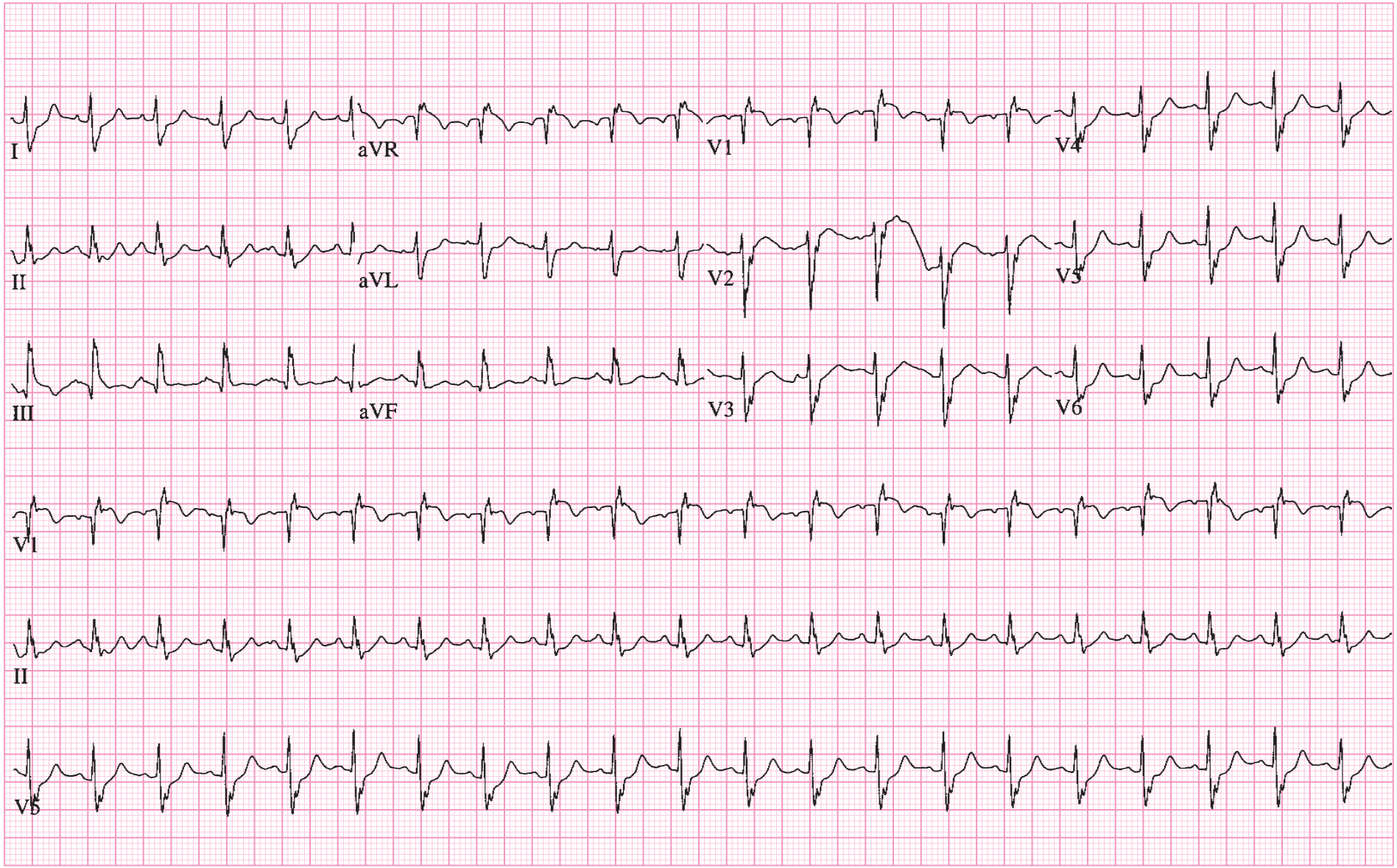
Instructions: Determine the clinical disorder that best corresponds to the ECG features listed below (see items 70-89 on score sheet for options).

ECG Features	Diagnosis
• Sinus bradycardia • PR, QRS, and QT prolonged) • Osborne ("J") wave: late upright terminal deflection of QRS complex • Atrial fibrillation in 50-60%	Hypothermia
• "Classic changes" usually occur in the precordial leads ▸ Large upright or deeply inverted T waves ▸ Prolonged QT interval (often marked) ▸ Prominent U waves • Other changes: ▸ ST segment changes: • Can mimic acute pericarditis or acute myocardia injury • ST depression may also occur ▸ Abnormal Q waves mimicking MI ▸ Almost any rhythm abnormality including sinus tachycardia or bradycardia, junctional rhythm, VPCs, ventricular tachycardia, etc.	CNS disorder
• Low QRS voltage in all leads • Sinus bradycardia • T wave flattened or inverted • PR interval may be prolonged • Frequently associated with pericardial effusion • Electrical alternans may occur	Myxedema

Don’t Forget!

- When a VPC originates on the same side as a bundle branch block, the resulting fusion complex can be narrow
- Think of parasystole when you see ventricular complexes with nonfixed coupling and fusion beats
- Look for ventricular capture complexes and fusion beats as markers for VT in the setting of a wide QRS tachycardia
- Classical Wenckebach periodicity may not always be evident, especially when sinus arrhythmia is present or an abrupt change in autonomic tone occurs
- 2:1 AV block can be Mobitz Type I or Type II
- In WPW, the PJ interval (beginning of P wave to end of QRS complex) is constant and ≤ 0.26 seconds
- Think of WPW when atrial fibrillation or flutter is associated with a QRS that varies in width (generally wide) and has a rate >200 per minute

ECG 27. 39-year-old male with acute shortness of breath:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
- ☐ 11. Sinus pause or arrest
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- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 20. AV junctional premature complexes
- ☐ 21. AV junctional escape complexes
- ☐ 22. AV junctional rhythm/tachycardia

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- ☐ 23. Ventricular premature complexes
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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
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- ☐ 79. Atrial septal defect, primum
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- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

PACED RHYTHMS

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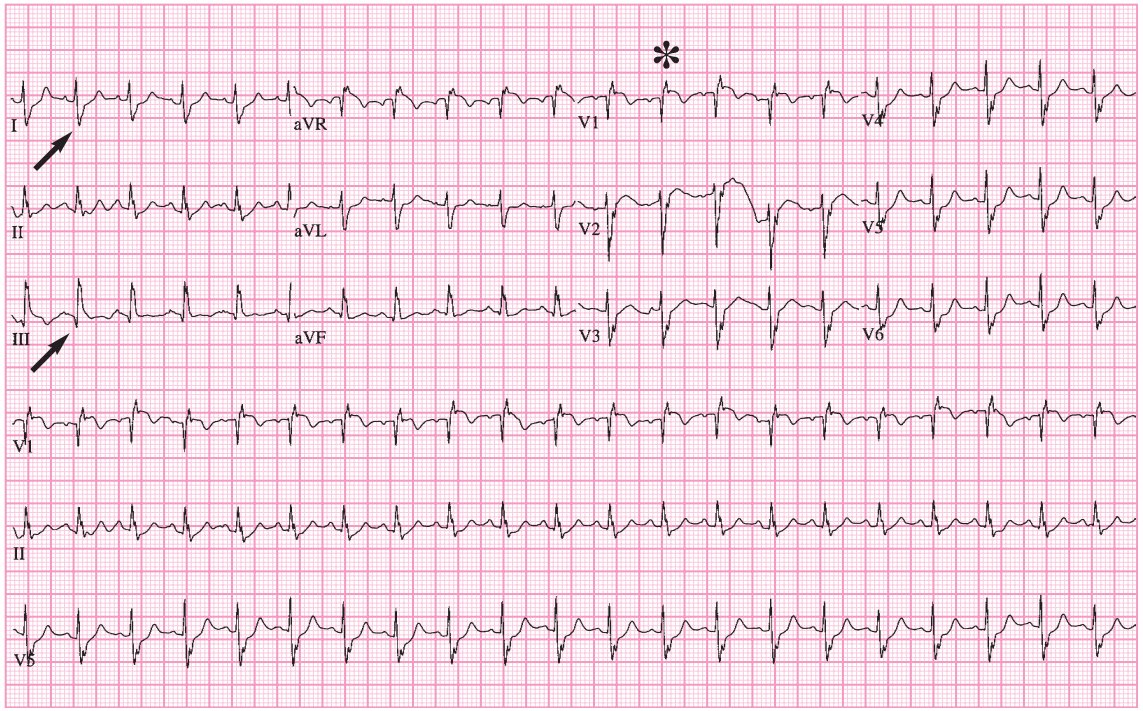
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 27 was obtained in a 39-year-old male with acute shortness of breath. The ECG shows sinus tachycardia at 127 beats/minute with right axis deviation (QRS axis = 104°) and a prominent S wave in lead I and Q wave in lead III (the classic S_1Q_3 pattern) (arrows). These findings suggest the diagnosis of pulmonary embolism. Further clues include the presence of incomplete RBBB (asterisk) and ischemic looking repolarization abnormalities in V_1-V_3.

Codes:

10	Sinus tachycardia
37	Right axis deviation
44	RBBB, incomplete
63	Nonspecific ST and/or T wave abnormalities
82	Acute cor pulmonale, including pulmonary embolism



Questions: ECG 27

1. Pulmonary embolism can mimic acute myocardial infarction based on clinical presentation and ECG appearance:

   a. True
   b. False

2. The most common ECG finding in acute pulmonary embolism is:

   a. Sinus tachycardia
   b. RBBB
   c. Inverted T waves in lead V_1 to V_4
   d. S_1Q_3

Answers: ECG 27

1. The clinical presentation and ECG findings of acute pulmonary embolism (PE) can be confused with acute inferior or anterior myocardial infarction (MI). T wave inversions and Q waves are often seen inferiorly in the setting of PE, although a Q wave in lead II is uncommon in PE and suggests MI. T wave inversions in leads V_1-V_4 is a sign of right ventricular strain and can sometimes be associated with ST elevation in leads V_1 and V_2 (as noted in the current case). Symptoms such as dyspnea, chest discomfort, tachycardia, and syncope can be seen in both PE and acute MI. Clinically, it is important to remember that both conditions may exist simultaneously; pulmonary embolism can complicate acute MI and vice versa. (Answer: a)

2. Sinus tachycardia is a very common finding in the setting of significant pulmonary embolism. Other classic ECG findings for pulmonary embolism – RBBB, S_1Q_3, inverted T waves in the precordial leads, right axis deviation – are seen less frequently than sinus tachycardia. ECG abnormalities are often transient in PE, although sinus tachycardia usually persists. Evidence for acute right ventricular strain and clockwise rotation of the heart may be absent despite persistent embolus. Most ECG findings of pulmonary embolism are secondary to acute right ventricular strain. (Answer: a)

— Quick Review 27 —

Acute cor pulmonale, including pulmonary embolism	
• ___ 1 ___ 3 or ___ 1 ___ 3 ___ 3 occurs in up to 30% of cases and last 1-2 weeks	$S_1\ Q_3$ or $S_1\ Q_3\ T_3$
• (Right/left) bundle branch block, either incomplete or complete, may be seen in up to 25% of cases and usually lasts less than 1 week	right
• (Inverted/peaked) T waves secondary to right ventricular strain may be seen in the (right/left) precordial leads and can last for months	inverted right
• Other ECG findings include (right/left) axis deviation, nonspecific ST and T wave changes, and P pulmonale	right
• Arrhythmias and conduction disturbances include _____ tachycardia (most common arrhythmia), atrial fibrillation, atrial flutter, atrial tachycardia, and (first/second) degree AV block	sinus first
• The clinical presentation and ECG of acute pulmonary embolism may sometimes be confused with acute (inferior/anterior) MI; however, a Q wave in lead II is (uncommon/common) in pulmonary embolism and suggests MI	inferior uncommon
• ECG abnormalities are often (transient/permanent)	transient
• A normal ECG may be recorded despite persistence of the embolus (true/false)	true

— POP QUIZ —

Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

B.

C.

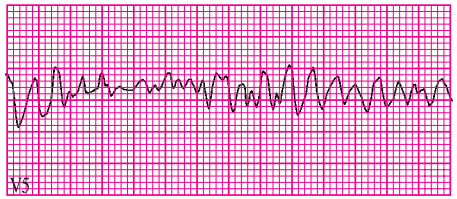
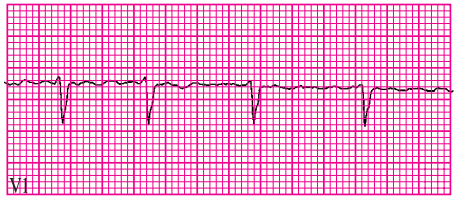
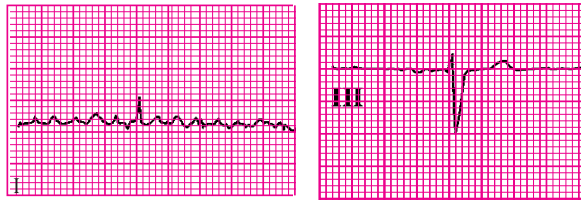
D.

Answer: Tracings A, C and D all show sinus tachycardia. Tracing C is a more subtle example of sinus tachycardia with the sinus P waves superimposed on the end of the preceding T waves. Tracing B represents supraventricular tachycardia with no evident P waves and is the imposter.

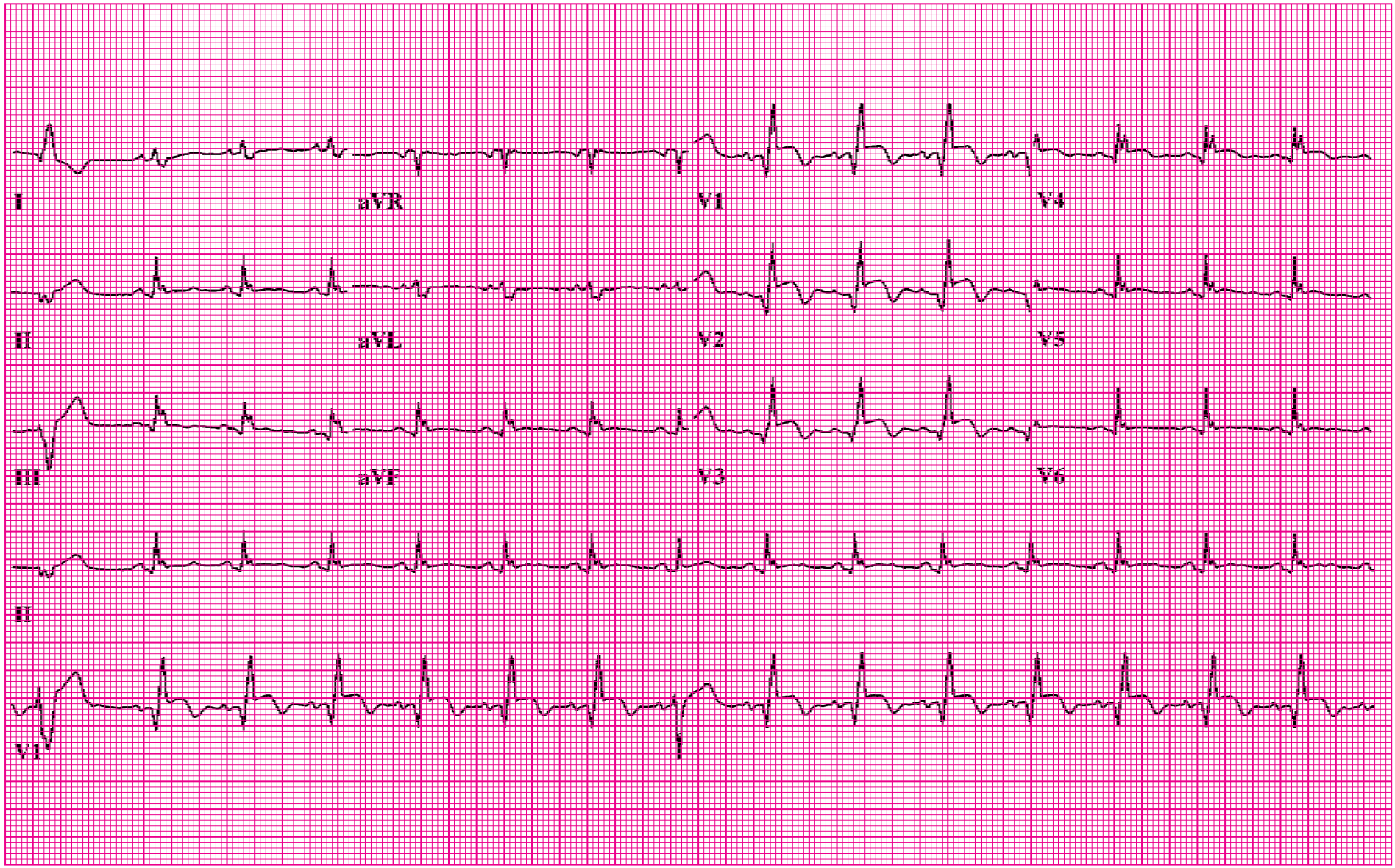
— POP QUIZ —

To Treat or Not to Treat, That Is the Question

Instructions: Select the best form of treatment for each of the following ECGs.

ECG	Choose Single Best Answer	Answer
	a. No treatment b. Digoxin c. Digoxin antibody d. Adenosine e. Cardioversion/defibrillation f. Stop aminophylline g. Pericardiocentesis h. Glucose + insulin	Early defibrillation is the major determinant of survival in cardiac arrest due to *ventricular fibrillation*. Resuscitation rates approach 100% if promptly managed in the first minute. When CPR and defibrillation are delayed for as little as 4-5 minutes, successful resuscitation occurs in only 25-35%. (Answer: e)
 Palpitations and dyspnea for 6 hours		*Atrial fibrillation* results in absent P waves, totally irregular atrial activity (causing random oscillation of the baseline), and an irregularly irregular ventricular rhythm. Treatment consists of anticoagulation, rate control (digoxin, beta-blocker, or calcium antagonist) followed by chemical or electrical cardioversion, depending on the duration of atrial fibrillation and the severity of symptoms. This patient is best treated with electrical cardioversion since the rate is already under control. (Answer: e)
		Patients with *physiologic tremor* can manifest P wave-like artifact at a rate of 7-9 cycles per second (~500 per minute), as in this ECG. Parkinson's tremor results in baseline artifact at a rate of 4-6 cycles per second (~300 per minute), which can be mistaken for atrial flutter. (The baseline undulations in this tracing are too fast for atrial flutter, which occurs at rates of 240-340 per minute.) Other causes of P wave artifact include alternating current (AC) electrical interference, skeletal muscle fasciculations (shivering), and IV infusion pump. (Answer: a)

ECG 28. 76-year-old female with chest pain and syncope:



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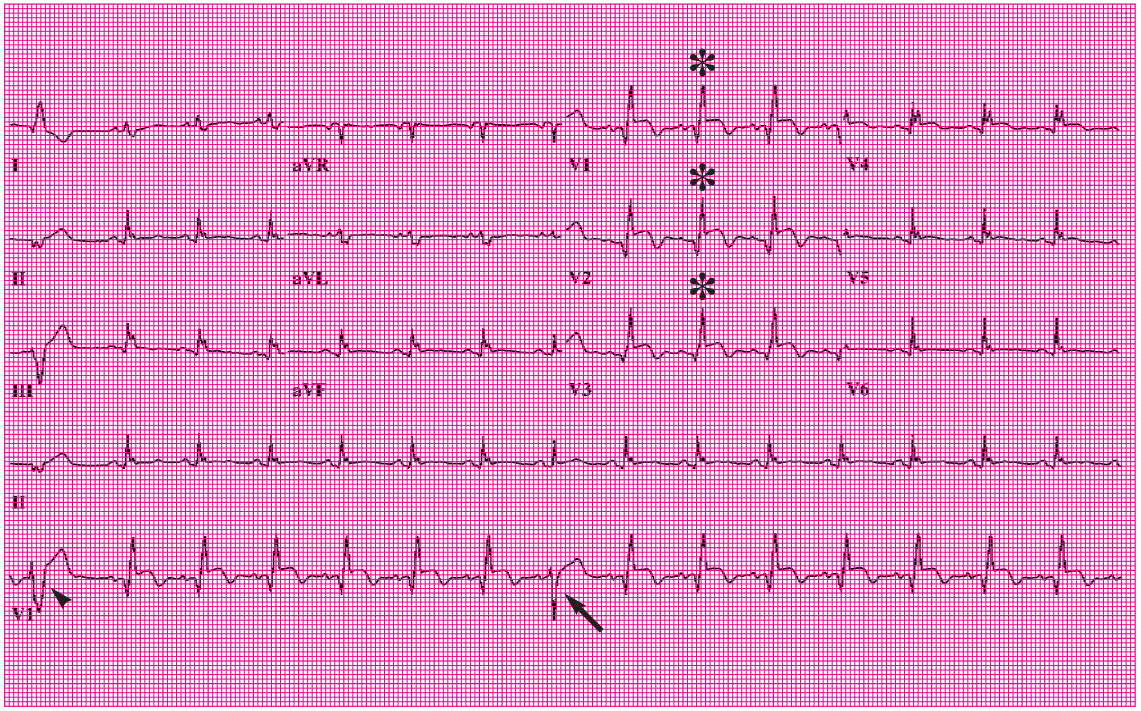
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 28 was obtained in a 76-year-old female with chest pain and syncope. The ECG shows normal sinus rhythm with ventricular premature complexes (arrowhead). The second VPC (arrow) is a fusion complex, which is intermediate in morphology between a regular sinus beat and a VPC. Also present are RBBB and acute anteroseptal myocardial infarction, with Q waves and ST segment elevation most notable in leads V_1-V_3 (asterisks). ST-T changes suggesting myocardial injury should also be coded.

Codes:

07	Sinus rhythm
23	Ventricular premature complexes
43	RBBB, complete
53	Anterior or anteroseptal Q wave MI (age recent or acute)
65	ST and/or T wave abnormalities suggesting myocardial injury



Questions: ECG 28

1. The presence of right bundle branch block (RBBB) invalidates the usual criteria for diagnosing acute anteroseptal myocardial infarction:

   a. True
   b. False

Answers: ECG 28

1. Patients with right bundle branch block without underlying structural heart disease have essentially the same prognosis as the general population. Among patients with coronary artery disease, RBBB is associated with a 2-fold increase in mortality compared to patients without bundle branch block. RBBB does not interfere with identification of abnormal Q waves or ST segment elevation of acute myocardial infarction, since the initial 0.08 seconds of the QRS complex is formed by conduction down the left bundle. (Answer: b)

— Quick Review 28 —

Ventricular premature complexes	
• A wide, notched or slurred ____ complex that is premature relative to the normal RR interval and is not preceded by a ____ wave	QRS P
• QRS duration is almost always > ____ seconds	0.12
• Initial direction of the QRS is often (similar to/different from) the QRS during sinus rhythm	different from
• Secondary ST & T wave changes in the (same/opposite) direction as the major deflection of the QRS (i.e., ST depression & T wave inversion in leads with a dominant _____wave; ST elevation and upright T wave in leads with a dominant _____ wave or ____ complex)	opposite R S QS
• Coupling interval is constant or varies by < ____ seconds	0.08
• Morphology of VPCs in any given lead is (the same/different)	the same
• Retrograde capture of atria may occur (true/false)	true
• A full ____ pause (PP interval containing the VPC is twice the normal PP interval) is usually evident	compensatory
Anterior or anteroseptal MI (age recent or acute)	
• Abnormal Q or QS deflection and ST elevation in leads ____ (and sometimes V_4)	V_1-V_3
• The presence of a Q wave in lead ____ distinguishes anteroseptal from anterior infarction	V_1

— Quick Review 28 (cont'd) —

ST and/or T wave changes suggesting myocardial injury	
• Acute ST segment (elevation/depression) with upward (convexity/concavity) in the leads representing the area of infarction	elevation convexity
• T waves invert (before/after) ST segments return to baseline	before
• Associated ST (elevation/depression) in the noninfarct leads is common	depression
• Acute ____ wall injury often has horizontal or downsloping ST segment depression with upright T waves in V_1-V_3, with or without a prominent R wave in these same leads	posterior

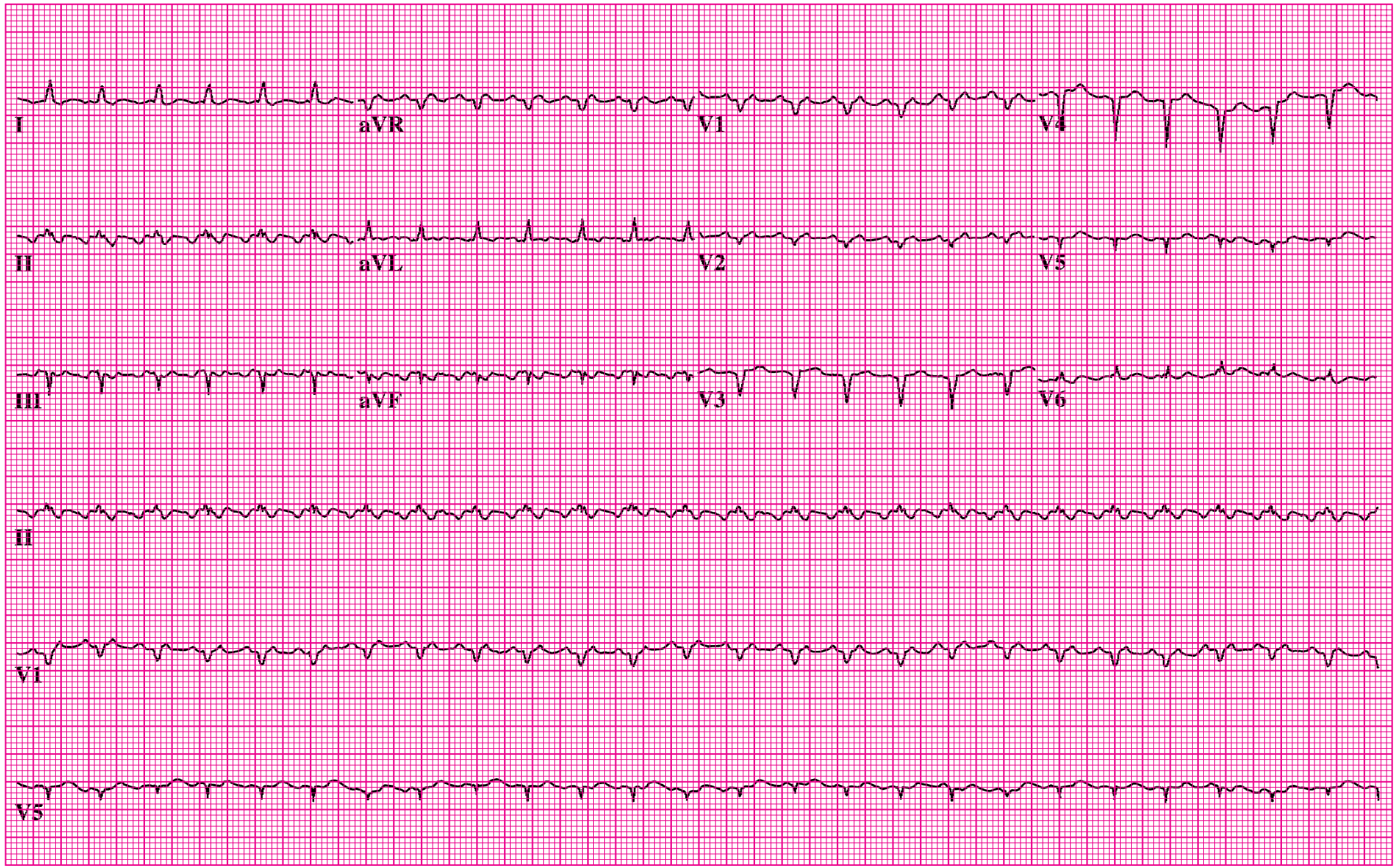
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the clinical disorder that best corresponds to the ECG features listed below (see answer sheet for options).

ECG Features	Answer
• Abnormal Q waves and ST elevation in leads I and aVL	Lateral MI, acute or recent
• Abnormal Q waves and ST elevation in at least two of leads II, III, and aVF • Associated ST depression usually evident in leads I, aVL, V_1-V_3	Inferior MI, probably acute or recent
• Abnormal Q waves (duration $\geq$ 0.03 seconds) and ST segment elevation in leads V_4-V_6	Anterolateral MI, acute or recent
• Abnormal Q or QS deflection with ST elevation in V_1-V_4	Anteroseptal or anterior MI, acute or recent
• Initial R wave $\geq$ 0.04 seconds in leads V_1 and V_2 with R wave amplitude > S wave amplitude, and ST segment depression with upright T waves • Usually seen in the setting of acute inferior MI	Posterior MI, probably acute or recent
• ST segment elevation $\geq$ 1 mm persisting 4 or more weeks after acute MI in leads with abnormal Q waves	Probable ventricular aneurysm

ECG 29. 56-year-old female with breast cancer, shortness of breath, and weakness:



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- ☐ 66. ST and/or T wave abnormalities suggesting electrolyte disturbances
- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

SUGGESTED CLINICAL DISORDERS

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

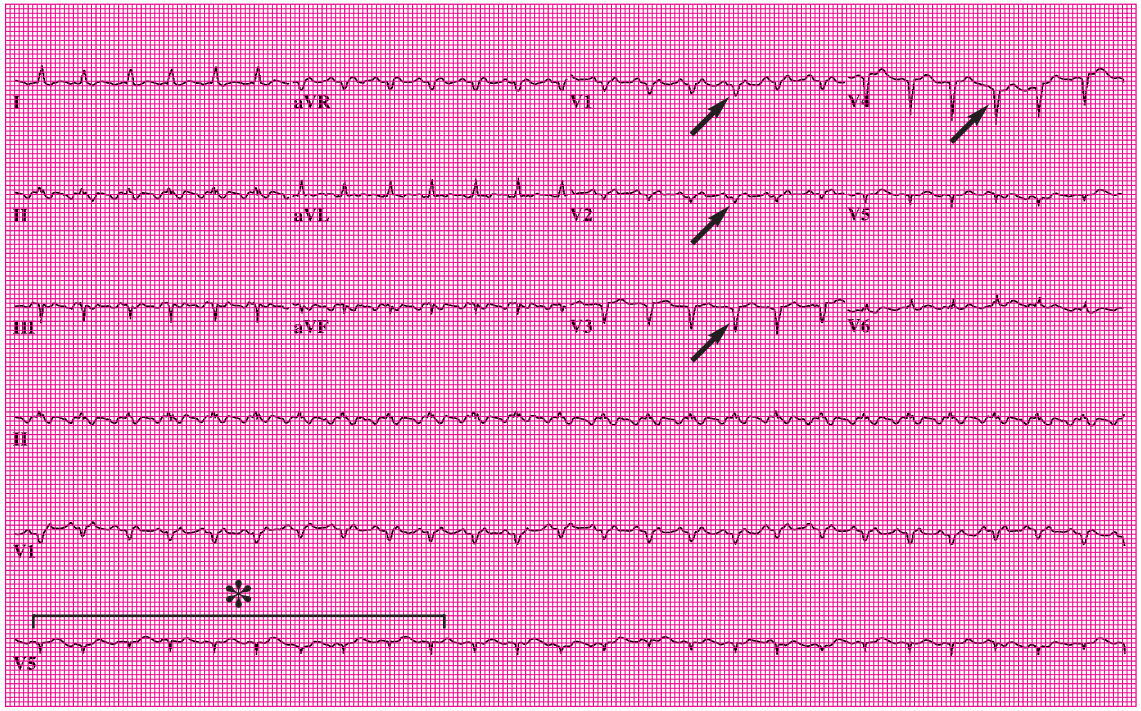
PACED RHYTHMS

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

ECG 29 was obtained in a 56-year-old female with breast cancer, shortness of breath, and weakness. The ECG shows atrial flutter with 2:1 AV block (2 flutter waves for every QRS complex). The flutter waves are inverted in lead II and upright in lead V_1, as is typically the case. Low voltage QRS is present, and there is subtle evidence for electrical alternans, which is most noticeable in the rhythm strip (asterisk). These findings are consistent with pericardial effusion (which is probably malignant in this case). Q waves in leads V_1-V_4 (arrows) suggest old anteroseptal/anterior myocardial infarction. Although Q waves are present in V_4-V_5, anterolateral MI should not be coded since abnormal Q waves are absent in V_6.

Codes:

18	Atrial flutter
32	AV block, 2:1
38	Electrical alternans
39	Low voltage
54	Anterior or anteroseptal Q wave MI (age indeterminate or old)
83	Pericardial effusion



Questions: ECG 29

1. The diagnosis of low voltage requires a QRS amplitude less than ___ mm in all limb leads and less than ___ mm in all precordial leads:

   a. 7, 10
   b. 5, 15
   c. 5, 10
   d. 10, 5

2. Atrial flutter with 2:1 block usually results in a ventricular rate of approximately ___ beats per minute:

   a. 100
   b. 150
   c. 300
   d. 180

3. ECG findings characteristic of pericardial effusion include:

   a. ST elevation
   b. Low voltage QRS complexes
   c. PR depression
   d. Electrical alternans

Answers: ECG 29

1. For board scoring purposes, a diagnosis of "low voltage" requires the presence of low voltage in all limb and precordial leads. Low voltage in the limb leads requires a total QRS amplitude (maximal deflection of R + S wave) < 5 mm; low voltage in the precordial leads requires a total QRS amplitude < 10 mm. Clinical conditions associated with low voltage QRS complexes include pleural or pericardial effusion, restrictive or infiltrative cardiomyopathies, diffuse myocardial disease with multiple prior infarctions, obesity, and emphysema (chronic lung disease). (Answer: c)

2. In typical atrial flutter, flutter (or "F" waves) occur at a rate of 300 per minute. Therefore, atrial flutter with 2:1 block usually results in a ventricular rate of 150 per minute. Flutter waves are sometimes difficult to recognize, and are usually best seen in the inferior leads (II, III, and aVF) and in lead V_1. The ventricular rhythm may be regular or irregular depending on whether AV nodal conduction is constant or variable. (Answer: b)

3. Low voltage QRS complexes and electrical alternans are consistent with (but neither sensitive nor specific for) the diagnosis of pericardial effusion. ECG findings of acute pericarditis (PR depression, ST segment elevation) may or may not be present. (Answer: b, d)

— Quick Review 29 —

Atrial flutter	
• Rapid (regular/irregular) atrial undulations ("F" waves) at a rate of ____ per minute	regular 240-340
• Flutter rate may (increase/decrease) in the presence of Types IA, IC or III antiarrhythmic drugs	decrease
• Flutter waves in leads II, III, AVF are typically (inverted/upright) (with/without) an isoelectric baseline	inverted, without
• Flutter waves in lead V_1 are typically small (positive/negative) deflections (with/without) a distinct isoelectric baseline	positive, with
• QRS complex may be normal or aberrant (true/false)	true
• AV conduction ratio (ratio of flutter waves to QRS complexes) is usually (fixed/variable)	fixed
▸ Conduction ratios of 1:1 and 3:1 are (common/uncommon)	uncommon
▸ In untreated patients, AV block ≥ ____ suggests the coexistence of AV conduction disease	4:1
Pericardial effusion	
• (High/low) voltage QRS	Low
• Electrical ____, especially if complicated by cardiac ____	alternans tamponade
• Other features of acute ____ may also be present	pericarditis

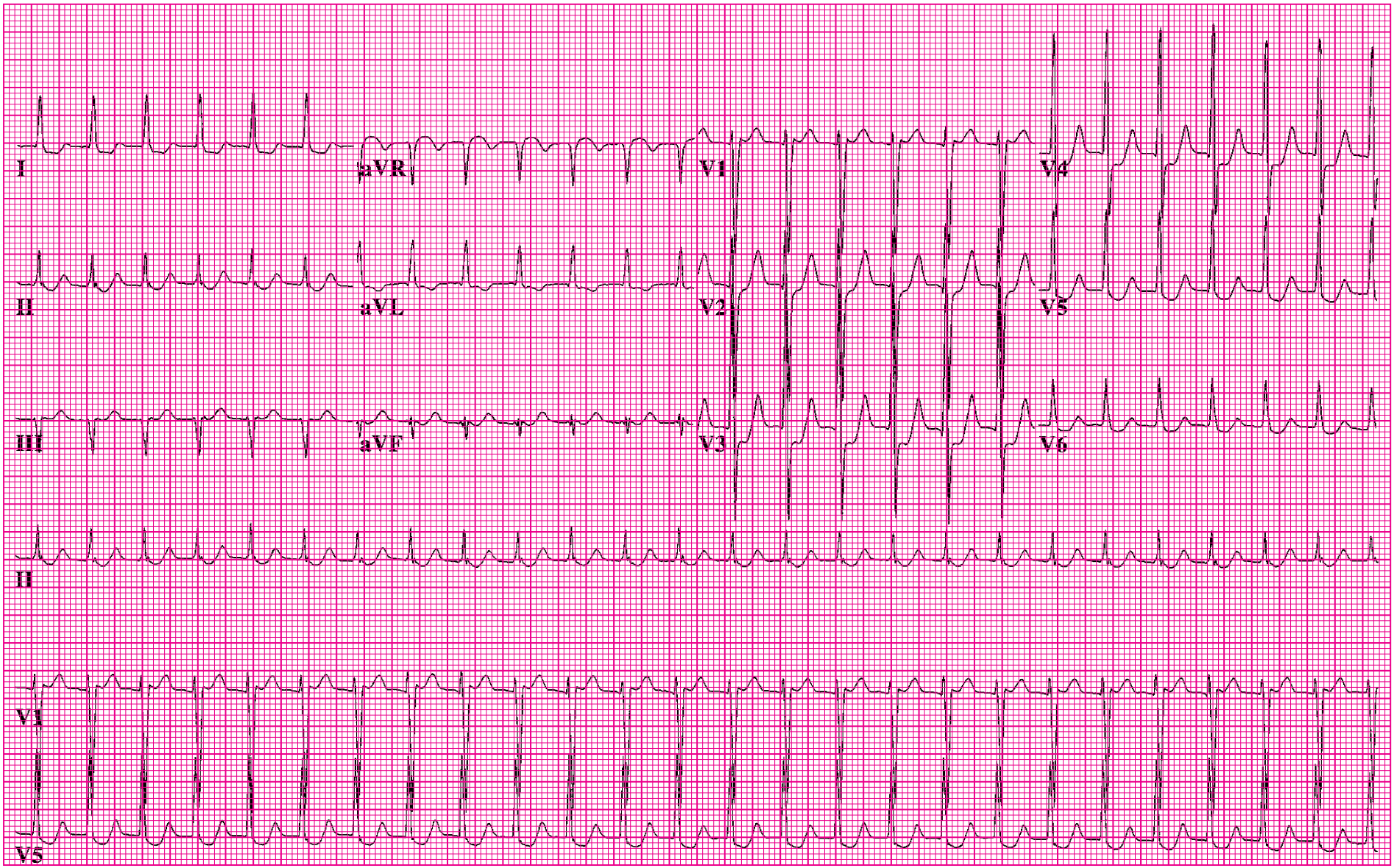
Differential Diagnosis

INTRAVENTRICULAR CONDUCTION DISTURBANCE

(QRS duration $\geq$ 0.11 seconds in duration but QRS morphology does not meet criteria for LBBB or RBBB, *or* abnormal notching of the QRS complex is present without prolongation)

- Antiarrhythmic drug toxicity (especially Type IA and IC agents)
- Hyperkalemia
- LVH
- Wolff-Parkinson-White
- Hypothermia
- Severe metabolic disturbances

ECG 30. 71-year-old male with palpitations:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
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- ☐ 31. AV block , 2°-Mobitz type II
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- ☐ 34. Wolff-Parkinson-White pattern
- ☐ 35. AV dissociation

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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
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- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

- ☐ 61. Normal variant, early repolarization
- ☐ 62. Normal variant, juvenile T waves
- ☐ 63. Nonspecific ST and/or T wave abnormalities
- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
- ☐ 65. ST and/or T wave abnormalities suggesting myocardial injury
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- ☐ 68. Prolonged QT interval
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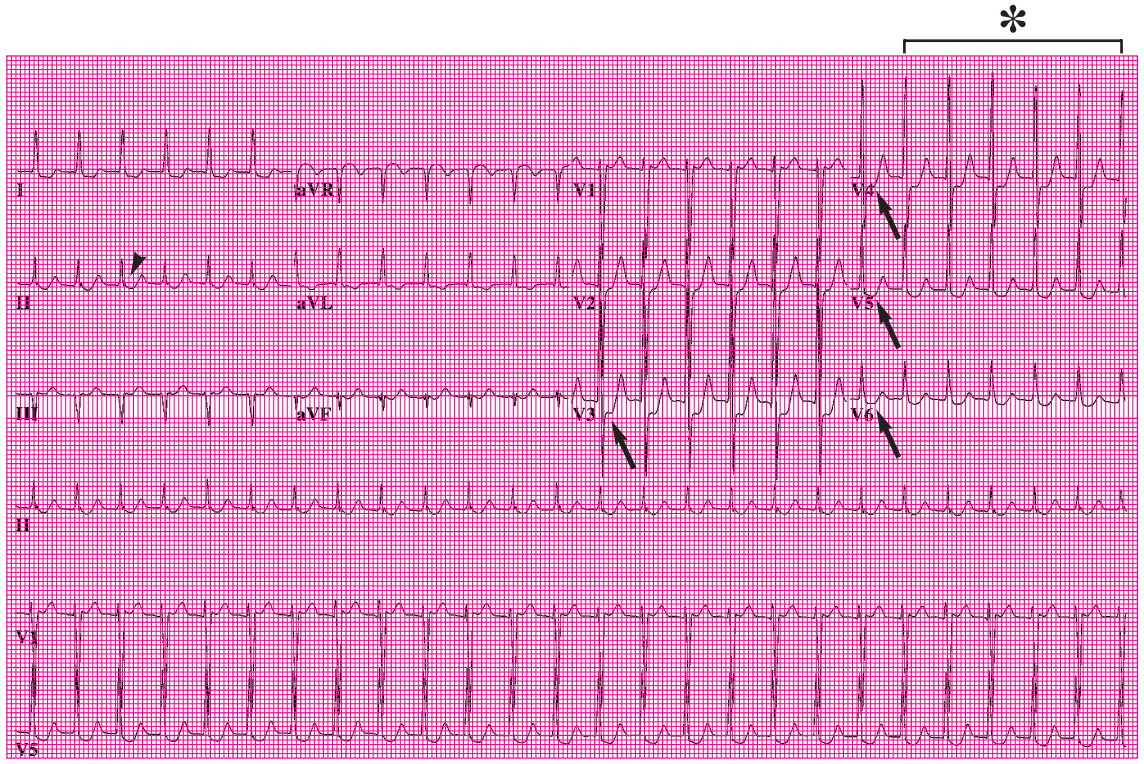
ECG 30 was obtained in a 71-year-old male with palpitations. The ECG shows a regular narrow complex tachycardia at 153 beats/minute with ST depression in V_3-V_6 suggesting subendocardial ischemia (arrows). Retrograde P waves are most noticeable in lead II, immediately following the QRS complex (arrowhead; also see full-size ECG on previous page). This tracing is most appropriately coded as supraventricular tachycardia. Electrical alternans is commonly seen in SVT and is present in this tracing (asterisk). Also noted is a prolonged QT interval. At electrophysiologic study, this patient was shown to have AV nodal re-entrant tachycardia.

Codes:

17	Supraventricular tachycardia, paroxysmal
38	Electrical alternans
64	ST and/or T wave abnormalities suggesting myocardial ischemia
68	Prolonged QT interval



Questions: ECG 30

1. Retrograde P waves are usually upright in leads II, III, and aVF:

   a. True
   b. False

2. ECG features consistent with a supraventricular origin rather than a ventricular origin for a tachycardia include:

   a. Capture or fusion beats
   b. Narrow QRS width
   c. Left axis deviation
   d. AV dissociation

Answers: ECG 30

1. Retrograde atrial activation results in inverted P waves in leads II, III, and aVF. The retrograde atrial wavefront moves in a superior direction away from the AV node and inferior leads, resulting in inverted P waves in these leads. Retrograde P waves typically occur with junctional beats and AV nodal re-entrant tachycardia, and sometimes with ventricular tachycardia or VPCs (if retrograde AV nodal conduction is present). (Answer: b)

2. The differentiation of a supraventricular from a ventricular rhythm is an important and frequent clinical dilemma.

   A <u>supraventricular</u> origin is favored if:
   - The QRS is narrow
   - QRS morphology is similar to that noted during a sinus rhythm or during an aberrantly conducted atrial premature complex
   - The tachyarrhythmia is initiated by an atrial premature complex

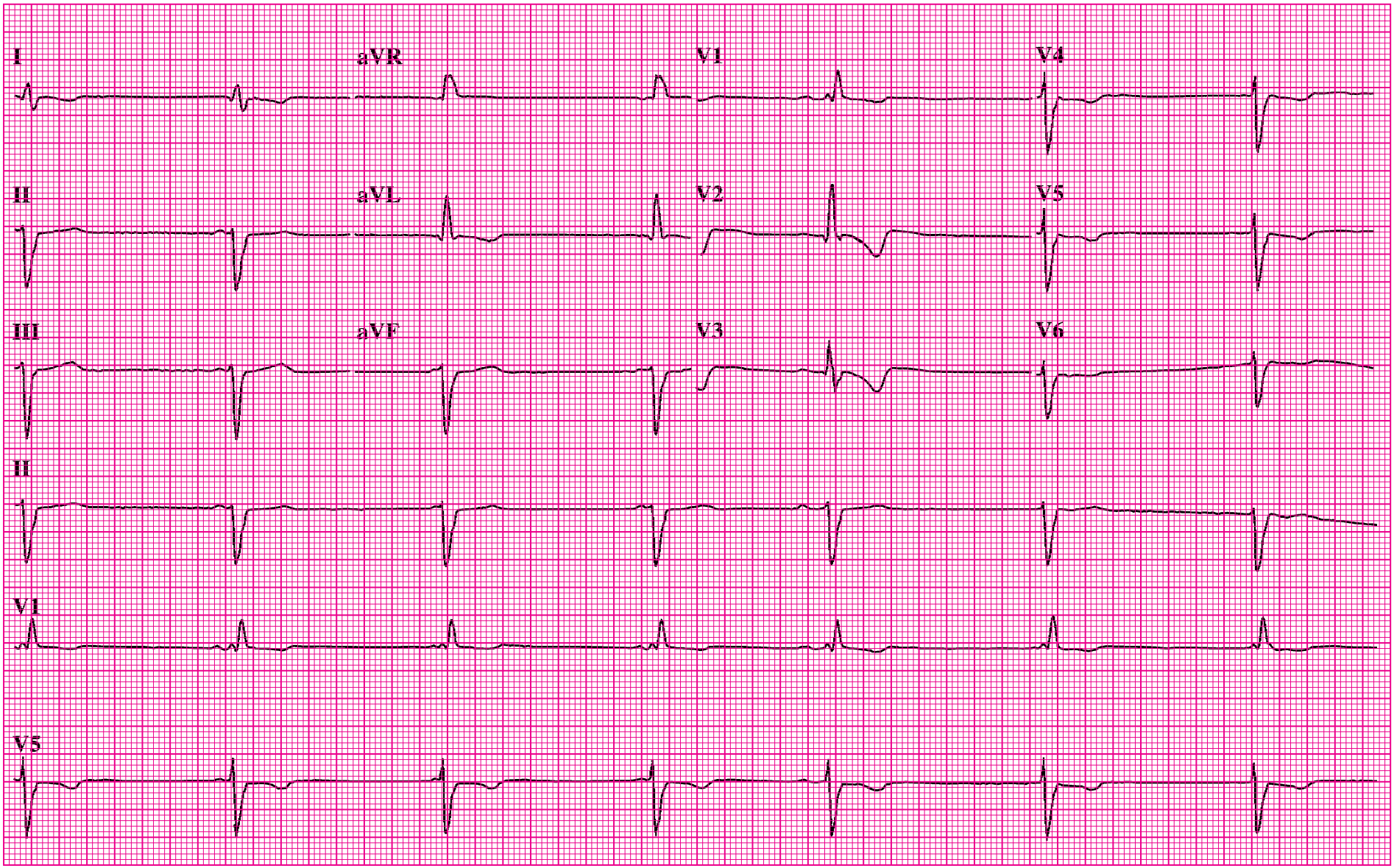
   A <u>ventricular</u> origin is favored if:
   - The QRS is wide (≥0.14 seconds in duration)
   - AV dissociation, capture beats, fusion beats are present
   - The QRS axis is leftward or northwest
   - Ventricular concordance is present
   - The dysrhythmia is initiated by a VPC

   (Answer: b)

— Quick Review 30 —

Supraventricular tachycardia	
• (Regular/irregular) rhythm	Regular
• Rate > ____ per minute	100
• P waves (always/sometimes) identified	sometimes
• QRS complex is usually (narrow/wide)	narrow
• If rate is 150 per minute, consider ____	atrial flutter with 2:1 block
Electrical alternans	
• Alteration in the ____ and/or ____ of the P, QRS and/or T waves	amplitude, direction

ECG 31. 78-year-old female with dizziness:



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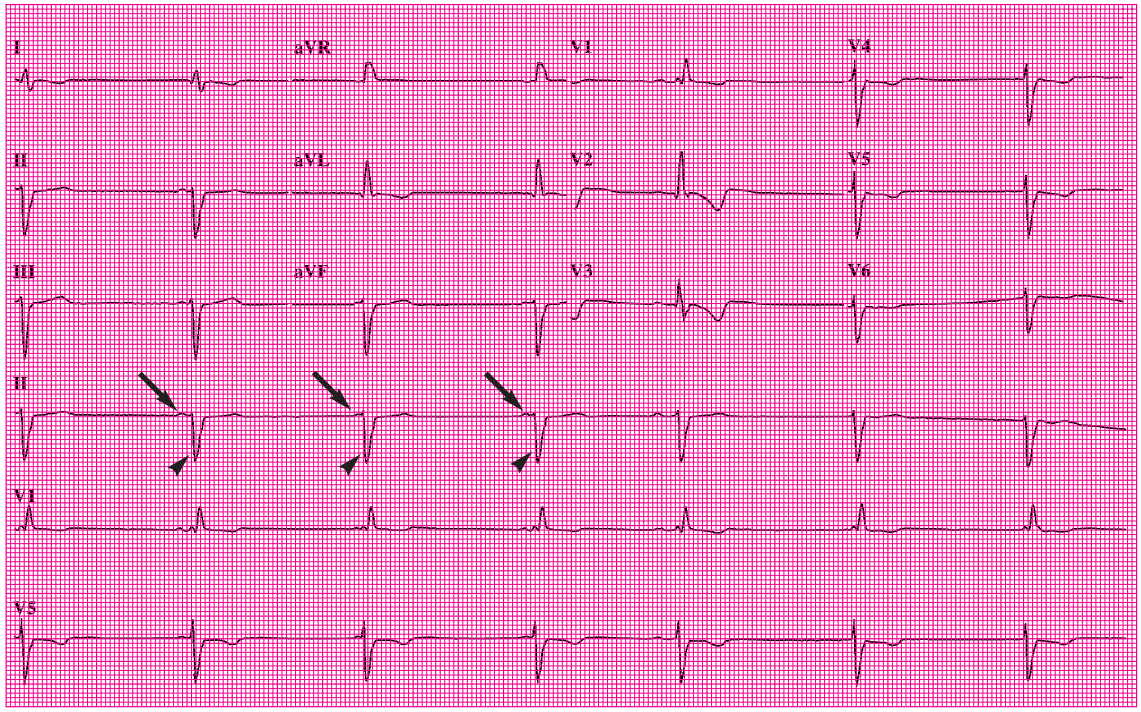
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 31 was obtained in a 78-year-old female complaining of dizziness. The ECG shows profound sinus bradycardia at 40 BPM (arrows mark sinus P waves) competing with a junctional rhythm (arrowheads mark junctional QRS complexes) and resulting in AV dissociation. Also present are sinus arrhythmia, RBBB with secondary ST-T changes, and left anterior fascicular block (left axis deviation does not require coding). The fifth complex on the tracing shows a normally conducted P wave resulting in a ventricular capture complex (which is premature compared to the junctional complexes). These findings are consistent with sick sinus syndrome.

Codes:

08	Sinus arrhythmia
09	Sinus bradycardia (< 60)
22	AV junctional rhythm/tachycardia
35	AV dissociation
43	RBBB, complete
45	Left anterior fascicular block
89	Sick sinus syndrome



Questions: ECG 31

1. Left anterior fascicular block requires an axis leftward of:

   a. – 30˚
   b. – 45˚
   c. 0˚
   d. – 90˚

2. Which of the following statements about junctional escape rhythms are true:

   a. AV dissociation is common
   b. Retrograde atrial activation is always evident
   c. The usual heart rate is 60-80 BPM
   d. The P wave may proceed the QRS

3. Incomplete RBBB and complete RBBB requires a QRS duration of ____ and ____ seconds, respectively:

   a. 0.9-0.12; ≥ 0.12
   b. 0.9-0.11; ≥ 0.11
   c. 0.9 to < 0.12; ≥ 0.12
   d. 0.11; 0.14

Answers: ECG 31

1. Left anterior fascicular block (LAFB) requires a QRS axis between - 45˚ and -90˚, and is typically associated with a normal to slightly prolonged QRS duration (0.08-0.10 seconds). Since LAFB is a diagnosis of exclusion, be sure to exclude other causes of left axis deviation (e.g., LVH, inferior infarction, LBBB, emphysema) before coding LAFB. (Answer: b)

2. The usual heart rate noted with a junctional escape rhythm is between 40-60 BPM. Junctional rhythms are often associated with isorhythmic AV dissociation (P waves and QRS complexes appear to bear a close relationship to each other but actually represent independent atrial and ventricular activation) or retrograde atrial activation (inverted P waves in leads II, III, and aVF). The P wave inscribed by a junctional pacemaker may proceed (by ≤ 0.11), be superimposed upon, or follow the QRS complex. (Answer: a, d)

3. Complete RBBB requires a QRS duration of ≥ 0.12 seconds (whereas incomplete RBBB requires a QRS duration between 0.09 and < 0.12 seconds). Lead V_1 is usually the most helpful lead for diagnosing RBBB, and typically displays an rSr' pattern. RBBB is not usually associated with extensive and diffuse ST-T wave (repolarization) abnormalities, although T wave inversions are often present in leads V_1 - V_3. (Answer: c)

— Quick Review 31 —

AV dissociation	
• Atrial and ventricular rhythms are ____ of each other	independent
• Ventricular rate is (</≥) than the atrial rate	≥
RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ___ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 > ____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ___	I, aVL, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
• RBBB (does/does not) interfere with the ECG diagnosis of ventricular hypertrophy or Q wave MI	does not
Left anterior fascicular block	
• ____ axis deviation with a mean QRS axis between ____ and ____ degrees	left – 45, –90
• (qR/rS) complex in leads I and aVL	qR
• (qR/rS) complex in lead III	rS
• Normal or slightly prolonged QRS duration (true/false)	true
• No other cause for left axis deviation should be present (true/false)	true
• Poor R wave progression is (common/uncommon)	common

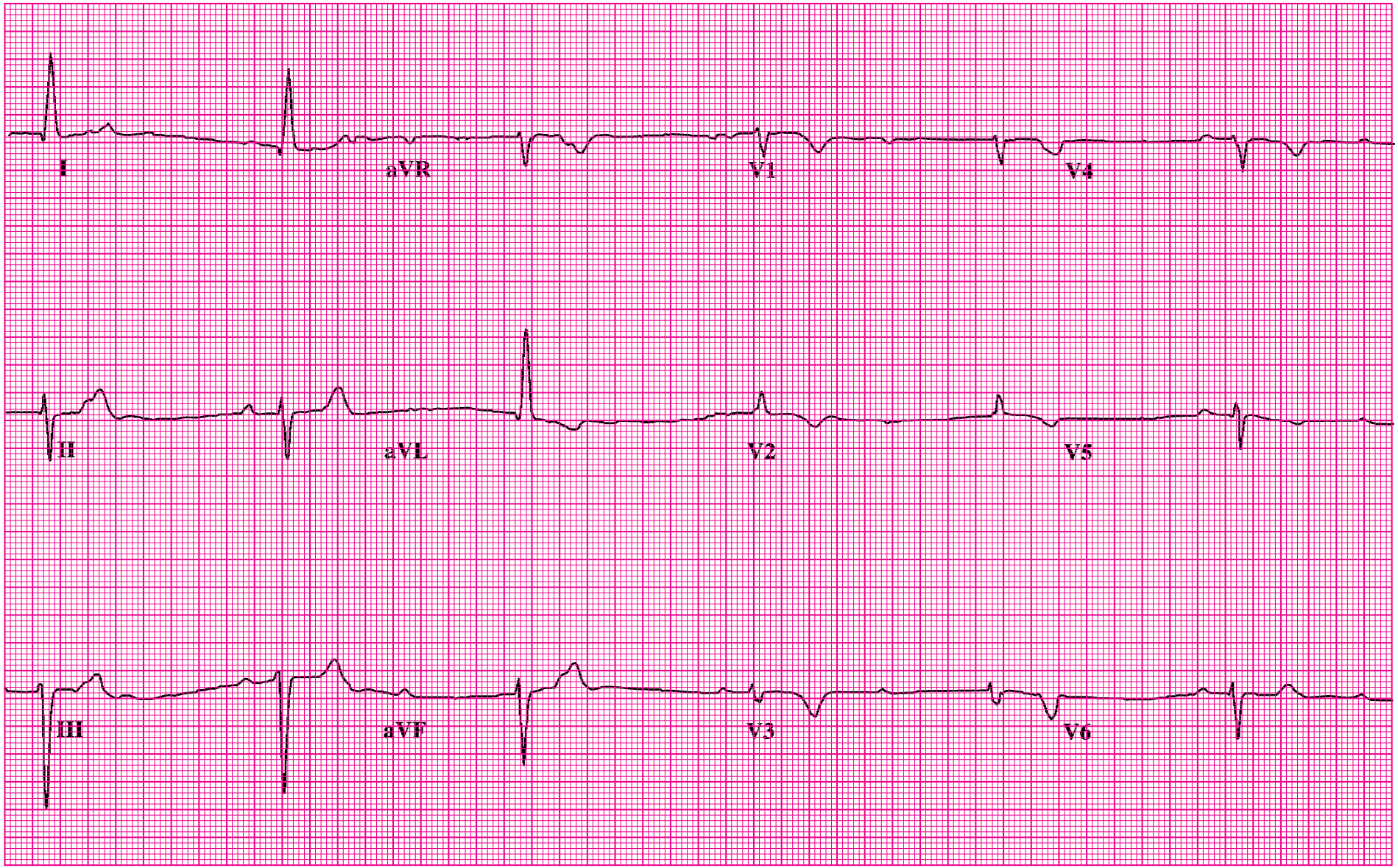
— POP QUIZ —

2:1 AV Block: Mobitz Type I or II

Instructions: Decide if the ECG features listed below favor Mobitz Type I (Wenkebach) or Mobitz Type II second-degree AV block

ECG Features	Mobitz Type I or II
AV block improves in response to maneuvers that reduce heart rate and AV conduction (e.g., carotid sinus massage)	Type II
AV block improves in response to maneuvers that increase heart rate and AV conduction (e.g., atropine, exercise)	Type I
2:1 block develops during anterior MI	Type II
Type I on another part of ECG	Type I
Wide QRS complex	Type II
History of syncope	Type II

ECG 32. 97-year-old female with confusion and weakness:



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- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

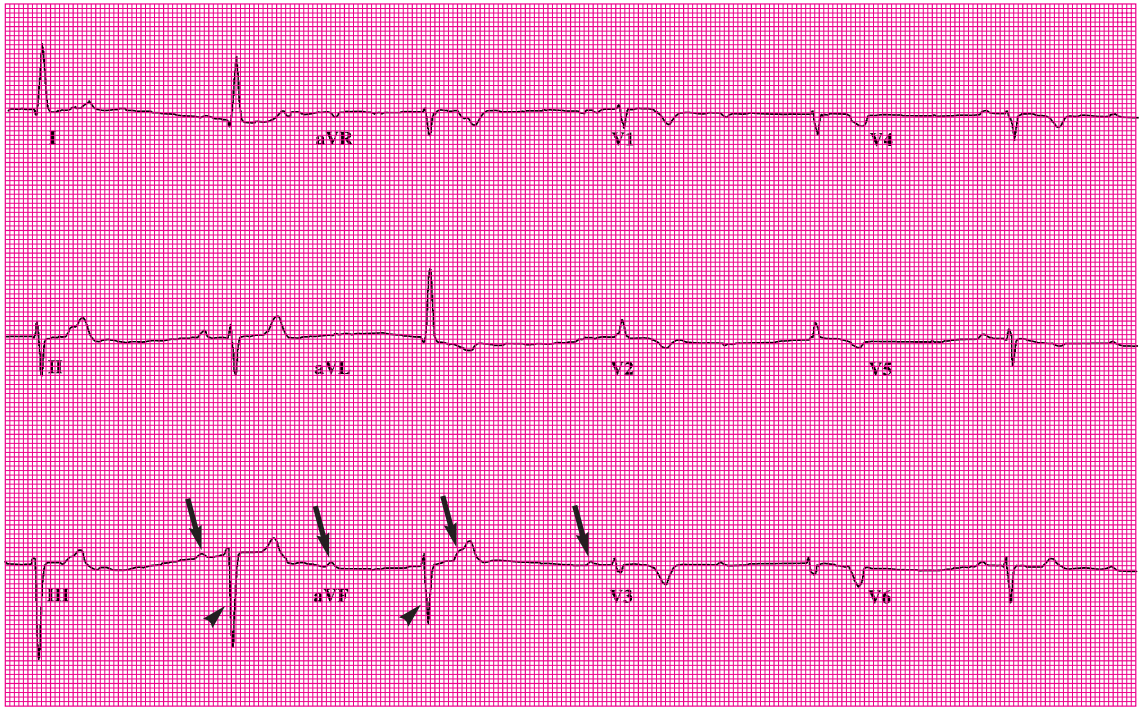
PACED RHYTHMS

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

ECG 32 was obtained from a 97-year-old female who presents with confusion and weakness. The ECG shows sinus bradycardia with complete heart block and a junctional escape rhythm at 35 BPM (arrows mark P waves; arrowheads mark junctional escape complexes). The junctional escape complexes show evidence for LVH (R wave in I $\geq$ 14 mm; R wave in aVL $\geq$ 12 mm; R wave in aVL + S wave in III > 20 mm), with associated ST-T changes, nonspecific IVCD, and left axis deviation. The sinus bradycardia, complete heart block, and junctional escape are all consistent with the diagnosis of sick sinus syndrome.

Codes:

09	Sinus bradycardia (< 60)
21	AV junctional escape complexes
33	AV block, 3^{o}
36	Left axis deviation (> -30^{o})
40	Left ventricular hypertrophy
57	ST and/or T wave abnormalities secondary to hypertrophy
89	Sick sinus syndrome



Questions: ECG 32

1. The distorted T wave best seen in the first beat in leads II, III, and aVF is due to:

   a. Repolarization abnormality
   b. Artifact
   c. Superimposed P wave

2. Is ventriculophasic sinus arrhythmia present in this ECG?

   a. Yes
   b. No

3. Can the diagnosis of left anterior fascicular block be made with certainty on this tracing?

   a. Yes
   b. No

4. Voltage criteria for LVH in the present ECG include:

   a. R wave in lead I
   b. R wave in lead aVL + S wave in lead III
   c. R wave in lead V_1 + S wave in lead V_6
   d. R wave in lead aVL
   e. S wave in lead aVR

5. Complete AV block can be diagnosed when the ventricular rate is faster than the atrial rate:

   a. True
   b. False

6. Causes of complete heart block include:

   a. Hyperkalemia
   b. Hypokalemia
   c. Endocarditis
   d. Acute MI
   e. Digitalis toxicity
   f. Lyme disease

7. In patients with complete congenital heart block, the site of block is typically the:

   a. AV node
   b. Bundle of His
   c. His-Purinkje system

Answers: ECG 32

1. Complete heart block occurs when atrial impulses consistently fail to reach the ventricles (i.e., atria and ventricles beat independently of each other). On ECG, the PP and RR intervals are constant, but the PR interval varies (since the P wave bares no constant relationship to the QRS complex). The P wave may precede, be buried within (and not visualized), or follow the QRS to deform the ST segment or T wave, as seen in several beats in the present tracing. The presence of bifid T waves in the first but not the second beats in leads II and III speaks against repolarization abnormality, which, if present in one beat, should be present in all beats in that lead. Artifact may deform the T wave, but this diagnosis is unlikely since the positive deflection appears in the early portion of the T wave and nowhere else on the ECG. (Answer: c)

2. Ventriculophasic sinus arrhythmia occurs in approximately 30% of cases of complete heart block, and is diagnosed when the PP interval containing a QRS complex is shorter than the PP interval not containing a QRS complex. In the present tracing, the PP intervals are regular, so ventriculophasic sinus arrhythmia is not present. (Answer: b)

3. Left anterior fascicular block (LAFB) is a diagnosis of exclusion, and can only be made with certainty when other conditions causing left axis deviation are absent, such as left ventricular hypertrophy (which is present on this tracing), inferior myocardial infarction, or chronic lung disease. (Answer: b)

4. Voltage criteria satisfied in this tracing include an R wave in lead I $\geq$ 14 mm, and an R wave in lead aVL $\geq$ 12 mm. (Answer: a, d)

5. The diagnosis of complete heart block requires that atrial and ventricular activity are independent of each other, and that the atrial rate is faster than the ventricular rate. When the ventricular rate exceeds the atrial rate, *AV dissociation* (as opposed to AV block) is said to be present; the ventricles may be refractory to incoming atrial impulses even though AV conduction is intact. (Answer: b)

6. Complete heart block may occur in advanced hyperkalemia, although death usually occurs from the development of ventricular tachyarrhythmias. In endocarditis, inflammation and edema of the septum and peri-AV nodal tissues may cause conduction failure and complete heart block; PR prolongation usually precedes this event. Five to fifteen percent of acute myocardial infarctions are complicated by complete heart block. In inferior MI, complete heart block is usually preceded by first-degree AV block or Type I second-degree AV block, typically occurs at the level of the AV node, is often transient (< 1 week), and is usually associated with a stable junctional escape rhythm (narrow QRS at a rate $\geq$ 40 BPM). In anterior MI, complete heart block occurs as a result of extensive damage to the left ventricle, and is typically preceded by Type II second-degree AV block or bifascicular block; mortality rates up to 70% may occur, and is usually due to pump failure rather than heart block per se. Digitalis toxicity is one of the most common causes of reversible complete AV block, and is usually associated with a junctional escape rhythm (narrow QRS) that is often accelerated. Lyme disease is caused by a tick-borne spirochete (Borrelia burgdorferi) and can also cause complete heart block. This disorder begins with a characteristic skin rash (erythema

chronicum migrans), and may be followed in subsequent weeks to months by joint, cardiac and neurological involvement. Cardiac involvement includes AV block, which can be partial or complete, usually occurs at the level of the AV node, and is sometimes accompanied by syncope. Other causes of complete heart block include infiltrative diseases of the myocardium (amyloid, sarcoid), myocardial contusion, acute rheumatic fever, aortic valve disease, and degenerative diseases of the conduction system (Lev's/Lenegre's disease). (Answer: a, c, d, e, f)

7. Complete congenital heart block usually occurs at the level of the AV node and is typically associated with a stable junctional escape rhythm. Very young patients often have escape rates > 55 BPM and usually do not require permanent pacing until age 25-30. (Answer: a)

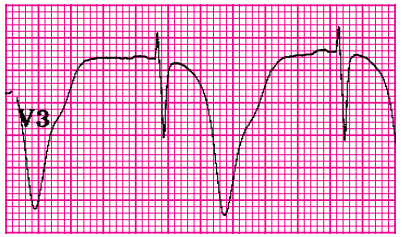
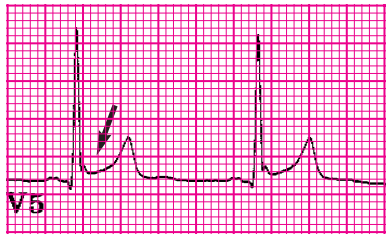
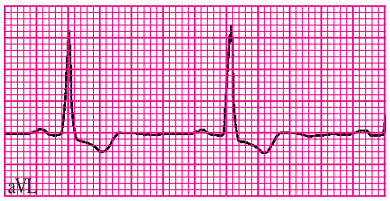
— Quick Review 32 —

AV junctional escape complexes	
• QRS complex occurs as a ____ phenomenon in response to decreased sinus impulse formation or conduction, or high-degree AV block	secondary
• Rate is typically ____ per minute	40-60
• Atrial mechanism may be sinus rhythm, paroxysmal atrial tachycardia, atrial flutter, or atrial fibrillation (true/false)	true
• QRS morphology is (similar to/different from) the sinus or supraventricular impulse	similar to
AV block, 3°	
• Atrial and ventricular rhythms are ____ of each other	independent
• Atrial rate is (faster/slower) than the ventricular rate	faster
Sick sinus syndrome	
• Marked sinus ____	bradycardia
• ____ arrest or ____ exit block	Sinus, sinoatrial
• Bradycardia alternating with ____	tachycardia
• Atrial fibrillation with ____ ventricular response preceded or followed by sinus bradycardia, sinus arrest, or sinoatrial exit block	slow
• Prolonged sinus node ____ time after atrial premature complex or atrial tachyarrhythmias	recovery
• AV junctional ____ rhythm	escape
• Additional conduction system disease is often present, including AV block, IVCD, and/or bundle branch block (true/false)	true

— POP QUIZ —

Pattern Recognition: ST & T Changes in Patients WITHOUT Chest Pain

Instructions: Match the following ECGs with all diagnoses/descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Early repolarization b. Normal variant c. Myocardial ischemia d. Can be treated with thrombolytic therapy e. Myocardial injury f. Total occlusion of right coronary artery g. Pericarditis h. Associated with PR segment depression i. CNS disorder j. Pseudo-ST depression k. Subtotal occlusion of left circumflex coronary artery l. Repolarization abnormality 2° to hypertrophy	Classic changes of *cerebral or subarachnoid hemorrhage* usually occur in the precordial leads, with large upright or deeply inverted T waves, prolonged QT interval (often marked), and prominent U waves. ST segment changes sometimes occur, including diffuse ST elevation (mimicking acute pericarditis), focal ST elevation (mimicking acute myocardial injury), or ST depression/abnormal Q waves (mimicking ischemia, MI). Almost any rhythm abnormality can be seen, including sinus tachycardia or bradycardia, junctional rhythm, VPCs, or VT. (Answer: i)
		*Normal variant early repolarization* results in elevated take-off of the ST segment at the junction between the QRS and ST segment (J junction), concave upward ST elevation ending with a symmetrical upright T wave (often of large amplitude), and distinct notching or slurring on the downstroke of the R wave. Early repolarization most commonly involves V_2-V_5 (sometimes II, III, aVF), and is not associated with reciprocal ST segment depression. Note: Some degree of ST elevation is present in the majority of young healthy individuals, especially in the precordial leads. (Answer: a, b)
		*Left ventricular hypertrophy (LVH)* results in tall R waves in the left precordial/limb leads, and ST and T wave changes opposite in direction to the major QRS deflection: ST depression in I, aVL, III, aVF, V_4-V_6, and ST elevation (< 0.5 - 3 mm) in V_1-V_3. Inverted T waves in I, aVL, V_4-V_6 and prominent upright or inverted U waves may also be seen. LVH repolarization abnormalities are often mistaken for myocardial ischemia (lateral wall) or myocardial infarction (anterior or inferior). Note: QRS voltage > 12 mm in aVL in this ECG meets criteria for LVH. (Answer: l)

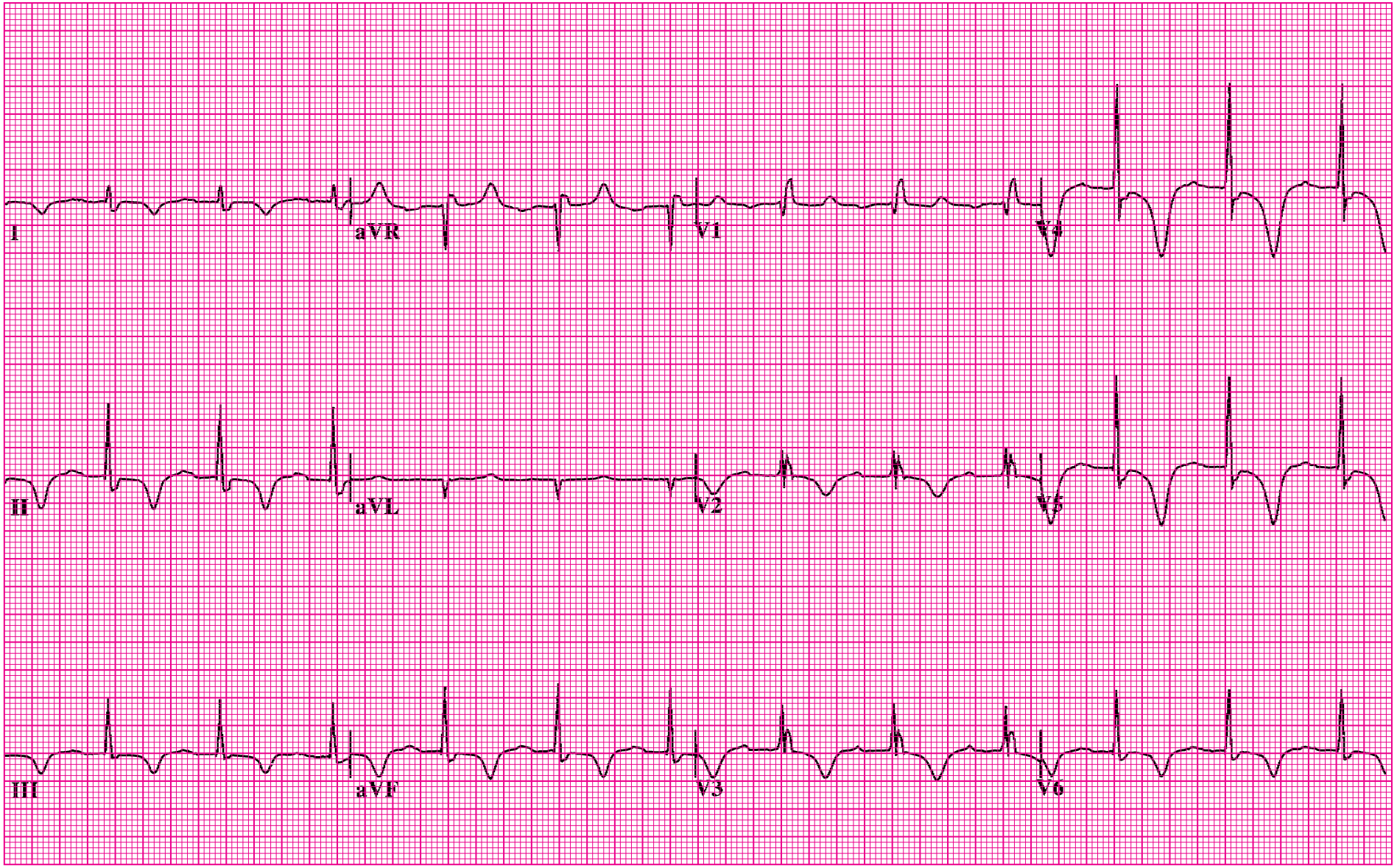
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the clinical disorder that best corresponds to each group of ECG features listed below (see score sheet for options)

ECG Features	Answer
• Amplitude of the entire QRS complex (R+S) < 10 mm in all precordial leads and < 5 mm in all limb leads	Low voltage
• Alternation in the amplitude and/or direction of the P, QRS and/or T waves	Electrical alternans
• Mean QRS axis $\geq 100^{o}$ • Dominant R wave V_1 • Secondary downsloping ST depression & T wave inversion in the right precordial leads • Right atrial abnormality	Right ventricular hypertrophy
• QRS duration $\geq$ 0.12 seconds • Onset of intrinsicoid deflection in leads I, V_5, V_6 > 0.05 seconds • Broad monophasic R waves in leads I, V_5, V_6, which are usually notched or slurred • Secondary ST & T wave changes opposite in direction to the major QRS deflection • rS or QS complex in the right precordial leads	LBBB, complete
• Upright P wave > 2.5 mm in leads II, III and aVF *or* > 1.5 mm in leads V_1 or V_2 • P wave axis $\geq$ 70 degrees	Right atrial abnormality
• Notched P wave with a duration $\geq$ 0.12 seconds in leads II, III or aVF, *or* • Terminal negative portion of the P wave in lead V_1 $\geq$ 1 mm deep and $\geq$ 0.04 seconds in duration	Left atrial abnormality

ECG 33. 48-year-old female with recent throat tightness and diaphoresis:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

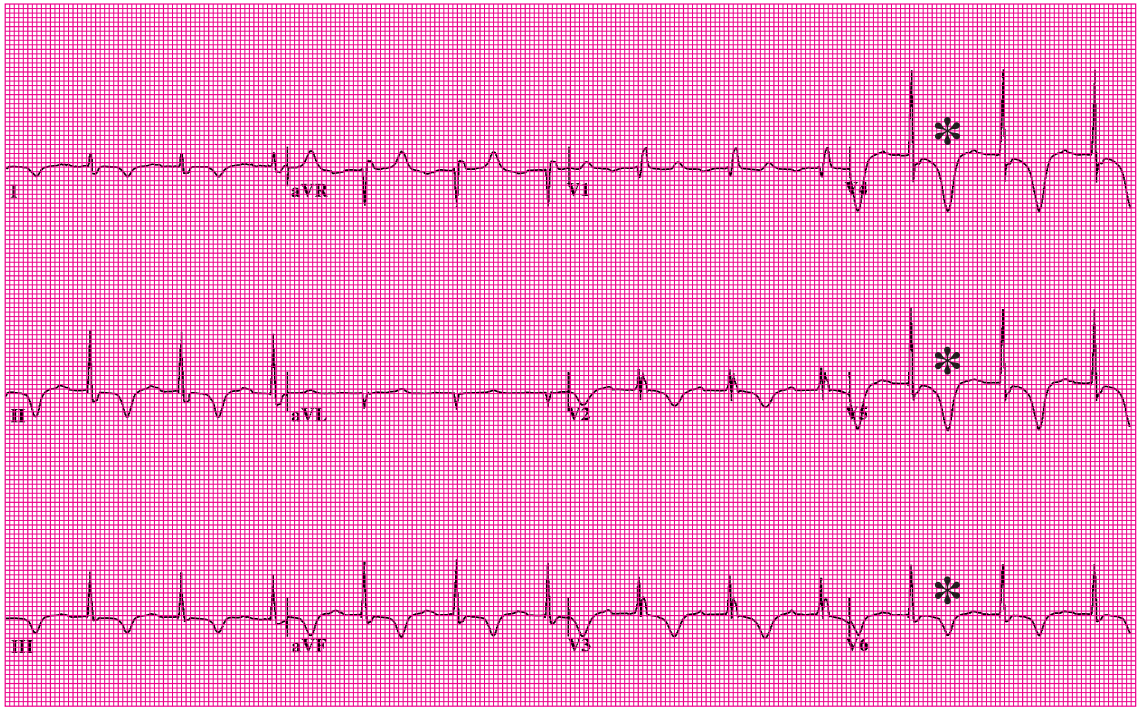
PACED RHYTHMS

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

ECG 33 was obtained in a 48-year-old female with recent throat tightness and diaphoresis. The ECG shows sinus rhythm with first-degree AV block, RBBB (widened rSR' complex in V_1 - V_2, and wide S waves in I, V_5, V_6), and striking T wave inversion and QT prolongation throughout most of the tracing (most prominent in the lateral precordial leads; asterisks). The deep T wave inversions in V_4-V_6, especially in a patient with chest pain, suggest myocardial ischemia or even a recent non-Q-wave myocardial infarction (note: secondary T waves of RBBB should be upright in these leads). Myocardial infarction should not be coded since abnormal Q waves are not present.

Codes:

07	Sinus rhythm
29	AV block, 1°
43	RBBB, complete
64	ST and/or T wave abnormalities suggesting myocardial ischemia
68	Prolonged QT interval



Questions: ECG 33

1. Deep T wave inversion in the precordial leads may be seen in:

   a. Non-Q-wave anterior MI
   b. Normal variant
   c. Hypertrophic cardiomyopathy
   d. Subarachnoid bleeding

2. QT prolongation can be seen in:

   a. CNS injury
   b. Hypercalcemia
   c. Quinidine effect
   d. Myocardial ischemia or injury

Answers: ECG 33

1. Deep T wave inversion in the precordial leads can be seen with non-Q-wave anterior MI, hypertrophic cardiomyopathy (especially the apical variant), subarachnoid hemorrhage, and following ventricular pacing. In contrast, normal variant T wave inversion is shallow, not deep, and is not associated with QT prolongation. (Answer: a, c, d)

2. QT prolongation can be seen with CNS injury (e.g., subarachnoid bleed), hypothermia, quinidine and other antiarrhythmic drugs, and myocardial ischemia or injury. Among electrolyte disturbances, hypocalcemia and hypokalemia result in QT prolongation; hypercalcemia and hyperkalemia result in QT shortening. (Answer: a, c, d)

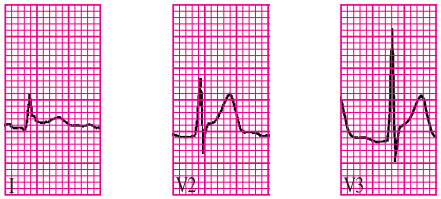
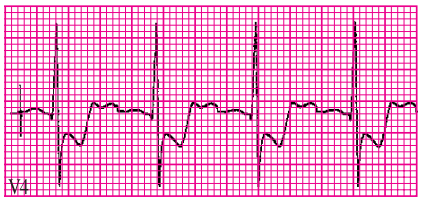
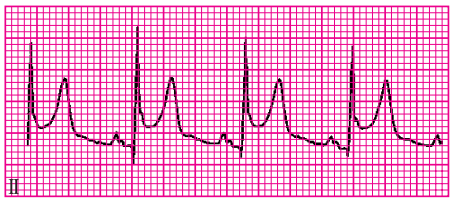
— Quick Review 33 —

ST and/or T wave abnormalities suggesting myocardial ischemia	
• Abnormally tall, symmetrical, (upright/inverted) T waves	inverted
• Horizontal or ____ ST segments with or without T wave inversion	downsloping
• Associated ECG findings:	
▸ QT interval is usually (normal/prolonged)	prolonged
▸ Reciprocal ____ wave changes may be evident	T
▸ Prominent U waves are often present and may be upright or inverted (true/false)	true
Prolonged QT interval	
• Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval	0.42-0.46 RR
• QT interval varies (directly/inversely) with heart rate	inversely
• The normal QT interval should be (less than/greater than) 50% of the RR interval	less than

— POP QUIZ —

Pattern Recognition: ST & T Changes in Patients WITH Chest Pain

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
 36 y.o. female with sharp chest pain relieved by sitting forward; similar ST changes in other leads	a. Early repolarization b. Normal variant c. Myocardial ischemia d. Can be treated with thrombolytic therapy e. Myocardial injury f. Total occlusion of right coronary artery g. Pericarditis h. Associated with PR segment depression i. CNS disorder j. Pseudo-ST depression k. Subtotal occlusion of left circumflex coronary artery l. Repolarization abnormality 2° to hypertrophy	*Acute pericarditis* results in upwardly concave ST segment elevation in almost all leads (except aVR) without reciprocal ST depression (except aVR). T wave inversion often occurs after ST segments return to baseline (in contrast to acute MI). Other findings may include sinus tachycardia, PR depression (early), or low voltage QRS and electrical alternans if pericardial effusion is present. Note: Focal pericarditis (e.g., post-pericardiotomy) results in regional (not diffuse) ST elevation. (Answer: g , h)
 72 y.o. female with GI bleed and chest pain		*Myocardial ischemia* results in horizontal or downsloping ST segments ± T wave inversion. T wave changes can be biphasic, symmetrical and deeply inverted, or upright and peaked (hyperacute), and may occur without significant ST segment depression. Prominent U waves (upright or inverted) and prolonged QT interval are sometimes seen. (Answer: c, k)
 65 y.o. male with 2 hrs of substernal chest pressure		*Myocardial injury* results in acute ST segment elevation with upward convexity in leads over the area of injury. ST & T wave changes evolve, and T waves invert before ST segments return to baseline. Reciprocal ST depression in noninfarct leads is common (unlike pericarditis). Hyperacute (upright and peaked) T waves are sometimes evident prior to ST segment elevation. Acute posterior wall injury results in ST segment *depression* with upright T waves in V_1-V_3 ± prominent R waves. (Answer: d, e, f)

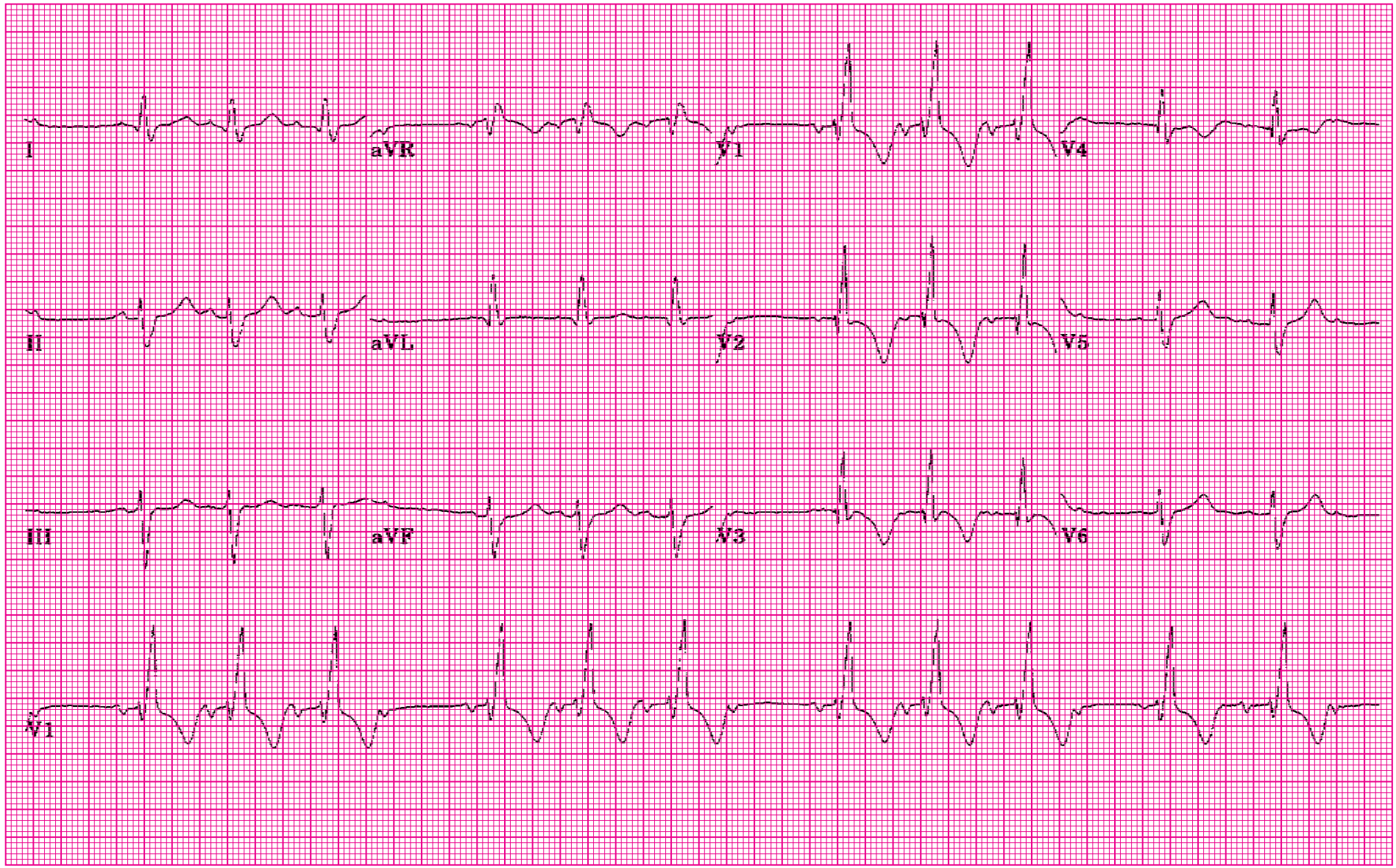
— POP QUIZ —

Differential Diagnosis: ST Segment

Instructions: Determine whether each diagnosis below is associated with: (a) ST elevation; (b) ST depression; or (c) both.

Diagnosis	Answer
Hyperkalemia	ST depression. Tall, peaked T waves and QRS widening are common. (Answer: b)
Hypokalemia	ST depression. Flattened T waves and prominent U waves are common. (Answer: b)
Myocardial injury	ST elevation (convex upward) in area of injury; ST depression in reciprocal leads. Q waves absent. (Answer: c)
Myocardial ischemia	ST depression (horizontal or downsloping). T waves usually inverted; Q waves absent. Prinzmetal's (variant) angina presents with ST elevation. (Answer: c)
Myocardial infarction	ST elevation (with reciprocal ST depression) or primary ST depression (non-ST-elevation MI or posterior MI). In the days-to-weeks post-MI, ST elevation without reciprocal ST depression can be seen in pericarditis or ventricular aneurysm. (Answer: c)
Digoxin	Sagging ST segment depression with upward concavity. T waves may be flattened, inverted or biphasic. QT shortening and PR prolongation may also occur. (Answer: b)
Early repolarization	ST elevation (concave upward) ending with a symmetrical (often tall) upright T wave, most often in V_2-V_6. No reciprocal ST depression. Distinct notching/slurring on downstroke of R wave. (Answer: a)
Intracranial hemorrhage	Can present with diffuse ST elevation (mimicking pericarditis), focal ST elevation (mimicking acute myocardial injury), or ST depression. Large upright or deeply inverted T waves, prolonged QT interval, and prominent U waves are common, especially in the precordial leads. (Answer: c)
Ventricular aneurysm	ST elevation $\geq$1 mm persisting four or more weeks after acute MI in leads with abnormal Q waves. (Answer: a)
Left ventricular hypertrophy (LVH)	ST and T wave displacement opposite to major QRS deflection: ST depression (upwardly concave) and T wave inversion when the QRS is mainly positive (leads I, V_5, V_6); subtle (< 1 mm) ST elevation and upright T waves when the QRS is mainly negative (leads V_1, V_2). (Answer: c)
Left bundle branch block (LBBB)	Secondary ST and T wave changes opposite in direction to major QRS deflection: ST depression and T wave inversion in leads I, V_5, V_6; ST elevation and upright T waves in leads V_1, V_2. (Answer: c)

ECG 34. 37-year-old male with palpitations:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

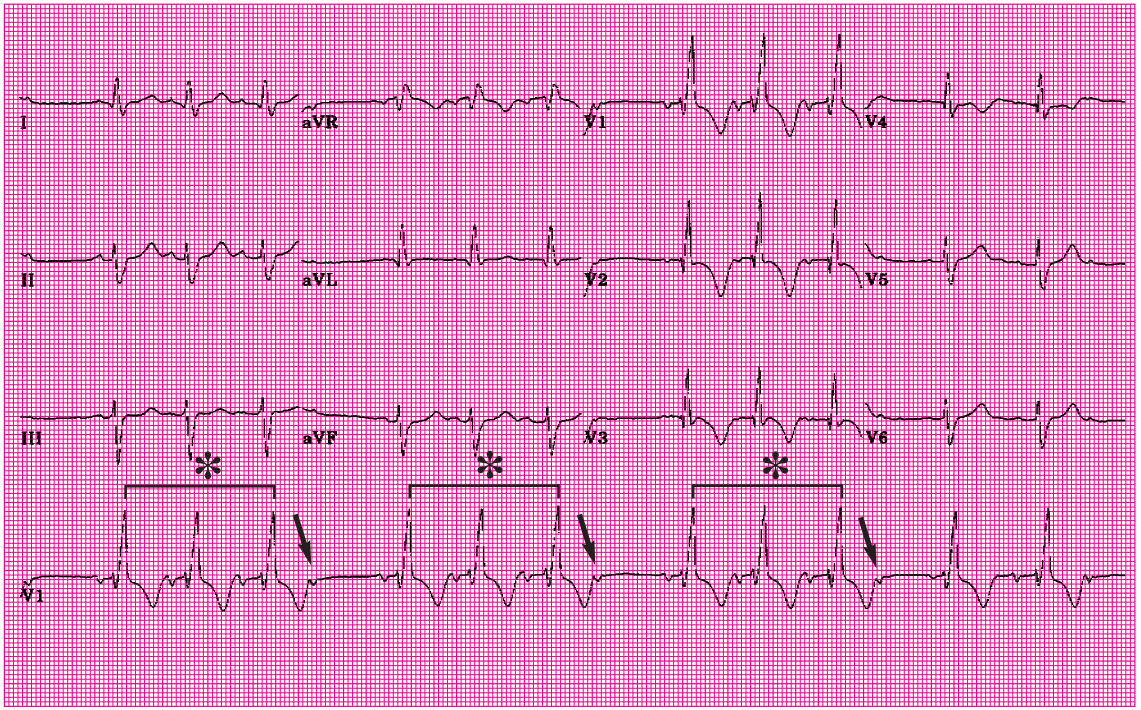
PACED RHYTHMS

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

ECG 34 was obtained from a 37-year-old male with palpitations. The ECG shows sinus rhythm at a rate of 94 beats/minute with occasional nonconducted P waves (arrows) resulting in grouped beating (asterisk). There is gradual prolongation of the PR interval leading up to the dropped beat, consistent with Mobitz type I - second-degree AV block. The conduction ratio is 4:3 (4 P waves for every 3 QRS complexes). Also noted are left atrial enlargement, RBBB with secondary repolarization changes (T wave inversions in leads V_1-V_3), left anterior fascicular block (QRS axis = –50°), and a prolonged QT interval (QTc = 0.48 seconds).

Codes:

06	Left atrial abnormality/enlargement
07	Sinus rhythm
30	AV block, 2°- Mobitz type I (Wenckebach)
43	RBBB, complete
45	Left anterior fascicular block
68	Prolonged QT interval



Questions: ECG 34

1. The diagnosis of left anterior fascicular block (LAFB) requires:

   a. QRS axis between -30° and -90°
   b. QRS axis between -45° and -90°
   c. QRS prolongation ≥ 0.11 seconds
   d. No other factor responsible for left axis deviation

2. Left anterior fascicular block (LAFB) can result in a false positive diagnosis of:

   a. Inferior myocardial infarction
   b. Anterior myocardial infarction
   c. Left ventricular hypertrophy
   d. Right ventricular hypertrophy

3. Bifascicular block (RBBB + LAFB) with Mobitz Type I second-degree AV block is a strong indication for a permanent pacemaker in an asymptomatic patient:

   a. True
   b. False

Answer: ECG 34

1. LAFB results in a mean QRS axis between -45° and -90°, and requires that no other cause of left axis deviation is present (e.g., LVH, inferior MI, left bundle branch block). QRS prolongation ≥ 0.11 seconds is not a diagnostic feature of LAFB, although the QRS is often slightly prolonged (0.08-0.10 seconds). (Answer: b, d)

2. In addition to left axis deviation, LAFB can produce diminished (sometimes absent) R waves in leads III and aVF, low anterior forces, and a tall R wave in lead aVL, which may be mistaken for inferior MI, anterior MI, and LVH, respectively. (Answer: a, b, c).

3. Bifascicular block with Mobitz I second-degree AV block is not a strong indication for a pacemaker in an asymptomatic person. In the setting of symptomatic bradycardia, a pacemaker would be an acceptable option. (Answer: b)

— Quick Review 34 —

AV block, 2° - Mobitz Type I (Wenckebach)	
• Progressive prolongation of the ____ interval and shortening of the ____ interval until a P wave is blocked	PR RR
• RR interval containing the nonconducted P wave is (less than/equal to/greater than) the sum of two PP intervals	less than
• Results in ____ beating due to the presence of nonconducted P waves	group
RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ____ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 > ____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ____	I, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
• RBBB (does/does not) interfere with the ECG diagnosis of ventricular hypertrophy or Q wave MI	does not
Left anterior fascicular block	
• ____ axis deviation with a mean QRS axis between ____ and ____ degrees	left -45, -90
• (qR/rS) complex in leads I and aVL	qR
• (qR/rS) complex in lead III	rS
• Normal or slightly prolonged QRS duration (true/false)	true
• No other cause for left axis deviation should be present (true/false)	true
• Poor R wave progression is (common/uncommon)	common

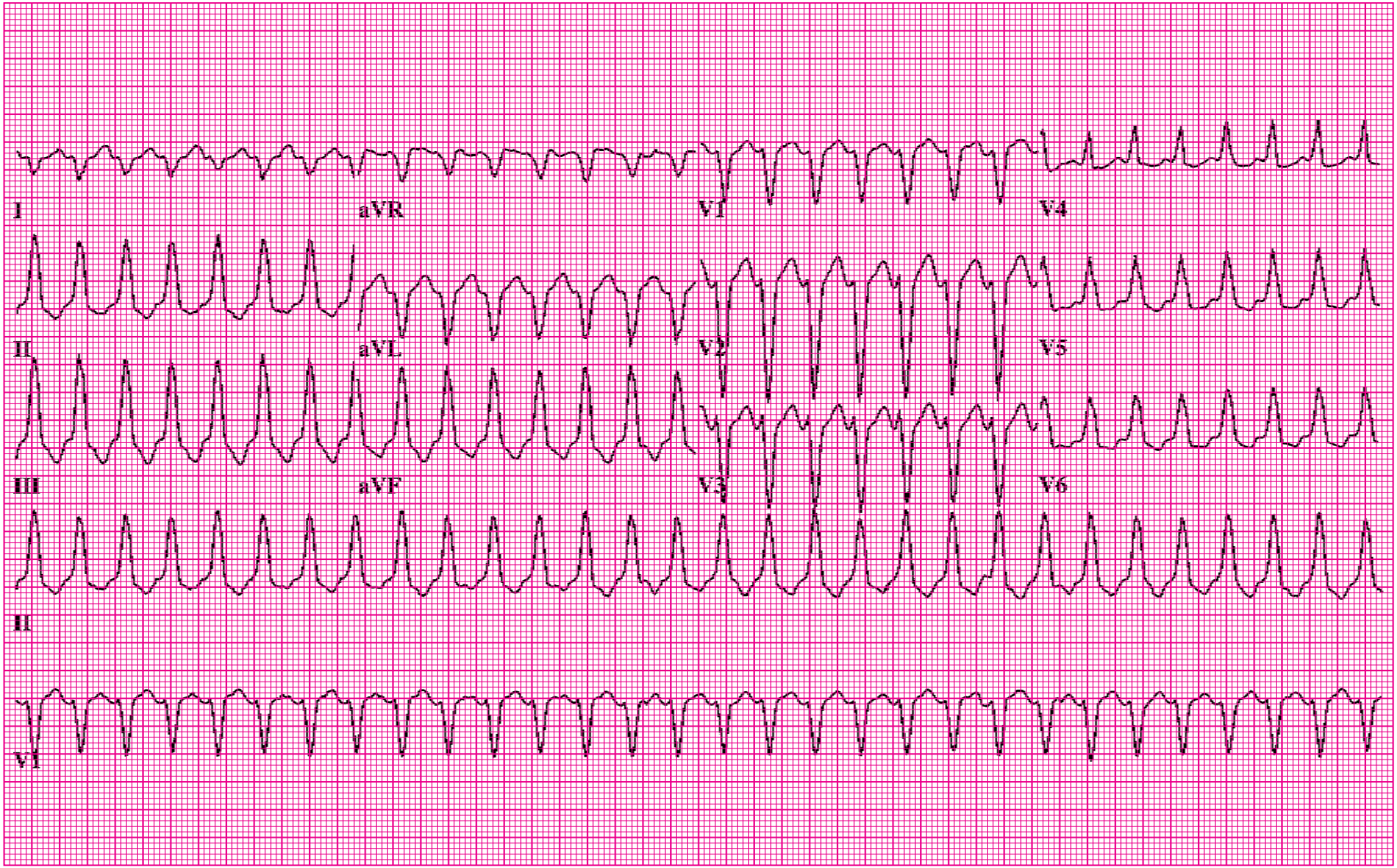
— POP QUIZ —

Find The Mistake

Instructions: Identify the incorrect ECG feature(s) for each ECG diagnosis listed below.

ECG Diagnosis and Features	Mistake
Left anterior fascicular block • Left axis deviation (-45 to -90 degrees) • qR complex in lead I, aVL, and III • Normal or slightly prolonged QRS duration • No other cause for left axis deviation present	There is an rS complex (not a qR complex) in lead III
Left posterior fascicular block • Right axis deviation (+100 to +180 degrees) • S wave in lead I and Q wave in lead III • Normal or slightly prolonged QRS duration • Other cause for right axis deviation may be present	LPFB should not be diagnosed when another cause for right axis deviation exists
RBBB, complete • QRS duration ≥ 0.12 seconds • Secondary R wave (R') in lead V_1 is usually shorter than the initial R wave • Onset of intrinsicoid deflection in V_1 and V_2 > 0.05 sec • ST segment depression and T wave inversion in V_1, V_2 • Wide slurred S wave in leads I, V_5, V_6 • QRS axis is usually rightward	R' is usually taller (not shorter) than the initial R wave in V_1, and QRS axis is usually normal (not rightward)
LBBB, complete • QRS duration ≥ 0.12 seconds • Onset of intrinsicoid deflection in I, V_5, V_6 > 0.05 sec • Broad monophasic R waves in leads I, V_5, V_6, which are usually notched or slurred • Secondary ST & T wave changes in same direction as the major QRS deflection • rS or QS complex in the right precordial leads	Secondary ST & T wave changes are in opposite (not the same) direction to the major QRS deflection

ECG 35. 24-year-old female with post-partum cardiomyopathy and palpitations:



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- ☐ 04. Artifact

P WAVE ABNORMALITIES

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- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 49. Nonspecific intraventricular conduction disturbance
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Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
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- ☐ 76. Hypercalcemia
- ☐ 77. Hypocalcemia
- ☐ 78. Atrial septal defect, secundum
- ☐ 79. Atrial septal defect, primum
- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
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- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
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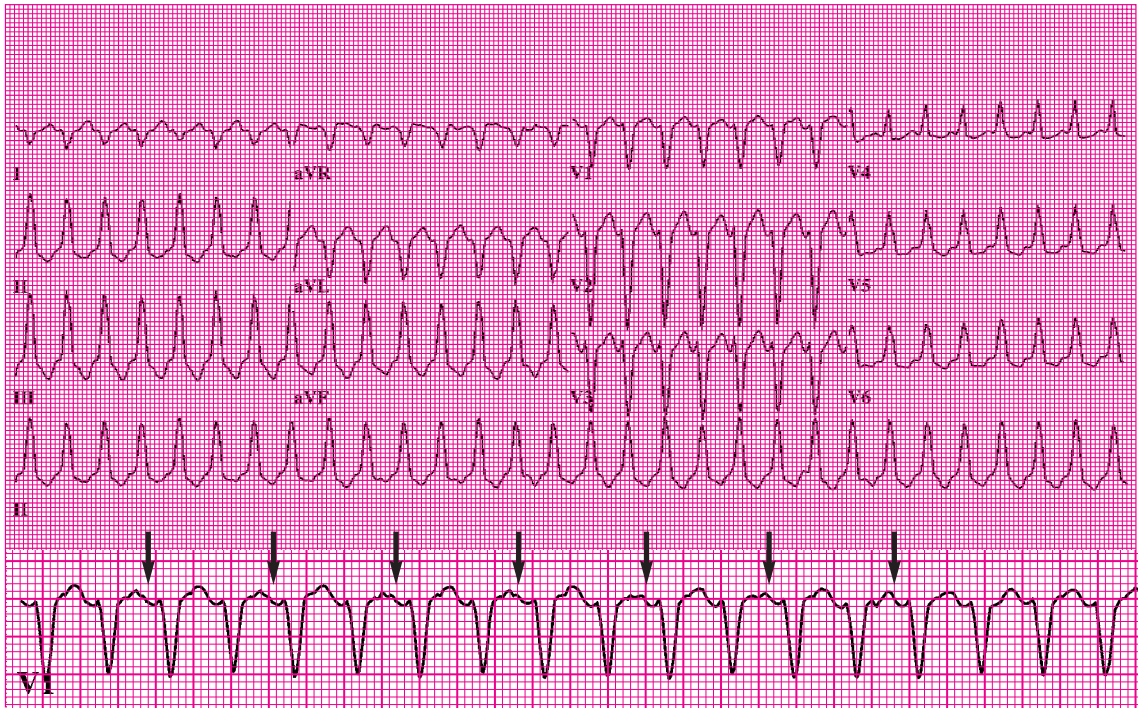
PACED RHYTHMS

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

ECG 35 was obtained in a 24-year-old female with post-partum cardiomyopathy under evaluation for palpitations. The ECG shows an underlying sinus rhythm at 90 beats/minute (arrows mark the P waves) and a wide complex tachycardia at 178 beats/minute consistent with ventricular tachycardia (VT) with AV dissociation. Ventricular tachycardia morphology shows a LBBB pattern with right axis deviation, localizing the site of origin of the VT to the right ventricular outflow tract. (Note: Right-sided VT results in a LBBB *pattern*, not a true LBBB).

Codes:

25 Ventricular tachycardia (≥ 3 consecutive complexes)
35 AV dissociation
37 Right axis deviation (> +100°)



Questions: ECG 35

1. In the setting of a wide QRS tachycardia, ECG findings that favor the diagnosis of ventricular tachycardia over SVT with aberrancy include:

   a. R' is taller than the R wave when an RSR' complex is present in V_1
   b. Capture beats
   c. QRS duration < 0.16 seconds if LBBB morphology is present (assuming the QRS is narrow during sinus rhythm)
   d. Some positive and some negative QRS deflections in the precordial leads
   e. AV dissociation

2. Ventricular tachycardia always manifests a QRS duration ≥ 0.12 seconds

   a. True
   b. False

Answers: ECG 35

1. In the setting of wide QRS tachycardia, the diagnosis of ventricular tachycardia is favored over SVT with aberrancy when: QRS morphology is similar to VPCs seen in an earlier tracing; when the tachycardia is initiated by VPCs; AV dissociation, capture beats, and/or fusion beats are present; QRS duration exceeds 0.14 seconds if RBBB morphology is present (or 0.16 seconds if LBBB morphology is present); QRS deflections in the precordial leads are concordant (all positive or all negative); or the R wave is taller than the R' wave in lead V_1. (Answer: b, e)

2. Although rare, if the ventricular focus is high in the septum (i.e., immediately below the bundle of His), ventricular tachycardia can present with a relatively narrow QRS complex. (Answer: b)

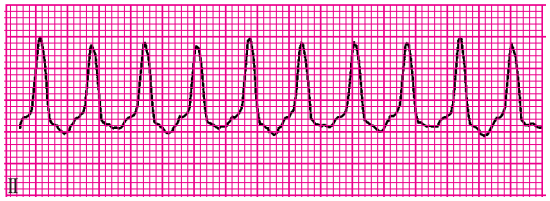
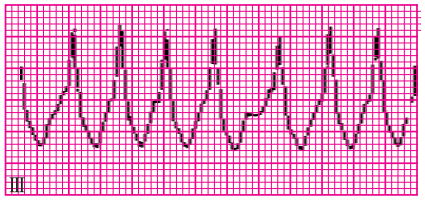
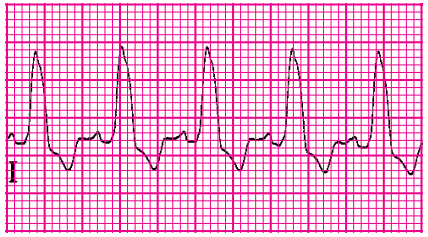
— Quick Review 35 —

Ventricular tachycardia	
• Rapid succession of three or more premature ventricular beats at a rate > ____ per minute	100
• RR intervals are usually regular but may be irregular (true/false)	true
• (Abrupt/gradual) onset and termination are evident	Abrupt
• AV ____ is common	dissociation
• Look for ventricular ____ complexes and ____ beats as markers for VT	capture, fusion
AV dissociation	
• Atrial and ventricular rhythms are ____ of each other	independent
• Ventricular rate is (</≥) than the atrial rate	≥

— POP QUIZ —

Rhythm Recognition: Wide QRS Tachycardia

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Ventricular tachycardia. Description: Regular (sometimes irregular) ventricular rhythm with ≥ 3 consecutive beats at a rate > 100 per minute. Onset and termination are usually abrupt. AV dissociation is common, and retrograde atrial activation and ventricular capture complexes sometimes occur. Seen in organic heart disease, hypokalemia, hyperkalemia, hypoxia, acidosis, drug toxicity, mitral valve prolapse, and occasionally in normals.
	Answer: Atrial fibrillation with Wolff-Parkinson-White (WPW) syndrome. Description: Irregular supraventricular rhythm with absent P waves, irregularly irregular RR intervals often at a rate > 200 per minute (can be < 200), and QRS complexes that vary in width (but are generally wide). Classic fibrillatory ("f") waves may be seen at slower rates (but may not be evident at faster rates, as in this ECG). The 12-lead ECG during sinus rhythm shows a short PR interval with initial slurring of the QRS (delta wave). The delta wave is due to pre-excitation of the ventricles from conduction across the bundle of Kent, an accessory AV pathway which bypasses the AV node (and AV nodal conduction delay). Variance in QRS width is due to different degress of fusion between electrical wavefronts conducted down the accessory pathway and AV node.
	Answer: Sinus tachycardia with bundle branch block. Description: Sinus tachycardia may present as a wide QRS tachycardia in the setting underlying bundle branch block (as in this ECG) or functional (rate-related) aberrancy. Although P waves are sometimes seen with ventricular tachycardia, they are either due to retrograde atrial activation (inverted in leads II, III) or are supraventricular in origin (sinus or atrial) and manifest AV dissociation (varying PR intervals). Fusion complexes and ventricular capture complexes are consistent with ventricular tachycardia, not sinus tachycardia with bundle branch block.

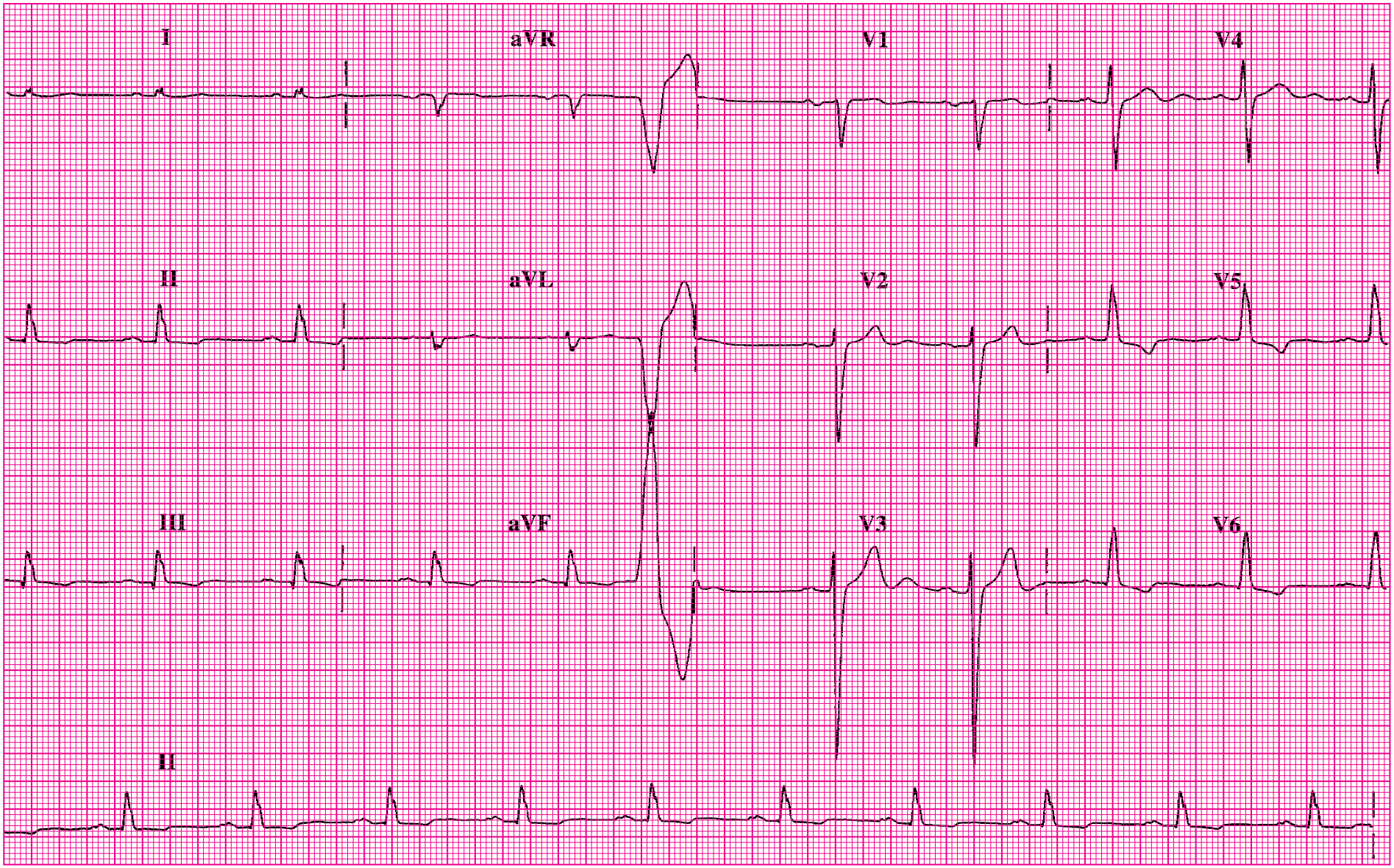
— POP QUIZ —

VT or Not VT: That is the Question

Instructions: In the setting of a wide QRS tachycardia, decide whether the ECG findings below favor ventricular tachycardia or SVT with aberrancy

ECG Feature	VT or SVT with Aberrancy
QRS morphology similar to VPCs	VT
Tachycardia initiated by APCs	SVT
AV dissociation absent	SVT
Capture beats present	VT
Fusion beats absent	SVT
QRS duration during tachycardia > 0.14 seconds if RBBB morphology or > 0.16 seconds if LBBB morphology (assuming QRS is narrow during sinus rhythm)	VT
QRS deflection in precordial leads are all positive or negative (concordance)	VT
RSR' in V_1: R wave shorter than R'	SVT

ECG 36. 48-year-old man with aortic stenosis:



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- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
- ☐ 65. ST and/or T wave abnormalities suggesting myocardial injury
- ☐ 66. ST and/or T wave abnormalities suggesting electrolyte disturbances
- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

SUGGESTED CLINICAL DISORDERS

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

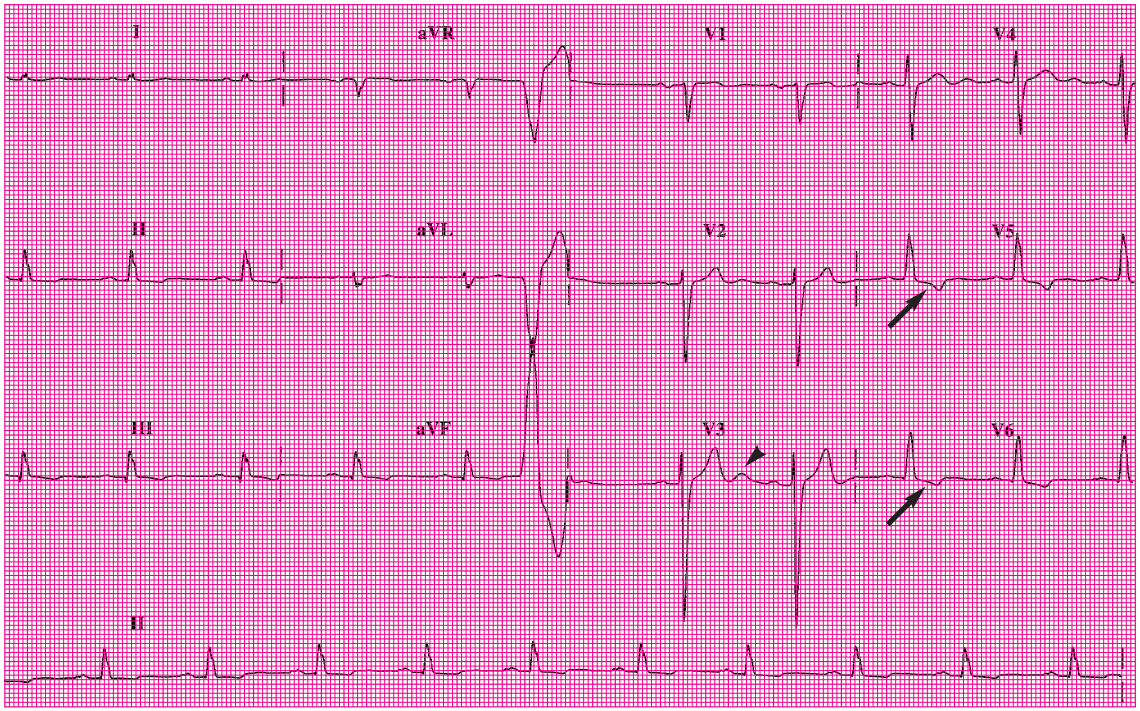
PACED RHYTHMS

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

ECG 36 was obtained in a 48-year-old male with aortic stenosis. The ECG shows a sinus rhythm with a single ventricular premature complex. LVH is apparent with an R wave in aVL + S wave in V_3 > 28 mm (Cornell criteria). Repolarization abnormalities (downsloping ST depression and asymmetrical T wave inversion) secondary to LVH are present (arrows) as well as prominent U waves (arrowhead), a common finding in LVH.

Codes:

07	Sinus rhythm
23	Ventricular premature complexes
40	Left ventricular hypertrophy
67	ST and/or T wave abnormalities secondary to hypertrophy
69	Prominent U waves



Questions: ECG 36

1. Findings in this ECG that can be attributed to LVH include:

   a. Left atrial abnormality
   b. Prominent U wave
   c. ST segment depression and T wave inversion
   d. Intraventricular conduction delay
   e. Poor R wave progression
   f. Absent Q wave in V_5
   g. ST elevation in V_3

2. The differential diagnosis for prominent U waves includes:

   a. Hypokalemia
   b. Hyperkalemia
   c. Digitalis
   d. Quinidine
   e. Amiodarone
   f. Central nervous system disorders
   g. LVH

3. Anatomical LVH is more likely to be present when repolarization (ST and T wave) changes accompany voltage criteria:

   a. True
   b. False

4. Which of the following ECG criteria is most specific (i.e., fewest false-positives) for the diagnosis of LVH:

   a. R in V_5 or V_6 + S in V_1 > 35 mm
   b. R in aVL > 12 mm
   c. Any R + S in the precordial leads > 45 mm
   d. R in aVL + S in V_3 > 28 mm (20 mm in females)

5. Which of the following ECG criteria is the most sensitive (i.e., fewest false-negatives) for the diagnosis of LVH:

   a. R in V_5 or V_6 + S in V_1 > 35 mm
   b. R in aVL > 12 mm
   c. Any R + S in the precordial leads > 45 mm
   d. Left axis deviation > – 30°
   e. R in aVL + S in V_3 > 28 mm (> 20 mm in females)

6. Factors/conditions reducing the sensitivity for the diagnosis of LVH by voltage criteria include:

   a. Obesity
   b. Thin body habitus
   c. Severe COPD
   d. Pericardial or pleural effusion
   e. Coronary artery disease
   f. Pneumothorax
   g. Sarcoidosis or amyloidosis of the heart
   h. Severe right ventricular hypertrophy
   i. Left bundle branch block
   j. Left anterior fascicular block

Answers: ECG 36

1. The ECG diagnosis of left ventricular hypertrophy (LVH) is based primarily on the presence of large amplitude QRS complexes generated from the hypertrophic left ventricle. LVH also frequently results in non-voltage based changes, some of which are evident in this ECG tracing. ***Left atrial abnormality***, while not a direct manifestation of LVH, increases the probability that LVH is present, and is given 3 points in the point score system for LVH by Romhilt and Estes (Table 1). A ***prominent U wave*** is often seen in the right precordial leads (V_2, V_3) but is neither sensitive nor specific for the diagnosis of LVH. ***ST and T wave changes*** are very common in advanced stages of LVH; when present, the ECG specificity for the diagnosis of anatomical LVH is increased: In the left precordial leads (V_4 - V_6), these changes typically consist of downsloping ST segment depression with a slight upward concavity, and asymmetrical T wave inversion, with more gentle sloping of the descending limb compared to the ascending limb. In the right precordial leads (V_1 - V_3), reciprocal ST segment elevation and tall T waves are often seen, which, in conjunction with ***poor R wave progression*** (or even Q waves or QS complexes) may mimic anteroseptal or anterior MI. In the limb leads, ST and T wave changes appear in a direction opposite from the main QRS forces (i.e., in leads with largely positive QRS complexes, ST depression and T wave inversion are present; in leads with largely negative QRS complexes, ST elevation and tall T waves are present). Other changes in this ECG consistent with LVH include ***delayed onset of intrinsicoid deflection*** (onset of QRS to peak R wave ≥ 0.05 seconds; due to a delay in intraventricular conduction), and ***inferior Q waves***, the mechanism of which is unknown. Findings not present in this tracing but often evident in LVH include ***notching of the QRS complex*** and ***left axis deviation***. (Answer: all)

2. Mild hyperkalemia (5.5 - 6.5 mEq/L) can result in T waves that are tall, peaked, and symmetrical, and shortening of the QT interval. Moderate hyperkalemia (6.5 - 8.0 mEq/L) can result in a decrease in P wave and R wave amplitudes, lengthening of the PR interval and QRS duration, and depression or elevation of the ST segment; ventricular premature complexes may also be seen. In severe hyperkalemia, the P wave may be undetectable and the QRS complex markedly widened, giving the appearance of a sine wave. Rhythms may include sinoventricular rhythm (no P waves apparent), idioventricular rhythm, accelerated idioventricular rhythm, ventricular tachycardia, ventricular fibrillation, and asystole. U waves, however, are not typically seen. (Answer: all except b)

3. The sensitivity and specificity for the ECG diagnosis of anatomical LVH depend on the ECG criteria. Although ST and T wave changes "typical" for LVH may be caused by other conditions (e.g., myocardial ischemia), their presence increases the specificity for the diagnosis of LVH by voltage criteria. (Answer: a)

4. An R wave in aVL ≥ 12 mm in the absence of left anterior fascicular block is highly specific for the diagnosis of LVH. However, only 11% of individuals with LVH. meet this criteria (i.e., poor sensitivity). **CAVEAT:** Since the presence of left anterior fascicular block results in large leftward forces (R waves) in leads I and aVL, voltage criteria using these leads (i.e., R wave in aVL > 12 mm; R wave in lead I + S wave in lead III > 28 mm; R wave in aVL + S wave in V_3 > 28 mm [Cornell

criteria]) will overestimate the diagnosis of LVH. (Answer: b)

5. Sensitivity for identification of LVH is highest (35-50%) for the Cornell criteria (R wave in aVL + S wave in V_3 > 28 mm in males or > 20 mm in females) and lower (10-30%) for other criteria. All standard voltage criteria for LVH are limited by low sensitivity. (Answer: e)

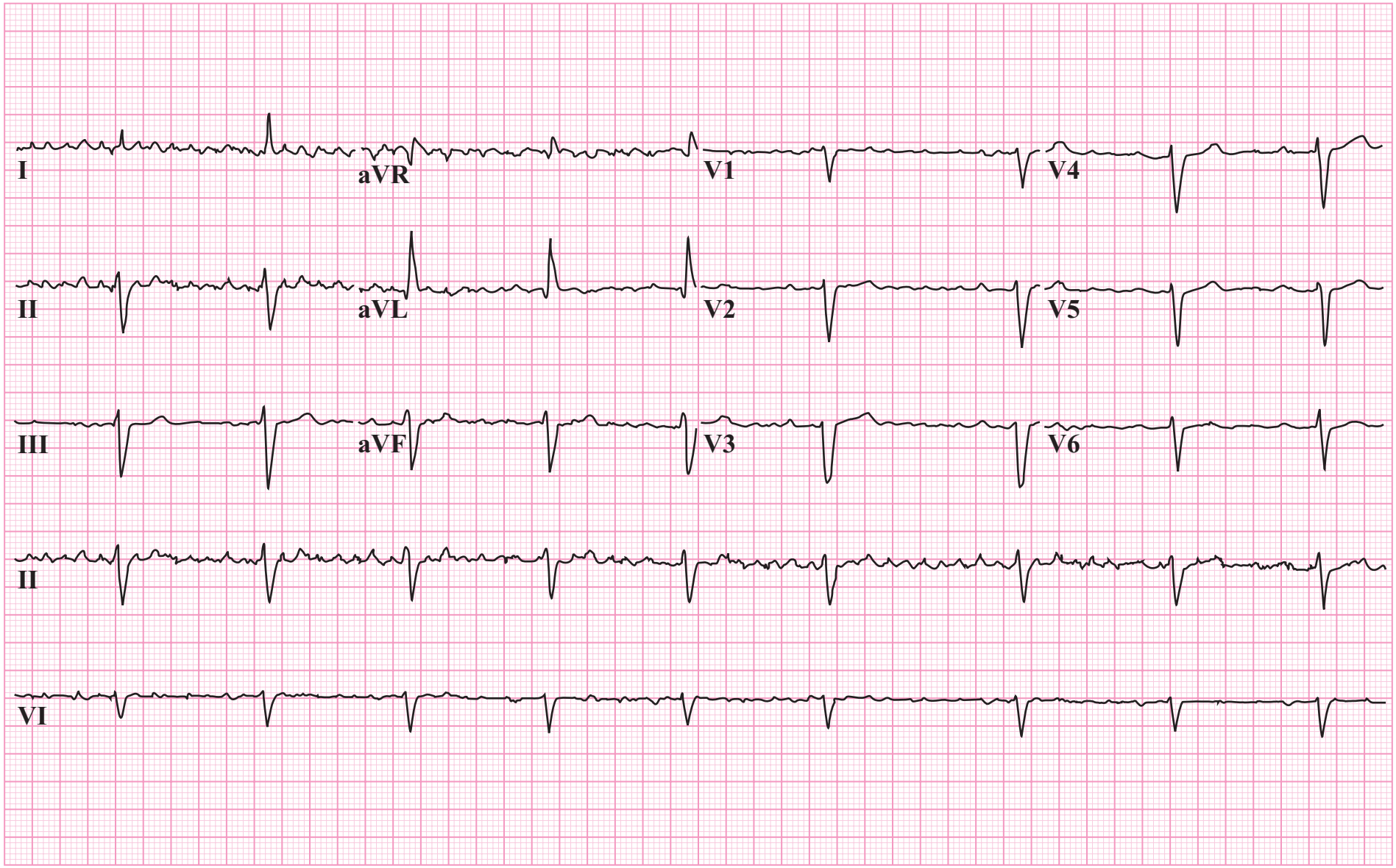
6. The amplitude of the QRS as recorded by the surface electrocardiogram (and the sensitivity for the diagnosis of LVH by voltage criteria) is often decreased by conditions that increase the amount of body tissue (obesity), air (COPD, pneumothorax), fluid (pericardial or plural effusion), or fibrous tissue (coronary artery disease, sarcoid or amyloid of the heart) between the myocardium and ECG electrodes. Severe RVH can also underestimate the ECG diagnosis of LVH by cancelling prominent QRS forces from the thickened LV. Left bundle branch block may also reduce QRS amplitude as well. In contrast, thin body habitus and the presence of left anterior fascicular block may increase QRS amplitude in the absence of LVH, thus decreasing the specificity of the voltage criteria. (Answer: all except b and j)

— Quick Review 36 —

LVH by both voltage and ST-T segment abnormalities

- Voltage criteria for LVH and one or more ST-T abnormalities:
  - ST segment and T wave deviation in (same/opposite) direction to the major deflection of QRS — opposite
  - ST segment (elevation/depression) in leads I, aVL, III, aVF, and/or V_4-V_6 — depression
  - Subtle (< 1-2 mm) ST (elevation/depression) in leads V_1-V_3 — elevation
  - Inverted ____ waves in leads I, aVL, V_4-V_6 — T
  - (Absent/prominent) U waves — prominent

ECG 37. 53-year-old asymptomatic male:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
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- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
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- ☐ 30. AV block, 2°-Mobitz type I (Wenckebach)
- ☐ 31. AV block , 2°-Mobitz type II
- ☐ 32. AV block, 2:1
- ☐ 33. AV block, 3°
- ☐ 34. Wolff-Parkinson-White pattern
- ☐ 35. AV dissociation

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- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
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- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
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- ☐ 59. Posterior (age recent or acute)
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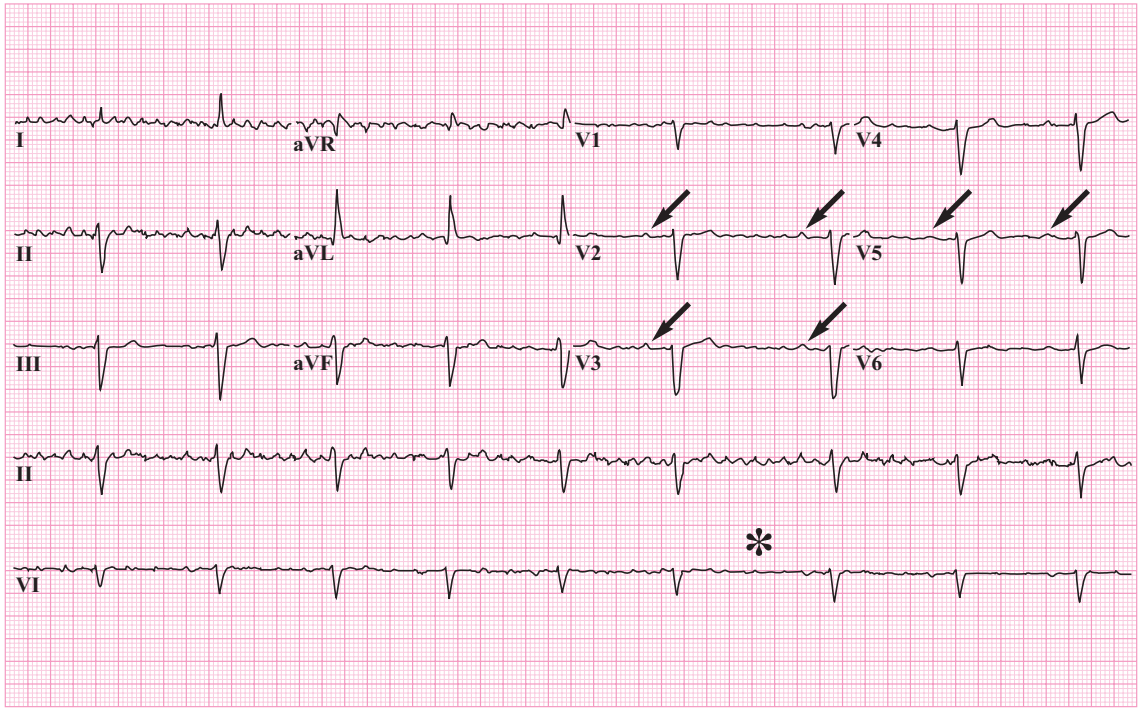
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 37 was obtained in an asymptomatic 53-year-old male. The ECG shows an irregular, undulating baseline at approximately at 8 cycles per second (cps). Upon close inspection of the precordial leads, sinus P waves are present (arrows), and the PR interval is 220 msec, consistent with first-degree AV block. The heart rate is 54 beats/minute, with sinus arrhythmia causing lengthening of the RR interval between the 6th and 7th beats (asterisk). Left axis deviation and a nonspecific intraventricular conduction disturbance (QRS duration = 120 msec) are also present. Left anterior fascicular block should not be coded as the cause of left axis deviation in this tracing since, by definition, the QRS duration in LAFB is 80-100 msec. The undulating baseline in the current tracing has a cycle length of 7-9 cps, characteristic of a physiologic tremor.

Codes:

04	Artifact
08	Sinus arrhythmia
09	Sinus bradycardia (< 60)
29	AV block 1°
36	Left axis deviation (> –30°)
49	Nonspecific intraventricular conduction disturbance



Questions: ECG 37

1. Match the following QRS durations with the associated conduction disturbance:

a.	0.08-0.10 seconds	1.	Incomplete LBBB
b.	≥ 0.12 seconds	2.	Complete LBBB
c.	0.11 seconds	3.	Left anterior fascicular block
d.	0.09-0.12 seconds	4.	Nonspecific intraventricular conduction defect

2. In sinus arrhythmia, the P wave morphology and axis are usually normal:

   a. True
   b. False

Answers: ECG 37

1. (Answer: a-3, b-2, c-4, d-1)

2. In sinus arrhythmia, phasic changes in the PP interval occur in response to respirations; the cycle is usually gradual but can sometimes change abruptly. By definition, the longest/shortest PP intervals vary by more than 160 msec or 10% of the PP interval. The P wave morphology and axis are usually normal in sinus arrhythmia, although left/right atrial enlargement can coexist with sinus arrhythmia. (Answer: a)

— Quick Review 37 —

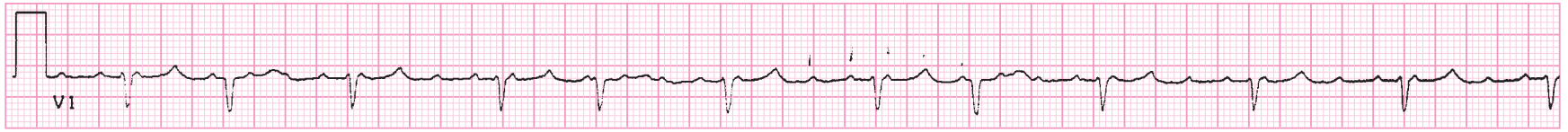
Artifact	
Commonly due to tremor	
• Parkinson's tremor simulates atrial ____ with a rate of _____ per second	flutter 4-6
• Physiologic tremor rate is _____ per second	7-9
• Tremor is most prominent in (limb/precordial) leads	limb
Sinus arrhythmia	
• (Sinus/nonsinus) P wave	Sinus
• Longest and shortest PP intervals vary by > ____ seconds or 10%	0.16
• Sinus arrhythmia differs from "ventriculophasic" sinus arrhythmia, the latter of which occurs in the setting of ____	heart block
Left axis deviation (> –30°)	
• Mean QRS axis between ___ degrees and ___ degrees	-30, -90

— POP QUIZ —

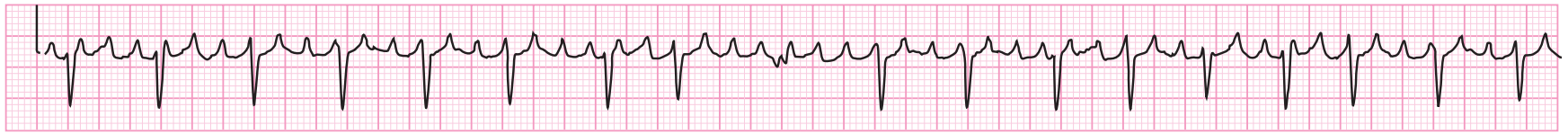
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

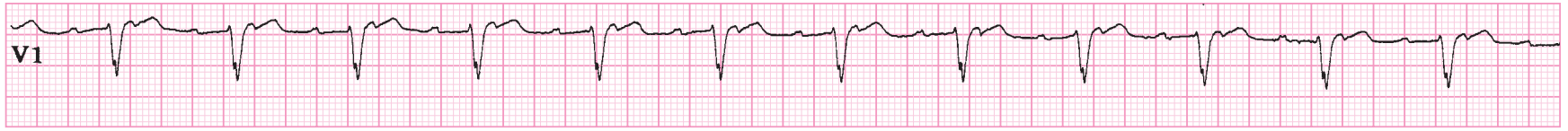
A.



B.



C.



D.

Answer: While all of the tracings show rapid atrial activity, A, B and D represent atrial flutter with atrial rates of 250–350 bpm. The imposter is tracing C, which shows atrial tachycardia with 2:1 AV block. The atrial rate of 160 bpm is much slower than that of atrial flutter, and the ventricular rate of 80 bpm is exactly one-half the atrial rate, which helps to identify the presence of 2:1 AV block. In this case, digitalis toxicity should be considered (although approximately one fourth of cases of atrial tachycardia with block occur in the absence of digitalis use or toxicity).

Common Dilemmas in ECG Interpretation

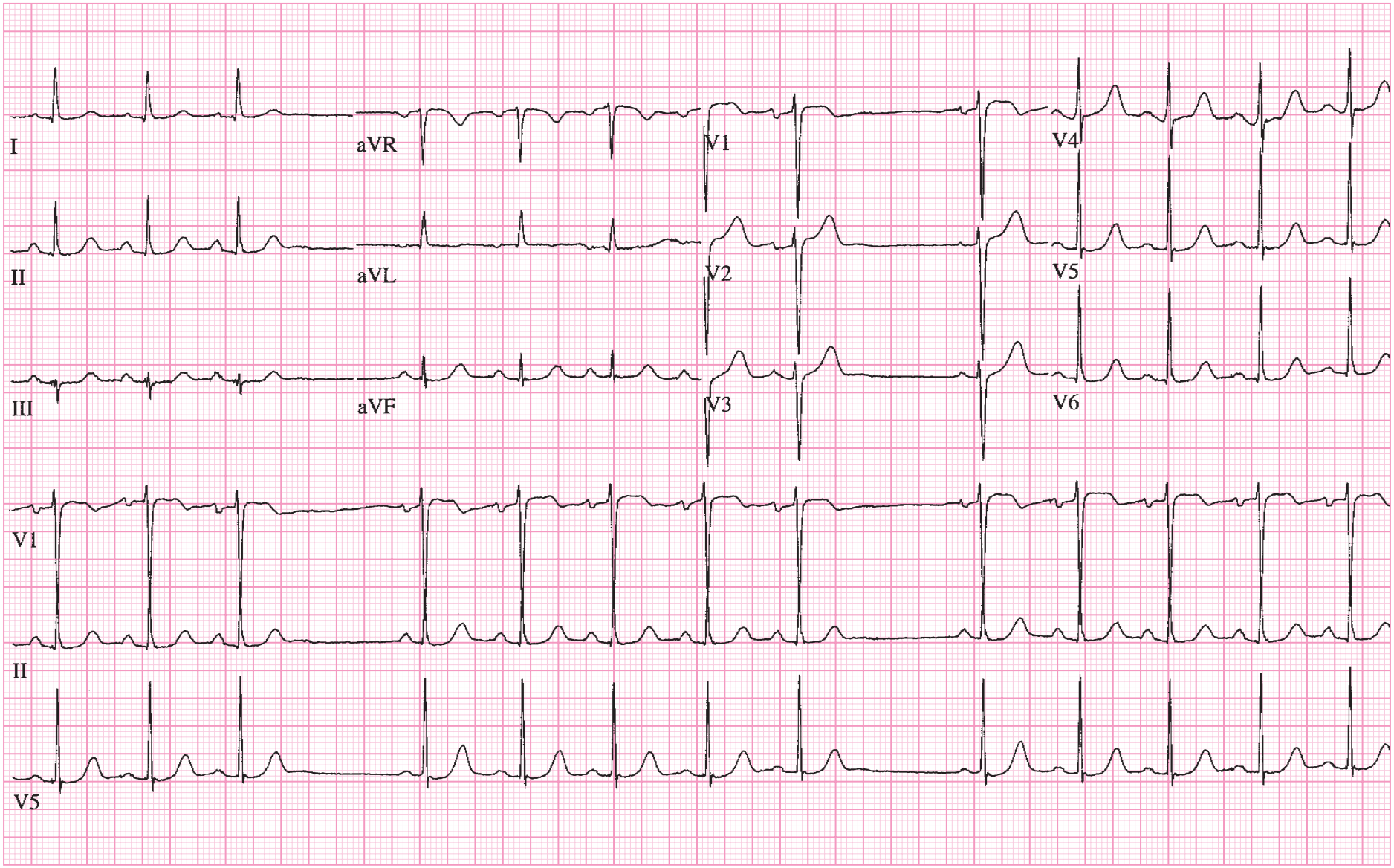
Problem

Ischemic-looking ST segment elevation is present without pathological Q waves in a patient with chest pain. Should acute myocardial infarction be coded?

Recommendation

No. Convex upward ST segment elevation without pathological Q waves should be coded as item 65 (ST and/or T abnormalities suggesting myocardial injury). Clinically, this usually represents the early stages of acute infarction or transient coronary spasm or occlusion. Nevertheless, in the absence of pathological Q waves (or pathological R waves in the case of posterior infarction), acute myocardial infarction should not be coded.

ECG 38. 87-year-old female with dizziness:



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- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
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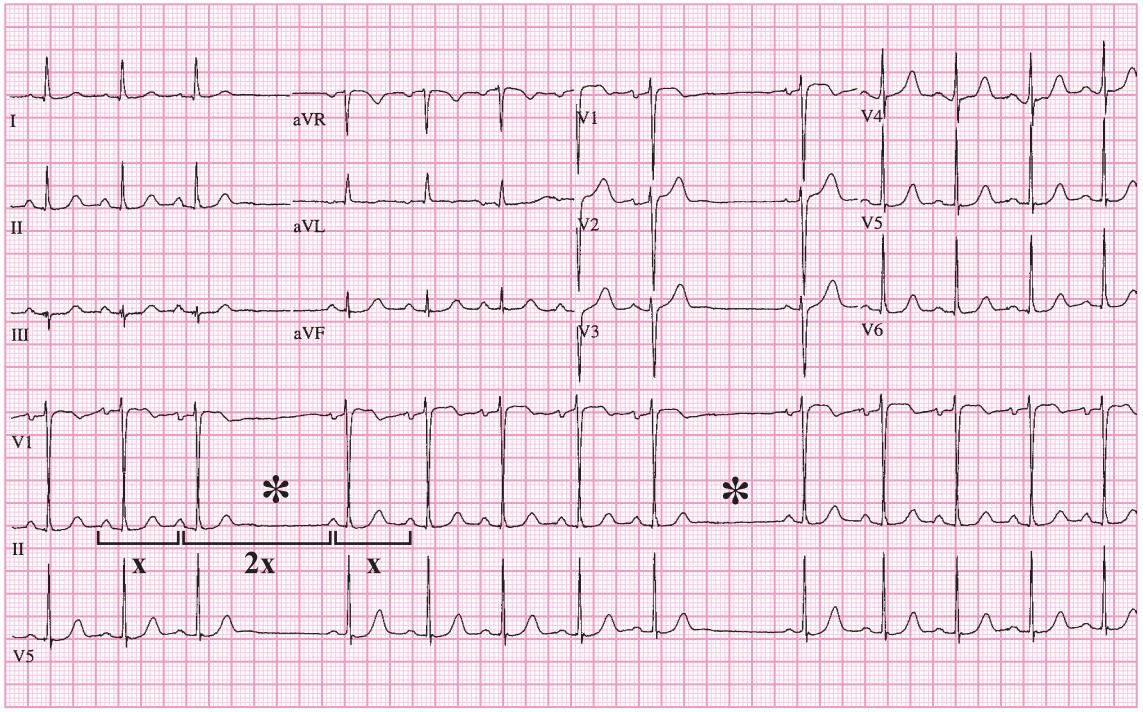
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 38 was obtained from a 87-year-old female with dizziness. The ECG shows sinus rhythm at 77 beats/minute with brief sinus pauses (asterisk) in which the PP interval is twice the basic PP interval. This finding indicates the presence of sinoatrial exit block.

Codes:

07	Sinus rhythm
12	Sinoatrial exit block



Questions: ECG 38

1. ECG manifestations of Mobitz II sinoatrial (SA) exit block include:

   a. Lengthening of the PR interval
   b. Sinus pauses that are a multiple of normal PP interval
   c. Narrowing of the QRS complex
   d. Shortening of the PP interval

2. The normal corrected QT intervals for heart rates of 60 and 80 beats/minute are ___ ± 0.04 seconds and ___ ± 0.04 seconds, respectively:

   a. 0.38; 0.42
   b. 0.44; 0.40
   c. 0.42; 0.38
   d. 0.40; 0.38

Answers: ECG 38

1. In ***Mobitz II*** SA exit block, sinus impulses occur at a constant rate but occasionally fail to capture the atria, resulting in intermittent absence of a P wave. The typical ECG finding is a PP pause that is a multiple (2x, 3x, etc.) of the basic PP interval. ***Mobitz I*** SA exit block is suggested by the presence of recurring PP pauses ("group beating") with PP intervals less than two times the basic PP interval. SA exit block is often a component of sick sinus syndrome, and is an important consideration when evaluating the etiology of a PP pause. (Answer: b)

2. The easiest method of calculating the corrected QT interval is to assume a normal QT interval of 0.40 ± 0.04 seconds for a heart rate of 70 BPM, then add (or subtract) 0.02 seconds for every 10 BPM change in heart rate below (or above) 70 BPM. Thus, at heart rates of 60 BPM and 80 BPM, the corrected QT intervals = 0.42 ± 0.04 seconds and 0.38 ± 0.04 seconds, respectively. At a heart rate of 50 BPM, the corrected QT interval = (0.40) + (2 x 0.02) = 0.44 ± 0.04 seconds. (Answer: b)

— Quick Review 38 —

Sinoatrial (SA) exit block	
First-degree: Conduction of sinus impulses to the atrium is (normal/delayed), but ____:1 response is maintained	delayed, 1
• First-degree SA exit block (is/is not) detectable on the surface ECG	is not
Second-degree: Some sinus impulses fail to ____the atria	capture
• Type I (Mobitz I):	
▸Sinus P wave (true/false)	true
▸"____ beating" with:	group
(1) (Shortening/lengthening) of the PP interval prior to absent P wave	shortening
(2) (Constant/variable) PR interval	constant
(3) PP pause < ____ normal PP interval	2
• Type II (Mobitz II): Constant PP interval followed by a pause that (is/is not) a multiple (2x, 3x, etc.) of the normal PP interval	is
Third-degree:	
• Complete failure of ____ conduction	sinoatrial
• Cannot be differentiated from complete ____	sinus arrest

— POP QUIZ —

Find The Mistake

Instructions: Identify the incorrect ECG feature(s) for each ECG diagnosis listed below.

ECG Diagnosis and Features	Mistake
Hypothermia • Sinus bradycardia • PR, QRS, and QT prolonged • Osborne ("J") wave: late upright terminal deflection of QRS complex • Atrial fibrillation in 50-60%	Atrial fibrillation occurs in 50-60%
CNS disorder • "Classic changes" usually occur in the limb leads ▸ Large upright or deeply inverted T waves ▸ Prolonged QT interval (often marked) ▸ Prominent U waves • Other changes: ▸ ST segment mimicking acute pericarditis or injury ▸ ST depression may also occur ▸ Abnormal Q waves mimicking MI ▸ Almost any rhythm abnormality, including sinus tachycardia or bradycardia, junctional rhythm, VPCs, ventricular tachycardia	"Classic changes" usually occur in the precordial (not limb) leads
Myxedema • Low QRS voltage in all leads • Sinus bradycardia • Peaked T waves • PR interval may be prolonged • Frequently associated with pericardial effusion • Electrical alternans may occur	T waves are flattened or inverted (not peaked)

Common Dilemmas in ECG Interpretation

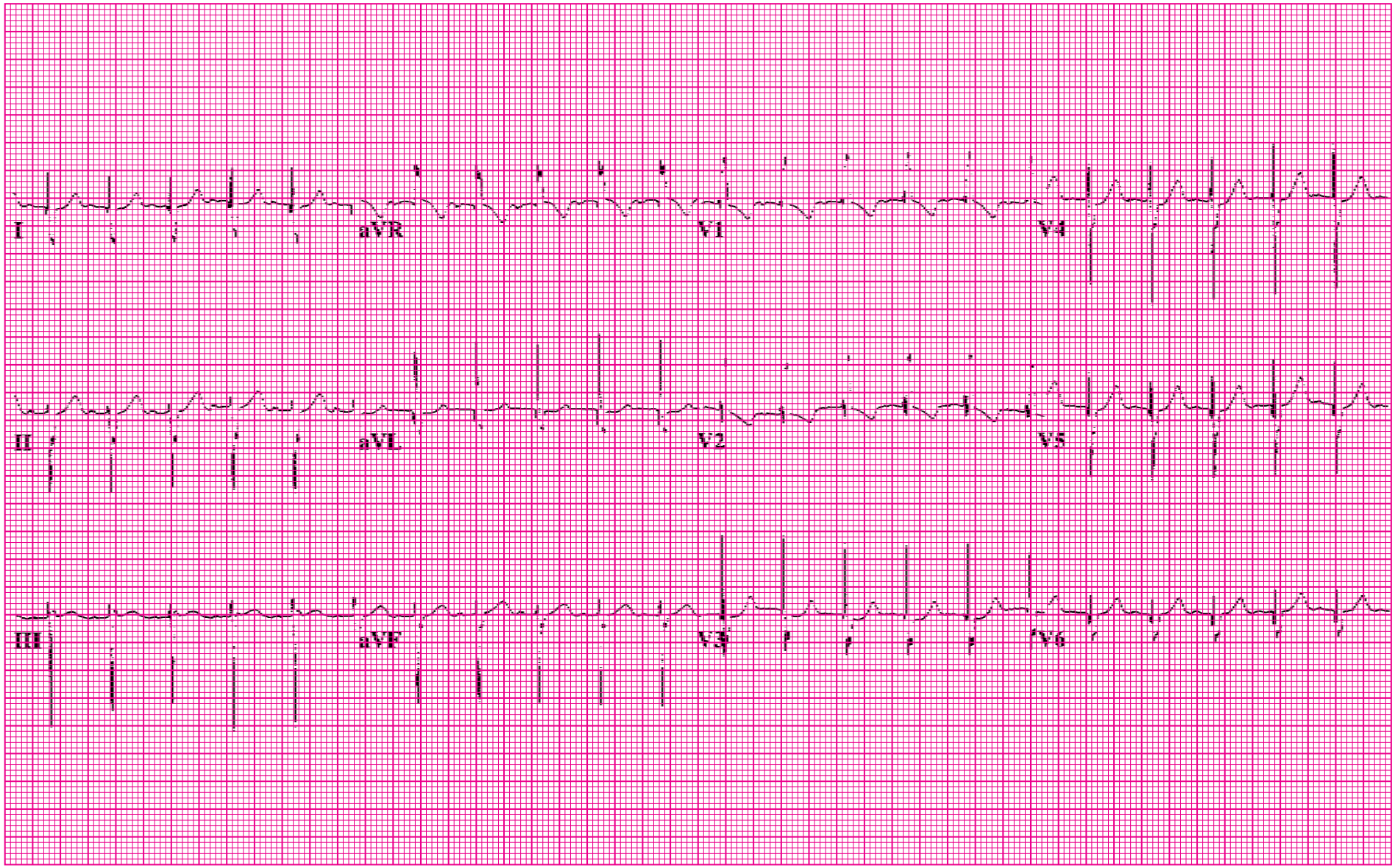
Problem

Should left axis deviation be coded when left anterior fascicular block (LAFB) is present? Similarly, should right axis deviation be coded when left posterior fascicular block (LPFB) is present?

Recommendation

No. A description of axis is redundant in LAFB or LPFB. If LAFB or LPFB is present, it is not necessary to code axis.

ECG 39. 1-year-old male with a heart murmur:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

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- ☐ 29. AV block, 1°
- ☐ 30. AV block, 2°-Mobitz type I (Wenckebach)
- ☐ 31. AV block , 2°-Mobitz type II
- ☐ 32. AV block, 2:1
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- ☐ 35. AV dissociation

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

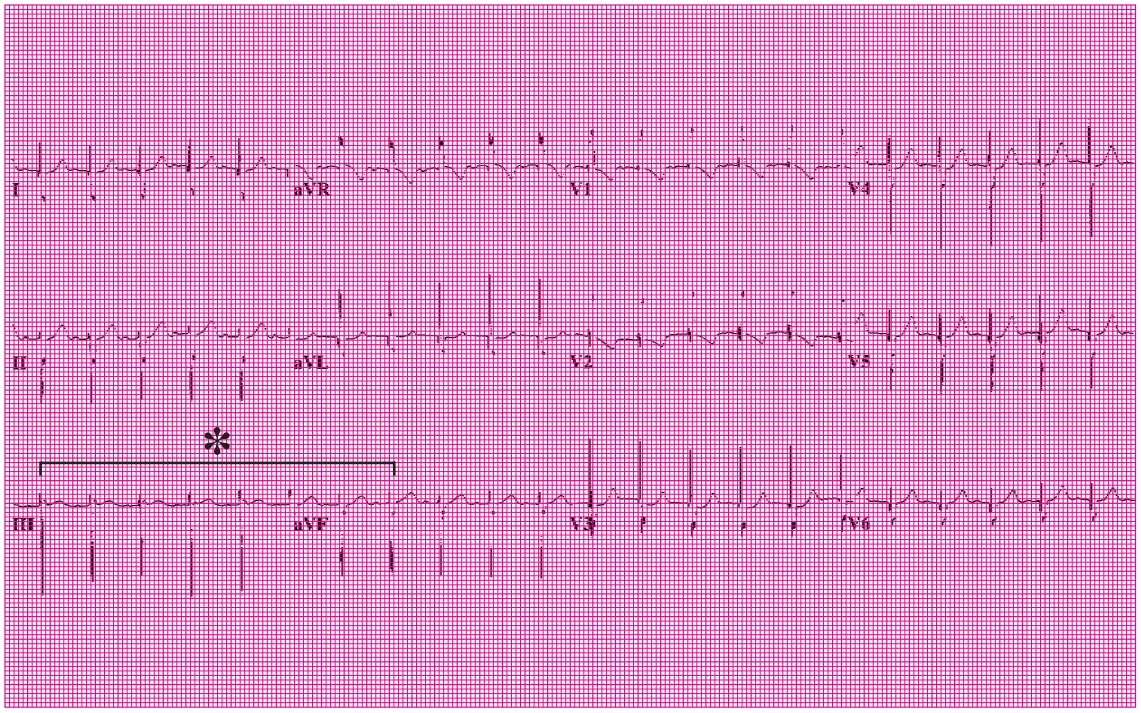
PACED RHYTHMS

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

ECG 39 was obtained in a 1-year-old male with a heart murmur. The ECG shows sinus tachycardia with incomplete RBBB and left axis deviation. This constellation of findings is typical for an ostium primum atrial septal defect. Electrical alternans is also present (asterisk). This child subsequently underwent cardiac surgery to repair a large primum ASD.

Codes:

10	Sinus tachycardia
36	Left axis deviation (> – 30°)
38	Electrical alternans
44	RBBB, incomplete
79	Atrial septal defect, primum



Questions: ECG 39

1. Primum atrial septal defect results in ___ axis deviation:

   a. Right
   b. Left

2. ECG findings suggestive of right ventricular hypertrophy (RVH) include:

   a. Left axis deviation
   b. Right atrial abnormality
   c. R > S in V_1
   d. R in aVL > 12 mm
   e. Downsloping ST segments and T wave inversion in V_1-V_3

Answers: ECG 39

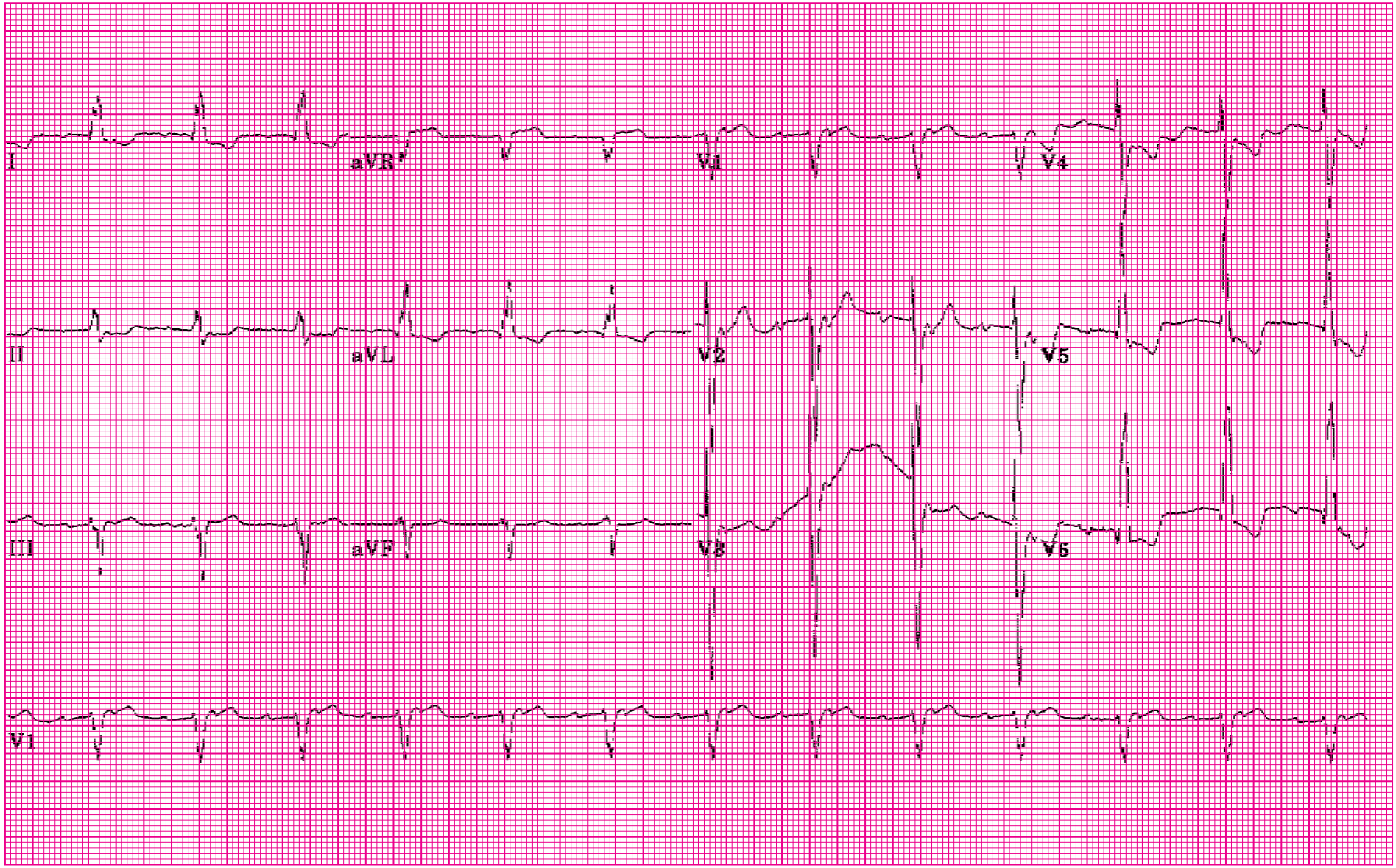
1. Primum atrial septal defect is associated with an rSr' complex in lead V_1 and left axis deviation. In 15-40% of cases, first-degree AV block is present. Advanced cases may demonstrate biventricular hypertrophy. (Answer: b)

2. ECG findings associated with right ventricular hypertrophy include right axis deviation, a dominant R wave in lead V_1 (R > S), right atrial abnormality, and repolarization abnormalities in the right precordial leads. An R wave in lead aVL > 12 mm is consistent with LVH, not RVH. (Answer: b, c, e)

— Quick Review 39 —

Sinus tachycardia	
• Rate > ____ per minute	100
• P wave amplitude often (increases/decreases) and PR interval often (lengthens/shortens) with increasing heart rate	increases shortens
Atrial septal defect, primum	
• RSR' complex in lead _____	V_1
• (Right/left) axis deviation, in contrast to (right/left) axis deviation in secundum ASD	Left, right
• ____ degree AV block in 15-40%	First
• Advanced cases have ____ hypertrophy	biventricular
• Primum ASDs represent 15% of all ASDs, and are due to deficient tissue in the lower portion of the ____. These ASDs are usually (small/large), may be accompanied by anomalous ____ venous drainage, and are associated with a cleft anterior ____ valve leaflet	septum, large pulmonary mitral

ECG 40. 75-year-old female with heart failure:



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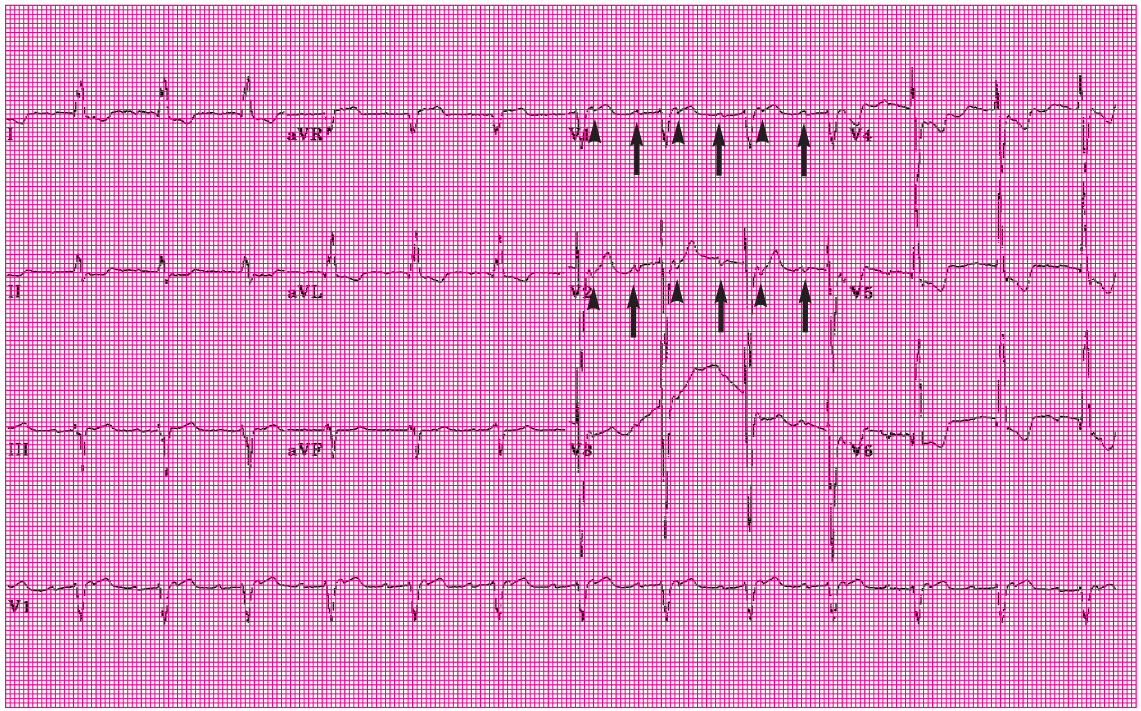
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 40 was obtained from a 75-year-old female with heart failure. At first glance, the ECG looks like an ectopic atrial rhythm at a rate of 80 beats/minute with first-degree AV block. However, on closer inspection, leads V_1 and V_2 show additional P waves immediately following each QRS complex (arrows mark conducted P waves; arrowheads mark nonconducted P waves), consistent with atrial tachycardia at a rate of 160 beats/minute with 2:1 AV block. In a patient with heart failure, this arrhythmia is commonly due to digitalis toxicity. Also noted are LVH (R wave in aVL + S wave in V_3 > 20 mm in females), nonspecific intraventricular conduction disturbance (QRS duration = 0.11 seconds), and ST-T wave changes in leads V_5-V_6 secondary to LVH (i.e., "strain" pattern).

Codes:

15	Atrial tachycardia
32	2:1 AV block
40	Left ventricular hypertrophy
49	Nonspecific intraventricular conduction disturbance
67	ST and/or T wave abnormalities secondary to hypertrophy
71	Digitalis toxicity



Questions: ECG 40

1. Atrial tachycardia with block is associated with:

   a. Mobitz Type I second-degree AV block
   b. Mobitz Type II second-degree AV block
   c. First-degree AV block
   d. Complete heart block

2. Arrhythmias associated with digitalis toxicity include:

   a. Ventricular fibrillation
   b. Ventricular tachycardia
   c. Paroxysmal atrial tachycardia
   d. Junctional tachycardia
   e. AV block with accelerated junctional rhythm
   f. Sinoatrial exit block
   g. Sinus node arrest

Answers: ECG 40

1. Atrial tachycardia with block is often a manifestation of digitalis toxicity, and results in a regular atrial rhythm with intermittent nonconducted P waves due to second-degree AV block, which can either Mobitz Type I or Type II. When 2:1 AV block is present, it is difficult to distinguish between these mechanisms based on surface ECG alone. (Answer: a, b)

2. Digitalis toxicity can induce nearly every known arrhythmia. Hypokalemia, hypomagnesemia, and hypercalcemia increase the risk of digitalis toxicity. (Answer: all)

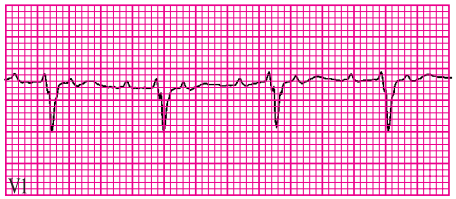
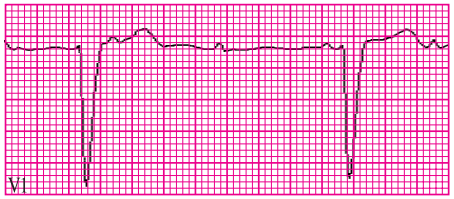
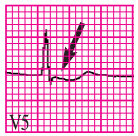
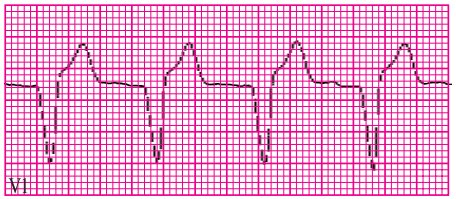
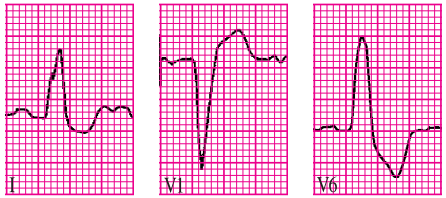
— Quick Review 40 —

Atrial tachycardia	
• Three or more consecutive (sinus/nonsinus) beats at an atrial rate of 100-240 bpm	nonsinus
• P wave is (always/sometimes) visualized	sometimes
• QRS follows each P wave unless ____ AV block is present	2° or 3°
AV block, 2:1	
• Regular sinus or ____ rhythm	ectopic atrial
• 2 ____ waves for every QRS complex	P
• Can be Mobitz type I or type II 2° AV block (true/false)	true
Nonspecific intraventricular conduction disturbance	
• QRS ≥ ____ seconds in duration but morphology does not meet criteria for LBBB or RBBB, *or* abnormal ____ without widening of the QRS complex	0.11 notching

— POP QUIZ —

Pattern Recognition: Digitalis Effect vs. Toxicity

Instructions: Determine which of the following ECGs are consistent with digitalis effect and which are consistent with digitalis toxicity.

Choose All That Apply	Answer
A.  **B.**  **C.**  **D.**  **E.** 	*Digitalis effects* include sagging ST segment depression with upward concavity (choice "c"), T wave changes (flat, inverted, or biphasic), shortening of the QT interval, and PR lengthening. ST changes are difficult to interpret in the setting of LVH, RVH, or bundle branch block; however, if typical sagging ST segments are present and the QT interval is shortened, digitalis effect should be considered. (Answer: c) *Digitalis toxicity* can cause almost any type of cardiac dysrhythmia or conduction disturbance, except bundle branch block (choice "e"). Examples include atrial tachycardia with block (choice "a"), atrial fibrillation with regular RR intervals (from complete heart block with junctional escape rhythm), second- or third-degree AV block (choice "b"), complete heart block with accelerated junctional or idioventricular rhythm (choice "d"), and SVT with alternating bundle branch block. Digitalis toxicity may be exacerbated by hypokalemia, hypomagnesemia, or hypercalcemia. Note: Electrical cardioversion of atrial fibrillation is contraindicated in the setting of digitalis toxicity due to the increased risk of protracted asystole or ventricular fibrillation. (Answer: a, b, d)

Don’t Get Confused!

Atrial Tachycardia with AV Block

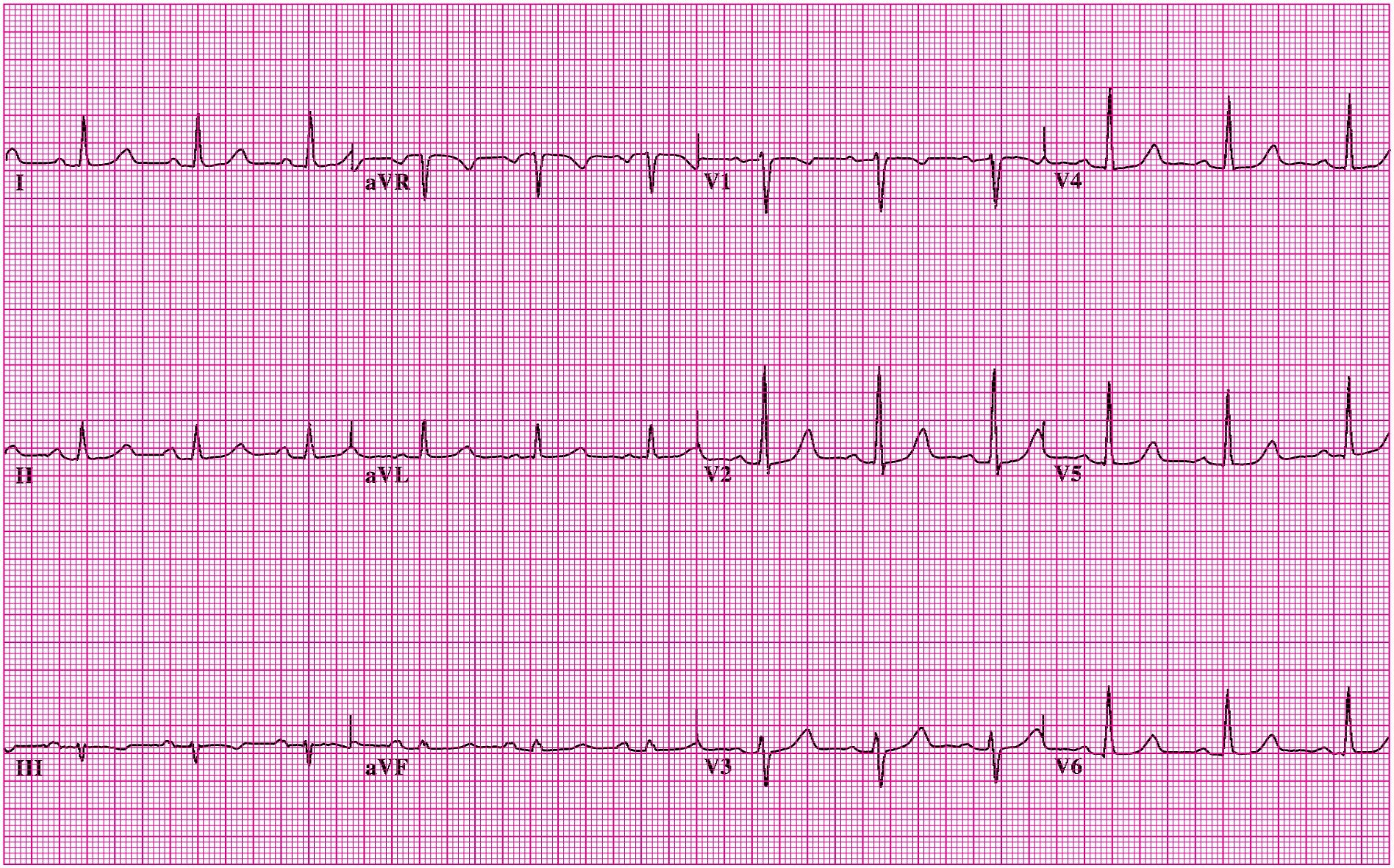
P wave axis or morphology different from sinus node, regular atrial rate of 100-240 per minute, isoelectric intervals between P waves in all leads, and second- or third-degree AV block with nonconducted P waves

<u>May be confused with</u>:

Atrial flutter

Atrial tachycardia with AV block has a distinct isoelectric baseline between P waves and an atrial rate of 100-240 per minute, whereas atrial flutter lacks an isoelectric baseline (except in lead V_1) and has an atrial rate of 240-340 per minute

ECG 41. Asymptomatic 61-year-old female:



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REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

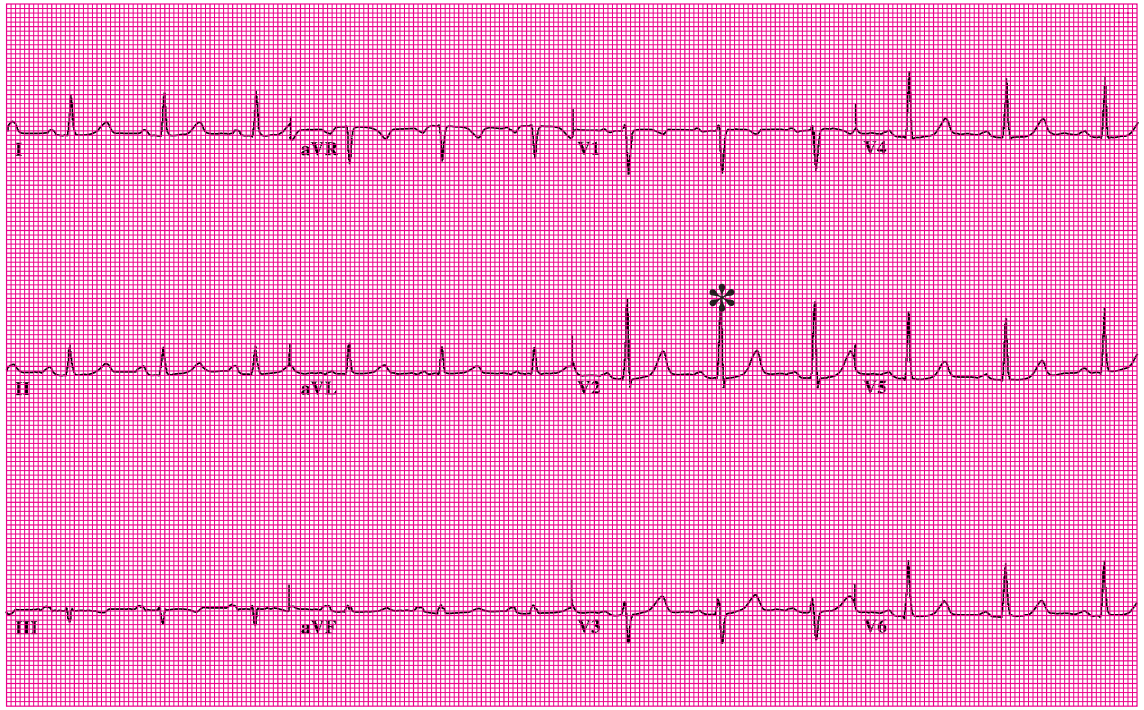
PACED RHYTHMS

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

ECG 41 was obtained in an asymptomatic 61-year-old female. The ECG shows a normal sinus rhythm with a PR interval at the upper limit of normal (0.20 seconds). There is a prominent R wave in V_2 (asterisk) with subsequent loss in R wave voltage from V_2 to V_3, which is due to V_2-V_3 electrode switch. This is otherwise a normal tracing.

Codes:

03	Incorrect electrode placement
07	Sinus rhythm



Questions: ECG 41

1. Both limb lead reversal and dextrocardia demonstrate:

   a. Inversion of the P-QRS-T in leads I and aVL
   b. Abnormal R wave progression in V_1-V_6
   c. Right ventricular hypertrophy
   d. Right atrial abnormality

2. Incorrect electrode placement of one of the precordial leads often manifests as unexplained loss of R wave voltage in the affected lead:

   a. True
   b. False

Answers: ECG 41

1. Limb lead reversal can be mistaken for dextrocardia, since both conditions manifest inversion of the P-QRS-T complex in leads I and aVL. Dextrocardia is associated with reverse R wave progression in leads V_1-V_6, right ventricular hypertrophy, and right atrial abnormality, none of which are present in limb lead reversal. (Answer: a)

2. Incorrect electrode placement in one of the precordial leads usually manifests as unexplained loss of R wave voltage in the affected lead (e.g., V_2), followed by return of normal R wave progression in the remaining leads (e.g., V_3-V_6). (Answer: a)

— Quick Review 41 —

Incorrect electrode placement

Limb lead reversal (reversal of right and left arm leads)

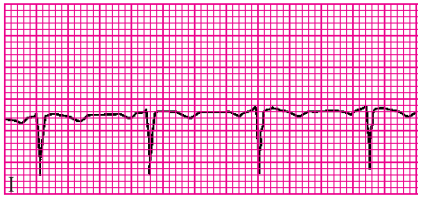
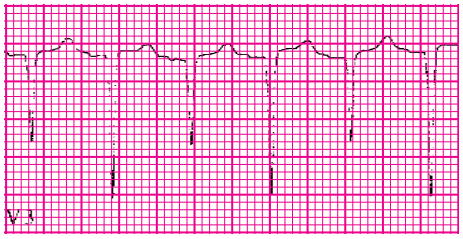
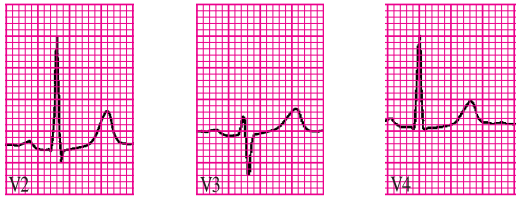
- Resultant ECG mimics dextrocardia with ____ of the P-QRS-T in leads ____ and aVL — inversion; I
- To distinguish between these conditions, look at precordial leads: dextrocardia shows (reverse/normal) R wave progression, while limb lead reversal shows (reverse/normal) R wave progression. — reverse; normal

Precordial lead reversal: Unexplained decrease in ____ voltage in two consecutive leads (e.g., V_1, V_2) with a return to normal progression in the following leads — R wave

— POP QUIZ —

Pattern Recognition: Clinical Disorders

Instructions: Determine the clinical diagnosis associated with each of the following ECGs.

ECG	Diagnosis	Answer
 Plus reverse precordial R-wave progression	a. Atrial septal defect, primum b. Atrial septal defect, secundum c. Dextrocardia d. Intracerebral hemorrhage e. Limb lead reversal f. Precordial lead reversal g. Digitalis effect h. Hypothermia i. Pericardial effusion	*Dextrocardia* results in an inverted ("upside down") P-QRS-T in leads I and aVL, and reverse R wave progression in the precordial leads (decreasing R wave amplitude from leads V_1-V_6). Inverted P-QRS-T in leads I and aVL with normal precordial R wave progression suggests limb lead reversal. (Answer: c)
		*Pericardial effusion* may present with electrical alternans (alternation in the amplitude or direction of P, QRS, and/or T waves) and low voltage QRS, but neither finding is sensitive or specific for the diagnosis: Only one-third of patients with QRS alternans have a pericardial effusion, and only 12% of patients with pericardial effusions have electrical alternans. If electrical alternans involves the P-QRS-T ("total alternans"), effusion with tamponade is often present. Sinus tachycardia is almost always present when pericardial effusion progresses to cardiac tamponade. (Answer: i)
		*Precordial lead reversal* results in an unexplained decrease in R wave voltage in two consecutive precordial leads (e.g., V_1, V_2), followed by a return to normal R wave progression (e.g., V_3-V_6). (Answer: f)

Common Dilemmas in ECG Interpretation

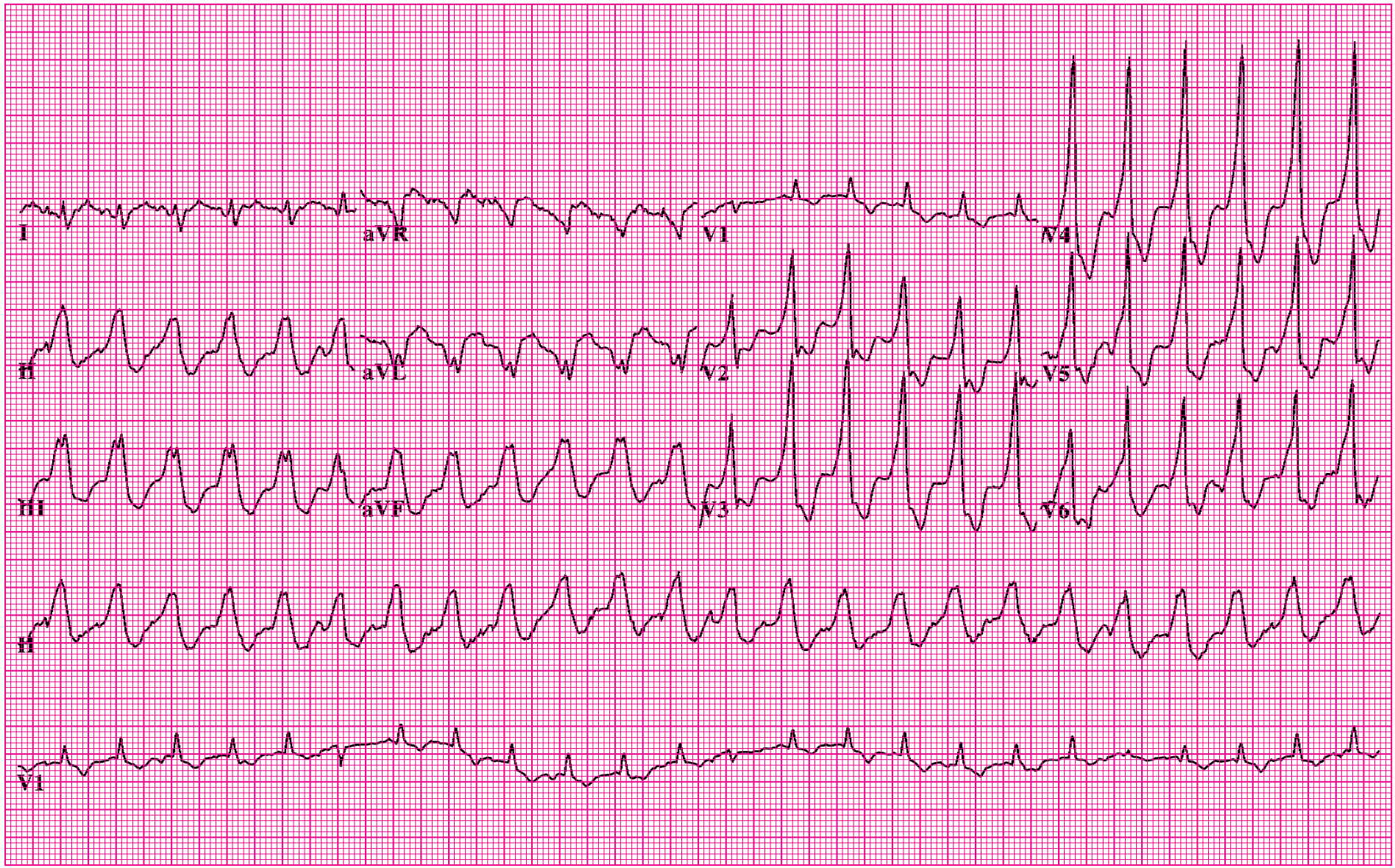
Problem:

The ECG shows an acute myocardial infarction. Should any other ECG diagnoses be coded?

Recommendation

Yes. It is also important to code item 65 (ST and/or T abnormalities suggesting myocardial injury) when acute myocardial infarction is present. Also code item 65 when ST segment depression is present in leads V_1 and V_2 in the setting of posterior MI.

ECG 42. 66-year-old male in the emergency room with palpitations and syncope:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
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AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
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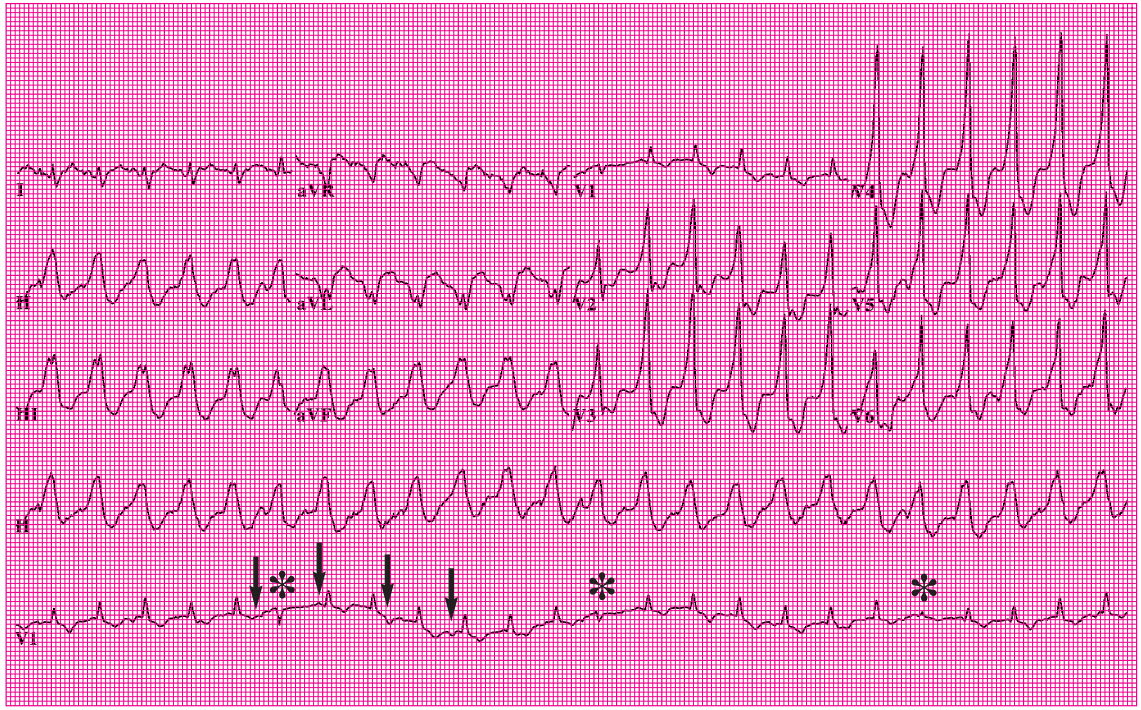
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 42 was obtained in a 66-year-old male who was taken to the emergency room with palpitations and syncope. The ECG shows a rapid wide complex tachycardia at 146 beats/minute. The wide QRS duration (0.15 seconds) suggests a ventricular origin for the tachycardia; this is confirmed by the presence of an underlying sinus tachycardia at approximately 120 beats/minute (arrows mark the P waves), resulting in AV dissociation, and the presence of ventricular capture complexes, manifesting as fusion complexes in the sixth beat on the rhythm strip and every seventh beat thereafter (asterisks). The QRS axis is rightward (measuring 98° by the computer), but does not meet criteria for right axis deviation (> 100°). Irregularity in the baseline in leads I and aVR is due to artifact.

Codes:

04	Artifact
10	Sinus tachycardia (> 100)
25	Ventricular tachycardia (≥ 3 consecutive complexes)
35	AV dissociation



Questions: ECG 42

1. ECG findings in this tracing favoring the diagnosis of ventricular tachycardia over supraventricular tachycardia include:

   a. AV dissociation
   b. Concordance of QRS complexes in V_1 - V_6
   c. Fusion beats
   d. Monophasic right bundle branch block pattern in V_1
   e. QRS > 0.14 seconds

2. Fusion complexes during wide QRS tachycardia favor the diagnosis of supraventriclar tachycardia over ventricular tachycardia:

   a. True
   b. False

3. The origin of the ventricular tachycardia in this tracing is:

   a. Right ventricle
   b. Left ventricle
   c. Unable to determine

Answers: ECG 42

1. Answer: All

2. Fusion complexes result from simultaneous activation of the ventricle from two different sources, resulting in a QRS complex intermediate in morphology between the QRS complexes of each source. Although not a common finding, fusion complexes in the setting of a wide QRS tachycardia are highly suggestive of ventricular tachycardia. (Answer: b)

3. In general, a positive QRS defection in lead V_1 suggests a left ventricular origin for ventricular tachycardia, while a negative QRS in lead V_1 suggests a right ventricular origin. (Answer: b)

— Quick Review 42 —

Ventricular tachycardia	
• Rapid succession of three or more premature ventricular beats at a rate > ____ per minute	100
• RR intervals are usually regular but may be irregular (true/false)	true
• (Abrupt/gradual) onset and termination are evident	Abrupt
• AV ____ is common	dissociation
• Look for ventricular ____ complexes and ____ beats as markers for VT	capture, fusion
Fusion complexes	
• Due to simultaneous activation of the ventricle from ____ sources, resulting in a QRS complex that is ____ in morphology between each source	2 intermediate
AV dissociation	
• Atrial and ventricular rhythms are ____ of each other	independent
• Ventricular rate is (</≥) than the atrial rate	≥

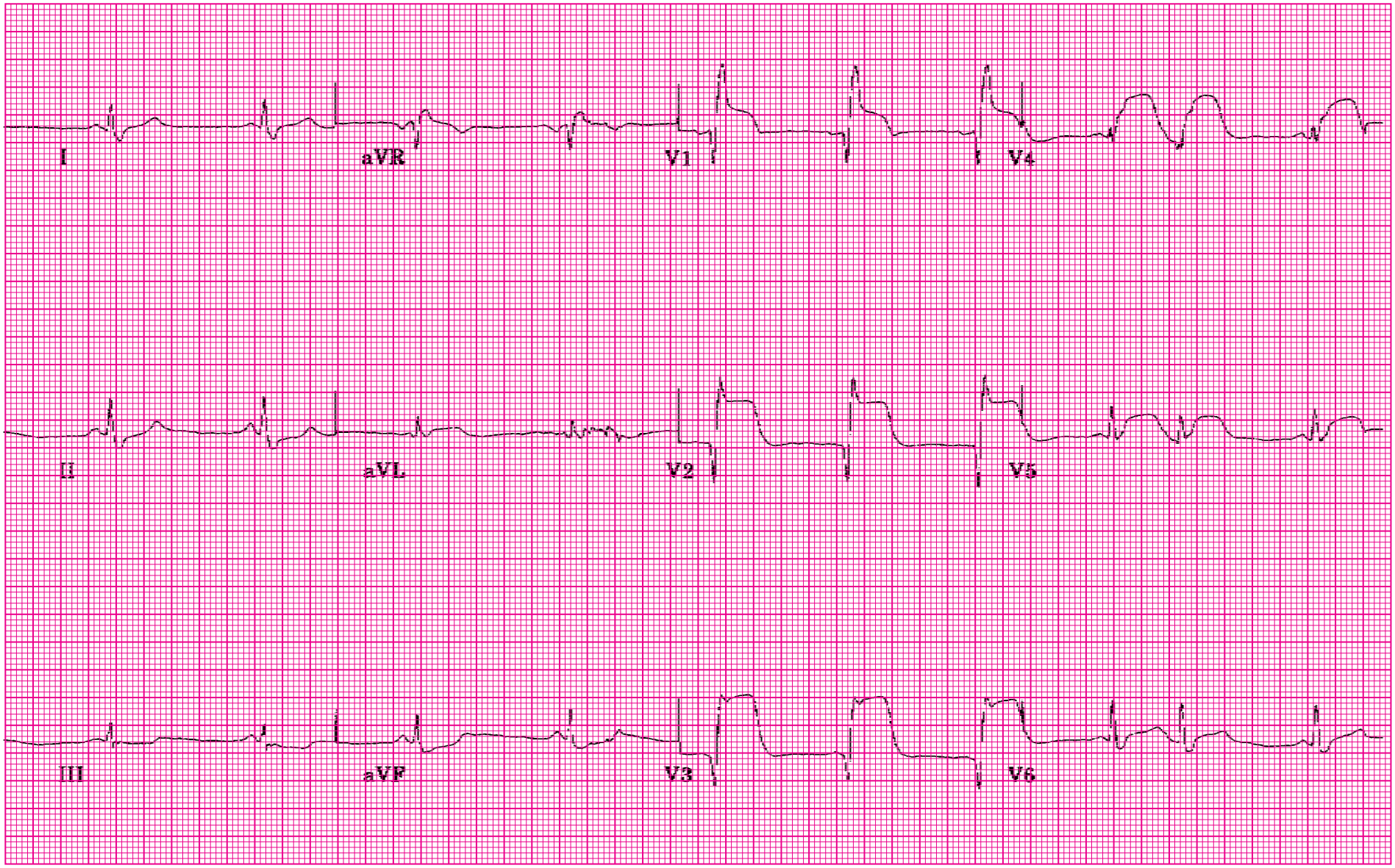
Differential Diagnosis

Low Voltage ECG

(Amplitude of the entire QRS complex (R+S) < 10 mm in all precordial leads and < 5 mm in all limb leads)

- Chronic lung disease
- Pericardial effusion
- Myxedema
- Obesity
- Pleural effusion
- Restrictive or infiltrative cardiomyopathies
- Diffuse coronary disease

ECG 43. 74-year-old diabetic male with sudden onset of dyspnea:



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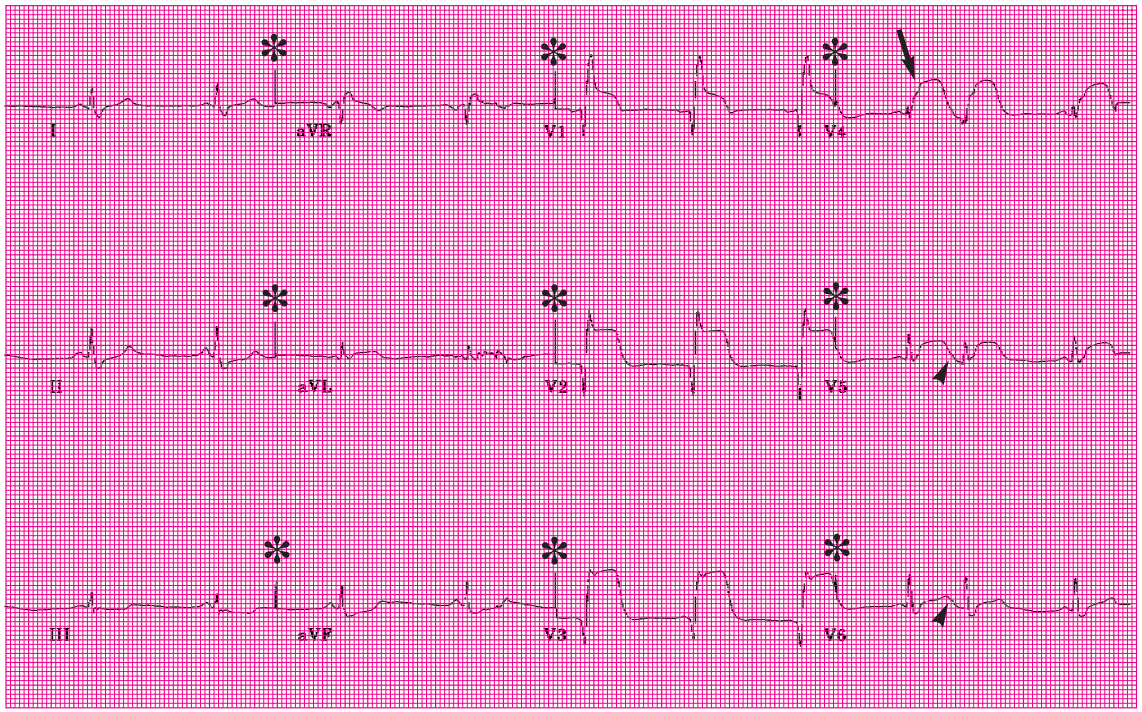
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 43 was obtained from a 74-year-old diabetic male with sudden onset of dyspnea. The ECG shows sinus rhythm at a rate of 64 beats/minute with RBBB. There is an ectopic supraventricular beat (arrow), which most likely represents an atrial premature complex (small deformity in downslope of T wave just preceding the premature QRS complex in leads V_5-V_6 is probably a P wave [arrowheads]). Most notable are the presence of abnormal Q waves and marked ST segment elevation in the precordial leads, consistent with acute anterior MI with lateral myocardial injury. Leads III and aVF show mild ST segment depression, which most likely represents reciprocal changes. The vertical lines in each lead (asterisks) represent lead switch markers, not pacemaker spikes.

Codes:

07	Sinus rhythm
13	Atrial premature complexes
43	RBBB, complete
53	Anterior or anteroseptal MI (age recent or acute)
65	ST and/or T wave abnormalities suggesting myocardial injury



Questions: ECG 43

1. The age of the myocardial infarction on this ECG is:

   a. Hours-to-days
   b. Days-to-weeks
   c. Weeks-to-months

2. Which of the following statements are about right bundle branch block (RBBB) are true:

   a. RBBB impairs the ability to diagnose LVH on ECG
   b. RBBB impairs the ability to diagnose Q wave myocardial infarction on ECG
   c. RBBB impairs the ability to determine QRS axis
   d. Most patients with RBBB have structural heart disease

Answers: ECG 43

1. Q waves usually develop in the hours-to-days after MI, and may persist indefinitely, regress, or infrequently disappear. ST elevation usually develops in seconds-to-minutes after MI and resolves in minutes-to-hours after reperfusion of the infarct artery. If reperfusion is not achieved, ST elevation resolves slowly over hours-to-days. ST elevation persisting beyond 48 hours post-MI is an adverse prognostic marker. T wave inversion begins before the ST segment returns to baseline. The present ECG shows Q waves and ST elevation, but no T wave inversion, suggesting an infarct that is hours-to-days old. (Answer: a)

2. Most patients with RBBB have either coronary artery disease (most common), hypertensive heart disease, myocarditis, cardiomyopathy, rheumatic heart disease, cor pulmonale (acute or chronic), degenerative disease of the conduction system (Lenegre's disease), or sclerosis of the cardiac skeleton (Lev's disease). Patients with RBBB and anatomical LVH may not manifest increased QRS voltage; however, LVH can still be diagnosed when voltage criteria are met. The first 0.04 - 0.06 seconds of the QRS is unaffected by RBBB, and can be used to identify QRS axis and abnormal Q waves of myocardial infarction. (Answer: a, d)

— Quick Review 43 —

RBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Secondary R wave (R') in lead ____ is usually (shorter/taller) than the initial R wave	V_1 taller
• Onset of intrinsicoid deflection in leads V_1 and V_2 >____ seconds	0.05
• ST segment ____ and T wave ____ in V_1, V_2	depression inversion
• Wide slurred S wave in leads ____	I, V_5, V_6
• QRS axis is usually (normal/leftward/rightward)	normal
ST and/or T wave changes suggesting myocardial injury	
• Acute ST segment (elevation/depression) with upward (convexity/concavity) in the leads representing the area of infarction	elevation convexity
• T waves invert (before/after) ST segments return to baseline	before
• Associated ST (elevation/depression) in the noninfarct leads is common	depression
• Acute ____ wall injury often has horizontal or downsloping ST segment depression with upright T waves in V_1-V_3, with or without a prominent R wave in these same leads	posterior

— POP QUIZ —

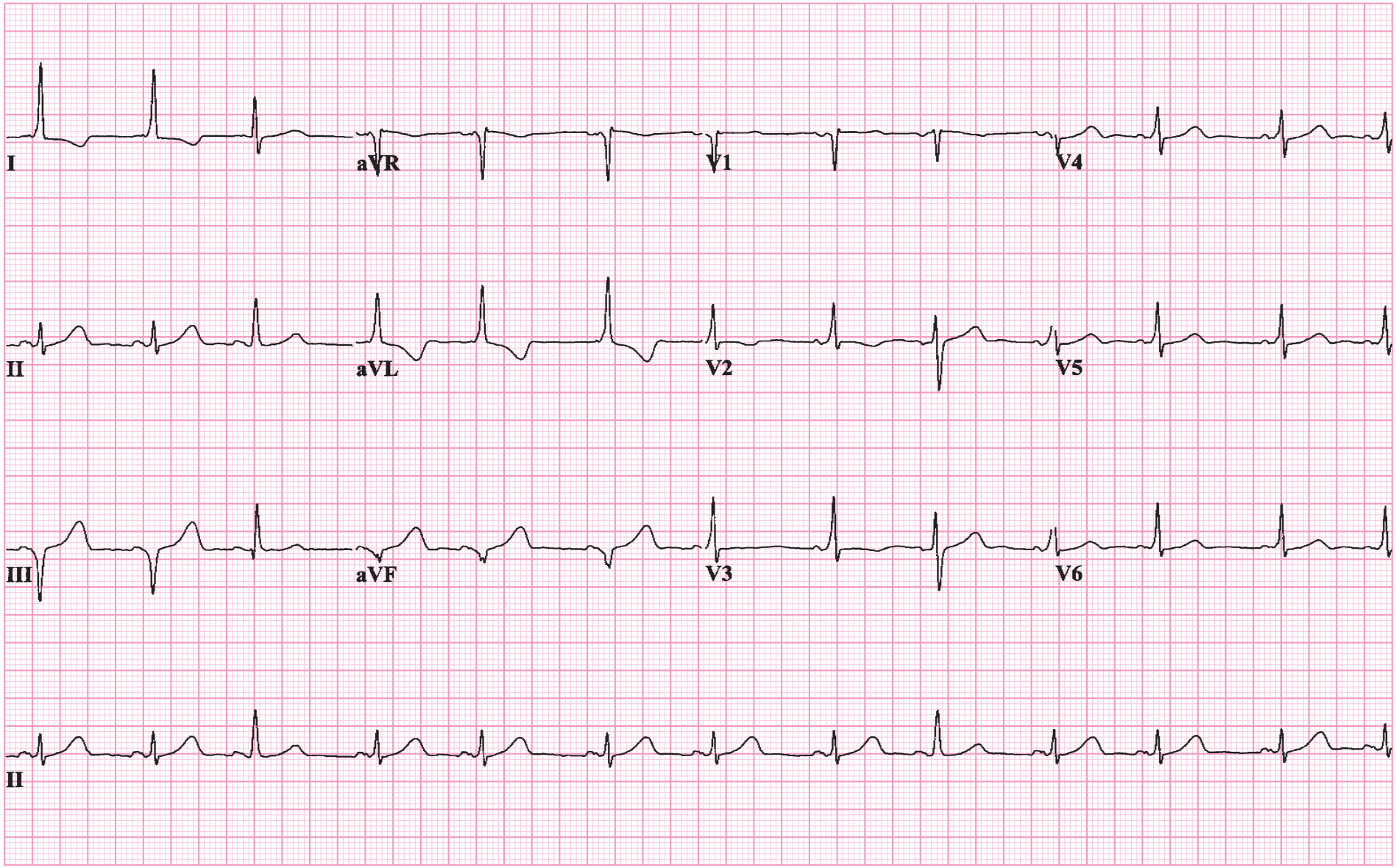
Differential Diagnosis: QRS Amplitude

Instructions: For each diagnosis below, select all QRS amplitude changes that apply:

a. Low voltage QRS
b. Tall (large amplitude) QRS
c. Prominent R wave in V_1
d. QRS alternans (alternation in amplitude)

Diagnosis	Answer
Left ventricular hypertrophy (LVH)	Tall QRS. QRS alternans sometimes in hypertensive heart disease. (Answer: b, d)
Left bundle branch block (LBBB)	Tall QRS. LBBB interferes with determination of QRS axis, ventricular hypertrophy, and MI. (Answer: b)
Thin body habitus	Tall QRS (may lead to false-positive diagnosis of LVH). (Answer: b)
Right ventricular hypertrophy (RVH)	Prominent R wave in V_1. Right axis deviation and deep S waves in V_5V_6 are common. (Answer: c)
Diffuse coronary disease	Low voltage QRS (infrequent); QRS alternans (infrequent). (Answer: a, d)
Right bundle branch block (RBBB)	Prominent R wave in V_1. (Answer: c)
Chronic lung disease (e.g., emphysema) with pulmonary hypertension	Low voltage QRS; prominent R wave in V_1 (if pulmonary hypertension with RVH is present). (Answer: a, c)
Pericardial effusion	Low voltage QRS; QRS alternans in some. If electrical alternans involves the P-QRS-T ("total alternans), pericardial effusion with cardiac tamponade is often present. (Answer: a, d)
Posterior myocardial infarction	Prominent R wave in V_1 and/or V_2 with ST depression and upright T waves. Inferior MI is common. (Answer: c)
Myxedema	Low voltage QRS. Sinus bradycardia and flattened or inverted T waves are common. (Answer: a)
Infiltrative cardiomyopathy	Low voltage QRS. Pseudoinfarct pattern (abnormal Q waves) may occur. (Answer: a)
Obesity	Low voltage QRS. Ability to detect ventricular hypertrophy based on voltage criteria is impaired. (Answer: a)

ECG 44. 57-year-old female with a routine ECG prior to elective surgery:



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- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
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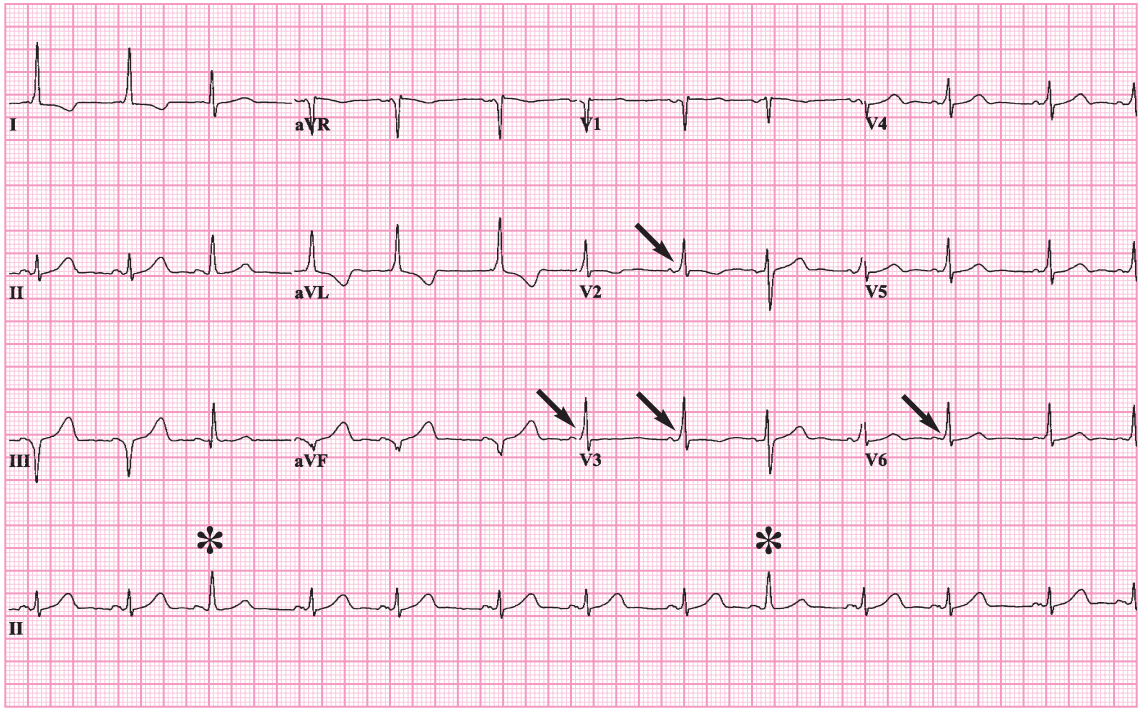
ECG 44 was obtained in a 57-year-old female on routine testing prior to elective surgery. The ECG shows sinus rhythm with changes in the PP interval consistent with sinus arrhythmia (PP interval varies by ≥ 0.16 seconds). In addition, the QRS morphology is observed to change during the recording; due to intermittent pre-excitation (intermittent Wolff-Parkinson-White pattern). Except for the 3rd and 9th beats (asterisks), all other beats demonstrate a short PR interval, delta wave (arrow), and prolonged QRS duration consistent with WPW pattern and conduction across an accessory bypass tract. The 3rd and 9th beats occur early, when the accessory pathway is still refractory (blocked), and demonstrate normal AV conduction and QRS complexes.

Codes:

07	Sinus rhythm
08	Sinus arrhythmia
34	Wolff-Parkinson-White pattern



Questions: ECG 44

1. In patients in WPW pattern on ECG, which feature is associated with a low risk for developing rapid ventricular rate during atrial fibrillation:

   a. The accessory pathway connects the left atrium and left ventricle
   b. Intermittent conduction over the accessory pathway in sinus rhythm
   c. Loss of accessory pathway conduction during atrioventricular reentry tachycardia

Answers: ECG 44

1. Patients with WPW pattern on their ECG are at risk for developing rapid conduction over the accessory pathway during atrial fibrillation. This rapid conduction can result in a very rapid ventricular rate and possibly syncope or even sudden cardiac death. The rapidity of accessory pathway conduction bears no relationship to its location in the heart. Similarly, when patients with WPW pattern develop typical atrioventricular (orthodromic) reentry, preexcitation is lost on ECG since the electrical impulse travels down the AV node and His-Purkinje system, resulting in normal activation of the ventricle. (The impulse returns to the atrium over the accessory pathway completing the reentrant circuit.) The presence of intermittent conduction over the accessory pathway in sinus rhythm is a reliable marker that the accessory pathway is not capable of rapid conduction during atrial fibrillation and therefore does not place the patient at risk for a rapid ventricular rate with syncope or sudden cardiac death. (Answer: b)

— Quick Review 44 —

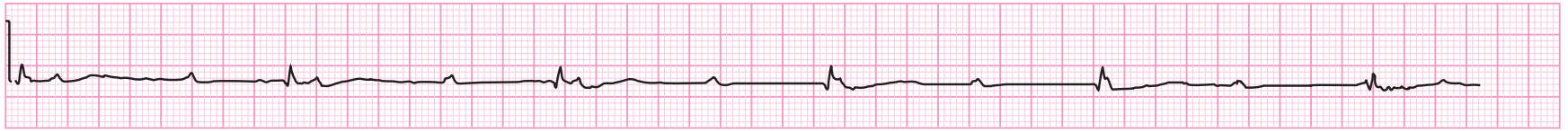
Sinus arrhythmia	
• (Sinus/nonsinus) P wave	Sinus
• Longest and shortest PP intervals vary by > ___ seconds or 10%	0.16
• Sinus arrhythmia differs from "ventriculophasic" sinus arrhythmia, the latter of which occurs in the setting of ____	heart block

— POP QUIZ —

Find The Imposter

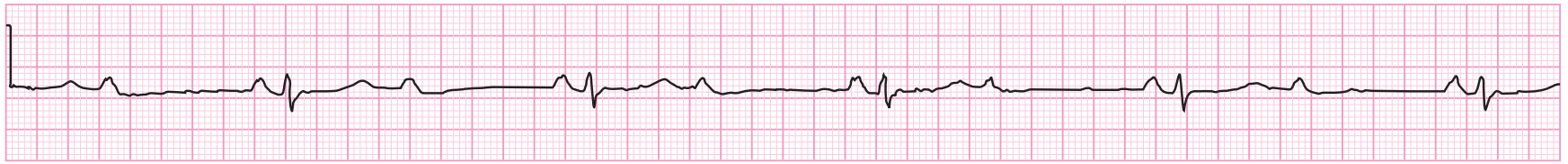
Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

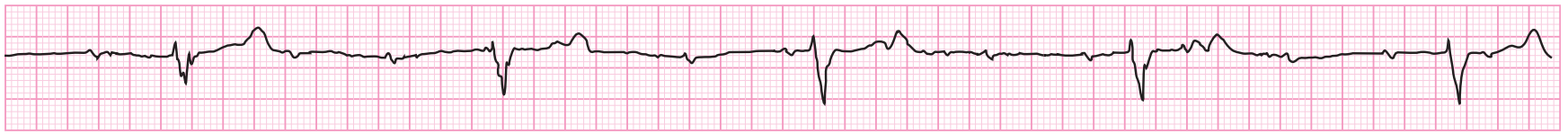


B.

C.



D.



Answer: Tracings A, B, and D are examples of complete heart block: P waves bear no consistent relationship to the QRS complexes and PP and RR intervals are constant. In these examples, the ventricular rhythm is maintained by a junctional (narrow complex) or ventricular (wide complex) escape rhythm. Tracing C shows sinus rhythm with 2:1 AV block and is the imposter. Every other P wave is conducted with a PR interval of 0.16 seconds.

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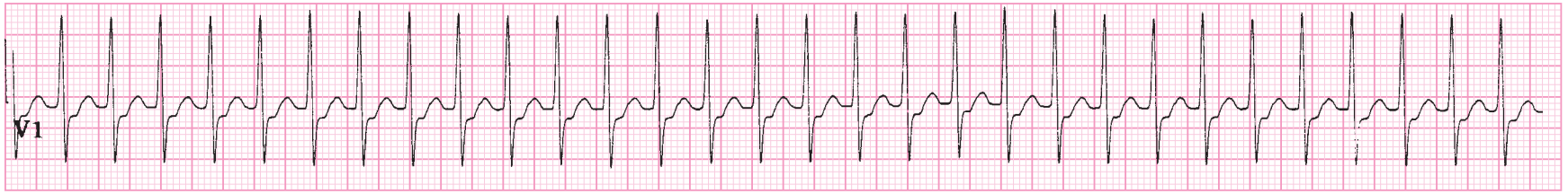

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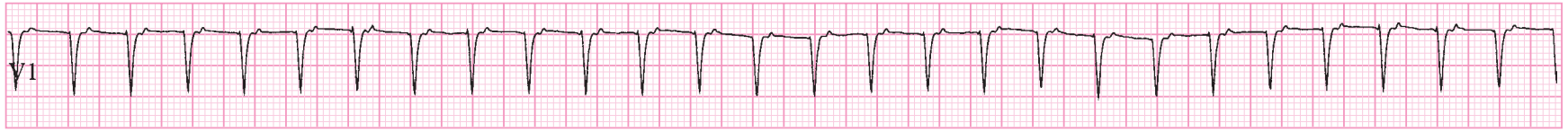
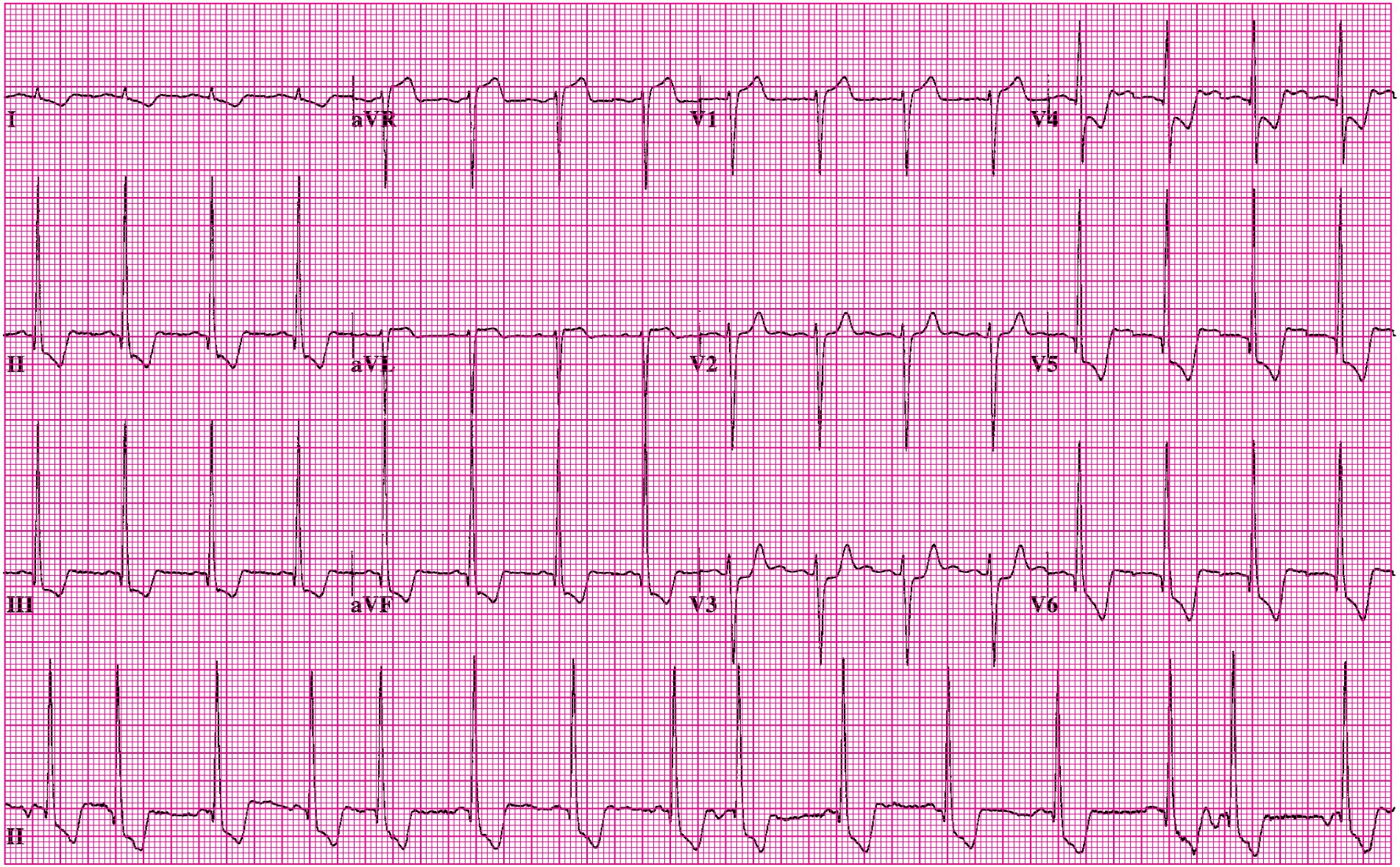


D.

Answer: Tracings B, C, and D demonstrate a regular, narrow QRS tachycardia with the suggestion of retrograde P waves at the end of each QRS complex. These rhythms are consistent with SVT, most likely reentry within the AV node or using a concealed accessory pathway. Tracing A shows atrial fibrillation and is the imposter. It shows a narrow QRS tachycardia that is irregular with no clear atrial activity. The irregular QRS intervals and the lack of clear P waves are consistent with atrial fibrillation.

ECG 45. 35-year-old male with syncope:



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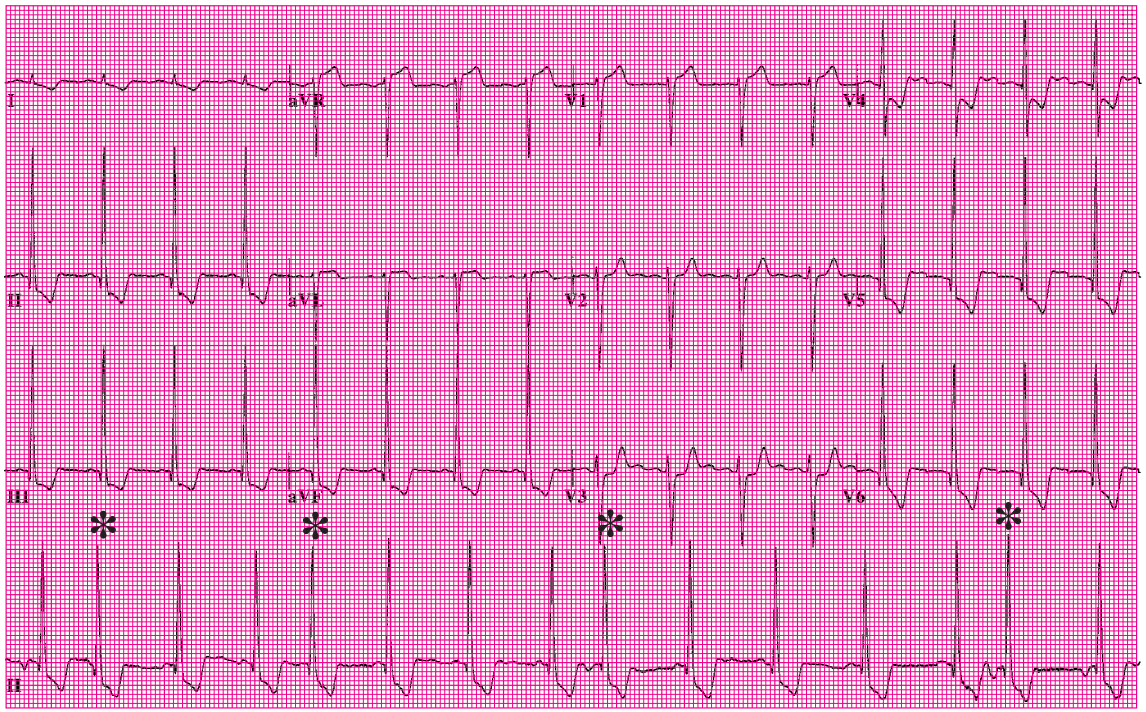
ECG 45 was obtained in a 35-year-old male with syncope and shows sinus rhythm with normally conducted APCs (asterisks). LVH (S wave in aVR $\geq$ 15mm; R wave in aVF $\geq$ 21mm; R wave in V_5 + S wave in $V_1 \geq$ 40mm; R wave in $V_6 >$ 20mm) with associated ST-T abnormalities are evident. Markedly increased QRS voltage and ST-T abnormalities in a young person with syncope suggest the diagnosis of hypertrophic cardiomyopathy (the inferior and anterolateral Q waves are secondary to hypertrophic cardiomyopathy, not previous infarction).

Codes:

07	Sinus rhythm
13	Atrial premature complexes
40	Left ventricular hypertrophy
67	ST and/or T wave abnormalities secondary to hypertrophy
85	Hypertrophic cardiomyopathy



Questions: ECG 45

1. Which of the following statements about hypertrophic obstructive cardiomyopathy are true?

   a. Left atrial abnormality is frequently seen
   b. Right axis deviation occurs in ~ 30% of cases
   c. LVH is present in > 90% of cases
   d. Pathological Q waves occur in 20-30% of cases
   e. ST and T wave changes are the most common finding
   f. Sinus node disease and AV block are common
   g. Nonsustained VT is a risk factor for sudden death

2. Causes of ST segment depression include:

   a. Hyperkalemia
   b. Hypokalemia
   c. Digoxin
   d. Quinidine
   e. Mitral valve prolapse

3. Which causes of ST segment depression in Question 2 are associated with:

   a Atrial fibrillation with a regular ventricular response
   b. Prominent U waves

4. LVH by voltage criteria is more likely to represent true anatomical LVH in younger patients compared to older patients:

   a True
   b. False

Answers: ECG 45

1. Hypertrophic cardiomyopathy is an uncommon disorder characterized by altered myocyte shape, size and alignment, which along with increased myocardial fibrosis, results in marked ventricular hypertrophy, LV stiffness, and diastolic dysfunction. The vast majority of patients have abnormal ECGs, with LVH in 50-65%, left atrial abnormality in 20-40%, and pathological Q waves (especially leads I, aVL, V_4 - V_5) in 20-30%. ST and T wave changes (repolarization abnormalities secondary to LVH) are the most common ECG findings, while right axis deviation is rare. The most frequent cause of mortality is sudden death, with risk factors including young age and a history of syncope and/or asymptomatic ventricular tachycardia on ambulatory monitoring. Sinus node disease and AV block are uncommon manifestations of this disorder. (Answer: a, d, e, g)

2. ST depression is a common manifestation of hypokalemia, along with decreased T wave amplitude and prominent U waves.

Classical digitalis effect produces ST depression that pulls down the first portion of the T wave to create a diphasic T wave, initially negative and then positive. ST depression can also be seen in patients taking quinidine, in conjunction with prolonged QT interval, flat or inverted T waves, and a prominent U wave. Approximately 20-40% of patients with mitral valve prolapse manifest some degree of ST depression and/or T wave inversion, especially in the inferior leads. ST segment depression is not a usual manifestation of hyperkalemia, although ST segment elevation can occur in advanced cases. (Answer: all)

3. Atrial fibrillation with a regular ventricular response should raise the suspicion of digitalis toxicity. In this setting, regularization of the ventricular response is due to complete heart block and accelerated junctional rhythm. Digitalis toxicity may be exacerbated by hypokalemia, hypomagnesemia, and hypercalcemia. Electrical cardioversion of atrial fibrillation is contraindicated in the setting of digitalis toxicity due to the risk of ventricular fibrillation. (Answer to 3a = c; Answer to 3b = b, c, d, e)

4. Increased QRS voltage is commonly observed in young adults with normal hearts. Many electrocardiographers are reluctant to diagnose LVH by voltage criteria alone in patients under the age of 40, and require other changes to be present (e.g., strain pattern, left axis deviation, delayed onset of intrinsicoid deflection, poor R wave progression). (Answer: b)

— Quick Review 45 —

Hypertrophic cardiomyopathy	
• (Right/left) atrial abnormality is common; (right/left) atrial abnormality on occasion	left right
• Majority have abnormal QRS complexes (true/false):	true
▸ (Small/large) amplitude QRS	large
▸ Large abnormal ____ waves (can give pseudoinfarct pattern in inferior, lateral, and anterior precordial leads)	Q
▸ Tall R wave with inverted T wave in V_1 simulating ____	RVH
▸ Nonspecific ST and/or T wave abnormalities are common (true/false)	false
▸ Apical variant of hypertrophic cardiomyopathy has deep T wave inversions in leads ____	V_4-V_6
▸ (Right/left) axis deviation in 20%	left

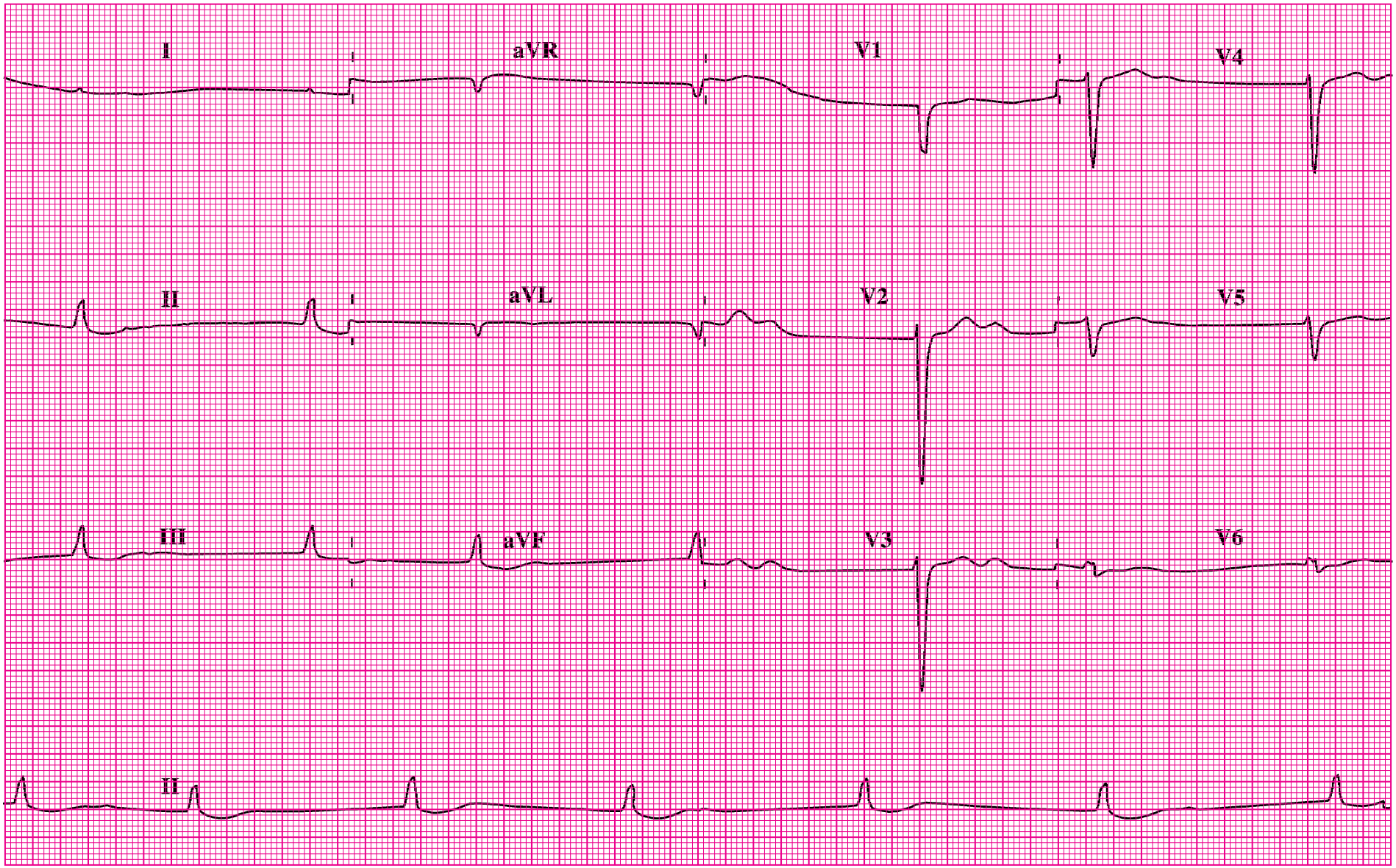
Differential Diagnosis

PROLONGED QT INTERVAL

(corrected QT interval $\geq$ 0.42-0.46 seconds)

- Drugs (quinidine, procainamide, disopyramide, amiodarone, sotalol, phenothiazine, tricyclics, lithium)
- Hypomagnesemia
- Hypocalcemia
- Marked bradyarrhythmias
- Intracranial hemorrhage
- Myocarditis
- Mitral valve prolapse
- Hypothyroidism
- Hypothermia
- Liquid protein diets
- Romano-Ward syndrome (normal hearing)
- Jervell and Lange-Nielson syndrome (deafness)

ECG 46. 68-year-old male with fatigue and dyspnea:



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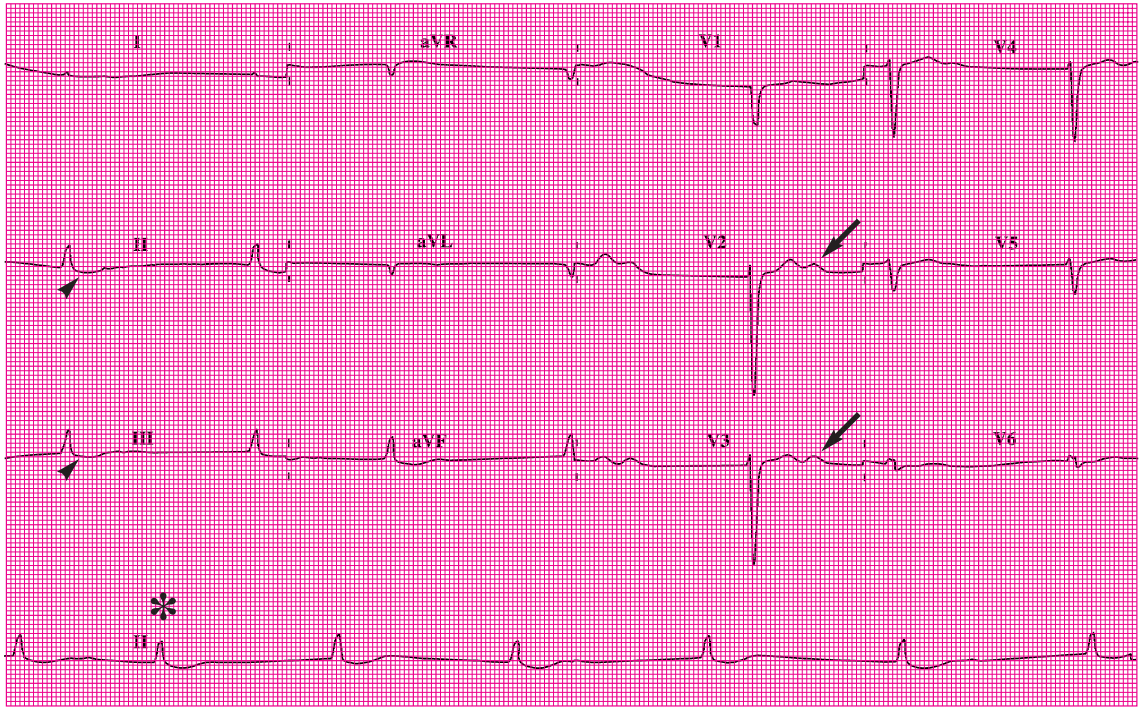
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 46 was obtained in a 68-year-old male with fatigue and dyspnea, and shows sinus arrest with a junctional rhythm at approximately 40 beats/minute. The slight irregularity in the early portion of the rhythm strip is due to the presence of an AV junctional premature complex (asterisk). The sagging ST segment depression (arrowheads) is typical for digitalis effect (even though it could also be coded as nonspecific ST-T abnormalities). Prominent U waves are present (arrows). This constellation of findings is consistent with digitalis toxicity.

Codes:

11	Sinus pause or arrest
20	AV junctional premature complexes
22	AV junctional rhythm/tachycardia
69	Prominent U waves
70	Digitalis effect
71	Digitalis toxicity



Questions: ECG 46

1. ECG findings attributable to digitalis effect as opposed to digitalis toxicity include:

   a. Right bundle branch block
   b. Paroxysmal atrial tachycardia with block
   c. Atrial fibrillation with regular ventricular response
   d. Bidirectional ventricular tachycardia
   e. Complete heart block
   f. Sagging ST segment depression
   g. Decreased T wave amplitude
   h. Shortening of the QT interval
   i. U waves
   j. Increased PR interval
   k. Left bundle branch block

2. Findings on this ECG consistent with hyperkalemia include:

   a. Flattened T waves
   b. Absent P waves
   c. Intraventricular conduction delay
   d. Prominent U waves

Answers: ECG 46

1. Typical digitalis effects include prolonged PR interval, sagging ST segment depression, decreased T wave amplitude, shortened QT interval, and prominent U waves. Arrhythmias and conduction disturbances associated with digitalis toxicity include paroxysmal atrial tachycardia (PAT) with block, atrial fibrillation with a regular ventricular response, junctional tachycardia, bidirectional ventricular tachycardia, and complete heart block. Digitalis does not produce bundle branch block or atrial flutter. (Answer: f, g, h, i, j)

2. The lack of P waves and the presence of IVCD are consistent with the diagnosis of hyperkalemia. However, normal T wave amplitude speaks strongly against this diagnosis, especially when hyperkalemia is acute. Prominent U waves are frequently observed in hypokalemia, not hyperkalemia. (Answer: b, c)

— Quick Review 46 —

Sinus pause or arrest	
• PP interval > _____ seconds	1.6-2.0
• Resumption of sinus rhythm at a PP interval that (is/is not) a multiple of the basic sinus PP interval	is not
• If sinus rhythm resumes at a multiple of the basic PP, consider _____	sinoatrial exit block
AV junctional rhythm	
• Rate ≤ ____ per minute	60
• QRS complex may be narrow or aberrant (true/false)	true
• Inverted P waves in leads ____ and upright P waves in leads ____ are common	II, III, aVF I, aVL
• RR interval of escape rhythm is usually (constant/variable)	constant
Digitalis toxicity	
• Digitalis toxicity can cause almost any type of cardiac dysrhythmia or conduction disturbance except _____	bundle branch block
• Typical abnormalities include:	
▸ Paroxysmal ____ tachycardia with block	atrial
▸ Atrial fibrillation with ____ heart block	complete
▸ Second or third-degree ____ block	AV
▸ Complete heart block with accelerated ____ or ____ rhythm	junctional idioventricular
▸ Supraventricular tachycardia with ____ bundle branch block	alternating

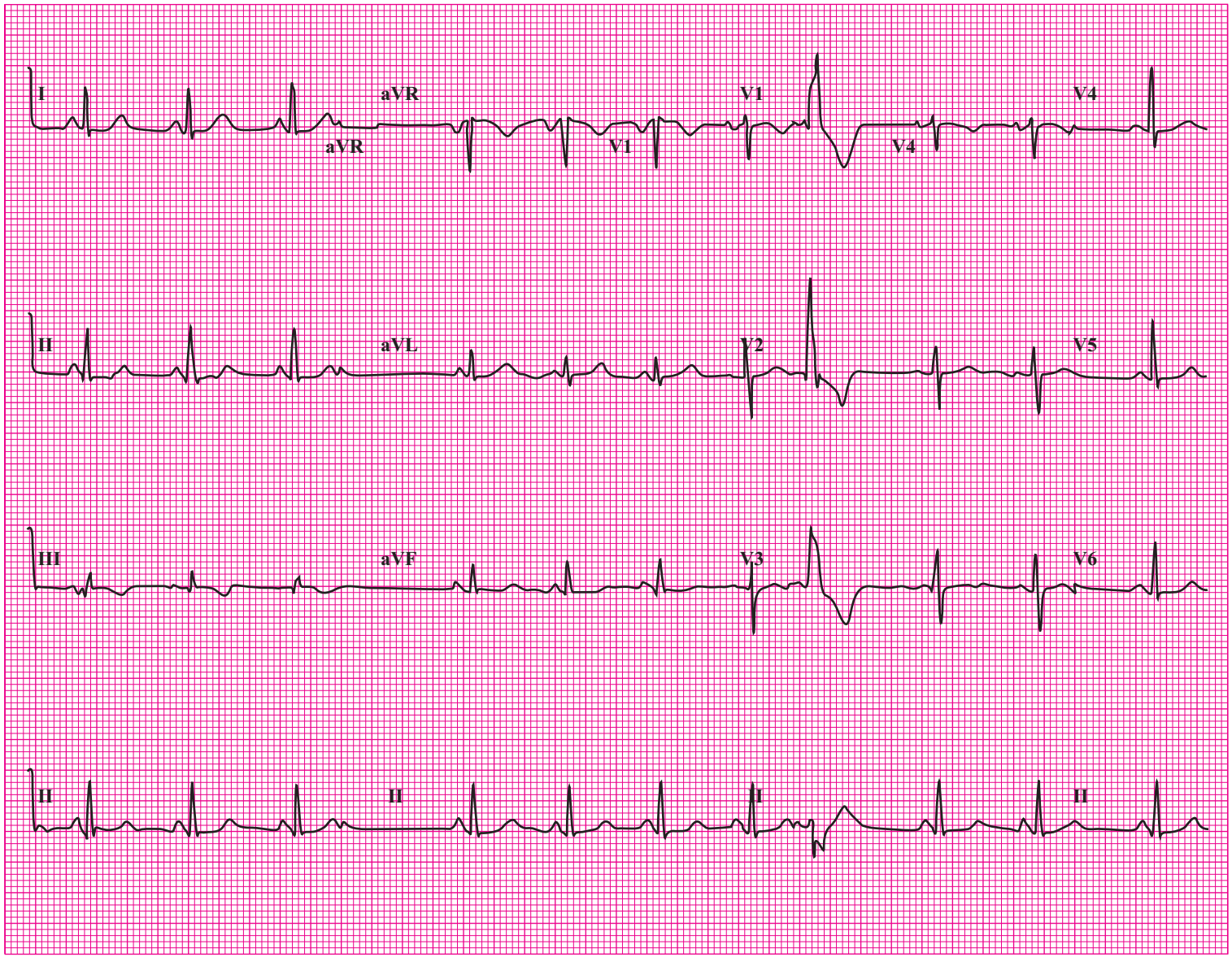
— POP QUIZ —

Find The Mistake

Instructions: Identify the incorrect ECG feature(s) for each of the ECG diagnoses listed below

ECG Features	Mistake
Atrial tachycardia with block • Sinus P waves • Atrial rate of 150-240 per minute • Isoelectric intervals between P waves in some but not all leads • Second- or third-degree AV block • Rhythm is regular	Nonsinus P waves are present; isoelectric intervals are present in all leads
Multifocal atrial tachycardia • Atrial rate > 100 per minute • P waves with $\geq$ 3 morphologies • PR, RR intervals vary • RP interval is constant	RP interval varies (not constant)
Atrial flutter • Rapid regular atrial undulations at 240-340 per minute • Undulations in leads II, III, AVF, and V_1 are typically inverted without an isoelectric baseline	In V_1, flutter waves are typically small positive deflections with a distinct isoelectric baseline
Atrial fibrillation • Totally irregular atrial activity manifests as undulations of varying amplitude, duration and morphology • Ventricular rhythm is irregularly irregular • Atrial activity may regularize with digitalis toxicity	Ventricular activity may regularize with digitalis toxicity, but atrial activity remains irregular

ECG 47. 68-year-old asymptomatic male:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

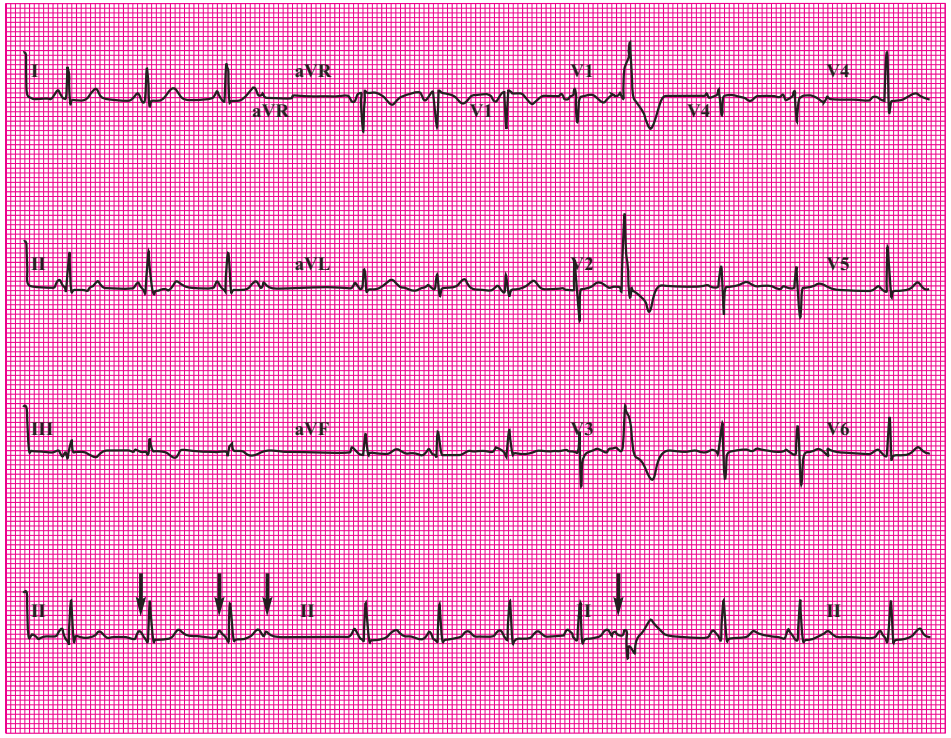
PACED RHYTHMS

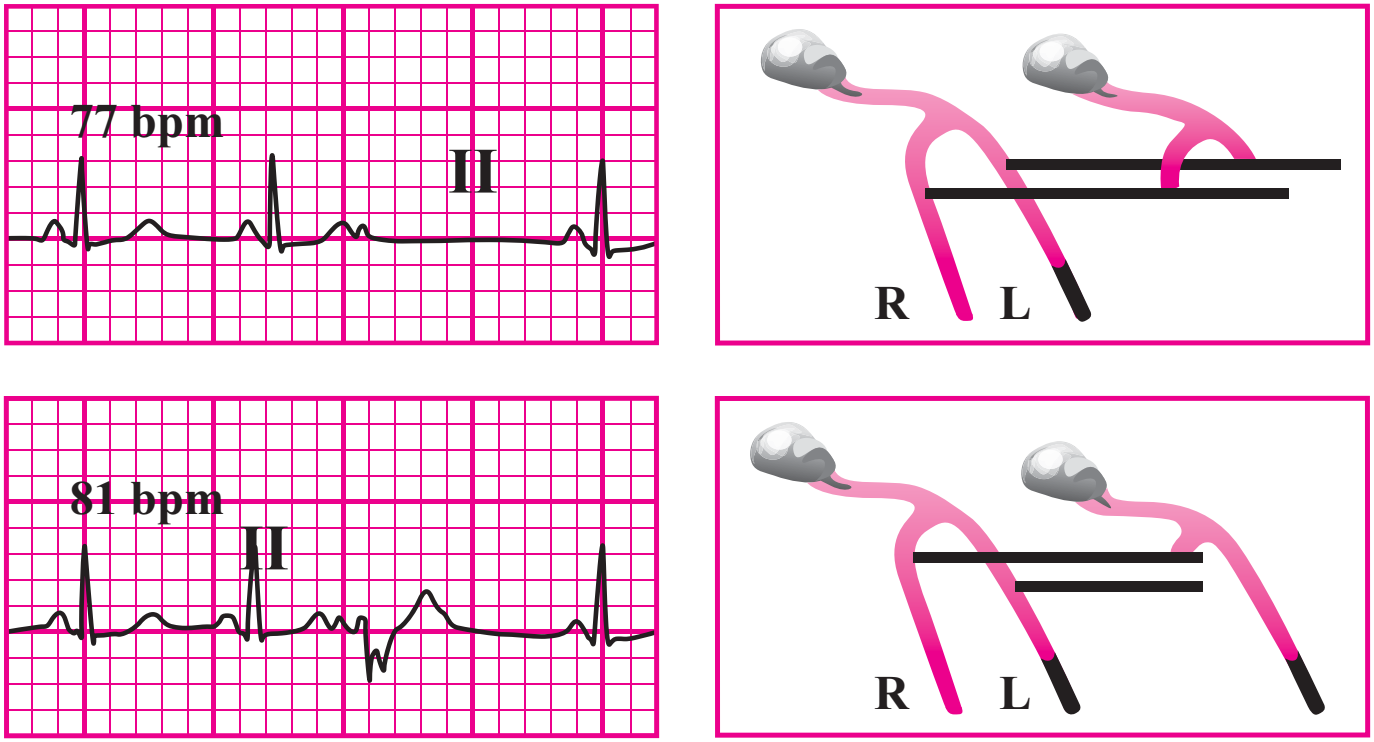
- ☐ 90. Atrial or coronary sinus pacing
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 47 was obtained in a 68-year old asymptomatic male. In the first half of the tracing, the patient is in a sinus rhythm at approximately 77 bpm. After the third QRS complex, the distortion in the T-wave is caused by a blocked APC. In the second half of the tracing, a second APC is noted; this one is conducted aberrantly, resulting in right bundle branch block QRS pattern. In the interim between the first blocked APC and the second conducted APC, the sinus rhythm increased from 77 bpm to 81 bpm. The fact that the first APC was blocked and the second one conducted (aberrantly) was a manifestation of Ashman's Phenomenon. The tracing also shows left atrial abnormality/enlargement and non-specific repolarization abnormalities, particularly noticeable in leads I, II, III, aVF, and V3.

Codes:

06	Left atrial abnormality / enlargement
07	Sinus rhythm
13	Atrial premature complexes
50	Functional (rate-related) aberrant intraventricular conduction
63	Nonspecific ST and/or T-wave abnormalities

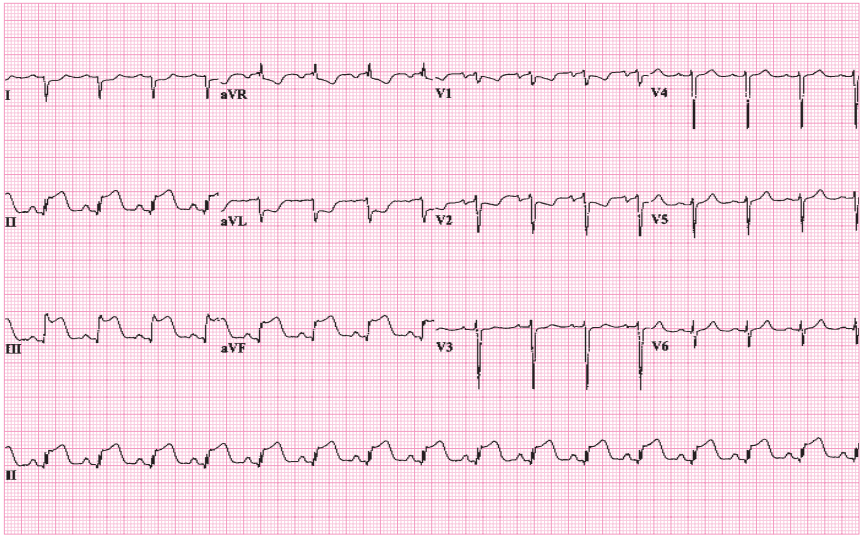




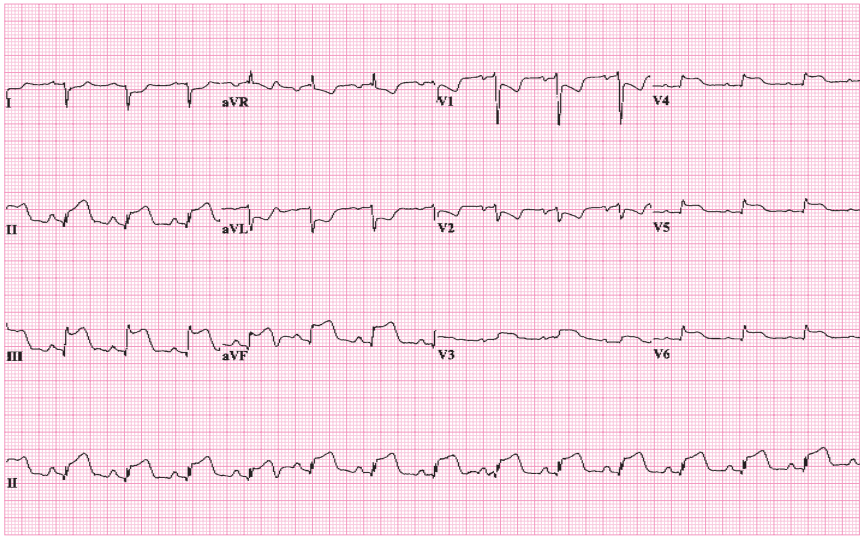
Ashman's Phenomenon describes the situation whereby the repolarization of the right and left bundles is rate related. At a faster sinus rate, the bundles repolarize more quickly and thus an APC is more likely to be conducted than when the sinus rate is slower at the time of the APC. The diagram above depicts how at the slower rate of 77 bpm the atrial impulse was blocked in both the right and left bundles (resulting in a blocked APC). However at the faster rate, the bundles repolarized more quickly and although the timing of the APC was identical to the first one, this impulse was conducted through to the ventricles via the left bundle (the right bundle remained refractory).

ECG 48. 54-year-old male with chest pain:

ECG A



ECG B



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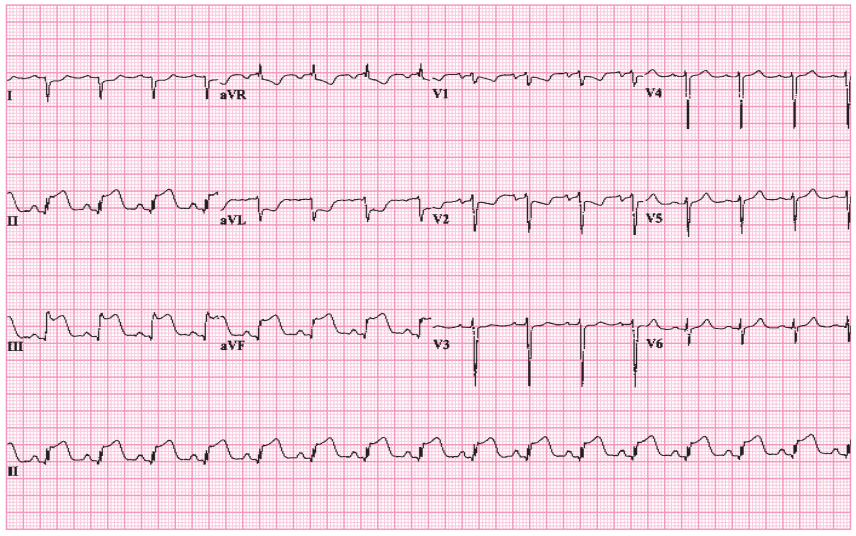
ECG 48A and 48B were obtained in a 54-year-old male with chest pain. ECG 85A shows an acute inferior myocardial infarction with ST-T wave changes of injury and diagnostic Q waves in leads II, III, and aVF. In addition, there is right axis deviation with a small R wave in leads I and aVL, consistent with left posterior fascicular block. The T wave depression in leads I, aVL, V_1 and V_2 may be due to ischemia, reciprocal changes (associated with the inferior injury), or posterior injury. In ECG 85B, right-sided chest leads are recorded – V_1 is placed in the usual V_2 position and the remaining chest leads are placed over the right chest – which allows recording of electrical activity as it passes through the right ventricle. By recording right-sided chest leads in this patient, and injury pattern is observed in leads V_3-V_6 consistent with acute right ventricular injury.

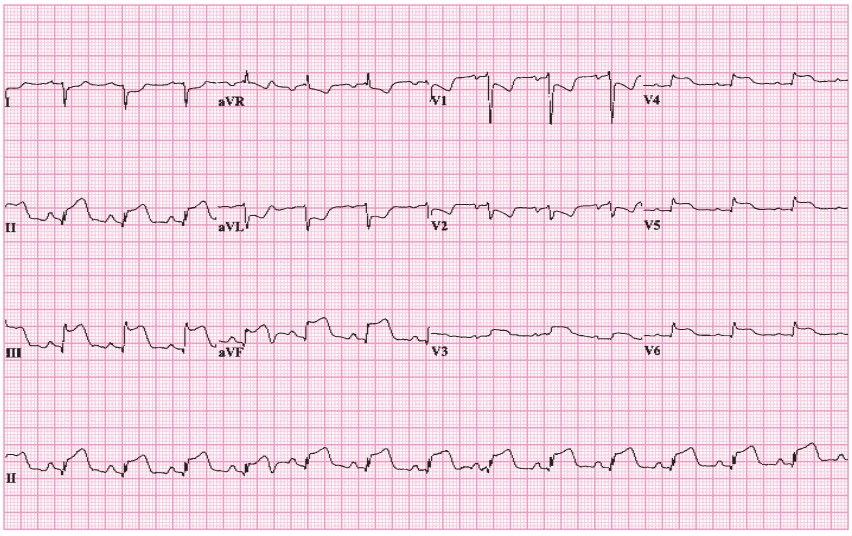
Codes:

06	Left atrial abnormality/enlargement
07	Sinus rhythm
46	Left posterior fascicular block
57	Inferior (age recent or acute)
65	ST and/or T wave abnormalities suggesting myocardial injury

ECG A



ECG B



Questions: ECG 48

1. Right-sided chest leads are superior to the standard 12-lead recording for identifying which of the following abnormalities affecting the right ventricular:

   a. Ischemia
   b. Infarction
   c. Injury
   d. Conduction delay

Answers: ECG 48

1. Typically, the right ventricular contribution to the standard 12-lead ECG is minimal due to the large myocardial mass of the left ventricule and lead positioning over the left chest. Therefore, if there is a concern that myocardial infarction may be involving the right ventricle, it is helpful to place leads on the right side of the chest to allow more accurate recording of the right ventricle. (For right-sided chest leads, V_1 is placed in the usual V_2 position, and V_3R-V_6R are placed in the same locations as V_3-V_6 but over the right chest.) However, the leads over the right ventricle are still strongly influenced by activation of the left ventricle, which proceeds away from the right ventricular leads, often resulting in a Q wave or QS complex even when there is no evidence of infarction. Similarly, the diagnosis of ischemia is difficult because of the influences of the left ventricle on right-sided chest lead recordings. Conduction delay involving the right ventricle, such as right bundle branch block, is still best recorded using the standard 12-lead ECG. In contrast, right-sided chest leads are very useful for the identification of right ventricular injury; ≥ 1 mm ST elevation in lead V_4R has > 90% sensitivity and specificity for right ventricular involvement in the setting of acute inferior wall MI. (Answer: c)

— Quick Review 48 —

Left posterior fascicular block	
• (Left/right) axis deviation with mean QRS axis between ___ and ___ degrees	right 100, 180
• QRS duration between ___ and ___ seconds	0.08, 0.10
• No other factor responsible for ___ axis deviation	right
Inferior MI, probably acute or recent	
• Abnormal Q waves and ST elevation in at least two of leads ____	II, III, aVF
• Associated ST depression is usually evident in leads I, aVL, V_1-V_3 (true/false)	true

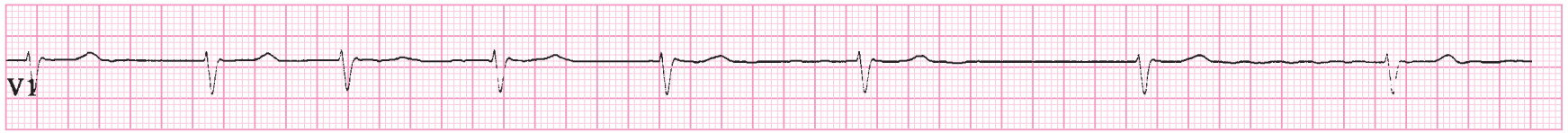
— POP QUIZ —

Find The Imposter

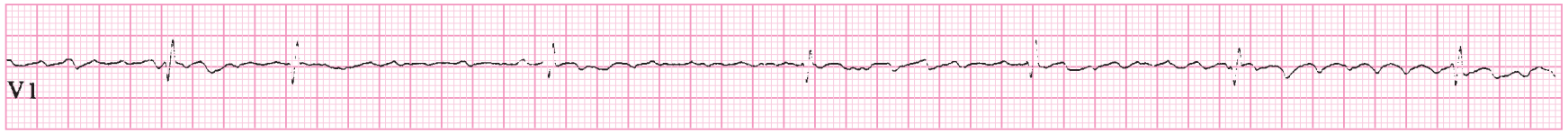
Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

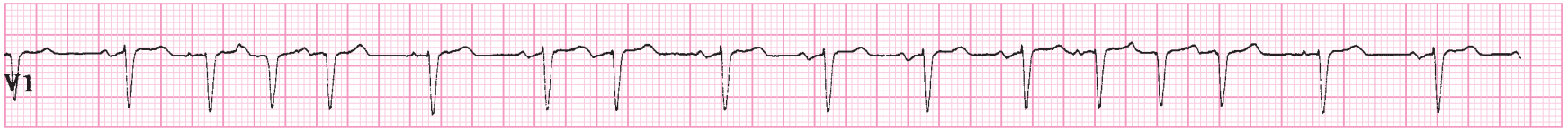
B.



C.



D.



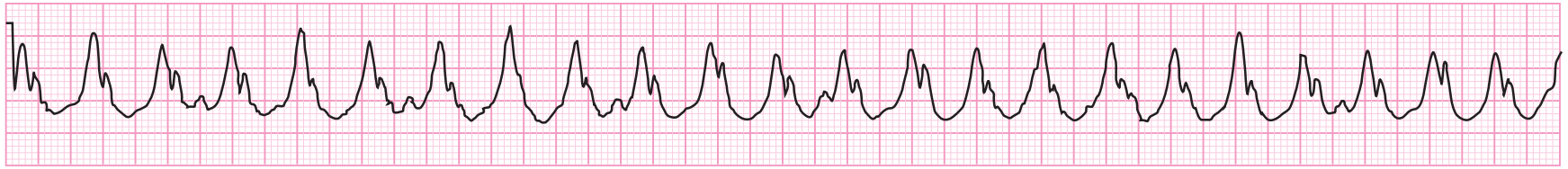
Answer: Tracings A and B have irregular QRS intervals with no clear atrial activity and are consistent with atrial fibrillation. Tracing C shows atrial fibrillation with evidence of fibrillatory waves giving a more coarse appearance to the baseline when compared with tracings A and B. Tracing D shows multifocal atrial tachycardia and is the imposter. It demonstrates irregular QRS intervals with each QRS complex being preceded by a P wave with more than three different P wave morphologies present. The finding of an irregular narrow QRS complex tachycardia with three or more P wave morphologies is consistent with multifocal atrial tachycardia.

— POP QUIZ —

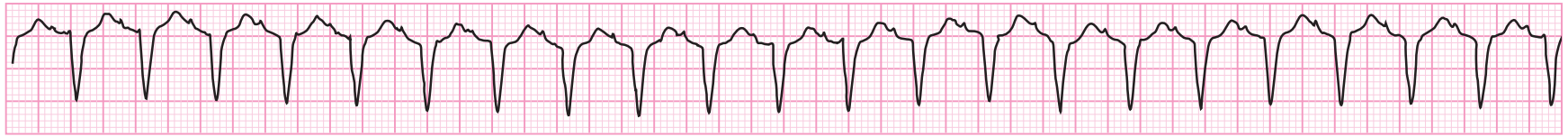
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

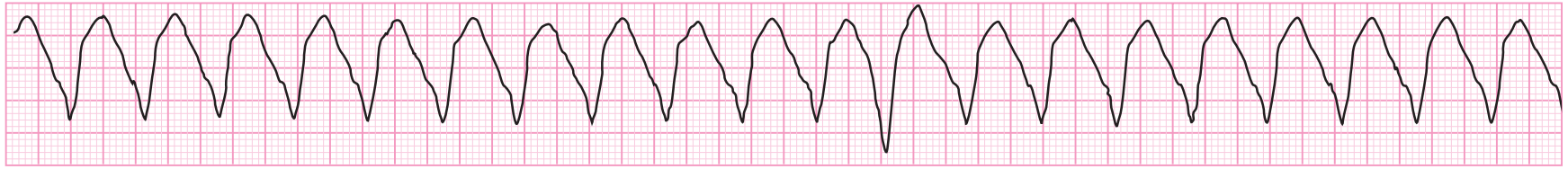
A.



B.



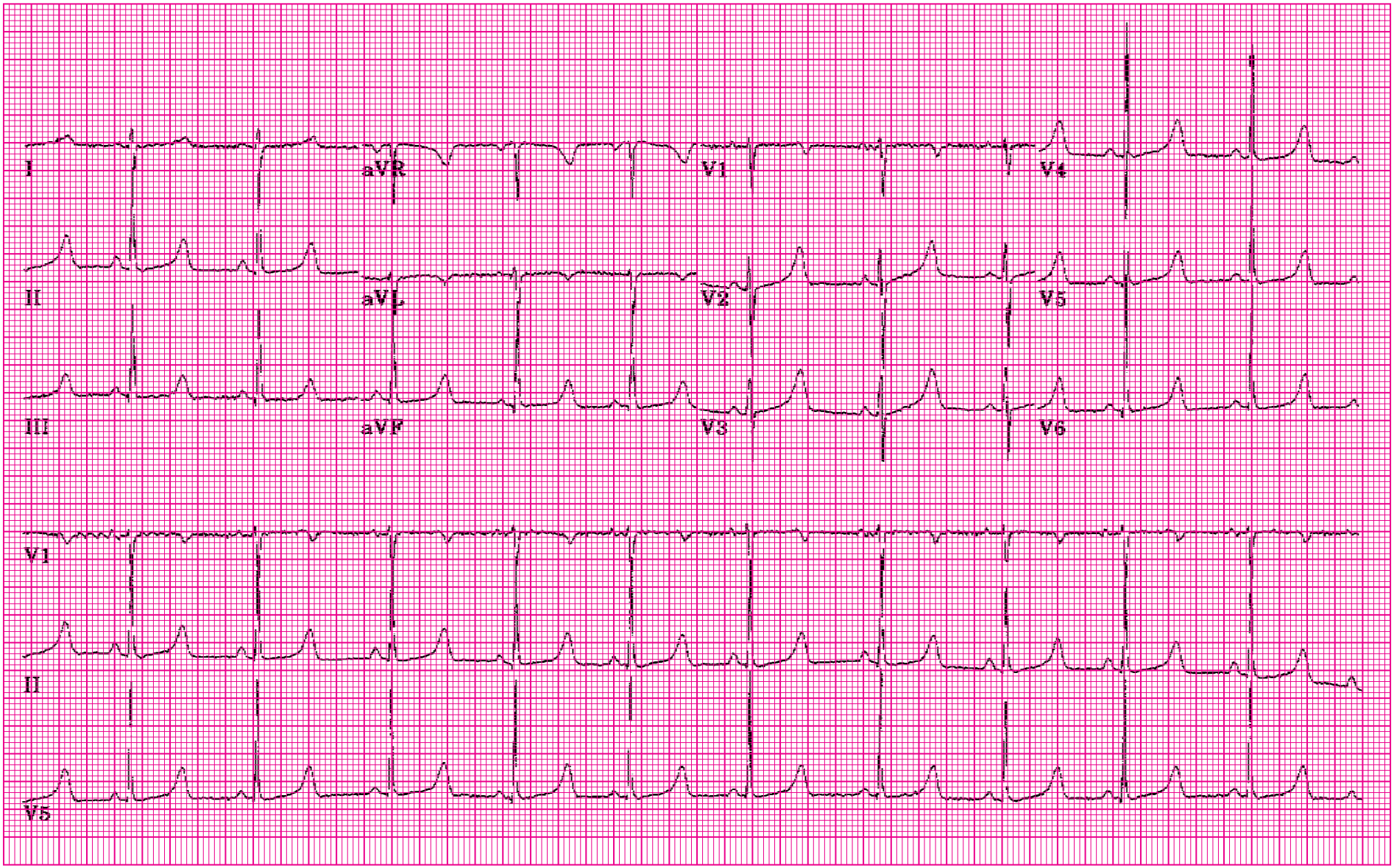
C.



D.

Answer: Tracing A shows a markedly widened QRS complex and P waves that "march through" the rhythm. The QRS prolongation and AV dissociation allow diagnosis of ventricular tachycardia. In tracing C the QRS complexes are markedly prolonged consistent with ventricular tachycardia, although no atrial activity can be identified. In the middle of tracing C is a QRS complex of different morphology, suggesting a "fusion" complex, a finding consistent with ventricular tachycardia. Tracing D demonstrates a relatively narrow QRS tachycardia. However, atrial activity can be observed to be "marching through" the tachycardia supporting the diagnosis of ventricular tachycardia with AV dissociation. Tracing B shows sinus tachycardia with a P wave preceding each QRS complex and is the imposter.

ECG 49. 28-year-old female with weakness:



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- ☐ 03. Incorrect electrode placement
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- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
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- ☐ 32. AV block, 2:1
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- ☐ 36. Left axis deviation (> –30°)
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- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
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- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
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- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
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- ☐ 62. Normal variant, juvenile T waves
- ☐ 63. Nonspecific ST and/or T wave abnormalities
- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
- ☐ 65. ST and/or T wave abnormalities suggesting myocardial injury
- ☐ 66. ST and/or T wave abnormalities suggesting electrolyte disturbances
- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

SUGGESTED CLINICAL DISORDERS

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

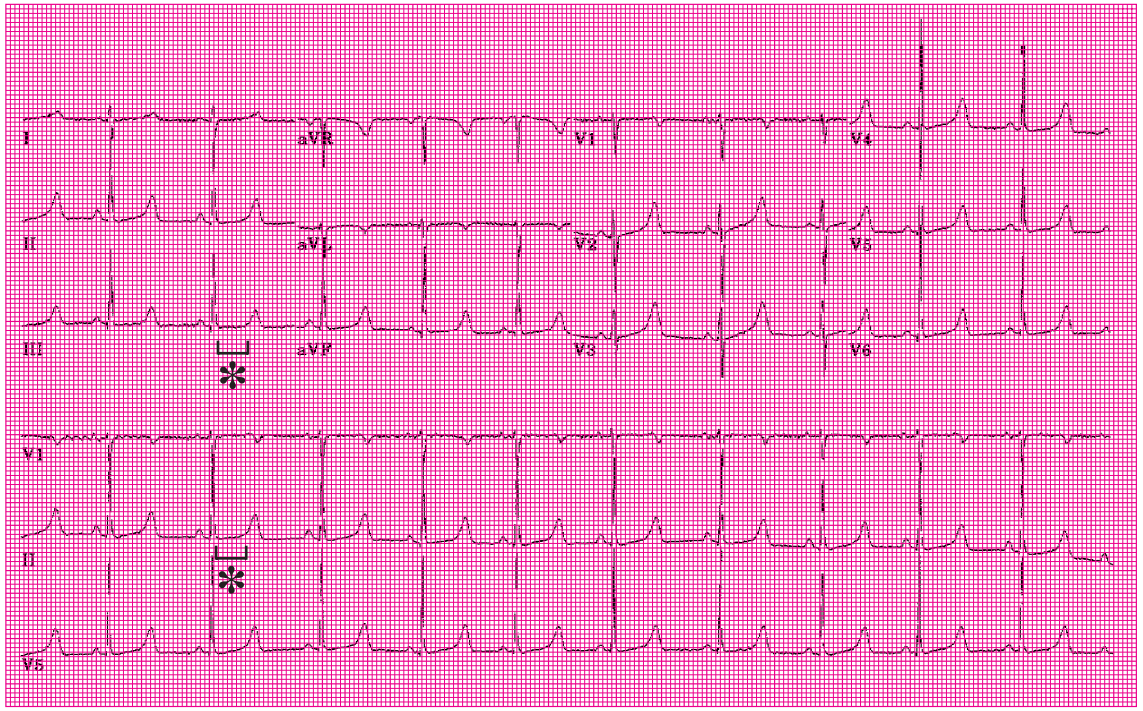
PACED RHYTHMS

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

ECG 49 was obtained from a 28 year-old female with weakness. The ECG shows sinus rhythm at a rate of 65 beats/minute. The corrected QT interval is prolonged (0.52 seconds), primarily due to lengthening of the ST segment (asterisk), which is characteristic of hypocalcemia. Baseline artifact is seen in several leads, especially in V_1. The patient was found to have a serum calcium level of 5.9 mg/dL.

Codes:

04	Artifact
07	Sinus rhythm
66	ST and/or T waves suggesting electrolyte disturbances
68	Prolonged QT interval
77	Hypocalcemia



Questions: ECG 49

1. Hypocalcemia results in prolongation of the QT interval due to:

   a. QT interval and U wave fusion
   b. ST segment and T wave prolongation
   c. Increased QT interval dispersion
   d. ST segment prolongation

2 At a heart rate of 50 BPM, the normal corrected QT interval = __ ± 0.04 seconds

   a. 0.36
   b. 0.38
   c. 0.42
   d. 0.44

Answers: ECG 49

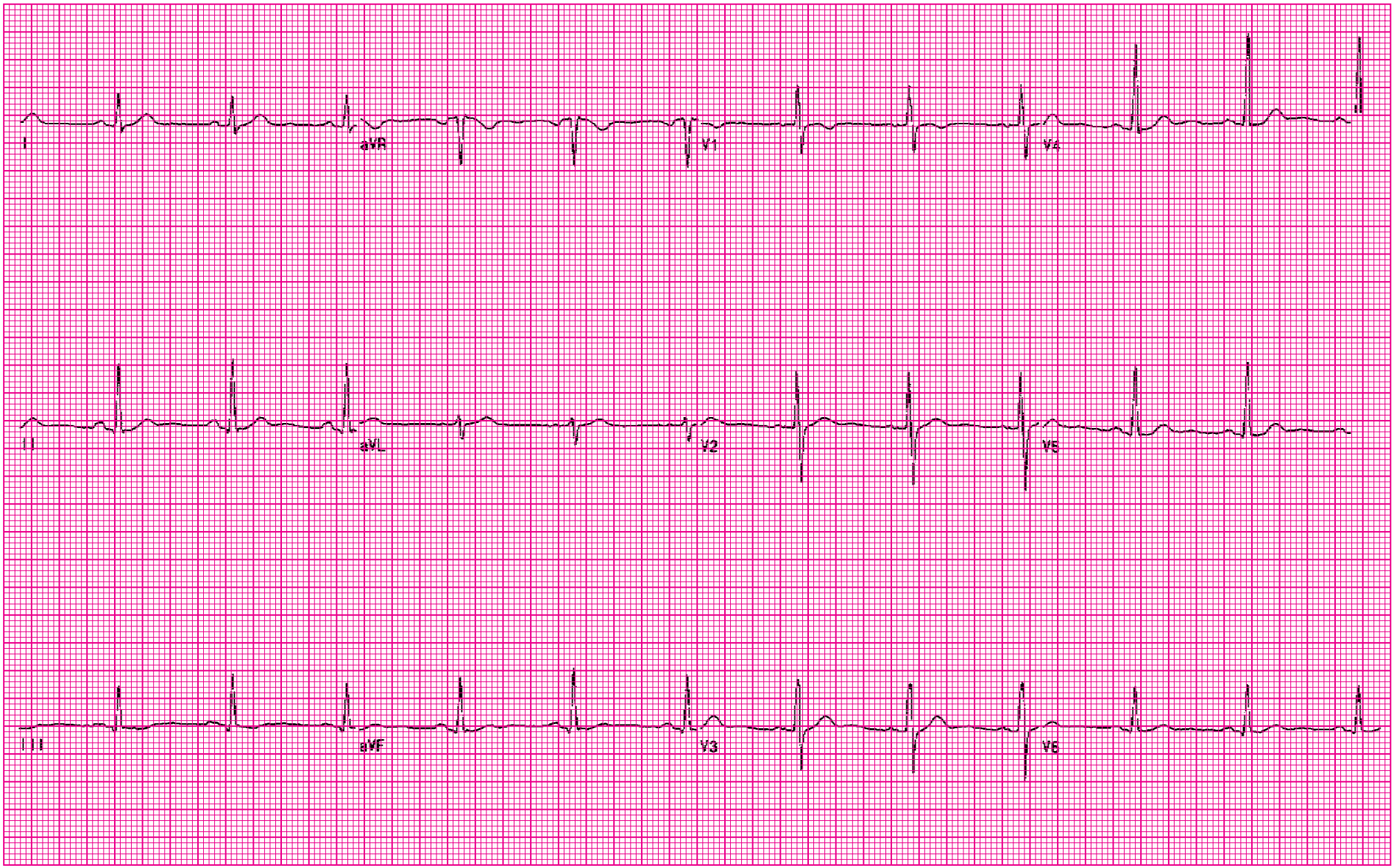
1. Hypocalcemia prolongs the QT interval by lengthening the ST segment (without changing T wave duration). Hypocalcemia is the only electrolyte abnormality associated with isolated ST segment prolongation. (Answer: d)

2. The normal corrected QT interval varies inversely with heart rate, and can be estimated by using 0.40 seconds as the normal QT interval for a heart rate of 70 BPM, then adding (or subtracting) 0.02 seconds for every 10 BPM below (or above) 70 BPM. For a heart rate of 50 BPM, the normal corrected QT interval = 0.04 + (2 x 0.02 seconds) = 0.44 ± 0.04 seconds. (Answer: d)

— Quick Review 49 —

Prolonged QT interval	
• Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval	0.42-0.46 RR
• QT interval varies (directly/inversely) with heart rate	inversely
• The normal QT interval should be (less than/greater than) 50% of the RR interval	less than
Hypocalcemia	
• Earliest and most common finding is prolonged ____ interval	QT
• Occasional flattening, peaking, or inversion of ____ waves	T

ECG 50. 58-year-old male with lung cancer:



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- ☐ 04. Artifact

P WAVE ABNORMALITIES

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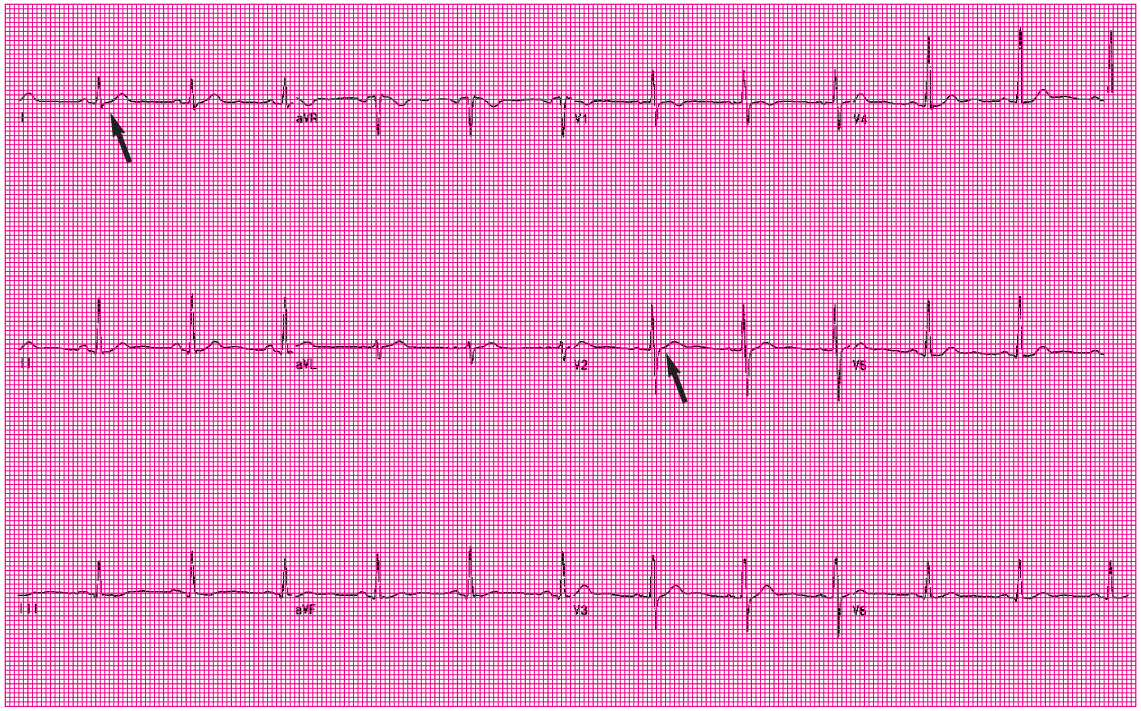
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 50 was obtained from a 58-year-old male with lung cancer. The ECG shows sinus rhythm at a rate of 72 beats/minute. Most notable is the presence of a short QT interval (0.32 seconds) with a very short ST segment (arrows); in the setting of a malignancy such as lung cancer, this finding strongly suggests the presence of hypercalcemia. At the time this ECG was obtained, the patient's serum calcium was 13.5 mg/dL.

Codes:

07	Sinus rhythm
66	ST and/or T waves suggesting electrolyte disturbances
76	Hypercalcemia



Questions: ECG 50

1. ECG changes associated with hypercalcemia include:

   a. Prolongation of the QT interval
   b. Shortening of the ST segment
   c. Flattening of the T wave
   d. Flattening of the P wave
   e. Increase in QRS duration

2. QT interval shortening can be seen with:

   a. Hypocalcemia
   b. Hypercalcemia
   c. Hyperkalemia
   d. Hypokalemia
   e. Beta blockers
   f. Digitalis

Answers: ECG 50

1. Hypercalcemia causes QT interval shortening, primarily due to shortening of the ST segment. There is little (if any) effect on the P wave, QRS complex, or T wave. (Answer: b)

2. Shortening of the QT interval occurs with hypercalcemia, hyperkalemia, digitalis, and beta-blockers. Hypocalcemia and hypokalemia prolong the QT interval. (Answer: b, c, e, f)

— Quick Review 50 —

Hypercalcemia	
• QT interval (lengthening/shortening), primarily due to shortening of the ___ segment	shortening ST
• (Marked/little) effect on the P-QRS-T complex	little

— POP QUIZ —

Electrolyte Abnormalities and the ECG

Instructions: Match the electrolyte disturbance with all ECG abnormalities that apply.

Electrolyte Abnormality	Choose All That Apply	Answer
1. Hyperkalemia 2. Hypokalemia 3. Hypercalcemia 4. Hypocalcemia	a. Widened QRS b. Prolonged ST segment c. Prolonged QT interval d. Shortened QT interval e. Peaked T waves f. Prominent U waves	1. Effects of *hyperkalemia* on the ECG depend on serum K^+ levels: $\underline{K^+ = 5.5 - 6.5 \text{ mEq/L}}$: Tall, peaked, narrow-based T waves, QT interval shortening, and reversible left anterior or left posterior fascicular block $\underline{K^+ = 6.5 - 7.5 \text{ mEq/L}}$: First-degree AV block, flattening and widening of the P wave, ST segment depression, and QRS widening $\underline{K^+ > 7.5 \text{ mEq/L}}$: ECG often shows disappearance of P waves (due to sinus arrest or "sinoventricular conduction"), and LBBB, RBBB, or markedly widened and diffuse intraventricular conduction delay (sine wave pattern). Arrhythmias and conduction disturbances include ventricular tachycardia, ventricular fibrillation, idioventricular rhythm, or asystole. (Answer: a, d, e)
		2. Effects of *hypokalemia* on the ECG include prominent U waves, ST segment depression and flattened T waves (seen in 80% of patients with K^+ levels < 2.7 mEq/L), increased P wave amplitude and duration, and occasional QT prolongation. Arrhythmias and conduction disturbances include paroxysmal atrial tachycardia (PAT) with block, first-degree AV block, Type I second-degree AV block, AV dissociation, VPCs, ventricular tachycardia, and ventricular fibrillation. (Answer: c, f)
		3. Effects of *hypercalcemia* on the ECG include QT shortening (usually due to shortening of the ST segment without a change in the duration of the T wave) and occasional PR prolongation. Typically, there is no effect on the P, QRS, or T wave. (Answer: d)
		4. The primary effect of *hypocalcemia* on the ECG is prolonged QT interval which is due to ST segment prolongation without a change in the duration of the T wave. (Answer: b, c)

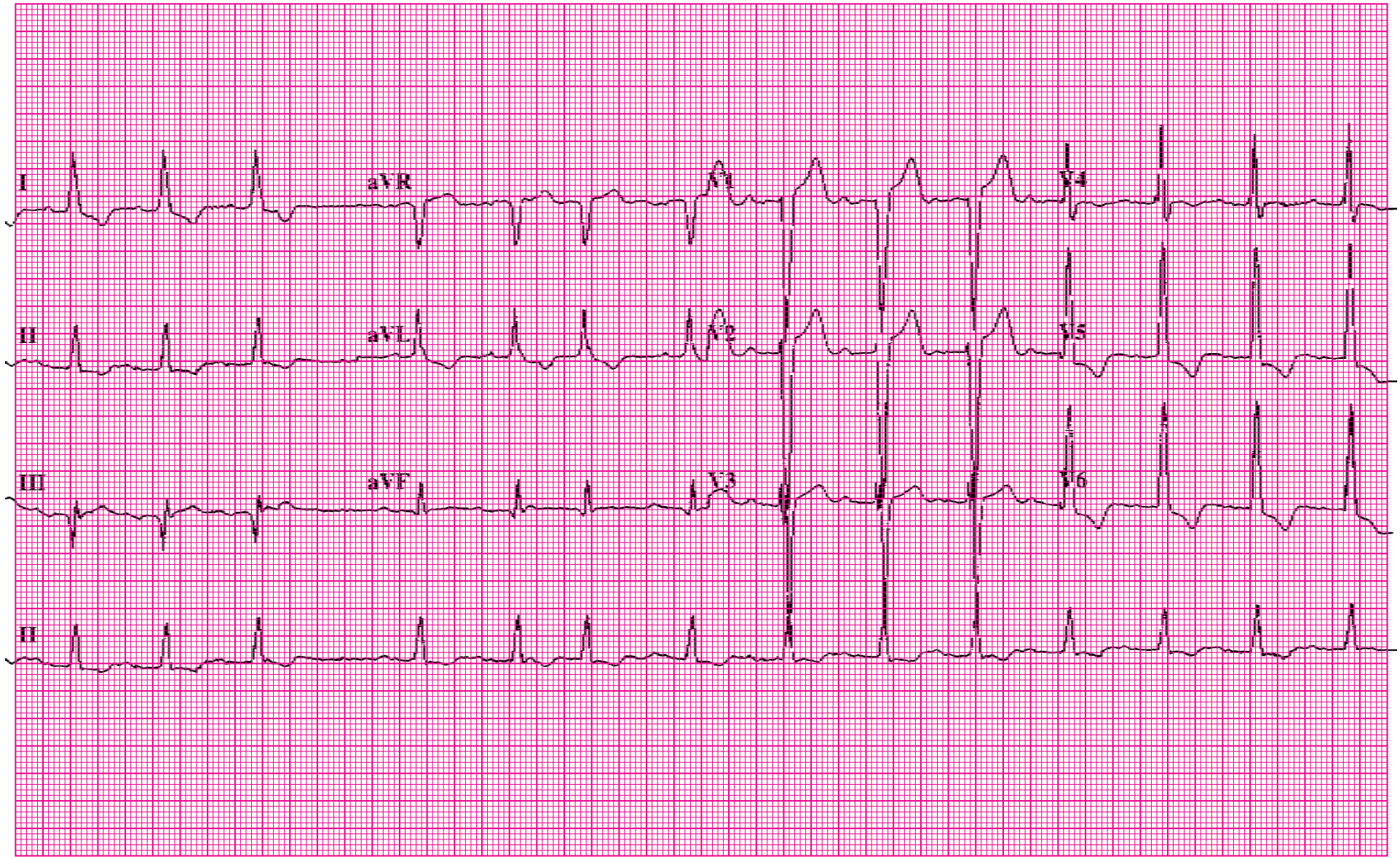
— POP QUIZ —

Differential Diagnosis: QT Interval

Instructions: Determine whether the diagnoses below are associated with a long QT interval or a short QT interval.

Diagnosis	Answer
Hypocalcemia	Long QT (earliest and most common finding), due to prolongation of the ST segment without a change in T wave duration.
Hypercalcemia	QT shortening (usually from shortening of ST segment). Prolongation of PR interval is sometimes seen.
Quinidine effect	Long QT. Prominent U waves and nonspecific ST and T wave changes are common. QRS widening may occur.
Hypomagnesemia	Long QT.
Intracranial hemorrhage	Long QT (often marked). Prominent U waves and large upright or deeply inverted T waves in the precordial leads are common.
Mitral valve prolapse	Long QT. May also see flattened or inverted T waves in II, III, aVF (sometimes in V_1, V_2), ST depression (sometimes in left precordial leads), and prominent U waves.
Myocarditis	Long QT. Q waves and ST elevation sometimes occur and mimic acute MI.
Hyperkalemia	QT shortening. Tall, peaked, narrow-based T waves are common.
Hypothermia	Long QT, due to prolongation of ST segment without a change in T wave duration (only hypothermia and hypocalcemia do this). Osborne (J) waves, prolongation of PR interval, and QRS widening also occur.
Romano Ward Syndrome	Long QT (congenital disorder with normal hearing). Jervell and Lange-Nielson syndrome presents with long QT and congenital deafness.
Digitalis effect	QT shortening. Sagging ST depression with upward concavity, T wave changes (flat, inverted, or biphasic), prominent U wave, and PR prolongation are common.

ECG 51. 82-year-old male with hypertension:



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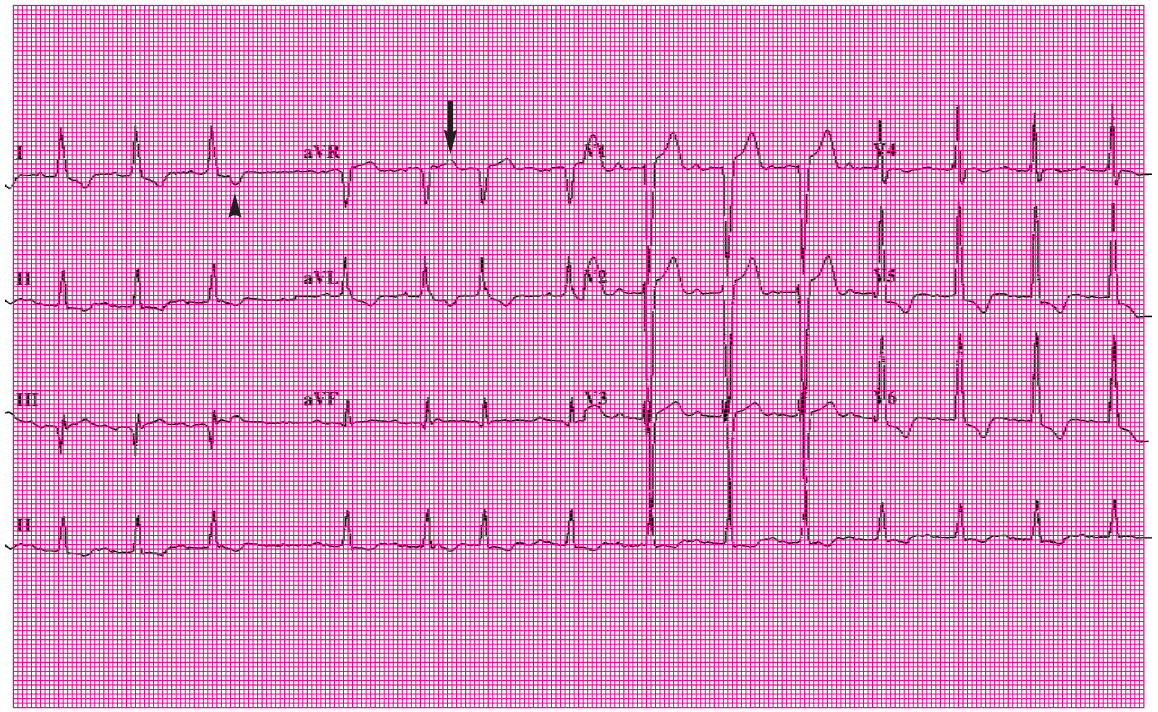
ECG 51 was obtained in an 82-year-old male with hypertension. The ECG shows sinus rhythm with a conducted atrial premature complex (APC) (arrow) and a nonconducted APC (arrowhead). First-degree AV block, nonspecific intraventricular conduct defect, and LVH with ST-T abnormalities are also present. Voltage-based criteria for LVH on this tracing include an R wave in aVL + S wave in V_3 > 28 mm (Cornell criteria), and an S wave in V_1 + R wave in V_5 or V_6 > 35 mm.

Codes:

07	Sinus rhythm
13	Atrial premature complexes
29	AV block, 1°
40	Left ventricular hypertrophy
49	Nonspecific intraventricular conduction disturbance
67	ST and/or T wave abnormalities secondary to hypertrophy



Questions: ECG 51

1. The pause following an atrial premature complex (APC) is typically a noncompensatory pause:

   a. True
   b. False

2. Aberrantly conducted APCs are characterized by:

   a. Initial QRS vector opposite in direction to initial QRS vector of normally conducted beats
   b. LBBB configuration
   c. RBBB configuration

Answers: ECG 51

1. APCs are usually followed by a noncompensatory pause, in which the PP interval containing the APC is less than twice the basic PP interval. In contrast, ventricular premature complexes (VPCs) are usually followed by a fully compensatory pause (PP interval containing the VPC is twice the basic PP interval). (Answer: a)

2. Aberrant conduction of APCs manifests as variable widening or distortion of the normal QRS. The initial QRS vector is in the same direction as the normally-conducted beats, while the more terminal portion of the QRS may be in a different direction. The longer refractory period of the right bundle (compared to the left bundle) increases the likelihood that an APC will conduct down the left bundle, resulting in RBBB morphology. (Answer: c)

— Quick Review 51 —

LVH by voltage only	
Cornell Criteria (most accurate): R wave in aVL + S wave in V_3 > ____ mm in males or > ____ mm in females	28, 20
• **Other commonly used voltage-based criteria**	
▸ Precordial leads (one or more)	
(1) R wave in V_5 or V_6 + S wave in V_1	
▸ > ____ mm if age > 40 years	35
▸ > ____ mm if age 30-40 years	40
▸ > ____ mm if age 16-30 years	60
(2) Maximum R wave + S wave in precordial leads > ____ mm	45
(3) R wave in V_5 > ____ mm	26
(4) R wave in V_6 > ____ mm	20
▸ Limb leads (one or more)	
(1) R wave in lead I + S wave in lead II ≥ ____ mm	26
(2) R wave in lead I ≥ ____ mm	14
(3) S wave in aVR ≥ ____ mm	15
(4) R wave in aVL ≥ ____ mm	12
(5) R wave in aVF ≥ ____ mm	21
• **Non-voltage related criteria for LVH**	
▸ (Left/right) atrial abnormality	left
▸ (Left/right) axis deviation	left
▸ Onset of intrinsicoid deflection > ____ seconds	0.05
▸ Small or absent R waves in leads ____	V_1-V_3
▸ Absent ____ waves in leads I, V_5, V_6	Q
▸ Abnormal ____ waves in leads II, III, aVF	Q
▸ Prominent ____ waves, especially in leads with large R and T waves	U
▸ R wave amplitude in V_6 (greater than/less than) V_5, provided there are dominant R waves in these leads	greater than

— Quick Review 51 —

ST and/or T wave changes secondary to IVCD or hypertrophy	
• ***LVH:*** ST (elevation/depression) & T wave inversion when QRS is mainly positive (leads ___); subtle ST (elevation/depression) & upright T waves when the QRS is mainly negative (leads V_1, V_2)	depression I, V_5, V_6 elevation
• ***RVH:*** ST segment depression & T wave inversion in leads ____ and sometimes in leads II, III, aVF	V_1-V_3
• ***LBBB:*** ST segment & T wave displacement (opposite to/ in same direction as) the major QRS deflection	opposite to
• ***RBBB:*** Uncomplicated RBBB has little ST displacement (true/false). T wave vector is (opposite to/in same direction as) the terminal slurred portion of the QRS	true opposite to

Don't Get Confused!

Multifocal Atrial Tachycardia

Atrial rate >100 per minute with ≥ 3 P wave morphologies and varying PR, RR, and RP intervals

<u>May be confused with</u>:

Sinus tachycardia with multifocal APCs

Demonstrates one dominant atrial pacemaker (i.e., the sinus node). In multifocal atrial tachycardia, *no* dominant atrial pacemaker (i.e., no dominant P wave morphology) is present.

Atrial fibrillation/flutter

Atrial fibrillation/flutter lacks an isoelectric baseline. In contrast, multifocal atrial tachycardia demonstrates a distinct isoelectric baseline and P waves.

ECG 52. 53-year-old male with severe chest pressure who lost consciousness during this ECG:

GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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REPOLARIZATION ABNORMALITIES

- ☐ 61. Normal variant, early repolarization
- ☐ 62. Normal variant, juvenile T waves
- ☐ 63. Nonspecific ST and/or T wave abnormalities
- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
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- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

SUGGESTED CLINICAL DISORDERS

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

PACED RHYTHMS

- ☐ 90. Atrial or coronary sinus pacing
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
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ECG 52 was obtained in a 53-year-old male who presented with severe chest and neck pressure and lost consciousness during the acquisition of this 12-lead ECG. The ECG shows chaotic, irregular deflections of varying amplitude without distinct P waves, QRS complexes, or T waves, consistent with ventricular fibrillation/ventricular flutter at a rate of 248 beats/minute.

Codes: 28 Ventricular fibrillation

Questions: ECG 52

1. The likelihood of successful resuscitation out of ventricular fibrillation decreases by approximately ___ % per minute from onset of the dysrhythmia:

   a. 2%
   b. 5%
   c. 7.5%
   d. 10%

2. The two most frequent causes of ventricular fibrillation are:

   a. Aortic stenosis
   b. Drug-induced or congenital long QT
   c. Coronary artery disease
   d. Pulmonary embolism
   e. Cardiomyopathy (including dilated and hypertrophic etiologies)

Answers: ECG 52

1. Ventricular fibrillation (VF) is a lethal dysrhythmia unless it is promptly terminated. Electrocardioversion is nearly always successful at restoring sinus rhythm when VF is shocked within the first minute. The success rate of cardioversion falls off rapidly with elapsed time. Overall, the rate of survival from VF in the community has been reported to vary between 4% and 33%, depending upon the rapidity of which the emergency medical personnel are able to attend to the victim. (Answer: d)

2. Coronary atherosclerosis and its consequences (myocardial ischemia or infarction) are responsible for approximately 80% of sudden cardiac death in the United States. Cardiomyopathy (ischemic, non-ischemic, and hypertrophic) is the second most common precipitating factor. The degree of left ventricular impairment is closely correlated with the risk of sudden cardiac death. Pulmonary embolism, aortic stenosis, and long QT syndromes are also associated with increased risk of ventricular fibrillation, but are less frequent causes compared to coronary artery disease and cardiomyopathy. (Answer: c, e)

— Quick Review 52 —

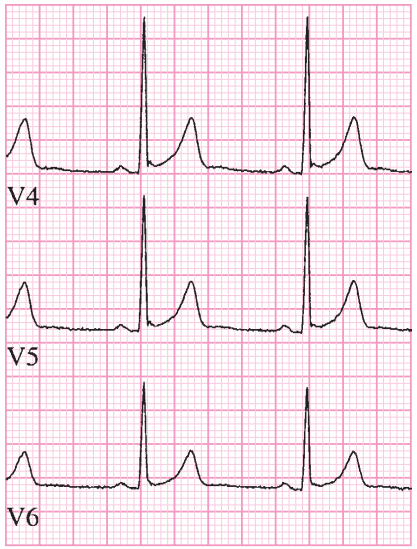
Ventricular fibrillation	
Extremely rapid and (regular/irregular) ventricular rhythm with:	irregular
• Chaotic, irregular deflections of (constant/varying) amplitude and duration	varying
• (Absence/presence) of distinct P waves, QRS complexes, and T waves	absence

— POP QUIZ —

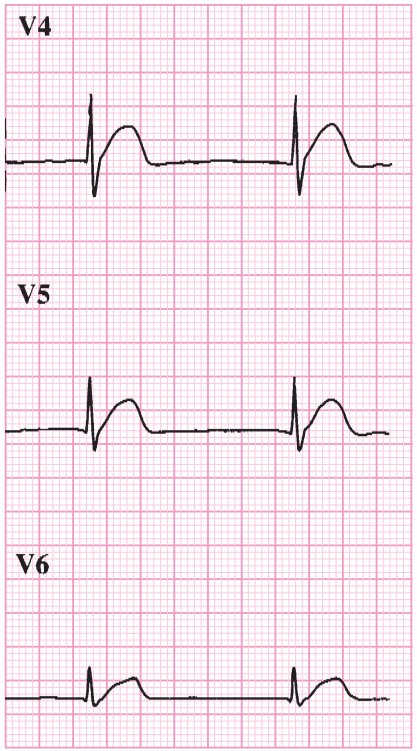
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

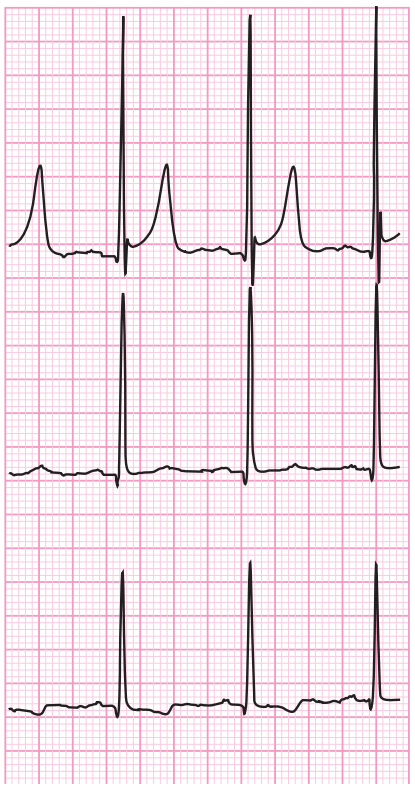
A.



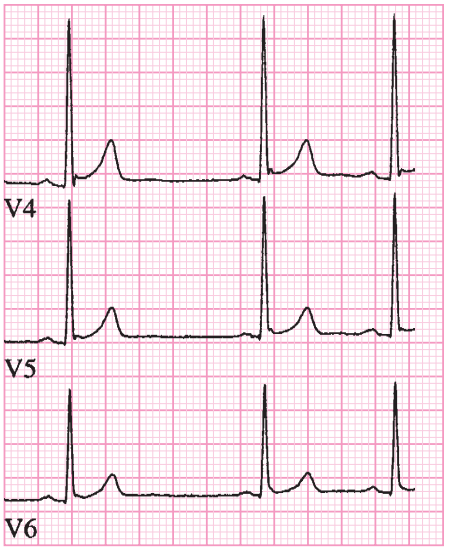
B.



C.



D.



Answer: Tracings A, C, and D are examples of normal variant early repolarization ST segment elevation, with a concave upward configuration of the ST elevation ending with a symmetrical upright T wave. Tracing B is shows a lateral myocardial injury pattern and is the imposter. In contrast to early repolarization, the configuration of the ST segment elevation of acute myocardial injury is convex upward.

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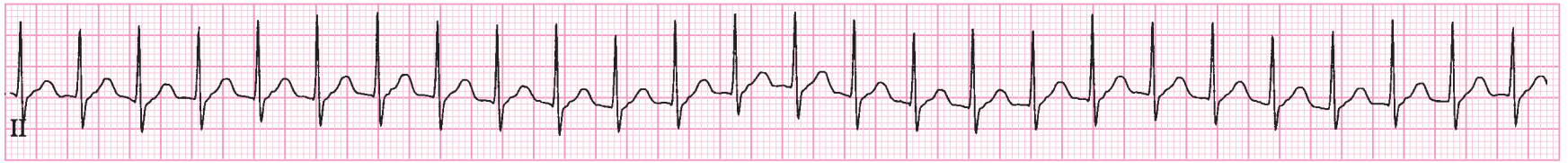
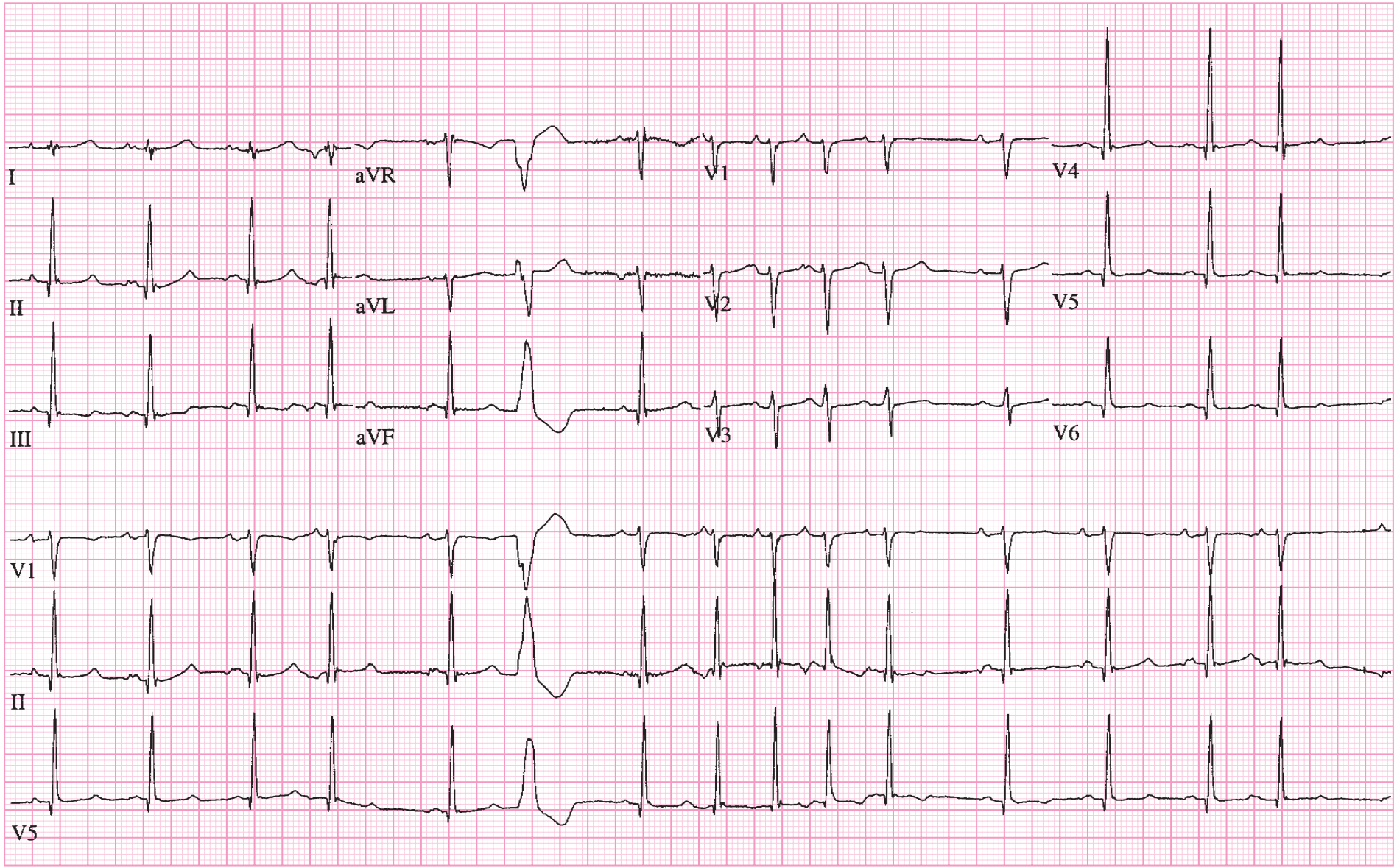


D.

Answer: Tracings A, B, and C demonstrate a regular, narrow QRS tachycardia with possible atrial activity at the end of the QRS complex in tracing C but no obvious atrial activity in tracings A or B. These regular, narrow QRS tachycardias with either P waves at the end of the QRS or no discernible atrial activity are consistent with SVT. Tracing D shows atrial flutter with 2:1 AV conduction and is the imposter. The flutter waves are best seen after each QRS complex. The flutter waves, which are negative, show an irregular relationship to the preceding QRS complex and therefore are not T waves but rather flutter waves following the QRS.

ECG 53. 59-year-old female with palpitations:



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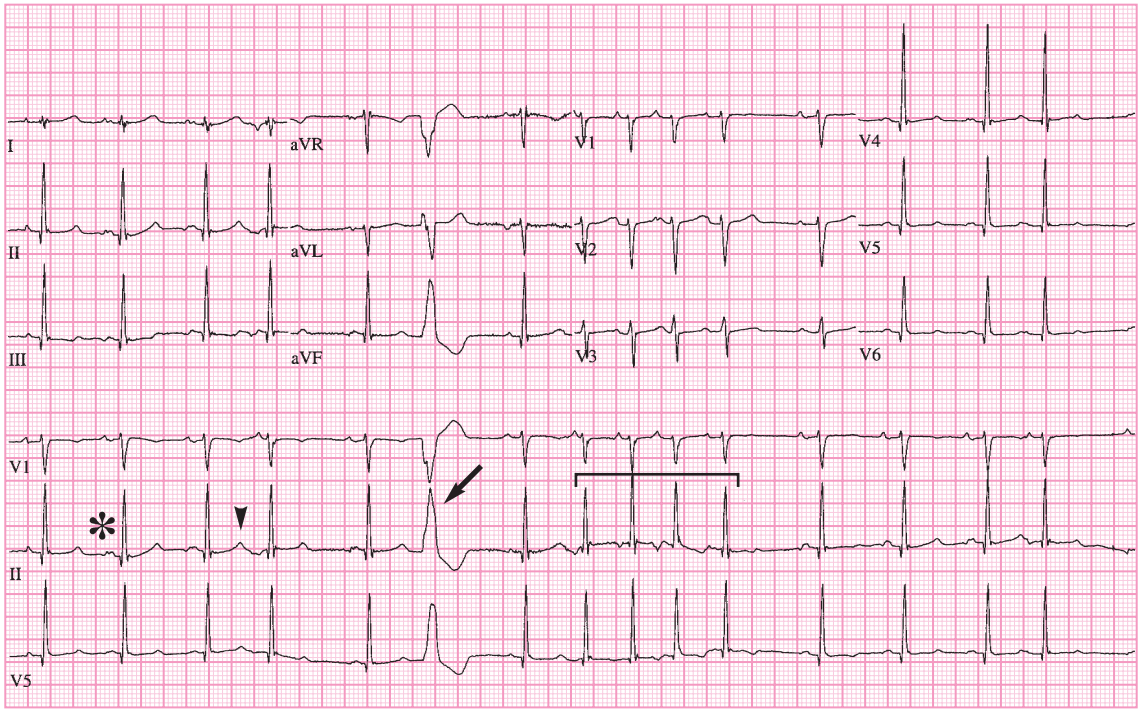
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 53 was obtained from a 59-year-old female with palpitations. The ECG shows sinus rhythm (asterisk on sinus beat) at a rate of 90 beats/minute with occasional APCs (arrowhead) and VPCs (arrow). In the middle of the tracing (double asterisk), there is a 4-beat run of atrial tachycardia at a rate of 150 beats/minute.

Codes:

07	Sinus rhythm
13	Atrial premature complexes
15	Atrial tachycardia
23	Ventricular premature complexes



Questions: ECG 53

1. Characteristics of atrial tachycardia include:

   a. Atrial rate between 120-180 per minute
   b. Regular rhythm
   c. P waves similar to sinus rhythm

2. Which of the following statement about atrial premature contractions are true:

   a. QRS complex is always similar in morphology to the QRS complex during sinus rhythm
   b. PR may be normal, increased, or decreased
   c. The post-extrasystolic pause is usually noncompensatory
   d. The QRS morphology of aberrantly conducted APCs is most often an RBBB pattern

Answers: ECG 53

1. Atrial tachycardia is a regular rhythm with nonsinus P waves at rates of 100-240 per minute. Atrial tachycardia should not be confused with multifocal atrial tachycardia, which is an irregular rhythm with ≥ 3 P wave morphologies that can be mistaken for atrial fibrillation. (Answer: a, b)

2. An atrial premature complexes (APC) is characterized by the presence of a P wave that is abnormal in configuration and premature relative to the normal PP interval. The QRS complex is usually similar in morphology to the QRS complex present during sinus rhythm. However, with aberrantly conducted APCs, the QRS morphology is most often RBBB pattern due to the longer refractory period of the right bundle compared to the left bundle, but it can be LBBB pattern or variable. The PR interval of APCs can be normal, increased, or decreased, and the post-extrasystolic pause is usually noncompensatory (i.e., the interval from the preceding normal P wave to the normal P wave following the APC is less than two normal PP intervals). (Answer: b, c, d)

— Quick Review 53 —

Atrial premature complexes

- P wave is (normal/abnormal) in configuration — abnormal
- QRS complex is (similar/different) in morphology to the QRS complex present during sinus rhythm — similar
- PR interval may be normal, increased, or decreased (true/false) — true
- The post-extrasystolic pause is usually (compensatory/noncompensatory) — noncompensatory

Atrial tachycardia

- Three or more consecutive (sinus/nonsinus) beats at an atrial rate of 100-240 bpm — nonsinus
- P wave is (always/sometimes) visualized — sometimes
- QRS follows each P wave unless ____ AV block is present — 2° or 3°

— Quick Review 53 —

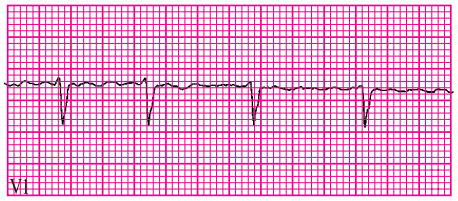
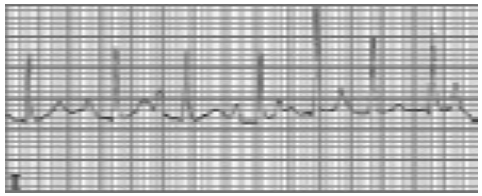
Ventricular premature complexes, uniform, fixed coupling

- A wide, notched or slurred ____ complex that is premature relative to the normal RR interval and is not preceded by a ____ wave — QRS; P
- QRS duration is almost always > ____ seconds — 0.12
- Initial direction of the QRS is often (similar to/different from) the QRS during sinus rhythm — different from
- Secondary ST & T wave changes in the (same/opposite) direction as the major deflection of the QRS (i.e., ST depression & T wave inversion in leads with a dominant _____wave; ST elevation and upright T wave in leads with a dominant _____ wave or ____ complex) — opposite; R; S, QS
- Coupling interval is constant or varies by < ____ seconds — 0.08
- Morphology of VPCs in any given lead is (the same/different) — the same
- Retrograde capture of atria may occur (true/false) — true
- A full ____ pause (PP interval containing the VPC is twice the normal PP interval) is usually evident — compensatory

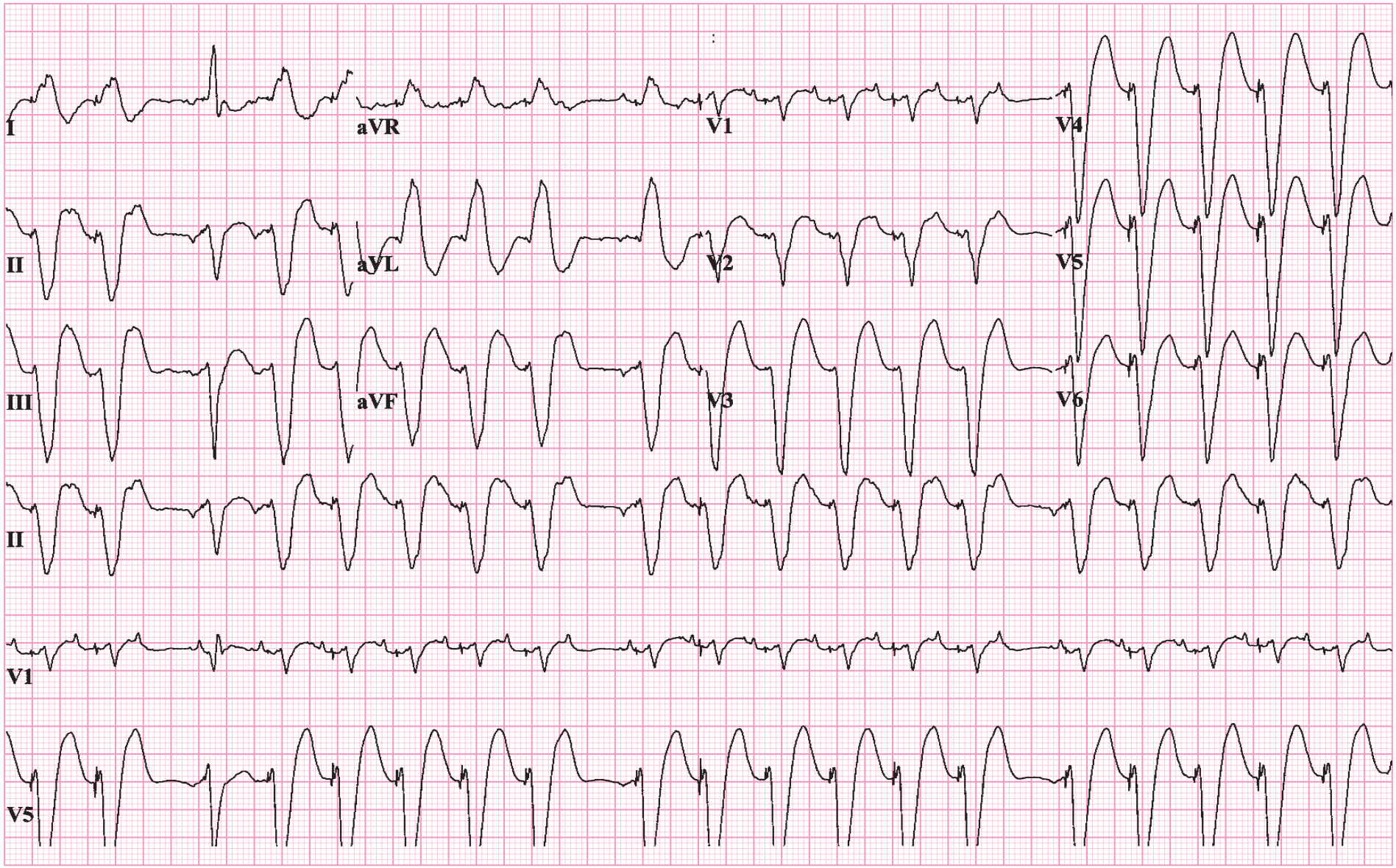
— POP QUIZ —

Rhythm Recognition: HR > 100; Narrow QRS; Irregular RR Interval

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Atrial fibrillation. Description: Absent P waves, with totally irregular atrial activity represented by fibrillatory (f) waves of varying amplitude, duration, and morphology causing random oscillation of the baseline. Ventricular rhythm is typically irregularly irregular, and occurs at a rate of 100-180 per minute in the absence of drugs. Atrial activity is best seen in leads V_1, V_2, II, III, and aVF. Digoxin toxicity can cause regularization of the QRS, representing complete heart block with junctional tachycardia. Note: In the absence of AV nodal blocking drugs, a ventricular rate < 100 per minute suggests coexistent AV conduction system disease. Conditions mimicking atrial fibrillation include multifocal atrial tachycardia, paroxysmal atrial tachycardia with block, or atrial flutter with variable AV block.
	Answer: Multifocal atrial tachycardia (MAT). Description: Irregular atrial rhythm with at least three different P wave morphologies (originating from separate atrial foci) at an atrial rate > 100 per minute with varying PP and PR intervals. P waves may be blocked (not followed by a QRS), or may be conducted with a narrow or aberrant (wide) QRS complex. Can be confused with atrial fibrillation/flutter or sinus tachycardia with multifocal APCs. MAT is usually associated with some form of lung disease (e.g., COPD, cor pulmonale, hypoxia, aminophylline therapy).
	Answer: Atrial flutter with variable AV block. Description: Rapid, regular atrial undulations (flutter or "F" waves) usually at a rate of 240-340 per minute. Flutter waves are typically inverted in leads II, III and aVF, and manifest small positive upright deflections in V_1; "atypical flutter" can show upright F waves in the inferior leads. QRS complexes may be narrow or wide (if underlying aberrancy or bundle branch block). AV conduction ratio (ratio of flutter waves to QRS complexes) is usually a fixed, even number (e.g., 2:1, 4:1), but variable conduction sometimes occurs (as in the present tracing). Flutter waves sometime deform the QRS, ST, T waves to mimic intraventricular conduction delay or myocardial ischemia/injury.

ECG 54. 26-year-old male with palpitations:



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

PACED RHYTHMS

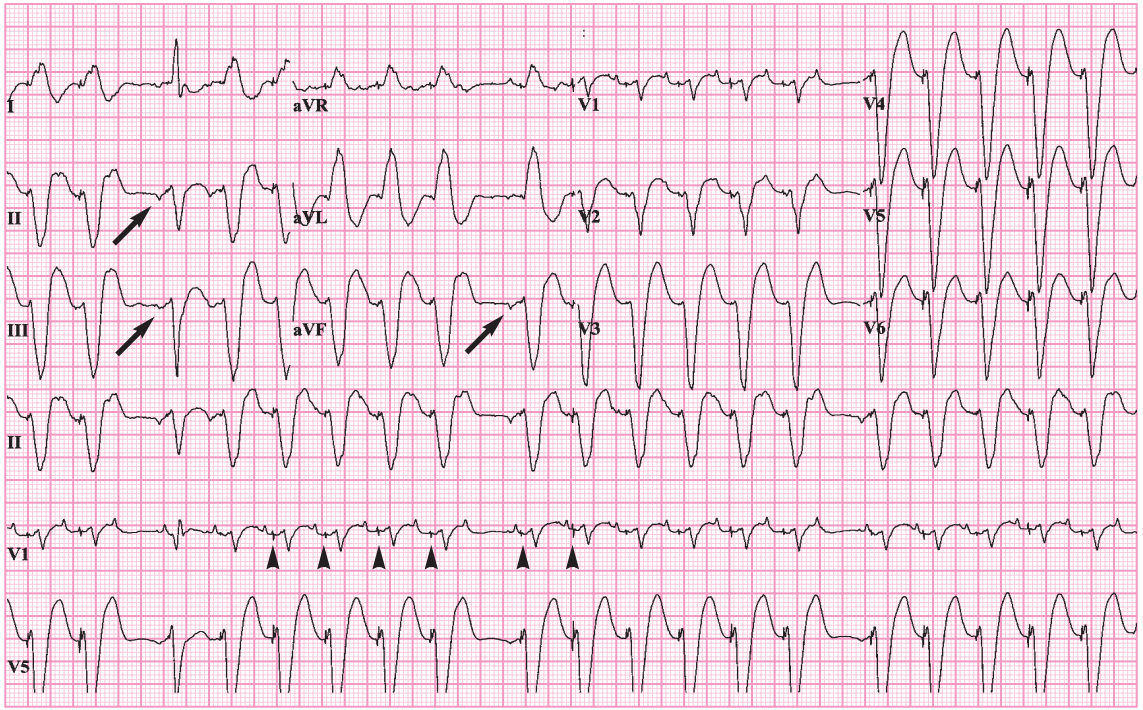
- ☐ 90. Atrial or coronary sinus pacing
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 54 was obtained in an 26-year-old male with palpitations. The ECG shows an atrial tachycardia at approximately 140 beats/minute with variable AV conduction and group beating. The P wave morphology is negative in leads II, III, and aVF (arrows), indicating an atrial focus remote from the sinus node (normal sinus P wave morphology is positive in leads II, III, and aVF). Pacing spikes are observed before each QRS complex (arrowheads), but no pacing spike is present before the P waves, consistent with a dual chamber pacemaker that is sensing the atrium and pacing the ventricle. (The ventricular lead is in the right ventricular apex, thus pacing the ventricle with the expected pattern for this pacing location of left bundle branch block with left axis deviation.) The unique finding on this ECG is the group beating plus P waves that are not followed by a paced QRS complex. At initial glance, this may be interpreted as a failure of pacemaker output. However, closer inspection demonstrates gradual prolongation of the AV delay between P wave and the pacemaker spike. In addition, the atrial tachycardia rate is rapid (140 beats/minute) and most likely above the programmed upper rate limit for the pacemaker. The pacemaker is demonstrating a feature termed "upper rate behavior pacing," in which the ventricular rate cannot exceed the upper programmed rate for the pacemaker even though the atrial rate is more rapid. To maintain the ventricular rate at or under the upper programmed rate, AV Wenckebach occurs, as depicted on this ECG. This is normal pacemaker behavior.

Codes:

15 Atrial tachycardia

92 Dual-chamber pacemaker (DDD)



Questions: ECG 54

1. Which of the following statements about pacemakers are true:

   a. DVI pacemakers pace only the ventricle, but senses both the atrium and ventricle
   b. VOO pacemakers pace the ventricle asynchronously
   c. VVI-R is a rate-responsive ventricular pacemaker
   d. DDD pacemakers can function in either a triggered or inhibited mode

2. A dual chamber (DDD) pacemaker senses:

   a. The ventricle only
   b. The atrium only
   c. The atrium and the ventricle
   d. Neither the atrium nor the ventricle

3. A normally-functioning DDD pacemaker results in:

   a. An atrial paced complex followed by a native QRS after an AV interval less than the programmed AV interval of the pacemaker
   b. An atrial paced complex followed by a ventricular paced complex at the programmed AV interval
   c. A native P wave followed by a paced ventricular complex at the programmed AV interval
   d. A native P wave followed by a native QRS at rates above the programmed pacemaker rate

Answers: ECG 54

1. Pacemakers are identified by a 3-letter pacemaker code. The first letter indicates the chamber *paced*: atrial (A), ventricular (V), or both (D). The second letter indicates the chamber *sensed*: atrial (A), ventricular (V), or neither (O). The third letter indicates the pacing *mode:* triggered (T), inhibited (I), dual (D), or asynchronous (O). A rate-responsive pacemaker is indicated by a fourth letter, R. All statements in question 1 are correct except "a", since a DVI pacemaker paces both the atrium and ventricle but senses only the ventricle. (Answer: b, c, d)

2. DDD pacemakers sense and pace the right atrium and ventricle. This results in inhibition or triggering of pulse generator impulses on the atrial and/or ventricular channels. (Answer: c)

3. A normally-functioning DDD pacemaker can show various combinations of atrial paced beats and/or native P waves followed by ventricular paced beats and/or native QRS complexes. The exact combination depends on the programmed AV interval/pacemaker rate and underlying rhythm. All four statements are correct. (Answer: all)

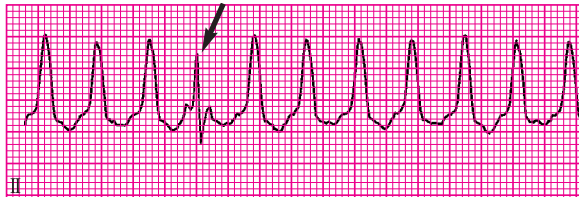
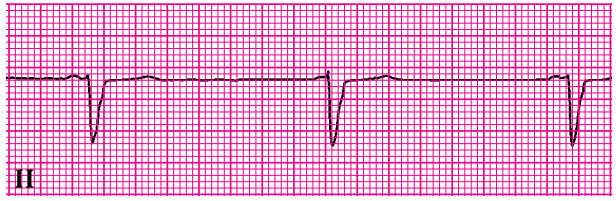
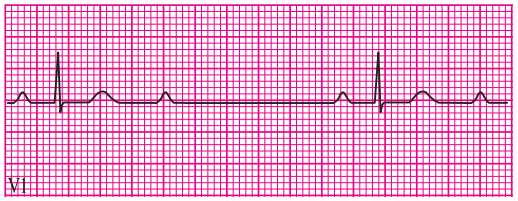
— Quick Review 54 —

Atrial tachycardia	
• Three or more consecutive (sinus/nonsinus) beats at an atrial rate of 100-240 bpm	nonsinus
• P wave is (always/sometimes) visualized	sometimes
• QRS follows each P wave unless ____ AV block is present	2° or 3°
Dual chamber, atrial-sensing pacemaker	
• For atrial sensing, need to demonstrate inhibition of (atrial/ventricular) output and/or triggering of the (atrial/ventricular) stimulus in response to intrinsic atrial depolarization	atrial ventricular
• Includes ____ and possibly VAT or VDD pacemakers	DDD

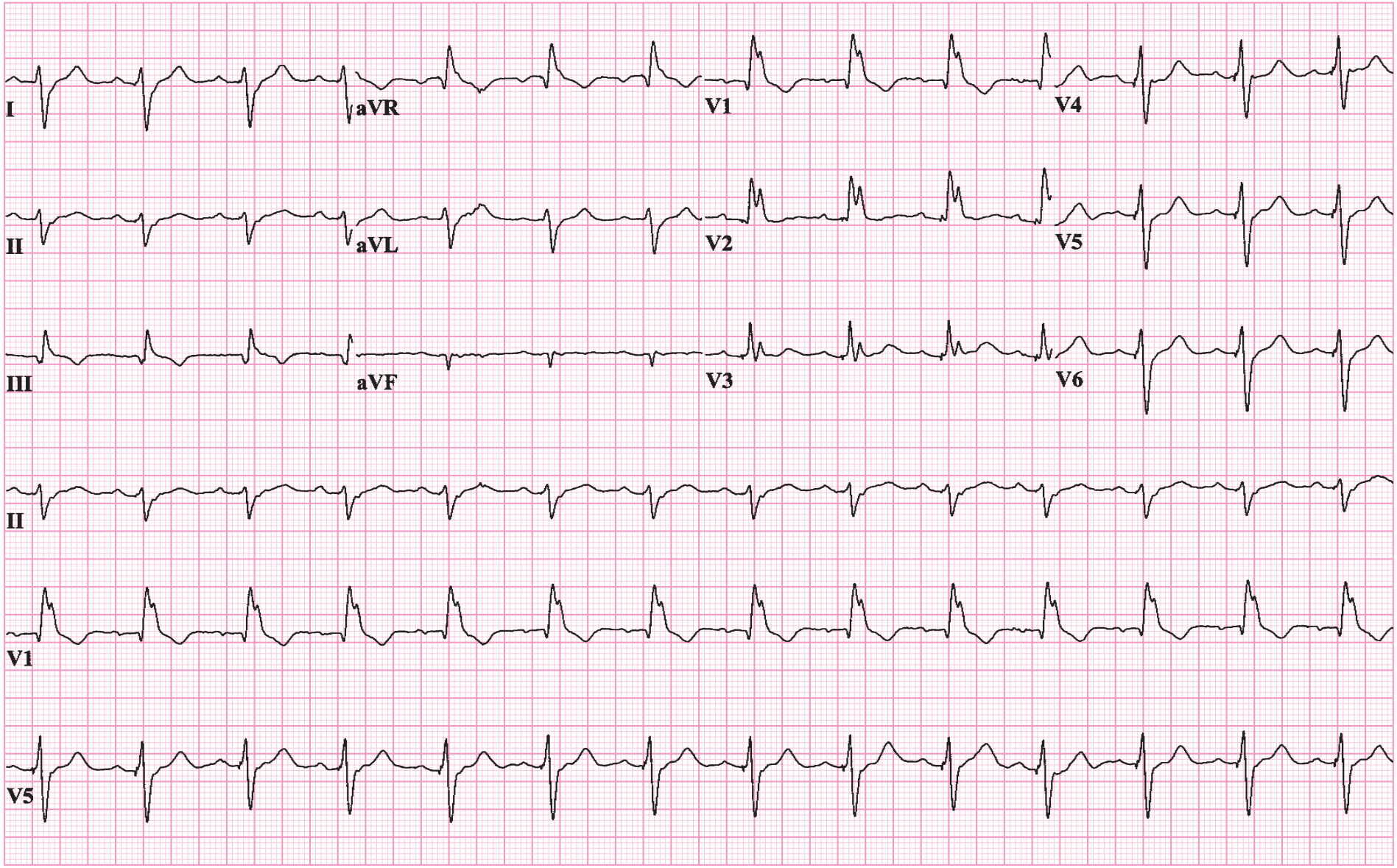
— POP QUIZ —

Pattern Recognition: A-V Interactions

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Fusion complex b. Can be seen with ventricular tachycardia c. Results from simultaneous activation of ventricle from 2 sources d. Echo beat e. Form of nonsustained reentry f. Capture complex g. Suggest diagnosis of SVT in setting of wide QRS tachycardia h. Occurs when atrial impulse stimulates the ventricle during VT i. Atrial and ventricular rhythms occur independant of each other j. AV dissociation k. Ventriculophasic sinus arrhythmia	*Ventricular capture complex* occurs when atrial impulse stimulates the ventricle during ventricular tachycardia. The "captured" ventricle results in a QRS complex similar to that during sinus rhythm. The presence of a ventricular capture complex(es) in the setting of a wide QRS tachycardia strongly suggests the diagnosis of ventricular tachycardia. (Answer: b, f, h)
		*AV dissociation* occurs when atrial and ventricular rhythms act independently of each other. In most cases, the ventricular rate is equal to or faster than the atrial rate, either due to acceleration of a subsidiary pacemaker above the atrial rate or slowing of the atrial rate below the intrinsic rate of the subsidiary ventricular pacemaker. Also applies to junctional rhythms. (Answer: b, i, j)
		*Ventriculophasic sinus arrhythmia* occurs during partial or complete AV block when the PP interval containing a QRS complex is shorter than the PP interval without a QRS complex. (Answer: k)

ECG 55. 73-year-old female on routine ECG:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

PACED RHYTHMS

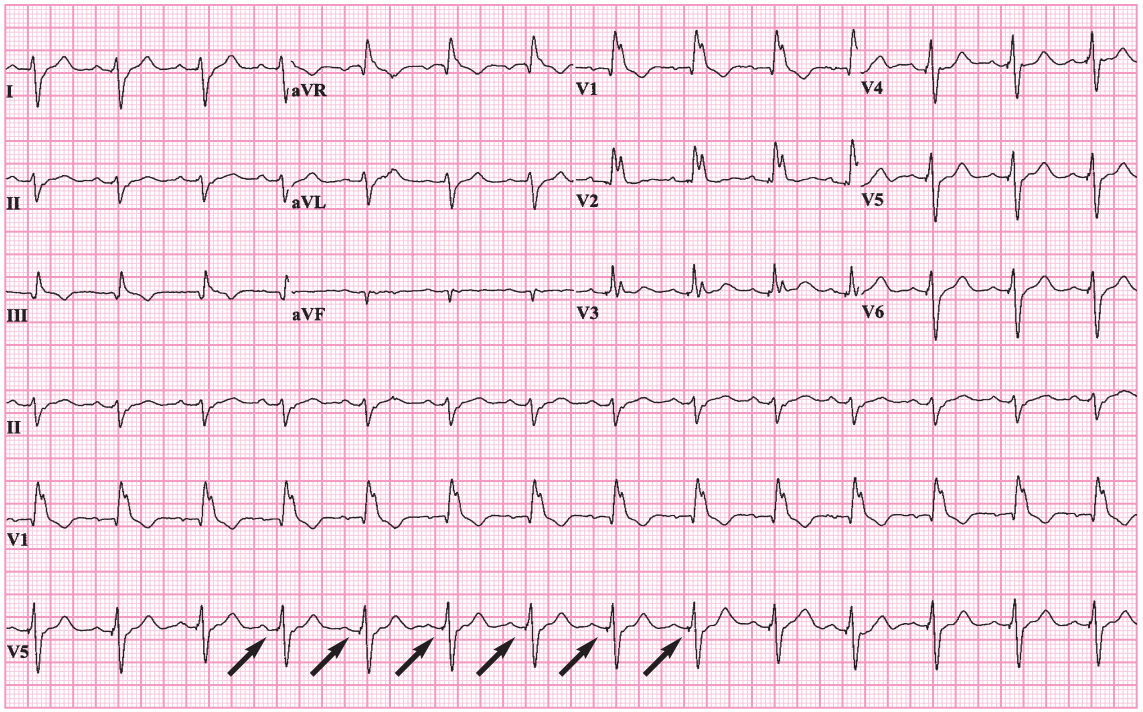
- ☐ 90. Atrial or coronary sinus pacing
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 55 was obtained in a 73-year-old female on routine ECG. The ECG demonstrates a unique clinical presentation of a permanent pacemaker with the ventricular lead in the left ventricle instead of the normal location in the right ventricular apex. The rhythm is sinus at a rate of 83 beats/minute, and there is a pacing spike before each QRS complex but not before the P wave (arrows). This is consistent with a dual chamber pacemaker that is properly sensing the atrium and pacing the ventricle. Normal ventricular lead placement in the right ventricular apex manifests right bundle branch morphology with left axis deviation ECG. Instead, the ventricular lead is in the left ventricle near the apex thus manifests a right bundle branch morphology and right axis deviation. In this patient the ventricular pacing lead was inadvertently passed through a patent foramen ovale into the left atrium, through the mitral valve, and into the left ventricle.

Codes:

07 Sinus rhythm

92 Dual-chamber pacemaker (DDD)



Questions: ECG 55

1. Which QRS morphology is associated with location of the ventricular pacing catheter in the right ventricular apex:

    a. RBBB + RAD
    b. RBBB + LAD
    c. LBBB + RAD
    d. LBBB + LAD

Answers: ECG 55

1. When the ventricles are activated by an impulse originating in the right ventricle, the left ventricle is activated by septal activation, avoiding the normal left bundle branch conduction system and giving rise to left bundle branch morphology on the surface ECG. This occurs with right-sided VPCs, ventricular tachycardia, and pacing from the right ventricle. Any electrical impulse that starts in the apex of the heart activates the ventricles away from the inferior leads and gives rise to left axis deviation ("superior axis"). In contrast, activation of the ventricles from the left or right ventricular outflow tract (i.e., just below the pulmonary or aortic valve) activates the heart toward the inferior leads and give rise to a normal axis or right axis deviation that is markedly positive in the inferior leads ("inferior axis"). (Answer: d)

— Quick Review 55 —

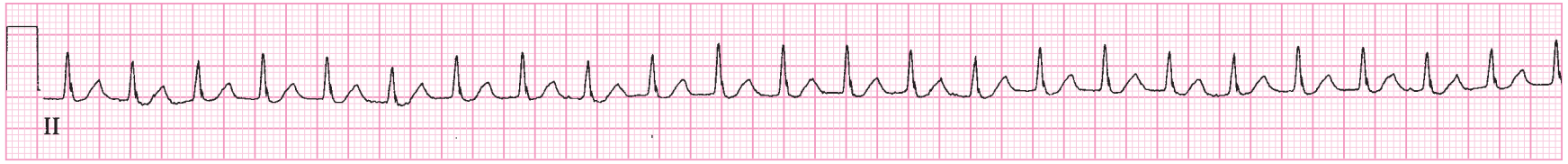
Dual chamber, atrial-sensing pacemaker	
• For atrial sensing, need to demonstrate inhibition of (atrial/ventricular) output and/or triggering of the (atrial/ventricular) stimulus in response to intrinsic atrial depolarization	atrial ventricular
• Includes ____ and possibly VAT or VDD pacemakers	DDD

— POP QUIZ —

Find The Imposter

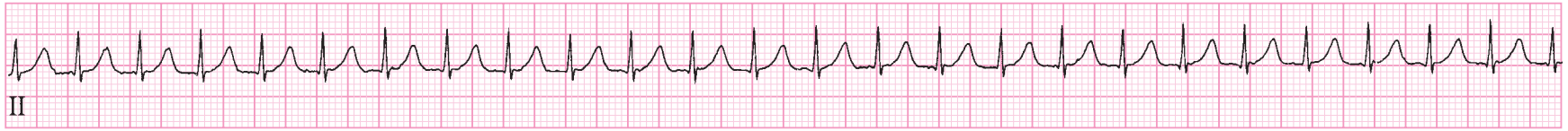
Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.



B.

C.



D.

Answer: Tracings A, B and C represent supraventricular tachycardia, with no clear P waves or atrial activity present. Tracing D shows atrial flutter with 2:1 AV block and is the imposter. Here the deeply negative flutter waves preceding the QRS are also evident in the late QRS complex and ST segment of the preceding beat. In fact, the slightly later appearance of the flutter wave superimposed on the S wave at the right of the tracing (compared to the left portion of the tracing) helps to further confirm the diagnosis of atrial flutter.

— POP QUIZ —

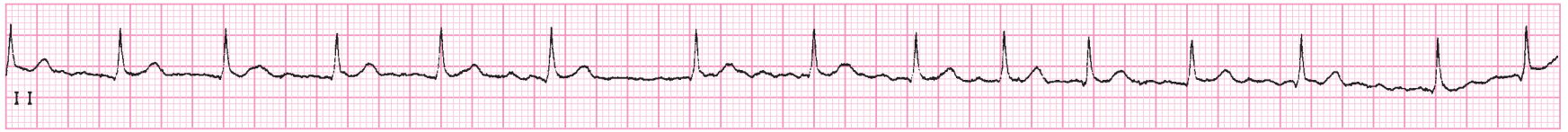
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

B.

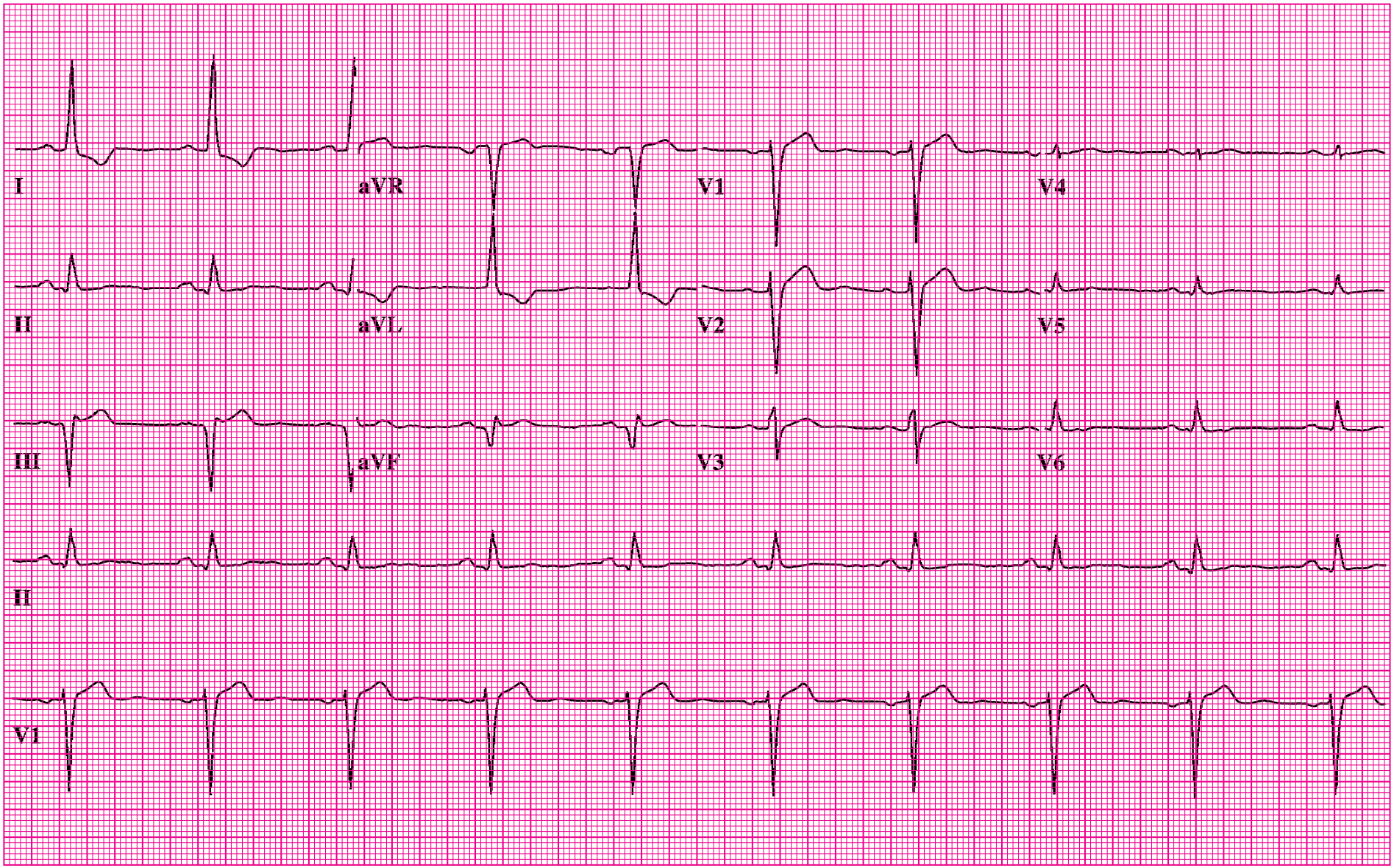
C.



D.

Answer: Tracings A, C, and D demonstrate a narrow QRS complex rhythm with irregular QRS intervals and no discrete atrial (P wave) activity. These findings are consistent with atrial fibrillation. Tracing B shows sinus rhythm with frequent APCs and is the imposter. It shows an irregular rhythm with one dominant P wave preceding most of the QRS complexes. The early QRS complexes also have a preceding P wave and are consistent with atrial premature complexes.

ECG 56. 58-year-old female with heart failure:



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- ☐ 59. Posterior (age recent or acute)
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- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

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- ☐ 71. Digitalis toxicity
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- ☐ 73. Antiarrhythmic drug toxicity
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- ☐ 78. Atrial septal defect, secundum
- ☐ 79. Atrial septal defect, primum
- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
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- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

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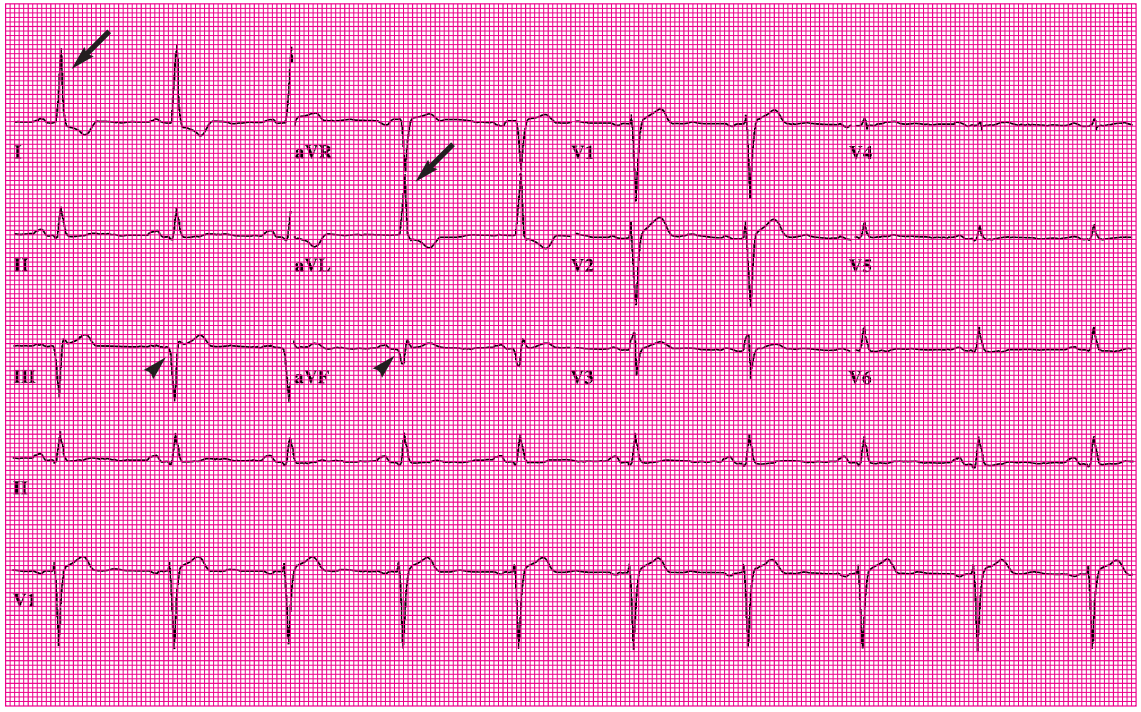
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 56 was obtained in a 58-year-old female with heart failure. The ECG shows sinus bradycardia at 58 beats/minute and LVH ® wave in I ≥ 14 mm; R wave in aVL ≥ 12 mm; arrows) with associated ST-T abnormalities. While some degree of QRS widening is often present with LVH, the QRS in this tracing measures 0.12 seconds, consistent with nonspecific IVCD. Evidence for an old inferior myocardial infarction (arrowheads) is present.

Codes:

09	Sinus bradycardia (<60)
40	Left ventricular hypertrophy
49	Nonspecific intraventricular conduction disturbance
58	Inferior Q wave MI (age indeterminate or old)
67	ST and/or T wave abnormalities secondary to hypertrophy



Questions: ECG 56

1. ECG leads in the present tracing that demonstrate LVH by voltage criteria include:

   a. R wave in I + S wave in II
   b. R wave in V_5 or V_6 + S wave in lead V_1
   c. R wave in I
   d. S wave in aVR
   e. R wave in aVL
   f. R wave in aVF

2. ECG findings associated with LVH include:

   a. ST elevation in leads V_1 - V_3
   b. ST segment depression and T wave inversion in leads I, aVL, V_4 - V_6
   c. Prominent U waves

Answers: ECG 56

1. In the present tracing, voltage criteria for LVH is satisfied by the presence of R waves in leads I and aVL > 14 mm and 12 mm, respectively. Criteria not satisfied on this ECG include an R wave in lead V_5 or V_6 + S wave in lead I > 35 mm; an R wave in lead I + S wave in lead II > 26 mm; an S wave in aVR > 15 mm; and an R wave in aVF > 21 mm. (Answer: c, e)

2. Non-voltage criteria for LVH include ST segment depression and T wave inversion in leads V_5 and V_6, ST elevation in the right precordial leads, and prominent U waves. Other non-voltage-based findings include left atrial abnormality, left axis deviation, nonspecific intraventricular conduction delay, delayed intrinsicoid deflection, poor R wave progression, absent Q waves in the left precordial leads, and abnormal Q waves in the inferior leads (due to left axis deviation). LVH may cause a "pseudoinfarct" pattern on ECG: poor R wave progression with ST elevation in V_1-V_3 can mimic anteroseptal MI, and inferior Q waves can mimic inferior MI. (Answer: all)

— Quick Review 56 —

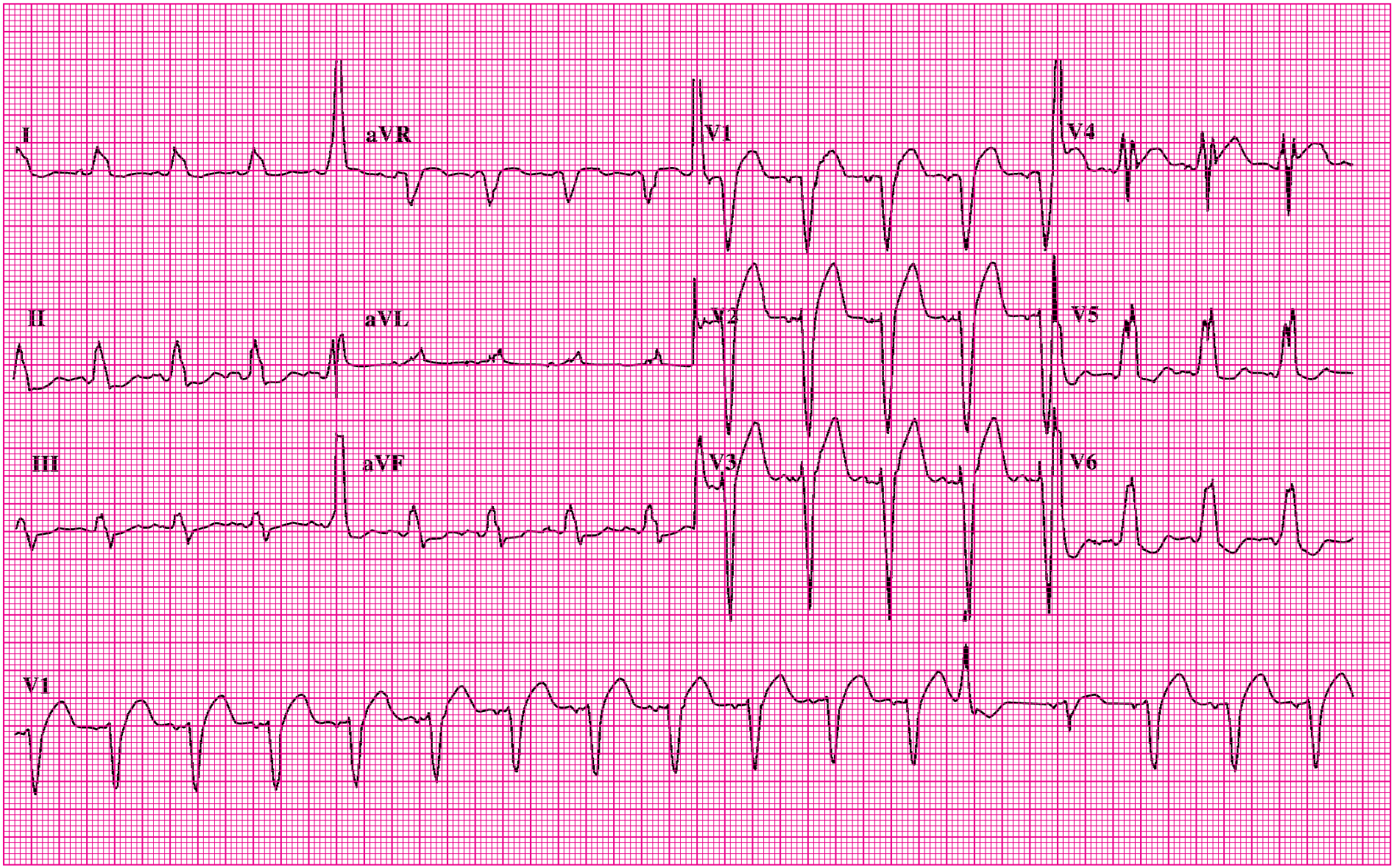
Nonspecific intraventricular conduction disturbance

- QRS ≥ ____ seconds in duration but morphology does not meet criteria for LBBB or RBBB, *or* abnormal ____ without widening of the QRS complex — 0.11 / notching

Left ventricular hypertrophy ST-T changes

- Voltage criteria for LVH and one or more ST-T abnormalities:
  - ST segment and T wave deviation in (same/opposite) direction to the major deflection of QRS — opposite
  - ST segment (elevation/depression) in leads I, aVL, III, aVF, and/or V_4-V_6 — depression
  - Subtle (< 1-2 mm) ST (elevation/depression) in leads V_1-V_3 — elevation
  - Inverted ____ waves in leads I, aVL, V_4-V_6 — T
  - (Absent/prominent) U waves — prominent

ECG 57. 55-year-old woman with a history of dilated cardiomyopathy:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
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- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
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- ☐ 12. Sinoatrial exit block
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- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
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- ☐ 27. Ventricular escape complexes or rhythm
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- ☐ 37. Right axis deviation (> +100°)
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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
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- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
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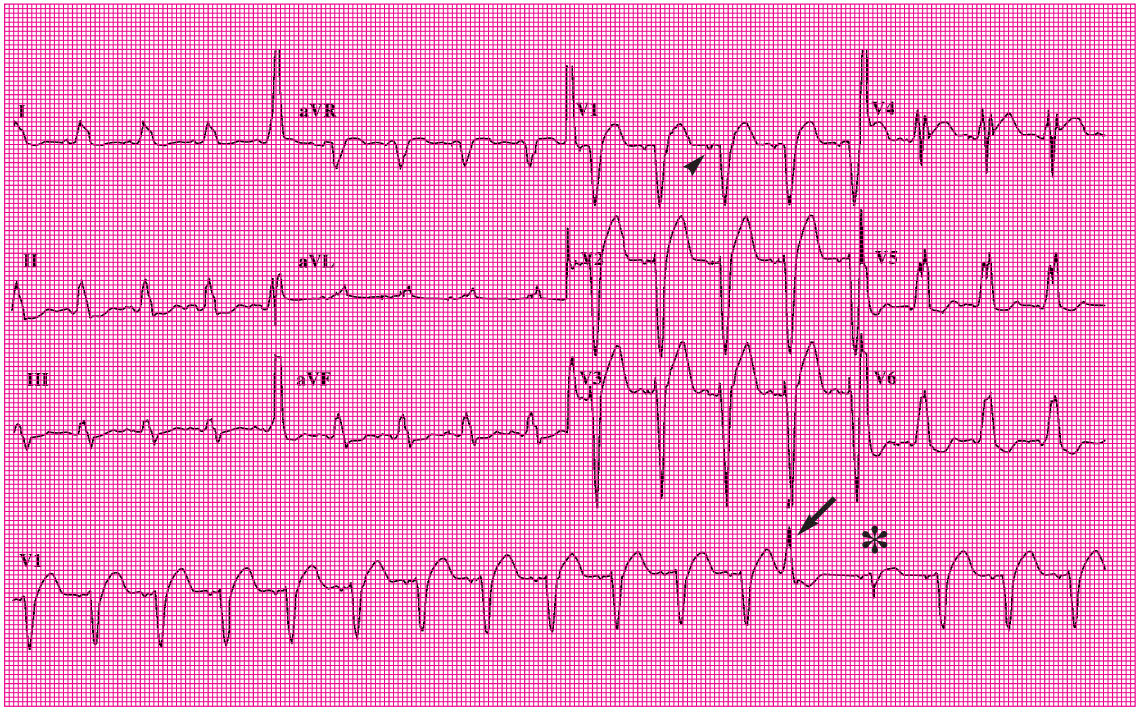
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 57 was obtained from a 55-year-old woman with a history of dilated cardiomyopathy. The ECG shows sinus tachycardia, left atrial abnormality (arrowhead), and LBBB with secondary ST-T abnormalities. The rhythm strip at the bottom of the tracing shows a ventricular premature complex (arrow) followed by a compensatory pause; the first beat after the compensatory pause (asterisk) shows a normal sinus beat without LBBB, establishing the diagnosis of rate-related LBBB.

Codes:

06	Left atrial abnormality/enlargement
10	Sinus tachycardia
23	Ventricular premature complexes
47	LBBB, complete
50	Functional (rate-related) aberrant intraventricular conduction



Questions: ECG 57

1. Intermittent left bundle branch block (LBBB) can be tachycardia- or bradycardia-dependent:

   a. True
   b. False

2. In the setting of LBBB, myocardial injury/infarction is suggested by the presence of ___ mm of discordant ST segment elevation in leads V_1-V_4:

   a. 2
   b. 3
   c. 4
   d. 5

Answers: ECG 57

1. Intermittent LBBB is more common at fast heart rates (tachycardia-dependent) than at slow heart rates, but may be bradycardia-dependent as well. The ventricular premature complex in the present ECG (arrow) results in a compensatory pause; this allows the refractory left bundle branch to recover, resulting in normal QRS conduction for a single beat. Sinus tachycardia resumes on the following beat, resulting in a shorter cycle length and resumption of left bundle branch block. (Answer: a)

2. In the setting of LBBB, discordant ST segment elevation (ST elevation in a direction opposite to the major QRS vector) ≥ 5 mm in height is worrisome for ischemia. Concordant ST segment elevation (ST segment elevation in the same direction as the major QRS vector) ≥ 1 mm is a more specific finding for transmural ischemia. (Answer: d)

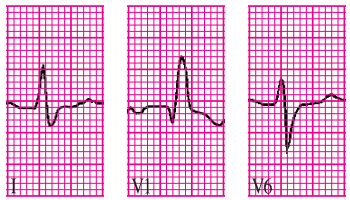
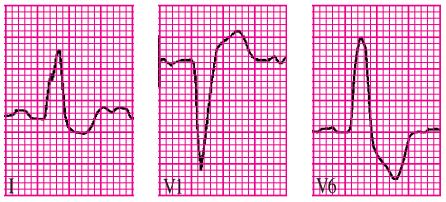
— Quick Review 57 —

Sinus tachycardia (>100)	
• Rate > ____ per minute	100
• P wave amplitude often (increases/decreases) and PR interval often (increases/decreases) with increasing heart rate	increases shortens
Left atrial abnormality/enlargement	
• Notched P wave with a duration ≥ ____ seconds in leads II, III or aVF, *or*	0.12
• Terminal negative portion of the P wave in lead V_1 ≥ 1 mm deep and ≥ ____ seconds in duration	0.04

— POP QUIZ —

Pattern Recognition: Intraventricular Conduction Disturbances

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Right bundle branch block b. QRS axis is usually normal c. Does not interfere with ECG diagnosis of ventricular hypertrophy or Q-wave MI d. Left anterior fascicular block e. Can result in false-positive diagnosis of LVH based on voltage criteria using only leads I or aVL f. Can mask the presence of inferior wall MI g. Left posterior fascicular block h. Can mask the presence of lateral wall MI i. Least prevalent conduction abnormality j. Left bundle branch block k. Commonly associated with secondary ST & T changes in opposite direction to main QRS complex	*Right bundle branch block* (RBBB) results in a prolonged QRS duration ($\geq$ 0.12 seconds) with delayed onset of intrinsicoid deflection (beginning of QRS to peak of R wave > 0.05 seconds); secondary R wave (R') in V_1 and V_2 (rsR' or rSR'), with R' usually taller than the initial R wave and secondary T wave inversion ± downsloping ST segments; and wide, slurred S waves in leads I, V_5 and V_6. Mean QRS axis is determined by the initial unblocked 0.06-0.08 seconds of the QRS, and should be normal unless left anterior fascicular block or left posterior fascicular block is present. RBBB does not interfere with the diagnosis of ventricular hypertrophy or Q-wave MI. (Answer: a, b, c, k in leads V_1 and V_2).
		*Left bundle branch block* (LBBB) results in a prolonged QRS ($\geq$ 0.12 seconds); delayed (> 0.05 seconds) onset of intrinsicoid deflection in leads I, V_5 and V_6; and broad monophasic R waves in leads I, V_5, and V_6 that are usually notched or slurred. Other changes include secondary ST and T wave changes opposite in direction to the major QRS deflection (ST depression and T wave inversion in leads I, V_5, V_6; ST elevation and upright T waves in V_1 and V_2), and rS or QS complexes in the right precordial leads. The axis is usually normal, but left axis deviation may be present. LBBB interferes with identification of QRS axis, ventricular hypertrophy, and acute MI. (Answer: b, j, k)

— POP QUIZ —

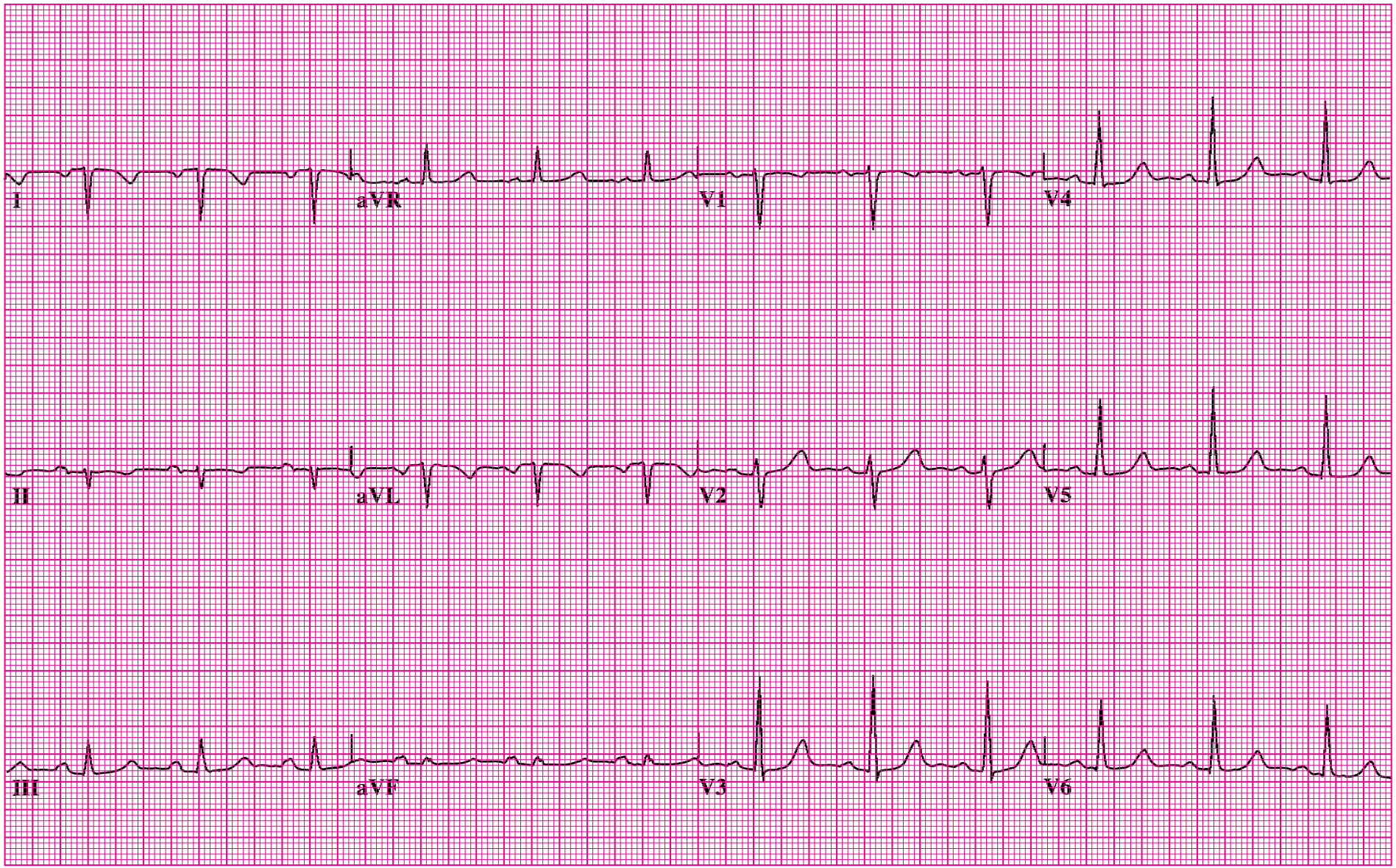
Differential Diagnosis: Precordial R-Wave Progression

Instructions: For each diagnosis below, select all precordial R-wave progression changes that apply:

- Early R-wave progression (tall R wave in V_1V_2; R/S wave amplitude > 1)
- Poor R-wave progression (precordial transition zone [R/S wave amplitude = 1] in V_5 or V_6)
- Reverse-R wave progression (decreasing R-wave amplitude across precordial leads)

Diagnosis	Answer
Hypertrophic cardiomyopathy	Poor R-wave progression. Q waves in I, aVL, V_4-V_6 (from septal hypertrophy) may lead to pseudoinfarct pattern.
Left ventricular hypertrophy (LVH)	Poor R-wave progression. May be accompanied by ST elevation in V_1-V_3 to mimic anteroseptal MI, or Q waves in II, III, aVF to mimic inferior MI.
Left anterior fascicular block (LAFB)	Poor R-wave progression. LAFB can result in pseudoinfarct pattern, mask inferior MI, and cause false-positive diagnosis of LVH based on voltage criteria using only leads I or aVL.
Right ventricular hypertrophy (RVH)	Early R-wave progression. Right axis deviation and deep S waves in V_5V_6 are common. Severe RVH may cancel out QRS forces from the LV and underestimate the presence of LVH.
Anterior MI	Poor R-wave progression; may be only manifestation of prior MI (no anterior Q-waves).
Posterior MI	Early R-wave progression. ST depression and upright T waves in V_1V_2 and inferior MI are common.
Right bundle branch block (RBBB)	Early R-wave progression. R' taller than r-wave in V_1 and T wave inversion in V_1V_2 are usual.
Chronic lung disease (e.g., emphysema)	Poor R-wave progression. Q waves are sometimes seen in right/mid-precordial or inferior leads, resulting in pseudoinfarct pattern.
Wolff-Parkinson-White syndrome (left-sided accessory pathway)	Early R-wave progression when the accessory pathway connects the left atrium and ventricle. Can lead to false-positive or false-negative diagnosis of ventricular hypertrophy, MI, or bundle branch block.
Duchenne's muscular dystrophy	Early R-wave progression.
Dextrocardia	Reverse R-wave progression. Inverted (upside-down) P-QRS-T in leads I and aVL is the key to diagnosis.

ECG 58. 61-year-old asymptomatic female:



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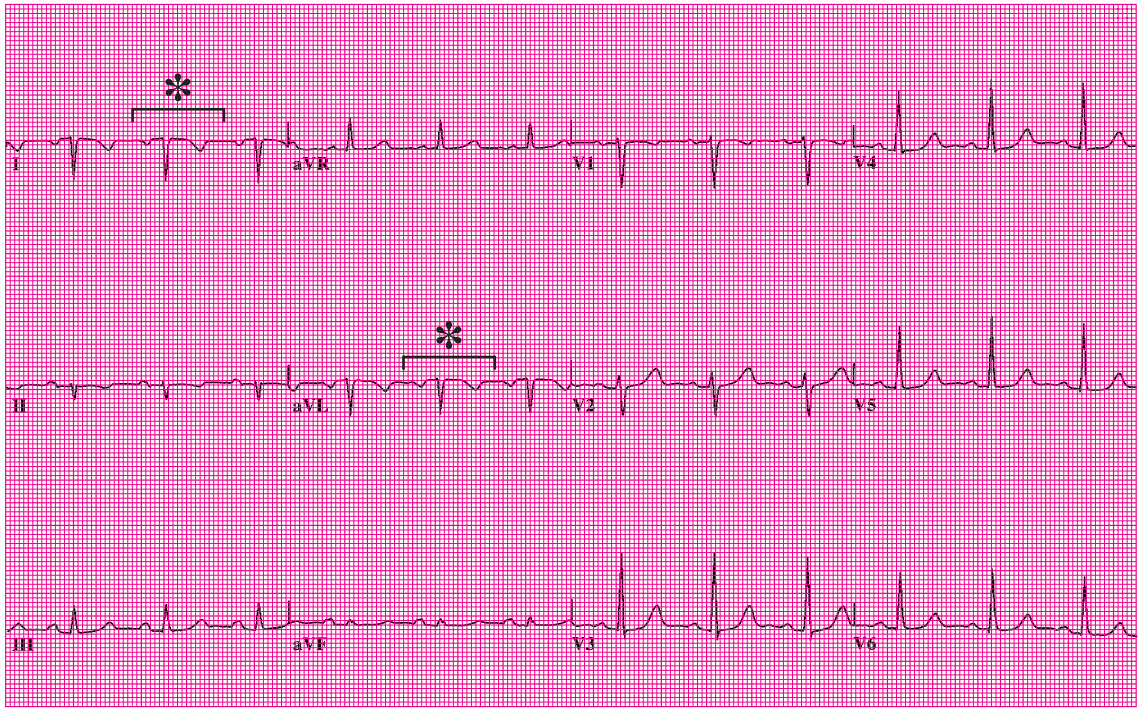
ECG 58 was obtained in a 61-year-old asymptomatic female. The P, QRS, and T waves are inverted in leads I and aVL (asterisks) and upright in aVR. These findings are can be seen both in incorrect limb lead electrode placement and dextrocardia. The normal R wave progression in the precordial leads is consistent with limb lead reversal; dextrocardia manifests reverse R wave progression. The axis is difficult to determine due to incorrect electrode placement, but is probably normal. First-degree AV block is also present.

Codes:

03	Incorrect electrode placement
07	Sinus rhythm
29	AV block, 1°



Questions: ECG 58

1. Limb lead reversal results in a "mirror-image" of the normal P-QRS-T in leads ____ and _____:

   a. I and II
   b. I and aVL
   c. II and III
   d. aVL and aVR

2. In contrast to dextrocardia, limb lead reversal is associated with reverse R wave progression in leads V_1-V_6:

   a. True
   b. False

Answers: ECG 58

1. Limb lead reversal results in inversion of the P-QRS-T in leads I and aVL. This gives the mistaken impression of right axis deviation, and may be confused with mirror-image dextrocardia. (Answer: b)

2. Dextrocardia and limb lead reversal both result in apparent right axis deviation and inversion of the P, QRS, and T waves in leads I and aVL. Dextrocardia is associated with reverse R wave progression in leads V_1-V_6; limb lead reversal shows normal precordial R wave progression. (Answer: b)

— Quick Review 58 —

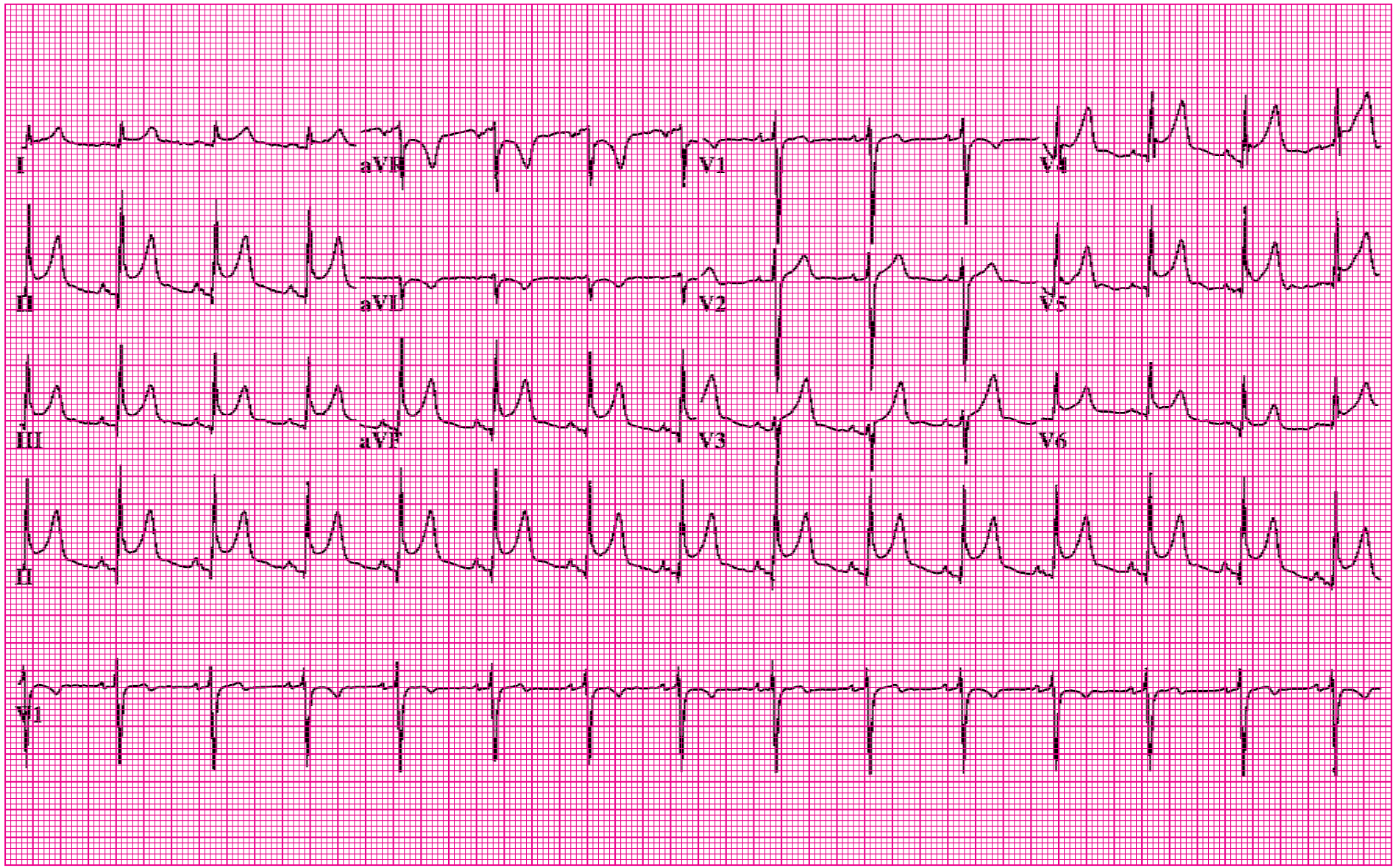
Incorrect electrode placement

Limb lead reversal (reversal of right and left arm leads)

- Resultant ECG mimics dextrocardia with ____ of the P-QRS-T in leads ____ and aVL — inversion, I
- To distinguish between these conditions, look at precordial leads: dextrocardia shows (reverse/normal) R wave progression, while limb lead reversal shows (reverse/normal) R wave progression. — reverse, normal

Precordial lead reversal: Unexplained decrease in ____ voltage in two consecutive leads (e.g., V_1, V_2) with a return to normal progression in the following leads — R wave

ECG 59. 59-year-old male with chest pain and cough of several days duration:



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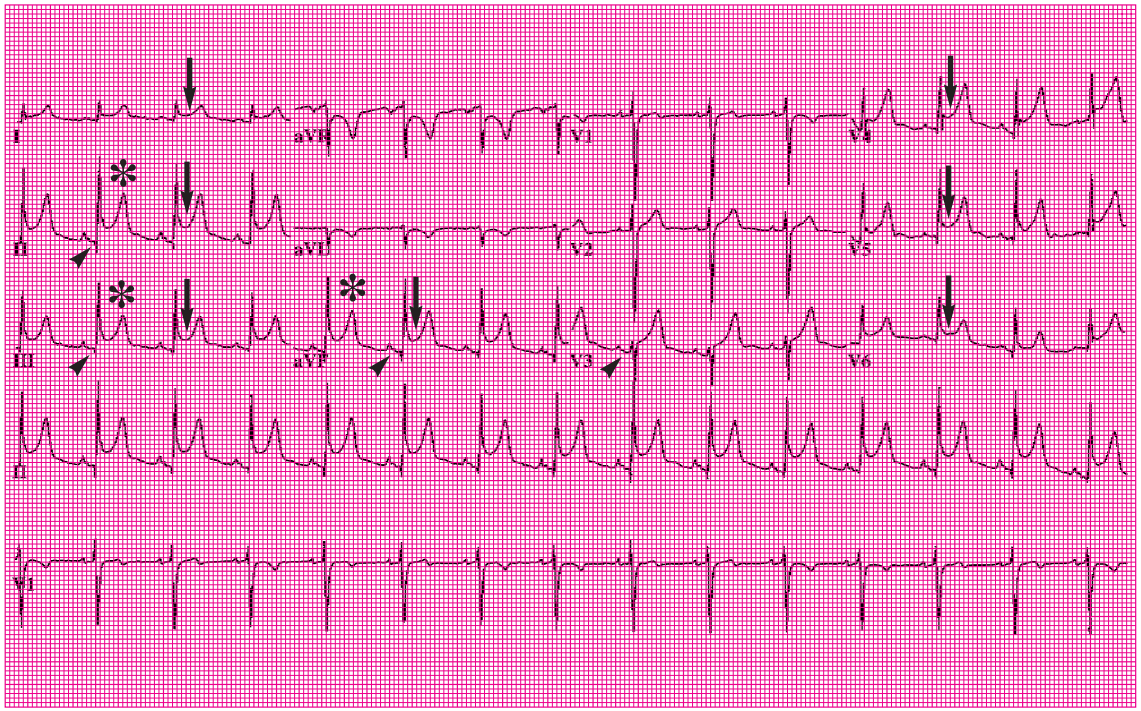
ECG 59 was obtained in a 59-year-old male with chest pain and cough of several days duration. The ECG shows sinus rhythm at 87 beats/minute. Diffuse ST segment elevation that is upwardly concave (arrows) is noted in nearly all leads. Some PR depression is apparent in leads II, III, aVF, and V_3 (arrowheads). Electrical alternans is most obvious in the lead II rhythm strip. Peaked T waves (> 6 mm in height) are also noted in the inferior leads (asterisks). These findings are consistent with acute pericarditis with pericardial effusion. Extensive myocardial infarction (left main or dominant left circumflex occlusion) is also a possibility, but the several day history of chest pain makes this diagnosis unlikely.

Codes:

07	Sinus rhythm
38	Electrical alternans
83	Pericardial effusion
84	Acute pericarditis



Questions: ECG 59

1. ST elevation can be seen in pericarditis in all leads except:

   a. aVF
   b. aVR
   c. III
   d. V_1

2. Diffuse loss of QRS voltage in the setting of pericarditis most likely suggests:

   a. Amyloidosis
   b. Obesity
   c. Pericardial effusion
   d. Associated myocardial infarction

Answers: ECG 59

1. ST segment elevation associated with acute pericarditis is typically diffuse and upwardly concave. All leads can (and often do) show ST elevation except aVR, which typically shows ST depression. (Answer: b)

2. Amyloidosis, obesity, and diffuse myocardial disease related to previous infarction can cause loss of QRS voltage. However, in the setting of pericarditis, the most likely cause is the development of a pericardial effusion. (Answer: c)

— Quick Review 59 —

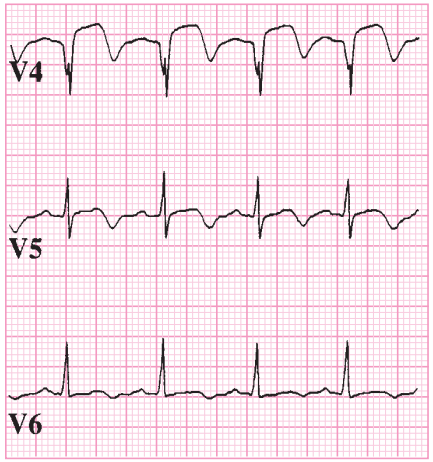
Electrical alternans	
• Alternation in the ____ and/or ____ of the P, QRS and/or T waves	amplitude, direction
ST and/or T wave changes suggesting acute pericarditis	
• Classic evolutionary pattern consists of ____ stages	4
▸ Stage 1: Upwardly concave ST segment ____ in almost all leads	elevation
▸ Stage 2: ST junction (J point) returns to baseline and T wave amplitude begins to (increase/decrease)	decrease
▸ Stage 3: T waves (invert/remain upright)	invert
▸ Stage 4: ECG (does/does not) return to normal	does
• Other clues to acute pericarditis:	
▸ Sinus ____	tachycardia
▸ PR ____ early (PR elevation in aVR)	depression
▸ (High/low) voltage QRS	low
▸ Electrical alternans if pericardial ____ is present	effusion
Peaked T waves	
• T wave > ____ mm in the limb leads or > ____ mm in the precordial leads	6, 10

— POP QUIZ —

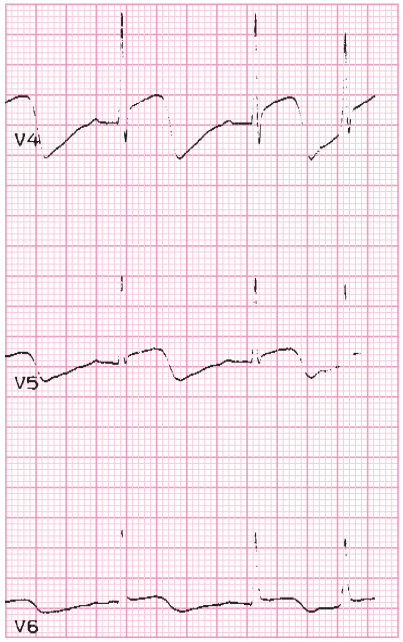
Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

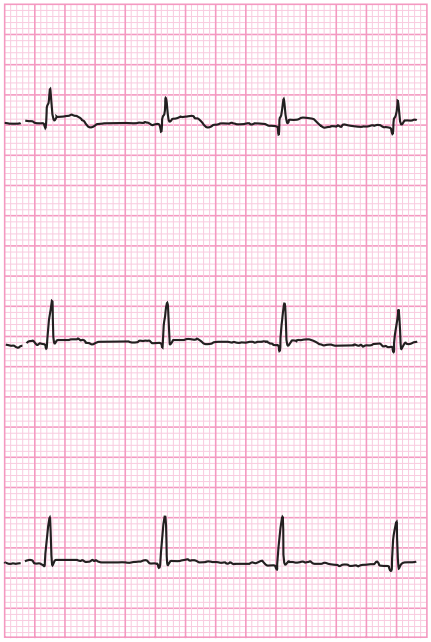
A.



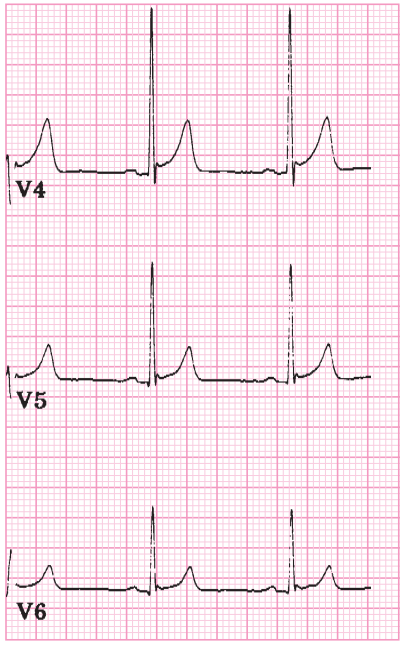
B.



C.



D.



Answer: Tracings A, B, and C demonstrate ST segment elevation with ST segments that are convex upwards with inverted T waves. These findings are consistent with myocardial injury. Tracing D shows concave upward ST segment elevation consistent with early repolarization and is the imposter.

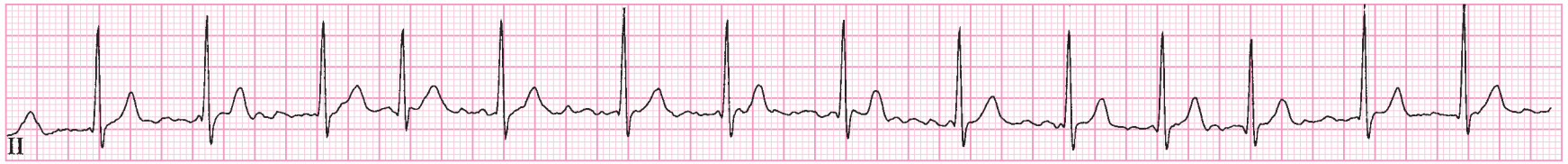
— POP QUIZ —

Find The Imposter

Instructions: Three of the following ECG tracings have a common diagnosis. Identify the common diagnosis and find the imposter.

A.

B.

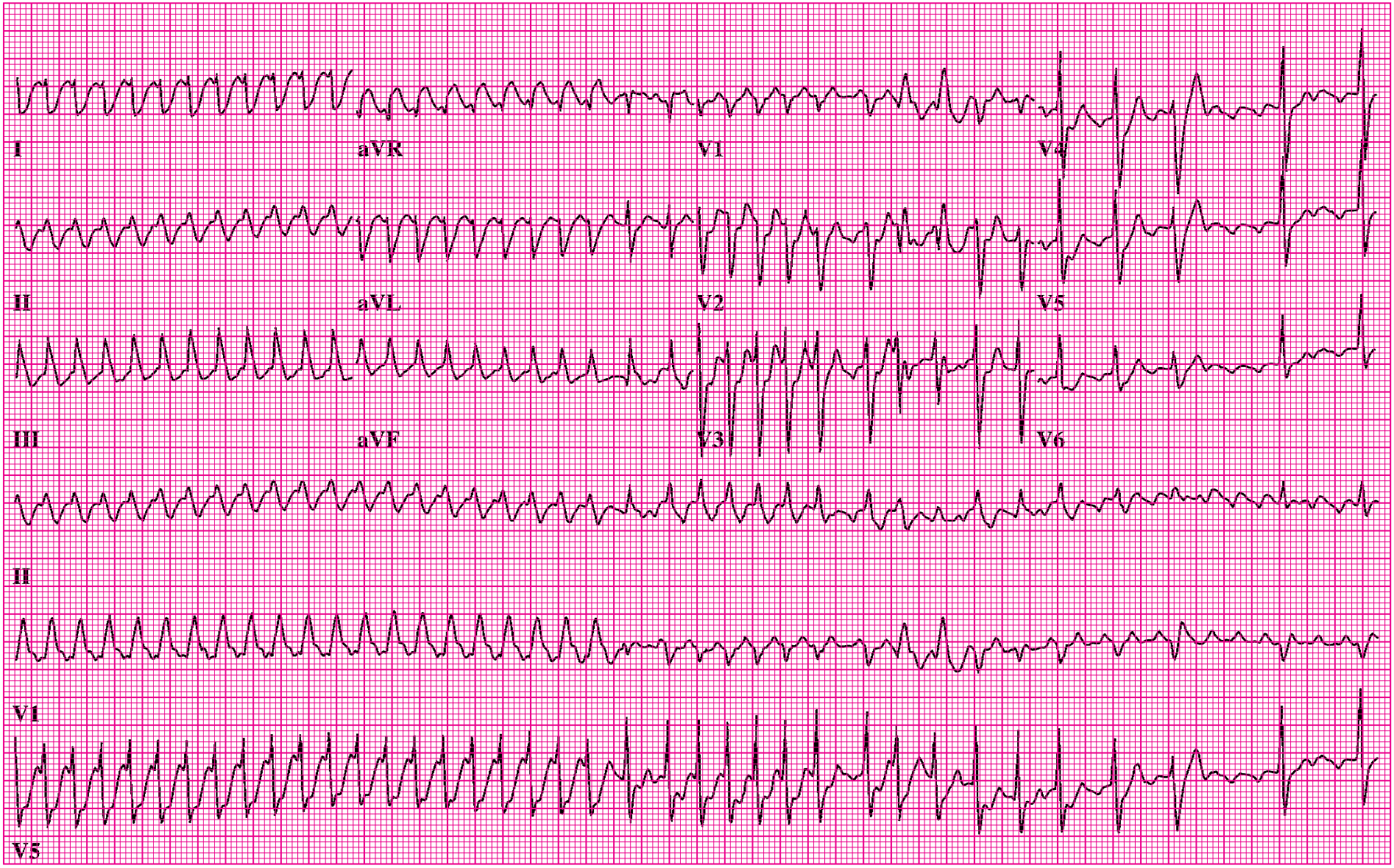


C.

D.

Answer: Tracings B, C, and D show atrial fibrillation with irregular QRS intervals and no discreet P waves. Tracing A shows atrial flutter with variable AV conduction and is the imposter. The tracing has regular atrial activity at a rate of approximately 300 beats per minute. This atrial activity is negative with a "saw tooth" pattern and consistent with atrial flutter. The atrial activity has a variable relationship with the preceding QRS complex and thus represents atrial activity and not a T wave.

ECG 60. 53-year-old male with chest fluttering and dyspnea:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

- ☐ 70. Digitalis effect
- ☐ 71. Digitalis toxicity
- ☐ 72. Antiarrhythmic drug effect
- ☐ 73. Antiarrhythmic drug toxicity
- ☐ 74. Hyperkalemia
- ☐ 75. Hypokalemia
- ☐ 76. Hypercalcemia
- ☐ 77. Hypocalcemia
- ☐ 78. Atrial septal defect, secundum
- ☐ 79. Atrial septal defect, primum
- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
- ☐ 82. Acute cor pulmonale including pulmonary embolus
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- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
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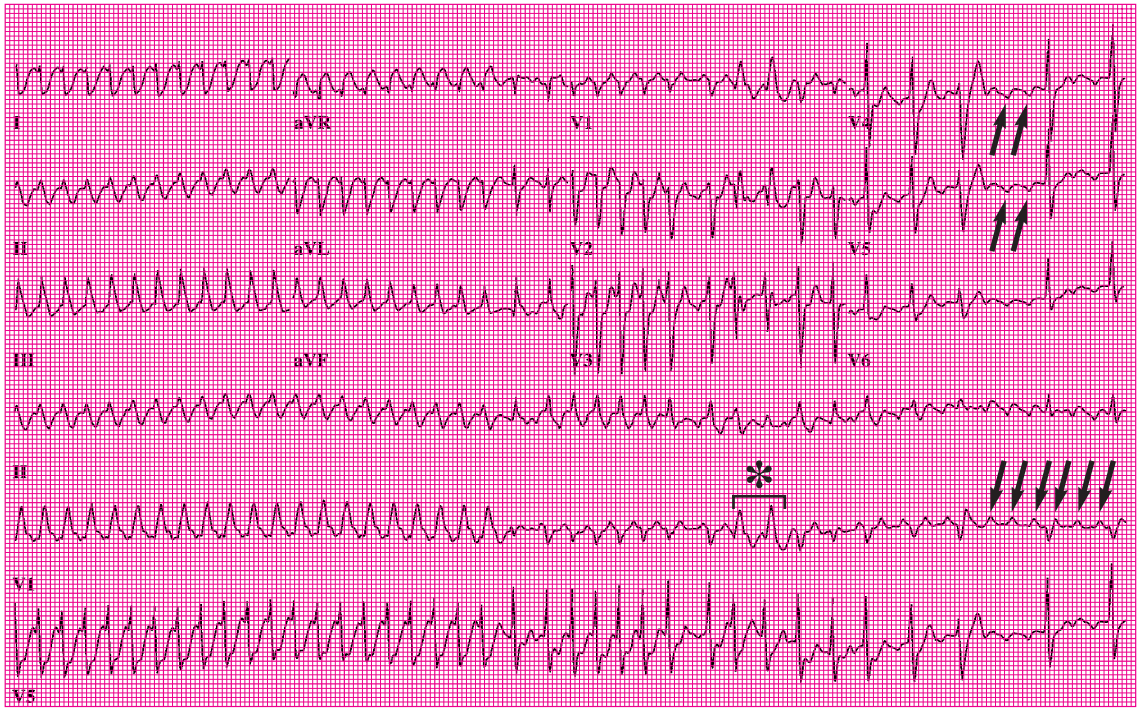
PACED RHYTHMS

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

ECG 60 was obtained in a 53-year-old male complaining of chest fluttering and dyspnea. The ECG shows atrial flutter, which is most apparent in the latter portion of the lead II rhythm strip and in leads V_4-V_6 (arrows mark flutter waves). Variable second-degree AV block is noted, at times resulting in a rapid (1:1) ventricular response with aberrant conduction (asterisk). During the tachycardia, the patient shows evidence for RBBB and left posterior fascicular block, which are transient findings related to the tachycardia and not essential for coding (although including these diagnoses would probably be given neutral credit).

Codes:

18	Atrial flutter
50	Functional (rate-related) aberrant intraventricular conduction



Questions: ECG 60

1. True statements about atrial flutter include:

   a. Ventricular response rates may vary
   b. The interval between flutter waves may vary
   c. Flutter rate is usually 240-340 bpm
   d. Carotid sinus massage frequently restores normal sinus rhythm

2. The most common AV conduction rate in atrial flutter is:

   a. 1:1
   b. 2:1
   c. 3:1
   d. 4:1
   e. > 4:1

3. QRS complexes in tachycardia-induced aberrancy are more likely to manifest:

   a. Left bundle branch block (LBBB) morphology
   b. Right bundle branch block (RBBB) morphology

Answers: ECG 60

1. Atrial flutter manifests as rapid regular atrial undulations (flutter or "F" waves) at a rate of 240-340 per minute. (In contrast, atrial fibrillation manifests totally irregular atrial fibrillatory (f) waves of varying amplitude, duration and morphology.) AV conduction ratio (ratio of flutter waves to QRS complexes) is usually fixed, but may vary, resulting in an irregular ventricular response, which is often due to two levels of block (e.g., 2:1 and 4:1 AV block) or concealed conduction. Atrial flutter typically responds to carotid sinus massage with a decrease in ventricular rate, which returns to baseline upon termination of this maneuver; restoration of normal sinus rhythm with carotid sinus massage is rare. (Answer: a, c)

2. Atrial flutter most commonly presents as 2:1 AV block. Conduction ratios of 1:1 (which may be mistaken for ventricular tachycardia) and 3:1 are uncommon. In untreated patients, AV block $\geq$ 4:1 suggests coexistent AV conduction system disease. (Answer: b)

3. Aberrant intraventricular conduction occurs when a supraventricular impulse finds one of the bundle branches conductive and the other refractory. Since the right bundle typically has a longer action potential and refractory period than the left bundle, QRS complexes in aberrancy usually manifest RBBB morphology. (Answer: b)

— Quick Review 60 —

Atrial flutter

- Rapid (regular/irregular) atrial undulations ("F" waves) at a rate of ____ per minute — regular; 240-340
- Flutter rate may (increase/decrease) in the presence of Types IA, IC or III antiarrhythmic drugs — decrease
- Flutter waves in leads II, III, AVF are typically (inverted/upright) (with/without) an isoelectric baseline — inverted,without
- Flutter waves in lead V_1 are typically small (positive/negative) deflections (with/without) a distinct isoelectric baseline — positive, with
- QRS complex may be normal or aberrant (true/false) — true
- AV conduction ratio (ratio of flutter waves to QRS complexes) is usually (fixed/variable) — fixed
  - Conduction ratios of 1:1 and 3:1 are (common/uncommon) — uncommon
  - In untreated patients, AV block ≥ ____ suggests the coexistence of AV conduction disease — 4:1

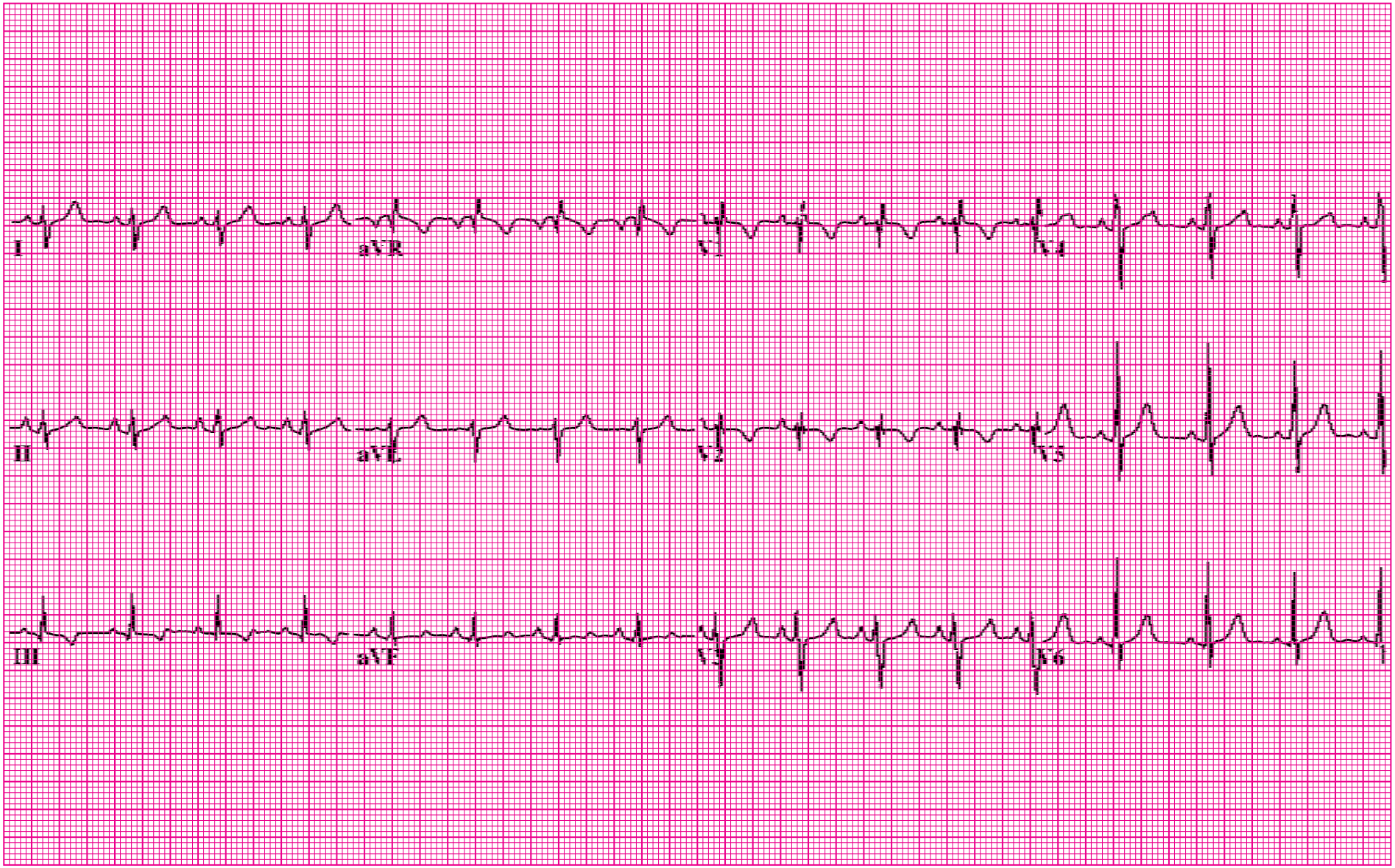
— POP QUIZ —

VT or Not VT: That is the Question

Instructions: In the setting of a wide QRS tachycardia, decide whether the ECG features below favor ventricular tachycardia or SVT with aberrancy.

ECG Feature	VT or SVT with Aberrancy
QRS duration during tachycardia < 0.14 seconds if RBBB morphology or < 0.16 seconds if LBBB morphology (assuming QRS is narrow during sinus rhythm)	SVT
Some QRS deflections in precordial leads are positive and some are negative (discordance)	SVT
RSR' V_1: R wave is taller than R'	VT
QRS morphology similar to sinus rhythm or aberrantly conducted APCs	SVT
Tachycardia initiated by VPCs	VT
AV dissociation present	VT
Capture beats present	VT
Fusion beats present	VT

ECG 61. 6-year-old female with a heart murmur:



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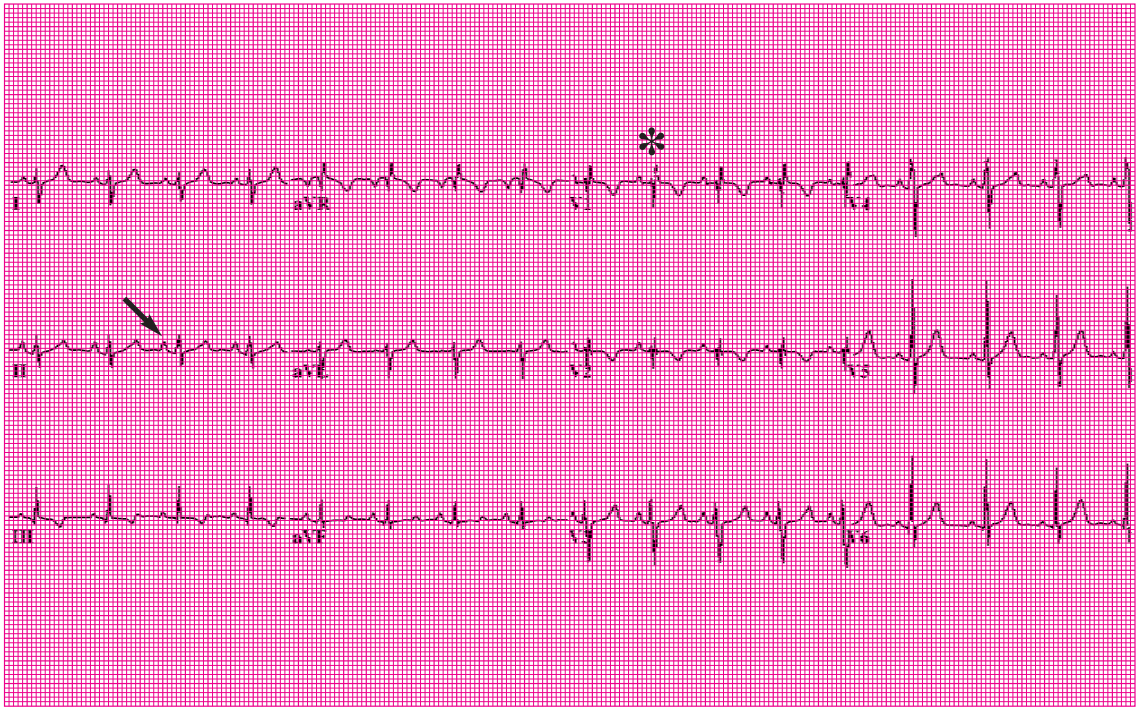
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 61 was obtained in a 6-year-old female with a heart murmur. The ECG shows sinus rhythm at 98 beats/minute. Incomplete RBBB (asterisk marks the rSR' complex in V_1, which is 0.10 seconds in duration) with secondary ST-T changes, right atrial abnormality (arrow), and right axis deviation are also present. These findings are consistent with ostium secundum atrial septal defect, which was confirmed by echocardiography.

Codes:

05	Right atrial abnormality/enlargement
07	Sinus rhythm
37	Right axis deviation (> +100°)
44	RBBB, incomplete
78	Atrial septal defect, secundum



Questions: ECG 61

1. Secundum atrial septal defect results in ____ axis deviation:

   a. Left
   b. Right

2. Causes of right axis deviation include:

   a. Right bundle branch block
   b. Right ventricular hypertrophy
   c. Lateral myocardial infarction
   d. Ostium secundum ASD
   e. Dextrocardia
   f. Chronic lung disease (e.g., emphysema)

Answers: ECG 61

1. Secundum atrial septal defect is typically associated with right axis deviation, incomplete RBBB, and right atrial enlargement. (Answer: b)

2. Right axis deviation can be seen as a normal variant, but is more often associated with COPD, cor pulmonale, right ventricular hypertrophy (RVH), lateral MI, left posterior fascicular block (LPFB), dextrocardia, lead reversal, ostium secundum ASD (ostium primum ASD is associated with left axis deviation), and Wolff-Parkinson-White syndrome. Right bundle branch block does not cause right axis deviation unless complicated by LPFB. Right axis deviation (QRS axis 90° to 180°) must be distinguished from right *superior* axis (-90° to -180°), which can be caused by RVH with or without left anterior fascicular block, left anterior fascicular block with lateral MI, or COPD. (Answer: all except a)

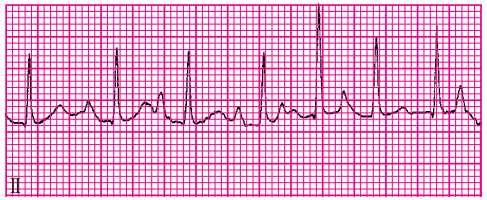
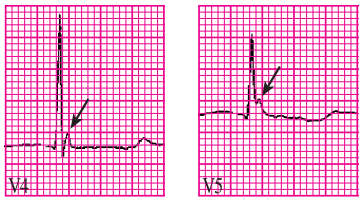
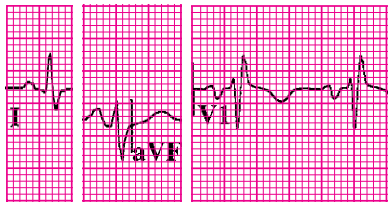
— Quick Review 61 —

Atrial septal defect, secundum	
• Incomplete (RBBB/LBBB)	RBBB
• (Right/left) axis deviation ± (right/left) ventricular hypertrophy	Right, right
• (Right/left) atrial abnormality in ~ 30%	Right
• ____ degree AV block in < 20%	First
• Secundum ASDs represent 70-80% of all ASDs, and are due to deficient tissue in the region of the____	fossa ovalis

— POP QUIZ —

Pattern Recognition: ECG/Clinical Correlation

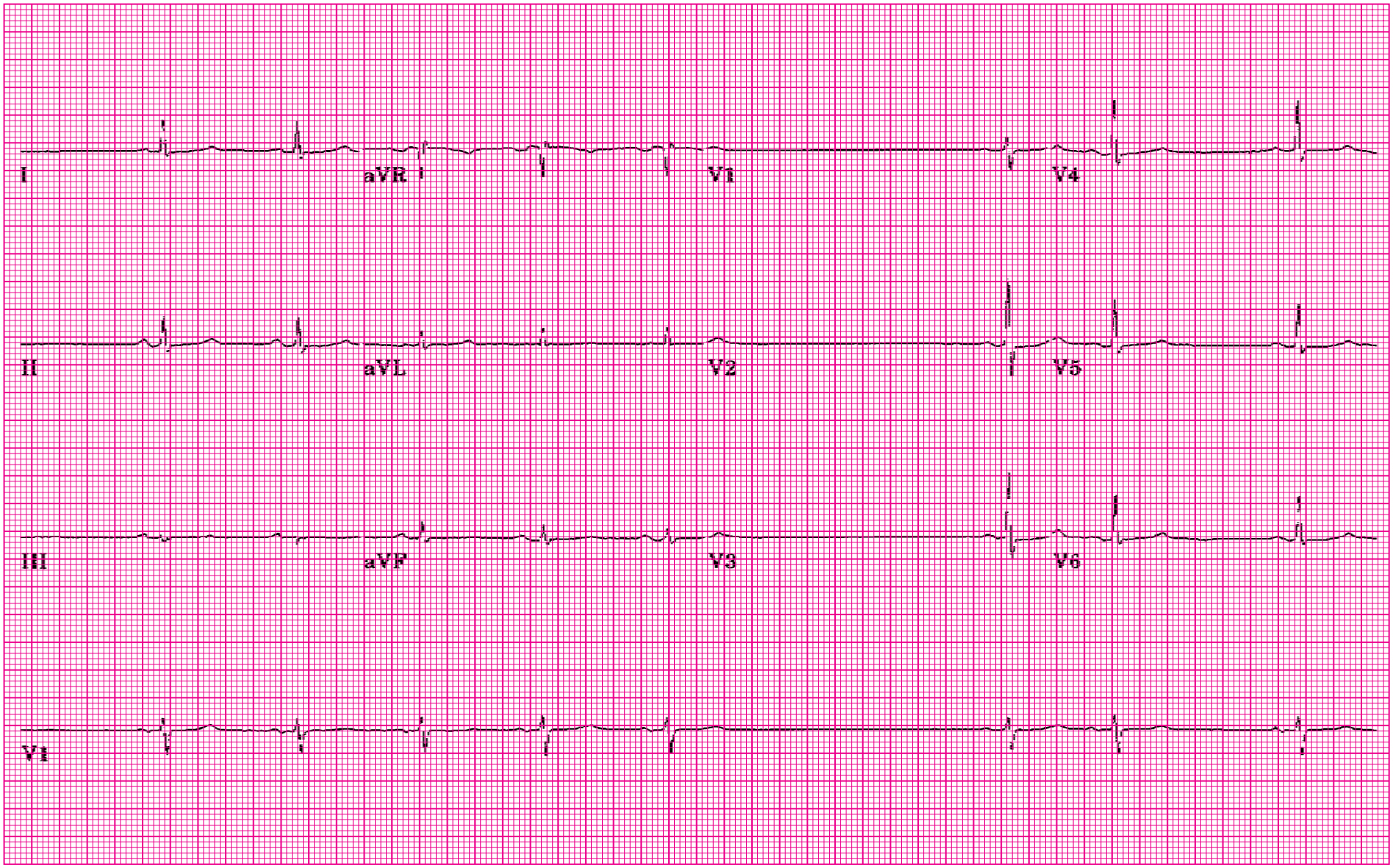
Instructions: Match the ECG with the most likely clinical presentation.

ECG	Choose Single Best Answer	Answer
	a. Acute hemiparesis, papilledema b. Dyspnea, constipation, impaired memory, fatigue c. Red-green color blindness, nausea, vomiting d. Acute oliguria 2° to rhabdomyolysis e. Murmur in a Down's Syndrome patient f. Acute exacerbation of chronic bronchitis g. Prolonged exposure to extreme cold h. Dyspnea and pulses paradoxus in a renal failure patient i. Acute onset of dyspnea in a patient with a DVT	*Multifocal atrial tachycardia* (MAT) results in an irregular rhythm at a rate > 100 per minute with at least three different P wave morphologies and varying PP and PR intervals. MAT is usually associated with some form of lung disease (COPD, cor pulmonade, hypoxia, heart failure), and can be precipitated by aminophylline. This ECG was obtained in a 67-year-old smoker patient with palpitations following acute exacerbation of chronic bronchitis. (Answer: f)
		*Hypothermia* is associated with Osborne ("J") waves, which are late, upright, terminal deflections of the QRS complex that become more pronounced as temperature declines. (Notching simulating Osborne waves may be seen in early repolarization.) Other ECG findings in hypothermia include sinus bradycardia, prolongation of PR, QRS, and QT intervals, and atrial fibrillation (in 50-60%). AV junctional rhythm, VT, and VF may occur. This patient was found unconscious following prolonged exposure to extreme cold. (Answer: g)
		*Ostium primum atrial septal defect (ASD)* represents 15-20% of ASDs, and is due to deficient tissue in the lower portion of the atrial septum. Primum ASDs are usually large, may be accompanied by anomalous pulmonary venous drainage, and are associated with cleft anterior mitral valve leaflet, mitral regurgitation, and Down's syndrome. ECG findings include RSR' in V_1, incomplete RBBB, and left axis deviation (in contrast to right axis deviation with ostium secundum ASD). First-degree AV block occurs in 15-40%, and biventricular hypertrophy is common in advanced cases. (Answer: e)

Don't Forget!

- An S_1 S_2 S_3 pattern (S wave in leads I, II, and III) is present in up to 20% of healthy adults
- Parkinson's tremor (~ 300 per minute) may be mistaken for atrial flutter
- If sinus bradycardia is present at a rate < 40 per minute, think of 2:1 sinoatrial exit block
- P wave amplitude often increases and PR interval often shortens with increasing heart rate (e.g., during exercise)
- The post-extrasystolic pause of normally conducted APCs is usually *noncompensatory* (i.e., PP interval containing the APC is less than two times the normal PP interval)
- In nonconducted (blocked) APCs, P waves are often hidden in the preceding T wave — search for a deformed T wave immediately preceding a PP pause to identify the presence of a nonconducted atrial premature beat

ECG 62. 80-year-old male with episodes of lightheadedness:



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- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

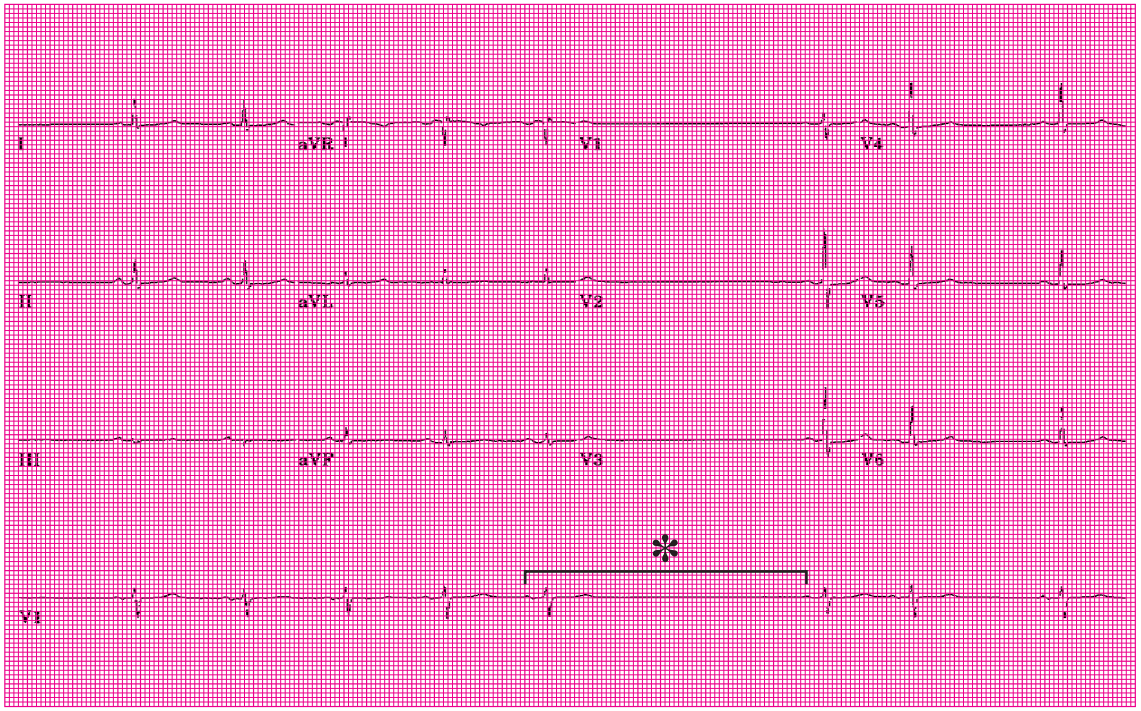
PACED RHYTHMS

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

ECG 62 was obtained from an 80-year-old male with episodes of lightheadedness. The ECG shows sinus rhythm at a rate of 60 beats/minute. Sinus arrhythmia (variability in PP intervals exceeds 0.16 seconds, especially after the pause) and a sinus pause lasting 2.4 seconds (asterisk) are present. Sinoatrial exit block should not be coded since the PP pause is not a multiple (2x, 3x, etc.) of the normal PP interval. The sinus pause and symptoms of lightheadedness are consistent with sick sinus syndrome. Early R wave progression (transition point between V_1 and V_2) is also noted.

Codes:

07	Sinus rhythm
08	Sinus arrhythmia
11	Sinus pause or arrest
89	Sick sinus syndrome



Questions: ECG 62

1. The most common cause of a sinus (PP) pause is:

   a. Sinoatrial exit block
   b. Ventricular premature complex (VPC)
   c. Blocked atrial premature complex (APC)
   d. High-grade AV block

2. The longest and shortest PP intervals in sinus arrhythmia vary by more than:

   a. 0.08 seconds
   b. 0.16 seconds
   c. 10%
   d. 5%

Answers: ECG 62

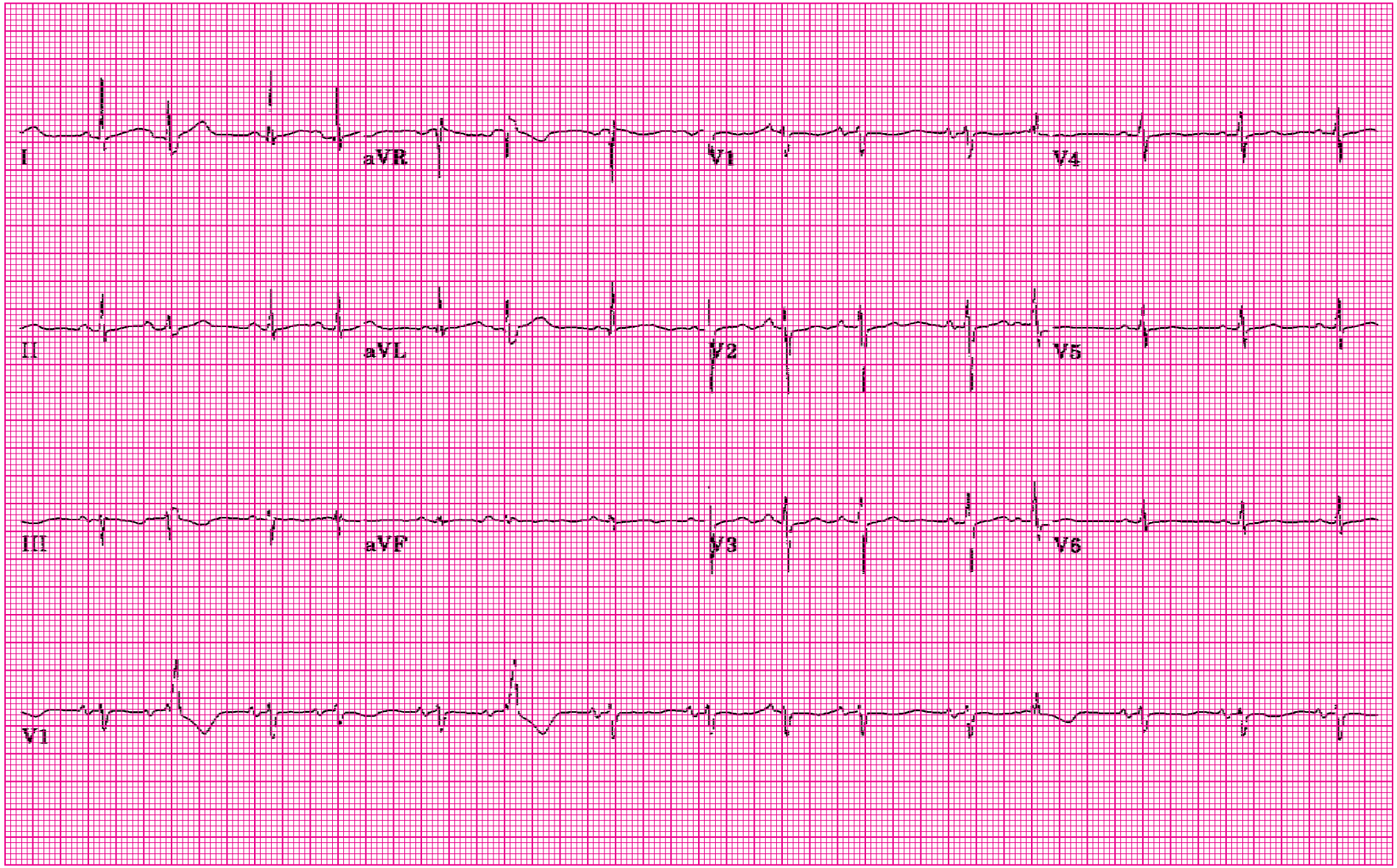
1. A blocked APC is the most common cause of a sinus pause. Close scrutiny of the T wave at the beginning of the pause frequently reveals some deformity caused by the premature atrial beat. (Answer: c)

2. Sinus arrhythmia results in gradual (sometimes abrupt) phasic change in the PP interval, with the longest and shortest PP intervals varying by > 0.16 seconds or 10%. (Answer: b, c)

— Quick Review 62 —

Sinus pause or arrest	
• PP interval > _____ seconds	1.6-2.0
• Resumption of sinus rhythm at a PP interval that (is/is not) a multiple of the basic sinus PP interval	is not
• If sinus rhythm resumes at a multiple of the basic PP, consider _____ block	sinoatrial exit
Sinus arrhythmia	
• (Sinus/nonsinus) P wave	Sinus
• Longest and shortest PP intervals vary by > ____ seconds or ____%	0.16 10
• Sinus arrhythmia differs from "ventriculophasic" sinus arrhythmia, the latter of which occurs in the setting of ____	heart block
Sick sinus syndrome	
• Marked sinus ____	bradycardia
• _____ arrest or _____ exit block	Sinus, sinoatrial
• Bradycardia alternating with ____	tachycardia
• Atrial fibrillation with ____ ventricular response preceded or followed by sinus bradycardia, sinus arrest, or sinoatrial exit block	slow
• Prolonged sinus node ____ time after atrial premature complex or atrial tachyarrhythmias	recovery
• AV junctional ____ rhythm	escape
• Additional conduction system disease is often present, including AV block, IVCD, or bundle branch block (true/false)	true

ECG 63. 47-year-old female with palpitations:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
- ☐ 11. Sinus pause or arrest
- ☐ 12. Sinoatrial exit block
- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 39. Low voltage
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- ☐ 58. Inferior (age indeterminate or old)
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- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

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- ☐ 71. Digitalis toxicity
- ☐ 72. Antiarrhythmic drug effect
- ☐ 73. Antiarrhythmic drug toxicity
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- ☐ 86. Central nervous system disorder
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- ☐ 88. Hypothermia
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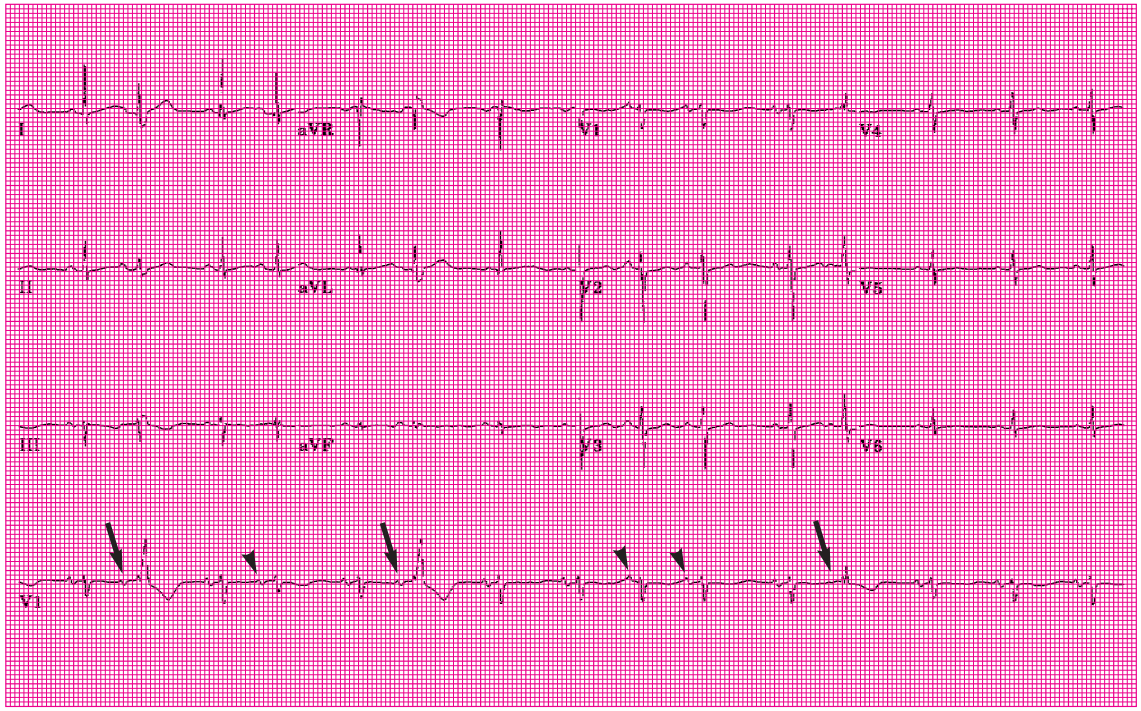
PACED RHYTHMS

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

ECG 63 was obtained from a 47-year-old female with palpitations. The ECG shows sinus rhythm at a rate of 84 beats/minute with frequent atrial premature complexes, some of which conducted normally (narrow QRS) (arrowheads), others of which conduct aberrantly (wide QRS) (arrows). The intermittent RBBB pattern is a result of aberrant intraventricular conduction, not true bundle branch block. Nonspecific ST-T changes are evident, and left atrial abnormality is present in the sinus beats in lead V_1.

Codes:

06	Left atrial abnormality/enlargement
07	Sinus rhythm
13	Atrial premature complexes
50	Functional (rate-related) aberrant intraventricular conduction
63	Nonspecific ST and/or T wave abnormalities



Questions: ECG 63

1. In the setting of a wide complex premature beat, factors that favor an atrial origin over a ventricular origin include:

   a. Compensatory pause
   b. Presence of other normally conducted APCs
   c. Presence of other aberrantly conducted APCs
   d. Initial QRS forces in the same direction as a normally conducted beat

2. The QRS configuration typically seen with aberrantly conducted APCs is:

   a. LBBB pattern
   b. RBBB pattern

Answers: ECG 63

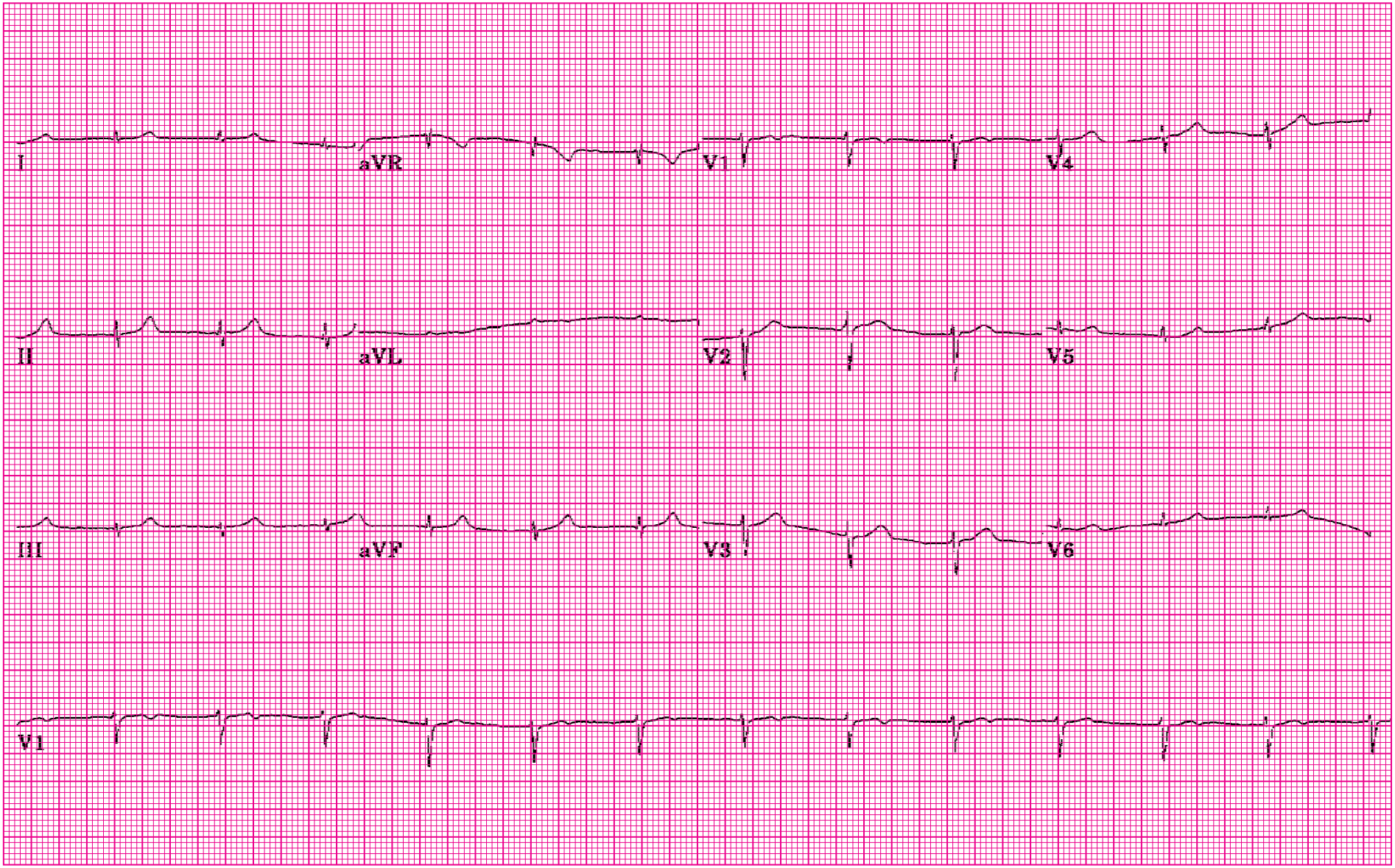
1. Factors favoring an aberrantly conducted atrial premature complex (APC) over a ventricular premature complex (VPC) include the presence of other normally conducted or aberrant APCs, and initial QRS forces in the same direction as a normal sinus beat. APCs typically reset the sinus node, resulting in a non-compensatory pause (i.e, PP interval containing the APC is less than twice the normal PP interval). In contrast, VPCs are usually accompanied by a full compensatory pause (i.e., PP interval containing the VPC is twice the normal PP interval). (Answer: b, c, d)

2. Aberrantly conducted APCs are characterized by P waves that are abnormal in configuration and occur early relative to the normal PP interval, and variable widening/distortion of the QRS complex. The longer refractory period of the right bundle compared to the left bundle increases the likelihood that an APC will conduct down the left bundle, resulting in QRS morphology with RBBB configuration. (Answer: b)

— Quick Review 63 —

Atrial premature complexes, aberrantly conducted	
• P wave is (normal/abnormal) in configuration	abnormal
• QRS complex is (similar/different) in morphology to the QRS complex present during sinus rhythm, and usually manifests (RBBB/LBBB) pattern	different RBBB
• PR interval may be normal, increased, or decreased (true/false)	true
• The post-extrasystolic pause is usually (compensatory/noncompensatory)	noncompensatory
Left atrial abnormality	
• Notched P wave with a duration ≥ ____ seconds in leads II, III or aVF, *or*	0.12
• Terminal negative portion of the P wave in lead V_1 ≥ 1 mm deep and ≥ ____ seconds in duration	0.04

ECG 64. 72-year-old male with chronic heart failure:



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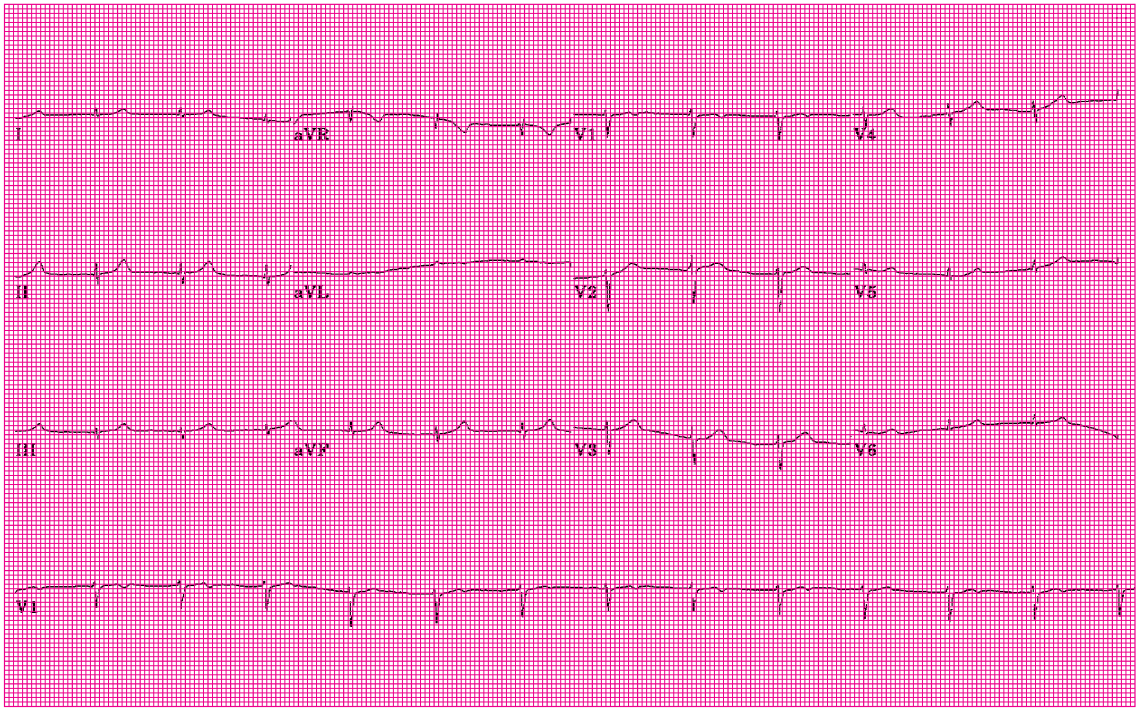
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 64 was obtained from a 72-year-old male with chronic heart failure. The ECG shows a narrow QRS complex rhythm at a rate of 78 beats/minute without P waves, consistent with accelerated junctional rhythm. In the setting of chronic heart failure, this is often caused by digitalis toxicity. Low voltage is evident in the limb leads (QRS amplitude < 5 mm), which may be due to pleural effusion, pericardial effusion, or restrictive, infiltrative, or severe ischemic cardiomyopathy.

Codes:

22 AV junctional rhythm/tachycardia

71 Digitalis toxicity



Questions: ECG 64

1. The P wave in a junctional rhythm:

    a. Follows the QRS complex
    b. Is buried in the QRS complex
    c. Precedes the QRS complex

2. Accelerated junctional rhythm with underlying complete heart block is a common manifestation of:

    a. Sick sinus rhythm
    b. Acute respiratory decompensation
    c. Acute myocardial infarction
    d. Digitalis toxicity

3. During accelerated junctional rhythm:

    a. P waves (when evident) are usually inverted in leads II, III, and aVF
    b. The QRS complex is aberrantly conducted
    c. Rate exceeds 100 per minute
    d. PR interval is prolonged

Answers: ECG 64

1. Depending on the site of origin of the junctional rhythm within the AV node, the P wave can precede, be buried in, or follow the QRS complex. (Answer: all)

2. Digitalis toxicity can cause a wide variety of arrhythmias and conduction disturbances, including paroxysmal atrial tachycardia with block, second- or third-degree AV block, accelerated junctional or idioventricular rhythm with complete heart block, and supraventricular tachycardia with alternating bundle branch block. Regularization of the ventricular response in atrial fibrillation is often indicates the development of complete heart block. Digitalis toxicity may be exacerbated by hypokalemia, hypomagnesemia, or hypercalcemia. (Answer: d)

3. Because the AV node lies at the base of the right atrium, electrical activation of the atria usually proceeds in an inferior to superior direction, resulting in inverted P waves in the inferior leads. (In contrast, the sinoatrial node activates the atrium in a superior to inferior direction, resulting in upright P waves in the inferior leads.) Other features of junctional rhythms include QRS complexes that are typically narrow (but may be wide if aberrancy or pre-existing bundle branch block) and occur at rates > 60 per minute. P waves usually occur within 0.12 seconds before or after the QRS complex. (Answer: a)

— Quick Review 64 —

Accelerated AV junctional rhythm

- Rate > ____ per minute — 60
- QRS complex may be narrow or aberrant (true/false) — true
- Inverted P waves in leads ____ and upright P waves in leads ____ are common — II, III, aVF; I, aVL
- P wave may precede, be buried in, or follow the QRS (true/false) — true

Low voltage

- Amplitude of the entire QRS complex (R+S) < ____ mm in all precordial leads and < ____ mm in all limb leads — 10; 5

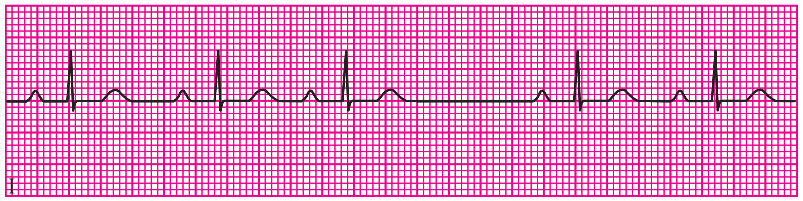
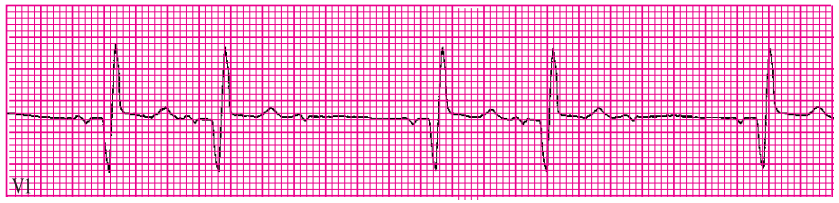
Digitalis toxicity

- Digitalis toxicity can cause almost any type of cardiac dysrhythmia or conduction disturbance except _____ — bundle branch block
- Typical abnormalities include:
  - Paroxysmal ____ tachycardia with block — atrial
  - Atrial fibrillation with ____ heart block — complete
  - Second or third-degree ____ block — AV
  - Complete heart block with accelerated ____ or ____ rhythm — junctional; idioventricular
  - Supraventricular tachycardia with ____ bundle branch block — alternating

— POP QUIZ —

Rhythm Recognition: HR < 100; Narrow QRS; Irregular RR interval

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Sinoatrial exit block, Mobitz Type I. Description: Somewhat irregular sinus rhythm with occasional absence of a P wave, caused by failure of sinus impulses to intermittenly capture the atria. Unlike Mobitz Type II SA exit block, there is *shortening* of the PP interval up to the pause, and the PP pause is *less than* twice the normal PP interval. PR interval is constant. Often a manifestation of the sick sinus syndrome.
	Answer: Sinoatrial exit block, Mobitz Type II. Description: Regular sinus rhythm with occasional absence of a P wave, caused by failure of sinus impulses to intermittently capture the atria. PP intervals before and after the pause are constant, and the PP pause is a multiple (e.g., 2x, 3x) of the normal PP interval. PR interval is constant. Often a manifestation of the sick sinus syndrome.
	Answer: 2° degree AV block, Mobitz Type II. Description: Regular sinus or atrial rhythm with intermittent nonconducted P waves and no evidence for atrial prematurity. PR interval in the conducted beats is constant, and the RR interval containing the nonconducted P wave is equal to two PP intervals. Type II AV block usually occurs within or below the bundle of His, and is associated with a wide QRS in 80% of cases.

ECG 65. Rhythm strip from a 62-year-old male with chest pain:

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- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

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- ☐ 10. Sinus tachycardia (>100)
- ☐ 11. Sinus pause or arrest
- ☐ 12. Sinoatrial exit block
- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

PACED RHYTHMS

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

ECG 65 is a 3-lead rhythm strip obtained from a 62-year-old male with chest pain. The first four beats are sinus beats at a rate of 98 beats/minute. Sinus rhythm is followed by the onset of ventricular tachycardia (arrowhead), which rapidly degenerates into ventricular fibrillation. The ST elevation in lead V_5 (arrow) is consistent with acute myocardial injury. The patient was successfully defibrillated.

Codes:

07	Sinus rhythm
25	Ventricular tachycardia
28	Ventricular fibrillation
65	ST and/or T wave abnormalities suggesting myocardial injury

Questions: ECG 65

1. Drugs commonly associated with proarrhythmia include:

   a. Amiodarone
   b. Flecainide
   c. Quinidine
   d. Propafenone

2. Factors associated with increased risk of ventricular tachycardia or sudden cardiac death after myocardial infarction include:

   a. Left ventricular dysfunction
   b. Heart rate variability
   c. Syncope
   d. Nonsustained ventricular tachycardia

Answers: ECG 65

1. Quinidine (by prolonging the QT interval), and flecainide and propafenone (by prolonging ventricular conduction, i.e., QRS complex) increase the risk of proarrhythmia. Amiodarone is associated with hypothyroidism, hyperthyroidism, pulmonary toxicity, hepatic toxicity, skin discoloration, and severe bradyarrhythmias. Proarrhythmia with ventricular tachycardia, however, is less common with amiodarone, but still occurs. (Answer: all)

2. Left ventricular ejection fraction < 40%, nonsustained ventricular tachycardia, syncope, and reduced heart rate variability are risk factors for ventricular tachycardia and sudden cardiac death following acute myocardial infarction. Sinus bradycardia and maintained beat-to-beat heart rate variability (sinus arrhythmia) are associated with reduced risk status after MI. (Answer: a, c, d)

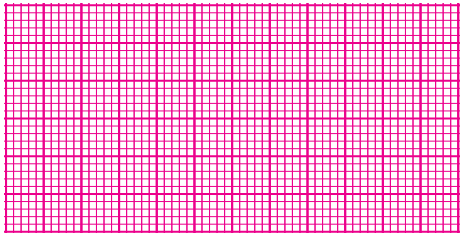
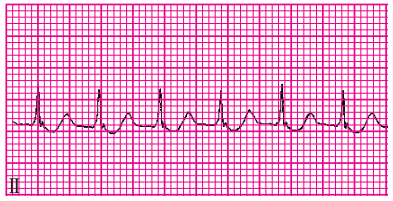
— Quick Review 65 —

Ventricular tachycardia	
• Rapid succession of three or more premature ventricular beats at a rate > ____ per minute	100
• RR intervals are usually regular but may be irregular (true/false)	true
• (Abrupt/gradual) onset and termination are evident	abrupt
• AV ____ is common	dissociation
• Look for ventricular ____ complexes and ____ beats as markers for VT	capture, fusion
Ventricular fibrillation	
• Extremely rapid and (regular/irregular) ventricular rhythm with:	irregular
▸ Chaotic, irregular deflections of (constant/varying) amplitude and duration	varying
▸ (Absence/presence) of distinct P waves, QRS complexes, and T waves	absence

— POP QUIZ —

To Treat or Not to Treat, That Is the Question

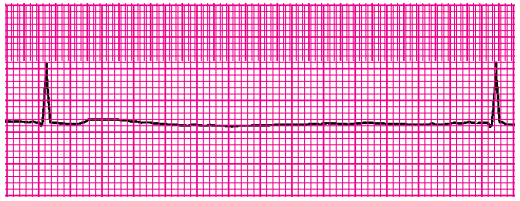
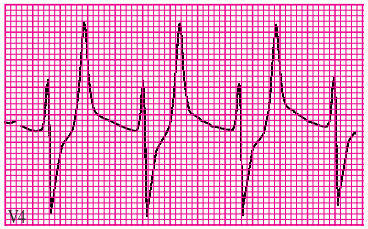
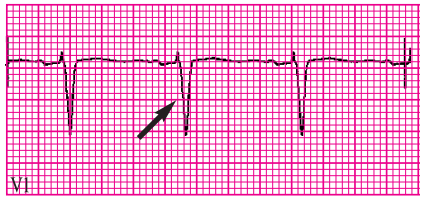
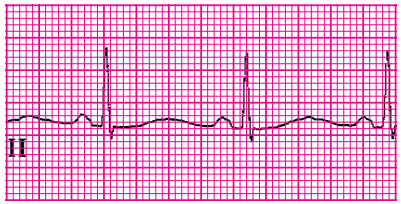
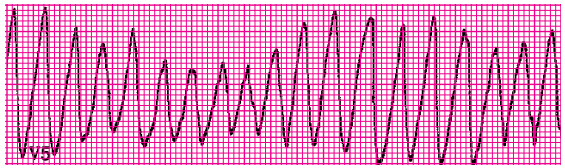
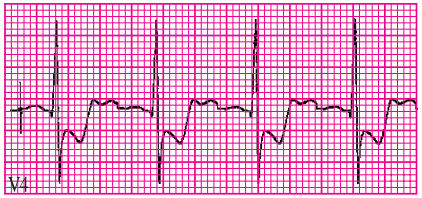
Instructions: Select the best form of treatment for each condition suggested by the following ECGs.

ECG	Choose Single Best Answer	Answer
 34 y.o. female: top (baseline 2 weeks ago); bottom (now during palpitations)	a. No treatment b. Digoxin c. Digoxin antibody d. Adenosine e. Stop aminophylline f. Procainamide (IV) g. Pericardiocentesis h. Glucose + insulin	Approximately 50% of patients with Wolff-Parkinson-White (WPW) syndrome manifest tachyarrhythmias, of which 80% is AV reentrant tachycardia, 15% atrial fibrillation, and 5% atrial flutter. *Atrial fibrillation in WPW* (seen in this ECG) is associated with a QRS that varies in width (generally wide), resulting in a rapid, irregular, wide QRS complex tachycardia that resembles VT and can degenerate into VF. The 12-lead ECG during sinus rhythm shows a short PR interval with initial slurring of the QRS (delta wave) due to pre-excitation of the ventricles from conduction over an accessory AV pathway (bundle of Kent). Treatment of atrial fibrillation in WPW consists of IV procainamide or electrical cardioversion (Answer: f)
 Pulsus paradoxus, severe dyspnea, hypotension		Causes of *electrical alternans* include pericardial effusion, severe left ventricular failure, hypertension, coronary artery disease, rheumatic heart disease, and supraventricular or ventricular tachycardia. Only one-third of patients with QRS alternans have a pericardial effusion, and only 12% of patients with pericardial effusions have electrical alternans. The clinical history in this case suggests cardiac tamponade with impending hemodynamic collapse, which should be treated with emergency pericardiocentesis. (Answer: g)
		This ECG shows a narrow complex SVT at a rate of 155 per minute, consistent with *AV nodal reentrant tachycardia* (AVNRT). The P wave is buried in the QRS complex. AVNRT is often initiated by APCs, and accounts for 60-70% of SVTs. Carotid sinus massage frequently terminates the tachycardia. Adenosine is highly effective at interrupting the reentrant loop and restoring sinus rhythm. (Answer: d)

— POP QUIZ —

Pattern Recognition: Antiarrhythmic Drug Effect vs. Toxicity

Instructions: Determine which of the following ECGs are consistent with antiarrhythmic drug effect and which are consistent with antiarrhythmic drug toxicity.

Choose All That Apply		Answer
A. 	**B.** 	*Antiarrhythmic drug effect* is suggested by mild increase in QT interval, prominent U waves (one of the earliest findings), nonspecific ST and/or T wave changes, or a decrease in atrial flutter rate. (Answer: none) *Antiarrhythmic drug toxicity* is suggested by marked prolongation of the QT interval (choice "d"), widening of the QRS complex (choice "c"), AV block, marked sinus bradycardia, sinus arrest (choice "a"), sinoatrial exit block, or ventricular arrhythmias including torsade de pointes (choice "e"). Peaked T waves (choice "b") or marked ST depression with deeply inverted T waves (choice "f") are not associated with antiarrhythmic drug toxicity. (Answer: a, c, d, e)
C. 	**D.** 	
E. 	**F.** 	

ECG 66. 51-year-old female with orthopnea and paroxysmal nocturnal dyspnea:

GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
- ☐ 11. Sinus pause or arrest
- ☐ 12. Sinoatrial exit block
- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
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- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

- ☐ 20. AV junctional premature complexes
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- ☐ 32. AV block, 2:1
- ☐ 33. AV block, 3°
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- ☐ 35. AV dissociation

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- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
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- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

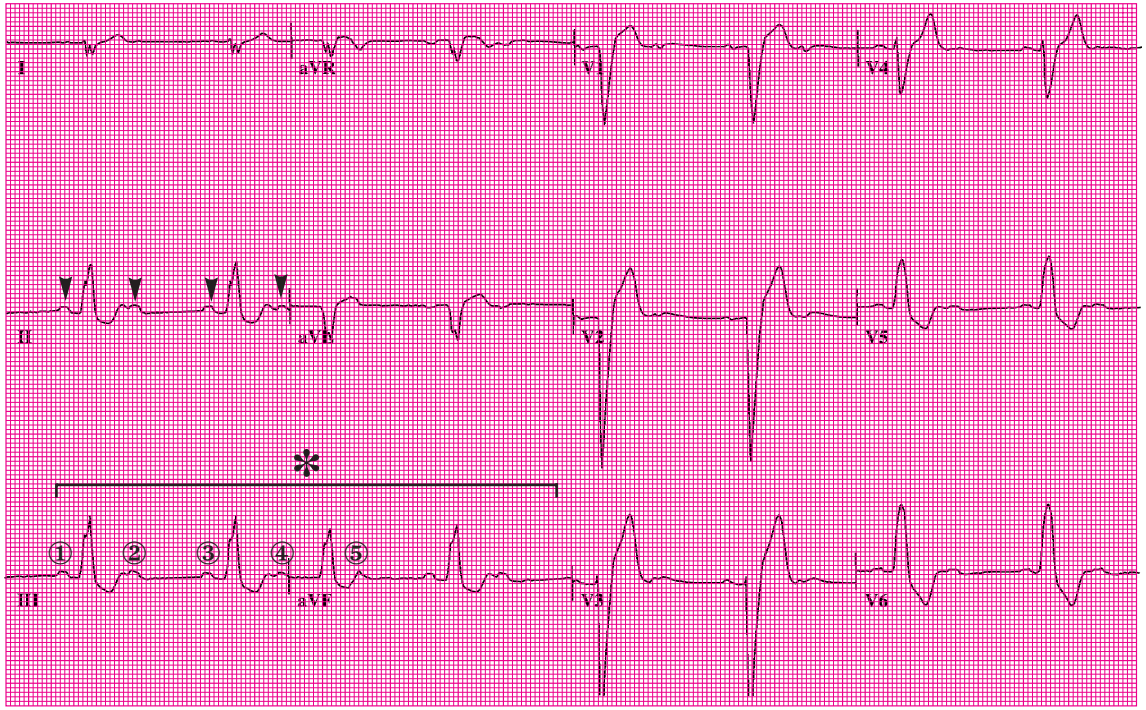
PACED RHYTHMS

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

ECG 66 was obtained in a 51-year-old female with a history of orthopnea and paroxysmal nocturnal dyspnea. The ECG shows sinus rhythm with predominantly 2:1 AV block (arrowheads mark P waves), left bundle branch block (LBBB) with secondary ST-T changes, and right axis deviation. Close inspection reveals evidence for Mobitz Type I (Wenckebach) second-degree AV block (asterisk) — the third P wave conducts at a normal PR interval, the fourth P wave at a prolonged PR interval, and the fifth P wave (hidden in the T wave) is blocked. The Q waves and ST elevation in leads V_1 - V_3 are most likely due to LBBB, rather than acute anteroseptal MI (LBBB often results in a pseudoinfarct pattern).

Codes:

07	Sinus rhythm
30	AV block, 2º- Mobitz type I (Wenckebach)
32	AV block, 2:1
37	Right axis deviation (> +100º)
47	LBBB, complete



Questions: ECG 66

1. Right axis deviation is defined by a QRS axis rightward between:

   a. 60° - 100°
   b. 90° - 180°
   c. 100° - 270°
   d. 110° - 270°

2. In the setting of 2:1 AV block, the presence of a wide complex QRS makes the mechanism of AV block more likely to be:

   a. Mobitz Type I
   b. Mobitz Type II

Answer: ECG 66

1. Right axis deviation is defined as a QRS axis between 100° and 270°. (Answer: c)

2. It is often difficult to distinguish Mobitz I from Mobitz II second-degree AV block when 2:1 AV block is present throughout the tracing. If classic Mobitz I (Wenckebach) is present on another ECG or on monitoring strips, the mechanism of block is probably Mobitz I. If abnormal QRS conduction is present (e.g., LBBB or bifascicular block), Mobitz II is more likely. (Answer: b)

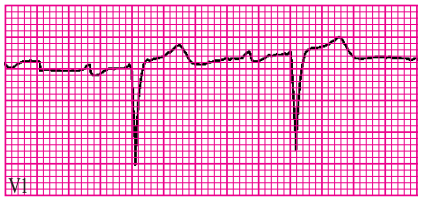
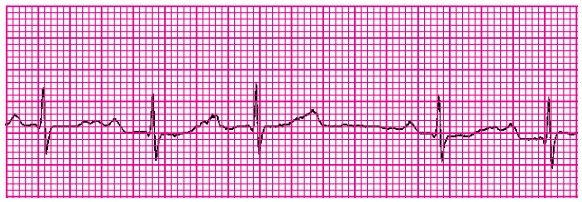
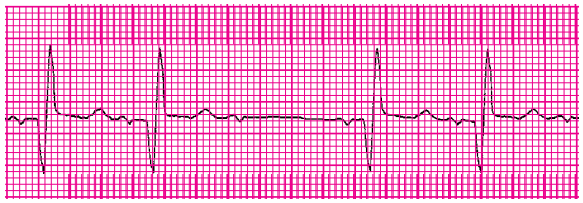
— Quick Review 66 —

AV block, 2° - Mobitz Type I (Wenckebach)	
• Progressive prolongation of the ____ interval and shortening of the ____ interval until a P wave is blocked	PR RR
• RR interval containing the nonconducted P wave is (less than/equal to/greater than) the sum of two PP intervals	less than
• Results in ____ beating due to the presence of nonconducted P waves	group
LBBB, complete	
• QRS duration ≥ ____ seconds	0.12
• Onset of intrinsicoid deflection in leads I, V_5, V_6 > ____ seconds	0.05
• Broad monophasic R waves in leads ____, which are usually notched or slurred	I, V_5, V_6
• Secondary ST & T wave changes in the (same/opposite) direction to the major QRS deflection	opposite
• ____ or ____ complex in the right precordial leads	rS or QS
• LBBB (does/does not) interfere with determination of QRS axis and the diagnoses of ventricular hypertrophy and acute MI	does

— POP QUIZ —

Pattern Recognition: AV Conduction Abnormalities

Instructions: Match the following ECGs with all descriptions that apply.

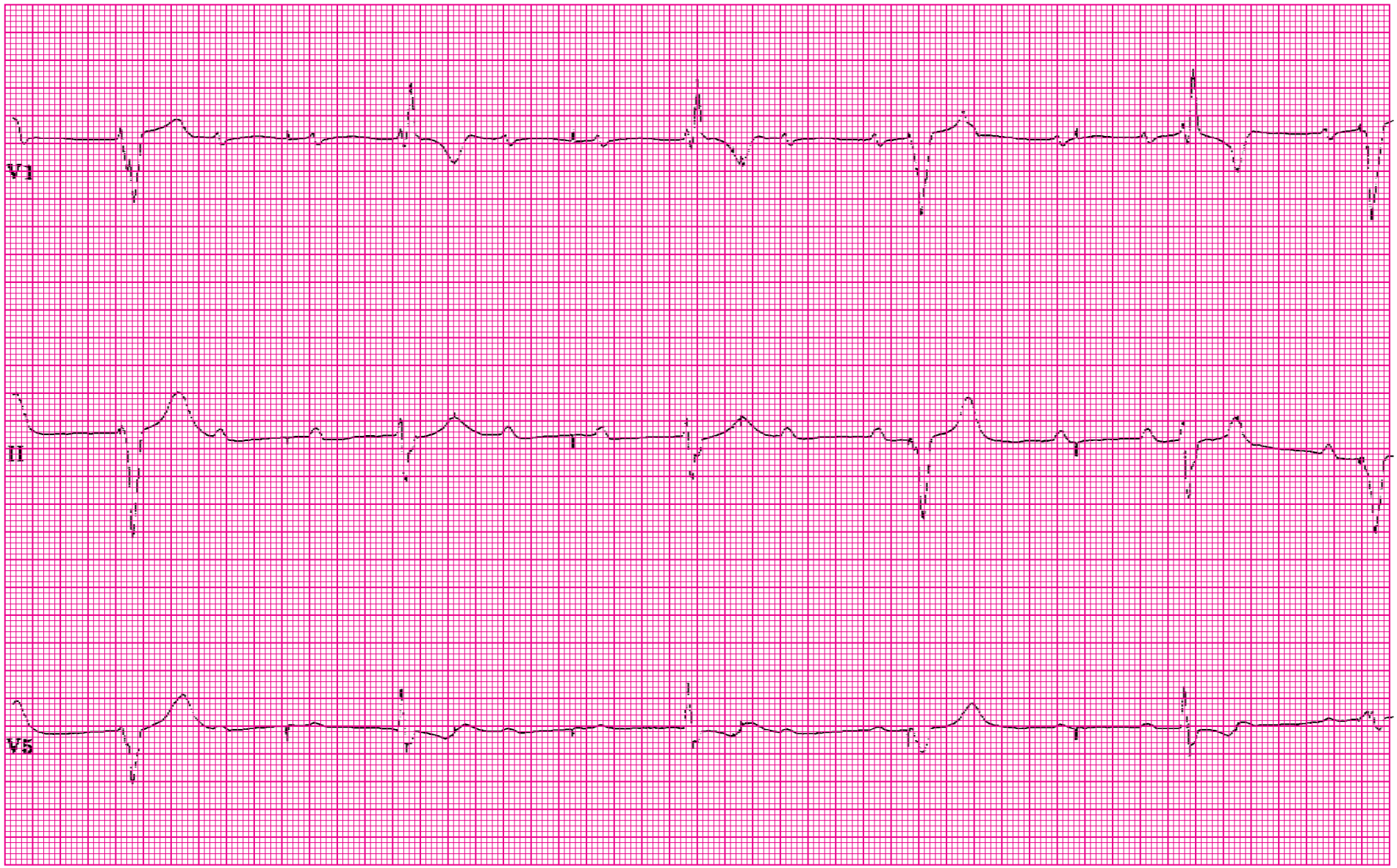
ECG	Choose All That Apply	Answer
	a. Reflects prolonged conduction from the sinus node to atrial tissue b. 1° AV block c. Can be seen in normal individuals d. 2° AV block, Type I e. Grouped beating due to nonconducted P waves f. Block usually occurs at level of AV node g. More common in inferior MI than anterior MI h. 2° AV block, Type II i. Block usually within or below bundle of His j. Block may improve with carotid sinus massage and worsen with atropine k. Can be either Mobitz Type I or II l. Atrial and ventricular rhythms are independent of each other m. 3° AV block	*1° AV block* represents delay from the onset of atrial depolarization to the onset of ventricular repolarization, and manifests as a PR interval ≥ 0.20 seconds. Each P wave is followed by a QRS complex. Causes include high vagal tone, drugs, acute rheumatic fever, myocarditis, and congenital heart disease; occasionally seen in normals. (Answer: b, c, f, g)
		*Mobitz Type I (Wenkebach) 2º AV block* results in a regular sinus or atrial rhythm with intermittent nonconducted P waves (resulting in "grouped beating"), and progressive prolongation of the PR interval and shortening of the RR interval until a P wave is blocked; the RR interval containing the nonconducted P wave is less than two PP intervals. Block usually occurs within the AV node, and is associated with a narrow QRS complex. AV block may improve with maneuvers that increase heart rate (e.g., atropine) and worsen with maneuvers that reduce heart rate (e.g., carotid sinus massage). Sometimes seen in normals. (Answer: c, d, e, f, g)
		*Mobitz Type II 2º AV block* results in a regular sinus or atrial rhythm with intermittent nonconducted P waves, a constant PR interval in the conducted beats, and an RR interval containing the nonconducted P wave equal to two RR intervals. Block usually occurs within or below the bundle of His, and is associated with a wide QRS complex. AV block may worsen with maneuvers that increase heart rate and improve with maneuvers that reduce rate. (Answer: e, h, i, j)

Differential Diagnosis

Right Axis Deviation (> +100°)

- Right ventricular hypertrophy
- Vertical heart
- Chronic obstructive pulmonary disease
- Pulmonary embolus
- Left posterior fascicular block
- Lateral wall myocardial infarction
- Dextrocardia
- Limb lead reversal
- Ostium secundum atrial septal defect

ECG 67. Rhythm strip from a 54-year-old female with lightheadedness:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
- ☐ 11. Sinus pause or arrest
- ☐ 12. Sinoatrial exit block
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- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
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AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

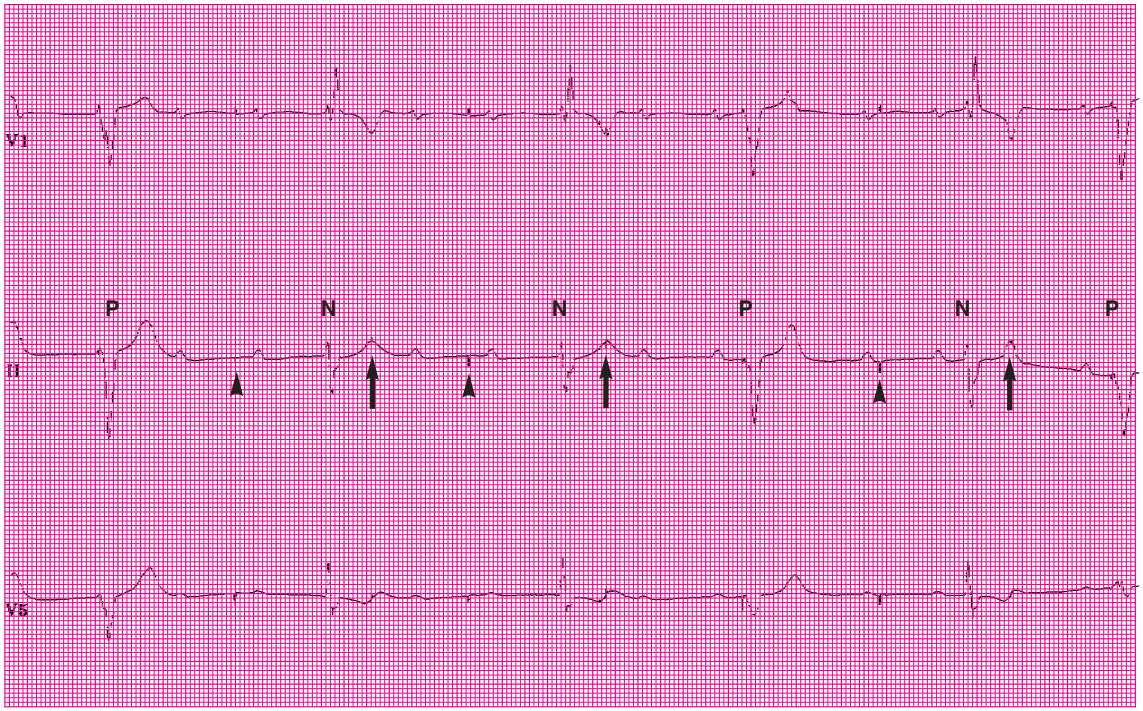
PACED RHYTHMS

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

ECG 67 is a 3-lead rhythm strip obtained from a 54-year-old female with lightheadedness. The ECG shows ventricular pacing with intermittent failure to capture and failure to sense: Failure to capture results in pacemaker spikes not followed by QRS complexes (arrowheads), and failure to sense results in premature pacemaker spikes (arrows) relative to the V-V interval of the pacemaker (separation between the first and second pacemaker spikes), the key timing interval for the pacemaker. The intrinsic rhythm is sinus at a rate of 88 beats/minute (P waves best seen in lead II) with complete heart block and a ventricular escape rhythm at a rate of 29 beats/minute. The sinus and ventricular escape rhythms are independent of each other, resulting in AV dissociation. (The RBBB pattern is due to abnormal ventricular activation from the ventricular escape rhythm, not true bundle branch block.) Left atrial enlargement is also present. N = native beat; P = paced beat.

Codes:

06	Left atrial abnormality/enlargement
07	Sinus rhythm
27	Ventricular escape complexes or rhythm
33	AV block, 3°
35	AV dissociation
93	Pacemaker malfunction, not constantly capturing (atrium or ventricle)
94	Pacemaker malfunction, not constantly sensing (atrium or ventricle)



Questions: ECG 67

1. Twiddler's syndrome is:

    a. Lightheadedness associated with atrial contraction against closed atrio-ventricular (AV) valves with VVI pacemakers
    b. Displacement of a pacemaker lead caused by patient manipulation of the subcutaneous lead wires
    c. A form of pacemaker-mediated tachycardia first described by Dr. Felix Twiddler

2. Paced ventricular beats with RBBB morphology is consistent with:

    a. Perforation of the pacemaker lead across the septum and into the left ventricle
    b. A left ventricular epicardial lead
    c. Normal lead placement in the right ventricular apex

Answers: ECG 67

1. Twiddler's syndrome refers to patient manipulation of pacemaker lead wires, resulting in rotation of the pulse generator in the pocket lead and dislodgement with failure to capture and/or sense. Pacemaker syndrome refers to episodic lightheadedness from enhanced baroreflex response to atrial contraction against closed AV valves. This is usually associated with cannon "a" waves in the jugular pulse, and occurs mainly with VVI pacemakers. No novel syndromes have been attributed to a Dr. Twiddler. (Answer: b)

2. Normally-positioned pacemaker leads in the right ventricular apex show paced QRS complexes with LBBB pattern. Paced beats with RBBB morphology should raise concern that the pacing lead has entered the left ventricle, either from perforation of the septum or, in unusual cases, passage of the catheter across an atrial septal defect or patent foramen ovale. RBBB morphology can also be seen in patients with left ventricular epicardial leads placed years ago via thoracotomy. (Answer: a, b)

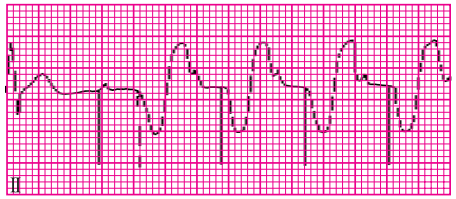
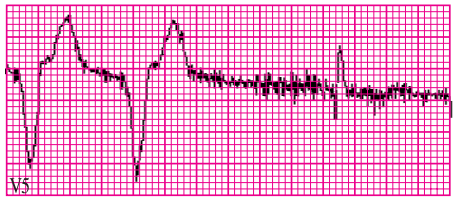
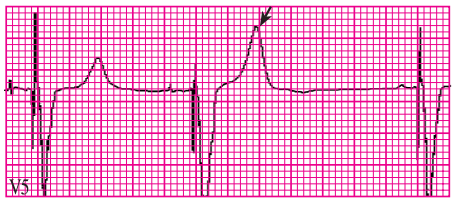
— Quick Review 67 —

Pacemaker malfunction, not constantly sensing (atrium or ventricle)	
• Pacemakers in the inhibited mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	inhibited
• Pacemakers in the triggered mode: Pacemaker fails to be triggered by an appropriate ____ depolarization	intrinsic
• Premature depolarizations may not be sensed if they fall within the programmed ____ period of the pacemaker, *or* have insufficient ____ at the sensing electrode site	refractory amplitude
Pacemaker malfunction, not constantly capturing (atrium or ventricle)	
• Failure of pacemaker stimulus to be followed by a ____	depolarization
• Rule out "pseudo-malfunction" (i.e., pacer stimulus falls into the ____ period of ventricle)	refractory

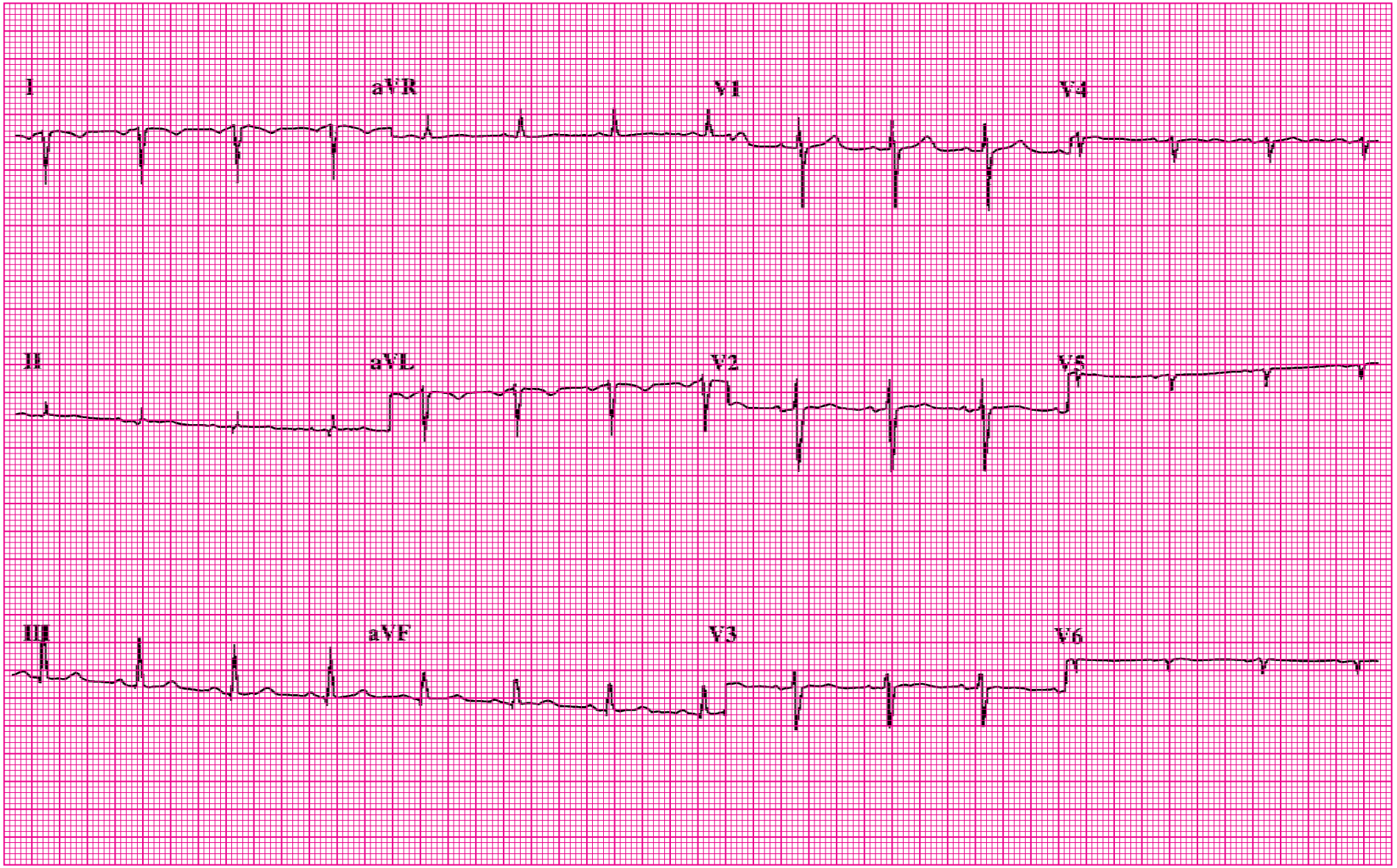
— POP QUIZ —

Pattern Recognition: Pacemaker Malfunction

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Failure to capture b. Causes include lead displacement, lead fracture, and increased pacing threshold c. Failure to sense d. Occurs with low amplitude signals e. Failure to fire f. Oversensing T waves g. Myopotential inhibition h. Pacemaker-mediated tachycardia (PMT) i. Can be corrected by increasing the post-ventricular atrial refractory period (PVARP) j. Proper pacing (no malfunction)	*Oversensing of T waves* occurs when T waves are mistaken for R waves, which resets the pacemaker clock and results in failure of the pacemaker to discharge at the appropriate time. The ventricular stimulus escape (V-A) interval is timed from the T wave, instead of the R wave. This problem can be corrected by reprogramming the sensitivity of the chamber that is oversensing. (Answer: f)
		*Oversensing of myopotentials (myopotential inhibition)* occurs when myopotentials (muscular potentials from arm movements) are inappropriately sensed as cardiac potentials, which resets the pacemaker clock and inhibits pacemaker output. Paced RR intervals tend to be irregular. This problem is more common with unipolar pacemakers. (Answer: g)
		*Pacemaker-mediated tachycardia* (PMT) occurs with atrial-sensing dual chamber pacemakers, and manifests as rapid pacing at or near the programmed upper rate limit. PMT is due to a repetitive cycle of ventricular paced beat (or VPC) → retrograde atrial activity → atrial sensing → ventricular paced beat, and can usually be corrected by increasing the post-ventricular atrial refractory period (PVARP) of the pacemaker. (In this ECG, retrograde P waves deform the downslope of the T waves.) (Answer: h, i)

ECG 68. 18-year-old asymptomatic male:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

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

SUGGESTED CLINICAL DISORDERS

- ☐ 70. Digitalis effect
- ☐ 71. Digitalis toxicity
- ☐ 72. Antiarrhythmic drug effect
- ☐ 73. Antiarrhythmic drug toxicity
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- ☐ 88. Hypothermia
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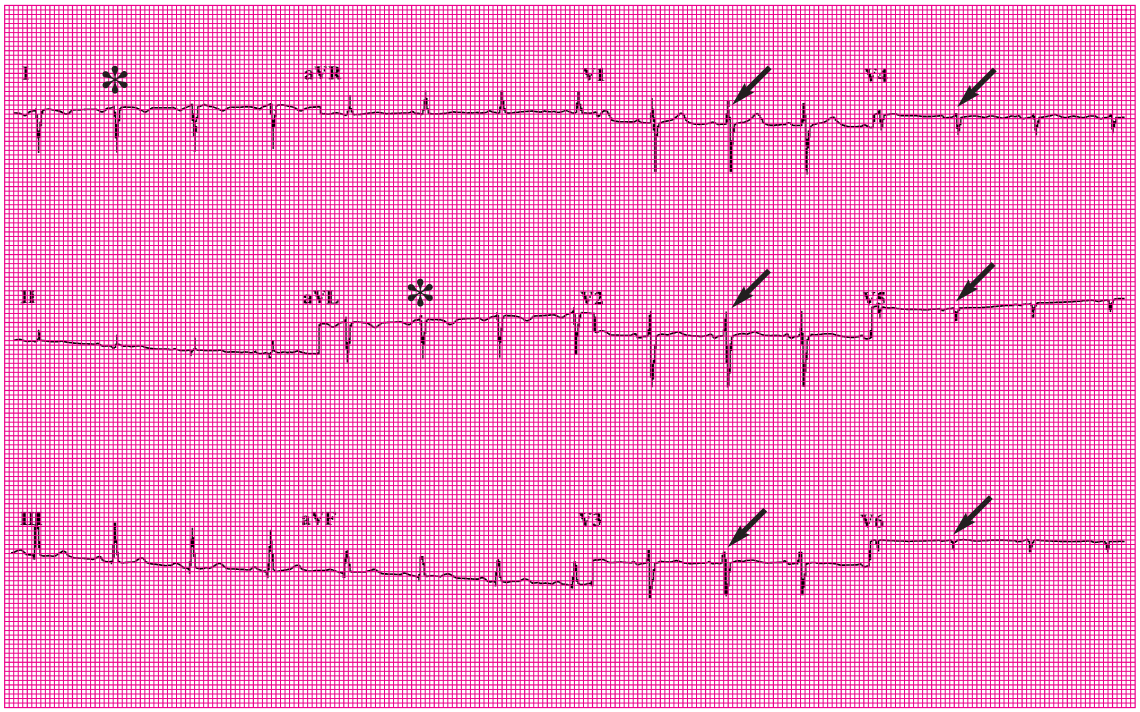
PACED RHYTHMS

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

ECG 68 was obtained in a 18-year-old asymptomatic male being screened for participation in high school basketball. The most notable feature of the ECG is the negative P-QRS-T in leads I and aVL (asterisks), which may be seen in both dextrocardia and limb lead reversal. The diminishing (reverse) R wave amplitude (arrows) across the precordium (V_1-V_6) confirms the diagnosis of dextrocardia. Right axis deviation and nonspecific ST-T abnormalities are also present.

Codes:

07	Sinus rhythm
37	Right axis deviation (> +100°)
63	Nonspecific ST and/or T wave abnormalities
80	Dextrocardia, mirror image



Questions: ECG 68

1. Dextrocardia is associated with:

   a. QT prolongation
   b. Low voltage in the limb leads
   c. Inverted P, QRS, and T waves in leads I and aVL
   d. Prominent R wave voltage in the left precordial leads (V_4-V_6)

2. Isolated dextrocardia (dextrocardia without inversion of other viscera) is almost invariably associated with other serious congenital cardiac malformations:

   a. True
   b. False

Answers: ECG 68

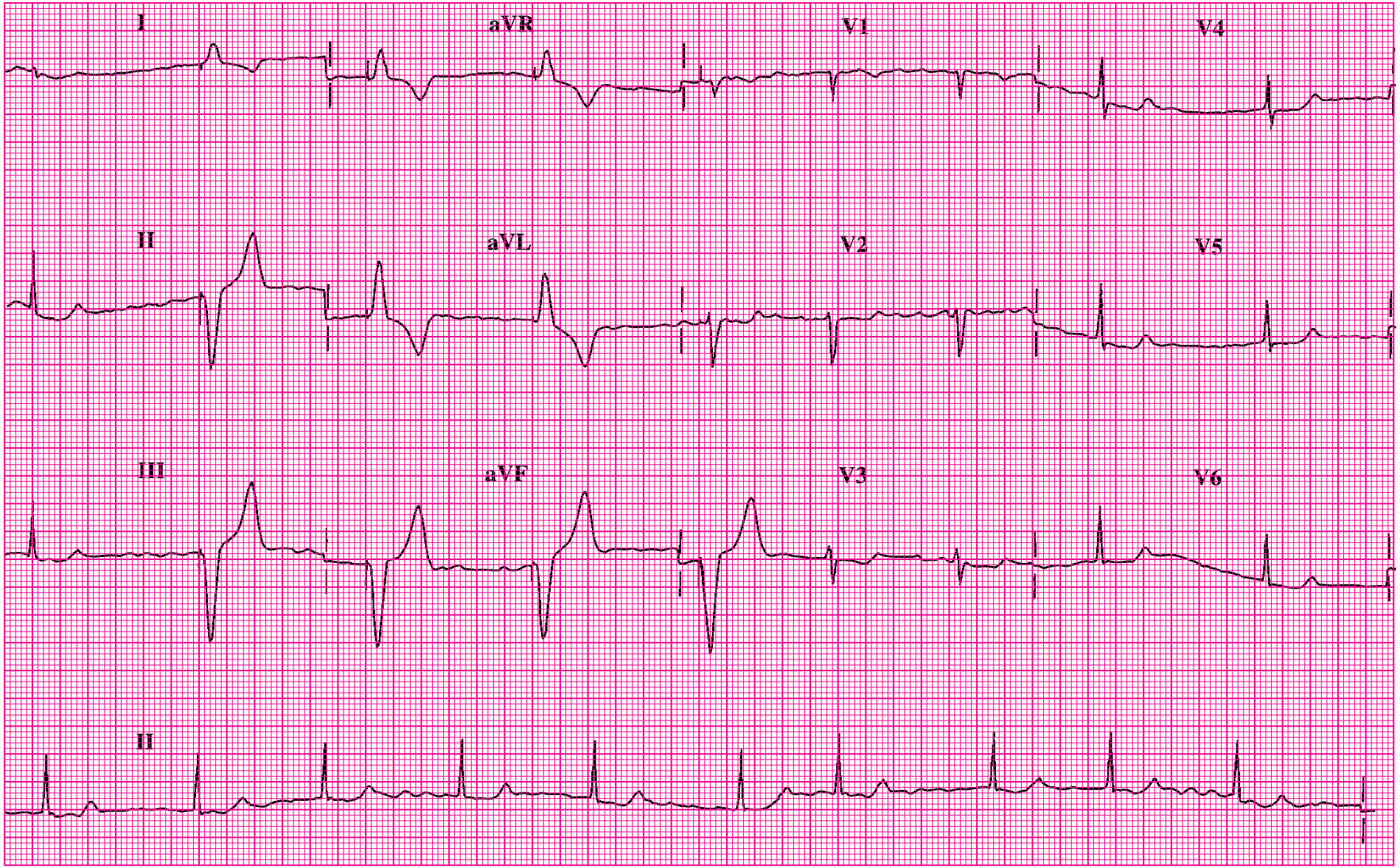
1. Dextrocardia is a rare condition characterized by congenital malpositioning of the heart in the right side of the chest. ECG features include inversion of the P-QRS-T in leads I and aVL, and decreasing R wave amplitude from leads V_1-V_6. (Answer: c)

2. In ***mirror-like dextrocardia***, the most common form of dextrocardia, the abdominal and thoracic viscera (in addition to the heart) are transposed to the side opposite their usual locations (dextrocardia with "situs inversus"). This form of dextrocardia is generally not associated with severe congenital cardiac abnormalities (other than the malposition, which does not affect cardiac function). In ***isolated dextrocardia***, the heart is rotated to the right side of the chest but other viscera remain in their usual locations. This type of dextrocardia is almost always associated with serious congenital cardiac abnormalities, resulting in clinical difficulties in infancy or early childhood. (Answer: a)

— Quick Review 68 —

Right axis deviation	
• Mean QRS axis between ____ and ____ degrees	101, 270
Dextrocardia, mirror image	
• P-QRS-T in leads ____ are inverted or "upside down"	I, aVL
• Decreasing ____ wave amplitude from leads V_1-V_6	R
• Dextrocardia and ____ can both produce an upside down P-QRS-T in leads I and aVL. To distinguish between these conditions, look at the R wave pattern in V_1 - V_6:	lead reversal
▸ Reverse R wave progression suggests (dextrocardia/lead reversal)	dextrocardia
▸ Normal R wave progression suggests (dextrocardia/lead reversal)	lead reversal

ECG 69. 76-year-old asymptomatic female:



GENERAL FEATURES

- ☐ 01. Normal ECG
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- ☐ 03. Incorrect electrode placement
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P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
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- ☐ 22. AV junctional rhythm/tachycardia

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- ☐ 31. AV block , 2°-Mobitz type II
- ☐ 32. AV block, 2:1
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- ☐ 35. AV dissociation

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- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 40. Left ventricular hypertrophy
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- ☐ 42. Combined ventricular hypertrophy

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- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
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- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
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- ☐ 69. Prominent U waves

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- ☐ 71. Digitalis toxicity
- ☐ 72. Antiarrhythmic drug effect
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- ☐ 75. Hypokalemia
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- ☐ 77. Hypocalcemia
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PACED RHYTHMS

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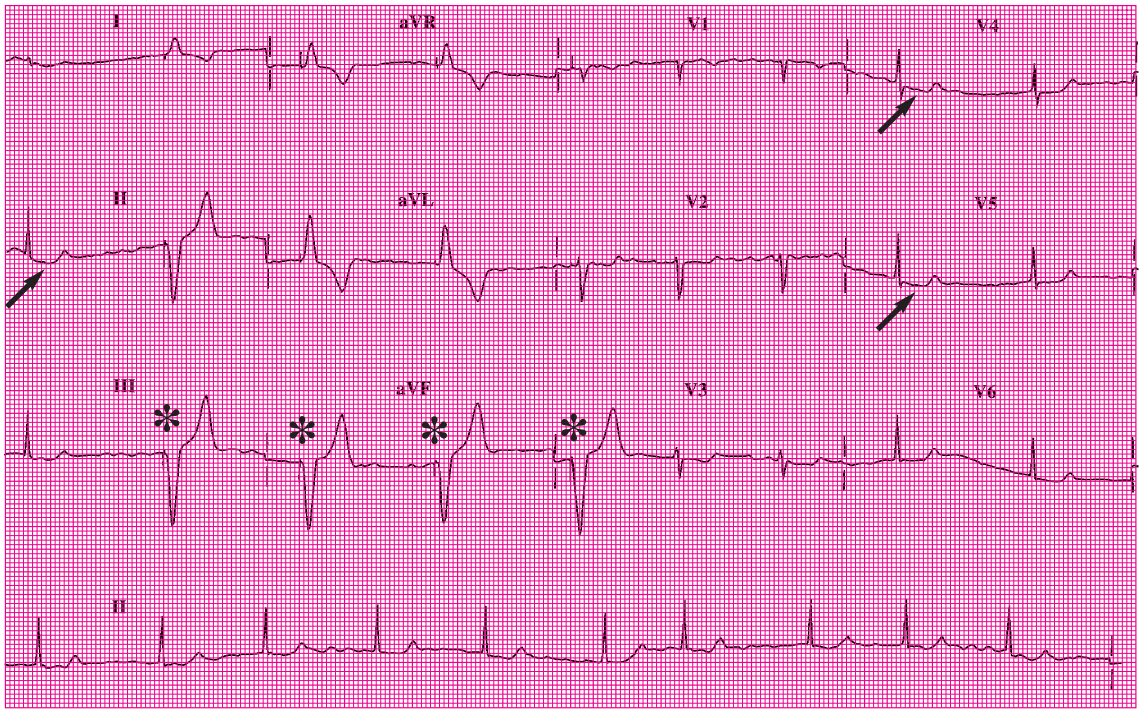
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 69 was obtained from a 76-year-old asymptomatic female, and shows atrial fibrillation with appropriate ventricular demand pacing (asterisks) at 50 beats/minute. Nonspecific repolarization abnormality is noted in native QRS complexes. Sagging ST segment depression (arrows) is consistent with digitalis effect.

Codes:

19	Atrial fibrillation
63	Nonspecific ST and/or T wave abnormalities
70	Digitalis effect
91	Ventricular demand pacemaker (VVI), normally functioning



Questions: ECG 69

1. The diagnosis of ventricular demand (VVI) pacing requires:

   a. Inhibition of atrial output in response to native atrial activity
   b. Variable pacing rates
   c. Retrograde VA conduction
   d. Inhibition of ventricular output in response to an intrinsic QRS complex

Answers: ECG 69

1. A ventricular demand (VVI) pacemaker senses and paces only in the ventricle, and is oblivious to native atrial activity. If constant ventricular pacing is noted throughout the tracing, it is impossible to distinguish ventricular demand from asynchronous ventricular pacing. Thus, the diagnosis of ventricular demand pacing requires evidence of appropriate inhibition of pacemaker output in response to a native QRS (at least one). Retrograde VA conduction may occur, but is not required for the diagnosis. The pacing rate of a VVI pacemaker is generally constant at the programmed pacing rate; in contrast, VVI-R pacing allows for variable pacing rates in response to physiologic needs. (Answer: d)

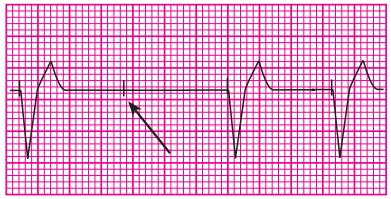
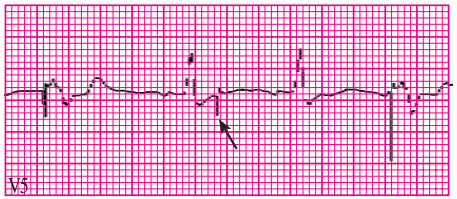
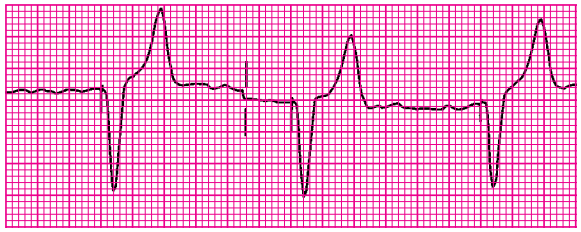
— Quick Review 69 —

Ventricular demand pacing	
• Pacemaker stimulus followed by a QRS complex that has (the same/different) morphology compared to the intrinsic QRS	different
• Must demonstrate ____ of pacemaker output in response to intrinsic QRS	inhibition
Digitalis effect	
• ____ ST segment depression with upward (concavity/convexity)	Sagging concavity
• T wave flat, inverted, or ____	biphasic
• QT interval (shortened/prolonged)	shortened
• U wave amplitude (increased/decreased)	increased
• PR interval (shortened/lengthened)	lengthened

— POP QUIZ —

Pattern Recognition: Pacemaker Malfunction

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Failure to capture b. Causes include lead displacement, lead fracture, and increased pacing threshold c. Failure to sense d. Failure to fire e. Oversensing T waves f. Myopotential inhibition g. Pacemaker-mediated tachycardia (PMT) h. Can be corrected by increasing the post-ventricular atrial refractory period (PVARP) i. Proper pacing (no malfunction)	*Failure to capture* occurs when a pacing spike (atrial or ventricular) is not followed by an appropriate depolarization. Causes include lead displacement, perforation, increased pacing threshold (from MI, flecainide, amiodarone, hyperkalemia), lead fracture or insulation break, pulse generator failure (from battery depletion), or inappropriate reprogramming. Failure to capture must be differentiated from "pseudo-malfunction," in which the pacer stimulus falls into refractory period of ventricle. (Answer: a, b)
		*Failure to sense* occurs when the pacemaker is not inhibited by an appropriate intrinsic depolarization, resulting in an extra early asynchronous pacing spike. Pacemaker timing is not reset by intrinsic or ectopic beats, resulting in a paced rhythm that competes with the intrinsic rhythm. Failure to sense occurs with low amplitude signals (especially VPCs), inappropriate programming of pacemaker sensitivity, and all causes of failure to capture, above. Reprogramming the sensitivity of the pacemaker often corrects the problem. (Answer: b, c)
		Proper ventricular demand pacing is seen (no malfunction). (The perforated vertical line between the first and second beats is a lead switch indicator, not a pacemaker spike.) (Answer: i)

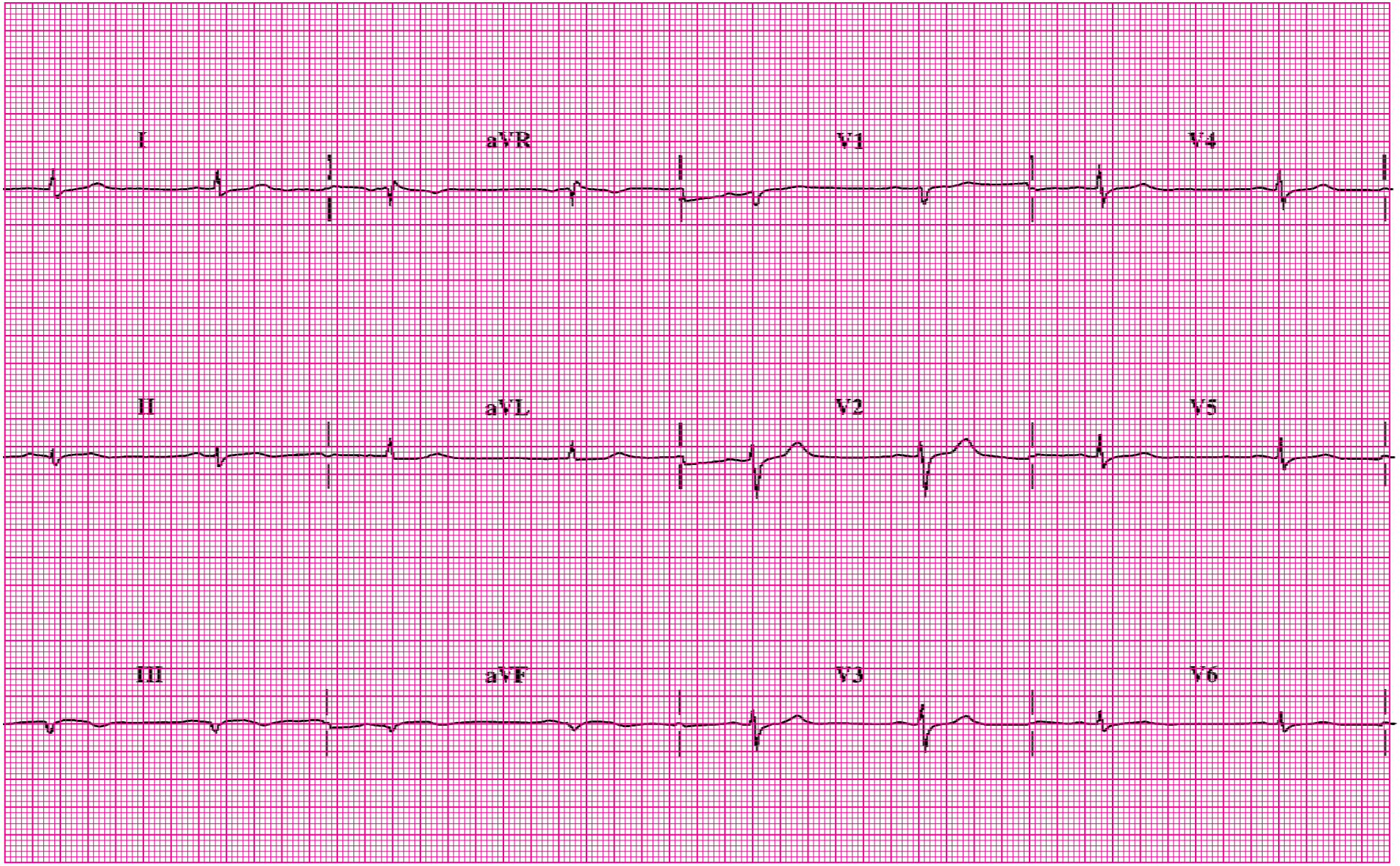
— POP QUIZ —

Find The Mistake

Instructions: Identify the incorrect ECG feature(s) for each ECG diagnosis listed below.

ECG Diagnosis and Features	Mistake
Antiarrhythmic drug effect • Prominent U waves (one of the earliest findings) • Prolonged QT interval • Nonspecific ST and/or T wave changes • Widening of the QRS complex • AV block	Widening of the QRS complex and AV block are consistent with drug toxicity (not drug effect)
Pericarditis • Classic evolutionary ST-T pattern consists of 4 stages: 1) Diffuse upwardly concave ST elevation; 2) T waves invert; 3) ST junction returns to baseline & T wave amplitude decreases; 4) ECG returns to normal • Other clues include sinus tachycardia, PR depression late, and low voltage QRS	ST junction usually returns to baseline before (not after) T waves invert; PR depression occurs early (not late)
Digitalis effect • Sagging ST segment depression with upward convexity • T wave flat, inverted, or biphasic • QT interval shortened • U wave amplitude increased • PR interval lengthened	ST segments have upward concavity (not convexity)
Digitalis toxicity • Typical abnormalities include paroxysmal atrial tachycardia with block, atrial fibrillation with complete heart block, second- or third-degree AV block, complete heart block with accelerated junctional or idioventricular rhythm, and bundle branch block	Isolated bundle branch block is not a manifestation of digitalis toxicity

ECG 70. 49-year-old woman with dry skin, weakness, cold intolerance, constipation & weight gain:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

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- ☐ 21. AV junctional escape complexes
- ☐ 22. AV junctional rhythm/tachycardia

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
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- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
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- ☐ 51. Anterolateral (age recent or acute)
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- ☐ 63. Nonspecific ST and/or T wave abnormalities
- ☐ 64. ST and/or T wave abnormalities suggesting myocardial ischemia
- ☐ 65. ST and/or T wave abnormalities suggesting myocardial injury
- ☐ 66. ST and/or T wave abnormalities suggesting electrolyte disturbances
- ☐ 67. ST and/or T wave abnormalities secondary to hypertrophy
- ☐ 68. Prolonged QT interval
- ☐ 69. Prominent U waves

SUGGESTED CLINICAL DISORDERS

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

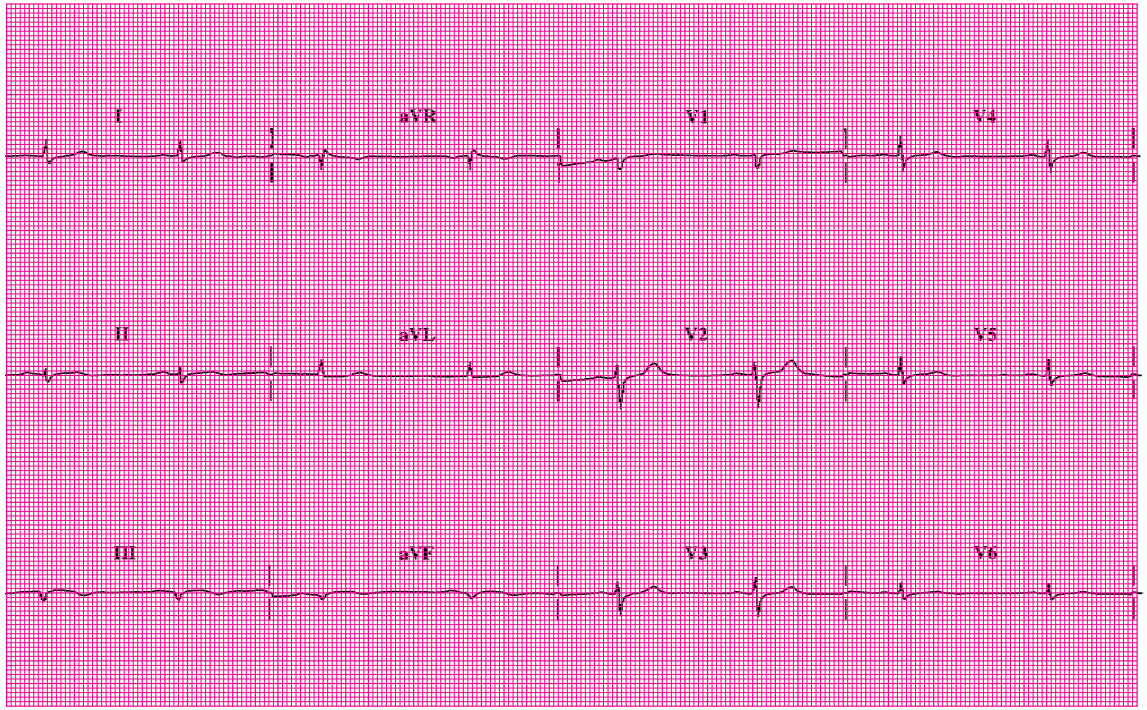
PACED RHYTHMS

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

ECG 70 was obtained in a 49-year-old woman with complaints of dry skin, weakness, cold intolerance, constipation, and weight gain. The ECG shows sinus bradycardia with left axis deviation, relatively low voltage QRS complexes (which do not quite meet criteria for formal coding of low voltage), age indeterminate or old inferior myocardial infarction, and minor nonspecific ST-T abnormalities. In the context of the clinical presentation, these ECG findings are consistent with myxedema. This woman had a past history of inferior MI and was shown to be profoundly hypothyroid.

Codes:

09	Sinus bradycardia (< 60)
58	Inferior Q wave MI (age indeterminate or old)
87	Myxedema



Questions: ECG 70

1. The differential diagnosis for low voltage ECG includes:

   a. Sarcoidosis of the heart
   b. Myxedema
   c. Congestive heart failure
   d. Pericardial effusion
   e. Pleural effusion
   f. Amyloid heart
   g. COPD
   h. Diffuse coronary artery disease
   i. Obesity
   j. Pectus excavatum

— Quick Review 70 —

Low voltage, limb and precordial leads	
• Amplitude of the entire QRS complex (R+S) < ___ mm in all precordial leads and < ____ mm in all limb leads	10 5
Inferior MI, age indeterminate or probably old	
• Abnormal Q waves (with/without) ST elevation in at least two of leads ____	without II, III, aVF
Myxedema	
• (High/low) QRS voltage in all leads	Low
• Sinus (tachycardia/bradycardia)	bradycardia
• T wave flattened or (upright/inverted)	inverted
• PR interval may be (shortened/prolonged)	prolonged
• Frequently associated with pericardial ____	effusion
• Electrical alternans may occur (true/false)	true

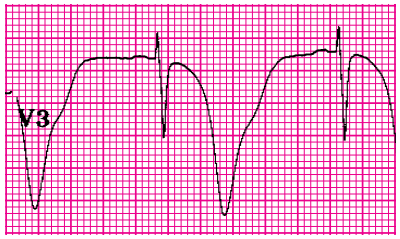
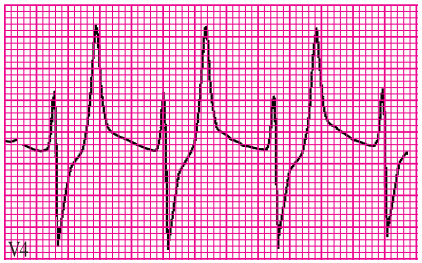
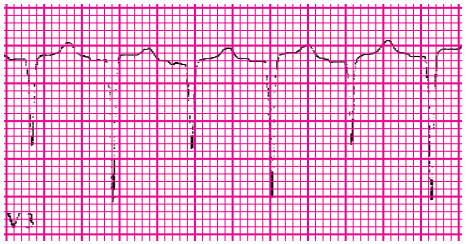
Answers: ECG 70

1. The amplitude of the QRS complex is often decreased by conditions that increase the amount of body tissue (obesity), air (COPD, pneumothorax), fluid (pericardial or pleural effusion), fibrous tissue (coronary artery disease) or other infiltrative substances (sarcoid, amyloid, myxedema) between the myocardium and surface ECG electrodes. Pectus excavatum (funnel chest) often increases QRS amplitude. (Answer: all except j)

— POP QUIZ —

Pattern Recognition: ECG/Clinical Correlation

Instructions: Match the ECG to the most likely clinical presentation.

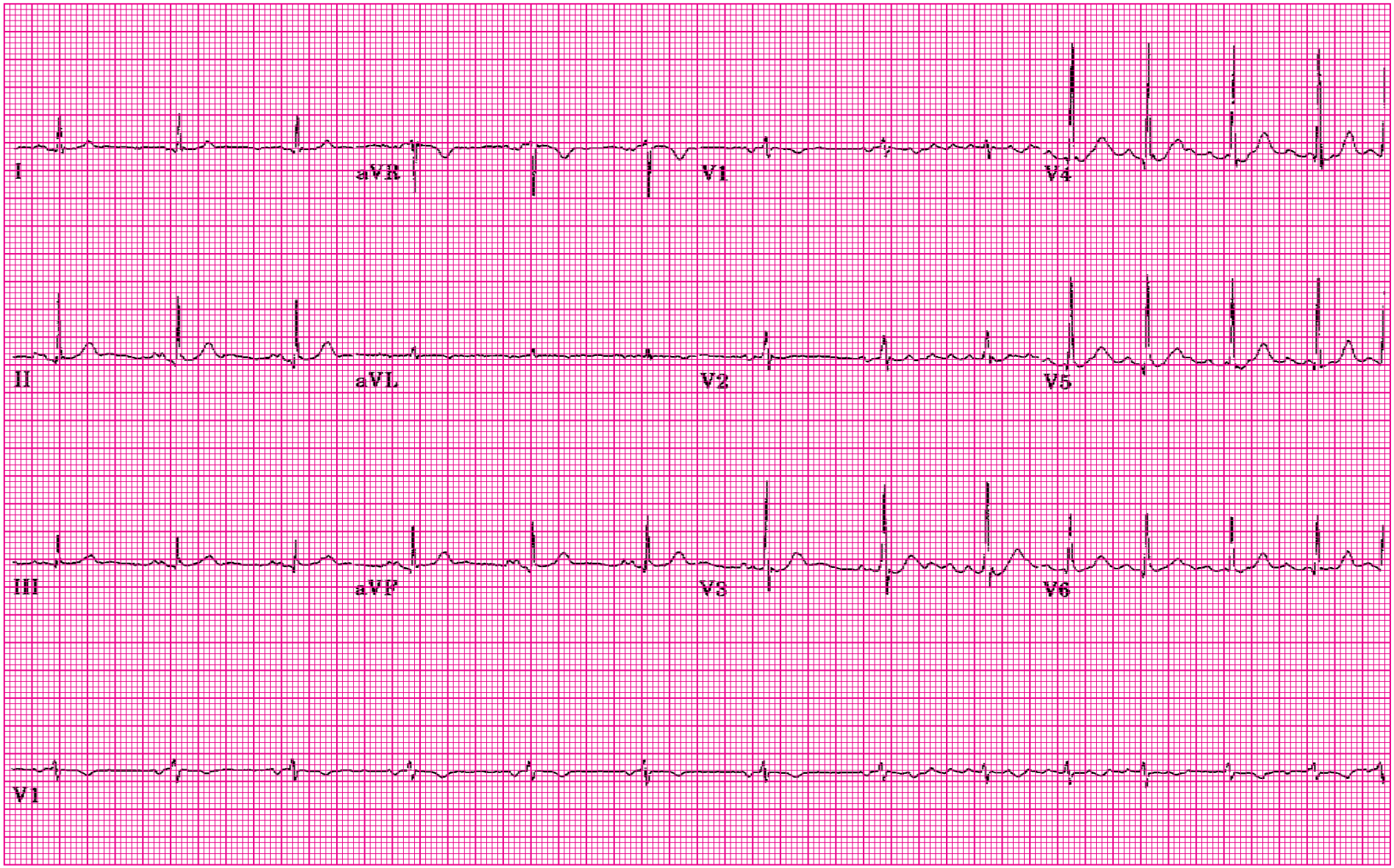
ECG	Choose Single Best Answer	Answer
	a. Severe headache, papilledema b. Dyspnea, constipation, impaired memory, fatigue c. Red-green color blindness, nausea, vomiting d. Acute oliguria 2° to rhabdomyolysis e. Murmur in a Down's Syndrome patient f. Acute exacerbation of chronic bronchitis g. Prolonged exposure to extreme cold h. Dyspnea and pulses paradoxus in a renal failure patient i. Acute onset of dyspnea in a patient with a DVT	Classic ECG changes of *cerebral or subarachnoid hemorrhage* usually occur in the precordial leads, and consist of large upright or deeply inverted T waves and a prolonged QT interval. Other changes may include prominent U waves, T wave notching with loss of amplitude, ST segment elevation or depression, or abnormal Q waves mimicking MI. This ECG was obtained in a patient with a ruptured Berry aneurysm and increased intracranial pressure. (Answer: a)
		*Peaked T waves* are defined by T wave amplitude exceeding 6 mm in the limb leads or 10 mm in the precordial leads. Causes include hyperkalemia (more common when rise in serum potassium is acute), acute MI, intracranial bleeding, LVH, RVH, and LBBB. This patient presented with acute oliguria 2° to rhabdomyolysis and a serum K^+ of 8 meq/L. (Answer: d)
		*QRS (electrical) alternans* may be seen in association with pericardial effusion, severe left ventricular failure, hypertension, coronary artery disease, rheumatic heart disease, and supraventricular or ventricular tachycardia. This ECG was obtained from a renal failure patient with uremic pericarditis and cardiac tamponade. (Answer: h)

Differential Diagnosis

Left Axis Deviation (> – 30°)

- Left anterior fascicular block (if axis > – 45°)
- Inferior wall MI
- Left bundle branch block
- Left ventricular hypertrophy
- Ostium primum atrial septal defect
- Chronic lung disease (e.g., emphysema)
- Hyperkalemia

ECG 71. 53-year-old male with palpitations:



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- ☐ 04. Artifact

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- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
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- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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VENTRICULAR RHYTHMS

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- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 58. Inferior (age indeterminate or old)
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- ☐ 80. Dextrocardia, mirror image
- ☐ 81. Chronic lung disease
- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
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- ☐ 86. Central nervous system disorder
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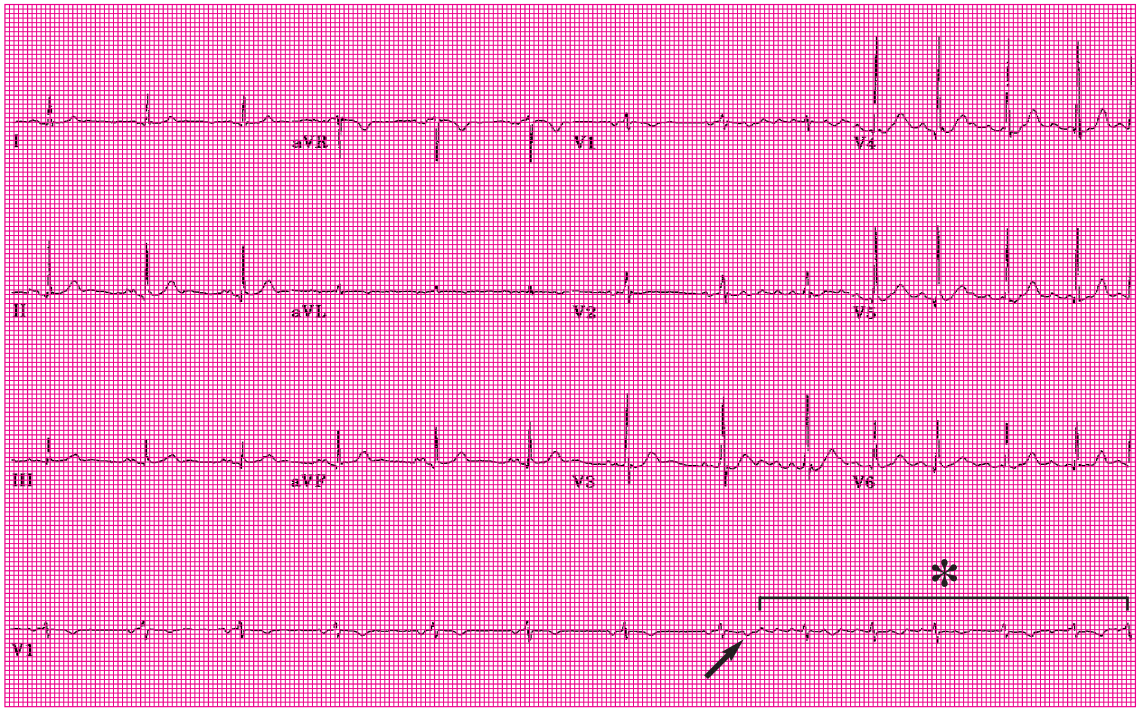
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 71 was obtained from a 53 year-old male with palpitations. The ECG begins with sinus rhythm at a rate of 72 beats/minute. After the 8th sinus beat, there is a nonconducted atrial premature complex (best seen in lead V_1, arrow) that initiates a run of atrial fibrillation (asterisk). Although the rhythm initially resembles atrial flutter, it quickly becomes more chaotic and typical of atrial fibrillation. This ECG emphasizes the importance of reviewing the *entire* rhythm strip.

Codes:

07	Sinus rhythm
13	Atrial premature complexes
19	Atrial fibrillation



Questions: ECG 71

1. In addition to atrial fibrillation, causes of an irregularly irregular rhythm include:

   a. Atrial flutter with variable AV block
   b. AV nodal reentrant tachycardia (AVNRT)
   c. Atrial tachycardia with 2:1 block
   d. Multifocal atrial tachycardia
   e. Accelerated junctional rhythm
   f. Sinus rhythm with frequent APCs

2. Atrial fibrillation with a ventricular response >200 per minute suggests:

   a. Tachy-brady syndrome (sick sinus syndrome)
   b. Concealed bypass tract
   c. Digitalis toxicity
   d. Wolff-Parkinson-White syndrome

Answers: ECG 71

1. Multifocal atrial tachycardia is an irregular rhythm with $\geq$ 3 P wave morphologies, varying PR, RR, and RP intervals, and a 1:1 relationship between P waves and QRS complexes. Sinus rhythm with frequent APCs can resemble MAT, but a dominant sinus P wave is evident. Atrial flutter with variable AV block results in sawtooth flutter waves and an irregularly irregular ventricular response. Atrial tachycardia with 2:1 AV block results in a regular rhythm with 2 P waves for every QRS complex. AVNRT and accelerated junctional rhythm are almost always regular rhythms. (Answer: a, d, f)

2. Patients with Wolff-Parkinson-White (WPW) syndrome are capable of conducting atrial impulses antegrade across their bypass tracts at very rapid rates, resulting in ventricular rates in atrial fibrillation up to 300-350 per minute. Patients with concealed bypass tracts conduct retrograde (not antegrade) across their bypass tracts; ventricular rates in atrial fibrillation are similar to normal patients (100-180 per minute). Ventricular rates > 200 per minute rarely occur in other clinical settings. (Answer: d)

— Quick Review 71 —

Atrial premature complexes	
• P wave is (normal/abnormal) in configuration	abnormal
• QRS complex is (similar/different) in morphology to the QRS complex present during sinus rhythm	similar
• PR interval may be normal, increased, or decreased (true/false)	true
• The post-extrasystolic pause is usually (compensatory/noncompensatory)	noncompensatory
Atrial fibrillation	
• ____ waves are absent	P
• Atrial activity is totally ____ and represented by fibrillatory (f) waves of varying amplitudes, duration and morphology	irregular
• Atrial activity is best seen in the ____ and ____ leads	right precordial, inferior
• Ventricular rhythm is (regularly/irregularly) irregular	irregularly
• ____ toxicity may result in regularization of the RR interval due to complete heart block with junctional tachycardia	Digitalis
• Ventricular rate is usually ____ per minute in the absence of drugs	100-180
▸ Think ____ if the ventricular rate is > 200 per minute and the QRS is > 0.12 seconds	Wolff-Parkinson-White

Common Dilemmas in ECG Interpretation

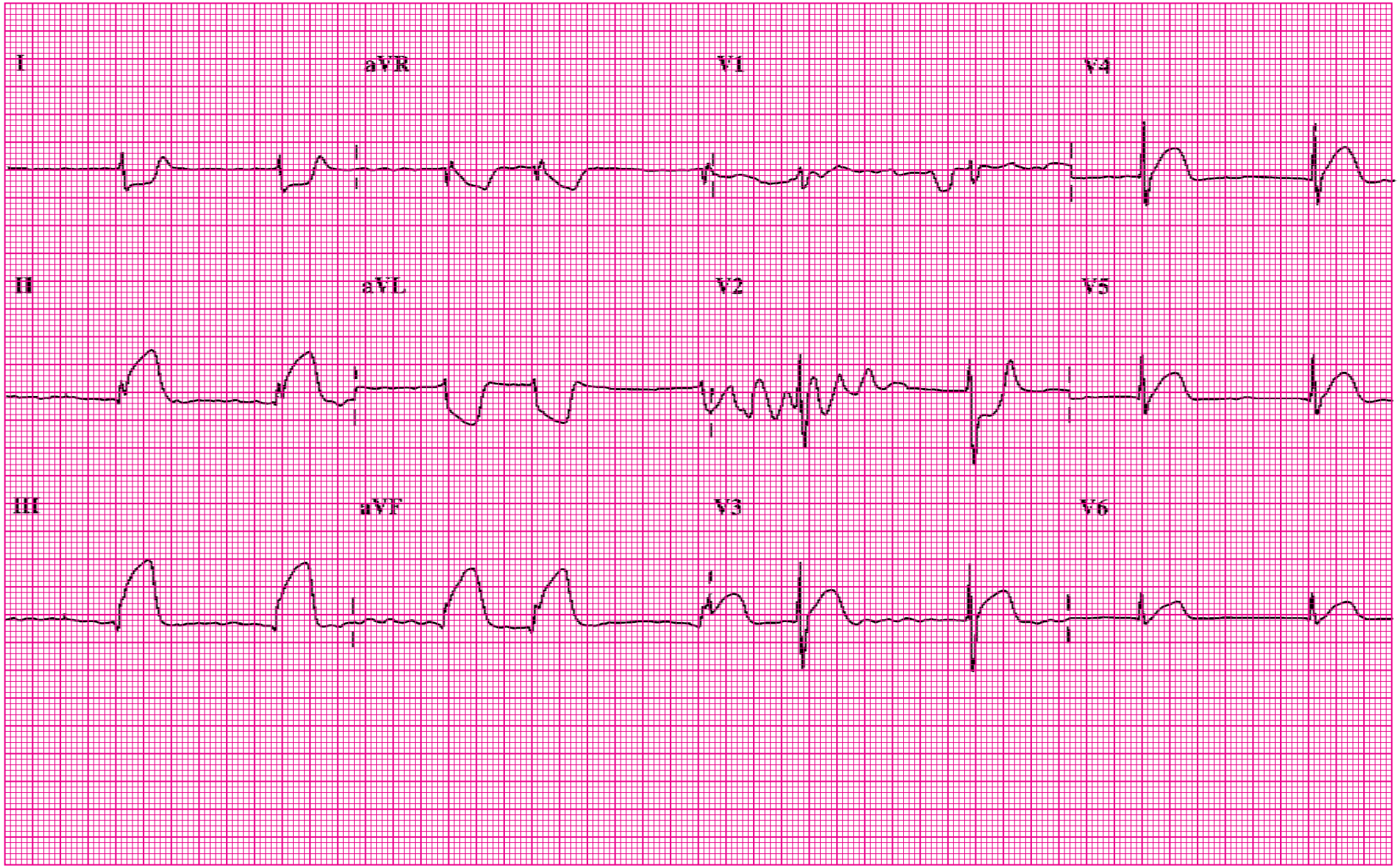
Problem

Atrial fibrillation is present with intermittent episodes of atrial flutter. Should atrial fibrillation or atrial flutter be coded?

Recommendation

Atrial fibrillation should be coded, not atrial flutter. Atrial fibrillation often manifests as "fib/flutter;" however, on formal testing, you must choose one or the other. The best strategy in this setting is to code atrial fibrillation; atrial flutter should be reserved for tracings that show continuous atrial flutter without interspersed episodes of fibrillation.

ECG 72. 52-year-old male with chest pain:



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- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

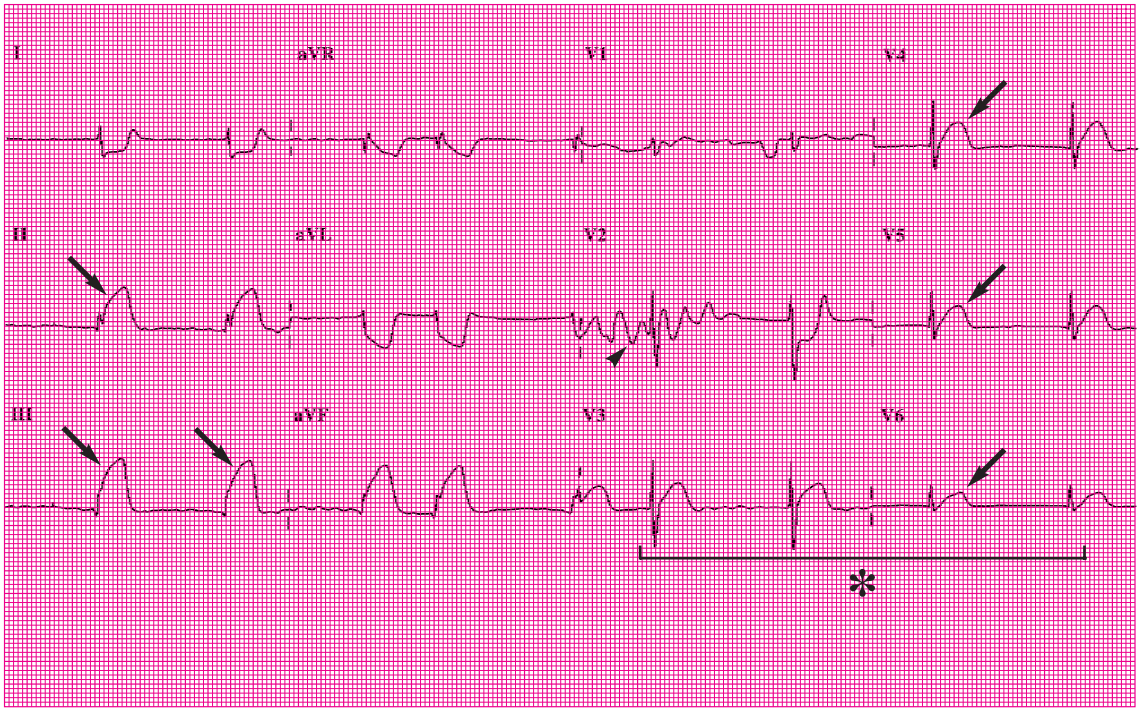
PACED RHYTHMS

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

ECG 72 was obtained from a 52-year-old male with chest pain. The ECG shows atrial fibrillation with marked ST-T wave changes (arrows) consistent with acute myocardial injury to the inferior, posterior, and anterolateral walls (which will likely evolve into an extensive Q wave MI). In addition, ST-T wave changes consistent with myocardial ischemia are evident in the high lateral leads (I and aVL). The coarse baseline fluctuation noted in lead V_2 (arrowhead) is compatible with artifact. Regularization of the atrial fibrillation in the last half of the tracing (asterisk) is consistent with AV block with an AV junctional escape rhythm secondary to digitalis toxicity. Right axis deviation is also present.

Codes:

04	Artifact
19	Atrial fibrillation
21	AV junctional escape complexes
37	Right axis deviation (> +100°)
64	ST and/or T wave abnormalities suggesting myocardial ischemia
65	ST and/or T wave abnormalities suggesting myocardial injury
71	Digitalis toxicity



Questions: ECG 72

1. The most likely cause of a tall R wave in lead V_1 in the setting of inferior myocardial injury is:

   a. Right ventricular hypertrophy
   b. Right bundle branch block
   c. Normal variant
   d. Posterior MI

2. What is the likely age of the myocardial infarction on this ECG:

   a. Hours
   b. Days
   c. Weeks

3. What is responsible for the change in QRS morphology between the 4th and 5th beats in the bottom row of the ECG record:

   a. Fusion beat
   b. Lead change
   c. Artifact
   d. Aberrancy

4. Right axis deviation in this tracing is due to left posterior fascicular block:

   a. True
   b. False

5. Findings in this tracing consistent with hyperkalemia include:

   a. Atrial fibrillation
   b. ST elevation
   c. Tall T waves

6. The most likely cause for the ST depression in I and aVL is:

   a. Ventricular aneurysm
   b. Lateral wall myocardial ischemia
   c. Localized pericarditis
   d. Reciprocal changes secondary to acute myocardial infarction
   e. Digitalis effect

7. What is the cause of the baseline undulations in V_2:

   a. Coarse atrial fibrillation
   b. Flutter waves
   c. Tremor due to Parkinson's disease
   d. Artifact

Answers: ECG 72

1. Posterior MI is the most likely cause of a tall R wave in lead V_1 in the setting of inferior myocardial injury. Due to the loss of posterior QRS forces, unopposed anterior QRS forces manifest a prominent R wave in leads overlying the anterior wall, such as lead V_1 and/or V_2. (Answer: d)

2. The development of Q waves and evolutionary changes in the T wave and ST segment can be used to approximate the age of myocardial infarction:

   - **T waves**: The development of large upright T waves is often the earliest manifestations of acute MI, occurring within minutes and lasting for minutes to hours. T wave inversion, which begins while ST segments are still elevated, may last for months to years, persist indefinitely, or regress to nonspecific T wave changes.

   - **ST segment**: ST elevation usually develops in the minutes to hours following acute MI. Resolution may occur within hours, but usually requires a few days for complete return to baseline. Persistence beyond 4 weeks should raise the suspicion of ventricular aneurysm.

   - **Q waves**: Abnormal Q waves usually develop in the first several hours to days following acute infarction. In most patients, they persist indefinitely; on occasion, Q waves may regress to no longer meet criteria for abnormal Q waves. In fewer than 15% of patients, Q waves disappear entirely.

   In the present ECG, inferior and anterolateral Q waves accompanied by convex upward ST segment elevation suggest the myocardial infarction is acute. (Answer: a)

3. The abnormal QRS morphology seen in the fifth beat in the bottom row of the ECG record is due to lead change midway through the inscription of the QRS morphology. The first half of the QRS complex represents recording from aVF; the second half represents recording from V_3. (Answer: b)

4. Before right axis deviation can be attributed to left posterior fascicular block (LPFB), other causes of right axis deviation must be excluded, including lateral wall MI, right ventricular hypertrophy, and pulmonary emphysema. The presence of lateral MI in this tracing precludes the diagnosis of LPFB, which is a diagnosis of exclusion. (Answer: b)

5. Although this patient is not hyperkalemic, elevated potassium levels can induce ECG changes that mimic acute MI. Findings in this ECG that can also be seen in hyperkalemia include tall T waves and ST elevation. However, the T waves in hyperkalemia are usually peaked and narrow, and ST elevation is usually diffuse and does not show reciprocal ST segment depression, as seen here in leads I and aVL. (Answer: b, c)

6. The ST segment depression in leads I and aVL is most likely due to high lateral wall ischemia, although reciprocal changes are possible. In general, ST depression associated with ST elevation in other leads is a marker for a larger region of jeopardized myocardium. Digitalis may cause ST depression, but is typically diffuse and not confined to two leads as in the present tracing. Pericarditis and ventricular aneurysm cause ST segment elevation, not depression. (Answer: b)

7. The most likely cause of baseline undulation in lead V_2 is artifact, probably due to a loose lead. Coarse atrial fibrillation is unlikely since fine atrial fibrillation is present throughout the rest of the tracing. Variability in the peak-to-peak intervals of the undulations makes atrial flutter unlikely, and the lack of artifact in the limb leads makes tremor due to Parkinson's disease unlikely. (Answer: d)

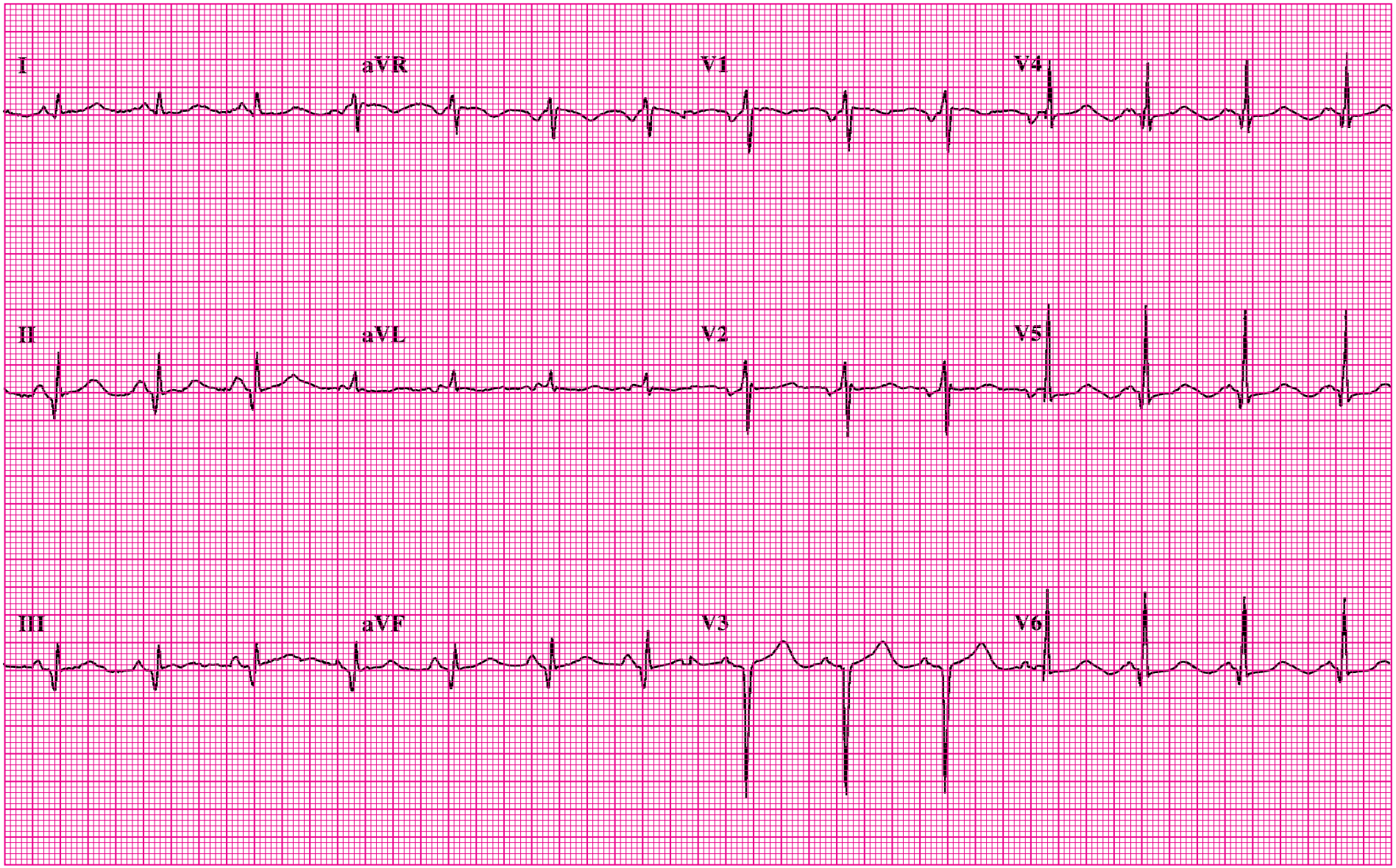
— Quick Review 72 —

Atrial fibrillation	
• ____ waves are absent	P
• Atrial activity is totally ____ and represented by fibrillatory (f) waves of varying amplitudes, duration and morphology	irregular
• Atrial activity is best seen in the ____ and ____ leads	right precordial, inferior
• Ventricular rhythm is (regularly/irregularly) irregular	irregularly
• ____ toxicity may result in regularization of the RR interval due to complete heart block with junctional tachycardia	Digitalis
• Ventricular rate is usually ____ per minute in the absence of drugs	100-180
▸ Think ____ if the ventricular rate is > 200 per minute and the QRS is > 0.12 seconds	Wolff-Parkinson-White

— Quick Review 72 —

AV junctional escape complexes	
• QRS complex occurs as a ____ phenomenon in response to decreased sinus impulse formation or conduction, or high-degree AV block	secondary
• Rate is typically ____ per minute	40-60
• Atrial mechanism may be sinus rhythm, paroxysmal atrial tachycardia, atrial flutter, or atrial fibrillation (true/false)	true
• QRS morphology is (similar to/different from) the sinus or supraventricular impulse	similar to
Posterior MI, recent or probably acute	
• Initial R wave ≥ ____ seconds in leads ____ and ____ with:	0.04, V_1 V_2
▸ R wave amplitude (greater than/less than) S wave amplitude, *and* ST segment (elevation/depression) with (upright/inverted) T waves	greater than depression, upright
• Posterior MI is usually seen in the setting of acute inferior MI (true/false)	true
• RVH, WPW and RBBB (do/do not) interfere with the ECG diagnosis of posterior MI	do
Peaked T waves	
• T wave > ____ mm in the limb leads or > ____ mm in the precordial leads	6, 10

ECG 73. 79-year-old asymptomatic male:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

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- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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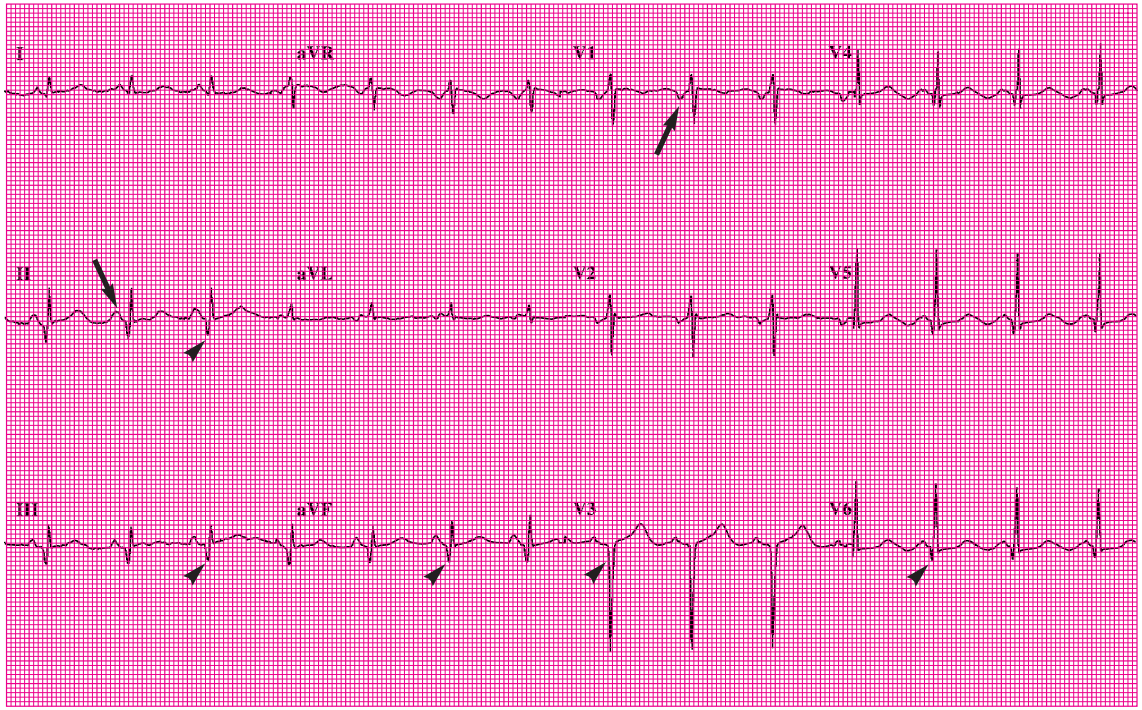
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 73 was obtained from a 79-year-old asymptomatic male. The ECG shows sinus rhythm, left and right atrial abnormalities (arrows), and Q waves meeting criteria for age indeterminate anterior, anterolateral, and inferior myocardial infarctions (arrowheads). Nonspecific ST-T abnormalities are present (the subtle ST segment elevation does not meet 1 mm in the inferior leads and is not diagnostic of aneurysm or acute injury). The corrected QT interval is prolonged (0.48 seconds).

Codes:

05	Right atrial abnormality/enlargement
06	Left atrial abnormality/enlargement
07	Sinus rhythm
52	Anterolateral Q wave MI (age indeterminate or old)
54	Anterior or anteroseptal Q wave MI (age indeterminate or old)
58	Inferior Q wave MI (age indeterminate or old)
63	Nonspecific ST and/or T wave abnormalities
68	Prolonged QT interval



Questions: ECG 73

1. Conditions associated with pathological Q waves that can mimic myocardial infarction include:

   a. Pericarditis
   b. Wolff-Parkinson-White syndrome
   c. Left bundle branch block
   d. COPD
   e. Pneumothorax
   f. Severe right ventricular hypertrophy
   g. Cardiomyopathy
   h. Infiltrative diseases of the myocardium (e.g., tumor, sarcoid)
   i. Pulmonary embolism

2. Drugs that can prolong the QT interval include:

   a. Amiodarone
   b. Sotalol
   c. Disopyramide
   d. Tricyclic antidepressants
   e. Lithium
   f. Procainamide
   g. Quinidine
   h. Phenothiazines

3. The likely age of the MI in the present ECG is:

   a. Minutes to hours
   b. Hours to days
   c. Months to years

4. The presence of Q waves can be used to distinguish transmural from subendocardial myocardial infarction:

   a. True
   b. False

5. The absence of Q waves can be used to distinguish subendocardial from transmural myocardial infarction:

   a. True
   b. False

Answers: ECG 73

1. While abnormal Q waves are most commonly associated with myocardial infarction (MI), several other conditions may produce abnormal Q waves on ECG, including WPW syndrome, left bundle branch block (LBBB), COPD, pneumothorax, cardiomyopathy, pulmonary embolism and others. In the WPW syndrome, negative delta-waves can occur and mimic MI. In left bundle branch block, QS complexes in leads V_1 - V_4 (often accompanied by 1 - 2 mm of ST elevation) can be mistaken for anteroseptal MI. In COPD, Q waves usually occur in the inferior and/or right/mid precordial leads; other findings include poor R wave progression, P pulmonale, low voltage QRS, and $S_1S_2S_3$ pattern. Pneumothorax can cause a loss of R waves in the right precordial leads (QS complex), and along with the presence of symmetrical T wave inversion can mimic anterior MI. In hypertrophic cardiomyopathy, abnormal Q waves are frequently seen in leads I, aVL, V_4 - V_6 due to septal hypertrophy. Abnormal Q waves may also be seen in infiltrative diseases of the myocardium when electrically-active tissue is replaced by fibrous tissue or electrically-inert substances (e.g., amyloid). Finally, Q waves may be seen in lead III and sometimes in aVF in pulmonary embolism, which can be accompanied by ST and T waves changes and confused with acute inferior MI; however, unlike inferior MI, Q waves in lead II are rare. (Answer: All except a, f)

2. Many drugs increase ventricular repolarization to cause prolongation of the QT interval, especially Type IA antiarrhythmics (quinidine, procainamide, disopyramide), sotalol and amiodarone. Significant QT prolongation increases the risk of torsade de pointes, syncope, and sudden cardiac death. (Answer: All)

3. The development of Q waves, and evolutionary changes in the T wave and ST segment can be used to approximate the age of myocardial infarction:

   - **T waves**: The development of large upright T waves is often the earliest manifestations of acute MI, occurring within minutes and lasting for minutes to hours. T wave inversion, which begins while ST segments are still elevated, may last for months to years, persist indefinitely, or regress to nonspecific T wave changes.

   - **ST segment**: ST elevation usually develops in the minutes to hours following acute MI. Resolution may occur within hours, but usually requires a few days for complete return to baseline. Persistence beyond 4 weeks should raise the suspicion of ventricular aneurysm.

   - **Q waves**: Abnormal Q waves usually develop in the first several hours to days following acute infarction. In most patients, they persist indefinitely, but may regress to no longer meet the criteria for abnormal Q waves; in some patients (< 15%), Q waves disappear entirely.

   In the ECG in question, the presence of inferior Q waves accompanied by isoelectric ST segments and upright T waves suggest that the infarction is months or years in age, not acute. (Answer: c)

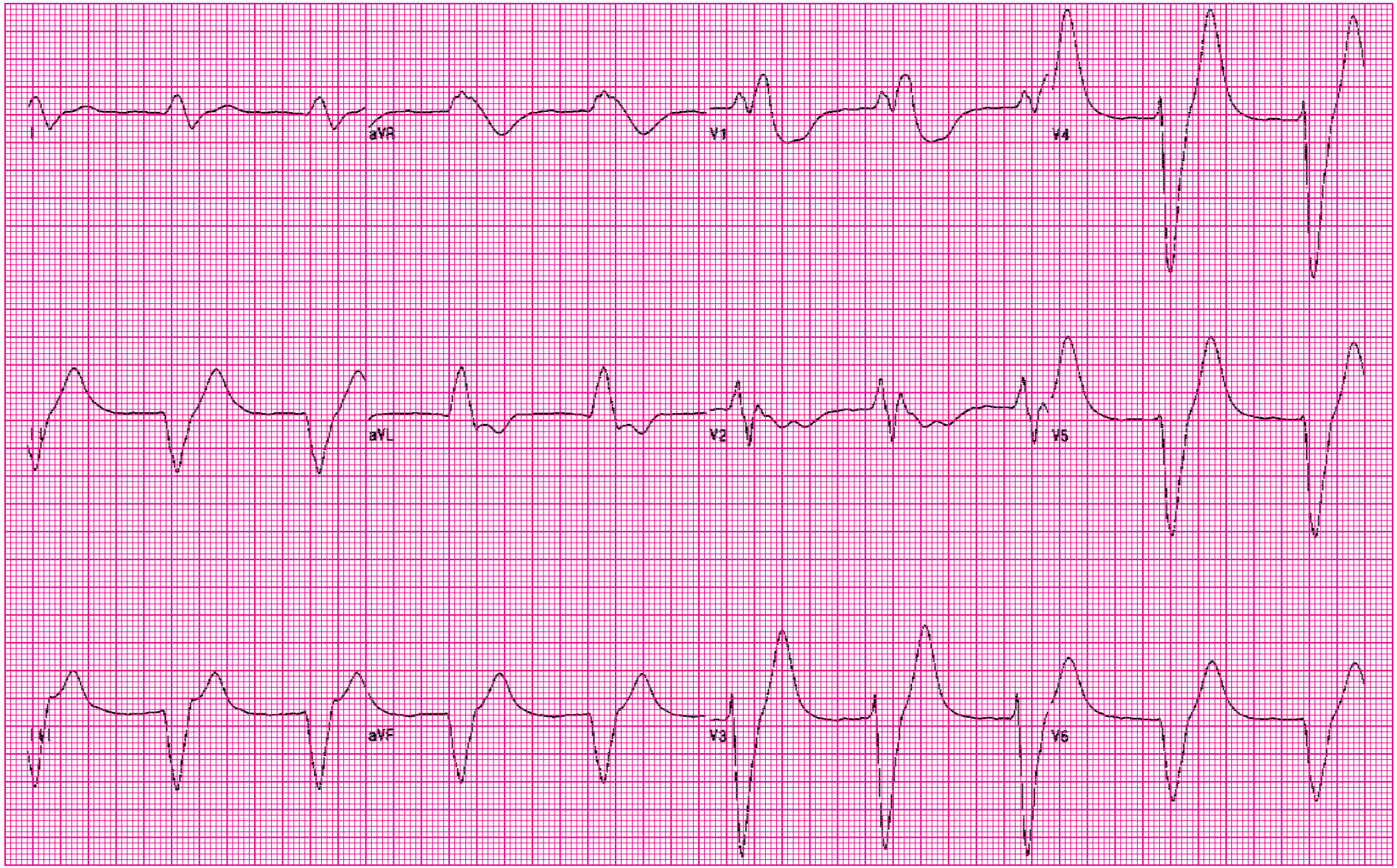
4. Q waves were once thought to be the hallmark of transmural infarction, but pathological studies have confirmed that Q waves can occur in subendocardial infarction as well. The presence of a Q wave cannot be used to reliably distinguish transmural from subendocardial MI. (Answer: b)

5. Non-Q-wave MI can be seen in both transmural infarction (especially when the culprit vessel is the left circumflex coronary artery) and subendocardial infarction. (Answer: b)

— Quick Review 73 —

Anterolateral MI, age indeterminate or probably old	
• Abnormal Q waves (with/without) ST segment elevation in leads ____	without V_4-V_6
Anterior MI, age indeterminate or probably old	
• rS in lead ____, followed by either QS or QR complexes (with/without) ST segment elevation in leads ____ or (increasing/decreasing) R wave amplitude from V_2-V_5	V_1 without, V_2-V_4 decreasing
Inferior MI, age indeterminate or probably old	
• Abnormal Q waves (with/without) ST elevation in at least two of leads ____	without II, III, aVF

ECG 74. 80-year-old unconscious female:



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- ☐ 81. Chronic lung disease
- ☐ 82. Acute cor pulmonale including pulmonary embolus
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- ☐ 85. Hypertrophic cardiomyopathy
- ☐ 86. Central nervous system disorder
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- ☐ 88. Hypothermia
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- ☐ 90. Atrial or coronary sinus pacing
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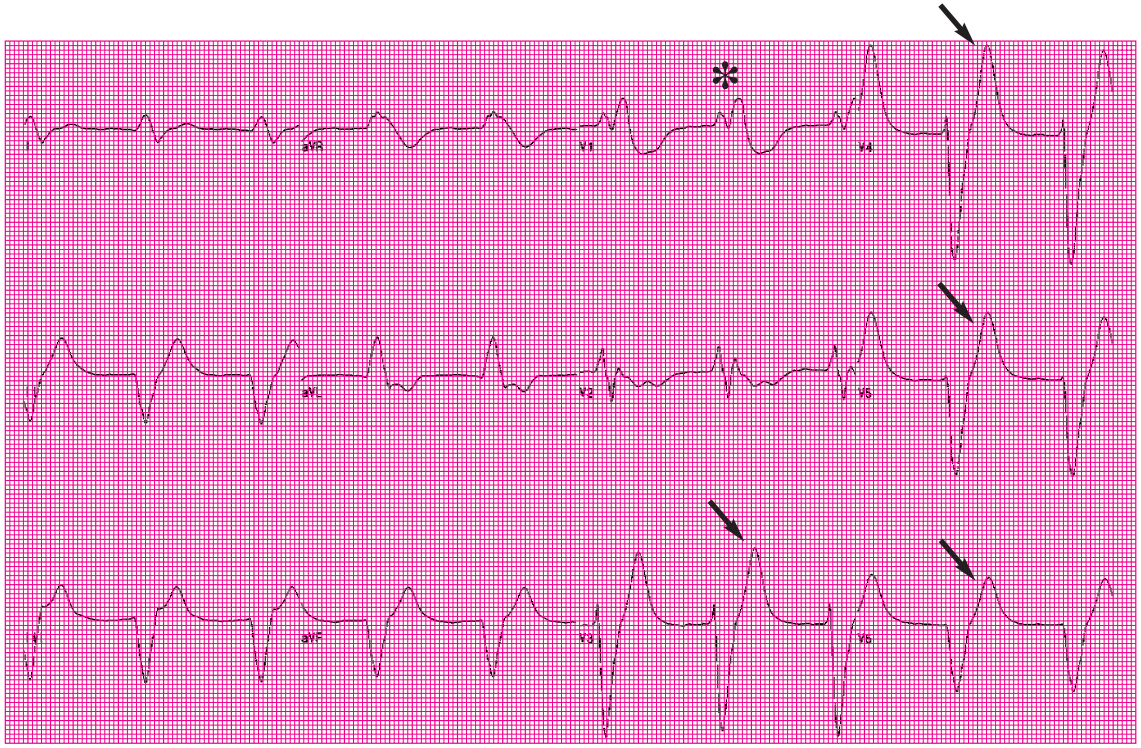
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 74 was obtained from an 80-year-old unconscious female. The ECG shows a regular, wide QRS complex rhythm at a rate of 57 beats/minute with no preceding P waves, consistent with accelerated idioventricular rhythm. The extremely wide QRS complexes (0.24 seconds in lead V_1) (asterisk) have an early sine-wave-like appearance, suggestive of hyperkalemia. The tall T waves in leads V_3-V_6 (arrows) are also consistent with hyperkalemia. Neither bundle branch block nor myocardial infarction should be coded in the setting of idioventricular rhythm. The patient was found to have a serum K^+ level of 8.5 mmol/L.

Codes:

26	Accelerated idioventricular rhythm
74	Hyperkalemia



Questions: ECG 74

1. Hyperkalemia is associated with all of the following ECG findings except:

   a. First-degree AV block
   b. Left anterior fascicular block
   c. Prolonged QT interval
   d. Sinus arrest
   e. Tall peaked T waves
   f. Intraventricular conduction disturbance (IVCD)

Answers: ECG 74

1. Hyperkalemia results in significant slowing of atrial, AV nodal, and ventricular conduction, manifesting as sinus arrest, first-degree AV block, IVCD, bundle branch block and/or fascicular block. Tall peaked T waves, flattening of the P wave, idioventricular rhythm, ventricular tachycardia, and ventricular fibrillation may also occur. Hyperkalemia increases the speed of ventricular repolarization, resulting in *shortening* of the QT interval. (Answer: c)

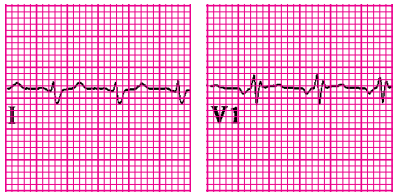
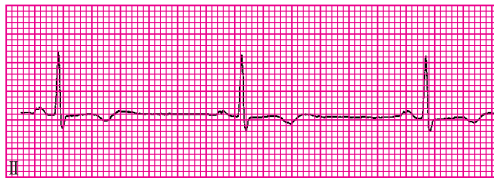
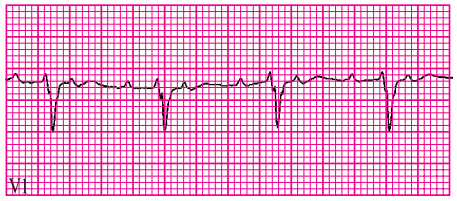
— Quick Review 74 —

Accelerated idioventricular rhythm	
• Highly irregular ventricular rhythm (true/false)	false
• Ventricular rate of ____ per minute	60-110
• QRS morphology is similar to ____	VPCs
• Ventricular ____ complexes, ____ beats, and AV ____ are common	capture, fusion dissociation
Hyperkalemia	
• ***K^+ = 5.5 - 6.5 mEq/L***	
▸ Tall, peaked, narrow based ____ waves	T
▸ QT interval (shortening/lengthening)	shortening
▸ (Reversible/irreversible) left anterior or posterior fascicular block	Reversible
• ***K^+ = 6.5 - 7.5 mEq/L***	
▸ ____ degree AV block	First
▸ Flattening and widening of the ____ wave	P
▸ ST segment (depression/elevation)	depression
▸ ____widening	QRS
• ***K^+ > 7.5 mEq/L***	
▸ Disappearance of ____ waves	P
▸ LBBB, RBBB, or markedly widened and diffuse intraventricular conduction delay resembling a _____ wave pattern	sine
▸ Arrhythmias and conduction disturbances including VT, VF, idioventricular rhythm, asystole (true/false)	true

— POP QUIZ —

Pattern Recognition: ECG/Clinical Correlation

Instructions: Match the ECG with the most likely clinical presentation.

ECG	Choose Single Best Answer	Answer
 Normal ECG 2 weeks earlier	a. Acute hemiparesis, papilledema b. Dyspnea, constipation, impaired memory, fatigue c. Red-green color blinders, nausea, vomiting d. Acute oliguria 2° to rhabdomyolysis e. Murmur in a Down's Syndrome patient f. Acute exacerbation of chronic bronchitis g. Prolonged exposure to extreme cold h. Dyspnea and pulses paradoxus in a renal failure patient i. Acute onset of dyspnea in a patient with a DVT	*Large pulmonary emboli* cause elevated pulmonary artery pressures, right ventricular dilation/strain, and clockwise rotation of the heart. Associated ECG changes include S_1Q_3 or $S_1Q_3T_3$ (occurs in 30% and lasts for 1-2 weeks), incomplete or complete right bundle branch block (occurs in 25% and lasts < 1 week), and inverted T waves in the right precordial leads (from right ventricular strain; can last for months). Other ECG findings include right axis deviation, nonspecific ST and T wave changes, and P pulmonale. Arrhythmias and conduction disturbances include sinus tachycardia (most common), atrial fibrillation, atrial flutter, atrial tachycardia, and first-degree AV block. (Answer: i)
		*Sinus bradycardia* results in sinus P waves at a rate < 60 per minute. Causes include high vagal tone (normals [especially during sleep], trained athletes, Bezold-Jarisch reflex, pulmonary embolism), myocardial infarction (usually inferior), drugs, hypothyroidism, hypothermia, obstructive jaundice, hyperkalemia, increased intracranial pressure, and sick sinus syndrome. This ECG was obtained in a patient with dyspnea, constipation, impaired memory, and fatigue secondary to hypothyroidism. (Answer: b)
		*Atrial tachycardia with block* results in nonsinus P waves, regular atrial rate (usually 150-240 per minute), isoelectric intervals between P waves (in contrast to atrial flutter), and nonconducted P waves (from second-degree AV block). Digoxin toxicity is responsible for 75% of cases and organic heart disease for 25%. This ECG was obtained in a patient who developed red-green color blindness and GI complaints from digitalis toxicity. (Answer: c)

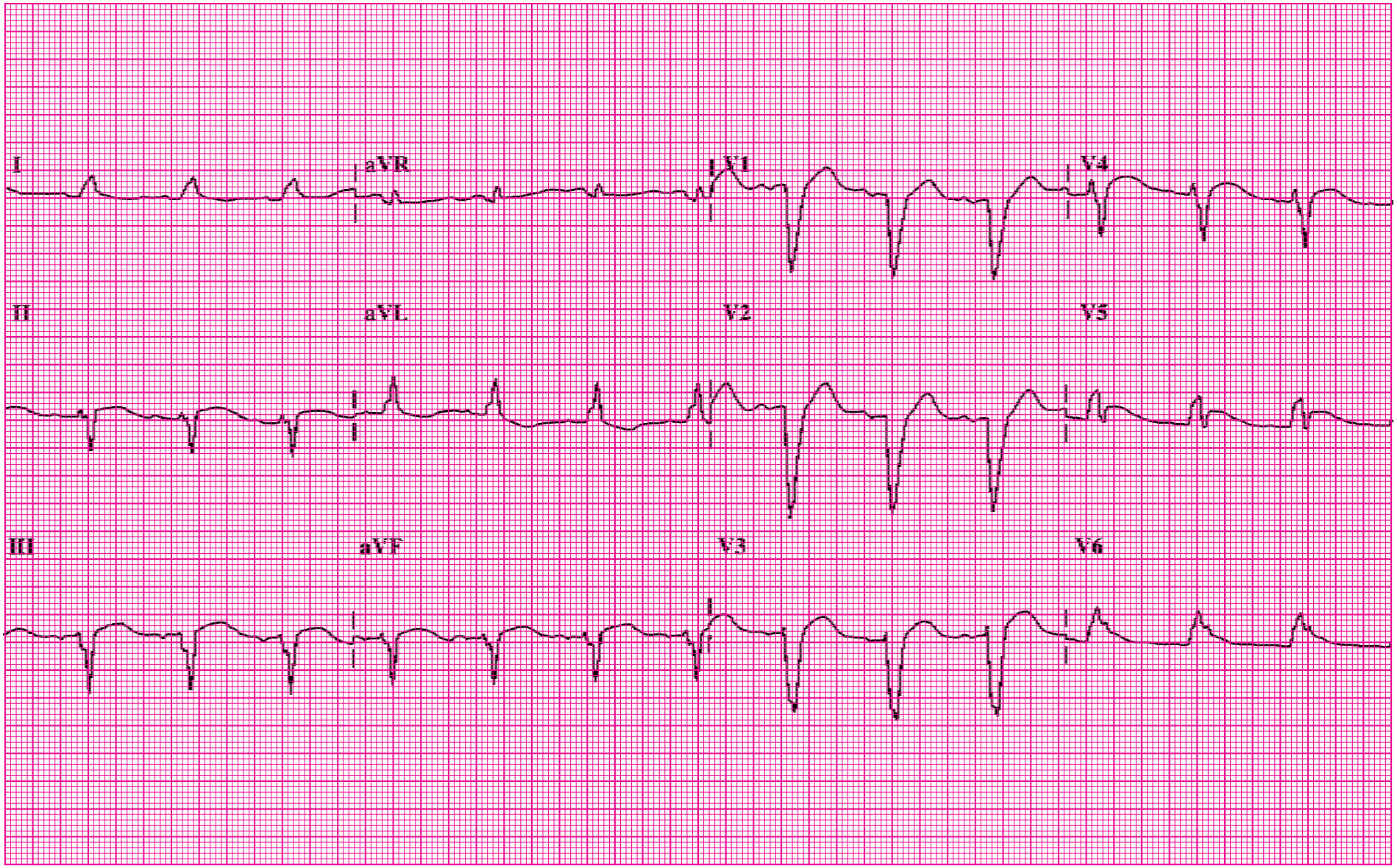
— POP QUIZ —

2:1 AV Block: Mobitz Type I or II

Instructions: Decide if the ECG features listed below favor Mobitz Type I (Wenkebach) or Mobitz Type II second-degree AV block.

ECG Feature	Mobitz Type I or II
2:1 block develops during inferior MI	Type I
Type I on another part of ECG	Type I
History of syncope	Type II
Narrow QRS complex	Type I
AV block worsens in response to maneuvers that increase heart rate & AV conduction (e.g., atropine, exercise)	Type II
AV block worsens in response to maneuvers that reduce heart rate & AV conduction (e.g., carotid sinus massage)	Type I

ECG 75. 76-year-old female with severe substernal chest pressure:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

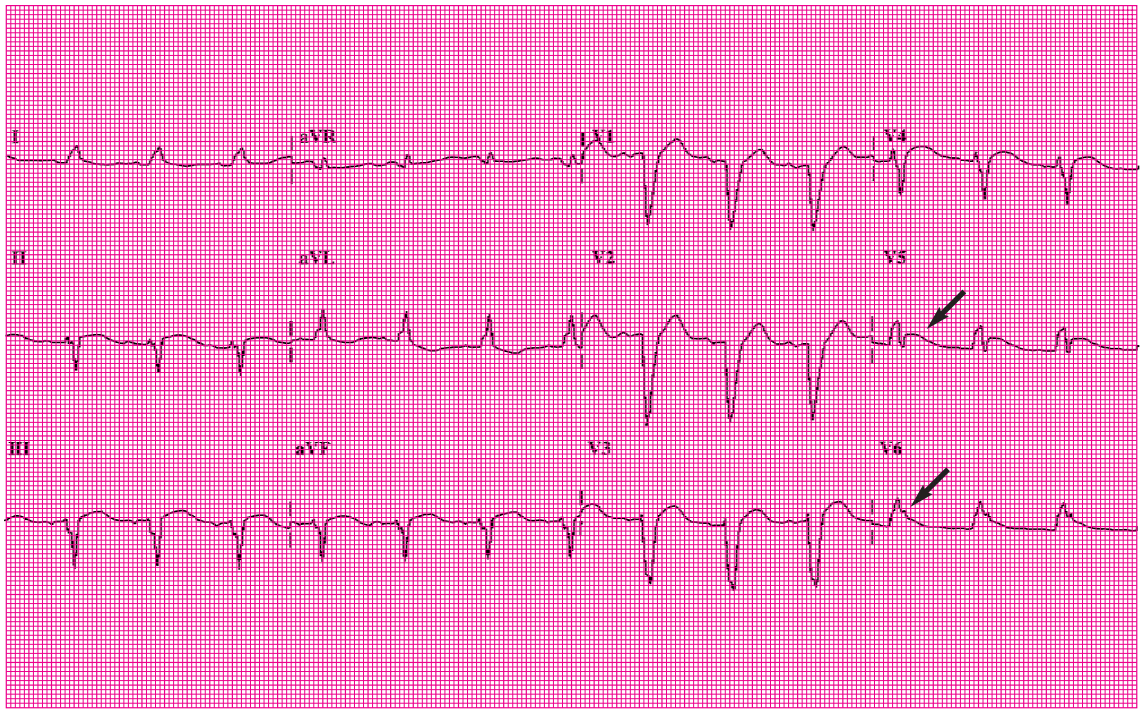
PACED RHYTHMS

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

ECG 75 was obtained in a 76-year-old female with severe substernal chest pressure, diaphoresis, and pallor. The ECG shows sinus rhythm, left bundle branch block, and left axis deviation. Concordant ST segment elevation is apparent in leads V_5 and V_6 (arrows), consistent with acute myocardial injury. However, since pathological Q waves are not present, Q wave myocardial infarction should not be coded.

Codes:

07	Sinus rhythm
36	Left axis deviation (> -30°)
47	LBBB, complete
65	ST and/or T wave abnormalities suggesting myocardial injury



Questions: ECG 75

1. The most specific ECG finding for acute myocardial injury in the setting of LBBB is:

   a. ST segment elevation > 1 mm opposite in direction (discordant) to the major QRS deflection
   b. Q waves in leads V_1-V_3
   c. Concordant ST segment depression
   d. ST segment elevation in the same direction (concordant) as the major QRS deflection

2. Left bundle branch block (LBBB):

   a. Interferes with the ECG diagnosis of RVH
   b. Interferes with the ECG diagnosis of LVH
   c. Does not interfere with the ECG diagnosis of myocardial infarction

Answers: ECG 75

1. Acute myocardial infarction is very difficult to diagnosis in the setting of LBBB, and the usual criteria do not apply. Q waves are often present in the anteroseptal leads and cannot be considered pathological. ST and T wave changes opposite in direction to the major QRS complex are secondary to LBBB, and lack specificity for acute ischemia. Concordant ST segment elevation > 1 mm is an unusual finding in LBBB, and is generally considered to be a sign of acute myocardial injury. (Answer: d)

2. Left bundle branch block (LBBB) interferes with the ECG diagnosis of right and left ventricular hypertrophy and myocardial infarction. Since more than 80% of patients with LBBB have increased LV mass on echo, for practical purposes, LBBB can be considered a marker for LVH. However, LVH should not be coded unless voltage criteria are present.
   (Answer: a, b)

— Quick Review 75 —

LBBB, complete with ST-T waves suggestive of acute myocardial injury or infarction	
• ST elevation ≥ ____ mm concordant to (same direction as) the major deflection of the QRS	1
• ST depression ≥ ____ mm in V_1, V_2, or V_3	1
• ST elevation ≥ ____ mm discordant with (opposite direction to) the major deflection of the QRS	5
ST and/or T wave changes suggesting myocardial injury	
• Acute ST segment (elevation/depression) with upward (convexity/concavity) in the leads representing the area of infarction	elevation convexity
• T waves invert (before/after) ST segments return to baseline	before
• Associated ST (elevation/depression) in the noninfarct leads is common	depression
• Acute ____ wall injury often has horizontal or downsloping ST segment depression with upright T waves in V_1-V_3, with or without a prominent R wave in these same leads	posterior

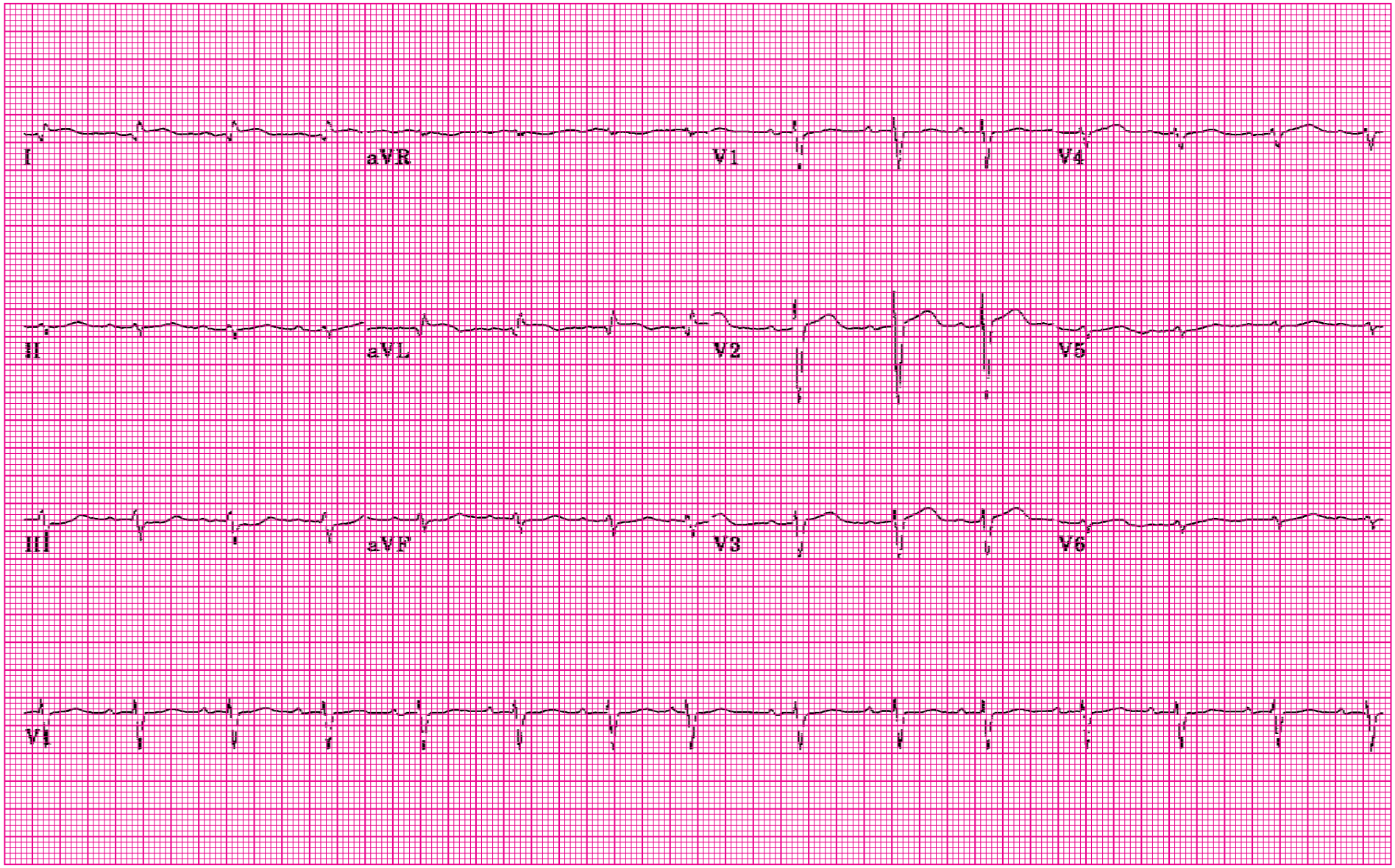
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the clinical disorder that best corresponds to the ECG features listed below (see answer sheet for options).

ECG Features	Answer
• Abnormally tall, symmetrical, inverted T waves • Horizontal or downsloping ST segments with or without T wave inversion	ST-T changes of myocardial ischemia
• Elevated take-off of the ST segment at the J junction • Concave upward ST elevation ending with a symmetrical upright T wave, which is often of large amplitude • Distinct notch or slur on downstroke of R wave • Most commonly involves leads V_2-V_5	Normal variant, early repolarization
• Persistently negative T waves, which are usually not symmetrical or deep, in leads V_1-V_3 in normal adults • Upright T waves in leads I, II, V_5, V_6 • Most frequently seen in young healthy females	Normal variant, juvenile T waves
• Acute ST segment elevation with upward convexity in the leads representing the area of infarction • T waves invert before ST segments return to baseline	ST-T changes of myocardial injury

ECG 76. 65-year-old male with chest pain:



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- ☐ 82. Acute cor pulmonale including pulmonary embolus
- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
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- ☐ 86. Central nervous system disorder
- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

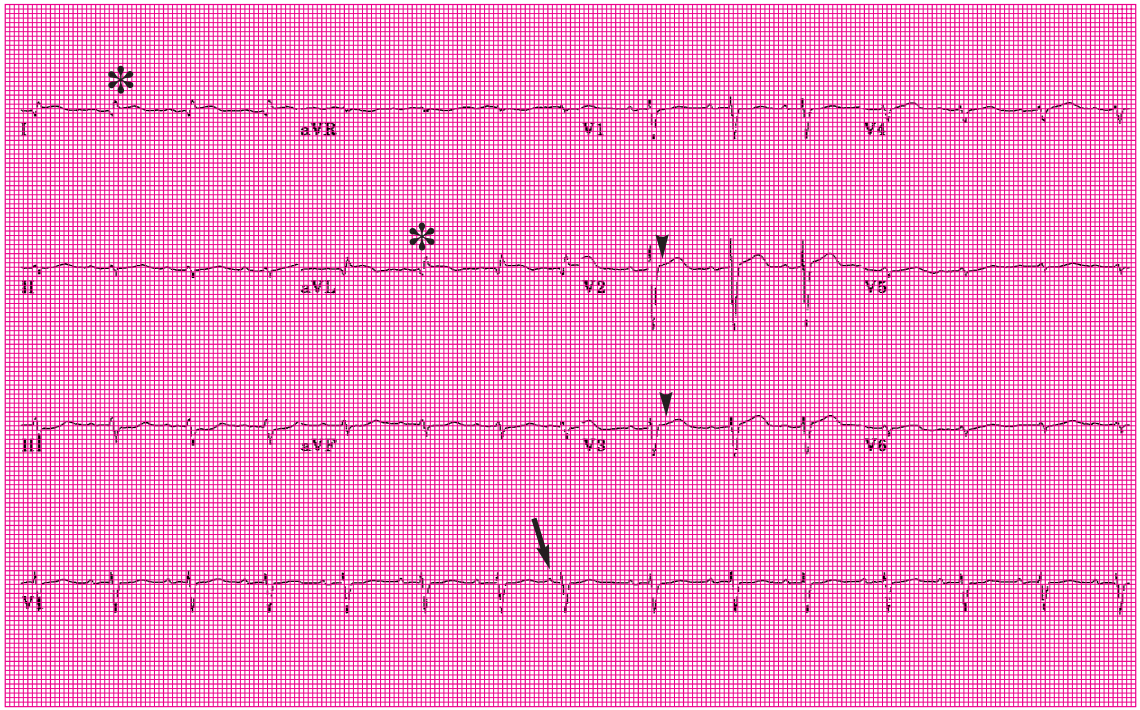
PACED RHYTHMS

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

ECG 76 was obtained from a 65-year-old male with chest pain. The ECG shows sinus rhythm at a rate of 87 beats/minute. The 8th beat (arrow) is an atrial premature complex; the short PR interval (0.12 seconds) indicates the APC originated relatively close to the AV node. There is slight irregularity of the rhythm, but sinus arrhythmia should not be coded since the PP intervals vary by < 0.16 seconds. There are abnormal Q waves and ST segment elevation leads I and aVL (asterisks), consistent with recent or acute lateral myocardial infarction. The loss of R wave amplitude in the left lateral leads is due to the infarct. There is subtle ST elevation in V_2-V_3 (arrowheads) which, given the clinical context, likely represents anterior myocardial injury. Prolonged QT interval (QTc = 0.47 seconds) is most evident in leads II and III.

Codes:

07	Sinus rhythm
13	Atrial premature complexes
55	Lateral Q wave MI (age recent or acute)
65	ST and/or T wave abnormalities suggesting myocardial injury
68	Prolonged QT interval



Questions: ECG 76

1. Clinical conditions associated with abnormal Q waves include:

   a. Primary and metastatic tumors of the heart
   b. Scleroderma of the heart
   c. Muscular dystrophy
   d. Amyloid heart
   e. Hypertrophic obstructive cardiomyopathy
   f. Myocardial contusion
   g. Mitral valve prolapse

Answers: ECG 76

1. Patients with hypertrophic cardiomyopathy often demonstrate abnormal (> 0.04 seconds in duration) Q waves in leads I, aVL, and V_4 - V_6, reflecting exaggerated septal Q waves from marked septal hypertrophy. Abnormal Q waves are also seen in conditions where electrically active tissue is replaced by fibrous tissue or electrically inert substances, as in muscular dystrophy, scleroderma, amyloid, or primary/metastatic tumors of the heart. Abnormal Q waves can also be seen in areas of intramyocardial hemorrhage and edema following myocardial contusion (inconjunction with nonspecific ST and T wave changes and various degrees of heart block if the conduction system is involved). Mitral valve prolapse has rarely been associated with abnormal Q waves in leads III and aVF. Other causes of abnormal Q waves include left bundle branch block, left anterior fascicular block, left and right ventricular hypertrophy, and dilated cardiomyopathy. The "Q" waves in WPW syndrome are actually negative delta waves. (Answer: all)

— Quick Review 76 —

Lateral or high lateral MI (age recent or probably acute)

- Abnormal Q waves and ST elevation in leads I and _____ — aVL
- An isolated Q wave in aVL (does/does not) qualify as a lateral MI — does not

ST and/or T wave changes suggesting myocardial injury

- Acute ST segment (elevation/depression) with upward (convexity/concavity) in the leads representing the area of infarction — elevation, convexity
- T waves invert (before/after) ST segments return to baseline — before
- Associated ST (elevation/depression) in the noninfarct leads is common — depression
- Acute _____ wall injury often has horizontal or downsloping ST segment depression with upright T waves in V_1-V_3, with or without a prominent R wave in these same leads — posterior

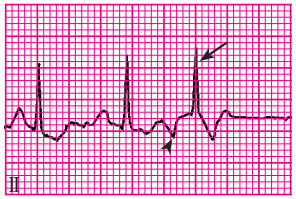
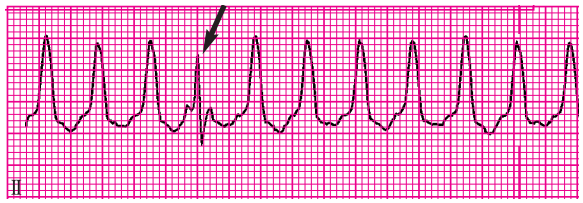
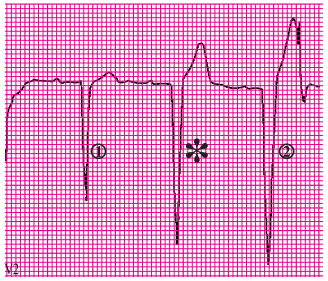
Prolonged QT interval

- Corrected QT interval (QTc) ≥ _____ seconds, where QTc = QT interval divided by the square root of the preceding _____ interval — 0.44, RR
- QT interval varies (directly/inversely) with heart rate — inversely
- The normal QT interval should be (less than/greater than) 50% of the RR interval when the ventricular rate is between 65–90. — less than

— POP QUIZ —

Pattern Recognition: A-V Interactions

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Fusion complex b. Can be seen with ventricular tachycardia c. Results from simultaneous activation of ventricle from 2 different sites of origin d. Echo beat e. Form of nonsustained reentry f. Capture complex g. Suggest diagnosis of SVT in setting of wide QRS tachycardia h. Occurs when atrial impulse stimulates the ventricle during VT i. Atrial and ventricular rhythms occur independant of each other j. AV dissociation k. Ventriculophasic sinus arrhythmia	*Reciprocal (echo) complex* is a form of nonsustained reentry that occurs when an electrical impulse activates a chamber (atria or ventricle), and then returns to the site of origin to reactivate the same chamber again. In the present ECG, an ectopic atrial impulse (inverted P wave; arrowhead) triggers a QRS complex (arrow), and then returns in a retrograde fashion to reactivate the atria (negative P wave immediately following the QRS complex). (Answer: d, e)
		*Ventricular capture complex* occurs when an atrial impulse stimulates the ventricle during ventricular tachycardia. The "captured" ventricle results in a QRS complex similar to that during sinus rhythm (narrow QRS in this ECG). The presence of a ventricular capture complex in the setting of a wide QRS tachycardia strongly suggests the diagnosis of ventricular tachycardia. (Answer: b, f, h)
		*Fusion complex* result from simultaneous activation of the ventricle from 2 sites of origin, resulting in a QRS complex intermediate in morphology between the QRS complexes of each source. Can be seen with ventricular premature complexes, ventricular tachycardia, ventricular parasystole, accelerated idioventricular rhythm, Wolff-Parkinson-White Syndrome, and paced rhythms. (Answer: a, b, c, i, j)

Common Dilemmas in ECG Interpretation

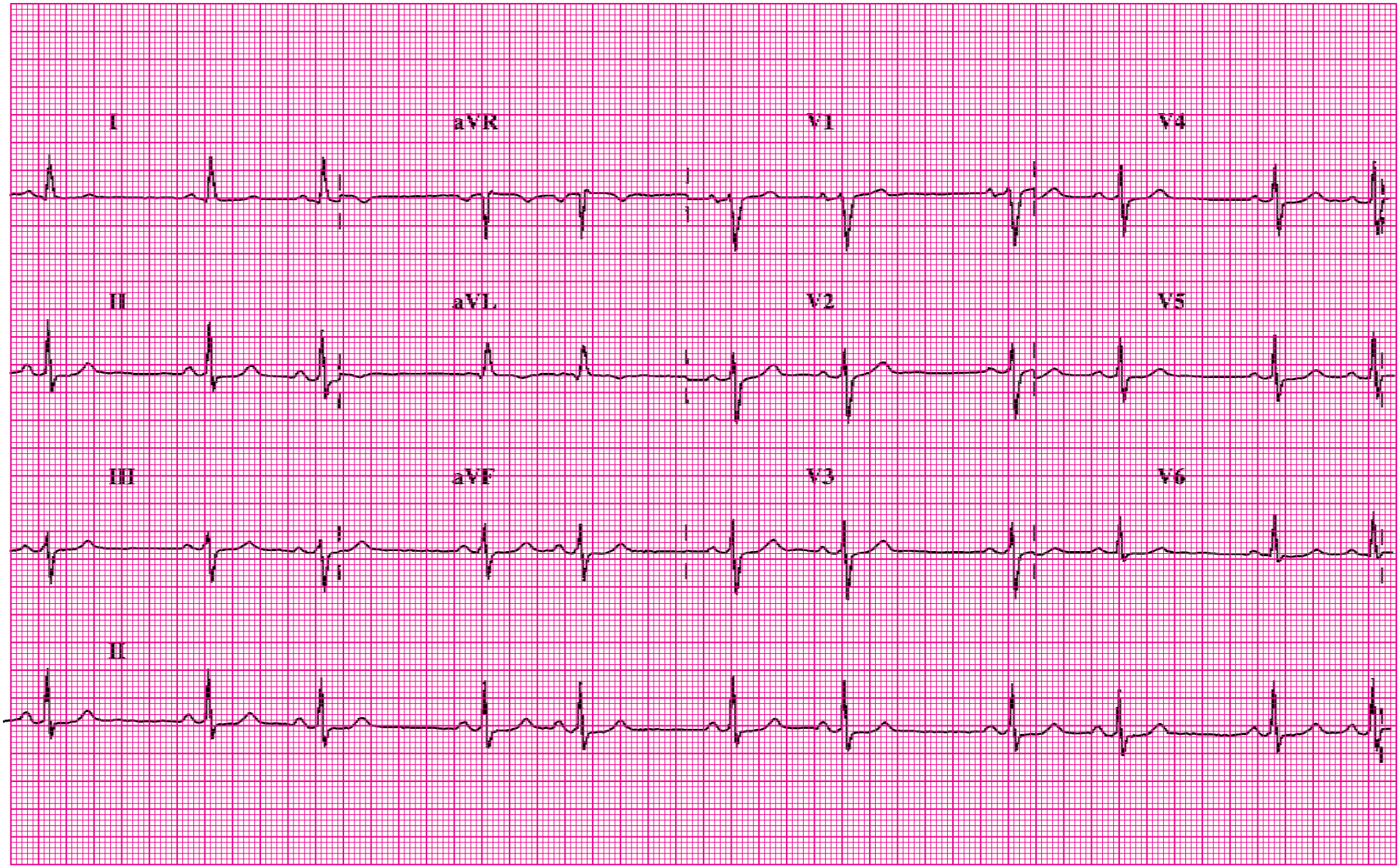
Problem

With so many different criteria for the diagnosis of LVH, which should be used as the "gold-standard?"

Recommendation

The Cornell criteria (R wave in aVL + S wave in V_3 > 28 mm in males and > 20 mm in females) is probably the most accurate of the voltage criteria. However, many ECGs meet voltage criteria in one area of the tracing but not in the others. Therefore, the best policy is know most or all of the various criteria used to diagnose LVH. Remember to code item 67 (ST and/or T wave abnormalities secondary to hypertrophy) in addition to item 40 (left ventricular hypertrophy) when a "strain" pattern is associated with LVH.

ECG 77. 72-year-old diabetic male with hypertension:



GENERAL FEATURES

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SUPRAVENTRICULAR RHYTHMS

- ☐ 07. Sinus rhythm
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- ☐ 12. Sinoatrial exit block
- ☐ 13. Atrial premature complexes
- ☐ 14. Atrial parasystole
- ☐ 15. Atrial tachycardia
- ☐ 16. Atrial tachycardia, multifocal
- ☐ 17. Supraventricular tachycardia, paroxysmal
- ☐ 18. Atrial flutter
- ☐ 19. Atrial fibrillation

JUNCTIONAL RHYTHMS

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

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- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
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- ☐ 31. AV block , 2°-Mobitz type II
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- ☐ 37. Right axis deviation (> +100°)
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- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
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Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
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- ☐ 86. Central nervous system disorder
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- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

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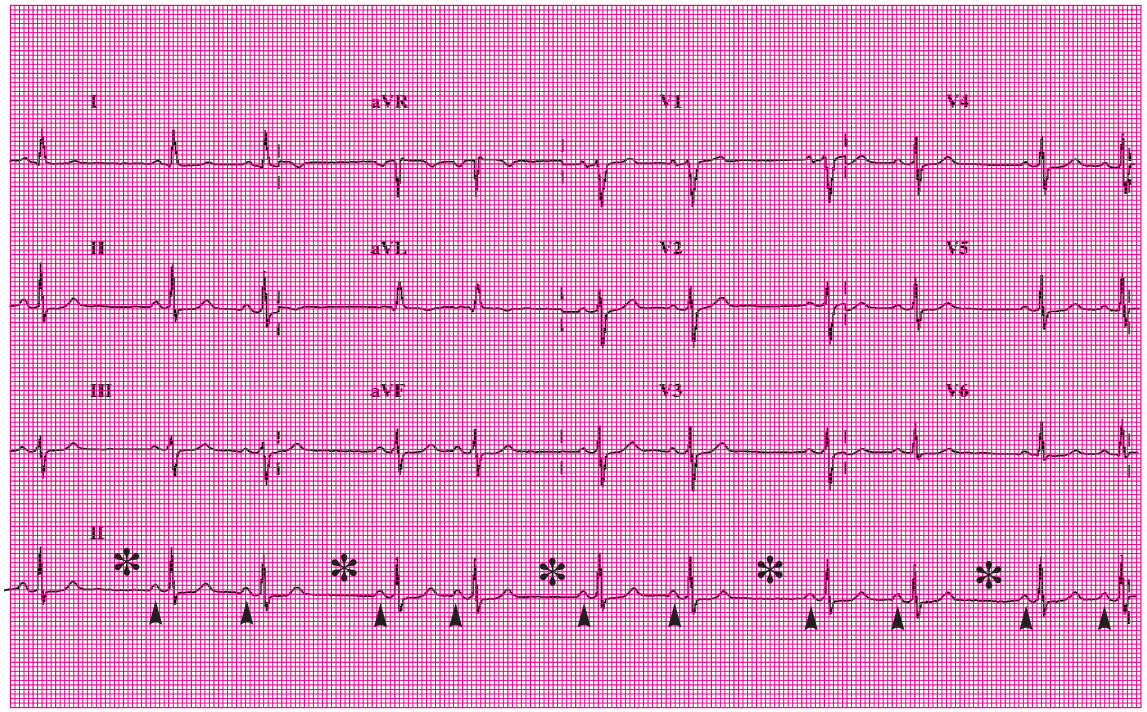
- ☐ 90. Atrial or coronary sinus pacing
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 77 was obtained in a 72-year-old male with hypertension and diabetes. The ECG shows sinus rhythm at approximately 75 beats/minute with "grouped beating." The recurring sequence throughout the tracing consists of two normally conducted P waves (which all have the same morphology; arrowheads mark the P waves) followed by a pause (asterisks) that is somewhat less than two times the usual PP interval. These findings are consistent with the diagnosis of 3:2 sinoatrial exit block (a manifestation of sick sinus syndrome). Sinus arrhythmia should also be coded.

Codes:

07	Sinus rhythm
08	Sinus arrhythmia
12	Sinoatrial exit block
89	Sick sinus syndrome



Questions: ECG 77

1. ECG features of Mobitz Type I sinoatrial exit block include:

   a. Constant PR interval
   b. Group beating
   c. Shortening of the PP interval
   d. PP pause less than two times the normal PP interval

2. Mobitz Type II SA exit block results in a PP pause that is ___ times the usual PP interval:

   a. 2
   b. 3
   c. 4
   d. Any of the above

Answers: ECG 77

1. Mobitz Type I sinoatrial exit block results in intermittent failure of the sinus impulse to capture the atria, resulting in a pause without a P wave. Additional ECG manifestations include shortening of the PP interval leading up to the pause, group beating, a PP pause less than two times the normal PP interval, and a constant PR interval. (Answer: all)

2. Mobitz Type II sinoatrial exit block results in a PP pause that is a multiple of the usual PP interval. PP pauses that are 2, 3, or 4 times the basic PP interval are often due to Mobitz Type II sinoatrial exit block. (Answer: d)

— Quick Review 77 —

Sinus arrhythmia	
• (Sinus/nonsinus) P wave	Sinus
• Longest and shortest PP intervals vary by > ___ seconds or 10%	0.16
• Sinus arrhythmia differs from "ventriculophasic" sinus arrhythmia, the latter of which occurs in the setting of ____	heart block
Sinoatrial (SA) exit block	
First-degree: Conduction of sinus impulses to the atrium is (normal/delayed), but ____:1 response is maintained	delayed, 1
• First-degree SA exit block (is/is not) detectable on the surface ECG	is not
Second-degree: Some sinus impulses fail to ____ the atria	capture
• Type I (Mobitz I):	
▸ Sinus P wave (true/false)	true
▸ "____ beating" with:	Group
(1) (Shortening/lengthening) of the PP interval prior to absent P wave	Shortening
(2) (Constant/variable) PR interval	Constant
(3) PP pause < ____ normal PP interval	2
• Type II (Mobitz II): Constant PP interval followed by a pause that (is/is not) a multiple (2x, 3x, etc.) of the normal PP interval	is
Third-degree:	
• Complete failure of ____ conduction	sinoatrial
• Cannot be differentiated from ____	complete sinus arrest

— POP QUIZ —

Make The Diagnosis

Instructions: Determine the clinical disorder that best corresponds to the ECG features listed below (see items 70-89 on answer sheet for options).

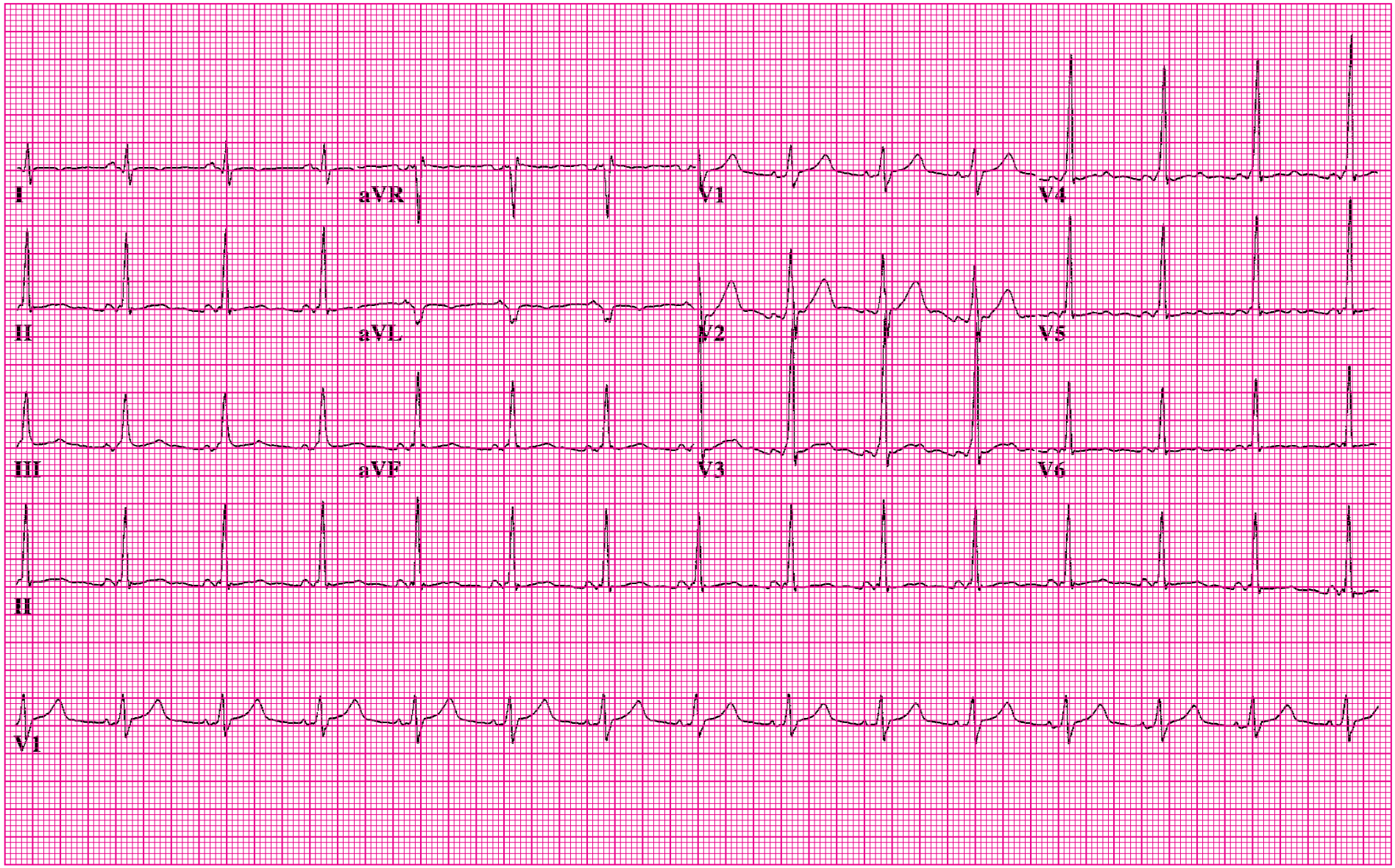
ECG Features	Diagnosis
• RSR' complex in lead V_1 • Left axis deviation • First-degree AV block in 15-40% • Advanced cases have biventricular hypertrophy	Atrial septal defect, primum
• Typical RSR' or rSR' complex in lead V_1 with a QRS duration < 0.11 seconds • Incomplete RBBB • Right axis deviation ± right ventricular hypertrophy • Right atrial abnormality in ~ 30% • First-degree AV block in < 20%	Atrial septal defect, secundum
• P-QRS-T in leads I and aVL are inverted or "upside down" • Decreasing R wave amplitude from leads V_1-V_6	Dextrocardia
• Right ventricular hypertrophy • Right axis deviation • Right atrial abnormality • Shift of transitional zone counterclockwise • Low voltage QRS • Pseudoinfarct pattern in the anteroseptal leads • S_1 S_2 S_3 pattern • May also see sinus tachycardia, junctional rhythm, various degrees of AV block, IVCD, and bundle branch block	Chronic lung disease

Differential Diagnosis

PP Pause Greater Than 1.6-2.0 Seconds

- Sinus pause/arrest: Due to transient failure of impulse formation at the SA node; sinus rhythm resumes at a PP interval that is not a multiple of the basic sinus PP interval
- Sinus arrhythmia: Phasic change in PP interval in response to breath cycle
- Second-degree sinoatrial exit block, Mobitz I (Wenckebach): Progressive shortening of PP interval until a P wave fails to appear
- Second-degree sinoatrial exit block, Mobitz II: Resumption of sinus rhythm at a PP interval that is a multiple (e.g., 2x, 3x, etc.) of the basic sinus rhythm
- Third-degree sinoatrial exit block: Complete failure of sinoatrial conduction; cannot be differentiated from complete sinus arrest on surface ECG
- Abrupt change in autonomic tone (e.g., vagal reaction)
- "Pseudo" sinus pause due to nonconducted APCs: P wave appears to be absent but is actually buried in the T wave — look for subtle deformity of the T wave just preceding the pause to detect nonconducted APC

ECG 78. 31-year-old male with palpitations:



GENERAL FEATURES

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- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

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- ☐ 08. Sinus arrhythmia
- ☐ 09. Sinus bradycardia (<60)
- ☐ 10. Sinus tachycardia (>100)
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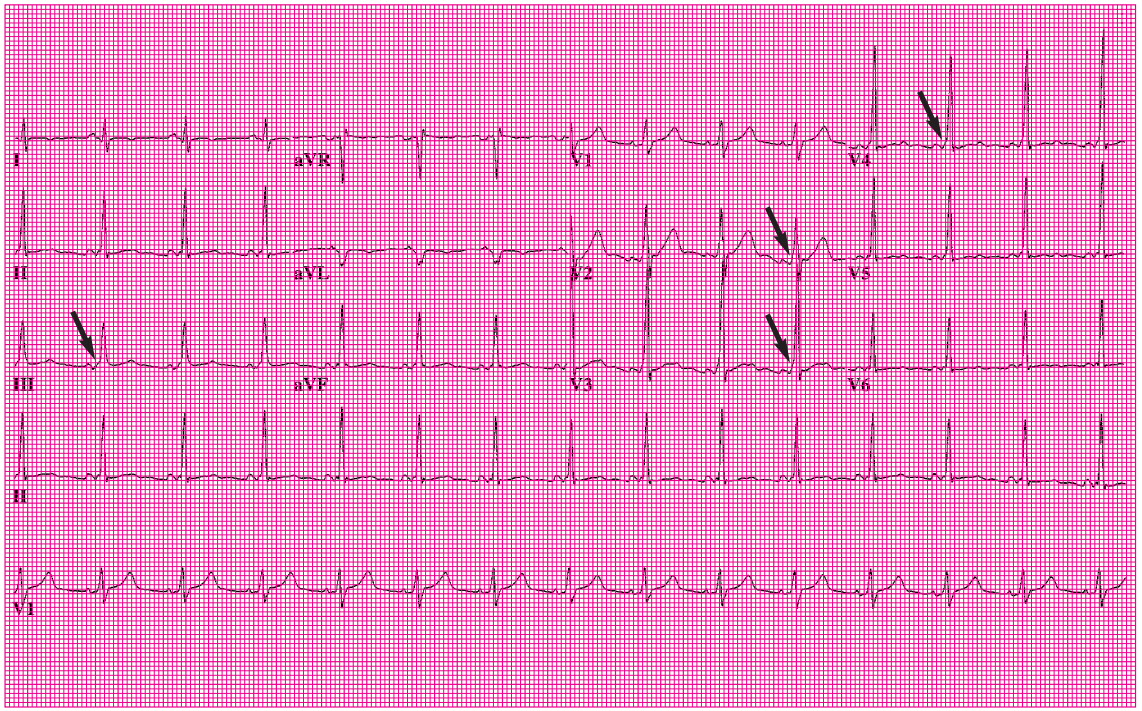
- ☐ 91. Ventricular demand pacemaker (VVI), normally functioning
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 78 was obtained in a 31-year-old male with palpitations. The tracing shows sinus rhythm with a short PR interval, delta waves (arrows), and a prolonged QRS (> 0.10 seconds), consistent with Wolff-Parkinson-White pattern.

Codes:

07	Sinus rhythm
34	Wolff-Parkinson-White pattern



Questions: ECG 78

1. Fusion complexes can be seen with:

   a. Wolff-Parkinson-White syndrome
   b. Atrial premature complexes
   c. Paced beats
   d. Ventricular tachycardia

2. Conditions associated with a short PR interval include:

   a. AV junctional rhythm
   b. Wolff-Parkinson-White syndrome
   c. Lown-Ganong-Levine syndrome
   d. Normal variant
   e. Pericarditis

3. Supraventricular tachycardia is needed to make the diagnosis of Wolff-Parkinson-White pattern:

   a. True
   b. False

4. Which of the following statements about Wolff-Parkinson-White syndrome are false:

   a. WPW may interfere with ECG recognition of left and right ventricular hypertrophy
   b. WPW may interfere with ECG recognition of bundle branch block
   c. WPW may interfere with ECG recognition of acute myocardial infarction
   d. The polarity of the delta waves can be used to accurately predict the location of the bypass tract
   e. A short QT interval is common in WPW

Answers: ECG 78

1. Fusion complexes result from simultaneous activation of the ventricle from two sources, resulting in a QRS complex intermediate in morphology between the QRS complex of each source. Fusion complexes can be seen with Wolff-Parkinson-White, paced beats, ventricular tachycardia, or isolated VPCs. Atrial premature complexes do not result in fusion complexes. (Answer: a, c, d)

2. The PR interval represents the time from the onset of atrial depolarization to the onset of ventricular depolarization (i.e., conduction from the atria → AV node → bundle of His → Purkinje fibers → ventricle). AV junctional rhythms can result in a short PR interval when retrograde atrial activation occurs before the

antegrade impulse reaches the ventricles. In the WPW syndrome, the presence of an accessory AV pathway (bundle of Kent), which connects the atria directly to the ventricles and bypasses the normal conduction delay in the AV node, prematurely activates the ventricles to result in a short PR. In the Lown-Ganong-Levine (LGL) syndrome, many experts believe that the short PR interval is due to "enhanced AV node conduction" from an immature AV node — not, as was once thought, from conduction down distinct atrioHisian fibers. In LGL syndrome, the QRS is normal in duration and configuration, unlike the WPW syndrome, in which more than 2/3 of cases show initial slurring of the QRS (delta wave) with a QRS duration $\geq$ 0.11 seconds. A short PR interval may also occur as a normal variant, although it is much more common in the pediatric population (as opposed to adults) and at faster (as compared to slower) heart rates. Pericarditis is associated with PR segment depression, but a short PR interval is not a characteristic finding. (Answer: all except e)

3. Wolff-Parkinson-White pattern differs from Wolff-Parkinson-White syndrome: the former requires delta waves and a short PR interval; the latter requires delta waves, a short PR, and a history of supraventricular tachycardia or atrial fibrillation. (Answer: b)

4. Wolff-Parkinson-White syndrome (WPW) is characterized by the presence of an abnormal muscular network of specialized conduction tissue that connects the atrium to the ventricle and bypasses conduction through the AV node. It is found in 0.2-0.4% of the overall population and is more common in males and younger patients. Most patients with WPW do not have structural heart disease, although there is an increased prevalence of this disorder among patients with Epstein's anomaly (downward displacement of the tricuspid valve into the right ventricle due to anomalous attachment of the tricuspid leaflets), hypertrophic cardiomyopathy, mitral valve prolapse, and dilated cardiomyopathy. ECG manifestations include a short PR interval (< 0.12 seconds) and a widened QRS complex (> 0.10 seconds) with slurring of the initial 30-50 milliseconds (delta wave). Two types of accessory pathways (AP) exist: In manifest AP, antegrade conduction occurs over the AP and results in preexcitation on baseline ECG (which may be intermittent). In concealed AP, antegrade conduction occurs via the AV node and retrograde conduction occurs over the AP, so preexcitation is not evident on the baseline ECG. Approximately 50% of patients with WPW manifest tachyarrhythmias, of which 80% is AV reentry tachycardia, 15% is atrial fibrillation, and 5% is atrial flutter. Asymptomatic individuals have an excellent prognosis. For patients with recurrent tachycardias, the overall prognosis is good but sudden death may occur. The presence of delta waves and secondary repolarization abnormalities can lead to a false positive or false negative diagnosis of ventricular hypertrophy, bundle branch block, or acute myocardial infarction. The polarity of the delta waves can be used to predict the location of the bypass tract. (Answer: e)

— Quick Review 78 —

Fusion complexes	
• Due to simultaneous activation of the ventricle from ____ sources, resulting in a QRS complex that is ____ in morphology between each source	2 intermediate
Wolff-Parkinson-White pattern	
• (Sinus/nonsinus) P wave	sinus
• PR interval < ____ seconds	0.12
• Initial slurring of QRS (____ wave) resulting in QRS duration > ____ seconds	delta 0.10
• Secondary ST-T wave changes occur (true/false)	true
• PJ interval (beginning of P wave to end of QRS) (is constant/varies)	is constant

— POP QUIZ —

Differential Diagnosis: PR Interval/Segment

Instructions: For each diagnosis below, select all PR interval/segment changes that apply:

a. Prolonged PR interval
b. Short PR Interval
c. PR segment depression
d. PR segment elevation

Diagnosis	Answer
Low ectopic atrial rhythm	Short PR interval. Inverted P waves in II, III, and aVF may be present, especially when the ectopic focus is in the lower atrium (near the AV node). Prolonged PR interval is unusual, and would require marked conduction delay in the AV node. PR segment deviation does not occur. (Answer: b)
3° AV block	Independence of atrial and ventricular rhythms results in varying PR intervals, which may be prolonged, normal and/or short. PR segment deviation does not occur. (Answer: a, b)
Atrial premature contractions (APCs)	PR interval may be prolonged, normal, or short, depending on the degree of prematurity and origin of the APC. In general, the more premature the APC, the longer the PR interval. APCs originating near the AV node tend to have shorter PR intervals (and inverted P waves). PR segment deviation does not occur. (Answer: a, b)
Wolff-Parkinson-White (WPW) syndrome	Short PR interval, due to conduction over accessory AV pathway (bundle of Kent), which bypasses the AV node (and AV nodal conduction delay). Slurring of the QRS complex is due to fusion of electrical wavefronts from conduction down the accessory pathway (delta wave) and AV node. (Answer: b)
Junctional rhythm with retrograde atrial activation	Retrograde atrial activity (manifest as inverted P waves) may immediately precede the QRS (short PR interval), be buried in the QRS (no P wave), or immediately follow the QRS (long PR interval). PR segment deviation does not occur. (Answer: a, b)
Pericarditis	Diffuse PR segment depression. PR interval is normal. (Answer: c)
Atrial infarction	PR elevation in area of infarction; PR depression in reciprocal leads. PR interval is normal. (Answer: c, d)

Differential Diagnosis

Fusion Complexes

Simultaneous activation of the ventricle from two sources, resulting in a QRS complex intermediate in morphology between the QRS complexes of each source

- Ventricular premature complexes
- Ventricular tachycardia
- Ventricular parasystole
- Accelerated idioventricular rhythm
- Wolff-Parkinson-White syndrome
- Paced rhythm

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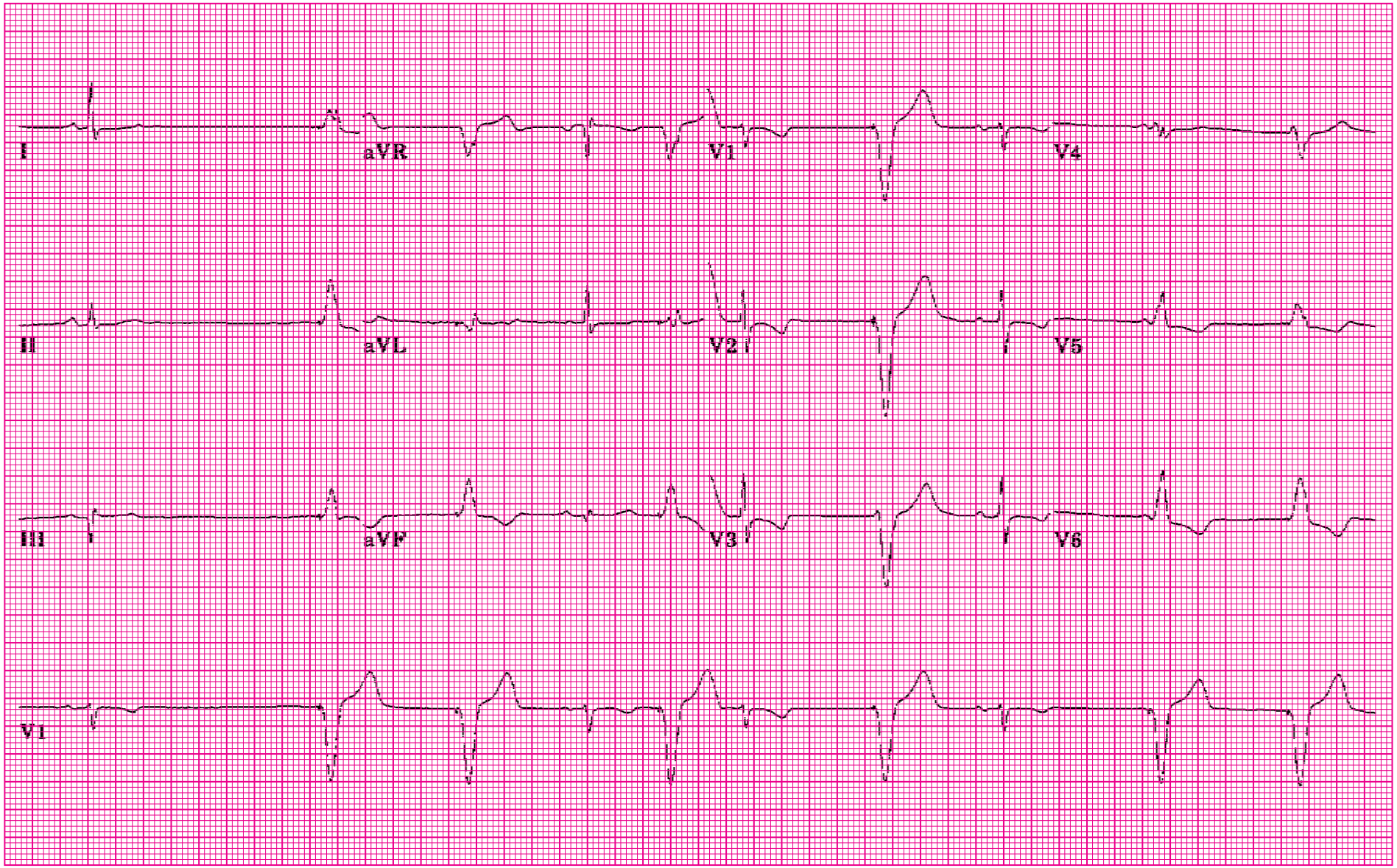

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- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

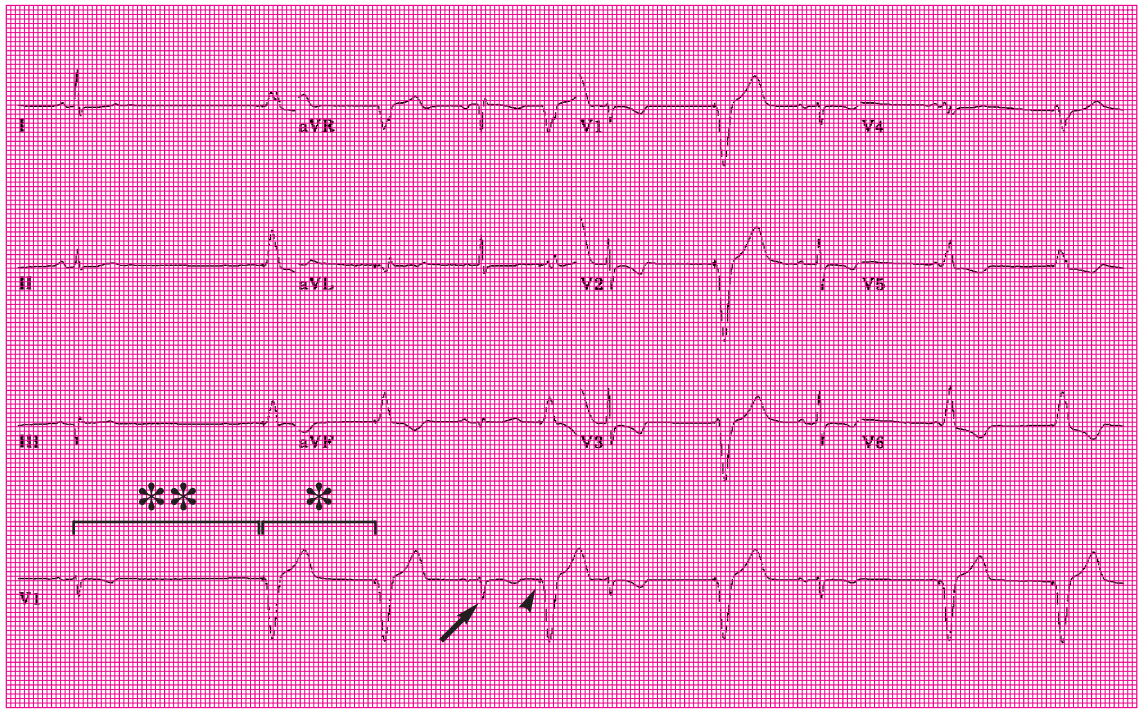
ECG 79 was obtained from an 80-year-old male with palpitations. The ECG shows an irregular rhythm with native and ventricular paced beats. The V-V interval of the pacemaker, evident from the separation between the 1st and 2nd ventricular pacing spikes (asterisk), represents the key timing interval of the pacemaker. The relatively long pause between the first and second beats (double asterisk) exceeds the V-V interval, indicating oversensing of electrical activity with inappropriate suppression of pacemaker firing. In addition, the pacemaker fails to sense the 2nd native QRS complex (arrow), resulting in premature firing of the pacemaker relative to the V-V interval (arrowhead). The 6th and 8th beats are appropriately sensed, so the sensing failure is intermittent. The native rhythm is marked sinus bradycardia. There is an abnormal Q wave in lead III, but inferior MI should not be coded since there is a small R wave in aVF. The native beats in leads V_1-V_3 show downsloping ST segment depression with T wave inversions, suggesting myocardial ischemia. This patient was found to have a crushed pacemaker lead.

Codes:

09 Sinus bradycardia

64 ST and/or T wave abnormalities suggesting myocardial ischemia

94 Pacemaker malfunction, not constantly sensing (atrium or ventricle)



Questions: ECG 79

1. Causes of pacemaker malfunction with failure to sense include:

    a. Type III antiarrhythmic drugs
    b. Electrolyte disorders
    c. Lead displacement
    d. Myocardial infarction
    e. Myopotential inhibition

2. Causes of pacemaker oversensing include:

    a. Lead fracture
    b. Myopotential inhibition
    c. T wave oversensing

Answers: ECG 79

1. Pacemaker malfunction with failure to sense can arise from any part of the pacing "circuit," including the pacemaker generator, the pacing lead, or lead contact with the ventricle. Nonviable myocardium, electrolyte abnormalities, and drugs such as Type III antiarrhythmics can also alter conductivity and result in sensing malfunction. (Answer: all)

2. T waves and muscle potentials may be sensed as atrial (P waves) or ventricular activity (QRS complexes), resulting in inhibition of pacemaker output. Oversensing of T waves and myopotential inhibition can be corrected by decreasing the sensitivity of the pacemaker. Lead fracture can cause erratic patterns of oversensing, undersensing, and failure to pace or capture; lead replacement is required. (Answer: all)

— Quick Review 79 —

ST and/or T wave abnormalities suggesting myocardial ischemia	
• Abnormally tall, symmetrical, (upright/inverted) T waves	inverted
• Horizontal or ____ ST segments with or without T wave inversion	downsloping
• Associated ECG findings:	
▸ QT interval is usually (normal/prolonged)	prolonged
▸ Reciprocal ____ wave changes may be evident	T
▸ Prominent U waves are often present and may be upright or inverted (true/false)	true
Pacemaker malfunction, not constantly sensing (atrium or ventricle)	
• Pacemakers in the inhibited mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	inhibited
• Pacemakers in the triggered mode: Pacemaker fails to be ____ by an appropriate intrinsic depolarization	triggered
• Premature depolarizations may not be sensed if they fall within the programmed ____ period of the pacemaker, *or* have insufficient ____ at the sensing electrode site	refractory amplitude

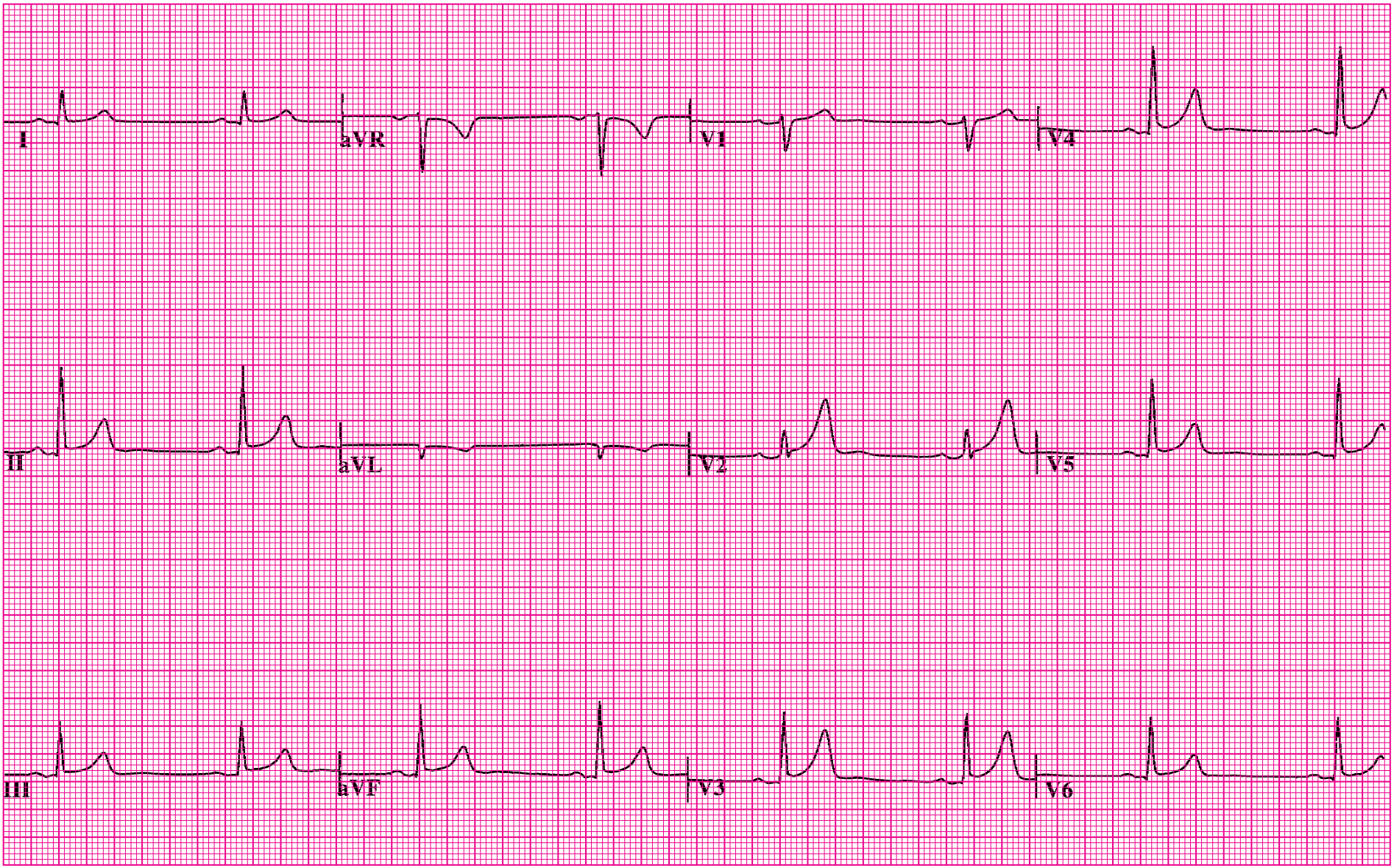
— POP QUIZ —

Make The Diagnosis

Instructions: Determine the ECG diagnosis that best corresponds to the ECG features listed below (see answer sheet for options).

ECG Features	Diagnosis
• Sensed atrial activity inhibits atrial output. If no ventricular activity is sensed by the end of the AV interval, ventricular pacing occurs	DDD pacing
• Pacemaker stimulus followed by an atrial depolarization	Atrial pacing
• Pacemaker stimulus followed by a QRS complex that has different morphology compared to the intrinsic QRS • Must demonstrate inhibition of pacemaker output in response to intrinsic QRS	Ventricular demand pacing
• Ventricular pacing without demonstrable output inhibition by intrinsic QRS complexes	Ventricular pacing, fixed rate, asynchronous
• Increase in stimulus intervals over the programmed intervals • Usually an indicator of battery end of life • Often noted first during magnet application	Pacemaker malfunction, slowing

ECG 80. Healthy 32-year-old male being screened for an insurance physical exam:



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- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

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- ☐ 06. Left atrial abnormality/enlargement

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- ☐ 08. Sinus arrhythmia
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- ☐ 10. Sinus tachycardia (>100)
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- ☐ 14. Atrial parasystole
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- ☐ 16. Atrial tachycardia, multifocal
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- ☐ 19. Atrial fibrillation

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- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

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- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
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- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

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- ☐ 76. Hypercalcemia
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- ☐ 79. Atrial septal defect, primum
- ☐ 80. Dextrocardia, mirror image
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- ☐ 83. Pericardial effusion
- ☐ 84. Acute pericarditis
- ☐ 85. Hypertrophic cardiomyopathy
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- ☐ 87. Myxedema
- ☐ 88. Hypothermia
- ☐ 89. Sick sinus syndrome

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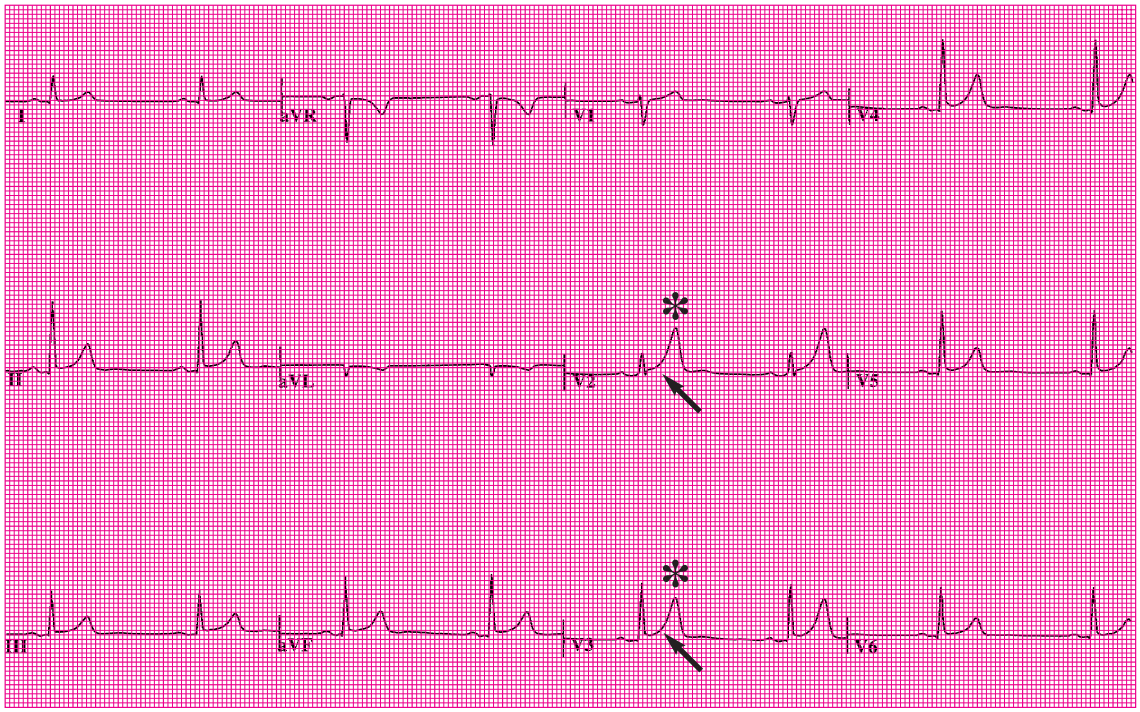
ECG 80 was obtained from a healthy 32-year-old male being screened for an insurance physical exam. The ECG shows sinus bradycardia at a rate of 47 beats/minute. Subtle notching of the J point (most apparent in leads II, III, aVF, V_2-V_6) with concave upward ST segment elevation (arrows) and tall upright T waves (asterisks) is consistent with normal variant early repolarization abnormality. All the findings in this tracing are consistent with normal variant ECG.

Codes:

02	Borderline normal ECG or normal variant
09	Sinus bradycardia (< 60)
61	Normal variant, early repolarization



Questions: ECG 80

1. Causes of diffuse ST elevation include:

   a. Acute MI
   b. Pericarditis
   c. Left ventricular hypertrophy
   d. Hyperkalemia
   e. LV aneurysm
   f. Variant (Printzmetal's) angina
   g. Early repolarization

2. Which of the following statements about ST elevation are true?

   Ventricular aneurysm:

   a. Q wave or QS is usually present in the same leads as ST segment elevation
   b. ST and T wave changes remain stable over time

   Pericarditis:

   a. Reciprocal ST depression is common
   b. Q waves are often evident
   c. ST and T wave changes remain stable over time
   d. T waves usually become inverted after ST segments return to baseline

3. "Normal variant" ECG findings include all of the following except:

   a. Small negative T waves in V_1 - V_3
   b. S waves in leads I - III
   c. Amplitude of R wave equal to depth of S wave in V_1
   d. Amplitude of R wave equal to depth of S wave in V_2
   e. ST elevation of 1-2 mm in V_2 and V_3
   f. Q wave duration $\geq$ 0.03 seconds
   g. ST depression in precordial leads
   h. U wave amplitude > 1.5 mm
   i. RSr' or rSR' in V_1 with a QRS duration < 0.10 seconds in V_1

Answers: ECG 80

1. Causes of diffuse ST elevation include pericarditis, severe hyperkalemia ("dialyzable current of injury"), and early repolarization (usually most apparent in leads II, III, aVF, and V_2-V_5). Focal ST elevation occurs in acute myocardial infarction, LV aneurysm, and variant angina, and is usually confined to the distribution of the culprit vessel. ST elevation with LVH is usually confined to leads V_1-V_4. (Answer: b, d, g)

2. The ST elevation of ventricular aneurysm differs from pericarditis in several ways: In ventricular aneurysm, ST elevation is localized, Q waves are usually present in the same

leads as ST elevation, and ST and T wave changes remain stable over time. In pericarditis, ST elevation is diffuse, Q waves are not evident (unless pericarditis follows acute MI), and ST and T wave changes evolve and are transient. The ST elevation of pericarditis differs from acute MI in that reciprocal ST depression does not occur, and T waves usually become inverted *after* the ST segment has returned to baseline. (Answers: ventricular aneurysm = a, b; pericarditis = d)

3. The transition zone is defined as the lead in which the amplitude of the positive and negative QRS deflections are equal (R/S = 1). The normal transition zone occurs in lead V_2, V_3, or V_4. A tall R wave in V_1 (R > S) is abnormal in adults, and may occur in posterior MI, right ventricular hypertrophy, WPW syndrome, or chronic lung disease. Q wave duration > 0.03 seconds is abnormal for most leads, and occurs in myocardial infarction, cardiomyopathy, pulmonary embolism, infiltrative myocardial disorders (e.g., amyloid, sarcoid, muscular dystrophy), CNS disorders, among others. ST depression or elevation of 1 mm in the limb leads, and ST elevation of 1-2 mm in the precordial leads (especially V_2, V_3) can be seen in normals, but ST depression in the precordial leads is abnormal. Shallow T wave inversion in leads V_1-V_3 is a common normal variant, especially in children and women. An incomplete RBBB pattern in lead V_1 can be seen in 2% of normals. (Answer: c, f, g)

— Quick Review 80 —

Normal variant, early repolarization	
• Elevated ____ of the ST segment at the J junction	take-off
• (Concave/convex) upward ST elevation ending with a symmetrical upright T wave, which is often of large amplitude	concave
• Distinct notch or slur on downstroke of ____ wave	R
• Most commonly involves leads ____	V_2-V_5
• Reciprocal ST segment depression is present (true/false)	false
• Some degree of ST elevation is present in the majority of young healthy individuals, especially in the precordial leads (true/false)	true

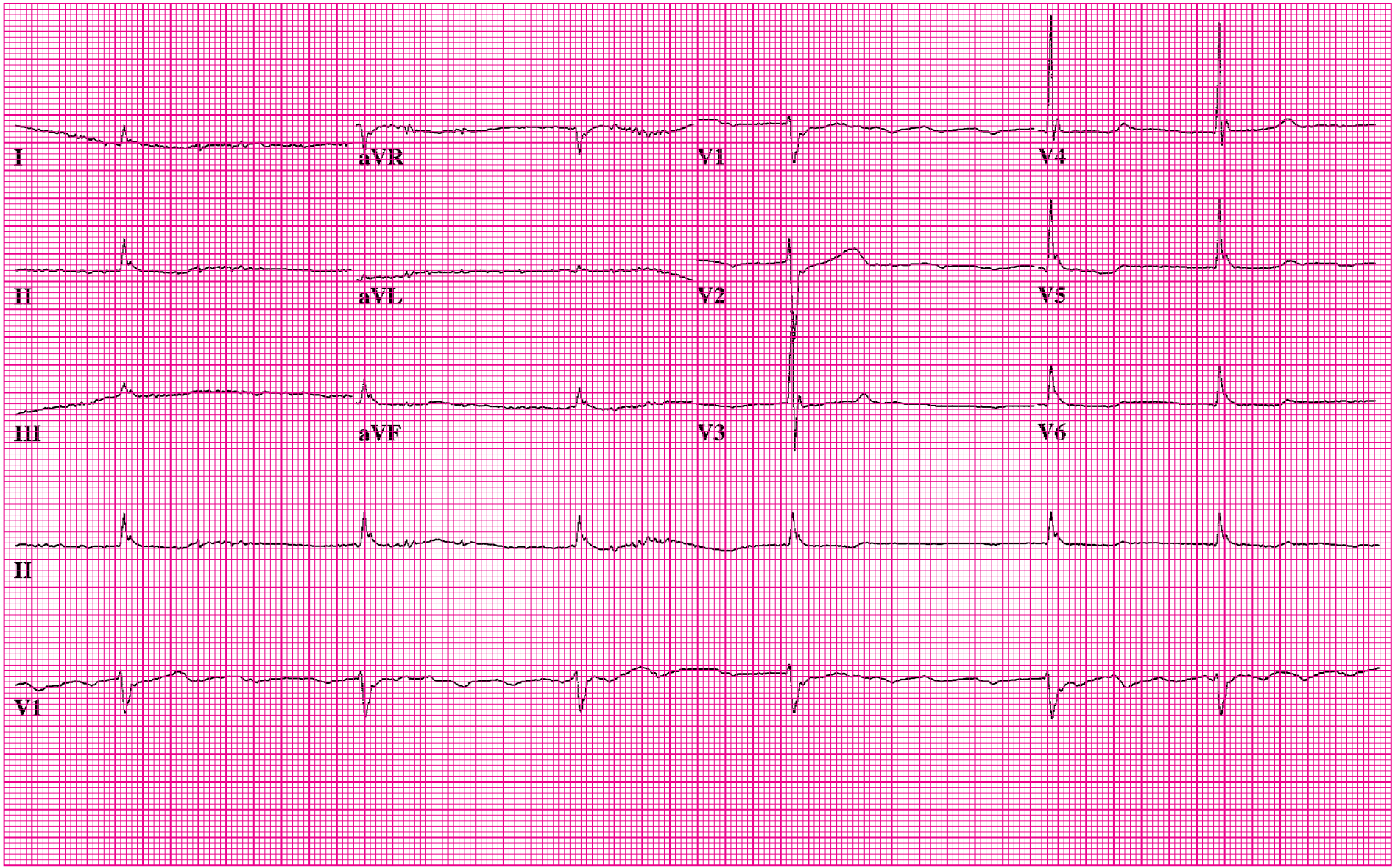
Differential Diagnosis

Electrical Alternans

(Alternation in the amplitude and/or direction of P, QRS, and/or T waves)

- Pericardial effusion. Only 12% of patients with pericardial effusion have electrical alternans. If electrical alternans involves the P, QRS, and T ("total alternans"), effusion with tamponade is often present.
- Severe left ventricular failure
- Hypertension
- Coronary artery disease
- Rheumatic heart disease
- Supraventricular or ventricular tachycardia
- Deep respirations

ECG 81. 64-year-old male found unconscious:



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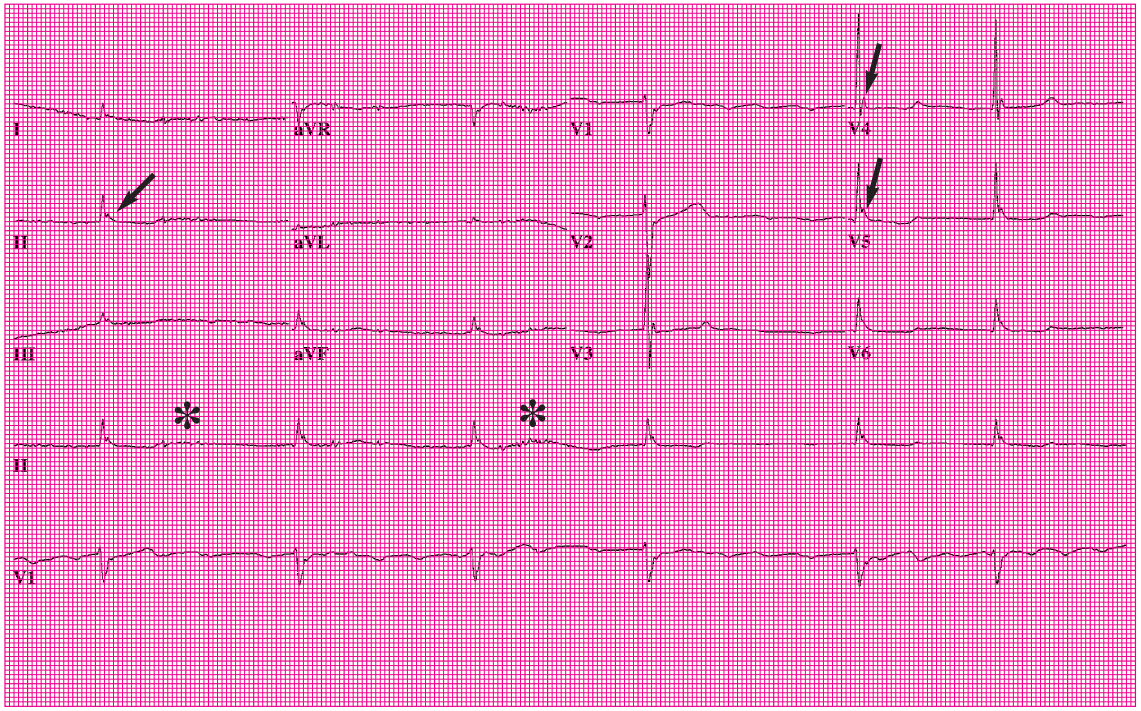
ECG 81 was obtained in a 64-year-old male found unconscious. The ECG shows atrial fibrillation with a very slow ventricular response, prominent J ("Osborne") waves (arrows), and nonspecific QRS widening. Artifact due to shivering (asterisks) is superimposed on the atrial fibrillation. These findings are consistent with hypothermia.

Codes:

04	Artifact
19	Atrial fibrillation
49	Nonspecific intraventricular conduction disturbance
88	Hypothermia



Questions: ECG 81

1. ECG findings consistent with hypothermia include:

   a. Osborne wave
   b. Junctional rhythm
   c. Atrial fibrillation with slow ventricular response
   d. Prolonged PR, QRS, and QT intervals
   e. Sinus bradycardia
   f. T wave inversions

2. The oscillations in the baseline seen in the present tracing (asterisks) are most likely due to:

   a. Fibrillation waves
   b. Parkinson's disease
   c. Loose ECG electrode
   d. Muscle tremor

Answers: ECG 81

1. Profound hypothermia (core temperature < 32° C) causes peripheral vasoconstriction, impaired enzymatic activity, decreased cardiac output, and reduced respirations. Complications include aspiration pneumonia, adult respiratory distress syndrome, pulmonary edema, rhabdomyolysis, acute tubular necrosis, gastric dilitation, upper GI bleed, hyperviscosity syndrome, and disseminated intravascular coagulation. The classic ECG finding of hypothermia is the Osborne wave (or "J" wave), which is an extra positive deflection between the terminal portion of the QRS complex and the beginning of ST segment. The Osborne wave is usually positive in the left precordial leads, and has an amplitude that is inversely proportional to body temperature. Other ECG changes caused by hypothermia include prolongation of the PR, QRS, and QT intervals; T wave inversion; and bradyarrhythmias consisting of sinus bradycardia, junctional rhythm, or atrial fibrillation with a slow ventricular response. (Answer: all)

2. Signals unrelated to cardiac conduction are seen frequently on the ECG. Muscle tremor (e.g., shivering or Parkinson disease) can be continuous or intermittent, and in some instances, crescendo-decrescendo in character (e.g., scratching). Physiologic tremor occurs at a rate of 7-9 cycles per second (~ 500 per minute); the tremor of Parkinson's disease occurs at a rate of 4-6 cycles per second (~ 300 per minute) and can simulate atrial flutter. AC electrical interference, particularly 60-cycle oscillations, can be severe in intensive care units, operating rooms, and cardiac catheterization laboratories. The extremely rapid, intermittent oscillations in this severely hypothermic patient were due to shivering. (Answer: d)

— Quick Review 81 —

Atrial fibrillation	
• ____ waves are absent	P
• Atrial activity is totally ____ and represented by fibrillatory (f) waves of varying amplitudes, duration and morphology	irregular
• Atrial activity is best seen in the ____ and ____ leads	right precordial, inferior
• Ventricular rhythm is (regularly/irregularly) irregular	irregularly
• ____ toxicity may result in regularization of the RR interval due to complete heart block with junctional tachycardia	Digitalis
• Ventricular rate is usually ____ per minute in the absence of drugs	100-180
▸ Think ____ if the ventricular rate is > 200 per minute and the QRS is > 0.12 seconds	Wolff-Parkinson-White
Hypothermia	
• Sinus (tachycardia/bradycardia)	bradycardia
• PR, QRS, and QT prolonged (true/false)	true
• Osborne ("J") wave: late upright terminal deflection of QRS complex; amplitude (increases/decreases) as temperature declines	increases
• Atrial ____ in 50-60%	fibrillation
• Other arrhythmias include AV junctional rhythm, ventricular tachycardia, ventricular fibrillation (true/false)	true

Don't Get Confused!

Wandering Atrial Pacemaker

P waves with ≥ 3 morphologies, atrial rate <100 per minute, and varying PR, RR, and RP intervals

<u>May be confused with</u>:

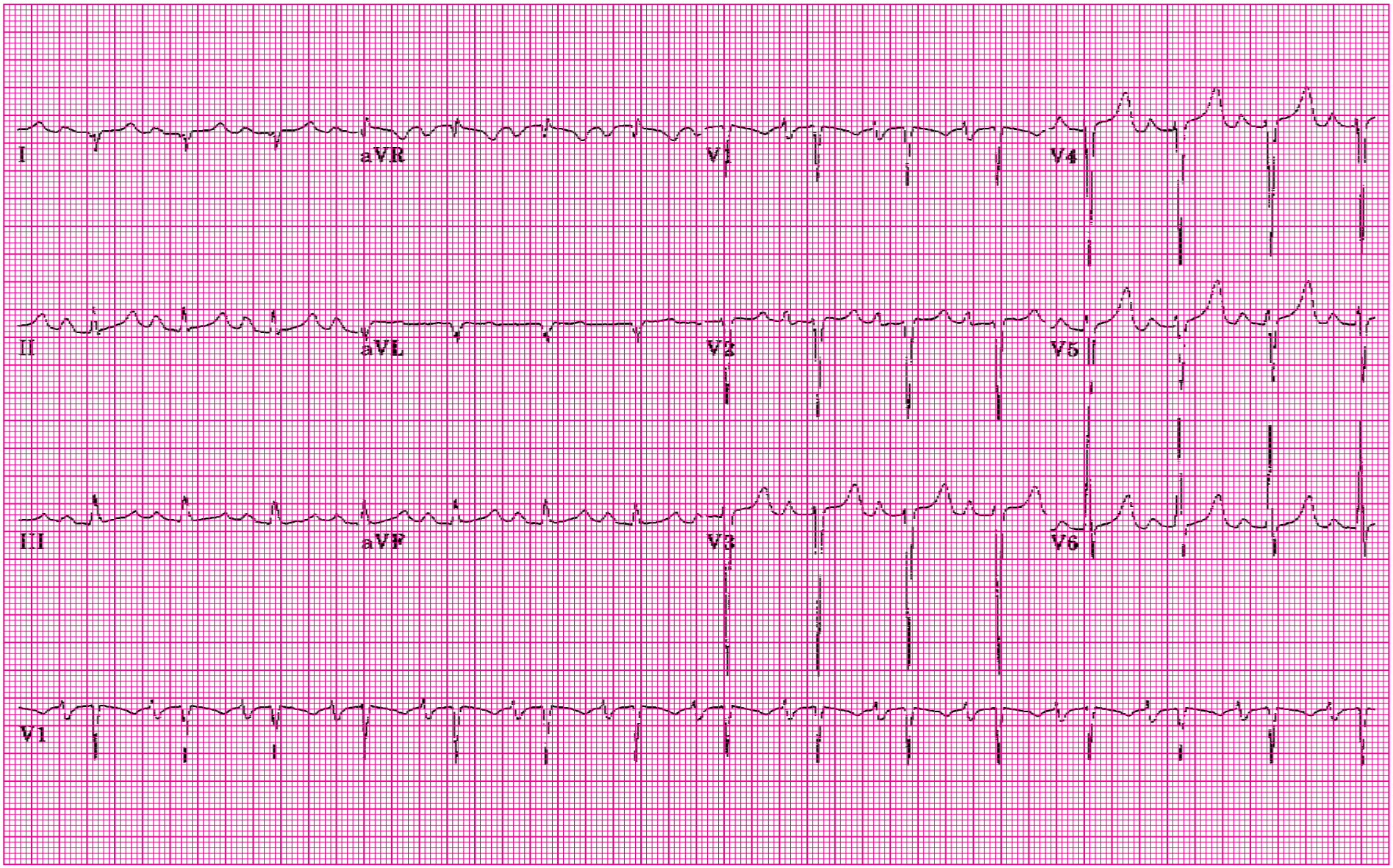
Sinus rhythm with multifocal APCs

Sinus rhythm with multifocal APCs demonstrates one dominant atrial pacemaker (i.e., the sinus node); in wandering atrial pacemaker, *no* dominant atrial pacemaker (i.e., no dominant P wave morphology) is present

Atrial fibrillation/flutter

In atrial fibrillation/flutter, there is lack of an isoelectric baseline; in wandering atrial pacemaker, a distinct isoelectric baseline is present

ECG 82. 58-year-old female with chest pain 2 days ago:



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Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
- ☐ 55. Lateral (age recent or acute)
- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

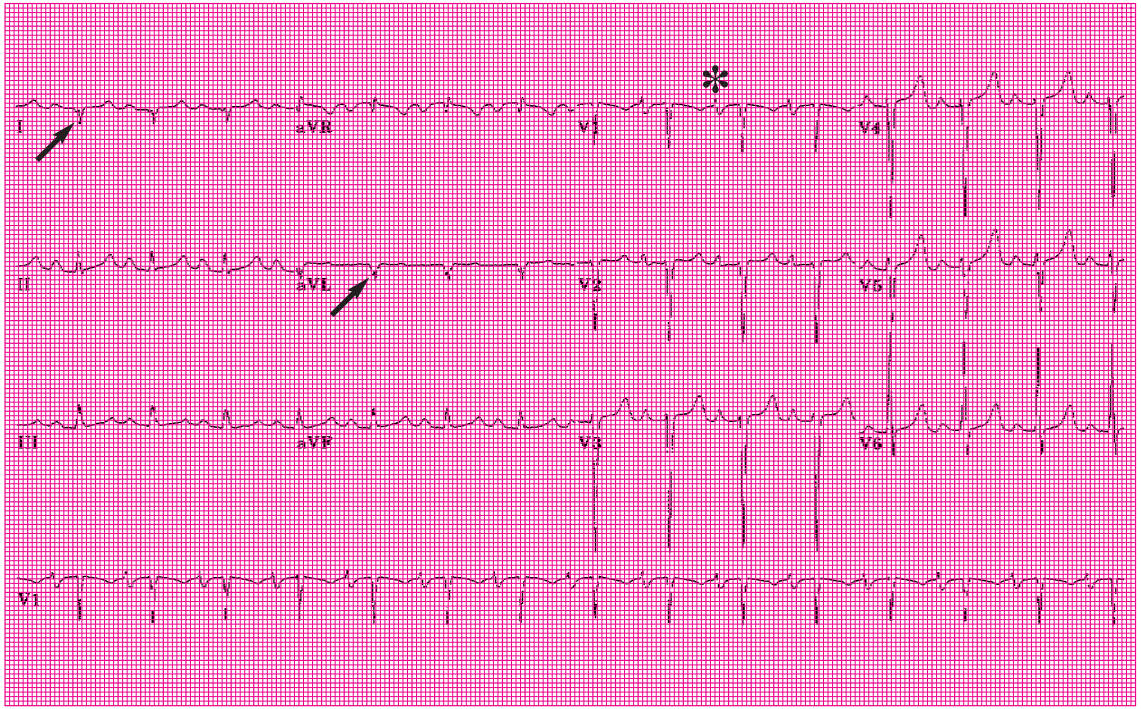
PACED RHYTHMS

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

ECG 82 was obtained from a 58-year-old female with chest pain two days ago. The ECG shows sinus rhythm at a rate of 91 beats/minute with first-degree AV block (PR interval = 0.21 seconds). Leads I and aVL show abnormal Q waves (arrows), consistent with old lateral wall myocardial infarction, which accounts for the right axis deviation. There is poor R wave progression (transition zone between V_5 and V_6), but in the absence of abnormal Q waves, anterior infarct should not be coded. Also present are left and right atrial enlargement (asterisk), LVH by Cornell criteria (R in aVL + S in V_3 > 20 mm in females), and prolonged QT interval (QTc = 0.49 seconds). The ST segment elevation in V_2-V_4 is most likely due to LVH.

Codes:

05	Right atrial abnormality/enlargement
06	Left atrial abnormality/enlargement
07	Sinus rhythm
29	AV block, 1°
37	Right axis deviation (>+100º)
40	Left ventricular hypertrophy
56	Lateral MI (age indeterminate or old)
68	Prolonged QT interval



Questions: ECG 82

1. Criteria for bi-atrial enlargement include:

    a. P wave amplitude > 2.5 mm and duration ≥ 0.12 seconds in leads II, III, or aVF
    b. P wave amplitude > 1.5 mm in leads V_1-V_3 with wide, notched P waves in leads II, III or aVF
    c. Biphasic P wave in lead V_1 with an initial positive amplitude > 1.5 mm and a terminal negative amplitude > 1 mm

2. Causes of right axis deviation include:

    a. Lateral myocardial infarction
    b. Right bundle branch block
    c. Right ventricular hypertrophy
    d. Ostium secundum ASD
    e. Dextrocardia
    f. Chronic lung disease (e.g., emphysema)

Answers: ECG 82

1. The diagnosis of bi-atrial enlargement is based on criteria used for individual atrial enlargement. All three choices are correct (Answer: all)

2. Right axis deviation can be seen as a normal variant, but is more often associated with COPD, cor pulmonale, right ventricular hypertrophy, lateral MI, left posterior fascicular block (LPFB), dextrocardia, lead reversal (apparent RAD), ostium secundum ASD, and Wolff-Parkinson-White syndrome. The mean QRS axis in right bundle branch block is normal. Right axis deviation (QRS axis 90° to 180°) must be distinguished from right *superior* axis (-90° to -180°), which can be caused by left anterior fascicular block with right ventricular hypertrophy or lateral MI, right ventricular hypertrophy alone, or COPD. (Answer: all except b)

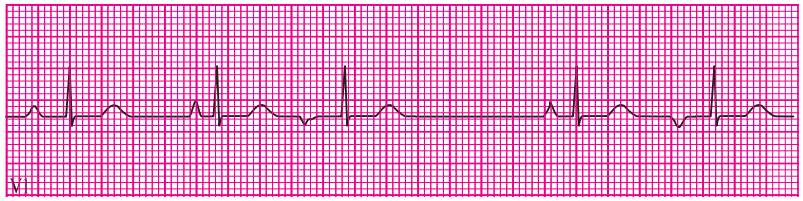
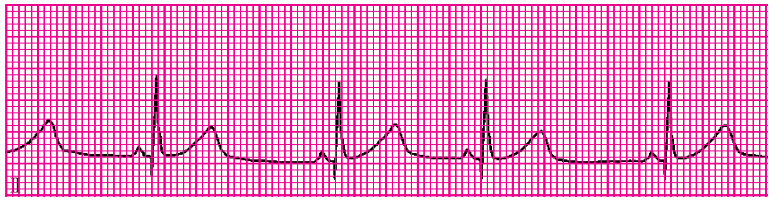
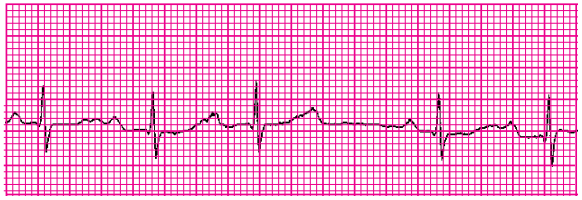
— Quick Review 82 —

Right atrial abnormality	
• Upright P wave > ____ mm in leads II, III and aVF	2.5
or > ____ mm in leads V_1 or V_2	1.5
• P wave axis ≥ ____ degrees	70
Left atrial abnormality	
• Notched P wave with a duration ≥ ____ seconds in leads II, III or aVF, *or*	0.12
• Terminal negative portion of the P wave in lead V_1 ≥ 1 mm deep and ≥ ____ seconds in duration	0.04
Right axis deviation	
• Mean QRS axis between ____ and ____ degrees	101, 270
Prolonged QT interval	
• Corrected QT interval (QTc) ≥ ____ seconds, where QTc = QT interval divided by the square root of the preceding ____ interval	0.44 RR
• QT interval varies (directly/inversely) with heart rate	inversely
• The normal QT interval should be (less than/greater than) 50% of the RR interval	less than

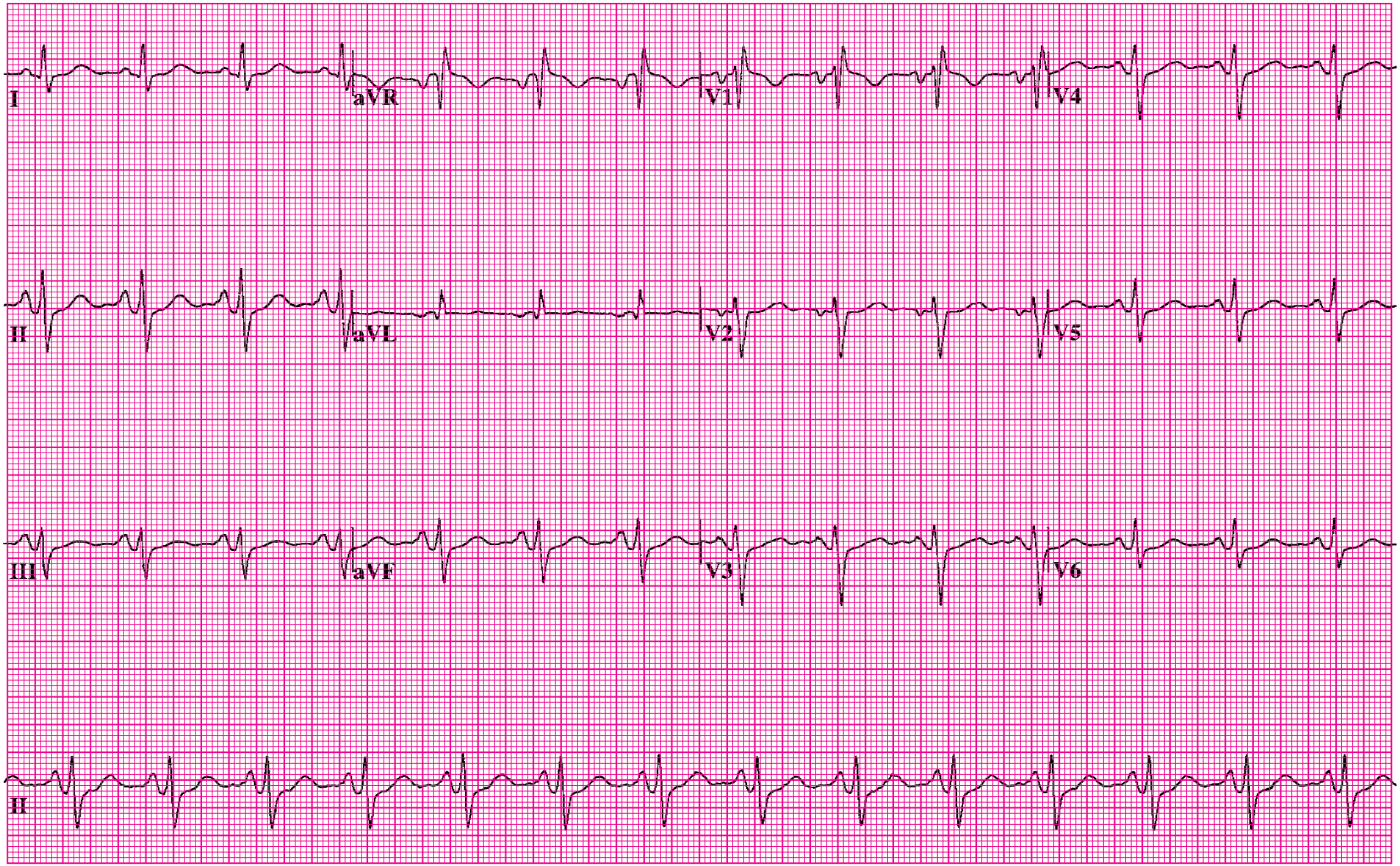
— POP QUIZ —

Rhythm Recognition: HR < 100; Narrow QRS; Irregular RR Interval

Instructions: Determine the cardiac rhythm for each of the following ECGs.

ECG	Diagnosis
	Answer: Wandering atrial pacemaker. Description: Irregular atrial (nonsinus) rhythm with at least three different P wave morphologies (originating from separate atrial foci) at an atrial rate < 100 per minute with varying PP and PR intervals. (Rhythm may be relatively constant if atrial foci are in close proximity to each other.) P waves may be blocked (not followed by a QRS complex), or may conduct with a narrow or aberrant (wide) QRS complex. Sometimes confused with atrial fibrillation/flutter or sinus rhythm with multifocal APCs. Seen in normals, athletes, and organic heart disease.
	Answer: Sinus arrhythmia. Description: Sinus rhythm with a gradual (sometimes abrupt) phasic change in PP interval, usually in response to the breath cycle. Longest and shortest PP intervals vary by > 0.16 seconds or 10%. Common in young adults and athletes. A marker for intact vagal activity.
	Answer: 2° degree AV block, Mobitz Type I (Wenkebach). Description: Regular sinus or atrial rhythm with intermittent nonconducted (blocked) P waves. Classic Wenkebach periodicity manifests as progressive lengthening of the PR interval and shortening of the RR interval until a P wave is blocked; the RR interval containing the nonconducted P wave is less than two PP intervals. Block usually occurs at the level of the AV node, resulting in a narrow QRS complex. Causes include drugs (e.g., digitalis, beta-blockers), myocardial infarction (especially inferior), acute rheumatic fever, and myocarditis; sometimes seen in normals and athletes. Note: Classical Wenckebach periodicity may not be evident in the presence of sinus arrhythmia or an abrupt change in autonomic tone (e.g., vagal reaction).

ECG 83. 46-year-old male with a history of rheumatic fever now with dyspnea on exertion:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
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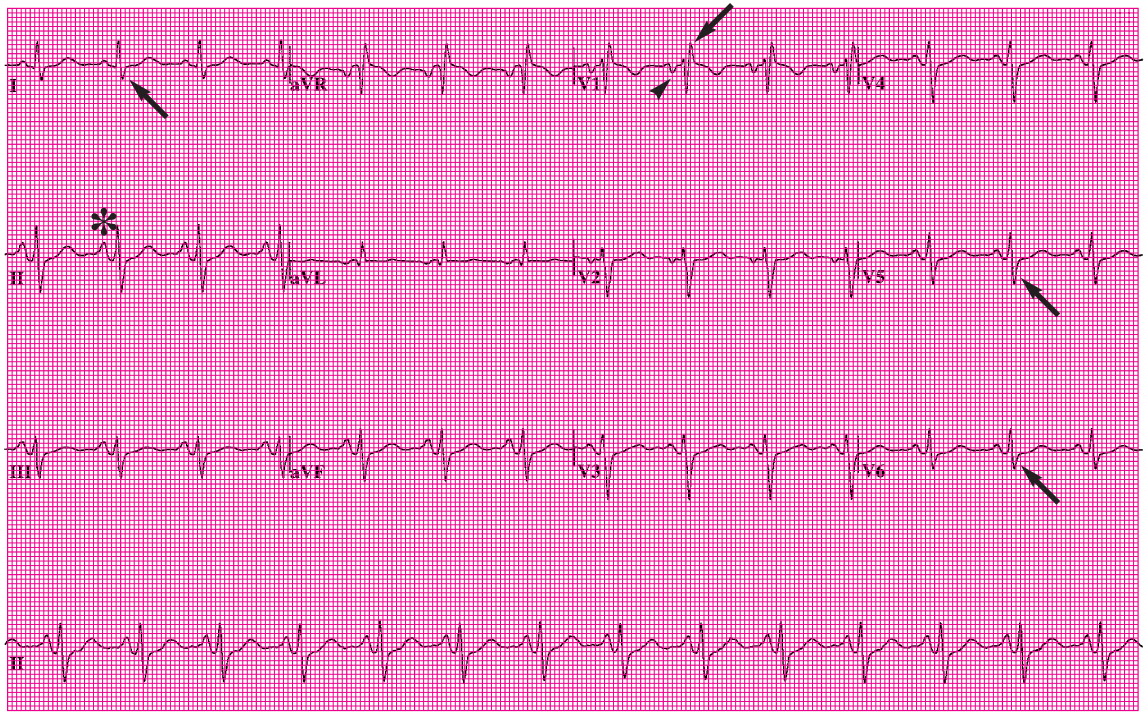
- ☐ 92. Dual-chamber pacemaker (DDD)
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 83 was obtained from a 46-year-old male with dyspnea on exertion and a history of rheumatic fever. The ECG shows sinus rhythm with RBBB (arrows mark wide rSR' complex in V_1, and wide, slurred S waves in I, V_5, V_6) and secondary ST-T abnormalities. Right atrial abnormality (asterisk), left atrial abnormality (arrowhead), and left axis deviation are evident. The axis is –35°, which does not meet criteria for left anterior fascicular block (i.e., axis > –45°). These findings are compatible with mitral valve disease.

Codes:

05	Right atrial abnormality/enlargement
06	Left atrial abnormality/enlargement
07	Sinus rhythm
43	RBBB, complete



Questions: ECG 83

1. Which of the following statements about the P wave are true:

    a. The right atrium is responsible for the electrical potential inscription in the late portion of the P wave
    b. The P wave is normally upright in leads I, II and aVF, and inverted in aVR
    c. Anatomical left atrial enlargement can exist with normal P wave amplitude, duration, and contour
    d. Left atrial enlargement can cause a P-pulmonale pattern

2. P-pulmonale can be seen in:

    a. Tetralogy of Fallot
    b. COPD without cor pulmonale
    c. Pulmonary embolism
    d. Normal variant

3. Notching and widening of the P wave (P-mitrale) may be caused by:

    a. Intra-atrial conduction delay
    b. Atrial dilatation
    c. Atrial hypertrophy

4. Which of the following statements about the PR interval/segment are true:

    a. The PR interval correlates with the period of atrial repolarization
    b. Leads with tall P waves are more likely to have PR depression than leads with smaller P waves
    c. PR elevation can be a normal finding
    d. PR depression can be a normal finding

Answers: ECG 83

1. The right and left atria are responsible for the electrical potential inscription in the early and late portions of the P wave, respectively. The P wave amplitude, duration, and contour lack sensitivity and specificity for left atrial enlargement (i.e., left atrial enlargement can exist with a normal P wave, and P-mitrale may occur without left atrial enlargement). Since the left atrium is responsible for the electrical potential inscription in the late portion of the P wave, left atrial enlargement can result in a pseudo-P-pulmonale pattern in the absence of right atrial enlargement. (Answer: b, c, d)

2. P-pulmonale, defined as a tall and peaked P wave (amplitude $\geq$ 2.5 mm in leads II, III, aVF) of normal duration, may be seen in pulmonary embolism (usually transient), COPD with or without cor pulmonale, or as a normal variant in patients with a thin body habitus or verticle heart. P-pulmonale can also be seen in

tetralogy of Fallot and other forms of congenital heart disease, including Eisenmenger's physiology, tricuspid atresia, pulmonary hypertension, and pulmonic stenosis. (Answer: all)

3. P-mitrale is defined by the presence of a notched and widened (≥ 0.12 seconds) P wave. While minor notching is common, pronounced notching (peak-to-peak interval > 0.04 seconds) is unusual. Mechanisms responsible for P-mitrale include left atrial hypertrophy or dilatation, intra-atrial conduction delay, increased left atrial volume, or an acute rise in left atrial pressure. (Answer: all)

4. The PR segment represents the time from the onset of atrial depolarization to the onset of ventricular depolarization. It is usually oriented in polarity opposite to that of the P wave, and is most pronounced in leads with taller P waves. PR depression < 0.8 mm is present on many normal ECGs, but PR depression > 0.8 mm is often abnormal. PR elevation in any lead other than aVR is abnormal. (Answer: b, d)

— Quick Review 83 —

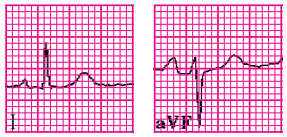
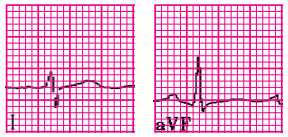
Mitral valve disease

- Mitral stenosis: Combination of (right/left) ventricular hypertrophy and (right/left) atrial abnormality is suggestive — right / left
- Mitral valve prolapse:
  - Flattened or inverted ____ waves in leads II, III and aVF (and sometimes in right precordial leads) ± ST segment depression in the left precordial leads — T
  - Prominent ____ waves — U
  - Prolonged ____ interval — QT

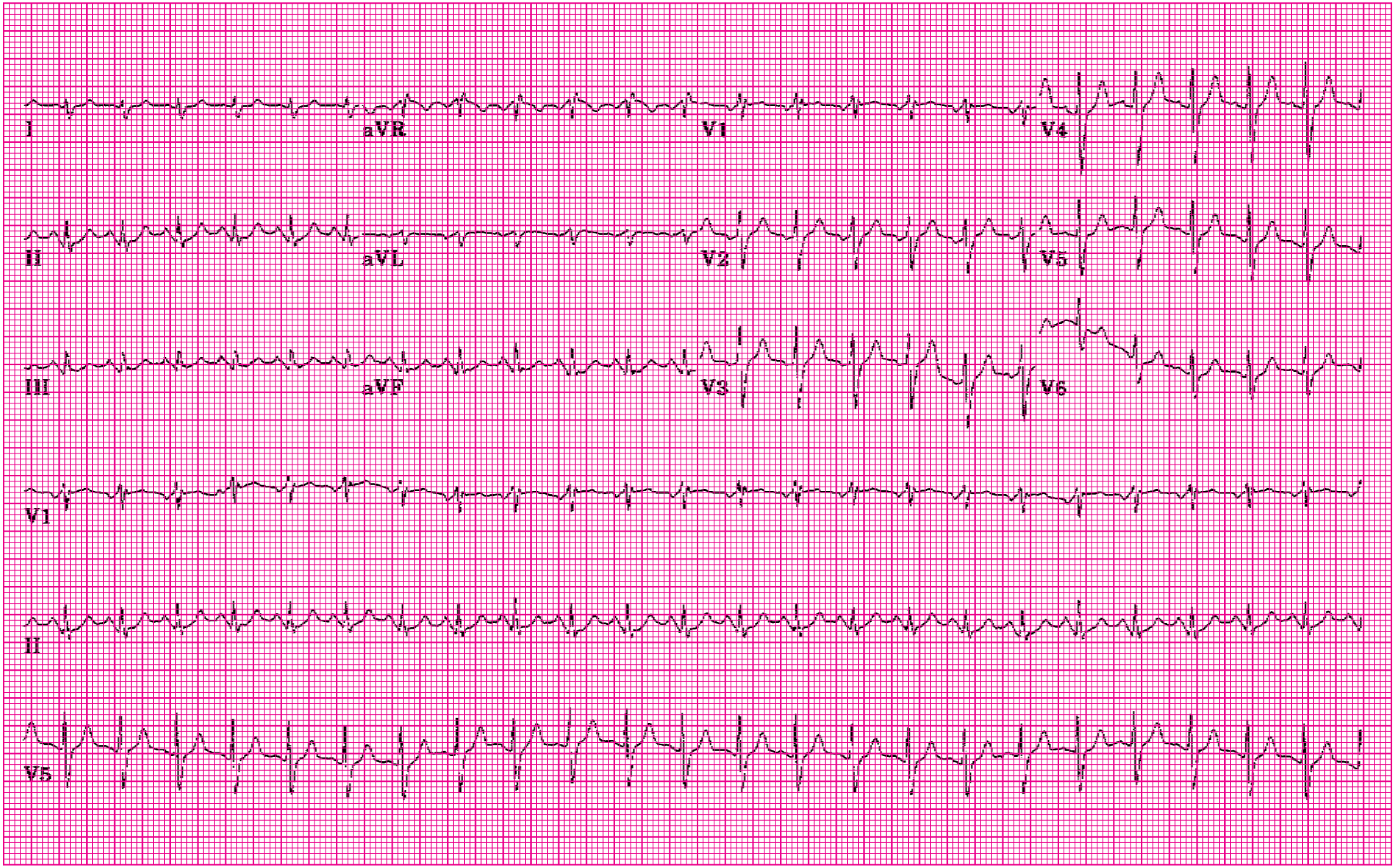
— POP QUIZ —

Pattern Recognition: Intraventricular Conduction Disturbances

Instructions: Match the following ECGs with all descriptions that apply.

ECG	Choose All That Apply	Answer
	a. Right bundle branch block b. QRS axis is usually normal c. Does not interfere with ECG diagnosis of ventricular hypertrophy d. Left anterior fascicular block e. Can result in false-positive diagnosis of LVH based on voltage criteria using leads I or aVL f. Can mask the presence of lateral wall MI g. Left posterior fascicular block h. Can mask the presence of inferior wall MI i. Least prevelant conduction abnormality j. Left bundle branch block k. Commonly associated with secondary ST & T changes in opposite direction to main QRS complex	*Left anterior fascicular block* (LAFB) results in left axis deviation (mean QRS axis between -45° and -90°); qR complexes (or an R wave) in leads I and aVL; rS complexes in lead III; and normal or slightly prolonged QRS duration (0.08-0.10 seconds). The diagnosis requires that no other cause of left axis deviation is present (LVH, inferior wall MI, chronic lung disease, left bundle branch block, ostium premum atrial septal defect, severe hyperkalemia). LAFB reduces the specificity of LVH based on voltage criteria using only leads I or aVL, and can mask the presence of inferior wall MI on ECG. LAFB is seen in organic heart disease, congenital heart disease, and rarely in normals. (Answer: d, e, h)
		*Left posterior fascicular block* (LPFB) results in right axis deviation (mean QRS axis between +101° and +180°); an S_1 Q_3 pattern (deep S wave in lead I and Q wave in lead III); and normal or slightly prolonged QRS duration (0.08-0.10 seconds). The diagnosis requires that no other cause of right axis deviation is present (RVH, vertical heart, chronic lung disease, pulmonary embolism, lateral wall MI, dextrocardia, lead reversal, ostium secundum ASD, Wolff-Parkinson-White syndrome). LPFB can mask the presence of lateral wall MI on ECG. Isolated LPFB is much less prevalent than left bundle branch block, right bundle branch block, or left anterior fascicular block. LPFB is seen most commonly with coronary artery disease, and is rare in normals. (Answer: c, f, g, i)

ECG 84. 69-year-old smoker with dyspnea:



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- ☐ 05. Right atrial abnormality/enlargement
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- ☐ 07. Sinus rhythm
- ☐ 08. Sinus arrhythmia
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- ☐ 33. AV block, 3°
- ☐ 34. Wolff-Parkinson-White pattern
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- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

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- ☐ 41. Right ventricular hypertrophy
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- ☐ 82. Acute cor pulmonale including pulmonary embolus
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- ☐ 86. Central nervous system disorder
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PACED RHYTHMS

- ☐ 90. Atrial or coronary sinus pacing
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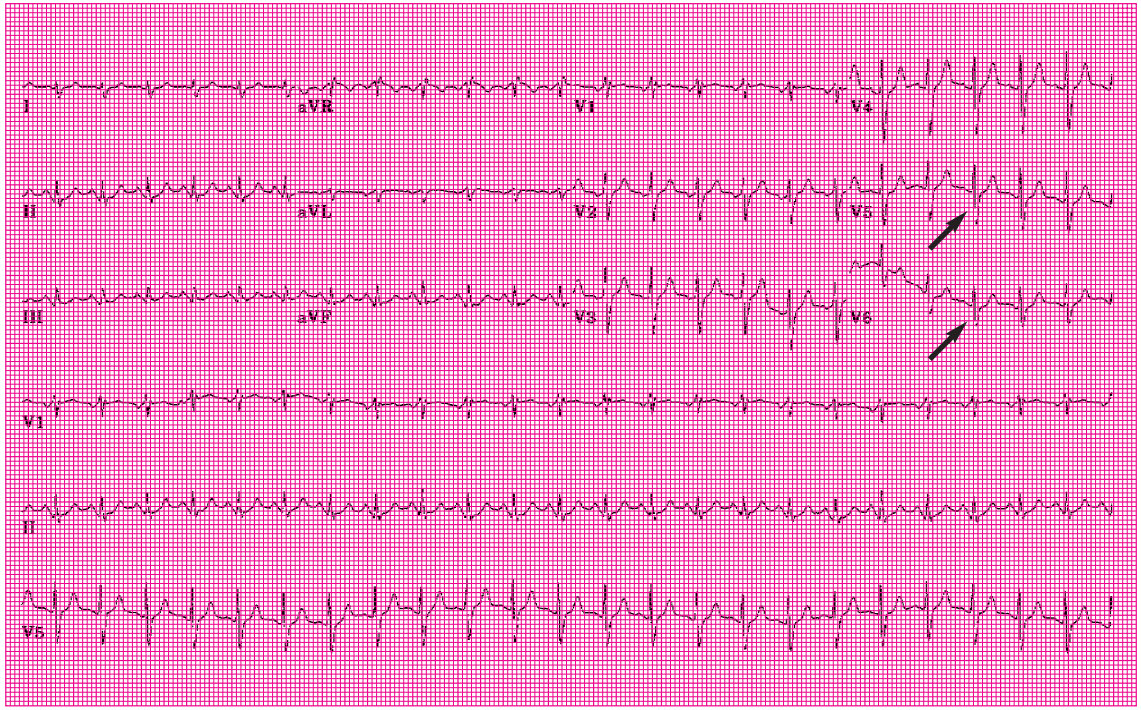
- ☐ 93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECG 84 was obtained from a 69-year-old smoker with dyspnea. The ECG shows sinus tachycardia at a rate of 144 beats/minute. There is a rightward axis (which does not quite meet criteria for right axis deviation) and prominent S waves in the left precordial leads (R/S < 1 in V_5-V_6) (arrows), consistent with right ventricular hypertrophy. Poor R wave progression in the precordial leads and low voltage in the limb leads are also present. This constellation of findings is consistent with chronic lung disease. Also noted are insignificant Q waves in II, III and aVF, and an RSR' pattern in V_1, which fail to meet criteria for Q-wave MI or incomplete RBBB.

Codes:

10	Sinus tachycardia (> 100)
41	Right ventricular hypertrophy
81	Chronic lung disease



Questions: ECG 84

1. Chronic lung disease is suggested by:

    a. Poor R wave progression in the precordial leads
    b. Early R wave progression in the precordial leads
    c. Right axis deviation
    d. Right atrial enlargement
    e. Low voltage QRS

2. Causes of right axis deviation include all of the following except:

    a. Left posterior fascicular block
    b. Lateral myocardial infarction
    c. Ostium primum atrial septal defect
    d. Limb lead misplacement

Answers: ECG 84

1. Chronic lung disease is characterized by poor R wave progression across the anterior precordial leads, which may be mistaken for prior anterior myocardial infarction. Other common findings include sinus tachycardia, right axis deviation, right atrial enlargement, right bundle branch block, and low voltage. Many of these findings can also be seen in acute cor pulmonale, including pulmonary embolism. Early R wave progression is not associated with chronic lung disease, unless it is complicated by pulmonary hypertension with right ventricular hypertrophy. (Answer: all except b)

2. Right axis deviation (QRS axis > +100°) is seen in many conditions, including left posterior fascicular block, lateral wall MI, right ventricular hypertrophy, chronic lung disease, pulmonary embolism, and dextrocardia. Transposition of ECG electrodes I and aVL can cause inversion of the P-QRS-T complex in these leads and apparent right axis deviation. Ostium secundum atrial septal defect (ASD) causes right axis deviation; primum ASD results in left axis deviation. Right axis deviation can also be a normal finding, especially in thin, young individuals. (Answer: c)

— Quick Review 84 —

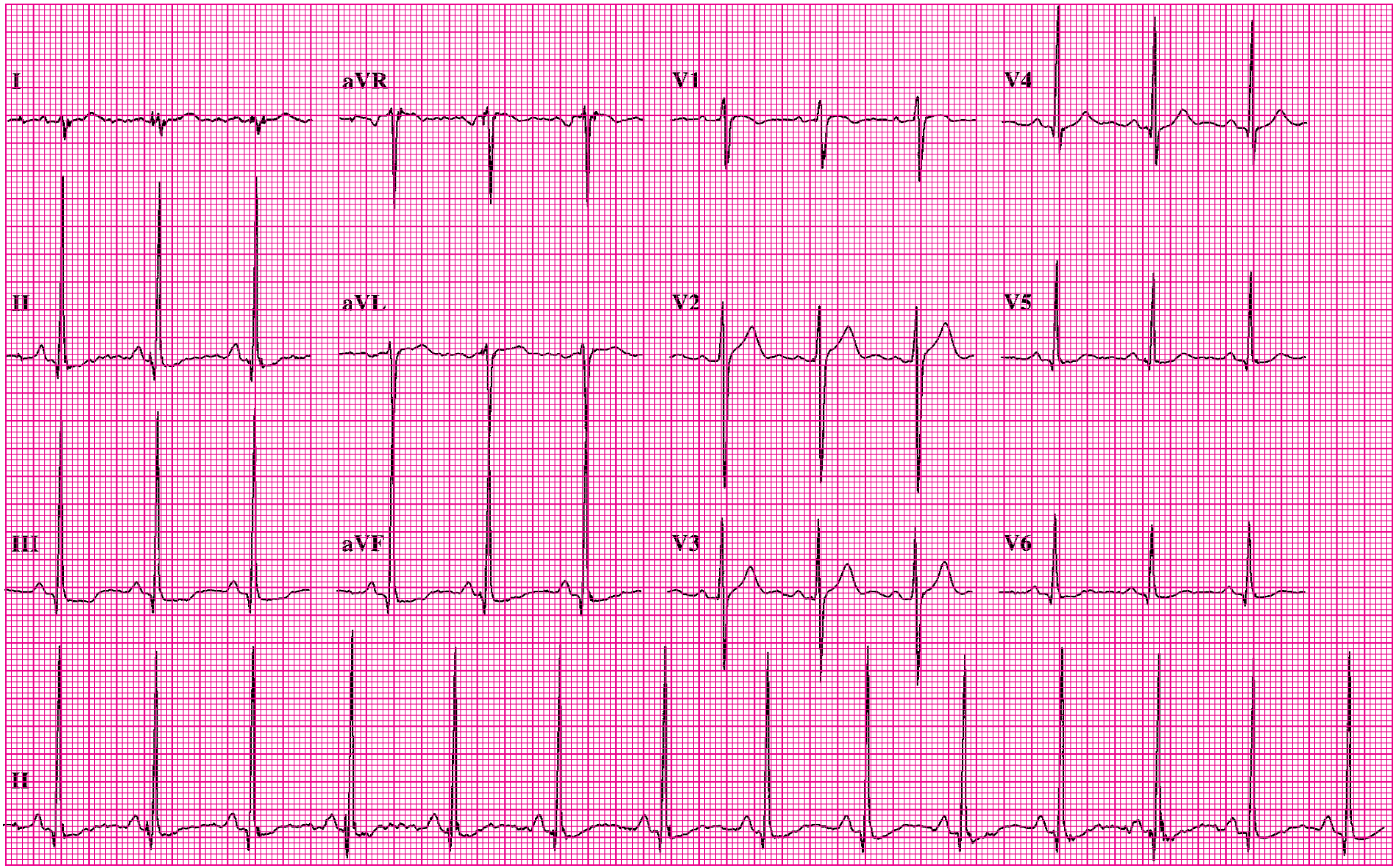
Right ventricular hypertrophy	
• Mean QRS axis ≥ ____ degrees	100
• Dominant ____ wave in V_1:	R
▸ R/S ratio in V_1 or V_{3R} (<, =, >) 1, or R/S ratio in V_5 or V_6 (≤, >) 1	> ≤
▸ R wave in V_1 ≥ ____ mm	7
▸ R wave in V_1 + S wave in V_5 or V_6 > ____ mm	10.5
▸ rSR' in V_1 with R' > ____ mm	10
• Secondary downsloping ST depression & T-wave inversion in the (right/left) precordial leads	right
• (Right/left) atrial abnormality	right
Chronic lung disease	
• (Right/left) ventricular hypertrophy	Right
• (Right/left) axis deviation	Right
• (Right/left) atrial abnormality	Right
• Shift of transitional zone (clockwise/counterclockwise)	clockwise
• (High/low) voltage QRS	low
• Pseudoinfarct pattern in the ____ leads	anteroseptal
• S waves in leads ____ (S_1 S_2 S_3 pattern)	I, II, and III
• May also see sinus tachycardia, junctional rhythm, various degrees of AV block, IVCD, and bundle branch block (true/false)	true

Differential Diagnosis

Right Axis Deviation and a Dominant R Wave in V_1 mimicking Right Ventricular Hypertrophy

- Posterior or inferoposterolateral wall MI
- Right bundle branch block
- Wolff-Parkinson-White syndrome
- Dextrocardia
- Left posterior fascicular block
- Normal variant (especially in children)

ECG 85. 40-year-old male athlete with palpitations:



GENERAL FEATURES

- ☐ 01. Normal ECG
- ☐ 02. Borderline normal ECG or normal variant
- ☐ 03. Incorrect electrode placement
- ☐ 04. Artifact

P WAVE ABNORMALITIES

- ☐ 05. Right atrial abnormality/enlargement
- ☐ 06. Left atrial abnormality/enlargement

SUPRAVENTRICULAR RHYTHMS

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

JUNCTIONAL RHYTHMS

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

VENTRICULAR RHYTHMS

- ☐ 23. Ventricular premature complexes
- ☐ 24. Ventricular parasystole
- ☐ 25. Ventricular tachycardia (≥ 3 consecutive complexes)
- ☐ 26. Accelerated idioventricular rhythm
- ☐ 27. Ventricular escape complexes or rhythm
- ☐ 28. Ventricular fibrillation

AV CONDUCTION ABNORMALITIES

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

ABNORMALITIES OF QRS AXIS

- ☐ 36. Left axis deviation (> –30°)
- ☐ 37. Right axis deviation (> +100°)
- ☐ 38. Electrical alternans

QRS VOLTAGE ABNORMALITIES

- ☐ 39. Low voltage
- ☐ 40. Left ventricular hypertrophy
- ☐ 41. Right ventricular hypertrophy
- ☐ 42. Combined ventricular hypertrophy

INTRAVENTRICULAR CONDUCTION ABNORMALITIES

- ☐ 43. RBBB, complete
- ☐ 44. RBBB, incomplete
- ☐ 45. Left anterior fascicular block
- ☐ 46. Left posterior fascicular block
- ☐ 47. LBBB, complete
- ☐ 48. LBBB, incomplete
- ☐ 49. Nonspecific intraventricular conduction disturbance
- ☐ 50. Functional (rate-related) aberrant intraventricular conduction

Q-WAVE MYOCARDIAL INFARCTIONS

- ☐ 51. Anterolateral (age recent or acute)
- ☐ 52. Anterolateral (age indeterminate or old)
- ☐ 53. Anterior or anteroseptal (age recent or acute)
- ☐ 54. Anterior or anteroseptal (age indeterminate or old)
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- ☐ 56. Lateral (age indeterminate or old)
- ☐ 57. Inferior (age recent or acute)
- ☐ 58. Inferior (age indeterminate or old)
- ☐ 59. Posterior (age recent or acute)
- ☐ 60. Posterior (age indeterminate or old)

REPOLARIZATION ABNORMALITIES

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

SUGGESTED CLINICAL DISORDERS

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

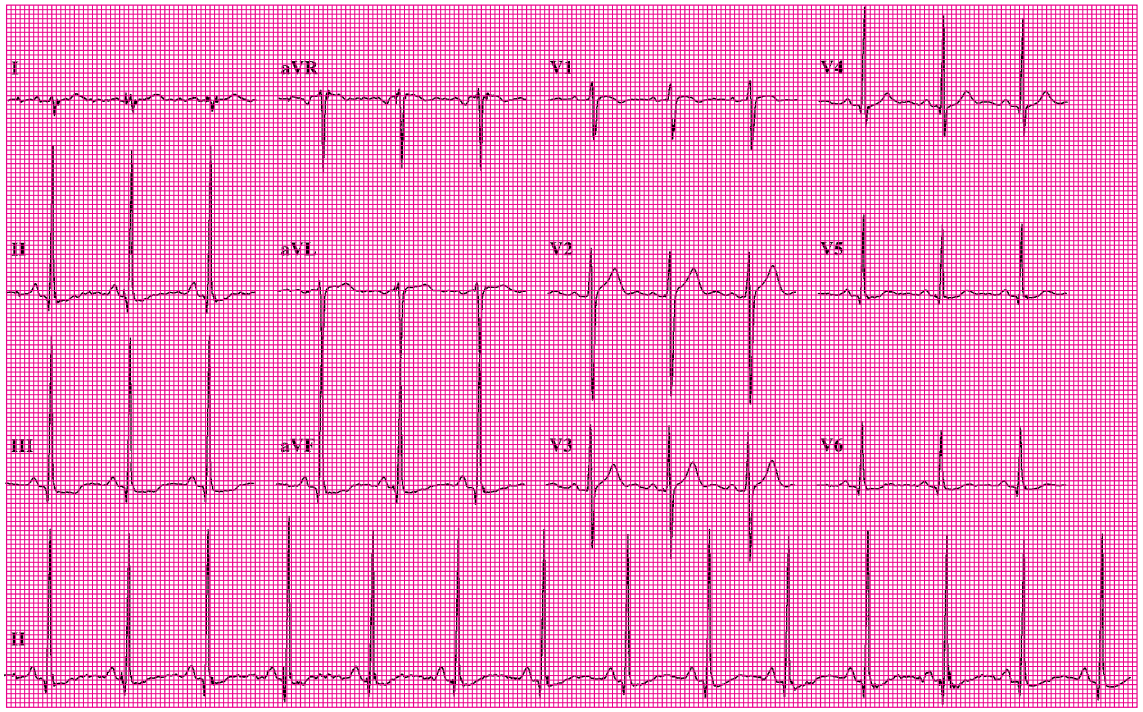
PACED RHYTHMS

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

ECG 85 was obtained from a 40-year-old male athlete with palpitations. The ECG shows normal sinus rhythm at a rate of 77 bpm. LVH with secondary repolarization abnormality is present (S wave in aVR $\geq$ 15 mm; R wave in aVF > 21 mm). The axis is shifted rightward but is < 100° (so right axis deviation should not be coded). Likewise, the P wave in lead II, Q waves in leads II, III, and aVF, and U waves in leads V_2 and V_3 do not meet criteria for right atrial abnormality, Q-wave MI, or prominent U waves. The rightward axis, large P wave in lead II, and prominent inferior voltage are consistent with a vertical heart, thin body habitus, and "physiologic hypertrophy," not uncommon in well-trained athletes.

Codes:

07	Sinus rhythm
40	Left ventricular hypertrophy
67	ST and/or T wave abnormalities secondary to hypertrophy



Questions: ECG 85

1. Causes of right axis deviation include:

   a. Reversal of right and left arm leads
   b. COPD
   c. Horizontal heart
   d. Left posterior fascicular block (LPFB)
   e. Dextrocardia
   f. Ostium primum atrial septal defect (ASD)
   g. Right ventricular hypertrophy (RVH)

2. Factors that reduce the specificity (i.e., increase the rate of false-positives) for the diagnosis of LVH by voltage criteria include:

   a. Severe COPD
   b. Thin body habitus
   c. Obesity
   d. Pericardial or pleural effusion
   e. Coronary artery disease
   f. Pneumothorax
   g. Sarcoidosis or amyloidosis of the heart
   h. Left anterior fascicular block (LAFB)
   i. Severe RVH

Answers: ECG 85

1. Causes of right axis deviation include RVH, vertical heart, COPD, pulmonary embolus, left posterior fascicular block, lateral wall MI, dextrocardia, reversal of right and left arm leads, and ostium secundum ASD. Horizontal hearts and ostium primum ASDs are associated with a leftward shift of the mean QRS axis. (Answer: a, b, d, e, g)

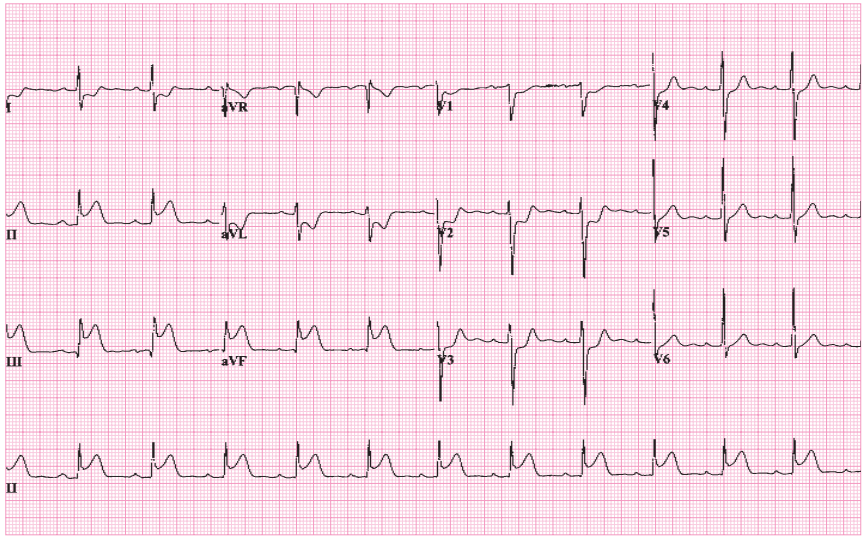
2. Conditions that increase QRS amplitude reduce the *specificity* for LVH by voltage criteria, including thin body habitus and left anterior fascicular block (LAFB increases QRS amplitude in leads I and aVL). Conditions that decrease QRS amplitude reduce the *sensitivity* (i.e., increase the rate of false-negatives) for LVH by voltage criteria, and include conditions that increase the amount of body tissue (obesity), air (COPD, pneumothorax), fluid (pericardial or plural effusion), or fibrous tissue (coronary artery disease, sarcoid or amyloid of the heart) between the myocardium and ECG electrodes. Severe RVH can also underestimate LVH by cancelling prominent QRS forces from the thickened LV. (Answer: b, h)

— Quick Review 85 —

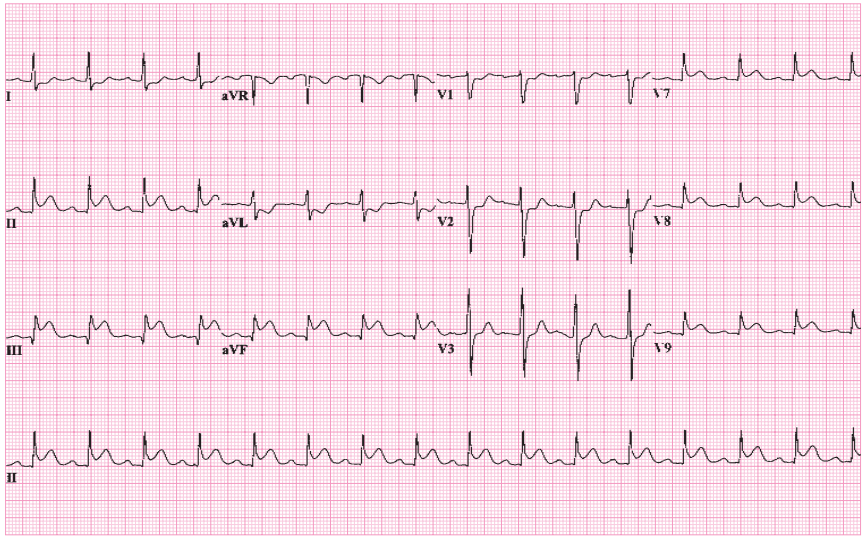
Right axis deviation	
• Mean QRS axis between ____ and ____ degrees	101, 270

ECG 86. 49-year-old male with chest pain:

ECG A



ECG B



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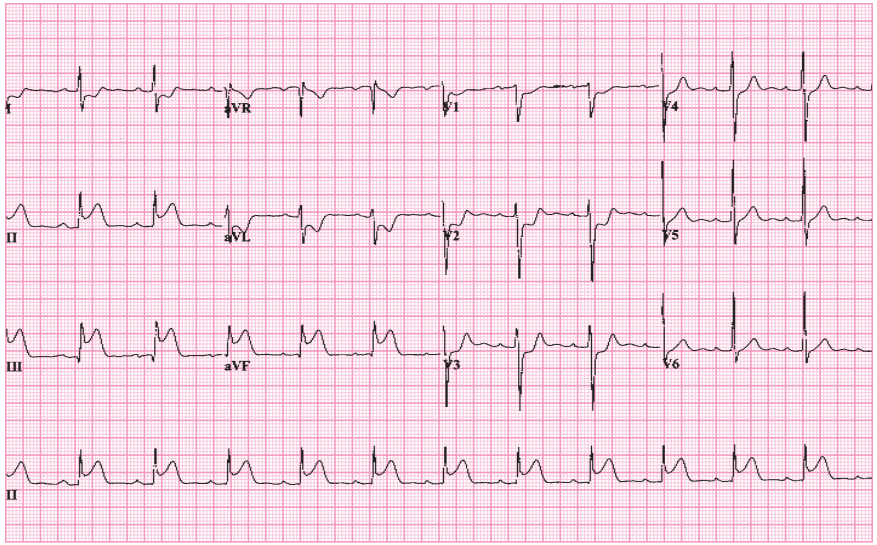
- ☐ 94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)

ECGs 86A and 86B were obtained in a 49-year-old male with chest pain. ECG 86A shows acute inferior injury with ST elevation in leads II, III, and aVF plus diagnostic Q waves in leads III and aVF. There is also ST depression in leads I, aVL and V_1-V_4, which may represent ischemia, reciprocal changes associated with acute inferior injury pattern, or posterior injury. To help identify posterior wall injury/infarction, posterior chest leads are recorded (ECG 86B). To record posterior chest leads V_7-V_9, leads V_4, V_5 and V_6 are placed in the 5th intercostal space (similar to the original V_4-V_6 leads) at the left posterior axillary line (V_7), left midscapular line (V_8), and just left of the spine (V_9). ECG 86B demonstrates ST elevation plus a small Q wave in leads V_7-V_9, consistent with acute posterior infarction. Posterior chest leads should be considered in patients with acute inferior infarction who demonstrate ST depression and upright T waves in leads V_1-V_2.

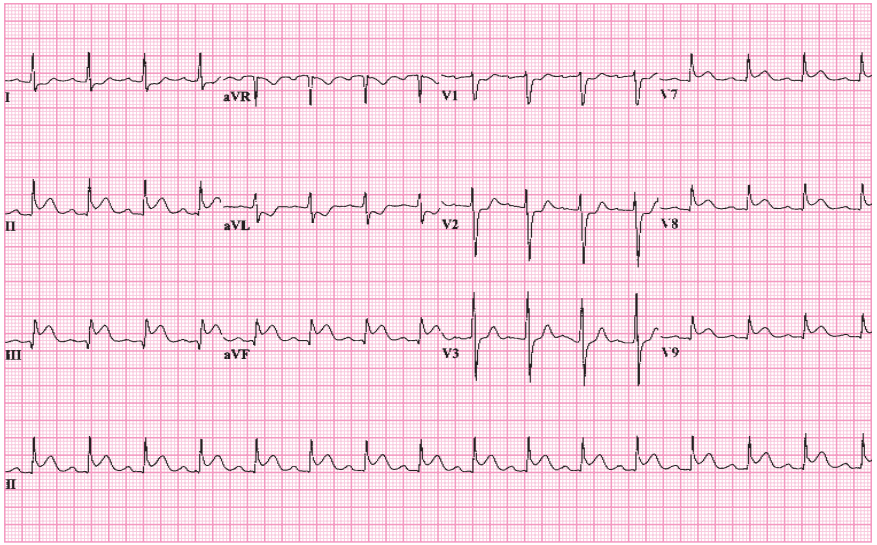
Codes:

07	Sinus rhythm
29	AV block, 1°
57	Inferior (age recent or acute)
59	Posterior (age recent or acute)

ECG A



ECG B



Questions: ECG 86

1. ST depression in leads V_1 and V_2 in a patient with an acute inferior myocardial infarction represents posterior injury when there is:

    a. An inverted T wave in lead V_2
    b. An injury pattern in right-sided chest leads
    c. An upright T wave in lead V_2
    d. Associated ST segment depression in leads I and aVL

2. Which coronary artery is most often involved in isolated posterior MI:

    a. Left anterior descending
    b. Left circumflex artery
    c. Right coronary artery

Answers: ECG 86

1. ST depression in leads V_1 and V_2 is common in the setting of acute inferior myocardial infarction. The mechanism of the ST depression can be anterior ischemia, reciprocal changes, or posterior injury. In this setting, posterior injury is the most common cause of a tall R wave (R > S) and an upright T wave in V_1. However, since none of the 12 ECG leads face the posterior wall, posterior injury is often overlooked/undetected. (Since V_1 records electrical activity from the opposite side from the posterior wall, the large R wave, ST depression, and upright T wave seen in posterior infarction are mirror image reflections of the Q wave, ST elevation, and inverted T wave usually seen in acute MI). ST elevation in posterior chest leads V_7-V_9 identifies patients with larger inferior MI's due to concomitant posterior wall involvement. (Answer: c)

2. Patients with ischemic-type chest pain and no evidence of ST elevation on standard 12-lead ECG benefit from posterior chest lead (V_7-V_9) placement. ST elevations in these leads are associated with cardiac enzyme elevations and posterior wall motion abnormalities on echocardiography in the vast majority of cases; mitral regurgitation is often present as well. On coronary angiography, the culprit vessel is usually the left circumflex artery. (Answer: b)

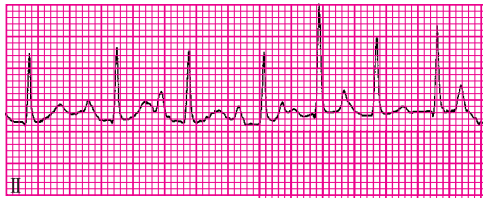
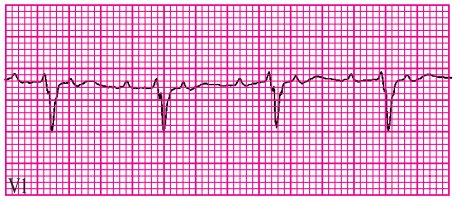
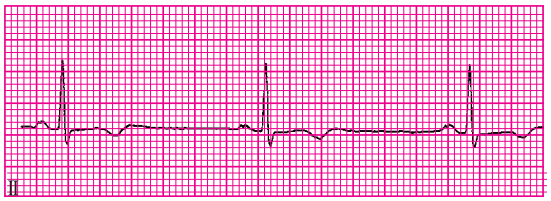
— Quick Review 86 —

AV block, 1°	
• PR interval ≥ ____ seconds	0.20
Posterior MI, recent or probably acute	
• Initial R wave ≥ ____ seconds in leads ____ and ____ with:	0.04, V_1 V_2
▸ R wave amplitude (greater than/less than) S wave amplitude *and* ST segment (elevation/depression) with (upright/inverted) T waves	greater than depression upright
• Posterior MI is usually seen in the setting of acute inferior MI (true/false)	true
• RVH, WPW and RBBB (do/do not) interfere with the ECG diagnosis of posterior MI	do

— POP QUIZ —

Pattern Recognition: Drug Effects and Rhythm Disturbances

Instructions: Choose all drugs commonly associated with each of the following rhythm abnormalities.

ECG	Choose All That Apply	Answer
	a. Amiodarone b. Atropine c. Aminophylline d. Digitalis e. Atorvastatin f. Ramipril g. Nitroglycerin h. Metoprolol i. Verapamil	*Multifocal atrial tachycardia* (MAT) results in an irregular atrial rate > 100 per minute with at least three different P wave morphologies (originating from separate atrial foci) and varying PP and PR intervals. MAT is usually associated with some form of lung disease (COPD, cor pulmonade, hypoxia), and can be precipitated by aminophylline. (Answer: c)
		*Paroxysmal atrial tachycardia (PAT) with block* results in nonsinus P waves at a regular atrial rate (usually 150-240 per minute), isoelectric intervals between P waves, and some nonconducted P waves due to 2° AV block. Digoxin toxicity is responsible for 75% of cases and organic heart disease for 25% of cases. Atropine may worsen Type II 2° AV block, but rarely causes this arrhythmia. Note: 2:1 AV block in this ECG may be either Mobitz Type I or Type II. (Answer: d)
		*Sinus bradycardia* results in a regular sinus (upright P waves in lead II) rhythm at a rate < 60 per minute. Common causes include beta-blockers, amiodarone, verapamil, diltiazem, digitalis, Type I antiarrhythmics, clonidine, α-methyldopa, reserpine, guanethidine, cimetidine, and lithium. Low-dose atropine may also cause a paradoxical slowing of heart rate. (Answer: a, b [low dose], d, h, i)

— POP QUIZ —

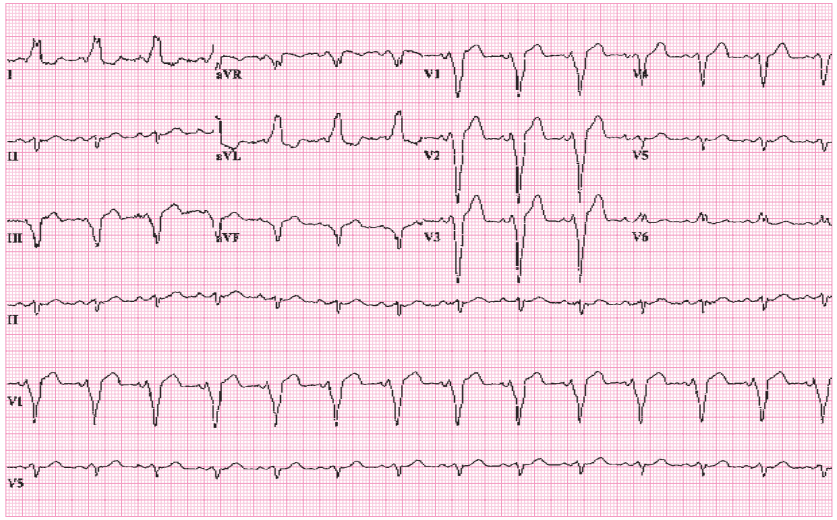
Differential Diagnosis: U-wave

Instructions: Determine whether the diagnoses below are associated with prominent upright U waves, inverted U waves, or both.

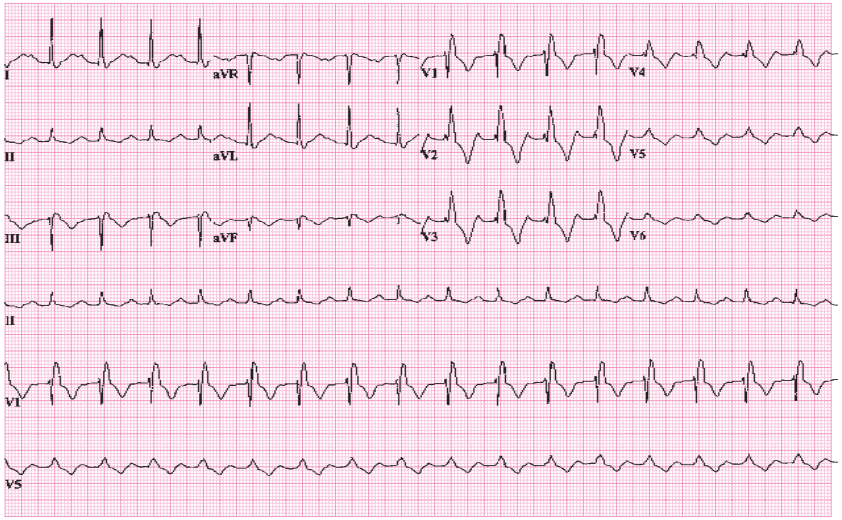
Diagnosis	Answer
Hypokalemia	Prominent upright U waves. ST depression and flattened T waves are common.
Left ventricular hypertrophy (LVH)	Prominent upright or inverted U waves.
Coronary artery disease	Prominent upright or inverted U waves.
Bradycardia	Prominent upright U waves.
Hypothermia	Prominent upright U waves. Osborne (J) waves and prolongation of PR, QRS, and QT are common.
Digoxin	Prominent upright U waves. Sagging ST depression with upward concavity and T wave changes (flat, inverted, or biphasic) are common. QT shortening and PR prolongation may occur.
Antiarrhythmic drugs	Prominent upright U waves (one of earliest findings). Prolonged QT interval and nonspecific ST and T wave changes are common.

ECG 87 (advanced). 29-year-old female with intermittent palpitations and a heart murmur:

ECG A



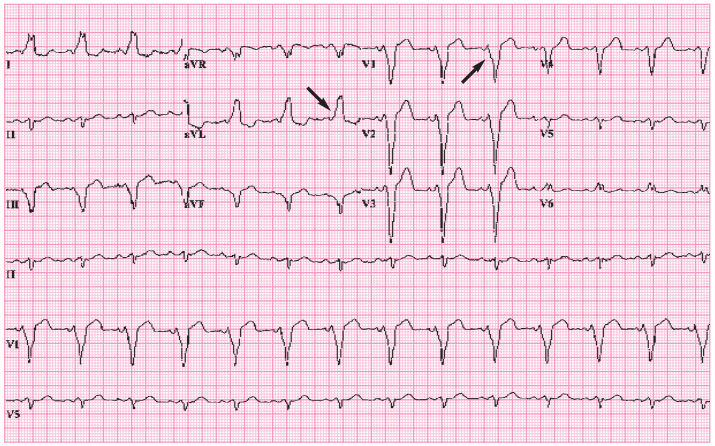
ECG B

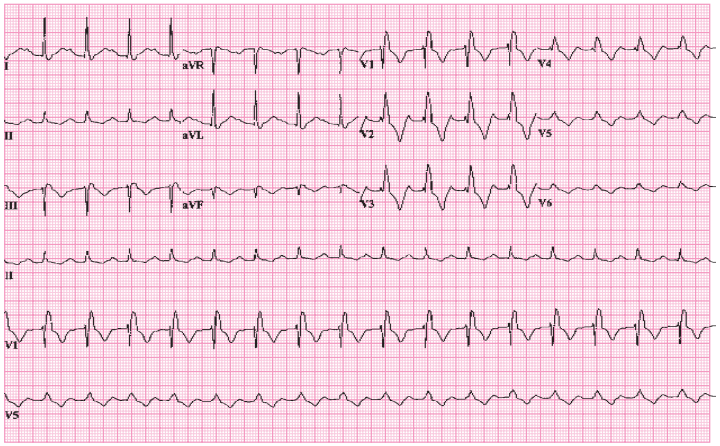


ECGs 87A and 87B were obtained in a 29-year-old female with intermittent palpitations and a heart murmur and are associated with a unique clinical presentation. This patient has Ebstein's anomaly with ventricular pre-excitation (Wolff-Parkinson-White pattern). In ECG 87A, pre-excitation is present with an accessory pathway connecting the right atrium and right ventricle. This gives rise to a left bundle branch block (LBBB) pattern since both ventricles are activated over the right-sided accessory pathway. The PR interval is short, a delta wave is present, and the QRS complex is prolonged, consistent with WPW pattern. In ECG 87B, pre-excitation is no longer present, and the more typical right bundle branch block (RBBB) pattern of conduction delay associated with Ebstein's anomaly is now apparent. The ventricle is activated by the electrical impulse traveling through the AV node and the His-Purkinje pathways, and Ebstein's anomaly has resulted in abnormal conduction through the right ventricle, consistent with a RBBB conduction pattern. First-degree AV block and ST-T changes suggestive of inferior wall ischemia are also evident. Ventricular pre-excitation due to a right-sided accessory AV pathway should be suspected whenever a patient with Ebstein's anomaly has an ECG showing absence of the expected RBBB pattern and instead shows normal or shortened AV conduction with a LBBB conduction pattern.

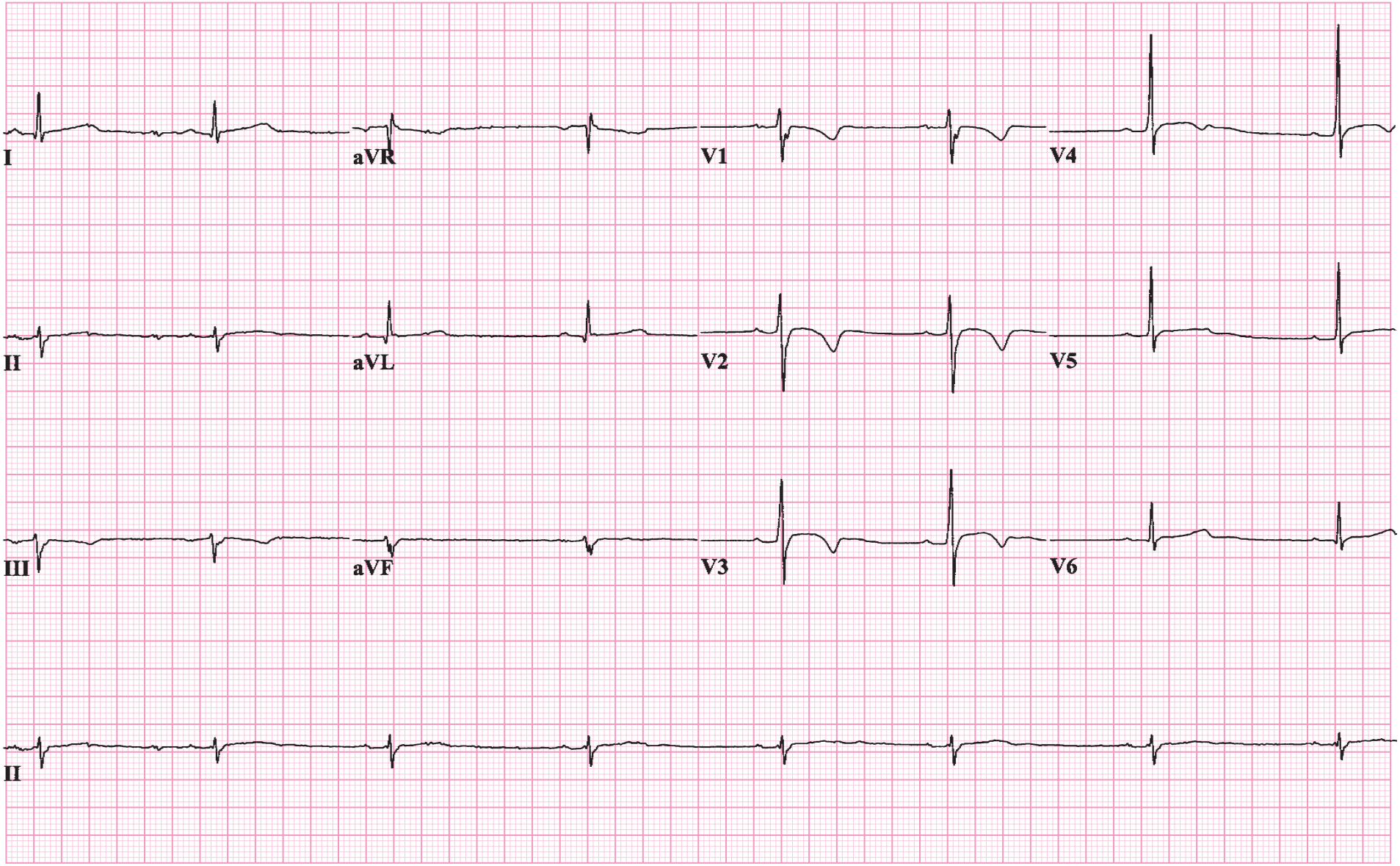
Codes:

ECG 87A.	06	Left atrial abnormality/enlargement
	07	Sinus rhythm
	34	Wolff-Parkinson-White pattern
ECG 87B.	10	Sinus tachycardia (> 100)
	29	AV block, 1°
	43	RBBB, complete
	64	ST and/or T wave abnormalities suggesting myocardial ischemia





ECG 88 (advanced). 30-year-old male with palpitations and syncope:



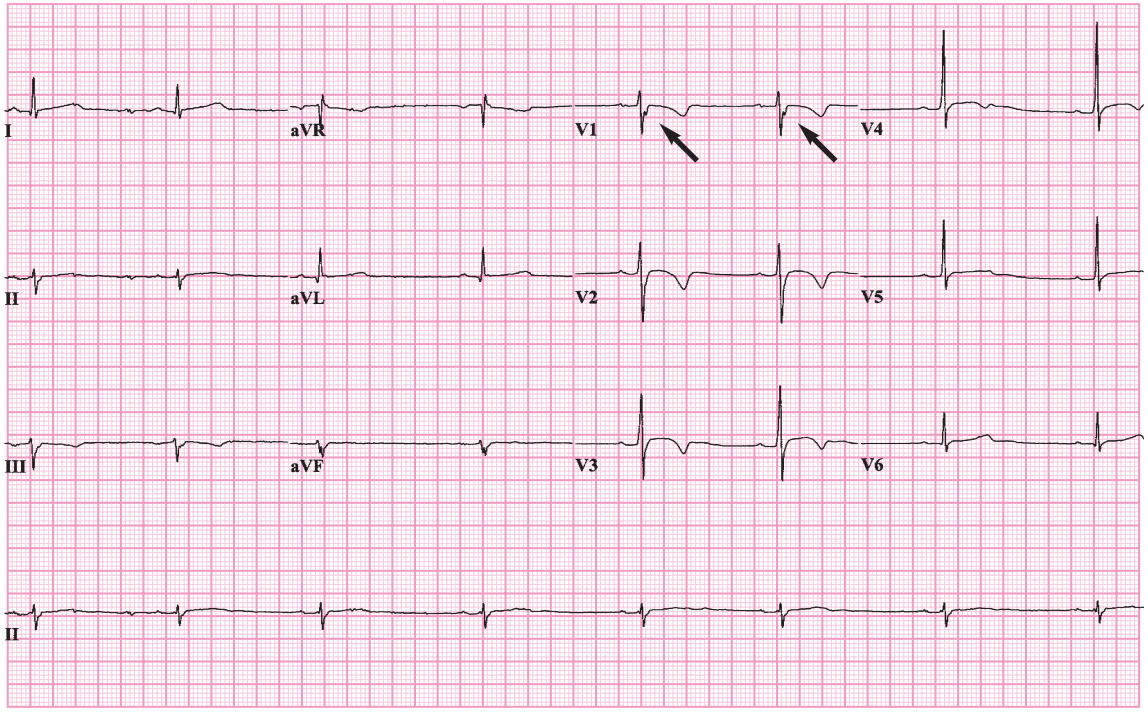
ECG 88 was obtained in a 30-year-old male with palpitations and syncope. The ECG shows the characteristic pattern for the unique clinical presentation of arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C). ECG findings consistent with ARVD/C include the epsilon wave in lead V_1 (arrow), a sharp deflection at the end of the QRS complex, and the inverted T waves in leads V_1-V_3. Patients with ARVD/C develop a cardiomyopathy of the right ventricle that at times can involve the left ventricle. The ventricular myocardium is replaced with fibrous and fatty tissue. These patients can develop heart failure and ventricular tachycardia due to reentry circuits occurring in the scarred right ventricle. The tachycardias are most commonly monomorphic ventricular tachycardia with a left bundle branch block morphology (since they originate in the right ventricle). Tachycardias arising near the right ventricular outflow tract will have a left bundle branch block, right axis (inferior axis) morphology; tachycardias originating from the mid-part of the right ventricle will have a left bundle branch block, normal axis morphology; and tachycardias originating from the right ventricular apex will have a left bundle branch block, left axis (superior axis) morphology. Other ECG findings include profound sinus bradycardia at 45 beats/minute and left axis deviation.

Codes:

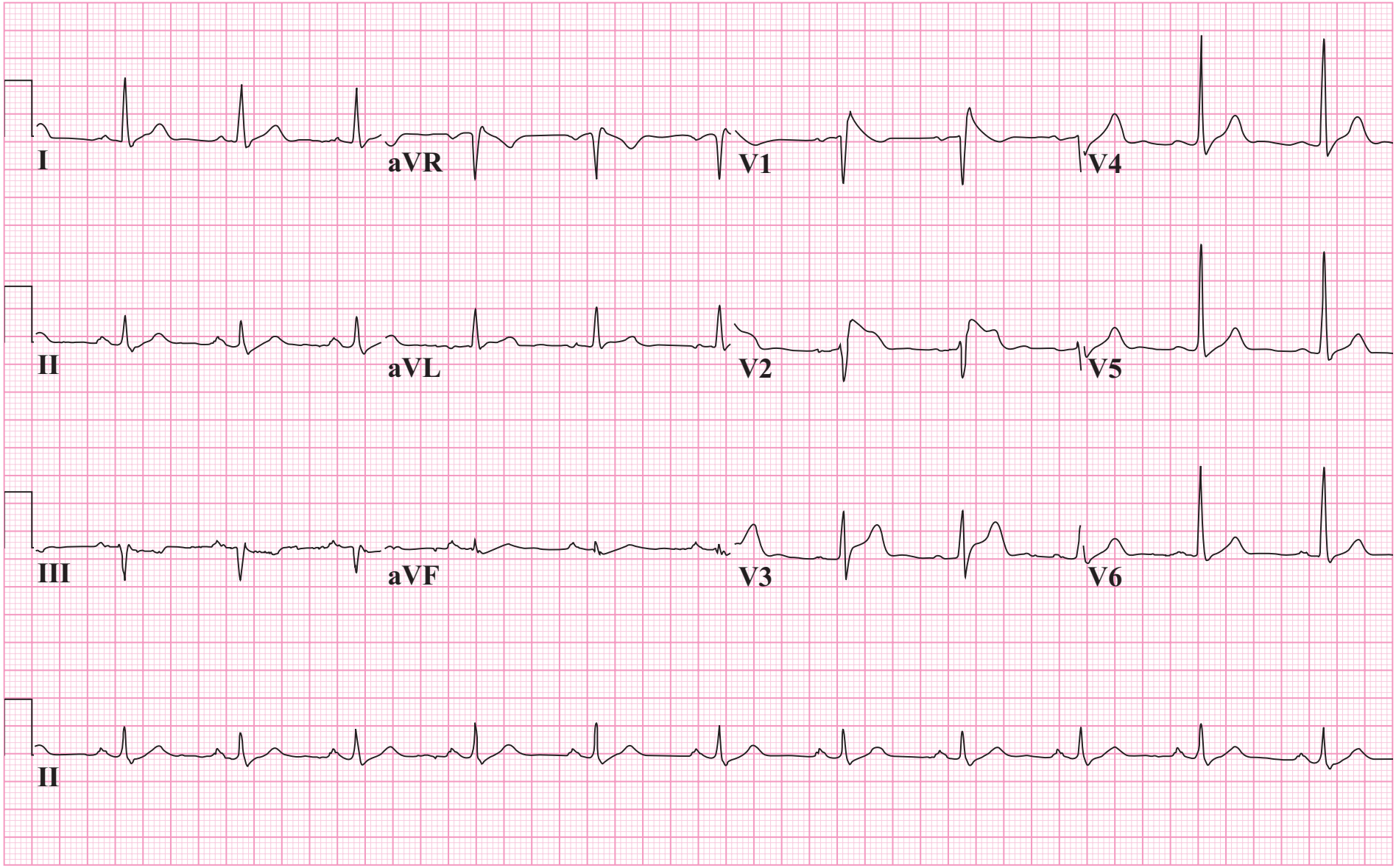
09 Sinus bradycardia (< 60)

37 Right axis deviation (> + 100°)

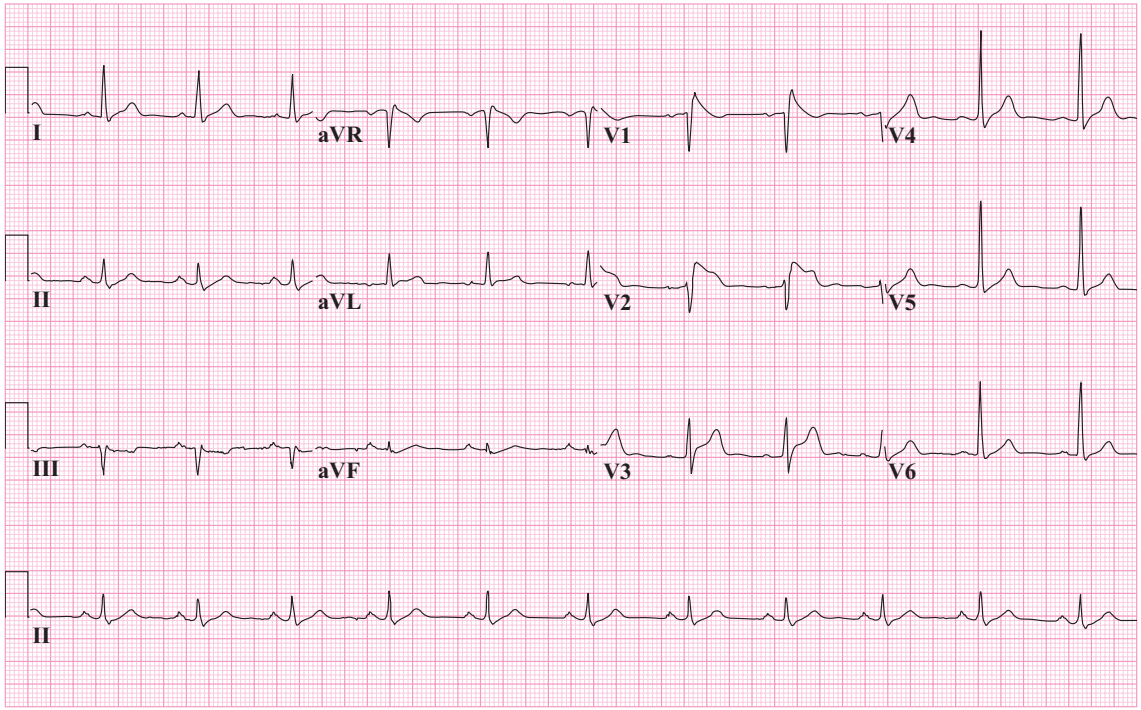
64 ST-T and/or T wave abnormalities suggesting myocardial ischemia



ECG 89 (advanced). 41-year-old male on routine ECG prior to elective surgery:



ECG 89 was obtained in a 41-year-old male on routine ECG prior to elective surgery. The ECG is associated with a distinctive QRS complex in leads V_1 and V_2 consistent with the *Brugada pattern of conduction*. Patients are considered to have the unique clinical presentation of the *Brugada Syndrome* when this ECG pattern is associated with ventricular tachycardia, which can result in syncope or sudden cardiac death. The *Brugada pattern* involves a depolarization abnormality characterized by an R' and ST elevation in lead V_1 and often in lead V_2. This ECG pattern is associated with a genetic defect of the SCN5A gene, which affects the sodium channel and is associated with life-threatening ventricular arrhythmias and sudden death in otherwise healthy individuals.



Section 4

ECG CRITERIA

(diagnoses are listed in order of appearance on answer sheet)

General Features

01. Normal ECG (no abnormalities of rate, rhythm, axis or P-QRS-T):

P Wave

Duration: 0.08 - 0.11 seconds

Axis: 0 - 75^0

Morphology: Upright in I, II; upright or inverted in aVF; inverted or biphasic in III, aVL, V_1, V_2; small notching may be present

Amplitude: Limb leads < 2.5 mm; V_1: positive deflection < 1.5 mm and negative deflection < 1 mm

PR Interval

Duration: 0.12 - 0.20 seconds

PR segment: Usually isoelectric; may be displaced in a direction opposite to the P wave; elevation is usually < 0.5 mm; depression is typically < 0.8 mm

QRS Complex

Duration: 0.06 - 0.10 seconds

Axis: -30^0 to $+105^0$

Transition zone (precordial leads with equal positive and negative deflection): V_2-V_4

Q wave: Small Q waves (duration < 0.04 seconds and amplitude < 2 mm) are common in most leads except aVR, V_1 and V_2

Onset of intrinsicoid deflection (beginning of QRS to peak of R wave): Right precordial leads < 0.035 seconds; left precordial leads < 0.045 seconds

ST Segment

Usually isoelectric. In limb leads, may vary from 0.5 mm below to 1 mm above baseline; in V_2 - V_3 (sometimes V_4) up to 3 mm concave upward elevation in precordial leads may be seen in young adults (early repolarization, item 61), but is usually < 2 mm if over age 40; in V_5-V_6 concave upward elevation more than 1 mm is uncommon

T Wave

Morphology: Upright in I, II, V_3-V_6; inverted in aVR, V_1; may be upright, flat or biphasic in III, aVL, aVF, V_1, V_2; T wave inversion may be present in V_1-V_3 in healthy young adults (juvenile T waves, item 62)

Amplitude: Usually < 6 mm in limb leads and < 10 mm in precordial leads

QT Interval

Corrected QT (QT interval divided by the square root of the RR interval) = 0.30 - 0.44 seconds; varies inversely with heart rate

U Wave

Morphology: Upright in all leads except aVR

Amplitude: 5-25% the height of the T wave (usually < 1.5 mm)

02. Borderline normal ECG or normal variant

- Early repolarization (item 61)
- Juvenile T waves (item 62)
- S wave in leads I, II, and III (S_1 S_2 S_3 pattern)
  Note: Present in up to 20% of healthy young adults.
- RSR' or rSr' in lead V_1 with QRS duration < 0.10 seconds, r wave amplitude < 7 mm, and r' amplitude smaller than r or S waves
  Note: Seen in 2% of normals, but can also be seen in:
  - RVH (item 41)
  - Posterior MI (items 59, 60)
  - Skeletal deformities (pectus excavatum, straight back syndrome)

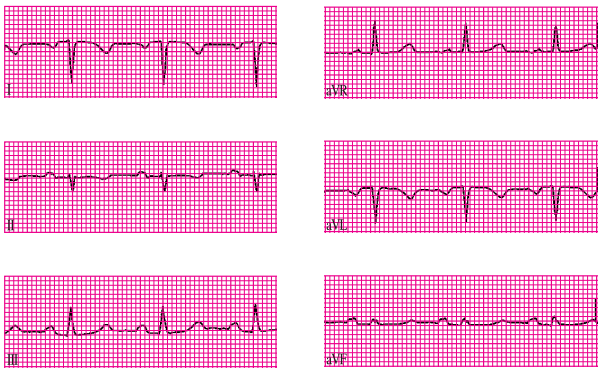
- ▸ High electrode placement of V_1 (in 3rd intercostal space instead of 4th)
- Tall P waves
- Notched P waves of normal duration

Note: Hyperventilation may cause prolonged PR, sinus tachycardia, and ST depression ± T wave inversion (usually seen in inferior leads).

Note: Large food intake may cause ST depression and/or T wave inversion, especially after a high carbohydrate meal.

03. Incorrect electrode placement

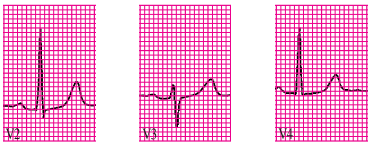
Limb lead reversal:



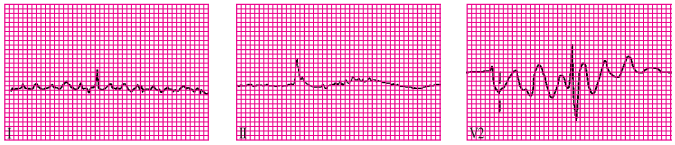
- Reversal of right and left arm leads
  - ▸ Resultant ECG mimics dextrocardia in limb leads with inversion of the P-QRS-T in leads I and aVL
  - ▸ Leads II and III transposed
  - ▸ Leads aVR and aVL transposed

  Note: To distinguish between these conditions, look at precordial leads: dextrocardia shows reverse R wave progression (with gradual loss of R wave voltage from V_1 -V_6); limb lead reversal shows normal R wave progression.
- Reversal of left arm and left leg leads
  - ▸ Leads I and II transposed
  - ▸ Leads aVF and aVL transposed
  - ▸ Lead III inverted
- Reversal of right arm and left leg leads
  - ▸ Leads I, II, and III inverted
  - ▸ Leads aVR and aVF transposed

Precordial lead reversal: Typically manifests as an unexplained decrease in R wave voltage in two consecutive leads (e.g., V_1, V_2) with a return to normal R wave progression on the following leads



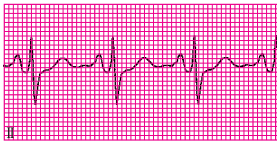
04. Artifact



- **AC electrical interference** (60 cycles per seconds): Due to an unstable or dry electrode, poor grounding of the ECG machine, or excessive current leak from an ECG machine too close to other electronic equipment. Rapid sine-wave changes make assessment of P waves and ST segment shifts unreliable.
- **Wandering baseline:** Due to an unstable electrode, deep respirations, or uncooperative patient. Evaluation of P waves, QRS voltage, and ST segment shifts are unreliable.
- **Skeletal muscle fasciculations** (e.g., shivering, anxiety with muscle tension)
- Commonly due to **tremor** (most prominent in limb leads)
  - ▸ Parkinson's tremor simulates atrial flutter with a rate of ~ 300 per minute (4-6 cycles per second)
  - ▸ Physiologic tremor rate is 500 per minute (7-9 per second)
- **Poor standardization:** 1 mV signal is not recorded, underdamped, or overdamped; ECG recorded at half-standard or double-standard. Voltages may be inaccurate.
- **ECG recorded at double-speed or half-speed**
- **Rapid arm motion** or lead movement (e.g., brushing teeth or hair): Can simulate VPCs or ventricular tachycardia; often mistaken for ventricular tachycardia on telemetry or Holter monitoring.
- **Cautery:** Pronounced baseline interference
- **IV infusion pump:** May give appearance of rapid P waves

P Wave Abnormalities

05. Right atrial abnormality/enlargement



- Tall upright P wave:
  - > 2.5 mm in leads II, III, and aVF (P-pulmonale), *or*
  - > 1.5 mm in leads V_1 or V_2
- P wave axis shifted rightward (i.e., axis ≥ 70°)

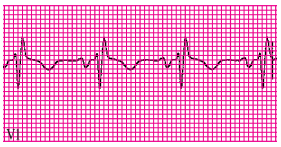
Note: In up to 30% of cases, P pulmonale may actually represent left atrial enlargement. Suspect this possibility when left atrial abnormality/enlargement (item 05) is present in lead V_1.

Note: Prominent atrial repolarization waves (Ta) can mimic Q waves and ST depression by deforming the PR and ST segments, respectively.

Note: P pulmonale can be seen in

- COPD with or without cor pulmonale (item 81)
- Pulmonary hypertension
- Congenital heart disease (such as pulmonic stenosis, Tetralogy of Fallot, tricuspid atresia, Eisemenger's physiology)
- Pulmonary embolism (usually transient) (item 82)
- Normal variant in patients with a thin body habitus and/or vertical heart

06. Left atrial abnormality/enlargement



- Terminal negative portion of the P wave in lead V_1 ≥ 1 mm deep and ≥ 0.04 seconds in duration (i.e., one small box deep and one small box wide), *or*
- Notched P wave with a duration ≥ 0.12 seconds in leads II, III or aVF (P-mitrale)

Note: Left atrial enlargement by echocardiography can exist with a normal P wave, and P mitrale may be present in the absence of left atrial enlargement.

Note: Prominent atrial repolarization waves (Ta) can mimic Q waves and ST depression by deforming the PR and ST segments, respectively.

Note: Mechanisms responsible for P mitrale include left atrial hypertrophy or dilation, intraatrial conduction delay, increased left atrial volume, and an acute rise in left atrial pressure.

Note: Can be seen in:

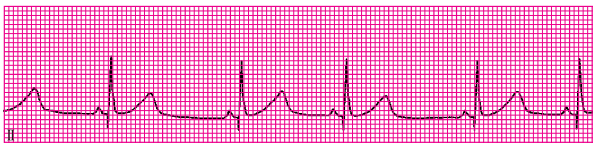
- Mitral valve disease
- Organic heart disease
- Aortic valve disease
- Heart failure
- Myocardial infarction
- Hypertension/LVH

Supraventricular Rhythms

07. Sinus rhythm

- Normal P wave axis and morphology
- Atrial rate is 60-100 per minute and regular (PP interval varies by < 0.16 seconds or < 10%)

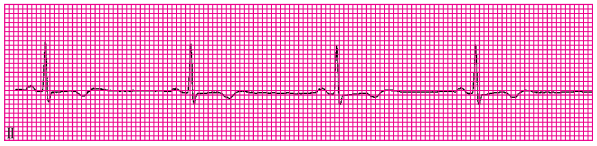
08. Sinus arrhythmia



- Normal P wave morphology and axis
- Phasic change in PP interval (onset may sometimes occur abruptly), usually in response to the breath cycle
- Longest and shortest PP intervals vary by > 0.16 seconds or 10%

Note: Sinus arrhythmia is a major factor in beat-to-beat heart rate variability (HRV). The presence of maintained HRV is a manifestation of active, healthy, vagal tone, and an important marker for good cardiovascular prognosis.

09. Sinus bradycardia (< 60)



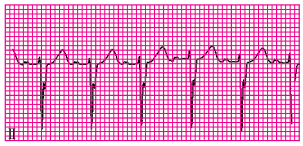
- Normal P wave axis and morphology
- Rate < 60 per minute

Note: If the atrial rate is < 40 per minute, think of 2:1 sinoatrial exit block (item 12)

Note: Causes include:

- High vagal tone (normals, especially during sleep; trained athletes; Bezold-Jarisch reflex; inferior MI, pulmonary embolism)
- Myocardial infarction (usually inferior)
- Drugs (beta-blockers, verapamil, diltiazem, digitalis, Type IA, IB, IC antiarrhythmics, amiodarone, sotalol, clonidine, α-methyldopa, reserpine, guanethidine, lithium)
- Hypothyroidism (item 87)
- Hypothermia (item 88)
- Obstructive jaundice
- Hyperkalemia (item 74)
- Increased intracranial pressure (item 86)
- Sick sinus syndrome (item 89)

10. Sinus tachycardia (> 100)



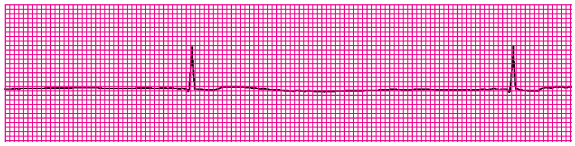
- Normal P wave axis and morphology
- Rate > 100 per minute

Note: P wave amplitude often increases and PR interval often shortens with increasing heart rate (e.g., during exercise)

Note: Causes include:

- Physiologic response to stress (exercise, anxiety, pain, fever, hypovolemia, hypotension, anemia)
- Thyrotoxicosis
- Myocardial ischemia/infarction
- Heart failure
- Myocarditis
- Pulmonary embolism (item 82)
- Pheochromocytoma
- AV fistula
- Drugs (caffeine, alcohol, nicotine, cocaine, amphetamines, albuterol and other beta-agonists, endogenous catecholamines, hydralazine, exogenous thyroid, atropine, aminophylline)

11. Sinus pause or arrest



- PP interval (pause) greater than 1.6-2.0 seconds
- Sinus pause is not a multiple of the basic sinus PP interval

  Note: If sinus pause is a multiple of the basic PP interval, consider sinoatrial exit block (item 12).

Note: Sinus pauses must be differentiated from:

- Sinus arrhythmia (item 08): Phasic, gradual change in PP interval
- Second-degree sinoatrial block, Mobitz I (Wenckebach) (item 12): Progressive shortening of PP interval until a P wave fails to appear
- Second-degree sinoatrial block, Mobitz II (item 12): Sinus pause is a multiple (e.g., 2x, 3x, etc.) of the basic sinus rhythm (PP interval)
- Abrupt change in autonomic tone (e.g., vagal reaction)
- "Pseudo" sinus pause due to nonconducted atrial premature complexes (APC; item 13): P wave appears to be absent but is actually buried in the T wave — look for subtle deformity of the T wave at the beginning of the pause to detect nonconducted APCs

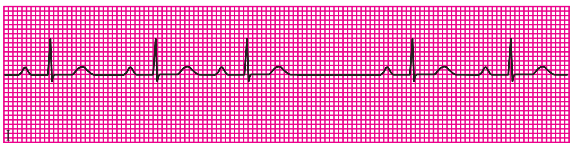
Note: Complete failure of sinoatrial conduction (third-degree sinoatrial block; item 12) cannot be differentiated from complete sinus arrest on surface ECG

Note: Sinus pause/arrest is due to transient failure of impulse formation at the SA node. Etiology is the same as sinoatrial exit block (item 12).

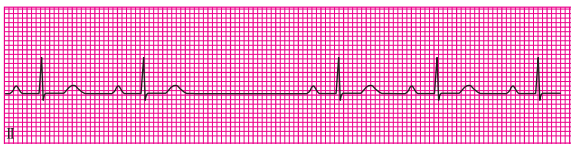
12. Sinoatrial exit block

- **SECOND-DEGREE:** Some sinus impulses fail to capture the atria, resulting in the intermittent absence of a P wave. Often a component of the Sick Sinus Syndrome (item 89)

- Type I (Mobitz I) sinoatrial exit block:



  - P wave morphology and axis consistent with a sinus node origin
  - "Group beating" with:
    (1) Shortening of PP interval up to pause
    (2) Constant PR interval
    (3) PP pause < 2x the normal PP interval
- Type II (Mobitz II) sinoatrial exit block

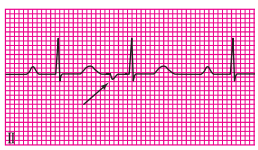


  - Constant PP interval followed by a pause that is a multiple (e.g., 2x, 3x, etc.) of the normal PP interval
  - The pause may be slightly less than twice the normal PP interval (usually within 0.10 seconds).

  Note: Causes include:
  - Drugs (digitalis, quinidine, flecainide, propafenone, procainamide)
  - Hyperkalemia (item 74)
  - Sinus node dysfunction
  - Organic heart disease
  - Myocardial infarction
  - Vagal stimulation

Note: First-degree sinoatrial exit block (conduction of sinus impulses to the atrium is delayed, but 1:1 response is maintained) is not detectable on surface ECG, and third-degree sinoatrial exit block (complete failure of sinoatrial conduction) cannot be differentiated from complete sinus arrest (item 11)

13. Atrial premature complexes



- P wave that is abnormal in configuration and premature relative to the normal PP interval
- QRS complex is usually similar in morphology to the QRS complex present during sinus rhythm. Exceptions include:
  - Aberrantly conducted APCs: QRS may be wide and bizarre; more likely to occur with very premature APCs. QRS morphology is most often RBBB pattern (due to the longer refractory period of the right bundle compared to the left bundle), but can be LBBB pattern or variable.
  - Blocked APCs: Very premature P wave not followed by a QRS complex. P waves are often hidden in the preceding T wave — look for a deformed T wave immediately after the first QRS of the RR pause to identify the presence of a nonconducted atrial premature complex.
- The PR interval may be normal, increased, or decreased.
- The post-extrasystolic pause is usually *noncompensatory* (i.e., the interval from the preceding normal P wave to the normal P wave following the APC is less than two normal PP intervals). However, an interpolated APC or a compensatory pause may be evident when sinoatrial (SA) "entrance block" is present and the SA node is not reset.

Note: Can be seen in normals, fatigue, stress, smoking, drugs (including caffeine and alcohol), organic heart disease, cor pulmonale

14. Atrial parasystole

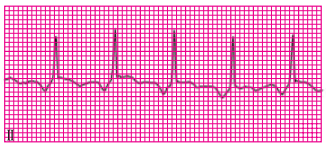
- Frequent atrial premature complexes of similar morphology that "march through" the tracing independent of the underlying sinus rhythm.
- Interectopic intervals are a multiple (2x, 3x, etc.) of the shortest interectopic interval (since the parasystolic focus fires at a regular rate and inscribes a P wave whenever the atria are not refractory)
- Resultant ectopic atrial complex varies in relationship to the preceding sinus beats (i.e., nonfixed coupling)

Note: Exit block from a parasystolic focus may occur and result in absence of an atrial ectopic beat when it would otherwise be expected to occur.

Note: Atrial parasystole is due to the presence of an ectopic atrial focus that activates the atria independent of the basic sinus rhythm, and is protected from depolarization by an entrance block. The atrial focus fires at a regular cycle length and results in an ectopic atrial beat that bears no constant relationship (nonfixed coupling) to the previous sinus beat.

Note: Think of atrial parasystole in the presence of atrial premature complexes of similar morphology with nonfixed coupling.

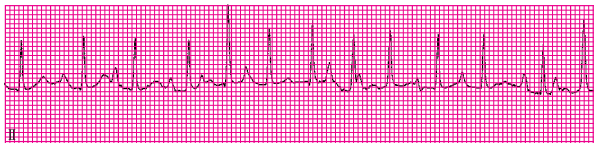
15. Atrial tachycardia



- Three or more consecutive ectopic atrial beats (nonsinus P waves) at an atrial rate of 100-240 per minute
- P wave may precede, be buried in (sometimes not visualized), or immediately follow the QRS complex
- QRS complex follows each P wave unless second- or third-degree AV block is present. Atrial tachycardia with block may be confused with atrial flutter. Atrial tachycardia with block has a distinct isoelectric baseline between P waves, atrial flutter does not (except occasionally in lead V_1). Atrial tachycardia with block is secondary to digitalis toxicity (item 71) in 75% and organic heart disease in 25%
- QRS morphology is usually narrow and resembles QRS morphology during sinus rhythm, but can be wide (if underlying bundle branch block or aberrancy)

  Note: Automatic atrial tachycardia and intraatrial reentrant tachycardia account for 10% of SVTs. Carotid sinus massage produces AV block but does not terminate the tachycardia. Nonsustained form is common in normals; the sustained form is more common in organic heart disease.

16. Atrial tachycardia, multifocal



- Atrial rate >100 per minute
- P waves with ≥ 3 morphologies (each originating from a separate atrial focus)
- Varying PP and PR intervals
- P waves may be blocked (i.e., not followed by a QRS complex), or may be conducted with a narrow or wide (if underlying bundle branch block or aberrancy) QRS complex.

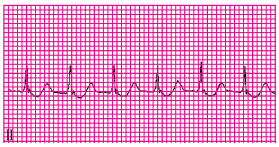
Note: Multifocal atrial tachycardia may be confused with:

- Sinus tachycardia with multifocal APCs, which demonstrates one dominant atrial pacemaker (i.e., the sinus node). In contrast, in multifocal atrial tachycardia, *no* dominant atrial pacemaker (i.e., no dominant P wave morphology) is present.
- Atrial fibrillation/flutter, in which there is lack of an isoelectric baseline. In contrast, multifocal atrial tachycardia demonstrates a distinct isoelectric baseline and P waves.

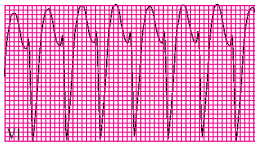
Note: Usually associated with some form of lung disease. Etiologies include:

- COPD/pneumonia
- Cor pulmonale
- Aminophylline therapy
- Hypoxia
- Organic heart disease
- Heart failure
- Post-op
- Sepsis
- Pulmonary edema

17. Supraventricular tachycardia, paroxysmal



Without aberrancy



With aberrancy

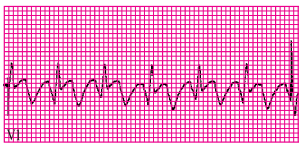
- Regular rhythm
- Rate >100 per minute
- P waves not easily identified
- QRS complex is usually narrow (but occasionally wide if underlying bundle branch block or aberrancy)
- Onset and termination of SVT is sudden, and SVT does not persist throughout the entire tracing
- Retrograde atrial activity may be present

Note: If rate is approximately 150 per minute, atrial flutter with 2:1 block may be present. Look for typical "sawtooth" flutter waves in inferior leads (II, III, aVF) or V_1; every other flutter wave may be buried in the QRS complex or ST segment.

Note: There are several different types of supraventricular tachycardia, the majority of which cannot be differentiated by surface ECG alone and may require an EP study to differentiate:

- AV nodal reentrant tachycardia accounts for 60-70% of SVTs, and is usually initiated by an APC. Reentry occurs in the AV node, with antegrade conduction down the slow (α) AV nodal pathway and retrograde conduction up the fast (β) AV nodal pathway. Carotid sinus massage slows and frequently terminates tachycardia. Occurs commonly in normals.
- Atypical AV nodal reentrant tachycardia accounts for 5-10% of AV node reentry and 2-5% of SVTs. Reentry circuit in AV node with antegrade conduction down the rapid (β) AV node pathway and retrograde conduction up the slow (α) pathway. May require an EP study to diagnose. Carotid sinus massage may terminate the tachycardia.
- AV reentrant tachycardia (orthrodromic SVT) occurs with Wolff-Parkinson-White syndrome and concealed bypass tracts. The hearts are usually normal in these conditions, but WPW can be associated with Ebstein's anomaly, cardiomyopathy, or mitral valve prolapse. Usually a short RP SVT, but can have a long RP interval and be incessant if there is slow retrograde (VA) conduction. Often initiated by APCs, and usually terminates suddenly with carotid sinus massage.
- In contrast to the other forms of atrial tachycardia, sinus node reentrant tachycardia manifests *sinus* P waves and is indistinguishable from sinus tachycardia. It involves reentry in or around the sinus node, and accounts for < 5% of SVTs. Carotid sinus massage produces AV block, but does not terminate the tachycardia. Occasionally seen in normals, but more common in organic heart disease.

18. Atrial flutter



- Rapid regular atrial undulations (flutter or "F" waves) usually at a rate of 240-340 per minute

  Note: Flutter rate may be faster (> 340 per minute) in children and slower (200-240 per minute) in the presence of antiarrhythmic drugs (Type IA, IC, III) and/or massively dilated atria.

  Note: ECG artifact due to Parkinsonian tremor (~ 4-6 cycles second) can simulate flutter waves. Look for evidence of distinct superimposed P waves preceding each QRS complex, especially in leads I, II, or V_1.
- Typical atrial flutter morphology is usually present:
  - Leads II, III, AVF: Inverted F waves without an isoelectric baseline ("picket-fence" or "sawtooth" appearance)
  - Lead V_1: Small positive deflections usually with a distinct isoelectric baseline
- Atypical atrial flutter can exhibit upright F waves in inferior leads
- QRS complex may be normal or wide (if underlying bundle branch block or aberrancy)
- Rate and regularity of QRS complexes depend on the AV conduction sequence
  - AV conduction ratio (ratio of flutter waves to QRS complexes) is usually fixed and an even number (e.g., 2:1, 4:1), but may vary.

    Note: Odd-numbered conduction ratios of 1:1 and 3:1 are uncommon. Atrial flutter with 1:1 AV conduction often conducts aberrantly, resulting in a wide QRS tachycardia that may be confused with VT. In untreated patients, ≥ 4:1 block suggests the coexistence of AV conduction disease.

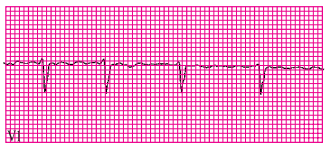
    Note: Carotid sinus massage typically causes a transient increase in AV block and slowing of the ventricular response, without a change in the atrial flutter rate. At times, no effect is seen. When atrial flutter with 2:1 AV block is suspected, carotid sinus massage may unmask flutter waves and help confirm the diagnosis. Upon discontinuation of carotid sinus massage, the usual response is return to the original ventricular rate.
  - Complete heart block with a junctional or ventricular escape rhythm may be present.

Note: Consider digitalis toxicity in the setting of atrial flutter with complete heart block and junctional tachycardia.

Note: Flutter waves can deform QRS, ST and/or T to mimic intraventricular conduction delay and/or myocardial ischemia.

Note: Etiology is the same as for atrial fibrillation (item 19).

19. Atrial fibrillation



- P waves absent
- Atrial activity is totally irregular and represented by fibrillatory (f) waves of varying amplitude, duration and morphology, causing random oscillation of the baseline

Note: Atrial activity is best seen in leads V_1, V_2, II, III, aVF.

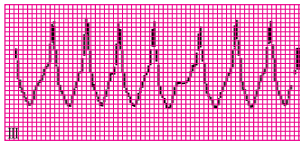
- Ventricular rhythm is typically irregularly irregular

  Note: If the RR interval is regular, second- or third-degree AV block may be present.

  Note: Digitalis toxicity may result in regularization of the QRS due to complete heart block with junctional tachycardia.
- Ventricular rate is usually 100-180 per minute in the absence of drugs

  Note: If the rate without AV blocking drugs is less than 100 beats per minute, AV conduction system disease is likely to be present.

  Note: Consider **Wolff-Parkinson-White syndrome** (item 34) if the ventricular rate is > 200 per minute and the QRS is > 0.12 seconds. The 12-lead ECG during sinus rhythm should show a short PR interval and a wide QRS complex with initial slurring (delta wave):



Note: Conditions mimicking atrial fibrillation include:

- Multifocal atrial tachycardia (item 16)
- Atrial flutter (item 18)

Note: Etiologies include:

- Mitral valve disease (especially if severe)
- Organic heart disease
- Hypertension
- Post-CABG (30% of patients)
- Myocardial infarction
- Thyrotoxicosis
- Pulmonary embolism (item 82)
- Post-operative state
- Hypoxia
- Chronic lung disease (e.g., emphysema) (item 81)
- Atrial septal defect (items 78, 79)
- Wolff-Parkinson-White syndrome (item 34)
- Sick sinus syndrome (tachy-brady syndrome) (item 89)
- Alcohol ("Holiday heart" syndrome)
- Normals (lone atrial fibrillation)

Junctional Rhythms

20. AV junctional premature complexes

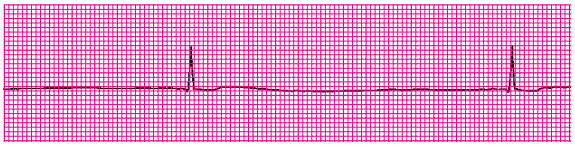
- Premature QRS complex (relative to the basic RR interval), which may be narrow or wide (if underlying bundle branch block or aberrancy)
- The P wave may precede the QRS by ≤ 0.11 seconds (retrograde atrial activation,), may be buried in the QRS (and not visualized), or may follow the QRS complex
- Inverted P waves in leads II, III, aVF and upright P waves in leads I and aVL are commonly seen due to the spread of atrial activation from near the AV node and in a superior and leftward direction (i.e., away from the inferior leads and toward the left lateral leads).

  Note: The atrium may occasionally be activated by the sinus node, resulting in a normal sinus P wave. This occurs when retrograde block exists between the AV junctional focus and the atrium, or the sinus node activates the atrium before the AV junctional impulse.

  Note: A constant coupling interval and noncompensatory pause are usually present.

  Note: Seen in normals and organic heart disease.

21. AV junctional escape complexes

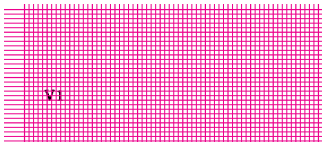


- Typically narrow QRS complex beat(s) that follow the previous conducted beat at a coupling interval corresponding to a rate of 40-60 per minute. QRS may be wide if underlying bundle branch block
- P wave may precede (PR < 0.11 seconds), be buried in, or follow the QRS complex (similar to AV junctional premature complexes; item 23)
- QRS morphology is similar to the sinus or supraventricular impulse

Note: QRS complex occurs as a secondary phenomenon in response to decreased sinus impulse formation or conduction,

high-degree AV block, or after a pause following termination of atrial tachycardia, atrial flutter, or atrial fibrillation.

22. AV junctional rhythm/tachycardia



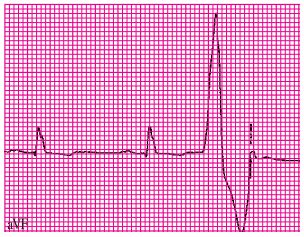
- RR interval is usually regular
- Heart rate is between 40-60 per minute for AV junctional rhythm, and > 60 per minute for junctional tachycardia
- P wave may proceed, be buried in, or follow the QRS complex
- QRS is usually narrow, but may be wide if underlying bundle branch block or aberrancy
- Relationship between atrial and ventricular rates may vary:
  - If retrograde (VA) block is present, the atria remain in sinus rhythm and *AV dissociation* (item 35) will be present
  - If retrograde atrial activation (inverted P waves in II, III, aVF) occurs, a constant QRS-P interval is usually present

Note: Consider digitalis toxicity (item 71) if atrial fibrillation or flutter with a regular RR is seen — this often represents complete heart block with junctional tachycardia

Note: Junctional tachycardia can be seen in acute myocardial infarction (usually inferior), myocarditis, digitalis toxicity, and following open heart surgery.

Ventricular Rhythms

23. Ventricular premature complexes



Requires all of the following:

- A wide, notched or slurred QRS complex that is:
  - Premature relative to the normal RR interval, *and*
  - Not preceded by a P wave (except when late coupled VPCs follow a sinus P wave; in this case, the PR interval is usually ≤ 0.11 seconds)

  Note: QRS is almost always > 0.12 seconds, but VPCs originating high in the interventricular septum may have a relatively normal QRS duration.

  Note: When a VPC occurs just distal to the site of bundle branch block and near the interventricular septum, the QRS of the VPC may be narrower than the QRS of the bundle branch block.

  Note: Initial direction of the QRS is often different from the QRS during sinus rhythm.
- Secondary ST & T wave changes in a direction opposite to the major deflection of the QRS (i.e., ST depression & T wave inversion in leads with a dominant R wave; ST elevation and upright T wave in leads with a dominant S wave or QS complex)
- Coupling interval (relation of VPCs to the preceding QRS) may be constant or variable

  Note: Non-fixed coupling should raise the suspicion of ventricular parasystole (item 24)
- Morphology of VPCs in any given lead may be the same (uniform) or different (multiform)

  Note: Although multiform VPCs are usually multifocal in origin (i.e., originate from more than one ventricular focus), a single ventricular focus can produce VPCs of varying morphology.

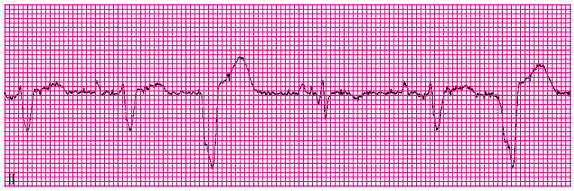
Note: Retrograde capture of atria may occur

Note: A full compensatory pause (PP interval containing the VPC is twice the normal PP interval) is usually evident, but this relationship may be altered if sinus arrhythmia is also present. A partial compensatory pause may follow a VPC when ventriculoatrial conduction penetrates and resets the sinus node. Less commonly, interpolated VPCs occur, manifesting as VPCs that are interposed between two consecutive sinus beats without disrupting the basic sinus rhythm; interpolated VPCs result in neither a partial nor a full compensatory pause.

Note: Clues on the electrocardiogram suggestive of a ventricular (rather than atrial) origin of an ectopic beat include an initial QRS vector different from the sinus beats, QRS duration > 0.12 seconds, retrograde P waves (caused by retrograde conduction through the AV node), and the presence of a full compensatory pause.

Note: Seen in normals and all causes of ventricular tachycardia (item 25).

24. Ventricular parasystole



- Frequent ventricular premature complexes (VPCs) usually at a rate of 30-50 per minute with the interectopic intervals a multiple (2x, 3x, etc.) of the shortest interectopic interval present (since the parasystolic focus fires at a regular rate and inscribes a QRS complex whenever the ventricles are not refractory)
- Resultant VPCs vary in relationship to the preceding sinus or supraventricular beats (i.e., nonfixed coupling)
- VPCs typically manifest uniform morphology (which resembles a VPC, item 23) unless fusion occurs

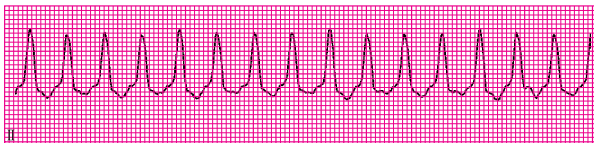
  Note: Fusion complexes, resulting from simultaneous activation of the ventricles by atrial and parasystolic impulses, are commonly seen but are not required for the diagnosis.

  Note: Exit block from a parasystolic focus may occur and result in absence of a ventricular ectopic beat when it would be expected to occur.

  Note: Ventricular parasystole is due to the presence of an ectopic ventricular focus that activates the ventricles independent of the basic sinus or supraventricular rhythm, and is protected from depolarization by an entrance block. The ventricular focus fires at a regular cycle length and results in a VPC that bears no constant relationship (nonfixed coupling) to the previous sinus beat. In contrast to ventricular parasystole, uniform VPC's due to local reentry initiated by prior sinus activation of the ventricle show fixed coupling.

  Note: Think of parasystole when you see ventricular premature complexes with nonfixed coupling and fusion beats.

25. Ventricular tachycardia



- Rapid succession of three or more ventricular premature complexes (item 23) at a rate > 100 per minute
- RR interval is usually regular but may be irregular
- Abrupt onset and termination of arrhythmia is evident
- AV dissociation (item 35) is common
- On occasion, retrograde atrial activation, fusion complexes, and ventricular capture complexes occur

Note: Ventriculoatrial (VA) conduction may occur at 1:1 or may manifest variable, fixed, or complete block; ventriculoatrial Wenckebach may also occur.

Note: In the setting of a wide QRS tachycardia, certain findings may help distinguish ventricular tachycardia from supraventricular tachycardia with aberrancy (Table).

Note: Rarely, VT can present as a narrow QRS tachycardia.

Note: Bidirectional VT is a rare type of VT in which the QRS complexes in any given lead alternate in polarity. It is most often caused by digitalis toxicity.

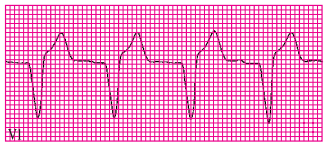
Note: Seen in:

- Organic heart disease
- Hypokalemia/hyperkalemia (items 74, 75)
- Hypoxia/acidosis
- Drugs (digitalis toxicity, antiarrhythmics, phenothiazines, tricyclics, caffeine, alcohol, nicotine)
- Mitral valve prolapse
- Occasionally in normals

Table. Origin of Wide QRS Tachycardia

	Favors VT	Favors SVT with Aberrancy
QRS morphology	Similar to VPCs	Similar to sinus rhythm or APCs with aberrancy
Initiation of tachycardia	VPCs	APCs
AV dissociation present	Yes	No
Capture or fusion complexes present	Yes	No
QRS duration when QRS is narrow during sinus rhythm	RBBB morphology (> 0.14 seconds); LBBB morphology (> 0.16 seconds)	QRS duration generally < 0.14 seconds
QRS deflection in precordial leads	Concordant (all positive or negative)	Discordant (some positive ; some negative)
QRS axis	Left or northwest	—
RSR' in lead V_1	R wave taller than R'	R' taller than R wave

26. Accelerated idioventricular rhythm



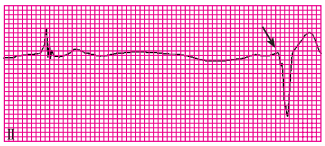
- Regular or slightly irregular ventricular (wide complex) rhythm
- Rate of 60-110 per minute
- QRS morphology similar to VPCs (item 23)
- AV dissociation (item 35), ventricular capture complexes, and fusion beats are common because of the competition between the normal sinus and ectopic ventricular rhythms.

Note: Unlike ventricular tachycardia, AIVR is not associated with an adverse prognosis.

Note: Seen in:

- Myocardial ischemia
- Following coronary reperfusion
- Digitalis toxicity (item 71)
- Occasionally in normals

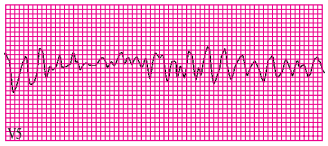
27. Ventricular escape complexes or rhythm



- Single beat or regular or slightly irregular ventricular rhythm
- Rate of 30-40 per minute (can be 20-50 per min)
- QRS morphology similar to VPCs (item 23)

Note: QRS escape complex/rhythm occurs as a secondary phenomenon in response to decreased sinus impulse formation or conduction (e.g., high vagal tone), high-degree AV block, or after the pause following termination of atrial tachycardia, atrial flutter, or atrial fibrillation.

28. Ventricular fibrillation

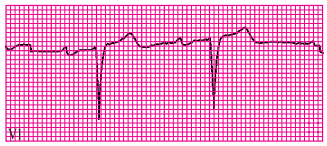


- An extremely rapid and irregular ventricular rhythm demonstrating:
  - Chaotic and irregular deflections of varying amplitude and contour
  - Absence of distinct P waves, QRS complexes, and T waves

Note: A lethal arrhythmia that can nearly always be converted into a stable rhythm when defibrillation occurs within the first minute. Successful cardioversion occurs in only 25% when delayed as little as 4-5 minutes.

AV Conduction Abnormalities

29. AV block, 1°



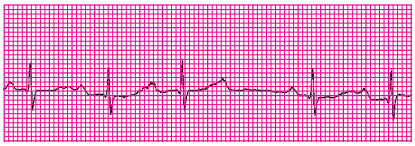
- PR interval ≥ 0.20 seconds (usually 0.21-0.40 seconds but may be as long as 0.80 seconds)
- Each P wave is followed by a QRS complex

Note: The PR interval represents the time from the onset of atrial depolarization to the onset of ventricular repolarization (i.e., conduction time from the atrium → AV node → His bundle → Purkinje system → ventricles). It does not reflect conduction from the sinus node to the atrial tissue. Therefore, a prolonged PR interval with a narrow QRS complex identifies the site of block in the AV node. If the QRS is wide, conduction delay or block typically occurs in the His-Purkinje system (although block in the AV node can manifest as a prolonged PR and wide QRS if bundle branch block or rate-dependant aberrancy is present).

Note: Etiologies include:

- Normals
- Athletes
- High vagal tone
- Drugs (digitalis, quinidine, procainamide, flecainide, propafenone, amiodarone, sotalol, popranolol, verapamil)
- Acute rheumatic fever
- Myocarditis
- Congenital heart disease (atrial septal defect, patent ductus arteriosus)

30. AV block, 2° - Mobitz Type I (Wenckebach)



- Progressive prolongation of the PR interval and progressive shortening of the RR interval until a P wave is blocked

  Note: The progressive shortening of the RR interval is due to a decrease in the beat-to-beat increment of PR prolongation.
- RR interval containing the nonconducted P wave is less than two PP intervals

Note: Classical Wenckebach periodicity may not always be evident, especially when sinus arrhythmia is present or an abrupt change in autonomic tone occurs.

Note: In Type I block with high conduction ratios (i.e., infrequent pauses), the PR interval of the beats immediately preceding the blocked P wave may be equal to each other, suggesting Type II block. In these situations, it is best to compare the PR intervals immediately before and after the blocked P wave; differences in the PR intervals suggest Type I block, whereas a constant PR interval suggests Type II block.

Note: Mobitz Type I results in "group" or "pattern beating" due to the presence of nonconducted P waves. Other causes of group beating include:

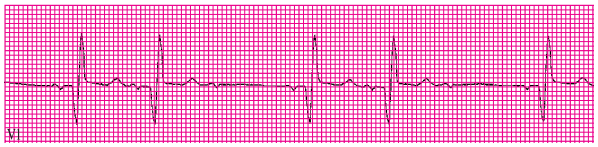
- Blocked APCs
- Type II second-degree AV block (item 31)
- Concealed His-bundle depolarizations: Premature His depolarizations render the AV node refractory to subsequent sinus beats, resulting in blocked P waves and pseudo-AV block.

Note: Type I block usually occurs at the level of the AV node, resulting in a narrow QRS complex. In contrast, Mobitz Type II block usually occurs within or below the bundle of His, and is associated with a wide QRS complex in 80% of cases.

Note: Etiologies include:

- Normals
- Athletes
- Drugs (digitalis, β-blocker, calcium blockers, clonidine, α-methyldopa, flecainide, sotalol, amiodarone encainide, propafenone, lithium)
- Myocardial infarction (especially inferior)
- Acute rheumatic fever
- Myocarditis

31. AV block, 2° - Mobitz Type II



- Regular sinus or atrial rhythm with intermittent nonconducted P waves and no evidence for atrial prematurity
- PR interval in the conducted beats is constant
- RR interval containing the nonconducted P wave is equal to two PP intervals

Note: Type II second-degree AV block usually occurs within or below the bundle of His; the QRS is wide in 80% of cases.

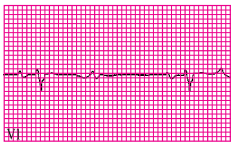
Note: 2:1 AV block can be Mobitz Type I or II (Table).

Note: In Type I block with high conduction rates (e.g., 10:9 conduction), the PR interval of the beats immediately preceding the blocked P wave may be equal, suggesting Type II block. In these situations, it is best to compare the PR interval immediately before and after the blocked P wave; differences in the PR interval suggest Type I block, whereas a constant PR interval is evidence for Type II block, which is almost always due to organic heart disease.

Table. Features Suggesting the Mechanism of 2:1 AV Block

Feature	Mechanism	
	Mobitz Type I	Mobitz Type II
QRS duration	Narrow	Wide
Response to maneuvers that increase heart rate & AV conduction (e.g., atropine, exercise)	Block improves	Block worsens
Response to maneuvers that reduce heart rate & AV conduction (e.g., carotid sinus massage)	Block worsens	Block improves
Develops during acute MI	Inferior MI	Anterior MI
Other	Mobitz I on another part of ECG	History of syncope

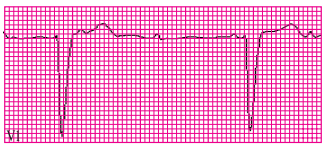
32. AV block, 2:1



- Regular sinus or atrial rhythm with two P waves for each QRS complex (i.e., every other P wave is nonconducted)

Note: Can be Mobitz Type I or II second-degree AV block (see Table on previous page).

33. AV block, 3°



- Atrial impulses consistently fail to reach the ventricles, resulting in atrial and ventricular rhythms that are independent of each other
- PR interval varies
- PP and RR intervals are constant
- Atrial rate is usually faster than ventricular rate
- Ventricular rhythm is maintained by a junctional or idioventricular escape rhythm or a ventricular pacemaker

Note: The P wave may precede, be buried within (and not visualized), or follow the QRS to deform the ST segment or T wave.

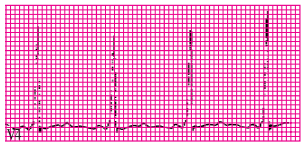
Note: Ventriculophasic sinus arrhythmia—PP interval containing a QRS complex is shorter than the PP interval without a QRS complex—is present in 30-50%

Note: Complete heart block is present when the atrial rate is faster than the ventricular escape rate (identified by the presence of nonconducted P waves when the AV node and ventricle are not refractory). In contrast, AV dissociation is usually present if the atrial rate is slower than the ventricular rate.

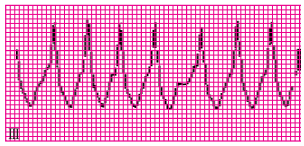
Note: Causes of complete heart block include:

- **MYOCARDIAL INFARCTION:** 5-15% of acute myocardial infarctions are complicated by complete heart block: In inferior MI, complete heart block is usually preceded by first-degree AV block or Type I second-degree AV block, usually occurs at the level of the AV node, is typically transient (< 1 week), and is usually associated with a stable junctional escape rhythm (narrow QRS; rate ≥ 40 per minute). In anterior MI, complete heart block occurs as a result of extensive damage to the left ventricle, is typically preceded by Type II second-degree AV block or bifascicular block, and is associated with mortality rates as high as 70% (due to pump failure rather than heart block per se)
- **DEGENERATIVE DISEASES** of the conduction system (Lev's disease, Lenegre's disease)
- **INFILTRATIVE DISEASES** of the myocardium (e.g., amyloid, sarcoid)
- **DIGITALIS TOXICITY:** One of the most common causes of reversible complete AV block; usually associated with a junctional escape rhythm (narrow QRS), which is often accelerated
- **ENDOCARDITIS:** Inflammation and edema of the septum and peri-AV nodal tissues may cause conduction failure and complete heart block; PR prolongation usually precedes this event
- **ADVANCED HYPERKALEMIA** (death is usually from ventricular tachyarrhythmias)
- **LYME DISEASE:** Caused by a tick-borne spirochete (Borrelia burgdorferi), this disorder begins with a characteristic skin rash (erythema chronicum migrans), and may be followed in subsequent weeks to months by joint, cardiac and neurological involvement. Cardiac involvement includes AV block that partial or complete, usually occurs at the level of the AV node, and may be accompanied by syncope
- **OTHERS:** Myocardial contusion, acute rheumatic fever, aortic valve disease

34. Wolff-Parkinson-White pattern



Sinus rhythm



Atrial fibrillation

- Normal P wave axis and morphology
- PR interval < 0.12 seconds (rarely > 0.12 seconds)

  Note: AV conduction over the accessory pathway (Bundle of Kent) bypasses the AV node (and AV nodal conduction delay), resulting in pre-excitation of the ventricles and a short PR interval
- Initial slurring of the QRS (delta wave), resulting in an abnormally wide QRS (> 0.12 seconds)

  Note: The QRS duration is ≤ 0.10 seconds in 30%. In these cases, the ventricles are depolarized almost entirely

by the normal AV conduction system, with minimal contribution from antegrade conduction along the accessory pathway.

Note: The widened QRS complexes represent fusion between electrical wavefronts conducted down the accessory pathway (delta wave) and the AV node. Differing degrees of pre-excitation (fusion) may be present, resulting in variability in the delta wave and QRS duration.

- Secondary ST-T wave changes (opposite in direction to main deflection of QRS)

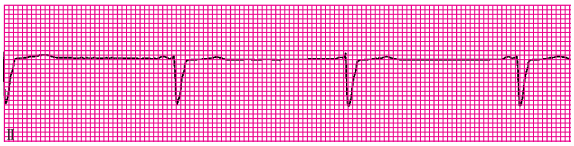
Note: The PJ interval (beginning of P wave to the J point (i.e., end of QRS complex) is constant and ≤ 0.26 seconds. This is due to an inverse relationship between the PR interval and QRS duration — if the PR interval shortens, the QRS widens; if the PR interval lengthens, the QRS narrows.

Note: Think WPW when atrial fibrillation or flutter is associated with a QRS that varies in width (generally wide) and has a rate >200 per minute

Note: Atrial fibrillation can conduct extremely rapidly, resulting in aberrant conduction and an irregular wide complex tachycardia, which resembles VT and can degenerate into VF.

OVERVIEW: Wolff-Parkinson-White syndrome (WPW) is characterized by the presence of an abnormal muscular network of specialized conduction tissue that connects the atrium to the ventricle and bypasses conduction through the AV node. It is found in 0.2-0.4% of the overall population and is more common in males and younger patients. Most patients with WPW do not have structural heart disease, although there is an increased prevalence of this disorder among patients with Epstein's anomaly (downward displacement of the tricuspid valve into the right ventricle due to anomalous attachment of the tricuspid leaflets), hypertrophic cardiomyopathy, mitral valve prolapse, and dilated cardiomyopathy. Two types of accessory pathways (AP) exist: In ***manifest*** AP, antegrade conduction occurs over the AP and results in pre-excitation on baseline ECG (which may be intermittent). In ***concealed*** AP, antegrade conduction occurs via the AV node and retrograde conduction occurs over the AP, so pre-excitation is not evident on the baseline ECG. Approximately 50% of patients with WPW manifest tachyarrhythmias, of which 80% is AV reentry tachycardia, 15% is atrial fibrillation, and 5% is atrial flutter. Asymptomatic individuals have an excellent prognosis. For patients with recurrent tachycardias, the overall prognosis is good, but in rare instances sudden death may occur. The presence of delta waves and secondary repolarization abnormalities can lead to a false positive or false negative diagnoses of ventricular hypertrophy, bundle branch block, or acute myocardial infarction. The polarity of the delta waves can be used to predict the location of the bypass tract.

35. AV dissociation



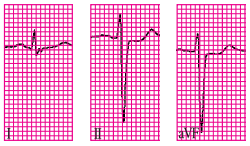
- Atrial and ventricular rhythms are independent of each other
- Ventricular rate is usually ≥ atrial rate

Note: AV dissociation is a secondary phenomenon resulting from some other disturbance of cardiac rhythm.

- AV dissociation may involve:
  - A ventricular rate that is faster than the normal atrial rate because of acceleration of a subsidiary pacemaker (e.g., junctional or ventricular tachycardia, myocardial ischemia, digitalis toxicity, post-operative state)
  - A ventricular rate that is faster than the normal atrial rate because of slowing of the atrial rate (sinus bradycardia, sinus arrest, sinoatrial exit block, high vagal tone, post-cardioversion, β-blockers) below the intrinsic rate of a subsidiary AV junctional or ventricular pacemaker
  - A ventricular rate that is slower than the atrial rate because of AV block

Abnormalities of QRS Axis

36. Left axis deviation



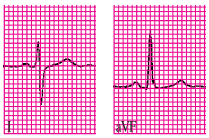
- Mean QRS axis between -30° and -90°

Note: Causes include:

- Left anterior fascicular block (if axis > –45°, item 45)
- Inferior wall MI (items 57, 58)
- LBBB (item 47)
- LVH (items 40)
- Ostium primum ASD (item 79)
- Chronic lung disease (item 81)

- Hyperkalemia (item 74)

37. Right axis deviation

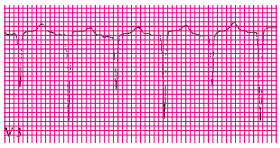


- Mean QRS axis between 100° and 270°

Note: Causes include:

- RVH (item 41)
- Vertical heart
- Chronic lung disease (item 81)
- Pulmonary embolus (item 82)
- Left posterior fascicular block (item 46)
- Lateral wall myocardial infarction (items 55, 56)
- Dextrocardia (item 80)
- Lead reversal (item 03)
- Ostium secundum ASD (item 78)

38. Electrical alternans



- Alternation in the amplitude and/or direction of P, QRS, and/or T waves

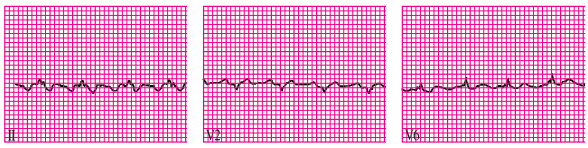
Note: Causes include:

- Pericardial effusion (item 83)

  Note: Electrical alternans is due to swinging of the heart in the pericardial fluid during the cardiac cycle. Only one-third of patients with QRS alternans have a pericardial effusion, and only 12% of patients with pericardial effusions have QRS alternans. If electrical alternans involves the entire P-QRS-T ("total alternans"), effusion with tamponade is often present (which is almost always associated with sinus tachycardia).
- Severe heart failure
- Hypertension
- Coronary artery disease
- Rheumatic heart disease
- Supraventricular or ventricular tachycardia
- Deep respirations

QRS Voltage Abnormalities

39. Low voltage

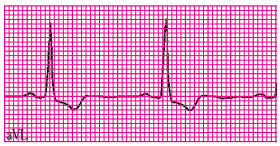


- Amplitude of the entire QRS complex (R+S) < 10 mm in all precordial leads and < 5 mm in all limb leads

Note: Causes include:

- Chronic lung disease (item 81)
- Pericardial effusion (item 83)
- Obesity
- Restrictive or infiltrative cardiomyopathies
- Coronary disease with extensive infarction of the left ventricle
- Myxedema (item 87)
- Pleural effusion

40. Left ventricular hypertrophy



VOLTAGE CRITERIA FOR LVH (sufficient for diagnosis without repolarization abnormalities)

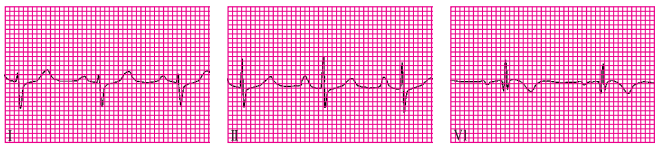
- **Cornell Criteria** (most accurate):
  R wave in aVL + S wave in V_3:
  - > 28 mm in males
  - > 20 mm in females
- **Other commonly used voltage-based criteria**
  - **PRECORDIAL LEADS** (one or more)
    - R wave in V_5 or V_6 + S wave in V_1
      - > 35 mm if age > 40 years
      - > 40 mm if age 30-40 years
      - > 60 mm if age 16-30 years
    - Maximum R wave + S wave in precordial leads > 45 mm
    - R wave in V_5 > 26 mm
    - R wave in V_6 > 20 mm
  - **LIMB LEADS** (one or more)
    - R wave in lead I + S wave in lead II ≥ 26 mm

- R wave in lead I ≥ 14 mm
- S wave in aVR ≥ 15 mm
- R wave in aVL ≥ 12 mm (a highly specific finding, except when associated with left anterior fascicular block)
- R wave in aVF ≥ 21 mm

Note: The amplitude of the QRS (and sensitivity for the diagnosis of LVH by voltage criteria) is often decreased by conditions that increase the amount of body tissue (obesity), air (COPD, pneumothorax), fluid (pericardial or plural effusion), or fibrous tissue (coronary artery disease, sarcoid or amyloid of the heart) between the myocardium and ECG electrodes. Severe RVH can also underestimate the ECG diagnosis of LVH by canceling prominent QRS forces from the thickened LV. Left bundle branch block may also reduce QRS amplitude as well. In contrast, thin body habitus, left mastectomy, LBBB, WPW, and left anterior fascicular block may increase QRS amplitude in the absence of LVH, decreasing the specificity of the voltage criteria.

- **NON-VOLTAGE RELATED CHANGES** (often present but not required for the diagnosis of LVH)
  - Left atrial abnormality/enlargement (item 06)
  - Left axis deviation (item 36)
  - Nonspecific intraventricular conduction disturbance (item 49)
  - Delayed onset of intrinsicoid deflection (beginning of QRS to peak of R wave > 0.05 seconds)
  - Small or absent R waves in V_1-V_3 (low anterior forces)
  - Absent Q waves in leads I, V_5, V_6
  - Abnormal Q waves in leads II, III, aVF (due to left axis deviation)
  - Prominent U waves (item 69)
  - R wave in V_6 > V_5, provided there are dominant R waves in these leads
- **REPOLARIZATION (ST AND/OR T WAVE) ABNORMALITIES SUGGESTING LVH** (see item 67)

41. Right ventricular hypertrophy



- Right axis deviation with mean QRS axis ≥ + 100°
- Dominant R wave
  - R/S ratio in V_1 or V_{3R} > 1, *or* R/S ratio in V_5 or V_6 ≤ 1
  - R wave in V_1 ≥ 7 mm
  - R wave in V_1 + S wave in V_5 or V_6 > 10.5 mm
  - rSR' in V_1 with R' > 10 mm
  - qR complex in V_1
- Secondary ST-T changes (downsloping ST depression, T-wave inversion) in right precordial leads (if present, be sure to code item 67)
- Right atrial abnormality/enlargement (item 06) is common
- Onset of intrinsicoid deflection (beginning of QRS to peak of R wave) in V_1 < 0.05 seconds

Note: For ECG features of RVH in the setting of chronic lung disease, see item 81.

Note: Severe RVH can also underestimate the ECG diagnosis of LVH by canceling prominent QRS forces from the thickened LV.

Note: Conditions that can present with right axis deviation and/or a dominant R wave and possibly mimic RVH include:

- Posterior or inferoposterolateral wall MI (items 59, 60). When a tall R wave is present in lead V_1, other ECG findings can help distinguish right ventricular hypertrophy (RVH) from posterior MI: T wave inversions in V_1V_2 and right axis deviation favors the diagnosis of RVH, while inferior Q waves suggestive of inferior MI favors the diagnosis of posterior MI.
- Right bundle branch block (items 43, 44)
- Wolff-Parkinson-White syndrome (type A) (item 34)
- Dextrocardia (item 80)
- Left posterior fascicular block (item 46)
- Normal variant (especially in children)

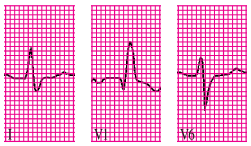
42. Combined ventricular hypertrophy

Suggested by any of the following:

- ECG meets one or more diagnostic criteria for LVH (item 40) and RVH (item 41)
- Precordial leads show LVH but QRS axis is >90°
- LVH *plus:*
  - R wave > Q wave in aVR, *and*
  - S wave > R wave in V_5, *and*
  - T wave inversion in V_1
- Large amplitude, equiphasic ® = S) complexes in V_3 and V_4 (Kutz-Wachtel phenomenon)
- Right atrial abnormality/enlargement (item 05) with LVH pattern (item 40) in precordial leads

Intraventricular Conduction Abnormalities

43. RBBB, complete



- Prolonged QRS duration (≥ 0.12 seconds)
- Secondary R wave (R') in leads V_1 and V_2 (rsR' or rSR') with R' usually taller than the initial R wave
- Delayed onset of intrinsicoid deflection (beginning of QRS to peak of R wave > 0.05 seconds) in V_1 and V_2
- Secondary ST & T-wave changes (T wave inversion; downsloping ST segment may or may not be present) in leads V_1 and V_2
- Wide slurred S wave in leads I, V_5, and V_6

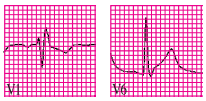
Note: In RBBB, mean QRS axis is determined by the initial unblocked 0.06-0.08 seconds of QRS, and should be normal unless left anterior fascicular block (item 45) or left posterior fascicular block (item 46) is present.

Note: RBBB does not interfere with the ECG diagnosis of ventricular hypertrophy or Q-wave MI.

Note: Can be seen in:

- Occasionally in normal adults (incidence ~ 2/1000) without underlying structural heart disease (unlike LBBB). These patients have essentially the same prognosis as the general population. However, among patients with coronary artery disease, RBBB is associated with a 2-fold increase in morality (compared to patients with coronary disease but without bundle branch block).
- Hypertensive heart disease
- Myocarditis
- Cardiomyopathy
- Rheumatic heart disease
- Cor pulmonale (acute or chronic)
- Degenerative disease of the conduction system (Lenegre's disease) or sclerosis of the cardiac skeleton (Lev's disease)
- Ebstein's anomaly

44. RBBB, incomplete

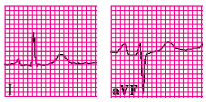


- RBBB morphology (rSR' in V_1; item 43) with a QRS duration between 0.09 and 0.12 seconds

Note: Other causes of RSR' pattern < 0.12 seconds in lead V_1 include:

- Normal variant (present in ~ 2% of healthy adults) (item 02)
- Right ventricular hypertrophy (item 41)
- Posterior wall MI (items 59, 60)
- Incorrect lead placement (electrode for lead V_1 placed in 3rd instead of 4th intercostal space) (item 03)
- Skeletal deformities (e.g., pectus excavatum)
- Atrial septal defect (items 78, 79)

45. Left anterior fascicular block



- Left axis deviation with mean QRS axis between –45° and –90° (item 36)
- qR complex (or an R wave) in leads I and aVL
- rS complex in lead III
- Normal or slightly prolonged QRS duration (0.08-0.10 seconds)
- No other factors responsible for left axis deviation:
  - LVH (items 40)
  - Inferior wall MI (items 57, 58)
  - Emphysema (chronic lung disease) (item 81)
  - Left bundle branch block (item 47)
  - Ostium primum atrial septal defect (item 79)
  - Severe hyperkalemia (item 74)

Note: LAFB may result in a false-positive diagnosis of LVH based on voltage criteria in leads I or aVL.

Note: Poor R wave progression is common.

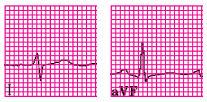
Note: Left anterior fascicular block can mask the presence of inferior wall MI.

Note: When QS complexes are present in the inferior leads, inferior MI and LAFB may both be present, but inferior MI alone should be coded.

Note: The anterior fascicle of the left bundle branch supplies the Purkinje fibers to the anterior and lateral walls of the left ventricle.

Note: Seen in organic heart disease, congenital heart disease, and rarely in normals.

46. Left posterior fascicular block



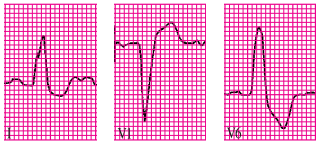
- Right axis deviation with mean QRS axis between +100° and +180° (item 37)
- Normal or slightly prolonged QRS duration (0.08-0.10 seconds)
- No other factors responsible for right axis deviation:
  - RVH (item 41)
  - Vertical heart
  - Emphysema (chronic lung disease) (item 81)
  - Pulmonary embolism (item 82)
  - Lateral wall MI (items 55, 56)
  - Dextrocardia (item 80)
  - Lead reversal (item 03)
  - Wolff-Parkinson-White (item 34)

Note: Left posterior fascicular block can mask the presence of lateral wall MI.

Note: Compared to the left anterior fascicle, the left posterior fascicle is shorter, thicker, and receives blood supply from both left and right coronary arteries. Isolated left posterior fascicular block (LPFB) is much less prevalent than left bundle branch block, right bundle branch block, or left anterior fascicular block.

Note: Coronary artery disease is the most common cause of LPFB; when it develops during acute MI, multivessel coronary disease and extensive infarction are usually present, and the prognosis is poor. LPFB is rarely seen in normals.

47. LBBB, complete



- Prolonged QRS duration (≥ 0.12 seconds)
- Delayed onset of intrinsicoid deflection (i.e., beginning of QRS to peak of R wave > 0.05 seconds) in leads I, V_5, V_6
- Broad monophasic R waves in leads I, V_5, V_6 that are usually notched or slurred
- Secondary ST & T wave changes opposite in direction to the major QRS deflection (i.e., ST depression & T wave inversion in leads I, V_5, V_6; ST elevation & upright T wave in leads V_1 and V_2)
- rS or QS complex in right precordial leads

Note: Left axis deviation may be present (item 36).

Note: LBBB interferes with determination of QRS axis and identification of ventricular hypertrophy and acute MI. Although the formal diagnosis of LVH should not be made in the setting LBBB, echocardiographic and pathological studies show that ~ 80% of patients with LBBB have abnormally increased LV mass

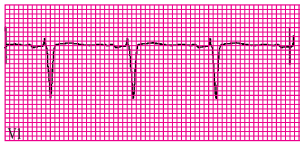
Note: Seen in:

- LVH (item 40)
- Myocardial infarction
- Organic heart disease
- Congenital heart disease
- Degenerative conduction system disease
- Rarely in normals

48. LBBB, incomplete

- LBBB morphology (item 47) with a QRS duration ≥ 0.09 seconds and < 0.12 seconds

49. Nonspecific intraventricular conduction disturbance

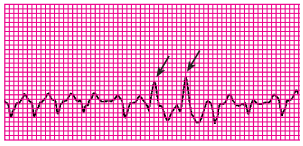


- QRS ≥ 0.11 seconds in duration but morphology does not meet criteria for LBBB (item 47) or RBBB (item 43), *or*
- Abnormal notching of the QRS complex without prolongation

Note: Nonspecific IVCD may be seen with:

- Antiarrhythmic drug toxicity (especially Type IA and IC agents) (item 73)
- Hyperkalemia (item 74)
- LVH (item 40)
- Wolff-Parkinson-White (item 34)
- Hypothermia (item 88)
- Severe metabolic disturbances

50. Functional (rate-related) aberrant intraventricular conduction



- Wide (> 0.12 seconds) QRS complex rhythm due to underlying supraventricular arrhythmia, such as atrial fibrillation, atrial flutter, other SVTs.

  Note: Since the right bundle has a longer refractory period than the left bundle, aberrant conduction usually occurs down the left bundle, resulting in QRS morphology with RBBB pattern.

  Note: May resemble VT (see item 25 for criteria to distinguish between SVT with aberrancy vs. VT).

  Note: Return to normal intraventricular conduction may be accompanied by T wave abnormalities.

Q Wave Myocardial Infarctions

MYOCARDIAL ISCHEMIA VS. INJURY VS. INFARCTION

- Ischemia: ST segment depression: T waves usually inverted; Q waves absent
- Injury: ST segment elevation; Q waves absent
- Infarction: Abnormal Q waves; ST segment elevation or depression; T waves inverted, normal, or upright & symmetrically peaked

  Note: Prior MI may be present without Q waves in: (1) Anterior MI: May only see low anterior R wave forces with decreasing R wave progression in leads V_2-V_5; and (2) Posterior MI: Dominant R wave in V_1 and/or V_2, usually in the setting of inferior MI. ST depression is often present during acute infarction in leads V_1-V_3

SIGNIFICANT ST ELEVATION

- New ST segment elevation at the J point (where QRS complex meets the ST segment) in ≥ 2 contiguous leads
  - ST elevation ≥ 2 mm in leads V_1, V_2, or V_3
  - ST elevation ≥ 1 mm in other leads
- Usually with upwardly convex ("out-pouching") configuration
- Can persist 48 hours to 4 weeks after MI

  Note: Persistent ST elevation beyond 4 weeks suggests the presence of a ventricular aneurysm

T WAVE INVERSION typically begins while the ST segments are still elevated (in contrast to pericarditis) and may persist indefinitely

Note: Acute infarction can occur without significant ST segment elevation or depression: up to 40% of patients with acute occlusion of the left circumflex coronary artery and 10-15% of patients with RCA or LAD occlusions may not have significant ECG changes.

ABNORMAL Q WAVES

- Any Q wave in leads V_1 - V_3
- Q wave ≥ 0.03 seconds in leads I, II, aVL, aVF, V_4, V_5, or V_6
- Q wave changes must be present in at least 2 contiguous leads, and must be ≥ 1 mm in depth

Note: The presence of a Q wave cannot be used to reliably distinguish transmural from subendocardial MI.

Note: Abnormal Q waves regress or disappear over months to years in up to 20% of patients with Q-wave MI.

AGE OF INFARCT CAN BE APPROXIMATED FROM THE ECG:

- Age Recent or Acute: The repolarization abnormalities associated with acute myocardial infarction typically evolve in a relatively predictable fashion. Usually, the earliest finding is marked peaking of the T waves ("hyperacute T waves") in the region of the infarct; these are often missed since they occur very early (< 15 minutes) in the course of the acute event and are transient. If transmural ischemia persists for more than a few minutes, the peaked T waves evolve into ST segment elevation, which should be ≥ 1 mm in height to be considered significant. The ST segment elevation of myocardial infarction is usually upwardly ***convex*** (in contrast to acute pericarditis or normal variant early repolarization, in which the ST elevation is usually upwardly ***concave)***. As the acute infarction continues to evolve, the ST segment elevation decreases and the T waves begin to invert. The T waves usually become progressively deeper as the ST segment elevation subsides. Abnormal Q waves develop within the first few hours to days after an infarction.
  - Acute MI: Abnormal Q waves, ST elevation (associated ST depression is sometimes present in noninfarct leads). Hyperacute (tall, peaked) T waves are seen very early (transient)
  - Recent MI: Abnormal Q waves, isoelectric ST segments, ischemic (usually inverted) T waves
- Age Indeterminate or Old: Abnormal Q waves, isoelectric ST segments, nonspecific or normal T waves

  Note: Exception: MI may be present without Q waves in: (1) Anterior MI: May only see low anterior R wave forces with decreasing R wave progression in leads V_2-V_5; and (2) Posterior MI: Dominant R wave and ST depression in leads V_1-V_3

PSEUDOINFARCTION PATTERN: See pages 17-18 for conditions causing "pseudoinfarcts" (ECG pattern mimicking myocardial infarction).

DIAGNOSIS OF Q WAVE MI IN THE PRESENCE OF BUNDLE BRANCH BLOCK

- RBBB: Does not interfere with the diagnosis of Q wave MI; Q wave criteria apply for all infarctions

- ▸ LBBB: Difficult to diagnose any infarct in the presence of LBBB. However, acute injury is sometimes apparent

51. Anterolateral MI (age recent or acute)

- Abnormal Q waves with significant ST segment elevation in leads V_4 - V_6

52. Anterolateral MI (age indeterminate or old)

- Abnormal Q waves in leads V_4 - V_6 *without* significant ST segment elevation

53. Anterior anteroseptal MI (age recent or acute)

- Abnormal Q waves with significant ST segment elevation in at least 2 consecutive leads between V_1 - V_4

Note: The presence of a Q wave in V_1 distinguishes anteroseptal from anterior infarction, although the distinction between the two in not necessary for testing purposes.

Note: Many ECG texts consider decreasing R wave voltage from V_2 - V_5 consistent with age indeterminate anterior MI, even in the absence of abnormal Q waves. However, because the board score sheet lists the various MIs under the subheading of "Q-wave infarction," loss of R wave voltage in the precordial leads in the absence of abnormal Q waves should not be coded as an MI.

54. Anterior or anteroseptal MI (age indeterminate or old)

- Abnormal Q waves in at least 2 consecutive leads between V_1 - V_4 *without* significant ST segment elevation

55. Lateral MI (age recent or acute)

- Abnormal Q waves with significant ST segment elevation in leads I and aVL

Note: An isolated Q wave in aVL does not qualify as a lateral MI.

56. Lateral MI (age indeterminate or old)

- Abnormal Q waves in leads I and aVL *without* significant ST segment elevation

57. Inferior MI (age recent or acute)

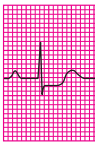
- Abnormal Q waves with significant ST segment elevation in at least two of leads II, III, aVF

Note: Associated ST depression is usually evident in leads I, aVL, V_1-V_3.

58. Inferior infarct (age indeterminate or old)

- Abnormal Q waves in at least two of leads II, III, aVF *without* significant ST segment elevation

59. Posterior MI (age recent or acute)



- Initial R wave ≥ 0.04 seconds in V_1 or V_2 with R wave amplitude ≥ S wave amplitude (R/S > 1) and significant (usually ≥ 2 mm) ST segment depression
  - ▸ Upright T waves are usually evident in same leads as dominant R wave

Note: The posterior wall of the left ventricular differs from the anterior, inferior, and lateral walls by not having ECG leads directly overlying it. Instead of Q waves and ST elevation, acute posterior MI presents with mirror-image changes in the anterior precordial leads (V_1-V_3), including dominant R waves (the mirror-image of abnormal Q waves), and horizontal ST segment depression (the mirror-image of ST elevation). Acute posterior infarction is often associated with ECG changes of acute inferior or inferolateral myocardial infarction, but may occur in isolation.

Note: RVH (item 41), WPW (item 34), and RBBB (item 43) may interfere with the ECG diagnosis of posterior MI.

60. Posterior MI (age indeterminate or old)

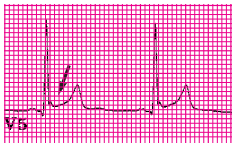
- Dominant R wave (R/S > 1) in leads V_1 or V_2 without significant ST segment depression

Note: Must be distinguished from other causes of a tall R wave in leads V_1 or V_2, including RVH, Wolff-Parkinson-White, RBBB, and incorrect electrode placement.

Note: Evidence of inferior wall ischemia or infarction is often present

Repolarization Abnormalities

61. Normal variant, early repolarization

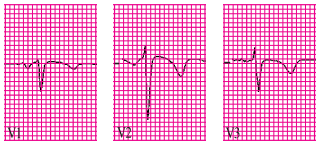


- Elevated take-off of ST segment at the junction between the QRS and ST segment (J junction)
- Concave upward ST elevation ending with a symmetrical upright T wave (often of large amplitude)

  Note: ST elevation should be less than 25% of the height of the T wave in lead V_6
- Distinct notch or slur on downstroke of R wave
- Most commonly involves V_2-V_5; sometimes II, III, aVF
- No reciprocal ST segment depression

Note: Some degree of ST elevation is present in the majority of young healthy individuals, especially in the precordial leads.

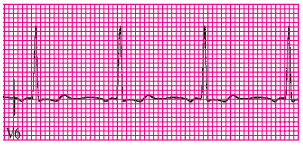
62. Normal variant, juvenile T waves



- Persistently negative T waves (usually not symmetrical or deep) in leads V_1-V_3 in normal adults
- T waves still upright I, II, V_5, V_6

Note: Juvenile T waves is a normal variant ECG finding commonly seen in children, occasionally seen as a normal variant in adult women, but only rarely seen in adult men.

63. Nonspecific ST and/or T wave abnormalities



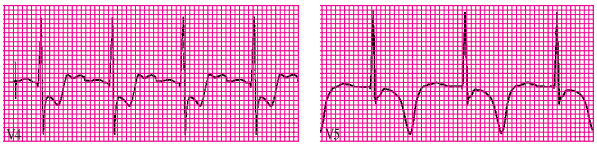
- Slight (< 1mm) ST depression or elevation, *and/or*
- T wave flat or slightly inverted

Note: Normal T waves usually ≥ 10% the height of R wave

Note: Can be seen in:

- Organic heart disease
- Drugs (e.g., quinidine)
- Electrolyte disorders (e.g., hyperkalemia, hypokalemia)
- Hyperventilation
- Myxedema (item 87)
- Recent large meal
- Stress
- Pancreatitis
- Pericarditis (item 84)
- CNS disorders (item 86)
- LVH (item 40)
- RVH (item 41)
- Bundle branch block (items 43, 47)
- Healthy adults (normal variant) (item 02)
- Persistent juvenile pattern: T wave inversion in V_1-V_3 in young adults

64. ST and/or T wave abnormalities suggesting myocardial ischemia



- Ischemic ST segment changes:
  - Horizontal or downsloping ST segments with or without T wave inversion

  Note: Flutter waves or prominent atrial repolarization waves (as can be seen in left/right atrial enlargement, pericarditis, atrial infarction) can deform the ST segment and result in "pseudodepression."
- Ischemic T wave changes:
  - Biphasic T waves with or without ST depression
  - Symmetrical or deeply inverted T waves; QT interval is often prolonged

Note: Reciprocal T wave changes may be evident (e.g., tall upright T waves in inferior leads with deeply inverted T waves in anterior leads).

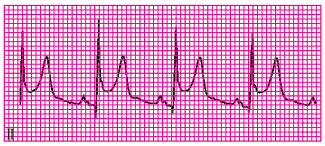
Note: T waves may become less inverted or upright during acute ischemia ("pseudonormalization).

Note: Prominent U waves (upright or inverted) (item 69) are often present.

Note: Tall upright T waves may also be seen in:

- Normal healthy adults (item 02)
- Hyperkalemia (item 74)
- Early myocardial infarction
- LVH (item 40)
- CNS disorders (item 86)
- Anemia

65. ST and/or T wave abnormalities suggesting myocardial injury



- Acute ST segment elevation ≥ 1 mm with upward convexity (may be concave early) in the leads representing the area of jeopardized myocardium/acute infarction
- ST and T wave changes evolve: T waves invert before ST segments return to baseline
- Associated ST depression in the noninfarct leads is common
- Acute posterior wall injury often has horizontal or downsloping ST segment depression with upright T waves in V_1 and/or V_2 with prominent R wave in these same leads

Note: It is important to consider the clinical context, since ST segment elevation suggesting myocardial injury can also be seen in:

- Acute pericarditis (item 84)
- Ventricular aneurysm
- Early repolarization (item 61)
- LVH (item 40)
- Hyperkalemia (item 74)
- Bundle branch block (items 43, 47)
- Myocarditis
- Apical hypertrophic cardiomyopathy (item 85)
- Central nervous system disease (item 86)
- Normals (ST elevation up to 3 mm may be seen in leads V_1-V_3)

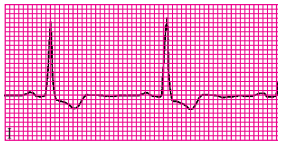
66. ST and/or T wave abnormalities suggesting electrolyte disturbances

- Any abnormalities suggesting hyperkalemia, hypokalemia, hypercalcemia, or hypocalcemia (see items 74-77)

  Note: Hypomagnesemia causes changes similar to hypocalcemia (QT prolongation)

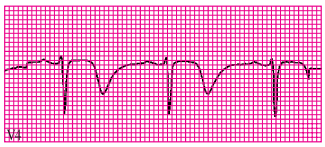
  Note: Renal failure often results in multiple electrolyte derangements with a wide variety of associated ECG abnormalities

67. ST and/or T wave abnormalities secondary to hypertrophy



- **LVH:** ST segment and T wave displacement opposite to the major QRS deflection:
  - ST depression (upwardly concave) and T wave inversion when the QRS is mainly positive (leads I, V_5, V_6)
  - Subtle (< 1 mm) ST elevation and upright T waves when the QRS is mainly negative (leads V_1, V_2); with more extreme voltage, ST elevation up to 2-3 mm can be seen in leads V_1-V_2
- **RVH:** ST segment depression and T wave inversion in leads V_1-V_3 and sometimes in leads II, III, aVF

68. Prolonged QT interval



- Corrected QT interval (QTc) ≥ 0.44 seconds, where *QTc = QT when the heart rate is 60 BPM = QT interval divided by the square root of the preceding RR interval*

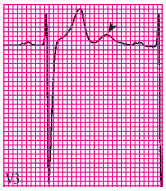
  Note: Be sure to measure the QT interval in a lead with a large T wave and distinct termination. Also look for the lead with the longest QT.
- Easier method to determine QT interval:
  - Use 0.40 seconds as the normal QT interval for a heart rate of 70. For every 10 BPM change in heart rate above (or below) 70, subtract (or add) 0.02 seconds. (Measured value should be within ± 0.04 seconds of the calculated normal.) Example: For a heart rate of 100 BPM, the calculated normal QT interval = 0.40 seconds – (3 x 0.02 seconds) = 0.34 ± 0.04 seconds. For a heart rate of 50 BPM, the calculated normal QT interval = 0.40 seconds + (2 x 0.02 seconds) = 0.44 ± 0.04 seconds.
  - In general, the normal QT interval should be less than 50% of the RR interval

Note: The QT interval represents the period of ventricular electrical systole (i.e., the time required for ventricular depolarization and repolarization to occur), varies inversely with heart rate, and is longer while asleep than while awake (presumably due to vagal hypertonia).

Note: Conditions associated with a prolonged QT interval include:

- Drugs (quinidine, procainamide, disopyramide, amiodarone, sotalol, dofetilide, azimilide, phenothiazines, tricyclics, lithium)
- Hypomagnesemia
- Hypocalcemia (item 77)
- Marked bradyarrhythmias
- Intracranial hemorrhage (item 86)
- Myocarditis
- Mitral valve prolapse
- Myxedema (item 87)
- Hypothermia (item 88)
- Very high protein diets
- Romano-Ward syndrome (congenital; normal hearing)
- Jervell and Lange-Nielson syndrome (congenital; deafness)

69. Prominent U waves



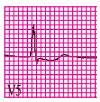
- Amplitude ≥ 1.5 mm

  Note: The U wave is normally 5-25% the height of the T wave, and is largest in leads V_2 and V_3

  Note: Causes include:
  - Hypokalemia (item 75)
  - Bradyarrhythmias
  - Hypothermia (item 88)
  - LVH (item 40)
  - Coronary artery disease
  - Drugs (digitalis, quinidine, amiodarone, isoproterenol)

Suggested Clinical Disorders

70. Digitalis effect



- Sagging ST segment depression with upward concavity
- T wave flat, inverted, or biphasic
- QT interval shortened
- U wave amplitude increased
- PR interval lengthened

Note: ST changes are difficult to interpret in the setting of LVH, RVH, or bundle branch block. However, if typical sagging ST segments are present and the QT interval is shortened, consider digitalis effect.

71. Digitalis toxicity

- Digitalis toxicity can cause almost any type of cardiac dysrhythmia or conduction disturbance except bundle branch block. Typical abnormalities include:
  - Paroxysmal atrial tachycardia with block
  - Atrial fibrillation with complete heart block (regular RR intervals)
  - Second or third-degree AV block
  - Complete heart block (item 33) with accelerated junctional rhythm (item 22) or accelerated idioventricular rhythm (items 26)
  - Supraventricular tachycardia with alternating bundle branch block

Note: Digitalis toxicity may be exacerbated by hypokalemia, hypomagnesemia, and hypercalcemia.

Note: Electrical cardioversion of atrial fibrillation is contraindicated in the setting of digitalis toxicity since protracted asystole or ventricular fibrillation can occur. (Digitalis levels should always be checked prior to elective electrical cardioversion).

72. Antiarrhythmic drug effect

Suggested by the following:

- Mild prolongation of QT interval (item 68)
- Prominent U waves (one of the earliest findings) (item 69)
- Nonspecific ST and/or T wave abnormalities (item 63)
- Decrease in atrial flutter rate

73. Antiarrhythmic drug toxicity

Suggested by the following:

- Marked prolongation of QT interval (item 68)
- Ventricular arrhythmias including "Torsade de Pointes" (paroxysms of irregular ventricular tachyarrhythmia at a rate of 200-280 BPM with sinusoidal cycles of changing QRS amplitude and polarity in the setting of a prolonged QT interval)
- Wide QRS complex
- Various degrees of AV block
- Marked sinus bradycardia (item 09), sinus arrest (item 11), or sinoatrial exit block (item 12)

74. Hyperkalemia

ECG changes depend on serum K^+ level and rapidity of rise:

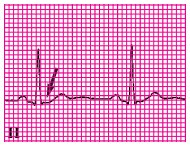
- $K^+ = 5.5 - 6.5$ mEq/L
  - Tall, peaked, narrow based T waves
    Note: Generally defined as > 10 mm in precordial leads and > 6 mm in limb leads). May also be seen as normal variant or in acute MI, LVH, or LBBB
  - QT interval shortening
  - Reversible left anterior fascicular block (item 45) or left posterior fascicular block (item 46)
- $K^+ = 6.5 - 7.5$ mEq/L
  - First-degree AV block (item 29)
  - Flattening and widening of the P wave
  - QRS widening
- $K^+ > 7.5$ mEq/L
  - Disappearance of P waves, which may be caused by:
    - Sinus arrest (item 11), *or*
    - "Sinoventricular conduction" (sinus impulses conducted to the ventricles via specialized atrial fibers *without* atrial depolarization)
  - LBBB (items 47, 48), RBBB (items 43, 44), or markedly widened and diffuse intraventricular conduction disturbance (item 49) resembling a sine-wave pattern
  - ST segment elevation
  - Arrhythmias and conduction disturbances including ventricular tachycardia (item 25), ventricular fibrillation (item 28), idioventricular rhythm (item 26, 27), asystole

75. Hypokalemia

Suggested by the following:

- Prominent U waves (item 69)
- ST segment depression and flattened T waves
  Note: The ST-T and U wave changes of hypokalemia are seen in approximately 80% of patients with potassium levels < 2.7 mEq/L, compared to 35% of patients with levels of 2.7-3.0 mEq/L, and 10% of patients with levels >3.0 mEq/L.
- Increased amplitude and duration of the P wave
- Prolonged QT sometimes seen
  Note: If potassium replacement does not normalize the QT interval, suspect hypomagnesemia.
- Arrhythmias and conduction disturbances, including paroxysmal atrial tachycardia with block, first-degree AV block (item 29), Type I second-degree AV block (item 30), AV dissociation (item 35), VPCs (item 23), ventricular tachycardia (item 25), and ventricular fibrillation (item 28).

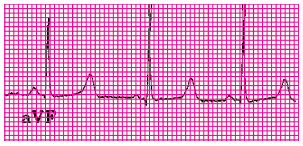
76. Hypercalcemia



- QTc shortening (usually due to shortening of the ST segment)
- May see PR prolongation

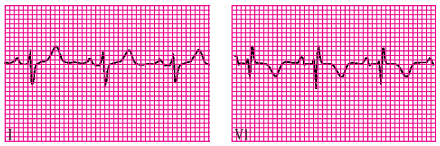
Note: Little if any effect on P, QRS, or T wave.

77. Hypocalcemia



- Prolonged QTc (item 68) (earliest and most common finding) due to ST segment prolongation without changing the duration of the T wave (seen only with hypocalcemia or hypothermia)
- Occasional flattening, peaking, or inversion of T waves

78. Atrial septal defect, secundum

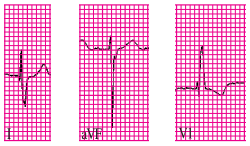


Suggested by the following:

- Typical RSR' or rSR' complex in V_1 with a QRS duration < 0.11 seconds (incomplete RBBB, item 44)
- Right axis deviation (item 37) ± right ventricular hypertrophy (item 41)
- Right atrial abnormality/enlargement (item 05) in ~ 30%
- First-degree AV block (item 29) in < 20%

Note: Ostium secundum ASDs represent 70-80% of all ASDs, and are due to deficient tissue in the region of the fossa ovalis.

79. Atrial septal defect, primum

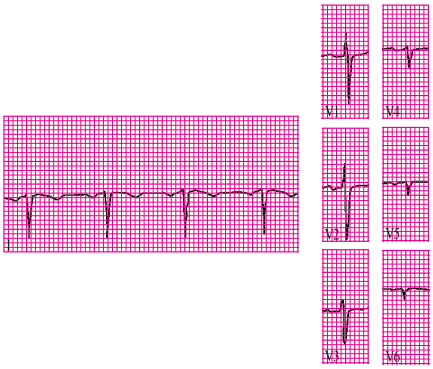


Suggested by the following:

- RSR' complex in V_1
- Incomplete RBBB (item 44)
- Left axis deviation (item 36) (in contrast to right axis deviation in ostium secundum ASD)
- First-degree AV block (item 29) in 15-40%
- Advanced cases have combined ventricular hypertrophy (item 42)

Note: Ostium primum ASDs represent 15-20% of all ASDs, and are due to deficient tissue in the lower portion of the septum. These ASDs are usually large and may be accompanied by anomalous pulmonary venous drainage. Primum ASDs are often associated with a cleft anterior mitral valve leaflet, mitral regurgitation, and Down's syndrome.

80. Dextrocardia, mirror image



Suggested by the following:

- P-QRS-T in leads I and aVL are inverted or "upside down"

  Note: Dextrocardia and lead reversal (item 03) can both produce an upside down P-QRS-T in leads I and aVL. To distinguish between these conditions, look at the R wave pattern in V_1 - V_6:
  - Reverse R wave progression (i.e., decreasing R wave amplitude form leads V_1-V_6) suggests dextrocardia
  - Normal R wave progression suggests lead reversal

Note: In ***mirror-image dextrocardia***, the most common form of dextrocardia, the abdominal and thoracic viscera (in addition to the heart) are transposed to the side opposite their usual locations (dextrocardia with "situs inversus"). This form of dextrocardia is generally not associated with severe congenital cardiac abnormalities (other than the malposition, which does not affect cardiac function). In ***isolated dextrocardia***, the heart is rotated to the right side of the chest but other viscera remain in their usual locations. This type of dextrocardia is almost always associated with serious congenital cardiac abnormalities, resulting in clinical difficulties in infancy or early childhood.

81. Chronic lung disease

- ECG features suggestive of COPD include:
  - Right ventricular hypertrophy (item 41)
  - Right axis deviation (item 37)
  - Right atrial abnormality/enlargement (item 05)
  - Poor precordial R wave progression
  - Low voltage (item 39)
  - Pseudo-anteroseptal infarct pattern (low anterior forces)

- S waves in leads I, II, and III (S_1 S_2 S_3 pattern)

- May also see sinus tachycardia (item 10), junctional rhythm (item 22), multifocal atrial tachycardia (item 16), various degrees of AV block, nonspecific IVCD (item 49), or bundle branch block (item 43, 44, 47, 48)

Note: Right ventricular hypertrophy in the setting of chronic lung disease is suggested by:

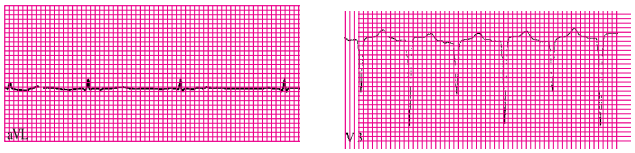
- Rightward shift of QRS
- T wave inversion in V_1, V_2
- ST depression in leads II, III, aVF
- Transient RBBB
- RSR' or QR complex in V_1

82. Acute cor pulmonale including pulmonary embolus

- ECG changes often accompany large pulmonary emboli and are associated with elevated pulmonary artery pressures, right ventricular dilation and strain, and clockwise rotation of the heart:
  - **S_1 Q_3 or S_1 Q_3 T_3** occurs in up to 30% of cases and lasts for 1-2 weeks
  - **Right bundle branch block** (incomplete or complete) may be seen in up to 25% of cases and usually lasts less than 1 week
  - **Inverted T waves** secondary to right ventricular strain may be seen in the right precordial leads and can last for months.
  - Other ECG findings include right axis deviation, nonspecific ST and T wave changes, and P pulmonale.
  - Arrhythmias and conduction disturbances include sinus tachycardia (most common arrhythmia), atrial fibrillation, atrial flutter, atrial tachycardia, and first-degree AV block.
- The clinical presentation and ECG of acute pulmonary embolism may sometimes be confused with acute inferior MI: Q waves and T wave inversions may be seen in leads III and aVF in both conditions, however, a Q wave in lead II is uncommon in pulmonary embolism and suggests MI.

Note: ECG abnormalities are often *transient*, and a normal ECG may be recorded despite persistence of the embolus. Sinus tachycardia, however, is usually present even when other ECG features of acute cor pulmonale are absent.

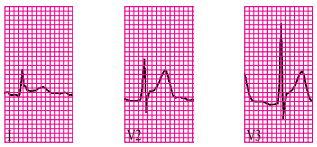
83. Pericardial effusion



- Low voltage QRS (item 39) (left strip) and/or electrical alternans (item 38) (right strip)

  Note: Low voltage QRS complexes and electrical alternans are consistent with (but not very sensitive or specific for) the diagnosis of pericardial effusion.
- Other features of acute pericarditis (item 84) may or may not be present

84. Acute pericarditis



- Classic evolutionary ST and T wave pattern consists of 4 stages (but is not always present):
  - Stage 1: Upwardly concave ST segment elevation in almost all leads except aVR; no reciprocal ST depression in other leads except aVR
  - Stage 2: ST junction (J point) returns to baseline and T wave amplitude begins to decrease
  - Stage 3: T waves invert
  - Stage 4: ECG returns to normal

  Note: T wave inversion usually occurs *after* the ST segment returns to baseline (in contrast to myocardial infarction, where T wave inversion typically begins while the ST segments are still elevated).

  Note: Pericarditis may be focal (e.g., post-pericardiotomy) and result in regional (rather than diffuse) ST elevation.

  Note: Classic ST and T wave changes are more likely to occur in purulent pericarditis as opposed to idiopathic, rheumatic, or malignant pericarditis.
- Other clues to acute pericarditis include:
  - Sinus tachycardia (item 10)
  - PR depression early (PR elevation in aVR)
  - Low voltage (item 39)

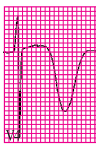
- Electrical alternans (item 38) if pericardial effusion (item 83)

85. Hypertrophic cardiomyopathy

- Majority have abnormal QRS
  - Large amplitude QRS
  - Large abnormal Q waves (can give pseudoinfarct pattern in inferior, lateral, and anterior precordial leads)
  - Tall R wave with inverted T wave in V_1 simulating RVH
- Left axis deviation (item 36) in 20%
- ST and T wave changes
  - Nonspecific ST and/or T wave abnormalities are common (item 63)
  - ST and/or T wave changes secondary to ventricular hypertrophy or conduction abnormalities
  - Apical variant of hypertrophic cardiomyopathy has deep T wave inversions in V_4-V_6 (item 85)
- Left atrial abnormality/enlargement (item 06) is common; right atrial abnormality/enlargement (item 05) on occasion

Note: The vast majority of patients with hypertrophic cardiomyopathy have abnormal ECGs, with LVH in 50-65%, left atrial abnormality/enlargement in 20-40%, and pathological Q waves (especially leads I, aVL, V_4 - V_5) in 20-30%. ST and T wave changes (repolarization abnormalities secondary to LVH) are the most common ECG findings, while right axis deviation is rare. Sinus node disease and AV block are occasional manifestations of this disorder. The most frequent cause of mortality is sudden death, with risk factors including young age and a history of syncope and/or asymptomatic ventricular tachycardia on ambulatory monitoring.

86. Central nervous system disorder



- "Classic changes" of cerebral and subarachnoid hemorrhage usually occur in the precordial leads
  - Large upright or deeply inverted T waves
  - Prolonged QT interval (often marked) (item 68)
  - Prominent U waves (item 69)
- Other changes:
  - T wave notching with loss of amplitude
  - ST segment changes:
    - Diffuse ST elevation mimicking acute pericarditis, *or*
    - Focal ST elevation mimicking acute myocardial injury, *or*
    - ST depression
  - Abnormal Q waves mimicking MI
  - Almost any rhythm abnormality (sinus tachycardia or bradycardia, junctional rhythm, VPCs, ventricular tachycardia, etc.)

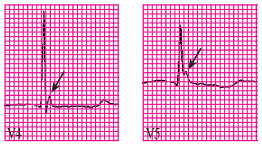
Note: ECG findings in CNS disease can mimic those of:

- Acute myocardial infarction
- Acute pericarditis (item 84)
- Drug effect or toxicity (items 70-73)

87. Myxedema

- Low voltage (item 39)
- Sinus bradycardia (item 09)
- T wave flattened or inverted
- PR interval may be prolonged (item 29)
- Frequently associated with pericardial effusion (item 83)
- Electrical alternans (item 38) may occur

88. Hypothermia



- Sinus bradycardia (item 09)
- Prolongation of PR, QRS, and QT (items 29, 49, 68)
- Osborne ("J") wave: late upright terminal deflection of QRS complex ("camel hump" sign); amplitude increases as temperature declines

  Note: Notching simulating an Osborne wave may be seen in early repolarization
- Atrial fibrillation (item 19) in 50-60%
- Other arrhythmias include AV junctional rhythm (item 22), ventricular tachycardia (item 25), ventricular fibrillation (item 28)

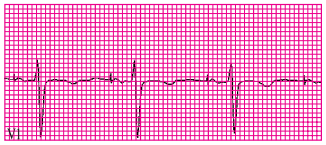
89. Sick sinus syndrome

One or more of the following:

- Marked sinus bradycardia (item 09)
- Sinus arrest (item 11) or sinoatrial exit block (item 12)
- Bradycardia alternating with tachycardia
- Atrial fibrillation with slow ventricular response preceded or followed by sinus bradycardia, sinus arrest, or sinoatrial exit block
- Prolonged sinus node recovery time after atrial premature complex or atrial tachyarrhythmias
- AV junctional escape rhythm
- Additional conduction system disease is often present, including AV block (items 29-33), nonspecific IVCD (item 49), and/or bundle branch block (items 43, 44, 47, 48)

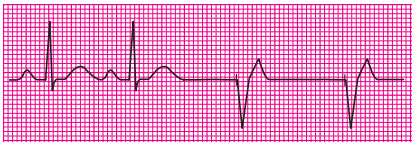
Paced Rhythms

90. Atrial or coronary sinus pacing



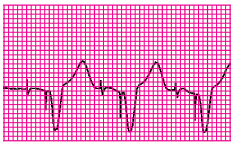
- Pacemaker stimulus followed by an atrial depolarization
- If the rate of the intrinsic rhythm falls below that of the pacemaker, atrial paced beats occur and will be separated by a constant (A-A) interval.
- Appropriately sensed intrinsic atrial activity (P wave) resets pacemaker timing clock. After an interval of time (A-A interval) with no sensed atrial activity, an atrial paced beat occurs.

91. Ventricular demand pacemaker (VVI), normally functioning



- Pacemaker stimulus followed by a QRS complex of different morphology than intrinsic QRS
- A ventricular demand (VVI) pacemaker senses and paces only in the ventricle and is oblivious to native atrial activity. If constant ventricular pacing is noted throughout the tracing, it is impossible to distinguish ventricular demand from asynchronous ventricular pacing. Thus, the diagnosis of ventricular demand pacing requires evidence of appropriate inhibition of pacemaker output in response to a native QRS (at least one).
- Appropriately sensed ventricular activity (QRS complex) resets pacemaker timing clock. After an interval of time (V-V interval) with no sensed ventricular activity, a ventricular paced beat is delivered and a new cycle begins.
- A spontaneous QRS arising before the end of the V-V interval is sensed and the ventricular output of the pacemaker is inhibited. A new timing cycle begins.
- For rate-responsive VVI-R pacemakers, ventricular paced rate increases with activity (up to a defined upper rate limit).

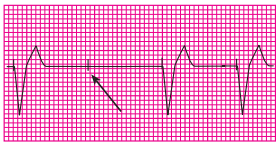
92. Dual-chamber pacemaker (DDD)



- Atrial and ventricular pacing and sensing
- For atrial sensing, need to demonstrate inhibition of atrial output and/or triggering of ventricular stimulus in response to intrinsic atrial depolarization
- If pacemaker rate exceeds rate of intrinsic rhythm, there will be atrial (A) and ventricular (V) paced beats with defined intervals between the A and V spikes (A-V interval) and from the V spike to the subsequent A spike (V-A interval);
- Following V sensed activity (either QRS or paced [V] beats), the timing clock is reset. If intrinsic atrial activity (P) is sensed prior to the end of the V-A interval, atrial output of the pacemaker will be inhibited. If no intrinsic atrial activity (P) is sensed by the end of the V-A interval, an atrial paced beat will occur.
- Following atrial sensed activity (either intrinsic (P) or paced (A) beats), the timing clock is reset. If intrinsic ventricular activity (QRS) is sensed prior to the end of the AV interval, ventricular output of the pacemaker will be inhibited. If no intrinsic ventricular activity (QRS) is

sensed by the end of the A-V interval, a ventricular paced beat will occur.

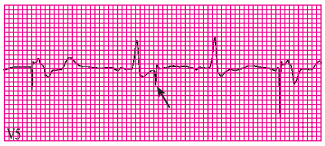
93. Pacemaker malfunction, not constantly capturing (atrium or ventricle)



- Pacing spike is not followed by appropriate depolarization (at a time when myocardium is not refractory).
- May be due to lead displacement, perforation, increased pacing threshold (from MI, flecainide, amiodarone, hyperkalemia), lead fracture or insulation break, pulse generator failure (from battery depletion), or inappropriate reprogramming.

Note: Rule out "pseudo-malfunction" (i.e., pacer stimulus falls into refractory period of ventricle)

94. Pacemaker malfunction, not constantly sensing (atrium or ventricle)



- Pacemakers in "inhibited" mode: Failure of pacemaker to be inhibited by an appropriate intrinsic depolarization
- Pacemakers in "triggered" mode: Failure of pacemaker to be triggered by an appropriate intrinsic depolarization
- Pacemaker timing is not rest by intrinsic or ectopic beat, resulting in asynchronous firing of pacemaker (paced rhythm competes with the intrinsic rhythm)
- Occurs with low amplitude signals (esp. VPCs) and inappropriate programming of the sensitivity. All causes of failure to capture (item 93) can also cause fail to sense.

Note: Can often be corrected by reprogramming the sensitivity of the pacemaker.

Note: Watch for "pseudomalfunction" (i.e., pacer stimulus falls into refractory period of ventricle)

Note: Premature depolarizations may not be sensed if they:

- Fall within the programmed refractory period of the pacemaker
- Have insufficient amplitude at the sensing electrode site

Note: Any stimulus falling early within the QRS complex probably does not represent sensing malfunction; commonly seen with right ventricular electrodes in RBBB.

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