

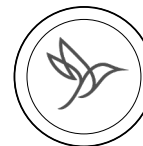


Evaluating the 12-lead ECG: Concepts for interpretation of acute and chronic changes with case study analysis

Sally K. Miller

PhD, APRN, AGACNP-BC, AGPCNP-BC,
FNP-BC, PMHNP-BC, FAANP

1



Sally K. Miller

PhD, APRN, AGACNP-BC, AGPCNP-BC,
FNP-BC, PMHNP-BC, FAANP

Faculty,

Fitzgerald Health Education Associates, Lawrence, MA

Clinical practice, Sahara Family Practice
and iCarePsychiatry, Las Vegas, NV

Clinical Professor, Drexel University CNHP, Philadelphia, PA

2

Disclosure

No real or potential conflict of interest
to disclose.

No off-label, experimental or investigational
use of drugs or devices will be presented.

3

Objectives

At the conclusion
of this session,
the attendee will
be able to:

- 01 Describe a systematic approach
to assessing the 12-lead ECG.
- 02 Identify critical ECG changes seen
in ischemia, injury, and infarct.
- 03 Analyze axis using the hexaxial plot.

4

Objectives (continued)

At the conclusion of this session, the attendee will be able to: (cont.)

- 04 Evaluate morphologic changes consistent with chronic disease and insult.
- 05 Perform a case study analysis of conditions characterized by ECG changes.

5

Tips



- References
 - Listed throughout and at the end of the presentation
- To facilitate your learning
 - Specific tables/images can be viewed full page at the end of your handout.

6

Vector Analysis and Axis Determination

- Initiation and propagation – Sequence of cardiac activation
 - The SA node depolarizes spontaneously.
 - Atrial muscle depolarizes rapidly.
 - The wave of depolarization funnels to AV node where it is delayed.
 - Current travels to the bundle of His.

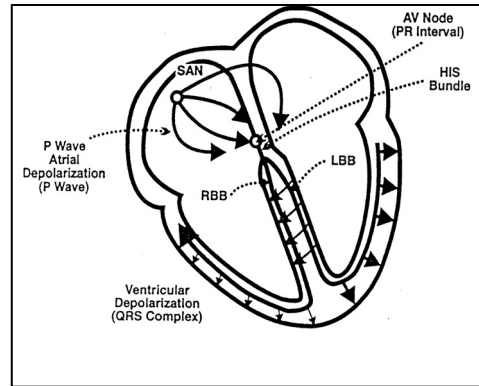
7

Vector Analysis and Axis Determination (continued)

- Initiation and propagation (cont.)
 - Current divides into right and left bundles.
 - Depolarization of interventricular septum is left to right.
 - Current moves simultaneously through the right and left bundle branches.
 - Ventricles repolarize.

8

Initiation and Propagation



9

Limb Leads

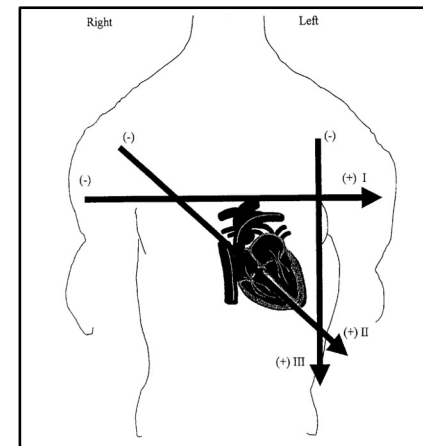
- Vector is a voltage force that has direction as well as amplitude.
 - Electrical events in the heart occur in three dimensions.
 - ECG paper converts those dimensions to a two-dimensional picture – hence 12 leads.
 - Using 12 leads allows us to visualize events from the anterior, inferior, and lateral perspective.

10

Limb Leads (continued)

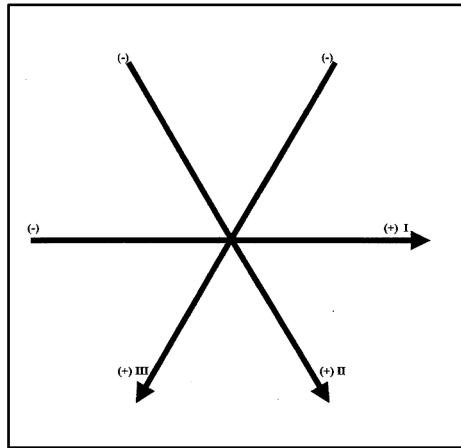
- The limb leads
 - Offer a lateral and inferior view
 - Axis is plotted based on the hexaxial system.
 - Find the limb lead with the voltage closest to 0.
 - Identify its right-angle lead.
 - On the ECG, see if that lead is positive (+) or negative (-).

11



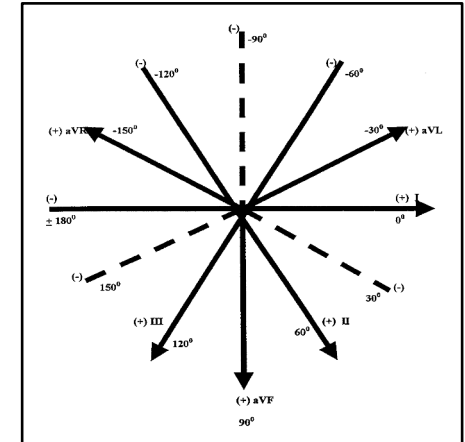
12

The Hexaxial Plot



13

The Hexaxial Plot



14

Axis Determination

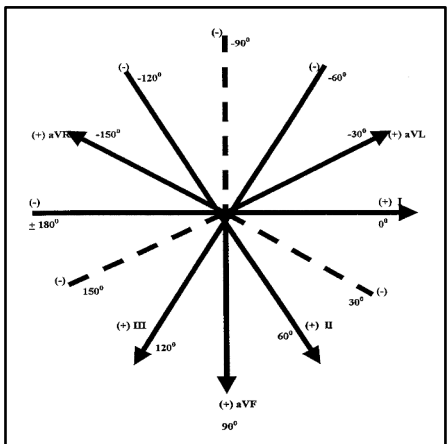
- Determine the corresponding direction on the hexaxial plot.
- Because the net vector is normally down and to the left, the normal axis should be in the vicinity of 60° ; a range of -30° to $+110^\circ$ is normal.
- If the axis deviates to the left of -30° , this represents a left axis deviation.
- If the axis deviates to the right of $+110^\circ$, this represents a right axis deviation.

15



16

The Hexaxial Plot



17



18



19

The System of ECG Interpretation

1. Rate
2. Rhythm
3. Intervals
4. Axis
5. Morphology

20

Rate

- Determine the R-R interval.
- Each large square is 0.2 seconds.
- Divide the number of large squares between R waves into 300 to determine rate.
- Normal rate is 60 to 100 bpm.

21



22

Rhythm

- Rhythm interpretation is presumed as a prerequisite to this presentation.
- The second step in 12-lead ECG assessment is identification of the rhythm, e.g., NSR, SB, ST, A-V block, atrial dysrhythmia, ventricular dysrhythmia, etc.

23

Intervals

- P-R interval represents A-V conduction.
 - Should be 0.12 to 0.22 seconds
 - Prolonged P-R interval indicates a first-degree block.
 - Shortened P-R interval indicates a junctional rhythm with retrograde conduction.
- QRS duration represents ventricular depolarization.
 - Should be <0.12 seconds
 - Prolonged duration indicates a block in the bundle branches or a ventricular ectopic foci.

24

Intervals (continued)

- Q-T(c) interval represents repolarization of the ventricle
 - Q-T interval should be $< \frac{1}{2}$ the R-R interval
 - Long Q-T interval increases the risk of ventricular dysrhythmia and sudden death

25

QRS Axis

- Identify the lead where the net voltage of the QRS is closest to 0.
- Look for the perpendicular lead.
- If the deflection of the perpendicular lead is +, then the axis is at the positive end of the pole.
- If the deflection of the perpendicular lead is -, then the axis is toward the negative end of the pole.

26

Abnormalities Caused by Drugs and Metabolic Conditions

27

Abnormalities of Rate

- Sinus bradycardia
 - Beta-adrenergic antagonists
 - Calcium channel antagonists
 - Digitalis
 - Adenosine
 - Hypoxemia
 - Hypothyroidism
 - Hypothermia
 - Hyperkalemia

28

Sinus Tachycardia

- Catecholamines
- Caffeine
- Amphetamines
- Hyperthyroidism
- Anemia
- Fever

29

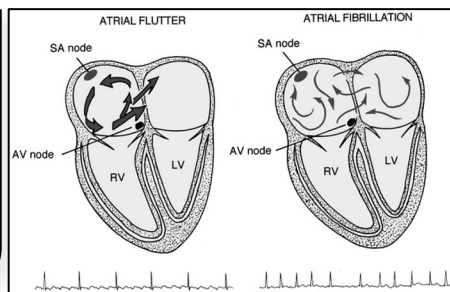
Heart Block

- Digitalis
- Beta adrenergic blockers
- Calcium channel blockers
- Adenosine
- Hyperkalemia

30

Atrial Flutter/Fibrillation

- Both are conditions of aberrant atrial discharge.
- Some degree of physiologic AV block typically present.



31

Ventricular Tachycardia – Torsade de pointe

- Electrical dysfunction**
- Class I antidysrhythmic drugs
- Amiodarone
- Phenothiazine derivatives
- Tricyclic overdose
- Long QT syndrome

32

Ventricular Fibrillation

- Most antidysrhythmic drugs
- Digoxin
- Tricyclic overdose
- Hypokalemia
- Hypomagnesemia
- Hypocalcemia

33

Analysis of the 12-Lead ECG Part 2

Morphologic Changes

34

Morphologic Changes

- The V leads (V_1 to V_6), aka precordial leads, represent the anterior wall of the heart.
 - V leads may be referred to as “anterior” leads.
 - The limb leads represent the inferior and lateral walls of the heart.

<u>Inferior Wall</u>	<u>Lateral Wall</u>	<u>Anterior Wall</u>
II, III, aVF	I, aVL, (V_6)	V leads

35

P Wave Abnormalities

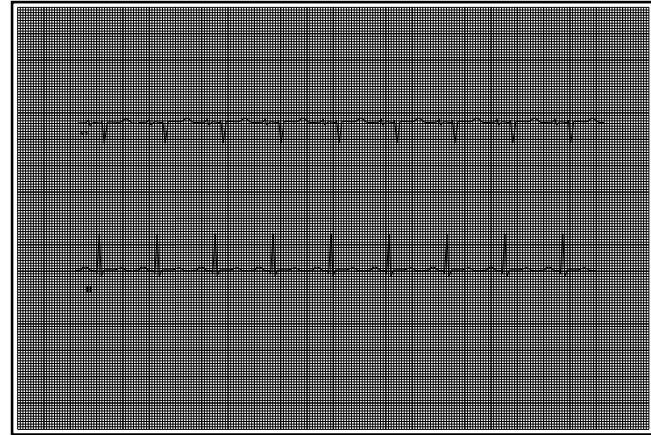
- The P wave represents atrial depolarization. An abnormal P wave would logically suggest an atrial abnormality.
- Left atrial abnormalities
 - Biphasic P wave in V_1 is most common; must be 1×1 mm to be significant
 - Biphasic P waves occur in conditions that increase LVEDP.
 - CHF, LVH, hypertensive heart disease may all cause this abnormality.

36

P Wave Abnormalities (continued)

- Broad, notched P waves in limb leads suggest left atrial dilation.
- These occur in conditions such as mitral stenosis and regurgitation.

37



38

Right Atrial Abnormalities

- P wave >2.5 mm in any lead
- Occurs in conditions such as lung disease and pulmonary artery hypertension

39



40

QRS Abnormalities

- Remember the normal flow of current and how it reflects on an ECG.
 - ECG will record normal left to right activation. V_1 initial deflection is positive
 - LV depolarization produces a downward deflection in V_1 .
 - LV and RV depolarize simultaneously, so LV depolarization dominates the picture.
 - After ventricles repolarize, return to baseline.

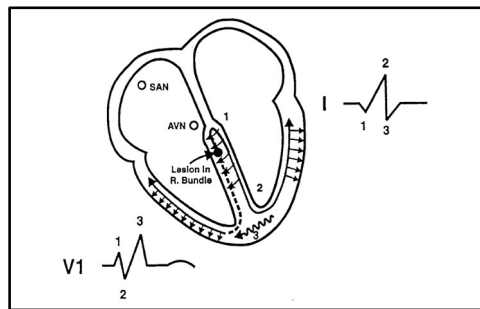
41

QRS Abnormalities (continued)

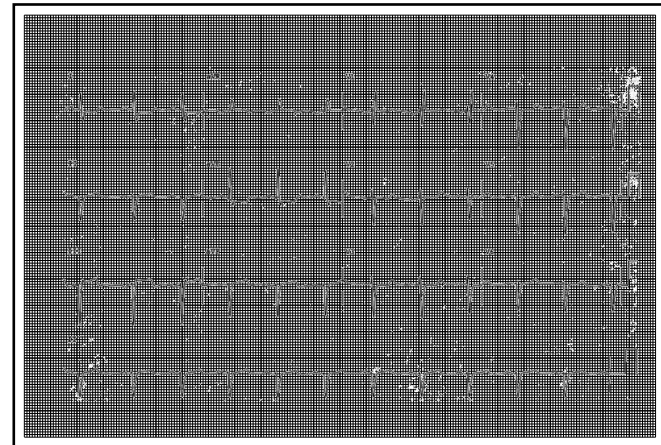
- Right bundle branch block (RBBB)
 - QRS >0.12 seconds
 - Current normally moves left to right in the intraventricular septum.
 - ECG will record normal left to right activation in V_1 .
 - This is followed by normal LV activation, but right bundle is blocked.
 - Late current LV to RV results in second upward deflection in V_1 .
 - After RV activation, return to baseline.

42

Right Bundle Branch



43



44

Incomplete RBBB

- Usually a normal variant
- May reflect RV hypertrophy or dilation
- Very common with atrial septal defect
- RSR pattern in V_1
- QRS is <0.12 seconds.

45



46

Left Bundle Branch Block

- Sequence is opposite RBBB.
- Loss of initial normal left to right activation
- Interventricular septum is activated from right to left, causing an abnormal upward deflection in the left lateral leads.
- QRS is >0.12 seconds.
- Septum is activated from right to left, but the blocked left bundle limits the impulse.

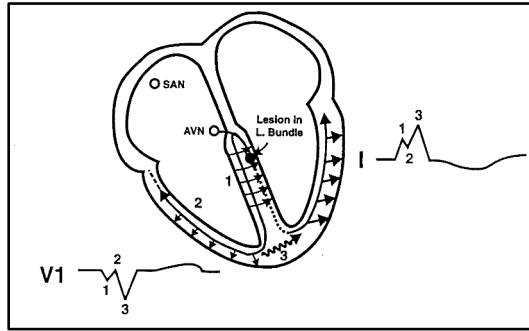
47

LBBB (continued)

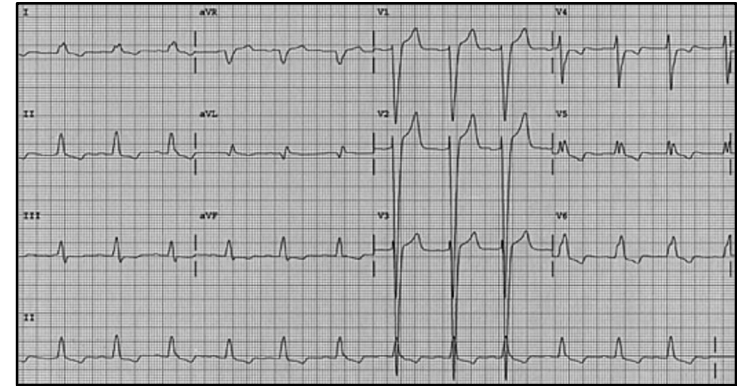
- Right side depolarizes first. It is thin walled, so it produces a small current.
- After RV depolarization, the current travels around to left ventricle.
- Late left depolarization produces terminal QRS force.

48

LBBB



49



50

Fascicular Blocks

- The left bundle branch divides into two fascicles: the anterior and posterior.
- LBBB is when both fascicles are blocked. QRS is wider than 0.12 seconds.
- When only one of the fascicles is blocked, the diagnosis is either “left anterior fascicular block” or “left posterior fascicular block.”

51

Fascicular Blocks (continued)

- Diagnosis of fascicular block is made when there is a shift in axis.
- The QRS is not necessarily wider than normal.
- LAFB is extreme left axis deviation, at least -45° and not caused by IWMI.
- LPFB is diagnosed by right axis deviation, at least $>90^\circ$, usually $>110^\circ$ to 120° .

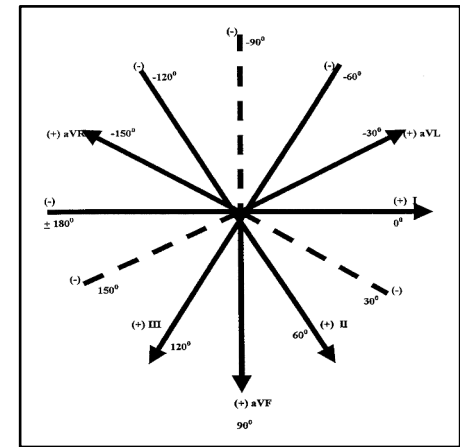
52

LAFB



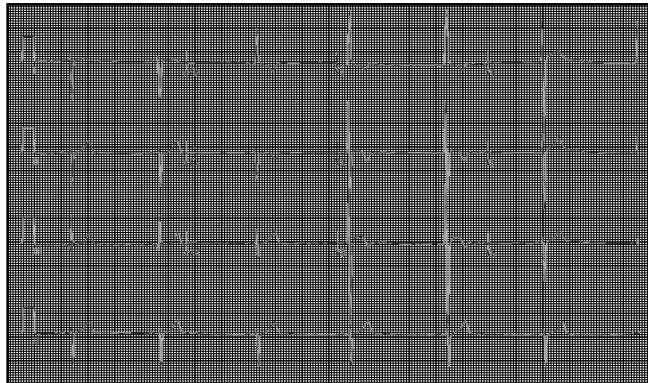
53

The Hexaxial Plot



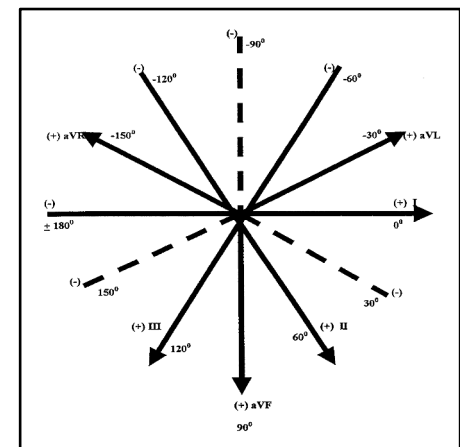
54

LPFB



55

The Hexaxial Plot

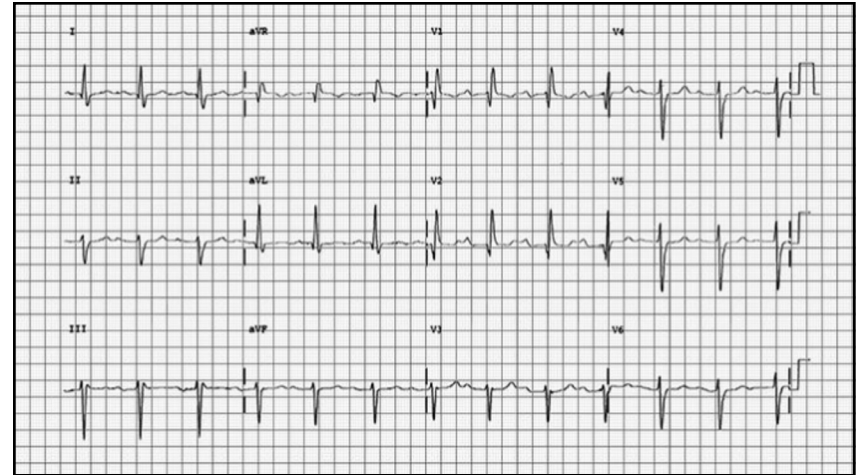


56

Bifascicular Block

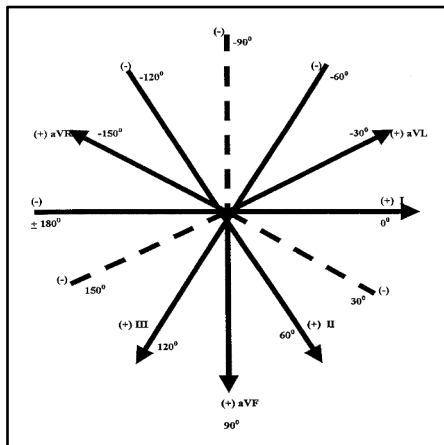
- A right bundle branch block
 - RSR pattern in V_1
 - QRS >0.12 seconds
- A coincident block of either the left anterior or posterior fascicle
- AKA – a RBBB with either left or right axis deviation

57



58

The Hexaxial Plot



59

Left Ventricular Hypertrophy

- When you have hypertrophy of muscle a variety of changes occur.
 - The larger muscle mass produces more voltage.
 - The increased size changes axis of electrical conduction.
 - Resultant high pressure in left atria may change character of voltage movement through left atria.

60

Romhilt + Estes Point Score System

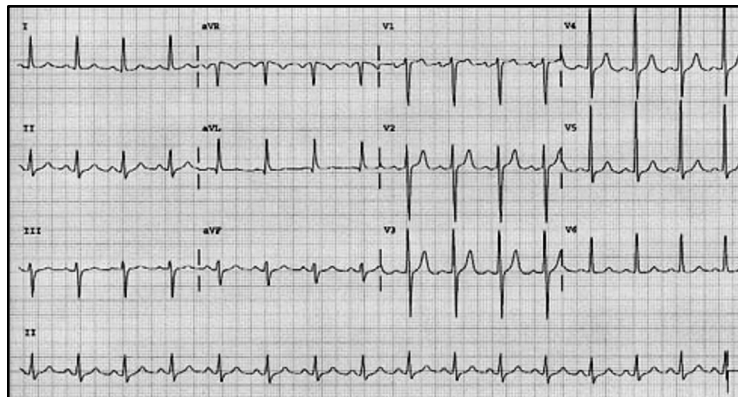
- Amplitude – any of the following= **3 points**
 - Largest R or S wave in any limb lead ≥ 20 mm
 - S wave in V_1 or $V_2 \geq 30$ mm
 - R wave in V_5 or $V_6 \geq 30$ mm
- ST-T strain (change in lateral leads)
 - On digitalis= **1 point**
 - Not on digitalis= **3 points**

61

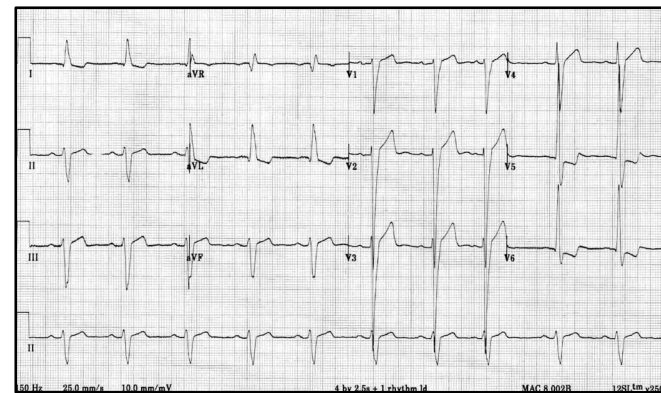
Romhilt + Estes Point Score System (continued)

- Left atrial abnormality= **3 points**
 - LAD $> -30^\circ$ = **2 points**
 - QRS duration ≥ 0.09 sec= **1 point**
 - Intrinsicoid deflection in V_5 or $V_6 \geq 0.05$ sec= **1 point**
- 5 or more points= LVH
4 points= Probable LVH

62



63



64

Right Ventricular Hypertrophy

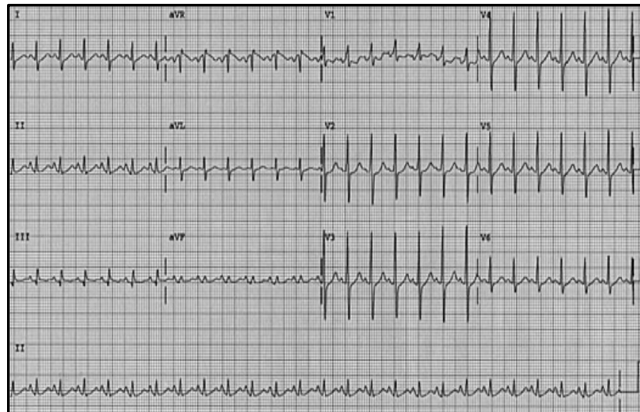
- Most voltage in the QRS generated by LV
- When the right ventricle hypertrophies significantly, it can generate a lot of voltage. A more “rightward shift” occurs in V_1 .

65

RVH

- Diagnostic criteria
 - R/S in $V_1 \geq 1$ **or**
 - R in $V_1 + S$ in $V_6 > 10.5$ mm
- Supportive criteria
 - Right axis deviation $\geq 110^\circ$
 - Right atrial abnormality
 - ST depression + T wave inversion in V_1 or V_2

66



67

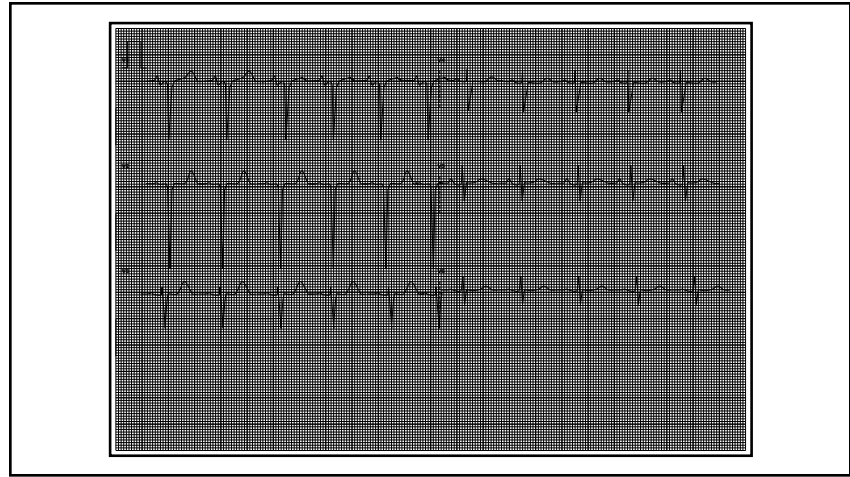
Poor R Wave Progression

- In the normal ECG, the transition from negative V_{1-2} to positive V_{5-6} deflection occurs during V_{3-4} .
- A delay or absence of this transition on ECG just means that anatomically the transition point has moved.

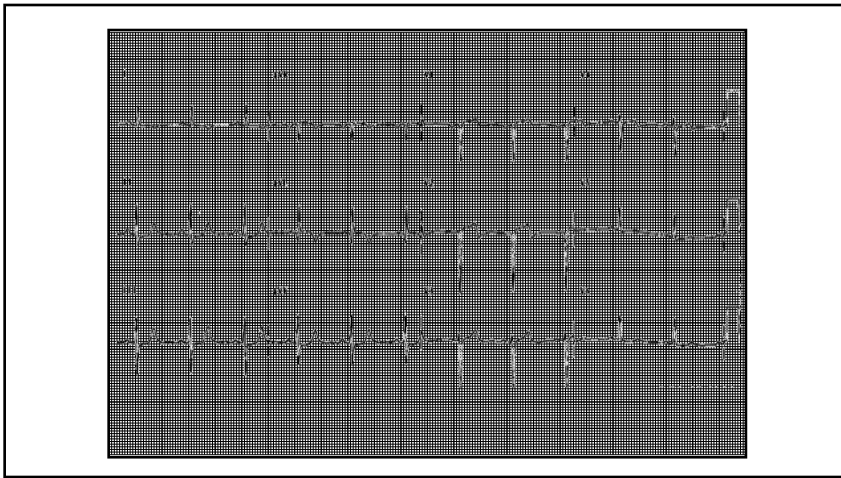
68



69



70



71

Causes of PRWP

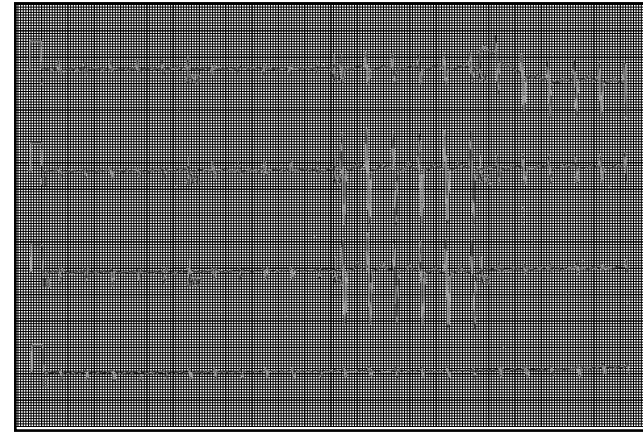
- COPD
- LV dilation
- Anterior wall MI
- Misplaced precordial leads

72

Low QRS Voltage

- QRS amplitude <5 mm in all limb leads
- QRS amplitude in V leads usually <10 mm, but not necessary for diagnosis

73



74

Causes of Low QRS Voltage

- Effusion
- Cardiomyopathy
- Hypothyroidism
- Obesity
- Emphysema
- Normal variant

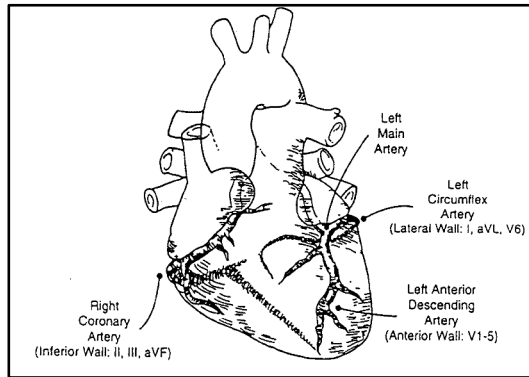
75

ST-T Wave Abnormalities

- Ischemia and infarction tend to be regional events.
- Depending upon anatomy, there may be some overlap.
- An event in a large RCA that loops around the lateral wall might cause inferolateral ECG changes
- An event in a large anterior descending artery that has branches to the lateral wall may cause an anterolateral event
- An event in the left main artery may cause an anterolateral event
- Global ST-T changes are more typically caused by pericarditis.

76

Arteries and Corresponding Leads



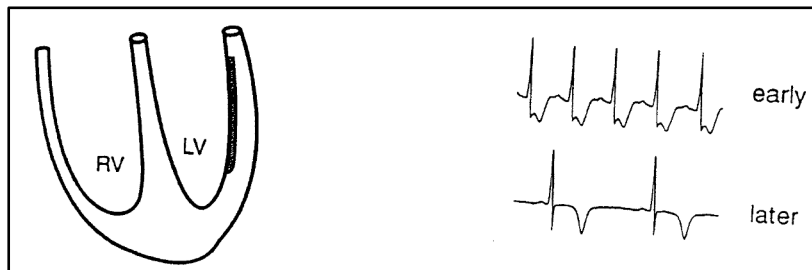
77

ST Segment Depression

- Stenosed artery with some retrograde flow
- O₂ demand exceeds supply.
- Subendocardial ischemia
- Region of myocardium furthest from the stenosed artery is occluded.
- If ischemia persists and myocardial injury occurs, a subendocardial MI occurs.
 - Later changes will show T wave inversion.

78

Subendocardial Injury



79

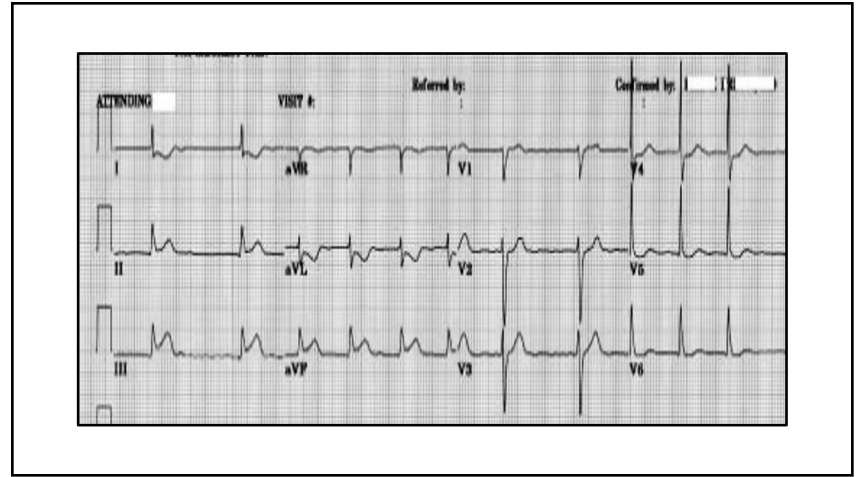
ST Segment Elevation

- Most common cause is transmural MI.
- Affected artery is totally occluded.
- Is the primary ECG indication for thrombolytic therapy
- Prinzmetal's angina (acute vasospasm) usually produces complete vessel occlusion; will produce ST segment elevation if ECG recorded during event
- The size of the inferior and lateral MI is proportional to the sum of the elevation in the appropriate leads.
- The size of the anterior wall MI is proportionate to the number of anterior leads with elevation.

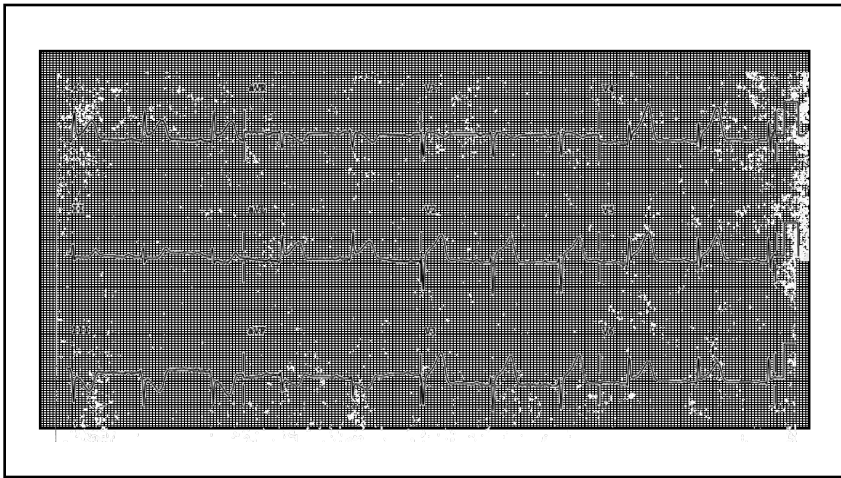
80



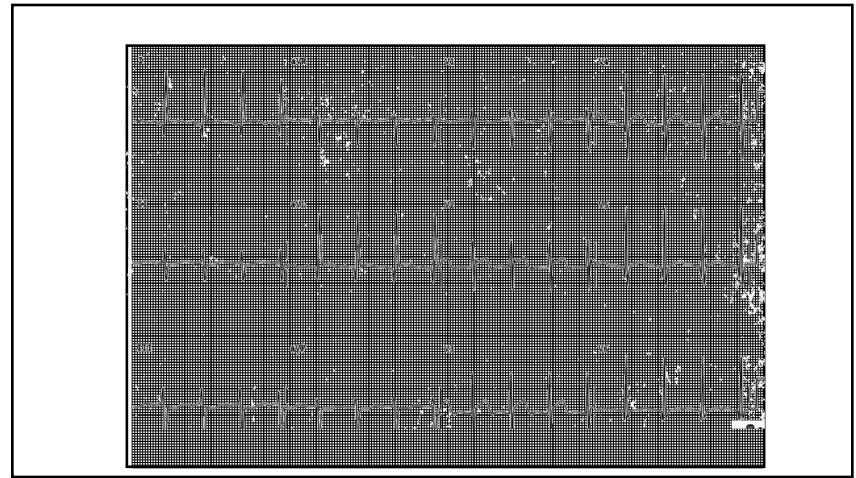
81



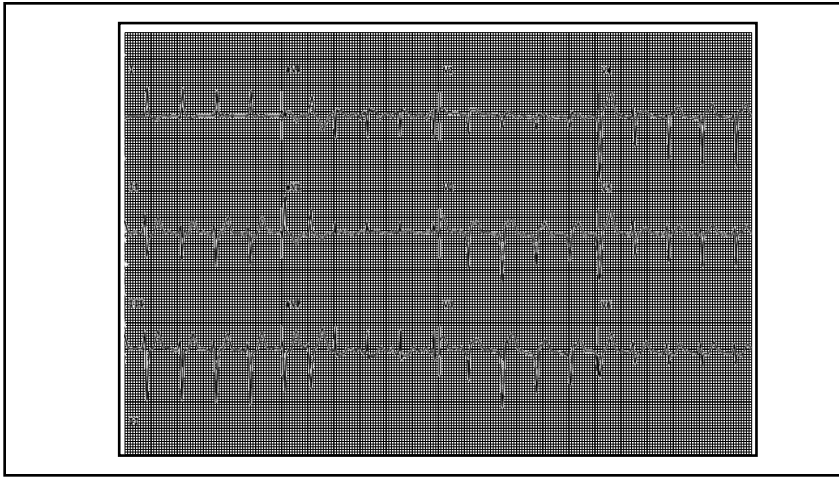
82



83



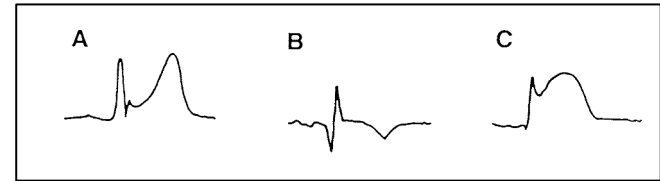
84



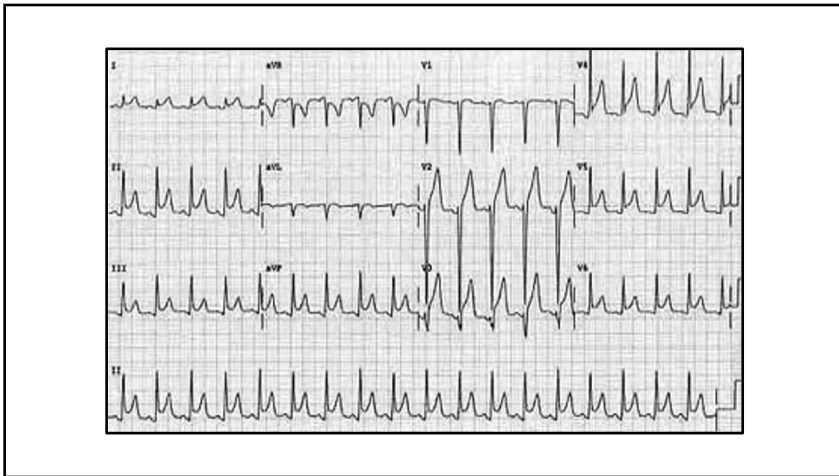
85

Other Causes of ST Elevation

- There are causes of ST elevation that are not specific to myocardial damage.
 - Pericarditis
 - Early repolarization



86



87

Non-specific ST Changes

- A label typically applied to ST depression that is not placed in a clinical context
- Specific ST changes
 - During exercise ECG
 - During chest pain

88

T Wave Inversion

- Reflects altered repolarization of ventricular muscle during ischemia/injury event
- May reflect permanent injury with scar formation and loss of muscle; permanent atypical path of repolarization

89

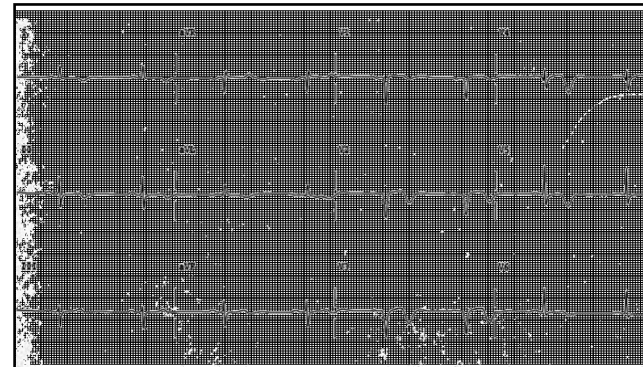


90

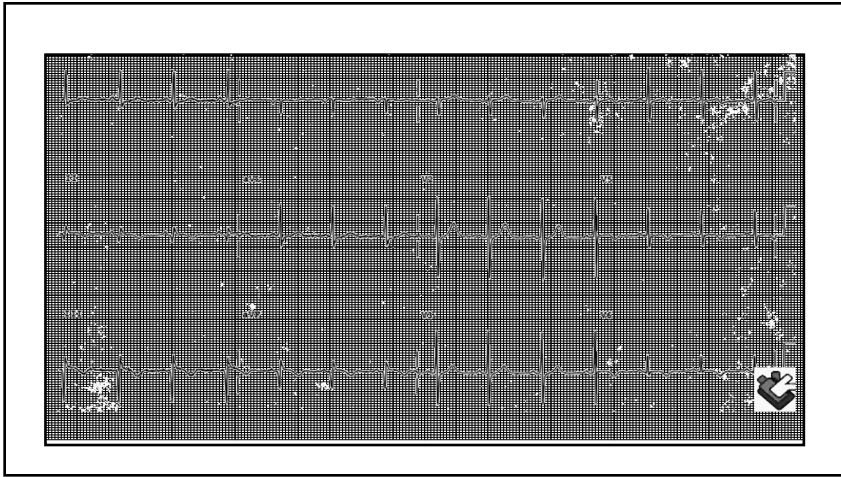
Q Waves

- Initial negative deflection of the QRS complex
- Must be 1 mm deep and 1 mm wide to be significant
- May be normal in leads III and V₆
- A Q wave indicates transmural injury.

91



92

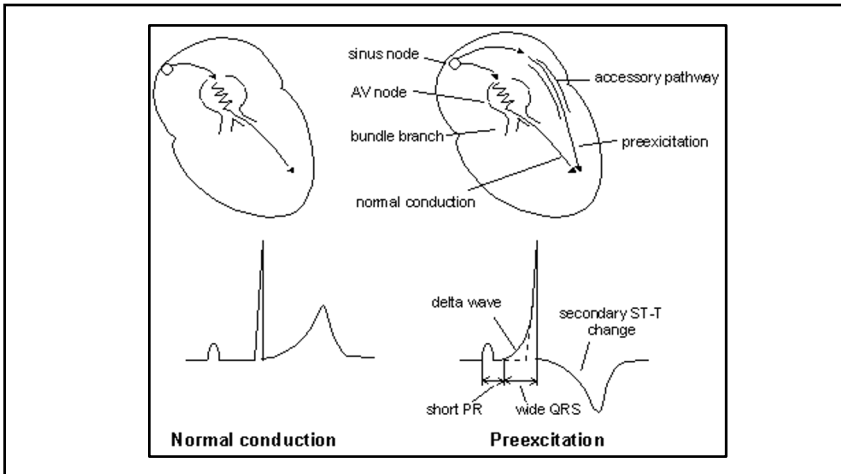


93

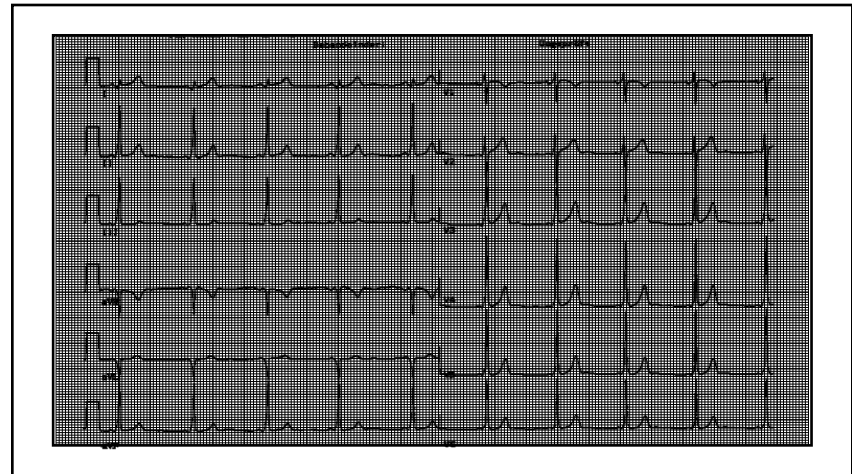
WPW Syndrome

- Activation of accessory pathway results in preexcitation of the ventricle
- Delta wave may appear to be a Q wave
- No history of MI
- Normal echocardiogram
- Short P-R interval

94



95



96

Case Study

41-year-old Female

97

Chief Complaint

- A 41-year-old female presents with a chief complaint of chest pain and getting out of breath too easily.
- She power walks/jogs each day and actually had to stop because she was so out of breath.

98

History of Present Illness

- The patient reports that she is generally in good health. She has just noticed that in the last few weeks or so she gets tired easily.
- She was finally prompted to seek care when she had to stop her job for SOB.

99

History of Present Illness (continued)

- She admits to a kind of “dull” chest discomfort that is hard to describe. She is aware of it. It comes and goes but it doesn't really stop her from doing anything.
- It is not sharp or easy to localize.

100

History of Present Illness

- She specifically denies
 - Radiation of the discomfort
 - Pain or discomfort to neck, arm, jaw
 - No associated sx, e.g., diaphoresis, n/v
 - Association with rest or activity

101

Review of Systems

- Otherwise, noncontributory
- She was queried specifically regarding history of
 - Constitutional sx
 - Other cardiopulmonary sx
 - Hemoptysis
 - Bleeding (skin, GI, GYN)

102

PMH/PSH

- Dyslipidemia
- Hypertension
- Gastric bypass procedure 2 years ago
 - Her two previous medical problems resolved entirely with wt loss.

103

Medications

- Vitamin B₁₂ 500 mcg daily
- Vitamin D and calcium combination supplement daily
- MVI daily

104

Family Hx

- Mother aged 70 y
 - Obesity
 - Dyslipidemia
 - T2DM
 - HTN
- Father died age 52 y of MI
- Brother aged 48 y
 - Obesity
 - HTN
 - T2DM

105

Social Hx

- Pt lives with her husband. She has no children.
- Works as a telephone tech support person
- Denies tobacco or recreational drug use
- Rare ETOH <6 × year
- Monogamous with husband

106

Case Study (continued)

Physical exam

- 41-year-old female 5'3" (160 cm) 164 lbs (74.4 kg)
- 97.5 °F (36.4°C) P=60 bpm, RR=16 bpm, BP=134/82 mm Hg
- Well-groomed
- In NAD
- Skin color normal, warm/dry

107

Case Study (continued)

Physical exam (cont.)

- CN II-XII grossly normal
- HEENT WNL
- Neck without nodes, bruit, thyromegaly but + minimal JVD
- Lung sounds are clear
- Cardiac exam reveals
 - A grade II/VI systolic murmur at 4th ICS LSB
 - Very loud S₂

108

Case Study (continued)

Physical exam (cont.)

- Peripheral pulses are normal.
- Examination of the extremities reveals 1+ pitting edema from mid-calf down. There is no hyperpigmentation.

109

Case Study (continued)

Physical exam (cont.)

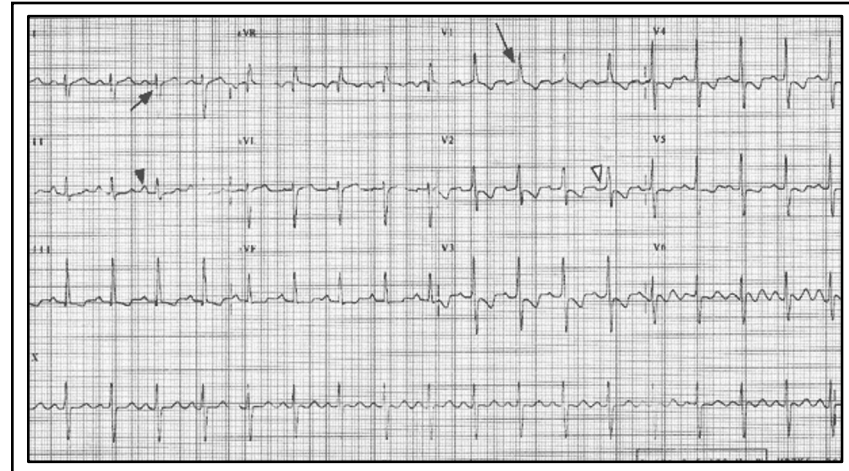
- The abdomen is basically normal. Organ palpation is limited due to large amount of excess skin.
 - Large well-healed scar is apparent.
 - No bruit
 - No organomegaly
 - No pulsations

110

Office Diagnostics

- Routine labs are drawn.
- 12-lead ECG
 - Right atrial abnormality
 - Right bundle branch block
 - Right axis deviation

111



112

Office Spirometry

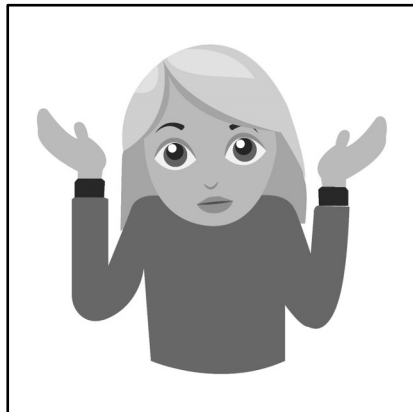
- Essentially WNL
- No obstructive dysfunction
- Total lung capacity (TLC) 72% predicted

113

Laboratory Results

- CMP was WNL.
- CBC
 - Significant for Hgb/ HCT of 17.8 g/dL (178 g/L)/ 53%
 - WBC differential was normal.

114



115

Case Study 44-year-old Male

116

Case Study: 44-year-old Male

- The patient presents with dull retrosternal chest pain.
 - Began acutely with a tearing sensation
 - 3 days duration
- Unable to "get comfortable"
- Denies any recent viral infection or significant medical history

117

Case Study (continued)

- No family history of cardiovascular disease
- The patient is taking no medications and he denies illicit drug use.

118

Case Study (continued)

Physical exam

- The patient is alert, uncomfortable, and afebrile.
- Blood pressure is 160/102 mm Hg.
- Equal and symmetric pulses in both carotid and brachial arteries.

119

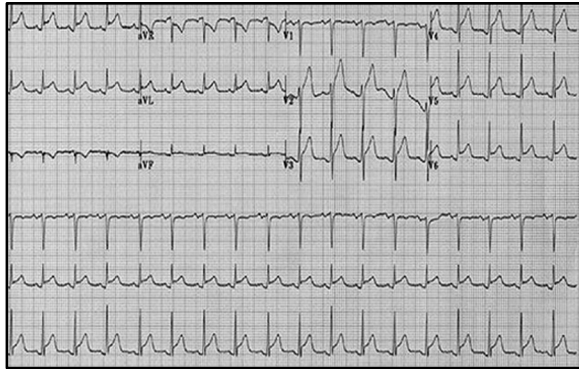
Case Study (continued)

Physical exam (cont.)

- There is a diastolic murmur in the aortic region.
 - No gallop or pericardial rub
- Heart sounds are distant.
- The patient's pulmonary, abdominal, and neurologic examinations are unremarkable.

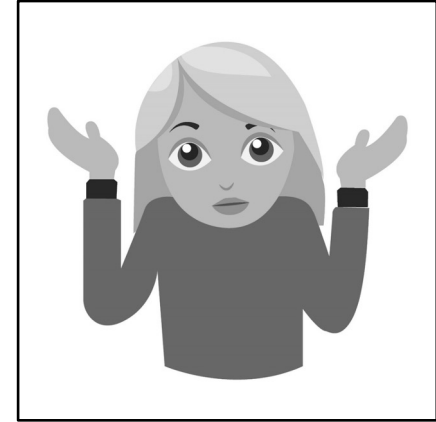
120

What are the findings on the ECG?



121

**Aortic
Dissection**



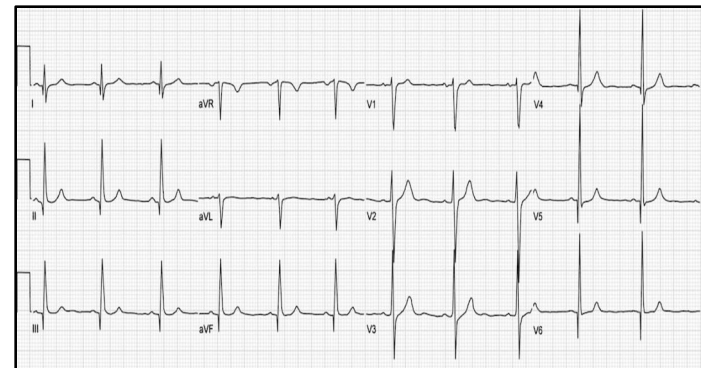
122

Myocarditis



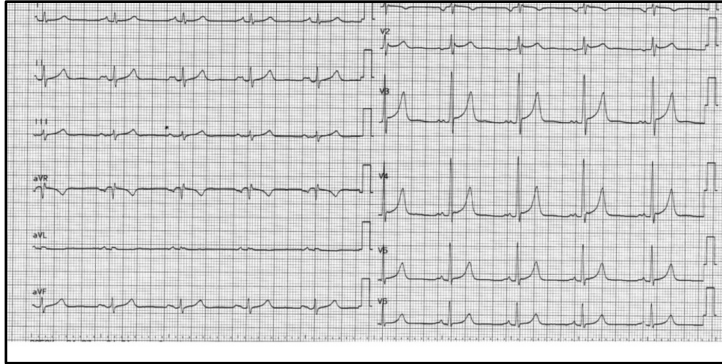
123

Hypertrophic Cardiomyopathy



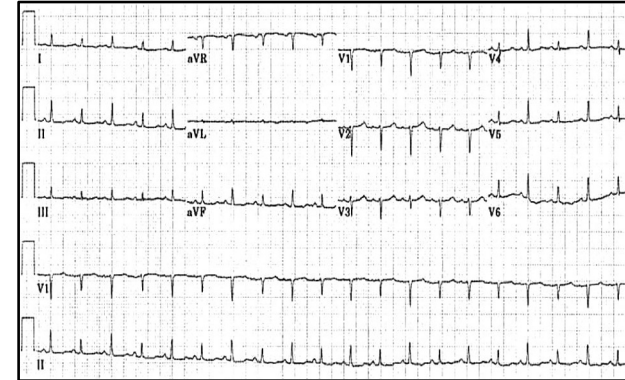
124

Mitral Valve Prolapse



125

Effusion



126

End of Presentation!

Thank you for your time and attention.

Sally K. Miller

PhD, APRN, AGACNP-BC, AGPCNP-BC,
FNP-BC, PMHNP-BC, FAANP

www.fhea.com

sally@fhea.com

127

References

- Miller, S.K. (2019). 12-lead electrocardiographic interpretation for nurse practitioners. *The Journal for Nurse Practitioners*, 15(1), 110-7.
- Shade, B. (2022). *Interpreting ECGs: A practical approach*. 3rd edition. NY: McGraw Hill.
- Thaler, M.S. (2015). *The Only EKG book you'll ever need* (8th ed.). Philadelphia: LWW.
- Yanowitz, F.G. (2015). ECG Learning Center. University of Utah. Last accessed November 2022. <http://ecg.utah.edu/>

128

Image sources

All images used with permission from Dr. S.K. Miller, except for slides 116 and 123 which are from Shutterstock.

129

Copyright Notice

Fitzgerald Health Education Associates
All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopy, recording or any information storage and retrieval system, without permission from Fitzgerald Health Education Associates

Requests for permission to make copies of any part of the work should be mailed to:

Fitzgerald Health Education Associates
15 Union Street, Suite 512
Lawrence, MA 01840

130

Statement of Liability

- The information in this program has been thoroughly researched and checked for accuracy. However, clinical practice and techniques are a dynamic process and new information becomes available daily. Prudent practice dictates that the clinician consult further sources prior to applying information obtained from this program, whether in printed, visual or verbal form.
- Fitzgerald Health Education Associates disclaims any liability, loss, injury or damage incurred as a consequence, directly or indirectly, of the use and application of any of the contents of this presentation.
- All websites listed active at the time of publication.

131

Fitzgerald Health Education Associates

15 Union Street, Suite 512
Lawrence, MA 01840
978.794.8366 Fax-978.794.2455

Website: fhea.com

Learning & Testing Center: fhea.com/npexpert



https://www.instagram.com/fitzgerald_health_ed/



www.facebook.com/fitzgeraldhealth



@npcert

132