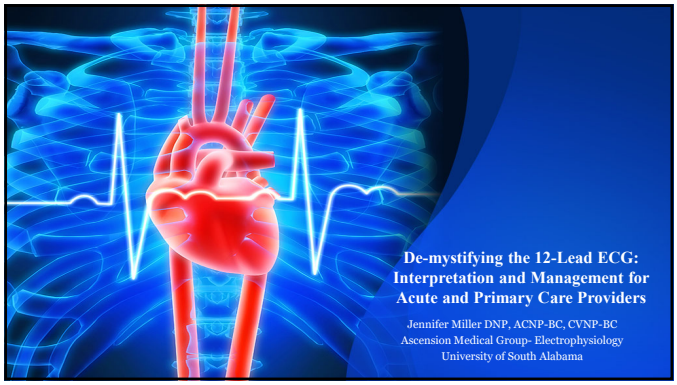
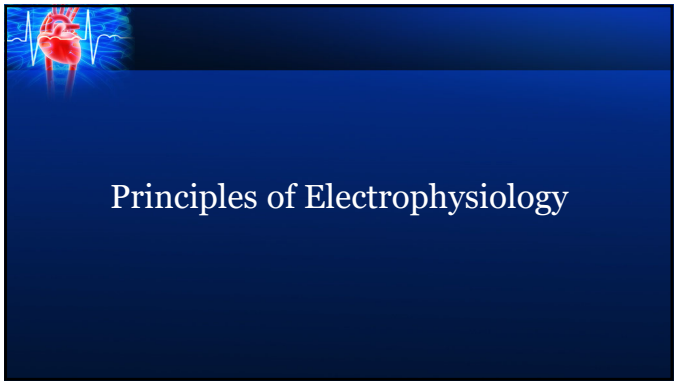
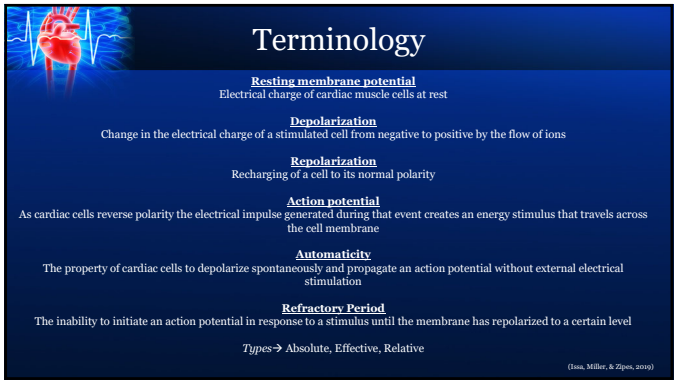

De-mystifying the 12-Lead ECG: Interpretation and Management for Acute and Primary Care Providers
Jennifer Miller DNP, ACNP-BC, CVNP-BC
Ascension Medical Group- Electrophysiology
University of South Alabama

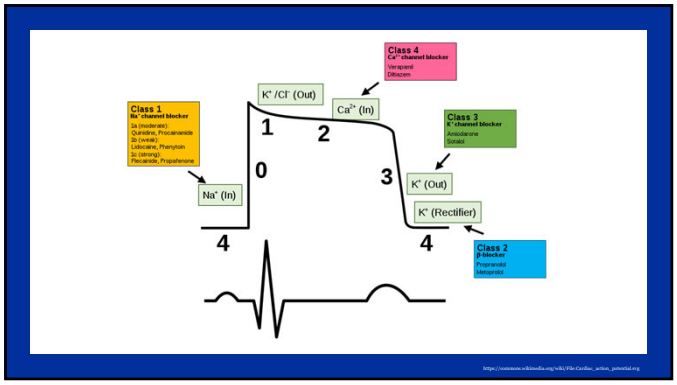
1


Principles of Electrophysiology

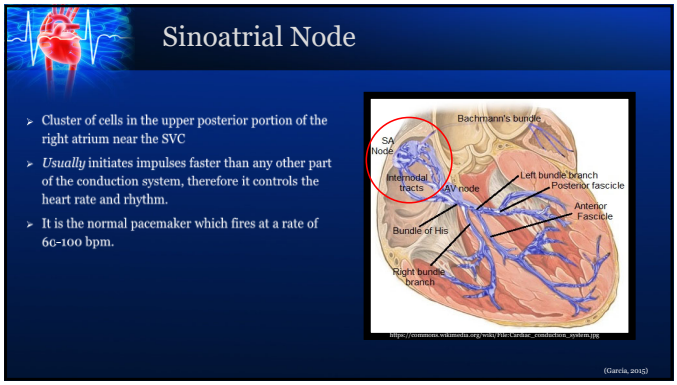
2


Terminology
Resting membrane potential
Electrical charge of cardiac muscle cells at rest
Depolarization
Change in the electrical charge of a stimulated cell from negative to positive by the flow of ions
Repolarization
Recharging of a cell to its normal polarity
Action potential
As cardiac cells reverse polarity the electrical impulse generated during that event creates an energy stimulus that travels across the cell membrane
Automaticity
The property of cardiac cells to depolarize spontaneously and propagate an action potential without external electrical stimulation
Refractory Period
The inability to initiate an action potential in response to a stimulus until the membrane has repolarized to a certain level
Types→ Absolute, Effective, Relative
(Issa, Miller, & Zipes, 2019)

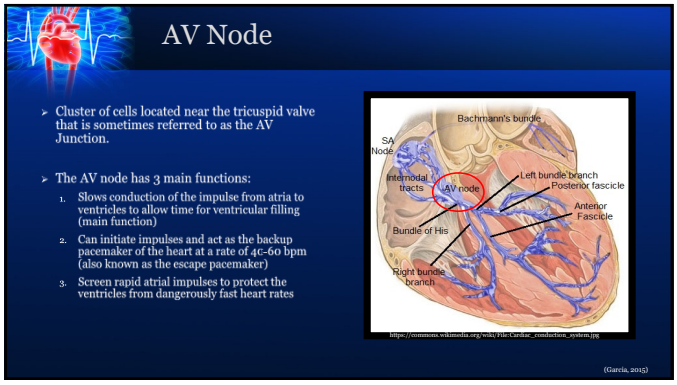
3


Class 4
Ca2+ channel blocker
Verapamil
Diltiazem
K+ /Cl- (Out)
Ca2+ (In)
Class 1
Na+ channel blocker
1a (moderate):
Quinidine, Procainamide
1b (weak):
Lidocaine, Phenytoin
1c (strong):
Flecainide, Propafenone
1
2
Class 3
K+ channel blocker
Amiodarone
Sotalol
0
3
Na+ (In)
K+ (Out)
K+ (Rectifier)
4
4
Class 2
β-blocker
Propranolol
Metoprolol

4


Sinoatrial Node
Cluster of cells in the upper posterior portion of the right atrium near the SVC
Usually initiates impulses faster than any other part of the conduction system, therefore it controls the heart rate and rhythm.
It is the normal pacemaker which fires at a rate of 60-100 bpm.
Bachmann's bundle
SA Node
Internodal tracts
AV node
Left bundle branch
Posterior fascicle
Anterior Fascicle
Bundle of His
Right bundle branch
(Garcia, 2015)

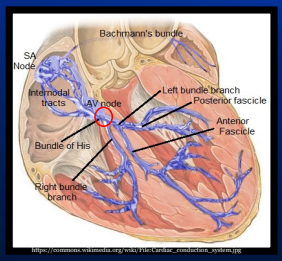
5


AV Node
Cluster of cells located near the tricuspid valve that is sometimes referred to as the AV Junction.
The AV node has 3 main functions:
1. Slows conduction of the impulse from atria to ventricles to allow time for ventricular filling (main function)
2. Can initiate impulses and act as the backup pacemaker of the heart at a rate of 40-60 bpm (also known as the escape pacemaker)
3. Screen rapid atrial impulses to protect the ventricles from dangerously fast heart rates
Bachmann's bundle
SA Node
Internodal tracts
AV node
Left bundle branch
Posterior fascicle
Anterior Fascicle
Bundle of His
Right bundle branch
(Garcia, 2015)

6

Bundle of HIS

- Located partially in the RA and in the upper portion of the interventricular septum
- Connects the AV node and the 2 bundle branches



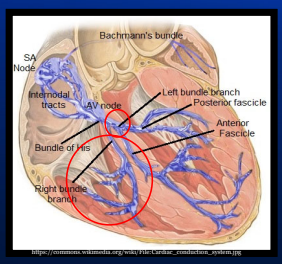
https://commons.wikimedia.org/wiki/File:Cardiac_conduction_system.jpg

(Garcia, 2015)

7

Bundle Branches

- Fibers located in the septum that carry impulses into the right and left ventricles.
- Inherent rate is approximately 40-45 bpm
- Left Bundle Branch
  - Innervates the LV and the left side of the intraventricular septum
  - Gives rise to the LAF & LPF
- Right Bundle Branch
  - Innervates the RV and the right side of the intraventricular septum
  - Gives rise to the Purkinje Fibers



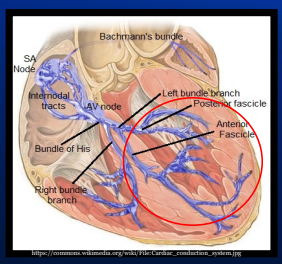
https://commons.wikimedia.org/wiki/File:Cardiac_conduction_system.jpg

(Garcia, 2015)

8

Fascicles & Purkinje Fibers

- Left Anterior Fascicle (LAF)
  - Innervates the anterior and posterior LV via Purkinje cells
  - Single stranded
- Left Posterior Fascicle (LPF)
  - Innervates the posterior and inferior LV via Purkinje cells
  - Fan-like structure
- Purkinje Fibers
  - Hair like fibers that spread out from the bundle branches into the ventricles
  - Innervate myocardial cells directly
  - Initiate ventricular depolarization
  - Pace at a rate of 26-40 bpm

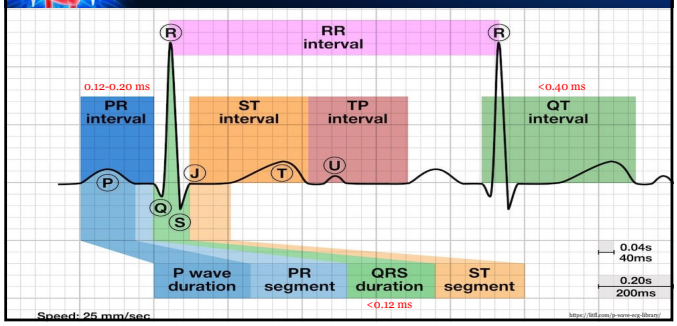


https://commons.wikimedia.org/wiki/File:Cardiac_conduction_system.jpg

(Garcia, 2015)

9

Waves & Intervals



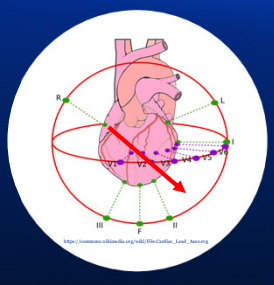
10

Vectors & Lead Placement

11

Vectors

- Demonstrates the strength & direction in which action potentials generated during depolarization are moving
- The heart produces many action potentials moving in various directions during depolarization
- Normal heart→ Left to right, superior to inferior
- The sum of all vectors = the electrical axis of the heart



(Garcia, 2015)

12

Electrical Activity

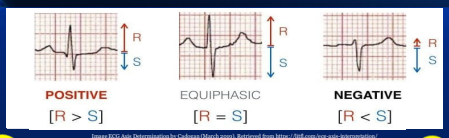


Image ECG Axis Determination by Cadogan (March 2015). Retrieved from https://litfl.com/ecg-axis-interpretation/

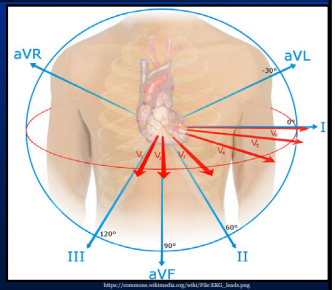
- **Negative waves→** produced by a positive impulse moving ***AWAY*** from a lead
- **Positive waves→** produced by a positive impulse moving ***TOWARDS*** a lead
- **Isoelectric waves→** produced by an equal amount of positive and negative energy affecting a lead

(Garcia, 2015)

13

ECG Leads

- The standard EKG has 12 leads
  - 3 Standard Limb Leads
  - 3 Augmented Limb Leads
  - 6 Precordial Leads
- The axis of a particular lead represents the viewpoint from which it looks at the heart
- Leads measure the difference in electrical potential between them
  1. Two different points on the body (bipolar leads)
  2. One point on the body and a virtual reference point with zero electrical potential, located in the center of the heart (unipolar leads)

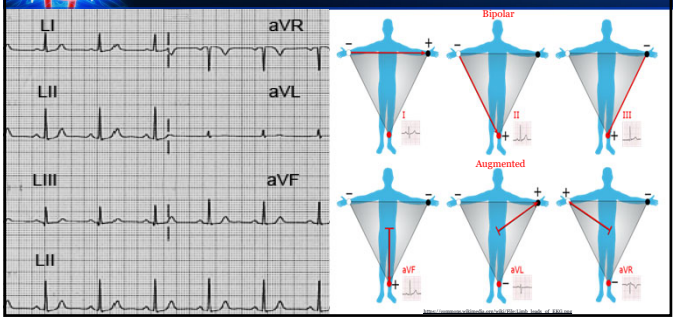


https://commons.wikimedia.org/wiki/File:EKG_leads.png

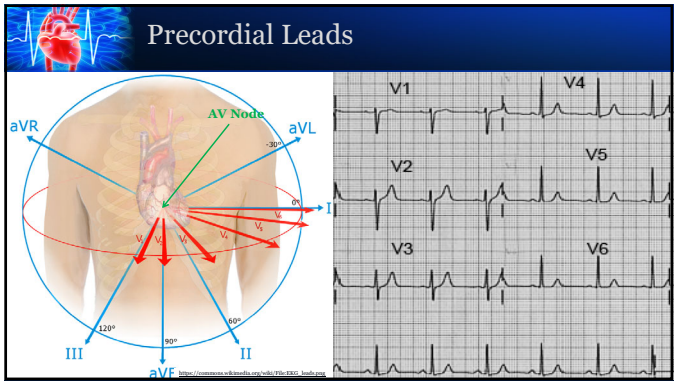
(Garcia, 2015; Wagner & Strauss, 2014)

14

Limb Leads



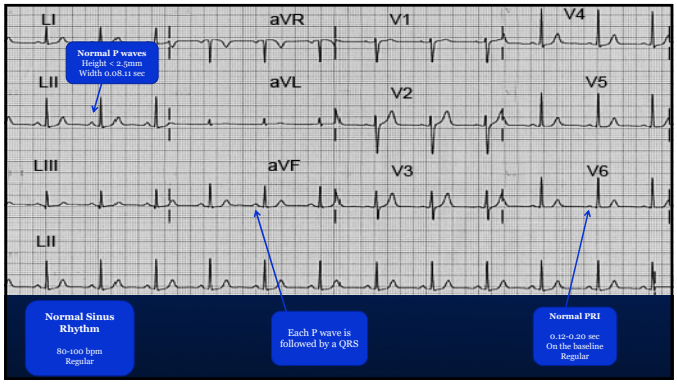
15



16

Normal 12-Lead ECG

17



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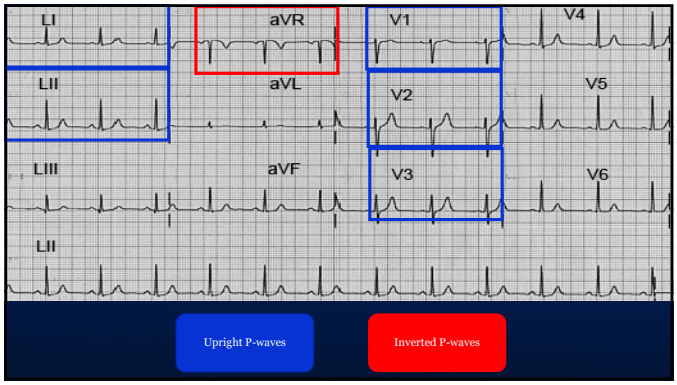
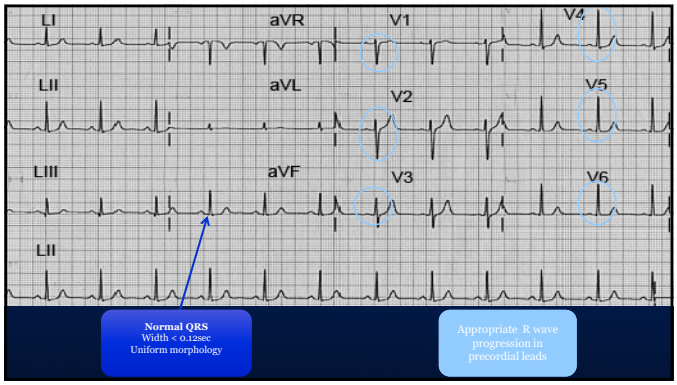
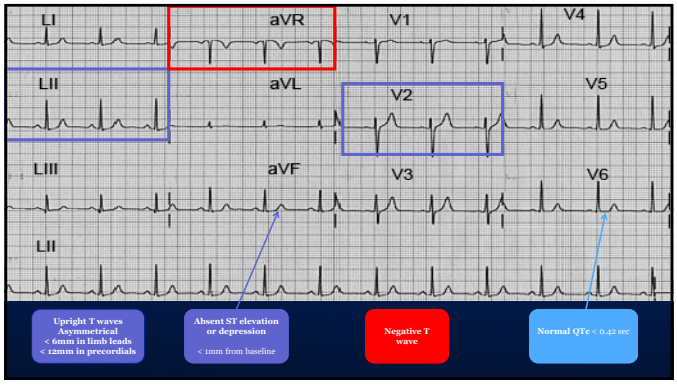

LI
aVR
V1
V4
LII
aVL
V2
V5
LIII
aVF
V3
V6
LII
Upright P-waves
Inverted P-waves

19


LI
aVR
V1
V4
LII
aVL
V2
V5
LIII
aVF
V3
V6
LII
Normal QRS
Width < 0.12sec
Uniform morphology
Appropriate R wave progression in precordial leads

20


LI
aVR
V1
V4
LII
aVL
V2
V5
LIII
aVF
V3
V6
LII
Upright T waves
Asymmetrical
< 6mm in limb leads
< 12mm in precordials
Absent ST elevation or depression
< 1mm from baseline
Negative T wave
Normal QTc < 0.42 sec

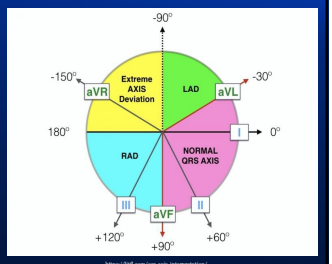
21

Axis Determination

22

Electrical Axis

- Mean QRS Vector
  - Sub-total of all the vectors generated by the action potentials of the individual ventricular myocytes
  - AV node is the starting point
- Vectors point
  - TOWARDS hypertrophy
  - AWAY from infarction

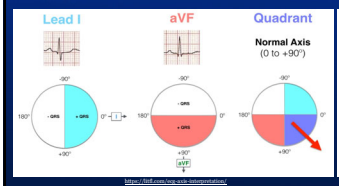


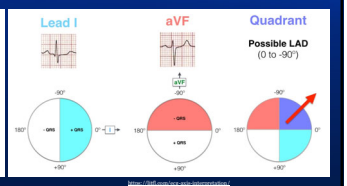
https://litfl.com/ecg-axis-interpretation/

(Garcia, 2015; Wagner & Strauss, 2014)

23

Quadrant Method





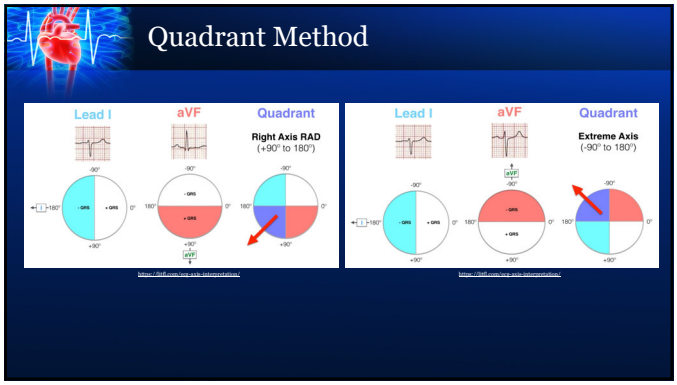
https://litfl.com/ecg-axis-interpretation/

https://litfl.com/ecg-axis-interpretation/

Why do we use Lead I & aVF?

(Garcia, 2015)

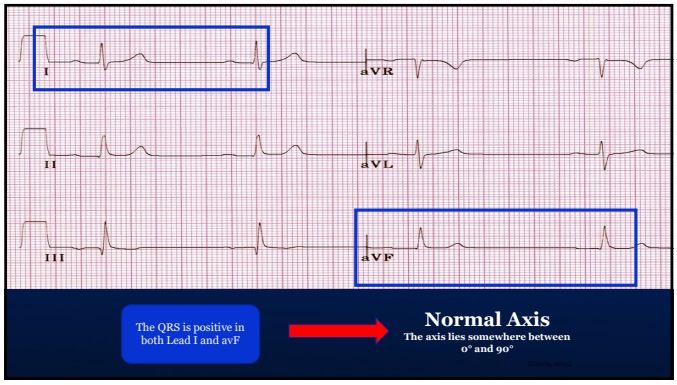
24



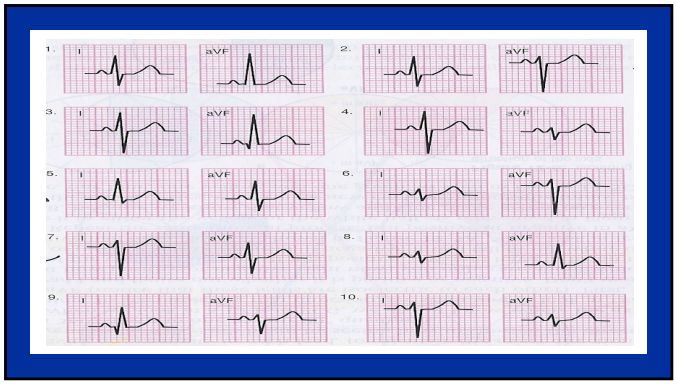
25

Lead 1	Lead aVF	Quadrant	Axis
POSITIVE	POSITIVE		Normal Axis (0 to +90°)
POSITIVE	NEGATIVE		**Possible LAD (0 to -90°)
NEGATIVE	POSITIVE		RAD (+90° to 180°)
NEGATIVE	NEGATIVE		Extreme Axis (-90° to 180°)

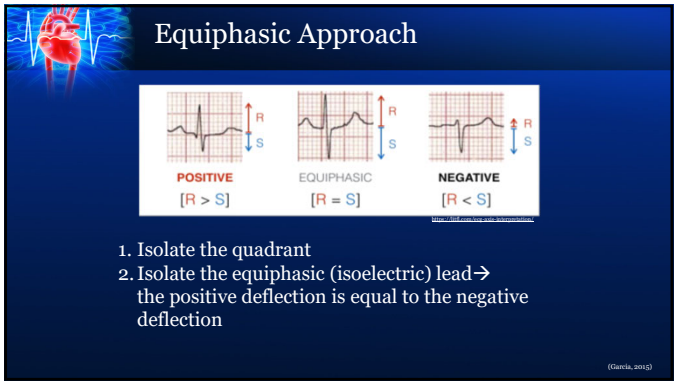
26



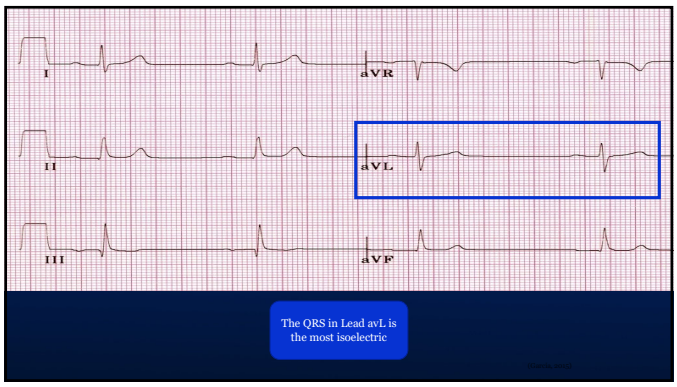
27


1.
I
aVF
2.
I
aVF
3.
I
aVF
4.
I
aVF
5.
I
aVF
6.
I
aVF
7.
I
aVF
8.
I
aVF
9.
I
aVF
10.
I
aVF

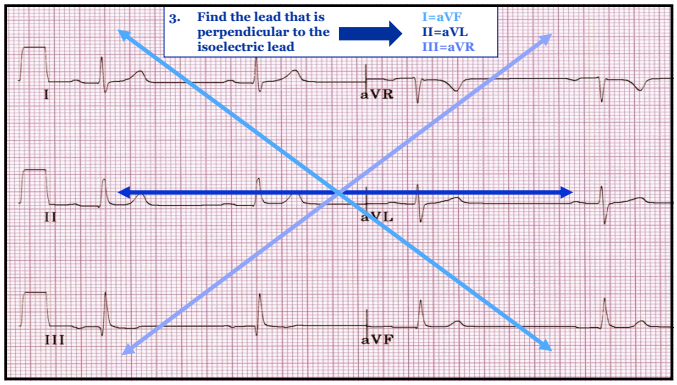
28


Equiphasic Approach
R
S
POSITIVE
[R > S]
R
S
EQUIPHASIC
[R = S]
R
S
NEGATIVE
[R < S]
1. Isolate the quadrant
2. Isolate the equiphasic (isoelectric) lead→ the positive deflection is equal to the negative deflection
(García, 2015)

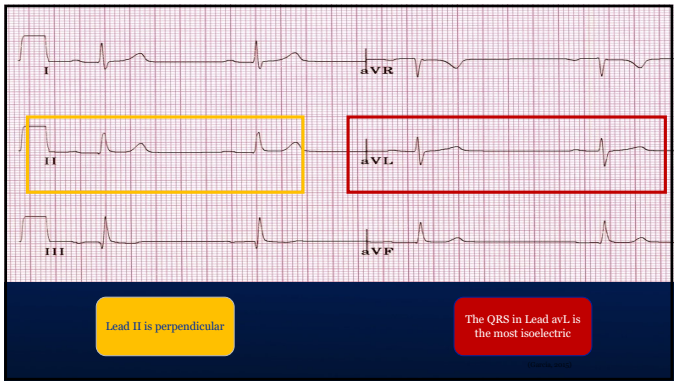
29


I
aVR
II
aVL
III
aVF
The QRS in Lead avL is the most isoelectric

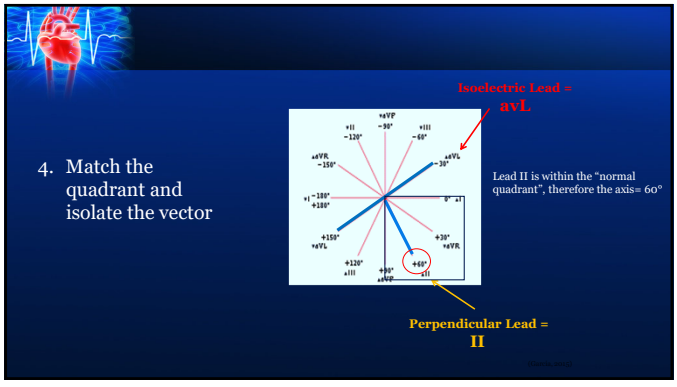
30


3. Find the lead that is perpendicular to the isoelectric lead
I=aVF
II=aVL
III=aVR
I
aVR
II
aVL
III
aVF

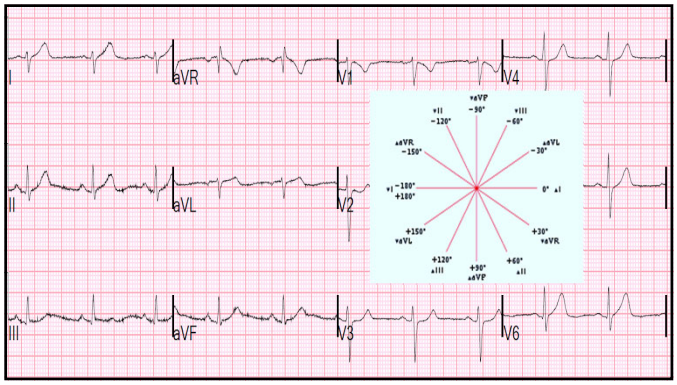
31


I
aVR
II
aVL
III
aVF
Lead II is perpendicular
The QRS in Lead avL is the most isoelectric

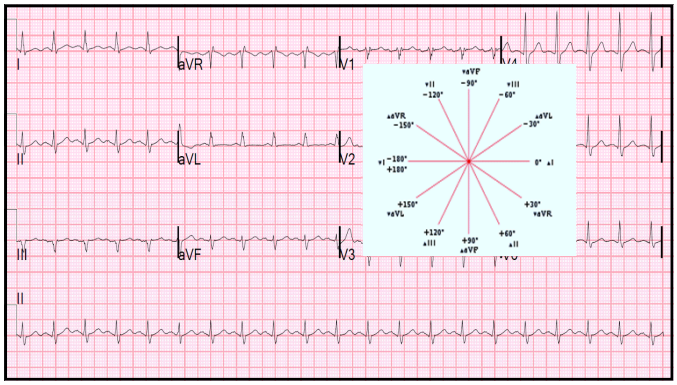
32


4. Match the quadrant and isolate the vector
Isoelectric Lead = avL
Lead II is within the "normal quadrant", therefore the axis= 60°
Perpendicular Lead = II

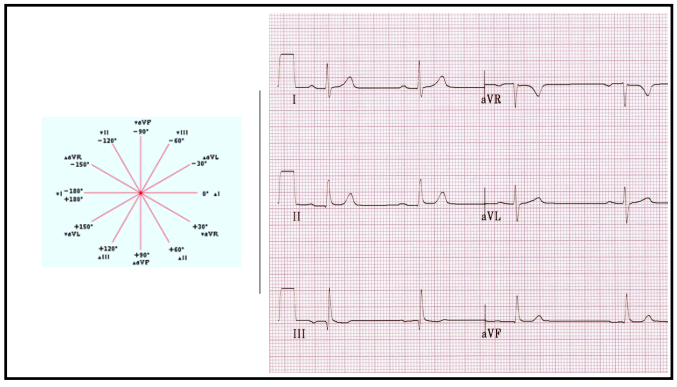
33


I
aVR
V1
V4
II
aVL
V2
III
aVF
V3
V6

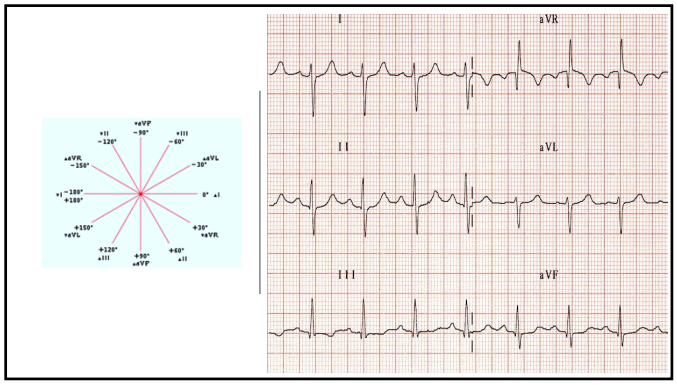
34


I
aVR
V1
II
aVL
V2
III
aVF
V3
II

35


I
aVR
II
aVL
III
aVF

36



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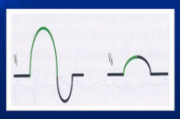
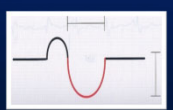
Hypertrophy

38

Atrial Hypertrophy



RAE *The first half of the P wave in V1 is greater than the first half in V6*



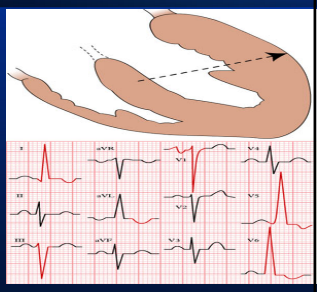
LAE *Second half of the P wave is wider & deeper than 0.30*

(Garcia, 2015)

39

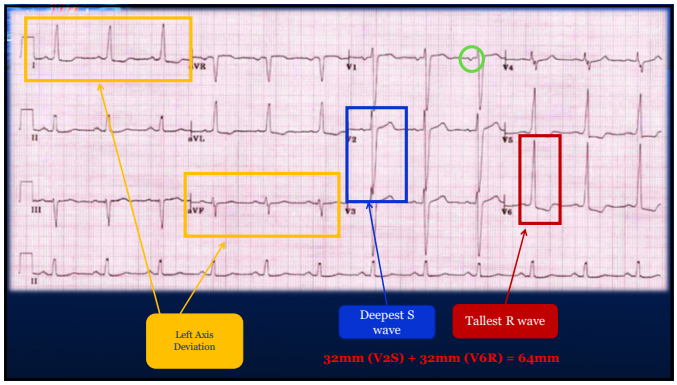
Left Ventricular Hypertrophy

- **Sokolow-Lyon Criteria**
  1. S wave depth in V1 or V2 (deepest) + R wave height in V5 or V6 (tallest) ≥ 35mm
- **Additional Criteria**
  1. Precordial R wave ≥ 45mm
  2. aVL R wave ≥ 11
  3. Lead I R wave ≥ 12
  4. Lead aVF R wave ≥ 20
  5. LAD



(Garcia, 2015; Wagner & Strauss, 2014)

40



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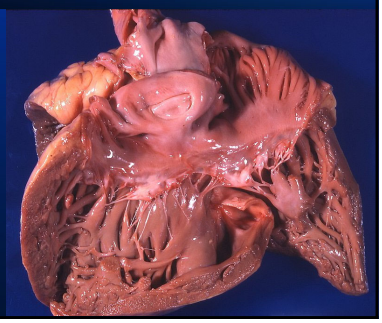
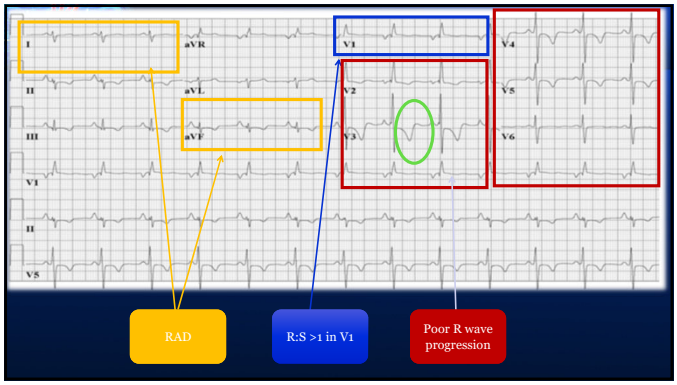
Right Ventricular Hypertrophy

- **Main Criteria**
  1. Dominant R wave in V1→ R:S in V1 is > 1
- **Additional Criteria**
  1. Dominant S wave in V5 or V6→ R:S <1
  2. RAD
  3. RV strain pattern
  4. RAE
  5. QRS <0.12
  6. Poor R wave progression

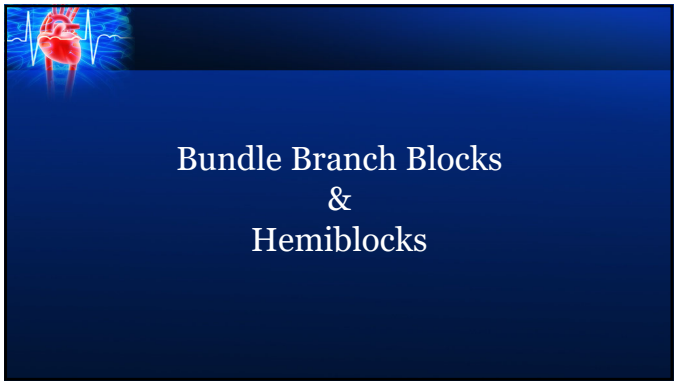


(Garcia, 2015; Wagner & Strauss, 2014)

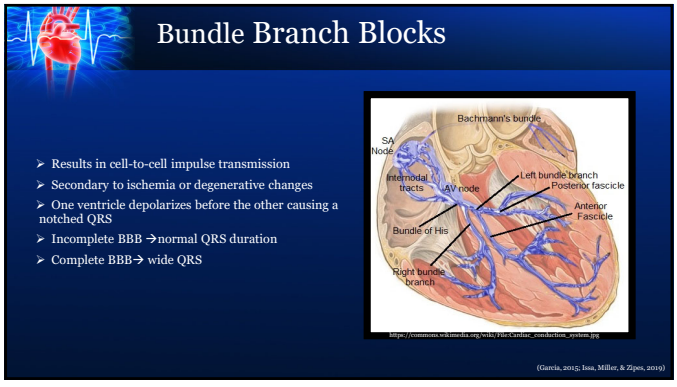
42


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
RAD
R:S >1 in V1
Poor R wave progression

43


Bundle Branch Blocks
&
Hemiblocks

44


Bundle Branch Blocks
Results in cell-to-cell impulse transmission
Secondary to ischemia or degenerative changes
One ventricle depolarizes before the other causing a notched QRS
Incomplete BBB →normal QRS duration
Complete BBB→ wide QRS
Bachmann's bundle
SA Node
Internodal tracts
AV node
Left bundle branch
Posterior fascicle
Anterior Fascicle
Bundle of His
Right bundle branch
(García, 2015; Issa, Miller, & Zipes, 2019)

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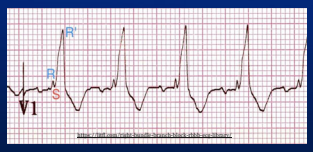
Right Bundle Branch Block

- Left ventricular activation is via the normal pathway
- Right ventricular activation is delayed→ new vector
  - Secondary R wave (R') in V1-V3
  - Slurred S wave in V6 & Lead I
  - Repolarization abnormalities→ ST depression & TWI in the right precordial leads
- There are many manifestations of the RsR'



https://bit.ly/ecg-right-bundle-branch-block-rbbb-in-3-steps/

(García, 2015; Issa, Miller, & Zipes, 2019)

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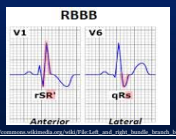
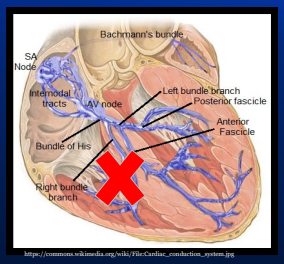
Right Bundle Branch Block Criteria

1. QRS ≥ 0.12 sec
2. RSR´in V1→ **Positive**
3. Slurred S wave in I & V6
4. *Incomplete→ above criteria except the QRS is <0.12 sec*



https://commons.wikimedia.org/wiki/File:Left_and_right_bundle_branch_block.png



https://commons.wikimedia.org/wiki/File:Cardiac_conduction_system.jpg

(García, 2015; Issa, Miller, & Zipes, 2019)

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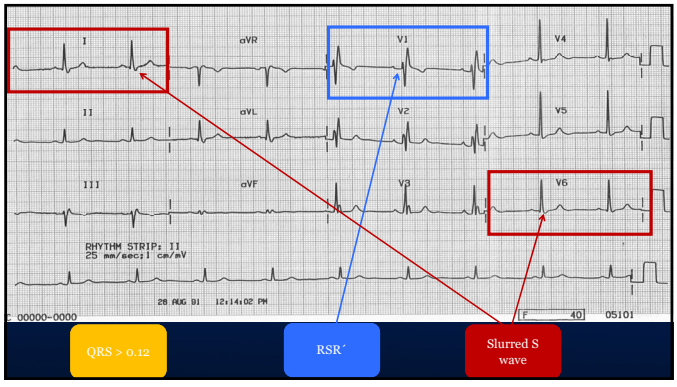

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Left Bundle Branch Block

- Right ventricular activation is via the normal pathway
- Left ventricular activation is delayed→ new vector
  - Delayed LV activation→ slurred S wave in V1
  - Broad monomorphic R in V6 & Lead I
- The proximal LBB is large & compact leading into the intraventricular septum and the fascicles

https://litfl.com/left-bundle-branch-block-lbbb-ecg-library/

(García, 2015; Issa, Miller, & Zipes, 2019)

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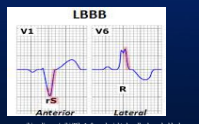
Left Bundle Branch Block Criteria

1. QRS ≥ 0.12 sec
2. Broad, monomorphic R wave in I & V6 that are either both positive or both negative
3. Broad, monomorphic S wave in V1→ **Negative**
4. May see RsR´ in V5 or V6
5. ST depression/elevation & broad T waves



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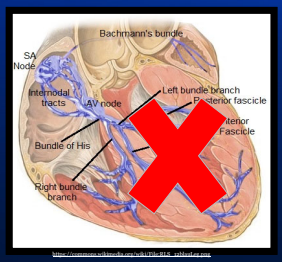

https://commons.wikimedia.org/wiki/File:BBB_pathology.png

(García, 2015; Issa et al., 2019)

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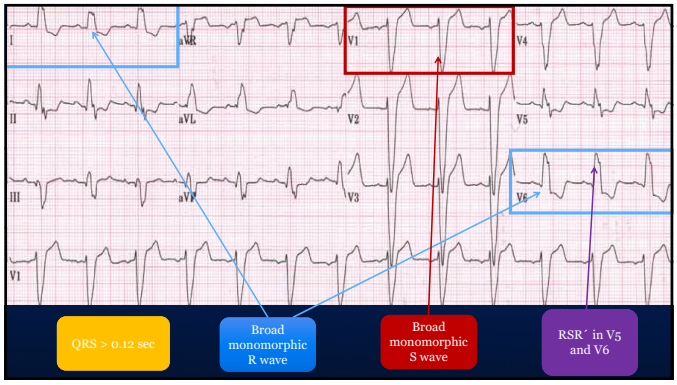

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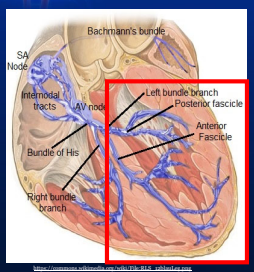
Fascicular Blocks



- AKA "Hemiblocks"
- Purkinje system is considered "Tri-fascicular"
  - Small proximal RBB→ RV
  - Left anterior fascicle→ anterosuperior papillary muscle
  - Left posterior fascicle→ posteroinferior papillary muscle

Unifascicular block
Isolated RBBB, LAH (most common)
Isolated LPH (rare)

Bifascicular block
Complete LBBB
RBBB + LAH or LPH

Trifascicular block
RBBB + LAH + LPH

(García, 2015; Issa et al., 2019)

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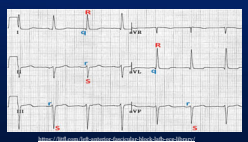
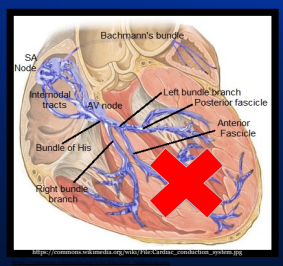
LAFB Criteria

1. Left axis deviation- typically
2. Either a qR complex or an R wave in lead I
3. An rS complex in lead III, possibly leads II and aVF





(García, 2015; Issa et al., 2019)

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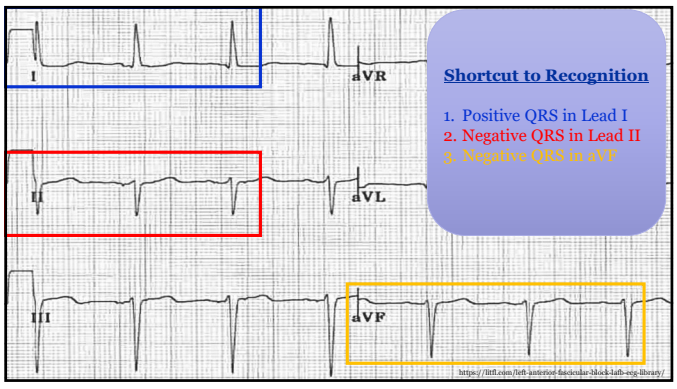

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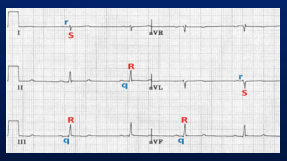
LPFB Criteria

1. Right axis deviation
2. An s wave in lead I
3. A q in lead III
4. No evidence of RVH



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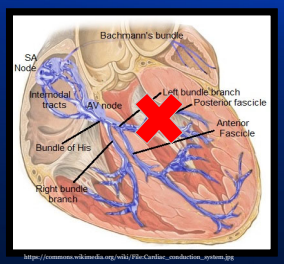


https://commons.wikimedia.org/wiki/File:Cardiac_conduction_system.jpg

(García, 2015; Issa, Miller, & Zipes, 2019)

55

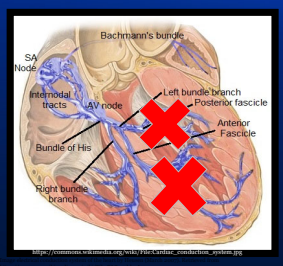
Bifascicular Block Criteria

RBBB + LAFB (most common)

1. Slurred S wave in leads I and V_6
2. RsR' in V_1 with delayed QRS > 0.12 sec

RBBB + LPFB

1. RBBB pattern
2. Small q wave in Lead III



https://commons.wikimedia.org/wiki/File:Cardiac_conduction_system.jpg

(García, 2015; Issa et al., 2019)

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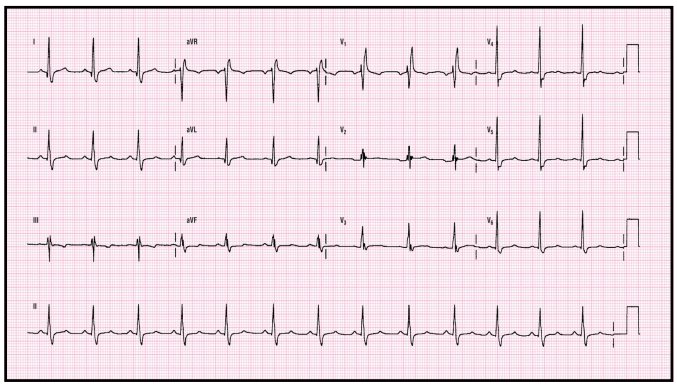

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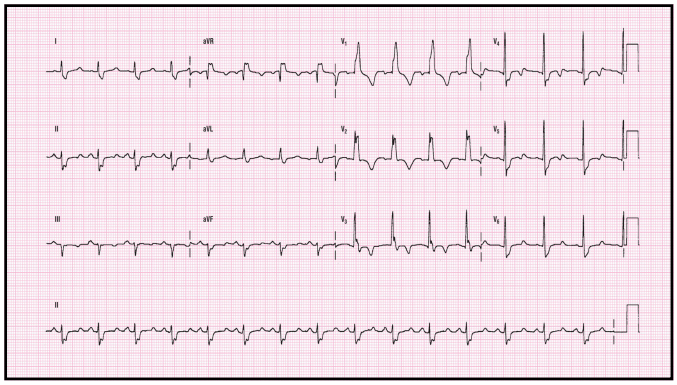

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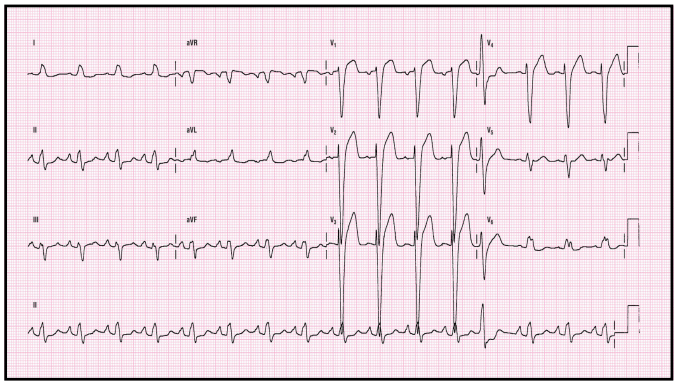

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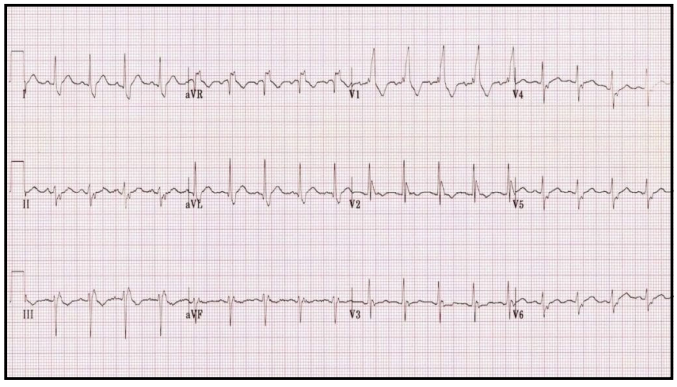

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ST Segments & T Waves

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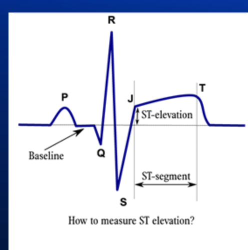
ST Segment

- ST segment→ the ventricles are between electrical depolarization and repolarization
- Measured from *J point* to beginning of T wave
- Important parts of the ECG as they reflect myocardial ischemia or injury
- Pathologic Changes
  - ST-segment depression + T waves in opposite direction from normal→ ischemia
  - ST-segment elevation +/- T wave changes → myocardial injury/infarction



https://commons.wikimedia.org/wiki/File:Stelevatie_en_(CardioNetworks_ECGpedia).png

(Garcia, 2015; Issa et al., 2019)

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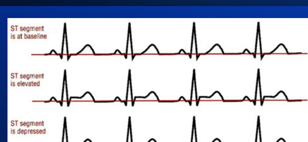
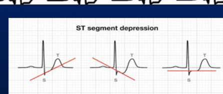
ST Elevation vs. Depression

What is the relationship to the baseline?

- Measured based upon the tp segment
- T wave and p waves may overlap making the determination difficult
- ST elevation of < 1mm is considered normal in the limb leads
- ST elevation of > 1mm in >2 contiguous leads + ischemic signs is indicative of a pathological process
- Horizontal or downsloping ST depression ≥ 0.5 mm in ≥ 2 contiguous leads→ myocardial ischemia





https://litfl.com/myocardial-ischaemia-ecg-library/

(Garcia, 2015; Issa et al., 2019)

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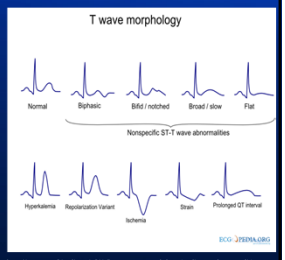
T Wave Morphology

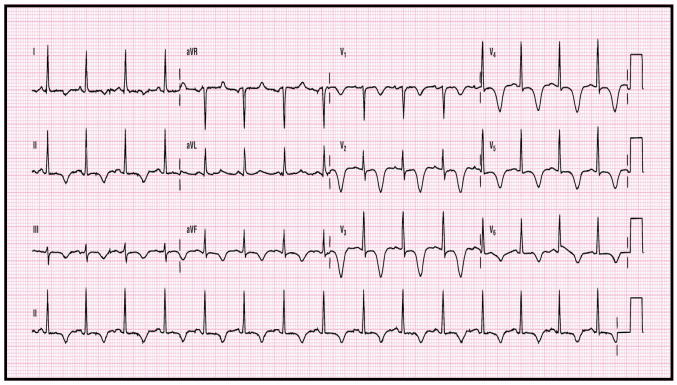
- T waves are typically **positive** in leads I, II, and V_3-V_6, and **negative** in lead aVR & possibly V1
- When evaluating the T waves, pay particular attention to the following:
  - Shape→ broad, symmetrical, etc.
  - Polarity→ positive, negative, or biphasic
  - Height/Depth→ tall or short
- Symmetric or flat T waves are always a sign of underlying pathology
- T waves should not be more than:
  - 6 mm high in limb leads
  - 12 mm high in precordial leads
- T wave > 2/3 height of R wave is always abnormal



https://commons.wikimedia.org/wiki/File:Te-T_wave_morphology_(CardioNetworks_ECGpedia).png

(García, 2015; Issa, Miller, & Zipes, 2019)

64



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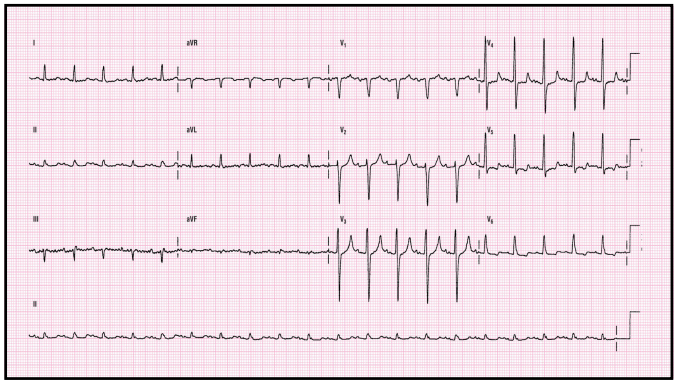
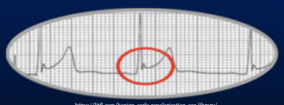


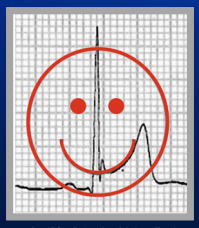
66

Benign Early Repolarization

- Common in healthy adults <50 years
- Characteristics
  - Widespread, concave ST elevation prominent in V2-V5
  - Notched or slurred J point
  - Prominent, concordant T waves that are slightly asymmetrical
  - STE is typically< 2mm in the precordial leads & < 0.5mm in the limb leads
  - No reciprocal ST depression to suggest STEMI
  - ST changes are not progressive



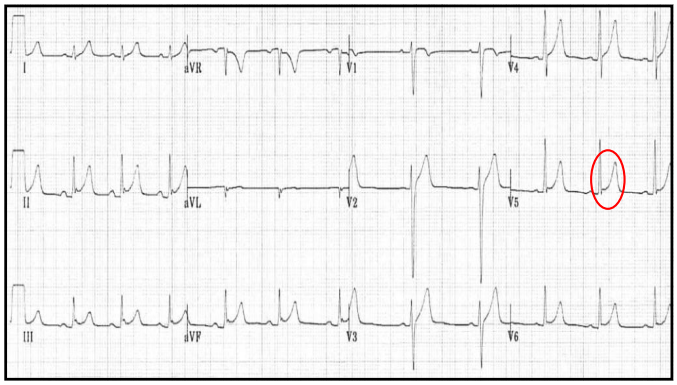
https://litfl.com/benign-early-repolarisation-ecg-library/



https://litfl.com/benign-early-repolarisation-ecg-library/

(García, 2015; Zipes et. al., 2019)

67



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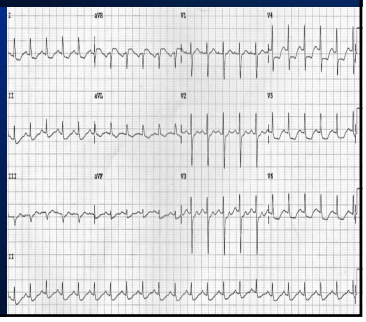
Myocardial Ischemia

- Reversible
- Affects a wedge-shaped section of the heart
- ECG signs
  - ST depression, particularly in a regional distribution
  - Symmetrical T waves
  - Biphasic T waves are initially negative
  - ST segment is flat or down sloping
- Energy production is via anaerobic metabolism
- Susceptibility is dependent upon blood supply proximity, distance from the major coronary arteries, & workload
- "Demand" or subendocardial ischemia

(García, 2015; Issa et al., 2019)



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Acute Myocardial Infarction

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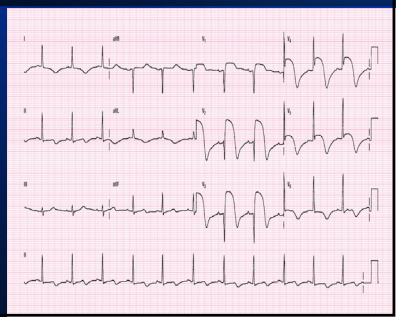
Myocardial Infarction

NOT REVERSIBLE

- Dead tissue is unable to generate action potentials and acts as an electrical "window" through the myocardium
- Energy stores are depleted→ necrosis
- Causes significant ST elevation, flipped T waves, & possibly Q waves

(García, 2015; Issa et al., 2019)



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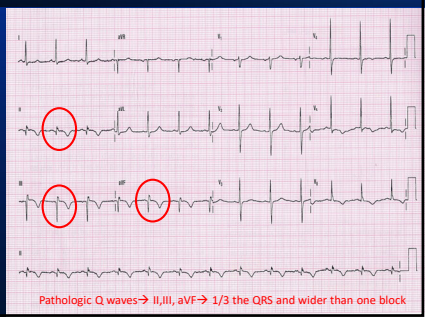
Q-Waves

- ECG representation of left-right intraventricular septum depolarization
- Benign
  - Septal Q waves→ Leads I and aVL
  - QS waves→ Isolated to V1
  - Q wave→ Isolated to lead III
  - Respiratory Variation
- Pathologic
  - Indicative of dead myocardium
  - May indicate an old MI
  - Extends through V_1 to V_2 or even V_3
  - Found in II or aVF→ Inferior MI

(García, 2015; Issa et al., 2019)



Pathologic Q waves→ II,III, aVF→ 1/3 the QRS and wider than one block

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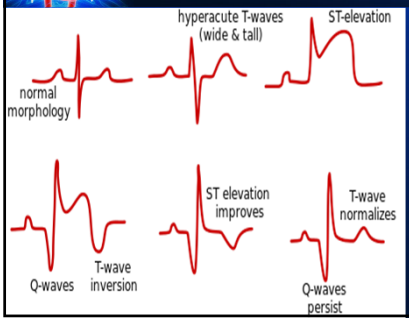
Infarction Progression



Hyperdynamic T-waves are often the first sign of myocardial changes

(Garcia, 2015; Wagner & Strauss, 2014)

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Reciprocal Changes

"Mirror image" that occurs when 2 electrodes are viewing the same AMI from opposite angles

Electrode A

1. Unopposed vector heading AWAY→ Q wave
2. Positive zone of injury→ ST elevation
3. Repolarization abnormality→ T wave inversion

Electrode B

1. Unopposed vector heading TOWARDS→ Tall R wave
2. Zone of injury/Ischemia→ ST depression
3. Repolarization abnormality→ T wave upright

(Garcia, 2015; Wagner & Strauss, 2014)

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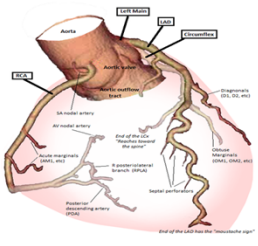
Left Anterior Descending Artery

- The LAD travels in the anterior interventricular groove and continues up to the apex of the heart.
- **Septal branches**→ supply the anterior septal wall
- **Diagonal branches**→ supply the anterior wall of the left ventricle
- Supplies most of the left ventricle and the AV-bundle
- Supplies a portion of the RBB, Left anterior division & posterior division of the LBB
- EKG changes→ leads V1-V4 depending upon proximity

(Garcia, 2015; Issa et al., 2019)



https://commons.wikimedia.org/wiki/File:Anatomy_of_coronary_arteries.png

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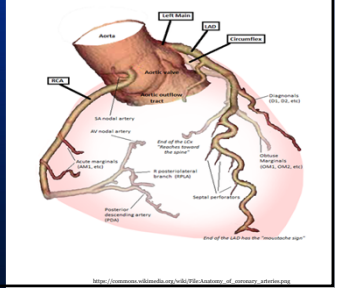
Circumflex Artery

- Situated in the AV groove between the LA & LV
- **Obtuse marginals**(OM1/OM2)→ they supply the lateral *margin* of the left ventricle and branch off with an *obtuse* angle.
- In most cases the CX ends as an obtuse marginal branch, however 10-15% of patients have a **left dominant** circulation in which the Cx also supplies the posterior descending artery (PDA).
- EKG changes→ V5, V6, I, aVL

(Garcia, 2015; Issa et al., 2019)



https://commons.wikimedia.org/wiki/File:Anatomy_of_coronary_arteries.png

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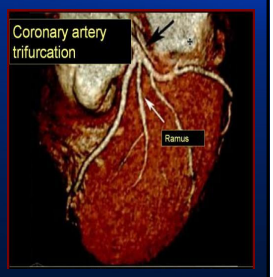
Ramus

- It generally lies in the angle between the LAD and the LCX
- Behaves like a large OM or a Diag→ supplies the lateral free wall of the LV
- Ramus could present as ST elevation in 1/avL /V5,V6
  - Lateral MI
  - Apical MI
  - High lateral MI
- Whenever the ECG changes are not fitting with typical ASMI or a lateral MI→ suspect a ramus lesion



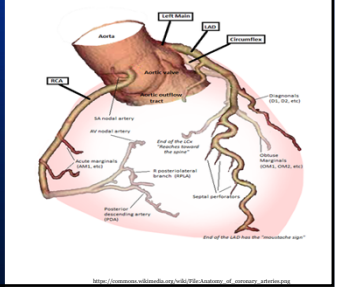
(Garcia, 2015; Issa, Miller, & Zipes, 2019)

77

Right Coronary Artery

- Lies in the AV groove b/t the RA & RV to the inferior septum
- Supplies the RA, RV, SA node, AV node, a portion of the left posterior division of the LBB & the septum
- **Conus Branch**→ supplies the RV outflow tract
- **SA node branch**→ may also arise from the CX in approx 40% of the population
- **Acute marginal**→ runs along the *margin* of the right ventricle above the diaphragm.
- **AV node branch**
- **PDA**→ supplies the inferior wall of the left ventricle and the posterior 1/3 of the intraventricular septum (if right dominant)
- EKG changes→ II, III, aVF

(Garcia, 2015; Issa, Miller, & Zipes, 2019)



https://commons.wikimedia.org/wiki/File:Anatomy_of_coronary_arteries.png

78

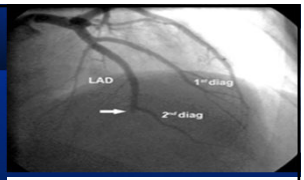
I Lateral	aVR	V1 Septal	V4 Anterior
II Inferior	aVL Lateral	V2 Septal	V5 Lateral
III Inferior	aVF Inferior	V3 Anterior	V6 Lateral

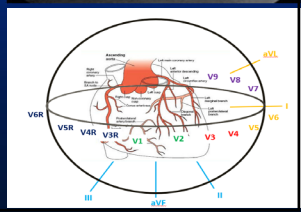
79

Anterior Wall MI

- Rarely occurs in isolation
  - Anteroseptal→ V1-V4
  - Anterolateral→ V3-V6, *I & aVL
  - Anteroseptal with Lateral Extension→ V1-V6, I, & aVL
- Typically confers a larger infarct region
- Secondary to an acute LAD or LMCA occlusion
- Proximal vs Distal is an essential prognostic factor
  - Proximal to S1→ Basal septal
  - Proximal to D1→ High lateral
- Anterior MI + RBBB→ poor prognosis

(Garcia, 2015; Wagner & Strauss, 2014)





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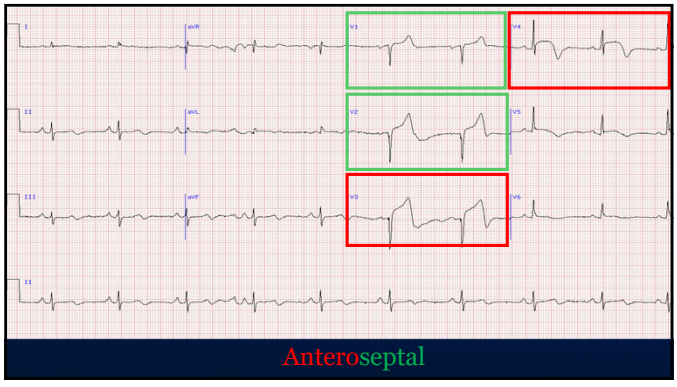
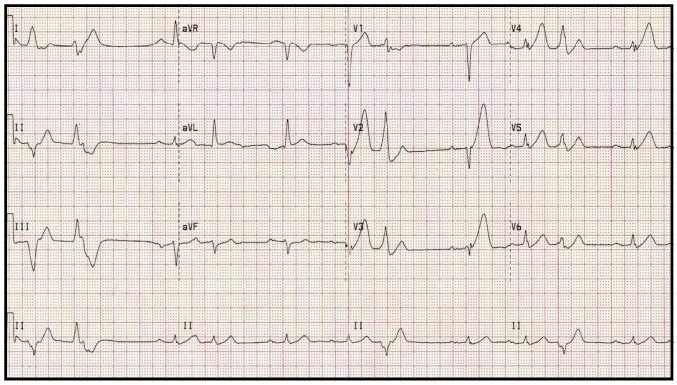
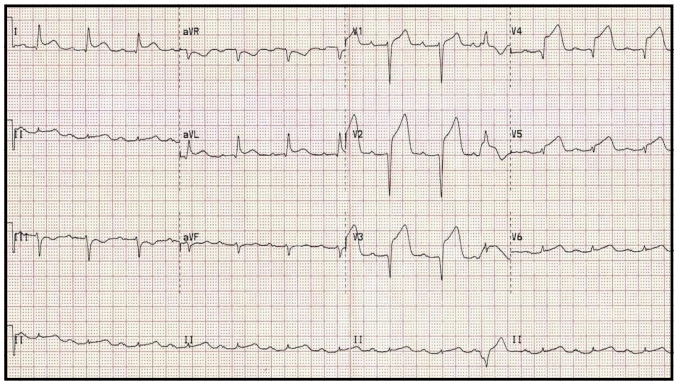


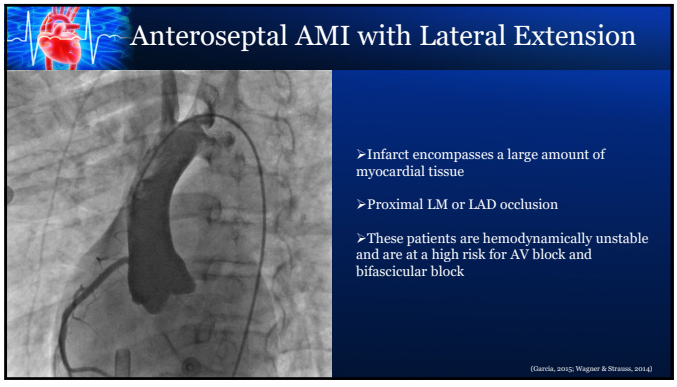
81


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
II
II
II

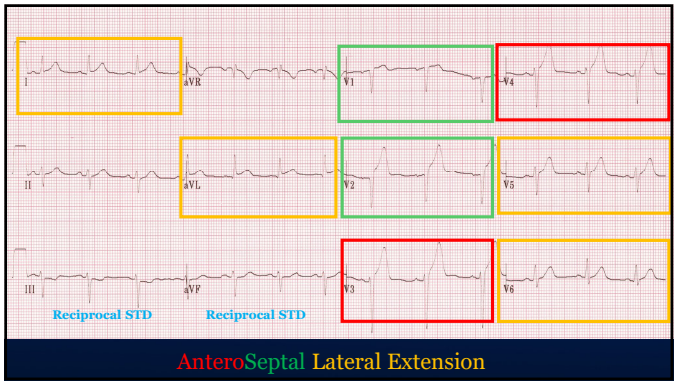
82


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
II
II
II

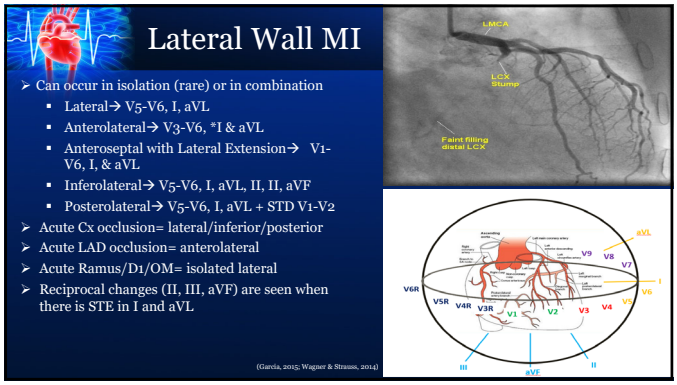
83


Anteroseptal AMI with Lateral Extension
➢Infarct encompasses a large amount of myocardial tissue
➢Proximal LM or LAD occlusion
➢These patients are hemodynamically unstable and are at a high risk for AV block and bifascicular block
(Garcia, 2015; Wagner & Strauss, 2014)

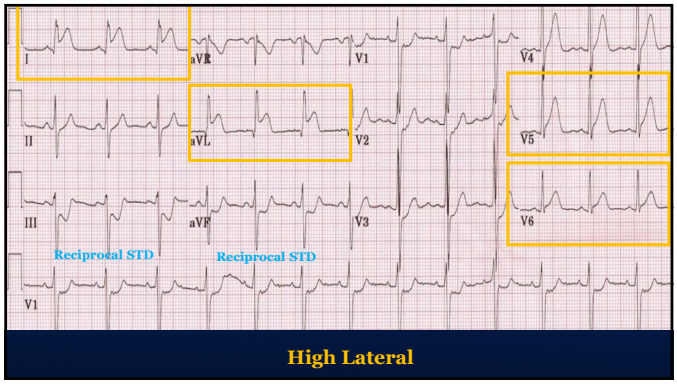
84


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Reciprocal STD
Reciprocal STD
AnteroSeptal Lateral Extension

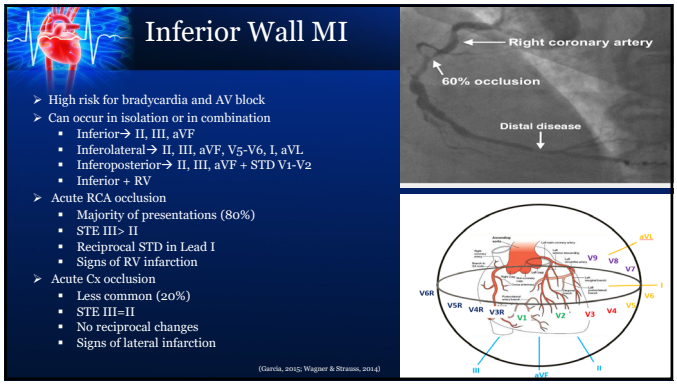
85


Lateral Wall MI
Can occur in isolation (rare) or in combination
Lateral→ V5-V6, I, aVL
Anterolateral→ V3-V6, *I & aVL
Anteroseptal with Lateral Extension→ V1-V6, I, & aVL
Inferolateral→ V5-V6, I, aVL, II, aVF
Posterolateral→ V5-V6, I, aVL + STD V1-V2
Acute Cx occlusion= lateral/inferior/posterior
Acute LAD occlusion= anterolateral
Acute Ramus/D1/OM= isolated lateral
Reciprocal changes (II, III, aVF) are seen when there is STE in I and aVL
(García, 2015; Wagner & Strauss, 2014)
LMCA
LCX Stump
Faint filling distal LCX
aVL
I
V6
V5
V4
V3
V2
V1
V4R
V3R
V5R
V6R
V7
V8
V9
III
aVF
II

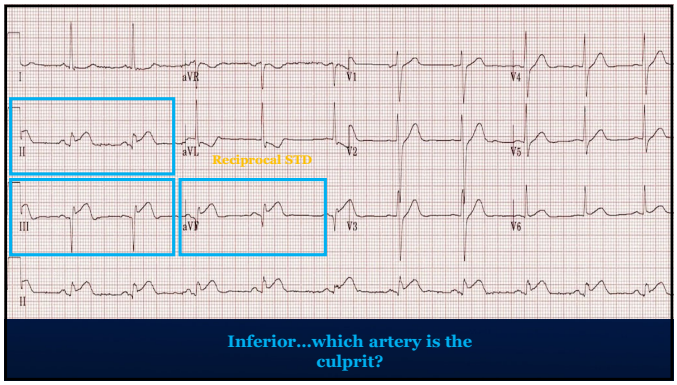
86


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Reciprocal STD
Reciprocal STD
V1
High Lateral

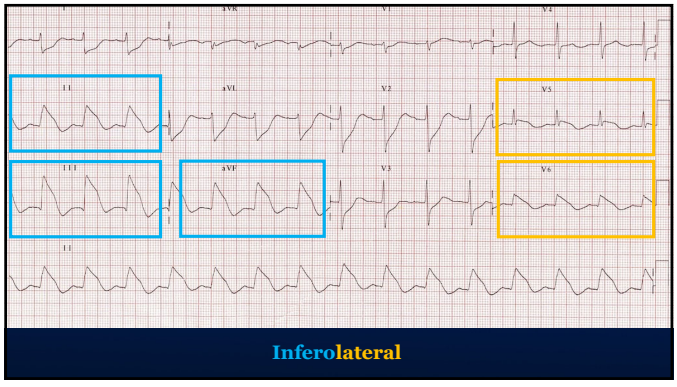
87


Inferior Wall MI
High risk for bradycardia and AV block
Can occur in isolation or in combination
Inferior→ II, III, aVF
Inferolateral→ II, III, aVF, V5-V6, I, aVL
Inferoposterior→ II, III, aVF + STD V1-V2
Inferior + RV
Acute RCA occlusion
Majority of presentations (80%)
STE III> II
Reciprocal STD in Lead I
Signs of RV infarction
Acute Cx occlusion
Less common (20%)
STE III=II
No reciprocal changes
Signs of lateral infarction
(Garcia, 2015; Wagner & Strauss, 2014)
Right coronary artery
60% occlusion
Distal disease

88


Reciprocal STD
Inferior…which artery is the culprit?

89

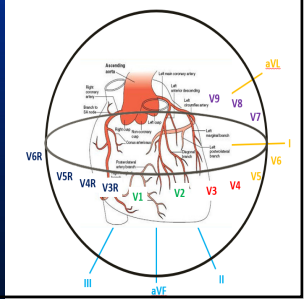

Inferolateral

90

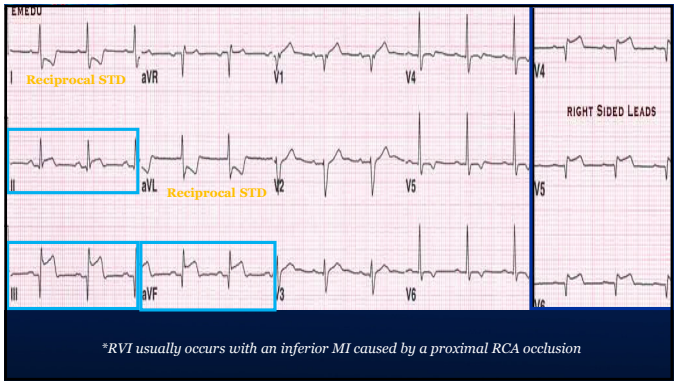
Right Ventricular MI

- Complicates ≈40% of Inferior MI's, therefore be suspicious in any patient presenting with an IWMI!
- ***Preload dependent → avoid Nitrates, diuretics, etc.***
- Criteria
  1. STE lead III > II
  2. STE V1
  3. STD V2 < ½ STE in aVF
  4. > 1mm STE in RV leads V4R-V6R

(García, 2015; Wagner & Strauss, 2014)



91



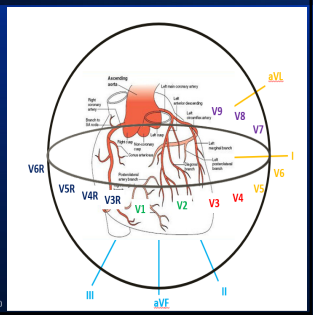
**RVI usually occurs with an inferior MI caused by a proximal RCA occlusion*

92

Posterior Wall MI

- Isolated posterior MI is less common
  - Inferoposterior
  - Posterolateral
- Criteria is based upon reciprocal changes in V1-V2
  - Tall, flat R wave
  - ST depression
  - Upright T wave, asymmetric
- The T wave critical in making this diagnosis vs. anterior ischemia where the T wave is inverted and symmetric
- You can also assess the posterior leads V7-V10

(García, 2015; Wagner & Strauss, 2014)



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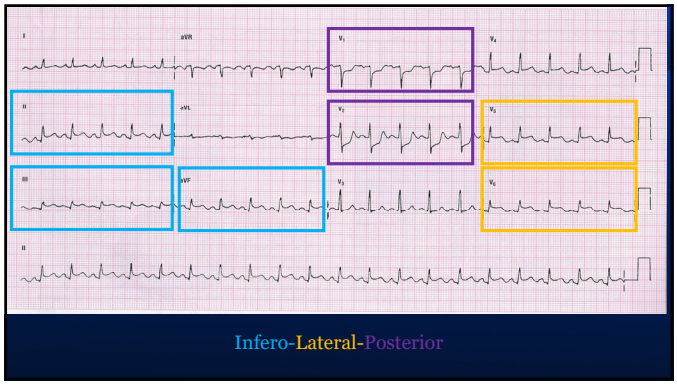

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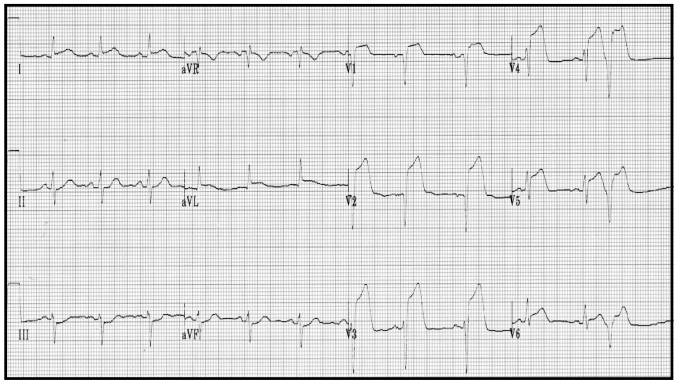
Regional Distribution	Arterial Supply	Leads	Reciprocal Changes
Inferior	RCA or CX	II, III, aVF	I, aVL
Inferior-RV-Posterior	Proximal RCA	V7-V10 II, III, aVF	V1-V2 I, aVL
Inferoposterior	RCA or CX	V7-V9 II, III, aVF	V1-V2 I, aVL
Inferolateral	RCA, CX	V5-V6 II, III, aVF I, aVL if high lateral	None
Isolated RV	CX	V1-V2 V4-V6R II, III, aVF	I, aVL
Isolated Posterior	RCA or CX	V7-V10	V1-V2
Anterior	LAD	V3, V4	None
Anteroseptal	LAD	V1-V4	None
Anteroseptal-Lateral extension	Proximal LAD	V1-V6, I, aVL	II, III, aVF
Lateral	CX	V5-V6, I, aVL	II, III, aVF
Apical	RCA dominant	V2-V6, I, aVL II, III, aVF	None

95



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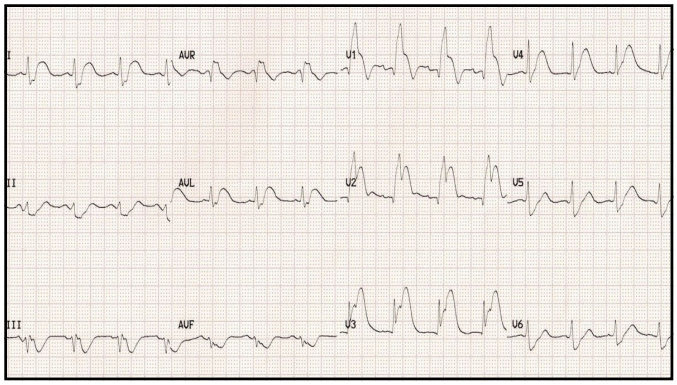
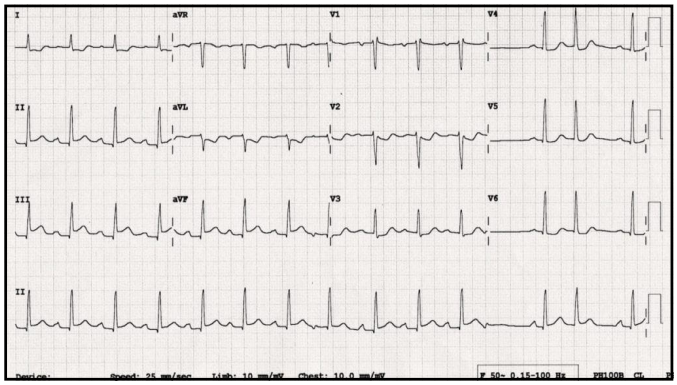
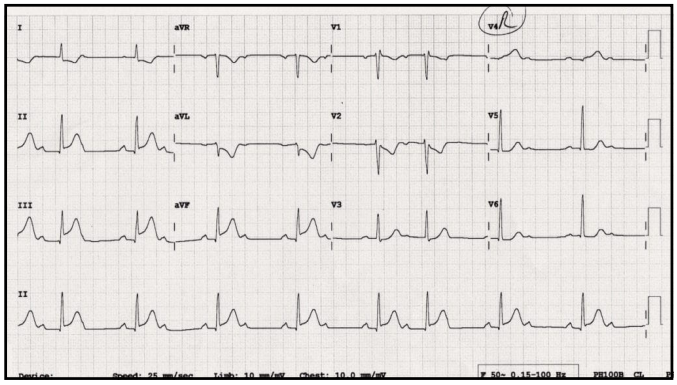

I
AUR
U1
U4
II
AUL
U2
U5
III
AUF
U3
U6

97


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

98


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

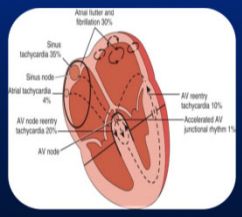
99

Arrhythmia Recognition & Management

100

Supraventricular Arrhythmias

- Tachyarrhythmias in which atrial and/or ventricular rates exceed 100 bpm at rest
- Site of origination is at or above the HIS bundle leading to a narrow complex (<120ms)
- May exhibit a regular or irregular ventricular response
- Types
  - Sinus tachycardia
  - Supraventricular tachycardia (SVT/PSVT)
  - AV reentrant (or reciprocating) tachycardia (AVRT)
  - AV nodal reentrant tachycardia (AVNRT)
  - Focal Atrial tachycardia (AT)
  - Inappropriate ST (IST)
  - Atrial fibrillation
  - Atrial flutter
  - Multifocal AT (MAT)



(Issa, Miller, & Zipes, 2019)

101

Sinus Tachycardia

Clinical Characteristics & Presentation

- SA node driven physiologic response secondary to
  - Sympathetic activation
  - Parasympathetic withdrawal
  - Demand for increased CO
- Types
  - Physiologic
  - Postural Orthostatic Tachycardia Syndrome
  - Inappropriate Sinus Tachycardia
- Most patients are asymptomatic, or symptoms may be related to the underlying pathophysiology
- HR >100 bpm

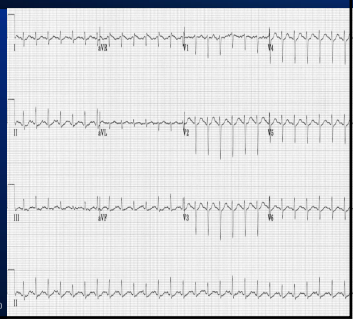
Diagnostic Work-up

- Diagnostic maneuvers to differentiate ST from another supraventricular arrhythmias
  - Carotid massage
  - Valsalva maneuver
  - AV node blocking agents
- Further evaluation is directed towards isolating the underlying cause (septic shock, thyroid dysfunction, etc.)

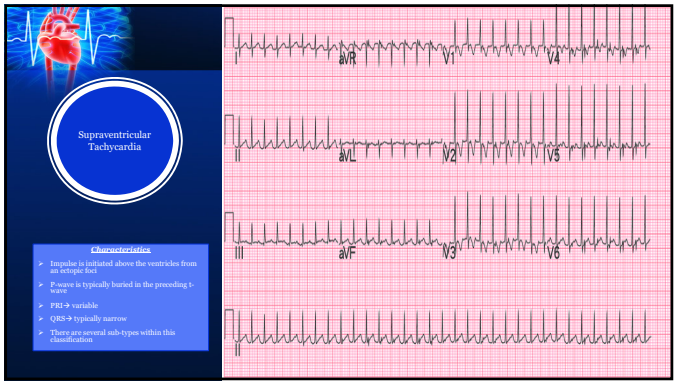
Management

- Aimed at treating the underlying cause
- Specialty referral if indicated

(Issa et al., 2019; Zipes et. al., 2019)



102



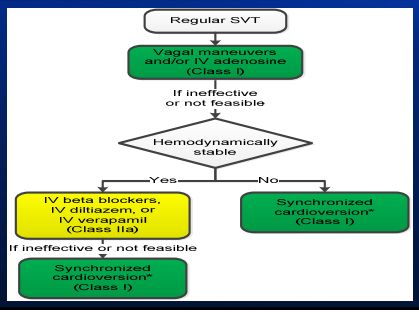
103

Initial Evaluation of PSVT

- Thorough history & physical exam
- 12-lead ECG
- Ambulatory Monitoring
  - Holter monitor→ useful in frequent, self-terminating tachycardias
  - Event monitor→ useful for less frequent episodes
  - Implantable loop recorder→ useful for rare episodes associated with hemodynamic instability
- Echocardiography
  - Documented sustained SVT to r/o SHD
- Exercise Testing
  - Rarely utilized unless there is a clear trigger related to exertion
- EP Testing
  - Not indicated unless catheter ablation is planned
  - Considered in patients with disabling symptoms without ECG arrhythmia documentation

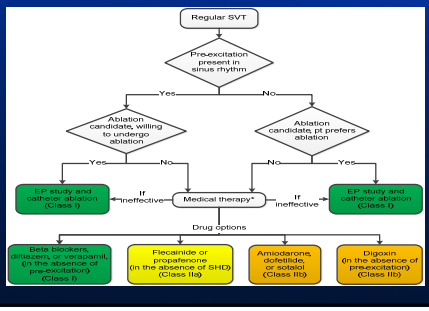
104

2015 AHA/ACC/HRS Guideline for the Management of Patients with Supraventricular Tachycardia: *Acute Management*



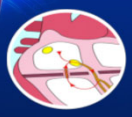
105

2015 AHA/ACC/HRS Guideline for the Management of Patients with Supraventricular Tachycardia: *Ongoing Management*



106

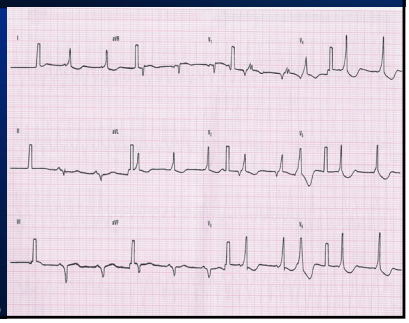
Wolff Parkinson White



- 1st Line Treatment
  - Procainamide (Class Ia)
  - Flecainide (Class Ic)
  - Sotalol, Amiodarone (Class III)
- Hemodynamic Instability→ Cardioversion

At high risk for SCD from lethal arrhythmias

(Issa et al., 2019; Zipes et. al., 2019)



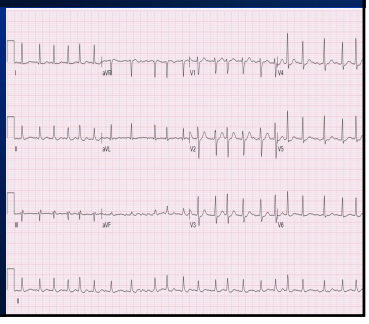
107

Atrial Fibrillation

Atrial Activity
- Rapid, irregular fibrillatory (f) waves with an undulating baseline
- Variable F wave morphology & amplitude
- Most prominent in II, III, avL, & V1
- Atrial rate→ >350 bpm

AV Conduction
- Irregularly, irregular
- Untreated patients→ V rate 90-170 bpm
- AVN disease→ V rate <60 bpm
- Physiologic/Pathophysiologic influence→ V rate >200

QRS Morphology
- Typically identical to a sinus beat
- Wide QRS etiology
  - Rate related aberrancy
  - BBB
  - Pre-excitation



108

Atrial Fibrillation

- Triggering factors & sustainability
  - SNS/PNS stimulation
  - PAC's
  - AFL
  - SVT
  - Acute atrial stretch
  - Inflammatory states
  - Common foci→ pulmonary veins
- Commonly occurs in the context of other pathological conditions
- Atrial Remodeling
  - Dilatation with progressive interstitial fibrosis
  - Electrical remodeling
- Genetic Component
  - Associated with a 40% increased risk for development in 1° relatives
  - Strongest with development at a younger age
  - Association with ion channelopathies

109

2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation

Term	Definition
Paroxysmal AF	• AF that terminates spontaneously or with intervention within 7 d of onset. • Episodes may recur with variable frequency.
Persistent AF	• Continuous AF that is sustained >7 d.
Long-standing persistent AF	• Continuous AF >12 mo in duration.
Permanent AF	• The term "permanent AF" is used when the patient and clinician make a joint decision to stop further attempts to restore and/or maintain sinus rhythm. • Acceptance of AF represents a therapeutic attitude on the part of the patient and clinician rather than an inherent pathophysiological attribute of AF. • Acceptance of AF may change as symptoms, efficacy of therapeutic interventions, and patient and clinician preferences evolve.
Nonvalvular AF	• AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair.

110

Initial Evaluation

- Essential components
  - Characterization of the arrhythmia
  - Determining underlying causes
  - Defining associated conditions
  - Identifying potential complications
- Thorough history & physical exam
- 12-lead ECG
- Thromboembolic vs. Hemorrhagic risk
- Evaluation of potential triggers
  - Ischemic evaluation
  - Electrolytes
  - CBC
  - Renal function
  - Hepatic function
  - Thyroid function
- Echocardiography→ evaluation for SHD, cardiac function, atrial size, LA thrombus
- Stress Test→ evaluation for ischemic heart disease, preparation for Class Ic antiarrhythmic therapy
- EP study

111

Four Pillars of Management

Stroke Prevention	Rate Control	Rhythm Control	Risk Factor Modification
• NOAC • Warfarin • LAA Occluder	• AV Nodal Blockers • Digoxin • AV Node Ablation + PPM	• Antiarrhythmic Therapy • Cardioversion • Ablation	• Hypertension • Diabetes • Weight Loss • Sleep Apnea • Tobacco Abuse • EtOH Abuse

112

2019 Focused Update of the AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation:
Antithrombotic Regimen Risk vs Benefit

Recommendations for Selecting an Anticoagulant Regimen—Balancing Risks and Benefits

COR	LOE	Recommendations
I	A	For patients with AF and an elevated CHA_2DS_2-VASc score of 2 or greater in men or 3 or greater in women, oral anticoagulants are recommended.
	B	
	B	Options include:
	B	• Warfarin (LOE: A)
	B-R	• Dabigatran (LOE: B) • Rivaroxaban (LOE: B) • Apixaban (LOE: B) or • Edoxaban (LOE: B-R)
I	A	NOACs (dabigatran, rivaroxaban, apixaban, and edoxaban) are recommended over warfarin in NOAC-eligible patients with AF (except with moderate-to-severe mitral stenosis or a mechanical heart valve).
I	B	In patients with AF (except with moderate-to-severe mitral stenosis or a mechanical heart valve), the CHA_2DS_2-VASc score is recommended for assessment of stroke risk.
I	B	For patients with AF who have mechanical heart valves, warfarin is recommended.
I	B	Selection of anticoagulant therapy should be based on the risk of thromboembolism, irrespective of whether the AF pattern is paroxysmal, persistent, or permanent.

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2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation:
Stroke Risk Stratification

Definition and Scores for $CHADS_2$ and CHA_2DS_2-VASc

	Score
$CHADS_2$	
Congestive HF	1
Hypertension	1
Age ≥75 y	1
Diabetes mellitus	1
Stroke/TIA/TE	2
Maximum score	6
CHA_2DS_2-VASc	
Congestive HF	1
Hypertension	1
Age ≥75 y	2
Diabetes mellitus	1
Stroke/TIA/TE	2
Vascular disease (prior MI, PAD, or aortic plaque)	1
Age 65–74 y	1
Sex category (i.e., female sex)	1
Maximum score	9

Stroke Risk Stratification With the $CHADS_2$ and CHA_2DS_2-VASc Scores

	Adjusted stroke rate (% per y)
$CHADS_2$*	
0	1.9
1	2.8
2	4.0
3	5.9
4	8.5
5	12.5
6	18.2
CHA_2DS_2-VASc	
0	0
1	1.3
2	2.2
3	3.2
4	4.0
5	6.7
6	9.8
7	9.6
8	6.7
9	15.20

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Landmark NOAC Trials

RE-LY
- Evaluated the safety and efficacy of Dabigatran (Pradaxa) vs. Warfarin
- Results→ less intracranial hemorrhage, however a higher incidence of GI bleeding
- Superior to Warfarin at the 150mg BID dose and non-inferior at the 110mg BID dose
- Reduce to 75mg BID for CrCl 15-30 mL/min

ROCKET-AF
- Evaluated the safety and efficacy of Rivaroxaban (Xarelto) vs. Warfarin
- Results→ less intracranial hemorrhage, however a higher incidence of GI bleeding
- Non-inferior to Warfarin at the 20mg once daily dose
- Dose reduction→ 15mg once daily for CrCl <50 mL/min
- Administer *with food*

(Fisher, et al., 2012; Patel, H. al., 2011)

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Landmark NOAC Trials

ARISTOTLE
- Evaluated the safety and efficacy of Apixaban (Eliquis) vs. Warfarin
- Results→ significantly lower risk of major bleeding and all cause mortality
- Superior to Warfarin in stroke prevention at the 5mg BID dose or the reduced dose
- Dose reduction→ 2.5mg BID for patients with at least 2 of these risk factors (Age ≥80, Weight ≤60kg, Cr ≥1.5 mg/dL

ENGAGE-AF
- Evaluated the safety and efficacy of Edoxaban (Savaysa) vs. Warfarin
- Results→ lower risk for major bleeding and hemorrhagic stroke
- Non-inferior to Warfarin in stroke prevention at the 60mg once daily dose or the reduced dose
- Dose reduction→ 30mg daily for CrCl ≤50 mL/min
- FDA boxed warning→ do not use in patients with a CrCL ≥95 mL/min due to decreased efficacy

(Giugliano et. al., 2013; Granger, et. al., 2011)

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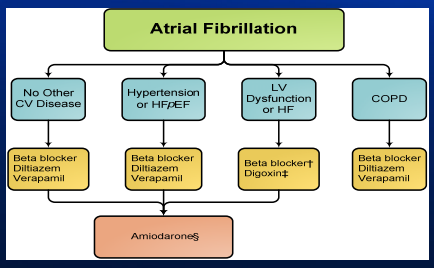
2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation:
Rate Control



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2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation:
Rhythm Control- DCCV

Recommendations	COR	LOE
In pursuing a rhythm-control strategy, cardioversion is recommended for patients with AF or atrial flutter as a method to restore sinus rhythm. If cardioversion is unsuccessful, repeated attempts at direct-current cardioversion may be made after adjusting the location of the electrodes, applying pressure over the electrodes or following administration of an antiarrhythmic medication.	I	B
Cardioversion is recommended when a rapid ventricular response to AF or atrial flutter does not respond promptly to pharmacological therapies and contributes to ongoing myocardial ischemia, hypotension, or HF.	I	C
Cardioversion is recommended for patients with AF or atrial flutter and pre-excitation when tachycardia is associated with hemodynamic instability.	I	C
It is reasonable to perform repeated cardioversions in patients with persistent AF, provided that sinus rhythm can be maintained for a clinically meaningful period between cardioversion procedures. Severity of AF symptoms and patient preference should be considered when embarking on a strategy requiring serial cardioversion procedures.	IIa	C

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2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation:
Rhythm Control- Pharmacologic Cardioversion

Recommendations	COR	LOE
Flecainide, dofetilide, propafenone, and intravenous ibutilide are useful for pharmacological cardioversion of AF or atrial flutter, provided contraindications to the selected drug are absent.	I	A
Administration of oral amiodarone is a reasonable option for pharmacological cardioversion of AF.	IIa	A
Propafenone or flecainide ("pill-in-the-pocket") in addition to a beta blocker or nondihydropyridine calcium channel antagonist is reasonable to terminate AF outside the hospital once this treatment has been observed to be safe in a monitored setting for selected patients.	IIa	B
Dofetilide therapy should not be initiated out of hospital because of the risk of excessive QT prolongation that can cause torsades de pointes.	III: Harm	B

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2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation:
Pharmacologic Maintenance of NSR

Recommendations	COR	LOE
Before initiating antiarrhythmic drug therapy, treatment of precipitating or reversible causes of AF is recommended.	I	C
The following antiarrhythmic drugs are recommended in patients with AF to maintain sinus rhythm, depending on underlying heart disease and comorbidities: a. Amiodarone b. Dofetilide c. Dronedarone d. Flecainide e. Propafenone f. Sotalol	I	A
The risks of the antiarrhythmic drug, including proarrhythmia, should be considered before initiating therapy with each drug.	I	C
Because of its potential toxicities, amiodarone should only be used after consideration of risks and when other agents have failed or are contraindicated.	I	C
A rhythm-control strategy with pharmacological therapy can be useful in patients with AF for the treatment of tachycardia-induced cardiomyopathy.	IIa	C
It may be reasonable to continue current antiarrhythmic drug therapy in the setting of infrequent, well-tolerated recurrences of AF when the drug has reduced the frequency or symptoms of AF.	IIb	C
Antiarrhythmic drugs for rhythm control should not be continued when AF becomes permanent,...	III: Harm	C
...including dronedarone.		B
Dronedarone should not be used for treatment of AF in patients with New York Heart Association class III and IV HF or patients who have had an episode of decompensated HF in the past 4 weeks.	III: Harm	B

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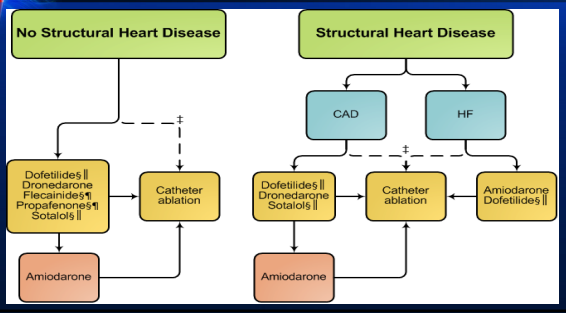
2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation



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Atrial Flutter

Typical
- Often utilizes the cavotricuspid isthmus as the reentry circuit
- Rarely presents as the primary pathology

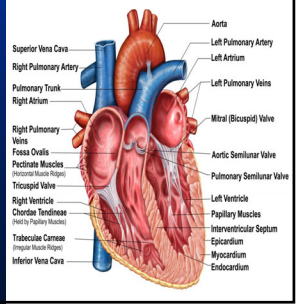
Atypical
- Waves rotate around the pulmonary veins and mitral annulus most commonly

Flutter Waves
- Atrial complexes of consistent morphology & polarity
- Atrial rate→ 240-340 bpm
- Slower rates (<150) occur with SAN dysfunction, scarring, or antiarrhythmic drugs

AV Conduction
- Even, integral conduction ratios most commonly
- Most common 2:1
- Variable conduction can occur

QRS Morphology
- Typically identical to a sinus beat
- Can be temporally distorted by superimposed flutter waves

(Issa et al., 2019; Zipes et. al., 2019)



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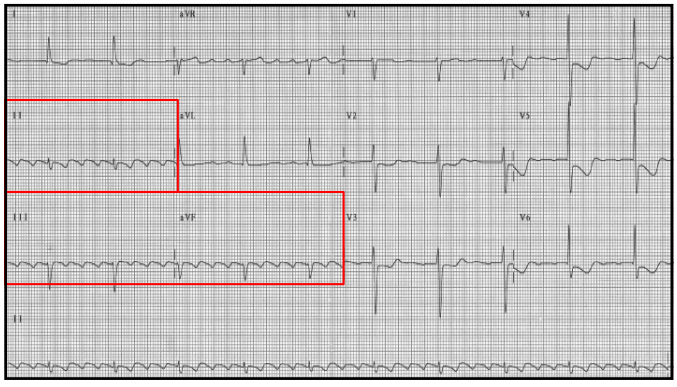

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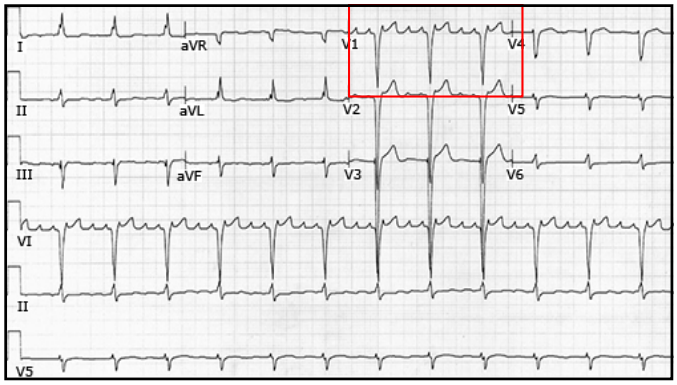
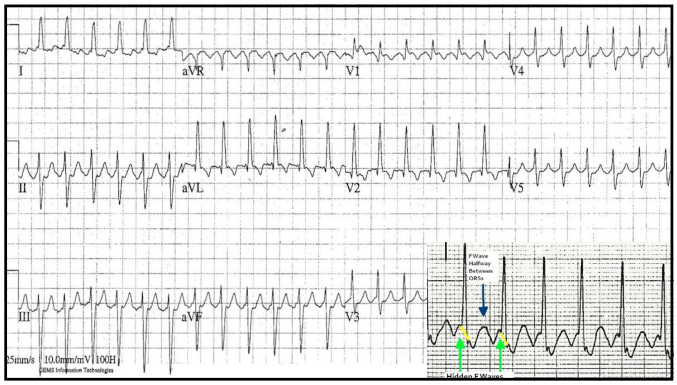

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
VI
II
V5

124


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
P Wave Halfway Between QRSs
Hidden P Waves

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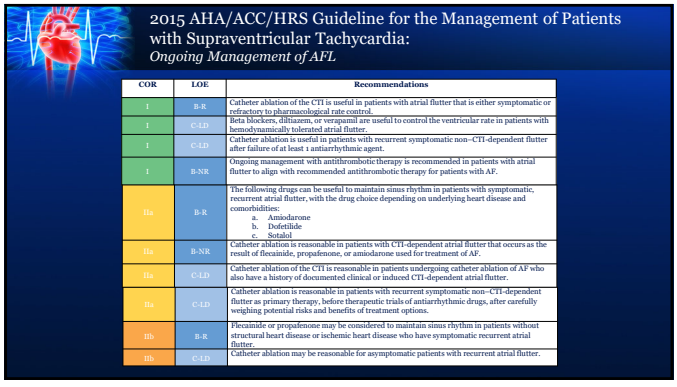
2015 AHA/ACC/HRS Guideline for the Management of Patients with Supraventricular Tachycardia:
Ongoing Management of AFL
COR
LOE
Recommendations
I
B-R
Catheter ablation of the CTI is useful in patients with atrial flutter that is either symptomatic or refractory to pharmacological rate control.
I
C-LD
Beta blockers, diltiazem, or verapamil are useful to control the ventricular rate in patients with hemodynamically tolerated atrial flutter.
I
C-LD
Catheter ablation is useful in patients with recurrent symptomatic non–CTI-dependent flutter after failure of at least 1 antiarrhythmic agent.
I
B-NR
Ongoing management with antithrombotic therapy is recommended in patients with atrial flutter to align with recommended antithrombotic therapy for patients with AF.
IIa
B-R
The following drugs can be useful to maintain sinus rhythm in patients with symptomatic, recurrent atrial flutter, with the drug choice depending on underlying heart disease and comorbidities:
a. Amiodarone
b. Dofetilide
c. Sotalol
IIa
B-NR
Catheter ablation is reasonable in patients with CTI-dependent atrial flutter that occurs as the result of flecainide, propafenone, or amiodarone used for treatment of AF.
IIa
C-LD
Catheter ablation of the CTI is reasonable in patients undergoing catheter ablation of AF who also have a history of documented clinical or induced CTI-dependent atrial flutter.
IIa
C-LD
Catheter ablation is reasonable in patients with recurrent symptomatic non–CTI-dependent flutter as primary therapy, before therapeutic trials of antiarrhythmic drugs, after carefully weighing potential risks and benefits of treatment options.
IIb
B-R
Flecainide or propafenone may be considered to maintain sinus rhythm in patients without structural heart disease or ischemic heart disease who have symptomatic recurrent atrial flutter.
IIb
C-LD
Catheter ablation may be reasonable for asymptomatic patients with recurrent atrial flutter.

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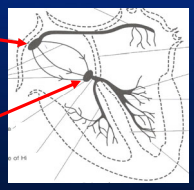
Bradyarrhythmia's

There are two types of bradyarrhythmia's

- Those related to problems with impulse formation
  - Sinus Arrest
  - Sinus Bradycardia
  - Chronotropic Incompetence
  - Tachy-Brady syndrome
- Those related to problems with impulse conduction
  - Sinus Exit Block
  - First Degree AV block
  - Second Degree AV block
    - Mobitz Type 1 – Wenckebach
    - Mobitz Type 2
  - Third Degree AV block – Complete heart block



(Issa et al., 2019; Zipes et. al., 2019)

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SA Node Dysfunction

A range of abnormalities encompassing sinus node and atrial impulse generation & propagation → inability of the SAN to generate a HR that meets physiologic needs

Intrinsic	Extrinsic
Cardiomyopathy (ischemic or nonischemic)	Autonomic perturbation
Congenital heart disease	• Carotid sinus hypersensitivity
Degenerative fibrosis	• Neurally-mediated syncope/presyncope
Infection/inflammation	• Physical conditioning
• Chagas disease	• Situational syncope
• Diphtheria	○ Cough
• Infectious endocarditis	○ Defecation
• Lyme disease	○ Glottic stimulation
• Myocarditis	○ Medical procedures
• Sarcoidosis	○ Micturition
• Toxoplasmosis	○ Vomiting
Infiltrative disorders	• Sleep (with or without sleep apnea)
• Amyloidosis	Metabolic
• Hemochromatosis	• Acidosis
• Lymphoma	• Hyperkalemia
Ischemia/infarction	• Hypokalemia
Rheumatological conditions	• Hypothermia
• Rheumatoid arthritis	• Hypothyroidism
• Scleroderma	• Hypoxia
• Systemic lupus erythematosus	
Surgical or procedural trauma	
• Cardiac procedures such as ablation or cardiac catheterization	
• Congenital heart disease surgery	
• Septal myomectomy for hypertrophic obstructive cardiomyopathy	
• Valve surgery (including percutaneous valve replacement)	

(AHA/ACC, 2018)

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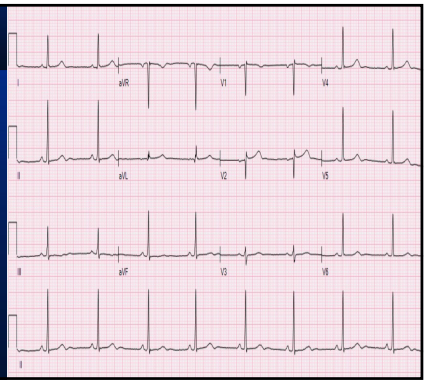
Sinus Bradycardia

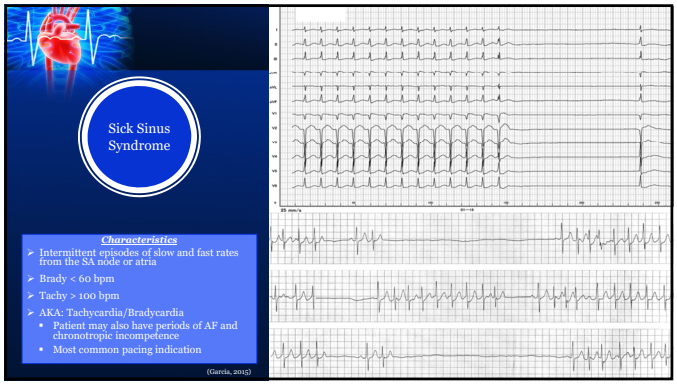
Characteristics

- Impulse arises from the SA node with a rate of <60 bpm
- Regular rhythm
- For each QRS, there is 1 P-wave, which is has an appropriate morphology
- PRI→ 0.12-0.20 sec
- QRS→ <0.12 sec
- Due to SA node disease or increased vagal tone



(García, 2015)

129


Sick Sinus Syndrome
Characteristics
Intermittent episodes of slow and fast rates from the SA node or atria
Brady < 60 bpm
Tachy > 100 bpm
AKA: Tachycardia/Bradycardia
Patient may also have periods of AF and chronotropic incompetence
Most common pacing indication
(Garcia, 2015)

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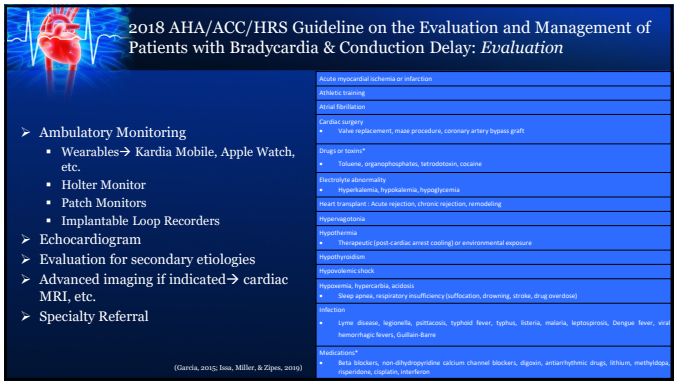
2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: Evaluation
Ambulatory Monitoring
Wearables→ Kardia Mobile, Apple Watch, etc.
Holter Monitor
Patch Monitors
Implantable Loop Recorders
Echocardiogram
Evaluation for secondary etiologies
Advanced imaging if indicated→ cardiac MRI, etc.
Specialty Referral
(Garcia, 2015; Issa, Miller, & Zipes, 2019)
Acute myocardial ischemia or infarction
Athletic training
Atrial fibrillation
Cardiac surgery
Valve replacement, maze procedure, coronary artery bypass graft
Drugs or toxins*
Toluene, organophosphates, tetrodotoxin, cocaine
Electrolyte abnormality
Hyperkalemia, hypokalemia, hypoglycemia
Heart transplant : Acute rejection, chronic rejection, remodeling
Hypervagotonia
Hypothermia
Therapeutic (post-cardiac arrest cooling) or environmental exposure
Hypothyroidism
Hypovolemic shock
Hypoxemia, hypercarbia, acidosis
Sleep apnea, respiratory insufficiency (suffocation, drowning, stroke, drug overdose)
Infection
Lyme disease, legionella, psittacosis, typhoid fever, typhus, listeria, malaria, leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre
Medications*
Beta blockers, non-dihydropyridine calcium channel blockers, digoxin, antiarrhythmic drugs, lithium, methyldopa, risperidone, cisplatin, interferon

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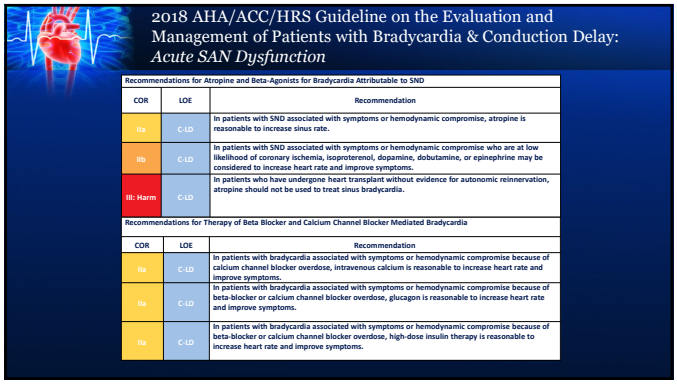
2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: Acute SAN Dysfunction
Recommendations for Atropine and Beta-Agonists for Bradycardia Attributable to SND
COR | LOE | Recommendation
IIa | C-LD | In patients with SND associated with symptoms or hemodynamic compromise, atropine is reasonable to increase sinus rate.
IIb | C-LD | In patients with SND associated with symptoms or hemodynamic compromise who are at low likelihood of coronary ischemia, isoproterenol, dopamine, dobutamine, or epinephrine may be considered to increase heart rate and improve symptoms.
III: Harm | C-LD | In patients who have undergone heart transplant without evidence for autonomic reinnervation, atropine should not be used to treat sinus bradycardia.
Recommendations for Therapy of Beta Blocker and Calcium Channel Blocker Mediated Bradycardia
COR | LOE | Recommendation
IIa | C-LD | In patients with bradycardia associated with symptoms or hemodynamic compromise because of calcium channel blocker overdose, intravenous calcium is reasonable to increase heart rate and improve symptoms.
IIa | C-LD | In patients with bradycardia associated with symptoms or hemodynamic compromise because of beta-blocker or calcium channel blocker overdose, glucagon is reasonable to increase heart rate and improve symptoms.
IIa | C-LD | In patients with bradycardia associated with symptoms or hemodynamic compromise because of beta-blocker or calcium channel blocker overdose, high-dose insulin therapy is reasonable to increase heart rate and improve symptoms.

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Acute SAN Dysfunction*

Recommendations for Temporary Pacing for Bradycardia Attributable to SND

COR	LOE	Recommendation
IIa	C-LD	In patients with persistent hemodynamically unstable SND refractory to medical therapy, temporary transvenous pacing is reasonable to increase heart rate and improve symptoms until a PPM is placed or the bradycardia resolves.
IIb	C-LD	In patients with SND with severe symptoms or hemodynamic compromise, temporary transcutaneous pacing may be considered to increase heart rate and improve symptoms until a temporary transvenous or PPM is placed or the bradycardia resolves.
III: Harm	C-LD	In patients with SND with minimal and/or infrequent symptoms without hemodynamic compromise, temporary transcutaneous or transvenous pacing should not be performed.

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Chronic SAN Dysfunction*

Recommendations for Permanent Pacing for Chronic Therapy/Management of Bradycardia Attributable to SND

COR	LOE	Recommendation
I	C-LD	In patients with symptoms that are directly attributable to SND, permanent pacing is indicated to increase heart rate and improve symptoms.
I	C-EO	In patients who develop symptomatic sinus bradycardia as a consequence of guideline-directed management and therapy for which there is no alternative treatment and continued treatment is clinically necessary, permanent pacing is recommended to increase heart rate and improve symptoms.

Recommendations for Permanent Pacing for Chronic Therapy/Management of Bradycardia Attributable to SND

COR	LOE	Recommendation
IIa	C-EO	For patients with tachy-brady syndrome and symptoms attributable to bradycardia, permanent pacing is reasonable to increase heart rate and reduce symptoms attributable to hypoperfusion.
IIa	C-EO	In patients with symptomatic chronotropic incompetence, permanent pacing with rate-responsive programming is reasonable to increase exertional heart rates and improve symptoms.
IIb	C-LD	In patients with symptoms that are likely attributable to SND, a trial of oral theophylline may be considered to increase heart rate, improve symptoms, and help determine the potential effects of permanent pacing.

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Chronic SAN Dysfunction*

Recommendations for Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to SND

COR	LOE	Recommendation
I	B-R	In symptomatic patients with SND, atrial-based pacing is recommended over single chamber ventricular pacing.
I	B-R	In symptomatic patients with SND and intact atrioventricular conduction without evidence of conduction abnormalities, dual chamber or single chamber atrial pacing is recommended.

Recommendations for Permanent Pacing Techniques and Methods for Chronic Therapy/Management of Bradycardia Attributable to SND

COR	LOE	Recommendation
IIa	B-R	In symptomatic patients with SND who have dual chamber pacemakers and intact atrioventricular conduction, it is reasonable to program the dual chamber pacemaker to minimize ventricular pacing.
IIa	C-EO	In symptomatic patients with SND in which frequent ventricular pacing is not expected or the patient has significant comorbidities that are otherwise likely to determine the survival and clinical outcomes, single chamber ventricular pacing is reasonable.

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Chronic SAN Dysfunction*

Recommendations for General Principles of Chronic Therapy/Management of Bradycardia Attributable to SND

COR	LOE	Recommendation
III: Harm	C-LD	In asymptomatic individuals with sinus bradycardia or sinus pauses that are secondary to physiologically elevated parasympathetic tone, permanent pacing should not be performed.
III: Harm	C-LD	In patients with sleep-related sinus bradycardia or transient sinus pauses occurring during sleep, permanent pacing should not be performed unless other indications for pacing are present.
III: Harm	C-LD	In patients with asymptomatic SND, or in those in whom the symptoms have been documented to occur in the absence of bradycardia or chronotropic incompetence, permanent pacing should not be performed.

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AV Conduction Disturbances

A block or delay of a cardiac impulse from the atria to the ventricles due to functional or anatomical impairments

Congenital/genetic
- Congenital AV block (associated with maternal systemic lupus erythematosus)
- Congenital heart defects (e.g., L-TGA)
- Genetic (e.g., SCN5A mutations)

Infectious
- Lyme carditis
- Bacterial endocarditis with perivalvar abscess
- Acute rheumatic fever
- Chagas disease
- Toxoplasmosis

Inflammatory/infiltrative
- Myocarditis
- Amyloidosis
- Cardiac sarcoidosis
- Rheumatologic disease: Systemic sclerosis, SLE, RA, reactive arthritis (Reiter's syndrome)
- Other cardiomyopathy–idiopathic, valvular

Ischemic
- Acute MI
- Coronary ischemia without infarction—unstable angina, variant angina
- Chronic ischemic cardiomyopathy

Degenerative
- Lev's and Lenegre's diseases

Vagotonic-associated with increased vagal tone
- Sleep, obstructive sleep apnea
- High-level athletic conditioning
- Neurocardiogenic

Metabolic/endocrine
- Acid-base disorders
- Poisoning/overdose (e.g., mercury, cyanide, carbon monoxide, mad honey)
- Thyroid disease (both hypothyroidism and hyperthyroidism)
- Adrenal disease (e.g., pheochromocytoma, hypoaldosteronism)

Other diseases
- Neuromuscular diseases (e.g., myotonic dystrophy, Kearns-Sayre syndrome, Erb's dystrophy)
- Lymphoma

Iatrogenic
- Medication related
  - Beta blockers, verapamil, diltiazem, digoxin
  - Antiarrhythmic drugs
  - Neutraceuticals
- Catheter ablation
- Cardiac surgery, especially valve surgery
- TAVR, alcohol septal ablation

(AHA/ACC,2018)

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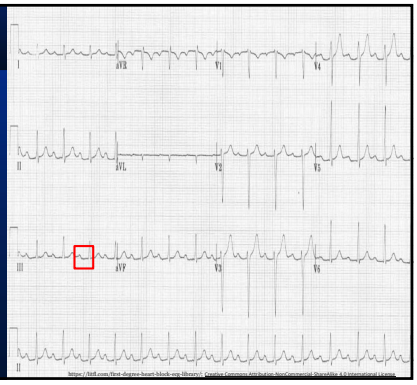
1° AV Block

- PRI→ >0.20 sec
- Delay in conduction from the atria to the ventricles
- Typically benign

(Issa et al., 2019; Zipes et. al., 2019)



https://litfl.com/first-degree-heart-block-ecg-library/ | Creative Commons Attribution-NonCommercial-ShareAlike 4.0 International License

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2° AV Block Type I (Wenckebach)
Conduction block at the level of the AV node
Typically benign with minimal hemodynamic consequences
Most patients are asymptomatic
(Issa et al., 2019; Zipes et. al., 2019)

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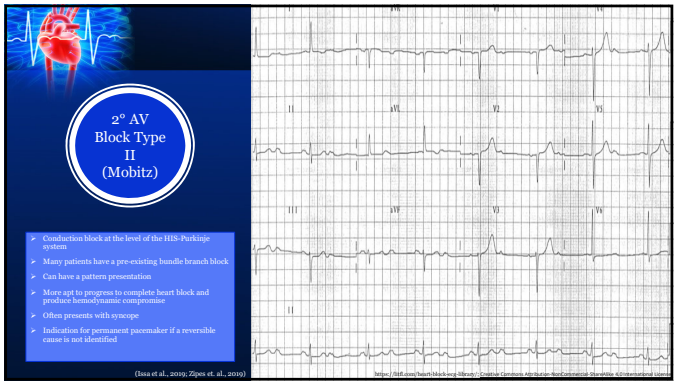
2° AV Block Type II (Mobitz)
Conduction block at the level of the HIS-Purkinje system
Many patients have a pre-existing bundle branch block
Can have a pattern presentation
More apt to progress to complete heart block and produce hemodynamic compromise
Often presents with syncope
Indication for permanent pacemaker if a reversible cause is not identified
(Issa et al., 2019; Zipes et. al., 2019)

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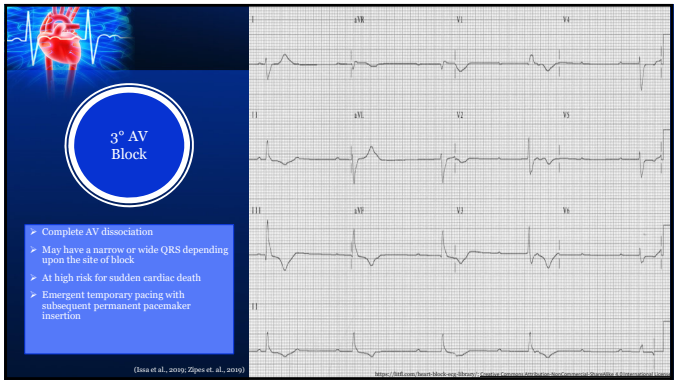
3° AV Block
Complete AV dissociation
May have a narrow or wide QRS depending upon the site of block
At high risk for sudden cardiac death
Emergent temporary pacing with subsequent permanent pacemaker insertion
(Issa et al., 2019; Zipes et. al., 2019)

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Acute AV Block*

Recommendations for Acute Management of Reversible Causes of Bradycardia Attributable to Atrioventricular Block		
COR	**LOE**	**Recommendation**
I	B-NR	Patients with transient or reversible causes of atrioventricular block, such as Lyme carditis or drug toxicity, should have medical therapy and supportive care, including temporary transvenous pacing if necessary, before determination of need for permanent pacing.
IIa	B-NR	In selected patients with symptomatic second-degree or third-degree atrioventricular block who are on chronic stable doses of medically necessary antiarrhythmic or beta-blocker therapy, it is reasonable to proceed to permanent pacing without further observation for drug washout or reversibility.

Recommendations for Acute Management of Reversible Causes of Bradycardia Attributable to Atrioventricular Block		
COR	**LOE**	**Recommendation**
IIa	B-NR	In patients with second-degree or third-degree atrioventricular block associated with cardiac sarcoidosis, permanent pacing, with additional defibrillator capability if needed and meaningful survival of greater than 1 year is expected, without further observation for reversibility is reasonable.
IIb	C-LD	In patients with symptomatic second-degree or third-degree atrioventricular block associated with thyroid function abnormalities but without clinical myxedema, permanent pacing without further observation for reversibility may be considered.

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Acute AV Block*

Recommendations for Acute Medical Therapy for Bradycardia Attributable to Atrioventricular Block		
COR	**LOE**	**Recommendation**
IIa	C-LD	For patients with second-degree or third-degree atrioventricular block believed to be at the atrioventricular nodal level associated with symptoms or hemodynamic compromise, atropine is reasonable to improve atrioventricular conduction, increase ventricular rate, and improve symptoms.
IIb	B-NR	For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise and who have low likelihood for coronary ischemia, beta-adrenergic agonists, such as isoproterenol, dopamine, dobutamine, or epinephrine, may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms.
IIb	C-LD	For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise in the setting of acute inferior MI, intravenous aminophylline may be considered to improve atrioventricular conduction, increase ventricular rate, and improve symptoms.

Recommendations for Temporary Pacing for Bradycardia Attributable to Atrioventricular Block		
COR	**LOE**	**Recommendation**
IIa	B-NR	For patients with second-degree or third-degree atrioventricular block associated with symptoms or hemodynamic compromise that is refractory to medical therapy, temporary transvenous pacing is reasonable to increase heart rate and improve symptoms.
IIa	B-NR	For patients who require prolonged temporary transvenous pacing, it is reasonable to choose an externalized permanent active fixation lead over a standard passive fixation temporary pacing lead.
IIb	B-R	For patients with second-degree or third-degree atrioventricular block and hemodynamic compromise refractory to antibradycardic medical therapy, temporary transcutaneous pacing may be considered until a temporary transvenous or PPM is placed or the bradyarrhythmia resolves.

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2018 AHA/ACC/HRS Guideline on the Evaluation and Management of Patients with Bradycardia & Conduction Delay: *Chronic AV Block*

Recommendations for General Principles of Chronic Therapy/Management of Bradycardia Attributable to Atrioventricular Block		
COR	**LOE**	**Recommendation**
III: Harm	C-LD	In patients with first-degree atrioventricular block or second-degree Mobitz type I (Wenckebach) or 2:1 atrioventricular block which is believed to be at the level of the atrioventricular node, with symptoms that do not temporally correspond to the atrioventricular block, permanent pacing should not be performed.
III: Harm	C-LD	In asymptomatic patients with first-degree atrioventricular block or second-degree Mobitz type I (Wenckebach) or 2:1 atrioventricular block which is believed to be at the level of the atrioventricular node, permanent pacing should not be performed.

Recommendations for Potentially Reversible or Transient Causes of Atrioventricular Block		
COR	**LOE**	**Recommendation**
I	C-LD	In patients with symptomatic atrioventricular block attributable to a known reversible cause in whom the atrioventricular block does not resolve despite treatment of the underlying cause, permanent pacing is recommended.
III: Harm	C-LD	In patients who had acute atrioventricular block attributable to a known reversible and nonrecurrent cause and have had complete resolution of the atrioventricular block with treatment of the underlying cause, permanent pacing should not be performed.
III: Harm	C-LD	In patients with asymptomatic vagally mediated atrioventricular block, permanent pacing should not be performed.

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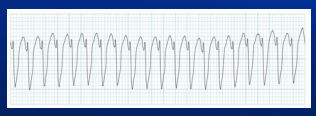
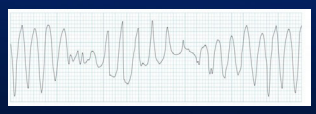
Ventricular Tachycardia

- Hemodynamic Consequences
  - Decreased CO
  - Decreased myocardial perfusion
  - Cardiovascular collapse
- Classification
  - Morphology→ Mono, Poly, etc.
  - Duration→ Sustained vs NSVT
  - Hemodynamic stability
- Treatment
  - ID and treat reversible causes
  - Antiarrhythmic therapy→ Amiodarone, etc.
  - ICD Placement for secondary prevention



https://litfl.com/ventricular-tachycardia-monomorphic-ecg-library/



Retrieved from https://litfl.com/polymorphic-vt-and-torsades-de-pointes-tdp/

(Issa et al., 2019; Zipes et. al., 2019)

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Wide QRS Differentials

Wide QRS Differentials →

- Hyperkalemia
- Ventricular tachycardia
- Idioventricular rhythms
- Drug effects/Overdoses
- Wolff-Parkinson-White
- Bundle branch blocks
- IVCD's
- PVC's
- Aberrantly conducted complexes
- Inherited Channelopathies

(Issa et al., 2019; Zipes et. al., 2019)

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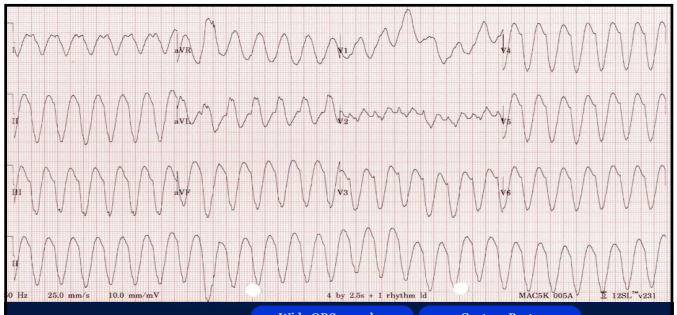

ECG Characteristics of Monomorphic VT

Wide QRS complex
ERAD
AV Dissociation
Precordial Concordance

Capture Beats
Fusion Beats
Brugada Sign
Josephson Sign

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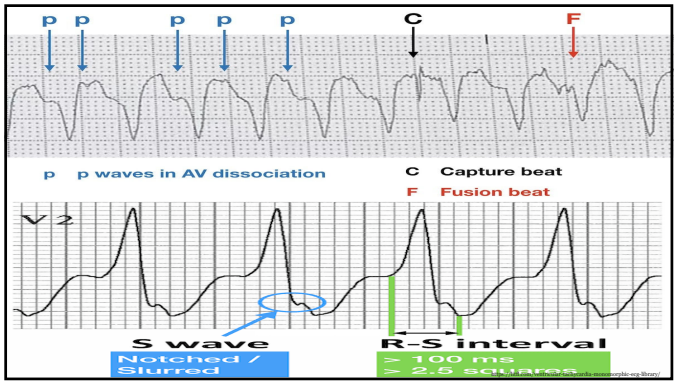
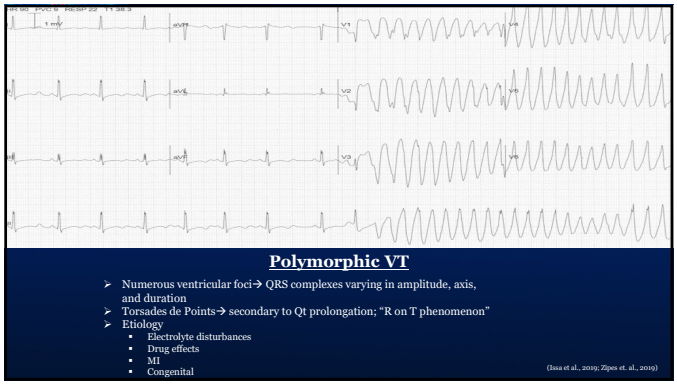

p p p p p C F
p p waves in AV dissociation
C Capture beat
F Fusion beat
V 2
S wave
Notched / Slurred
R-S interval
> 100 ms
> 2.5 squares

148


Polymorphic VT
Numerous ventricular foci→ QRS complexes varying in amplitude, axis, and duration
Torsades de Points→ secondary to Qt prolongation; "R on T phenomenon"
Etiology
Electrolyte disturbances
Drug effects
MI
Congenital
(Issa et al., 2019; Zipes et. al., 2019)

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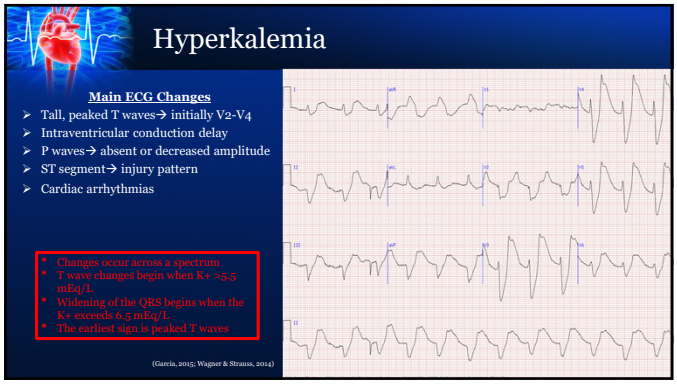
Hyperkalemia
Main ECG Changes
Tall, peaked T waves→ initially V2-V4
Intraventricular conduction delay
P waves→ absent or decreased amplitude
ST segment→ injury pattern
Cardiac arrhythmias
Changes occur across a spectrum
T wave changes begin when K+ >5.5 mEq/L
Widening of the QRS begins when the K+ exceeds 6.5 mEq/L
The earliest sign is peaked T waves
(Garcia, 2015; Wagner & Strauss, 2014)

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Effect	Agent	Dose	Onset	Duration
Membrane Stabilization	Calcium Gluconate (10%)	10mL IV over 10 min	Immediate	30 – 60 minutes
	Hypertonic (3%) Normal Saline	50mL IV push	Immediate	Unknown
Shifters	Insulin (Short Acting)	10 units IV push with 25 – 40 g dextrose (50% solution)	20 minute	4 – 6 hours
	Albuterol	10 – 20 mg in 4 mL of Normal Saline nebulized over 10 minutes	30 minute	2 hours
Excreters	Furosemide	40 – 80 mg IV x1	15 minute	2 -3 hours
	Sodium Bicarbonate	150mmol/L IV at variable rate	Hours	Duration of Infusion
	Sodium Polystyrene Sulfonate	15 – 30 g in 15 – 30 mL (70% sorbitol orally)	> 2 hours	4 – 6 hours
Definitive	Hemodialysis	-----	Immediate	3 hours

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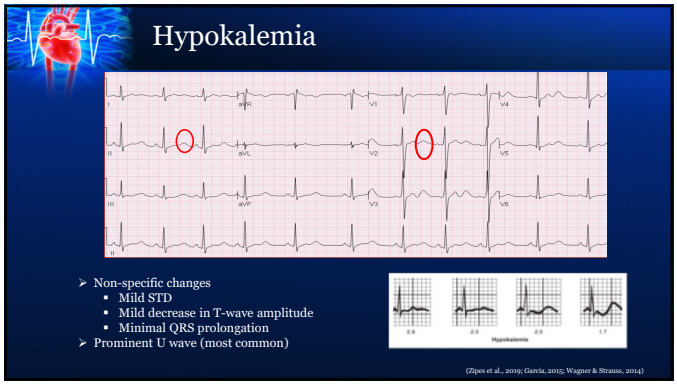
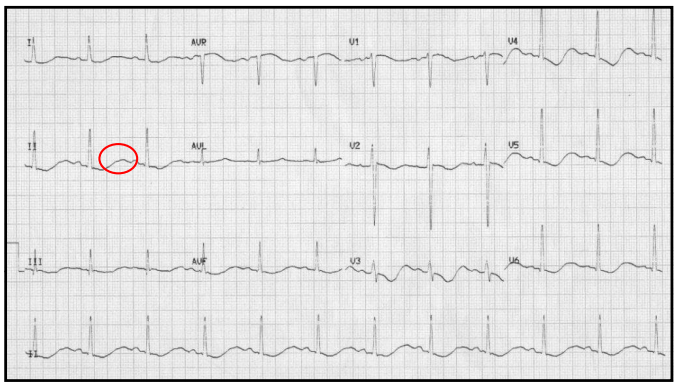


152



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Hypercalcemia/Hypocalcemia

Normal serum calcium = 8.5-10.5 mg/dL
Ionized calcium= 4.6-5.3 mg/dL

Hypercalcemia

ECG Changes
- Short QT interval→ typically too minimal to clinically correlate
- Severe→ Osborn J waves, VF/VT

Causes
- Hyperparathyroidism
- Malignancy associated
- Sarcoidosis
- Iatrogenic

Hypocalcemia

ECG Changes
- QT prolongation…Differentials?
- Torsades may occur

Causes
- Hypoparathyroidism
- Vitamin D deficiency
- Acute pancreatitis
- Other electrolyte disturbances
- Diuretics
- Congenital disorders
- Critical illness (e.g. sepsis)

(Wagner & Strauss, 2014; García, 2015; Zipes et al., 2019)

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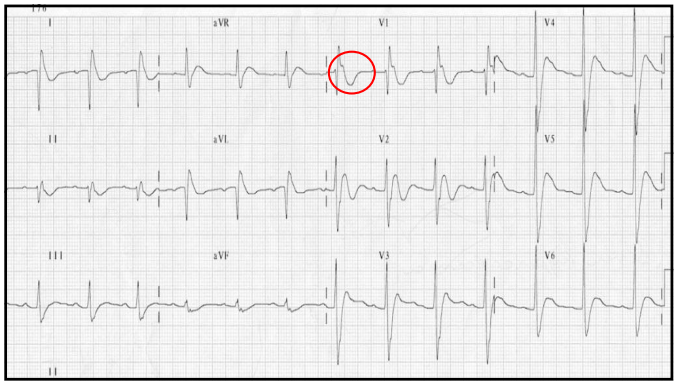

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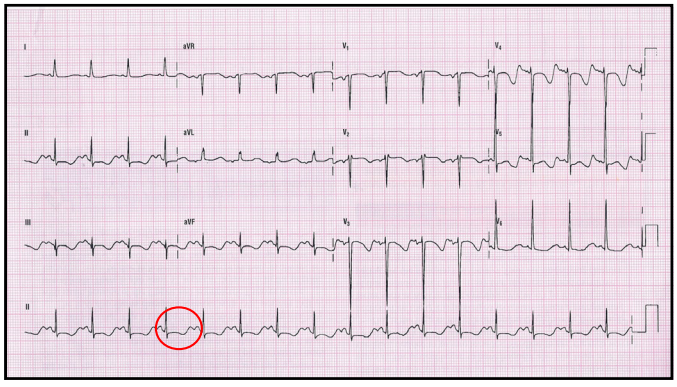

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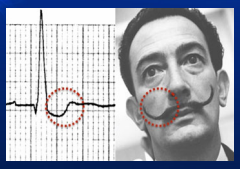
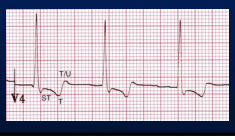
Digoxin Effect/Toxicity





General Considerations

- Very narrow therapeutic range → 0.8-2.0 ng/mL
- Toxicity
  - Potentiated by hypokalemia
  - Can cause any and all arrhythmias secondary to increased automaticity or decreased AV conduction
- Effect→ normal therapeutic dose
  - Downsloping, scooped ST segment
  - T waves→ Flat, inverted, biphasic
  - QT interval→ short
  - Prominent U waves
  - Mild PR prolongation

(Issa et al., 2019; Zipes et. al., 2019)

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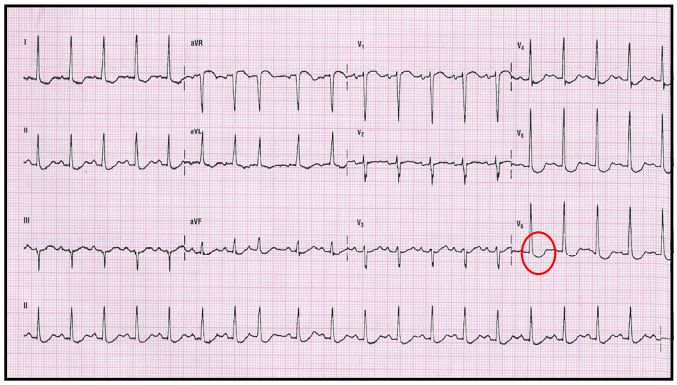

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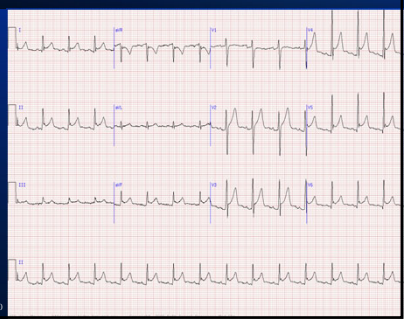
Pericarditis

Main Criteria

- PR depression
- Diffuse ST elevation
- Scooping, upwardly concave ST segment
- QRS notching



(Issa et al., 2019; Zipes et. al., 2019)

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Drugs that Cause QT Prolongation

- Antidepressants
  - SSRI's
  - TCA's
  - Lithium
  - Bupropion
  - Venlafaxine
- Antipsychotics
  - Haloperidol
  - Ziprasidone
  - Chlorpromazine
  - Quetiapine
- Antiemetics
  - Ondansetron
- Antimicrobials
  - Fluoroquinolone's
  - Macrolide's
  - Fluconazole, Voriconazole
- Antiarrhythmics
  - Amiodarone
  - Sotalol
  - Flecainide
  - Procainamide
- Other Drugs
  - Hydroxychloroquine
  - Methadone

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Putting It All Together

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1. Evaluate the ECG for a general impression

a. General rate (tachy/brady)
b. General rhythm (regular/irregular)
c. Scan for overt abnormalities that would point to any emergent/life threatening issues that need to be addressed immediately (AMI, hyperkalemia, etc).
d. Proceed to a full interpretation

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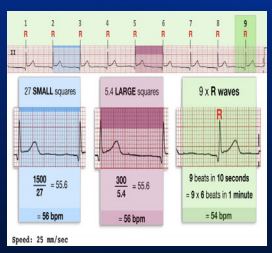
2. Calculate the Rate



163

3. Evaluate Waves & Intervals

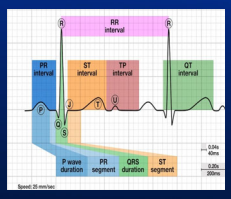
a. P waves & PRI
   - ✓ Are p waves present?
   - ✓ What is the p morphology (consistency)?
   - ✓ What is the general PRI length (short vs long) & duration?
   - ✓ Are the PRI's consistent, is there depression?

b. QRS
   - ✓ What is the general QRS width (narrow vs wide) & duration?
   - ✓ What is the QRS morphology?
   - ✓ What is the P:QRS ratio?
   - ✓ What is the RR interval

c. Is the J point sharp or diffuse?

d. QT interval→ normal, prolonged, short?

e. U waves→ are they present



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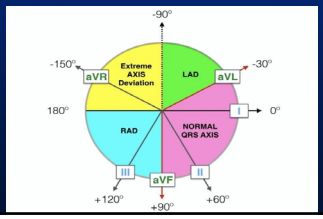
4. Calculate the Axis

a. Isolate the quadrant

b. Isolate the equiphasic (isoelectric) lead→ the positive deflection is equal to the negative deflection

c. Find the lead that is perpendicular to the isoelectric lead

d. Match the quadrant and isolate the vector to 30°



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5. Evaluate for the Presence of Hypertrophy

a. Left Ventricular Hypertrophy
- ✓ **S wave depth in V1 or V2 (deepest) + R wave height in V5 or V6 (tallest) ≥ 35mm**
- ✓ Precordial R wave ≥ 45mm
- ✓ aVL R wave ≥ 11
- ✓ Lead I R wave ≥ 12
- ✓ Lead aVF R wave ≥ 20
- ✓ LAD
- ✓ Strain pattern

b. Right Ventricular Hypertrophy
- ✓ **Dominant R wave in V1→ R:S in V1 is > 1**
- ✓ Dominant S wave in V5 or V6→ R:S <1
- ✓ RAD
- ✓ RV strain pattern
- ✓ RAE
- ✓ QRS <0.12
- ✓ Poor R wave progression

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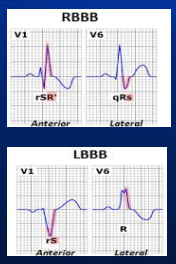
6. Evaluate for the Presence of a BBB & LAFB/LPFB

a. Is there a wide QRS present?
b. Evaluate V1 First as this will help lead you to your answer. Do not go straight to Lead I and V6.
c. Is the QRS Wide POSITIVE with a possible RsR'? If yes, this leads you towards a RBBB
d. Is the QRS Wide NEGATIVE? If yes, this leads you towards a LBBB
e. Evaluate V6 and Lead I for a slurred S wave (RBBB) vs. broad monomorphic R waves (LBBB)
f. Is the criteria for **LAFB** present?
- ✓ Positive QRS in Lead I
- ✓ Negative QRS in Lead II
- ✓ Negative QRS in aVF

g. Is the criteria for **LPFB** present?
- ✓ Right axis deviation
- ✓ An s wave in lead I
- ✓ A q in lead III
- ✓ No evidence of RVH



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7. Evaluate for Ischemia/Infarction

a. Perform a regional review→ You can start with any region that you choose, just be systematic.
b. Evaluate the T-waves and ST segments
- ✓ Where is the J point?
- ✓ Is the ST segment concave up/down, flat, depressed, elevated, or in a tombstone pattern? If there is depression/elevation is it present in 2 or more contiguous leads? How many mm?
- ✓ Is the T wave abnormal? Is it symmetric (abnormal) or asymmetrical (normal)? Is it tall, peaked, broad, inverted, biphasic?

c. Are reciprocal changes present?
d. Is there posterior wall or RV involvement?→ If an inferior MI is present, always assess for an RV infarction and/or posterior wall involvement as well. These often go together
e. Correlate your EKG findings with the patient presentation as different regions produce different hemodynamic alterations.

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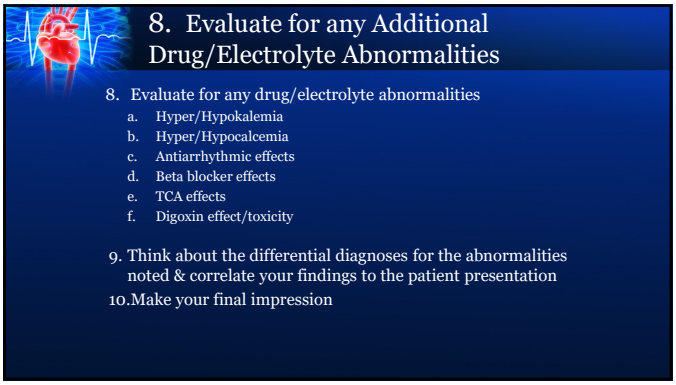
8. Evaluate for any Additional Drug/Electrolyte Abnormalities
8. Evaluate for any drug/electrolyte abnormalities
a. Hyper/Hypokalemia
b. Hyper/Hypocalcemia
c. Antiarrhythmic effects
d. Beta blocker effects
e. TCA effects
f. Digoxin effect/toxicity
9. Think about the differential diagnoses for the abnormalities noted & correlate your findings to the patient presentation
10. Make your final impression

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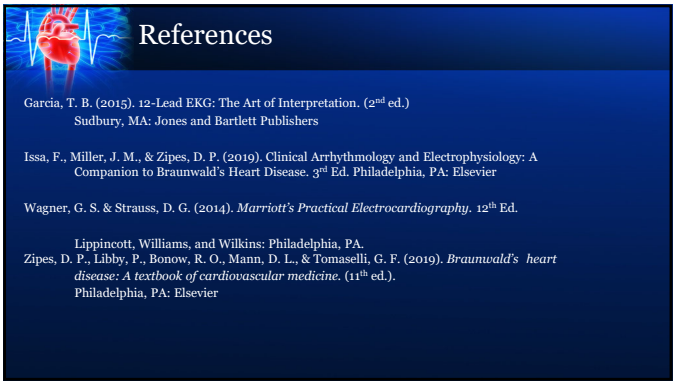
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Wagner, G. S. & Strauss, D. G. (2014). Marriott's Practical Electrocardiography. 12th Ed. Lippincott, Williams, and Wilkins: Philadelphia, PA.
Zipes, D. P., Libby, P., Bonow, R. O., Mann, D. L., & Tomaselli, G. F. (2019). Braunwald's heart disease: A textbook of cardiovascular medicine. (11th ed.). Philadelphia, PA: Elsevier

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