Pediatric EKG Interpretation

Laila Tutunji, MD

Introduction

What you need before you begin?

- 1 Calculator
- 2 Percentile Charts (Harriet Lane, Davignon)
- 3 Callipers are a plus

Introduction

- Values are dynamic and change with age.

Introduction

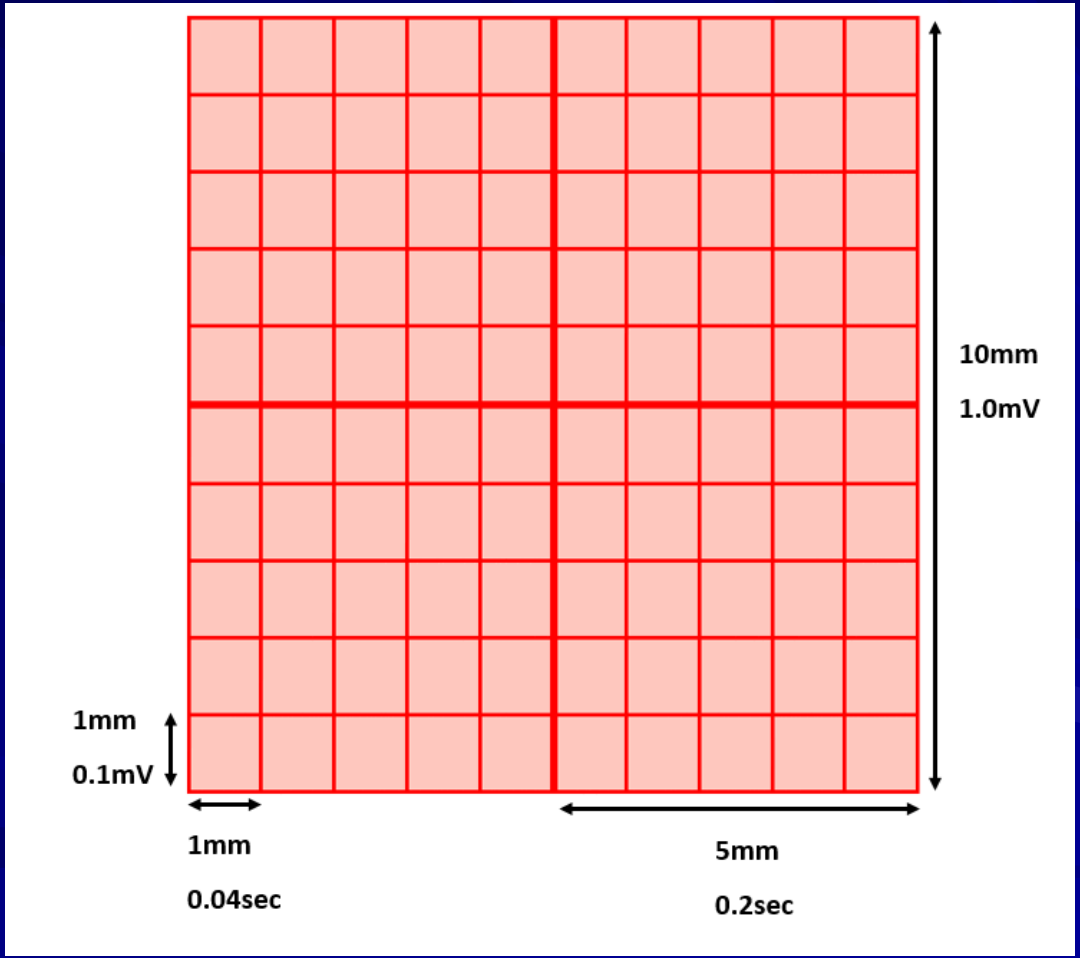
- 12 lead EKG
- Anatomy of an impulse

12 Lead ECG

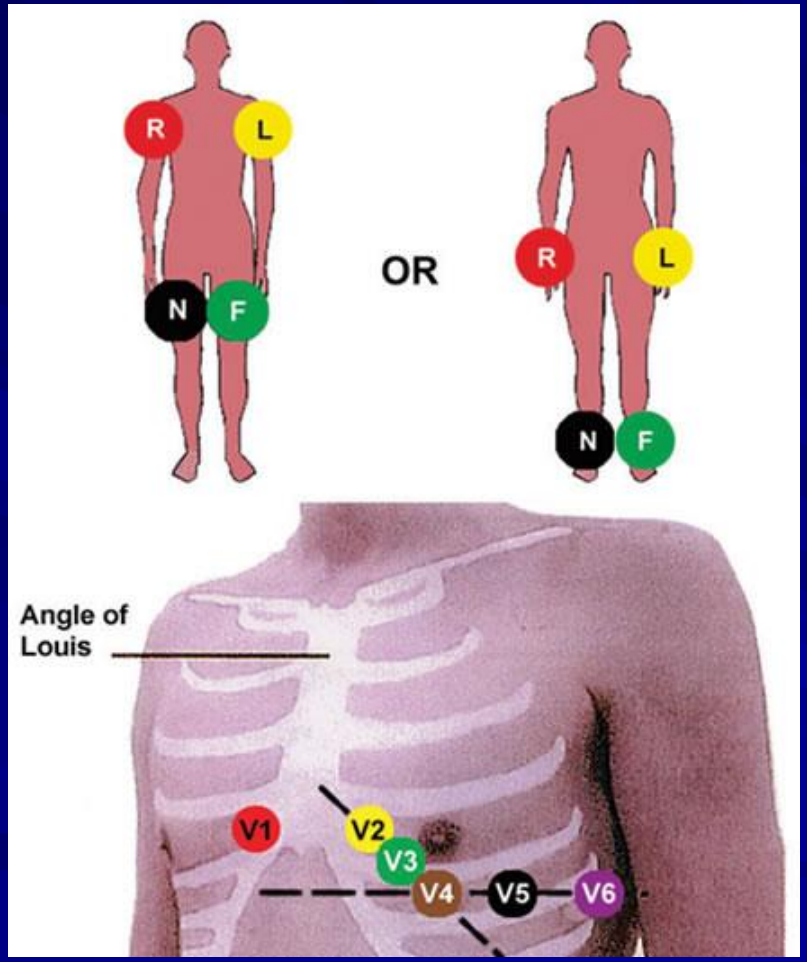
- EKG is a voltmeter ie measures voltage which has magnitude and direction.

- Voltage displayed on the Y axis (1mV=10mm) and time is displayed on the x axis (1 little block = 0.04 seconds)

ECG Paper



ECG Lead Placement



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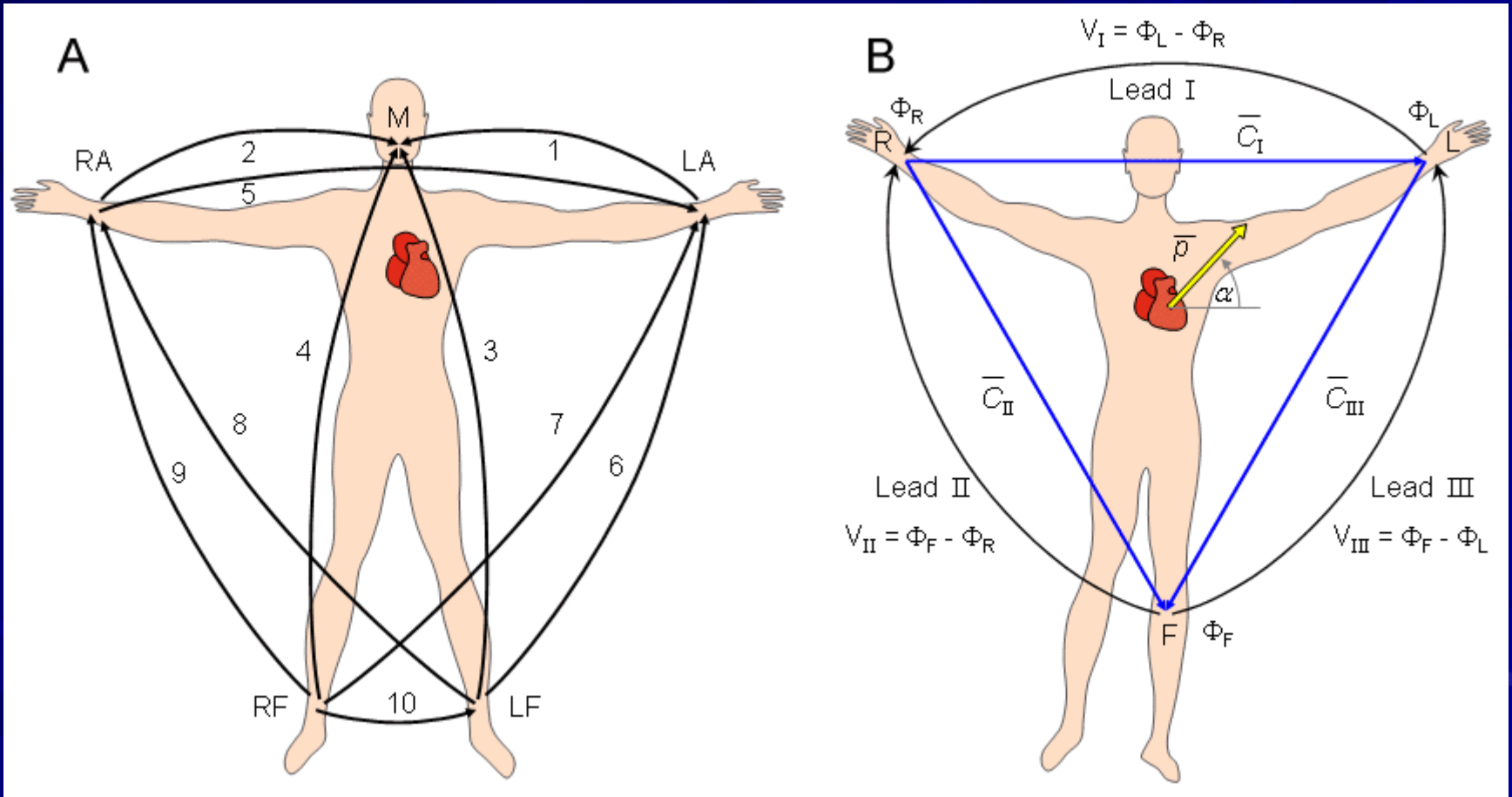
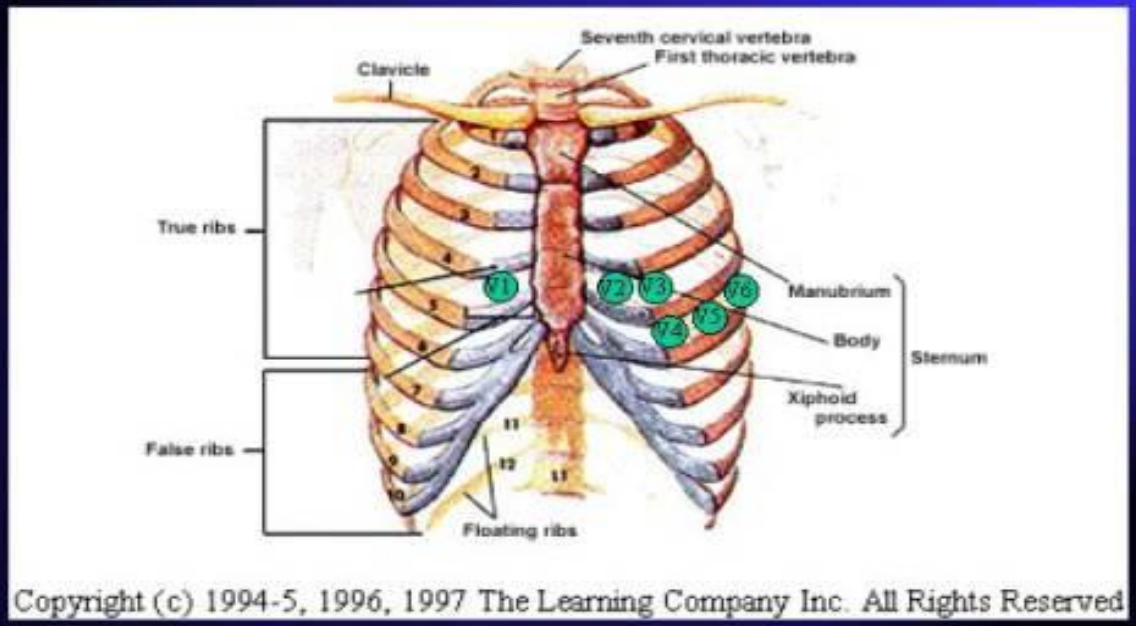
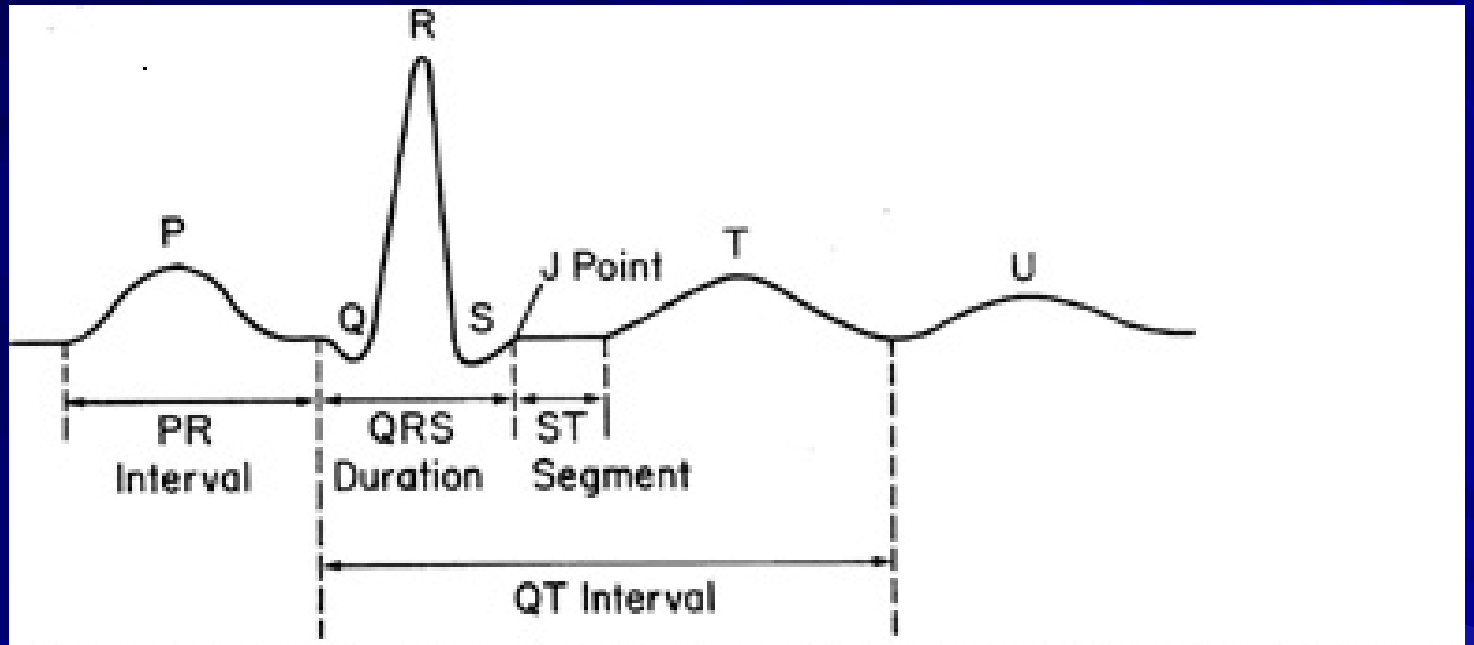


ECG Lead Placement

12-Lead EKG Placement



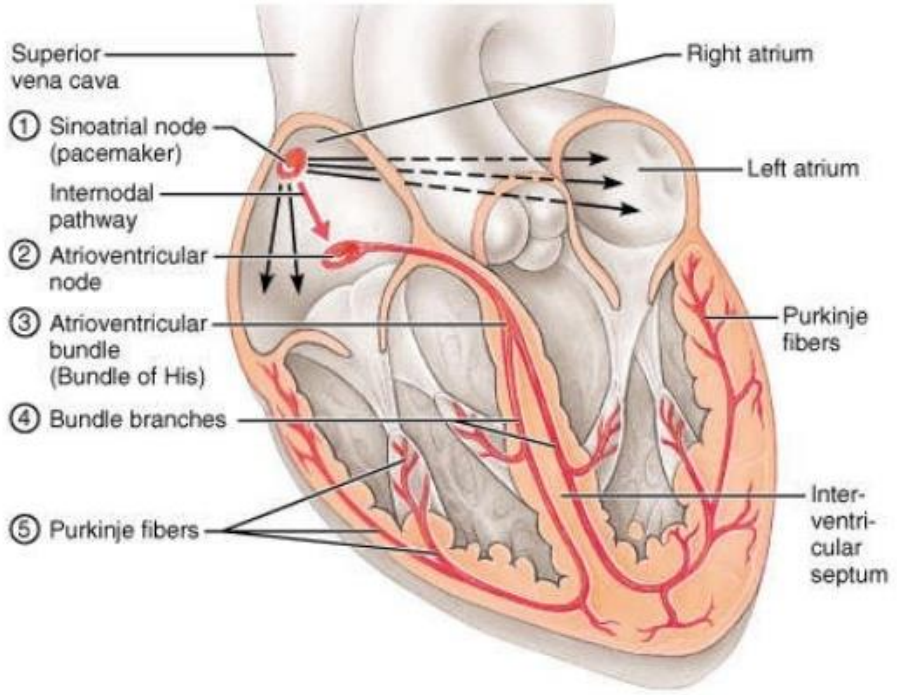
QRS



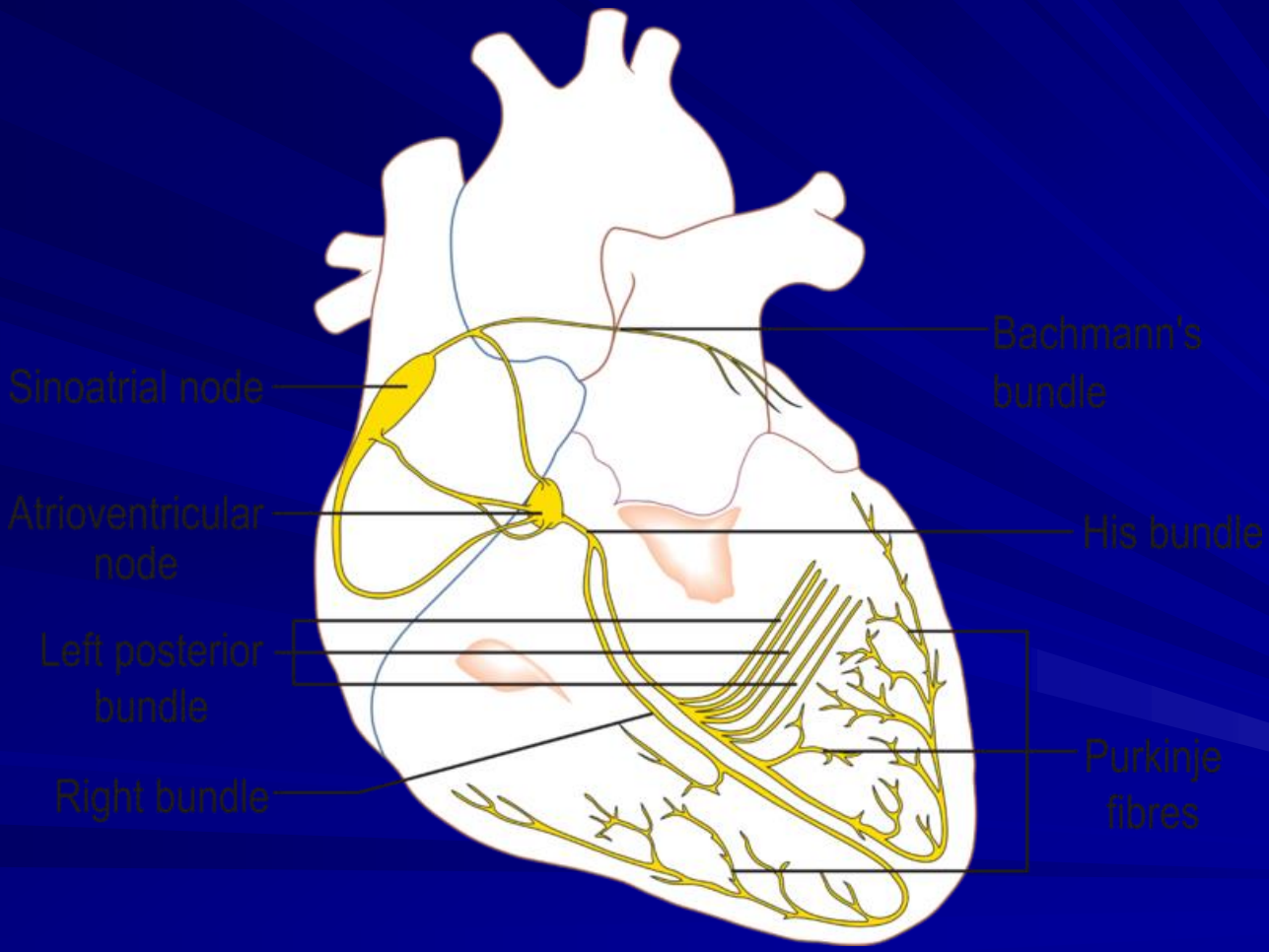
Anatomy of an Impulse

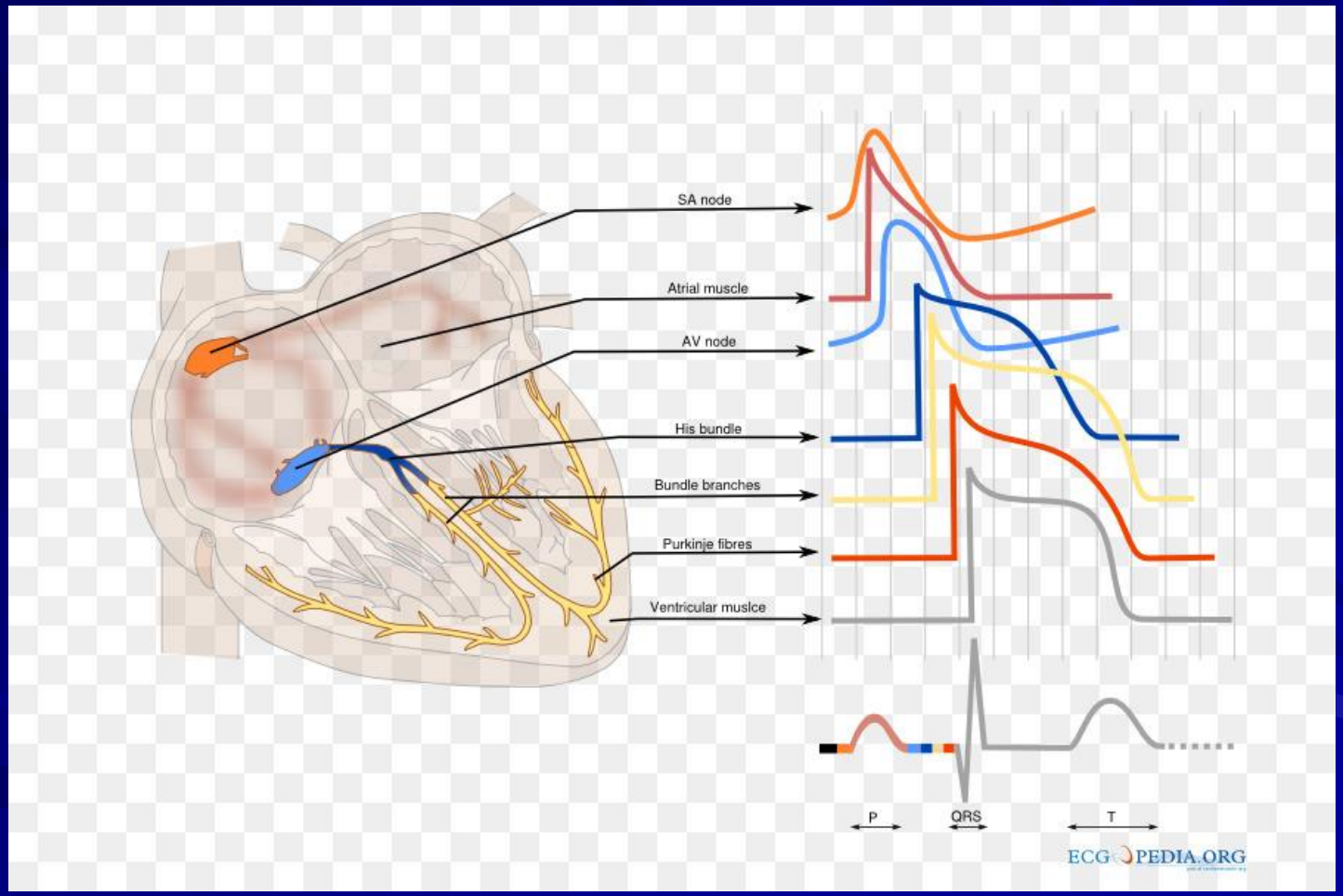
Conducting System

- Network of specialized tissue that stimulates contraction
- Modified cardiac myocytes
- The heart can contract without any innervation



Electrical Conduction System




SA node
Atrial muscle
AV node
His bundle
Bundle branches
Purkinje fibres
Ventricular muscle
P
QRS
T
ECG PEDIA.ORG

ECG Analysis

Always read an EKG systematically

1. Rhythm
2. Rate
3. QRS axis
4. Intervals :
   - PR interval
   - QRS duration
   - QT interval
5. QRS amplitude, R/S ratio, abnormal Q waves
6. ST-segment and T wave abnormality

1. Rhythm

Sinus or not

- Sinus rhythm:
- P before every QRS
- P wave morphology
- Regular PR interval
- Normal P wave Axis

1. Rhythm

- P wave duration < 0.07 sec in infants, < 0.09 in children
- LAH duration > 0.08 sec in infants and >0.1 sec in children
- P wave amplitude < 3 mm
- RAH > 3 mm
- Combined atrial hypertrophy

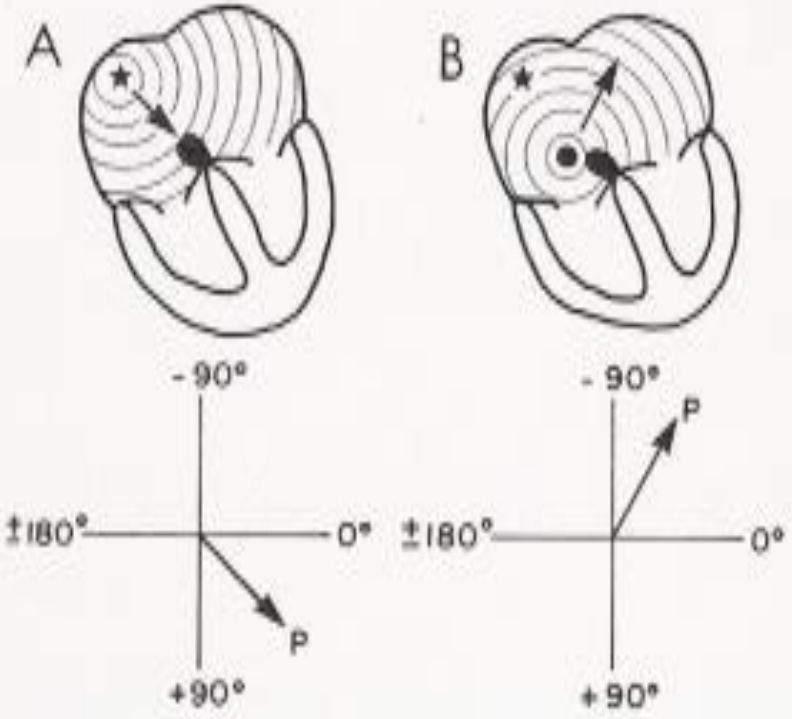


FIG. 3–3.
Comparison of P axis in sinus rhythm **(A)**, and low atrial rhythm **(B)**. In sinus rhythm, the P waves are upright in leads I and aVF. In low atrial rhythm, the P wave is inverted in lead aVF.

2. Rate

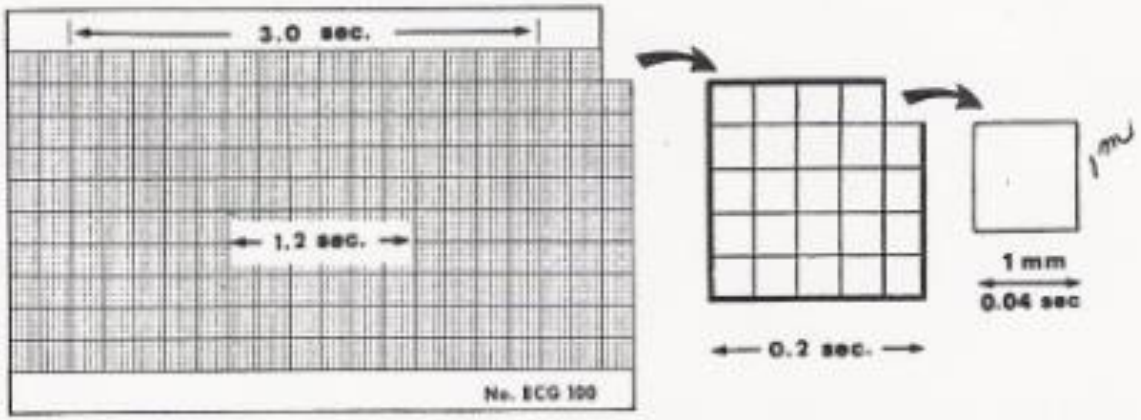


FIG. 3–4.
ECG paper. Time is measured on the horizontal axis. Each 1 mm = 0.04 second, and each 5 mm (a large division) = 0.20 second; 30 mm (or 6 large divisions) = 1.2 second or 1/50 minute. Every 7.5 cm marked on the top margin of the paper = 3.0 second or 1/20 minute. (From Park MK, Guntheroth WG: *How to read pediatric ECGs,* ed 3, St Louis, 1992, Mosby.)

2. Rate

1 mm = 0.04 sec, 5 mm = 0.2 sec

Measure between R – R’

- measure duration in seconds, Rate = 60/duration
- measure large divisions, Rate = 300/ number of large divisions
- 1 minute = 60 seconds, and 300 large divisions

2. Rate

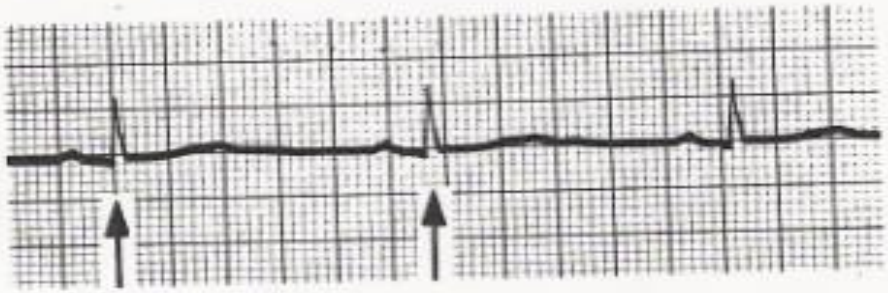


FIG. 3–6.
Heart rate of 52 beats/min. There are 5.8 large divisions between the two *arrows*. Therefore the heart rate is 300 ÷ 5.8 = 52.

FIG. 3–7.
Quick estimation of heart rate. When the R-R interval is 5 mm, the heart rate is 300 beats/min. When the R-R interval is 10 mm, the rate is 150 beats/min, and so on.

2. Rate

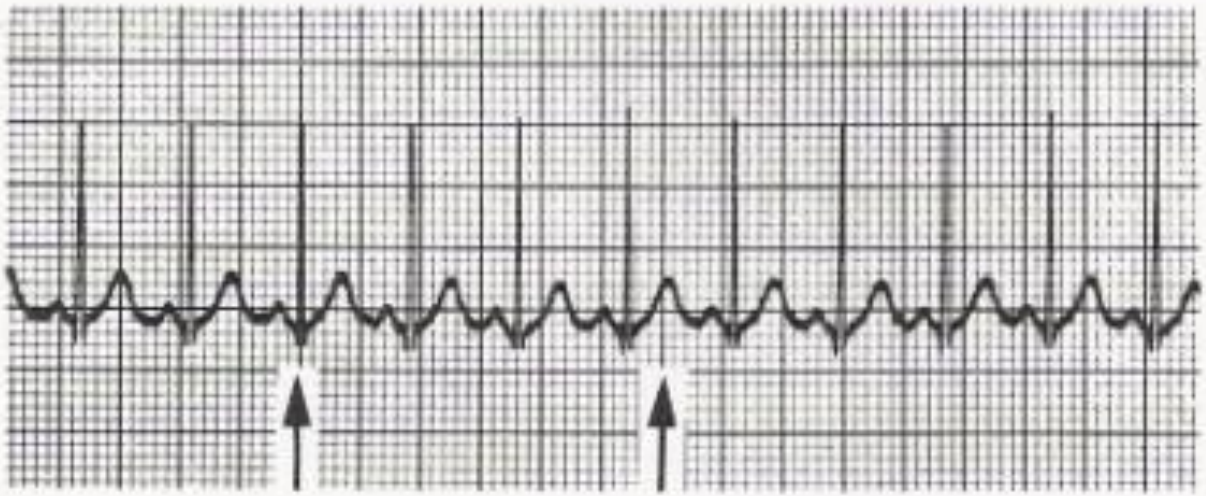


FIG. 3–5.
Heart rate of 165 beats/min. There are about 3.3 cardiac cycles (R-R intervals) in six large divisions. Therefore the heart rate is $3.3 \times 50 = 165$.

2. Rate

Count R-R cycles

- In 6 large divisions, multiply cycles by 50
- In 3 seconds = marks on top margin of paper , multiply cycles by 20

Quick and easy; 300/150/100/75/60/50

Tachycardia and Bradycardia, check normal values for age.

3. QRS Axis

- Hexaxial System, Limb leads
- Frontal Plane

- Left vs right, superior and inferior
- Lead I left (positive) vs right (negative)
- AvF downward (positive) vs upward (negative)

3. Axis

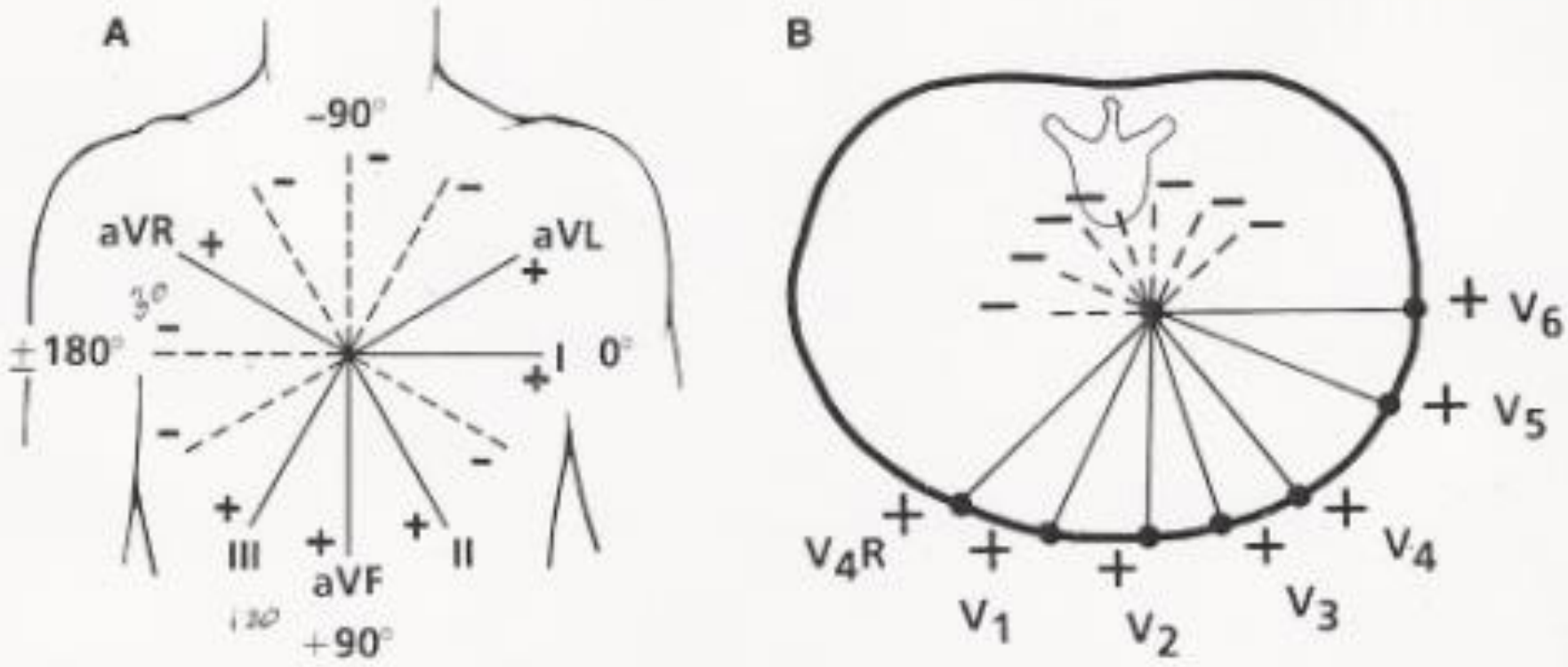


FIG. 3–8.
Hexaxial **(A)** and horizontal **(B)** reference systems. (From Park MK, Guntheroth WG: *How to read pediatric ECGs,* ed 3, St Louis, 1992, Mosby.)

Horizontal Reference System

- Right and left precordial leads

- V2 is perpendicular to V6
- V2 anterior (positive) posterior (negative)
- V6 left (positive) right (negative)
- V1 anterior and right (positive) posterior and left (negative)

The QRS axis is perpendicular to the lead with an equiphasic QRS complex in the predetermined quadrant.

Example: Determine the QRS axis in Fig. 3-11.

Step 1: The axis is in the left lower quadrant (i.e., 0 to +90 degrees), since the R waves are upright in leads I and aVF.

Step 2: The QRS complex is equiphasic in aVL. Therefore the QRS axis is +60 degrees, which is perpendicular to aVL.

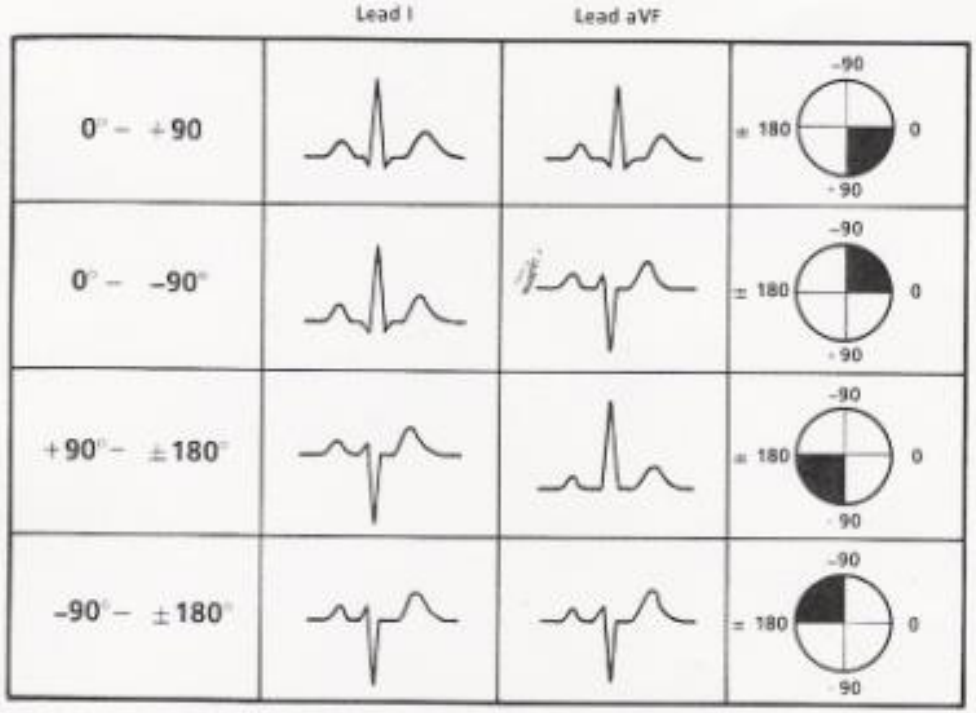


FIG. 3–10.
Locating quadrants of mean QRS axis from leads I and aVF. (From Park MK, Guntheroth WG: *How to read pediatric ECGs*, ed 3, St Louis, 1992, Mosby.)

3. Axis

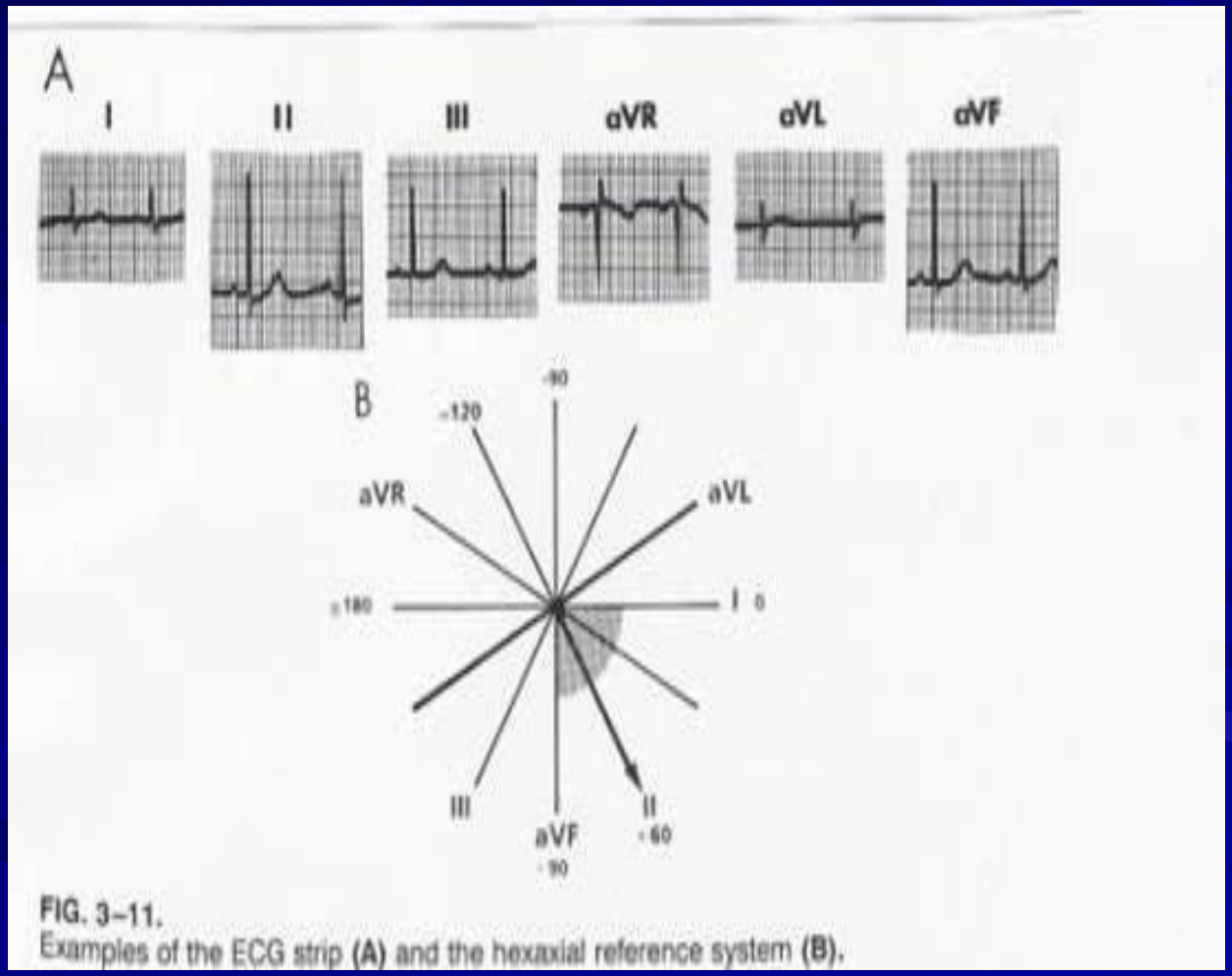


FIG. 3–11.
Examples of the ECG strip (A) and the hexaxial reference system (B).

3. QRS Axis

- RAD, LAD look at normal ranges for age

- Superior Axis

DDX:

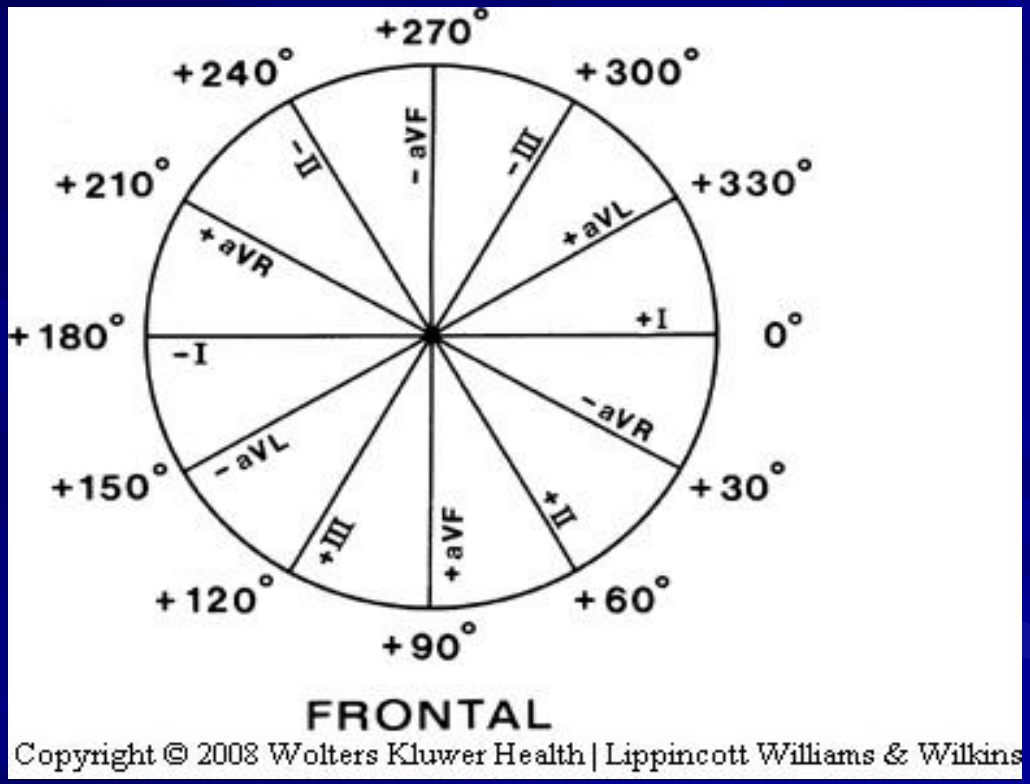
- Endocardial cushion defect
- Tricuspid atresia
- RBBB
- Overlap with LAD may occur with Left anterior hemiblock

3. QRS Axis

Successive approximation method

- Locate a quadrant
- Locate an equiphasic QRS complex, The QRS axis will be perpendicular to the lead with the equiphasic complex i.e. Lead II and aVL, and Lead III and aVR

3. AXIS

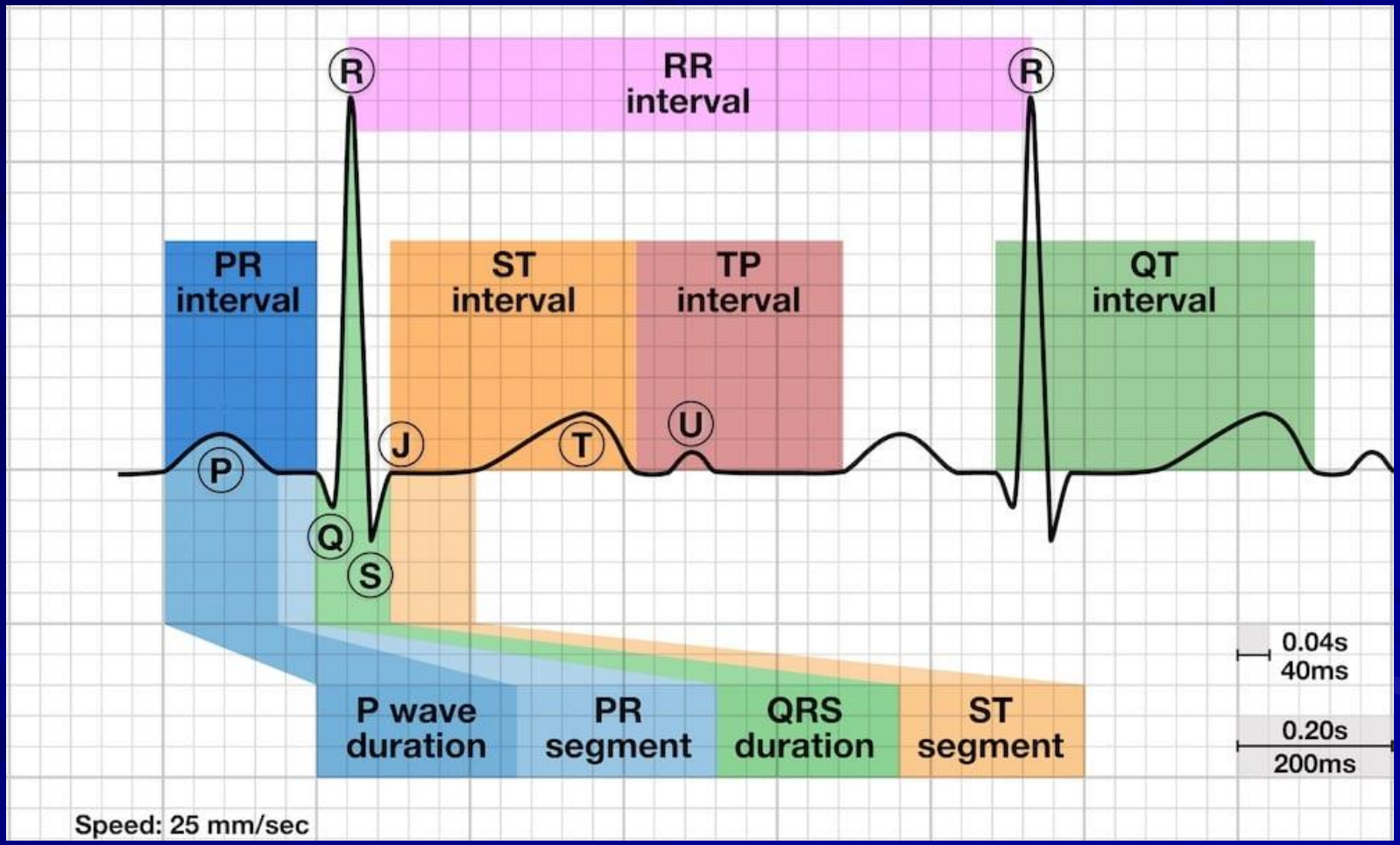


3. Axis

- T Axis
- QRS-T angle, > 60 unusual, >90 abnormal
- DDX:
- VH with strain
- V conduction disturbances
- Myocardial dysfunction of a metabolic or ischemic nature

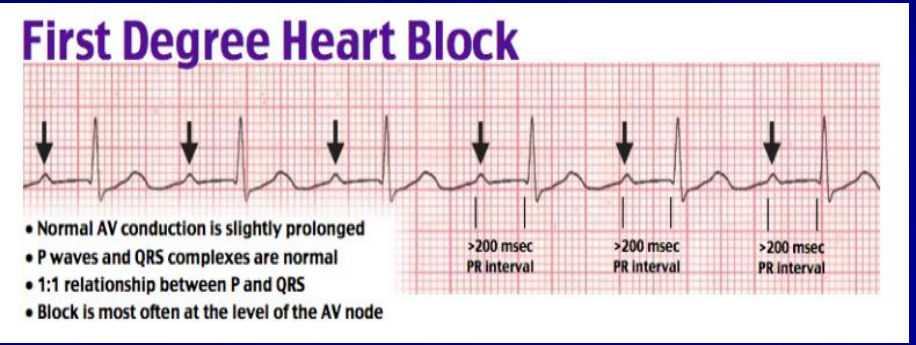
4. Intervals

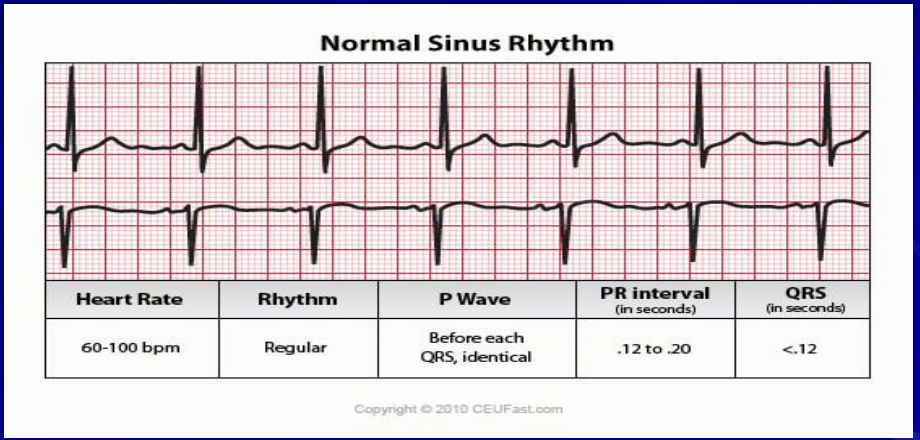
PR Interval QRS Duration and QTC Interval



PR Interval

- PR interval
- Varies with age and rate
- Increases with age and decreases with rate





4. Intervals

- Increased PR interval, DDx

- First degree AV block
- Myocarditis, rheumatic or viral
- Digitalis toxicity
- ECD, ASD, Ebsteins’s anomaly

4. Intervals

- Decreased PR interval, DDx:

- Preexcitation
- WPW
- Lown-Ganong-Levine

4. Intervals

- QRS duration
- Increases with age
- Ventricular conduction Disturbances
- Ventricular rhythms

QRS Duration

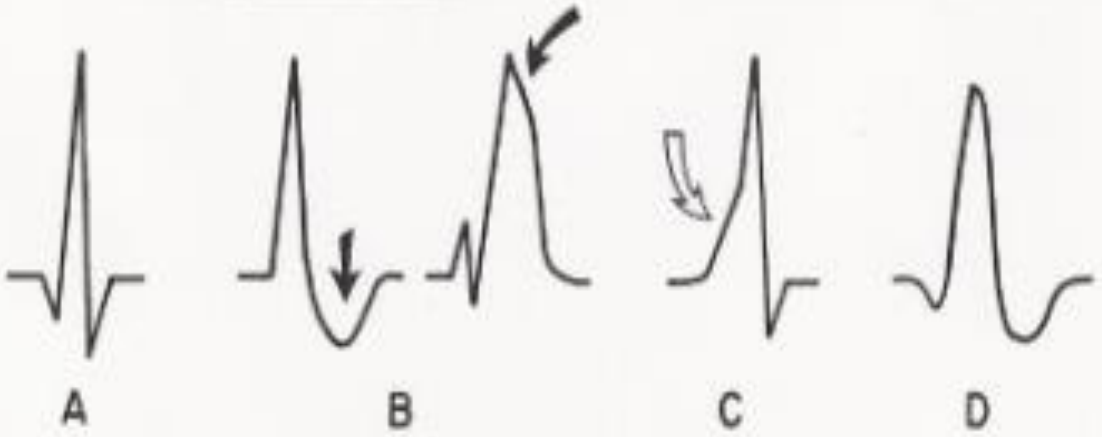


FIG. 3–18.
Schematic diagram of three types of ventricular conduction disturbances. **A,** Normal QRS complexes. **B,** QRS complex in RBBB or PVCs with prolongation of the QRS duration in the terminal portion (*black arrows*, terminal slurring). **C,** A preexcitation with delta wave (*open arrow*, initial slurring). **D,** Intraventricular block in which the prolongation of the QRS complex is throughout the duration of the QRS complex.

QRS Duration

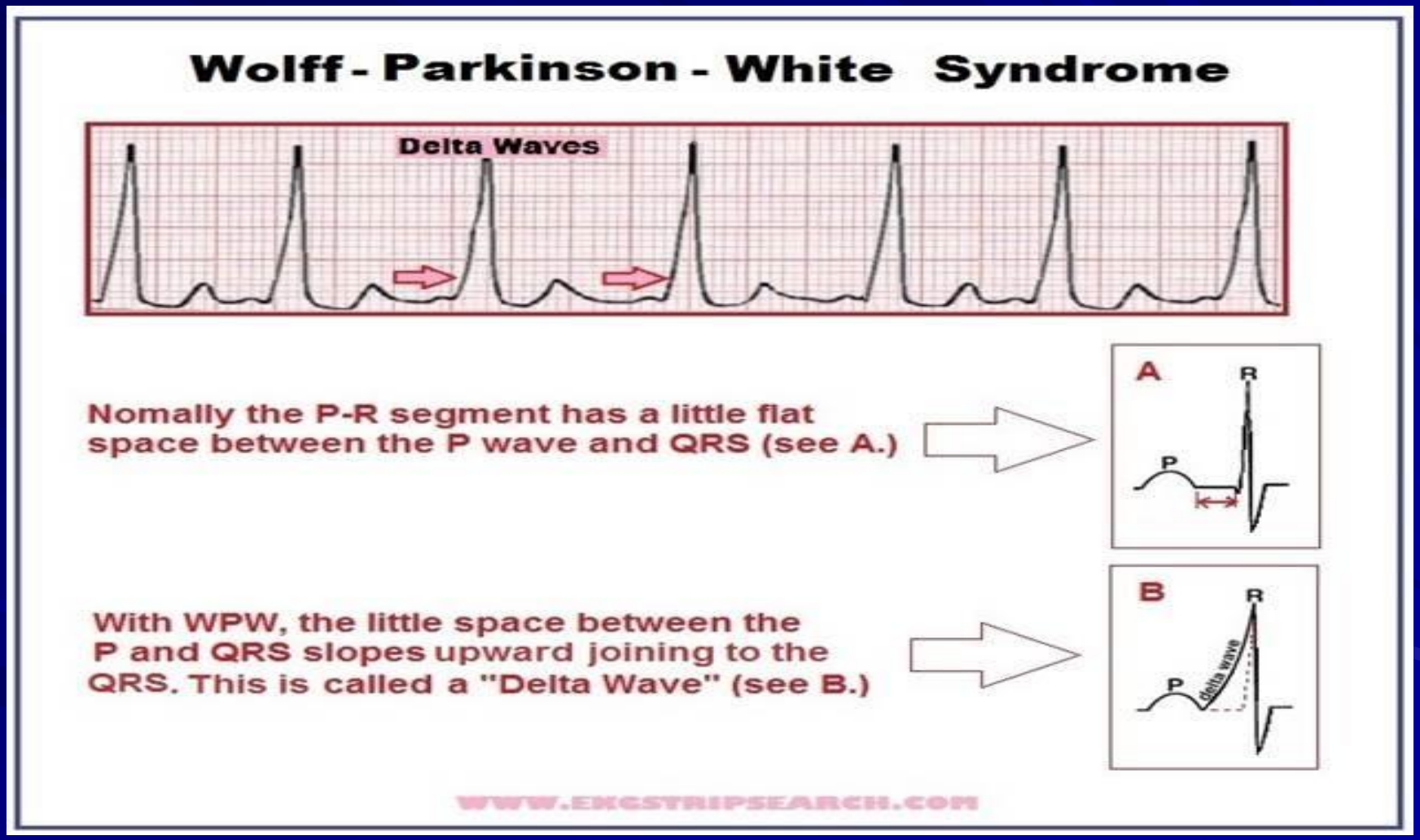
- Initial slurring: Preexcitation, WPW
- Ventricular Conduction Disturbances
- Terminal slurring: RBBB, LBBB
- Diffuse slurring: Intraventricular block

  Hyperkalemia, procainamide, quinidine, myocardial fibrosis, myocardial dysfunction of metabolic or ischemic nature

Preexcitation

- WPW
- Short PR interval < 0.1 (check tables)
- Delta waves, initial slurring of QRS
- Wide QRS duration
- May mimic VH or RBBB

Pre-excitation WPW



Ventricular Conduction Disturbances

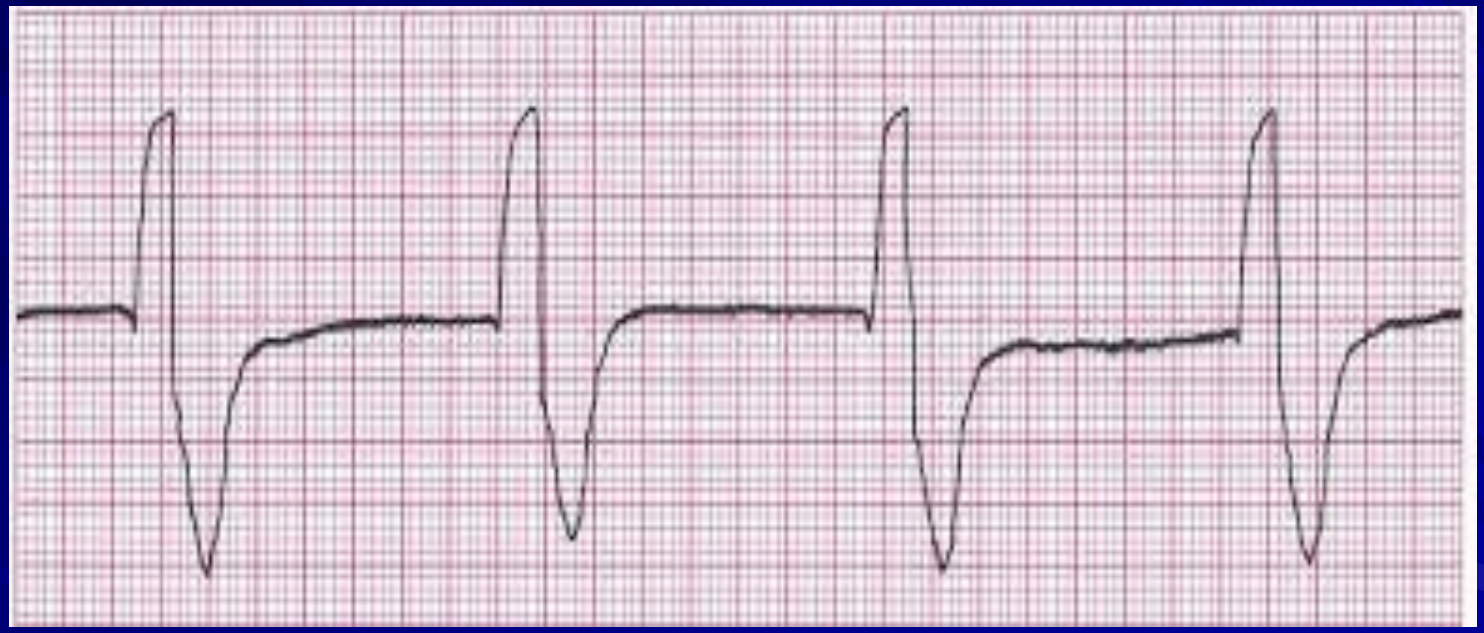
- Initial Slurring

  Preexcitation
- Diffuse Slurring

  Intraventricular block
- Terminal Slurring

  Vent rhythm, RBBB, LBBB

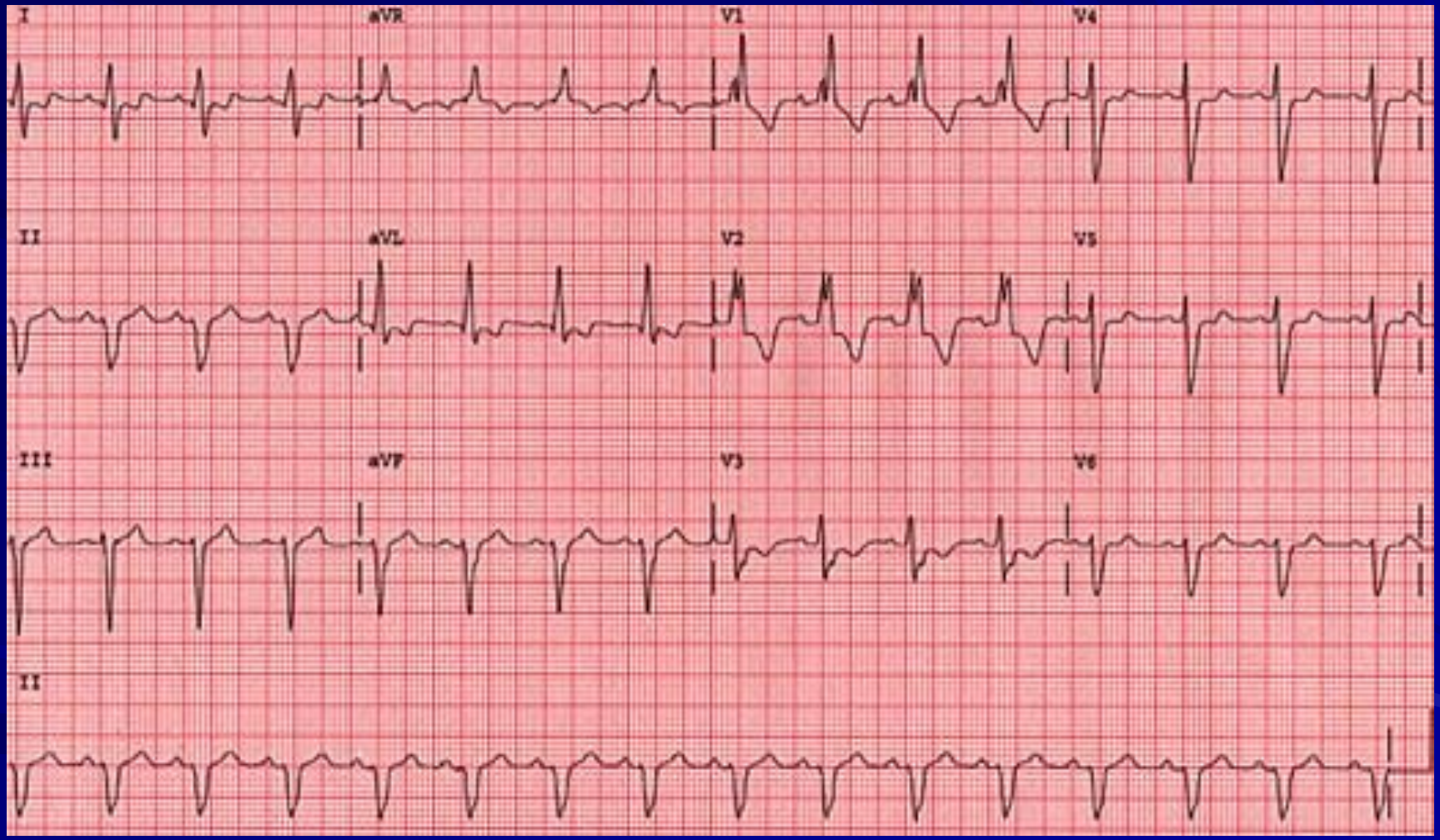
Ventricular Rhythm



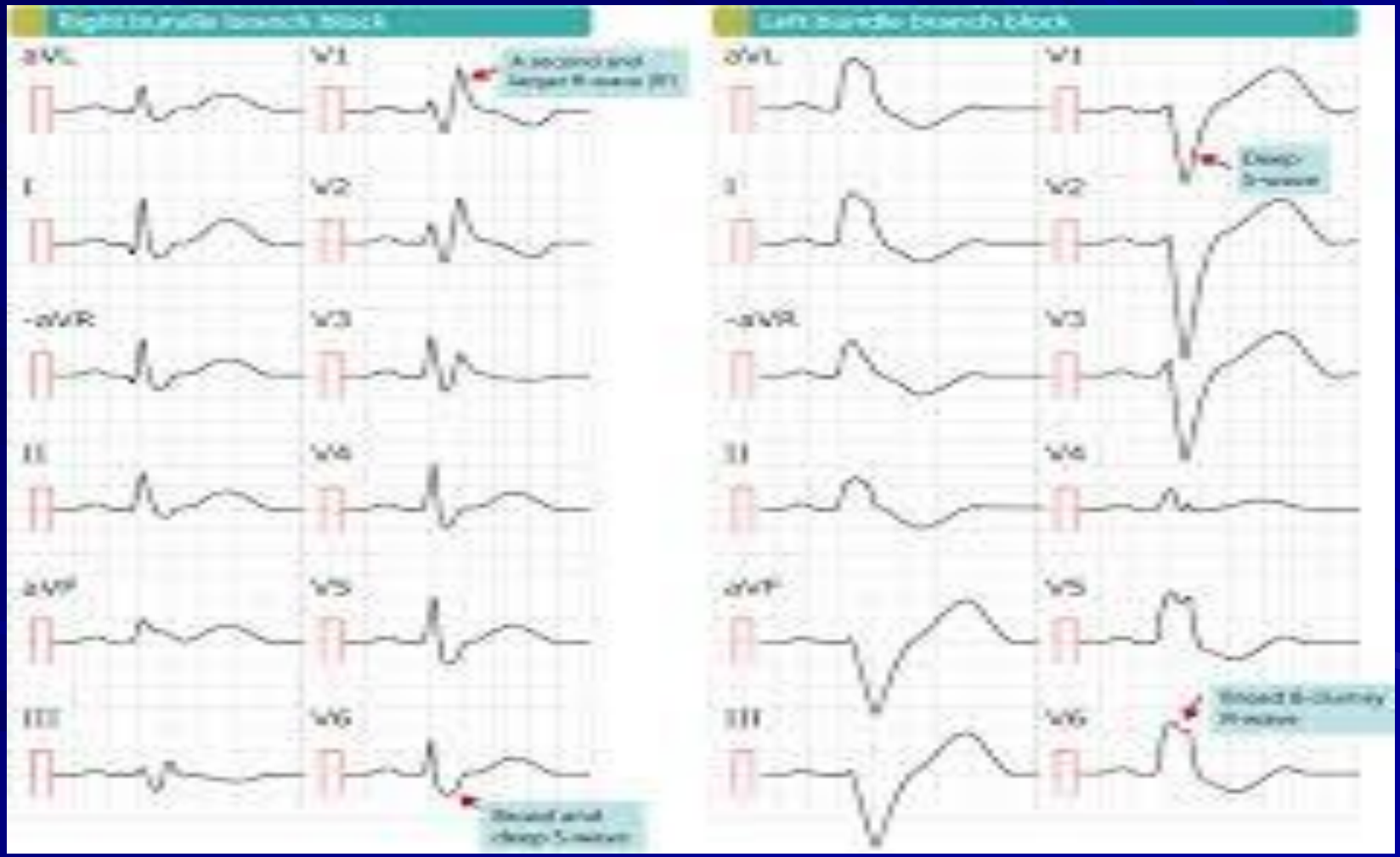
RBBB

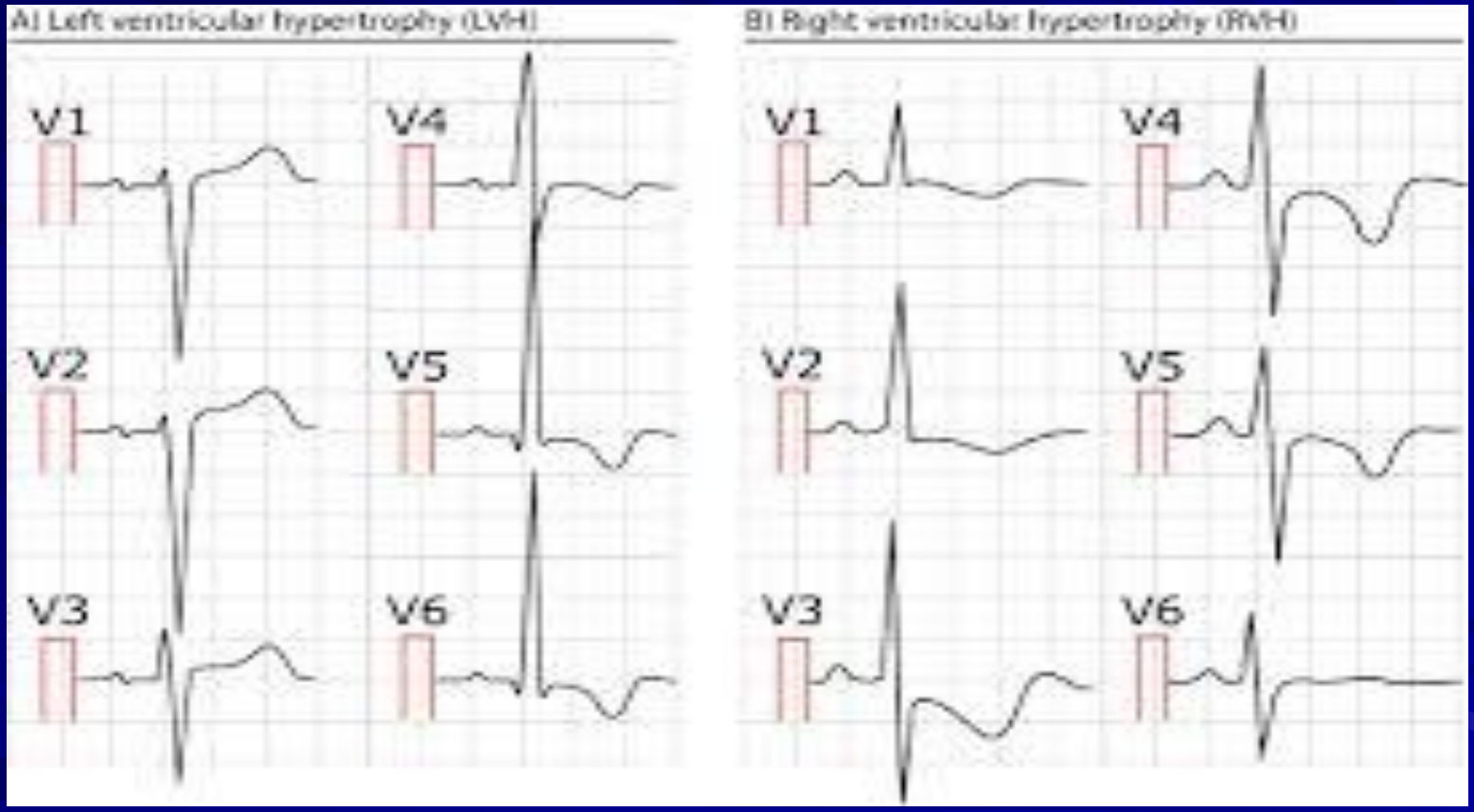
- Terminal Slurring is right and anterior
- RAD for terminal portion
- Prolonged QRS duration
- Wide slurred S in I, V5 V6
- Terminal slurred R’ in aVR, V4R, V1, V2
- T waves inversion common in adults, but not in children

RBBB



RBBB and LBBB




A) Left ventricular hypertrophy (LVH)
V1
V4
V2
V5
V3
V6
B) Right ventricular hypertrophy (RVH)
V1
V4
V2
V5
V3
V6

QRS duration

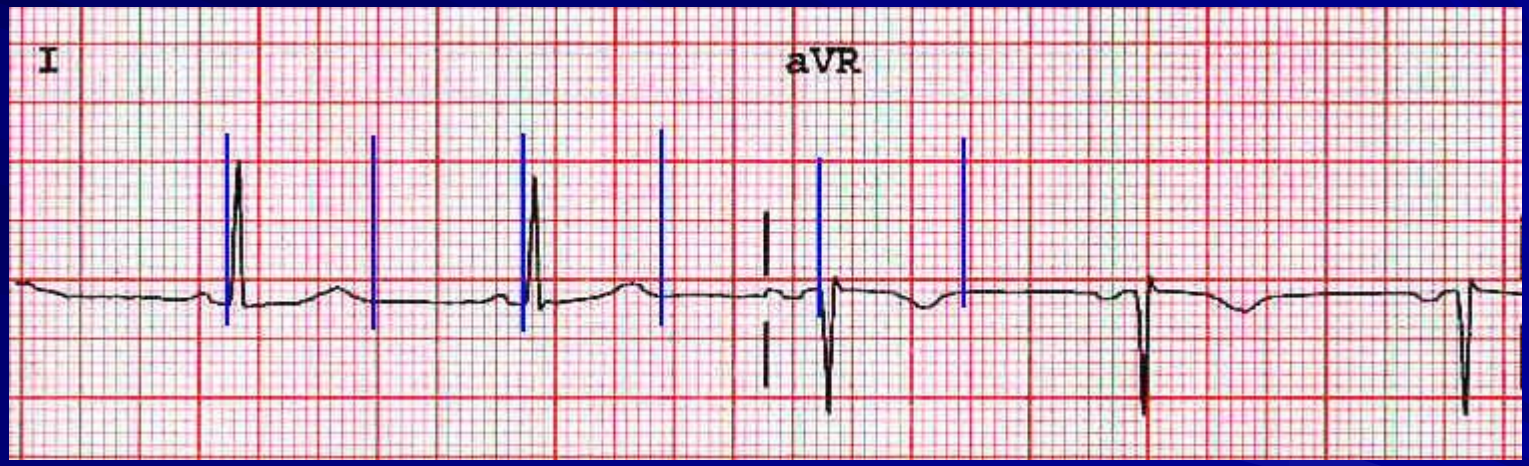
- Ventricular Rhythms:

- Premature Ventricular Conctractions
- Ventricular Tachycardia
- Implanted Ventricular Pacemaker

QTc interval

- QTc = QT/√ R-R interval
- <0.44
- <0.49 in infants

QTc



QTc Interval

- Increased QTc DDX;

Cardiac Causes:

- Myocarditis, diffuse myocardial disease (hypertrophic and dilated cardiomyopathy)
- Long QT syndrome (Jervell and Lange – Nielsen , Romano-Ward syndrome

Prolonged QTc Interval

None cardiac causes:

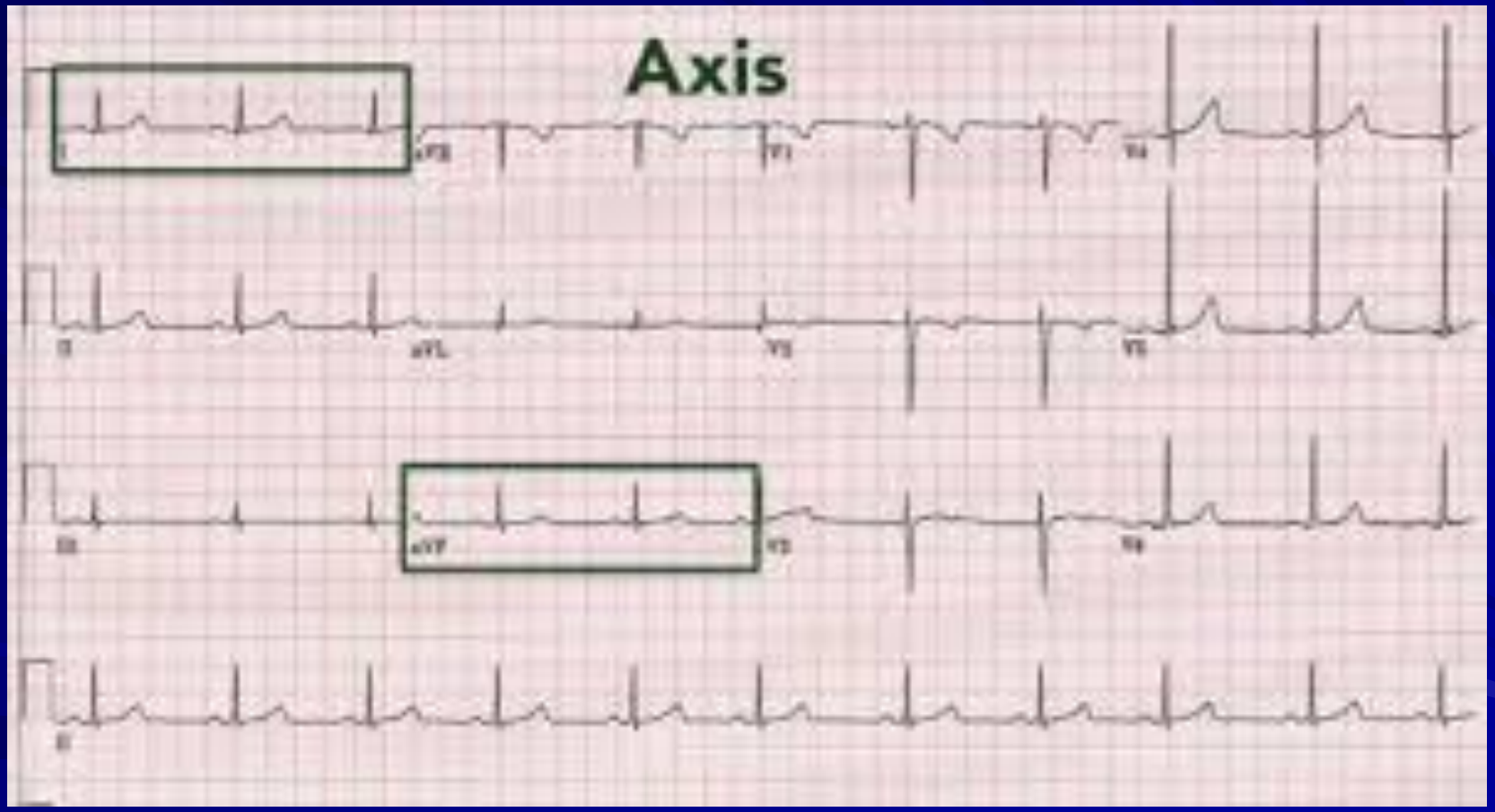
- Hypocalcemia, head trauma, malnutrition
- Drugs;

  Antibeotics (Amp, Em,TMP-Sulfa, Amantadine) Anti psychotic (phenothiazines) Anti depressants (tricyclic) Anti histamines (Seldane) and Anti arrhythmic drugs, arsenics, organophosphates

Decreased QT

- Digitalis effect
- Hypercalcemia

5. Forces and R-R Progression



Hypertrophy

- RPL V3R, V4R, V1
- LPL V5, V6, V7
- BVL V2-V4

- QRS Axis:

  Directed towards the hypertrophied lead, more seen with RVH

Hypertrophy

- QRS voltage:
- Increases in the direction of the respective ventricle.
- Normal QRS duration
- LVH increased R voltages in Leads I, II, aVL, sometimes aVF and III, tall R's in V5-7 with deep S's in V1-2 and V3R and V4R

Hypertrophy

- RVH increased R in aVR and III, and deep S in lead I, increased R in V1-2, V3R and V4R and deep S ‘s in V5-6

Hypertrophy

- R/S ratio
- Maybe just change in ratio without change in absolute voltages.
- R/S ratio increase in RPL and decrease in LPL = RVH
- R/S ratio increase in LPL and decrease in RPL = LVH

Hypertrophy

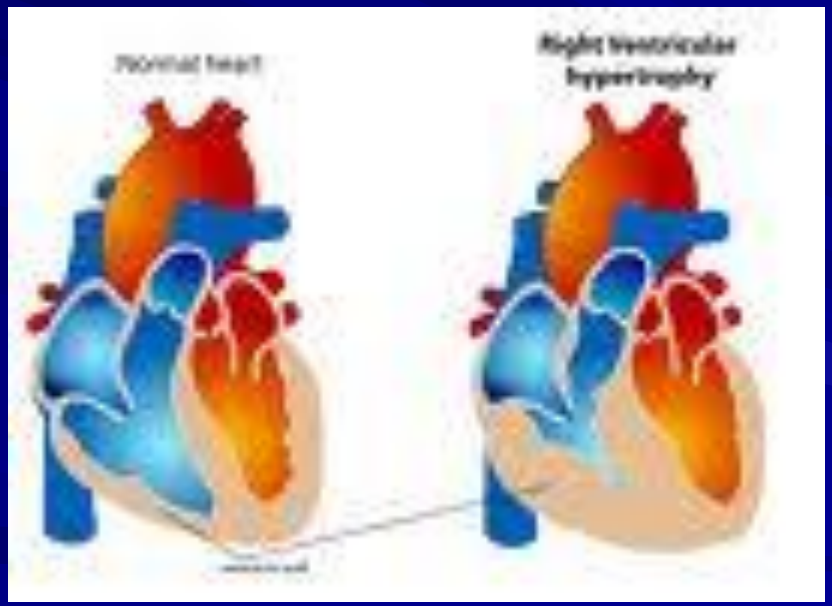
- Changes in T Axis
- Abnormal T axis with increased QRS-T angle = strain
- Upright T waves in RPL after day 3 of life and up to adolescence = strain
- Inverted T waves in LPL = strain

Hypertrophy

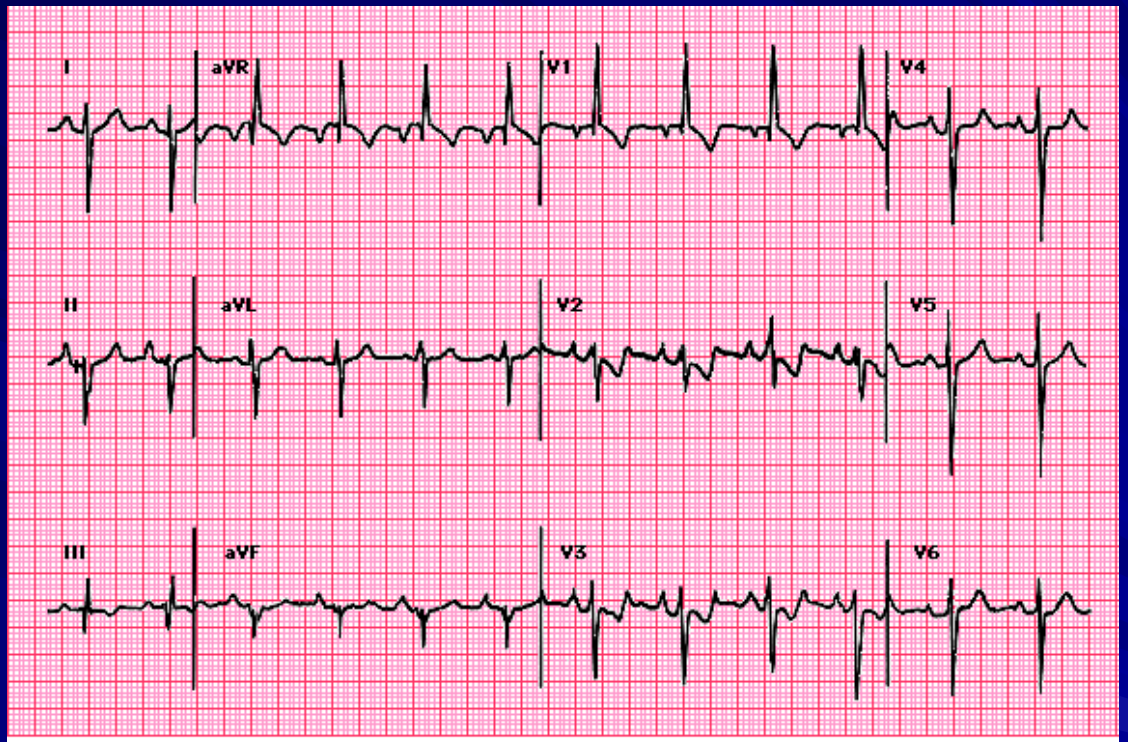
- Q waves
- Abnormal Q waves are either deep or wide or both.
- Q waves are normally present in LPL and absent in RPL
- Deep and wide Q's are present in myocardial incfarction
- Deep Q's are present in volume overload VH
- Presence of Q's in RPL (RVH or V inversion) Absence in LPL (LBBB or V inversion)

RVH

- RVH



RVH



Right ventricular hypertrophy Right ventricular hypertrophy due, in this case, to primary pulmonary hypertension. The characteristic features include marked right axis deviation (+210° which is equal to −150°), tall R wave in V1 (as part of a qR complex), delayed precordial transition zone with prominent S waves in leads V5 and V6, inverted T waves and ST depression in V1 to V3 consistent with right ventricular "strain", and peaked P waves in lead II consistent with concomitant right atrial enlargement. Courtesy of Ary Goldberger, MD.

RVH

RAD

Normal QRS duration and increased voltages in RPL, tall R in V1-2, aVR and deep S's in lead I, V6

R/S ratio increased in RPL, decreased in LPL

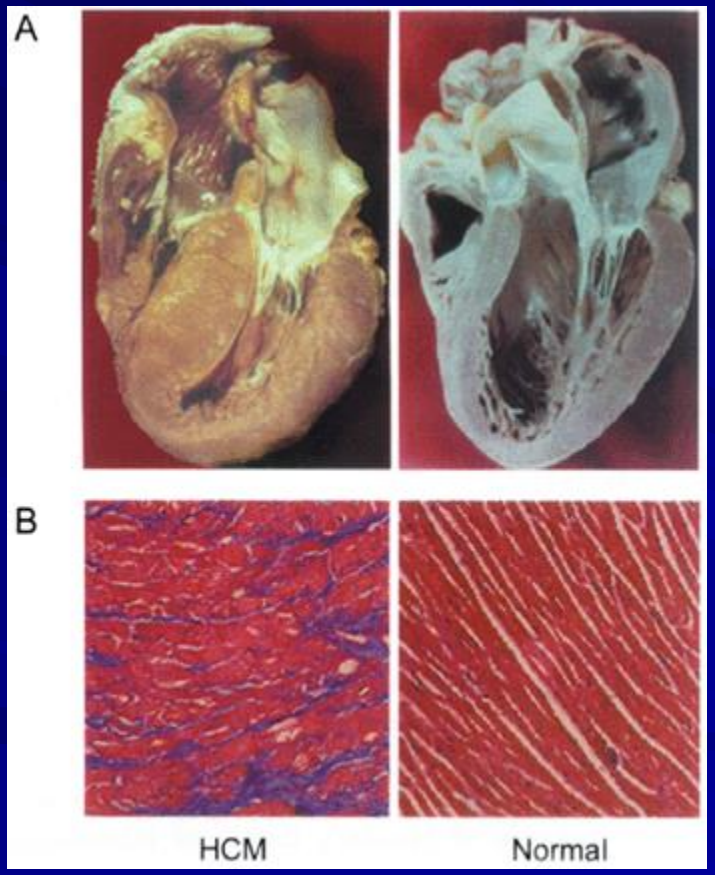
q in V1 (qR or qRs)

rsR', or rR' where R' is always > R

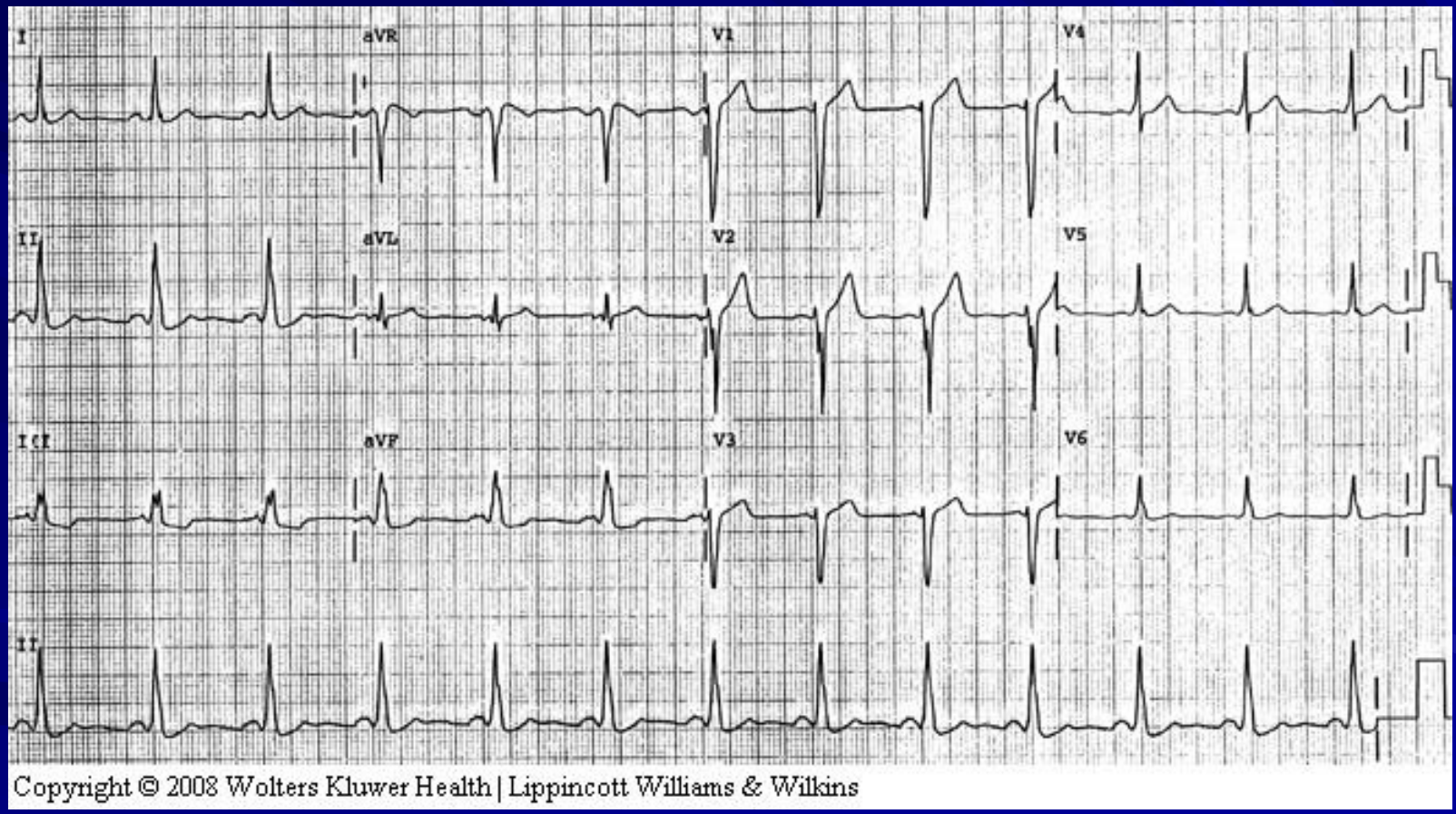
T waves upright in RPL with strain

LVH

LVH



LVH



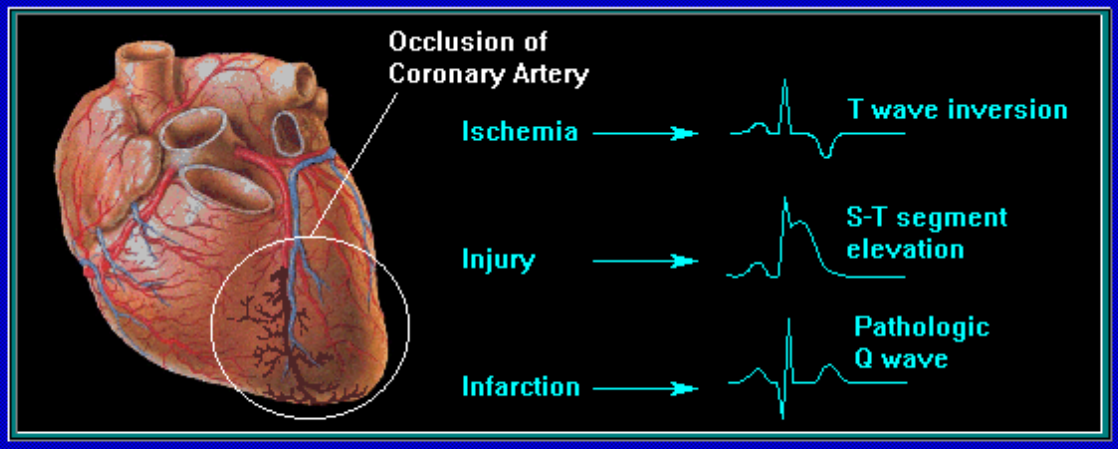
LVH

- LAD
- Tall R in lead I, II, aVL, aVF, V5, V6
- Deep S's in V1, V2
- R/S increased in LPL and decreased in RPL
- Deep Q in V5, V6 > 5 mm, and tall symmetric T waves (LV diastolic overload)
- Inverted T waves in lead I, aVF and V5-6 = strain

CVH

- Presence of RVH and LVH criteria
- Positive criteria for RVH or LVH and large voltages for the other
- Large equiphasic QRS complexes in 2 or more limb leads and the mid precordial leads

Ischemia



ECG CHANGES IN ACUTE MYOCARDIAL ISCHEMIA, INJURY, AND INFARCTION

Typically, three phenomena may occur on the ECG that are characteristic of the evolution of a myocardial infarction (MI):

1. T wave inversion, indicating ischemia.
2. S-T segment elevation, indicating injury and the acuteness of the MI.
3. The presence of an abnormal Q wave, indicating tissue death (necrosis).

The above abnormalities are usually seen in the ECG leads representing the area of damage.

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ST Segments

- Up to 1mm elevation or depression is acceptable in children
- Examples of nonpathologic ST segment shift: Early repolarization, J point depression

ST Segment

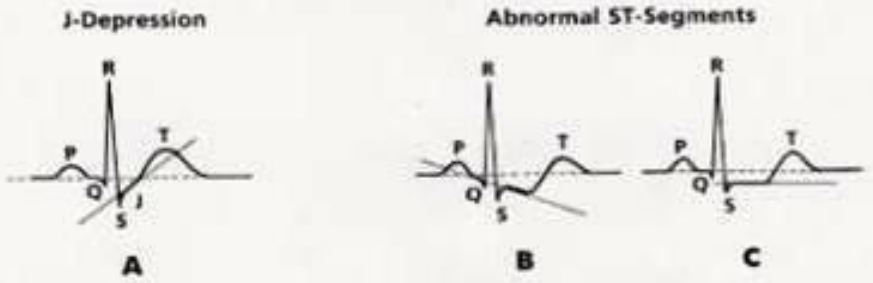


FIG. 27–1.
Nonpathologic (nonischemic) and pathologic (ischemic) ST-segment and T-wave changes. **A,** characteristic nonischemic ST-segment change called *J-depression;* note that the ST slope is upward. **B** and **C,** Examples of pathologic ST-segment changes; note that the downward slope of the ST-segment **(B)** or the horizontal segment is sustained **(C).** (From Park MK, Guntheroth WG: *How to read pediatric ECGs,* ed 3, St Louis, 1992, Mosby.)

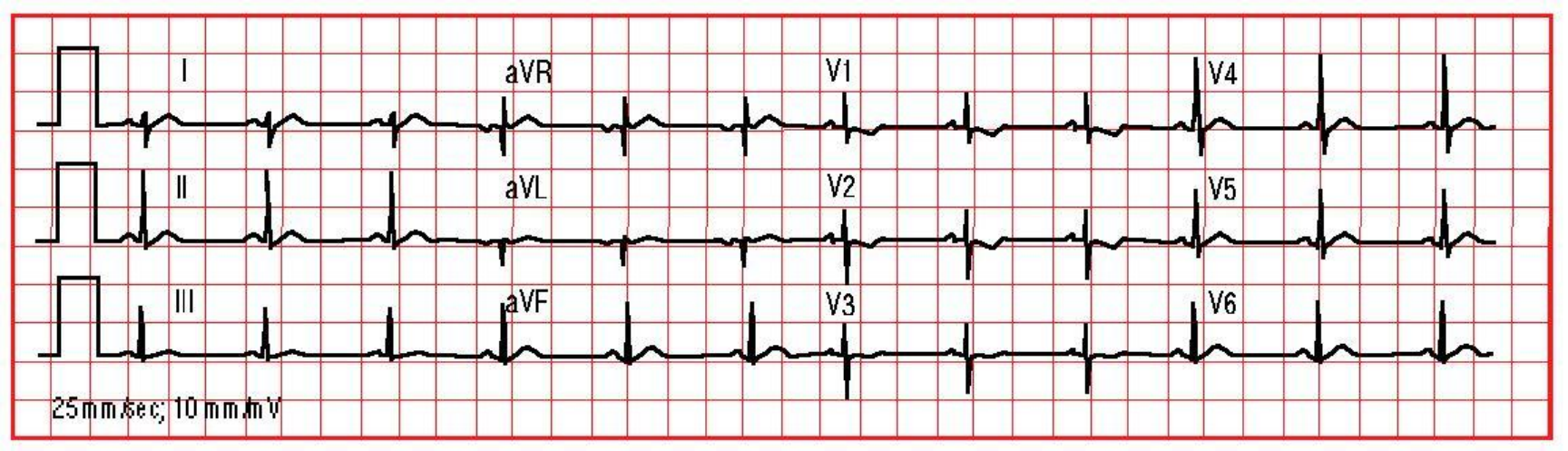
St Segment

- Pathologic depression;
- Downward slant with a diphasic or inverted T wave
- Horizontal elevation or depression sustained for over 0.08 seconds
- ST depression; hypertrophy, strain, ischemia, digoxin effect
- ST elevation; pericarditis, injury

T waves

- < ½ of QRS
- Positive in I, II, aVL, V4-6
- Negative in aVR, V3R, V1-2
- Abnormal inverted T waves: ischemia, hypertrophy and hyperventilation
- Flattened T waves: hypokalemia
- Peaked T waves : hyperkalemia, ventricular hypertrophy or BBB

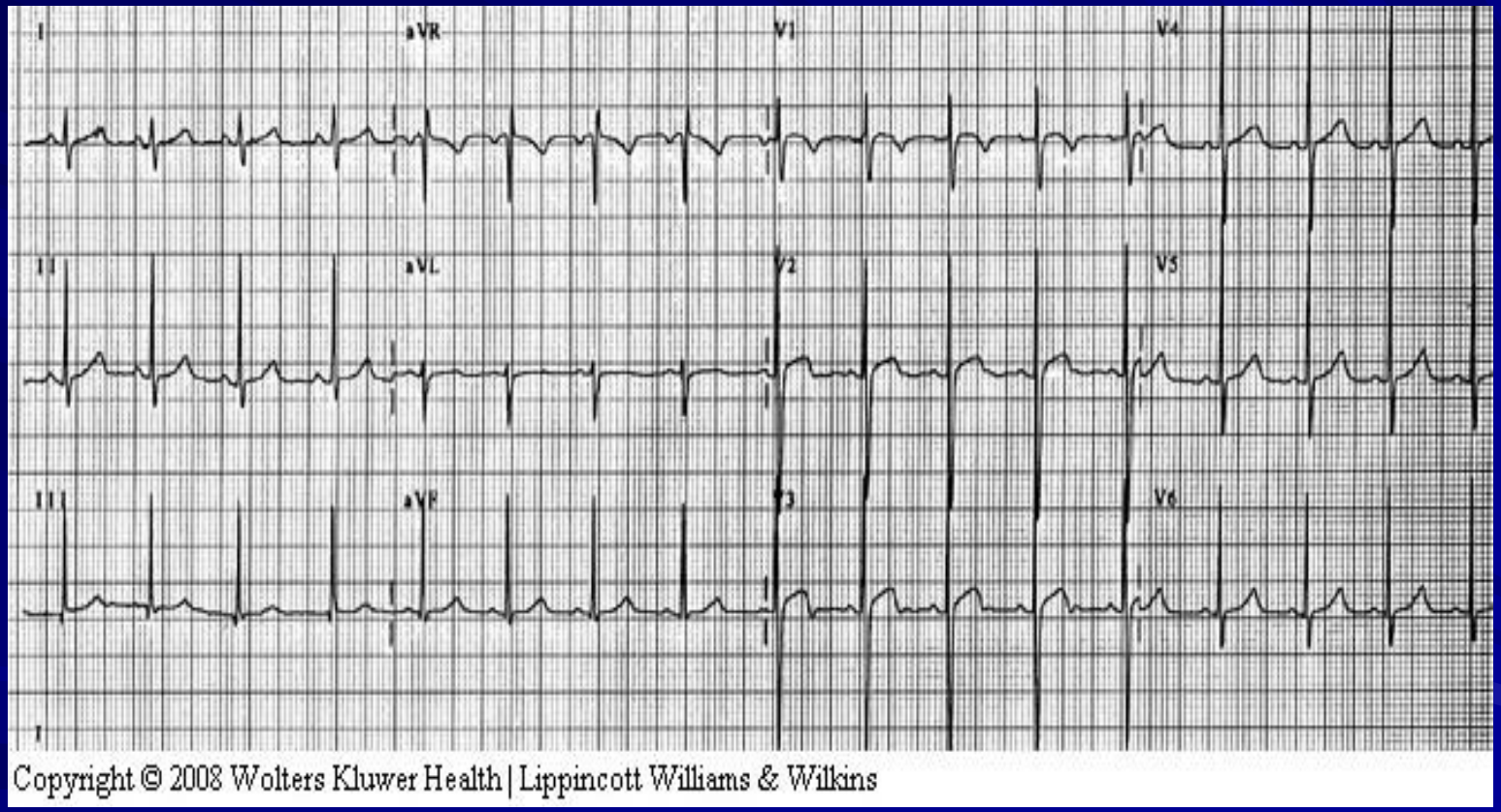
Normal Infant ECG

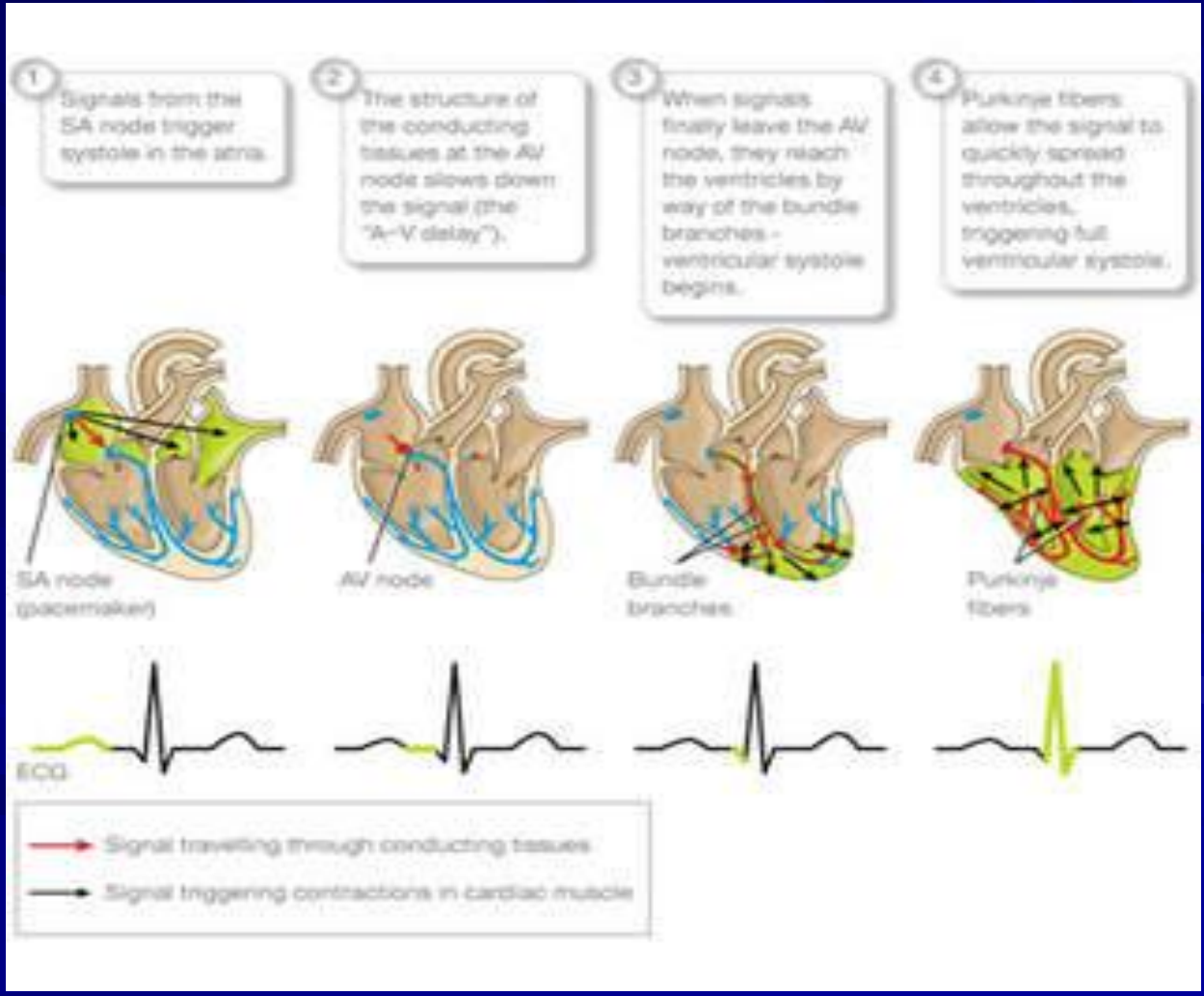


New born RVH

- QRS Axis> 180
- R waves in aVR> 8mm, pure R in V1>10 mm, and R wave in V1> 25 mm
- S wave in lead I> 12 mm
- qR pattern in V1
- Upright T waves in RPL

Early Repolarization




1
Signals from the SA node trigger systole in the atria.
2
The structure of the conducting tissues at the AV node slows down the signal (the "A–V delay").
3
When signals finally leave the AV node, they reach the ventricles by way of the bundle branches - ventricular systole begins.
4
Purkinje fibers allow the signal to quickly spread throughout the ventricles, triggering full ventricular systole.
SA node (pacemaker)
AV node
Bundle branches
Purkinje fibers
ECG
Signal traveling through conducting tissues
Signal triggering contractions in cardiac muscle