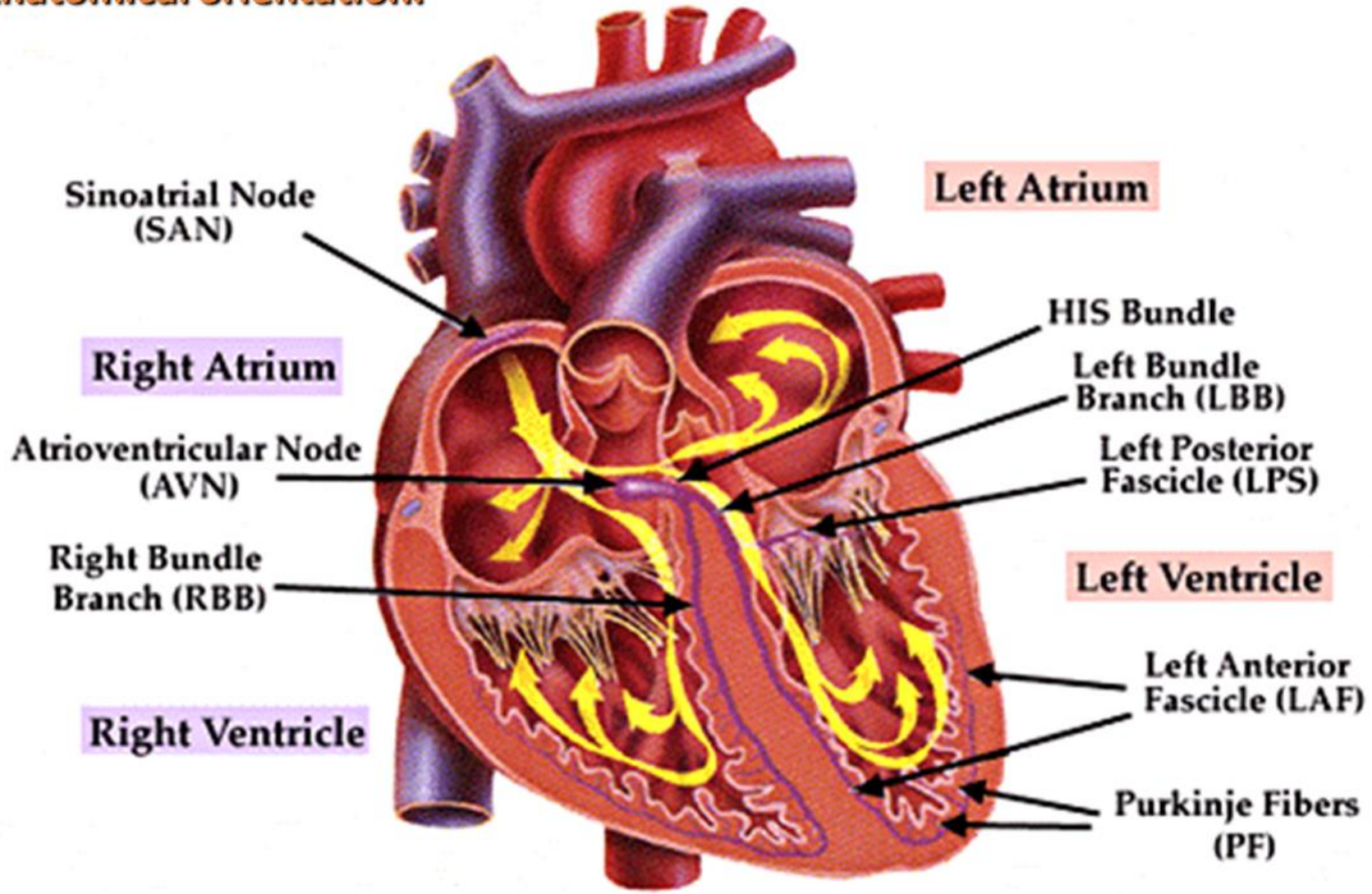
Introduction To ECG.

Anatomical orientation:



Action Potential

Phase 0 rapid depolarisation (inflow of Na^{+})

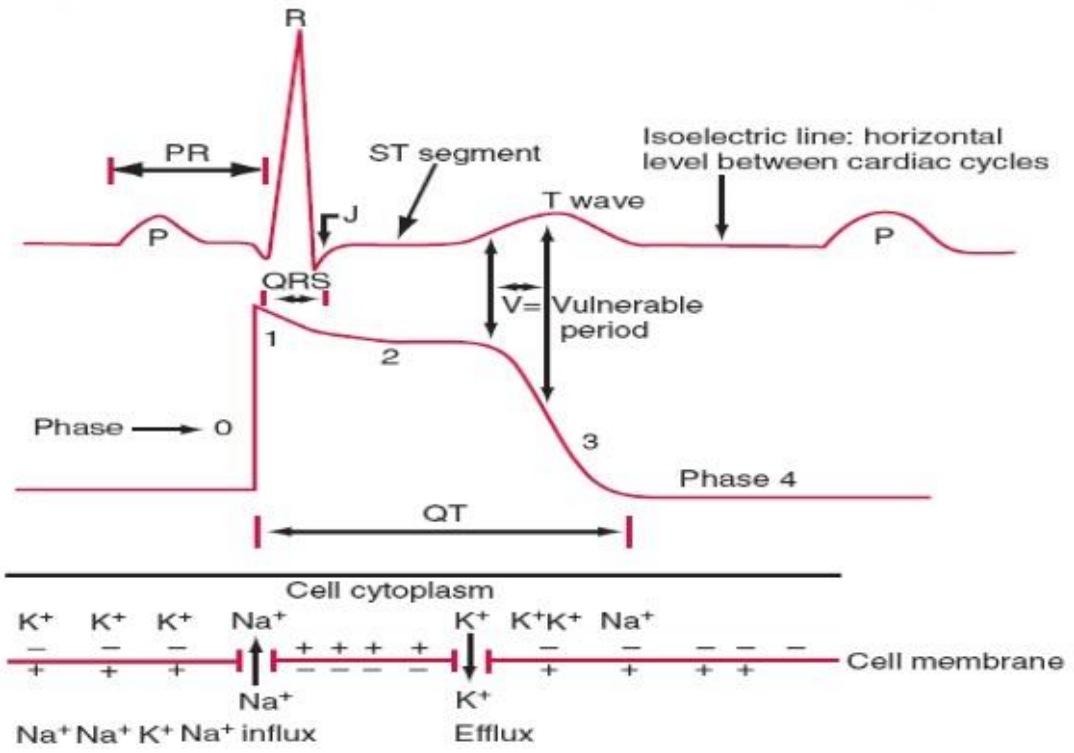
Phase 1 partial repolarisation (inward Na^{+} current deactivated, outflow of K^{+})

Phase 2 plateau (slow inward calcium current)

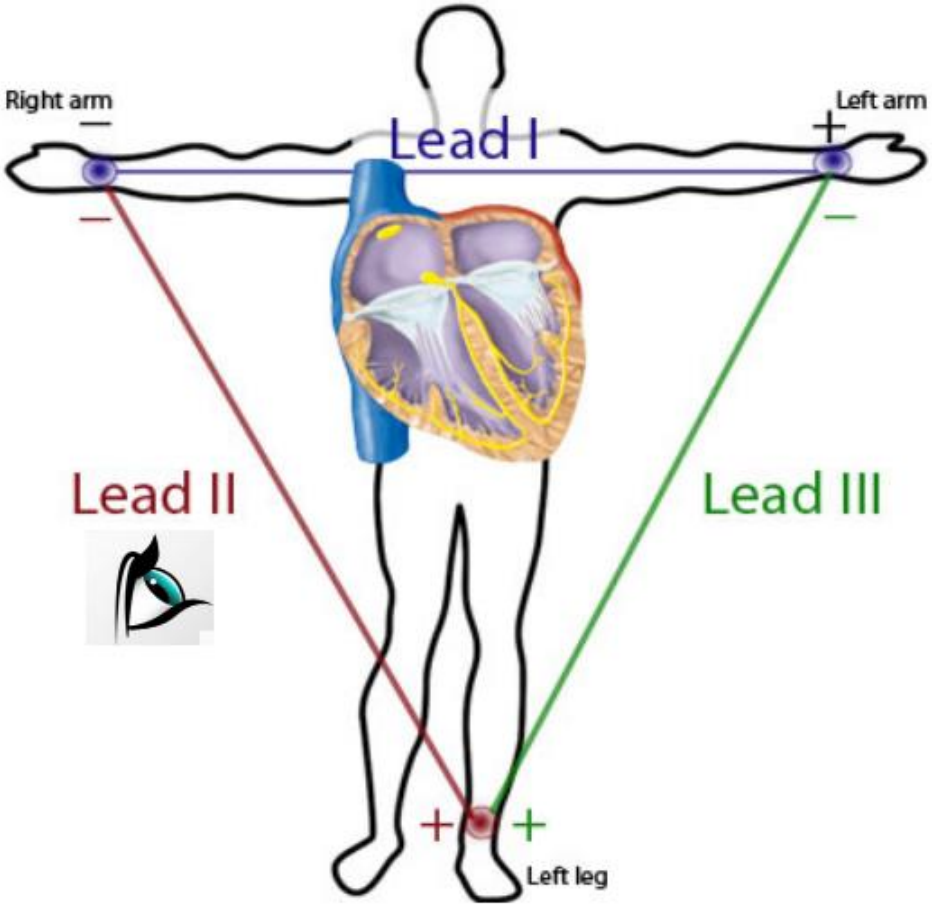
Phase 3 repolarisation (calcium current inactivates, K^{+} outflow)

Phase 4 pacemaker potential (Slow Na^{+} inflow, slowing of K^{+} outflow) 'autorhythmicity'

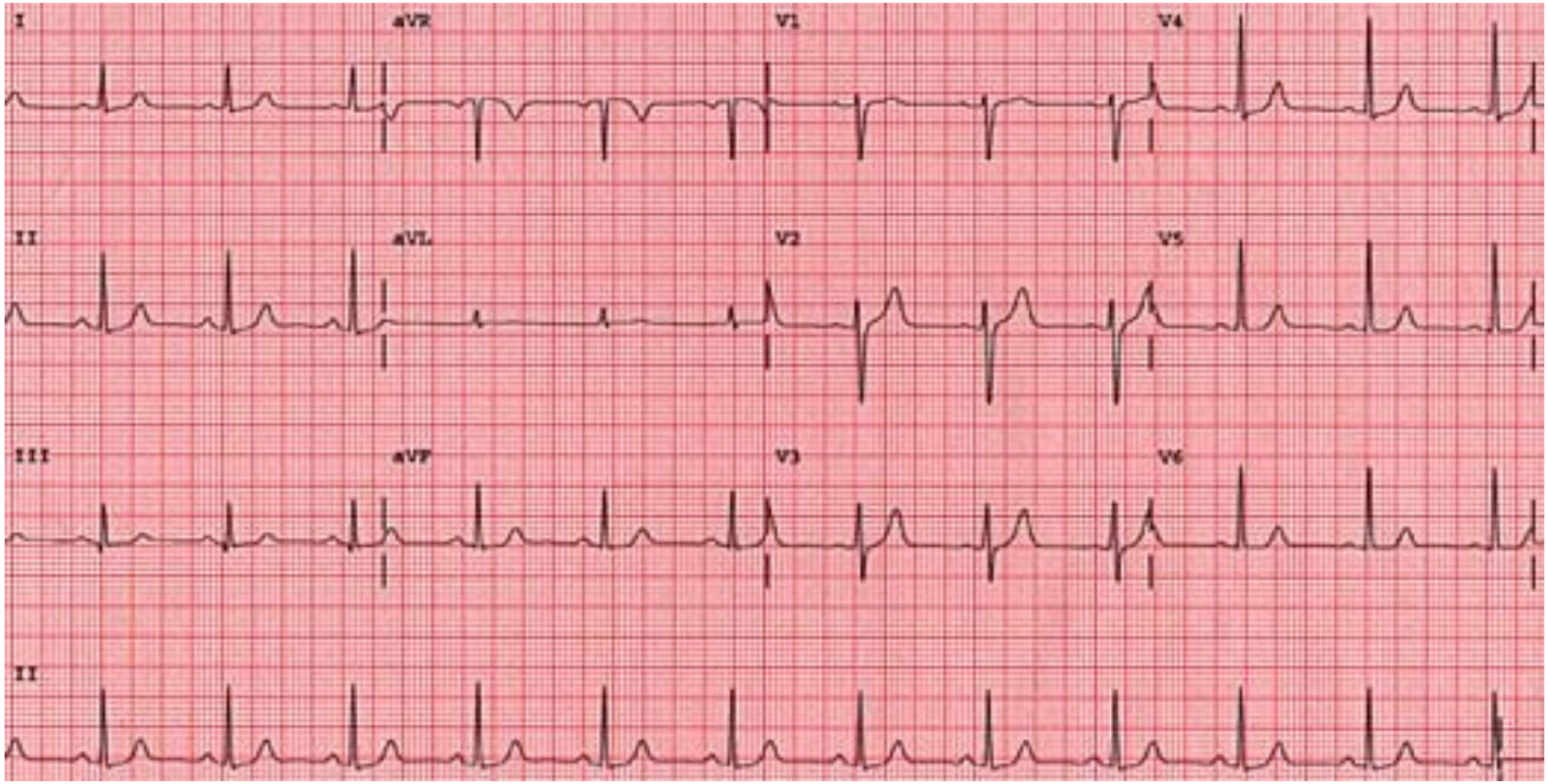
Refractory period (phases 1-3)

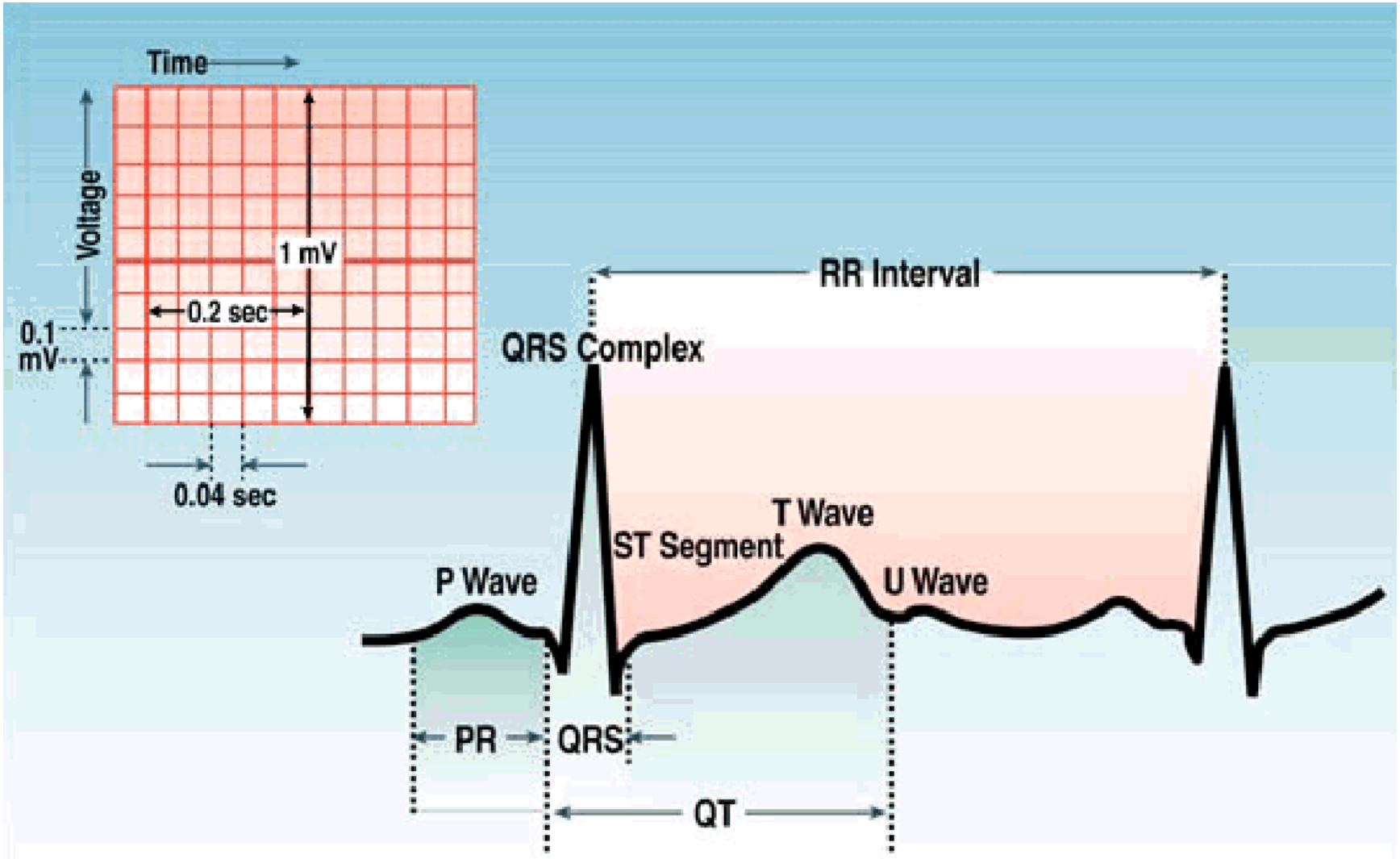


Einthoven's Triangle



Normal ECG




Time
Voltage
1 mV
0.2 sec
0.1 mV
0.04 sec
RR Interval
QRS Complex
T Wave
ST Segment
P Wave
U Wave
PR
QRS
QT

PR Interval

(Measured from beginning of P to beginning of QRS in the frontal plane)

Normal: 0.12 - 0.20s

Short PR: < 0.12s

Preexcitation syndromes: *WPW (Wolff-Parkinson-White) Syndrome: An accessory pathway (called the "Kent" bundle) connects the right atrium to the right ventricle or the left atrium to the left ventricle, and this permits early activation of the ventricles (delta wave) and a short PR interval.*

AV Junctional Rhythms *With retrograde atrial activation (inverted P waves in II, III, aVF): Retrograde P waves may occur before the QRS complex (usually with a short PR interval), in the QRS complex (i.e., hidden from view), or after the QRS complex (i.e., in the ST segment).*

Ectopic atrial rhythms *originating near the AV node (the PR interval is short because atrial activation originates close to the AV node; the P wave morphology is different from the sinus*

First degree AV block (PR interval usually constant)

Prolonged PR: >0.20s

Intra-atrial conduction delay (uncommon)

Slowed conduction in AV node (most common site)

Slowed conduction in His bundle (rare)

Slowed conduction in bundle branch (when contra lateral bundle is blocked)

Second degree AV block (PR interval may be normal or prolonged;

Some P waves do not conduct)

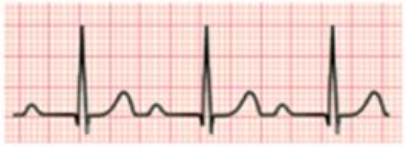
Type I (Wenckebach): Increasing PR until nonconducted P wave occurs

Type II (Mobitz): Fixed PR intervals plus nonconducted P waves

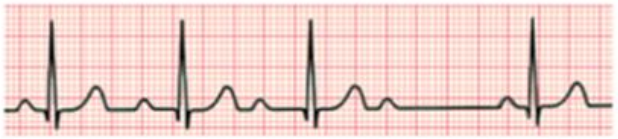
Third degree AV block (Complete dissociation)

AV dissociation: Some PR's may appear prolonged, but the P waves and QRS complexes are dissociated

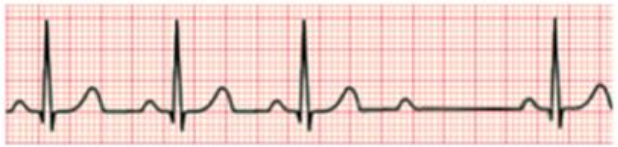
First degree AV block



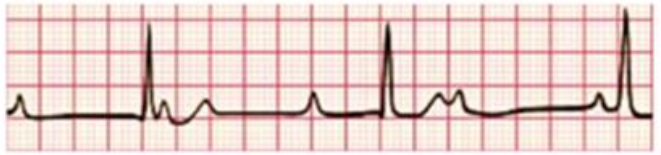
Second degree AV block (Mobitz I or Wenckebach)



Second degree AV block (Mobitz II)

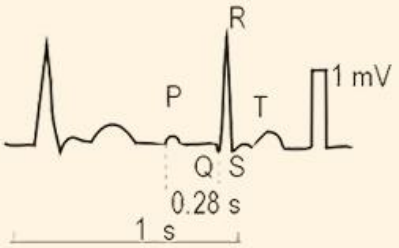


Third degree AV block with junctional escape

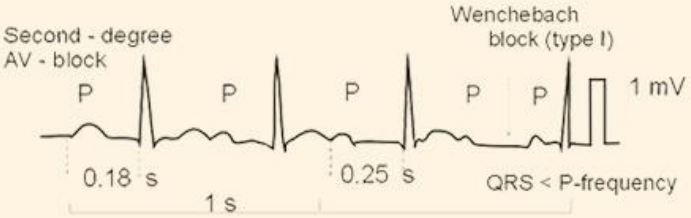


The Heart Block Poem

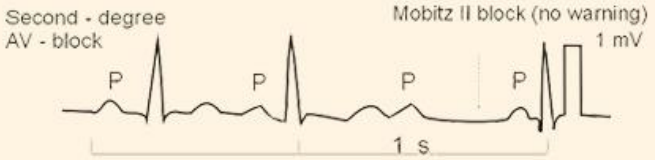
If the **R** is far from **P**,
then you have a **FIRST DEGREE**.



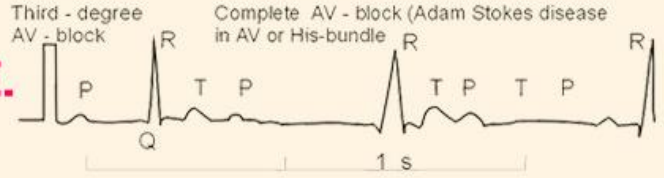
Longer, longer, longer, drop!
Then you have a **WENKEBACH**.



If some **P**s don't get through,
then you have **MOBITZ II.**



If **P**s and **Q**s don't agree,
then you have a **THIRD DEGREE.**



QRS Duration

(Duration of QRS complex in frontal plane):

Normal: 0.06 - 0.10s

Prolonged QRS Duration (>0.10s): QRS duration 0.10 - 0.12s

Incomplete right or left bundle branch block

Non-specific intraventricular conduction delay (IVCD)

Some cases of left anterior or posterior fascicular block

QRS duration > 0.12s

Complete RBBB or LBBB

Non-specific IVCD

Ectopic rhythms originating in the ventricles (e.g., ventricular tachycardia, pacemaker rhythm)

QT Interval

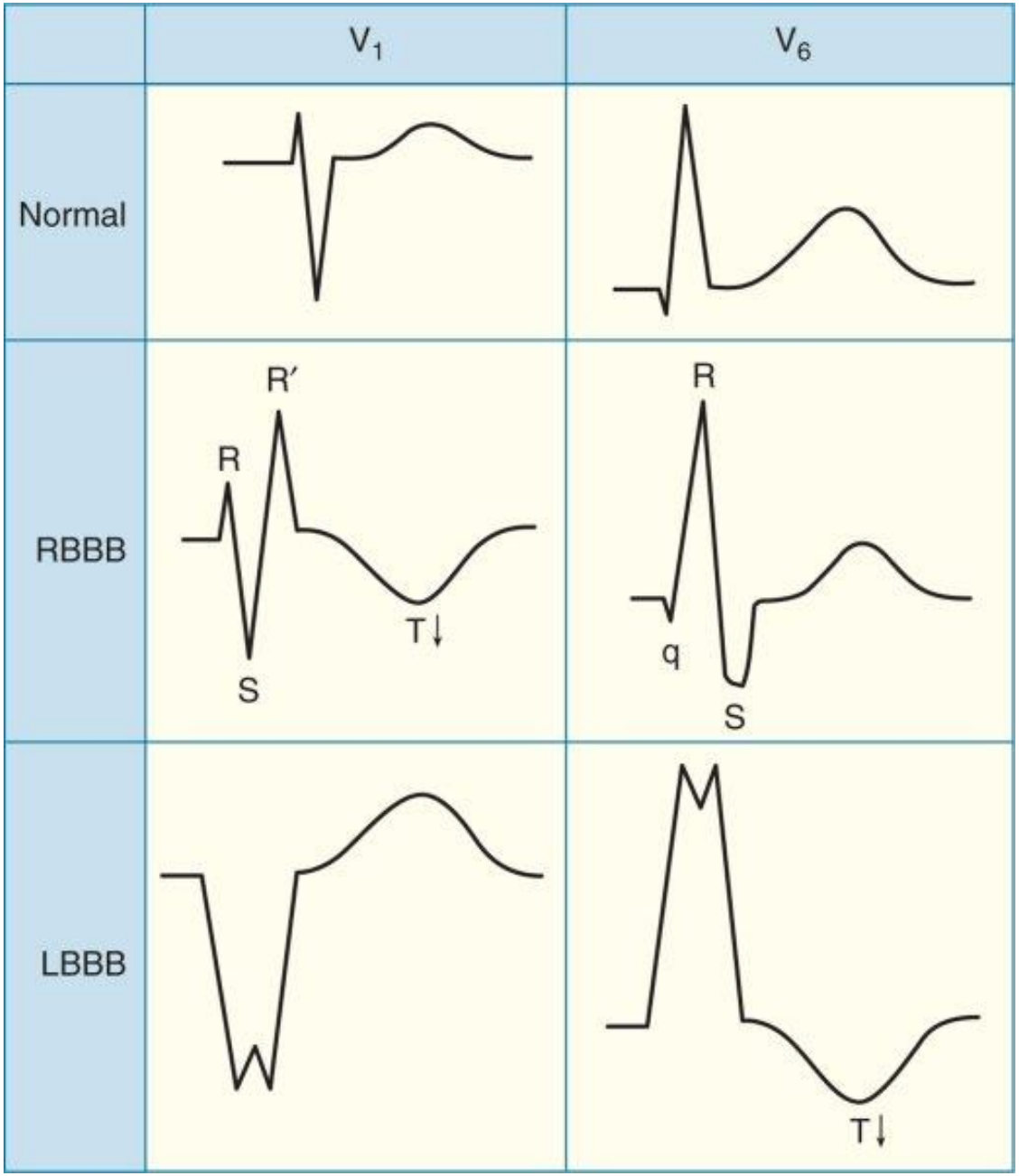
(Measured from beginning of QRS to end of T wave in the frontal plane)

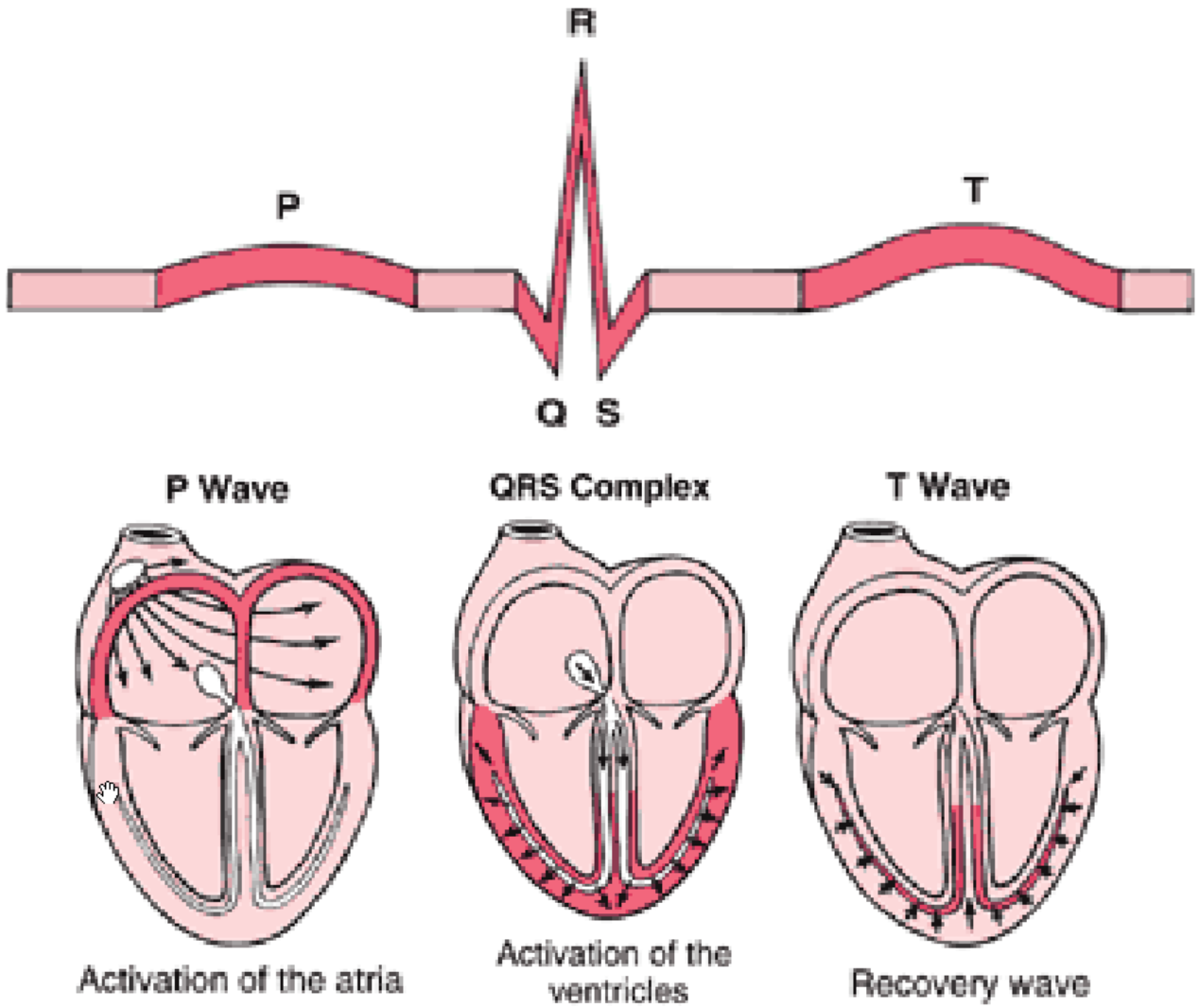
Normal: heart rate dependent

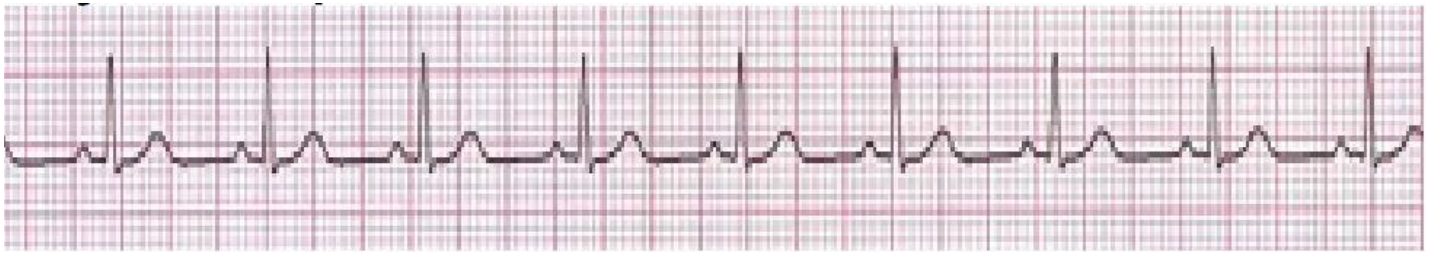
Long QT Syndrome - "LQTS" *(based on upper limits for heart rate) This abnormality may have important clinical implications since it usually indicates a state of increased vulnerability to malignant ventricular arrhythmias, syncope, and sudden death. The prototype arrhythmia of the Long QT Interval Syndromes (LQTS) is Torsade-de-pointes, a polymorphic ventricular tachycardia characterized by varying QRS morphology and amplitude around the isoelectric baseline.*

Causes of LQTS include the following:

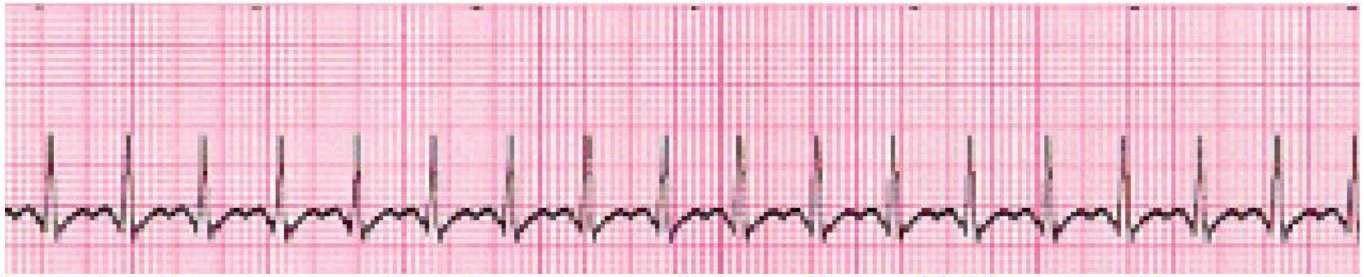
- *Drugs (many antiarrhythmics, tricyclics, phenothiazines, and others)*
- *Electrolyte abnormalities (↓K+, ↓Ca++, ↓Mg++)*
- *CNS disease (especially subarrachnoid haemorrhage, stroke, trauma)*
- *Hereditary LQTS (e.g., Romano-Ward Syndrome)*
- *Coronary Heart Disease (some post-MI patients)*


V1
V6
Normal
RBBB
R′
R
S
T↓
R
q
S
T↓
LBBB

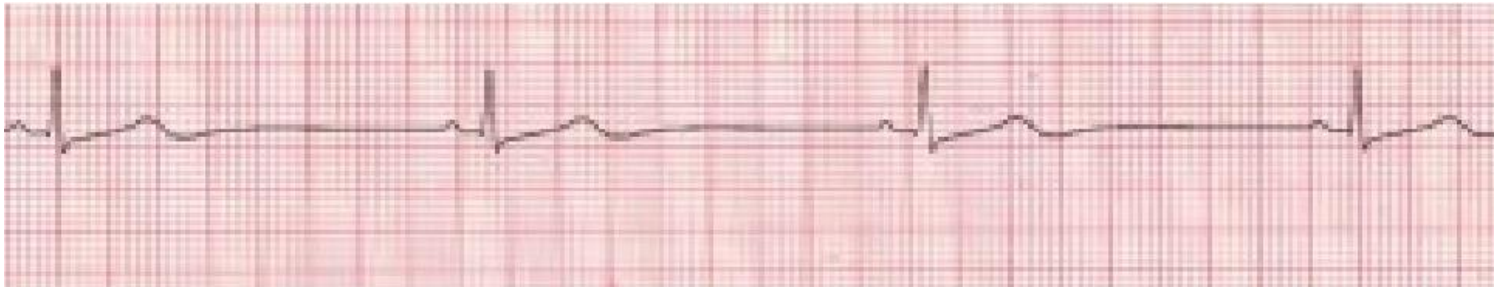

R
P
T
Q
S
P Wave
QRS Complex
T Wave
Activation of the atria
Activation of the ventricles
Recovery wave



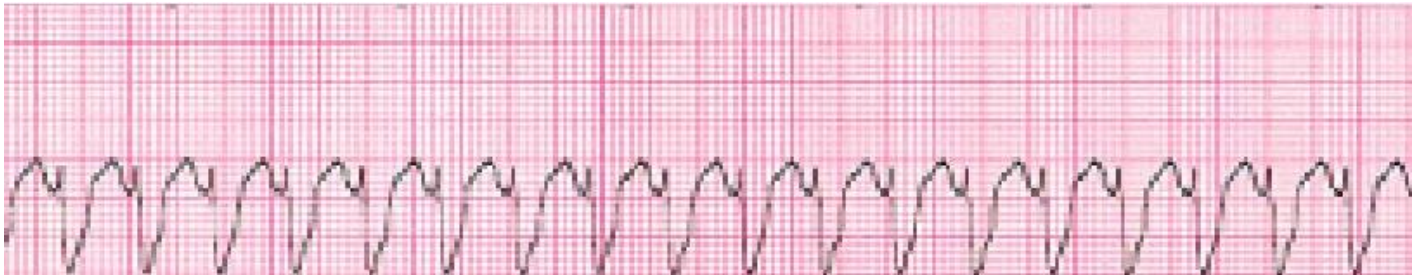
Normal Sinus rhythm – regularly spaced QRS Complexes.(60 – 80bpm)



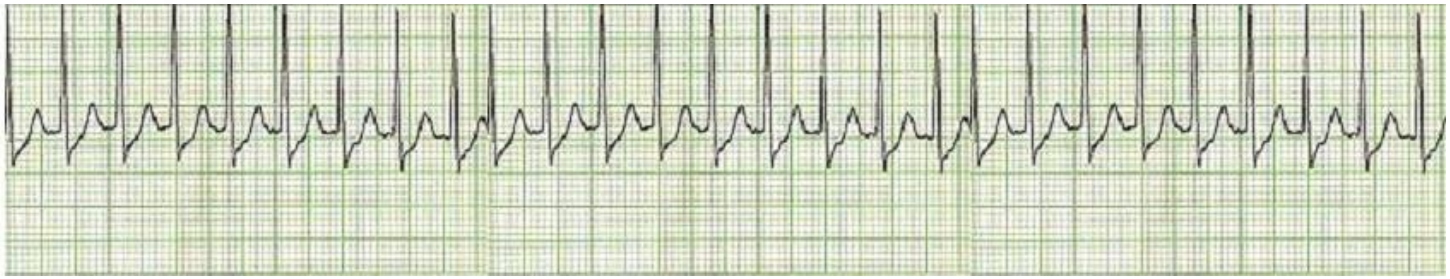
Sinus tachycardia – fast heart rate (more than 100bpm) with normal QRS complex



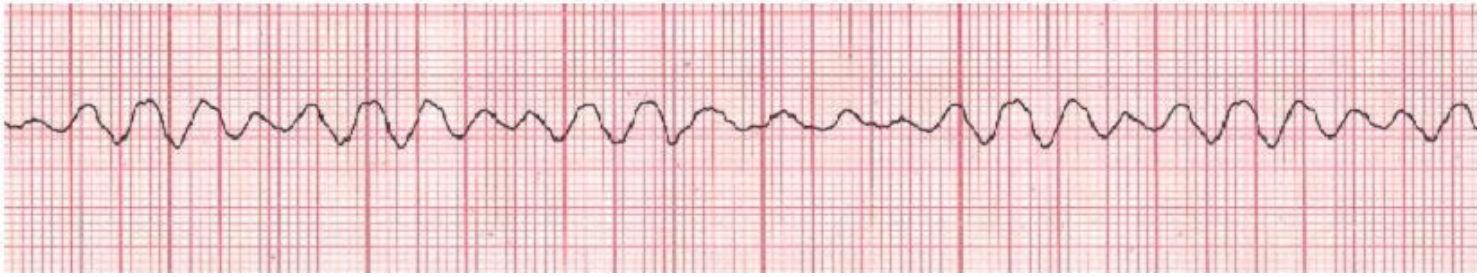
Sinus Bradycardia – regular QRS complexes at a slow rate(less than 60bpm)



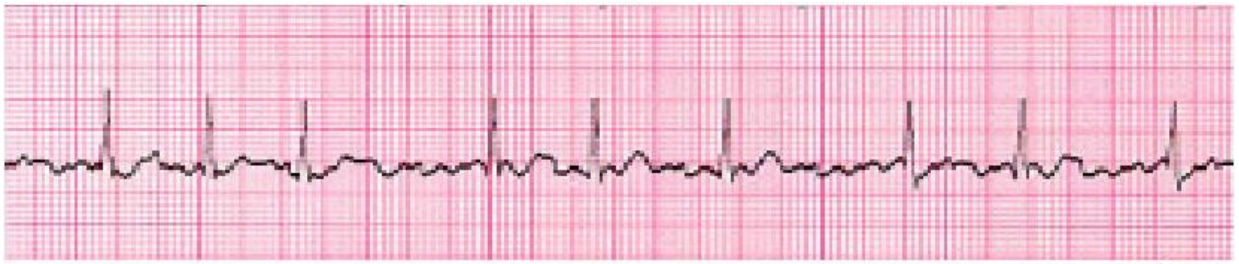
Ventricular Tachycardia – origin in ventricle, fast regular rhythm, can be life threatening if it has no pulse.



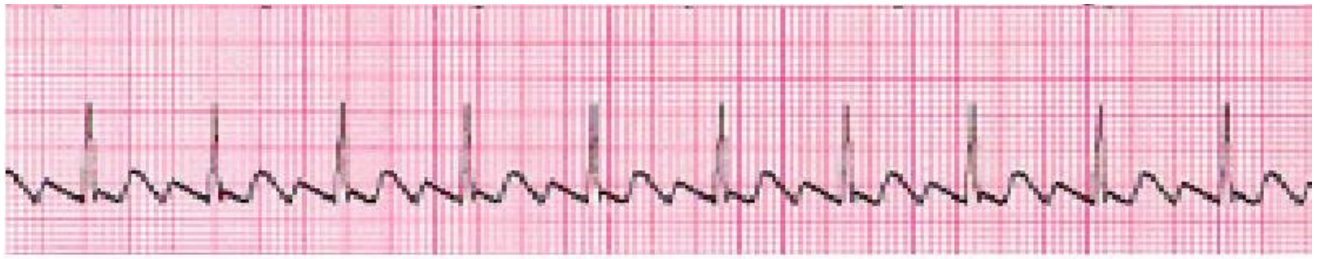
Supraventricular Tachycardia – very fast regular rhythm (more than 150bpm) can be felt as palpitations. The origin is above the ventricles.



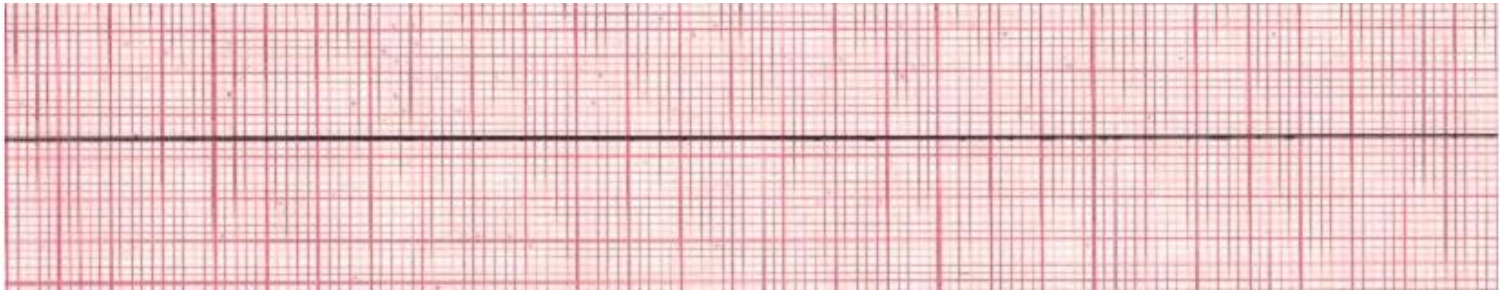
Ventricular Fibulation – Life Threatening – no QRS complex, no pulse



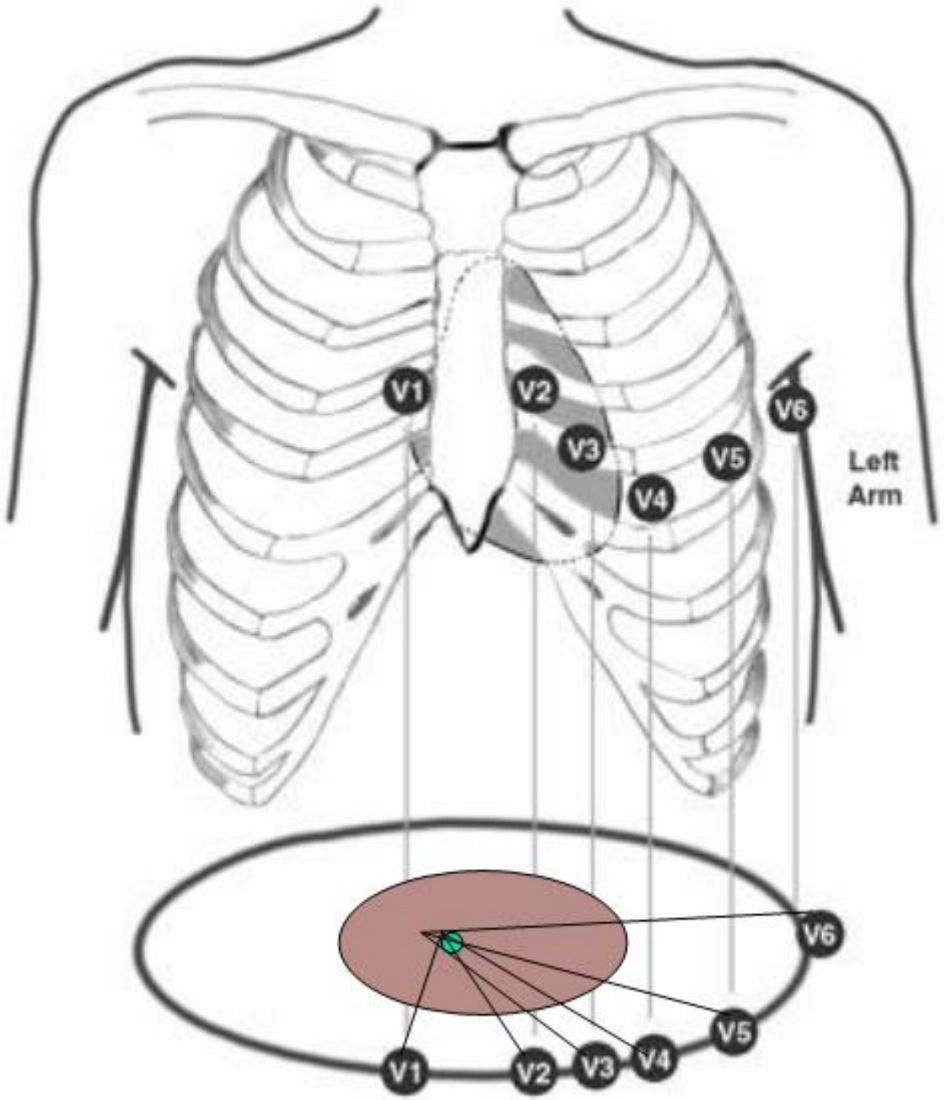
Atrial Fibulation – irregular fast heart rate with no or incomplete QRS complex



Atrial Flutter – fast heart rate with normal QRS complex and added regular p waves.

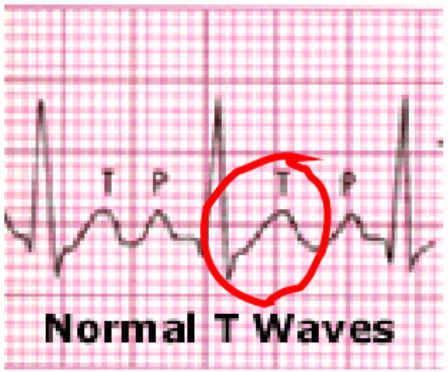


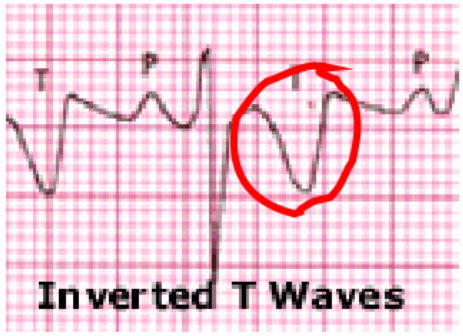
Asystole – the absence of any electrical activity.

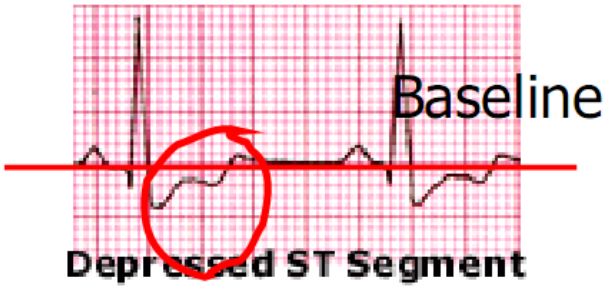

V1
V2
V3
V4
V5
V6
Left
Arm
V6
V5
V4
V3
V2
V1

Ischemia

- T-wave inversion (flipped T)
- ST segment depression
- T wave flattening
- Biphasic T-waves

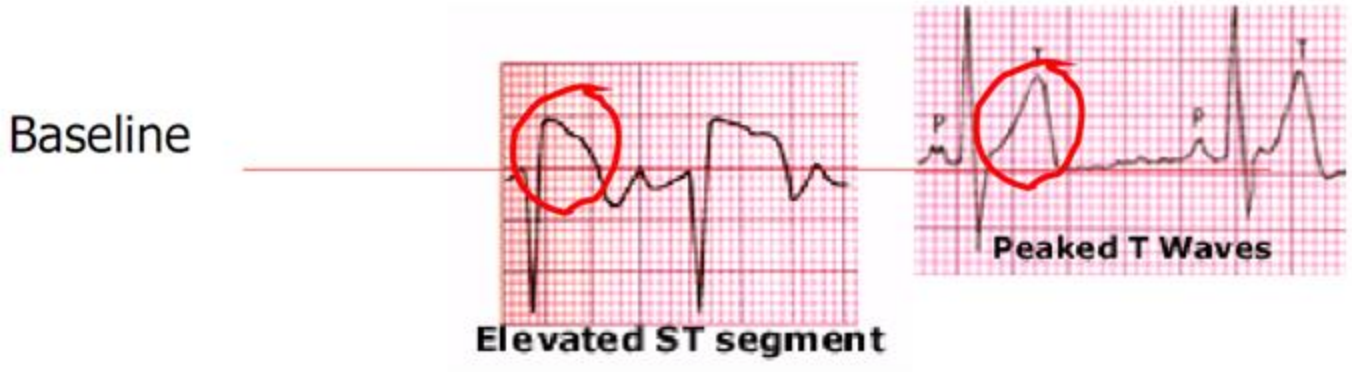






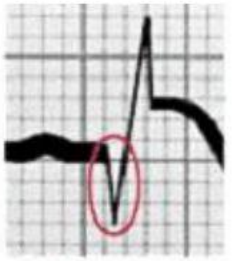
Injury

- ST segment elevation of greater than 1mm in at least 2 contiguous leads
- Heightened or peaked T waves
- Directly related to portions of myocardium rendered electrically inactive



Infarct

- Significant Q-wave where none previously existed
  - Why?
    - Impulse traveling away from the positive lead
    - Necrotic tissue is electrically dead
- No Q-wave in Subendocardial infarcts
  - Why?
    - Not full thickness dead tissue
    - But will see a ST depression
    - Often a precursor to full thickness MI
- Criteria
  - Depth of Q wave should be 25% the height of the R wave
  - Width of Q wave is 0.04 secs
  - Diminished height of the R wave



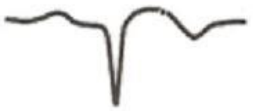
Q wave

Acute Myocardial Infarct

Preadmission

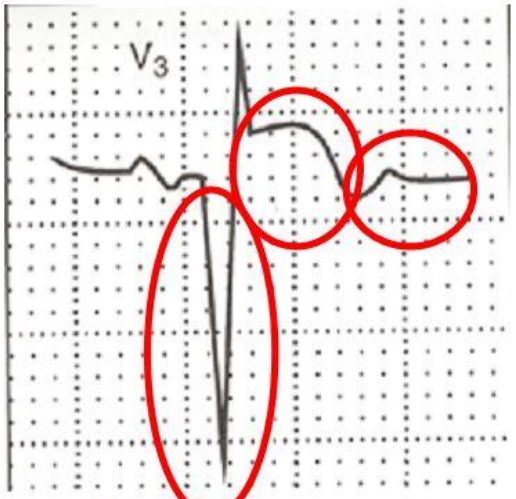
Admission

1 Hour



24 Hours

1 year



Q wave

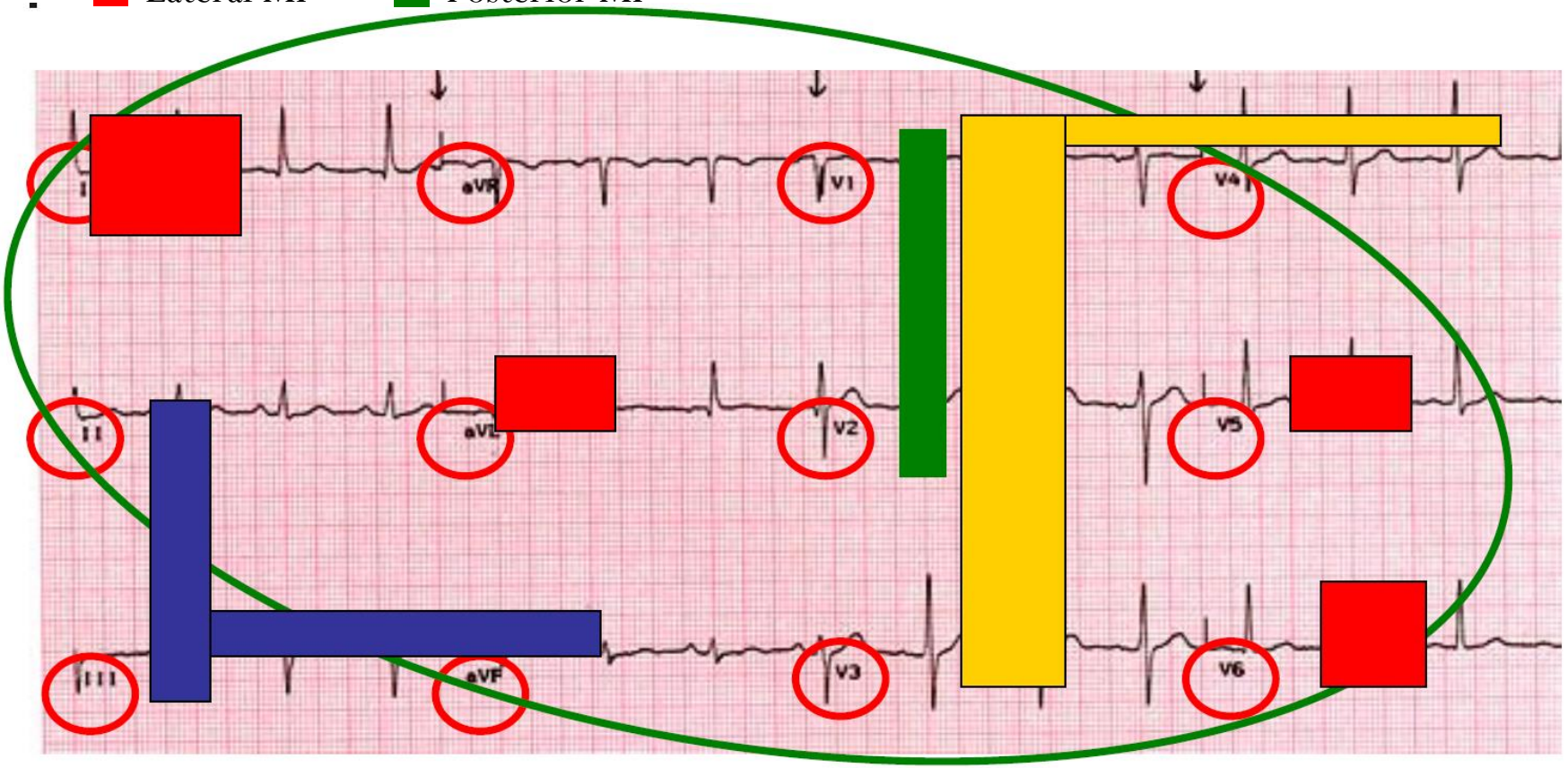
ST Elevation

T wave inversion

ECG Colour Codes for MI Diagnosis

- Anterior MI
- Inferior MI
- Lateral MI
- Posterior MI

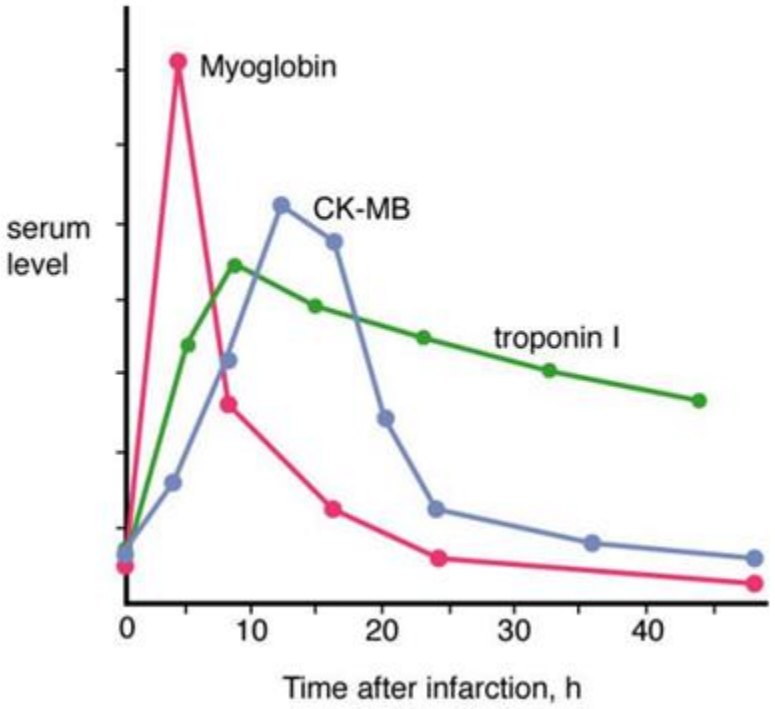
SubEndo MI – Q wave anomalies.



Troponin

Troponins·

- Rises after 3-6 hours
- Negative Troponin within 6 hours of onset of S&S rules out the MI
- Peaks at about 20 hours
- May be raised for 14 days



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