Physiology

DPT

Awais Hamza

Rate of Rhythmic Discharge of Excitatory & Conductive System

- **Rate of Rhythmic Discharge**
- SA Node → **70–80 / min**
- AV Node → **40–60 / min**
- Purkinje Fibers → **15–40 / min**
- **Additional Points**
- SA node has the **highest intrinsic rate**, hence acts as the **normal pacemaker**.
- Lower centers fire only when SA node fails (escape rhythm).

Normal Heart Rate by Age & Sex

- **Adult Male** HR = **72 (70–80) beats/min**
- **Adult Female**
- Slightly higher than male→ Lower BP + more sympathetic tone.
- **Old Age**
- Slightly higher HR→ Compensatory adjustment for age-related circulatory changes.
- **Children** HR = **95–100 beats/min**

Factors Affecting Heart Rate

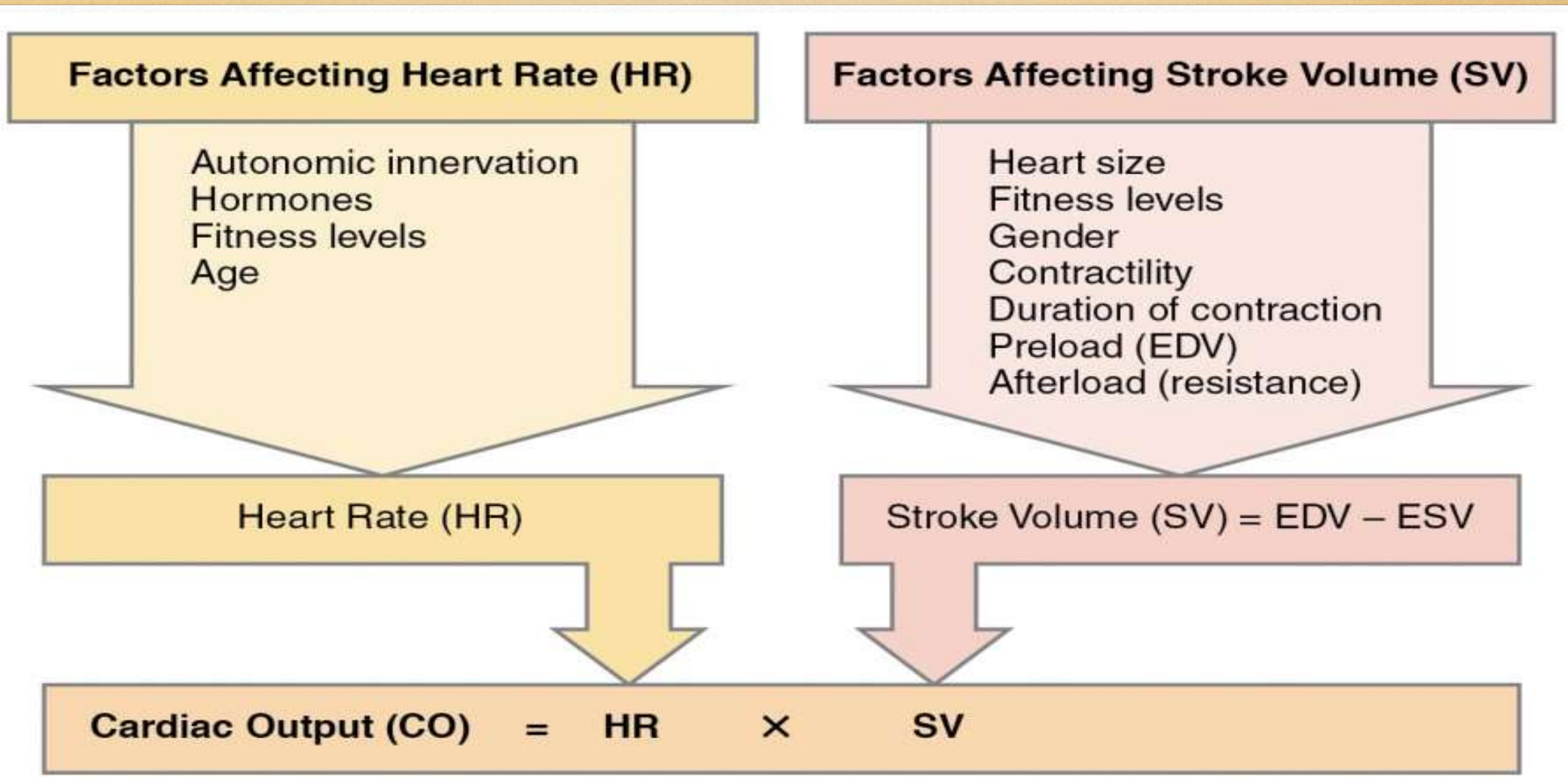
- **1) Higher Centers (Frontal Lobe & Hypothalamus)**
- Excitement → ↑HR
- Sudden Shock → ↓HR
- **2) Respiration**
- Inspiration → ↑HR
- Expiration → ↓HR

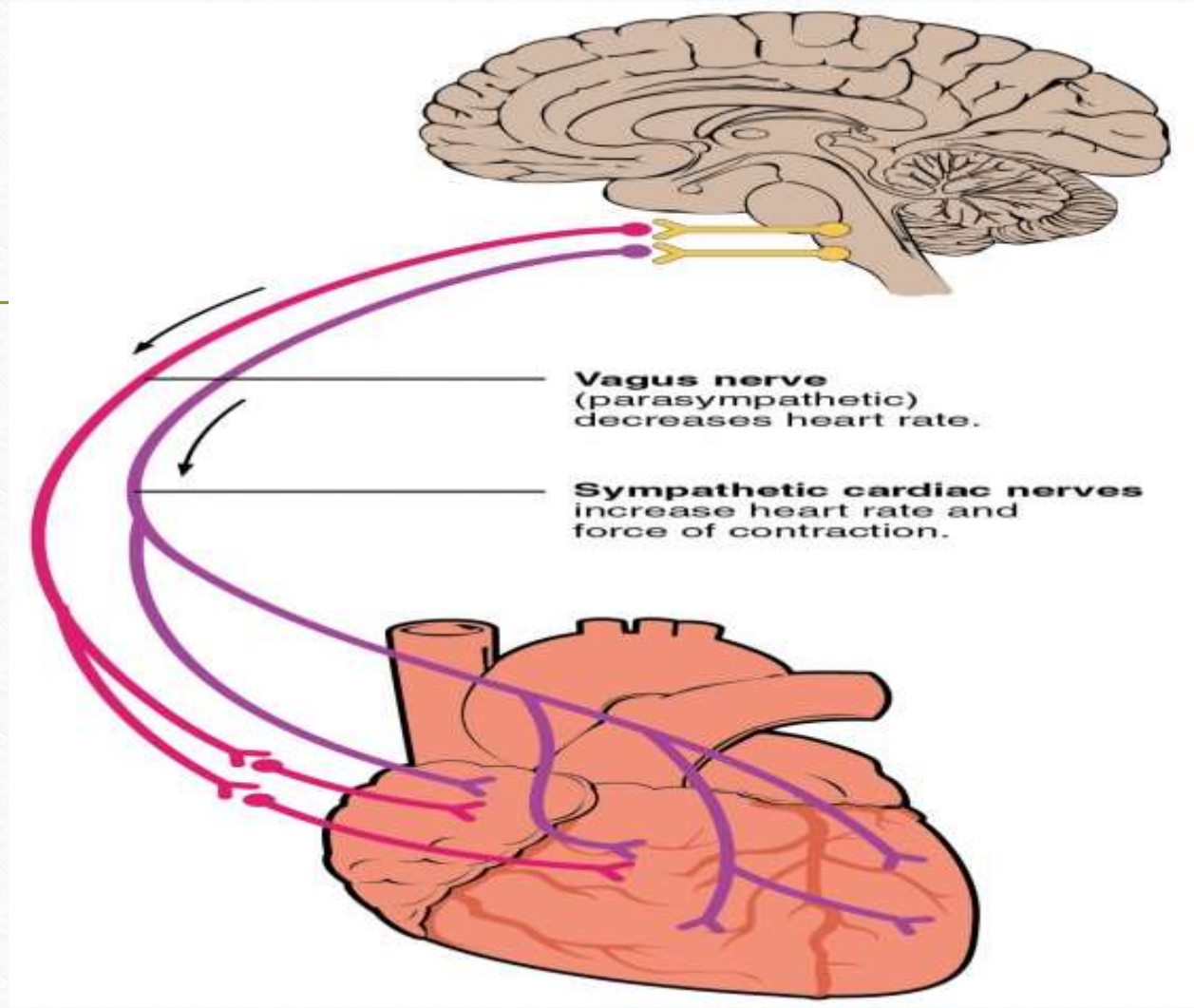
Factors Affecting Heart Rate

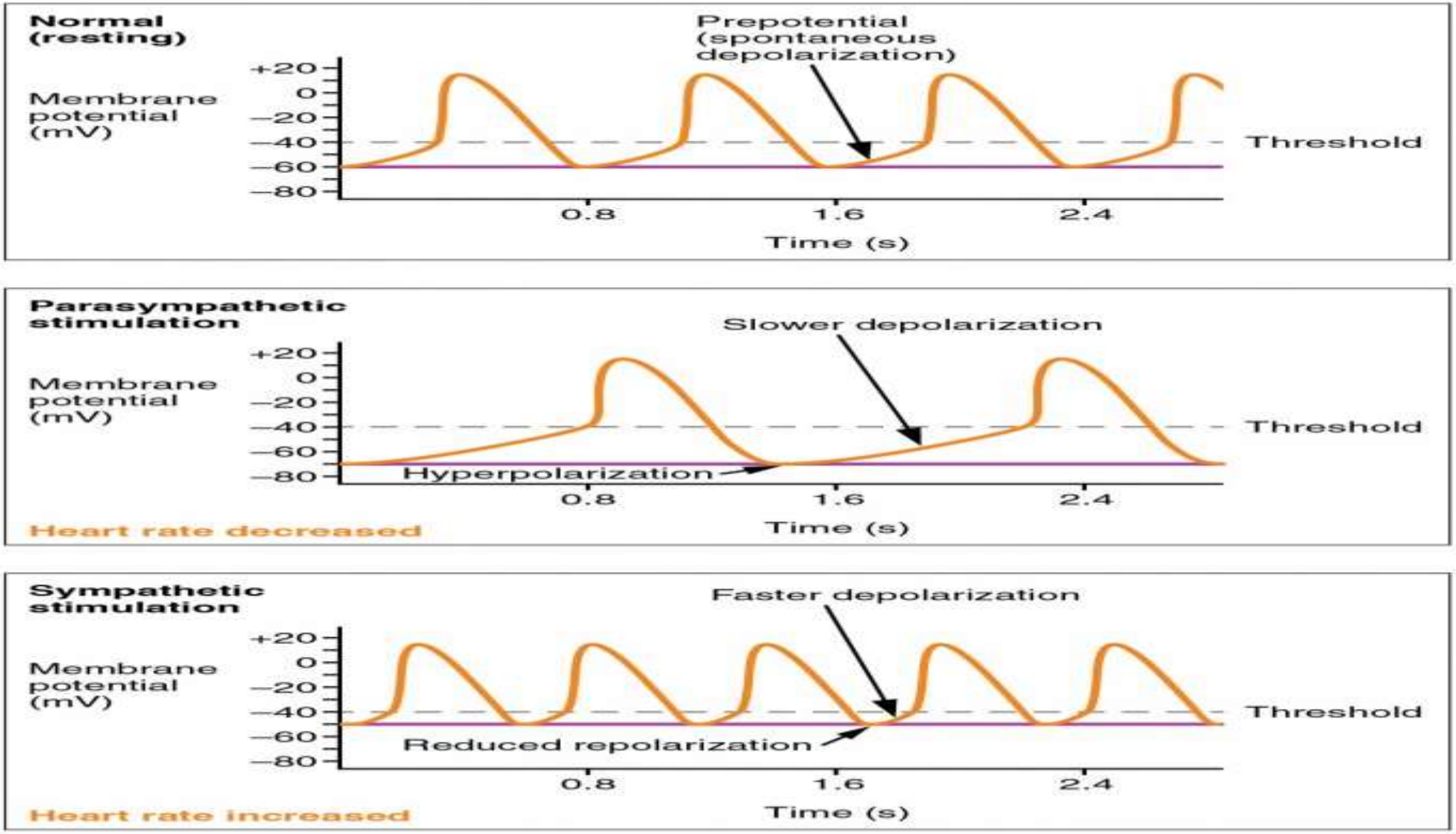
- **3) Reflexes**
- **Baroreceptor (Marey's reflex):** ↑BP → ↓HR
- **Chemoreceptor reflex:** ↓BP → ↑HR
- **Bainbridge reflex:** ↑Venous return → ↑HR

Factors Affecting Heart Rate

- **4) Anoxia → ↑HR**
  5) Moderate ↑CO_2 → ↑HR; severe ↑CO_2 → ↓HR
  6) ↑Body temperature (fever/hyperthyroidism) → ↑HR
  7) ↑Intracranial pressure → ↓HR (vagal stimulation)
  8) Sympathetic stimulation → ↑HR
  9) Thyroxine → ↑BMR → ↑HR
  10) Exercise → ↑HR


Factors Affecting Heart Rate (HR)
Autonomic innervation
Hormones
Fitness levels
Age
Factors Affecting Stroke Volume (SV)
Heart size
Fitness levels
Gender
Contractility
Duration of contraction
Preload (EDV)
Afterload (resistance)
Heart Rate (HR)
Stroke Volume (SV) = EDV – ESV
Cardiac Output (CO) = HR × SV

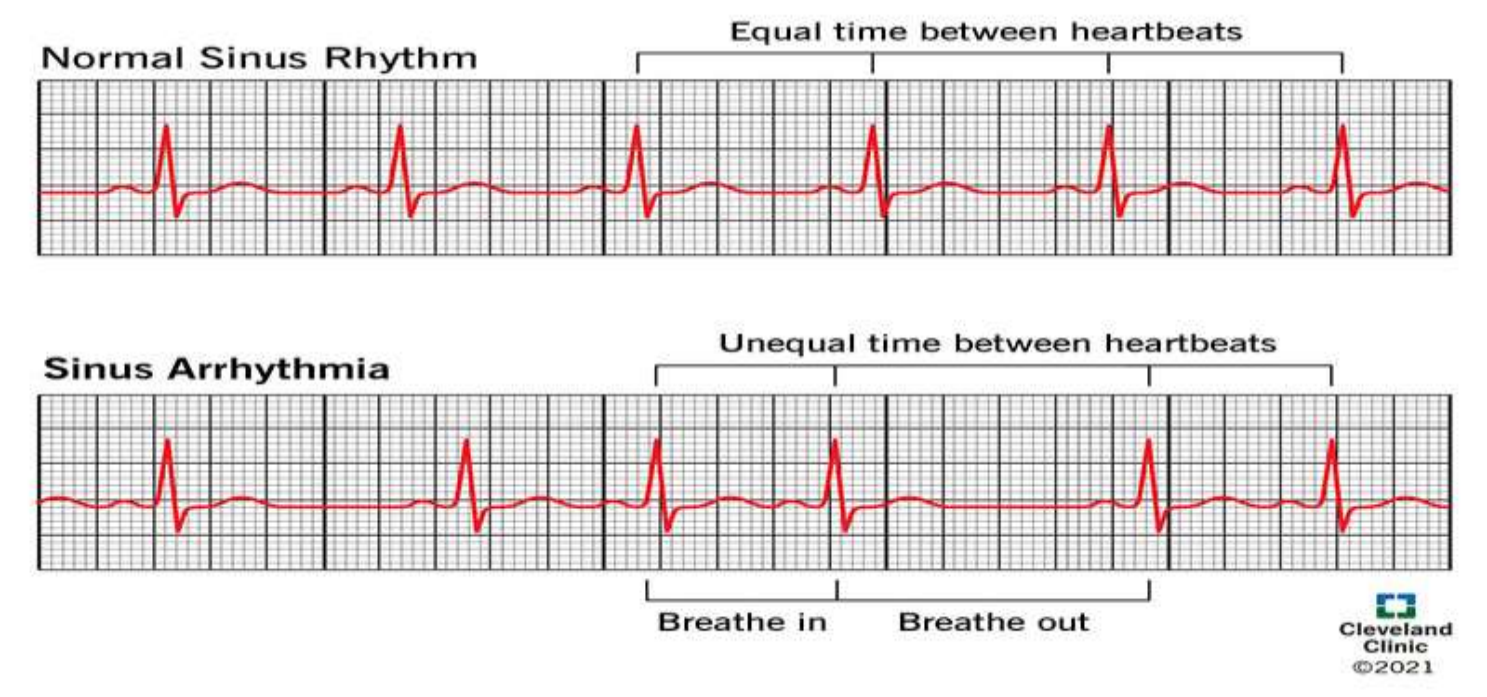

Vagus nerve
(parasympathetic)
decreases heart rate.
Sympathetic cardiac nerves
increase heart rate and
force of contraction.


Normal
(resting)
Prepotential
(spontaneous
depolarization)
Membrane
potential
(mV)
+20
0
−20
−40
−60
−80
Threshold
0.8
1.6
2.4
Time (s)
Parasympathetic
stimulation
Slower depolarization
Membrane
potential
(mV)
+20
0
−20
−40
−60
−80
Threshold
Hyperpolarization
0.8
1.6
2.4
Time (s)
Heart rate decreased
Sympathetic
stimulation
Faster depolarization
Membrane
potential
(mV)
+20
0
−20
−40
−60
−80
Threshold
Reduced repolarization
0.8
1.6
2.4
Time (s)
Heart rate increased

Sinus Arrhythmia

- **Definition**
- HR increases during inspiration and decreases during expiration.
- **Cause**
- Inspiration → ↑Venous return → ↑RA stretch → **Bainbridge reflex** → ↑HR.
- **Marey's Law**
- HR is *inversely proportional* to BP (except during exercise).

Sinus Arrhythmia



How is sinus arrhythmia treated?

- Since respiratory sinus arrhythmia is normal, people without symptoms rarely need treatment.
- With nonrespiratory sinus arrhythmia or ventriculophasic sinus arrhythmia, generally, you don't need any further testing or intervention

Regulation of Heart Rate

- HR is regulated by:
- **1) Local Mechanism**
- SA node generates rhythmic impulses (≈72 bpm).
- Any factor influencing SA node rhythmicity changes HR.

Regulation of Heart Rate

- **2) Nervous Mechanism**
- **Sympathetic system**
  - NE release → ↑Na^+ & Ca^{2+} permeability
  - ↑SA discharge rate → ↑HR
  - ↑Impulse conduction
  - ↑Force of contraction

Regulation of Heart Rate

- **Parasympathetic system**
  - ACh release → ↑K^+ permeability → Hyperpolarization
  - ↓SA discharge rate → ↓HR
  - ↓Impulse conduction
  - ↓Force of contraction

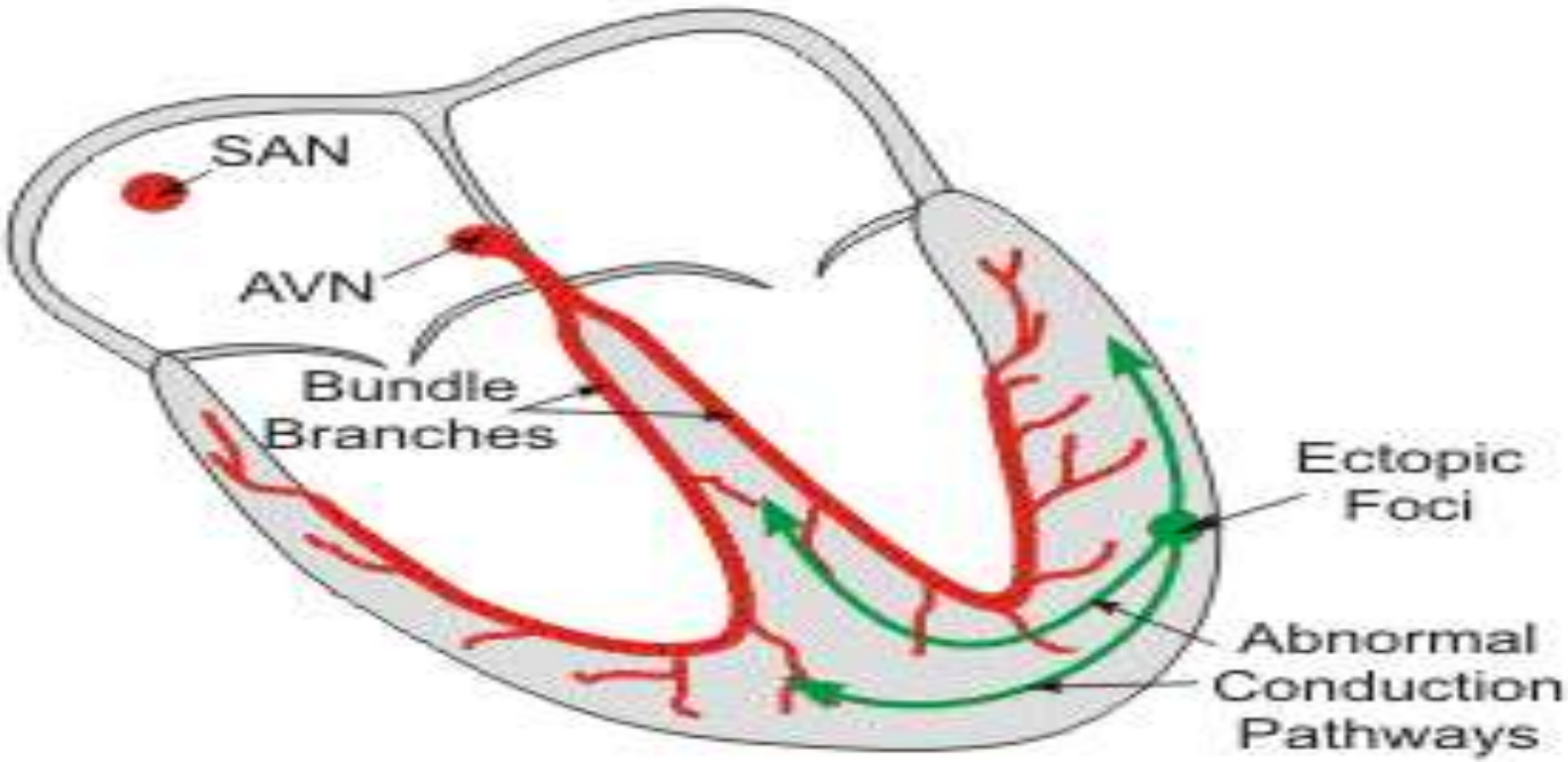
Pacemaker of Heart

- **Definition**
- Site that originates rhythmic cardiac impulses.
  OR
- Structure that determines HR.
- **Normal Pacemaker: SA Node**
  Reason: Highest discharge rate (70–80/min).

Ectopic Pacemaker & Overdrive Suppression

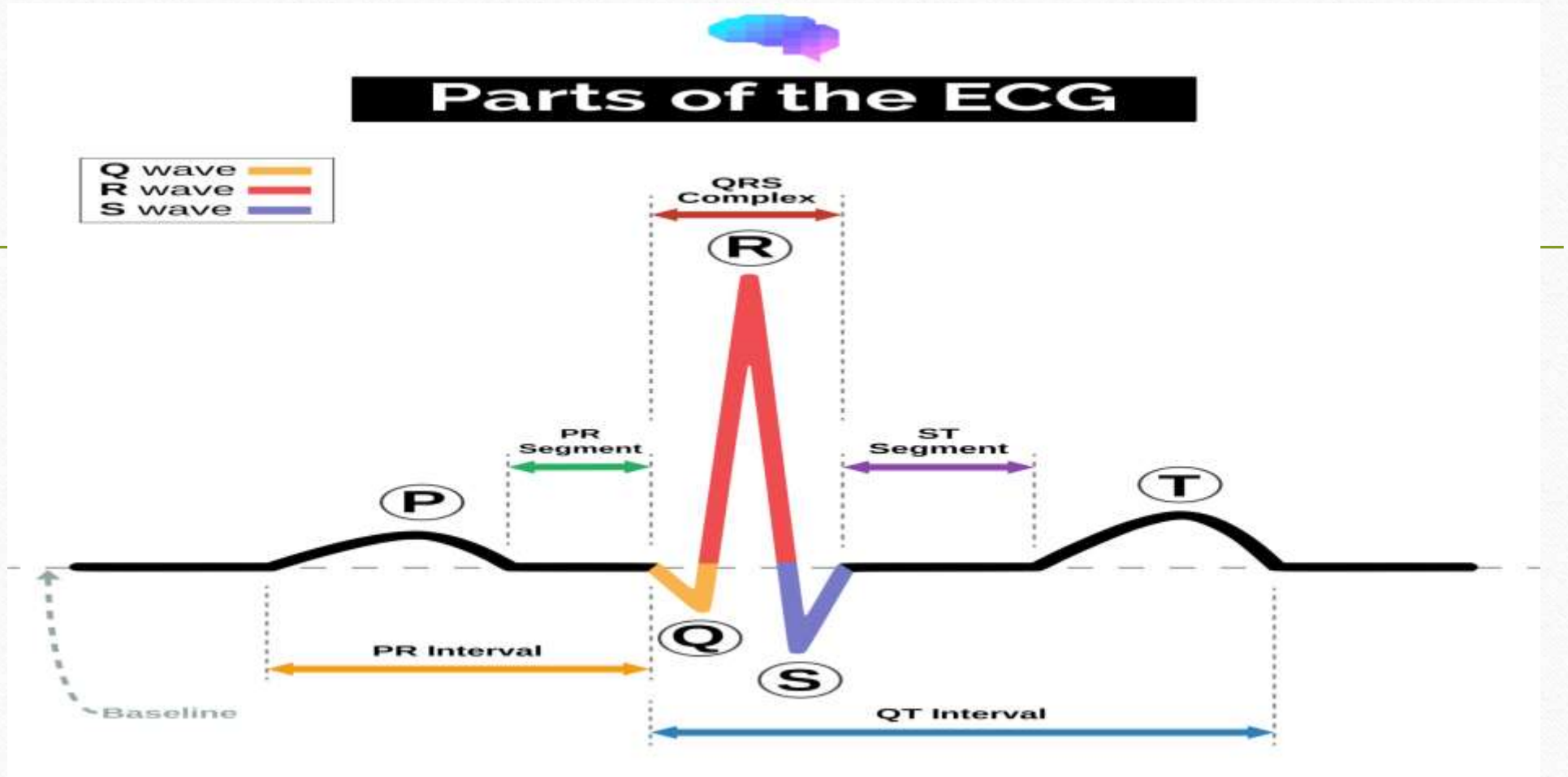
- **Ectopic Pacemaker**
- Any pacemaker outside SA node.
- **Causes**
- AV node or Purkinje fibers firing > SA node rate
- Block of SA node impulse transmission
- **Overdrive Suppression**
- SA node (70–80/min) suppresses Purkinje system (15–40/min).
- Fast rhythm → Temporary suppression of lower centers.

Abnormal Electrical Conduction due to Ventricular Ectopic Foci

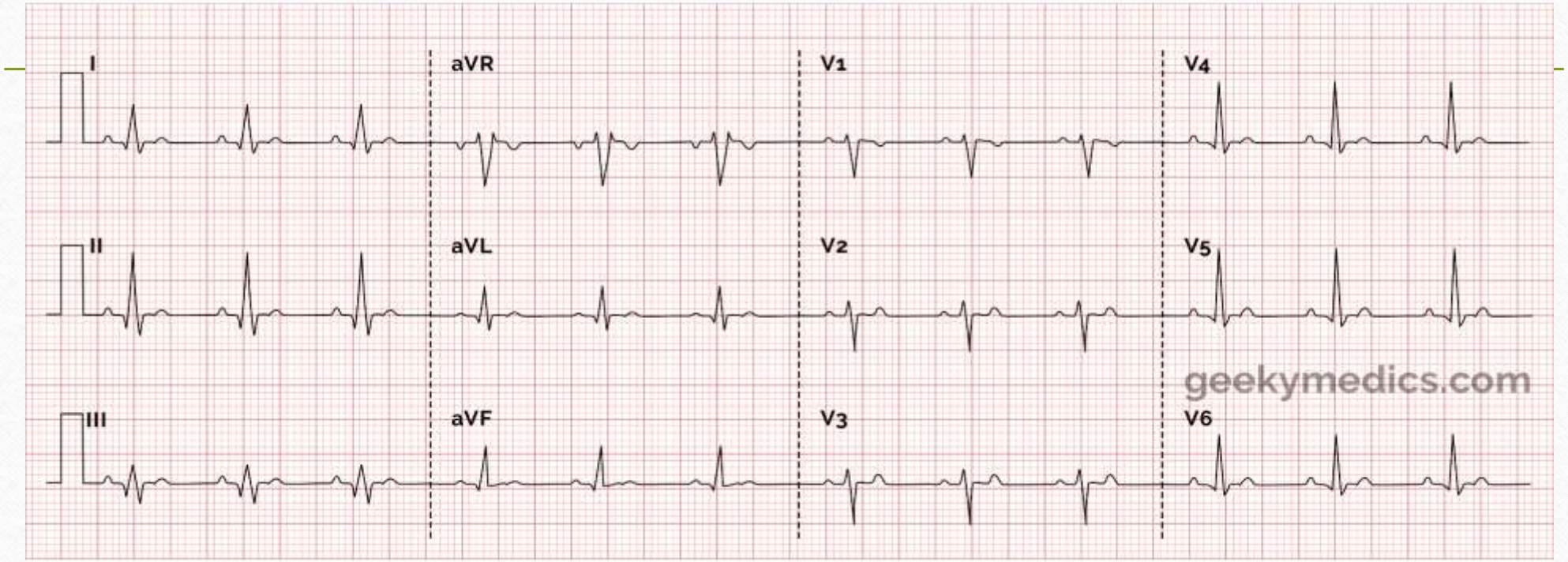


Electrocardiogram (ECG)

- **Definition**
- Graphic recording of electrical potentials during depolarization & repolarization.
- **Father of ECG:** Einthoven (1903)
- **Significance**
- Identify pacemaker site, Calculate HR, Recognize rhythm
- Detect voltage changes
- Diagnose heart diseases


Parts of the ECG
Q wave
R wave
S wave
QRS Complex
R
PR Segment
ST Segment
P
T
Q
S
PR Interval
QT Interval
Baseline

Normal ECG



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ECG Waves

- **P Wave**
- Atrial depolarization
- **QRS Complex**
- Ventricular depolarization
- **T Wave & ST Segment**
- Ventricular repolarization

ECG Waves

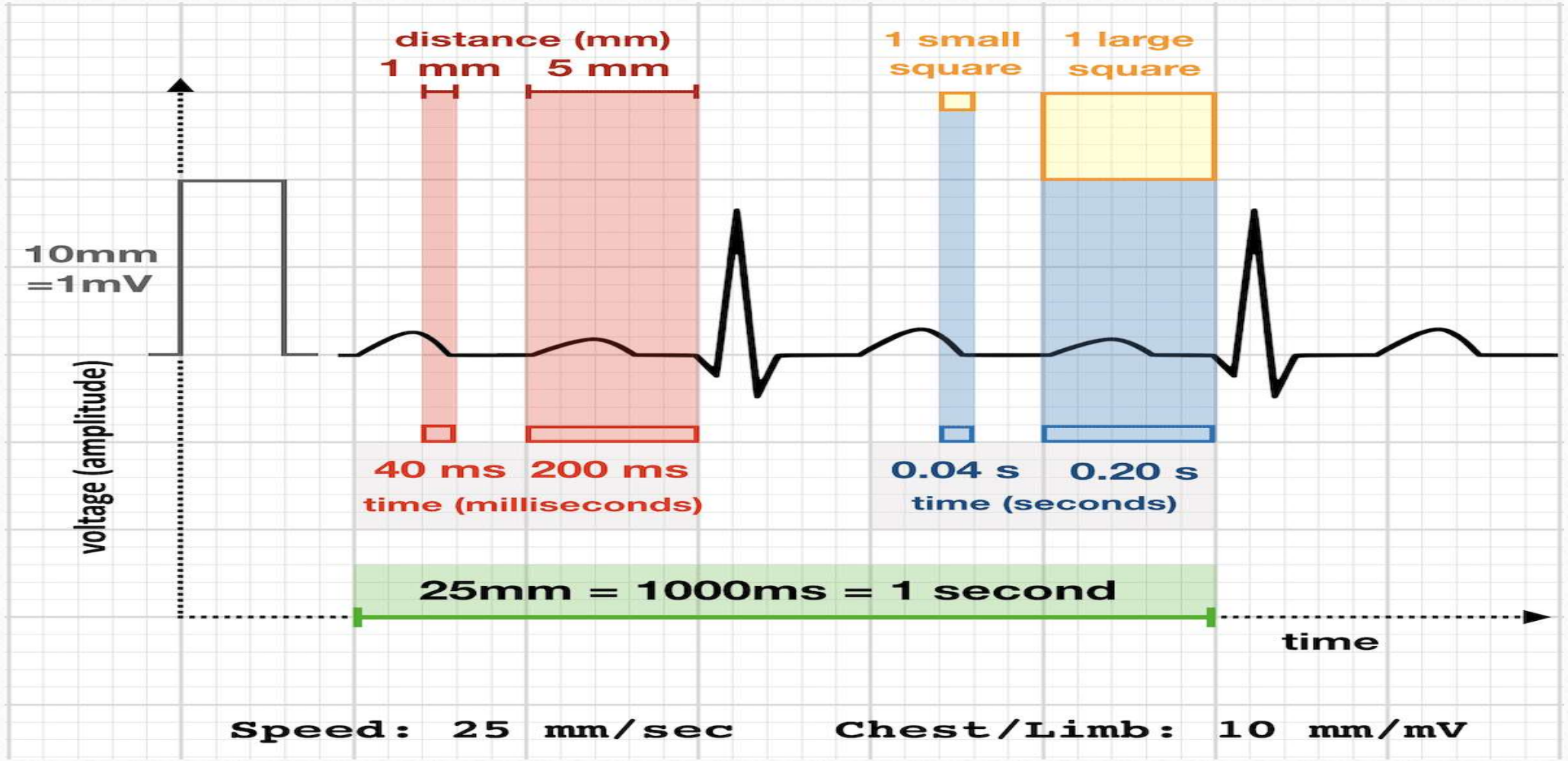
- **Atrial T Wave**
- Atrial repolarization (hidden in QRS)
- **U Wave**
- Papillary muscle repolarization
- Prominent in **hypokalemia**

ECG Intervals & Durations

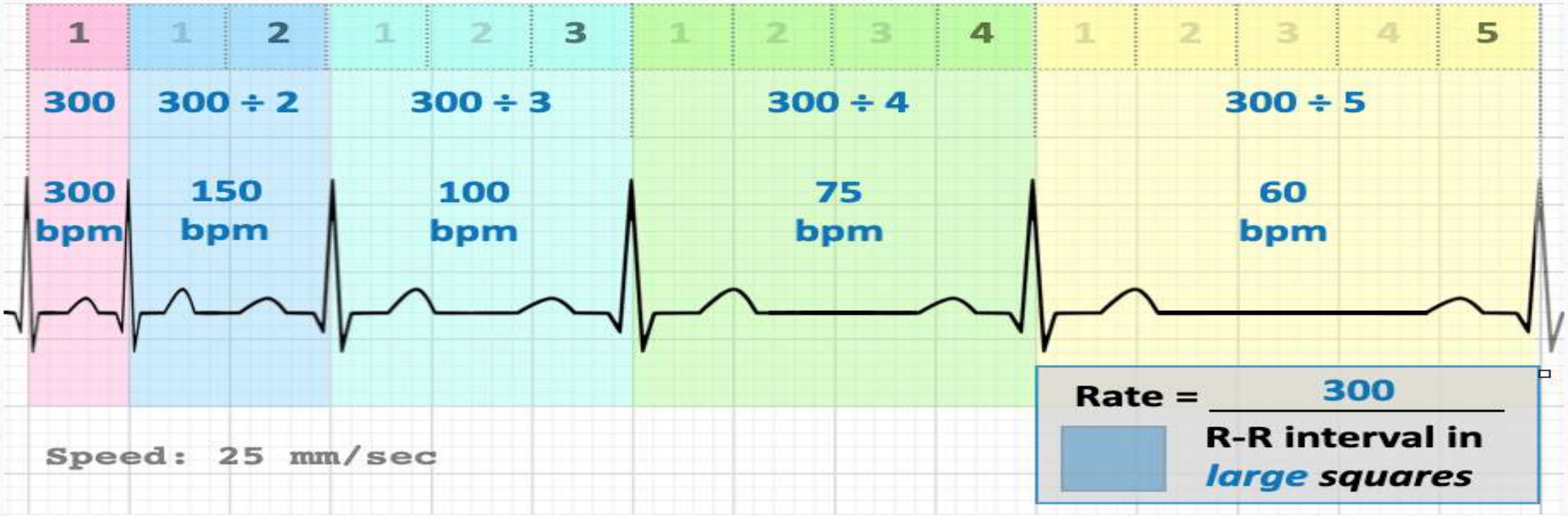
- **PR Interval**
- Onset of atrial → onset of ventricular contraction **0.16 sec (120–210 ms)**
- **QT Interval**
- Ventricular contraction duration **0.35 sec**
- **Total ECG Time (one heartbeat) 0.83 sec**
- **HR Calculation**
- HR = 60 / 0.83 = **72 bpm**

ECG Calculation Methods

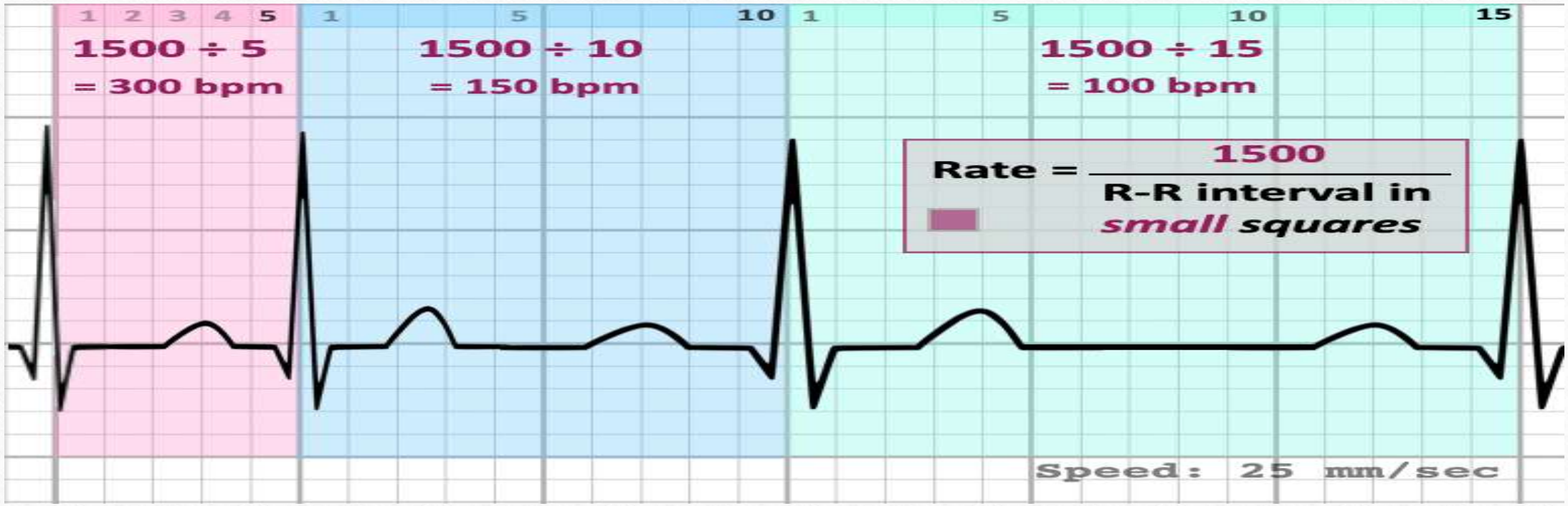
- Small squares between R–R / 1500
- Large squares between R–R / 300
- For irregular rhythm:
  Count QRS in 15 large squares × 20


distance (mm)
1 mm
5 mm
1 small square
1 large square
10mm =1mV
voltage (amplitude)
40 ms
200 ms
time (milliseconds)
0.04 s
0.20 s
time (seconds)
25mm = 1000ms = 1 second
time
Speed: 25 mm/sec
Chest/Limb: 10 mm/mV

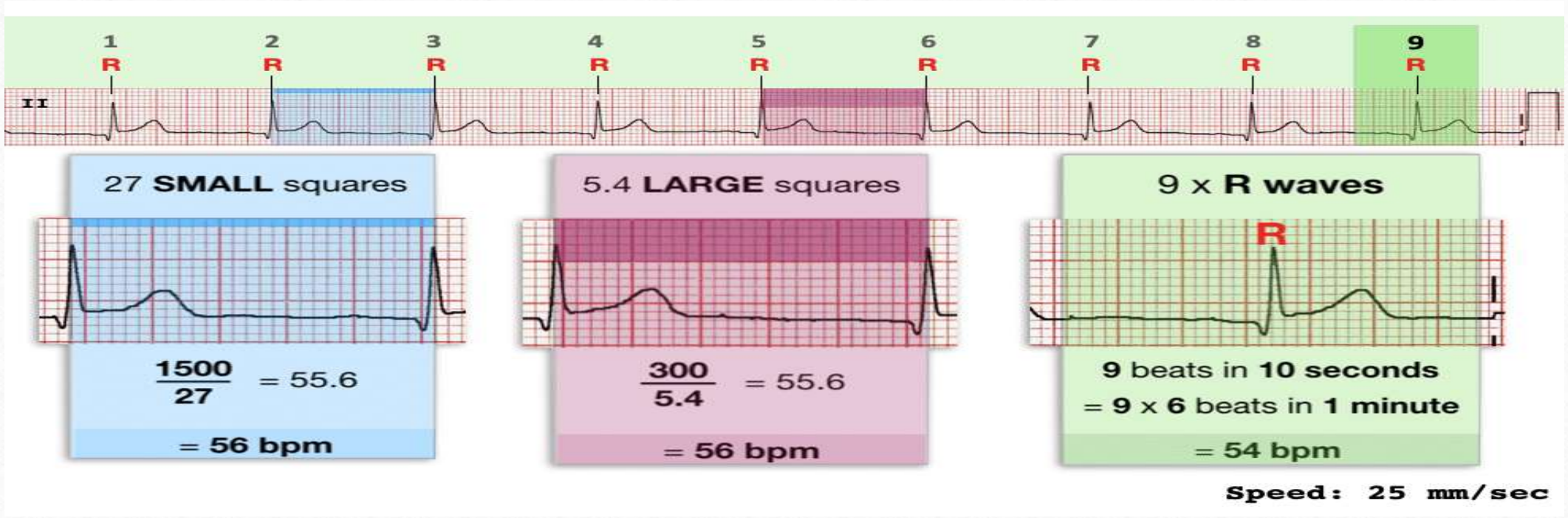
Large square method



Small square method



R wave method



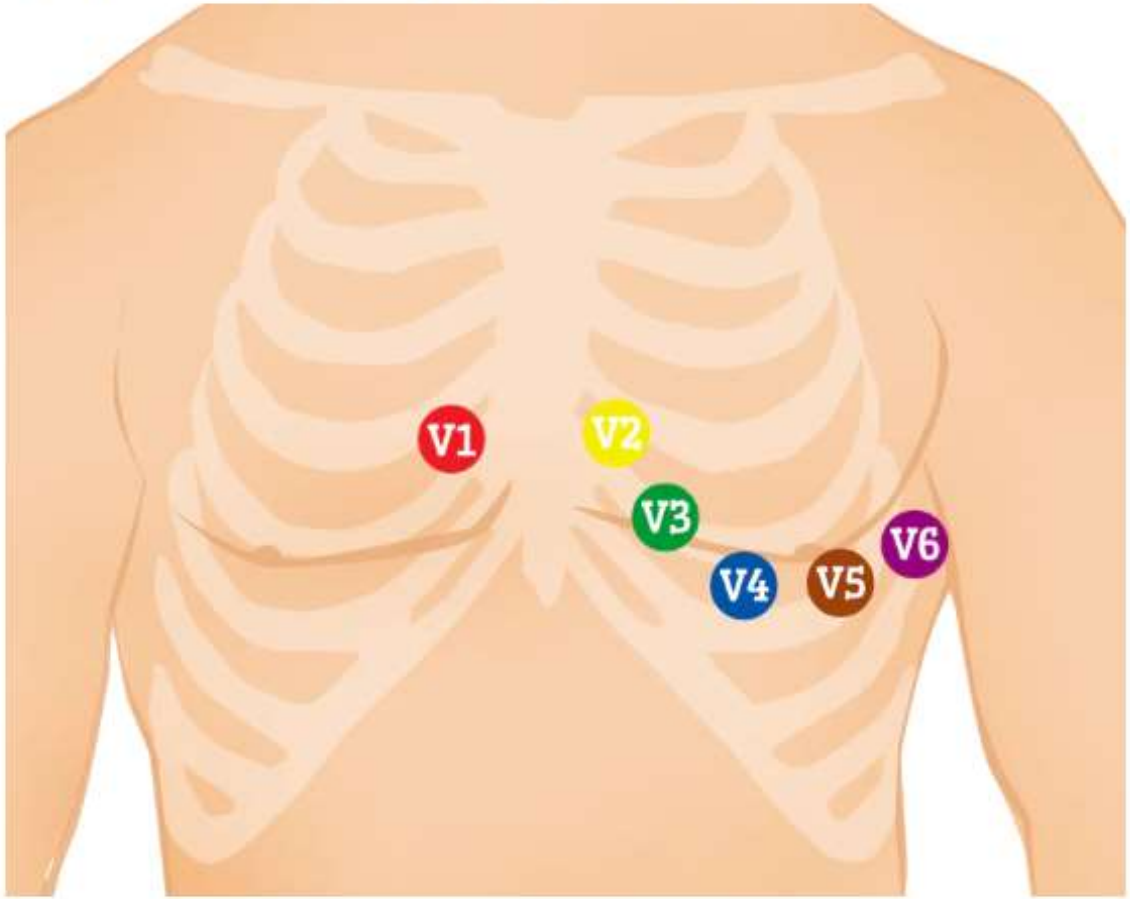
Electrocardiographic Leads

- **Bipolar Limb Leads**
- **Lead I:** RA (–) → LA (+)
- **Lead II:** RA (–) → LL (+)
- **Lead III:** LA (–) → LL (+)
- **Precordial (Chest) Leads**
- V1 → 4th ICS right
- V2 → 4th ICS left

Electrocardiographic Leads

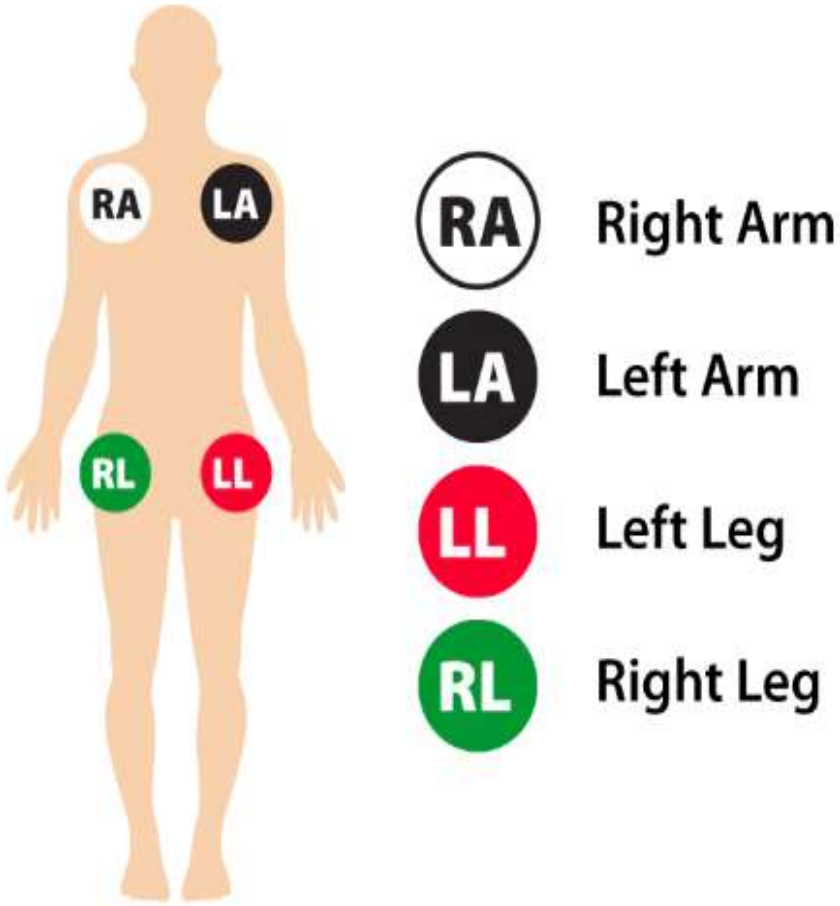
- V3 → Midway V2 & V4
- V4 → 5th ICS MCL
- V5 → Anterior axillary line
- V6 → Mid-axillary line
- **Augmented Unipolar Limb Leads**
- aVR → Right arm
- aVL → Left arm
- aVF → Left leg

Chest (Precordial) Electrodes and Placement



» V1 - Fourth intercostal space on the right sternum
» V2 - Fourth intercostal space at the left sternum
» V3 - Midway between placement of V2 and V4
» V4 - Fifth intercostal space at the midclavicular line
» V5 - Anterior axillary line on the same horizontal level as V4
» V6 - Mid-axillary line on the same horizontal level as V4 and V5

Limb (Extremity) Electrodes and Placement



RA Right Arm

LA Left Arm

LL Left Leg

RL Right Leg

» RA (Right Arm) - Anywhere between the right shoulder and right elbow

» RL (Right Leg) - Anywhere below the right torso and above the right ankle

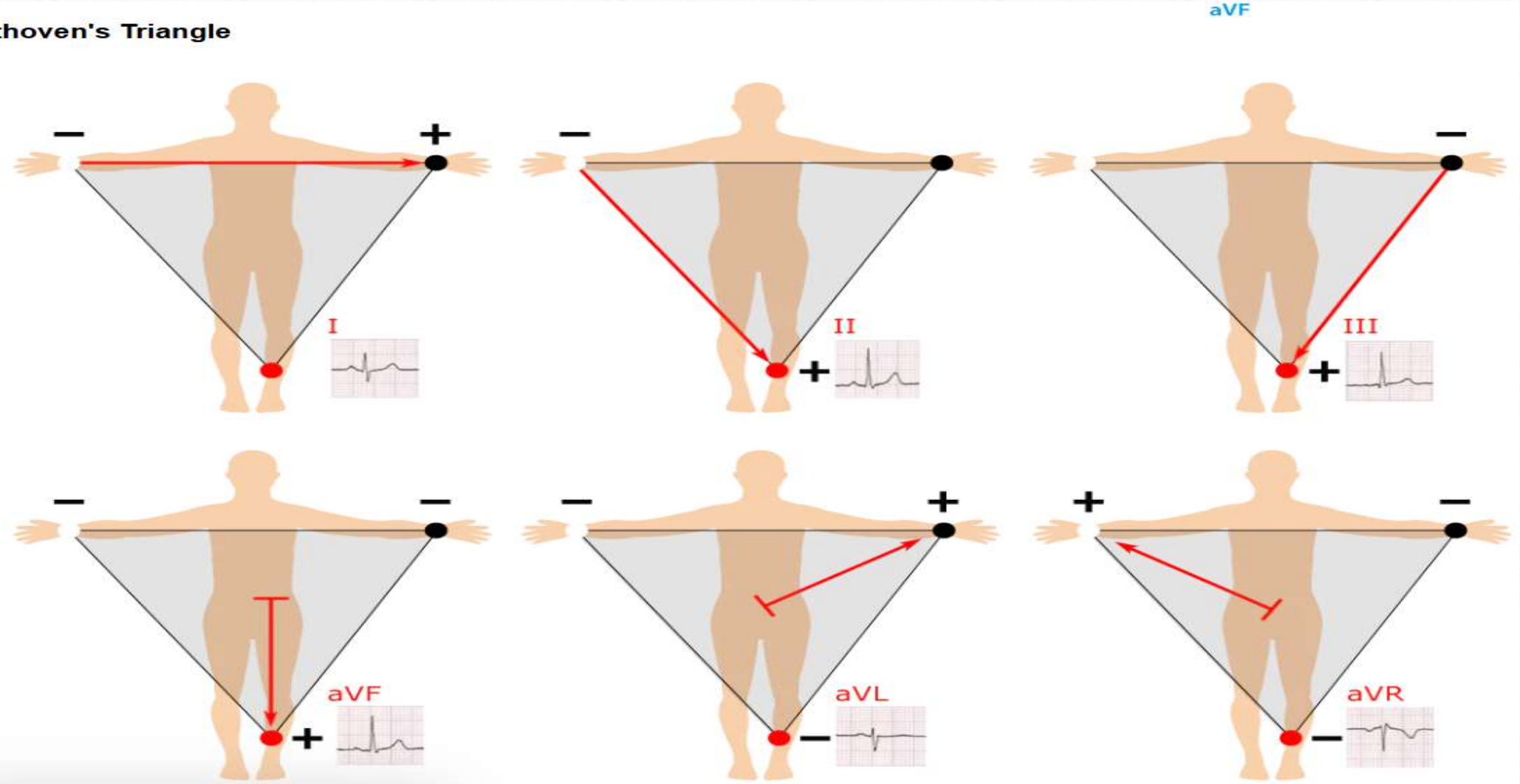
» LA(Left Arm) - Anywhere between the left shoulder and the left elbow

» LL (Left Leg) - Anywhere below the left torso and above the left ankle

Einthoven's Triangle & Law

- **Einthoven's Triangle**
- Equilateral triangle around heart (RA, LA, LL)
- **Einthoven's Law**
- Lead II = Lead I + Lead III

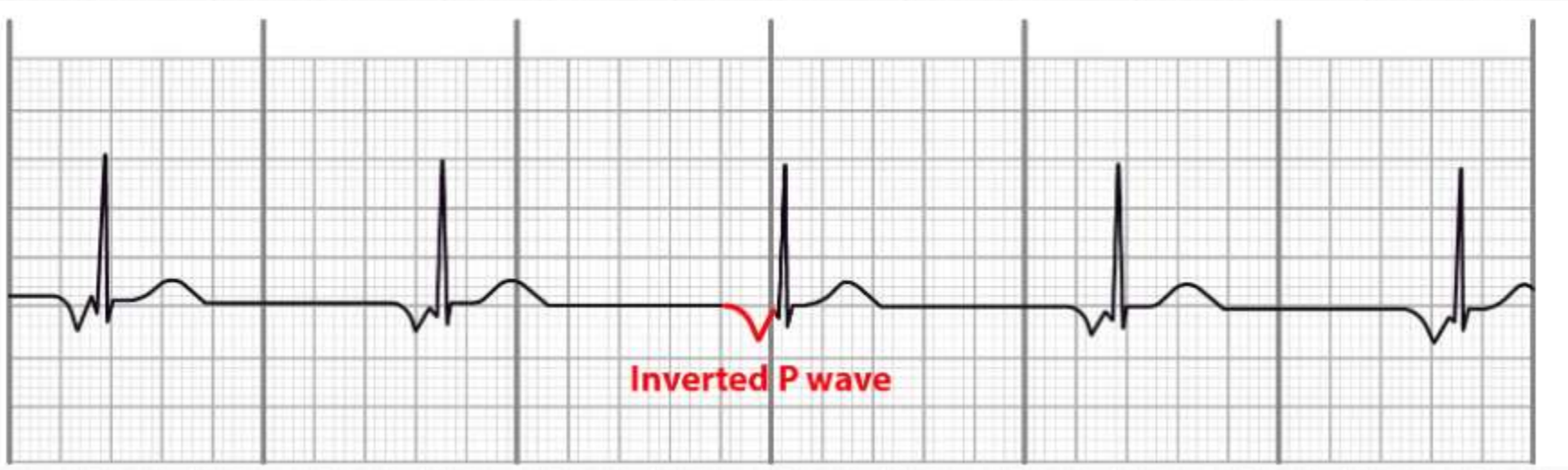
Einthoven's Triangle



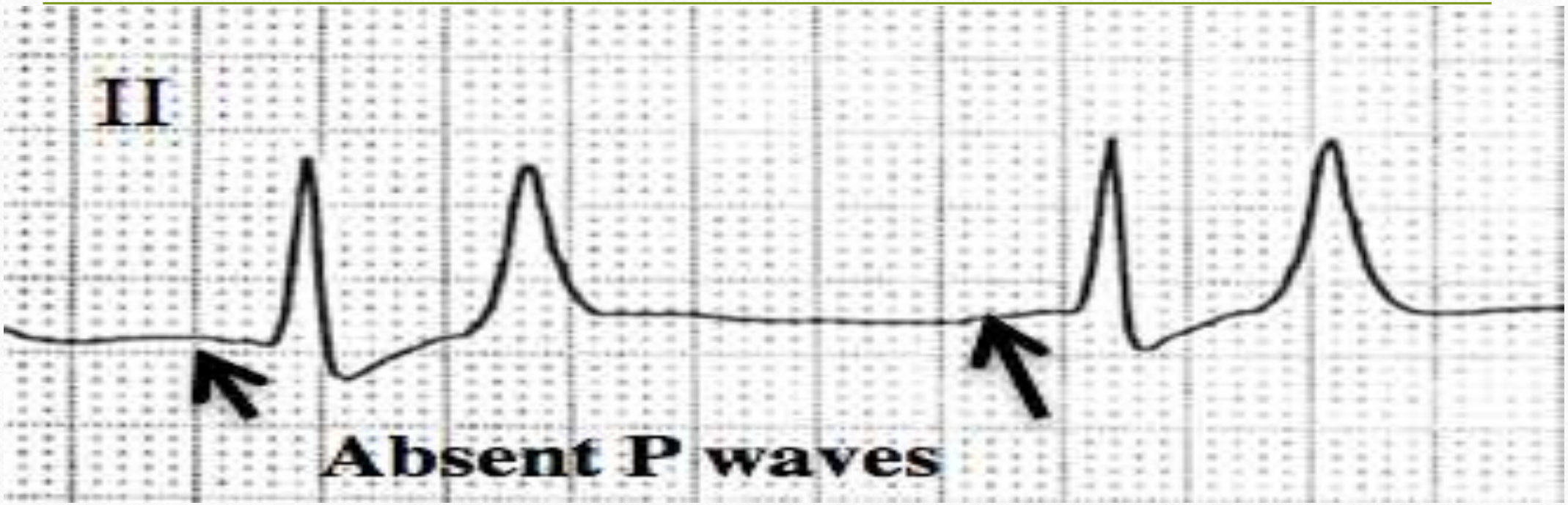
Abnormalities of P Wave

- **Inverted P:** Pacemaker shifted to AV node
- **Absent P:** Atrial fibrillation
- **P-mitrale:** Left atrial hypertrophy (notched P)
- **P-pulmonale:** Right atrial hypertrophy (tall P)
- **Nodal rhythm:** Reversed P direction

Inverted P



Absent P



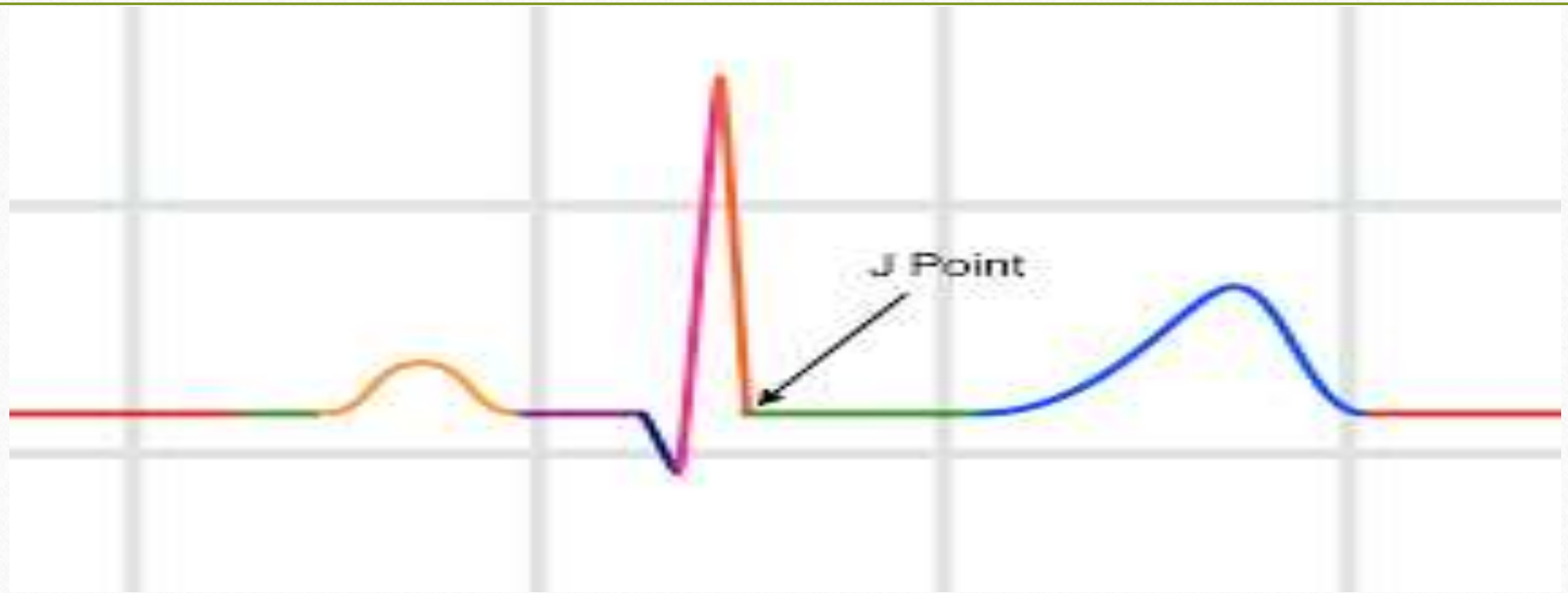
Abnormalities of QRS Complex

- **High Voltage:** RVH / LVH
- **Low Voltage:** Pericardial effusion, old MI
- **Prolonged QRS:** Ventricular hypertrophy, bundle block
- **Bizarre QRS:** Scar tissue, conduction block

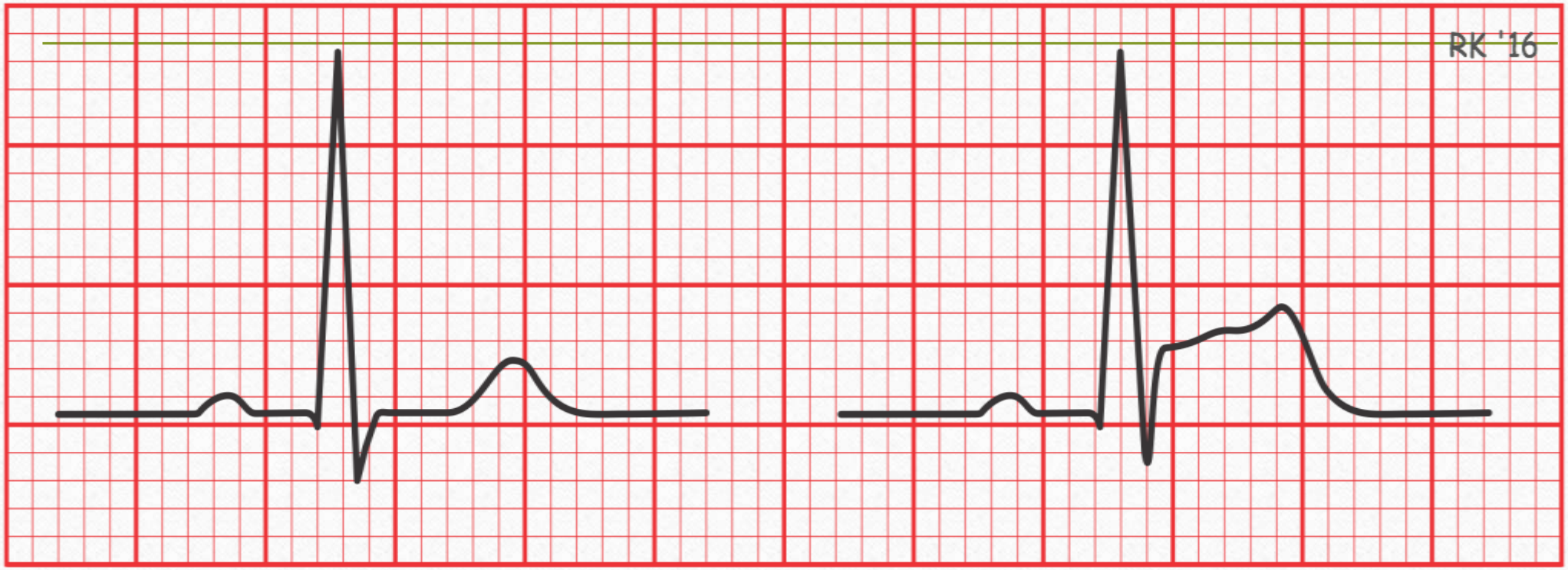
Current of Injury, J Point & ST Shift

- **Current of Injury**
- Flow from injured (–) to normal (+) tissue.
- **Causes**
- Trauma, infection, ischemia
- **J Point**
- End of QRS where potential = zero
- **ST Segment Shift**
- ST ≠ TP level → Present in ischemia/injury

J Point

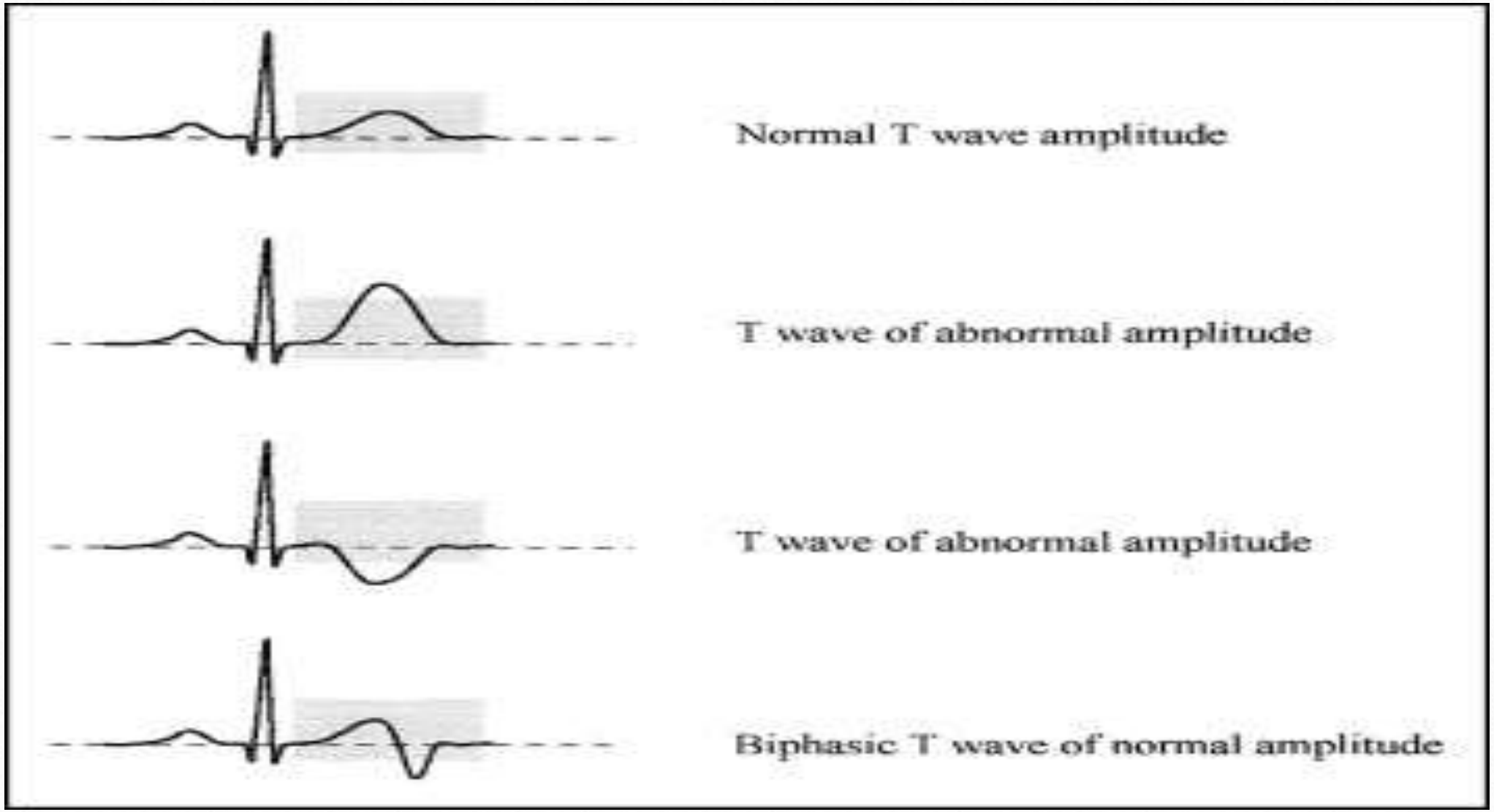


ST Segment Shift



T Wave Abnormalities

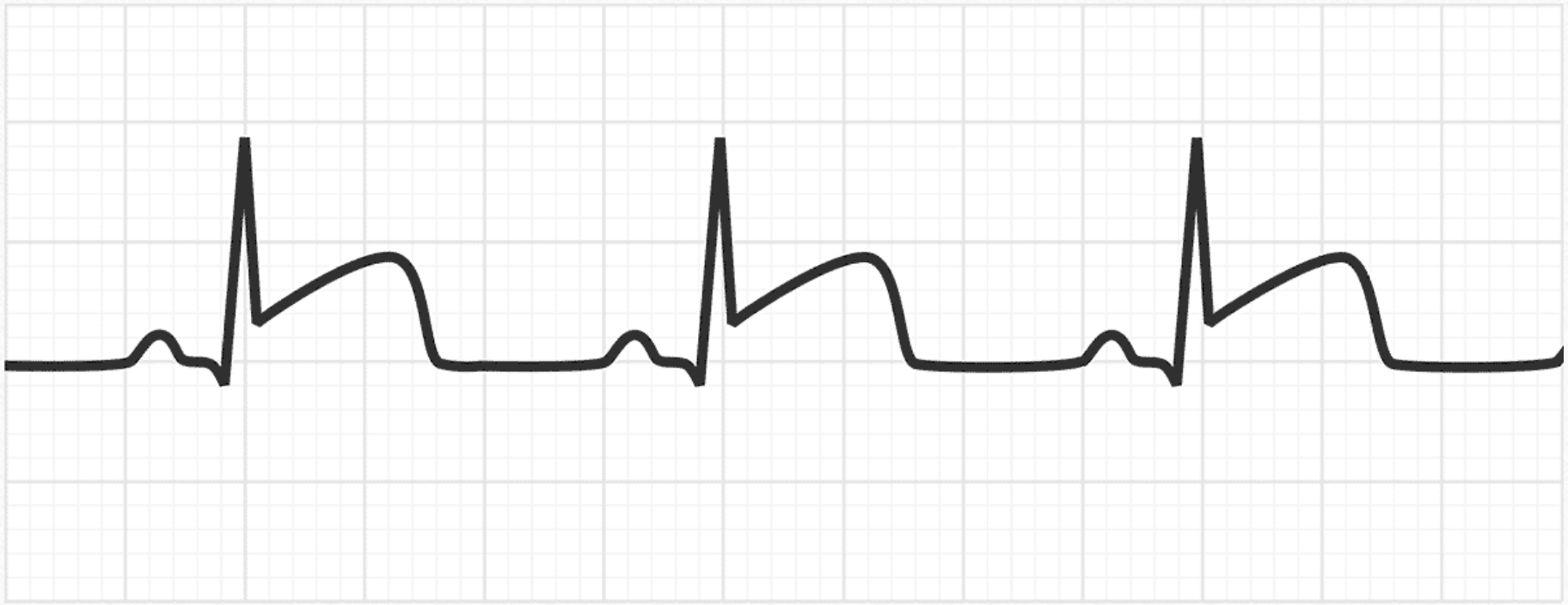
- **Opposite T wave polarity:** Bundle branch block
- **Inverted T wave:** Ischemia
- **Biphasic T:** Digitalis toxicity


Normal T wave amplitude
T wave of abnormal amplitude
T wave of abnormal amplitude
Biphasic T wave of normal amplitude

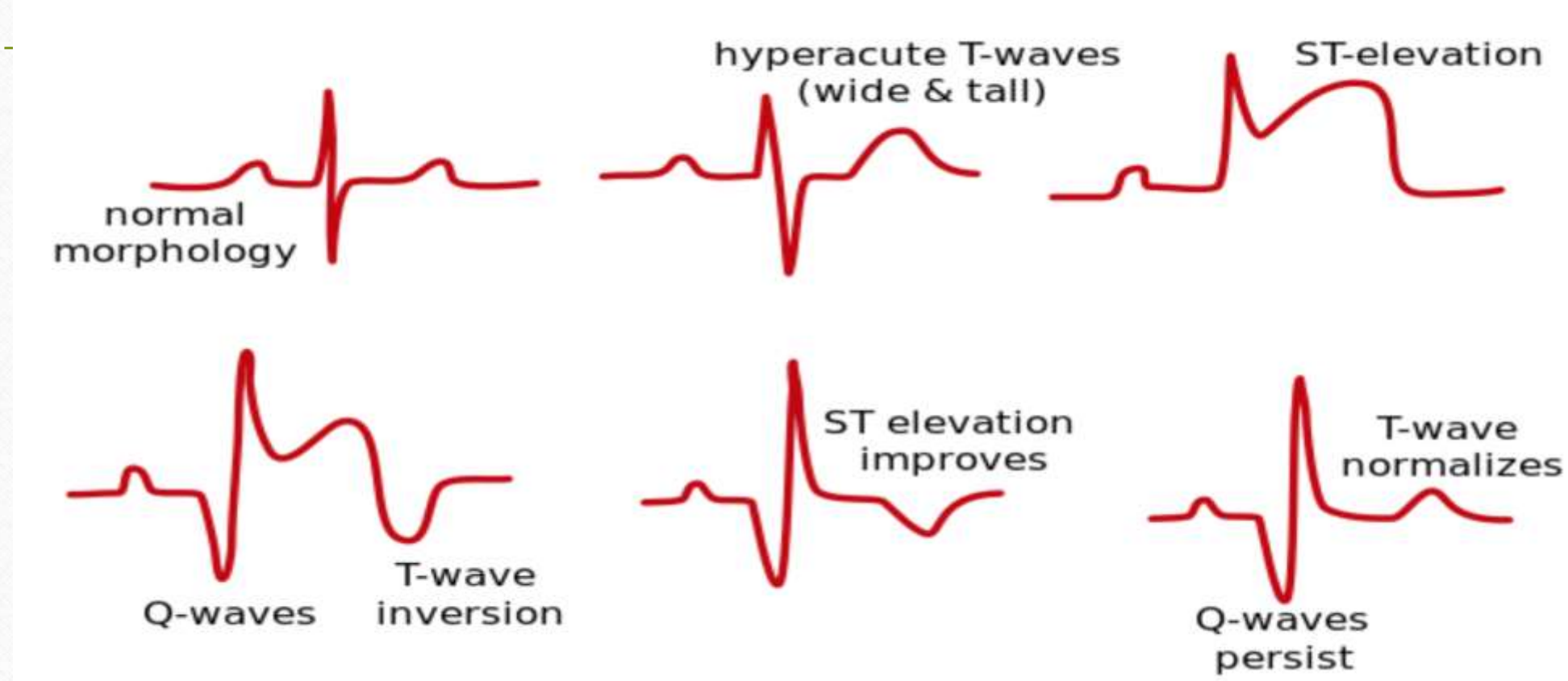
ECG in Myocardial Infarction

- **T wave inversion**
- **ST elevation**
- **Deep Q wave**

ECG MI



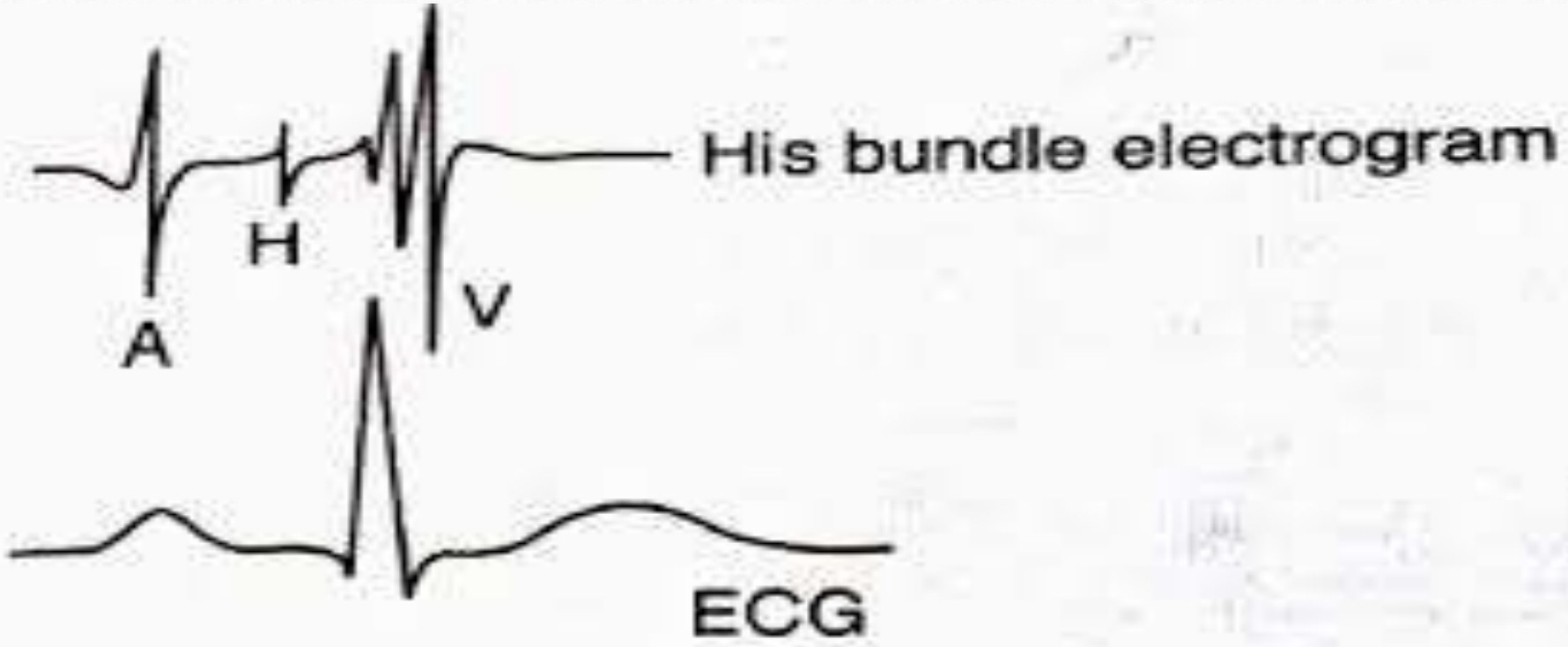
Post MI Changes



His Bundle Electrogram (HBE)

- A deflection
- H spike
- V deflection
- Used to locate AV conduction blocks.

His Bundle Electrogram



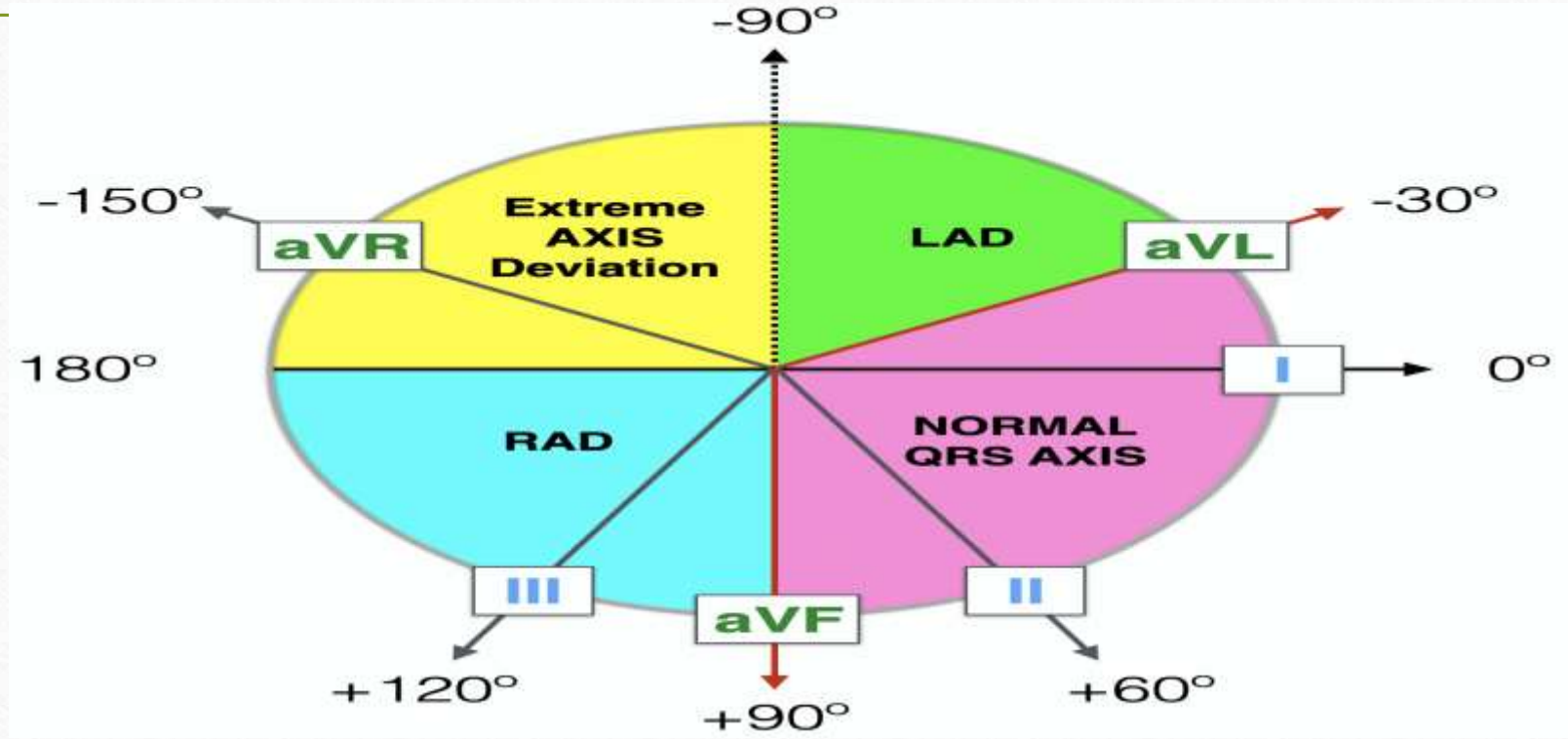
Vector & Vectorcardiogram

- **Vector**
- Arrow indicating direction & magnitude of electrical current
- **Resultant Vector**
- From depolarized base (–) → polarized apex (+)
- **Vectorcardiogram**
- Loop showing changes during cardiac cycle

Mean Electrical Axis

- **Definition**
- Preponderant direction of ventricular depolarization
- **Normal value**
- **+59°**
- **Axis Deviations**
- **Left Axis Deviation**
- Expiration, Lying down,Obesity

ECG Axis Interpretation



Mean Electrical Axis

- Obesity
- LV hypertrophy
- LBBB
- **Right Axis Deviation**
- Inspiration
- Standing
- Tall person
- RV hypertrophy
- RBBB

Cardiac Arrhythmias

- **Normal Sinus Rhythm**
- SA node origin
- **Bradycardia**
- HR < 60/min

Bradycardia

Found In

- **Sleep**, **Athletes** (increased vagal tone)
- **Vagal stimulation**, **Carotid sinus syndrome**
- **Additional High-Yield**
- Seen in hypothyroidism, **Drugs:** β-blockers, Ca^{++} channel blockers, digitalis
- May indicate conduction disease (SA/AV node dysfunction)

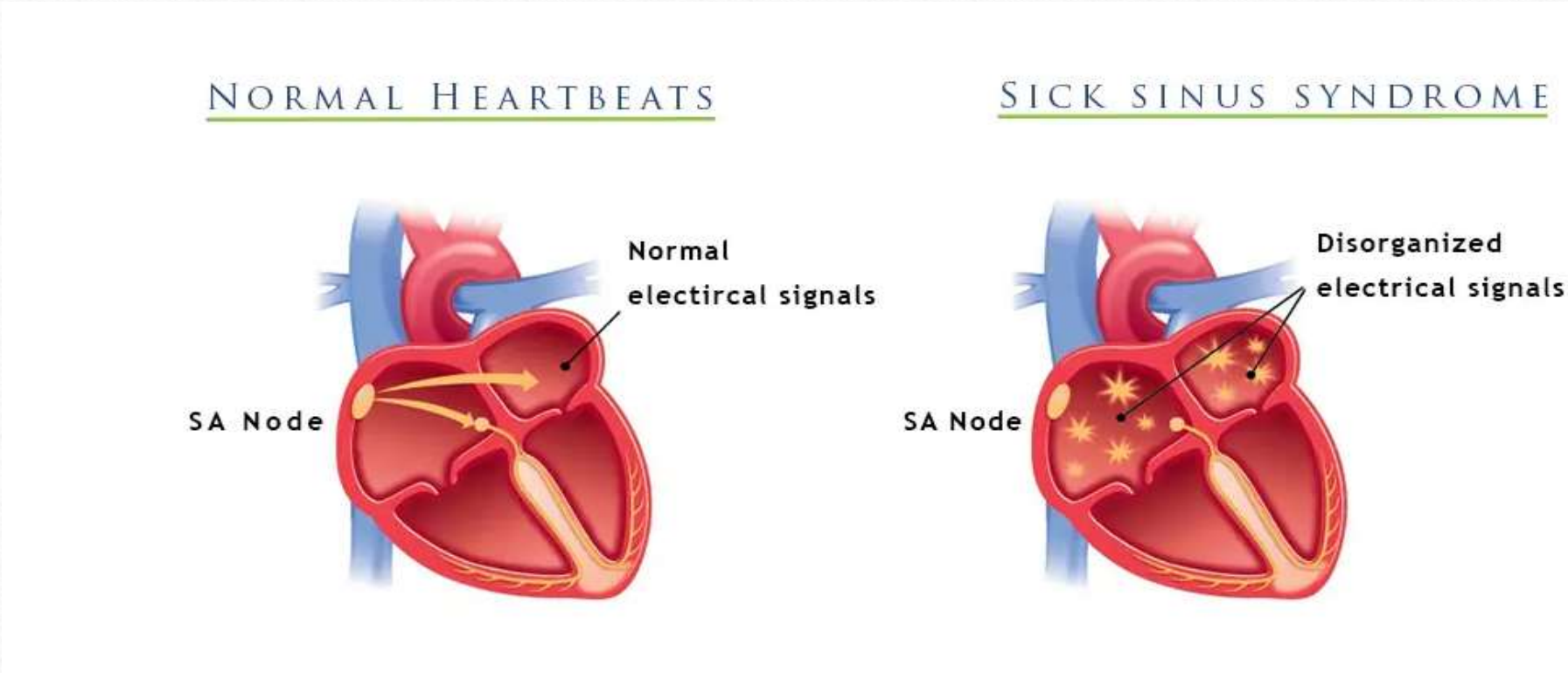
Tachycardia - HR > 100 beats/min

- **Found In**
- Fever, Emotion, Exercise
- Sympathetic stimulation
- **Additional High-Yield**
- Hyperthyroidism, hypovolemia, anemia
- Pain, anxiety
- Early heart failure

Sick Sinus Syndrome

- **Definition**
- Dysfunction of SA node → **Bradycardia**, dizziness, syncope.
- **Additional Points**
- May alternate with tachycardia (“tachy-brady syndrome”)
- Seen in elderly due to fibrosis of SA node
- Often requires pacemaker

Sick Sinus Syndrome



Cardiac Arrhythmia Overview

- **Definition**
- Variation in normal rhythm OR abnormal rhythm.
- **Causes**
- Abnormal rhythmicity of pacemaker
- Shift from SA node to another pacemaker
- Conduction blocks
- Abnormal pathways
- Spontaneous ectopic impulses

Re-Entry / Circus Movement

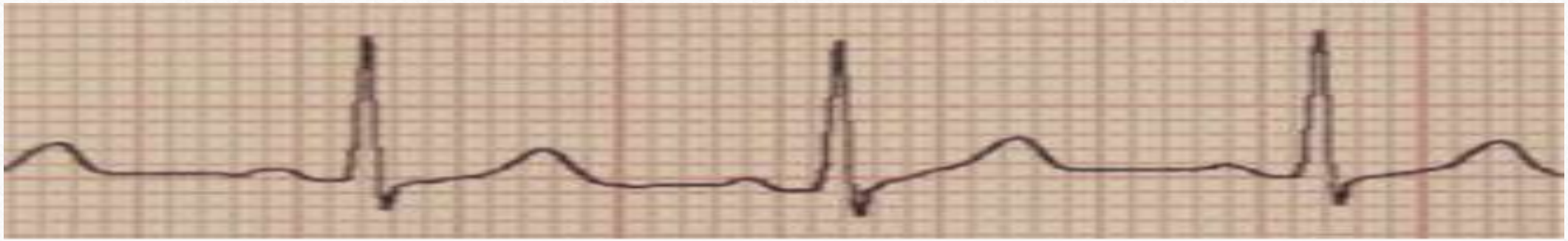
- **Definition**
- Cardiac impulse travels continuously around a loop of tissue → arrhythmia.
- **Characteristics**
- HR 200–300 beats/min
- Contraction still coordinated (in flutter)

Re-Entry / Circus Movement

- **Causes**
- Long pathway (dilated heart)
- Slow conduction (Purkinje block)
- Short refractory period (epinephrine)
- **Example**
- Atrial flutter
  → Synchronous circular movement in dilated atria (valvular disease)

Flutter vs Fibrillation

- **Flutter**
- 200–300/min
- Coordinated but too fast
- Often progresses to fibrillation
- **Fibrillation**
- Rapid, **incoordinated**
- No effective pumping
- Types: Atrial & Ventricular



(a) Normal sinus rhythm

(b) Atrial fibrillation

(c) Atrial flutter

Causes

- **Causes of Atrial Fibrillation**
- Atrial over-dilation (flutter → fibrillation)
- Hypertension, valvular disease
- Hyperthyroidism
- **Causes of Ventricular Fibrillation**
- Ventricular dilation
- Purkinje block
- Myocardial ischemia/infarction

Ectopic Focus

- **Definition**
- An over-excitable spot producing premature impulses.
- **Causes**
- Re-entry signals, Local ischemia, Toxic irritation (caffeine, nicotine)
- Lack of sleep, anxiety,Calcified plaques
- **Produces**
- Premature contractions (extrasystole)

Premature Contractions + Compensatory Pause

- **Premature Contraction**
- Contraction before normal systole → **extra beat**
- Most commonly due to ectopic focus
- **Compensatory Pause**
- Interval between premature & next normal contraction is prolonged.
- **Additional Note**
- Common in stress, stimulants, ischemia

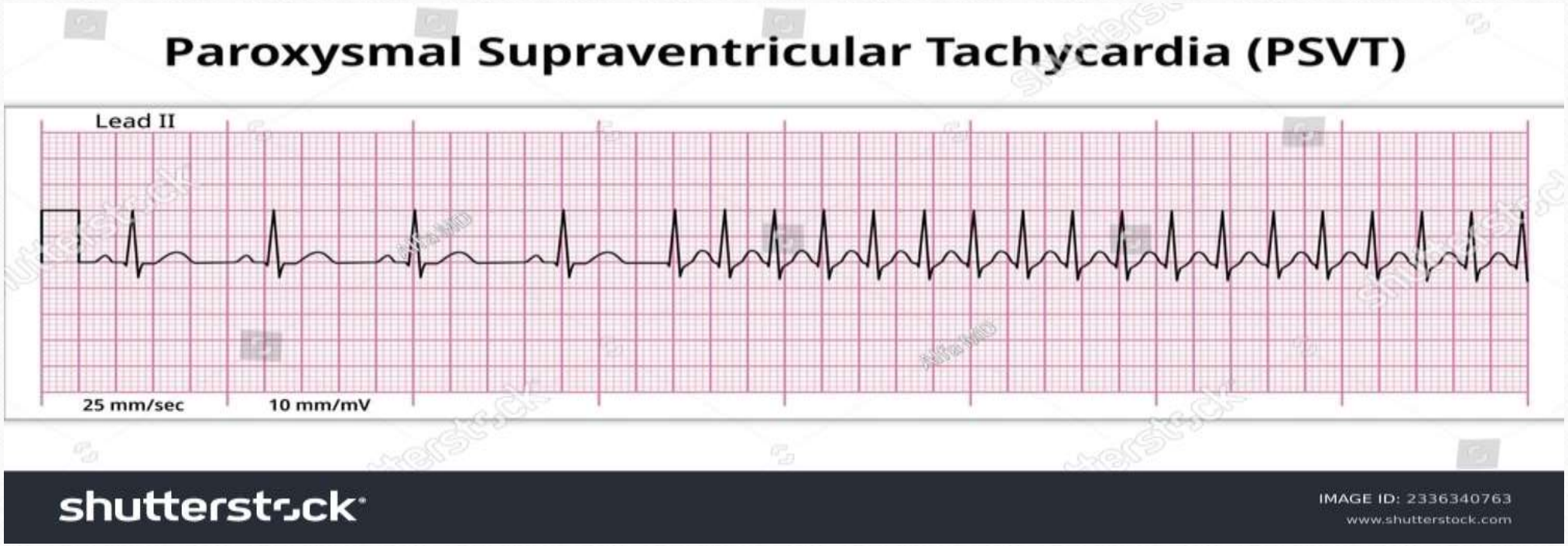
Paroxysmal Tachycardia

- **Definition**
- Sudden onset of very rapid HR occurring in paroxysms.
- **Mechanism**
- Irritable focus acts as temporary pacemaker due to re-entry
- Emits rapid impulses
- Then reverts back to SA node

Paroxysmal Tachycardia

- **Types**
- Atrial
- AV nodal (SVT)
- Ventricular
- **HR**
- 150–220 bpm (atrial/AVN)
- 180–250 bpm (ventricular)

Ecg Paroxysmal Supraventricular Tachycardia



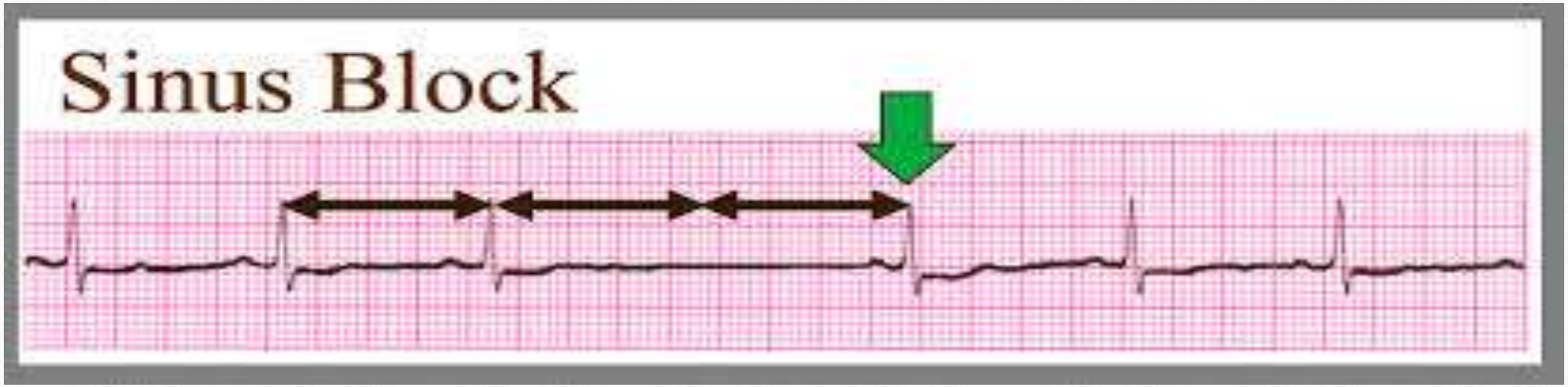
Heart Block (Overview)

- **Definition**
- Block of impulse transmission from SA node to ventricles.
- **Types**
- Sinoatrial block
- AV block
- Bundle branch block
- Arborization block

Sino-Atrial Block

- **Mechanism**
- SA node impulse blocked before entering atria.
- **Characteristics**
- Sudden disappearance of P wave
- Missing one full heartbeat
- AV node may take over rhythm (escape rhythm)

Sino-Atrial Block



Atrioventricular (AV) Block — Causes

- Ischemia of AV node (CAD)
- Compression by scar/calcification
- Myocarditis (diphtheria, rheumatic fever)
- Excess vagal stimulation (carotid sinus)

AV Block Types

- **1) First-Degree Block**
- All impulses are transmitted, PR interval **prolonged > 220 ms**
- **2) Second-Degree Block**
- Some impulses blocked (dropped beats)
- **Mobitz I (Wenckebach):** Progressive PR lengthening → dropped QRS
- **Mobitz II:**Sudden dropped QRS, No PR prolongation
- More dangerous (often needs pacemaker)

Third-Degree Block (Complete Block)

- No impulses reach ventricles
- Atria & ventricles beat independently
- P waves > QRS waves
- Requires pacemaker

Dr Lal PathLabs

What is Heart Block?

Normal

Fist-Degree AV Block

Second-Degree AV Block(2:1)

Third-Degree AV Block

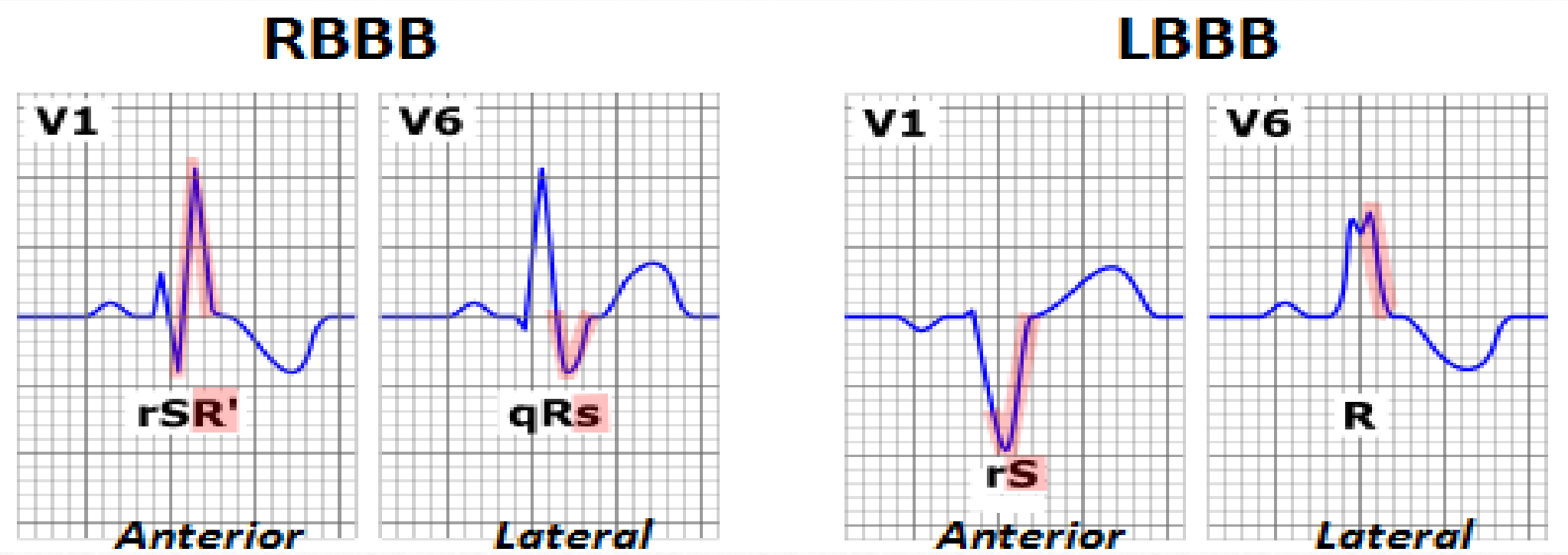
Arborization Block

- **Definition**
- Impulse blocked in Purkinje fiber arborization.
- **Found In**
- Chronic myocardial damage (scarring)
- Cardiomyopathies

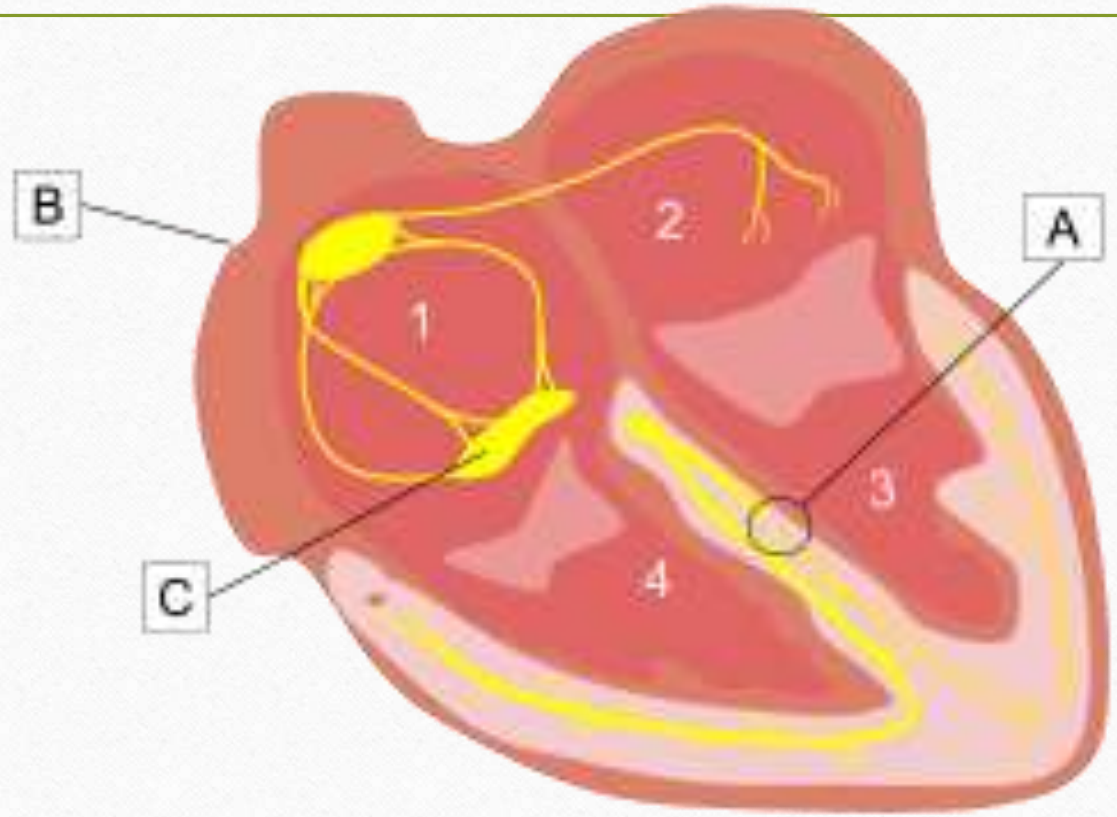
Bundle Branch Block (Right/Left)

- **Definition**
- Impulse blocked in either bundle branch.
- **Features**
- Normal ventricle contracts first
- Blocked ventricle contracts later
- Produces splitting of S1
- QRS complex prolonged (>120 ms)

RBB vs LBB


RBBB
V1
rSR'
Anterior
V6
qRs
Lateral
LBBB
V1
rS
Anterior
V6
R
Lateral

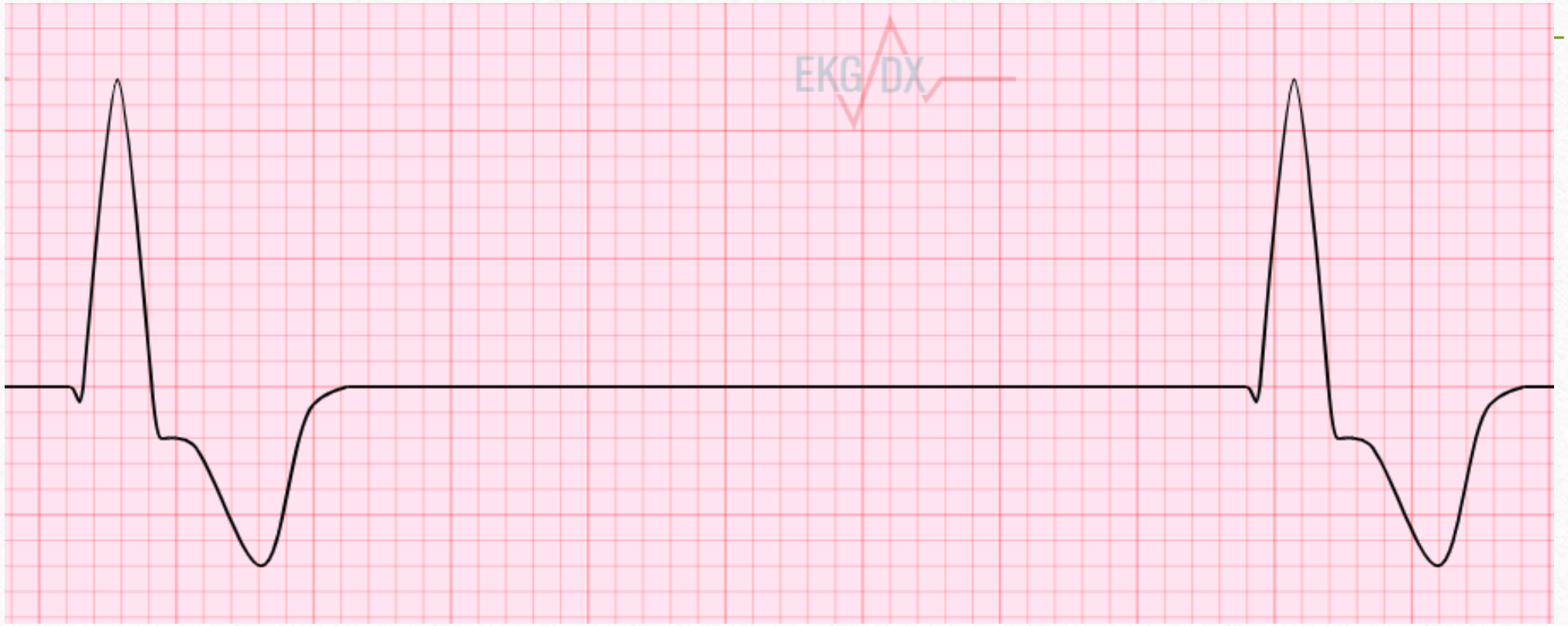
This image shows **bundle branch block** where the beating rhythm is hindered, but not stopped



Ventricular Escape

- **Occurs When**
- Ventricles stop when AV block occurs
- After 4–10 sec → Purkinje fibers become pacemaker
- **Rate**
- 15–40 bpm

Ventricular Escape



Stokes–Adams Syndrome

- **Mechanism**
- In complete AV block → ventricles stop
- Cerebral ischemia for 5–30 sec
- Patient faints (syncope)
- **May Cause**
- Seizure-like movements
- Sudden death if prolonged

Cardiac Arrest

- **Definition**
- Sudden cessation of rhythmic contractions.
- **Causes**
- Severe hypoxia
- Deep anesthesia
- SA node ischemia
- Severe myocardial disease

Dextrocardia

- **Definition**
- Heart located on right side
- Apex pointing right
- **Additional**
- May be part of situs inversus
- ECG leads must be reversed for interpretation

Chest X-ray


Any
Question?


Thank You