Electrocardiography (ECG)

DR. AMIR SAMADIAN M.D.

DEPARTMENT OF PHYSIOLOGY

SPRING 2020

Electrical activation of Heart

In order for the Heart to pump blood :

1. **Ventricles must be electrically activated**
2. **Contract**

For that :

Generation of **Action Potential** in **Pacemaker cell (SA node)** → conduction and propagation of AP by **specialized conductive tissue (GAP junctions!)**→ Excitation/**depolarization of contractile cardiomyocytes** → **Contraction of cardiac muscle cells**

2 major type of cardiac cells

1. Conductive cells (pacemaker cells) :

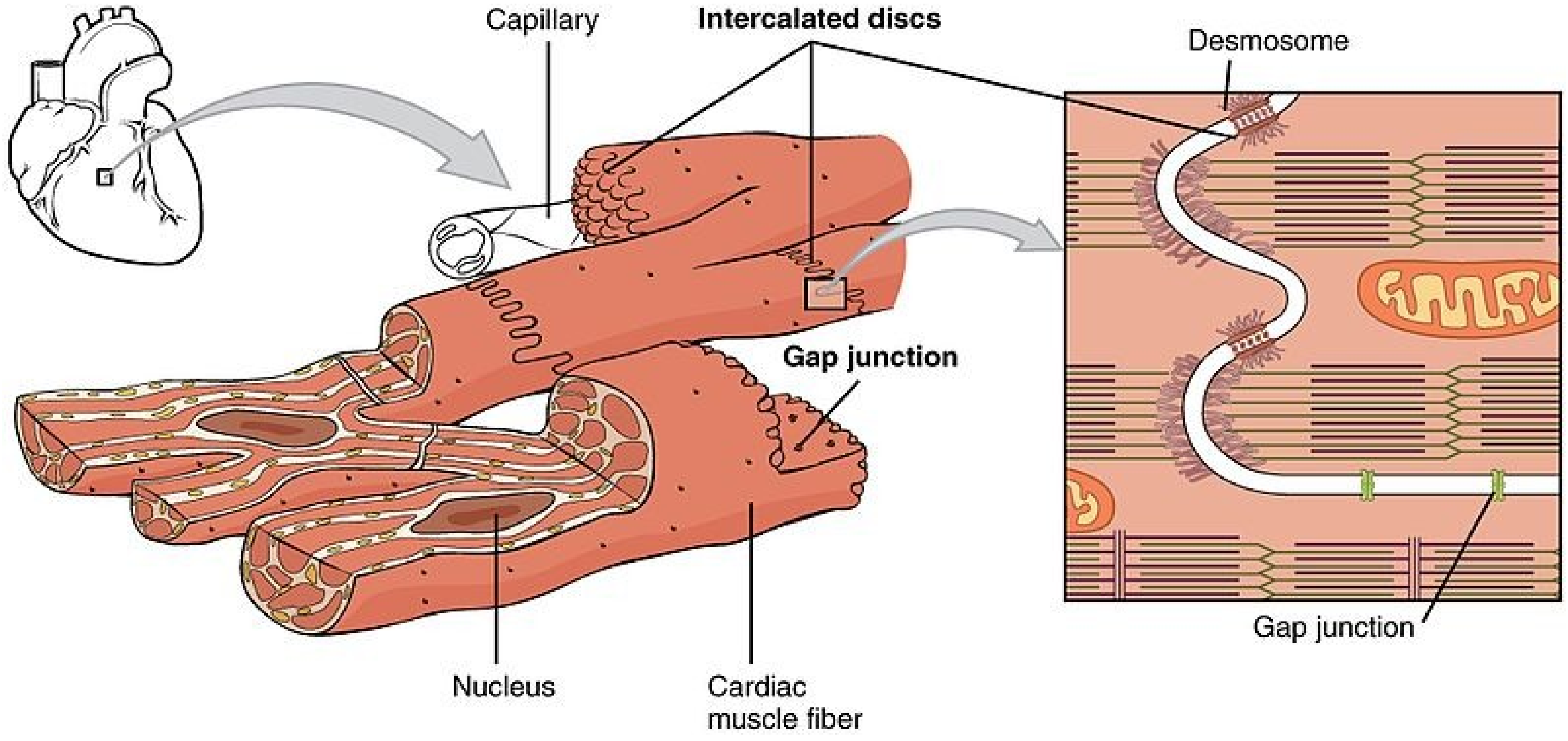
-Automatic excitation/Generation of AP (pacemaker activity)

-Conduction of excitation

2. Contractile cells :

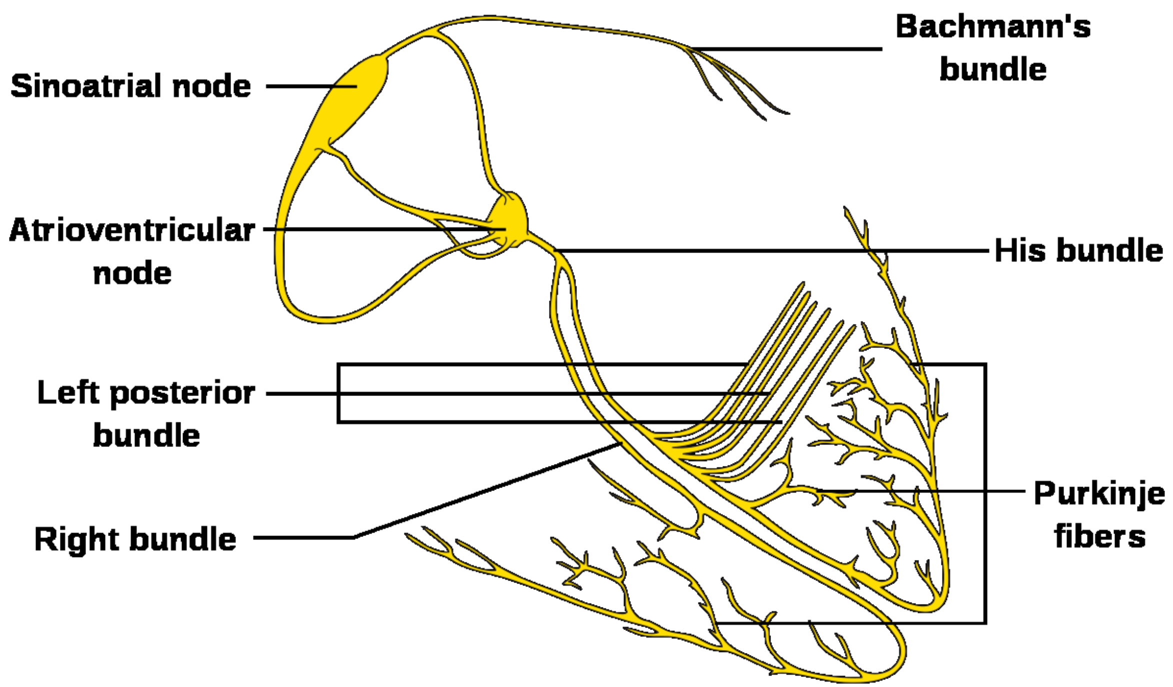
-Contraction & generation of force (Atrial & ventricular working cardiomyocytes)

Cardiac cells (Gap Junctions)

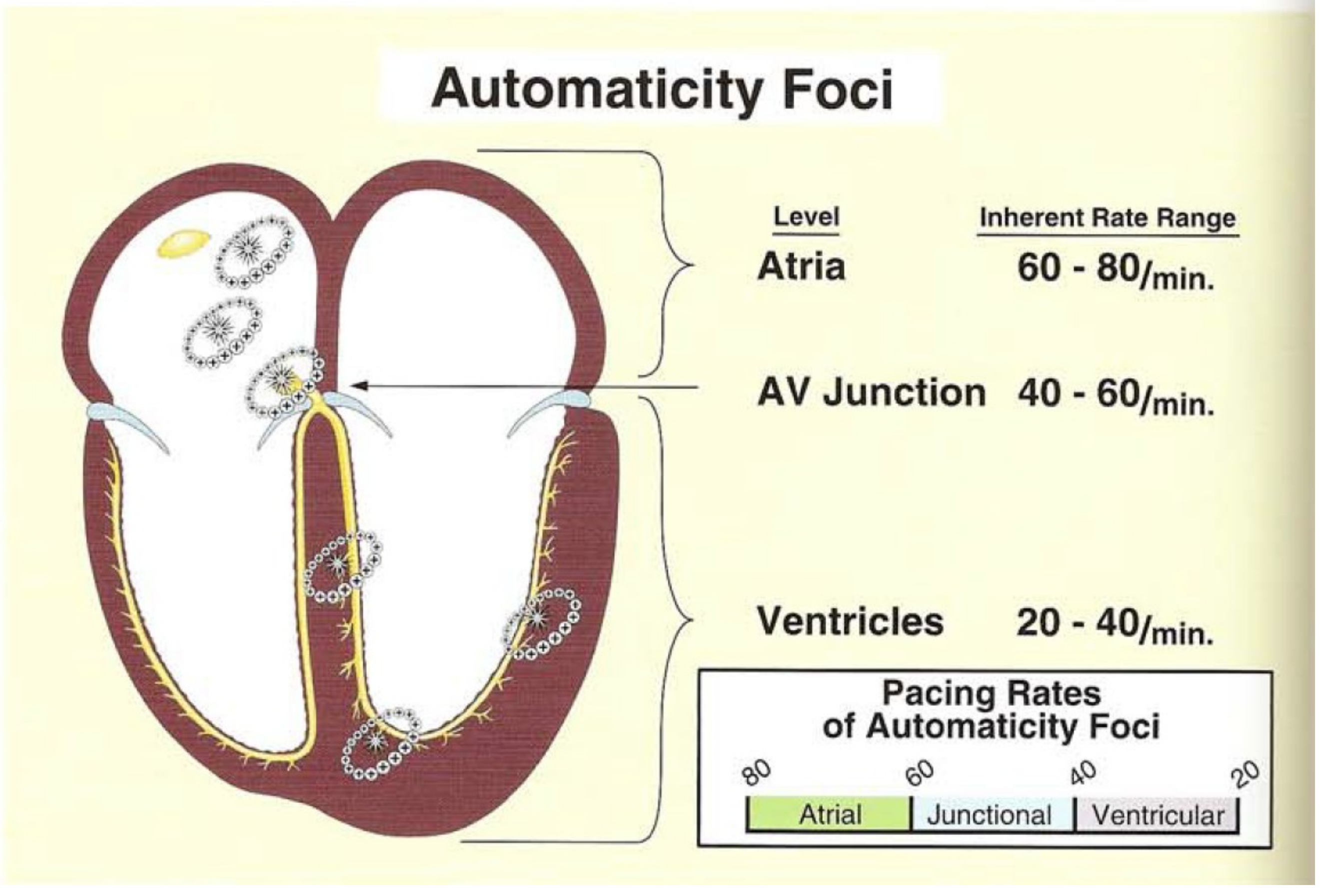


Cardiac conductive cells

1. Sinoatrial node (SA NODE) → **main pacemaker (Automaticity!) → 60-100 bpm**
2. Atrioventricular node (AV NODE)
3. Bundle of his
4. L + R bundle branches
5. Purkinje system



Pacemaker cells

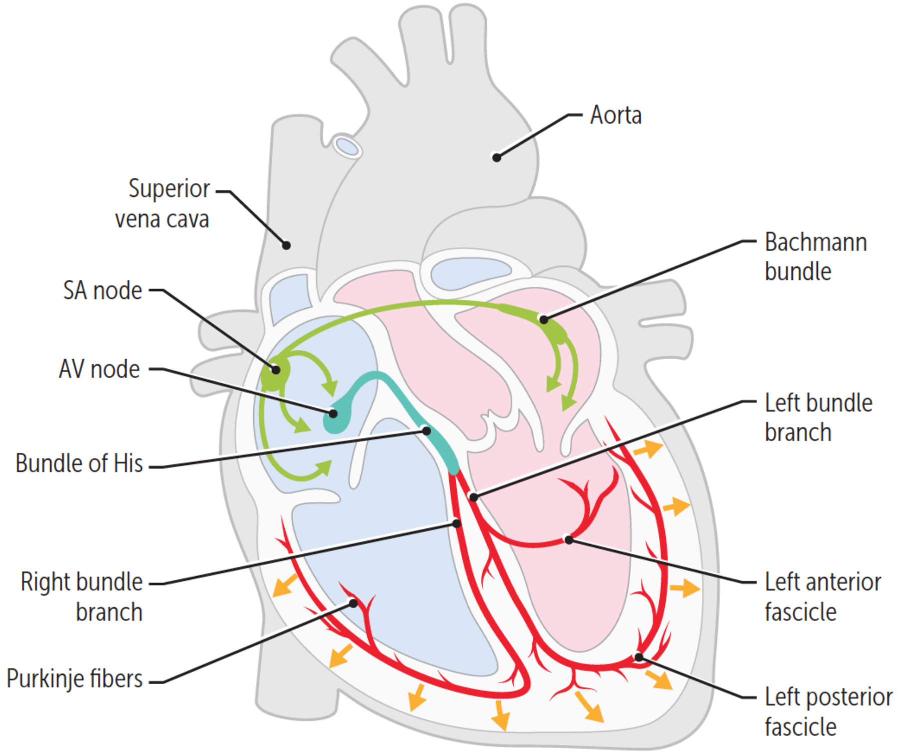


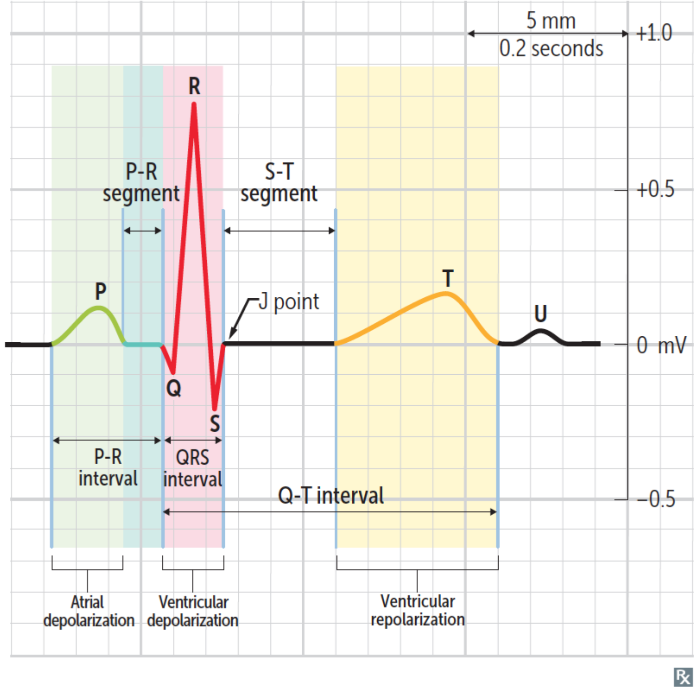
Conductive pathway

Conduction pathway: SA node → atria → AV node → bundle of His → right and left bundle branches → Purkinje fibers → ventricles; left bundle branch divides into left anterior and posterior fascicles.

Pacemaker rates: SA > AV > bundle of His/ Purkinje/ventricles.

Speed of conduction: His-Purkinje > Atria > Ventricles > AV node. He Parks At Ventura Avenue.





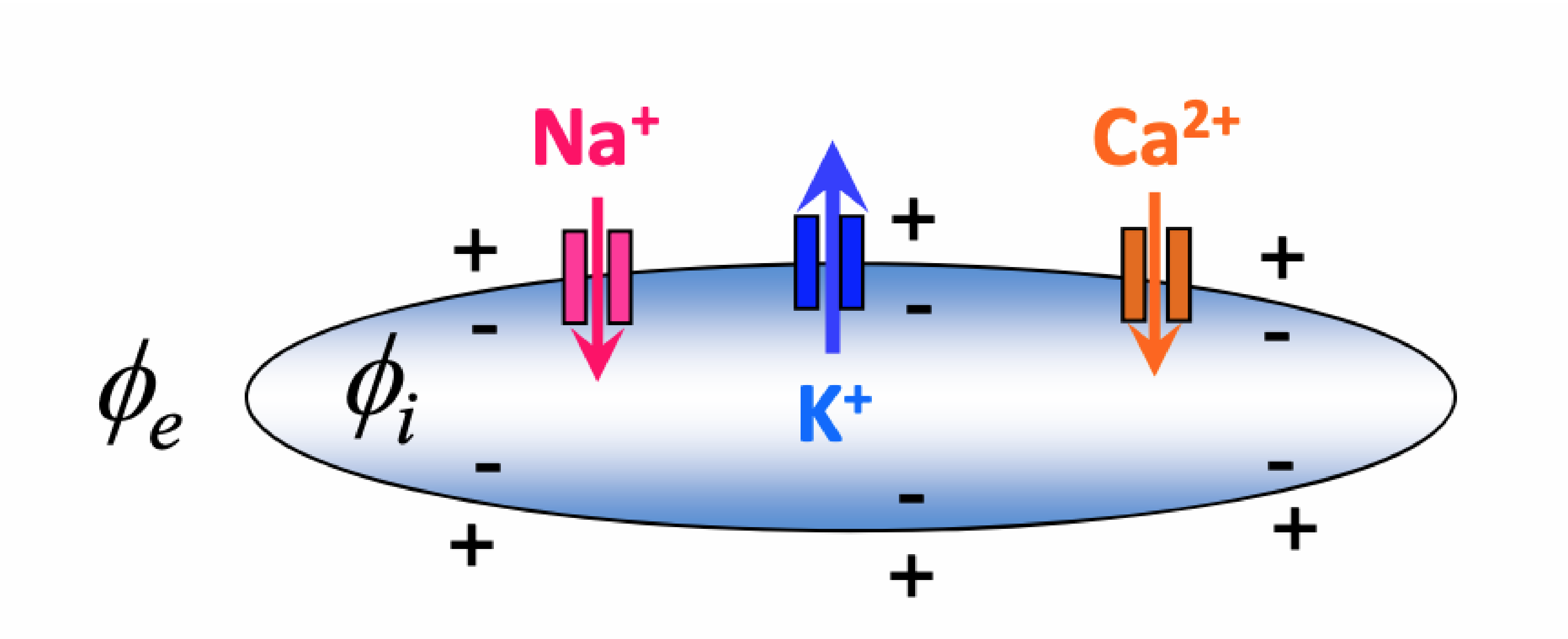
CONDUCTION VELOCITY

- Reflects the time required for excitation to spread throughout cardiac tissue.
- Depends on the **size of the inward current during the upstroke** of the action potential. The larger the inward current, the higher the conduction velocity.
- Conduction fastest in the Purkinje system.
- Conduction is **slowest in the AV node** (seen as the PR interval on the ECG), allowing time for **ventricular filling** before ventricular contraction. If conduction velocity through the AV node is increased, ventricular filling may be compromised.

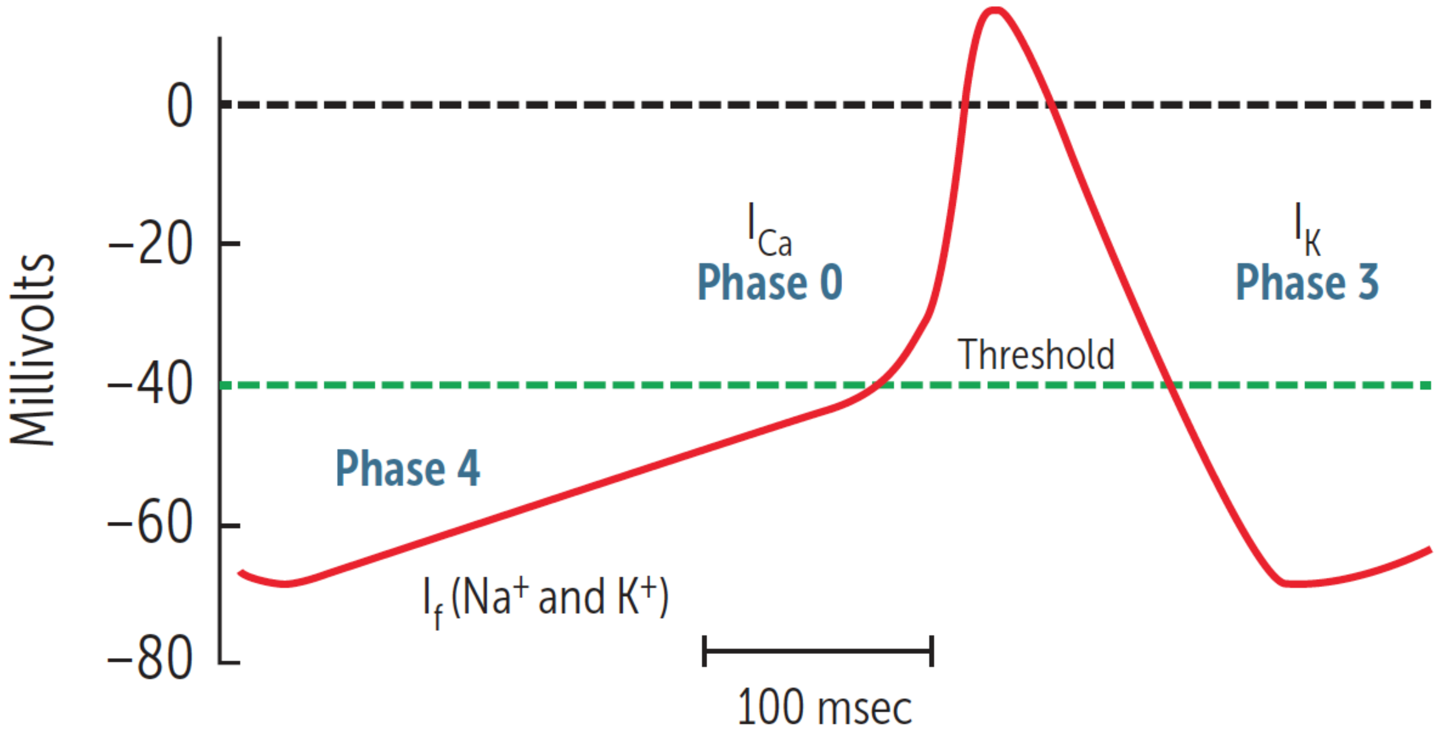
Table 21-4 Conduction Velocity in Different Cardiac Tissues

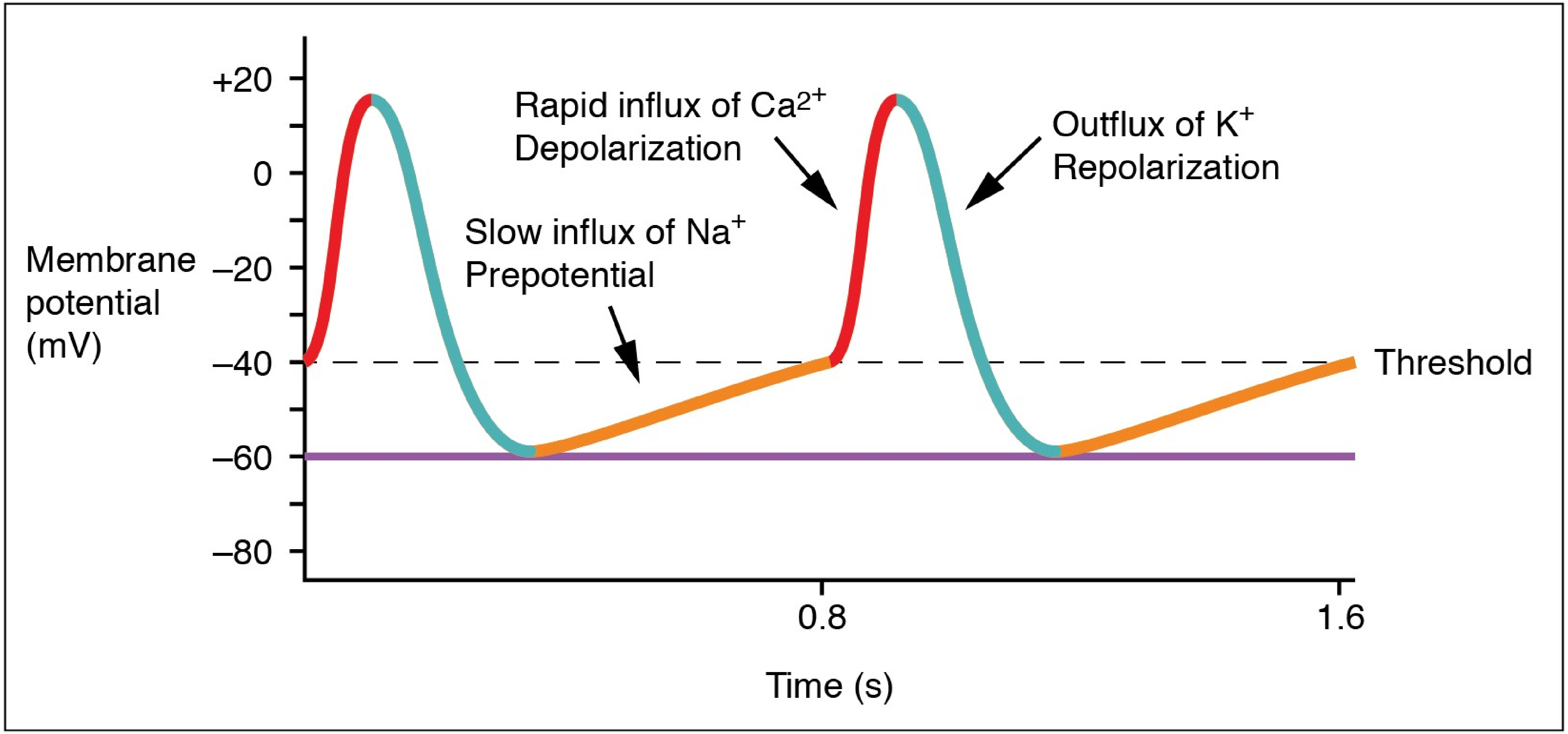
Tissue	Conduction Velocity (m/s)
SA node	0.05
Atrial pathways	1
AV node	0.05
Bundle of His	1
Purkinje system	4
Ventricular muscle	1

Cardiac action potential

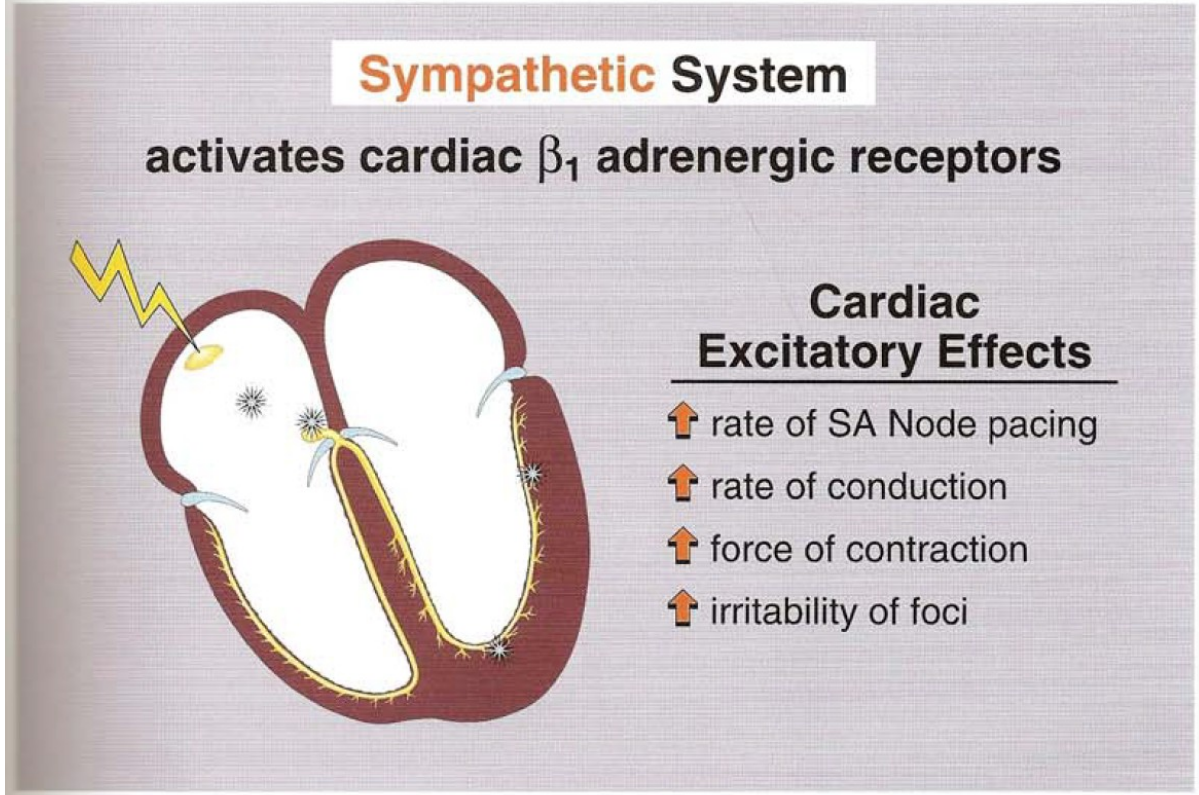


Pacemaker action potential (HIGH YIELD)

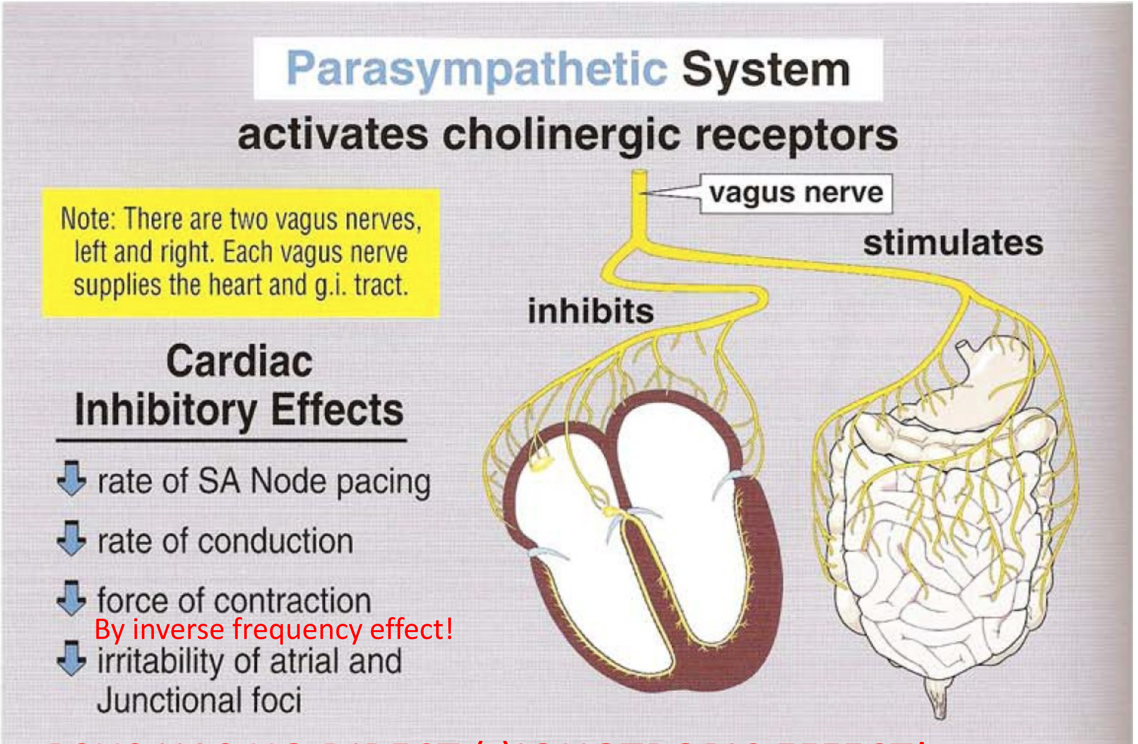



+20
0
–20
–40
–60
–80
Membrane
potential
(mV)
Rapid influx of Ca2+
Depolarization
Outflux of K+
Repolarization
Slow influx of Na+
Prepotential
Threshold
0.8
1.6
Time (s)

AUTONOMIC EFFECTS ON HR AND CV



The heart is stimulated by the sympathetic system through its terminal boutons. The boutons deliver N-epi to the β_1 **(adrenergic)** receptors; this activates the β_1 receptors,* producing an *excitatory* response at the cellular level.



PSNS HAS NO DIRECT (-)IONOTROPIC EFFECT!

Parasympathetic nerves release the neurotransmitter acetylcholine (ACh), which activates cardiac cholinergic receptors (most are within the atria) to produce a cardiac *inhibitory* effect. Conversely, the gastrointestinal tract is stimulated by its parasympathetic innervation.

AUTONOMIC EFFECTS ON HR AND CV

Catecholamines

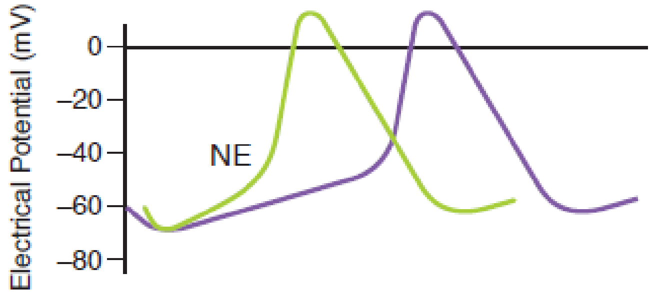


Figure II-3-3. Sympathetic Effects on SA Nodal Cells

- Norepinephrine (NE) from postganglionic sympathetic nerve terminals and circulating epinephrine (Epi)
- β-1 receptors; Gs—cAMP; stimulates opening of HCN and Ca^{2+} channels
- Increased slope of pacemaker potential (gets to threshold sooner)
- Functional effect
  - Positive chronotropy (SA node): increased HR
  - Positive dromotropy (AV node): increased conduction velocity through the AV node

AUTONOMIC EFFECTS ON HR AND CV

Parasympathetic

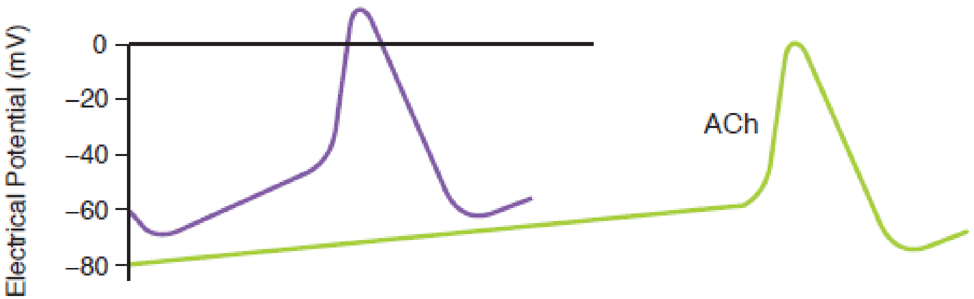
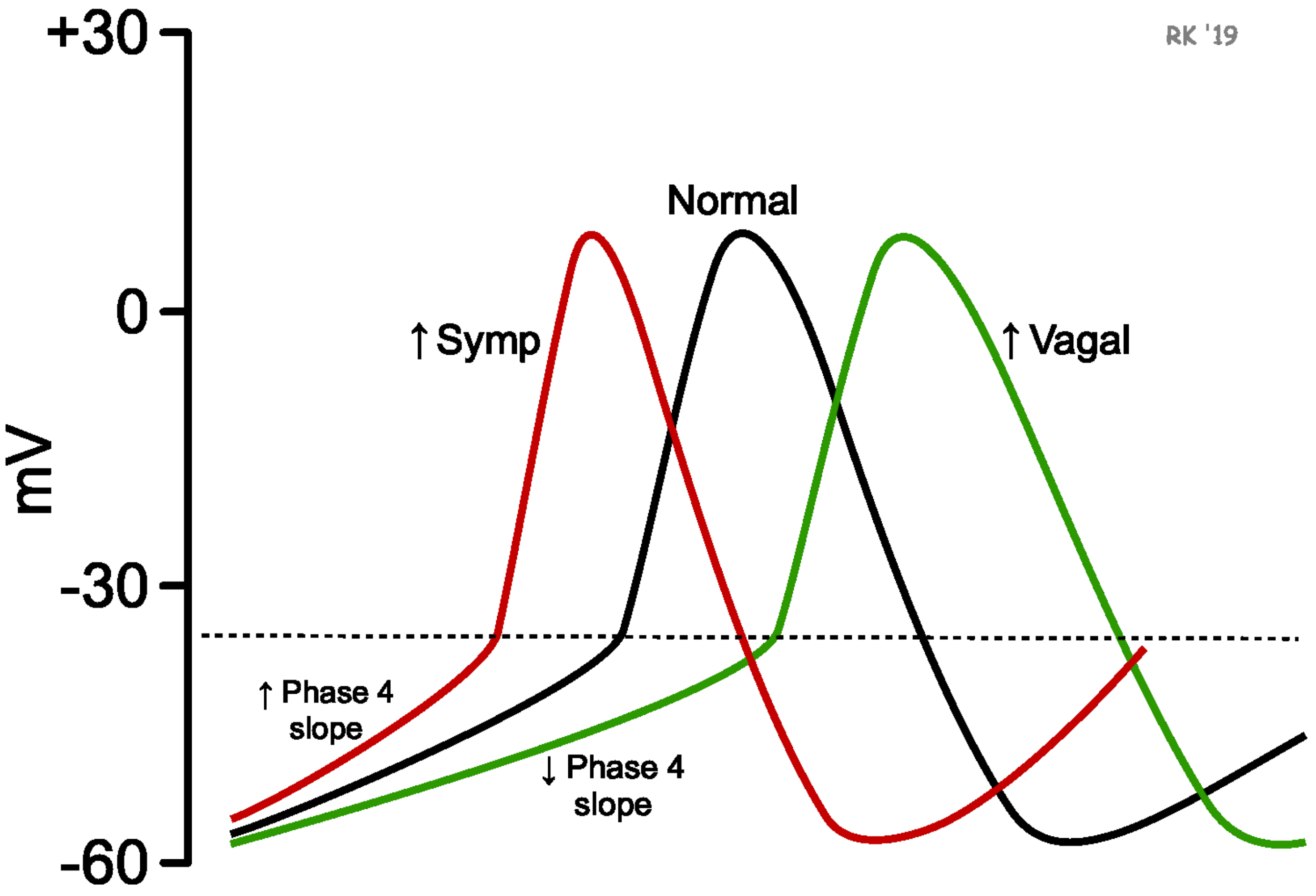


Figure II-3-4. Parasympathetic Effects on SA Nodal Cells

- Ach released from post-ganglionic fibers.
- M_2 receptor; Gi-Go; Opens K^+ channels and inhibits cAMP
- Hyperpolarizes; reduced slope of pacemaker potential
- Functional effect
  - Negative chronotropy (SA node): Decreased HR
  - Negative dromotropy (AV node): Decreased conduction velocity through the AV node

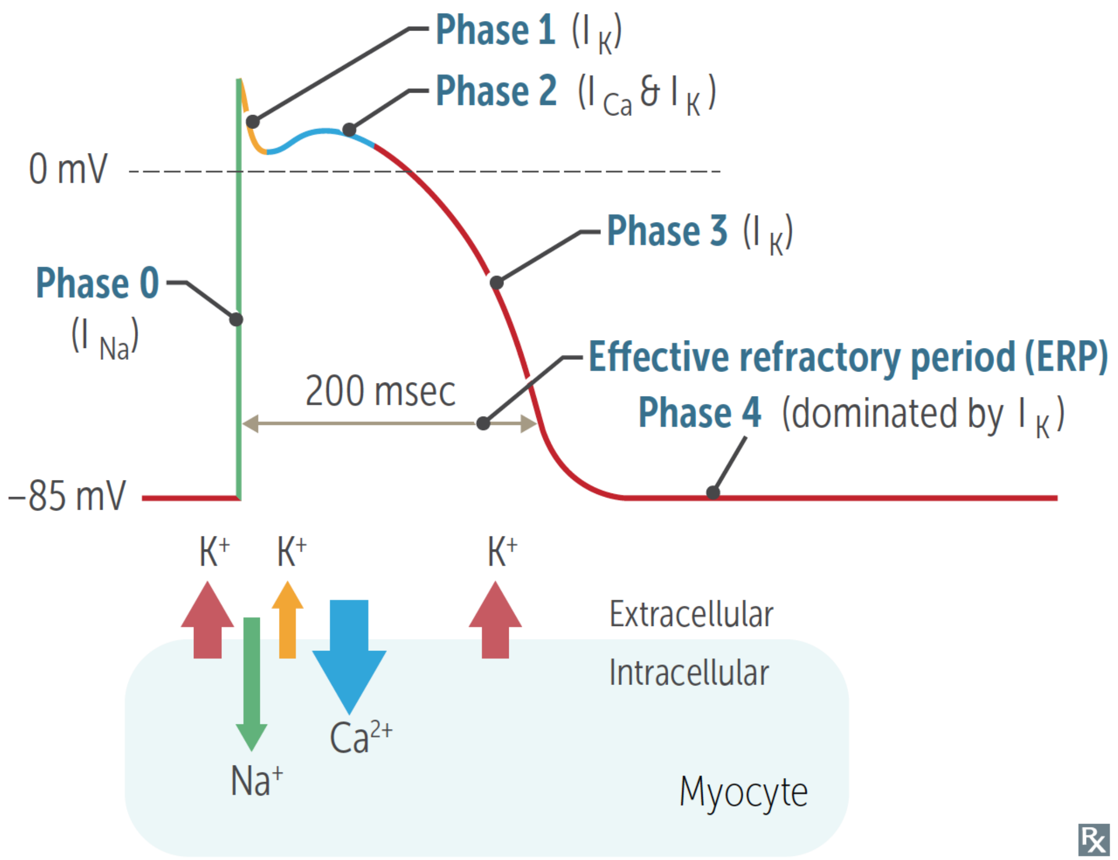
Effect of ANS on Pacemaker cells & HR



AUTONOMIC EFFECTS ON HR AND CV

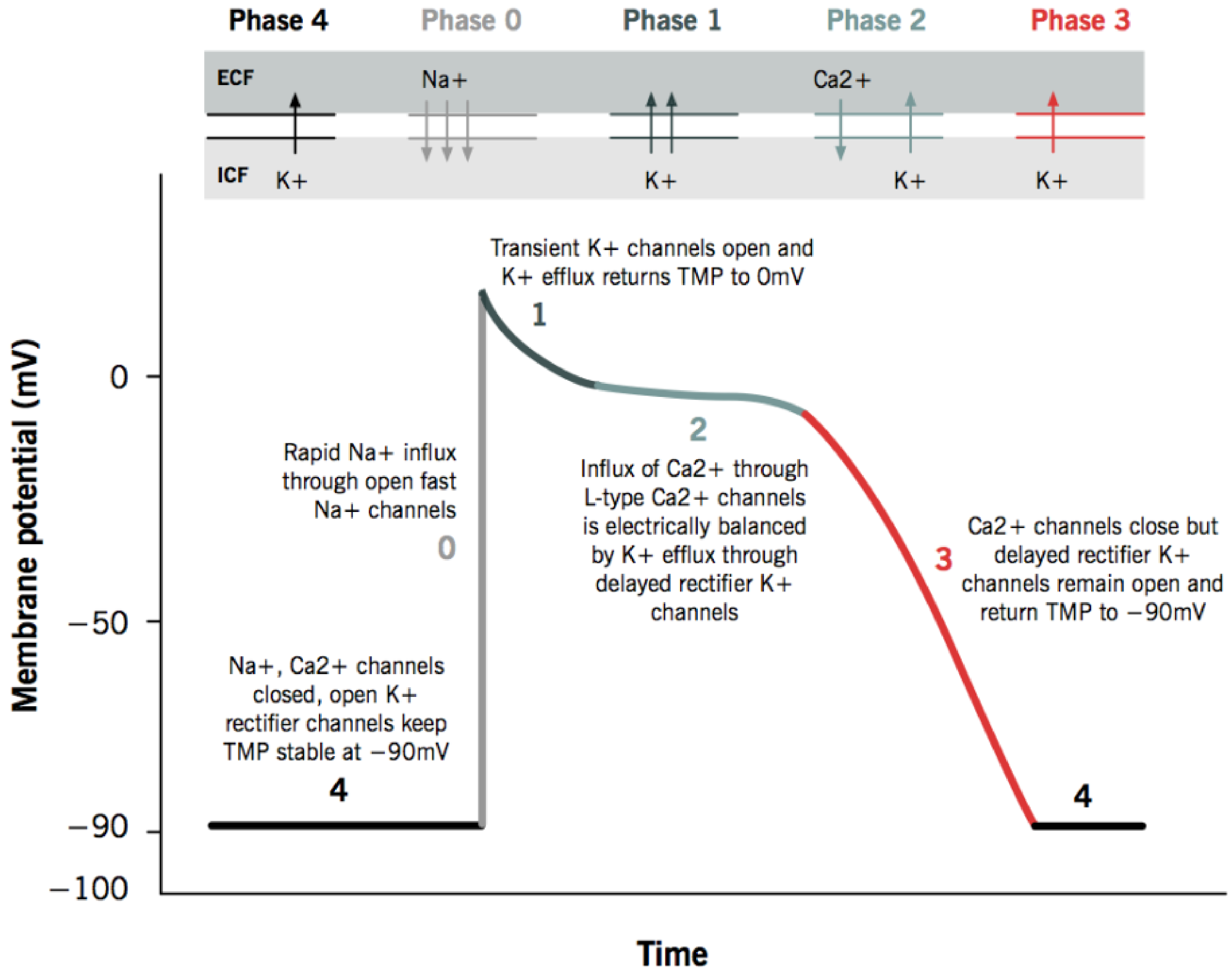
	Sympathetic		Parasympathetic	
	Effect	*Receptor*	*Effect*	*Receptor*
Heart rate	↑	β_1	↓	Muscarinic
Conduction velocity (AV node)	↑	β_1	↓	Muscarinic
Contractility	↑	β_1	↓ (Atria only)	Muscarinic
Vascular smooth muscle				
Skin, splanchnic	Constriction	α_1		
Skeletal muscle	Constriction	α_1		
	Relaxation	β_2		

Contractile cardiomyocytes action potential

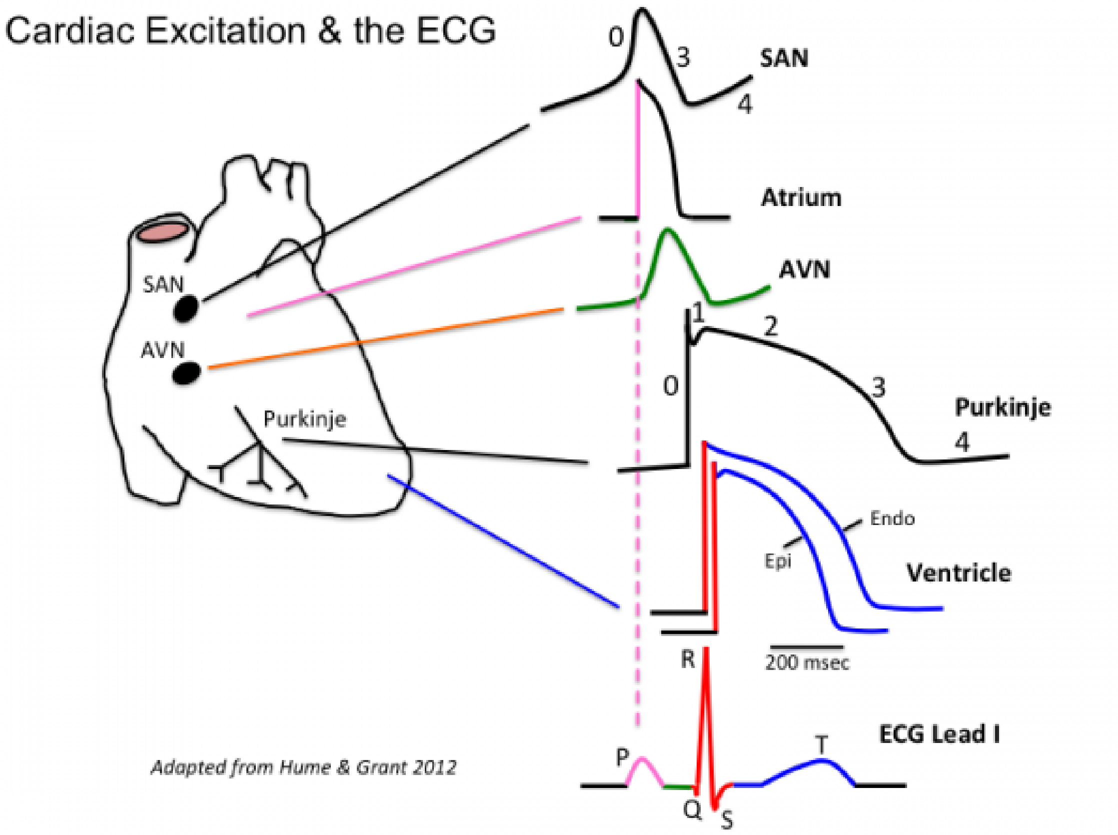


Action potential of cardiac muscles

Grigoriy Ikonnikov and Eric Wong



COMPARISON OF DIFFERENT ACTION POTENTIALS

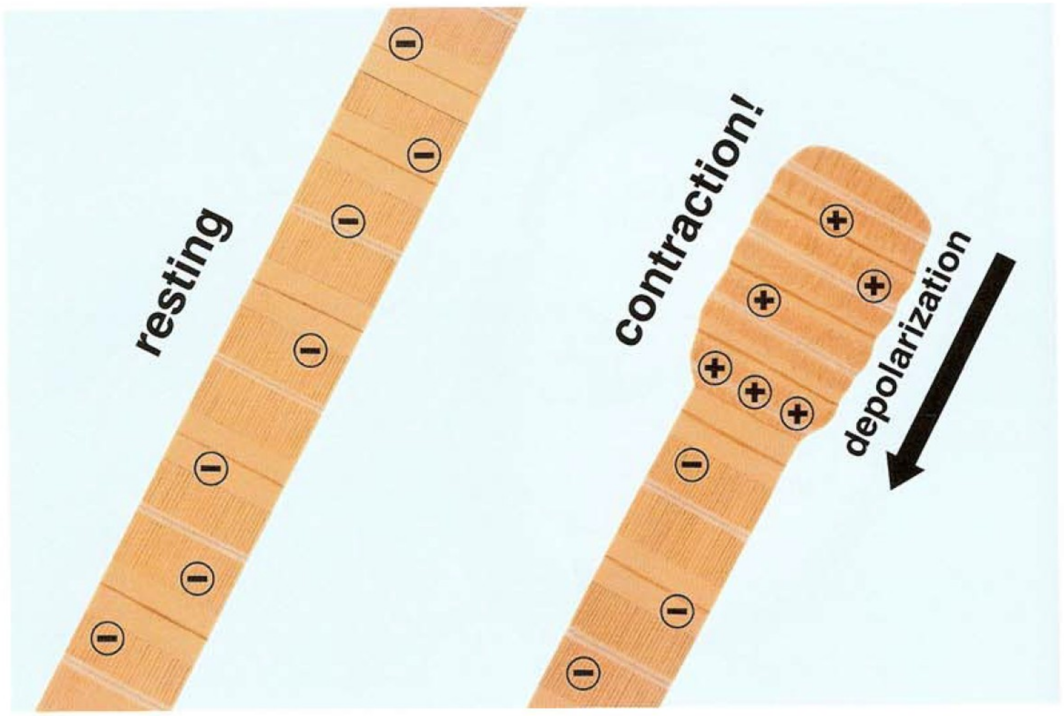


EKG - Basics

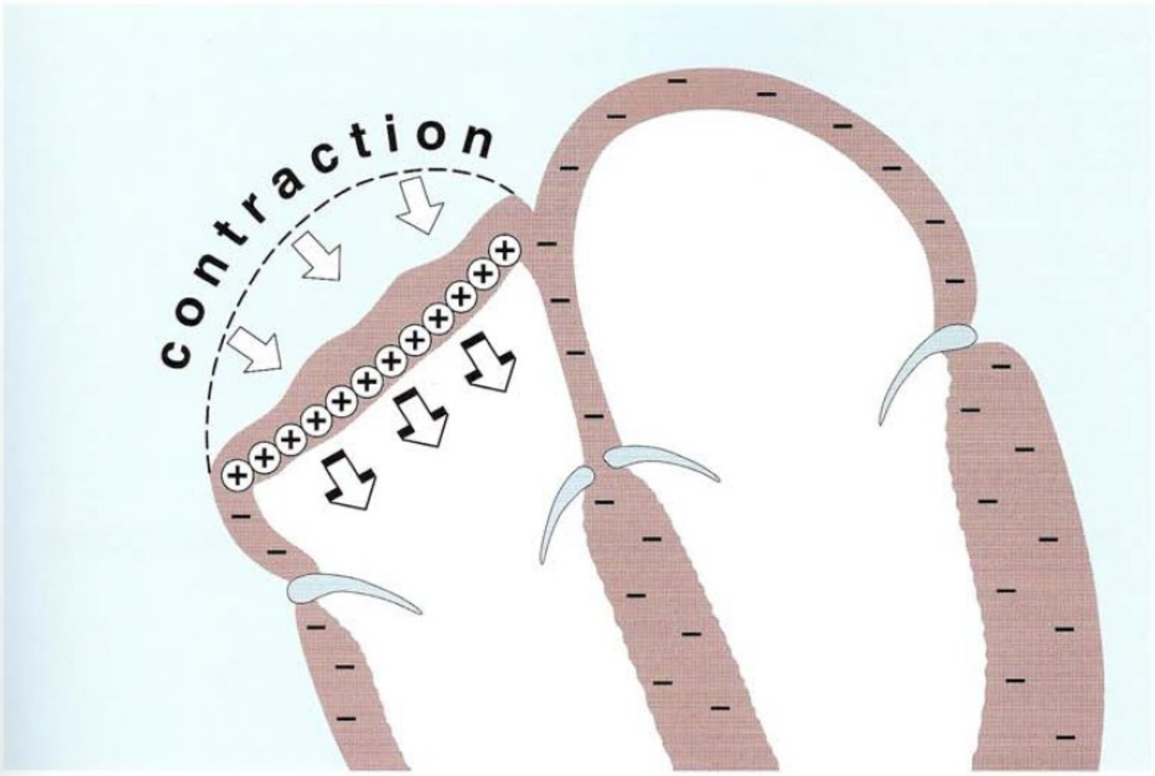
1. When Heart cells go from polarized (-resting state) to depolarized (+action potential) → a **wave of electrical current** is generated
2. this wave of depolarization (current) moves through Cardiac cells → causes contraction !
3. ECG records this electrical activity by mean of **10 skin electrodes** (4limb + 6chest)
4. Electrodes give us **12 real time picture of heart from different angles (12 leads)**
5. Information is recorded on a ECG paper as **Positive(+) or Negative(-) deflection**
6. A wave of **depolarization moving toward** a **positive electrode** produces a **+ deflection.**
7. A wave of **depolarization moving away from** a **positive electrode** records a **- deflection**.

Wave of depolarization

Chapter 1: Basic Principles

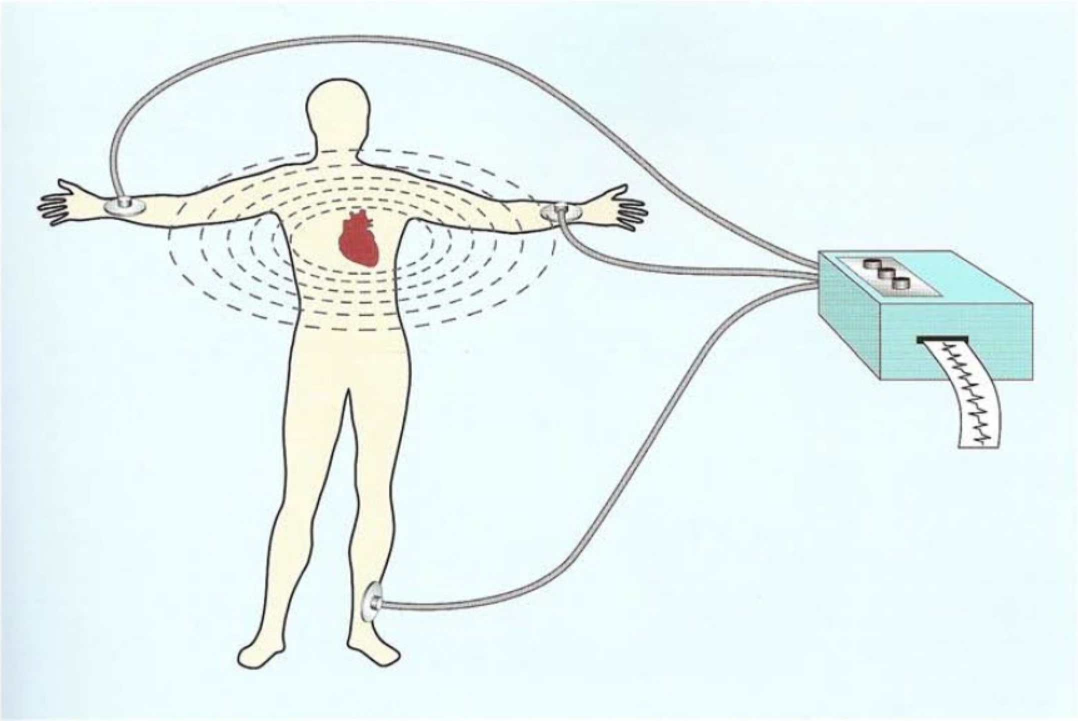


The interiors of heart muscle cells (myocytes*) are negative ("polarized") at rest, but when "depolarized" their interiors become positive and the myocytes contract.

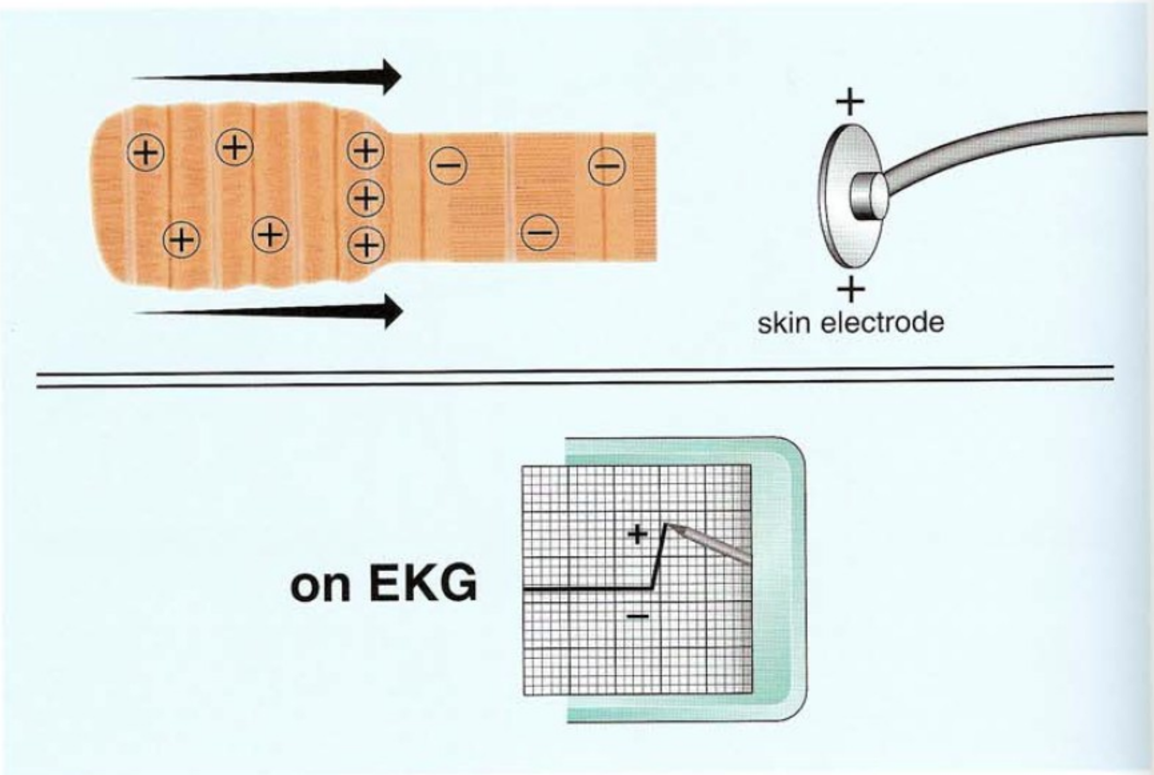


As a wave of depolarization progresses through the heart, it causes contraction of the myocardium.

ECG Electrodes

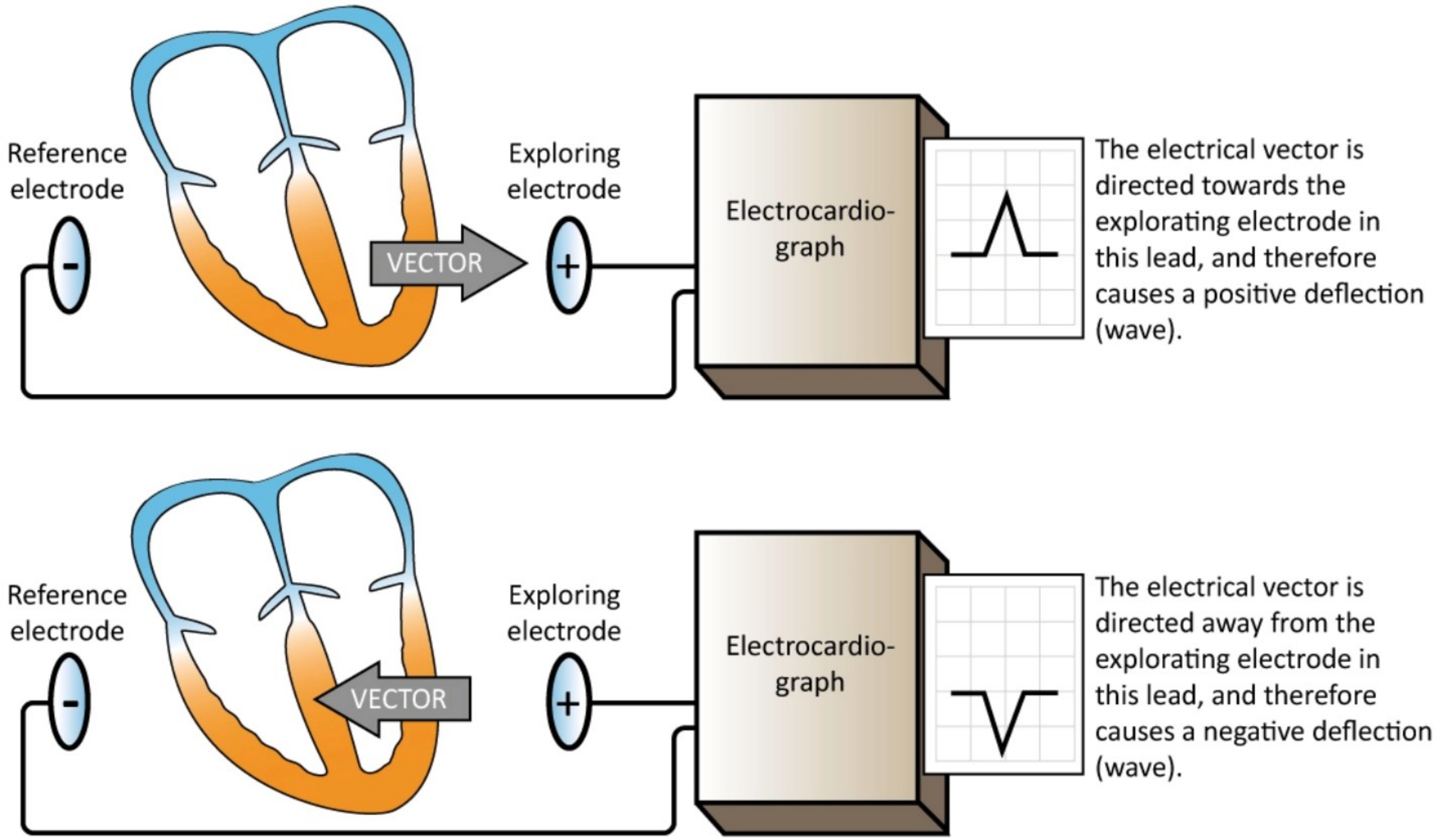


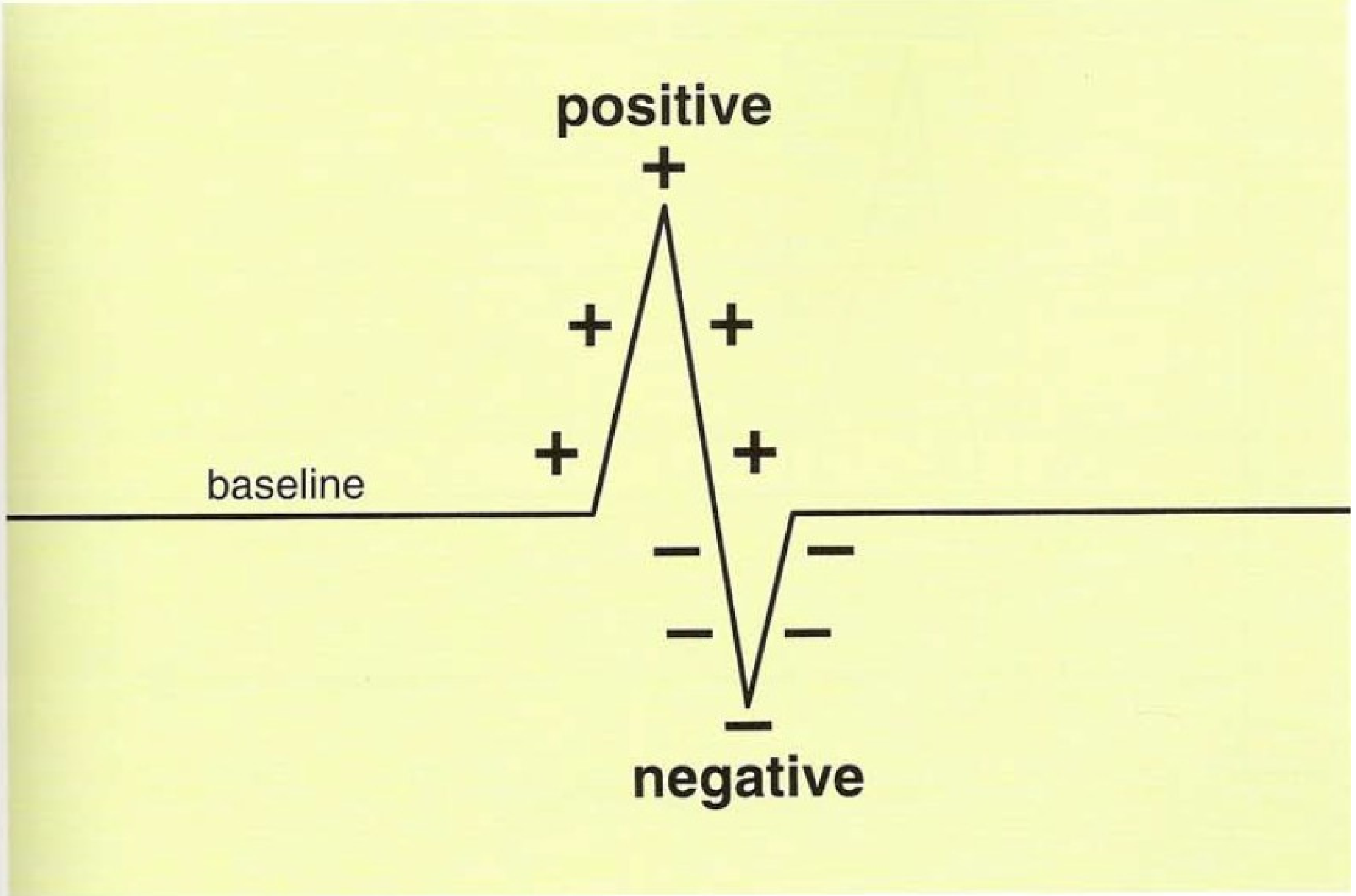
Sensors called "electrodes" are put on the skin to detect the heart's electrical activity. The EKG machine records this activity on moving paper as an electrocardiogram.



As the positive wave of depolarization within the myocytes flows toward a positive electrode, there is a positive (upward) deflection recorded on EKG.

Positive & Negative Deflections on ECG paper



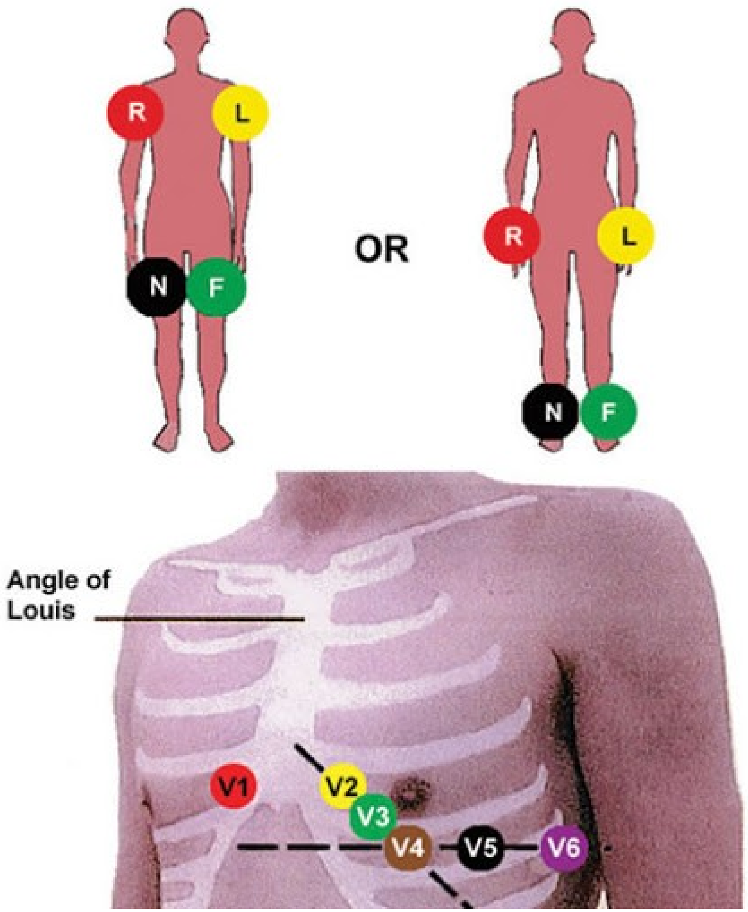


Upward deflections are called "positive" deflections. Downward deflections are called "negative" deflections.

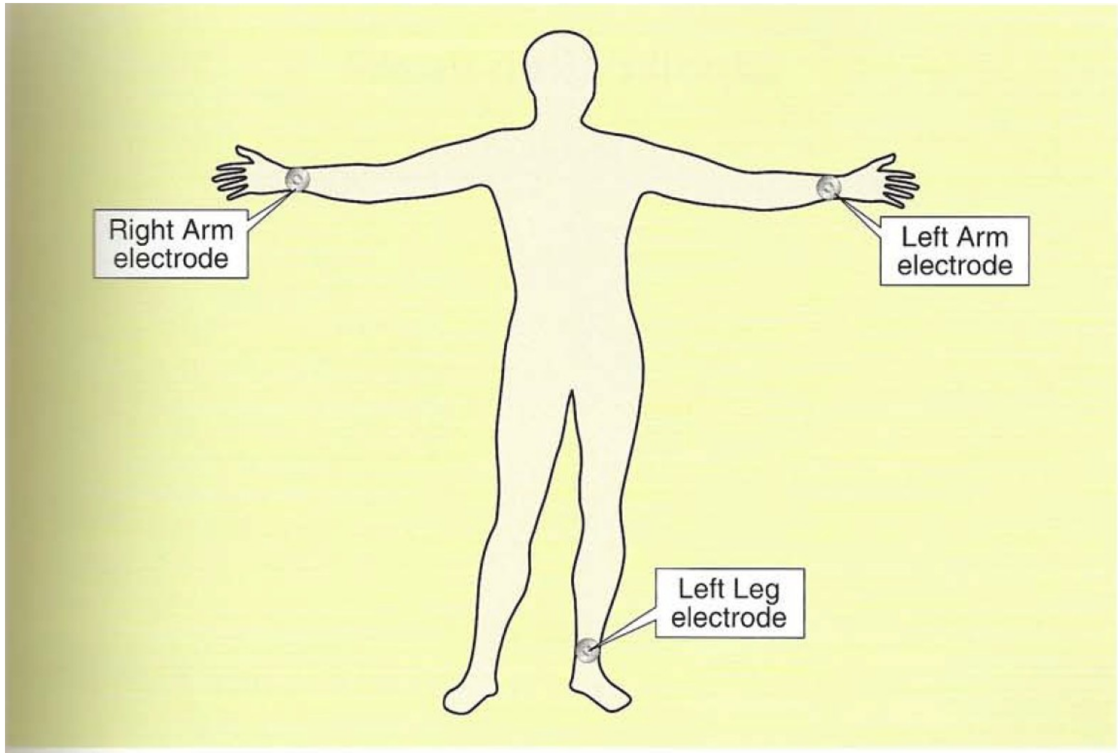
12 lead ECG

1. **6 limb lead** (I , II, III, aVL, aVR, aVF) & **6 chest lead** (V1-V6)
2. **Bipolar leads (I , II, III)** : using voltage difference between 2 active electrode to create an image (2 eye vision example :))
3. **Unipolar leads (aVL, aVR, aVF, V1-V6)** : measuring voltage in a single electrode while others are set as earth! (one eye vision ;))

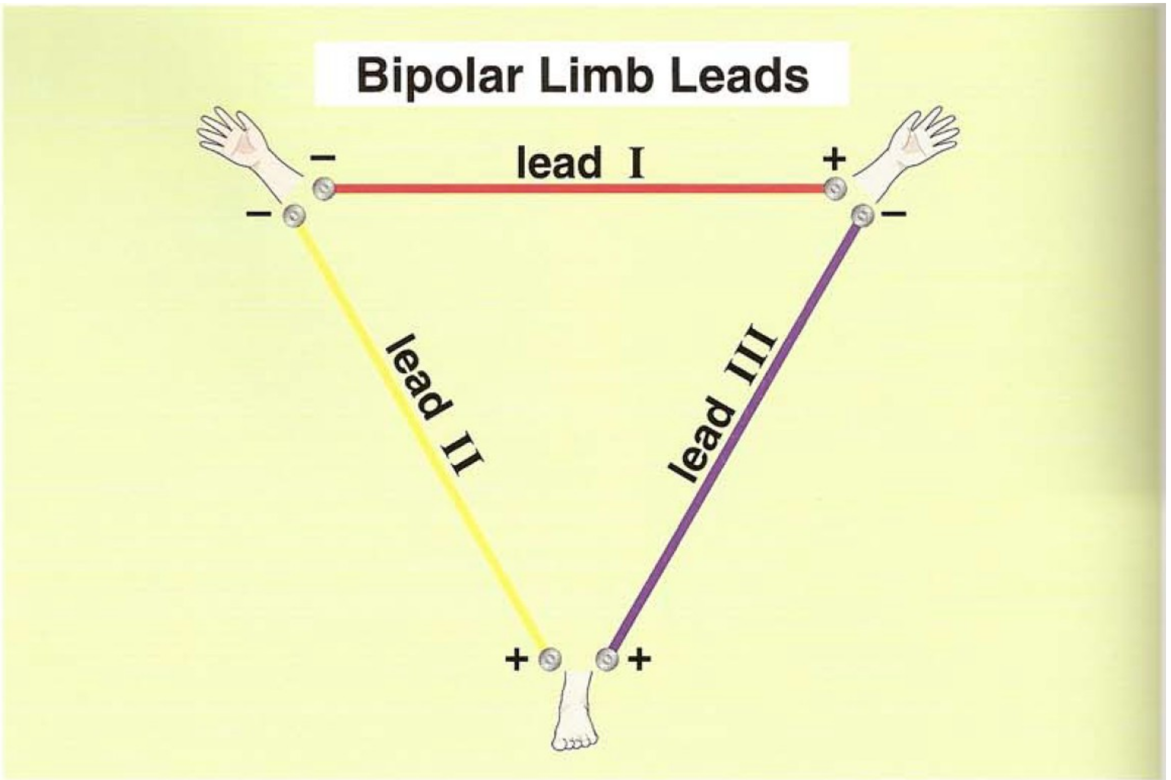
ELECTRODE PLACEMENT



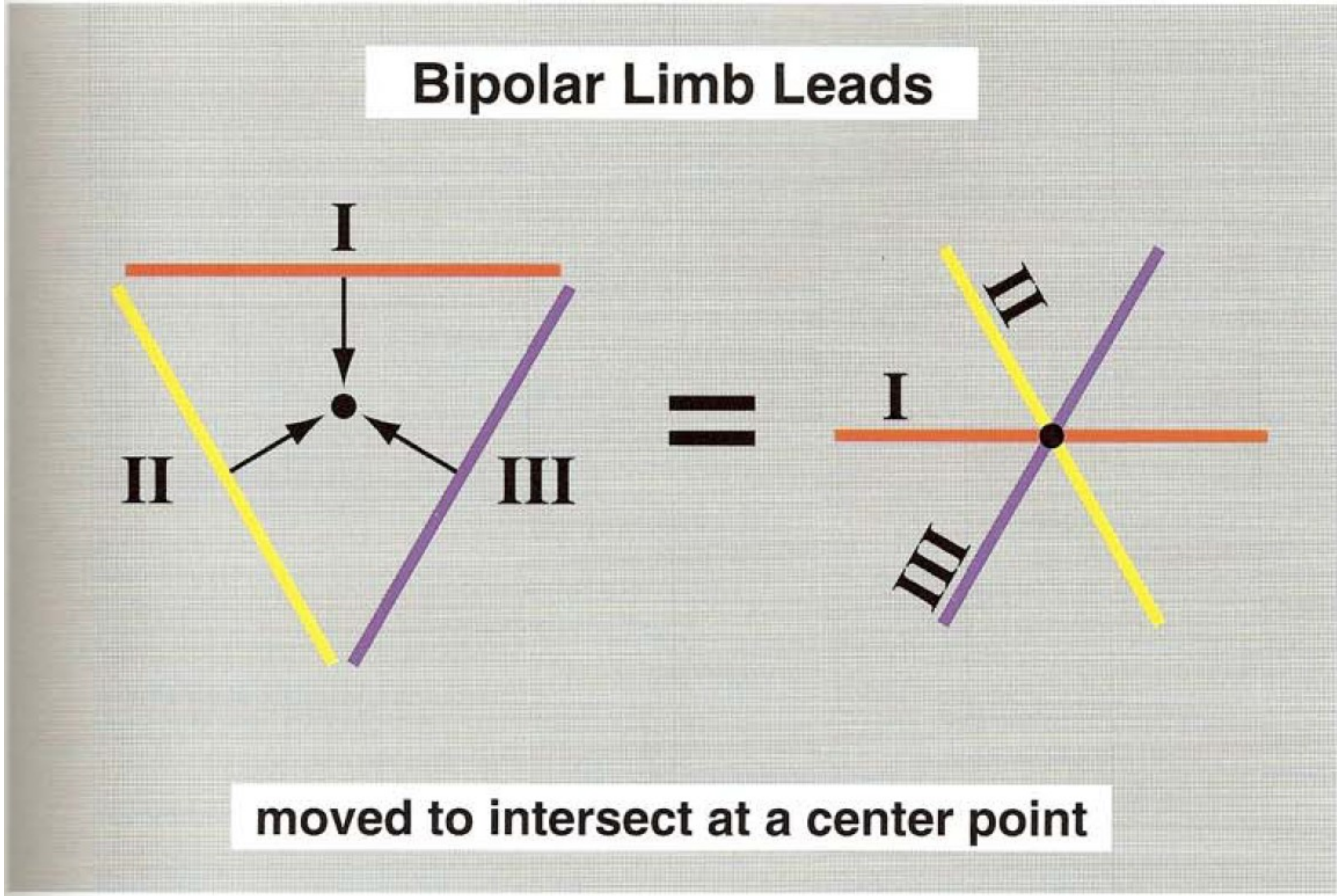
Bipolar limb Leads



To obtain the **limb leads**, electrodes are placed on the right arm, the left arm, and the left leg. A pair of electrodes is used to record a lead.

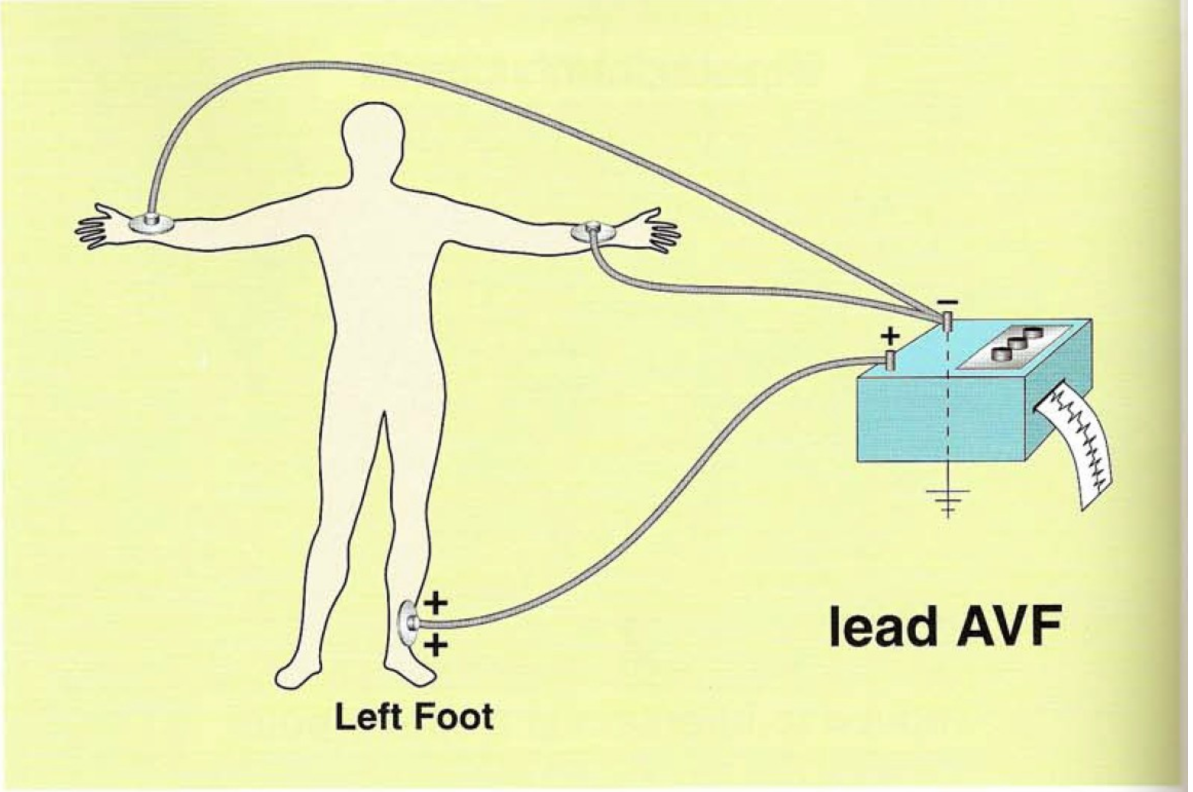


Each **bipolar limb lead** is recorded using two electrodes. So by selecting a different pair of electrodes for each lead, we create three separate bipolar limb leads (**lead I, lead II, and lead III**) for recording.

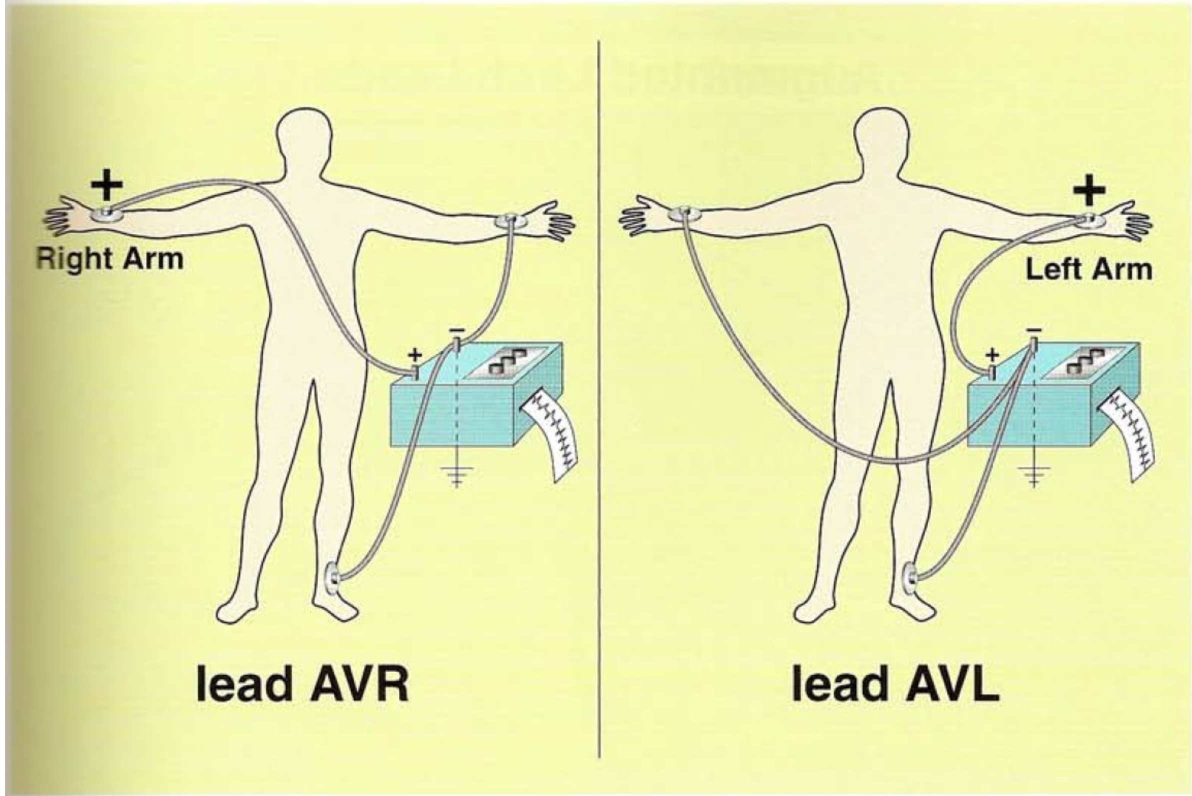


By pushing the three (bipolar) limb leads to the center of the triangle, we produce three intersecting lines of reference.

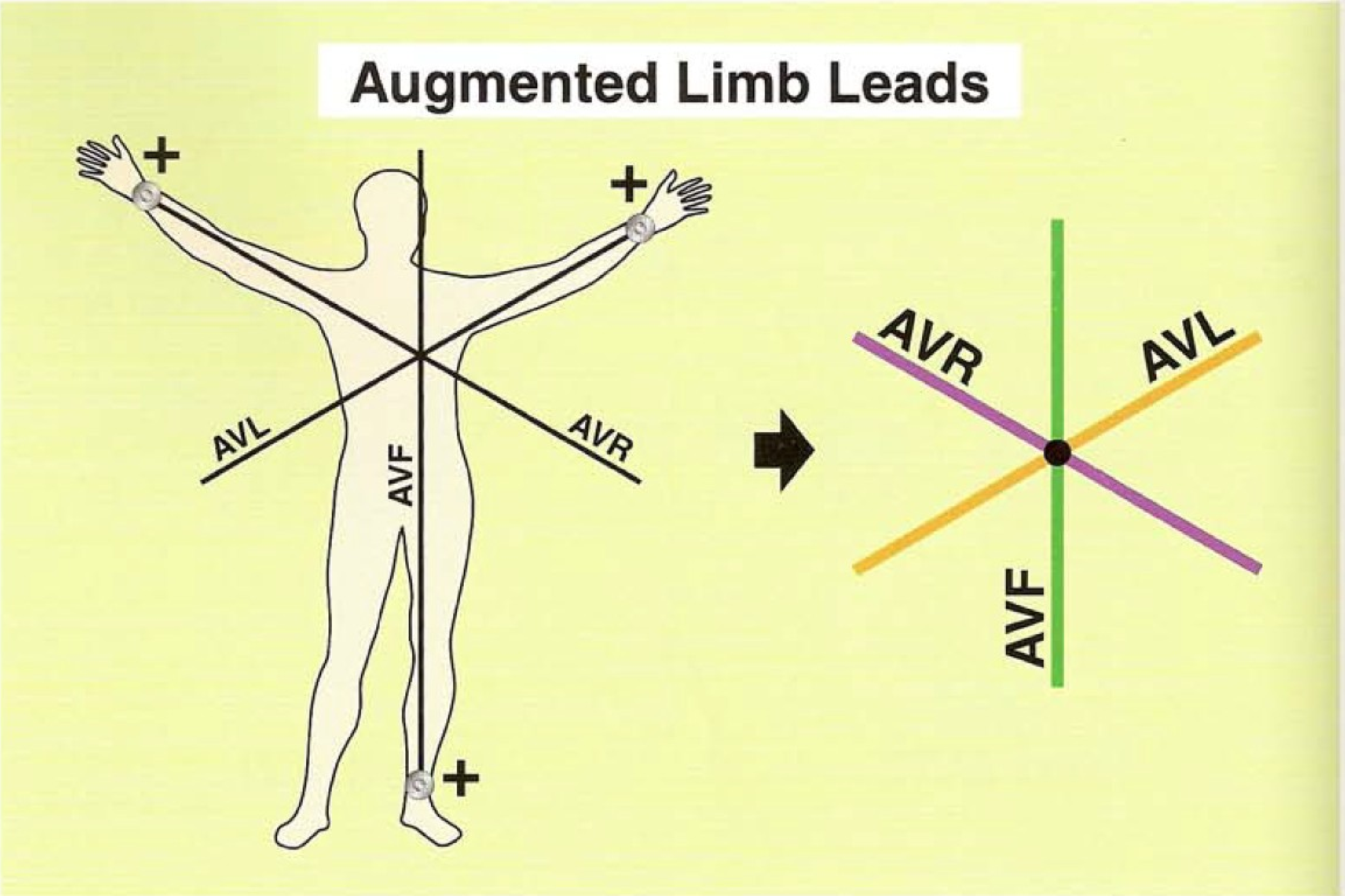
Augmented unipolar limb leads



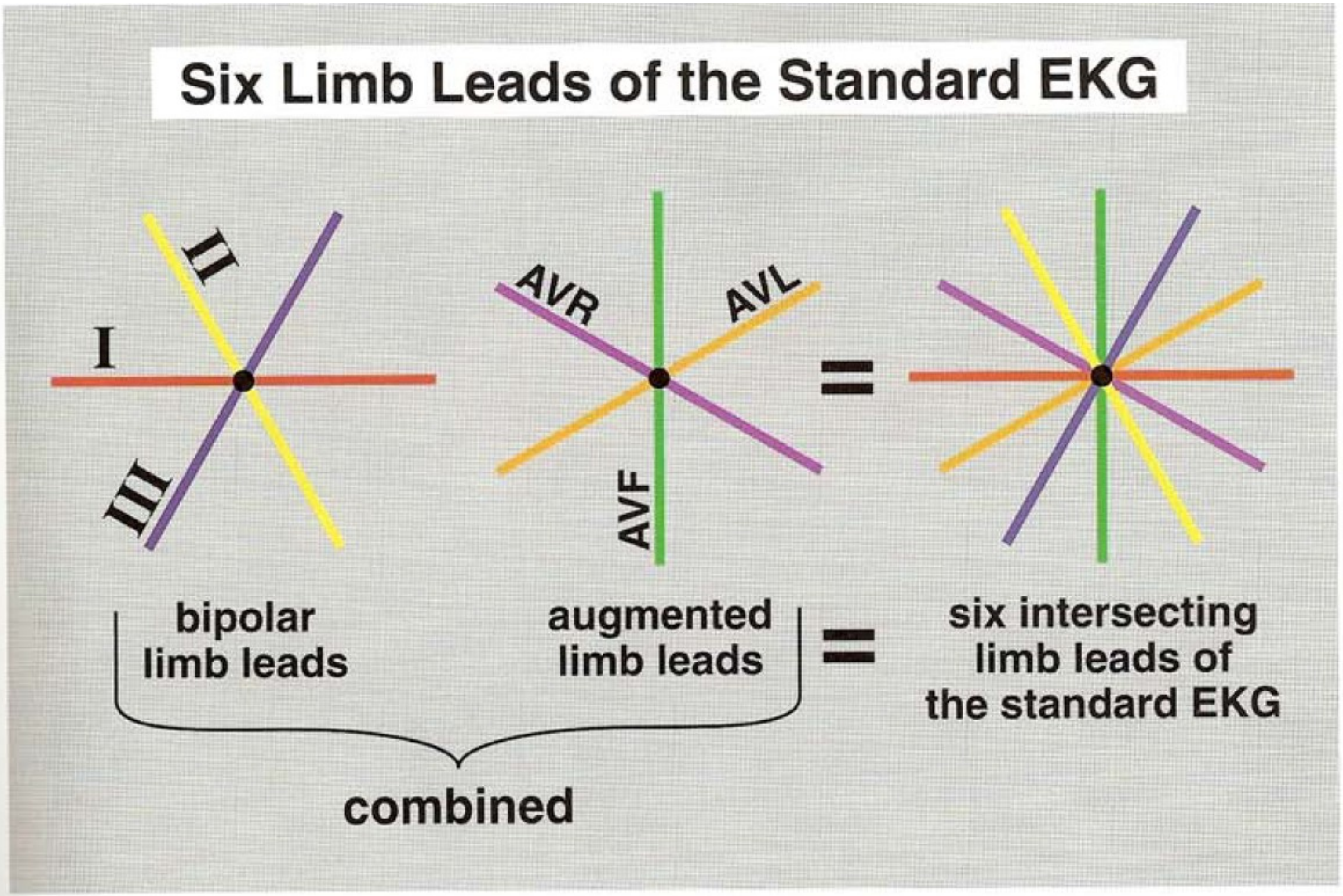
Another standard lead is the **AVF** lead. The AVF lead uses the left foot electrode as positive and both arm electrodes as a common ground (negative).



The remaining two augmented limb leads, **AVR** and **AVL**, are obtained in a similar manner.

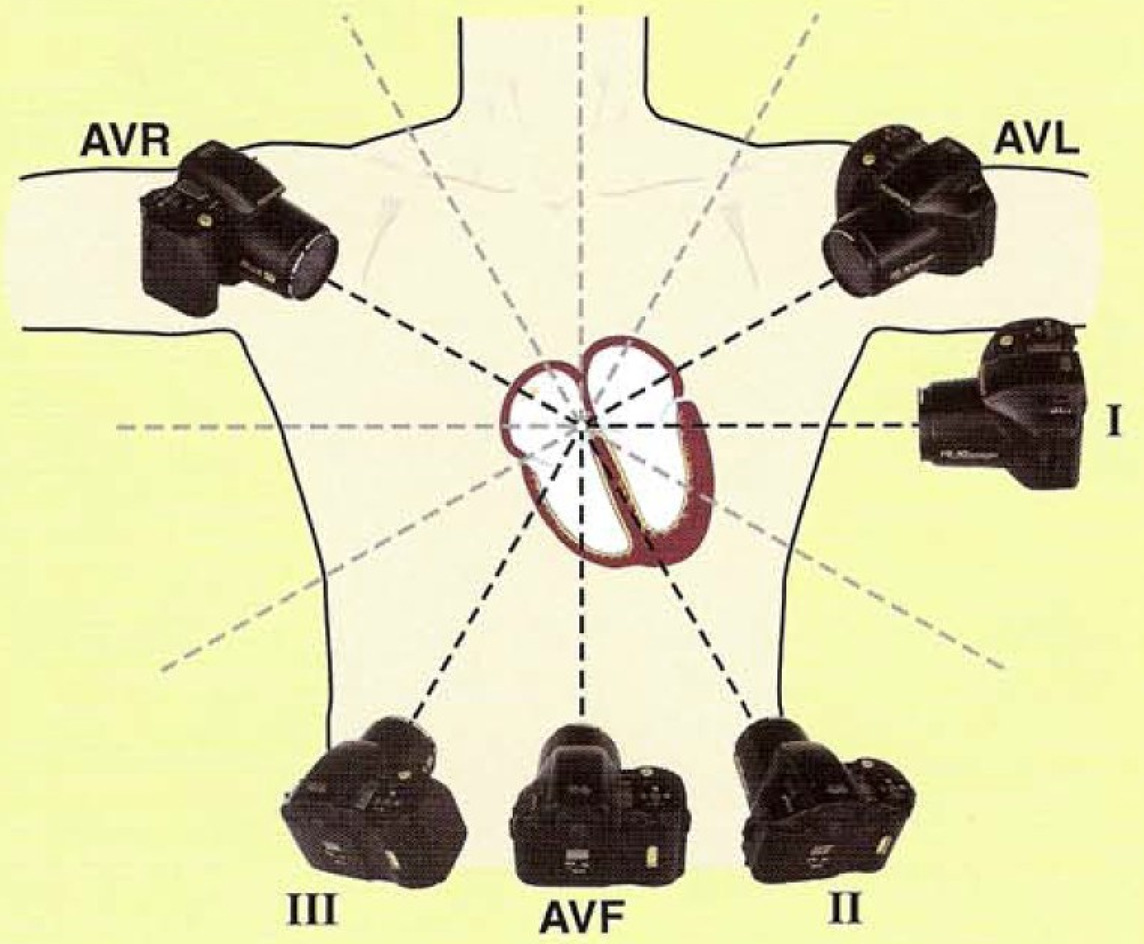


The **augmented limb leads**, AVR, AVL, and AVF, intersect at different angles than those produced by the bipolar limb leads, and they produce three other intersecting lines of reference.



All six limb leads (I, II, III, and AVR, AVL, and AVF) meet to form six intersecting leads that lie in a flat "**frontal**" plane on the patient's chest.

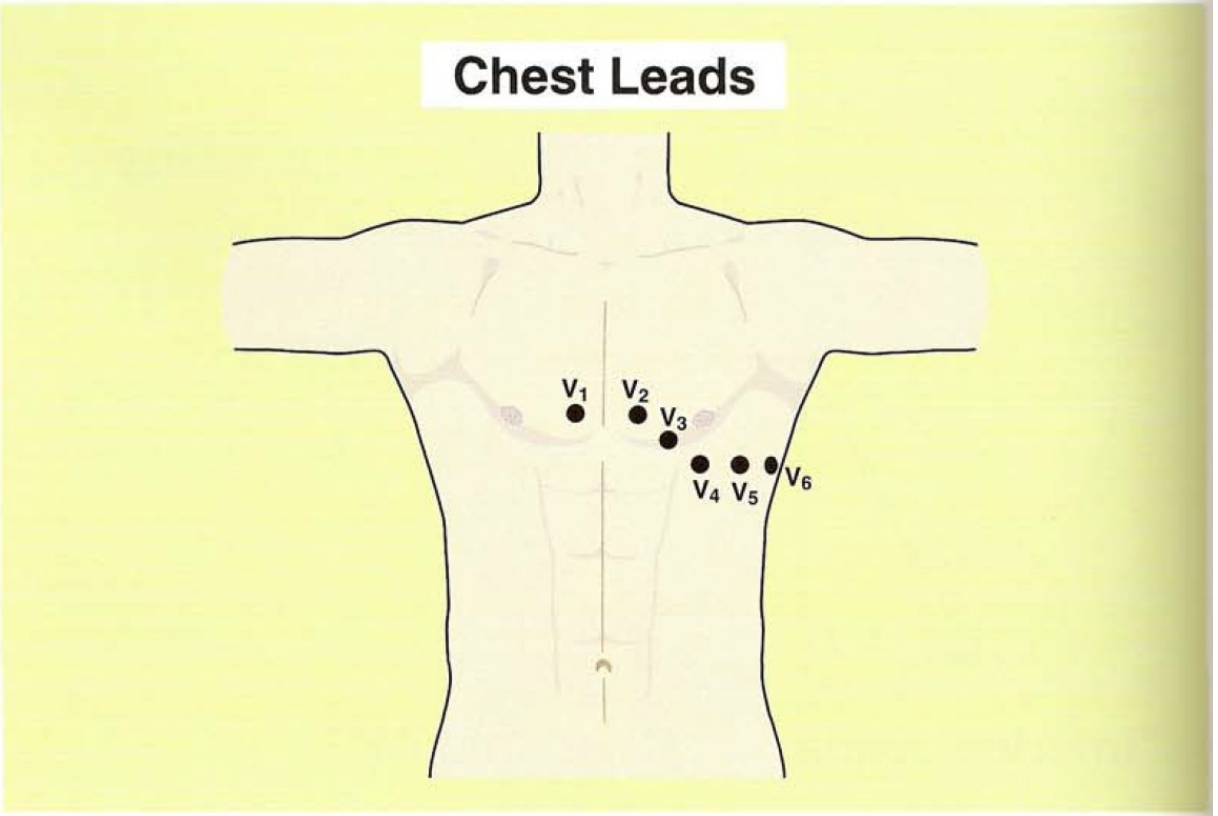
Six Limb Leads are at Six Different Angles



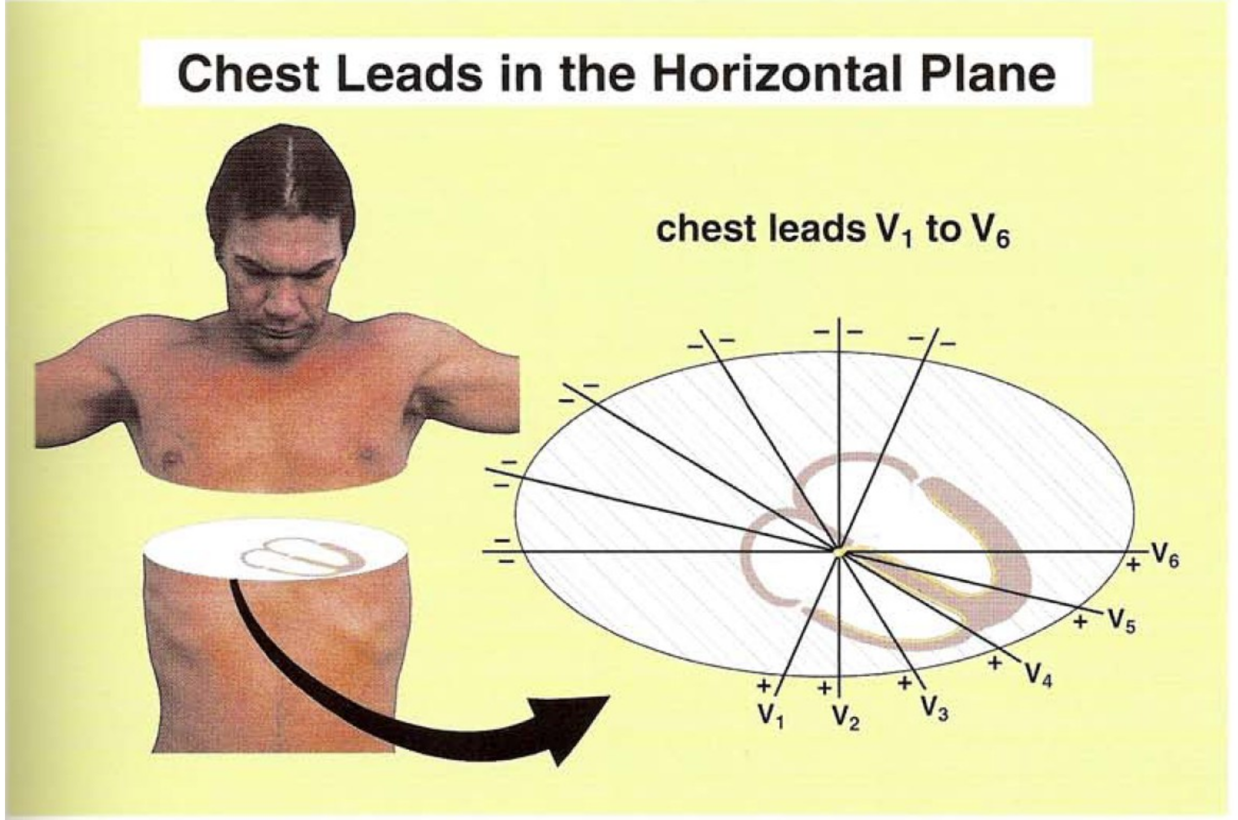
Each camera* position represents the positive electrode of a standard limb lead. Each limb lead (I, II, III, AVR, AVL, and AVF) records from a different angle (viewpoint), to provide a different view of the same cardiac activity.

By observing the same object from six different angles, you will obtain a great deal of information, and in this case, perhaps even identify the car.

Chest leads

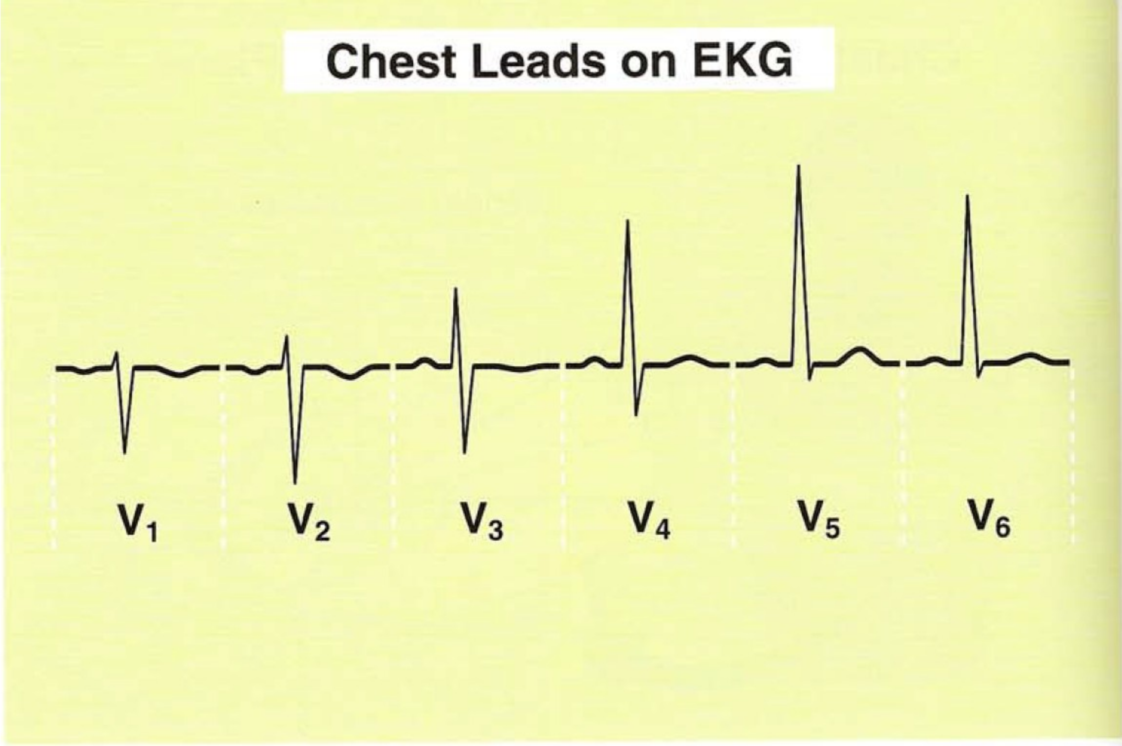


To obtain the six standard **chest leads**, a positive electrode (suction cup) is placed at six different positions (one for each lead) on the chest.

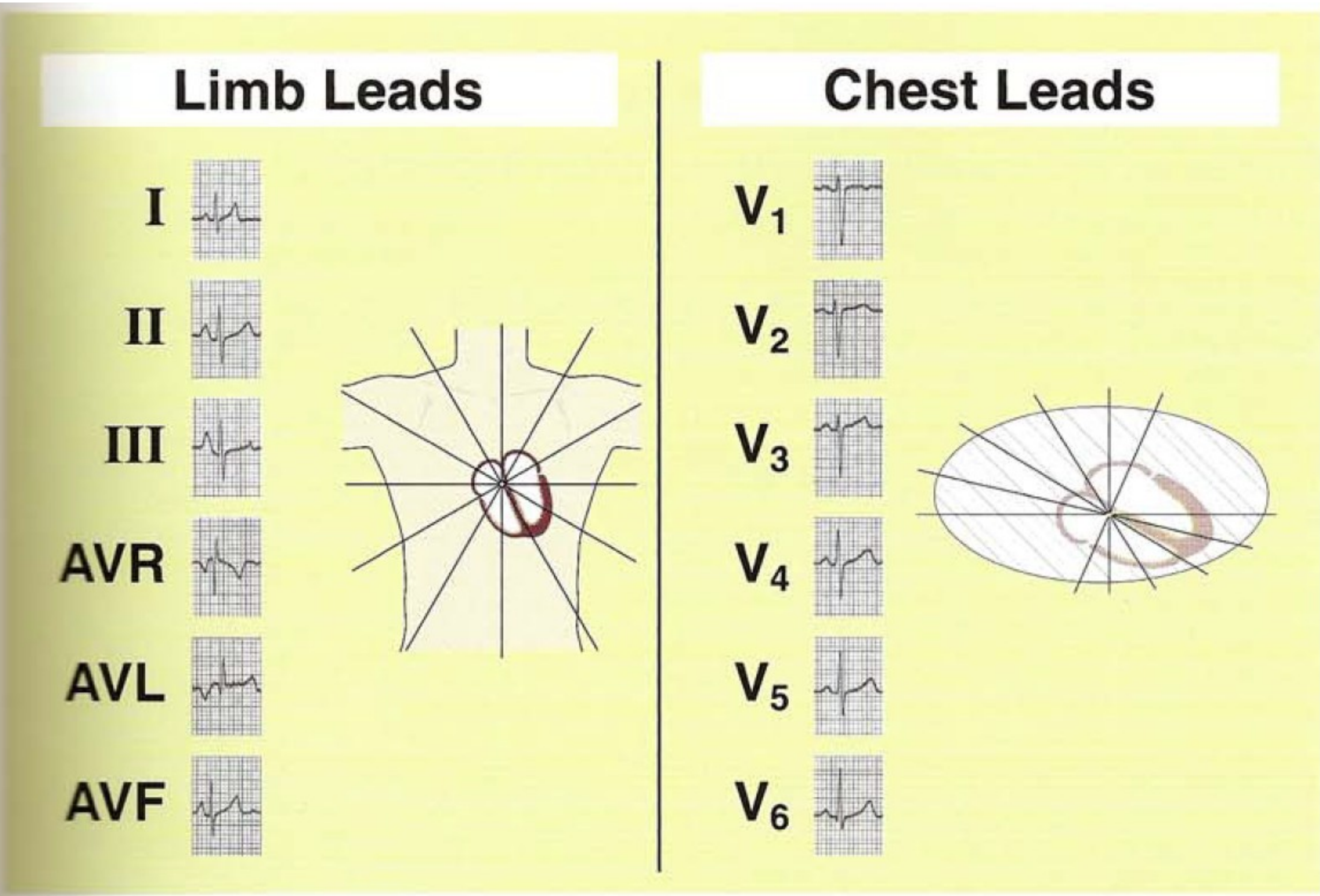


In general, each of the chest leads* is oriented through the AV node and projects through the patient's back, which is negative.

Chest leads

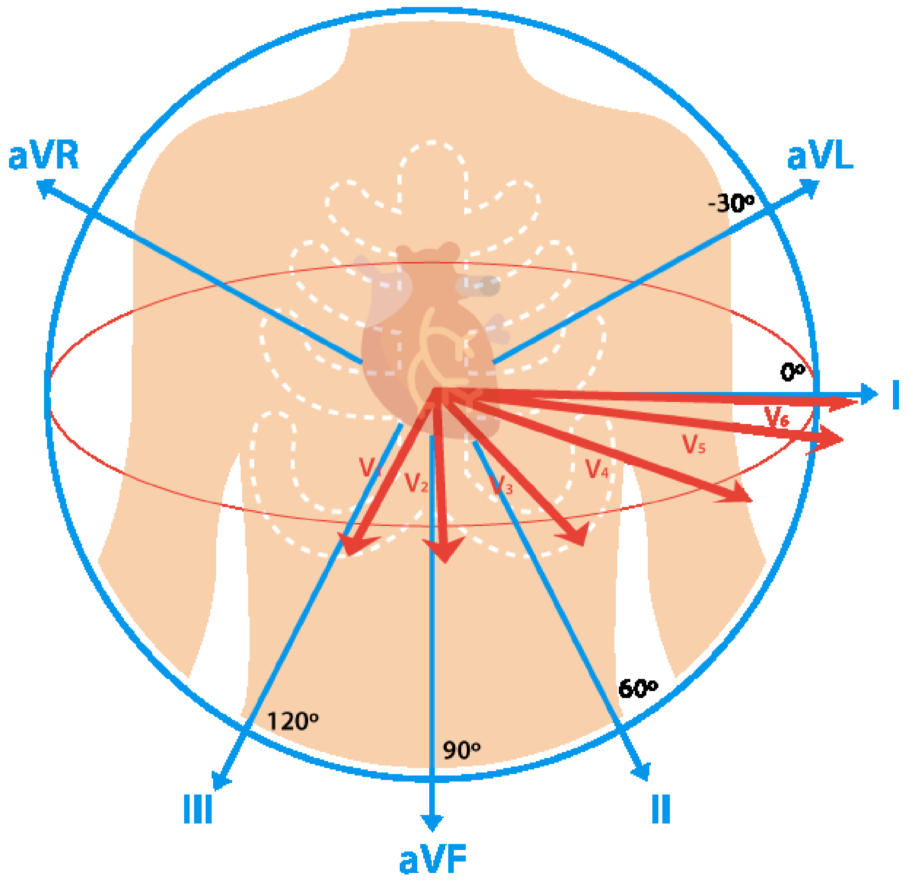


By examining an EKG, you will notice that the waves in the six chest leads show progressive changes from V_1 to V_6.

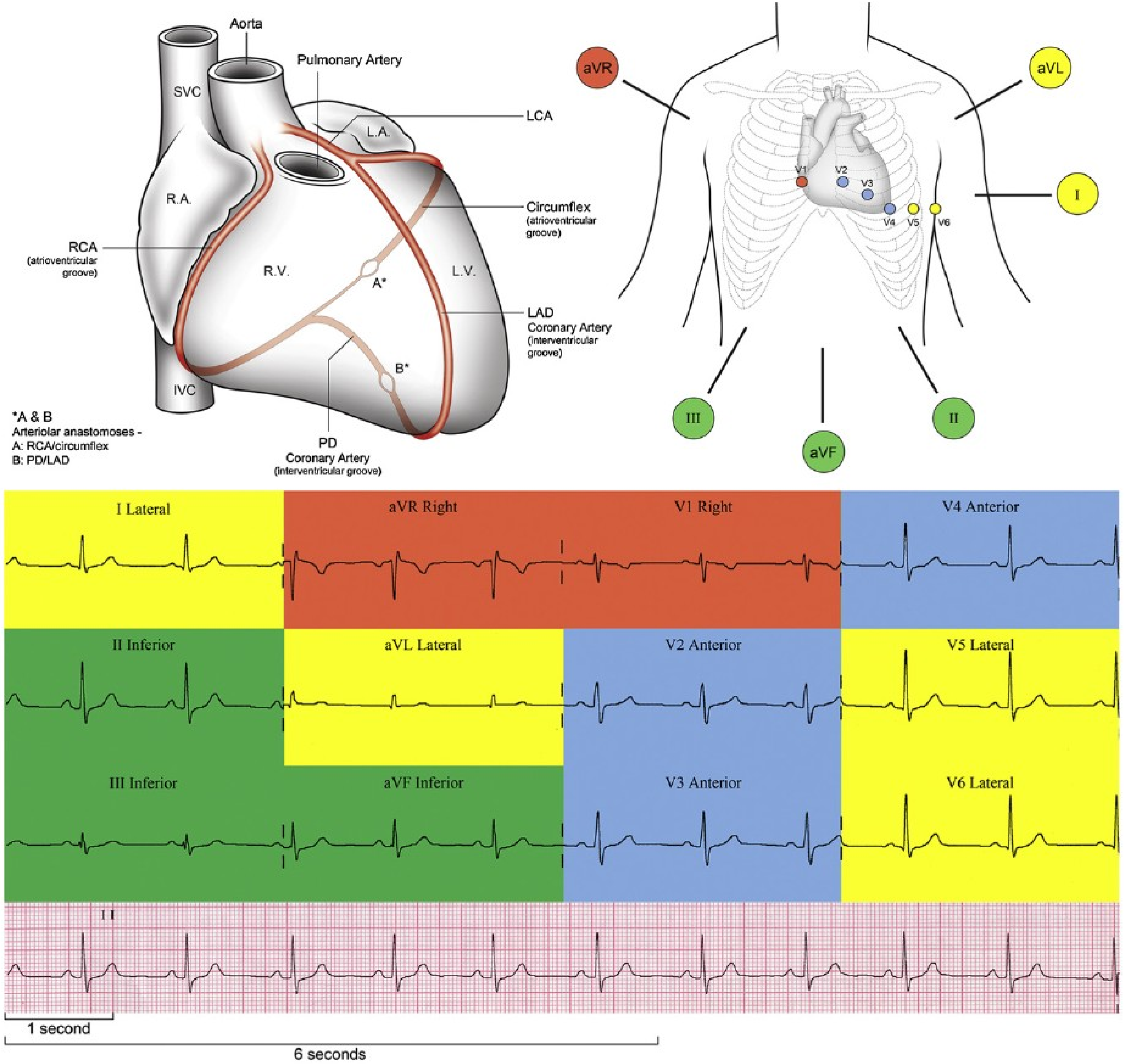


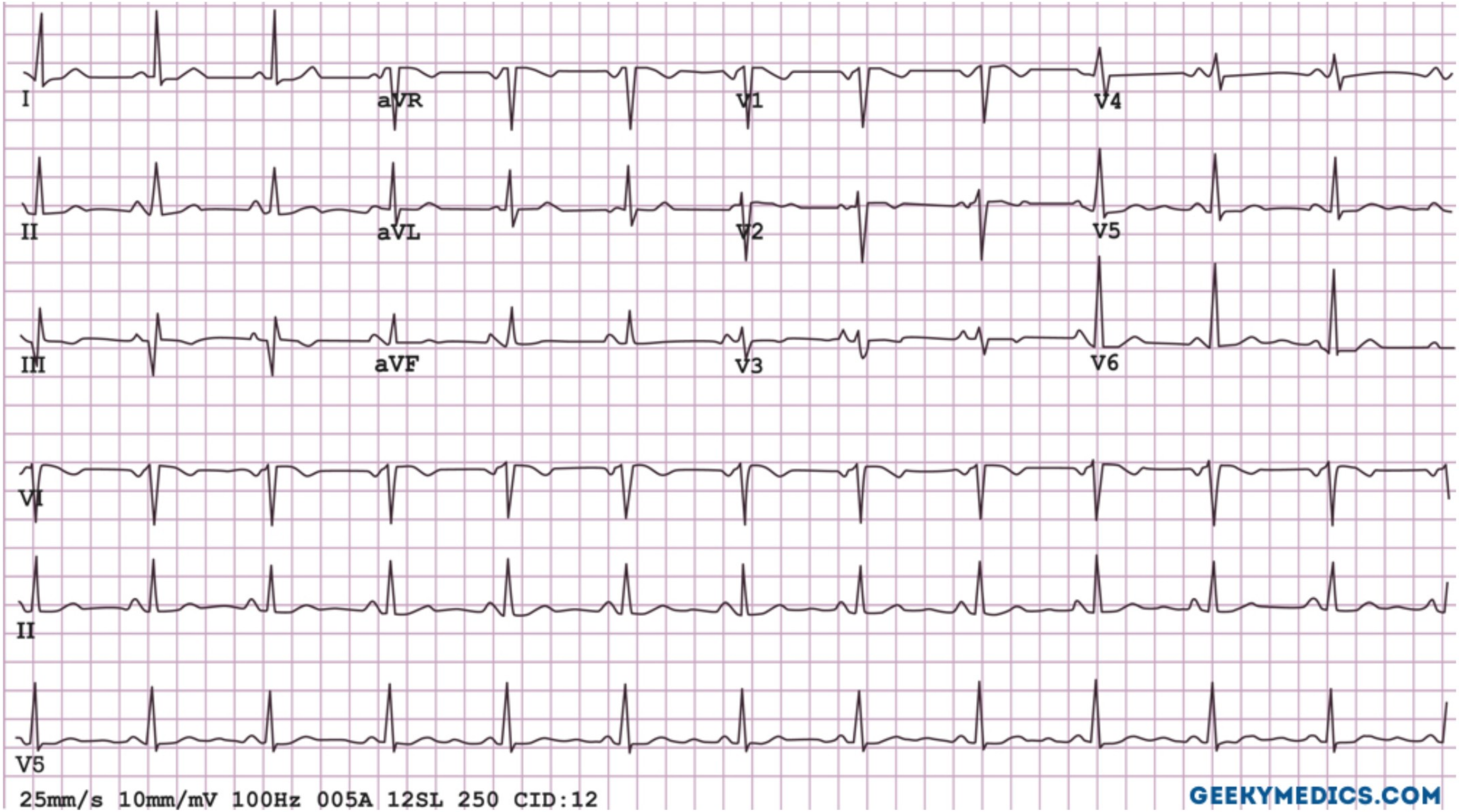
On the standard EKG tracing there are six chest leads and six limb leads. This is the 12 lead electrocardiogram.

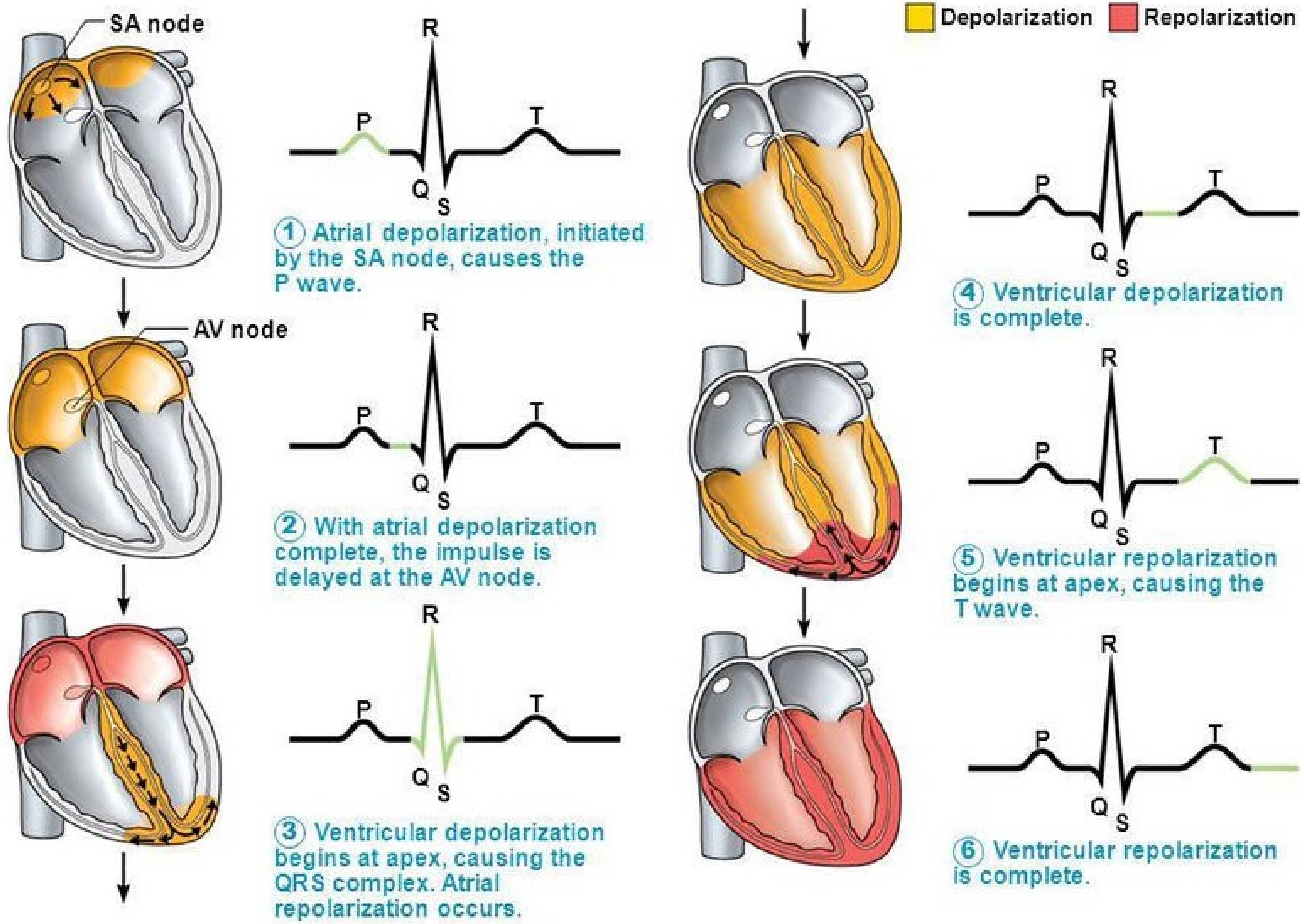
FRONTAL & HORIZONTAL PLANE VIEW



View of heart	Leads
Inferior	II, III, aVF
Lateral	I, aVL,V5,V6
Anterior	V3,V4
Septal	V1,V2


Aorta
Pulmonary Artery
SVC
L.A.
LCA
R.A.
Circumflex
(atrioventricular groove)
RCA
(atrioventricular groove)
R.V.
A*
L.V.
LAD
Coronary Artery
(interventricular groove)
B*
IVC
*A & B
Arteriolar anastomoses -
A: RCA/circumflex
B: PD/LAD
PD
Coronary Artery
(interventricular groove)
aVR
aVL
V1
V2
V3
V4
V5
V6
I
III
aVF
II
I Lateral
aVR Right
V1 Right
V4 Anterior
II Inferior
aVL Lateral
V2 Anterior
V5 Lateral
III Inferior
aVF Inferior
V3 Anterior
V6 Lateral
II
1 second
6 seconds

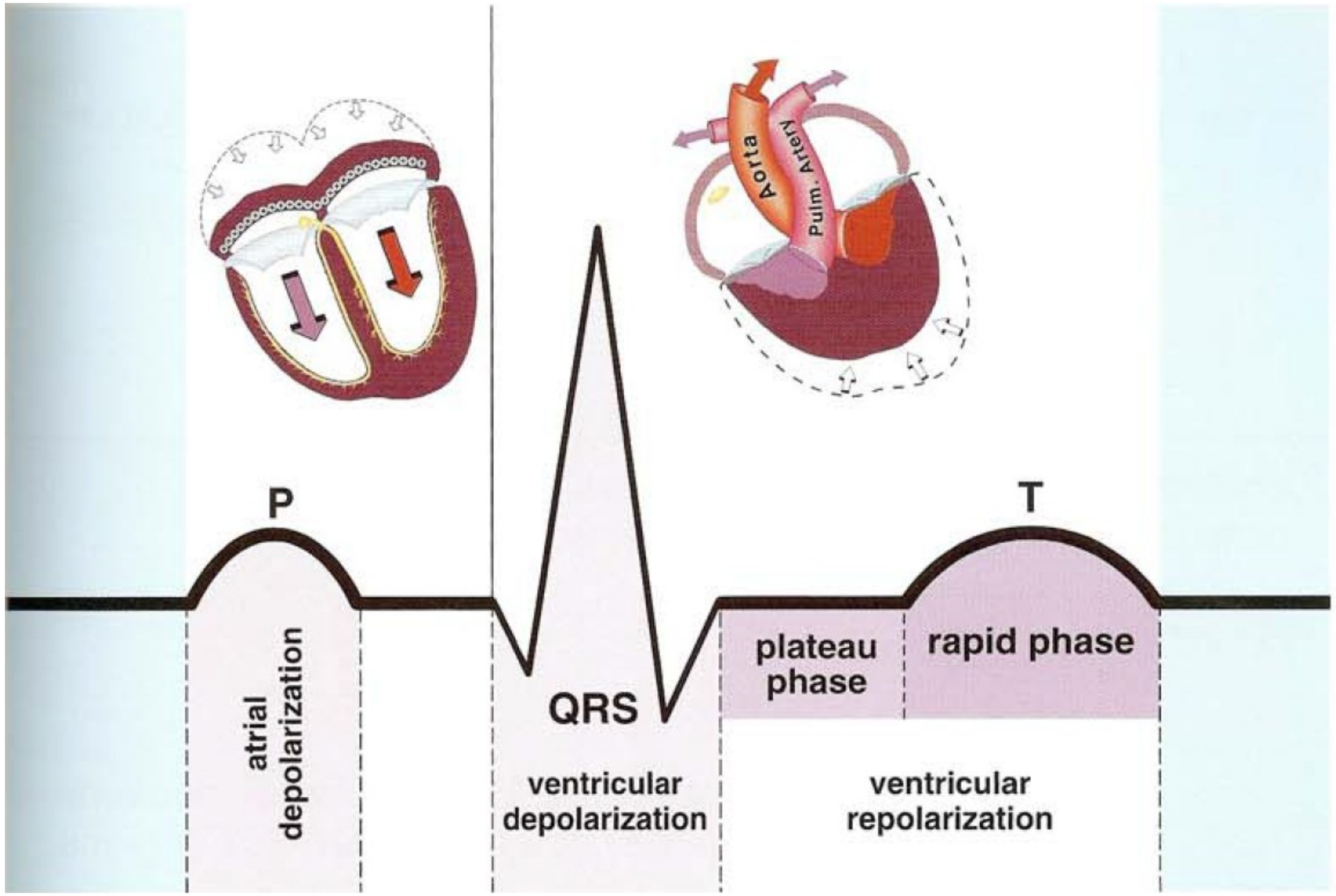

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5
25mm/s 10mm/mV 100Hz 005A 12SL 250 CID:12
GEEKYMEDICS.COM



Depolarization
Repolarization
SA node
AV node
R
P
T
Q
S
1 Atrial depolarization, initiated by the SA node, causes the P wave.
2 With atrial depolarization complete, the impulse is delayed at the AV node.
3 Ventricular depolarization begins at apex, causing the QRS complex. Atrial repolarization occurs.
4 Ventricular depolarization is complete.
5 Ventricular repolarization begins at apex, causing the T wave.
6 Ventricular repolarization is complete.

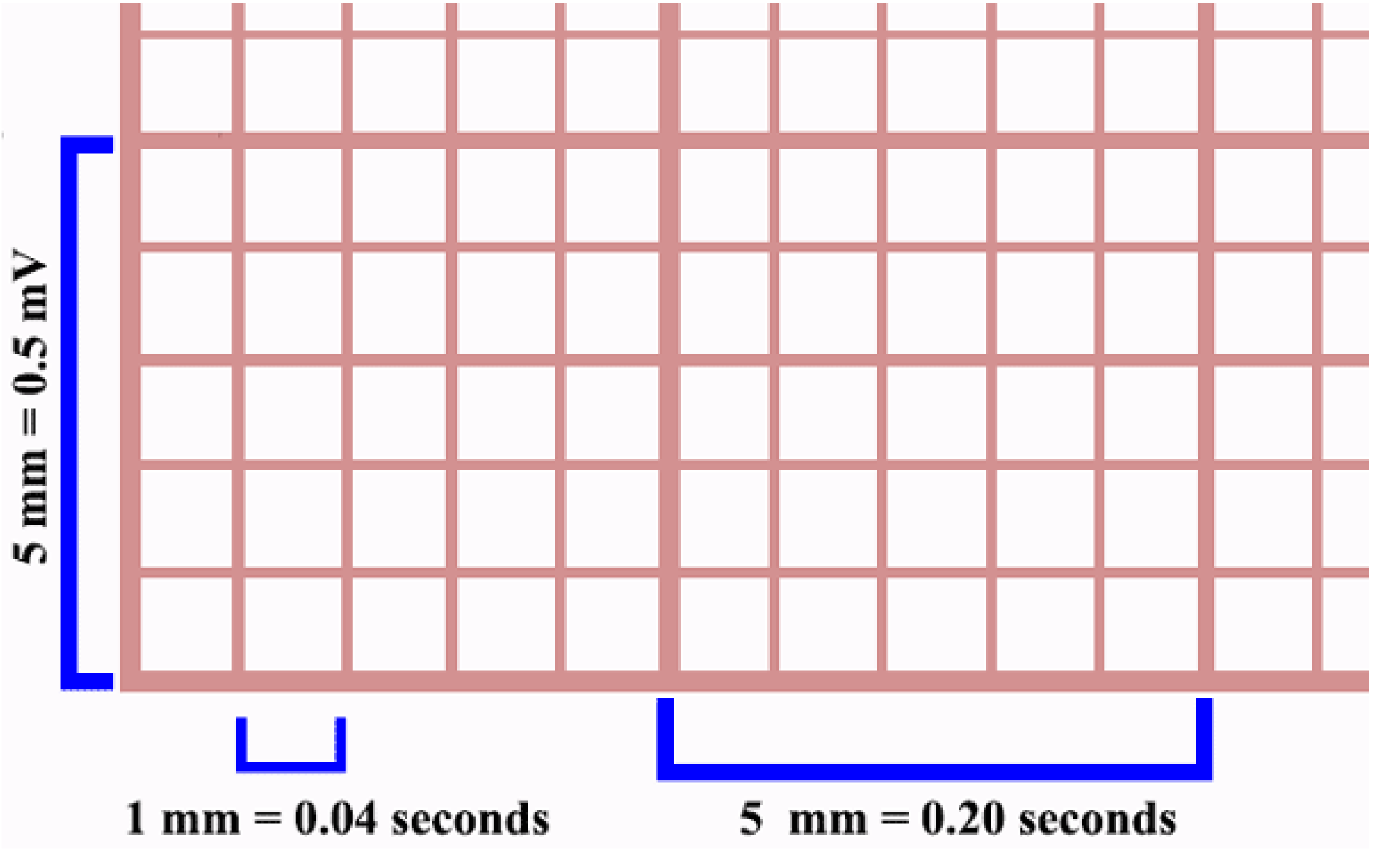
Classic ECG recording (Lead II)

SING IT TO ME !

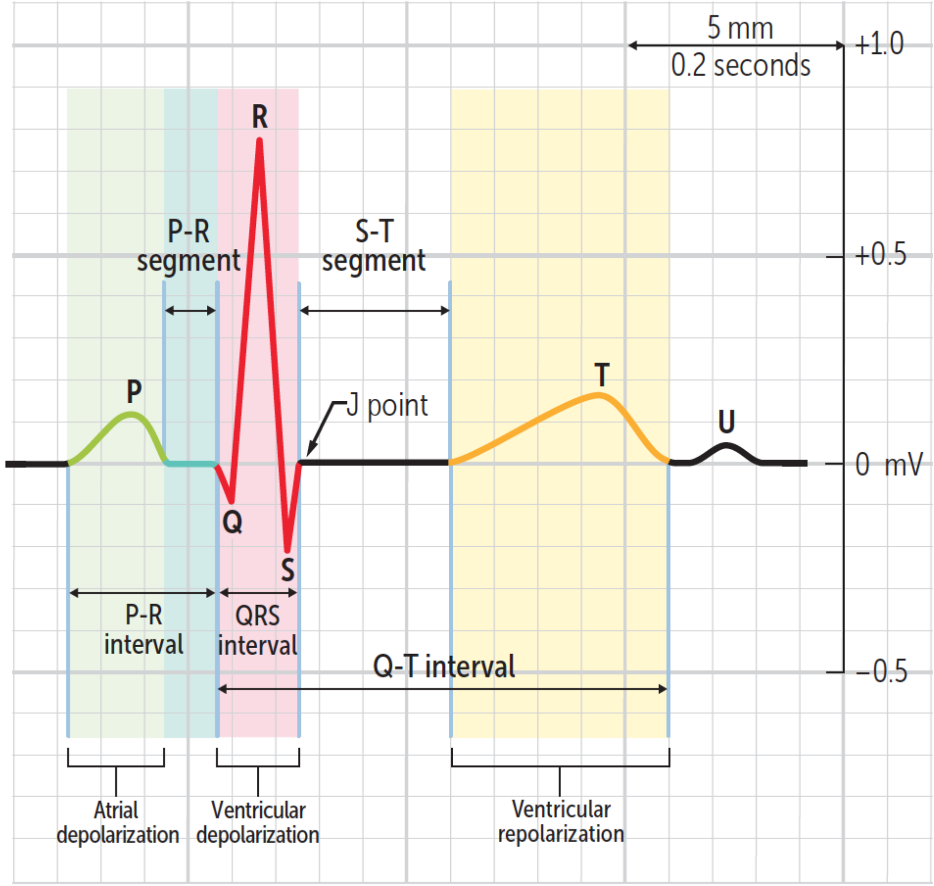


Aorta
Pulm. Artery
P
T
QRS
atrial depolarization
plateau phase
rapid phase
ventricular depolarization
ventricular repolarization

ECG paper



Classic ECG on Lead II



How to Read ECG ?

1. **RATE**
2. **RHYTHM**
3. **AXIS**
4. **INTERVALS**
5. **WAVEFORMS**

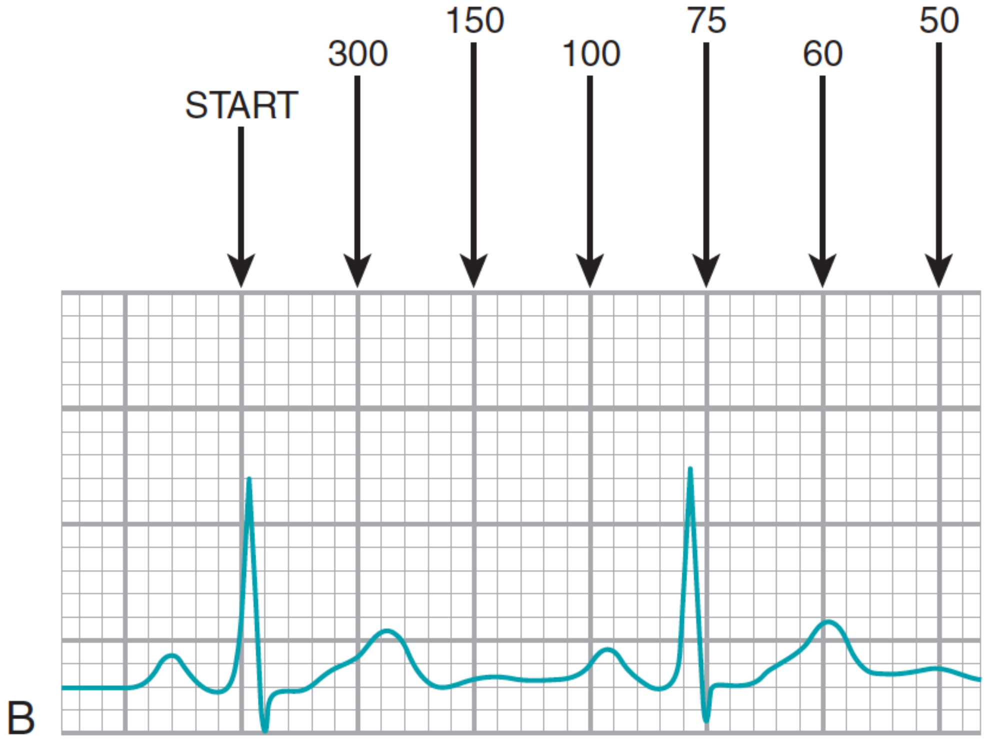
RATE

Normal HR : 50-100 bpm

HR > 100 → <u>TACHYCARDIA</u>

HR < 50 → <u>BRADYCARDIA</u>

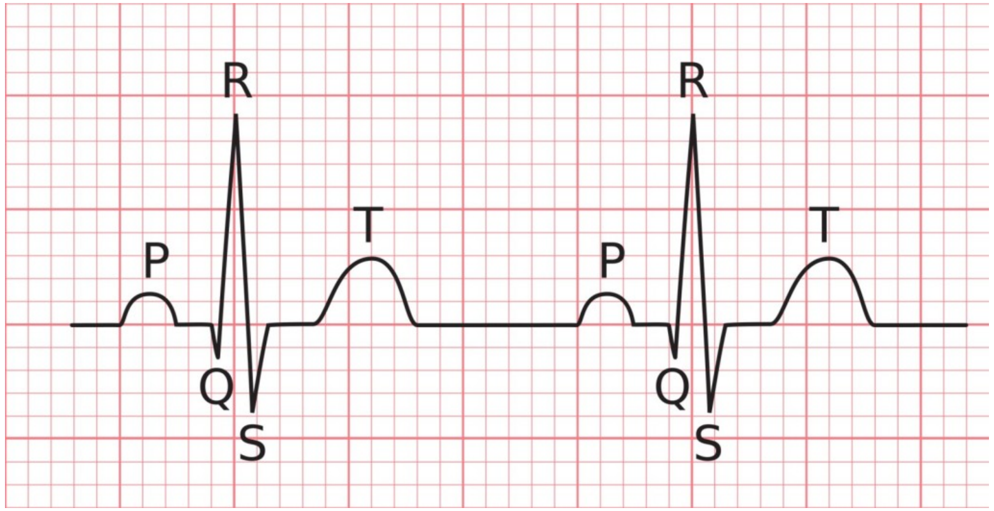
300/ Number of **large boxes between 2 R**



Rythm

Is it Regular Sinus rhythm ?

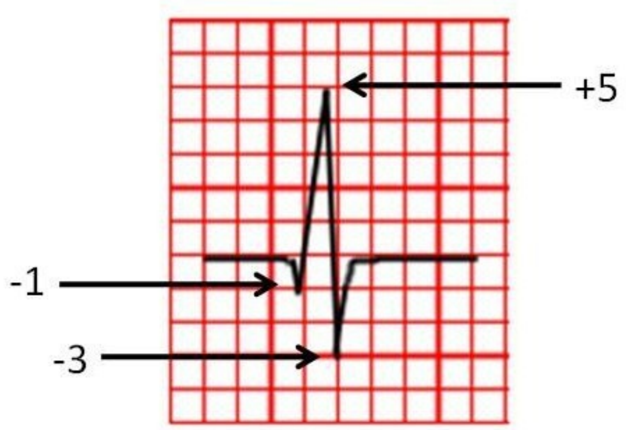
1. Normal morphology of P wave
2. P before QRS and QRS after every P
3. RR and PP intervals are equal in all leads



MEAN ELECTRICAL AXIS (MEA)

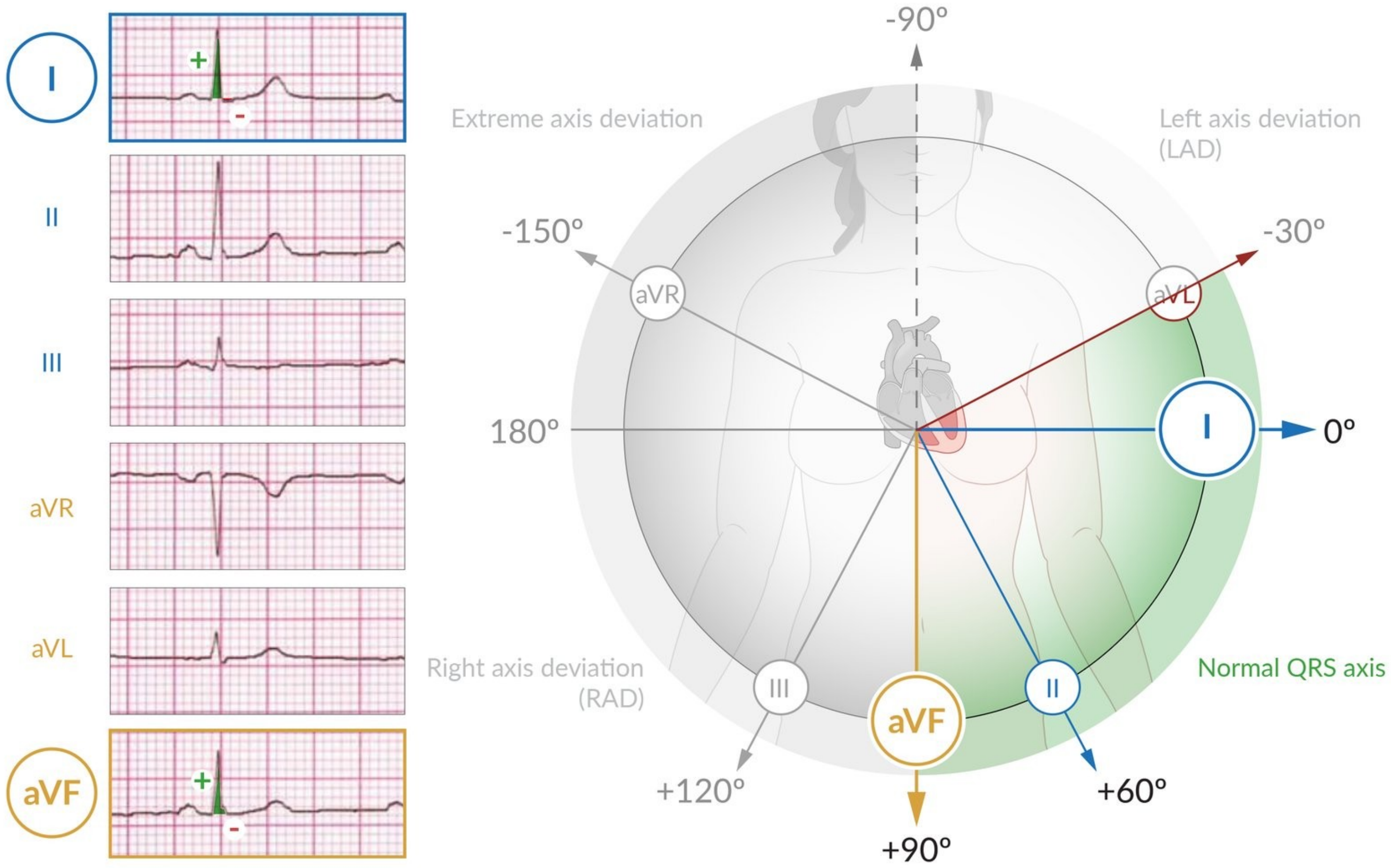
The MEA indicates the net direction (vector) of current flow during ventricular depolarization.

Determine the net QRS deflection (upward minus downward) in lead I and aVF. Using these 2 leads allows us to partition the mathematical grid into 4 basic quadrants (upper left panel of Figure II-3-9).



Net deflection = 5 – 1 – 3
= +1

AXIS



Rule of thumb: the heart axis (QRS) rotates **towards** hypertropy and **away** from infarction

ECG intervals

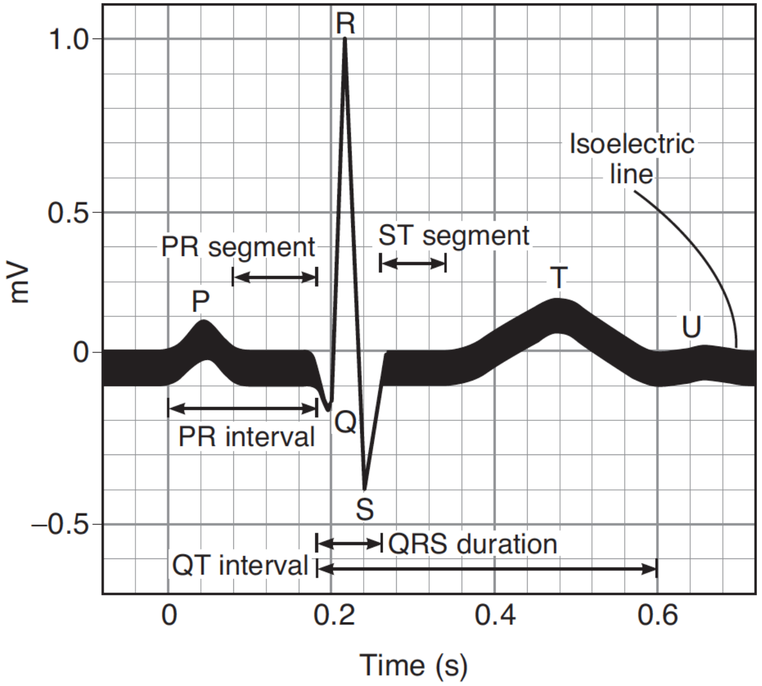


FIGURE 30–5 **Waves of the ECG.**

TABLE 30–2 **ECG intervals.**

Intervals	Normal Durations: Average	Normal Durations: Range	Events in the Heart during Interval
PR interval[a]	0.18[b]	0.12–0.20	Atrial depolarization and conduction through AV node
QRS duration	0.08	to 0.10	Ventricular depolarization and atrial repolarization
QT interval	0.40	to 0.43	Ventricular depolarization plus ventricular repolarization
ST interval (QT minus QRS)	0.32	. . .	Ventricular repolarization (during T wave)

[a]Measured from the beginning of the P wave to the beginning of the QRS complex.

[b]Shortens as heart rate increases from average of 0.18 s at a rate of 70 beats/min to 0.14 s at a rate of 130 beats/min.

Arrythmia

1. Altered automaticity
2. Altered conduction

Arrythmias

- Based on Rate → **Bradyarrythmia Vs Tachyarrythmia**
- Based on Location of origin → **Supraventricular Vs Ventricular**
- Base on mechanism of origin →

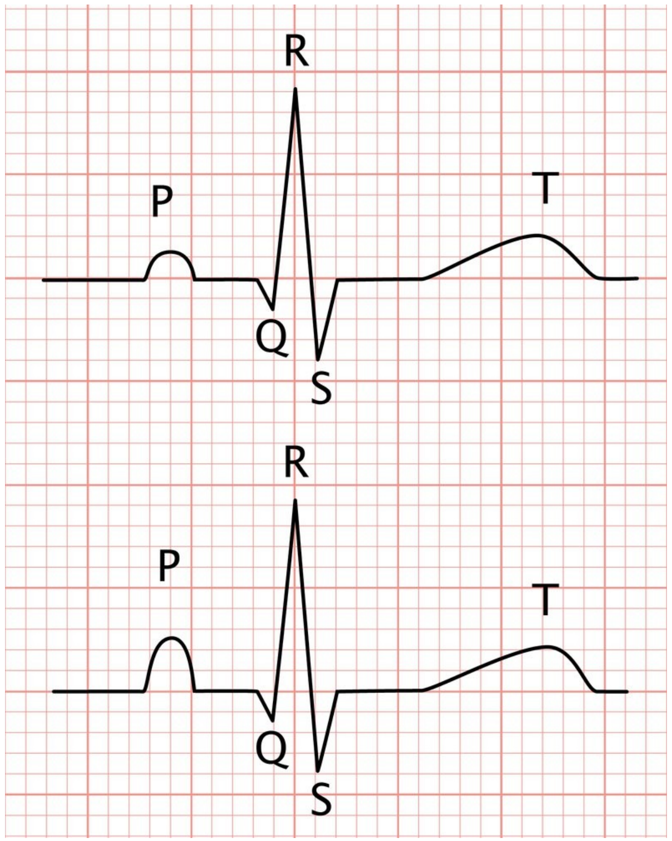
- **Irregular rythms**
- **Escape**
- **Premature beats**
- **Tachy-Arrythmias**

P Pulmonale & Mitrale

P Pulmonale

Causes **Peaked** P wave

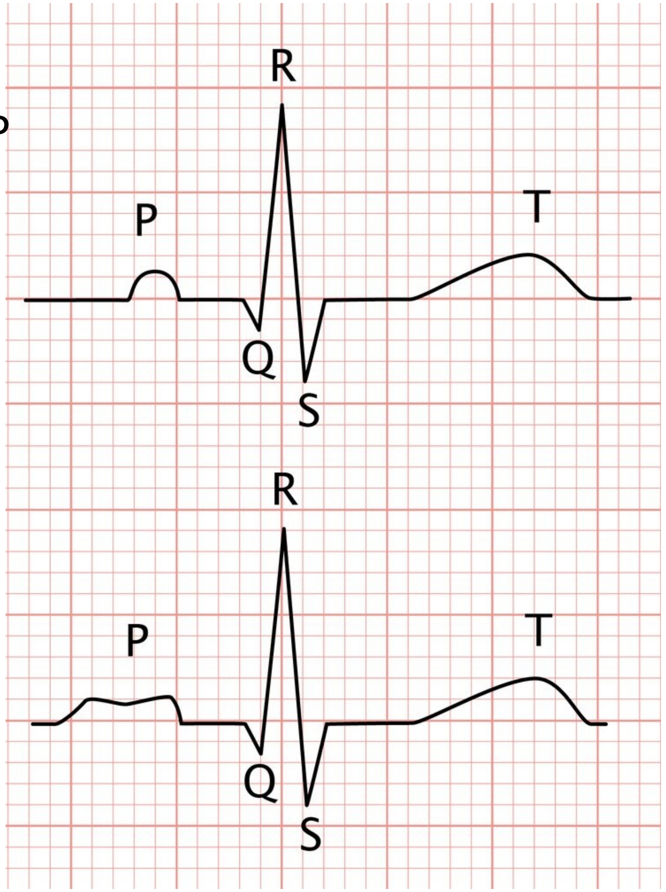
Elevation of P wave > 0.25mv



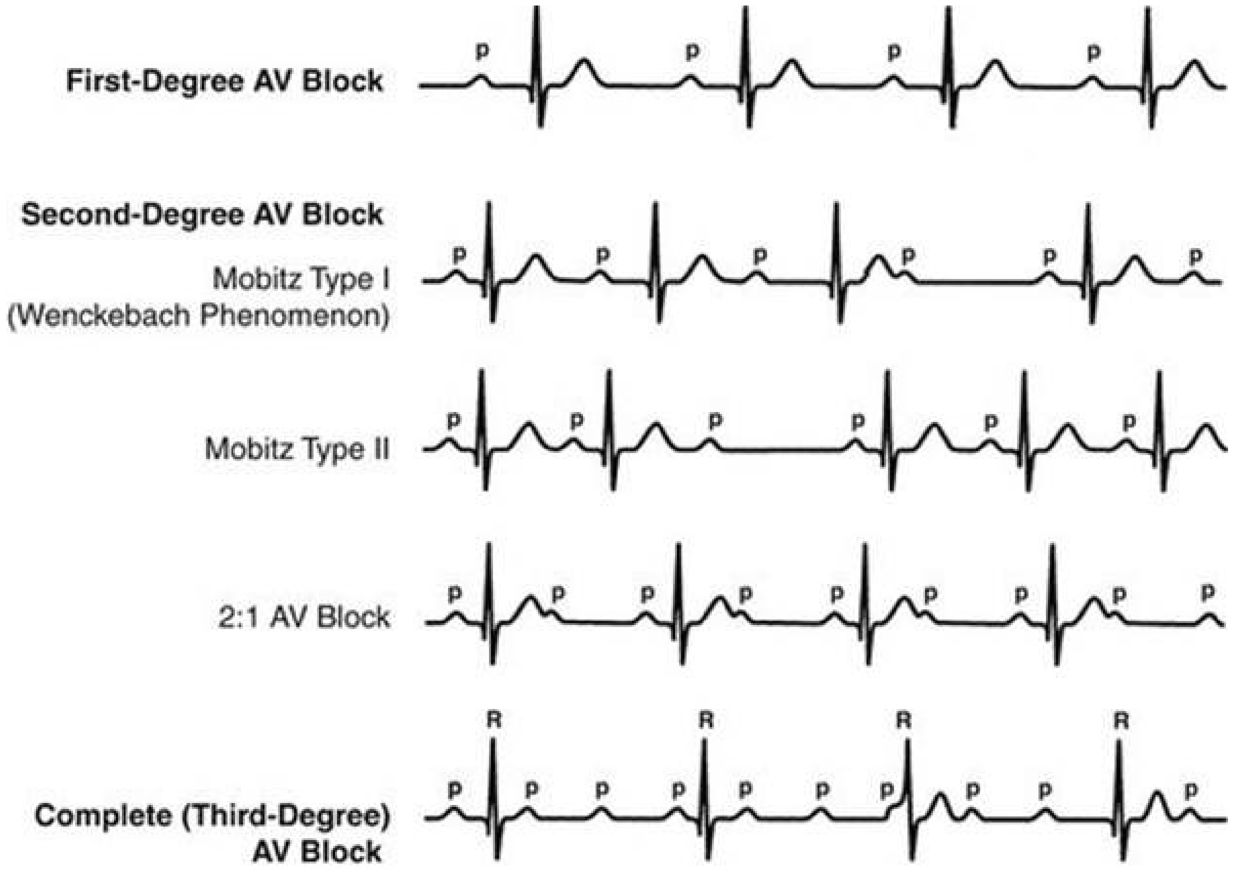
P Mitrale

Causes **M-shaped** P wave

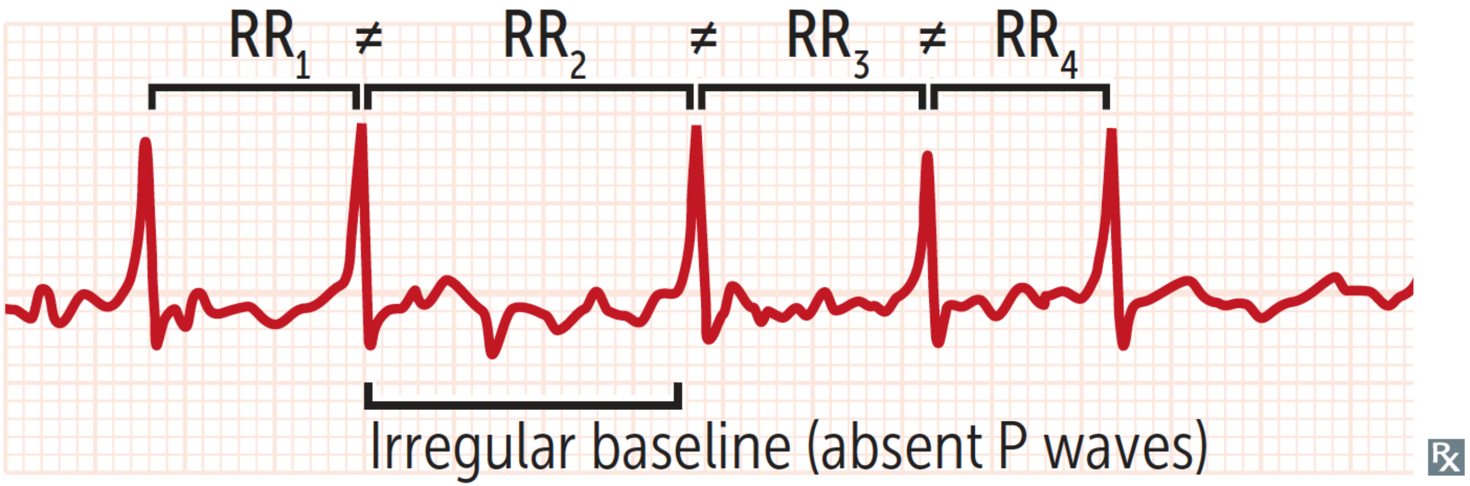
Prolongation of P > 0.10s



PR interval pathology (AV BLOCK)



Atrial fibrillation

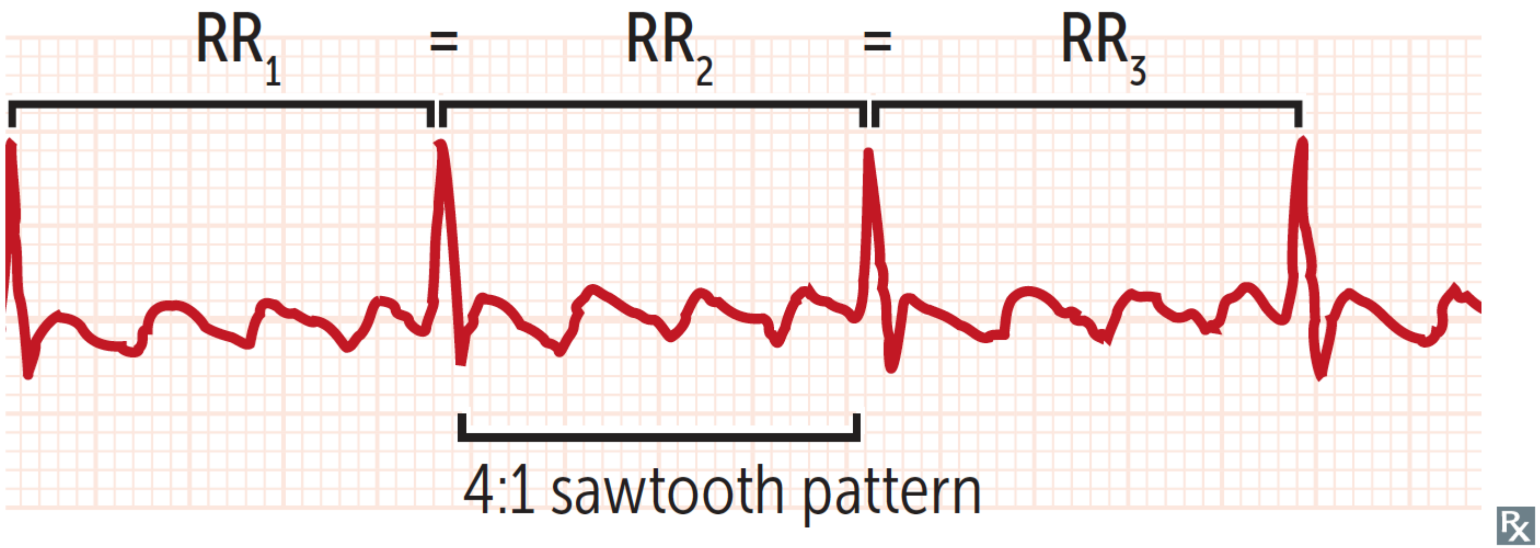


Atrial fibrillation

Chaotic and erratic baseline with no discrete P waves in between irregularly spaced QRS complexes. Irregularly irregular heartbeat. Most common risk factors include hypertension and coronary artery disease (CAD). Can lead to thromboembolic events, particularly stroke.

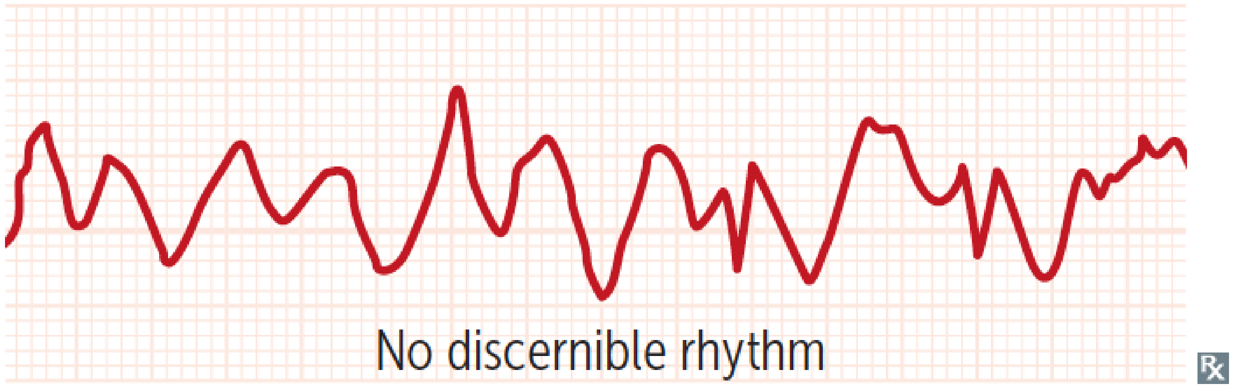
Treatment includes anticoagulation, rate control, rhythm control, and/or cardioversion.

Atrial Flutter



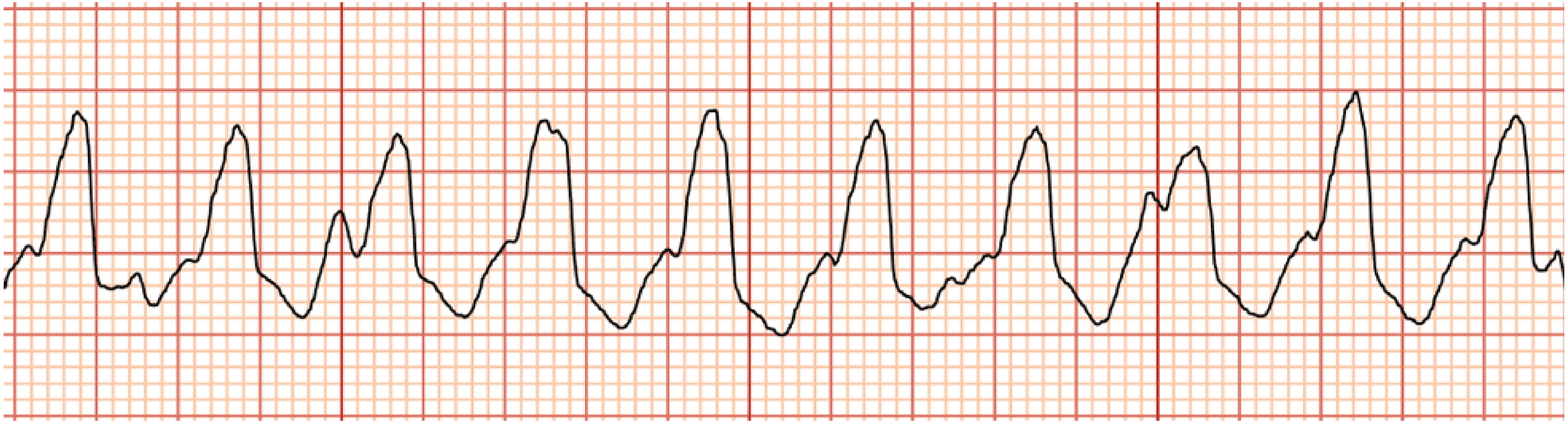
Atrial flutter	A rapid succession of identical, back-to-back atrial depolarization waves. The identical appearance accounts for the "sawtooth" appearance of the flutter waves. Treat like atrial fibrillation. Definitive treatment is catheter ablation.

Ventricular fibrillation (VF)

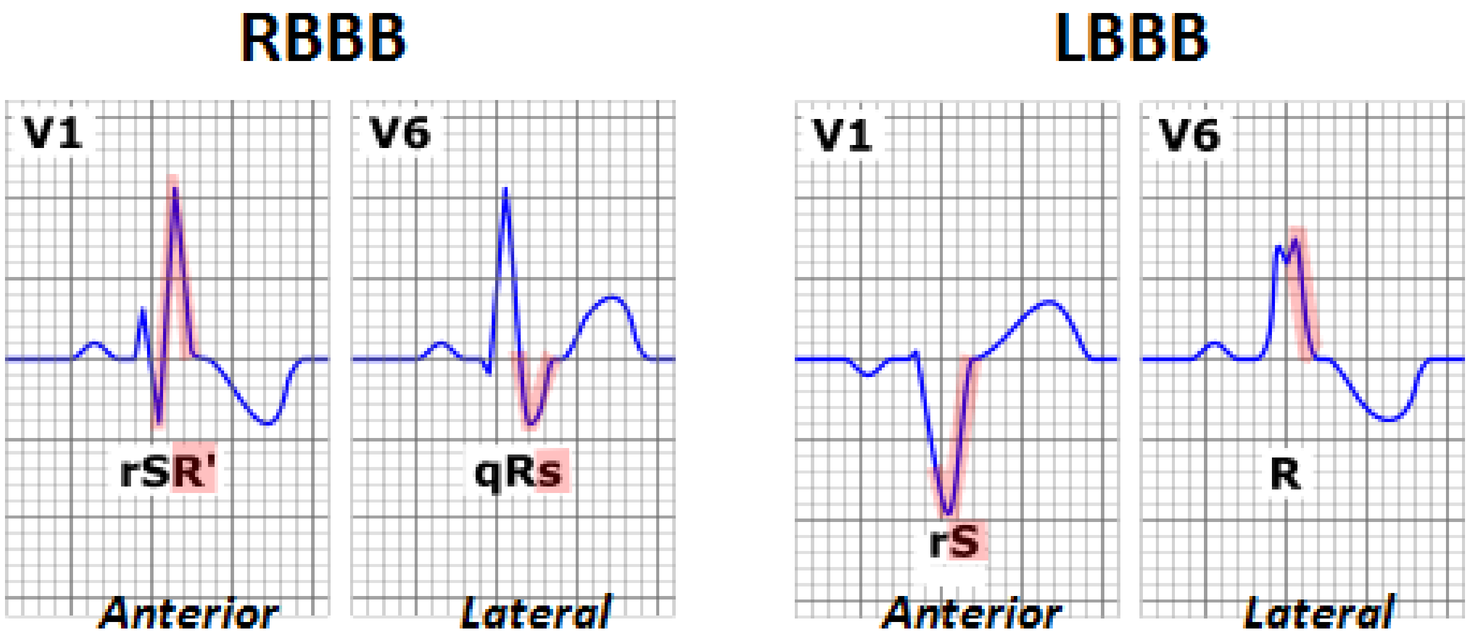


Ventricular fibrillation	A completely erratic rhythm with no identifiable waves. Fatal arrhythmia without immediate CPR and defibrillation.

Ventricular tachycardia

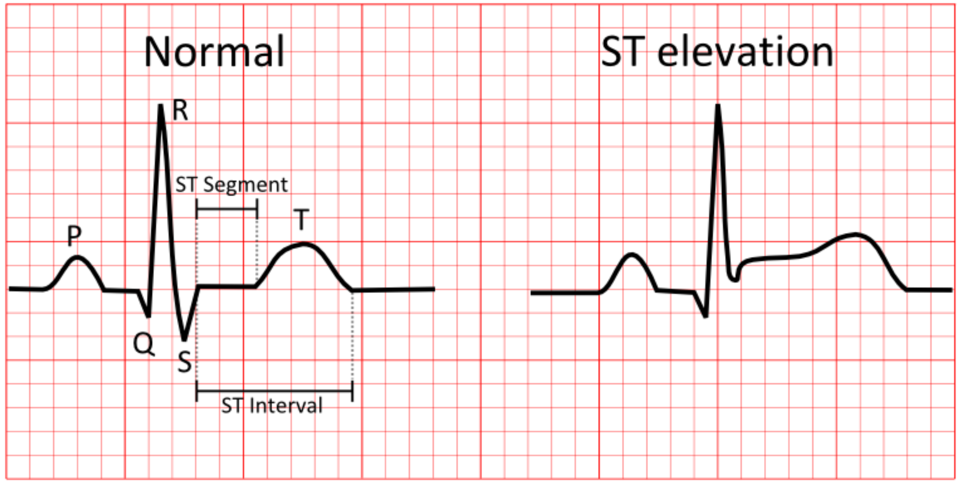


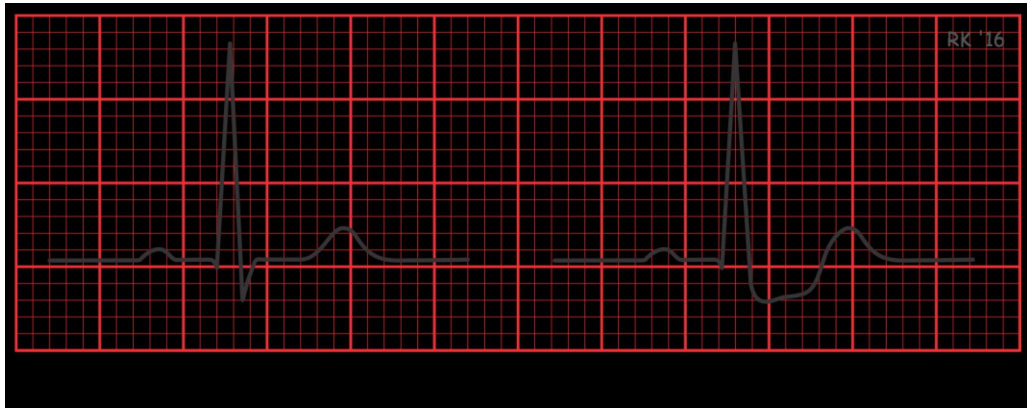
Prolonged QRS (>120) -BBB



The name William Morrow can help you identify LBBB and RBBB by looking at the QRS morphology in V_1 and V_6. In LBBB the QRS looks like a W in V_1 and an M in V_6 (WiLLiaM), in RBBB the QRS looks like an M in V_1 and a W in V_6 (MoRRoW).

ST segment elevation and depression

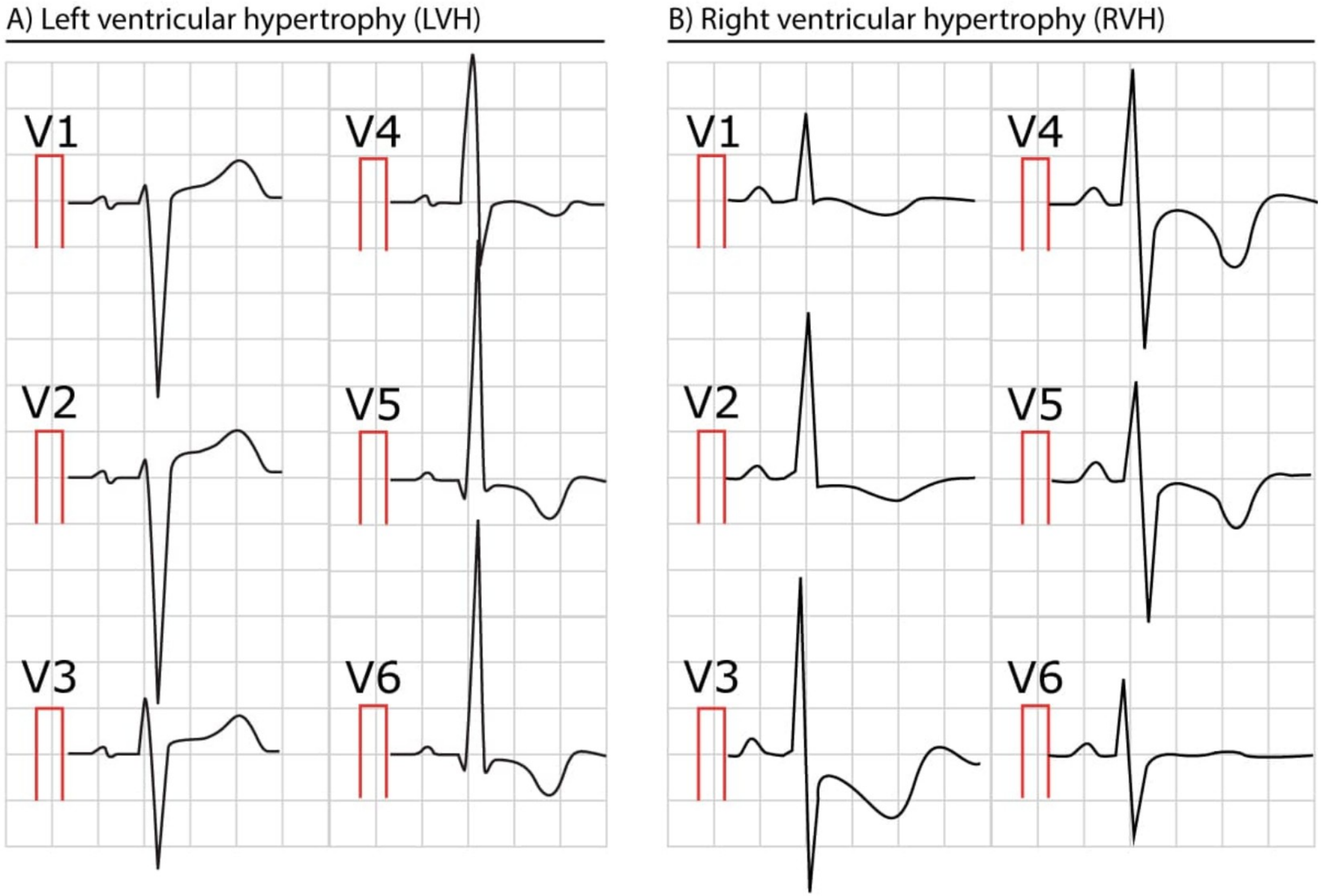




QRS amplitude

Left ventricular hypertrophy (LVH): $S_{V1\ or\ 2} + R_{V5\ or\ 6} \geq 3.5$ mV

Right ventricular hypertrophy (RVH): $R_{V1\ or\ 2} + S_{V5\ or\ 6} \geq 1.05$ mV

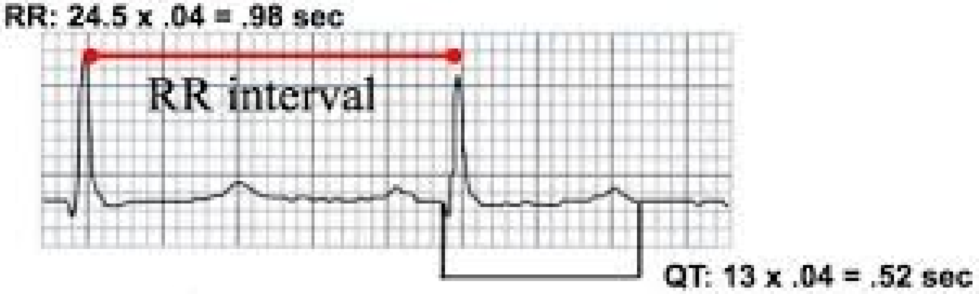


Corrected QT interval

Physiological

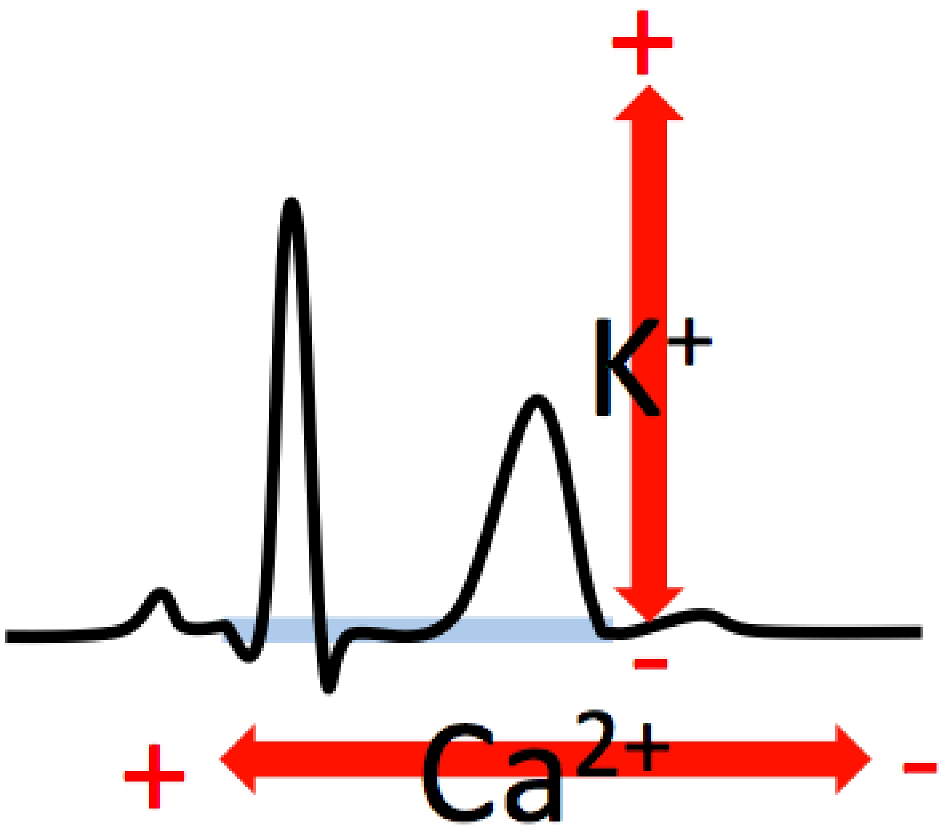
- Measured from the beginning of the Q wave to the end of the T wave
- Represents the entire duration of ventricular depolarization
- Varies with heart rate, so correction for the heart rate is necessary (=**QTc**)
  - QTc normally **< 350–440 ms**

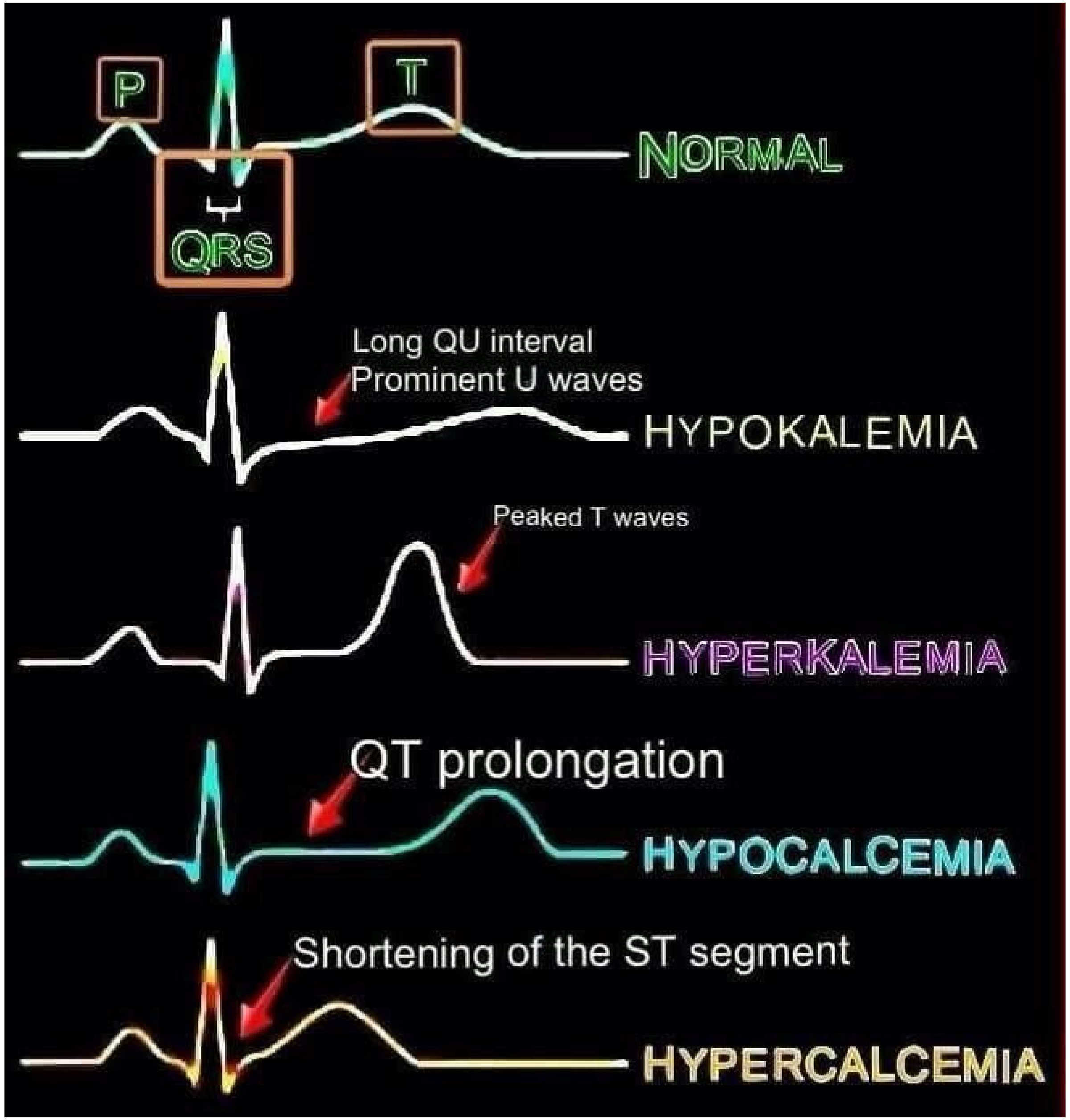
$$QTc = \frac{\overline{QT}\,(ms)}{\sqrt{RR\,(sec)}}$$



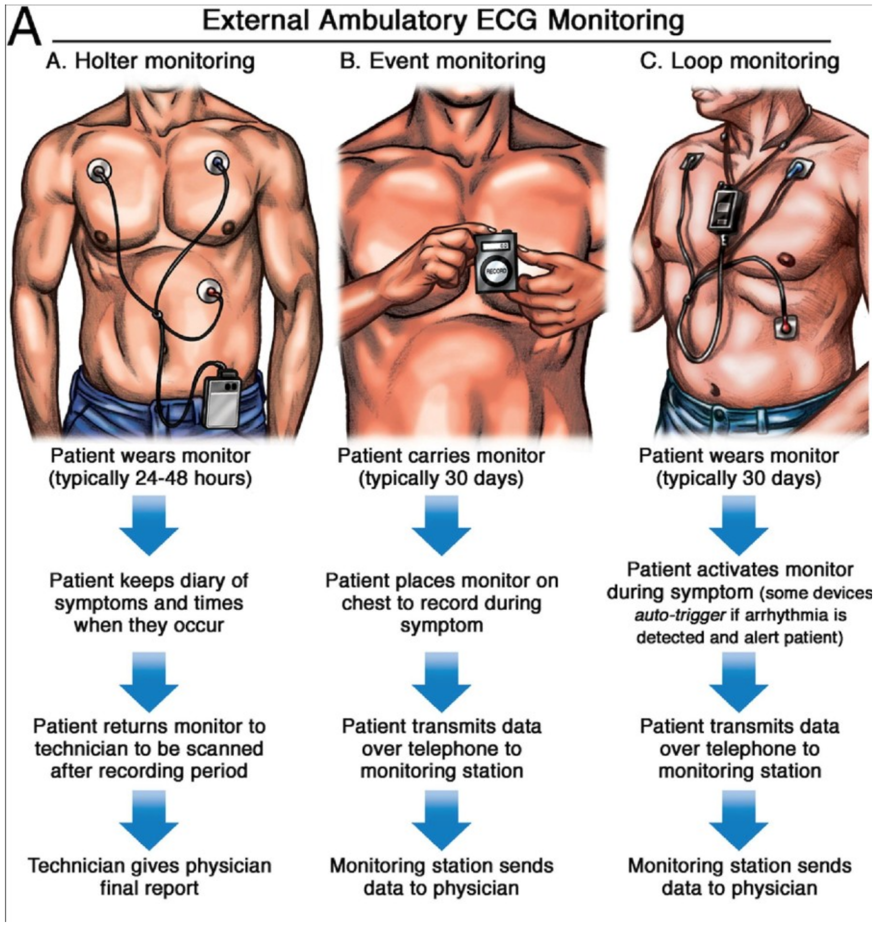
$$QTc = \frac{QT}{\sqrt{RR}} = \frac{.52}{\sqrt{.98}} = \frac{.52}{.989} = .525 \text{ or } .53 \text{ sec}$$

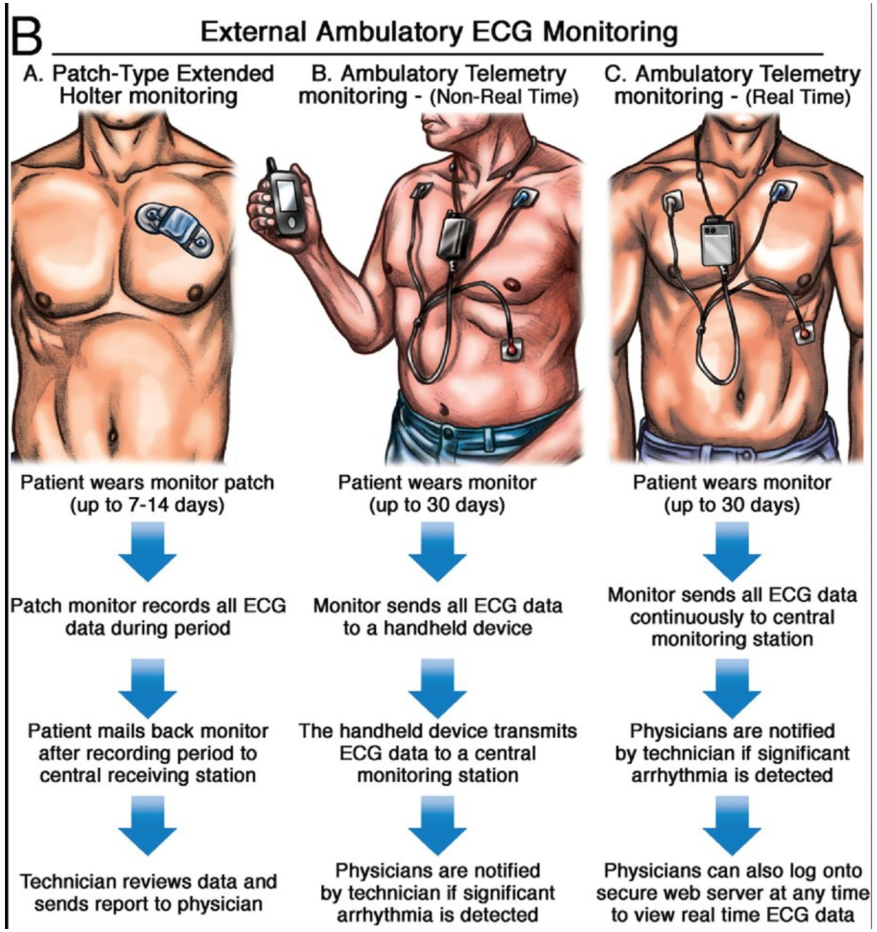
Electrolyte disturbances and ECG



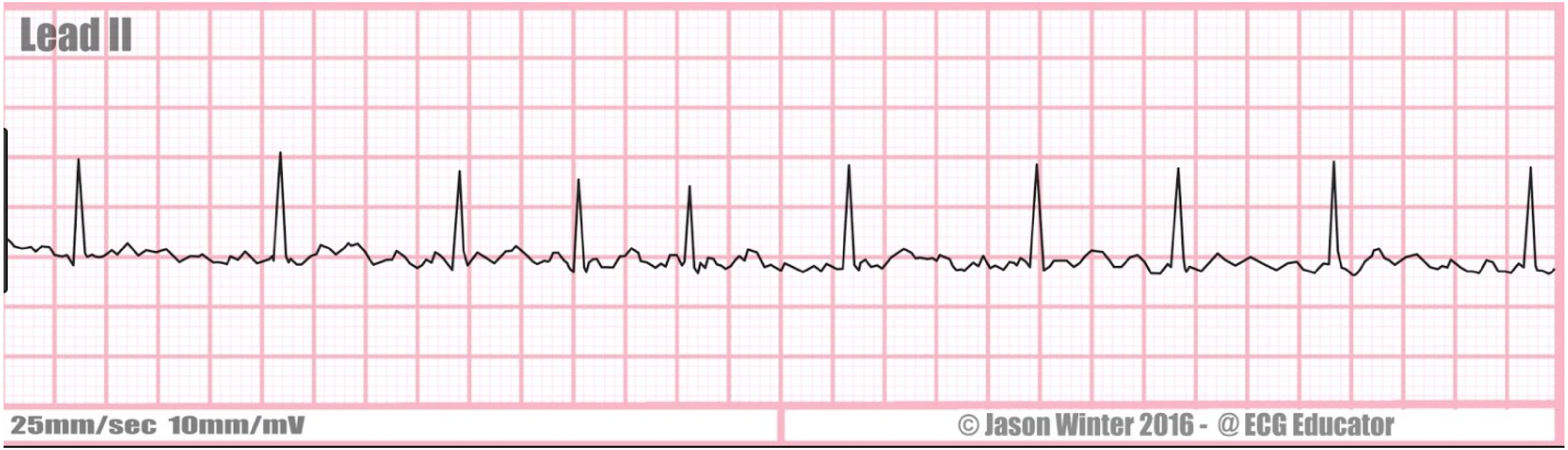

P
T
QRS
NORMAL
Long QU interval
Prominent U waves
HYPOKALEMIA
Peaked T waves
HYPERKALEMIA
QT prolongation
HYPOCALCEMIA
Shortening of the ST segment
HYPERCALCEMIA

Ambulatory monitoring





Quiz 1



Answer

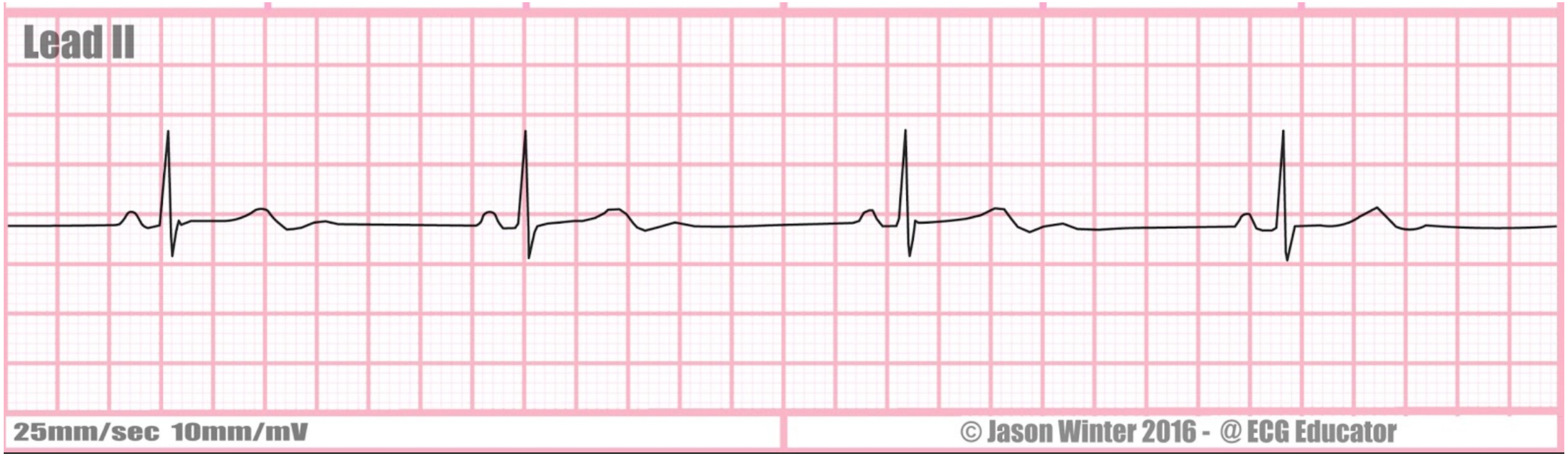
A-FIB

Quiz 2

Answer

AV blcok (mobitz 1)

Quiz 3



Answer

Sinus Bradycardia

Quiz 4

Answer

A-Flutter

Quiz 5

Answer

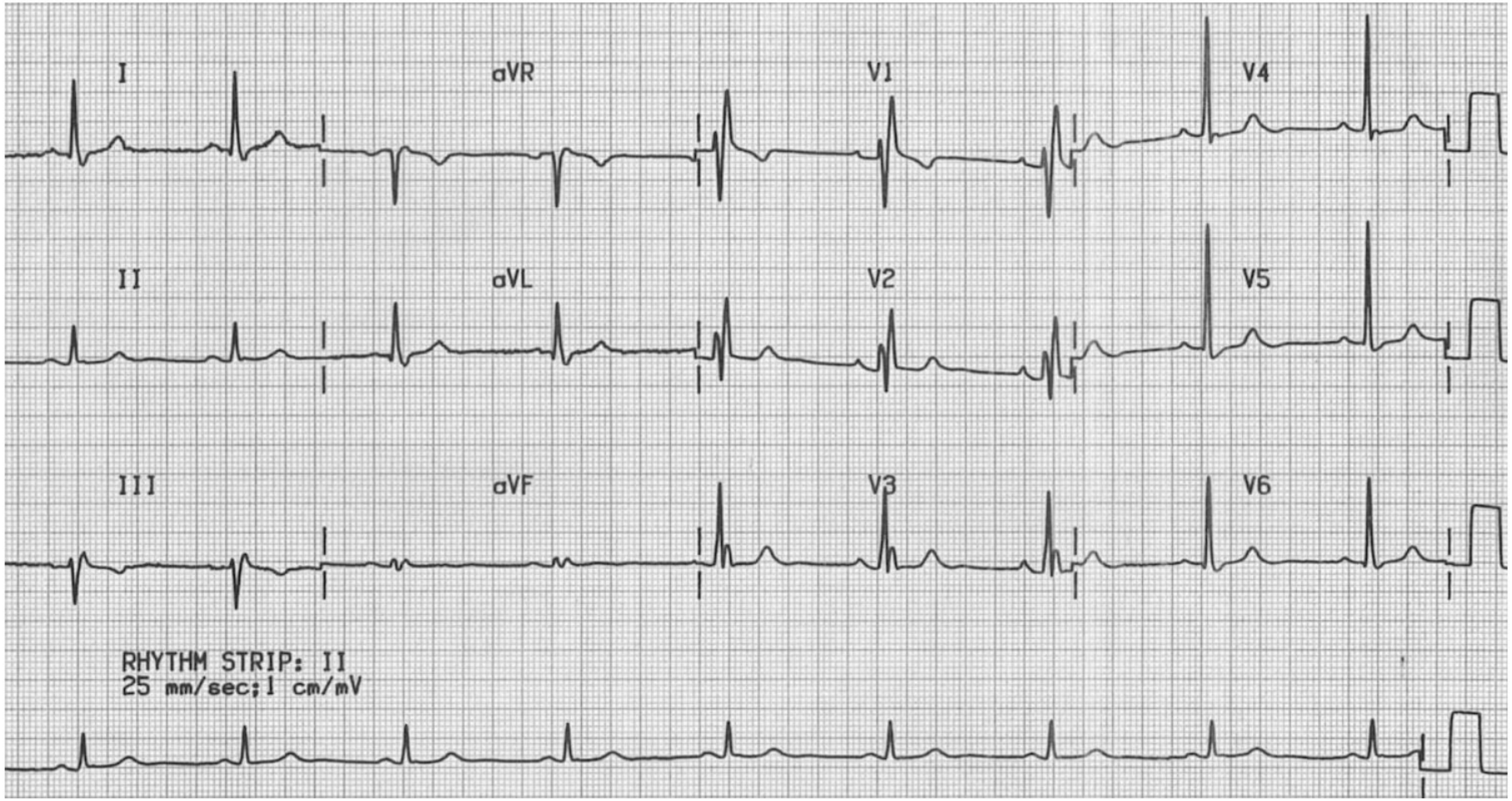
V-Fib

Quiz 6

Answer

Sinus Tachycardia

Quiz 7

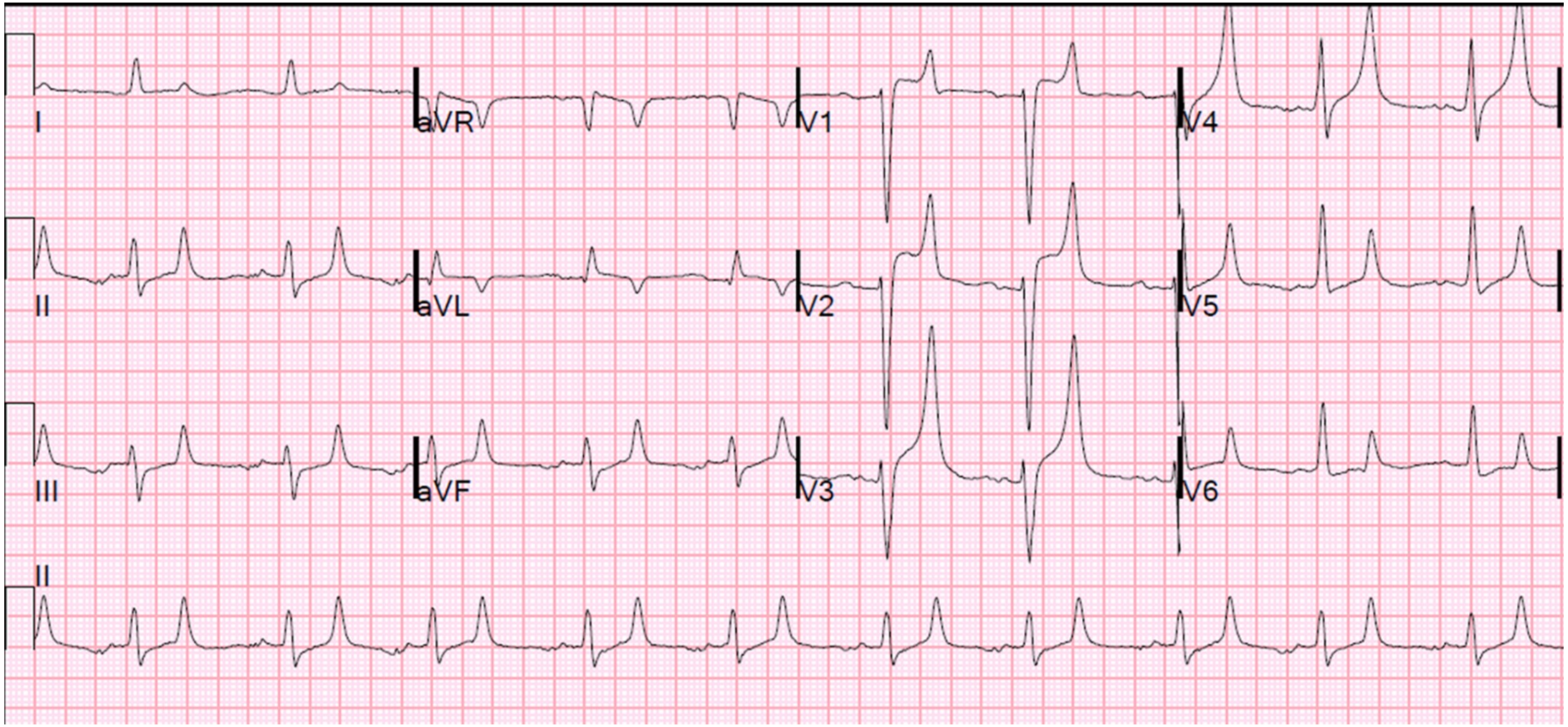


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
RHYTHM STRIP: II
25 mm/sec;1 cm/mV

Asnwer

RBBB

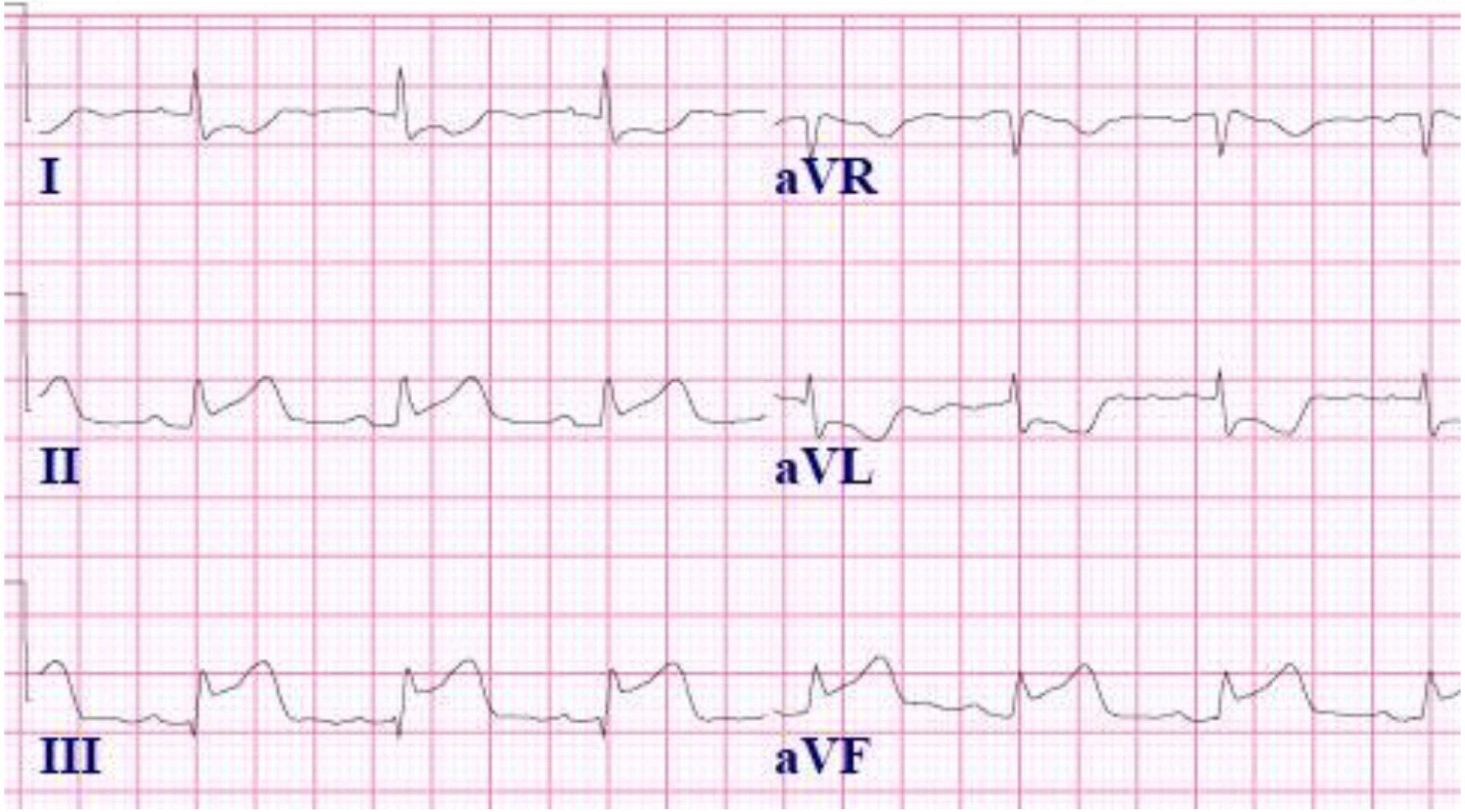
Quiz 8



Answer

HyperKalemia

Quiz 9



Answer

Inferior MI

THANK YOU

Resources ;

- RAPID INTERPRETATION OF ECG DALE DUBIN
- FIRST AID FOR USMLE 2020
- KAPLAN PHYSIOLOGY
- BORON PHYSIOLOGY