The cardiac muscle

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objectives

- **Cardiac action potential**
- **Pacemaker potential**
- **AV delay - significance**
- **ECG – different waves represent ?**
- **Frank starling law. Define preload**
- **HR regulation – factors**
- **Cardiac cycle – events list**
- **Cardiac contractility is increased under the influence of hormone Norepinephrine.**

Functions of the Heart

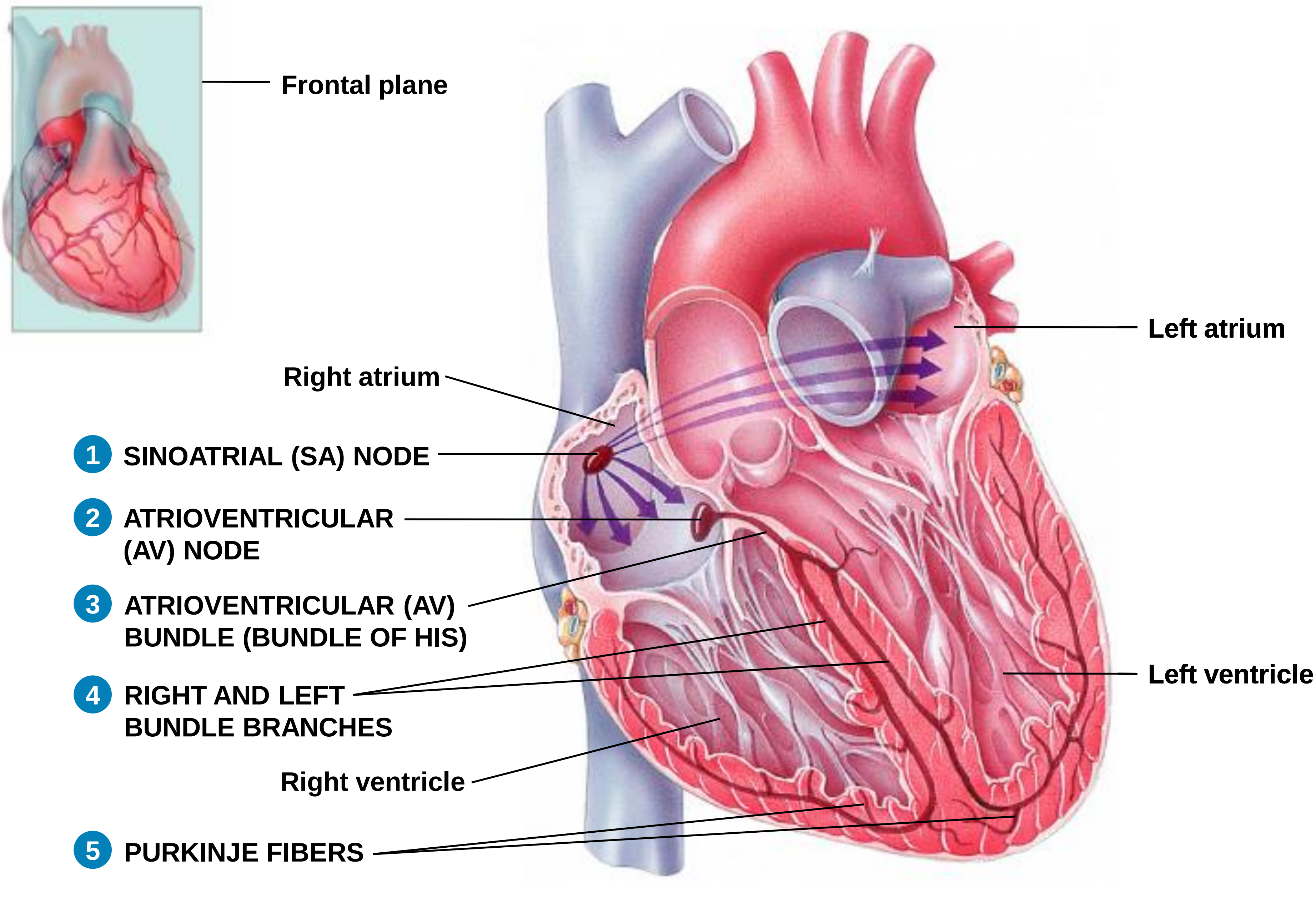
- Generating blood pressure
- Routing blood: separates pulmonary and systemic circulations
- Ensuring one-way blood flow: valves
- Regulating blood supply
  - Changes in contraction rate and force match blood delivery to changing metabolic needs
  - Exercise
  - Sleeping
  - Sitting

Autorhythmic Fibers

- Specialized cardiac muscle fibers
- Self-excitable
- Repeatedly generate action potentials that trigger heart contractions
- 2 important functions
  1. Act as pacemaker
  2. Form conduction system

Conduction system

1. Begins in sinoatrial (SA) node in right atrial wall
   - Propagates through atria via gap junctions
   - Atria contract
2. Reaches atrioventricular (AV) node in interatrial septum
3. Enters atrioventricular (AV) bundle (Bundle of His)
   - Only site where action potentials can conduct from atria to ventricles due to fibrous skeleton
4. Enters right and left bundle branches which extends through interventricular septum towards apex
5. Finally, large diameter Purkinje fibers conduct action potential to remainder of ventricular myocardium
   - Ventricles contract



Anterior view of frontal section

Conduction System

- SA node acts as natural pacemaker
  - Faster than other autorhythmic fibers
  - Initiates 100 times per second
- Nerve impulses from autonomic nervous system (ANS) and hormones modify timing and strength of each heartbeat
  - Do not establish fundamental (constant) rhythm

Action Potentials and Contraction

- Action potential initiated by SA node spreads out to excite "working" fibers called contractile fibers

1. Depolarization
2. Plateau
3. Repolarization

Action Potentials and Contraction

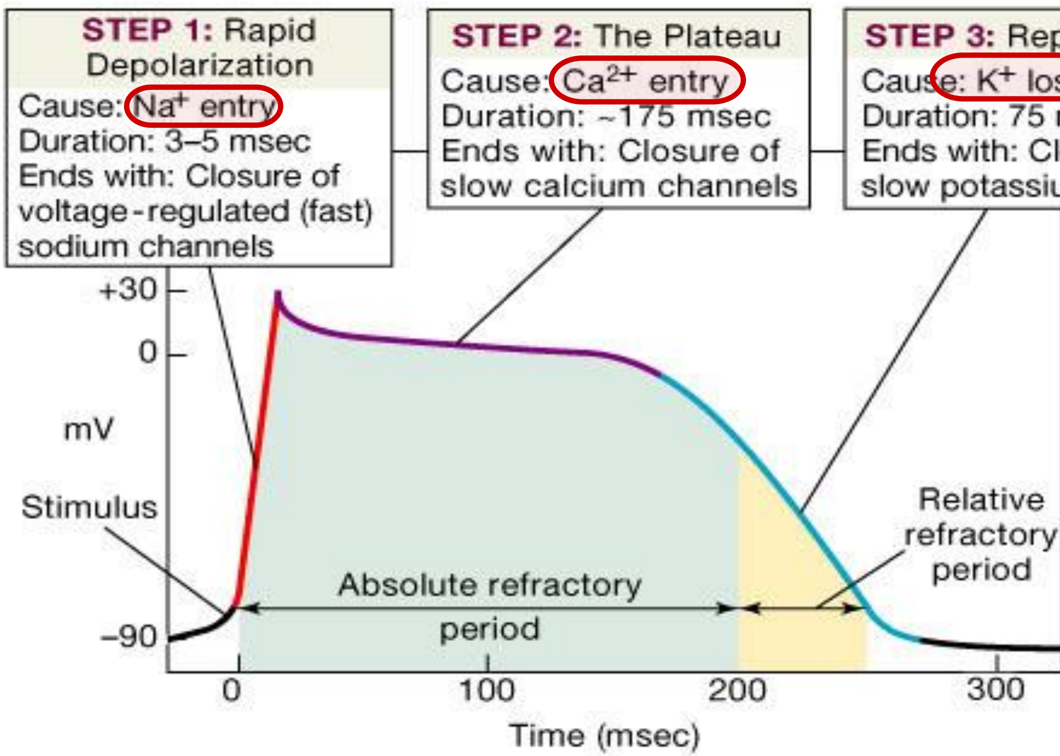
1. Depolarization – contractile fibers have stable resting membrane potential
   - Voltage-gated fast Na^+ channels open – Na^+ flows in
   - Then deactivate and Na^+ inflow decreases
2. Plateau – period of maintained depolarization
   - Due in part to opening of voltage-gated slow Ca^{2+} channels – Ca^{2+} moves from interstitial fluid into cytosol
   - Ultimately triggers contraction
   - Depolarization sustained due to voltage-gated K^+ channels balancing Ca^{2+} inflow with K^+ outflow

Action Potentials and Contraction

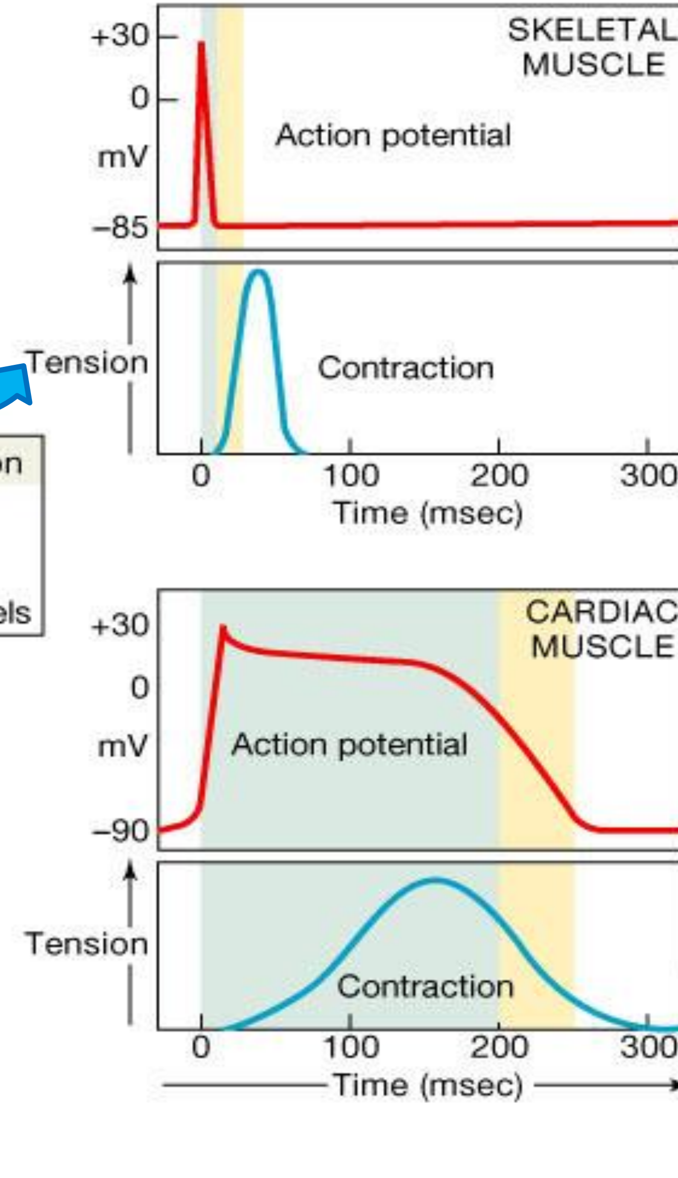
3. Repolarization – recovery of resting membrane potential
    - Resembles that in other excitable cells
    - Additional voltage-gated K^+ channels open
    - Outflow of K^+ restores negative resting membrane potential
    - Calcium channels closing

- Refractory period – time interval during which second contraction cannot be triggered
    - Lasts longer than contraction itself
    - Hence Tetanus (maintained contraction) cannot occur
        - Then, Blood flow would cease

TWITCH

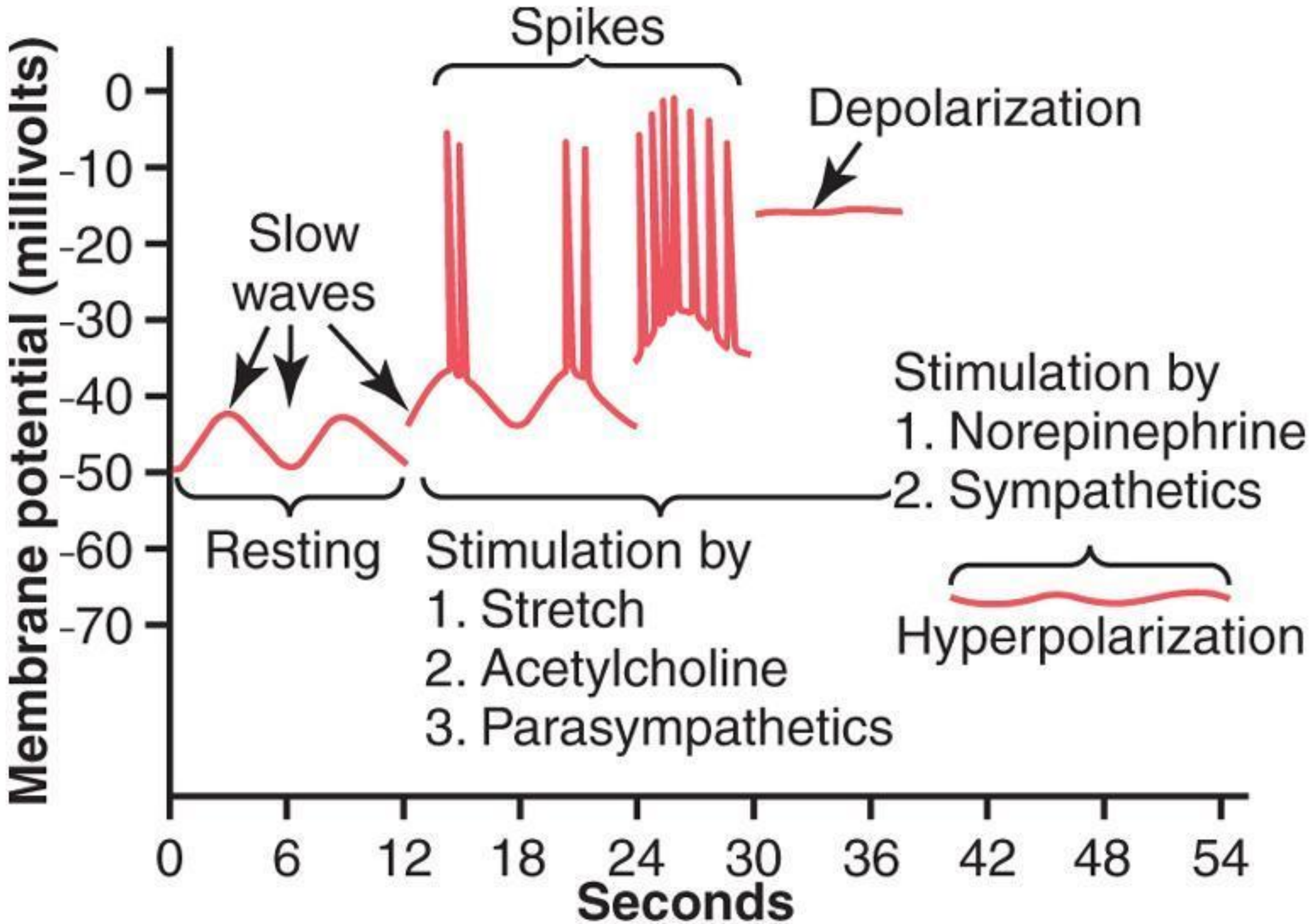


(a) Cardiac muscle



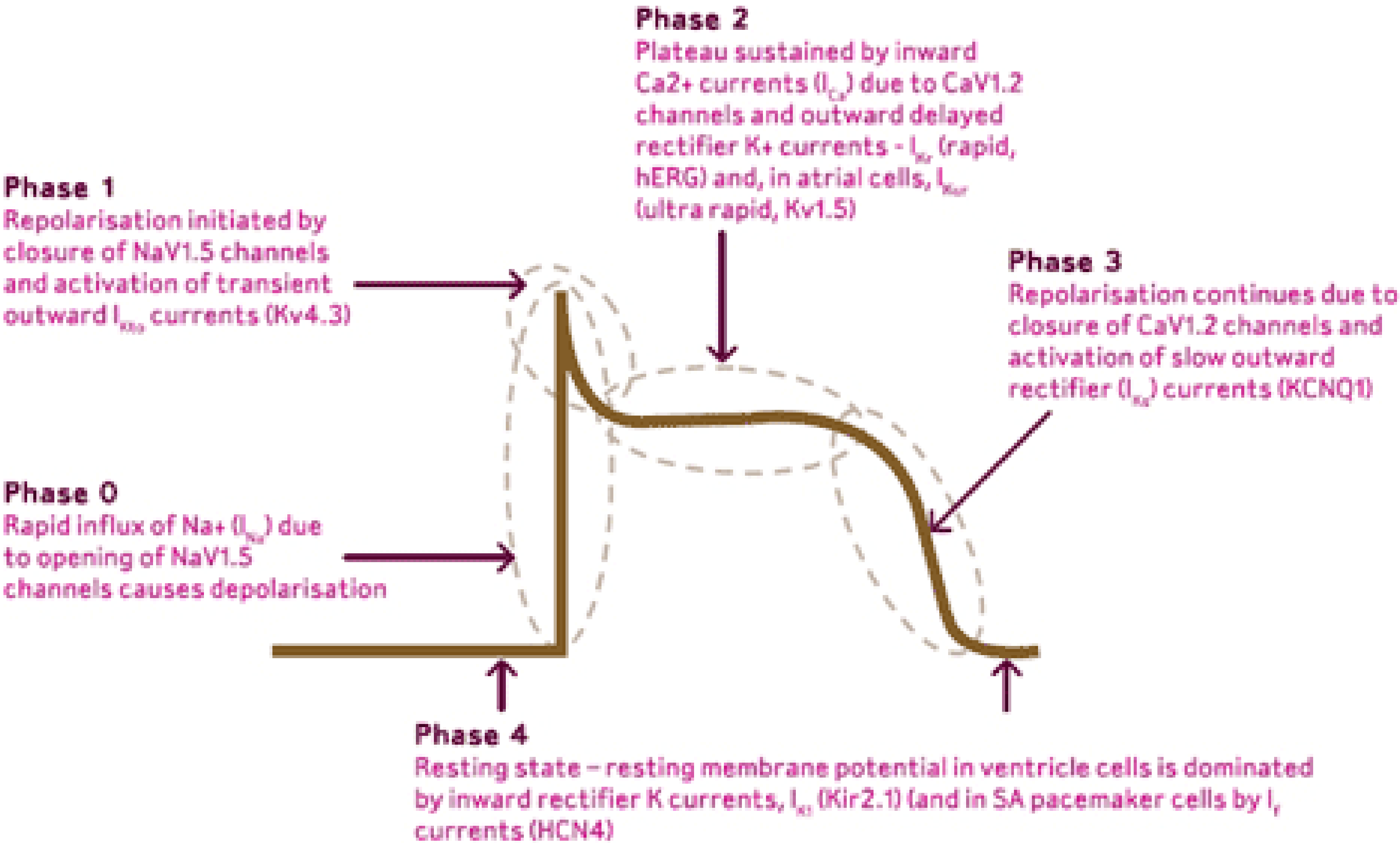
(b)

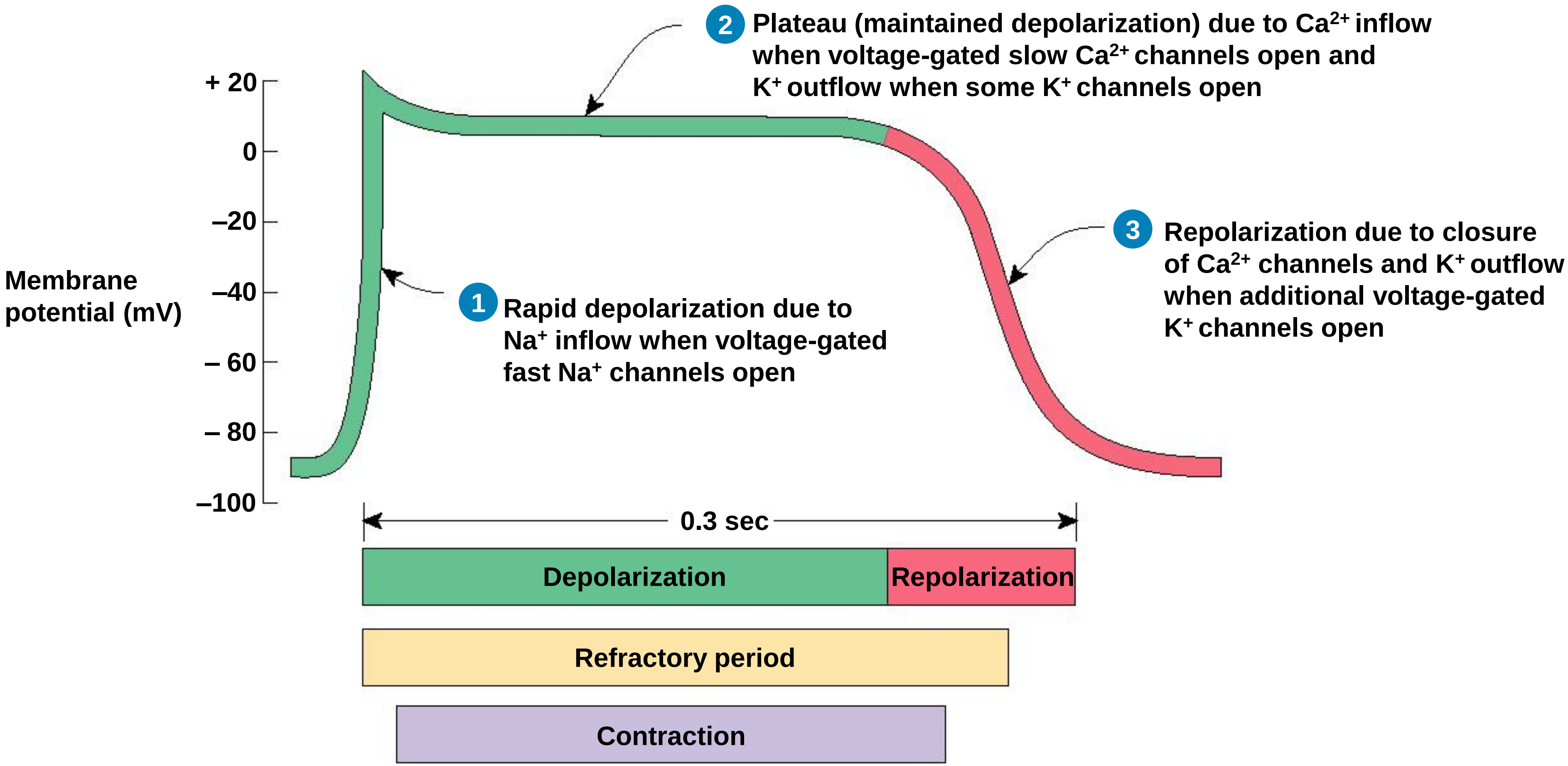
Smooth muscles action potentials in Gut



Guyton’s textbook

cardiac


Phase 2
Plateau sustained by inward Ca2+ currents (I_{Ca}) due to CaV1.2 channels and outward delayed rectifier K+ currents - I_{Kr} (rapid, hERG) and, in atrial cells, I_{Kur} (ultra rapid, Kv1.5)
Phase 1
Repolarisation initiated by closure of NaV1.5 channels and activation of transient outward I_{Kto} currents (Kv4.3)
Phase 3
Repolarisation continues due to closure of CaV1.2 channels and activation of slow outward rectifier (I_{Ks}) currents (KCNQ1)
Phase 0
Rapid influx of Na+ (I_{Na}) due to opening of NaV1.5 channels causes depolarisation
Phase 4
Resting state – resting membrane potential in ventricle cells is dominated by inward rectifier K currents, I_{K1} (Kir2.1) (and in SA pacemaker cells by I_f currents (HCN4)



Refractory period

When a muscle fiber contracts, it temporarily cannot respond to another action potential

Skeletal muscle has a refractory period of 5 milliseconds

Cardiac muscle has a refractory period of 300 milliseconds- so no tetanus.

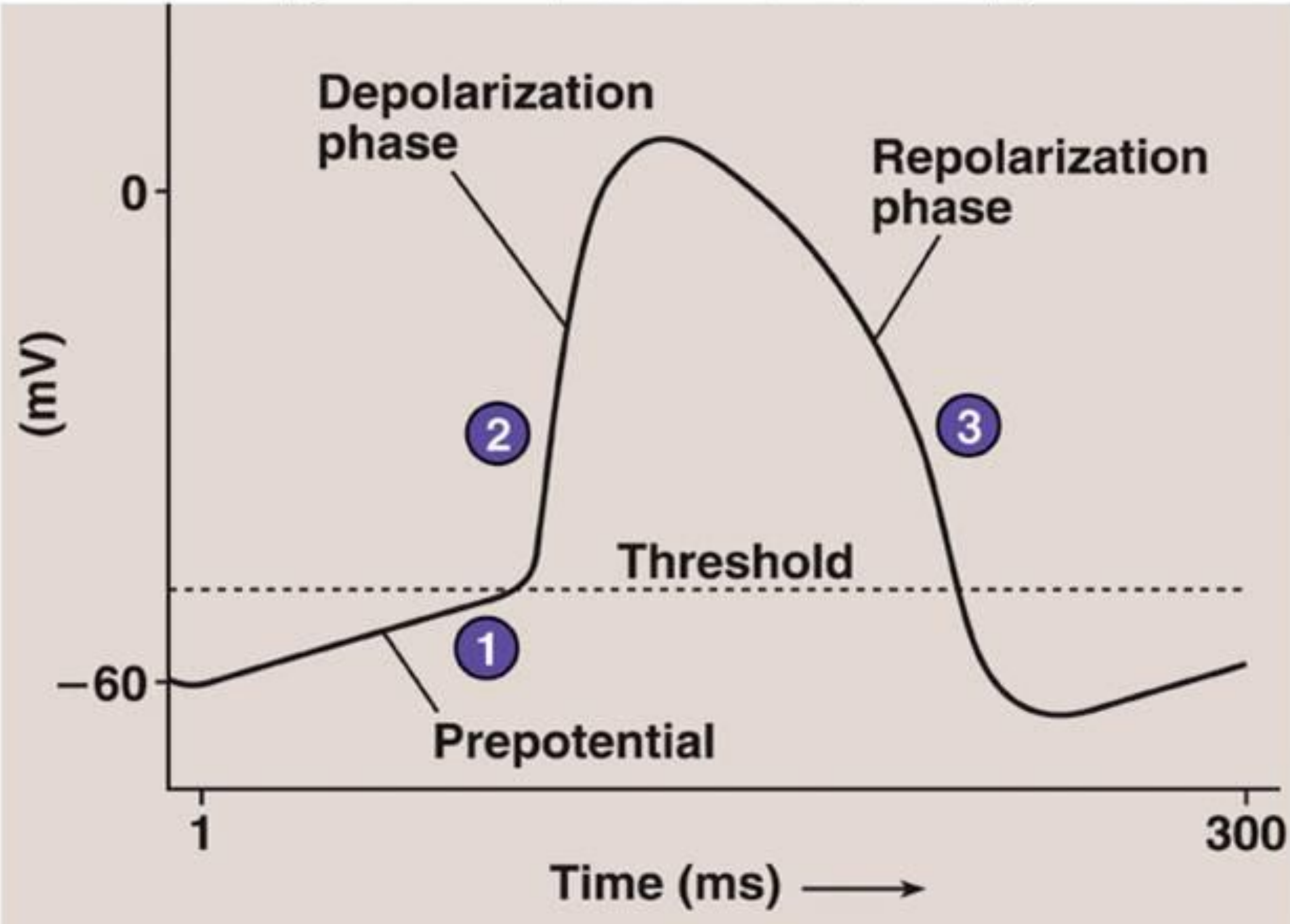
- because Na^+ channels are inactivated during this time, additional depolarizing stimuli do not lead to new action potentials.

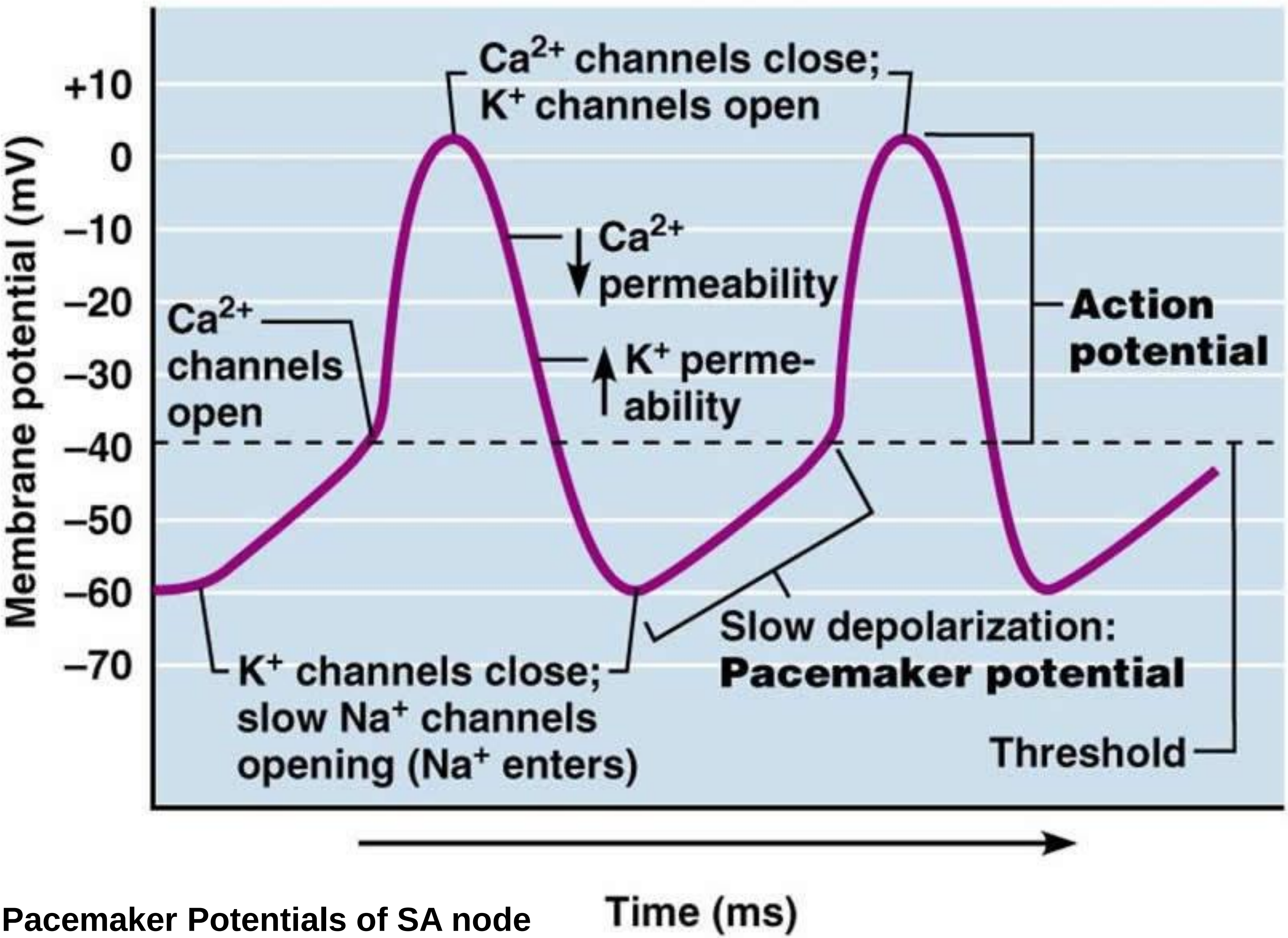
- Cardiac muscle cannot be tetanised because of long refractory period

Mechanism of Sinus nodal rhythmicity

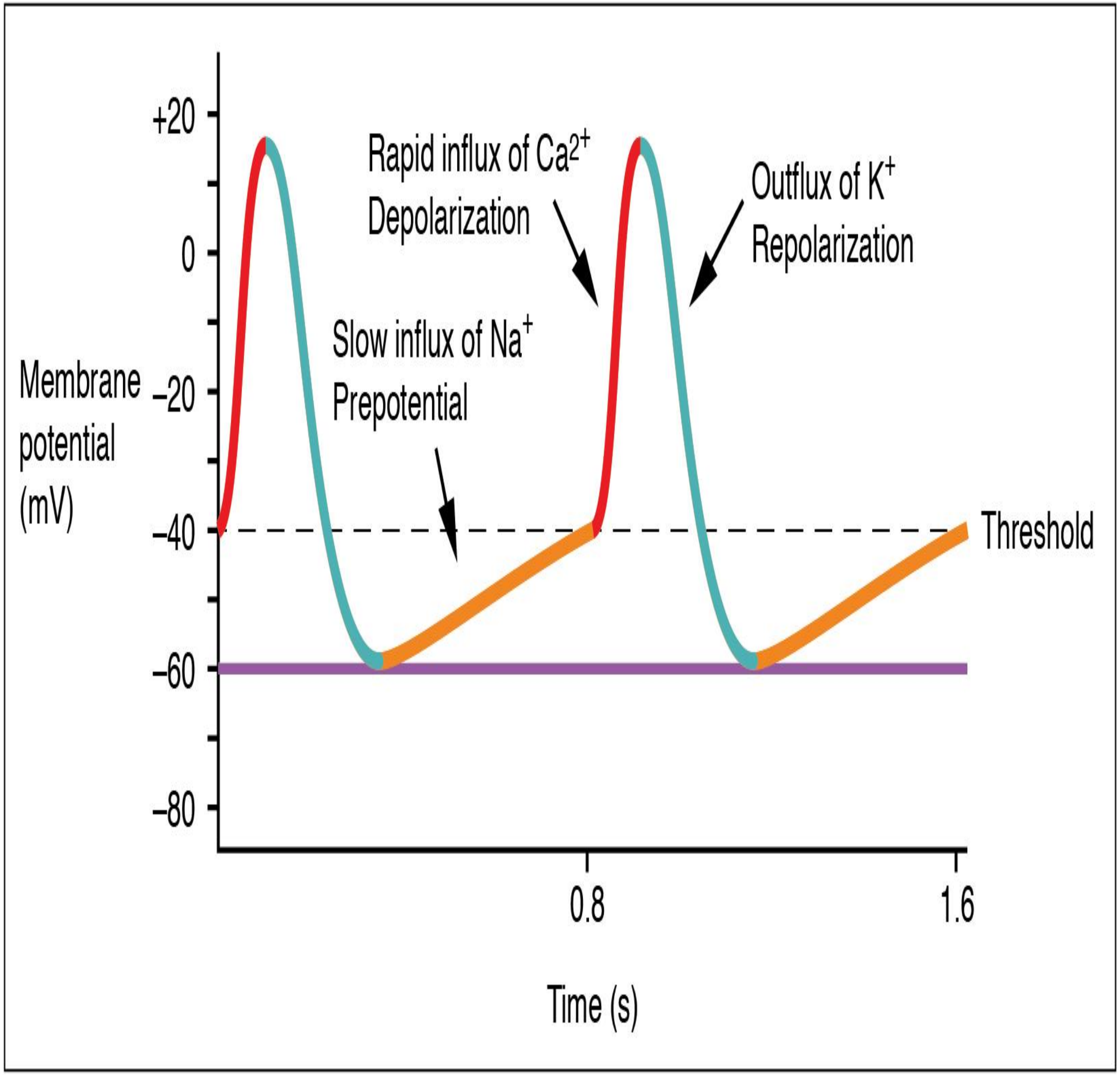
- A process that cause automatic rhythmical discharge and contraction.
- RMP of sinus nodal fiber (SAN) is -55 to -60mv.(unstable RMP)
- **The inherent leakiness of sinus nodal fibers to sodium and calcium ions cause their self excitation.**

Only in living cells


Depolarization phase
Repolarization phase
0
(mV)
−60
2
3
Threshold
1
Prepotential
1
300
Time (ms)



Pacemaker Potentials of SA node



+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)

- Pacemaker potential or Pre-potential -

The slow rising RMP in between action potentials is k/a pacemaker potential/prepotential.

Observe this heart !!

Are both atria and ventricles beating at the same time ?

What makes the heart to contract atria first then the ventricles?

AV node

The AV node is located in the posterior wall of the right atrium immediately behind the tricuspid valve.

AV nodal Delay

- Due to the slow conduction in AV node there is a delay of **0.09** sec in the AV node, before the impulse reaches the penetrating portion of AV bundle.
- There is a delay of **0.04** sec in the penetrating portion of AV bundle.
- There is a total delay of **0.13** sec in the AV node and AV bundle system.
- There is a delay of **0.03** sec from the sinus node to the AV node, thus making **total delay of 0.16 sec** before the excitatory signal finally reaches the contracting muscle of ventricles.

Cause of Slow Conduction in the A-V Node

The cause of slow conduction is mainly due to diminished number of gap junctions between the successive cells in the conducting pathways.

As a result of which there is great resistance to conduction of excitatory ions from one conducting fiber to the next.

Significance of AV nodal delay

- The cardiac impulse does not travel from the atria to the ventricles too rapidly.
- This delay allows time for the atria to empty their blood into the ventricles before ventricular contraction begins. This increases the efficiency of the pumping action of the heart.
- It is primarily the AV node and it's adjacent fibers that delay this transmission into the ventricles

AV Bundle or Bundle of His

- From the AV node arises a special conducting pathway called the <u>bundle of His</u>.
- Except for the very small part which <u>penetrates</u> through the AV <u>fibrous tissue</u> and has low conduction velocity, the bundle of His gives rise purkinje fibers which possess maximum conduction velocity in the heart.

- Purkinje fibers are very large fibers and they transmit action potentials at a velocity of 1.5 to 4.0 m/sec.
- The rapid transmission of action potentials through the Purkinje fibers is believed to be caused by a very high level of permeability of gap junctions at the intercalated discs between the successive cells of Purkinje fibers.
- The rapid conduction through the purkinje fibers ensures that different parts of ventricles are excited almost simultaneously; this greatly increases the efficiency of heart as a pump.

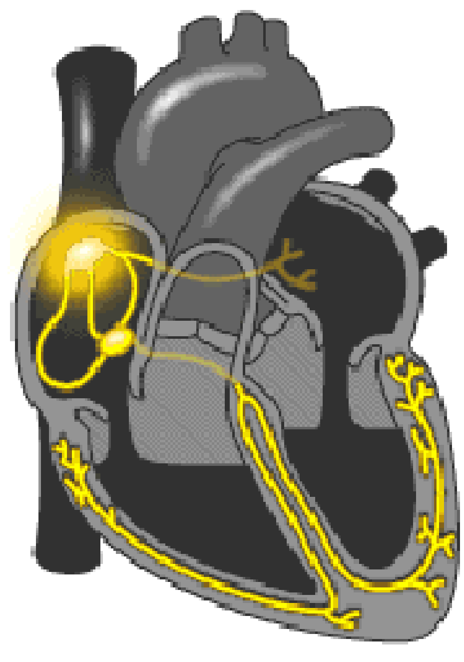
- Normally the Bundle of His is the only conducting mass *between* the atrial and ventricular musculature and it transmits the cardiac impulses from the AV node to the ventricles.

Right and Left Bundle Branches

- After penetrating the fibrous tissue between the atrial and ventricular muscle, the distal portion of the A-V bundle passes downward in the ventricular septum for 5 to 15 mm toward the apex of the heart.
- Then the bundle of His splits into two branches which are called right and left bundle branches that lie on the respective sides of the ventricular septum.

- Each branch spreads downward toward the apex of the ventricle, progressively dividing into smaller branches.
- These branches in turn course sidewise around each ventricular chamber and back toward the base of heart.
- The ends of Purkinje fibers penetrate about one third of the way into muscle mass and finally become continuous with cardiac muscle fibers.

- From the time the cardiac impulse enters the bundle branches until it reaches the terminations of Purkinje fibers , the total elapsed time averages only 0.03 sec.



- **Observe atria, then the ventricles**

One- way Conduction through AV bundle

- A special characteristic of the A-V bundle is it’s inability, except in the abnormal states , of action potentials to travel backward from the ventricles to the atria.
- This prevents re-entry of cardiac impulse by this route from the ventricles to the atria.
- The atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier which acts as an insulator to prevent the passage of cardiac impulse between the atrial and ventricular muscle through any other route besides forward conduction through A-V bundle itself.

Conduction in the Cardiac Muscle

- Once the impulse reaches the ends of the Purkinje fibers it is transmitted through the ventricular muscle mass by the ventricular muscle fibers themselves.
- For transmission of the cardiac impulse from the endocardial surface to the epicardial surface recquires another 0.03 sec.
- Thus the total time for transmission of cardiac impulse from the initial bundle branches to the last of the ventricular muscle fibers in the normal heart is about 0.06 sec.

Conduction speed in Cardiac tissues

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

Normal Rate of Action potential Discharge in

Tissue	Action Potentials Per Minute
SA node (normal pacemaker)	70-80
AV node	40-60
Bundle of His and Purkinje fibers	20-40

- **Sinoatrial (SA) node** normally generates the action potential, i.e. the electrical impulse that initiates contraction.
  - The SA node excites the right atrium (RA), travels through Bachmann's bundle to excite left atrium (LA).
  - The impulse travels through internodal pathways in RA to the **atrioventricular (AV) node.**

Questions ?

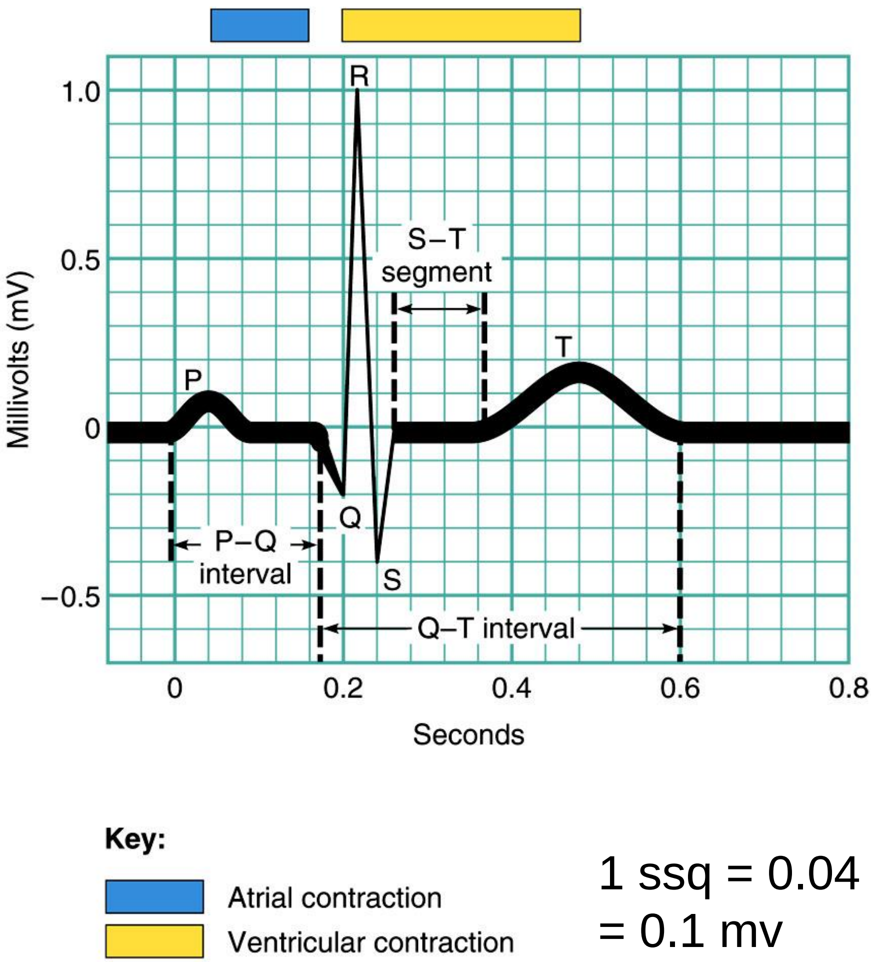
Does the action potential spreads from atrial muscles to the ventricles directly?

Learning objectives

- Limb leads- I,II,III
- Augmented limb leads- avR, avL, avF
- Unipolar chest leads- V1-9
- Positive wave
- Negative wave
- Vector
- Mean Cardiac axis

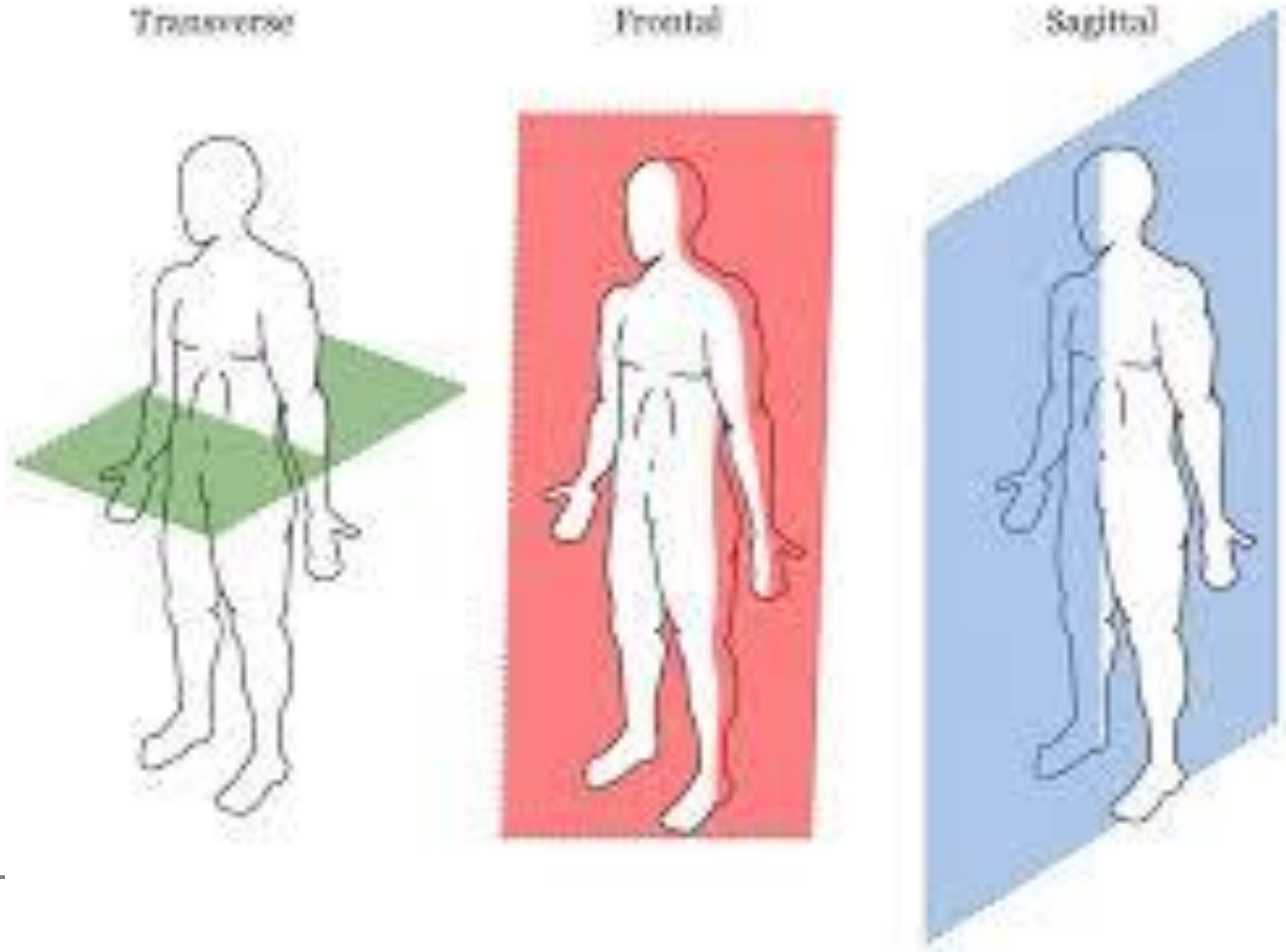
Electrocardiogram

- ECG or EKG
- Composite record of action potentials produced by all the heart muscle fibers from skin surface
- Compare tracings from different leads with one another and with normal records
- 3 recognizable waves
  - P, QRS, and T

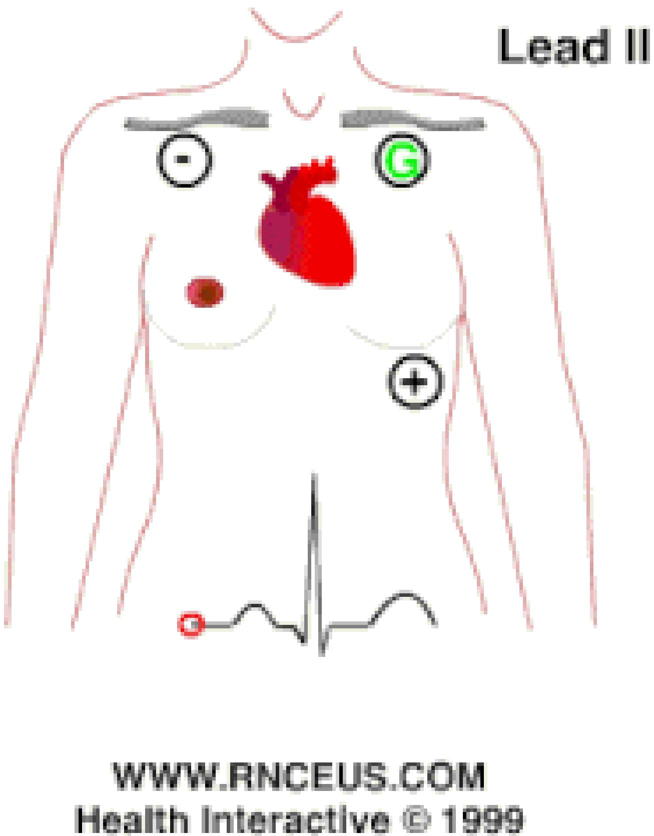


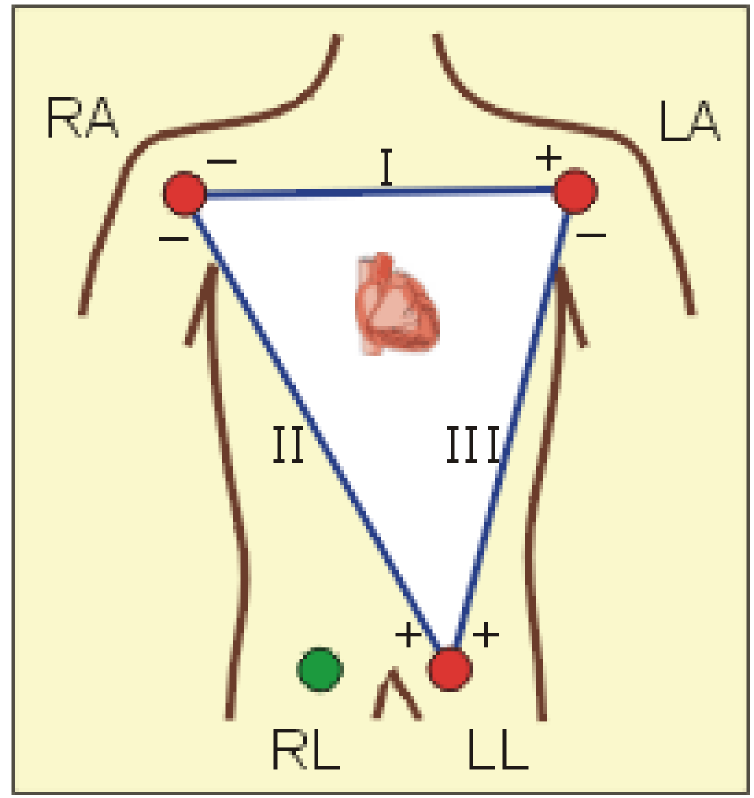
1 ssq = 0.04 sec
= 0.1 mv

TRANSVERSE PLANE **FRONTAL PLANE**



Einthoven's triangle connection leads

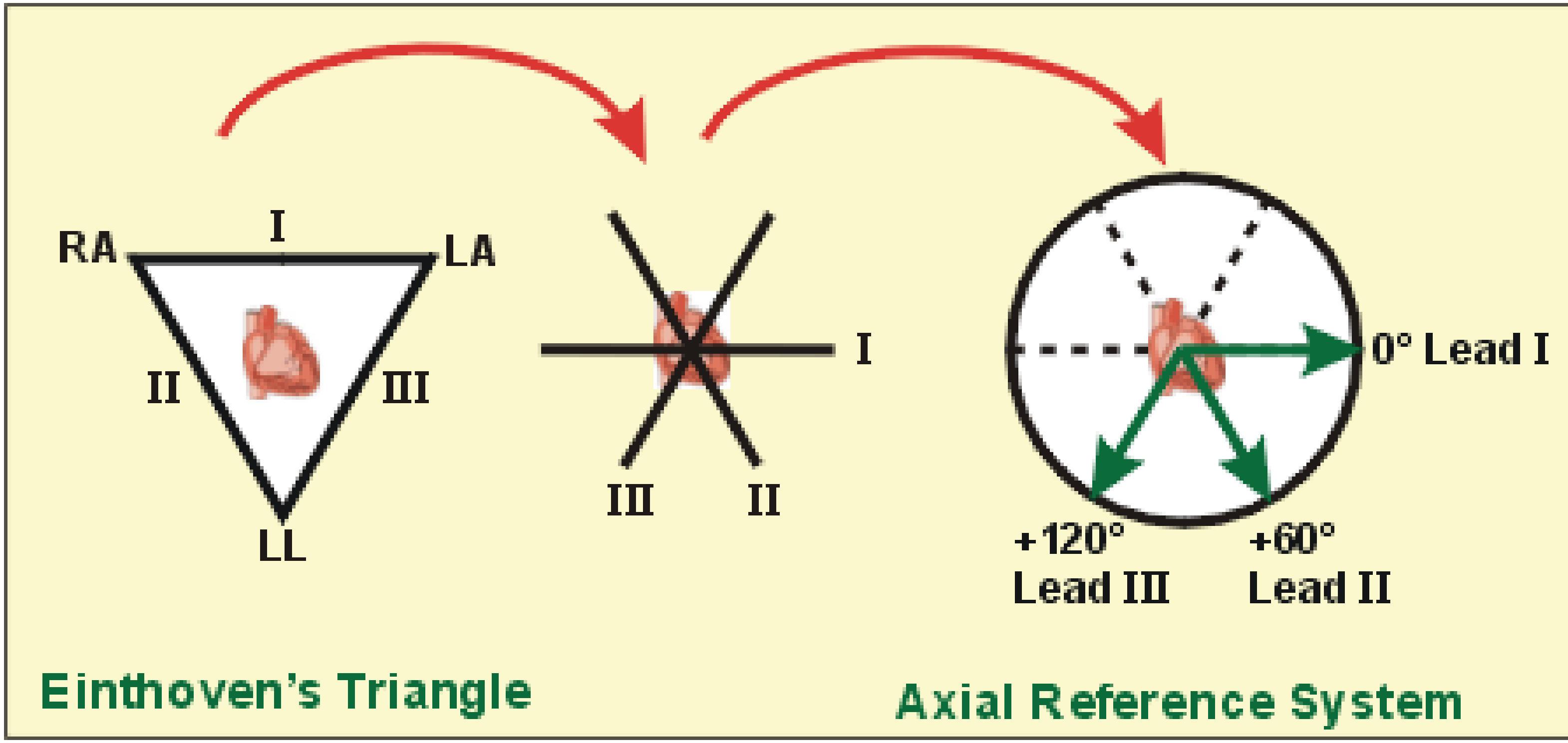




Electrocardiogram - paper

- **Depolarization – if towards lead II – positive wave**
- **Away from lead II – negative wave**
- **Repolarization – if towards lead II – negative wave**
- **Away lead II – positive wave**

- **Vector = used to determine the direction of depolarization (or. repolarization) of the atria and ventricles**
- **Cardiac axis = mean direction of the wave of ventricular depolarization (QRS) in the frontal plane**
- **Dipole = one positive and one negative charge at a place**

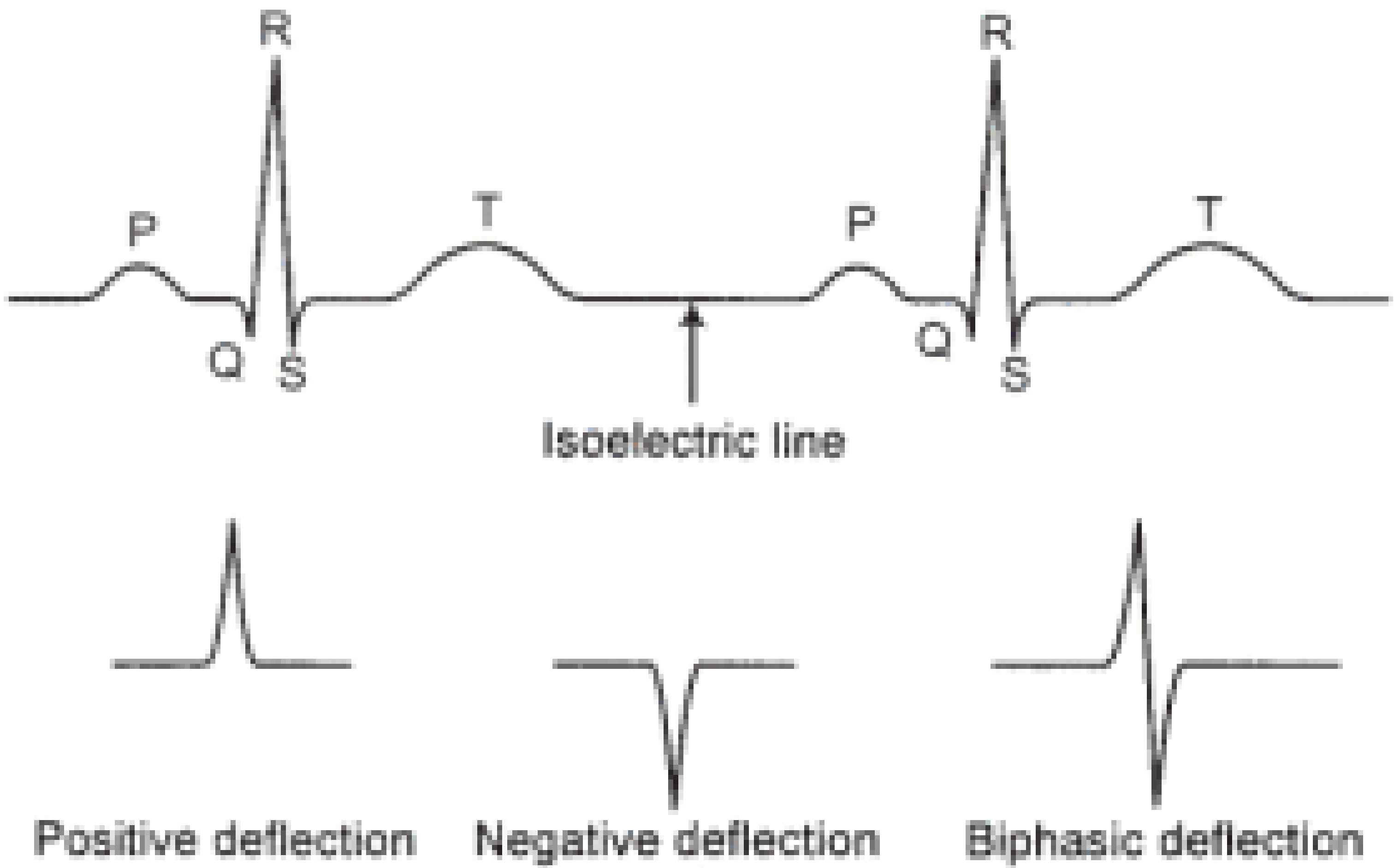


Triangle is anti clockwise I,II,III

Position & Axis is clockwise I,II,III

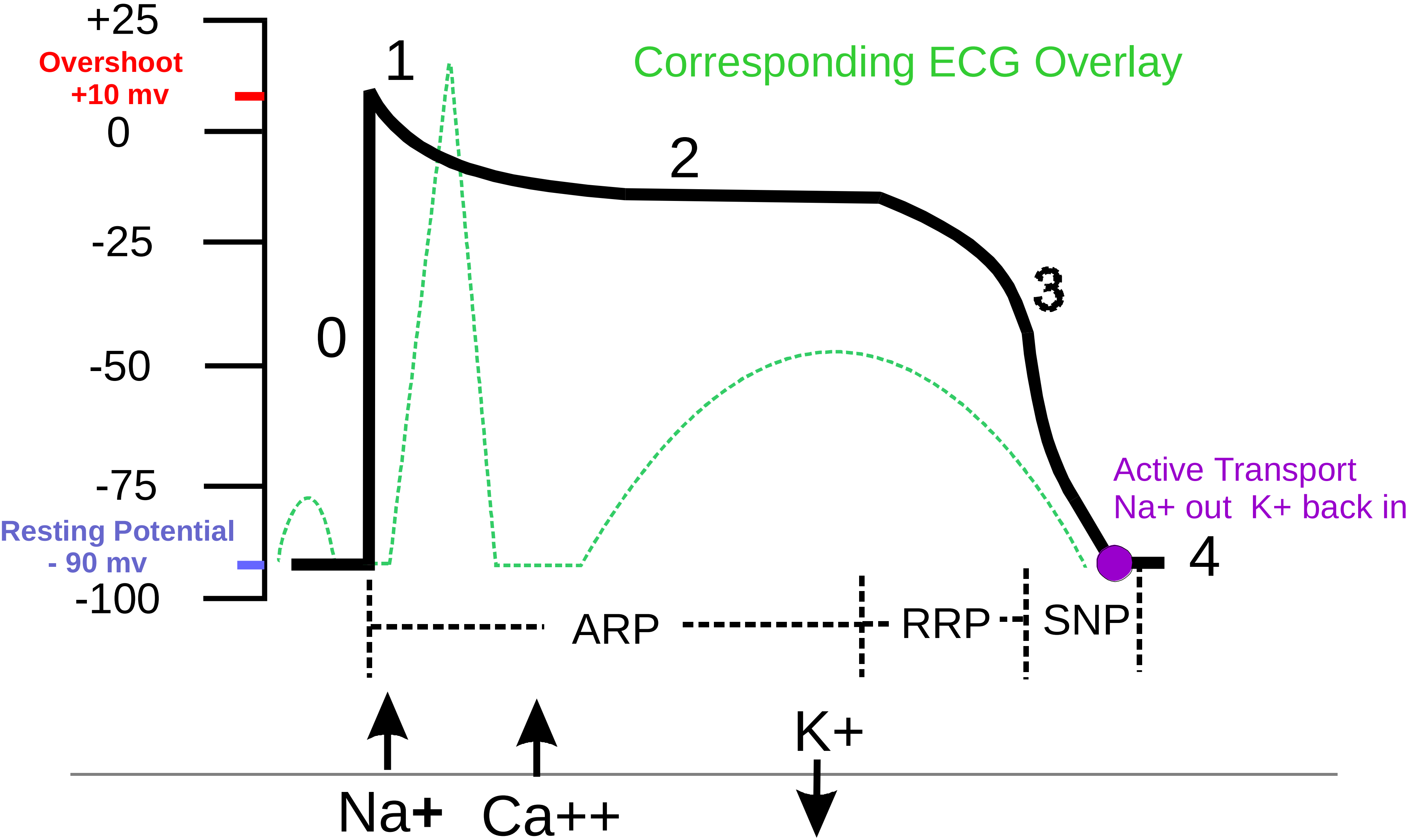
- P wave = discharge spreads over atria
- Q wave = interventricular septum depolarizes from left to right
- R wave = from sub endocardial muscle to subepicardial layer
- S wave = posterobasal portion of L ventricle
- T wave = repolarization – occurs from epicardial to endocardial surface is slower than depolarization

Waves in ECG - draw in book



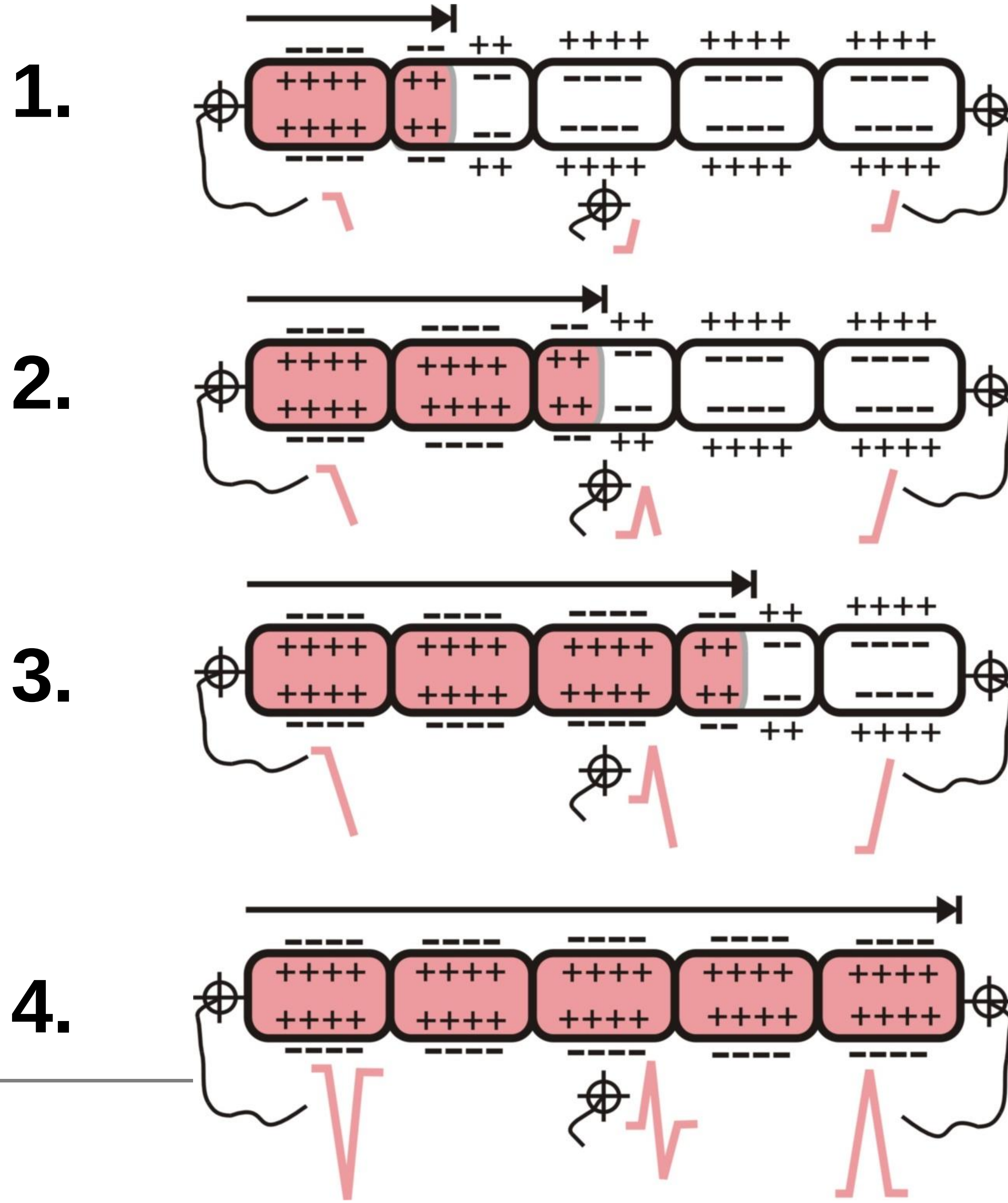
Relationship between waveforms and the isoelectric line.

Myocardium Muscle Action Potential



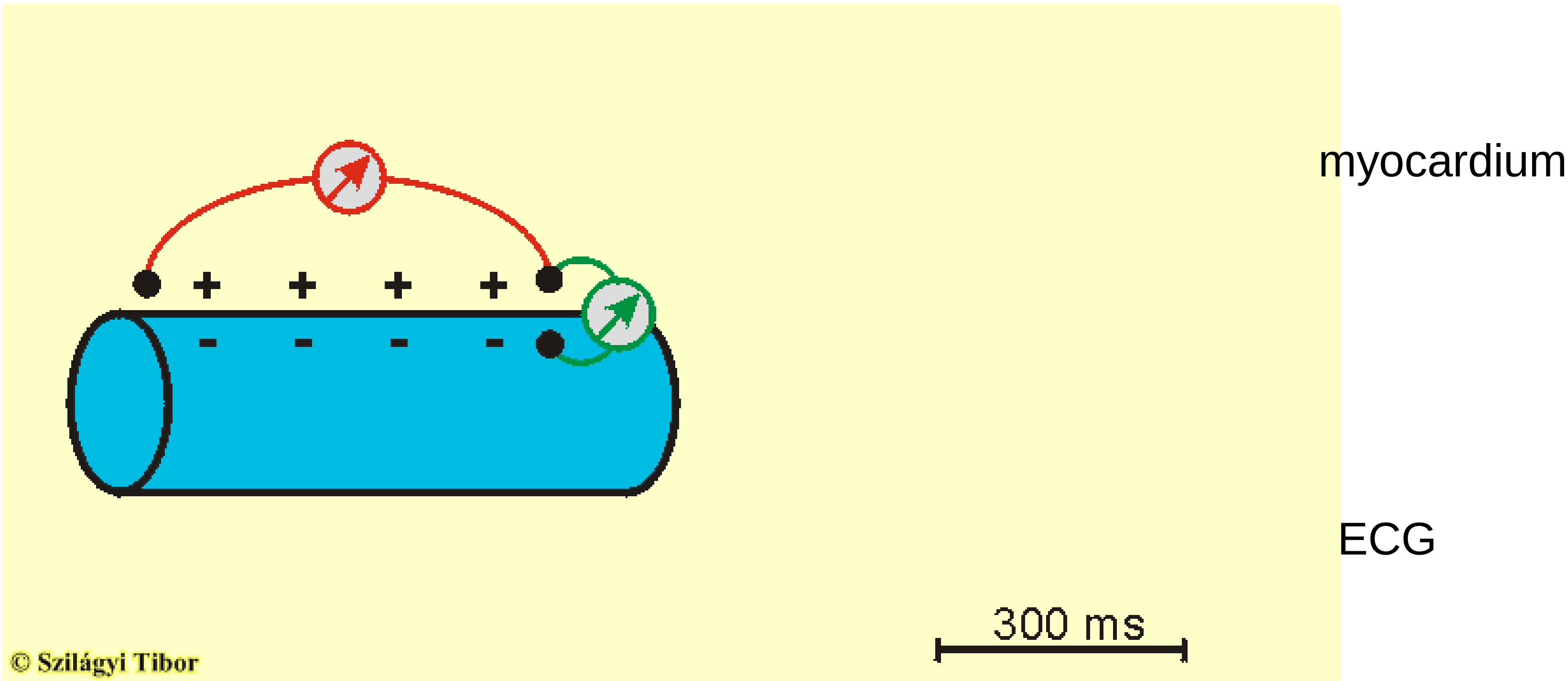
Concept 1

Depolarization progressing from left to right



Depolarization Sequence of a "Strip" of 5 Myocardial Cells And 3 leads

Genesis of depolarization in ECG



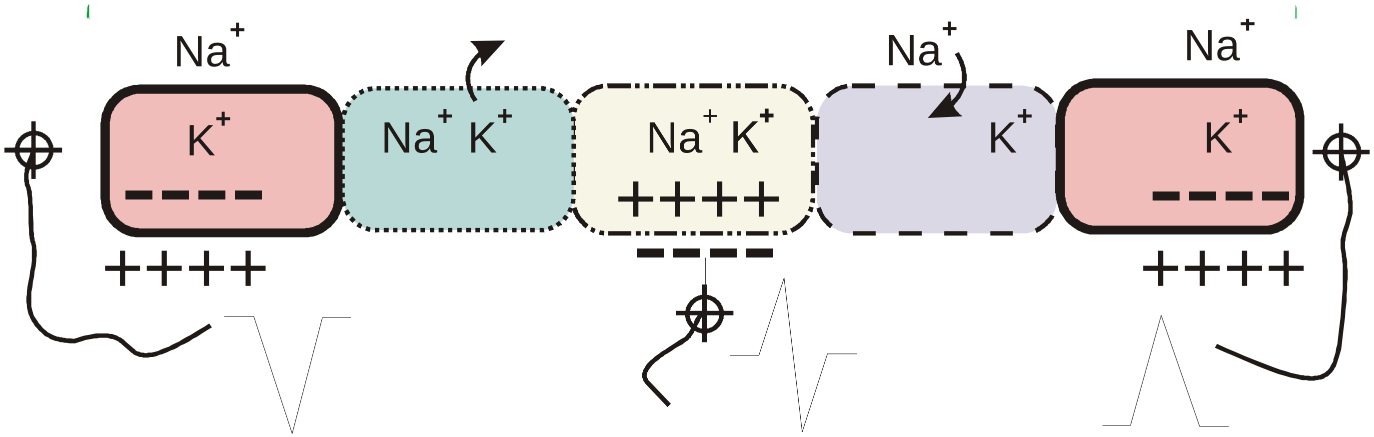
In ECG, Depolarization towards = positive = up

In ECG, Repolarization towards = negative = down

Depolarization Wave of a Strip of Nerve Cells
(or Myocardial Muscle Cells minus the depiction of Ca^{++} influx)

“Wave of Depolarization“ or ”Propigation of Action Potential” moving from left to right

Concept 2

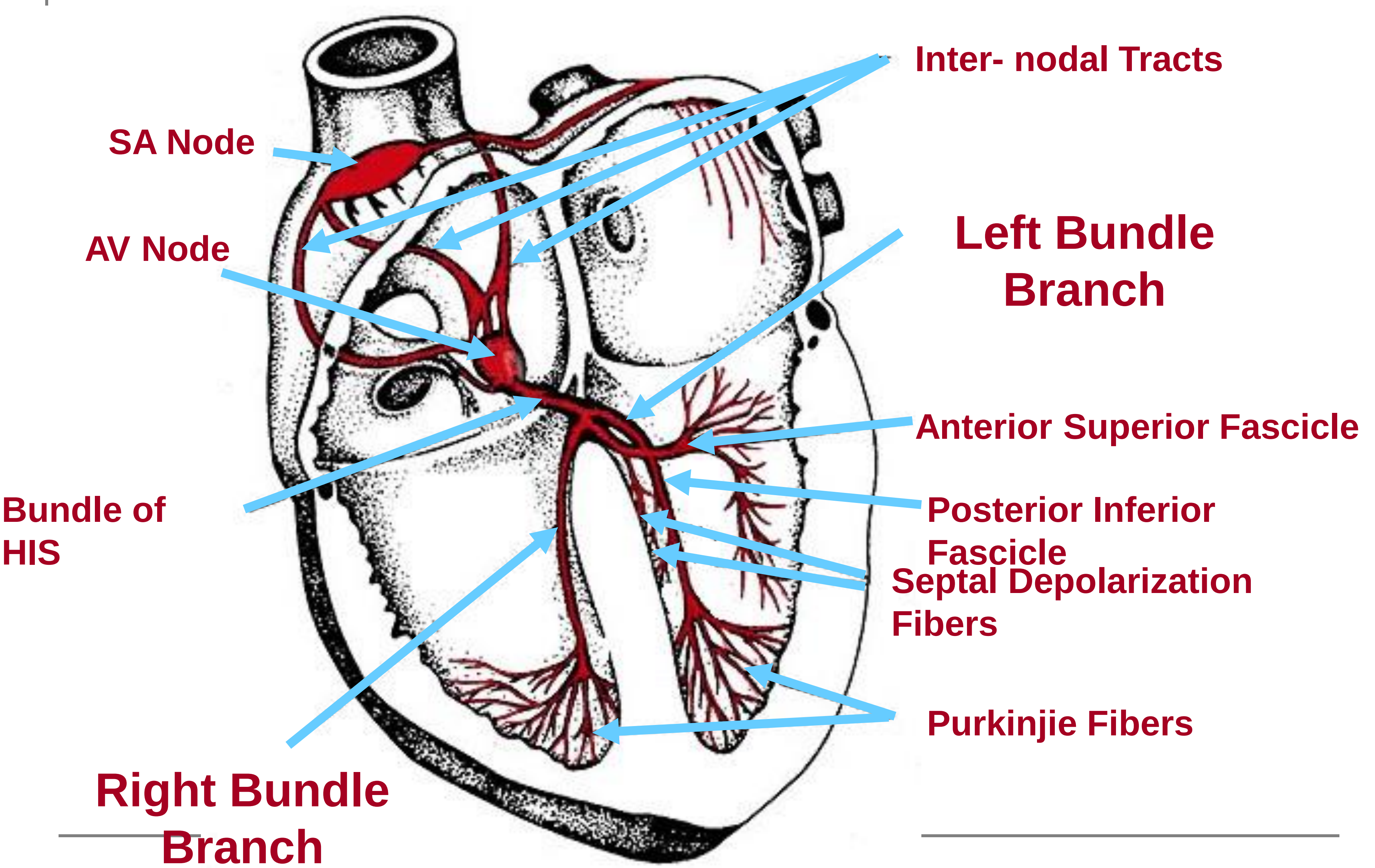


The needle of this recording electrode inscribes a totally <u>negative</u> complex because the wave of depolariztion is moving <u>away</u> from it during the entire time the strip is depoarizing

The needle of this recording electrode is <u>biphasic</u> because half of the time the wave of depolarization is moving <u>towards</u> it while the other half of the time it is moving <u>away</u>

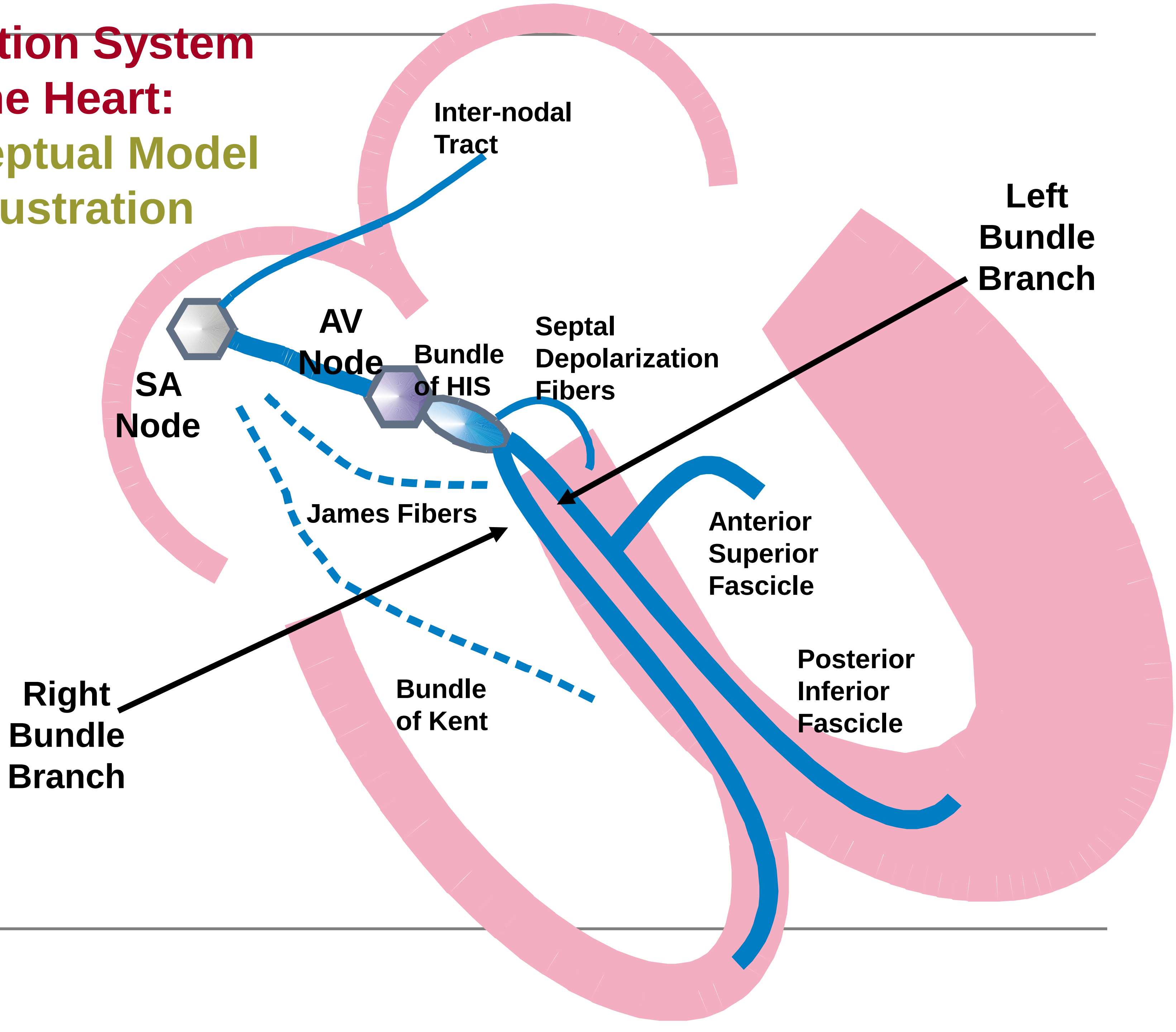
The needle of this recording electrode inscribes a totally <u>positive</u> complex because the wave of depolariztion is moving <u>towards</u> it during the entire time the strip is depolarizing

The Electrical System of the Heart



Conduction System of the Heart:

A Conceptual Model for Illustration

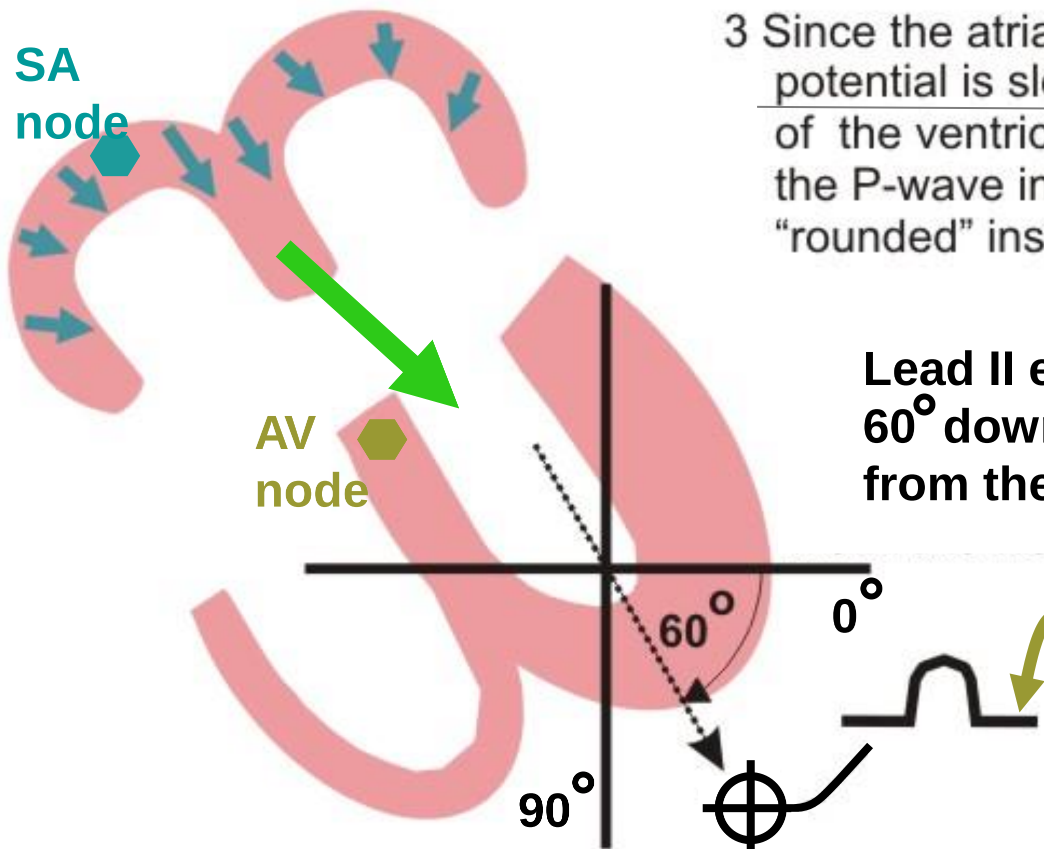


Atrial Depolarization and the Inscription of the P-wave

1. Atrial depolarization proceeds from the top of the atria downward in all directions.

2. Summing these vectors of depolarization results in the main atrial depolarization vector oriented as shown (large green arrow). It is moving towards the positive electrode of the lead, resulting in an upward deflection of the ECG stylus.

3 Since the atrial muscle action potential is slow, relative to that of the ventricular mycoardium the P-wave inscription is “rounded” instead of a spike.



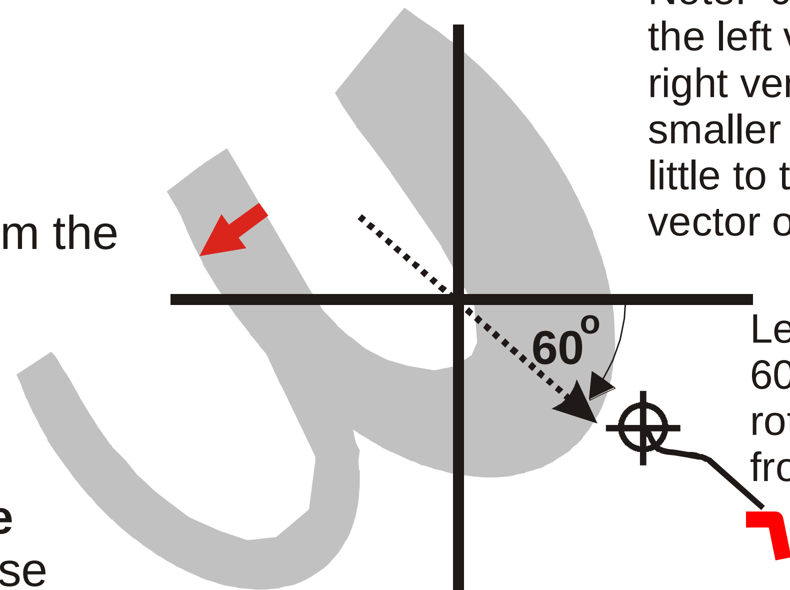
Lead II electrode:
60° downward rotation from the horizontal 0°

Delay (no electrical activity) before the beginning of ventricular depolarization due to AV node function

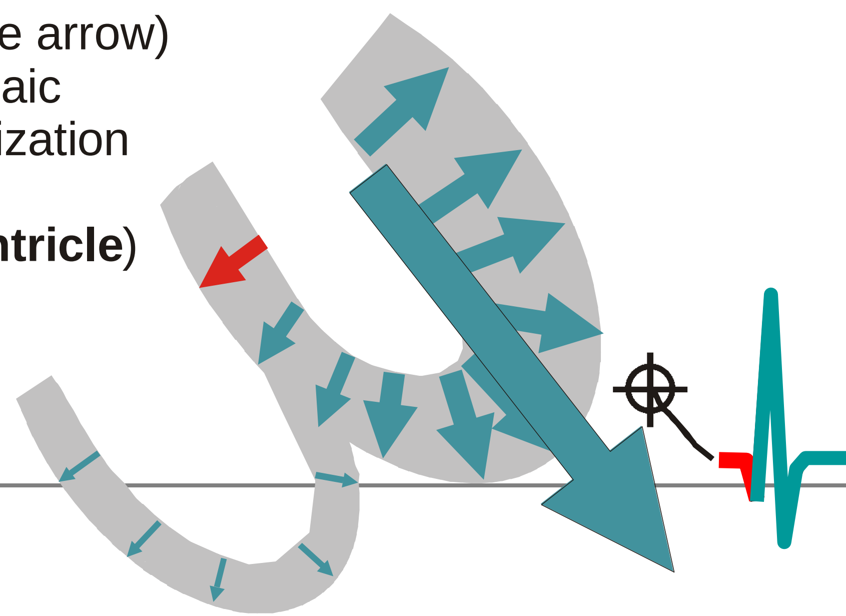
Ventricular Depolarization and the Inscription of the QRS complex

1. The **septum** depolarizes from the inside out and the resulting depolarization wave moves away from the electrode recording Lead II
2. The rest of the **left ventricle** depolarizes counter-clockwise from the inside out and creates the **main cardiac vector** (large arrow) which is essentially, the algebraic sum of all of the small depolarization vectors (including the small contribution from the **right ventricle**) In a normal heart, this vector is always moving directly toward Lead II, generating a mostly positive QRS complex

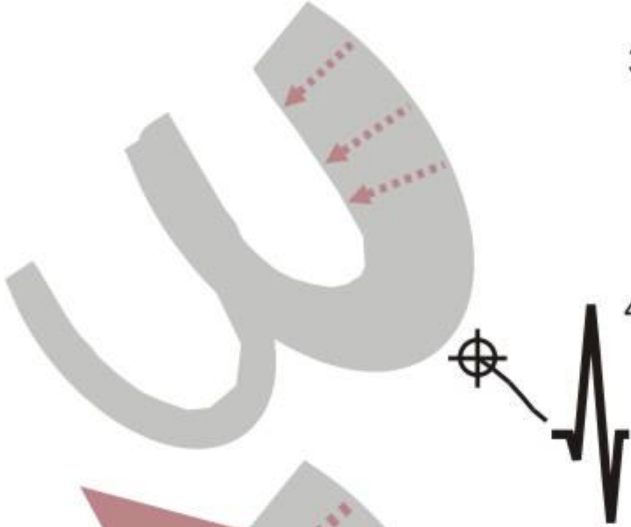
Note: compared to the left ventricle, the right ventricle is much smaller and contributes little to the overall main vector of depolarization



Lead II electrode 60° downward rotation angle from the horizontal 0°

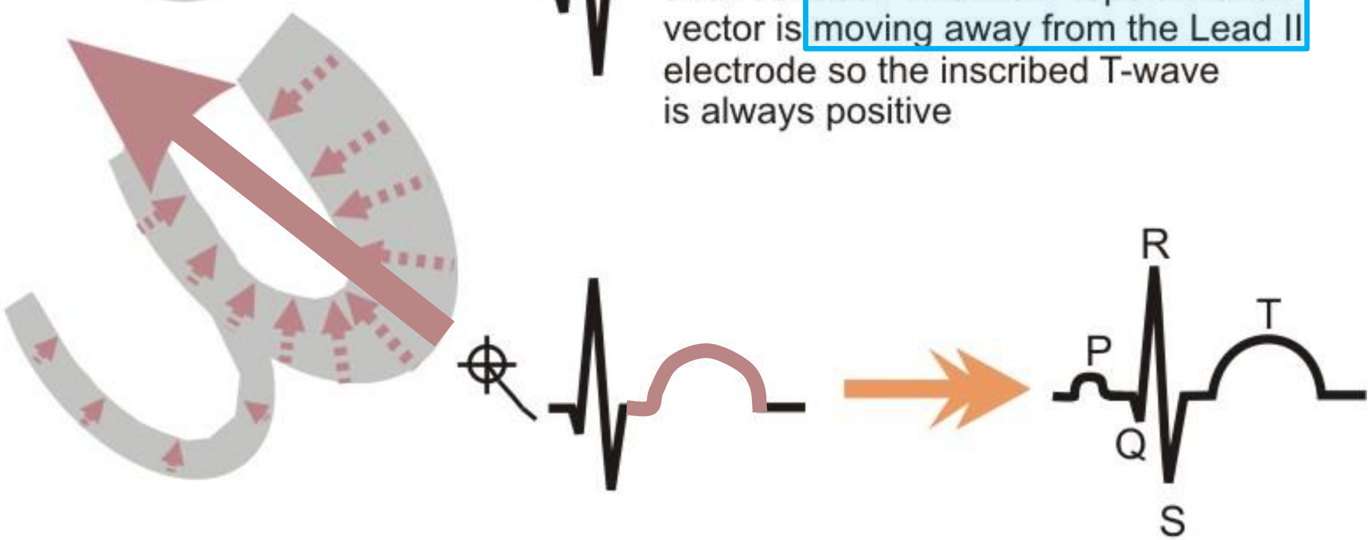


Ventricular Repolarization and the Inscription of the T-wave



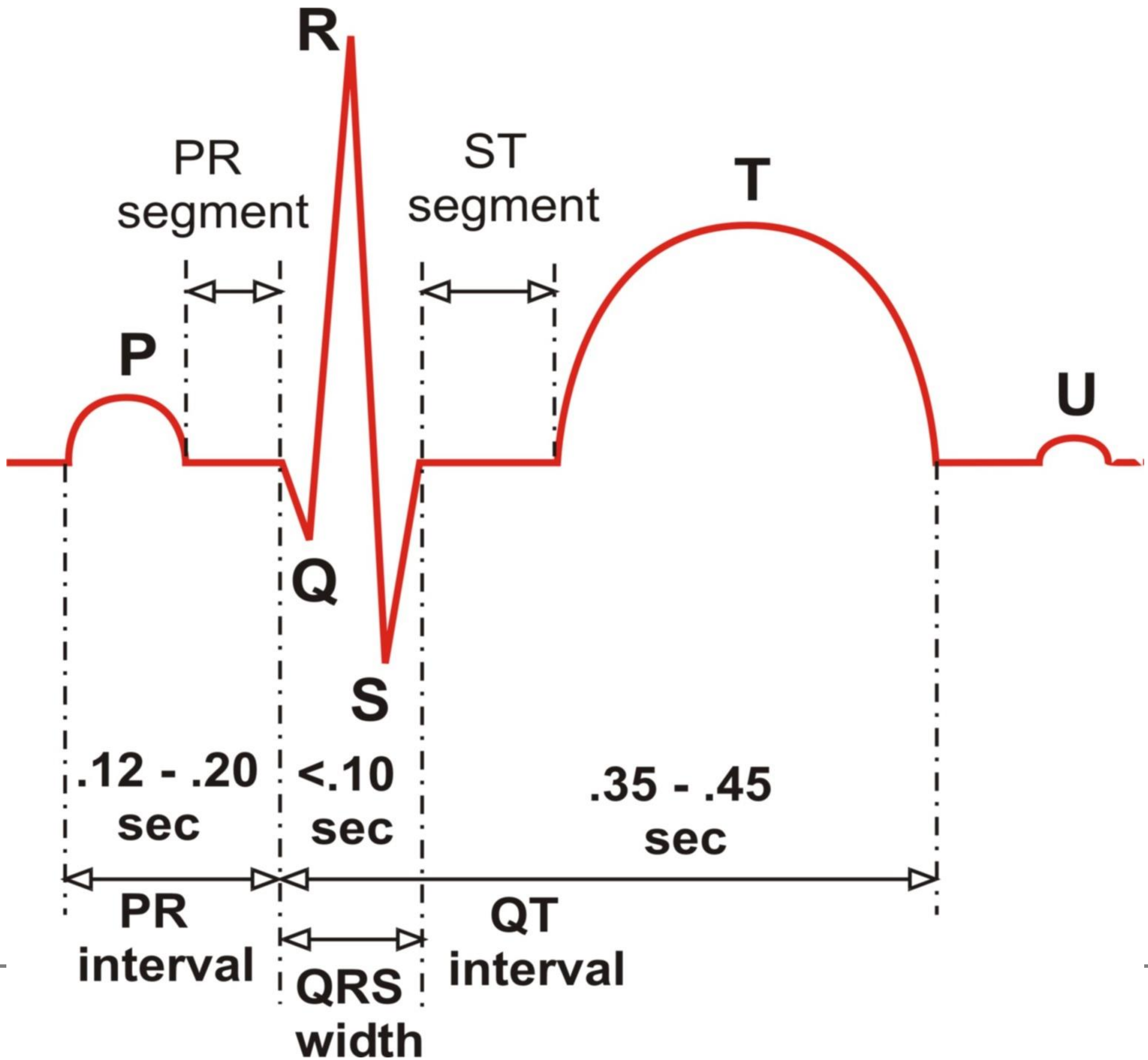
3. Repolarization can be thought of as beginning where depolarization left off and proceeding clockwise from the lateral wall back to the septum..

4. The repolarization process proceeds at a much slower rate than depolarization so the wave inscribed (T-wave) is wide and rounded. The main repolarization vector is moving away from the Lead II electrode so the inscribed T-wave is always positive

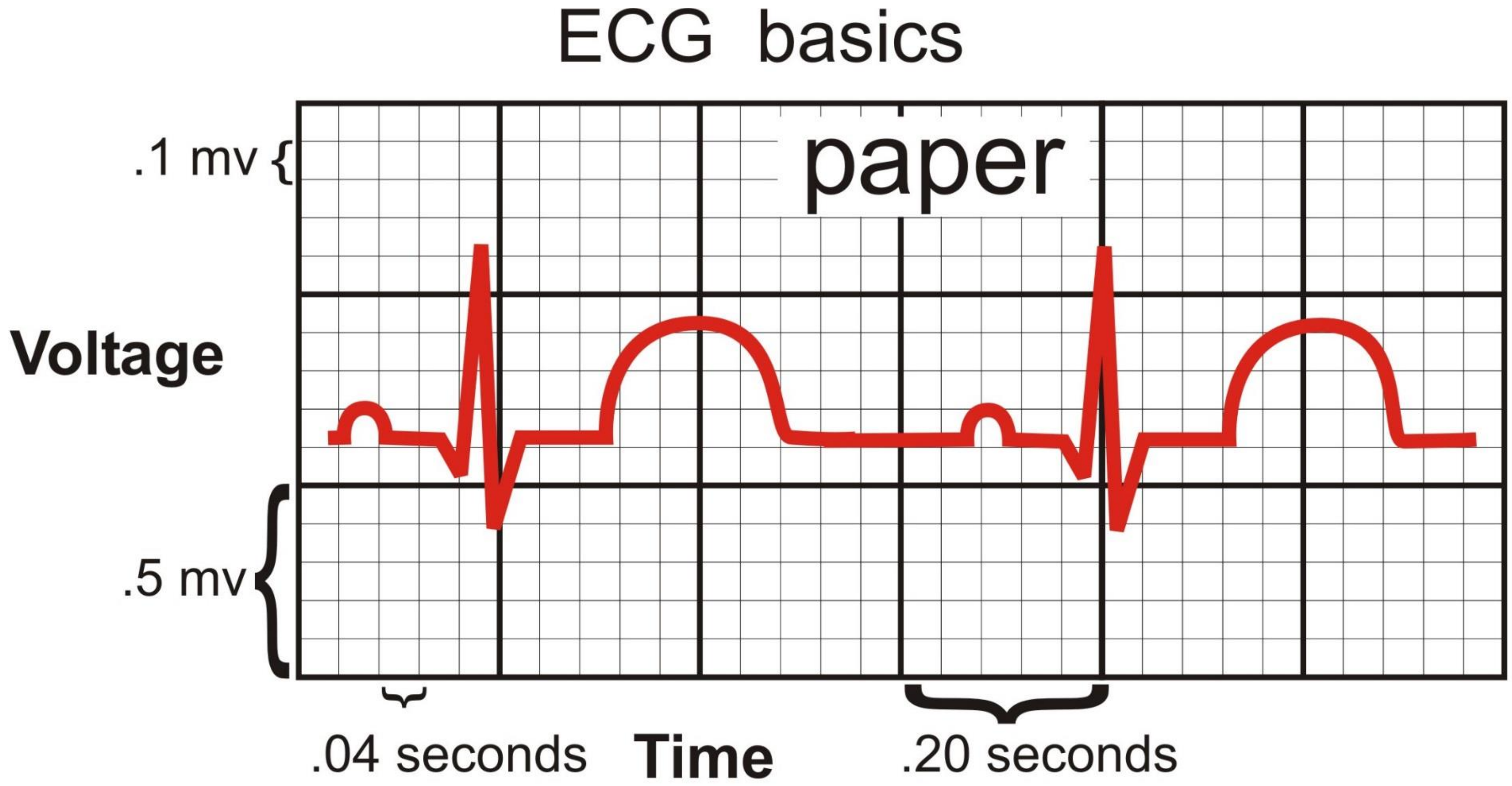


5. Putting the P-wave with the ventricular generated complex yields the entire ECG complex, representing atrial depolarization, atrial repolarization (hidden in ventricular depolarization), ventricular depolarization, and ventricular repolarization

The ECG Complex with Interval and Segment Measurements



ECG Paper and related Heart Rate & Voltage Computations



Paper speed = 25mm / second

Heart Rate = number of R-waves in a 6 second strip divided by 10

Memorize These 2

= 1500 divided by the number of small boxes between consecutive R-waves

= large square estimation counts (300 - 150 - 100 - 75 - 60 - 50 - 43)

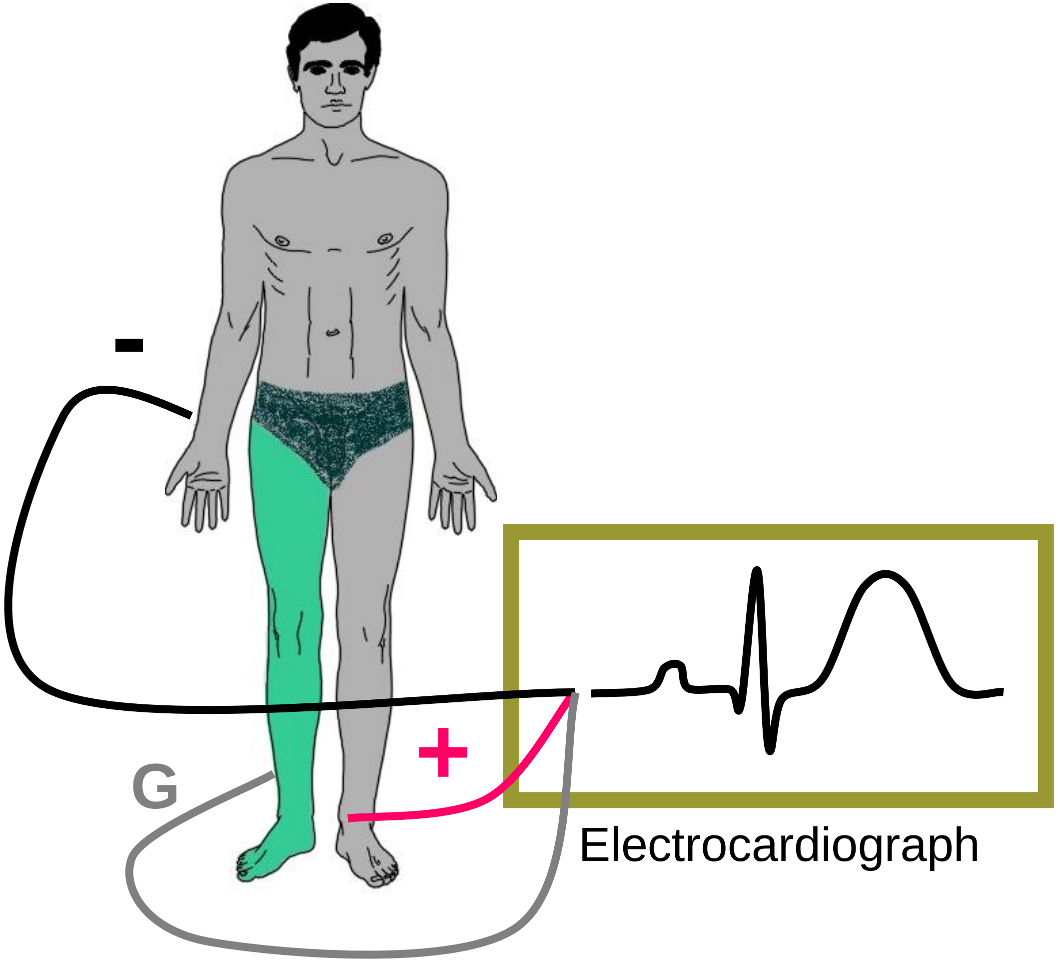
Questions ?

The Concept of a "Lead"

Lead II

• Right arm (RA) negative, left leg (LL) positive, right leg (RL) is always the ground.

•This arrangement of electrodes enables a "directional view" recording of the heart's electrical potentials as they are sequentially activated throughout the entire cardiac cycle

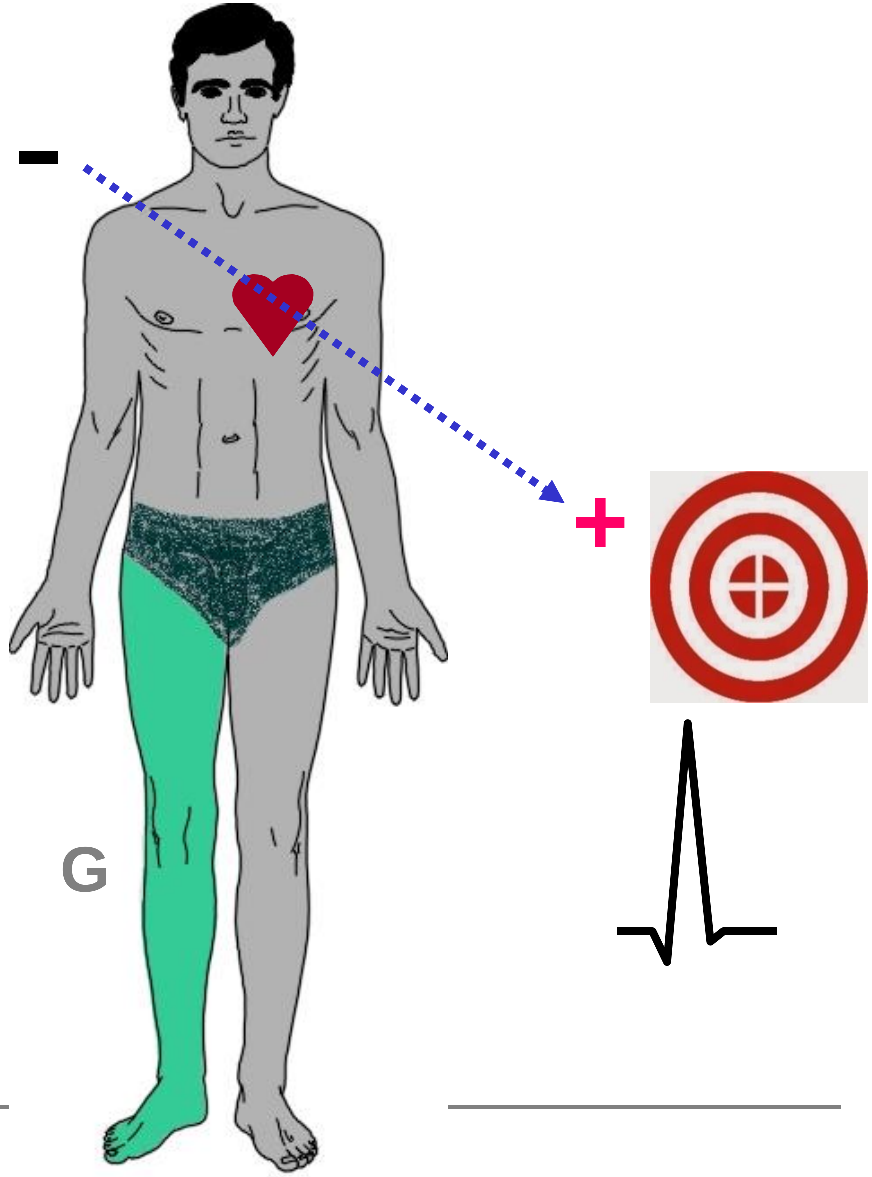


The Concept of a "Lead"

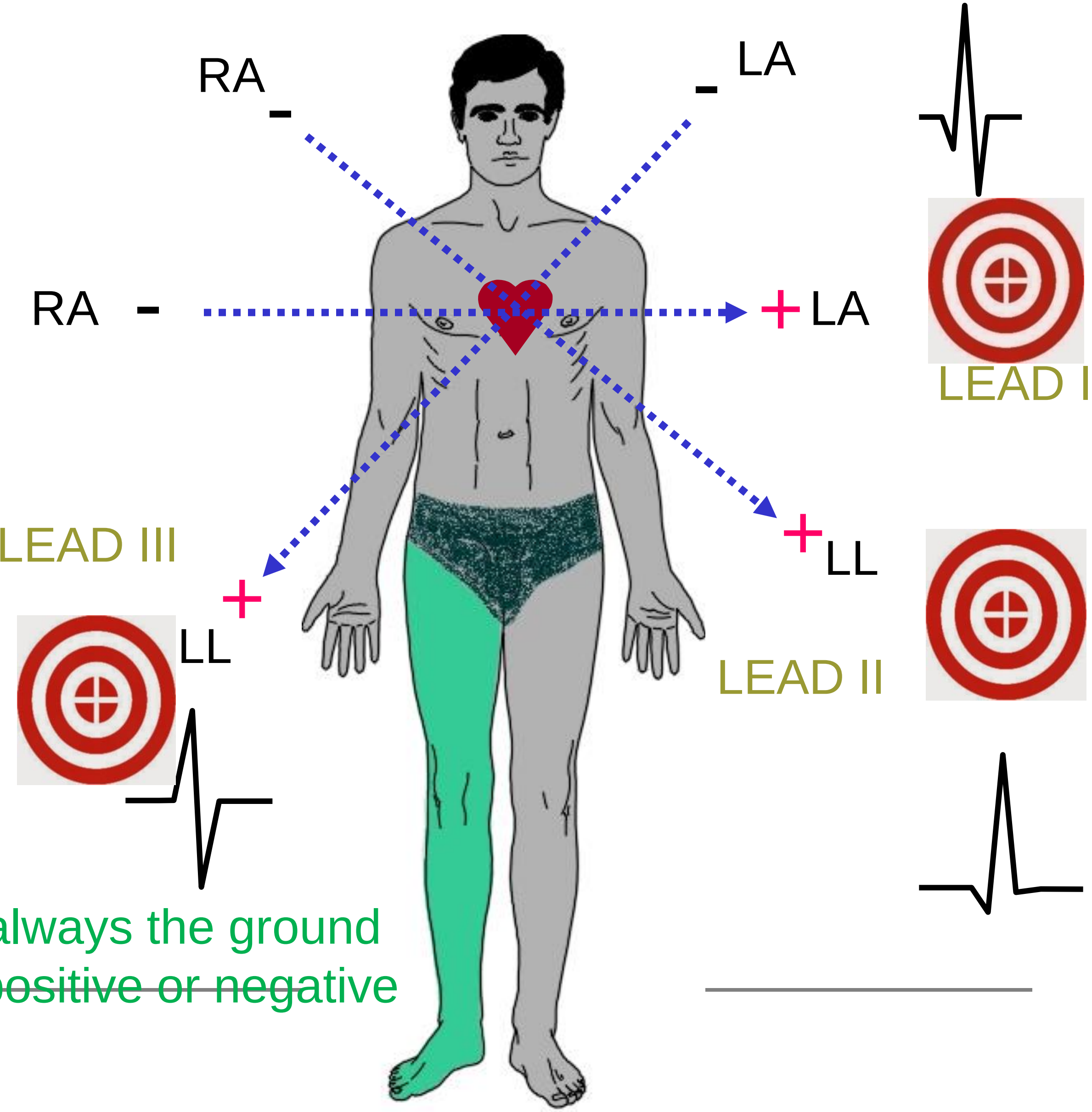
Lead II

- The directional flow of electricity from Lead II can be viewed as flowing <u>from the RA toward the LL</u> and passing through the heart (RA is negative LL is positive.

•Imagine Towards bulls eye



Leads I, II, and III

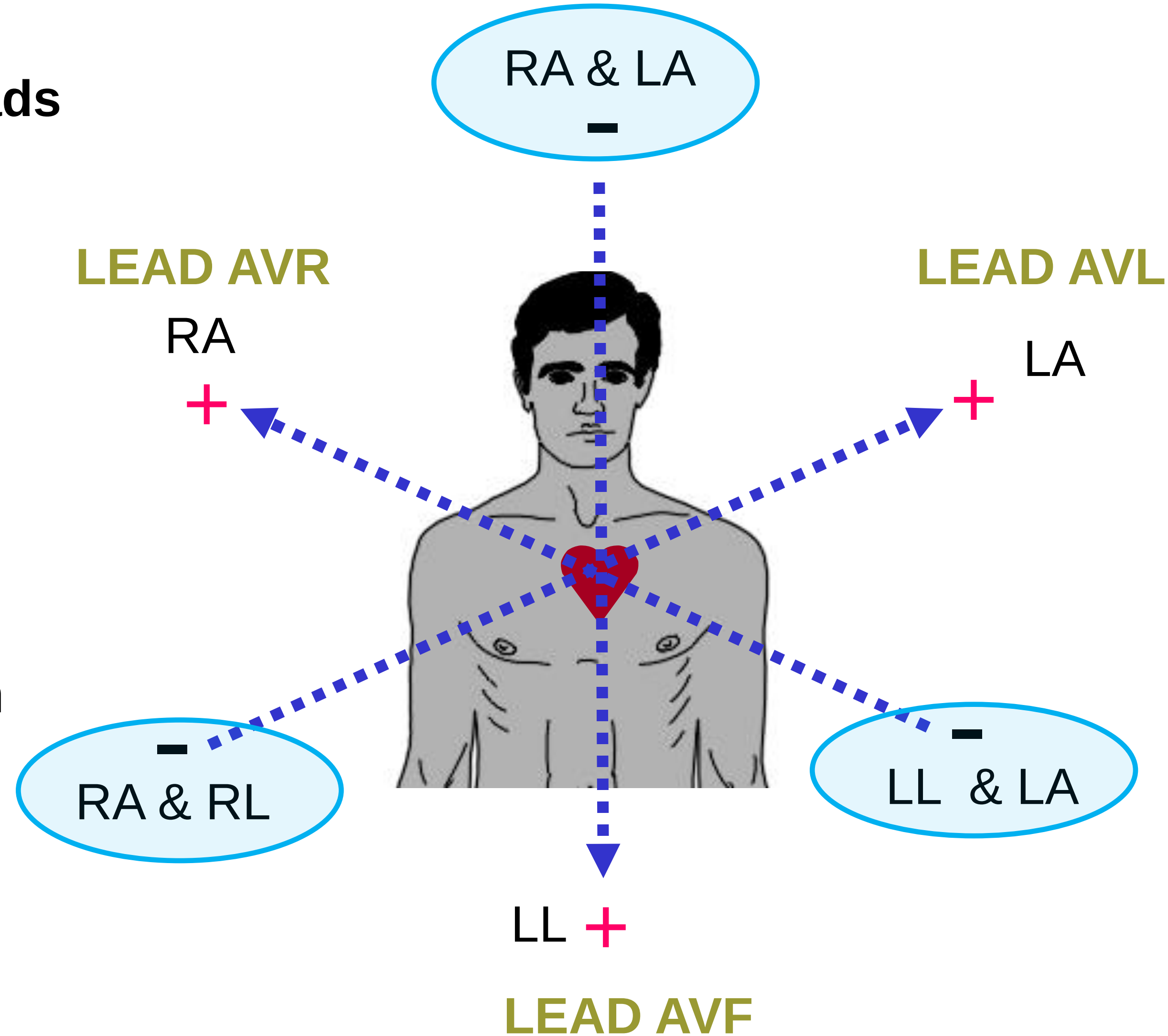


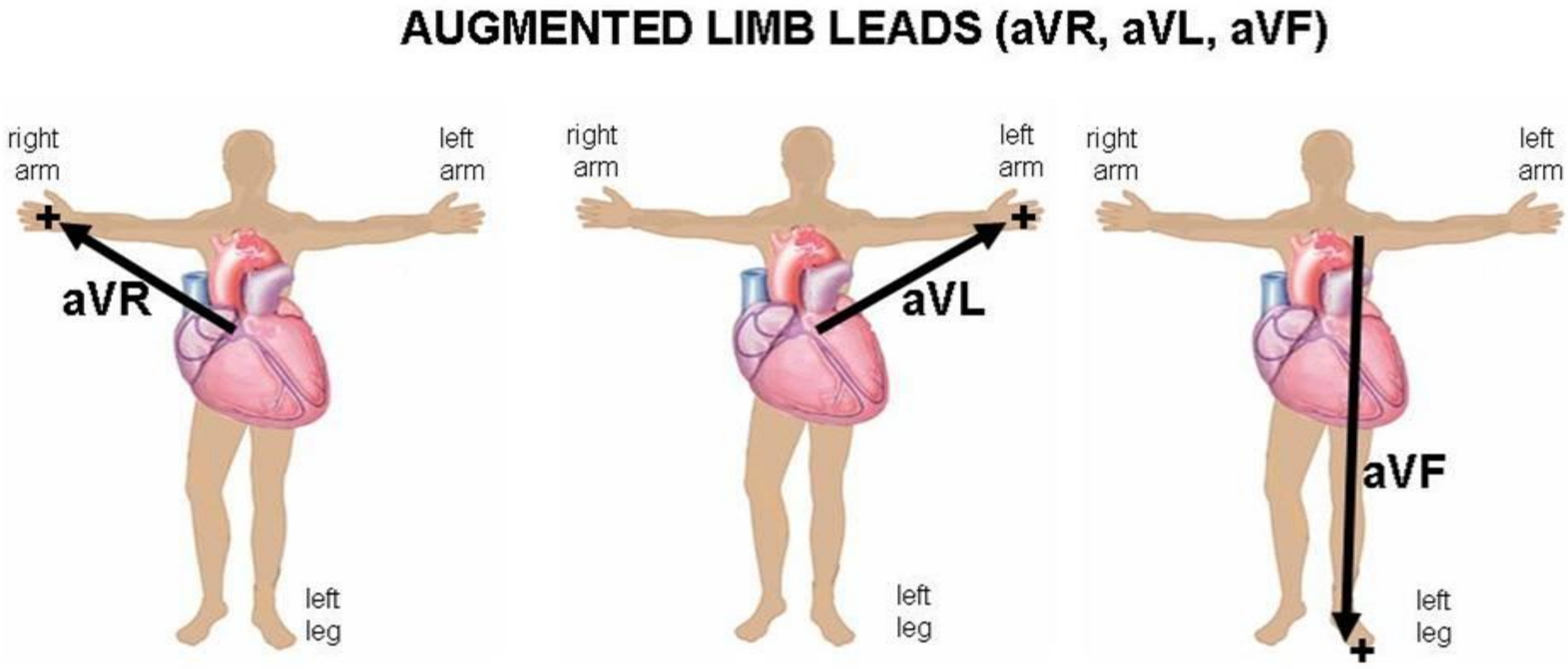
Remember, the **RL** is always the ground and never takes on a positive or negative charge.

The Concept of a "Lead"

Augmented Voltage Leads
AVR, AVL, and AVF

By combining certain limb leads into a **central terminal**, which serves as the negative electrode, other leads could be formed to "fill in the gaps" in terms of the angles of directional recording. These leads required **augmentation of voltage** to be read and are thus labeled.



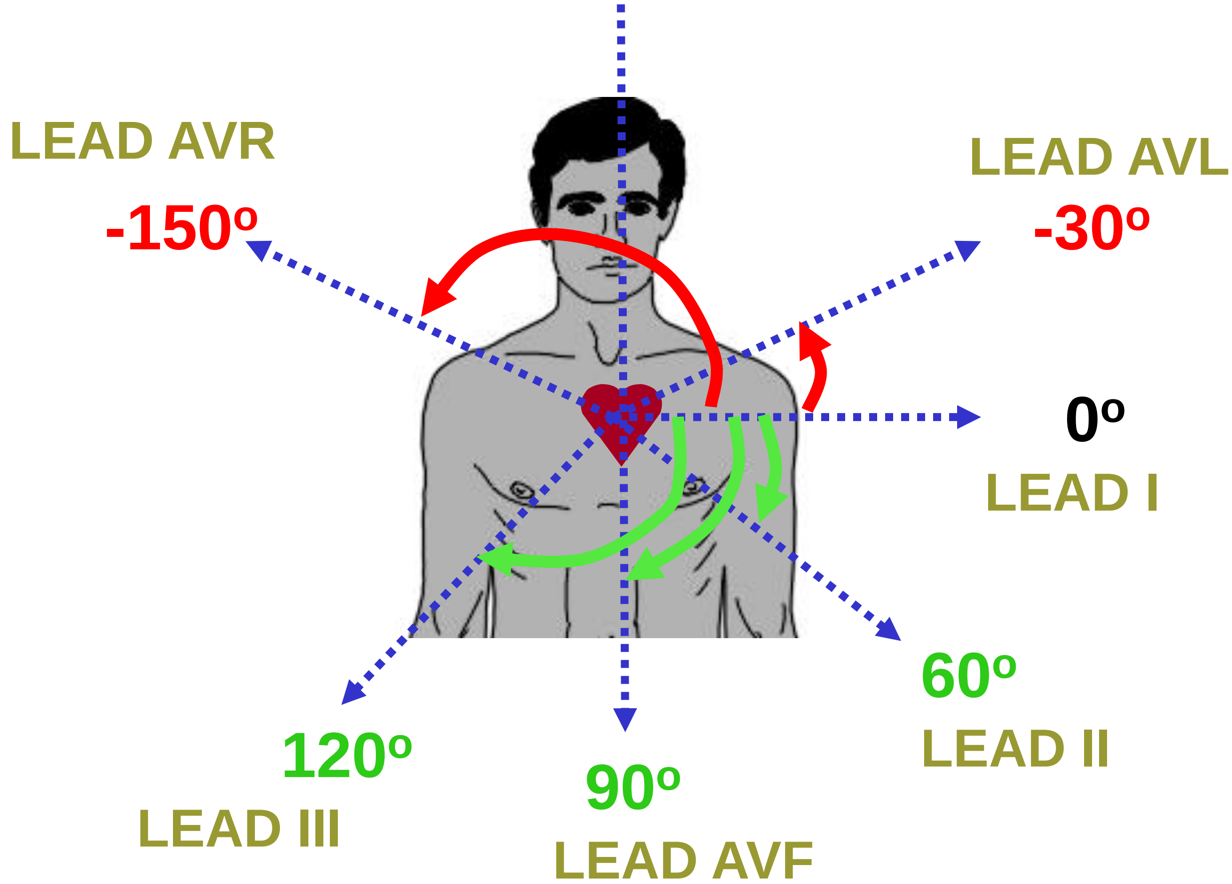


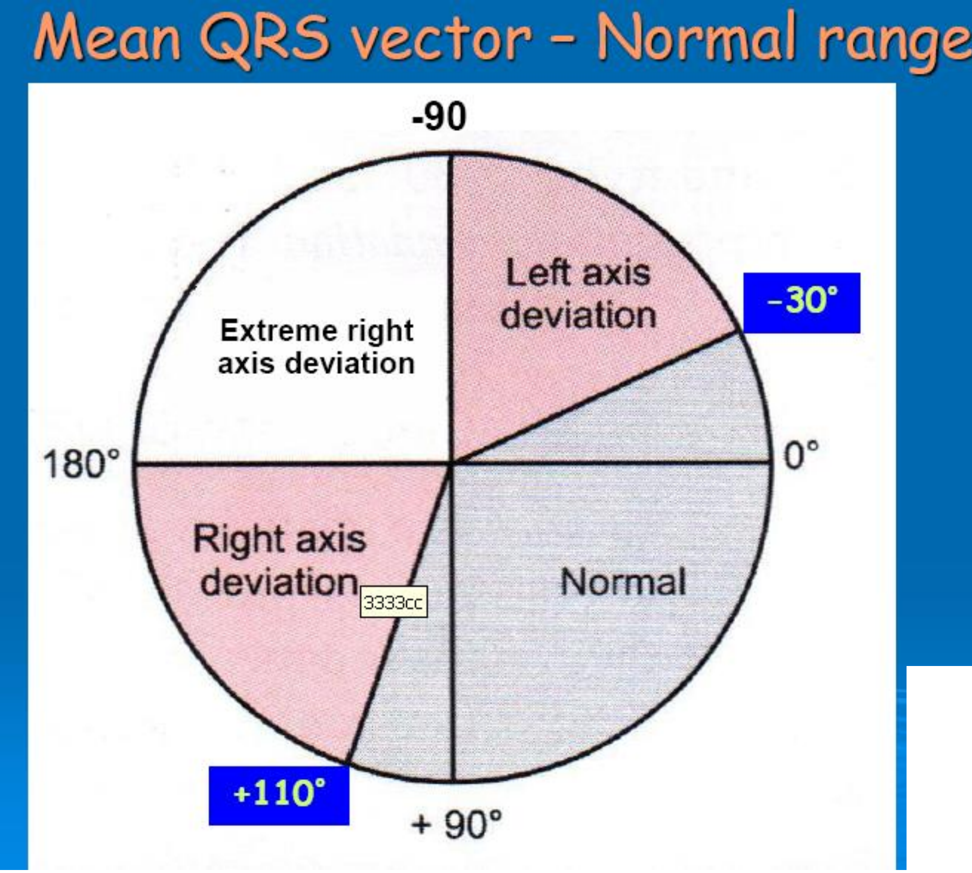
Other two combine to become negative electrode. A single positive electrode that is referenced against a combination of the other **limb** electrodes.

Rotation of a "Lead" - degrees

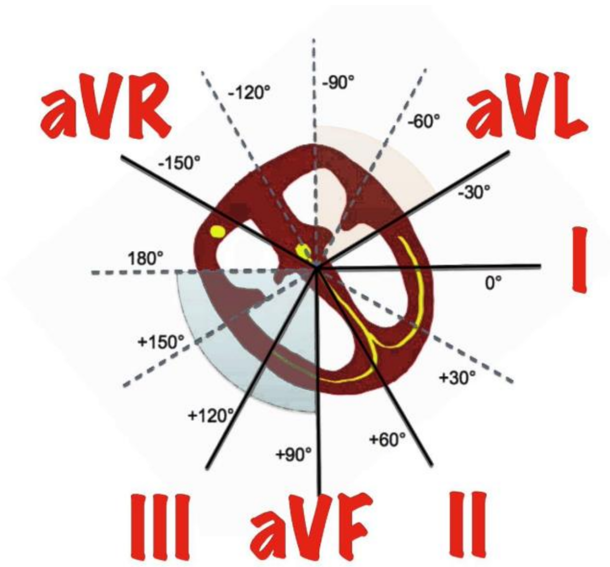
Any "Limb Leads"

Each of the limb leads (I, II, III, AVR, AVL, AVF) can be assigned an angle of **clockwise** or **counterclockwise** rotation to describe its position in the frontal plane. Downward rotation from 0 is positive and upward rotation from 0 is negative.





A Normal Axis lies between **+90 and -30 degrees.**



Cardiac Axis

Left axis deviation:

left ventricular hypertrophy (LVH)
left anterior fascicular block (or hemiblock) and
Inferior myocardial infarction

Right axis deviation:

Right ventricular hypertrophy
Left Posterior Fascicular Block
Lateral Myocardial Infarction

Review Exercise

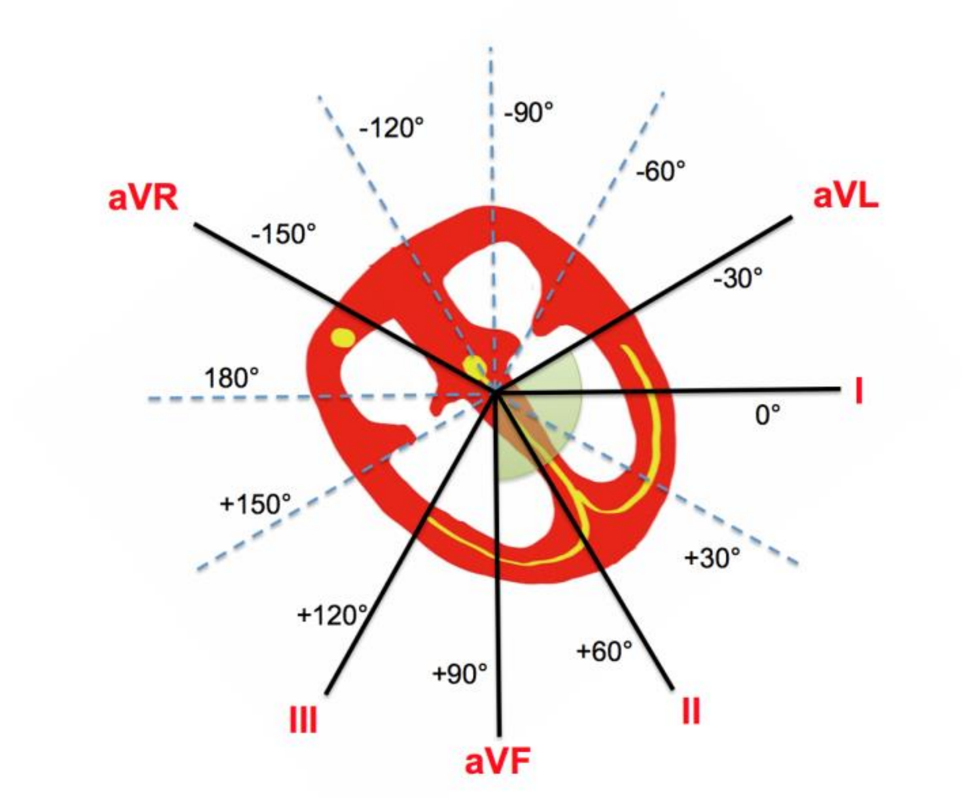
An anaesthetist recently asked
“Please can you explain how to calculate the cardiac Axis........ideally, as if explaining to a student.

Needed Items:

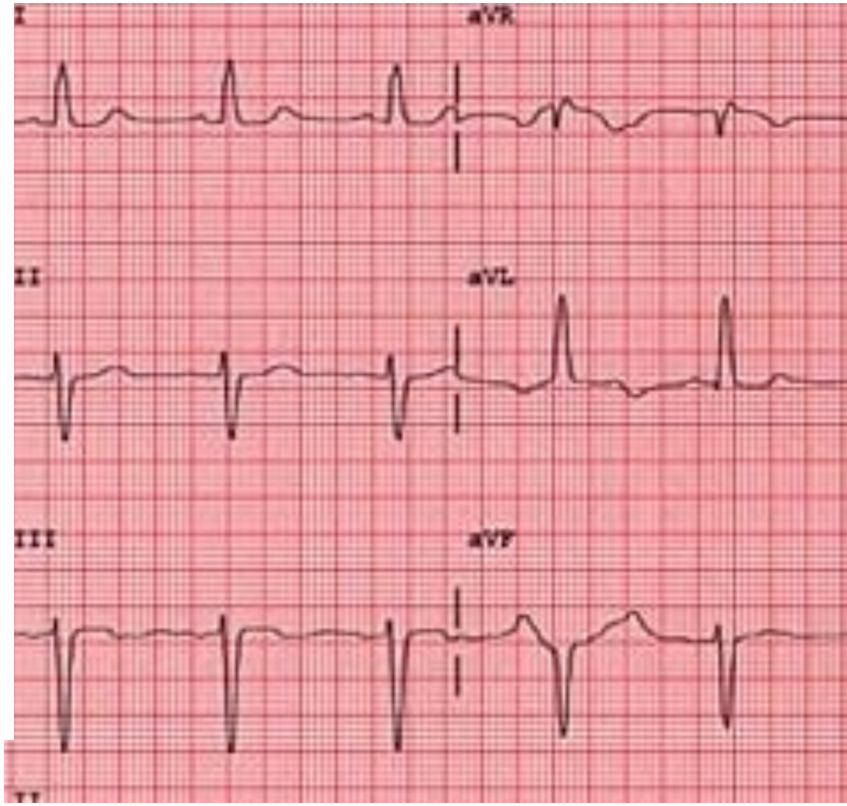
1)aVR, aVL, AvF, I, II and III on axis

2)Any ECG record

Axis paper (in pocket)



Normal ECG



Identify the most isoelectric lead (on ecg) =

Identify the line at right angles to the most isoelectric line (on the axis paper) =

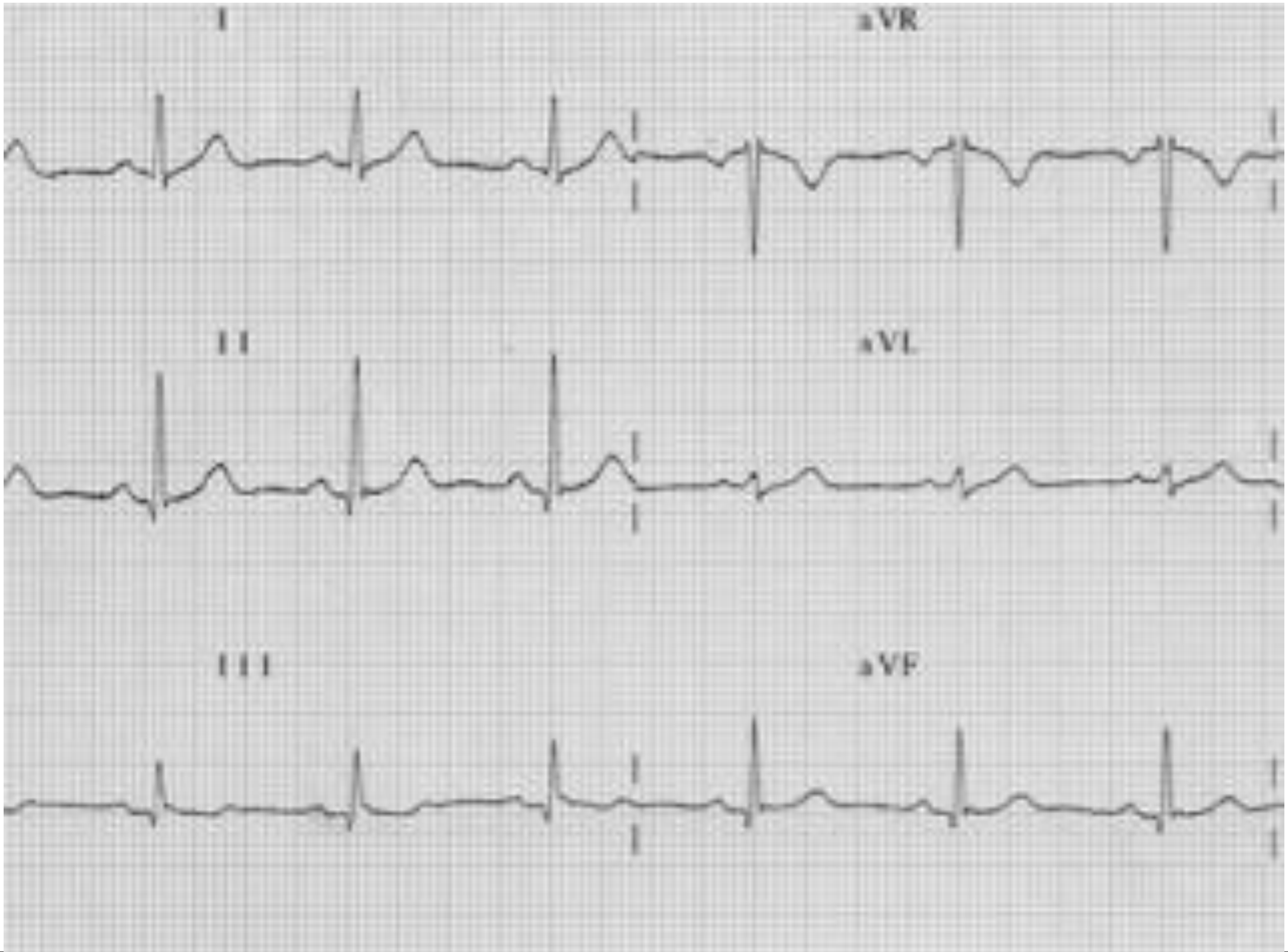
So far so good?

Is the direction positive or negative in that line?
If negative means, travelling away from that lead

Look at the number degree associated with that direction.

Hence THE CARDIAC AXIS ON THIS ECG IS -60 DEGREES

Without axis paper, impossible to find cardiac axis



Example 2

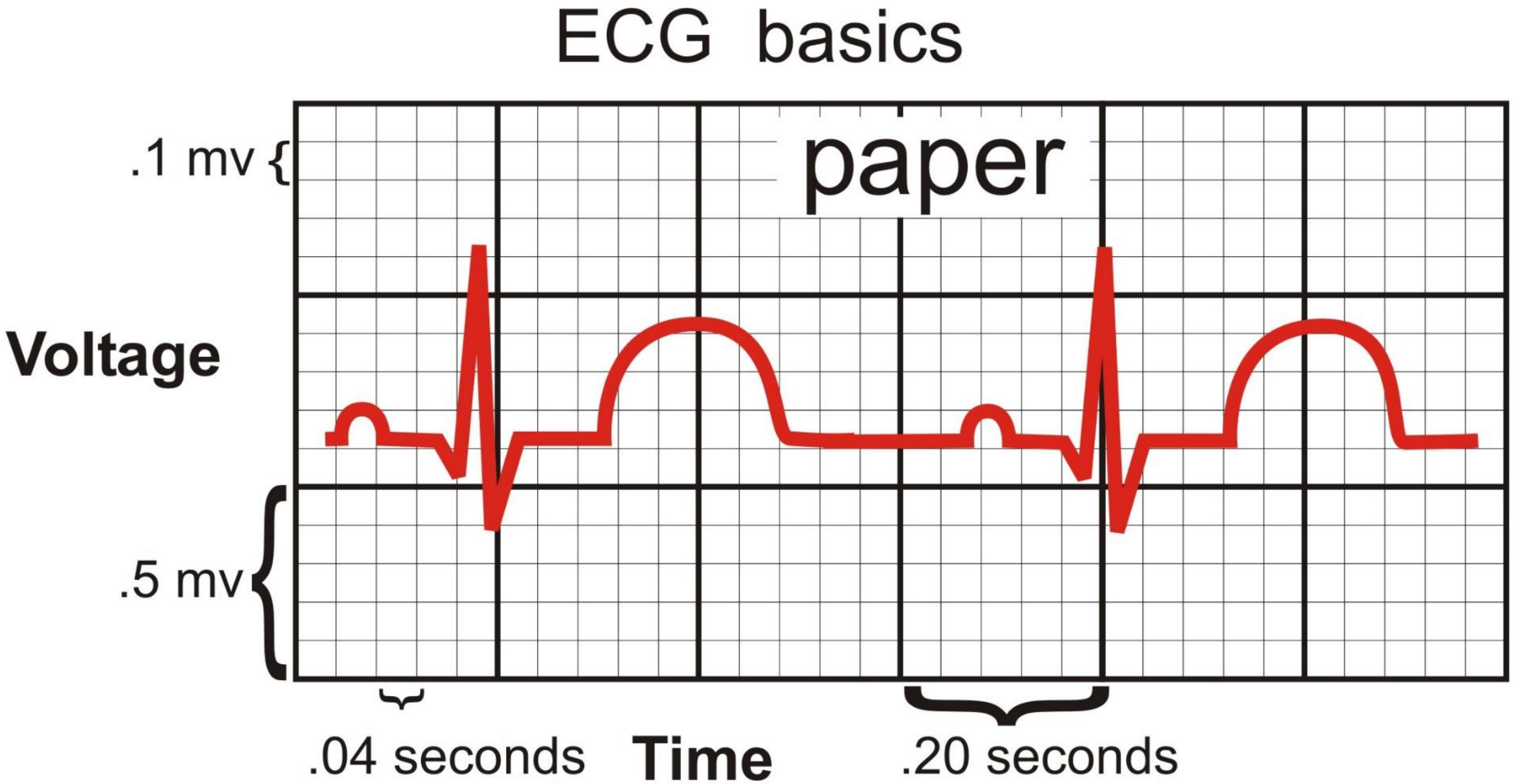
Identify the most isoelectric lead (on ecg) =
Identify the line at right angles to the most isoelectric line (on the axis paper) =

Check wave towards or away

So degrees?

Cardiac axis is + 60

ECG Paper and related Heart Rate & Voltage Computations



Paper speed = 25mm / second

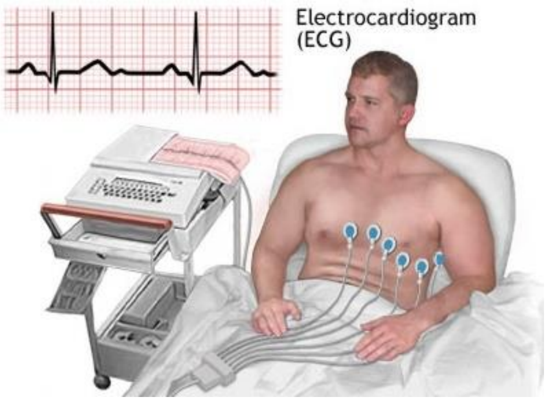
Heart Rate = number of R-waves in a 6 second strip divided by 10

Memorize These 2

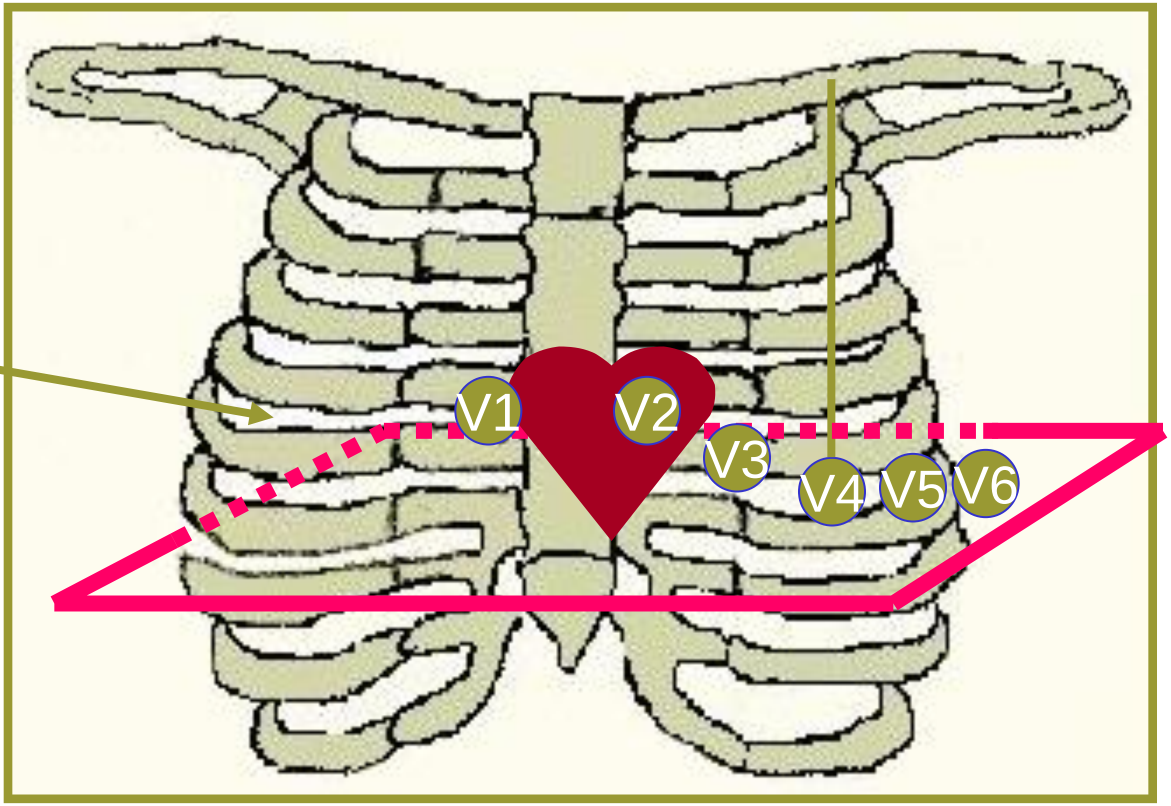
= 1500 divided by the number of small boxes between consecutive R-waves

= large square estimation counts (300 - 150 - 100 - 75 - 60 - 50 - 43)

The Concept of a "Lead"



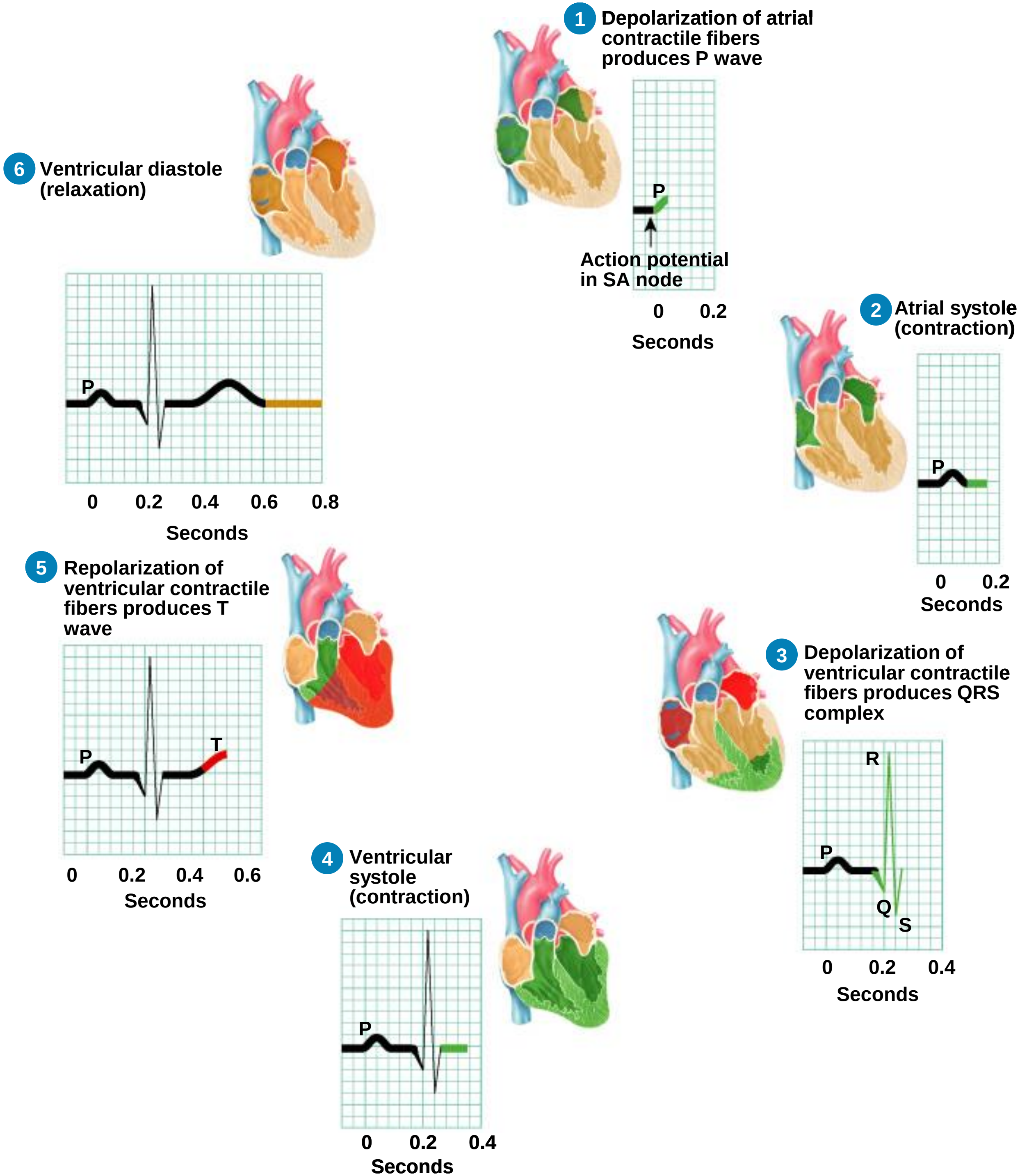
The "Precordial Leads"



Each of the 6 precordial leads is unipolar (1 electrode constitutes a lead) and is designed to view the electrical activity of the heart in the **horizontal** or **transverse plane**

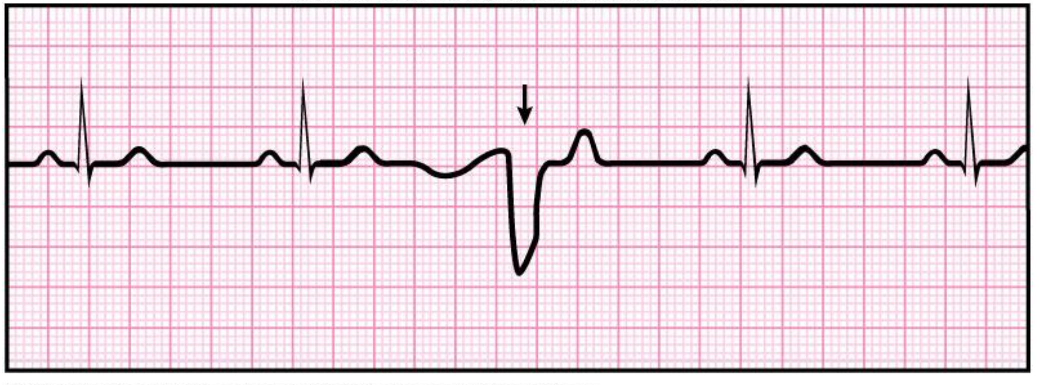
V1 - 4th intercostal space - right margin of sternum
V2 - 4th intercostal space - left margin of sternum
V3 - linear midpoint between V2 and V4
V4 - 5th intercostal space at the mid clavicular line
V5 - horizontally adjacent to V4 at anterior axillary line
V6 - horizontally adjacent to V5 at mid-axillary line

Correlation of ECG Waves and Systole



1 Depolarization of atrial contractile fibers produces P wave
P
Action potential in SA node
0 0.2
Seconds
2 Atrial systole (contraction)
P
0 0.2
Seconds
3 Depolarization of ventricular contractile fibers produces QRS complex
R
P
Q
S
0 0.2 0.4
Seconds
4 Ventricular systole (contraction)
P
0 0.2 0.4
Seconds
5 Repolarization of ventricular contractile fibers produces T wave
P
T
0 0.2 0.4 0.6
Seconds
6 Ventricular diastole (relaxation)
P
0 0.2 0.4 0.6 0.8
Seconds

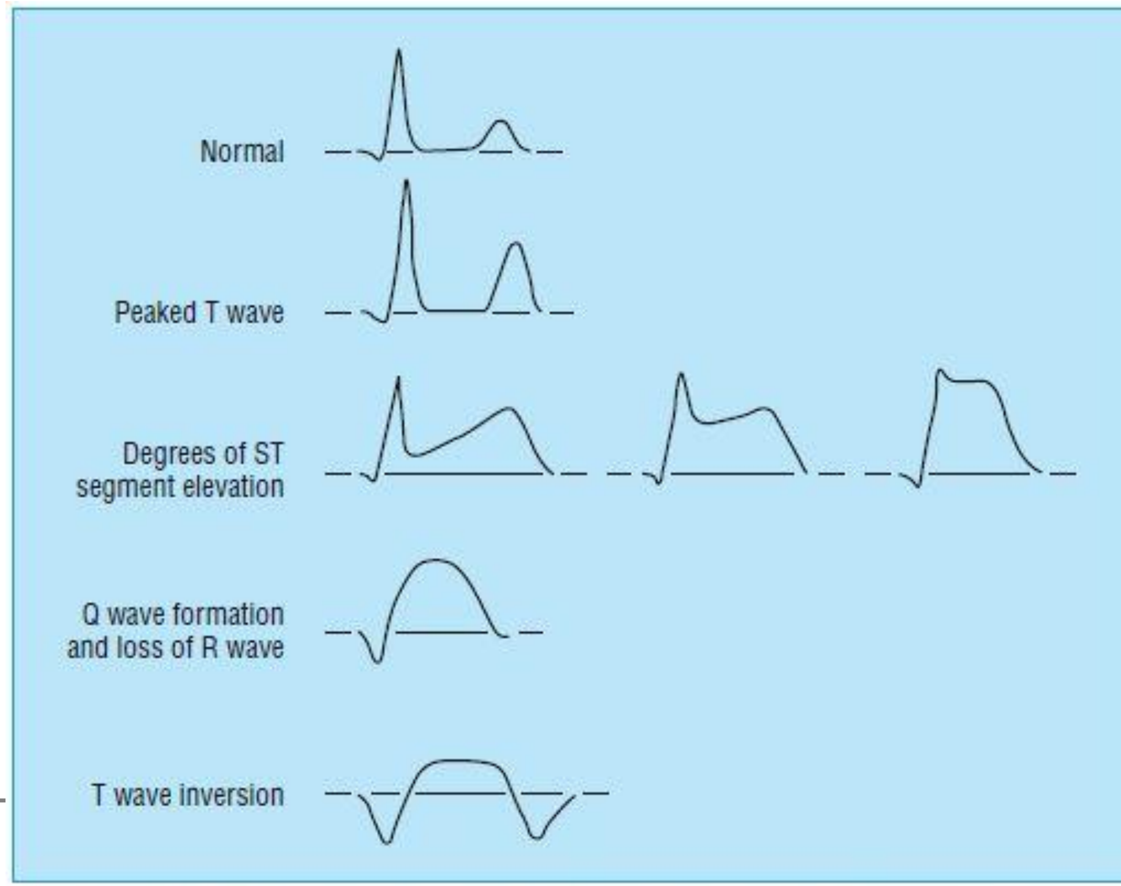
- **PR interval** or **<u>PQ interval</u>**: 0.16 sec
  - Extends from start of atrial depolarization to start of ventricular depolarization (QRS complex) contract and begin to relax
  - Can <u>indicate damage to **conducting pathway** or AV node</u> if greater than 0.20 sec (200 msec)
- **Q-T interval**: time required for **<u>ventricles</u>** to undergo a single cycle of depolarization and repolarization
  - Can be lengthened by **<u>electrolyte disturbances</u>**, conduction problems, coronary <u>ischemia, **myocardial damage**</u>
- -



(d) Premature ventricular contraction

Extrasystole : note <u>inverted QRS complex,</u> misshapen QRS and T and absence of a P wave preceding this contraction.

- Physiological basis of ECG changes in MI
- Rapid repolarization of infracted tissue
- Decreased RMP
- Delayed depolarization



Sequence of changes seen during evolution of myocardial infarction

Questions ?

In class Assignment   0n

Applied vignette

Ms Seeta is a 67 year old retired lecturer who arrives at the Emergency Department complaining of difficulty in breathing and severe fatigue. She recently has been experiencing occasional chest pain and shortness of breath, particularly on exertion while climbing staircase on the way to lecture hall. Her physical signs include tachycardia, engorged neck veins, moderate cyanosis, ankle edema and palpable, and a tender liver. Her blood pressure is 120/85. After admitted to the Sungai long Medical center for further management, her stroke volume was 40 ml/beat and pulse rate was 90/minute.

- What is Ms Seeta's cardiac output?
- What are the factors affecting her stroke volume and cardiac output ?
- What volume of her resting cardiac output is being supplied to the muscles?

Ans: please refer below:

- http://btc.montana.edu/olympics/physiology/pb01.html