Cardiovascular Physiology, Part 2

concepts:

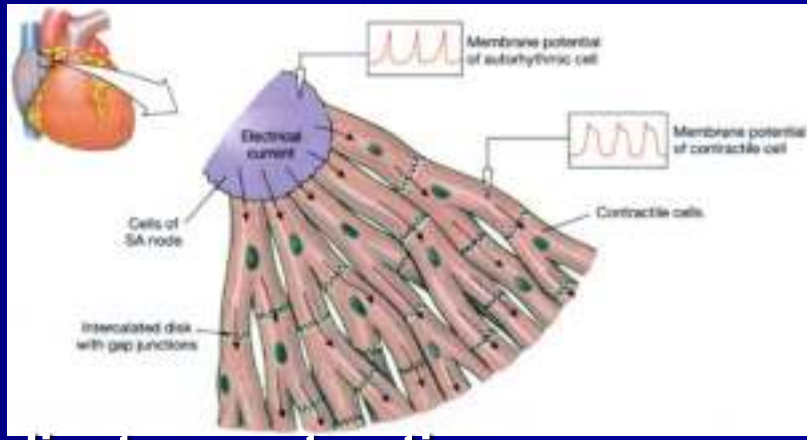
- **Fluid flow**
- **APs in contractile & autorhythmic cells**
- **Cardiac cycle** (elec. & mech. events)
- **HR regulation**
- **Stroke volume & cardiac output**

Modulation of Heart Rate by ANS

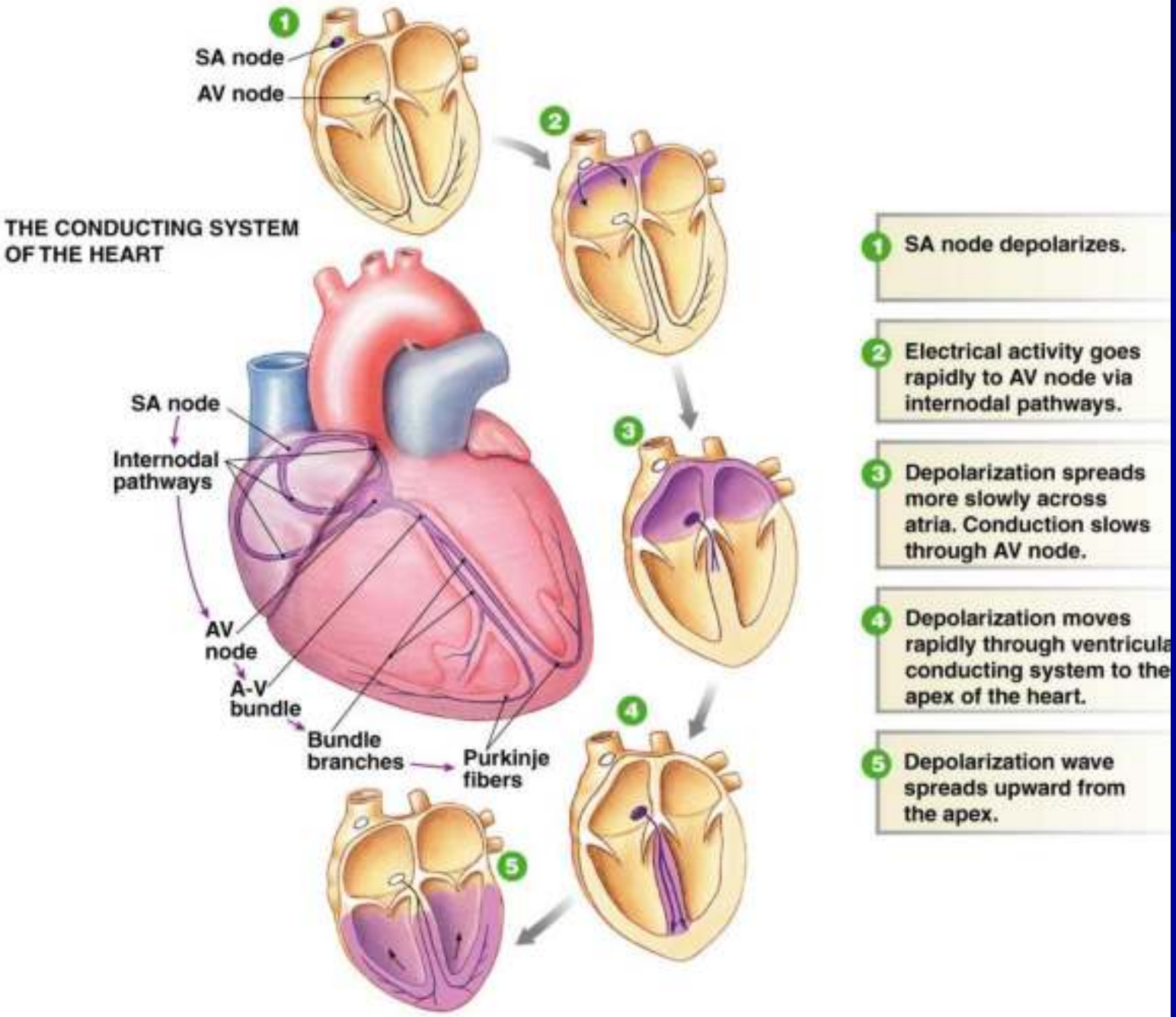
- ANS can alter permeability of autorhythmic cells to different ions
- NE/E (i.e. sympathetic stimulation): ↑ flow through I_f and Ca^{2+} channels
  - Rate AND force of contraction go up
- Ach (parasympathetic): ↑ flow through K^+ channels ↓ flow through Ca^{2+} channels
  - Membranes become hyperpolarized

The Heart as a Pump

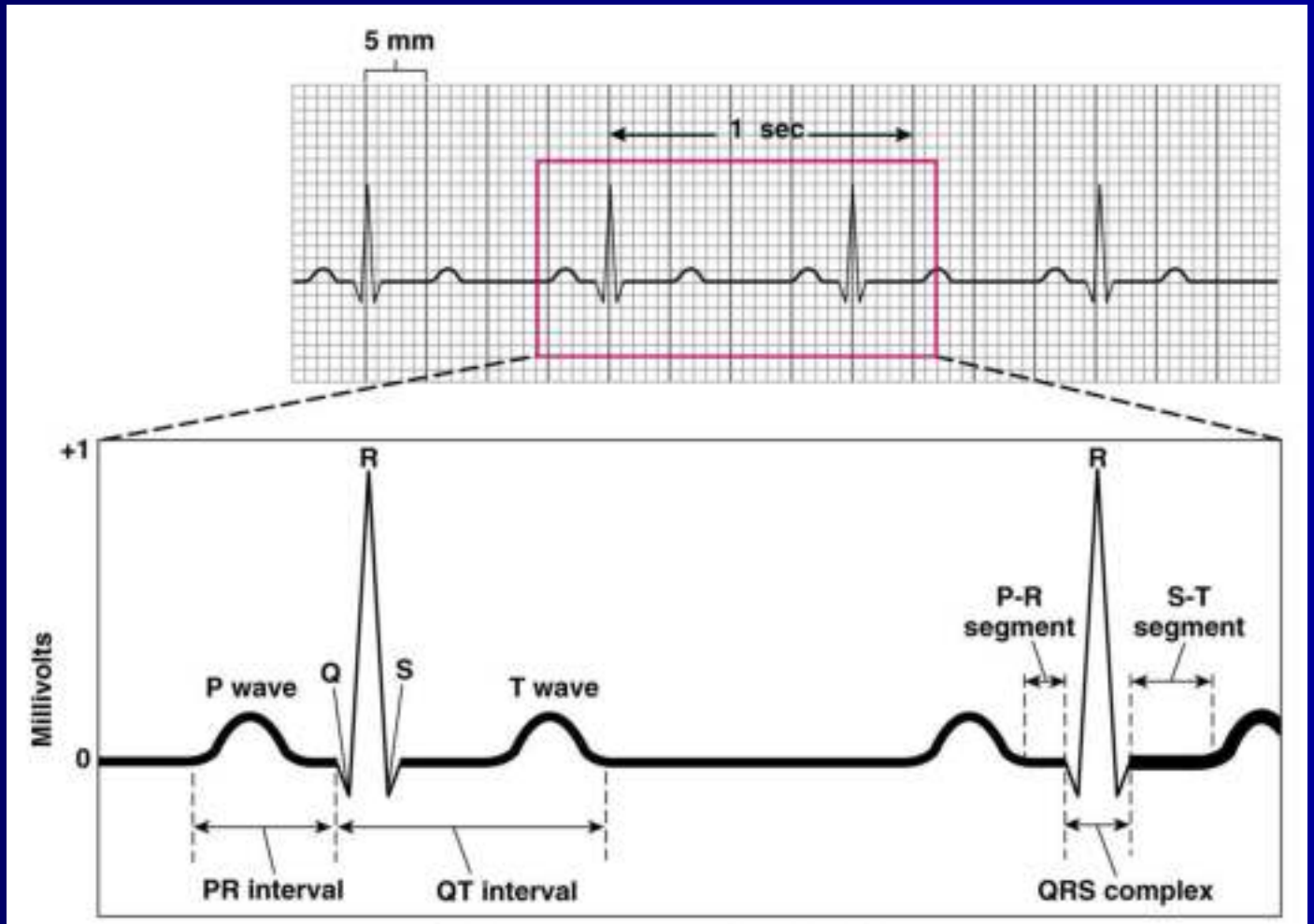
- Communication starts in autorhythmic cells in the SA node (the Pacemaker)
- Move from events in single cell to events in whole heart
- Cardiac cycle
  1. electrical events
  2. mechanical events



- Electrical conduction in heart coordinates contraction


THE CONDUCTING SYSTEM OF THE HEART
1
SA node
AV node
2
SA node
Internodal pathways
AV node
A-V bundle
Bundle branches
Purkinje fibers
3
4
5
1 SA node depolarizes.
2 Electrical activity goes rapidly to AV node via internodal pathways.
3 Depolarization spreads more slowly across atria. Conduction slows through AV node.
4 Depolarization moves rapidly through ventricula conducting system to the apex of the heart.
5 Depolarization wave spreads upward from the apex.

Electrocardiogram ECG (EKG)



Electrocardiogram ECG (EKG)

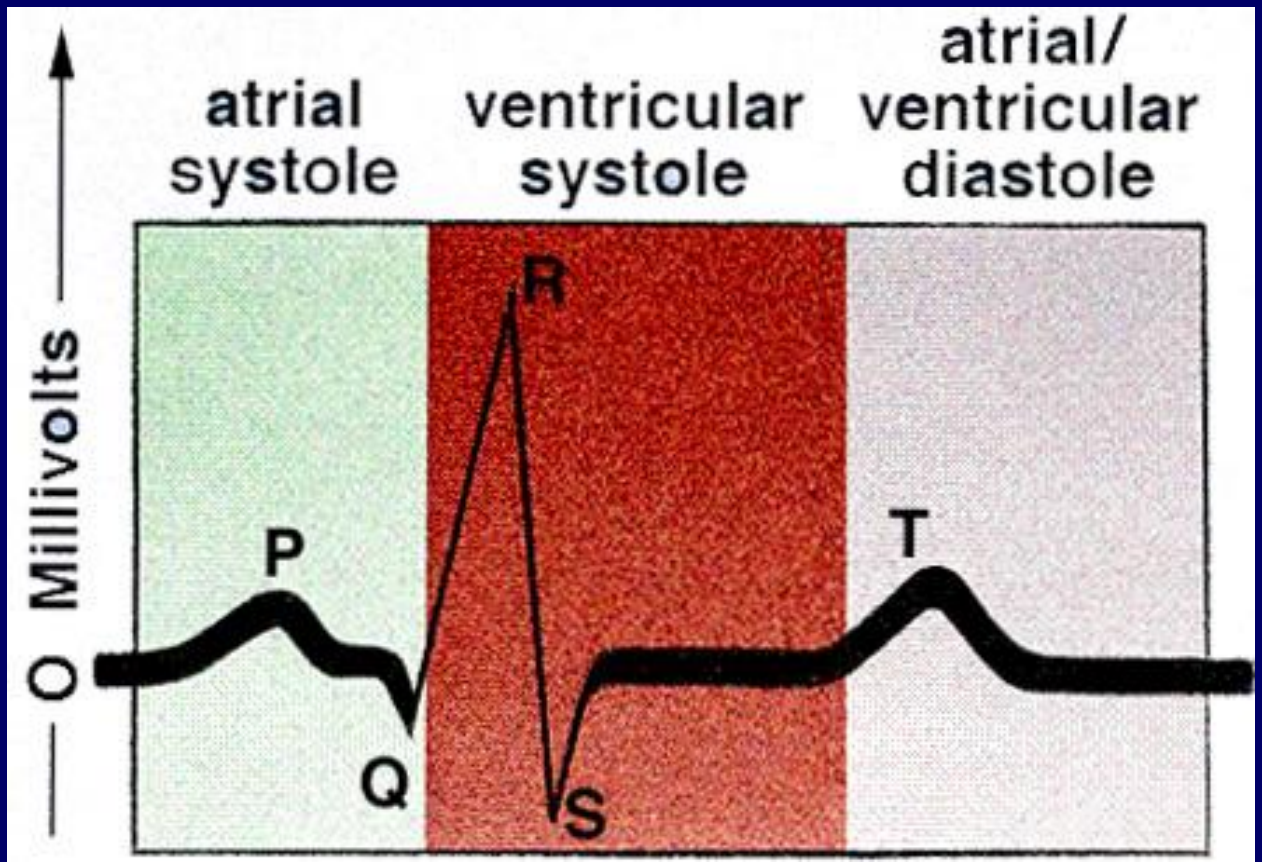
- Surface electrodes record electrical activity deep within body - *How possible?*

- Reflects electrical activity of whole heart not of single cell!
- EC fluid = "salt solution" (NaCl) $\Rightarrow$ good conductor of electricity to skin surface
- Signal very weak by time it gets to skin
  - ventricular AP = ? mV
  - ECG signal amplitude = 1mV
- **EKG tracing** = Σ of all electrical potentials generated by all cells of heart at any given moment

Since:

Depolarization = signal for contraction

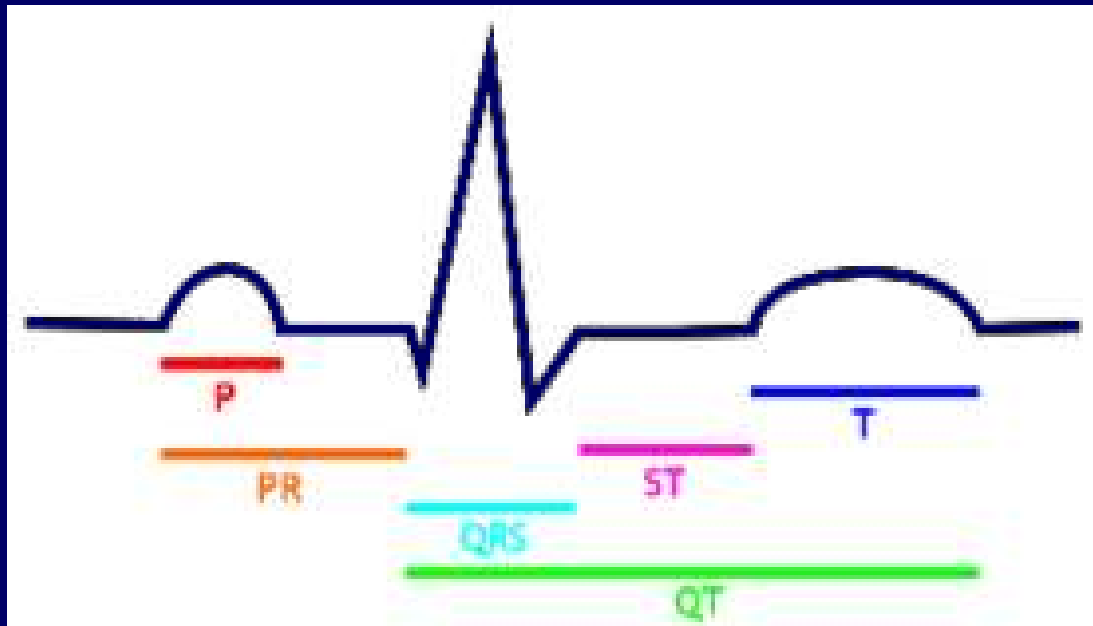
Segments of EKG reflect mechanical heart events

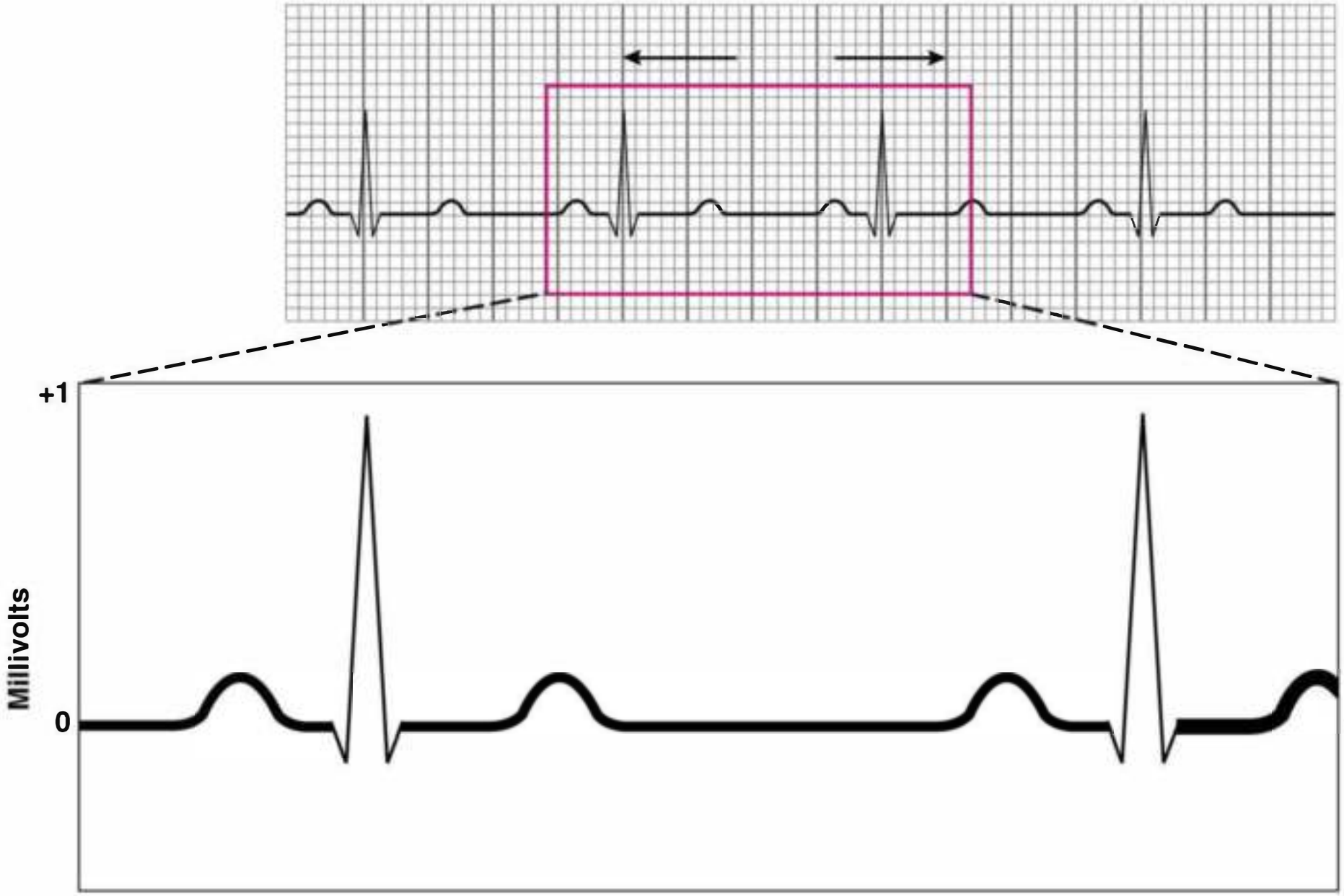


Components of EKG

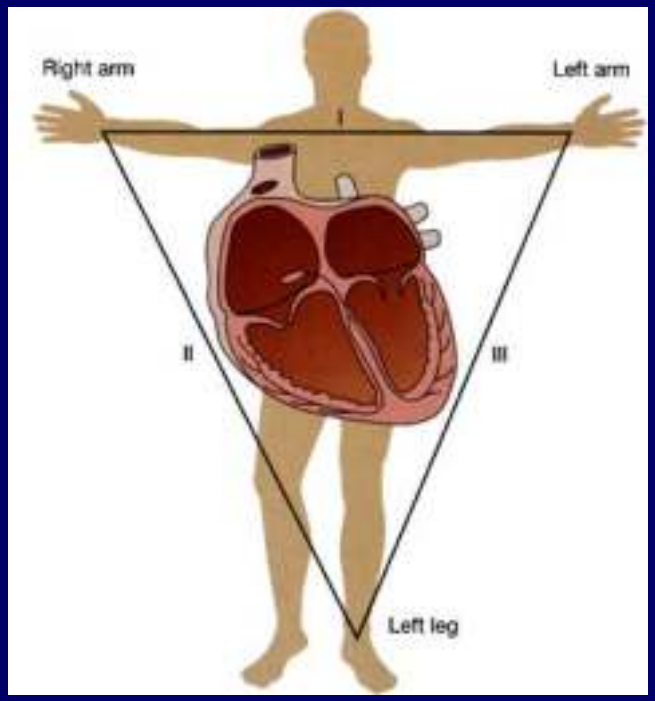
- **Waves** (P, QRS, T)
- **Segments** (PR, ST)
- **Intervals** (wave- segment combos: PR, QT)

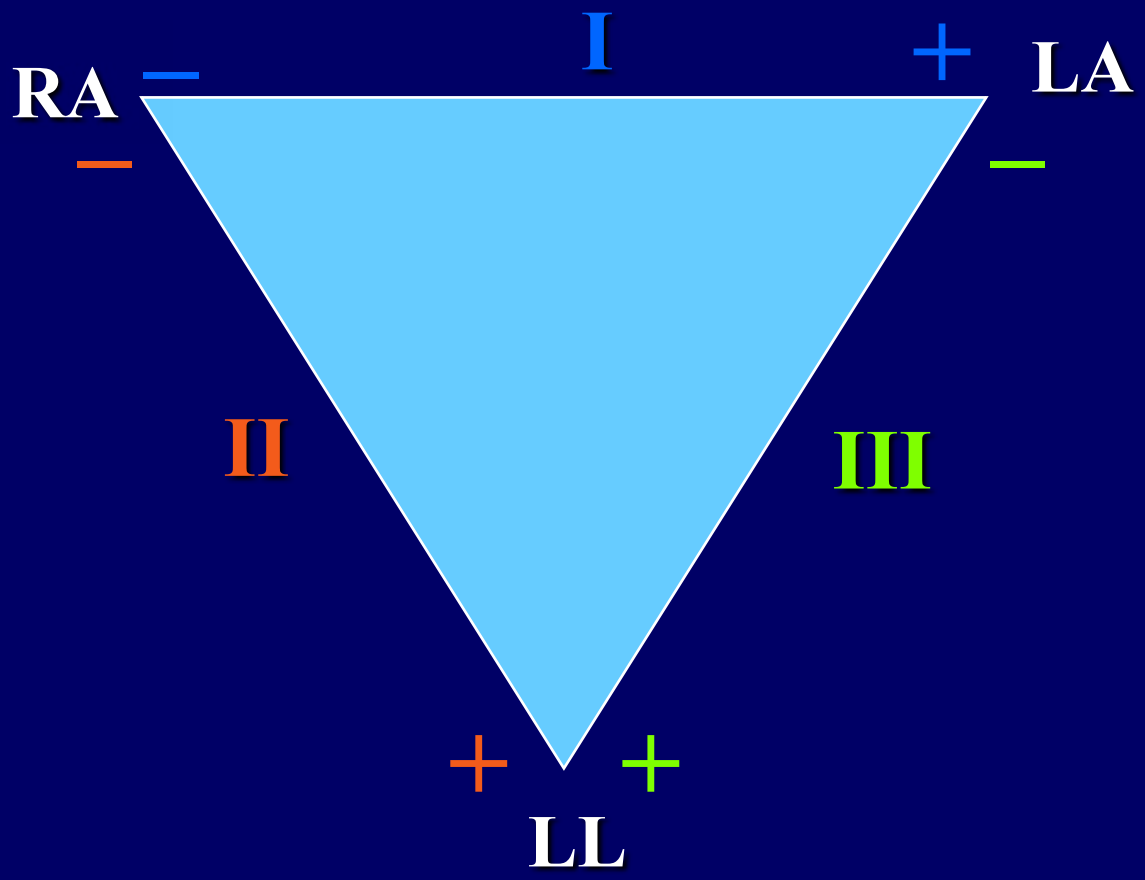
Mechanical events lag slightly behind electrical events.



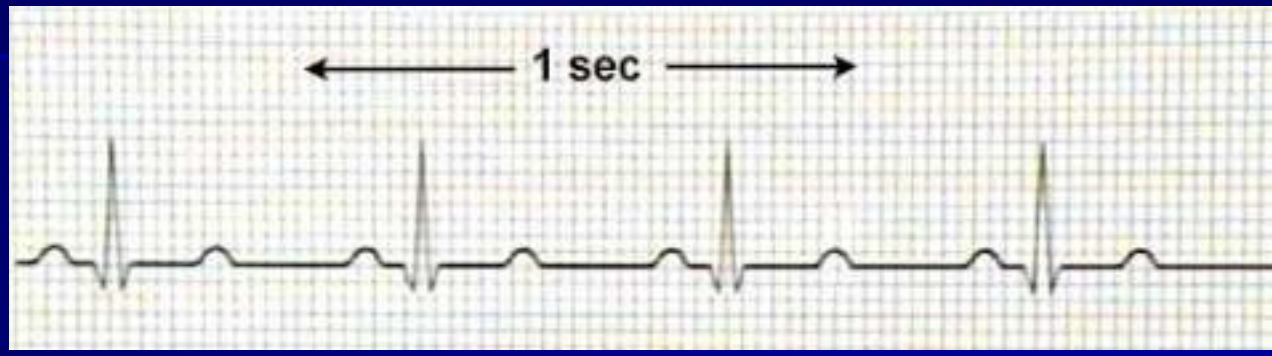

+1
Millivolts
0

Einthoven's Triangle and the 3 Limb Leads:

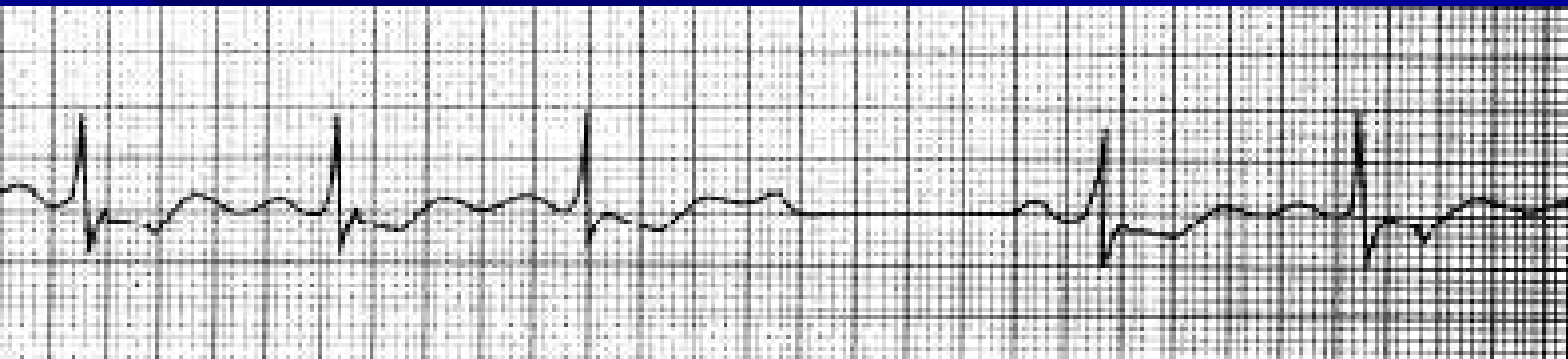




Info provided by EKG:



1. HR
2. Rhythm
3. Relationships of EKG components
   - each P wave followed by QRS complex?
   - PR segment constant in length? etc. etc.

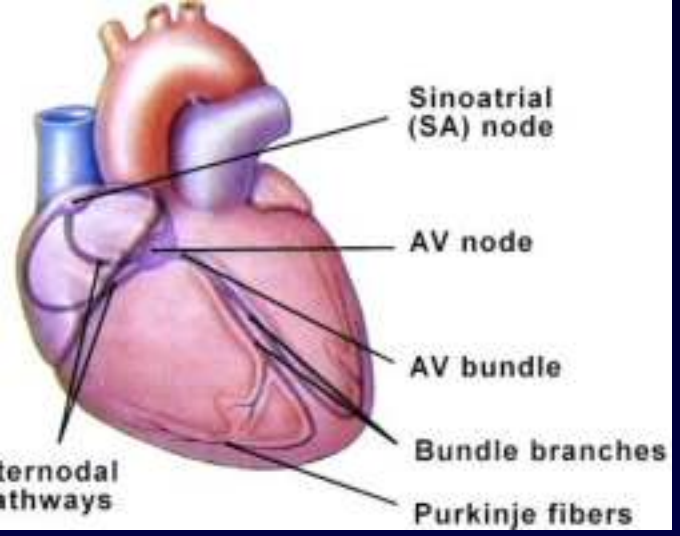


For the Expert:

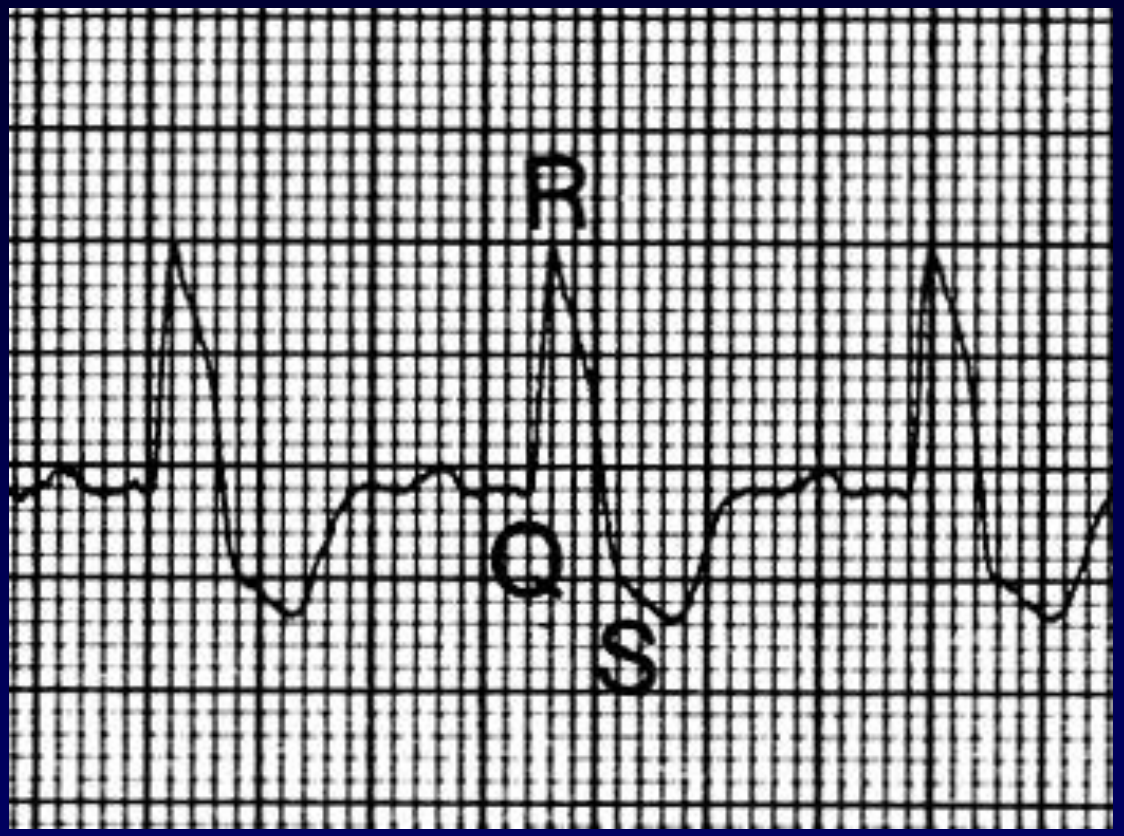
Find subtle changes in shape or duration of various waves or segments.

Indicates for example:

- **Change in conduction velocity**
- **Enlargement of heart**
- **Tissue damage due to ischemia (infarct!)**



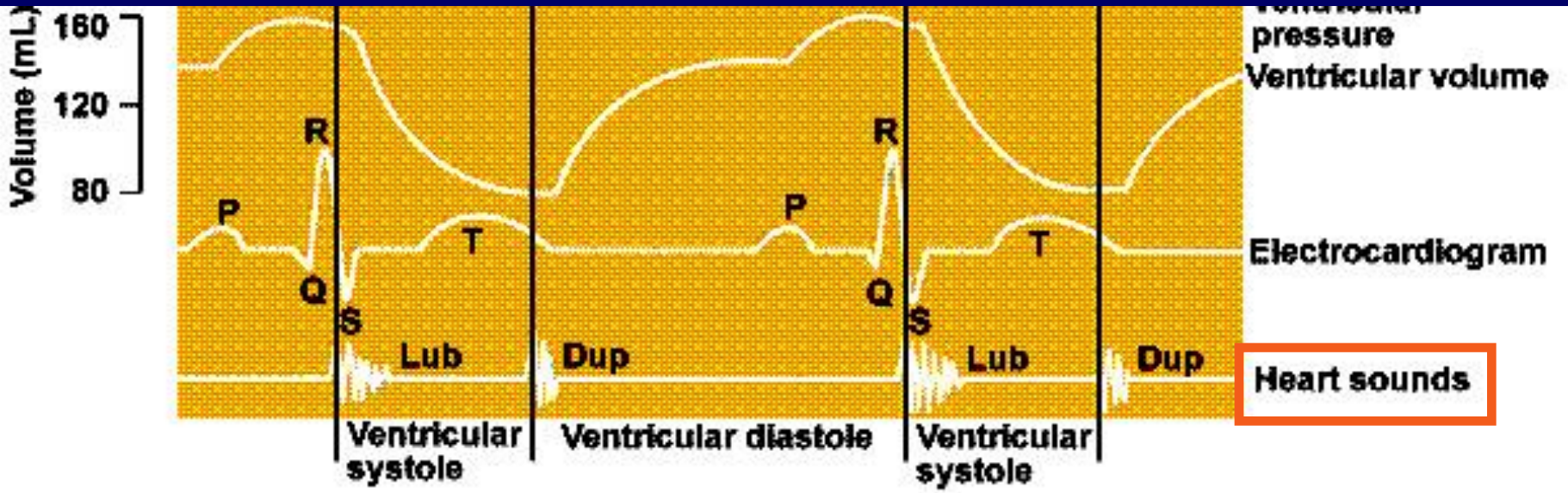
Prolonged QRS complex



Injury to AV bundle can increase duration of QRS complex (takes longer for impulse to spread throughout ventricular walls).

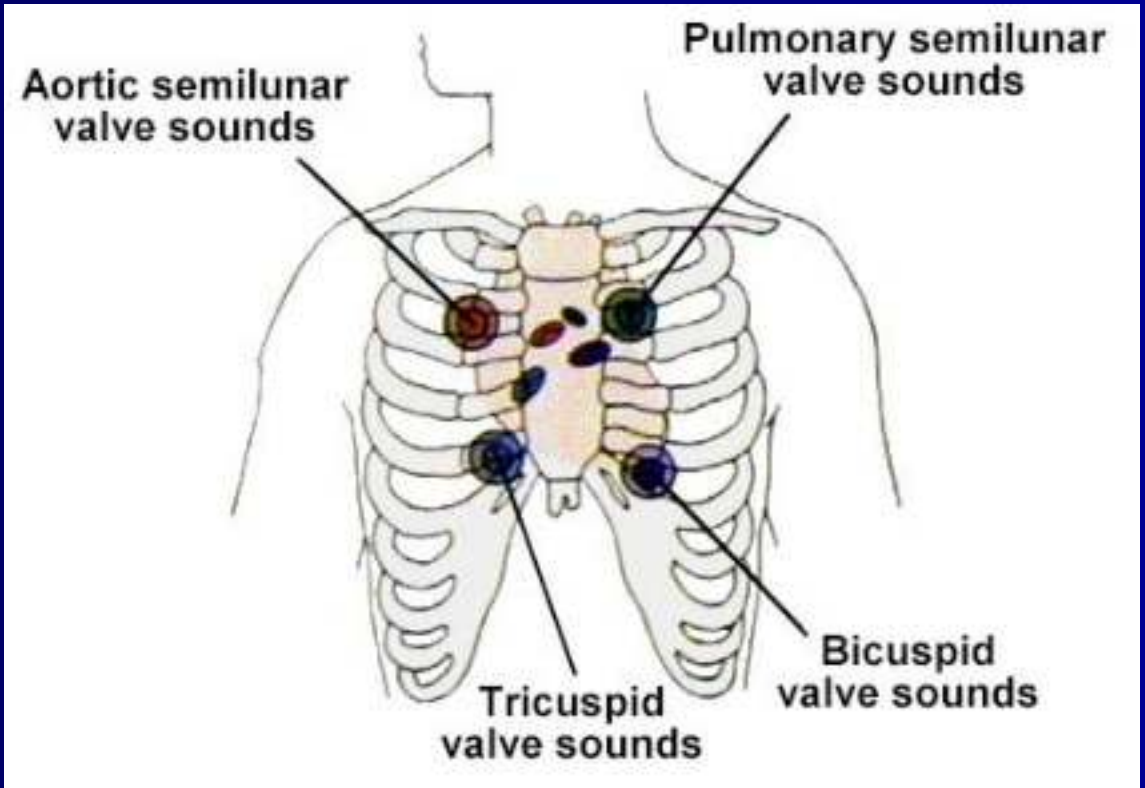
Heart Sounds (HS)

- **1st HS:** during early ventricular contraction ⇒ AV valves close
- **2nd HS:** during early ventricular relaxation ⇒ semilunar valves close



Gallops, Clicks and Murmurs

Turbulent blood flow produces heart murmurs upon auscultation



Cardiac Cycle: some definitions

- **Systole** (time during which cardiac muscle contracts)
  - atrial
  - ventricular
- **Diastole** (time during which cardiac muscle relaxes)
  - atrial
  - ventricular\
- **EDV** = End diastolic volume
- **ESV** = End systolic volume
- **SV** = Stroke Volume—that which is pumped in one stroke
- Heart at rest: **atrial & ventricular diastole**

SV	=	EDV	-	ESV
70mL	=	135 mL	-	65 mL

Cardiac Output (CO) – a Measure of Cardiac Performance

CO = HR x SV

calculate for average person!

- **HR controlled by ANS (p 475)**
  - parasympathetic influence ?
  - sympathetic influence ?
  - without ANS, SA node fires 90-100x/min
- *What happens with ANS when resting HR goes up (e.g. during exercise)?*

$$CO = HR \times SV$$

Force of contraction

Length of muscle fibers (Starling curve/law) due to **venous return**, *influenced by* skeletal muscle pump and respiratory pump

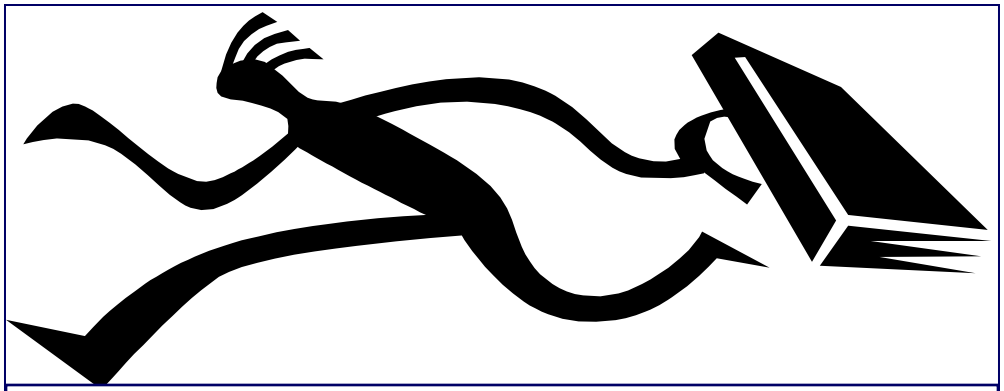
Sympathetic activity (and adrenaline)

venous constriction by sympathetic NS and

Increased Ca^{2+} availability

Frank-Starling Law (p 490)

- SV α EDV
  - i.e., the heart pumps all the blood sent to it via venous return
- Therefore, Venous Return = SV
- Preload = the amount of load, or stretch of the myocardium before diastole
- Afterload = Arterial resistance and EDV combined
- Ejection Fraction = % of EDV that is actually ejected; e.g., 70 ml/135ml x 100 = 52% at rest



Myocardial Infarction