

# Cardiac Muscle Contraction

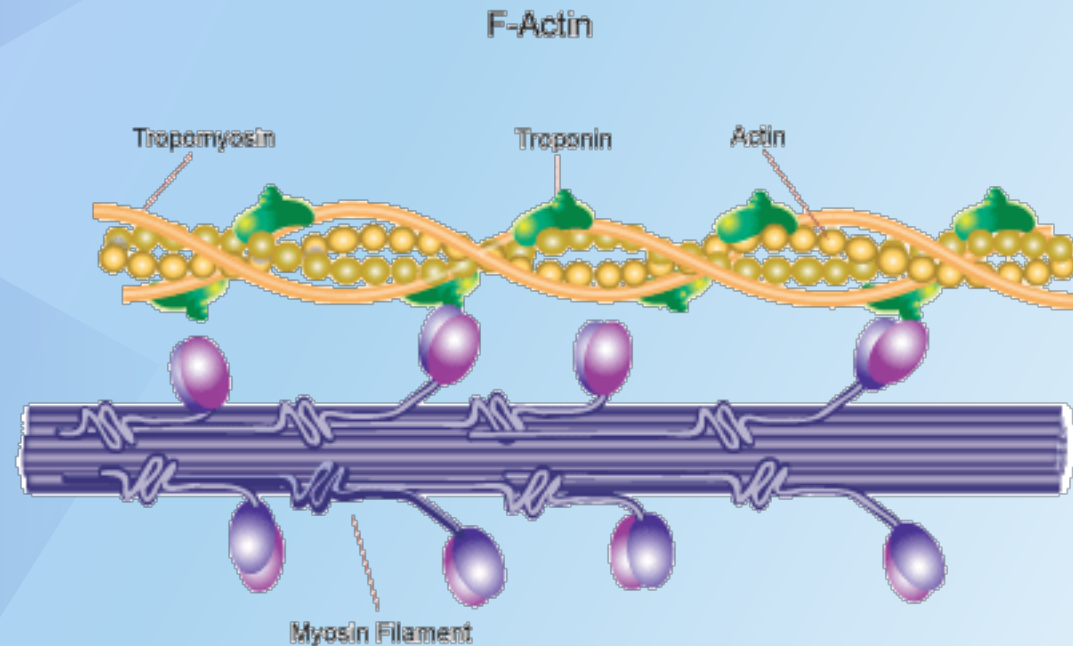
# Muscle fiber structure

## Thick filament

- Myosin

## Thin filament

- Actin
- Tropomyosin
- Troponin



*Organized in sarcomeres*

Let's draw! 😊

# Contractility (*inotropism*)

## Positive inotropic effects

- Increase contractility
- Increase:
  - Rate of tension development
  - Peak tension

## Negative inotropic effects

- Decrease contractility
- Decrease:
  - Rate of tension development
  - Peak tension

*Contractility is directly correlated with the intracellular  $[Ca^{2+}]$*



# Autonomic Effects

Back to the board!

# Effects of heart rate on contractility

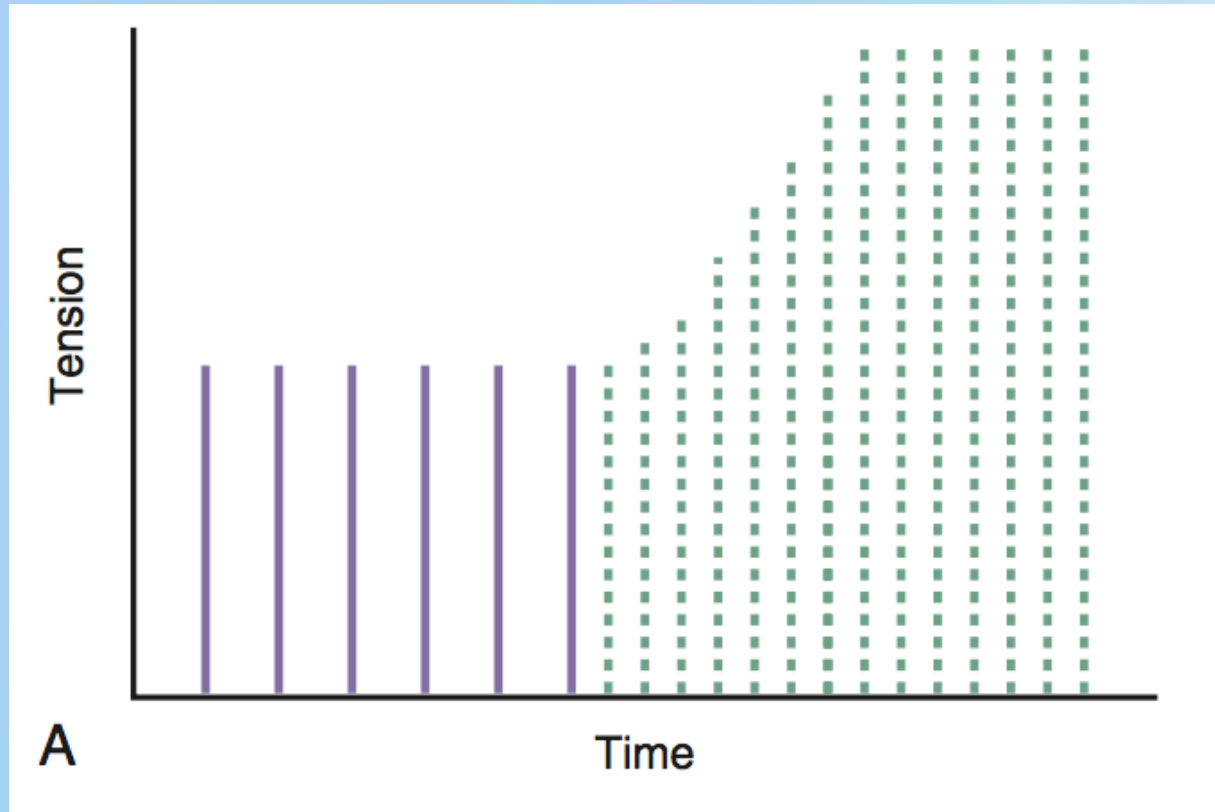
- Increased heart rate → increased contractility

Why?

1. Increased heart rate = more action potentials
  - This increases the total volume of trigger  $\text{Ca}^{2+}$  that enters the cell
  - If the increase in heart rate is caused by sympathetic stimulation, then the volume of  $\text{Ca}^{2+}$  influx per action potential is also increased
2. Increased  $\text{Ca}^{2+}$  influx = more  $\text{Ca}^{2+}$  stored in sarcoplasmic reticulum
  - $\text{Ca}^{2+}$  uptake is further increased if caused by sympathetic stimulation (*phospholamban*)

# Positive staircase effect

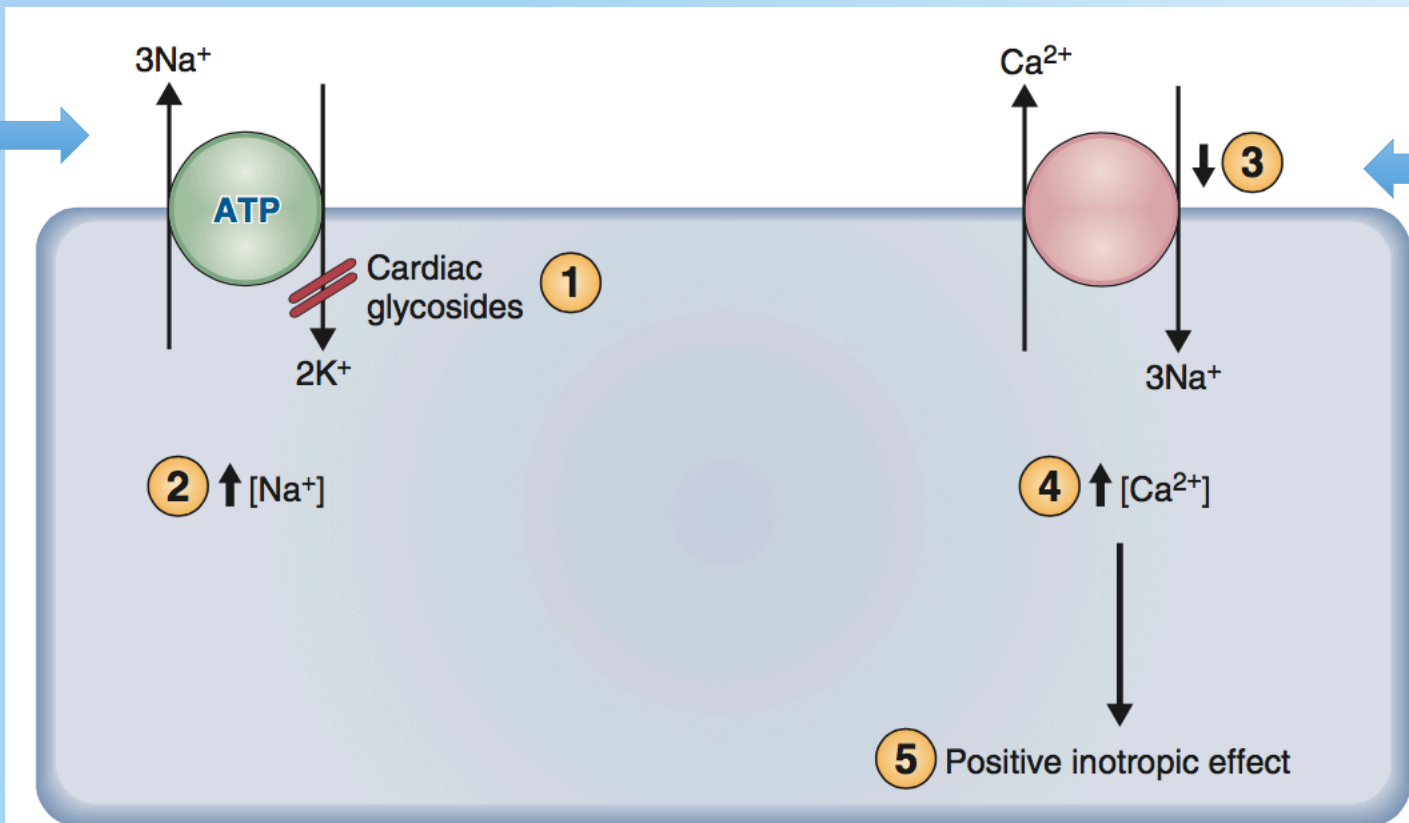
- Pattern of increase in tension when heart rate is increased



# Cardiac glycosides

- Positive inotropic agents (e.g digoxin, ouabain)
- Inhibit  $\text{Na}^+\text{-K}^+$  ATPase

Activity is dependent on ATP



Activity is dependent on  $[\text{Na}^+]$  gradient



# Length-tension relationship

- Contractility is based on length/ tension of muscle fibers
- $L_{\max}$  tension > long fiber tension > short fiber tension
  - $L_{\max}$  = 2.2 micrometers
- Increasing muscle length increases  $\text{Ca}^{2+}$  sensitivity of troponin C and  $\text{Ca}^{2+}$  release from SR



# Frank-Starling relationship

BUT FIRST.....

# ~~definitions~~

- Preload – the degree of overlap of muscle filaments
  - Same as end-diastolic fiber length
- Afterload – the pressure that the heart must pump against

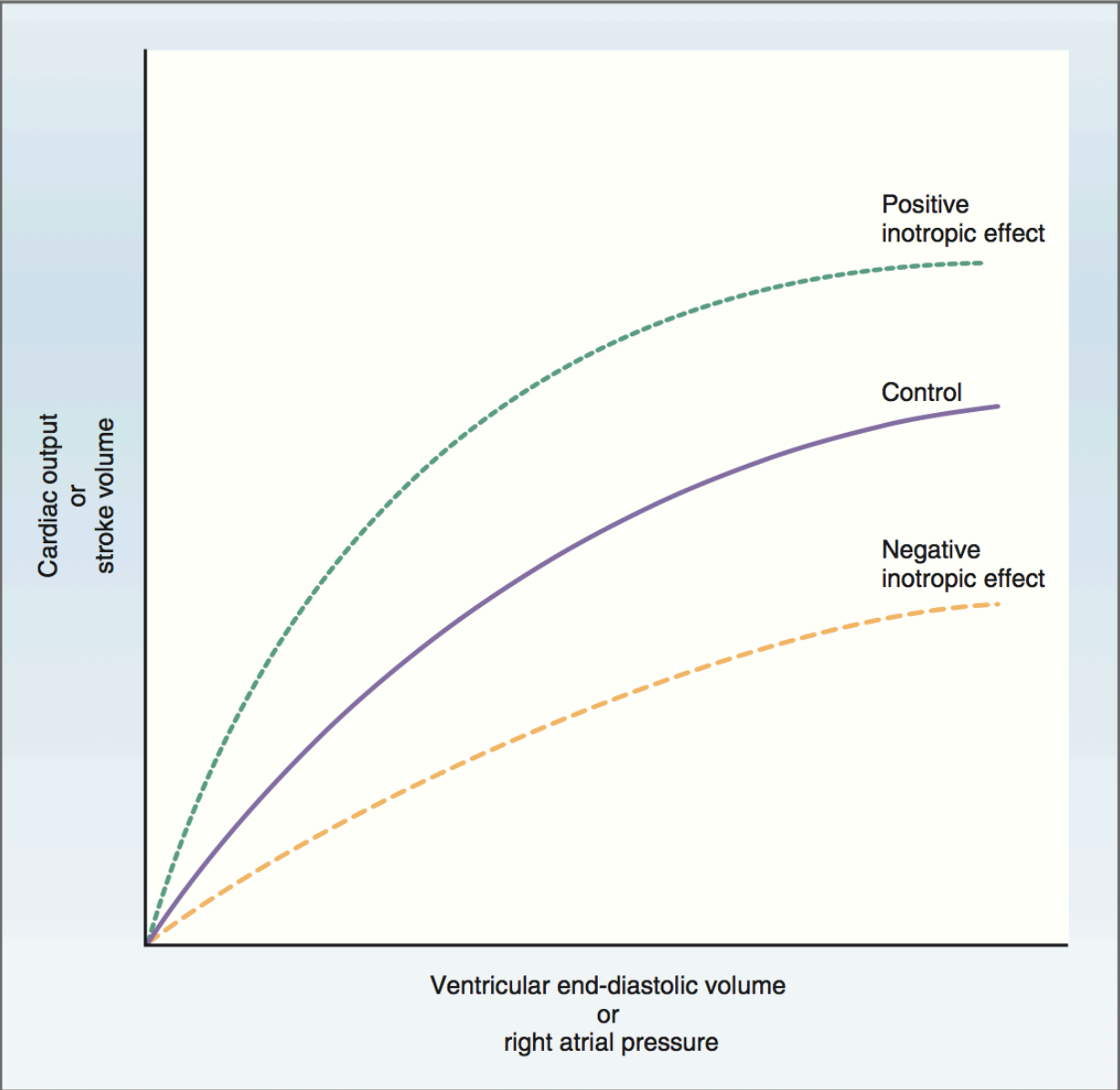
## ~~~more definitions~~~

- Stroke volume – amount of blood ejected per contraction
  - Usually about 70mL
- Ejection fraction – fraction of end-diastolic volume ejected per contraction
  - Usually around 55%
  - Indicator of contractility
- Cardiac output – amount of blood ejected per unit time
  - $CO = SV * HR$
  - E.g 5L/min

# Frank-Starling relationship

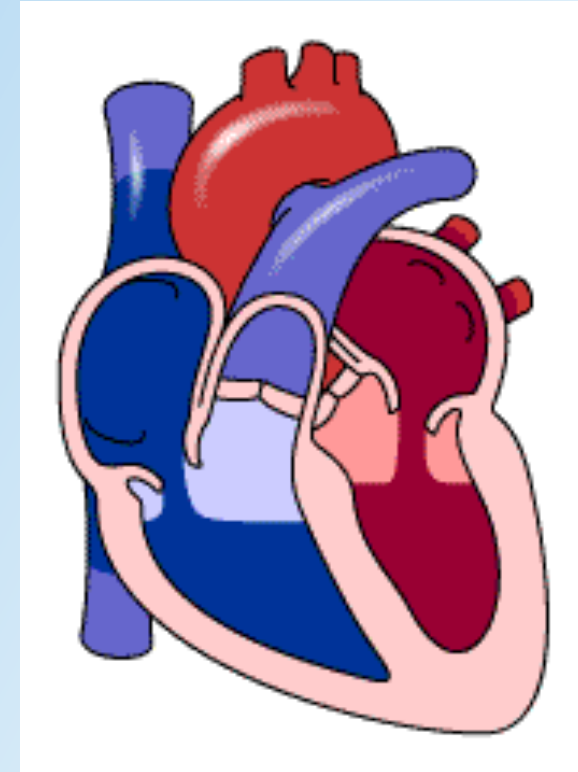


- *“the volume of blood ejected by the ventricle depends on the volume present in the ventricle at the end of diastole”*
- CO and SV are dependent on preload
  - Preload = Venous Return
- If VR is increased, CO will increase
- If VR is decreased, CO will decrease
  
- Increasing volume stretches muscle fiber length (preload!)



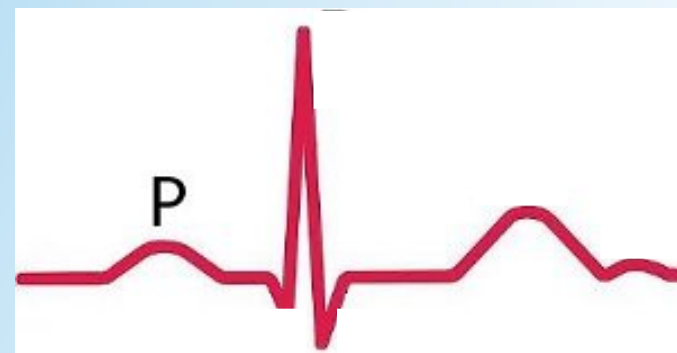
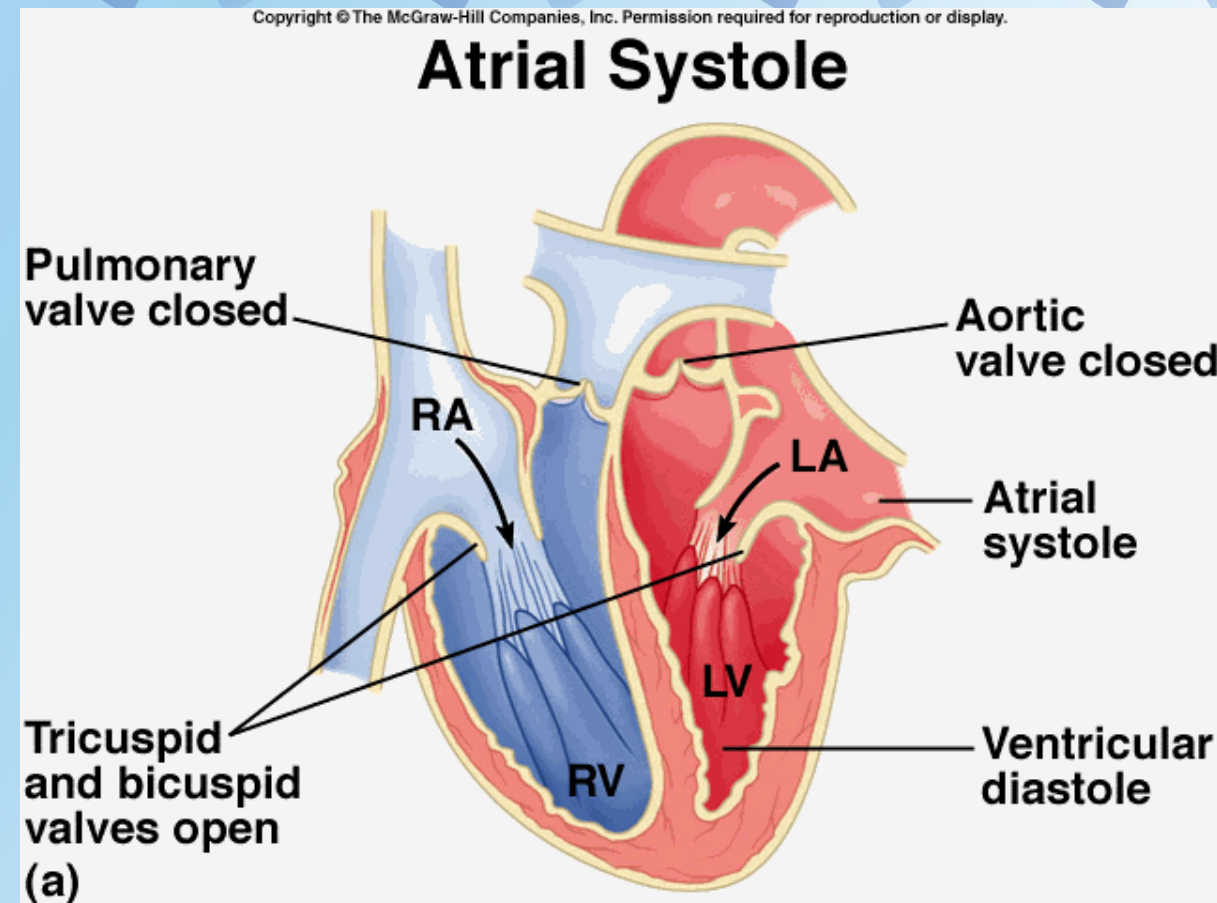
# Cardiac cycle

1. Atrial systole
2. Isovolumetric ventricular contraction
3. Rapid ventricular ejection
4. Reduced ventricular ejection
5. Isovolumetric ventricular relaxation
6. Rapid ventricular filling
7. Reduced ventricular filling



# Atrial systole

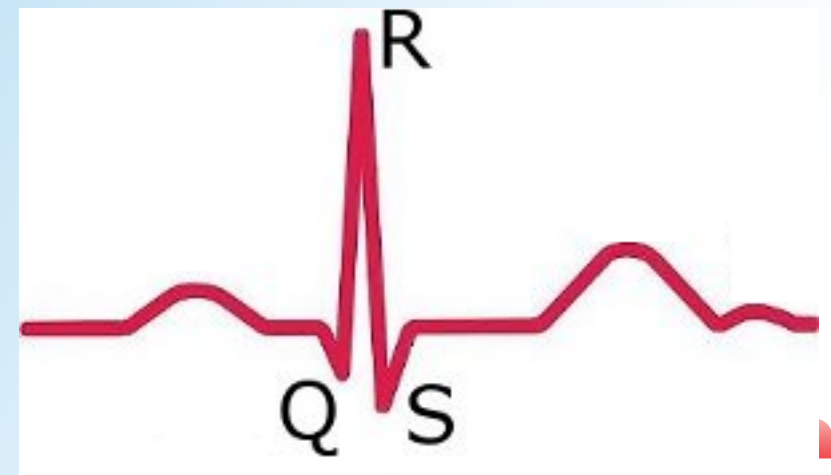
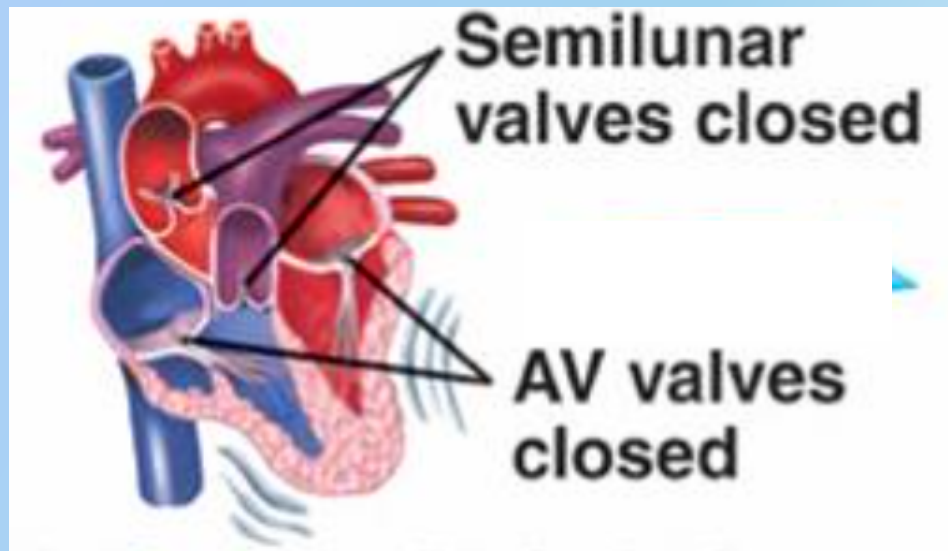
- Contraction of the left atrium
- Preceded by P wave on ECG
- Mitral valve is open
  - Passive ventricular filling precedes atrial systole





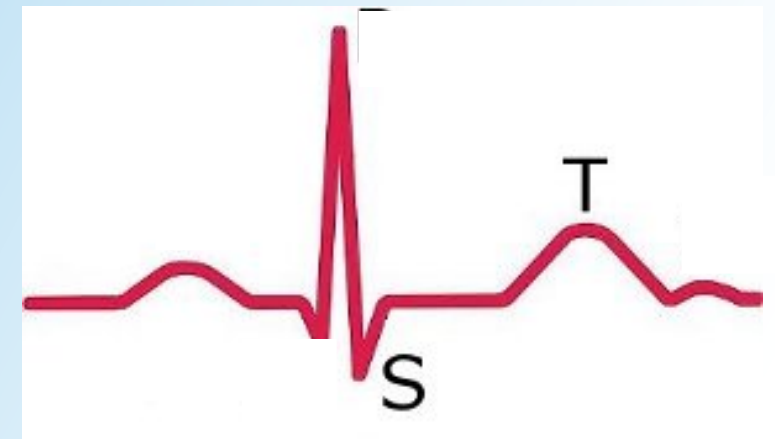
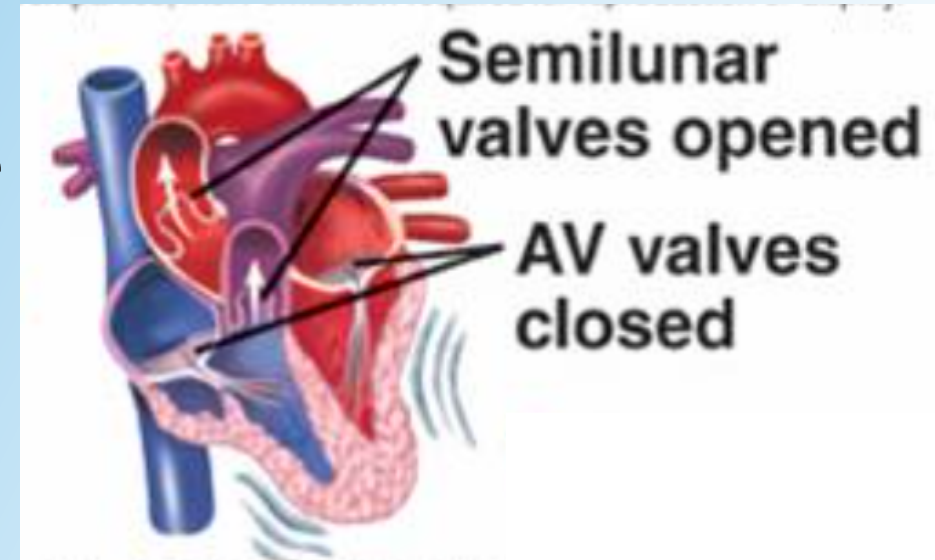
# Isovolumetric ventricular contraction

- Begins during QRS complex
- Closes mitral valve (S1)
- Ventricular volume stays the same; pressure increases



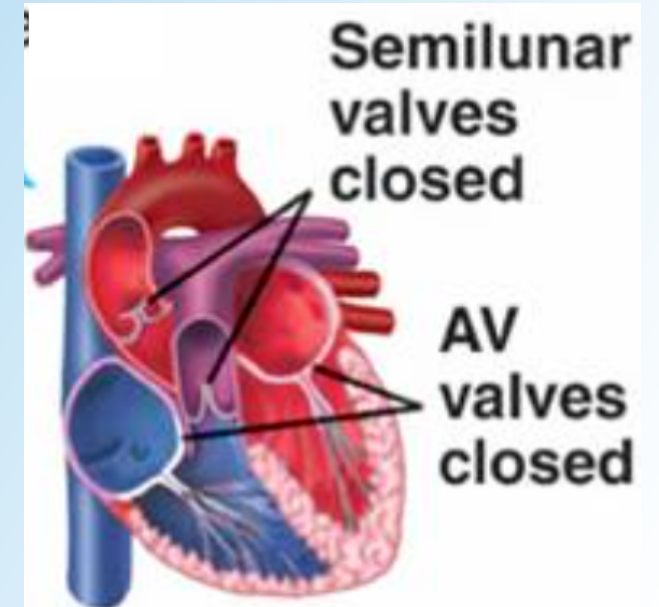
# Ventricular ejection

- Aortic valve opens when ventricular pressure becomes greater than aortic pressure
- Rapid ejection
  - Large pressure gradient
  - Most of stroke volume is ejected here
  - Atria begin to fill for next cardiac cycle
  - ST segment
- Reduced ejection
  - Small pressure gradient/ volume ejection
  - Ventricles are no longer contracting
  - T wave



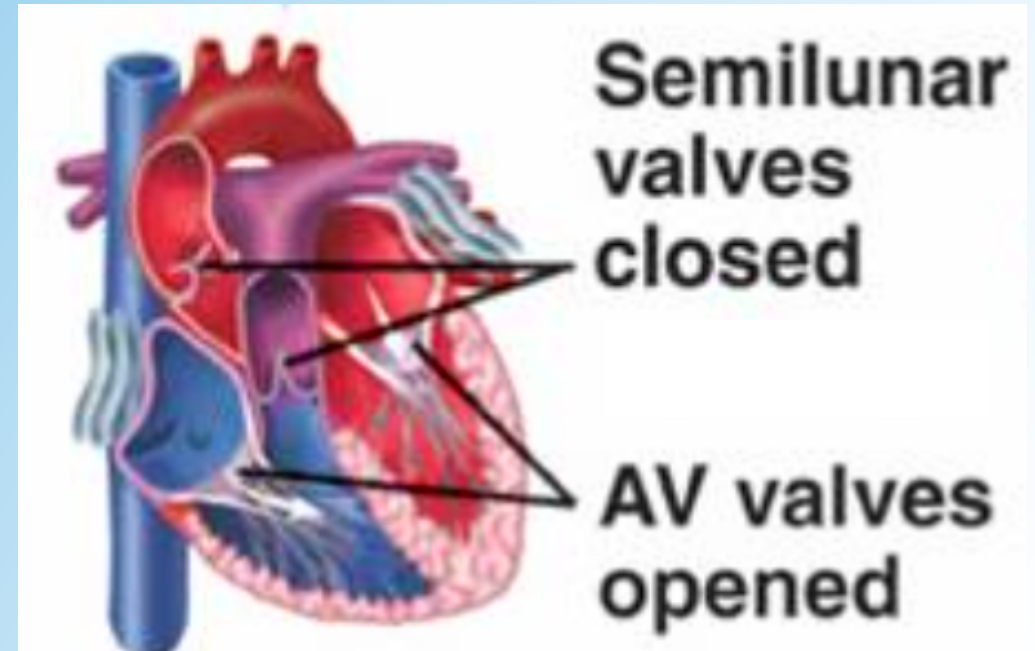
# Isovolumetric ventricular relaxation

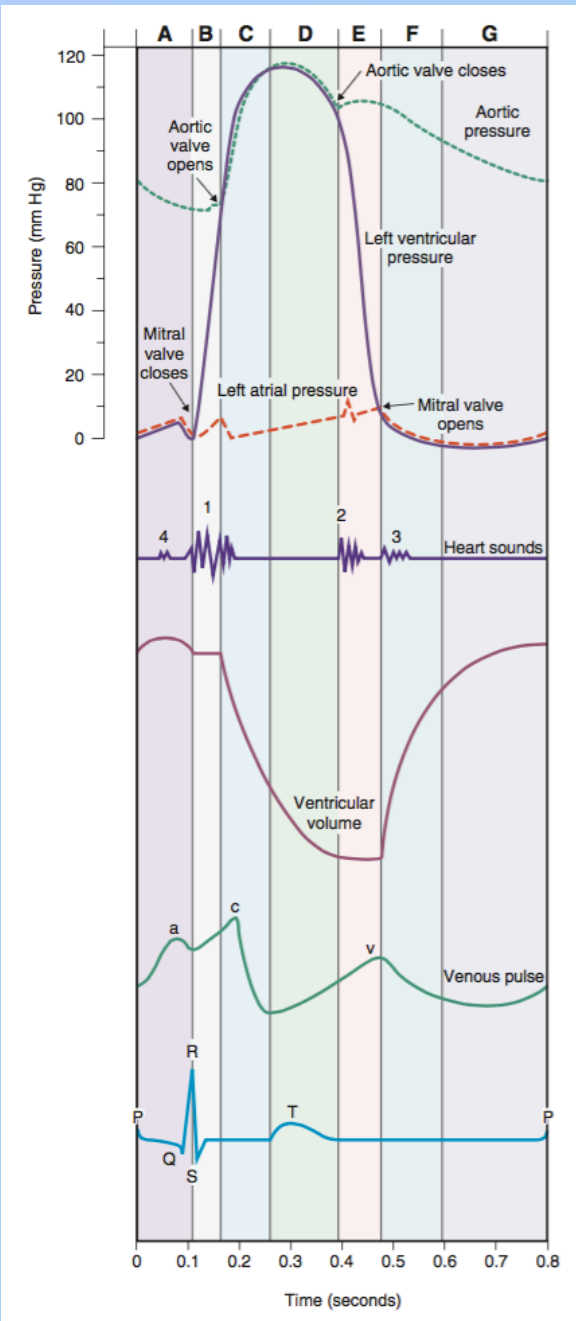
- Ventricles are fully repolarized
- End of T wave
- Left ventricular pressure decreases
- Aortic valve closes (S2)



# Ventricular filling

- Mitral valve opens
- Rapid
  - Ventricular pressure remains low
- Reduced (*Diastasis*)
  - Longest phase of cardiac cycle





An ECG marks electrical events – this marks electrical AND mechanical events

atria + ventricles  
relax + fill

atria contract

isovolumetric  
contraction

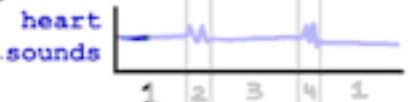
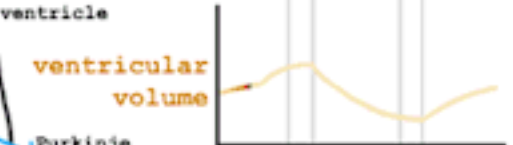
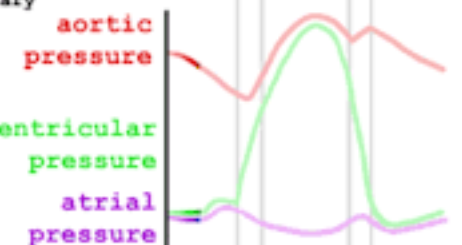
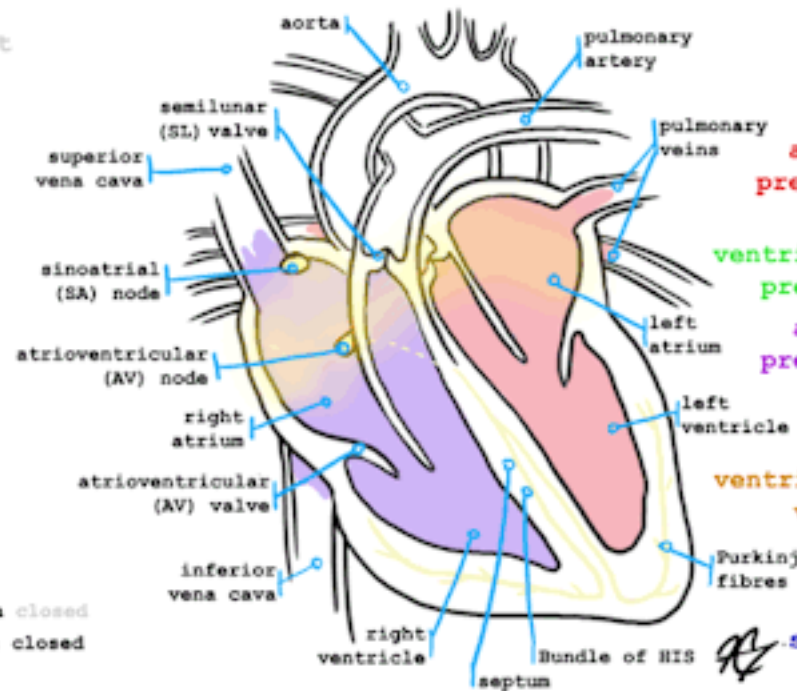
ventricles  
contract;  
ejection

isovolumetric  
relaxation

Time: 0.1s

diastole  
systole

AV valves open closed  
SL valves open closed



# Pressure-Volume Loop

- Putting it all together 😊
- Back to the board!