

# Lect. 7: Cardiovascular System

## Basics and Mammalian Heart

ASST.: READ CH. 12

### I. ROLE OF CARDIOVASCULAR SYSTEM?

#### A. Comparative Physiology provides clues

1. Comparing highly evolved systems that are well suited to that animals lifestyle
2. Don't get in habit that all animals are "striving" to get to human design
  - a) Turtle heart with incomplete division of ventricle does not have a septal defect awaiting evolutionary repair
  - b) There are benefits and costs of all design options

#### B. What is role of vascular system in most animals?

1. Movement of O<sub>2</sub> is critical
  - a) O<sub>2</sub> very insoluble in water
  - b) August Krogh: low solubility of O<sub>2</sub> limits rate of diffusion in living systems to tissues that are less than 1 mm from O<sub>2</sub> source
  - c) Drives much of evolution of cardiovascular systems
  - d) Also, results in evolution of **respiratory pigments**
  - e) CO<sub>2</sub> less of a problem because more soluble
2. Movement of nutrients
3. Removal of nitrogenous wastes
4. Distribution of water, ions and even heat,
5. Coordination of bodily functions via chemical messengers
6. Regulation of pH (through CO<sub>2</sub> regulation)
7. Locomotion
  - a) Molluscan Hydrostatic skeletons
  - b) Echinoderm tube feet
  - c) Insect wing expansion
  - d) Spider leg extension

**C. Small animals and protzoans have cells very close to outside environment**

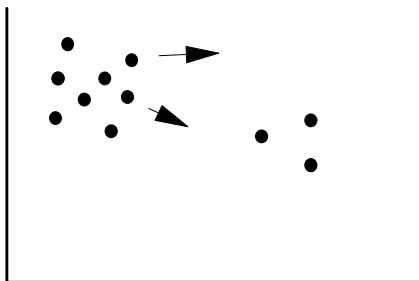
1. single cells
2. single cell layers of tissue
3. very narrow or flat body

**D. Fick's Law**

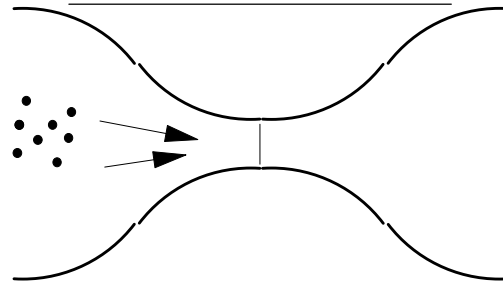
**1. Rate of Diffusion**

- a) Fick's Law describes factors that affect diffusion rate

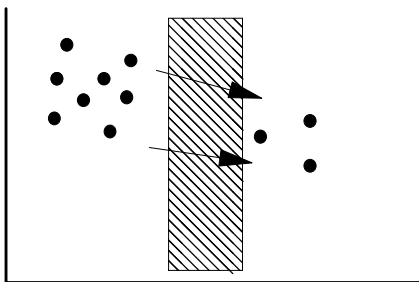
(1)  $C_1 - C_2$



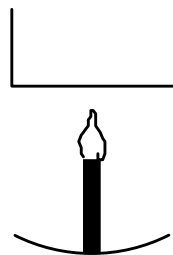
(2) (A) = Cross-Sect. Area



(3) Distance (L)



(4) Temperature



(5) Particle Size (D)



2. If you want to get big you need to overcome *Distance* factor for Fick's law
3. Circ. system provides plumbing to bring nutrients to and waste away from cells

**II. BASIC COMPONENTS**

**A. Circulating fluid - Blood**

**B. Vessels to move blood**

### C. Vehicles to exchange nutrients and waste

1. **Capillaries**
2. **Blood Sinus - Hemocoel**

### D. Pump(s)

1. **Tubular hearts:** contractile muscular tubes (arthropods and annelids)
2. **Ampular hearts:** usually accessory propulsive systems to boost fluid flow through specific peripheral networks
3. **Pulsating vessels:** propel circulatory fluid, often by peristaltic contraction
4. **Chambered hearts:** hearts that use muscular chambers to propel fluid

## III. TYPES OF SYSTEMS

### A. **Open system** (See diagrams in text pp. 468-469)

1. **Blood in vessels only part of the time**
2. **Hemolymph moves out of vessels and percolates through tissues**
3. **Exchange occurs in sinuses (hemocoel) where blood simply bathes tissues**
4. **Examples**
  - a) **Molluscs**
    - (1) Generally **Open** system
      - (a) exception - Cephalopods are closed system
    - (2) Blood pumped by chambered heart
    - (3) Moves through arteries to **Hemocoel**
    - (4) Back to myogenic heart through open sinuses
  - b) **Arthropods**
    - (1) Generally **Open**
    - (2) Heart receives blood from pericardium through holes (ostia)
    - (3) Several arteries
    - (4) No veins present - return through series of sinuses
    - (5) Heart is
      - (a) Tubular – Insects
      - (b) Or single chambered neurogenic organ that lies above gut - crustacea

### B. **Closed system**

1. **Blood always contained in defined tubes (vessels)**
2. **Plasma exits as Lymph into interstitial space.**
3. **examples**
  - a) **annelids**
    - (1) complete segmentation mandates use of closed system
    - (2) Dorsal and ventral main vessels
    - (3) Circumintestinal vessels are pulsatile and (along with dorsal vessel) act as pumps
    - (4) Capillaries for exchange
    - (5) Gas exchange in skin
      - (a) also parapodia
      - (b) gills where present

- b) vertebrates
- (1) More later

### C. Advantages of Open system

1. less resistance
2. lower energy requirement
3. less efficient pump required

### D. Advantage of Closed system

1. Greater pressure
  - a) faster exchange
  - b) can have ultrafiltration in kidneys
2. Can direct blood to and from specific areas as needs arise
3. Capillaries provide large surface area for exchange

### E. So most large and active animals have Closed system

1. E.g. look at cephalopods in contrast to other molluscs that have open system
2. Exception: *Insects* very active
  - a) limiting factor is gas exchange
  - b) although insects have open circ. system, gas exchange is in another system
  - c) Tracheal system provides **Closed system** for gas exchange

## IV. VERTEBRATE SYSTEM

### A. Basic Components

1. Pump - Heart
  - a) peristaltic pump
2. Transport - Arteries & Veins
  - a) Arteries
    - (1) muscular, very elastic walls
    - (2) high pressure
    - (3) windkessel effect smoothes out pressure differences
  - b) Veins
    - (1) thin walls
    - (2) very compliant
    - (3) extends
    - (4) acts as a reservoir for large quantity of blood
3. Exchange - Capillaries
  - a) thin wall
  - b) pressure forces fluids out

- c) requires **Lymphatic system** for return

## V. HEART

### A. Types

#### 1. Neurogenic

- a) Crustacea, Insects
- b) Beat is set by collection of **neurons** located on heart
- c) Cardiac ganglion
- d) These excite **follower cells** which stimulate heart muscle like any other cells

#### 2. Myogenic

- a) Molluscs and Vertebrates
- b) Beat initiated by muscle cells
- c) Rate can be influenced by neural input from **ANS**
- d) Heart can beat completely absent neural connections

### B. Mammalian Heart

#### 1. Cardiac muscle is modified striated muscle

- a) smaller cells
- b) electrically linked to form functional syncytium
- c) Actually 3 types of heart muscle cells
  - (1) SMALL CELLS
    - (a) only weakly contractile
    - (b) Generate rhythm
    - (c) In Pacemaker regions
      - (i) sinus node
      - (ii) AV node
  - (2) LARGE CELLS
    - (a) in ventricular endocardium
    - (b) Also weakly contractile
    - (c) Specialized for **fast conduction**
    - (d) spreads signal over ventricle quickly
  - (3) INTERMEDIATE CELLS
    - (a) Bulk of heart
    - (b) Strongly contractile
- d) Various parts of heart are specialized for
  - (1) pacemaker
  - (2) conduction

#### 2. Regions of Heart

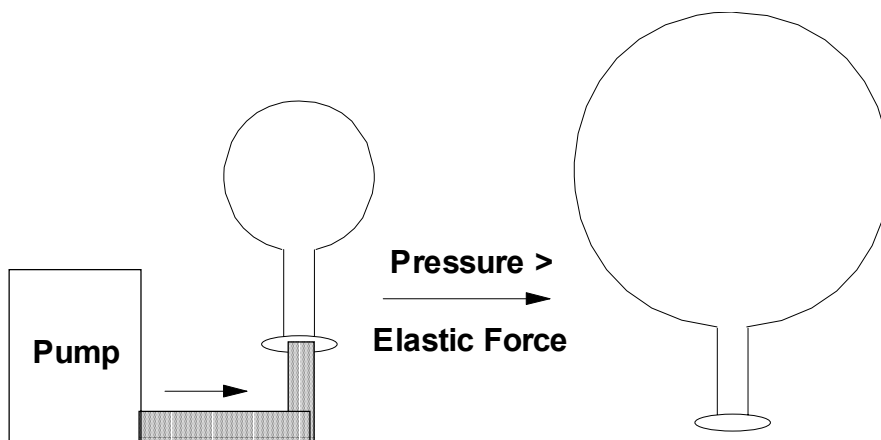
- a) **Atria** - Receive venous blood
- b) **Ventricles** - pump blood away from heart
- c) *Right side Ventricle*

- (1) receives deoxygenated blood from body
- (2) sends it to lungs
- d) *Left side Ventricle*
  - (1) receives oxygenated blood from lungs
  - (2) sends it to body
  - (3) since it has farther to go this ventricle is much larger
- e) NOTE: **Arteries always Away from Heart; Veins Toward Heart**
- f) In Pulmonary System
  - (1) veins carry Oxygenated blood
  - (2) Arteries deoxygenated blood
- g) In **Systemic Circulation** opposite is true

### 3. Basic Heart Beat

- a) **Diastole**
  - (1) Atria and Ventricles Fill
- b) **Systole**
  - (1) Atria and then Ventricles contract
- c) Movement of blood
  - (1) caused by Pressure differences

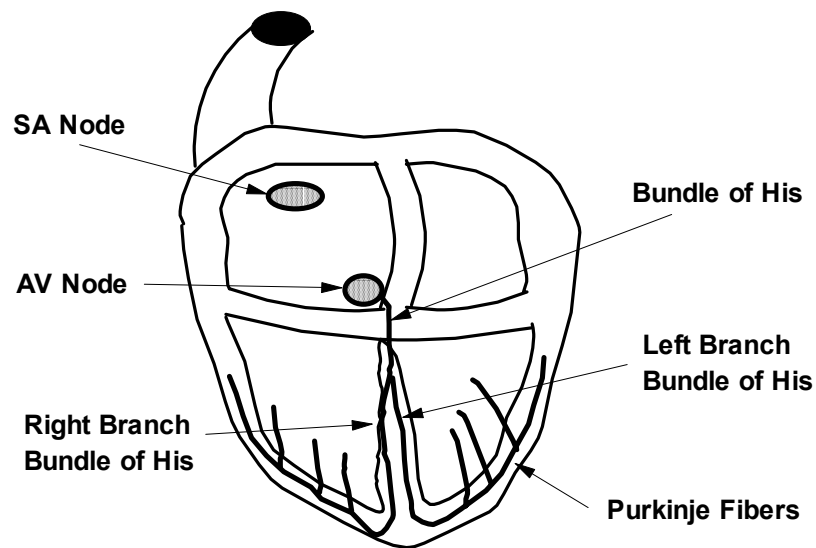
$$\text{Flow} = \frac{\Delta P}{R}$$



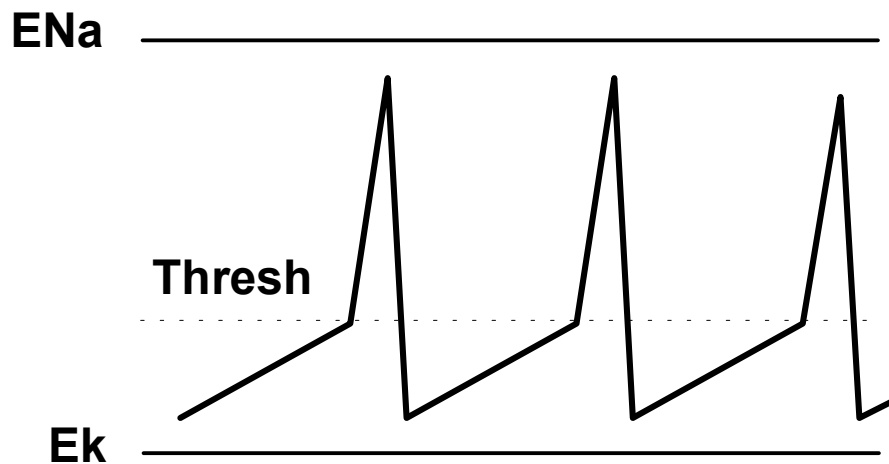
- (2) controlled by **Valves** where pressure diff. would make it go wrong way
- d) Pattern of contractions result of elaborate **Electrical system**

### 4. Electrical system

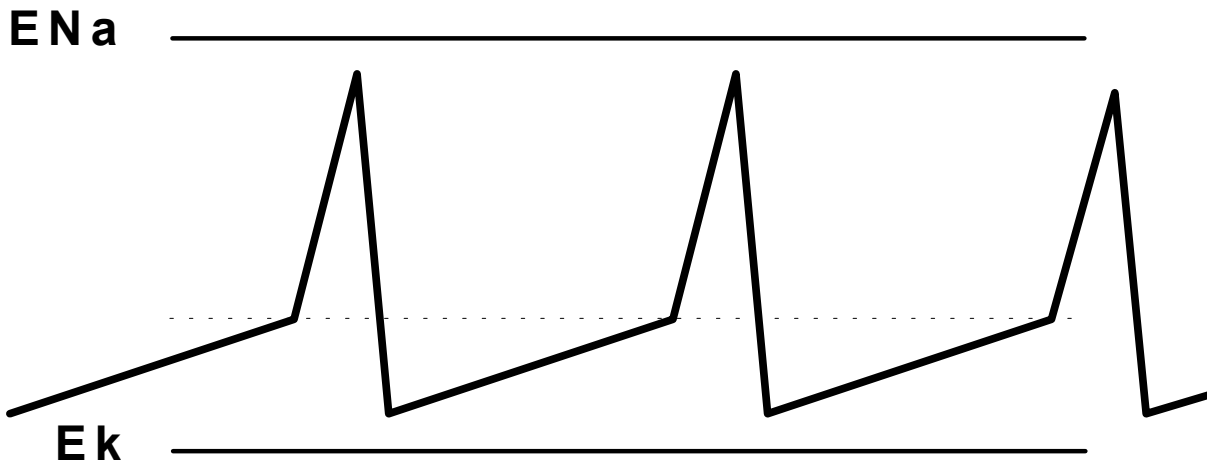
- a) Pacemaker: Sinoatrial (sinus) Node: Located in Sinus Venosus



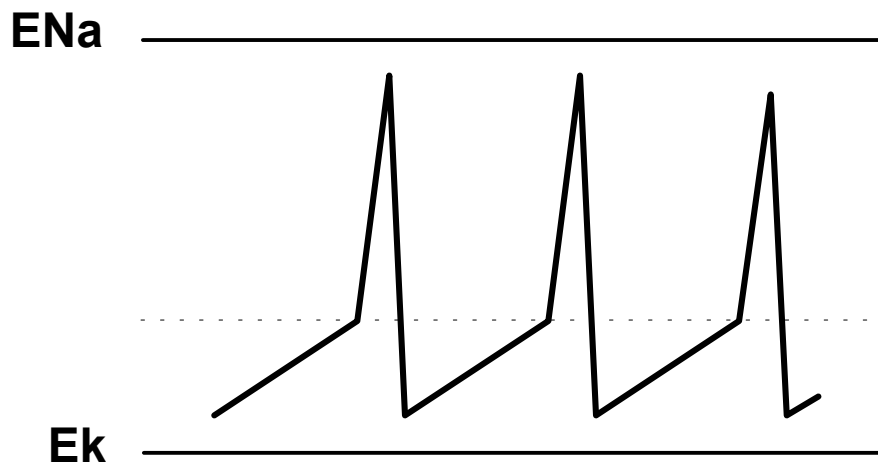
- b) Cells undergo steady depolarization to threshold
- (1) Pacemaker pot  $\rightarrow$  Cardiac Action pot.
  - (2) Na cond. is normally moderately high
  - (3) Immediately after AP, K cond. also high
  - (4) K cond. slowly decreases and Memb. Pot. drifts upward
  - (5) When it reaches threshold  $\rightarrow$  A.P.



- c) Control of Heart Rate
- (1) Transmitters can alter pacemaker rhythm
  - (2) **ACh (vagus) increases K cond.**
    - (a) this delays rise of pacemaker pot.
    - (b) threshold is reached slower
    - (c) heart rate delayed

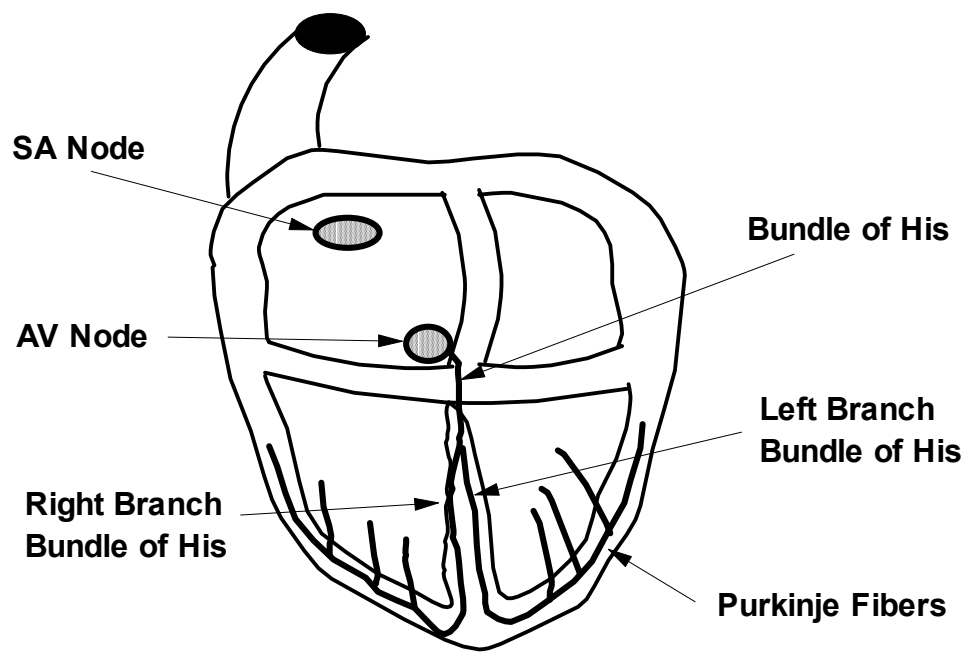


- (3) **Nor-epinephrine (Sympathetic Nerves) decreases time-dependent K cond.**
- (a) also inc. Na and Ca cond.
  - (b) this speeds up depol.
  - (c) so threshold is reached faster



### C. Spread of Excitation

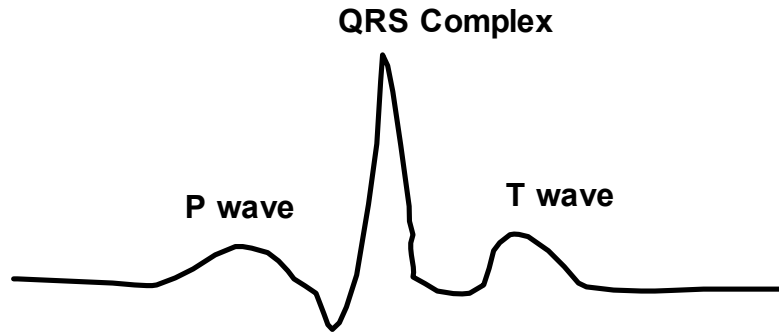
1. From Pacemaker excitation spreads over both Atria at velocity of  $\sim 0.8$  m/sec
2. Atria electrically connected to Ventricles *only via Atrioventricular (AV) node*



3. **Other regions are non-conductive connective tissue**
4. **From AV node excitation spreads through a series of Junctional fibers**
  - a) slowed to  $\sim 0.05$  m/sec
  - b) AV  $\rightarrow$  Junctional fibers  $\rightarrow$  nodal fibers  $\rightarrow$  Bundle of His
  - c) **Bundle of His**
    - (1) Right and left bundles
    - (2) covers endocardium of ventricles
    - (3) Conduction speeds up to 4-5 m/sec
  - d) **Purkinje Fibers** spread waves rapidly over entire area of **ventricle**
    - (1) **wave spreads from apex upward**
    - (2) pushes blood up and out
5. **Master Pacemaker: Sinoatrial (SA) Node: Located in Right Atrium**
  - a) Other nodes (e.g. Bundle of His) also have pacemaker capacity
  - b) But are slower
  - c) Thus, SA drives system
  - d) Blockage or damage to SA node can release other nodes
  - e) Results in slower rhythm
  - f) Bundle of His (25 - 40 beats / min.)

#### **D. EKG**

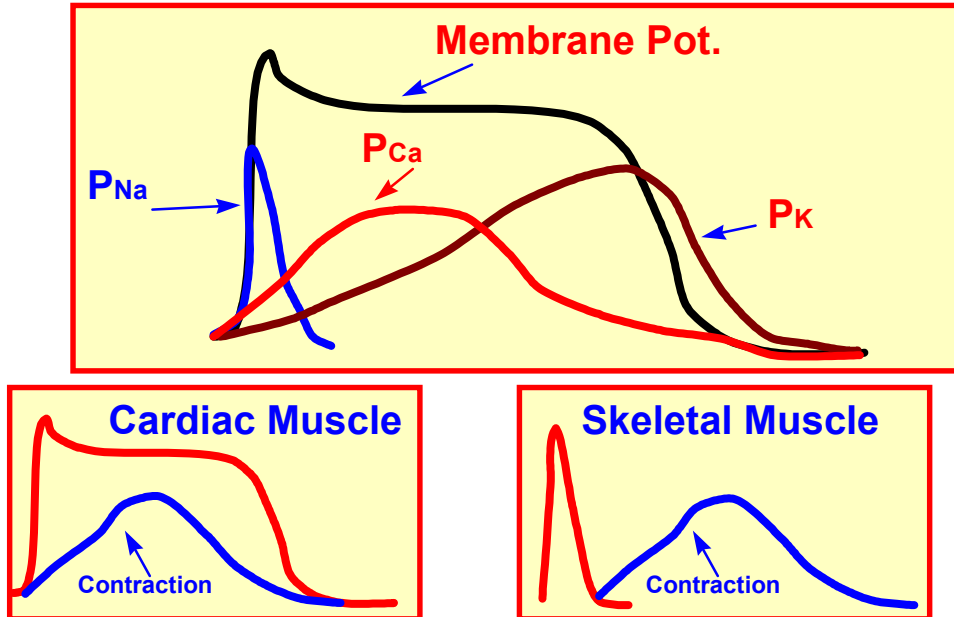
1. **because of large number of cells involved, can detect electrical activity all over body**
2. **EKG has standard form that represents electrical events in heart**
3. **EKG waveform**



- a) **P wave:** Depol. of atrium
- b) **QRS complex:** Depol. of ventricle
  - (1) QR is large because of synch. in conduct. from AV node
  - (2) RS is wave traveling in opposite direction
- c) **T wave** is Repol. of ventricles
  - (1) not very synchronized
  - (2) thus small wave

## E. Action Potentials

### 1. Broader than striated muscle



- 2. As a result of broad AP cardiac muscle is in a refractory state until heart has returned to relaxed state
- 3. No chance for summation and tetanus
- 4. Rapid depol. due to inc. g<sub>Na</sub>
- 5. g<sub>K</sub> is delayed

6. **Depol. maintained by inc. in gCa**
7. **Inc. in gCa provides Ca for contraction**
  - a) no highly specialized sarc. retic. for Ca store
8. **Activity spreads over heart via gap junctions between muscle cells**

## F. Mechanical

1. **Blood moves from area of high pressure to one of low pressure**
2. **Chambers contract to increase pressure and move blood**
3. **Valves prevent movement in opposite direction when pressure gradient is reversed**
4. **Valves**
  - a) Atrioventricular (AV) valves
    - (1) Guard atrium to ventricle path
    - (2) **Bicuspid: Left**
    - (3) **Tricuspid: Right**
    - (4) Flaps of tissue anchored with cords to fibrous muscles in wall
  - b) Aortic valves
    - (1) cups
    - (2) pressure builds in aorta and ventricle relaxes
    - (3) blood tries to move back into ventricle
    - (4) cups close and prevent backflow
5. **Cardiac Cycle (Diagram P. 478)**
  - a) **Passive filling**
    - (1) mid-diastole
    - (2) AV valves open
    - (3) blood enters atria from venous systems
    - (4) passes through atria and fills ventricles directly
    - (5) most of ventricle fills passively
  - b) **Atrial contraction (*systole*)**
    - (1) Atria contract
    - (2) Active filling "**tops off**" ventricles
  - c) **Isovolumetric Ventricular Contraction**
    - (1) Ventricles contract
    - (2) AV valves close
    - (3) Aortic valves have not opened yet, because pressure in aorta > pressure in ventricle
    - (4) Tension builds but **NO DECREASE IN VOLUME**
  - d) **Ejection**
    - (1) Pressure in ventricle > pressure in aorta
    - (2) Aortic valves **open**
    - (3) blood is ejected into aorta and pulmonary artery
  - e) **Isovolumetric Ventricular Relaxation**
    - (1) Ventricles begin to relax
    - (2) blood tries to flow back into ventricle (down pressure gradient)
    - (3) Aortic valves close to prevent backflow
    - (4) With both sets of valves closed relaxation is **isometric**
  - f) **Return to Passive Filling**

- (1) AV valves open and passive filling begins
- g) So contraction is both *Isometric* and then *Isotonic* at different times in the cycle
- h) Energy expended during cardiac cycle
  - (1) Work
    - (a) pressure changes
    - (b) flow of blood
  - (2) Frictional forces
  - (3) Heat
- i) **Use figure 12-11 p. 478 to correlate Mechanical and Electrical cycles**
- j) Compare Left and Right Ventricles
  - (1) Left Pressure >> Right Pressure
  - (2) Left Volume = Right Volume
    - (a) Required for Closed system with completely separate chambers

## 6. Cardiac Output

- a) Volume of Blood Pumped **per Unit Time**
- b) In mammals defined as volume ejected from *either* left or right ventricle, not combined
- c) these must be equal in mammals

## 7. Stroke Volume

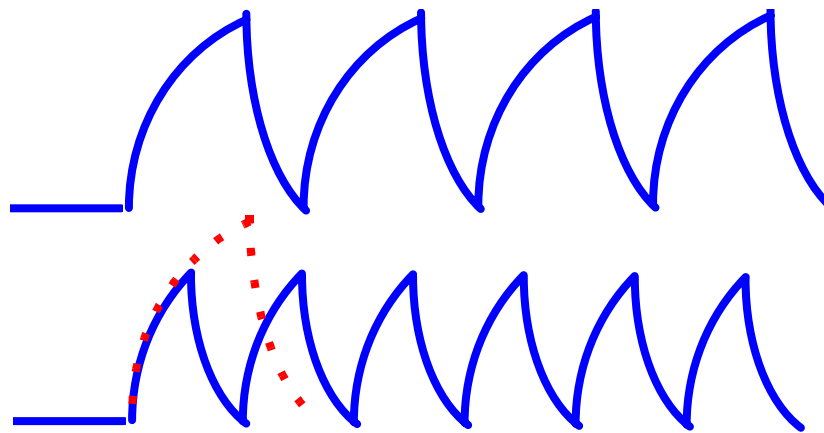
- a) vol. of blood ejected by each beat
- b) Measured as difference between vol. of vent. just before contraction (**end-diastolic vol.**) and vol. at end of contraction (**end-systolic vol.**)

## 8. Cardiac Output = Stroke Vol. X Heart Rate

- a) At rest 0.07 L/beat X 72 b/min = 5.0 L/min
- b) Total blood vol. is ~ 5L
- c) So entire vol. is circulated every minute
- d) Can calculate mean stroke vol by dividing Cardiac Output by heart rate

## 9. Can increase cardiac output by either

- a) increasing contraction **and** keeping rate constant
- b) increasing rate **and** keeping contraction constant
- c) **if one goes up and other goes down you get nowhere**
  - (1) With faster rate, **less time** for contraction
  - (2) should result in **lower** Stroke Volume



**10. Alter heart rate by**

- a) Changing pacemaker
- b) Parasymp. (Vagus) slows H.R. (ACh)
- c) Symp. increases H.R. (norepi)
- d) Epinephrine from adrenal medulla also inc. H.R.

**11. Alter Stroke volume by**

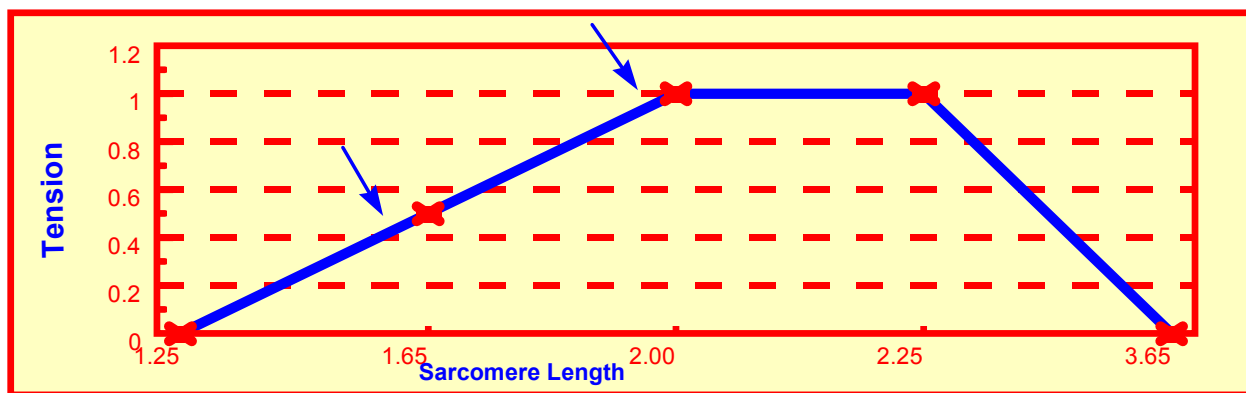
- a) Extrinsic effects (e.g. A.N.S.)
- b) Intrinsic effects

**12. Starling Mechanism**

- a) Inc. Venous filling pressure
  - (1) End-diastolic vol. increases
  - (2) End-systolic vol. increases only slightly
  - (3) So stroke vol. increases
  - (4) SO GREATER VOL. IN BLOOD MAKES FOR MORE POWERFUL CONTRACTION
- b) Inc. **Arterial pressure** (by constriction)
  - (1) Also get inc. end-diastolic vol. (harder to get blood out against pressure gradient)
  - (2) But end-systolic vol. also increases
  - (3) So stroke vol. remains constant

**13. Otto Frank**

- a) measured length-tension relationship for frog myocardium
- b) just like skeletal muscle
- c) but cardiac cells only operate in range of rising tension
- d) So more stretch on muscle → greater tension when stimulated



- e) Also, affinity of Troponin C to Ca increases with increased volume of blood

#### 14. Frank-Starling Mechanism

- More blood fills ventricle
- Stretches muscle cells more
- Results in more tension
- More forceful contraction increases Stroke Volume
- Happens *without* neural control

#### 15. Sympathetic (Neural) Control

- Allows for increase in **Cardiac Output** via Increased **Heart Rate**
- Must inc. rate **AND** keep stroke volume at least constant
- Effect of **Epinephrine** and **Norepinephrine** release
  - Heart rate increases as a result of **cond. changes** in pacemaker cells
  - Cond. Velocity over heart inc. → near synchron. contraction of ventricle
  - Rate of ATP production increases
  - 2 and 3 → stronger contraction
  - So heart rate can rise up to 250/min with little change in stroke volume
  - Blood moves faster to capillaries
- Mechanisms
  - B1 receptors
  - Activates G Protein and 2nd messenger cascade
  - Protein Kinase activates
    - More Ca per twitch
    - Increased phosphorylation of Myosin
      - makes inc. rate of myosin cyclign
      - inc rate of tension development
    - Inc. rate of Ca re-uptake
      - Inc. rate of relaxation**

#### 16. ACh from vagus (parasymp) has opposite effect

- lowers rate to 20-30/min

b) slight decrease in strength of contraction

**17. So Cardiac output can be controlled by either**

a) **Intrinsic factors** (Frank-Starling mech)

b) Extrinsic factors (ANS input)

c) ANS is more controlled

d) After heart transplant symp. and parasymp. nerves not reconnected

e) rely solely on intrinsic factors