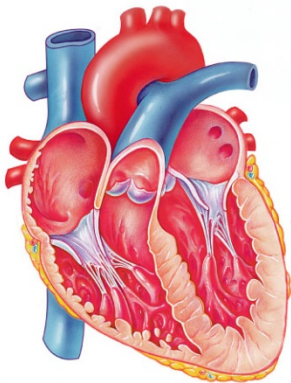
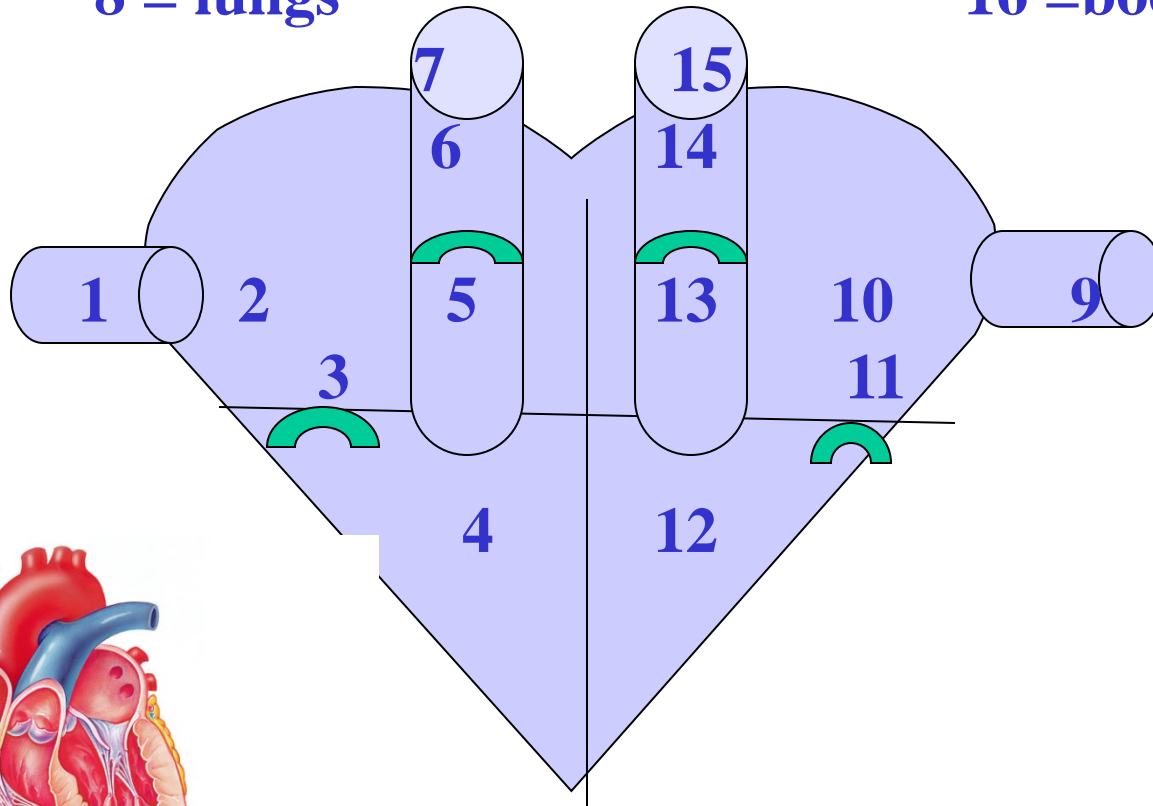


# Engineer's Heart

8 = lungs

16 = body



(e)  
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# Heart Flow

Oxygen-poor

Mixed

Oxygen-rich

## 8. Pul. Circ.

1. vena cava (2)

2. RA

3. tricuspid valve

4. RV

5. pul. semi. valve

6. pulmonary trunk

7. pulmonary arteries (2)

9. pul. veins (4)

10. LA

11. bicuspid valve

12. LV

13. aortic semi. v.

14. aorta

15. aortic art. (4)

## 16. Sys. Circ.

# Sequence

## **Diastole:**

- 1. E SA node: initiate heart beat**
- 2. E atrial muscles (gap junction): receive SA signal**  
**M atrial muscles contract (simultaneous)**

## **Systole:**

- 3. E AV node: conduct SA signal**
- 4. E Bundle of His: conduct AV signal**
- 5. E Bundle Branches: conduct Bundle signal**
- 6. E Purkinje fibers: conduct Branch signals**
- 7. E ventricular muscles: receive Purkinje fibers**  
**M ventricular muscles contract (simultaneous)**

**\*E = electrical event; M = muscle event**

# Neural Reg. Notes

- 1) **SA node**
  - several potential areas in atrial vicinity
  - prob. if in ventricular vicinity
- 2) **pacemaker**
  - artificial, battery powered SA node
  - initiate beat; hard to change tempo
- 3) **ventricular contraction**
  - > BP, more imp. than atrial contraction
- 4) **atrial contraction**
  - less critical; more imp. during exercise
- 5) **left side of heart**
  - more developed than right  
(thicker heart wall; larger chambers)
  - > systemic circuit

# EKG - Waves

**waves = electrical/neural events**

- **height = amount of neural excitation**  
 **$\alpha$  force prod. by cardiac muscle**
- **width = amount of time**

**3 waves:**

- 1) P wave: atrial depol. (.08 sec)\***  
-  **$\downarrow$  height  $\rightarrow$   $\downarrow$  PD  $\rightarrow$   $\downarrow$  force**
- 2) QRS wave: ventr. depol; atrial repol. (.08 sec)**  
-  **$\uparrow$  height  $\rightarrow$   $\uparrow$  PD  $\rightarrow$   $\uparrow$  force**
- 3) T wave: ventr. repol. (.16 sec)**

**\*based on HR=75 bpm, 1 cycle = .8 sec**

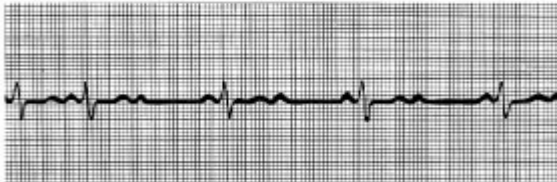
# Arrhythmias



(a)



(b)



(c)



(d)

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**normal**

**atrial fibrillation**

- no P waves
- slower HR

**heart block**

- #P wave > # QRS

**vent. fibrillation**

- erratic pattern
- heart attack

# Preload

**preload = stretch**

**-stretch in cardiac muscle  $\alpha$  force cardiac contraction**

**-more blood volume -> stronger systole**

**length-tension relationship:**

**↑ stretch in muscle fibers**

**→ ↑ # cross bridge attachments**

**→ ↑ force of contraction**

**heart: force of contraction matches**

**volume of blood to be ejected**

**- more volume -> stronger contraction**

# Frank-Starling Law

- $\uparrow$ preload thru exercise  $\rightarrow \uparrow$ SV
- $\downarrow$ HR  $\rightarrow \uparrow$ venous return  $\rightarrow \uparrow$  EDV  $\rightarrow \uparrow$ stretch  
 $\rightarrow \uparrow$ contraction force  $\rightarrow$  stronger systole  $\rightarrow \uparrow$ SV

## 1) untrained individual

- @ rest:  $\downarrow$ HR  $\rightarrow$  more time for ventr. fill-up  
 $\rightarrow \uparrow$  venous return  $\rightarrow \uparrow$ SV
- @ exercise:  $\uparrow$ HR  $\rightarrow$  less time for ventr. fill-up  
 $\rightarrow \downarrow$  venous return  $\rightarrow \downarrow$ SV

## 2) trained individual:

- @ rest & exercise,  $\downarrow$ HR  $\rightarrow \uparrow$ SV  
- combine both benefits



# Contractility

**contractility = cardiac tone**

- **healthy cardiac muscles** → ↑ **contractile strength**
- **independent of stretch and EDV**
- ↑ **tone** → ↑ **contraction force**

**Factors:**

**1) Sympathetic NS**

↑ **epinephrine/nor-epi.** → **Ca<sup>+</sup> influx**

→ ↑ **contractility** → ↑ **blood ejection** → ↓ **ESV** → ↑ **SV**

**2) Ca<sup>+</sup> ions**

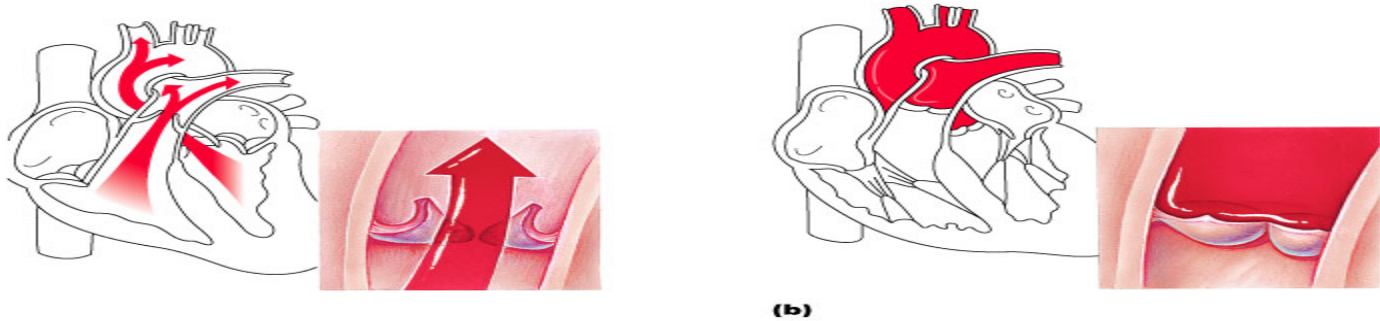
**3) glucagon**

**4) thyroxine**

**5) digitalis (heart stimulant)**

**\*Fill in the functions of 2 thru 5**

# Afterload



(a)  
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(b)

- afterload = back pressure to open heart valves**
- onset of systole, to eject arterial blood
  - aortic semilunar valve: 80 mm Hg  
( → afterload = 80+ mm Hg)
  - pul. semilunar valve: 8 mm Hg

**BP = opposing force to afterload**

**hypertension: 140/90 ( mm Hg)**

- afterload = 90+ mm Hg
- ↓ time heart valves open → ↓SV

# HR - ANS

**HR: compensates SV when ESV & EDV changes**

**ANS - reg HR**

**a) stress  $\rightarrow$   $\uparrow$ symp. NS  $\rightarrow$   $\uparrow$ epineph. release**

**$\rightarrow$  a)  $\uparrow$ HR**

**b)  $\downarrow$ EDV (less time for vent. fill-up)**

**b) grief  $\rightarrow$   $\uparrow$  parasymp. NS  $\rightarrow$   $\uparrow$  ACh release**

**$\rightarrow$  a)  $\downarrow$ HR**

**b)  $\uparrow$ EDV (more time for vent. fill-up)**

**note: sustained stress or grief brings new problems  
- compensations only temporary**

# HR - Chem.

## Chemicals reg HR:

### a) hormones

- 1) epinephrine (adrenal gland - ↑HR),
- 2) thyroxine (thyroid - sustained ↑HR)

### b) ions

- too high or low levels

→ irritable heart (spastic, little rest)

→ contraction prob., heart block, cardiac arrest

- ion disorders (look up symptoms in text)

- hypo/hyper-calcemia (calcium)

- hypo/hyper-natremia (sodium)

- hypo/hyper-kalemia (potassium)

# HR - Other Factors

- 1) **age : fetus (140 - 160 bpm), elderly < 60**
- 2) **gender: female adult (72 - 80), male adult (64 -72)**
- 3) **exercise:  $\uparrow$ symp. NS  $\rightarrow$   $\uparrow$ HR**
  - **untrained people:  $\uparrow$ HR  $\rightarrow$   $\downarrow$ SV**
  - **trained athletes  $\rightarrow$   $\downarrow$ HR  $\rightarrow$   $\uparrow$ SV**
- 4) **temperature**
  - **heat:  $\uparrow$ metabolism  $\rightarrow$   $\uparrow$ HR**
  - **cold: opposite effect**

# CHF

**congestive heart failure:**

**1) coronary atherosclerosis**

- **clog coronary vessels w/ fat**  
→ **heart muscle atrophy**

**2) ↑BP: → ↑afterload → heart muscle hypertrophy**

**3) myocardial infarcts**

- **cartilage replaces cardiac muscle cells**  
→ **↓contraction force**

**4) DCM - dilated cardiomyopathy**

- **ventricles stretch, flabby, deteriorate**  
→ **harder cardiac work w/ less results**

# Congestion

**heart = double pump**

**- each side can fail independently → congestion of circuit**

**a) pulmonary congestion:**

**right side works → blood to pulmonary circuit**

**left side fails → blood remains in pul. circuit**

**→ pulmonary edema (lungs engorged w/ blood)**

**→ suffocation (fast death)**

**b) peripheral congestion:**

**left side works → blood to systemic circuit**

**right side fails → blood remains in sys. circuit**

**→ systemic edema (organs & muscles engorged)**

**→ extremities swollen → poor circulation (slow death)**

# Resistance

**R = resistance to blood flow (F), friction**

**- 3 sources ( $\eta$ , l, r)**

**1)  $\eta$  (viscosity): fluid - thicker to thinner**

**- anemia:  $\downarrow\eta \rightarrow \downarrow R$**

**- higher altitudes: thicker blood  $\rightarrow \uparrow R$**

**2) l (BV length)**

**3) r (BV radius)**

**-  $\uparrow$  distance from heart**

**$\rightarrow \uparrow l$  &  $\downarrow r$  of blood vessels**

**$\rightarrow \uparrow R \rightarrow$  poorer circulation**



# Radius notes

a) type of blood flow dep. on location:

1) laminar flow: center, no walls, less friction ( $\downarrow R$ )

2) peripheral flow: near walls, more friction,  
impeded flow ( $\uparrow R$ )

3) turbulent flow: protruding objects, ( $\uparrow R$ )

b) radius of BV:

- large: mostly laminar flow ( $\downarrow R$ )

- small or objects: impeded, turb. flows ( $\uparrow R$ )

- 4th power:  $R = 1 / \text{radius}^4$  (exponent)

eg r doubled,  $R \rightarrow 1 / 16$  ( $\downarrow\downarrow R$ )

r halved,  $R \rightarrow 16$  ( $\uparrow\uparrow R$ )

# BP

**"BP" = arterial blood pressure (systemic)**

**2 factors:**

- a) stretch - arterial wall elasticity**
- b) volume -  $\uparrow$  vol  $\rightarrow$   $\uparrow$  BP**

**2 types of BP:**

- a) systolic pressure (adult 120 mm Hg)**
  - highest aortic pressure**
  - aortic semilunar valves open**
- b) diastolic pressure (adult 80 mm Hg)**
  - lowest aortic pressure**
  - aortic semilunar valves closed**