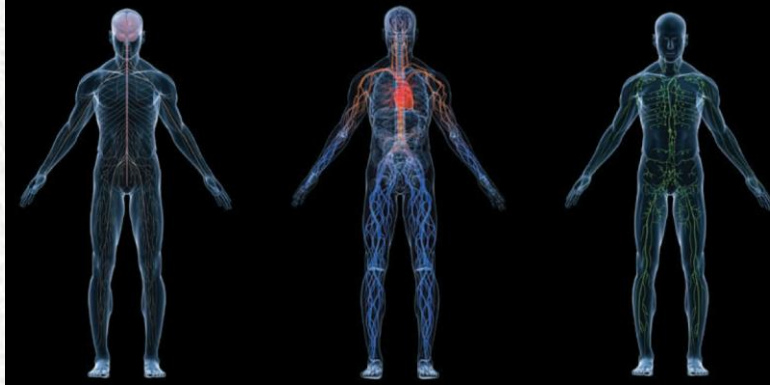


UNIT III

GUYTON AND HALL TEXTBOOK OF **MEDICAL PHYSIOLOGY** THIRTEENTH EDITION

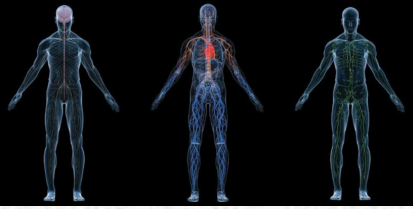
Chapter 9:



Cardiac Muscle; The Heart as a Pump and Function of the Heart Valves

Slides by R. Davis Manning, Jr., PhD

Presented by: Dr. Avnish Dave



The Heart

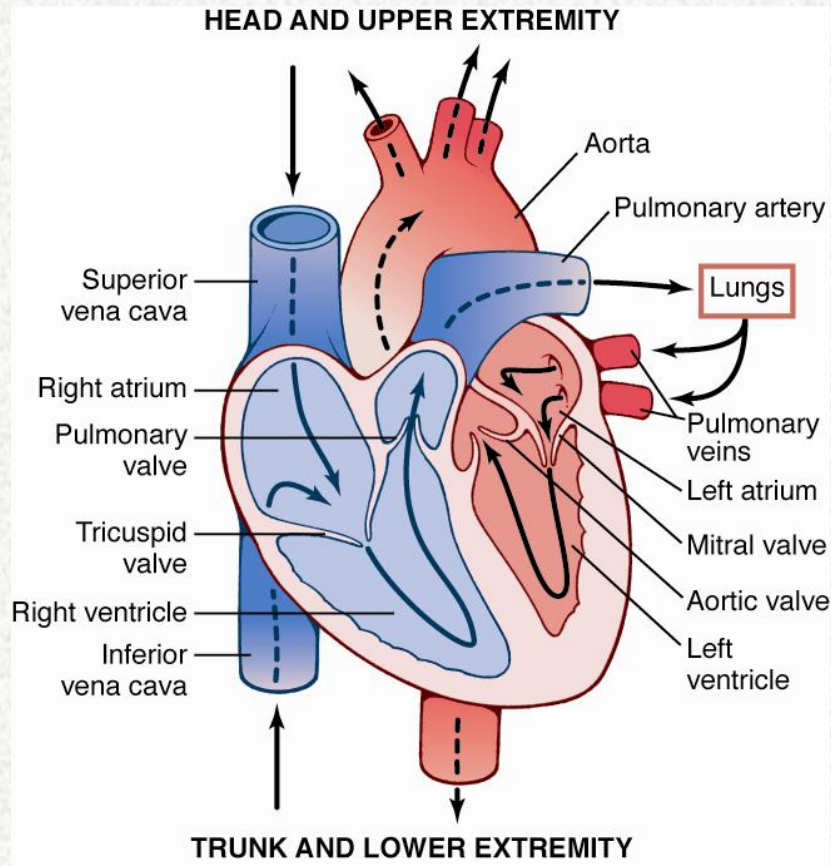
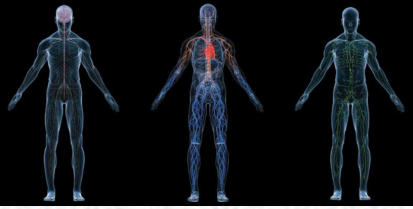


Figure. 9-1

- Atrial syncytium
- Ventricular syncytium
- Fibrous insulator exists between atrium and ventricle (why?)



Cardiac Muscle

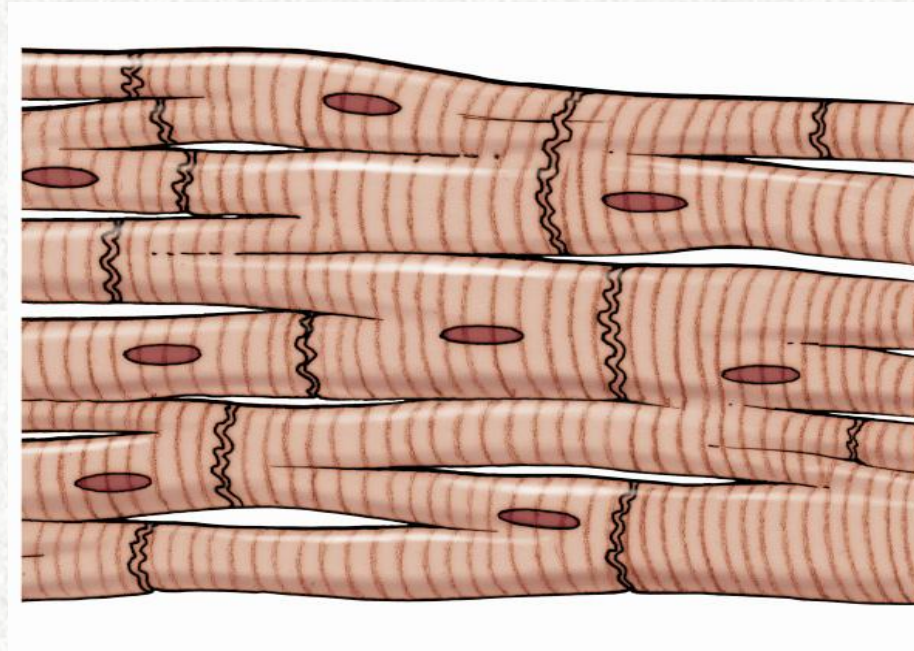
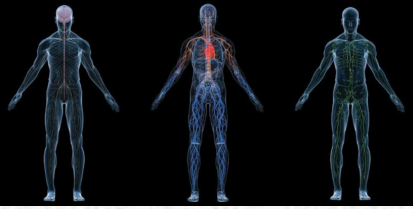


Figure. 9-2

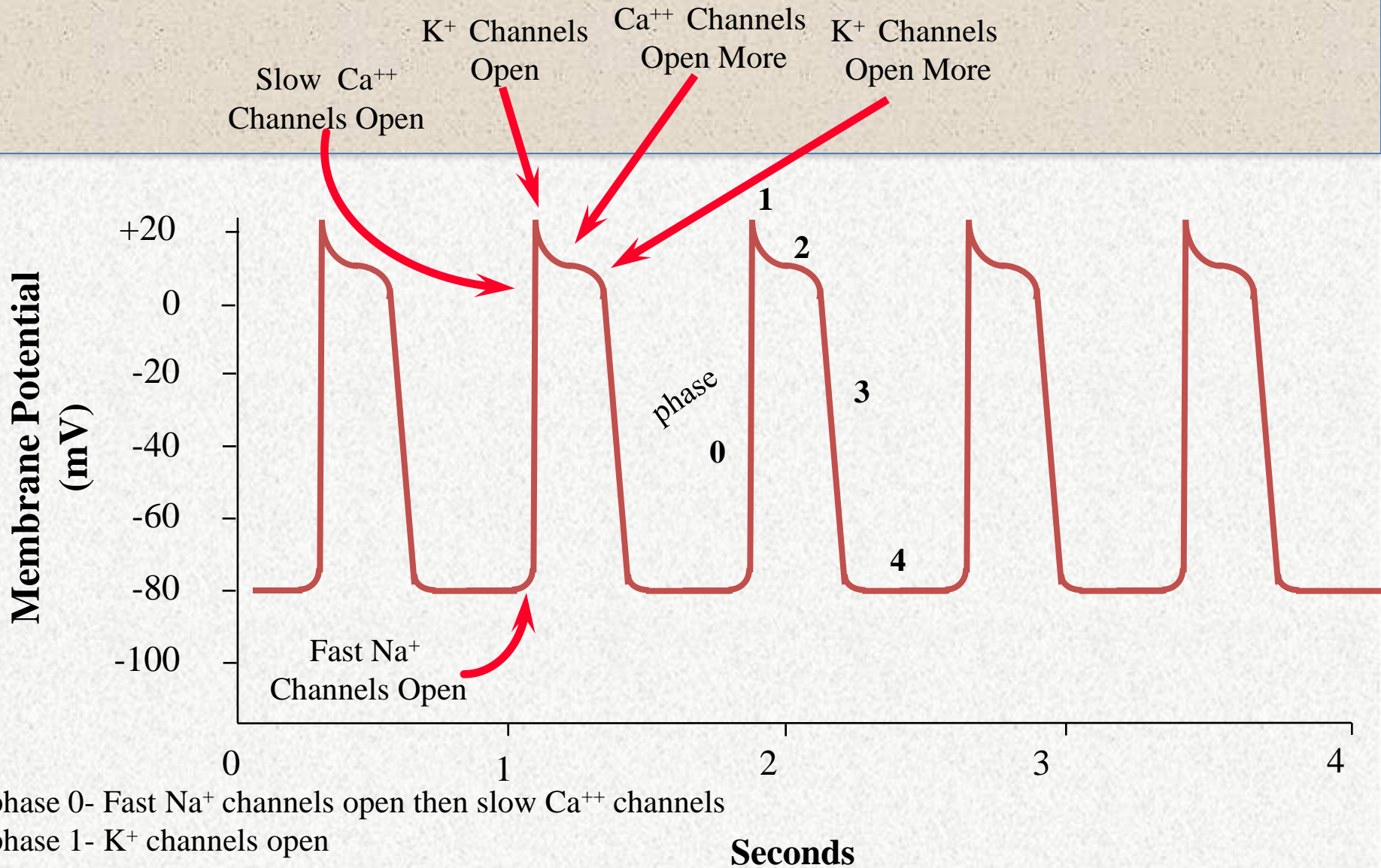
- Has actin and myosin filaments
- Has low resistance intercalated disks (1/400 the resistance of cell membrane)



Action Potentials

- Resting membrane potential of cardiac muscle is -85 to -95 millivolts
- Action potential is 105 millivolts
- Plateau lasts ~0.2-0.3 sec in ventricular muscle (much longer than skeletal muscle)

Ventricular Muscle Action Potential



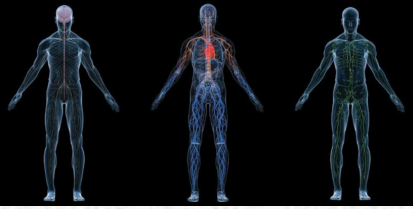
phase 0- Fast Na^+ channels open then slow Ca^{++} channels

phase 1- K^+ channels open

phase 2- Ca^{++} channels open more

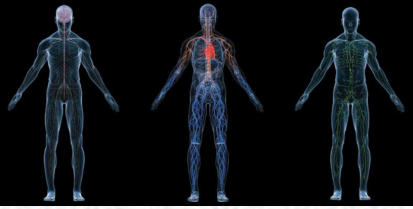
phase 3- K^+ channels open more

phase 4- Resting membrane potential



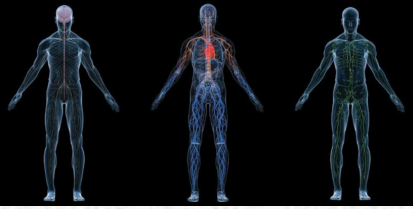
Refractory Period

- During this time cardiac muscle cannot be re-excited
- Lasts 0.25-0.30 sec in ventricles
- Lasts 0.15 sec in atria
(Does this help atria control rate?)



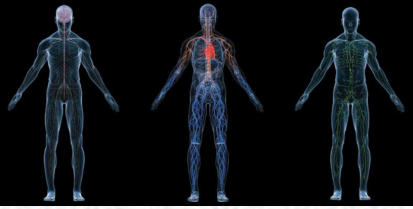
Results of Action Potential

- Ca^{++} release from T- tubules, which are large, are a very important source of Ca^{++} . T- tubule Ca^{++} depends strongly on extracellular Ca^{++} concentration. Mucopolysaccharides bind Ca^{++} .
- Ca^{++} release from sarcoplasmic reticulum (after stimulation of ryanodine receptors)



Cardiac Cycle

- Systole – ventricular muscle stimulated by action potential and contracting
- Diastole – ventricular muscle reestablishing $\text{Na}^+/\text{K}^+/\text{Ca}^{++}$ gradient and is relaxing
- EKG - P-atrial wave
QRS - Ventricular wave
T - ventricular repolarization



Cardiac Cycle (cont'd)

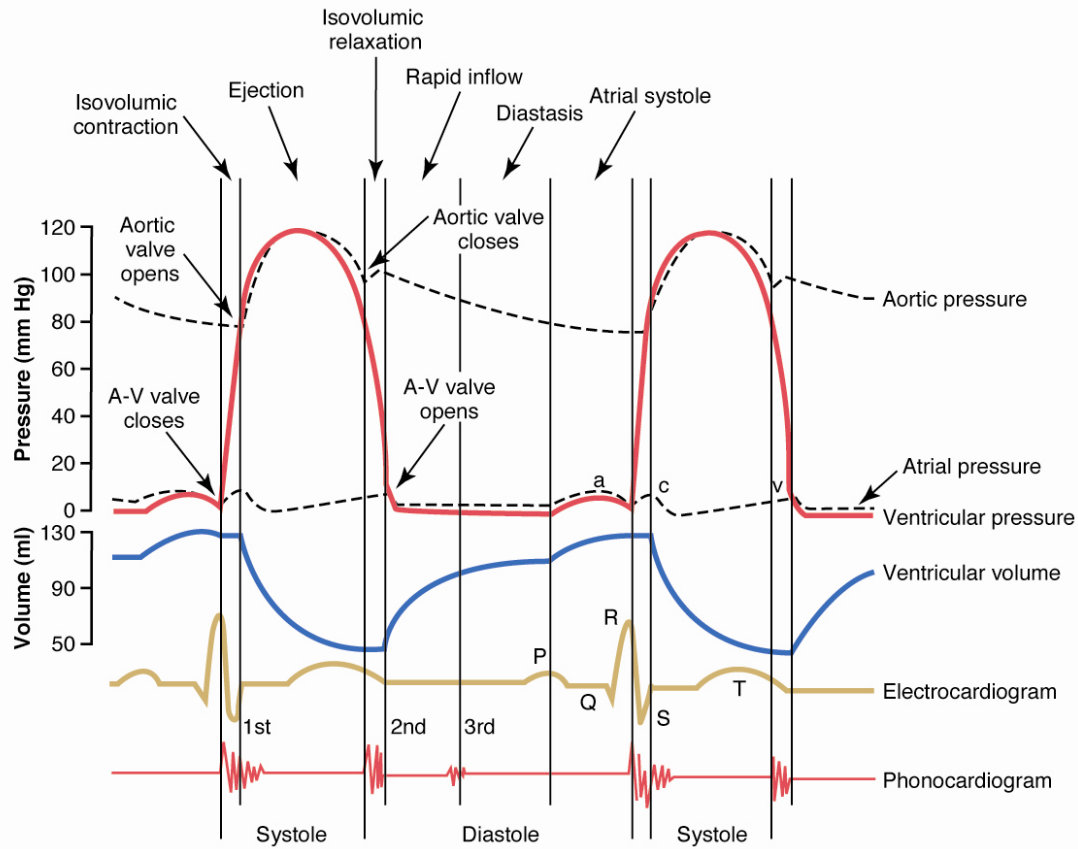
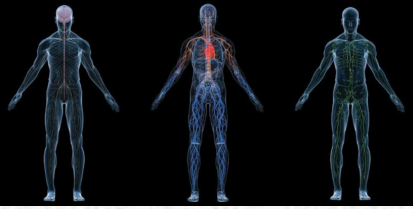
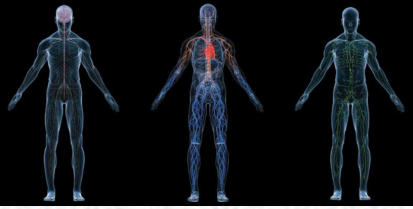


Figure. 9-6



Cardiac Cycle (cont'd)

- Atrial press wave -
 - a-wave - atrial contraction
 - c-wave - ventricular contraction
(A-V valves bulge)
 - v-wave - flow of blood into atria



Ventricular Pressure and Volume Curves

- Diastole

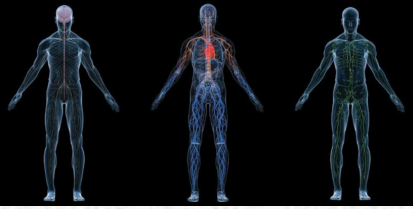
Isovolumic relaxation

A-V valves open

Rapid inflow

Diastasis - slow flow into ventricle

Atrial systole - extra blood in and this just follows P wave. Accounts for 25% of filling



Cardiac Cycle (cont'd)

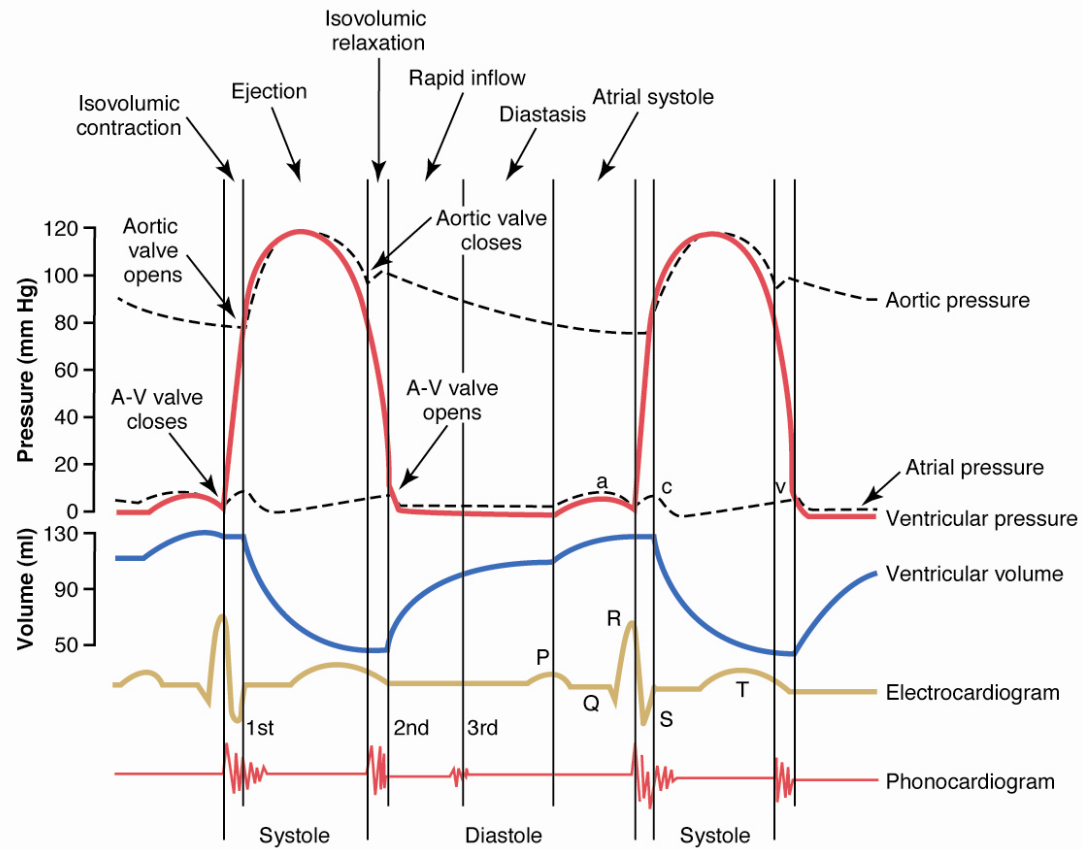
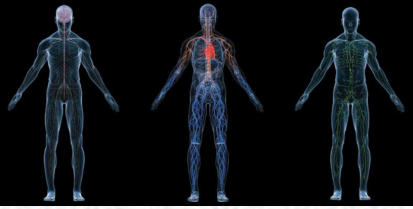


Figure. 9-6



Ventricular Pressure and Volume Curves (cont'd)

- Systole

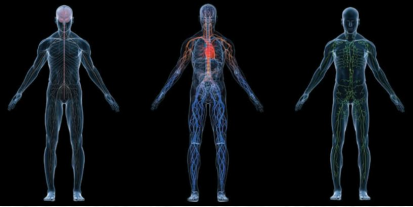
Isovolumic contraction

A-V valves close (ventricular press > atrial press)

Aortic valve opens

Ejection phase

Aortic valve closes



Cardiac Cycle (cont'd)

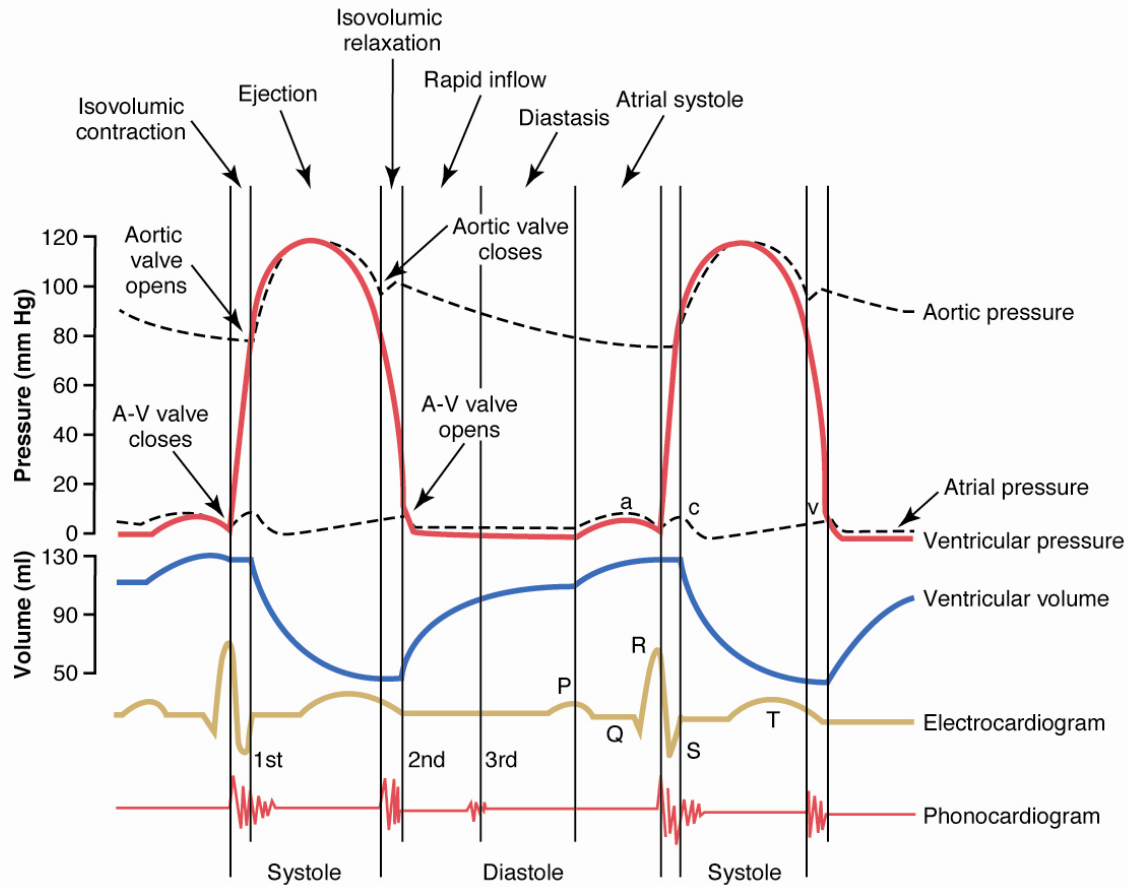
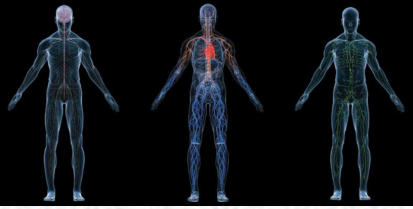
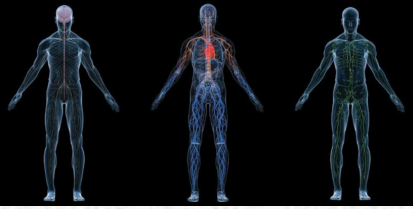


Figure. 9-6



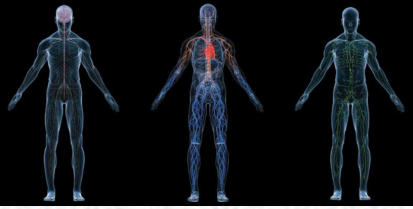
Ventricular Pressure and Volume Curves (cont'd)

- During the latter part of the ejection phase how can blood still leave the ventricle if pressure is higher in the aorta?
- Total energy of blood = $P + mV^2/2$ = pressure + kinetic energy
- Total energy of blood leaving ventricle is greater than in aorta.



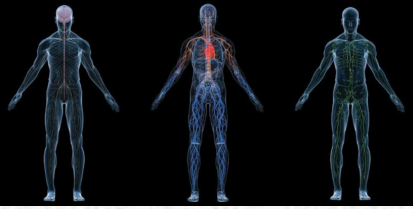
Ejection Fraction

- End diastolic volume = 120 ml
- End systolic volume = 50 ml
- Ejection volume (stroke volume) = 70 ml
- Ejection fraction = $70 \text{ ml} / 120 \text{ ml} = 58\%$
(normally 60%)
- If heart rate (HR) is 70 beats/minute, what is cardiac output?
- Cardiac output = HR * stroke volume
= 70/min. * 70 ml
= 4900 ml/min.



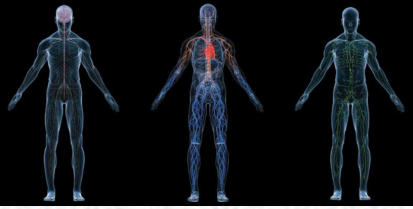
Ejection Fraction (cont'd)

- If $HR = 100$, end diastolic volume = 180 ml, end systolic vol. = 20 ml, what is cardiac output?
- $C.O. = 100/\text{min.} * 160 \text{ ml} = 16,000 \text{ ml/min}$



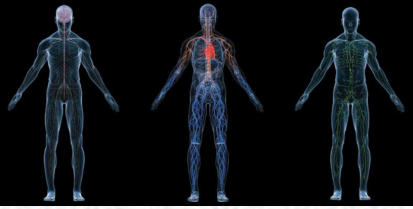
Aortic Pressure Curve

- Aortic pressure starts increasing during systole after the aortic valve opens.
- Aortic pressure decreases toward the end of the ejection phase.
- After the aortic valve closes an incisura occurs because of sudden cessation of back-flow toward left ventricle.
- Aortic pressure decreases slowly during diastole because of the elasticity of the aorta plus blood flow to the periphery.

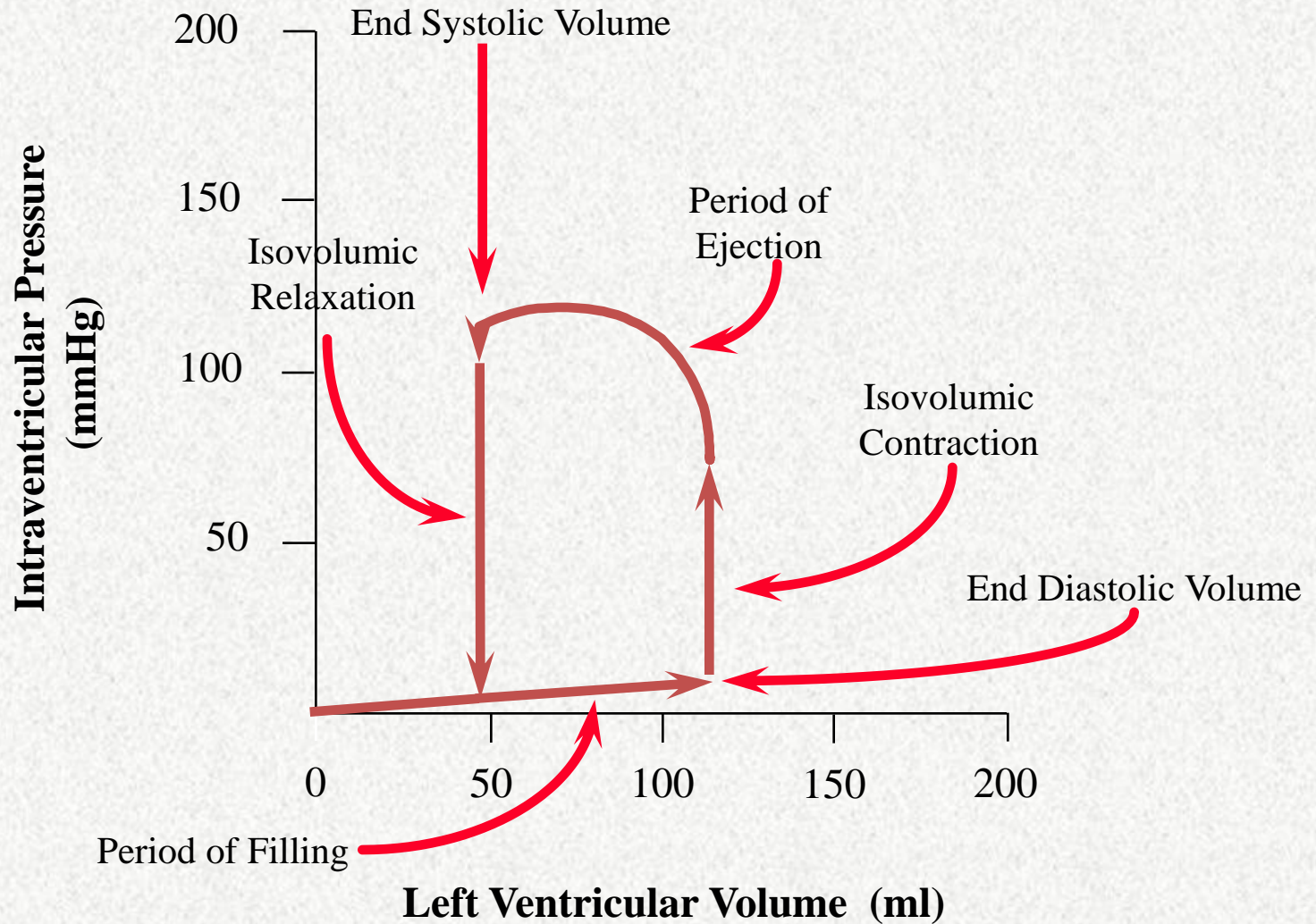


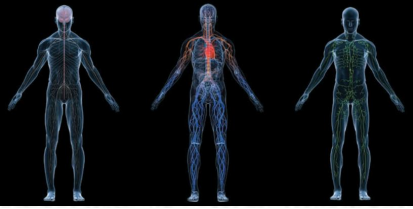
Valvular Function

- To prevent back-flow
- Chordae tendineae are attached to A-V valves
- Papillary muscle, attached to chordae tendineae, contract during systole and help prevent back-flow.
- Because of smaller opening, velocity through aortic and pulmonary valves exceed that through the A-V valves.

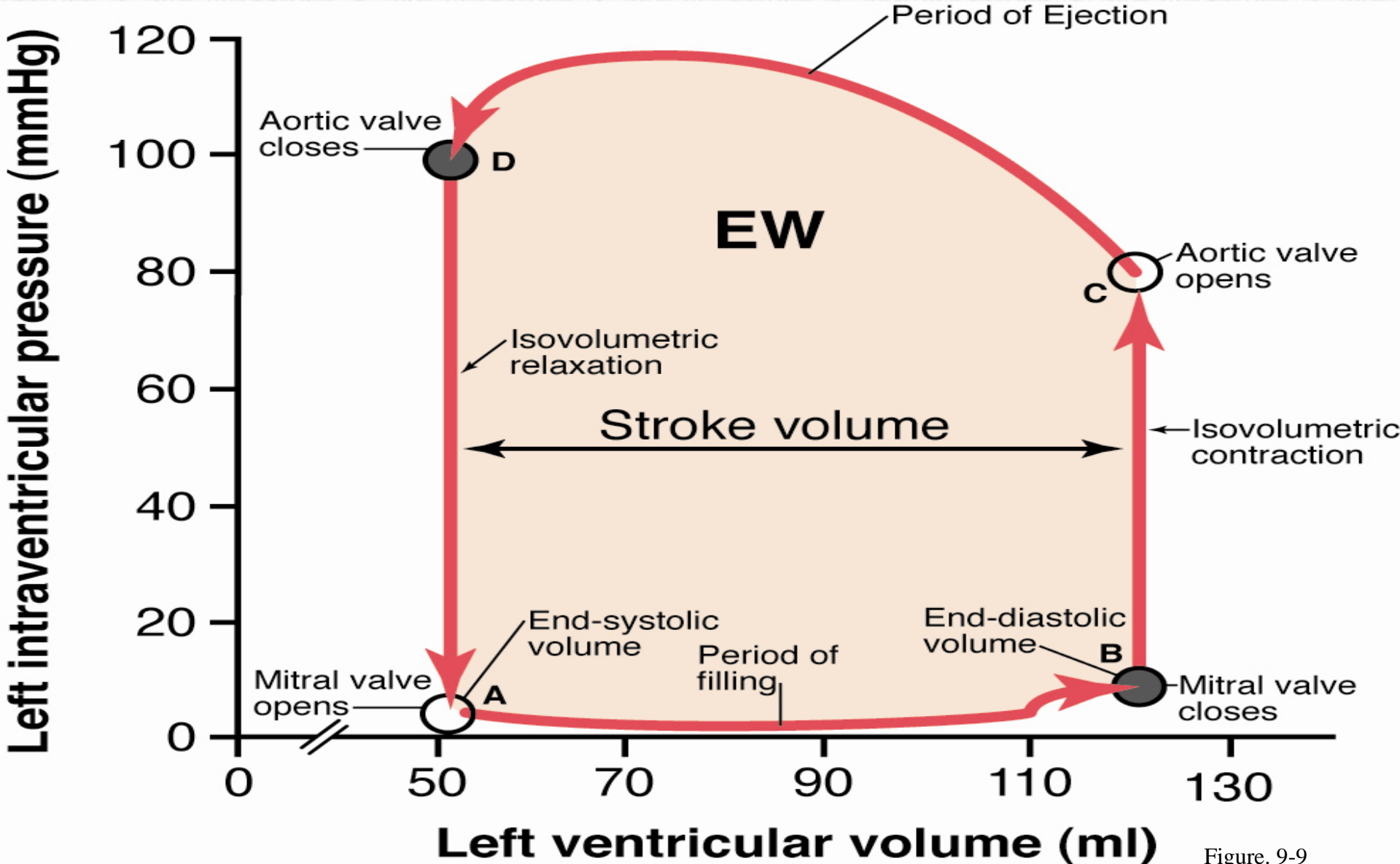


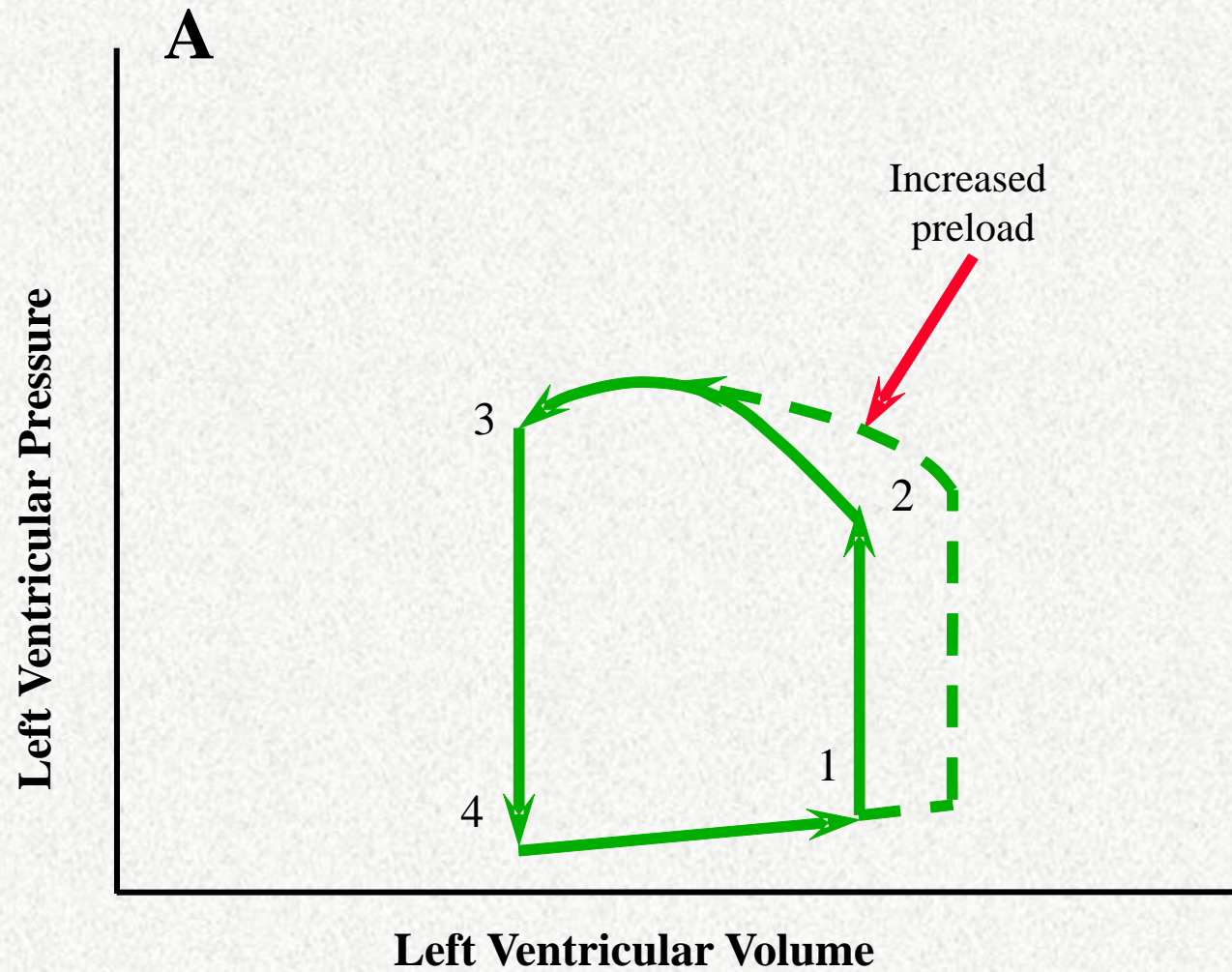
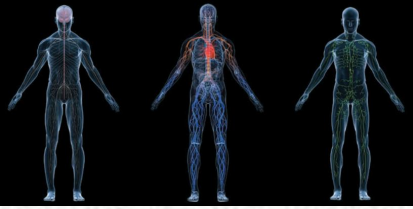
Work Output of the Heart

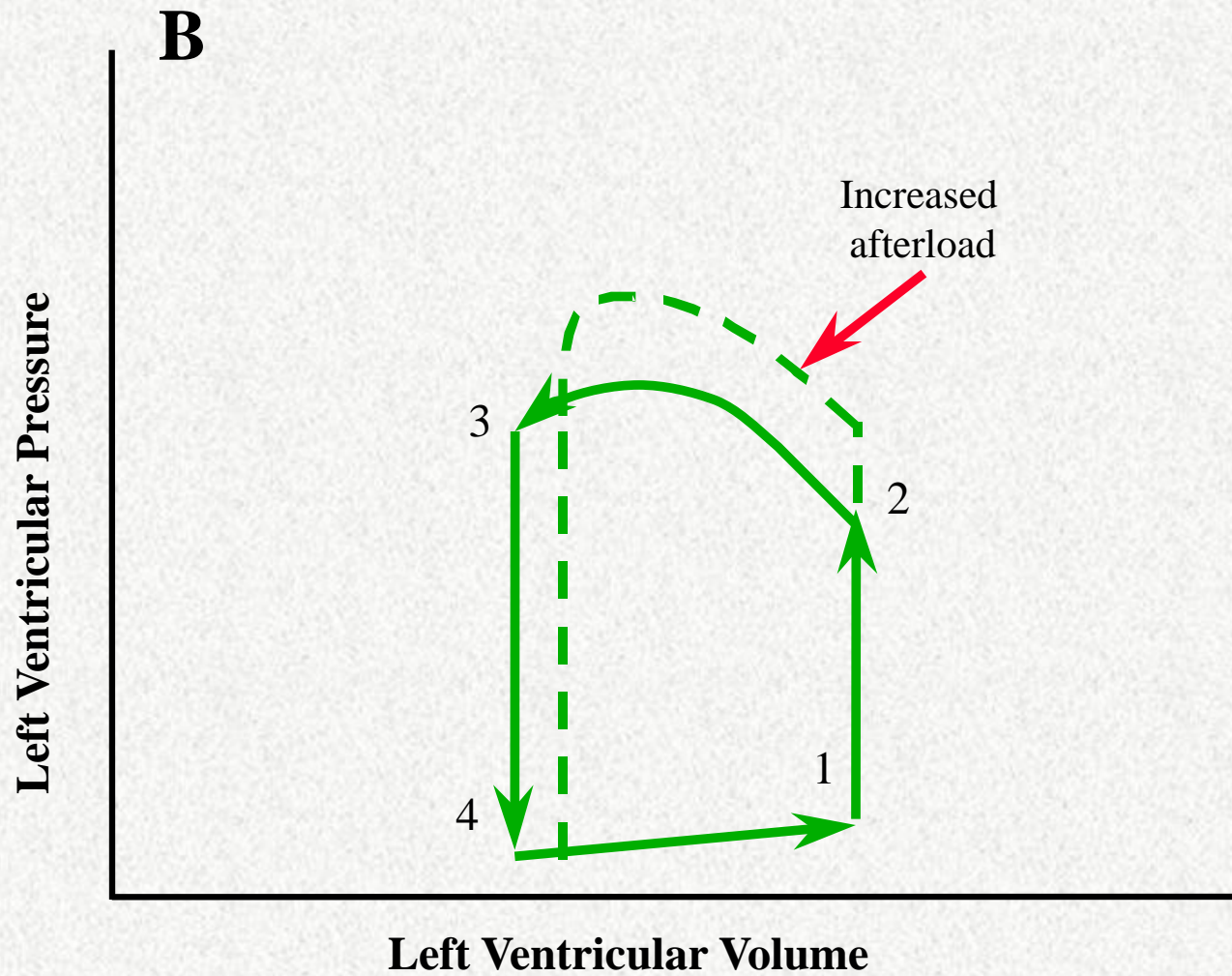
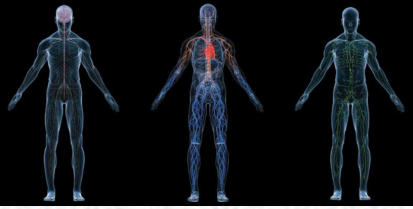


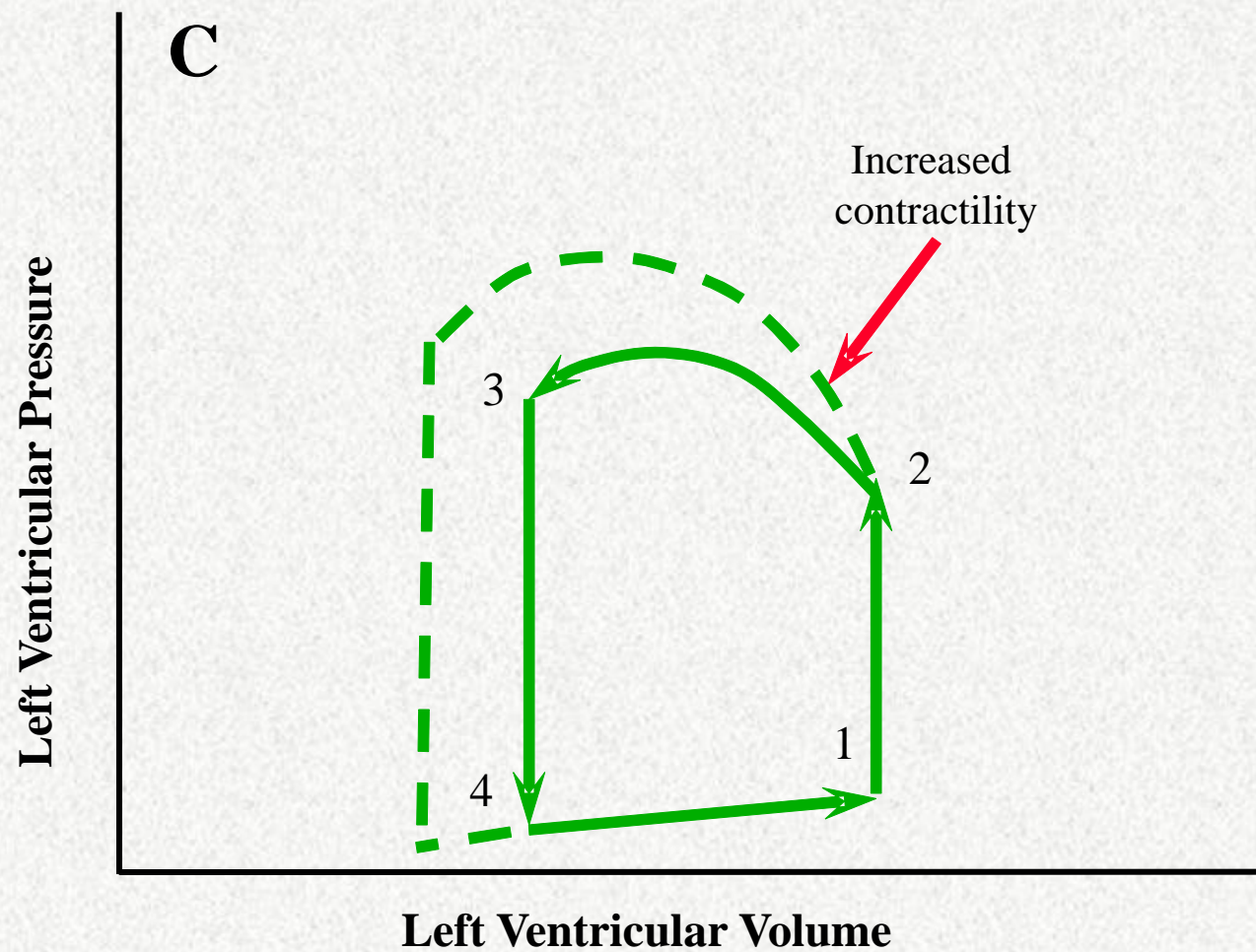
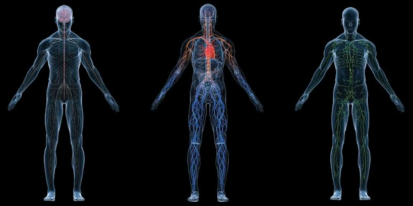


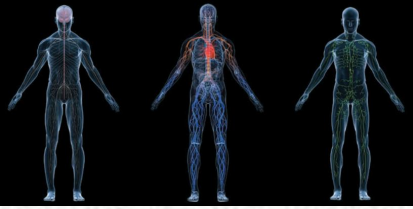
Work Output of the Heart





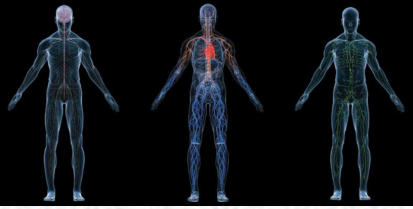






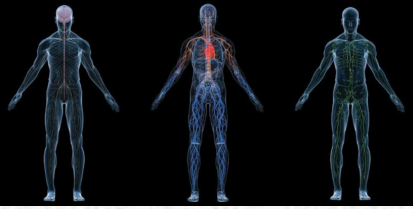
Valvular Function (cont'd)

- Most work is external work or pressure-volume work.
- A small amount of work is required to impart kinetic energy to the heart ($1/2 mV^2$).
- What is stroke-volume in above figure?
- External work is area of P-V curve.
- Work output is affected by “preload” (end-diastolic pressure) and “afterload” (aortic pressure).

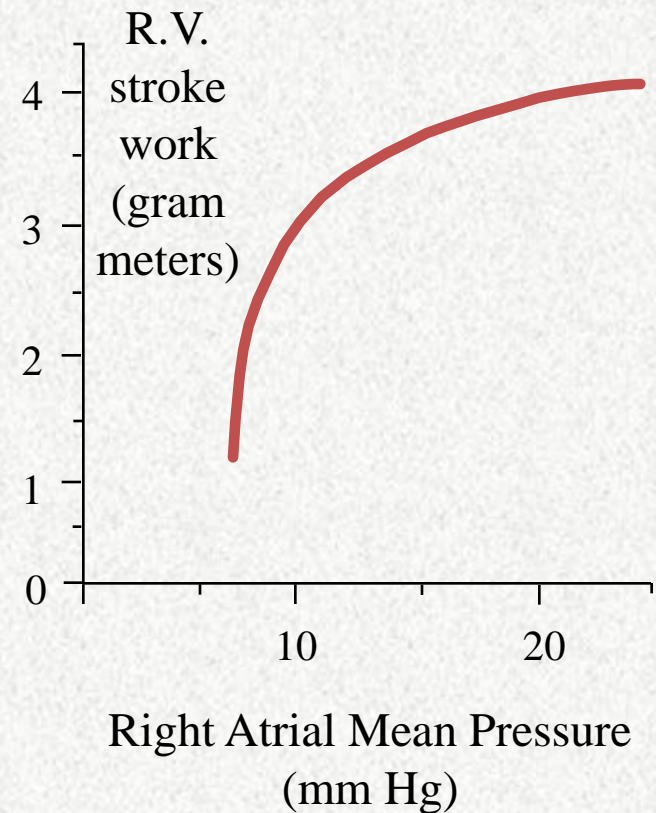
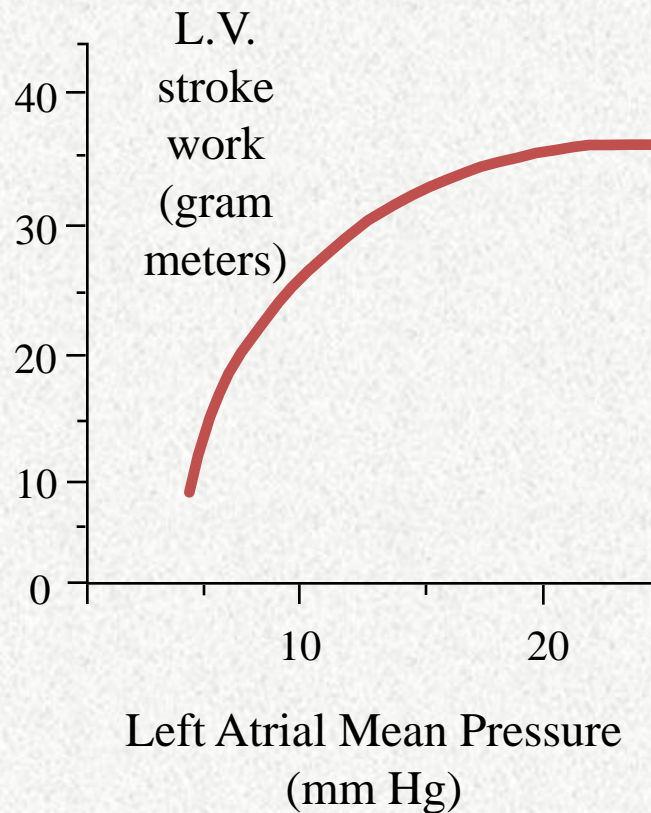


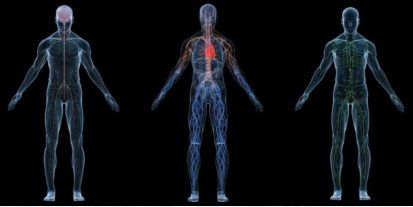
Frank-Starling Mechanism

- Within physiological limits the heart pumps all the blood that comes to it without excessive damming in the veins.
- Extra stretch on cardiac myocytes makes actin and myosin filaments interdigitate to a more optimal degree for force generation.



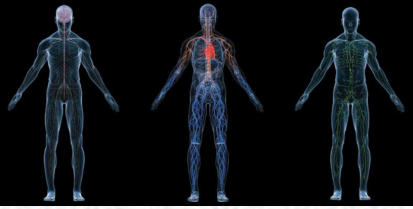
Ventricular Stroke Work Output



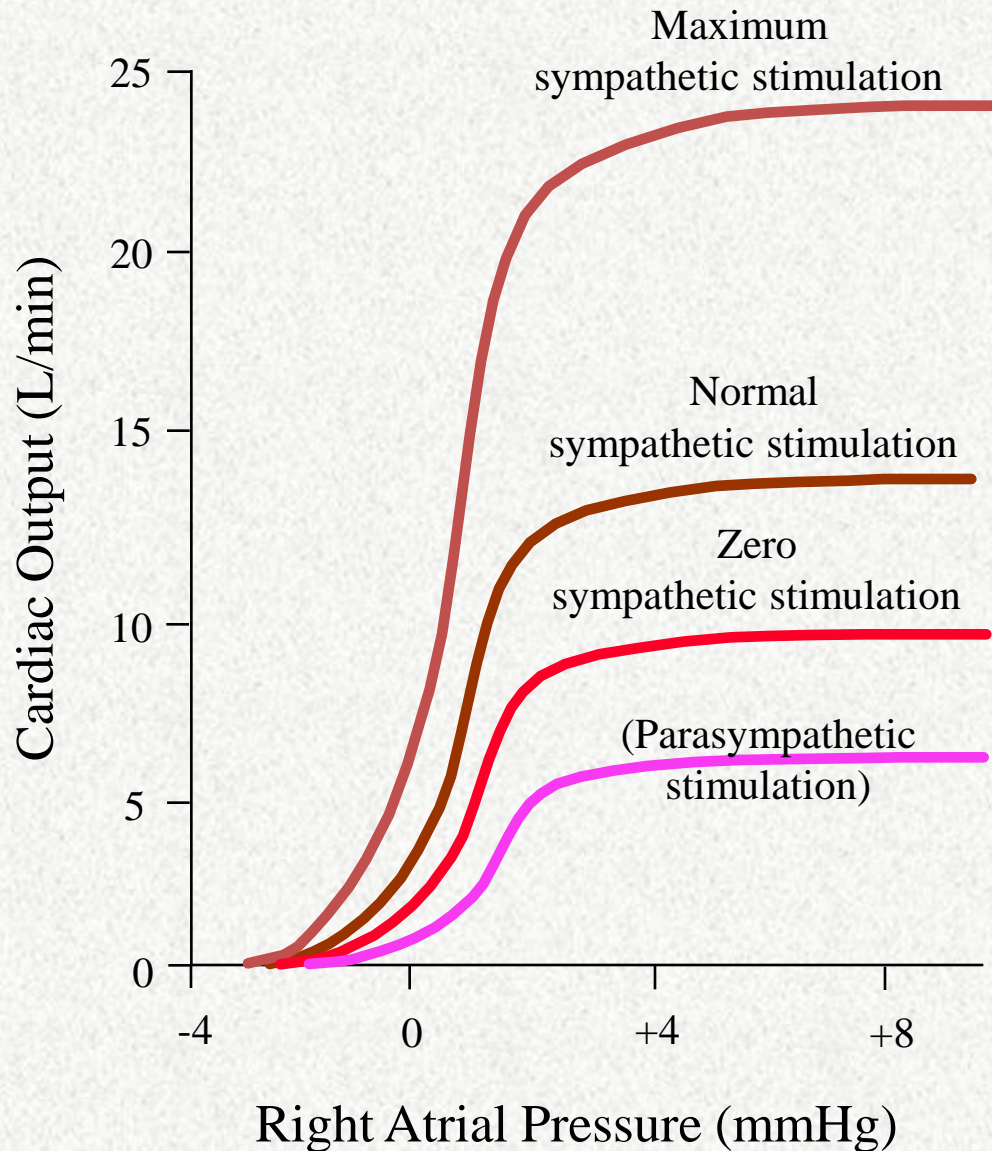


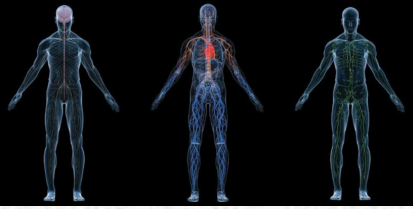
Autonomic Effects on Heart

- Sympathetic stimulation causes increased HR + increased contractility with HR = 180-200 and C.O. = 15-20 L/min.
- Parasympathetic stimulation decreases HR markedly and decreases cardiac contractility slightly. Vagal fibers go mainly to atria.
- Fast heart rate (tachycardia) can decrease C.O. because there is not enough time for heart to fill during diastole.



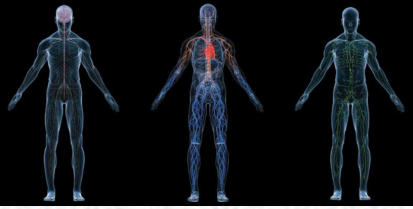
Effect of Sympathetic and Parasympathetic Stimulation on Cardiac Output





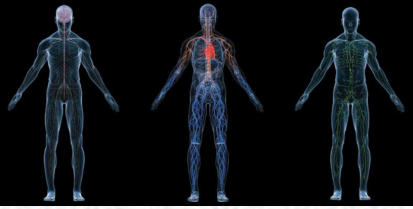
Cardiac Contractility

- Best is to measure the C.O. curve, but this is nearly impossible in humans.
- dP/dt is not an accurate measure because this increases with increasing preload and afterload.
- $(dP/dt)/P_{\text{ventricle}}$ is better. $P_{\text{ventricle}}$ is instantaneous ventricular pressure.
- Excess K^+ decreases contractility (heart dilated)
- Excess Ca^{++} causes spastic contraction, and low Ca^{++} causes cardiac dilation.



Chapter 9 Objectives

1. Understand what transcellular movement of ions causes the cardiac action potential.
2. Learn the definition of a refractory period.
3. Learn the role of calcium ions in the excitation-contraction coupling in cardiac muscle.
4. Understand the relationships between the electrocardiogram, atrial pressure, ventricular pressure, aortic pressure, ventricular volume and the heart sounds.



Chapter 9 Objectives

5. Learn the phases of the ventricular pressure volume curve and how to calculate cardiac work from this curve.
6. Learn how to calculate ejection fraction and cardiac output from end diastolic volume, end systolic volume, and heart rate.
7. Learn the function of the cardiac valves.
8. Understand the Frank-Starling mechanism and the effect of the autonomic nervous system on cardiac contractility.