

Human Phys PCB4701

# **Heart 1**

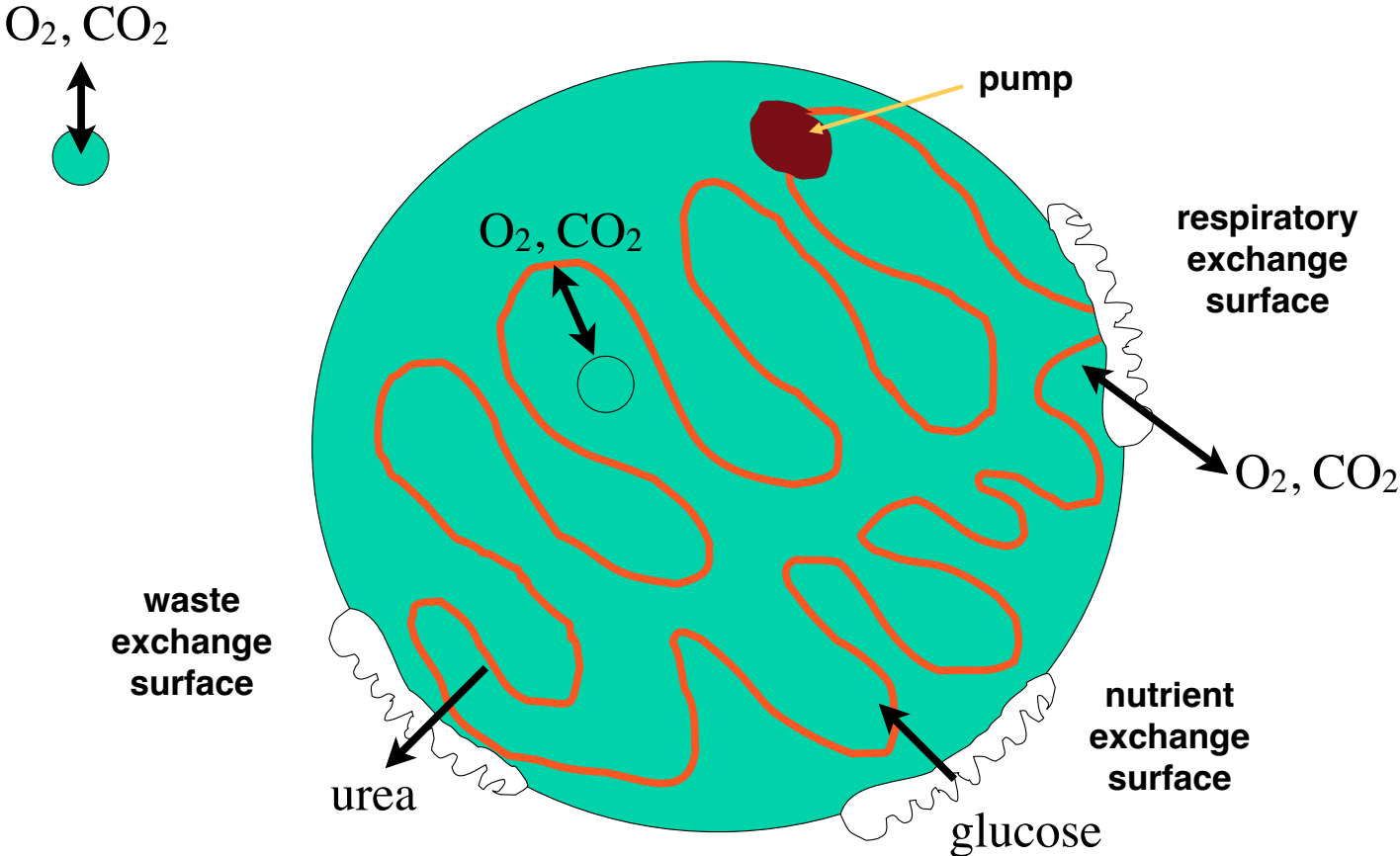
## **Fox Chapter 13 part 1 (Chapter 12.6 Cardiac Muscle)**

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**Anatomy of The Heart**  
**Cardiac Cycle**  
**Cardiac Action Potential and ECG**

# Circulatory System: Active Pumping

to transport gases from respiratory surface to tissues



## **Atrium**

receives incoming blood, passes it to ventricle

## **Ventricle**

more muscular pump sending blood to a separate circulation (either pulmonary circulation (lungs) or systemic circulation).

## **Arteries (arterial blood)**

vessels carrying blood from heart towards the capillaries.

Thick muscular walls to keep pressure up.

High in oxygen (except for pulmonary arteries).

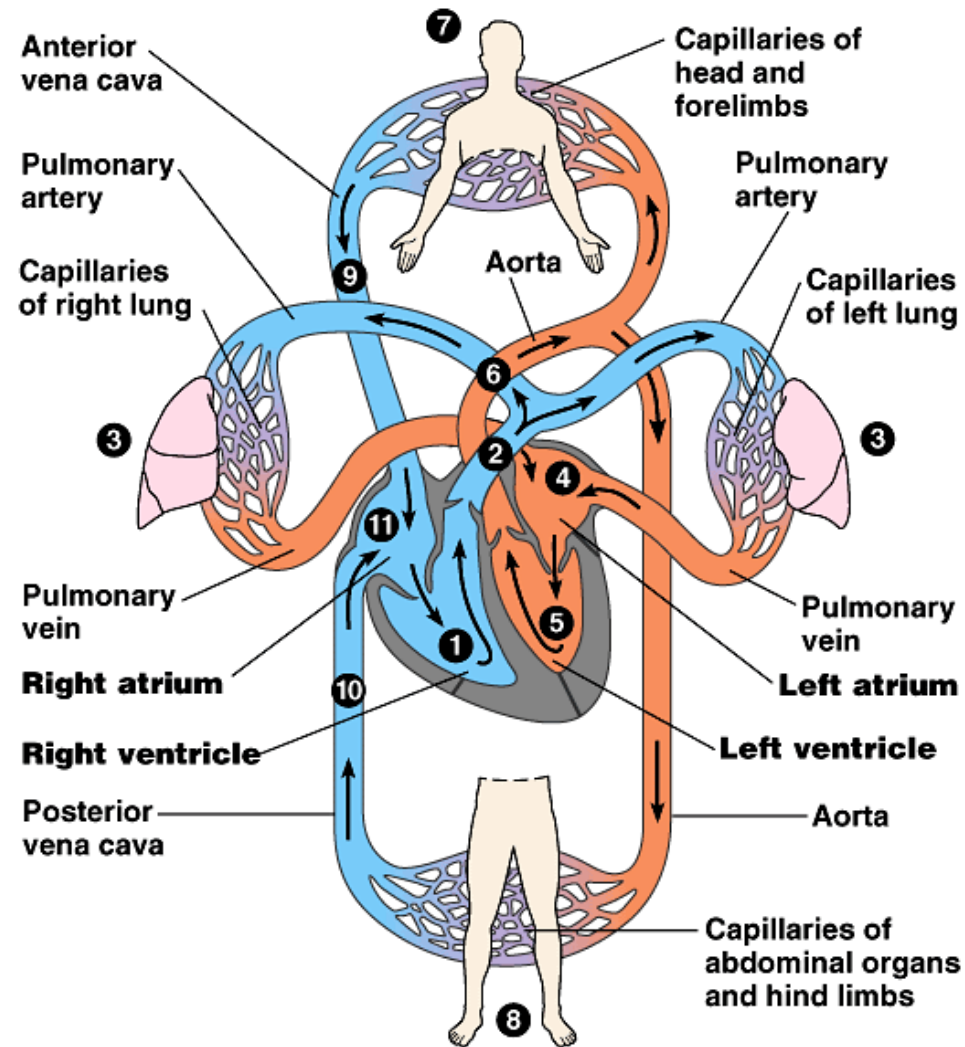
## **Veins (venous blood)**

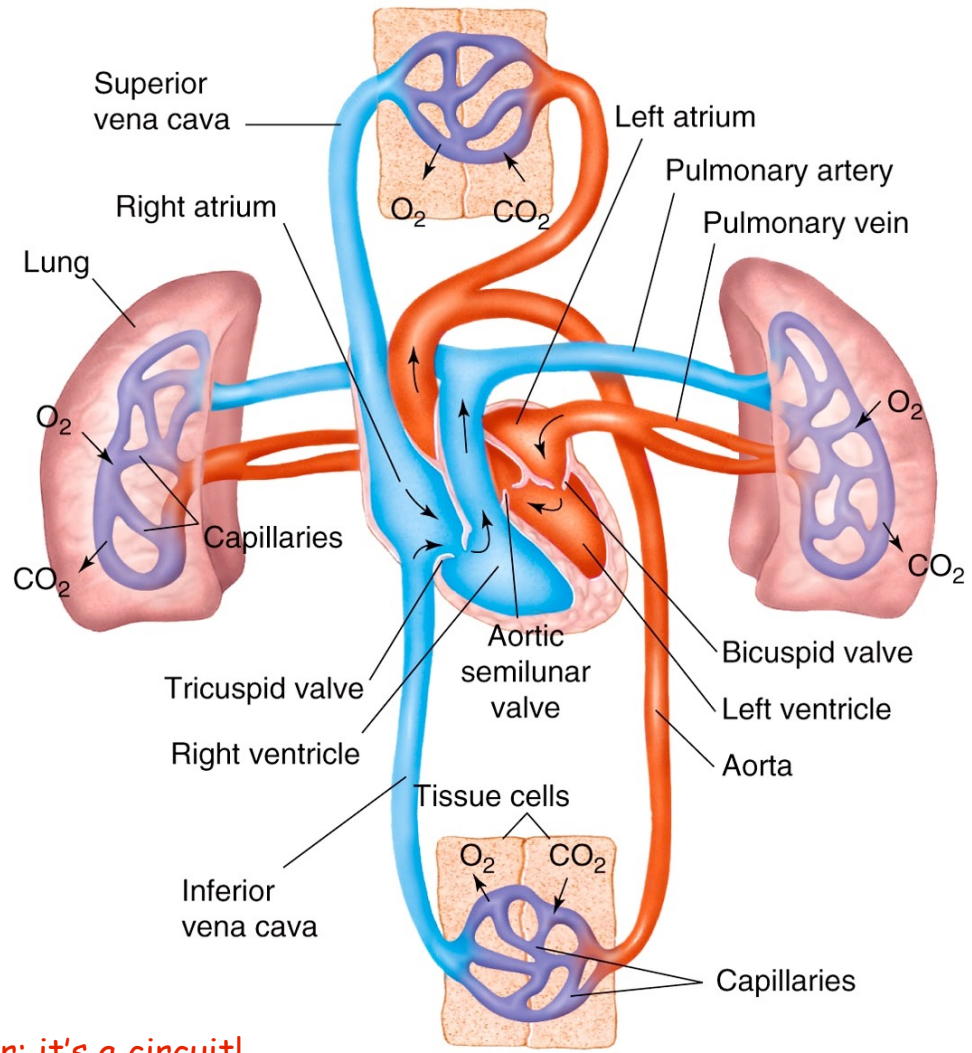
vessels carrying blood from capillaries back to heart. Very thin flabby walls with low pressure, but have one-way valves to prevent blood from backing up. Low in oxygen (except for pulmonary veins).

## **Capillaries**

very small vessels (one blood cell wide) that perfuse all the tissues.

Figure 42.4 The mammalian cardiovascular system: an overview





remember: it's a circuit!

Figure 13.10

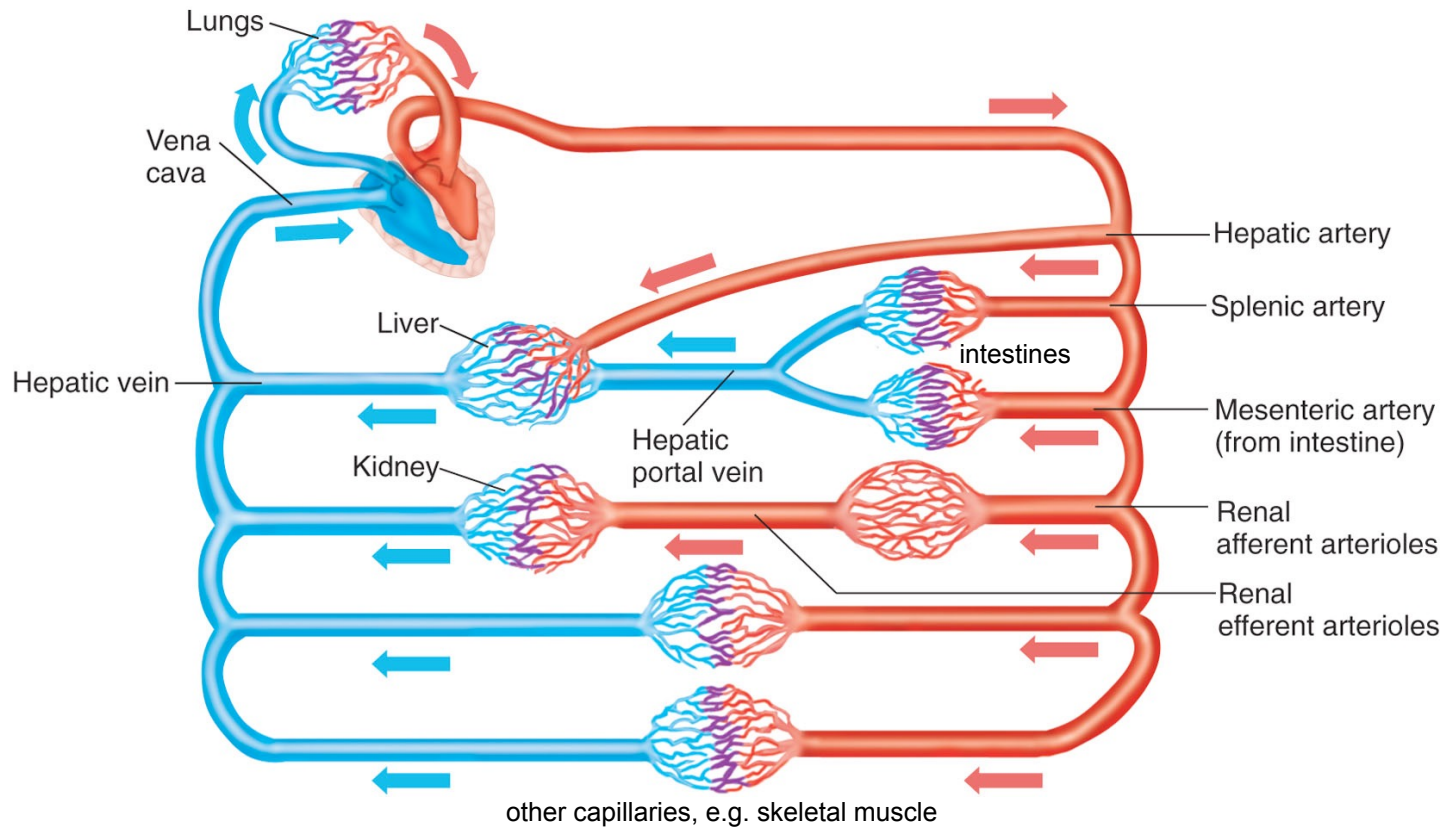


Figure 14.17

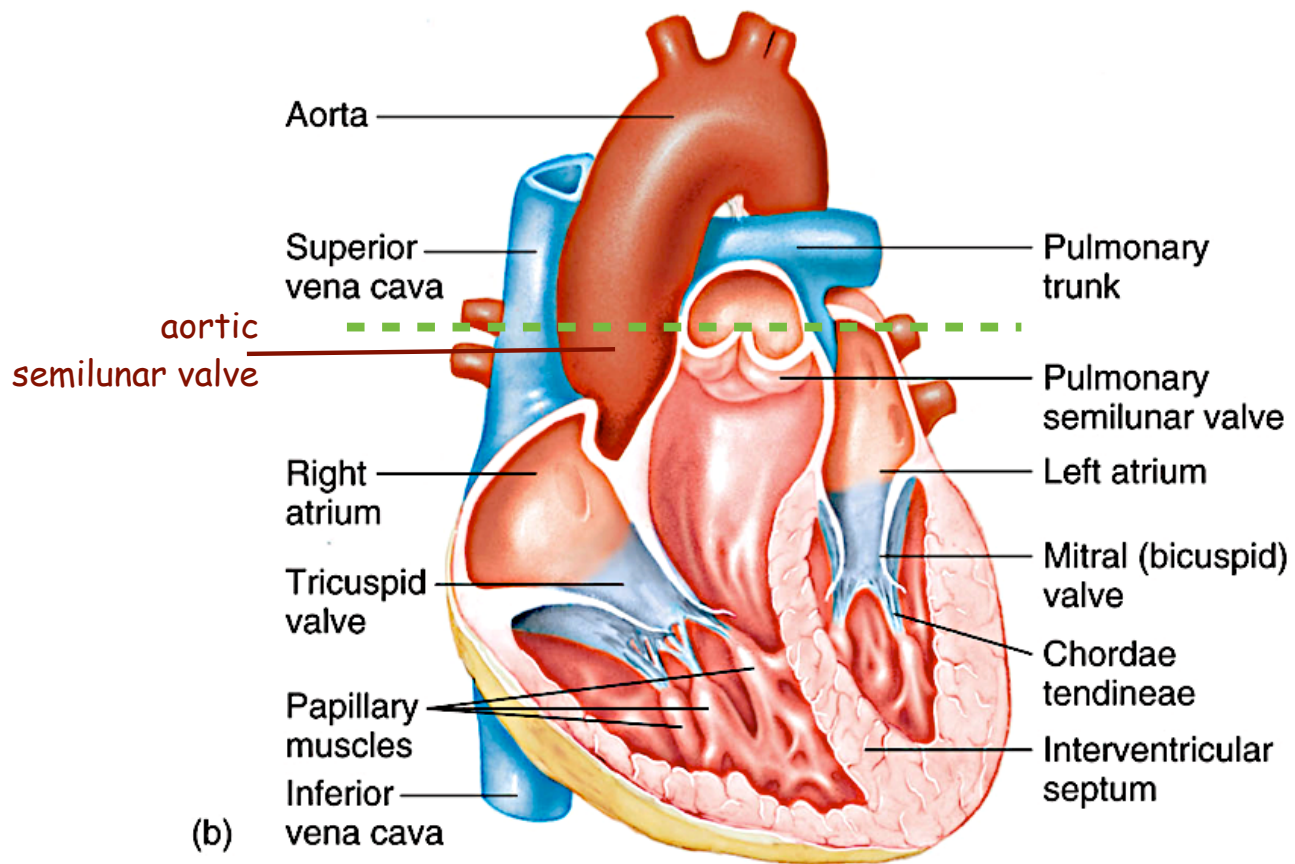
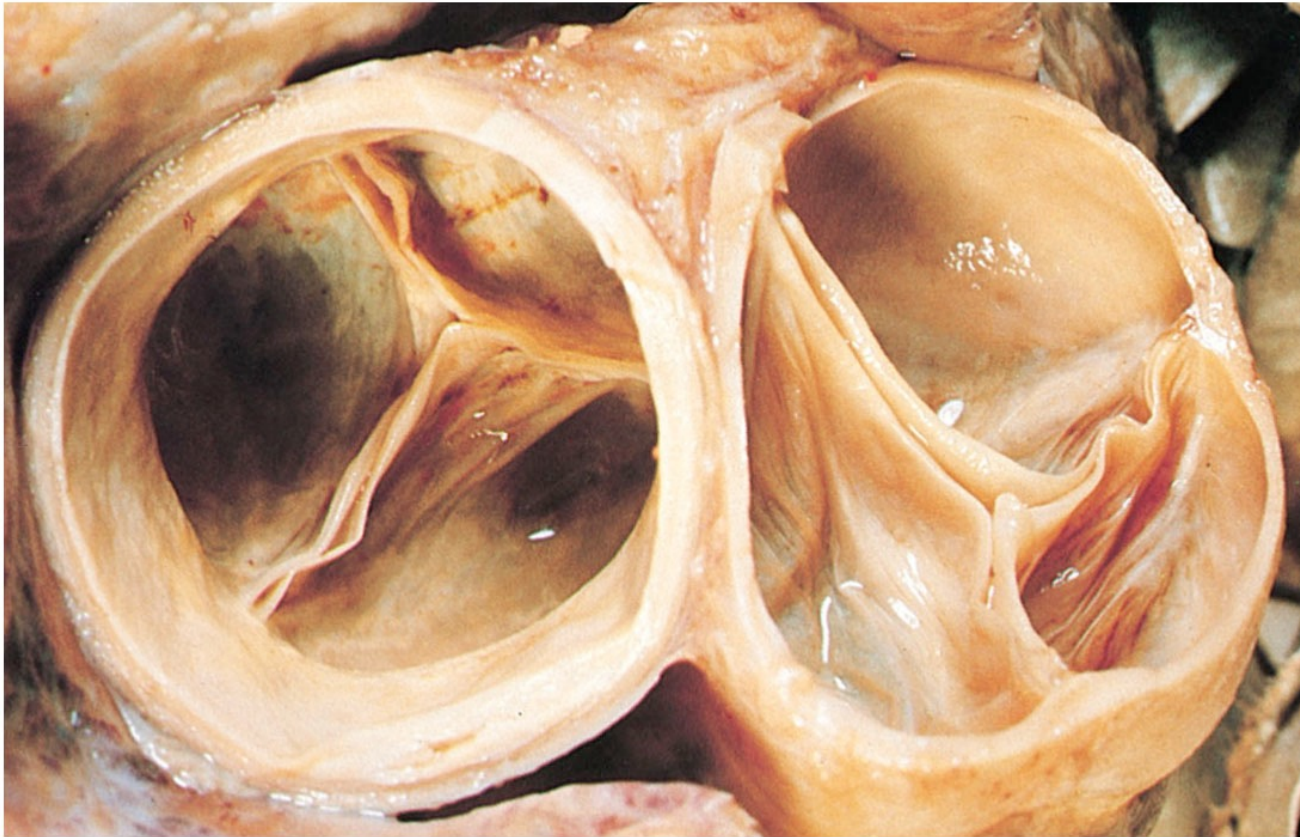


Figure 13.11



aortic semilunar valves

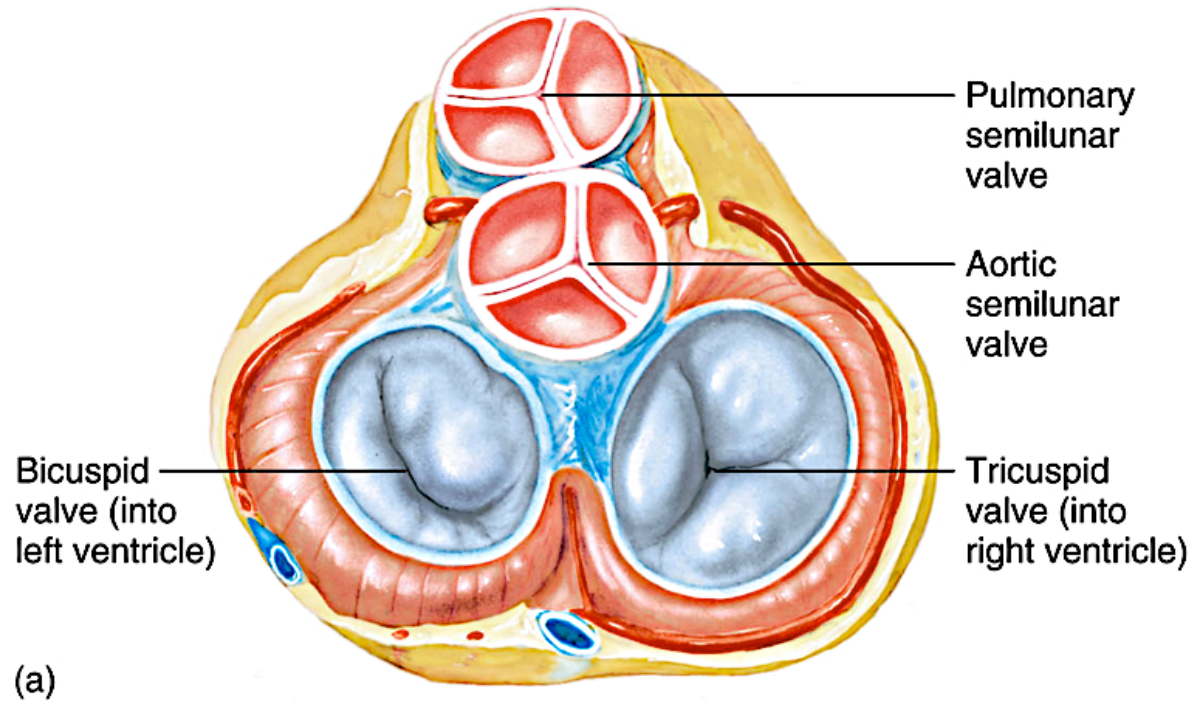
pulmonary semilunar valves



Igaku-Shoin, Ltd., Tokyo, Japan

Figure 13.12

“dub” = closing of semilunar valves = S2



“lub” = closing of AV valves = S1

Figure 13.11

## **The Cardiac Cycle**

### **Diastole**

chambers are relaxed, blood can flow in

### **Atrial Systole**

atria contract, pushing blood into ventricles

### **Ventricular Systole**

ventricles contract with high pressure, pushing blood into the lungs and systemic circulation

### **Diastolic pressure (bottom number)**

arterial pressure when ventricle is relaxed

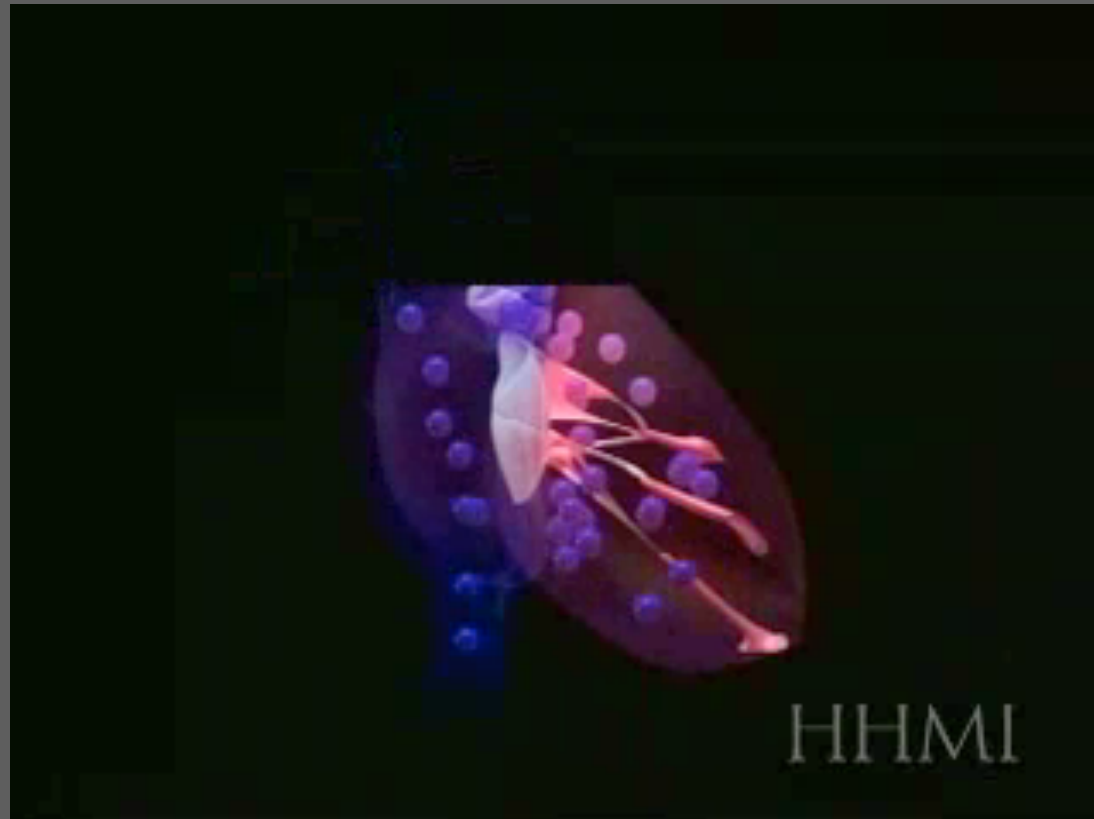
### **Systolic pressure (top number)**

arterial pressure when ventricle contracts and pumps

<http://www.hhmi.org/biointeractive/cardiovascular/animations.html>

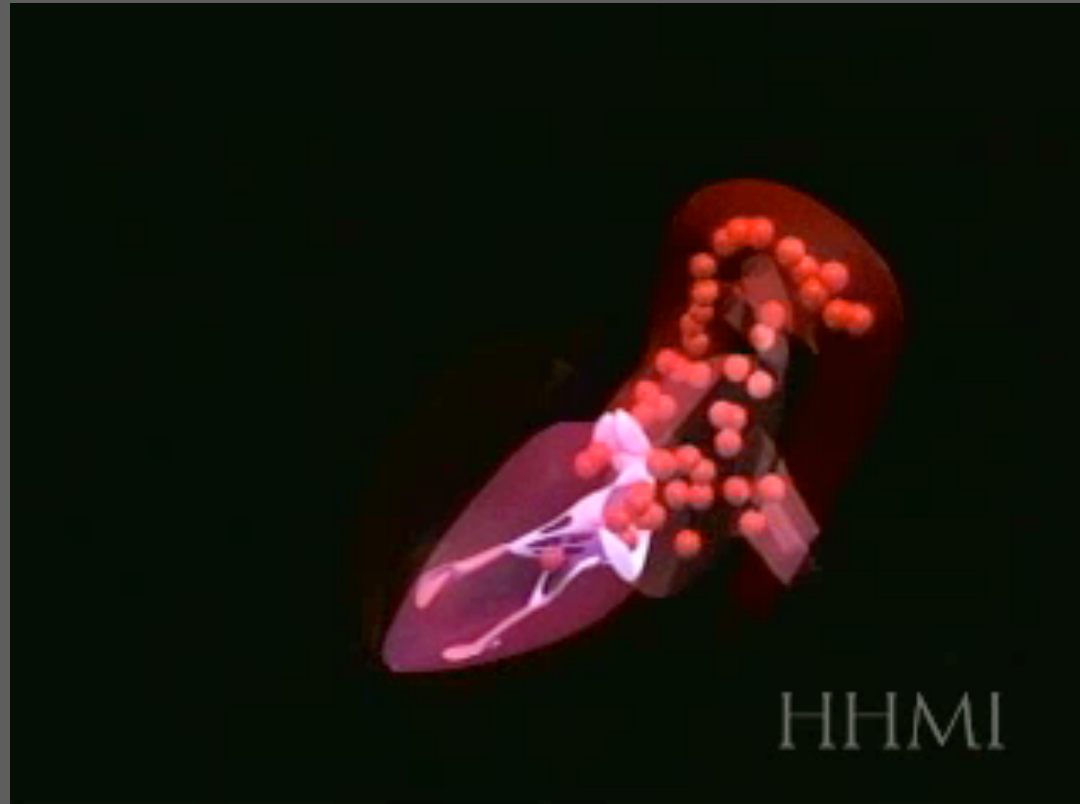
lower lights

## RIGHT atrium & ventricle



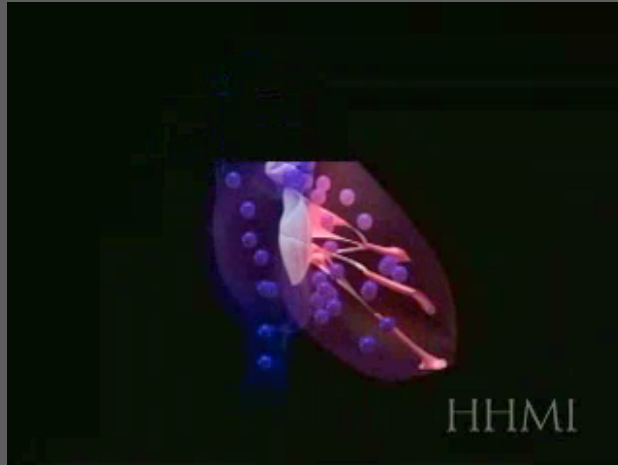
systemic veins -> vena cava -> right atrium ->  
right ventricle -> pulmonary artery -> lungs

## LEFT atrium & ventricle

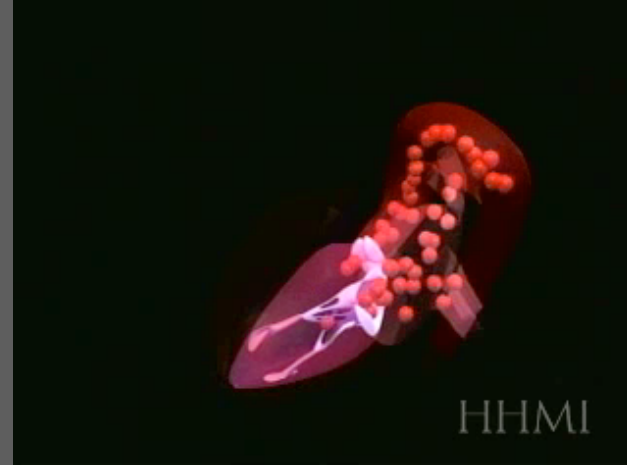


lungs -> pulmonary vein -> left atrium -> left  
ventricle -> aorta -> systemic arteries

RIGHT atrium & ventricle



LEFT atrium & ventricle



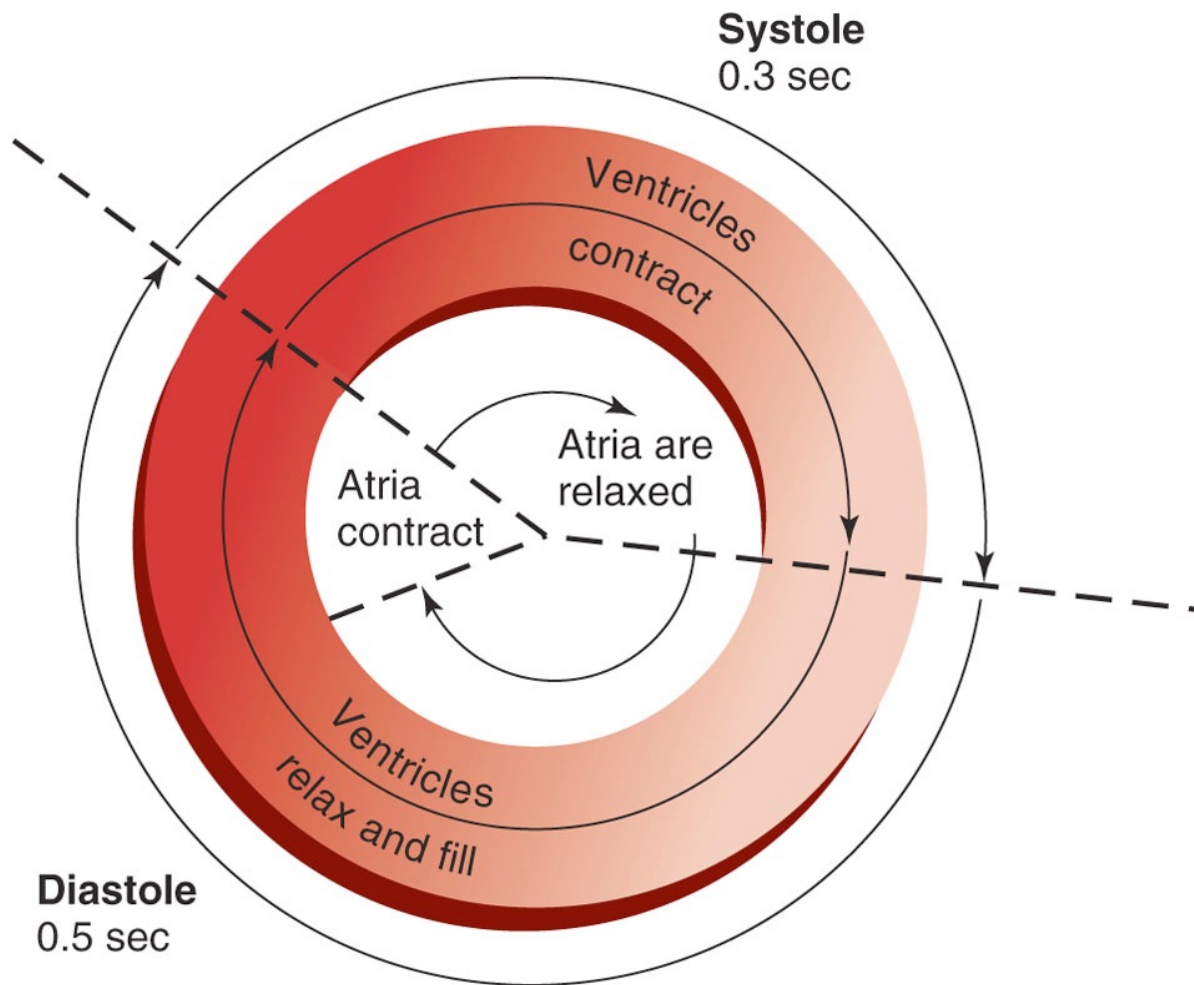
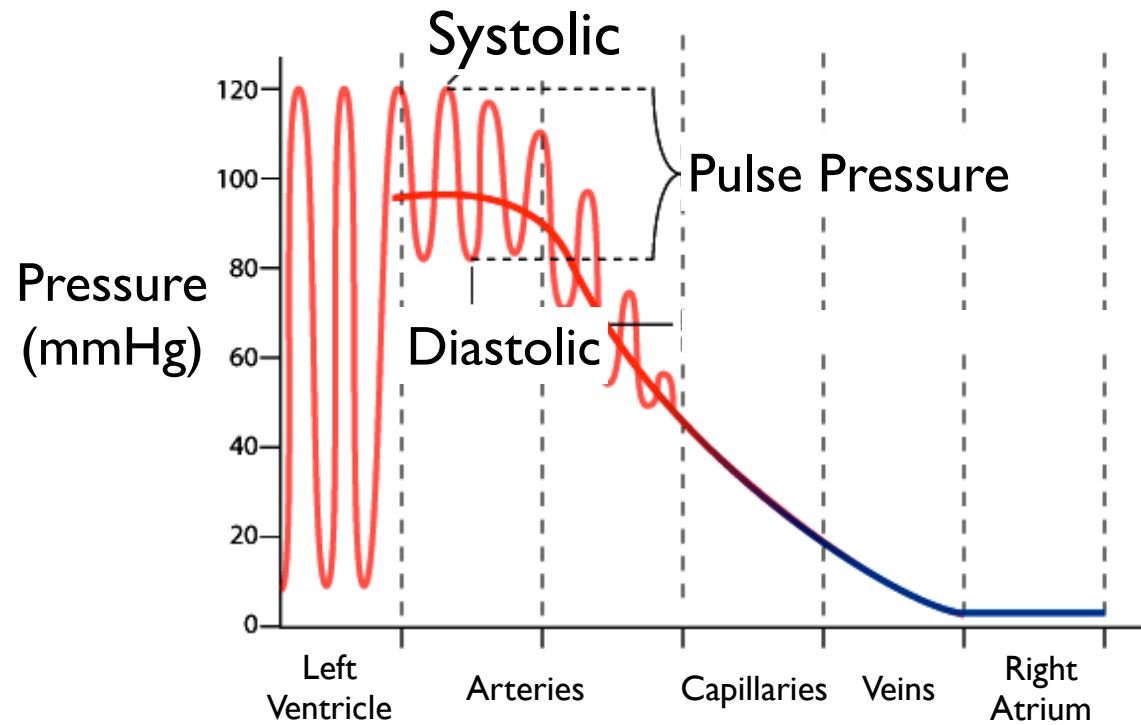


Figure 13.16



## Blood Pressure: Arterial Pressure > Venous Pressure



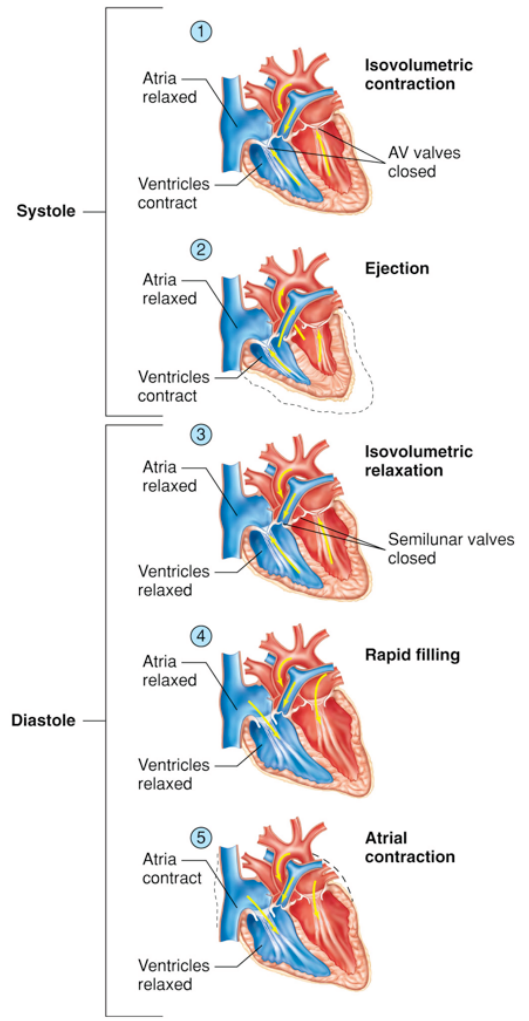
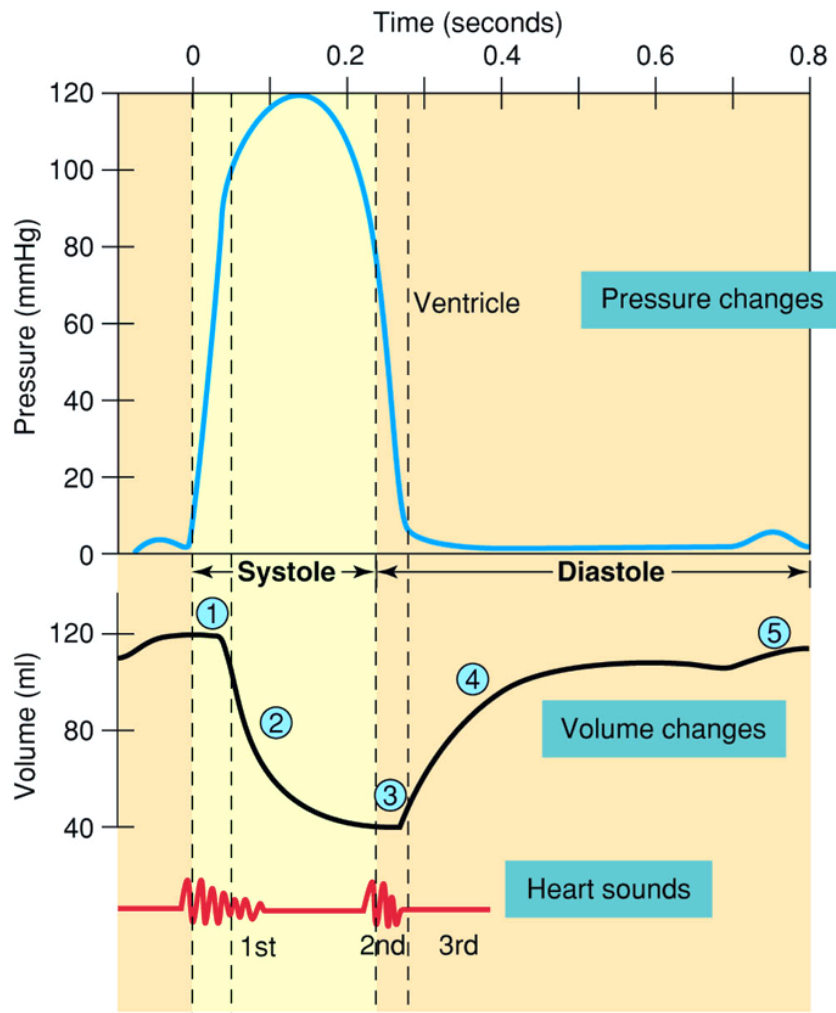
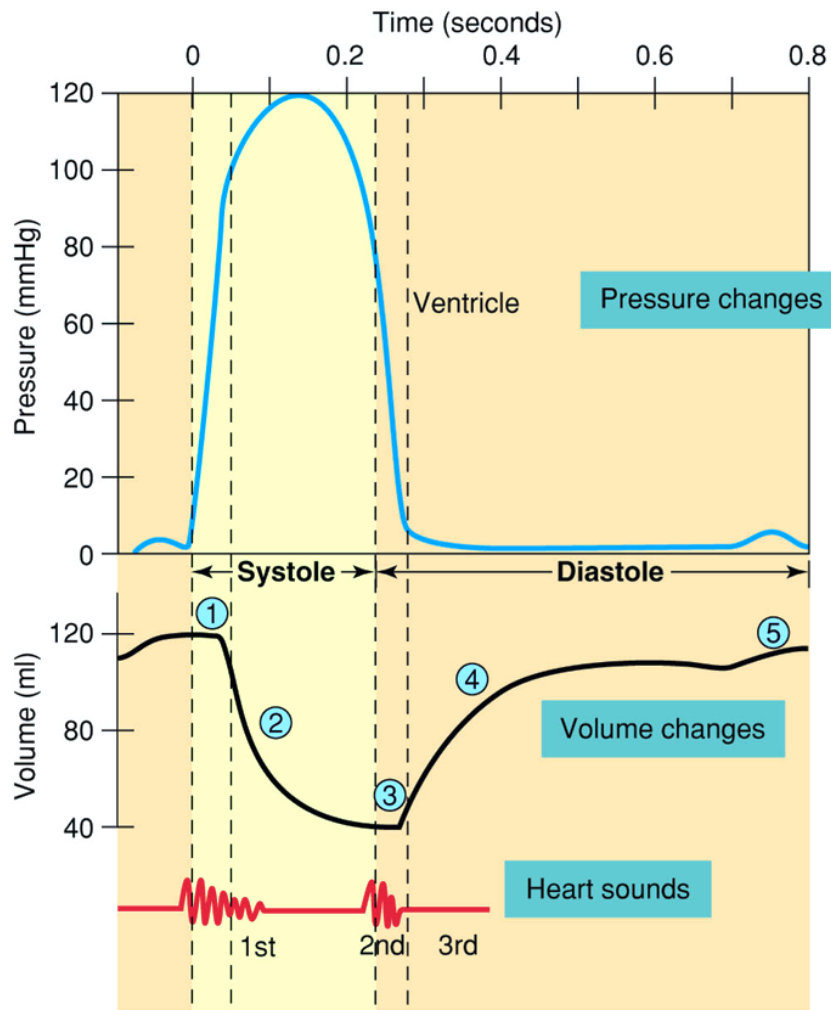


Figure 13.17



### Ventricular Systole

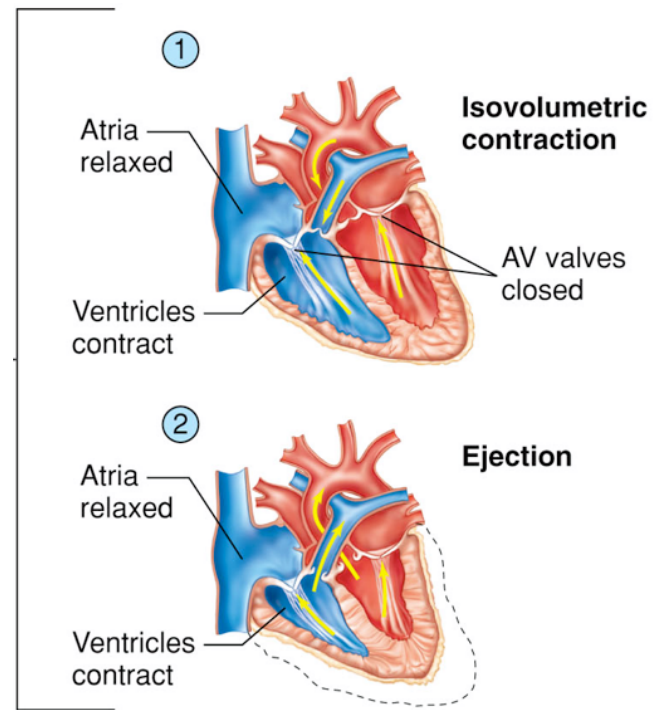
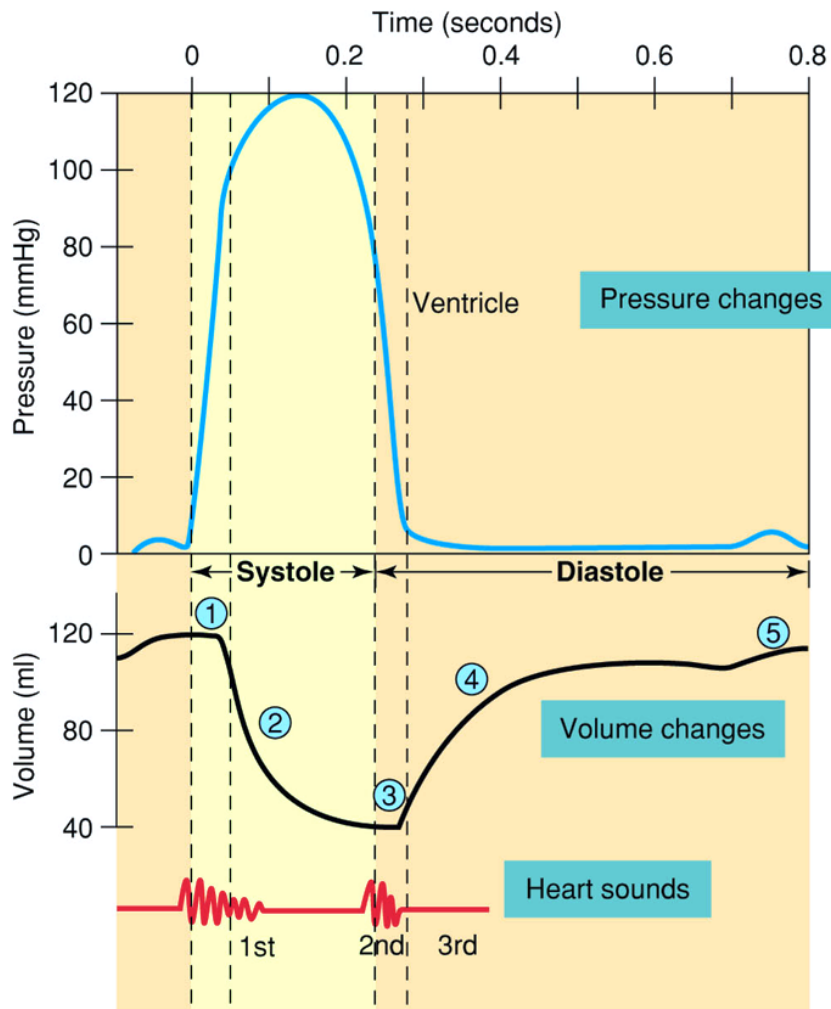


Figure 13.17



### Ventricular Diastole

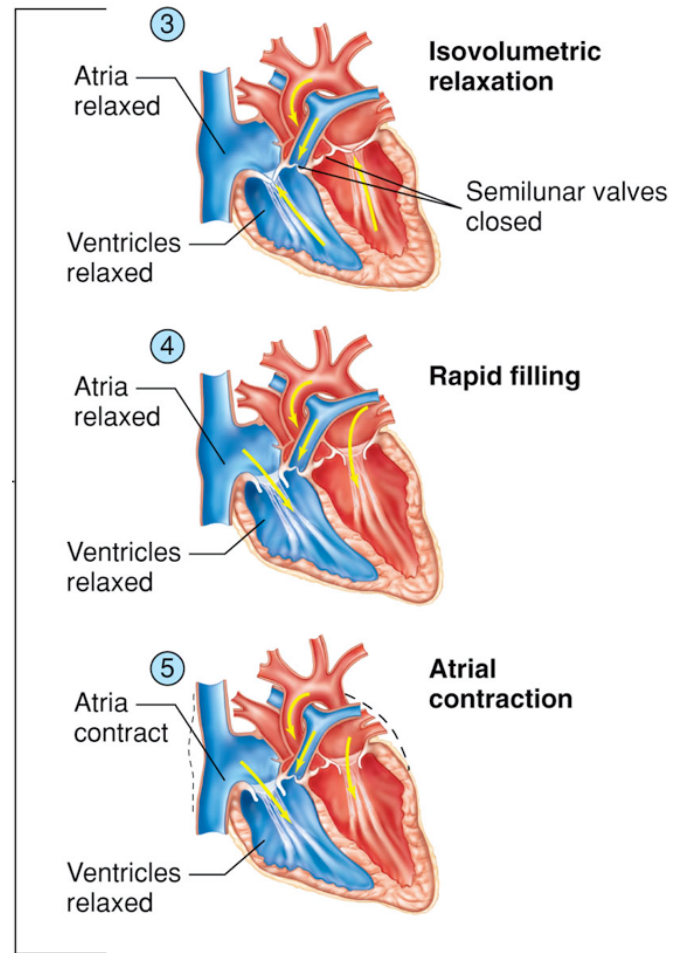


Figure 13.17

## Events of the Cardiac Cycle

Atria	AV Valves	Ventricles	Semi-Lunar Valves	Blood Flow
diastole	open	<b>diastole</b>	closed	into atria (from vena cava, lungs)
systole	open	<b>diastole</b>	closed	into ventricles
diastole	closed	<b>systole</b>	open	into lungs, aorta
diastole	open	<b>diastole</b>	closed	into atria (from vena cava, lungs)

*diastole = relaxed, systole = contracting*

## Heart Beat

1. Generate rhythmic stimulation to start cardiac action potential
2. Action potential will cause contraction of cardiac muscle
3. Allow action potential to spread across the heart; introduce delay between atria and ventricles so they don't contract at the same time

## Contraction of Cardiac Muscle (Myocardium)

Action Potential starts from pacemaker cells in **sinoatrial node** (SA node)

HCN channels open when hyperpolarized (**H**yperpolarization-activated **C**yclic **N**ucleotide-gated channels)

-> spontaneous depolarization of pacemaker cells to -40 mV

-> opening of voltage-gated Ca<sup>++</sup> channels

-> + 20 mV -> action potential across myocardium

Myocardial Action Potential is longer than neural action potential:

fast Na<sup>+</sup> channels -> fast depolarization

slow Ca<sup>++</sup> channels -> plateau phase

voltage-gated K<sup>+</sup> channels cause repolarization

Myocardium forms a functional **syncytium**, via **gap junctions**

### **Myocardial Contraction**

Voltage-gated Na<sup>+</sup> channels open, causing depolarization

Voltage-gated Ca<sup>++</sup> channels open in transverse tubules

Influx of Ca<sup>++</sup> causes release of Ca<sup>++</sup> from sarcoplasmic reticulum

Ca<sup>++</sup> binds to troponin to allow contraction

Ca<sup>++</sup> ATPase pump returns Ca<sup>++</sup> into sarcoplasmic reticulum

Na<sup>+</sup>/Ca<sup>++</sup> exchanger pumps Ca<sup>++</sup> from cytoplasm into extracellular fluid

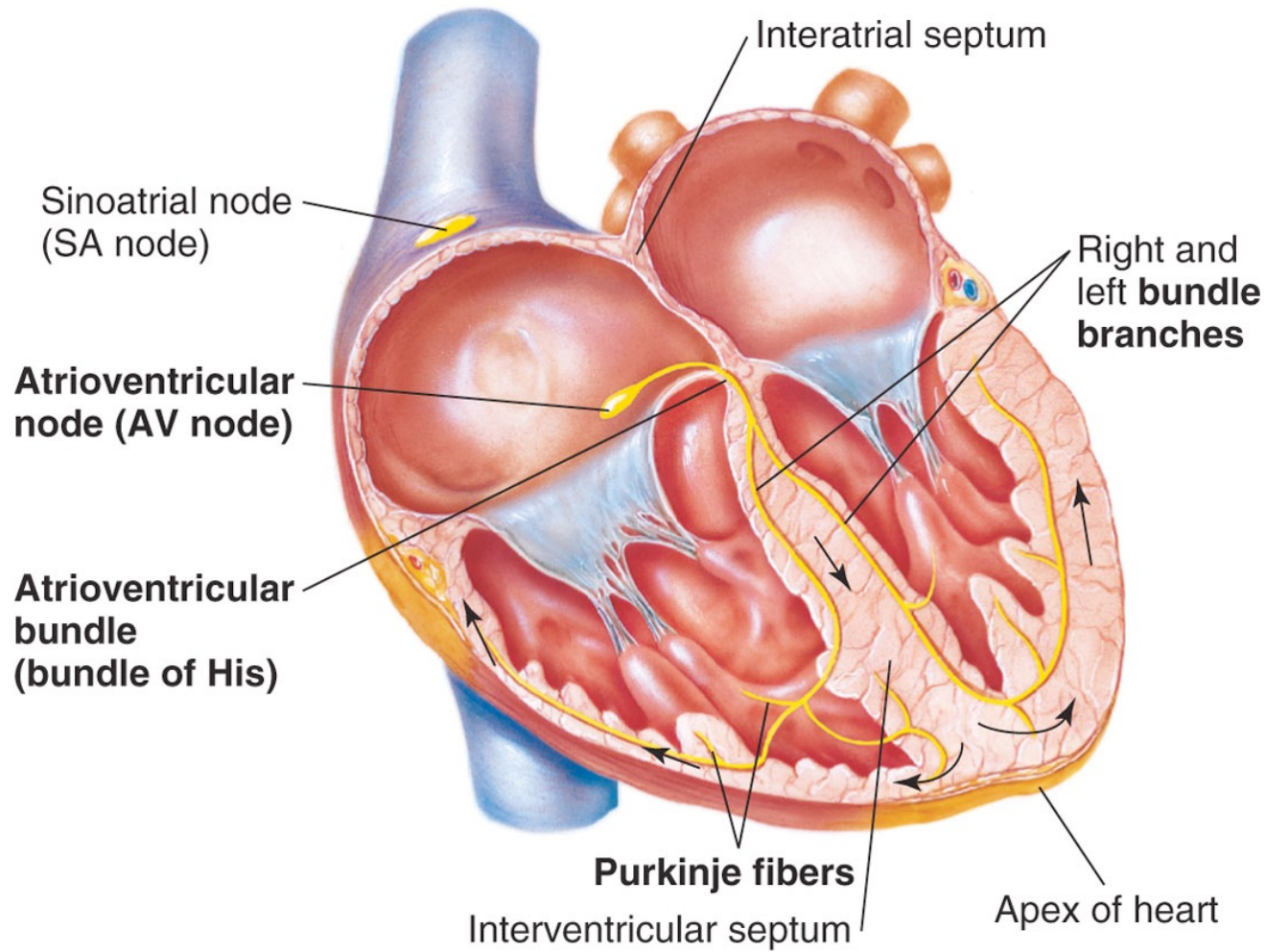
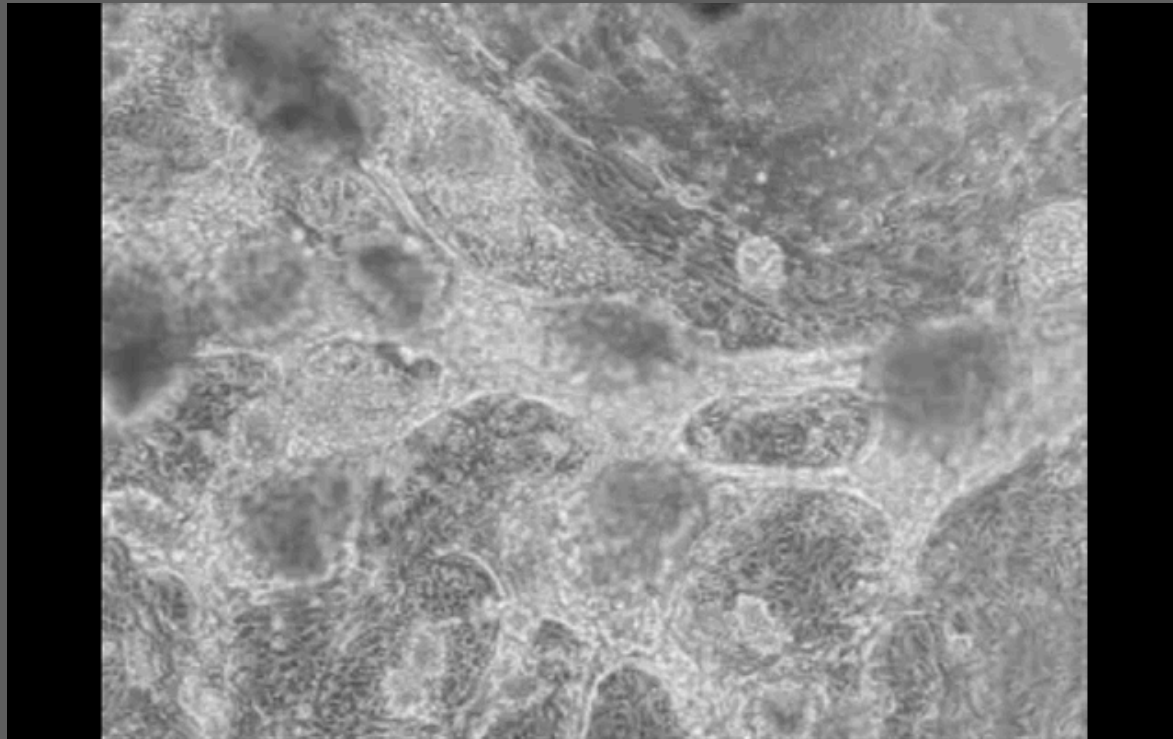


Figure 13.20



Stem cells differentiate into cardiac pacemaker cells  
*in vitro* (in a petri dish)



Drexel University

<http://io9.com/5882963/whoa-a-petri-dish-that-has-a-pulse>

<https://www.youtube.com/watch?v=BqzW9Jq-OVA>

# Spontaneous Depolarization of Pacemaker Cells

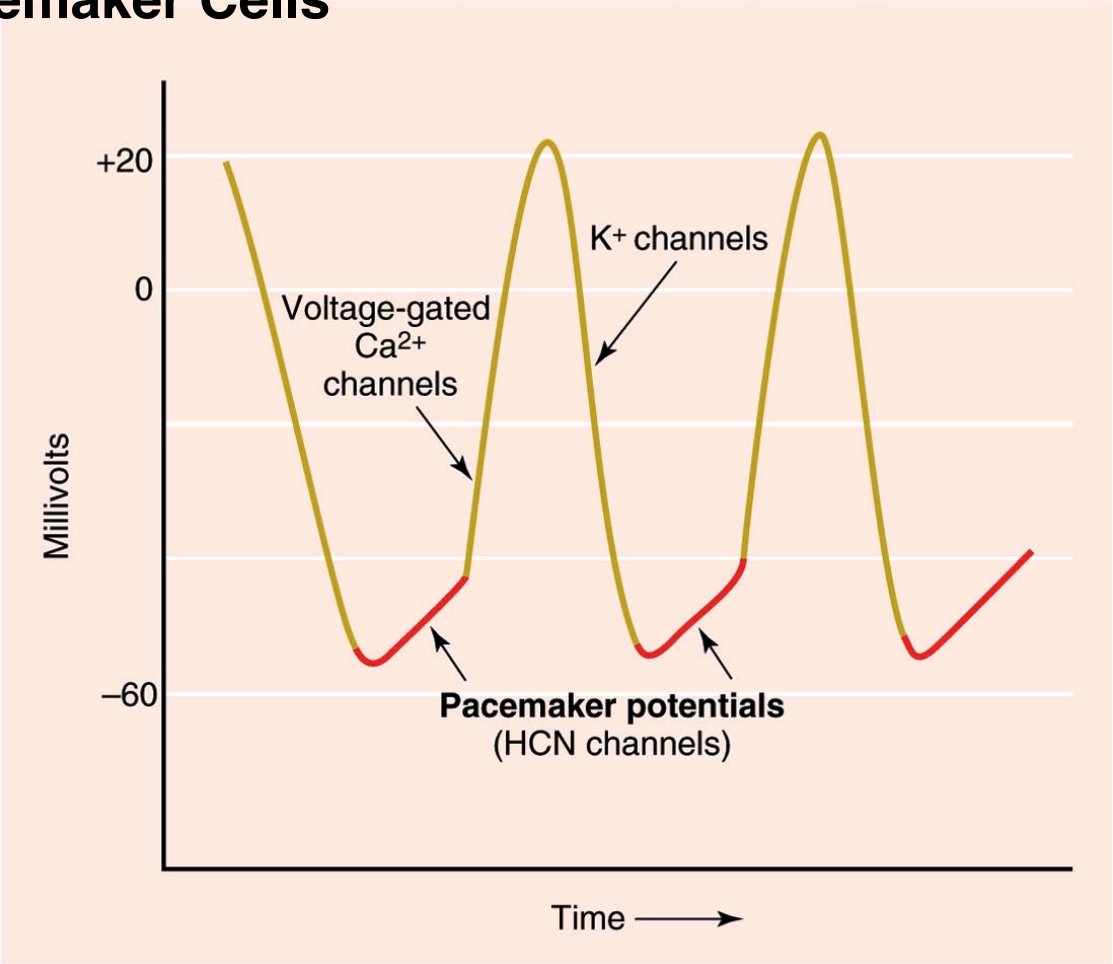
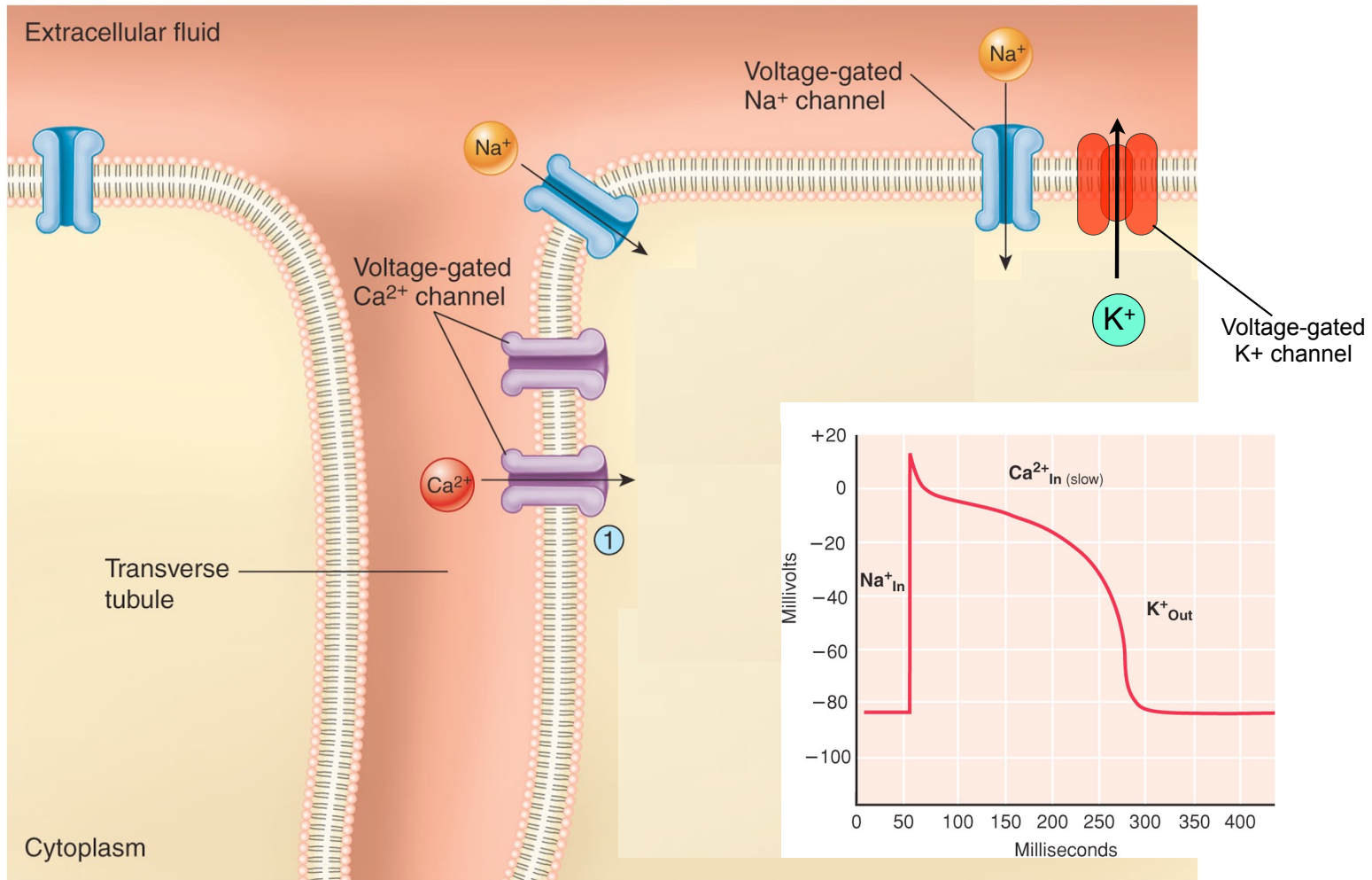


Figure 13.18



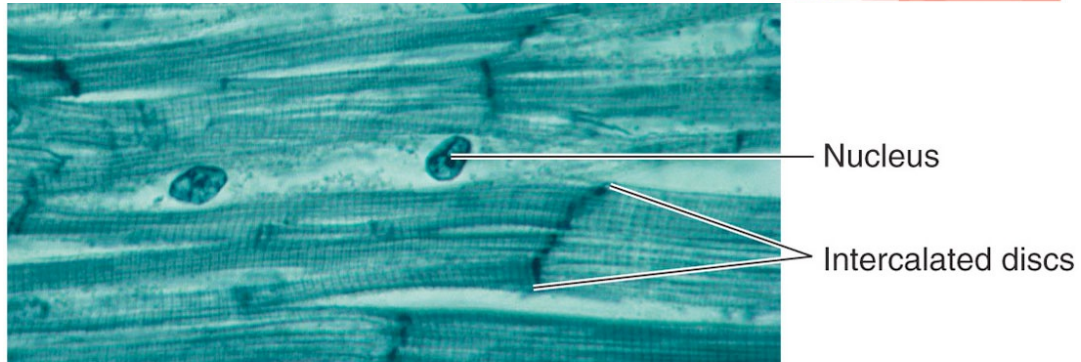
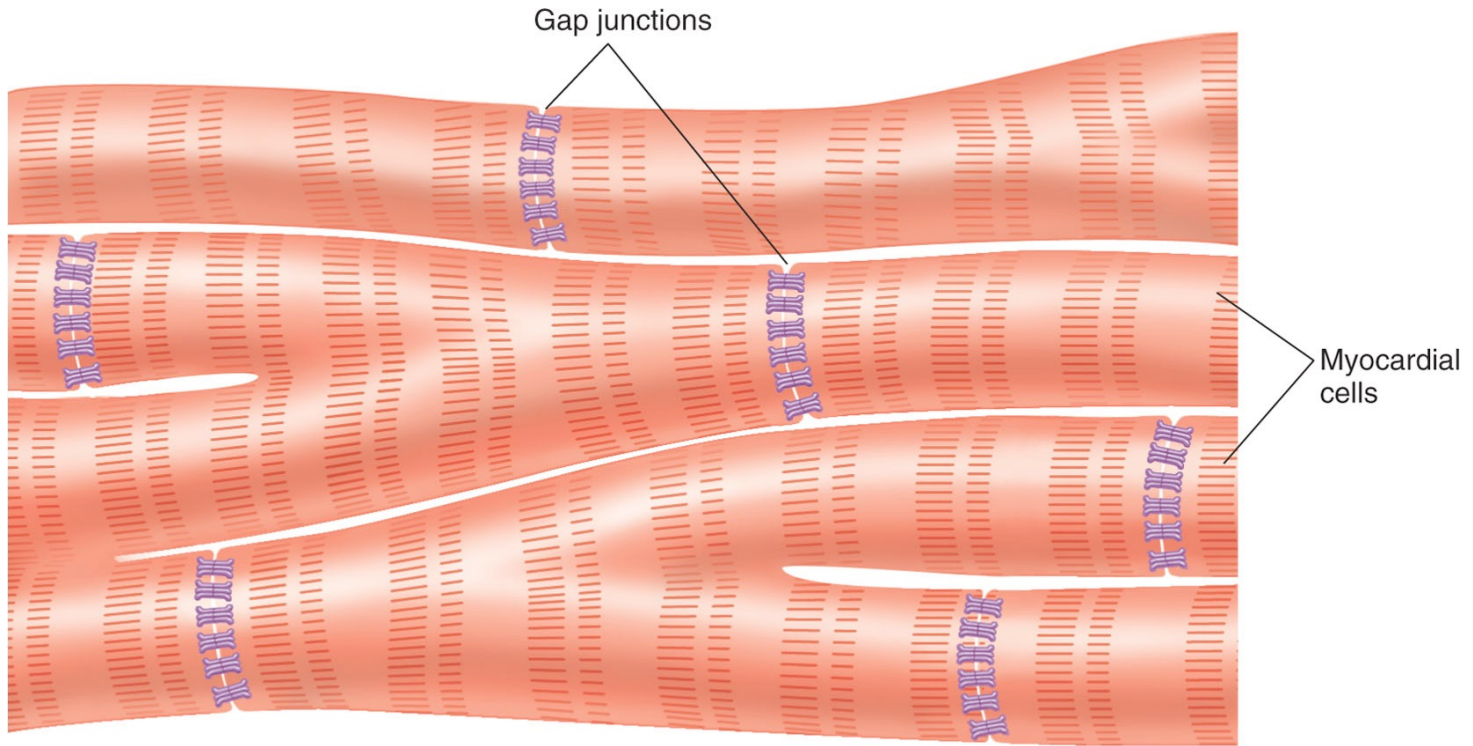
frog heart does not beat in  $\text{Ca}^{++}$  free buffer

## Comparison of Skeletal Muscle and Cardiac Muscle

Skeletal Muscle	Cardiac Muscle
Striated; actin and myosin arranged in sarcomeres	Striated; actin and myosin arranged in sarcomeres
Well-developed sarcoplasmic reticulum and transverse tubules	Moderately developed sarcoplasmic reticulum and transverse tubules
Contains troponin in the thin filaments	Contains troponin in the thin filaments
Ca <sup>2+</sup> released into cytoplasm from sarcoplasmic reticulum	Ca <sup>2+</sup> enters cytoplasm from sarcoplasmic reticulum and extracellular fluid
Cannot contract without nerve stimulation; denervation results in muscle atrophy	Can contract without nerve stimulation; action potentials originate in pacemaker cells of heart
Muscle fibers stimulated independently; no gap junctions	Gap junctions present as intercalated discs

myocardial infarction (tissue damage due to lack of oxygen)  
 -> cardiac troponin in the blood

Table 12.8



© Ed Reschke

Figure 12.32

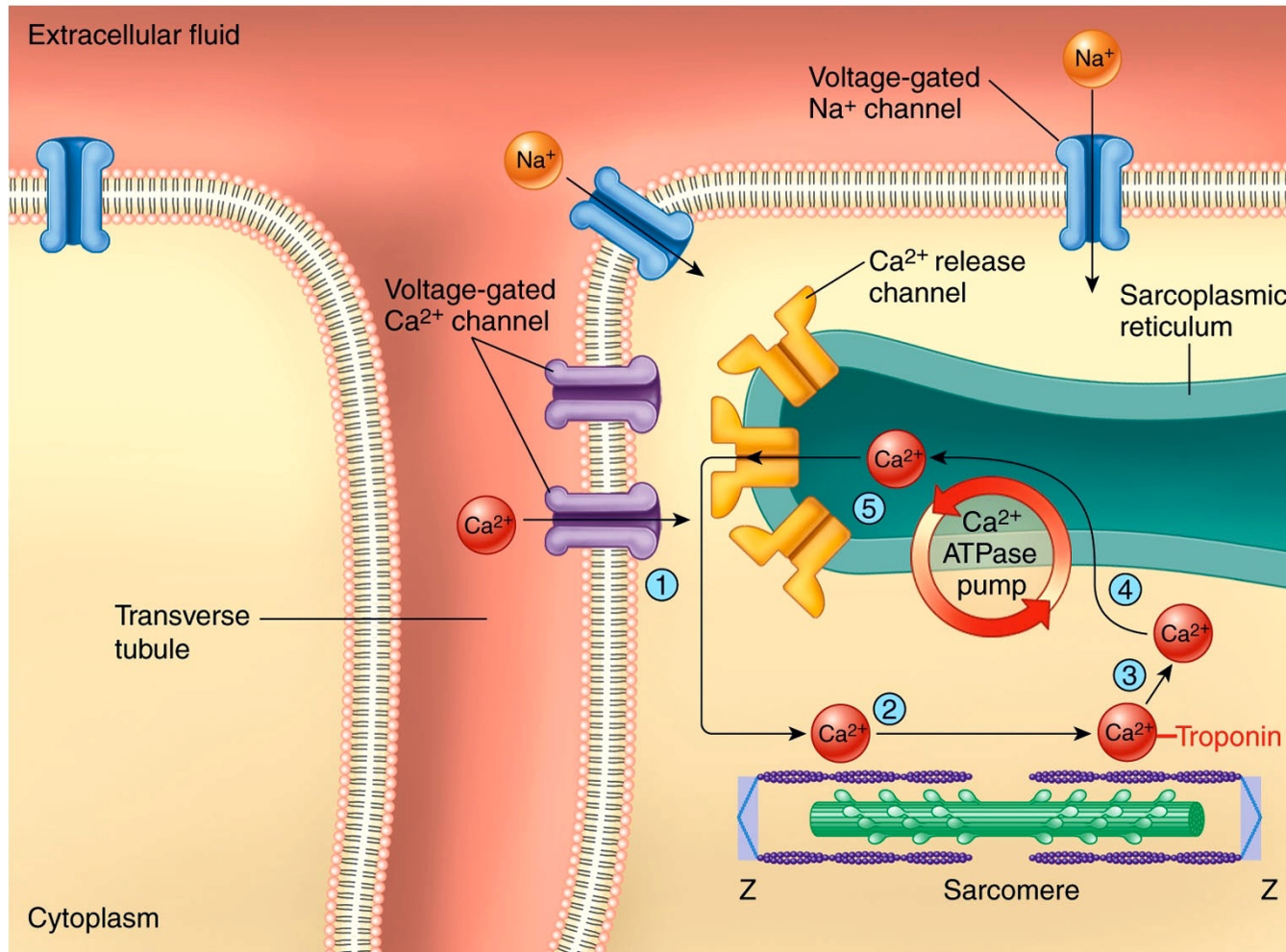


Figure 12.34

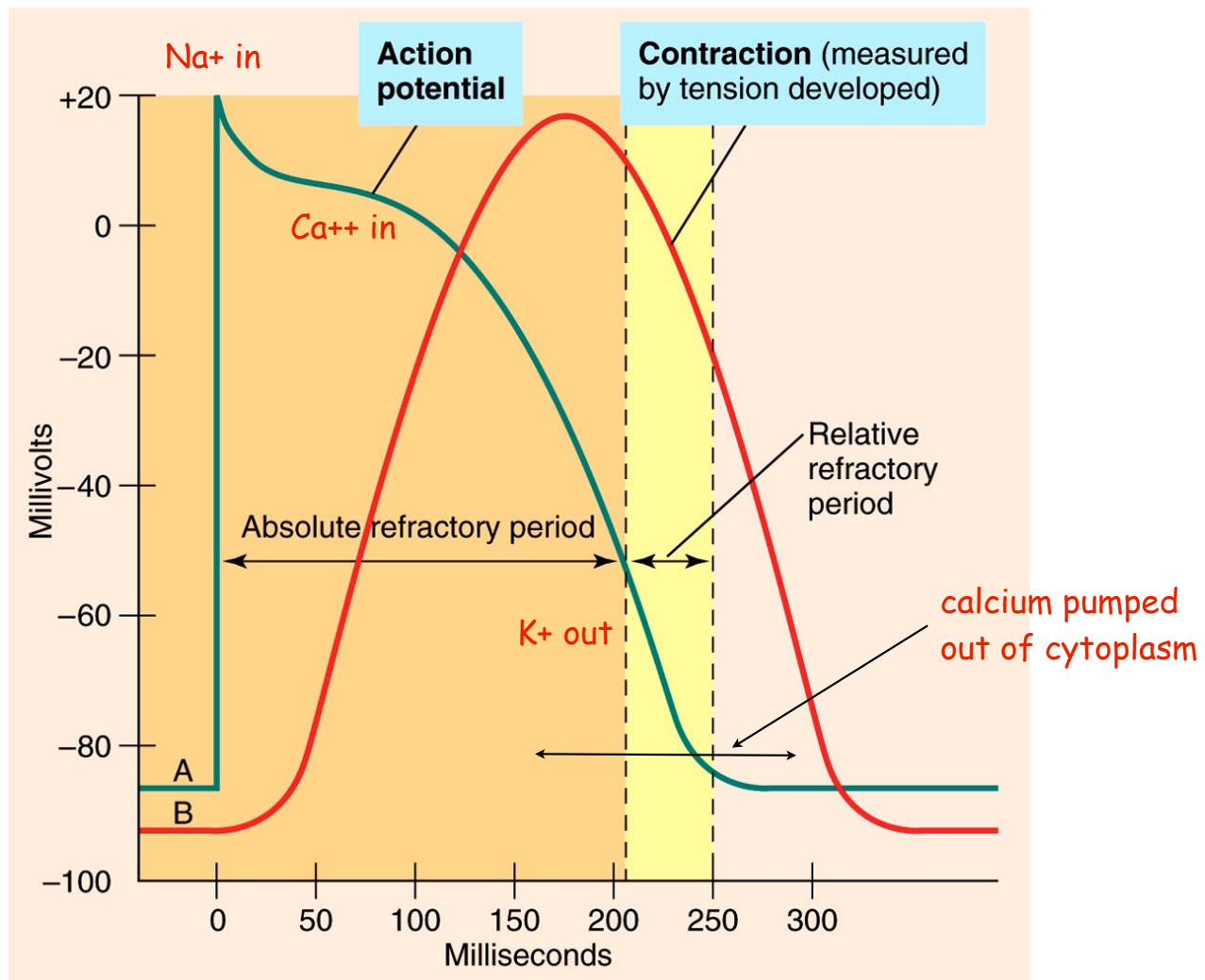


Figure 13.21

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# Conduction of action potential across heart and Electrocardiogram (ECG)

Action Potential (AP) spreads from pacemaker cells in **SA node**.

Myocardium forms a functional **syncitium**, via **gap junctions**

AP spreads rapidly across atria to cause depolarization and atrial systole (contraction). **[P wave]**

AP cannot cross directly to ventricles: must pass through **atrioventricular node** (AV node).

Slow conduction through AV node causes delay between atrial and ventricular contraction.

AP spreads from AV node through bundle of His and along Purkinje fibers in the walls of the ventricles. [Atria repolarize.]

Ventricles depolarize and contract. **[QRS wave]**

Ventricles repolarize. **[T wave]**

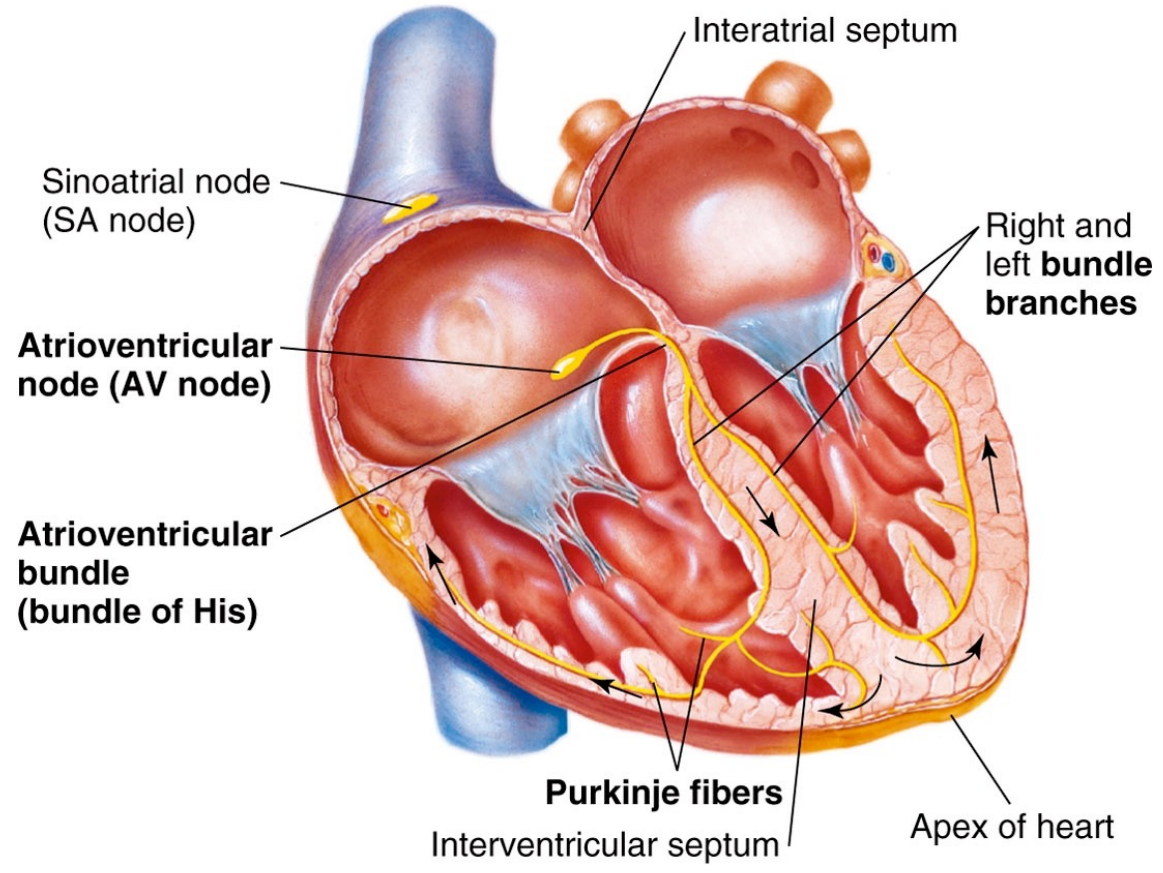


Figure 13.20

Figure 42.7 The control of heart rhythm

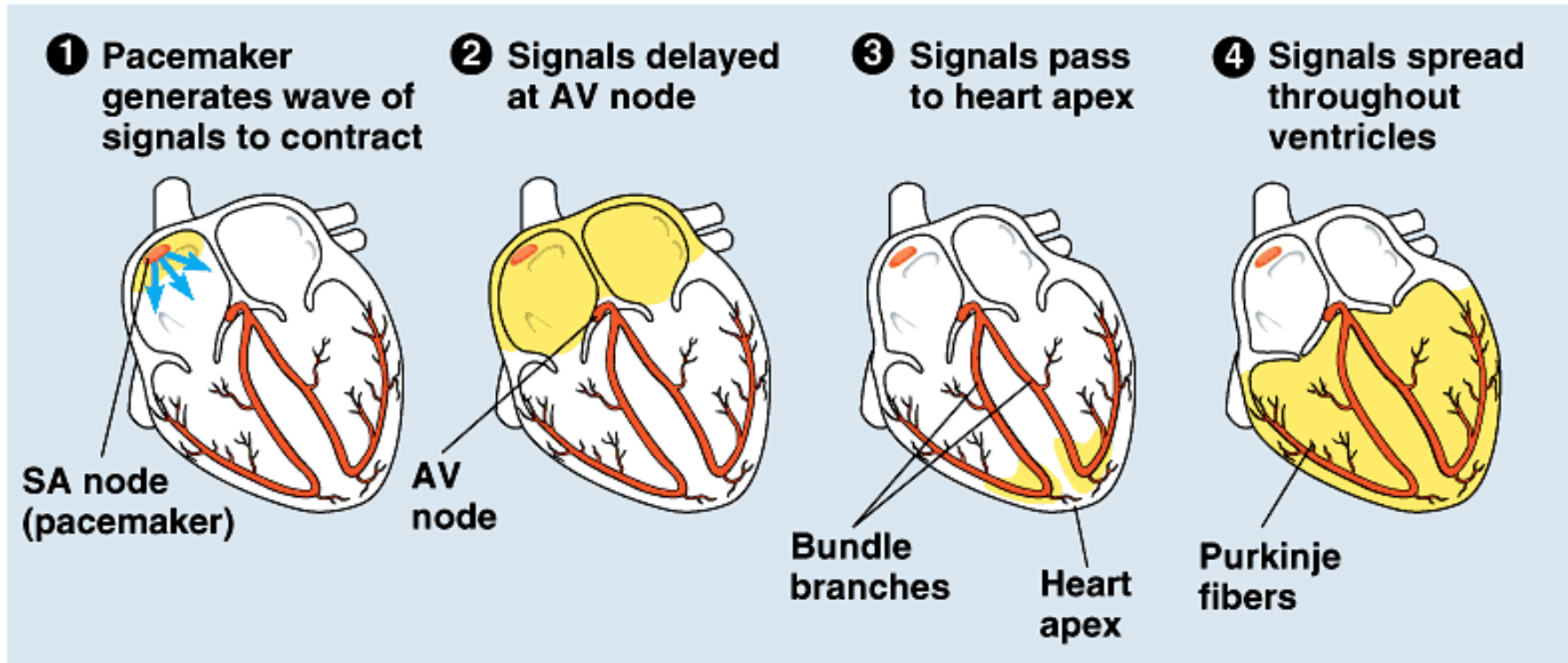
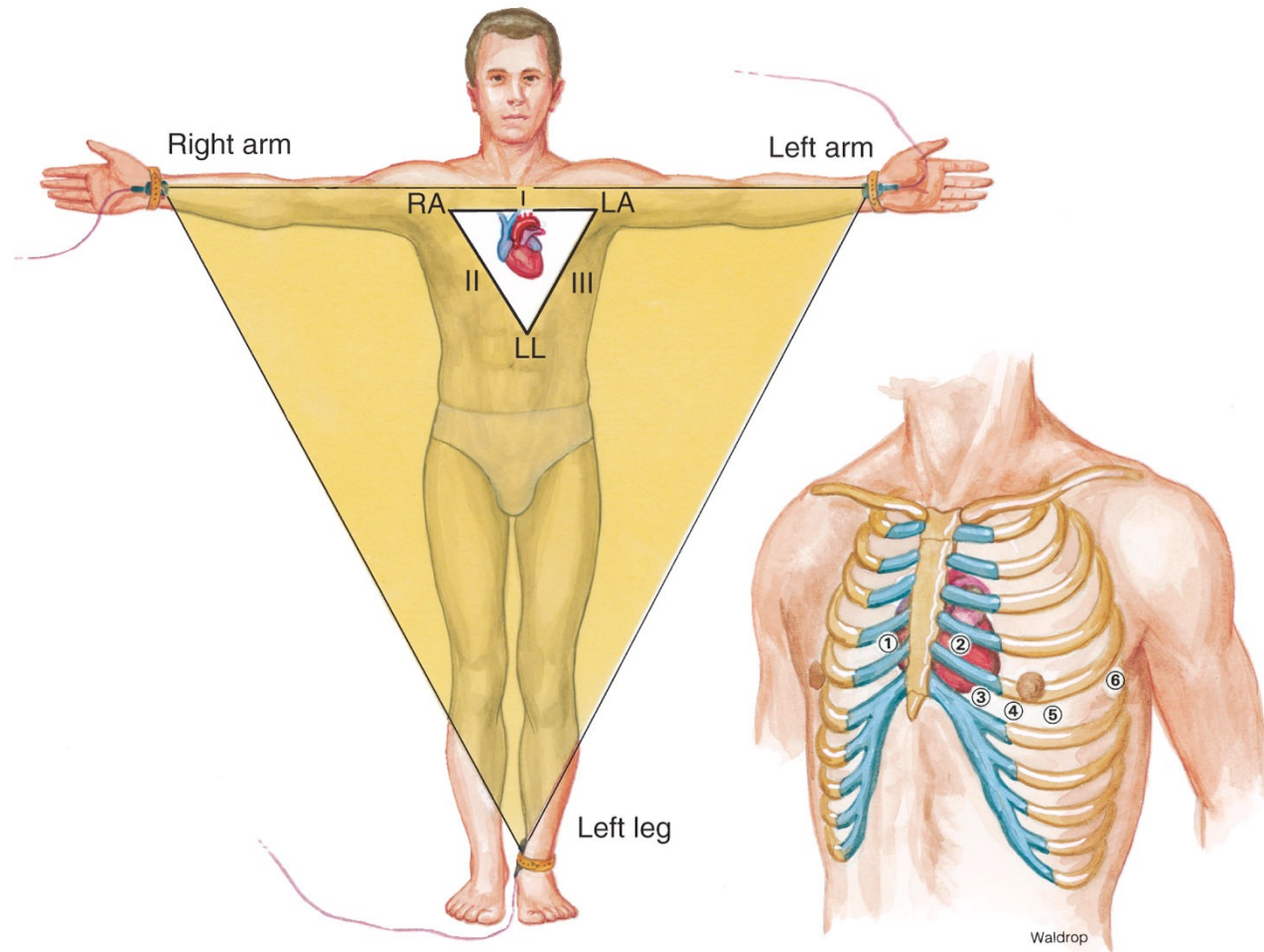
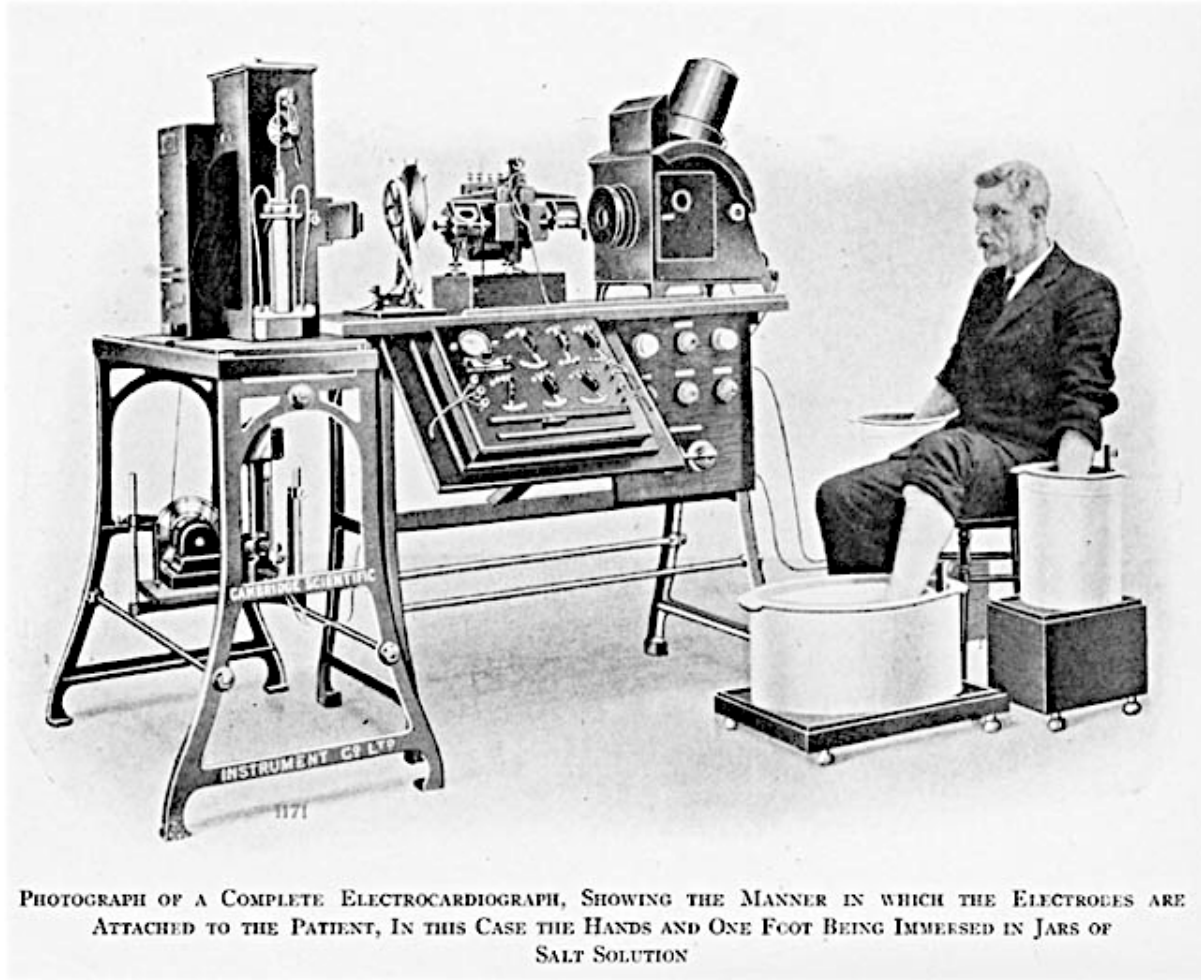


Figure 13.24

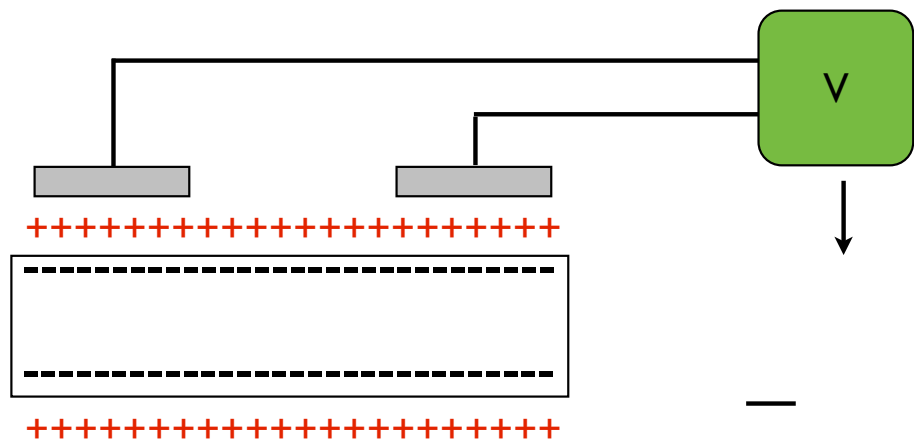


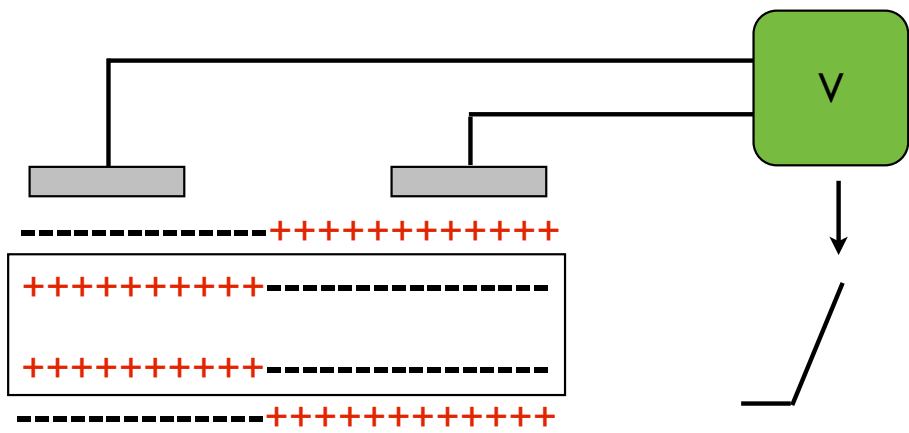


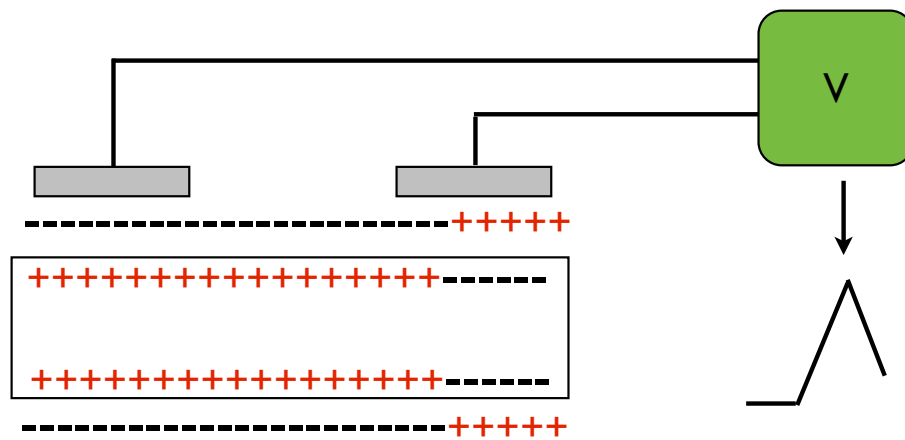
PHOTOGRAPH OF A COMPLETE ELECTROCARDIOGRAPH, SHOWING THE MANNER IN WHICH THE ELECTRODES ARE ATTACHED TO THE PATIENT, IN THIS CASE THE HANDS AND ONE FOOT BEING IMMersed IN JARS OF SALT SOLUTION

1911

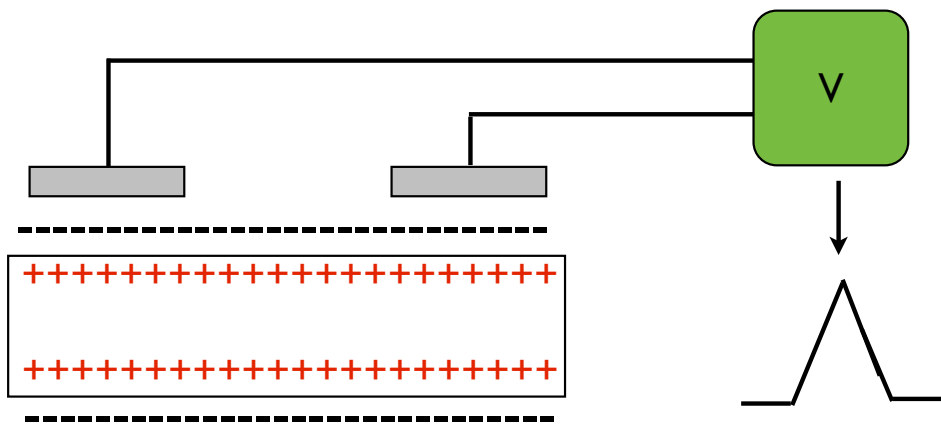
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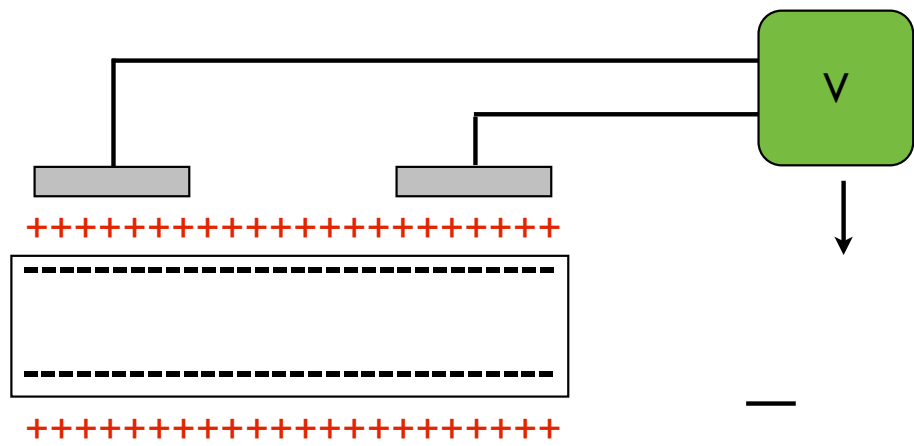


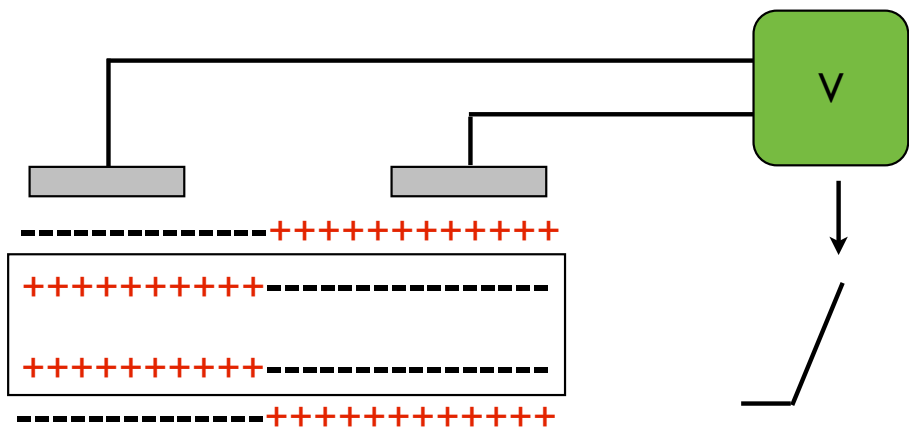


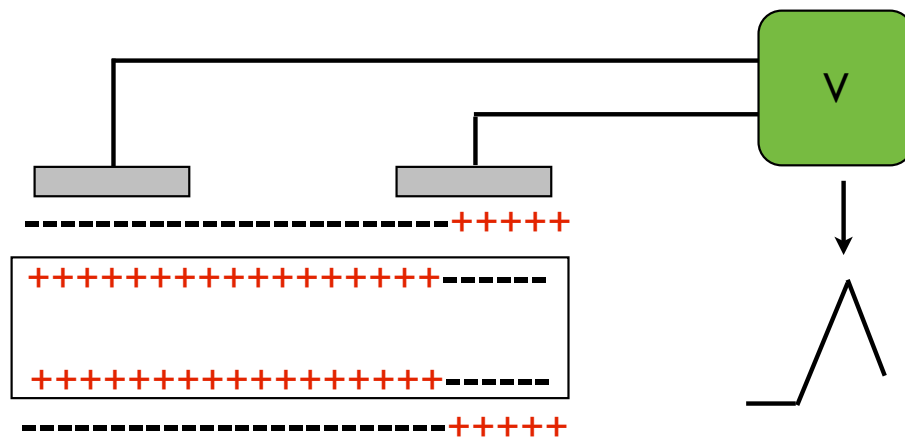


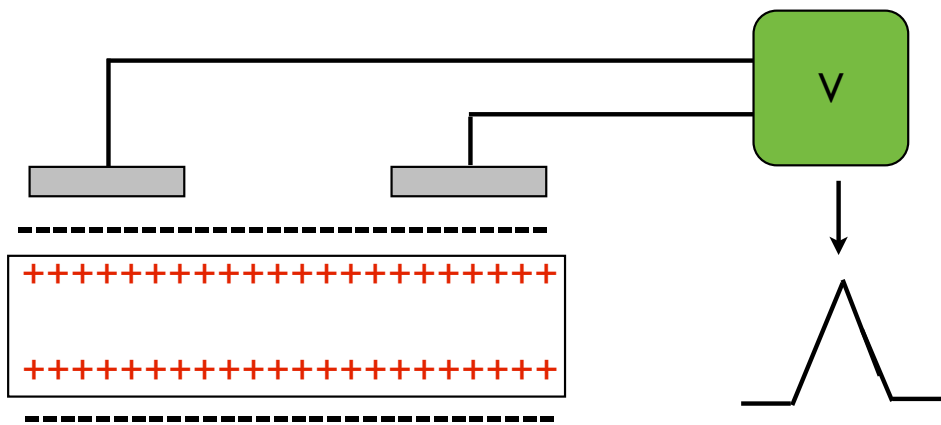


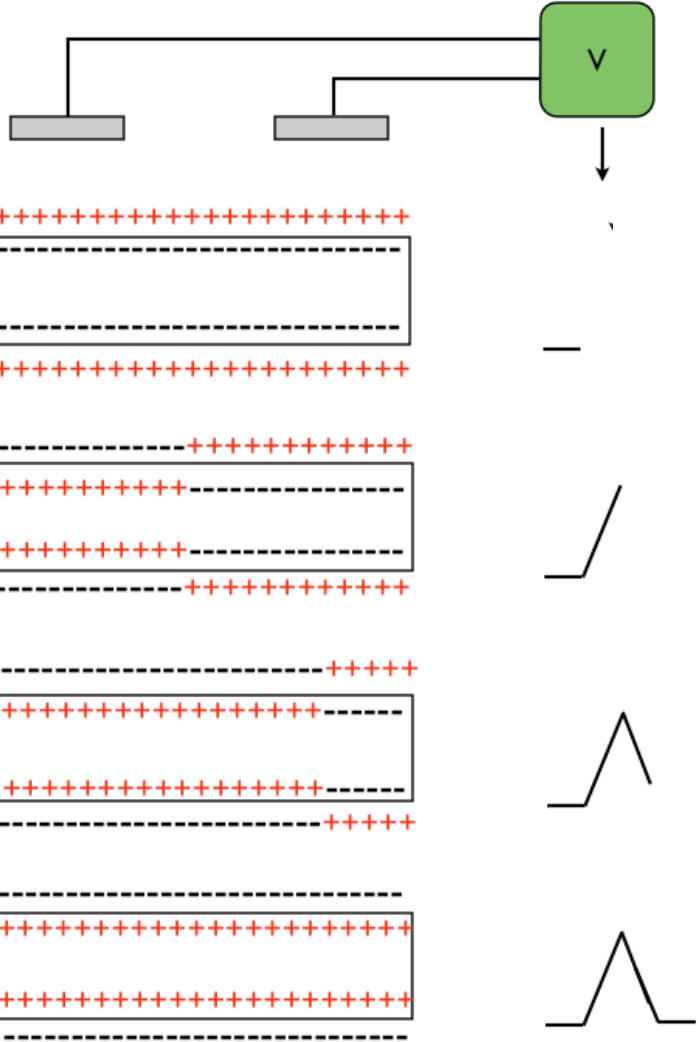


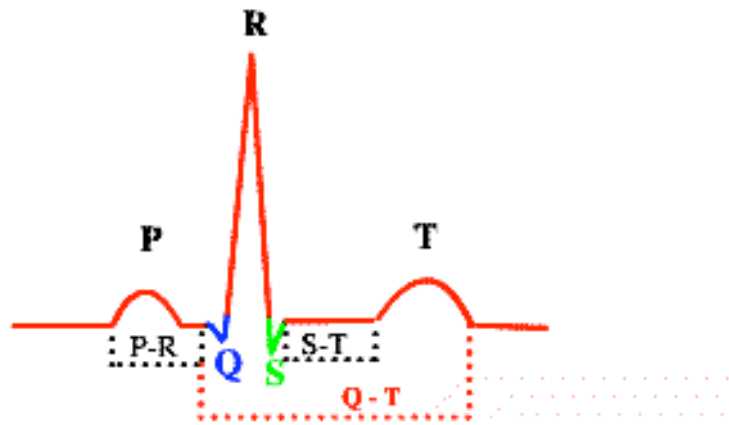
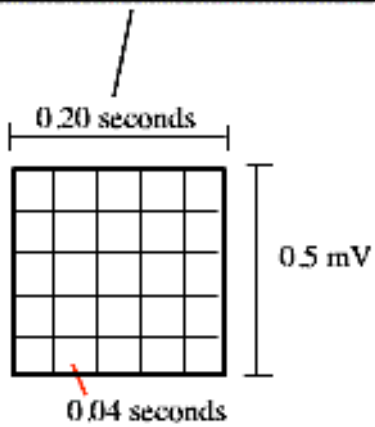
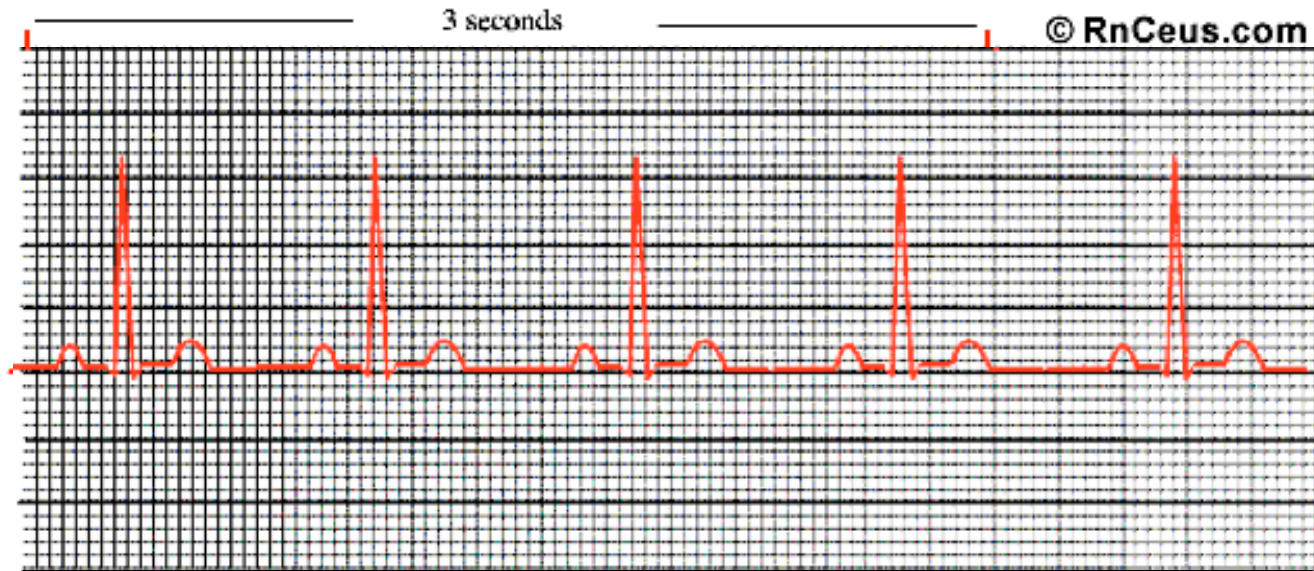












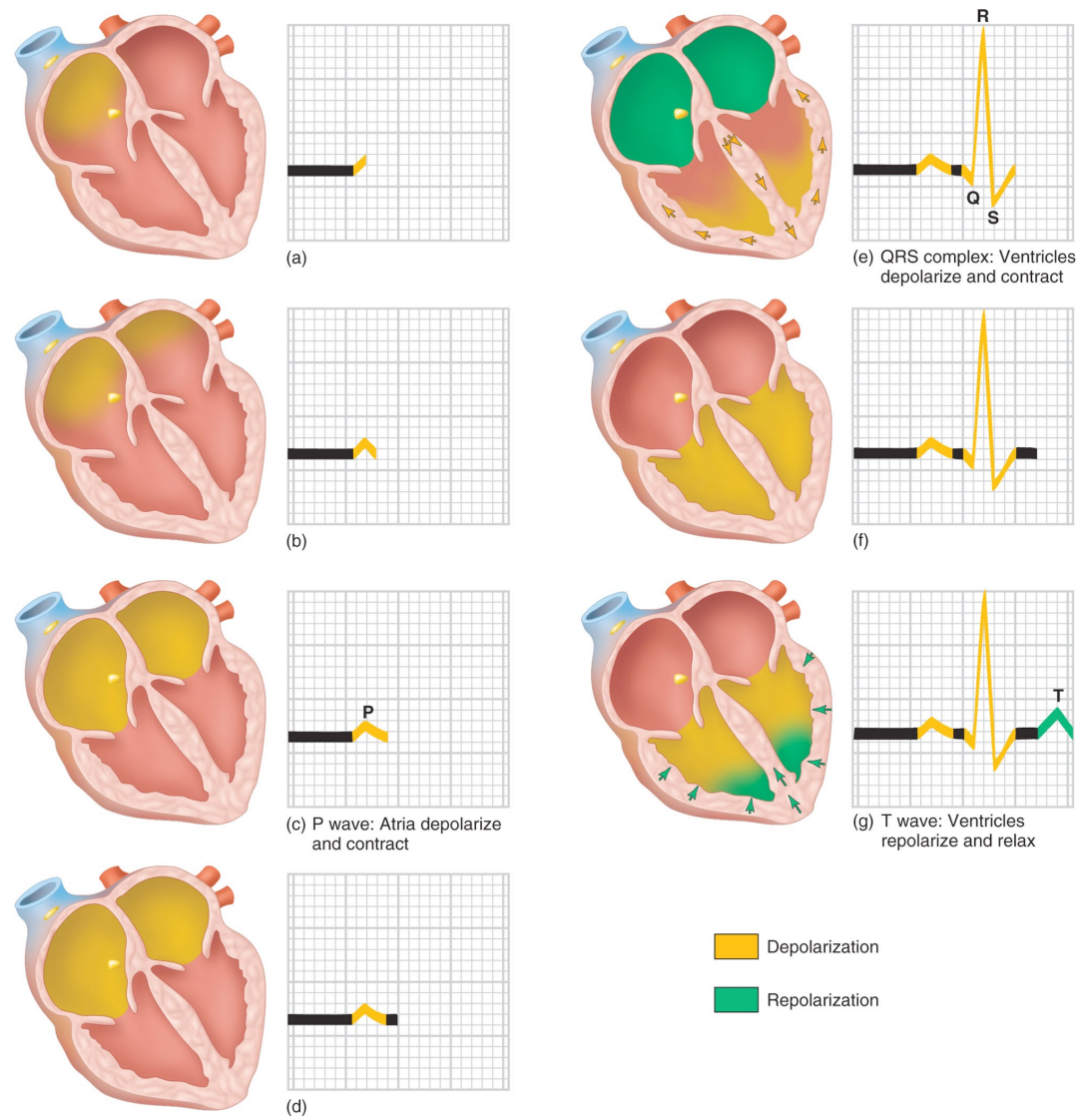


Figure 13.23



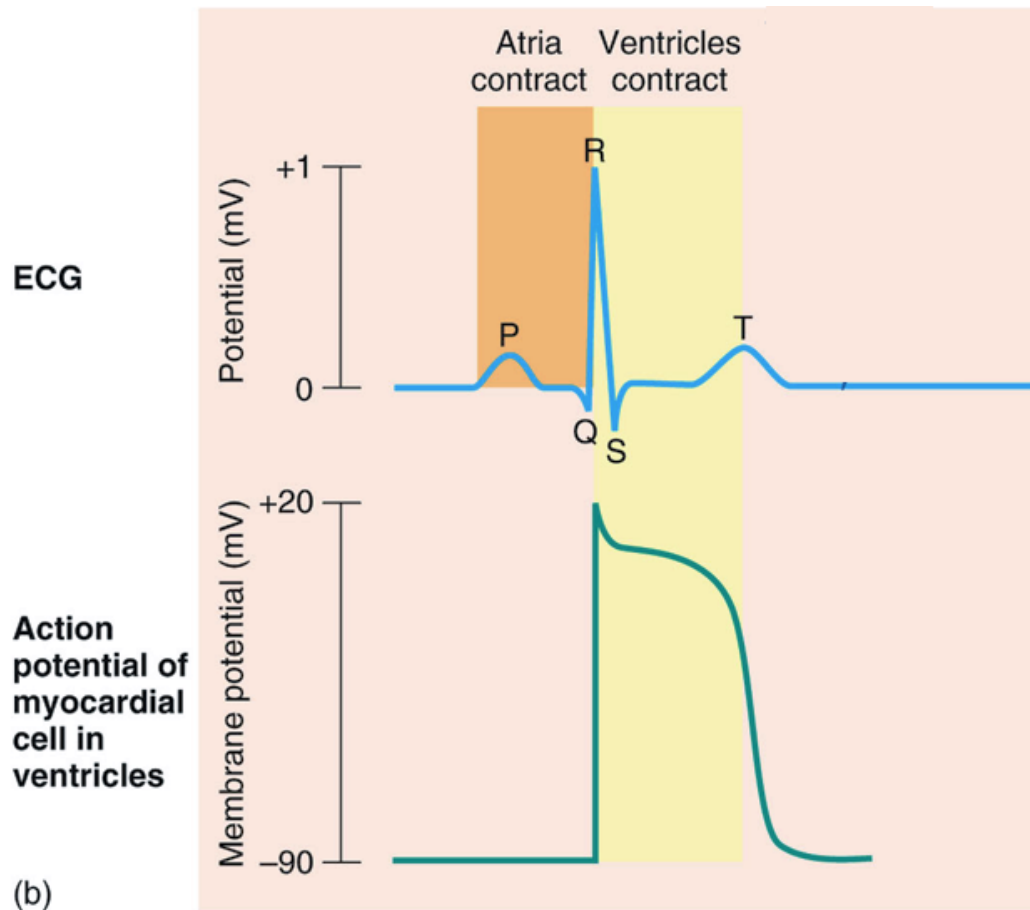


Figure 13.22

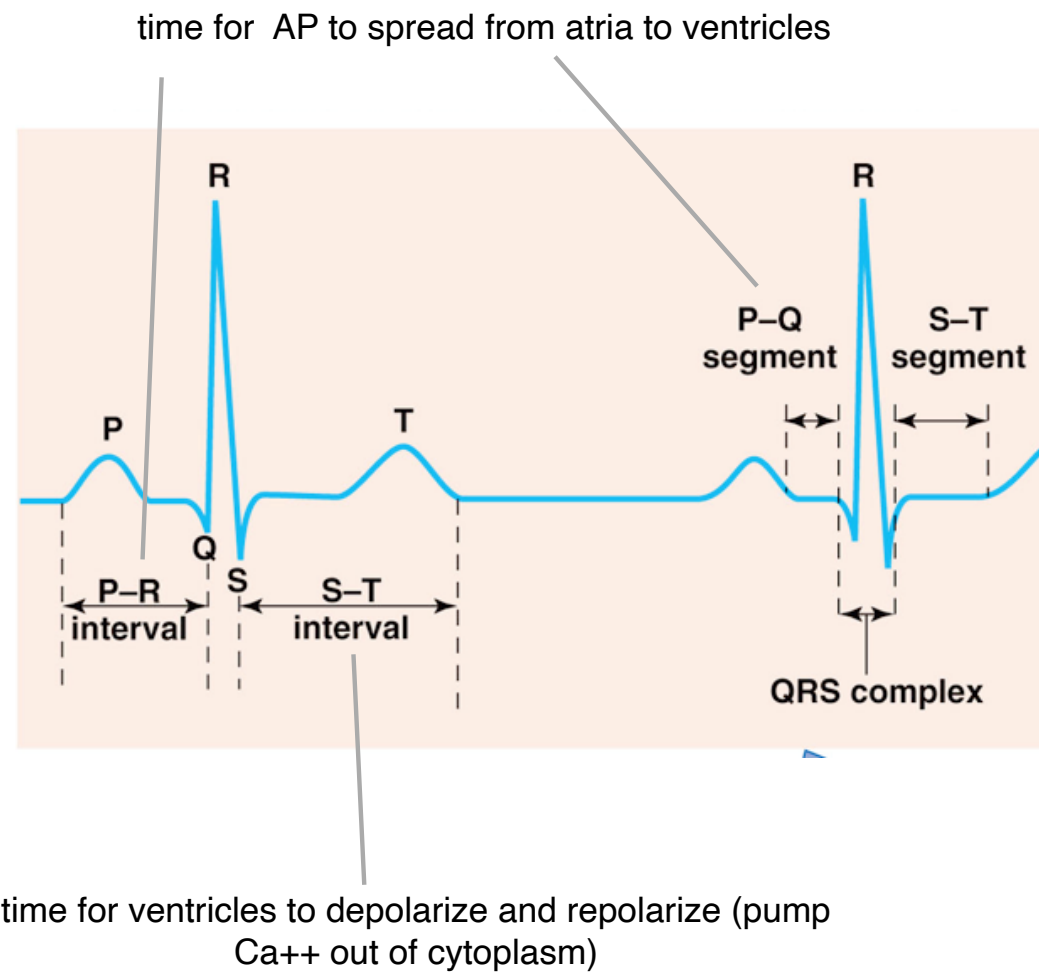
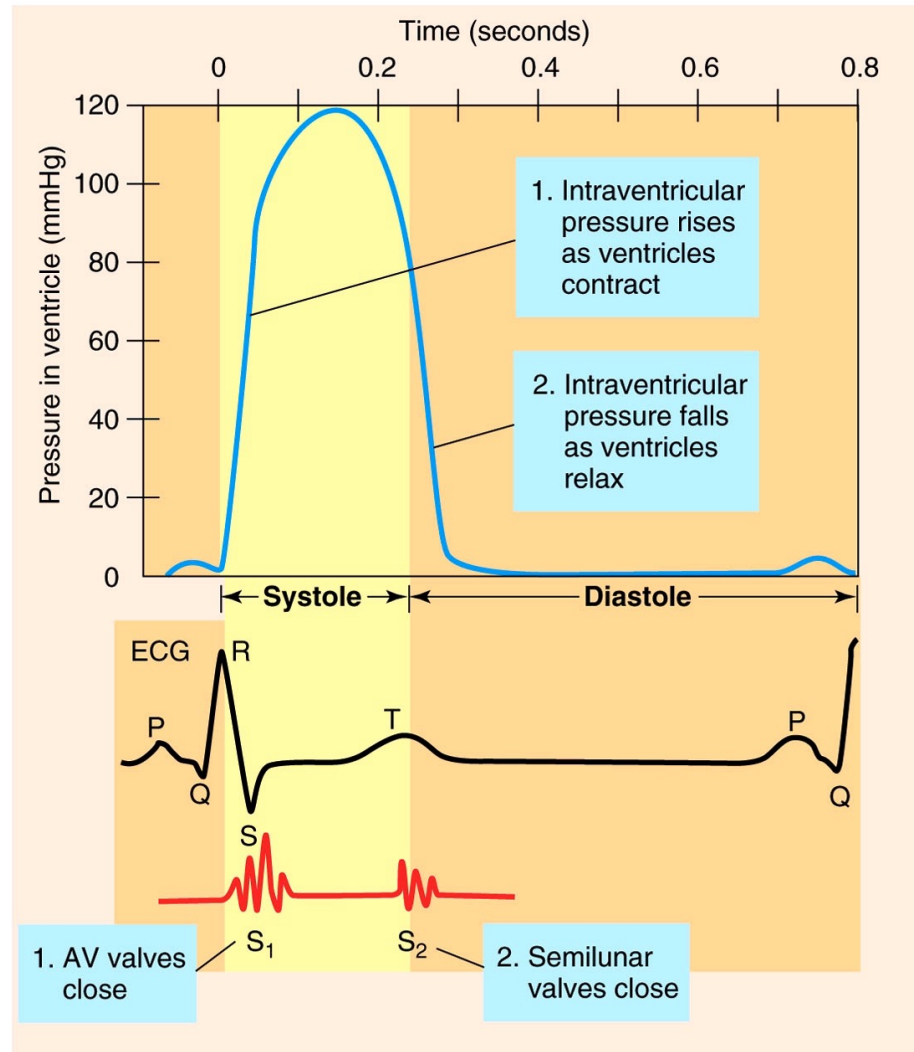


Figure 13.22

Figure 13.25



# Events of the Cardiac Cycle

Electrical	ECG	Atria	AV Valves	Ventricles	Semi-Lunar Valves	Blood Flow
between beats		diastole	open	diastole	closed	into atria (from vena cava, lungs)
SA node fires, spreads to AV node	P	systole	open	diastole	closed	into ventricles
spreads down bundle of His to Apex		systole	open	diastole	closed	
			<i>“lub”</i>			
spreads thru Purkinje fibers	QRS	diastole	closed	systole	open	into lungs, aorta
					<i>“dub”</i>	
between beats	T	diastole	open	diastole	closed	into atria (from vena cava, lungs)

*diastole = relaxed, systole = contracting*