

# Heart 1

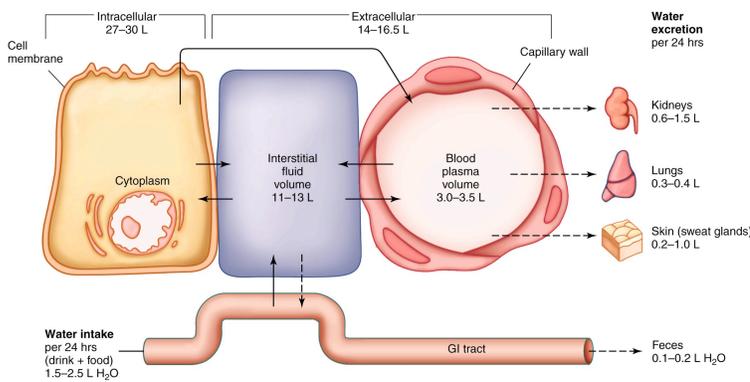
## Fox Chapter 13 part 1

(Chapter 12.6 Cardiac Muscle)

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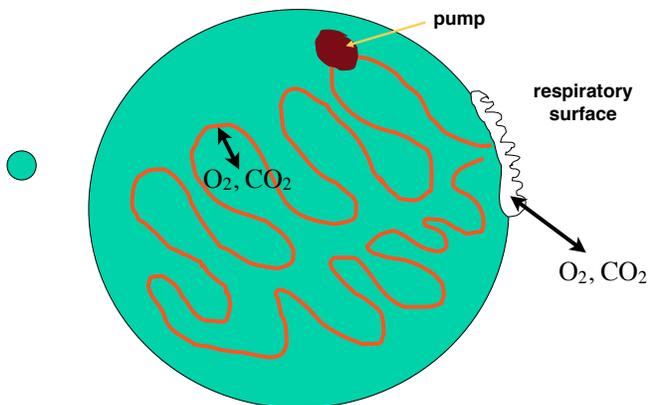
Figure 14.8



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### Circulatory System: Active Pumping

to transport gases from respiratory surface to tissues



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## 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)

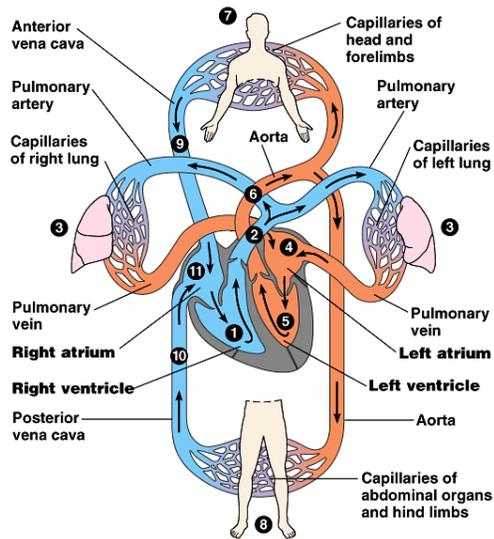
vessels carrying blood from capillaries back to heart. Very thin flabby walls with low pressure, but have one-way valves to prevent blood from backing up. Low in oxygen (except for pulmonary veins).

## Capillaries

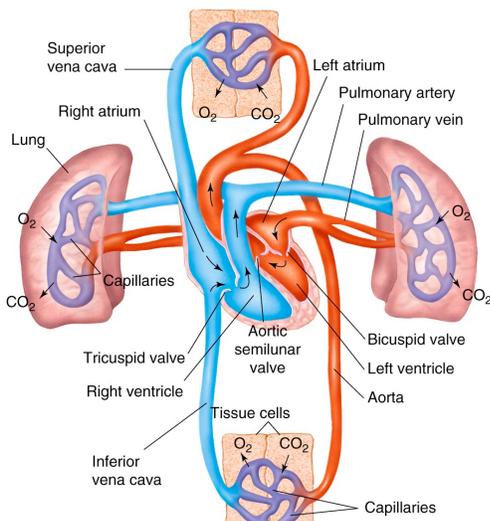
very small vessels (one blood cell wide) that perfuse all the tissues.

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Figure 42.4 The mammalian cardiovascular system: an overview



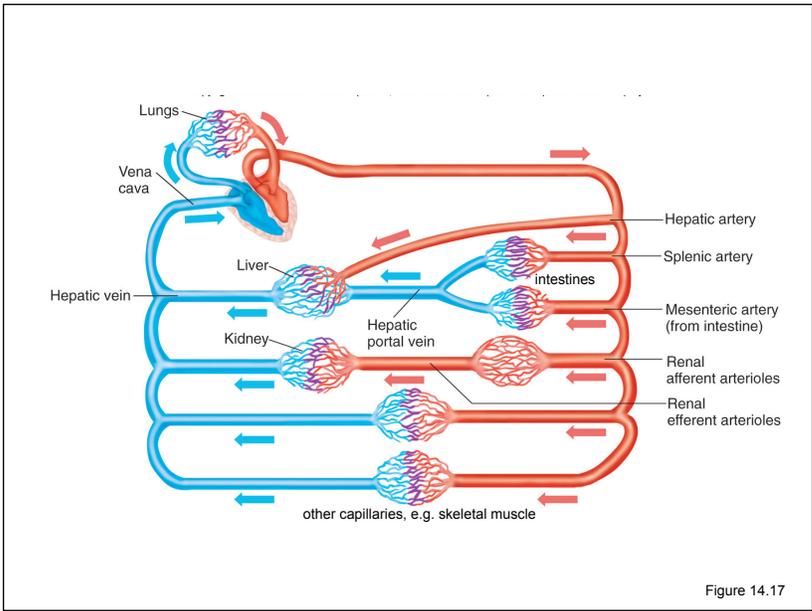
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remember: it's a circuit!

Figure 13.10

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### William Harvey, MD

1578-1657  
Physician to Kings James I and Charles I

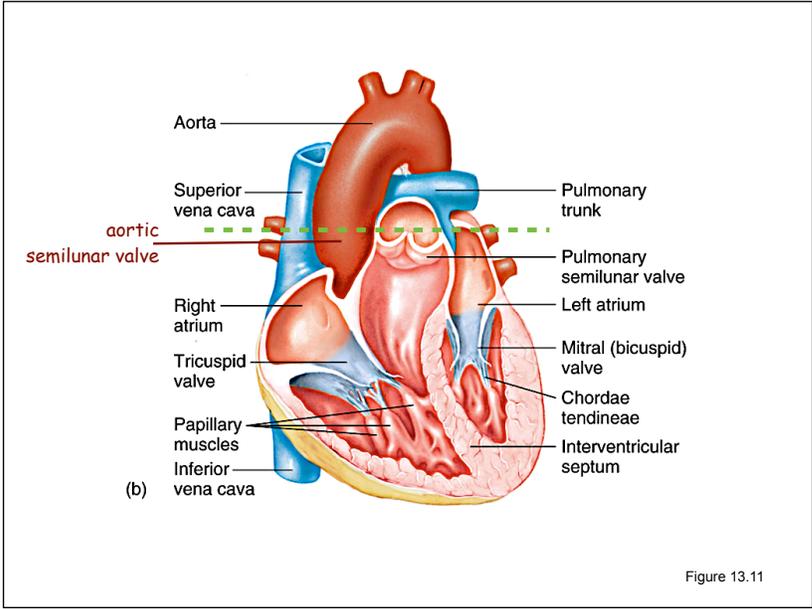
Author of *de Motu Cordis et Sanguinis*  
(An Anatomical Exercise on the Motion of the Heart and Blood in Living Beings), 1628

"This book is important both for the discovery of the complete circulation and for the experimental, quantitative and mechanistic methodology which Harvey introduced. He looked upon the heart, not as a mystical seat of the spirit and faculties, but as a pump analyzable along **mechanical lines**.

He observed that with each beat two ounces of blood leave the heart; so that with 72 heart beats per minute, the heart throws into the system 540 pounds of blood every hour. Where could all this blood come from? The answer seems to be that it is the same blood that is always returning. Moreover, the one-way valves in the heart, like those in the veins, indicate that ... the blood goes out to all parts of the body through the arteries and returns by way of the veins. The blood thus makes a **complete closed circuit**."

described pulmonary vs. systemic circulation, cardiac cycle, pacemaker cells, arteries vs. veins...(but couldn't see capillaries)

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aortic semilunar valves

pulmonary semilunar valves

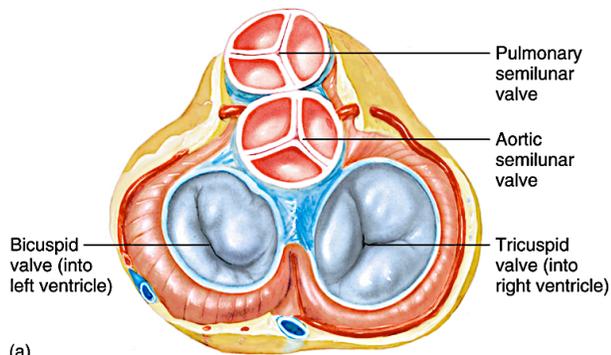


Igaku-Shoin, Ltd., Tokyo, Japan

Figure 13.12

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"dub" = closing of semilunar valves = S2



(a)

"lub" = closing of AV valves = S1

Figure 13.11

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## 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

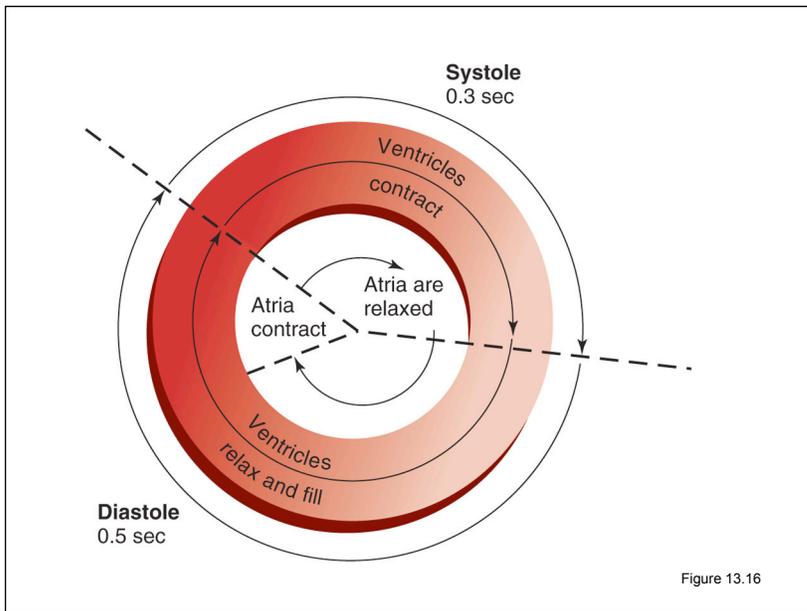
### Diastolic pressure (bottom number)

arterial pressure when ventricle is relaxed

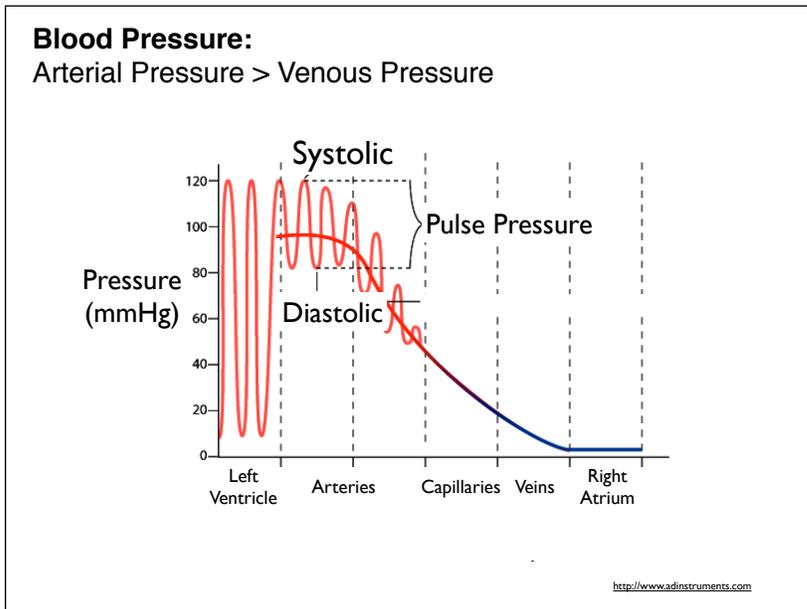
### Systolic pressure (top number)

arterial pressure when ventricle contracts and pumps

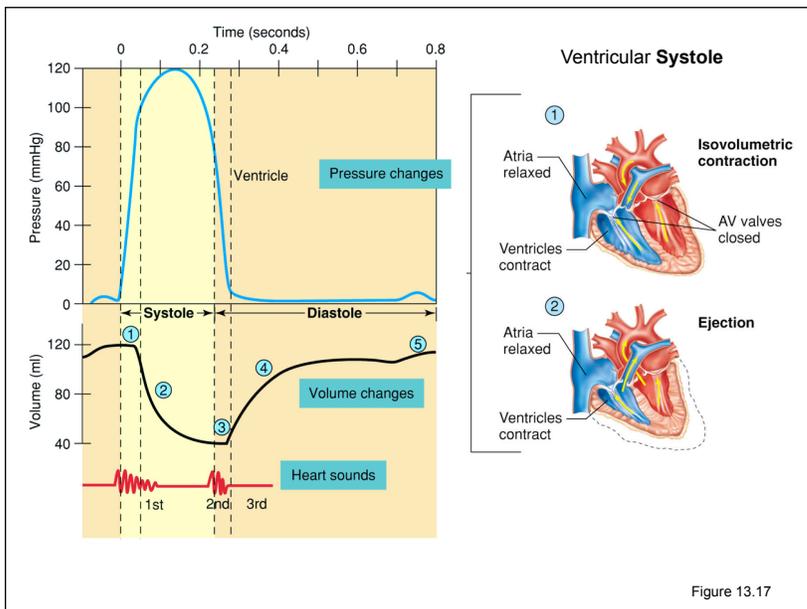
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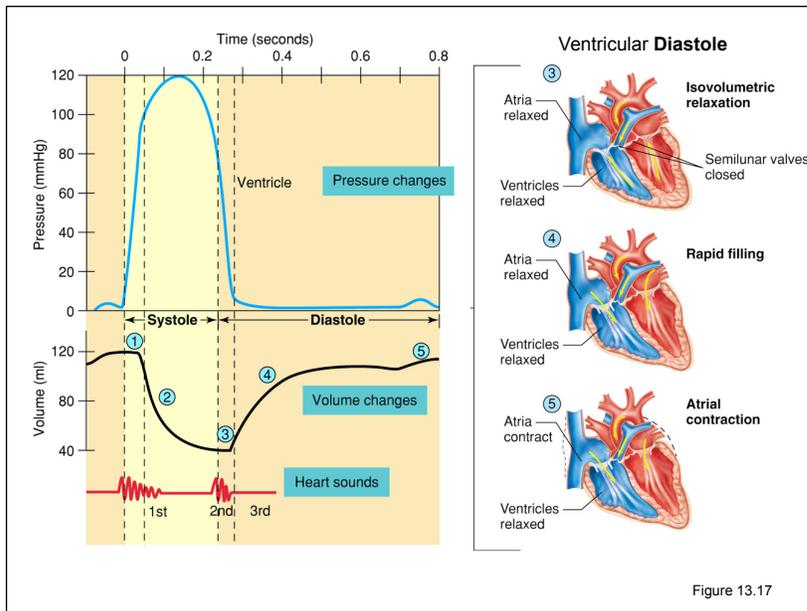
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### Events of the Cardiac Cycle

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 "lub" ↓	<b>systole</b>	open	into lungs, aorta
diastole	open	<b>diastole</b>	closed "dub" ↓	into atria (from vena cava, lungs)

*diastole = relaxed, systole = contracting*

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- ### 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

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## Contraction of Cardiac Muscle (Myocardium)

Action Potential starts from pacemaker cells in **sinoatrial node (SA node)**  
 HCN channels open when hyperpolarized (Hyperpolarization-activated Cyclic Nucleotide-gated channels)  
 -> spontaneous depolarization of pacemaker cells to -40 mV  
 -> opening of voltage-gated  $\text{Ca}^{++}$  channels  
 -> +20 mV -> action potential across myocardium

Myocardial Action Potential is longer than neural action potential:  
 fast  $\text{Na}^+$  channels -> fast depolarization  
 slow  $\text{Ca}^{++}$  channels -> plateau phase  
 voltage-gated  $\text{K}^+$  channels cause repolarization

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

### Myocardial Contraction

Voltage-gated  $\text{Na}^+$  channels open, causing depolarization  
 Voltage-gated  $\text{Ca}^{++}$  channels open in transverse tubules  
 Influx of  $\text{Ca}^{++}$  causes release of  $\text{Ca}^{++}$  from sarcoplasmic reticulum  
 $\text{Ca}^{++}$  binds to troponin to allow contraction  
 $\text{Ca}^{++}$  ATPase pump returns  $\text{Ca}^{++}$  into sarcoplasmic reticulum  
 $\text{Na}^+/\text{Ca}^{++}$  exchanger pumps  $\text{Ca}^{++}$  from cytoplasm into extracellular fluid

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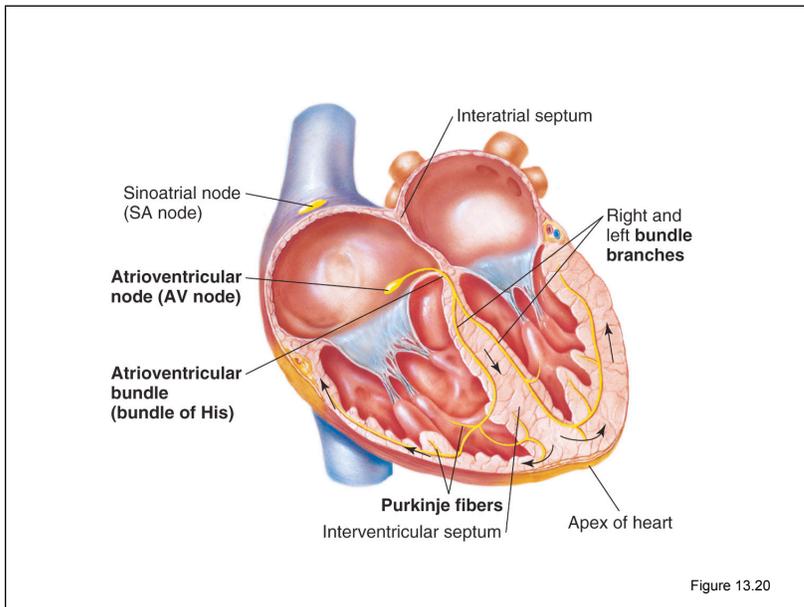


Figure 13.20

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## Spontaneous Depolarization of Pacemaker Cells

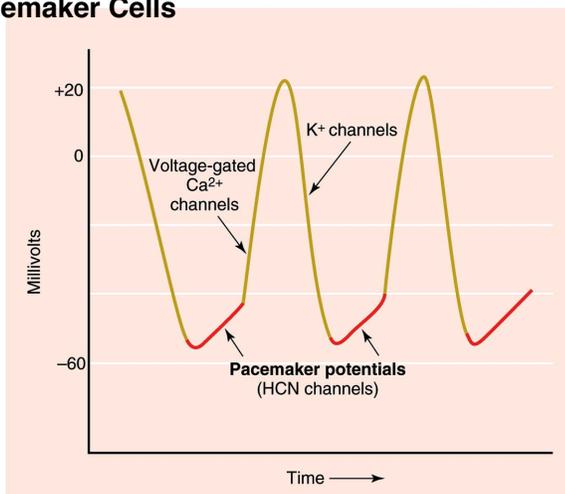
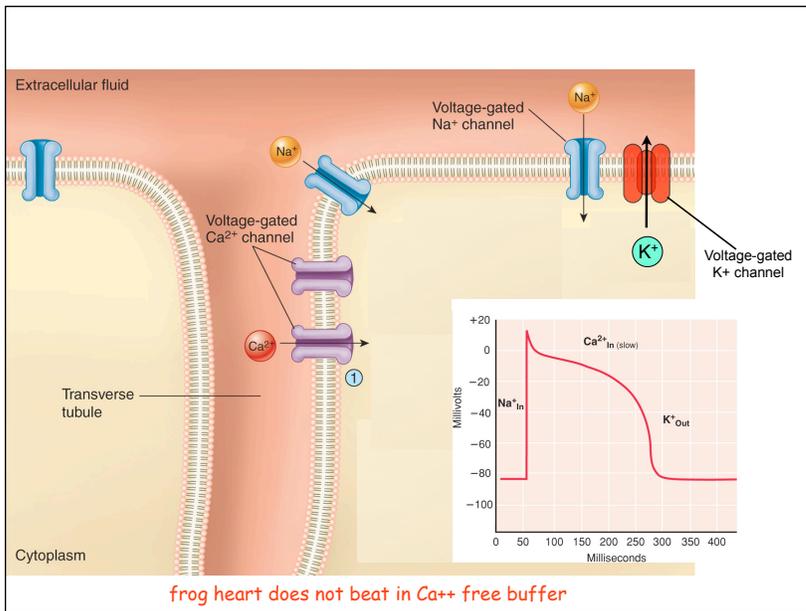


Figure 13.18

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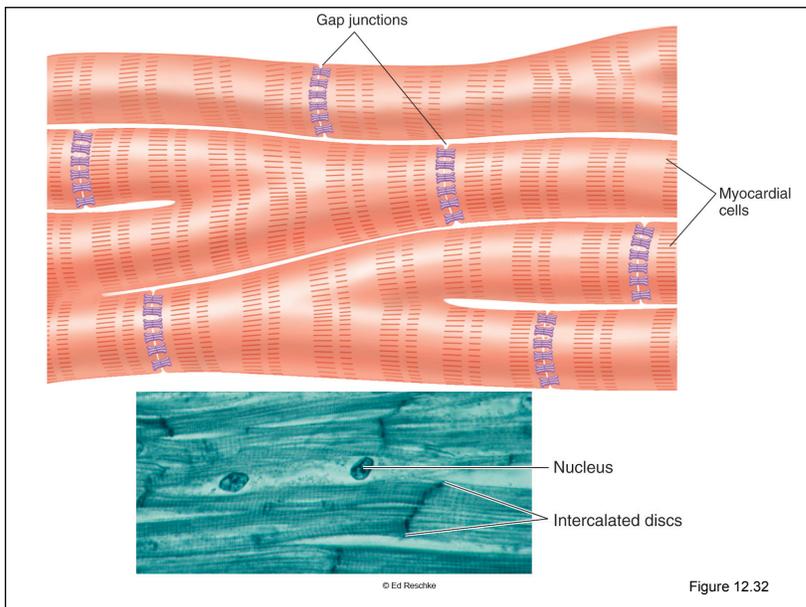
### Comparison of Skeletal Muscle and Cardiac Muscle

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
$\text{Ca}^{2+}$ released into cytoplasm from sarcoplasmic reticulum	$\text{Ca}^{2+}$ 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

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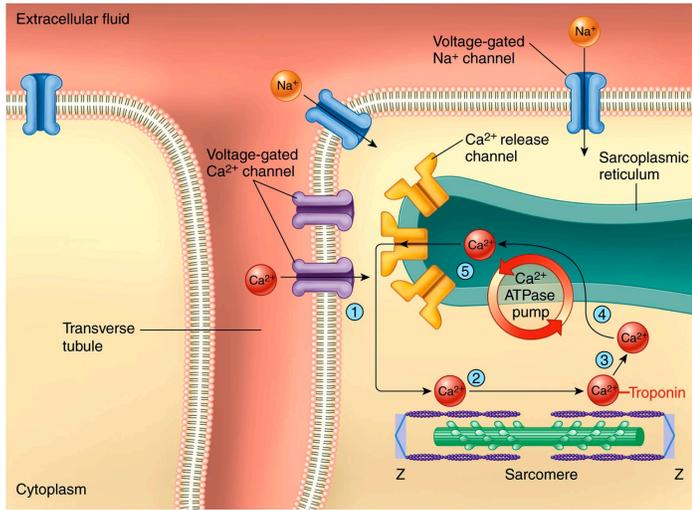


Figure 12.34

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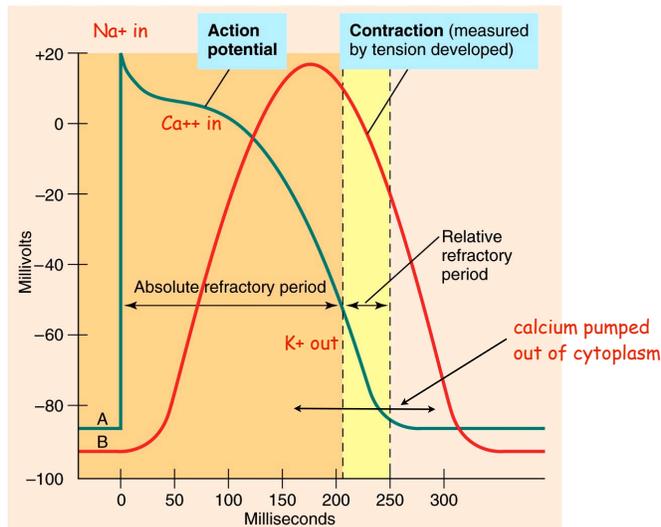


Figure 13.21

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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**]

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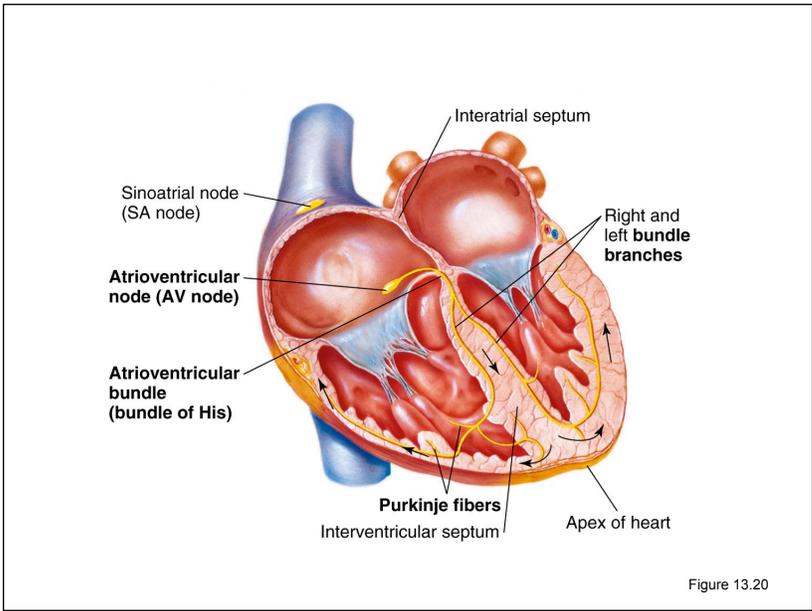
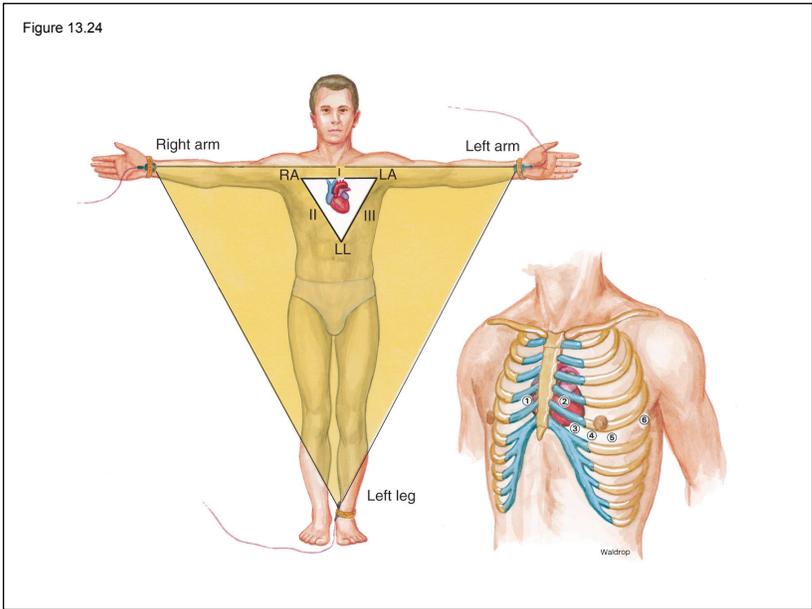
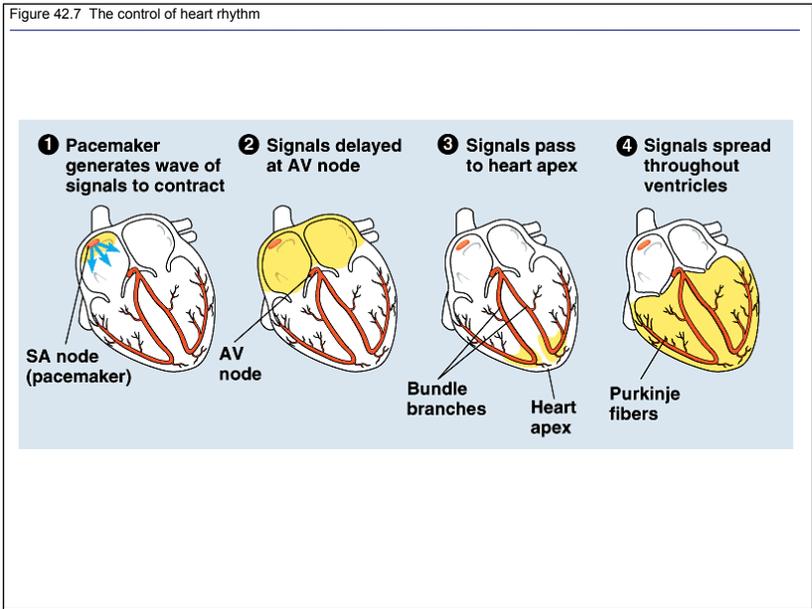
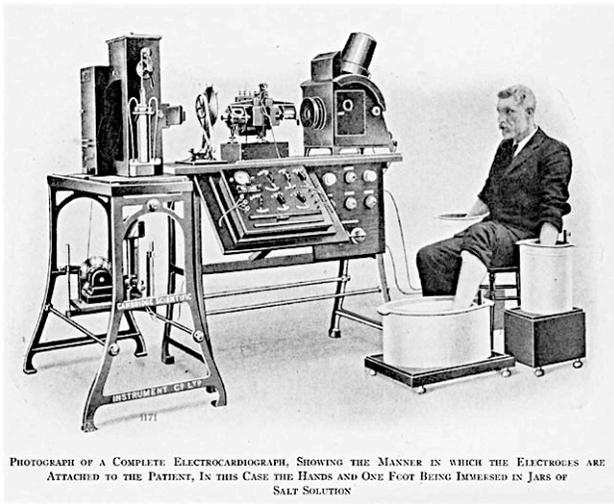


Figure 13.20



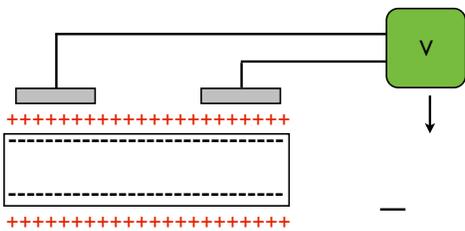


PHOTOGRAPH OF A COMPLETE ELECTROCARDIOGRAPH, SHOWING THE MANNER IN WHICH THE ELECTRODES ARE ATTACHED TO THE PATIENT, IN THIS CASE THE HANDS AND ONE FOOT BEING IMMESSED IN JARS OF SALT SOLUTION

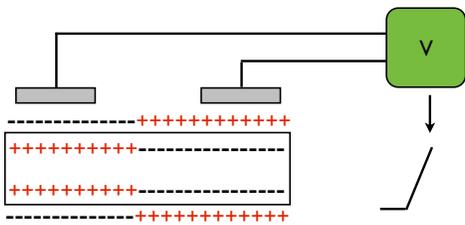
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[http://en.wikipedia.org/wiki/Image:Willem\\_Einthonen\\_ECG.jpg](http://en.wikipedia.org/wiki/Image:Willem_Einthonen_ECG.jpg)

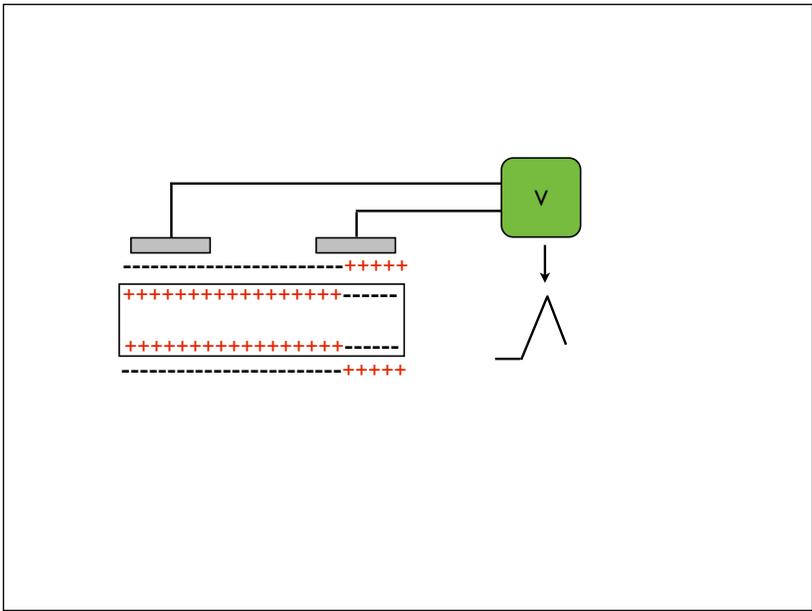
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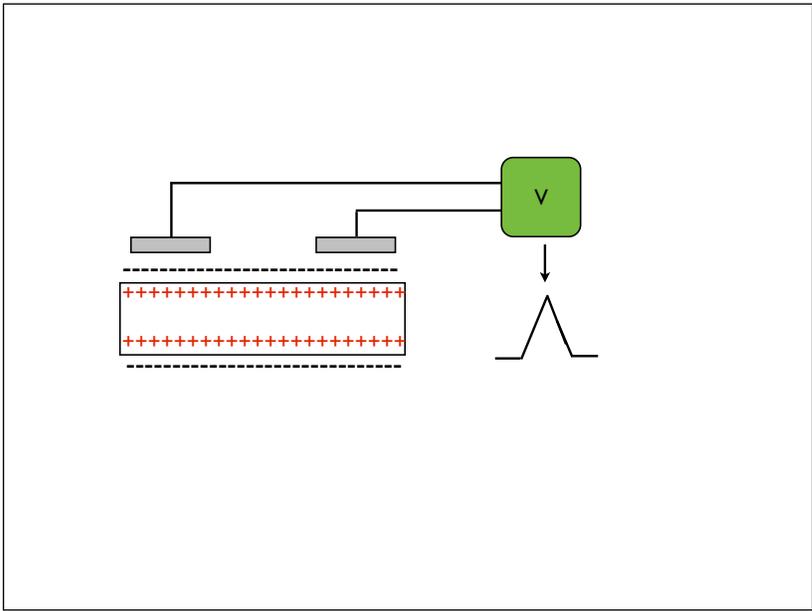
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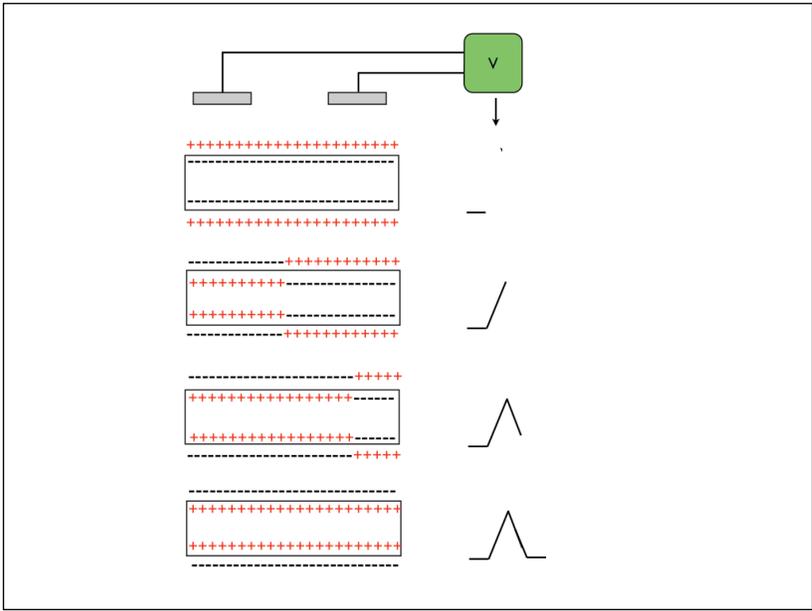
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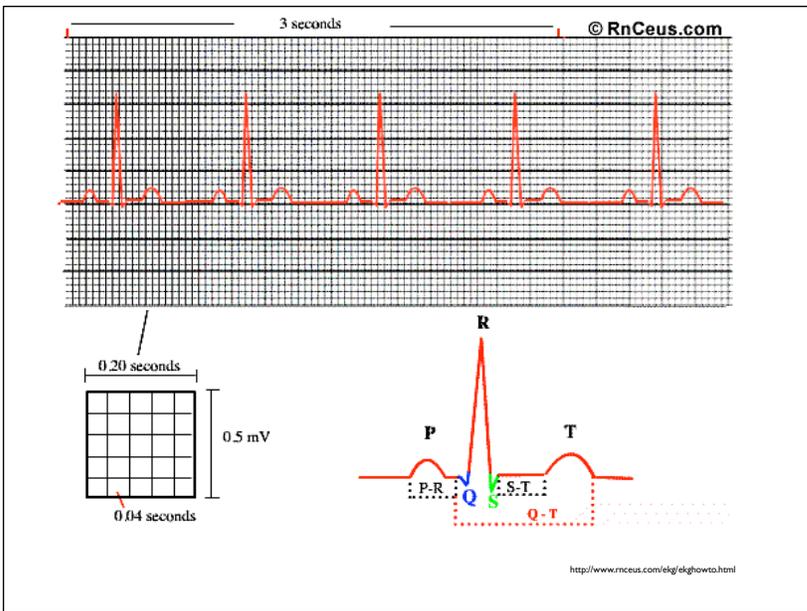
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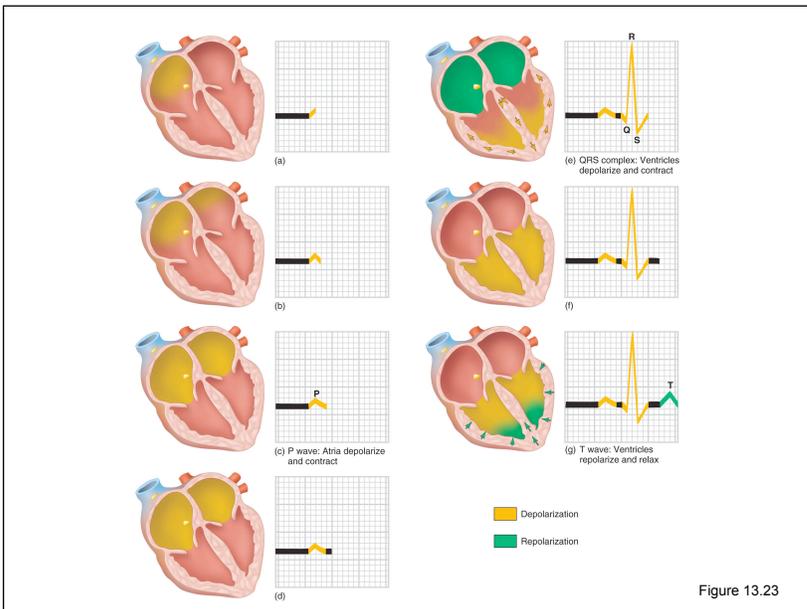
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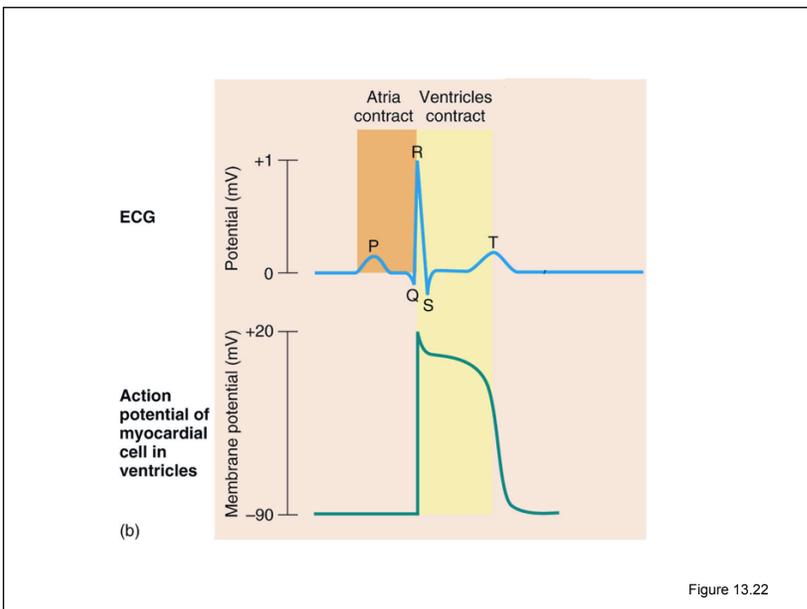
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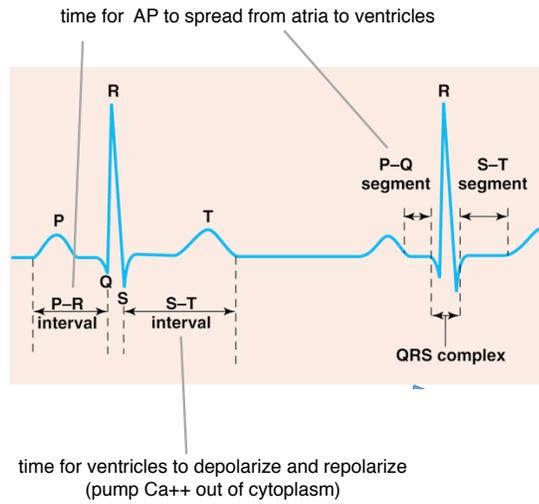
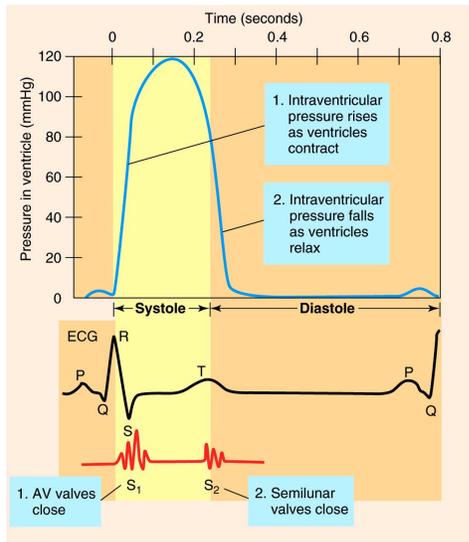


Figure 13.22

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Figure 13.25



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## 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	
			"lub"			
spreads thru Purkinje fibers	QRS	diastole	closed	systole	open	into lungs, aorta
					"dub"	
between beats	T	diastole	open	diastole	closed	into atria (from vena cava, lungs)

diastole = relaxed, systole = contracting

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