

# Cardiac Cycle

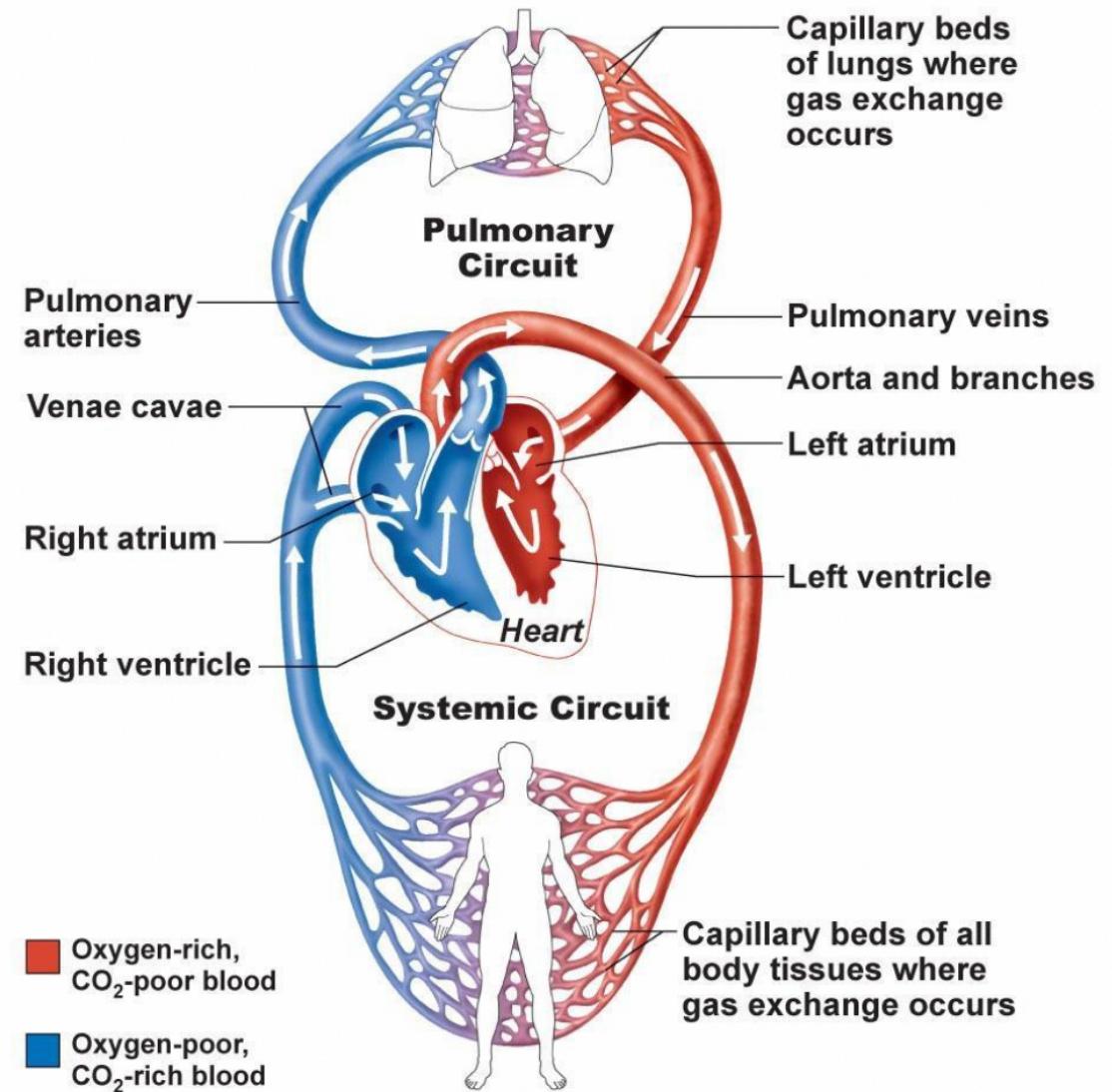
Central Circulation, Electrocardiogram  
(and Circulatory Regulation)

ZOOL 430

# Circulatory System

- Central Circulation:
  - Heart
- Peripheral Circulation:
  - Arteries
  - Veins
  - Capillaries

Mammalian 4-chambered heart



# *Myogenic Heart*

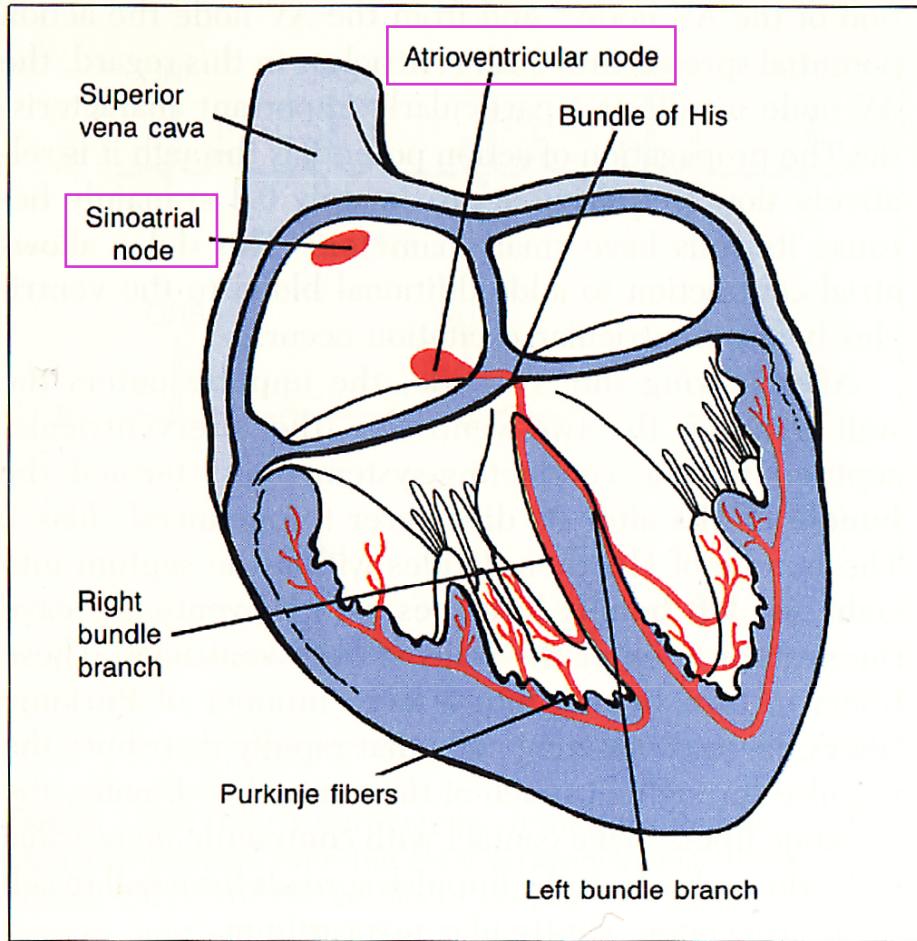
electrical signals

Pacemaker beats on its Own!

*Myogenic* = modified muscle cells

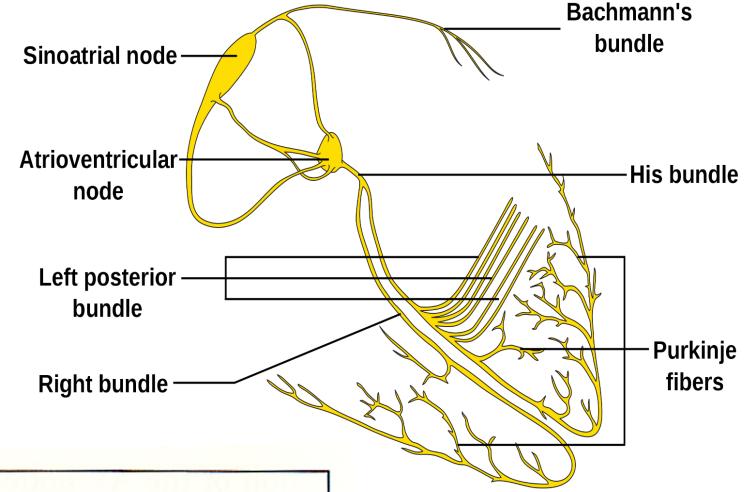
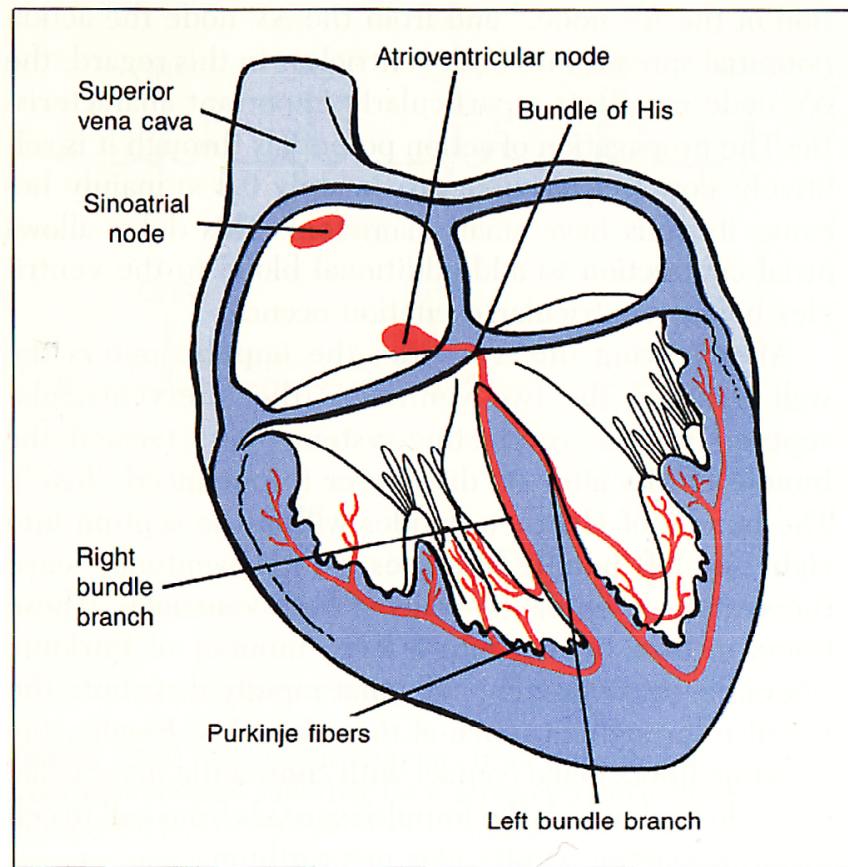
Found in two nodes

- Vertebrate Pacemaker Cells
  1. Sinoatrial (SA) node
  2. Atrioventricular (AV) node



# Pacemaker Cells

- Initiate cardiac action potential
- Separate from nervous system
- A natural pacemaker: setting up the rhythmic beating of the heart
- Transfers cardiac action potential:  
SA node → AV node → Bundle of His → Purkinje fibers



# What moves the heart?

- Pacemaker Cells

electrical signals

1. Sinoatrial (SA) node
2. Atrioventricular (AV) node

- Cardiomyocyte (Muscle cell)

- Cardiac muscle cells
- Conduction fibers: larger, connected by gap junctions

Allows flow of electrical signal between myocytes

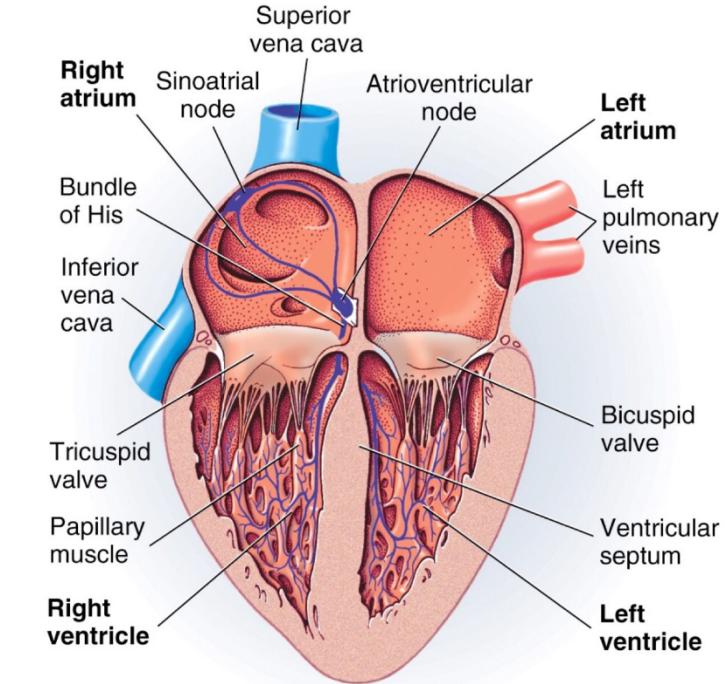
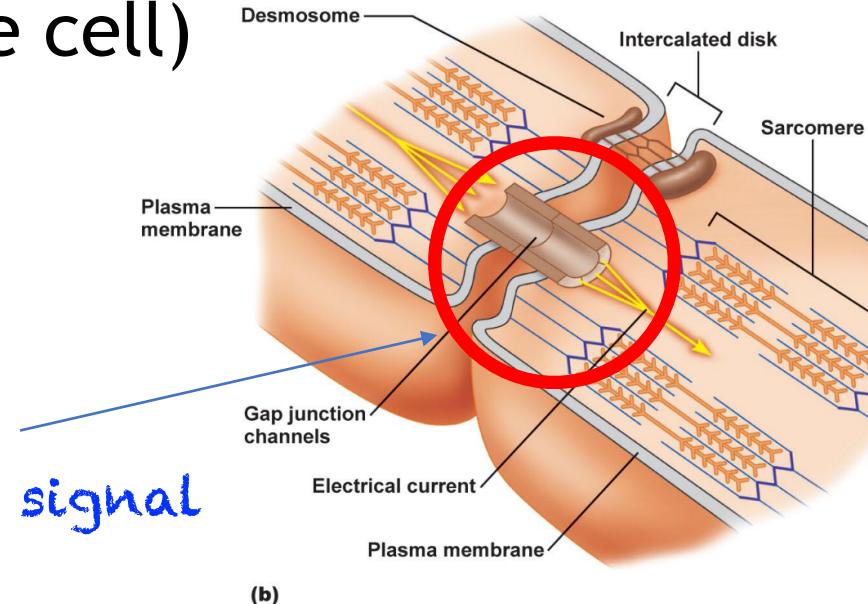
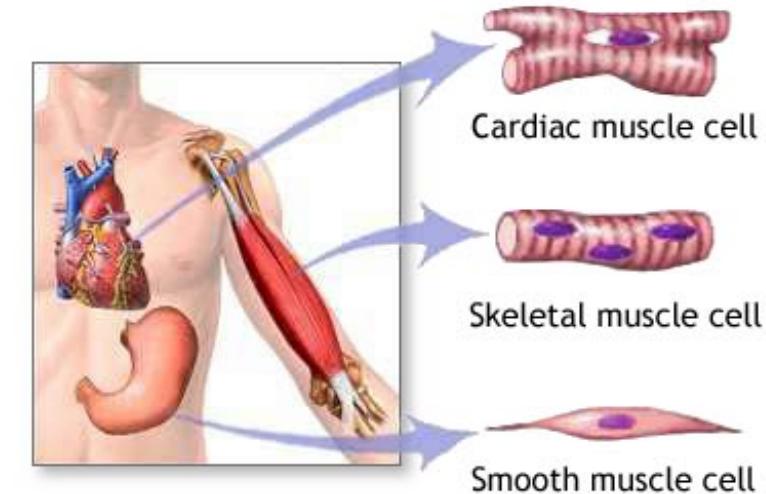


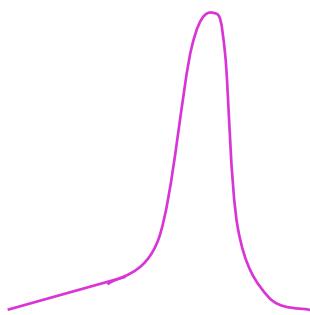
Figure 1. The 4 chambered heart



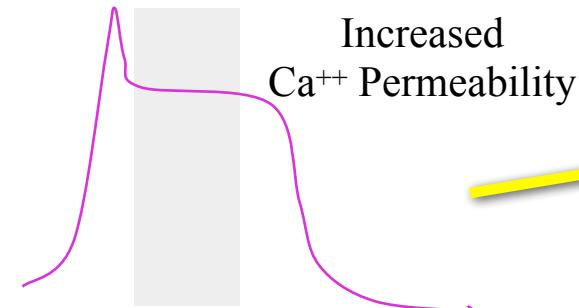
# Pacemaker and Cardiac Potentials

(special types of action potentials)

Pacemaker Potential

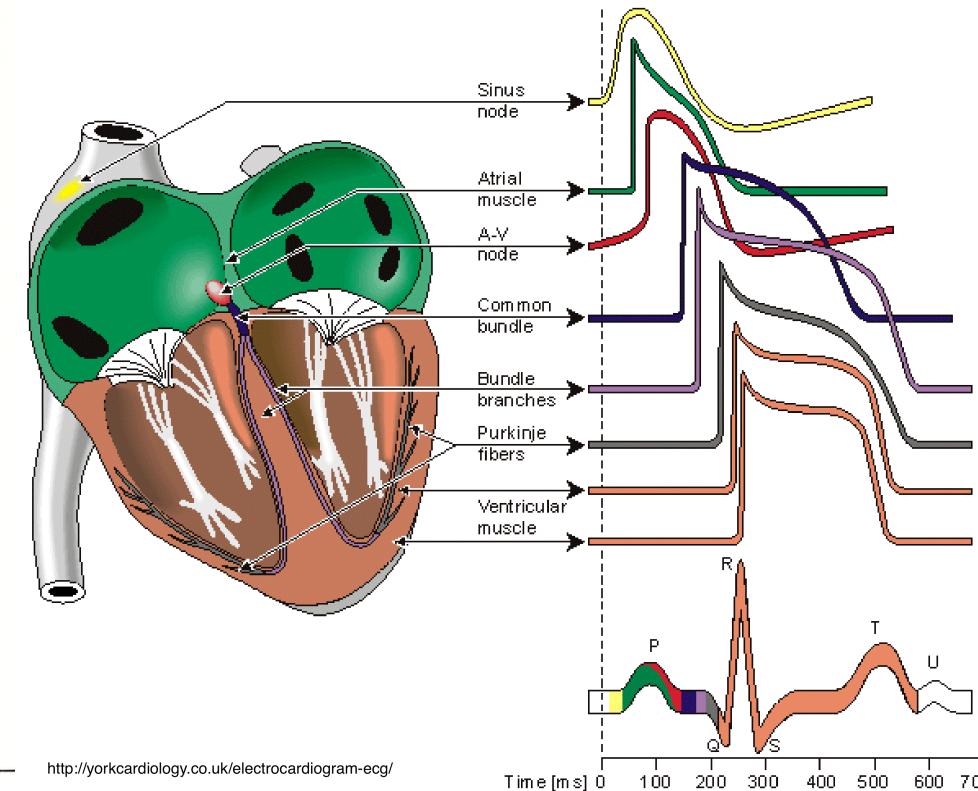
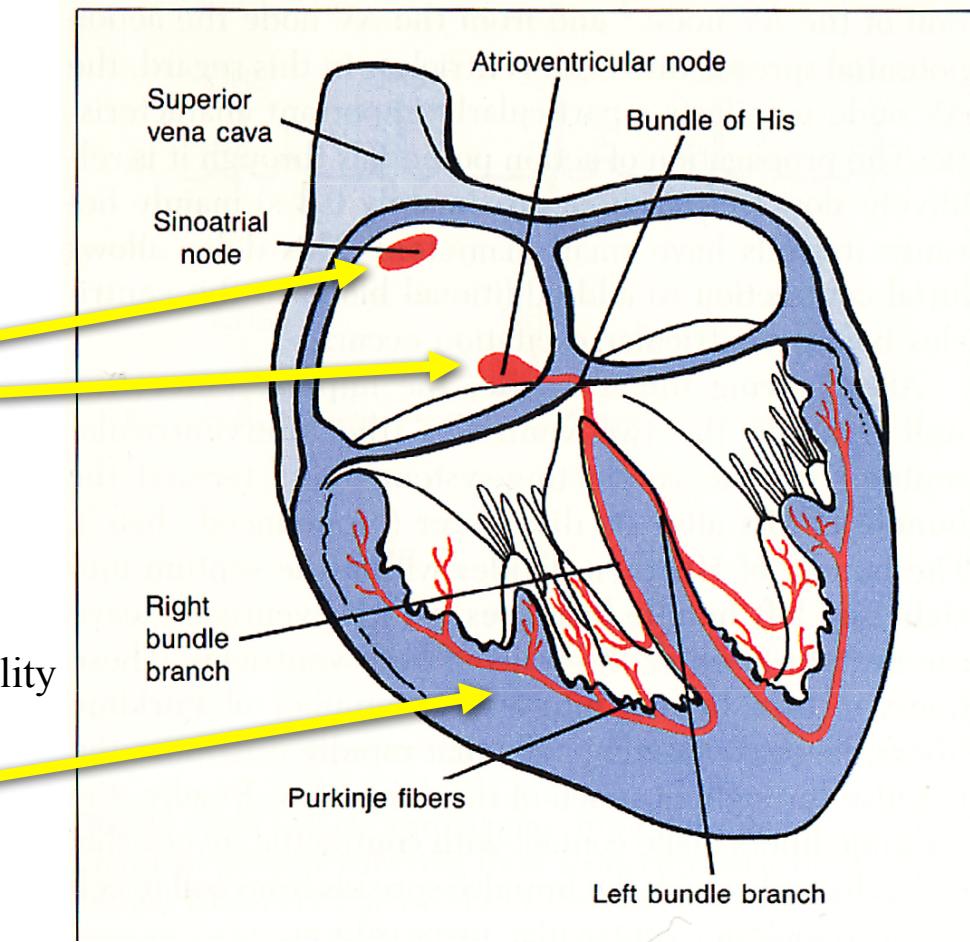


Cardiac Potential



Prolonged Potential

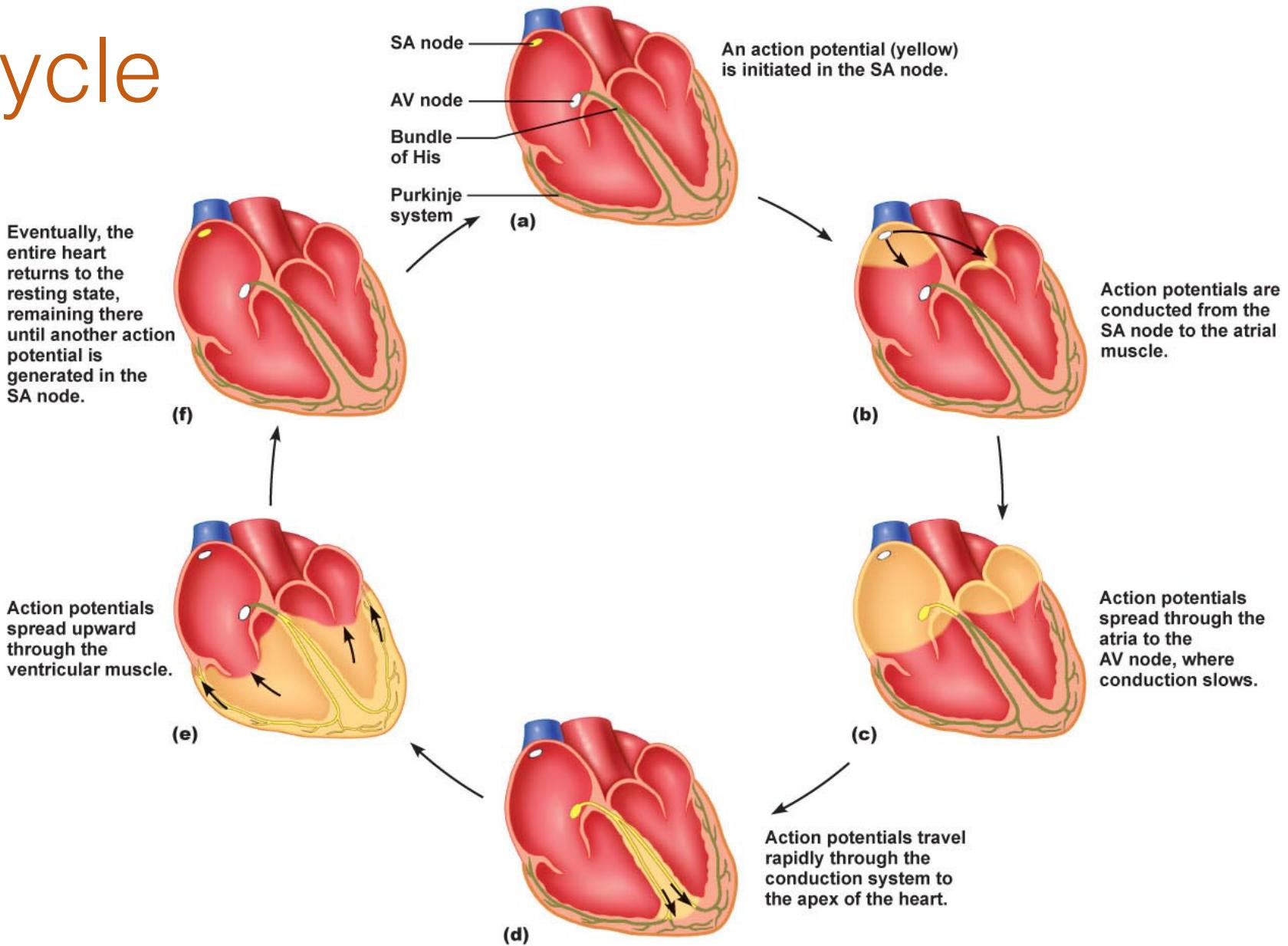
Synchronizes Contraction



<http://yorkcardiology.co.uk/electrocardiogram-ecg/>

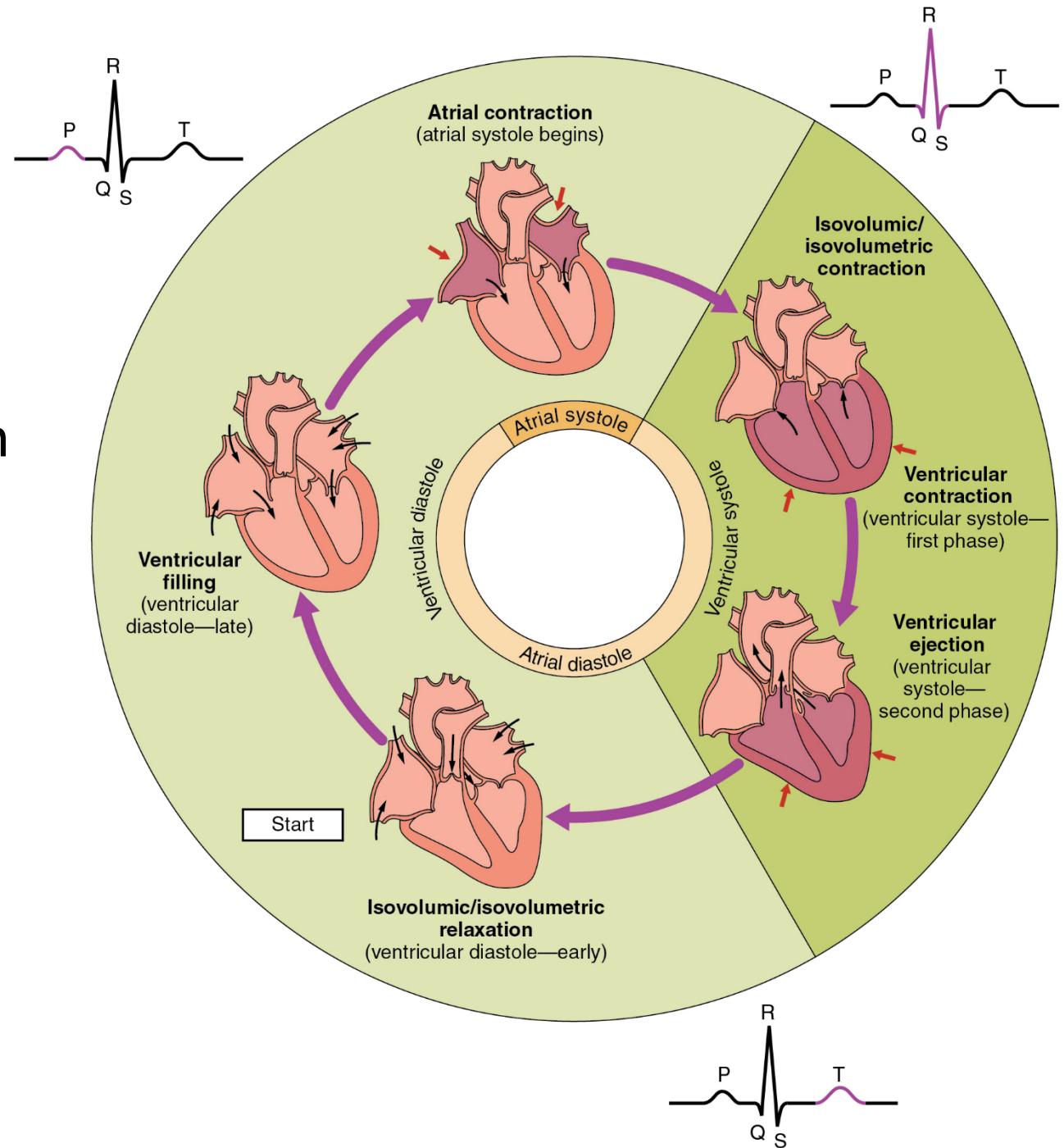
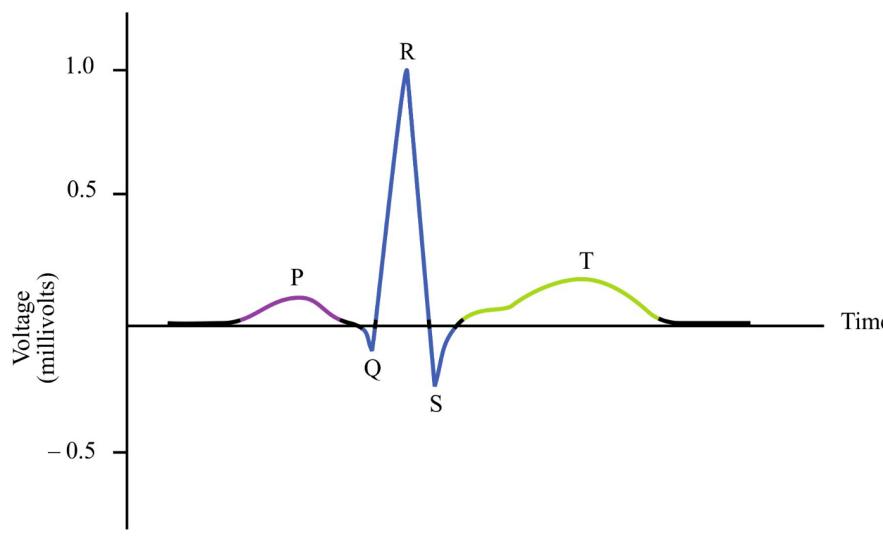
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# Cardiac Cycle



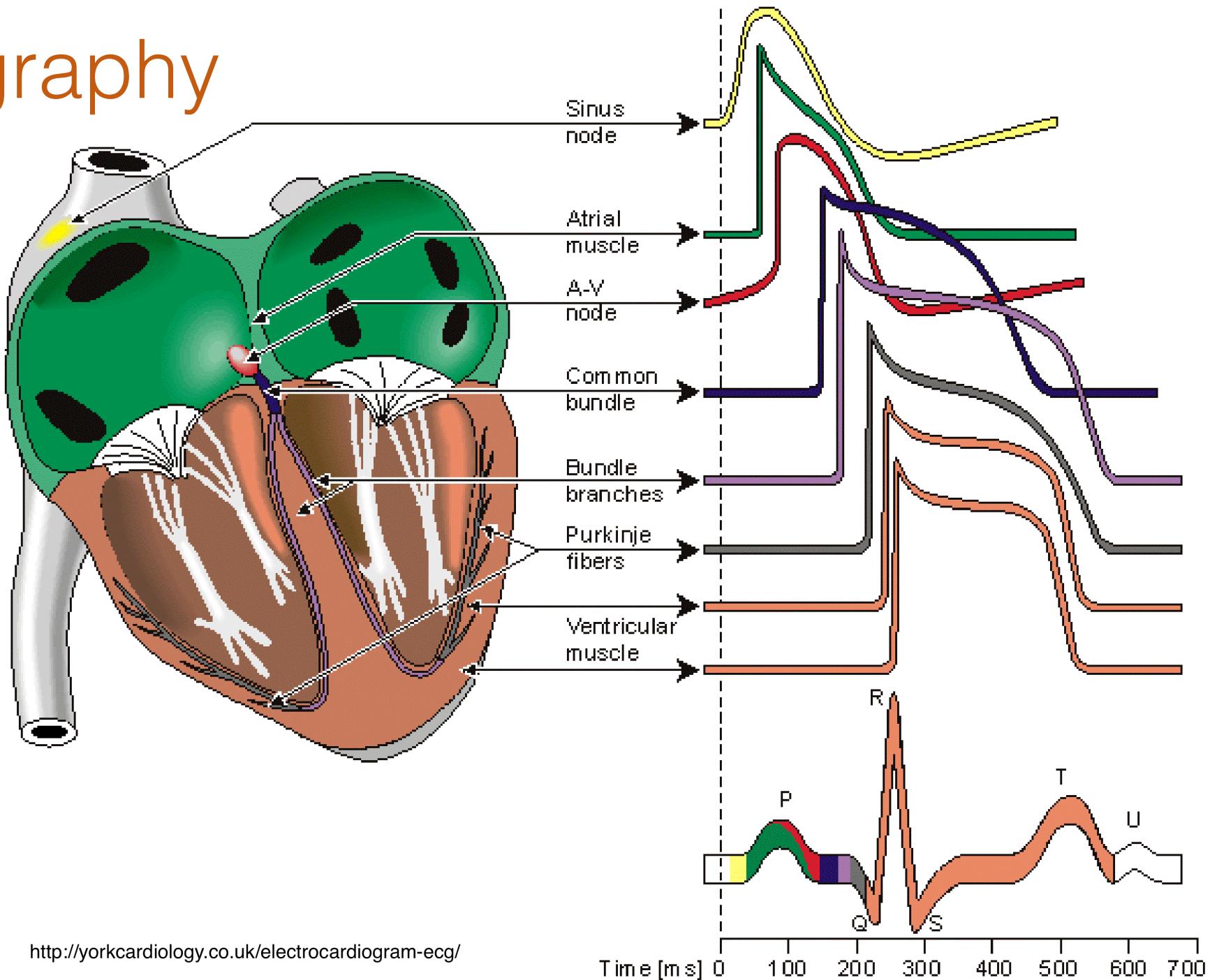
# Electrocardiography

- Measure electrical changes caused by cardiac AP using electrodes attached to the surface of the skin
- Signals detected would be the combination of atrial and ventricular activities.



# Electrocardiography

EKG's reveal the sum of all electrical potentials (pacemaker, cardiac), across the heart during a cardiac cycle



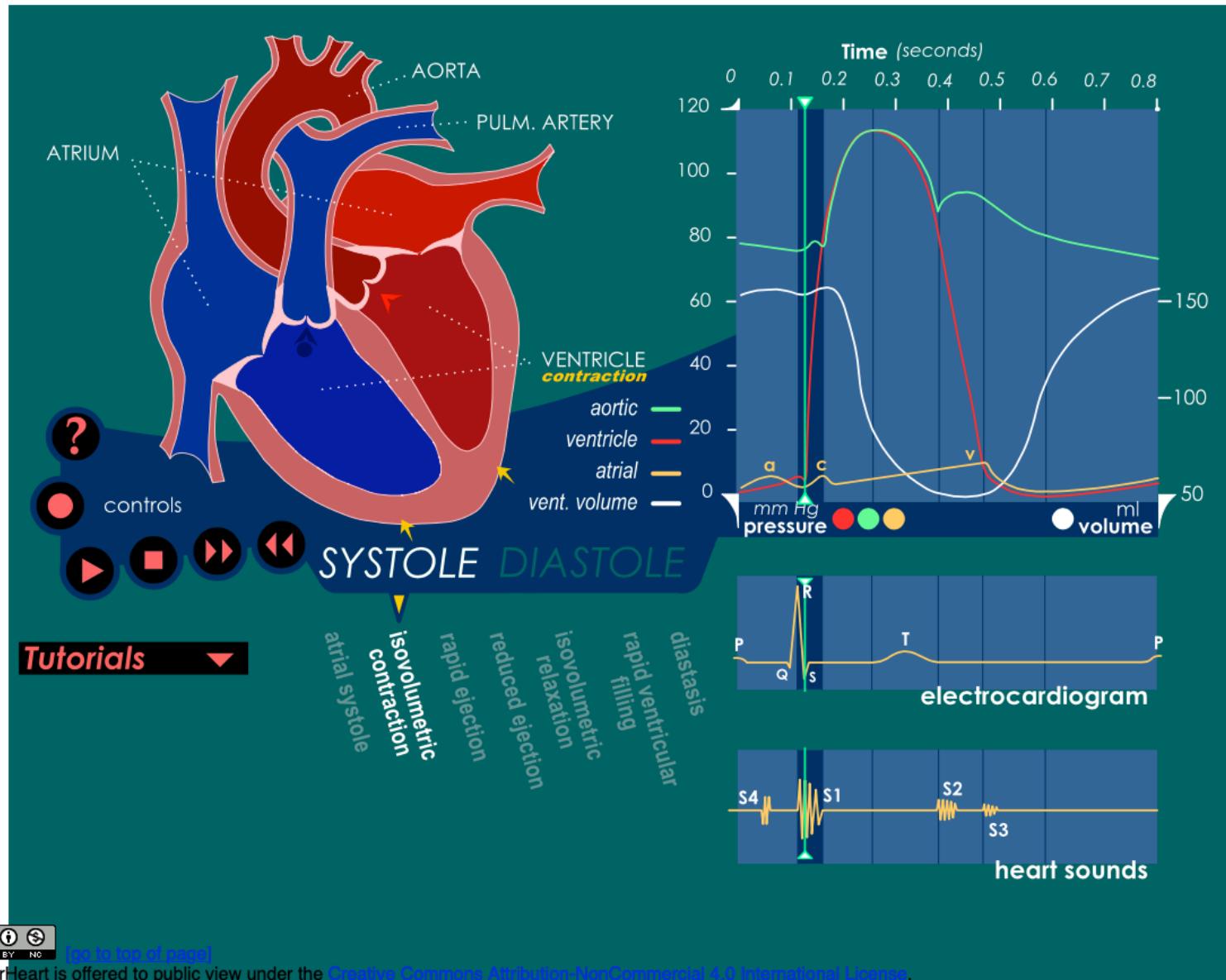
# HyperHeart

Interactive animation updated from Flash to HTML 5 by Quentin Roper of [Massey University, New Zealand](#). It is offered to public view under the Creative Commons commercial re-use with required attribution license ([see below](#)).

[Click here](#) to position animation.

[Spencer S. Eccles Health Sciences Library home](#) | [Knowledge Weavers projects](#)

<https://library.med.utah.edu/kw/pharm/hyperheart/>



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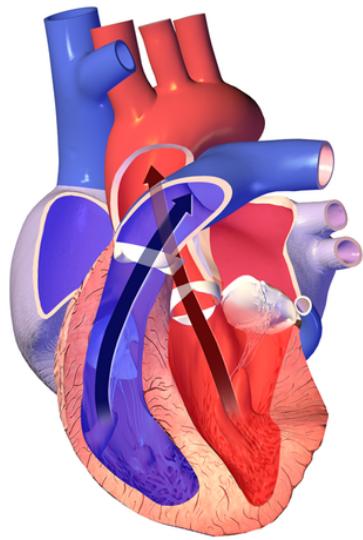


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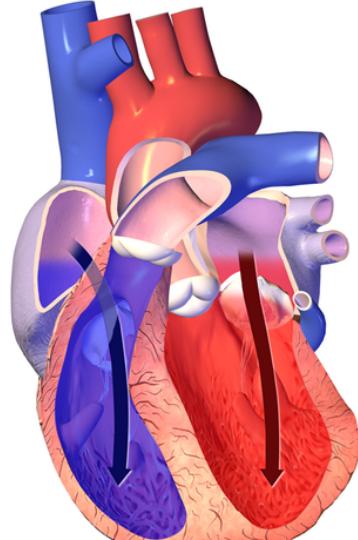
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# Cardiac Cycle

The performance of the heart from the start of one heartbeat to the next



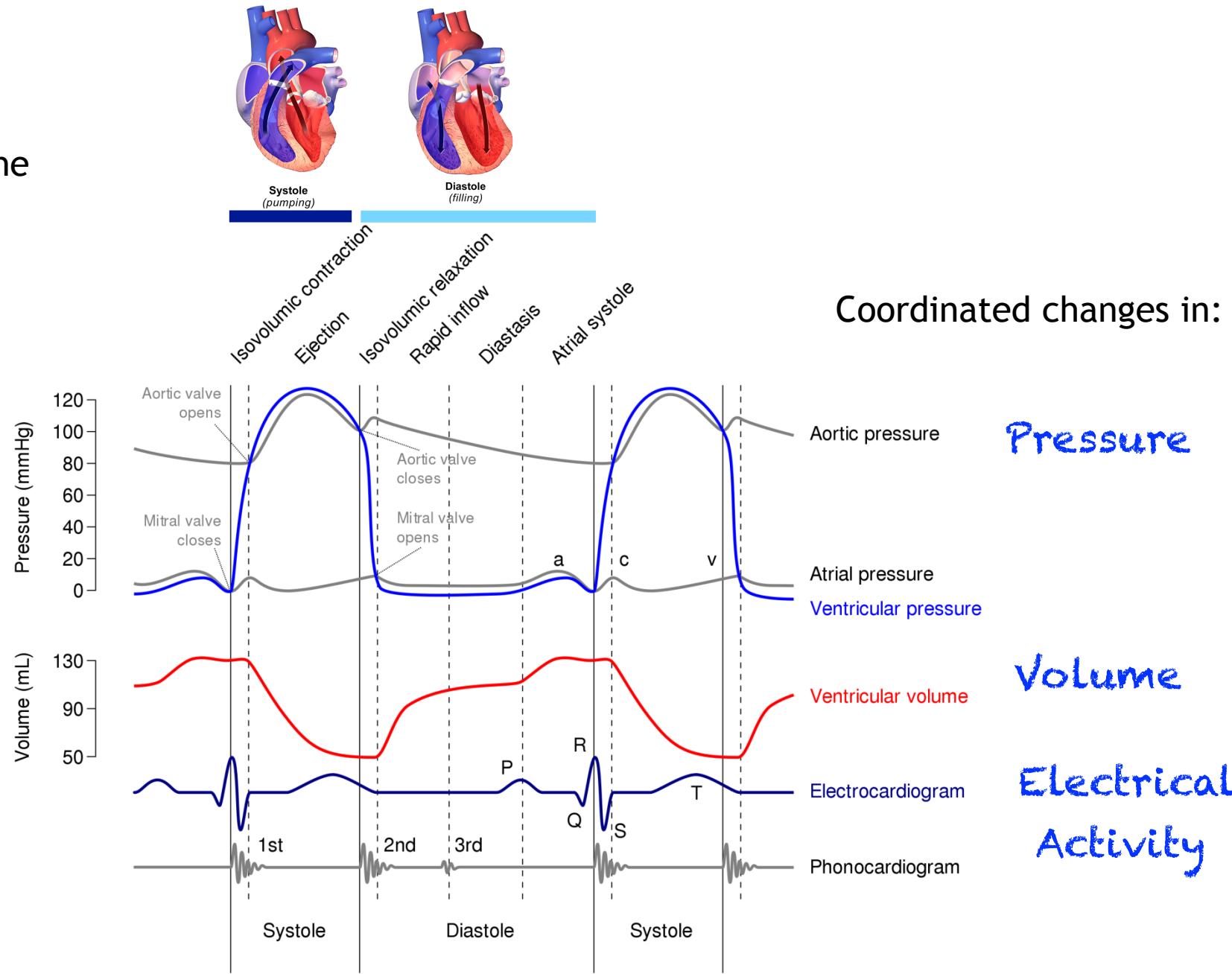
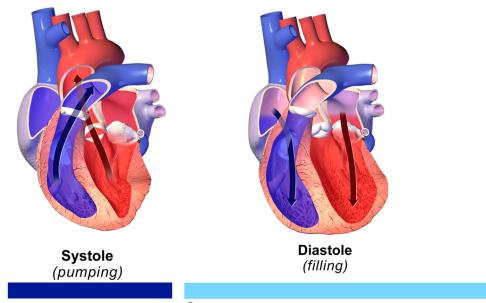
Systole  
(pumping)



Diastole  
(filling)

Ventricular  
Contraction

Ventricular  
Relaxation

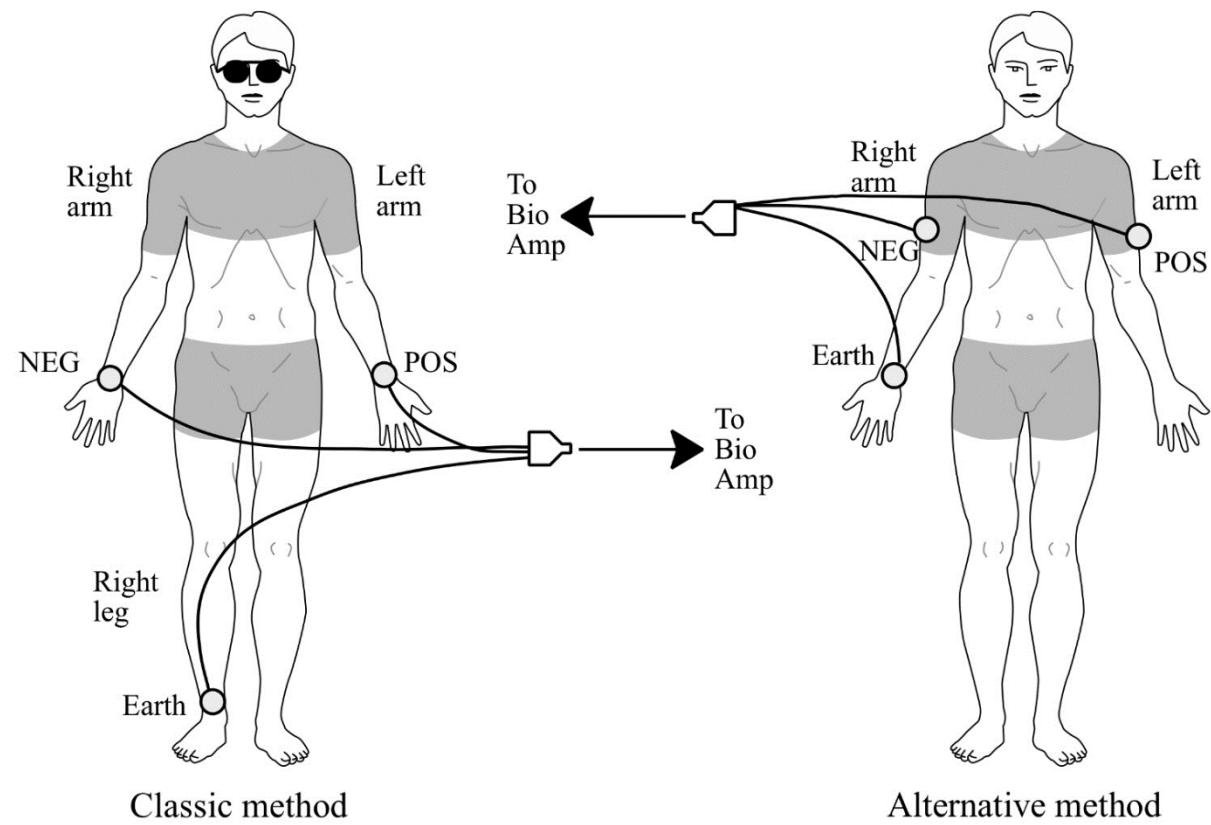


# Today's Lab Electrocardiogram



# Experiment

- What you need: PowerLab, BioAmp, electrodes, Push Button
- Make sure the colors or PowerLab and BioAmp match
- Remove any jewelry/watch
- Fill the electrode cups with conducting gel. Make sure there is no air between electrode and skin!
- Tape the electrode securely.



# Starling's Law of the Heart

In the early 1900s, Ernest Starling demonstrated that the *energy of cardiac contraction is proportional to the initial length of the cardiac muscle fibers.*

More stretch → More SV!

Allows for a passive (but dynamic!) ability of the heart to adjust contraction to meet demand

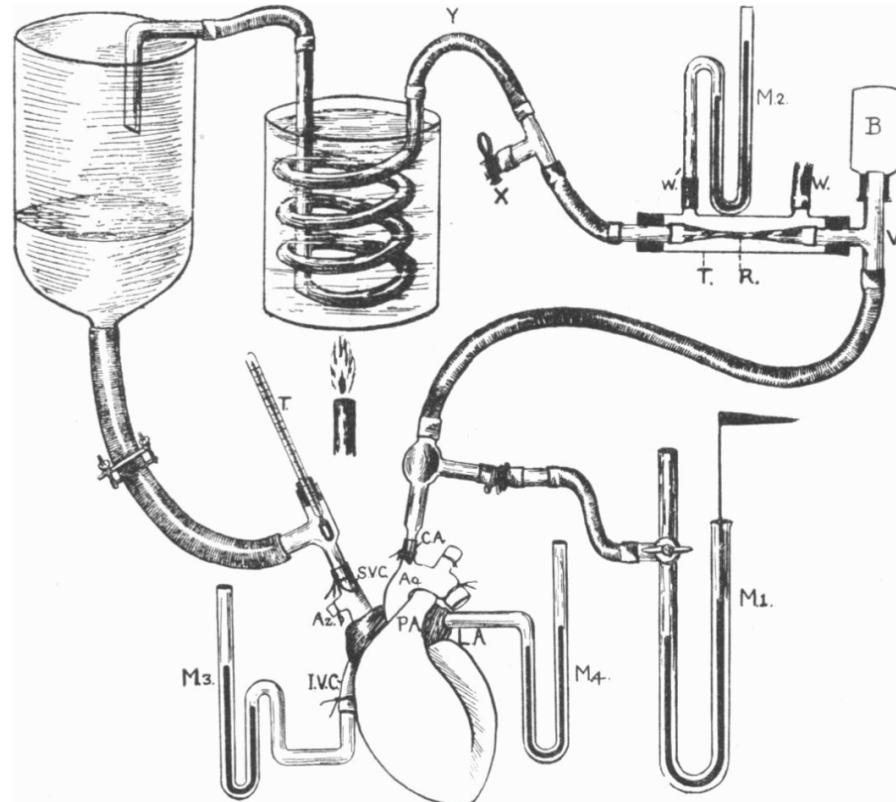


Fig. 1.

Figure 1 Starling's heart-lung preparation. The coronary and pulmonary circulations are left intact and the lungs are not shown. Image reproduced with permission from [1].

\*as the heart fills up with more blood during DIASTOLE → it contracts harder and pumps out more blood during SYSTOLE

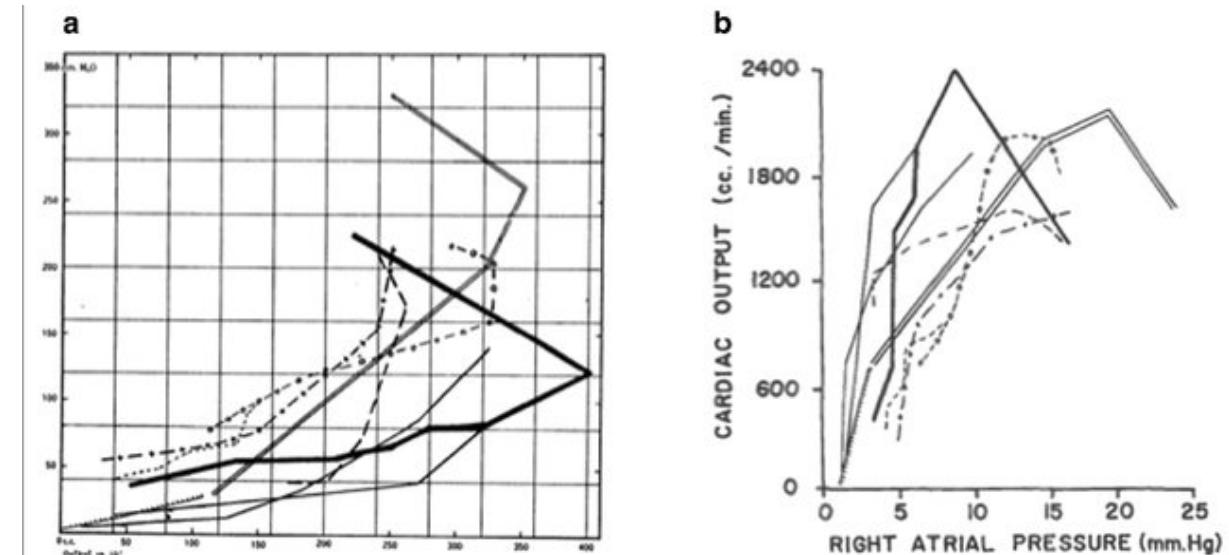
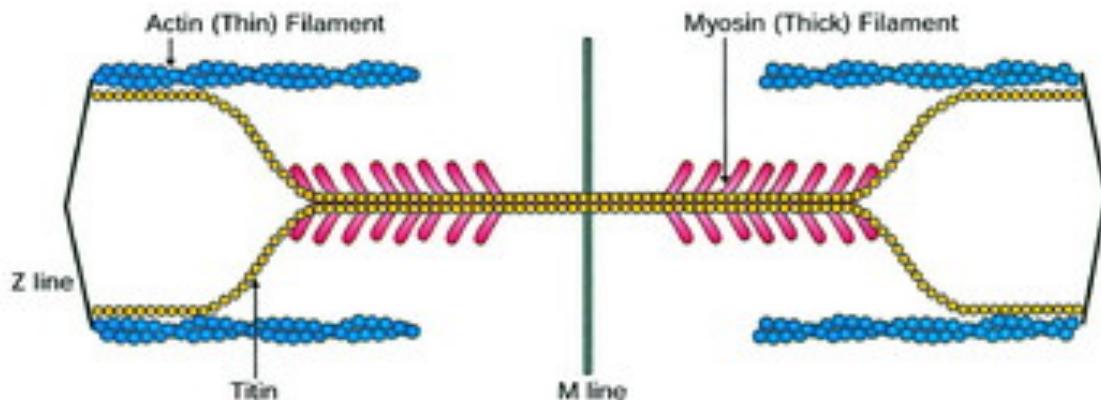
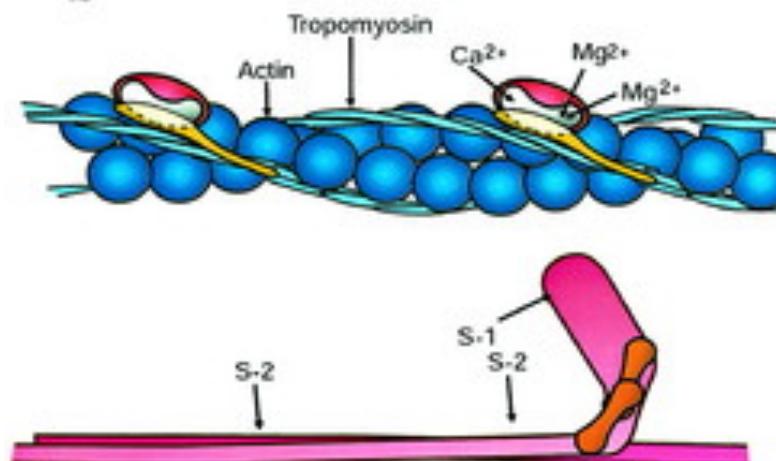


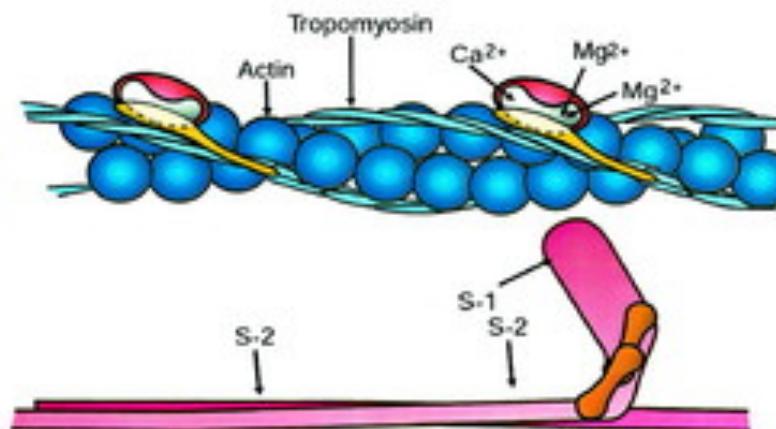
Figure 2 Original presentation and reproduction of the Starling curve. (a) Original presentation of the Starling curve. The y axis is right atrial pressure (mmH<sub>2</sub>O). The x axis is cardiac output (cm<sup>3</sup>/minute). Image reproduced with permission from [1]. (b) Reproduction in Guyton and colleagues' textbook on circulatory physiology. Image reproduced with permission from [9].

**A**

Really cool, but mechanism?

**B**

*Short Sarcomere Length*



*Long Sarcomere Length*

Figure 1. Myocardial sarcomere and myofilaments.

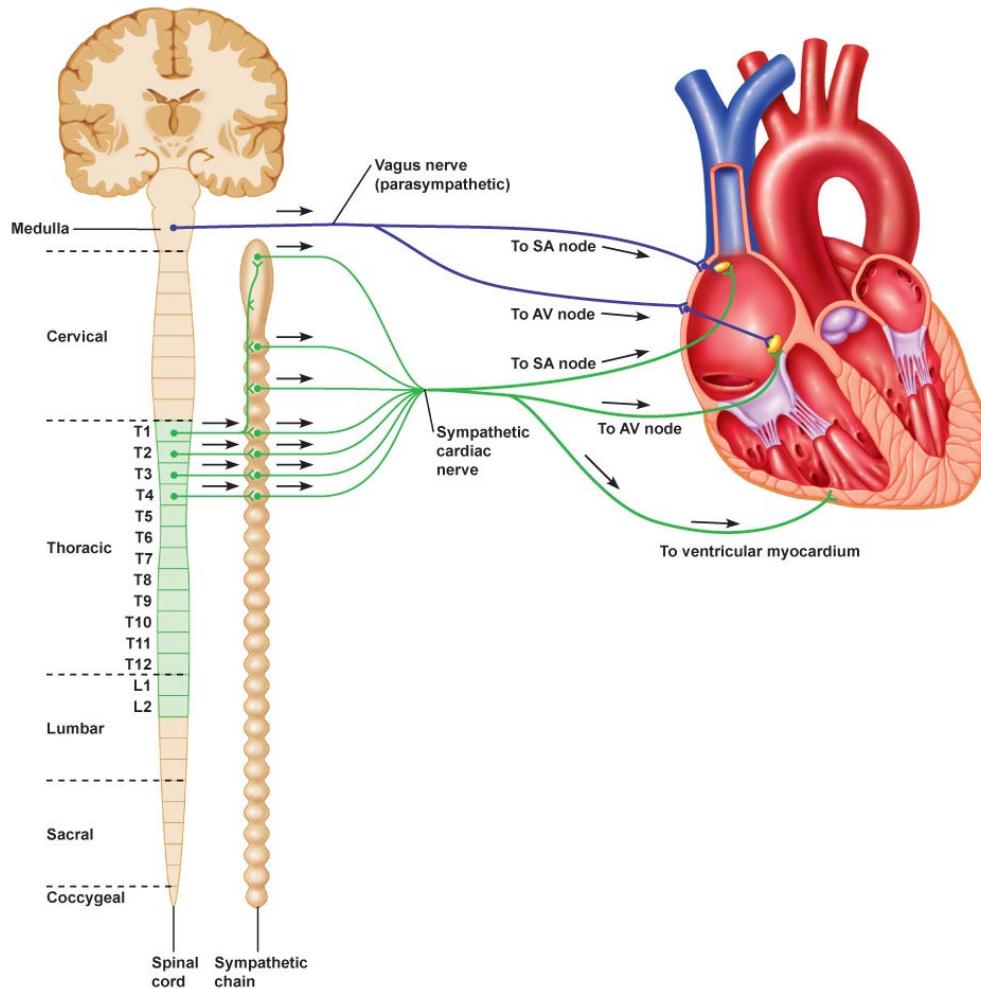
A, Diagram of the sarcomere showing approximate **spatial relationships of thick and thin filaments and putative interactions of titin with the filaments, which would give rise to radial and axial restorative forces when the sarcomere is stretched.**<sup>11</sup>

B, Diagram of the thick and thin filaments illustrating the decrease in lateral separation at long lengths. The probability of crossbridge interaction increases at long lengths due to closer proximity to actin.



# Regulation of Heart Rate

## Nervous System



Parasympathetic & Vagus Nerve  
Ach -> SA node

Sympathetic  
Epinephrine

Norepinephrine (synthesized from  
Dopamine) increases blood pressure

# *Pacemaker Potentials*

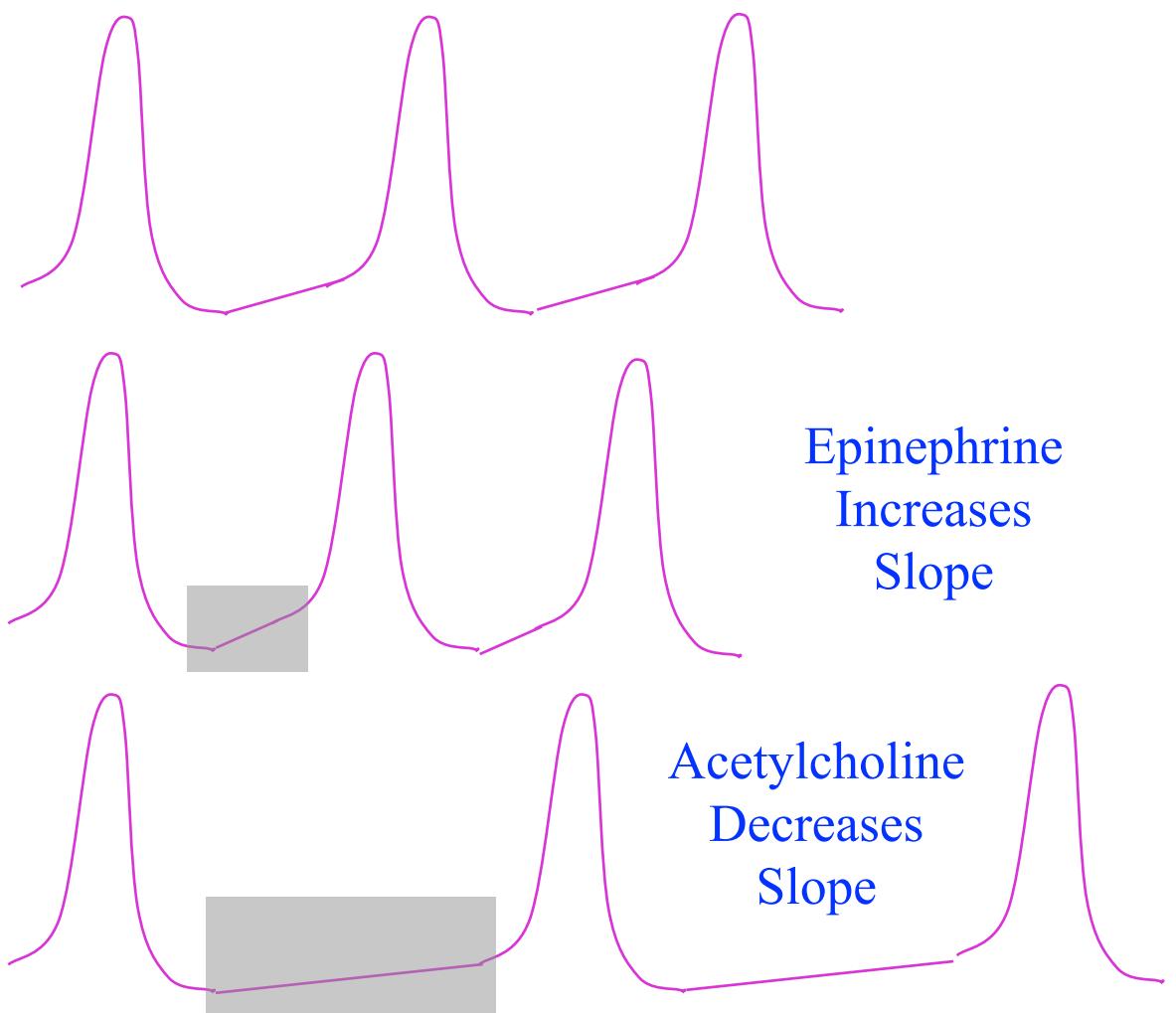


Slow decrease in  $K^+$  permeability

← →  
time

Heart rate = # beats / time

# *Pacemaker Potentials*



Normal

Increase  
Heart Rate

Decrease  
Heart Rate

# Regulation of Circulation

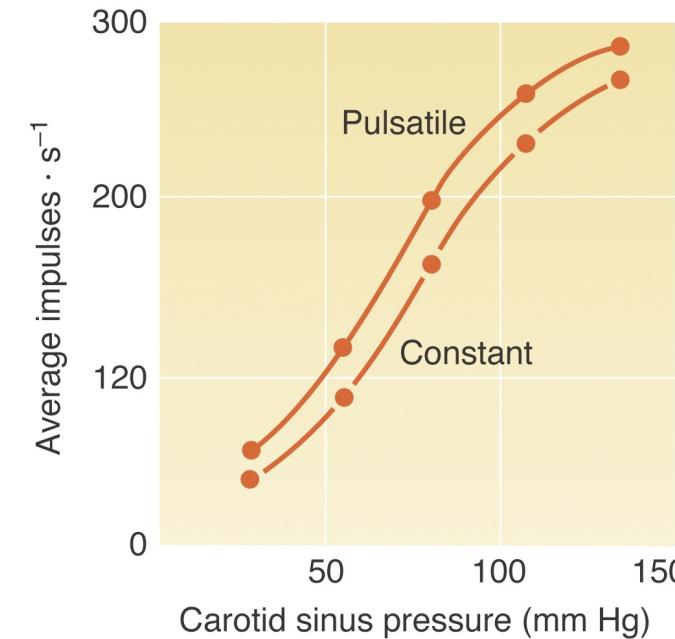
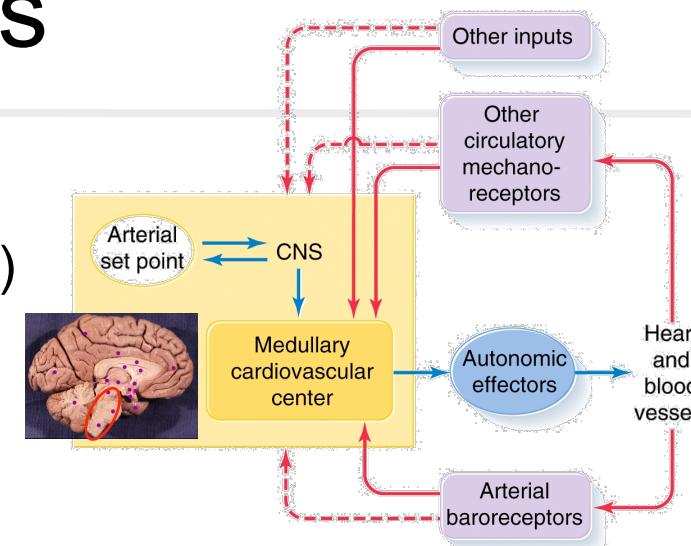
# Regulation of Circulation

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- Priorities:
  - Ensure adequate supply of blood to brain and heart
    - Stroke, Myocardial Infarction (heart attack)
  - Supply blood to other systemic organs
  - Control capillary pressure
    - Control tissue volume
    - Maintain interstitial fluid composition
- Control Mechanisms:
  - Baroreceptors: pressure sensitive sensory neurons
  - Chemoreceptors: ventilation AND circulation
  - Endocrine (hormonal) and Chemical messengers (NO)

# Arterial Baroreceptors

- Pressure-sensing mechanoreceptors
- Receptor signals transmitted to Medullary Cardiovascular Center (MCC) and integrated with signals from other neuronal circuits
- Baroreceptors activate or inhibit two MCC functional regions:
  - Pressor center (increases b.p.)
    - Sympathetic activation
  - Depressor center (decreases b.p.)
    - Parasympathetic activation
- Arterial baroreceptors:
  - Mammals: carotid sinus, aortic arch, clavian, common carotid, and pulmonary arteries
  - Increase firing with increases in b.p.
  - Initiate reflexes to reduce arterial b.p. by decreasing cardiac output (both HR and SV) and peripheral vascular resistance



# Arterial Chemoreceptors

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- Chemoreceptors in carotid and aortic body
  - Sense  $CO_2$ , pH,  $O_2$ 
    - Ventilation Regulation
  - Increases in these chemoreceptor impulses also
    - Reduces Heart Rate
    - Increases Peripheral Vasoconstriction
      - Increases Blood Pressure
  - Important in control of dive response

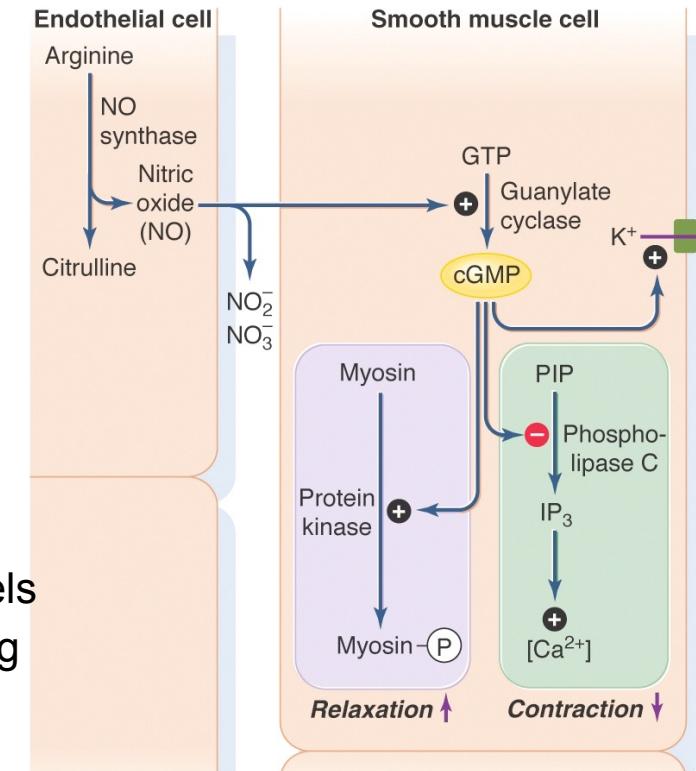
# Cardiac Sensory Systems

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- Atrial **Mechanoreceptor** fibers:
  - A-fiber: sense changes in **heart beat rate**
    - Signals sent to Medullary Cardiovascular Center
  - B-fiber: sense changes in **rate of filling of atria**
    - Increased venous pressure stimulates B-fibers, which increase HR through innervation of SA node
    - Increase **diuresis** (urine prod.) which reduces blood volume + pressure
      - Mediated by decreased antidiuretic hormone (ADH) in blood
  - C-fibers: sense **stretch** at junction of veins and atria
    - Stimulation maintains HR
- Atrial **Stretch-sensitive** secretory cells:
  - Produce atrial natriuretic peptide (ANP)
    - Increases **Na<sup>+</sup> excretion** (natriuresis) and **urine production**
      - Reduces blood volume + pressure
    - ANP also inhibits pathways to resorb Na and **increase blood volume**
- Ventricular mechanoreceptors:
  - **Low coronary blood flow** stimulates and **increases cardiac output**
  - High stimulation causes **stomach relaxation** and produces vomiting

# Capillary blood flow control

- Hyperemia/Ischemia control (too much/too little blood)
  - Increase/decrease
- Neuronal control
  - **Norepinephrine** (sympathetic): vasoconstriction, flow reduction, incr. b.p.
  - **Acetylcholine** (parasympathetic): vasodilation, flow increase, decr. b.p.
- Local control by Nitric Oxide (NO)
  - Produced in vascular endothelial cells
  - Diffuses into vascular smooth muscle
  - Binds to receptors at metal ion or S
  - Activates guanulate cyclase which produces second messenger cGMP
    - like adenylate cyclase
  - cGMP relaxes muscles by affecting multiple pathways
- NO synthases are activated by  $\text{Ca}^{2+}$ 
  - $\text{Ca}^{2+}$  enters through stretch-receptive channels
  - Potentially NO synthases are activated during every pulse



# Just say NO: molecule of the year in 1992

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- Nitric Oxide is an important diffusible messenger in many physiological processes, and has many important applications
  - Smooth Muscle Relaxation
    - Vasodilation:
      - Viagra, Levitra, Cialis enhance NO effects by inhibiting the enzyme that degrades cGMP
      - Nitroglycerine releases NO when it dissolves
    - Gastrointestinal peristalsis
    - Genito-urinary tract - birth
  - Kidney function
  - Inflammation and immune responses
  - Hormonal secretion
  - Nervous system function
    - Neurotransmitter
    - Ventilation control (complexed with Hemoglobin)
  - Reproductive physiology - egg fertilization
  - Firefly flashing
  - Plant pathogen defenses, reproduction