Cardiac Cycle

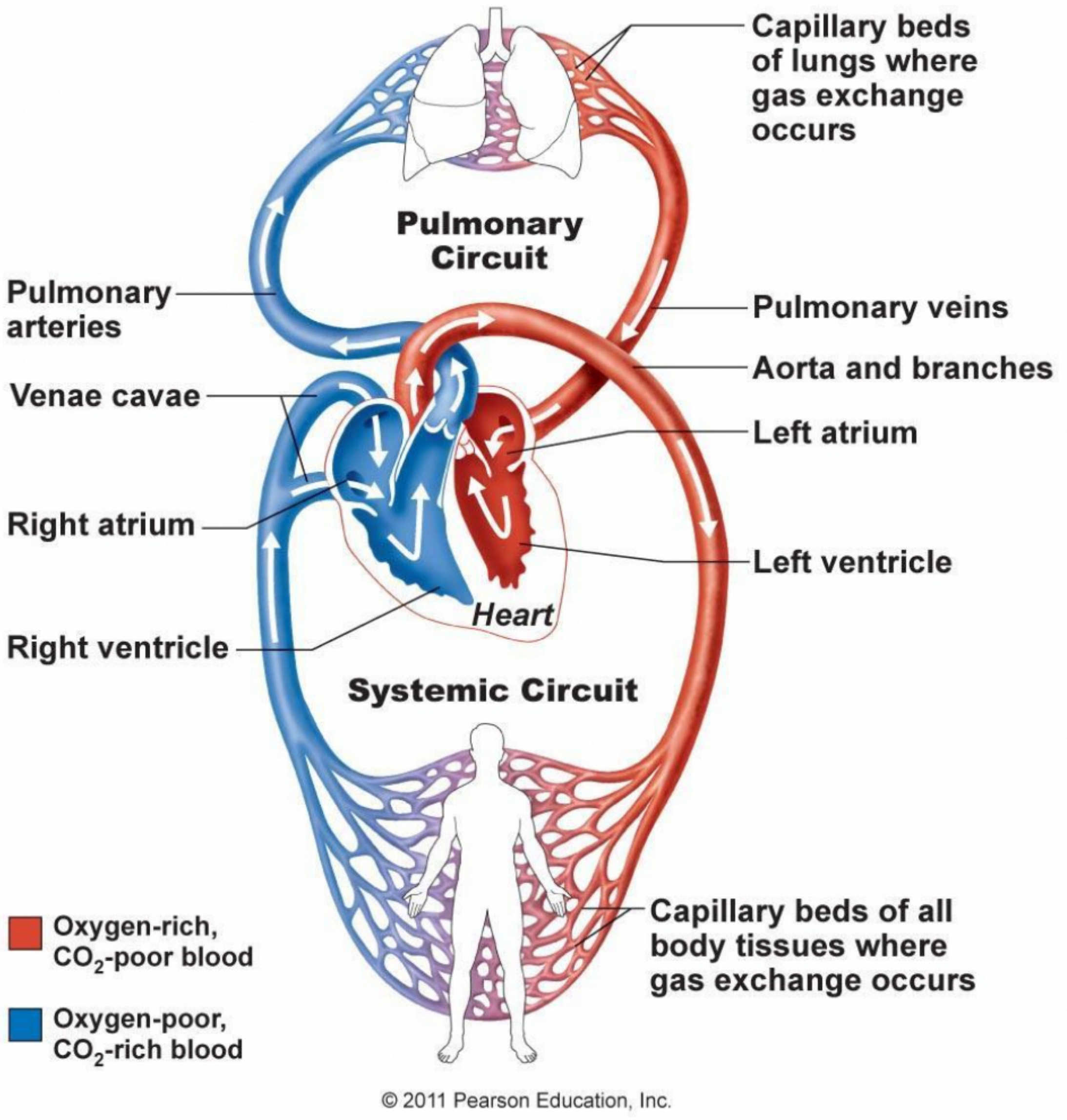
Central Circulation, Electrocardiogram (and Circulatory Regulation)

ZOOL 430

Circulatory System

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

Mammalian 4-chambered heart



Myogenic Heart

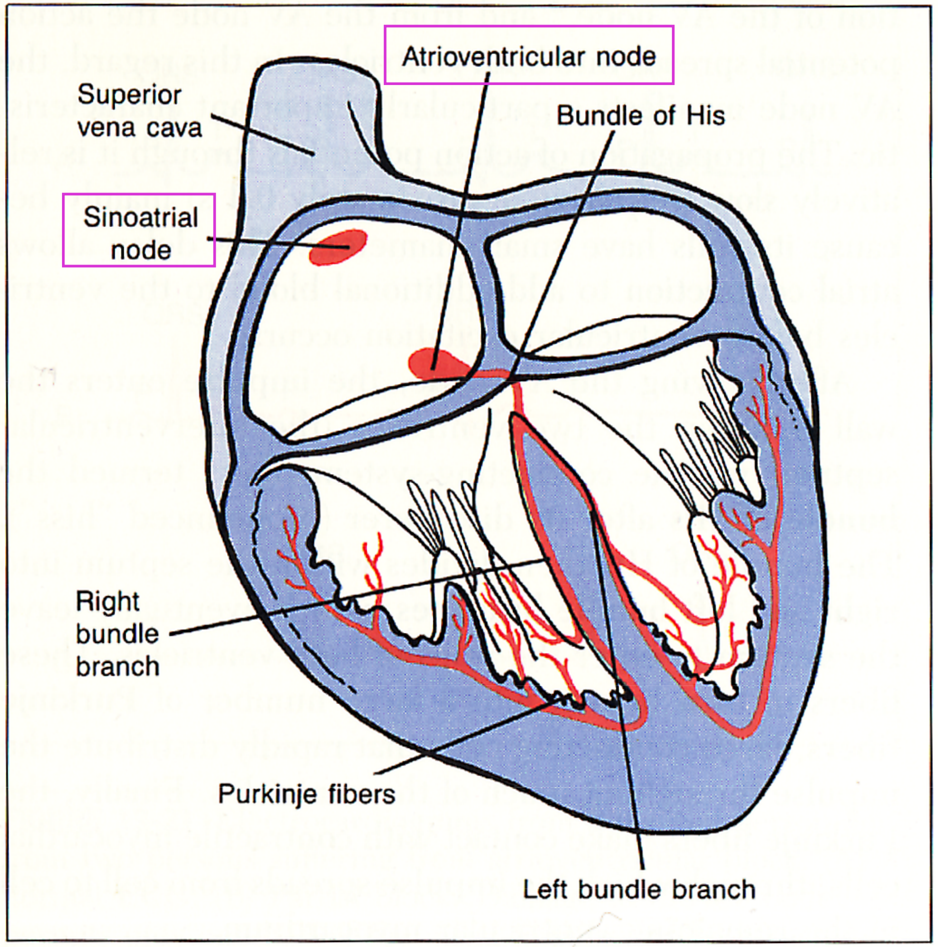
electrical signals

Pacemaker beats on its Own!

Myogenic = modified **muscle** cells

Found in two nodes

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



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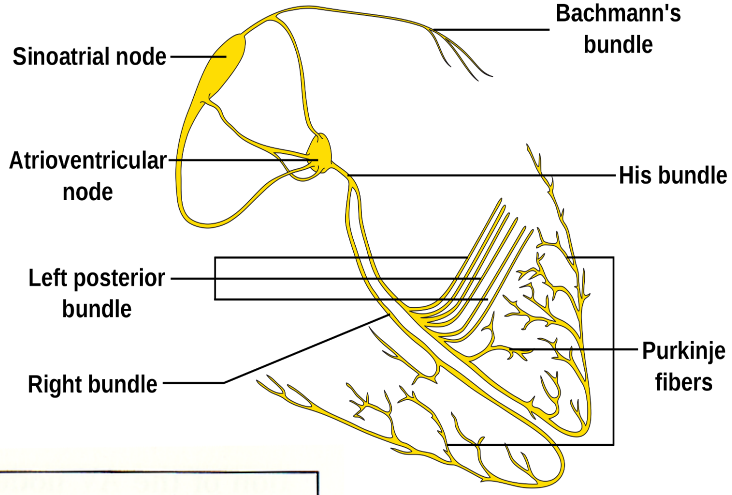
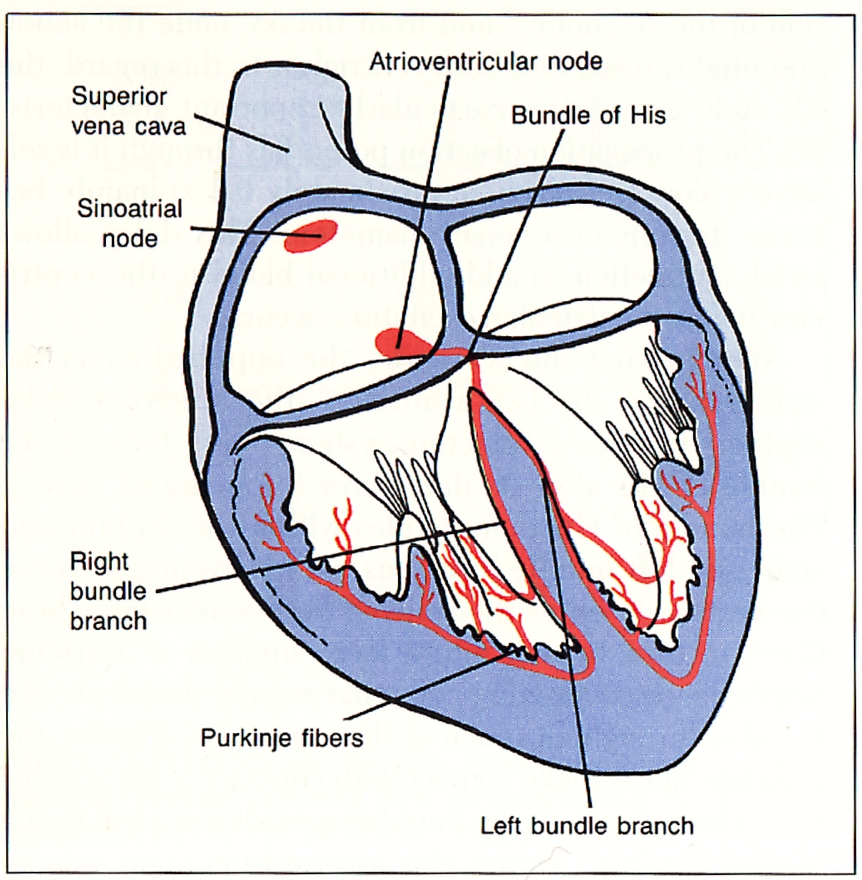
Pacemaker Cells

- **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

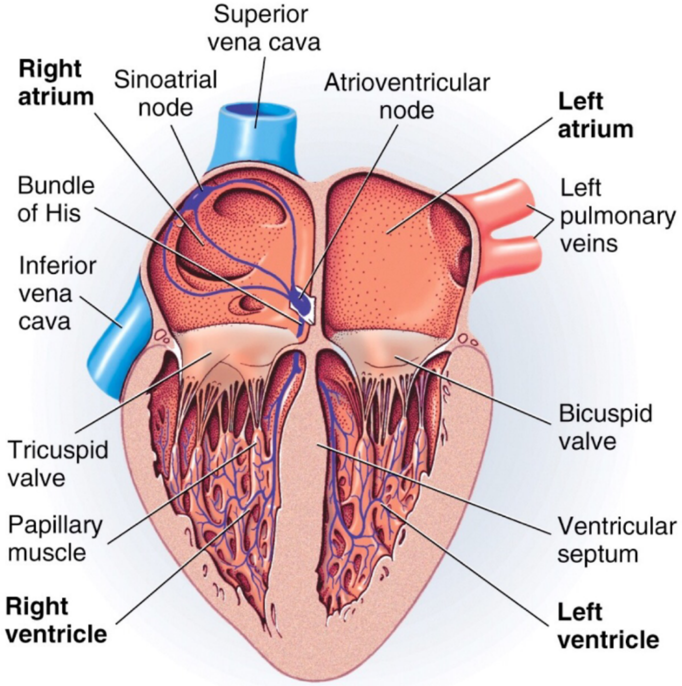
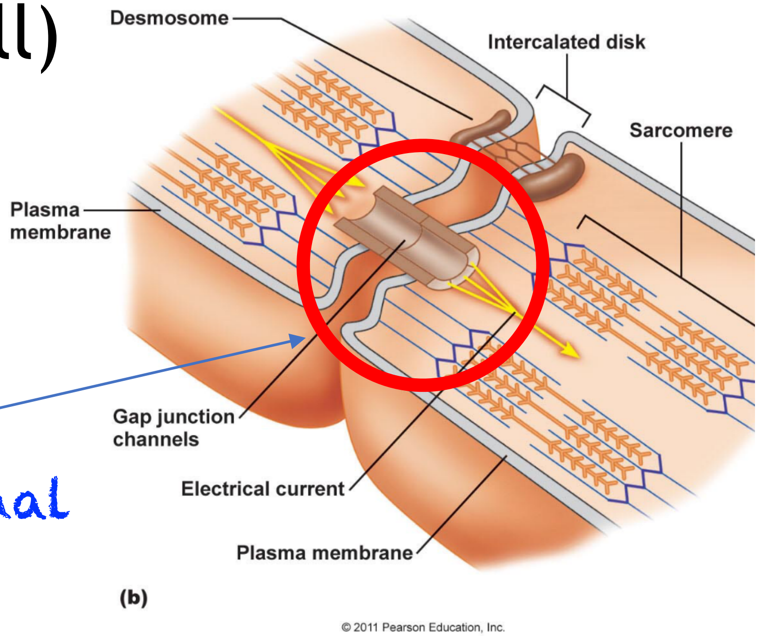
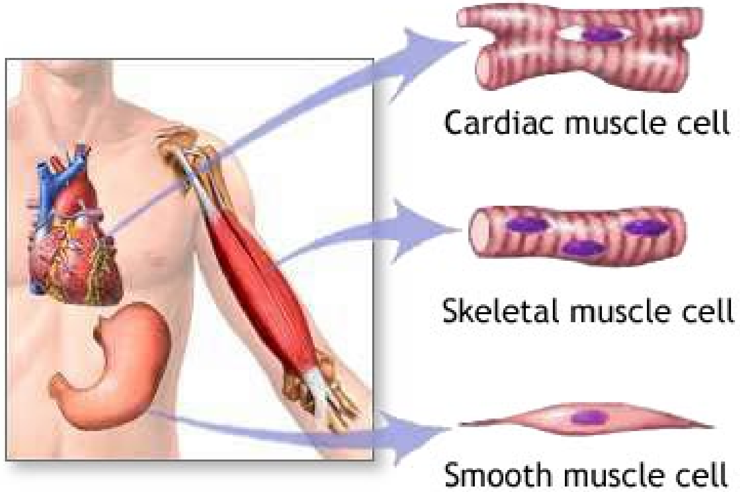


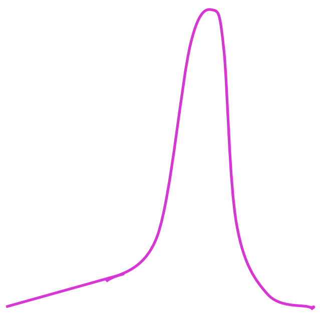
Figure 1. The 4 chambered heart



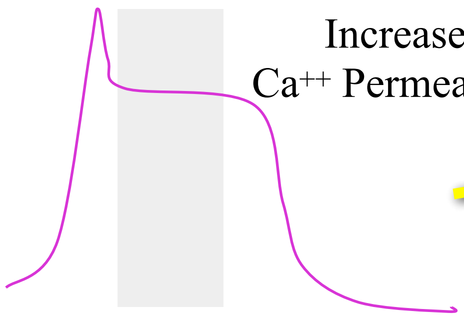


Pacemaker and Cardiac Potentials (special types of action potentials)

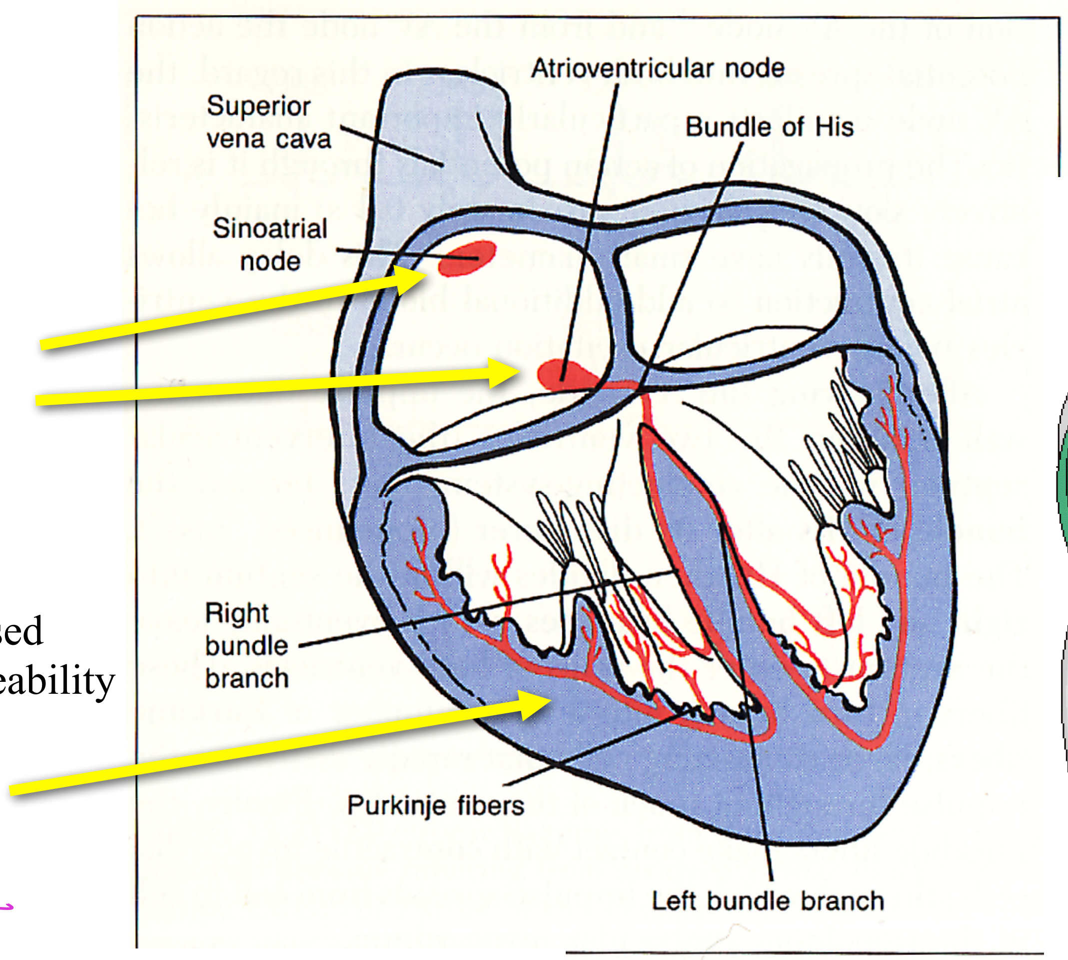
Pacemaker Potential

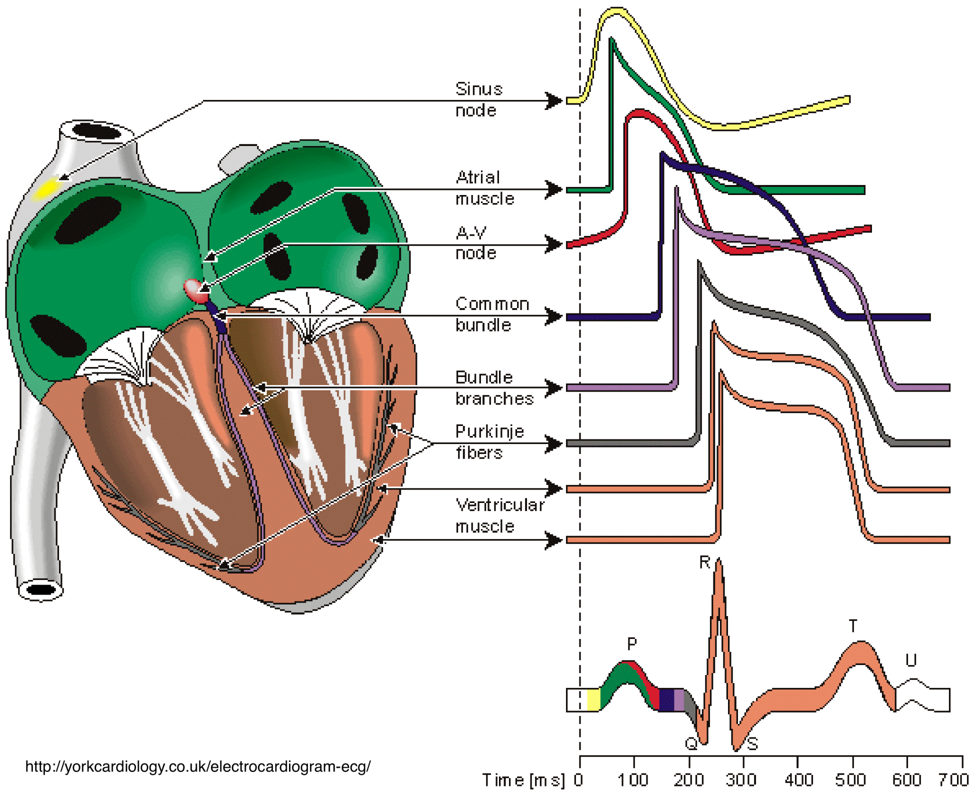


Cardiac Potential



Increased
Ca^{++} Permeability





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

Prolonged Potential
Synchronizes Contraction

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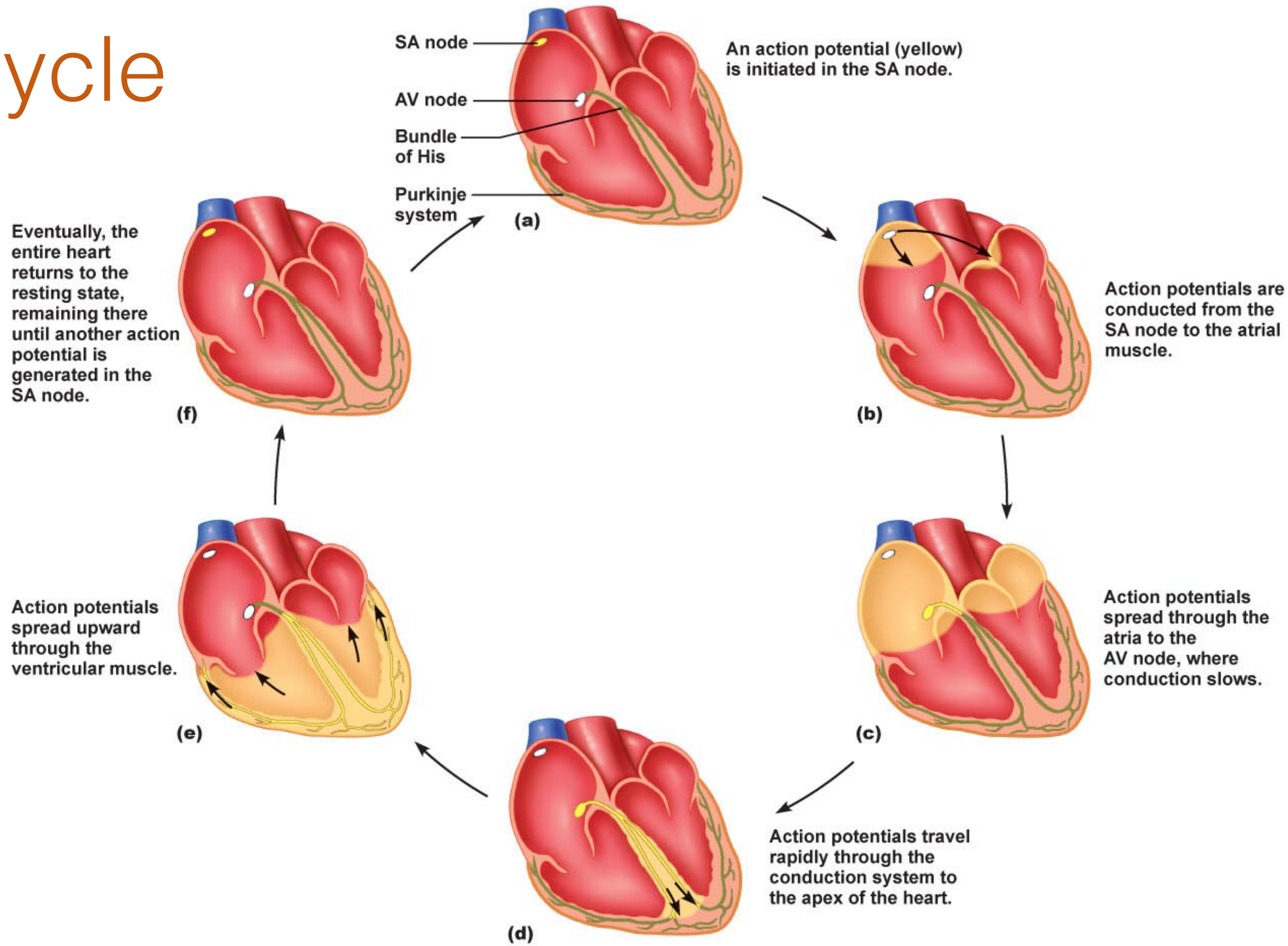
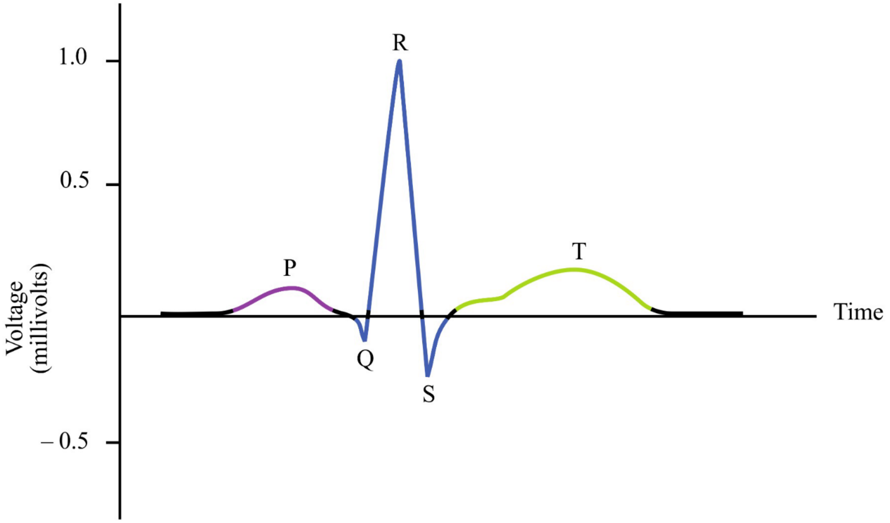
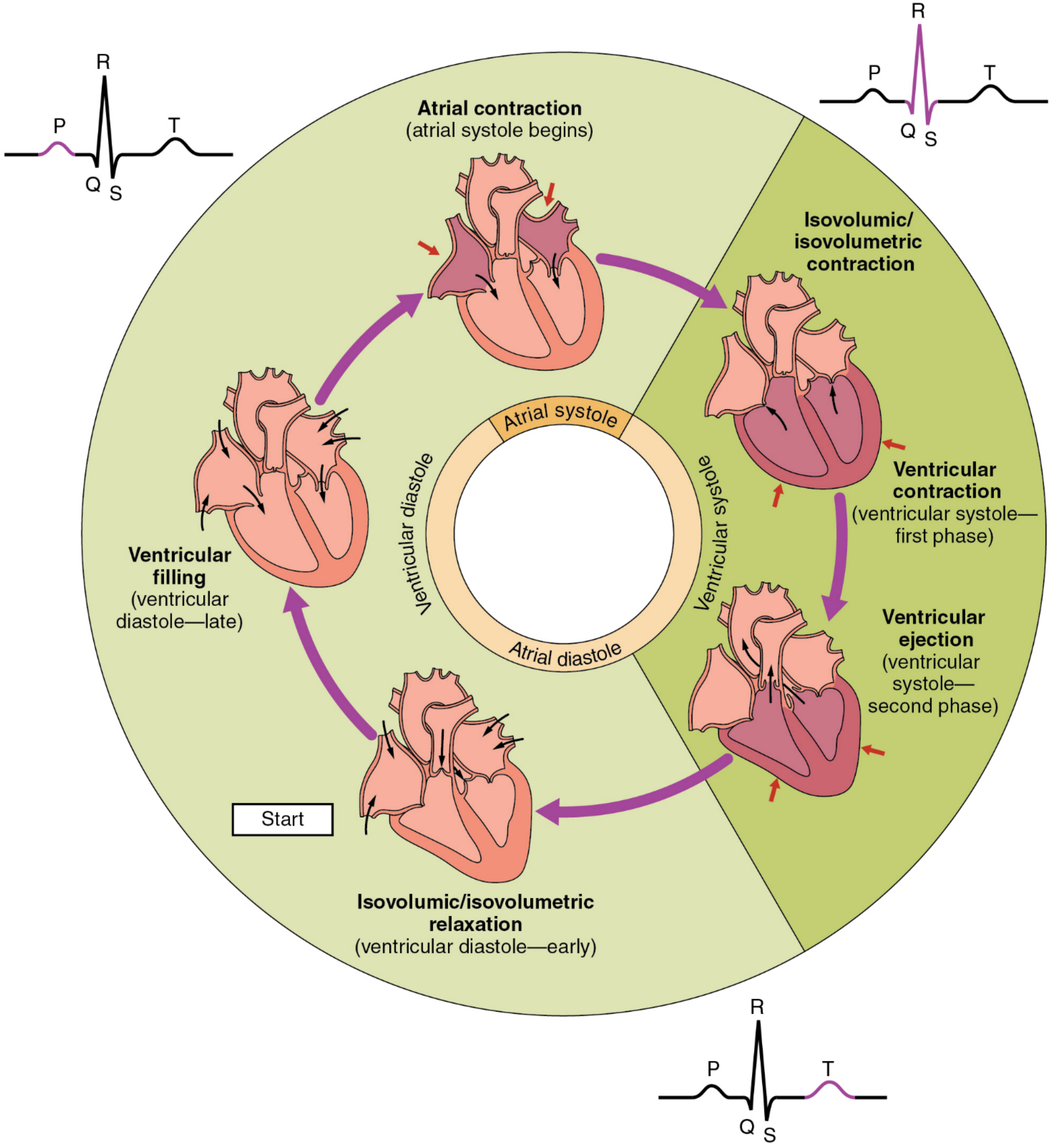
Cardiac Cycle



Electrocardiography

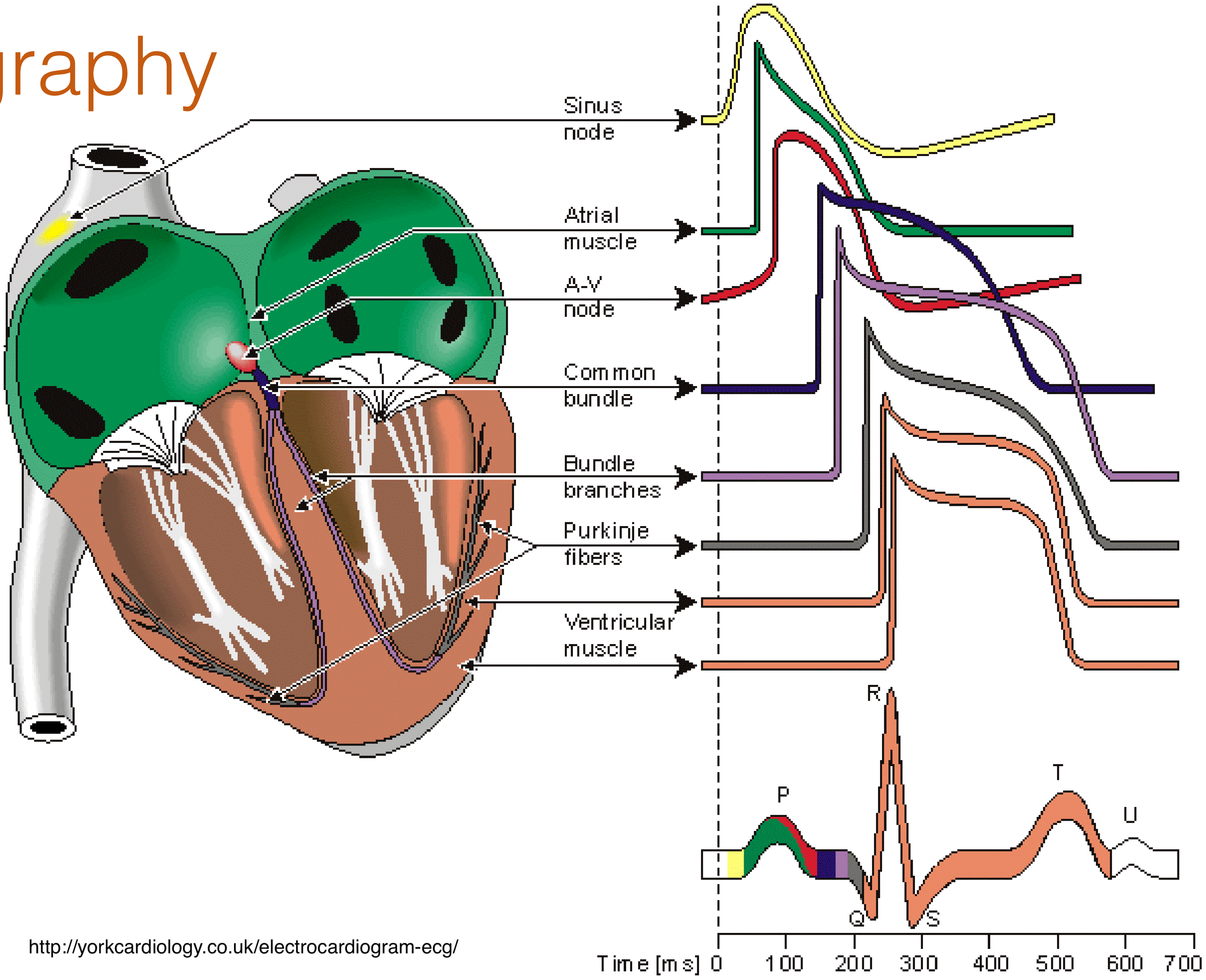
- Measure electrical changes caused by cardiac AP using electrodes attached to the surface of the skin
- Signals detected would be the combination of atrial and ventricular activities.





Electrocardiography

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



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

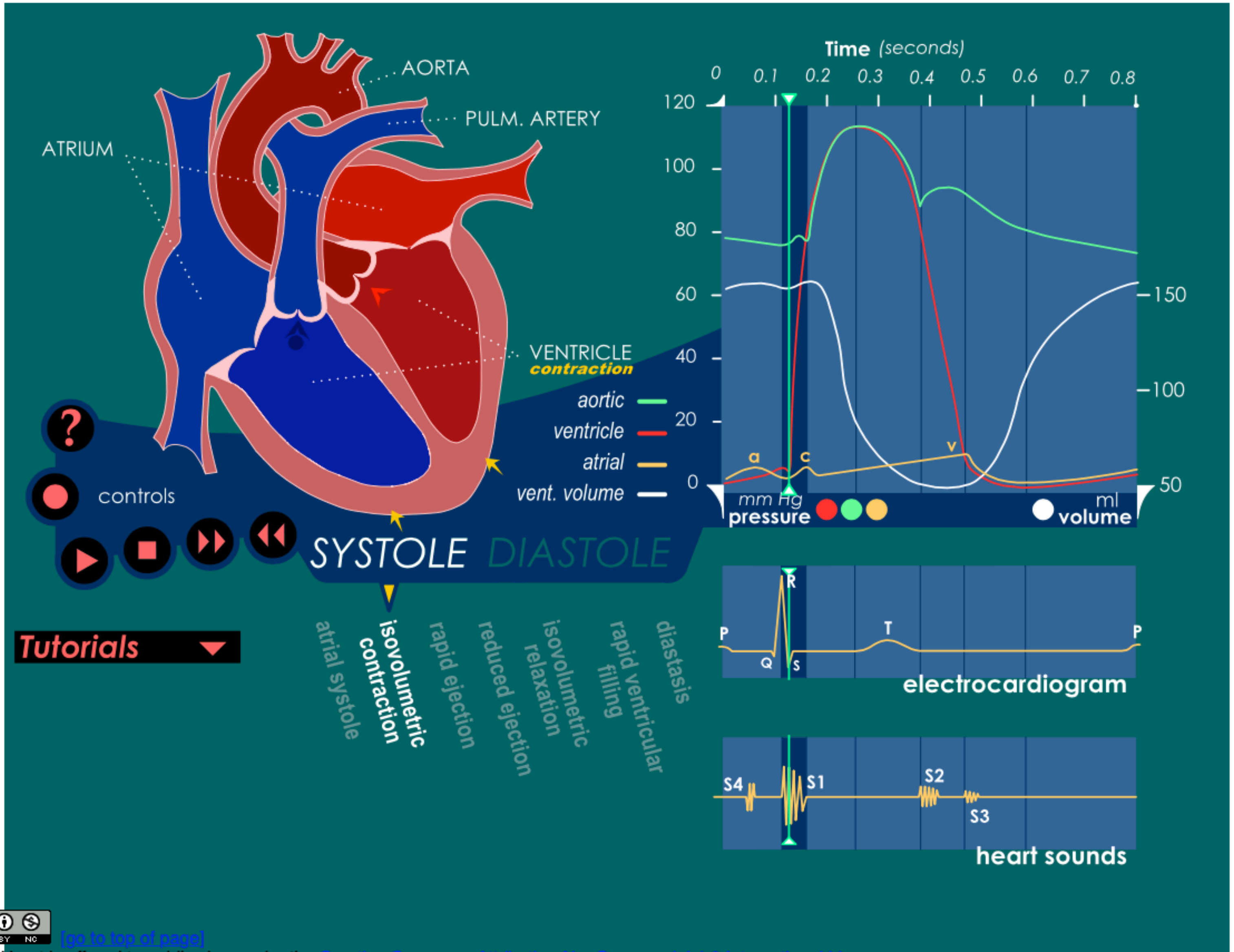
HyperHeart

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 Common
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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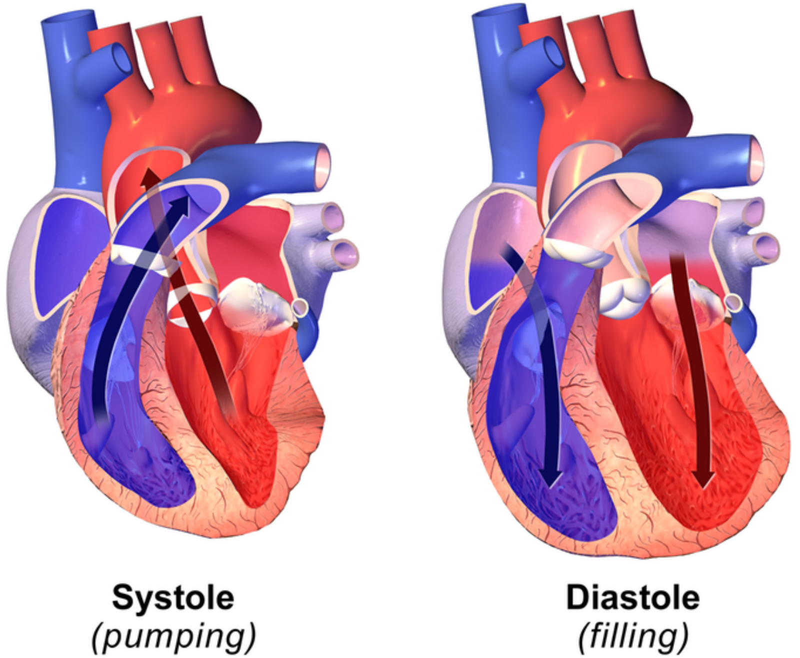
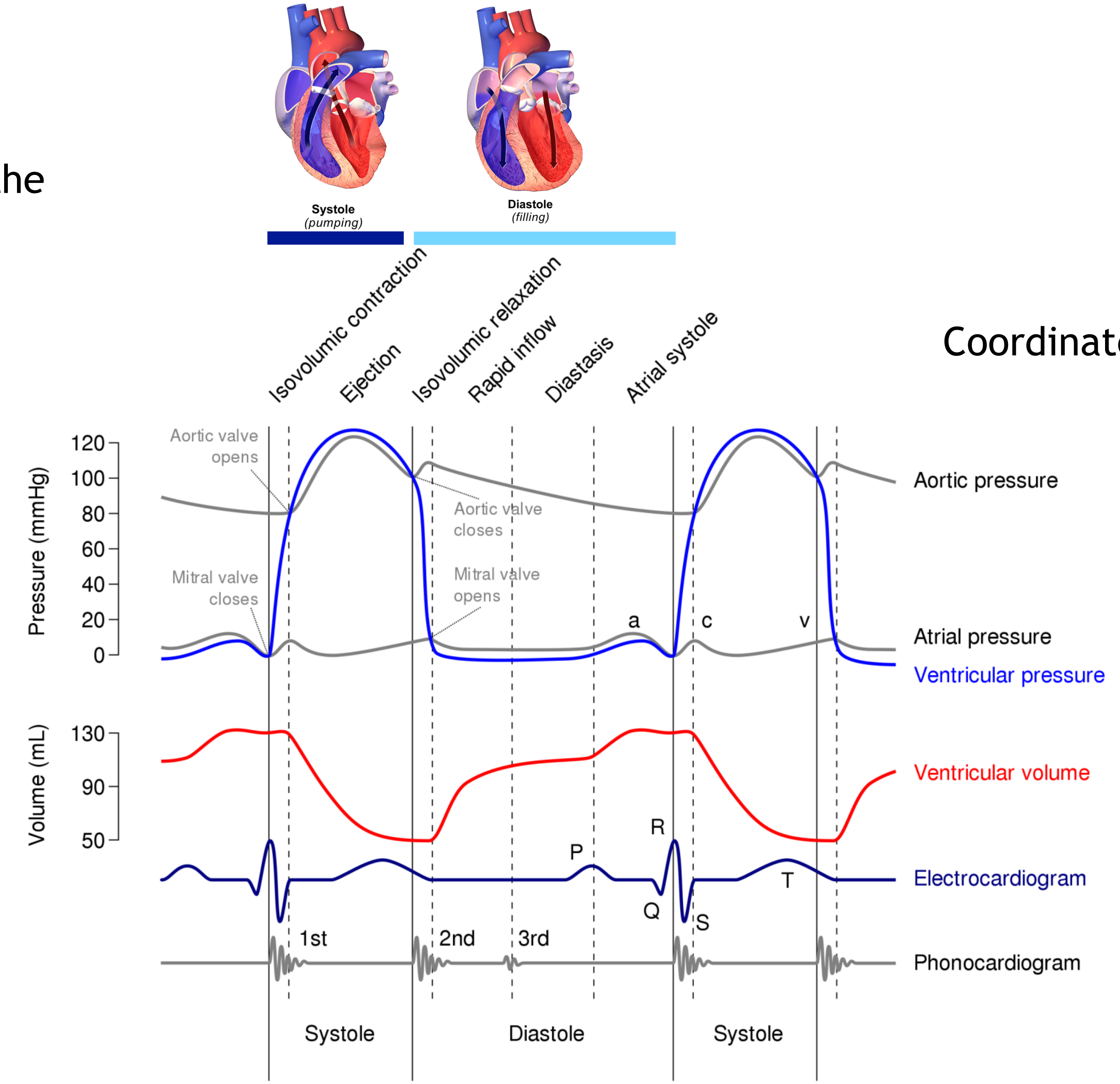
Cardiac Cycle

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



Ventricular Contraction

Ventricular Relaxation



Coordinated changes in:

Pressure

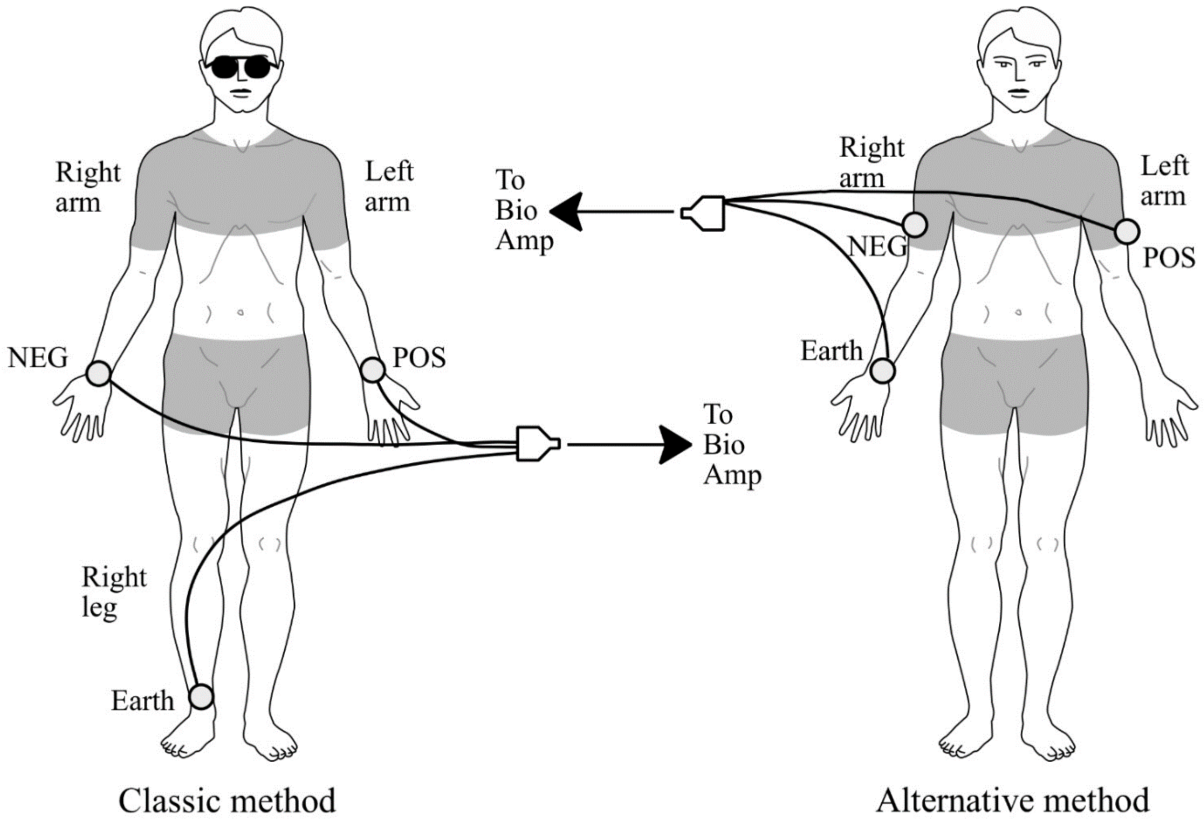
Volume

Electrical Activity

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

More stretch → More SV!

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

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

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

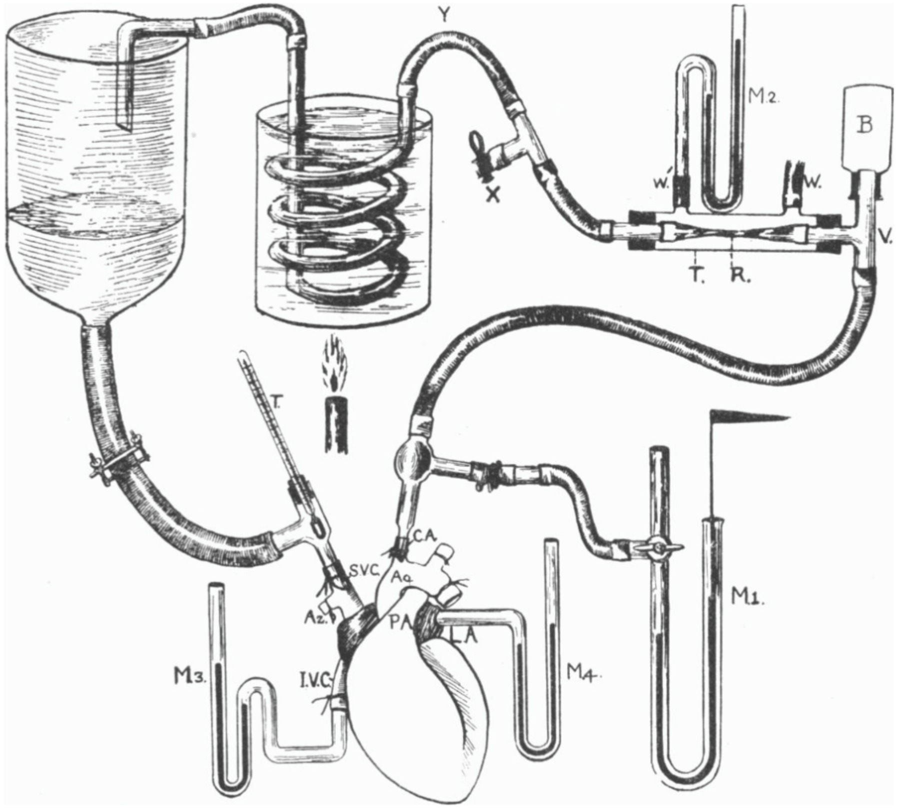


Fig. 1.

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].

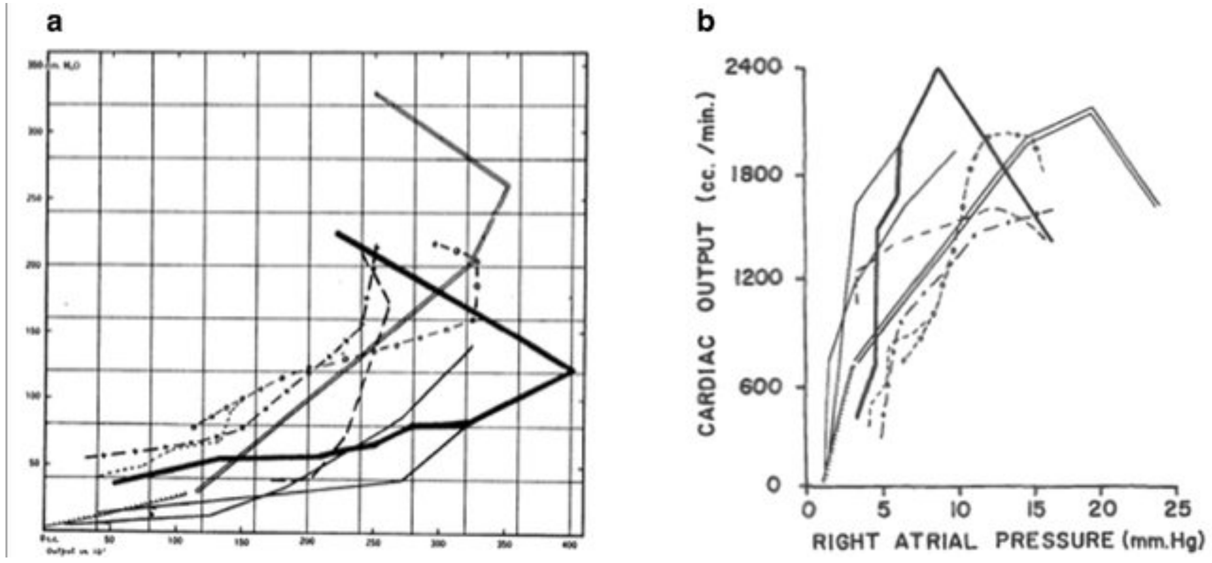


Figure 2 Original presentation and reproduction of the Starling curve. (a) Original presentation of the Starling curve. The y axis is right atrial pressure (mmH 2 O). The x axis is cardiac output (cm 3 /minute). Image reproduced with permission from [1]. (b) Reproduction in Guyton and colleagues' textbook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

Berlin, D.A., Bakker, J. Starling curves and central venous pressure. *Crit Care* **19,** 55 (2015).

Really cool, but mechanism?

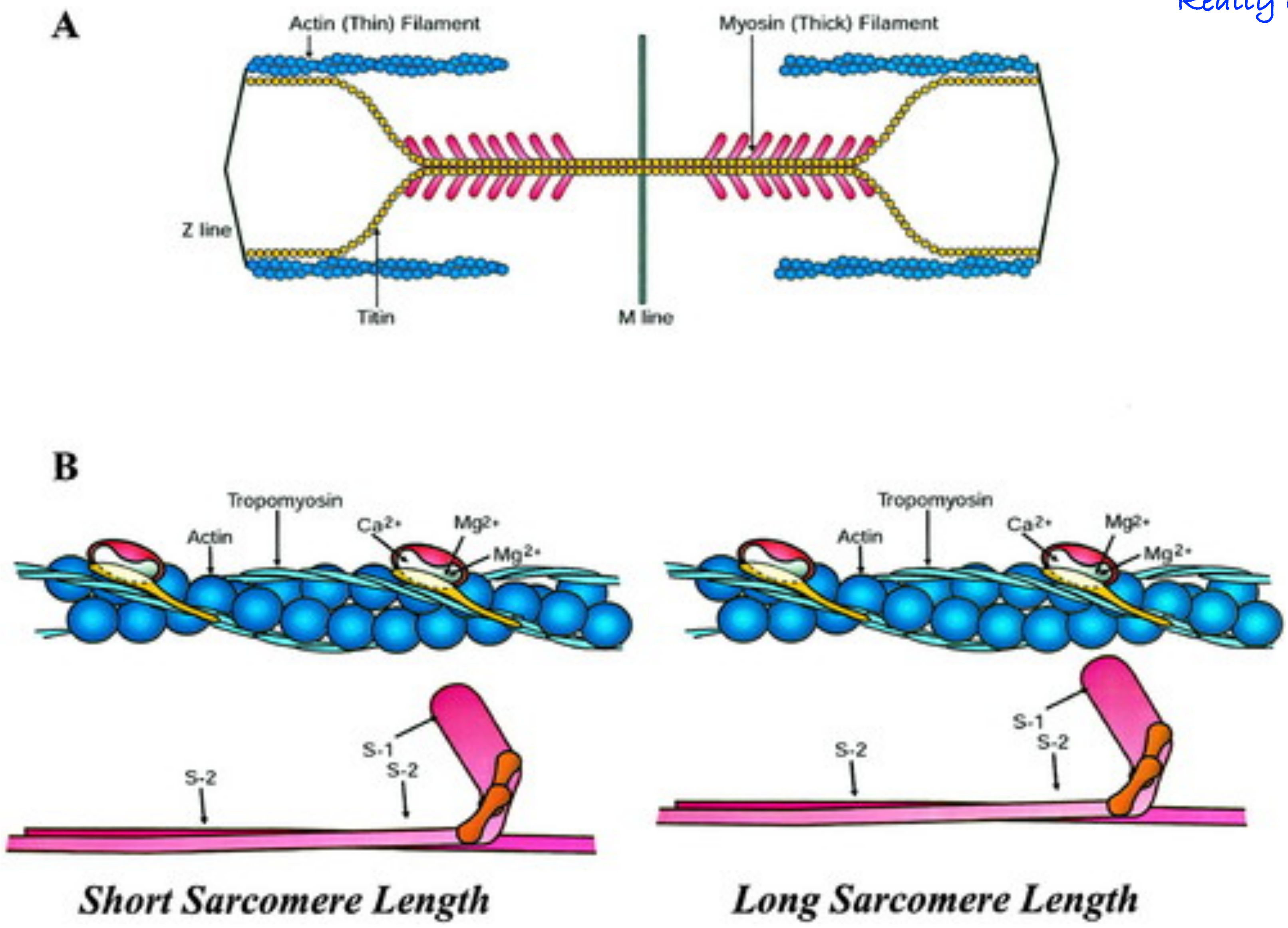


Figure 1. Myocardial sarcomere and myofilaments.

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.**11

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.

Richard L. Moss. 2002. Circulation Research. Frank-Starling Relationship, Volum
90, Issue: 1, Pages: 11-13, DOI: 10.1161/res.90.1.11

Starling's Law

Stroke volume <-> End diastolic volume

Force of contraction is proportional to length of the myofibrils (cardiac muscle cells)

Cardiac Output <-> Stretch

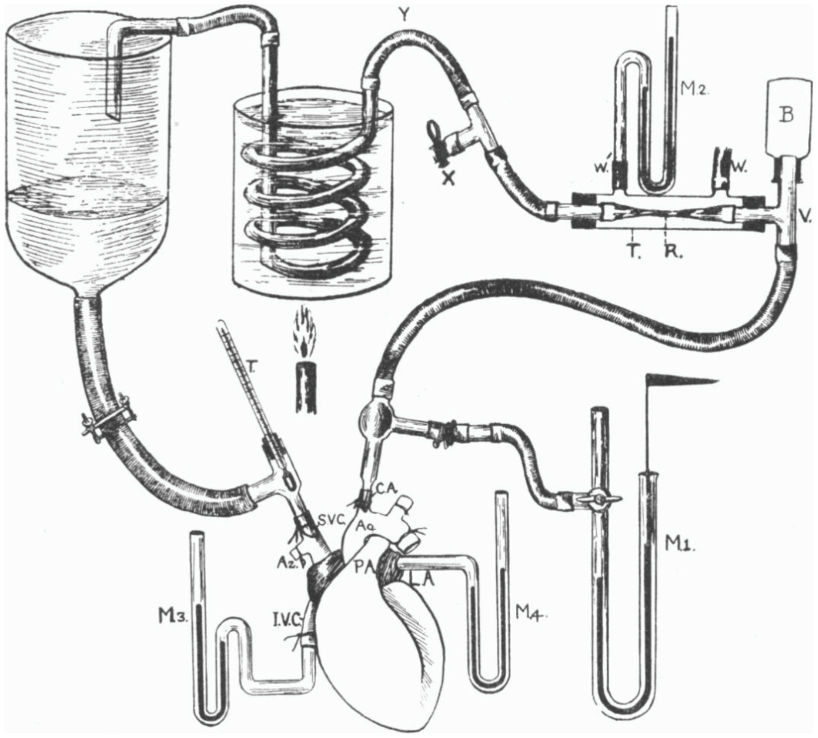
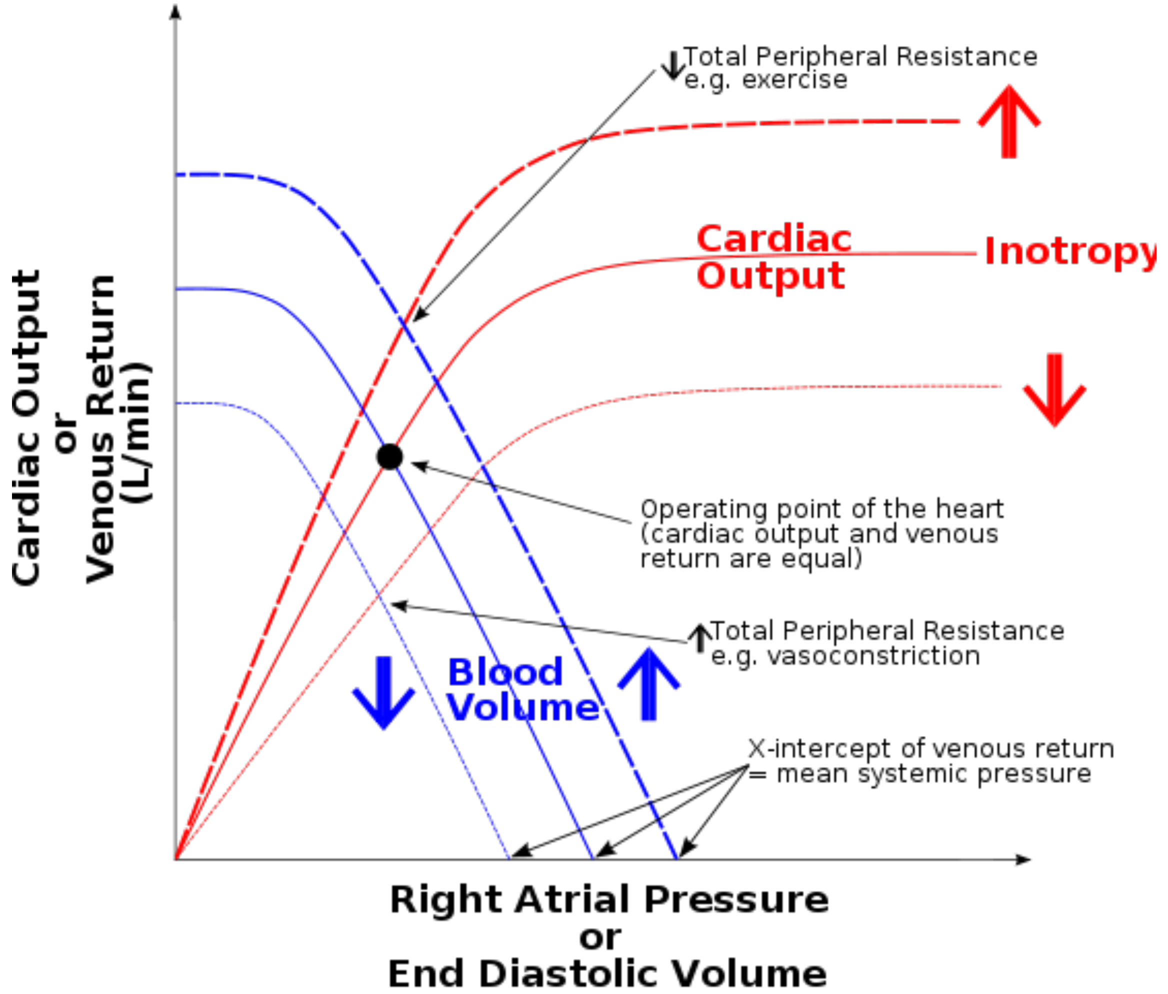


Fig. 1.



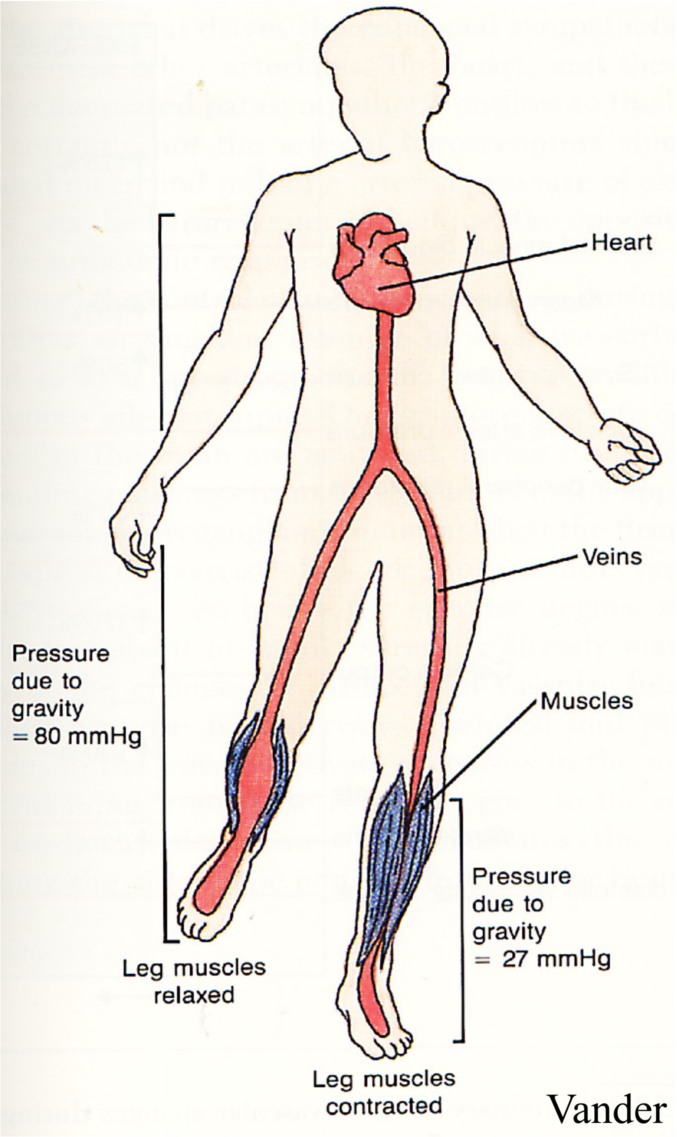
Muscle Pumps

High Pressure at Feet leads to venous pooling.

Blood in feet must be pumped against the weight of the column of blood above it.

Leg muscle acts as pumps.

Veins have valves to prevent back flow.



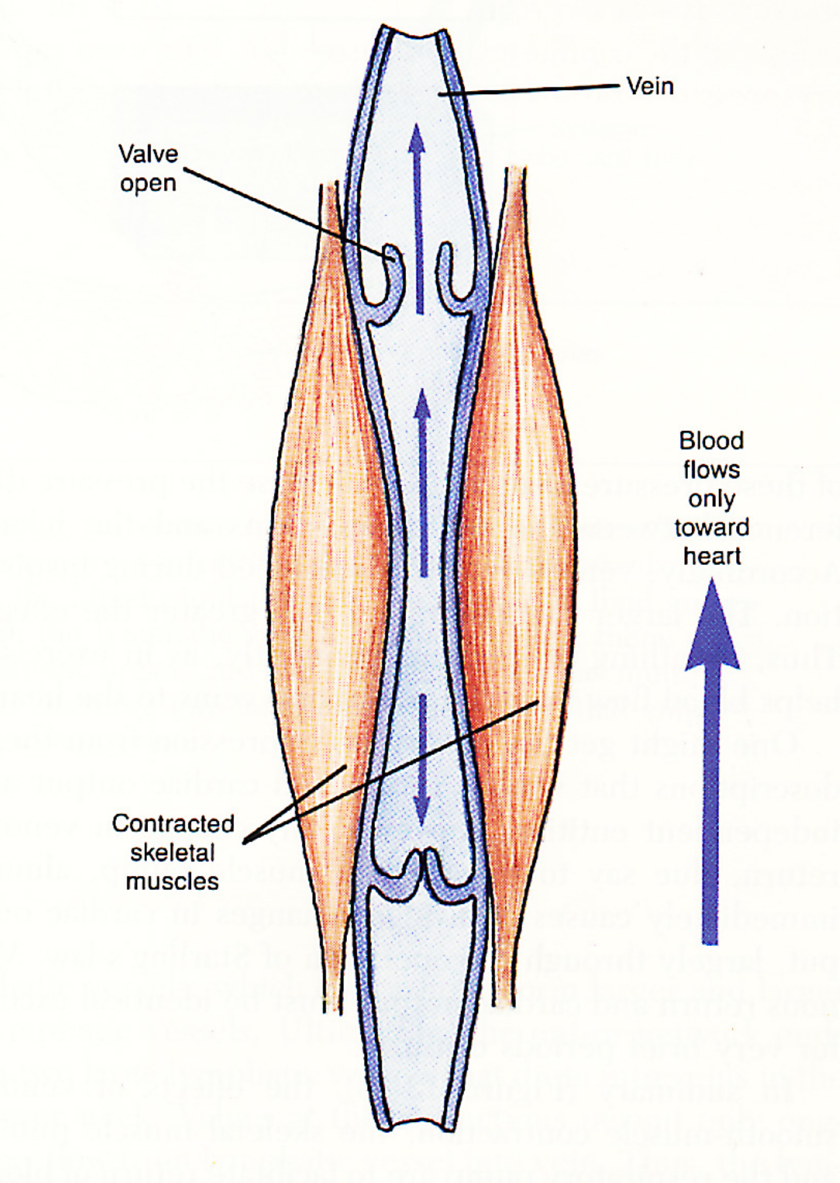
Vander et al.

Veins with ***One-Way*** Valves

Muscles contract and move blood toward heart.

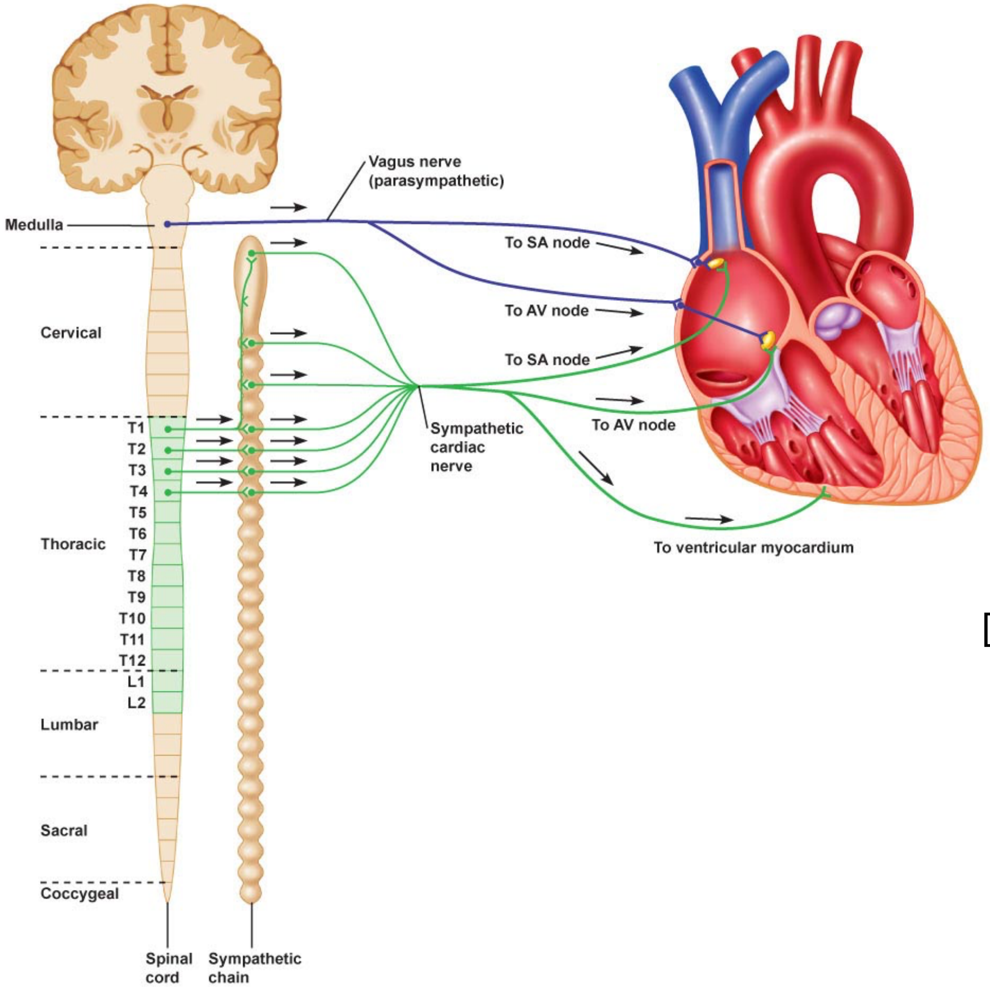
Valves open in direction of heart.

One-way valves prevent back flow.



Regulation of Heart Rate

Nervous System

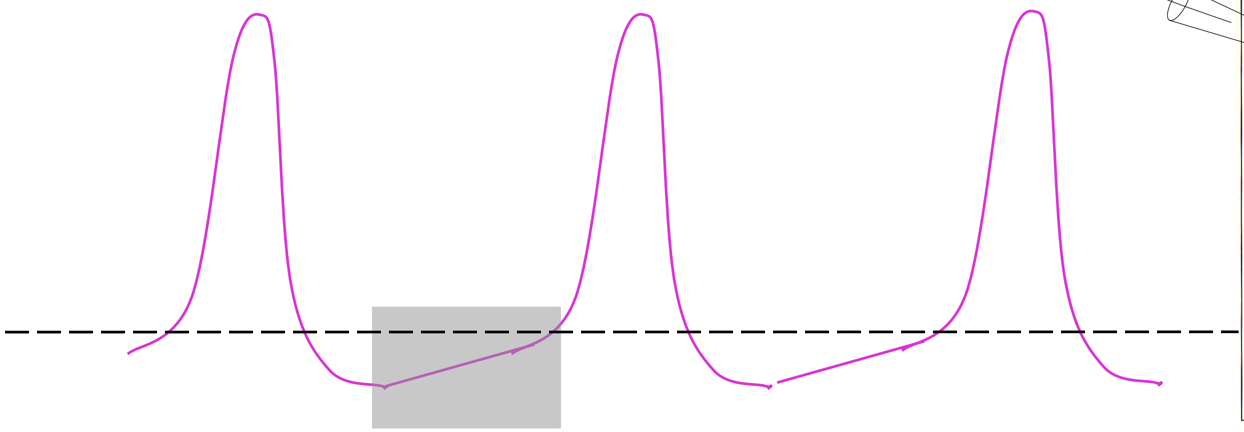


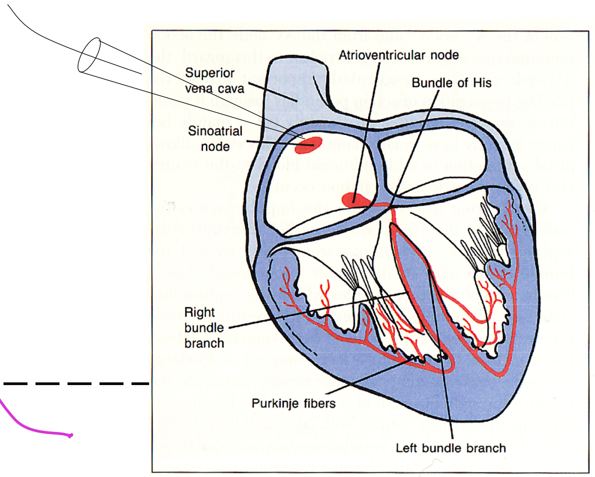
Parasympathetic & Vagus Nerve
Ach (acetylcholine) -> SA node

Sympathetic
Epinephrine

Norepinephrine (synthesized from Dopamine) increases blood pressure

Pacemaker Potentials



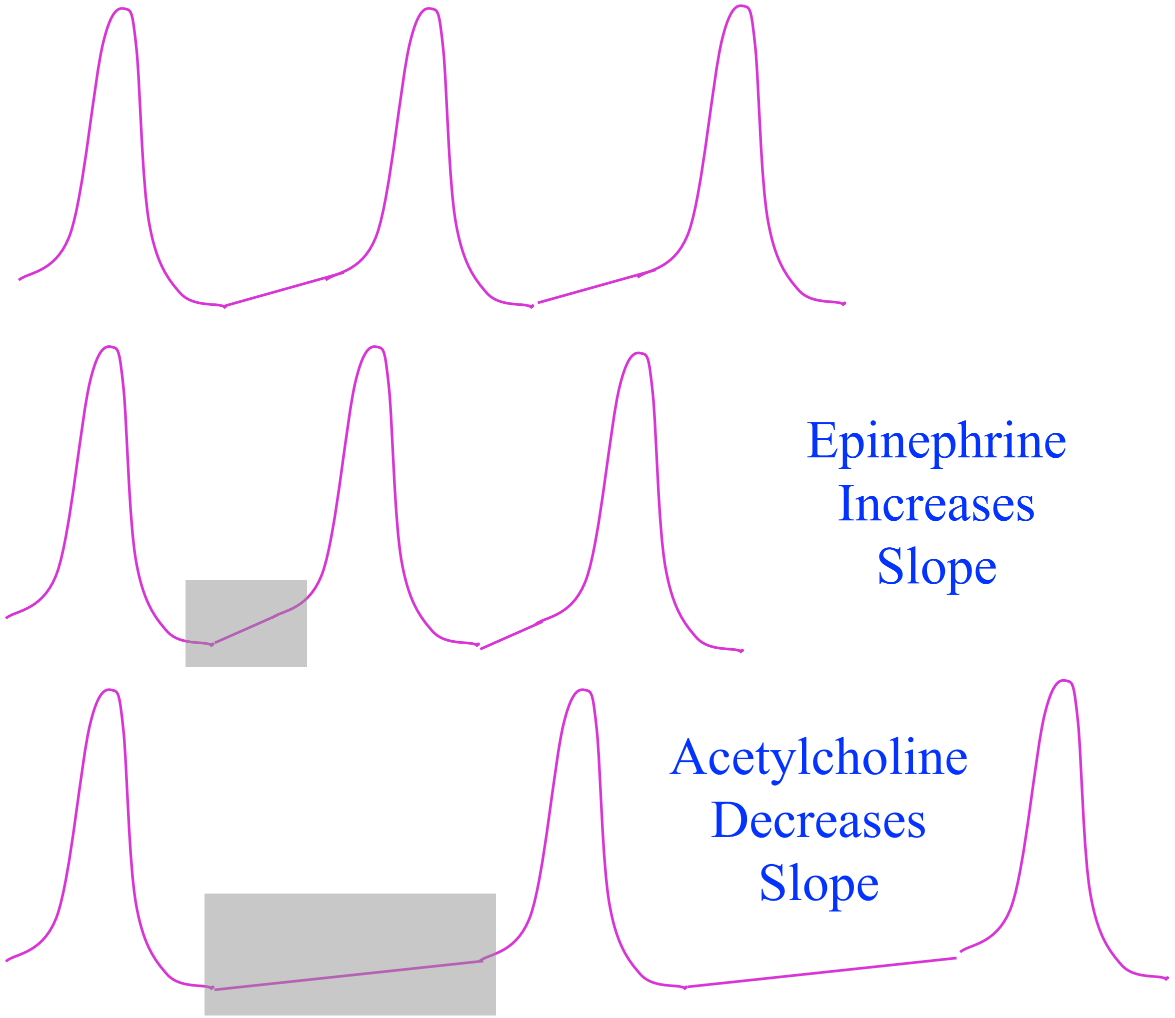


Slow decrease in K^+ permeability

time

Heart rate = # beats / time

Pacemaker Potentials



Normal

Increase
Heart Rate

Decrease
Heart Rate

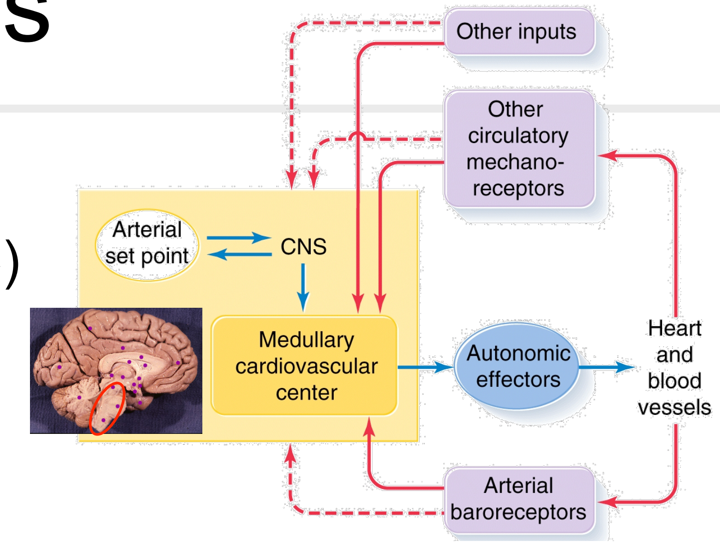
Regulation of Circulation

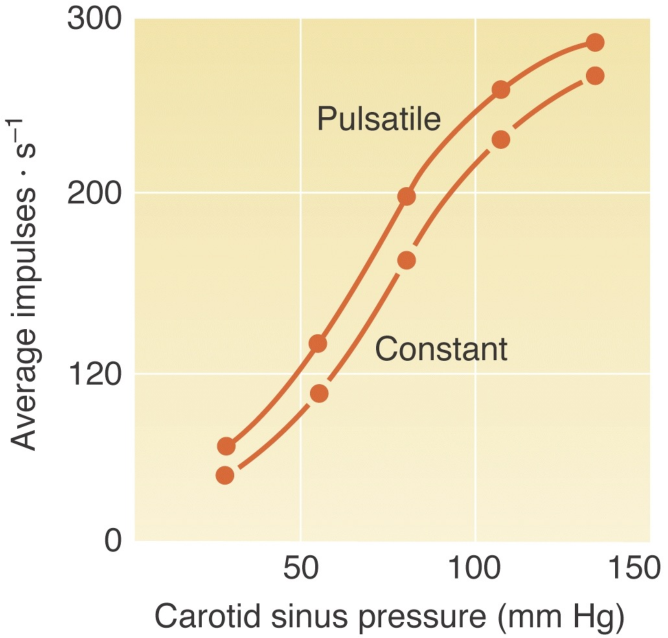
Regulation of Circulation

- 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

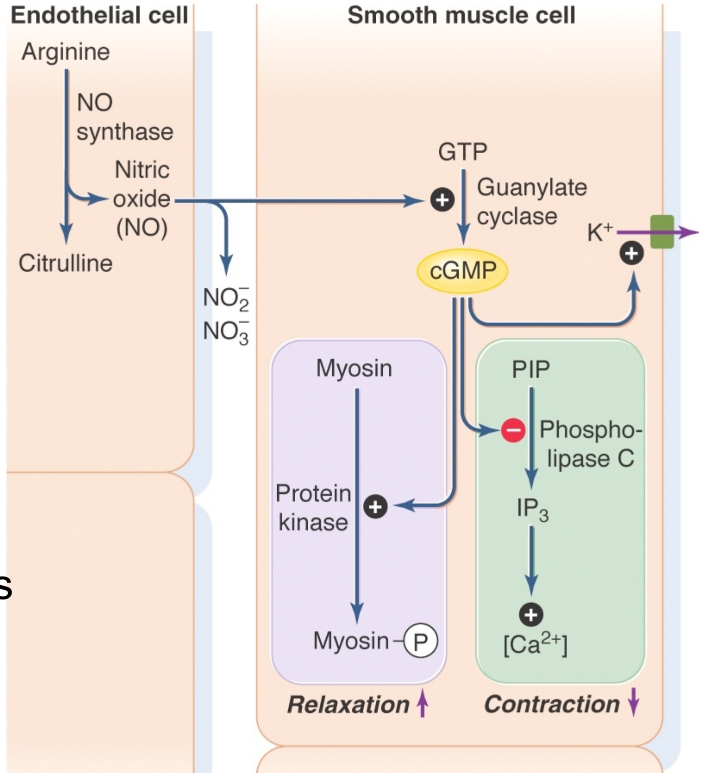
- Chemoreceptors in carotid and aortic body
  - Sense CO_2, pH, O_2
    - Ventilation Regulation
  - Increases in these chemoreceptor impulses also
    - Reduces Heart Rate
    - Increases Peripheral Vasoconstriction
      - Increases Blood Pressure
  - Important in control of dive response

Cardaic Sensory Systems

- 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+ 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 Ca^{2+}
  - Ca^{2+} enters through stretch-receptive channels
  - Potentially NO synthases are activated during every pulse



Just say NO: molecule of the year in 1992

- 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