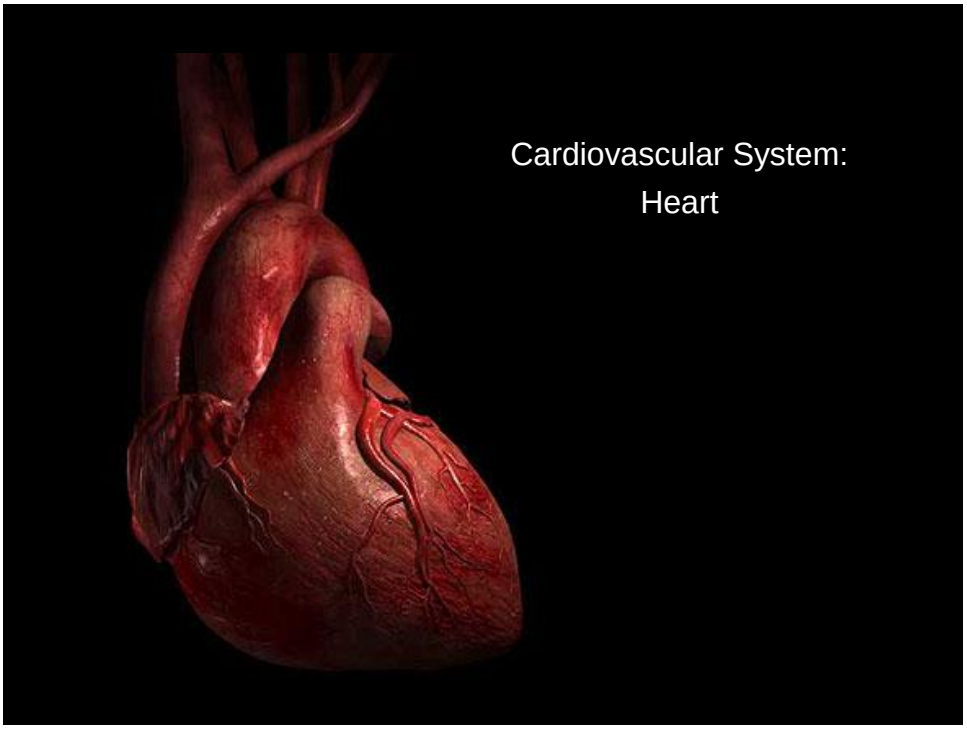
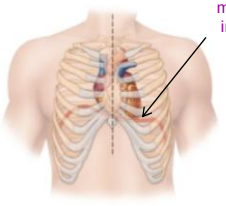


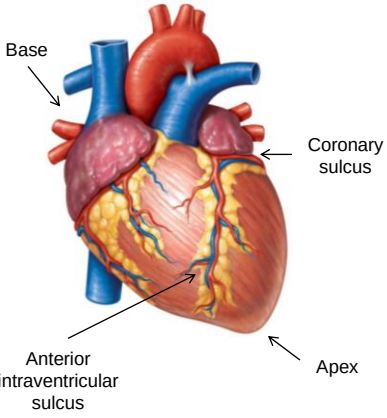
Cardiovascular System – Heart

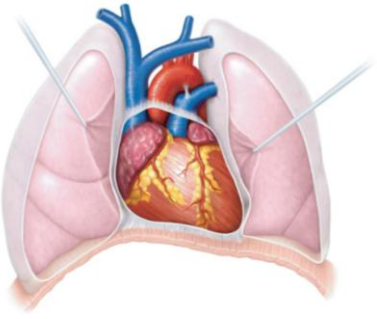
Heart:

- Roughly size of human fist (~ 250 – 350 grams)
- Located in the mediastinum (medial cavity of thorax)
- "Double pump" composed of cardiac muscle



2/3 of heart mass lies left of mid-sternal line



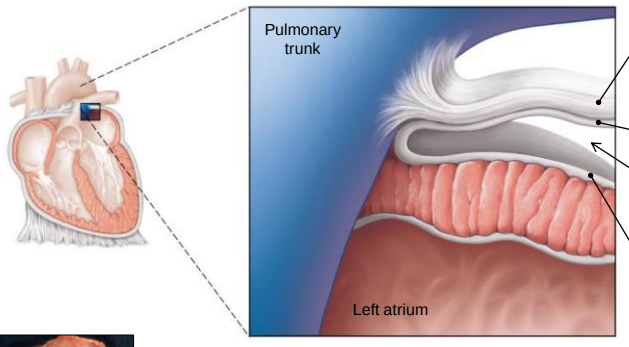


Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figures 18.1 / 18.4

Heart:

Pericardial sac: Double-walled sac enclosing the heart

Cardiac Tamponade:
Compression of heart due to fluid / blood build up in pericardial cavity

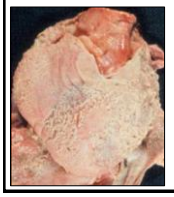


Fibrous pericardium
- Protects heart
- Anchors heart
- Prevents overfilling

Parietal pericardium

Pericardial cavity
- Contains serous fluid (friction-free environment)

Visceral pericardium



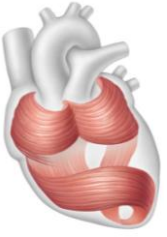
Pericarditis:
Inflammation of the pericardial sac

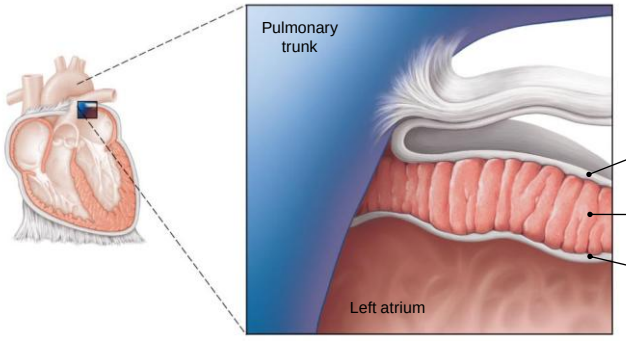
Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.2

Heart:

Heart layers:

- Anchors cardiac fibers
- Reinforces heart structures
- Directs electrical signals





Epicardium
- Often infiltrated with fat

Myocardium
- Contains fibrous skeleton

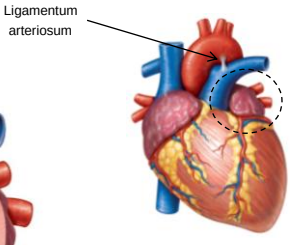
Endocardium

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figures 18.2 / 18.3

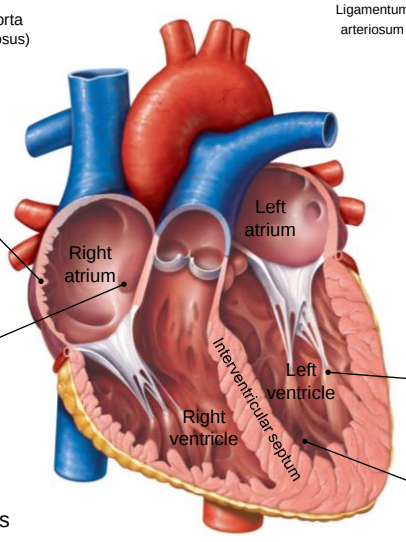
Heart – Chambers, Vessels & Valves

Atria:
- Receiving chambers
- Small, thin-walled

Ligamentum arteriosum:
Remnant of fetal duct between aorta and pulmonary truck (ductus arteriosus)



Pectinate muscles:
Muscle bundles; assist in atrial contraction



Auricles: ('little ears')
Increase atrial volume

Fossa ovalis:
Shallow depression; remnants of hole between atria in fetal heart

Papillary muscle:
Cone-like muscle; assists in valve closure

Trabeculae carneae:
Muscle ridges; assist in maintaining momentum

Ventricles:
- Discharging chambers
- Large, thick-walled

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.4

Heart – Chambers, Vessels & Valves

(largest artery in body)

Aorta
Carries blood to body

Superior vena cava
Returns blood above diaphragm

Pulmonary veins (4)
Returns blood from lungs

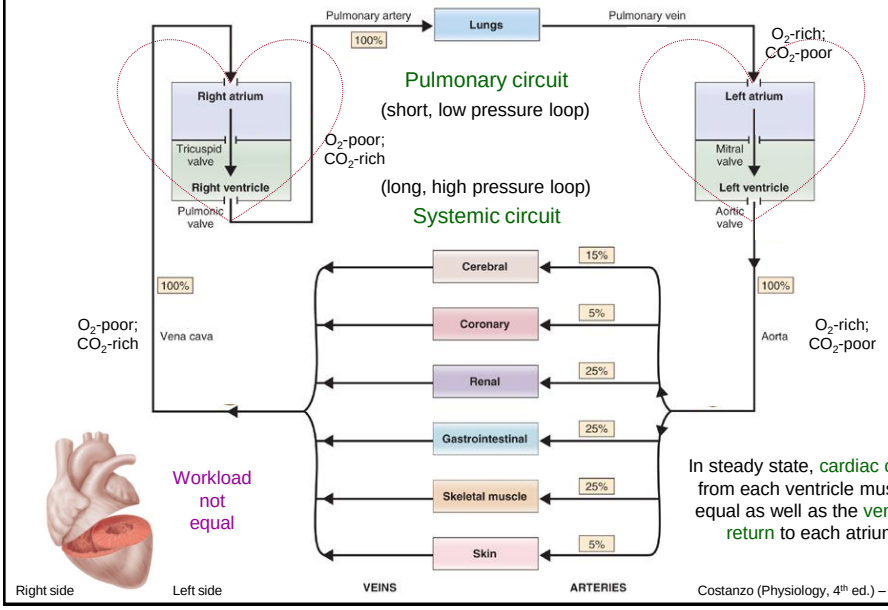
Pulmonary trunk
Carries blood to lungs

Inferior vena cava
Returns blood below diaphragm

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.4

Heart – Chambers, Vessels & Valves

Side-by-side pumps of the heart serve separate circuits



Costanzo (Physiology, 4th ed.) – Figure 4.1

Heart – Chambers, Vessels & Valves

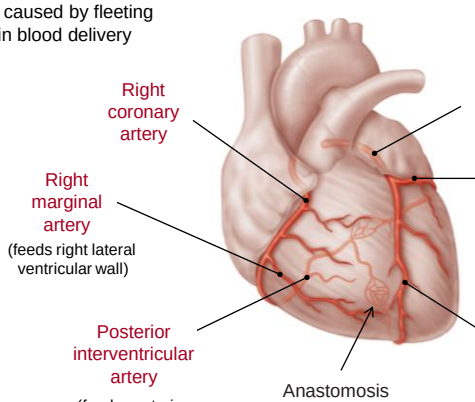
Blood in the atria / ventricles provides little nourishment to heart tissue

How does the heart itself get nourishment?

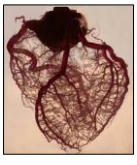
Answer: Coronary circulation

Angina pectoris:
Thoracic pain caused by fleeting deficiency in blood delivery

(Heart attack)
Myocardial infarction:
Myocardial cell death resulting from prolonged coronary blockage



1/200 body's mass; 1/20 body's blood supply



Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.7

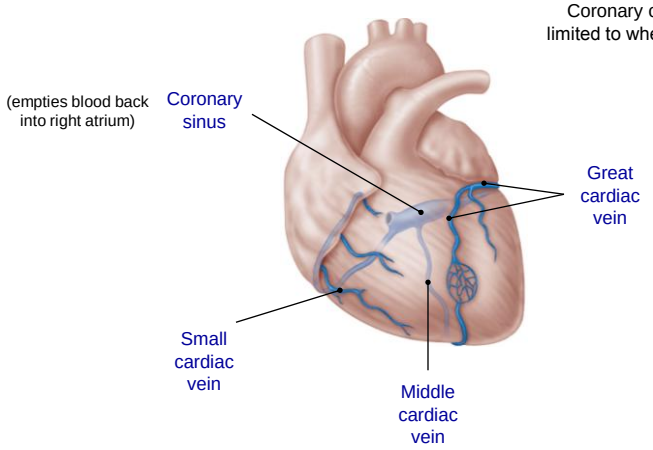
Heart – Chambers, Vessels & Valves

Blood in the atria / ventricles provides little nourishment to heart tissue

How does the heart itself get nourishment?

Answer: Coronary circulation

Coronary circulation delivery limited to when heart is relaxed…

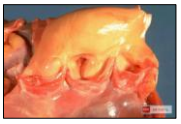


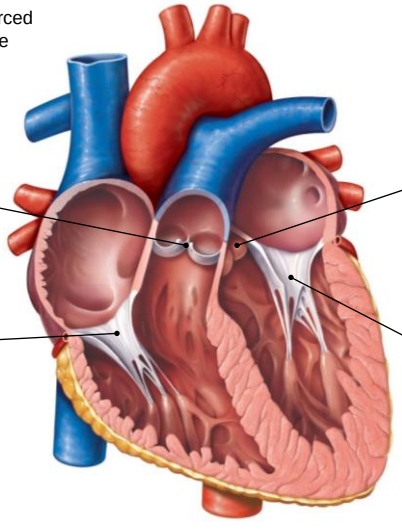
Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.7

Heart – Chambers, Vessels & Valves

Blood flows through the heart in a single direction due to the presence of valves

Cusp:
Flap of endocardium reinforced by connective tissue core



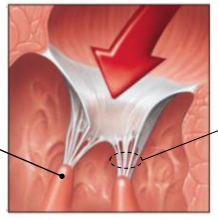


Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.4

Heart – Chambers, Vessels & Valves

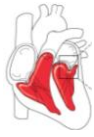
Valves open / close based on pressure differences

Atrioventricular valves
(prevent backflow into atria)



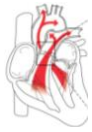
Papillary muscle

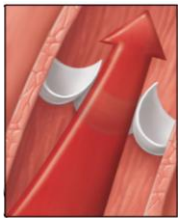
(collagen cords)
Chordae tendineae



(papillary muscles tighten)

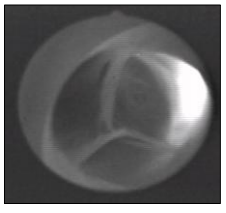
Semilunar valves
(prevent backflow into ventricles)





Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figures 18.9 / 18.10

Heart – Chambers, Vessels & Valves



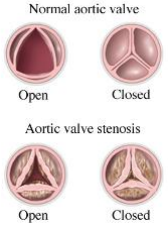
Aortic semilunar valve (pig)

Pathophysiology:

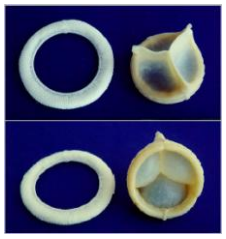
Valvular Regurgitation:
Valve does not close properly; blood regurgitated

Causes:
- Congenially deformed valve
- Post-inflammatory scarring
- Infective endocarditis
- Rupture of cord / muscle

Valvular stenosis:
Valve flaps become stiff; opening constricted



Causes:
- Congenially deformed valve
- Post-inflammatory scarring
- Calcification of valve

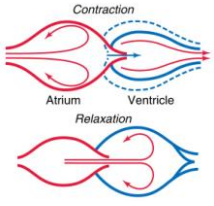


Treatment = Valve replacement

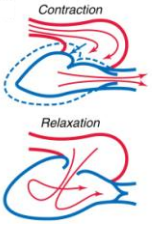
Heart – Chambers, Vessels & Valves

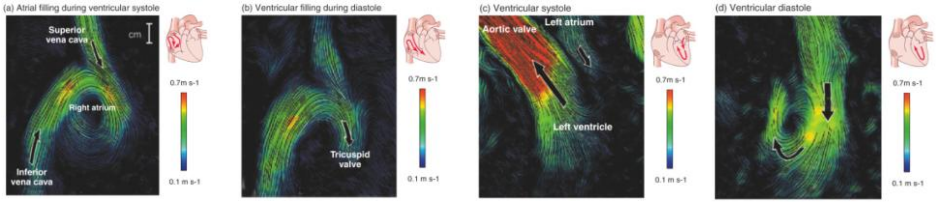
Heart designed to create complex flow patterns

(direct / maintain blood momentum)



1) Chambers arranged in loop pattern
2) Delivery vessels curved
3) Grooves / ridges within chambers



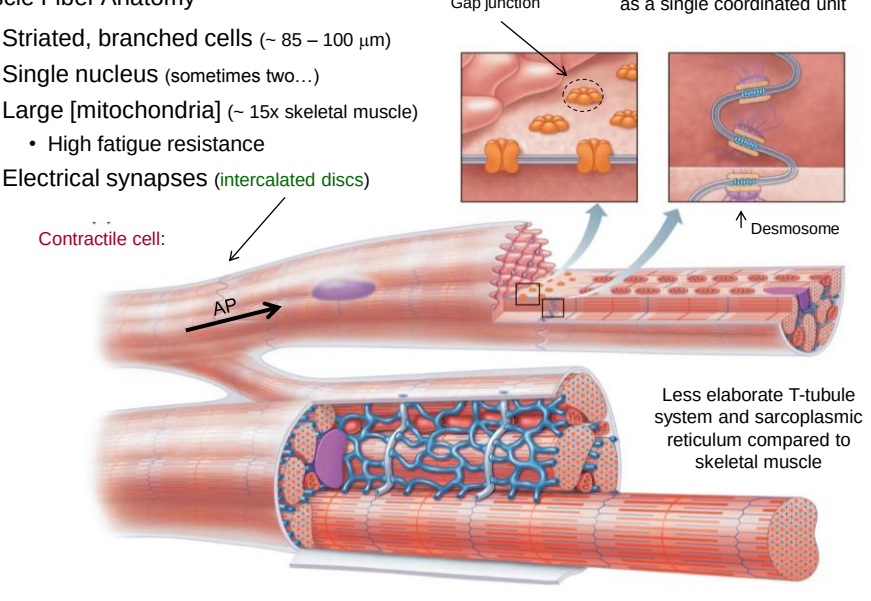


Randall et al. (Animal Physiology, 5th ed.) – Figures 12.4 / 12.10

Muscle Fiber Anatomy

- Striated, branched cells (~ 85 – 100 μm)
- Single nucleus (sometimes two…)
- Large [mitochondria] (~ 15x skeletal muscle)
  - High fatigue resistance
- Electrical synapses (intercalated discs)

Functional syncytium:
The entire myocardium behaves as a single coordinated unit



Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure18.11

Cardiac Electrophysiology

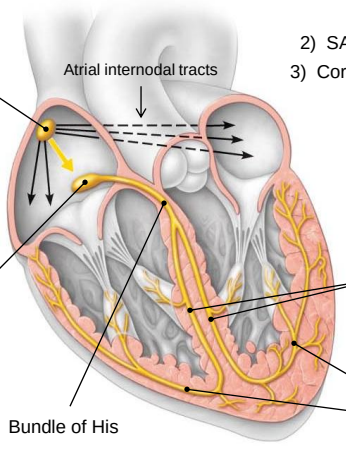
Conducting cells:
Cardiac cells specialized to quickly spread action potentials across myocardium
- Weak force generators

System allows for orderly, sequential depolarization and contraction of heart

Intrinsic Conduction System:

Normal sinus rhythm:
1) AP originates at SA node
2) SA node fires at 60 – 100 beats / min
3) Correct myocardial activation sequence

Sinoatrial node: (SA node)
- Located in right atrial wall
- Initiates action potentials (APs)
  - Pacemaker (~ 80 beats / min)

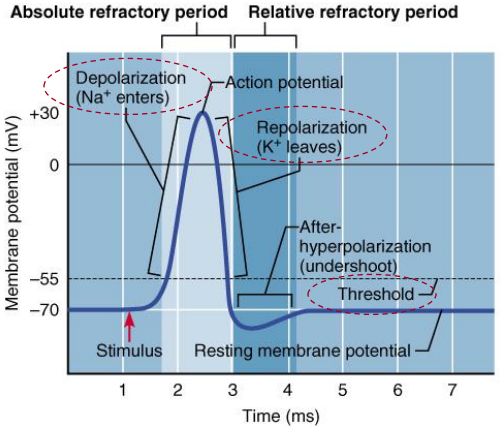


Atrioventricular node: (AV Node)
- Connects atria to ventricles
- Slowed conduction velocity
  - Ventricular filling

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.14

Cardiac Electrophysiology

The concepts applied to cardiac APs are the same concepts as applied to APs in nerves / skeletal muscle

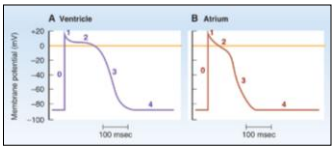


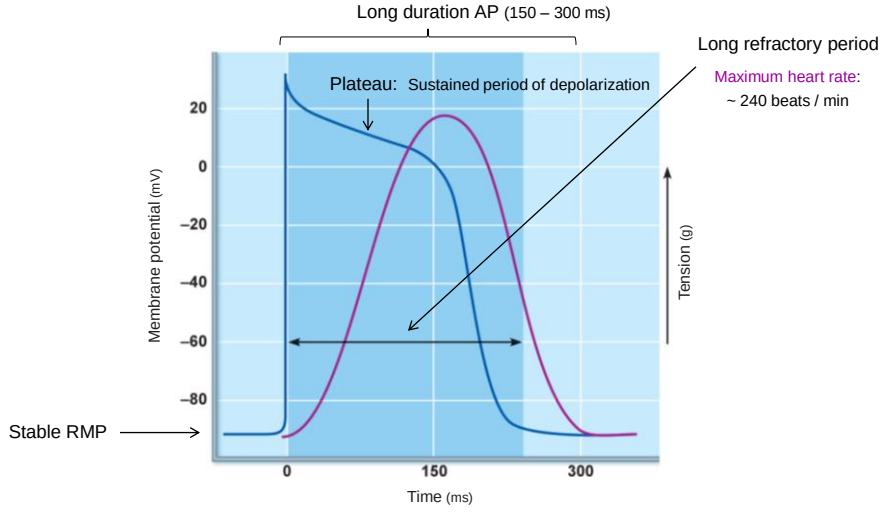
Review:
- Membrane potential determined by relative conductances / concentrations of permeable ions
- Ions flow down electrochemical gradient toward equilibrium potential (Nernst equation)
- Membrane potential expressed in mV; inside cell expressed relative to outside
- Resting membrane potential determined primarily by K^+ ions (leaky K^+ gates at rest)
- Na^+ / K^+ pumps maintain [gradients] across membranes
- Changes in membrane potential caused by flow of ions into / out of cell
- Threshold potential represents the point at which a depolarization even becomes self-sustaining (voltage-gated channels)

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 11.14

Cardiac Electrophysiology

APs of Atria, Ventricles & Purkinje System:





Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.12

Costanzo (Physiology, 4th ed.) – Figure 4.12

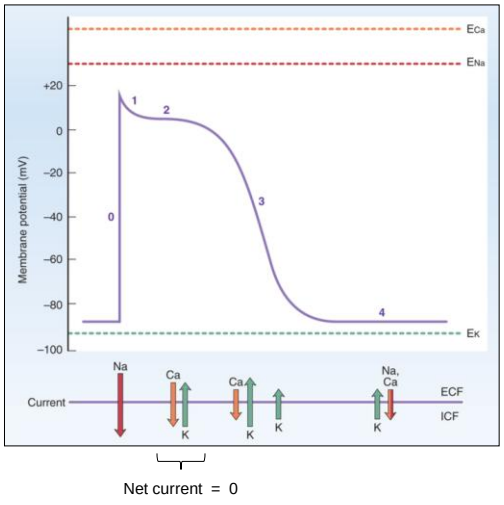
Cardiovascular System – Heart

g_X = conductance

VG = voltage-gated

Cardiac Electrophysiology

APs of Atria, Ventricles & Purkinje System:



Costanzo (Physiology, 4th ed.) – Figure 4.13

Phases of the Action Potential:

Phase 0 – Upstroke

- Period of rapid depolarization
  - Na^+ enters via VG channels (↑ g_{Na})

Phase 1 – Initial repolarization

- Brief period of repolarization
  - Na^+ channels close (↓ g_{Na})
  - K^+ exits via VG channels (↑ g_K)

Steep electrochemical gradient

Phase 2 – Plateau

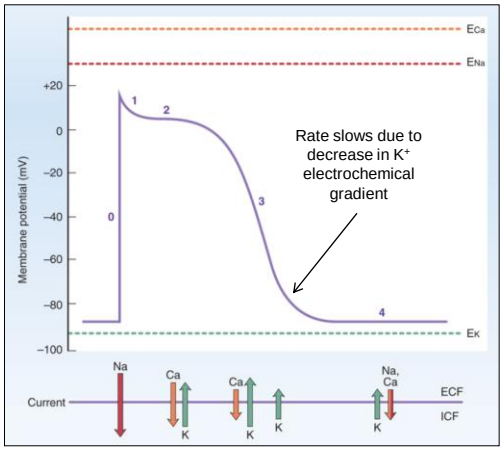
- Stable, depolarized membrane potential
  - K^+ exits via VG channels (↑ g_K)
  - (L-type) Ca^{++} enters via VG channels (↑ g_{Ca})

Ca^{2+} entry initiates release of more Ca^{2+} from intracellular stores
(excitation-contraction coupling)

Cardiac Electrophysiology

g_X = conductance
VG = voltage-gated

APs of Atria, Ventricles & Purkinje System:



Net current = 0

Costanzo (Physiology, 4th ed.) – Figure 4.13

Phases of the Action Potential:

Phase 3 – Repolarization

- Period of rapid repolarization
  - Ca^{2+} cannels close (↓ g_{Ca})
  - K^+ exits via VG channels (↑ g_K)

Phase 4 – Resting membrane potential

- Membrane potential stabilizes
  - All VG channels closed
  - K^+ exits via "leaky" channels
  - Na^+ / K^+ pumps restore [gradients]

Changes in RMP (due to [gradient] issues) directly affect responsiveness of heart

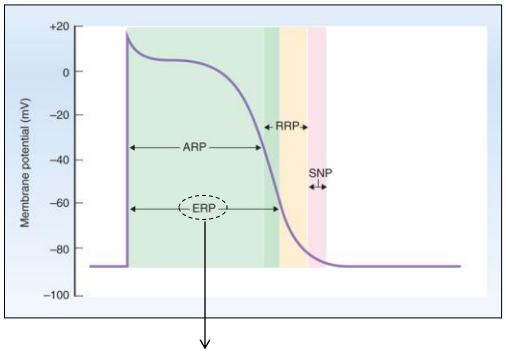
# of VG Na^+ channels available to respond decreases as RMP becomes more (+)

Cardiac dysrhythmia = irregular heartbeat

Cardiovascular System – Heart

Cardiac Electrophysiology

APs of Atria, Ventricles & Purkinje System:



Effective refractory period (ERP)
(not enough Na^+ channels have recovered)

Costanzo (Physiology, 4th ed.) – Figure 4.15

Refractory Periods:

Absolute refractory period (ARP)

- Na^+ channels closed (reset at ~ -50 mV)

Relative refractory period (RRP)

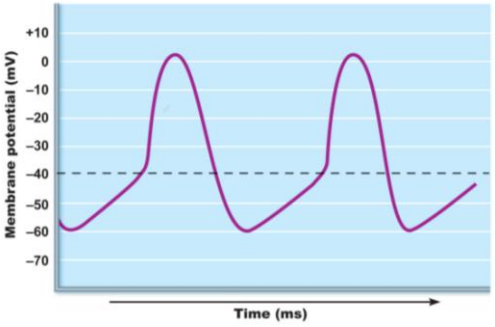
- Greater than normal stimulus required to generate AP (some Na^+ channels recovered)

Supranormal period (SNP)

- Cell is more excitable than normal due
  - Full Na^+ channel recovery
  - Potential closer to threshold than at rest

Cardiac Electrophysiology

APs of the Sinoatrial Node:



Pacemaker of the Heart:

1) Exhibits automaticity (spontaneous AP generation)
2) Unstable resting membrane potential
3) No sustained plateau

Phase 0 – Upstroke

- Slower than other cardiac tissue (T-type)
- Ca^{++} enters via VG channels (↑ g_{Ca})

Phase 1 / Phase 2

Absent

Phase 3 – Repolarization

- Similar to other cardiac cells

Phase 4 – Spontaneous depolarization

- Accounts for automaticity of SA node
- Na^{+} enters via VG channels (↑ g_{Na})
  - Open via repolarization event

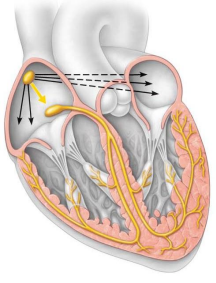
Once threshold reached, VG Ca^{2+} channels open (return to Phase 0)

- Rate of depolarization sets heart rate

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.13

Cardiac Electrophysiology

Other myocardial cells also have the capacity for spontaneous phase 4 depolarization; these are called latent pacemakers



Location	Intrinsic firing rate (impulses / min)
Sinoatrial node	70 – 80
Atrioventricular node	40 – 60
Bundle of His	40
Purkinje fibers	15 – 20

The pacemaker with the fastest rate controls the heart rate

Overdrive suppression:
Latent pacemakers own capacity to spontaneously depolarize is suppressed by the SA node.

Ectopic pacemaker: Latent pacemaker takes over and becomes the pacemaker

1) SA node firing rate decreases (e.g., damage / drug suppression)
2) Intrinsic rate of latent pacemakers increases
3) Blockage in normal conduction pathway (e.g., disease)

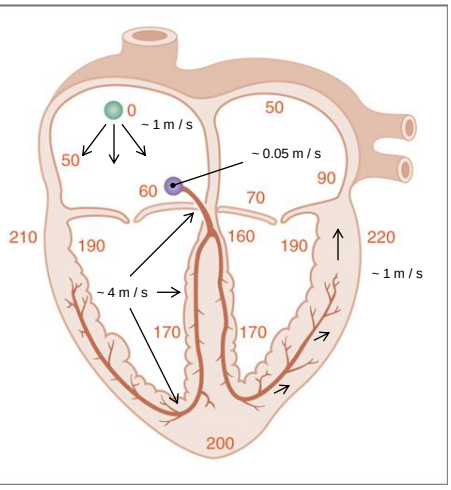
Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.14

Cardiac Electrophysiology

Allows for proper timing of heart events

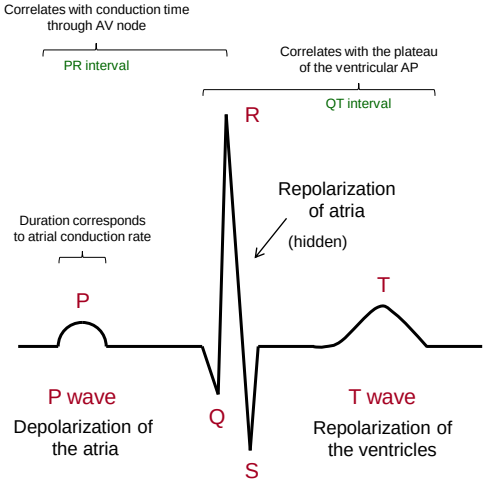
Conduction velocity (speed at which APs propagate in tissues) differs among myocardial tissues

Conduction Velocity	
Atria	1 m/sec
AV node	0.01–0.05 m/sec
Only connection: atria → ventricles	
His-Purkinje	2–4 m/sec
Ventricle	1 m/sec

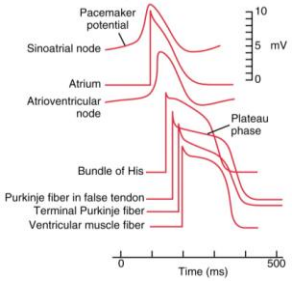


Costanzo (Physiology, 4th ed.) – Figure 4.14

Cardiac Electrophysiology



Electrocardiogram (ECG or EKG):
Graphical recording of electrical currents generated and transmitted through heart

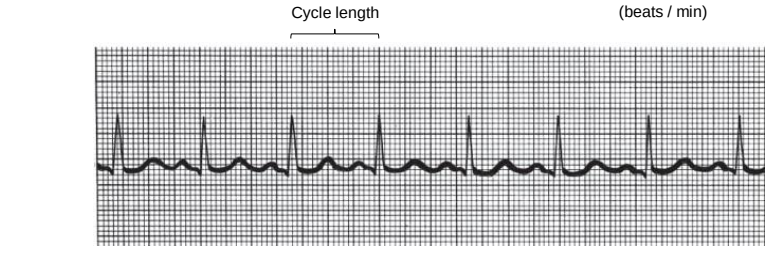


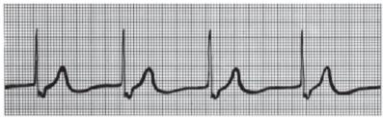
Randall et al. (Animal Physiology, 5th ed.) – Figure12.8

Cardiac Electrophysiology

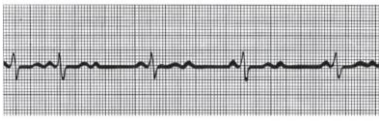
Cycle length:
Time between one R wave and the next

Heart rate = 60 / cycle length
(beats / min)

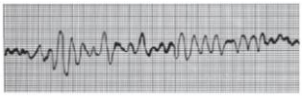




Junctional Rhythm:
SA node nonfunctional



Heart block:
Poor conduction at AV node



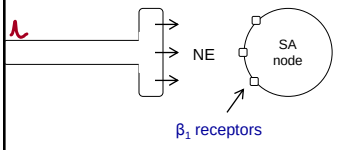
Fibrillation:
Out-of-phase contractions

Cardiac Electrophysiology

The autonomic nervous system can directly affect the heart rate;
these effects are called chronotropic effects

Positive chronotrophic effects:
(increase heart rate)

- Under sympathetic control



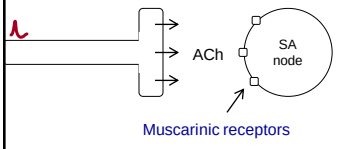
β_1 receptors

Leads to ↑ g_{Na}; cells reach threshold more rapidly

Pharmacology:
β-blockers (e.g., propanolol)

Negative chronotrophic effects:
(decrease heart rate)

- Under parasympathetic control

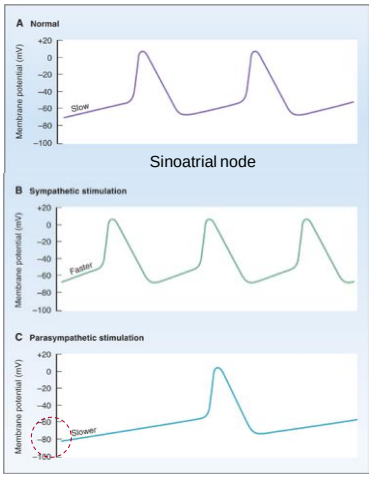


Muscarinic receptors

Leads to ↓ g_{Na}; cells reach threshold less rapidly

Leads to ↑ g_K; cells hyperpolarized during repolarization stage
(further from threshold)

Recall: spontaneous depolarization = VG Na^+ channels



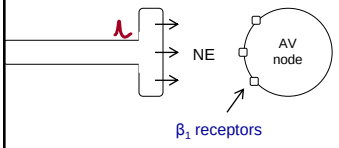
Costanzo (Physiology, 4th ed.) – Figure 4.16

Cardiac Electrophysiology

The autonomic nervous system can also directly conduction velocity at the AV node; these effects are called dromotropic effects

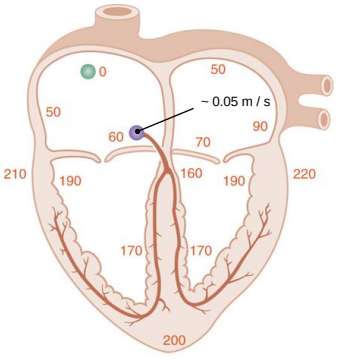
Positive dromotropic effects:
(increase conduction velocity)

- Under sympathetic control



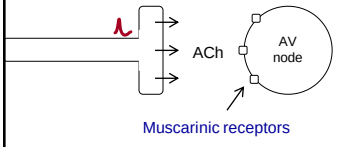
Leads to ↑ g_{Ca}; cells depolarize more rapidly following threshold

Recall: AV node slow point in intrinsic conduction pathway



Negative dromotropic effects:
(decrease conduction velocity)

- Under parasympathetic control



Leads to ↓ g_{Ca} & ↑ g_K; cells depolarize more slowly following threshold

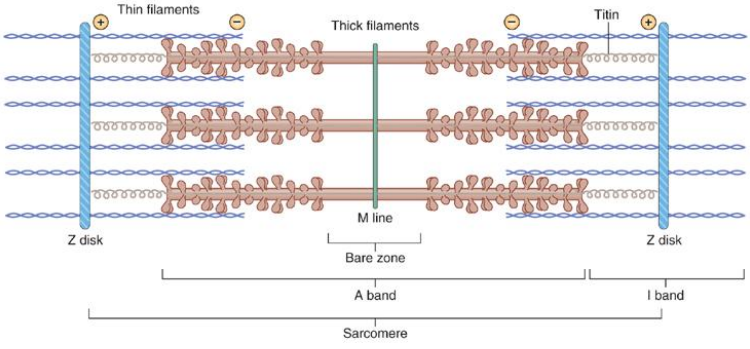
Heart block:
Signals fail to be conducted at AV node

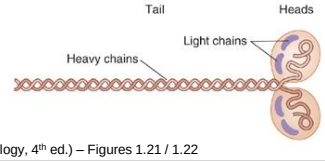
Costanzo (Physiology, 4th ed.) – Figure 4.14

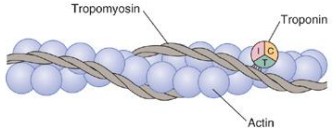
Cardiac Muscle Contraction

Contraction occurs according to sliding filament model

The basic contractile machinery between cardiac and smooth muscle is similar







Costanzo (Physiology, 4th ed.) – Figures 1.21 / 1.22

Cardiac Muscle Contraction

Excitation-contraction coupling translates the action potential into the production of tension

Cardiac AP initiated in membrane

Dihydropyridine receptors

- Spreads to cell interior via T-tubules
- Triggers L-type Ca^{2+} channels in membrane

Ryanodine receptors

Ca^{2+} enters cell (plateau phase)

- Triggers Ca^{2+}-induced Ca^{2+} release from SR

Ca^{2+} floods cytoplasm

Mechanisms for changing tension production:

1) ↑ Ca^{2+} in SR = ↑ Ca^{2+} release to cytoplasm
2) ↑ Ca^{2+} inward current = ↑ Ca^{2+} in cytoplasm

Cross-bridging cycling initiated

- Ca^{2+} binds to troponin C; cross-bridging occurs

Tension

The magnitude of the tension developed is proportional to the intracellular [Ca^{2+}]

1) Ca^{2+} reaccumulated in SR
   - Ca^{2+} ATPase
2) Ca^{2+} extruded from cell
   - Ca^{2+} ATPase
   - Ca^{2+} / Na^{+} exchanger

Relaxation

Similar to Costanzo (Physiology, 4th ed.) – Figure 4.18

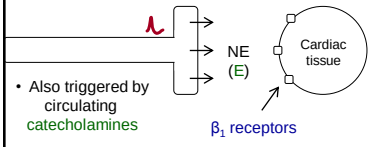
Cardiac Muscle Contraction

Inotropism:
Intrinsic ability of myocardial cells to develop force at a given length

The autonomic nervous system can directly affect heart contractility; these effects are called inotropic effects

Positive inotropic effects:
(increase contractility)

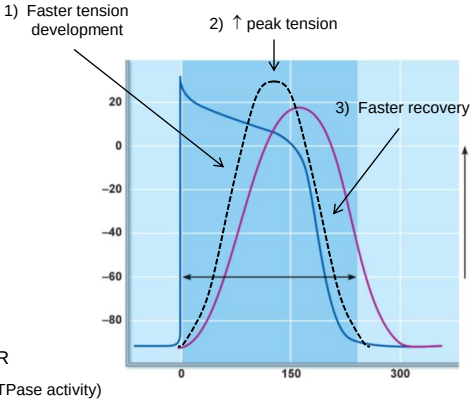
- Under sympathetic control



- Also triggered by circulating catecholamines

Mechanisms of action:

1) Phosphorylation of Ca^{2+} channels in sarcolemma
   - ↑ Ca^{2+} enters during plateau / released from SR
2) Phosphorylation of phospholamban (regulates Ca^{2+} ATPase activity)
   - ↑ uptake / storage of Ca^{2+} in SR
     - Faster relaxation time
     - Increased peak tension during subsequent 'beats'



- Shorter twitch time allows for more time for ventricle to fill
- Increased tension generation equals stronger contraction

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.12

Inotropism:
Intrinsic ability of myocardial cells to develop force at a given length

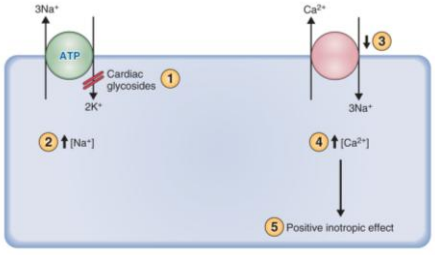
Cardiac Muscle Contraction

The autonomic nervous system can directly affect heart contractility; these effects are called inotropic effects

Positive inotropic effects:
(increase contractility)

Digitalis purpurea

Digoxin
Digitoxin
Ouabain



1) Cardiac glycosides inhibit Na^+-K^+ ATPase
2) Intracellular $[Na^+]$ increases
3) Change in Na^+ gradient slows down Ca^{2+}-Na^+ exchanger
4) Intracellular $[Ca^{2+}]$ increases
5) ↑ $[Ca^{2+}]$ = ↑ tension development

Cardiac glycosides are a class of drugs that act as positive inotropic agents

Used extensively for the treatment of congestive heart failure

Costanzo (Physiology, 4th ed.) – Figure 4.20

Cardiovascular System – Heart

Inotropism:
Intrinsic ability of myocardial cells to develop force at a given length

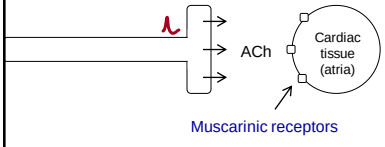
Cardiac Muscle Contraction

The autonomic nervous system can directly affect heart contractility; these effects are called inotropic effects

Only affect myocardium in atria

Negative inotropic effects:
(decrease contractility)

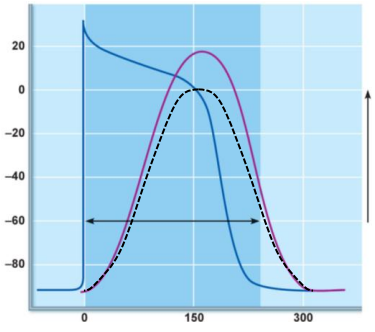
- Under parasympathetic control



Muscarinic receptors

- ACh decreases inward Ca^{2+} current during plateau
- ACh increases outward K^+ current (shorten plateau phase)

Both ↓ Ca^{2+} entering cell and thus the amount of Ca^{2+} available for tension development



Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.12

Cardiac Muscle Contraction

Changes in heart rate also produce changes in cardiac contractility

Example:

Increase in heart rate = Increase in cardiac contractility

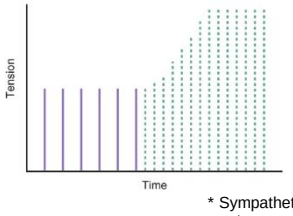
1) ↑ heart rate = ↑ APs per unit time = ↑ total amount of Ca^{2+} entering cell per unit time

AND

2) ↑ Ca^{2+} entering cell per unit time = ↑ accumulation of Ca^{2+} in SR for future release

Positive staircase effect

As heart rate increases, the tension developed on each beat increases stepwise to a maximal value



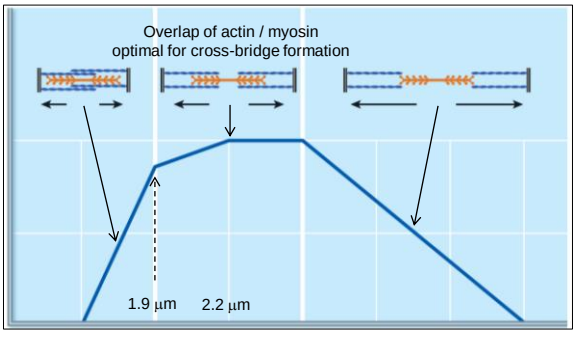
* Sympathetic input will enhance response

Costanzo (Physiology, 4th ed.) – Figure 4.19

Cardiac Muscle Contraction

The maximum tension that can be developed by a myocardial cell depends on its resting length (similar to skeletal muscle)

Sarcomere length of ~ 2.2 μm = L_{max} for cardiac muscle



Myocardium cells maintain a 'working length' of ~ 1.9 μm

Additional Length-dependent Mechanisms:

1) Increasing muscle length increases Ca^{2+}-sensitivity of troponin C
2) Increasing muscle length increases Ca^{2+} release from SR

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 9.22

Cardiac Muscle Contraction

Systole = Contraction of heart
Diastole = Relaxation of heart

Ventricular function is described by several parameters:

1) Cardiac Output: Total volume of blood ejected by each ventricle per unit time (usually one minute)

Cardiac output (ml / min) = Heart rate (beats / min) x Stroke volume (ml / beat)

2) Stroke Volume: Volume of blood ejected by each ventricle per heart beat

Stroke volume (ml) = End diastolic volume (ml) - End systolic volume (ml)

3) Ejection Fraction: Fraction of the end diastolic volume ejected in each stroke volume

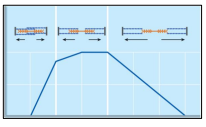
$$\text{Ejection fraction} = \frac{\text{Stroke volume (ml)}}{\text{End diastolic volume (ml)}}$$

Heart rate = 75 beats / min
End diastolic volume = 140 ml
End systolic volume = 70 ml

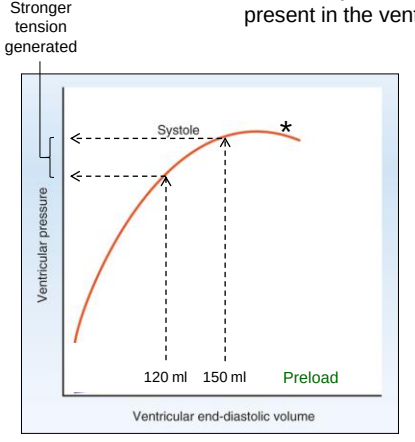
Calculate:

Stroke volume: 70 ml
Ejection fraction: 0.50
Cardiac output: 5250 ml / min

Cardiac Muscle Contraction



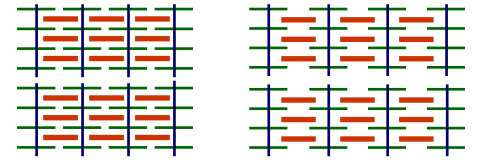
Frank-Starling Law of the Heart:
The volume of blood ejected by the ventricle depends on the volume present in the ventricle at the end of diastole



(end diastolic cardiac fiber length)

The more blood that collects in the ventricle, the more the cardiac cells are stretched

optimal overlap = optimal tension



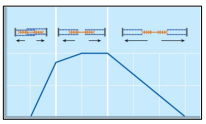
Sarcomere length = ~1.9 μm
Sarcomere length = ~ 2.2 μm

Preload:
The resting length from which cardiac muscle contracts

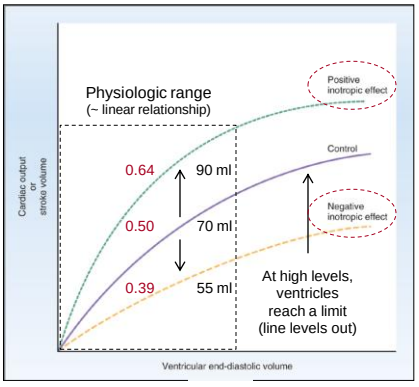
* Cardiac muscle normally 'held' on the ascending limb of the length-tension curve; much 'stiffer' than skeletal muscle

Costanzo (Physiology, 4th ed.) – Figure 4.21

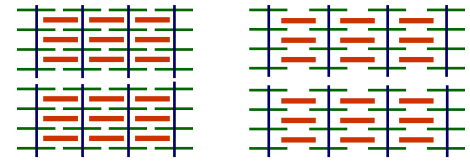
Cardiac Muscle Contraction



Frank-Starling Law of the Heart:
The volume of blood ejected by the ventricle depends on the volume present in the ventricle at the end of diastole



optimal overlap = optimal tension



Sarcomere length = ~1.9 μm

Sarcomere length = ~ 2.2 μm

This relationship ensures that the volume the heart ejects in systole equals the volume it receives in venous return

Positive inotropic effect = ↑ ejection fraction

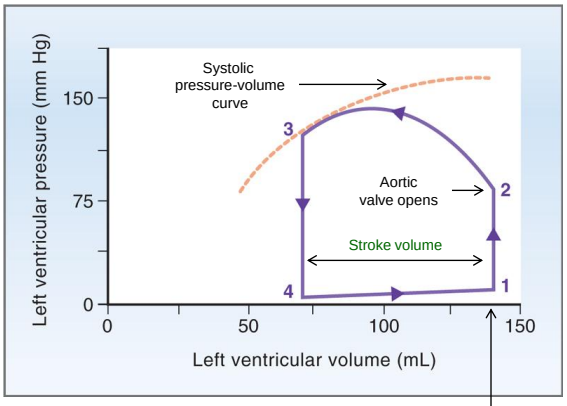
Negative inotropic effect = ↓ ejection fraction

Costanzo (Physiology, 4th ed.) – Figure 4.22

Cardiac Muscle Contraction

A ventricular pressure-volume loop allows for the function of a ventricle to be observed for a single heart beat

Left ventricle



- Atrium 'tops off' blood in ventricle
- Ventricle relaxed (late diastole)

Isovolumetric contraction (1 → 2)

- Ventricle activates (systole)

↓ space + no change in blood volume = ↑ pressure

- Aortic / mitral valves closed

$P_{aorta} > P_{ventricle} > P_{atrium}$

Ventricular ejection (2 → 3)

- Aortic valve opens

$P_{ventricle} > P_{aorta}$

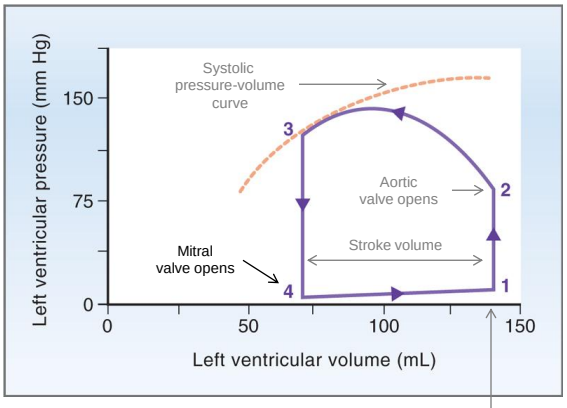
- Blood rapidly ejected (↓ volume)

Costanzo (Physiology, 4th ed.) – Figure 4.23

Cardiac Muscle Contraction

A ventricular pressure-volume loop allows for the function of a ventricle to be observed for a single heart beat

Left ventricle



- Atrium 'tops off' blood in ventricle
- Ventricle relaxed (diastole)

Isovolumetric relaxation (3 → 4)

- Ventricle relaxes (early diastole)

↑ space + no change in blood volume = ↓ pressure

- Aortic / mitral valves closed

$P_{aorta} > P_{ventricle} > P_{atrium}$

Ventricular filling (4 → 1)

- Mitral valve opens

$P_{atrium} > P_{ventricle}$

- Ventricle refills

Slight pressure increase due to passive filling of compliant ventricle

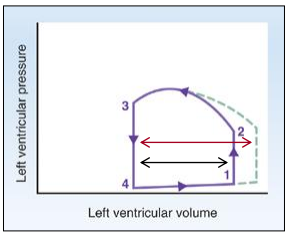
Costanzo (Physiology, 4th ed.) – Figure 4.23

Cardiovascular System – Heart

Cardiac Muscle Contraction

A ventricular pressure-volume loop allows for the function of a ventricle to be observed for a single heart beat

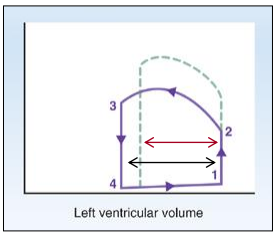
Increased preload



- Increased end diastolic volume
- Increased stroke volume

(Frank-Starling Law)

Increased afterload

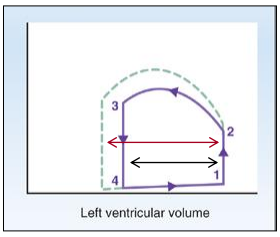


Afterload:

Pressure in the vessel leaving the heart (e.g., aorta) that must be overcome to eject blood

- Increased internal pressure
- Decreased stroke volume

Increased contractility



- Increased tension / pressure
- Decreased end systolic volume
- Increased stroke volume

Costanzo (Physiology, 4th ed.) – Figure 4.24

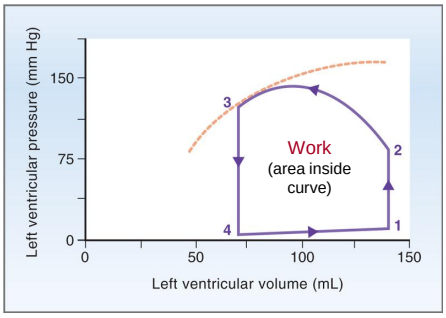
Cardiac Muscle Contraction

Force = aortic pressure
Distance = stroke volume

The stroke work is defined as the work the heart performs on each beat

Work = force x distance

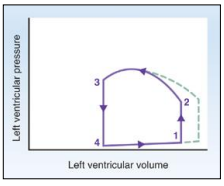
Left ventricle



Cardiac minute work:
Work performed by the heart during a unit time (e.g., minute)

Force = aortic pressure (pressure work)
Distance = cardiac output (volume work)

Increases in cardiac output or increases in aortic pressure will increase work of the heart



(e.g., preload)

Costanzo (Physiology, 4th ed.) – Figures 4.23 / 4.24

Cardiac Muscle Contraction

Aortic stenosis results in greatly increased O_2 consumption, even though cardiac output reduced

The myocardial O_2 consumption rate correlates directly with the cardiac minute work

↑ cardiac minute work = ↑ O_2 consumption

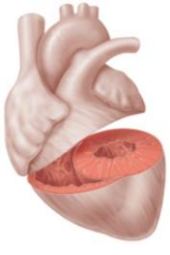
HOWEVER

(aortic pressure)

The largest percentage of O_2 consumption is for pressure work

THUS

Mean aortic pressure = 100 mm Hg
Mean pulmonary pressure = 15 mm Hg



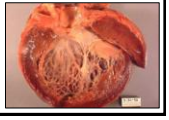
Left ventricle thicker than right ventricle

Law of Laplace:
In a sphere (e.g., heart), pressure correlates directly with tension and wall thickness and correlates inversely with radius

P = Pressure
H = Thickness (height)
T = Tension
R = Radius

$$P = \frac{2HT}{r}$$

What are the ramifications if a person exhibits systemic hypertension?



Cardiac Muscle Contraction

Recall:
Cardiac output equals the total volume of blood ejected by a ventricle per unit time

The cardiac output can also be measured using the Fick principle
(conservation of mass)

All measurable qualities →

In the steady state, the rate of O_2 consumed by the body must equal the amount of O_2 leaving the lungs (pulmonary veins) minus the amount of O_2 returning to the lungs (pulmonary artery)

$$O_2 \text{ consumption} = CO_{\text{left ventricle}} \times [O_2]_{\text{pulmonary vein}} - CO_{\text{right ventricle}} \times [O_2]_{\text{pulmonary artery}}$$

equal

Solve for cardiac output:

$$\text{Cardiac Output} = \frac{O_2 \text{ consumption}}{[O_2]_{\text{pulmonary vein}} - [O_2]_{\text{pulmonary artery}}}$$

Fick principle also applicable to measuring blood flow to individual organs

A man has a resting O_2 consumption of 250 mL O_2 / min, a femoral arterial O_2 content of 0.20 mL O_2 / mL blood, and a pulmonary arterial O_2 content of 0.15 mL O_2 / mL blood.

What is his cardiac output?

$$\text{Cardiac Output} = \frac{O_2 \text{ consumption}}{[O_2]_{\text{pulmonary vein}} - [O_2]_{\text{pulmonary artery}}}$$

$$\text{Cardiac Output} = \frac{250 \text{ mL } O_2 \text{ / min}}{0.20 \text{ mL } O_2 \text{ / mL blood} - 0.15 \text{ mL } O_2 \text{ / mL blood}}$$

$$\text{Cardiac Output} = 5000 \text{ mL / min}$$

Mechanical / Electrical Overview:

Cardiac Cycle: Mechanical and electrical events during single heart beat

Phases of the cardiac cycle:

Atrial Systole: (A)

Ventricle 'topped off' (20%)

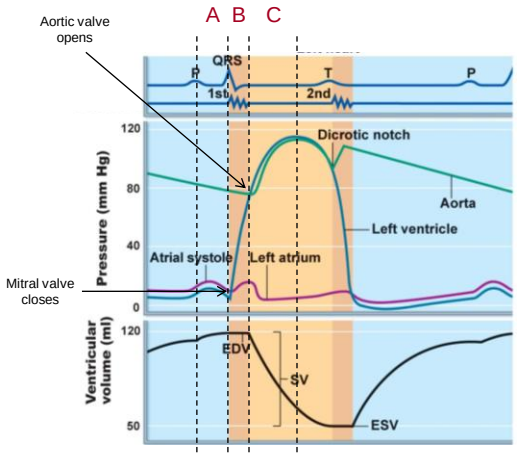
- Preceded by P wave on ECG
- Increased tension in left atrium
- Ventricular volume / pressure increases

Isovolumetric Ventricular Contraction: (B)

- Begins during QRS wave on ECG
- Ventricular pressure increases (systole)
- Mitral valve closes (1st heart sound – 'Lub')
- NO VOLUME CHANGE

Rapid Ventricular Ejection: (C)

- Aortic valve opens ($P_{ventricle} > P_{aorta}$)
- Majority of stroke volume ejected
- Aorta pressure increases
- Atrial filling begins



Left pump

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.20

Cardiovascular System – Heart

Mechanical / Electrical Overview:

Cardiac Cycle: Mechanical and electrical events during single heart beat

Phases of the cardiac cycle:

Reduced Ventricular Ejection: (D)

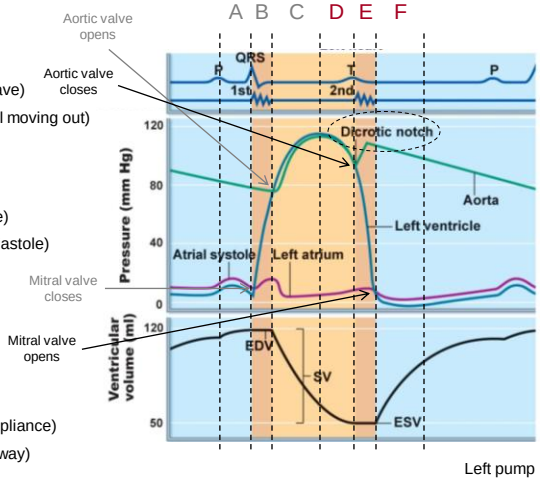
- Ventricles begin to repolarize (start of T wave)
- Ventricular / atrial pressure falls (blood still moving out)
- Atrium continues to fill (pressure rising)

Isovolumic Ventricular Relaxation: (E)

- Ventricles fully repolarize (T wave complete)
- Left ventricular pressure drops rapidly (diastole)
- Aortic valve closes (2nd heart sound – 'Dub')
- NO VOLUME CHANGE

Rapid Ventricular Filling: (F)

- Mitral valve opens ($P_{atrium} > P_{ventricle}$)
- Ventricular volume increases rapidly
- Little change in ventricular pressure (compliance)
- Aortic pressure decreases (blood carried away)



Left pump

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.20

Mechanical / Electrical Overview:

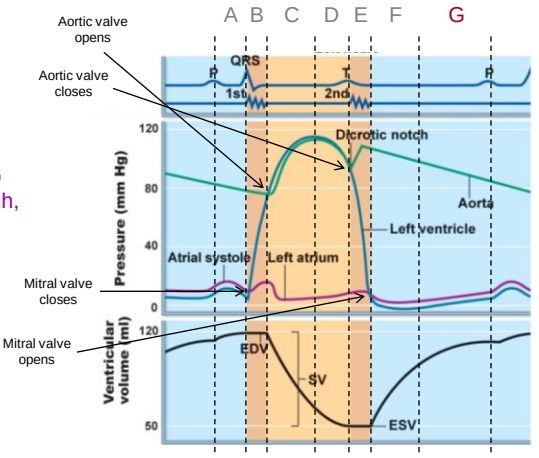
Cardiac Cycle: Mechanical and electrical events during single heart beat

Phases of the cardiac cycle:

Reduced Ventricular filling: (G)

- Longest phase of cardiac cycle
- Final portion of ventricular filling

Increase in heart rate reduces G phase interval; if heart rate too high, ventricular filling compromised



http://www.youtube.com/watch?feature=endscreen&NR=1&v=xS5twGT-epo

Left pump

Marieb & Hoehn (Human Anatomy and Physiology, 8th ed.) – Figure 18.20