

BIOL 260: Human Physiology

Spring 2020 23-205 TR, Mar. 17-19

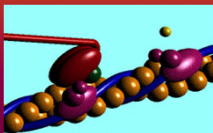
<http://accounts.smccd.edu/staplesn/biol260/>

1. Pre-Lab Writeups: Not assigned for now.
2. This Week: Heart and Vascular system (Chapters 14 and 15 ONLINE!). See video presentations to come! ☺
3. Lab: Keep up with Mastering A&P assignments, until (hopefully) live labs return. Respiratory labs are planned.
 - Blood Glucose and Urinary labs unlikely. ☹
4. Review Sessions in Lab THIS Tuesday & Thurs., on Canvas/ZOOM!!
 - a) 10am-12pm. ALL students are expected to participate during your regular lab time!
 - b) *** Review Sheet #2 will be updated by this weekend!
5. Midterm #2 on TUESDAY, 3/24 at 8:10AM on CANVAS/online!! (same interface as quizzes). Exam format will be very similar to usual.
 - Essay spaces will be given. Be VERY descriptive and explanatory as possible! You can upload digital drawings or photos of hand drawings in a separate page.
6. QUIZ #4 is DUE this Thursday!! (first attempt!).
7. Muscles lab due by Saturday!! UPLOAD to Canvas.

1

Ch. 14: Heart

1. Compare the anatomy, regulation, and mechanics of contraction in **Skeletal, Smooth, and Cardiac muscle**.
** Consider cellular connections/coordination, proteins involved, and responses to IC Ca**.



Today's Objectives: Students should be able to....

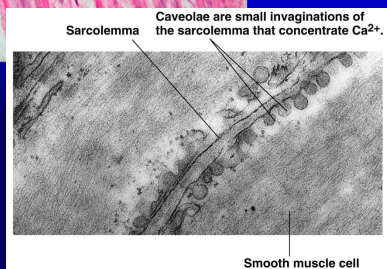
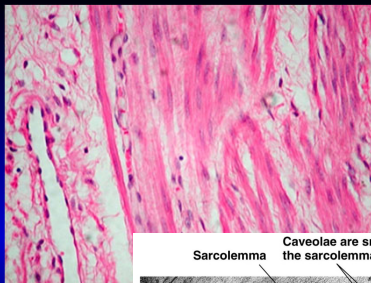
1. **Ch. 14: Contrast the purpose of the right and left halves of the heart.**
 - ❖ Draw a simple diagram showing the path of blood flow through the heart, lungs, & upper and lower body.
2. Describe the several different factors controlling the **rate and velocity of blood flow** through vessels. (*resistance: length, radius, viscosity; pressure, C-S area*)
3. Describe and diagram how the heart and blood vessels assure **one-way blood flow** through the heart, lungs, and body. (*ie: valves, pressure changes*)
4. Diagram the **how AP generates contraction in a muscle cell**. Compare/contrast cardiac muscle cell contraction with skeletal and smooth muscle.
5. Diagram a **contractile myocardial AP**. Describe refractory periods & summation in cardiac muscle.
6. Describe the **major pacemakers of the heart**, and how they spontaneously generate action potentials. Diagram the **path of heart AP conduction**, and the sequence of contraction in the four chambers.

❖ **These objectives are your HOMEWORK between classes!!!**

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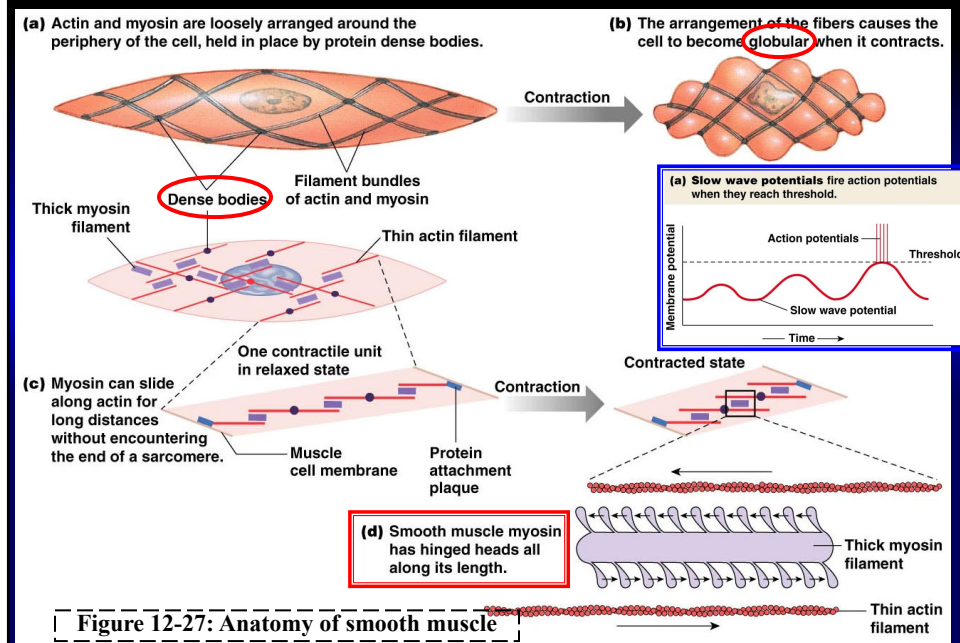
B. Smooth Muscles: Characteristics

1. Stimulation
 - a) Electrically coupled
 - Gap junctions
 - b) Hormones
 - c) Paracrines
 - d) Various receptors
 - e) **Single Unit** – gap jxns
 - f) **Multiple unit** - varicosities
2. Single tapered cells
3. **Longer actin & myosin**
 - a) Actin joined at **Dense Bodies**
 - b) Diagonal/oblique arrangement
 - c) Continuous myosin heads on thick filaments
4. Can contract without depolarization!! & **Latch State**



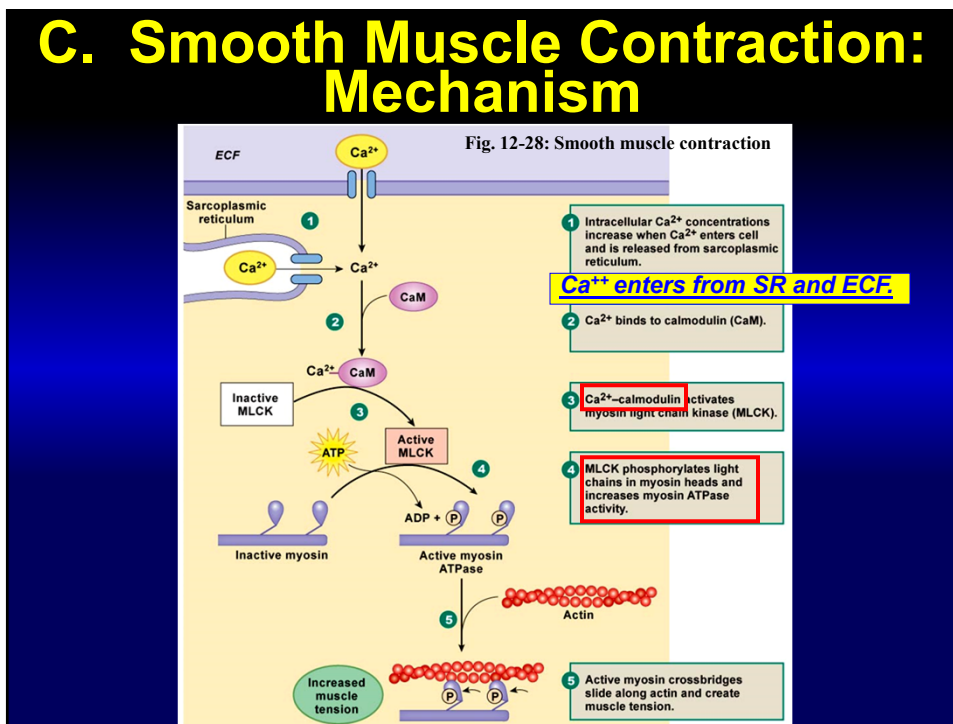
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Smooth Muscle Characteristics



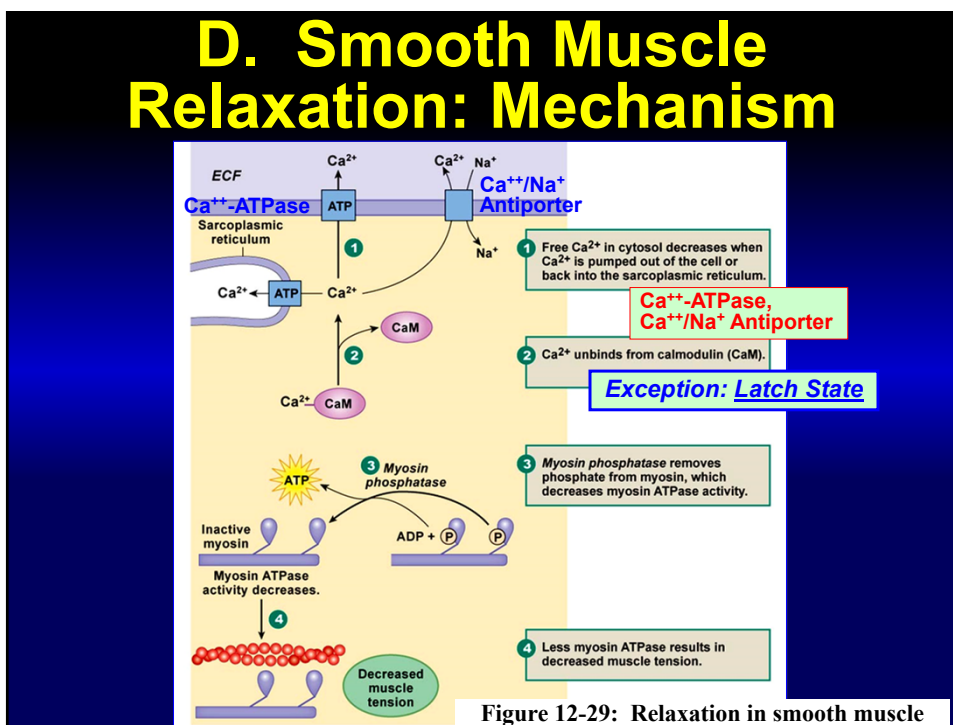
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C. Smooth Muscle Contraction: Mechanism



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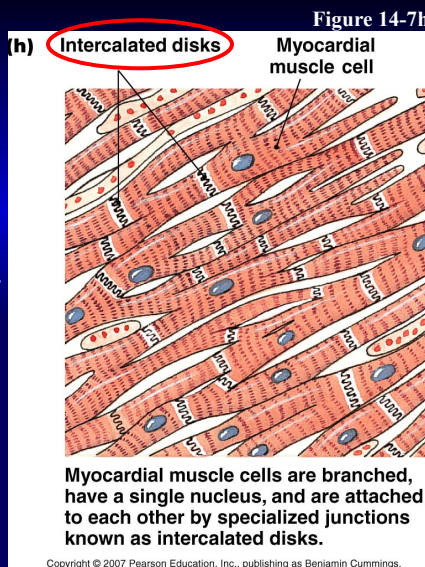
D. Smooth Muscle Relaxation: Mechanism



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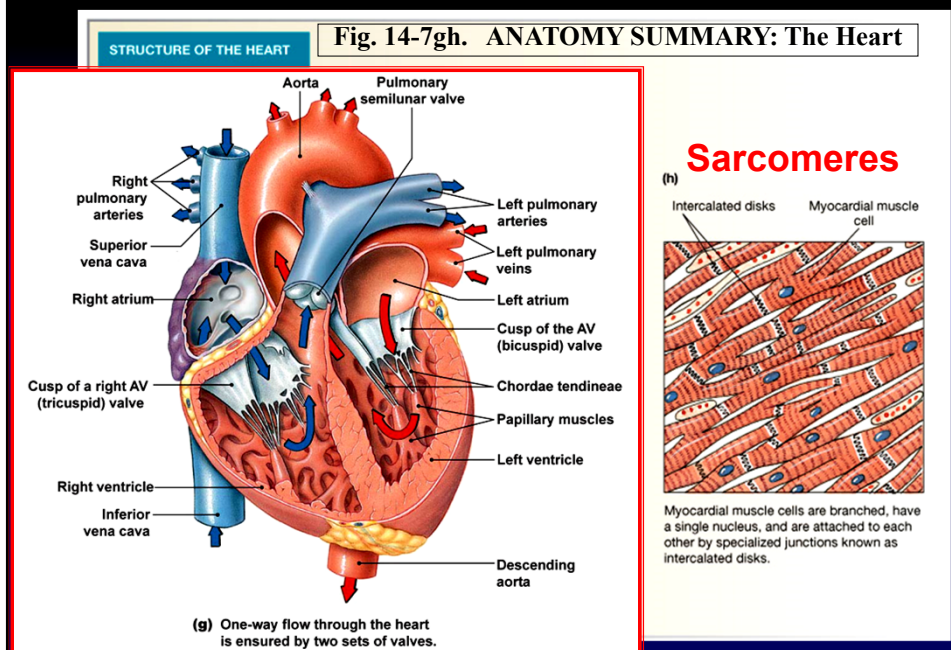
12.11) Cardiac Muscle: Contrasted to Skeletal

1. Shorter, branched fibers.
2. Single nucleus/fiber.
3. Intercalated discs between fibers.
 - *Desmosomes* allow force to be transferred
 - *Gap junctions* provide electrical connection
4. Gap junctions - *syncytia*
5. T-tubules are larger and branch.
6. Sarcoplasmic reticulum is smaller
7. Mitochondria occupy one-third of cell volume.
8. Stimulation:
 - a) *Pacemaker* (smooth too)
 - Waves of depolarization
 - ❖ Leaky ion/Ca⁺⁺ channels
 - b) Autonomic
 - Nervous & Hormonal



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Cardiac Muscle: Contrasted to Skeletal



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** Summary: Comparison of Three Muscle Types **

	SKELETAL	SMOOTH	CARDIAC
Appearance under light microscope	Striated	Smooth	Striated
Fiber arrangement	Sarcomeres	Oblique bundles	Sarcomeres
Fiber proteins	Actin, myosin; troponin and tropomyosin	Actin, myosin, tropomyosin	Actin, myosin; troponin and tropomyosin
Control	<ul style="list-style-type: none"> • Voluntary • Ca^{2+} and troponin • Fibers independent of one another 	<ul style="list-style-type: none"> • Involuntary • Ca^{2+} and calmodulin • Fibers electrically linked via gap junctions 	<ul style="list-style-type: none"> • Involuntary • Ca^{2+} and troponin • Fibers electrically linked via gap junctions
Nervous control	Somatic motor neuron	Autonomic neurons	Autonomic neurons
Hormonal influence	None	Multiple hormones	Epinephrine
Location	Attached to bones; a few sphincters close off hollow organs	Forms the walls of hollow organs and tubes; some sphincters	Heart muscle
Morphology	Multinucleate; large, cylindrical fibers	Uninucleate; small spindle-shaped fibers	Uninucleate; shorter branching fibers
Internal structure	T-tubule and sarcoplasmic reticulum	No t-tubules; sarcoplasmic reticulum reduced or absent	T-tubule and sarcoplasmic reticulum
Contraction speed	Fastest	Slowest	Intermediate
Contraction force of single fiber twitch	All-or-none	Graded	Graded
Initiation of contraction	Requires input from motor neuron	Can be autorhythmic	Autorhythmic

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Muscles: Review

- Skeletal:** Calcium initiation at Motor End Plate
- Actin/Myosin complexes:** contraction begins with Ca^{++} , ATP, troponin (TN), tropomyosin.
 - *Sliding Filament Theory*
 - (*sarcomere, myofibril, muscle fiber, fascicle, muscle*)
- Force:** Optimum length, motor unit recruitment
- Smooth muscle:** oblique filaments, slow & sustained, gap jxns, endo/paracrine, **CaM** (no TN), Ca^{++} depoln, K^+ repoln
- Cardiac Muscle:** autonomic, striated, intercalated discs/gap jxns, pacemaker potentials (leaky channels)
- Ca^{++} actions:** NT vesicle release, SR release for contraction → troponin or calmodulin. Depolarizes smooth muscle → AP.

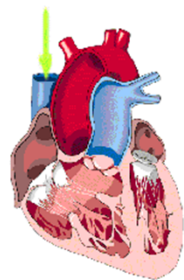
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Chapter 14

Cardiovascular Physiology

<http://www.heartpoint.com/theheart.html>



- Right Atrium
- Tricuspid Valve
- Right Ventricle
- Pulmonic Valve
- Pulmonary Arteries
- Pulmonic Veins
- Left Atrium
- Mitral Valve
- Left Ventricle
- Aortic Valve
- Aorta

1. Blood flow pumping & distribution
2. Anatomy and histology of the heart
3. Mechanism of cardiac contraction
4. Heart beat sequence—how the pump works
5. Regulators of heart beat and volume pumped

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Overview of the Cardiovascular System

- Heart and Blood vessels
- Products transported to sustain all cells

TABLE 14-1 Transport in the Cardiovascular System

SUBSTANCE MOVED	FROM	TO
Materials entering the body		
Oxygen	Lungs	All cells
Nutrients and water	Intestinal tract	All cells
Materials moved from cell to cell		
Wastes	Some cells	Liver for processing
Immune cells, antibodies, clotting proteins	Present in blood continuously	Available for any cell that needs them
Hormones	Endocrine cells	Target cells
Stored nutrients	Liver and adipose tissue	All cells
Materials leaving the body		
Metabolic wastes	All cells	Kidneys
Heat	All cells	Skin
Carbon dioxide	All cells	Lungs

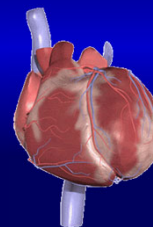


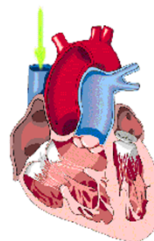
Table 14-1: Transport in the Cardiovascular System

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14.1) Circulation Reviewed

- Heart – "four chambered"

- Right atrium & ventricle
- *Pulmonary circuit*
- Left atrium & ventricle
- Systemic circuit



- Right Atrium
- Tricuspid Valve
- Right Ventricle
- Pulmonic Valve
- Pulmonary Arteries
- Pulmonic Veins
- Left Atrium
- Mitral Valve
- Left Ventricle
- Aortic Valve
- Aorta

- Blood Vessels – "closed circulation"

- Arteries – from heart
- *Capillaries* – cell exchange
- Veins – to heart

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Circulation Reviewed

❖ Circulatory System:
transports oxygen,
nutrients, Heat, & wastes

1. Right side of heart:
delivers deoxygenated blood
from the body to the lungs.

- Pulmonary arteries –
 - ✓ away from heart,
 - ✓ deoxygenated blood

2. Left side of heart:
delivers oxygenated blood from
the lungs to the body.

- Pulmonary veins –
 - ✓ towards heart,
 - ✓ oxygenated blood

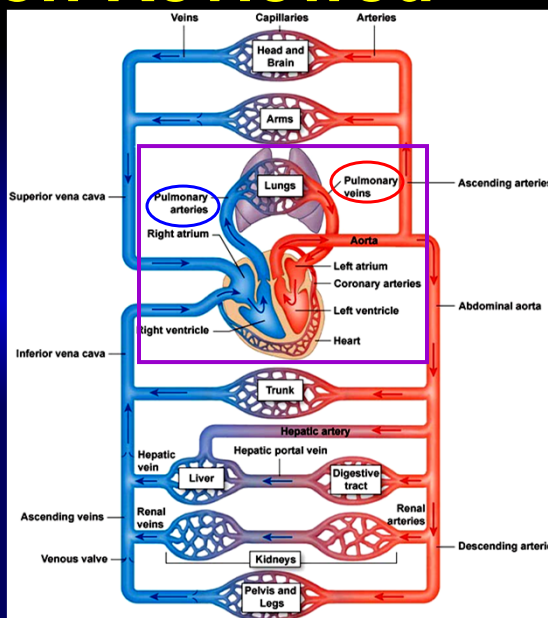


Figure 14-1: Overview of Cardiovascular system anatomy

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14.2) Blood Flow: A. Pressure Changes

1. Flows down a pressure gradient
2. Highest at the heart (driving P), decreases over distance
3. **Hydrostatic** (really **hydraulic**) pressure in vessels
4. Decreases 90% from aorta to vena cava
 - Resistance due to vessel walls and suspended cells

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Blood Flow: Pressure Changes

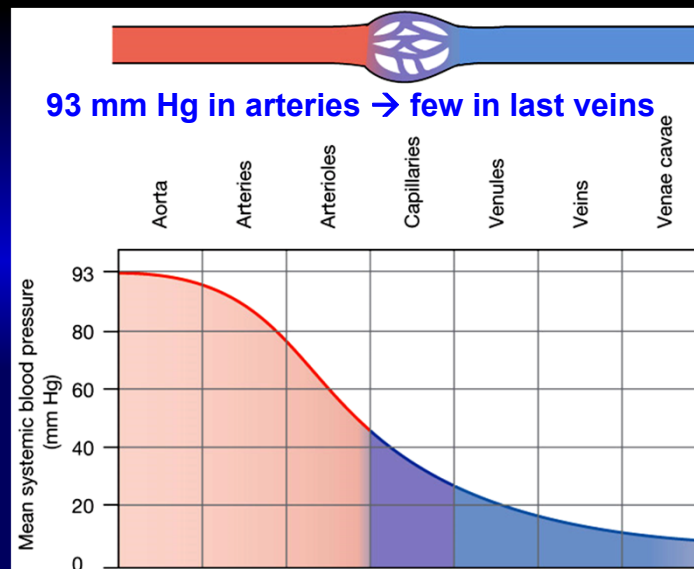


Figure 14-2 : Pressure gradient in the blood vessels

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B. Some Physics of Fluid Movement: Blood Flow

- **Flow rate** = vol/time (L/min)
- **Flow velocity** = rate/C-S area of vessel

- **Resistance** slows flow
 - ↑ Vessel diameter = ↓ R
 - ↑ Blood viscosity = ↑ R
 - ↑ Tube length = ↑ R

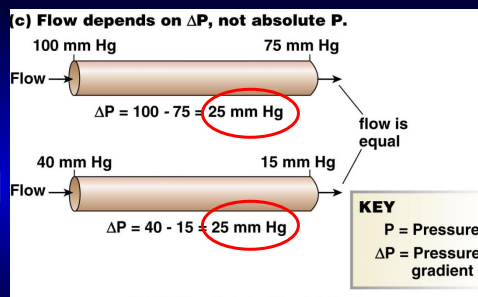
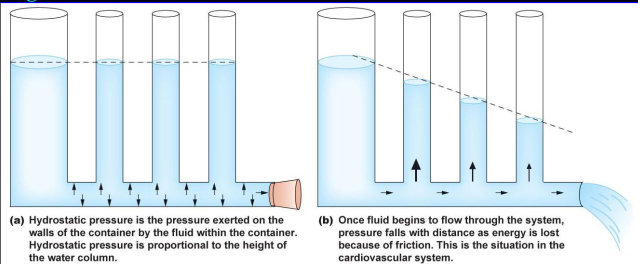


Figure 14-4 c: Pressure differences of static and flowing fluid

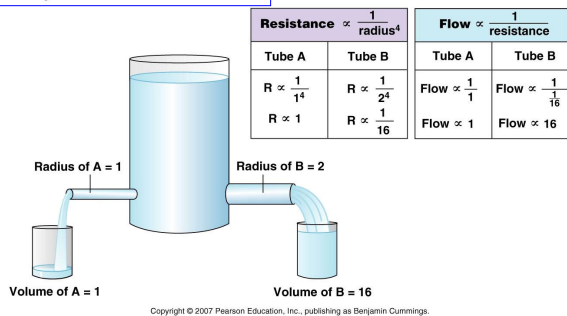
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Pressure differences in static and flowing fluid

Figure 14-3



Resistance and flow



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Some Physics of Fluid Movement: Blood Flow

• Mean arterial pressure \propto cardiac output \times peripheral resistance

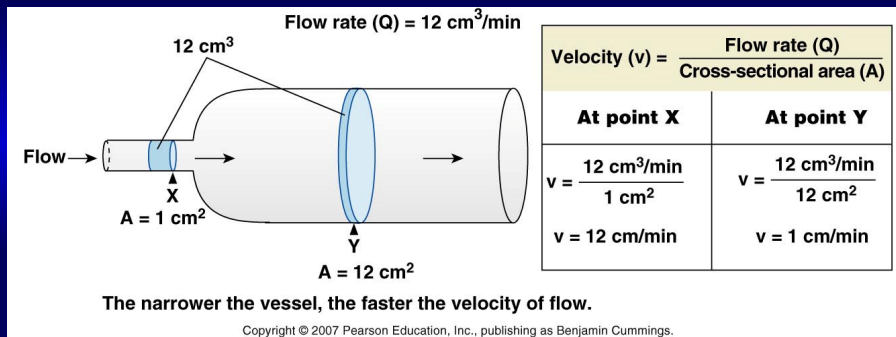


Figure 14-6: Flow rate versus velocity of flow

- ❖ **Velocity:** narrower vessel \rightarrow faster velocity of flow.
- ❖ **Rate:** how much volume flows per min/sec.

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14.3) Heart Structure

1. Pericardium
2. Chambers
3. Coronary vessels
4. Valves- (one-way-flow)
5. Myocardium

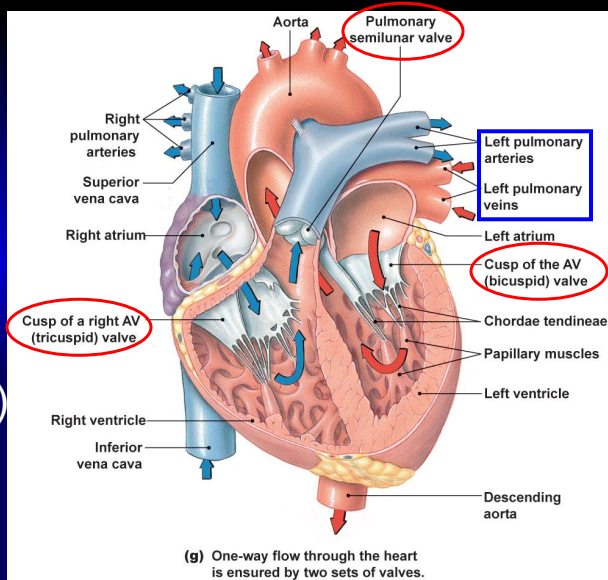


Figure 14-7 g: ANATOMY SUMMARY: The Heart

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Cardiac Muscle Cells:

1. Autorhythmic
2. Myocardial Cells
 - a) Intercalated discs
 - 1) **Desmosomes**
 - Hold together
 - 2) **Gap Junctions**
 - Fast signals
 - Cell to cell
 - b) Many mitochondria
 - c) **Large T tubules**

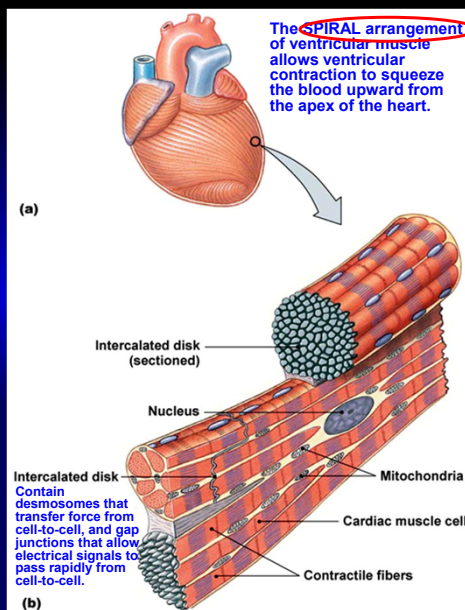
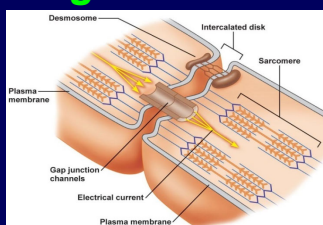


Figure 14-10: Cardiac muscle

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14.4) Mechanism of Cardiac Muscle Excitation, Contraction & Relaxation

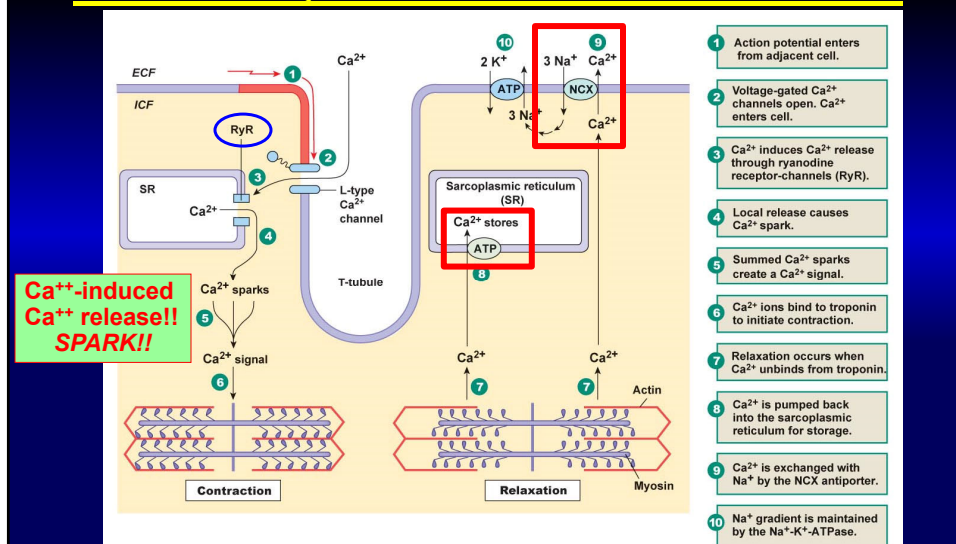


Figure 14-11: Excitation-contraction coupling and relaxation in cardiac muscle

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A. Modulation of Contraction

1. **Graded Contraction**: proportional to # crossbridges formed.
 - **More $[Ca^{++}]$** : \uparrow crossbridges, \uparrow force & speed (\uparrow Troponin- Ca^{++}).
2. \uparrow **Ca^{++} ATPase activity** \rightarrow quicker, more frequent contractions (faster relaxation).
3. Autonomic & epinephrine modulation
 - a) **Epi/norepi** \rightarrow \uparrow speed & force (β_1 -R's).
 - b) **ACh** \rightarrow slows, \downarrow force (mACh-R's).

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Modulation of Contraction

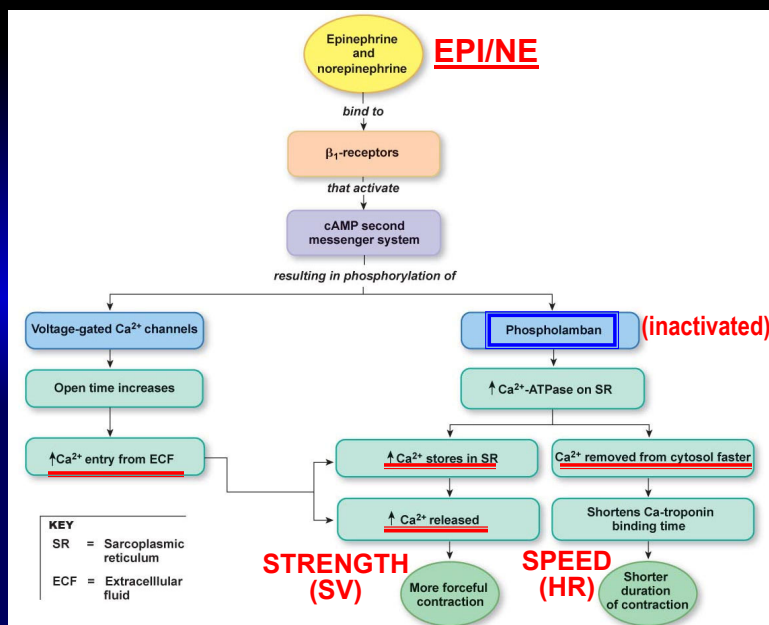


Figure 14-30: Modulation of cardiac contraction by catecholamines

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B. More Characteristics of Cardiac Muscle Contraction

1. Stretch-Length relationship

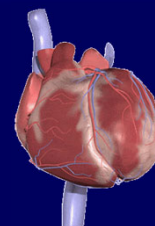
- a) \uparrow stretch, \uparrow Ca^{++} entering
- b) \uparrow contraction force

Including stretching of heart muscle by Diaphragm!!!!

2. Long action potential

3. Long refractory period

- a) *No summation!!*
- b) *No tetanus!!*



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1. Myocardial Length-Tension Relationship

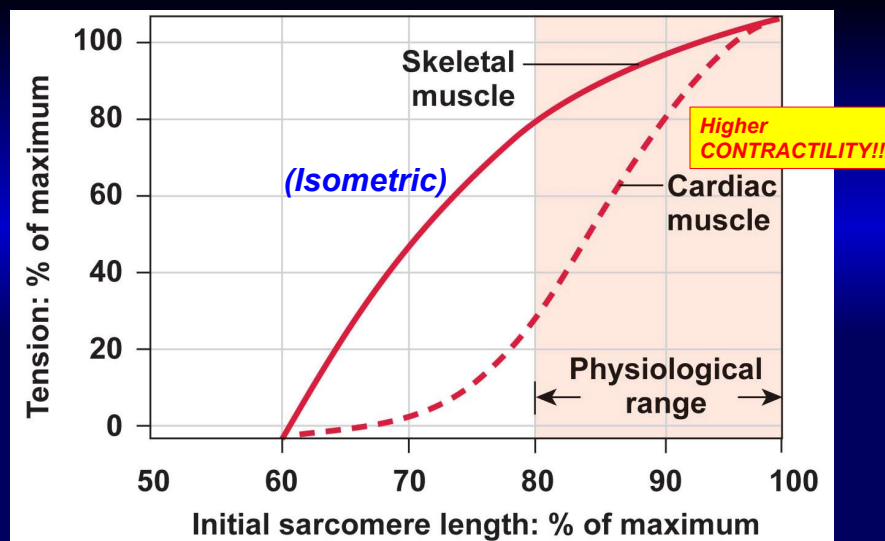


Figure 14-12: Length-tension relationships in skeletal and cardiac muscle

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2. AP in Cardiac Contractile Cell

1. **Phase 4** = resting V_m (-90mV) = E_K !!
2. **0** = Depol'n; influx thru **VG Na^+ channels (double-gated)**
3. **1** = initial repol'n; Na^+ Ch. Close, K^+ leaves (**slow opening**)
4. **2** = Plateau: → long refr.
 - a) *some K^+ Ch. Close;*
 - b) *very slow Ca^{++} Ch. Open!!!*
5. **3** = Rapid Repol'n; Ca^{++} Ch. Close; K^+ influx again

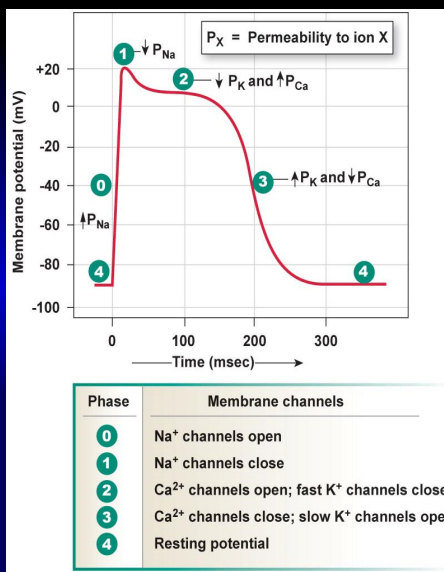


Figure 14-13: AP in cardiac muscle cell

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3. Cardiac Muscle Contraction: Refractory Periods → long!!!

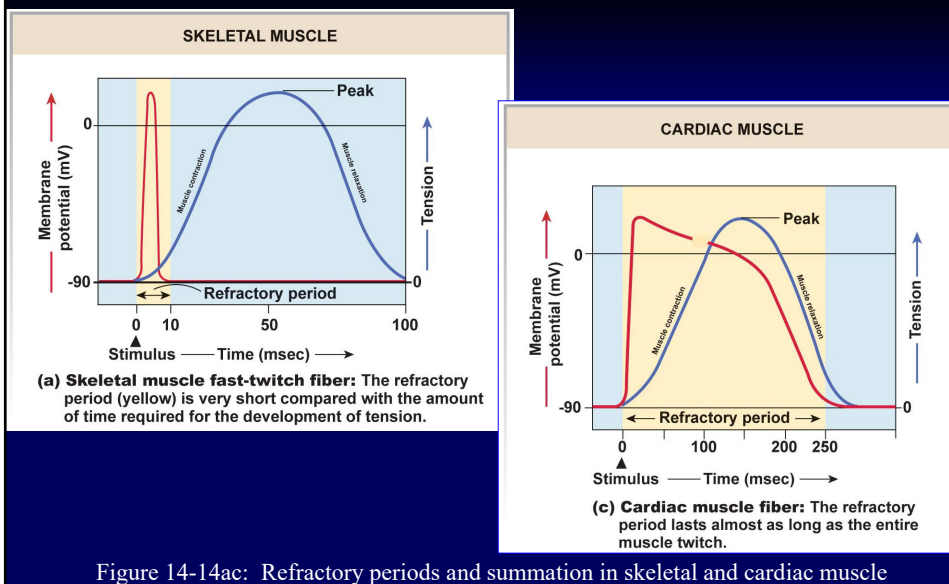


Figure 14-14ac: Refractory periods and summation in skeletal and cardiac muscle

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C. Autorhythmic Cells: Initiation of Signals

1. **Pacemaker membrane potential.**
2. **I_f channels** (“funny current”):
spontaneous Na^+ influx \rightarrow depol’n / pacemaker.
3. **Ca^{++} channels** – influx, to AP.
– Cardiac action potentials!!!
4. **Slow K^+ open** – repolarization.

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Autorhythmic Cells: Initiation of Signals

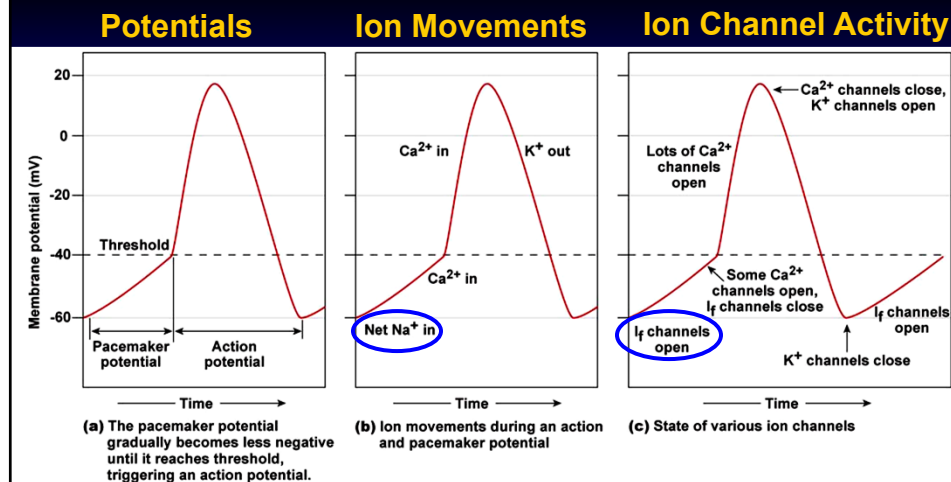


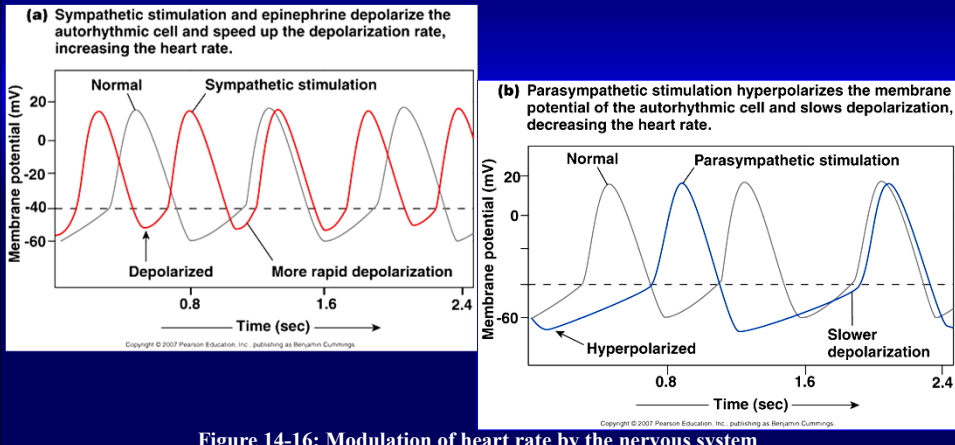
Figure 14-15: Action potentials in cardiac autorhythmic cells

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D. Sympathetic and Parasympathetic

❖ **Sympathetic** – speeds heart rate by \uparrow Ca^{++} & I-f channel flow.

❖ **Parasympathetic** – slows rate by \uparrow K^+ efflux & \downarrow Ca^{++} influx.



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14.5) Coordinating the Pump: Electrical Signal Flow

1. AP from autorhythmic cells in sinoatrial node (SA)
2. Spreads via gap junctions down internodal pathways and across atrial myocardial cells (atrial contraction starts)
3. Pause – atrioventricular (AV) node delay
4. AV node to Bundles of His (A-V Bundle), Branches, & Purkinje fibers (fast-conducting cells)
5. Right and left ventricular contraction from apex \rightarrow upward; due to AV pause and resistance between connective tissues in atria and ventricles

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A. Electrical Signal Flow: Cellular

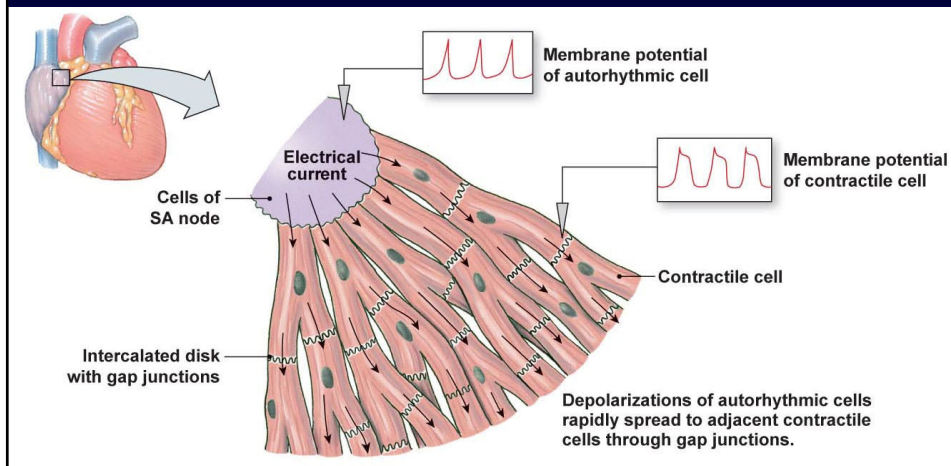


Figure 14-17: Electrical conduction in myocardial cells

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B. Electrical Signal Flow: Heart Muscle

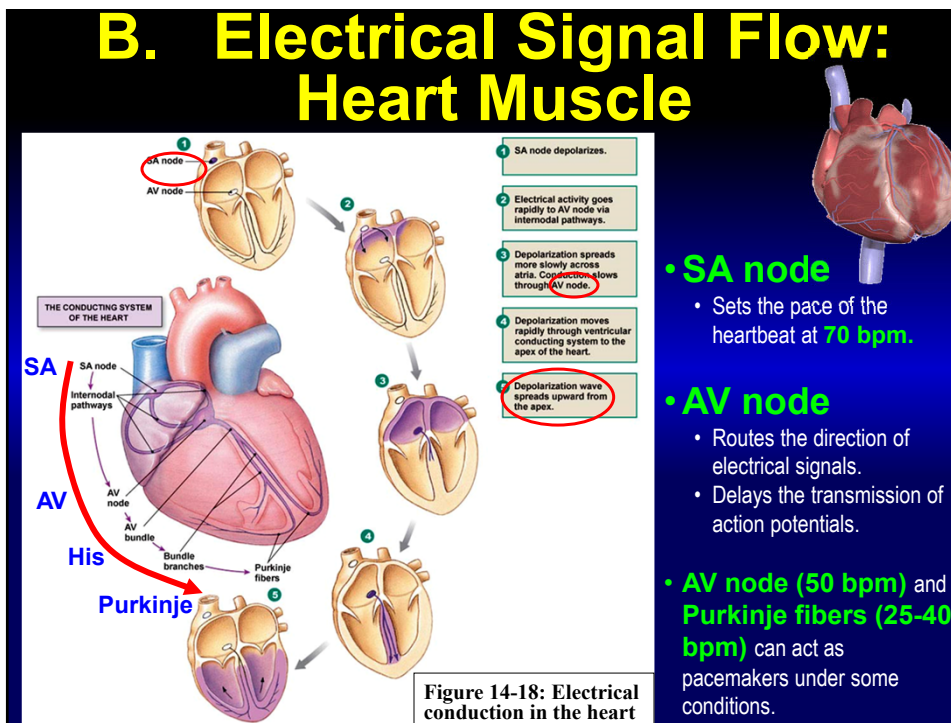


Figure 14-18: Electrical conduction in the heart

• SA node

- Sets the pace of the heartbeat at **70 bpm**.

• AV node

- Routes the direction of electrical signals.
- Delays the transmission of action potentials.

- **AV node (50 bpm)** and **Purkinje fibers (25-40 bpm)** can act as pacemakers under some conditions.

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C. Electrocardiogram (ECG): Electrical Activity of the Heart

- Einthoven's triangle

1. **P-Wave** – atria depolarize
2. **QRS-wave** – ventricles depol./ atr. repol
3. **T-wave** – Ventr. repolarization

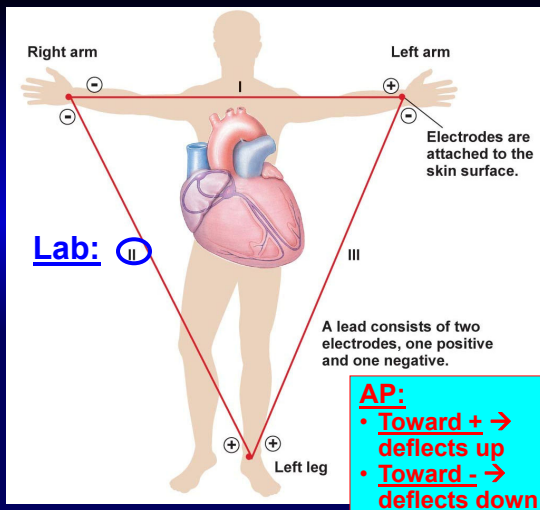


Figure 14-19: Einthoven's triangle

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Electrocardiogram (ECG): Electrical Activity of the Heart

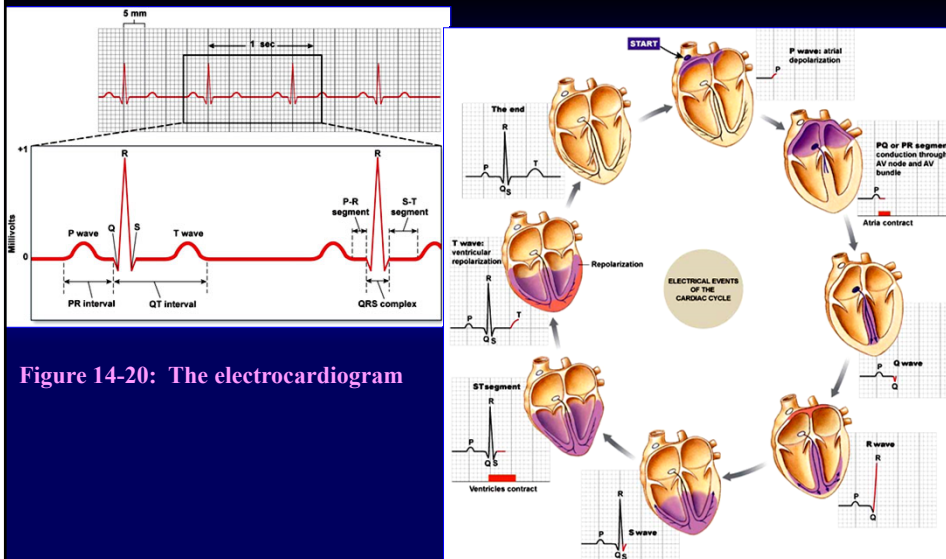


Figure 14-20: The electrocardiogram

Figure 14-21: Cardiac ECG Events

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ECG Information Gained

1. (Non-invasive!!)
2. Heart Rate
 - 60-100bpm?
3. Signal conduction
 - SA block? P-QRS?
 - P-R length?
 - Rhythm? Normal waves?
4. Heart tissue
5. Conditions

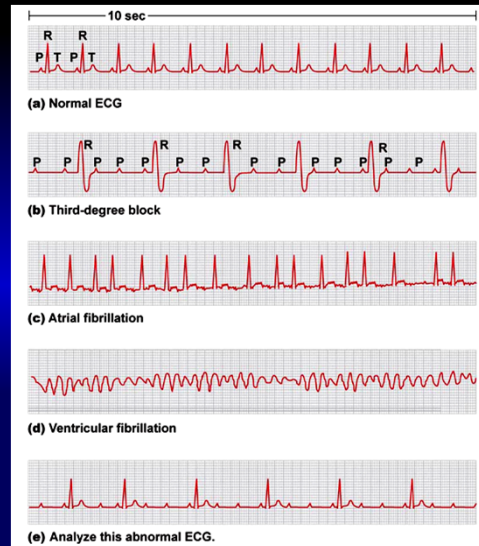


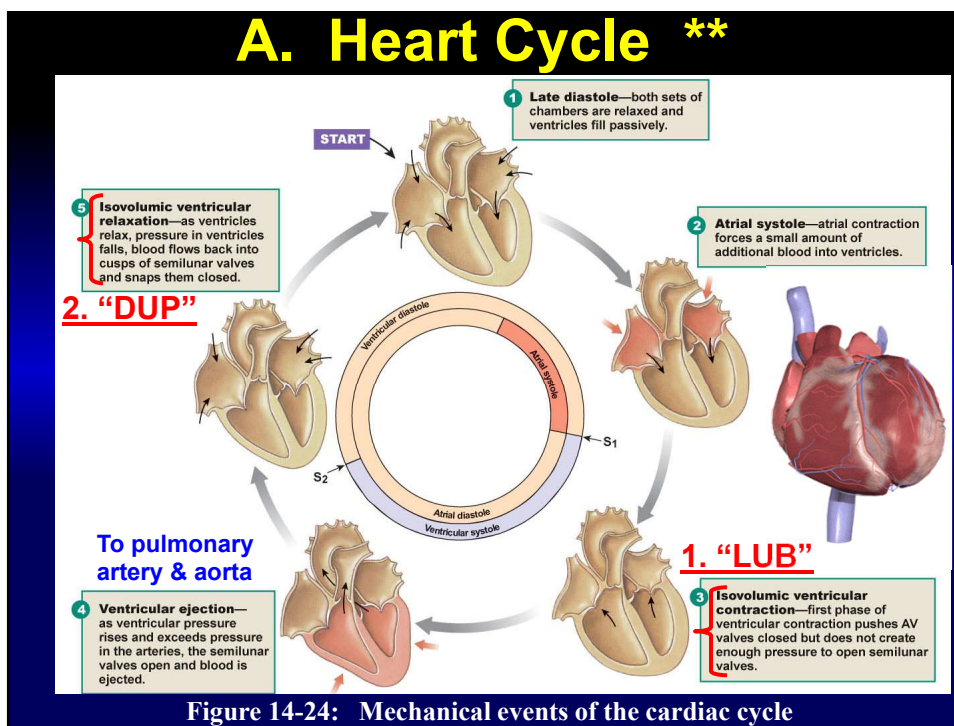
Figure 14-23: Normal and abnormal electrocardiograms – (e.) identify waves, their patterns, and what has happened to electr. Condn.

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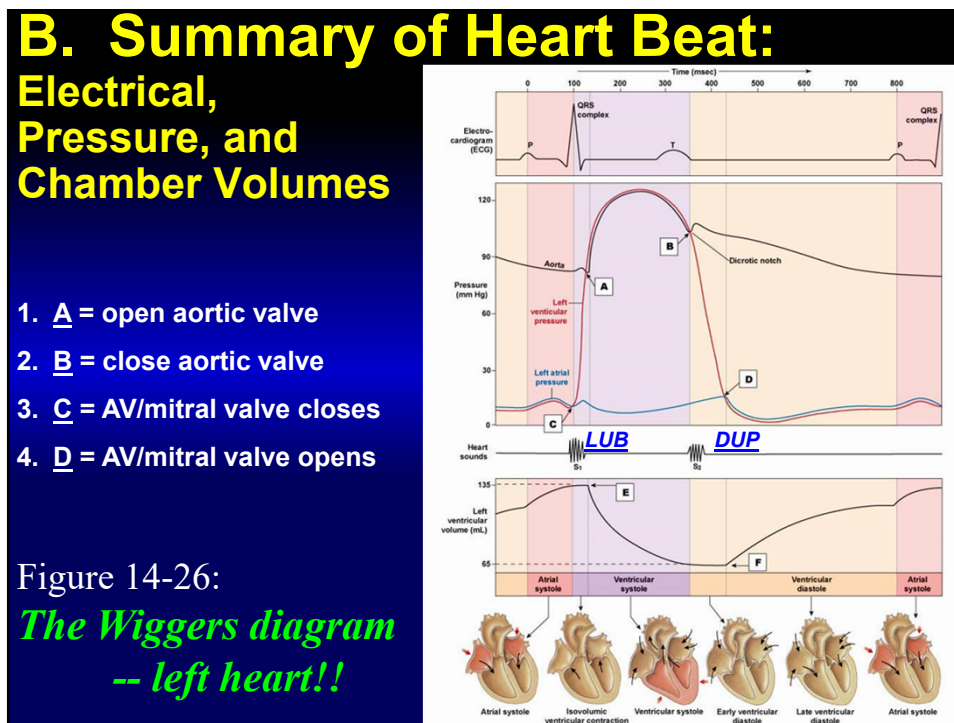
14.6) Heart Cycle: Heart Chambers and the Beat Sequence → Finish and Around To the Start

1. Late diastole: all chambers relax, filling with blood
2. Atrial systole: atria contract, add 20% more blood to ventricles
3. Isovolumic ventricular contraction: closes AV valves (“**LUB**” = AV valves close), builds pressure
4. Ventricular ejection: pushes open semi lunar valves, blood forced out
5. Ventricular relaxation: aortic back flow slams semi lunar valves shut (“**DUP**” = semilunar valves close)
 - Aortic valve and Pulmonary Artery valve (both semilunar)
 - AV valves open; refilling starts – back to start of cycle

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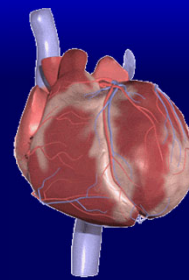
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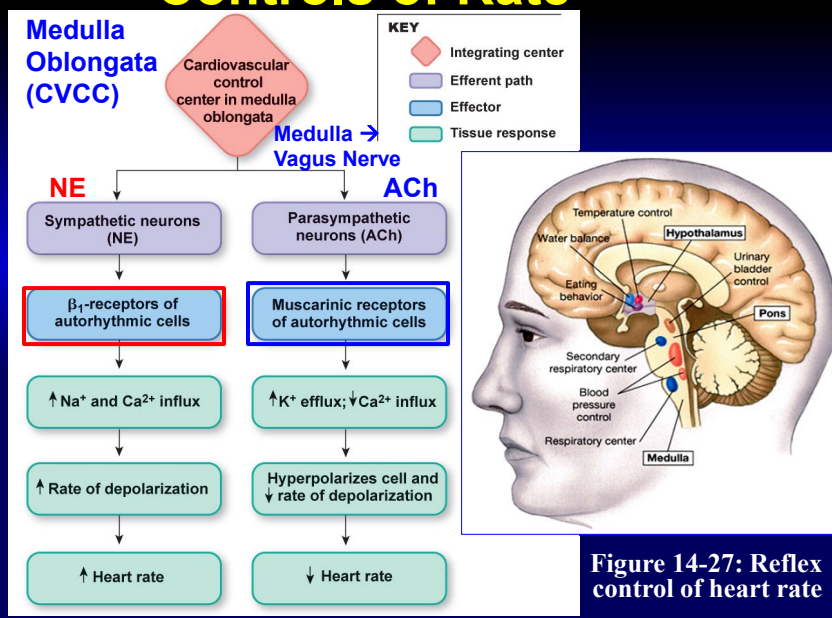
C. Regulators of the Heart: Reflex Controls of Rate

1. Range: about 50 – near 200
2. Typical resting: near 70.
 - SA node fires 90-100 AP's per min.....
3. AP conduction
4. Muscle Contraction
5. Parasympathetic → slows ↓
6. Sympathetic → speeds ↑



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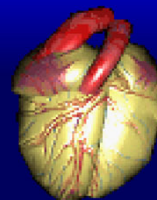
Regulators of the Heart: Reflex Controls of Rate



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14.7) Cardiac Output: Heart Rate x Stroke Volume

1. Around 5L :
(72 beats/min \times 70 ml/beat = 5,040 ml/min)
2. Heart Rate = beats per minute
3. **Stroke Volume** = ml per beat
 - a) = EDV - ESV
 - b) Residual (about 50%, at rest!)



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A. Cardiac Output: Heart Rate x Stroke Volume

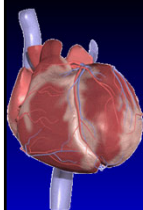
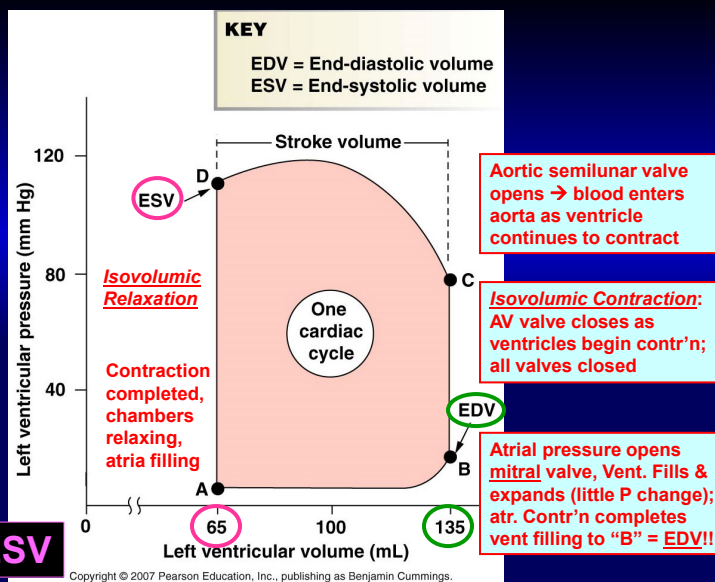


Figure 14-25:
Left Ventr.
P-V changes

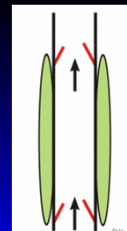


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B. Regulators of the Heart: Factors Influencing Stroke Volume

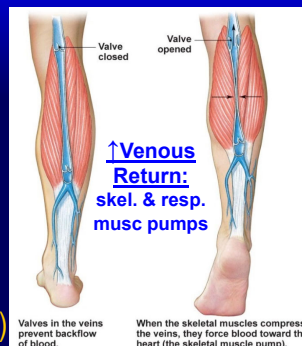
1. **Starling's Law** – stretch \rightarrow \uparrow Ca⁺⁺ entry \rightarrow \uparrow force of contraction (**Frank-Starling Law**)

- \uparrow Stroke volume as \uparrow EDV!!
- \uparrow EDV as \uparrow venous return



2. **Force** of contraction (w/ NE) – affected by:

- Stroke volume
- **Length** of muscle fiber and
- **contractility** of the heart



3. **Venous return:**

- Skeletal pumping**
- Respiratory pumping** (diaphragm)
 - Negative thoracic pressure
 - Positive abdominal pressure
- Venous constriction** (sympathetic innerv.)

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Factors Influencing Stroke Volume

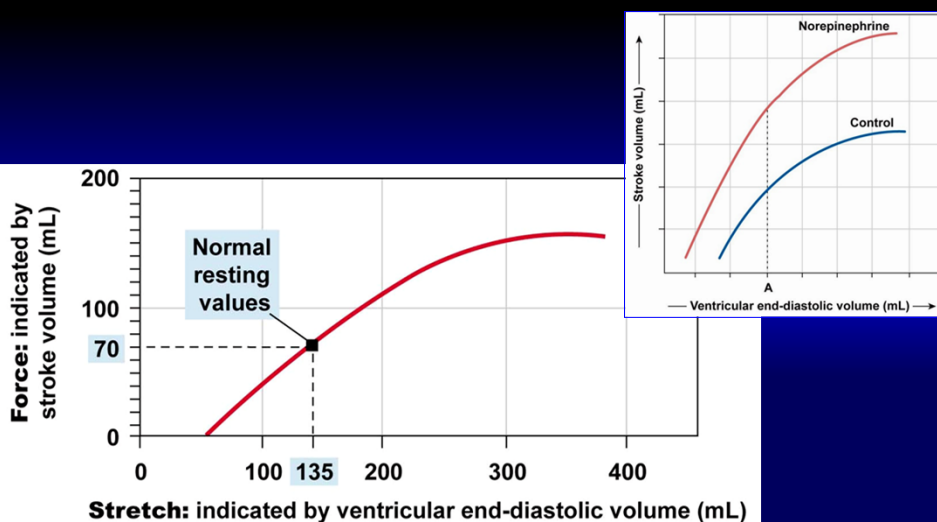


Figure 14-28: Length-force relationships in the intact heart: Starling Curve!

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Factors Influencing Stroke Volume

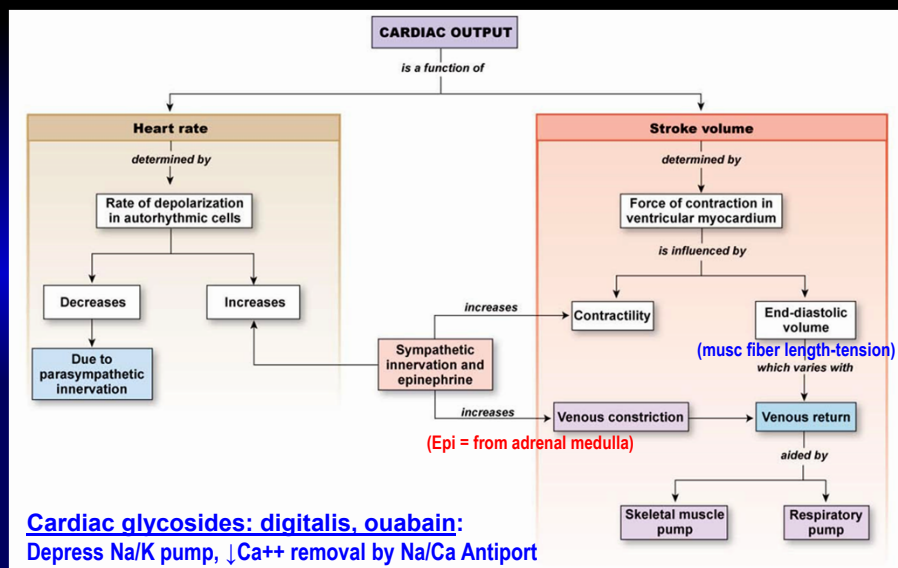
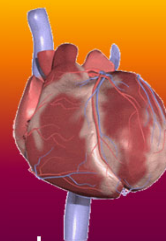


Figure 14-31: Factors that affect cardiac output

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Ch. 14: Summary

1. Anatomy and physiology of the **Heart**: chambers, muscles, valves, and its pacemaker
2. Mechanism of **cardiac muscle** stimulation and contraction – I-f channels, Ca⁺⁺ ind'd Ca⁺⁺ release
3. Blood vessels and fluid flow down a **pressure gradient** – ventricles & aorta, radius
4. Electrical control of the beat sequence (pacemakers/ autorhythmic cells), and **ECG** information – SA, AV, Purkinje
5. Influence of beat rate and stroke volume by ANS & hormones – epi → β1 Adr., mACh,



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