Ch 7 & 8: Blood and Cardiac Physiology

PowerPoint updated 1/5/25

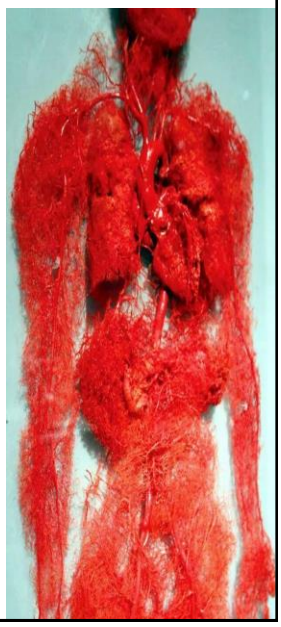
Objectives

Ch 8:

1. Review: Heart Anatomy, and Systemic & Pulmonary circuits.
2. The Cardiac Cycle and Heart Sounds
3. The Heart's Conduction Cycle & the ECG
4. Regulation of Heart's Pacemaker (heart rate)
5. Blood Pressure
6. Cardiac output and its Regulation
7. Three Ways the Body Regulates Blood Pressure
8. Abnormal Blood Pressure
9. Cardiovascular terms you need to know

Ch 7:

10. Blood Physiology



1

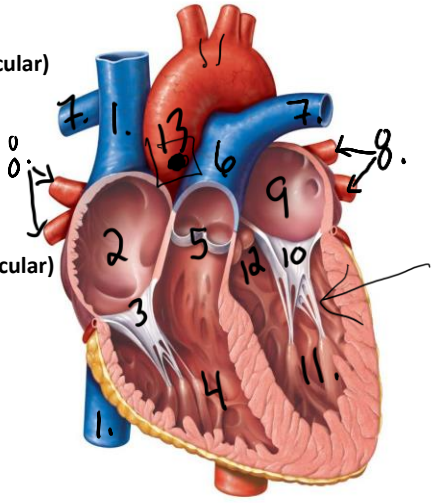
1. Review of Heart Anatomy and Circulatory System

Page 138 in text

ANATOMY REVIEW!

Other vessels attached to heart:
Brachiocephalic a.
L common carotid a.
L subclavian a.

1. Superior & inferior vena cava
2. Right atrium
3. tricuspid valve (R atrioventricular)
4. Right ventricle
5. pulmonary semilunar valve
6. Pulmonary trunk
7. Pulmonary arteries (O2-poor) to lungs
8. Pulmonary veins (O2-rich)
9. Left atrium
10. bicuspid valve (L atrioventricular)
11. Left ventricle
12. aortic semilunar valve
13. Aorta
    - > ascending
    - > arch
    - > descending



2

2

Circulatory Systems REVIEW!

Page 139 & 142 in text

Systemic Circuit

> From left atrium, arteries, tissues, veins, to vena cava.

> Systemic Arteries = always travel away from heart, carry O2-rich blood

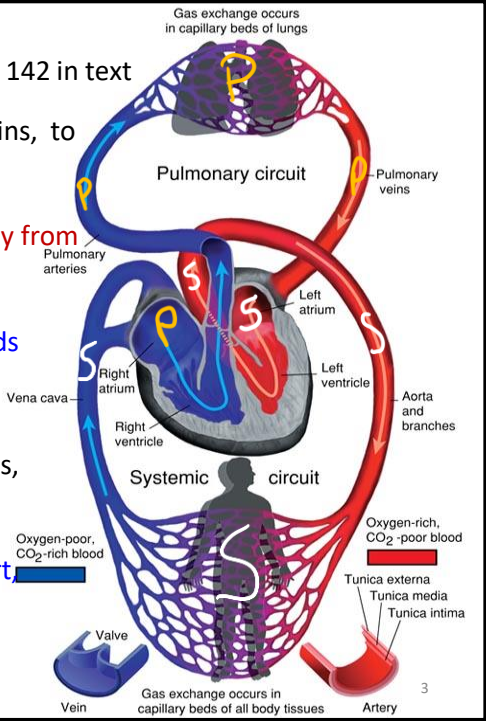
> Systemic Veins = always travel towards heart, carry O2-poor blood.

Pulmonary Circuit

> From right atrium, pulmonary arteries, lungs, and pulmonary veins.

> Pulmonary arteries = away from heart, towards lungs w/O2-poor blood.

> Pulmonary veins = towards heart, w/O2-rich blood.



3

3

2: The Cardiac Cycle & Heart Sounds Page 144- 146 in text

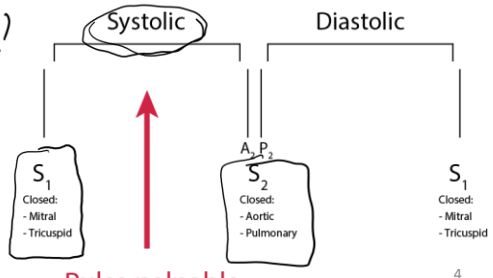
S1 or Lub sound = simultaneous closure of tricuspid & bicuspid valves.

> Ventricular systole = ventricles contract simultaneously.

S2 or Dub
~~S1 or Lub~~ **sound =** simultaneous closure of semilunar valves.

> Ventricular diastole = ventricles relaxed & fill w/blood from atria.

Asystole = no heart sounds!



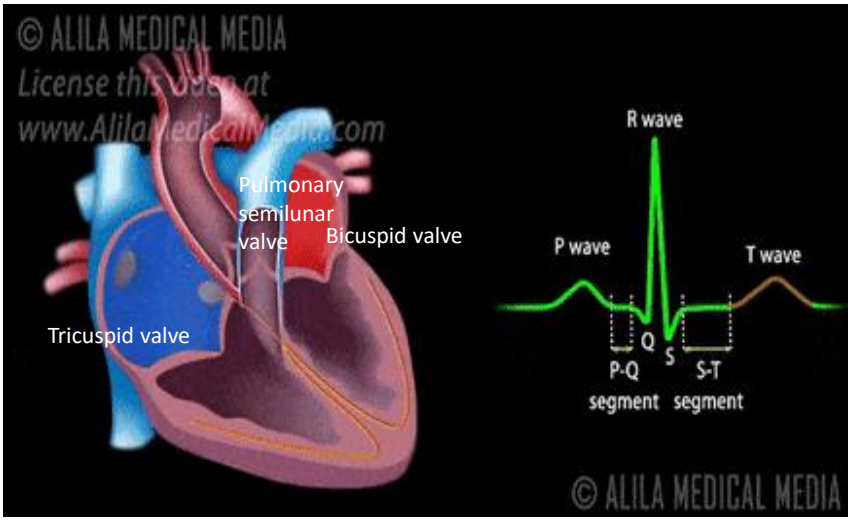
Click **HERE** for Normal heart sounds

4

4

2: The Cardiac Cycle & Heart Sounds Page 144- 146 in text

GIF

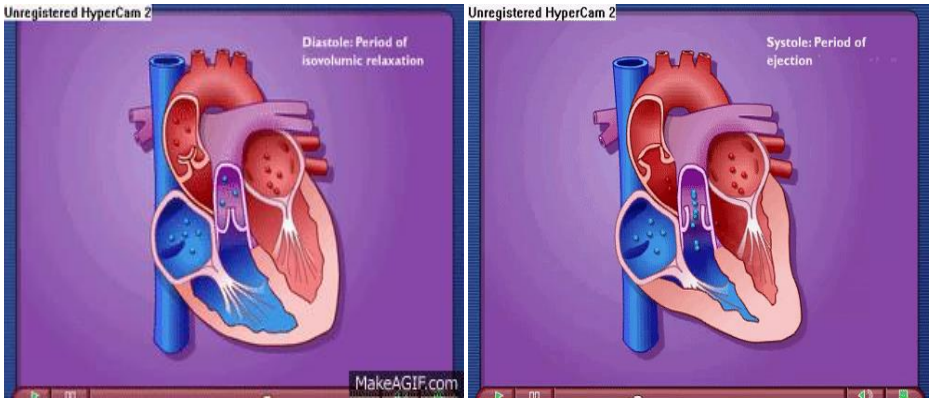


5

5

Ventricular Diastole

Ventricular Systole



GIF

6

6

Click **HERE** for Normal heart sounds

Heart Murmurs: unusual sound(s) during heart during cycle

> Innocent (benign) murmur = quiet whoosh sound in between the lub (S_1) and dub (S_2). Can be normal in children, who typically outgrow it.

> Pathologic murmur = loud whoosh sound between S_1 & S_2 Abnormal if murmur develops in an adult.

Murmurs typically caused by valve disease:

E.g.

- **valve prolapse** = Valve (often bicuspid or mitral) bulges into ventricle

- **stenosis** = stiffening valve(s) in heart.

- **rheumatic heart disease** = autoimmune attack on valves (bicuspid)

Chronic inflammation, leads to scar tissue & stiffening

7

7

Click **HERE** for Normal heart sounds

Heart Murmurs: unusual sound(s) during heart during cycle

Click links below to hear normal & abnormal heart sounds. (Works best with ear buds)

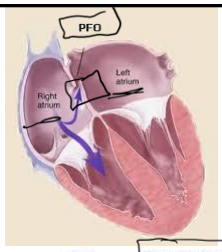
Click **HERE** for innocent (benign) murmur sound
Click **HERE** for aortic stenosis sound
Click **HERE** for split S2 (split dub) sound
Click **HERE** for bicuspid (mitral) valve regurgitant flow sound.

These, and more, heart sounds can be found **HERE**

8

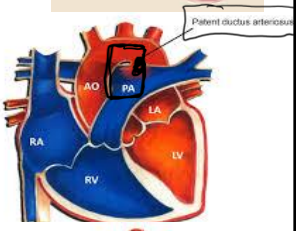
8

Heart defects: Clinical App

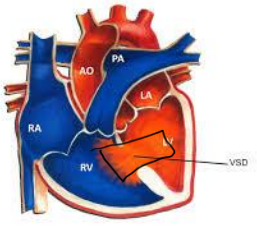


Septal Defects:

1. Patent foramen ovale = fetal hole in between (R) and (L) atria.



2. Patent ductus ateriosus = fetal hole in between pulmonary trunk & Aorta.



3. Ventricular septal defect = fetal hole in interventricular septum.

9

9

Review

- **The cardiovascular system (pulmonary & systemic circuits)**
- **Cardiac cycle & heart sounds**
  - ventricular diastole Vs systole
  - Lub / Dub or S1 / S2 sounds (normal sounds & innocent murmur)
  - Abnormal heart sounds (pathological murmurs)
- **Heart defects**
  - Patent foramen ovale
  - Patent ductus arteriosus
  - Ventricular septal defect

10

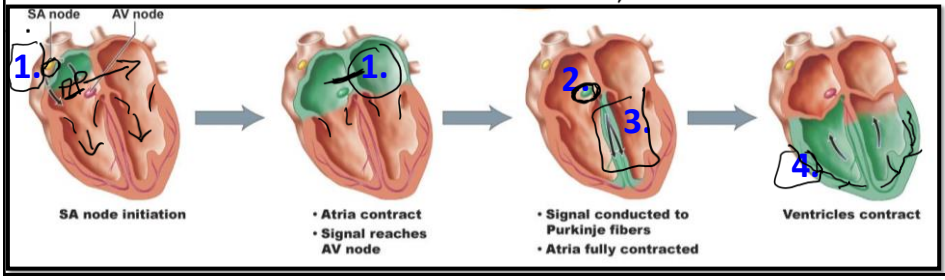
10

3: The Heart's Conduction System & ECGs

Heart is "autorhythmic" = starts its own signal for contraction.

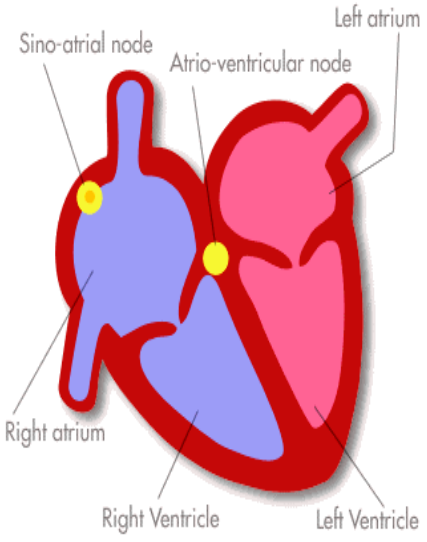
1. **SA node** = (sino atrial) "pacemaker" where AP starts. AP jumps to (A) atria & atria contract.
2. **AV node** = (atrio ventricular) where the AP pauses briefly to allow ventricular filling.
3. **Bundle of HIS** = AP is in the interventricular septum.
4. **Purkinje fibers** = AP is in outer walls of ventricles & they contract.

Page 146- 148 in text

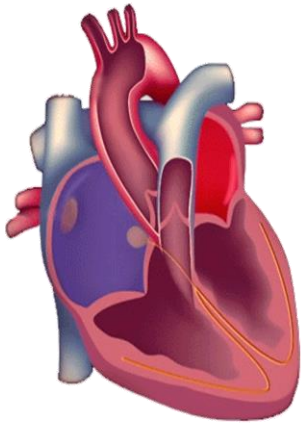


11

Heart Conduction System



GIF



GIF

12

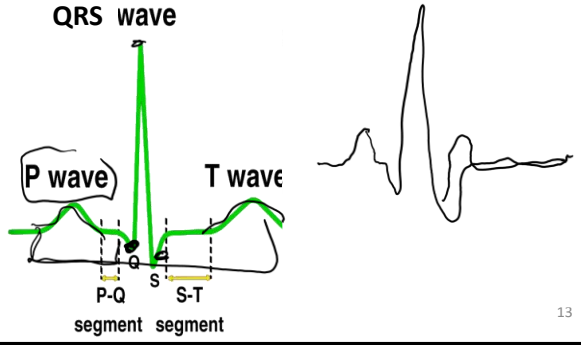
12

Electrocardiogram (ECG or EKG) =

P-wave = atrial contraction.

QRS wave = ventricles contract

T-wave = ventricles relaxed & filling with blood.



Page 148- 149 in text

13

13

4: The Heart's Pacemaker & Its Regulation

Regulated by cardiac center in medulla oblongata!

Sympathetic innervation with thoracic & cardiac nerves:

- **Neurotransmitter =** Epinephrine.
- **Receptor =** β_1 adrenergic
- **Heart rate** ↑

Parasympathetic innervation with vagus nerves:

- **Neurotransmitter =** Ach
- **Receptor =** muscarinic cholinergic
- **Heart rate** ↓

14

14

4: The Heart's Pacemaker & Its Regulation

Spontaneous APs start in pacemaker myocardial cells

1. Heart pacemaker cell depolarization (stimulation):
> opening of Na^+ & Ca^{+2} channels
> causes AP (or EPSP)
>Myocardial cells contract! (Signal started!)

Epinephrine binding to β1 adrenergic receptors on pacemaker increase rate of depolarization

2. Heart pacemaker cell repolarization (rest):
> opening of K^+ channels
> Myocardial cells relax!

ACh binding to muscarinic cholinergic receptors on pacemaker decrease rate of depolarization

APs started in SA node pacemaker muscle cells travel through rest of conductions system (i.e. AV node, Bundle of HIS, & purkinje fibers).

15

15

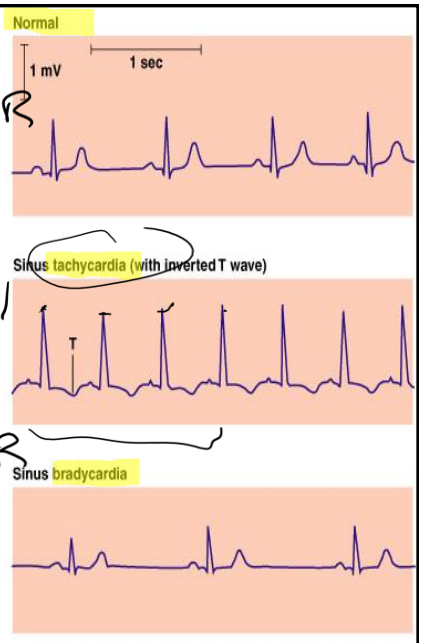
Arrhythmias = abnormal heart rate
Clinical App

Tachycardia = higher than normal HR
Treatments: for tachycardia also treat high BP
> Na+ channel blockers (*~~quinidine, lidocaine~~*)
> Ca^{+2} channel blockers (*~~verapamil~~*)

> Beta blockers B_1 & B_2
ex. *General beta blocker =* Propranolol
Review *B1-specific blocker =* Atenolol.

Bradycardia = lower than normal HR
Treatments: for bradycardia also treat low BP
> Digitalis – increases Ca+2 available to increase contractile strength.

> B1-agonist = Dobutamine
> MAO-I A = → makes more epinephrine available



16

16

Hypertension Drugs

Can you think of any drugs (especially ones we've covered) that can lower BP?

Propranol
Atenolol
Nat & Ca+2 blockers

17

17

Beta blockers – have specific indications, contraindications, and side effects that must be considered.

You DO NOT need to memorize the extra beta blockers shown here.
This just gives you a preview of nursing pharmacology. ☺

First generation beta blocker drugs were non-selective - Sotalol and **propranolol.** Propranolol is the first of its class used in clinical practice. Can block both B1 and B2 adrenergic receptors.

Second generation beta blocker drugs were selective (specific to the B1 receptor on heart muscle) – **atenolol**, metoprolol, and labetalol.

not memorize
Third generation drugs – can be non-selective or selective, BUT they also cause arterial vasodilation by stimulating NO and cGMP.

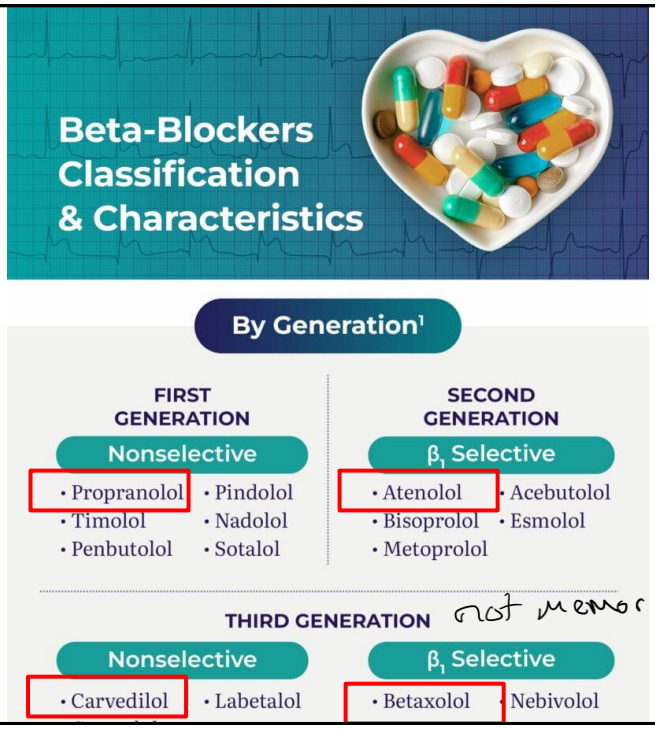
> Non-selective for B1 receptor (blocks B2) – Carvedilol, Carteolol, and Labetalol.

> Selective for B1 receptor – **Betaxolol** and Nebivolol.

Click **HERE** for a good summary of beta blockers for hypertension

18

18



19

Review

- **Cardiac conduction system**
  - SA node, AV node, Bundle of HIS, Purkinje fibers
- **Monitoring electrical activity of the heart (EKG)**
  - P wave, QRS wave, T wave
- **The Heart's Pacemaker**
  - parasympathetic and sympathetic regulation
- **Arrhythmias (tachycardia & bradycardia)**
  - Drugs to treat arrhythmias

20

20

5: Blood Pressure

Blood Pressure = pressure of arterial blood against vessel wall.

Systolic BP = pressure resulting from ventricular contraction.
- always the higher number.

Systolic arterial BP normal range = 80 – 160 mmHg

Diastolic BP = pressure with ventricular relaxation.
- always the lower number

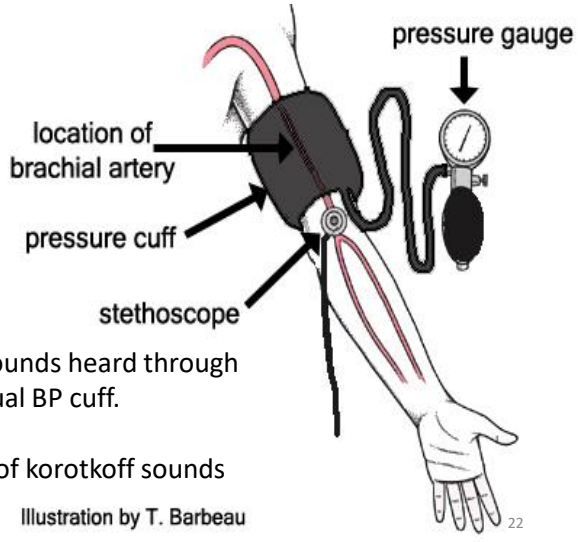
S 120 mmHg
D 80

Page 149 – 150 in text

21

21

Measuring Blood Pressure (Systolic & Diastolic) using a sphygmomanometer



"korotkoff sounds" = blood sounds heard through stethoscope when using manual BP cuff.

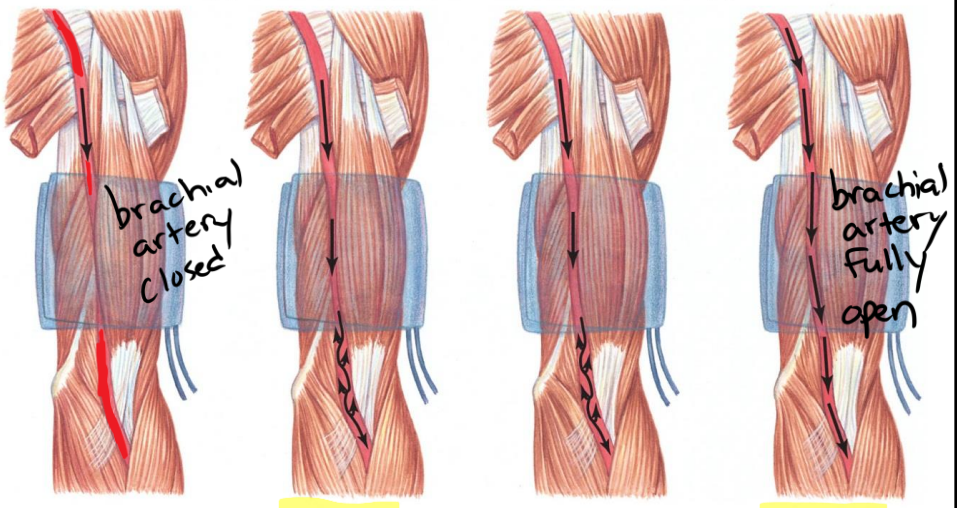
Click **HERE** for YouTube video of korotkoff sounds

Illustration by T. Barbeau

22

22

Measuring Blood Pressure (Systolic & Diastolic)



No sounds	First Korotkoff sounds	Sounds at every systole	Last Korotkoff sounds sound gone
Cuff pressure = 140	Cuff pressure = 120	Cuff pressure = 100	Cuff pressure = 80
	Systolic pressure = 120 mmHg		**Diastolic pressure = 80 mmHg**

Blood pressure = 120/80

23

23

6: Cardiac Output

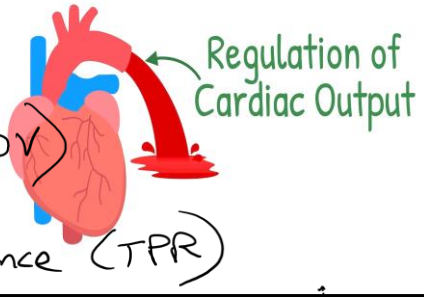
Cardiac Output = ml of blood leaving heart in 1 min.

Cardiac Output = Stroke Volume X Heart Rate
(ml/min) (ml/beat) (bpm)
blood

Average HR varies (~60 – 80 bpm)
AVG stroke volume = 70 – 80 ml/beat
AVG cardiac output = 5500 ml/min (5.5L/min)

Cardiac Output influenced by:

1. HR
2. Stroke volume
3. End diastolic volume (EDV)
4. Contractility of heart
5. Total Peripheral resistance (TPR)



24

Things That Influence Cardiac Output:

1. Heart Rate – changes with sympathetic or parasympathetic stimulation by medulla's cardiac center.

As HR ↑, cardiac output ↑
As HR ↓, cardiac output ↓

What are some drugs we've covered that can ↑ HR, thus will ↑ cardiac output? B_1 agonist Dobutamine
MAO-I A
Digitalis

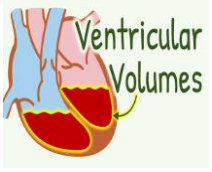
What are some drugs we've covered that can ↓HR, thus will ↓ cardiac output? B_1 B_2 blocker Propranolol
B_1 blocker Atenolol.

25

25

Things That Influence Cardiac Output:

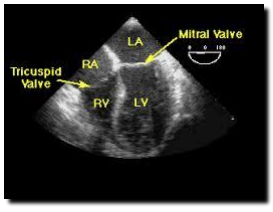
2. End Diastolic Volume (EDV) – = volume of blood in ventricles at end of diastole.

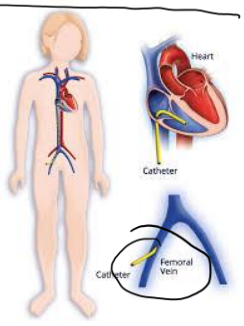


As EDV ↑, cardiac output ↑
As EDV ↓, cardiac output ↓

EDV is used to evaluate heart function in patients.

Can measure EDV using echocardiogram, or use catheterization of left ventricle.





Click **HERE** to read more about **EDV**, and how it is used to calculate **stroke volume (SV),** and **ejection fraction.**

26

26

Things That Influence Cardiac Output:

3. Stroke Volume: volume of blood leaving the heart with each cardiac cycle (Lub Dub)

As stroke volume ↑, cardiac output ↑
As stroke volume ↓, cardiac output ↓

DO NOT NEED TO MEMORIZE info below! This just demonstrates how these things are used in clinical setting to evaluate patient's heart function.

Doctors use both **end-diastolic volume (EDV)** and **end-systolic volume (ESV)** to calculate **stroke volume**. End-systolic volume is the amount of blood remaining in the ventricle at the end of systole, after the heart has contracted

Stroke volume = end-diastolic volume – end systolic volume.

Ejection fraction = percentage of blood that the heart pumps out of the left ventricle during each beat. Used to evaluate how well a person's heart is functioning.

Ejection fraction = (stroke volume / end-diastolic volume) x 100.

27

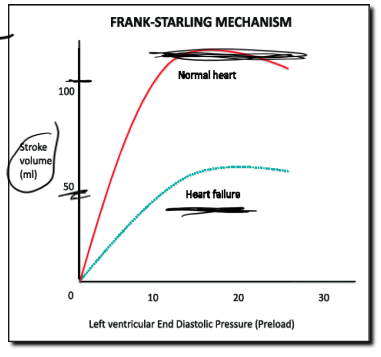
27

Things That Influence Cardiac Output:

4. Heart contractility (influenced by how much ventricles **stretched**)

when cardiac muscle is stretched it contracts harder.

Contractility = measure of how strong heart muscles contract



Frank-Starling Law of the Heart

So….
As EDV ↑, ventricles stretch more, contractility ↑, CO ↑. (more blood filling; cardiac output)

As EDV ↓, ventricles stretch less, Contractility ↓, CO ↓.

28

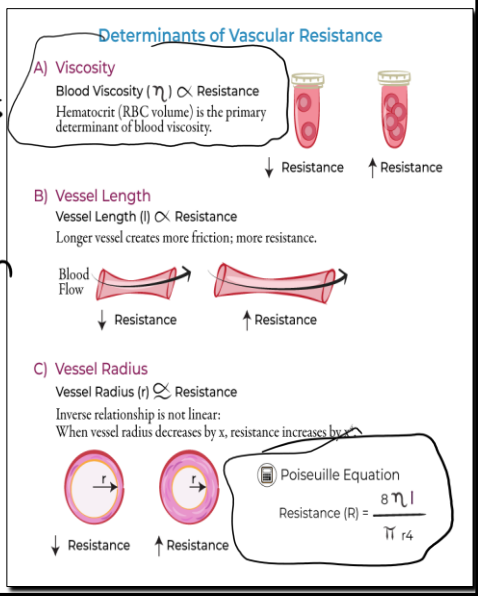
28

Things That Influence Cardiac Output:

5) Total peripheral resistance (TPR) =

thickness (viscosity) of blood influences how hard heart has to work to pump blood through vessels.

AS TPR ↑, C.O. ↓
TPR ↓, C.O. ↑



29

Things That Influence Cardiac Output:

In summary:

As HR ↑, cardiac output ↑
As HR ↓, cardiac output ↓

As stroke volume ↑, cardiac output ↑
As stroke volume ↓, cardiac output ↓

As EDV ↑, cardiac output ↑
As EDV ↓, cardiac output ↓

↑ ventricular stretch, ↑ heart contractility, so cardiac output ↑
↓ ventricular stretch, ↓ heart contractility, so cardiac output ↓

And

As TPR ↑, stroke volume and cardiac output ↓
As TRP ↓, stroke volume and cardiac output ↑

To re-cap:
Click HERE for a really good YouTube video explaining effect of HR, stroke volume, EDV, ventricular stretch, and TPR on cardiac output.

30

30

Review

- **Blood pressure**
- **Factors that influence cardiac output:**
  there are MANY factors, but we covered:
  Heart rate
  EDV
  Stroke volume
  Heart contractility (Frank Starling's "stretching" Law)
  TPR

31

31

7: Three Ways the Body Regulates Blood Pressure

Blood pressure is directly influenced by blood volume:

The kidneys have the most important control of **blood volume**, by how much water they retain (keep in bloodstream) versus how much they excrete as urine.

Kidneys affected by sympathetic and parasympathetic control, AND by hormones.

32

32

7: Three Ways the Body Regulates Blood Pressure

The Quick Fix to BP **vs** **The Slow Fix to BP**

for < 1 min of effect

Artery baroreceptors & medulla oblongata (cardiac and vasomotor centers)

Fixes BP quickly, but does not last long

Hormones that affect kidney water reabsorption.

Takes more time to fix BP, but its effect lasts longer (hours or days)

33

33

7: Three Ways the Body Regulates Blood Pressure

1. Baroreceptor reflexes

"baroreceptors" = pressure/stretch receptors that detect stretching of arteries OR the heart chambers.

2. Hypothalamus

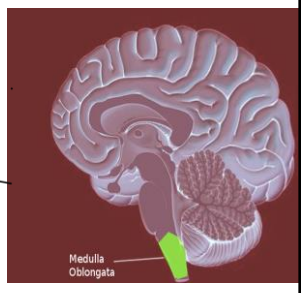
3. Kidney regulation

34

34

1. Arterial baroreceptor reflex (the "Quick Fix")

Stimulus = change in arterial blood pressure outside 80 – 160 mmHg
Sensor = aortic arch & carotid artery baroreceptors
Integrating center = medulla cardiac & vasomotor centers



A) If stimulus of ↓ BP (below 80mmHg)
Autonomic response is sympathetic
- Neurotransmitter = Epinephrine
- Pacemaker muscle receptors = β_1 adrenergic, HR ↑
- arterial vasoconstriction
- BP ↑

B) If stimulus of ↑ BP (above 160 mmHg)
Autonomic response is parasympathetic
- neurotransmitter = Ach
- pacemaker muscle receptors = muscarinic cholinergic, HR↓
- arterial vasodilation
- BP ↓

35

35

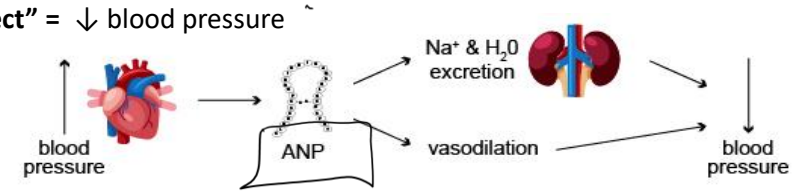
2. Heart baroreceptors reflex (hormonal response & "Long Fix")

Stimulus = ↑ blood pressure (heart chamber stretched)
Sensor, integrating center, and effector = heart

Heart secretes ANP (atrial natriuretic peptide)

ANP functions to
Cause arterial vasodilation
↑ filtration rate at kidneys, which
↓salt & water reabsorption & ↑ urine output (pee more) losing body water
↓ blood volume

"effect" = ↓ blood pressure



36

36

3. Hypothalamus – Blood Osmolarity Center setpoint of 280 – 290 mOsm

stimulus = ↑ blood osmolarity blood salt content.
(above 290 mOsm) Antidiuretic hormone

sensor & integrating center, & effector
= hypothalamus, which releases **ADH**. Preoptic nucleus

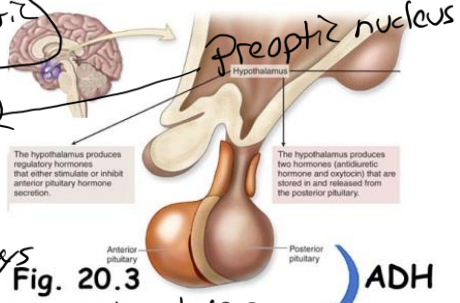
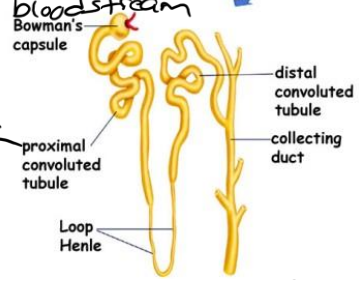


Fig. 20.3

effect of ADH =
↑ water retention by kidneys
urine output ↓, water returns to bloodstream
↑ blood volume, ↑ BP BUT
↓ blood osmolarity (water dilutes the salts)

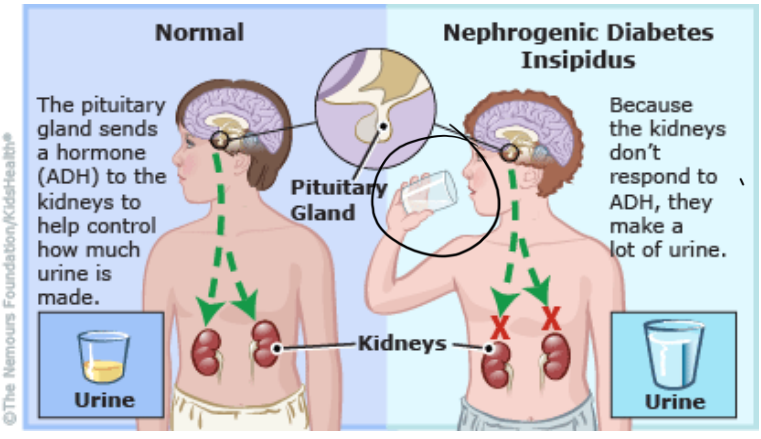


Stimulus = ↓ blood osmolarity
hypothalamus inhibits ADH release
opposite things happen

37

Diabetes insipidus = insufficient ADH release by hypothalamus.
you are always sipping water because you pee out body water are dehydrated

↓ water reabsorption at kidneys
↑ urine output (pee more)
↓ blood volume and BP
- become chronically dehydrated



38

38

4. Kidney –renin-angiotensin-aldosterone system ("long fix")

Stimulus = low BP at renal artery of kidneys

Sensor, integration center, & effector = kidney juxtaglomerular apparatus (JGA)

"effect" = ultimately, an increase in BP

39

39

IF blood volume & BP ↓:

> Sensed by JGA

> JGA releases **Renin**

> Renin causes liver to convert **angiotensinogen** → **angiotensin 1**

> Angiotensin 1 → **angiotensin 2** by **ACE** in lungs

> Angiotensin 2 stimulates Adrenal cortex make **aldosterone**

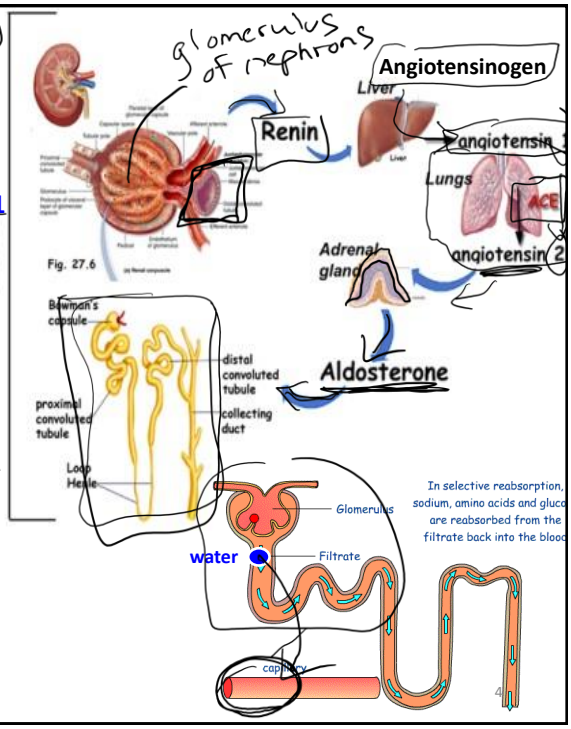
> **Al**do**st**erone ↑ salt reabsorption

> ↑ Water reabsorbed w/salt

> ↑ blood volume, **↑ BP**

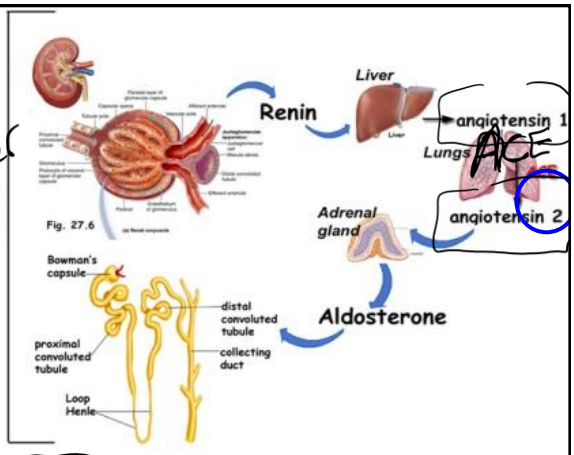
IF blood volume & BP ↑:

> Renin release is inhibited



40

won't retain salt.
pee out salt.
pee out body water
Blood volume ↓
BP ↓



ACE = angiotensin converting enzyme

ACE inhibitors – Clinical App
↳ block lungs from converting Angiotensin 1 to angiotensin 2.
↓ Aldosterone secretion

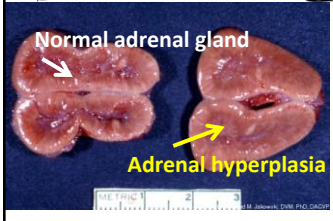
41

41

Addison's Disease = low aldosterone production by adrenal cortex.
↓ salt (reabsorption by kidneys)
↓ water reabsorption by kidneys

Clinical presentation?
Hyponatremia = low blood salt (Na^+), Peeing out salt.
Hyperkalemia = high blood potassium (K^+)
Bradycardia = low HR
Polyuria = pee a lot. Urine output ↑. Peeing out body water.
Hypovolemia=
Hypotension =
Skin bronzing

Conn's Syndrome = "hyperaldosteronism" or excess aldosterone



Clinical presentation?
Hypernatremia = retain salt
Hypokalemia = pee out K^+
Oliguria = reduced urine output
retain water
Hypervolemia=
Hypertension =

42

42

See blank and key flow diagrams on the online syllabus!

For the quick fix and long fix for **LOW BP** and the **KEY**
(This will involve a **quick fix** by the medulla, and a **long fix** by the renin angiotensin aldosterone system)

For the quick fix and long fix for **HIGH BP** and the **KEY**
(This will involve a **quick fix** by the medulla, and a **long fix** by the heart secreting ANP)

For the fix of **HIGH BLOOD OSMOLARITY** and the **KEY**
(This involves the hypothalamus and ADH)

And click **HERE** for a PDF outlining which systems engage when BP is too low or too high. These will each involve a **quick fix** by the medulla, and a **long fix** by a hormone. Also, this goes over blood osmolarity regulation by the hypothalamus and ADH.

43

43

Review

- **Blood volume & blood pressure regulated by**
  - **Medulla baroreceptor response (Quick fix)**
    - **Low BP causes sympathetic arteriole vasoconstriction and ↑ in HR & BP**
    - **High BP causes parasympathetic arteriole vasodilation and ↓ in HR & BP**

  Slow fixes
  - **Heart baroreceptor response (ANP)**
  - **Hypothalamus (ADH release)**
  - **Kidney (renin-angiotensin-aldosterone system)**

44

44

8: Abnormal Blood Pressure

Hypotension = low BP

Hypertension = high BP. Can be due to MANY factors.

45

45

2 types of Hypertension:

1. Primary (idiopathic) Hypertension = exact cause unknown.
2. Secondary Hypertension = result of disease (i.e. kidney or cardiac problem)

Know cause of high BP

Pausible causes of 2° Hypertension

- **Hypervolemia** = high blood volume. Drink excess water
  Can occur with:
  - Excess ADH secretion
  - "Conn's syndrome" (hyperaldosteronism) = Excess aldosterone secretion
- **Stress** too much salt retention, too much water retention
- **Pheochromocytoma** = high epinephrine from adrenal medulla
- **Atherosclerosis** – narrowing of arteries from cholesterol deposits
- **Renal artery disease** (↑ renin or increased angiotensin 2 secretion)
- **Pre-eclampsia (gestational hypertension) =** vasoconstriction of maternal arteries or problems with placenta. Causes still largely unknown.

46

46

Hypertension Drugs

Has hypertension

Can you think of any drugs (especially ones we've covered) that can lower BP?

- Propranolol (blocks β_1 & β_2 adrenergic receptors)
- Atenolol (blocks β_1 adrenergic receptor)
- Na^+ & Ca^{+2} channel blockers
- ACE inhibitors.
- water pill (Lasix)

47

47

Circulatory Shock = inadequate blood flow to all body tissues

Page 155 in text

Many types:

1. Hypovolemic shock = drop in blood volume and BP (blood loss, dehydration)

body response = ↑ heart rate (compensatory to ↑ BP)
= vasoconstriction (to ↑ BP)

2. Septic shock = drop in blood volume and BP from infection (sepsis). Caused by bacterial toxins in blood. Causes vasodilation & ↓ BP

body response = same as for #1.

3. Anaphylactic shock = drop in blood volume and BP due to massive histamine release which causes vasodilation and ↓ BP.

body response = same as #1

4. Congestive heart failure = drop in blood volume and BP due to heart not working. Body response = same as #1

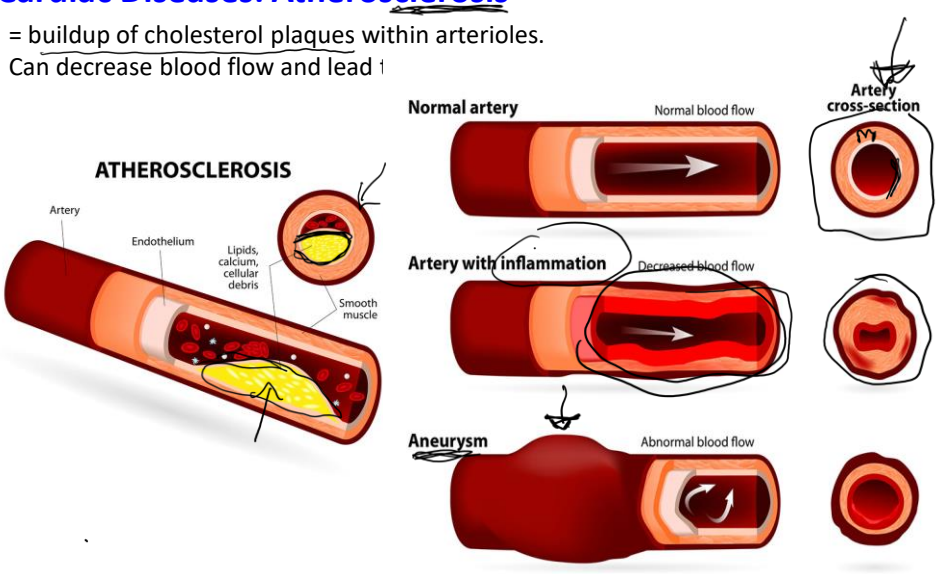
48

48

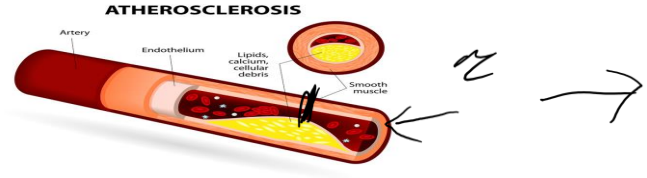
9: Cardiovascular disease vocabulary you must know.

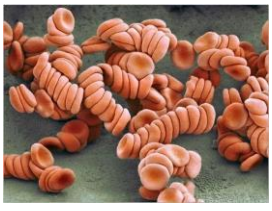
Cardiac Diseases: Atherosclerosis

= buildup of cholesterol plaques within arterioles.
Can decrease blood flow and lead t



49





Formation of **thrombus** = blood clot ↑ risk of thrombus with narrowed artery.

Embolism = floating debris (often a thrombus) that in arteries.

Ischemia = blocked blood flow.

Ex. Stroke = blocked blood flow to brain.

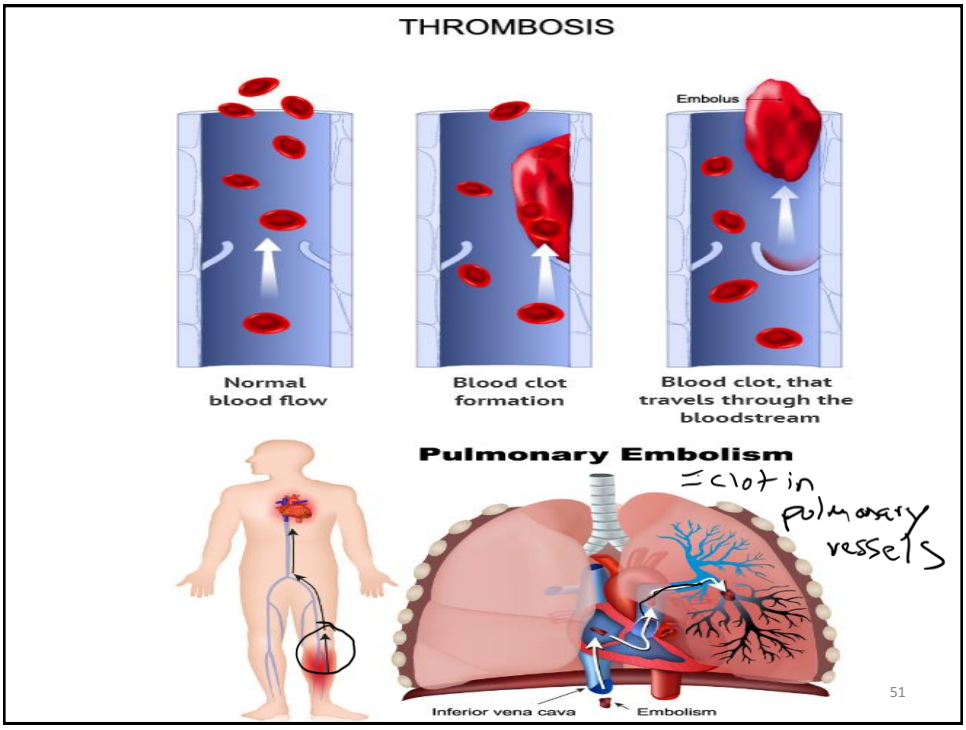
Ex. Heart attack = blocked blood flow to heart.

Arteriosclerosis = scarring of arteries (chronic inflammation)

Aneurysm = swollen artery (weakened by)

50

50



51

Click **HERE** for a brief YouTube diagramatic explanation of an embolism.

Click **HERE** for YouTube video of an actual surgical removal of an embolism. (*Warning: graphic content*)

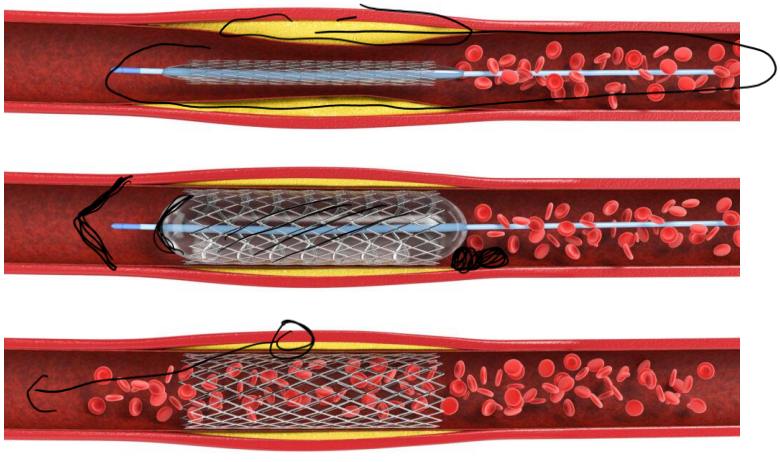
52

52

Angiogram = diagnostic test that uses x-rays to take pictures of your blood vessels. A long flexible catheter is inserted through the blood stream to deliver dye (contrast agent) into the arteries making them visible on the x-ray

53

Balloon Angioplasty with Stent = a catheter with inflatable "balloon", surrounded by a metal mesh, is positioned at the blockage. The balloon squashes plaque to open artery, and then is deflated, leaving the stent in place behind.



54

Review

- **Abnormal blood pressure**
  - **Hypotension**
  - **Hypertension (1° and 2°)**
  - **Some causes of 2° hypertension**
- **Circulatory shock**
  - **Hypovolemic shock**
  - **Septic shock**
  - **Body's response to shock**
- **Atherosclerosis leads to many other circulatory problems.**

55

55

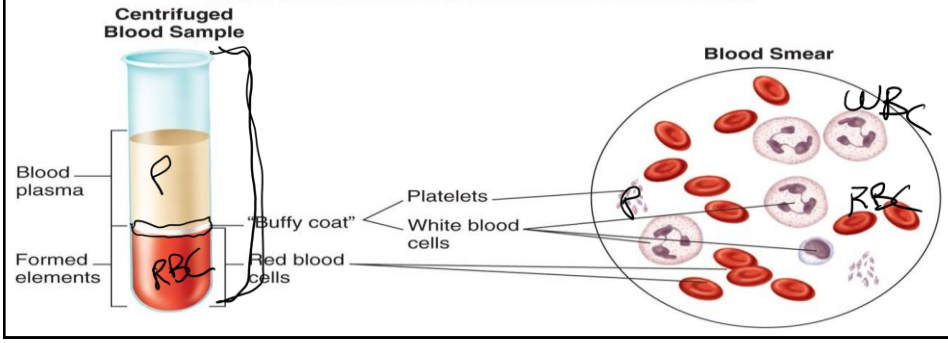
10: Blood Physiology

Blood Composition: Page 123 in text

Whole blood = liquid portion of blood along with the cells.

Plasma = liquid portion of blood (with things dissolved in it like salts, hormones, proteins, etc…)

Cellular portion = RBCs, WBCs, and platelets.



56

Types Blood Cells

red cell

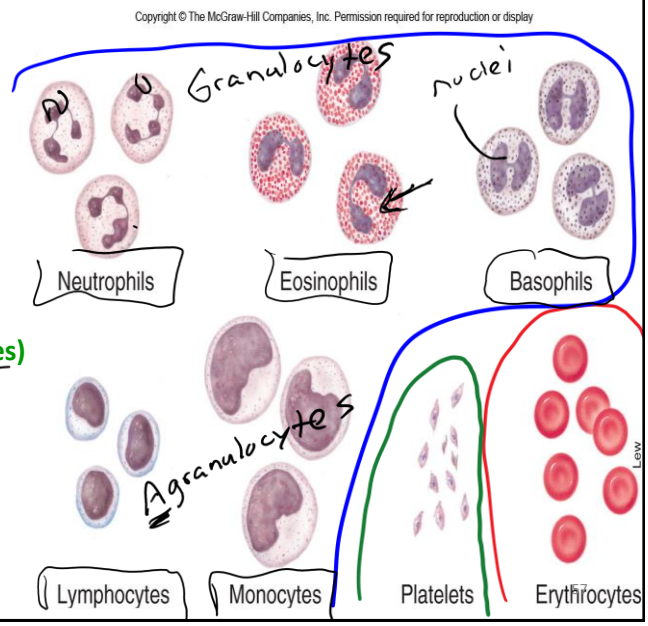
a) RBCs (erythrocytes)

white

b) WBCs (leukocytes)

c) Platelets (thrombocytes)

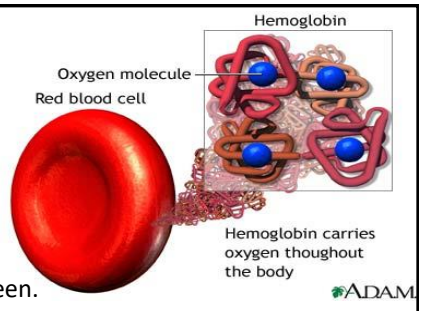
- Never
- Let
- Monkeys
- Eat
- Bananas



57

Page 124 in text

RBCs (Erythrocytes)



> Carry O2 bound to hemoglobin (heme + iron).
> ~500 million new RBC each day!
> RBCs last~120 days then removed by liver & spleen.
- Heme broken into bilirubin (yellow pigment), which liver removes.
- Iron in hemoglobin re-used in new RBCs.

- **jaundice** = yelllowing of the skin & mucus membranes due to liver failure. Because liver failing – not removing bilirubin.

- **erythropoeisis** = process by which new RBCs are made (in bone marrow). Stimulated by hormone **erythropoietin** (released by liver & kidneys.)

58

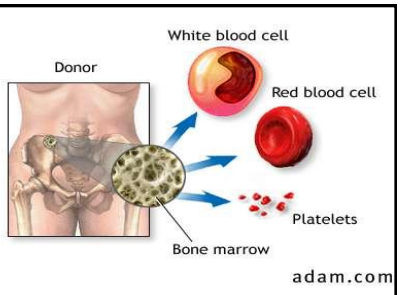
58

Polycythemia = higher than normal RBC count.

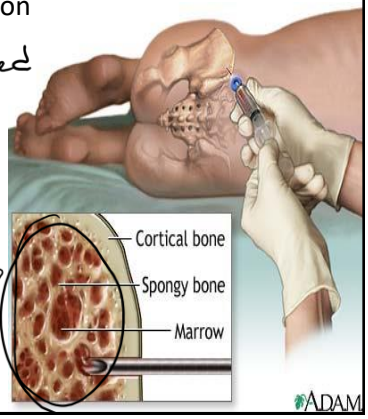
Anemia = lower than normal RBC count.
Clinical App

Can be due to many factors:

- **Iron deficiency anemia** = low iron in diet.



- **Pernicious anemia** = poor vitamin B12 absorption (need to make RBCs)
- **Aplastic anemia** = bone marrow defect (often from chemotherapy treatment.



- **Renal anemia** = low erythropoietin production by kidneys.
- **Autoimmune hemolytic anemia** = immune attack on RBCs (see with Rh disease)

59

Blood Typing

2 Major RBC antigens:

1) ABO antigens
2) Rh antigen

1) ABO

Blood Type A – have A antigens & anti-B antibodies
– receives type A or O blood

Blood Type B – has B antigens & anti-A antibodies
– receives type B or O blood

Blood Type AB – has A & B antigens & no anti-A anti-B antibodies
– "universal recipient", can receive A, B, AB, O blood

Blood Type O – has No antigens, & has anti-A and anti-B antibodies
– "universal donor", but can receive only type O blood

2) Rh factor

Rh+ = have Rh antigen on RBCs (~85% of popluation)

Rh- = not have Rh antigen (~15% of population)

60

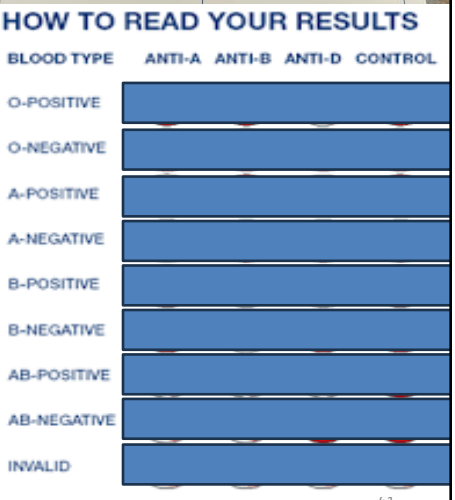
60

Blood Type Test

You put blood sample into each of 3 wells, then add antibodies against the possible Antigens.

If see clotting (**agglutination**)
The RBCs must have antigen to that antibody.

Add patient's blood to test card:
Add anti-A antibodies - if clots = **Type A**
Add anti-B antibodies – if clots = **Type B**
If no clots in either A or B **= Type O**
Add anti-Rh (D) antibodies
– if clots = Rh+
– no clot = Rh-



61

61

Rh incompatibility in pregnancy

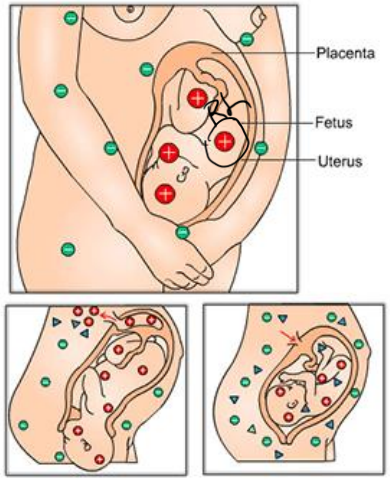
Page 129 in text

If Rh- woman pregnant from Rh+ man – 50% chance baby is Rh+

Risk of exposure of mom's blood stream to fetal RBCs with Rh+ antigens.
(Ex. During miscarriage or tissue tearing during birth or C-section)

Mom's immune system would develop anti-Rh antibodies within 2 weeks of exposure.

- During her next pregnancy if baby Rh+, maternal antibodies cross placenta
- Maternal antibodies attack (hemolyze) fetal RBCs
- "**autoimmune hemolytic anemia**" = immune destruction of RBCs in baby from mom's antibodies



Clinical App

62

Rh incompatibility in pregnancy

Page 129 in text

Prevention:

> If doctor suspects exposure to Rh+ blood in mom's first pregnancy.

> Give injection of anti-Rh antibodies to mom

> antibodies destroy and fetal Rh+ fetal RBCs in mom's body BEFORE her immune system detects & makes own antibodies.

63

63

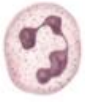
b) WBCs (Leukocytes)

Page 125 in text

Slide updated 3/19/25

2 Groups:

1) Granulocytes = WBCs with granules in cytoplasm

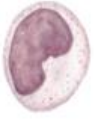


50-70% - **Neutrophils** = **1st responders** to infection/inflammation.

2-4% - **Eosinophils** = see w/chronic inflammation, infection**, allergies, parasites**
(also known to play role in inflammation in asthma, and new treatments try to decrease these WBCs)

<1% - **Basophils** = non-phagocytes, produce histamine & heparin in allergic reaction.

2) Agranulocytes = lack granules.



2-8% - **Monocytes** = phagocytes that seek out , engulf, & destroy pathogens

20-30% - **Lymphocytes** = defense from pathogens
> T cells =
> B cells = become plasma cells to produce antibodies.

Never Let Monkeys Eat Bananas *(neutrophils, lymphocytes, monocytes, eosinophils, basophils)*

64

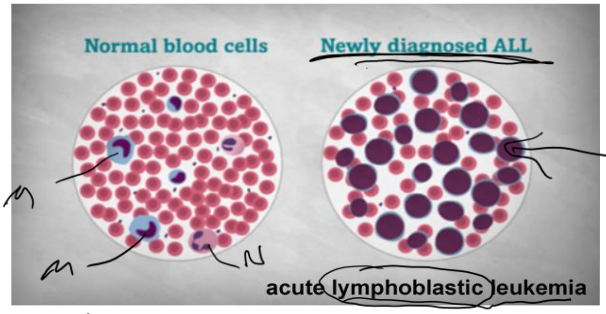
64

Leukocyte disorders – Clinical App

Slide updated 3/19

> **Leukocytosis** = ↑ WBC count (infections!)

> **Leukemia** = ↑ in immature numbers of WBCs, especially lymphocytes. (immature cells not protective)



➢ **Leukopenia** = ↓ WBC count (with immunosuppression, ~~radiation~~ chemotherapy Tx)

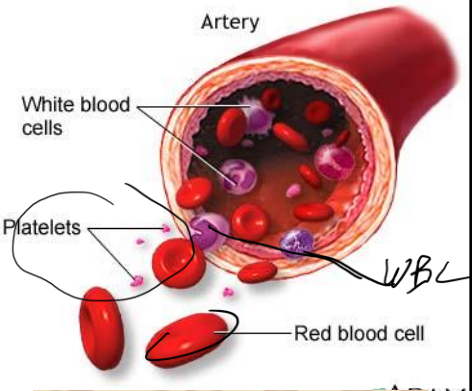
65

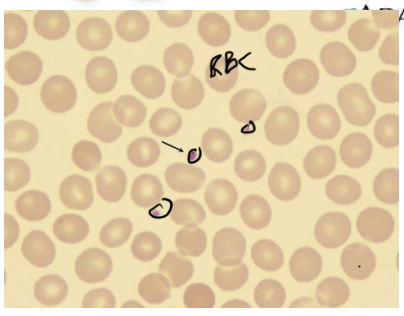
65

c) Platelets (Thrombocytes)

Page 126 in text

- Circulate ~ 5-9 days
- Function to start clot formation
- "**thrombopoietin**" = hormone from liver & kidney that stimulates platelet production by bone marrow.

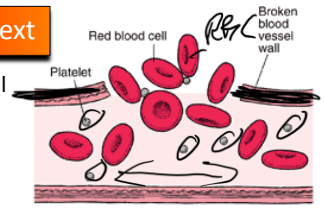


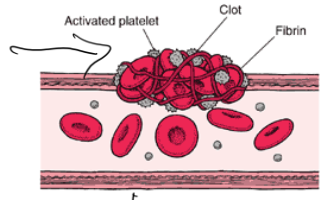


66

3: Physiology of Blood Clotting Page 126 -127 in text

"**Hemostasis**" = stopping bleeding from damaged blood vessel
> A blood vessel is damaged:
> arterioles contrict
> Platelets form a "plug"
- Platelets convert **prothrombin** to **thrombin**
- **Thrombin** activates **fibrinogen**
- Fibrinogen converts into **fibrin threads**
- fibrin threads "knit" the wound closed.

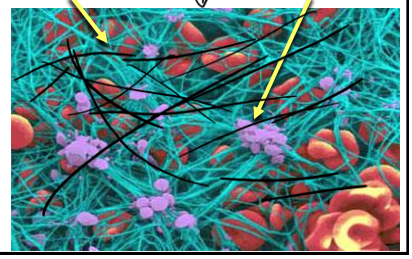




Clotting Disorder Tests:

1. **Bleeding time** w/skin prick (< 1 – 3 min)
2. **Prothrombin time** (PTT test) – treat blood plasma w/citrate and thromboplastin, and add Ca+2 then measure time to clot (< than 12 sec OK, but longer (>35 sec) = prothrombin deficiency)

Fibrin threads

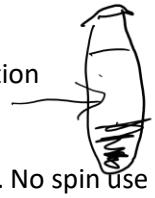


Clotting and Anticoagulants
Clinical App

67

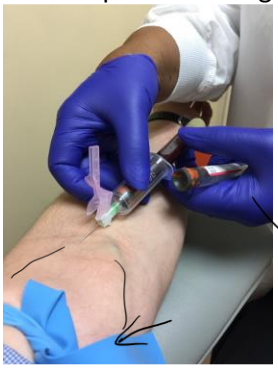
Collecting & Examining Blood Components:

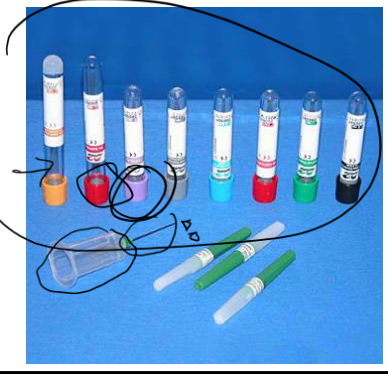
Vacutainer tubes = use vacuum to draw blood into tube.
> **Red top** = no anticoagulant. After spin get serum as fluid portion (use in serological tests)



> **Purple top** = has EDTA anticoagulant. After spin get "plasma". No spin use for blood counts, combs test, disease testing.

Green = heparin anticoagulant. Chromosome testing, ammonia, lactate.





Click **HERE** for Wikipedia description & uses of various vacutainer tubes.

68

68

Review

- **The 3 ways the body regulates blood volume and blood pressure**
  - **Heart baroreceptors: heart secretes ANP when BP is too high (a long fix)**
  - **Arterial baroreceptors and the medulla's cardiac and vasomotor center (provides a quick fix to BP that is either too low or too high)**
  - **Hypothalamic ADH secretion when blood osmolarity rises too high.**
  - **Renin angiotensin aldosterone system, which engages when BP is too low**
- **Blood composition**
  - **Plasma**
  - **Erythrocytes, leukocytes, platelets**
    - **Granulocytes (basophils, eosinophils, neutrophils)**
    - **Agranulocytes (lymphocytes, monocytes)**
- **Blood Typing**
- **Blood clotting with platelets**
- **Techniques for Collecting & Examining Blood**

69

69