

Ch. 7 & 8: Cardiovascular Physiology (updated 1/4/25)

Ch. 8. Objectives:

1. Review: Heart Anatomy, and Systemic & Pulmonary circuits.
2. The Cardiac Cycle and Heart Sounds
3. The Heart's Conduction Cycle & the EKG
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. Objectives

10. Blood Physiology

Anatomy of heart (Review – go over QUICKLY)

Circulatory Circuits (Anatomy review – go over QUICKLY)

- > **Systemic circuit** – from L atrium, arteries, tissues, veins, to vena cava
- > **Pulmonary circuit** – from R atrium, pulmonary arteries, lungs, and pulmonary veins.

Cardiac cycle =& sounds

- > **Ventricular systole** = when both L & R atria close simultaneously, the valves snap shut –**Lub or S1 sound**. At this point the ventricles contract & push blood into pulmonary trunk & aorta
- > **Ventricular diastole** = ventricles relax and fill with blood, both pulmonary & aortic semilunar valves snap shut, producing **Dub or S2 sound**.
- > **Asystole** = no heart sounds

Heart Defects (see online clinical app)

> Heart Murmurs:

- **Innocent murmur** = quiet whoosh sound between S1 & S2 sounds. Can be normal in children (they outgrow it) BUT pathology if adult develops a murmur.
 - **Pathological murmur** = loud whoosh sound between S1 & S2.
 - > could be valves not closing completely, leading to **regurgitant flow** (backflow of blood)
- Causes:
- > Valve prolapse – bicuspid valve can bulge into L ventricle.
 - > **aortic stenosis** (stiffening of aortic valve) could lead to it
 - > **rheumatic heart disease** (autoimmune attack on mitral valve) can lead to it.

Septal Defects (these are before baby is born. These Tholes supposed to close by birth. Not all do so.)

- 1) **Patent foramen ovale** (patent means open) = fetal hole between L & R atria
- 2) **Patent ductus arteriosus** = fetal hole between pulmonary trunk & aorta
- 3) **Ventricular septal defect** = fetal hole in interventricular septum

Review slide

Heart's Conduction System

1. **SA (sinoatrial) node** = heart's pacemaker in upper R atrium. Where an action potential spontaneous happens. Signal jumps to L atrium, and both atria contract simultaneously.
2. **AV (atrioventricular) node** = where the signal stops briefly at base R atrium, allowing time for ventricles to fill.

3. **Bundle of HIS** = signal is within interventricular septum
4. **Purkinje fibers** = signal is within ventricle walls, both ventricles contract simultaneously.

Measuring Heart's Conduction System with EKG

- > **P wave** = when atria contract
- > **QRS wave** = when ventricles contract
- > **T wave** = when ventricles relax
- > there are normal time intervals between these events. Slower or faster intervals = problem!

Heart's Pacemaker Regulation (sympathetic vs parasympathetic)

Concept: action potentials spontaneously occur in pacemaker, BUT the RATE of pacemaker depolarization changes.

1. Pacemaker cells depolarize (muscle cells contract) with sympathetic stimulation

- > opening of Na⁺ and Ca²⁺ channels
- > causes AP or EPSP
- > cells contract

2. Pacemaker cells repolarize (rest) with parasympathetic inhibition

- > opening of K⁺ channels
- > cells relax

Regulation of pacemaker depolarization (heart rate) controlled by cardiac center in Medulla

- > Sympathetic innervation of heart through Thoracic & Cardiac nerves (Review of Ch 6 & 4)
- > **ASK: "What is the neurotransmitter for sympathetic change in HR?" ANS = epinephrine.**
- > **ASK: "What is the receptor for epinephrine on the heart?" ANS = β 1 adrenergic**
- > **ASK: "What ion channels are opened?" ANS = Na⁺ and Ca²⁺ channels**
- > **ASK: "What does that do to HR?" ANS = increased**

> Parasympathetic innervation of heart through Vagus nerves

- > **ASK: "What is the neurotransmitter for parasympathetic?" ANS = Acetylcholine (ACh)**
- > **ASK: "What is the receptor on heart for ACh?" ANS = muscarinic cholinergic**
- > **ASK: "What ion channel opened?" ANS = K⁺ channel**
- > **ASK: "What does that do to HR?" ANS = decreases**

Arrhythmias = abnormal HR (see online clinical app)

- > **Tachycardia** = faster than normal HR, causes hypertension. Treatments to lower HR also lower BP.

Treatment:

- Na⁺ channel blockers
- Ca⁺ channel blockers
- Beta blockers – block β 1 adrenergic receptor on heart to slow HR
 - Ex. General beta blocker = **propranolol** (blocks both β 1 & β 2)
 - β 1 specific blocker = **atenolol** (blocks only the β 1)

- > **Bradycardia** = slower than normal HR

Treatment:

- **Digitalis** = makes Ca²⁺ more available and increases strength of heart contraction
- **β 1 Agonist** = ASK if they can remember from Ch 4 & 6. ANS = **Dobutamine (makes the heart beat faster)**
- **MAO-I class A** = ASK if they can remember what this does. ANS = prevents breakdown of epinephrine in synapse with heart muscle. More epinephrine means increased HR.

Updated slide – drugs to treat hypertension (beta blockers – out with the old, in with the new?)

1st, 2nd, and 3rd generation beta blockers can be non-selective or selective for β 1 adrenergic receptor on heart muscle.

Review slide

Blood Pressure = pressure of arterial blood against vessel wall. Can measure with blood pressure cuff.

> **Systolic BP** = pressure from ventricles contracting. Normal range arterial systolic BP = 80 – 160 mmHg

> **Diastolic BP** = pressure when ventricles relaxed.

Measuring BP with a Sphygmomanometer (they did their first lab experiment of semester doing this. Is REVIEW)

What is happening when BP cuff is inflated vs deflating (**Korotkoff sounds** through stethoscope)

Cardiac Output, Cardiac Rate & Stroke Volume

Cardiac output = volume of blood ejected by each ventricles *per minute*

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

> Average cardiac rate = 70 bpm

> Average stroke volume = 70–80 ml/beat

> Average cardiac output = 5,500 ml/minute [5.5 L/min] **This is ~ same volume of blood in body!*

Things that Influence Cardiac Output:

1) Heart rate

2 Stroke volume

3) End Diastolic Volume (EDV) = the amount of blood in the ventricles during diastole. EDV is affected directly by venous return of blood to heart. As venous return ↑, EDV ↑

As venous return ↓, EDV ↓

4) Stretching of the ventricles (as they fill in diastole) or “contractility”

Frank Starling (Stretching) Law of the Heart – states that:

As EDV ↑, ventricle stretch ↑, and so cardiac output ↑

As EDV ↓, ventricles stretch ↓, and so cardiac output ↓

As contractility ↓, stroke volume ↓, so cardiac output will ↓

5) Total peripheral resistance (TPR) = resistance of blood flow in arteries (depends on blood viscosity, how long vessels are, and their diameter). *[imagine blood that is thick like corn syrup, there would be larger resistance of that blood in artery than if blood was less viscous, like water.]*

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

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

Review Slide

3 ways body regulates cardiac output, blood volume, & blood pressure:

Quick Fix = arterial baroreceptors and medulla’s sympathetic or parasympathetic response

Long Fix = hormonal fix that affects salt and/or water retention at kidneys.

1. Baroreceptor reflex

a) Heart baroreceptors (aortic arch & carotid sinus) – if BP high ANP release, if BP low – inhibit ANP

b) Medulla oblongata baroreceptors – cardiac & vasomotor center response. Sense low or high BP

2. Hypothalamus – ADH release

3. Kidneys - renin-angiotensin-aldosterone system

There are practice flow diagrams on the online syllabus for these!

Baroreceptors = blood pressure receptors located within aortic arch & carotid sinus. Baroreceptors involved in regulating cardiac output (heart rate) depending on blood pressure in heart. Fig. 14.27

Artery Baroreceptors sense change in BP & signal medulla oblongata.

"stimulus" = either increased or decreased blood pressure.

"sensor" = medulla *cardiac* center and *vasomotor center* (autonomic regulation of cardiac functions)

"motor response"

> sympathetic **a)** adrenergic (epinephrine) stim. of B1 receptors of myocardial cells (subsequent activation of G-proteins, cAMP, and Ca⁺ release) AND ALSO SIMULTANEOUSLY

b) adrenergic (epinephrine) stim of endothelium of arterioles (vasoconstrict)

>parasympathetic = Ach stim of nicotinic receptors of myocardial cells & endothelium of arterioles

"effectors" = myocardial cells (changes heart rate) and endothelium of arterioles (vasodilate)

IF baroreceptors sense BP decreased (<80 mmHg)

Autonomic response is sympathetic

Neurotransmitter = epinephrine

Epineph binds to B1-adrenergic receptors on heart (HR increases)

Systemic arteries vasoconstrict

BP increases back to normal

IF baroreceptors sense BP increased (> 160 mmHg):

Autonomic response is parasympathetic

Neurotransmitter = ACh

ACh binds to muscarinic cholinergic receptors on heart (HR decreases)

Systemic arteries vasodilate

BP goes back down to normal.

Heart – Baroreceptor Reflex

"stimulus" = **increased BP (hypertension, systolic arterial pressure > 160 mmHg)**

"sensor" = baroreceptors in aortic arch & carotid sinus

"response" = ANP release from heart

"effect" = ANP causes:

> increased glomerular filtration rate (GFR)

> decreased water reabsorption (by DCT & CD)

> increased urine output (pee more)

> decreased blood volume (hypovolemia)

> **decreased BP**

2) Hypothalamus – ADH release

Stimulus = IF ↑ in osmolarity above setpoint (NOT a change in BP)

Sensor, integrating center, effector = hypothalamus, which secretes ADH (antidiuretic hormone)

Effect - ↑ water reabsorption by kidneys (retain water)

- ↓ urine output (pee less)

- ↑ blood volume (which does ↑ BP, BUT that was not the goal of ADH)

- **↓ blood osmolarity back to normal**

Stimulus = - If ↓ blood osmolarity

> Inhibition of ADH release (get opposite effects)

↓ water retention

↑ urine output

↓ blood volume

↑ **blood osmolarity back to normal**

Diabetes insipidus = inadequate ADH production by hypothalamus (OR ADH receptor problem with tubules)

- > insufficient water reabsorption from kidney nephrons.
- > Urine output increased (pee more) = “polyuria”
- > Less water returned to bloodstream, blood volume decreased,
- > decreased BP
- > Patients thirsty!! drink a lot of water to compensate. = “polydipsia” Chronic state of combating dehydration.

** always Sipping on water!

3. Kidneys - Renin-Angiotensin-Aldosterone System

*Your kidneys filter total blood volume (5.5 L) about 30 times a day and collect about 180 L/day of filtrate
98-99% of that is reabsorbed. Water reabsorption controlled by several hormones.

“stimulus” = low BP in renal artery of kidneys

sensor, integrating center, & effector = juxtaglomerular apparatus (JGA) of kidney, which secretes renin
Effect = BP increases back to normal

IF BP is too low:

- > sensed by **JGA**, which releases renin
- > Renin causes liver to convert **angiotensinogen** to **angiotensin 1**
- > at lungs, **angiotensin 1** converted to **angiotensin 2** by **ACE** (angiotensin-converting enzyme)
- > Angiotensin 2 causes: a) arterial vasoconstriction & b) adrenal cortex to secrete **aldosterone**
- > Aldosterone ↑ salt retention, then water retention (water ALWAYS follows salt) from the kidneys.
- > ↑ blood volume and ↑ BP

See online clinical app on use of ACE inhibitors to decrease BP in people with hypertension.

If BP low this process inhibited

ACE inhibitors – hypertension drug that prevents angiotensin 1 from being converted into angiotensin 2 by lungs.

Without angiotensin 2 you won’t have direct arterial vasoconstriction, AND there won’t be stimulation for aldosterone secretion by adrenal cortex. End effect is a drop in blood pressure through arterial vasodilation and reduced salt and water retention by kidneys (due to lack of aldosterone). Pee out salt and water reduces blood volume and BP.

Addison’s disease = insufficient **Aldosterone** – not enough Na⁺ reabsorbed (Review from Ch 11!)

- hypoglycemia & weight loss
- ↓ Na⁺, ↓ water reabsorbed, ↓ blood volume (hypovolemia) & BP (hypotension)
- “bronzing” of skin – overstimulation of melanocytes
- “hyperkalemia” (excess K⁺ reabsorbed, leads to ↓ HR and heart failure)

Conn’s Syndrome (Hyper-aldosteronism) = excess aldosterone

ASK: if Conn’s syndrom is opposite of Addison’s, what would be the clinical presentation?

ANS- ↑ salt & water reabsorption, ↑ blood volume (hypervolemia) and ↑ BP (hypertension)

- “hypokalemia” – excess loss of K⁺,

Review slide

Abnormal blood pressure

- > Hypotension
- > Hypertension

Primary (idiopathic) hypertension = when exact cause not known

Secondary hypertension = when cause is known

Possible causes (many!):

- > hypervolemia = higher than normal blood volume (due to excess ADH, Conn's syndrome)
- > chronic stress
- > pheochromocytoma = excess epinephrine secretion by adrenal medulla (review of Ch 8)
- > renal artery disease (increased renin secretion)
- > **pre-eclampsia** = gestational hypertension from vasoconstriction of maternal arteries

drugs to treat hypertension:

- > **diuretics** = block salt retention in kidneys, so water not retained. Causes excess urination to decrease BP. Ex. Lasix "loop diuretic (furosemide), carbonic anhydrase inhibitors, thiazides.
- > **Propranolol** (1st generation non-selective beta blocker) – B1 & B2 blocker
 - > **Atenolol** (2nd generation selective beta blocker) – ASK what does it do? Review of CH 6 & 4. ANS = it's a B1-adrenergic block that decreases heart rate to decrease BP
 - > **Ca²⁺ and Na⁺ channel blockers**
 - > **ACE inhibitors**
- > can anyone remember a 3rd generation beta selective beta blocker?? – ANS = **Betaxolol**

Circulatory Shock = systemic drop in blood flow to all body tissues

Types:

- > **hypovolemic shock** = drop in blood volume & BP (blood loss or dehydration)
- > **septic shock** = drop in BP from systemic vasodilation caused by bacterial toxins in blood
- > **anaphylactic shock** = drop in BP from systemic vasodilation caused by massive histamine release by mast cells in response to an allergy.
- > **congestive heart failure** = drop in BP because heart not working properly.

Body responds the same to all of these – tries to increase HR and vasoconstrict arteries to increase BP.

Cardiovascular Disease Vocabulary You Must Know:

Atherosclerosis = buildup of cholesterol plaques within arteries, which narrows them & decreases blood flow. Can lead to many secondary problems:

- > **thrombus** = blood clot forms in arteries. Clot can break off leading to...
- > **embolism** = clot (of blood cells or cholesterol) breaks free in blood, which could lead to ...
- > **ischemia** = blocked blood flow
 - Ex. Stroke or heart attack
- > **arteriosclerosis** = hardening or scarring of arteries from chronic inflammation from presence of blood clots or cholesterol plaques. Scarring weakens arteries making them prone to...
- > **aneurysm** = swelling of artery from weakened walls. Can lead to rupture.
- > **angiogram** – x rays used to view arterial vessels to determine blockage.
- > **balloon angioplasty** – thread a catheter with inflatable balloon into blocked artery to open it.

Review slide

10. Blood physiology

Whole blood is composed of:

- > plasma = liquid portion of blood. Mostly water with dissolved proteins, salts, glucose, and other things
- > cellular portion = RBCs, WBCs, and platelets

Types of Blood Cells:

- > **RBCs (erythrocytes or red cells)**
- > **WBCs (leukocytes or white cells)**
- > **Platelets (thrombocytes)**

RBCs

- > carry O₂ bound to hemoglobin pigment
- > fast reproducing cells! Make 500 million new ones/day
- > live ~120 days before body destroys them, components recycled
 - iron reused
 - heme broken into **bilirubin** (yellow pigment) by liver

Jaundice = yellowing of skin, mucous membranes, and sclera of eyes due to buildup of bilirubin in blood with liver failure.

Erythropoiesis = making of new RBCs (by bone marrow) in response to hormone erythropoietin (released by liver & kidneys)

Erythropoiesis stimulated by low blood O₂

Stimulus = low blood O₂

Sensor and effectors = liver and kidneys, which secrete erythropoietin

Effect = bone marrow makes new RBCs (process actually takes ~ 2weeks)

Anemias – see online clinical app

- > **polycythemia** = higher than normal RBCs produced (not an anemia, and is usual condition)
- > **anemia** = lower than normal RBCs produced (can be due to many things):
 - **pernicious anemia** = due to poor vitamin B12 absorption in GI (easy to treat w/supplements)
 - **aplastic anemia** = due to bone marrow problem. Can happen w/chemotherapy
 - **renal anemia** = low erythropoietin production by kidneys
 - **autoimmune hemolytic anemia (erythroblastosis fetalis or Rh disease)** = when RH – mother carries an RH+ fetus, and there is mixing of blood, mom's body makes antibodies against the RH factor. Subsequent pregnancies risk lysis of fetal RBC in utero from antibody attack. See online clinical app.

Blood typing!

Two major human RBC antigens: 1) ABO system, and 2) Rh system

1) ABO – based on presence of antigens on RBCs

Blood type A = have A antigens, and anti-B antibodies. Can receive only type A or type O blood. Would have reaction to any type B blood.

Blood type B = has B antigens, and anti-A antibodies. Can receive only type B or type O blood

Blood type AB = has both A and B antigens, and no antibodies. Can receive all types of blood (A, B, AB, & O). Universal recipient.

Blood type O = has no A or B antigens, but has anti-A and anti-B antibodies. Can only receive type O blood

2) Rh factor

Rh+ = have the Rh antigen on RBCs, **Rh-** = don't have them.

Examples of blood type test – if agglutination present when antibodies added to blood sample, that shows type

GO OVER WITH STUDENTS what each card shows:

ANS: In order: O+, O-, A+, A-, B+, B-, AB+, and AB-

Rh problems with pregnancy

> If Rh- woman carries Rh+ fetus, and there is mixing of maternal & fetal blood (during c-section, or difficult birth), mom's immune system responds by making anti-Rh antibodies within about 2 weeks. This would cause problems with future pregnancy with Rh+ baby and lead to autoimmune hemolytic anemia in baby.

Treatment: common practice now for Rh- mother to receive an injection of antibodies against Rh antigen. If any fetal blood cells in her body, the antibodies will destroy before her immune system can detect them and form antibodies.

WBCs – 2 groups:

1) **granulocytes** = because they have granules in the cytoplasm. 3 kinds:

- > **Neutrophils** – responds first and fast to pathogens.
- > **Eosinophils** – respond with chronic inflammation, allergies, and parasites
- > **Basophils** – produce histamine & heparin in allergic reactions

2) **Agranulocytes** = lack granules in cytoplasm. 2 kinds:

- > **Monocytes** = phagocytes that seek out, consume, & destroy pathogens
- > **Lymphocytes** = defense from pathogens
 - T cells = includes cytotoxic cells that can directly kill pathogens & activate other immune cells
 - B cells = become plasma cells that produce antibodies.

Leukocyte disorders – see online clinical app

- > **leukocytosis** = higher than normal WBC count (infections!)
- > **leukemia** = increased number immature WBCs in blood (especially lymphocytes). Indicates bone marrow working hard to push out new WBC and releases them before maturity.
- > **Leukopenia** = lower than normal WBCs (seen with immunosuppression as with AIDS or radiation or chemotherapy treatment)

Platelets = involved in clot formation to repair wounds.

> they are produced by bone marrow in response to **thrombopoietin** = hormone from liver and kidneys.

Clot formation

Hemostasis = stopping bleeding from damaged vessel

- > blood vessel is damaged
- > arterioles constrict
- > platelets gather and:
 - form plug
 - convert **prothrombin** to **thrombin**
 - thrombin causes **fibrinogen** to become **fibrin** threads, which “knit” the wound closed

Clotting disorder test:

1) **Bleeding time** with skin prick (should be less than 1 – 3 min)

2) **Prothrombin time** – treat blood plasma with citrate & thromboplastin, & add Ca²⁺, then measure time-to-clot (should be < 12 sec).

see online clinical app

Collecting & examining blood:

Vacutainer tube system – uses tube with vacuum and needle in plastic sheath. Most common tubes:

- > **Red top tube** – no anticoagulant, so blood will clot and separate into **serum** liquid portion and cell portion.
- > **Purple top tube** – has EDTA coagulant. Can centrifuge to separate plasma liquid from cellular portion, or keep blood mixed for blood counts and whole blood tests (assays)
- > **Green top tube** – has heparin anticoagulant. For blood chemistry panels.

Review slide