

## Cardiovascular System I: The Heart Chapter 17

**CV system** = \_\_\_\_\_  
[heart *pumps* blood into blood vessels throughout the body]

### → **Module 17.1 Overview of the Heart**

#### LOCATION & STRUCTURE OF THE HEART

- **Heart**

- cone-shaped organ
- located slightly to left side in thoracic cavity  
(\_\_\_\_\_)
- rests on diaphragm
- \_\_\_\_\_: inferior aspect
- ~ 250 to 350 grams (< 1 lb.)

- Chambers and external anatomical features:

**Chambers** – **RA** and **LA** atria (**atrium**)

**RV** and **LV** ventricles

\_\_\_\_\_ sulcus

- external indentation between the atria and ventricles

\_\_\_\_\_ sulcus

- external depression *between* RV and LV

**Veins** - carry blood \_\_\_\_\_

**Arteries** carry blood \_\_\_\_\_

- **Great vessels** = main veins and arteries that bring blood to and from heart  
[SVC, IVC, pulmonary V., pulmonary A., aorta]

#### PULMONARY & SYSTEMIC CIRCUITS

#### **Pulmonary Circuit:**

- Right side of heart (**pulmonary pump**) pumps blood to lungs
  - \_\_\_\_\_ deliver *oxygen-poor* (**deoxygenated**) blood to lungs
  - **Gas exchange** between **alveoli** and **pulmonary capillaries**

- \_\_\_\_\_ deliver oxygen-rich (**oxygenated**) blood to left side of heart

### Systemic Circuit:

- **Systemic pump (left side of heart)**

- receives \_\_\_\_\_ blood from pulmonary veins and *pumps* it to rest of body
- **Systemic arteries** pump **oxygen-rich** (\_\_\_\_\_) blood to all systems of body (not lungs)
- Gas exchange at **systemic capillaries**
- \_\_\_\_\_ return **oxygen-poor (deoxygenated) blood to RA**
- Pulmonary circuit *-low-pressure circuit* → \_\_\_\_\_
- Systemic circuit *high-pressure circuit* → \_\_\_\_\_

### FUNCTIONS OF THE HEART

- Heart helps maintain BP (blood pressure)
  - \_\_\_\_\_ of contraction influence BP and blood flow to organs
- Atria produce hormone: **atrial natriuretic peptide (ANP)**
  - ANP \_\_\_\_\_ BP by decreasing Na<sup>+</sup> *retention* in kidneys → decr. osmotic H<sub>2</sub>O reabsorption

### → Module 17.2 Heart Anatomy and Blood Flow Pathway

### PERICARDIUM

**Pericardium** – membrane surrounding heart

1. **Fibrous pericardium** – outermost layer
2. **Serous pericardium** – produces **serous fluid**
  - \_\_\_\_\_  
[pericardial cavity]
  - **Visceral pericardium** – (aka \_\_\_\_\_)

–

### Pericardial cavity

- contains serous fluid (**pericardial fluid**)
- acts as a \_\_\_\_\_

### HEART WALL

1. Epicardium - outmost layer
2. \_\_\_\_\_
  - middle muscle layer
  - [What type of muscle??] \_\_\_\_\_
  - fibrous skeleton (dense irregular collagenous CT)
3. Endocardium - innermost endothelial layer



### Cardiac Tamponade

- Pericardial cavity fills with excess fluid → cardiac tamponade
- Causes:
- Fibrous pericardium - strong but not very flexible, excess fluid in pericardial cavity *squeezes* heart; reduces filling of ventricles
- Treatment

### CORONARY CIRCULATION

Coronary vessels (supply heart wall):

- Branch off ascending aorta:
  - 1. \_\_\_\_\_ →
    - post. interventricular (post. descending a.)
    - marginal branch
  - 2. left coronary artery →
    - \_\_\_\_\_ → ant. interventricular a.
    - (left ant. descending) \_\_\_\_\_

- \_\_\_\_\_
  - **Great cardiac vein**
  - **Small cardiac vein** → \_\_\_\_\_ → \_\_\_\_\_
  - **Middle cardiac vein**
- **Coronary artery disease (CAD)**
  - buildup of \_\_\_\_\_ (fatty material) in coronary arteries
  - decreases blood flow to myocardium → \_\_\_\_\_
  - Symptoms: angina pectoris
  - leading cause of death worldwide
- **Myocardial infarction (MI) or heart attack**
  - Most dangerous potential consequence of CAD
  - Occurs when \_\_\_\_\_
  - Clot forms → myocardial tissue infarct
  - **Symptoms** include chest pain *radiates* to left arm shortness of breath, sweating, anxiety, and nausea and/or vomiting
  - Women may present with \_\_\_\_\_
  - Survival after MI depends on *extent* and *location* of damage
  - Dead cells are replaced with \_\_\_\_\_
  - Death of part of myocardium increases \_\_\_\_\_
  - **Risk factors** include smoking, incr. BP, poorly controlled diabetes, high levels of certain lipids, obesity

\_\_\_\_\_ diagnostic test for CAD

#### Treatments

- modify *Lifestyle*
  - *medications*
  - then invasive treatments
- **Coronary** \_\_\_\_\_ - balloon is *inflated* in blocked artery and \_\_\_\_\_ inserted
  - **Coronary artery bypass grafting (CABG)**
    - other vessels are *grafted* onto diseased coronary artery to *bypass* blockage

PATH OF BLOOD THROUGH THE HEART

- Heart consists of four chambers:
    - 2 Atria
      - \_\_\_\_\_
      - pump through atrioventricular (AV) valves into ventricles
    - 2 Ventricles
      - \_\_\_\_\_
      - carry blood through systemic **or pulmonary circuit**
- {
- Superior vena cava (\_\_\_\_\_)
  - Inferior vena cava (\_\_\_\_\_)
  - \_\_\_\_\_

1. Right Atrium (RA)

<Right atrioventricular (AV) valve>  
(\_\_\_\_\_)

2. Right Ventricle (RV)

chordae tendineae  
papillary muscles  
< Pulmonary semilunar valve>

→ pulmonary trunk

→ LUNGS → \_\_\_\_\_

3. Left Atrium (LA)

<left Atrioventricular (AV) valve>  
(\_\_\_\_\_)

4. Left Ventricle (LV)

chordae tendineae  
papillary muscles

< aortic semilunar valve >

→ Ascending aorta:

→ \_\_\_\_\_

→ **Aortic Arch**

- Brachiocephalic artery
- \_\_\_\_\_ (**LCC**) artery
- \_\_\_\_\_ artery

GREAT VESSELS, CHAMBERS, AND VALVES

- **Pectinate muscles** – muscular ridges inside RA
- **Interatrial septum** – wall between RA & LA
- **Fossa ovalis** – indentation in interatrial septum; *remnant* of opening (\_\_\_\_\_) from fetal circulation
- **Trabeculae carneae** – ridged surface in Ventricles “*beams of flesh*”

RV – \_\_\_\_\_

LV – \_\_\_\_\_

LV wall = 3x \_\_\_\_\_ than RV

HEART VALVES

Tricuspid (\_\_\_\_\_)

Pulmonary semilunar

Bicuspid (\_\_\_\_\_)

Aortic semilunar

Pulmonary semilunar valve - \_\_\_\_\_



**Valvular Heart Diseases**

- Diseases of heart valves
  - \_\_\_\_\_ (present at birth) or \_\_\_\_\_ (infection, cancer, or immune system disorder)

- Two major types of valvular defects:
  - Insufficient valve
    - fails to *close* fully, blood *leaks backward*
  - \_\_\_\_\_ valve (narrowing)
    - calcium deposits → hard and inflexible
- Both valve disorders may cause
- Symptoms: enlargement of heart, fatigue, dizziness, and heart palpitations
- Mitral and aortic valves are ones most commonly affected

→ **Module 17.3 Cardiac Muscle Tissue Anatomy and Electrophysiology**

ELECTROPHYSIOLOGY

- Cardiac muscle exhibits
- Cardiac muscle cells contract in response to electrical excitation in form of **APs**
- Cardiac muscle cells do not require stimulation from nervous system to *generate APs*
- \_\_\_\_\_
  - specialized cardiac muscle cells (=1% of cardiac muscle cells)
  - coordinate cardiac electrical activity
  - *rhythmically and spontaneously* generate APs to other type of cardiac muscle cell (\_\_\_\_\_)

HISTOLOGY OF CARDIAC MUSCLE TISSUE AND CELLS

- Cardiac muscle cells
  - 
  -

- 
- generate tension through sliding-filament mech.
  - Ex. of Structure-Function Core Principle
- Like skeletal muscle fibers, cardiac muscle cells contain *selective*
- *Opening & closing* action of these ion channels
  - both pacemaker & contractile cardiac APs

#### ELECTROPHYSIOLOGY OF CARDIAC MUSCLE

- **Cardiac conduction system**
  - Pacemaker cells undergo *rhythmic, spontaneous depolarizations* → APs
- \_\_\_\_\_
  - Permits heart to contract as a *unit* and \_\_\_\_\_
- Sequence of events of *contractile cell AP* resembles that of *skeletal muscle fiber AP* with one exception: \_\_\_\_\_
  - Plateau phase *lengthens* cardiac AP → \_\_\_\_\_ providing time required for heart to *fill* with blood;
  - also increases \_\_\_\_\_;
  - \_\_\_\_\_ (sustained contraction) in heart by *lengthening refractory period*
  - Refractory period in cardiac muscle cells is so long that cells cannot maintain a *sustained contraction*
  - allows heart to \_\_\_\_\_ before cardiac muscle cells are stimulated to contract again

#### CARDIAC CONDUCTION SYSTEM

- \_\_\_\_\_ **node (SA node)**
  - located in upper RA
  - **60 to 100** bpm influenced by SNS & PSN



\_\_\_\_\_ **node (AV node)**

- located near tricuspid valve
- 40 bpm
- AV node delay

**Purkinje fiber system**

- Purkinje fiber system:
  - Atrioventricular bundle (\_\_\_\_\_)
  - Right and left \_\_\_\_\_
  - Purkinje fibers
    - located in ventricular walls

ELECTROPHYSIOLOGY OF CARDIAC MUSCLE

**AV node delay**

- allows atria to depolarize (and *contract*) before ventricles, giving ventricles time to *fill* with blood
- also helps to prevent current from flowing *backward* from \_\_\_\_\_ into AV node and atria
- SA node = *main pacemaker* of heart
- **Sinus rhythms** = \_\_\_\_\_

• **Electrocardiogram (ECG)**

- \_\_\_\_\_ in cardiac muscle cells over time
- *electrodes* placed on patient's skin (6 on chest, 2 on each leg)
- detects *disturbance* in electrical rhythm = \_\_\_\_\_ or **arrhythmia** (= no rhythm)

- ECG represents *depolarization* or *repolarization* of parts of heart

- **P wave** represents \_\_\_\_\_

- **QRS complex** represents \_\_\_\_\_
- **T wave** represents \_\_\_\_\_

What's missing??



## Dysrhythmias

**Cardiac dysrhythmias** have 3 basic patterns:

1. Disturbances in *heart rate (HR)*:

- \_\_\_\_\_ =  $HR < 60$  bpm
- **Tachycardia** =  $HR > 100$  bpm
  - sinus tachycardia** = *regular, fast rhythm*

2. Disturbances in *conduction pathways*

– disrupted by accessory pathways between upper & lower chambers  
or by \_\_\_\_\_

- Heart block at **AV node**;

- *P-R interval* is longer than normal, due to incr. time for impulses to spread to ventricles through AV node;  
extra P waves are present, indicates that some APs from SA node are not being conducted through AV node

- **Right or left bundle branch block**

- generally widens *QRS complex* due to depolarization taking longer to spread through ventricles

3. **Fibrillation** = electrical activity goes haywire → parts of heart to depolarize and contract while others are repolarizing and not contracting

- *bag of worms writhing*

– **Atrial fibrillation**

- generally not life threatening
- atrial contraction isn't necessary for ventricular filling
- ECG tracing "irregularly irregular" rhythm (one that has no discernible pattern) that lacks P waves

- **Ventricular fibrillation**
  - immediately life-threatening
  - ECG exhibits chaotic activity
    - **defibrillation** (an electric shock to heart) depolarizes all ventricular muscle cells simultaneously
    - SA node will *resume* pacing heart after shock is delivered (ideally)
- “Flat-lining” = **asystole**
- defibrillation is not used for asystole because heart is not fibrillating and there is no electrical activity to reset
- instead, treated with **CPR** and pharmacological agents that stimulate heart such as **atropine** and **Epi**

### → **Module 17.4 Mechanical Physiology of the Heart: The Cardiac Cycle**

#### INTRODUCTION TO MECHANICAL PHYSIOLOGY

- **Mechanical physiology** - actual processes by which blood *fills* and is pumped out of chambers
- **Heartbeat** =
- **Cardiac cycle** - sequence of events that take place from one heartbeat to next (systole followed diastole for each chamber)

#### PRESSURE CHANGES, BLOOD FLOW, AND VALVE FUNCTION

Blood flows in response to *pressure gradients* (**Gradients Core Principle**); as ventricles contract and relax, pressure in chambers changes, causing blood to *push* on valves and open or close them:

- \_\_\_\_\_ (contraction phase)
  - Both of AV valves are forced *shut* by blood pushing against them
  - Both of semilunar valves are forced *open* by outgoing blood
- \_\_\_\_\_ (relaxation phase) –

Press. In ventricles falls below those in atria and in pulmonary trunk and aorta

→ forces AV valves *open*, \_\_\_\_\_

→ Higher pressures in pulmonary trunk and aorta push cusps of semilunar valves *closed*

• **Stethoscope** – used to listen to (**auscultate**) rhythmic **heart sounds**:

– **S1** (“lub”) = \_\_\_\_\_

– **S2** (“dub”) = \_\_\_\_\_



### Heart Murmurs and Extra Heart Sounds

- **Heart murmur** - *turbulent* blood flow through heart often due to *defective valves*, defective chordae tendineae, or holes in interatrial or interventricular septum

• **Cardiac cycle** =

- Cycle is divided into four main phases that are defined by actions of ventricles and positions of valves: **filling**, **contraction**, **ejection**, and **relaxation**

#### 1. Ventricular filling phase of cardiac cycle

- blood drains \_\_\_\_\_
- Pressures in LV and RV are lower than in atria, pulmonary trunk, and aorta
  - Higher pressures in pulmonary trunk and aorta cause semilunar valves to be *closed*; prevents backflow of blood into ventricles

#### → **Module 17.5 Cardiac Output and Regulation**

##### INTRODUCTION TO CARDIAC OUTPUT AND REGULATION

#### Heart rate (HR)

= 60–80 cardiac cycles or bpm

#### Stroke volume

= ~70 ml/beat (amt. of blood ejected from each \_\_\_\_\_ in a beat)

#### Cardiac output (CO)

= \_\_\_\_\_ into pulmonary & systemic circuits \_\_\_\_\_

##### DETERMINATION OF CARDIAC OUTPUT

- **C.O.** = *heart rate* x *stroke volume*:

- $72 \text{ beats/min} \times 70 \text{ ml/beat} = 5040 \text{ ml/min}$   
~5 liters/min (C.O.)
- Resting C.O. ~ averages about 5 liters/min;  
RV pumps ~ 5 liters into pulmonary circuit  
LV pumps same *amt.* to systemic circuit

Normal adult blood volume = ~ 5 liters

[∴ \_\_\_\_\_]

#### FACTORS THAT INFLUENCE STROKE VOLUME

##### Frank-Starling law

- Increased ventricular muscle cells *stretch*, leads to → \_\_\_\_\_
- *Ensures that vol. of blood discharged from heart is equal to vol. that enters it*
- Important during exercise, when C.O. must increase to meet body's needs



##### Ventricular Hypertrophy

#### FACTORS THAT INFLUENCE HEART RATE

- HR due to rate at which SA node generates APs
- \_\_\_\_\_ at which SA node depolarizes = **chronotropic agents**
  - *Positive* chronotropic agents
    - SNS, some hormones, increased body temp.
  - *Negative* chronotropic agents
    - PSN, decreased body temperature

#### REGULATION OF CARDIAC OUTPUT

Heart is autorhythmic but still requires *regulation* to ensure C.O. meets body's needs at all times

- Regulated by \_\_\_\_\_ (ANS) and \_\_\_\_\_ systems  
SNS (NEpi) → \_\_\_ HR, \_\_\_ force of contraction

PSN (ACh) → \_\_\_\_ HR, \_\_\_\_ force of contraction

- \_\_\_\_\_
  - \_\_\_\_\_ – affected by SNS → Epi and NEpi
  - thyroid hormone and glucagon
- \_\_\_\_\_
  - Aldosterone and antidiuretic hormone increase blood vol. → incr. C.O.
  - ANP decreases blood vol. → reduces C.O.
- Other factors that influence cardiac output:
  - [Electrolyte] in ECF
  - \_\_\_\_\_
    - SA node fires more *rapidly* at higher body temp. and more *slowly* at lower body temp.
  - Age
  - Exercise

#### HEART FAILURE

Heart failure (formerly CHF) = any condition that reduces heart's ability to pump *effectively*:

- \_\_\_\_\_ and/or M.I, valvular heart diseases, any disease of heart muscle (cardiomyopathy) and electrolyte imbalances
- Heart failure → decreased SV → \_\_\_\_\_
- Signs and symptoms of heart failure depend on *type* of heart failure and *side* of heart that is affected
  - LV failure, blood often backs up within pulmonary circuit; known as **pulmonary congestion** → \_\_\_\_\_
- Both RV and LV failure → **peripheral edema**, in which blood *backs up* in systemic capillaries (**systemic congestion**)

- \_\_\_\_\_ in legs and feet
- Peripheral edema exacerbated by kidneys *retain* excess fluid
- **Treatment** – increase cardiac *output*
  - **Lifestyle modifications** -weight loss and mild exercise, dietary sodium and fluid restrictions
  - **Drug therapy**
  - **Heart transplant** and/or **pacemaker**

**Cardiovascular System II: The Blood Vessels**  
**Chapter 18**

**Vasculature** = \_\_\_\_\_ 60,000 miles of vessels  
Capillaries alone would circle the world (25,000 miles)

→ **Module 18.1 Overview of Arteries and Veins**

INTRODUCTION TO THE VASCULATURE

• **Blood vessels**

- Transport blood to *tissues* (gases, nutrients, and wastes are exchanged) and back \_\_\_\_\_ to *heart*

- \_\_\_\_\_ to tissues

- \_\_\_\_\_

- Secrete a variety of *chemicals*

- \_\_\_\_\_ – transports blood between heart (RV) and \_\_\_\_\_

- **Systemic circuit** – transports blood between heart (LV) and \_\_\_\_\_

- **Coronary circuit**: circulation of blood to \_\_\_\_\_  
(coronary arteries & veins)

• **3 types of vessels**

1. **Arteries**

– *distribution system* of vasculature

-

2. **Capillaries**

– *exchange system* of vasculature

- smallest vessels

-

3. **Veins**

- *collection system* of vasculature

-



- **3 basic layers or tunics** of vessel wall:
  - **Tunica intima**
    - innermost layer
    - \_\_\_\_\_
  - **Tunica media**
    - middle layer
    - \_\_\_\_\_ (VC and VD) and elastic fibers
  - **Tunica externa (adventitia)**
    - \_\_\_\_\_
    - **Vaso vasorum**

STRUCTURE AND FUNCTION OF ARTERIES AND VEINS

- Artery vs vein:
  - Arteries
    - \_\_\_\_\_ → reflects arteries' role in controlling *BP* and *blood flow*
    - more extensive internal and external elastic → reflects arteries are under much higher press.
- 3 classes of arteries
  - 1. \_\_\_\_\_ **(conducting) arteries**
    - aorta and immediate branches
    - highest pressure
  - 2. \_\_\_\_\_ **(distributing) arteries**
    - well dev. tunica media of SMC
    - smaller diameter (named branches to organs)
  - 3. \_\_\_\_\_
    - smallest diameter
    - thin tunica media ( 1-3 layers of SMC)

- Arterioles
  - \_\_\_\_\_ = smallest arterioles that directly feed capillary beds
  - precapillary sphincter SMC that encircles metarteriole-capillary junc.

Certain arteries monitor pressure and chemicals:

*Baroreceptors* –

*Chemoreceptors* –

- Veins
  - outnumber arteries
  - larger lumens
  - serve \_\_\_\_\_ (70% of *total blood* located in veins (systemic & pulmonary veins))
  - 
  - fewer elastic fibers
  - less SMC
- **Veins** classified by *size*:
  - **Venules** – smallest veins; *drain* blood from capillary beds
    - 3 tunics become more *distinct* as venules *merge* → larger venules → veins
    - thin tunica media
    - \_\_\_\_\_ prevent backflow of blood



## Atherosclerosis

- **Atherosclerosis** – leading cause of death in developed world; characterized by formation of **atherosclerotic plaques** (buildups of lipids, cholesterol, calcium salts, and cellular debris within arterial tunica intima)
- Plaques tend to form at branching points where blood undergoes sudden changes in *velocity* and *direction*
- Plaques form due to endothelial injury
- Vessel wall becomes inflamed, which attracts **phagocytes** to “clean up” area → damage to blood vessel → plaque formation
- SMC proliferation → secrete ECM
- Clot may form → MI or stroke
- 10% of world pop. may have Atherosclerosis
- 

**Treatment:**

### → **Module 18.2 Physiology of Blood Flow**

#### INTRODUCTION TO HEMODYNAMICS

**Hemodynamics** – physiology of *blood flow*

- Heart provides *force* that drives blood through blood vessels by creating a *pressure gradient*  
(ex. of **Gradients Core Principle**)

- Pressure is *highest near*

- Blood flows *down* pressure gradient from area of higher P (near heart) to area of lower P (in peripheral vasculature)

- **Blood pressure** (mmHg) – *outward* force that blood exerts on walls of blood vessels
  - *Varies*
    - \_\_\_\_\_ in large systemic arteries
    - and
    - \_\_\_\_\_ in large systemic veins

**Blood flow** (vol. of blood/min) determined by:

- 1. **Magnitude of** \_\_\_\_\_
  - Generally, blood flow *matches* C.O. (avg. ~ 5–6 L/min)
  - Blood flow *directly proportional* to pressure gradient, (blood flow increases when pressure gradient incr.)
- 2. \_\_\_\_\_ (**R**) = any impedance to blood flow
  - Blood flow inversely proportional to R
- 3. \_\_\_\_\_ related to X-sec. area
  - incr. branching → incr. total x-sec. area
  - fastest in aorta, slowest in capillaries

#### FACTORS THAT DETERMINE BLOOD PRESSURE

- BP influenced by 3 main factors:
  1. \_\_\_\_\_ (PR)
    - any factor that *hinders* blood flow
    - PR is greatest further away from heart
    - as PR increases, BP increases
    - vessel radius, viscosity, vessel length
  2. \_\_\_\_\_ = SV x HR
  3. \_\_\_\_\_ – influenced by water loss and gain

#### BP IN DIFFERENT PORTIONS OF CIRCULATION

- Pulmonary circuit ~ 15 mmHg
  - Systemic circuit ~ 95 mm Hg (Fig. 18.5, 18.6; Table 18.2)
    - \_\_\_\_\_ **pressure** averages ~ 120 mm Hg
    - \_\_\_\_\_ **pressure** averages ~80 mm Hg (at rest)
- Pulse pressure** = systolic - diastolic pressures  
= ~ 40 mm Hg
- MAP** = diastolic pressure + 1/3 (pulse pressure)

- Increase venous return:
  - \_\_\_\_\_ prevent backward flow
  - \_\_\_\_\_ in vein walls VC by SNS
  - \_\_\_\_\_
  - **Respiratory pump** (difference in P between abdominal & thoracic cavity)



### Varicose Veins

- Varicose veins
  - characterized by *dilated, bulging, hardened* veins
  - located in superficial veins of lower limb
- Hemorrhoids

High pressure in abdominopelvic cavity during defecation or childbirth decreases return of venous blood from anal veins; also superficial and not well supported by surrounding tissues, and thus may *weaken* and *dilate* because of high pressure

### → Module 18.3 Maintenance of Blood Pressure

#### SHORT-TERM MAINTENANCE OF BP

- **Neural and Hormonal Control**

1. \_\_\_\_\_  
    **SNS** → \_\_\_\_\_ → VC → \_\_\_ BP  
  
    **PSN** → \_\_\_\_\_ → decr. C. O. → \_\_\_ BP  
    (CN X → SA node, AV node)

#### **Baroreceptor reflex:**

\_\_\_\_\_ →  
→ via CN IX to medulla oblongata

→ PSN response = decr. BP  
or SNS response = incr. BP

– **Valsalva maneuver**

- Subject bears down and tries to expire against a closed glottis (airway in larynx), as occurs during coughing, sneezing, defecation, and heavy lifting
- Raises pressure in thoracic cavity and reduces return of venous blood to heart
- → drop in *BP*; should trigger **baroreceptor reflex and generate increased HR**

– **Effects of chemoreceptor stimulation:**

- **Peripheral chemoreceptors** play a role in reg. *breathing*, but also affect BP; receptors respond to \_\_\_\_\_
- **Central chemoreceptors** respond to decreases \_\_\_\_\_; triggers another feedback loop that indirectly increases SNS; → VC and \_\_\_ BP

▪ \_\_\_\_\_ **responses** are much *slower*

1. Hormones that control \_\_\_\_\_  
**Epi, NEpi, thyroid hormone**
2. Hormones that control \_\_\_\_\_
  - Adrenal medulla → Epi, NEpi → VC
  - Atria → ANP → VD
  - Angiotensin II → VC
3. Hormones that reg. \_\_\_\_\_  
Kidneys → Renin → Angiotensin II → aldosterone → conserve H<sub>2</sub>O  
→ ADH → conserve H<sub>2</sub>O

DISORDERS OF BLOOD PRESSURE

- \_\_\_\_\_
  - **Essential (primary) hypertension** – cause is unknown
  - **Secondary hypertension** – cause can be determined
- **Hypotension** – systolic pressure < 90 mm Hg and/or diastolic pressure < 60 mm Hg

- **Circulatory shock** = severe hypotension
  - due to **hypovolemia**

→ **Module 18.4 Capillaries and Tissue Perfusion**

CAPILLARY STRUCTURE AND FUNCTION

Capillary Exchange via:

1. Diffusion & osmosis
  2. Diffusion
  3. Transcytosis
- **Types of capillaries** –
    - \_\_\_\_\_ – skin, nervous, CT, muscle
      - Most capillaries
    - **Fenestrated capillaries** – kidneys, endocrine, S.I.
    - \_\_\_\_\_ – liver, lymphoid

BLOOD FLOW THROUGH CAPILLARY BEDS

When precapillary sphincters are open:

When precapillary sphincters are closed:

LOCAL REGULATION OF TISSUE PERFUSION

- **Autoregulation** (self-regulation)
  - ensures that correct amount of blood is delivered to match a tissue's *level of activity*
- \_\_\_\_\_ ~ 25% of body's capillary beds are fully open

→ **Module 18.5 Capillary Pressures and Water Movement**

PRESSURES AT WORK IN A CAPILLARY

\_\_\_\_\_ drives movement of water across cap. wall (passive process)

- **Pressures at work across capillary bed:**
  - \_\_\_\_\_ (**HP**) moves water out of cap.
    - **35 mmHg** (arterial end) → **15 mmHg** (venule end)
  - \_\_\_\_\_ (**OP**) draws fluid into cap.
    - **25 mmHg** throughout cap. bed
- **Hydrostatic pressure** –
- \_\_\_\_\_
  - Solute particles in a solution exert a force, or “pull,” on water molecules called **osmotic pressure (OP)**
  - Osmotic pressure is determined by \_\_\_\_\_
- \_\_\_\_\_
  - OP of capillary blood = 25 mmHg
    - *Plasma proteins pull fluid into cap.*
  - OP of interstitial fluid = 3 mmHg
    - *Proteins in interstium pull fluid out of cap.*
  - \_\_\_\_\_ (**COP**) =  
 $25 - 3 = 22\text{mmHg}$
- **Capillary net filtration pressure (NFP)**
  - colloid OP and HP gradient drive water in *opposite* directions
  - \_\_\_\_\_ (**NFP**)  
**HP – COP = NFP**
  - At arteriolar end:
    - **35 mm Hg – 22 mmHg = \_\_\_\_\_** (out of cap.)
  - At venule end:
    - **15 mmHg – 22 mmHg = \_\_\_\_\_** (into cap.)
- NFP is not exactly even at 2 ends of cap. bed
  - overall NFP favors filtration of water *out* of capillary
- Excess fluid in interstitium returned to blood \_\_\_\_\_
- **Edema =**



Causes:

- increase in *CHP gradient* due to HT
- decrease in *COP* due to liver disease, cancer, or starvation
- **Peripheral edema** - in hands and feet due to *gravity*
- Ascites – accumulation of interstitial fluid in *abdomen*

### → **Module 18.6 Anatomy of the Systemic Arteries**

#### ANATOMY OF THE SYSTEMIC ARTERIES

**Aorta** (4 sections)

**1. Ascending aorta**

- Rt & Lt coronary arteries

**2. Aortic arch**

- 
- 
- 

**3. Descending thoracic aorta**

**4. Descending abdominal aorta**

- Rt and Lt common iliac A.



#### **Cerebrovascular Accident**

- Cerebrovascular accident (CVA), or stroke
  - damage to brain caused by a *disruption* to blood flow
  - 4<sup>th</sup> most common cause of death (US)
- Causes
  - (1) *blockage* of cerebral arteries due to a clot
  - (2) *loss* of blood (hemorrhage) due to ruptured cerebral artery
- Symptoms
  - sudden-onset paralysis (paresis or weakness)
  - loss of vision,
  - difficulty speaking or understanding speech
  - Headache
- **Risk factors**

- 
- 
- 
- 
- 

- **Treatment**

- medications to dissolve clot and thin blood
- surgery to repair damaged vessels

PULSE POINTS

- **Pulse** = Pressure changes cause arteries to *expand* and *recoil* with each heartbeat
  - 
  - **Pulse points**

→ **Module 18.7 Anatomy of the Systemic Veins**

INTRODUCTION TO THE SYSTEMIC VEINS

Systemic veins carry \_\_\_\_\_

Superior to diaphragm:

Rt and Lt **brachiocephalic veins** merge to form \_\_\_\_\_ → RA

Blood draining *lower limbs* and *pelvis*: → **external and internal iliac veins** merge to form **common iliac veins** → merge to form \_\_\_\_\_ → RA

VEINS OF THE HEAD AND NECK

Head and neck:

- internal jugular veins
- \_\_\_\_\_
- external jugular veins

VEINS OF THE THORAX AND ABDOMEN

- Hepatic portal circulation:
    - Drains nutrient- rich, oxygen-poor blood from digestive organs
    - Superior and inferior mesenteric veins
      - \_\_\_\_\_
- Liver then detoxifies substances including drugs
- blood then goes to IVC

**Blood:**  
**Chapter 19**

Blood = 5 L. of fluid CT, 8% TBW  
comprised of \_\_\_\_\_

→ **Module 19.1 Overview of Blood**

BLOOD OVERVIEW

- Plasma – \_\_\_\_\_ ECM of blood
- Formed elements - \_\_\_\_\_ suspended in plasma
  - \_\_\_\_\_ – also known as red blood cells (RBCs)
  - \_\_\_\_\_ – also known as white blood cells (WBCs)
  - \_\_\_\_\_ – small cellular *fragments* (thrombocytes)
- **Centrifuged** blood sample
  - Top layer – **plasma**
  - Middle layer – leukocytes and platelets (**buffy coat**)
  - Bottom layer – **erythrocytes**
    - **hematocrit** =

OVERVIEW OF BLOOD FUNCTIONS

Functions:

- Exchanging gases – O<sub>2</sub> and CO<sub>2</sub>
- \_\_\_\_\_ – transports *ions, nutrients, hormones, and wastes*, and regulating [ions]
- Immune functions – both *leukocytes* and immune system *proteins* are transported in blood
- \_\_\_\_\_

- \_\_\_\_\_ – platelets
- Acid-Base balance: 7.35 – 7.45 pH
- BP: determined by blood vol.

PLASMA

- Plasma
  - Pale yellow liquid
  - 90% *water*, determining viscosity
  - \_\_\_\_\_ (9% of plasma vol.)
    - Albumins (COP)
    - Immune & Transport (Gamma globulins, lipoproteins)
    - Clotting (Fibrinogen)
  - Other Solutes: glucose, a.a., gases, wastes



### Cirrhosis

- *Liver disease* (cirrhosis) has many causes, including cancer, alcoholism, and viral hepatitis
- Common in US; 10th leading cause of death for men; 12th for women
- Results in progressive decrease in *production of plasma proteins*; leads to decreased \_\_\_\_\_; results in fluid loss to extracellular spaces, producing *severe edema* in the abdomen; termed \_\_\_\_\_
- Decline in \_\_\_\_\_ levels also causes *easy bruising* and *delays clotting*; may be fatal

→ **Module 19.2 Erythrocytes and Oxygen Transport**

ERYTHROCYTE STRUCTURE

Erythrocyte, or red blood cell (RBC)

- \_\_\_\_\_
- anucleated, more space for O<sub>2</sub>-binding
- Hemoglobin (Hb)
  - 2 alpha (α) chains and 2 beta (β) chains
  - heme group = \_\_\_\_\_
  - Fe ion in each heme group is *oxidized* when it *binds to oxygen*  
→ \_\_\_\_\_
- **Hemoglobin :**
  - *Releases oxygen* into tissues where oxygen conc. is low
  - Binds to **CO<sub>2</sub>** → \_\_\_\_\_ where oxygen levels low

ERYTHROCYTE LIFESPAN

- Life span of an erythrocyte:
- Hematopoiesis – process in red bone marrow where *formed elements* in blood are produced by hematopoietic stem cells (HSCs)
- Erythropoiesis produces *erythrocytes* from HSCs
- 

ERYTHROPOIESIS

- Regulation of Erythropoiesis
  - \_\_\_\_\_ (EPO) triggers *neg. feedback*
  - maintains hematocrit within normal
  - Stimulus: Blood levels of oxygen fall *below normal*
  - Receptor: *Kidney cells* detect falling oxygen levels
  - Control center: Kidneys produce more *EPO*
  - Effector/Response: RBC production increases

Homeostasis:

ERYTHROCYTE DEATH

- Erythrocyte *destruction*:
  1. Erythrocytes trapped in sinusoids of \_\_\_\_\_
  2. Spleen macrophages digest erythrocytes
  3. Hemoglobin is broken down into *a.a*, *Fe*, and (biliverdin→) *bilirubin*
    - 4a. *Bilirubin* → \_\_\_\_\_
    - 4b. *Fe* and *a.a.* recycled → \_\_\_\_\_

ANEMIA

- Anemia =

Causes: decreased *Hb*, decreased *Hct*, and abnormal *Hb*

Symptoms: *pallor*, *weakness*, *fatigue*, *incr. HR*

Types: Iron-deficiency anemia (decr. *Hb*)

Pernicious anemia (decr. *Hct*)

SCA (abnormal *Hb*)

- **Abnormal hemoglobin**
  - most common ex. **sickle-cell disease (SCD)**
  - Individuals with *single copy* of defective gene have \_\_\_\_\_
  - Individuals with *two defective copies* of gene have **sickle-cell disease**;
  - produce abnormal hemoglobin called **hemoglobin S (HbS)**
- **Abnormal hemoglobin** (continued):
  - When *oxygen levels are low*, RBCs containing HbS change into a sickle shape; leads to *erythrocyte destruction* in small blood vessels and a reduction in circulating erythrocytes

→ **Module 19.3 Leukocytes and Immune Function**

LEUKOCYTES

- Leukocytes or white blood cells (WBCs)
  - larger than erythrocytes
  - nucleated
  - use blood-stream as transportation only

Two *basic categories*:

- \_\_\_\_\_ contain *cytoplasmic granules*
- Agranulocytes \_\_\_\_\_

GRANULOCYTES

- Granulocytes
  - readily distinguished by their unusual nucleus
  - 3 *categories* based on granule color
  - light lilac, dark purple, or red when stained with Me blue or acidic (eosin) dye
  - \_\_\_\_\_ 60-70%
  - Eosinophils <4%
  - Basophils <1%
- **Neutrophils (PMNs)**
  - most numerous leukocyte
  - *light lilac* color
  - *phagocytosis*
  - nucleus composed of \_\_\_\_\_
- **Eosinophils**
  - \_\_\_\_\_
  - appear *red* due to uptake of eosin dye
  - *Phagocytes* that ingest foreign molecules
  - Respond to parasitic infections and *allergic rxn.*
  - Granules contain *enz.* specific to \_\_\_\_\_



- **Basophils** – least numerous leukocyte
  - *S-shaped nucleus* and appear *dark purple* due to methylene blue dye
  - Chemicals in granules \_\_\_\_\_

AGRANULOCYTES

- **Agranulocytes**

- Lymphocytes** 20-25%

- 2<sup>nd</sup> most common leukocyte
    - contain *large, spherical nuclei* and *light blue rim of cytoplasm*
  - **B lymphocytes (B cells)**
    -
  - **T lymphocytes (T cells)**
    -

- Monocytes** 3-8%

- *largest* leukocyte
    - *large U-shaped nuclei*
    - Some mature into \_\_\_\_\_
    - **Macrophages** – *phagocytic* cells that ingest dead and dying cells, bacteria, antigens, and other cellular debris



### Complete Blood Count

- **Complete Blood Count (CBC)** – important test for *anemia* and other conditions
- Blood sample is drawn and examined under the *microscope* and by an *automated analyzer* to evaluate number and characteristics of blood cells:
  - 
  - 
  - RBC characteristics – size, volume, and concentration of hemoglobin in cytosol

- Platelet count and volume
- Numbers and types of leukocytes

#### LEUKOPOIESIS

- **Leukopoiesis** – formation of WBCs from \_\_\_\_\_ (**HSCs**):
  - **Myeloid cell line** – produces most formed elements (RBCs, monocytes, and platelets)
  - **Lymphoid cell line** – produces **lymphoblasts**, committed to becoming B and T lymphocytes
  - 
  -



#### Leukemia

- Leukemias are *cancers of blood cells or bone marrow*;
- Also classified by *cell line* from which abnormal cells derive:
  - Lymphocytic – from *lymphoid* cell line; generally *abnormal B lymphocytes*
  - Myelogenous – from *myeloid* cell line; can involve any of myeloid cells

#### → Module 19.4 Platelets

#### PLATELETS

- **Platelets**
  - *small cell fragments* of megakaryocyte
  - involved in \_\_\_\_\_ (*stops blood loss* from an injured blood vessel)
  - several types of **granules**: contain clotting factors, enzymes
  - Lifespan:
  -

→ **Module 19.5 Hemostasis**

HEMOSTASIS

- **Hemostasis** - forms **blood clot** to plug broken vessel
  - to *limit significant blood loss*
  - Part 1: **Vascular Spasm**
  - Part 2: **Platelet Plug Formation**
  - Part 3: **Coagulation** (Intrinsic and Extrinsic Pathway)
  - Part 4: **Clot Retraction**
  - Part 5: **Thrombolysis**

HEMOSTASIS – VASCULAR SPASM

- **Hemostasis Part 1: Vascular Spasm** begins immediately when a *blood vessel is injured* and blood leaks into ECF with following two responses:
  - \_\_\_\_\_ and increased *tissue pressure* both act to decrease blood vessel diameter
  - Blood loss is minimized as both *BP* and *blood flow* are reduced locally by these responses

HEMOSTASIS – PLATELET PLUG

HEMOSTASIS – COAGULATION

CONCEPT BOOST: Making Sense of the Coagulation Cascade

- What's the best way to approach the coagulation cascade? Remember that the entire process has three simple goals:
  - Produce factor Xa – goal of both intrinsic and extrinsic pathways, activates prothrombin

- Produce thrombin – produces enzyme thrombin
- Produce fibrin – thrombin, in turn, accomplishes third goal of coagulation: producing fibrin to hold platelet plug together and seal wound

#### HEMOSTASIS – CLOT RETRACTION

#### HEMOSTASIS – THROMBOLYSIS

#### REGULATION OF CLOTTING

- Blood clotting is produced by a \_\_\_\_\_; example of Feedback Loops Core Principle; must be tightly regulated to prevent mishaps
  - Endothelial cells → two chemicals that regulate 1st and 2<sup>nd</sup> stages of clot formation
    - Prostacyclin – prostaglandin; inhibits platelet aggregation
    - Nitric oxide – causes vasodilation
  - Endothelial cells and hepatocytes produce anticoagulants; inhibit coagulation:
    - Antithrombin III (AT-III) – protein that binds and inhibits activity of both factor Xa and thrombin; also prevents activation of new thrombin
    - Heparin sulfate – polysaccharide that enhances antithrombin activity
    - Protein C – when activated by protein S, catalyzes reactions that degrade clotting factors Va and VIIIa

#### DISORDERS OF CLOTTING

- **Clotting** Disorders
  1. Bleeding disorders:
    - Hemophilias –
  2. Hypercoagulable conditions:

---

DVT (deep vein thrombosis) → PE pulmonary embolism



## Anticlot Medications

- Patients with thrombi or emboli are treated with drugs that *prevent* clotting process
- Anticoagulants – widely used group of medications; manage and prevent emboli; include:
  - Heparin
  - Warfarin (Coumadin)
- Antiplatelet drugs:
  - Aspirin –
  - Clopidogrel –
- Thrombolytic agents (tPA or urokinase)
- 

### → **Module 19.6 Blood Typing and Matching**

#### BLOOD TRANSFUSIONS

- Blood transfusions
  - blood taken from a donor is given to a recipient
  - Discovery of \_\_\_\_\_ (surface marker) found on all cells, including RBCs; genetically determined CHO chain
  - Antigens on erythrocytes (*genetically determined* carbohydrate chains) give rise to different blood groups
  - Two groups of the 30 different antigens found on erythrocytes are particularly useful for clinical use: \_\_\_\_\_ blood group and \_\_\_\_\_ blood group
  -

### BLOOD TYPING

ABO blood group features two antigens, A and B antigens; gives rise to four ABO types:

- Type A – only \_\_\_\_\_ is present on RBC
- Type B – only \_\_\_\_\_ is present
- Type AB – both A and B antigens are present
- Type O – neither \_\_\_\_\_ antigens are present
- **Rh blood group**
- **Rh antigen** first discovered in rhesus monkeys; individuals with Rh antigen (**D antigen**)
- **Rh-positive (Rh+)** \_\_\_\_\_
- **Rh-negative (Rh-)** \_\_\_\_\_
- Type **O+** is most common blood type in U.S. populations while **AB-** is least common
- Blood typing in the lab uses **antibodies (agglutinins)** that *bind to antigens* on RBCs
- Causes them to *clump together* or \_\_\_\_\_
- Ultimately, agglutination promotes \_\_\_\_\_
- 



### BLOOD TRANSFUSIONS

- Note that *anti-A* and *anti-B antibodies* are *pre-formed*; they are present in plasma even if individual has never been *exposed to those antigens*
- *Anti-Rh antibodies*, however, are produced only if a person \_\_\_\_\_  
\_\_\_\_\_
- Therefore, an Rh- individual generally has no *anti-Rh antibodies* unless he or she has been *exposed* (sensitized) to *Rh+ erythrocytes*
- Antigens and antibodies are basis for **blood matching**; blood taken from a donor is *screened for compatibility* prior to its administration to a recipient
  - A **match** occurs if *donor blood type* is compatible with *recipient blood type*

- **Transfusion reaction** – recipient antibodies bind to donor antigens; causes agglutination that *destroys donor erythrocytes*, possibly leading to **kidney failure** and death



### Hemolytic Disease of the Newborn (HDN)

- Also known as \_\_\_\_\_; occurs when an *Rh-* mother gives birth to an *Rh+* fetus
- During birth *fetal RBCs enter mother's blood*; stimulates her immune system to produce *anti-Rh antibodies*
- First pregnancy is not typically at risk; in subsequent pregnancies maternal anti-Rh antibodies can cross placenta and *hemolyze Rh+ fetal RBCs*
- Effectively prevented with *blood type screening*; if woman is Rh-, can be given Rh<sub>0</sub> (D) immune globulin; contains anti-Rh antibodies that *bind fetal cells in maternal circulation*; prevents maternal production of anti-Rh antibodies
- Universal donor – Blood type \_\_\_\_\_ 
  - 
  - Can be given to *any other blood type* in an *emergency* when blood matching is not an option
- Universal recipient – blood type \_\_\_\_\_ 
  - These individuals *do not make antibodies* to A, B, or Rh antigens
  - Individuals with AB+ blood type can generally *receive blood from any blood type donors*
  - Matching is still safest practice

**The Lymphatic System and Immunity  
Chapter 20**

Immune System =

Lymphatic System works with immune system

→ **Module 20.1 Structure and Function of the Lymphatic System**

INTRODUCTION TO THE IMMUNE AND LYMPHATIC SYSTEMS

- Lymphatic system
  - group of organs and tissues that work with immune system
  - functions \_\_\_\_\_
  - 2 main components:
    - Lymphatic vessels: blind-ended tubes
    - Lymphatic tissue and organs: tonsils, lymph nodes, \_\_\_\_\_

FUNCTIONS OF THE LYMPHATIC SYSTEM

- Lymphatic system functions:
  1. Regulation of \_\_\_\_\_
    - return excess fluid lost from plasma to CV system
  2. Absorption of \_\_\_\_\_
    - breakdown products of fats in diet are too *large* to pass into blood cap.  
(absorbed into \_\_\_\_\_)
  3. Immune functions
    - filter pathogens from lymph and blood



LYMPHATIC VESSELS AND LYMPH CIRCULATION

- Lymph-collecting vessels

→ lymph trunks → cisterna chyli

2 lymph ducts

Right lymphatic duct

Thoracic duct

Right Subclavian Vein

Left Subclavian Vein

Lymphatic vessels

- **low-pressure** circuit because no main pump to drive lymph through vessels, and most of them are transporting lymph against gravity
- **Valves** \_\_\_\_\_



### Lymphedema

- \_\_\_\_\_ (swelling) is an accumulation of excess interstitial fluid; many conditions can cause mild to moderate edema, including trauma, vascular disease, and heart failure
- However, edema seen with lymphedema is typically severe and can be disfiguring
- Lymphedema is generally due to *removal* of lymphatic vessels during surgery or *blockage* of vessels from pathogens such as parasites
- Both conditions prevent lymphatic vessels from transporting excess interstitial fluid back to cardiovascular system; fluid therefore *accumulates* in tissues of affected body part, causing it to enlarge
- Photo shows a case of lymphedema in arm of a breast cancer patient resulting from surgical removal of lymph nodes

LYMPHOID TISSUES AND ORGANS

- Mucosa- Associated Lymphatic Tissue (MALT)
  - Tonsils (palatine, pharyngeal, lingual)
  - Peyer's patches (aggregated lymphoid nodules)
  - Appendix
- Lymph nodes
- Spleen

