N3: ANATOMY AND PHYSIOLOGY
1st Semester, Academic Year 2023-2024

LECTURE: CARDIOVASCULAR SYSTEM: THE HEART
STUDY GUIDE

Introduction
The cardiovascular system or the circulatory system consists of the heart, which is a muscular pumping device and a closed system of vessels with arteries, veins, and capillaries. Blood, which was discussed in the previous module, is pumped by the heart around a closed circle or circuit of vessels again and again throughout the body supplying cells with oxygen and nutrients, carrying wastes from cells and transports important hormones, enzymes, cellular elements of the blood and other chemicals.

The vital role of the cardiovascular system in maintaining homeostasis depends on the continuous and controlled movement of blood through the network of capillaries that reach every cell in the body.

Numerous control mechanisms help to regulate and integrate the diverse functions and component parts of the cardiovascular system in order to supply blood to specific body areas according to need. These mechanisms ensure a constant internal environment surrounding each body cell regardless of differing demands for nutrients or production of waste products.

I. Learning Outcomes:
At the end of the session, you should be able to:

1. Describe the anatomical features of the heart and its relevance to the functions of the heart.
2. Describe systemic and pulmonary circulations
3. Describe the structural and functional characteristics of cardiac muscle tissue and the cardiac conduction system
4. Describe the basic ECG waves and their correlation with atrial and ventricular systole
5. Describe the pressure and volume changes during a cardiac cycle
6. Define cardiac output and the factors which affect it

II. Activities

ACTIVITY	DESCRIPTION
1. Synchronous lecture	Powerpoint slides and video presentation
2. Supplementary Activities	Submission in discussion forum in VLE

III. References:
Tortora, & Derrickson. (2012). Chapter 20 The Cardiovascular System: The Heart. In *Principles of anatomy and physiology* (13th ed.) or its equivalent in other editions.

IV. General Instructions
- Read Chapters 20 of the reference book. Use the study guide provided.
- After reading, you can watch the slide presentation provided for this module. The link is posted separately.

- There are other links to videos which are optional viewing.

- There are activities provided in this guide to further facilitate learning. There will be a forum assigned to each of the activities. This is where you will make your submissions.

V. Topic and Study Guide

THE HEART

A. Anatomy of the Heart

1. Location
    - Lies in the mediastinum which is the anatomical region from the sternum to the vertebral column, from the first rib to the diaphragm, and between the lungs
    - What are the structures in the boundaries of the heart?
    - Identify the apex and the base of the heart.

2. Structures of the heart

a. Pericardium
    1) Fibrous pericardium
    2) Serous pericardium- made up of parietal and visceral pericardium
        - Pericardial cavity and pericardial fluid

b. Layers of the heart wall- describe its histology, features and functions
    1) Epicardium
    2) Myocardium
    3) Endocardium

c. Chambers of the heart- know the blood vessels connected to each chamber and the valves connected to each chamber
    1) Right atrium
        - Tricuspid valve or right atrioventricular valve
        - Presence of interatrial septum with fossa ovalis
        - Superior vena cava, inferior vena cava, coronary sinus→ bring deoxygenated blood to right atrium→right ventricle
    2) Right ventricle
        - Connections to the pulmonary valve→ pulmonary trunk→ left and right pulmonary artery→left and right lungs
    3) Left atrium
        - Lungs→bring oxygenated blood through 4 pulmonary veins→left atrium→ left ventricle →systemic circulation
        - Bicuspid (mitral) valve OR left atrioventricular valve
    4) Left ventricle
        - Thickest chamber of the heart
        - Forms apex of the heart
        - Left ventricle→through aortic valve→ascending aorta (coronary arteries branch out from this)→arch of the aorta→descending aorta
        - Ligamentum arteriosum (remnant of ductus arteriosus)

- Physiologic correlations with anatomy of the heart
    - The thickness of the myocardium of the four chambers varies according to each chamber's function.
    - Walls of ventricles thicker than atria because atria deliver blood under less pressure into the adjacent ventricles.

- Left ventricular wall is thicker than right ventricular wall right side has a much smaller workload. It pumps blood a short distance to the lungs at lower pressure, and the resistance to blood flow is small. The left ventricle pumps blood great distances to all other parts of the body at higher pressure, and the resistance to blood flow is larger.

d. Heart valves- The structure and anatomical features of the heart valves allow flow from atria to ventricles and prevent backflow of blood into atria (for AV valves) and allow ejection of blood into arteries and prevent backflow into ventricles (for semilunar valves) so that diseases which damage the valves will result in hemodynamic compromises

1) Atrioventricular valves
   a) Bicuspid OR mitral OR left atrioventricular valve
   b) Tricuspid OR right atrioventricular valve
2) Semilunar valves
   a) Aortic valve
   b) Pulmonary valve

Additional resources:
* Refer to Figure 20.1 (page 759, 13th edition Tortora): Position of the heart and associated structures in the mediastinum

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=R1kfeURl9Rk&t=59s
The Circulatory system part 1: The Heart by Professor Dave Explains

B. Circulation of blood

a. Describe and differentiate systemic from pulmonary circulation
Refer to Figure 20.7 Systemic and pulmonary circulation (p. 769, Tortora et al, 13th ed)

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=NDk8fmIl9V8
Pulmonary and Systemic Circulations by Educational channel

b. Coronary circulation

Coronary arteries

- The myocardium has its own network of blood vessels, the **coronary** or **cardiac circulation**. The **left and right coronary arteries** branch from the ascending aorta and encircle the heart like a crown encircles the head. While the heart is contracting, little blood flows in the coronary arteries because they are squeezed shut. When the heart relaxes, however, the high pressure of blood in the aorta propels blood through the coronary arteries, into capillaries, and then into **coronary veins**.
- Most parts of the body receive blood from branches of more than one artery, and where two or more arteries supply the same region, they usually connect. These connections, called **anastomoses**, provide alternate routes, called **collateral circulation**, for blood to reach a particular organ or tissue.
- The myocardium contains many anastomoses. They provide detours for arterial blood if a main route becomes obstructed. Thus, heart muscle may receive sufficient oxygen even if one of its coronary arteries is partially blocked.

Coronary Veins

- Most of the deoxygenated blood from the myocardium drains into a large vascular sinus in the coronary sulcus on the posterior surface of the heart, called the **coronary sinus**. (A vascular sinus is a thin-walled vein that has no smooth muscle to alter its diameter.) The deoxygenated blood in the **coronary sinus empties into the right atrium**.

C. Cardiac muscle tissue and the Cardiac conduction system

1. Histology of cardiac muscle tissue

a. Characteristics of cardiac muscle tissue

- Shorter with branching
- Usually one cell nucleus
- Intercalated discs: transverse thickenings of the sarcolemma called which connect one cardiac Muscle fiber to other fibers
    - Contain desmosomes and gap junctions
- Gap junctions allow muscle action potentials to conduct from one muscle fiber to its neighbors. Gap junctions allow the entire myocardium of the atria or the ventricles to contract as a single, coordinated unit.
- Mitochondria are more numerous than in skeletal muscles
- Same arrangement of actin and myosin as skeletal muscles
- Transverse tubules of cardiac muscles are wider and less abundant than skeletal muscles
- Sarcoplasmic reticulum are smaller than SR of skeletal muscles so cardiac muscles have smaller reserves of calcium ions

* Refer to Figure 20.9 Histology of the cardiac muscle tissue (p. 773, Tortora, 13th ed)

b. Autorhythmic fibers: The conduction system

- The heart has inherent and rhythmical electrical activity.
- The source of this is a network of specialized cardiac muscle fibers called **autorhythmic fibers.**
- Functions:
    1) Act as **pacemaker-** sets the rhythm of electrical excitation that causes contraction
    2) They form the cardiac conduction system- a network of specialized cardiac muscle fibers that provide a path for each cycle of cardiac excitation to progress through the heart. The conduction system ensures that cardiac chambers become stimulated to contract in a coordinated manner, which makes the heart an effective pump

2. Cardiac action potentials (cardiac AP) and specialized cardiac muscle fibers

a) Cardiac AP begins at the **sinoatrial node (SA node)**

- Located in the right atrium, just below the superior vena cava
- Natural pacemaker
- Unstable resting potential so they repeatedly depolarize to threshold spontaneously
- The spontaneous depolarization is called pacemaker potential.
- Each AP propagates throughout both right and left atria through gap junctions so that both atria contract at the same time.
- Pacing rate is about 60 to 100/minute-faster than the other conducting fibers of the heart

b) Atrioventricular node (AV node)

- Cardiac AP potential reaches AV node, in the interatrial septum
- AP slows down
- Provides time for atria to empty their blood into the ventricles

c) Atrioventricular bundle (bundle of His)

- Only site where AP can conduct from atria to ventricles

d) Right and left bundle branches

- AP conducted through the interventricular septum towards the apex

e) Purkinje fibers

- Rapidly conducts AP from the apex upward to the rest of the ventricular myocardium

* Refer to Figure 20.10 The conduction system (p. 774, Tortora, 13th ed)

3. Action potentials and contraction of contractile fibers

The action potential initiated by the SA node travels along the conduction system and spreads out to excite the "working" atrial and ventricular muscle fibers, called **contractile fibers.** An action potential occurs in a contractile fiber as follows:

a) Depolarization

- Contractile fibers have stable resting membrane potential (about –90 mV)
- When an action potential from a neighboring muscle fiber brings the membrane potential to threshold, **voltage-gated fast Na^+ channels** open→ **rapid depolarization**→ within a few milliseconds, the Na+ channels automatically inactivate

b) Plateau

- Period of maintained depolarization
- Due to opening of **voltage-gated slow Ca^{2+} channels** in the sarcolemma→ calcium ions move from interstitial fluid to cytosol (extracellular fluid to intracellular fluid)→more Ca^{2+} go into cytosol from sarcoplasmic reticulum→triggers contraction
- **Voltage-gated K^+ channels** also open just before plateau phase begins→ K^+ goes out into interstitial space (outflow)
- Therefore, depolarization is sustained during the plateau phase because Ca^{2+} inflow just balances K^+ outflow.

c) Repolarization

- The recovery of the resting membrane potential during the repolarization phase of a cardiac action potential resembles that in other excitable cells.
- After a delay (which is particularly prolonged in cardiac muscle), additional voltage-gated K^+ channels open.
- Outflow of K^+ restores the negative resting membrane potential (-90 mV). At the same time, the calcium channels in the sarcolemma and the sarcoplasmic reticulum are closing, which also contributes to repolarization.

d) Refractory period

- The refractory period of a cardiac muscle fiber lasts longer than the contraction itself. As a result, another contraction cannot begin until relaxation is well under way.
- For this reason, tetanus (maintained contraction) cannot occur in cardiac muscle as it can in skeletal muscle.

- The advantage is apparent if you consider how the ventricles work. Their pumping function depends on alternating contraction (when they eject blood) and relaxation (when they refill). If heart muscle could undergo tetanus, blood flow would cease.
- Mechanism of contraction of the contractile muscle fibers is the same as in skeletal muscles.

* Refer to Figure 20.11 Action potential in a ventricular contractile fiber (p. 775, Tortora, 13th ed)

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=v7Q9BrNfIpQ
Cardiac Action Potential, Animation by Alila Medical Media

4. ATP production in cardiac muscles
   - Sources of ATP:
     - Aerobic cellular respiration (in mitochondria)
     - At rest, ATP comes from fatty acids (60%) and glucose (35%), the rest from lactic acid, amino acids, ketone bodies
     - During exercise, lactic acid source comes from actively contracting skeletal muscles
     - ATP from creatine phosphate through creatine kinase enzyme

5. Electrocardiogram
   - What is an electrocardiogram or ECG?
   - Describe the ECG waves and what these represent.

* Refer to Figure 20.12 Normal ECG (lead II) (p. 777, Tortora, 13th ed)
* Refer to Figure 20.13 Timing and route of actiona potential depolarization and repolarization through the conducting system and myocardium (p. 778, Tortora, 13th ed)

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=RYZ4daFwMa8
Cardiac Conduction System and Understanding ECG, Animation by Alila Medical Media

D. The Cardiac Cycle

1. Cardiac cycle
   - consists of systole and diastole of the atria plus the systole and diastole of the ventricles
   - The term systole refers to the phase of contraction
   - The term diastole refers to the phase of relaxation
   - Phases of cardiac cycle
     a) Atrial contraction
        - SA node firing→atrial depolarization→atrial contraction (atrial systole)→increases in pressure in atria relative to pressure in ventricles→blood goes to ventricles through AV valves
        - The end of atrial systole Coincides with end of ventricular diastole
        - The volume of blood in each ventricle at the end of its relaxation period (diastole) is called the **end-diastolic volume (EDV).**

     b) Isovolumetric contraction
        - During ventricular systole, the atria are in atrial diastole

- Isovolumetric contraction: Ventricular depolarization→ventricular contraction/systole with both AV valves and semilunar valves are closed

c) Ventricular ejection
   - As the ventricles continue to contract, the pressures in the right and left ventricles exceed the pressure in the aorta and pulmonary truck→semilunar valves open→ blood goes to pulmonary and systemic circulation
   - The volume remaining in each ventricle at the end of systole is **end-systolic volume**
   - Period of ventricular systole when the semilunar valves open is **ventricular ejection**
   - **Stroke volume** is the volume ejected per beat from each ventricle
   - **Stroke volume= EDV-ESV**

d) Isovolumetric relaxation
   - Ventricular repolarization→ventricular diastole→pressure within the chambers falls→blood in the aorta and pulmonary trunk begins to flow backward toward the regions of lower pressure in the ventricles→semilunar valves close
   - Isovolumetric relaxation: period after the semilunar valves close, when ventricular blood volume does not change because all four valves are closed.

e) Ventricular filling
   - As ventricles relax→pressure falls below atrial pressure→AV valves open→ventricular filling begins

Phase of cardiac cycle	Steps/Events
Atrial contraction	1. Atrial depolarization
	2. Atrial systole→AV valves open
	3. end of atrial systole coincides with end of ventricular diastole→ contributes final volume of blood to ventricles at end of ventricular diastole (called end-diastolic volume (EDV)
	4. start of ventricular depolarization
Isovolumetric contraction	5. Ventricular depolarization→start of ventricular systole AV and semilunar valves are closed
Ventricular ejection	6. Ventricles continue to contract→when pressures in the ventricles surpass pressure in aorta and pulmonary trunk→semilunar valves open→**ventricular ejection** of blood form the heart
	7. End of ventricular systole, the volume of blood remaining in the ventricles is called **end-systolic volume (ESV)** - **Stroke volume (SV):** volume of blood ejected from each ventricle per beat
	8. Onset of ventricular repolarization (T wave in ECG)
Isovolumetric relaxation	9. Ventricular repolarization→Ventricular diastole
Ventricular filling	10. Ventricular pressure drops below atrial pressure→ventricular filling

* Refer to Figure 20.14 Cardiac cycle (p. 780, Tortora, 13th ed)
* Refer to Figure 20.13 Timing and route of actiona potential depolarization and repolarization through the conducting system and myocardium (p. 778, Tortora, 13th ed)

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=IS9TD9fHFv0
The Cardiac Cycle, Animation by Alila Medical Media

2. Heart sounds
   - What causes the normal heart sounds?

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=-4kGMI-qQ3I
Lub Dub | Circulatory system physiology by Khan Academy

E. Cardiac Output

1. Cardiac output (CO)-volume of blood ejected from the left ventricle or right ventricle into the aorta or pulmonary truck each minute.

CO (ml/min)= stroke volume (SV in ml/beat) x heart rate (HR in beats/min)

SV= 70 ml/beat HR= 60 to 100/min

CO= 70 x 75 = 5250 ml/min

2. Cardiac reserve- difference between maximum cardiac output and cardiac output at rest

3. Regulation of stroke volume
   - A healthy heart will pump out the blood that entered its chambers during the previous diastole. In other words, if more blood returns to the heart during diastole, then more blood is ejected during the next systole.
   - The following factors regulate SV:
     a) Preload- degree of stretch on the heart before it contracts
     b) Contractility- the forcefulness of contraction
     c) Afterload- the pressure that must be exceeded before ejection of blood from ventricles

4. Preload
   - **Frank–Starling law of the heart**: the more the heart fills with blood during diastole, the greater the force of contraction during systole.
   - The preload is proportional to the end-diastolic volume (EDV) (the volume of blood that fills the ventricles at the end of diastole). The greater the EDV, the more forceful the next contraction.
   - Two key factors determine EDV:
     (1) the duration of ventricular diastole
     (2) **venous return,** the volume of blood returning to the right ventricle
   - The Frank–Starling law of the heart equalizes the output of the right and left ventricles and keeps the same volume of blood flowing to both the systemic and pulmonary circulations.

5. Contractility
   - the strength of contraction at any given preload
   - Positive inotrope- substances that increase contractility
     - Examples: Agents that promote Ca^{2+} inflow during cardiac AP
       - Sympathetic nervous system stimulation- epinephrine and norepinephrine
       - Increased calcium in interstitial fluid
       - Drug digitalis

- Negative inotropic agents- substances that decrease contractility
    - Examples: Inhibition of sympathetic division of ANS
        - Anoxia, acidosis, some anesthetics
        - Increased K^+ in the interstitial fluid

6. Afterload
    - the pressure that must be overcome before a semilunar valve can open
    - Increase in afterload→decreased stroke volume
    - Factors that increases afterload: hypertension (increase in blood pressure); narrowing of the arteries by atherosclerosis

7. Regulation of heart rate (HR)
    a. Factors affecting HR
        - Centers in the brain: cardiovascular center in the medulla oblongata- connections with limbic system and cerebral cortex and sensory receptors
        - Sensory receptors that provide input to cardiovascular center in the brain:
            - Proprioceptors
            - Chemoreceptors
            - Baroreceptors (carotid arteries and aorta)-detect changes in blood pressure

        - Autonomic regulation
            - Sympathetic stimulation→NE released→ in SA and AV nodes, increase rate of depolarization of increase in HR
            - Sympathetic stimulation→NE released→contractile fibers in atria and ventricles→enhances Ca^{2+} entry→ increase in contractility
            - Parasympathetic stimulation→ vis vagus nerve (CN X)→ release of Ach in SA, node, AV node and atria→decrease in heart rate; no effect on contractility
        - Chemical regulation of HR
            - Hypoxia—decrease HR
            - Acidosis (low pH)—decrease HR
            - Alkalosis (high pH)—decrease HR
            - Hormone
                - Epinephrine and norepinephrine—increase HR
                - Exercise, stress→release of more hormones
                - Thyroid hormones→increase HR
            - Cations
                - Elevated K^+ or Na^+ →decreased HR and contractility
                - Increase Ca^{2+} →increase heart rate and contractility
            - Other factors
                - Age
                - Gender
                - Physical fitness
                - Body temperature

* Refer to Figure 20.17 Factors that increase cardiac output (p. 785, Tortora, 13th ed)

*You can view this video to further illustrate (optional):

https://www.youtube.com/watch?v=hpQFToprlH8
Cardiovascular System Physiology - Cardiac Output (stroke volume, heart rate, preload and afterload) by Armando Hasudungan