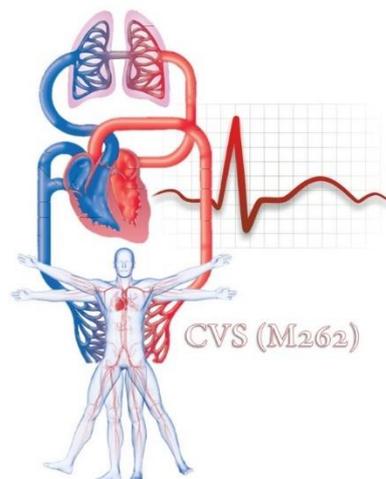




# Cardiovascular System (CVS) Module Integrated System Course (MED 364)

Faculty of Medicine  
Jordan University of Science and Technology

2018-2019  
Course Curriculum



<b>Course title:</b>	Cardiovascular System (CVS) Module (MED 364)
<b>Course code:</b>	MED 364
<b>Credit Hours:</b>	6 Credit Hours
<b>Duration:</b>	6 Calendar weeks
<b>Sequence:</b>	Year 3
<b>Coordinator:</b>	Dr. Khalid Kheirallah <a href="mailto:kakheirallah@just.edu.jo">kakheirallah@just.edu.jo</a>
<b>Clinical Coordinator:</b>	Dr. Alia Al-Mohtaseb <a href="mailto:ahmohtaseb@just.edu.jo">ahmohtaseb@just.edu.jo</a>
<b>Module Secretary:</b>	Mrs Rania Ababneh

## **Course Description**

This is an interdisciplinary integrated module of the cardiovascular system. Basic sciences of anatomy, physiology, biochemistry, pathology, and pharmacology of the cardiovascular system are correlated with a set of clinical disorders of the system. The goal of this integrated course is to provide medical students with comprehensive knowledge about components of the cardiovascular system related to clinical manifestations of diseases. The teaching methods include lectures, laboratories, as well as small group discussions of clinically oriented problems to enhance self-directed learning. This knowledge is supported by skills-developing laboratory activities and clinically oriented activities. Research ideas with specific embedded objectives are also included to emphasize social responsibility, evidence-based medicine, community service, and innovative thinking.

## **Course Learning Outcomes**

Upon completion of this course, students should be able to:

1. Understand the anatomy of mediastinum, heart, and the great vessels and their distribution.
2. Know the microscopic appearance of different parts of the cardiovascular system; their normal embryological development and common congenital abnormalities.
3. Describe and understand the physiological concepts of electrocardiogram, cardiac cycle, hemodynamics, regulation of blood flow and blood pressure, microcirculations, and mechanisms of circulatory shock.
4. Understand the metabolism of cardiac muscles and the value of cardiac enzymes and troponins in the diagnosis of cardiac muscle diseases.
5. Recognize the role and types of lipoprotein disorders and the pathogenic mechanisms behind the formation of atherosclerosis.
6. Be familiar with the most common types of cardiovascular diseases with emphasis on: etiology, mechanisms, morphology, pathological aspects, and clinical manifestations.
7. Understand the mechanisms of action, pharmacokinetics, uses, and adverse effects of commonly used drugs in the treatment of cardiac failure, cardiac arrhythmias, hypertension, angina, and hyperlipidemias.
8. Recognize the major cardiovascular risk factors in health and diseases.
9. Identify the nutritional and dietetic components in the etiology, management, and prevention of cardiovascular diseases.

**Recommended Textbooks and Atlases:**

<b>Subject</b>	<b>Book (Resources)</b>
<b>Anatomy</b>	<ul style="list-style-type: none"><li>• Clinical Anatomy for Medical Students. By Snell, Latest Edition.</li><li>• Grants Atlas of Anatomy or any other Atlas of Human Anatomy, Latest Edition.</li><li>• Basic Histology. By Junqueira, Latest Edition.</li><li>• Before we are born. By Morre and Persaud, Latest Edition.</li></ul>
<b>Physiology</b>	<ul style="list-style-type: none"><li>• Textbook of Medical physiology. By Guyton and Hall, Latest Edition.</li><li>• Human Physiology, from Cells to Systems. By Sherwood, Latest Edition.</li></ul>
<b>Biochemistry</b>	<ul style="list-style-type: none"><li>• Textbook of Biochemistry with Clinical Correlations. By Thomas Devlin, 7<sup>th</sup> edition.</li></ul>
<b>Pathology</b>	<ul style="list-style-type: none"><li>• Basic Pathology. By Kumar, Cotran, and Robbins, Latest Edition.</li></ul>
<b>Pharmacology</b>	<ul style="list-style-type: none"><li>• Lippincott's Illustrated Reviews: Pharmacology, 6<sup>th</sup> edition, 2014.</li></ul>
<b>Community Med.</b>	<ul style="list-style-type: none"><li>• Lecture Handouts.</li></ul>
<b>Clinical Lectures</b>	<ul style="list-style-type: none"><li>• Lecture Handouts.</li></ul>

## Specific Learning Objectives:

After studying the material covered in lectures, practical sessions, clinical seminars, and case discussions of this course, and after using his/her private self-learning time in a productive way, the student is expected to achieve the following specific objectives mentioned in the table for each lecture and lab:

### A-Lectures"

#	Lecture Title	Lecture Objectives
1	Introductory lecture	<ol style="list-style-type: none"><li>1. Understand the general outline of the CVS module (MED 364).</li><li>2. Be familiar with the modalities of teaching throughout the course.</li><li>3. Be familiar with the grading system and passing requirements of the course.</li><li>4. Understand the current status of CV diseases in Jordan.</li></ol>
2	The Mediastinum & heart coverings (Pericardium)  <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"><li>1. Describe divisions of the mediastinum.</li><li>2. Describe the outline and normal position of the heart.</li><li>3. Understand and identify relations of different parts of the heart in the middle mediastinum.</li><li>4. Identify and list various contents and relations of the mediastinum.</li><li>5. Define the pericardium and describe its covering layers.</li><li>6. Describe its attachment to the diaphragm to the pericardium and to the great vessels in the mediastinum.</li></ol>
3	Pericardial spaces and External features of the heart <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"><li>1. Discuss the pericardial space and its recesses.</li><li>2. Describe innervations of the fibrous pericardium.</li><li>3. Describe the external features of the heart.</li><li>4. Identify the surface anatomy of the heart.</li></ol>
4	Heart chambers, valves, conductive system, and innervation of the heart <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"><li>1. Describe divisions of the heart into four chambers and the internal of each chamber.</li><li>2. Identify papillary muscles and describe their locations and importance.</li><li>3. Describe the atrio-ventricular valves and their position and the attachment of the cusps to papillary muscles and their functional importance.</li><li>4. Describe the aortic and pulmonary semilunar valves and their position and functional importance.</li><li>5. Describe different parts of the conductive system of the heart and its distribution and function within the myocardium.</li></ol>
5	Histology of the heart and blood vessels <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"><li>1. Describe the microscopic structure of the cardiac muscle.</li><li>2. Describe the ultra structural appearance of the of cardiac muscle fiber.</li><li>3. Describe the histological features of endocardium and epicardium.</li><li>4. Describe the histological appearance of arteries and veins and their differences.</li><li>5. List the different types of capillaries and their distribution.</li></ol>
6	Organization of the CVS <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"><li>1. Describe the systemic and pulmonary circulations and their differences.</li><li>2. Explain the functional parts of the CVS.</li><li>3. Discuss blood velocity, volumes, and pressures in different parts of the CVS.</li><li>4. Discuss the basic functions of the CVS.</li></ol>

7	Physiology of cardiac muscle <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Describes the cardiac conductive system and its function.</li> <li>2. Describe the cardiac muscle action potential and its components.</li> <li>3. Define the refractory period and the excitation-contraction coupling.</li> </ol>
8	Cardiac cycle <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Define the systolic and diastolic contraction "durations".</li> <li>2. Discuss the changes of pressure and volumes in left ventricle, left atrium, and the aorta during cardiac cycle.</li> <li>3. Identify the periods of cardiac cycle, i.e. atrial systole, isovolumetric contraction, ejection, and isovolumetric relaxation.</li> <li>4. Discuss the volume-pressure relationship in the left ventricle.</li> </ol>
9	Pumping of the heart and regulation of cardiac output <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Discuss the intrinsic and extrinsic control of cardiac output.</li> <li>2. Explain the Frank-Starling mechanism.</li> <li>3. Describe the effect of high K<sup>+</sup> and Ca<sup>++</sup> on heart function.</li> <li>4. Define cardiac index.</li> <li>5. Discuss the importance of venous return and cardiac reserve and their effects on cardiac output.</li> <li>6. Explain the effect of increased sympathetic activity and blood volume on cardiac output.</li> <li>7. Study the methods for measurement of cardiac output.</li> </ol>
10	Endocarditis, valvular diseases, and myocarditis <b><u>(Pathology)</u></b>	<ol style="list-style-type: none"> <li>1. Know the different types of endocarditis.</li> <li>2. Classify infective endocarditis.</li> <li>3. Discuss the pathogenesis and list organisms causing endocarditis.</li> <li>4. Compare and contrast acute &amp; subacute bacterial endocarditis.</li> <li>5. List the possible complications of bacterial endocarditis.</li> <li>6. Describe briefly Marantic &amp; Libman-sack endocarditis.</li> <li>7. Classify and understand the pathological consequences of the various valvular heart diseases</li> <li>8. List the major etiological factors and clinical presentations of myocarditis.</li> </ol>
11	ECG <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Identify waves of ECG and the cause of each.</li> <li>2. Define the normal intervals and segments of ECG.</li> <li>3. Discuss the bipolar and unipolar limb and chest leads.</li> <li>4. Discuss the bipolar limb lead and the cardiac axis.</li> </ol>
12	Coronary circulation & venous drainage of the heart <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"> <li>1. Describe the origin of left and right coronary arteries and their course, branches, and distribution.</li> <li>2. Describe sites of anastomosis between branches of coronary arteries.</li> <li>3. Describe venous drainage of the heart and cardiac veins (their names, location, and drainage areas).</li> <li>4. Describe the location and termination of the coronary sinus and its tributaries.</li> </ol>
13	Blood vessels I- Arterial system <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"> <li>1. List the parts of aorta.</li> <li>2. List the branches of arch of aorta.</li> <li>3. Describe the blood supply of the head and neck.</li> <li>4. Describe the blood supply of upper limb.</li> <li>5. Describe the branches of thoracic aorta.</li> <li>6. Describe the branches of the abdominal aorta.</li> <li>7. Describe the blood supply of the pelvis.</li> <li>8. Describe the blood supply of the lower limb.</li> </ol>

<b>14</b>	Blood vessels II– Venous system <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"> <li>1. Describe the caval system (course and relations of superior and inferior vena cava).</li> <li>2. Describe tributaries of the superior vena cava draining the head, neck, and upper limbs.</li> <li>3. Describe tributaries of the inferior vena cava draining the abdomen, pelvis, and lower limbs.</li> <li>4. Describe the azygous system and its drainage area.</li> <li>5. Describe the portal venous system.</li> <li>6. Describe and porto caval anastomosis.</li> </ol>
<b>15</b>	Cardiac arrhythmias <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Define different ectopic foci of excitation and the mechanism of re-entry phenomena.</li> <li>2. Describe different types of arrhythmia and the ECG changes in each type.</li> <li>3. Discuss different types of conduction block.</li> </ol>
<b>16</b>	Antiarrhythmic drugs <b><u>(Pharmacology)</u></b>	<ol style="list-style-type: none"> <li>1. Describe the distinguishing features of the 5 major groups of antiarrhythmic drugs.</li> <li>2. Describe mechanisms of action of each group.</li> <li>3. Understand the pharmacokinetics, clinical uses, and major toxic effects of the drugs used in the treatment of arrhythmias.</li> </ol>
<b>17</b>	Rheumatic heart disease <b><u>(Pathology)</u></b>	<ol style="list-style-type: none"> <li>1. Know the main features of rheumatic fever (RF)</li> <li>2. Discuss the etiology and pathogenesis of RF.</li> <li>3. Be familiar with the pathognomonic lesions of RF &amp; RHD.</li> <li>4. Describe the changes in the heart and other organs in RF.</li> <li>5. Describe the chronic sequale of RHD.</li> </ol>
<b>18</b>	Blood flow to tissues <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Describe the local mechanism that controls blood flow to tissues, including acute and long-term control.</li> <li>2. Discuss the metabolic and myogenic theory for control of blood flow.</li> <li>3. Discuss humoral regulation of blood flow, by vasoconstrictor and vasodilator agents.</li> <li>4. Describe the flow of blood to capillaries and the effect of pre-capillary sphincter.</li> <li>5. Discuss the exchange of different substances between blood and interstitial fluid and factors that affect this exchange.</li> <li>6. Identify the primary forces that control fluid movement through capillary membrane.</li> <li>7. Explain normal coronary blood flow during systole and diastole to different parts of the myocardium.</li> <li>8. Discuss the local factors for control of coronary blood flow, local metabolism as primary factor and the oxygen demand.</li> <li>9. Describe the effect of autonomic nervous system on coronary arteries, role of alpha and beta receptors.</li> </ol>
<b>19</b>	Cardiomyopathy, tumors of the heart, and pathology of pericardium <b><u>(Pathology)</u></b>	<ol style="list-style-type: none"> <li>1. To discuss briefly the main features and effects of the main types of cardiomyopathies.</li> <li>2. To classify pericarditis according to type of exudate.</li> <li>3. To describe the pathology of the common types of heart tumors.</li> </ol>
<b>20</b>	Hemodynamics <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Explain the relationship between pressure, flow, and resistance.</li> <li>2. Discuss laminar and turbulent blood flow.</li> <li>3. Understand methods for measurement of blood flow.</li> <li>4. Study and understand Laplace law.</li> </ol>

		<ol style="list-style-type: none"> <li>5. Describe arterial pressure pulsation and transmission of pressure pulses to the peripheral arteries.</li> <li>6. Discuss the function of the veins, venous pressure, venous resistance, venous valve, and venous pump.</li> </ol>
21	Regulation of blood pressure <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Define the blood pressure systole, diastole, and the pulse pressure.</li> <li>2. Define mean arterial blood pressure, circulatory filling pressure, and central venous pressure.</li> <li>3. Explain the effect of gravity on the blood pressure in different parts of the CVS during different positions.</li> <li>4. Discuss the methods of blood pressure measurements.</li> <li>5. Describe the reflex mechanisms for maintaining normal pressure, role of baroreceptors, chemoreceptors, and low-pressure receptors. i.e fast acting mechanisms.</li> <li>6. Discuss the central nervous system ischemic response factor in regulating arterial pressure.</li> <li>7. Discuss the long-term mechanisms for regulation of blood pressure, i.e. renin-angiotensin system, aldosterone, and ADH.</li> </ol>
22	Development of the vascular system <b><u>(Anatomy)</u></b>	<ol style="list-style-type: none"> <li>1. Describe the formation of dorsal aorta.</li> <li>2. Describe the formation of aortic arches and their fate.</li> <li>3. Revise the process of transformation of fetal into adult circulation and the major changes that occur.</li> <li>4. Describe major congenital malformations incurred during these stages and their clinical implications.</li> </ol>
23	Vasculitis <b><u>(Pathology)</u></b>	<ol style="list-style-type: none"> <li>1. Define vasculitis &amp; list the possible causes of this condition.</li> <li>2. Discuss the mechanism of vasculitis.</li> <li>3. Understand the relation between ANCA and vasculitis.</li> <li>4. Classify vasculitis.</li> <li>5. Describe the main features of the different types of vasculitis.</li> </ol>
24	Antihypertensive drugs I <b><u>(Pharmacology)</u></b>	<ol style="list-style-type: none"> <li>1. List major groups of antihypertensive drugs and give an example of drugs in each group.</li> <li>2. Describe the mechanism of action and the values of diuretics used in the treatment of hypertension.</li> <li>3. List the major sites of action of sympathoplegic drugs and give an example of drugs that act at each site.</li> <li>4. List the major indications, contraindications, pharmacokinetics, and adverse effects of each drug mentioned in the above groups.</li> <li>5. Understand the role of vasodilators in the treatment of hypertension.</li> <li>6. Understand the mechanisms of action, pharmacokinetics, indications, contraindications, and adverse effects of commonly used vasodilators.</li> </ol>
25	Antihypertensive drugs II <b><u>(Pharmacology)</u></b>	<ol style="list-style-type: none"> <li>7. List major groups of antihypertensive drugs and give an example of drugs in each group.</li> <li>8. Describe the mechanism of action and the values of diuretics used in the treatment of hypertension.</li> <li>9. List the major sites of action of sympathoplegic drugs and give an example of drugs that act at each site.</li> <li>10. List the major indications, contraindications, pharmacokinetics, and adverse effects of each drug mentioned in the above groups.</li> <li>11. Understand the role of vasodilators in the treatment of hypertension.</li> </ol>

		12. Understand the mechanisms of action, pharmacokinetics, indications, contraindications, and adverse effects of commonly used vasodilators.
26	Surgical aspects of CAD and valvular heart disease <b>(Surgery)</b>	1. Understand the possible surgical complications and consequences of CAD and valvular heart disease. 2. Outline the general surgical interventions used in the management of CAD and valvular heart disease.
27	Cholesterol synthesis and degradation <b>(Biochemistry)</b>	1. Discuss the sources of cholesterol pool in the liver and the major input and output routes. 2. Discuss the cholesterol biosynthetic pathway including the key enzymes and sites of regulation. 3. Discuss the cholesterol degradation pathway including bile acid synthesis pathway and major sites of regulation. 4. Describe the enterohepatic circulation of bile acids and its role in controlling the cholesterol pool in the liver.
28	Arteriosclerosis atherosclerosis <b>(Pathology)</b>	1. Define the terms of arteriosclerosis and atherosclerosis (AS). 2. List the three patterns and two morphologic variants of arteriosclerosis. 3. Describe the main pathological features and disease associations of: medial calcification, hyaline, and hyperplastic arteriosclerosis and histological features of AS. 4. List the risk factors associated with AS. 5. Outline the different theories proposed for the pathogenesis of AS, with special emphasis on response to injury hypothesis. 6. Define aneurysm and list its types. 7. Discuss the pathology of syphilitic aortitis and its effects on the aorta and heart. 8. Define and discuss the etiology, mechanism, and possible outcome of dissecting hematoma.
29	Plasma lipoproteins and familial hypercholesterolemia <b>(Biochemistry)</b>	1. Describe the basic structure, components and types of the various lipoproteins including the various functions of the apoproteins. 2. Describe the metabolic pathway of chylomicrons, VLDL, LDL, and HDL. 3. Describe the biochemical basis of familial hypercholesterolemia.
30	Lipid-lowering drugs <b>(Pharmacology)</b>	4. Understand therapeutic strategies to lower blood lipids. 5. Classify lipid-lowering drugs. 6. Understand the indications, mechanisms of action, toxic effects, pharmacokinetics, and pharmacodynamics of the various classes of lipid-lowering drugs.
31	Cardiac enzymes and other protein markers <b>(Biochemistry)</b>	1. Discuss the clinical relevance of creatine kinase, LDH, troponins, myoglobin, and other peptide markers in the diagnosis of cardiac diseases.
32	Ischemic and hypertensive heart diseases <b>(Pathology)</b>	1. Define the term IHD. 2. Understand the pathogenesis of IHD and list the syndromes associated with it. 3. Correlate the type of angina pectoris with the pathology of coronary arteries. 4. Describe the pathology of myocardial infarction (MI) including: types, gross, histology and sites.

		<ol style="list-style-type: none"> <li>Describe the main features of chronic ischemic heart disease.</li> <li>List causes of sudden cardiac death and outline the mechanism of SCD.</li> <li>List the criteria of HHD and describe its gross and histological features.</li> </ol>
33	Antianginal drugs <b><u>(Pharmacology)</u></b>	<ol style="list-style-type: none"> <li>Define the therapeutic strategies in angina pectoris.</li> <li>List the groups of drugs commonly used in the treatment of angina.</li> <li>Classify and describe the pharmacokinetics, mechanism of action, clinical uses and method of administration, and major toxic effects of nitrates.</li> <li>Understand the role of calcium channel blockers in the treatment of angina.</li> <li>List the most commonly used calcium channel blockers in the treatment of angina with their pharmacokinetics, indications, contraindications and adverse effects.</li> <li>Understand the role of Beta-blockers in the treatment of angina.</li> </ol>
34	Clinical aspects of ischemic heart diseases (IHD) <b><u>(Medicine)</u></b>	<ol style="list-style-type: none"> <li>Describe the clinical presentation (history and physical examination) of the various types of IHD.</li> <li>Understand the possible complications and consequences of IHD.</li> <li>Describe the major investigations used in the diagnosis of IHD.</li> <li>Outline a general management plan for patients with IHD.</li> </ol>
35	Epidemiology of cardiovascular disease (CVD) <b><u>(Com. Med.)</u></b>	<ol style="list-style-type: none"> <li>Appreciate the magnitude and significance of cardiovascular diseases (CVD) as a public health problem.</li> <li>Understand the epidemiology of CVD by person, time, and place.</li> <li>List CVD risk factors with emphasis on modifiable risk factors.</li> <li>Be familiar with the Framingham study, Massachusetts, USA.</li> </ol>
36	Surgical aspects of peripheral vascular diseases <b><u>(Surgery)</u></b>	<ol style="list-style-type: none"> <li>Describe the clinical presentation (history and physical examination) of peripheral vascular diseases (PVD).</li> <li>Understand the possible complications and consequences of PVD.</li> <li>Outline the general surgical interventions used in the management of PVD.</li> </ol>
37	Circulatory shock and heart failure <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>Define circulatory shock, and the difference between cardiogenic and hypovolumic shock.</li> <li>Discuss the stages of shock; non-progressive and progressive.</li> <li>Describe sympathetic reflex compensation in shock.</li> <li>Discuss the effects of shock on the human body.</li> </ol>
38	Varicose veins, lymphatic pathology, and tumors of blood vessels <b><u>(Pathology)</u></b>	<ol style="list-style-type: none"> <li>Discuss the pathogenesis of varicose veins (VV).</li> <li>Know the different sites where VV can occur.</li> <li>List the sequelae of VV and other lymphatic disorders.</li> <li>Know the criteria that differentiate between benign, borderline, and malignant blood vessel tumors.</li> <li>Give examples of the different types of vascular tumors.</li> </ol>
39	Drugs used in the treatment of heart failure <b><u>(Pharmacology)</u></b>	<ol style="list-style-type: none"> <li>Understand the therapeutic strategies in congestive cardiac failure.</li> <li>Classify and give examples of digitalis glycosides.</li> <li>Describe the pharmacokinetics of digitalis.</li> <li>Understand the mechanism of action and the effects of digitalis.</li> <li>List the major toxic effects and their treatment of digitalis.</li> </ol>

		6. Describe the role of diuretics, ACE inhibitors, vasodilators, and B1-selective adrenoceptor agonists in the treatment of congestive cardiac failure.
40	Prevention and control of CVD <b><u>(Com. Med.)</u></b>	1. Distinguish high risk group vs. population-based approach to control CVD. 2. List the levels of control of CVD. 3. Case study: North Karelia, Finland program.

### B-Practical Laboratory Sessions:

#	Lab. Title	Objectives
1	Gross anatomy of the cardiovascular system  <b><u>(Anatomy)</u></b>	<p><b><u>Anatomy of the heart and mediastinum:</u></b></p> <ol style="list-style-type: none"> <li>1. Describe the normal location and surface markings of the heart, its valves and great vessels.</li> <li>2. Identify the heart and its great vessels in-situ in cross sections.</li> <li>3. Appreciate important relations of the heart in the middle mediastinum.</li> <li>4. Examine external and internal features of the heart including its pericardium in plastic models.</li> <li>5. Identify location, subdivisions, and the contents of the mediastinum.</li> <li>6. Identify images of the heart and its blood supply in plain chest X-ray, angiograms and CT scans.</li> </ol> <p><b><u>Anatomy of blood vessels - arterial and venous systems:</u></b></p> <ol style="list-style-type: none"> <li>1. Identify main arteries and veins and their branches in the thorax, upper limbs, and lower limbs.</li> <li>2. Identify main arteries and veins in the head and neck and their branches.</li> <li>3. Identify main arteries and veins in the abdomen and their branches.</li> <li>4. Study and identify the above arteries in angiograms and cross sections.</li> <li>5. On the living subject, locate and feel the important pulses in the above regions (common carotid, superficial temporal, subclavian, abdominal aorta axillary, brachial, radial, ulnar, femoral, popliteal, dorsal pedal and posterior tibial).</li> </ol>
2	ECG  <b><u>(Physiology)</u></b>	<ol style="list-style-type: none"> <li>1. Locate the position of different bipolar and unipolar leads.</li> <li>2. Be familiar with ECG machine and how to record the ECG.</li> <li>3. Identify different waves of the ECG and the shapes and amplitude of each.</li> <li>4. Understand the methods of calculation the heart rate and the cardiac axis from the recording ECG.</li> </ol>
3	Pathology of the heart  <b><u>(Pathology)</u></b>	<ol style="list-style-type: none"> <li>1. Understand the gross and microscopic appearances of the following cardiac pathologies: <ul style="list-style-type: none"> <li>• Endocarditis (infective vs. non-infective).</li> <li>• Myocarditis.</li> <li>• Pericarditis.</li> <li>• Cardiomyopathies.</li> </ul> </li> </ol>

4	Histology of the heart and blood vessels  <u>(Anatomy)</u>	<ol style="list-style-type: none"> <li>1. Examine the detailed microscopic structure of the cardiac muscle.</li> <li>2. Examine, compare and understand the microscopic structure of walls of different caliber blood vessels.</li> <li>3. Examine and study the ultrastructure of blood capillaries and sinusoids by the aid of electron micrographs.</li> </ol>
5	Blood pressure and heart sounds  <u>(Physiology)</u>	<ol style="list-style-type: none"> <li>1. Explain methods of blood pressure measurement (palpation and auscultation) during systole and diastole.</li> <li>2. Explain the advantages and disadvantages of each method</li> <li>3. Discuss the difference of pressure values in different parts of the body during different positions.</li> <li>4. Discuss the cause of heart sounds and their relation to the ECG.</li> <li>5. Identify the location of different regions on the chest wall to hear the maximal intensity of the component of each heart sound.</li> </ol>
6	Pathology of blood vessels  <u>(Pathology)</u>	<ol style="list-style-type: none"> <li>1. Identify the gross histology of atherosclerosis.</li> <li>2. Study the histological features of vasculitis and common types of blood vessel tumours.</li> </ol>

### Evaluation Items and Regulations:

Grade Categories	Grade %
<b>Attendance</b>	<b>5%</b>
<b>Students' Activities</b>	<b>10%</b>
<b>Midterm Exam</b>	<b>45%</b>
<b>Final Exam</b>	<b>40%</b>
<b>Total</b>	<b>100%</b>

- All exams will be held online and in an integrated format.
- *Midterm exam covers lectures of basic medical sciences (37 lectures), while final exam covers clinical lectures (3 lectures), practical sessions (6 labs), and small group discussions (2 cases).*
- The final exam will be at the end of the module course, NOT at the end of the semester.
- Any act of misconduct, as determined by the course coordinator according to University's regulations, would be subtracted from activity grades.

### Participating Staff Members:

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### Dates, Times, and Places:

- **Lectures' Venue: TBD.**
- **Course begins:** Sunday 8<sup>th</sup> of March 2020.
- **Lectures end:** Thursday 2<sup>nd</sup> of April 2020.
- **Midterm Exam:** Monday 6/4/2020. Locations to be assigned by registrar.
- **Final Exam:** Thursday 9/4/2020. Locations to be assigned by registrar.
- **Last Date for Submission of Activities:** Thursday April 2<sup>nd</sup> 2020.
- **Small Group Discussions (SGA) Locations:** Separate attached schedule.

## **Small Group Discussions:**

### **Case I. Acute ST Elevation Myocardial Infarction**

This 49 year-old married male school teacher was a 2 pack/day smoker with a history of diabetes mellitus, hyperlipidemia, and obesity, and a family history of coronary artery disease. He was awakened from his sleep at 03:00 A.M. with crushing substernal chest pain, which radiated to his left arm, and was accompanied by shortness of breath. When paramedics arrived, they found the patient cool, clammy, bradycardic, and hypotensive. Intravenous fluids and atropine were given, and he was transported to a tertiary hospital.

On arrival to the emergency department at the hospital, the patient was in considerable distress. He was still bradycardic (heart rate 54 bpm). He had no jugular venous distention. He had decreased breath sounds with occasional expiratory wheezes. At 04:01 A.M., his white blood cell count was 7,900/cu mm, hematocrit 45.8%, platelets 246,000/cu mm, creatine phosphokinase (CPK) 89 IU/L, and troponin-I <0.4 ng/ml. Electrocardiogram (ECG) showed ST-segment elevation in leads II, III, AVF, and V4-V6. Chest x-ray (CXR) showed borderline cardiomegaly without signs of pulmonary edema.

### **Discussion:**

1. Identify the major and minor risk factors for cardiovascular disease in this patient.
2. Discuss the acute coronary syndrome: types, criteria for diagnosis, and general guidelines for management.
3. In our patient, what is the most likely affected coronary vessel?
4. In our patient, CPK and troponin-I were normal at presentation, discuss the cardiac markers' profile changes in acute myocardial infarction.
5. What is the explanation for the bradycardia in our patient?

### **Case II. Heart Failure**

A 61 year-old male comes to the emergency department complaining of one-week duration of breathlessness on minimal exercise, orthopnea, lower limb edema, and nocturnal paroxysmal dyspnea. He reported that this recent clinical worsening of symptoms occurred after an attack of "cold". He denied chest pain, palpitations, fever, or other symptoms.

Besides being obese (BMI = 35.4 kg/m<sup>2</sup>), he denied other known cardiovascular risk factors, although he didn't seek medical care for the last 10 years. His past medical history was unremarkable, except for knee pain, with intermittent use of NSAIDs (ibuprofen). He had no relevant family history.

On physical examination, his blood pressure was 180/100 mmHg, heart rate 108 bpm, oxygen saturation 93%. He had rales over the lower third of both lung fields, raised jugular venous pressure, and bilateral lower limb edema. On cardiac auscultation, a grade II/VI aortic murmur was heard. He had no fever.

An ECG was done and showed signs of left ventricular hypertrophy (LVH), but no ischemic changes were found. Chest X-ray (CXR) was done and showed cardiomegaly, bilateral

prominent vascular markings in the lung fields, with lung congestion. An echocardiogram was performed later and showed concentric LVH, LV dilatation, and global LV hypokinesia with overall moderately impaired systolic function (Ejection Fraction = 35%). A heavily calcified aortic valve was observed with associated aortic stenosis: valve functional area was 0.9 cm<sup>2</sup>.

### **Discussion:**

1. What is the cause of breathlessness in this patient?
2. Discuss types and causes of heart failure.
3. What is the most likely cause of the left ventricular hypertrophy and impairment in our patient?
4. Discuss the general guidelines for managing this patient.

### **Students' Activities**

#### **Students' Responsibilities:**

- Students are requested to attend lectures sitting on their dedicated seats.
- Students are requested to pay good attention maintain a professional behavior during lectures, labs, and small group discussions.
- Students are requested to attend laboratories and small group discussions.
- During the course of the module, students will be divided into small groups conducting research activities. These activities are intended to enhance student active participation in learning and they will be subject to evaluation.

#### **Students' Activities:**

These activities will include:

1. Performance of small-scale research projects.
2. Writing short essays on the most recent advancements in translational medicine and or up-to-date clinical guidelines.

## **ACTIVITY # 1: SMALL SCALE RESEARCH PROJECTS:**

Herein, students are requested to fulfil a pre-defined task related to the CVS system, as assigned to them by the course coordinator. Topics of interest are listed below. This activity aims at encouraging the students' expression of innovative abilities in addition to enhancing their eventual integration into the clinical setting. Students are requested to submit a brief summary of their work (*four pages long, double spaced, narrow margins A4 sized, Times New Roman font*). **Plagiarism is prohibited and will severely affect your grade.**

#	Topic	Instructor
1	Compare blood pressure readings taken manually or by automated machines in 20 subjects.	Dr. A. Alzoubi
2	How does smoking affect exercise tolerance? Let a chronic smoker perform the treadmill test at KAUH and report results.	Dr. S. Rawashdeh
3	Effect of exercise on heart rate and blood pressure. Try going upstairs for example to report such effects on 10 subjects.	Dr. I. Matalka
4	Compare resting heart rates between 20 athletes and 20 non-athlete subjects.	Prof. Nabil Khouri
5	Short video about anatomical landmarks of major arteries used in the measurement of heart rate.	Prof. Nabil Khouri
6	Measure waist circumference and body mass index, and correlate them with blood pressure and heart rate readings in 20 subjects.	Dr. I. Matalka
7	Report postural changes of blood pressure readings in 20 subjects; supine vs. standing.	Prof. S. Khatib
8	Survey the prevalence of hypertension, ischemic heart disease, and heart failure in the families of your class; report relation to age and gender.	Dr. A. Alzoubi
9	Survey the use of antihypertensive drugs in the families of your class; report classes, dosing, and side effects.	Dr. N. Rabadi
10	Survey the prevalence of myocardial infarction cases at KAUH over one year.	Dr. I. Matalka
11	Design a brochure about healthy diets for CVS diseases. Your target audience are the public.	Dr. H. Kanan
12	Design a brochure about risks of smoking on cardiovascular health. Your target audience are the public.	Dr. H. Kanan
13	Design a social media campaign to quit smoking on JUST campus. Outline the cardiovascular impact of smoking.	Dr. A. Al-Zoubi
14	Outline the major risk factors for peripheral vascular diseases in Jordan, by visiting Dr. Qusai Al-Jarrah clinic at KAUH.	Dr. Q. Jarrah
15	Examine the effect of exercise on ECG; report the changes in ECG with exercise / during exercise in shape of waves and interval durations.	Prof. S. Khatib
16	Visit Dr. Sukainah Rawashdeh clinic at KAUH and report your experience with demonstration of echocardiography procedure.	Dr. S. Rawashdeh
17	Report radiographic changes of heart failure in the form of short video, outlining the normal features of chest X ray.	Dr. A. Kasasbeh

**EVALUATION CRITERIA:**

This activity will be evaluated based on the following criteria:

<b>ITEM</b>	<b>SCORE</b>
<b>English Language</b>	----- out of 2
<b>Flow of Logic</b>	----- out of 2
<b>Effective Use of Illustrations</b>	----- out of 1
<b>Originality (No plagiarism)</b>	----- out of 2
<b>Proper Citations</b>	----- out of 2
<b>Adherence to Format Guidelines</b>	----- out of 1
<b>Total</b>	<b>----- out of 10</b>

## **ACTIVITY # 2: WRITING SHORT ESSAYS:**

Herein, students are requested to submit a brief summary (*four pages long, double spaced, narrow margins A4 sized, Times New Roman font*) on the most recent advancements in translational medicine, and up-to-date clinical guidelines of the diagnosis and management of defined conditions related to the topics covered in the module. Topics of interest are listed below, and student groups will be randomly assigned to each of the topics by the course coordinator. This activity aims at empowering the students with the most relevant research skills, in addition to potentiating the concept of continuous medical education in the students. **Plagiarism is prohibited and will severely affect your grade.**

#	Topic	Instructor
1	Write a critical review on the following article: " <i>Natriuretic peptide-guided heart failure Management</i> " Richard Troughton, G. Michael Felker, and James L. Januzzi Jr. <i>European Heart Journal</i> (2014) 35, 16–24.	Dr. N. Bashir
2	Clinical approach to hypertensive crises.	Dr.A. Kasasbeh
3	The role of angiogenesis in ischemic heart diseases.	Dr. I. Matalaka
4	Funny Sodium Channels in the heart; what are these and how do they function?	Prof. S. Khatib
5	What is metabolic syndrome X? Briefly summarize the clinical significance of the condition.	Dr. A. Al-Mohtaseb
6	Variations in the anatomy of superficial palmar artery.	Dr. N. Khouri
7	Atrial and brain natriuretic peptides; where do we use these clinically?	Dr. N. Bashir
8	Omega 3 containing food for the normalization of serum lipids: facts vs. myths.	Dr. N. Rabadi
9	Right heart catheterization, where is it indicated and how is it clinically significant?	Dr. A. Kasasbeh
10	Clinical approach to treatment of atrial fibrillation.	Dr. S. Rawashdeh
11	Role of adult progenitor cells and stem cells in heart diseases.	Dr. A. Al-Mohtaseb
12	What is the new heart disease staging system?	Dr. A. Al-Mohtaseb
13	Structural cardiac remodeling in heart failure; summary of the pathophysiology.	Dr. A. Al-Mohtaseb
14	The emerging role of hypercholesterolemia in tumorigenesis.	Dr. N. Bashir
15	Ibn Alnafis; what did he achieve in the history of medicine?	Dr. A. Alzoubi
16	Explore and compare different types of Hyperlipoproteinemia.	Dr. N. Bashir
17	Clinical presentation of abdominal aortic aneurysms (AAA).	Dr. N. Waqfi
18	The effect on using Sildenafil preoperative and postoperative surgery on pulmonary hypertension in ventricular septal defect in children	Prof. S. Khatib

<b>19</b>	The effects of Sildenafil on respiratory and CVS parameters, like blood pressure and oxygen consumption during exercise	Prof. S. Khatib
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**EVALUATION CRITERIA:**

This activity will be evaluated based on the following criteria:

<b>ITEM</b>	<b>SCORE</b>
<b>English Language</b>	----- out of 2
<b>Flow of Logic</b>	----- out of 2
<b>Effective Use of Illustrations</b>	----- out of 1
<b>Originality (No plagiarism)</b>	----- out of 2
<b>Proper Citations</b>	----- out of 2
<b>Adherence to Format Guidelines</b>	----- out of 1
<b>Total</b>	<b>----- out of 10</b>

## Practical Sessions Manuals:

### Physiology LAB:

#### Lab 1: Electrocardiography (ECG)

##### Objectives:

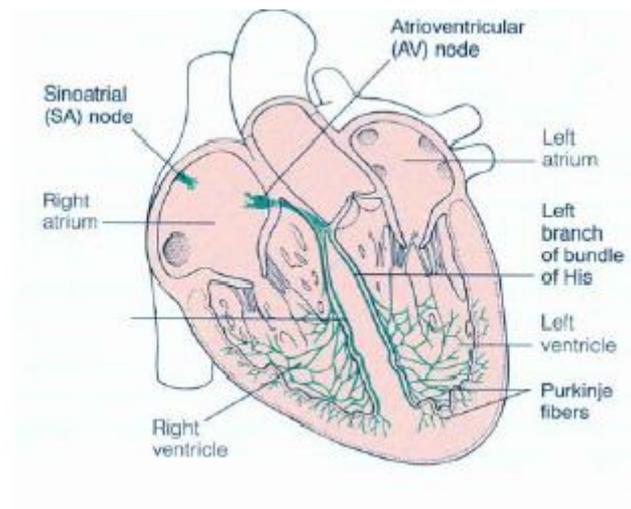
1. To locate the position of different bipolar and unipolar leads.
2. To be familiar with ECG machine and how to record the ECG.
3. To identify different waves of the ECG and the shapes and amplitude of each.
4. To understand the methods of calculation the heart rate and the cardiac axis from the recording ECG.

##### Heart conduction system:

The conduction system of the heart is a network of specialized cardiac cells designed for starting each heart contraction and for rapid and coordinated spread of excitation.

The components of the conduction system (**Figure 1**) are:

1. Sinoatrial node (SA node or pacemaker).
2. Atrioventricular node (AV node).
3. Atrioventricular bundle (AV bundle or Bundle of His).
4. Right and left branches.
5. Purkinje fiber.



**Figure 1:** Specialized conducting system of the heart

## What Is Electrocardiogram?

An electrocardiogram is a common, painless test that records the electric currents produced by the heart and converts it into lines called "waveforms" that can be seen on a monitor or printed out on paper. The waveforms created by the ECG can be divided into time segments to measure the rate of movement of the heart's electrical impulses. It measures the current through the skin with electrodes that are attached to the chest. **Electrodes are harmless devices** with wires that lead to a recording machine.

**The electrocardiograph:** is the machine used for recording the electrocardiogram (**Figure- 2**).

**An electrocardiogram (Electrocardiography):** is a recording of the electrical activity of the heart made directly from the body surface.



**Figure 2:** The Electrocardiograph

## Principles of Electrocardiography:

Electrical impulses drive the heart. Just before contraction a wave of depolarization is spreading along the muscle fibers of the heart and this generates an electrical current which has both magnitude and direction. Because the body cells and fluids contain electrolytes the body can be considered to be conductor and the electrical activity of the heart can be picked up from the surface of the heart by means of electrodes placed on the skin. However, the voltages are quite small and have to be amplified sufficiently. For recording the ECG metallic electrodes attached to wires are placed on the skin using electrolyte paste.

**A lead is** a pair of electrodes placed on the body in designated anatomical locations and connected to an ECG recorder or oscilloscope. Each lead has a positive and negative pole. The standard ECG has 12 leads.

1. **Bipolar (3) leads:** where they record the potential difference between two points (the positive and negative poles).
2. **Unipolar leads (9):** where they record the electrical potential at a particular point by means of a single exploring electrode.

**12 standard leads are used in ECG, 3 of these are bipolar leads** that measure the voltage difference between the arms or an arm and leg, and **9 are unipolar leads** record the electrical potential at a particular point by means of a single exploring electrode. Together the 12 leads provide a fairly comprehensive picture of the electrical activity of the heart.

### Routine ECG Examination:

In the routine ECG examination, the recordings are made from two planes; the frontal plane and the transverse plane (**Figure 3**).

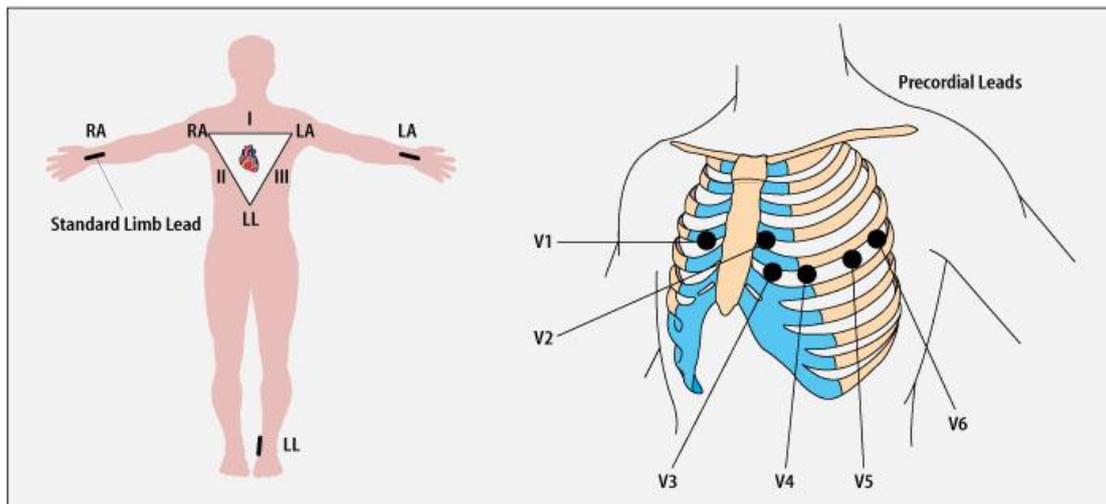
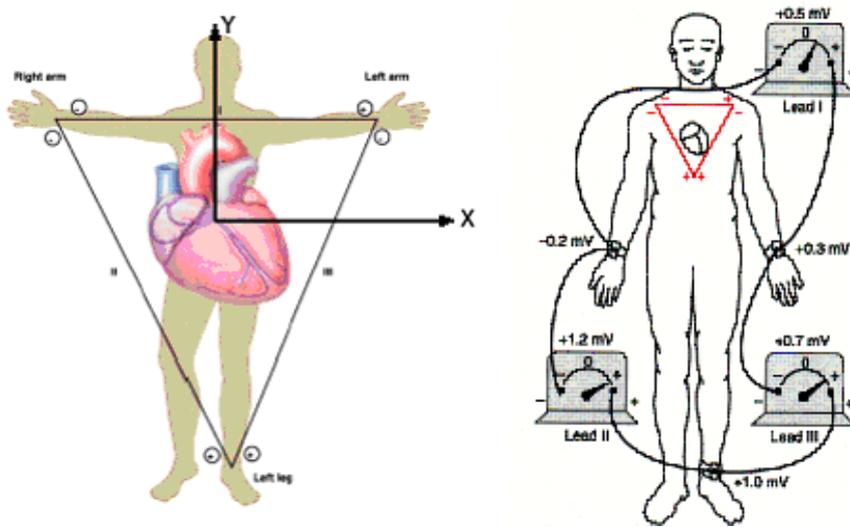


Figure 3: Recording the ECG is made in 2 planes, the frontal and transverse plane

#### A- Frontal plane leads:

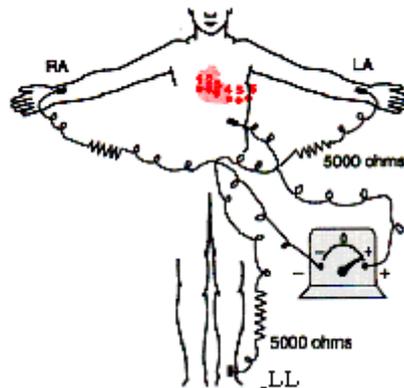
##### 1. Standard bipolar limb leads (Figure 4):

- Measures “potential differences” between two points. They are:
  - **Lead I: Left arm(+) & Right arm(-)**
  - **Lead II: Right arm(-) & left leg (+)**
  - **Lead III: Left arm(-) & left leg(+)**



**Figure 4:** Limb leads system of the ECG

2. **Augmented unipolar leads:** same 3 electrodes used in standard limbs leads (Figure 5).
  - **aVR: Augmented voltage of right arm.**
  - **aVL: Augmented voltage of left arm.**
  - **aVF: Augmented voltage of left foot.**



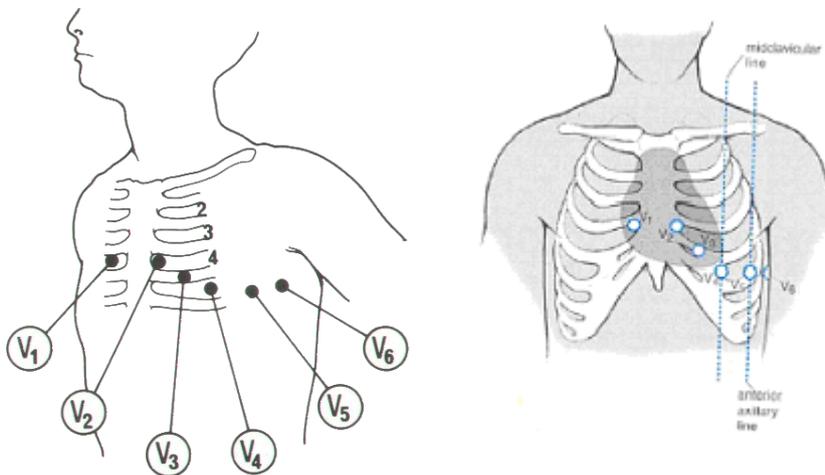
**Figure 5:** Unipolar Leads Connections

**B- Transverse or horizontal plane leads:**

There are six unipolar leads recorded in the transverse plane with the usual ECG and they provide information on how the instantaneous cardiac vectors are directed anteriorly and posteriorly. These are termed the **precordial leads or chest leads**. These 6 unipolar leads are numbered V1-V6.

**The standard chest lead positions are as follows (Figure 6):**

1. V<sub>1</sub> Fourth intercostal space, right sternal edge.
2. V<sub>2</sub> Fourth intercostal space, left sternal edge.
3. V<sub>4</sub> (place the fourth electrode before the third) Fifth intercostal space in the mid-clavicular line.
4. V<sub>3</sub> bang it half way between the second and fourth electrodes.
5. V<sub>5</sub> Lies on the fifth rib in the anterior axillary line.
6. V<sub>6</sub> On an imaginary horizontal line with V<sub>5</sub> in the mid axillary line.



**Figure 6:** Precordial leads

### **Characteristics of Electrocardiogram:**

The ECG is composed of P wave, QRS complex, and T wave (**Figure 7**), and some related intervals (**Figure 8**).

#### **P Wave**

The P wave is the wave of atrial depolarization. As the atria depolarize, the P wave shows up on the ECG. In a patient with normal physiology and with the SA node acting as the pacemaker of the heart, the P wave has these characteristics:

- Smooth and rounded
- Not more than 3 mm tall
- Upright in leads I, II, aVF.

#### **PR Interval**

The next component is the PR interval, which includes the P wave and the space up until the beginning of the QRS complex. The PR interval represents the time it takes the electrical impulse to travel from the SA node to the ventricles. By the end of the PR interval, the atria are beginning to repolarize and the ventricles are beginning to depolarize or become electrically stimulated.

The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. The normal PR interval duration is 0.12 to 0.20 seconds or 120 – 200-ms.

**QRS Complex:**

The QRS complex is the wave of ventricular depolarization. It is generally calling the wave of ventricular depolarization a “QRS complex”. Technically, the Q wave is the first downward stroke. An R wave is the first positive stroke, and an S wave is a negative stroke that follows a positive upstroke.

The QRS should be at least 5 mm and not more than 20 mm tall. The width of the QRS is measured from the beginning of the Q wave to the end of the S. Normal QRS duration is 0.06 to 0.10 seconds.

**ST Segment**

This segment at the end of QRS complex up to the beginning of T wave. The ST segment indicates the period of time between the end of ventricular depolarization and the beginning of ventricular repolarization. Generally the ST segment is ISOELECTRIC, or on the “baseline”. A deviation of the ST segment from the baseline (either a depression or elevation) may be indicative of coronary artery disease.

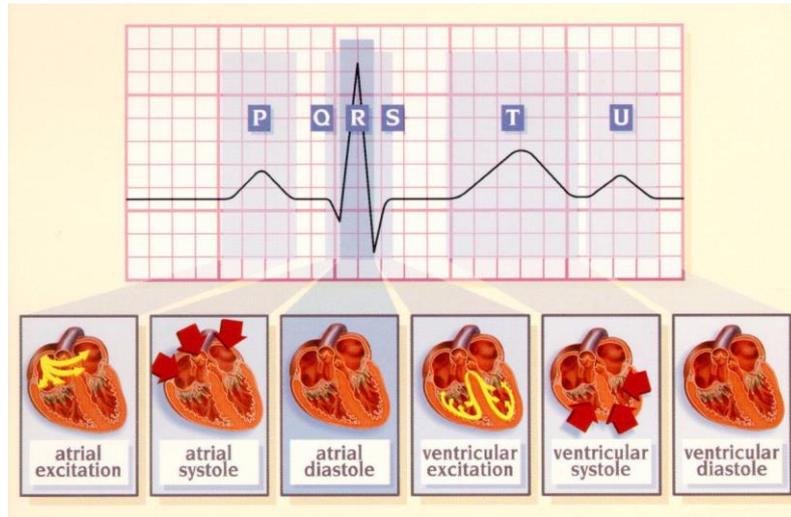
**T Wave**

The T wave is the wave of ventricular repolarization. The T wave usually deflects in the same direction as the QRS complex, and should be smooth and rounded. The period from the beginning of the T wave to nearly the end is called the “relative refractory period”. At this time, the ventricles are vulnerable. Table 1 list some ECG related intervals and the duration of each one.

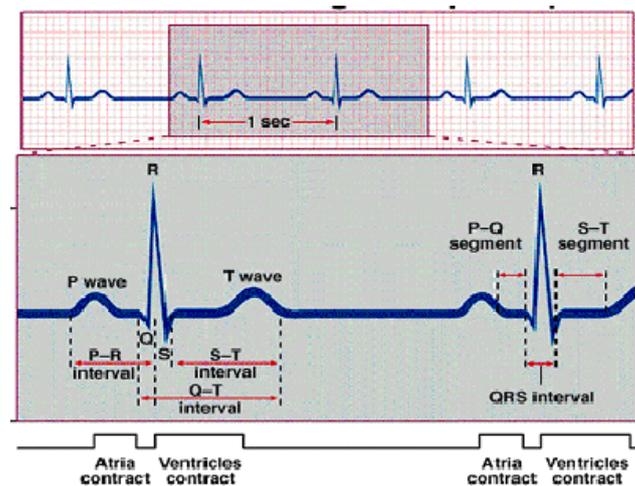
Intervals	Normal durations	Events in Heart during interval
PR interval	0.12 – 0.20	Atrial depolarization and conduction through the AV node
QRS duration	0.08 – 0.10	Ventricular depolarization and atrial repolarization
QT interval	0.40 – 0.43	Ventricular depolarization plus ventricular repolarization

\*Depends on heart rate

**Table 1:** ECG intervals



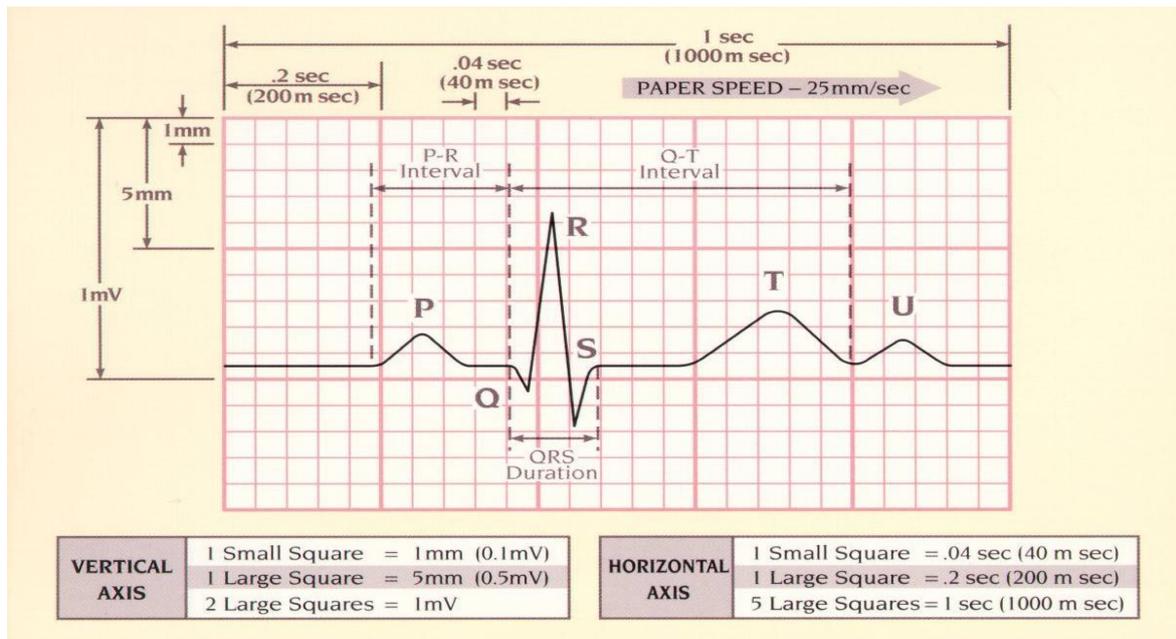
**Figure7:** The waves of standard ECG



**Figure 8:** ECG waves and intervals

### ECG Paper: What Does It Means?

**Figure 9** below shows the design and the calculations of the ECG paper, which is used to study the patient heart.



**Figure 9:** The design and the calculations regard the ECG paper.

- 1 mm intervals (vertical and horizontal)
- Every 5 mm the line is accentuated (bold).
- Speed of the record is **25 mm/sec**
- 5 mm distance = **0.2 sec = 200 mSec**
- 1 mm distance = **0.04 sec = 40 mSec**
- **1 sec = 5 bold lines = 25 mm**
- 1.0 mV = 10 mm of vertical deflection on the grid

### ECG Interpretation:

1. Rate.
2. Rhythm.
3. Duration time for waves and intervals.

#### 1. Heart Rate:

In normal sinus rhythm, a resting heart rate of below 60 bpm (beat per minute) is called bradycardia and a rate of above 100 bpm is called tachycardia.

#### **How to calculate heart rate using the ECG paper:**

- Each small horizontal axis is 0.04 second, and each large horizontal square is 0.2 second.
- P-P, R-R is one cycle.

**Heart rate** = 1500/ number of small squares.

**Example: if the distance between R-R is 18 mm, the heart rate is  $1500/18 = 83.3$**

## **2. Rhythm evaluation:**

- The most difficult part of ECG interpretation
- To assess the rhythm, one must identify the P waves and QRS complexes and determine the relationship between them.
- In the normal cardiac rhythm there is a constant distance between similar waves (R wave to R, P to P etc.)
- Arrhythmias are abnormal (inconsistent) cardiac rhythms

## **How electrocardiogram is performed?**

1. Electrodes are placed on the chest, arms and legs.
2. The examiner attaches electrodes to the chest, wrists, and legs with a special gel.
3. A recording machine will trace a wave pattern for each wire lead.
4. For a resting ECG, the person must remain still and quiet. The test takes about five to ten minutes.

## **Artifacts of ECG:**

**The causes of ECG artifacts are:**

- Poor skin contact.
- Gel drying up due to exposure to air for long time.
- Damaged cables.
- Skeletal muscle contraction.

## **Physiology LAB:**

### **Lab 2: Blood Pressure and Heart Sounds**

#### **Objectives:**

1. Explain methods of blood pressure measurement (palpation and auscultation) during systole and diastole.
2. Explain the advantages and disadvantages of each method
3. Discuss the difference of pressure values in different parts of the body during different positions.
4. Discuss the cause of heart sounds and their relation to the ECG.
5. Identify the location of different regions on the chest wall to hear the maximal intensity of the component of each heart sound.

#### **Blood Pressure:**

Blood pressure is measured in the peripheral artery of arm or leg(usually in the brachial artery). An occluding pressure is applied to the surface of the limb through a pneumatic cuff.

Pneumatic cuff consist of:

1. Cuff: a rubber bag usually the adult size 13 by 23 cm.
2. Inflator: rubber bulb with two valves that let the air move in one direction.
3. Pressure indicator: Hg manometer

**The systolic pressure** is the maximum pressure in an artery during ventricular contraction.

**The diastolic pressure** is the lowest pressure in an artery during ventricular relaxation.

Systolic blood pressure could be measured by two methods:

1. Palpatory Method: measures systolic pressure only)
2. Auscultatory Method: measures both systolic and diastolic pressures.

Diastolic blood pressure could be measured by two methods:

1. Auscultatory Method: measures both systolic and diastolic pressures.
2. Observation of maximum oscillation: measures diastolic pressure only.

### How is blood pressure measured?

Here you are going to use the following instruments (Fig.9)

1. A sphygmomanometer (blood pressure cuff) to measure systolic pressure by palpatory method.

It is important that the subject is relaxed and rested for at least 5-10 minutes before having his blood pressure taken. Deflate the cuff and place it around the upper arm so it fits not too tightly. If you're right handed, you should hold the bulb/pump in your left hand to inflate the cuff. Hold it in the palm so your fingers can easily reach the valve at the top to open/close the outlet valve. Observer palpate radial artery and inflate the cuff until radial pulsation no longer perceptible and take the manometer reading, pressure is then reduced slowly until pulsation again appear, manometer reading is taken as systolic pressure.



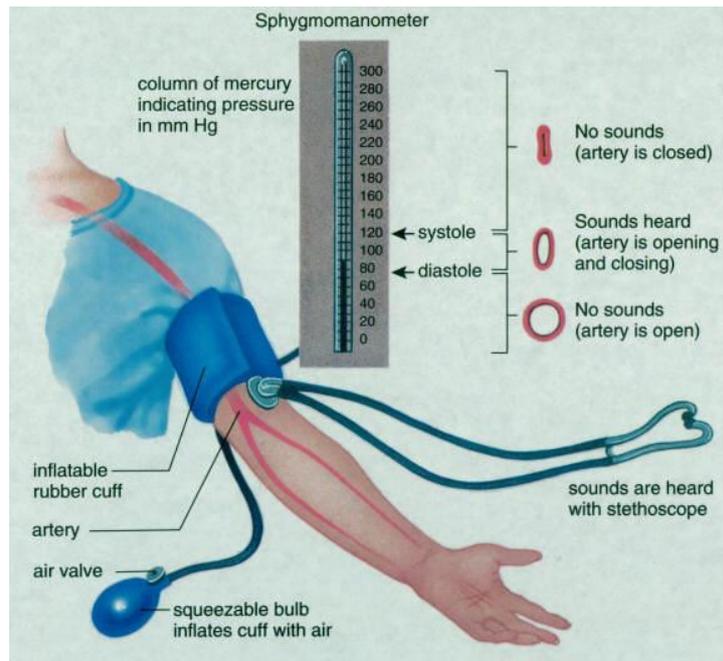
**Figure 9:** A Stethoscope and a Sphygmomanometer.

2. A sphygmomanometer (blood pressure cuff)& stethoscope to measure both systolic & diastolic blood pressure by auscultatory method

It is important that the subject is relaxed and rested for at least 5-10 minutes before having his blood pressure taken. Deflate the cuff and place it around the upper arm so it fits snugly, but not too tightly. If you're right handed, you should hold the bulb/pump in your left hand to inflate the cuff. Hold it in the palm so your fingers can easily reach the valve at the top to open/close the outlet valve. Put the head of the stethoscope just under the edge of the cuff, a little above the crease of the person's elbow. Hold it there firmly with your right hand. Put the ear pieces of the stethoscope in your ears. . Inflate the cuff with brisk squeezes of the bulb. Watch the pressure manometer as you do it. For most adult people, you shouldn't need to go over 180 (the markings indicate "pressure" in mm Hg).

At 180 mmHg, slightly open the valve on the air pump (held in your left hand, as above). This part takes practice. It's important that you don't let the air out too suddenly. Likewise, your friend will be quite irritated with you if you let it out too slowly. Now, pay attention to what you hear through the stethoscope as the mercury column falls. The first time you hear the sound, note what the reading was on the pressure manometer. This value represents the **systolic blood pressure**. The sounds should continue and become louder in intensity. Note the pressure reading when you hear the sound for the last time. This value represents the **diastolic blood pressure**.

The sound from the systolic until it is disappeared is grouped into five phases known as Korotkov sounds: sudden appearance of sharp sound (systolic); slightly muffled around 20mm; sound of increased intensity and shortness; less intense sound; the sound disappears. Afterwards, open the air valve completely to release any remaining pressure.



**Figure 10:** Principle of blood pressure measurement

### Heart Sounds:

Two heart sounds are normally heard by a stethoscope during each cardiac cycle.

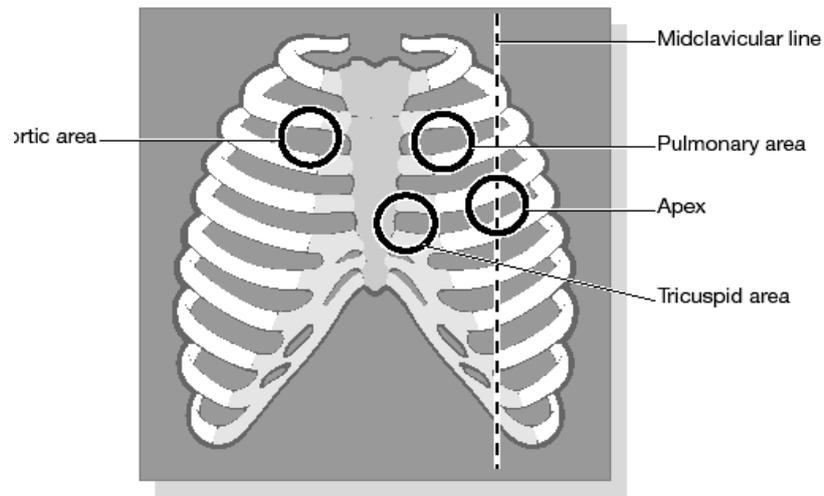
**First Heart Sound (S1):** The first heart sound is caused by the mitral and tricuspid valves closure (AV-valves) at the beginning of systole. The mitral valve closes before the tricuspid valve, although it is not usually possible to appreciate the dual nature of the sound.

**Second heart sound (S2):** The second heart sound is caused by the closure of the aortic and then pulmonary valves (semilunar valves). Because the delay is greater than in the first heart sound this usually can be heard. The splitting becomes wider with inspiration and narrower with expiration.

### The four main areas usually used to listen to the heart sounds

- Mitral valve: at the position of the apex beat.
- Tricuspid valve: over the xiphoid process.
- Aortic valve: in the 2<sup>nd</sup> right intercostal space.

- Pulmonary valve at the 2<sup>nd</sup> left intercostal space.

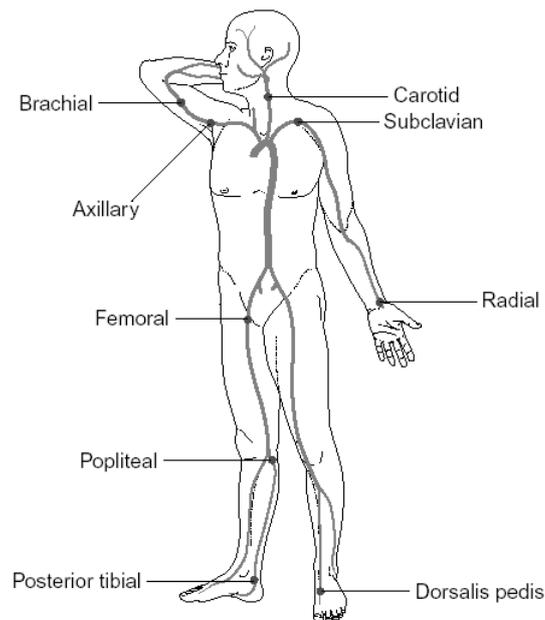


**Figure 8:** Auscultation of precordium.

### **Arterial pulse:**

The **arterial pulse**: is a pressure wave distending the arterial wall starting from the aorta toward peripherally. Arterial pulse reflects the number of heart beats per minute, and it is measured at different parts of the body. (**Fig 4**). Most common part:

1. Radial artery in the wrist
2. Brachial artery in the elbow
3. Common carotid artery in the neck

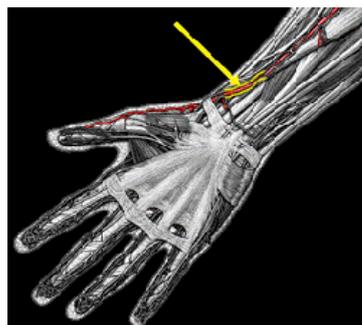


**Figure 3: Arterial pulses**

When you examine the arterial pulse, you are going to study the following characteristics:

1. Heart Rate: number of beats/min.
2. Rhythm: regularity of intervals.
3. Condition of the artery(soft or hard)
4. Character of the pulse wave.

The radial pulse is usually used to assess for rate, rhythm, character and volume. All the pulses should be palpated and the volume compared with the other side (not simultaneously in the case of the carotid pulse). **Radial artery (Fig. 5)**

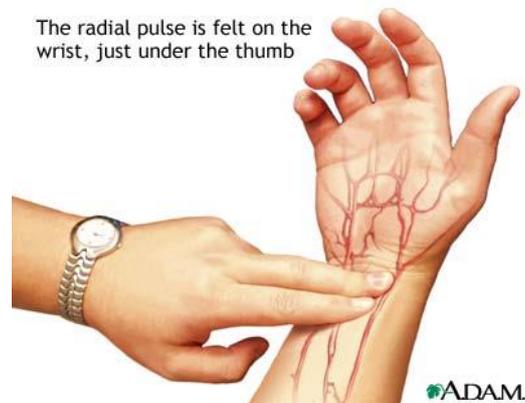


**Figure 5: Radial artery**

Assess the following, using the right radial pulse.

- A- Rate:** To measure the pulse at the wrist, place the index and middle finger over the underside of the opposite wrist, below the base of the thumb. Press firmly with flat fingers until you feel the pulse in the radial artery, To determine heart

rate, one feels the beats at a pulse point like the inside of the wrist for 30 seconds, and multiplies this numbers by two. This is the per-minute total (Fig.6).



**Figure 6:** The measurement of radial pulse

**Abnormal rates could be:**

- Bradycardia (rate below normal). Below 60
- Tachycardia (rate above normal). Above 100

**B- Rhythm:** can be classified into 2 categories:*regular* or *irregular*:

**C- Character:**The character of the pulse may be one of the following weak or strong.

**Anatomy LAB:**

Anatomy Laboratory checklist

Lab 1: Cardiovascular system

Identify the following structures on plastic and plastinated models.

The Heart

- Right atrium  Left atrium  Auricle  Pectinate muscle  Tricuspid valve  Mitral or bicuspid valve  Right ventricle  Left ventricle  Trabeculae carneae  Papillary muscle
- Chordae Tendineae  Pulmonary valve  Aortic valve

Major vessels

- Pulmonary trunk  Pulmonary arteries  Pulmonary veins  Aorta  Coronary arteries
- Cardiac veins  Coronary sinus  Superior vena cava  Inferior vena cava  Brachiocephalic artery and veins  Common carotid arteries  Internal jugular veins

Subclavian arteries and veins □ Axillary arteries and veins □ Brachial arteries and veins □  
Radial arteries and veins □ Ulnar arteries and veins □ Common iliac arteries and veins □  
Internal iliac arteries and veins □ External iliac arteries and veins □ Femoral arteries and  
veins □ Popliteal arteries and veins □ Anterior tibial arteries and veins □ Posterior tibial  
arteries and veins

## HISTOLOGY LAB 2:

### CVS

**I. General structure of blood vessels** (Fig 12.6, p. 310); this is most clear in medium-sized muscular arteries (and with TEM).

The sub-endothelial connective tissue and adventitial

#### A. tunica intima

- Endothelium
- Basal lamina
- Subendothelial connective tissue
- Internal elastic membrane

#### B. tunica media

- Smooth muscle
- Elastic fibers, if stained for

C. tunica adventitia (Adventitia being on the outside can be partially torn off - artifact!)

- External elastic membrane
- Connective tissue
- Vasavasorum (vessels of vessel)

## **II. ARTERIES & ARTERIOLES**

A. arterioles When does it stop being a small artery and become an arteriole. Two cells thick is an arteriole; 8 cells thick and above makes it an artery, so what about 3 through 7 layers of smooth muscle?

#### B. muscular arteries

- Tunica intima
- Tunica media
- Tunica adventitia

C. elastic arteries

- Tunica intima
- Tunica media
- Tunica adventitia
- Vasavasorum
- Lymphatic vessels

**III. VEINS**

A. Venules

B. Veins

1. Tunica intima often has valves, but these may be missed even in longitudinal sections

C. Large veins

- Tunica intima
- Tunica media
- Tunica adventitia
- Smooth muscle
- Vasavasorum

**IV. CAPILLARIES** endothelium Recognize

A. With the light microscope - note the very small size capillaries.

1. Endothelium,
2. Fenestrated capillary
3. Continuous capillary

**V. HEART**

A. Endocardium

1. Inner layer

- Endothelium
- Subendothelial layer (sometimes with smooth muscle)

2. Outer, subendocardial layer, only present at certain sites

3. Purkinje fibers in subendocardium

## B. Myocardium

- Cardiac muscle
- Small amount of connective tissue

## C. Epicardium

- Mesothelium (may have been rubbed off)
- Connective tissue, often with fat
- Blood vessels (coronary vessels) and nerves

## **Pathology LAB:**

Webpath images:

Lab (1): 62-97.

137- 144.

Lab (2): 12- 61.

161- 173.