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BLOCK BOOK AND STUDENT’S GUIDE OF **THE CARDIOVASCULAR SYSTEM**

YEAR 1

**College of Medicine**

**Medical Education Department**

**2015 - 2016**

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**College of Medicine**

**Medical Education Department**



**BLOCK BOOK AND STUDENT’S GUIDE**

**OF THE**

**CARDIOVASCULAR SYSTEM**

(Academic year 1436-1437)

**FEMALE GROUP**

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**WELCOME ADDRESS**

**Dear Students,**

**We are pleased to welcome you in the college of Medicine, Cardiovascular Block attachment. We hope you will find this block both useful and enjoyable.**

 **Dr. Mona Soliman**

**GENERAL INFORMATION**

Block Title: **Cardiovascular Block**

Block Code & Number: Cardio113

Credit Hour: 7

Block Duration: 6 Weeks

Block Dates: 21st of February 2016 – 7th April 2016

Block Chairman: Dr. Mona Abdulhafeeth Soliman

**Teaching Staff Contact Information**

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**Problem-Based Learning Cases**

The table below summarizes the PBL cases to be discussed in the Cardiovascular System Block.

|  |  |  |
| --- | --- | --- |
| **Week** | **Case No.** | **Case title** |
| Week 1 | **NO CASE** |
| **Week 2 (Sunday & Wednesday)** | **Case 1** | **“I have to travel to Al-Mozahmia daily”** |
| Week 3 | **NO CASE** |
| Week 4  | **Mid-year vacation** |
| **Week 5 (Sunday & Wednesday)** | **Case 2** | **“Because of sudden severe pain”** |
| **Week 6**  | **Consolidation** |

**Instructions:**

The cases listed above will be discussed by students in their small groups. Each group is about 8 to 12 students. Each case will be discussed in two tutorials, on Sunday and Wednesday. Each tutorial is two hours long.

**Attendance of Small Group Learning tutorials:**

Students must attend all small group learning tutorials. If a student is not well, she needs to provide a medical certificate from their family doctor. If a student fails to attend four tutorials, she might not be allowed to attend the final examination (Dr. Mona Soliman /Prof. Hamza Abdulghani to add re the college regulations).

**Students Roles in Small Group Learning Tutorials:**

The design of the curriculum encourages small group discussion and student-centered learning. To achieve these goals there is a need for establishing good group dynamics, interpersonal skills, and effective communication. These elements will ensure that learning is an enjoyable process and rewarding to each member in the group. Therefore, students play a vital role in making a difference in their groups. To achieve these changes and improve your learning outcomes, We recommend that you use the paper by Professor Samy Azer, titled “Becoming a Student in a PBL Tutorial”, a copy is enclosed in the Appendix. Your continuous reflection on these tips and working on identifying your role in your group will help you in reaching these goals and building up your group.

**Goals and Objectives of the Cardiovascular Block**

By the end of the course the students must be able to:

1. Identify and correlate normal anatomical structures of the cardiovascular system (the heart and the major blood vessels) to their functions.
2. Discuss the pathology and the pathogenesis of common diseases effecting the heart and major blood vessels.
3. Use basic sciences to interpret symptoms and signs of patients with common diseases effecting the cardiovascular system.
4. Identify risk factors for the development of the cardiovascular diseases and discuss the pathology and the pathogenesis atherosclerosis and the development of acute myocardial infraction.
5. Identify approaches used in preventing common cardiovascular disorder,
6. Discuss the pharmacology of drugs used in the management of cardiovascular disorders.
7. Understand the scientific basis of the common investigations used in assessing patients with cardiovascular diseases such as the physiological basic Electrocardiogram (ECG).
8. Demonstrate the ability to take simple history and examine the cardiovascular system and measure the blood pressure, record radial pulse, take temperature and uses the breathing rate in assessing a patient with a cardiovascular disease

**Teaching and Learning Modes:**

In an integrated curriculum like our curriculum, we use a wide range of teaching and learning strategies to ensure that learning meets the different needs of the students. These strategies include:

* Small group discussion
* Lectures
* Student-led seminars
* Practical classes.
* Clinical skills
* Independent learning
* Writing an essay or mini thesis.

**Contents of the Cardiovascular Block**

**Week 1:**

**Theme “Normal Heart , Arrhythmias and Cardiac Cycle”**

**Lectures**

1. Anatomy of the heart (Anatomy)
2. Structure of the cardiac muscle (Histology)
3. Contractile mechanism in cardiac muscle (Physiology)
4. Cardiac electrical activity (Physiology)
5. The Cardiac Cycle 1 (Physiology)
6. The Cardiac Cycle 2 (Physiology)
7. The Electrocardiogram (Physiology)
8. Development of the Heart (Anatomy)
9. Alpha adrenergic blockers (Pharmacology)
10. Beta adrenergic blockers (Pharmacology)
11. Arryhthmias (Cardiac Science Department )
12. Antiarrhythmic drugs 1 (Pharmacology)
13. Antiarrhythmic drugs 2 (Pharmacology)

**Practicals**

1. Anatomy of the heart (Anatomy)
2. Histology of the cardiac muscle (Histology)
3. The electrocardiogram (Physiology)

**Week 2:**

**Theme “Valve disease, The Heart as a Pump and heart failure ”**

**Lectures**

1. Regulation of stroke volume (preload, contractility &afterload) & heart failure (Physiology)
2. Heart sounds and murmurs (Physiology)
3. Venous return & cardiac output (Physiology)
4. Stroke volume (Physiology)
5. Microbiology of myocarditis & pericarditis (microbiology)
6. Lactic acidosis (Biochemistry)
7. Drug therapy for heart failure 1 (Pharmacology)
8. Drug therapy for heart failure 2 (Pharmacology)
9. Rheumatic heart disease (Immunology)
10. Pathology of rheumatic fever, endocarditis and heart valves (Pathology)
11. Risk factors and pathogenesis of atherosclerosis (Pathology)
12. Infective endocarditis (Microbiology)
13. Pathology and pathogenesis of ischemic heart diseases(Pathology)

**Practicals**

1. Heart sounds (Physiology)
2. The recording of Jugular venous and carotid arterial pressures (Physiology)
3. Pathology of cardiovascular disease 1 (Pathology)
4. Pathology of cardiovascular disease2 (Pathology)

**Problem- based learning:** Case # 1

**Week 3:**

 **Theme “Normal Blood Pressure and Hypertention”**

**Lectures**

1. Anatomy of large blood vessels- arteries (Anatomy)
2. Histology of the blood vessels (Histology)
3. Arterial blood pressure (Physiology)
4. Anatomy of large blood vessels- veins (Anatomy)
5. Regulation of blood pressure (Physiology)
6. Treatment of hypertension 1 (Pharmacology)
7. Treatment of hypertension 2 (Pharmacology)
8. Shock (Physiology)
9. Capillary circulation (Physiology)
10. Cholesterol Metabolism (Biochemistry)

**Practicals**

1. Measurement of arterial blood pressure (Physiology)
2. Anatomy and histology of the major arteries and veins (Anatomy & Histology)

 **Week 4:**

 **Mid-year Vacation (2nd semester)**

**Week 5:**

**Theme “Atherosclerosis and Myocardial Infarction “**

**Lectures**

1. Anatomy of the arterial supply and venous drainage of the heart (Anatomy)
2. Coronary circulation (Physiology)
3. Pathology & pathogenesis of hypertension (Pathology)
4. Lipoprotein metabolism (Biochemistry)
5. Thrombolytic therapy (Pharmacology)
6. Lipoprotein and Atherosclerosis (Biochemistry)
7. Pathology of thromboembolism (Pathology)
8. Pathology of vasculitis (Phathology)
9. Drugs for hyperlipidemia 1 (Pharmacology)
10. Drugs for hyperlipidemia 2 (Pharmacology)
11. Oxidative stress (Biochemistry)
12. Biochemical markers of myocardial infarction (Biochemistry)
13. Antianginal drugs 1 (Pharmacology)
14. Antianginal drugs 2 (Pharmacology)

**Practicals**

1. History taking for cardiac disease and clinical examination of the cardiovascular system

***Anatomy of the heart(Anatomy)***

**Objectives:**

By the end of the lecture, the student should be able to:

* Describe the anatomy of the pericardium.
* Describe the surface anatomy of the heart and heart valves.
* Describe the anatomy of the four heart chambers.
* Describe the surfaces and borders of the heart and the relation for each.
* Define the auscultatory points of the heart valves.

**Background and summary:**

The **pericardium** is a fibro-serous sac that encloses the heart and the beginning of the great vessels entering or leaving the heart.

The **heart** is a hollow muscular organ in the middle mediastinum that has apex, base, sternocostal and diaphragmatic surfaces, and right and left borders.

It is formed of four chambers, two atria and two ventricles.

Each **atrium** consists of main cavity and a small out pouching, the auricle.

The **right atrium**, receives the superior and inferior vena cavae and the opening of the coronary sinus.

Many small orifices of small veins also open into the right atrium.

The **right atrium** occupies the right part of the sternocostal surface.

The **left atrium** receives the four pulmonary veins, and occupies most of the base of the heart, which is directed posteriorly.

Each **atrium** empties its blood into the corresponding ventricle through an atrioventricular valve which allows one way passage.

The right atrioventricular valve is called **tricuspid** while the left is called the **mitral.**

The interior of each atrium has a smooth and rough part due to the presence of **musculi pectinati**.

The **ventricular** wall is much thicker than the atrial wall, and that of the left ventricle is three time thicker than the right one.

The ventricular wall shows several projections called **trabeculae carneae**, which form the **papillary muscles** which are connected to the cusps of the mitral and tricuspid valves by chordae tendineae. The ventricular cavities shows rough inflow parte and smooth (infundibulum and aortic vestibule), outflow part.

***Structure of the Cardiac Muscle (Histology)***

**Objectives:**

By the end of this lecture the student should be able to:

* Identify the different layers of the wall of the heart.
* Describe the detailed microscopic structures of the endocardium, myocardium and epicardium.
* Describe the microscopic structure of the cardiac valves.

**Background:**

Studying the microscopic structure of both the wall of the chambers of the heart as well as the cardiac valves is essential for students to interpret the histopathological changes in these structures in different heart lesions.

**Summary:**

The wall of the heart is formed of 3 layers; endocardium, myocardium and epicardium. The endocardium is formed of endothelium and connective tissue layers. The myocardium is formed mainly of cardiac muscle fibers. The epicardium is formed of mesothelium and a connective tissue layer. The cardiac valves are formed of a core of connective tissue covered by single layer of endothelial cells.

***Contractile mechanism in cardiac muscle (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* Define cardiac muscle contractility
* Describe the mechanism of excitation-contraction coupling.
* Understand the mechanism of isovolumetrjc and isometric contraction.
* Factors affecting cardiac contractility.

**Lecture outline:**

Contractility is the force of contraction for a given fiber length and is essential for the pumping action of heart. The mechanism of contraction is known as excitation-contraction coupling. Following are its steps; i) initiated by impulse on the surface of myocardium; ii) the impulse as depolarization wave pass through T -tubule; iii) cause calcium to enter the cell through calcium voltage operated channel; iv) calcium entry trigger the release of calcium from sarcoplasmic reticulum; v) this cause increase in intracellular calcium; vi) calcium then binds to troponin C to start crossbridge cycle; vii) hydrolysis of ATP occurs and provides the energy for sliding of myosin over actin causing contraction; viii) relaxation occurs when calcium is removed from troponin C and is due to re-uptake of calcium by the sarcoplasmic reticulum.

The student with this knowledge will be able to understand inotropic action (+ve and -ve). Also it is essential to understand the mechanism of isovolumetric and isometric contraction.

***Cardiac electrical activity (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* Discuss the cardiac conductive system and its function.
* Describe the action potential of the cardiac muscle and its components.
* Define the refractory period and the excitation-contraction coupling
* Discuss the control of excitation and conduction of the heart.

***Lecture outline:***

The students should understand how membrane potentials are created across semipermeable membrane by transmembrane ion concentration differences. Heart muscle when denervated it still goes on contracting and is known as rhythmicity. This is due to action potential generated by sino-atrial node. Two general types of cardiac action potentials are seen e.g., pacemaker potentials are slow response action potential. The examples are SA node, A V node potentials. The other type is non-pacemaker potential or fast response action potentials e.g., atrial, ventricular action potentials. All are divided into five phases. At the end of lecture student should know the ionic basis of these potentials.

Role of sympathetic and parasympathetic nervous system to be understood on prepotential or pacemaker potential with ionic basis involved in it. Phenomenon of refractory period should also be defined and understood.

***The Cardiac Cycle 1 (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* General principles of the cardiac cycle
* Identify events occurring during cardiac cycle: mechanical, electrical, volume & pressure changes, heart sounds.
* Understand the various phases of the cardiac cycle

**Lecture outline:**

The cardiac events that occur during one heart beat are called the cardiac cycle. Basically two events are occurring e.g., mechanical event and electrical event (ECG). The total duration of the cardiac cycle at rest when heart rate 72 beats per minute is 0.8 sec. If heart rate is fast the cardiac cycle is short. The cardiac cycle is divided into seven phases e.g., 1) atrial systole (0.1 sec): AV valves open, aortic and pulmonary valves closed; 2) isovolumetric contraction: all valves are closed; 3) rapid ejection: aortic and pulmonary valves open, A V valves remain closed; 4) reduced ejection: aortic and pulmonary valves open: AV valves remain closed; 5) isovolumetric relaxation: all valves closed; 6) rapid filling: A V valves open, aortic and pulmonary valves closed; 7) reduced filling: A V valves open, aortic and pulmonary valves closed. In this lecture students are expected to understand the various phases of the cardiac cycle.

***The Cardiac Cycle 2 (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* Identify the systolic and diastolic period.
* Discuss the changes of pressure and volumes in left ventricle, left atrium and the aorta during cardiac cycle
* Explain the meaning of isovolumetric contraction, period of ejection and isovolumetric relaxation.
* Discuss the volume-pressure relationship in the left ventricle.

**Lecture outline:**

In this lecture students should be able to know the pressure and volume changes in the atria, the ventricle and the aorta during each phase ofthe cardiac cycle. Able to define with normal value about: 1) ventricular end-diastolic volume, end-systolic volume, stroke volume, diastolic pressure and peak systolic pressure; 2) aortic diastolic pressure, systolic pressure and pulse pressure. Students should know similarities and differences between mechanical events in the left and right heart pump. It is important that students should note the site of origin of the heart sounds over the ventricular pressure curve. In the end of the lecture student should be able to understand and draw the left ventricular pressure volume loop. This volume-pressure curve demonstrate changes in intraventricular volume and pressure during a cardiac cycle.

***The Electrocardiogram (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* Identify waves of ECG and the physiological cause of each.
* Define the normal intervals and segments.
* Discuss the bipolar and unipolar leads and their locations.
* Discuss the bipolar limb lead and the cardiac axis (Quick method).

**Lecture outline:**

The student should be able to understand the definition of ECG. It is a record of the sum of electrical changes occurring during one heart beat and is propagated to the body surface. This recording is 12-lead electrocardiogram and consists of a combination of bipolar and unipolar records from limb electrode and chest electrode. At the end of lecture students are required to know PQRST waves, P-R interval, QRS wave, T-wave and U-wave if any. Also they should be able to determine the mean electrical axis of the heart. Able to recognized signs of ischemic injury, and infarction in ECG recording. The students should have a full background knowledge of conductive tissue of the heart.

***Arrhythmias (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* identify normal heart rhythm
* Identify the main pathophysiological mechanisms underlying cardiac arrhythmias.
* identify sinus arrhythmias.
* Define different ectopic foci of excitation and the mechanism of re-entry phenomena.
* Identify conduction block.
* Identify electrolyte abnormalities (K+, Ca++).

**Lecture outline:**

The causes of cardiac arrhythmias are due to abnormalities in conductivity or abnormalities in rhythmicity. It can be classified physiologically as:, abnormal rhythmicity of the pacemaker, shift of the pacemaker from the sinus node to another place in the heart, blocks at different points in the spread of the impulse conduction, abnormal pathways of impulse transmission through the heart, premature atrial or ventricular contraction. and atrial fibrillation and ventricular fibrillation.

***Regulation of stroke volume (preload, contractility and afterload) and heart failure (Physiology)***

**Objectives:**

By the end of this lecture the students are expected to:

* Explain how cardiac contractility affect stroke volume.
* Calculate CO using Fick’s principle equation.
* Explain pathophysiology of heart failure and differentiate between left and right failure.
* Explain how the pathophysiology associated with heart failure results in typical signs and symptoms.

**Lecture outline**

The contractility of the myocardium exerts a major influence on SV. Contractility is increased in response to sympathetic stimulation and this is reflected by shifting the pressure volume-loop upward and to the left (positive inotropic effect). Changes in heart rate and rhythm also affect myocardial contractility. Measuring cardiac output using Fick’s principle equation depends on measuring O2 consumption per minute and arterio-venous oxygen difference. Heart failure occurs when the heart loses its function as a pump which may result from ischemia, hypertension, cardiomyopathy,etc… Heart failure could be right or left-sided. There are differences between them regarding causes, effect on body systems and clinical manifestations. The path- physiological mechanisms of heart failure include: systolic dysfunction or diastolic dysfunction.

***Heart sounds and murmurs (Physiology)***

**Objectives:**

By the end of this lecture students are expected to:

* List the major types of normal heart sounds
* Understand the physiological basis for the production of normal heart sounds
* Understand the pathophysiological basis for the production of heart murmurs

**Lecture outline:**

Normal heart sounds and murmur is heard with the help of a stethoscope. Normal heart valves make sound when they close but not when they open. First sound is due to mitral and tricuspid closure and is best heard at the apex (LUBB). Second sound is due to aortic and pulmonary valve closure, it is best heard at the left sterna edge, it is louder than first sound (DUB). Third heart sound is caused by rapid ventricular filling and is a low pitched early diastolic sound. It is a normal finding in children, young adults and during pregnancy. Fourth heart sound is due to flow of blood into the ventricular due to atrial wall contraction hence it is also known as atrial sound. Heart murmurs are produced by turbulent flow of blood across a valve, it is caused by abnormality of the valve but it could be normal known as innocent murmurs. It could be diastolic murmur or systolic murmur, signifying stenosis or regurgitation at the valve.

***Venous return and cardiac output (Physiology)***

By the end of this lecture the students are expected to:

* Define cardiac output, stroke volume, end-diastolic and end-systolic volumes.
* Define physiological conditions affecting CO
* List causes of high and low output pathological states.
* Define venous return and describe factors controlling venous return.

**Lecture outline:**

Cardiac output is the amount of blood pumped by each ventricle per minute (5L/min). It varies physiologically with age, body mass index, physical activity, sleep, meals, pregnancy, etc.. But there are pathological conditions that lead to a significant increase in CO including hyperthyroidism, anemia and conditions decreasing CO as myocardial infarction. CO is well controlled and regulated by Many Factors: venous return, ABP , blood volume and nervous regulation. This lecture will focus on Venous return as an important factor determining CO. Venous return represents the amount of blood returning to the heart per minute. Venous return is controlled by many factors:1) Frank-Starling’s mechanism, 2) mean systemic filling pressure, 3) tissue metabolism, 4) thoracic pump, 5) Gravity, 6) Muscle pump, 7) blood volume.

***Stroke volume(Physiology)***

By the end of this lecture the students are expected to:

* Understand the concept of preload and afterload.
* Determine factors affecting the end-diastolic volume.
* Explains how cardiac contractility affects SV,
* Describe and explain the pressure-volume loop .

**Lecture outline**

This lecture will focus on explaining the concept of preload which is the venous return and how it differs from the afterload which is represented by the ABP and how each affect the CO. The effect of external pressure outside the heart as pericardial pressure, intrathoracic pressure on pre-load is of clinical relevance. Conditions that increase intrathoracic pressure can reduce CO significantly. Because the end-diastolic volume (EDV) represents the volume of the blood filling the ventricle by the end of diastole, it determines the stroke volume (SV) and consequently the CO. therefore factors affecting EDV will be reflected on CO. these factors include; blood volume, venous tone, ……

***Microbiology of myocarditis and pericarditis (Microbiology)***

**Objectives:**

* Describe the epidemiology, risk factor for myocarditis
* Explain the pathogenesis of myopericarditis
* Differential between the various types of myocarditis and pericarditis
* Name various etiological agents causing myocarditis and pericarditis
* Describe the clinical presentation and differential diagnosis of myocarditis and pericarditis
* Discuss the microbiological and non-microbiological methods for diagnosis of myocarditis and pericarditis
* Explain the management ,complication and prognosis of patient with myocarditis and/or pericarditis
* **Take home messages**
* Myocarditis and Pericarditis are an inflammatory disease of myocardium and pericardia respectively.
* Viral infection is the most etiological agents but other infectious and noninfectious agents may also instigate an inflammation of the heart muscle and pericardium.
* Recognizing the clinical symptoms and signs of myocarditis are critical for better management of the patients with these syndromes.
* It is important to send the required laboratory test as soon as possible either microbiological (blood culture, serologies etc.) and non-microbiological tests in addition to radiological and immunological investigation to confirm the diagnosis.
* Supportive care, bed rest and anti-inflammatory are the common approach for management of these patients.
* Specific treatment like antibiotics and antiviral indicated on a case by case basis.

***Lactic acidosis (Biochemistry)***

**Objectives:**

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

* know the conditions associated with excessive blood lactate production
	+ recognize the importance and consequences of lactate production
	+ Identify fates of lactate
	+ relate lactic acidosis Vs skeletal muscle cramp
	+ evaluate lactic acidosis as a medical emergency

**Background:**

Normally, lactate is released into the blood as an end product of consumption of glucose by anaerobic glycolysis by exercising skeletal muscle and by cells that lack mitochondria e.g., RBCs. This lactate is taken up by the liver and converted back into glucose, which is released back into the circulation (Cori cycle).

When there is a collapse of circulatory system, for example shock, there will be failure to bring adequate amount of oxygen to tissues. The results will be impaired oxidative phosphorylation and ATP synthesis. To survive the cells use anaerobic glycolysis as a backup system for generating ATP, with the production of lactate as an end product.

**Key principles to be discussed:**

Pyruvate is the end product of glycolysis in cells with mitochondria and an adequate supply of oxygen (aerobic glycolysis). In absence of oxygen, there is, however, obligatory reduction of pyruvate into lactate to allow oxidation of NADH into NAD+ and the continuation of anaerobic glycolysis (refer to glycolysis). The production of even meager amounts of ATP by anaerobic glycolysis may be life-saving during the period required to reestablish adequate blood flow to the tissues.

Increased lactate production might be congenital or acquired. Pyruvate dehydrogenase enzyme deficiency causes congenital lactic acidosis with developmental defects especially of the brain, muscular spasticity and early death. Circulatory system collapse, for example shock, myocardial infarction, pulmonary embolism and uncontrolled hemorrhage, results in acquired increase of blood lactate level and lactic acidosis.

Measuring of blood lactate level is a useful indicator for the presence and severity of shock and to monitor the patient’s recovery and its response to treatment.

Excess lactate production in the skeletal muscle, for example during severe exercise, results in muscle cramps (compare lactic acidosis to muscle cramps).

**Take home messages:**

* Excess lactate production is a life-saving backup system for production of small, but important and critical amount of ATP for tissues exposed to insufficient blood supply.
* Lactic acidosis (increased blood lactate level) may be congenital or acquired.
* Lactic acidosis is a medical emergency that requires intensive therapy (ICU).
* Measurement of blood lactate in patients with circulatory system collapse is a useful prognostic marker for monitoring of patient’s recovery.

**Further reading (Prescribed book):**

Lippincott’s Illustrated Reviews; Biochemistry, 4th edition, Editors: Pamela Champe, Richard A. Harvey, 2008

**Keywords**:

Lactate, lactic acidosis, circulatory system collapse, anaerobic glycolysis, muscle cramps

***Rheumatic heart disease (Immunology)***

**Objectives:**

* To understand basis of rheumatic fever as an immunologically mediated late complication of Streptococcal infection
* To know that autoimmunity results from production of cross reacting antibodies against Streptococcal antigens
* To describe rheumatic heart disease as one of the several manifestations of rheumatic fever
* To know the signs, symptoms, pathogenesis, treatment and prophylaxis of rheumatic heart disease

**Background:**

Sore throat is a common condition frequently observed in adults and children. Detection of the microorganisms involved in throat infections is important as frequent streptococcal throat infections may predispose an individual especially a child to rheumatic fever. Autoimmune process resulting as a late complication of streptococcal sore throat can cause severe damage to heart valves in children and is known to cause a significant mortality and morbidity. Development of symptoms is usually associated with an advanced valvular damage often requiring valve replacement. The level of incapacity in patients with rheumatic heart can be tremendous and in order to avoid it the main emphasis is currently been focused on the prevention or prophylaxis of the disease.

**Key principles to be discussed:**

Rheumatic fever being an autoimmune disease resulting from repeated throat infections with streptococcus may involve vital organs such as the heart and the kidneys. The key issues to be discussed are:

* Importance of frequent attacks of streptococcal sore throat.
* Awareness of the late complications of such infections
* Basic mechanisms involved in the disease process
* Clinical signs and symptoms associated with rheumatic fever and the associated complications
* Preventive or prophylactic measures

**Take home message**

* Awareness of the importance of streptococcal sore throat with regards to development of rheumatic fever
* Correct diagnosis and management of rheumatic fever
* Importance of prolonged antibiotic cover for prevention of future attacks.

**Further reading:**

Kuby Immunology

***Pathology of rheumatic fever, endocarditis and heart valves (Pathology)***

**Objectives:**

At the end of this lecture, the students should be able to:

* Understands the clinicopathological features of rheumatic heart disease which is a major cause of acquired mitral and aortic valve diseases in the Kingdom of Saudi Arabia.
* Know the pathological causes and pathophysiological consequences of stenosis and incompetence of all the cardiac valves but particularly the mitral and aortic valves.
* Understands the pathology of infective endocarditis so as to be able to identify patients at risk and when appropriate ensure prophylactic treatment is given.

**Key principles to be discussed:**

* Pathology and manifestations of rheumatic heart disease as a major cause of valvular diseases in the Middle East and Saudi Arabia.
* Complications of rheumatic heart disease including atrial fibrillation, valvular and atrial thrombus formation with systemic embolism, cardiac failure and infective endocarditis.
* Infective endocarditis: predisposing factors, clinical acute and subacute forms, common pathogenic bacteria in IE and complications including valve perforation, thrombosis and septic embolization of the vegetations.
* Causes and consequences of valvular heart disease with special emphasis on aortic and mitral valve including "floppy or prolapsed" mitral valve.

***Infective Endocardites (Microbiology)***

**Objectives:**

By the end of this lecture, the student should be able to:

* Define infective endocarditis
* Recognize that bacteria like *Streptococcus viridans* and *Staphyloccocus aureus* are the most important causes of infective endocarditis.
* Know that the disease can present as acute or sub-acute bacterial endocarditis.
* Recognize the comments clinical presentations of infective endocarditis i.e. fever as the disease is one of the most important causes of pyrexia of unknown angiun (P.U.O)
* Know the bacterial factors and host factors that affected the severity and outcome of the disease.
* Recognize the sources of the bacteria that causes bacterimia and then infective endocarditis.
* Know the pathogenesis of the infective endocarditis. The formation and presence of bacterial vegetation.
* Know the predisposing factors for infective endocarditis e.g previous congenital heart lesions, rheumatic heart lesions presence of prosthetic valve as well as intravenous drug users.
* Know the common bacterial causes and relationship of different bacteria causes to different clinical conditions.
* Know the diagnostic methods for endocarditis including
1. clinical features
2. Laboratory tests mainly blood culture
3. Echo cardiography for presence of bacterial vegelations.
* Know the different anti- microbial used for different types of organism.
* Know the possible complications of infective endocarditis.

**Back ground :**

Infective endocarditis is the microbial infection of the endothelial surface of the heart. The valves of the heart are the most affected structures, but the infection can also occur at a septal defect, the chordate tendons and other lesions like patent ductus arteriosus, arteriovenous fistula and coartication of the aorta. The syndrome is caused mainly by bacterial so it is sometimes called *Bacterial endocarditis* which can be divided into sub-acute or acute bacterial endocarditis on the basis of clinical presentation. It is important to recognize that infective endocarditis is one of the causes of the syndrome pyrexia of unknown origin (P.U.O). Some predisposing factors increase the chance of having endocarditis e.g. in intravenous drug users or prosthetic valve etc. The main laboratory test for diagnosis is blood culture. Mortality before use of antibiotic was high and it is also considerable even with use of antibiotics.

**Key principle points to be discussed**

Definition of infective endocarditis as the infection of the cardiac endocardium mainly affecting the valve, but can also affect septal defects, patent ducts arteriosus and coarcitation of the aorta.

The use of clinical presentation to know the clinical divisions of the syndrome into acute and sub-acute will addressed.

Pathogenis of the condition shall be discussed and clarified as the characteristic lesion is the vegetation including bacteria and other constituents. Pathogenesis depends on the:

1. Endothelial damage due to blood flow dynamics.
2. Bacterimia originating from the month, GTT or urogenital.
3. Bacterial factors: ability to adhere to the thrombus formed
4. Some bacteria e.g. staphylococcus can infect intact endothelium.

**The underlying or predisposing factors:**

1. Heart disease either congenital heart disease, rheumatic heart disease degenerative heart disease or syphilic heart disease, intravenous drug user can also predispose to infective endocarditis.

Clinical features, could be acute or sub-acute the most important general feature are fever which goes on for more that 2 weeks (P.U.O) should always suggest the possibility of infective endocarditis. Signs can include splenomegaly, petechiae, clubbing, retinal haemorrhage and maybe haematuria.

**Cardiac signs:** when valve are affected like the mitral valve which is the one most affected, there can be heart murmurs.

**Signs of congestive heart failure which can also happen as a complication**

**Diagnosis:** Non-specific finding, high ESR, C-reactive protein which manifest tissue destruction and bacterial infection respectively, peripheral high white blood count.

**Specific:** the most important is blood culture where two samples of blood culture are taken.

**Echocardiography:** can also help in detecting vegetation in the endocardium.

**Treatment:** the regimen of antibiotic drugs for different organism will be discussed in details stressing on the fact that therapy depends on the antimicrobial susceptibility of the causative bacterial agent.

Blactam antibiotics like penicillin can be used in case of *Streptococcus viridians* endocarditis. Penicillin plus an aminoglycoside can be used as combinedtherapy.

Prophylaxis against endocarditis with use of penicillin in high risk patient shall also be dicussed.

**Causative Bacterial agent:**

The full details of bacteria causing endocarditis will be discussed in the following theme:

* *Streptococcus viridian* e.g. *S. Mutans* and *S. Sanguis* are the main causes in Rheumatic heart damaged valve.
* Staphylococci are causes of infection in prosthetic or intravenous drug addicts.
* *Enterococcus faecalis* in elderly men with prostatic hyperplasia who are catherized.

The topic of *Hacek endocarditis* group of organism or cause of will also be discussed.

**Diagnosis depends on :**

1. Clinical features
2. Laboratory test that mainly include blood culture, ESR, C-reactive protein and high white cell count.
3. Echocardiography to look for vegetation.

Therapy includes beta laclam alone or in combination with aminogycosal vancomycin can be used in gram positive organism e.g. MRSA and VRF.

**Take away home message:**

* Infective endocarditis means infection of the heart endocardium that affects mainly damaged valves due to congenital rheumatic and other congenital like skeptical defects can also be infected.
* Most causative organisms are *Streptococcus viridian, Staphylococci* and maybe Enterococci.
* Other organism includes Hacek group, chlamydia, coxiellia and fungi.
* Presenting clinical features include :
1. Fever in most cases.
2. Heart murmurs.
3. Peripheral signs like emobic phenomenons, petechiae, hematuria and finger clubbing.

***Pathology and pathogenesis of ischemic heart diseases(Pathology)***

***Objectives:***

At the end of these two lectures, the student should:

* Understand the pathogenesis and clinical consequences of atherosclerosis.
* Be able to discuss pathology and complications of ischaemic heart diseases with special emphasis on myocardial infarction.
* Know how lifestyle modifications can reduce the risk of ischaemic heart disease.

**Key principles to be discussed:**

* Risk factors of atherosclerosis.
* Pathogenesis of the fibrolipid atherosclerotic plaque.
* Clinical complications of atherosclerosis.
* Commonest sites for the clinically significant coronary atherosclerosis.
* Macroscopic and microscopic changes in myocardial infarction.
* Biochemical markers of myocardial infarction.
* Complications of myocardial infarction: immediate and late.

***Anatomy of large blood vessels- arteries (Anatomy)***

**Objectives:**

At the end of the session the student should be able to:

* Define the systemic and pulmonary circulation.
* Describe the origin, course, parts and branches of aorta.
* Describe the main arteries of the head & neck.
* Describe the main arteries of the thorax.
* Describe the main arteries of the abdomen & pelvis.
* Describe the main arteries of the limbs.
* Describe the pulmonary circulation.

**Summary**

* Two types of circulation: Systemic & Pulmonary
* The arteries of the body are divided into:
	+ Systemic arteries, which carry oxygenated blood from the heart to the whole body
	+ Pulmonary arteries, which carry deoxygenated blood from the heart, to the lungs
* Aorta: Major artery of the systemic circulation
	+ Origin: Left ventricle
	+ Part: ascending aorta, arch, descending aorta (thoracic & abdominal)
	+ Branches:
	+ Ascending aorta: Right & left coronary arteries
	+ Arch of aorta: brachiocephalic, left commom carotid. Left subclavian
	+ Descending aorta--Thoracic part: intercostal, bronchial, esophageal and phrenic arteries
	+ Descending aorta—abdominal part: celiac trunk, superior and inferior mesenteric, renal, gonadal, inferior phrenic, lumbar and common iliac arteries
* Arteries of the head & neck: Common carotid, external and internal carotid
* Arteries of the thorax: intercostal arteries
* Arteries of the abdomen& pelvis: celiac trunk, superior and inferior mesenteric, renal, gonadal, and internal iliac arteries
* Arteries of the upper limb: subclavian, axillary, brachial, radial, ulnar, palmer arches, digital arteries
* Arteries of the lower limb: External iliac, femoral, popliteal, tibial, dorsalis pedis, digital arteries

***Histology of the blood vessels (Histology)***

**Objectives:**

By the end of this lecture the student should be able to describe the microscopic structures of the wall of:

* Large elastic arteries, e.g. aorta.
* Medium-sized arteries (muscular arteries).
* Arterioles.
* Different types of blood capillaries.
* Medium-sized veins.
* Large veins.

**Background:**

Studying the microscopic structure of the wall of different blood vessels enable the students to understand the physiological processes occurring through the walls of blood vessels and interpret the histopathological changes confronted in the different vascular diseases.

**Summary:**

The microscopic structure of the wall of most of the blood vessels is formed of 3 coats; tunica intima, tunica media and tunica adventitia. The structural differences between the different types of blood vessels are essential for these vessels to adapt their specific functions. Similarly, the wall of blood capillaries is significantly reduced to adapt their functions. The wall of most blood vessels is formed of:

* Tunica intima: endothelium, subendothelial connective tissue and internal elastic lamina.
* Tunica media: contains smooth muscle cells, fenesterated elastic membranes, collagen fibers and reticular fibers.
* Tunica adventitia: formed of a connective tissue layer.

***Arterial blood pressure (Physiology)***

By the end of this lecture the students are expected to:

* Understand the concept of mean blood pressure, systolic, diastolic, and pulse pressure.
* Calculate mean BP
* Understand normal variations in ABP.
* Understand the relationship between CO, BP and total peripheral resistance.
* Describe and understand factors determining blood pressure.

**Lecture outline:**

Arterial blood pressure is the force exerted on the lateral wall of the arteries while blood is flowing through them. The maximum pressure in the arteries during systole is the systolic pressure and during diastole represents the diastolic. Blood pressure is affected by age, emotions, physical and mental stress, sleep, meals, pregnancy. Mean Bp= diastolic+1/3 pulse pressure. Blood pressure is determined by: 1) Cardiac output, 2) Total peripheral resistance, 3) Blood viscosity, and 4) Blood volume.

***Anatomy of large blood vessels- veins (Anatomy)***

**Objectives:**

At the end of the lecture the student should be able to:

* List the name of the major veins of the body.
* Describe the anatomy of each major vein regarding the surface anatomy, beginning, termination, important relations and important tributaries.
* Correlate between the anatomy of major veins and arteries of the body.

**Background:**

The right atrium of the heart receives the openings of three veins:

* Superior vena cava that drains the upper half of the body, including head, neck, upper limbs & thorax.
* Inferior vena cava that drains the lower half of the body, including abdomen, pelvis, perineum & lower limbs.
* Coronary sinus that drains the heart itself.

Knowing the anatomy of the superior and inferior venae cavae, as well the major veins draining into each of them is essential for medical students in order to understand the venous circulation of the body and to correlate between the anatomy of major veins and major arteries.

**Summary:**

 Regarding ***the superior vena cava***, it is formed by the union of the two brachiocephalic veins, opposite the sternal end of the right first costal cartilage. It runs a vertical course in the superior and middle mediastinum. It receives the azygos vein draining most of the thoracic region. It ends by opening into the right atrium, opposite the sternal end of the right third costal cartilage. Each ***brachiocephalic vein*** is formed by the union of the corresponding subclavian and internal jugular veins, behind the sternal end of the clavicle. Its important tributaries are the inferior thyroid and vertebral veins. ***The subclavian vein***, a continuation of axillary vein, opposite the outer border of the first rib, runs a transverse course in the posterior triangle of the neck, behind the clavicle and receives the external jugular vein draining the superficial structures in the head and neck. ***The internal jugular vein***, a continuation of sigmoid sinus at the jugular foramen, runs a vertical course in the neck, inside the carotid sheath. In addition of draining the cranial cavity, the internal jugular vein receives many veins in the neck, namely common facial, lingual, pharyngeal, superior & middle thyroid veins.

 Regarding ***the inferior vena cava***, it is formed by the union of the two common iliac veins, opposite the fifth lumbar vertebra. It ascends in the abdomen, to the right side of the abdominal aorta, in front of lumbar vertebrae. It receives the lumbar, renal, right suprarenal, right gonadal, phrenic and hepatic veins. It passes to the thorax through the vena caval opening of the diaphragm, opposite the eighth thoracic vertebra It ends by opening into the right atrium, opposite the sternal end of the right sixth costal cartilage. Each ***common iliac vein*** is formed by the union of the corresponding external and internal iliac veins. ***The external iliac vein***, a continuation of the femoral vein, behind the inguinal ligament, drains most of the lower limb and anterior abdominal wall (through the inferior epigastric vein). ***The internal iliac vein*** is formed by the union of many venous plexuses draining the pelvic viscera, as well as veins draining the posterior abdominal wall, pelvic wall, perineum and gluteal region of the lower limb.

***Regulation of blood pressure (Physiology)***

By the end of this lecture the students are expected to:

* List the short, intermediate and long-term mechanisms regulating ABP.
* Understands how baroreceptors prevent significant changes in BP and why they act for short-term control.
* Understand why chemoreceptors work under emergency conditions to control BP.
* List the anatomical components of baroreceptors/chemoreceptors.
* Explains the role of the kidney in the long-term regulation of ABP.

**Lecture outline:**

Arterial blood pressure is regulated by many mechanisms ranging from short-term (nervous) , intermediate and long-term (Role of the kidney). The short-term mechanisms depends on many reflexes:1) the baroreceptor reflex, 2) the chemoreceptor reflex, and the 3) CNS-ischemic response. The baroreceptor reflex is involved in minute by minute regulation of ABP and because baroreceptors are rapidly adapted they are involved in the short-term regulation of ABP. The other two reflexes works during emergency situations. These reflexes work through connections to the CNS, the cardiovascular centers in the medulla oblongata. These centers control heart rate, cardiac contractility and blood vessel tone through efferent outputs. The intermediate –term mechanism depends on capillary fluid shift between vascular compartment and ECF compartment. The long-term regulation of arterial blood pressure depends on the kidney which regulates fluid and salt excretion. Pressure-diuresis and pressure-natriuresis are two mechanisms. Hormones included are the rennin-angiotensin-aldosterone system and atrial natriuretic peptide.

***Shock (Physiology)***

**Objectives:**

By the end of this lecture the students are expected to:

* Define circulatory shock.
* List types and causes of shock.
* Understand the body compensatory mechanisms during the reversible phase of hemorrhagic shock.
* Understands the mechanisms responsible for the irreversible phase of hemorrhagic shock.

**Lecture outline:**

Circulatory shock indicates Inadequate tissue perfusion with relatively or absolutely inadequate cardiac output. There are many classifications of shock but this one could help in understanding the cause and mechanisms involved. The types and causes include: 1) Hypovolemic shock, 2) Distributive shock., 3) Cardiogenic shock, and 4) Obsteructive shock. The hypovolemic shock could be due to blood loss, fluid loss or plasma loss. Blood loss as in accident or surgery can result in hemorrhagic shock depending on amount of blood lost. As it is useful to consider the effect of hemorrhage in details this lecture. Hypotension, Rapid thready pulse., Cold, pale skin, Intense thirst, Rapid respiration, Restlessness are manifestations of shock during early phase and during that phase physiological mechanisms work to prevent excess drop in blood pressure as baroreceptor reflex, chemoreceptor and CNS ischemic response. Vasoconstriction of cutaneous blood vessels occurs due to activation of sympathetic nervous system. The irreversible phase occurs because of delayed management or severe blood loss. Series of positive feedback mechanisms mediates this phase.

***Cholesterol metabolism (Biochemistry)***

**Objectives:**

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

* know the structure and functions of cholesterol
* relate hypercholesterolemia and atherosclerosis
* define cholesterol biosynthesis and its regulation
* list the factors that decrease blood cholesterol level
* identify bile salt functions and cholesterol execution

**Background:**

Cholesterol is a structural component of all cell membranes and of the outer layer of the plasma lipoproteins. It is synthesized in many tissues and is the precursor of all other steroids in the body including corticosteroids, sex hormones, bile acids and bile salts, and vitamin D. Cholesterol is a major constituent of gallstones and is a very important factor in the genesis of atherosclerosis.

**Key principles to be discussed:**

During this lecture the students will become familiar with the characteristic steroid alcohol of animal tissues- cholesterol. The students will know the major sources of cholesterol from diet and from extrahepatic tissues. They will also develop an understanding of the structure of cholesterol and its ester and how it is synthesized and converted into bile acids and bile salts. The ring structure of cholesterol cannot be metabolized to CO2 and H2O in humans. Rather, the intact sterol nucleus is eliminated from the body by conversion to bile acids and bile salts, which are excreted in the feces, and by secretion of cholesterol into the bile. Bile salts are important in the digestion and absorption of lipids from the intestine. Students will also get an understanding of the action mechanism of cholesterol lowering drugs.

**Take home messages:**

* The liver plays a central role in the regulation of the body's cholesterol homeostasis
* Cholesterol synthesis occurs in the cytoplasm, with enzymes in both cytosol and the membrane of the endoplasmic reticulum
* HMG-CoA reductase, is the most important enzyme catalyzing the rate-limiting step in cholesterol biosynthesis
* Bile salts are important in the digestion and absorption of lipids from the intestine
* Bile salts together with phospholipids are important to keep cholesterol soluble in bile

**Further reading (Prescribed book):**

Illustrated Reviews of Biochemistry by Lippincott 4th edition.

**Key words:**

Cholesterol, cholesteryl ester, HMG CoA, bile salts and acids, statins

***Anatomy of the arterial supply and venous drainage of the heart (Anatomy)***

**Objectives:**

By the end of the lecture, the student should be able to:

* Describe the origin, course and distribution of the two coronary arteries.
* Describe the venous drainage of the heard and the coronary sinus.
* Identify which part of the cardiac muscle would be affected in case of occlusion of each of the coronary arteries and its main branches.

**Background and summary:**

The heart muscle receives its arterial supply from the two coronary arteries which arise from the ascending aorta just above the aortic valve.

Both coronary arteries are distributed over the surface of the heart lying within the subpericardium to supply the pericardium.

Both coronary arteries lie in the atrioventricular groove (coronary Sulcus).
While their branches lie in the *interventricular* grooves.

*Right Coronary Artery:*

It arises from anterior aortic sinus, at the root of ascending aorta

It passes forwards between right auricle & pulmonary trunk.

 Then it passes downwards and to the right in coronary groove to reach the lower margin of the heart

 Then it curves backward to runs in the posterior part of the coronary sulcus.

Termination: By anastomosis with the *circumflex* branch of left coronary.

 Left coronary artery
 Origin: From Left posterior Aortic sinus, (larger than the Rt. Coronary)

Lies between left auricle and root of pulmonary trunk, till the anterior interventricular groove, where it divided into:

1- Circumflex artery.

2- Anterior interventricular a.

Branches:

1- Anterior interventricular.

2- Circumflex artery.

3- Diagonal artery.

4- Left marginal branch.

***Coronary circulation (Physiology)***

**Objectives:**

By the end of this lecture the student should be able to:

* define autoregulation and its mechanism (metabolic & myogenic).
* identify the vasodilator metabolites.
* list vasodilator and vasoconstrictor agents and those secreted by the endothelium.
* identify vessels supplying the heart (coronaries).
* Understand and list factors affecting coronary blood flow (cardiac cycle, autoregulation, chemical factors & neural factors.

**Lecture outline:**

Microcirculations are tightly regulated by mechanisms, an important one is the autoregulation, which means ability of the tissues to regulate their own blood flow according to their metabolic needs. This mechanism works well in the heart, kidney,… There are substances that either works systemically or locally to control blood flow or blood pressure. The local ones include, histamine, nitric oxide, prostaglandins, endothelin,.. while the circulating agents include: angiotensin II, vasopressin, adrenaline…

Coronary circulation is one of special circulations as it supplies the heart and it is well controlled by specific factors: 1)Pressure gradient during the cardiac cycle and aortic pressure, 2) Autoregulation, 3) chemical factors, and 4) neural factors. When flow through the coronaries is reduced to a point that the myocardium becomes hypoxic, substance 'P' accumulates and angina develops.

***Pathology and pathogenesis of hypertension (Pathology)***

**Objectives:**

At the end of this lecture, the students should be able to:

* Know the aetiology, risk factors and complications of hypertension, so as to be able to identify patient risk factors amenable to treatment by lifestyle modification, and to investigate patients appropriately for causes of secondary hypertension.

**Key principles to be discussed:**

* Raised systemic blood pressure is a major cause of morbidity and mortality.
* Hypertension can cause or contribute to: atherosclerosis, left ventricular hypertrophy, chronic renal failure, cerebrovascular disease and retinopathy.
* Normal values for blood pressure.
* Causes of secondary hypertension.
* Genetic and environmental factors contributing to the aetiology of essential hypertension.
* Pathology of blood vessels (blood vessels changes) in both primary and secondary hypertension.

***Lipoprotein metabolism (Biochemistry)***

***Lipoprotein and Atherosclerosis (Biochemistry)***

**Objectives:**

Upon completion of these two lectures, the students should be able to:

* know the composition of plasma lipoproteins (chylomicrons, VLDL, LDL and HDL).
* recognize the metabolism and functions of plasma lipoproteins
* identify the functions of apolipoproteins
* outline the clinical aspects of abnormal lipoprotein metabolism.

**Background:**

The plasma lipoproteins are spherical macromolecular complexes of lipids and specific proteins (apolipoproteins or apoproteins). The lipoprotein particles include chylomicrons, very-low- density lipoproteins (VLDL), low – density lipoproteins (LDL), and high- density lipoproteins (HDL). They differ in lipid and protein composition, size, density, and site of origin. Lipoproteins function both to keep their component lipids soluble as they transport them in the plasma and to provide an efficient mechanism for transporting their lipid contents to (and from) the tissues.

**Key principles to be discussed in the first lecture:**

Lipoproteins are composed of neutral lipid core (containing triacylglycerol, and cholesteryl esters) surrounded by a shell of amphipathic apolipoproteins, phospholipids, none esterified cholesterol). These amphipathic compounds are oriented so that their polar portions are exposed on the surface of the lipoprotein, thus making the particle soluble in aqueous solution. The plasma lipoproteins can be separated by either ultracentrifugation or electrophoresis. The apolipoproteins associated with lipoprotein particles have a number of diverse functions, such as providing recognition sites for cell – surface receptors, and serving as activators or coenzymes for enzymes involved in lipoprotein metabolism. Some of the apolipoproteins are required as essential structural components of the particles and cannot be removed, whereas others are transferred freely between lipoproteins.

The chylomicrons are formed by the intestinal mucosal cells and they transfer dietary lipids from the intestine to the tissues. VLDLs are produced in the liver and their function is to transport triacylglycerol from the liver to the peripheral tissues. There, the triacylglycerol is degraded by lipoprotein lipase and thus converting VLDC to LDL. Intermediate sized particles, the intermediate – density lipoproteins (IDL) or VLDL remnants are observed during this transition.

**Key principles to be discussed in the second lecture:**

 LDL particles contain much less triacylglycerol than their VLDL precursors, and have a high concentration of cholesterol and cholesteryl esters. The primary function of LDL particles is to provide cholesterol to the peripheral tissues (or return it to the liver). They do so by binding to cell surface membrane LDL receptors. A deficiency of functional LDL receptors causes a significant elevation in plasma LDL and, therefore, of plasma cholesterol. Patients with such deficiencies have type II hyperlipidemia (familial hypercholesterolemia) and premature atherosclerosis.

HDL particles are formed by the liver and intestine. HDL is a reservoir of apolipoproteins for the other lipoprotein particles. HDL particles are excellent acceptors of unesterified cholesterol (both from other lipoprotein particles and from cell membrane) and they esterify cholesterol by the enzyme lecithin: cholesterol acyltransferase (LCAT). HDL particles are responsible for the reverse cholesterol transport from the peripheral tissues to the liver for excretion. This is the basis for the inverse relationship between plasma HDL concentration and atherosclerosis, and for the HDL’s designation as the “good” cholesterol carrier.

**Take home messages:**

* Plasma lipoproteins are important for the transport of lipids from one tissue to the other.
* Plasma lipoproteins can be separated by either ultracentrifugation or electrophoresis.
* Plasma lipoproteins include: chylomicrons, VLDL, LDL, and HDL
* Chylomicrons are formed in the intestine and they transfer dietary lipids to the tissues.
* VLDL is formed in the liver and it transfers the triacylglycerol synthesized by the liver to the peripheral tissues.
* LDL is formed in the blood from VLDL. It transfers the cholesterol synthesized by the liver to the peripheral tissues.
* HDL is formed in the liver and intestine. It is important for the reverse transfer of cholesterol from the tissues to the liver.

**Further reading (Prescribed book):**

Lippincott Illustrated Reviews, Biochemistry (4th edition)

**Keywords:** Lipoproteins, chylomicrons, lipoprotein lipase, VLDL, LDL, HDL.

***Pathology of vasculitis (Pathology)***

**Objectives:**

At the end of these two lectures, the students should be able to:

1. Understand the basic pathology of thrombogenesis and the risk factors for development of deep vein thrombosis.
2. Know the types of embolus than can occur and the pathology of pulmonary embolism.
3. Know the common causes of vasculitis with special emphasis on the clinic-pathological features and mechanism of:
4. Giant cell arteritis.
5. Polyarteritis nodosa.
6. Wegener's granulomatosis.
7. Cutaneous hypersensitivity vasculitis and Henoch Schonlein purpura.

***Key principles to be discussed:***

1. Factors involved in thrombogenesis: vessel wall abnormality, vascular stasis or turbulent flow and increased blood coagulability.
2. Causes of thrombus and embolism formation.
3. Predisposing factors for deep vein thrombosis.
4. Pathology of pulmonary thrombo-embolism.
5. Bried description of other forms of emboli like: fat embolism, air embolism, atherosclerotic plaque embolism, amniotic fluid embolism, nitrogen embolism and infective endocarditis.
6. Pathology of vasculitis: giant cell arteritis, polyarteritis nodosa, Wegener's granulomatosis, Henoch-Schonlein purpura and cutaneous hypersensitivity vasculitis.

***Oxidative stress (Biochemistry)***

**Objectives:**

Upon completion of these two lectures, the students should be able to:

* know the reactive oxygen species (ROS)
* define the sources of ROS
* recognize the toxic effects of ROS
* identify the protective mechanisms against ROS
* describe glucose 6-phosphate dehydrogenase deficiency

**Background:**

Reactive oxygen species (ROS) is a family of toxic compounds that are formed of hydrogen peroxide (H2O2), superoxide and hydroxyl radical. These compounds are formed continuously as by - products of aerobic metabolism, through reactions with drugs and environmental toxins, or when the level of antioxidants is diminished, all creating the condition of oxidative stress.

**Key principles to be discussed in the first lecture:**

The highly reactive oxygen intermediates can cause serious chemical damage to DNA, proteins, and unsaturated lipids, and can lead to cell death. These reactive oxygen species have been implicated in a number of pathologic processes including reperfusion injury, cancer, inflammatory disease and aging. The role of ROS in the development of atherosclerosis is important. The cell has several protective mechanisms that minimized the toxic potential of these compounds. One of these mechanisms is the presence of enzymes that catalyze antioxidant reactions e.g. superoxide dismutase, catalase, glutathione reductase and glutathione peroxidase.

**Key principles to be discussed in the second lecture:**

The second protective mechanism against ROS is the antioxidant chemicals. A number of intracellular reducing agents, such as ascorbate, vitamin E, and B-carotene, are able to reduce and, thus, detoxify oxygen intermediates in the laboratory. Consumption of foods rich in these antioxidant compounds has been correlated with a reduced risk for certain types of cancers, as well as decreased frequency of certain other chronic health problems. The sources and mode of action of these antioxidant agents are important topics to study.

Glucose 6-phosphate dehydrogenase (G6PD) deficiency is an inherited disease characterized by hemolytic anemia caused by the inability to detoxify oxidizing agents. G6PD deficiency is the most common disease-producing enzyme abnormality in humans, affecting more than 200 million individuals worldwide. G6PD deficiency is x-linked, and is, in fact, a family of deficiencies caused by more than 400 different mutations in the gene coding for G6PD. Only some of these mutations cause clinical symptoms. The pathogenesis and precipitating factors for hemolysis and the treatment of the disease are important topics to discuss.

**Take home messages:**

* Reactive oxygen species (ROS) is a family of toxic compounds that are formed of H2O2, superoxide ions and hydroxyl radical.
* ROS can cause cell death and a number of diseases e.g. cancer, inflammatory disease and cardiovascular diseases.
* The cell has several protective mechanism against ROS.
* The first protective mechanism is by the enzymes: superoxide dismutase, catalase, glutathione reductase and glutathione peroxidase.
* The second protective mechanism is by the antioxidant chemicals: ascorbate, vitamin E and β-carotene.
* G6PD deficiency is an inherited disease characterized by hemolytic anemia caused by the inability to detoxify oxidizing agents.

**Further reading (Prescribed book):**

Lippincott Illustrated Reviews, Biochemistry (4th edition)

**Key words:** ROS, H2O2, superoxide, hydroxyl radical, superoxide dismutase, catalase, glutathione peroxidase, ascorbic, vitamin E, β-carbonate, G6PD deficiency, hemolytic anemia.

***Biochemical markers of myocardial infarction (Biochemistry)***

**Objectives:**

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

* Recognize the importance of a need for specific & sensitive markers for diagnosis of myocardial infarction of all stages.
* Know the standards that should be considered for recruiting a biochemical marker for diagnosis & follow up of myocardial infarction
* Be aware of recommendations highly suggested for using biochemical markers for diagnosis & follow up of myocardial infarction.
* List the currently used biomarkers for diagnosis of myocardial infarction and know the advantages & limitations of use of each.

**Background:**

Myocardial infarction is considered one of the most important causes of sudden death worldwide. According to recommendations of the WHO, three items have to be considered for diagnosis and follow up of cases of myocardial infarction, clinical examination, ECG and biomarkers. The proper use of the appropriate biomarkers for diagnosis is of great help in diagnosis and follow up and that requires the utilization of the guidelines set for this purpose.

**Key principles to be discussed:**

The lecture begins with a short introduction to myocardial infarction in respect of causes, brief discussion clinical manifestations & diagnosis. Then, mechanism of release of myocardial markers into plasma is discussed in brief. The criteria for ideal biochemical markers for diagnosis & follow up of myocardial infarction is discussed.

Detailed review of individual markers was introduced: cardiac Enzymes (CK-MB activity & CK-MB mass) and cardiac proteins: (cardiac troponins & myoglobin) with respect to advantages and disadvantages of each marker. Finally, a guidelines and instructions for the use of biomarkers in myocardial infarction is presented as a conclusion at the final stage of the lecture with some interaction with the students.

**Take home messages:**

* The student has got to know that 100% ideal biochemical marker for diagnosing and follow up of myocardial infarction is not available yet.
* He/she should have the ability to evaluate new markers in the future by exploiting the criteria that have been discussed in the lecture.

**Further reading (Prescribed book):**

Bishop Duben-Engelkirk Fody, Clinical chemistry principles, procedure, correlations (fourth edition).

**Keywords**

Biomarkers criteria, interpret results, apply markers

**ACADEMIC SUPPORT TEAM:**

The College of Medicine and the Department of Medical Education are working on ensuring that our students receive optimal support to their learning. The list of academics shown below represent the departments involved in the teaching and learning of this block. If a student needs help in their teaching and learning they might consult one academic from the list. He/she might email them and arrange a time to see them if needed, otherwise email might be of help.

|  |  |
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| Prof. Hanan Habib | Microbiology | Extension : 71014 | hahabib@ksu.com.sa |
| Prof. Ammar Rikabi | Pathology | Extension : 71893 | ammar\_rikabi12@yahoo.com |
| Dr. Sufia Hussain | Pathology | Extension : | sufiahusain@hotmail.com |
| Dr. Osama Yousif | Pharmacology | Extension : 71327 | oymjahrasoul@hotmail.com |
| Dr. Abeer Almasri | Physiology | Extension: 81983  | Eblmasri@hotmail.comaelmasri@ksu.edu.sa |
| Prof. Adel Almogren | Immunology | Extension: 71925  | almogren@ksu.edu.sa |

**Schedule**

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| **Week 1 - Cardiovascular block (Female)** |
| **Week (1) Starting : 21/02/2016 to 25/02/2016 (12/05/1437 to 16/05/1437)** |
| **Normal Heart, Arrhythmias and Cardiac Cycle** |
| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****21 February 2016** | **Monday****22 February 2016** | **Tuesday****23 February 2016** | **Wednesday****24 February 2016** | **Thursday****25 February 2016** |
| **8:00 – 9:00 am**Self – directedLearning | **8:00 – 9:00 am**Contractile mechanism in cardiac muscle**(Physiology)****Dr. Mona Soliman** | **8:00 – 9:00 am**Cardiac cycle 2**(Physiology)****Dr. Abeer Al Masri** | **8:00 – 9:00 am**Beta adrenergicBlockers**(Pharmacology)****Prof. Hanan Hagar** | **8:00 – 10:00 am****(Practical)**Histology of the cardiac muscle**(Histology)****All Staff** |
| **9:00 – 10:00 am**Anatomy of the heart**(Anatomy)****Dr. Sanaa Alshaarawi** | **9:00 – 10:00 am**Self – directedLearning | **9:00 – 10:00 am**The ElectrocardiogramECG**(Physiology)****Dr. Mona Soliman** | **9:00 – 10:00 am**Arrhythmias**(Cardiac Science Department)****Dr. Hanan Baker** |
| **10:00 – 11:00 am**Structure of the cardiac muscle**(Histology)****Dr. Raeesa Abdultawab** | **10:00 – 11:00 am**Cardiac electric activity**(Physiology)****Dr. Mona Soliman** | **10:00 – 11:00pm**The Development of the Heart**(Anatomy)****Dr. Jamilah El-Medany** | **10:00 – 12:00nn****(Practical)**The ElectrocardiogramECG**(Physiology)** | **10:00 – 11:00 am**Antiarrhythmicdrugs 1**(Pharmacology)****Prof. Abdulrahman Almotrefi** |
| **11:00am – 12:00nn**Introduction to SAQs & using Blackboard**(Medical Education)****Dr. Mona Soliman****Mrs. Bashayier Alyousfi** | **11:00am – 12:00nn**Cardiac cycle 1**(Physiology)****Dr. Abeer Al Masri** | **11:00am – 12:00nn**Alpha adrenergic blockers & sympatholytic**(Pharmacology)****Prof. Hanan Hagar** | **11:00am – 12:00nn**AntiarrhythmicDrugs 2**(Pharmacology)****Prof. Abdulrahman Almotrefi** |
| **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** |
| **1:00 – 3:00 pm****(Practical)**Anatomy of the heartGroup F1**(Anatomy)****All Staff** | **1:00 – 3:00 pm****(Practical)**Anatomy of the heartGroup F2**(Anatomy)****All Staff** | **1:00 – 2:00 pm**Self – directedLearning | **1:00 – 3:00 pm****S A L A M** | **1:00 – 2:00 pm**Self – directedLearning |
| **2:00 – 3:00 pm**Self – directedLearning | **2:00 – 3:00 pm**Self – directedLearning |

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| **Week 2 - Cardiovascular block (Female)** |
| **Week (2) Starting : 28/02/2016 to 03/03/2016 (19/05/1437 to 23/05/1437)** |
| **Valve disease, The Heart as a Pump and heart failure** |
| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****28 February 2016** | **Monday****29 February 2016** | **Tuesday****01 March 2016** | **Wednesday****02 March 2016** | **Thursday****03 March 2016** |
| **8:00 – 10:00 am****Problem-based****Learning****Case 1 Tutorial 1** | **8:00 – 9:00 am**Microbiology of myocarditis & pericarditis**(Microbiology)****Prof. Hanan Habib** | **8:00 – 9:00 am**Rheumatic heart disease**(Immunology)****Dr. Amani Ballo** | **8:00 – 10:00 am****Problem-based****Learning****Case 1 Tutorial 2** | **8:00 – 9:00 am**Self – directedLearning |
| **9:00 – 10:00 am**Venous return & cardiac output**(Physiology)****Dr. Mona Soliman** | **9:00 – 10:00 am**Pathology of rheumatic fever, endocarditis & heart valves**(Pathology)****Dr. Sufia Husain** | **9:00 – 10:00 am**Self – directedLearning |
| **10:00 – 11:00am**Regulation of stroke volume (preload, contractility & afterload) & heart failure**(Physiology)****Dr. Mona Soliman** | **10:00 – 11:00 am**Stroke volume**(Physiology)****Dr. Mona Soliman** | **10:00 – 11:00 am**Risk factors & pathogenesis of atherosclerosis**(Pathology)****Dr. Sufia Husain** | **10:00 – 12:00nn****(Practical)**The recording of Jugular venous & carotid arterial pressures**(Physiology)****All Staff** | **10:00 – 11:00 am**Pathology & pathogenesis of ischemic heart diseases**(Pathology)****Dr. Sufia Husain** |
| **11:00 – 12:00nn** Heart sounds & murmurs**(Physiology)****Dr. Mona Soliman** | **11:00am –12:00nn**Lactic acidosis**(Biochemistry)****Dr. Sumbul Fatma** | **11:00am – 12:00nn**Infective endocarditis**(Microbiology)****Dr. Fawzia** | **11:00am –12:00nn**Self – directedLearning |
| **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** |
| **1:00 – 3:00 pm****(Practical)**Heart sounds**(Physiology)****All Staff** | **1:00 – 2:00 pm**Drug therapy for heart failure 1**(Pharmacology)****Prof. Abdulrahman Almotrefi** | **1:00 – 3:00 pm****(Practical)**Pathology of cardiovascular diseases 1**(Pathology)****Dr. Shaesta Zaidi****Dr. Sufia Husain** | **1:00 – 3:00 pm****S A L A M** | **1:00 – 3:00 pm****(Practical)**Pathology of cardiovascular diseases 2**(Pathology)****Dr. Shaesta Zaidi****Dr. Sufia Husain** |
| **2:00 – 3:00 pm**Drug therapy for heart failure 2**(Pharmacology)****Prof. Abdulrahman Almotrefi** |

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| **Week 3 - Cardiovascular block (Female)** |
| **Week (3) Starting : 06/03/2016 to 10/03/2016 (26/05/1437 to 01/06/1437)** |
| **Normal Blood Pressure and Hypertension** |
| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****06 March 2016** | **Monday****07 March 2016** | **Tuesday****08 March 2016** | **Wednesday****09 March 2016** | **Thursday****10 March 2016** |
| **8:00 – 9:00 am**Anatomy of large blood vessels – arteries**(Anatomy)****Dr. Jamilah El-Medany** | **8:00 – 9:00 am**Regulation of blood pressure**(Physiology)****Dr. Abeer Al Masri** | **8:00 – 9:00 am**Shock**(Physiology)****Dr. Abeer Al Masri** | **8:00 – 9:00 am**Self – directedLearning | **8:00 – 9:00 am**Self – directedLearning |
| **9:00 – 10:00 am**Histology of the blood vessels**(Histology)****Dr. Raeesa Abdultawab** | **9:00 – 11:00 am****(Practical)**Measurement of arterial blood pressure**(Physiology)****All Staff** | **9:00 – 10:00 am**CholesterolMetabolism**(Biochemistry)****Dr. Sumbul Fatma** | **9:00 – 10:00 am**Self – directedLearning | **9:00 – 10:00 am**Self – directedLearning |
| **10:00 – 11:00 am**Arterial blood pressure**(Physiology)****Dr. Abeer Al Masri** | **10:00 – 11:00 am**Capillary circulation**(Physiology)****Dr. Abeer Al Masri** | **10:00 – 12:00nn****(Practical)**Anatomy and histology of the major arteries and veinsGroup F1**(Anatomy & Histology)****All Staff** | **10:00 – 12:00nn****(Practical)**Anatomy and histology of the major arteries and veinsGroup F2**(Anatomy & Histology)****All Staff** |
| **11:00am – 12:00nn**Anatomy of large blood vessels – veins**(Anatomy)****Dr. Sanaa Alshaarawi** | **11:00am – 12:00nn**Treatment of hypertension 1**(Pharmacology)****Prof. Yieldez Bassioni** | **11:00am – 12:00nn**Treatment of hypertension 2**(Pharmacology)****Prof. Yieldez Bassioni** |
| **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** |
| **1:00 – 2:00 pm**Self – directedLearning | **1:00 – 2:00 pm**Self – directedLearning | **1:00 – 2:00 pm**Self – directedLearning | **1:00 – 3:00 pm****S A L A M** | **1:00 – 2:00 pm**Self – directedLearning |
| **2:00 – 3:00 pm**Self – directedLearning | **2:00 – 3:00 pm**Self – directedLearning | **2:00 – 3:00 pm**Self – directedLearning | **2:00 – 3:00 pm**Self – directedLearning |

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| **Week 4 - Cardiovascular block (Female)** |
| **Week (4) Starting : 13/03/2016 to 17/03/2016 (04/06/1437 to 08/06/1437)** |
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| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****13 March 2016** | **Monday****14 March 2016** | **Tuesday****15 March 2016** | **Wednesday****16 March 2016** | **Thursday****17 March 2016** |
| **2nd Semester** **Vacation**  |

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| **Week 5 - Cardiovascular block (Female)** |
| **Week (5) Starting : 20/03/2016 to 24/05/2016 (11/06/1437 to 15/06/1437)** |
| **Atherosclerosis and Myocardial Infarction** |
| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****20 March 2016** | **Monday****21 March 2016** | **Tuesday****22 March 2016** | **Wednesday****23 March 2016** | **Thursday****24 March 2016** |
| **8:00 – 10:00 am****Problem-based****Learning****Case 2 Tutorial 1** | **8:00 – 10:00 am****Clinical Skills****History taking for cardiac disease & clinical examination of the cardiovascular system****Group F2** | **8:00 – 9:00 am**Lipoprotein & Atherosclerosis**(Biochemistry)****Dr. Reem Sallam** | **8:00 – 10:00 am****Problem-based****Learning****Case 2 Tutorial 2** | **8:00 – 9:00 am**Self – directedLearning |
| **9:00 – 10:00 am**Pathology of thromboembolism**(Pathology)****Dr. Sufia Husain** | **9:00 – 10:00 am**Oxidative stress**(Biochemistry)****Dr. Reem Sallam** |
| **10:00 – 12:00nn****Clinical Skills****History taking for cardiac disease & clinical examination of the cardiovascular system****Group F1** | **10:00 – 11:00 am**Pathology & pathogenesis of hypertension**(Pathology)****Dr. Sufia Husain** | **10:00 – 11:00am**Pathology of vasculitis**(Pathology)****Dr. Sufia Husain** | **10:00 – 11:00 am**Antianginal drugs 1**(Pharmacology)****Dr. Osama Yousef** | **10:00 – 11:00 am**Biochemical markers of myocardial infarction**(Biochemistry)****Dr. Rana Hassanato** |
| **11:00am – 12:00nn**Lipoprotein metabolism**(Biochemistry)****Dr. Reem Sallam** | **11:00am – 12:00nn**Drugs for hyperlipidemia 1**(Pharmacology)****Prof. Yieldez Bassioni** | **11:00am –12:00nn**Antianginal drugs 2**(Pharmacology)****Dr. Osama Yousef** | **11:00am-12:00nn**Drugs for hyperlipidemia 2**(Pharmacology)****Prof. Yieldez Bassioni** |
| **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** |
| **1:00 – 2:00 pm**Anatomy of the arterial supply and venous drainage of the heart**(Anatomy)****Dr. Jamilah El-Medany** | **1:00 – 2:00 pm**Thrombolytic therapy**(Pharmacology)****Prof. Hanan Hagar** | **1:00 – 2:00 pm**Self – directedLearning | **1:00 – 3:00 pm****S A L A M** | **1:00 – 2:00 pm**Self – directedLearning |
| **2:00 – 3:00 pm**Coronary circulation**(Physiology)****Dr. Abeer Al Masri** | **2:00 – 3:00 pm**Self – directedLearning | **2:00 – 3:00 pm**Self – directedLearning | **2:00 – 3:00 pm**Self – directedLearning |

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| **Week 6 - Cardiovascular block (Female)** |
| **Week (6) Starting : 27/03/2016 to 31/03/2016 (18/06/1437 to 22/06/1437)** |
| **Consolidation Week** |
| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****27 March 2016** | **Monday****28 March 2016** | **Tuesday****29 March 2016** | **Wednesday****30 March 2016** | **Thursday****31 March 2016** |
| **Consolidation** | **Consolidation** | **Consolidation** | **Consolidation** | **Consolidation** |

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| **Week 7 - Cardiovascular block (Female)** |
| **Week (7) Starting : 03/04/2016 to 07/04/2017 (25/06/1437 to 29/06/1437)** |
| **Examination Week** |
| **CHAIR PERSON : Dr. Mona Soliman** |
| **CO-CHAIR : Dr. Hanan Baker** |
| **Sunday****03 April 2016** | **Monday****04 April 2016** | **Tuesday****05 April 2016** | **Wednesday****06 April 2016** | **Thursday****07 April 2016** |
| **8:00 – 9:00 am****Self – directed****Learning** | **8:00 – 11:30 am****Final MCQ** | **8:00 – 9:00 am****Self – directed****Learning** | **8:00 – 9:00 am****Self – directed****Learning** | **8:00 – 9:00 am****Self – directed****Learning** |
| **9:00 – 10:00 am****Self – directed****Learning** | **9:00 – 10:00 am****Self – directed****Learning** | **9:00 – 10:00 am****Self – directed****Learning** | **9:00 – 10:30 am****OSPE** |
| **10:00 – 11:00 am****Self – directed****Learning** | **10:00 – 11:00am****Self – directed****Learning** | **10:00 – 11:00 am****Self – directed****Learning** |
| **11:00am – 12:00nn****Self – directed****Learning** | **11:30am – 12:00nn****Self – directed****Learning** | **11:00am – 12:00nn****Self – directed****Learning** | **11:00am – 12:00nn****Self – directed****Learning** | **10:30am – 12:00nn****Self – directed****Learning** |
| **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** | **Lunch****12:00 – 1:00 pm** |
| **1:00 – 2:00 pm****Self – directed****Learning** | **1:00 – 2:00 pm****Self – directed****Learning** | **1:00 – 2:00 pm****Self – directed****Learning** | **1:00 – 2:00 pm****Self – directed****Learning** | **1:00 – 2:00 pm****Self – directed****Learning** |
| **2:00 – 3:00 pm****Self – directed****Learning** | **2:00 – 3:00 pm****Self – directed****Learning** | **2:00 – 3:00 pm****Self – directed****Learning** | **2:00 – 3:00 pm****Self – directed****Learning** | **2:00 – 3:00 pm****Self – directed****Learning** |

**Plagiarism**

Plagiarism is a voluntary act to copy sentences and give a misleading impression that the text is created by the person whose name appears on the work. For example an assignment submitted as part of the requirements of assessment of a subject.

Plagiarism may include plagiarism of ideas and or plagiarism of text (sentences or paragraphs). It also may include the use of diagrams, tables, images, cartoons etc without acknowledging the original creator of the work.

The act of copy-and-paste writings even if the aim is to produce a good assignment with well-structured English statements is unethical and when discovered could cause serious consequences including disciplinary action. Students need to construct statements in their own words and refer to the correct references related to what they have written and included in their assignment/work. Giving credit and acknowledgement to the original authors/creators are valued by the academic community as it reflects an ethical and professional attitude.

*Why is plagiarism wrong?*

Universities, higher education institutes and scientific communities consider plagiarism as a major problem for a number of reasons:

* It is an act of stealing ideas and the work of original authors/creators.
* It does not represent acceptable professional, ethical or scientific behaviour.
* It raises doubts about the credibility of the person/group of people who committed such act.

*How can teachers/college discover an act of plagiarism?*

There are a number of software programs such as authenticate and many others available to detect the act of plagiarism. Some of these programs are available free online.

These tools can locate the places and sentences where students have copied and the original resource (articles, manuscripts, papers, books, websites) for such statements/paragraphs or images.

*What are the consequences of plagiarism?*

Students who commit plagiarism will be exposed to disciplinary action including the failure of the subject concerned provided that such act has been confirmed with evidence.

**Assessment of Students in the Block**

In order to pass the block, you must obtain a minimum final block grade of D (the grading guide attached as appendix¹), this grade is a composition from several block requirements, which can be subdivided as:

1. Attendance
2. Tutor assessment
3. Written Exams
4. OSPE (Objective Structured Practical Examination)

The final grade is a composition of the grades obtained for the specified block requirements, calculated as follows:

* Continuous Assessment (Tutor Assessment in PBL sessions) : 5%
* Written Examinations:
	+ SAQs : 20%
	+ MCQ : 40%
* Final Block Exam (40%)
* OSPE : 35 %

**TOTAL 100%**

1. **Attendance :**

Students are required to attend not less than 75% of all educational activities during the block. These include small group teaching, lectures, practical sessions, skills training sessions and integrated clinical sessions.

Your attendance will be recorded during all sessions. Failure to meet this requirement without a valid explanation will result in exclusion from the final examination. On the other hand, your presence will be rewarded by assigned marks.

1. **Tutor Assessment in Large and Small groups (Continuous Assessment):**

During each session, your individual efforts will be evaluated by your tutor. The tutors are instructed to evaluate two aspects:

1. The extent to which you demonstrate that you study and prepare yourself thoroughly between the two sessions (i.e., preparation).
2. The extent to which you actively contribute during group discussion (i.e., participation). Your grade for each session depends upon both your preparation and your participation. The grade will be on the scale from “5”, “4”, “3”, “2”, or “1”. Which have the following general descriptors:

5 = Outstanding (Excellent)

4 = Very good

3 = Good

2 = Average

1 = Poor

The block contains two sessions each week, so the maximum amount of ‘participation points’ you are able to obtain will be from two sessions multiplied by the number of weeks. The total participation points will be recalculated according to the weight for each participation in the total assessment.

Your tutor can give you more information about the evaluation of your participation. The details of these evaluation also given in “Tutor Assessment of Student” form.

1. **Written Examination:**
2. Final written exam 40%: at the end of the block in form of MCQs, that are prepared mainly from sessions and presented to the students. This exam will consist of 80-100 MCQs that will assess factual knowledge too.
3. **Objective Structured Practical Examination** **(OSPE** **):**

This contains 35% of the marks. It is a practical examination at the end of the block. The OSPE examination will consist of 15-20 OSPE stations. The purpose of the OSPE stations is to test your deeper understanding of the basic sciences. The OSPE will take place at the end of each block.

1. **Short Answer Questions (SAQs)**

This comprises 20% of the marks. It contains 3 to 5 cases which include 12-20 integrated SAQs. The time allocated for each question is 3 minutes. The purpose of SAQs is to test the knowledge and its application in integrated manner and to increase the validity and reliability of the written exam. The SAQ will take place at the end of each block

***Methods of student’s formative assessment:***

* Self evaluation
* Peer evaluation
* Tutor evaluation (both summative & formative)
* Log book

**Block Evaluation**

The block evaluation uses the following three data sources:

1. Student Feedback In form of **DREEM** – Dundee Ready Educational Environment Measure
2. Tutor Feedback
3. Student Results

**LEARNING RESOURCES**

The list below comprises the key textbooks and learning resources which have been prescribed and recommended for use in the undergraduate medical course at King Saud University. It is expected that you have your own copy of prescribed textbooks and use them as one of your main resources in learning. Before making any purchases, you might carefully examine all other recommended textbooks in an area and chose the text that matches with your needs and your learning style. Although all these texts are available in the Medical Library, you might need to purchase texts that you use frequently in these years as the demand upon library texts is usually high.

**Medical Dictionary**

*Prescribed :*

Martin EA (2010). Oxford Concise Medical Dictionary. Oxford: Oxford University Press.

*Recommended textbooks:*

Dorland (2010). Dorland’s Pocket Medical Dictionary with CD-ROM, Twenty-eighth Edition, Elsevier, UK.

Dorland (2007). Dorland’s Illustrated Medical Dictionary with CD-ROM, Thirty-first Edition, Elsevier, UK.

**Anatomy & Embryology**

*Prescribed textbook:*

Drake RL, Vogl W and Mitchell AWM (2005). Gray’s Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.

Snell RS (2005). Clinical Anatomy for Medical Students. 7th ed. Philadelphia: Lippincott Williams & Wilkins.

Larson WJ (2001). Human Embryology. New York: Churchill Livingstone.

*Recommended textbooks:*

McMinn RH (2004). McMinn’s Color Atlas of Human Anatomy. Fifth Edition. Mosby Publisher, UK.

Moore KL and Dalley AF (2005). Clinically Oriented Anatomy. Philadelphia: Lippincott Williams & Wilkins.

Netter FH (2006). Atlas of Human Anatomy. 4th ed. Philadelphia: Saunders WB.

Agur AMR and Dalley AF (2005). Grant’s Atlas of Anatomy. 11th ed. Philadelphia: Lippincott Williams & Wilkins.

More KL (2002). The Developing Human. Philadelphia: Saunders WB.

Sadler TW. (2005) Langman’s Essential Medical Embryology. Philadelphia: Lippincott Williams & Wilkins.

Sadler TW. (2006) Langman’s Medical Embryology. 10th ed. Philadelphia: Lippincott Williams & Wilkins.

**Histology**

*Prescribed textbook:*

Gartner LP and Hiatt JL (2002). Color Textbook of Histology. 2nd ed. Philadelphia: Saunders WB.

*Recommended textbooks:*

Young B, Lowe JS, Stevens A and Heath JW (2006). Wheater’s Functional Histology. 5th ed. London: Churchill Livingstone.

**Physiology**

*Prescribed textbook:*

Rhoades R and Pflanzer R (2003). Human Physiology, 4th ed. London: Brooks/Cole.

Hall JE. Guyton and Hall Textbook of Medical Physiology (2010). Twelfth Edition. Churchill Livingstone, UK.

*Recommended textbooks:*

Berne RM, Levy MN, Koeppen BM and Stanton BA. (2005) Physiology. 5th ed. London: Mosby

Sherwood L. (2006). Human Physiology: From Cells to Systems. 4th ed.Brooks/Cole Pub.Co: Sydney.

Fox SI. (2008). Fundamentals of Human Physiology. 9th ed. McGraw-Hill: Boston.

Saladin KS (2009). Anatomy and Physiology. McGraw Hill Lange, USA

Barrett KE, Barman SM, Boitano S, Brooks HL (2009). Ganong’s Review of Medical Physiology. Twenty Third Edition. McGraw-Hill Publisher, UK.

**Pharmacology**

*Prescribed textbook:*

Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone, Elsevier, UK.

*Recommended textbooks:*

Katszing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Medical Biochemistry**

*Prescribed textbook:*

Lieberman M, Marks AD (2008). Mark’s Basic Medical Biochemistry: A Clinical Approach. Lippincott Williams & Wilkins, New York.

Champe PC, Harvey RA, Ferrier DR (2005). Lippincott’s Illustrated Reviews Biochemistry. 3rd ed. Philadelphia: Lippincott Williams & Wilkins.

*Recommended textbooks:*

Murray RK, Roolwell VW, Bender D, Botham KM, Weill A, Kennelly PJ (2009). Harper’s Illustrated Biochemistry. Twenty -eighth Editions. McGraw Hill, Lange, New York.

Baynes J and Dominiczak M (2005). Medical Biochemistry. 2nd ed. London: Mosby.

Bhagavan NV (2002). Medical Biochemistry. Fourth-Edition, Elsevier, UK.

**Microbiology & Parasitology**

*Prescribed textbook:*

Goering R, DoCkrell H, Zuckerman M, Wakelin D, Riott I, Mims C (2008). Mims’ Medical Microbiology. Fourth Edition. Mosby, UK.

John DT, Petri Jr (2006). Markell and Voge’s Medical Parasitology. Ninth Edition. Elsevier, UK.

*Recommended textbooks:*

Greenwood D, Slack RC, Peutherer JF, Barer MR (2007). Medical Microbiology. Seventh Edition. Churchill Livingstone, UK.

Strohol WA. Lippincotts Illustrated Review Microbiology (2006). Second Edition. Lippincott Williams & Wilkins, New York.

Brooks GF, Butel JS, and Morse SA. (2004). Jawetz, Melnick, and Adelberg’s Medical Microbiology. 23rd ed. New York: McGraw-Hill Co and Lange Appleton.

Engleberg NC, DiRita V, and Dermody TS. (2007). Schaechter’s Mechanisms of Microbial Disease. 4th ed. Philadelphia: Lippincott Williams & Wilkins.

Neva FA, Brown HW. (1994). Basic Clinical Parasitology. 6th ed. Connecticut: Prentice-Hall International Inc.

Chamberlain NR (2008). Medical microbiology & immunology. McGraw Hill Lange Publisher, UK.

Levinson WE (2010). Review of Medical Microbiology and Immunology. Eleventh-Edition, McGraw-Hill Publisher, UK

**Pathology**

*Prescribed textbook:*

Kumar V and Cotran RS (2007). Robbins Basic Pathology. 8th ed. Philadelphia: Saunders WB.

*Recommended textbooks:*

Kumar V, Abbas AK, and Fausto N (2004). Robbins and Cotran Pathologic Basis of Disease. 7th ed. Philadelphia: Saunders WB.

Stevens A, Lowe JS, Young B (2008). Wheaters Basic Histopathology. A Colour Atlas and Text. Churchill Livingstone, Elsevier, UK.

**Immunology**

*Prescribed textbook:*

Delves PJ, Martin SJ, Burton DR, Riott IM (2006). Riott’s Essential Immunology. Eleventh Edition. Blackwell Publishing, UK.

*Recommended textbooks:*

Male D, Brostoff J, Roth DB, and Roitt I. (2006). Immunology. 7th ed. Edinburgh: Mosby.

**PBL and Learning Skills**

*Prescribed textbook:*

Azer SA (2006). Core Clinical Cases in Basic Biomedical Sciences. Hodder-Arnold, UK.

Azer SA (2008). Navigating Problem-Based Learning. Elsevier Australia, Australia.

*Recommended textbook:*

Kushner TK and Thomasma DC (2001). Dilemmas for Medical Students and Doctors in Training. Cambridge: University Press.

**Communication Skills & Introduction to Clinical Medicine**

*Prescribed textbook:*

Lloyd M, Bor R (2006). Communication Skills for Medicine. Churchill Livingstone. UK.

Munro JF, Campbell IW (2006). Macleod’s Clinical Examination. Tenth Edition. Churchill Livingstone, UK.

 Talley NJ and O’Connor S. (2006). Pocket Clinical Examination. Melbourne: Blackwell Science.

**Medicine**

Kumar P and Clark M (2010). Clinical Medicine. 7th ed. Edinburgh: Elsevier Saunders.

Edwards C and Bouchier IA. (2003). Davidson’s Principles and Practice of Medicine. 14th ed. Edinburgh: Churchill Livingstone.

*(In the preclinical years these two textbooks may help you in the preparation of your learning issues, you will also need them in the clinical years).*

**Professionalism**

*Prescribed textbook:*

Feldman MD, Christensen JF (2007). Behavioural Medicine. A Guide for Clinical Practice. McGraw-Hill Lange, UK.

Stern DT (2005). Measuring Medical Professionalism. Oxford University Press, UK.

Spandorfer J, Pohl CA, Rattner SL, Nasca TJ (2010). Professionalism in Medicine. A case-based Guide for Medical Students. Cambridge University Press, UK.



KING SAUD UNIVERSITY

College of Medicine

Department of Medical Education

**Feedback to Students on PBL Performance**

**Cardiovascular Block**

**Year 1 (Academic Year 2015-2016)**

**Student’s ID no : …………………………………………………Group number:…………**

**Student’s name:………………………………………………………………………………..**

**Tutor’s name……………………………………………………………………………….......**

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You will receive feedback on your performance in PBL tutorials from your tutor. After completing the 2nd PBL case, your tutor will meet with each student in your group on individual basis. He or she will use the following criteria for providing feedback on your performance. Feedback items are grouped under two main headings.

1= Deficient/lacking/or poor; 2= Working on it; 3= showing some improvement; 4 = developed; 5=well developed (marks are allocated as follows: 1 for rank 1, 2 mark for rank 2, 3 marks for rank 3, and 4 marks for rank 4, and 5 marks for rank 5, maximum mark is 5 for each group)

**1. Learning and cognitive skills:**

Ability to: 1 2 3 4 5

* Identify problems in the case
* Generate hypotheses
* Build mechanisms
* Collect new information
* Interpret findings
* Identify learning issues
* Apply knowledge learnt

 Mark= /5

**2. Interaction and participate to the group function:**

Ability to: 1 2 3 4 5

* Work collaboratively with other members
* Take active roles such as scribing
* Communicate effectively
* Arrive to tutorials on time
* Demonstrate good manners
* Keep the group focused
* Share resources with others

 Mark = /5

Tutor’s Name: Signature: Total Mark= /10

Comments

………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………



KING SAUD UNIVERSITY

College of Medicine

Department of Medical Education

**Assessment of student’s Performance in PBL**

**Cardiovascular Block**

**Year 1 (Academic Year 2015-2016)**

**Student’s ID no.: ............................................................................ Group number:…………………**

**Student’s name: ………………………………………….......................................................................................**

**Tutor’s name: …………………………………………….........................................................................................**

**1=Unsatisfactory ; 2=Poor; 3=Good, 4=Very good; 5=Excellent**

 **1. Preparation and participation:**

Ability to:

* Contribute actively to discussion 1 2 3 4 5
* Use evidence when debate an issue 1 2 3 4 5
* Demonstrate critical analysis skills 1 2 3 4 5
* Integrate knowledge 1 2 3 4 5
* Demonstrate deep understanding 1 2 3 4 5

**Total Marks = 25**

**2. Professional behaviour:**

Ability to:

* Come to tutorials on time 1 2 3 4 5
* Communicate effectively 1 2 3 4 5
* Demonstrate good manners 1 2 3 4 5
* Keep the group focused 1 2 3 4 5
* Give and receive feedback 1 2 3 4 5

 **Total marks = 25**

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**Tutor’s Name: Signature: Total maximum Marks for the case = 50 /10 = 5 marks**

**Comments**

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**KING SAUD UNIVERSITY**



**COLLEGE OF MEDICINE**

**MEDICAL EDUCATION DEPARTMENT**

**STUDENT’S EVALUATION OF THEIR PBL TUTOR**

**Date: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Tutor’s Name: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_ Group No.:\_\_\_\_\_\_\_\_**

**Student: Peer: Other: Name (Optional):\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**How well did the tutor facilitate group process in the following regards? Please put a check (✓) in the box.**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| 1. Appropriately facilitated the brainstorming sessions.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately facilitated the hypothesis reorganization sessions.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately facilitated the reporting sessions.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately manage the time flow.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Help to keep the group focused on its task
 | **1** | **2** | **3** | **4** | **5** |
| 1. Provided a well balanced intervention within the group process, but avoided dominating.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Intervened when chairman or reporter needed.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Provided constructive positive and constructive feedback to the group as needed.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Encouraged positive and constructive feedback within the group about its performance
 | **1** | **2** | **3** | **4** | **5** |
| 1. Showed enthusiasm.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Helped to create a supportive group climate.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Encouraged logical and critical thinking.
 | **1** | **2** | **3** | **4** | **5** |
| 1. Overall rating of the tutor.
 | **1** | **2** | **3** | **4** | **5** |

Number Code Values:

5- EXCELLENT 4- VERY GOOD 3-GOOD 2- FAIR 1- POOR

**KING SAUD UNIVERSITY**



**COLLEGE OF MEDICINE**

**MEDICAL EDUCATION DEPARTMENT**

  **STUDENT RATING OF LECTURES**

**Date: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_Subject: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_Instructor:\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Purpose:**

This form is designed as an observation tool to rate the performance of each instructor in the different sessions. It is intended to provide a tool for lecturer improvement.

**Directions:**

Using the anchors below, check (✓) your rating for each item below. Check (✓) N/A for items that do not apply.

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **No.** | **Standard Procedure** | **5** | **4** | **3** | **2** | **1** | **N/A** |
| **1** | Started and ended class on time. |  |  |  |  |  |  |
| **2** | Presented overview of content and objectives. |  |  |  |  |  |  |
| **3** | Presented information according to objectives. |  |  |  |  |  |  |
| **4** | Used relevant examples and illustrations (graphs, etc.) to explain major ideas |  |  |  |  |  |  |
| **5** | Used alternative explanations when necessary. |  |  |  |  |  |  |
| **6** | Made efficient use of questions with students. |  |  |  |  |  |  |
| **7** | Covered all contents/objectives. |  |  |  |  |  |  |
| **8** | Exhibited enthusiasm. |  |  |  |  |  |  |
| **9** | Encouraged students to express themselves. |  |  |  |  |  |  |
| **10** | Asked questions prior to closure |  |  |  |  |  |  |
| **11** | Summarized major points/related contents to objectives. |  |  |  |  |  |  |
| **12** | Amount you learned in the class was: |  |  |  |  |  |  |

**Mention 3 strong points in this lecture:**

1. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
2. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
3. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Mention 3 points for Improvement:**

1. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
2. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**
3. **\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_**

**Your name: (optional)\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_­­­**