
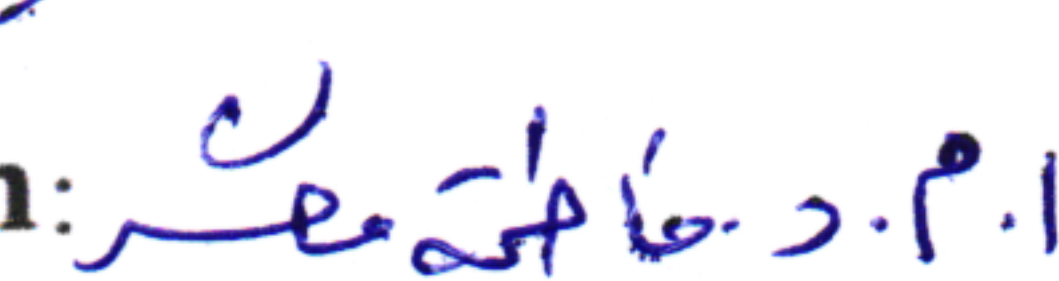



## Course Description Template

University Name: Warith Al-Anbiya  
Faculty/Institute: College of medicine  
Scientific Department: medical education / 2nd stage / 5th unit  
Academic or Professional Program Name: Integration System  
Final Certificate Name: .....  
Academic System: Integration System  
Description Preparation Date: 25/8/2025  
File Completion Date: 25/8/2025

Signature:   
Head of Branch: 

Date: 27/8/2025

Signature:   
Vice Dean for Scientific  
Affairs: Dr. Luth M. Abbas  
Date: 27/8/2025

The file is checked by:

Department of Quality Assurance and University Performance

Director of the Quality Assurance and University Performance

Department: Professor Dr. Ali Al Mousawi

Date: 27.8.2025

Signature: 



Dean's approval

الأستاذ الدكتور  
علي عبد السيد  
عميد كلية الطب





Ministry of Higher Education and Scientific Research

UNIVERSITY OF WARITH ALANBAYAA

COLLEGE OF MEDICINE

# **Academic Program and Course Description Guide**

2025



# Course Description Form

1. Course Name:
Unit 5 Cardiovascular unit .
2. Course Code:
medu20Σ
3. Semester / Year:
2020-2021
4. Description Preparation Date:
2020
5. Available Attendance Forms:
6. Number of Credit Hours (Total) / Number of Units (Total)
10 HOURS
7. Course administrator's name (mention all, if more than one name)
8. Course Objectives
At the end of this unit students should be able to:
1. Describe the normal anatomy of the heart, great vessels, and coronary circulation.
2. Explain the physiology of cardiac muscle, conduction system, cardiac cycle, and hemodynamics.
3. Recognize the pathophysiology of major cardiovascular disorders:
o Ischemic heart disease
o Heart failure
o Valvular heart diseases
o Hypertension
o Arrhythmias
o Congenital heart diseases
4. Identify the clinical manifestations of common cardiovascular diseases (chest pain, palpitations, dyspnea, edema, syncope).
5. Interpret basic cardiovascular investigations (ECG, echocardiography, cardiac enzymes, chest X-ray, catheterization findings).



٦. Outline the principles of treatment ( <b>lifestyle measures, pharmacological management, interventional and surgical options</b> ). ٧. Understand preventive cardiology <b>and the role of controlling risk factors (smoking, diabetes, obesity, dyslipidemia, hypertension</b>
٩. Teaching and Learning Strategies
١. Theoretical lectures ٢. Practical training and skill lab ٣. Seminars and group discussion ٤. PBL
١٠. Course Structure
A.curriculum map

week s	discipline	objectives	hours	Practical sessions & hours
١	<b>Anatomy</b>	١. Describe the basic anatomy of sympathetic system ٢. Describe the basic anatomy of parasympathetic system	٢	
	<b>Physiology</b>	١. Define and Compare terms and concepts related to the sympathetic and parasympathetic systems, including: the central location of cell body of origin, number of synapses between CNS and effector organs, degree of myelination, and general effects on target tissues. ٢. Define and compare pre- and postganglionic autonomic neurons, and white and gray rami communicants.	٤	



		<ul style="list-style-type: none"> <li>ϣ. Describe the sensory input and roles for visceral afferent fibers of the ANS.</li> <li>Ϙ. Describe the synaptic characteristics, receptors, and neurotransmitters for the parasympathetic and sympathetic division of the ANS.</li> <li>ο. Describe the function of non-adrenergic, non-cholinergic fibers in the ANS.</li> <li>ϧ. Explain the relatively diffuse action of the sympathetic division compared with the parasympathetic division.</li> <li>ϣ. Describe the ANS signaling mechanism and the effects of sympathetic and parasympathetic stimulation of lungs, heart, arteries, and veins; gastrointestinal function; renal function; and sexual function.</li> <li>⋈. Explain the Cardiovascular reflexes</li> <li>Ϡ. Explain the Cardiorespiratory interactions</li> <li>ϣ⋄. Describe signs and symptoms of ANS dysfunction that may accompany lesions that affect the ANS. Including Horner's Syndrome, medullary dysfunction, common visceral dysfunction, and multiple system atrophy (Shy-Drager syndrome).</li>   <li>ϣ. Explain the Receptor mechanisms regulating central autonomic function</li> </ul>		
	Pharmacology		ϣ	







	<b>Pathology</b>	<p>Hemodynamic disorder:</p> <ol style="list-style-type: none"> <li>١. Define edema and describe its types</li> <li>٢. Explain the pathophysiology of edema</li> <li>٣. Describe hperaemia and congestion as terms</li> <li>٤. Explain pathogenesis of thrombosis with reference to Virchow's triad</li> <li>٥. Describe morphological features of different types of thrombi,</li> <li>٦. Differentiate arterial versus venous thrombosis</li> <li>٧. Describe the fate of thrombi.</li> <li>٨. Define and describe embolism and its types,</li> <li>٩. Explain the consequences of thromboembolism. pulmonary embolism</li> <li>١٠. Define shock and list its types</li> <li>١١. Describe the stages of shock</li> <li>١٢. Recognize the causes of cardiogenic shock</li> </ol> <p>Explain the pathogenesis of septic shock</p>	٢	
	<b>Pharmacology</b>	<p>Anti-arrhythmic drugs:</p> <ol style="list-style-type: none"> <li>١. Classes of antiarrhythmic drugs &amp; their clinical uses.</li> <li>٢. Mechanism of action of each class of antiarrhythmic drugs, commonly used drugs, alternative drugs, clinically important interactions &amp; their adverse effects</li> </ol>	٢	
	<b>Clinical resources</b>	<p>A. Syncope</p> <ol style="list-style-type: none"> <li>١. Definition, etiology of syncope</li> </ol>	١	







		<p>٢. The pressure and volume changes in the heart during each phase of the cardiac cycle.</p> <p>٣. Relate the phases of the cardiac cycle to the ECG.</p> <p>٤. The role of the heart valves in the cardiac cycle.</p> <p>٥. Clinical correlation between heart diseases and cardiac cycle</p>		
	<b>Pharmacology</b>	<p>A. Anti- arrhythmic drugs (٢):</p> <p>١. General principals of antiarrhythmic therapy</p> <p>٢. Classification of antiarrhythmic drugs</p> <p>٣. Differences between antiarrhythmic drugs</p> <p>B. Clinical pharmacology of antiarrhythmic drug</p> <p>١. Differentiate between supraventricular and ventricular arrhythmia treatment\</p> <p>Treatment of common and serious arrhythmias: AF, SVT,VT,VF</p>	٢	
	<b>Microbiology</b>	<p><b>Rheumatic fever</b></p> <p><b>Infective endocarditis</b></p> <p>١ .Causative pathogens</p> <p>Including list, Microbiological and biochemical features, virulence factors and laboratory diagnosis.</p> <p>٢. The mechanism of pathogenesis of each condition</p>	١	
	<b>Clinical resources</b>	<p><b>Management of AS</b></p> <p>١. Significance of valvular heart disease.</p> <p>٢. Role of valvular heart disease in dyspnoea</p> <p>٣. How to distinguish AV sclerosis from stenosis</p> <p>٤. Clinical signs of severe AS</p> <p>٥. Tests to diagnose AS</p> <p>٦. Surgery indication</p>	١	







		١. Definition of shock ٢. Causes and types of shock ٣. The dangers of cardiogenic shock and how it leads to death ٤. Management of cardiogenic shock		
	<b><u>Biochemistry</u></b>	Cardiac enzymes: ١. Understand Isozymes as markers of myocardial infarction ٢. Understand Troponin as biomarker; know when to order it and what does the test results mean! ٣. Distinguish between angina and myocardial infarction	١	
	<b><u>Pharmacology</u></b>	Management of MI: ١. Rationale for Drug Therapy in MI, ٢. Classes of Drugs Used to Treat MI, ٣. Their mode of action, Clinical uses and common side effects. Hypolipidemic agents: ١. General outlines of treatment of hyperlipidemia ٢. Different classes of hypolipidemic agents, ٣. Pharmacology of every agent regarding: Mechanism of action, Pharmacokinetics, Clinical effects, Side effects and interaction with other drugs	٢	
	<b><u>Clinical resources</u></b>	Management of MI ١. DD of chest pain ٢. Analysis of pain ٣. Sign & symptoms of ischemic coronary syndromes ٤. Examination & diagnosis ٥. Management of MI ٦. Complications of acute MI	١	







		<p>٥. Histology of the heart and histological features of cardiac muscle</p>		
	<b>Physiology</b>	<p>Regulation of arterial blood pressure:</p> <p>١. How vasomotion influence blood flow</p> <p>٢. The neural and hormonal influences on vasomotion and arterial blood pressure,</p>	٢	
	<b>Biochemistry</b>	<p>Cholesterol and Hypercholesterol:</p> <p>١. Functions of cholesterol.</p> <p>٢. Endogenous and exogenous cholesterol.</p> <p>٣. Cholesterol synthesis.</p> <p>٤. Fates of cholesterol</p> <p>٥. Lipoproteins synthesis</p> <p>٦. Entero-hepatic circulation of cholesterol</p> <p>٧. Functions of lipoproteins</p> <p>٨. Structure and function of the LDL-receptor</p> <p>٩. Good and bad cholesterol</p> <p>١٠. Foam cells and how they lead to atherosclerosis</p> <p>١١. Familial hypercholesterolemia</p>	٢	
	<b>Psychology</b>	<p>Issues of patient compliance:</p> <p>١. Importance of compliance /adherence</p> <p>٢. Types of noncompliance</p> <p>٣. Causes and consequences of noncompliance</p> <p>٤. Methods to improve compliance in patients</p>	١	
	<b>Community medicine</b>	<p>Epidemiology of hypertension</p>	١	



	<b>Clinical resources</b>	<p>Management of Hypertension:</p> <ol style="list-style-type: none"> <li>١. Definition &amp; classification</li> <li>٢. Hypertension management in various countries</li> <li>٣. Pathophysiology of HT</li> <li>٤. Factors influencing prognosis</li> <li>٥. Documentation of HT</li> <li>٦. Defining target BP</li> <li>٧. Examination and lab investigations in hypertensive patients</li> <li>٨. Guidelines for management of HT.</li> </ol>	١	
	<b>Pharmacology</b>	<p>Diuretics</p> <ol style="list-style-type: none"> <li>١. Different classes of diuretics, their mode and site of action ,their clinical uses,</li> <li>٢. Adverse effects and their interaction with other drugs</li> </ol> <p>Antihypertensive drugs:</p> <ol style="list-style-type: none"> <li>١. Regulation of blood pressure by normal body mechanisms</li> <li>٢. Autonomic control of blood pressure</li> <li>٣. Different classes of drugs used for treatment of hypertension, their mechanism of action, agents commonly used, their adverse effects, and clinically important drug interactions</li> <li>٤. Different strategies for treatment of hypertension</li> </ol>	٢	
٦	<b>Anatomy</b>	<p>Heart Anomalies:</p> <ol style="list-style-type: none"> <li>١. Fetal circulation</li> <li>٢. Changes in circulation at birth</li> <li>٣. Remnants of the embryonic vessels</li> <li>٤. Congenital heart disorders according to position, atresia or stenosis, abnormal growth, defective septa.</li> <li>٥. Combined defects, anomalies of relationship of chambers to great vessel</li> </ol>	٢	<p>١. <u>Practical Lab</u></p> <p>a. <u>biochemistry lab</u></p> <p><u>lab results interpretaion</u></p>
	<b>Embryology</b>	Embryology of the heart with correlation to congenital heart anomalies)	١	



	<b>Physiology</b>	<p>Venous Return:</p> <ol style="list-style-type: none"> <li>١. The importance of venous return</li> <li>٢. The characteristics of veins that allow them to be able to hold large volumes of blood and to have low resistance to flow</li> <li>٣. The importance of venous pressure, and the factors that determine venous pressure</li> </ol> <p>Microcirculation and tissue fluid formation:\</p> <ol style="list-style-type: none"> <li>١. Define the Starling equation and discuss how each component influences fluid movement across the capillary wall</li> <li>٢. Explain how edema develops in response to different situations.</li> </ol>	٢	
	<b>Biochemistry</b>	<p>Frederickson Classification of Lipid Disorders*.</p> <p>Type. Average of overnight serum. Elevated particles, Associated clinical disorders, Serum TC, Serum TG</p>	١	
	<b>Clinical resources</b>	<p>Management of CHF</p> <ol style="list-style-type: none"> <li>١. Etiology</li> <li>٢. Causes of right and left sided HF</li> <li>٣. Pathophysiology of CHF</li> <li>٤. Sign &amp; symptoms</li> <li>٥. Diagnosis, management &amp; prognosis of CHF</li> </ol> <p>CVS Imaging</p> <ol style="list-style-type: none"> <li>١. Different imaging modalities used in examination of the CVS.</li> <li>٢. Protocol for reading the normal chest x-ray.</li> <li>٣. Appearance of some of the common and important abnormalities on Chest x-ray</li> </ol>	١	
	<b>Pharmacology</b>	<p>Inotropic agents &amp; heart failure:</p> <ol style="list-style-type: none"> <li>١. Inotropic agents [positive &amp; negative] types &amp; various cardiovascular conditions in which such agents are applied</li> <li>٢. Types of drugs that are used in the treatment of HF, their mechanism of action,</li> </ol>	٢	



V	Microbiology	<p>interaction with other drugs &amp; adverse effects</p> <p>٧. Treatment options for various stages of HF.</p> <p>Angiotensin-converting enzyme inhibitors (ACEI) and Angiotensin II Receptor Blockers (ARBs):</p> <p>٨. Role in management of HF</p> <p>٩. Mode of action, clinical uses, adverse effects and clinically important Drug Interactions</p> <p>Infections lead to dilated cardiomyopathy</p> <p>١. Causative pathogens</p> <p>Including list, Microbiological and biochemical features, virulence factors and laboratory diagnosis.</p> <p>٢. The mechanism of pathogenesis of each condition</p>	١	Lab Anatomy
	Anatomy	<p>Demonstrate the origin, course and branches of the major arteries that supply the gluteal region, hip, thigh, leg, ankle and foot. Explain the functional significance of anastomoses between branches of these arteries at the hip and knee.</p> <p>Demonstrate the locations at which the femoral, popliteal, posterior tibial and dorsalis pedis arterial pulses can be palpated.</p> <p>Demonstrate the course of the principal veins of the lower limb. Explain the role of the perforator veins between the superficial and deep veins and the function of the ‘muscle pump’ for venous return to the</p>		



	<p><b>Pathology</b></p>	<p>heart. Describe the surface landmarks</p> <p>Describe the basic histology of arteries and veins</p> <p>A. Pathology of blood vessels</p> <ol style="list-style-type: none"> <li>١- define and differentiate between aneurysms and dissections</li> <li>٢- Define vasculitis and Describe its types and pathogenic mechanism.</li> <li>٣- Describe pathogenesis and clinical features of thromboangiitis obliterans (buerger disease).</li> <li>٤- Explain Pathology of vasculitis associated with other diseases.</li> <li>٥- Explain Pathology of Blood Vessel Hyperreactivity (Raynaud phenomenon).</li> <li>٦- Explain Pathogenesis of Varicose veins.</li> <li>٧- Describe the basic pathology of the tumors of the blood vessels</li> </ol> <p>B. Pathology of thrombosis</p> <ol style="list-style-type: none"> <li>١٣. Describe the factors that predispose to thrombosis ; acquired and inherited.</li> <li>١٤. Define thrombophilia and its classification</li> <li>١٥. Recognize the inherited and acquired causes of thrombophilia</li> <li>١٦. Lab monitoring of common anticoagulant drugs(warfarin and heparin)</li> </ol>	<p>٢</p>	
	<p><b>Pharmacology</b></p>	<p>A. Anticoagulants:</p> <ol style="list-style-type: none"> <li>١. Warfarin,</li> <li>٢. Unfractionated and LMW heparin ,</li> <li>٣. Direct thrombin inhibitors</li> </ol> <p>B. Thrombolytic agents</p> <ol style="list-style-type: none"> <li>١. Streptokinase</li> <li>٢. t-PA</li> <li>٣. Tenecteplase</li> <li>٤. Reteplase</li> </ol>	<p>٢</p>	



	<p><b><u>Microbiology</u></b></p> <p><b>clinical resources</b></p>	<p>C. Inhibitors of anticoagulation and fibrinolysis</p> <p>Mycotic aneurysm          ١. .Causative pathogens          Including list, Microbiological and biochemical features, virulence factors and laboratory diagnosis.          ٢. The mechanism of pathogenesis of each condition</p> <p><u>Deep venous thrombosis</u></p> <ol style="list-style-type: none"> <li>١. Define and describe risk factors for developing DVT</li> <li>٢. Define and describe the symptoms and signs of DVT and PE.</li> <li>٣. Generate a prioritized differential diagnosis of DVT/PE</li> <li>٤. Describe the indications for and utility of various diagnostic tests of DVT/PE</li> <li>٥. Define and describe, and develop an appropriate management plan for DVT/PE</li> <li>٦. Identify the appropriate duration of anticoagulation therapy in a patient with DVT and PE based on the clinical picture</li> <li>٧. Define and describe methods of DVT/PE prophylaxis, their indications and efficacy</li> </ol> <p><u>Peripheral arterial disease(PAD)</u></p> <ol style="list-style-type: none"> <li>١. Describe the risk factors, clinical presentation, and evaluation of PAD</li> <li>٢. Recognize the relationship between atherosclerosis and PAD</li> <li>٣. Recognize different modalities of testing for PAD</li> <li>٤. recognize the options of medical therapy for this disorder</li> </ol>	<p>١</p> <p>١</p> <p>١</p>	
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		<p>◦. recognize the non surgical and surgical intervention in the treatment of PAD</p> <p><u>Varicose veins</u></p> <p>١. Explain the pathophysiology underlying varicose veins (primary venous vs chronic venous insufficiency)</p> <p>٢. Describe the signs and symptoms of varicose veins</p> <p>٣. Identify the important steps in diagnosis of varicose veins including investigations</p> <p>٤. Outline the important modalities of treatment of varicose veins</p>	١	
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# Clinical skill theme

Week	Clinical skill	Clinical skill objectives
١	examination	Blood pressure assesments & pulse exam
٢	History taking	CVS general history
٣	examination	Examination of murmur
٤	History and Exam	hstory of heart failure
٥	History and Exam	History of Atrial Fibrillation ( palpitation )
٦	History and Exam	History of Shortness of Breath
٧	History and Exam	CVS exam & examination of Edema



# Small Group PBL Tutorials:

Every week, students study a problem in a small group in the presence of a tutor. Students meet with the tutor on Sunday (first session) and Thursday (second session) every week. In the first PBL tutorial session, students: a) Read and interpret the case scenario (triggers) and define technical terms. b) Identify the key issues of the problem. c) Brainstorm, ask questions and generate hypotheses (possible causes and consequences). d) Indicate additional information, procedures, required to sort through the hypotheses and what you expect to learn from the additional information. e) Identify their learning needs i.e. objectives. \ • In between the first and second sessions, students follow a self-directed learning approach, using the relevant learning resources in studying the identified learning needs. In the second PBL tutorial session, students: - Present the newly gathered knowledge. - Relate it to the context of the problem. - Integrate the physical, biological and behavioral components in every problem. - Evaluate their tutorial performance

## Summary of the Unit Problems

week	Case presentaion	Summary
١	Catecholamines Crisis	A ٢١-year-old female with ٩-month history of recurrent headaches, palpitations, sweating, and paroxysmal hypertension was found to have a right adrenal mass. Biochemical tests (↑ catecholamines, positive VMA) and CT confirmed pheochromocytoma. She underwent right adrenalectomy after proper α then β blockade, with postoperative normalization of blood pressure.
2	<u>The man collapses at the shrine of the Imam AL-Hussein</u>	<b>A ٦٧ year old man collapses while standing, CPR was done as first aid, t consciousness. A hospital check-ups, confirmed sinus bradycardia syndrome on ECG. Cardiac pace maker implantation was done s failure of conventional antiarrhythmic drugs.</b>
٣	Dizzy feeling after stood up	A ٦٢ year old man often felt like fainting in garden of in his house. Past history revealed a recurrent chest pain upon exertion for the past ٥ years. Clinical examination revealed systolic thrill over the aortic valve region with a loud ejection systolic murmur, S٢ sound was heard. ECG showed left axis deviation



		for a LV hypertrophy, cardiac catheterization revealed aortic stenosis. Subsequently aortic valve replacement was done successfully.
Σ	Hard time with drama of life	Abdul-Hussein, 51 year old obese smoker, having stressful life with family history of cardiac diseases. Three years back, he was diagnosed to have hypertension , hypercholesterolemia. He started to experience retrosternal pain with exertion, relieved by rest one year back. He is on atenolol, isosorbate dinitrate, aspirin, lipid lowering medication and oral hypoglycemic agent. Two weeks back, his chest pain attacks becoming more frequent. He was admitted to A& E emergency unit, diagnosed with myocardial infarction. He had fibrinolytic therapy. He developed ventricular tachycardia and complicated with left ventricular failure and cardiogenic shock. He underwent coronary angiography and angioplasty. He has to change his life style for prevention of further cardiac attacks. His doctor advised him to join support group program.
0	Yellowish spots below eyes	60-year-old man presented with yellowish spots below the eye since one year and recently developed recurrent headache. Lipid profile showed elevated cholesterol, LDL and triglycerides. His blood pressure remained elevated on the second visit. In addition to medication, he was advised to modify his diet and life style. His compliance was poor. Nine months later he presented with recurrent headache. His hypertension was associated with left ventricular hypertrophy and retinal hemorrhages. He was successfully treated with atenolol, enalapril, and lipitor. He was reassured that hypertension is a chronic but treatable disease. Complications should the patient do not comply were explained.
٦	Samira have difficult in breath	60 year old, mother of four, works as administrative assistant at a bank Al-Rashid in holy Karbala. Approximately ٦ months ago, she began to experience difficult breathing, even when lying down and effort related fatigue. She is heavy smoker and overweight. On examination, her doctor found swollen neck veins, evidence of elevated venous pressure and peripheral pitting oedema. Her doctor wanted to admit her to hospital
V	Suha has swollen leg	50 year old, mother of two, works as a computer employee at the University of Karbala. She is obese with sedentary life style using OCP for birth control. Her leg has been swollen, red and painful for ٧ weeks. Clinical evaluation using Well's score with D-Dimer assay and compression ultrasonography confirmed the diagnosis of DVT. She was prescribed anticoagulant therapy as treatment.

## Summary of the Unit Mini-PBLs

week	Case presentaion	Summary
2	Atrial Fibrillation	٦٧-year-old woman with hypertension presented with recurrent palpitations, now prolonged with dizziness, diaphoresis, nausea, and syncope. On exam: irregularly irregular pulse, tachycardia ١٦٠-١٨٠ bpm, stable BP, no murmurs, lungs clear. ECG shows atrial fibrillation with rapid ventricular response.
٣	Rheumatic Fever	9-year-old boy with fever, migratory polyarthritis, sore throat, and new-onset heart failure was



		<p>diagnosed with <b>acute rheumatic fever</b> (ASO <math>\Lambda^{++}</math>, prolonged PR, ESR/CRP high). Echo showed <b>severe mitral regurgitation</b> with cardiomegaly. He improved on corticosteroids and salicylates, but a residual murmur persisted. He was discharged on <b>long-term benzathine penicillin prophylaxis</b> every 2 weeks.</p>
Σ	Myocardial Infarction	<p>Kadhemeea, a 78-year-old woman with a prior MI, was readmitted with a new posterior MI and left ventricular failure. Her lipid profile shows <b>high LDL (160), high triglycerides (240), and low HDL (32)</b>, indicating severe dyslipidemia. Family screening revealed her brother with moderately high LDL and very low HDL, and her sister with isolated hypertriglyceridemia—suggesting a familial lipid disorder with different phenotypic expression</p>
0	Familial hypercholesterolemia	<p>Abdulla, a 37-year-old obese man (BMI <math>\approx 40.6</math>, waist 54 in), presented with hypertension, hyperglycemia (fasting 162, PP 200), and severe dyslipidemia (cholesterol 296, TG 290, LDL 221, HDL 22). He also has LVH with reduced EF (27%) and a family history of premature MI.</p> <p>Diagnosis: <b>familial hypercholesterolemia with metabolic syndrome</b>. He was started on <b>pravastatin, cholestyramine, clofibrate, and aspirin</b>, with lifestyle modification advice.</p>
7	Cardiovascular Hemodynamics	<p>arrha, a 177 cm, 81 kg athlete, presented with hypotension, ectopic beats, low cardiac output, and lab results showing <b>hyponatremia, hypokalemia, hypocalcemia, hypoglycemia, iron deficiency, and severe hypoproteinemia</b>. Despite intensive training, she was found to be malnourished with edema and low energy intake. She was diagnosed with the <b>female athlete triad (disordered eating,</b></p>



		<b>amenorrhea, and osteoporosis risk), and referred for counseling.</b>
	Tetralogy of Fallot	Faris, a 7-month-old boy, had <b>cyanosis, squatting episodes, systolic murmur, polycythemia, low O<sub>2</sub> sat, RVH on ECG, boot-shaped heart on CXR, and echo showing VSD, overriding aorta, and RVOT obstruction</b> — classic <b>Tetralogy of Fallot</b> . Cardiac catheterization confirmed anatomy, and he underwent <b>total surgical repair with VSD patch and RVOT relief</b> , with improved postoperative ECG.
V	PERIPHERAL ARTERIAL DISEASE	atima, a 77-year-old woman with HTN and hyperlipidemia, presents with <b>exertional calf pain relieved by rest (intermittent claudication)</b> . Exam shows <b>cool left leg, hair loss, and diminished posterior tibial pulse</b> , consistent with <b>peripheral arterial disease (PAD)</b> due to atherosclerosis.

# Course Evaluation

Evaluation of the students in this unit will consist of the following:

END OF UNIT SUMMATIVE ASSESSMENT

The exam will cover:

Unit 0: CVS Dates & timetables will be announced later.

The exam will comprise the followings:

Written paper: MCQ + lab materials

OSCE ASSESSMENT OF THE PBL SESSION PBL

assessment form is provided in Appendix

PORTFOLIO:

Detailed content of portfolio will be delivered to the students separately.

**MASTERY SKILLS:** Separate exams for the mastery skills will be assigned. Important note: students fail to pass the mastery skill exam with complete competency will not be allowed to enter the final year exam.



## Learning and Teaching Resources

- Braunwald's Heart Disease
- Harrison's Principles of Internal Medicine
- Davidson's Principles and Practice of Medicine
- Kumar & Clark's Clinical Medicine
- Clinical Examination by Talley & O'Connor
- ESC Guidelines (European Society of Cardiology)

## Appendix : PBL assessment form

	<b>PBL</b>	<b>knowledge</b>	<b>Critical thinking /reasoning</b>	<b>Communication skill and participation</b>	<b>Attitude and collaborative work</b>
unsatisfactory	1	Has no recall of previous knowledge	Identify problems(events) in the case	Not participating spontaneously most of the time	Negative influence • Interrupts others • does not respect others views • Does not help the group to identify the learning objectives
margina	2	• Has limited recall of previous knowledge	Prioritize patient problems • Differentiate important information from others	Rarely asks questions. • Limited participation in discussions	• rarely participates in identify the learning objectives • takes up tasks only one asked by others
satisfactory	3	Apply previous knowledge to the problem	• Give explanations to the patient problems	Occasionally ask questions. • Occasionally present ideas clearly	Sometimes participates in identify the learning objectives •



					Volunteer to perform tasks
good	4	Recognizes integration of knowledge and its application to the case	Can identify interrelationship between different concepts with guidance • Can identify learning objectives with guidance	Regularly asks questions that stimulate discussions. • Often present ideas and help in clarifying ideas	always participates in identify the learning objectives
excellent	5	Can recognize knowledge gap	Can identify interrelationship between different concepts without guidance • Can identify learning objectives without guidance	Leads discussion most of the time • Present clear ideas • Give summaries on the subject	Help and encourage the engagement of other members. • Explain difficult concepts to others willingly