Ministry of Healthcare of Ukraine
Poltava State Medical University

GUIDELINES FOR STUDENTS INDEPENDENT WORK IN THE PRACTICAL CLASSES PREPARING

Academic discipline	Internal medicine
Module	Current practice of internal medicine
Content module	Management of the patients with main symptoms and syndromes in cardiology clinic
Study subject	**Management of the patients with heart murmurs** **Management of the patients the with cyanosis**
Course	VI
Faculty	of foreign students training

1.The aims of the training course:

To Know:

Differential diagnosis of functional and organic, systolic and dyastolic murmurs.
Plan of survey, additional instrumental examination methods (radioscopy lung and heart, ECG, Echo-CG)

To be able to:

- Conduct surveys and patients examination with major cardiological syndromes
- To draft survey the patients with heart diseases, to justify the use of major invasive and non-invasive diagnostic techniques witch are using in cardiology, to identify indications and contraindications for their conduction, possible complications
- Identify different options for the course and complications of heart disease
- Carry out differential diagnosis, justify and formulate diagnoses for major cardiac syndromes based on laboratory analysis and test tool
• Prescribe treatment, determine prognosis, to conduct primary and secondary prevention in heart disease
• Register and interpret the ECG in 12 assignments
• Measure and interpret blood pressure
• Diagnose and assist in syncope
• Diagnose and assist in hypertensive crisis
• Diagnose and assist with arterial hypotension
• Diagnose and assist in the paroxysmal disorders of cardiac rhythm
• Diagnose and assist syndrome Morhany-Edems-Stoks
• Conduct pulmonary heart reanimation
• Demonstrate knowledge of moral principles medical specialist and professional principles of subordination

The contents of topic:

Text

Heart Murmurs

Systolic

Early systolic
- Physiologic (innocent)
- Small ventricular septal defect
- Large ventricular septal defect with pulmonary hypertension
- Severe acute mitral or tricuspid regurgitation
- Tricuspid regurgitation without pulmonary hypertension

Midsystolic
- Physiologic (innocent)
  - Vibratory murmur
  - Hyperkinetic states
  - Pulmonary ejection murmur
  - Aortic ejection murmur of old age
- Obstruction to left ventricular outflow
  - Valvular aortic stenosis
  - Supravalvular aortic stenosis
  - Hypertrophic cardiomyopathy
  - Aortic valve prosthesis
- Aortic dilatation
- Murmurs of mitral regurgitation (occasionally)

- Aortic flow murmur in aortic regurgitation
- Coarctation of aorta
- Supraclavicular arterial bruit
- Obstruction to right ventricular outflow
  - Supravalvular pulmonary arterial stenosis
  - Pulmonic valvular stenosis
  - Subpulmonic (infundibular) stenosis
- Flow murmur of atrial septal defect
- Idiopathic dilatation of pulmonary artery
- Pulmonary hypertension of any cause (occasionally)

Late systolic
- Mitral valve prolapse
- Tricuspid valve prolapse

Holosystolic
- Mitral regurgitation
- Tricuspid regurgitation secondary to pulmonary hypertension
- Ventricular septal defect
- Patent ductus arteriosus or aorticopulmonary window with pulmonary hypertension

Diastolic

Early diastolic
- Aortic regurgitation
- Pulmonic regurgitation associated with pulmonary hypertension, congenital or valvular disease

Middiastolic
- Mitral stenosis
- Mitral valve prosthesis
- Tricuspid stenosis
- Atrial myxoma
- Left atrial ball-valve thrombus
- Austin Flint murmur
- Increased diastolic atrioventricular flow
  - Hyperkinetic states
  - Mitral and tricuspid regurgitation
  - Left-to-right shunt (e.g., ventricular septal defect)
  - Acute rheumatic valvulitis
  - Complete heart block
- Coronary artery stenosis

Presystolic
- Mitral stenosis
- Tricuspid stenosis
- Atrial myxoma
- Left-to-right shunt
- Complete heart block
- Severe pulmonic stenosis
- Fourth heart sound
- Severe aortic insufficiency

Heart Murmur, Diastolic:

A diastolic murmur is a finding that provides a clue to an underlying disease process. Unlike their systolic counterparts, diastolic murmurs almost always indicate underlying heart disease.

Approach

Given the broad range of factors that may cause diastolic murmurs, a two-part scheme is used to narrow the differential diagnosis. First, the timing of the murmur (early, mid-, or end-diastolic) helps in localizing the anatomic abnormality to a specific valve or other heart structure. Second, historical features such as congestive heart failure, rheumatic disease, congenital abnormalities, or connective tissue and collagen vascular disease further characterize the underlying cause.

History

A. Symptoms. Many patients with diastolic murmurs will not present with specific complaints; rather, the murmurs will be found in the course of a routine medical examination. With symptomatic lesions, the patient may experience dyspnea, chest pain, or palpitations. Pulmonary regurgitation (PR) is usually asymptomatic except in its most severe forms. More specific symptoms include chest or neck pounding in aortic regurgitation (AR); hemoptysis, embolism, or hoarseness (left recurrent laryngeal nerve compression from the left atrium) in mitral stenosis (MS); failure to thrive or frequent respiratory infections with congenital MS; edema in tricuspid stenosis (TS); and fever, anemia, weight loss, embolism, digital clubbing, arthralgias, syncope, rash, and Raynaud's phenomenon with an atrial myxoma (1).

B. Past medical history. Does the patient have a history of rheumatic fever (RF)? RF is the most common cause of all diastolic murmurs (mitral → aortic → tricuspid → pulmonic) (2). Of patients with mitral stenosis, 50% will have a history of rheumatic fever (3).

1. Endocarditis. Vegetations can lead to either AR/PR or MS/TS.

2. Pulmonary hypertension with PR is classically associated with the Graham Steell murmur, heard in the left third interspace near the sternum and propagated down the sternum.

3. Connective tissue and collagen vascular diseases predispose to aortic root dilatation and AR.

4. Congenital heart malformations can be associated with multiple valvular lesions, left ventricular (LV) outflow tract abnormalities, or shunts (with resultant volume overload).

5. Atrial myxoma is a rare cause of *variable* AV valve obstruction.

6. Syphilis can cause aortitis and AR.

Physical examination (PE)

A. Table 7.3 lists characteristic PE findings of diastolic murmurs.

B. Fine points of the physical examination

1. Is the murmur of AR louder at the right sternal border? If so, consider aortic root dilation. Remember, whereas the duration of the *chronic* AR murmur is directly proportional to the severity of the regurgitation, the duration of the *acute* AR murmur may not predict its severity (3).

2. Is the murmur of MS shorter, or does it extend closer to S_2? The length of this murmur, not its intensity, is directly proportional to the severity of the stenosis (3). In addition, the murmur may not be audible with increased heart rates because of shortening of diastole.

3. Does the murmur of MS vary from examination to examination? If so, and especially if it is introduced by a "plop" sound, consider atrial myxoma.

Testing

Echocardiogram is the essential test for confirming the anatomic location of the murmur and its severity. Transthoracic echocardiography (ECHO) is generally sufficient, unless endocarditis is suspected, in which case a transesophageal ECHO is preferred to evaluate for vegetations. If aortic root dilatation is present on ECHO, a computed tomography or magnetic resonance imaging scan may help to delineate the anatomy further. Additional laboratory testing may be warranted to further evaluate the underlying cause (e.g., serologic studies for collagen vascular disease, serologic test for syphilis, and so on).

Diagnostic assessment

With a careful examination and thorough history, the valve causing the murmur and the probable cause of the valvular lesion can be identified prior to ordering the definitive test (ECHO). The most common cause of all diastolic murmurs is still rheumatic heart disease, even though the incidence of acute rheumatic fever has decreased. Mitral stenosis is almost invariably caused by rheumatic heart disease (98% in one study of excised valves) (3,4), with the remainder caused by vegetations (from endocarditis) or congenital factors (4). Tricuspid stenosis is also predominantly rheumatic in origin and is rarely an isolated lesion. Other causes of TS include carcinoid and congenital malformations. Rheumatic heart disease is the leading cause of chronic AR, followed by congenital bicuspid valves and aortic root dilatation (Marfan's syndrome, Ehlers-Danlos syndrome, ankylosing spondylitis, and syphilitic aortitis). If chronic, AR can result in LV dilation and compensation; if acute, it can be associated with severe LV overload and significant symptoms. Acute AR is most often related to endocarditis, aortic dissection, and trauma. Pulmonary regurgitation without hypertension has multiple causes, including pulmonary trunk dilation, endocarditis, carcinoid, trauma (from balloon-tipped catheters), and rheumatic fever. The nonstenotic physiologic murmurs are related to high-flow states across an otherwise normal mitral
or tricuspid valve. For a mitral flow murmur, the primary lesions are usually mitral regurgitation, ventricular septal defects, or patent ductus arteriosus. For a tricuspid flow murmur, an atrial septal defect or severe tricuspid regurgitation is the most common cause. The Austin–Flint murmur, caused by increasing left ventricular pressure pushing the anterior mitral leaflet into the flow of blood coming from the atrium, is the result of significant aortic regurgitation

Table 7.3 Physical examination findings of diastolic murmurs

Timing	Murmur	Pitch	Location	Shape or quality	Maneuvers and other features
Early diastolic murmurs	Aortic regurgitation (AR)	High	Left lower sternal border (LLSB)	Decrescendo	Have patient lean slightly forwar hold breath in deep expiration, press diaphragm firmly against chest; increases with hand grip and squatting; listen for Austin Flint murmur.
	Pulmonary regurgitation (PR) *with* pulmonary hypertension	High	LLSB	Decrescendo	Distinguish from AR by prominer jugular A-wave; right ventricular (RV) heave; RV S_4 that increases with inspiration
	PR *without* pulmonary hypertension	High	LLSB	Crescendo-decrescendo (short duration)	RV heave (sometimes); palpable thrill over pulmonic area may signify dilation of pulmonic artery; *follows silent gap after S* (unlike other PR murmur)
Middiastolic murmurs	Mitral stenosis	Low	Point of maximal impulse (PMI)	Rumbling; may have presystolic accentuation	Have patient on left side, hold be lightly on chest; may be preceded by opening snap; increased with handgrip; decreased with inspiration.
	Tricuspid stenosis	Low	LLSB (near xyphoid)	Rumbling	Inspiratory augmentation.
	Nonstenotic rumbles	Low	PMI or LLSB	Rumbling	No presystolic component; preceded by low-pitched S_3 instead of high-pitched opening snap.
	Austin-Flint (mitral origin, associated with AR)	Low	PMI	Rumbling	Begins after S_3; increased with handgrip and squatting. In severe AR, this murmur may be absent.

Heart Murmur, Systolic

Systolic murmurs can herald significant clinical deterioration and sudden death, or they can represent stable or clinically insignificant conditions. Although technologic advances in cardiac diagnostic testing continue, auscultation remains the mainstay of diagnosis and is the key to the cost-effective use of technology.

Approach

Systolic murmurs can develop in all age groups. Close attention should be given to the history [looking for associated symptoms (e.g., dyspnea)] and to the physical examination (looking for specific murmur characteristics described below).

History

A. General issues in the history. The history can provide important clues as to whether the murmur is clinically significant. Any history of rheumatic fever, previously known valvular disease, congenital heart disease, or intravenous drug abuse would be important to ascertain.

Murmurs of early adulthood suggest congenital or rheumatic disease, whereas murmurs with onset later in life are consistent with degenerative valvular changes.

B. Patient symptoms. Patients should be asked about shortness of breath, dyspnea on exertion, orthopnea, and paroxysmal nocturnal dyspnea. Patients with these symptoms warrant an expedited evaluation because these symptoms suggest cardiac decompensation. Advanced aortic stenosis specifically is associated with chest pain, syncope, and heart failure, although a gradient across the valve can exist for years prior to symptom onset. Chest discomfort is often present in advanced disease, but sudden death occurs in 15% of patients with no previous symptoms (1).

C. Association of a murmur with a specific disease. Recent myocardial infarction endocarditis could cause papillary muscle dysfunction resulting in mitral or tricuspid regurgitation. Mitral regurgitation can be seen in connective tissue disease, coronary artery disease, and congenital disease, but is commonly associated with conditions leading to left ventricular dilatation such as congestive heart failure (CHF)

Endocarditis, myocardial infarction, trauma, prolapse, or congenital heart disease usually precede tricuspid regurgitation. Mitral valve prolapse, which is clinically characterized by

palpitations, fatigue, and chest pain, is often associated with anxiety. Hypertrophic cardiomyopathy can be seen in patients with a family history and usually presents between the ages of 20 and 40 years. Presenting symptoms include dyspnea on exertion, chest pain, palpitations, or syncope. It is an important cause of sudden death in athletes. A history of anemia, thyroid disease, or fever should also be elicited from patients being evaluated for a systolic murmur as each of these conditions can cause a murmur from increased flow.

Physical examination

A. Technique. Auscultate the heart with the bell to best detect lower frequencies and the heart sounds (S_1-S_4). The quality of the murmur is best heard with the diaphragm. Inspiration increases the audibility of right ventricular sounds.

B. Murmur characteristics. Table 7.4 presents a summary of the characteristics of different causes of systolic murmurs (2,3). Etchell et al. (3) have prepared a comprehensive review on the usefulness of specific physical examination findings in the diagnosis of systolic murmurs.

Testing

Testing of an undiagnosed cardiac murmur can include an electrocardiogram (ECG), a chest x-ray study (CXR), and an echocardiogram. Echocardiograms, although useful for quantification of stenotic valvular disease, can overestimate the degree of regurgitation.

A. Aortic stenosis. Specific ECG findings in aortic stenosis can include left ventricular hypertrophy (LVH), left axis deviation, conduction disturbances, and atrial hypertrophy. On CXR, cardiac size remains normal until stenosis is severe, then signs of CHF may be present. The echocardiogram may reveal thickened or calcified aortic leaflets, bicuspid valve, and LVH. The size of the valve can be estimated and the pressure gradient across the valve can be assessed. Cardiac catheterization can also be used to assess the size of the valve and the gradient. Even though echocardiography is accurate in measuring valve area and gradient, catheterization is usually indicated because 50% of patients above age 40 years have coronary artery disease.

B. Mitral regurgitation. In mitral regurgitation, the ECG may reveal LVH with left atrial enlargement and later in the course, atrial fibrillation. In severe disease, CXR usually reveals cardiomegaly without pulmonary venous congestion. The echocardiogram reveals valvular anatomy, but can overestimate the severity of the regurgitation. Exercise testing can be used

to determine clinical deterioration in mitral regurgitation. Catheterization is used to assess the contractile state of the ventricle as well as the regurgitant and forward stroke volume.

C. Other disease processes. The ECG with tricuspid insufficiency often reveals atrial fibrillation. The CXR may show right atrial hypertrophy, and the echocardiogram shows valvular anatomy. Pulmonic stenosis will lead to ECG findings consistent with right ventricular hypertrophy. Hypertrophic cardiomyopathy is best diagnosed by echocardiography. ECG may reveal LVH and occasionally a shortened PR interval is seen. Cardiac catheterization can be used to quantify the gradient caused by the hypertrophic lesion.

Table 7.4 Characteristics of systolic murmurs

Systolic murmurs	Murmur type	Maneuvers
Tricuspid regurgitation	Early systolic (low pressure) Holosystolic (high pressure)	Inspiration intensifies murmur
Aortic stenosis	Midsystolic crescendo, decrescendo with delayed and decreased peripheral pulses	Palpable thrill in aortic area on full expiration
Mitral valve prolapse	Late systolic	Squatting and prompt standing may augment murmur
Tricuspid insufficiency	Holosystolic on left sternal border	Large jugular waves; increased murmur with inspiration
Mitral regurgitation	Apical holosystolic radiating to axilla and left sternal border	If left ventricle dilatation present, grade II–IV murmur with S_3 mid to late or holosystolic with palpable thrill
Pulmonic stenosis	Midsystolic	Murmur increases with inspiration
Ventricular septal defect	Harsh, holosystolic murmur	Differentiated from mitral regurgitation by radiating to right of sternum rather than apex
Hypertrophic cardiomyopathy	Midsystolic; if murmur present, associated with S_4 and heard best at left lower sternal border	

Problem definition: differentiation between AS and MR murmurs

A typical normal heart sound signal that corresponds to a heart cycle consists of four structural components:

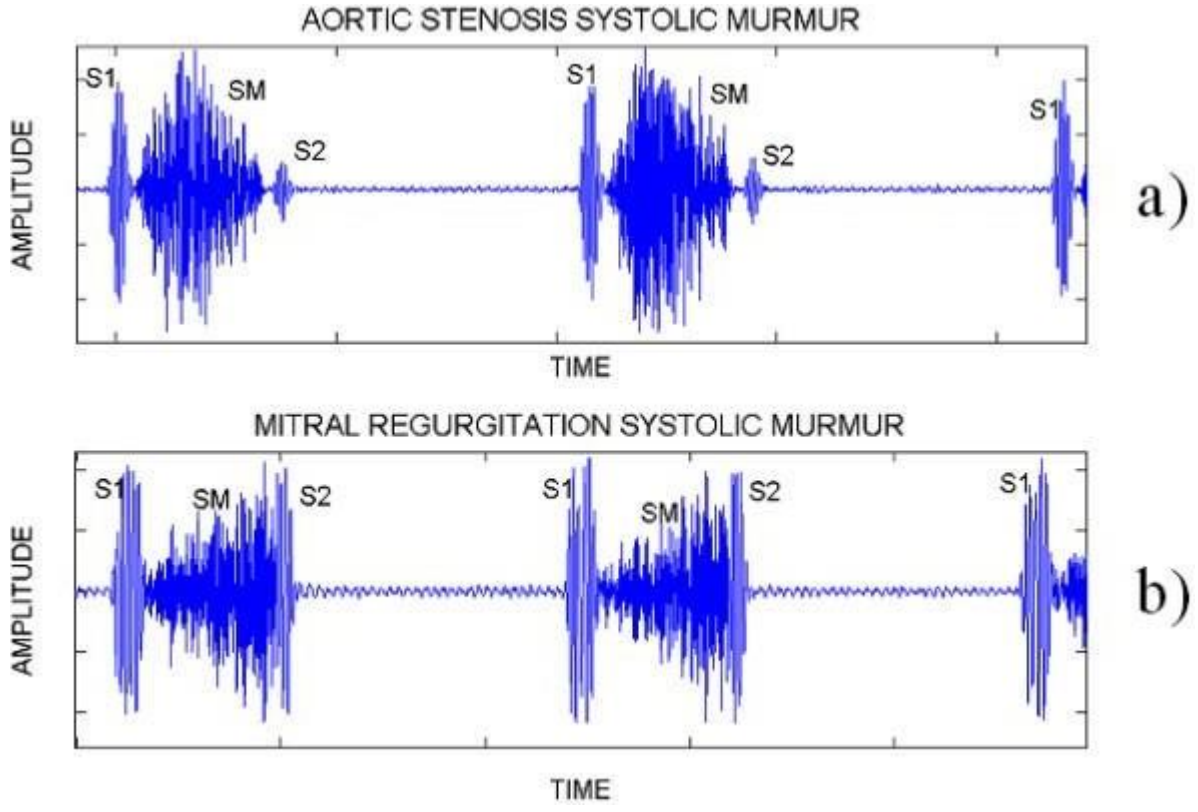
– The first heart sound (S1, corresponding to the closure of the mitral and the tricuspid valve).

– The systolic phase.

– The second heart sound (S2, corresponding to the closure of the aortic and pulmonary valve).

– The diastolic phase.

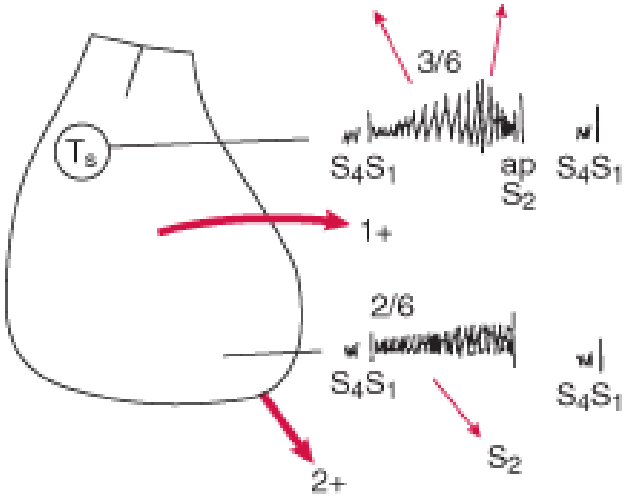
Heart sound signals with various additional sounds are observed in patients with heart diseases. The tone of these sounds can be either like murmur or click-like. The murmurs are generated from the turbulent blood flow and are named after the phase of the heart cycle where they are best heard, e.g. systolic murmur (SM), diastolic murmur (DM), pro-systolic murmur (PSM) etc. The heart sound diagnosis problem consists in the diagnosis from heart sound signals of a) whether the heart is healthy, or not and b) if it is not healthy, which is the exact heart disease. In AS the aortic valve is thickened and narrowed. As a result, it does not fully open during cardiac contraction in systolic phase, leading to abnormally high pressure in the left ventricle and producing a systolic murmur that has relatively uniform frequency and rhomboid shape in magnitude (Figure (Figure1a).1a). In MR the mitral valve does not close completely during systole (due to tissue lesion) and there is blood leakage back from the left ventricle to the left atrium. MR is producing a systolic murmur that has relatively uniform frequency and magnitude slope (Figure (Figure1b).1b). The spectral content of the MR systolic murmur has faintly higher frequencies than the spectral content of the AS systolic murmur. The closure of aortic valve affects the second heart sound and the closure of the mitral valve affects the first heart sound.



Two heart cycles of AS and MR heart sounds.

It can be concluded by the visual insplection that the AS systolic murmur has very similar characteristics with the MR systolic murmur and therefore the differentiation between these two diseases is a difficult problem in heart sound diagnosis, especially for young inexperienced clinicians. All these facts define the problem of differentiation between the AS and the MR murmurs. In the following sections we propose a method based on time-frequency features and decision tree classifiers for solving this problem.

Diagram of physical findings in a patient with aortic stenosis and mitral regurgitation.



Murmur, character, intensity, and radiation are depicted. Sound of pulmonic closure exceeds that of aortic closure. Left ventricular (LV) thrust and right ventricular (RV) lift (heavy arrows) are identified. A 4th heart sound (S_4) and systolic thrill (T_S) are present. a = aortic closure sound; p =pulmonic closure sound; S_1 = 1st heart sound; S_2 =2nd heart sound; 3/6 = grade of crescendo-diminuendo murmur (radiates to both sides of neck); 2/6 = grade of pansystolic apical crescendo murmur; 1+ = mild precordial lift of RV hypertrophy (arrow shows direction of lift);
2+ = moderate LV thrust (arrow shows direction of thrust).

Systolic heart sounds: Systolic sounds include the following:

- 1st heart sound (S_1)
- Clicks

S_1 and the 2nd heart sound (S_2, a diastolic heart sound) are normal components of the cardiac cycle, the familiar “lub-dub” sounds.

S_1 occurs just after the beginning of systole and is predominantly due to mitral closure but may also include tricuspid closure components. It is often split and has a high pitch. S_1 is loud in mitral stenosis. It may be soft or absent in mitral regurgitation due to valve leaflet sclerosis and rigidity but is often distinctly heard in mitral regurgitation due to myxomatous degeneration of the mitral apparatus or due to ventricular myocardial abnormality (eg, papillary muscle dysfunction, ventricular dilation).

Clicks occur only during systole; they are distinguished from S_1 and S_2 by their higher pitch and briefer duration. Some clicks occur at different times during systole as hemodynamics change. Clicks may be single or multiple.

Clicks in congenital aortic or pulmonic stenosis are thought to result from abnormal ventricular wall tension. These clicks occur early in systole (very near S_1) and are not affected by hemodynamic changes. Similar clicks occur in severe pulmonary hypertension. Clicks in mitral

or tricuspid valve prolapse, typically occurring in mid to late systole, are thought to result from abnormal tension on redundant and elongated chordae tendineae or valve leaflets.

Clicks due to myxomatous degeneration of valves may occur any time during systole but move toward S_1 during maneuvers that transiently decrease ventricular filling volume (eg, standing, Valsalva maneuver). If ventricular filling volume is increased (eg, by lying supine), clicks move toward S_2, particularly in mitral valve prolapse. For unknown reasons, characteristics of the clicks may vary greatly between examinations, and clicks may come and go.

Diastolic heart sounds: Diastolic sounds include the following:

- 2nd, 3rd, and 4th heart sounds (S_2, S_3, and S_4)
- Diastolic knocks
- Mitral valve sounds

Unlike systolic sounds, diastolic sounds are low-pitched; they are softer in intensity and longer in duration. Except for S_2, these sounds are always abnormal in adults.

S_2 occurs at the beginning of diastole, due to aortic and pulmonic valve closure. Aortic valve closure normally precedes pulmonic valve closure unless the former is late or the latter is early. Aortic valve closure is late in left bundle branch block or aortic stenosis; pulmonic valve closure is early in some forms of preexcitation phenomena. Delayed pulmonic valve closure may result from increased blood flow through the RV (eg, in atrial septal defect of the common secundum variety) or complete right bundle branch block. Increased RV flow in atrial septal defect also abolishes the normal respiratory variation in aortic and pulmonic valve closure, producing a fixed split S_2. Left-to-right shunts with normal RV volume flow (eg, in membranous ventricular septal defects) do not cause fixed splitting. A single S_2may occur when the aortic valve is regurgitant, severely stenotic, or atretic (in truncus arteriosus when there is a common valve).

S_3 occurs in early diastole, when the ventricle is dilated and noncompliant. It occurs during passive diastolic ventricular filling and indicates serious ventricular dysfunction in adults; in children, it can be normal. RV $S_3$is heard best (sometimes only) during inspiration (because negative intrathoracic pressure augments RV filling volume) with the patient supine. LV S_3 is best heard during expiration (because the heart is nearer the chest wall) with the patient in the left lateral decubitus position.

S_4 is produced by augmented ventricular filling, caused by atrial contraction, near the end of diastole. It is similar to S_3 and heard best or only with the bell of the stethoscope. During inspiration, RV S_4 increases and LV S_4 decreases. S_4 is heard much more often than S_3 and indicates a lesser degree of ventricular dysfunction, usually diastolic. S_4 is absent in atrial fibrillation (because the atria do not contract) but is almost always present in active myocardial ischemia or soon after MI. S_3, with or without S_4, is usual in significant systolic LV dysfunction; S_4 without S_3 is usual in diastolic LV dysfunction.

A **summation gallop** occurs when S_3 and S_4 are present in a patient with tachycardia, which shortens diastole so that the 2 sounds merge. Loud S_3 and S_4may be palpable at the apex with the patient in the left lateral decubitus position.

A **diastolic knock** occurs at the same time as S_3, in early diastole. It is not accompanied by S_4 and is a louder, thudding sound, which indicates abrupt arrest of ventricular filling by a noncompliant, constricting pericardium.

An **opening snap** may occur in early diastole in mitral stenosis or, rarely, in tricuspid stenosis. Mitral opening snap is very high pitched, brief, and heard best with the diaphragm of the stethoscope. The more severe mitral stenosis is (ie, the higher the left atrial pressure), the closer the opening snap is to the pulmonic component of S_2. Intensity is related to the compliance of the valve leaflets: The snap sounds loud when leaflets remain elastic, but it gradually softens and ultimately disappears as sclerosis, fibrosis, and calcification of the valve develop. Mitral opening snap, although sometimes heard at the apex, is often heard best or only at the lower left sternal border.

Etiology of Murmurs by Timing

Timing	Associated Disorders
Mid systolic (ejection)	Aortic obstruction (supravalvular stenosis, coarctation of the aorta, aortic stenosis, aortic sclerosis, hypertrophic cardiomyopathy, subvalvular stenosis) Increased blood flow across the aortic valve (hyperkinetic states, aortic regurgitation) Dilation of ascending aorta (atheroma, aortitis, aneurysm of aorta) Pulmonic obstruction (supravalvular pulmonary artery stenosis, pulmonic stenosis, infundibular stenosis) Increased blood flow across the pulmonic valve (hyperkinetic states, left-to-right shunt from atrial septal defect, ventricular septal defect) Dilation of pulmonary artery
Mid-late systolic	Mitral valve prolapse, papillary muscle dysfunction
Holosystolic	Mitral regurgitation, tricuspid regurgitation, ventricular septal defect
Early diastolic (regurgitant)	Aortic regurgitation: acquired or congenital valve abnormality (myxomatous or calcific degeneration, rheumatic fever, endocarditis), dilation of valve ring (aortic dissection, annuloaortic ectasia, cystic medial necrosis, or hypertension), widening of commissures (syphilis); congenital bicuspid valve with or without ventricular septal defect Pulmonic regurgitation: acquired or congenital valve abnormality, dilation of valve ring (pulmonary hypertension, Marfan syndrome), tetralogy of Fallot, ventricular septal defect

Mid diastolic	Mitral stenosis (rheumatic fever, congenital stenosis, cor triatriatum) Increased blood flow across nonstenotic mitral valve (mitral regurgitation, ventricular septal defect, patent ductus arteriosus, high-output states, complete heart block) Tricuspid stenosis Increased blood flow across nonstenotic tricuspid valve (tricuspid regurgitation, atrial septal defect, anomalous pulmonary venous return) Left or right atrial tumors, atrial ball-valve thrombi
Continuous	Patent ductus arteriosus, coarctation of the pulmonary artery, coronary or intercostal arteriovenous fistula, ruptured aneurysm of sinus of Valsalva, aortic septal defect, cervical venous hum, anomalous left coronary artery, proximal coronary artery stenosis, mammary souffle (venous hum from engorged breast vessels during pregnancy), pulmonary artery branch stenosis, bronchial collateral circulation, small (restrictive) atrial septal defect with mitral stenosis, coronary-cameral fistula, aortic–right ventricular or atrial fistula

Maneuvers That Aid in Diagnosis of Murmurs

Maneuver	Effect on Blood Flow	Effect on Heart Sounds
Inspiration	Simultaneously increases venous flow into the right heart, decreases venous flow into the left heart	Augments right heart sounds (eg, murmurs of tricuspid stenosis and regurgitation, those of pulmonic stenosis* [immediately] and regurgitation [usually]); reduces left heart sounds
Valsalva maneuver	Reduces size of left ventricle (LV); decreases venous return to the right heart and subsequently to the left heart	Augments murmur of hypertrophic obstructive cardiomyopathy and mitral valve prolapse, and diastolic murmur of mitral stenosis; reduces murmurs of aortic stenosis, mitral regurgitation, and tricuspid stenosis
Release of Valsalva maneuver	Increases volume of LV	Augments murmur of aortic stenosis, that of aortic regurgitation (after 4 or 5 beats), and those of pulmonic regurgitation or pulmonic stenosis* (immediately); reduces murmur of tricuspid stenosis
Isometric handgrip	Increases afterload and peripheral arterial resistance	Reduces murmurs of aortic stenosis and hypertrophic obstructive cardiomyopathy; reduces murmur of mitral valve prolapse or papillary muscle dysfunction; augments murmurs of mitral

		regurgitation and aortic regurgitation and diastolic murmur of mitral stenosis
Squatting	Simultaneously decreases venous return to the right heart and increases afterload and peripheral resistance	Augments murmurs of aortic regurgitation, aortic stenosis, mitral valve prolapse, and mitral regurgitation and diastolic murmur of mitral stenosis; reduces murmur of hypertrophic obstructive cardiomyopathy and mitral valve prolapse or papillary muscle dysfunction
Amyl nitrite	Causes intense venodilation, which reduces venous return to the right heart	Augments murmurs of hypertrophic obstructive cardiomyopathy and mitral valve prolapse; reduces murmur of aortic stenosis

*Patient may need to be standing for effect on pulmonic stenosis to be heard.

All patients with heart murmurs are evaluated by chest x-ray and ECG. Most require echocardiography to confirm the diagnosis, determine severity, and track severity over time, usually followed by a cardiac consultation if significant disease is suspected.

Systolic murmurs: Systolic murmurs may be normal or abnormal. They may be early, mid, or late systolic, or holosystolic (pansystolic). Systolic murmurs may be divided into ejection, regurgitant, and shunt murmurs.

Ejection murmurs are due to turbulent forward flow through narrowed or irregular valves or outflow tracts (eg, due to aortic or pulmonic stenosis). They are typically mid systolic and have a crescendo-diminuendo character that usually becomes louder and longer as flow becomes more obstructed. The greater the stenosis and turbulence, the longer the crescendo phase and the shorter the diminuendo phase.

Systolic ejection murmurs may occur without hemodynamically significant outflow tract obstruction and thus do not necessarily indicate a disorder. In normal infants and children, flow is often mildly turbulent, producing soft ejection murmurs. The elderly often have ejection murmurs due to valve and vessel sclerosis.

During pregnancy, many women have soft ejection murmurs at the 2nd intercostal space to the left or right of the sternum. The murmurs occur because a physiologic increase in blood volume and cardiac output increases flow velocity through normal structures. The murmurs may be greatly exaggerated if severe anemia complicates the pregnancy.

Regurgitant murmurs represent retrograde or abnormal flow (eg, due to mitral regurgitation, tricuspid regurgitation, or ventricular septal defects) into chambers that are at lower resistance. They are typically holosystolic and tend to be louder with high-velocity, low-volume regurgitation or shunts and softer with high-volume regurgitation or shunts. Late systolic

murmurs, which may or may not be preceded by a click, are typical of mitral valve prolapse or papillary muscle dysfunction. Various maneuvers are usually required for more accurate diagnosis of timing and type of murmur.

Shunt murmurs may originate at the site of the shunt (eg, patent ductus arteriosus, ventricular septal defects) or result from altered hemodynamics remote from the shunt (eg, pulmonic systolic flow murmur due to an atrial septal defect with left-to-right shunt).

Diastolic murmurs: Diastolic murmurs are always abnormal; most are early or mid diastolic but may be late diastolic (presystolic). Early diastolic murmurs are typically due to aortic or pulmonic regurgitation. Mid diastolic (or early to mid diastolic) murmurs are typically due to mitral or tricuspid stenosis. A late diastolic murmur may be due to rheumatic mitral stenosis in a patient in sinus rhythm.

A mitral or tricuspid murmur due to an atrial tumor or thrombus may be evanescent and may vary with position and from one examination to the next because the position of the intracardiac mass changes.

Continuous murmurs: Continuous murmurs occur throughout the cardiac cycle. They are always abnormal, indicating a constant shunt flow throughout systole and diastole. They may be due to various cardiac defects

Some defects produce a thrill; many are associated with signs of RVH and LVH. As pulmonary artery resistance increases in shunt lesions, the diastolic component gradually decreases. When pulmonary and systemic resistance equalize, the murmur may disappear.

Patent ductus arteriosus murmurs are loudest at the 2nd intercostal space just below the medial end of the left clavicle.Aorticopulmonary window murmurs are central and heard at the 3rd intercostal space level. Murmurs of systemic arteriovenous fistulas are best heard directly over the lesions; those of pulmonic arteriovenous fistulas and pulmonary artery branch stenosis are more diffuse and heard throughout the chest

During pregnancy, a continuous venous hum from breast vessels (mammary souffle) may be mistaken for a continuous cardiac murmur.

Pericardial friction rub: A pericardial friction rub is caused by movement of inflammatory adhesions between visceral and parietal pericardial layers. It is a high-pitched or squeaking sound; it may be systolic, diastolic and systolic, or triphasic (when atrial contraction accentuates the diastolic component during late diastole). The rub sounds like pieces of leather squeaking as they are rubbed together. Rubs are best heard with the patient leaning forward or on hands and knees with breath held in expiration.

Extremity and Abdominal Examination

The extremities and abdomen are examined for signs of fluid overload, which may occur with heart failure as well as noncardiac disorders (eg, renal, hepatic, lymphatic).

Extremities: In the extremities (primarily the legs), fluid overload is manifest as edema which is swelling of soft tissues due to increased interstitial fluid. Edema may be visible on inspection, but modest amounts of edema in very obese or muscular people may be difficult to recognize visually. Thus, extremities are palpated for presence and degree of pitting (visible and palpable depressions caused by pressure from the examiner's fingers, which displaces the interstitial fluid). The area of edema is examined for extent, symmetry (ie, comparing both extremities), warmth, erythema, and tenderness. With significant fluid overload, edema may also be present over the sacrum, genitals, or both.

Tenderness, erythema, or both, particularly when unilateral, suggests an inflammatory cause (eg, cellulitis or thrombophlebitis). Nonpitting edema is more suggestive of lymphatic or vascular obstruction than fluid overload.

Abdomen: In the abdomen, significant fluid overload manifests as ascites arked ascites causes visible abdominal distention, which is tense and nontender to palpation, with shifting dullness on abdominal percussion and a fluid wave. The liver may be distended and slightly tender, with a hepatojugular reflux present.

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Self preparation at class:
Listen information;
Work with patients (with cardiac pathology);
Ask about the problems that have not been found in information given.
Self preparation at home:

Compose the plan of your answer;
Answer the questions to the topic;
Do the test given above.

Task. A 59-year-old female with exertional dyspnea and a loud holosystolic murmur at the apex presented with sinus rhythm. However, intermittent atrial fibrillation had been documented on previous occasions. Atrial enlargement was suggested by the ECG, which was otherwise normal.

The mechanism of mitral regurgitation is:

A. Mitral valve prolapse (posterior leaflet).
B. Mitral valve prolapse (both leaflets).
C. Posterior flail mitral leaflet.
D. Ruptured papillary muscle.

The optimal surgical approach in this patient is:

A. Mitral valve replacement.
B. Mitral annuloplasty.
C. Mitral valve repair.
D. Commissurotomy.

Recommended literature:

I. Main:

1. Internal Medicine: in 2 books. Book 1. Diseases of the Cardiovascular and Respiratory Systems: textbook / N.M. Seredyuk, I.P. Vakaliuk, R.I. Yatsyshyn et al. Київ, Медицина., 2019. - 664 + 48 кольор. вкл.).
2. Internal medicine: Part 1 (cardiology, rheumatology, haematology): textbook for English-speaking students of higher medical schools / edited by Professor M.A. Stanislavchuk and Professor V.A. Serkova. - Vinnytsia: Nova Knyha, 2019. - 392 p.
3. Медицина за Девідсоном: принципи і практика / Навчальний посібник: пер. 23-го англ. вид.: у3 т. Т.3 С. Ралстона, Я. Пенмана, М. Стрекена, Р. Гобсона; К.: ВСВ «Медицина», 2021. – 642 с.
4. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)/
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II. Additional literature:

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3. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
4. Principles and practice of interventional pulmonology / Ernst A., Herth F. -Springer, 2012. - 757 p.
5. Clinical respiratory medicine / Spiro S., Silvestri G., Agusti A. - Saunders, 2012. - 1000 p.
6. Petrov Y. The chief symptoms and syndromes in patients with cardiovascular pathology : The practical handbook fur medical students / Ye. Petrov, Yu. Goldenberg, N. Chekalina; UMSA. - Poltava : TexcepBic, 2010. - 143 .
7. Gastroenterology and Hepatology Board Review: Pearls of Wisdom, Third Edition (Pearls of Wisdom Medicine) by John K. DiBaise (May 11, 2012)
8. Clinical Pulmonology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Oct 30, 2011) - Kindle eBook
9. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Hematology: Clinical Principles and Applications, 4e by Bernadette F. Rodak MS MLS (Feb 18, 2017)
12. Rheumatology, 2-Volume Set: EXPERT CONSULT - ENHANCED ONLINE FEATURES AND PRINT, 5e by Marc C. Hochberg MD MPH, Alan J. Silman MD, Josef S. Smolen MD and Michael E. Weinblatt MD (Oct 19, 2019)
13. Endocrine Pathology: Differential Diagnosis and Molecular Advances by Ricardo V. Lloyd (Nov 5, 2018)
14. Clinical Endocrinology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2017) - Kindle eBook
15. Williams Textbook of Endocrinology: Expert Consult-Online and Print, 12e by Shlomo Melmed, Kenneth S. Polonsky MD, P. Reed MD Larsen and Henry M. Kronenberg MD (May 27, 2016)

16. Electrocardiography, 3e with Student CD (Booth, Electrocardiography for Health Care Personnel) by Kathryn A. Booth (Jan 27, 2017)
17. Echocardiography Review Guide: Companion to the Textbook of Clinical Echocardiography: Expert Consult: Online and Print, 2e (Expert Consult Title: Online + Print) by Catherine M. Otto (Mar 7, 2017).

Answers.

Posterior flail mitral leaflet.
Mitral valve repair.

The mechanism of mitral regurgitation can be determined by echocardiographic assessment of color Doppler jet direction, leaflet motion, and morphology. Note the excessive motion of the posterior leaflet that swings into the left atrium during systole. The regurgitant jet is deflected away from the diseased leaflet and is directed medially (and anterior). Hyperkinetic left ventricular function and left ventricular enlargement suggests severe regurgitation.

The feasibility of repair depends on the mechanism of regurgitation. The easiest type to repair is a flail or prolapsing posterior leaflet (success rate is in the range of 80% to 90%). Furthermore, each mechanism demands specific surgical techniques for optimal correction. For a flail posterior leaflet the most commonly applied technique is quadrilateral resection of the unsupported leaflet tissue, suture closure of the gap or "sliding plastic" and implantation of an annuloplasty ring.

Methodical recommendations consisted by Kulishov S.K.