Valvular heart diseases

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Summary

Valvular heart diseases can take the form of stenosis, insufficiency (regurgitation), or a combination of the two. These defects are typically acquired as the result of infections, underlying heart disease, or degenerative processes. However, certain congenital conditions can also cause valvular heart diseases.

Acquired defects are found primarily in the left heart as a result of higher pressure and mechanical strain on the left ventricle.

Valvular stenosis leads to a greater pressure load and concentric hypertrophy, while insufficiencies are characterized by volume overload and an eccentric hypertrophy of the preceding heart cavities.

Diagnostic procedures typically include ECGs, chest x-ray, and echocardiograms. Management consists of interventional or surgical procedures to reconstruct or replace valves, as well as medical treatment of possible heart failure.

Epidemiology

- **Aortic stenosis**
  - Most common valve defect in industrialized countries
  - Mostly degenerative

- **Aortic regurgitation**
  - Onset at 40–60 years
  - Severity increases with age

- **Mitral stenosis**: symptom onset at 20–30 years
- **Mitral regurgitation**
  - Second most common valve defect
  - More common in women
- **Tricuspid valve** defects occur in < 1% of the population
- **Pulmonary valve** defects rare outside of congenital conditions

Etiology

Valvular heart defects may either be acquired or congenital. Acquired defects are more common and typically occur secondary to infections (postinflammatory), degenerative processes, or heart disease.

		Valve stenosis	Valve regurgitation
Left heart	Mitral valve	• Rheumatic fever • Rheumatic diseases (e.g., SLE, RA)	• Mitral valve prolapse • Dilated cardiomyopathy • Ischemic heart disease (e.g., following myocardial infarction) • Degenerative calcification

	Aortic valve	• Degenerative calcification (most common)
		• Rheumatic endocarditis
		• Congenital (e.g., unicuspid, bicuspid, or hypoplastic valve)
			• Acute: infective endocarditis, aortic dissection type A, chest trauma
			• Chronic
			  • Bicuspid aortic valve
			  • Connective tissue diseases (e.g., Marfan syndrome, Ehlers-Danlos syndrome)
			  • Rheumatic fever
			  • Rheumatic diseases (e.g., Behcet's disease, RA, SLE)

		Valve stenosis	Valve regurgitation
Right heart	Tricuspid valve	• Rheumatic fever • Infective endocarditis (mostly IV drug abuse)	• Right ventricular dilation (e.g., in right-sided heart failure) • Infective endocarditis (IV drug use) • Rheumatic fever • Connective tissue diseases (e.g., Marfan syndrome)
	Pulmonary valve	• Congenital	• Pulmonary hypertension ”**PHTN “** (primary- e.g., tetralogy of Fallot, ventricular septal defects, or secondary PHTN)

Clinical features

All valvular defects can eventually lead to symptoms of heart failure as a result of excessive strain on the ventricles.

- Aortic stenosis
- Aortic regurgitation
- Mitral stenosis
- Mitral regurgitation

Auscultation in valvular defects			
	Maximum point	Murmur	Characteristics
Aortic stenosis	• Aortic valve (parasternal 2nd right intercostal space) • Erb's point	• Harsh crescendo-decrescendo systolic ejection murmur	• Radiation into the carotids • Possibly ejection click
Aortic regurgitation	• Aortic valve (parasternal 2nd right ICS) • Erb's point	• Diastolic murmur with a decrescendo • Possible additional quiet systolic murmur	• Immediately following the 2nd heart sound (“immediate diastolic murmur”) • Austin Flint Murmur

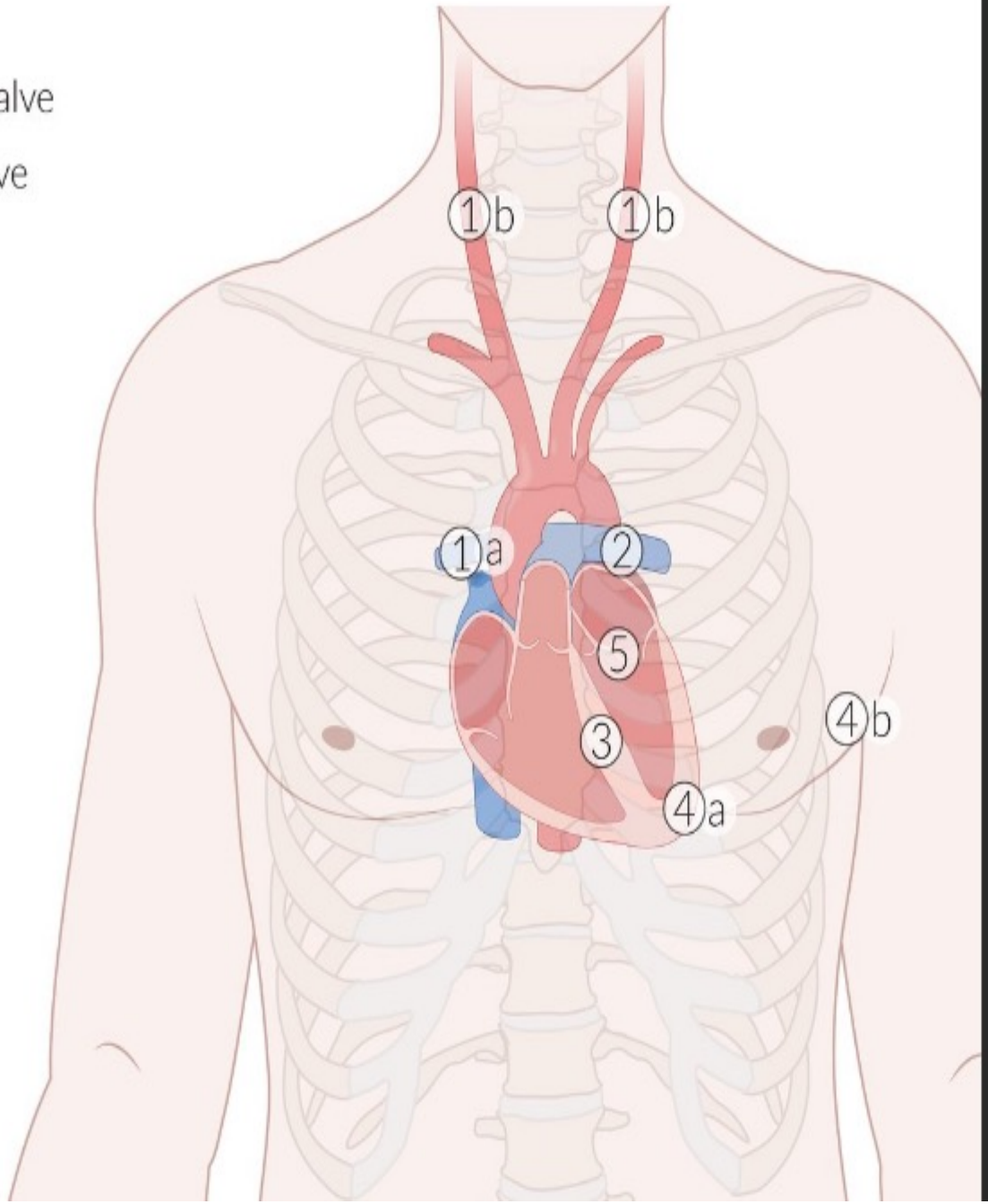
Auscultation in valvular defects			
	Maximum point	Murmur	Characteristics
Mitral stenosis	• Heart apex (midclavicular 5th left ICS)	• Delayed diastolic murmur with a decrescendo with presystolic accentuation	• "Tympanic" 1st heart sound • Mitral opening murmur /opening snap (OS)
Mitral valve prolapse	• Heart apex (midclavicular 5th left ICS)	• Late-systolic crescendo	• Mid-systolic high-frequency click

Auscultation in valvular defects			
	Maximum point	Murmur	Characteristics
Mitral regurgitation	• Heart apex (midclavicular 5th left ICS) • Left axilla	• Holosystolic murmur • 3rd heart sound audible • Quiet 1st heart sound	• Blowing • Radiation into the axilla
Pulmonary stenosis	• Pulmonary valve (parasternal 2nd left ICS)	• Crescendo-decrescendo ejection systolic murmur	• Possible radiation into the back • Possible early systolic pulmonary ejection click and/or widely split 2nd heart sound

Auscultation in valvular defects			
	Maximum point	Murmur	Characteristics
Pulmonary regurgitation	• Pulmonary valve (parasternal 2nd left ICS)	• Diastolic murmur with a decrescendo	• Graham Steel murmur: high-frequency decrescendo diastolic murmur
Tricuspid stenosis (extremely rare)	• Tricuspid valve (parasternal 4th left ICS)	• Delayed diastolic murmur with a decrescendo • Possible pre-systolic crescendo	

Auscultation in valvular defects			
	Maximum point	Murmur	Characteristics
Tricuspid regurgitation (extremely rare)	• Tricuspid valve (parasternal 4th left ICS)	• Holosystolic murmur	• Augmentation of the murmur's intensity with inspiration (Carvallo's sign)

① Aortic valve
② Pulmonary valve
③ Tricuspid valve
④ Mitral valve
⑤ Erb's point



Auscultatory sites of the heart valves

Aortic valve
Auscultation: 2nd right ICS, parasternal line (1a) and carotid arteries (1b)

Pulmonary valve
Auscultation: 2nd left ICS, parasternal line (2)

Tricuspid valve
Auscultation: 4th right ICS, parasternal line (3)

Mitral valve
Auscultation: 5th left ICS, midclavicular line (4a) and left axilla (4b)

Erb's point
Auscultation: 3rd left ICS, parasternal line

Treatment

Symptomatic

- Treatment of heart failure
- Endocarditis prophylaxis
- Prevention of thromboembolism (if necessary)

- **Valve reconstruction (annuloplasty)**
  - Procedure: ring-shaped device attached to the outside of the valve opening to re-establish shape and function of valve
  - Reduced thromboembolic risk compared to mechanical valve replacement; but high risk of recurring stenosis
  - Lower mortality rate than valve replacements, though replacements are more durable

Prosthetic heart valve		
	Mechanical prosthetic valve	Biological prosthetic valve
Pros	• Valve has a long lifespan	• Anticoagulation only necessary for 3 months post operation

Prosthetic heart valve		
	Mechanical prosthetic valve	Biological prosthetic valve
Cons	• Life-long anticoagulation necessary (warfarin, aspirin)	• Short lifespan due to sclerotic degeneration • May need to be replaced every ten years

Prosthetic heart valve		
	Mechanical prosthetic valve	Biological prosthetic valve
Indications	• Younger patients • Previously anticoagulated patients (e.g., with pre-existing atrial fibrillation)	• Older patients • Patients with a high risk of bleeding • Women with a desire to have children

- **Interventional procedures via catheter**
  - Valve replacement, e.g., **transcatheter aortic valve replacement (TAVR)** or **transcatheter mitral valve replacement (TMVR)**
- Percutaneous balloon valvuloplasty for stenoses

Biological prosthetic heart valve

This is a photo of a bovine or porcine, biological prosthetic valve, a xenograft.

Mechanical prosthetic valve

Alloplastic, bileaflet mechanical prosthetic valve.

Aortic valve stenosis

Summary

Aortic stenosis (AS) is a valvular heart disease characterized by narrowing of the aortic valve. As a result, the outflow of blood from the left ventricle into the aorta is obstructed. This leads to chronic and progressive excess load on the left ventricle and potentially left ventricular failure.

The patient may remain asymptomatic for long periods of time; for this reason, AS is often detected late, i.e., when it first becomes symptomatic (dyspnea on exertion, angina pectoris, or syncope). Auscultation reveals a harsh, crescendo-decrescendo murmur in systole that radiates to the carotids, and pulses are delayed with diminished carotid upstrokes.

Echocardiography is the gold standard for diagnosis. Patients with asymptomatic aortic stenosis are treated conservatively. Symptomatic patients or those with severe aortic valve stenosis require valve replacement.

Epidemiology

- Most common valvular heart disease in industrialized countries
- **Prevalence**:
  - Increases with age
  - May reach up to 12.4% among individuals ≥ 75 years

By etiology

- Congenital:
  - **Bicuspid aortic valve**: Caused by a fusion of two of the three aortic-valve leaflets in utero
    - Most common congenital heart defect, 3:1 ♂ predominance
    - Predisposes the valve to **dystrophic calcification** and degeneration
    - Patients present with symptoms of aortic stenosis earlier than in regular aortic valve calcification

- Acquired
  - **Calcific aortic stenosis**: most common cause of aortic stenosis
    - Characterized by calcification and fibrosis of aortic valve leaflets that occur at an increasing rate as patients age (prevalence is 65% in those aged 75–84 years)
    - Similar pathophysiology to atherosclerosis (see generic risk factors for development of arteriosclerosis)
  - Rheumatic fever is a rare cause of AS in developed countries, but continues to remain a significant cause in developing countries.

Pathophysiology

- Narrowed opening area of the aortic valve during systole → obstruction of blood flow from left ventricle (LV) → increased LV pressure → left ventricular concentric hypertrophy →
  - Increased LV oxygen demand
  - Impaired ventricular filling during diastole → left heart failure
  - Reduced coronary flow reserve
- Initially, cardiac output (CO) can be maintained. Later, the decreased distensibility of the left ventricle reduces cardiac output and may then cause backflow into the pulmonary veins and capillaries → higher afterload (pulmonic pressure) on the right heart → right heart failure (congestive heart failure)

Clinical features

- The disease may remain asymptomatic for years (particularly with mild or moderate stenosis).
- Symptoms typically present on exertion, unless AS is severe
- Dyspnea
- **Angina pectoris**
- Dizziness and **syncope**
- Small blood pressure amplitude, decreased pulse pressure

- Cardiac exam (cardiovascular examination)
  - Delayed and weak pulse (**Pulsus parvus et tardus**)
  - Palpable systolic thrill over the bifurcation of the carotids and the aorta

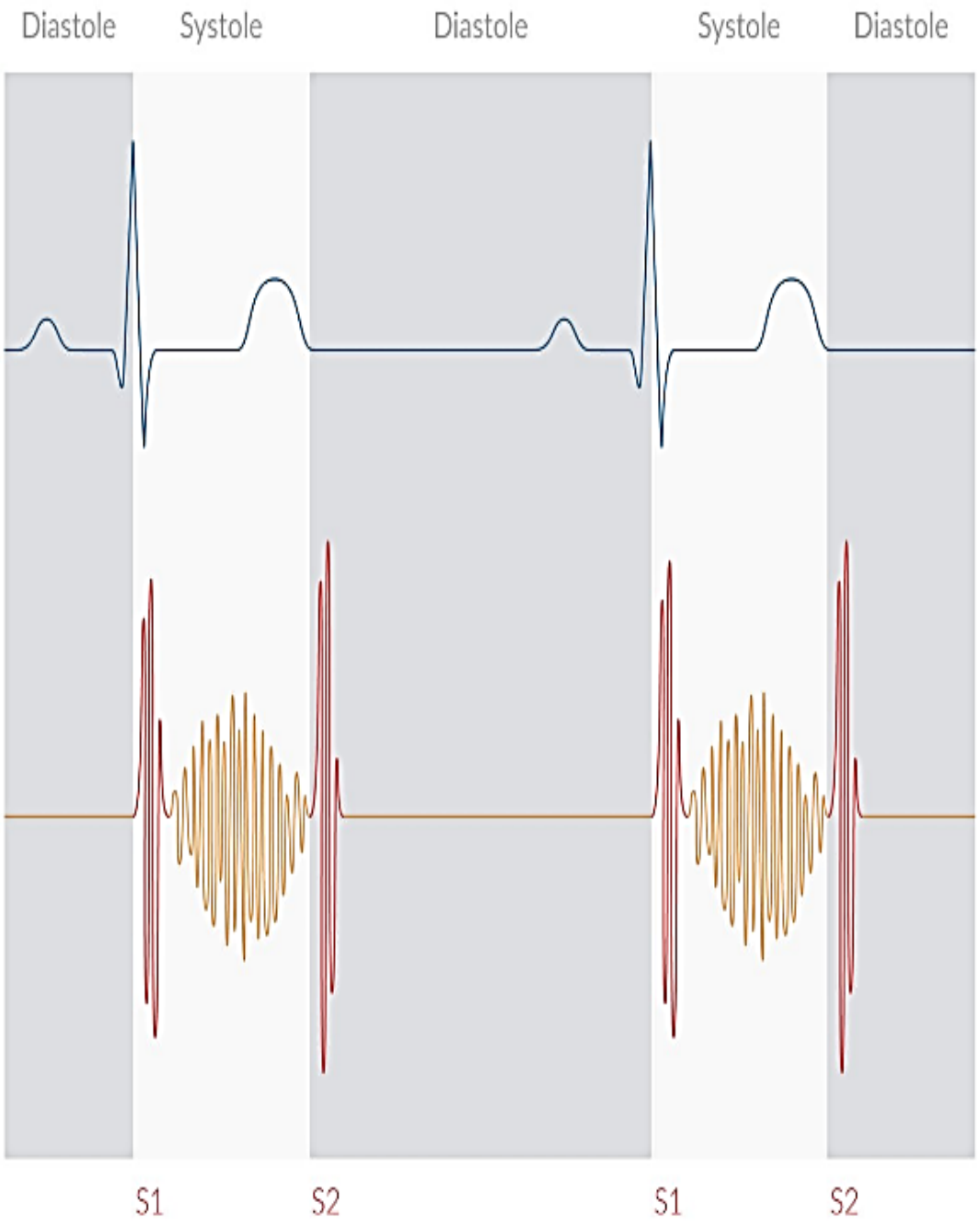
- **Harsh crescendo-decrescendo** (diamond-shaped), **late systolic ejection murmur** that radiates bilaterally to the **carotids**
  - Best heard in the 2nd right intercostal space
  - Hand grip decreases the intensity of the murmur.
  - Valsalva and standing from squatting **decreases** or does not change the intensity of the murmur (in contrast to hypertrophic cardiomyopathy).

- **Soft** S2
- **S4** is best heard at the apex.
- Early systolic **ejection click**
- Frequently associated with aortic regurgitation (see diagnosis of aortic regurgitation)

- Additional signs specific to infants: wheezing and difficulty feeding

SAD (syncope, angina, dyspnea)

Without definite treatment (surgery), more than 50% of the symptomatic patients with severe aortic stenosis will die within the first 2 years of diagnosis!



Heart murmur in hypertrophic obstructive cardiomyopathy, aortic stenosis, and pulmonary stenosis

A crescendo–decrescendo late systolic murmur is depicted. In the case of aortic stenosis, the murmur may radiate to the carotid arteries.

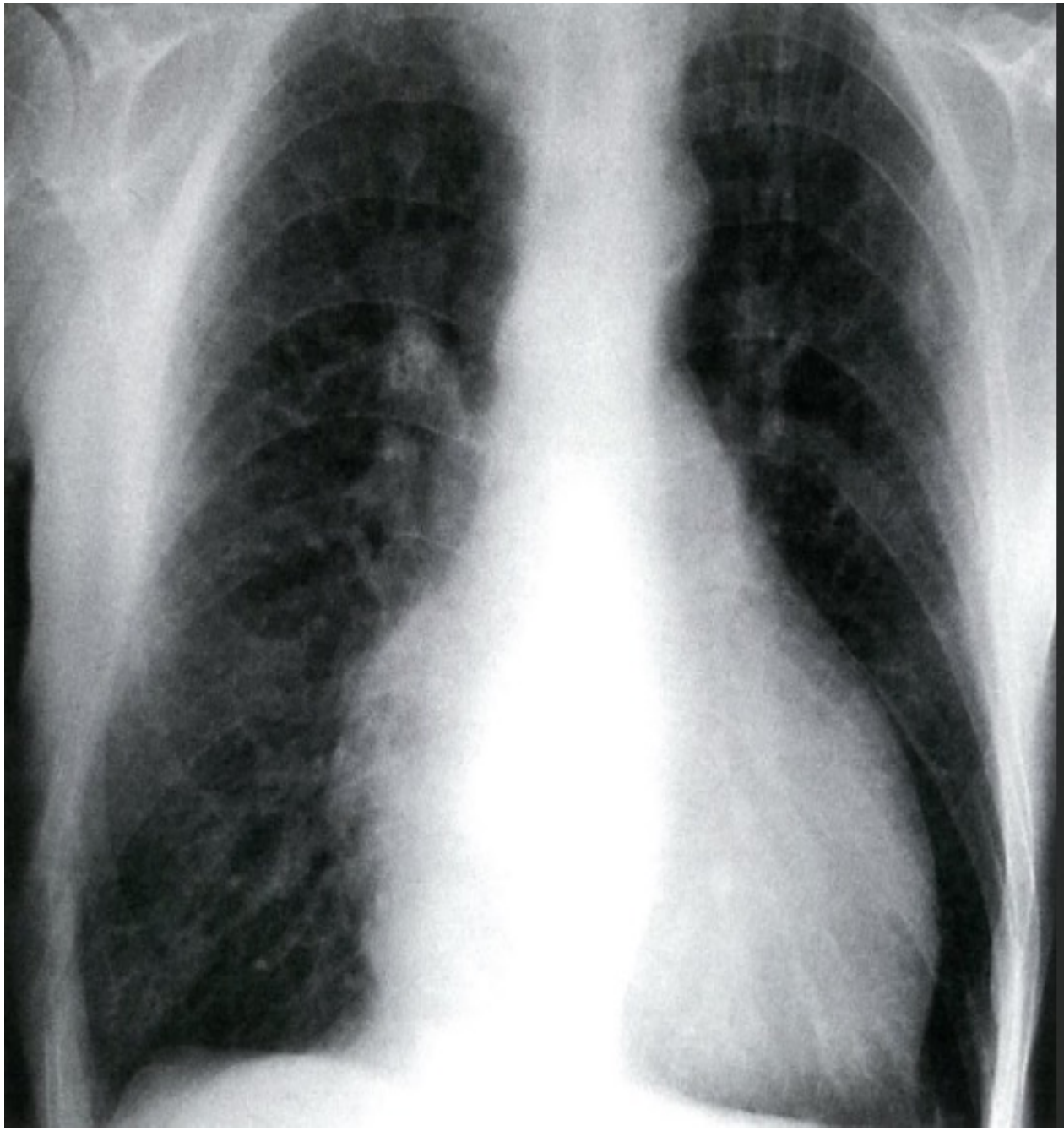
Diagnostics

- **ECG**
  - Nonspecific for AS
  - Signs of left ventricular hypertrophy (e.g., left axis deviation, positive Sokolow-Lyon index)

- **Chest x-ray**
  - Findings of left ventricular hypertrophy, such as left ventricular enlargement and rounded heart apex, usually only in decompensated aortic stenosis, and possibly left atrial enlargement as well
  - Narrowing of retrocardiac space (lateral view)
  - Calcification of aortic valve: signs of more severe disease

- **Echocardiography**
  - Transthoracic (TTE) or transesophageal (TEE): preferred primary test and noninvasive gold standard
    - Findings include concentric hypertrophy, narrowing of the opening of the aortic valve, and increased mean pressure gradient across the aortic valve.
    - Also utilized to determine the severity of stenosis by parameters such as the mean gradient and cross-sectional area of the opening of the valve

- **Left-heart catheterization**
  - Definitive diagnostic test
  - Indication: inconclusive echocardiogram
  - Risk of cerebral embolization



Left ventricular enlargement in aortic valve stenosis

Chest x-ray (PA view)

Enlargement of the heart at its left border can be seen (hatched overlay).

This enlargement is consistent with left ventricular enlargement, which leads to a cardiac-to-thoracic width ratio > 0.5.

Treatment

- **Conservative management**: regular follow-ups indicated for asymptomatic patients with mild aortic stenosis

- **Surgical** (heart valve prostheses)
  - Indications
    - Symptomatic patients
    - Asymptomatic patients with severe AS and either significantly ↓ LV EF

- Aortic valve replacement (AVR): 3 possible approaches
  - Surgical AVR: patients with low surgical risk.
  - Transcatheter AVR (TAVR): patients with high surgical risk or contraindication
  - Catheter balloon valvuloplasty: children without AV calcification

The presence of exertional symptoms (dyspnea on exertion, angina pectoris, syncope) is an indication for surgery!

Prognosis

- Asymptomatic patients: The mortality rate is < 1% in a given year.
- Symptomatic patients: The mortality rate in the first 2 years is > 50%.

Patients with severe AS have fixed cardiac output and fixed afterload, besides being preload dependent. So AF if develops in severe AS patients it will have deleterious effects in these patients .

ACE inhibitors and other afterload-reducing drugs (calcium channel blockers and ACE inhibitors / or ARBs) are contraindicated because they cause dilation of peripheral blood vessels, which may lead to cardiovascular decompensation!
Besides diuretics and hydralazine slould be given cautiously in these patients .

Aortic regurgitation (Aortic insufficiency)

Summary

Aortic regurgitation (AR) is a valvular heart disease characterized by incomplete closure of the aortic valve that leads to reflux of blood from the aorta into the left ventricle (LV) during diastole

Aortic regurgitation may be acute (occurring primarily after bacterial endocarditis or aortic dissection) or chronic (due to congenital bicuspid valve or rheumatic fever) and may be caused by valvular disease or an abnormality of the aorta. In most cases, acute AR leads to rapid deterioration of LV function with subsequent pulmonary edema and cardiac decompensation.

Frequently, chronic AR may remain compensated for a long period of time, becoming symptomatic only when left heart failure develops. Auscultation reveals an S3 and a high-pitched, decrescendo early diastolic murmur. Another characteristic diagnostic finding is widened pulse pressure.

Echocardiography is the most important diagnostic tool, both for confirming the diagnosis and determining the severity of disease. In asymptomatic patients, conservative treatment consists of symptom management and physical activity as tolerated. However, symptomatic patients or those with severely reduced LV function should undergo surgical aortic valve replacement.

Etiology

- **Acute AR**
  - **Infective endocarditis**
  - Aortic dissection (ascending aorta)
  - Chest trauma

- **Chronic AR**
  - **Congenital bicuspid valve**: most common cause of AR in young adults and in **developed countries**
  - **Rheumatic heart disease**: most common cause of AR in **developing countries**
  - Distortion or dilation of the ascending aorta and aortic root
    - Connective tissue disorders (e.g., Marfan syndrome, Ehlers-Danlos syndrome)
    - Tertiary syphilis

Pathophysiology

- General
  - Regurgitation of blood from the aorta into the left ventricle (LV)
    - → Increased systolic blood pressure and decreased diastolic pressure
    - → Widened pulse pressure → **water hammer pulse** (“Diagnostics” below)

- Acute AR
  - Because LV cannot sufficiently dilate in response to regurgitant blood, LV end-diastolic pressure increases rapidly → pressure transmits backwards into pulmonary circulation → pulmonary edema and dyspnea
  - Decreased cardiac output if severe → cardiogenic shock and myocardial ischemia

- Chronic AR:
  - Initially, a **compensatory increase in stroke volume** can maintain adequate cardiac output despite regurgitation (compensated heart failure)
  - Over time, increased left ventricular end-diastolic volume → LV enlargement and **eccentric hypertrophy of myocardium** → left ventricular systolic dysfunction → decompensated heart failure

Clinical features

- **Acute AR**
  - Sudden, severe **dyspnea**
  - Rapid cardiac decompensation secondary to heart failure
  - **Pulmonary edema**
  - Symptoms related to underlying disease (e.g., fever due to endocarditis, chest pain due to aortic dissection)

- **Chronic AR**
  - May be asymptomatic for up to decades despite progressive LV dilation
  - **Palpitations**
  - **Symptoms of left heart failure**
    - Exertional **dyspnea**
    - **Angina**
    - Orthopnea
    - Easy fatigability
    - **Syncope**
  - Symptoms of high pulse pressure (e.g., head pounding, rhythmic nodding, or bobbing of the head in synchrony with heartbeats- de Musset sign)

Diagnostics

Physical examination

- For detailed information about the individual tests, (cardiovascular examination).
- **High pulse pressure**
  - **Water hammer pulse** of peripheral arteries characterized by rapid upstroke and downstroke
  - Pulsing of carotid arteries with rapid upstroke and downstroke
  - Visible capillary pulse
  - Nodding of the head with each pulse
- Point of maximal impulse (PMI): displaced inferolaterally, diffuse, and hyperdynamic

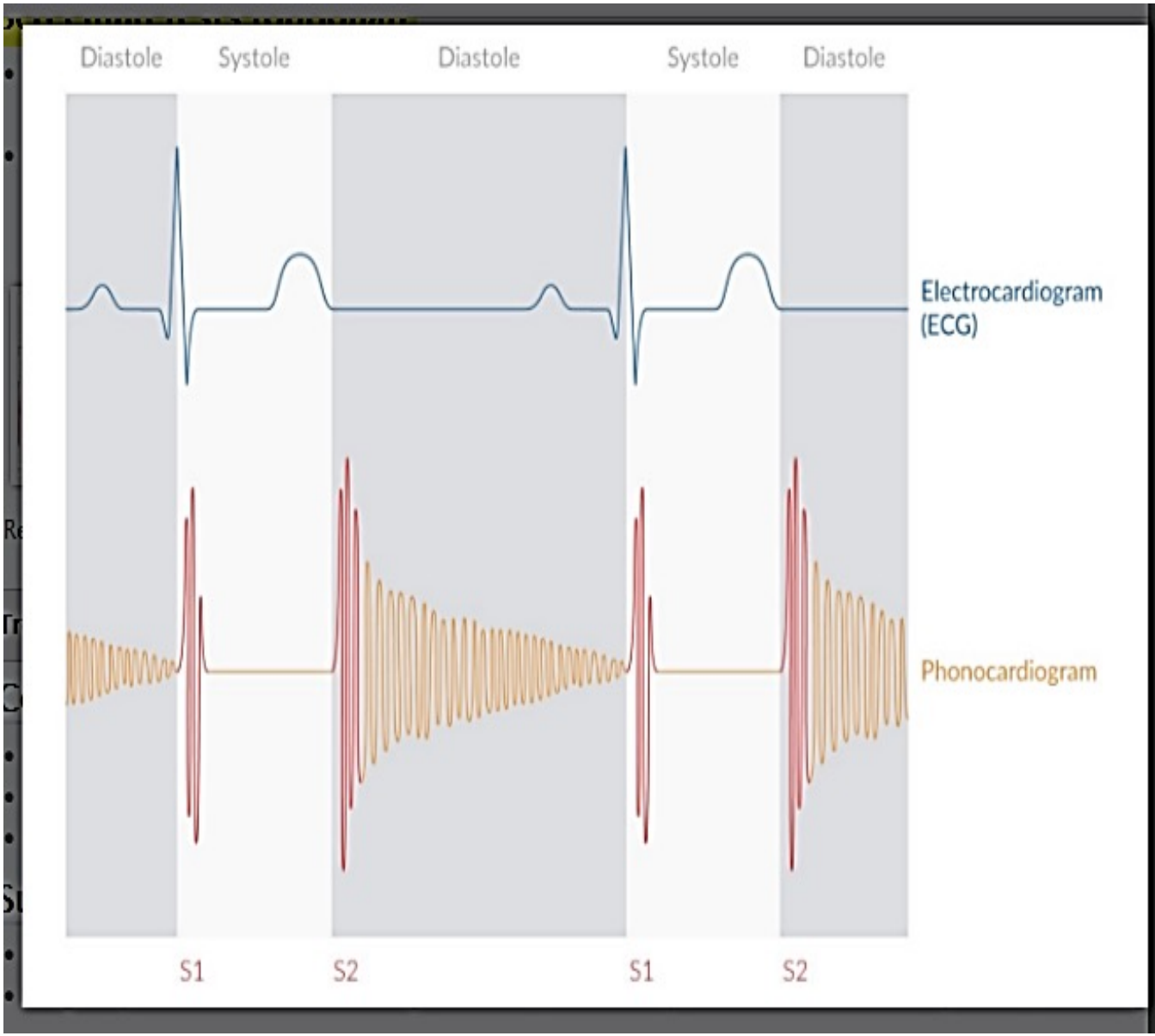
- **Auscultation**
  - **S3**
  - **High-pitched, blowing, decrescendo early diastolic murmur**
    - AR due to valvular disease: best heard in the left third and fourth intercostal spaces and along the left sternal border
    - AR due to aortic root disease (e.g., aortic dissection): best heard along the right sternal border
    - Worsens with squatting and handgrip
  - **Austin Flint murmur** (Apical mid-diastolic or presystolic murmur).
  - In more severe stages, possibly a **harsh, crescendo-decrescendo mid-systolic murmur** that resembles the ejection murmur heard in aortic stenosis

Confirmatory tests

- **Transthoracic echocardiogram (TTE)**
  - Indicated for suspected AR as well as to monitor confirmed AR to determine optimal timing of surgery
  - Findings
    - Abnormal valve leaflets
    - Regurgitant AR jet on Doppler
    - Increased LV size and volume
    - LV ejection fraction < 55%
- Transesophageal echocardiogram (TEE): indicated if suboptimal or nondiagnostic TTE

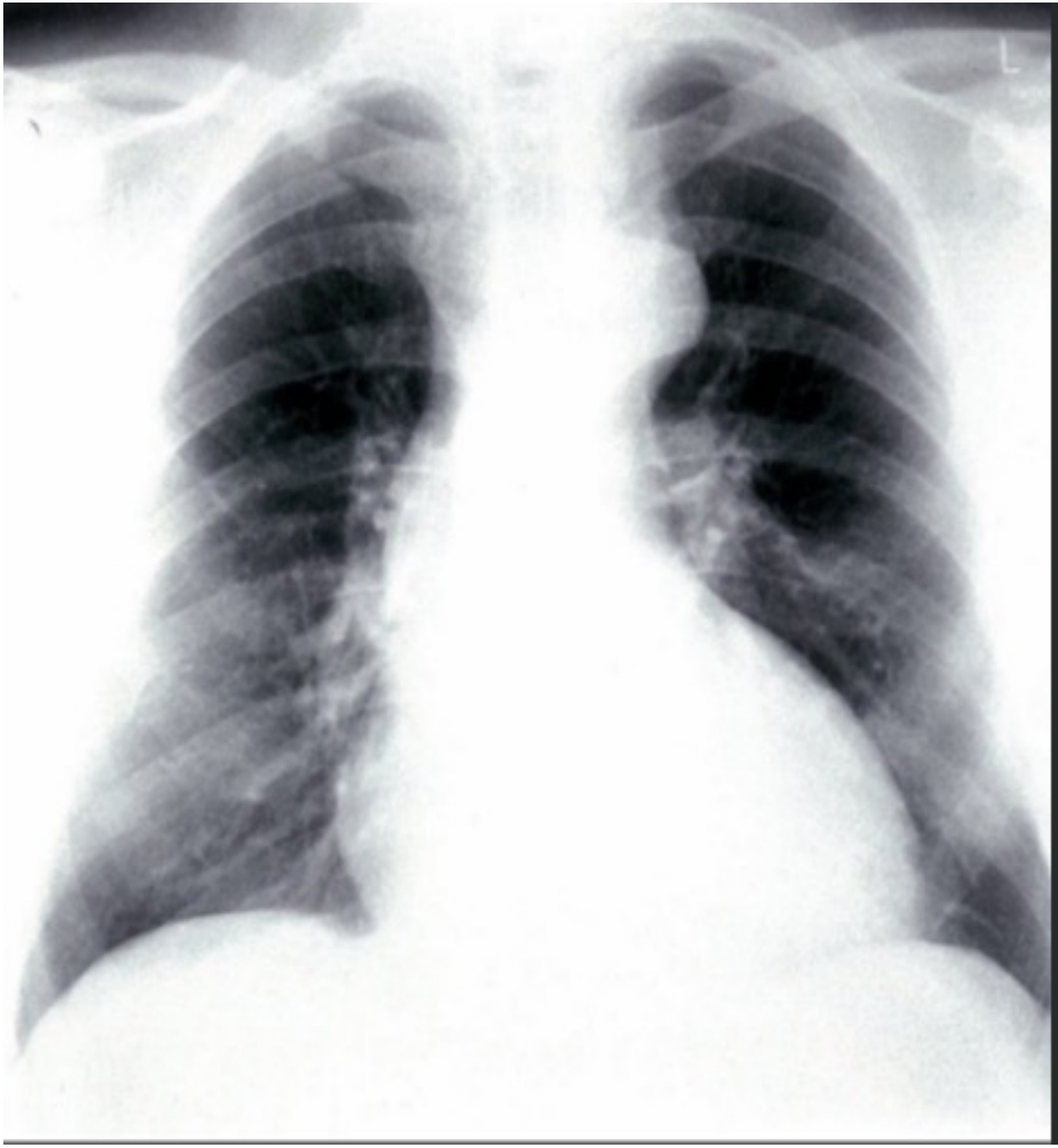
Screening tests (optional)

- ECG
  - Signs of left ventricular hypertrophy
- Chest x-ray
  - Prominent aortic root/arch
  - Enlarged cardiac silhouette



Heart murmur in aortic and pulmonary insufficiency

Early–diastolic decrescendo murmur (directly after the second heart sound), consistent with aortic regurgitation and/or pulmonary regurgitation.



Aortic regurgitation

Posteroanterior chest x-ray: enlargement of the left ventricle and accenuated left heart border, accompanied with dilation of the ascending aorta and dilation and elongation of the aortic arch.

Treatment

- **Conservative**
  - Indication: asymptomatic patients and symptomatic patients who are not candidates for surgical treatment
  - **Treatment of heart failure**
  - Physical activity , but without excessive straining
  - Remember that patients with severe AR are sensitive to bradycardia and drugs that cause bradycardia.

- **Surgical**
  - Indication: patients with acute severe AR and chronic AR with symptoms or with significantly reduced pump function
  - Surgical procedure: **aortic valve replacement** (occasionally valve reconstruction is possible) and long-term anticoagulation therapy for mechanical valve

Prognosis

- Asymptomatic patients with normal EF: progression to symptoms or LV dysfunction at a rate of < 6% per year
- Asymptomatic patients with decreased EF: progression to symptoms at a rate of > 25% per year
- Symptomatic patients: mortality rate is > 10% per year

Mitral valve stenosis

Summary

Mitral stenosis (MS) is a valvular anomaly of the mitral valve that leads to obstruction of blood flow into the left ventricle. The most common cause of MS is rheumatic fever. The clinical manifestations depend on the extent of stenosis: reduced mitral opening leads to progressive congestion behind the stenotic valve. Initial dilation of the left atrium (complications: atrial fibrillations, emboli) is followed by progressive congestion of the lungs and subsequent cardiac asthma (coughing, dyspnea).

Acute decompensation can cause pulmonary edema. Echocardiography is the main diagnostic tool for evaluating the mitral valve apparatus, left atrial size, and pulmonary pressure. In the event of high grade and/or symptomatic stenosis, percutaneous valvuloplasty or surgical valve replacement is often required.

Etiology

- Most commonly due to **rheumatic fever**
- Autoimmune diseases: **systemic lupus erythematosus**, rheumatoid arthritis
- Congenital
- Some conditions may mimic mitral stenosis: bacterial endocarditis of the mitral valve with large vegetation, left atrial myxoma

Pathophysiology

- Mitral valve stenosis → **obstruction** of blood flow into the left ventricle (LV) → limited diastolic filling of the LV (**↓ end-diastolic LV volume**) → decreased stroke volume → decreased cardiac output (**forward heart failure**)
- Mitral valve stenosis → increase in left atrial pressure → backup of blood into lungs → increased pulmonary capillary pressure
  → **cardiogenic pulmonary edema** → pulmonary hypertension → backward heart failure and right ventricular hypertrophy

Clinical features

- Initially asymptomatic (onset ~ 10 years after acute rheumatic carditis)
- **Dyspnea** (paroxysmal nocturnal dyspnea) and orthopnea, especially when supine
- **Hemoptysis**
- Atrial fibrillation
- Later stages: signs and symptoms of right-sided heart failure

Diagnostics

- **Auscultation** (auscultation in valvular defects)
  - **Diastolic murmur** typically heard best at the **5th left intercostal space at the mid-clavicular line (the apex)**
    - Heard loudest when the patient is lying on his/her left side.
  - **Loud first heart sound (S_1)**
  - **Opening snap** of the mitral valve after S_2; **shorter interval** between opening snap and S_2 indicative of more **severe disease**, because left atrial pressure is greater than left ventricular end diastolic pressure (LVEDP)

- **Chest x-ray**
  - **Posterior-anterior image**
    - **LA enlargement** with prominent left auricle (left atrial appendage) → straightening of the left cardiac border
    - Signs of pulmonary congestion (see X-ray findings in pulmonary congestion)
  - **Lateral image**
    - Dorsal displacement of the esophagus (visible in barium swallow test)
    - Signs of right ventricular hypertrophy

- **ECG**
  - **P mitrale**
  - Atrial fibrillation
  - Signs of right ventricular hypertrophy (Sokolow-Lyon index)

- **Echocardiography**: most important diagnostic method for detecting and assessing valvular abnormalities
  - Abnormal valve mobility
  - Subvalvular thickening
  - Leaflet thickening
  - **Calcification**
- Coronary angiography may be conducted prior to surgical interventions to assess the associated risk of coronary artery disease

Treatment

- **Conservative treatment**
  - Treatment of heart failure: only **diuretics** may be administered!
  - Beta blockers or calcium channel blockers: ↓ heart rate and ↓ cardiac output
  - Endocarditis prophylaxis (“Infective endocarditis”)

- Remember that patients with severe MS are sensitive to tachycardias and drugs that cause it. So AF has deleterious effects in these patients.

- **Interventional**
  - Indication: high grade and/or symptomatic mitral stenosis
  - First-line: **percutaneous balloon commissurotomy** of the mitral valve
  - Alternatives: **open commissurotomy** and **surgical valve replacement** (mechanical prosthetic valve or biological prosthetic valve)

Complications

- **Atrial fibrillation** → thromboembolic events
- Progressive congestion of the lungs, **pulmonary edema, pulmonary hypertension**
- **Congestive heart failure**
- Enlarged left atrium (rare) → esophageal compression, recurrent laryngeal nerve palsy

Mitral regurgitation
(Mitral valve regurgitation)

Summary

Mitral regurgitation (MR) refers to the leakage of blood from the left ventricle to the left atrium due to incomplete closure of the mitral valve. The most common causes are primary diseases of the valve (e.g., mitral valve prolapse), although damage may also result secondary to other heart conditions such as left ventricular dilation and myocardial infarction.

Symptoms such as palpitations or dyspnea appear late in the course of chronic compensated MR in which cardiac output can still be maintained. In acute or chronic decompensated MR, pulmonary edema and pulmonary hypertension often cause dyspnea, coughing, jugular venous distention, and pitting edema.

Echocardiography is the most important diagnostic tool and allows for assessment of severity. It may also be used for preoperative evaluation. Treatment options include surgical mitral valve repair or replacement and percutaneous reconstruction. Early intervention is favored in most cases of MR with evidence of left ventricular dysfunction, regardless of symptoms.

Etiology

- **Primary MR**
  - Degenerative mitral valve disease (**mitral valve prolapse**, mitral annular calcification, ruptured chordae tendineae)
  - **Rheumatic fever**
  - **Infective endocarditis**

- **Secondary (functional) MR**
  - **Coronary artery disease**, prior myocardial infarction causing papillary muscle involvement
  - Dilated cardiomyopathy and left-sided heart failure

Pathophysiology

- **Acute MR** → ↑ LV end-diastolic volume → rapid ↑ LA and pulmonary pressure → pulmonary venous congestion → pulmonary edema
- **Chronic (compensated) MR**: progressive dilation of the LV (via eccentric hypertrophy) → ↑ volume capacity of the LV (preload and afterload return to normal values) → ↑ end-diastolic volume → maintains ↑ stroke volume (normal EF)

- **Chronic (decompensated) MR**: progressive LV enlargement and myocardial dysfunction → ↓ stroke volume → ↑ end-systolic and end-diastolic volume → ↑ LV and LA pressure → pulmonary congestion, possible acute pulmonary edema, pulmonary hypertension, and right heart strain

Clinical features

- **Acute mitral regurgitation**
  - Symptoms of left-sided heart failure
  - Signs and symptoms of pulmonary edema
  - Cardiogenic shock

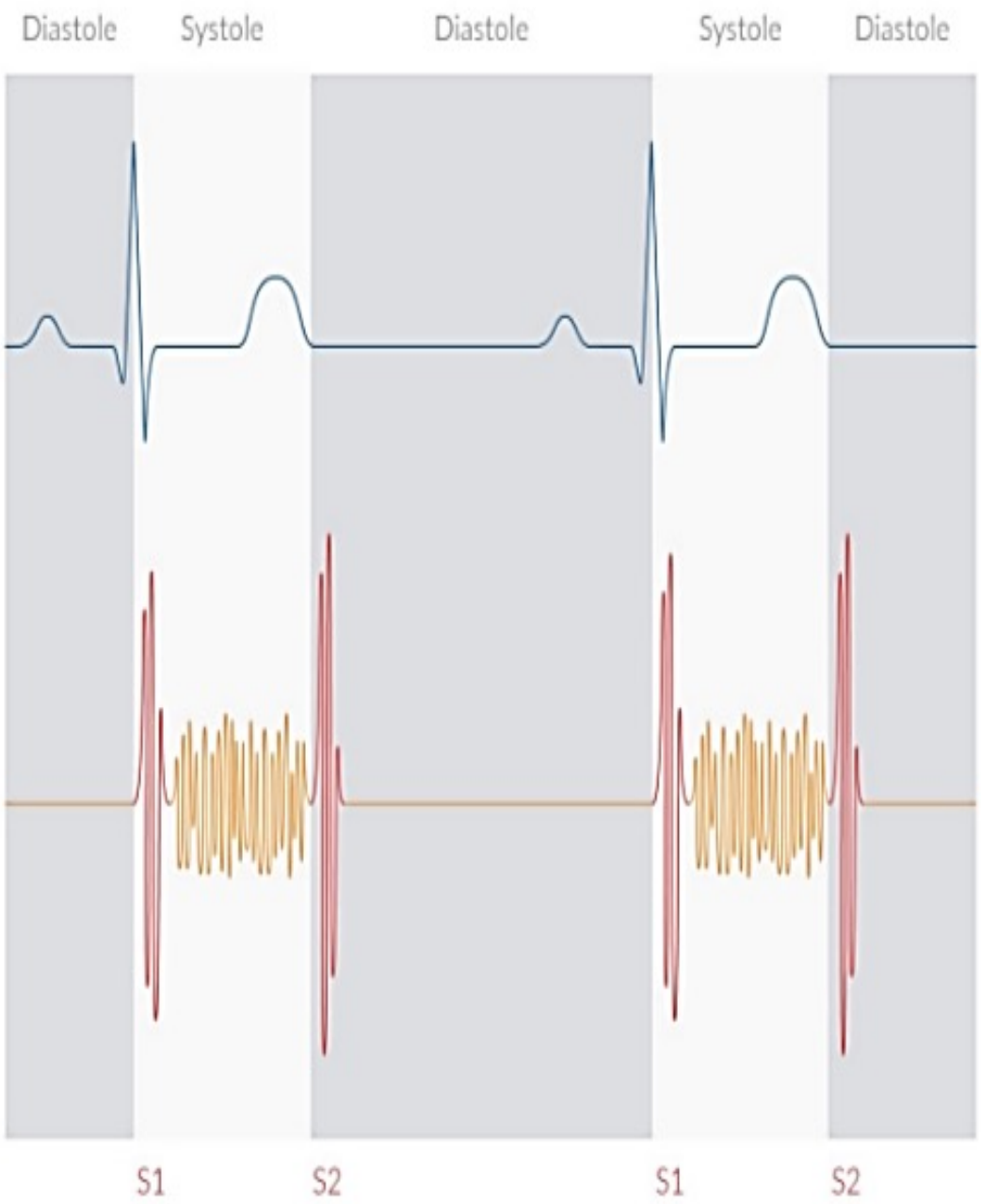
- **Chronic mitral regurgitation**: late onset of symptoms
  - (Exertional) dyspnea, dry cough
  - **Palpitations**
  - Symptoms of left-sided heart failure

Diagnostics

- **Auscultation**
  - "Auscultation in valvular defects"
  - Quiet first heart sound (S1)
  - S3 gallop in advanced stages of disease
  - **Holosystolic murmur**
    - Radiates to the left axilla and heard loudest over the apex (5th ICS at the left mid-clavicular line)
    - Intensity increases with increased systemic vascular resistance (hand grip, squatting)
  - Cardiac impulse is often prominent

- **ECG**
  - **Left cardiac hypertrophy and P mitrale**
  - Later, signs of right heart strain with P pulmonale
- **Chest X-ray**
  - Posterior-anterior image
    - **LV enlargement**: laterally displaced left cardiac border
    - **LA enlargement**: straightening of the left cardiac border
    - Signs of pulmonary congestion in late stages of disease (“X-ray findings in pulmonary congestion”)
  - Lateral image: Narrowing of the retrocardiac space

- **Echocardiography**: **most important** diagnostic method for detecting and assessing valvular abnormalities
  - Valve apparatus (e.g., dimensions of valve opening area, calcification, rupture of the chordae tendineae) and mobility
  - LV and LA size and function
- **Coronary angiography**: prior to surgical intervention



Heart murmur in ventricular septal defect, mitral regurgitation, and tricuspid regurgitation

Holosystolic murmur (often referred to as "pansystolic murmur" in the case of a ventricular septal defect)

Myocardial infarction must be ruled out in patients presenting with acute MR!

Treatment

- **Acute mitral regurgitation**
  - Hemodynamic stabilization: diuretics, nitrates, antihypertensive drugs
  - Intra-aortic balloon pump if pharmacologic management is insufficient

  - Urgent surgical **valve repair or replacement**

- **Chronic mitral regurgitation**
  - **Asymptomatic patients**
    - Without evidence of LV dysfunction (EF > 60%): conservative management (regular follow-ups, avoidance of physical exertion), management of underlying and/or secondary diseases
    - With evidence of LV dysfunction (EF < 60%): early surgical valve repair or replacement

- **Symptomatic patients**
  - Without evidence of severe LV dysfunction (EF > 30%): early valve repair or replacement (mechanical prosthetic valve or biological prosthetic valve)
  - With severe LV dysfunction (EF < 30%): surgical valve repair or replacement only if the patient is hemodynamically stable ;
  - Alternatives for severe LV dysfunction:
    - Percutaneous reconstruction
    - Medical management of heart failure

Complications

- Heart failure, cardiac decompensation, and pulmonary edema
- **Atrial fibrillation** and arterial emboli
- Endocarditis