4. 4. 1. Aortic stenosis

**Definition.** Reduction of the aortic valve opening due to pathological processes that lead to thickening, fibrosis and calcification of the cusps and therefore to an obstruction of the left ventricular outflow tract.

**Etiology.** The aortic stenosis may be congenital or acquired (rheumatic or degenerative). In younger patients most of them are congenital lesions (bicuspid or monocuspid valve) that undergo a process of fibrosis and calcification in time. Although rare nowadays in the developed countries, the rheumatic etiology is still frequent throughout the world: a process of fibrosis leads to fused commissures. The degenerative senile etiology is becoming the most frequent one: it affects older people by a process of calcification that immobilizes the aortic cusps.

![Fig 4.1 Severe calcified aortic stenosis in an old patient. (Department of Cardiac Surgery, Innsbruck, Austria)](image)

**Pathophysiology.** By obstructing the left ventricular (LV) ejection, the aortic valve stenosis causes an increase of the afterload and therefore of the heart labor, expressed by a huge pressure gradient between LV and aorta. The heart reacts by concentric left ventricular hypertrophy that enables LV to overcome the obstacle, the compensated stage. When this compensatory mechanism exhausts in time, the dilatation of the LV occurs, leading to important decrease of the systolic function and to heart failure phenomena – the decompensated stage.

**Clinical aspect.** Three main symptoms may occur in patients with aortic stenosis: dyspnea, angina and syncope. The exercise may produce any of the above symptoms, but may also lead to sudden death. In the late, decompensated stage, heart failure appears, with atrial fibrillation, cerebral or systemic embolism from the calcium deposits and secondary pulmonary hypertension. There is always the risk of infection and endocarditis. Some patients remain asymptomatic for a longer time, although the aortic stenosis became severe: even in such cases there is the risk of sudden death, therefore a correct follow-up and treatment is important. **Degree of aortic stenosis.** Normally aortic orifice area in adults is 3-4 cm². In a mild aortic stenosis the effective orifice area has less than 2cm², between 1 and 1.5 cm² is moderate, while for less than 1 cm² the aortic stenosis is
severe. In case of severe stenosis with normal cardiac output, mean transvalvular gradient is increased above 50 mmHg.

**Natural evolution.** Patients with AS remain asymptomatic for years despite a serious obstruction. Though, 40% of patients with hemodynamically significant stenosis develop symptoms within the next 1-2 years. The installment of the symptoms (dyspnea, angina and syncope after a long period of evolution of the stenosis marks the beginning of cardiac decompensation and time for a therapeutical decision to avoid sudden death and to prolong survival, otherwise reduced to a few years. Most studies have shown reduced incidence of 1% of sudden death in asymptomatic patients, but more than 20-30% for the symptomatic. Life expectancy is 1-2 years for patients with heart failure phenomena, 2-3 years for those with syncope and 4-5 years for those with angina. Dintre pacientii cu stenoza aortica severa, tratati medical, 50% decedeaza la 2 ani, jumatate dintre ei prin moarte subita. Knowledge of the natural evolution helps us make the most appropriate decisions in a certain moment of the disease.

**Clinical and paraclinical diagnosis.** Diagnosis by physical examination of the patient is easily made by characteristic elements: intense, rough systolic murmur in the upper right sternal border that irradiates to the neck vessels, sustained apex beat, slow upstroke and low volume arterial pulse “parvus et tardus”. Routine ECG and chest X-ray is indicated. **Echocardiographic study,** by M-mode, two-dimensional and Doppler confirms and permits staging of the evolving aortic stenosis.

Table 4.1 Information brought by echocardiography in aortic stenosis.

<table>
<thead>
<tr>
<th>Information brought by echocardiography in aortic stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• It specifies the diagnosis and severity of the aortic stenosis; it calculates the transaortic gradient and the aortic orifice area, it detects calcifications on the aortic annulus, on the cusps and on the ascending aorta.</td>
</tr>
<tr>
<td>• It assesses the heart size and function: the systolic and diastolic LV diameters and volumes, the degree of the LV hypertrophy and the ejection and shortening fractions.</td>
</tr>
<tr>
<td>• It evaluates the other valves(mitral, tricuspid) and measures the pulmonary hypertension if present</td>
</tr>
<tr>
<td>• Periodic follow-up of asymptomatic patients</td>
</tr>
<tr>
<td>• Follow-up of the hemodynamic changes in pregnant women with AS.</td>
</tr>
</tbody>
</table>

Exercise test is not indicated in patients with severe SA because it may precipitate the symptoms and may even lead to sudden death. **Cardiac catheterization.** It is no longer necessary in all patients with AS, the main diagnostic tool is nowadays the echocardiography. Though, when there are discrepancies between clinical factors and echocardiographic data cardiac catheterization is indicated, accurately measuring
transvalvular flow, the gradient between the LV and the aorta, calculating the effective aortic orifice area and the LV function. Angiocoronarography is nevertheless performed in all patients proposed for surgical intervention that have risk factors for coronary disease, to assess the coronary system.

**Medical and surgical treatment**

Medical treatment is of reduced importance in patients with AS. Endocarditis prophylactic measures are taken and restricted sodium diet and restriction of the physical exercise are imposed. Associated hypertension is also handled accordingly.

Indications for surgery. Evolution in patients with severe AS, medically treated is worse, with 50% mortality at two years, half of them by sudden death.

**Table4.2 Indications for surgery in aortic stenosis**

<table>
<thead>
<tr>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Severe AS in neonates and infants, by balloon dilatation or emergency surgery</td>
</tr>
<tr>
<td>• Severe symptomatic AS at any age</td>
</tr>
<tr>
<td>• Moderate AS in patients with coronary lesions associated that have an indication for revascularization.</td>
</tr>
<tr>
<td>• Moderate AS in patients with associated valvular lesions associated that have an indication for surgery.</td>
</tr>
<tr>
<td>• Asymptomatic patients with severe AS that present with ventricular dysfunction, severe left ventricular hypertrophy, ventricular tachycardia or abnormal response to exercise</td>
</tr>
</tbody>
</table>

**Surgical treatment**

With the use of the cardiopulmonary by-pass and the substitutes for the aortic valve in the 60’s, surgery became the treatment of choice for aortic stenosis. The goal of surgery is to eliminate the symptoms and the risk of sudden death, to increase the life expectancy and the quality of life, with active reinsertion of the patient within family and society.

**Surgical technique.**

Aortic valve replacement or repair is made using the cardiopulmonary by-pass CBP. The classical approach uses median sternotomy, cannulation of the ascending aorta, single venous cannula inserted through the right atrium, normothermic or moderately hypothermic blood cardioplegia administered antegradeley through the coronary ostia or retrogradely through the coronary sinus. Aortic valve is excised, the aortic orifice is measured and valve prosthesis is chosen according to the size and is sutured with separate stitches with or without pledgets. Valve selection is based on patient’s age, aortic valve pathology, surgeon’s and patient’s preferences. At neonates, infants and children it is desirable to try a conservative method of repairing the valve or dilating it by balloon
valvulotomy. Likewise, for the elderly and the very high risk patients, balloon valvulotomy may be the procedure of choice.

Fig. 4.2 Surgical technique for aortic stenosis

**Operative risk.** It should be less than the risk of death by natural evolution of the disease. It is based on the following parameters:

Table 4.3 Operative risk factors for surgery in aortic stenosis

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elective or emergency surgery</td>
<td>(endocarditis, pulmonary edema)</td>
</tr>
<tr>
<td>Age over 70 years</td>
<td></td>
</tr>
<tr>
<td>NYHA functional class III-IV</td>
<td></td>
</tr>
<tr>
<td>Severely impaired left ventricular EF &lt;40%</td>
<td></td>
</tr>
<tr>
<td>Association of coronary artery disease (CAD)</td>
<td></td>
</tr>
<tr>
<td>Association with other valvular lesions (mitral, tricuspid)</td>
<td></td>
</tr>
<tr>
<td>Chronic atrial fibrillation</td>
<td></td>
</tr>
<tr>
<td>History of stroke</td>
<td></td>
</tr>
<tr>
<td>Associated renal or hepatic failure</td>
<td></td>
</tr>
<tr>
<td>Reintervention for aortic valve prosthesis clogging or endocarditis of the prosthesis</td>
<td></td>
</tr>
</tbody>
</table>

**Early and late complications. Results**

Operative mortality in elective surgery is lower than 3%, increasing to extreme ages (infants, elderly), in case of emergency surgery, in case of endocarditis or if associated pathology is present. Early postoperative complications are dominated by bleeding, arrhythmia, cerebral embolic stroke, low cardiac output syndrome, renal failure and respiratory failure. Survival is 75% at 5 years and 60% to 10 years, with the risk of prosthetic endocarditis, complications of anticoagulant treatment, bleeding or valve clogging, cerebral or peripheral embolism. All these complications depend on patient
parameters before surgery (cardiac function, age), surgical technique and postoperative care of the patient

4. 4. 2 Aortic regurgitation

Definition. Due to pathological processes, aortic cusps fail to close the aortic orifice during ventricular diastole, allowing a certain amount of blood to return into LV.

Etiology. Pathologic processes may affect various constituents of the aortic root resulting in an acute or chronic aortic regurgitation:

Table 4.4 Etiology of acute and chronic aortic regurgitation:

<table>
<thead>
<tr>
<th>Acute aortic regurgitation</th>
<th>Chronic aortic regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Acute aortic dissection</td>
<td>• Rheumatic</td>
</tr>
<tr>
<td>• Endocarditis</td>
<td>• Congenital</td>
</tr>
<tr>
<td>• Trauma</td>
<td>• Aneurysm of ascending aorta</td>
</tr>
<tr>
<td>• Postinterventional</td>
<td>• Marfan syndrome</td>
</tr>
<tr>
<td></td>
<td>• Annular ectasia</td>
</tr>
<tr>
<td></td>
<td>• Longterm severe arterial hypertension</td>
</tr>
</tbody>
</table>
**Clinical aspect.** Patients with chronic regurgitation are asymptomatic for a long time. When symptoms appear, the patients have dyspnea, palpitations, fatigue, syncope, heavy sweating and angina. Physical findings include: rapidly upstroke and collapsing arterial pulse, celer and altus (Corrigan’s pulse), de Musset’s sign (rhythmic nodding of the head), low diastolic arterial pressure, large “en dome” apex beat and soft, aspirative diastolic murmur in the aortic area.

**Paraclinical diagnosis.**

Chest X-ray highlights the size of LV and of the aorta. In early forms is normal, then the lower left arch gets longer expressing the LV increase in size, ascending aorta dilates, the pulmonary fields become loaded due to pulmonary stasis. ECG may be normal in the first stage, later on LV hypertrophy, conduction disturbances atrial fibrillation and even ischemic changes may appear.

**Echocardiography.** Transthoracic and transesophageal echocardiography are the key methods in diagnosing aortic regurgitation:

Table 4.5 Value of echocardiography in assessment of aortic regurgitation.

<table>
<thead>
<tr>
<th>Value of echocardiography in assessment of aortic regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Confirms and assesses the severity of acute aortic regurgitation</td>
</tr>
<tr>
<td>• Confirms the diagnosis of chronic aortic regurgitation</td>
</tr>
<tr>
<td>• May specify the etiology of regurgitation, may detect aortic dissection, endocarditis with vegetation</td>
</tr>
<tr>
<td>• Assesses effects on the LV size, enddiastolic pressure, ejection fraction</td>
</tr>
<tr>
<td>• Evaluates the other valves (mitral, tricuspid, pulmonary) and quantifies the pulmonary hypertension</td>
</tr>
<tr>
<td>• Periodical evaluation of asymptomatic patients until the moment of surgical indication</td>
</tr>
<tr>
<td>• Postoperative follow-up, showing fecoery of LV function or the functional state of valve prosthesis</td>
</tr>
</tbody>
</table>

**Coronary angiography.** It is performed routinely in all patients over 40 years to investigate the coronary system, to visualize an eventual aortic root aneurysm or to detect any carotid or peripheral arterial lesions. If cardiac catheterization is performed, the severity of the aortic regurgitation may be assessed, along with pressures and even pulmonary resistances.

**Natural evolution.** It depends on the etiology, on the onset of regurgitation and on the occurrence of cardiovascular complications. In case of acute aortic regurgitation, the evolution is severe towards cardiogenic shock and death unless urgent action is taken. Postrheumatic aortic alterations occur after a few years of evolution, then for a long time patients are asymptomatic and when they become symptomatic, this will lead to exitus in...
another 10 years. The mortality rate in symptomatic patients is higher than 10% / year. The risk of infection on such pathologic aortic valve complicates furthermore the evolution of these patients.

**Medical Treatment** Asymptomatic patients do not require any treatment in the absence of hypertension; only recommended general measures, dietary elimination of cardiovascular risk factors (salt, smoking and obesity), infectious endocarditis prophylaxis and regular monitoring by a cardiologist. If the patient becomes symptomatic, moderate vasodilator therapy, with ACE inhibitors, may be administered, but the patient needs to be reevaluated for surgery before severe decompensation of the heart occurs.

**Surgical treatment** Optimal timing of surgery is more difficult than for aortic stenosis due to the long asymptomatic evolution, therefore the indication for surgery in asymptomatic patients with severe regurgitation is controversial. On the other hand, once the cardiac decompensation advances, it may reduce the chances of postoperative recovery of cardiac function and of survival after surgery.

Table 4.6 Surgical indications in aortic regurgitation

<table>
<thead>
<tr>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic patients with</td>
</tr>
<tr>
<td>- Progressive increase of heart size – X-ray, echocardiography</td>
</tr>
<tr>
<td>- Progressive LV hypertrophy - ECG, echocardiography</td>
</tr>
<tr>
<td>- Reduction of EF &lt; 45%, SF &lt; 25%</td>
</tr>
<tr>
<td>- Enddiastolic diameter&gt; 70 mm, endsystolic diameter&gt; 55 mm</td>
</tr>
<tr>
<td>Symptomatic patients with angina pectoris and heart failure</td>
</tr>
<tr>
<td>Acute aortic regurgitation</td>
</tr>
<tr>
<td>Acute bacterial endocarditis with severe regurgitation and risk of septic embolism</td>
</tr>
</tbody>
</table>

Table 4.7 Factors predicting an increased surgical risk and suboptimal results

<table>
<thead>
<tr>
<th>Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>FS &lt; 25%</td>
</tr>
<tr>
<td>LVESD &gt; 55mm, LVEDD &gt; 70 mm</td>
</tr>
<tr>
<td>regurgitant volume/ enddiastolic volume &lt; 25%</td>
</tr>
<tr>
<td>EF &lt; 50%</td>
</tr>
<tr>
<td>Cardiac index &lt; 2,2 L/min/ m2</td>
</tr>
<tr>
<td>Wedge pressure &gt; 12 mmHg</td>
</tr>
<tr>
<td>Indexed endsystolic volume &gt; 70-90 mL/m2</td>
</tr>
<tr>
<td>Indexed enddiastolic volume &gt; 180 mL/m2</td>
</tr>
</tbody>
</table>
Surgical techniques.

If the aortic valve is damaged by rheumatic, degenerative or infectious processes with deformation of the cusps, shortening, perforation, rupture, then there is no alternative but to replace the valve. In situations where aortic regurgitation is the result of sinus Valsalva dilatation or of the annulus dilatation and aortic cusps are not altered, an attempt of reconstruction procedures of aortic valve and aortic root should be made.

The approach is by median sternotomy, on CPB with mild hypothermia or normothermia, aortic cannulation, single venous cannulation, LV venting through right superior pulmonary vein or through apex and blood cardioplegia administered through coronary ostia and through coronary sinus. The valve is examined for possible repair, and if not possible is excised, the aortic annulus is measured and a mechanical prosthesis (mono – or bi-leaflet), a biological prosthesis (stentless or stentmounted, bovine pericardium or porcine) or a human allograft (homograft) is chosen. Choice of valve is based on patient’s age, pathology, the surgeon’s or patient’s preference and local availability of the aortic substitute.

Special situations.

a) – Aortic regurgitation associated with aortic aneurysm or dissection of the ascending aorta. In this situation the aortic root should be replaced completely by Bentall operation with reimplantation of coronary arteries using a composite graft that combines a vascular prosthesis with a mechanical or a biological valve, or using a stentless prosthesis (Freestyle, aortic root of pork) or a homograft, eventually associated with a tubular Dacron prosthesis which restores the ascending aorta.

When aortic cusps are not damaged, reconstructive procedures as Tirone-David or Jacoub techniques may be used, preserving the cusps reintegrated into a Dacron tubular prosthesis that replaces the ascending aorta; coronary arteries need to be reimplanted also.

b) – Aortic regurgitation from infectious endocarditis. Endocarditis process can lead to massive destruction of the aortic root and of the aortic valve, sometimes requiring complex reconstruction of the aortic annulus due to annulus abscesses and destructions. Since the mechanical valves have a higher susceptibility to reinfection, biological valves or homografts should be used whenever it is possible.

c) – Aortic regurgitation in children and women. To avoid anticoagulation therapy in women who plan childbearing or to avoid a mismatch inserting a mechanical valve in children that will grow, it is recommended the use of Ross operation, which consists of harvesting and implanting patient’s own pulmonary valve (autograft) in the aortic position and replacing the pulmonary outflow tract using a homograft.
d) – Aortic regurgitation associated with other pathology. If the patient has associated coronary artery disease or other valve lesions, all of these should be addressed concomitantly, by coronary artery by-pass grafting and/or by other valve repair or replacement.

e) – Acute aortic regurgitation. The fast decompensation of the left heart that occurs in acute aortic regurgitation, with acute pulmonary edema and cardiogenic shock needs to be addressed accordingly by emergency surgery.

Postoperative complications. Results

The early complications are the common complications of cardiac surgery: bleeding, low cardiac output syndrome, acute renal failure, arrhythmias. Postoperative results are good for chronic aortic regurgitation in compensated stages, but may worsen according to the predictors mentioned in Table 4.7

4. 4. 3. Mitral stenosis

Definition. It consists of an impair of the passage of blood from AS to LV during diastolic ventricular filling due to mitral valvular apparatus alteration that leads to reduced mitral orifice area.

Etiology. The vast majority of cases have rheumatic etiology. Rare causes are: congenital mitral stenosis, left atrial myxoma, Liebmann-Sachs endocarditis or calcification of the mitral annulus in the elderly. Association of atrial septal defect with mitral stenosis is described as Lutembacher syndrome.

Pathophysiology. Normal mitral orifice has an opening of 4-6 cm². Narrowing of the mitral orifice below 2 cm² leads to appearance of symptoms and signs characteristic to mitral stenosis. The obstruction of the blood flow from left atrium to left ventricle results in increased LA pressure, that is transmitted retrogradely to the pulmonary veins and to pulmonary capillaries, causing severe dyspnea and occasional episodes of acute pulmonary edema. Later on, due to functional pulmonary arterial hypertension, pulmonary arterioles react by middle layer hypertrophy, transforming the functional arterial hypertension into an organic one, with functional tricuspid regurgitation and increase in size of the right heart chambers. The left atrium may enlarge, leading to atrial fibrillation, that often precipitates the onset of the symptoms.

Clinical aspect. Patients remain asymptomatic for a long time (nearly 10-15 years) after the rheumatic episode until mitral stenosis becomes severe. The first symptom is dyspnea, its onset may be precipitated by stress, infections, pregnancy, emotional or by atrial fibrillation recently installed. On auscultation there are observed a typical diastolic rumbling murmur, an unusually loud first heart sound and a typical opening snap after the second heart sound. In advanced stages are the patients are asthenic, with weight loss, rest dyspnea, hepatomegaly and peripheral edema.

Paraclinical diagnosis. ECG may reveal sinus rhythm with mitral P wave or atrial fibrillation and RV hypertrophy in advanced stages. Chest X-ray shows enlargement of the left atrium, pulmonary stasis and later on enlargement of the right chambers. Kerley
lines are linear shadows that appear due to fibrosis and lymphatic stasis with increasing pulmonary capillary pressure.

Fig. 4.3 Chest X-ray showing the mitral configuration of the heart and pulmonary stasis. ECG reveals mitral P wave and QRS axis deviation to the right.

**Echocardiography.** It has a special value in diagnosing the disease and in assessing its severity. Routinely it starts with transthoracic echocardiography:

Table. 4.8 Information brought by transthoracic echography in the mitral stenosis

- It confirms the diagnosis of MS
- It assesses the hemodynamical consequences, it calculates the transmitral gradient, the mitral orifice area, pulmonary artery pressure and the size and function of the right ventricle.
- Description of mitral valve morphology: leaflets, chordae tendinae, papillary muscles, mitral annulus.
- Evaluation of the other cardiac valves (aortic, tricuspid, pulmonary).
- Periodical reassessment of asymptomatic patients
- It may detect atrial thrombosis

If images are blurred transthoracically or data are not conclusive, transesophageal echography should be performed:

Table 4.9. Value of transesophageal echography in assessment of the mitral stenosis

- It detects accurately the presence of thrombi in the left atrium or the left atrial appendage
- It offers clearer images of valve morphology.
- Some cardiologists use it routinely to assess the hemodynamic and morphology of the mitral valve.

**Coronary angiography.** It is routinely performed for excluding coronary artery disease for all patients over 45 years or for patients with cardiovascular risk factors (diabetes, hypertension, hyperlipoproteinemia and smoking) regardless of age.
Natural evolution. Mitral stenosis was characterized as a progressive disease. Asymptomatic patients have a survival rate of 80% at 10 years, while this drops in symptomatic patients at 15-20%. The severe pulmonary hypertension decreases further the survival rate. Death of these patients is consequent to cerebral embolism, infection, or progressive heart failure.

Medical treatment. It is important the primary prevention of rheumatic disease, which in western countries has almost been eradicated, while in our country was introduced later, therefore a consistent pathology still exists. Once the diagnosis of mitral stenosis is made, medical treatment is going to prevent specific complications: anticoagulation therapy for preventing left atrial thrombosis and cerebral or peripheral embolism, antiarrhythmic, inotropic or diuretic therapy. Also important is endocarditis prophylaxis in patients with mitral stenosis who undergo dental, surgical or gynecological therapy.

Surgical treatment.

The goal of surgery is to remove the obstacle represented by the mitral valve apparatus, either by valve reconstruction or by valve replacement.

Indications for surgical treatment. They depend on the severity of stenosis, the patient's clinical condition and age and on the complications (pulmonary edema, peripheral or cerebral embolism, presence of atrial fibrillation).

Table 4.10 Indications for surgery in mitral valve stenosis

- Symptomatic patients with severe mitral stenosis, mitral area <1 cm$^2$ transmitral gradient> 12 mmHg, moderate or severe pulmonary hypertension, either in sinus rhythm or atrial fibrillation
- Slightly symptomatic patients, but data which attest echocardiographic severity of the mitral stenosis, to prevent postoperative complications and to achieve a better result.

a) - Closed mitral commissurotomy

Mitral stenosis was practically the first valvular lesion to be approached surgically, given the advantage of using the left atrial appendage as a waiting room, with surgeon's finger trying to dilate mitral opening. This possibility was suggested by Samways in 1898 and it was brought into clinical practice in 1923 by Cuttler. Progressively, it has been abandoned in favor of open commissurotomy or of valve replacement. It was practiced until recently in some less developed countries. In many cases it enlarged the mitral valve opening and brought symptomatic amelioration and delay in progression of the disease.

b) – Open mitral commissurotomy

The routine use of the CPB allowed a direct, visual approach of the mitral valve stenosis. The fused, merged commissures are carefully divided and instrumental dilatation of the
mitral valve is performed. Though, the recurrency rate is high (60% at 10 years), rendering necessary the mitral valve replacement later on.

c) - **Commissurotomy with percutaneous balloon dilatation.** A more recent technique appeared in cardiac catheterization laboratories (1980), which consist of introducing percutaneous via femoral vessel a balloon catheter that is placed at a level of the mitral valve and it dilates it. It may lead to severe complications, like embolism, myocardial infarction and cardiac perforation. It may be used in elderly or pregnant women in critical situations to prevent pulmonary edema during labor.

d) – **Mitral valve replacement.** It represents the method by which the mitral valve apparatus is removed and replaced with a mechanical or biological valve. Mitral valve replacement is performed using the classic approach with median sternotomy and standard CPB or may be performed by minimal right thoracotomy and assisted videoscopy using the Heart Port System. In the recent years, the operation has been performed even totally endoscopic, by robotic control. Mitral valve is excised totally with modified chordae to avoid interference with disc prosthesis. After measuring the mitral annulus, mechanical or biological valve prosthesis is inserted using continuous suture or separate stitches armed with pledgets. When it is possible, it is desirable to keep the posterior leaflet together with its chordae to preserve left ventricular geometry and to achieve a better long term result.

**Complications. Postoperative results.** There are common complications related to heart surgery, like bleeding, arrhythmias, infection and specific complications of mitral valve surgery, like suturing the circumflex artery with myocardial infarction and atrio-ventricular disruption if excessive excision of the mitral valve apparatus is performed or extensive retraction is applied. Postoperative mortality decreased a lot by improving the techniques of myocardial protection and postoperative intensive care, as well as by applying the indication for surgery before occurrence of complications and cardiac decompensation, being below 5%. Survival rate at 10 years is 95%, freedom of reoperation rate is 84% at 10 years and freedom of thromboembolic events is 91% at 10 years.

**4. 4. 4. Mitral insufficiency**

**Definition.** Return of the ventricular blood volume in the left atrium during ventricular systole due to the incomplete closure of the mitral valve apparatus characterizes mitral regurgitation.

**Etiology.** Various pathological processes can affect one or more constituents of the mitral valve, resulting in lack of coaptation between the two leaflets. There is an acute form, with sudden onset, produced by endocarditis, chordal rupture, traumatic or papillary muscle infarction. The chronic mitral regurgitation may be produced by rheumatic disease, still common in some underdeveloped countries, by ischemic heart disease, which leads to tethering of chordae and restricted motion of the posterior leaflet, by mitral valve prolapse, a very common disease or by different processes that lead to mitral annulus dilatation.
**Pathophysiology.** It is different in the acute form of MR, when the amount of regurgitant blood produces an important increase of the LA pressure and subsequently of the pulmonary veins and capillaries, leading to acute pulmonary edema. Since the stroke volume is decreased, the drop of the cardiac output leads to cardiogenic shock. In the chronic form, the left ventricle adapts itself through a mechanism of eccentric hypertrophy, but the systolic function is well preserved for a long time, although the left ventricle and the left atrium increase in size. After a long time, decompensation may occur with heart failure and severe pulmonary hypertension.

**Clinical aspect.** Chronic mitral regurgitation may be well tolerated for many years without symptoms. In time, there appear fatigue, dyspnea, or topnea, bouts of nocturnal dyspnea, palpitations, peripheral edema. On auscultation a holosystolic high pitched murmur may be heard at the apex and towards the axillary region. In the acute form, the symptoms are severe, often evolving to pulmonary acute edema and sometimes to cardiogenic shock.

**Paraclinical diagnosis.** Once the suspicion of mitral regurgitation is raised by clinical examination, the patient is fully investigated and the severity of the disease is assessed. Electrocardiogram may show LV hypertrophy, mitral P wave, ischemic alterations or even Q waves of myocardial infarction. Atrial fibrillation once installed, can remain even after surgery. Chest X-ray may be normal in initial stages, but in advanced stages increase in LV and LA size, together with pulmonary stasis are noticed.

**Echocardiography.** It is an indispensable investigation by information brought about the etiology of mitral disease and its consequences. Furthermore, transesophageal echocardiography is essential intraoperatively for assessing the result of mitral valve reconstruction.

Table. 4.11 Value of echocardiography in the diagnosis of mitral regurgitation

| • Confirms and quantifies the mitral regurgitation and its consequences on the LV function. |
| • Identifies the mechanism of regurgitation. |
| • Detects other valve pathology (aortic, tricuspid, pulmonary) |
| • Rates tricuspid functional or structural regurgitation, pulmonary hypertension. |
| • Guides surgical option since this stage, for replacement or mitral plasty. |
| • Identify the consequences of a myocardial infarction, parietal mobility |
| • Periodical follow-up of moderate to severe asymptomatic forms to choose the optimal moment for surgery. |
| • Visualizes more accurately the presence or absence of atrial thrombosis. |

**Cardiac catheterization.** More than in mitral stenosis is required to confirm or rule out coronary artery disease as etiological mechanism. It may quantify with high sensitivity the regurgitant volume, the severity of MR and its consequences on LV function, the
pulmonary hypertension and the pulmonary resistances.

**Natural evolution.** The prognosis of these patients depends on the etiology, severity of regurgitation and ventricular function. Evolution even in patients with significant regurgitation may be spread over decades to decompensation. But once installed decompensation, operative risk increases and postoperative results are worse, actions to be taken into account when choosing an operative moment. Dilatation of the left atrium with installation of atrial fibrillation brings to 10-20% the incidence of cerebral and peripheral embolic complications. It was noted that 80% of patients with mitral regurgitation stay alive in five years, and 60% after 10 years. Natural evolution is much worse in those with ischemic mitral regurgitation.

**Medical treatment.** Prophylaxis of infective endocarditis in patients with mitral regurgitation is very important. Vasodilator drugs, antiarrhythmics and anticoagulation therapy when atrial fibrillation, diuretics improve the patient's clinical status, but should not lead to indefinite postponement of surgical decision, until severe LV decompensation.

**Surgical treatment.** It is the only rational at a certain point in the evolution of mitral regurgitation. Surgical indication and its best timing is more difficult to determine than for the mitral stenosis. The current trend is to recommend faster intervention before ventricular decompensation and to use the techniques of reconstructive rather than valve replacement.

**Surgical indication.** It is made according to severity of mitral regurgitation, clinical status of patients, symptomatic or asymptomatic and the repercussions on LV function.

Table 4.12 Surgical indication for mitral regurgitation

- symptomatic patients with severe mitral regurgitation and ventricular dysfunction EF <60%, end-systolic LV diameter > 45 mm.
- asymptomatic patients when the echocardiographic and angiographic data show dysfunction of LV
- Before irreparable destruction of the mitral valve apparatus
- If atrial fibrillation appeared, due to better results in case of recent onset are likely to return to sinus rhythm postoperatively.

Table 4.13 Factors predictive of a suboptimal response to surgery in mitral regurgitation surgical correction.

<table>
<thead>
<tr>
<th>Echo - LV shortening fraction &lt;0.32</th>
</tr>
</thead>
<tbody>
<tr>
<td>- End-diastolic diameter index &gt; 40 mm/m2</td>
</tr>
<tr>
<td>- End-diastolic diameter &gt; 50 mm</td>
</tr>
<tr>
<td>- End-systolic diameter index &gt; 26 mm/m2</td>
</tr>
<tr>
<td>Angio - EF &lt;55%</td>
</tr>
<tr>
<td>- LVEDP &gt; 12 mmHg</td>
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Surgical technique. There are two types of operations that are performed when mitral regurgitation mitral valve reconstruction and valve replacement.

a) - mitral valve reconstruction - is made whenever it is possible, based on favorable anatomy and surgeon’s experience. There are several valve repair techniques that address specific anatomical lesion: annulus enlargement, valve prolapse, elongation of chordae, chordae rupture, papillary muscle rupture. Accurate preoperative assessment and intraoperative transesophageal echocardiography and especially the surgeon's direct examination assist in decision making regarding surgical repair and use of the proper techniques.

The classical mitral valve repair technique is quadrangular resection of posterior cusps in case of a mitral prolapse and insertion of a mitral ring.

Fig. 4.4 Quadrangular resection of posterior mitral valve and mitral ring insertion

In case of rupture or elongation of the chordae, these are repaired or replaced with artificial chordae from Goretex stitches and a mitral ring is inserted. Mitral rings are artificial products, metal or plastic, covered with textile fabric to help their insertion. Rings have different forms, they may be complete or incomplete, rigid or flexible. They help stabilize mitral annulus, which is often increased.

Fig. 4.5 Mitral rings: complete (Carpentier-Edwards), incomplete (Labcor) and flexible (Komp)
Although difficult, if possible the postendocarditic mitral regurgitation may be repaired after excision of the infected part and the reconstruction of mitral apparatus with as little foreign material as possible to prevent reinfection.

Fig. 4.6 Posterior native mitral valve endocarditis in a young patient, 34 years, pregnant: the posterior leaflet was partly resected and replaced with pericardial patch and a Carpentier mitral ring was inserted. (L. Muller & G. Gaspar, Innsbruck, Austria, 2002)

b) - mitral valve replacement. When mitral valve repair is not possible it is replaced by mechanical or biological valve prosthesis. It is recommended to keep the posterior leaflet and its chordae in order to preserve LV geometry and to achieve a better long term result. Technically this is achieved by folding the posterior leaflet with the stitches that fix the prosthesis.

**Mitral valve prolapse**

**Definition.** Protrusion of a mitral leaflet into the left atrium during ventricular systole, leading to imperfect closure of the mitral valve. Initially, the mitral regurgitation is minor, but this may evolve to get significant. Prevalence is 1-6% of all population. Its incidence is higher in patients with Graves' disease, Duchenne muscular dystrophy, Marfan syndrome and interatrial septal defect.

**Clinical aspect.** Most patients are asymptomatic, being discovered occasionally, with a short systolic mitral murmur. When the mitral regurgitation is important, symptoms may occur: palpitations, fatigue, dyspnea on exertion, chest pain.

**Diagnosis.** It is clinical and confirmed by echocardiography, which may quantify the degree of regurgitation.

**Treatment.** It does not require any specific treatment, but infectious endocarditis prophylaxis during invasive maneuvers or dental treatments. It is recommended periodical echocardiographic examination and avoid high level competitive sports that require intense exercise. In case the regurgitation progresses to grade III-IV, then it is considered a severe mitral regurgitation and enters into the diagnostic and therapeutic algorithm of this disease.

4. 4. 5. **Tricuspid stenosis**
In most cases of rheumatic etiology. Other causes may be congenital tricuspid atresia, right atrial tumors, rarely endomyocardial fibrosis or endocarditis.

**Pathophysiology.** Diastolic gradient between AD and VD increases, transvalvular flow is reduced and result in systemic venous stasis with swelling of the internal jugular veins, hepatomegaly, edema and ascites.

**Clinical aspect.** The patient has fatigue, dyspnea, turgid jugular, hepatomegaly, edema and ascites in advanced stage.

**Diagnosis.** It is suggested by the clinical symptoms and by the physical examination of patient and confirmed by laboratory investigations including echocardiography, which is essential. Usually there are also other valves affected in the rheumatic disease.

**Treatment.** Medical treatment, with dietary salt restriction and diuretics may temporarily improve the clinical condition. Surgery is trying to normalize blood flow through the tricuspid valve by direct digital or instrumental commissurotomy. In case of a poor result, the tricuspid valve is replaced preferably with a biological valve. Mechanical valves in tricuspid position are to be avoided when possible due to increased rates of thrombosis and clogging.

**Tricuspid regurgitation**

It is usually the result of the dilatation of the right ventricle secondary to valve diseases of the left heart or to myocardial infarction and only rarely is isolated, like in endocarditis.

**Clinical aspect.** In the absence of pulmonary hypertension with medication is well tolerated. When the pulmonary hypertension is severe, right heart failure installs: turgid jugular veins, hepatomegaly, ascites and peripheral stasis.

**Diagnosis.** Clinical examination reveals signs of RV decompensation (edema, hepatomegaly, jugular turgid, ascites) and at auscultation increased systolic murmur in inspiration (Carvallo sign). Echocardiographic examination estimates the severity of tricuspid regurgitation, the size of heart cavities, pulmonary hypertension and RV function.

**Treatment.** The surgical indication depends on the type of the TR (functional or organic), on its severity, on the pulmonary hypertension’s value. If regurgitation is functional grade II / III without severe pulmonary hypertension then only the main cause that led to RV dilatation is addressed (mitral stenosis, pulmonary stenosis, coronary artery disease); if TR is severe, a tricuspid ring may be inserted or DeVega annuloplasty performed. If the valve cannot be repaired, then a biological valve is used to replace the tricuspid valve.

4. 4. 6. **Pulmonary stenosis or regurgitation**

The common etiology for pulmonary stenosis is congenital, simple or within complex malformations (tetralogy of Fallot) and only rarely is acquired, rheumatoid or carcinoid syndrome.

Pulmonary regurgitation is most often secondary to pulmonary hypertension or pulmonary dilation.
Diagnosis is made by echocardiography and/or right heart catheterization. Surgical treatment requires valve plasty. When the pulmonary valve must be changed due to valvular stenosis or regurgitation, the choice is a biological conduit, homograft or pulmonary xenograft, Medtronic Freestyle, Contegra Conduct.

4. 4. 7. **Multivalvular lesions**

Rheumatic disease often affects one or more valves. Endocarditis on a valve may be extended by the neighboring valve. Organic damage of a valve may lead to functional damage of the other. Full recognition is important for a proper valve affected surgical indication and solving simultaneously. Transthoracic and transesophageal echocardiography examination sometimes three-dimensional reconstruction allows assessment correct, complete and appropriate therapeutic decision making. Replacement surgery two or three valves (valve plasty) is burdened by an increased surgical risk and postoperative outcome of heart disease depends.

4. 4. 8. **Types of prosthetic valves**

Medical research efforts to find a substitute for mechanical or biological valves over more than five decades and resulted in a wide range of commercial products tend to approach the function of heart valves. However the results are far from ideal.

Ideal characteristics of a prosthetic valves.
- To achieve a good hemodynamic, central flow without turbulence, without gradient
- Resistance to infection
- Nonthrombogenic
- Do not destroy the figurative elements of blood – non hemolytic
- Easy to implant
- Reasonable cost
- Readily available
- Strength in time

a) - haemodynamic feature has been extensively studied in clinical and laboratory conditions, using duplicating to reproduce the long time heart activity in different conditions of rest and exercise. Pressional transvalvular gradient criterium is very important when a valve is implanted. It can be calculated by measuring the Doppler echocardiographic maximum instantaneous gradient (MIG-maximal instantaneous gradient). All valves have a gradient. Performance index (performance index PI) of a valve is given by the ratio of effective orifice area (EOA, effective orifice area) and area of the suture ring. The higher this index is higher with both valve hemodynamic performance is. For example, porcine valves have a PI 0.35-0.40, the bovine 0.65 and double disc valves 0.65-0.70.

b) - valvular thromboembolism. Thrombus formation on the valve and cerebral or peripheral embolism is a delicate issue in valve replacement. To prevent valve thrombosis anticoagulant therapy is given to the long. Biological valves are more resistant to
thrombosis, homografts being the best. They do not require anticoagulant therapy if the patient is in sinus rhythm. Mechanical valves need lifelong anticoagulation should be mandatory, with an INR between 2.5-3.5.
c) - Valve clogging. Is a severe complication of thrombus formation and consists of the valve blockage. It is a medical and surgical emergency that threatens the patient's life. Treatment consists of fresh thrombus lysis with fibrinolytic medication if it succeeds and if not, emergency surgery, thrombus extraction with or without the need to change the valve. This complication occurs mainly in patients who do not comply with anticoagulant treatment.
d) - Bleeding. Anticoagulant therapy besides beneficial effect may also lead to bleeding accidents, which can be powerful, cerebral, even fatal or minor bleeding, gum, bladder. Check INR and adjust dose of anticoagulant.
e) - Infections. Prosthetic valves are susceptible to infection, and of these the mechanical are the most affected and the most resistant are homografts. Treatment is antibiotics and surgical treatment is indicated when paravalvular leak occurs or infection sites can not be controlled by medication.
f) - Hemolysis. Destruction of figurative elements of blood is another disadvantage, especially of mechanical valves. Is mostly due to a paravalvular leak, which produces blood turbulence.
g) - Structural durability. Mechanical valves are the most durable and the biological are undergoing a process of degeneration in time which would have required reintervention and their change after 10-15 years.

**Classification of prosthetic valve**
After the materials that are made are divided into mechanical valves and biological valves.
I mechanical prostheses. Ring and disks are made of metal or carbon Pirol and the ring is covered with a plastic texture that help to fix them.
• Ball - Starr-Edwards - is no longer used

![Mechanical ball valves, Starr-Edwards](image.png)

• The single-disc Medtronic-Hall, Sorin Carbocast, Allcarbon, Omnisience (Fig.)
II biological valves. Biological materials are made of animal origin (bovine pericardium, porcine valves) specially treated and mounted on a metal frame and textile fabric belts (biological valves with stenting) and without metallic frame casing (stentless biological valves).

a) - Heterografts or xenografts - valves harvested from animals (Fig.)
- Porcine - stentmounted - Carpentier-Edwards valve
  - Stentless - Medtronic Freestyle
- Bovine pericardium - stentmounted - Ionescu-Shiley, Mitroflow
- Stentless Freedom
Selection of heart valves

A valve selection is based on patient age, valve pathology, patient’s and surgeon’s preference, availability and type of valve experience doctor.

a) - mechanical valves. It is recommended to:
• young patients with expectation of long survival without contraindications to anticoagulation treatment
• young women who childbeared or are unwilling to take charge

Mechanical valves have a long resistance, but have the disadvantage of anticoagulant therapy with its implications for life and are more susceptible to infections.

b) - Bioproteze –porcine or bovine xenografts.
• For women who want to have children to avoid anticoagulation therapy.
• elderly patients over 70 years, the risk is high anticoagulant treatment
• to patients in remote areas unable to control anticoagulation treatment
• Contraindication of anticoagulation therapy (gastrointestinal bleeding, coagulopathy)
• endocarditis, when homografts are not available.

Do not require anticoagulant therapy, but have the disadvantage of degeneration and the necessity of reintervention in order to change the valve.

c) - Homografts
• Children who are growing
• Young people who do not wish to take anticoagulant therapy
• endocarditis