

HEART VALVE DISEASES

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Introduction

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Although the prevalence of heart valve diseases is low in the general population - around 2.5% - it is much higher in patients aged 75 years and older, reaching 10% to 15%.

In Europe, the most common valve diseases are mitral regurgitation (mitral insufficiency) and aortic stenosis, which affect three out of four patients. Then comes aortic regurgitation (aortic insufficiency), followed at some distance by mitral stenosis. Today, the three most common valvular heart diseases are degenerative conditions. Other causes are possible but rarer: ischaemic, congenital or due to endocarditis. Mitral stenosis is mainly due to acute articular rheumatism (AAR). This is very rare in Western countries, occurring mainly in developing countries.*

Improvements in healthcare during the past 30 years have contributed to changes in the distribution of the causes of valvular heart disease. Thus, rheumatic valve disease, which once was predominant in young subjects, has gradually become less common. On the other hand, with the increase in life expectancy, degenerative causes have become more frequent.

Moreover, it remains likely that the number of cases will continue to grow as the population ages. As for the prognosis for valvular heart disease overall, it will no doubt change considerably as a result of improvements in diagnosis and treatment, in particular recent developments in surgical techniques.



Cardiac cycle and heart valve diseases

SOME ANATOMICAL REMINDERS

Basically, the heart is divided into two halves. The left side is the seat of arterial circulation: it consists of the left atrium (LA) and the left ventricle (LV), separated by the mitral valve. The right side is the seat of venous circulation: it consists of the right atrium (RA) and the right ventricle (RV), which are separated by the tricuspid valve.

The cardiac cycle is a continuous pattern of events. The blood arrives from the lungs via the pulmonary veins; it enters the left atrium then the left ventricle via the mitral valve. It is then ejected into the aorta through the aortic valve to be carried around the rest of the body.

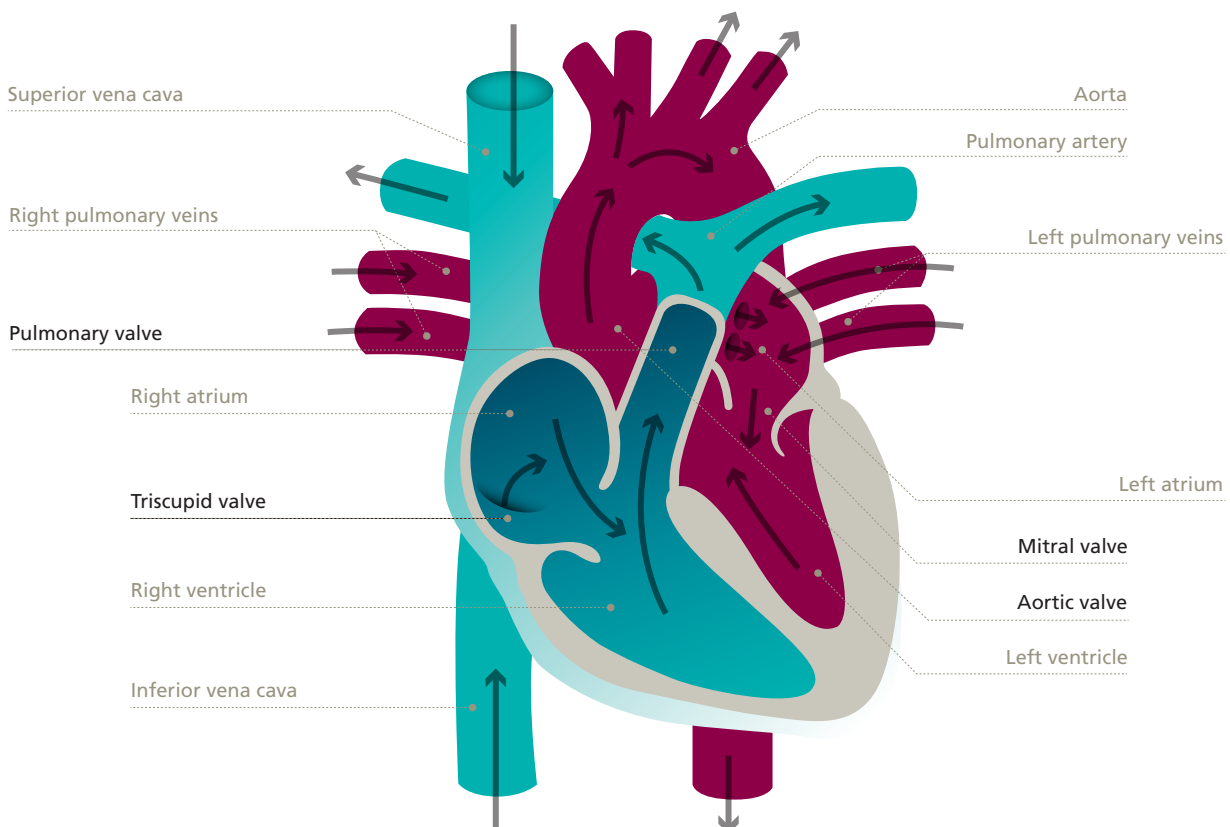
Then the blood returns to the heart on the right side via the inferior and superior vena cava, entering the right atrium, passing through the tricuspid valve to the right ventricle then through the pulmonary valve. The pulmonary artery carries the blood to the lungs, where it picks up oxygen, before returning to the pulmonary veins and so on.

The blood leaves the atria to fill up the ventricles: this takes place during the phase known as diastole*, which is followed by systole*, the phase when the ventricles eject the blood.

The heart valves operate in the same way as mechanical valves:

- as inlet valves that receive the blood: the mitral and tricuspid valve,
- as outlet valves since the blood flows out of them: the aortic valve on the left side and pulmonary valve on the right side.

ANATOMICAL REMINDER: SITE OF THE FOUR VALVES



HEART VALVE DISEASES: CAUSES AND DIAGNOSIS

Problems affecting the heart valves can be of two sorts:

- Stenosis (or narrowing) of the valve opening, where an obstruction prevents the blood getting through.
- Regurgitation (also called insufficiency), where the valve does not close tightly, leading to backward leakage.

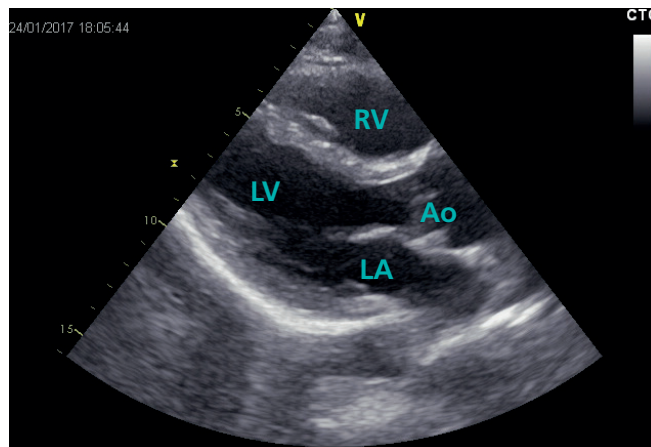
In some cases valvular heart disease involves concomitant stenosis and regurgitation. Thus, aortic valve disease may combine aortic regurgitation with aortic stenosis.

Generally speaking, valve diseases are quite easy to diagnose given that symptoms are almost always identical: exertional dyspnea, palpitations, chest pain and lower limb edema, especially in the case of heart failure. A cardiac murmur is the major sign of valve disease. It is discernible on auscultation, allowing a diagnosis to be suspected. It may be systolic*, diastolic* or systolic-diastolic* and may already indicate the origin of the valve disease during the clinical examination. Other signs may be present, such as rhythm disorders or other signs of heart failure. An electrocardiogram (ECG) is a test that can detect such heart rhythm disorders.

Echocardiography is paramount to diagnose, assess the severity and guide the treatment of valvular heart diseases. This is always combined with a color flow Doppler: the echo shows the size, structure and function of different parts of the heart and large blood vessels, while the Doppler ultrasound is used to estimate the velocity of the blood flows.

2D ECHOCARDIOGRAM (LONG-AXIS PARASTERNAL* VIEW)

Conventional parasternal view provides a two dimensional image of the left ventricle (LV) and allows the thicknesses of the walls in the two phases of the cardiac cycle to be appreciated: in systole, the thickness is greater than in diastole. The measurements taken are used to calculate the left ventricular ejection fraction (LVEF), that is to say the contractility of the LV: its ability to eject the blood into the aorta.



Source: Dr. Christophe Genel, Cardiologist

2D NORMAL ECHOCARDIOGRAM VALUES FOR THE DIMENSIONS OF LV AND OF LVEF

The latest measurements published by the American Society of Echocardiography provide some benchmarks. For a male, the internal diameter of the left ventricle is therefore between 42 and 58.4 mm in telediastole (end of diastole). In telesystole, it is from 25 to 39.8 mm. There are also volume data and body surface area-indexed volume data: for example, the heart of a woman measuring 1.60 m and weighing 55 kilos will not be the same weight and size as that of a man measuring 1.90 m and weighing 110 kg. The LVEF* for a man is 62% (+/-5) and for a woman 64% (+/-5).

| PARAMETERS | MALE | | FEMALE | |
|------------------------------------|--------------|-----------|--------------|-----------|
| | MEAN +/- 2SD | NORMS | MEAN +/- 2SD | NORMS |
| LV INTERNAL DIAMETERS | | | | |
| Telediastolic (mm) | 50,2 +/- 4,1 | 42,0-58,4 | 45,0 +/- 3,6 | 37,8-52,2 |
| Telesystolic (mm) | 32,4 +/- 3,7 | 25,0-39,8 | 28,2 +/- 3,3 | 21,6-34,8 |
| LV VOLUMES | | | | |
| Telediastolic (ml) | 106 +/- 22 | 62-150 | 76 +/- 15 | 46-106 |
| Telesystolic (ml) | 41 +/- 10 | 21-61 | 28 +/- 7 | 14-42 |
| BSA-INDEXED LV VOLUMES | | | | |
| Telediastolic (ml/m ²) | 54 +/- 10 | 34-74 | 45 +/- 8 | 29-61 |
| Telesystolic (ml/m ²) | 21 +/- 5 | 11-31 | 16 +/- 4 | 8-24 |
| LVEF (BIPLANE METHOD) | 62 +/- 5 | 52-72 | 64 +/- 5 | 54-74 |

Source: JASE, 2015, Vol.28, Issue 1, p.7 (January 2015)

SD: standard deviation
 BSA: body surface area
 LV: left ventricle
 LVEF: left ventricular ejection fraction

AORTIC STENOSIS

Aortic narrowing, more commonly known as aortic stenosis (AS), is a common form of valve disease. It is characterised by an obstruction preventing ejection from the left ventricle (LV) into the aorta, leading to an increase in the afterload. In other words AS slows the ejection of blood by the left ventricle. To combat the force of this increased resistance, the LV adapts by developing hypertrophied walls, before becoming exhausted and dilating.

The causes of aortic stenosis are mainly degenerative. This is why the condition is mainly seen in people over age 70 with valve calcifications. However, in 15% of cases, AS is congenital and therefore occurs in younger people, usually around the age of 40: this is the case of bicuspid aortic valve* (the aortic valve is normally tricuspid). AS can also be linked to acute articular rheumatism, although this is infrequent.

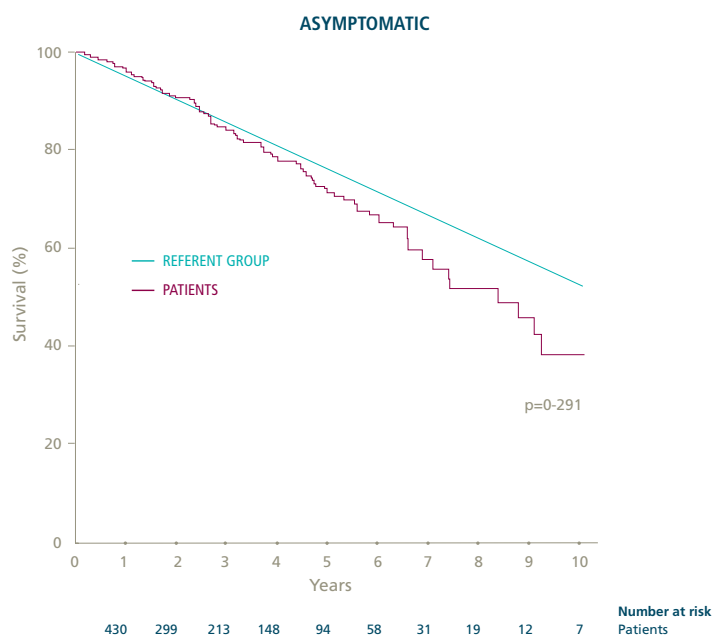
For a long time AS remains asymptomatic. But survival is impacted as soon as the symptoms appear. And so, once the signs of heart failure appear, survival is then about two years. For angina pectoris or dyspnea*, median survival is of the order of four to five years. In patients with both left ventricular dysfunction and severe dyspnea, the prognosis is poor: only 20 to 25% of patients survive more than three years. And in some rare cases, when AS is very severe, the blood flow can no longer be ejected into the aorta, leading to sudden death.

AS is the valve disease with the worst prognosis.



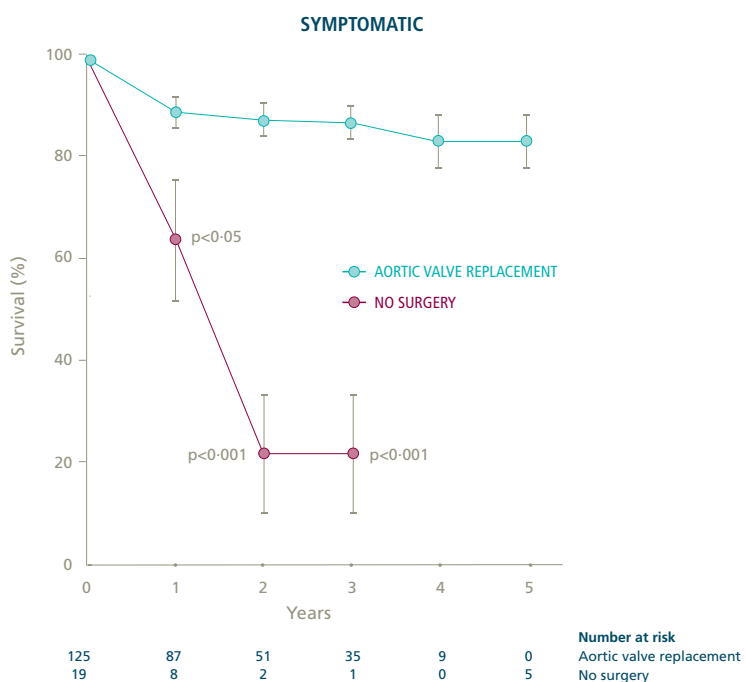
Aortic stenosis is the valve disease with the worst prognosis.”

NATURAL HISTORY OF CALCIFIC AS



The ESC (European Society of Cardiology) classification grades the severity of AS: grade I (mild), II (moderate) or III (severe). Severe AS is characterised by an ejection velocity more than 4 m/s and a valve area of less than 1 cm². Indexed by body surface area (valve area index), the threshold is 0.6 cm²/m². Grade III AS is also characterised by a mean gradient higher than 40 mmHg. The presence of an obstruction leads to a difference in pressure between the LV and the aorta, with higher pressure in the LV and lower pressure in the aorta: it is this gradient that is measured.

Source : Pellikka et al. *Circulation* 2005;111:3290-5



Source : Schwartz et al. *Circulation* 1982;66:1105-10

AORTIC REGURGITATION

Aortic regurgitation (AR), also called aortic insufficiency, is in a way the "aortic" counterpart of mitral regurgitation. Here the valve is leaking and blood regurgitates into the left ventricle.

More precisely, in systole, the blood flows into the aorta, the aortic valve closes, but some blood leaks back into the left ventricle.

This is known as acute aortic regurgitation when there is aortic dissection*, endocarditis or thoracic trauma. It can also be chronic when it is linked to annuloaortic ectasia*, bicuspid aortic valve, acute articular rheumatism*, subacute endocarditis* or aortitis*.

QUALITATIVE AND QUANTITATIVE PARAMETERS USEFUL IN ASSESSING THE SEVERITY OF AORTIC REGURGITATION

This table provides benchmarks for assessing the severity of AR. When the effective regurgitant orifice area (EROA) is less than 0.1 cm², the aortic regurgitation is mild; conversely, when it is greater than 0.3 cm², it is considered severe. Vena contracta (VC) corresponds to the original diameter of the jet and is also a very useful piece of information: when it is greater than 6 mm, it is likely to indicate that the AR is severe. At less than 3 mm it suggests mild AR. Finally, the repercussions on the heart chambers also provide clues: a dilated left ventricle generally indicates a substantial leak.

| | MILD | MODERATE | SEVERE |
|--|---------------------------------|-----------------------|---|
| STRUCTURAL PARAMETERS | | | |
| LV size | Normal | Normal or dilated | Usually dilated |
| Aortic leaflets | Normal or abnormal | Normal or abnormal | Abnormal/flail, or wide coaptation defect |
| DOPPLER PARAMETERS | | | |
| Jet width in LVOT - Color Flow | Small in central jets | Intermediate | Large in central jets; variable in eccentric jets |
| Jet density - CW | Incomplete or faint | Dense | Dense |
| Jet deceleration rate - CW (PHT, ms) | Slow >500 | Medium 500-200 | Early peaking-triangular |
| Diastolic flow reversal in descending aorta - PW | Brief, early diastolic reversal | Intermediate | Systolic flow reversal |
| QUANTITATIVE PARAMETERS | | | |
| VC width (cm) | <0,3 | 0,3-0,6 | >0,6 |
| Jet width /LVOT width (%) | <25 | 25-45 46-64 | ≥65 |
| Jet CSA /LVOT CSA (%) | <5 | 5-20 21-59 | ≥60 |
| R Vol, ml/beat | <30 | 30-44 45-59 | ≥60 |
| RF (%) | <30 | 30-39 40-49 | ≥50 |
| EROA (cm ²) | <0,1 | 0,1-0,19 0,20-0,29 | ≥0,3 |

Source : JASE, 2003, Vol.16, Issue 7, 777-802 (july 2003)

LV: left ventricle
 LVOT: left ventricular outflow tract
 CW: continuous wave
 PHT: pressure half-time
 PW: pulse wave
 VC: vena contracta
 CSA: cross sectional area
 EROA: effective regurgitant orifice area

In terms of prognosis, the left ventricular ejection fraction (LVEF) is an essential piece of information: when it is low (under 35%), survival compared to the general population will be considerably reduced, of the order of 10% after 15 years. When the LVEF is moderately affected, between 35 and 50%, the repercussions remain serious.

On the other hand, a LVEF is considered as normal when it is 50% or over.

Symptoms play an important role in disease prognosis. In highly symptomatic patients, mainly those with dyspnea, survival is affected.

ASCENDING AORTIC ANEURYSM



Dilatation of the ascending aorta is sometimes associated with aortic valve disease, either because it leads to aortic regurgitation or because it is associated with a bicuspid aortic valve. These dilations of the ascending aorta have their own prognosis, with a risk of aortic dissection*, which is an extremely serious complication as it leads to an in-hospital mortality rate of 25%, even after surgery.

Thoracic aortic dilatations have various etiologies. It includes genetic conditions (Marfan syndrome in particular), bicuspid aortic valve and degenerative causes. Consequently diagnosis occurs at different ages, and there is an impact on the indications for treatment as progression will be different even though there are many similarities in histology and in spite of imprecise nosological limits. Whatever the etiology, the more dilated the aorta, the higher the risk of death. The annual death rate from an aortic wall complication is dependent on the body surface area-indexed aortic diameter: the annual rate of aortic dissection, rupture or death is 20% when the ASI (Aortic Size Index) is greater than 4.25 cm/m².

Intervention thresholds are codified in the recommendations. In the case of Marfan syndrome, a dilated aorta diameter of more than 50 mm is the threshold for intervention; this also applies to a bicuspid aortic valve with risk factors. If there are risk factors associated, especially rapid progression or a family history of aortic dissection or sudden death, the operation will take place earlier, at 45 mm. In all other cases, the operation must take place when the aorta is dilated to more than 55 mm. This 55 mm threshold is therefore a formal indication.

Patients should nevertheless be assessed individually, taking into account their natural history, the progression of the dilation and whether or not there is an associated valve disease.

The mitral valve

MITRAL REGURGITATION

The role of the mitral valve is ensuring communication between the left atrium and the left ventricle when it is open and to provide a perfect seal when it is closed. It has two cusps or leaflets: the anterior and posterior cusps, whose edges adhere perfectly when the valve is operating correctly; this is known as coaptation of the cusps.

Mitral regurgitation (MR), also called mitral insufficiency, is due to the valve cusps not completely sealing during systole, when the left ventricle contracts and ejects the blood into the aorta. As the valve does not close tightly, a part of the blood flows into the aorta, but another part leaks back into the LA. The bigger the leak, the more it dilates the LA and also the LV, which will dilate to cope with the overload. When the LV is exhausted and can no longer adapt due to the extent of the leakage, heart failure occurs.

MR has the particularity of being either organic or functional.

In organic (or primary) MR, the valve structure is abnormal, with lesions to the valve or subvalvular apparatus whose causes are for the most part degenerative; however, they can also be due to acute articular rheumatism or endocarditis. This type of heart valve leak results in left ventricular dysfunction. The main mechanism in degenerative MR is valve prolapse*: for example in posterior valve prolapse, the anterior cusp is normal but the posterior cusp is displaced into the left atrium, causing a major tightness defect. Such valve prolapses are linked above all to lesions (rupture or elongation) of the chordae tendinae (colloquially known as the "heart strings") which attach the valves to the heart. Lesions to the cusps or mitral annulus (to which the valves are attached) may also be involved.

Conversely, in functional (or secondary) MR, the structure of the valve is normal, but the valve leaks due to a distortion of the subvalvular apparatus, which is itself linked to left ventricular remodeling. In functional MR, left ventricle dysfunction is the cause of the problem and not the consequence: the causality pattern is the opposite of organic MR. The indications for treatment are therefore very different.

In addition, mitral regurgitation can be acute or chronic.

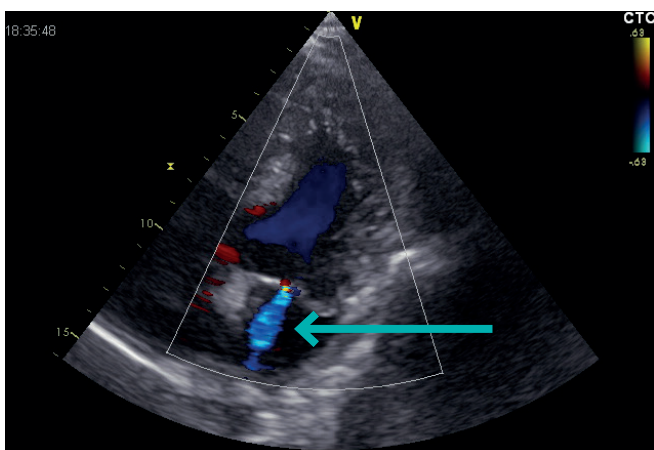
Acute mitral regurgitation is mainly due to rupture of the chordae, leading to sudden regurgitation. It can also occur in ischaemic heart disease* or on myocardial infarction. Likewise, infective endocarditis can destroy the valve and cause acute mitral regurgitation; more rarely, trauma can be the cause (accident).

However, the most common case of chronic mitral regurgitation is degenerative in origin. The chronic disease may progress over 20 to 30 years: it concerns elderly subjects in more than 50% of cases. Only rarely linked to acute articular rheumatism, it is due to dystrophy* with mitral valve prolapse (Barlow's disease), characterized by excess tissue in the valve. Mitral regurgitation can also be functional, secondary to annular dilatation, related to LV dilatation in case of dilated cardiomyopathy*.

The natural progression or natural history of MR depends on the extent of the leak and the ejection fraction: the more severe is the leak and LVEF below 50%, the more pejorative is the outcome.

APICAL VIEW WITH COLOR FLOW DOPPLER

This apical view of the two chambers is a color flow Doppler image of a mitral regurgitation. The light blue flow is a mitral regurgitant flow.

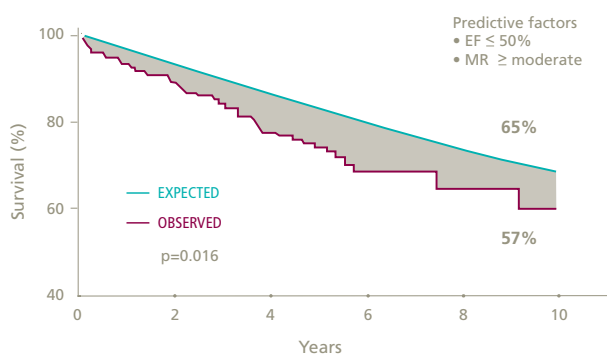


Source : Dr. Christophe Genel, Cardiologist

The severity of heart valve disease is a prognostic factor in itself. Half of patients with severe regurgitation, with an EROA greater than 40 mm² do not survive more than five years. And an even greater proportion suffer cardiac events.

Severe MR has a very poor prognosis, and the symptoms, which occur belatedly, have a pejorative prognostic value. Unlike aortic stenosis, patients need to undergo surgery before becoming very symptomatic: indeed, if they are operated on in NYHA (New York Heart Association) class III or IV, their survival will be lower than that of a population of the same age and sex. However, if they are operated on in the asymptomatic stage (NYHA class I) or in the mildly symptomatic stage (class II), their life expectancy is comparable to that of the population of the same age and sex. The decision for intervention needs to be made, therefore, at an early stage. For surgery, the impact of the left ventricular ejection fraction* (LVEF) is important. If patients are operated on with a LVEF higher than 60%, survival is excellent. If it is under 50% before the operation, the rate of survival falls to 30% after 10 years. Moderate left ventricular dysfunction (EF between 50 and 60% inclusive) leads to delayed excess mortality.

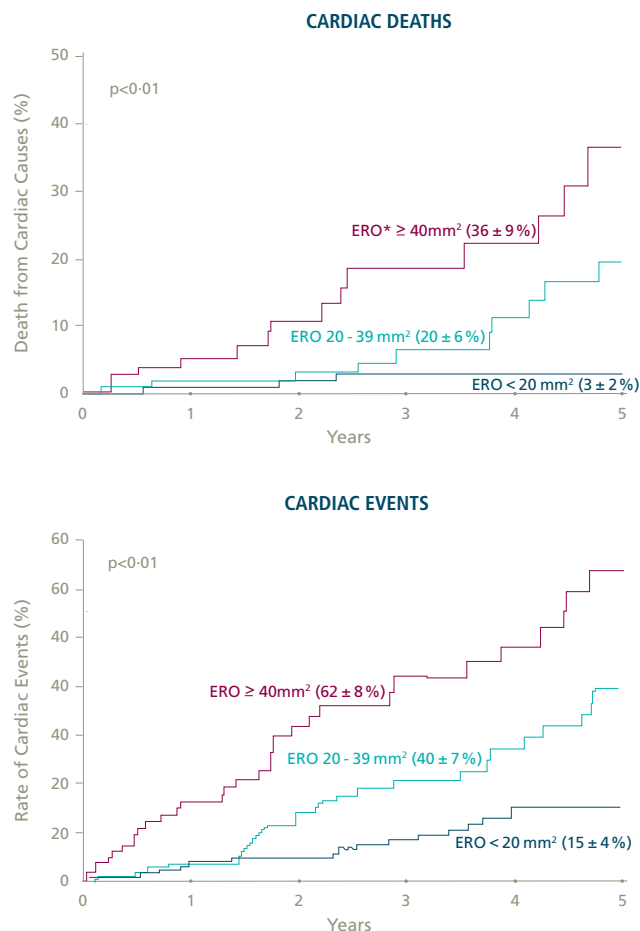
NATURAL HISTORY OF MR DUE TO VALVE PROLAPSE



Source : Avierinos et al. *Circulation* 2002;106:1355-61

SEVERITY OF MR AND NATURAL HISTORY

456 asymptomatic patients (1991-2001). Progression according to MR quantification



Source : Enriquez-Sarano et al. *N Engl J Med* 2005;352:875-83

QUANTITATIVE AND QUALITATIVE PARAMETERS USEFUL IN ASSESSING THE SEVERITY OF MITRAL REGURGITATION

There are qualitative and quantitative parameters for assessing the severity of MR. When the LA or the LV is dilated, the leak is substantial and has repercussions on the chambers of the heart: this is a first way of assessing the extent of the regurgitation. Other measurements then provide more precise informations, such as VC (vena contracta) width: a diameter greater than 7 mm suggests severe regurgitation. Likewise, the effective regurgitant orifice area (EROA) is an important parameter: an area greater than 0.4 cm² indicates severe regurgitation.

| | MILD | MODERATE | SEVERE |
|--------------------------------------|---|---------------------------|--|
| STRUCTURAL PARAMETERS | | | |
| LA size | Normal | Normal or dilated | Usually dilated |
| LV size | Normal | Normal or dilated | Usually dilated |
| Mitral leaflets or support apparatus | Normal or abnormal | Normal or abnormal | Abnormal/Flail leaflet Ruptured papillary muscle |
| DOPPLER PARAMETERS | | | |
| Color flow jet area | Small, central jet (usually <4 cm ² or <20 % of LA area) | Variable | large central jet (usually >10 cm ² or >40 % of LA area) or variable size wall-impinging jet swirling in LA |
| Mitral inflow - PW | A wave dominant | Variable | E wave dominant |
| Jet density - CW | Incomplete or faint | Dense | Dense |
| Jet contour - CW | Parabolic | Usually parabolic | Early peaking-triangular |
| Pulmory vein flow | Systolic dominance | Systolic blunting | Systolic flow reversal |
| QUANTITATIVE PARAMETERS | | | |
| VC width (cm) | <0,3 | 0,3 - 0,69 | ≥ 0,7 |
| R Vol (ml/beat) | <30 | 30 - 44 45 - 59 | ≥ 60 |
| RF (%) | <30 | 30 - 39 40 - 49 | ≥ 50 |
| EROA (cm ²) | <0,2 | 0,2 - 0,29 0,30 - 0,39 | ≥ 0,4 |

Source : JASE, 2003, Vol.16, Issue 7, 777-802 (july 2003)

PW: pulsed wave
 CW: continuous wave
 VC: vena contracta
 EROA: effective regurgitant orifice area

MITRAL STENOSIS

Mitral narrowing, more commonly known as mitral stenosis, is characterized by an obstruction in the mitral valve. The blood in the left atrium which should flow into the left ventricle is hindered by an obstruction usually linked to acute articular rheumatism (AAR), a rare condition in Western countries. In exceptional cases, MS can be congenital.

The natural history of MS is much slower than that of other heart valve diseases, often involving a long latent period of 20 to 40 years between the occurrence of AAR and the appearance of the symptoms. Once the symptoms appear, the survival rate is impacted immediately, from 0% to 15% after 10 years. Overall, the 10-year survival rate of untreated patients oscillates around 50% to 60%, depending on the symptoms. On the other hand, in asymptomatic subjects, survival is more than 80% at 10 years.

The main problem with mitral stenosis is the risk of thromboembolism.

The risk increases with patient age, the presence of atrial fibrillation*, the left atrium size, the degree of MS and the presence of a spontaneous echo contrast.

There is a real benefit in operating on patients - excellent survival, far higher than that of the natural history.



The natural history of mitral stenosis is much slower than that of other heart valve diseases.”

Treatment of heart valve disease



Whatever the type of heart valve disease, there is no medical treatment available. Treatments only deal with the complications (heart rhythm disorders, heart failure) not with the valve disease itself. The only effective treatment for heart valve disease is surgery.

In all types of aortic valve disease and for about half of cases of mitral valve disease, surgery involves valve replacement, with a mechanical or biological prosthesis.

Mechanical valves have been used longer and have proven their effectiveness. The very first ones, implanted in 1960, were caged ball valves. They were followed by tilting-disc valves, then by the bi-leaflet valves used today. They are made from an inert composite material and have a long lifespan. They are not usually changed unless complications occur. Their main drawback is that they require that the patient take anticoagulants for life: vitamin K antagonists (the newer anticoagulants are formally contraindicated). This treatment is closely monitored and optimised, as coming off the drugs even temporarily can lead to serious complications, primarily thrombosis* of the prosthesis, which can become blocked and lead to sudden death.

Biological heart valves, or bioprosthetic valves, generally made from pig heart valves, are an alternative solution. They are made of biological tissue treated to make it less antigenic. These valves therefore do not require treatment with anticoagulant drugs (except in the case of rhythm disorders, atrial in particular). However, they have the drawback of deteriorating over time and therefore having a shorter lifespan than mechanical valves. After about 10 years, an annual deterioration rate is observed. This degeneration depends to a large extent on the patient's age; it occurs faster in young patients and much more slowly in elderly patients. After the age of 70, a bioprosthetic valve will often be chosen. In younger patients, the decision requires a case-by-case approach taking into account the patient's preferences and lifestyle.

ST JUDE-TYPE BI-LEAFLET MECHANICAL HEART VALVE



Source: P. Dutoit SAM-CHUV

BIOPROSTHETIC HEART VALVES



Source: P. Dutoit SAM-CHUV

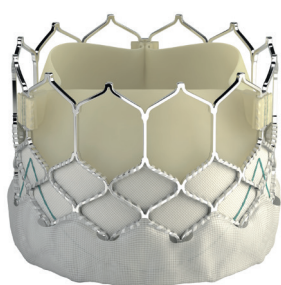
There is no difference in long-term survival between patients who are given a mechanical valve and those receiving a biosprosthetic valve, so there is no decisive argument in favor of one or the other. However, over time there has been a steady reduction in the number of mechanical valves fitted, with bioprosthetic valves now accounting for 70% of all artificial valves implanted.

AORTIC STENOSIS AND TAVI

For a long time, surgery was the only option for aortic stenosis, with a surgical risk of somewhere around 4% or 5% in the general population and much higher for elderly patients. The risk remains acceptable because patients, once they have undergone surgery, have a life expectancy considerably higher than that of the natural history and even comparable to that of a general population of the same age and sex. Surgery therefore brings a considerable benefit. However, relative survival is decreased in patients who undergo surgery at a young age, as they are exposed for longer to complications with the artificial valve.

In the past 10 years, a new minimally invasive procedure has made its appearance. TAVI (Transcatheter Aortic Valve Implantation) consists of percutaneous replacement of the aortic valve. This involves conserving the valve and inserting an aortic prosthesis via the femoral artery.

VALVE FOR TAVI



Source: Edwards Lifesciences

This very promising, less invasive technique is being used more frequently in patients for whom surgery is contraindicated (patients advanced in age, with too high a surgical risk). With TAVI, mortality is of the order of 5% to 10% in patients with a high surgical risk. However, for patients with a moderate, low or intermediate risk, surgery remains the preferred treatment.



The only effective treatment for heart valve disease is surgery.”

AORTIC REGURGITATION

Here the treatment is surgery. Depending on the results, patients operated on in NYHA class III or IV show excess mortality compared to the general population. However, if they are operated on in class I or II, their rate of survival is better, of the order of 80% compared to 50% in class III-IV at 10 years.

MITRAL REGURGITATION AND THE MITRACLIP[®]

Currently there are two solutions available for MR: surgical treatment and a transcatheter repair procedure.

Here the preferred surgical treatment is valve reconstruction or repair (unlike the aortic valve where the only option is replacement with an artificial valve); however, valve replacement may also be necessary in certain cases.

In organic MR, all symptomatic patients with severe regurgitation undergo surgery. When they are asymptomatic, the decision to operate depends on left ventricular involvement. Mortality linked to mitral valve replacement is higher than for aortic valve replacement: between 4% and 6% without a bypass compared to 6% to 10% with a bypass.

An instrumental treatment is currently being developed for the mitral valve. MitraClip, a transcatheter repair procedure, is the first non-surgical procedure for MR. It consists of a device that grips the valve cusps to secure them, not with a surgical suture but using a clip.

MITRAL STENOSIS

Generally in MS the mitral valve is dilated by a procedure called percutaneous balloon valvuloplasty. This consists of inserting a catheter into the valve in order to inflate a balloon at its tip to enlarge the stenotic mitral valve orifice. This is the technique of choice; if it does not work, the valve will be replaced. Outcomes at 20 years show that survival is excellent, about 85%. Valve replacement continues to be the preferred treatment for patients more severely affected.

MITRACLIP[®] - TRANSCATHETER MITRAL VALVE REPAIR



Source : abbott.com / Media-center

CONCLUSION

The etiology of heart valve disease has changed over the last few decades with the decline in acute articular rheumatism. Currently degenerative causes are most common, with aortic stenosis and mitral regurgitation dominating.

Although valve stenosis is progressive, discreet or moderate leaks can be well tolerated for decades. Improved diagnosis, monitoring using Doppler echocardiography and the progress made in surgical techniques have led to a considerable reduction in surgical risks and an improvement in long-term results.

The main prognostic factors for heart valve diseases are the severity of the dysfunction, symptomatology and deterioration of left ventricular ejection fraction.

The prognosis for severe heart valve disease is poor once symptoms have appeared and the LVEF has deteriorated. Prognosis for severe asymptomatic valve disease is better. The decision to operate must be taken in good time and sometimes immediately in selected patients.

Moderate valve disease has a good prognosis, and there are no indications for treatment. However, patients must be informed of the potential development of the disease, which is individually very difficult to predict. Regular monitoring is necessary to check the valve.

Surgery is effective. It restores a life expectancy comparable to that of the general population if it is not undertaken too late. Risk scores allow a reliable assessment of the surgical risk for most patients; their accuracy is lower in high-risk patients, making assessment on a case-by-case basis necessary. Patients who undergo surgery are exposed to risks specific to the type of artificial valve fitted, and specific monitoring is required.

The indications for surgery for ascending aortic disease depend essentially on aorta diameter and etiology.



APPROACH TO RISK ASSESSMENT AND CLAIMS MANAGEMENT

APPROACH TO RISK ASSESSMENT

PRINCIPLES

Find out the date of the aortic valve disease diagnosis

Track the development of the aortic valve disease until surgery becomes necessary

Be aware of the average duration of sick leave following aortic valve replacement, in the absence of any complications (approximately one and a half months)

FILE CONSTITUTION

Hospital discharge summary, surgery notes and follow-up reports

Results of examinations, in particular the examination leading to the diagnosis of aortic valve disease

APPROACH TO CLAIMS MANAGEMENT

A nuanced approach to the claim depending on:

The benefits being claimed for aortic valve disease: short-term TIW, death if complications

The type of replacement (mechanical valve or bioprosthetic valve)

The risk inherent in the treatment received, the drug therapy

The associated risk factors and comorbidities

ACUTE ARTICULAR RHEUMATISM (AAR)

Inflammatory disease resulting from a bacterial infection liable to lead to heart valve disease

ANNULOARTIC ECTASIA

Heart disease involving an ascending aorta aneurysm, dilation of the aortic annulus and an abnormality in the aortic valves (vulgaris medical)

AORTIC DISSECTION

A tear in the wall of the aorta

AORTITIS

Inflammation of the aorta

ATRIAL FIBRILLATION

Cardiac arrhythmia where the regular sinus rhythm is replaced by an irregular rhythm due to very fast and out of sync contractions of the atria

BICUSPID AORTIC VALVE

Malformation of the aortic valve, which has only 2 cusps (or leaflets) instead of 3

DIASTOLE-DIASTOLIC

Heart muscle relaxation phase when the left ventricle fills

DILATED CARDIOMYOPATHY

Frequent cardiomyopathy characterized by dilated ventricles and systolic dysfunction

DYSPNEA

Respiratory distress, shortness of breath

DYSTROPHY

Abnormal development of tissue or organ

EJECTION FRACTION (EF) OR LVEF

Left Ventricle Ejection Fraction: the quantity of blood ejected by the left ventricle in systole

ENDOCARDITIS

Bacterial infection of the heart valves

EROA / ERO

Effective Regurgitant Orifice Area

ISCHAEMIC HEART DISEASE

A condition affecting the coronary arteries

PARASTERNAL

Parallel plane to the sternum

SYSTOLE/SYSTOLIC

Phase when the heart muscle contracts; contraction of the left ventricle

SYSTOLIC-DIASTOLIC

Occurring in systole and in diastole

THROMBOSIS

Obstruction/blockage of a blood vessel by a blood clot

VALVE PROLAPSE

Condition in which valve's leaflet bulge back into the upstream chamber

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