

## Why do surgical mitral valve repairs fail?



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Mitral valve repair or reconstruction has become the gold standard to treat mitral regurgitation (MR), especially in degenerative MR. However, its long-term results are only known and published in very few selected centers with high volume, very low mortality, and close to a 100% repair rate.

The initial goal is to achieve a perfect immediate surgical result, which is the key to good long-term results—although not enough. How are immediate good results achieved?

We tend to believe that there is neither a magic solution nor a magic technique to obtain a perfect repair; rather, whatever the technique being used there are some rules that are intangible. Among these rules, 2 are essential:

- Achieve a harmonious closure line,<sup>1,2</sup> which means that the closure line should remain parallel to the posterior rim or to the ring annuloplasty, keeping all the way through a ratio of 2:3 for the anterior leaflet (AL) and 1:3 for the posterior leaflet (PL), from 1 commissure to the other. To avoid systolic anterior motion (SAM) the closure line can even be further posterior and the ratio could be 3:4 for the AL and 1:4 for the PL.
- Restore a good coaptation height,<sup>1,2</sup> which should be in between 7 and 9 mm at the level of A2/P2 after weaning from cardiopulmonary bypass. This parameter is not provided in any publication and perhaps would be worth knowing. It should be stressed that percutaneous therapies do not provide coaptation height.

Mechanisms of repair failure are procedure-related in approximately 50% of the cases for the Cleveland Clinic,<sup>3</sup> whereas procedure-related failure represent only 23% of the cases for Nishida and colleagues,<sup>4</sup> and 38% for Anyanwu and colleagues<sup>5</sup> at Mount Sinai. This failure mode takes place a soon as at discharge and within the first months after index operation and tend to occur within the first 3 years after initial repair. The causes are multiple, among which are incomplete initial repair, suture dehiscence, SAM with left ventricular obstruction, chordal



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### CENTRAL MESSAGE

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rupture (native chord or artificial neo chord), or hemolysis. Failures are valve-related in approximately the other 50% of the cases at the Cleveland Clinic,<sup>3</sup> but 71% for Nishida and colleagues<sup>4</sup> and 62% for Anyanwu and colleagues,<sup>5</sup> who segregate progression of original disease from new disease. Failure can also be related to both in some instances.

### WHAT ABOUT RESIDUAL MR?

#### Incomplete Initial Repair

Residual MR is key to avoid MR progression that can lead, in turn, to reintervention. It is now well known that even 1+ MR is a risk factor and that at the time of index operation, more than trace or mild is an indication for a second pump run to correct the residual regurgitation. Imielski and colleagues<sup>6</sup> published a series of 1137 patients with AL and bileaflet prolapse, as well as PL prolapse showing an extremely low incidence of reoperation at 1.9% at 10 years and a freedom from 2+ MR or more of 98.3% at 10 years.

Residual MR following cardiopulmonary bypass weaning is, most often, procedure-related and should be addressed as soon as possible. It is only recently and coming from expert centers that it well recognized that only trace or <1+ MR can be considered as acceptable.<sup>4</sup> The dilemma remains because there is a need to identify the cause and locate it precisely on 1 hand (requiring a team approach between the surgeon and the echocardiographer), and the risk of a second crossclamp and an increase in ischemic myocardial injury on the other. In

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some instances, the tissues are slightly stiff and a regurgitant jet at the closure line level can be produced. It can be useful to wait a little time for a better systolic pressure as well as for an improvement in diastolic dysfunction. It may be true in Barlow valves and might explain why resection is sometimes better to get a smooth surface of coaptation. Otherwise, any regurgitant jet providing a residual MR >1+ should be corrected immediately. Needless to say, if the residual MR originates from the body of the leaflet or close to the annulus it has to be fixed immediately because it can only progress—and often quite rapidly. This type of regurgitation is related to leaflet resection (quadrangular or triangular, or to a sliding plasty). Its mechanism is quite straightforward because once you have a suture within the leaflets, or between the leaflet and the annulus, such sutures must be tight to avoid any dead space or a tiny hole that could produce a leak. Resection is a mainstay and even those who advocate to respect rather than resect do resect when excess tissue is obvious. There is no specific resection technique leading to intraleaflet regurgitation, but rather the careful and tight suturing of the edges left after resection, to avoid intraleaflet regurgitation. There are no differences between isolated sutures and running sutures.

### **Suture Dehiscence**

When referring to such etiology, the problem is rarely a leaflet tissue dehiscence because great attention is given a not to overresect, as this would result in a suture under tension. Suture dehiscence is mainly related to ring annuloplasty, which is either too small—thus creating excess tension on the sutures—or due to annuloplasty stitches placed either into the leaflet tissue or outside the annulus. In both instances, the sutures were not placed properly. The most frequent dehiscence area is at the level of the AL. It is a complex area because it is the most fibrotic, at the level of the fibrous trigone, but also the most dangerous because bites that are too deep may hit the bundle of His and create an atrioventricular conduction disorder that requires a secondary permanent pacemaker. Not to mention the risk of encroaching on the aortic annulus or on an aortic leaflet thereby creating aortic insufficiency. Securing the ring annuloplasty at the fibrous trigone may seem like good advice, but it may be difficult to identify the fibrous trigone.

### **SAM Causing Left Ventricular Outflow Tract Obstruction**

Intraoperative SAM is a different entity than follow-up SAM, which takes place after discharge but usually quite early on. It has not been studied if SAM in these cases were diagnosed as soon as cardiopulmonary bypass was weaned and had resolved under medical management or if

such delayed SAM could occur in patients without any predicting signs.

Intraoperative SAM is always related to a PL remaining too high, along with some risk factors such as a thick septum and a pronounced angle between the left ventricle cavity and the left ventricular outflow tract. If SAM is identified during and after cardiopulmonary bypass weaning, many scenarios can occur. The worst is a high left ventricular outflow tract gradient >50 mm Hg along with more-than-moderate MR. Sometimes SAM is so severe that cardiopulmonary bypass cannot be weaned. At that point, going back immediately is mandatory. The options are an Alfieri stitch to limit the AL excursion or taking down the ring annuloplasty, restricting or reducing the PL height, and implanting a bigger annuloplasty ring. If the situation is less obvious, such as a gradient <50 mm Hg and mild-to-moderate MR, it is time to assist the heart to allow diastolic dysfunction to recover; stop, if possible, any inotropes and pace the heart sequentially. If the blood pressure is low (<90 mm Hg), introduce careful volume loading along with pure vasopressors such as norepinephrine. It becomes more crucial than ever to monitor left ventricle filling by transesophageal echocardiography. Filling associated with these maneuvers resolves the MR completely and decreases the left ventricular outflow tract gradient so the situation can be acceptable. However, after decannulation and protamine being given, there is always a transition phase with a drop in blood pressure and a left ventricle that looks empty. If at that stage the SAM recurs, again filling slowly is necessary. If despite all these adjunctive methods SAM remains, the decision has to be made to go back on pump and address the SAM surgically.

Secondary SAM is rare, and obviously there would have been signs that were ignored or a resolved SAM at the index operation. It has been mentioned that a small left ventricle is a risk factor. The more asymptomatic a patient is at the time of surgery, the more his or her left ventricle is not very dilated.

If a significant gradient >50 mm Hg is still found and there is still some MR, an exercise test will show if there is any significant increase or symptoms. If that is the case, redo surgery will be required. Literature does clarify in which clinical settings such a scenario occurs and which percentage of reoperations it represents. Obviously if there are signs of SAM, it is far better to address them at the time of surgery.

### **Chordal Rupture or Inadequate Height (Native or Artificial)**

Native chord rupture is rare and was reported when using the initial chordal shortening described by Imielski and colleagues<sup>6</sup> using a trench in the tip of a papillary muscle, and

then burying the chord into the trench, with a monofilament suture using a figure of 8. Some ruptures were reported by the Cleveland Clinic—and others—and the technique was progressively abandoned.

Conversely, chordal transfer and chordal transposition are very safe and effective, and we use them preferably to any other technique. Recently, Imielski and colleagues<sup>6</sup> reported more 1000 patients with 10 years' follow-up and showing, using chordal transfer and no neochordae, a 98.1% freedom from reoperation and a 98.3% freedom from 2+ MR or more. Such results reemphasize how much native chords can be efficient and safe in the long-term.

The use of neochords is widely adopted as chordal substitutes nowadays. However, it is to be stressed that there are more than 100 techniques described to use them adequately, which may explain some unexpected results. Among many techniques, the loop technique, described by Imielski and colleagues,<sup>6</sup> seems to be the most reliable. But even this technique has recently been subjected to revision because the trend is to shorten the loop length from an average of 18 to 16 mm once the left ventricle has improved and its size reduced by reverse remodeling, to avoid a new prolapse due to too-long artificial chordae. Placing artificial chordae can be easy if limited, but gains complexity when there are too many because the aim is to get the most evenly distributed coaptation line. Another issue that is rarely reported is the rupture of artificial chordae: Some cases have been published, but not enough to assess the magnitude of the problem.

### Hemolysis After Mitral Valve Repair

It is quite uncommon and is always related to high velocity jets. This subset may happen in 2 instances: either a tiny hole in a sutured leaflet (after resection or a sliding plasty) or in relation to a knot of the sutures to secure an annuloplasty ring or band. Any hemolysis can be controlled medically by iron supplement drugs and sometimes by using erythropoietin. Escalating to blood transfusion is a turning point that might indicate the need for more aggressive management. Obviously, in such cases the risk and influence of hemolysis has to be outweighed by the risk of redo surgery and will depend on the severity of hemolysis and the patient's comorbidities and age allowing or not a redo surgery. It warrants close surveillance. If hemolysis influences the hemoglobin level, requires regular blood transfusions, and influences renal function, it then requires reintervention. New percutaneous plugs may be used because the technology improves constantly and may reduce hemolysis to an acceptable level and avoid redo surgery if successful.

### Valve-Related Failure

The incidence of valve-related failure cannot be controlled and has nothing to do with the initial repair. It is either due to progression of the degenerative disease or to endocarditis. Progression of the degenerative disease is a new lesion in an area of the valve deemed nonprolapsed at the initial repair. It seems quite difficult to be sure that there is no billowing, which increases the tension on a localized area and creates a secondary prolapse.<sup>7</sup> The posterior commissure is the weakest part of the mitral apparatus because there are no surrounding structures to support it. Therefore, any billowing at this level might create favorable ground for a delayed prolapse.

An uneven coaptation closure line witnesses an irregular closure line, which does not create any residual regurgitation at index operation, but at the level of a short coaptation height, the tension increases and may become a prolapse later on. The frontier between a new lesion due to the progression of degenerative disease and a pre-existing favorable condition not identified initially is very tiny. That is why the artificial classification mentions that mitral valve repair failure may be both repair-related and valve-related.

A pannus formation is a new lesion and can be enhanced by the type of annuloplasty ring. Similarly, stiffening of the leaflet may create an obstacle such as mitral stenosis and, at the same time, a regurgitation due to the lack of pliability of the leaflets. On the contrary, endocarditis is a true new entity and creates lesions on a repaired valve similar to those in an unrepaired native valve. The indications to intervene remain similar to any endocarditis if not earlier because there is a prosthetic ring.

### Incidence of Mitral Repair Failure

The incidence of mitral repair failure is not well known and to be precise should be derived from a longitudinal study to have a real-time picture when it takes place. Three parameters are commonly used: mortality at 10 years, MR >2+ at 10 years, and reoperation rate at 10 years. Mortality at 10 years depends not only on hospital mortality, but also on risk factors of the cohort, that give an indication. Similarly, reoperation at 10 years is highly subjective because some are showing 3+/4+ MR and remain poorly symptomatic with no left ventricle dilatation. The only objective parameter is MR grading at regular intervals. For instance, David and colleagues<sup>8,9</sup> provide a reoperation rate at 20 years of 4.5%, which is very low but a 6% MR rate >2+ at 10 years and a 13% MR rate >2+ at 20 years.

However, the reoperation rate is not an objective parameter because some patients showing a residual/recurrent MR grade 3+/4+ might remain asymptomatic and do not dilate their left ventricle and therefore do not undergo reoperation for some time. Moreover, some patients are lost to

follow-up; therefore, the real rate might be inaccurate, especially given that all published studies are retrospective.<sup>10</sup>

Reoperation rates vary from 4.5% at 20 years for David and colleagues<sup>8</sup> to 4% at 10 years for Pfannmueller and colleagues<sup>11</sup> to 7.8% at 10 years for Lang and colleagues<sup>12</sup> but showing quite different outcomes according to AL (16%) and PL (7.1%). For Nishida and colleagues,<sup>4</sup> it reaches 5.6% and for Dumont and colleagues<sup>3</sup> it reaches 5.2% at 10 years.

It seems from the literature that reoperation takes place after a failed mitral repair rather than within the first 4 years for procedure-related failures and that otherwise the failures tend to disappear, whereas valve-related failures occur during all the follow-up points and that both mechanisms tend to plateau after 10 years.<sup>3</sup> On the contrary, for Anyanwu and colleagues,<sup>5</sup> failures tend to occur in a linearized rate of 1% to 4% per year.

Very recently, Imielski and colleagues<sup>6</sup> published a series of more than a 1160 mitral valve repairs with a longitudinal follow-up in degenerative disease showing striking results with a freedom from reoperation at 10 years of 99.4% that has become the landmark and freedom from >2+ MR of 98.3% at 10 years. Also, and quite usefully, these authors introduced the concept of mitral valve repair failure, which is a composite of >2+ MR and reoperation reaching a staggering 98.1% at 10 years.<sup>6</sup> This article should become the benchmark against which any other approach or technique, surgical or transcatheter, is compared.<sup>5</sup> Using the repair failure rate described previously, David and colleagues<sup>8,9</sup> report 4.5% reoperation and 13% recurrent residual MR at 20 years, bringing the mitral repair failure rate to 17.5% at 20 years.

Many well-recognized groups have published results that are difficult to understand, such as Pfannmueller and colleagues,<sup>11</sup> with a report of 2134 cases in which there is mention of 30 days' mortality and survival at 10 years along with a reoperation rate at 10 years but nothing on residual/recurrent MR, which is the only objective parameter to assess one's results in mitral valve repair. On the contrary, Lang and colleagues<sup>12</sup> published a series of 346 patients providing a complete analysis with survival at 10 years but also an incidence of recurrent MR at 13.3% at 10 years adding more data by providing the difference between AL showing 27.13% MR at 10 years, whereas the PL reached 13.09%. Then the reoperation rate reached 7.84% with again a significant difference in between AL and PL. If the mitral repair failure criteria are applied, in the first series it is unknown and in the second it reaches

21.15%, which has to be compared with Imielski and colleagues' 1.9%.

Finally, too few surgical groups know their longitudinal results of recurrent MR at 10 years, data that are the only objective way to see if repair results are within the best standards. In the era of challenging percutaneous therapies, surgery should be able to provide the best results and remain the first option when dealing with degenerative MR.

### Conflict of Interest Statement

The authors reported no conflicts of interest.

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