WHY DO SURGICAL MITRAL VALVE REPAIRS FAIL?

Gilles Dreyfus\textsuperscript{a}, MD, Filip Dulguerov\textsuperscript{b} PhD,

\textsuperscript{a}Hôpital Européen Georges Pompidou, Department of Cardiac Surgery, Paris, France
\textsuperscript{b}University Hospital of Lausanne, Department of Cardiac Surgery, Lausanne, Switzerland

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**Corresponding author:** Gilles Dreyfus, MD, PhD, Hôpital Européen Georges Pompidou, Department of Cardiac Surgery, Paris, France. E-mail: gillesdreyfus1@gmail.com

**Central Picture Legends:** Picture 1: Gilles Dreyfus; Picture 2: Filip Dulguerov
Abbreviations

AL – Anterior Leaflet
CPB – Cardiopulmonary Bypass
LVOT – Left Ventricular Outflow Tract
MR – Mitral Regurgitation
PL – Posterior Leaflet
SAM – Systolic Anterior Motion
TOE – Transoesophageal Echocardiography

Central Message

Mitral valve repair is the gold standard to treat degenerative mitral regurgitation. The initial goal is to achieve a “perfect” immediate surgical result which is the key to good long-term results.
Mitral valve repair or reconstruction has become with time 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 centres, 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 to achieve immediate good result?**

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

1) **Achieve a harmonious closure line** [1,2], 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 one commissure to the other. In order 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.

2) **Restore a good coaptation height** [1,2], which should be in between 7 to 9 mm at the level of A2/P2 after cardiopulmonary bypass being weaned. 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:**

1) **Procedure related** in approximately 50% of the cases for the Cleveland Clinic [3], whereas it represents only 23% of the cases for Nishida [4], and 38% for Anyanwu [5] 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 incomplete initial repair, suture dehiscence, SAM with left ventricular obstruction, chordal rupture (native chord or artificial neo chord) or haemolysis.

2) **Valve related** in approximately the other 50% of the cases at the Cleveland Clinic [3], but 71% for Nishida [4] and 62% for Anyanwu [5], who segregates progression of original disease from new disease

3) **Both** in some instances.

### What about residual MR?

1) Incomplete initial repair

Residual MR is key to avoid MR progression and 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. Mc Carthy [6] published a series of 1137 patients with AL and bi leaflet prolapse, as well as PL prolapse showing an extremely low incidence of reoperation at 1.9% at ten years and a freedom from 2+MR or more of 98.3% at ten 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 centres that it well recognized that only trace or less than 1+ MR can be considered as acceptable [4]. The dilemma however remains, as there is a need to identify the cause and locate it precisely on the one hand (requiring a team approach in between the surgeon and the echocardiographer), and the risk of a second cross clamp and an increase in ischemic myocardial injury on the other hand. In some instances, the tissues are slightly stiff, and it can produce a regurgitant jet at the closure line level. 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 in order to get a smooth surface of coaptation. Otherwise, any regurgitant jet providing a residual MR greater than 1+ should be corrected immediately. Needless to say, that if the residual MR originates from the body of the leaflet or close to the annulus it has to fixed immediately as it can only progress and often quite rapidly. This type of
regurgitation is related to leaflet resection (quadrangular, triangular or to a sliding plasty). Its mechanism is quite straightforward, as once you have a suture within the leaflets, or in between the leaflet and the annulus, such sutures must be tight to avoid any dead space or a tiny hole, which in turn could produce a leak. Resection is a mainstay and even those who advocate “respect rather than resect “do resect when excess tissue is obvious. There is no specific resection technique leading to intra leaflet regurgitation but rather the careful and tight suturing of the edges left after resection, to avoid intraleaflet regurgitation. There are no difference in between isolated sutures or running sutures.

2) Suture dehiscence

When referring to such aetiology it is rarely a leaflet tissue dehiscence as wide quadrangular resection have been abandoned, and care is taken when performing resection not to over resect in order to leave enough tissue and avoid suturing 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 but in both instances not placed properly. The most frequent dehiscence area is at the level of the anterior leaflet. It is a complex area as it is the most fibrotic, at the level of the fibrous trigone, but also the most dangerous as too deep bites may hit the bundle of His and create an atrioventricular conduction disorder requiring a secondary permanent pacemaker and not to mention the risk to encroach the aortic annulus or worse an aortic leaflet thereby creating aortic insufficiency. When some advocate to secure the ring annuloplasty at the fibrous trigone it may seem a good advice, but it may be difficult to identify where is the fibrous trigone.

SAM causing LVOT 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 these SAM were diagnosed as soon as
Cardiopulmonary Bypass (CPB) was weaned and had resolved under medical management or if such delayed SAM could occur in patients without any predicting signs before.

Intraoperative SAM is always related to a remaining too high PL, along with some risk factors, such as a thick septum and a pronounced angle in between the LV cavity and the LVOT. If SAM is identified during and after CPB weaning many scenarios can occur. The worst is a high Left Ventricular Outflow Tract (LVOT) gradient above 50 mm of Hg along with more than moderate MR. Sometimes SAM is so severe that CPB cannot be weaned, then going back immediately is mandatory. The options are either 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 less than 50 mm of Hg and a mild to moderate MR, then comes the time to assist the heart to allow diastolic dysfunction to recover, stop, if possible, any inotropes and pace the heart sequentially and if the blood pressure is low (below 90 mm Hg) introduce careful volume loading along with pure vasopressors such as Norepinephrine. It becomes more crucial than ever to monitor LV filling by Transesophageal Echocardiography (TOE). Either filling associated with all these manoeuvres resolves the MR completely and decreases the LVOT gradient 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 an LV which 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 made to go back on pump and address SAM surgically.

Secondary SAM is rare and obviously there would have been premonitory signs that were ignored or a resolved SAM at index operation. It has also been mentioned that “small LV” is a risk factor. The more the patients are being asymptomatic at the time of surgery the more their LV is not very dilated. If a reverse remodelling takes place as quickly as one month after surgery, the ratio in between the surface of the ring annuloplasty and the amount of leaflet tissue is a key factor to produce a secondary SAM. It can also be enhanced by the introduction of vasodilators, often indicated after MV repair in order to promote reverse remodelling.

The first step would be when recognized at echocardiogram and the LVOT gradient being assessed to quantify both the MR grade (if there is any) and the gradient. Then optimize medical treatment, withdrawing or decreasing diuretics, and vasodilators and introducing or increasing beta blocking agents.
If a significant gradient above 50 mmHg 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 not allow to find in which clinical setting such scenario occurs and which percentage of reoperation 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 Carpentier [6] 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 eight. 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 it preferably to any other technique. Recently, McCarthy reported above a thousand patients with ten years follow up and showing, using chordal transfer and no neo chordae, a 98.1% freedom from reoperation and a 98.3% freedom from 2+ MR or more [6]. Such results reemphasize how much native chords can be efficient and safe in the long term.

The use of neo chord is widely adopted as chordal substitute, nowadays. However, it is to be stressed that there are over hundred techniques described to use them adequately, which may explain some unexpected results. Among many techniques the loop technique, described by Mohr seems to be the most reliable [6]. But even this technique has recently been subjected to revision as the trend is to shorten the loop length, from an average of 18 mm to 16 mm, as once the LV has improved and reduced its size by reverse remodelling, 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, as the aim is to get the most evenly distributed coaptation line. Another issue which is rarely reported is the rupture of artificial chordae, some cases have been published but not enough to assess the magnitude of the problem.
Haemolysis after mitral valve repair

It is quite uncommon and is always related to high velocity jets. This subset may happen in two instances. Either a tiny hole in a sutured leaflet (after resection or a sliding plasty), or in relation with a knot of the sutures to secure an annuloplasty ring or band. As any haemolysis it can either be controlled medically by iron supplement drugs and sometimes by using erythropoietin. Escalating to blood transfusion is a turn point which might indicate the need of more aggressive management. Obviously in such cases the risk and impact of haemolysis has to be outweighed with the risk of redo surgery and will depend on the severity of haemolysis and the patients comorbidities and age allowing or not a redo surgery. It warrants a close surveillance. If haemolysis impacts the haemoglobin level, requires regular blood transfusions and might even impact renal function it then requires reintervention. New percutaneous plugs may be used as the technology improves constantly and may reduce haemolysis to an acceptable level and avoid redo surgery if successful.

Valve related failure

Its incidence cannot be controlled and has nothing to do with the initial repair. It is either due to progression of the degenerative disease or to an endocarditis.

Progression of the degenerative disease is a new lesion in an area of the valve deemed non prolapsed at the initial repair. It seems quite difficult to be sure that there was no billowing, which increases the tension on a localized area, and creates secondarily a prolapse [8]. The posterior commissure for example is the weakest part of the mitral apparatus as there are no surrounding structures to support it: therefore, any billowing at this level might create a favourable 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 in between a new lesion due to the progression of degenerative disease and a pre-existing favourable condition not identified initially is very tiny. That is why the artificial
A pannus formation is a new lesion and can be enhanced by the type of annuloplasty ring. Similarly, the stiffening of the leaflet may create an obstacle such a 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 a unrepaired native valve. The indications to intervene remain similar to any endocarditis if not earlier as 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 more than 2+ at 10 years and reoperation rate at 10 years. Mortality at 10 years depends not only on hospital mortality but on risk factors of the cohort which gives an indication but has some kind of non-accurate data. Similarly, reoperation at 10 years is highly subjective as some are showing 3+/4+ MR and remain poorly symptomatic with no LV dilatation. The only objective parameter is the MR grading at regular interval. For instance, T David provides a reoperation rate at 20 years of 4.5% which is very low but a 6% MR rate greater than 2+ at 10 years and a 13% MR rate greater than 2+ at 20 years [9,10].

It is the reoperation rate at 10 years for instance. However, it is not an objective parameter as some patients showing a residual/recurrent MR grade 3+/4+ might remain asymptomatic and do not dilate their LV and therefore are not reoperated for some time. Moreover, some patients are lost for follow up, therefore the real rate might be inaccurate, especially given that all published studies are retrospective [11].

Reoperation rates vary from 4.5% at 20 years for David [9], to 4% at 10 years for Pfanmuller [11], to 7.8% at 10 years for Lang but showing quite different outcome according to anterior leaflet at 16%, and for posterior leaflet at 7.1%. For Nishida [4] it reaches 5.6 % and for Dumont [3] it reaches 5.2% at 10 years.
It seems from the literature that reoperation take place after a failed mitral repair rather 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 and that both mechanisms tend to plateau after 10 years [3]. On the contrary for Anyanwu failures tend to occur in a linearized rate of 1 to 4% per year [5].

Very recently Mc Carthy published [6] 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% which has become the landmark and freedom from more than 2+ MR of 98, 3% at 10 years. Also, and quite usefully, he has introduced the concept of mitral valve repair failure: which is a composite of more than 2+ MR, and reoperation reaching a staggering 98,1% at 10 years. This manuscript should become the benchmark results to which any other approach or technique, surgical or transcatheter should be compared to [5]. Using the repair failure rate described previously T. David has 4,5% reoperation and 13% recurrent residual MR at 20 years bringing mitral repair failure rate at 17,5% at 20 years.

Many well recognized groups have published results that are difficult to understand such as Pfanmuller [12] with a report of 2134 cases in which there is mentioned 30 days mortality and survival at 10 years along with reoperation rate at 10 years but nothing on residual / recurrent MR which is the only objective parameter to assess one’s results in MV repair. On the contrary Lang [13] has published a series of 346 patients providing a complete analysis with a survival at 10 years but also an incidence of recurrent MR at 13, 3% at 10 years adding more data by providing the difference in between anterior leaflet showing 27,13% of MR at 10 years whereas the posterior leaflet 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, in the second one it reaches 21, 15% which has to be compared to Mc Carthy’s 1, 9%.

Finally, too few surgical groups do not know their longitudinal results of recurrent MR at 10 years which is the only objective data to see if the repair results are within the best standards. In the era of challenging percutaneous therapies surgery should be able to provide the best results to remain the first option when dealing with degenerative MR.
References


