No ring at all in mitral valve repair: indications, techniques and long-term outcome

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Received 1 March 2013; received in revised form 11 May 2013; accepted 17 May 2013

Abstract

OBJECTIVES: In mitral valve (MV) repair, we adhere to a biological concept of preservation of the native valves and avoidance of any prosthetic materials except for sutures whenever possible. Untreated autologous pericardium is the biological tissue of choice we use to support the repair. We report our 25-year institutional experience with no-ring MV repair in terms of indications, repair techniques and long-term results.

METHODS: Patients with ruptured chordae or posterior leaflet prolapse from degenerative MV disease, active infective endocarditis (IE), ischaemic mitral incompetence (IMI), annular dilatation with or without ruptured chordae along the posterior leaflet, and various lesions of the MV and its subvalvar apparatus underwent suture-repair techniques tailored to their valve morphology. These are personal series of modified Gerbode–Hetzer posterior leaflet plication and modified Paneth–Hetzer posterior annulus shortening techniques. Indications for the use of each technique and technical details are described in this report.

RESULTS: Modified Gerbode–Hetzer posterior leaflet plication: mean duration of the follow-up is 15.84 ± 0.58 years. Overall freedoms from reoperation and cumulative survival rate are 55.4 ± 4.7 and 44.7 ± 5.4%, respectively. Freedom from reoperation is 83.5 ± 4.3%, in ruptured chordae from degenerative disease (n = 161), 74.4 ± 10.1% in active infectious endocarditis (IE) (n = 22) and 100% from both ruptured chordae of ischaemic origin (n = 10) and deceleration trauma (n = 1), respectively. Likewise, freedoms from reoperation at a mean duration of the follow-up of 11.2 ± 7.2 years in 62 children stratified based on age groups are: <3 months: 61.4 ± 2.7%; 3 months to 2 years: 78.7 ± 3.7%; 2–18 years: 97.1 ± 2.4%. Modified Paneth–Hetzer posterior annulus shortening: Mean duration of the follow-up is 11.98 ± 1.14 years. Overall freedoms from reoperation and cumulative survival rate in 179 patients are 82.95 ± 4.1 and 63.4 ± 8.5%, respectively. Freedom from reoperation is 85.9 ± 1.3% in patients with annular dilatation from any form of cardiomyopathy (n = 81), 78.4 ± 5.6% in those with IMI (n = 75) and 100% in those who underwent asymmetric valve repair (n = 23). In IMI, mean New York Heart Association functional class, ejection fraction and degree of mitral incompetence (MI) were significantly abated (P = 0.001). In 78 children, freedoms from reoperation at a mean duration of the follow-up of 11.2 ± 7.2 years stratified based on age groups are as follows: <3 months: 82.79 ± 3.5%; 3 months to 2 years: 71.6 ± 5.3%; 2–18 years: 85.1 ± 4.4%.

CONCLUSIONS: No-ring MV repair using the aforementioned techniques in patients with MI resulting from chordal rupture, degenerative valve disease, IE, annular dilatation and posterior leaflet prolapse and from IMI as well as various MV lesions in children offers excellent long-term functional results with satisfactory freedom from reoperation.

Keywords: Mitral valve · Mitral incompetence · Posterior leaflet plication · Annuloplasty · Autologous pericardium · Systolic anterior motion

INTRODUCTION

Preservation of the normal physiology and mobility of the mitral valve (MV) has long been a goal of MV repair. To remodel the annulus to its correct shape and size, preserve its sphincter function, and decrease tension on the sutures, Carpentier [1] introduced, in 1969, the use of prosthetic rings for annuloplasty. Since then, numerous synthetic rings in various geometrical forms for annuloplasty support have become commercially available [2–7]. Although several groups reported good results without prosthetic ring stabilization [8], there is broad consensus among surgeons that ring annuloplasty is a mandatory step in MV repair. The few long-term studies with different types of rings, such as a report on 1072 patients from the Cleveland Clinic [9], albeit not prospective-ly randomized, strongly supported the need for ring annuloplasty but did not show outcome differences between the different types of rings. Moreover, aside from high cost and limited size availability, concerns have been raised about LV outflow obstruction and impairment of left ventricular function caused by a prosthetic ring [10].

For over two decades, we adhered to a biological concept of preservation of the native valves precluding any prosthetic materials, except for sutures, whenever possible. The basic repair principles being followed include: (i) achievement of rapid and
complete closure of the mitral orifice by an anterior leaflet that is very mobile by effecting a large coaptation area when the posterior leaflet is brought closer to the anterior leaflet, (ii) no placement of sutures along the anterior annulus, (iii) achievement of a minimal final MV orifice of 3.5 cm² in adults and not <10% below norm according to body surface area in children, (iv) accomplishment of primary repair only by sutures, (v) use of a pericardial strip to stabilize the suture-dependent repair and enhance the posterior leaflet coaptation surface area without further annulus shortening. (vi) application of additional sutures to secure anchorage of running sutures, pericardial pledgets and pericardial strip, when necessary and (vii) cautious circumvention to avoid systolic anterior motion (SAM) caused by excessive annulus reduction and a coaptation area made excessively wide.

To realize this concept, the authors use untreated autologous pericardium to support and stabilize posterior annuloplasty. It is an excellent and stable biological reconstruction material to support posterior annulus shortening, and increases leaflet coaptation area while allowing MV growth [11], which is particularly important in children. There has been no incidence of infection or thromboembolism, no shrinkage, no distension, no calcification and no tear or rupture. Furthermore, anticoagulation is not necessary. Pericardial strip annuloplasty corrects and prevents further annular dilatation, maintains the physiological annular movements, ensures the durability of the repair and avoids the negative effects of rigid fixation of the annulus.

The mitral annulus is dynamic—it changes shape, becoming circular during diastole and elliptical in systole [12]. The anterior annulus is part of the fibrous skeleton of the heart: dilatation involves only the posterior part. Our aim, aside from stabilizing the repair, is to restore a normal annulus shape with a technique that best preserves its physiological contractile properties, while allowing valve remodelling.

The pericardium

Autologous pericardium is not only costless and readily available with simple preparation in the operating room, but its flexibility maintains the physiological movement of the mitral annulus. It can be applied to the posterior annulus, the main target of mitral annuloplasty, to correct the annular dilatation, increase the leaflet coaptation, reinforce the annulus sutures and prevent future annular dilatation. Its ease of handling and its malleability make it an obviously excellent choice to correct or eliminate a defect. We do not treat the pericardial strip with glutaraldehyde, which was reported to enhance the tissue durability, prevent calcification, endow the pericardium with greater resistance to retraction and degeneration and increase the ease of handling while its intrinsic pliability was retained [13]. In our institution, we are convinced that using untreated autologous pericardium makes it more flexible to maintain the physiological annular movement and construct a soft reinforcement that conforms to the natural tri-dimensional geometry of the mitral annulus. With absence of thrombogenicity and calcification as evidenced during reoperations, it offers long-term durability. Posterior annulus stabilization with untreated autologous pericardium has a positive influence on left ventricular function while preserving the contractile properties of the mitral annulus. These benefits, in addition to the effective functioning of the remodelled valve, the preservation of the natural shape of the valve and allowing the valve to grow as the patient grows older, make this technique a useful surgical tool for MV reconstructive surgeries. Our institutional experience has proven this concept to be reliable and long-lasting.

This report provides compelling evidence that no-ring MV repair techniques, to correct mitral insufficiency for the lesions described using an untreated autologous pericardial strip to stabilize the repair produces optimal and highly reproducible long-term results.

PATIENTS AND METHODS

The Institutional Review Board approved this retrospective/prospective study and waived the need for patient consent.

Indications and techniques of no-ring MV repair

Techniques have been based on concepts dating back to the pioneering time in the 1960s [14, 15] and 1970s [16, 17] which the authors have further modified and advanced [18, 19]. These consist of the modified Gerbode–Hetzer posterior leaflet plication and modified Paneth–Hetzer posterior annulus shortening technique [20].

No-ring modified Gerbode–Hetzer posterior leaflet plication.

The no-ring modified Gerbode–Hetzer posterior leaflet plication is generally indicated in rupture of chordae of the posterior leaflet, which mostly occurs in the central scallop (P2). There are very few cases with excessive prolapse where a localized plication on other areas of the posterior leaflet is strongly advisable and reliable.

Ruptured chordae are found typically in A. degenerative MV disease, prolapse syndrome occurring mostly in males between 20 and 60 years of age with peak occurrence at 40–50 years, from an unknown mechanism; in Barlow’s disease, and in floppy valves in Marfan’s syndrome; in the latter two the pathophysiology is a prolapse or presence of a minimal coaptation area that tends to eventually rupture; B. active endocarditis, often on the basis of pathophysiological conditions specified in A; C. in very few cases of ischaemic disease, when a tip of papillary muscle becomes infarcted and D. rarely, as a sequel of blunt or deceleration trauma.

Table 1 summarizes the demographic profile of 194 patients with MV insufficiency from ruptured chordae where modified Gerbode–Hetzer posterior leaflet plication technique was employed.

Surgical technique

MV repair was performed through a median sternotomy or right anterior thoracotomy in the fifth intercostal space under cardiopulmonary bypass. Myocardial protection was provided mostly with blood cardioplegia under normothermia or antegrade intermittent cold crystalloid cardioplegia with topical hypothermia. Through a left atriotomy along the interatrial groove, the mitral annulus, leaflets, chordae tendineae and papillary muscles were exposed and meticulously inspected to determine the precise nature of the lesion. Leaflet coaptation was assessed with a forceful transvalvular injection of saline with a bulb syringe. Using a nerve hook, the coaptation of the anterior and posterior leaflet with regard to the presence of sufficient tissues along the coaptation plane was assessed. The valve orifice area was measured with a Hegar dilator and, more recently, with a Ziemer–Hetzer valve sizer (Fehling Instruments GmbH, Karlstein, Germany).
Prolapse can occur anywhere along the posterior leaflet, although it is most commonly found in the region of P2 (Fig. 1A), which may lead to chordal rupture. The *modified Gerbode–Hetzer posterior leaflet plication* technique is a repair strategy wherein the flail segment is plicated towards the left ventricle in a V-shaped fashion with interrupted mattress sutures pledged with untreated autologous pericardium attaching the P1–P3 segment (Fig. 1B). When competence and size are satisfactory (Fig. 1C), a strip of autologous pericardium is sutured continuously onto the posterior annulus (Fig. 1D) with precautions to avoid further annular narrowing (Fig. 1E).

**Active infective endocarditis.** The goals of surgery in infective endocarditis (IE) are to eradicate the focus of infection, repair destroyed valve structures, and prevent a relapse of infection. MV repair preserves not only the valve and its subvalvar apparatus but the ventricular function as well. Standard operative principles are adequate debridement and resection of all infected tissues and thorough washing with povidone-iodine solution. When sufficient valve tissue remains after meticulous removal of vegetations and all infected tissues [21, 22], valve repair becomes feasible. Repair using untreated autologous pericardial strips and pledgets for reinforcement may be acceptable with minimal insufficiency, to allow the infection to heal. Reoperation, when necessary, may then be performed in an unaffectected valve status. We have performed MV repair in 22 patients using the modified Gerbode–Hetzer posterior leaflet plication technique (Fig. 2A–C). Leaflet perforations, when found, were either closed directly or with an autologous pericardial patch. Reimplantation of ruptured chordae was done in 1 patient. In annular abscess, implantation of a stentless xenograft without any prosthetic material is highly recommended. In abscess formation and destruction of the aorto-mitral curtain, the authors performed MV replacement with a stentless xenograft, connecting this to the anterior mitral leaflet of the homograft in the aortic position to restore the cardiac skeleton.

**No-ring modified Paneth–Hetzer posterior annulus shortening technique.** The no-ring modified Paneth–Hetzer posterior annulus shortening technique is indicated in A. all cases of dilated annulus seen in idiopathic cardiomyopathy, secondary cardiomyopathy in valve disease with impaired LV function (ischaemic or non-ischaemic); B. ischaemic mitral incompetence (IMI) from either global myocardial ischaemia, postinfarction ischaemia or transient ischaemia. In these situations, the MV insufficiency is mostly due to incompetence around the anterior and/or posterior leaflets, related to ischaemia of the inferoposterior wall of the left ventricle. This occurs mostly in the obstruction of the right coronary artery and/or left circumflex coronary artery and C. in all cases of asymmetric valve repair, such as in closure of a leaflet perforation and unilateral commissuroplasty, to stabilize the repair. In this situation, a pericardial strip applied in the posterior annulus becomes particularly important.

Table 1 summarizes the demographic profile of 179 patients in whom the no-ring modified Paneth–Hetzer posterior annulus shortening technique was employed.

**Surgical technique.**

**No-ring modified Paneth–Hetzer posterior annulus shortening.** The *no-ring modified Paneth–Hetzer posterior annulus shortening* is performed by running a pericardium-pledgeted 3-0 polypropylene suture through the fibrous body of the trigone and tying it (Fig. 2A), after which it is run continuously along the annulus from one trigone towards the midsection of the posterior annulus. The same is done on the opposite trigone. These sutures are then tied over the aforementioned valve sizer to prevent over-narrowing of the valve orifice. When competence is assured, both sutures using the same needles are passed onto an autologous pericardial strip that is then attached to the posterior annulus from the midsection towards the trigone (Fig. 2B) by continuous sutures in a through-and-through fashion. Leaflet coaptation is tested by a forceful transvalvular saline injection to look for residual regurgitation. In anterior leaflet prolapse, the then-wider coaptation plane will eliminate prolapse. Shortening the posterior annulus produces wide and even coaptation, in such a way that when the anterior mitral leaflet closes, the border between the smooth and rough surface of the anterior leaflet forms the closure line with the posterior leaflet, without folding (Fig. 2C).

We are cautious to obviate the occurrence of the SAM phenomenon seen as ‘folding’ of the anterior leaflet when the valve is tested with transvalvular saline instillation. The folding appears when the valve opening is made too narrow by overshortening of the posterior annulus [20].

The *philosophy of a functional valve repair technique in IMI* In IMI, mitral insufficiency may be caused by dilatation of the annular-ventricular apparatus or dysfunction of the LV wall and papillary muscles or a combination of the two, resulting in failure

Table 1: Demographic profile of 194 patients with MV insufficiency from ruptured chordae in whom modified Gerbode–Hetzer plication plasty technique was employed

<table>
<thead>
<tr>
<th>Aetiology of ruptured chordae</th>
<th>n</th>
<th>Mean (median, range) years</th>
<th>NYHA functional class</th>
<th>Severity of mitral insufficiency* (mean grade)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (degenerative n = 108, Barlow’s disease n = 37, floppy valve in Marfan’s syndrome n = 18)</td>
<td>161</td>
<td>46.5 ± 14.8 (45.0, 20.9–86.2)</td>
<td>III–IV</td>
<td>3.8</td>
</tr>
<tr>
<td>B (active IE)</td>
<td>22</td>
<td>57.5 ± 13.9 (59.5, 23.9–82.9)</td>
<td>III–IV</td>
<td>3.5</td>
</tr>
<tr>
<td>C (IMI)</td>
<td>10</td>
<td>66.8 ± 10.2 (66.5, 53.7–82.7)</td>
<td>III–IV</td>
<td>3.8</td>
</tr>
<tr>
<td>D (deceleration trauma)</td>
<td>1</td>
<td>14.8</td>
<td>III</td>
<td>4</td>
</tr>
</tbody>
</table>

a*Echocardiographic evaluation of mitral insufficiency based on regurgitant volume (regurgitant fraction, %): 0—no regurgitation, 1—<30 ml (<20%), 2—30–59 ml (20–40%); 3—>60 ml (40–60%); and 4—>60%.*
of leaflet coaptation. Because in IMI the MV structure appears ostensibly normal, the lesion is ideal for valve repair.

In our early years of experience, modified Kay–Wooler annuloplasty and a modified Paneth annuloplasty were used in patients with ischaemic annular dysfunction without structural abnormality of the leaflets and the subvalvar apparatus. The repair technique now applied since 1992 is a combination of posterior annulus shortening of a modified Paneth type (Fig. 2A) to a degree which reduces the mitral orifice to between 22 and 24 mm in diameter, resulting in an MV orifice of between 3.5 and 4.5 cm². This

Table 2: Demographic profile of 179 patients in whom the no-ring modified Paneth–Hetzer posterior annulus shortening technique technique was employed

<table>
<thead>
<tr>
<th>Aetiology of MV incompetence</th>
<th>n</th>
<th>Mean (median, range) age, years</th>
<th>NYHA functional class</th>
<th>Severity of mitral insufficiency&lt;sup&gt;a&lt;/sup&gt; (mean grade)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (annular dilatation)</td>
<td>81</td>
<td>43.9 ± 27.75 (47.5, 22.5–80.20)</td>
<td>III</td>
<td>3.5</td>
</tr>
<tr>
<td>B (IMI)</td>
<td>75</td>
<td>64.56 ± 10.37 (66.0, 35.0–86.1)</td>
<td>III–IV</td>
<td>3.8</td>
</tr>
<tr>
<td>C (asymmetric valve repair)</td>
<td>23</td>
<td>42.87 ± 22.68 (44.0, 18.7–81.0)</td>
<td>III</td>
<td>3.3</td>
</tr>
</tbody>
</table>

<sup>a</sup>Echocardiographic evaluation of mitral insufficiency based on regurgitant volume (regurgitant fraction, %): 0–no regurgitation; 1—<30 ml (<20%); 2—30–59 ml (20–40%); 3—<60 ml (40–60%); and 4—(>60%).
residual size is sufficient for adults and ensures an adequately broad leaflet coaptation area. The posterior annulus shortening suture is reinforced by adding a strip of untreated autologous pericardium, which has been shown to obviate redilatation (Fig. 2B and C).

It has been observed that this technique was successful in IMI through augmentation of the posterior annulus by the pericardial strip tissue. The area that the posterior leaflet offers to the anterior leaflet for coaptation during valve closure is enhanced by the pericardial tissue, making valve closure possible even in cases of advanced leaflet restriction.

Mitral regurgitation from IMI may also be caused by chordal rupture, which leads to leaflet prolapse (Fig. 1A). This is typical for IMI, leading to asymmetrical valve closure along the postero medial commissure. In this series, it was seen in patients with a large area of infarcted posterior wall and, interestingly, in those with 90% or complete occlusion of the right coronary artery. It is always accompanied by alterations in annular geometry, which leads to a reduction of surface area for leaflet coaptation. For this condition, a modified Gerbode–Hetzer posterior leaflet plication (Fig. 1B–E) is best suited.

No-ring MV repair in children

No-ring modified Gerbode–Hetzer posterior leaflet plication technique. The no-ring modified Gerbode–Hetzer posterior leaflet plication technique was used in 62 patients with leaflet prolapse (n = 40), chordal rupture (n = 10) and papillary muscle elongation (n = 12).

Modified Kay–Wooler annuloplasty with Hetzer’s modifications. Modified Kay–Wooler annuloplasty with Hetzer’s modifications was most frequently used in newborns and small infants with annular dilatation (n = 30). We modified [21, 22] this technique by shortening the segments of the posterior annulus next to both trigones by polypropylene sutures pledgeted with untreated autologous pericardium.

No-ring modified Paneth–Hetzer posterior annulus shortening technique. The no-ring modified Paneth–Hetzer posterior annulus shortening technique was used in 48 children and adolescents with severely dilated annulus of any origin.

Table 3 summarizes the MV lesions of 140 children with MV incompetence in whom the modified Gerbode–Hetzer posterior leaflet plication, modified Kay–Wooler annuloplasty with Hetzer’s modification and modified Paneth–Hetzer posterior annulus shortening techniques were employed.

Evaluation of the adequacy of repair

It is obligatory to assess the valve function after MV repair, before atrial closure and separation from cardiopulmonary bypass, which is done by transvalvular saline injection. Any remaining areas contributing to significant incompetence are attended to before atrial closure. Once de-airing has been completed and extracorporeal circulation is discontinued, the repair result is evaluated with intraoperative transoesophageal echocardiography to demonstrate adequate mitral orifice, lack of residual incompetence, absence of myocardial ischaemia due to coronary kinking and absence of the SAM phenomenon. Immediate and prompt correction must be made if the repair is shown to be unsatisfactory. Regardless of the underlying pathology and techniques used, no patient should be discharged from the operating room with more than minimal MI.
Follow-up. The follow-up data were provided by the Department of Clinical Studies, Deutsches Herzzentrum Berlin, and written correspondence from the referring physicians and/or telephone interviews with patients or families. All patients have complete follow-up.

Statistical analysis. All data were analysed with the SPSS 16.0 (SPSS, Inc., Chicago IL, USA) software programme. Data are expressed as absolute and percentage frequency values and continuous data as mean ± standard deviation, as appropriate. Variables such as New York Heart Association (NYHA) functional class, left ventricular function and severity of mitral insufficiency were analysed with the Pearson χ² tests, the two-tailed displayed proportions and odds ratios. Differences among groups were analysed using the Mann-Whitney U-test, log rank (Mantel-Cox) and generalized Wilcoxon. A P-value of ≤0.05 is considered significant. Freedom from reoperation and survival rates were analysed according to Kaplan-Meier estimates with 95% confidence interval (CI).

RESULTS

Between May 1986 and December 2012, we have performed a total of 29 503 valve operations, of which 9742 were MV surgeries (4570 replacements and 5172 repairs). The cases in this report are single surgeon’s series wherein the no-ring MV repair techniques were employed within this time period.

Outcome of no-ring modified Gerbode–Hetzer posterior leaflet plication technique

There was no hospital morbidity or mortality. Overall mean duration of the follow-up is 17.24 ± 0.53 (median 19.63, range 1.3–24) years.

A. Mean duration of the follow-up in this group is 8.39 ± 2.2 (median 8.53, range 1–24) years. In 161 patients with ruptured chordae from degenerative MV disease, prolapsed leaflet in Barlows’s disease and floppy valve in Marfan’s syndrome, freedoms from reoperation are 96.3 ± 1.5, 93.6 ± 2.0, 88.9 ± 2.6, 86.8 ± 3.0 and 83.5 ± 4.3% at 30 days, 1, 5, 10 and 20 years, respectively (Fig. 3A: line A).

B. Mean duration of the follow-up in this group is 10.54 ± 1.9 (median 10.95, range 1–19) years. In 22 patients with ruptured chordae from active endocarditis, freedom from reoperation is 95.5 ± 4.4, 81.1 ± 8.5 and 44.4 ± 10.1% at the 1-, 5- and 19-year follow-up, respectively (Fig. 3A: line B). Freedoms from reinfection for isolated native MV endocarditis were 100% at 30 days, 97.5 ± 1.7% at 1 year and 91.9 ± 3.6% at 5, 10 and 15 years.

C. Mean duration of the follow-up in this group is 8.63 ± 1.5 (median 8.65, range 3–15) years. In 10 patients with ruptured chordae from ischaemic disease, freedoms from reoperation are 100%, 93.5 ± 4.6, and 78.3 ± 2.2% at 30 days, 1 and 15 years (Fig. 3A: line C).

D. Chordal rupture as a sequel of blunt or deceleration trauma was seen in a 15-year old male. He is now 26 years old, without any MI.

In all, severity of MI decreased significantly from grade 3.27 ± 0.6 (median 3, range 3–4) to absent or mild regurgitation of grade 0.72 ± 0.6 (median 1, range 0–2) at the long-term follow-up (P < 0.001). Complete absence of MI was found in 96% of patients. There was no worsening of MI, except in 3 patients; 1 of them had suture dehiscence, who then underwent repeat repair, and two underwent MV replacement. The increase in severity of MI in these 3 patients, however, occurred in the late postoperative period (>30 days postoperatively).

At the latest follow-up, overall freedom from reoperation and cumulative survival rate are 55.4 ± 4.7% (Fig. 3B) and 44.7 ± 5.4% (Fig. 3D), respectively.

Outcome of no-ring modified Paneth–Hetzer posterior annulus shortening technique

Overall mean duration of the follow-up is 11.98 ± 1.14 (median 14.47, range 4.8–24.09) years.

A. Mean duration of the follow-up in this group is 12.27 ± 1.4 (median 9.45, range 1.5–19) years. In 83 patients with annular dilation seen in idiopathic and secondary cardiomyopathy, with impaired left ventricular function, freedoms from reoperation were employed.

Table 3: Indications of no-ring modified Gerbode–Hetzer posterior leaflet plication and modified Paneth–Hetzer posterior annulus shortening techniques in 140 children

<table>
<thead>
<tr>
<th></th>
<th>&lt;3 months (n = 17)</th>
<th>3 months to 2 years (n = 48)</th>
<th>&gt;2–18 years (n = 75)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severity of mitral insufficiency (mean grade)*</td>
<td>2.6</td>
<td>3.2</td>
<td>3.5</td>
</tr>
<tr>
<td>NYHA functional class</td>
<td>II–III</td>
<td>II–III</td>
<td>III</td>
</tr>
<tr>
<td>Modified Kay–Wooler annuloplasty with Hetzer’s modification</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annular dilatation</td>
<td>13</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>Modified Paneth–Hetzer posterior annulus shortening</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Annular dilatation</td>
<td>10</td>
<td>38</td>
<td></td>
</tr>
<tr>
<td>Modified Gerbode–Hetzer posterior leaflet plication</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Leaflet prolapse</td>
<td>3</td>
<td>14</td>
<td>23</td>
</tr>
<tr>
<td>Chordal rupture</td>
<td>2</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>Papillary muscle elongation</td>
<td>1</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

*Echocardiographic evaluation of mitral insufficiency based on regurgitant volume (regurgitant fraction, %): 0—no regurgitation; 1—<30 ml (<20%); 2—>30–59 ml (20–40%); 3—>60 ml (40–60%); and 4—(>60%).
were 95.0 ± 2.4, 90.0 ± 3.7, 88.6 ± 6.3 and 85.9 ± 13.9%, at 30 days, 1, 5 and 19 years, respectively (Fig.4A: line A)).

Severity of MI decreased significantly from grade 3.7 ± 0.08 to absent or mild regurgitation of grade 0.52 ± 0.3 at the long-term follow-up (P < 0.001). Significant postoperative improvement in MI severity was found in all patients. In 2 patients, there was a recurrence of MI because of suture dehiscence; they underwent repeat repair by resuturing of the dehiscent area. Both were performed 2 years postoperatively.

B. Mean duration of the follow-up in this group is 7.62 ± 0.66 (median 8.53, range 3.6–18.9) years. In 75 patients with IMI who had either previous or concomitant coronary revascularization, 26 (34.6%) (mean age 60.6 ± 11.9, median 61.07, range 35–82.8 years) underwent isolated MV repair. These patients had mild MI upon discharge after a previous coronary revascularization that progressed in severity over time.

Freedoms from reoperation are 97.3 ± 1.9, 83.8 ± 2.7 and 78.4 ± 5.6% at 1, 5 and 18 years (Fig. 4A: line B), respectively, in patients who had had a previous coronary revascularization and then a subsequent modified Paneth–Hetzer posterior annulus shortening technique. Reoperation consisted of MV replacement in 2 patients at 1 and 5 years after the initial repair, and 1 was reoperated due to annular suture dehiscence 3 years later. Two reoperations unrelated to the MV repair consisted of mechanical circulatory assist device implantation and another 2 patients underwent orthotopic heart transplantation a year later. These are the patients who preoperatively had ejection fraction of 15%, NYHA functional class IV and MI of grade 4, with associated triple-vessel disease and left ventricular aneurysm.

Freedoms from reoperation in patients who underwent isolated MV repair for ischaemic MI are 96.2 ± 3.8, 92.0 ± 5.5 and

Figure 3: Kaplan–Meier curve showing freedom from reoperation after modified Gerbode–Hetzer plication plasty. (A) Based on indications (ruptured chordae from A = degenerative disease, B = active IE, C = IMI). (B) Overall freedom from reoperation with patients at risk at 95% CI. (C) Cumulative survival based on indications. (D) Overall survival rate.
80.7 ± 8.9% at 30 days, 1 and 5 years, respectively. Overall freedom from reoperation among patients at risk is 62.9 ± 6.3% (Fig. 4A and B).

Noteworthy is that with the previously described concept of leaflet augmentation for IMI, 66.7 and 62.2% of patients with preoperative NYHA functional classes III and IV, respectively, improved to class I on the latest follow-up. Likewise, ejection fraction and severity of MI have significantly improved.

C. Mean duration of the follow-up in this group is 15.83 ± 2.4 (median 8.53, range 4–24.09) years. In 22 cases of asymmetric valve repair performed, such as closure of a leaflet perforation and unilateral commissuroplasty, freedom from reoperation was 100% at 1 year sustained through 24 years (Fig. 4: line C).

Severity of MI decreased significantly from grade 3.3 ± 0.7 to absent or mild regurgitation of grade 0.42 ± 0.3 (P < 0.001). Absent to trivial MI was found in 93% of patients at the latest follow-up.

Overall freedom from reoperation and cumulative survival rate in this population who underwent the modified Paneth–Hetzer posterior annulus shortening technique are 82.95 ± 4.1% (Fig. 4B) and 63.4 ± 8.5% (Fig. 4D), respectively.

Outcome of MV repair in children

While it is extremely challenging to repair MV in infants and children, primarily because of their size, immature and fragile leaflet tissues particularly in infants, and associated congenital cardiac abnormalities, this population gains maximally from repair.

In a series of 140 children (mean age 7.5 ± 5.9, median 5 years, range 20 days to 17.8 years), with MV insufficiency, of whom 126 had congenital MV lesions and 34 acquired lesions, repair results in this group are highly satisfactory. At a mean follow-up of
11.2 ± 7.2 years (median 8.2, range 1.2–23.7 years), freedom from repeat reconstruction and replacement after modified Kay-Wooler annuloplasty and modified Paneth-Hetzer posterior annulus shortening technique (n = 62) stratified based on age groups were as follows: age group <3 months: 82.1 ± 2.3 and 78.9 ± 3.5% at 1 and 5 years onwards, respectively (Fig. 5A: line A); age group 3 months to 2 years: 80.2 ± 1.4 and 71.6 ± 5.3% at 1 and 5 years onwards, respectively (Fig. 5A: line B); and for age group 2–18 years: 98.2 ± 1.3, 92.5 ± 15, and 85.1 ± 4.4% at 1, 5, and 20 years, respectively (Fig. 5A: line C). Overall survival is 83% (Fig. 5B).

Likewise, freedom from repeat reconstruction and replacement after modified Gerbode-Hetzer posterior leaflet plication technique (n = 78) stratified based on age groups were: age group <3 months: 88.1 ± 1.3, 80.9 ± 3.5 and 61.4 ± 2.7% at 1, 5, and 10 years onwards, respectively (Fig. 5C: line A); age group 3 months to 2 years: 97.2 ± 3.4, 90.5 ± 2.4 and 78.7 ± 3.7% (Fig. 5C: line B); and for age group 2–18 years: 99.2 ± 0.3, 98.5 ± 1.5, and 97.1 ± 2.4% (Fig. 5C: line C), all at 1, 5, and 10 years onwards, respectively. Overall survival is 92% (Fig. 5D).

These results are encouraging, particularly in this population where 46.4% of patients were under 2 years of age. It is also important to note that at 23.7 years, 94% were in Ross/NYHA functional classes I and II with normal growth and development. In our previous publication [11], it was shown that the pericardial strip, aside from preventing further posterior annular dilatation, likewise allows the anterior MV leaflet and its annulus to grow in relation to body size over time, while preserving the flexible properties of the MV orifice.

In all reoperations, the pericardial strip was found perfectly attached to the annulus and covered by a layer of fibrous tissues without calcification, thrombosis or retraction. It appeared completely endothelialized and somewhat indistinguishable from the native annular tissues.

**DISCUSSION**

The ideal type of annuloplasty in MV repair has remained controversial. For many years, Carpentier’s ring was considered the gold standard for annuloplasty. Increased knowledge of the anatomy and physiology of the MV annulus, and increased experience with the evolution of various repair techniques have raised doubts about this and other annular devices. Insertion of a ring prosthesis deforms the natural saddles of the MV annulus and may lead to left ventricular outflow obstruction [10]. Fixation of the annulus with a rigid ring also has a negative effect on systolic left ventricular function [23].

Analysis of our >20-year institutional experience shows that MV repair without prosthetic rings using suture repair techniques and posterior annuloplasty stabilization with untreated autologous
pericardium are highly reliable in restoring MV competence and achieving highly satisfactory long-term results when employed in ruptured chordae and/or posterior leaflet prolapse from degenerative disease, active endocarditis and IMI and in traumatic chordal rupture. Likewise, satisfactory long-term outcome is seen in all cases of annular dilatation. There are only very few and sporadic published reports on posterior annuloplasty using untreated autologous pericardium. Annular remodelling with pericardial reinforcement was used by several groups for mitral regurgitation due to degenerative disease wherein an autologous pericardial tube was placed along the posterior annulus just beyond the anatomical commissures in 55 patients with good, albeit very short-term, results [24]. The same group reported posterior annuloplasty using autologous pericardium treated with 0.625% glutaraldehyde for MI caused by myxomatous degenerative disease in 113 patients, with freedom from reoperation of 97 and 89.7% at 1 and 5 years, respectively. Another group [25] reported using 0.625% glutaraldehyde-treated autologous pericardial strips for posterior mitral ‘overreductive’ annuloplasty in 31 patients—in 19 with dilated cardiomyopathy and in 12 with postischaemic MI. However, they reported only the optimal length (1 cm wide, 5 cm long) of pericardial strip, and advocated this fixed pericardial length to achieve predictable and haemodynamically satisfying results based on 4.6 ± 3.5 months follow-up.

We are firmly convinced that annular stabilization is the crucial element of the correction; it not only reduces the annular size but also distributes the tension of the suture on the posterior annulus and prevents further or late annular dilatation. Our technique of posterior annular stabilization with an untreated autologous pericardial strip avoids the negative effects of rigid fixation of the annulus.

The rationale of posterior annulus shortening in IMI is to facilitate the return of the zone of coaptation, particularly along the posteromedial commissure, thus correcting the insufficiency. Use of an autologous pericardial strip to stabilize the shortened posterior annulus not only reinforces the repair but thus helps in maintaining the MV geometry. This technical strategy offers an incremental benefit over standard annuloplasty or implantation of prosthetic devices, since the shortening of the posterior annulus and the geometrical restoration by posterior leaflet augmentation provide adequate coaptation surface and thus help improve the LV function.

In active IE, this biological repair is aimed at immediate restoration of mitral competence, after resection of all infected materials. A variety of repair techniques we have previously described are necessary to achieve this; otherwise, when repair is impossible due to extensive destruction of leaflets and subvalvar apparatus, we implanted a xenograft valve with pericardial tissue as sewing ring [21, 22]. With the techniques previously described, the incidence of reinfection and reoperation has remained low; however, reoperation after healing of endocarditis must be anticipated.

In our institution, MV repair is the surgical technique of choice for any kind of MV lesions in childhood. It is believed that repair allows continuous somatic and valve growth [11], delays or eliminates the need for future valve replacement with lifetime anticoagulation, and obviates the known complications of valve replacement that frequently necessitate subsequent reoperation to implant a larger prosthesis. It must be assumed that the majority of, if not all, valves repaired during childhood will have to be replaced eventually at some time in life. The concept of repair in childhood is primarily aimed at growth of the patient to an age when, if necessary, an adult-size prosthesis can be implanted.

In this population, annular dilatation and prolapsed leaflet were frequently present. The authors tried to avoid the placement of a rigid ring prosthesis for stabilization or to correct annulus abnormalities because of concerns about anticoagulation, subsequent somatic growth problems and the risk of a rigid prosthesis causing distortion of the heart cavities and/or contributing to left ventricular outflow tract obstruction [10]. Several studies consider ring annuloplasty for MV incompetence obligatory in children over 2 years of age. This concept is supported by their experience of a 25% incidence rate of significant residual MI after repair without ring insertion. Other groups have demonstrated that other annuloplasty techniques can be employed successfully in children and that prosthetic rings are not indispensable for achieving favourable results.

Systolic anterior motion

Every surgeon who repairs the MV must give particular attention to the SAM phenomenon. It is very important to evaluate the presence of SAM intraoperatively, while the patient is still on cardiopulmonary bypass. Failure to recognize its presence may be disastrous, if not fatal. This phenomenon is virtually absent in our series, since we have learned to recognize its presence during the assessment of the adequacy of our repair. An inward folding of the anterior leaflet during transvalvular saline instillation is a definite indication for SAM, which is due to overreduction of the posterior annulus [20].

CONCLUSION

This institutional series of 513 patients, from 20 days to 86 years old, is by far the largest series of suture repair techniques with the longest follow-up (≥20 years) ever reported. No-ring MV repair (modified Gerbode-Hetzer posterior leaflet plication and modified Paneth-Hetzer posterior annulus shortening technique) with posterior annulus stabilization with an untreated autologous pericardial strip in annular dilatation, chordal rupture, ischaemic MI and IE, and in various MV lesions in children offers excellent long-term functional results with a highly satisfactory 20-year freedom from reoperation. In our long-term experience with these techniques, there has never been an issue of left ventricular outflow tract obstruction or impairment of LV function. Based on our results, we advocate no ring at all for MV repair, particularly in the above-mentioned indications.

ACKNOWLEDGEMENTS

We thank Christine Detschades for providing us updated data, Julia Stein for statistical analysis, Astrid Benhennour for providing us with literature and Anne Gale for editorial help. We highly appreciate the assistance of Helge Haselbach and Carla Weber for the graphical illustrations.

Conflict of interest: none declared.
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