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Mitral Valve Mechanics Following Posterior Leaflet Patch Augmentation

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Abstract

Background and aim of the study—Attention towards the optimization of mitral valve repair methods is increasing. Patch augmentation is one strategy used to treat functional ischemic mitral regurgitation (FIMR). The study aim was to investigate the force balance changes in specific chordae tendineae emanating from the posterior papillary muscle in a FIMR-simulated valve, following posterior leaflet patch augmentation.

Methods—Mitral valves were obtained from 12 pigs (body weight 80 kg). An in vitro test set-up simulating the left ventricle was used to hold the valves. The left ventricular pressure was regulated with water to simulate different static pressures during valve closure. A standardized oval pericardial patch (17 × 29 mm) was introduced into the posterior leaflet from mid P2 to the end of the P3 scallop. Dedicated miniature transducers were used to record the forces exerted on the chordae tendineae. Data were acquired before and after 12 mm posterior and 5 mm apical posterior papillary muscle displacement to simulate the effect from one of the main contributors of FIMR, before and after patch augmentation.

Results—The effect of displacing the posterior papillary muscle induced tethering on the intermediate chordae tendineae to the posterior leaflet, and resulted in a 39.8% force increase ($p = 0.014$). Posterior leaflet patch augmentation of the FIMR valve induced a 31.1% force decrease ($p = 0.007$). There was no difference in force between the healthy and the repaired valve simulations ($p = 0.773$).

Conclusion—Posterior leaflet patch augmentation significantly reduced the forces exerted on the intermediate chordae tendineae from the posterior papillary muscle following FIMR simulation. As changes in chordal tension lead to a redistribution of the total stress exerted on the valve, patch augmentation may have an adverse long-term influence on mitral valve function and remodeling.

Functional mitral regurgitation is a complex valvular heart disease that commonly occurs in patients with left ventricular (LV) dysfunction in ischemic or dilated cardiomyopathy, with an anatomically normal leaflet and chordal morphology (1,2). When the heart is exposed to a posterior myocardial infarction, the left ventricle dilates, and this may result in papillary muscle displacement and annulus dilation and flattening (3–5). Ventricular dilatation causes a displacement of the papillary muscle tips away from the annulus in the apical, lateral, and posterior directions, thereby inflicting an increased tethering force on the chordae tendineae (2,6,7). Consequentially, the motion of the posterior leaflet is restricted in the medial half of P2 and the P3 scallops (4,6,8), which in turn results in an asymmetric coaptation, mitral valve incompetence, and regurgitation. The papillary muscle displacement with tethering of the anterior and posterior leaflets (Carpentier type IIIb) (9), along with the annular dilatation (Carpentier type I) (9), are considered to be the two major contributors to functional ischemic mitral regurgitation (FIMR) (1,6,9). Typically, FIMR is seen in between 11% and 59% of patients after myocardial infarction (1). Even with a mild degree of mitral regurgitation, these patients have a mortality risk that is two- to fourfold higher than in those patients without mitral regurgitation within 2 to 16 days after infarction (10).

Valve repair is generally preferred as opposed to replacement, as the former approach is associated with increased survival and a lower risk of thromboembolic complications (11). Repair also facilitates the preservation of LV function and eliminates the need for long-term anticoagulation therapy (11,12). Several repair methods for FIMR exist. One particular technique that has been transferred from the repair of other mitral valve diseases is leaflet augmentation, using a pericardial patch (11,12). Patch augmentation increases the membrane area covering the annular orifice, thereby bringing the leaflets together while reforming the coaptation zone and ensuring valve competence. Despite several surgical techniques being available, the problem stated widely among such studies is that the long-term outcomes of repair methods have not been fully investigated and hence this subject remains controversial (3,5,11,13,14).

The aim of the present study was to investigate the possible force changes in the strut and intermediate chordae tendineae emanating from the posterior papillary muscle to the anterior and posterior leaflets by using posterior leaflet augmentation as a repair method of FIMR. In this study, FIMR was simulated by displacing only the posterior papillary muscle (Carpentier type IIIb), thereby isolating this effect from the confounding annular dilatation and flattening, as seen in FIMR. This should provide insight into explaining how a patch repair can affect the force balance and the long-term outcome of this procedure.

Materials and methods

Left heart simulator

A static LV pressure was obtained in an altered version of the left heart simulator developed by Ostli et al. (15). The simulator (Fig. 1) consists of a ventricular chamber in which pressure can be induced by water, and annulus- and papillary muscle holders in which a mitral valve can be mounted. The system provides the possibility of adjusting the height of the annulus holder as well as the posterior papillary muscle, thereby providing full control of the valve positioning. The annulus holder was produced in a D-shape with saddle shape with a 15% annular height-to-commissural ratio to simulate the healthy valve in systole (16). The distance from commissure to commissure was 5 cm, and the anterior-posterior distance was 4 cm.

Valve preparation

The hearts of 12 pigs (body weight 80 kg) were retrieved from a local abattoir and taken to the laboratory. The mitral valves were excised from each heart and examined for abnormalities that would make them inadequate for use in the left heart simulator. The valves were fixed in a formaldehyde (4%) bath for 65 min to ease handling of the tissue and to prolong the period of possible use. The valves were stored in 0.9% saline solution in separate containers at 3–5°C, and used within one week of their excision.

Valve implantation and patch augmentation

The mitral valve was secured circumferentially at four points onto the annulus holder using 4-0 polypropylene sutures, with continuous interlocking stitches between the fastening points. An incision was made 2 mm from the annulus, and extended circumferentially from the middle of P2 and through the entirety of the P3 scallop, as demonstrated previously by de Varennes et al. (3) and others (8). A standardized oval-shaped porcine formaldehyde-treated pericardial patch (17 × 29 mm) was then implanted in the incision by securing it first at two points, and then suturing continuously between the points using a 5-0 polypropylene suture. Taking into account that approximately 2 mm would be used for the suture itself, the resulting enlarged area was 13 × 25 mm. A reefing stitch with retraction sutures was placed through the annulus and patch, in order to allow the patch to be furled into the annulus when simulating a healthy valve, and unfurled to augment the leaflet as shown by Langer et al. (17) (Fig. 2). Dacron vascular graft material was sutured onto the papillary muscle to allow fixation of the papillary muscle holder.

Data acquisition: Force measurements

Dedicated miniature strain gauge transducers (dimensions of 2 × 2 × 5 mm) (18), were used to measure the forces on specific chordae tendineae. The transducers were calibrated before and after measurements to assure correct functionality. The mean value of the two calibration constants was used. The transducers were sutured onto the upper part of the chordae tendineae near the leaflet before the first branching of the chordae. When branching of the chordae prohibited insertion at the very top, the transducer was inserted directly below the branching. After insertion of the transducer, the chordae was cut in between the suture points to ensure complete measurement of the forces (Fig. 3). Transducers were sutured onto the intermediate and strut chordae tendineae emanating from the posterior papillary muscle attached onto the posterior and anterior leaflets, respectively. These chords were chosen as they are thicker, more load-bearing, and are responsible for maintaining LV geometry (19,20).

The annulus holder was mounted in the left heart simulator and water supply connected to the chamber to create a LV pressure ranging from 0 to 150 mmHg. Measurements were obtained in the following simulations:

1. Healthy valve
2. Healthy valve with patch augmentation
3. FIMR
4. FIMR with patch augmentation.

The healthy valve simulation was defined as: Correct valve closure without visual retrograde flow, and correct papillary muscle positioning as seen clinically (6,21). The FIMR simulation was defined as: Posterior and apical displacement, 12 mm and 5 mm respectively, of the posterior papillary muscle, as shown by Jensen et al. (6) and others (2,7).

The angle of both papillary muscles and the position of the anterolateral papillary muscle were preserved, as in the healthy simulation.

The LV pressure was obtained using Millar catheters (Millar Instruments Inc., Houston, Texas, USA). Transducers were connected to a module rack (NI cDAQ 9172; National Instruments Corp., Austin, Texas, USA) with two input modules (NI 9215, NI 9237). Data were recorded with virtual instrumentation using an in-house-built data acquisition program (LabVIEW 8.6; National Instruments) with a sampling frequency of 1,613 Hz.

All simulations were performed without removing the valve from the system, thereby preserving the settings and reproducibility.

Statistical analysis

For all experimental set-up simulations, three separate measurements were made and their mean was calculated and used for the prospective statistical analysis. A three-way ANOVA was performed using the following parameters: (i) valve; (ii) before/after patch; and (iii) before/after papillary muscle displacement. A linear regression analysis was used to verify the linearity between the ventricular pressure and chordal tension. The statistical analysis was approved by a professional biostatistician, and the level of significance selected for all tests was defined at $p = 0.05$. When applicable, numerical results were presented as mean $\pm$ SD.

Results

For all measurements, specific results were presented at a LV pressure of 100 mmHg. Mean values are listed in Table I, while the effects of the different variables are shown in Table II (as percentages). The pressure and force measurements correlated linearly for the anterior and posterior measurements ($R^2 = 0.996$ and $R^2 = 0.990$, respectively); an example of the correlation is shown in Figure 4.

Posterior leaflet

The effect of both displacing the posterior papillary muscle and augmenting the posterior leaflet has a significant effect on the force in the intermediate chordae tendineae emanating from the posterior papillary muscle attached to the posterior leaflet. When the healthy simulation was compared with the posterior papillary muscle-displaced simulation, a force increase of 39.8% ($p = 0.014$) was found in the intermediate chordae tendineae. When the patch augmentation was implemented, simulating a repair of the valve, the forces were decreased by 31.1% ($p = 0.007$). Furthermore, by comparing the healthy and repaired FIMR valve, no significant difference in force was found ($p = 0.773$).

Anterior leaflet

The FIMR simulation increased the tethering of the strut chordae emanating from the posterior papillary muscle by 64.3% ($p < 0.0001$). However, patch augmenting the posterior leaflet did not have a significant impact on the forces of the strut chordae, but rather showed a force that was decreased by 6.6% ($p = 0.165$). Furthermore, as the test set-up provided full visual access of the valve in both the ventricular and atrial views, it was possible to make visual assessments of the patch augmentation. The insertion points of the two chordae tendineae used for measurements are marked in Figure 5. As the patch augmentation was put into effect, the insertion point of the intermediate chordae tendineae was moved into the coaptation zone, whereas the insertion point of the anterior leaflet strut chordae remained in position.

Discussion

Several different repair methods of the mitral valve have been investigated, some of which have been put into practical use, including the papillary muscle sling (22), papillary muscle relocation (13), and selective chordal cutting (7). The most commonly used surgical procedure to correct FIMR is coronary artery bypass grafting combined with ring annuloplasty, or either method alone (5,7,10). The results of recent studies (3,17) have shown that 30% of patients had a recurrence of significant ischemic mitral regurgitation during a six-month period after surgery, and that this was increased to 60% after three to five years (5). The reason for this might be that annuloplasty ring (downsizing and true-sizing) primarily addresses annular dilatation and not papillary muscle displacement (5,13,17).

Another reservation towards some of these repair methods is their comprehensiveness, as they often consist of several procedures that prolong the operating time and place the patient at greater risk (5,17). Furthermore an easily reproducible procedure is preferable at those institutions which do not have many FIMR patients and therefore have a limited experience in valve repair.

In 1966, Sauvage and Wood (23) were the first to use a pericardial patch as part of mitral valve repair in patients. Since then, leaflet augmentation has been used in different mitral valve diseases, most recently in conjunction with an annuloplasty ring to repair FIMR (3–5,8,17). Whilst several of these studies have reported good short-term results, long-term data and more specific knowledge of the complexity of the mitral valve are required in order to thoroughly evaluate the efficiency of this procedure.

The choice in the present study to simulate displacement of the papillary muscle posteriorly and apically was made on the basis of different studies examining the displacement of LV geometry. The posterior displacement was based on the results of Jensen et al. (6), with a more subtle apical displacement being chosen as different measurements have been reported elsewhere (2,7,24).

The force decrease found in the present study was similar to that reported by Granier et al. (25), who showed that the anterior leaflet was augmented in an effort to investigate the forces in a prolapsed valve, though employing a technique comparable to that used in the present study. When augmenting the anterior leaflet, the forces exerted on the secondary chordae tendineae to the leaflet showed a mean decrease of 36.1% - a force reduction which was within the decrease interval found in the present study. This indicated that the effect of augmenting the posterior leaflet would be the same as for augmenting the anterior leaflet on the secondary chordae tendineae attached to the posterior and anterior leaflets, respectively. The force measurements made in the present study were comparable with those of other in vitro studies, such as that conducted by Jimenez et al. (24). The statistical analysis confirmed that the force measurements between the valves would vary considerably. This variance means that the individuality of valves has a high significance on the force measurements ($p < 0.001$), as seen in other studies (24,25). In other words, the effect of displacing the papillary muscle and augmenting the posterior leaflet differs from valve to valve. Furthermore, the results obtained in the present study indicated that, although the tethering force is of high concern in the posterior leaflet, it is also very significant in the chordae on the anterior leaflet.

Findings from Kuwahara et al. (26) state that the use of an annuloplasty ring in patients with FIMR provides satisfactory results, initially by reducing mitral regurgitation, although patients who developed recurrent regurgitation had an increased posterior leaflet tethering. The anterior leaflet tethering remained improved and stable in these patients after

annuloplasty, leading to an asymmetric tethering of the anterior and posterior leaflets (26). Others have stated that the use of an annuloplasty ring moves the posterior annulus anteriorly because the anterior side of the annulus is fixed at the aortic root; this gives the valve a more monocusp function, as the posterior leaflet remains tethered (7,16). The present study demonstrated a reduction in tethering of 31.1% of the posterior leaflet as a result of patch augmentation. On the basis of the above-mentioned data, and the results of the present study, it might be speculated that the use of an annuloplasty ring in conjunction with posterior leaflet augmentation represents a solution to address both annular dilatation and posterior and anterior tethering. Dobre and coworkers (4) have shown that patch augmentation with ring annuloplasty reduces mitral regurgitation, as tethering of the posterior leaflet is one of the primary causes of the regurgitation. This point was also suggested by Rendón et al. (8), where the annuloplasty ring was used to maintain the annulus shape, thereby preventing further dilation. Furthermore, these authors believed that this repair technique would be reproducible and usable in most FIMR patients with restricted leaflet motion.

The reason for the force decrease found when patch augmenting the posterior leaflet is thought to be a displacement of the insertion point of the chordae, as shown in Figure 5; the theory of this is shown schematically in Figure 6. First, the distance between the insertion point and the posterior part of the annulus is increased. If the leaflet is viewed as a lever arm held in position by the chordae insertion point and the annulus as its hinge point, the laws of physics state (27) that an increase in the distance between the hinge and the insertion point will decrease the force exerted on the chordae. Second, the insertion point is observed to move into the coaptation zone, and is therefore thought to move closer to the posterior papillary muscle, thus decreasing the tension of the chord. As it is moved into the coaptation zone, the pressure conditions are significantly altered. There is a transmitral tension induced by the pressure difference between the ventricle and the atrium which affects the intermediate chordae tendineae when its insertion point is located on the belly of the leaflet towards the atrium. However, in the coaptation zone the pressure is the same on both sides, which means that the pressure applied in the anterior region of the coaptation zone (Fig. 6, horizontal arrow pointing right) is neutralized by the pressure applied on the posterior side (Fig. 6, horizontal arrow pointing left). It is believed that these three theories are the main reasons for the force reduction found in the present study.

The study results however raise the question of, ‘Where do the forces go?’, and ‘Why is this important?’. A free body diagram of the mitral valve may solve the LV ‘tug of war’, which dictates that when the force is decreased in one part of the valve, it must be increased in another part to meet static equilibrium. The results of the present study have demonstrated that tethering the force is relieved by 31.1% by the augmentation, but are the forces transferred to other parts of the valve or the myocardium, and what consequences does this bring? The theory of the lever arm, as described previously, would for instance indicate a transfer of the reduced force from the chordae to the annulus. This puzzle may be resolved by simulating patch augmentation through the finite element method, among others.

Study limitations

Kunzelman et al. (28) described the porcine mitral valve as being a suitable substitute when there was a need to explore and comprehend the structure of the human mitral valve. Furthermore, Jimenez et al. (24) stated that there is no significant difference in the chordal force between human and porcine valves of similar size. Anatomic differences were observed on the valves, however, for example in chordal thickness, branching of the chordae, and leaflet size. This variability comes into effect in the measurements which were observed to vary from valve to valve, as seen in other in vitro studies (24). The normal positioning of the valve was of a more qualitative than quantitative nature. Variation in

defining normal papillary muscle position may have resulted in a variance of the force change induced by the patch augmentation. Each mitral valve is incorporated as a variable in the statistical analysis to take the varying measurements into account.

In vitro studies often hold many advantages, including accurate adjustments, with the possibility of measuring mechanistic factors independently, in a controlled manner. However, it also simplifies a very complex multifactorial system that removes many factors from the equation. The limitations that are introduced are applicable in all scenarios before and after measurements; possible errors introduced can therefore to some extent be disregarded. These limitations are:

- Inserting the valve into a rigid three-dimensional (3D) print removes all the flexibility of the annulus. However, it is not necessary to achieve flexibility as the measurements are taken momentarily in systole, compensated by the saddle-shaped 3D print. Similarly, the contraction ability of the papillary muscles is made static.
- The annulus is often flat in FIMR, but this could not be simulated as it was necessary to simulate all four scenarios without tampering with the valve by removing it from the system.
- Formaldehyde fixation introduces a change in the biomechanical qualities of the valve (29).
- Anatomical variations of the chordae, namely branching near the leaflet, prohibited placing the transducer at the exact same point on all valves. However, this was not a limitation per se as the statistical analysis was made for each valve before and after patch augmentation.

The absence of retrograde flow was assessed by visual inspection, but a more valuable way of determining this would have been by ultrasound, had the in vitro system allowed for such measurements. FIMR can result from a complex combination of papillary muscle displacement and annular size changes. In the present study the annulus changes were not considered, and this will be a topic of future investigations among the authors' group.

In conclusion, FIMR in vitro simulation induces an increase in the chordae force of 39.8% in the posterior intermediate chord, and 64.3% in the anterior strut chord. Valve repair of FIMR using posterior leaflet patch augmentation effectively reduces the force exerted on the intermediate chord to the posterior leaflet by 31.1%, but not on the strut chord to the anterior leaflet. Most importantly of all, when comparing the healthy and the repaired simulation, no significant difference was found ($p = 0.773$), which means that the forces exerted on the intermediate chordae of the repaired valve were the same as in the healthy state. These findings may prove useful when developing therapeutic interventions with regards to surgical approaches.

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Figure 1.
Schematic diagram of the left heart simulator.

Figure 2. Atrial views of the mitral valve following displacement of the posterior papillary muscle, with and without patch. P: Posterior; A: Anterior.

Figure 3.
Photograph showing the transducer inserted onto the strut chordae tendineae.

Pressure-Force correlation

Figure 4.
Pressure-force correlation of the chordae tendineae to the anterior and posterior leaflets.

Figure 5. Positions of the chordae tendineae insertion points on the posterior and anterior leaflets (denoted by asterisks).

Figure 6.

Cross-sectional schematic of the mitral valve before and after patch augmentation. 1, Posterior leaflet; 2, anterior leaflet; 3, coaptation zone; 4, intermediate chordae tendineae to posterior leaflet; 5, strut chordae to anterior leaflet; 6, posterior papillary muscle; 7, anterior papillary muscle; 8, patch implanted in the posterior leaflet; 9, intermediate chordae tendineae to posterior leaflet moved to the coaptation zone. The asterisks indicate the insertion points for the strut and intermediate chordae to the anterior and posterior leaflets, respectively.

Table I

Force measurements ^{*} of posterior intermediate and anterior strut chordae tendineae (at LV pressure of 100 mmHg).

Post papillary muscle location	Healthy		Displaced	
	No patch	Patch	No patch	Patch
Posterior	0.28 ± 0.16	0.25 ± 0.18	0.42 ± 0.29 [*]	0.29 ± 0.18
Anterior	0.64 ± 0.34	0.61 ± 0.31	1.06 ± 0.60 [*]	0.99 ± 0.57 [#]

All values are expressed as mean ± SD (in Newton; n = 12).

^{*} Statistically significant difference between 'healthy - no patch' and 'displaced - no patch' (p <0.05).

[#] Statistically significant difference between 'healthy - no patch' and 'displaced - patch' (p <0.05).

Table II

Effect of functional ischemic mitral regurgitation (FIMR) simulation and implementing patch augmentation on the chordal force of the intermediate chordae tendineae to the posterior leaflet and strut chordae to the anterior leaflet (at LV pressure of 100 mmHg).

Chordae	Force change (%)	CI (%)	p-value
Posterior			
Healthy vs. FIMR	+39.8	+7.5 to +81.7	0.014
FIMR vs. repaired	-31.1	-47.0 to -10.4	0.007
Healthy vs. repaired	-3.7	-25.9 to +25.2	0.773
Anterior			
Healthy vs. FIMR	+64.3	+49.0 to +81.1	<0.001
FIMR vs. repaired	-6.6	-15.3 to +3.0	0.165
Healthy vs. repaired	+53.4	+39.2 to +69.2	<0.001