

# Comprehensive left ventricular outflow tract management beyond septal reduction to relieve obstruction

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Lara Gharibeh<sup>1,2</sup> , Nicholas G Smedira<sup>3</sup>  and Juan B Grau<sup>1,4</sup>

## Abstract

The surgical management of patients with hypertrophic obstructive cardiomyopathy can be extremely challenging. Relieving the left ventricular outflow tract obstruction in these patients is often achieved by performing a septal myectomy. However, in many instances, septal reduction alone is not enough to relieve the obstruction. Interventions on the sub-valvular apparatus, including the anomalous chordae tendineae and the abnormal papillary muscles, are often required. In this review, we summarize the embryology and the pathophysiology of the different elements that may contribute to the left ventricular outflow tract obstruction in the setting of hypertrophic obstructive cardiomyopathy. In addition, we highlight the different surgical procedures that a surgeon may adopt to relieve the left ventricular outflow tract obstruction, beyond the septal myectomy.

## Keywords

Hypertrophic cardiomyopathy, left ventricular outflow tract obstruction, mitral valve, papillary muscles, chordae tendineae

## Introduction

Hypertrophic cardiomyopathy (HCM) is a cardiac pathology that results in an abnormal thickening of the interventricular septum (IVS), with cardiac myocytes showing a disorganized array on microscopic examination.<sup>1</sup> It is caused by autosomal dominant mutations in sarcomeric protein genes and can exhibit several phenotypic variations with different distribution of myocardial hypertrophy: asymmetric septal (classic HCM), apical, neutral, concentric, and reverse septal.<sup>2,3</sup> In most cases, HCM has been classified as a nonobstructive disease, given that many patients are asymptomatic and do not show any abnormal left ventricular outflow tract (LVOT) gradient under basal conditions.<sup>4</sup> LVOT obstruction (LVOTO), defined as a peak instantaneous gradient >30 mmHg, defines the obstructive form of this disease. When this obstruction occurs, the condition becomes known as hypertrophic obstructive cardiomyopathy (HOCM).<sup>4,5</sup>

For many decades, the LVOTO was thought to result mainly from a thick septum.<sup>6</sup> Imaging modalities such as Cine computed tomography (CT), dynamic echocardiography/stress echo, and magnetic resonance imaging (MRI) have allowed us to identify the involvement of other elements that contribute to the LVOTO in addition to the

septum.<sup>7</sup> This has been reflected in recent published studies where the pathophysiology of HOCM involving all these structural abnormalities was described in detail. This is very relevant in patients with thin septae (<17–18 mm) who may be considered for surgical intervention as they normally present with severe symptoms and significant LVOT gradients during dynamic conditions.<sup>8</sup> The anatomic elements that contribute to the LVOTO, deriving from an abnormal embryology, consist of the sub-valvular mitral valve (MV) apparatus including anterior papillary muscle (PM) displacement with abnormal insertion, aberrant chordal attachments to the anterior leaflet of the MV (AMVL), and abnormally elongated anterior and posterior leaflet of the MV.<sup>9,10</sup>

<sup>1</sup>Division of Cardiac Surgery, University of Ottawa Heart Institute, Canada

<sup>2</sup>Department of Biochemistry, Microbiology and Immunology, University of Ottawa, Canada

<sup>3</sup>Department of Thoracic/Cardiovascular Surgery, Cleveland Clinic Foundation, USA

<sup>4</sup>Division of Cardiothoracic Surgery, The Valley Hospital, New Jersey, USA

## Corresponding author:

Juan B Grau, Division of Cardiac Surgery, University of Ottawa Heart Institute, 40 Ruskin Street, Ottawa, ON, Canada, K1Y 4W7.

Email: grauju@valleyhealth.com

These structures can interact with the septum, causing systolic anterior motion (SAM) of the MV leading to LVOTO and mitral regurgitation (MR), the hallmarks of the condition. This LVOTO in HOCM patients can vary from one patient to another depending on which elements are involved. For example, some HOCM patients present with only a thickened septum.<sup>11</sup> Some others present with PM hypertrophy in addition to IVS and/or left ventricular (LV) hypertrophy.<sup>12</sup> Some others have abnormal length of the MV leaflet with the presence of SAM of the MV<sup>13,14</sup> (Figure 1).

Under static conditions, it can be difficult to ascertain the contribution of each of these elements to the obstruction. The LVOT gradient, at rest, has been shown to be an independent element of predicting disease progression and cardiovascular-related mortality.<sup>4,5</sup> In many patients, it is only under provocation that one can assess how these elements interact to generate the obstruction. Studies have revealed that the LVOT gradient can be triggered using exercise, pharmacological interventions, or provocative (Valsalva) maneuvers, to reproduce the gradient increase in symptomatic HCM patients with no obvious obstruction at rest.<sup>15</sup> Given the heterogeneity in the presentation of HOCM that exists among patients, their assessment and management of their symptoms remain to be a big challenge for cardiologists and surgeons. It is only through a deeper understanding of the different abnormal embryonic elements involved in LVOTO that one can determine the best approach for therapy (Figure 1).

Embryology and pathophysiology of the septum and the MV apparatus in the HOCM setting

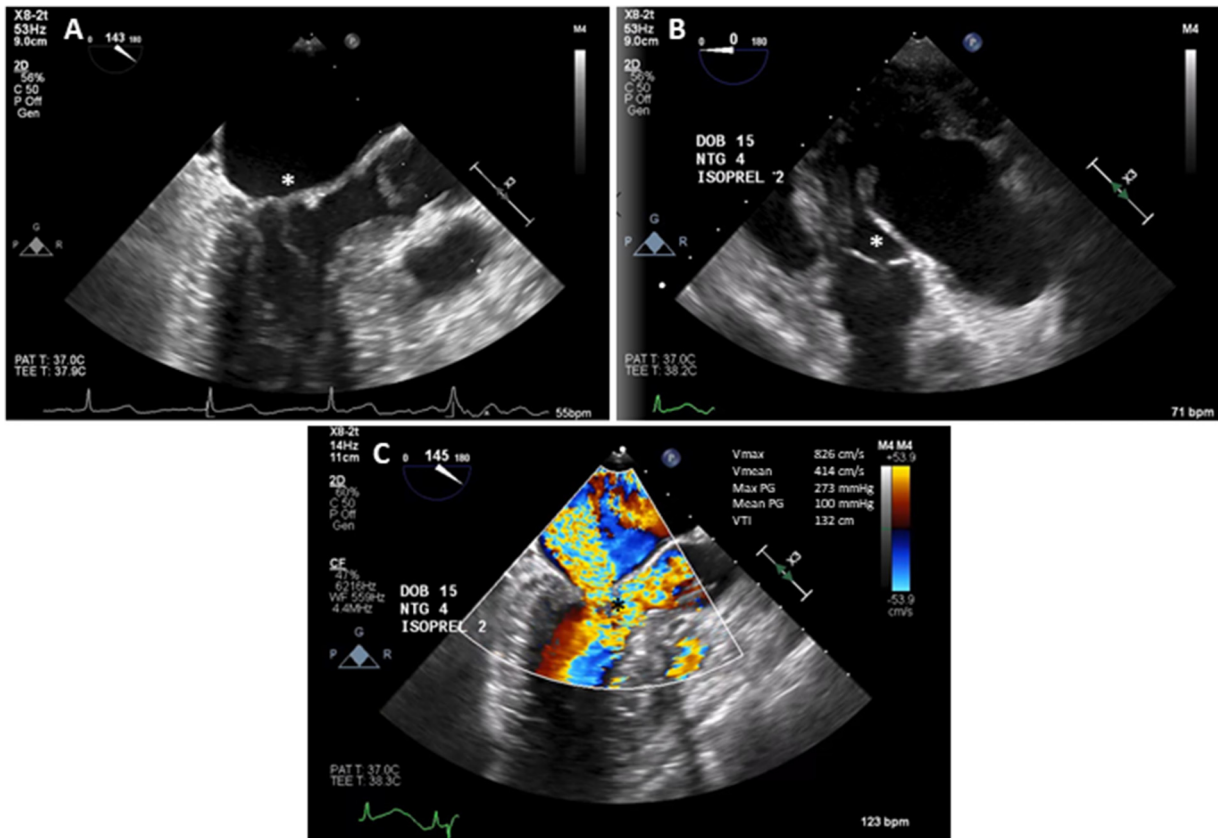
Knowledge of the influence of the MV and IVS on the pathophysiology of HCM is crucial for understanding the mechanism of obstruction in HOCM patients. The MV is composed of the anterior and posterior leaflets, the annulus, the chordae tendineae, and the PMs. The MV is a bicuspid valve where the two leaflets are divided into anterior and posterior portions and can have different forms and variable length.<sup>16</sup> The MV derives from the atrioventricular cushions, also called endocardial cushions, structures that form early on during heart development. These cushions, mainly composed of myocytes, endocardial cells, and a matrix rich in hyaluronic acid, undergo a series of cell proliferation, cell differentiation, and matrix remodeling processes to give rise to the thin anterior and posterior leaflets of the MV. In addition to the MV leaflets, the chordae tendineae will also derive from the endocardial cushions.<sup>16</sup> During this process, the fusion of the inferior and superior endocardial cushions will divide the atrioventricular canal into right and left atrioventricular junctions.

The PMs arise from the apical and two-thirds of the LV wall. Myocardial delamination, consisting of the gradual loosening of the ventricular myocytes, gives rise to two separate equal-sized PMs, arranged in an anterolateral and posteromedial positions.<sup>17</sup> From their tips extend tendine-resembling fibrous cords composed of connective tissue known as

the chordae tendineae. The MV leaflets connect to the PMs within the ventricles via these chordae tendineae.<sup>16</sup> When one of the two PMs does not delaminate properly from the left ventricle, its tip remains attached to the ventricle and results in asymmetric PM development. Furthermore, the abnormal compaction of the ventricular myocardium will result in what is known as a true parachute MV.<sup>16</sup>

MV anomalies of the leaflet, chordae, and/or PMs, in the setting of HOCM can contribute directly or indirectly to LVOTO. A study comparing HOCM patients to control groups revealed that the MV leaflets were 1.5–1.7 cm longer in the HOCM patients.<sup>18</sup> Studies have shown that this leaflet elongation can be potentially caused by sarcomeric gene mutation, concomitant familial MV disease, the stretch effect, or paracrine effects from the adjacent hypertrophic ventricle.<sup>19</sup> SAM of the MV occurs when the MV leaflets deviate toward the ventricular septum during systole.<sup>20</sup> The SAM of the MV is a major contributor to the LVOTO. The severity of the obstruction is also highly influenced by the SAM: when the MVs are dragged anteriorly for an extended period of time, an increase in flow velocity and a narrowing in the LVOT occurs.<sup>21,22</sup> It has been shown that in HOCM patients, the MV leaflets coapt in the central portion rather than at the tips of the leaflets.<sup>18</sup> Occasionally, the posterior leaflet contacts the septum, protruding past the coaptation point of the anterior leaflet.<sup>23</sup> During SAM, and as the leaflets are pulled into the LVOT, their apposition becomes incomplete and asymmetric, leading to the creation of a MR jet that is posteriorly directed.<sup>24</sup> In fact, it has been shown that leaflet length and mobility can affect the severity of the MR.<sup>14</sup> When the posterior leaflet is shorter than the elongated anterior MV leaflet or is not mobile an interleaflet gap is created with the subsequent mal-coaptation commonly seen in HOCM patients.<sup>22</sup> Leaflet coaptation can also be affected by the presence of chordal and PM displacement since they limit the mobility of the leaflets.

PM anomalies in HOCM patients are major contributors to LVOTO. The presentation of these anomalies varies between hypertrophy of PM, fusion of PM to the ventricular septum or to the ventricular wall, double bifid PM, and insertion of PM directly in the AMVL.<sup>25–27</sup> Increased PM mobility was also found to be contributing to the LVOTO in HOCM patients.<sup>28</sup> In fact, a higher peak resting LVOT gradient was found in HOCM patients who had anteroapical displacement of the anterolateral PM and a double bifid PM when compared to patients who did not show these physical traits on cardiac MRI. This increase in the LVOTO was shown to be independent of the presence of thickening of the septum.<sup>29</sup> Furthermore, patients with HOCM were shown to have medial and anterior displacement of both PMs when compared to controls, a determinant that was independently associated with LVOTO. The results of this study were assessed by a real-time three-dimensional echocardiography analysis and revealed that this anomaly results in an inter-PM distance that is narrower than in normal cases.<sup>30</sup>



**Figure 1.** Intra-operative resting and after provocation demonstrating SAM and severe MR. (a) At rest and (b) following provocation intra-operative trans-esophageal echocardiography images showing a long axis view of the left ventricle prior to intervention and after isoproterenol provocation. The white asterisk shows severe SAM. (c) Intra-operative trans-esophageal echocardiogram image demonstrating significant LVOT flow acceleration (black asterisk) following isoproterenol infusion with significant MR at a posteriorly directed jet. SAM: systolic anterior motion; MR: mitral regurgitation; LVOT: left ventricular outflow tract.

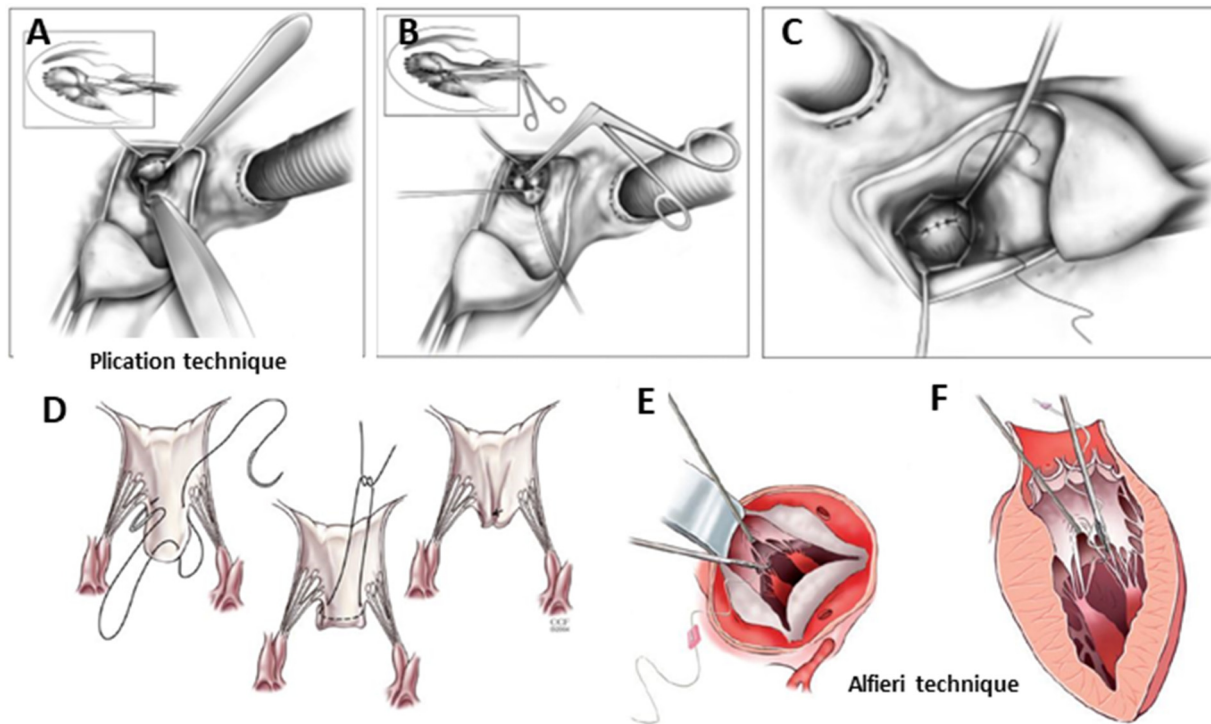
Despite remarkable progress in the past few years in the understanding of the pathophysiology of HOCM, the exact causes and pathways underlying the phenotypic differences observed in HOCM patients remain undefined. This is very likely due to the multifactorial nature of HOCM where mutations in one sarcomeric gene can lead to many different phenotypes. Further research is necessary to better couple the phenotypic differences observed in HOCM patients with their underlying genotypic signature. This will shed more light onto the basis of the observed anatomic variability in imaging and will help tailor *n* surgical intervention in this patient population.

### Techniques beyond septal reduction to alleviate LVOTO

#### *Techniques that deal with the anterior leaflet of the MV and its attachments in the setting of HOCM*

Most of the techniques that have been published address the anterior leaflet of the MV through a trans-aortic approach.

The reason for this is the limited exposure available to the surgeon through the trans-aortic route, where only the anterior leaflet (AL) of the MV and its connections can be clearly visualized. One of the techniques that has been described for shortening the AMVL is the plication of the AMVL (Figure 2(a)–(c)), where a suture line is created immediately below the sub-aortic curtain plicating and shortening the AMVL with this maneuver. This was described by Swistel and Sherrid.<sup>34</sup> With this technique, surgeons do not only shorten the length of the AMVL but also change the coaptation point between the anterior and posterior leaflets closer to the free edge of both leaflets. This technique creates a vertical mattress plication suture line with 5.0 Prolene below and parallel to the sub-aortic curtain, shortening the length of the AL. The AL must be at least 3.2–3.5 cm in length, pliable, non-fibrotic, and without abnormal chordae attached to it. The benefit of this procedure is that it is reversible by removing the sutures if MR is found post-plication. A study by Balaram et al.<sup>35</sup> has used this technique on 82/132 patients with HCM with mean age 55.5 years. Results from this study revealed a significant decrease in LVOT gradient



**Figure 2.** Techniques that deal with the anterior leaflet of the MV and its attachments in the setting of HOCM: plication and Alfieri techniques. (a) *Plication technique.* Resection of hypertrophied septum is carried out with a number 10 blade knife, starting at the level of the right coronary ostia and moving anteriorly (leftward). Traction is supplied by a trefoil hook placed deeply within the ventricular cavity. (b) The hypertrophied papillary muscles are released and thinned with a pituitary rongeur. Often the papillary muscles have grown into the anterior septum or there are abnormal attachments, which can be divided without fear of causing mitral insufficiency. (c), (d) Plication of elongated anterior mitral leaflet. Base (c) versus free edge (d). (c) Plication of the anterior leaflet of the MV is accomplished with the placement of four to six horizontal mattress sutures of 5-0 prolene. (e), (f) Alfieri technique. View from the aortic orifice and (e) sagittal view. The edge-to-edge U suture is placed in the central portion of the leaflets (A2 and P2) on either side. (a)–(c) Were reprinted with permission from Swistel and Balam. <sup>31</sup> (d) Was reprinted with permission from Hodges et al. <sup>32</sup> (e), (f) were reprinted with permission from Obadia et al. <sup>33</sup> MV: mitral valve; HOCM: hypertrophic obstructive cardiomyopathy.

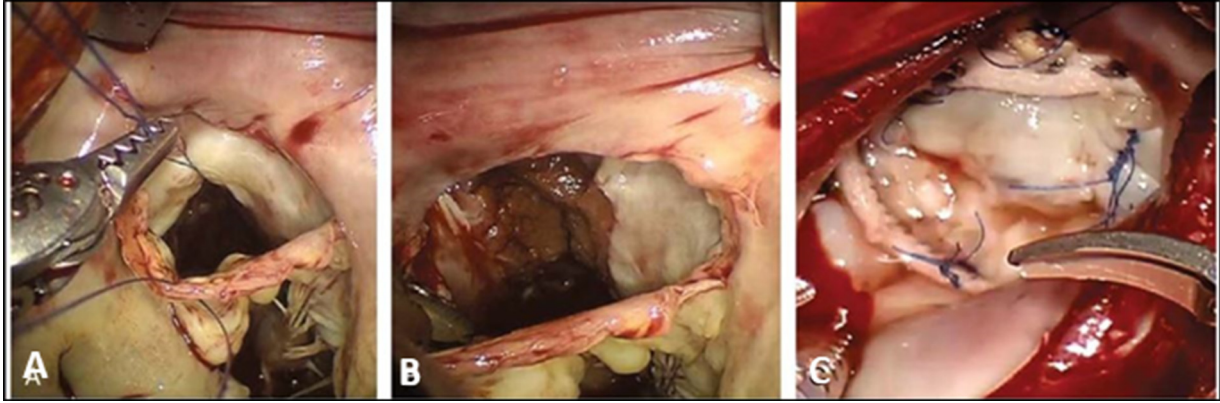
following procedure and resolution of SAM in 96.2% of patients. No operative mortality was observed and on follow-up (average 5.6 + 3.9 years) results showed no significant changes in terms of LVOT gradient and MR.<sup>35</sup> Another option is the free-edge plication of the ALMV described by NGS where a U-suture is placed plicating the excess tissue at the level of the A2 free edge (Figure 2(d)).

The second option is to use the Alfieri technique (Figure 2(e), 2(f)). This technique fixes the coaptation point between the anterior and posterior leaflets of the MV using a couple of sutures. First, a standard septal myectomy is performed. Then, the MV is examined through the aortotomy, followed by placement of two 4-0 polyester figure-of-eight sutures, between A2 and P2. These suturing points are identified as the junctions between the anteromedial and posterolateral PM chords.<sup>36,37</sup> The dragging that exists in these HOCM cases, where the AL is pulled toward the LVOT, following the Alfieri repair technique, can no longer occur, since the PL holds the AL away from the LVOT. The study by Shah et al.<sup>37</sup> used this technique on 24 patients who

underwent septal myectomy and MV repair, all having documented SAM. Following this procedure, the mean LVOT gradient decreased drastically, with none of the patients showing any residual SAM or requiring a second aortic cross-clamping for mitral repair postoperatively. In addition, some patients showed improvement in their MR with a mean postoperative MV gradient of  $4.5 \pm 3.0$  mmHg.

The third option, instead of dealing with the AL length, addresses the coaptation of the leaflets. The coaptation in HOCM patients is abnormal and results from the extended length of the AL. This technique patches the AL making it even longer and it is known as the MV extension technique (mitral valve leaflet extension (MLE)) (Figure 3(a)–(c)). MLE was first described by Kofflard et al.<sup>39</sup> in 1996, and is a procedure always done in the setting of a myectomy by this group. These operations are approached through a left atriotomy as detachment of the AMVL from the annulus is required. When detached, patching allows for a tension-free reattachment of the AL to the annulus. This is the only technique that approached the myectomy and





**Figure 3.** Techniques that deal with the anterior leaflet of the MV and its attachments in the setting of HOCM: mitral leaflet extension and resection of ectopic secondary chordae. (a) Mitral leaflet extension technique. Detached anterior mitral leaflet for exposure. (b) View of septum after septal myectomy. (c) Final view of the MV. (a)–(c) were reprinted with permission from Bayburt et al.<sup>38</sup> MV: mitral valve; HOCM: hypertrophic obstructive cardiomyopathy.

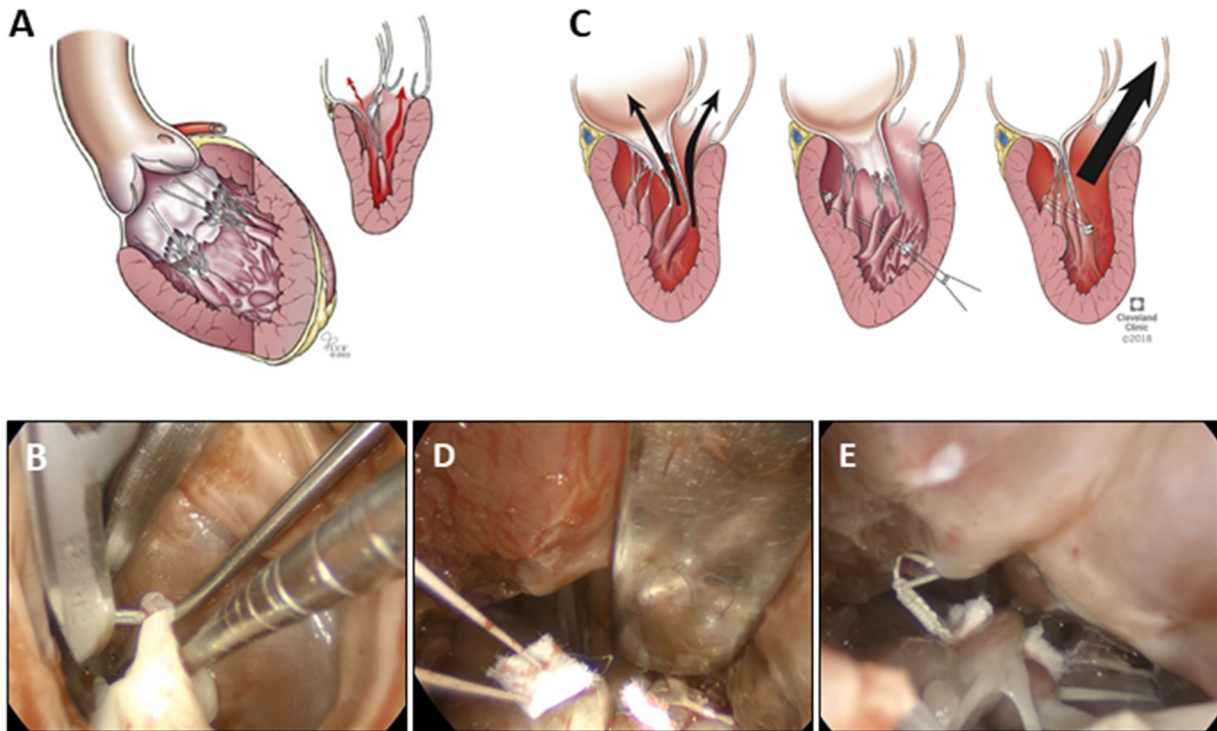
SAM through the left atriotomy instead of the universally used trans-aortic approach. Patching of the MV in this technique is necessary to displace the coaptation point between the anterior and posterior leaflet of the MV more posteriorly. In the investigation by Van Der Lee et al.,<sup>40</sup> 29 patients with HOCM were studied with a mean follow-up of  $3.4 \pm 2.1$  years.<sup>40</sup> Following the procedure, the results of the study showed a reduction in LVOTO, in addition to a decrease in SAM and MR of the MV. The patch in one of the patients detached and therefore the patient required reoperation. The authors believe that this technique first stiffens the central part of the collapsing AL, stopping its abnormal mobility. In addition, by increasing the width of the leaflet, a lateral shift and stretching of the chordae that are attaching to the center of the valve occur, leading to the enhancement of leaflet coaptation and maintenance of valve tethering.<sup>40,41</sup> There are two main limitations in this study. The first is highlighted by the absence of any provocation during the procedure in the operation room. The second is the fact that they performed a limited myectomy, where the myectomy did not pass the free edge of the ALMV.<sup>40</sup> Another study by Gutermann et al.<sup>42</sup> also used this approach on 12 symptomatic patients with diffuse hypertrophy. Results of the study revealed comparable disappearance of the LVOT gradient and correction of MV abnormalities including SAM and MR. Only one patient died due to LV diastolic dysfunction.<sup>42</sup> Calcification of this patch is a potential long-term risk of this technique. Long-term results are not available either.

In patients with thin septum, the presence of fibrotic abnormal secondary chordae inserting into the aortic side of the AMVL tethers the AL and displaces the MV apparatus toward the IVS. In normal anatomy, the MV chordae insert on the ventricular side of the AMVL and go from the free edge to the base (primary, secondary, and tertiary

chordae). Resection of this fibrotic abnormal chordae restores mobility of the entire leaflet of the MV moving the coaptation point posteriorly in the LV (Figure 4(a), (b)). This prevents the anterior displacement of the MV leaflets during systole reducing the possibility of LVOTO. A  $23 \pm 2$  months follow-up series was published by Ferrazzi et al.<sup>43</sup> looking at a total of 268 patients with HOCM where 39 patients underwent a surgical cutting of MV secondary chordae combined with a shallow septal myectomy. Results showed resolution of the LVOTO, with no patient showing postoperatively MV prolapse or flail, nor requiring MV replacement; one patient had residual moderate-to-severe MR at the most recent evaluation. In addition, no patient died during hospitalization or follow-up.<sup>43</sup> The benefit of this approach is that it increases the AMVL: annulus ratio indicating that a proper repositioning of the leaflet coaptation point has occurred, which allows for an increase in LVOT size. This procedure also decreases the MV tenting area, which reduces the systolic displacement of the MV leaflets toward the apex. One limitation of the study is their selection criteria for the studied patient population (patients who underwent secondary chordae cutting) which only included patients with relatively thin septums ( $\leq 19$  mm), an LVOT gradient ( $\geq 50$  mmHg), and disabling symptoms with patients unresponsive to treatment.<sup>43</sup>

#### *Techniques that address the sub-valvular MV apparatus*

Another abnormality is when PMs insert directly onto the AMVL, therefore tethering the AMVL the same way abnormal ectopic chordae would. In this case, it is a muscular band that creates a traction force onto the AL of the MV limiting its mobility.<sup>44</sup> The PMs in HOCM patients are



**Figure 4.** (a), (b) Resection of ectopic secondary chordae causing tethering of the anterior leaflet of the MV and SAM. Tethering secondary chordae are often present on the anterior mitral leaflet that pull the leaflet into the left ventricular outflow tract. Resecting these chordae allows the zone of coaptation to move posteriorly, away from the outflow tract. (c)–(e) Technique that addresses the MV sub-valvular apparatus by re-aligning the papillary muscles posteriorly using a pledgeted 4-0 Goretex suture. Large, excessively mobile or anteriorly displaced papillary muscles can contribute directly to the LVOTO and pull the anterior leaflet toward the outflow tract, potentiating SAM. Papillary muscle reorientation is performed by tacking anterior papillary muscle heads to posterior heads, moving the papillary muscles and MV zone of coaptation away from the outflow tract. (a), (c) were reprinted with permission from Hodges et al.<sup>32</sup> SAM: systolic anterior motion; LVOTO: left ventricular outflow tract obstruction; MV: mitral valve.

hypertrophic in many instances. They are larger than in normal patients, and therefore occupy more space. In addition, the insertion of the PMs is abnormal when compared to the normal anatomy. That contributes, in some patients, to the mechanism of obstruction. Another technique is to identify this abnormality and mobilize the PM and realign it posteriorly, toward the posterior PM group placing sutures, moving the anterior lateral PM group away from the LVOT preventing the mechanism of obstruction from occurring again (Figure 4(c)–(e)). Surgical results of PM reorientation were presented by the Cleveland Clinic group.<sup>28,45</sup> In their study, a total of 204 patients were divided in three groups: isolated myectomy, myectomy plus MV repair/replacement, PM reorientation with and without myectomy. The results of the study highlighted the effects of PM reorientation in reducing the symptomatic LVOT gradient in these patients.<sup>28</sup> This was even true in patients with a septal thickness <1.5 cm. This technique confirmed the observation that in some patients with LVOTO, abnormal PM morphology has been observed. It is well-known that anterior apical displacement and bifid PM anatomy is frequently observed in patients with HOCM.<sup>29,46</sup> In the latter study, the authors used

cardiovascular magnetic resonance to evaluate PM morphology. Interestingly, even in the absence of significant septal hypertrophy, they were able to reproduce significant LVOTO and provokable SAM in many of these patients.

The surgical intervention involved a trans-aortic approach for all cases and PM realignment was done using a pledgeted mattress suture that was placed through the head of the posterior medial PM, that then will be passed through the anterior head of the anterolateral PM.<sup>28</sup> The authors show that attention needs to be placed on passing the sutures through the fibrous portion of the PM to avoid ischemia. The number of sutures (1–2) is variable from patient to patient with a final goal of realigning the anterolateral PM group toward the posterior medial group. The video that is included with this manuscript demonstrates very clearly the mobilization of the base of the anterolateral PM group. The suture is being passed through the head of the posterior medial PM, toward the anterior lateral group, displacing the anterolateral group away from the IVS. Interestingly, the maximal basal septal thickness in group 3 in the series reported by the Cleveland group was <1.5 cm and therefore an aggressive myectomy was limited by this very dimension.<sup>28</sup> In

addition, none of the patient's requiring PM reorientation required pacemaker implantation when compared to groups 1 and 2 in that paper. One of the patients in this group required a MV replacement due to the persistence of SAM of the MV and moderate-to-severe MR after this type of procedure.

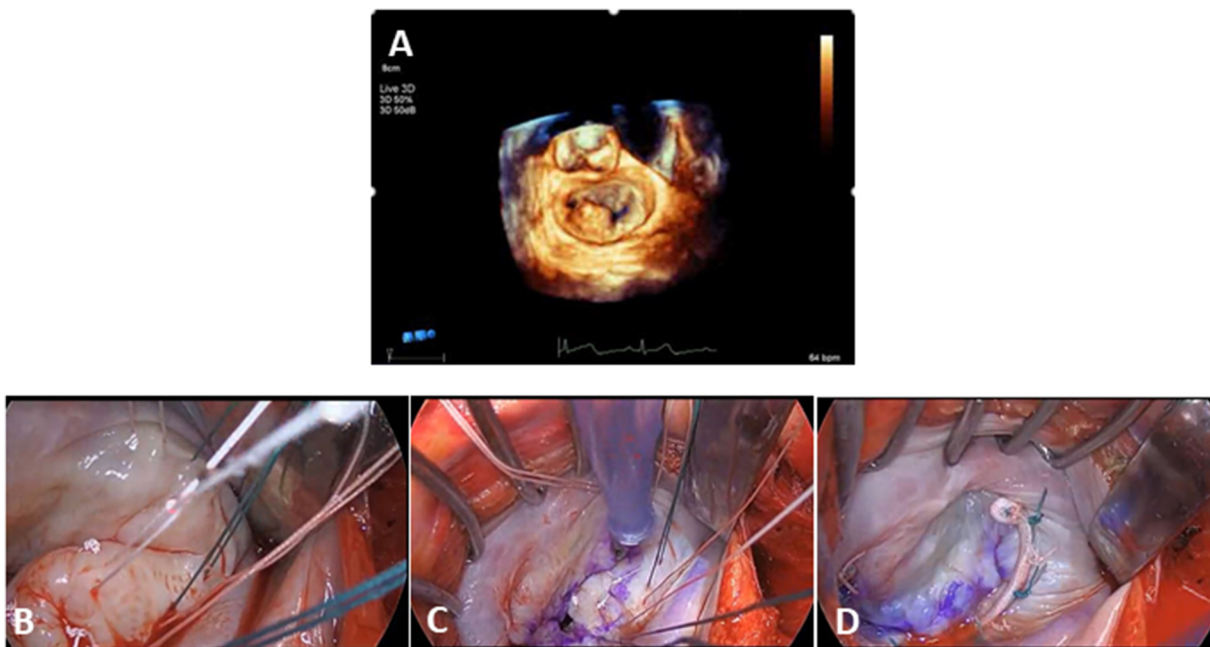
In some instances, patients with HOCM will present with myxomatous MV prolapse (Figure 5(a)) requiring repair in addition to relief of the LVOTO via myectomy (Figure 5(b)–(d)). This is a small group of patients with complex pathologies and in whom the preoperative assessment and intra-operative planning must be exquisite. When the MV repair for MV prolapse is necessary in cases of HOCM, one must be very cautious to not undersize the annuloplasty device too aggressively. This will invariably displace the AMVL toward the LVOT. In addition, when the posterior leaflet requires repair, the operator needs to maintain the height of the prolapsing segment close or identical to the height of P1, which normally is not prolapsing in most cases. This will allow for a short posterior leaflet against which the anterior leaflet can coapt as posteriorly as possible within the mitral orifice, and therefore away from the outflow tract. We prefer the use of artificial chordae to lower the height of P2, as described by Perrier et al.<sup>47</sup>

## Discussion

What is clear is that all patients with HOCM are in need of an extended septal myectomy. What is not as clear is

whether all patients with septal hypertrophy and LVOTO will substantially improve both at rest and during dynamic testing with just an operation that addresses the septum by doing an extended septal myectomy only. The diagnostic modalities and the management tools that we have been able to develop and use for the evaluation of patients with this pathology have improved quite significantly over the past few decades. Our ability to understand the dynamic mechanisms of obstruction has transformed the way we conduct our operations. Through these new imaging technologies, we have been able to appreciate the contribution of the anterior leaflet of the MV, the hypertrophic PMs, and the septum, to the LVOTO. This becomes even more relevant with patients with thin septa where a deep myectomy is just simply not possible without incurring significant complications. It is in these cases, where addressing the long AMVL via different techniques and displacing the anterior lateral PM posteriorly, becomes necessary to relieve the obstruction of the LVOT.

All the techniques on the AMVL mentioned in this review have one thing in common. Whether a resection of the anomalous chordae is done, or a shortening or patch extension of the AMVL is performed, the goal is always the same and that is displacing the coaptation point between the anterior and the posterior of the MV posteriorly and away from the IVS. Choosing one strategy over the other depends heavily on the level of comfort that the operator has. For instance, surgeons not familiar with HOCM may choose an atrial approach with patch extension versus a trans-aortic approach, as they may be more familiar with inspecting the



**Figure 5.** Myxomatous valve. (a) P2 prolapse with a tall prolapsing segment. (b)–(d) Mitral valve repair: (b) posterior chordae significantly reduced in height. (c), (d) Coaptation of the posterior leaflet inside the ventricle.



MV through a left atriotomy. However, the use of the Alferi technique via a trans-aortic approach seems to be preferred by others due to its efficacy and high reproducibility. Some surgeons have adopted and described plication and shortening of the AMVL as an easy, accessible, and reversible technique to address the same problem.

In our opinion, intervention on the sub-valvular apparatus, including the abnormal connections of the MV to the PMs and/or septum, is also crucial to help relieve the LVOTO and restore the normal mobility to that part of the valve. In many cases, the PMs are also hypertrophied and displaced apically generating a space conflict between these displaced hypertrophic PMs and the IVS. The posterior realignment of the anterior–lateral PM group addresses these anatomic abnormalities by moving the anterior–lateral PM away from the outflow tract of the ventricle by using simple pledgeted sutures.

To understand how different elements are contributing to the obstruction, which is in most cases absent at rest, dynamic testing either preoperatively or intra-operatively is required as it uncovers the presence of inducible severe mitral insufficiency caused by SAM as the heart rate increases and the blood pressure drops. In these patients as the mitral insufficiency (MI) appears, the turbulent flow to the LVOT becomes more and more permanent. We believe that every patient should have provocative testing intra-operatively before and after the operation is completed to document successful static and dynamic resolution of the obstruction.

With this disease, no generalization can be done; one operation does not fit all. More personalized patient-tailored approaches need to be adapted by the HOCM team based on the knowledge of the available techniques and the complexity of the case. Around the world, only few surgeons operate on HOCM cases, and few centers provide these services. This makes it difficult to improve and expand any techniques when the denominator of surgeons performing this operation remains so small. Any effort to expand the number of centers and operators helping patients with this condition would demand the protocolization and simplification of the surgical techniques used to relieve the LVOTO so it can be performed by many surgeons around the world. It is mandatory for any center embarking in the treatment of patient with HOCM to provide life-long follow-up to these patients so the surgical community can learn which techniques work best in the long term.

## Conclusion

The elements involved in generating the LVOTO are multiple and varied in patients with HOCM, and include the septum, the ALMV, the chordae tendineae, and the PMs. Addressing the contribution to the obstruction by each one of these elements, in addition to the hypertrophic septum, is a crucial step to plan a successful surgical

intervention. The techniques described in this review are meant to be resolute and complementary to each other with the aim of decreasing complications.

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## Informed consent

Not applicable.

## ORCID iDs

Lara Gharibeh  <https://orcid.org/0000-0002-9520-3403>

Nicholas G Smedira  <https://orcid.org/0000-0002-4963-8588>

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