

Systolic Anterior Motion after Mitral Valve Repair: Hemodynamic Optimization or Provocation for Surgical Correction in the Operating Room?

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Systolic anterior motion (SAM) of the mitral valve (MV) is a known complication of MV repair for myxomatous mitral valve disease.¹ Several preoperative echocardiographic predictors and preventive surgical techniques have been described in the literature.² Despite best practices of echocardiographic prediction and operative adaptation to reduce the incidence during mitral repair, SAM continues to occur in 4 to 11% of patients after MV repair.³⁻⁶ Evidence supports conservative medical management with rate control, volume optimization, maintenance of afterload with adequate blood pressure and avoidance of diuretics and vasodilators. Few patients with SAM require surgical intervention. The question remains is where to draw the line for expectant conservative medical management and definitive eliminative surgical therapy. Several groups addressed their protocols for the management of SAM after MV repair.³⁻⁶

Brown et al from Mayo Clinic published a large series (n = 1589) and reported an incidence of 11% SAM (n = 174) after MV repair for myxomatous valve disease.³ They did not believe in predictors or preventive techniques and instituted medical management in all of their patients with SAM. They even advocated medical management for patients with SAM and left ventricular outflow obstruction (LVOTO). LVOT gradients ranged from 16 to 64 mm Hg. They hypothesized that remodeling of the left ventricle over time with medical therapy will lead to resolution of SAM and LVOTO. One unique aspect of the Mayo series is their repair technique utilizes all flexible partial band annuloplasties. None of their patients required late reoperation > 4 weeks because of SAM and no postoperative mortality was observed related to SAM on a median follow-up of 5.4 years (0-13.2 years). Only two patients with SAM had intraoperative re-repair considering their advanced age and two others had early postoperative re-repair < 4 weeks because of SAM related complications (pulmonary edema, high LVOT gradient and hemolytic anemia). Since, there are risks associated with second pump run, their results condoned a 'leave it alone' strategy

unless there is obvious hemodynamic decompensation or other surgical reasons (e.g. inadequate leaflet resection). However, this study remains controversial to many surgeons when faced with more extreme situations (e.g. SAM and significant LVOTO).

Two other large institutions recently published their experience with SAM. They both instituted a step-wise approach to the management of SAM after MV repair compared to Mayo group. Varghese et al reported their experience in 785 patients who underwent MV repair.⁴ The incidence of SAM was 6.6% in their study. Technically, inappropriate repair, symptoms, hemodynamic instability and persistent LVOT gradient >50 mm Hg or more than mild MR in spite of medical optimization were indications for re-repair. Six out of 41 (14.6%) required surgical intervention on posterior leaflet for SAM in their series. In their median follow-up of 1.3 years, no adverse events related to SAM were reported. Crescenzi et al in their series of 608 patients undergoing MV repair reported SAM in 60 patients (9.8%).⁵ They instituted medical optimization in the post-bypass period similar to Varghese et al's study but surgical intervention was considered for any persistent SAM with even lower LVOT gradients (20-50 mm Hg) and mild to moderate MR. Only five patients required surgical intervention in their series and long-term follow-up was not reported. Both of these two groups developed a well-defined algorithm for the management of SAM after MV repair. Most surgeons appear to follow such a protocol-based approach.

Though the somewhat benign nature of SAM is generally accepted, several isolated reports of late onset of complications related to SAM keep appearing in the literature.⁷⁻⁹ This limits the wider application of the results of retrospective studies to individual patient management in the operating room. Furthermore, many patients with myxomatous valve disease are young and healthy and often present for repair with minimal symptoms and thus continuation of beta-blocker with requirement for frequent follow-up echocardiograms can be considered as partial surgical failure.

In this issue of the *Journal of Perioperative Echocardiography*, Bardia et al report a patient with SAM and stable hemodynamics in the operating room that went on to decompensate in the early postoperative period.¹⁰ Is

there a strategy to identify such patients? The answer is 'yes'. Manecke et al described a 'systolic anterior motion tolerance test' in such borderline patients with SAM.¹¹ This is a completely opposite strategy to the typical conservative hemodynamic optimization strategy. Optimization strategy produces favorable hemodynamic conditions (volume infusion, avoidance of inotropic drugs, heart rate control with beta-blockers and increased afterload with vasoconstrictors) to suppress the development of SAM. In SAM tolerance testing, Manecke et al challenged a patient of SAM and stable hemodynamics with nitroglycerine (decreased pre and afterload), rapid pacing and dopamine (tachycardia). The patient tolerated these SAM aggravating conditions without decompensation and it was decided not to intervene SAM in the operating room. This can be compared to a pharmacologic stress test to detect ischemia in patients with coronary arterial disease.

At present, there are no clinical studies validating such a protocol in patients with SAM after MV repair. Several centers employ provocation test protocol for septal myectomy before and after bypass. Patients with no SAM at rest or on provocation are considered to have undergone successful myectomy. Ashikhmina et al reported the use of isoproterenol for provocative test in patients with hypertrophic cardiomyopathy who underwent septal myectomy.¹² In the post-bypass period, eight patients with higher outflow gradients after provocation study required repeat bypass for completion myectomy.

Typical conservative medical optimization strategy banks on good postoperative regimen to maintain optimal hemodynamic conditions until LV remodeling takes place to naturally eliminate SAM. In contrast, systolic tolerance testing and provocative strategies aim to eliminate trouble early by identification of patients who can decompensate when challenged by SAM aggravating hemodynamic scenarios. Such hemodynamic situations are not uncommon early after surgery during hospitalization or after discharge (sympathetic stimulation with tachycardia, tachyarrhythmias, dehydration or low blood pressure). The merits of employing provocative SAM tolerance testing in selected patients when we sense problem in the operating room may be best answered by future clinical trials.

For now, SAM is commonly considered predictable and preventable in most high volume mitral repair programs. When the common predictors for SAM are identified on the preoperative TEE², the surgical strategy of posterior leaflet height reduction and true-sized or slightly oversized

annuloplasty remain the surgeon's primary methods for SAM avoidance.

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