REVIEW ARTICLE

SYSTOLIC ANTERIOR MOTION OF THE MITRAL VALVE AFTER SURGICAL REPAIR: RISK FACTORS, DIAGNOSIS, AND MANAGEMENT ALGORITHM

ABSTRACT

The exponential growth of mitral repair surgery has transformed the prognosis of patients with severe mitral regurgitation. All expert consensus and current clinical guidelines recognize the preponderance of mitral reconstruction. The incidence of complications inherent to more conservative repair techniques, such as systolic anterior motion (SAM), has also increased. The mechanisms of SAM are complex and depend directly on the characteristics of the mitral apparatus after plasty and, of course, on the hemodynamic state of the left ventricle. In this context, echocardiography has an important diagnostic and prognostic role. Approximately 90% of cases of SAM can be resolved conservatively; therefore, an orderly multidisciplinary approach is critical in understanding the substrate of the problem and knowing when and how to intervene. A simple algorithm for the management of perioperative MAS is presented below.

Keywords: *mitral repair surgery, severe mitral regurgitation, systolic anterior motion.*

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INTRODUCTION

At the height of the boom in mitral reconstruction and more conservative techniques based on the exclusive use of neo-strings, perioperative SAM has been reported in up to 15% of patients[1]. Moreover, according to the latest publications, the medium and long-term incidence may be as high as 7%[2]. SAM rarely occurs in patients with degenerative disease, whatever the size of the leaflets or the degree of myxomatous degeneration. For this reason, the occurrence of MAR after reconstructive plasty is considered an iatrogenic phenomenon[3].

The degree of SAM after reconstructive surgery depends directly on the characteristics of the mitral valve apparatus, which includes the annulus, leaflets, chordae tendineae, papillary muscles, and left ventricle. The hemodynamic status of the latter is critical to the onset, assessment, evolution, and treatment of SAM. The onset of SAM is typically associated with mitral insufficiency and LVOT obstruction, which, without treatment, can lead to acute pulmonary edema, heart failure, and sudden death. Despite multiple techniques and surgical maneuvers to avoid it, its incidence is still high[4]. Consequently, specialists involved in the post-surgical care of patients after mitral plasty must have the capacity to manage this complication.

Although most cases of SAM resolve with conservative management (medical and hemodynamic treatment), about 10% of cases may require early reintervention[5]. Furthermore, the cardiovascular specialist should not only be comfortable with SAM's diagnosis and essential medical management but also with risk analysis and coordination of an orderly multidisciplinary approach to understand the substrate of the problem and know when and how to intervene. A simple algorithm based on echocardiographic calculations and hemodynamic findings for the perioperative management of SAM may be especially valuable for managing any patient presenting with this complication.

DEFINITION

SAM is defined as the displacement of the distal portion of the anterior leaflet of the mitral valve into the left ventricular outflow tract (LVOT) in systole. The main trigger for SAM after mitral reconstruction is the mismatch between the annulus dimension (annuloplasty-induced alteration) and the resulting amount of leaflet tissue (directly proportional to the repair techniques employed). Although several potential mechanisms have been studied and described, the Venturi effect (pull) and the drag phenomenon (push) are among the predominant ones. In this context, both mechanisms describe the displacement of the anterior leaflet into the LVOT with consequent interruption of outflow[6].

As the left ventricle contracts and expels blood into the aorta through the outflow tract, it pulls and pushes the anterior leaflet tissue into the LVOT, interrupting the laminar flow. The greater the presence of tissue below the coaptation line (subannular tissue or coaptation surface), the greater the push or pull effect. Likewise, turbulence in the LVOT causes the Venturi effect of the anterior leaflet and mitral insufficiency. Undoubtedly, the drag phenomenon plays a much more critical role than the Venturi effect, significantly the closer the coaptation surface is to the LVOT[7].

There are several risk factors for the development of SAM. These can be divided into clinical or anatomical factors specific to the patient, hemodynamic factors, and technical factors related to the repair (*Table 1*). Early recognition of MAR as a significant cause of hypotension during the perioperative period is critical, as many routine measures to increase blood pressure significantly aggravate MAR.

ECHOCARDIOGRAPHIC CHARACTERIZATION

Echocardiography is still relevant during perioperative anamnesis of the functional anatomy of a dynamic structure such as the mitral valve. In addition, handheld echocardiography has revolutionized the care of unstable patients and, of course, the management of patients with perioperative MAS. Accurate knowledge of the anatomical characteristics of valve lesions and their hemodynamic impact is essential to proceed with an individualized therapeutic plan for each patient[8].

More than four decades ago, based on the echocardiographic motion of the leaflets, Carpentier established the pathophysiological triad of mitral pathology for an orderly and systematic approach to the disease[9]. The triad highlights the importance of differentiating between the disease that leads to insufficiency or stenosis (etiology), the resulting lesions, and how these lesions affect leaflet mobility, i.e., the type of dysfunction they cause. Over time, cardiovascular specialists have adopted this triad or classification, and today. However, its use still needs to be more generalized and widespread; it is conducive to a perfect understanding among cardiologists, cardiac imaging specialists, and surgeons (*Figure 1*).

Clinical	• Age (<60 years) • Male sex • Advanced degenerative disease (severe myxomatosis) • Prolapse of both leaflets • Elongated anterior leaflet • Anterior coaptation line: anterior:posterior ratio <1.3 mm • Limited end-systolic diameter (small ventricles) • Hypertrophic obstructive cardiomyopathy (HOCM) • Reduced mitroaortic angulation (<20°) • Abnormalities of the subvalvular apparatus (anomalous chordal insertion)
Hemodynamic	• Hypercontractility (hyperdynamic ventricle or tachycardia) • Limited preload (short ventricular filling time or hypovolemia) • Limited afterload (hypotension)
Technical	• Excess residual posterior leaflet (anterior coaptation line) • Excess coaptation surface area (>10 mm) • Exclusive use of neo-strings in unusually redundant leaflets • Wrong prosthetic ring size (too small) • Untreated underlying HOCM during the procedure

TABLE 1. Risk factors for systolic anterior motion.

The classification of the different mitral dysfunctions is based on the position of the leaflet margins in relation to the plane of the mitral annulus:

- Type I dysfunction: normal mobility of the leaflets with severe annular dilatation resulting in a central regurgitant jet, perforation, or clefting of one of the leaflets.
- Type II dysfunction: excessive mobility of the leaflets, usually secondary to pathological elongation or rupture of the chordae tendineae. In this case, the regurgitant jet is directed to the opposite side of the affected leaflet.
- Type III dysfunction: restricted mobility due to retraction of the subvalvular apparatus (frequent in rheumatic disease or inflammatory processes) (IIIA) or displacement of the papillary muscle (ischemic remodeling or dilated cardiomyopathy) causing apical displacement (ventriculization or tethering) of the leaflet (IIIB). The jet is directed to the same side of the affected leaflet.

The original classification of the pathophysiological triad included only preoperative dysfunctions. However, if we include dysfunctions secondary to other clinical identities (e.g., hypertrophic cardiomyopathy) or postoperative (e.g., SAM or dysfunction opposing postoperative ventricular failure), there are two more dysfunctions:

- Type IV dysfunction: anterior systolic motion due to hypertrophic cardiomyopathy, induced by hemodynamic disturbances or after mitral repair. A closing jet or leaflet snapping jet of erratic direction is produced in this case.
- Type V dysfunction: hybrid conditions such as posterior prolapse due to anterior valve thrust (a particular form of SAM), opposite dysfunction, anterior pseudo prolapse, or advanced pathologies due to endocarditis[10].

In expert hands, systematic examination of the mitral valve by echocardiography should provide generalized (anterior, posterior, or bivalve pathology) and segmented information on both leaflets (analysis of each segment and commissure), identify excess or scarcity of tissue in the leaflets, evaluate annular dimensions, detail the state of the subvalvular apparatus and estimate ventricular resistance. As for the specific evaluation of the SAM, the parasternal view is of choice. The parasternal plane (long axis), if transthoracic echocardiography is used or the sagittal view if the transesophageal probe is rotated to 120°, cuts perpendicularly across the coaptation line, crossing P2 (on the left of the image) and A2 (on the right of the image). This view is relevant, especially in evaluating the SAM, as the coaptation surface and extent are clearly visible[10]. Additionally, this view allows us to evaluate the annular surface, extrapolating the annular diameter with the anterior leaflet surface.

TYPE I	Normal leaflet mobility	Annular dilatation (without ventriculization) Perforation of leaflets Cleavage of leaflets	Atrial fibrillation Ischemic cardiomyopathy/dilatation of atrial fibrillation Endocarditis (“windsock” deformity) Congenital pathologies
TYPE II	Excessive velocity mobility	“Billowing” findings Localized prolapse Prolapse of both leaflets	Degenerative disease* Endocarditis Rheumatic disease Trauma Ischemic cardiomyopathy Marfan/Ehler-Danlos diseases
TYPE IIIA	Restricted leaflet mobility	Systolic and diastolic restriction	Rheumatic disease Carcinoid syndrome Radiotherapy Systemic lupus erythematosus Mucopolysaccharidosis Hypereosinophilia
TYPE IIIB	Restricted leaflet mobility	Symmetric systolic restriction Asymmetric systolic restriction Annular dilatation (with ventriculization)	Ischemic/dilated cardiomyopathy
TYPE IV	Anterior systolic motion	After mitral repair Hemodynamic induction Hypertrophic cardiomyopathy	Hypertrophic obstructive cardiomyopathy Hypovolemia Inotropic stimulation Tachycardia (arrhythmias)
TYPE V	Opposite dysfunction	Anterior pseudoprolapse** Postoperative ventricular failure Posterior prolapse due to SAM Untreated intrinsic pathology Severe endocarditis	Barlow's disease Ischemic/dilated cardiomyopathy Hypovolemia Inotropic stimulation Tachycardia (arrhythmias) Infectious process

FIGURE 1. Echocardiographic classification of mitral pathology.
SAM: Systolic anterior motion.
*Degenerative disease includes fibroelastic deficiency, advanced fibroelastic deficiency, forme frustre forms, and Barlow's disease.
**Pseudoprolapse: defines the echocardiographic image of anterior leaflet prolapse due to posterior leaflet lesions (posterior leaflet repair is sufficient for resolution).
Adapted from: Castillo & Adams, Valvular Heart Disease: A Companion to Braunwald's Heart Disease, Chapter 19:370, 5th Edition, 2020.

Echocardiographic evaluation of SAM should be performed in the presence of a full ventricle (maximized end-systolic diameter after optimization of preload and afterload) and normodynamics (adequacy of heart rate and ventricular contractility). Although the surgeon assesses the position of the coaptation line during the saline test (as well as the height of the coaptation surface using ink), the line can also be assessed by three-dimensional echocardiography (glass view). The remaining structural and geometric variables to be collated include: a) the ratio between the anterior and posterior leaflet height; b) changes in annular diameter; c) the distance between the coaptation line and the interventricular septum; d) the end-systolic and end-diastolic diameters; and e) the thickness of the interventricular septum (if addressed during surgery).

Any SAM phenomena should be characterized and documented, including LVOT gradient (ideally ≤ 30 mmHg) and the degree of turbulent flow and mitral insufficiency[11].

PERIOPERATIVE MANAGEMENT OF SYSTOLIC ANTERIOR MOTION

With any threat or suspicion of SAM after mitral repair, the checklist of preventive maneuvers should be reviewed, which includes: (a) posterior leaflet height ≤ 1.5 cm, precisely what an open surgical clamp measures, (b) appropriate ring or band size, and (c) dye saline test showing a posterior coaptation line (reflecting the distance between the anterior leaflet and interventricular septum) and a coaptation surface ≤10 mm (some experts are permissive up to 12 mm, the larger the coaptation surface, the greater the entrainment effect in systole) (*Figure 2*). Optimal management of SAM depends on two factors: identification and localization of the anatomical lesion responsible and echocardiographic calculation of its severity[12]. The first step in any algorithm should be conservative, consisting of medical therapy focused on ventricular filling. This will involve volume, immediate and short-acting beta-blockers (e.g., esmolol) and, in some cases, agents with selective alpha-adrenergic effect (e.g., phenylephrine)[13]. Arterial vasoconstriction is also accompanied by venous vasoconstriction. This increases blood pressure and reflex bradycardia, which will be needed in the presence of SAM. The potent arterial vasoconstriction increases systemic vascular resistance (increase in afterload). The overall result is a reduction in cardiac output.

Furthermore, the approach to SAM, whatever the patient's condition, should be a logical and orderly stepwise process. First, cessation of any inotropic medication is indicated to avoid ventricular hyper dynamism and to control the heart rate (the slower the rate, the longer the ventricular filling time, and, consequently, the larger the end-systolic diameter). We aim to bring the heart rate to 60 bpm. In an overlapping manner, we will administer volume to increase preload, maximize ventricular filling, and try to raise afterload as well. The aim is to achieve a mean arterial pressure ≥80 mmHg so that the increase in afterload hinders ventricular emptying to some extent and thus maintains the end-systolic diameter (agents with selective alpha-adrenergic effect can be introduced here, and some surgeons compress the aorta manually for seconds to recreate the scenario). If this fails, beta-blockers are also indicated to try to minimize the hyperdynamic state of the ventricle and the number of beats per minute (as tolerated or allowed by the mean arterial pressure)[14].

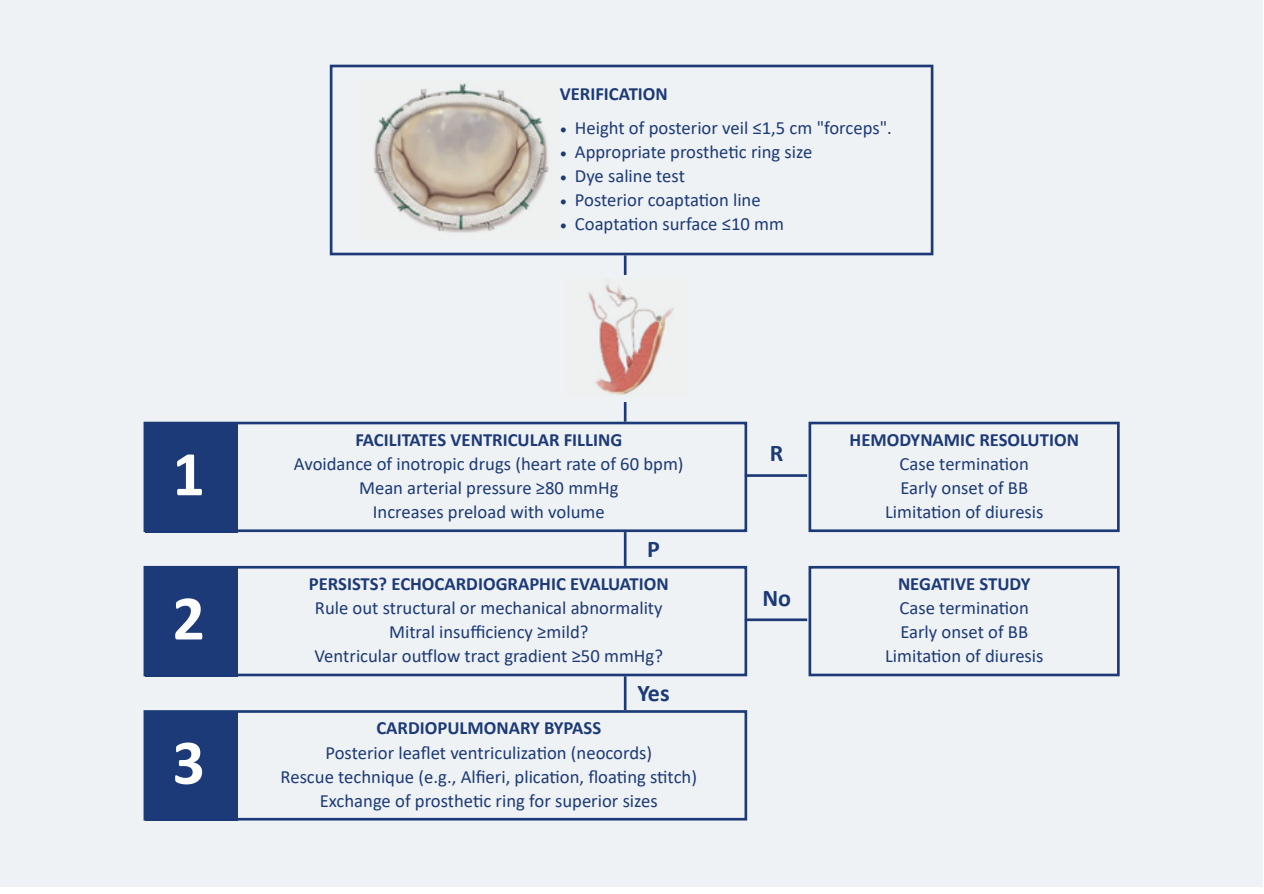

FIGURE 2. Intraoperative management algorithm.
BB: beta-blocker, P: persists, R: resolution.

Resolution of SAM after conservative management entails termination of the case, early initiation of beta-blocker, and, of course, limitation of diuresis (or controlled administration adjusted to preoperative body weight). The persistence of SAM makes it necessary to perform a new echocardiographic study in search of underlying structural or mechanical abnormalities and to calculate the degree of mitral insufficiency and the gradient in the LVOT. If the regurgitation is mild or greater, or the gradient is ≥50 mmHg, we will proceed with a second bypass time for SAM correction. Furthermore, suppose there is a clear excess of tissue in either leaflet, whatever the response to hemodynamic interventions. In that case, the threshold will be too low to reestablish cardiopulmonary bypass and proceed with surgical correction[15]. This eliminates the possibility of SAM in the future. In young patients, especially those with asymptomatic mitral regurgitation, the threshold for surgical correction of SAM is equally low, regardless of the response to conservative treatment. There is a crucial axiom about MAR: if MAR was successfully eliminated with conservative management in the operating room, it could also be managed in the inpatient ward regardless of its severity.

The management of MAS generated in the inpatient ward during the postoperative stay can be much more complex and risky. However, even so, the conservative approach is always the initial one (*Figure 3*). If regurgitation is less than mild or the gradient is <50 mmHg, proceed to increase the dose of oral beta-blocker (as tolerated) and avoid the use of diuretics and antihypertensives if the systolic blood pressure is <135 mmHg. If there is improvement or normalization in the next 48 hours, the patient is discharged, and a follow-up echocardiogram will be performed in four weeks[16]. If there is no improvement, we should assemble a team of cardiovascular specialists to plan individualized management and reoperation (*Table 2*).

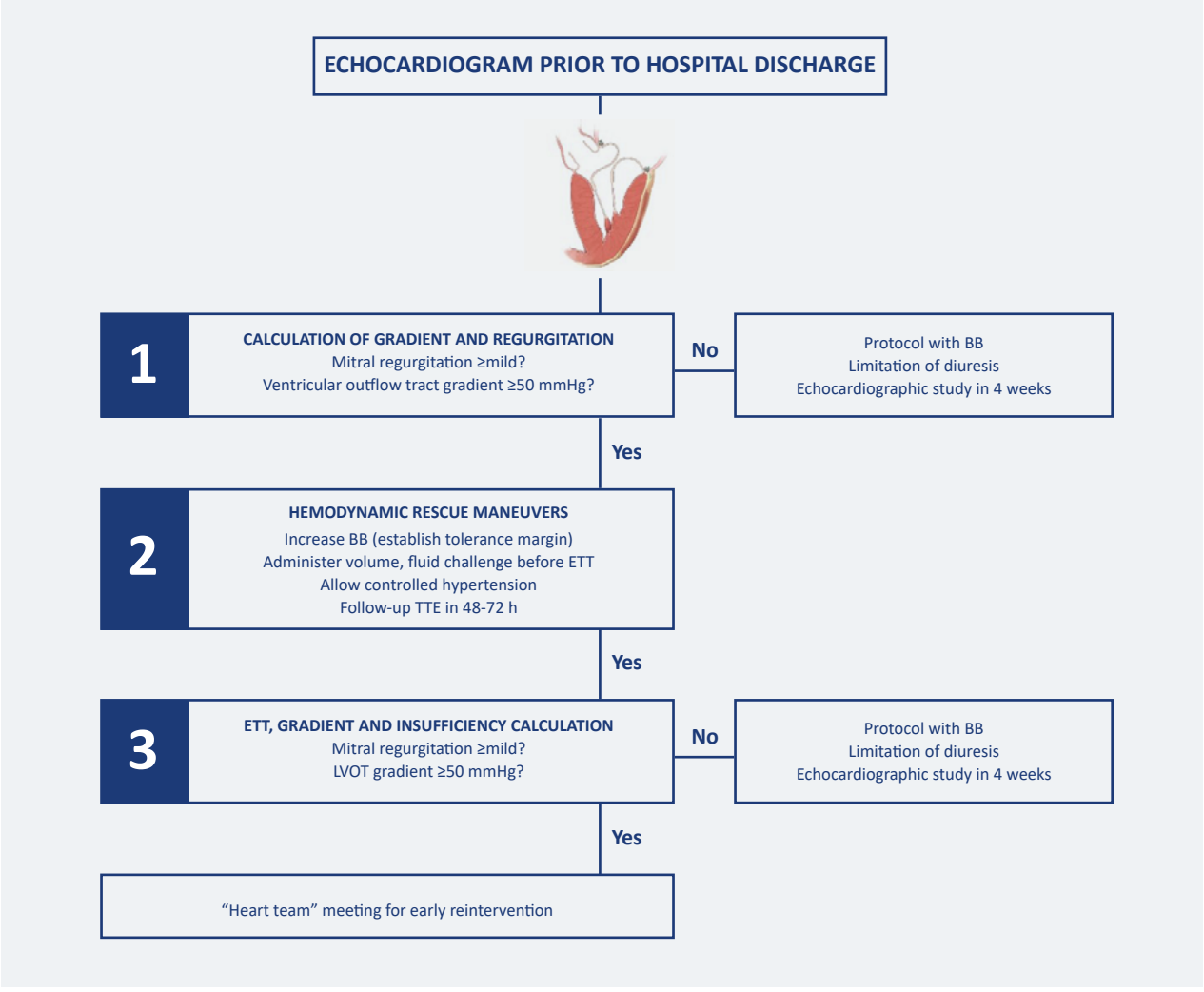

FIGURE 3. Postoperative management algorithm.
BB: beta-blocker, TTE: transthoracic echocardiogram, LVOT: left ventricular outflow tract.

Salvage techniques	Features
Posterior leaflet displacement (ventriculization) with neo-strings	First option due to its speed and simplicity. Ventriculization with neo-strings (Gore-Tex™ CV-5) of the posterior leaflet leads to more significant deployment of the anterior leaflet and displacement towards the posterior annulus of the coaptation line (greater distance between the anterior leaflet and the interventricular septum).
Alfieri's point (symmetric or asymmetric)	• Displacement of the coaptation line and anchoring of the anterior leaflet. • May not work in very redundant leaflets, distorts the native morphology of the mitral valve (dehiscence), and generates higher postoperative gradients.
Floating stitch	• Gore-Tex™ stitch between the free edge of A2 and the posterior prosthetic annulus (P2) to deploy the anterior leaflet and facilitate its anchoring without altering the movement of the posterior leaflet. Variants with two lateral points have been described. • A priori, the possibility of dehiscence is lower.
Posterior leaflet longitudinal plication (shortening)	• Posterior displacement of the coaptation line after reducing the height of the posterior leaflet by about 5 mm. 4-0 polypropylene suture is used, and four mattress stitches are made with pledget support, U-stitches, or cut and sew technique. • It takes an additional 10 to 15 minutes.
Anterior leaflet longitudinal plication (shortening)	• As described above for the posterior leaflet. • It requires the certainty that the problem is not the height of the posterior leaflet since, with this technique, the anterior leaflet is shortened, and the coaptation line can be displaced towards the LVOT.
Replacement of the prosthetic ring with a larger size or a band	• Remodeling annuloplasty with a larger annulus size (either because of a technical measurement error or to accommodate changes in the native annulus) leads to increased deployment of the anterior leaflet. This also happens if bands are used instead of rings (greater anteroposterior distance). • It is much more time-consuming.

TABLE 2. Surgical techniques to correct systolic anterior motion after mitral repair.

CONCLUSION

The occurrence of SAM after reconstructive mitral surgery is considered a mainly iatrogenic phenomenon. It is a complex mechanism involving the entrainment of the anterior leaflet into the LVOT in the systolic phase. Outflow tract obstruction causes turbulent flow that can lead to mitral regurgitation, acute pulmonary edema, and even sudden death. Perioperative diagnosis is eminently echocardiographic. The management of MAS requires a logical, stepwise approach ranging from conservative medical management to reoperation. Decision-making should be based on the severity of the MAS and the experience of the multidisciplinary team in charge of the patient.

Declarations
The authors declare no conflict of interest.

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