Review

The surgical management of giant left atrium

Efstratios Apostolakis a, Jeffrey H. Shuhaiber b, *

a Cardiothoracic Surgery Department, University Hospital of Rion, 26500 Rion Patras, Greece
b Department of Thoracic and Cardiovascular Surgery, Loyola University Stritch School of Medicine, Chicago, IL 60153, United States

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Summary

Giant left atrium (GLA) is a condition defined when the left atrial diameter exceeds 65 mm. GLA is commonly associated with mitral valve regurgitation due to excess intracavitary pressure resulting in strain and dilation of the left atrial chamber. The mechanism of lone GLA remains unknown but is possibly related to inherent weakening of the atrial wall tissue. The enlarged left atrium leads to expansion of left atrial volume, which in turn can place pressure on the main bronchus, lung, and left ventricle with corresponding cardiopulmonary embarrassment. Because GLA can increase the risk of sudden death, its existence merits careful evaluation and surgical intervention when needed. Careful review of the literature reveals that the presence of GLA in the context of severe mitral valve regurgitation with or without atrial fibrillation is the most common indication for surgical intervention. Indications for intervening on lone GLA are rare except when compressive symptoms manifest. Partial resection of inferior and or superior left atrial wall is the most common surgical technique. With the evolution of atrial fibrillation surgery, atrial size matters and is determinant of long term performance following successful ablation. Surgical management of GLA achieves good clinical outcome with respect to cardiopulmonary performance including restoration of sinus rhythm among patients suffering from atrial fibrillation. Surgeons should be aware of current modalities for atrial volume reduction when indicated to retain the function and structure of the left atrium.

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Keywords: Giant left atrium; Left atrium volume reduction; Atrial fibrillation; Maze procedure

1. Introduction

The exact etiology of giant left atrium (GLA) remains unknown, but a strong association occurs with chronic rheumatic mitral valve disease. Other conditions such as left ventricular failure, chronic atrial fibrillation, and left-to-right shunts such as patent ductus arteriosus and ventricular septal defects can also lead to left atrial enlargement [1—4], but this is rarely excessive.

According to Di Eusanio et al. [5], about 19% of patients requiring an operation for a mitral valve disease had GLA. GLA patients always have a long history of mitral valve disease and atrial fibrillation, and very often present with hemodynamic and/or respiratory complications, as well as atrial thrombus formation [3,5—7]. According to some studies, the size of the left atrium is a predictor of sudden death [8—11]. Analysis of data from the Framingham study showed that an increase in left atrial chamber in the presence of atrial fibrillation was associated with an increased risk of stroke and sudden death [12]. Thus, GLA poses a significant mortality risk and therefore once the existence of GLA is established it needs to be evaluated, with intention to treat if possible.

Currently, there is no consensus regarding the management of GLA during mitral valve surgery. Most surgeons fix the mitral valve, and do little to an oversized left atrium. Others occlude the left atrial appendage [1,4,5,12]. We believe that a good proportion of surgeons think that successful mitral valve surgery alone will result in the eventual remodeling of the left atrium and size reduction. In this article, we shall review the available literature and provide evidence regarding the pathophysiology, indications for intervention and surgical management of GLA. Our intention is to summarize and review GLA in the context of contemporary mitral valve and arrhythmia surgery.

2. Background history

Hewett was the first to describe GLA in 1849 [4]. He reported autopsy findings of an ‘aneurysmal dilatation of left atrium with thickening and contraction of left atrioventricular opening’ in the context of mitral valve disease [13]. Few reports exist after 1849 and the next report published was in 1967 when the first successful attempt to manage GLA in a symptomatic patient was performed [14]. The authors
3. Definition of GLA

There have been several definitions ranging from 6 to beyond 10 cm in diameter [15—18]. However, more recent reports follow the definition by Kawazoe et al.: GLA is characterized by the following two echocardiographic findings: (a) large left atrium depicted by M-mode ECHO with diameter >65 mm, and (b) left ventricular postero-basal wall bent inward and lying between the dilated left atrial cavity, and left ventricular cavity. The postero-basal segment usually bends more than 30 mm (in the long axis view on the 2-mode ECHO) after the onset of diastole [3,15] (Fig. 1D) [15—18].

3.1. Etiology

GLA is the end product of severe and prolonged pressure and volume overload, occurring mainly during mitral insufficiency, stenosis, and rarely in mitral valve prolapse alone [19,20]. Willis Hurst pointed out that GLA is closely related to rheumatic mitral valve regurgitation or mixed mitral disease with predominant regurgitation, and does not occur in patients with mitral regurgitation due to other causes [21]. However, several physicians and surgeons claim that occasionally the left atrium can reach extreme sizes even under normal mitral valve function in both adults and children [4,18,22—24]. Therefore, the hypothesis that GLA is a consequence of chronic pressure alone in the left atrium is still debatable. Some authors postulate that excessive left atrial enlargement is not solely due to mitral regurgitation, but is also due to the quality of the left atrial wall tissue itself [21,14]. In fact, rheumatic pancarditis not only causes mitral valve regurgitation but it also damages the entire heart by weakening its tissues. The diseased left atrial wall therefore is primed to dilate more easily especially when significant valve regurgitation co-exists. For that reason, GLA is rare in patients with non-rheumatic mitral regurgitation [21].

Interestingly, the histological findings from the atrial wall in patients with GLA do not usually reveal Aschoff bodies (fibrinoid necrotic centre found in the myocardium surrounding blood vessels, and other regions of the body) but instead fibrosis with chronic inflammatory findings [4].

3.2. Pathophysiology of GLA

Dilatation of left atrium with mitral valve disease compensates for increased stress by an initial increase in compliance [16]. According to Hurst, pulmonary edema occurs less often in patients with GLA than in patients with mitral stenosis [21]. This reservoir effect of a large left atrium is considered to be beneficial in reducing pulmonary congestion and protecting the lungs from pulmonary hypertension and edema [25,16]. According to Marijon et al. [22], the pressure decay across the mitral valve depends on mitral valve area, the transmitral gradient, as well as the atrioventricular compliance. Atrioventricular compliance implies compliance characteristics of both atrium and ventricle, working as a single unit [22,26]. When the compliance of either of these chambers is normal or near normal, any abnormality of atrioventricular compliance reflects an abnormal compliance of the other [22,27]. In the case of mitral regurgitation, the left atrial size is proportionally related to compliance, and it may explain a near normal pulmonary artery pressure in some patients with chronic severe mitral regurgitation [22,28]. Nonetheless, Plaschkes et al. [4] observed markedly increased left atrial pressure (mean: 20—60 mmHg), and increased pulmonary pressure (30—95/10 to 50 mmHg), in cases of GLA. And so, GLA can be associated with increased pulmonary venous pressure due to elevated left atrial pressure.

Very large left atria provide the substrate for atrial fibrillation [29]. It has been shown that an enlarged LA with diameter >60 mm is a substrate for development and maintenance of AF [30]. This was also shown by experimental studies with isolated perfused rabbit hearts [31]. In this study, the investigators provoked acute dilatation of the atria by volume load and measured the right and left effective refractory periods, as well as the generation of atrial fibrillation by simple premature stimuli. There was a decrease in threshold and an increase in the ability to induce AF from 0% to 100% (drastic shortening of atrial effective refractory period). If the atrial pressure was greater than 10 mmHg, inhibition of atrial wall stress contributed to prompt cardioversion. Another important finding in this study showed that the induced atrial fibrillation in a dilated left atrium contributes to further atrial dilatation. Thus, atrial enlargement can develop as a...
consequence of atrial fibrillation, as in the case of mitral valve disease confirmed by Sanfilippo et al. [32].

Mathematical modeling can help understand the left atrium better. One such example is Laplace’s law

\[ T = \frac{P \times R}{M} \]

where \( T \) is the tension in the walls, \( P \) is the pressure difference across the wall, \( R \) is the radius of the cylinder, and \( M \) is the thickness of the wall and can be helpful in explaining GLA formation. The analogy is similar to the biophysical properties of dilated ventricle. Therefore to create the same pressure during ejection of the blood, much larger wall tension (\( T \)) has to be developed by the atrium initially. The dilated atrium requires more energy to pump the same amount of blood as compared to the heart of normal size. However, as the atrial volume increases with worsening mitral regurgitation in time it goes on to succumb to deformational change limiting Laplace’s law application in chronic mitral regurgitation. It is possible that patients with rheumatic mitral valve disease lose the elastic properties due to myocarditis, so it becomes larger than expected at any given mean left atrial pressure. Matsuda et al. [40] showed that there is a close correlation between the left atrial diameter measured by ECHO or CT, and left atrial volume. Accordingly, for a left atrial diameter of 6 cm, the estimated volume of the atrium exceeds 300 ml [40]. With this increasing volume reported complications of GLA include; thrombus formation due to potential stasis with potential thromboembolic events, arrhythmia (atrial fibrillation), and decreased cardiac output. The hemodynamic derangements are caused by the following: (a) compression of right cardiac chambers and of caval veins (reduced preload) [4,14]. According to Minagoe et al. [41], in patients with mitral stenosis, GLA can obstruct venous return at the IVC orifice, via marked displacement of atrial septum towards the right atrium, and (b) compression of the postero-lateral wall of left ventricle by downward extension of the enlarged left atrium [3,4,7,14,17]. The latter complication is clinically rare. The extracardiac thoracic organs at risk of compression include: (a) bronchial tree (increase of carinal angle >120°) with severe respiratory dysfunction [3,6,7], (b) basal segments of lower lobes [6,17], (c) esophagus with dysphagia, or the hemidiaphragm [3,5,21], and (d) descending thoracic aorta without apparent clinical symptoms or signs [5].

### 3.3. Clinical findings

It is difficult to distinguish GLA symptoms from the conditions giving rise to initial increase in left atrial size. Nonetheless, GLA may be asymptomatic in a minority of cases. Badui et al. [42] reported an asymptomatic GLA with a diameter of 145 mm. When symptomatic, most common manifestations are: arrhythmia, palpitations, chest pain, shortness of breath, and fatigue dyspnea, orthopnea, atrial fibrillation, nocturnal paroxysmal dyspnea, and thromboembolic events. The symptoms in GLA originate from the anatomic relation of enlarged posterior wall of the left atrium pressing the esophagus and airways [43,44]. Konstantinov et al. [45] reported their experience with 40 symptomatic mitral valve disease patients with GLA and demonstrated that the symptoms of GLA in their patients were due to either compression of the postero-basal part of the left ventricle (hemodynamic instability), or constriction of the lumen of the left main bronchus, or compression of the inferior and middle lobes of the right lung (atelectasis, infection, secretion formation). Kawazoe et al. reported on 40 GLA patients, and showed that 23 patients had manifestation of both left ventricular and bronchopulmonary compression (defined as type II), whereas the remaining 17 had isolated left ventricular compression (defined as type I) [3,17]. Hara et al. [40] reported a case of GLA causing stenosis of the left main bronchus, and widening of the tracheal bifurcation, secondary to compression by the left atrium as it always underlies the carinal angle. In a few case reports direct atrial pressure on the esophagus manifested as dysphagia [17,21,43,46]. Rarely, it may compress the left laryngeal nerve, manifested by hoarseness due to left vocal cord paralysis (Ortner’s syndrome) [46,47].

### 4. Diagnosis

Chest radiography is a useful initial screening tool when it shows increased cardiothoracic ratio and features characteristics of an enlarged left atrium. Transthoracic echocardiography is the easiest, most reliable, and least expensive method for diagnosing and monitoring GLA; this should be done at the time of mitral valve assessment (Fig. 1) [3,18,48,49]. However, in cases of severe mitral regurgitation, the auriculo-ventricular compliance is more difficult to evaluate [22]. The CT scan is valuable in diagnosing GLA when symptoms of compression occur [50]. It is reliable not only in confirming the diagnosis, but useful in the evaluation of those patients in whom the symptoms are non-cardiac [50]. Sinatra et al. [6] consider MRI a more reliable tool for a precise estimation size of the atrium and its relations with other adjacent organs (Fig. 2). Overall clinically, when the right atrial diameter measured by ECHO or CT, and left atrial volume. Accordingly, for a left atrial diameter of 6 cm, the estimated volume of the atrium exceeds 300 ml [40]. With this increasing volume reported complications of GLA include; thrombus formation due to potential stasis with potential thromboembolic events, arrhythmia (atrial fibrillation), and decreased cardiac output. The hemodynamic derangements are caused by the following: (a) compression of right cardiac chambers and of caval veins (reduced preload) [4,14]. According to Minagoe et al. [41], in patients with mitral stenosis, GLA can obstruct venous return at the IVC orifice, via marked displacement of atrial septum towards the right atrium, and (b) compression of the postero-lateral wall of left ventricle by downward extension of the enlarged left atrium [3,4,7,14,17]. The latter complication is clinically rare. The extracardiac thoracic organs at risk of compression include: (a) bronchial tree (increase of carinal angle >120°) with severe respiratory dysfunction [3,6,7], (b) basal segments of lower lobes [6,17], (c) esophagus with dysphagia, or the hemidiaphragm [3,5,21], and (d) descending thoracic aorta without apparent clinical symptoms or signs [5].

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border of the cardiac shadow touches the right thoracic wall, the diagnosis of GLA should be entertained among a list of differentials including: right atrial tumors or congenital abnormalities [51], left atrial congenital anomalies [52], tumors of the mediastinum, or even pleural effusion of the right hemithorax [6,53]. Congenital intrapericardial aneurysm of the left appendage is very rare (saccular aneurysm at the left atrial appendage, with three myocardial layers, and normal sized left atrium body) and should be differentiated from GLA [52,54].

5. Indications for surgical management

A review of the literature reveals that most GLA cases are managed at the time of mitral valve surgery. All authors agree that the main indication for its surgical management is the presence of intracardiac or extracardiac compressive symptoms from neighboring organs (see Section 3.3) [3,5,7,17,43–47]. They claim that by reducing the left atrial size, the pressure effect is reduced with a favorable effect on the postoperative course. A second indication in our opinion is the presence of thrombus and a history of thromboembolic events. Left atrial volume reduction can in theory prevent recurrent thrombosis by reducing intrathromboembolic events. Left atrial volume reduction can be classified into three categories: partial plication or unroofing of both inferior and superior atrial walls, and partial autotransplantation of the heart. We summarize the current techniques below.

groups of patients who underwent radiofrequency ablation alone or in combination with left atrial size reduction [9]. Their study endpoint was restoration of sinus rhythm. In the atrial size reduction arm, sinus rhythm was restored in 77.3% of patients, whereas in the group without reduction it was restored only in 61.1% of patients.

Pre- and postoperative echocardiographic left and right atrial dimensions demonstrated that attaining normal atrial size was important in restoring sinus rhythm [33–35].

Finally, the long-term success rate of maze procedure and its modifications is dependent on the size of the left atrium [36]. Chen et al. showed, that preoperative left atrial size and left atrial diameter (cut-off value of 56.25 mm) could predict successful conversion to sinus rhythm. More than 90% of the patients with a preoperative left atrial size <57.8 mm regained sinus rhythm postoperatively [37]. Kosaki et al. [38] showed that when the left atrium diameter was <45 mm, atrial fibrillation was ablated in 100% of the patients, but when the diameter was >85 mm, the success rate was 0%. This has been confirmed again [39]. Yuda et al. [15] examined the effectiveness of maze procedure in restoring normal atrial contraction in patients with or without GLA. They compared two groups of patients after the maze procedure: 19 patients with GLA (group A), and 32 patients with normal-size LA (group B). Regular rhythm was restored in 53% and 81%, respectively, and effective atrial contraction was observed in 21% and 66%, respectively.

Most surgeons believe that successful mitral valve surgery alone will result in the eventual reduction of left atrial size as the volume and mean atrial pressure decline. In our opinion this is not true. One major reason is that the changes observed in the left atrium of patients with GLA are quite considerable and sometimes irreversible. Choo et al. [58] compared GLA patients to another group of patients that had an atrial diameter of less than 65 mm, who underwent the maze procedure. They demonstrated that in both groups the size of the left atrium was reduced early on postoperatively, but no further reduction was observed for the next 2 years on follow-up, suggesting that these changes may not be sustained. However, Hagihara et al. [59] reported the early and long-term surgical results and changes in the left atrial dimension among 30 GLA patients. They observed that the diameter of LA decreased significantly from 69.0 ± 8.5 mm to 53.7 ± 9.1 mm shortly after surgery, and maintained at 5 years of follow-up. As shown by these studies, the reduction in size is not significant and probably does not alleviate the symptoms caused by GLA. The most important point to note here is that despite the mitral valve surgery, the enlarged left atrial size is unlikely to reduce in size without direct surgical management of GLA.

6. Surgical management

The current methods for left atrial volume reduction may be classified into three categories: partial plication or excision of inferior atrial wall, partial plication or excision of both inferior and superior atrial walls, and partial autotransplantation of the heart. We summarize the current techniques below.
6.1. Partial plication or resection of inferior atrial wall

6.1.1. The classical wall plication \([3,8,14,16,18,43,60]\)

This dates back to Dr Danielson’s work \([14]\). After the classical left atriotomy, he occludes the left atrial appendage and plicates the inferior (or posterior) wall of left atrium. Fugita et al. \([60]\) in 1982 described the method of para-annular plication of the inferior atrial wall for the treatment of patients with GLA. They claimed that in some of the cases of severe mitral valvular disease and GLA, compression and bending of the basal portion of the posterior wall of the left ventricle occurs and is accompanied by paradoxical movement. The para-annular plication technique stemmed from repeated observation that the strut of the mitral prosthetic mitral valve was leaning against the ventricular septum and the inflow stream of blood was directed towards the left ventricular outflow tract. Kawazoe et al. \([3]\) in 1983 provided a full description of plication technique in 30 patients. Other significant reported studies with the same method are that of Isomura et al. \([16]\) in 38 patients and of Schrerer et al. \([8]\) in 27 patients.

6.1.1.1. Technique. Following atriotomy along the interatrial groove, para-annular plication parallel to the posterior mitral annulus approximately 10–20 mm is performed (Fig. 3). The posteroinferior wall of left atrium between the ostia of inferior pulmonary veins and the posterior mitral annulus is plicated in a semilunar fashion. It is plicated first with stay-sutures, at a distance 20–30 mm from each other, and 15–20 mm from the ostia and 10 mm from the annulus. Then, a second row of continuous suture (over-and-over) is fashioned. When GLA compresses the bronchus, a superior plication is performed alone or in combination with the para-annular plication (Fig. 3). The superior plication is performed in the area between the right and left pulmonary veins, and then circumferentially around the left pulmonary veins reaching the mitral annulus. Disadvantages of these techniques include: (a) reduction of the left atrial posterior wall only, (b) lack of releasing the adhesions of posterior wall to mediastinal organs, (c) the from inside plication technique may injure mediastinal structures due to its blind process.

6.1.2. Mercedes-plasty of the inferior wall

Dzemeshkevich et al. \([61]\) combined GLA-plasty with preservation of mitral valve apparatus during MVR.

6.1.2.1. Technique. After exposing the mitral valve through the interatrial groove, the inferior wall of left atrium between the four ostia of pulmonary veins is plicated. During dissection the authors said to remain more laterally on the left side to include the left atrium in the plication and the inner part of the left atrial appendage. The two lateral branches of plication are situated between the inferior pulmonary veins and the posterior mitral annulus (Fig. 3). All three sutures are tied up to each other in the center of the inferior wall, slightly above the line, between the ostia of inferior pulmonary veins.

6.1.3. Triangular resection of the inferior wall

This was described by Sinatra et al. \([6]\).

6.1.3.1. Technique. The left atrium is exposed through an incision parallel to the atrial septum, posterior to the IVC and superior to the atrial roof and to the left fibrous trigone of the mitral valve. Three triangular segments of posterior left wall are excised (Fig. 3). The first triangle has its base between RSPV and behind the retracted SVC whilst its apex is between RIPV and LIPV. The second has its base between RSPV and RIPV, and its apex towards the ostium of LIPV. The third has its base between RIPV and behind the IVC, and its apex towards the LIPV and posterior mitral annulus. The three residual defects of the atrial wall are then approximated with three continuous sutures. The left atrial appendage is closed from outside. The method has all the already above mentioned disadvantages of posterior wall plication. The main advantage is that it is not blind, and the surgeon has direct vision to free any adhesions, and avoid injury behind the posterior wall. Given the extent of incision with this technique, there may be a theoretical advantage in that it may further interrupt the wavelets-circuits around the pulmonary veins adding to the beneficial effects of inhibiting the propagation of atrial fibrillation foci. Further studies need to prove this.

Apart from the already mentioned disadvantages, triangular resection of the inferior wall allows the surgeon to directly visualize the posterior mediastinal structures.

6.2. Partial plication or resection of both, inferior and superior atrial walls

6.2.1. Plication of superior and inferior atrial walls

This technique combines both superior and trans-septal approaches \([62]\). The postero-inferior wall as in the previous method and the roof of the left atrium are plicated.

6.2.1.1. Technique. The IVC is cannulated low, and SVC is directly cannulated as far distal as possible. The right atrium is opened obliquely reaching the IVC. The interatrial septum is open longitudinally through the fossa ovalis. Next, the incision is extended caudally to the inferior limbus, and
through the roof of left atrium to the left auricle in a cephalad manner (Fig. 4). The postero-inferior wall of left atrium between posterior mitral annulus and ostium of the inferior pulmonary veins is plicated in a semilunar fashion similar to that reported by Kawazoe et al. [3]. After mitral valve replacement or repair, the plication is continued along the incision line, from the caudal border of the appendage, and upward through the cephalic part of the atrium.

6.2.2. Spiral resection of left atrial wall

This method was introduced by Sugiki et al. [7], and is based on a combined superior trans-septal approach to the mitral valve which was also proposed independently by Berreklouw et al. [63] in 1991.

6.2.2.1. Technique.

The IVC is cannulated low, while the SVC is directly cannulated. The right atrium is opened obliquely and the septum longitudinally at the fossa ovalis to enter the left atrium (Fig. 5). The incision is then extended cranially through the roof of the atrium, and then laterally towards the left atrial auricle. Thereafter, the incision is extended to the postero-inferior wall of the left atrium, between both ostia of inferior pulmonary veins, and 2—3 cm from the mitral annulus. The incision is extended cranially, parallel to the atrial groove reaching the left atrial wall between the right superior pulmonary vein and superior vena cava. The end result is a spiral-shape incision extending from the atrial septum to the right lateral wall of the left atrium through the roof, lateral, posterior and inferior wall of left atrium. Along this long incision, a strip, 3—4 cm in width of left atrial wall is excised. The cut margins are re-approximated using a non-absorbable stitch. The advantages of these methods include those already mentioned above. The main disadvantage is the risk of bleeding from the anastomotic site, and possible atrioventricular conduction abnormalities.

6.3. Partial heart auto-transplantation

This was introduced by Lessana et al. [64] in 1999.

Fig. 4. Yuasa et al.’s [62] method involves exposure of the left atrium and mitral valve, and plication of the inferior wall, as well as of the superior wall along the incision-line.

Fig. 5. Schematic presentation of Sugiki’s operation. The mitral valve is exposed through the Berreklouw trans-septal method. (A) The incision is extended in the posterior wall, or in the direction of left auricle (line 1), or between right and left pulmonary veins (line 2). (B) The incision is further extended in the entire inferior wall and comes around to reach the right side atrial wall (C) [7].

6.3.1. Technique

CPB is instituted following bicaudal cannulation. After cross-clamping and cardioplegia infusion, the superior vena cava, aorta, and pulmonary artery are detached. The heart is then displaced superiorly and to the left. The left atrium is completely incised and a strip with a width of 3—5 cm is resected circumferentially. Finally, the diameter of left atrium is decreased to 5 cm. Mitral valve replacement is easily performed in this position, and the pulmonary artery,
aorta, and superior vena cava are re-attached. A further modification was proposed by Garcia-Villarreal et al. [65] 2 years later. They called their method a functional-anatomic unit concept because they consider LA and MV as a functional-anatomic unit. It is more conservative than the Lessana’s method because they transect only the SVC. The advantages of these two methods are that they obtain significant reduction of left atrium size, an excellent exposure of mitral valve, and may interrupt the wavelets-circuits around the pulmonary veins. The disadvantages include: (a) non-plication of interatrial septum (the anterior wall of left atrium), (b) involvement of non-diseased anatomic structures like IVC (for the Garcia-Villarreal modification), pulmonary artery, and aorta with potential for mortality and morbidity, (c) the transection of SVC and its re-approximation can produce brain edema, and (d) prolonged myocardial ischemic time.

7. Results of GLA surgery

The co-existence of GLA associated with mitral valve disease has been reported as a significant risk factor in mitral valve surgery with surgical mortality ranging from 8% to 23% [2,3]. Currently, high mortality rates following mitral valve surgery above 5% are not acceptable with increased safety of [2,3]. Currently, high mortality rates following mitral valve surgery with surgical mortality ranging from 8% to 23% [2,3]. Currently, high mortality rates following mitral valve surgery with surgical mortality ranging from 8% to 23% [2,3].

8. Conclusions

The literature provides evidence that the left atrium should be reduced in size whenever it coexists with operable mitral valve disease, as well as in cases of maze procedure for chronic atrial fibrillation. However, the quality of the observational data cannot justify any strong recommendations to our patients especially when mortality can be high. The simplest modality for size reduction is plication of the inferior left atrial wall. The plication (or partial excision) of superior wall is more radical and carries a higher greater risk for bleeding and atrioventricular node blockade. Left atrial remodeling can reduce pressure symptoms on neighbouring organs, and improve early postoperative course, with the ability to restore and maintain sinus rhythm. Cardiovascular surgeons should carefully consider the contemporary benefits of the various surgical options when managing GLA.

References


