Review Article

Surgical Ventricular Restoration (SVR) for Ischemic Heart Failure

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Abstract:

Surgical ventricular restoration (SVR) is a surgical procedure developed in an attempt to reverse the negative remodeling that occurs following myocardial infarction. SVR has evolved from a treatment for ventricular aneurysms and has become a treatment of ischemic heart failure.SVR procedure is usually performed after coronary artery bypass grafting (CABG) and may proceed or be followed by mitral valve repair or replacement. This article will review the procedure.

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Introduction:

Despite continued advances in medical and surgical approaches for patients with ischemic cardiomyopathy, congestive heart failure (CHF) remains a growing cause of morbidity and mortality in the United States and worldwide.[1,2] There have been few medical and surgical options for patients with end-stage CHF who are ineligible for heart transplantation.[3-5] However, there are several surgical therapies under investigation that promise to reverse or restore the remodeled left ventricle toward its more normal shape and function. This review focuses on surgical ventricular restoration (SVR).

Ventricular remodeling is a natural process after infraction. Following infarction, the left ventricle (LV) undergoes a well – described process of ventricular remodeling. Left unchecked, this process can lead to a progressive dilation of the ventricle, resulting in an increase in the end diastolic diameter and volume, an increase in LV wall stress and oxygen demand, a loss of the ventricle's natural elliptical shape with the development of a more rounded contour, the development of mitral insufficiency, and ultimately a worsening of global systolic function.[6,7] The development of mitral regurgitation is due to factors specific to ventricular remodeling as well as unrelated leaflet issues. The first factor related to the remodeling process is annular dilation due to global ventricular enlargement. The second is restricted leaflet motion and reduced coaptation due to the global LV dilation or involvement of the papillary muscles with the infarction itself. These factors combine to prevent leaflet coaptation of limit it in the proper plane, resulting in central regurgitation. Superimposed leaflet pathology can worsen the functional regurgitation. Postinfarction dysfunction is further affected by both the electrical and mechanical dysynchrony that develops following infarction.

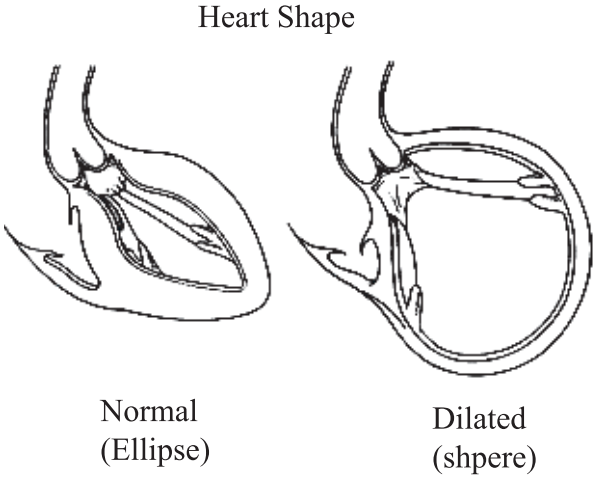

Fig.1: *Left ventricular shape. The normal LV has an elliptical shape, with a well-defined apex. The dilated LV becomes spherical, and loses its oblique fiber angle orientation.*

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The prognosis of patients with ischemic cardiomyopathy is related to the size of the LV and the impact that remodeling has had on the function of the remote noninfarcted zones.[7] Progressive thinning and dilation of the remote areas leads to the development of a spherical rather than the normal elliptical shape of the heart. Clinically, the ongoing remodeling generates a progressive reduction in contractile force, thus worsening congestive heart failure and ultimately leading to death.[8, 9]

The paradigm of progressive left ventricular dilatation is grossly oversimplified but simply understood through Laplace' Law. Left ventricular wall stress/tension (ä) is proportional to the radius (r) and pressure (P) within the left ventricular chamber and inversely proportional to left ventricular wall thickness (T) with average wall stress estimated by the following equation: ä = rP/(2)T. Whether approaching the concept from the level of the individual myocyte (tension-length) or from the level of the left ventricular chamber (pressure-volume), increasing cardiac myocyte stress becomes an impediment to effective contraction. Therefore, a solution is to reduce left ventricular chamber radius/volume and thereby reduce myocardial wall stress. Surgical ventricular restoration was initially advocated for patients with post infarction LV aneurysms, with discrete akinetic/dyskinetic areas and remaining areas of contractile myocardium. Later, it was done in patients with dilated hypokinetic ventricles where the benefits were seen due to decrease in wall stress by decreasing the ventricular radius (Laplace' law). And, it is really under the auspices of left ventricular aneurysmectomy, technically refined most notably by Cooley, Jatene and Dor, that volume reduction surgery had a beginning in the early 1970's.[10-12] Vincent Dor's approach was unique in that it approached akinetic areas and dyskinetic aneurysms equally. He latter began to apply this technique to treat congestive heart failure and altered the way in which changes in ventricular morphology are approached. The concept off reconstructing the ventricle to a prescribed size based on the patient's body size was introduced by Dor and popularized by Menicannti.[12]

Additional technical approaches that merit mention include a linear closure and septoplasty approach described by Mickleborough and a concentric purse-string or "cerclage" technique followed by linear closure utilized by McCarrthy.[13,14] The end result of two decades of surgical innovation is four techniques used for ventricular remodeling procedures today.

Coronary artery revascularization has been shown to improve survival in both normal and abnormal ventricles. Improved blood flow cannot improve the function off the scarred, noncontractile areas of the heart. Furthermore, it does not change the evident dyssynchrony. Surgical techniques have been developed to arrest the progression and reverse the morphologic changes induced by the pathologic process of postinfarction ventricular remodeling. Such techniques are commonly referred to as surgical ventricular remodeling (SVR). The goal of SVR is to reduce the enlarged spherical hearts so as to improve cardiac function.SVR is often performed in conjunction with coronary artery revascularization and mitral valve repair / replacement.[15]

Definition and unification of nomenclature: Surgical ventricular restoration (SVR) is a procedure designed to restore or remodel the left ventricle to its normal, spherical shape and size in patients with akinetic segments of the heart, secondary to either dilated cardiomyopathy or post infarction left ventricular aneurysm. The SVR procedure is usually performed after coronary artery bypass grafting (CABG) and may proceed or be followed by mitral valve repair or replacement and other procedures such as endocardectomy and cryoablation for treatment of ventricular tachycardia. A key difference between surgical ventricular restoration and ventriculectomy (i.e., for aneurysm removal) is that in SVR the ventricle is reconstructed using patches of autologous or artificial material that are placed to close the defect while maintaining the desired ventricular volume and contour.[17]

Additionally, SVR is distinct from partial left ventriculectomy (i.e., the Bastista procedure[16]) which does not attempt to specifically resect akinetic segments and restore ventricular contour. The SVR procedure may also be referred to as surgical ventricular remodeling, surgical anterior ventricular endocardial restoration (SAVER) or the Dor procedure after Vincent Dor, MD. Dr. Dor

pioneered expansion of techniques for ventricular reconstruction and is credited with treating congestive heart failure patients with SVR in conjunction with CABG.[17]

Goal in SVR is to approach the spherical chamber and reconstruct a more normal elliptical ventricular form, but we must unify our language to use a common term to achieve this goal. Guidelines are garnered from the word "remodeling", that is generally accepted as (a) an adaptive alteration in ventricular shape and size after infarction, and (b) the morphologic spherical size and shape change that is the underpinning of congestive heart failure in dilated hearts. Webster's dictionary describes remodel as "take away from normal". Medical and electrical interventions that beneficially improve function are called useful by "reverse remodeling", when form returns toward normal. Current surgical interventions toward this morphologic goal are called reconstruction, rebuilding, and restoration. Webster's dictionary also defines "restoration" as "bring back toward normal", thereby introducing the antonym of remodel. Since "restoration" includes the surgical goal in its definition, this phrase adapts to the common objective and should replace the current reverse remodeling, reconstruction, and rebuilding terminology.[18]

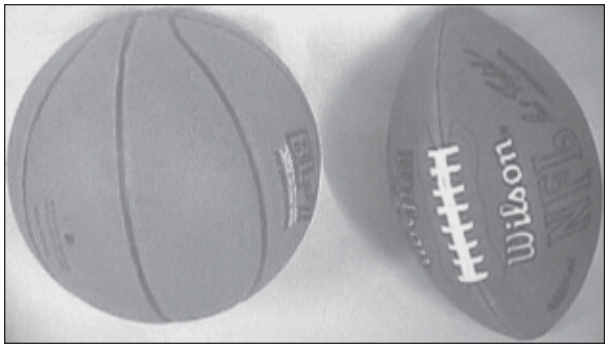

Fig.-2: *The athletic corollary to shape changes between the normal, elliptical football and the globular, spherical basketball shape in heart failure. The surgical objective is to remake the elliptical football. (Reproduced with permission of Hisayoshi Suma, MD).*

Who really benefits:

Classically, patients are candidates for SVR if they have had an anterior myocardial infarction, have a large area of akinesis or dyskinesis, and have clinical evidence of CHF. Ideal candidates have akinesis/ dyskinesis in the anteroseptal area, have retained function of the basilar and lateral portions of the heart, and have good right ventricular function.[15]

Specific characteristics of patients who have successfully undergone SVR are-

- Previous anterior myocardial infarction (either Q or non-Q) as evaluated by electrocardiogram or cardiac magnetic resonance.
- LV dysfunction with dilatation of the ventricle and regional asynergy (either akinetic or dyskinetic). When LV asynergy is severe and diffuse, SVR can be performed only if regions remote from the scar have some degree of contraction detectable at rest or under inotropic stimulus (like dobutamine test).
- HF symptoms are the first indication for SVR, but also patients presenting with ventricular arrhythmias and/or angina who need surgical revascularization represent an indication for SVR (if the previous conditions are present) to avoid further dilatation.
- For patients who are asymptomatic despite postinfarction LV dysfunction, we suggest that serial echocardiographic studies be performed to detect the first signs of deterioration (i.e, LV progressive enlargementor decline in EF).

The following are contraindications-

- Severe right ventricular dysfunction (biventricular dilated cardiomyopathy) (absolute).
- Severe pulmonary hypertension not associated with MR (relative).
- Severe regional asynergy without LV dilatation (absolute).
- Restrictive diastolic pattern associated with high functional class and MR (absolute)[19].

Surgical procedure:

Surgical management, until recently, was directed toward the underlying pathology of coronary occlusion and the secondary mitral insufficiency that evolves as the heart dilates. Coronary grafting reperfuses the responsible vessel and mitral repair fixes the valve that loses effectiveness from dilation, but surgeons do not systematically

approach the ventricle, the underlying cardiac structure that causes progressive dilation and produces this wide range of symptoms. The disease of congestive heart failure is considered the "Triple V"- vessel, valve and ventricle,[18] and this review issues will define how the combined entity is approached.

Surgical ventricular restoration is commonly performed in conjunction with CABG. Standard arterial and venous cannulation techniques, in conjunction with myocardial protection protocols, are utilized during this surgical procedure. If mitral valve repair or replacement is a strong possibility, cannulation of both inferior and superior cava is recommended. The left ventricle can be vented directly, through the right superior pulmonary vein, or through the aortic root.

Once the patient is on cardiopulmonary bypass, the sequence of procedures performed is dependent upon personal preference. Most commonly, CABG is performed first. Once this is accomplished, the SVR or the mitral valve replacement can be performed next. The surgeon performs the mitral valve replacement to avoid potential disruption of the ventricular repair with retraction.

With the heart vented, the left ventricle will often collapse, demonstrating the area of thinned out scar. This does not always happen, and the absence of collapse does not contraindicate SVR. An incision is made into the anterior wall of the left ventricle through the area of scar. This is extended to the apex and proximally parallel to the course of the left anterior descending coronary artery. Retention sutures are placed to aid in achieving and maintaining exposure.

The ventricle is inspected. Any thrombus is removed. If there is suspicion that there is a large thrombus burden in the left ventricle preoperatively, it may be beneficial to perform a left atriotomy, as if approaching the mitral valve, and placing a sponge into the left atrium before opening the ventricle. This will prevent any particulate matter from falling through the mitral valve into an irretrievable location in the pulmonary veins. This is later removed through the mitral valve and out the left ventricle after the ventriculotomy is performed, the thrombus is removed, and the ventricle is irrigated.

During inspection of the ventricle, the extent as well as the transmural nature of the scar is noted. A palpable transition zone between infarcted and non-infarcted muscle is often palpable. This is particularly so with full thickness infarctions. The presence or absence of a transition zone is not known to be of clinical significant.

A ventricular sizing device, or mannequin, is used to help reconstruct the ventricle. The ventricular sizing device is inserted into the ventricle, seated on the mitral valve annulus, and the site of the new apex is selected. This is one of the most important parts of the operation to ensure a properly sized and shaped ventricle. The device is selected based on a volume of 50–70 cc/m^2 body surface area, with 60 cc/m^2 being chosen for most patients. Obese patients will have the volume titrated down to 50 cc/ m^2, and cachetic patients will have the volume titrated up to 70 cc/ m^2. Once this is done, a purse string of 2-0 polypropylene suture is placed to define the limits of the new anterior wall. This is commonly referred to as the "Fontan" stitch. The stitch is begun at the new apex and is run cephalad across the septum using the anterior margin of the filled sizing device as a guide. It is recommended to include no more than one half of the septum, regardless of the amount of scar left behind. An alternative method is to use a marking pen to mark the endocardium with the device filled and then deflating it while the sutures are placed. The suture is carried across the anterior wall and down the lateral wall to the new apex. Deep partial thickness bites into tough scar is recommended. If two or more purse strings are used, they should be placed equidistant from the previous purse string. This is not a necessary step. The purse string is tied defining the outline of the new distal anterior wall. The sizing device is left inflated while the purse string is tied. The purse string should not be tied in an attempt to close the ventriculotomy except in the rare instances of very small reconstructions. Doing so may cause the suture to pull through the endocardium, especially in patients without well-formed scar tissue.

The anterior wall is reconstructed utilizing a variety of techniques. A patch of Dacron (polyester; Invista, Wichita, KS) is used if the remaining defect is >2–3 cm long. An ovalshaped patch is cut to the

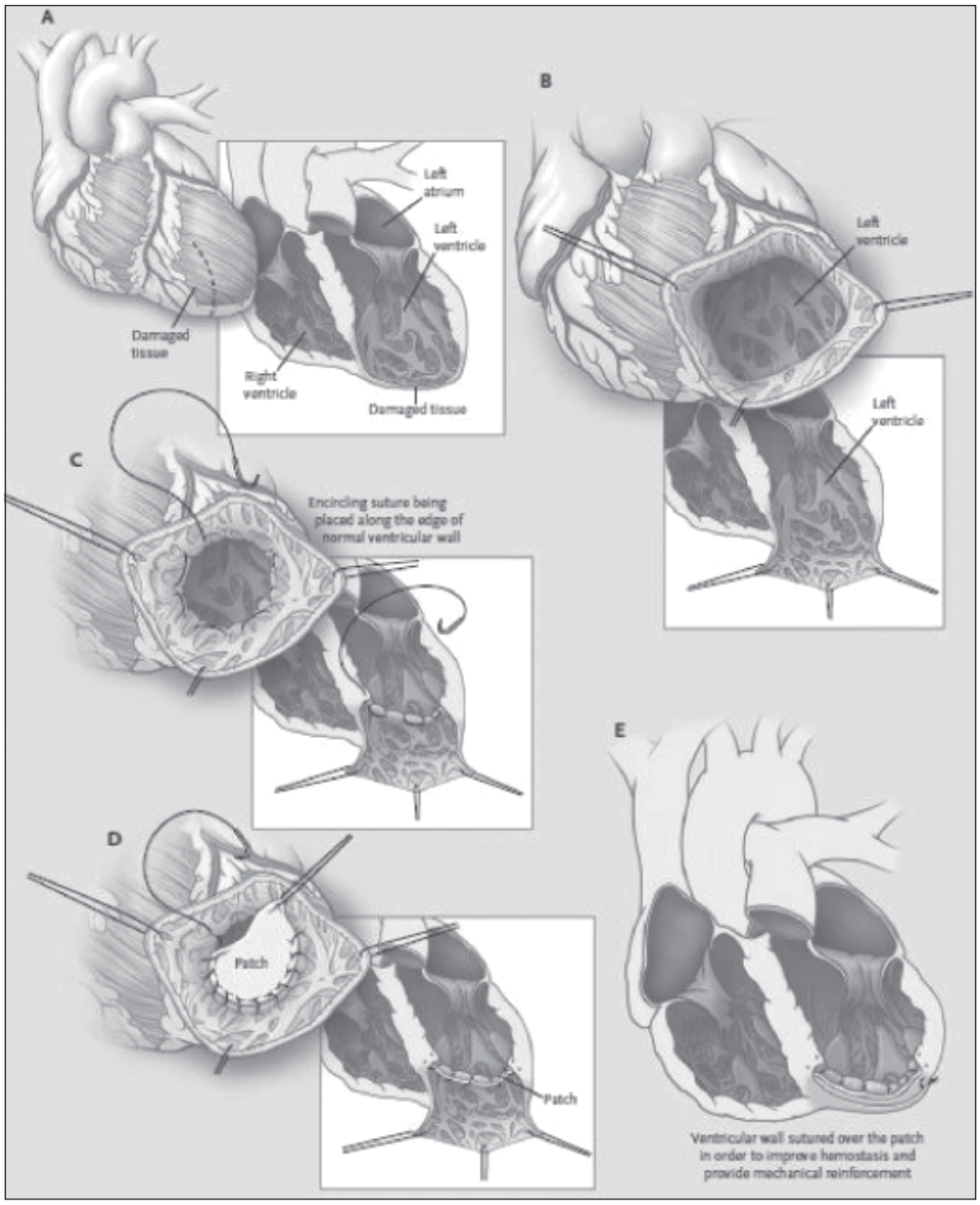

Fig.-3: *Surgical Ventricular Restoration- An incision is made through the area of scarring in the left ventricle (Panel A), which is opened to identify the boundary between damaged and healthy myocardium (Panel B). A purse-string suture is placed to encircle the scar, and the healthy portions of the ventricular wall are brought together (Panel C). The suture is tightened or a patch is placed (Panel D), and the scarred sections left outside the chamber are closed (Panel E).*

appropriate size and is sutured in place closing the defect. Care is made to place the patch sutures around the anterior purse string. The patch may be sutured using a continuous or interrupted technique. The sizing device is often left in place while half of the patch is sewn into place and is deflated and removed after this. Prior to completing the patch, the left ventricular vent is shut off, allowing the ventricle to fill with blood and forcing air to escape the ventricle through the partially completed closure. After the patch is sutured into place, it is checked for hemostasis. In the event of a small residual defect too small for patching, a linear closure is performed superficial to the patch or purse string. Horizontal mattress sutures, buttressed with bovine pericardium, are

usually used as a first layer of closure. With well-formed scar, the pericardial strips are not used. The second layer is a continuous running stitch of 2-0 polypropylene. If the defect is moderate in size, but not large enough for patching, a series of anterior purse string sutures are placed to narrow the ventricular defect before placing the mattress sutures.[20]

The mitral repair, if necessary, can be done at any time utilizing any technique preferred. An intraventricular repair as described by Menicanti, et al.[21] is done through the ventriculotomy before performing the SVR**.** Indication for mitral repair is moderate to severe mitral regurgitation (grade ≥3/4) or mitral annulus dilatation (≥38 mm) if mitral regurgitation is mild (grade ≤2). The technique of mitral repair is posterior annulus suture through the ventricular opening in all cases. A double arm stitch running from trigone to trigone is tied on a 26-mm sizer, inserted through the mitral orifice, to undersize the mitral area.

A word should be mentioned about performing the SVR with the heart beating. One advantage is that poorly functioning, often ischemic ventricles are not further injured during the period of cardiac arrest. Another is that the degree of mitral regurgitation and the result of mitral repair can be assessed in a beating heart, which is felt by some to be advantageous. A final advantage is it allows the border between contracting and non-contracting segments of the heart to be seen more clearly from within the ventricle and may aid in performing the SVR. The disadvantage is a much more difficult time in placing the sizing device on the mitral annulus and keeping it there to allow accurate placement of sutures. This disadvantage can be partially compensated for by tracing the outline of the margins of the sizer on the endocardium and then deflating the sizer. No good evidence supports any approach; however, the novice may be aided with cardiac arrest until experience is gained.[20]

Discussion:

The published literature on ventricular remodeling is largely single-center studies from centers with extensive experience with such techniques. These experiences have demonstrated success of the operation in improving measured parameters of function while reducing the size of the remodeled ventricle.[22- 24] More recently, the importance of reestablishing an elliptical shape has been recognized and appreciated.[22-25]

Data from two randomized, controlled trials are available. Ribeiro and colleagues randomized 74 patients with viable anterior wall myocardium following anterior myocardial infarction to coronary artery bypass (CABG) alone or CABG plus surgical ventricular restoration (SVR).[26] Indications for revascularization included angina, heart failure or both. Patients were randomized on a 1:1 ratio. Patients randomized to the SABG + SVR arm received endoventricular reconstruction developed by Dor. Patients in both groups were followed for two years. After surgery both groups exhibited improvement in LVEF: CABG only patients improved from 32 to 40.1 and CABG + SVR improved from 34.5 to 44.2. After two years, there was a further significant difference in LVEF between the groups; 41 versus 49 respectively. The two-year survival was not significantly different between the groups. The CABG + SVR group had significantly improved freedom from heart failure compared with the CABG only group ($p = 0.016$). As the authors noted in their discussion and as noted in an accompanying editorial, (13, 14) while SVR provided significant improvement in left ventricular volumes compared to CABG alone, the number of patients was small and the follow-up short term. Recurrence of heart failure is likely to occur at higher rates after more time has passed. The authors further stated that it is not clear whether SVR can revert or stop the remodeling processes after myocardial infarction.

Jones and colleagues randomized 1000 patients to either CABG alone (n = 499) or CABG with SVR (n = 501). At median follow-up of 48 months, reduction in end-systolic volume index remained significantly greater in the SVR group than in the CABG alone group (19% and 6%, respectively). There was no between-group difference for the primary endpoint, which was a composite of death from any cause and hospitalization for cardiac causes.[27]

The remainder of the published literature consists primarily of case series reports and retrospective reviews from single centers with the exception of publications from the multi-center RESTORE

Group (Reconstructive Endoventricular Surgery, returning Torsion Original Radius Elliptical Shape to the LV). The RESTORE Group is an international group of cardiologists and surgeons from 13 centers that has investigated SVR in over 1000 patients with ischemic cardiomyopathy following anterior infarction in the past 20 years.[28-34] The following discussion summarizes a representative sample of some of the reports on SVR.

Athanasuleas and colleagues from the RESTORE Group, reported on early and 3-year outcomes in 662 patients who underwent SVR following anterior myocardial infarction during the period of January 1998 to July 2000.[31] In addition to SVR, patients also concomitantly underwent CABG (92%), mitral repair (22%), and mitral replacement (3%). The authors reported overall mortality during hospitalization was 7.7%; postoperative ejection fractions increased from 29.7% ± 11.3% to 40.0% ± 12.3% (P <. 05). The survival rate and freedom from hospitalization for heart failure at 3 years was 89.4% ± 1.3% and 88.7% respectively. In a separate publication on 439 patients from the RESTORE Group, Athanasuleas and colleagues reported outcomes improved in patients with lower patient age, higher ejection fractions and lack of need for mitral valve replacement.[34]

Lorenzo Menicanti from the Cardiac Surgery Department, San Donato Hospital, Milano, Italy reported 1161 patients had anterior SVR with or without CABG and with or without mitral repair/ replacement From June 1989 to October 2005. It describes the largest single-center series of patients with anterior SVR, and it is the largest that extends follow-up observation beyond 5 years. Major findings from their results are as follow: (1) Significant volume reduction and improvement in EF are observed at the predischarge echo evaluation, and this improvement is still significant at late follow-up, despite a slight but significant increase in volumes and in MR, in respect to early after surgery. The late increase in volume could be ascribed to loading conditions that are lower when the patient is hospitalized, at rest. (2) Clinical status, as evaluated by NYHA functional class, is significantly improved. (3) Eighty-two percent of patients are free from cardiac rehospitalization. (4) Very few patients required an internal cardioverter device and biventricular pacing implantation. (5) Long-term survival is promising also in patients with depressed cardiac function and high functional class.[19]

Mickleborough and colleagues reported on 285 patients who underwent SVR by a single surgeon for class III or IV congestive heart failure, angina or ventricular tachyarrhythmia during the period of 1983 to 2002. In addition to SVR, patients also concomitantly underwent CABG (93%), patch septoplasty (22%), arrhythmia ablation (41%), mitral repair (3%), and mitral replacement (3%). SVR was performed on the beating heart in 7% of patients. The authors reported hospital mortality of 2.8%; postoperative ejection fractions increased 10% ± 9% from 24% ± 11% (p<.000) and symptom class in 140 patients improved 1.3 ± 1.1 functional class per patient. Patients were followed up for up to 19 years (mean, 63 ± 48 months) and overall actuarial survival was reported as 92%, 82%, and 62% at 1, 5 and 10 years respectively. The authors suggested wall-thinning should be used as a criterion for patient selection.[35]

Bolooki and colleagues reported on 157 patients that underwent SVR by a single surgeon for class III or IV congestive heart failure, angina, ventricular tachyarrhythmia or myocardial infarction using 3 operative methods during the period of 1979 to 2000. SVR procedures consisted of radical aneurysm resection and linear closure (n=65), septal dyskinesis reinforced with patch septoplasty (n = 70), or ventriculotomy closure with an intracavitary oval patch (n = 22). The authors reported hospital mortality of 16%. The mean preoperative ejection fraction was 28% ± 0.9%. Patients were followed up for up to 22 years and overall actuarial survival was reported as 53%, 30%, and 18% at 5, 10 and 15 years respectively. The authors found factors improving long term survival included SVR with intraventricular patch repair and ejection fraction of 26% or greater preoperatively.[36]

Sartipy and colleagues reported on 101 patients who underwent SVR using the Dor procedure at a single center for class III or IV congestive heart failure, angina and ventricular tachyarrhythmia during the period of 1994 to 2004. In addition to SVR, patients also concomitantly underwent CABG (98%), arrhythmia ablation (52%) and mitral valve

procedure (29%). The authors reported early mortality (within 30 days of operation) was 7.9%; left ventricular ejection fraction increased from 27% ± 9.9% to 33% ± 9.3% postoperatively. Patients were followed up 4.4 ± 2.8 years and overall actuarial survival was reported as 88%, 79%, and 65% at 1, 3 and 5 years respectively.[37]

Another article reported on the contemporary performance of SVR based on data from the Society of Thoracic Surgeons' (STS) Database. From January 2002 to June 2004, 731 patients underwent procedures at 141 hospitals. The operative mortality was 9.3%; combined death or major complications occurred in 33.5%. The authors commented that further studies of SVR are needed to improve patient selection and procedural performance.[38]

Conclusion:

SVR is a procedure that has evolved from a treatment for ventricular aneurysms and has become a treatment of CHF. It has been shown to improve ventricular size, morphology, LV ejection fraction, stroke-volume index, endocrine markers of CHF, ventricular energetics, ventricular synchrony, and mechanical efficiency. Clinically, it results in improved functional capacity (NYHA class) and an excellent 5-year survival in very sick patients. It is an excellent treatment option in appropriately selected patients with ischemic cardiomyopathy. Where SVR will fit in the armamentarium of a heart failure team will be institution-dependent based on their expertise and experience. Further studies are needed to better define the appropriate patients, the basis of the beneficial response (ventricular physiology versus relief of ischemia), the optimal technique, and the appropriate time to perform the procedure. Much remains to be worked out.

Conflict of Interest- None.

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