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Surgical ventricular reconstruction for ischaemic heart failure: state of the art

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Patients with ischaemic cardiomyopathy and left ventricular (LV) systolic dysfunction represent the highest-risk population with heart failure (HF). The cornerstone of treatment remains guideline-driven medical therapy, which is associated with significant improvement in survival and quality of life. The most commonly considered surgical interventions are coronary artery bypass graft surgery, at times combined with surgical ventricular reconstruction (SVR) and surgery for mitral valve regurgitation. Surgical ventricular reconstruction has been introduced as an optional therapeutic strategy aimed to reduce LV volumes through the exclusion of the scar tissue, thereby restoring the physiological volume and shape and improving cardiac function and clinical status. This review will briefly discuss the rationale to surgically reverse LV remodelling, the technique, and the indications, to the best of our knowledge, coming from the Center with the largest worldwide experience. The evolving data on the role of SVR for the treatment of ischaemic HF will be addressed as well.

Introduction

The estimated population prevalence of heart failure (HF) in the developed world is 1–2%.[1] Heart failure is associated with ischaemic heart disease in a percentage of patients ranging from 46 to 68%[2] and for this population the prognosis is poor. The 5-year survival rate of patients diagnosed with HF is still <50%, and might even be underestimated.[3] Furthermore, patients with ischaemic left ventricular (LV) systolic dysfunction have significantly higher mortality rates than those with non-ischaemic aetiologies.[4]

Research has been very effective in delivering major advances in therapy of ischaemic HF patients, including medical therapy, devices, and surgery.[5] However, despite advances in different therapeutic strategies, the prognosis remains unfavourable. Indeed, HF is a syndrome with a broad spectrum of heterogeneous symptoms and signs caused by cardiac dysfunction and resulting in a wide range of clinical expressions.[6] Treating generically HF syndrome is reductive and misleading: to be really successful, the underlying disease, named LV remodelling, should be addressed and treated.

Left ventricular remodelling: mechanisms and characteristics

Left ventricular remodelling is a complex and dynamic process that may occur after a myocardial infarction (MI), leading to chamber dilatation, altered configuration, and increased wall stress.[7] Left ventricular remodelling usually begins within the first few hours after an MI and results from fibrotic repair of the necrotic area with scar formation, elongation, and thinning of the infarcted zone.[8] Left ventricular volumes increase, a response that is sometimes considered adaptive, associated with stroke volume augmentation in an effort to maintain a normal cardiac output as the ejection fraction declines. However, beyond this early stage, the remodelling process is driven predominantly by eccentric hypertrophy of the non-infarcted remote regions, resulting in increased wall mass, chamber

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enlargement and geometric distorsion.[8] The decline in performance of hypertrophied myocyte, along with increased neurohormonal activation, collagen synthesis, fibrosis, and remodelling of the extracellular matrix within the non-infarcted zone, leads towards a progressive decline in ventricular performance.[9] Left ventricular hypertrophy, dilatation, and contractile dysfunction, left untreated, may progress indefinitely as evidenced by progressive increases in LV volumes (*Figure 1*).

Furthermore, mitral regurgitation (MR) may occur as a consequence of the LV remodelling worsening the prognosis.[10] The papillary muscle displacement, which may occur as a consequence of the LV dilatation, results in tenting of the mitral valve at closure with lack of a proper coaptation, in turn leading to secondary MR. In addition, ventricular dilatation results in annular enlargement, which further increases valve incompetence.

The rationale to surgically reverse left ventricular remodelling

The concept behind the ventricular reconstruction is based on the exclusion of the scar tissue, thereby reducing the ventricle to a more physiological volume, reshaping the distorted chamber, and improving cardiac function through a reduction of LV wall stress in accordance with the principle of Laplace's law. Since LV wall stress is directly proportional to LV internal radius and pressure and inversely proportional to wall thickness, any intervention to optimize this relationship would be beneficial in terms of either improving wall compliance and reducing filling pressure or, as wall stress is a crucial determinant of afterload, in terms of enhancing contractile performance of LV by increasing the extent and velocity of systolic fibre shortening.[11] Furthermore, myocardial revascularization usually combined with the reconstruction of failing ventricles aims to treat the underlying coronary artery disease. Finally, although the matter of functional chronic ischaemic MR, in terms of whether, when and how it should be corrected is still considerably controversial, it should be pointed out that surgical ventricular reconstruction (SVR) offers either the possibility to repair the mitral valve through the LV opening or the potential of

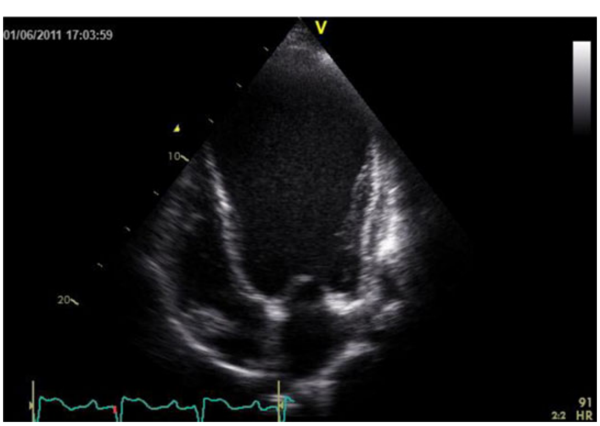

Figure 1 Left ventricular remodelling following a previous anterior myocardial infarction in apical four-champer view.

improving mitral functioning by reducing LV volumes and papillary muscles distance (which is a main determinant of functional MR).[12–14]

Surgical ventricular reconstruction technique

The technique is performed under total cardiac arrest with antegrade cristalloid cardioplegia.[14] Complete myocardial revascularization is performed first with particular attention to revascularize the proximal left anterior descending segment, to preserve the upper part of the septum. After that, the ventricle is opened with an incision parallel to the left anterior descending artery, starting at the middle scarred region and ending at the apex. The cavity is inspected and any thrombus is removed if present. After a careful identification of the transitional zone between scarred and non-scarred tissue, a pre-shaped mannequin (TRISVR™, Chase Medical Richardson, TX) is inserted into the LV chamber and inflated with saline. The size of the mannequin is selected according with the BSA (inflated with 50 mL/m^2). The mannequin is useful in giving the surgeon the correct position of the apex and in maintaining the long axis of the ventricle in a physiological range (7.5/8.5), reducing thereby the risk of sphericalization of the new ventricle. The exclusion of dyskinetic or akinetic LV free wall is performed through an endoventricular circular suture passed in the transitional zone. The ventricle is closed over the mannequin respecting the longitudinal diameter; if the dilatation involves also the inferior wall, a short plication of the inferior wall is performed to avoid amputation of the apex. The final result has to be an elliptical shape of the ventricle; when the dilatation starts close to the aortic valve, a running suture is conducted from the inner of the ventricle over the mannequin towards the apex. The mannequin is deflated and removed before completing the closure of the ventricle. The opening is closed with a direct suture if it is <3 cm large or with an elliptical, synthetic patch if >3 cm to avoid distortion of the cavity. The positioning of the patch is crucial in determining the residual shape of the new ventricle. To this aim, we pay attention to positioning the patch with an oblique orientation, towards the aortic outflow tract.

When indicated, mitral valve is repaired through the ventricular opening with a double arm stitch running from one trigone to the other one, embedding the two arms in the posterior anulus of the mitral valve. To avoid tears of the posterior leaflet of the mitral valve, the suture is reinforced with a Teflon strip. A restrictive mitral annuloplasty with a ring implantation may be performed in selected patients, when the LV opening is not big enough to have a good exposition of the mitral valve.

Tailored approaches

The surgical procedure as described above is usually performed to reverse LV remodelling after an anterior MI. However, the procedure may be tailored to approach a different pattern of post-infarction LV remodelling, varying from the classic posterior aneurysm with a bulging of the inferior wall (*Figure 2*) to a global LV dilatation with regional

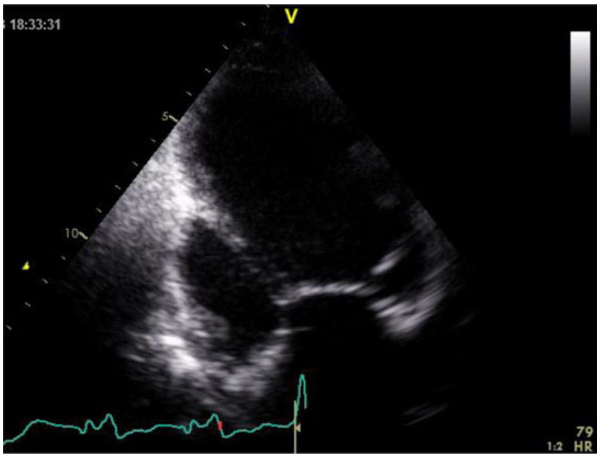

Figure 2 Localized left ventricular remodelling involving the basal segment of the inferior wall in apical two-champer view.

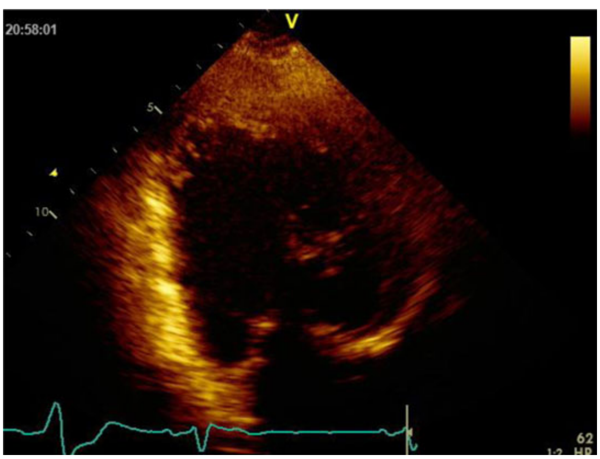

Figure 3 Extensive left ventricular remodelling involving the basal and medium segment of the inferior wall in apical two-champer view.

wall dysfunction at the inferior and posterior region, according to the site of coronary occlusion (*Figure 3*). Surgery for the posterior aneurysm generally involves a patch to close the neck of dilatation. Otherwise, the treatment of global dilatation of the infero-posterior wall is more complex and varies according to the relationship between localization of the scar and the dilatation (with or without involvement of the posterior septum) (*Figure 4*).[15]

San Donato experience

Nowadays, our Center has the largest worldwide series (*Figure 5*) and represents a reference Center for the International surgical community. The series is changed over the course of 25 years of experience, either in terms of number of patients treated or for type of patient. The decline in the number is mainly due to the advances in the treatment of acute MI, which had also an impact on survivors, in the meaning that patients with the classical dyskinetic remodelling of the apex decreased while we observed an increase of patients showing LV chambers severely distorted with a more global LV dilatation. In the former group, the indication to perform SVR is clear, while in the latter more caution is required for the lack of a well-defined zone of transition between the scarred tissue and the remote myocardium. To this regard, the use of cardiac magnetic resonance (CMR) with late-gadolinium enhancement (LGE) for the detection of myocardial scar has gained a major role in the patient selection, allowing the exclusion of those patients for whom the final result is expected to be unfavourable.[16] Furthermore, since July 2001 we started to collect the data in a prospectively way, making a serial follow-up over time; doing so, we improved our knowledge and we have been able to optimize the selection of those patients who will benefit from this procedure at most. Lastly, the released neutral results of the STICH trial in spring 2009 have called into question the additional benefit of the SVR compared with CABG alone, making the decision for referral HF patients less evident.[17]

Suggested indications

According to our experience, we consider the following to be the indications for SVR:

- Previous anterior or posterior MI, as evaluated by electrocardiogram or CMR
  CMR should be preferred, when available and not contraindicated;
- LVESVI $>$60 mL/m^2
  Pre-operative LVESV should be carefully evaluated to avoid the selection of patients with small ventricles for which the likelihood for diastolic function worsening is high;[18]
- LV dysfunction with regional asynergy, either dyskinetic or akinetic; when LV asynergy is severe and diffuse, SVR should be performed only if regions remote from the scar show some degree of detectable contraction
  CMR should be mandatory;
- Predominant HF symptoms [New York Heart Association (NYHA) class III/IV]
  The indication can also be expanded to patients presenting with ventricular arrhythmias and/or angina who need surgical revascularization if the previous conditions are present, to avoid further remodelling.

Suggested controindications

- Severe right ventricular dysfunction (biventricular dilated cardiomyopathy) (absolute)
  In our experience, right ventricular dysfunction, as reflected by an impaired TAPSE, correlates with LV dysfunction and it is an important predictor of long-term outcome in HF patients undergoing SVR[19]
- Restrictive diastolic pattern associated with high functional class and MR (absolute)
  We showed that diastolic dysfunction (E/A ratio $>$2) increases the operative risk of mortality when associated with mitral regurgitation and a New York Heart Association class greater than II[14]

How to make the decision

The choice to perform SVR should be based on a careful evaluation of patients, including HF symptoms, which should be

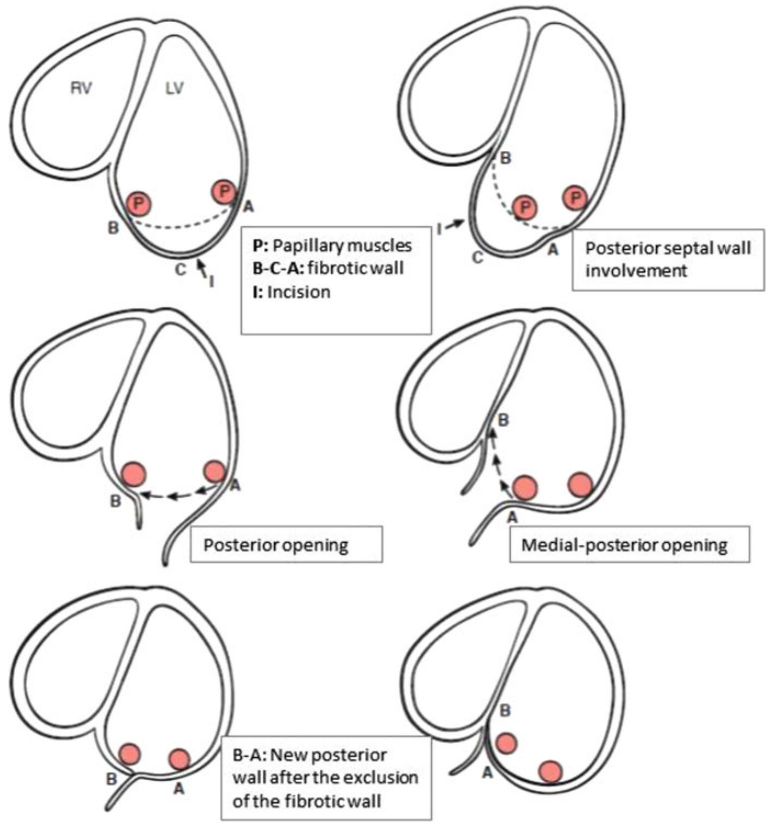

Figure 4 Schematic surgical ventricular reconstruction technique according to the location of the posterior remodelling.

predominant over angina, accurate measurements of LV geometric and haemodynamic parameters, careful evaluation of mitral valve, assessment of the transmural extent of myocardial scar tissue, and viability of regions remote from the scar, and should be performed only in centres with a high level of surgical expertise.[20]

A comprehensive echocardiographic evaluation is the first-choice diagnostic imaging tool, providing accurate information about LV dimensions and cardiac function.[21] However, the feasibility of a reliable echocardiographic examination is sometime limited by a poor acoustic window, an inadequate endocardial border definition or, when the ventricle is particularly enlarged, by incomplete visualization of the apex.

Cardiac magnetic resonance is increasingly being used for the non-invasive imaging of the HF population and it is nowadays the gold standard imaging technique to assess myocardial anatomy, regional and global function, and the extension of the scar.[22] The functional information derived from cine CMR includes global LV and RV volumes and mass, without the need to make any geometrical assumptions, and therefore applies to ventricles of all sizes and shapes, even extensively remodelled. The greatest advantage of CMR is the detection of myocardial scar with LGE. Late-gadolinium enhancement imaging visualizes irreversible damage due to an accumulation of contrast agent in areas with increased extracellular space. At the same time, CMR offers the opportunity to assess thickness and function of the remaining non-enhanced viable myocardial tissue ('the remote regions'), which may be hibernating (ischaemic but viable myocardium likely for functional recovering after CABG) or non-ischaemic but dysfunctional because of the high local tension that reduces shortening and likely for functional improvement after volume reduction obtained through SVR, as previously demonstrated.[23] On the other hand, the detection of scar by LGE in the remote regions, especially at the level of basal segments, may predict an unsatisfactory LV systolic and diastolic functional recovery and adverse clinical outcomes after SVR.[16]

Major limitations, at this time, include the exclusion of patients with pacemakers or devices for cardiac resynchronization therapy and the potential reduction of image quality in patients with significant arrhythmia or severe shortness of breath.

Insights from the literature

After the first consistent results on SVR reported by Dor and co-authors,[11,24,25] observational data in unblinded series suggested that SVR was relatively safe, and was associated with reduced LV volume, improved LV systolic function, improved symptoms, and high survival rates at 5 years.[14,26,27] Furthermore, beneficial effects from SVR include an improvement in LV mechanical synchrony, resulting in more efficient myocardial pump function.[28] However, all these studies were not randomized; only one single-centre study randomized a small number of patients

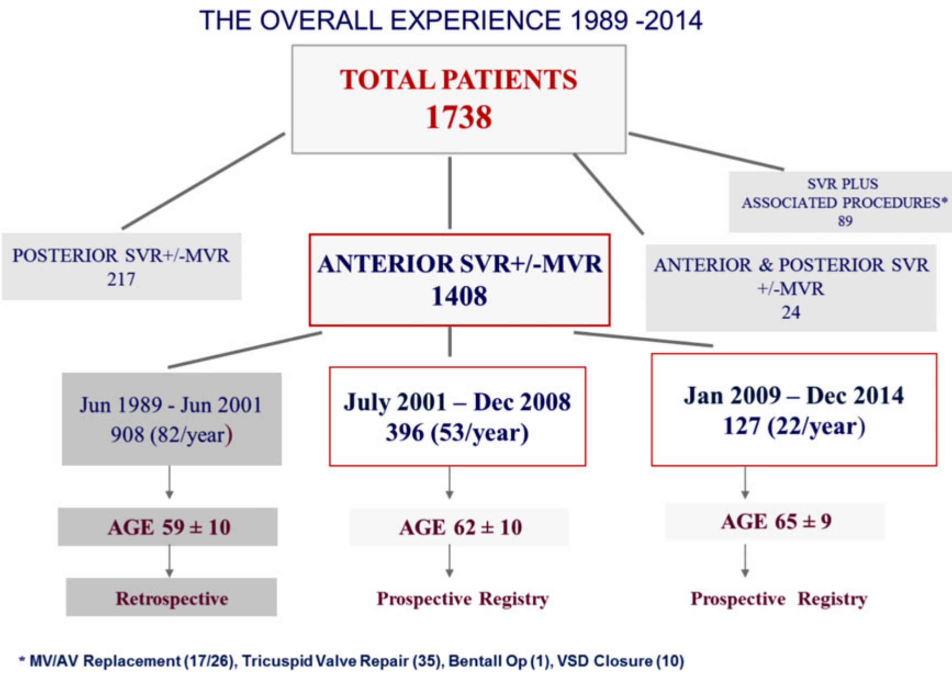

Figure 5 The overall Center experience.

($n = 74$) with dyssynergic myocardium to CABG with or without SVR, and reported that the outcome of CABG + SVR was better than that of CABG alone.[29]

The STICH trial is the only prospective, randomized, controlled trial to specifically compare CABG alone the combined procedure of CABG with SVR in patients with CAD amenable to CABG, an LVEF of 35% or less, and a dominant anterior region of myocardial akinesia or dyskinesia amenable for SVR.[17] No difference was observed in the occurrence of the primary outcome (a composite outcome of death from any cause or hospitalization for cardiac causes) between the two groups at 4 years of median follow-up. However, the relative small percentage of ESVI reduction observed in the combined group (19%, lower than the percentage of reduction reported in previous observational series, ranging from 30 to 50%), raised concerns on the extent of the SVR procedure that was applied in this trial.

We hypothesized that the lack of additional improvement in terms of survival in the SVR group observed in the STICH trial might be due to the inadequate volume reduction, which left the patients in the two arms at identical risk.[30] Later, this observation has been confirmed by Witkowski *et al.* showing that a residual postsurgical LVESVI of at least 60 mL/m^2 was independently associated with a fivefold increase of death and HF rehospitalization at 2-years follow-up after SVR.[31] Lastly, a post hoc analysis from the STICH trial showed that a post-operative LVESVI of 70 mL/m^2 or lower resulted in improved survival compared with CABG alone.[32] In agreement with these results, the most recently released guidelines on Myocardial Revascularization (ESC/EACTS) confirmed the merit of SVR which has been included as a surgical option combined with CABG in selected HF patients with a scar in the LAD territory, especially if a post-operative LVESV index <70 mL/m^2 can be predictably achieved (Class of Recommendation IIb; level of evidence B).[33]

Concurrently, we analysed follow-up data from the institutional registry including 501 consecutive patients who underwent SVR at our Institution.[34] Although it was a retrospective analysis of uncontrolled cohort, the cumulative survival rate was at around 75% at 5 years, without a significant difference between patients affected by anterior or posterior remodelling, supporting the possible additional benefit of this therapeutic strategy for patients with post-MI LV remodelling and LV dysfunction.

Surgical ventricular reconstruction and mitral valve surgery

Chronic ischaemic MR occurs in ~20–25% of patients after an anterior MI, raising up to 50–60% in case of inferior LV infarction[35] and, overall, in 50% of those with post-infarct congestive HF. Mitral regurgitation has clearly been shown to affect the natural history of patients with previous MI and CHF,[10] as well as it adversely affects survival after percutaneous or surgical myocardial revascularization.[36,37] Recently, our group addressed the differences between anterior and posterior remodelling in patients with previous MI undergoing SVR.[34] In patients with previous inferior MI, the remodelling can occur between the two papillary muscles or between the posteromedial papillary muscle and the posterior septum, which is usually deeply involved. Anyway, MR, if occurs, is mainly related to a localized inferobasal LV remodelling causing lateral displacement of posteromedial PM, increasing of internal diameter and mitral valve posterior leaflet tethering. Hence, MR occurs with less global LV remodelling and dysfunction, and especially without any involvement of the antero-septal LV wall. In patients with previous anterior MI, MR occurs mainly in the setting of global LV dilatation and severe dysfunction, reflecting a more advanced stage of disease, with tethering of both mitral valve leaflets due to apical displacement of PMs. As consequence, we found that pre-operative severe MR resulted in an independent predictor of late mortality in anterior group, but not in the posterior one.

Although significant MR and advanced LV dysfunction represent a deadly combination, the management strategy of this poor condition is not clearly delineated in the current literature.

Until recently, several studies had reported the lack of additional survival advantage from combining MV surgery with CABG in patients with IMR.[38] In contrast, results from STICH Hypothesis 1 patients with moderate or severe IMR (among 104 patients assigned to CABG with moderate to severe MR, 91 underwent CABG, and 49 received an adjunctive concomitant mitral valve procedure) suggest that the combination of CABG and MV surgery may improve long-term survival when compared with CABG alone or MED alone (50% of mortality risk at 5 years in the latter).[39] The most recent released paper by Samad and co-workers seems to confirm the survival advantage of MV surgery in a slightly larger population of patients with moderate or severe IMR and severe LV dysfunction retrieved from the Duke databases.[40]

Lately, we analysed data from a subgroup of 175 HF patients undergoing SVR combined with MV repair between January 2001 and October 2014.[41] After the operation, all but one patient had an MR grade equal or <2; only one patient had moderate MR (3+). At a median follow-up of 36 months, the majority of patients was in NYHA class I/II (113/150, 75%); no one was in NYHA class IV. The actuarial survival rate of the whole patient population at 3, 5 and 8 years following surgery was $72 \pm 4\%$, $65 \pm 4\%$ and $45 \pm 6\%$, respectively.

Although the comparison between populations of different studies is always difficult because of differences in baseline characteristics, our results, coming from a larger population with a longer follow-up, show that combining MV repair with SVR added to CABG in the majority may further improve survival at 5 years (59% in the STICH population vs. 65% in our series). The additional survival benefit observed in our population could be ascribed to the role of SVR in improving the LV adverse remodelling.[42] To this regard, it is not surprising that the Cardiothoracic surgery trials network (CTSN) trial examining the addition of MV surgery to CABG among patients with moderate ischaemic MR showed no additional benefit in terms of LV remodelling neither in survival at 1 year.[43]

Conclusions

Despite controversies which probably continue for a long time, SVR seems to have still a role in treatment of ischaemic HF patients, especially if a post-operative LVESV index <70 mL/m^2 can be predictably achieved.[32,33] The choice to add SVR to CABG should be based on a careful selection of patients, coming from a tight collaboration between surgeons, cardiologists, and radiologists.[33]

Conflict of interest: none declared.

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