Surgical treatment of congestive heart failure in coronary artery disease

Benetis R*

Clinic of Cardiac Surgery, Kaunas Heart Center, Kaunas Medical University, Lithuania

Abstract

Heart failure (HF) is a patophysiological condition, when the heart can not provide adequate blood flow to the body organs. The main cause of HF is now ischemic heart disease (IHD), and the number of patients with HF in aging society is growing. HF is becoming the leading cause of death. Medical therapy does not provide satisfactory results in respect of symptoms and survival (5 year survival 28-40%). Therefore there is a trend towards early invasive methods of IHD treatment: percutaneous or surgical revascularisation and surgical reconstruction of myocardial damage.

Most common surgical procedure in IHD is coronary artery bypass grafting (CABG). This treatment is safe and effective in patients with normal ventricular function (operative mortality 0.5%, 5 year survival >92%). Results in patients with impaired left ventricular (LV) function are better than conservative therapy, but still not satisfactory (operative mortality 8.4%, 5 year survival 65%). The modern surgical concept for improvement of ventricular function is left ventricular (LV) shape and volume restoration (SVR) accompanied by CABG. In cases of severe damage of myocardium resulting in left ventricular aneurysm or akinesia, SVR improves LV function and prevents further LV remodeling. At present it is under investigation whether SVR is of benefit for moderate-sized ventricles and NYHA class II symptoms. In case of ischemic mitral insufficiency mitral valve repair is a method of choice. The results of combined procedures in Heart Failure group (CABG + MV reconstruction or SVR) are better than CABG alone. Other surgical alternatives for HF treatment are: heart transplantation, ventricular assist devices (VAD), dynamic cardiomyoplasty, constrictive devices and cellular transplantation therapy. Heart transplantation is reserved for younger patients with less comorbidities. Shortage of donor organs and poor long-term results remains a main problem of such a treatment. VAD at present is still very expensive, and serves particularly as a "bridge to heart transplantation" or "bridge to recovery" rather then destination therapy. Despite of all achievements in medical or invasive HF treatment further basic and clinical works as well as new organization systems are necessary to find optimal strategies to reduce cost of care, improve quality of life and survival.

Key words: congestive heart failure, coronary artery, surgical treatment.

Definition of heart failure (HF) currently describes pathophysiological status when heart muscle as pump can not provide adequate blood flow to the body organs to meet metabolic needs of above tissue. Following last years, incidence of heart failure has been increasing. Reaching annual prevalence of 14-16 patients per 1 000 health care system patients [1]. However, more recent data showed kind of plateau regarding mortality from heart failure in 5 years adjusted for age. The same have been followed analyzing changes in relative risk of death after heart failure onset within a different age cohorts. Relative risk of death in younger group of patients (mean age of 60 years) showing signs of heart failure, have been reduced from 0.84 during 1985-1990 to 0.63 in a year 1991-1995 and to 0.48 during 1996-2000. However, within a group of more elderly patients (mean age 80 years) relative risk of death from heart failure at the same time frames have been reduced only slightly: 0.85-0.88-0.72. The same trend has been followed in both sex groups [2]. Also it is well recognized that ischemic heart disease (IHD) together with cerebrovascular disease defined as atherothrombosis remains leading cause of death and heart failure [3].

* CORRESPONDING AUTHOR:
Department of Cardiac Surgery
Kaunas Medical University
Eiveniu 2 LT-50009 Kaunas, Lithuania
Tel: +37 37733412; Fax: +37 37733412
e-mail: rimantas.benetis@kmuk.lt (Rimantas Benetis)

Received 05.01.2005 Accepted 16.04.2005

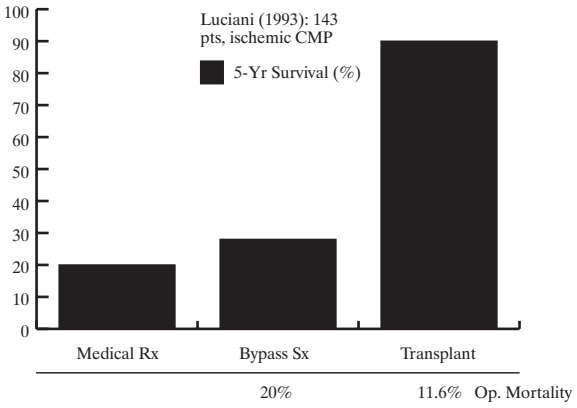
Table 1. **Arts II Arterial Revascularization Therapies Study Part II: Sirolimus-Eluting Bx Velocity Stent for the Treatment of Multivessel de Novo Coronary Artery Lesions (Luis Gruberg MD) Presenter: Patric W. Serruys, on Behalf of the ARTS II Investigators**

MACCE: 1 – Month follow-up

Characteristic	ARTS II (n=606)	ARTS I	
		CABG (n=605)	PCI (n=600)
Death (%)	0	0.5	1.5
CVA (%)	0.2	1.0	0.5
MI (%)	0.3	2.3	2.5
Reapeat CABG (%)	1.4	0.2	2.0
Reapeat PCI (%)	1	0.2	1.7
Total MACCE (%)	2.9	4.2	8.2

CABG = coronary artery bypass graft; CVA = cerebrovascular accident; MACCE = major adverse cardiac and cerebrovascular events; PCI = percutaneous coronary intervention

Figure 1. **Myocardial Revascularization**



After myocardial infarction (MI) there is a direct correlation between the quantity of HF biomarkers like Troponin I (Tn I), C-reactive protein (CRP) for B type natriuretic peptide (BNP) and incidence of onset of HF or death from it. Applying multimarkers approach to determine 30 day mortality risk in acute coronary ischemia cases in an OPUS-TIMI (16) and TACTICS-TIMI (18) studies, all 3 biomarkers increase correlated well with the increased risk of 30 days mortality rate from progressing heart failure after MI [4]. Those, whom sustain from MI, develop minor or major symptoms of HF because of left ventricular (LV) remodeling. HF is diagnosed in approximately 1% of population at the age of more then 65 years and remains as a main cause at hospitalization for the patients of the same age. More then 30% of them being rehospitalized within 90 days because of progression of HF symptoms, inadequate medical treatment and inadequate follow-up of patients: discrepancies between planned treatment and treatment after discharge.

Medical treatment of HF remains as a treatment core and includes: diuretics, digoxine, aldosterone antagonists, B blokers, ACE inhibitors and currently angiotensine II receptors inhibitors. However, they do not substantially improve symptoms of HF either prolong survival of HF patients [5].

Speaking about treatment of IHD as from the main cause of HF, there is well accepted trend towards early and aggressive mechanical revascularization of stenotic or acutely occluded coronary vessel.

PCI and stenting studies within international cardiological society, however, still showed rather high rate of death, MI and major adverse cardiac event rate in a large group of studies: TAXUS VI, ENDEAVOR I, ARTS II, TROPICAL, SVELTE, SICTO, PISCES, ISAR, IMPRESS-2 MVD, etc. [6] (*Tab. 1*).

Large numbers of invasive coronary procedures seems did not reduce, rather increased financial burden to cardiosurgical and intensive care unit budgets, because of higher numbers of more elderly and more sick patients. For instance in Denmark number of coronary artery bypass grafting (CABG) operations have decreased approximately by 20% since 2002, at the same time treatment costs within ICU have increased approximately 20% (personal communication with Danish CABG registry).

Surgical treatment of IHD complicated by HF creates number of socioeconomical, ethical and medical problems. Though, currently there is little doubt and controversy regarding indications for surgery for high risk myocardial revascularization or ischemic mitral valve repair. Indications for surgery of LV volume and shape restoration operations remain unclear.

CABG operations can provide rather low (less then 0.5%) mortality and good survival 5 years after surgery (>92%) in a group of patients with good LV function (ejection fraction /EF/>50%). However, decreased EF correlates with increased operative mortality: 6.2% 5 year survival 65%, respectively EF<0.30 – operative mortality – 8.4% (CASS registry) (*Fig. 1*)

High risk myocardial revascularization without concomitant valve or LV size and shape correction may carry high operative mortality – up to 20% comparing to 11.6% mortality rate in a heart transplant group. However, late 5 year survival is better (80%) in CABG group (80%) and heart transplantation group (85%) compared to only medical group, where 5 year survival is at best from 28% to 40% of patients [5].

We have analyzed our low EF <30% CAGB patient group. Operative mortality being 3.9% and when this group of patients was divided in to a group with restrictive diastolic dysfunction, operative mortality reached 8.7%. Analysis of HF symptoms or functional capacity after 1 year following surgery within a group of patients with low LVEF <30% showed general shift of patients from NYHA class III (66.5%) before surgery to NYHA class II postoperatively (70.4%), however 28% of pts remaining in NYHA class III. While analyzing group of pts with restrictive diastolic dysfunction after 1 year following CABG, there was a positive trend towards improvement of a postoperative NYHA class comparing to preoperative. Being 78.3% of pts in a NYHA class III and 17.4% in a NYHA class IV preoperatively and postoperatively NYHA class III – 42.9% but NYHA class IV – 28.6% (*Fig. 2*).

Currently it is still unclear whether the patients will sustain high risk CABG surgery and will benefit from it regarding reduction of HF symptom. We do continue operating on them irrespectively with or without clear evidence of myocardical viability, tests, providing optimal myocardial preservation

Figure 2. **NYHA changes in a PTS group with restrictive LV diastolic dysfunction**

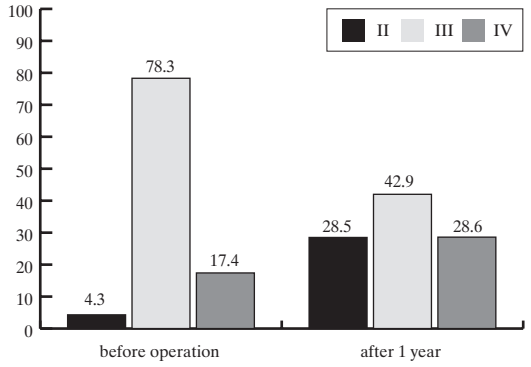


Figure 3. **Mechanisms of IMR**

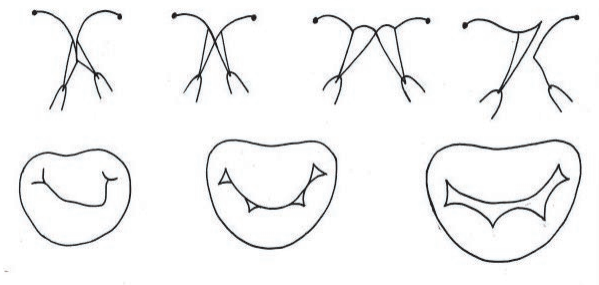


Figure 4. **Papillary muscle shortening (total) or partial with or without papillotomy including translocation**

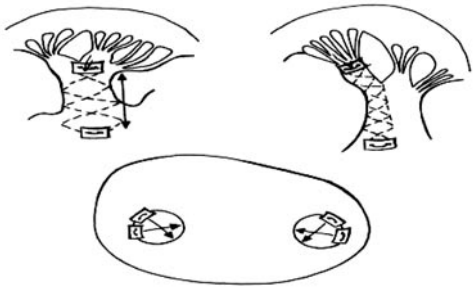


Figure 5. **Interpapillary muscle distance shortening**

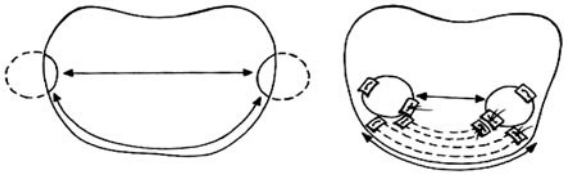


Figure 6. **Papillary muscle base – AV ring distance shortening**

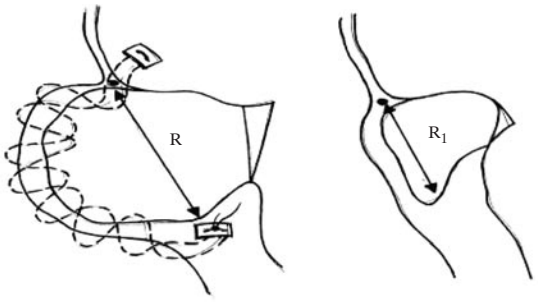
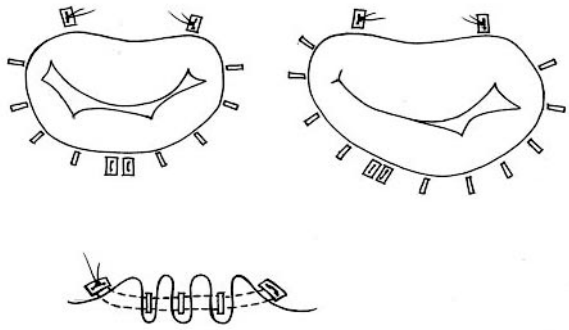


Figure 7. **Annuloplasty (symmetric or asymmetric)**



and revascularization techniques in order to facilitate optimal postoperative medical treatment, and for some young patients as a first step cardiac operation before advocating heart transplantation.

Generally the same is applied for the patients with ischemic mitral regurgitation (IMR). The mechanism of IMR depends on a changes localy and remote from MI area: post infarction remodeling of LV, which in general leads to general LV enlargement, different local LV deformities, depending on a localization of MI, leading to distortion of all MV apparatus: MV annulus dilatation, restriction or prolapse of leaflets, elongation or dysfunction of papillary muscles. Complex changes of all the LV and MV apparatus geometry would finally lead to IMR: central, paracommissural or both (*Fig. 3*).

Undersized annuloplasty for IMR and dilative CMD proposed by Steven F. Bolling remains as a good alternative to treat IMR along with CABG. Providing low operative mortality and better postoperative results comparing to mitral valve replacement. However, up to 8-12% of ischemic mitral valve annuloplasty repairs will fail within 1-3 years because of further LV remodeling and progressive LV failure. Some of the surgeons at this stage would advocate mitral valve replacement by valve substitute, for instance A. Calafiore: when valve leaflet coaptation point is beyond atrioventricular ring line more then 1 cm. Having more then 550 cases of mitral valve repair including 470 patients with IMR repair, we would advocate "future oriented ischemic mitral valve repair". Those would include: correction of the whole mitral valve apparatus: papillary muscle elongation, partial or total shortening (*Fig. 4*) or translocation techniques (*Fig. 5*) along with LV basal segment, mitral valve annulus and papillary muscle base or interpapillary muscle distance correction (*Fig. 7*). All procedure is selected after careful transesophageal echo investigation before surgery. Effectiveness of mitral valve repair is controlled in operating room, also when the heart starts beating and generating physiological haemodynamic parameters. Second attempt of mitral

Figure 8. **Results OR IMV REPAIR II**

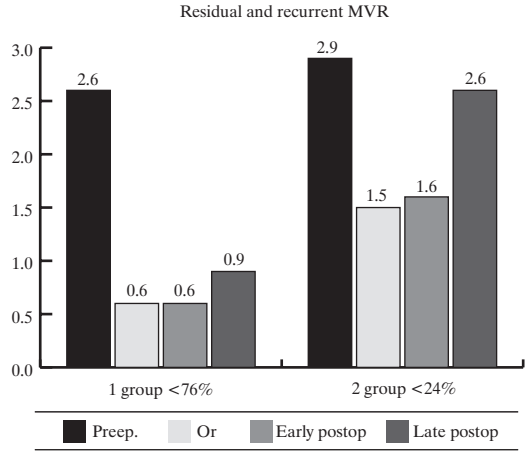
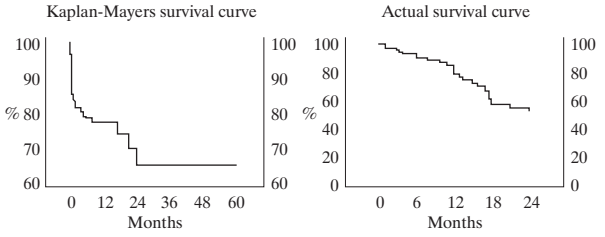


Figure 9. **Results of IMV III**



valve repair is undertaken if residual MR is more or equal to grade two. Whenever effective mitral valve repair can not be achieved, mitral valve is replaced, in our experience – 4 cases (less then 1%). Currently all annuloplasties now are performed using two double 2/0 Ethibond semi pursue, Teflon pledget reinforced sutures (*Fig. 7*). Carefully following suture annuloplasty procedure, both symmetric and asymmetric, suture tear through tissue can be completely avoided, providing long-term effective stabilization of MV annulus and subsequently clinical improvement of HF symptoms postoperatively. In our IMR repair group 86% of pts showed minor or negligible residual and recurrent MR, no more then grade 1 (mean 0.9). 14% of pts in whom TEE intra and early postoperatively revealed MR grade 1 and more, but no more then grade 2, (mean intraoperative MR 1.5; early postoperatively – 1.6) in a late postoperative period (more then 12 month) had recurrent MR with mean MR grade 2.6 (*Fig. 8*). However, only 10% (7 pts) required redo surgery on mitral valve in a late postoperative period. All valves have been replaced using mechanical (2 pts) and biological (5 pts) valve substitutes with 0% mortality. Most of them have been operated on using fibrillating heart technique. This restrospective single center analysis would confirm the indications to correct even moderate IMR, whenever left ventricular end diastolic diameter reaches or exceeds 50 mm. Nevertheless it is worth to mention that actuarial survival of our pts is 65% in 2 years after surgery (*Fig. 9*).

As LV remodeling certainly have an effect onto IMR it does have impact onto LV function impairment also. Segmental MI leads to increased LV end diastolic pressure and volume subsequently to increased wall tension and stress, and finally congestion. At present only paramount experience of Vincent Dor (Centre Cardiothoracique de Monaco) led surgeons to turn onto LV reconstruction – aneurysmectomies. At present RESTORE (Reconstructive Endoventricular Surgery Returning Torsion Original Radius Elliptical Shape to the Left Ventricle) registry demonstrated five-year findings in 1198 pts operated on between 1998-2003. Surgical ventricular restoration (SVR) using patch in 80% and without it in 20% was accompanied by CABG in 95%, MV repair in 22% and MV replacement in 1%. SVR can be performed relatively safe with overall 30 day mortality – 5.3%: 8.7% with mitral valve repair, vs 4.0% without repair. LVEF improvement is significant, from mean preoperative EF of 29.6 % to 39.5% postoperatively. Preoperative LV end systolic volume index decreased from a mean of 80.4 ml/m^2 to 56.6 ml/m^2 postoperatively. NYHA class being 3-4 in 67% of pts, in postoperative period 85% of pts were in NYHA class 1 or 2. Over all 5 year survival was 68.6% and survival was better in the group of pts who had dyskinetic as compared with akinetic LV (80% vs 65%). Risk factors for death were EF<30%, LVESVI>8 ml/m^2, NYHA class 3-4 and advanced age >75% [7].

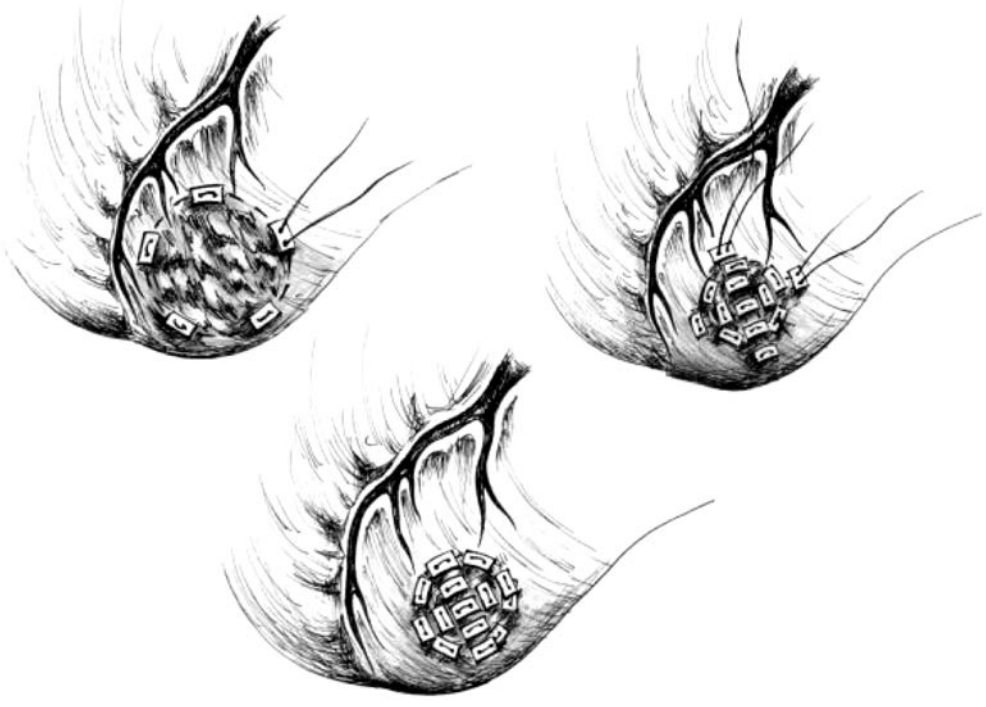
At present STICH; Surgical Treatment for Ischemic Heart Failure trial may help to answer the question of wether SVR is of benefit to those with more moderate-sized ventricles and NYHA class 2 HF symptoms. STICH is randomizing pts with ischemic cardiomyopathy to medical therapy, CABG alone, or CABG with SVR [8]. Currently we have introduced aneurismal compression plication technique for small aneurysmus or diffuse hipo-akinetic areas (*Fig. 10*).

Other alternatives in treating heart failure of ischemic origin are less effective and some of them have more or less experimental or historical importance. That would be dynamic cardiomyoplasty (A. Carpentier), cellular transplantation, constrictive devices, cardiac resynchronization therapy along with ICD implantation and least but not last heart transplantation and ventricular assist devices (VAD).

However, perhaps only heart transplantation and VAD therapy are under closer investigation. Although, heart transplantation techniques are well established and results quite well understood and accepted, this method of treatment remains reserved for younger patients with les comorbidities. Heart transplantation results because of dilative cardiomyopathy are certainly better comparing to those of ischemic. Also shortage of donor organs does not warrant to become heart transplantation as a method of choice for those with four advanced ischemic heart failure.

VAD therapy may become another, yet still expensive alternative for those with IHF. From existing registries it is well known, even FDA have approved the whole line of assist devices

Figure 10. **LV wall compression – reinforcement**



in treating terminal heart failure, 20-30% on VAD therapy will die before heart transplantation. Infection at the site of cables and cannulas or device endocarditis will reach to 70% in 2 years period. Thromboembolic complications also would account up to 25%. Only in USA 4000 pts with congestive heart disease awaits for heart transplantation, and only 2200 will get this type of surgery. For the VAD (biVAD's) only in USA there is a need of 100 systems per year.

Though it can be achieved rather good survival in all three treatment modalities: "bridge to transplantation", "bridge to recovery" and "destination therapy", vast majority of pts will die within 2 to 3 years, however, quality of life during those years may be improved dramatically.

Despite of all achievements in medical or invasive coronary artery disease therapy surgical approach to treat ischemic heart failure remains in majority of cases main method of treatment.

Finally establishment of new organization systems and models like multidisciplinary approach to IHF pts may reduce hospitalization rate more then 80%, more then by 85% can be reduced visits to emergency units, reduced cost of care, improved quality of life and survival.

References

1. McCullough PA, Philbin EF, Spertus JA, Kaatz S, Sandberg KR, Weaver WD. Confirmation of a heart failure epidemic: findings from the Resource Utilization Among Congestive Heart Failure (REACH) study. J Am Coll Cardiol, 2002; 39: 60-9.
2. Roger VL, Weston SA, Redfield MM, Hellermann-Homan JP, Killian J, Yawn BP. Trends in heart failure incidence and survival in a community-based population. JAMA, 2004; 292: 344-50.
3. The World Health Report 2001. Geneva, WHO, 2001.
4. Sabatine MS, Aretz HT, Fang LS, Dec GW. Images in cardiovascular medicine. Fibrillary/immunotactoid glomerulopathy with cardiac involvement. Circulation, 2002; 105: 120-1.
5. Young JB. Management of chronic heart failure: what do recent clinical trials teach us? Rev Cardiovasc Med, 2004; 5(Suppl. 1): S3-9.
6. Euro PCR 2004, the Paris Course on Revascularisation, May 25-28, 2004, Paris, France.
7. Athanasuleas CL, Buckberg GD, Stanley AW, Siler W, Dor V, Di Donato M. Surgical ventricular restoration in the treatment of congestive heart failure due to post-infarction ventricular dilation. J Am Coll Cardiol, 2004; 44: 1439-45.
8. Owen A. NICE try but a long way to go in heart faiture. Br J Cardiol, 2004; 95: 339-41.