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Ischemic dilatative cardiomyopathy and aneurysms of the left ventricular cavity: transplantation vs alternative surgery

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ABSTRACT: Patients with terminal end-stage heart failure due to severe coronary disease associated with dilatative cardiomyopathy have an annual mortality of 30-50%. Between July 1997 and December 1999, 21 patients at the University Hospital in Frankfurt, and 25 patients from Skopje underwent total circular repair with simultaneous coronary artery bypass. (Int J Artif Organs 2002; 25: 401-10)

KEY WORDS: Transplantation, Dilatative cardiomyopathy, Aneurysmatic dilatation of left ventricle

INTRODUCTION

Patients with terminal end-stage heart failure due to severe coronary disease associated with dilatative cardiomyopathy and low ejection fraction (EF< 25%) have an annual mortality of 30 to 50%, thus making :em the best candidates for heart transplantation and artificial heart. Transplantation has already been established as a last resort and its application has been further limited because of: donor shortage (1), world wide cultural-economical constraints and limited applicability in older patients or those with comorbid diseases. While the artificial heart is supposed to be free from such limitations, a totally implantable device is not yet available for unlimited use (2). These problems oblige us to search for another surgical strategy to manage patients with end-stage heart disease.

Increased wall stress and afterload (Law of Laplace)

As the ventricular wall expands due to infarct expansion, the stress on the ventricular wall increases. According to Laplace's law, the wall stress on the ventricle is equal to the pressure times the radius divided by two times the wall thickness (3). Mathematically, the formula is:

$$\text{Wall Stress} = \frac{\text{Pressure x Radius}}{\text{2x (Wall Thickness)}}$$

Infarct expansion dramatically alters ventricular wall stress by increasing the radius of the ventricle and reducing the wall thickness of the ventricle (3). Afterload on the ventricle is defined as the arterial blood pressure plus the walls stress of the ventricle. As wall stress increases, afterload increases, and as the afterload increases the myocytes have to work harder to contract and pump blood to the arterial system.

The increased work by the ventricle causes greater demand for oxygen and nutrients to the myocytes. If not satisfied, an escalating pattern occurs: infarct extension; infarct expansion; further increases in wall stress; higher demand for oxygen and nutrients. Ultimately, this pattern can lead to pump failure and death. In most patients with myocardial infarction the heart is able to reach stasis, although with the cost of reduced reserve capacity for increased work.

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Increased preload (Frank Starling Mechanism)

If the infarct is substantial (greater than 7% of the ventricular surface), then stroke volume decreases acutely due to the loss of contracting myocytes.The larger the infarct, the lower the resulting stroke volume. Hemodinamic mechanisms monitor stroke volume and try to compensate for decreases by increasing the diastolic volume (preload) in the ventricle. The increase in diastolic volume is beneficial in that the viable myocytes contract with greater force if there is greater distension of the individual myocytes. When the preload increases, the myocytes can generate more force to increase the stroke work of the ventricle to compensate for the loss of the infarcted myocytes. This is known as the Frank Starling Mechanism.

However, the increase in preload is only partially beneficial. Once the ventricle distends past a certain point, the force generated by the contracting myocytes decreases and the heart begins to fail to maintain the stroke volume necessary to meet the bodies demands. Additionally, the increase in preload dilates the ventricle and accelerates the process of ventricular remodeling.

The increases in afterload and preload after an acute myocardial infarction start a process of ventricular dilatation of the viable areas of the ventricle. The remodeling helps to maintain the stroke work of the ventricle. However, this normal stroke volume comes with a price of increased preload according to Frank Starlings principles and increased afterload due to increased wall stress via Laplace's Law. The myocytes also begin to grow in thickness (hypertrophy) to try to reduce the wall stress. The increase in preload means that the heart cannot respond as well to increased stress such as exercise.

Ventricular remodeling is thought to be compensatory in the first four weeks after an acute myocardial infarction. Patients that have small infarctions (less than 75% of the ventricular wall involved) have limited to no remodeling. Patients who have moderate infarctions (from about 15% of the ventricle involved) will have compensatory remodeling in the first few weeks which then stablizes. Even when the dilatation process is stable, left ventricular function is considered low when the ventricular end diastolic pressure is elevated and the stroke volume is normal or reduced. These stable patients often experience quality of life reduction because the heart has less reserve and cannot increase cardiac output to the same extent as a normal heart. The patient may experience angina or other cardiac symptoms with exercise. Additionally, if there is another myocardial infarction later in life, these patients compensation capacity is compromised. In patients with extensive infarcts (20% or greater ventricular involvement) the ventricular remodeling can occur after the initial four weeks and may continue for years depending on the medical therapy received. This remodeling is no longer considered compensatory but pathologic. As the ventricle continues to enlarge, electrical instability increases and the heart begins to fail due to greater preload and afterload. As this process continues, pump failure or arrhythmias can occur which may lead ultimately to cardiac death.

Alternative therapies for treating chronically failing hearts

It is recognized that preventing ventricular enlargement and/or reducing the volume of an enlarged ventricle is beneficial. Considering the limitations of traditional replacement surgery for advanced heart failure there are a few methods which can be considered as alternatives.

All of these methods contain the concept of volume reduction surgery and are built upon several solid surgical and physiological principles. Currently, there are four treatment options: medical treatment, surgical revascularization, surgical removal of scar tissue, and surgical removal of viable tissue.

Medical treatment

Initial medical treatment consists in treating the acute myocardial infarction with thrombolytics or angioplasty immediately after onset of symptoms to reopen the occluded coronary artery and to prevent further necrosis due to infarct extension. After the thrombolytic therapy, afterload reducing agents are given to help maintain stroke work and limit infarct expansion and ventricular remodeling. The most used afterload reducing agent is an Angiotensin Converting Enzyme inhibitor (ACE-inhibitor). Other medications can also be

given such as vasodilators, beta-blockers, growth hormone (5) etc. Medical therapy slows the progression of ventricular remodeling for patients with extensive infarcts, but does not halt it. Therefore, these patients treated medically are on a slow path to heart failure or sudden death due to electrical instability of the heart.

Surgical revascularization

Revascularization is well accepted and one of the most commonly used medical procedures. Coronary Artery Bypass Grafting (CABG) has been proven to inhibit infarct extension, restore hibernating myocardium and reduce the risk of further infarction of the ventricle. Generally, surgical revascularization is performed after the patient has stabilized and recovered from the acute phase of infarction.

Surgical removal of non-contracting scar tissue

Drs. Cooley (7), Dor (8-10), and others have developed surgical techniques to remove infarcted scar tissue and to try to restore the ventricle to a more normal geometry. These procedures can also treat infarcted septal defects within the ventricle.

Most patients with a large post infarction left ventricular aneurysm undergo operation through linear resection of the dyskinetic area (11). After direct linear closure of the left ventricular wall aneurysmectomy, the LVEDV decreased significantly, resulting in near normalization of preload reserve and reduction of tensile wall force (afterload) (12, 13). This generally leads to improvement in various hemodynamic variables such as ejection fraction (14) or NYHA classification.

This method has become a principal drawback to resection of the aneurysm by linear repair because it distorts left ventricular geometry.

Recent advances in surgical techniques for the repair of left ventricular aneurysm include the use of an endoventricular patch (15, 16) to resect or exclude the aneurysm. This remodels the left ventricular cavity and improves left ventricular function. The patch repair (excluding the aneurysm) described by Dor leaves foreign material (the patch) within the heart. The size of the patch is not standardized (17) and can lead to residual deformity of the left ventricular cavity due to its non-contractile properties which postoperatively hinder the contractile function of the myocardium.

Surgical removal of viable tissue to reduce wall stress

Dr. Batista of Brazil pioneered a new surgical technique to actually restore normal heart geometry to patients suffering from non-ischemic cardiomyopathies. In Ventricular Volume Reduction Surgery or VVRS (more commonly known as the Batista Procedure), the heart is incised and a wedge-shaped section of myocardium is taken from between the papillary muscles of the apex and the base of the left ventricle.

The original procedure involved LV lateral wall excision between the papillary muscles, which measures up to 60 mm over the short axis equator of the LV (4). Removing the lateral LV free wall theoretically allows a reduction in the short axis by up to 19 mm. In some patients with extreme LV dilatation, he later started removing the papillary muscles together with the mitral apparatus to allow greater diameter reduction. The mitral valve is then replaced (extended ventriculectomy). Initially the Batista procedure was effective for patients with end stage cardiomyopathy. End diastolic and systolic volumes were significantly reduced and ejection fraction improved. Many patients who survived did well in the intermediate term, however, high one-year morbidity and mortality and one-year survival rates as low as 55% in some studies have reduced the interest of many surgeons in this procedure.

Dorsal cardiomyoplasty

An experimental approach that holds considerable promise is the skeletal muscle ventricle (SMV), an auxiliary blood pump formed from a pedicle graft of *latissimus dorsi* muscle and connected to the circulation in a cardiac assist configuration (6). Adaptive transformation, or conditioning, by electrical stimulation enables the skeletal muscle to perform a significant proportion of cardiac work indefinitely without fatigue. The one year survival according to Sao Paulo University was 78%, and after two years 59% (18).

Total circular repair-technique

Median sternotomy was performed on all patients. With mild hypothermic cardiopulmonary bypass (32°C) and after blood cardioplegic arrest, aneurysmectomy resection is performed prior to total myocardial revascularisation. The surgical technique includes: 1) complete coronary revascularisation with use of LITA to LAD, 2) circular resection of the scar, 3) left ventricular reconstruction without using synthetic or pericardial patch. An incision is made in the center of the aneurysm and the endoventricular cavity is inspected to identify the border zone of fibrous tissue and vital myocardium. The length of the ventriculotomies ranged from 4.0 to 10.0 cm. After the careful removal of organized thrombus, the aneurysm was resected, leaving an at least 1.5 cm wide fibrous sewing cuff. For geometric reconstruction of the ventricle, a 2-0 prolene purse string suture was placed within the fibrous sewing ring, leaving about 1cm of fibrous border and pulled to reduce the ventricular orifice to a diameter of approximately 1cm. Next a 2-0 prolene suture (120 cm long) is used over two autologue pericardial strips to bring the circular cuff together. This suture to close the fibrous cuff is placed in such a fashion, that the suture lines retain the circular aspect. Excluded external tissue is folded to reinforce the suture line with the other side of the 2-0 prolene suture which is placed over the reconstruction so that an even epicardial surface results after the reconstruction (Fig. 1). The resected scarred myocardium did not include the septal scar; this was only excluded, especially in the distal third, because it was expected that hibernized myocardium would become viable after revascularization of LAD with internal thoracic artery through septal branches.

METHODS

Between July 1997 and December 1999, 21 consecutive, nonrandomized patients at the University Hospital in Frankfurt/M (group F), and 25 patients at the Institute of Pathology in Skopje (group S), between March 2000 and March 2001 underwent total circular repair with simultaneous coronary artery bypass grafting.

Every patient was assessed according to his preoperative cardiac history, angiographic, hemodynamic and intraoperative data and postoperative course and complications.

In all patients, trans-thoracic two-dimensional echocardiography was performed before and after operation. Measurements of left ventricular hemodynamic parameters were performed preoperative and postoperative with echocardiography and inserted pulmonary artery catheter. Histological samples were taken systematically from every patient who underwent these procedures. Observations were taken to ascertain the status of capillary myocardial net, atherosclerotic changes, existence of fibrous tissue, hypertrophied myocites, or present of inflammatory cells. We also took electron microscopy images, to show up abnormal structure of myocites, subcellular structure (mitochondria nucleus), or existence of apoptosis, as a form of cellular death. It is very important through this investigation to establish the difference between necrotic (hypoxia ischemia) cell death, and apoptoic (programmed-energy consuming) cell death.

Operations were performed with institutional approval and informed consent. All data are presented as mean±SD. Comparisons of preoperative and post-operative data were made using Wilcoxon's rank-sum test. Probability values of 0.05 or less were considered statistically significant.

RESULTS

In the Frankfurt group, 21 patients (18m/3f) were included in the study. Patients' age ranged from 42 to 70 (mean 73 ± 9.4). In-group S/25 patients (20m/5f) were evaluated with mean age 57±8.3. Mean number of myocardial infarcts was 1.19 in group F vs 1.2 in group S (p=ns). All patients showed symptomatic improvement after the operation. The mean *New York Heart Association* functional class improved from 3.33 to 1.62 ($p< 0.001$) in the group F, and from 3.76 to 1.6 ($p<0.001$) in group S.

Cardiac history data was similar in both groups, (Tab. I) with a tendency of younger patients in group S.

Catheterization (angio) data and distribution of coronary artery disease were not statistically different in

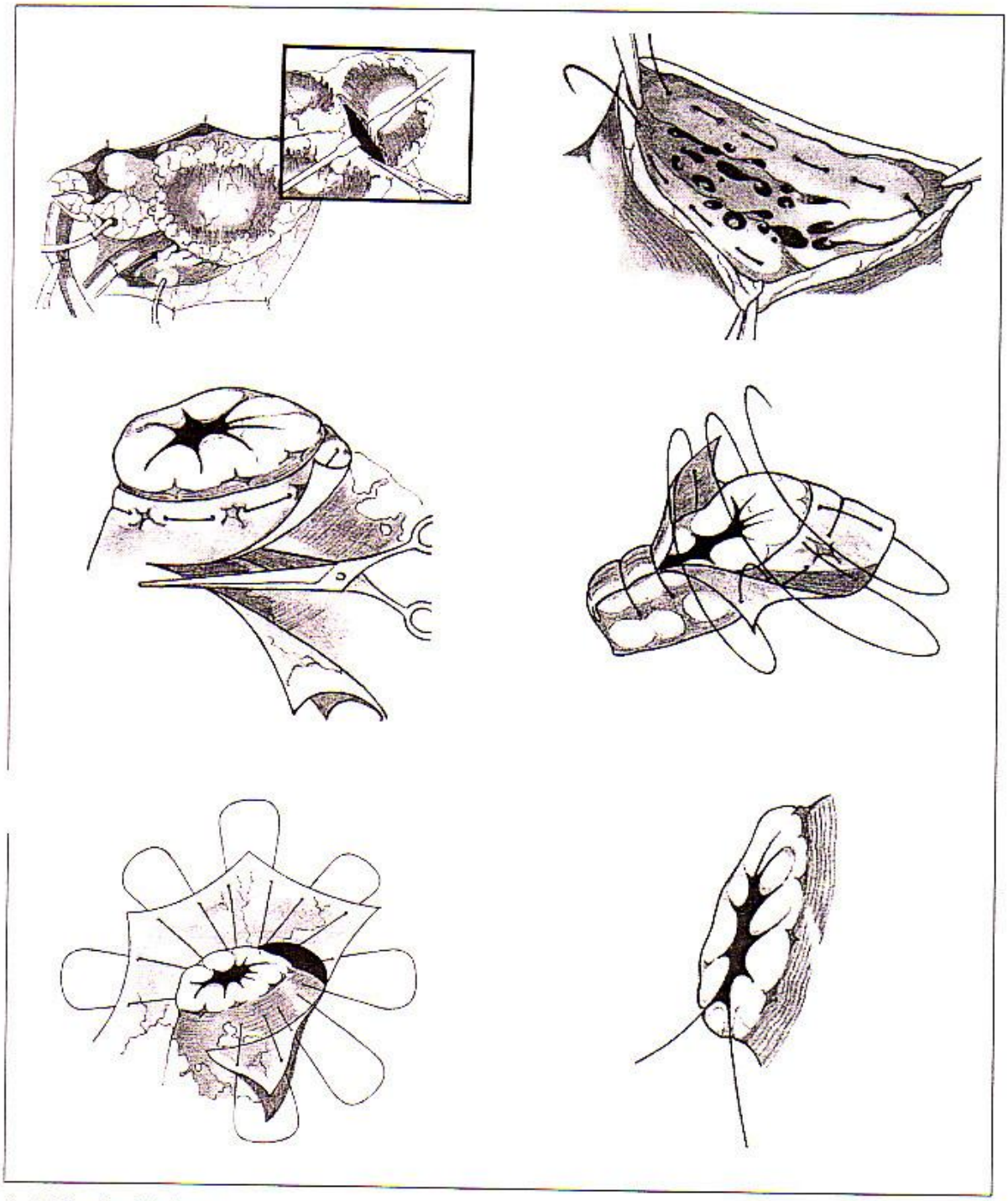

Fig. 1 - *Operative techniques.*

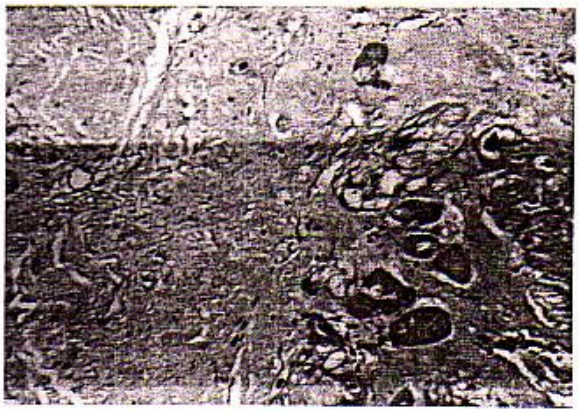

Fig. 2 - *Infiltration with inflammatory cells between myiocardial cells in the patients with dilatative cardiomyopathy.*

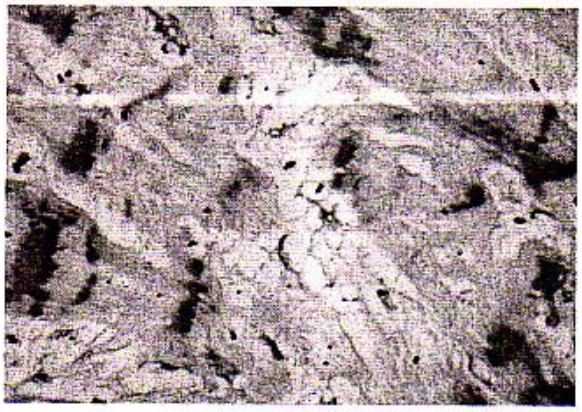

Fig. 3 - *See legend Fig. 2.*

the two groups. However, one patient in group F underwent mitral valve replacement because of severe mitral insufficiency, and two other patients (9.6%) in the same group had a carotid thrombendarterectomy. Other associated defects did not require surgical intervention.

In the Intraoperative Data Table II, comparing variables of the two groups showed no significant differences. Percentage of emergency operations was significantly higher in group F (4 patients (19%)). In group S no patients had emergency operations. The number of intracavitary

TABLE I - CARDIAC HISTORY DATA

Cardiac history data	Group F TCR (n=21) Frankfurt/M	Group S TCR (n=25) Skopje
Age	70	57
Sex ratio		
Male	18	20
Female	3	5
Previous MI-one	17 (81%)	20 (80%)
Previous MI->two	4 (19%)	5 (20%)
Location of MI		
Anterior	21	23
Posterior	2	2
MI/patient	1.19	1.2

MI = myocardial infarct

TABLE II - INTRAOPERATIVE DATA

Intraoperative data	Group F TCR (n=21) Frankfurt/M	Group S TCR (n=25) Skopje
Location of aneurysm		
Anterior	21 (100%)	24 (96%)
Posterior	0	1
Reoperation	4 (19%)	3 (12%)
Elective operation	17 (81%)	22 (88%)
Emergency operation	4 (19%)	0
By pass time/min	120	149.6
Crossclamping time/min	79	91.2
Cardioplegia	blood (100%)	blood (100%)
Size of aneurysm/cm²	20.51	22.4
Intracavitary thrombus	5 (24%)	3 (12%)
Concomitant procedures		
Aortic valve repair	0	0
Mitral valve repair	1 (4.8%)	2 (8%)
Carotid endarterectomy	2 (9.6%)	0
Postinfarction VSD closed	1 (4.8%)	0
No of venous grafts/patient	2.38	1.96
IMA implantation(%)	18 (85.7%)	22 (88%)
LAD revascularisation	19 (90.48%)	24 (96%)

IMA = internal mammarian artery
LAD = left anterior descending artery

Fig. 4 - *Electron microscopy image of early irreversibile damage with focal miofibrillar lysis.*

Fig. 5 - *Electron microscopy of myofibrillar lysis found in one of our patients.*

thrombus was higher in group F, 5 patients (24%) vs 2 patients (12%) in group S. There was a tendency towards more LAD revascularisation and more frequent use of the internal thoracic artery in group S (19-90.48% vs. 24-96%) and (18-85.7% vs. 22-88%) but this did not reach significance. The average number of vein grafts per patient was 2.38 in group F, and 1.96 in group S.

All preoperative hemodynamic data shows significant improvement early in the postoperative period. Thus ejection fraction improved from mean 28.6% preoperative to 45% postoperative ($p<0.001$) in group F and from mean 24.6% to 37.5% ($p<0.001$) in group S. The mean end-systolic volume decreased from 131 ml preoperative to 83 ml postoperative ($p<0.001$) in group F vs 177 ml to 113.9 ml in group S ($p<0.001$).

Left ventricle end-diastolic volume decreased from 180.3 ml preoperative to 105 ml ($p<0.001$) postoperative in group F, and from 244.9 ml to 175 ml ($p<0.001$) in group S without significant decreased in stroke volume from 49.4 ml to 46.7 ml in group F vs 59.6 ml to 58.8 ml in group S (p=ns) in both groups.

The same improvement was achieved with other hemodynamics (CO; CI; PAWP) (Tab. III).

Postoperatively one patient in group F (4.8%) and 5 patients (20%) in group S developed low cardiac output syndrome, but they were successfully treated with intra aortic baloonpump (IABP). Ventricular arrhythmias in both groups were successfully treated with antiarrhythmic drugs. There was one hospital death in

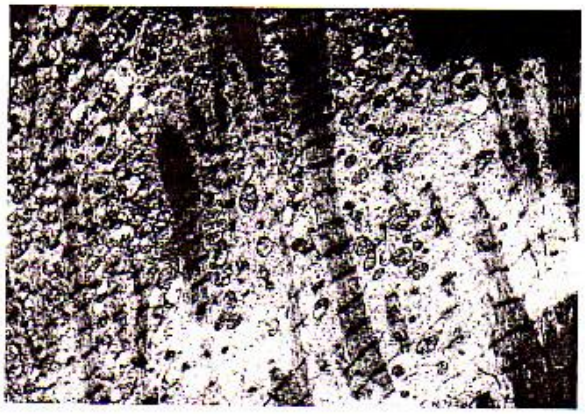

Fig. 6 - *See legend Fig. 5.*

both groups (4.8% in-group F vs 8% in group S). The cause of death was sepsis and heart failure in group S, and intractable ventricular arrhythmias in group F.

There were no cases of bleeding or sternal infection in either group and the intensive care stay was longer in group S, 5.1 days vs group F 1.48 ($p<0.001$).

Other postoperative complications were similarly distributed in the two groups (Tab. IV).

Histological results

Light microscopic examinations of resected myocardial wall showed dominant fibrosis multifocal

TABLE III - HEMODYNAMIC DATA

Hemodynamic data	Group F TCR (n=21) Frankfurt/M preop	Group F TCR (n=21) Frankfurt/M postop	Group S TCR (n=25) Skopje preop	Group S TCR (n=25) Skopje postop
Cardiac output (CO)	3.44	4.56	3.9	5.3
Cardiac index (CI)	2.03	2.8	2.2	3.1
End dyastolic volume	180.31	104.67	245	175
End systolic volume	130.94	57.98	175.8	113.9
Stroke volume	49.39	46.68	62.1	58.8
Ejecton fraction	28.62	44.95	24.6	37.7
LAP (PAWP)	15	12.1	20	14.5
NYHA CLASS	3.33	1.62	3.76	1.65

TABLE IV - POSTOPERATIVE COURSE AND COMPLICATIONS

Postoperative course complications	Group F TCR (n=21) Frankfurt/M	Group S TCR (n=25) Skopje	p
Bleeding	0	1 (4%)	n.s.
Embolism	1 (4.8%)	1 (4%)	n.s.
Rhythm disturbances			
Sinus rhythm	20 (95%)	25 (100%)	n.s.
Ventricular arrhythmia	5 (24%)	5 (20%)	n.s.
Supraventricular arrhythmia	4 (19%)	2 (12%)	n.s.
Myocardial infarction	0	0	n.s.
Low cardiac output	1 (4.8%)	5 (20%)	0.05
Permanent pacing	1 (4.8%)	0	n.s.
Sternal infection	0	1 (4%)	n.s.
ICU time/days	1.48 (1-4)	5.1 (1-2)	0.01
Mortality (%)	1 (4.8%)	2 (8%)	n.s.

degeneration of myocardial cells and atherosclerotic changes of the capillary net.

Special stain for viable myocites revealed existence of contraction band – the first sign of irreversible ischemic change in cells. The infiltration of the inflammatory cells which were present between myocardial cells points towards subacute phase of myocardial infarct or autoimmune reaction comparable with what has been found in human dilated cardiomyopathy (Figs. 2, 3).

Using electron microscopy the heart showed focal myofibrillar lysis, the loss of normal myofilament banding pattern, mitochondrial swelling and the deposition of dense granules in both sarcoplasm and myofibrilles-lipophuscin. Mitochondrial inner space showed swelling and disruption of crystas (Figs. 4-6).

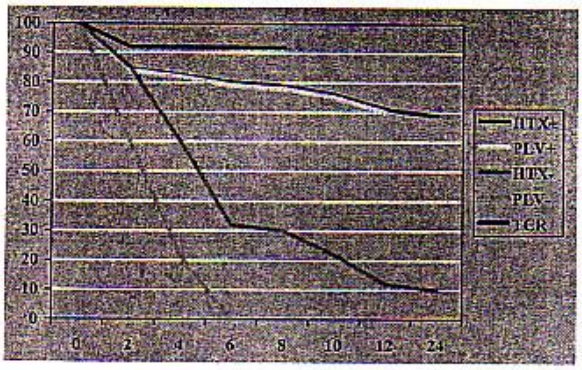

Fig. 7 - *Improvement of the clinical data.*

CONCLUSIONS

Cardiomyopathy and resulting congestive heart failure continue to be a major cause of death in the world. Despite improved medical therapy which reduces mortality, both diagnosis and quality of life remain dismal for clinically symptomatic cardiomyopathy.

Heart replacement by transplantation or use of mechanical device, is the only "cure" for such patients. Cardiac transplantation is a very limited option due to a lack of organs, numerous contraindications and dangerous side effects. Mechanical replacement and assist devices are still in their infancy because of severe complications, which are not acceptable, unless the mechanical devices are used as a bridge to transplantation. Disadvantages common to both methods are the high expense, the low availability of organs and the huge burden placed on the health care system.

The lack of medical alternatives for such a large group of patients, many of whom are relatively young, explains the enthusiasm with which total circular repair was greeted by many.

These preliminary results demonstrate that TCA may be performed with very low postoperative mortality and morbidity rates probably due to the improvement of hemodynamic data increase in ejection fraction by decrease of EDV without reduction in SV.

The left anterior descending artery, the artery responsible for the left ventricular aneurysm in many studies, was not bypassed in all patients or only in 50-70% cases). In contrast to TCR, where up to 90-100% LAD was revascularised with IMA.

The over all results of TCR are comparable and even better than those of Batista procedure dorsal cardiomyoplasty, or transplantation (Fig. 7). We are not talking only about survival, but also improving the quality of life.

Altered geometry of the infero-apical wall exerts disadvantageous effects on the contractile myocardium at the border of the aneurysm and negative curvature at the border of the infracted area indicates high local stress. The shape and function of the border zone are related to each other and to global left ventricular function. From the morphologic point of view, the TCA creates a twin apex heart with its own scar tissue which even when hypokinetic, makes the systolic ventricle nearly conical in shape.

Thus, the remodeling achieved by TCA is closer to the natural geometry, which is a desirable goal in every aneurysmatic and dilated heart.

We have successfully performed this technique in 46 patients and feel that it provides a simple technique for patients with large left ventricular aneurysm and dilated heart. We continue to follow these patients and plan to provide long-term clinical results as they become available.

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