

# Surgical options in ischemic cardiomyopathy

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The prevalence of ischemic dilated cardiomyopathy in western countries is increasing despite improvements in prevention, diagnosis, and treatment of cardiovascular disease. The management of patients with coronary artery disease and severe left ventricular dysfunction continues to be challenging and the mortality rate with medical therapy alone in this setting remains very high. Since heart transplantation represents a realistic option just for a very small number of patients, in recent years a variety of classic surgical interventions have been improved or optimized to address the complex and multifactorial pathophysiology of the ischemic heart failure picture. Myocardial revascularization, left ventricular restoration, mitral valve repair, passive containment device implantation, and surgical ablation of atrial fibrillation represent some of the "conventional" procedures which are currently in use or under development for the surgical treatment of ischemic cardiomyopathy. For several of them, the exact indications and results are not yet established and significant changes and improvements should reasonably be waited over the next few years. As techniques are refined and more data become available, the optimum surgical strategy for patients with advanced ischemic heart failure is likely to become clearer and more effective.

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## Introduction

The incidence of ischemic dilated cardiomyopathy is increasing, not only as a consequence of the aging of the population, but also because effective emergency interventions for otherwise fatal acute coronary events are extending the lives of many patients with ischemic congestive heart failure (CHF). Despite the major advances in both medical and surgical therapy, the management of patients with coronary artery disease and severe left ventricular (LV) dysfunction continues to be challenging due to the complex and multifactorial pathophysiology of this condition. In addition to the coronary artery disease potentially responsible for acute ischemia, scars and aneurysm resulting from previous myocardial infarction are often present together with variable amount of hibernating myocardium. Functional mitral regurgitation, atrial fibrillation and sustained ventricular arrhythmias may exacerbate the clinical picture of ischemic CHF (Table I). Since heart transplantation, when indicated, continues to be limited by several factors<sup>1</sup>, conventional surgical treatment for ischemic cardiomyopathy has gained increasing attention in recent years and a variety of ther-

**Table I.** Pathophysiologic components of ischemic dilated cardiomyopathy.

Ischemia-stunning
Hibernation
Fibrosis (akinesia-dyskinesia)
Dilation-remodeling
Mitral regurgitation
Atrial fibrillation
Dyssynchrony
Malignant arrhythmias

apeutic interventions have been developed or optimized<sup>2,3</sup>. This review will particularly focus on the role of myocardial revascularization, LV restoration, mitral valve repair, passive containment device implantation, and atrial fibrillation ablation for the treatment of ischemic CHF. Mechanical circulatory support and heart transplantation will not be included in the "conventional" surgical options here addressed.

## Myocardial revascularization

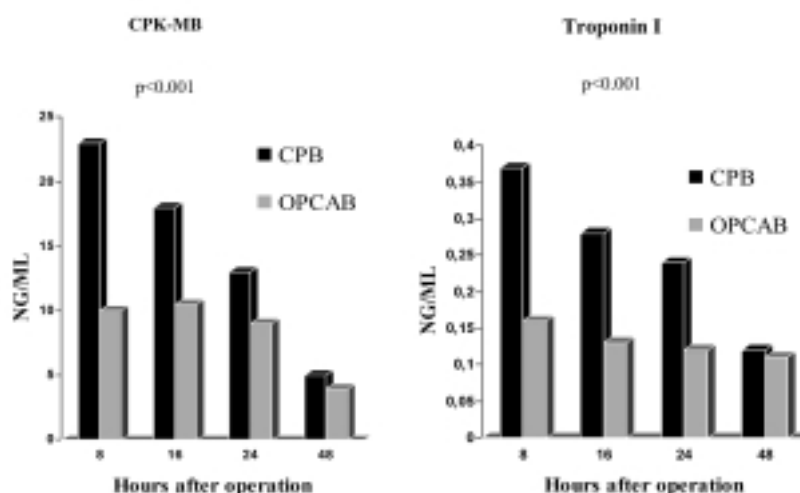
More than 50% of patients presenting with heart failure have ischemic heart disease. At beginning of coronary surgery several randomized and observational studies documented a significant survival benefit of

myocardial revascularization compared to medical therapy in patients with ischemic cardiomyopathy<sup>4-7</sup>. Since then, the early and long-term results of surgery have greatly improved but medical and electrical treatment for ischemic CHF have also been characterized by impressive advances. These progresses have raised the questions of whether revascularization is still indicated to improve the survival of such patients in the presence of modern pharmacological and electrical therapies. While this and other controversial issues will be definitively clarified by the ongoing multicenter randomized STICH trial (Surgical Treatment of Ischemic Heart Failure), some considerations need to be done on the basis of the data already available in the literature. First of all, although improvements in survival have been documented with modern medical therapy alone, even very recent trials still show a high mortality rate for patients with ischemic cardiomyopathy who have not been revascularized. In the ATLAS trial (Assessment of Treatment with Lisinopril and Survival), for example, a study comparing high and low-dose lisinopril for 2035 patients with ischemic cardiomyopathy, despite an 8% decreased mortality documented in the high-dose treatment group, the mortality rate of these patients was still 44% at 41 months. In many cases the cause of death was recurrent ischemia as confirmed by the fact that, out of 155 patients judged to have died for cardiovascular causes, 56 exhibited acute coronary pathology at autopsy<sup>8</sup>. Similarly, a randomized trial of spironolactone for 1663 patients with an ejection fraction < 35%, showed an improved survival for the treatment group but, at the same time, documented again the high mortality rate of these patients despite modern pharmacological therapy: 35% at 24 months in the study group vs 48% in the placebo group<sup>9</sup>. On the other hand, in presence of myocardial viability, it has been proved that surgery diminishes the risk of death and provides a survival advantage for such patients. For instance, in one SOLVD trial (Studies of Left Ventricular Dysfunction), designed to test the impact of enalapril on the survival rate of 4510 patients with an ejection fraction < 35%, those patients who had undergone previous bypass surgery (35% of all cases) experienced a 26% lower all-cause mortality during a 3-year follow-up compared with those who had not had myocardial revascularization<sup>10</sup>. This decreased death rate was mainly due to a decreased risk of sudden death. This finding is in accordance with the results of the Coronary Artery Bypass Graft (CABG) Patch trial, which compared arrhythmia occurrence and long-term survival in patients with an ejection fraction < 35% and abnormal signal-averaged electrocardiogram who underwent revascularization. This important trial showed that patients undergoing bypass surgery did not benefit from cardioverter-defibrillator implantation, probably because of the positive effect of grafting on the arrhythmia substrate<sup>11</sup>. Since in patients with ischemic cardiomyopathy ventricular arrhythmias are a major source of morbidity and mortality, the value of complete revas-

cularization in reducing arrhythmia recurrence should not be underestimated. The most important factors to proceed with coronary artery bypass surgery in patients with ischemic CHF are represented by the documentation of ischemic or hibernating myocardium in association with significant coronary artery stenosis and suitable distal vessels for surgical bypass. Many patients have a mixed pattern of scarring and hibernation and bypass surgery is indicated only when the extension of the hibernating areas is such that a significant impact on global LV function can be expected<sup>12</sup>. Hibernating myocardium can be identified by dobutamine stress echocardiography, single-photon emission computed tomography with thallium 201 or technetium 99 or positron emission tomography. If a significant amount of viable myocardium cannot be demonstrated, surgery is usually not the right option because no recovery of function is expected after coronary artery revascularization. Allman et al.<sup>13</sup> performed a meta-analysis of 24 studies concerning the ability of single-photon emission computed tomography, positron emission tomography or dobutamine stress echocardiography to predict survival of ischemic cardiomyopathy patients with or without myocardial revascularization. The studies included 3088 patients with an ejection fraction of  $32 \pm 8\%$ . At a mean follow-up of  $25 \pm 10$  months, in the presence of myocardial viability, revascularization decreased the risk of death by approximately 80% producing an annual mortality rate of 3.2% compared with 16% for patients who did not undergo revascularization. On the other hand, when no viable myocardium could be demonstrated, coronary surgery was not beneficial and did not improve survival compared to medical treatment.

The operation can be performed nowadays with a hospital mortality ranging from 3 to 10%<sup>14,15</sup>, which is still acceptable compared with the expected mortality rate with medical management. A careful selection of the patient is mandatory and particular attention should be paid in terms of surgical risk to the presence of right ventricular dysfunction, severe pulmonary hypertension, important LV dilation, high LV end-diastolic pressure and co-morbidities such as diabetes, peripheral vascular disease, renal or pulmonary dysfunction. Peri- and postoperative management must be optimized and the use of off-pump techniques, whenever possible, can produce additional benefits in terms of outcome<sup>16</sup>. In our own experience, the adoption of beating heart coronary revascularization in the presence of severe LV dysfunction (ejection fraction < 30%) has been associated with significantly lower serum levels of cardiac enzymes (creatinine kinase-MB and troponin I) and reduced use of postoperative intra-aortic balloon counterpulsation and inotropic support (Fig. 1).

In conclusion, most of the data available show that patients with ischemic cardiomyopathy, graftable coronary arteries and myocardial viability can significantly benefit in terms of survival and symptom status from



**Figure 1.** Postoperative serum levels of cardiac enzymes (creatine phosphokinase [CPK]-MB and troponin I) in 78 patients with an ejection fraction < 30% undergoing myocardial revascularization with (45 patients) and without (33 patients) cardiopulmonary bypass (CPB). OPCAB = off-pump coronary artery bypass grafting.

myocardial revascularization. Nevertheless, there are certainly subgroups of ischemic CHF patients for whom further investigations are required. For example, in the presence of anterior-apical akinetic areas, it is still unclear the amount of myocardial viability at which it makes sense to perform revascularization alone or in association with restoration procedures<sup>17</sup>. This, as well as other controversial issues, will hopefully be clarified by the ongoing multicenter STICH trial in the next few years.

### Left ventricular restoration

A large transmural acute myocardial infarction may result in complex alterations in the architecture and function of the left ventricle involving both the infarcted and the non-infarcted zone. These alterations, usually referred to as “LV remodeling”, can profoundly affect the patient’s prognosis. LV remodeling is a dynamic process, starting in the acute phase with infarct expansion and myocardial thinning, and progressing to LV dilation, geometric distortion, and impaired relaxation and contraction<sup>18-21</sup>. Patients who develop LV dilation following an acute myocardial infarction have significantly reduced survival: if the LV end-systolic volume index is > 60 ml/m<sup>2</sup> immediately after a myocardial infarction, the incidence of CHF and mortality exceeds 30% in 1 year<sup>22</sup>. Since LV volume is the single most important predictor of survival in patients with coronary heart disease<sup>23,24</sup>, surgical treatments to resize and reshape the distorted LV geometry have been developed. The partial left ventriculectomy, consisting of reducing LV volume by a wide resection of its lateral free wall, was originally introduced by Batista et al.<sup>25</sup> but the largest and best studied partial left ventriculectomy population was reported by the Cleveland Clinic<sup>26</sup>. In a 2-year period, 62

patients underwent this operation with a perioperative mortality of 3.2% and 26% event-free survival at 3 years. After initial improvement, most of the survivors had worsening heart failure 1 year after surgery. Because the overall benefit was so small and no predictors of outcome existed, the procedure was abandoned. Therefore, partial left ventriculectomy, despite the promising initial short-term results, is not considered anymore a reliable alternative to heart transplantation in western countries because of its unpredictable outcome and the poor late survival of the treated patients.

Contrary to the Batista operation, mainly performed in patients with diffuse LV dysfunction, surgical restoration of the size and shape of the LV chamber in patients with a post-infarction akinetic scar or dyskinetic area, is a widely accepted procedure performed with a large variety of techniques. Ventricular aneurysmectomy was first described by Sauerbruch in 1937 and a linear suture technique was proposed by Cooley in 1958<sup>27</sup>. Implantation of an endoventricular patch was introduced by Jatene<sup>28</sup> in 1985 and tends to restore the natural geometry and dynamics of the left ventricle. The goals of this procedure are to exclude with a patch all non-functional portions of the ventricular cavity, including the infarcted apical septum, and to change the spherical LV chamber to a more normal elliptical shape lowering wall stress and enhancing synchrony and contraction of the remote viable myocardium. The final result is a significant clinical improvement of CHF symptoms and a decreased incidence of ventricular arrhythmias. Whether such a procedure is called aneurysm resection, ventricular surgical remodeling, LV restoration surgery or surgical anterior ventricular endocardial restoration, the basic principles are the same. From a technical point of view, the infarcted apex of the left ventricle is opened and the transitional zone between scar and viable tissue is identified. A circular purse

string suture can be applied depending on surgeon's preference and finally the apex is closed with a patch taking care not to undersize the ventricular cavity. This endoventricular patch plasty repair<sup>29</sup>, associated with septal exclusion when needed, provides gratifying results in terms of survival and clinical improvements as documented by large multicenter studies<sup>30</sup>. Our own experience with 135 patients submitted to LV surgical restoration for post-infarction akinetic scar or dyskinetic aneurysm confirms these findings. The hospital mortality was 3.3% and a significant improvement in the ejection fraction was documented at a mean follow-up of  $32 \pm 14.7$  months (from  $30 \pm 7.9$  to  $48 \pm 8\%$ ). A significant decrease in end-diastolic and end-systolic volumes as well as in the mean pulmonary pressure was documented as well (respectively from  $145 \pm 37$  to  $86 \pm 16$  ml/m<sup>2</sup>, from  $103 \pm 37$  to  $51 \pm 18$  ml/m<sup>2</sup>, and from  $37 \pm 8.6$  to  $24 \pm 7.2$  mmHg). In 14.8% of patients, with history of ventricular arrhythmia, intraoperative ablation using endocardial excision in the border zone between scar and surrounding normal endocardium was performed and proved to be very effective in controlling the rhythm disturbances.

### Mitral valve repair

Functional mitral regurgitation is a frequent complication of end-stage ischemic cardiomyopathy. The pathophysiological components of functional mitral regurgitation are represented by annular deformation and dilation, alteration of the LV geometry with displacement of one or both papillary muscles, and reduction of the closing forces of the mitral leaflets due to severe LV dysfunction<sup>31</sup>. The alteration in size and geometry of the LV cavity produces a variable degree of tethering of one or both leaflets, causing incomplete coaptation. The severity of the morphological changes of the valvular apparatus can be specifically quantified by transesophageal echocardiography. By using the so-called "coaptation depth" and "tenting area", reliable measurements of the degree of tethering of the mitral leaflets can be obtained. The "coaptation depth" is the distance between the point of coaptation of the mitral leaflets and the plane of the mitral annulus, whereas the "tenting area" represents the surface delimited by the two mitral leaflets and the annular plane.

The development of significant mitral regurgitation strongly worsens the prognosis of patients with CHF<sup>32</sup> and its correction is intended to abolish chronic LV overload, promote reverse remodeling, improve symptoms and, hopefully, increase survival. Mitral valve repair with a complete undersized flexible ring annuloplasty has been introduced and popularized by Bolling<sup>33,34</sup> and is based on restoration of leaflet coaptation lost through progressive LV dilation and associated stretch of the papillary muscles. During a 7-year period, 140 patients with an ejection fraction < 25% un-

derwent mitral valve repair with this technique. Operative mortality was 5% and most patients improved their NYHA class, ejection fraction, cardiac output, and LV end-diastolic volume with a 5-year survival of 57%<sup>35</sup>. However, as shown by Tahta et al.<sup>36</sup>, this technique, despite achieving immediate valve competence in most of the cases, is associated with a 29% recurrence of significant mitral regurgitation ( $\geq 2+/4+$ ) at 3 years. This finding emphasizes the importance of more refined preoperative criteria to predict in which patients this procedure is going to be durable and when, on the other hand, a different repair approach or a replacement of the mitral valve should be performed. It has been suggested by Calafiore et al.<sup>37</sup> that, if the preoperative coaptation depth is > 1 cm, the geometric deformation of the mitral valve apparatus is so advanced to preclude the possibility of any conservative approach and a mitral valve replacement should be carried out. They came to this conclusion on the basis of their own clinical experience with mitral valve repair in dilated cardiomyopathy by using an over-reducing posterior annuloplasty with either homologous pericardium or a De Vega-like suture adapted to a 26-ring sizer.

So far, in our institution, 83 patients with dilated cardiomyopathy and an ejection fraction < 35%, underwent mitral valve repair or replacement for severe functional mitral regurgitation. The etiology of the LV dysfunction was idiopathic in 28 cases and ischemic in 55. Taking into consideration, for the purpose of this review, only the 55 patients with functional mitral regurgitation secondary to ischemic dilated cardiomyopathy, mitral valve repair was performed in 45 of them and mitral valve replacement, usually with a bioprosthesis, in the remaining 10. The preoperative characteristics of the repair group are reported in table II. Hospital mortality was 4.4% (2/45) and 1 patient died of sudden death 2 months after discharge. The actuarial survival was  $84 \pm 9.3\%$  at 3 years. From a technical point of view, at the beginning of our experience, we used an undersized annuloplasty with a semi-rigid ring or homologous pericardium in all cases. Because such an untailored approach was associated with a 30% recurrence of significant (2+/4+ or 3+/4) mi-

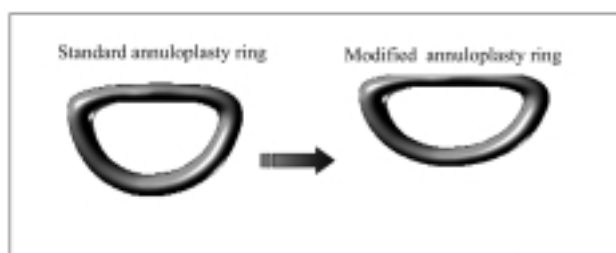
**Table II.** Preoperative characteristics of the patients submitted to mitral valve repair for functional mitral regurgitation in ischemic dilated cardiomyopathy at the San Raffaele University Hospital.

No. patients	45
Age (years)	$63 \pm 10.8$
NYHA class	$2.9 \pm 0.7$
Grade of regurgitation	$3.7 \pm 0.4$
Ejection fraction (%)	$29 \pm 6.2$
Left ventricular end-diastolic volume (ml)	$206 \pm 65$
Left ventricular end-systolic volume (ml)	$145 \pm 52.1$
Left ventricular end-diastolic diameter (mm)	$68 \pm 6.7$
Left ventricular end-systolic diameter (mm)	$53 \pm 7.4$
Systolic pulmonary artery pressure (mmHg)	$50 \pm 11.2$
Tenting area (cm <sup>2</sup> )	$2.8 \pm 1.1$
Coaptation depth (cm)	$1.2 \pm 0.4$



tral regurgitation, starting from 2001, our surgical strategy became more specific and individualized, being completely guided by the preoperative transesophageal echocardiogram. The echo findings in terms of mechanism of mitral regurgitation, location of the regurgitant jet, degree of annular dilation and severity of the leaflet tethering (coaptation depth and tenting area) were always used to decide about the type of operation to perform. In the presence of severe mitral regurgitation, associated with annular dilation and moderate leaflet tethering (defined as a coaptation depth  $< 1$  cm), surgical correction was obtained by an undersized annuloplasty alone. For this purpose, we used complete semi-rigid or, more recently, rigid rings, modified in their shape to preferentially over-reduce the septo-lateral annular diameter as shown in figure 2. On the other hand, when the tethering of the leaflets was more pronounced (coaptation depth  $\geq 1$  cm), an edge-to-edge suture was always added at the site of the regurgitant jet (central or commissural) to force leaflet coaptation and prevent recurrence of significant mitral regurgitation. Despite the shorter follow-up of the patients treated with this more recent strategy, such an echo-guided approach decreased, in our experience, the recurrence of 2+ or 3+/4 mitral regurgitation from 30 to 11%. Indeed we do believe that the undersized annuloplasty alone can be effective in functional mitral regurgitation only if the mechanism of mitral regurgitation is represented by prevalent annular dilation associated with a moderate degree of leaflet tethering. If a more advanced tethering of the mitral apparatus is present, something more, depending on the center's experience, should be added to the ring annuloplasty to increase the durability of the repair (secondary chordae resection, edge-to-edge technique, papillary muscle realignment). Moreover, the type of ring to use in this context remains controversial. Experimental and clinical studies have recently shown two interesting findings:

- even a localized infarct can produce a significant distortion of the entire mitral annulus and not only of its posterior portion;
- the septo-lateral dilation of the mitral annulus seems to play a more prominent role than the intercommissural distance in the pathophysiology of chronic ischemic mitral regurgitation<sup>38-40</sup>.

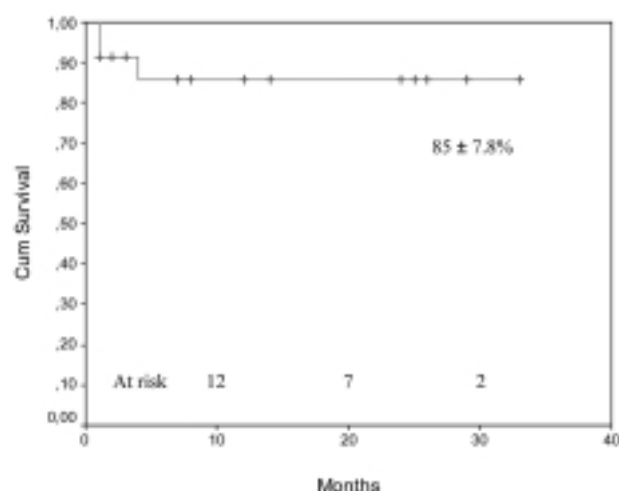


**Figure 2.** The use of undersized rigid rings modified in their shape to over-reduce the septo-lateral annular diameter could be more effective for the surgical treatment of ischemic functional mitral regurgitation.

According to these data, rings which are complete, rigid and with a shape that reduces preferentially the septo-lateral dimension of the mitral valve annulus, are likely going to be more effective and durable than partial or complete flexible rings<sup>36,41</sup>. Therefore, in our institution, the use of undersized complete rigid rings, modified in their shape to over-reduce the septo-lateral annular diameter, has become the method of choice for the treatment of functional ischemic mitral regurgitation with or without concomitant edge-to-edge technique, as described above.

Although we believe that measuring the severity of tethering of the mitral apparatus in functional mitral regurgitation is very important to decide if and how to proceed with a reconstructive approach, we do not entirely agree with Calafiore et al. who propose mitral valve replacement whenever the measured coaptation depth is  $> 1$  cm. As shown in table II, the mean coaptation depth in our patients was  $1.2 \pm 0.47$  cm (range 0.3-1.8 cm) and 23 patients had a coaptation depth  $> 1$  cm (mean  $1.3 \pm 0.2$  cm, range 1.1-1.8 cm). In these 23 cases, the addition of an edge-to-edge suture to the ring annuloplasty was associated with an actuarial freedom from recurrence of significant mitral regurgitation ( $\geq 2+$ /4+) of  $85 \pm 7.8\%$  at 20 months (Fig. 3). The association of the edge-to-edge to the undersized annuloplasty was never complicated by the development of mitral stenosis and allowed the successful repair of mitral valves with a coaptation depth between 1 and 1.5 cm (only 2 patients had a coaptation depth of 1.8 cm) which would have otherwise been replaced.

As far as the mitral valve replacement option is concerned, in our opinion, this choice should be preferred to mitral valve repair when the probability of performing a successful and durable reconstructive procedure is rather low, according to the preoperative echo findings. We decided to adopt this solution in 10 patients



**Figure 3.** Actuarial freedom from mitral regurgitation  $\geq 2+/4+$  in 23 patients with ischemic functional mitral insufficiency and coaptation depth  $> 1$  cm treated with the edge-to-edge technique and undersized ring annuloplasty.

who presented with one or more of the following conditions:

- significant fibrosis and/or retraction of the mitral leaflets;
- absence of annular dilation or presence of calcified mitral annulus;
- excessive tethering of the mitral leaflets, arbitrarily defined as a coaptation depth  $> 1.5$  cm and/or tenting area  $> 4$  cm<sup>2</sup>;
- sickest patients, with complex multiple regurgitant jets, in whom performing an effective repair could have required more than a single pump run.

### Passive containment devices

External compression of the left ventricle was first introduced as dynamic cardiomyoplasty by Carpentier<sup>42</sup> in 1987 by wrapping a conditioned skeleton muscle (latissimus dorsi) around the failing heart. Since no major benefits of this type of procedure could be demonstrated, dynamic cardiomyoplasty was totally abandoned. However, the concept of passive diastolic support to reduce the ventricular wall stress and prevent further cardiac dilation gained increasing attention promoting the development of specifically designed devices. In particular, we have experience with the Acorn cardiac support device (CorCap<sup>TM</sup>, Cardiac Support Device, Acorn Cardiovascular Inc., St. Paul, MN, USA) which is fitted around the dilated ventricles like a sock, providing diastolic support and avoiding further cardiac dilation. The basic principle behind such a device is that decreasing wall stress can promote reverse remodeling by reducing stretch proteins, hypertrophy and apoptosis. Preclinical trials and preliminary clinical experience have shown that improvements in compliance and contractility occur without constrictive effects. Oz et al.<sup>43</sup> recently reported their global clinical experience with this device in 48 dilated cardiomyopathy patients, of whom 33 also received concomitant cardiac surgery. There were no device-related adverse events or evidence of constrictive physiology. Eight early and 9 late deaths occurred with an actuarial survival of 73% at 12 months and 68% at 24 months. Ejection fraction and NYHA class improved whereas ventricular chamber dimensions significantly decreased in the overall patient cohort, as well as in the subgroup of patient who received only the cardiac support device without concomitant operations. On the basis of these results, the cardiac support device seems to be able to halt ventricular dilation promoting reverse remodeling and improving cardiac function. Randomized clinical trials are currently underway in Europe and in North America and results will be available in the next months.

Another external compression device is the Myosplint<sup>44</sup>, which consists of an implantable transventricular polyethylene braided splint, coated with expanded polytetrafluoroethylene and two polyester-coated epi-

cardial pads. The splint is positioned to bisect the dilated left ventricle, creating two smaller LV chambers and decreasing the wall tension according to Laplace's law. Animal experiments and initial clinical observations<sup>44,45</sup> showed the feasibility of the device implantation and led to the beginning of a non-randomized clinical trial in the United States. Both the CorCap and the Myosplint devices are easy to implant and may represent useful adjuncts to the surgical armamentarium for the treatment of ischemic dilated cardiomyopathy. Nevertheless, the hemodynamic improvements reported so far may be related to the concomitant cardiac operations performed in most of the cases and their effectiveness needs therefore to be further confirmed by the ongoing multicenter trials.

### Surgical ablation of atrial fibrillation

Patients with ischemic cardiomyopathy may develop paroxysmal, persistent or permanent atrial fibrillation which can significantly worsen the degree of LV dysfunction and the symptoms of CHF. Surgical ablation of atrial fibrillation can be very beneficial in this setting and easily performed by using the bipolar radiofrequency approach. In the bipolar radiofrequency system the two electrodes reside in the jaws of an atraumatic clamp which is particularly well suited to pulmonary vein isolation, since the shape of the clamp allows easy placement around these structures. The right pulmonary veins may be isolated off-pump, whereas the left ones are usually prepared on-pump with the heart beating. Each application takes about 30 s. Patients with paroxysmal atrial fibrillation are effectively treated by pulmonary vein isolation alone, whereas those with persistent or permanent atrial fibrillation usually receive additional left atrial lesions, particularly if they need concomitant mitral valve surgery. In our preliminary experience, the bipolar radiofrequency system has proven to be particularly advantageous in CHF patients because of its rapidity, safety and effectiveness: all lesions can be performed very quickly, with minimal risks to the adjacent structures and a high probability of transmural. Although further studies are required, this approach really seems to represent an important advance in the surgical treatment of atrial fibrillation in CHF patients.

Patients with ischemic cardiomyopathy often require a combination of all the above-described surgical procedures to address the multiple pathophysiological components of this complex clinical condition. The decision-making process, in the individual patient, can be particularly challenging and must take into account the surgical risk together with the potential benefits in survival and quality of life following the operation. Surgical results can be optimized with intense preoperative preparation and appropriate peri- and postoperative

management: prophylactic intra-aortic balloon counterpulsation, adequate myocardial protection, intra- and postoperative multisite pacing, tailored pharmacological treatment, and transesophageal echocardiographic monitoring, can certainly play an important role to lessen the morbidity and mortality of such a high-risk population. The exact indications and results of some of the above-described procedures are still under evaluation and significant changes and improvements should be waited over the next few years. As techniques are refined and more data become available, the optimum surgical strategy for patients with advanced ischemic heart failure is likely to become clearer and more effective.

## Riassunto

Il trattamento dei pazienti affetti da cardiopatia ischemica e severa disfunzione ventricolare sinistra rappresenta tuttora un problema complesso se si considera che la sola terapia farmacologica, in tale contesto, si associa ancora ad una mortalità decisamente elevata. Dal momento che il trapianto cardiaco rappresenta una realistica opzione terapeutica soltanto per un numero estremamente limitato di pazienti, negli ultimi anni una molteplicità di soluzioni chirurgiche cosiddette "convenzionali", ossia non trapiantologiche, sono state sviluppate ed ottimizzate al fine di migliorare la prognosi e la qualità di vita dei pazienti affetti da cardiomiopatia dilatativa postischemica (CMDI). Oltre alla patologia coronarica, potenzialmente responsabile di eventi ischemici acuti, i pazienti affetti da CMDI presentano spesso aree fibrotiche e/o aneurismatiche più o meno estese, associate a regioni di miocardio ibernato di variabile entità. Inoltre, la frequente comparsa di insufficienza mitralica funzionale, fibrillazione atriale ed aritmie ventricolari, contribuisce a rendere ancora più complesso un quadro fisiopatologico il cui trattamento prevede spesso la combinazione di diverse procedure chirurgiche: rivascolarizzazione miocardica, interventi di rimodellamento ventricolare sinistro, riparazione della valvola mitrale, impiego di sistemi di contenimento passivo delle camere ventricolari, ablazione chirurgica della fibrillazione atriale.

La rivascolarizzazione miocardica in pazienti affetti da CMDI si associa ad un significativo miglioramento in termini di sopravvivenza e qualità di vita purché sia possibile documentare preoperatoriamente la presenza di miocardio vitale.

Il rimodellamento chirurgico del ventricolo sinistro è, invece, indicato in presenza di aree acinetiche e/o discinetiche postinfartuali e consente di escludere le regioni funzionalmente inattive della cavità ventricolare sinistra, modificandone la geometria da sferica ad ellissoidale. La conseguente riduzione dello stress di parete ed il miglioramento della sincronia di contrazione del restante miocardio si traducono in sostanziali vantaggi sul piano clinico e prognostico.

La chirurgia riparativa della valvola mitrale, in caso di insufficienza mitralica funzionale associata, si propone di eliminare il sovraccarico volumetrico del ventricolo sinistro, favorire il processo di "rimodellamento inverso", migliorando la sintomatologia, la qualità di vita e la sopravvivenza dei pazienti trattati. La procedura di annuloplastica sottodimensionata, proposta ed introdotta a tale scopo, ha consentito a molti pazienti di raggiungere un importante miglioramento clinico-funzionale. I limiti che tale intervento ha tuttavia evidenziato in termini di efficacia e durata della riparazione mitralica, sono attualmente oggetto di valutazione. Alcune possibili soluzioni sono state già parzialmente individuate nell'impiego di anelli appositamente studiati e nell'aggiunta, alla sola annuloplastica, di altre tecniche riparative in grado di migliorarne i risultati.

L'impiego nella CMDI di sistemi di supporto passivo delle camere ventricolari è basato sul principio fisiopatologico secondo il quale riducendo lo stress di parete si determina una significativa diminuzione dei fenomeni di ipertrofia e di apoptosi miocardica e si inibiscono le cascate neuroumorali tipiche dello scompenso cardiaco, favorendo i processi di rimodellamento inverso e migliorando la funzione contrattile.

Infine, l'ablazione chirurgica della fibrillazione atriale, quando presente, può essere oggi eseguita in modo relativamente semplice adottando la tecnica della radiofrequenza bipolare che associa alla facilità e rapidità di esecuzione, l'efficacia derivante dall'elevata probabilità di transmuraltà delle lesioni prodotte.

Alcune di tali opzioni terapeutiche presentano indicazioni e risultati ancora in corso di definizione. Tuttavia, con il perfezionamento delle tecniche descritte ed il maggior numero di dati che si renderanno presto disponibili, le strategie chirurgiche per il trattamento della CMDI sono indubbiamente destinate a diventare progressivamente più diffuse ed efficaci.

## References

1. Deng MC, Smits JM, De Meester J, Hummel M, Schoendube F, Scheld HH. Heart transplantation indicated only in the most severely ill patient: perspectives from the German heart transplant experience. *Curr Opin Cardiol* 2001; 16: 97-104.
2. Gregoric I, Frazier OF, Couto WJ. Surgical treatment of congestive heart failure. *Congest Heart Fail* 2002; 8: 214-9.
3. Zeltsman D, Acker MA. Surgical management of heart failure: an overview. *Annu Rev Med* 2002; 53: 383-91.
4. The VA Coronary Artery Bypass Surgery Cooperative Study Group. Eighteen-year follow-up in the Veterans Affairs Cooperative Study of coronary artery bypass surgery for stable angina. *Circulation* 1992; 86: 121-30.
5. Passamani E, Davis KB, Gillespie MJ, Killip T. A randomized trial of coronary artery bypass surgery. Survival of patients with a low ejection fraction. *N Engl J Med* 1985; 312: 1665-71.
6. Alderman EL, Bourassa MG, Cohen LS, et al. Ten-year follow-up of survival and myocardial infarction in the ran-

- domized Coronary Artery Surgery Study. *Circulation* 1990; 82: 1629-46.
7. Yusuf S, Zucker D, Peduzzi P, et al. Effect of coronary artery bypass graft surgery on survival: overview of 10-year results from randomised trials by the Coronary Artery Bypass Graft Surgery Trialists Collaboration. *Lancet* 1994; 344: 563-70.
8. Uretsky BF, Thygesen K, Armstrong PW, et al. Acute coronary findings at autopsy in heart failure patients with sudden death: results from the assessment of treatment with lisinopril and survival (ATLAS) trial. *Circulation* 2000; 102: 611-6.
9. Pitt B, Zannad F, Remme WJ, et al. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. Randomized Aldactone Evaluation Study Investigators. *N Engl J Med* 1999; 341: 709-17.
10. Veenhuyzen GD, Singh SN, McAreavey D, Shelton BJ, Exner DV. Prior coronary artery bypass surgery and risk of death among patients with ischemic left ventricular dysfunction. *Circulation* 2001; 104: 1489-93.
11. Bigger JT, for the Coronary Artery Bypass Graft (CABG) Patch Trial Investigators. Prophylactic use of implanted cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary artery bypass graft surgery. *N Engl J Med* 1997; 337: 1569-75.
12. Alfieri O, La Canna G, Giubbini R, Pardini A, Zogno M, Fucci C. Recovery of myocardial function: the ultimate target of coronary revascularization. *Eur J Cardiothorac Surg* 1993; 7: 325-30.
13. Allman KC, Shaw LJ, Hachamovitch R, Udelson JE. Myocardial viability testing and impact of revascularization on prognosis in patients with coronary artery disease and left ventricular dysfunction: a meta-analysis. *J Am Coll Cardiol* 2002; 39: 1151-8.
14. Lytle BW. The role of coronary revascularization in the treatment of ischemic cardiomyopathy. *Ann Thorac Surg* 2003; 75 (Suppl): S2-S5.
15. Lorusso R, La Canna G, Ceconi C, et al. Long-term results of coronary artery bypass grafting procedure in the presence of left ventricular dysfunction and hibernating myocardium. *Eur J Cardiothorac Surg* 2001; 20: 937-48.
16. Bittner HB, Savitt MA. Off-pump coronary artery bypass grafting decreases morbidity and mortality in a selected group of high risk patients. *Ann Thorac Surg* 2002; 74: 115-8.
17. Bonow RO. Myocardial viability and prognosis in patients with ischemic left ventricular dysfunction. *J Am Coll Cardiol* 2002; 39: 1159-62.
18. Visser CA, Delemarre BJ, Peels K. Left ventricular remodeling following anterior wall myocardial infarction. *Am J Card Imaging* 1992; 6: 127-33.
19. Sodoshima J, Izumo S. The cellular and molecular response of cardiac myocytes to mechanical stress. *Annu Rev Physiol* 1997; 59: 551-71.
20. Pan J, Fukuda K, Saito M, et al. Mechanical stretch activates the JAK/STAT pathway in rat cardiomyocytes. *Circ Res* 1999; 84: 1127-36.
21. Swynghedauw B. Molecular mechanisms of myocardial remodeling. *Physiol Rev* 1999; 79: 215-62.
22. Migrino RQ, Young JB, Ellis SG, et al. End-systolic volume index at 90 to 180 minutes into reperfusion therapy for acute myocardial infarction is a strong predictor of early and late mortality. The Global Utilization of Streptokinase and t-PA for Occluded Coronary Arteries (GUSTO)-I Angiographic Investigators. *Circulation* 1997; 96: 116-21.
23. White HD, Norris RM, Brown MA, et al. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. *Circulation* 1987; 76: 44-51.
24. Yamaguchi A, Ino T, Adachi H, et al. Left ventricular volume predicts postoperative course in patients with ischemic cardiomyopathy. *Ann Thorac Surg* 1998; 65: 434-8.
25. Batista RJ, Santos JL, Takeshita N, Bocchino L, Lima PN, Cunha MA. Partial left ventriculectomy to improve left ventricular function in end-stage heart disease. *J Card Surg* 1996; 11: 96-7.
26. McCarthy PM. New surgical options for the failing heart. *J Heart Valve Dis* 1999; 8: 472-5.
27. Radovancevic B, Frazier OH. Surgical therapies for heart failure. *Curr Opin Cardiol* 2000; 15: 161-5.
28. Jatene AD. Left ventricular aneurysmectomy. Resection or reconstruction. *J Thorac Cardiovasc Surg* 1985; 89: 321-31.
29. Dor V. The endoventricular circular patch plasty ("Dor procedure") in ischemic akinetic dilated ventricles. *Heart Fail Rev* 2001; 6: 187-93.
30. Athanasuleas CL, Stanley AW Jr, Buckberg GD, Dor V, Di-Donato M, Blackstone EH. Surgical anterior ventricular endocardial restoration (SAVER) in the dilated remodeled ventricle after anterior myocardial infarction. RESTORE Group. *J Am Coll Cardiol* 2001; 37: 1199-209.
31. Koelling TM, Aaronson KD, Cody RJ, Bach DS, Armstrong WF. Prognostic significance of mitral regurgitation and tricuspid regurgitation in patients with left ventricular systolic dysfunction. *Am Heart J* 2002; 144: 524-9.
32. Grigioni F, Enriquez-Sarano M, Zehr KJ, Bailey KR, Tajik AJ. Ischemic mitral regurgitation: long-term outcome and prognostic implications with quantitative Doppler assessment. *Circulation* 2001; 103: 1759-64.
33. Badhwar V, Bolling SF. Mitral valve surgery: when is it appropriate? *Congest Heart Fail* 2002; 8: 210-3.
34. Bolling SF. Mitral reconstruction in cardiomyopathy. *J Heart Valve Dis* 2002; 11 (Suppl 1): S26-S31.
35. Bolling SF, Smolens IA, Pagani FD. Surgical alternatives for heart failure. *J Heart Lung Transplant* 2001; 20: 729-33.
36. Tahta SA, Oury JH, Maxwell JM, Hiro SP, Duran CM. Outcome after mitral valve repair for functional ischemic mitral regurgitation. *J Heart Valve Dis* 2002; 11: 11-9.
37. Calafiore AM, Gallina S, Di Mauro M, et al. Mitral valve procedure for dilated cardiomyopathy. Repair or replacement? *Ann Thorac Surg* 2001; 71: 1146-52.
38. Gorman JH, Gorman RC, Jackson BM, Enomoto Y, St John Sutton MG, Edmunds LH. Annuloplasty ring selection for chronic ischemic mitral regurgitation: lessons from the ovine model. *Ann Thorac Surg* 2003; 76: 1556-63.
39. Tibayan FA, Rodriguez F, Langer F, et al. Annular remodeling in chronic ischemic mitral regurgitation: ring selection implications. *Ann Thorac Surg* 2003; 76: 1549-55.
40. Kunzelman KS, Reimink MS, Cochran RP. Annular dilatation increases stress in the mitral valve and delays coaptation: a finite element computer model. *Cardiovasc Surg* 1997; 5: 427-34.
41. Adams DH, Filsoufi F, Aklog L. Surgical treatment of the ischemic mitral valve. *J Heart Valve Dis* 2002; 11 (Suppl 1): S21-S25.
42. Chachques JC, Grandjean PA, Tommasi JJ, et al. Dynamic cardiomyoplasty: a new approach to assist chronic myocardial failure. *Life Support Syst* 1987; 5: 323-7.
43. Oz MC, Konertz WF, Kleber FX, et al. Global surgical experience with the Acorn cardiac support device. *J Thorac Cardiovasc Surg* 2003; 126: 983-91.
44. McCarthy PM, Takagaki M, Ochiai Y, et al. Device-based change in left ventricular shape: a new concept for the treatment of dilated cardiomyopathy. *J Thorac Cardiovasc Surg* 2001; 122: 482-90.
45. Schenk S, Reichenspurner H, Boehm DH, et al. Myosplint implant and shape-change procedure: intra- and peri-operative safety and feasibility. *J Heart Lung Transplant* 2002; 21: 680-6.