Adaptive Cardiac Binding: A New Method for Treatment of Dilated Cardiomyopathy

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Abstract

Background- We propose a new surgical procedure for advanced heart failure - adaptive cardiac binding - which allows for a gradual increase in compression on the dilated heart with separate loads on the left and right ventricles.

Method- A canine model of biventricular heart failure (arteriovenous anastomosis – AVA, and doxorubicin administration) was created. Twenty-four dogs were divided into four groups: control, adynamic cardiomyoplasty (CMP), usual plastic cardiac binding (PCB), and adaptive cardiac binding (ACB). Systolic and diastolic area and volume and LVEF were measured before creation of heart failure, six weeks after, immediately after main operation, and 4 weeks later. In the animal group with ACB, liquid was added incrementally (35ml, 15ml, and finally 10ml) to each side of the pouch at weeks 1, 2, and 3.

Results- LVEF was 59±4 % before AVA and doxorubicin administration and dropped to 27±2% six weeks later. Immediately after the main operation, LVEF was 35±3% (CMP), 34±4% (PCB), and 35±4 (ACB) ($p>0.05$ between groups). Four weeks later, LVEF had not changed in the CMP (37±3%) and PCB (32±2%) groups but had significantly increased in the ACB group (48±5%, $p<0.05$). LVEF was 23±4% in the controls ($p<0.05$ vs. all groups).

Conclusion- Adaptive cardiac binding that gradually adapts to the heart's natural variations in tension and contractile strength is a promising new surgical approach for patients who have end-stage heart failure ***(Iranian Heart Journal 2006; 7 (2):5-14)***.

Key words: cardiomyoplasty■ myocardial remodeling■ biventricular assist device■ experimental surgery

In the United States alone, the prevalence of chronic heart failure has risen steadily to approximately 4-5 million people, with 400,000 new cases annually (an estimated 2000 cases per 1.5 million people).[1]

Cardiac transplantation remains the proven therapeutic modality to achieve long-term survival in patients who have end-stage heart failure.[2]

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Unfortunately, the prospect of heart transplantation proves to be a false hope for many patients because of the very limited number of donors and the strict selection criteria for recipients.[3] Artificial hearts and cardiac assist devices (bridge-to-recovery) are new and promising approaches, but their clinical application is limited mainly to the bridge-to-transplantation modality.[4]

For failing hearts, cellular cardiomyoplasty is the newly emerging procedure.[5,6] Two surgical methods that have been introduced clinically for treatment of heart failure are reduction ventriculectomy and dynamic cardiomyoplasty. These methods have controversial results. Although there are reports of its benefits, the Batista procedure has not undergone rigorous and objective scientific scrutiny to document its safety and efficacy. Data accumulated on ventriculectomy from centers in 14 countries show a 75% hospital survival rate.[7] In 2001, researchers at the Cleveland Clinic concluded that early and late failures preclude widespread use of partial left ventriculectomy, although it may be useful as a biological bridge-to-transplant.[8] Although survival rates were roughly comparable, despite initial improvement, 44% of post-ventriculectomy patients were returned to the waiting list for heart transplantation.[9] In 2001, Moreira's group in Saô Paolo (105 patients) concluded that this procedure is limited by high rates of mortality in the first postoperative months, the possibility of progressive heart failure, and arrhythmic events during follow-up.[10]

The beneficial effect of dynamic cardiomyoplasty depends on the successful systolic augmentation of the stimulated latissimus dorsi muscle (LDM) wrapped around the heart. Studies have shown[11] that the "girdling effect" from dynamic cardiomyoplasty significantly prevents the progression of left ventricular dilatation (Fig.1).

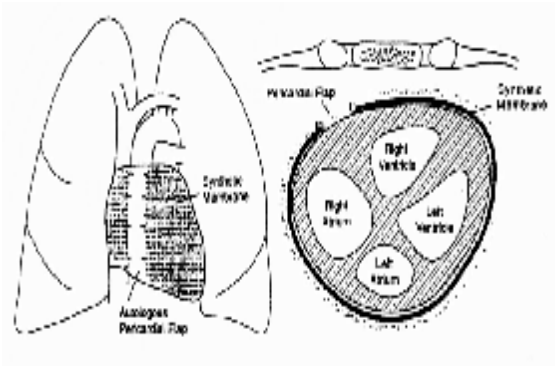


Fig. 1. Cardiac binding in experimental heart failure.

Real hemodynamic improvement has not been demonstrated in clinical trials, but all trials have demonstrated improvement in quality of life and functional status.[12,13] This may be attributed to the long-term girdling effect of dynamic cardiomyoplasty.

Studies by Vaynblat et al.[14] and Oh et al.[15] concluded that simple cardiac binding is the logical extension of the cardiomyoplasty girdling effect. Their studies involved wrapping the failing heart with a synthetic membrane to prevent dilatation. They also showed that cardiac binding reduces ventricular dilatation without exacerbating left ventricular dysfunction. The fundamental basis for this procedure is the restoration of a more normal, helical, ventricular architecture. The new ACORN Cardiac Support Device, formed from a polyester mesh fabric, was used clinically to provide high tensile strength while maintaining flexibility.[16,17]

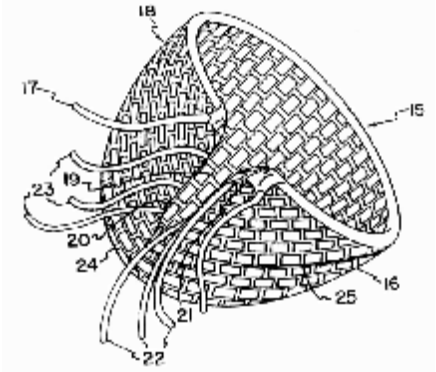


Fig. 2. Alferness, C.A. Cardiac Reinforcement Device U.S. Patent 6, 165,122 *(2000)*

This relatively simple technique is as an alternate method to restrain the ventricles from progressive dilatation. However, the device and related procedure has a significant limitation. If it becomes necessary to repeat the remodeling, another operation is needed.

We propose a new surgical device/technique: adaptive cardiac binding of the left and right ventricles. In patients with advanced heart failure, the procedure allows for a gradual increase in compression on the dilated heart with separate loads on the left and right ventricles (U.S. Patent No. 6,540,666, B1, 2003, Fig 3).[18]

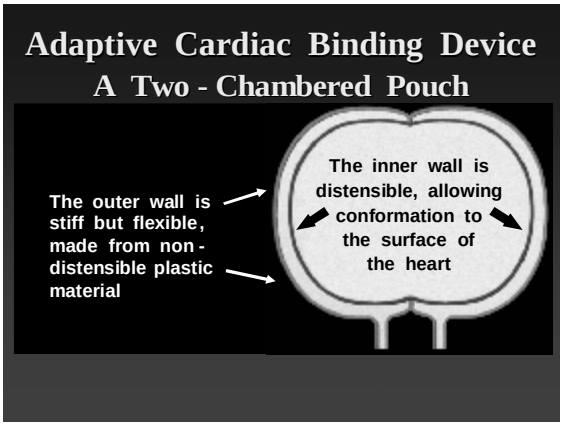
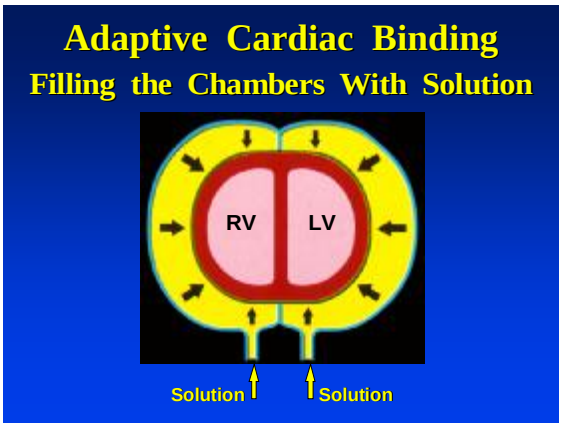


Fig. 3. Adaptive cardiac binding.

It was hypothesized that it is necessary to replace stable static heart compression with adaptive binding correlating with the heart's condition, which provides hemodynamically correct remodeling of the dilated heart. A special pouch for binding the dilated heart, sutured from two plastic bags, allows repeated volume increases inside the pouch (Fig.4). We compared the effect of this adaptive binding on congestive heart failure (CHF) with two surgical options: non-adaptive binding and adynamic cardiomyoplasty.



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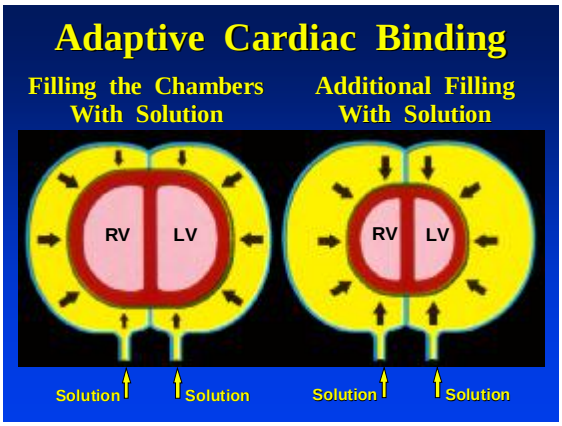

B

Fig. 4. A surgical pouch with separate chambers for the right ventricle (RV) and left ventricle (LV) wrapped around both ventricles up to the pericardial reflection.
A: first portion of solution administrated in each chamber
B: with final portion of solution, there is greater heart compression and decreasing volume of ventricles.

Methods

Our investigation was performed in two different centers: the Vakhidov Scientific Center of Surgery, Tashkent, Uzbekistan; adaptive cardiac binding and plastic cardiac binding), and the Institute for Biomedical Research, Clinic of Cardiac Surgery, Kaunas University of Medicine, Kaunas, Lithuania; adynamic cardiomyoplasty).

The animals undergoing procedures received humane care in compliance with state, federal, and local laws and policies governing animal experimentation. In Kaunas, the care conformed to the approved guidelines of the Experimental Laboratory of Kaunas University; and in Tashkent, the care was performed in compliance with the guidelines of the Experimental Department of the Center of Surgery.

Creation of the heart failure model

First surgery

Eighteen male dogs weighing 20-25 kg fasted for 18-24 hours prior to surgery. In Tashkent, anesthesia was induced with sodium pentothal (25mg/kg) and maintained with 1-1.5% halothane. Each dog was mechanically ventilated with 5 L/min of oxygen via 9 mm endotracheal tube using a Harvard Respirator. ECG leads I, II, and VI were monitored during the procedure. In Kaunas, each animal was premedicated with drugs such as atropine sulfate (0.04 mg/kg IM) and ketamine hydrocloride (10-20 mg/kg IM). In Tashkent, there were times when aceptomazine maleate (0.11-0.22 mg/kg IM) was added to the atropine sulfate and ketamine hydrocloride.

In order to create arteriovenous anastomosis (AVA), an incision (10 cm) was made on the right side of the neck just above the clavicle along the sternocleidomastoid muscle. The right jugular vein and right common carotid artery were isolated, and a side-by-side anastomosis (8-10mm) was created. Patency of the anastomosis was evaluated by auscultation (to detect continuous systolic/diastolic murmur) and palpation (to detect systolic thrill). After this procedure was complete, the first injection of doxorubicin (2.5 mg/kg IV) was administered. Another five injections were performed over the next five weeks with one-week intervals between the injections.

Six weeks after creation of the AVA and six doxorubicin injections, the plastic cardiac binding (PCB, Tashkent), adaptive cardiac binding (ACB, Tashkent), or adynamic cardiomyoplasty (CMP, Kaunas) procedures were performed. Controls (arteriovenous anastomosis and doxorubicin injections without the second main operation) were evaluated at Kaunas.

Adynamic and Adaptive Cardiomyoplasty

Second (main) surgery

a) Plastic cardiac binding (6 animals)

After anesthesia induction (see first surgery), a median sternotomy was performed. The pericardium was opened, and the heart was suspended in a cradle. A binding surgical pouch was shaped and sized according to the animal's heart. The pouch was made by suturing together two 100 ml IV bags (Viaflex, PL 146 plastic container, Baxter, Deerfield, IL). The heart was lifted gently, and the pouch was wrapped around both ventricles up to the pericardial reflection. One IV bag was placed close to the left ventricle and the other IV bag close to the right ventricle. The two lateral ends of the bags were sutured together to compress the heart just above the anterior border between the left and right ventricles. The binding was made tight enough to follow the contour of the heart without altering hemodynamic parameters. The free upper edge of the pouch was sutured to the pericardial flap, which anchored the pouch and prevented it from sliding down. Hemodynamic evaluation was performed immediately after surgery and four weeks later.

b) Adaptive cardiac binding (6 animals)

All the surgical procedures were carried out as previously stated. However, the pouch was infused with solution through ports on the end of the bags to change the volume in the pouch for adaptive heart compression. One week after binding, the first portion of solution was administrated into the pouch (35 ml into each chamber).

One week later, an additional portion of solution was added (15 ml in each chamber). On the third week, the final portion of solution was added (10 ml in each chamber). The total amount of solution injected into the pouch over this time period was 120 ml (60 ml in each chamber). Hemodynamic evaluation was performed immediately after surgery and four weeks later.

c) Adynamic cardiomyoplasty (6 animals)

The dogs were placed on their sides, and a 25 cm cutaneous incision was made from the left axilla towards the costrovertebral angle at the level of the lateral border of the scapula to the intersection between the iliac crest and the paravertebral muscles. The latissimus dorsi muscle (LDM) was dissected from the iliac crest, vertebra, inferior scapular angle and the 9^{th} to the 12^{th} rib attachments. Collateral blood vessels arising from the intercostal arteries were ligated. The muscle flap was then freed of its distal attachments with the neurovascular pedicle carefully preserved. A 4-cm segment of the anterior portion of the second rib was resected, and the LDM flap was transposed into the thoracic cavity. Cardiomyoplasty was performed through a median sternotomy with the pericardium widely open. The LDM flap was not sutured directly to the myocardium; rather, all the sutures were placed into the pericardium. The first two sutures were placed close to the left branches of the pulmonary artery and the inferior vena cava. Next, the apex of the left ventricle was elevated 2-3 cm, and the mid-portion of the LDM was placed under the heart. The heart was immediately returned to its natural position, and the electrocardiogram was monitored. If no severe arrhythmias were noted, the next suture was placed as deeply as possible at the level of the right ventricle. The free edges of the LDM flap were sutured together. Hemodynamic evaluation was performed immediately after the operation and four weeks later.

Hemodynamic studies

A two-dimensional echocardiographic study was performed using a Model 1500 echocardiographic system (Hewlett-Packard, Andover, MA) with a 2.5-MHz transducer. All the animals were anesthetized with intravenous thiopental (25 mg/kg) and placed in the left lateral decubitus position. Long-axis and short-axis views were obtained at the apex, left sternal border, and subcostal margin. Three sets of measurements were made in each echocardiographic view. The mean value of the three measurements was used to describe the quantitative data for each animal at each of the thirteen data points. Calculated parameters included left ventricular end-systolic and end-diastolic areas, volume, and ejection fraction.

Statistical Analysis

Results are expressed as mean ± 1 SEM. All the analyses were performed by one of the authors (MM) using appropriate software (StatView, SAS Institute, Inc., Cary, NC). All continuous variables were studied using one way ANOVA.[1] If a resultant fraction was found to be significant, i.e., established at $p<0.05$, a Scheff test was used to specify pair-wise differences.

Results

Prior to creation of arteriovenous anastomosis and doxorubicin injections:

Left ventricular systolic area (LVSA) was 6.8±0.5 cm^2. Left ventricular diastolic area (LVDA) was 10.2±0.9 cm^2. Left ventricular end systolic volume (LVESV) was 8.8±1.2 ml. Left ventricular end diastolic volume (LVEDV) was 21.4±1.7 ml. Left ventricular ejection fraction (EF) was 59±4%.

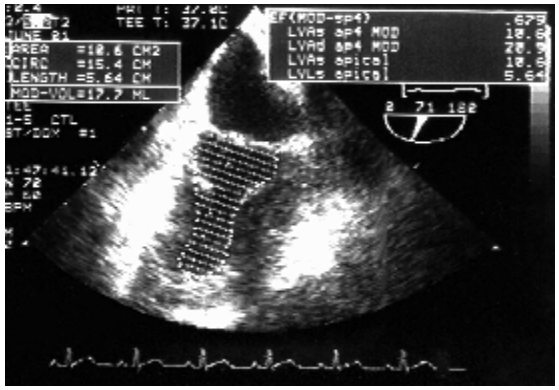

Fig. 5. Adaptive cardiac binding before AV anastamosis and doxorubicin adaptive cardiac binding.

Six weeks after AVA and six doxorubicin injections:

Left ventricular systolic area (LVSA) was 15.8±1.1cm^2. Left ventricular diastolic area (LVDA) was 19.8±2.0cm^2. Left ventricular end systolic volume (LVESV) was 26.3±4.1 ml. Left ventricular end diastolic volume (LVEDV) was 36.0±2.9 ml. Left ventricular ejection fraction (LVEF) was 27.0±2%.

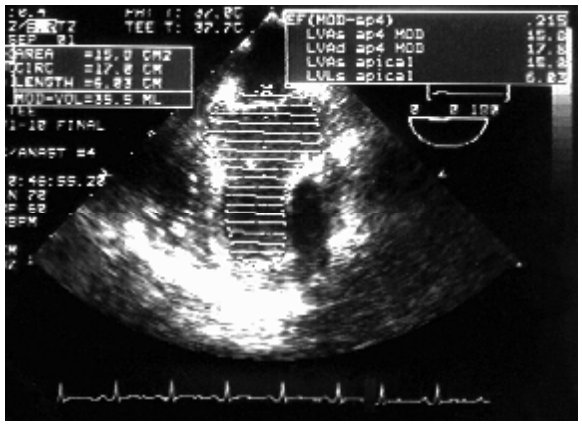

Fig. 6. 6 Weeks after AV anastamosis and 6 injections of doxorubicin

Immediately after second operation (results after initial procedure; post-procedure):

Control animals did not undergo the second operation. Test dogs were randomly divided into three groups: CMP, PCB, and ACB.

Immediately after these procedures, all the test animals showed improvement in left ventricular performance:

LVSA decreased to 13.1±0.5cm^2 in the ACB group, 12.4±0.9 cm^2 in the CMP group, and 12.9±0.7 cm^2 in the PCB group. No differences were noted between the three procedures after heart wrapping.

LVDA decreased to 15.7±1.4 cm^2 in the ACB group, 15.8±1.2 cm^2 in the CMP group, and 16.0±0.8 cm^2 in the PCB group. No differences ($p>0.05$) were noted between the three procedures after heart wrapping.

LVESV decreased to 18.6±2.2 ml in the ACB group, 18.9±2.1 ml in the CMP group, and 19.2±1.8 ml in the PCB group post-procedure. There were no differences ($p>0.05$) noted between the three procedures after heart wrapping.

LVEDV decreased to 28.6±1.3 ml in the ACB group; 29.1±2.3 in the CMP group, and 30.0±1.9 ml in PCB group. There were no differences ($p>0.05$) noted between the three procedures after heart wrapping.

LVEF increased from 27±2% pre-procedure to 35±4% in the ACB group, 35±3% in the CMP group, and 34±4% in the PCB group post-procedure. There were no differences ($p>0.05$) between the three groups after heart wrapping (Fig. 7).

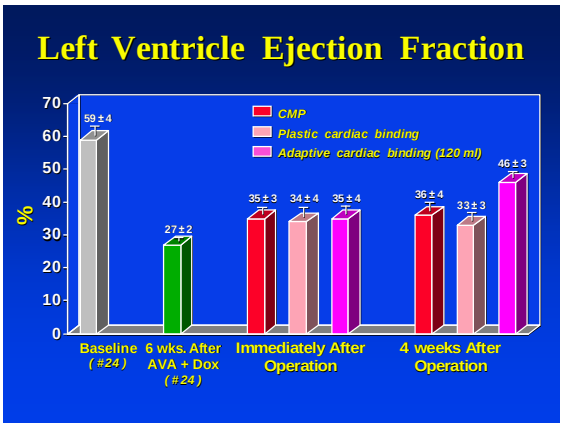

Fig. 7. Left ventricle ejection fraction.

Four weeks after second operation:

During this time, the surgical condition for controls and test animals with adynamic cardiomyoplasty, CMP, and plastic cardiac binding were the same.

Left ventricular function measurements were as follows:

Control Group: LVSA 17.9±0.9 cm^2, LVDA 23.1±1.8 cm^2, LVESV 29.2±3.8 ml, LVEDV 41.4±3.1 ml, LVEF 23±4% ($p<0.05$ vs. all other groups).

CMP Group: LVSA 11.0±0.6 cm^2, LVDA 14.3±1.1 cm^2, LVESV 18.5±1.1 ml, LVEDV 26.5±1.1 ml, LVEF 37±3%. All the left ventricular function values in the CMP group were statistically the same compared with data immediately after operation ($p>0.05$).

PCB Group: LVSA 12.9±0.8 cm^2, LVDA 15.8±0.8 cm^2, LVESV 19.6±1.8 ml, LVEDV 29.1±1.4 ml, and LVEF 32±2%. All the left ventricular function values in the PCB group were the same compared with data immediately after the operation ($p>0.05$).

ACB Group: LVSA decreased to 7.8±0.6 cm^2 and was significantly lower than both CMP and PCB ($p<0.05$). LVDA decreased to 11.2±0.6 cm^2 and was significantly lower than both CMP and PCB ($p<0.05$). LVESV decreased to 11.9±1.2 ml ($p<0.05$ vs. pre-surgery, 26.3±4.1 ml), significantly lower than both CMP ($p<0.05$, 18.5±1.1 ml) and PCB ($p<0.05$, 19.6±1.8 ml). LVEDV decreased to 21.5±1.4 ml ($p<0.05$ vs. pre-surgery 36.0±2.9 ml), significantly lower than both CMP ($p<0.05$, 26.5±1.1 ml) and PCB ($p<0.05$, 29.1±1.4 ml). LVEF increased significantly from 27.0±2% (pre-surgery) to 48±5 percent ($p<0.05$) and was significantly higher ($p<0.05$) than both CMP (37±3%) and PCB (32±2%, Fig. 8).

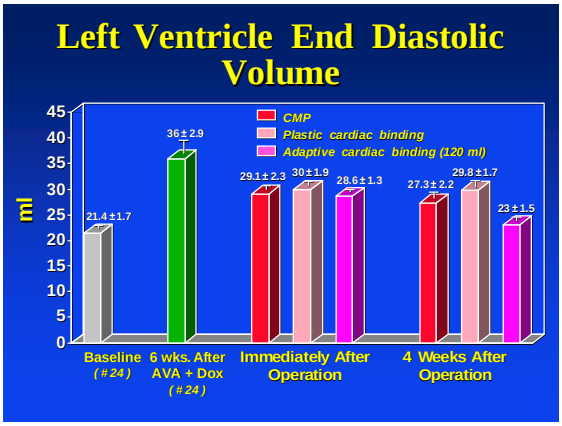

Fig. 8. Left ventricle end diastolic volume.

Discussion

A major goal in the treatment of heart failure is prevention or reduction of ventricular dilatation. Several years of clinical trials with dynamic cardiomyoplasty revealed that prevention of future ventricular dilatation is the first benefit of cardiomyoplasty. Most studies[11-13] support a beneficial girdling effect of cardiomyoplasty in limiting cardiac dilatation associated with heart failure. Nevertheless, objective evidence of hemodynamic benefit is difficult to trace. Unfortunately, a 10-year experience with clinical cardiomyoplasty showed that, after

initial elevation of left ventricular ejection fraction and decrease in left ventricular end-diastolic volume, both parameters returned to baseline levels.[13] The lack of hemodynamic improvement has been attributed to skeletal muscle damage and cardiomyoplasty-induced heart displacement, especially during contraction.

It is our opinion that the current consensus discarding dynamic cardiomyoplasty as a method of treatment for pre end-stage congestive heart failure is a mistake. If a patient with heart failure has a poor prognosis without surgical intervention, why would cardiomyoplasty not be an option if this procedure could offer the patient an additional 10 or more years of life? If heart transplants become available for all patients in need of them there would be no reason to search for alternatives. However, with the deficit of donor hearts, thousands of patients are dying without any other surgical options, simply because of the conclusion that "cardiomyoplasty did not improve LVEF after 10 years". There is urgent need for a viable option to heart transplantation to support the failing heart, not as a replacement for heart transplantation, the gold standard for end-stage heart failure, but to offer some alternative to those patients for whom heart transplantation is not available.

The concept of surgically modulating the ventricular remodeling process in heart failure was an offshoot of the experience with dynamic cardiomyoplasty. In some cardiomyoplasty patients, the cardiothoracic ratio was stabilized, or even appeared to decrease over time ("reverse remodeling").[13] Capoya et al.[11] confirmed that non-dynamic cardiomyoplasty might delay ventricular dilatation. It was proposed that the elastic stretch ability of the skeletal muscle allowed it to provide dynamic constraint against progressive cardiac dilatation. However, even synthetic material, if wrapped around the heart, reduced ventricular dilatation with doxorubicin-induced failure.[14]

Using a Marlex sheet for cardiac binding, Oh et al.[15] showed that ejection fraction was better preserved with non-dynamic cardiomyoplasty and cardiac binding with Marlex mesh, than without. In our experiments, LVEF immediately improved from 26% to 35% (cardiomyoplasty) and from 27% to 34% (plastic cardiac binding). The same improvement was seen in LVESV and LVEDV. Of course, in addition to the stretch ability, skeletal muscles undergo conformation changes, by the addition or deletion of the number of sarcomeres in the muscle fibers. This leads to a change in the pressure of the muscle on the ventricles and its resultant remodeling. Oh et al.[15] concluded that passive restraint against further dilatation alone is not sufficient to reverse remodeling and that this may necessitate the added benefit of active systolic assist available through dynamic cardiomyoplasty. We agreed with this conclusion, but, in time, when cardiomyoplasty survived the Hamlet question "to be or not to be",[18] we hypothesized that it is necessary to replace stable static heart compression with adaptive binding correlating with the heart's condition. This provides hemodynamically correct remodeling of the dilated heart. First, it was necessary to create a special pouch to wrap the heart. Oh et al.[15] used a special Marlex sheet. Power et al.[19] used a special custom polyester jacket. These devices were sutured around the heart and had one layer only. None were able to change their form, nor were they able to increase or decrease the pressure on the heart. Moreover, this operation could be done only one time during the process, and there was no way to remodulate the heart size postoperatively. Preclinical studies showed that a passive cardiac constraint device could promote reverse remodeling and

improve cardiac function. Using the ACORN Cardiac Support Device in 48 patients, Oz et al.[20] showed improvement in left ventricular ejection fraction 6 months postoperatively. Actuarial survival was 73% at 12 months and 68% at 24 months. With these encouraging results, we created a special preliminary pouch from two plastic bags, which allowed for repeated increase of the volume inside the pouch. This device allowed us to increase the volume of liquid up to 120 ml gradually, over a period of four weeks after cardiac binding. After the pouch reached a volume of 120 ml, the parameters measuring left ventricular function were significantly better than those in the groups with aplastic cardiac binding or non-dynamic cardiomyoplasty. Every week, these adaptations to the left ventricular hemodynamics were done to increase the load (pressure to LV from pouch), and each new portion of liquid had a beneficial effect. An interesting effect was described in our original proposition.[34] Left ventricular ejection fraction increased from 0.28±0.04 to 0.35 ±0.03 after binding and 0.42±0.02 after the addition of 60 ml of solution into the pouch. When the amount of solution in the pouch reached 120 ml, LVEF increased to 0.5±0.06. However, any additional portion of liquid compressed the heart too much and LVEF dropped to 0.34±0.06. When this additional amount of solution was removed, LVEF again increased to 0.49±0.02. Realizing that additional investigation is needed, we feel that this new procedure, adaptive cardiac binding, offers hope to patients with end-stage congestive heart failure who do not have the option of cardiac transplantation.

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