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CardioClasp: A New Passive Device to Re-Shape Cardiac Enlargement

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In dilated heart failure, geometric distortions place an extra load on the myocardial cells. If this extra burden can be eliminated, the myocardial wall stress would decrease leading to improved systolic ventricular performance. In a dilated heart failure model, we wanted to see whether the CardioClasp (which uses two indenting bars to reshape the left ventricle [LV] as two widely communicating "lobes" of reduced radius) could improve systolic performance by passively reshaping the LV and reducing the wall stress.

In mongrel dogs (n = 7; 25–27 kg), rapid ventricular pacing (210 ppm 1st week to 240 ppm 4th week) induced dilated heart failure. After 4 weeks, LV performance was evaluated at baseline and with the CardioClasp by measuring LV end-diastolic and peak LV systolic pressure, LV +dP/dt, LV −dP/dt, and cardiac output. With the Clasp on, LV wall stress was reduced to 58.6 ± 3.5 from 108.3 ± 8.2 g/cm^2. The fractional area of contraction (FAC) with the Clasp on (28.4 ± 4.4) was significantly increased ($p < 0.05$) from baseline (20.8 ± 4.6) and consistent with improved systolic performance. Cardiac output, LV peak systolic and end-diastolic pressures, and regional myocardial blood flow were unaltered.

The Clasp was able to acutely reshape the left ventricle, while preserving the contractile mass, and reduced the tension on the myocardial cells and increased the fractional area of contraction without decreasing the systolic blood pressure. *ASAIO Journal* 2002; 48:●●●–●●●.

Chronic heart failure remains a major cause of morbidity and mortality, with approximately 550,000 new cases diagnosed yearly in the United States.[1] Orthotopic heart transplantation remains the most effective treatment for end-stage heart disease. However, the limited donor supply, together with an increasing number of suitable transplant candidates, has made cardiac transplantation an effective treatment for only 3,400 patients in the United States in 1998, a level that remained unchanged over the past several years.[2]

In dilated heart failure, geometric distortions place an extra load on the myocardial cells. If this extra burden can be eliminated, myocardial wall stress would decrease leading to improved systolic ventricular performance. The CardioClasp is a passive cardiac support device that uses two indenting bars to reshape the left ventricle as two widely communicating "lobes" of reduced radius. In a dilated heart failure model, we tested whether the CardioClasp, by passively reshaping the left ventricle (LV) and reducing wall stress, could improve systolic performance.

Experimental Models and Methods

Theoretical Basis

The relationship between the radius of curvature and tension in the wall of a hollow structure is soundly supported by basic physics. A common example of the effect is seen in pneumatic tires. Racing bicycle tires, for example, sustain triple the pressure of an automobile tire, despite having a quarter the tear-strength. Physical analysis is conclusive: it is the local radius, not chamber volume, that determines the tensile wall stress for a given chamber pressure and wall thickness. Results are equivalent whether determined by LaPlace's original equations, by free-body diagrams, by conservation-of-energy methods, or by Castigliano's theorem: for a given chamber pressure, the local radius determines the wall tension in the plane of that radius.[3] The relationship has been specifically confirmed for left ventricular geometry by formulations of Sandler and Dodge.[4]

The wall tension, divided by the wall thickness, yields the wall tensile stress. The wall tensile stress, in turn, is the afterload against which weakened cardiomyocytes must contract in the dilated, failing, left ventricle. From basic muscle physiology, the lower the resisting tensile stress, or afterload, the faster the heart contracts and greater shortening will occur. Hill's classic experiments[5] showed this for skeletal muscle, and Sarnoff *et al.*,[6] and Suga *et al.*[7] confirmed this for the heart.

Induction of Heart Failure

All study procedures were performed in compliance with the *Principles of Laboratory Animal Care* formulated by the National Society for Medical Research, and with the *Guide for Care and Use of Laboratory Animals* issued by the National Academy of Sciences, and published by the National Institute of Health (NIH publication, volume 25, number 28, August 16, revised 1996).

Mongrel dogs (n = 7, 25–27 kg) underwent 4 weeks of continuous right ventricular (RV) pacing.[8] After an overnight fast, the animals were anesthetized with intravenous sodium thiopental (15–25 mg/kg) and intramuscular atropine (0.01

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mg/kg), intubated, and mechanically ventilated (North American Drägger, Model R-DRAGERAV, S/N. R-2035). Anesthesia was maintained with 1–2% isoflurane (Isoflurane Vaporizer, Oharda, Isotec 3, Aushell, GA) and oxygen. Surface electrocardiogram (Hewlett Packard Model 78346A) and oxygen saturation (SpO_2) were continuously monitored.

Under sterile conditions, a small (2–3 cm) incision was made over the right jugular vein. A bipolar, implantable, screw-in, transvenous myocardial pacing lead (Model 4058, 9Fr., IS-1BI connector) was introduced directly into the right jugular vein using a guided stylet (series 6058, lot J92-100914) and carefully placed at the right ventricular (RV) apex under fluoroscopy.[8]

The pacing lead was tunneled to the back of the neck where a single pulse pacemaker with attached magnet (Medtronic THERAs Model 8966i B) was implanted subcutaneously through a 4- to 5-cm incision. The pacemaker generator was secured with the underlying muscle using 3/0 Vicryl, and all the wounds were closed in layers. Lactated Ringer's solution, 250–350 ml/hr i.v., was infused perioperatively. Postoperatively, Buprenex hydrochloride (0.3 mg, i.m, b.i.d.) was administered as analgesia for 48 hours. Injection Baytril (Enrofloxacin 2.27%, 2.5 mg/kg i.m., Agriculture Division, Bayer Corporation, NADA 140-913, Animal Health, Kansas Division, KS) was given daily for 72 hours.

Rapid RV pacing was started on the 2nd postoperative day (POD) in all animals and programmed by the Medtronic PSA 9790. During the 1st week, pacing parameters were 210 ppm, 3.5 V pulse amplitude, 2.8 mV pulse sensitivity, and 1.5 ms pulse width. The pacing rate was increased consecutively to 220, 230, and 240 ppm during the 2nd, 3rd, and 4th weeks, respectively.[8] Heart failure was diagnosed upon loss of appetite, lethargy, weight loss, severe loss of skeletal muscle, and ascites. Visual inspection of the enlarged LV was carried out by two-dimensional echocardiography.

Surgical Placement of the CardioClasp

The heart failure animals were anesthetized using a combination of intravenous diazepam (1 mg/kg), fentanyl citrate (2–8 μg/kg), and 2% lidocaine (1 mg/kg). The animals were ventilated by means of an endotracheal tube with a positive-pressure respirator (North American Drägger, Model R-DRAGERAV, S/N. R-2035) and maintained by 1–2% isoflurane. ECG was continuously monitored. A SpO_2 monitor (52000 series, S/N. 9801168, WelchAllyn, Tycos Instruments, Inc., Arden, NC) to measure the oxygen saturation was used.

After administering 3,000 units of heparin, a 6 Fr micromanometer tipped pigtail catheter (Millar, Inc., Houston, TX) was inserted and advanced to the left ventricle for measurement of the left ventricular pressure. The side port of the catheter sheath was connected to a fluid transducer to measure the arterial pressure. With the animal in the right lateral position, a left lateral thoracotomy was performed at the 5th intercostal space. The pericardium was opened using fine scissors and the heart was suspended using a pericardial cradle. To measure aortic flow, a flow probe (A-series 16-mm, S/L no. 16A320, Transonic Systems, Inc., Ithaca, NY) was placed around the ascending aorta and connected to a Flow Meter (Model. T206, S/L no.1206-S-991539, Transonic Systems, Inc.).

The CardioClasp is an implantable medical device that consists of three primary components: Two rigid bars with pads and an adjustable tether. The rigid bars are 0.64 cm wide and have a defined curvature similar to the heart. Longitudinally and torsionally flexible contact pads, 1.8 cm, are attached to each bar. An adjustable tether connects the two pad-bar assemblies on either side of the left heart. The device, once implanted, was secured to the left heart with fixation means and adjusted to achieve an approximately 30% ($\pm$) end-diastolic anterior-posterior dimension reduction of the left ventricle. At 30% LV A-P dimension reduction, there were no changes in cardiac output or arterial pressure.

All pulmonary veins (PV) were mobilized from this superior to inferior aspects and a guiding catheter was introduced underneath the PV. Using a guiding catheter, the basal portion of the posterior bar of the Clasp was positioned on the posterior left atrial wall. The posterior bar was placed along the long axis of the left ventricle adjacent to the posterior descending artery. The anterior bar of the device was placed along the long axis of the left ventricle adjacent to the left anterior descending coronary artery. The anterior and posterior bars were connected by means of the tether and aligned at approximately a 180 degree placement.[3]

In one experiment ($n = 1$), two small piezoelectric crystals (Sonomicrometer Crystals, Sonometrics, Inc., Ontario, Canada) were implanted into the left ventricular free wall at the level of the papillary muscles. Regional wall motion was measured continuously with specialized digital circuitry (Sonometrics Data Acquisition System, Sonometrics, Inc.).

Measurements

At baseline, the ECG, arterial and left ventricular pressures, peak positive and negative first derivative of the left ventricular pressure (LV $+dP/dt$ and LV $-dP/dt$), and aortic flow were measured, recorded, and digitized on a recorder (VIPER, Gould, S/L no. 46-4,608-C, 338, OH). Echocardiographic study was performed using a Hewlett Packard Model Sonos 5,500 machine (SONOS 5500, Hewlett Packard, Model US97803994, serial no. M2424A, Palo Alto, CA). Direct epicardial two-dimensional echocardiographic images were obtained in the open thorax. The probe (S12; transducers of 7.5 MHz, 21380A, S/L no. US97301670, Hewlett-Packard, Sonos 5500) was placed onto the LV free wall using a stand-off gel pad, and the probe position was marked by fine epicardial suture for repositioning. LV internal dimensions and areas were obtained in the short axis view at the papillary muscle level. All the echocardiograms were performed by one of the authors (DC), a professional American board certified echocardiologist.

To measure regional myocardial blood flow, colored microspheres (7.5 million, 15 μm diameter, NuFlow, Los Angeles, CA) were injected into the left ventricle, while an arterial blood sample (7.75 ml/min) was withdrawn by means of a Harvard syringe pump.[9]

After obtaining the baseline measurements, the Clasp was placed on the heart and the LV anterior-posterior internal end-diastolic dimension was reduced by $31.8 \pm 3.0\%$. Echocardiograms were obtained. Hemodynamic data were acquired with the Clasp on. To measure the regional myocardial blood flow with the Clasp on, colored microspheres were injected as described earlier. Two data sets were obtained at

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baseline and with the Clasp on. At the end of the study, the animal was killed using 2 mEq/kg of potassium chloride (20–30 ml of KCl) i.v. and 15 mg/kg of sodium pentothal (10–15 ml i.v.). Myocardial tissue samples were obtained for blood flow analysis.

Data Analysis

Echocardiography. The standard algorithms in the echo machine were used to calculate the left ventricular end-diastolic and end-systolic areas (short axis view), and the area ejection fraction or fractional area of contraction. The echocardiographic images at end-systole and end-diastole were captured into the Hewlett-Packard echocardiography machine (Sonos 5500), where the endocardial and epicardial borders were traced. The dimensional and area information were used to calculate the fractional area of contraction and LV wall stress. Regional LV wall stress was calculated as (0.334 × LVP × LVD)/WT [1 + WT/LVD]), where LVP is left ventricular pressure, LVD is left ventricular anterior posterior diameter, and WT is LV wall thickness.[10–12]

Myocardial blood flow. For blood flow determinations, tissue samples were obtained from the epicardium and endocardium near the placement of the Clasp bars and kidneys (to determine adequate mixing of the microspheres). Ten myocardial samples were obtained on each side of the bar along the base-to-apex axis. The wet weight of each tissue sample was measured, after which the tissue samples were dried in a 70°C oven for 24 hours, the dry weight determined, and the samples sent overnight to BioPAL Laboratory Inc.(Worcester, MA) for myocardial blood flow analysis.[9] Regional epicardial and endocardial blood flow were measured in both nonrisk and risk areas. Risk areas include the myocardium supplied by the coronary vessels crossing under the bars. The ratio of endocardial to epicardial blood flow was measured and compared for significant differences between the nonrisk and risk areas.

Hemodynamics. Using software developed in Visual Basic for Excel (Microsoft Excel 7.0, Microsoft, Inc., Redwood, WA), hemodynamic variables were extracted from a digitally stored data file. Ectopic and postectopic cycles were excluded from the analysis. For each cardiac cycle, the end-diastolic pressure, the peak ventricular systolic pressure, LV +dP/dt, LV −dP/dt), peak and end-diastolic aortic pressures were determined, and stroke volume was calculated.

End-systolic pressure–segment-length relationship and end-systolic elastance. End-systolic pressure (ESP, P_{es}) was defined as peak pressure for isovolumic contractions. For ejecting beats, end-systolic segment-length (ESPS, L_{es}) and ESPs were defined by the point in the cardiac cycle at which the instantaneous ratio between pressure and segment-length attained maximal value. The relationships between ESP and ESPS (ESPSR) were analyzed by linear regression analysis applied to data from different segment-length beats according to the formula $P_{es} = E_{es} (L_{es}\text{-}L_o)$, where E_{es} is the end-systolic elastance and L_o is the segment-length axis intercept. From each pressure segment-length loop, the maximum ($P_{es}/[L_{es}\text{-}L_o]$) ratio was determined with L_o initially equal to zero. A least-squares linear regression was applied to those points, generating slope (E_{es}) and intercept (L_o) estimates. With the estimate of the intercept, the maximal ($P_{es}/[L_{es}\text{-}L_o]$) ratio for each cycle was obtained and a subsequent regression was used to determine new estimates for E_{es} and L_o. This process was repeated until there was no change in either parameter with subsequent interactions.[13,14]

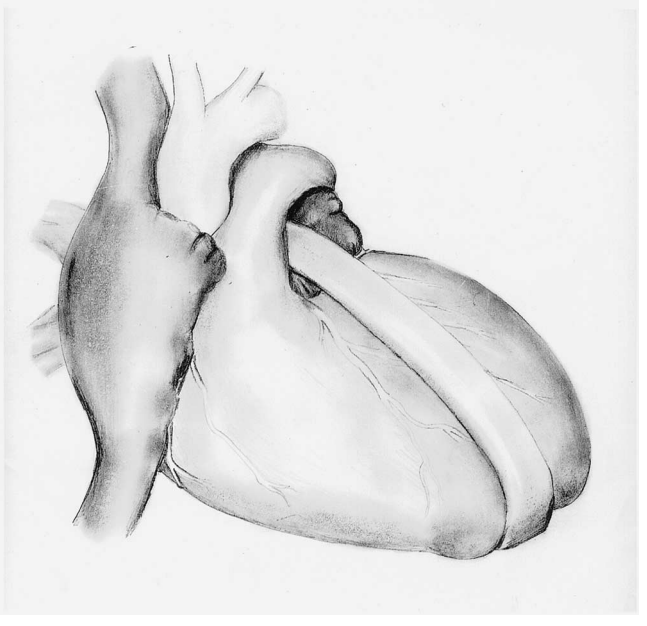

Figure 1. Representative images of the CardioClasp device over the left ventricular long axis to reshape the ventricle. The anterior bar is placed along the anterior long axis of the left ventricle adjacent to the left anterior descending coronary artery. The posterior bar is placed along the posterior long axis of the left ventricle near the posterior descending coronary artery and is not visible in this figure.

Data are expressed as mean ± standard error of the mean. The hemodynamic and echo variables at baseline were compared with the Clasp on by paired Student's t-test. A p value <0.05 was considered significant.

Results

Figure 1 shows the image of the CardioClasp device used to reshape the left ventricle. The anterior bar was placed along the anterior long axis of the left ventricle adjacent to the left anterior descending coronary artery. The posterior bar was placed along the posterior long axis of the left ventricle near the posterior descending coronary artery and not visible in this figure. F1

Figure 2 shows three representative left ventricle (LV) short axis echocardiographic views at the papillary muscle level at baseline and with the Clasp on. The LV short axis views were obtained at end-diastole. With the Clasp on, the LV anterior-posterior diameter was reduced by 28% (middle panel) and 15% (right panel). Fractional areas of contraction were increased by 21.9% at 28% diameter reduction and 11.9% at 15% diameter reduction. In group data, echo images revealed that the CardioClasp device reduced the LV anterior-posterior diameter by 31.8 ± 3.0%. Due to reduction in the LV anterior-posterior diameter, LV free and septal wall thickness increased slightly (10 ± 2.4%). F2

Figure 3 shows decreased myocardial LV wall stress with the Clasp on compared with baseline. With the Clasp on, LV wall F3

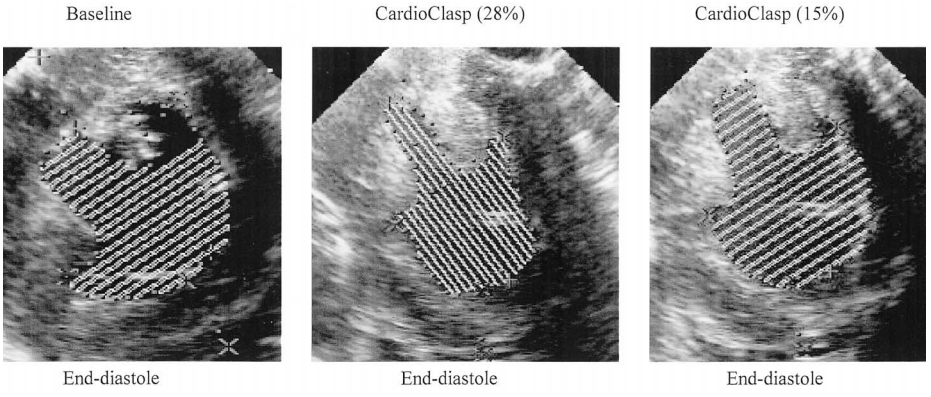

Figure 2. Representative left ventricle (LV) short axis end-diastolic echocardiographic views at papillary muscle level at baseline and with the Clasp on. With the Clasp on, the LV anterior-posterior diameter was reduced by 28% and 15%.

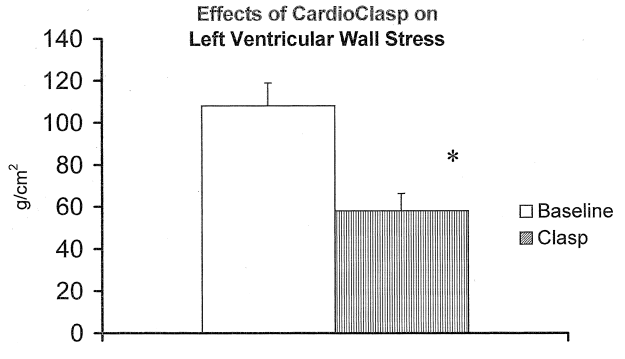

Figure 3. Effects of the CardioClasp device on left ventricular wall stress (LVWS). LVWS was significantly reduced with the CardioClasp on compared with baseline. (*$p < 0.05$; baseline LVWS *vs.* Clasp on LVWS)

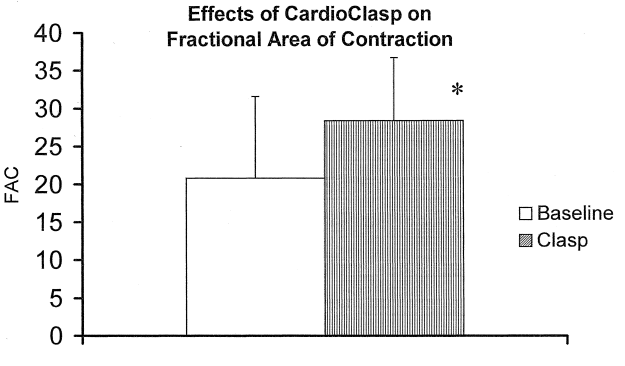

Figure 4. Effects of the CardioClasp device on the fractional area of contraction (FAC). (*$p < 0.05$; baseline FAC *vs.* Clasp on FAC)

stress was significantly decreased by approximately 46% ($p < 0.05$) from 108 to 59 g/cm^2. Baseline LV peak systolic pressure was not significantly altered compared with the Clasp on during LV wall stress measurement.

Figure 4 shows the fractional area of contraction at baseline and with the CardioClasp on. The fractional area of contraction significantly increased with the Clasp on ($p < 0.05$), changing from 20.8 ± 4.6 (baseline) to 28.4 ± 4.4 (with Clasp; $p < 0.05$), consistent with improved systolic performance.

Table 1 shows the hemodynamic parameters at baseline and with the Clasp on. With the Clasp on, cardiac output was unaltered (p = NS), as were peak LV systolic and end-diastolic pressures. There were no significant changes in LV +dP/dt (−2 ± 64 mm Hg/sec; $p = 0.26$) and LV −dP/dt (−25 ± 57 mm Hg/sec; $p = 0.38$) with the Clasp on.

In one experiment (n = 1), **Figure 5** shows three examples of end-systolic pressure-length loops generated during initial baseline and with the Clasp on at 15% and 25%. Also shown are the ESPSR measured during transient balloon obstruction of the inferior vena caval inflow. The data show a steepening of the ESPSR (with increased E_{es}) with simultaneous net leftward shift of the end-systolic pressure-length loop with the Clasp on at 15–25%. With the Clasp on, the pressure–segment-length relationship resulted in increased slope and end-systolic elastance (E_{es}), which reflected increased and improved contractility.

From one experiment, **Figure 6** shows regional myocardial blood flow at baseline and with the Clasp on. The dotted lines represent baseline regional myocardial blood flow, whereas the darker line represents blood flow with the Clasp on. Risk areas include the myocardium supplied by the coronary vessels crossing under the bar. In this example, regional myocar-

Table 1. Hemodynamic Parameters at Baseline and with the Clasp on

	LVEDP (mm Hg)	LVSP (mm Hg)	LV +dP/dt (mm Hg/s)	LV −dP/dt (mm Hg/s)	CO (L/min)
Baseline	13.1 ± 1.4	82.8 ± 2.7	950 ± 134	−860 ± 105	1.9 ± 0.3
Clasp	15.6 ± 2.0	80.7 ± 3.5	950 ± 129	−815 ± 178	1.8 ± 0.3

LVEDP, left ventricular end-diastolic pressure; LVSP, peak left ventricular systolic pressure; LV +dP/dt, maximum peak first positive derivative of the left ventricular pressure; LV −dP/dt, minimum peak first negative derivative of the left ventricular pressure; CO, cardiac output.

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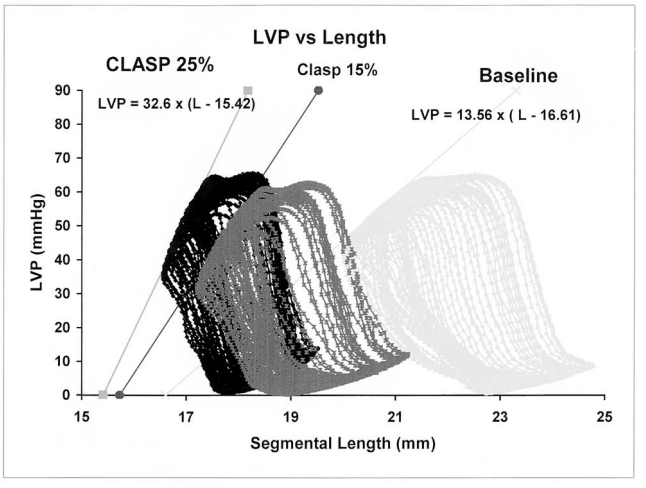

Figure 5. Pressure–segment-length loops generated during baseline and with the Clasp on at two levels. With the Clasp on, the pressure–segment-length loops were shifted to the left and the slope of the end-systolic pressure-length relationship increased. LVP, LV pressure.

dial blood flow in both risk and nonrisk areas was unaltered with the Clasp on.

T2 **Table 2** shows the nonrisk and risk areas of regional epi- and endomyocardial blood flow. With the Clasp on, regional epi- and endomyocardial blood flows were unaltered in both nonrisk and risk areas. The ratio of endocardial to epicardial blood flow showed no significant differences with the Clasp on.

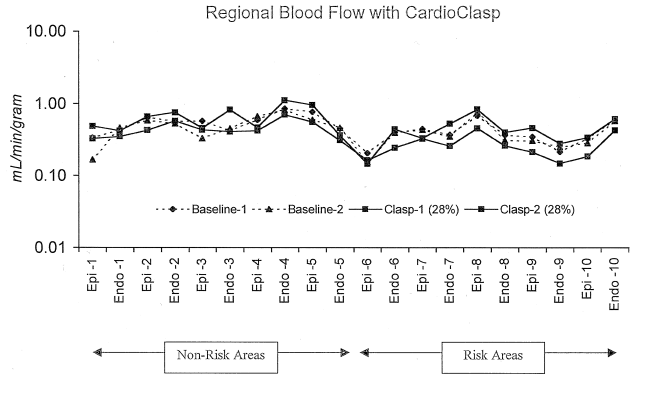

Figure 6. Effects of the CardioClasp on regional myocardial blood flow in risk and nonrisk areas. With the Clasp on, regional myocardial blood flow was unaltered compared with baseline. There were no significant differences in regional blood flow between risk and nonrisk areas.

Discussion

Melvin was the first to examine the concept of passive geometric ventricular remodeling using computational analysis.[3] The model showed that, by imposing a shape change on a dilated left ventricle, the ratio of wall tension to chamber pressure was reduced. If the reduction in wall tension occurred due to ventricular resection, then a substantial degree of contractile shortening improvement would be needed just to maintain baseline stroke volume. However, if that same reduction in wall tension occurred due to passive remodeling (due to the device), then that same proportional contractile shortening improvement would be associated with a stroke volume increase to 1.36 times baseline—because the salutary effect on the wall shortening fraction would not be negated by the severe reduction in baseline wall length that occurs with ventricular resection. He also showed that if contractile shortening was improved sufficiently to increase stroke volume to 1.2 and 1.5 times baseline with ventricular resection remodeling, the stroke volume would be increased to 1.7 and 2.1 times baseline with passive geometric ventricular remodeling.[3] Passive geometric ventricular remodeling has the added potential for preserving the contractile mass and circumferential length, is completely reversible, and would have minimal operative trauma.

Shimizu *et al.* examined the effects of the Clasp in an animal model of dilated heart failure.[15] In dogs, heart failure was produced using rapid ventricular pacing for 4 weeks. After heart failure was produced, the hearts were studied using an acute isolated heart preparation dog with support animal. The heart failure dog's heart was removed and attached to the system. A water filled balloon with a micromanometer tip transducer inside to measure LV pressure was placed into the LV. Coronary arterial pressure was held constant, and the perfusate was maintained at 35°C. The heart was paced at 10 to 15 bpm higher than the spontaneous rate.

The CardioClasp device was positioned on the heart and adjusted to reduce anterior-posterior end-diastolic dimension by 20–30%. The LV pressure during fluid withdrawal and rapid CardioClasp removal was measured. With the Clasp on, the end-systolic pressure–volume relationship (ESPVR) consisted of two ratios: boundary volume between these was 57.2 ml, and LV end-diastolic pressures at this LV volume were 13.6 mm Hg. ESPVR above boundary volume after placing the device was significantly increased from 1.4 at baseline to 2.4 mm Hg/ml, whereas ESPVR below boundary volume was not changed due to device placement. After taking off the device, ESPVR returned to almost baseline level (1.5) mm Hg/ml. They also showed that both systolic and diastolic pressures and developed pressure decreased with rapid removal of the CardioClasp.

Table 2. Regional Myocardial Blood Flow at Baseline and with the Clasp on

	Areas at Risk			Nonrisk Areas		
	Epi (ml/min/g)	Endo (ml/min/g)	Ratio (%)	Epi (ml/min/g)	Endo (ml/min/g)	Ratio (%)
Baseline	0.82 ± 0.26	0.76 ± 0.25	0.88 ± 0.07	0.84 ± 0.28	0.77 ± 0.28	0.87 ± 0.10
Clasp on	0.82 ± 0.18	0.79 ± 0.19	0.90 ± 0.07	0.74 ± 0.16	0.75 ± 0.16	1.01 ± 0.12

Epi, epicardial blood flow; Endo, endocardial blood flow; Ratio, ratio of epi- and endocardial blood flow.

In our experiments, the CardioClasp acutely reduced the LV anterior-posterior diameter by 31.8% as demonstrated by direct epicardial transthoracic echocardiography, and also significantly reduced the LV systolic wall stress. Our measurement of fractional area of contraction clearly showed the effective results from the passive CardioClasp device; fractional area of contraction was increased by 27%. Baseline LV systolic and diastolic pressures were not significantly altered compared with those with the Clasp on. Likewise, cardiac output was unaltered with the Clasp on. Regional blood flow was not affected by the Clasp.

Comparison with the Literature

In theory, the Myosplint is very similar to the CardioClasp. The Myosplint device allows the opposing walls of the failing heart to be drawn together, thereby decreasing the chamber radius with improved cardiac function. Each Myosplint implant consists of two epicardial pads or buttons connected by a tension member. A series of specialty instruments have been developed to allow for the implantation of the Myosplint device. Through the use of these instruments, the Myosplint buttons can be placed down the long axis of the left ventricle to create a bilobular shape.[16–19] In experimental studies, Takagaki *et al.* confirmed the shape changes by epicardial echocardiography and they were able to implant the device without any cardiopulmonary bypass. They showed that this passive device was associated with a 20% decrease in wall stress[17] and an increase in left ventricular ejection fraction. Cardiac output and ventricular pressures were unaltered. These benefits were maintained over 30 days.[16,17]

In humans, the Myosplint was placed just before removing the heart from a heart transplant patient. This passive device decreased peak LV systolic wall stress.[20]

Passive cardiac support. Cardiomyoplasty is a surgical treatment for heart failure, whereby the latissimus dorsi muscle is wrapped around the heart. Cardiomyoplasty with its passive support and girdling effects slow the progression of disease.[21,22] It has been suggested that preventing further ventricular dilatation may impede the progressive deterioration in cardiac function associated with heart failure. The hypothesis has been examined and found, in varying degrees and formats, that passive ventricular constraint alone improves outcome in comparison to control.[21,22]

The result from cardiomyoplasty has led to the development of passive devices to constrain the heart and prevent ventricular dilatation associated with heart failure. One such device is the AcornCorCap cardiac support device (CSD). The AcornCorCap CSD is a mesh-like jacket that is easily slipped around the heart and adjusted to provide ventricular support and prevent further dilatation.

In experimental studies, Sabbah *et al.* showed that percentage myocyte shortening was significantly increased in CSD treated heart failure dogs from 1.5% to 2.8%, and peak velocity of shortening (dS/dt) increased from 26 mm/sec to 48 μm/sec. He also reported shorter changes of peak velocity re-lengthening (dR/dt) from 13 to 40 mm/sec and time of 50% relaxation at stimulation frequency of 1 Hz from 129 mm to 56 mm.[23]

Power *et al.* reported increases of LV −dP/dt from 1,067 to 1,243 mm Hg/sec and decreases of left ventricular end-diastolic pressure from 11 to 7.8 mm Hg using the Acorn device in heart failure sheep and induced pacing.[24] They showed promising results with the Acorn CSD, although percentage reduction of the LV size and diameters are not obvious from their experiments.

Konertz *et al.* reported 10% decreases in LV end-diastolic diameter and 60% increases in LV ejection fraction using the Acorn device in patients.[25] They studied pressure-volume loops at 3 months postoperatively and concluded significant increases in systolic and diastolic function, no constriction of the heart, no coronary artery compression, and maintained coronary flow reserve to adenosine. Postoperatively, all their patients were in either Class II or I.

Summary and Conclusions

The goal of medical management of heart failure is to reduce afterload. This is usually achieved by lowering vascular resistance. The resulting lower arterial blood pressure can compromise blood flow to other vital organs, such as the brain and kidney. The CardioClasp device can reduce the tension on the myocardial cells without decreasing arterial blood pressure. This leads to improved LV systolic performance. Echo images showed that the CardioClasp reduced LV diameter thereby decreased LV wall stress and increasing the fractional area of contraction. The CardioClasp was able to reshape the left ventricle, while preserving the contractile mass. This reshaping was associated with maintained systolic pressures and cardiac output with increased fractional area of contraction.

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