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Less Invasive Surgical Management of Heart Failure by Cardiac Support Device Implantation on the Beating Heart

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INTRODUCTION

Heart failure is the leading cause for hospital admissions in the U.S. and accounts for a large fraction of health care costs. Myocardial dysfunction in chronic heart failure is due in part to progressive ventricular dilatation and remodeling. Medical management of symptoms without the reversal of the underlying process has been the mainstay of treatment. There are few good surgical therapies for heart failure. Passive ventricular containment allowing for reverse remodeling using the Acorn Cardiac Support Device (CSD) (Acorn Cardiovascular Inc., St. Paul, MN) has been shown to be feasible and safe in pre-clinical and early clinical trials.

Explanation

The remodeling process in heart failure is a result of neurohormonal activation and mechanical stress [Goldstein 1998]. The resultant ventricular dilatation increases biomechanical wall stress and creates stretch of the cardiac myocytes [Simpson 1999]. This myocte stretch causes change in the gene expression and stimulation of the neurohormonal activity with resultant myocte apoptosis and increases in the extracellular matrix [Baig 1999, Pan 1999, Minamisawa 1999]. Once begun, the remodeling process is auto-inductive, leading to further remodeling and progression of ventricular dysfunction and baring intervention, inexorably to end stage heart failure [Oz 2001]. The Acorn Cardiac Support Device is a passive constraint device that prevents ventricular dilatation and allows for ventricular remodeling. The device is made of a proprietary polyester mesh and is sutured to the epicardial surface of the heart. It is a multifilamentous yarn that is designed with bidirectional compliance to conform to the heart and assist in reshaping the heart to a more ellipsoid shape [Oz 2001]. It is attached to the heart encompassing both ventricles. The left ventricular end diastolic diameter (LVEDD) is reduced during surgery by 5 to 8 % Improvement in ventricular performance is not acute but occurs over time with prevention of left ventricular dilatation. By supporting the heart and reducing wall stress, the CSD may act to relieve stretching of the cardiomyoctyes, which in turn may help to downregulate adverse molecular changes responsible for remodeling. Stimulation of "reverse" remodeling may be responsible for the improvement in cardiac function seen in safety study patients. In preclinical studies using a microembolic canine model Sabbah and associates showed that in the CSD treated group there was a greater improvement in regional wall motion and ejection fraction than the control groupat the 3 month follow up [Sabbah 2000]. Mitral valve regurgitation was also eliminated in the CSD treated group.They also demonstrated that the CSD can minimize or prevent maladaptive gene expression and resultant phenotypic transformation [Sabbah 2000].

Clinical safety studies with a 3 and 6 month follow up were performed at Charite in Berlin Germany. Konertz and associates demonstrated that the CSD effectively limits progressive cardiac dilatation with a statistically significant decrease in heart size and increase in ejection fraction.improvement in quality of life was also seen in these patients [Konertz 2000]. Raman and his group in Melbourne Australia looked at a group that had coronary bypass with placement of the CSD. They also demonstrated that the device was well tolerated, all coronary bypasses were patent and cardiac dilatation was prevented [Raman 2000]. In both groups there was no evidence of constriction and no device related adverse events.

MATERIALS AND METHODS

Under the IRB approved protocol, a CSD was implanted in a 42-year-old male with dilated cardiomyopathy and NYHA Class III heart failure. The procedure was performed without cardiopulmonary bypass support or cannulation. Intraoperative transesophogeal echocardiography was used to determine pre and post implant cardiac dimensions (see Figure 1, ◎). Under general anaesthesia, a median sternotomy was performed. Cardiac sizing was performed to choose the appropriate CSD (see Figure 2, ◎). Using the Xpose (Guidant Corp. Cupertino, California) ,the heart was gently positioned and manipulated to allow hemodynamic stability for placement of the posterior sutures (Figure 3, ◎). The sized polyester mesh was then pulled up anteriorly, adjusted to fit the heart, (Figure 4, ◎).

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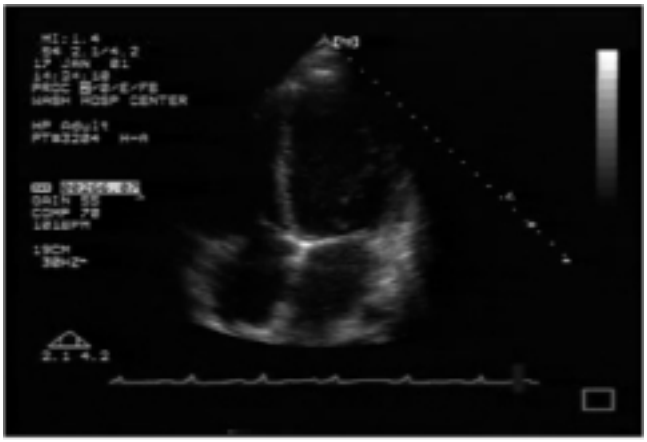

Figure 1. Intraoperative echocardiogram.

Figure 2. Sizing for CSD.

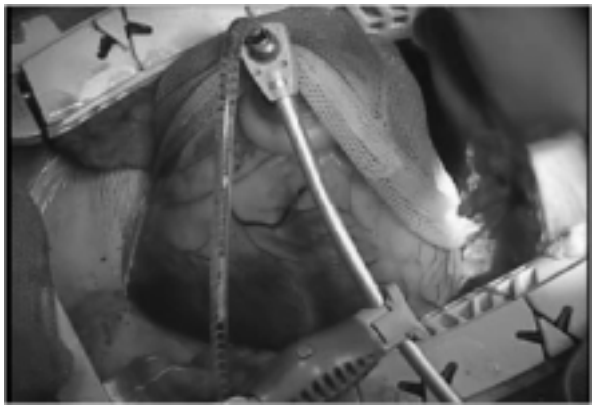

Figure 3. Placing posterior sutures.

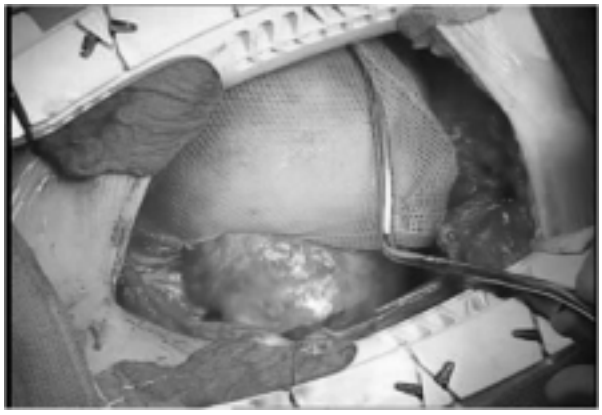

Figure 4. Containing and placing anterior sutures.

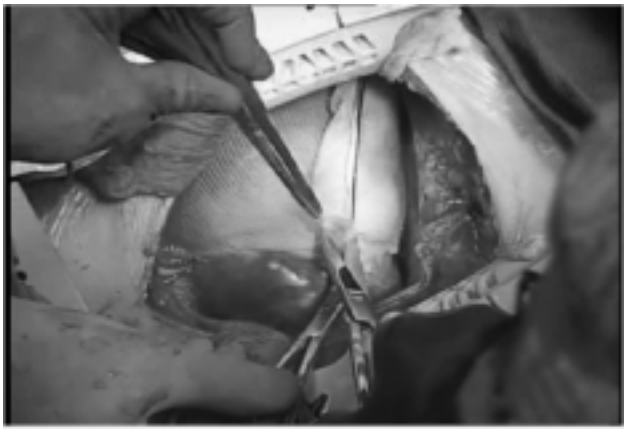

Figure 5. Trimming the anterior seam.

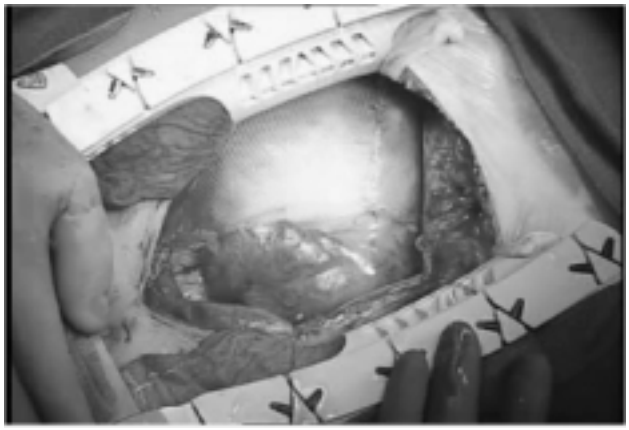

Figure 6. Final result.

A clamp was placed anteriorly on the CSD and the excess fabric was trimmed and the device tailored to fit snugly on the heart for the determined reduction (Figure 5, ◎). The final sutures anchoring the device to the anterior aspect of the heart were then placed (Figure 6, ◎).

RESULTS

The CSD was successfully implanted without the need for cardiopulmonary bypass. The patient maintained hemodynamic stability during the procedure.The Left Ventricular End Diastolic Diameter was reduced by 8% intraoperatively

Table 1. Pre and Post implant data.

	Pre-Implant	3 Months	6 Months
LVEDDI	35	33	32
LVEDD(MM)	61	57	56
LVESD(MM)	48	46	44
LVEF (%)	33	30	51
Peak VO2	19.1	NA	20.3
MR (0-4)	0	0	0
BP systolic (mmhg)	129	117	102
BP diastolic (mmhg)	90	70	60
Heart rate (bpm)	59	80	70
NYHA Class	III	II	II

LVEDDI – Left Ventricular End Diastolic Dimension Index, LVEDD - – Left Ventricular End Diastolic Dimension Index, LVESD – Left Ventricular End Systolic Dimension, LVEF – Left Ventricular Ejection Fraction, Mitral Regurgitation

(see Figures 4 and 5, ◎). The patient was discharged and three- and six-month follow-up data is shown in Table 1 (◎). At six months, the Left Ventricular End Diastolic Dimension Index, Left Ventricular End Diastolic Dimension, and Left Ventricular End Systolic Dimension decreased by at least 8%. The Left Ventricular Ejection Fraction increased by 63% from 33% to 51% and the patient New York Heart Association status improved from 3 to 2.

CONCLUSION

The Acorn Cardiac Support Device can be safely and successfully implanted with the less invasive beating heart approach for patients having this as sole surgical therapy for heart failure.

Disclosure

The U.S. randomized clinical study is sponsored by Acorn Cardiovascular, Inc., St. Paul, MN. Dr. Dullum is a paid consultant for Acorn Cardiovascular, Inc. in the development of implant procedures.

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