Echocardiographic findings using tissue velocity imaging following passive containment surgery with the Acorn CorCap™ cardiac support device

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Abstract

Objective: To echocardiographically evaluate the effects of passive containment surgery using the CorCap™ Cardiac Support Device in heart failure patients with dilated cardiomyopathy. Methods: Twelve patients with dilated cardiomyopathy subjected to cardiac surgery received the Cardiac Support Device. Patients with ischemic cardiomyopathy (n = 5) underwent coronary artery bypass surgery receiving 1–3 bypass grafts. In the idiopathic cardiomyopathy group (n = 7), mitral valve annuloplasty was performed in five patients while two patients received the Cardiac Support Device only. Results: Following surgery there was a gradual, sustained improvement in cardiac dimensions (decreased left ventricular end-diastolic diameter and left ventricular end-systolic diameter) combined with an increase in functional status (6-min walk and NYHA class). Concomitantly there was a marked decrease in right ventricular function (decrease in tricuspid annular systolic and diastolic velocities) while the left ventricular function (mitral annular systolic and diastolic velocities) and output (ejection fraction, stroke volume) remained unchanged. Conclusions: Addition of the Cardiac Support Device to conventional cardiac surgery improves patient status and decreases left ventricular size in heart failure patients with dilated cardiomyopathy. The positive effect on left ventricular dimensions is not accompanied by any improvement in cardiac output but rather right ventricular dysfunction, although the functional significance of this is unclear.

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Keywords: CorCap™ Cardiac Support Device; Dilated cardiomyopathy; Heart failure; Passive containment surgery; Tissue velocity imaging

1. Introduction

In spite of marked improvement in the medical treatment of advanced heart failure, the prognosis is still poor with a high mortality rate, especially for those with a dilated heart [1]. The dilating left ventricle (LV) of the failing heart exposes myocytes to increased wall stress leading to a number of adaptive mechanisms and eventually to further ventricular dilatation with additional deterioration of LV function. In this ongoing auto-inductive process, often referred to as ventricular remodeling, cardiac function will worsen until end-stage heart failure is reached.

Surgical treatment of advanced heart failure includes heart transplantation, which, however, is limited due to organ shortage, limited long-term survival, associated morbidity and high costs. Therefore, different surgical ventricular shape changing procedures have been evaluated for these patients [2-4]. One such device is passive ventricular volume containment as produced by the CorCap™ Cardiac Support Device (CSD; Acorn Cardiovascular, Inc., St Paul, MN, USA) [5]. The CSD is a mesh-like polyester fabric with bi-directional compliance, which is positioned around the heart during surgery in order to reduce wall stress and reshape the heart from a dilated, spherical shape to an ellipsoidal shape.

The CSD has been demonstrated to reduce adaptive mechanisms associated with increased wall stress and to attenuate further ventricular dilatation and improve cardiac function in various experimental heart failure models. In addition, short and intermediate term results with the CSD in heart failure patients with dilated LV indicate amelioration of symptoms and improved cardiac function [5,6].

In this paper, we present the echocardiographic findings in 12 patients with ischemic or non-ischemic cardiomyopathy (CM) subjected to cardiac surgery with the use of the CSD.

2. Methods

2.1. Patients

Between June 2001 and February 2004, 12 patients (10 males, 2 females) with idiopathic or ischemic CM received the CorCap™ CSD. The study was approved by the Local Ethical Committee at the Karolinska University Hospital and written consent was obtained from all patients.

Inclusion criteria which all had to be fulfilled included (I) left ventricular end-diastolic diameter (LVEDD) of >60 mm or indexed to >30 mm/m² body surface area, (II) an
Table 1 Pre-operative characteristics and surgical procedure of 12 patients subjected to passive containment surgery

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age</th>
<th>NYHA</th>
<th>MR</th>
<th>HF duration</th>
<th>Surgery (in addition to CSD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66</td>
<td>3</td>
<td>1</td>
<td>7</td>
<td>LITA-D1</td>
</tr>
<tr>
<td>2</td>
<td>69</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>LITA-LAD, SVG-RPD</td>
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<td>3</td>
<td>50</td>
<td>3</td>
<td>1</td>
<td>2</td>
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</tr>
<tr>
<td>4</td>
<td>58</td>
<td>4</td>
<td>2</td>
<td>8</td>
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</tr>
<tr>
<td>5</td>
<td>44</td>
<td>3</td>
<td>2</td>
<td>4</td>
<td>C-E Physioring, 30 mm</td>
</tr>
<tr>
<td>6</td>
<td>61</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>LITA-LAD, SVG-M2</td>
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<td>7</td>
<td>78</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>-</td>
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<tr>
<td>8</td>
<td>72</td>
<td>3</td>
<td>1</td>
<td>3</td>
<td>LITA-LAD, SVG-M1, RP1</td>
</tr>
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<td>9</td>
<td>33</td>
<td>2</td>
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<tr>
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<tr>
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<td>52</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>LITA-LAD, SVG-RPD, M1</td>
</tr>
<tr>
<td>12</td>
<td>59</td>
<td>2</td>
<td>3</td>
<td>8</td>
<td>C-E Physioring, 30 mm</td>
</tr>
</tbody>
</table>

LITA, left internal thoracic artery; LAD, left anterior descending coronary artery; D, diagonal branch of the LAD; RP1, right posterior descending coronary artery; M1, marginal branch of the circumflex artery; SVG, saphenous vein graft; HF, heart failure (duration in years); NYHA, New York Heart Association functional class; MR, mitral regurgitation (grade 1–4/4).

ejection fraction (EF) of 10–45%, (III) NYHA class III or IV, or when in class I only if a history of at least one previous class III or IV episode, (IV) stable and optimal drug therapy, (V) mitral regurgitation (MR) <2+ (unless accepted for MR surgery). Five of the patients were accepted for coronary artery bypass grafting (CABG; ischemic CM) and the remaining seven were initially accepted for mitral valve surgery although two of these patients received the CSD alone due to MR <1+ at the time of surgery (idiopathic CM). The patient characteristics are presented in Table 1.

2.2. Medications

Prior to surgery, all patients were on β-blockade, ACE-inhibitor (n=11) or All-blocker (n=1) and diuretics. This was maintained throughout follow-up. Major pharmacological treatment is presented in Table 2.

2.3. Device implant

All patients were operated on through a midline sternotomy by the same surgeon. Cardiopulmonary bypass (CPB) was initiated using a centrifugal pump (BP 80, Biomedicus Biomed, Houston, TX, USA) and a membrane oxygenator (Affinity, Medtronic Inc, Minneapolis, MN, USA) primed with acetated Ringer’s solution. During CPB, the temperature was allowed to drift to 34 °C. The cardioplegia solution was mixed 1:4 with blood and delivered at a temperature of 4 °C. The cardioplegia contained (mmol) KCl 100, MgSO4 8, glucose 28, THAM 20 and saline 0.9% to a volume of 1000 ml. Overall CPB was 94 ± 14 min (n=12) and cross-clamp was 58 ± 8 min (n=10). For the two patients receiving the CSD alone, cross-clamp was not used. CABG patients received an average of two grafts (1-3) with the left internal thoracic artery (LITA) used in all patients and when needed additional saphenous veins were used as graft material. Five patients in the idiopathic group underwent mitral valve surgery with a Carpentier-Edwards Physioring (see Table 1).

Before applying the CSD, the heart size was measured (apex-base and circumference) to select proper sizing as the device comes in six different sizes. With the CSD hemline positioned adjacent to the atroventricular groove, the device was stabilized by interrupted 3-0 Prolene sutures approximately 20 mm apart. Final adjustment was done on the fully beating heart to ensure a snug fit. Transesophageal echocardiography was used to obtain a final fit reducing the LVEDD by 6 ± 1% compared to baseline.

2.4. Anesthesia

All patients received their routine daily cardiac medications on the morning of surgery. All standard American Society of Anesthesiologists’ non-invasive monitors and peripheral intravenous and intra-arterial catheters were placed before induction. Anesthesia was induced using fentanyl (10-20 μg/kg) and midazolam (0.03-0.05 mg/kg). Intubation was facilitated with atracurium. Patients were ventilated to achieve normocapnia (PaCO2 35–45 mmHg). A flow directed pulmonary artery catheter and transesophageal echocardiography probe were inserted after induction. Anesthesia was maintained with intermittent fentanyl and isoflurane pre-CPB and an infusion of propofol (1-4 mg/kg per h) during and after CPB. Based on our experience with levosimendan (Simdax; Orion Corporation, Espoo, Finland) in patients with low EF undergoing cardiac surgery, all patients received levosimendan as an inotrope starting with a loading dose of 12 μg/kg prior to skin incision, followed by a continuous infusion of 0.1 μg/kg per min during 24 h.

2.5. Patient follow-up/echocardiography

Functional status was assessed by 6 min walk[7] and evaluation of NYHA class prior to surgery and at 3, 6 and 12 months postoperatively. At the same time, all patients underwent echocardiographic examinations including pulsed wave tissue velocity imaging (TVI) using a commercially available equipment (System 5, GE Vingmed, Horten, Norway) equipped with 2.5-MHz phased array transducers. All examinations were performed by the same physician with the subjects in the left lateral decubitus position. The parasternal long-axis view was used to derive the diameter of aorta and left atrial dimensions at end-systole. The diameter of the left ventricular outflow tract (LVOT) was measured during early systole from the junction of the aortic leaflets with septal endocardium, to the junction of the leaflet with the mitral valve posteriorly, using the inner edge to inner edge. Cross-sectional area of the LVOT (CSA, LVOT) was calculated as πr². Left ventricular (LV) cavity size was measured at end-diastole and end-systole and the wall thickness...
thickness at end-diastole. The apical views were used to calculate ejection fraction using the modified Simpson's rule. The end-expiratory diameter of the inferior vena cava (IVCexp) was measured from a subcostal view.

2.6. Doppler studies

Transmitral flow velocities were recorded during early inspiration and end-expiration with pulsed wave Doppler from the apical four-chamber view by placing the sample volume between the leaflet tips in the center of the flow stream. The transmitral peak rapid filling velocity (E), peak atrial filling velocity (A), E-wave deceleration time (DT), and E/A ratio were measured as indices of LV diastolic filling. Changes in peak rapid filling velocity during respiration were calculated as \((E_{\text{exp}} - E_{\text{insp}}) / E_{\text{exp}} \times 100\) and was used together with the transmitral flow velocities to detect LV constriction. The LVOT outflow velocity was recorded with pulsed wave Doppler from the apical five-chamber view by placing the sample volume about 5 mm proximal to the aortic valve. The sample volume position was then adjusted to get a high-quality signal that showed the closing click but not the opening click of the aortic valve. Velocity-time integral (VTI\textsubscript{LVOT}) and heart rate (HR) were also measured. Stroke opening click of the aortic valve. Velocity-time integral quality signal that showed the closing click but not the

The sample volume position was then adjusted to get a high-quality signal that showed the closing click but not the opening click of the aortic valve. Velocity-time integral (VTI\textsubscript{LVOT}) and heart rate (HR) were also measured. Stroke volume (SV) was then calculated as the product of the VTI\textsubscript{LVOT} and CSA\textsubscript{LVOT} and cardiac output as the product of SV and HR. All measurements were averaged from three beats.

2.7. Myocardial velocities using TVI

In the apical four-chamber, apical two-chamber and apical long axis views the mitral annular velocities were recorded at six sites corresponding to the septal, lateral, anterior, inferior, anteroseptal, and inferolateral LV wall (Fig. 1). Tricuspid annular velocities were recorded at the free RV wall in the apical four-chamber view.

The online analysis included measurements of peak systolic annular velocities \((S_m)\), early diastolic \((E_m)\) and late diastolic \((A_m)\) annular velocities (Fig. 1). All measurements were made during end expiration and averaged from three beats. A mean value of peak systolic, early diastolic and late diastolic mitral annular velocities from the above six different sites was used as a measurement of global LV systolic \((S_m)\) and diastolic \((E_m\) and \(A_m)\) function. The tricuspid annular velocities were used as a measurement of global RV systolic \((S_m)\) and diastolic \((E_m\) and \(A_m)\) function. The LV \(E_m\)/LV \(A_m)\) ratio and the RV \(E_m) /RV A_m)\) ratio were also calculated.

The ratio of early mitral inflow to early mitral annular velocity was calculated both as \(E/LV E_m\) and as the ratio of \(E \) and \(E_m\) registered from the septal mitral annulus \((E/\text{Septum E}_m)\) and were used as measurements of LV filling pressure.

2.8. Statistical evaluation

Data are presented as mean\(\pm\)SEM. For statistical analysis, repeated measures of variance (ANOVA) with Bonferroni comparison was used. A \(P<0.05\) was considered significant.

3. Results

All patients survived the surgical procedure and have left the hospital and the follow-up is complete.

Heart rate, LVEDD, LVESD, SV, CO and MR could be evaluated in all 12 patients. The rest of the echocardiographic parameters were obtained in 9-11 patients at each study point due to poor acoustic windows, atrial flutter or pacemaker rhythm.

Following surgery, there was a decrease in MR (from 1.7\(+\)0.2 to 1.0\(+\)0.1; \(P<0.05) and NYHA (from 2.8\(+\)0.2 to 1.8\(+\)0.2; \(P<0.001) while the 6 min walk improved (from 388\(+\)27 to 474\(+\)34; \(P<0.05) at 12 months follow-up.

3.1. Two-dimensional measurements

At 12-months follow-up, the LVEDD had decreased from 68\(+\)2 to 62\(+\)3 mm \(P<0.05) and LVESD had decreased from 60\(+\)2 to 53\(+\)3 mm \(P>0.05;\) Fig. 2a). The corresponding changes in the two patients receiving the CSD only were similar (from a mean of 65-56 mm in LVEDD and 57-45 mm in LVESD at 12-months follow-up, respectively). No changes were observed in the left atrium or inferior vena cava diameters.

3.2. Systolic variables

There were no statistical significant changes in EF, CO or SV during follow-up of the patients (Fig. 2b). Similarly, mitral annular peak systolic velocities \((LV S_m)\) remained unchanged \(Fig. 2c\), while tricuspid annular peak systolic velocities \((RV S_m)\) decreased from 9.5\(+\)0.7 to 7.4\(+\)0.4 cm/s \(P<0.05) 12 months postoperatively (Fig. 2d).

Fig. 1. Myocardial velocity measurement sites (■) in the apical four-chamber, two-chamber and long axis (APLA) views. The annular measurement principle of systolic velocity \((S_m)\) and early \((E_m)\) and late \((A_m)\) diastolic velocities are also shown.
3.3. Diastolic variables

The transmitral flow velocities (E, A), the diastolic mitral annular velocities (LV E\text{m}, LV A\text{m}), the E/A and LV E\text{m}/LV A\text{m} ratios, the E-wave deceleration time (DT), the E/LV E\text{m} and E/Septum E\text{m} ratios all remained unchanged throughout the study period (Fig. 2c). Also the change of peak rapid filling velocity (E) during respiration, calculated as (E_{\text{exp}}-E_{\text{insp}})/E_{\text{exp}}\cdot100 was unchanged following surgery.

Tricuspid annular diastolic velocities, i.e. RV E\text{m} decreased from 8.8±1.1 to 6.3±0.5 cm/s (P<0.01), and RV A\text{m} decreased from 14.1±1.7 to 6.5±0.9 cm/s (P<0.001) at 12 months after surgery (Fig. 2d). The corresponding changes in the two patients receiving the CSD only were similar (from a mean of 12.7 to 5.2 cm/s in RV E\text{m} and 14–7.2 cm/s in RV A\text{m} at 12 months follow-up, respectively)

4. Discussion

In this study, we have demonstrated that passive containment surgery in heart failure patients with dilated CM is associated with functional improvement (i.e. NYHA class and 6 min walk) and reverses ventricular dilatation. This occurs in spite of any clear-cut positive effects on LV function, and in combination with marked negative effect on RV function.

Congestive heart failure due to ischemic or idiopathic cardiomyopathy is characterized by left ventricular dilatation and ventricular remodeling. As a consequence, the left ventricle becomes spherical, the wall thinner with abnormal motion and myocardial oxygen consumption increases concomitantly with decreased EF. Hemodynamic unloading by cardiac support devices as well as surgical procedures such as partial left ventriculectomy can reverse the ventricular remodeling process of the failing heart [8,9].

LV diastolic and systolic dimensions decreased in the study group combined with a slight, non-significant increase in EF, although enough to maintain stroke volume and cardiac output. This suggests that although the MR decreased significantly following surgery, increased antegrade flow does not explain the effects on patient status. In accord, it has earlier been reported that CSD failed to improve total cardiac output [10]. Similarly we could not demonstrate any changes in mitral annular peak systolic or diastolic velocities throughout the study period. The beneficial effects on patient status by the surgery may be related to other effects on the LV such as decreased wall tension and attenuation of muscle over-extension resulting in decreased myocardial oxygen requirement with subsequent reversed ventricular remodeling. Indeed, earlier studies have demonstrated reduced left ventricular muscle mass following CSD implantation [10].

Diastolic variables did not indicate any obvious change in LV diastolic function and there were no signs of LV constriction or altered LV filling pressures. Experimental studies using pressure-volume relationships have earlier failed to demonstrate any increase in chamber stiffness or diastolic filling pressures by the CSD [11]. When the CSD reduces wall stress by compliance in the device with a gradual reduction of LV diameter, impairment of diastolic function is therefore not a major concern. This is in contrast to surgical shape changing procedures, i.e. partial left ventriculectomy, which has been shown to be associated with negative effects on diastolic pressure-volume
relationships countering the positive effects on systolic improvement [12].

Tricuspid annular velocities were evaluated as variables of RV systolic and diastolic function [13] in this study and according to the measurements, a marked reduction in RV systolic and particularly in RV diastolic function occurred postoperatively. No improvement was seen during the follow-up period. Impaired RV function is a known effect of cardiac surgery [13,14]. In our two groups of ischemic and non-ischemic dilated CM patients, we detected similar preoperative LV and RV function and no group differences in postoperative RV dysfunction were observed. This is important since RV function has been suggested to be more altered in idiopathic CM patients compared to ischemic patients [15]. Furthermore, the present observation on postoperative RV dysfunction is in agreement with findings on RV dysfunction following coronary bypass surgery [13], and thus unlikely to be related to any containment effect of the CSD per se. Although suggested to be of no clinical significance [13], the natural course of postoperative right ventricular function in CM patients is largely unstudied. Moreover, the long-term effects of the right ventricular function following passive containment surgery remain to be established.

4.1. Study limitations

All our patients were long-term treated (more than 3 months of unchanged medication preoperatively) with drugs known to partially prevent and also reverse ventricular remodeling [16,17]. In spite of this, ventricular function was poor suggesting that although beneficial, the sustained effects of pharmacological treatment may be limited in certain patients. Also mitral valve surgery [18,19] or CABG [20,21] may induce reversed remodeling of the dilated failing heart. Preoperatively all of our patients had mild (MR 1 +; n = 7) to moderate (MR 2–3 +; n = 5). MR. Of these, only five patients underwent mitral valve annuloplasty and in one of them the MR postoperatively is the same as before surgery (2 +). We therefore do not believe that mitral valve surgery plays any major role in the effects observed in these patients. To what extent adding mitral valve surgery to CSD implantation has any additional positive effect on LV dimensions compared to CSD implantation alone remains to be further evaluated [10]. With regards to effects of CABG surgery in LV dilatation and low EF, the best results are achieved in patients with proximal three-vessel disease [21]. This was the case of only two of five patients in this study. Although the present patient group is small in numbers we therefore believe that the effects on cardiac dimensions observed in our patients are achieved by the containment surgery. It is important to emphasize though that we cannot discriminate what surgical procedure underlies the observed patient functional improvement.

In conclusion, the present study demonstrates that passive containment surgery is associated with improved patient status and decreased left ventricular size in heart failure patients with dilated CM although without improvement in CO. The observed RV dysfunction is unlikely to be related to the CSD device per se.

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References


