



# Percutaneous implantation of the left ventricular partitioning device for chronic heart failure: a pilot study with 1-year follow-up

Dragan Sagic<sup>1</sup>, Petar Otasevic<sup>1\*</sup>, Horst Sievert<sup>2</sup>, Albrecht Elsasser<sup>3</sup>, Veselin Mitrovic<sup>3</sup>, and Sinisa Gradinac<sup>1</sup>

<sup>1</sup>Dedinje Cardiovascular Institute, Belgrade, Serbia; <sup>2</sup>Sankt Katharinen Hospital, Frankfurt, Germany; and <sup>3</sup>Kerckhoff Clinic, Bad Nauheim, Germany

Received 7 November 2009; revised 9 February 2010; accepted 12 February 2010

<b>Aims</b>	To assess short-term safety defined as the successful delivery and deployment of the ventricular partitioning device (VPD) implant, as well as 12-month functional, clinical, and haemodynamic effectiveness.
<b>Methods and results</b>	Ventricular partitioning device implantation was successful in 15/18 (83%) patients with anteroapical regional wall motion abnormalities following myocardial infarction. In one patient, the VPD was removed 3 days post implantation and the patient subsequently died due to extra-cardiac sepsis. When compared with baseline, there was significant improvement at 6 and 12 months following VPD implantation in NYHA class ( $2.21 \pm 0.57$ vs. $1.28 \pm 0.46$ vs. $1.23 \pm 0.43$ , respectively, $P < 0.001$ for both), left ventricular (LV) end-systolic volume ( $189 \pm 45$ vs. $142 \pm 29$ vs. $151 \pm 48$ mL/m <sup>2</sup> , respectively, $P < 0.001$ for both), and LV end-diastolic volume ( $260 \pm 47$ vs. $208 \pm 33$ vs. $222 \pm 58$ mL/m <sup>2</sup> , respectively, $P < 0.001$ for both). After 12 months, an improvement in LV ejection fraction was noted ( $28 \pm 7$ vs. $32 \pm 7$ vs. $33 \pm 9\%$ , respectively, $P = 0.02$ ) as well as improvement in 6 min walk distance ( $382 \pm 123$ vs. $409 \pm 7$ vs. $425 \pm 140$ m) when compared with pre-procedural values.
<b>Conclusion</b>	Our data indicate that VPD implantation is safe and feasible, and that VPD implantation improves LV haemodynamics and functional capacity in the 12 months following the procedure.
<b>Keywords</b>	Heart failure • Ventricular partitioning device • Remodelling

## Introduction

Patients with moderate or severe heart failure have a poor long-term prognosis and despite major advances, long-term medical treatment alone may be insufficient to improve outcomes. Thus the search for therapies with mechanisms of action other than neurohormonal antagonism appears justified.<sup>1,2</sup>

The deleterious changes in left ventricular (LV) size and shape following myocardial infarction (MI), termed ventricular remodelling, are increasingly recognized as potential targets for therapeutic intervention. Mechanical burden associated with LV remodelling leads to increased myocardial wall stress, which may lead to further impairment in LV function and symptomatic deterioration.<sup>3</sup> The ventricular partitioning device (VPD) (Cardiokinetix Inc., Menlo Park, CA, USA) is a new medical device, which is deployed

in the LV of patients with anteroapical regional wall motion abnormalities following a MI to partition the ventricle and segregate the dysfunctional region. The VPD implant is delivered percutaneously from the femoral artery using standard techniques for left heart catheterization. It is hypothesized that the VPD will reduce LV volumes, decrease myocardial wall stress, and, hence, improve LV haemodynamics.

The Percutaneous Ventricular Restoration In Chronic Heart Failure (PARACHUTE) study is a first-in-man, prospective, non-randomized, multicentre clinical trial with the primary aim to assess short-term safety of the VPD defined as the successful delivery and deployment of the device from implant through hospital discharge without the occurrence of major adverse cardiovascular events (death, MI, and stroke) or other serious adverse events. Secondary endpoints included device performance and long-term

\* Corresponding author. Tel: +381 113601669, Fax: +381 113601783, Email: potasevic@yahoo.com

Published on behalf of the European Society of Cardiology. All rights reserved. © The Author 2010. For permissions please email: journals.permissions@oxfordjournals.org.

safety (late major cardiovascular adverse cardiac or serious adverse events), as well as functional (symptom level, exercise capacity, and quality of life), and haemodynamic [LV volumes, LV ejection fraction (LVEF), and LV end-diastolic pressure (LVEDP)] evidence of device effectiveness at 6 and 12 months.

## Methods

### Patients

Between October 2005 and October 2006, a total of 41 patients were screened for VPD implantation in three centres (one in Serbia and two in Germany). Patient flow through the study is depicted in *Figure 1*. A total of 18 patients were selected for implantation. Other patients refused to participate in the study or did not meet inclusion/exclusion criteria. Major inclusion criteria were (i) anteroapical wall motion abnormality of the LV following MI, as detected by echocardiogram; (ii) LVEF <40% and evidence of LV dilation; (iii) NYHA class  $\geq 2$ ; (iv) eligibility for cardiac surgery; (v) age  $\geq 18$  years, and (vi) signed written informed consent. Major exclusion criteria were: (i) MI within 3 months prior to enrolment; (ii) ischaemia requiring percutaneous or surgical revascularization; (iii) revascularization procedure within 30 days of enrolment; (iv) significant valve disease requiring surgery, and (v) echocardiographic evidence of thrombus in the left ventricle. The study protocol was approved by the appropriate Institutional Ethics Committees, and the investigation conforms to the principles outlined in the Declaration of Helsinki. Written consent was obtained from all patients prior to the procedure.

### Device components

The VPD Implant System is comprised of three components: the Access System, the Delivery System, and the VPD implant. The VPD Access System consists of a guide catheter and dilator to provide

access to the LV. The guide catheter is 14F and the internal lumen is sized to accommodate a collapsed VPD implant. An injection port is attached to the proximal end of the guide catheter and allows for injection of contrast media. A radiopaque band is mounted at the distal tip of the guide catheter. The dilator is sized to accommodate a 6F pigtail catheter. The dilator is placed inside the guide catheter to prevent kinking while gaining access to the LV.

The delivery catheter is used to deliver and position the VPD implant in the LV (*Figure 2A*). The central lumen provides a channel for the torque shaft, at the distal end of which is a screw that engages the VPD implant. By rotating the detachment knob at the proximal end of the delivery catheter, the VPD implant can be attached or detached. The balloon is designed to push against the struts of the VPD implant when inflated, to ensure full expansion of the VPD implant and engagement of struts into the tissue of the LV wall.

The VPD implant, consisting of an ePTFE membrane bonded to an expanded Nitinol frame (*Figure 2B*), is comprised of a self-expanding frame, a membrane, and an atraumatic foot. The frame has 16 struts and the tip of each strut ends in an anchor. The purpose of the anchors is to engage the tissue, stabilize the VPD implant, and prevent dislodgment and migration after the VPD implant is detached from the delivery catheter. Once the VPD implant is expanded, the membrane provides a barrier to seal-off the static chamber on the distal side of the VPD implant.

### Ventricular partitioning device implantation technique

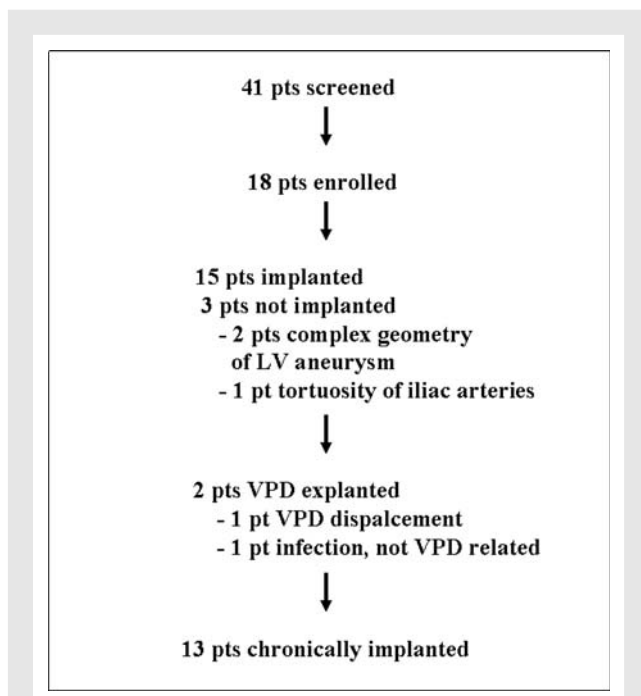
Prior to treating the first patient, the implanting physicians have completed VPD System training consisting of didactic session and hands on device training in an *in vitro* model along with implantation in a large animal model. Ventricular partitioning device implantation patients were prepared as for a left heart catheterization procedure according to the hospital's standard procedures. Left ventriculography was performed in two projections (left/right anterior oblique) to confirm LV sizes measured by baseline echocardiogram. Ventricular partitioning device implant landing zone diameter was measured 40 mm from the apex in both the short- and long-axis views or in two perpendicular long-axis views to confirm appropriate VPD implant size. Ventricular partitioning device implant size is determined by calculating a 30–60% over-sizing of the VPD implant diameter relative to the largest diameter of the LV measured at end-diastole. During this study, the VPD was available in 75 mm (for LV's with diameters at the VPD attachment zone of 47–53 mm) and 85 mm sizes (for LV's with diameters at the attachment zone of 54–60 mm).

In all patients, VPD implantation was performed via the right femoral artery using a 14F guide catheter. The collapsed VPD implant was attached to the delivery catheter, and advanced retrogradely through the guide catheter across the aortic valve and positioned in the LV apex. An LV angiogram was performed to assess the appropriate positioning. Once in place (*Figure 3A*), the VPD implant was expanded by the compliant balloon located proximal to the screw connector (*Figure 3B*), and released using the distal screw mechanism (*Figure 3C*).

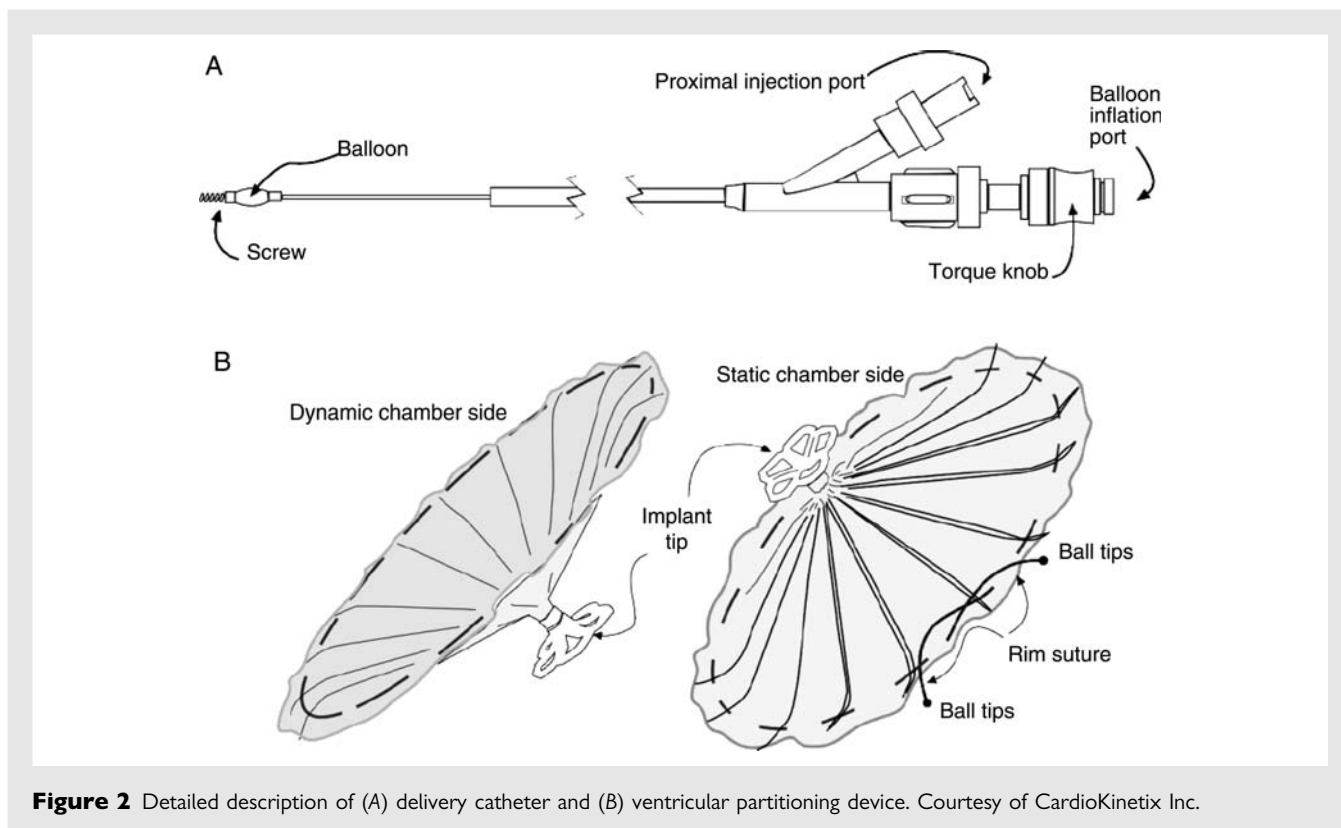
Control left ventriculography was performed to assess the VPD position and any possible residual leak between the walls of the LV and the device. *Figure 4* shows computerized tomography at 9-month follow-up, showing the VPD at the LV apex (black arrows) and masses on both sides of the device (white arrows).

### Concomitant medications

Four days prior to the procedure, all patients were placed on anti-platelet therapy consisting of 75 mg/day of clopidogrel and



**Figure 1** Patient flow through the study. Abbreviations: pt, patient; VPD, ventricular partitioning device.



325 mg/day of aspirin. During the procedure, patients received intravenous boluses of unfractionated heparin in sufficient doses to prolong the activated clotting time  $>250$  s. At the time of implantation and upon discharge, all patients also received optimal therapy for chronic heart failure consisting of an angiotensin converting enzyme inhibitor/angiotensin receptor blocker, beta-blocker, and diuretic. In addition, patients were required to continue on the 75 mg/day of clopidogrel and 325 mg/day of aspirin through 6 months post procedure. Coumadin (or warfarin) was recommended for the same period of time, and patients with suspected thrombi were continued on oral anticoagulants indefinitely with a target INR of 2.0–3.0.

### Echocardiography

Complete two-dimensional transthoracic echocardiographic and Doppler examinations were performed in all patients at baseline, and after 6 and 12 months. Left ventricular ejection fraction and LV volumes were determined from the apical four- and two-chamber views using Simpson's biplane formula. Competence of the VPD implant seal was evaluated by colour Doppler echo performed at the site of the VPD implant attachments to the myocardium. The extent of leakage was assessed semiquantitatively, and graded by the core echo lab as either no leakage, or mild, moderate, or severe leakage.

### Symptoms, functional capacity, and quality of life

Symptoms were evaluated by experienced cardiologists who were unaware of the patient's participation in the study. Dyspnoea on exertion was assessed at baseline, and after 6 and 12 months using the NYHA classification. Functional capacity was assessed using the standard 6 min walking test at the same three time points. In addition, change in quality of life when compared with baseline was determined

using the Minnesota Living with Heart Failure Questionnaire at 6 and 12 months.

### Left ventricular end-diastolic pressure

Left ventricular end-diastolic pressure was determined pre-procedure, immediately following VPD implantation and after 6 months, by standard left heart catheterization using fluid-filled catheters.

### Laboratory assessment

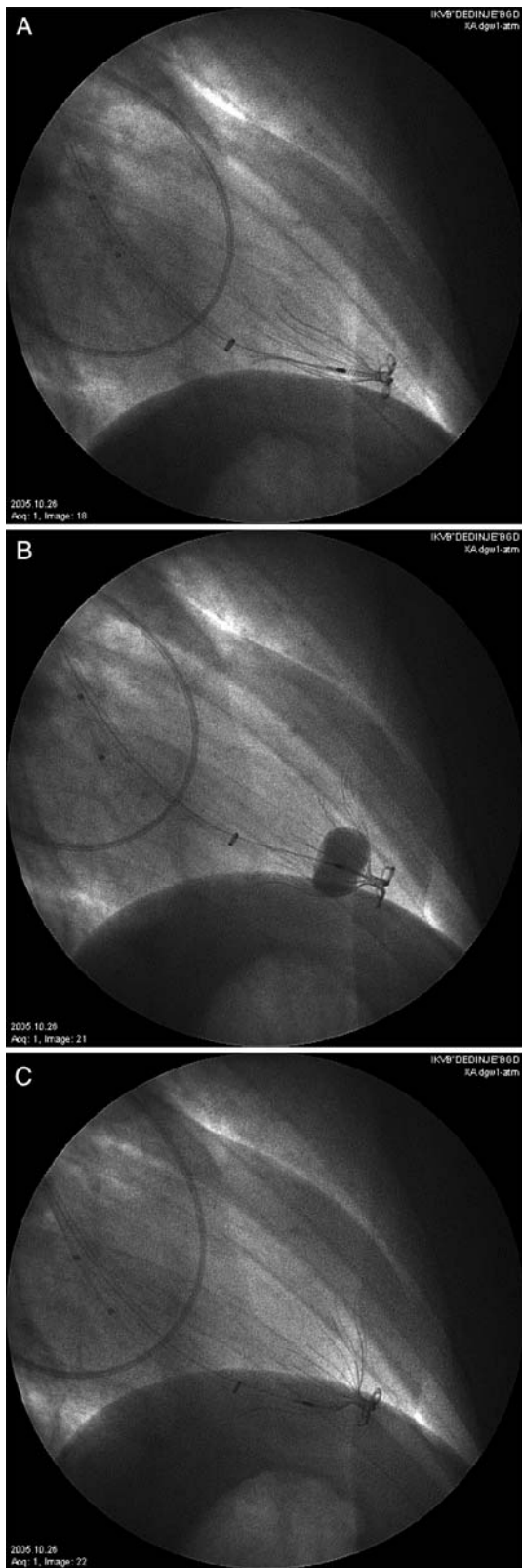
Serum creatinine and blood urea nitrogen were measured at baseline, 24 h after VPD implantation, and at discharge to assess the impact of the procedure on renal function. Additionally, troponin T was measured at the same time points, to assess possible myocardial damage associated with the procedure.

### Events

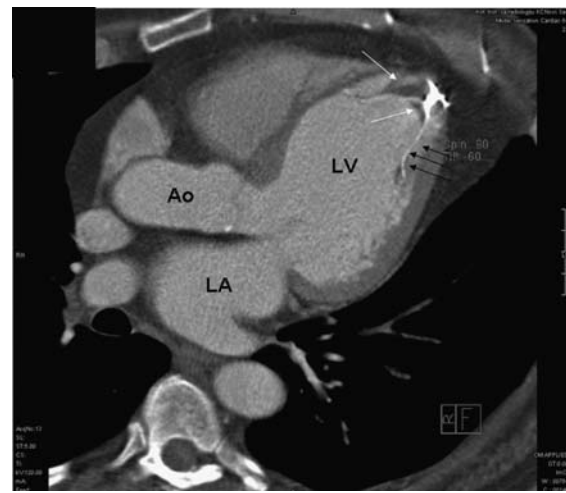
All patients were followed for 12 months. When either the patient or physician considered that a major adverse cardiovascular event or some other serious adverse event had occurred, the attending cardiologist was contacted and photocopies of original reports, letters, laboratory tests, and electrocardiograms were obtained to document these possible events. The possible events were then judged by an independent events committee.

### Statistical analysis

All data are expressed as mean  $\pm$  standard deviation. We performed a univariate repeated-measures analysis of variance using SPSS 11.3, with time as a fixed variable and patient ID as a dummy variable. Repeated-measures analysis of variance was followed by analysis of contrasts with a contrast set as a difference from a baseline value (a probability value of  $P < 0.05$  was considered significant).



**Figure 3** Implantation of the ventricular partitioning device (VPD) showing (A) positioning of the collapsed VPD at the left ventricular apex, (B) VPD expansion, and (C) VPD release. See text for details.



**Figure 4** Computerized tomography at 9-month follow-up, showing the ventricular partitioning device (VPD) at the left ventricular apex (black arrows) and masses on both sides of the device (white arrows). (Reprinted from ref. 14 with permission from Elsevier).

**Table 1** Baseline demographic data

Sex (male)	17/18
Age (years)	58 ± 9
NYHA class II	13/18
NYHA class III	5/18
Diabetes	3/18
Hypertension	9/18
Prior coronary revascularization	6/18

NYHA, New York Heart Association class.

## Results

The demographic characteristics of the patients included in the study are shown in Table 1. Ventricular partitioning device was successfully implanted in 15/18 (83%) patients. In two patients, the VPD was not implanted due to unfavourable anatomy of the anteroapical LV aneurysm, and in one due to tortuosity of the iliac arteries which rendered the delivery catheter too short to deliver the VPD at the apex. No catheter or device malfunctions were noted. In one patient, the VPD was inadequately attached and moved from the apex into the LV cavity. This patient underwent immediate open-heart surgery, the VPD was removed, and the patient received a bypass graft to the left anterior descending artery and LV aneurysmectomy. This patient's postoperative course was uneventful and she was discharged 10 days post-operatively in a stable condition. One patient developed signs and symptoms of infection during the post-procedure period. The VPD was surgically explanted after 3 days on the assumption that the prosthetic material might be the source of the infection. The VPD implant was cultured and was negative for infection.

The patient subsequently died 19 days following implantation due to sepsis. Autopsy revealed that the likely source of infection was an unrecognized perianal abscess. There were no other acute serious adverse events, except for an inguinal haematoma in one patient that did not require surgical treatment.

The mean diameter of the VPD implant attachment zone was  $56.4 \pm 6.1$  mm. A 75 mm size was implanted in eight patients, and an 85 mm size was implanted in the remaining seven. There were no peri-attachment leaks in seven patients, trace leaks were found in seven patients, and a moderate leak was seen in one patient. The procedure time averaged  $60.6 \pm 29.0$  min, with a mean fluoroscopy time of  $17.9 \pm 10.5$  min. An average of  $340 \pm 99$  mL of contrast agent was used per patient.

When compared with baseline, the mean LVEDP was slightly lower immediately post-procedure, but there was a significant decrease in LVEDP 6 months following VPD implantation ( $18.1 \pm 9.4$  vs.  $16.4 \pm 6.1$  vs.  $12.9 \pm 4.4$  mmHg, respectively,  $P = 0.007$ ).

Serum creatinine and blood urea nitrogen did not show significant changes at 24 h following procedure or at discharge, when compared with baseline values (Table 2). At the same time points, CK, CK-MB, and troponin T were consistently within reference range, indicating that no myocardial damage had occurred during VPD implantation.

On discharge, there was no leakage between the static and dynamic LV chamber in five patients, whereas in six patients the leakage was mild, and in two patients moderate. Only chronically

implanted patients were analysed. On control echocardiographic examinations, the extent of leakage remained exactly the same in all patients.

Data on symptom status, functional capacity, and haemodynamic characteristics during the 12-month follow-up are shown in Table 3. Briefly, there was significant improvement in NYHA class and LV volumes 6 months following VPD implantation when compared with baseline, with the effect sustained over 12 months. As for the LVEF and 6 min walk test, a statistically significant improvement was observed at 12 months, with a positive trend at 6 months. However, there was no significant difference in the quality of life score when compared with baseline.

No complex arrhythmias were noted during the implantation and follow-up. However, as the protocol did not mandate use of Holter monitoring or any other arrhythmia monitoring device, the true incidence and severity of arrhythmias remains unknown.

Regarding late serious adverse events, one patient (with post-procedural inguinal haematoma) underwent surgery at 3 months following VPD implantation due to femoral artery pseudo-aneurysm, and one patient suffered a transitory ischaemic attack at 9 months (the source of embolism was not identified). Both patients fully recovered. There were no deaths, or other major adverse cardiovascular events, during the follow-up. No patient was rehospitalized for symptoms of heart failure.

## Discussion

The low incidence of periprocedural and long-term complications reflects the safety of the procedure, adequate design of the device, and well tailored concomitant medical therapy. The only death was not device-related, but probably reflects poor patient selection. In one patient, the VPD was not adequately attached and dislocated during the procedure, but was safely removed. In this particular patient, the anterior wall and apex were very thin with severe fibrosis and calcification and it can be assumed that the struts failed to anchor adequately.

Pre-clinical studies in an animal model of MI have indicated that the VPD implant has beneficial short- and medium-term effects on LV function, as measured by a decrease in LV volumes and end-diastolic pressure, increased cardiac output, and improvement in EF. Pathohistologic analysis, performed 6 months after implantation, showed that the luminal surface of the VPD was covered by organized thrombus and was covered by a smooth lining. Inflammatory response was focal and mild consisting of histiolympocytic infiltrate surrounding the anchors and some portions of the graft with an occasional giant cell.<sup>4,5</sup>

This first in man clinical study demonstrates that the VPD implant has favourable effects on LV function, as measured by LVESV, LVEDV, EF, and LVEDP, as well as symptom status, and exercise tolerance. These are very important findings, as it has been shown that LVESV is one of the major prognostic predictors following MI.<sup>6</sup> Although the mechanisms of these beneficial effects are not completely established, we hypothesize the following. The VPD implant partitions the enlarged ventricle into dynamic and static chambers. The portion of the LV volume that comprises the static chamber is sealed and taken out of the circulation. In the static chamber, the segregated blood clots and forms

**Table 2 Serum creatinine and blood urea nitrogen concentrations**

	Pre	24 h post	Discharge
Creatinine (mmol/L)	$93.8 \pm 7.9$	$122.6 \pm 41.2$	$109.8 \pm 26.9$
BUN (mmol/L)	$6.2 \pm 1.6$	$7.5 \pm 2.8$	$8.6 \pm 3.8$

BUN, blood urea nitrogen; Pre, pre-implantation; Post, post-implantation.

**Table 3 Symptom status, functional capacity, and haemodynamic characteristics during follow-up**

	Baseline	6 months	12 months
NYHA class	$2.21 \pm 0.57$	$1.28 \pm 0.46^{\ddagger}$	$1.23 \pm 0.43^{\ddagger}$
LVEF (%)	$28 \pm 7$	$32 \pm 7$	$33 \pm 9^*$
LVESV (mL)	$189 \pm 45$	$142 \pm 29^{\ddagger}$	$151 \pm 48^{\ddagger}$
LVEDV(mL)	$260 \pm 47$	$208 \pm 33^{\ddagger}$	$222 \pm 58^{\ddagger}$
6 min walk test (m)	$382 \pm 123$	$409 \pm 129$	$425 \pm 140^*$
QoL (points)	$21.7 \pm 18.9$	$16.7 \pm 12.3$	$20.8 \pm 16.9$

LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume index; LVESV, left ventricular end-systolic volume index; NYHA, New York Heart Association; QoL, quality of life as assessed by Minnesota Living with Heart Failure Questionnaire.

\* $P < 0.05$  vs. baseline.

<sup>†</sup> $P < 0.01$  vs. baseline.

<sup>‡</sup> $P < 0.001$  vs. baseline.

organized thrombus behind the partitioning membrane, but this thrombus does not usually fill the whole static chamber. Stress in the partitioned myocardium and the forces transmitted to the apical segment appear to be decreased both in diastole and systole, thus possibly diminishing the stress that is responsible for dilation. In addition to this regional unloading, the reduction in size of the dynamic chamber results in a decrease in myocardial wall stress in the normal myocardium via Laplace's Law, providing a global unloading of the ventricle. Regional unloading decreases the myocardial oxygen demand of that regional tissue thereby increasing the relative myocardial oxygen supply to the non-isolated portion of the ventricular wall. An overall reduction in global myocardial stress may further decrease myocardial oxygen demand and improve the oxygen supply/demand ratio to the functioning myocardium. These beneficial haemodynamic effects are accompanied by improvement in the patient's symptomatic status as measured by improvement in NYHA class and exercise capacity (6 min walk distance) at 12 months post implant.

The clinical importance of leakage between the static and dynamic LV chamber is not clear. It can be assumed that higher degrees of leakage may have an unfavourable effect on LV remodelling, and that there may be increased risk of peripheral embolism through larger communications between the static and dynamic LV chambers. However, our group of patients was too small to allow further sub-analysis, and no severe leakage was noted in our patient population.

It is not clear why there was no observed improvement in the Minnesota Living with Heart Failure Questionnaire. It is possible that the use of this questionnaire in this group of Serbian and German patients may not have been appropriate due to diversities in their social, economic, and cultural backgrounds compared with North American patients. Another potential explanation is that the patient's quality of life was impaired due to requisite medication intake and dietary restrictions due to prolonged anti-coagulation therapy.

Several surgical approaches have been advocated for LV volume reduction, including surgical ventricular remodelling (Dor procedure) and partial left ventriculectomy (Batista procedure). The largest prospective trial that evaluated the effects of surgical ventricular restoration was the RESTORE trial, which included nearly 1200 patients. Thirty-day mortality after surgical ventricular remodelling was 5.3%. Overall 5-year survival was 70%, and it was better in the subgroup of patients that had dyskinetic when compared with akinetic apical wall motion. Overall freedom from re-admission to the hospital for congestive heart failure was 78%, and NYHA functional class improved from a mean of 2.9 preoperatively to 1.7, postoperatively.<sup>7</sup> The randomized STICH trial included 1000 patients with LVEF of 35% or less, coronary artery disease that was amenable to coronary artery bypass graft (CABG), and dominant anterior LV dysfunction that was amenable to surgical ventricular reconstruction. Patients were randomly assigned to undergo either CABG alone or CABG with surgical ventricular reconstruction. Although addition of surgical ventricular reconstruction to CABG reduced LV volumes, it did not appear to be associated with a greater improvement in symptoms or exercise tolerance or with a reduction in the rate of death or hospitalization for cardiac causes.<sup>8</sup> Partial left ventriculectomy has been virtually abandoned, largely because of the

unpredictability of the results compared with the morbidity and mortality associated with the procedure. It has been reported that hospital survival after this procedure is 83%,<sup>9</sup> and 10-year survival is only 16% (Sinisa Gradinac, personal communication). In surviving patients, improvement in symptomatic status, functional capacity, and LV haemodynamics were noted.

In summary, data from surgical trials indicate that the described procedures reduce LV volumes, with mixed effects on functional capacity and quality of life, and no effects on mortality, which is very similar to our findings.

Another more generalized approach to mechanical intervention includes the use of cardiac support devices, which either acutely alter ventricular geometry [e.g. the Myosplint device (Myocor, Maple Grove, USA)] or attempt to reverse LV remodelling in a slower fashion through a reduction in wall stress [e.g. the CorCap Cardiac Support Device (Acorn Cardiovascular, St Paul, USA)].<sup>10</sup> Fukamachi and McCarthy<sup>11</sup> reported on 21 patients with dilated cardiomyopathy in whom the Myosplint device was deployed. Of these patients, 9 received the Myosplint device as the only therapy and 12 received the Myosplint in combination with mitral valve repair. At 6-month post-implantation, there were no device-related complications such as thromboembolism, bleeding device instability, or vascular damage, suggesting that placement of the Myosplint device is safe. NYHA class, LV volumes, and LVEF were improved at 6 months when compared with baseline. ACORN's clinical trial of the CorCap Cardiac Support Device in patients with heart failure has been completed, and included 300 patients who received either the CorCap Cardiac Support Device alone, the CorCap Cardiac Support Device in combination with mitral valve surgery, or mitral valve surgery alone. It was concluded that implantation of the CorCap Cardiac Support Device is safe, as the incidence of periprocedural and long-term follow-up cardiac adverse events was low.<sup>12</sup> Implantation was associated with a significant reduction in LV volumes throughout the study period, but had inconsistent effects on LVEF.<sup>13</sup>

Implantation of a cardiac support device or surgical ventricular remodelling requires open-heart surgery. This may be a major advantage of the VPD implant over other mechanical interventions as it can be implanted percutaneously, thus avoiding the early mortality and morbidity associated with an open surgical intervention. Therefore, the VPD implant can be more readily used for patients in whom surgical revascularization or other concomitant procedures are not needed.

The VPD implantation procedure has an acceptable duration and fluoroscopy time. Despite the relatively large quantities of contrast agent used, there were no significant increases in serum creatinine or blood urea nitrogen concentrations. The fact that no myocardial damage occurred during VPD implantation may suggest that VPD was implanted in the region with no viable myocardium and/or that anchor penetration in the myocardium is minimal, but sufficient to provide device stability.

At this stage, it appears that a VPD implant should not be attempted in patients with complex geometry of the anteroapical wall, as exact positioning of VPD may be very complicated, if not impossible. Two other major challenges that should be avoided in patient selection include: (i) aberrant chordae tendinae in the vicinity of LV apex, which may interfere with VPD landing and deployment,

and (ii) possible interference of the VPD implant with papillary muscles in patients with dilated, but 'shallow' left ventricles.

Although initial experience and 12-month effects of VPD implantation are encouraging, further studies and longer follow-up are needed before the VPD can be placed in adequate clinical perspective. The major limitation of the current study is the small number of patients that were included. Additionally, the assessment of NYHA class may be confounded by the fact that the trial was unblinded.

In conclusion, our data indicate that VPD implantation is safe, feasible and that VPD implantation improves LV haemodynamics, functional class, and exercise capacity in the 12 months following the procedure.

## Acknowledgements

The authors would like to thank Zoran B. Popovic for his expert assistance in statistical analysis.

## Funding

The study was funded by Cardiokinetix Inc., Menlo Park, CA, USA, manufacturers of the Ventricular Partitioning Device.

**Conflict of interest:** none declared.

## References

1. Tonnessen T, Wold Knudsen C. Surgical left ventricular remodeling in heart failure. *Eur J Heart Fail* 2005;**7**:704–709.
2. Starling RC, Jessup M. Worldwide clinical experience with the Corcap cardiac support device. *J Card Fail* 2004;**10**(Suppl. 6):S225–S233.
3. Udelson JE, Konstam MA. Relation between left ventricular remodeling and clinical outcomes in heart failure patients with left ventricular systolic dysfunction. *J Card Fail* 2002;**8**:S465–S471.
4. Nikolic SD. Percutaneous intraventricular device for the treatment of heart failure: concept and experimental results. *Transcatheter Cardiovascular Therapeutics 2003 Annual Meeting (abstract)*. www.tctmd.com. Accessed 7 January 2009.
5. Nikolic SD, Khairkhahan A, Ryu M, Champsaur G, Breznock E, Dae M. Percutaneous implantation of an intraventricular device for the treatment of heart failure: experimental results and proof of concept. *J Card Fail* 2009;**15**:790–797.
6. White HD, Norris RM, Brown MA, Brandt PV, Whitlock RM, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. *Circulation* 1987;**76**:44–51.
7. Athanasuleas CL, Buckberg GD, Stanley AWH, Siler W, Dor V, DiDonato M, Menicanti L, de Oliveira SA, Beyersdorf F, Kron IL, Suma H, Kouchoukos NT, Moore W, McCarthy PM, Oz MC, Fontan F, Scott ML, Accola KA. Surgical ventricular restoration: The RESTORE group experience. *Heart Fail Rev* 2004;**9**:287–297.
8. Jones RH, Velazquez EJ, Michler RE, Sopko G, Oh JK, O'Connor CM, Hill JA, Menicanti L, Sadowski Z, Desvigne-Nickens P, Rouleau JL, Lee KL. Coronary bypass surgery with or without surgical ventricular reconstruction. *N Engl J Med* 2009;**360**:1705–1717.
9. Kawaguchi AT, Takeshita N, Bocchino L, Shimura S, Batista RJV. Angiographic and hemodynamic follow-up of patients after partial left ventriculectomy. *J Card Surg* 2005;**20**:S35–S38.
10. Blom AS, Mukherjee R, Pilla JJ, Lowry AS, Yarbrough WM, Mingoia JT, Hendrick JW, Stroud RE, McLean JE, Affuso J, Gorman RC, Gorman JH 3rd, Acker MA, Spinale FG. Cardiac support device modifies left ventricular geometry and myocardial structure after myocardial infarction. *Circulation* 2005;**112**:1274–1283.
11. Fukamachi K, McCarthy PM. Initial safety and feasibility clinical trial of the myosplint device. *J Card Surg* 2005;**20**:S43–S47.
12. Mann DL, Acker MA, Jessup M, Sabbah HN, Starling RC, Kubo SH. Clinical evaluation of the CorCap Cardiac Support Device in patients with dilated cardiomyopathy. *Ann Thorac Surg* 2007;**84**:1226–1235.
13. Starling RC, Jessup M, Oh JK, Sabbah HN, Acker MA, Mann DL, Kubo SH. Sustained benefits of the CorCap Cardiac Support Device on left ventricular remodeling: three year follow-up results from the Acorn clinical trial. *Ann Thorac Surg* 2007;**84**:1236–1242.
14. Otasevic P, Sagic D, Antonic Z, Nikolic SD, Khairakhan A, Radovancevic B, Gradinac S. First-in-man implantation of left ventricular partitioning device in a patient with chronic heart failure: 12-month follow-up. *J Card Fail* 2007;**13**:517–520.