Enhanced External Counterpulsation as Treatment for Chronic Angina in Patients With Left Ventricular Dysfunction: A Report From the International EECP Patient Registry (IEPR)

The International Enhanced External Counterpulsation (EECP) Patient Registry tracks acute and long-term outcome for consecutive patients treated for chronic angina. Although EECP has previously been shown to be a safe and effective treatment for angina, little information is available on its use in patients with left ventricular (LV) dysfunction. This report compares the acute outcome and 6-month follow-up for a group of patients with severe LV dysfunction and a group of patients without LV dysfunction. Of 1402 patients in the registry recruited in 1998–1999 who had recorded values of LV ejection fraction (LVEF) at baseline, 1090 (77.7%) had preserved LV function (LVEF >35%) and 312 (22.3%) had LV dysfunction (LVEF ≤35%). Six-month follow-up was available on 84% of these patients. Pre-EECP patients with LV dysfunction had a longer history of coronary artery disease (12.9 years vs. 9.1 years; p<0.001), a higher rate of congestive heart failure (60.6% vs. 20.1%; p<0.001) and myocardial infarction (83.5% vs. 61.9%; p<0.001). Patients with LV dysfunction had more severe pre-EECP angina, with 86.2% presenting with Canadian Cardiovascular Society Class III/IV vs. 73.6%; p<0.01. Patients with LV dysfunction, consistent with their more severe baseline profile, suffered more adverse events (death, unstable angina, and exacerbation of heart failure) during the treatment period and were less likely to complete the full course. Immediately post-EECP, angina decreased by at least one class in 67.8% of patients with LV dysfunction (vs. 76.2%; p<0.01), and 35.9% of LV dysfunction patients vs. 39.0% had discontinued nitroglycerin use (p=NS). At 6-month follow-up, patients with LV dysfunction showed higher rates of death (9.3% vs. 2.2%; p<0.001) and exacerbation of congestive heart failure (9.9% vs. 3.7%; p<0.001). Rates of the composite outcome of death/myocardial infarction/coronary artery bypass grafting/percutaneous coronary intervention (15.4% vs. 8.3%; p<0.001) were also higher for patients with LV dysfunction. However, patients not reporting such an event showed maintenance of their improved anginal status, with 81% of LV dysfunction vs. 83.8% of patients without LV dysfunction (p=NS) reporting angina at 6 months equal to or less severe than immediately post-EECP, and nitroglycerin use was still reduced at 46.1% for LV dysfunction vs. 37.4% (p<0.05). The rate of event-free angina maintenance at 6 months was 67.0% for patients with LV dysfunction and 70.6% of patients with preserved LV function (p=NS). Patients with LV dysfunction achieved a less robust reduction in angina than did those without LV dysfunction. For the majority of the patients in the registry, this reduction was maintained at 6 months. (CHF. 2002;8:297–302, 312) ©2002 CHF, Inc.

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For nearly half a century, investigators have tried to develop techniques that could lower cardiac afterload while at the same time increasing diastolic coronary flow in patients with acute and/or chronic coronary syndromes. Enhanced external counterpulsation (EECP) is a novel technology for accomplishing these goals. It is a noninvasive counterpulsation technique that has been shown to reduce angina and extend time to exercise-induced ischemia in patients with symptomatic coronary disease.[1] In addition to relieving myocardial ischemia, EECP is associated with improved quality of life.[2] EECP uses sequential inflation of three

sets of pneumatic cuffs wrapped around the lower legs, thighs, and upper thighs. The cuffs are inflated sequentially at the onset of diastole, producing aortic counterpulsation and diastolic augmentation (DA) and increased venous return. At the onset of systole, the external pressure in the cuffs is released, producing a decrease in systolic pressure [systolic unloading (SU)]. Subsequent modifications of the EECP prototype with microprocessors allowed for precise cuff inflation and deflation and gating with electrocardiography. The hemodynamic effects may be monitored noninvasively by assessing the finger plethysmographic waveforms. Although EECP has been shown to be beneficial in patients with angina and normal cardiac function, its safety and efficacy in patients with angina and left ventricular dysfunction (LVD) has not been evaluated. Indeed, previous studies either have not shown a relationship between ejection fraction (EF) and response to EECP,[3] or have excluded patients with a low EF altogether.[1] This report examines the safety and efficacy of EECP for relief of chronic angina in patients with impaired left ventricular (LV) function (LVEF ≤35%) who were enrolled in the International EECP Patient Registry. Data were obtained after completion of EECP and after the 6-month follow-up period and comparisons were made between patients with and without LVD.

Methods

The International EECP Patient Registry (IEPR) enrolls consecutive patients undergoing EECP for chronic angina. The IEPR began in January, 1998, and to date over 3000 patients have been enrolled from over 100 centers in the United States and other countries. Since the Registry aims to collect data on as broad a range of patients as possible, the criteria for entry are only that the patient give informed consent for participation in the registry and have at least 1 hour of EECP treatment for chronic angina.

For this analysis, only those patients (n=1402) with a recording of LVEF prior to the commencement of EECP therapy were included. In the statistical analyses, data are presented as percentages for categorical variables or as mean values and standard deviations for continuous variables. Comparisons between those with LVEF ≤35%, and those with LVEF <35% were done using chi-square tests or *t* tests, as appropriate.

Results

These 1402 patients were enrolled in the registry from 75 sites from 1/98 thru 12/99. Impaired LV function was defined as an EF ≤35% and occurred in 312 (22.3%) of patients. For these patients, the mean EF was 28.6%±6.2%. LV function was preserved (EF >35%, mean value 52.1%±9.2%) in 1090 patients (77.7%).

Baseline characteristic of the patients prior to receiving EECP treatment by EF category are shown in Table I. The patients with LVD were more likely to have been diagnosed with coronary artery disease for a longer period of time (12.9 years vs. 9.1 years; $p<0.001$), although the mean age for the two groups was not significantly different. Patients in this group also had more prior myocardial infarctions (MI), (83.5% vs. 61.9%; $p<0.001$), more previous bypass surgery (69.7% vs. 62.9%; $p<0.05$), and a higher incidence of congestive heart failure (60.6% vs. 20.1%; $p<0.001$). Risk factors were very similar in the two groups. However, symptoms of coronary disease were more severe in the LVD group, with 86.2% presenting with Canadian Cardiovascular Society class III or IV angina, vs. 73.6% for the group with higher EFs, and more frequent multivessel disease (89.8% vs. 76.5%; $p<0.001$). Only 9% (vs. 20%; $p<0.001$) were considered suitable for percutaneous coronary intervention (PCI), and 13% (vs. 25%; $p<0.001$) were considered suitable for coronary artery bypass grafting (CABG) at the time of commencing EECP.

The outcome immediately following EECP treatment is summarized in Tables II and III. Patients with LVD were less likely to complete a course of treatment (79.4% vs. 85.8%; $p<0.01$; mean hours, 32.8 vs. 34.4; $p<0.05$), and the reason for stopping treatment was significantly more likely to be because of a clinical event (14.4% vs. 7.2%; $p<0.001$). However, serious cardiac events during the treatment period were rare in both groups of patients. The combined rate of death/MI/CABG/PCI was 2.9% for patients with LV dysfunction, and 1.7% for those without, a difference that was not significant. However, some adverse events were more frequent in the LVD group—in particular, exacerbation of congestive heart failure (5.4% vs. 1.0%; $p<0.001$) and unstable angina (4.2% vs. 2.0%; $p<0.05$). All adverse events are summarized in Table II.

Patients without LV dysfunction achieved higher levels of diastolic augmentation than those with LV dysfunction (Table III). For patients without dysfunction, the mean value of the peak diastolic to systolic pressure at the completion of EECP was 1.2±0.7, compared to 1.00±0.6 for LVD. The corresponding figures for the ratios of area were 1.4±0.8 vs. 1.1±0.60. Both these differences are statistically significant ($p<0.001$). Despite these differences, both groups achieved a considerable reduction in angina (defined as a decrease in angina of at least one Canadian Cardiovascular Society classification angina class), although the reduction was significantly less for those with LV dysfunction (67.8% vs.76.2%; $p<0.01$),

with a concomitant decrease in angina counts and use of nitroglycerin. For those patients who completed the full course of treatment (at least 35 hours) there were similar rates of angina decrease between the two groups (80.1% for those with LVD vs. 83.8% for those without; p=NS).

Six-month follow-up was obtained for 80% of patients with LVD and 86% of those without LVD. The cumulative events and status at 6 months are summarized in Table IV. Patients with LVD, in keeping with their poor risk profile at baseline, had a substantially higher mortality (9.3%) than those without (2.2%). This difference was statistically significant, with a p value of <0.001. The combination outcome of death/MI/PCI/CABG was also substantially higher for the LVD patients (15.4% vs. 8.3%; $p<0.001$), as was the incidence of episodes of congestive heart failure (9.9 % vs. 3.3%; $p<0.001$). Hospitalizations, both for cardiac and noncardiac reasons, occurred with similar frequency in the two groups. For the majority of patients not undergoing an event (death/MI/PCI/CABG) during this period, angina levels immediately post-EECP were maintained. Angina class was reported as less than or equal to that immediately post-EECP in 81.0% of LVD patients vs. 83.8% of the group with preserved LV function (p=NS). The fact that the angina maintenance was lower in the LVD group is consistent with the increased event rate in this group. In fact, the combined end point of maintenance of angina reduction with no event was 67% in the LVD group vs. 70.6% in the group with preserved LV function (p=NS).

Discussion

The present study is the largest reported series of consecutive patients treated with EECP for chronic angina

Table I. Pre-EECP Patient Characteristics by Ejection Fraction (EF)

	EF >35%	EF ≤35%
Number of patients	1090	312
Demographics		
Age (years)	66.0±10.4	66.9±10.7
Male gender	75.5	80.4
Medical history		
Time since CAD diagnosis (years)†	9.1±7.5	12.9±8.7
Prior percutaneous coronary intervention (PCI)	60.2	58.6
Prior coronary artery bypass graft (CABG)*	62.9	69.7
Prior myocardial infarction†	61.9	83.5
History of congestive heart failure†	20.1	60.6
Risk factors		
Family history of CAD	75.6	72.5
History of diabetes	41.7	41.3
History of hypertension	68.7	63.7
History of hyperlipidemia	76.9	74.8
Noncardiac vascular disease	29.2	34.1
History of smoking	68.9	72.9
Angina characteristics**		
CCS class I	3.9	3.5
II	22.4	10.3
III	53.9	53.2
IV	19.7	33.0
Unstable angina	1.7	4.8
Angina episodes/week	8.7±13.1	9.9±12.7
Nitroglycerin use*	64.9	71.7
No. of times/week*	8.3±11.1	10.4±11.5
Multivessel disease†	76.5	89.8
Candidate for PCI†	20.5	9.1
Candidate for CABG†	25.2	12.7

EECP=enhanced external counterpulsation; CCS=Canadian Cardiovascular Society; CAD=coronary artery disease; *$p<0.05$; **$p<0.01$; †$p<0.001$

pectoris. These patients show a profile of long-standing coronary disease, with chronic angina unrelieved by medical means or conventional revascularization. Concomitant diseases, such as congestive heart failure and diabetes, were frequent. LVD was present in about one fifth of the patients. At the start of the treatment, patients with LVD had a longer history of coronary artery disease, more congestive heart failure and MIs, and more severe angina than the patients with preserved LV function. Most patients, regardless of whether or not they had a low EF, experienced relief of angina after a course of EECP treatment. Decreases in the number of angina episodes and use of nitroglycerin also were similar for those with and without LVD. As has been shown previously,[5] patients with LVD do not augment as well as those without LVD. However, the exact relationship between angina reduction and augmentation has not yet been elucidated.

Table II. Major Adverse Events During EECP Treatment Period by Ejection Fraction (EF)

	EF >35% (N=1090)	EF ≤35% (N=312)
Death**	3	1.6
Coronary artery bypass grafting (CABG)	0.4	0.0
Percutaneous coronary intervention (PCI)	0.6	0.3
Myocardial infarction (MI)	0.7	1.3
Unstable angina*	2.0	4.2
Exacerbation of congestive heart failure†	1.0	5.4
Skin breakdown	0.9	0.6
Musculoskeletal problems	0.6	1.6
Death/MI/CABG/PCI	1.7	2.9

Data are percentages.
EECP=enhanced external counterpulsation; *$p<0.05$; **$p<0.01$; †$p<0.001$

Table III. Post-EECP Results by Ejection Fraction (EF)

	EF >35%	EF ≤35%
No. of patients starting treatment	1090	312
No. of patients completed treatment	85.8	79.4
No. of patients stopped because of clinical event %†	7.2	14.4
No. of patients discontinued	7.0	6.2
Hours of treatment (mean)*	34.4±9.9	32.8±11.7
Diastolic augmentation at last hour:		
Peak ratio†	1.20±0.7	1.0±0.5
Area ratio†	1.36±0.8	1.12 0.6
Anginal status**		
No angina	20.2	15.6
CCS Class I	29.4	20.2
II	31.4	32.4
III	14.3	20.8
IV	4.9	11.2
Angina decreased ≥1 class*	76.2	67.8
Mean decrease in angina episodes/week	6.7±12.4	6.7±12.1
Nitroglycerin discontinued %**	39.0	39.5
Mean decrease in nitroglycerin use/week	6.2±10.0	7.2±11.3

Data are percentages of patients with information recorded.
EECP=enhanced external counterpulsation; CCS=Canadian Cardiovascular Society; *$p<0.05$; **$p<0.01$; †$p<0.001$

Importantly, there were few adverse effects of EECP. However, patients with LVD were less likely to complete the full course of treatment because of intervening clinical events. As might be expected in such a high-risk population, the rates of death, unstable angina, and exacerbations of congestive heart failure were significantly more frequent in the patients with LVD. These higher adverse event rates in the patients with LVD continued to 6 months. However, patients without these events maintained their reduced angina status. These initial results from the IEPR suggest that EECP treatment is a safe and effective method for the treatment of patients with chronic angina and LVD.

In the one fifth of Registry patients who had LVD, the majority tolerated EECP and showed considerable reduction in angina at the end of the treatment. We cannot exclude the possibility that patient selection affected our results. In particular, physicians may have been reluctant to refer patients with symptomatic LVD for EECP. Furthermore, many heart failure patients with significant coronary artery disease do not experience angina because their exertion is limited by shortness of breath. Regardless, this is the largest study to report acute and long-term outcomes for consecutive patients treated with angina who had LVD, as evidenced by an EF <35%. The benefits of intra-aortic balloon counterpulsation in patients with congestive heart failure have been well described, and the hemodynamic effects that it imparts in acute coronary syndromes are well recognized.[6] Furthermore, the benefits of EECP in patients with angina and compromised LV function are not inconsistent with the known hemodynamic effects of EECP, since EECP not only improves diastolic coronary flow, but also effectively reduces afterload.[7]

Another potential benefit of EECP is a salutary modulation of neurohormonal expression. It is well known that neurohormonal activation is deleterious in patients with heart failure.[8,9] For example, elevations in endothelin and decreases in plasma levels of the endogenous vasodilator nitric oxide have been identified in patients with heart failure, and the degree of change is directly related to increased mortality and/or alterations in functional capacity. Recent studies suggest that EECP effectively decreases the expression of the potent vasoconstrictor endothelin while increasing expression of the vasodilator nitric oxide.[9] Thus, while recent data suggest that EECP exerts beneficial effects in patients with angina and normal LV function by enhancing the development of collateral vessels, its effects in patients with coronary disease and compromised LV function may be far more complex.[7,9] Our results are consistent with earlier anecdotal studies suggesting that EECP is a safe and effective treatment for angina in patients with severe LVD, and the magnitude of improvement in angina classification and quality of life produced by EECP is independent of the degree of LVD before treatment.[10,11]

Table IV. Cumulative Events to 6 Months by Ejection Fraction (EF)

	EF >35%	EF ≤35%
	(n=1090)	(n=312)
Completed 6 months follow-up*	85.7	80.1
Death†	2.2	9.3
Coronary artery bypass grafting (CABG)	1.5	–
Percutaneous coronary intervention (PCI)	2.8	3.2
Myocardial infarction (MI)	3.1	3.5
Death/MI/PCI/CABG†	8.3	15.4
Repeat EECP	11.5	11.5
Cardiac hospitalization	12.8	12.3
Noncardiac hospitalization	6.4	7.4
Exacerbation of congestive heart failure†	3.7	9.9
Unstable angina	7.5	8.0
Patients without death/MI/PCI/CABG		
Angina same/less than post	83.8	81.0
Angina class I/II or none†	83.3	72.7
Nitroglycerin use*	37.4	46.1
No death/MI/PCI/CABG and angina same/less than post	70.6	67.0

Event data are percentages of all patients starting EECP. Events are not exclusive. Angina data are percentages of those patients reporting anginal status.
EECP=enhanced external counterpulsation; *$p<0.05$; †$p<0.001$

A limitation of the Registry is the lack of a control group. We cannot exclude the possibility that angina reduction was partly or largely due to a placebo effect. A clinical trial with patients with LVD assigned randomly to EECP vs. control would be needed to definitively determine the value of EECP for these patients. The Multicenter Study of Enhanced External Counterpulsation (MUST-EECP) clinical trial[1] demonstrated the efficacy of EECP for a general population of chronic angina patients. Since the observational data we report suggests benefit to those with LVD comparable to that for patients with normal LV function, this treatment should be considered for such patients.

Conclusions

Data from this prospective Registry indicate that EECP is safe in the treatment of chronic angina in patients with LVD. Most patients were able to undergo a course of treatment (usually 35 hours) without adverse effects. The incidence of major cardiac events was low (2.8%) and was not significantly different from the rate (1.8%) observed in patients with preserved LV function. Angina class and nitroglycerin use were reduced in the majority of patients. Although patients with LVD did not show as much reduction as other patients, the overall rates of reduction were high.

At 6 months of follow-up, patients with LVD had a significantly higher mortality rate, a significantly higher rate of the combined outcome of death/MI/PCI/CABG, and more episodes of exacerbation of congestive heart failure. However, these patients had a severe disease profile at baseline compared to those without LV dysfunction. For patients not suffering an adverse event, the reduction of angina achieved post-EECP was maintained in the majority, and event-free maintenance of angina reduction was not significantly different between the two groups.

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Appendix

IEPR Clinical Sites, Investigators, and Coordinators

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