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Congestive Heart Failure

FOR CLINICIANS TREATING CONGESTIVE HEART FAILURE AND ITS CO-MORBID CONDITIONS INCLUDING HYPERTENSION, CORONARY ARTERY DISEASE, ATHEROSCLEROSIS, AND DIABETES

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John E. Strobeck, MD, PhD, Co-Editor in Chief

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Ozlem Soran, MD
Bruce Fleishman, MD
Theresa Demarco, MD
William Grossman, MD
Virginia M. Schneider, RN
Karen Manzo, RN
Paul-André de Lame, MD
Arthur M. Feldman, MD, PhD

Editorial

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John E. Strobeck, MD, PhD, Co-Editor in Chief
From the Heart-Lung Center, Hawthorne, NJ

Address for correspondence:
John E. Strobeck, MD, PhD, Heart-Lung Center, 297 Lafayette Avenue, Hawthorne, NJ 07506

Enhanced External Counterpulsation (EECP) is a unique outpatient, noninvasive treatment method currently used to improve myocardial perfusion, reduce symptoms from obstructive coronary disease, and enhance patient functionality and quality of life. Its roots are in the science of intra-aortic balloon counterpulsation, but its effect on cardiac output and systemic vascular resistance, due to simultaneous venous and arterial sequential compression and simultaneous release, can be shown to be superior to the hemodynamic response typically seen with intra-aortic balloon counterpulsation. The device has undergone a significant evolution in design since the first units in 1953 used by Kantrowitz[1] and Amsterdam[2] and now, as used by the team from the University of Pittsburgh Medical Center in their paper in this issue of *Congestive Heart Failure*,[3] it is a streamlined system of external pneumatic cuffs, placed on the lower extremities and hips, sequentially inflated during early diastole, with careful online monitoring of the electrocardiogram, arterial pulse waveform/amplitude, and arterial oxygen saturation.

Lawson and colleagues[4] began reporting their experience in 1992 using EECP in chronic angina refractory to medical therapy and showed an overall improvement in stress myocardial perfusion in 78% of their patients exercised to the same cardiac workload. Post-treatment maximal stress testing showed an increase in peak exercise time and an increase in peak double-product in all patients with improved myocardial perfusion. These safety and efficacy results were extended in uncontrolled studies and confirmed by comparison with placebo in the Multicenter Study of Enhanced External Counterpulsation (MUST-EECP) completed and published in 1999.[5] Patients completing this trial were followed for 12 months after EECP treatment and demonstrated persistent improvement in health-related quality-of-life scores over placebo treatment.[6] An International EECP Patient Registry was begun in 1998 at the University of Pittsburgh Medical Center. Participation by EECP treatment centers was voluntary and during Phase I more than 5000 patients with angina pectoris were enrolled. Phase II has commenced and plans to enroll another 2500 patients with division into several important substudies such as type 2 diabetes, peripheral arterial disease, and sexual dysfunction in men.

Experienced EECP centers treating large numbers of patients with chronic obstructive coronary disease, including my own, have necessarily treated patients with low ejection fraction (EF) (<35%). Our experience, as documented by a recently published analysis of the International EECP Patient Registry data,[7] indicates that patients with low EF and obstructive coronary disease respond as well as patients with EF% >35% and do not suffer an increase of adverse events. The important study of Soran et al.[3] in this issue of *Congestive Heart Failure* extends these observations and forms the basis for a larger multicenter, randomized, single blind, controlled Prospective Evaluation of EECP in CHF (PEECH trial) currently enrolling patients in the United States.

Over the last two decades the preferred locus of care for patients with CHF has shifted from acute care inpatient settings to less-acute care inpatient and outpatient heart failure clinic settings. This shift has been in response to escalating inpatient health care costs while simultaneously attempting to improve access to care and outcomes of treatment. During the same time, the medical management of heart failure has become increasingly complex by virtue of: 1) trial results indicating the life-saving efficacy of several classes of medications and/or devices; and 2) the demonstrated preven-

Table. New Classification of Congestive Heart Failure[8]

STAGE		PATIENT DESCRIPTION
A	High risk for developing heart failure	Hypertension Coronary artery disease Diabetes mellitus Family history of cardiomyopathy
B	Asymptomatic heart failure	Previous myocardial infarction Left ventricular systolic dysfunction Asymptomatic valvular disease
C	Symptomatic heart failure	Known structural heart disease Shortness of breath and fatigue Reduced exercise tolerance
D	Refractory end-stage heart failure	Marked symptoms at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions)

tive value of initiating treatment early in patients at high risk for heart failure and those with asymptomatic heart failure (see Table).

The greatest challenges in managing symptomatic or refractory heart failure are: 1) obtaining objective data that signal disease progression or therapeutic effectiveness of specific treatment regimens; 2) inadequate dosing of medications with proven efficacy due to hypotension or bradycardia; 3) failure to detect the need for automatic implantable cardioverter-defibrillator/pacemaker insertion; and 4) poor patient compliance with polypharmacy regimens, salt/fluid intake, or scheduled follow-up leading to acute decompensations. Measurement of hemodynamic parameters such as cardiac output, contractility, systemic vascular resistance, and fluid content of the chest provide important information to augment medical decision-making, and is now possible noninvasively using impedance cardiography techniques.[9,10]

It is well known that the heart chambers, the vascular wall/endothelium, and systemic neurohormones are integrated in function to regulate the delivery of oxygen and blood to the tissues of the body. Disorders or altered reactivity of the vascular wall and/or endothelium can effect the delivery of oxygen regionally or systemically through release of vasoactive substances such as nitric oxide, endothelin, vascular growth factors, etc. Disorders of heart muscle function leading to CHF affect systemic oxygen delivery directly and indirectly through activation of neurohormonal systems (renin-angiotensin-aldosterone-system, sympathetic nervous system, and atrial natriuretic peptides), which exert their effects on both the heart and vascular wall/endothelium. Full activation of these systems in patients with systolic dysfunction typically creates a hemodynamic profile characterized by a low cardiac output, increased systemic vascular resistance, and increased intravascular fluid volume. Drug therapy that increases the heart's contractility by draining contractile reserves, reduces vascular resistance by inhibition of the renin-angiotensin-aldosterone system or the sympathetic nervous system, and reduces intravascular volume by causing diuresis will improve the CHF patient's hemodynamic profile but frequently creates an "over-medicated" state due to inadequate follow-up or reassessment of intravascular fluid status. This situation frequently leads to the development of renal dysfunction, further complicating therapy. There is currently no single oral medication treatment that is capable of increasing cardiac contractility, lowering vascular resistance, and increasing urine flow in patients with low EF. Additionally, the clinical efficacy of current medical therapy for CHF is maintained only while the medication is being taken. There is no long-term benefit to be obtained after short-term medical therapy for CHF.

EECP has been shown to have profound effects on central hemodynamics in patients with symptomatic or refractory left ventricular dysfunction. In our patients, already on optimal angiotensin-converting enzyme inhibitor, β blocker, and diuretic therapy, EECP has increased cardiac output during treatment by more than 75%, reduced systemic vascular resistance by 20%–30%, and improved urine flow enough to restore diuretic responsiveness or allow diuretic dosage reduction. These changes in cardiac output and systemic vascular resistance are larger than any reported responses to oral or intravenous vasodilators or inodilators. EECP was

shown to improve a load-independent measure of ventricular function, pre-load adjusted maximal power[11] indicating augmentation of intrinsic myocardial contractility. Additionally, EECP treatment increases nitric oxide production,[12,13] decreases endothelin levels,[14] increases vascular endothelial growth factor levels,[15] and decreases B-type natriuretic peptide levels[12] suggesting profound improvement in endothelial function, and a reduction of neurohormonal activation. Finally, EECP has demonstrated durable benefits in ischemic patients lasting for many multiples of the treatment duration.[6]

In summary, these results strongly suggest, in my view, that EECP (Vasomedical Inc., Westbury, NY) will play an important role as an adjunctive hemodynamic treatment for CHF. It can readily be used if the patient is hypotensive, bradycardic, or fatigued from other efficacious medication treatments. It can be used whether or not the patient has an arrhythmia or automatic implantable cardioverter-defibrillator/pacemaker. It enhances overall patient functionality as shown in the paper by Soran et al.[3] It should also help reduce poor patient compliance due to polypharmacy by reducing dosage or number of pills taken, and improve follow-up and disease process educational efforts by virtue of the need for daily visits for treatment. The results of the PEECH trial, currently in progress, are eagerly anticipated.

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Enhanced External Counterpulsation in Patients With Heart Failure: A Multicenter Feasibility Study

To assess the feasibility of using enhanced external counterpulsation to treat patients with heart failure, 26 patients with stable heart failure (New York Heart Association classes II–III), with a left ventricular ejection fraction at or below 35%, and without fluid overload, were treated with enhanced external counterpulsation (1 hour daily, 5 days a week, to a total of 35 hours). Patients were followed for 6 months after completing the course of enhanced external counterpulsation. The primary parameter was safety as reflected by adverse events or by changes in laboratory parameters. Secondary end points included changes in exercise capacity and quality of life. There were no clinically significant problems associated with the administration of enhanced external counterpulsation. Significant improvements were seen in exercise capacity (peak oxygen uptake and exercise duration), and in quality of life assessments, at 1 week and 6 months after the course of enhanced external counterpulsation. This study suggests that enhanced external counterpulsation is safe and well tolerated in patients with stable heart failure, and that a randomized, controlled study of enhanced external counterpulsation in these patients is warranted. (CHF. 2002;8:204–208, 227)

Ozlem Soran, MD;[1] Bruce Fleishman, MD;[2] Theresa Demarco, MD;[3] William Grossman, MD;[3] Virginia M. Schneider, RN;[1] Karen Manzo, RN;[2] Paul-André de Lame, MD;[4] Arthur M. Feldman, MD, PhD[1]
From the Cardiovascular Institute of the University of Pittsburgh Medical Center, Pittsburgh, PA;[1] Cardiovascular Research Institute, Columbus, OH;[2] Division of Cardiology, University of California, San Francisco, CA;[3] and Anabase International Corporation, Stockton, NJ[4]

Address for correspondence:
Arthur M. Feldman, MD, PhD, Chief, Cardiovascular Division, University of Pittsburgh, 200 Lothrop Street, S572 Scaife Hall, Pittsburgh, PA 15213-2582
E-mail: feldmanam@msx.upmc.edu
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Enhanced external counterpulsation (EECP; Vasomedical Inc., Westbury, NY) is a noninvasive therapy for patients with stable angina secondary to coronary artery disease[1] and normal or near-normal left ventricular function. By sequentially inflating a series of compressive cuffs wrapped around the lower legs, thighs, and upper thighs upon diastole, and then rapidly deflating the cuffs at end diastole, EECP increases diastolic and decreases systolic blood pressure. In this manner, EECP lowers systemic vascular resistance, decreases afterload, and increases cardiac output.[2–6] These effects achieve a significant reduction in left ventricular oxygen consumption and are accompanied by a transient increase in right ventricular filling pressures,[7] a decrease in serum endothelin, and an increase in serum induced nitric oxide synthetase. In a multicenter, randomized, controlled trial, EECP was effective in relieving angina and increasing time to exercise-induced ST-segment depression in patients with chronic angina pectoris.[8] Salutary effects of EECP have been associated with a significant decrease in reversible perfusion defects as seen on thallium scintigraphy.[6,9]

Because EECP increases right ventricular filling pressures by augmenting venous return during diastole, clinicians have conjectured that its use in patients with left ventricular dysfunction and heart failure would be contraindicated. However, the hemodynamic effects of EECP are similar to those of intra-aortic balloon counterpulsation, with similar diastolic augmentation and afterload reduction. Anecdotal reports have stated that EECP benefits patients with coronary disease and left ventricular dysfunction and an analysis of patients enrolled in the International EECP Patient Registry showed that a left ventricular ejection fraction of less than 35% was not associated with an increase in adverse events during treatment. Furthermore, benefits were similar when compared to patients with preserved left ventricular function.[10]

However, the long-term safety of EECP in patients with symptomatic heart failure and coronary disease and its role in patients with nonischemic heart failure secondary to left ventricular dysfunction have not

been evaluated. The present feasibility/safety trial was designed to address these questions.

Methods

This study was an open, prospective, nonrandomized (single-group) feasibility trial involving three study centers. The study was supervised by a Steering Committee, and monitored by a Data Safety Monitoring Board. Anabase International Corporation (Stockton, New Jersey) performed data collection and management, statistical analysis and monitoring services. The study was conducted in compliance with the Declaration of Helsinki.

A total of 32 patients were enrolled in the study, 11 with idiopathic dilated cardiomyopathy and 21 with ischemic cardiomyopathy. Details of these patients are given in Tables I and II. Informed consent was obtained from all patients prior to study enrollment. Six patients (four idiopathic, two ischemic) withdrew from the study prior to receiving therapy.

Patients of both sexes between the ages of 21 and 81 were eligible for the study if they had a diagnosis of heart failure (New York Heart Association [NYHA] class II or III) despite conventional therapy, left ventricular systolic dysfunction with an ejection fraction of 35% or less, the ability to exercise on a treadmill (Modified Naughton Protocol) with exertion limited by shortness of breath or fatigue, and clinical stability as demonstrated by an absence of medication changes over the 2 weeks prior to the first study visit. Patients were classified as having idiopathic cardiomyopathy if they had no hemodynamically significant coronary artery disease or other known causes of heart failure. Patients with heart failure and documented history of ischemic heart disease and/or diagnostic coronary arteriography were assumed to have ischemic cardiomyopathy.

Patients were excluded from the study if they met any of the following criteria: exercise limited by chest pain, electrocardiographic changes consistent with myocardial ischemia, or claudication; peripheral edema above the ankle; unstable angina; a serum digoxin level greater than 1.5 ng/mL; a serum creatinine level greater than 2 mg/dL; a myocardial infarction or coronary artery bypass grafting within the past 3 months; a cardiac catheterization within the past 2 weeks; acute myocarditis; a history of sudden death; the presence of an implantable cardioverter defibrillator or pacemaker; a history of clinically significant aortic stenosis, mitral stenosis, or aortic regurgitation; chronic obstructive pulmonary disease with forced expiratory volume at one second of 1.5 L or lower; coagulation abnormalities resulting in an international normalized ratio greater than 2.0; or severe hypertension. Because of the requirements for cardiac gating with EECP, patients were excluded if they had arrhythmias that would interfere significantly with triggering of the EECP device. In addition, patients were also excluded if they had a history of deep vein thrombosis, phlebitis, stasis ulcers, and/or pulmonary emboli, because of the potential risks associated with the use of pressurized cuffs around the lower extremities in such patients. Patients were also excluded from the study if they had any condition that precluded diastolic augmentation during initiation of EECP.

Heart failure treatment was optimized and stable prior to enrollment into the study. All patients were treated with an angiotensin-converting enzyme inhibitor unless contraindicated or not tolerated. In patients with ischemic heart disease, calcium channel blockers were permitted if unchanged

Table I. Patient Demographics and Characteristics

GROUP	IDIOPATHIC	ISCHEMIC	OVERALL
N	11	21	32
Age (years; means±SD)	41.0±15.1	64.4±9.6	56.3±16.1
Females (N, %)	2 (18.2)	4 (19.0)	6 (18.8)
LVEF (means±SD)	18.7±7.4	25.6±7.1	23.2±7.8
Angina			
N (%)	—	13 (61.9)	13 (40.6)
Years (means±SD)	—	8.9±7.2	8.9±7.2
Digoxin (N, %)	11 (100.0)	12 (57.1)	23 (71.2)
Carvedilol (N, %)	2 (18.2)	3 (14.3)	5 (15.6)

LVEF=left ventricular ejection fraction

for at least 1 month prior to the study. Likewise, β blockers were allowed provided dosage had been unchanged for at least 6 months. In patients with idiopathic cardiomyopathy, calcium channel blockers were not permitted. Patients were excluded if a calcium channel blocker or a β blocker had been withdrawn less than 3 months prior to the study.

Before treatment, patients were entered into a 2-week screening and baseline period, during which they underwent physical examinations, routine chemistry tests, and two baseline symptom-limited treadmill exercise tolerance tests. If the exercise tests differed by >20%, additional tests were performed; the average of two tests that were within a 20% range was used as the baseline value.

Each patient received 35 1-hour EECP sessions administered once a day, 5 days a week on average, for a total treatment period of 7 weeks. After assessing clinical stability, EECP was initiated and the inflation pressure applied was increased by 0.01 megapascals increments up to a maximum of 0.04 megapascals in all patients (0.04 MPa=300 mm Hg). Oxygen saturation during the treatments was measured by continuous pulse oxymetry. During the study, all drugs were kept as constant as possible, adjustments being made only when medically essential. Injected inotropic agents were not permitted.

Each patient was followed for a period of six months, with study visits at 1 week, 3 months, and 6 months after the end of the treatment period. All assessments performed at baseline were repeated at the 1-week and 6-month follow-up visits. In addition, a clinical assessment was made at the 3-month follow-up visit.

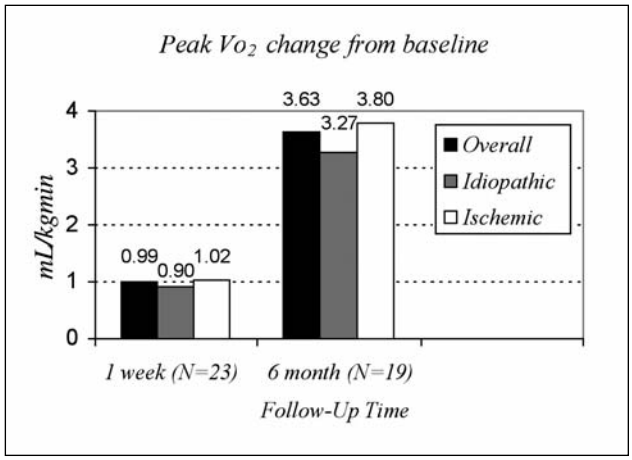

Figure 1. Change in peak oxygen uptake (Vo_2) p values (baseline vs. 1 week and 6 month): overall (p=0.08, p<0.01); idiopathic (p=0.54, p<0.05); ischemic (p=0.09, p<0.01)

The primary study parameter was safety as reflected by adverse events experienced during the administration of EECP, adverse events during the overall treatment period, or changes in laboratory parameters. Other parameters included changes in exercise capacity and quality of life.

Table II. Adverse Events

Occurring During Treatment Period	Related*	Not Related**
Cardiovascular	Arrhythmia, bradycardia	Arrhythmia, palpitations, atrial fibrillation, angina pectoris, worsening heart failure
Noncardiovascular	Shin tenderness, skin abrasion, back problem, muscle pain, swelling under knee	Lightheadedness, nausea, flu-like symptoms, ankle pain, hypothyroidism, edema
Occurring During Follow-up Period	**Related**	**Not Related**
Cardiovascular	—	Atypical chest pain, angina pectoris, arrhythmia, hypotension, electrocardiographic changes, worsening heart failure, pulmonary embolism, atrial fibrillation, shortness of breath, fatigue
Noncardiovascular	—	Upper respiratory tract infection, flu symptoms, gout, urinary retention, increased lipid levels, lightheadedness

*Includes definitely, probably, and possibly related adverse events; **includes probably not-related and definitely not-related adverse events. Some adverse events occurred more than once.

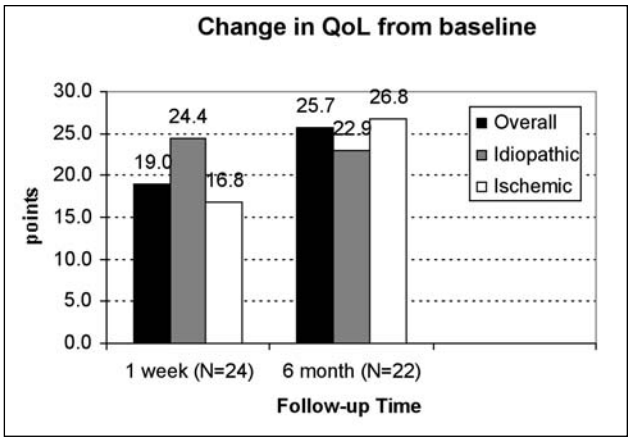

Figure 2. Change in quality of life (QoL) shown as inverse of actual score change; all **p** *values <0.01 except for idiopathic at 6 months (NS)*

Statistical analysis was performed by comparing mean changes of the primary parameters from baseline to study visits at either 1-week post-EECP treatment or at the end of the study, using paired *t* tests.

Clinical examinations and laboratory parameters were summarized at each visit. Change and percent change from baseline are expressed using descriptive statistics (N, Mean, Standard Error, etc.). Paired *t* tests were used within each etiology of heart failure (ischemic or idiopathic) to test whether the change or percent change from baseline was significant.

Peak exercise parameters, Minnesota Living with Heart Failure Questionnaire (MLHFQ) scores,[11] and NYHA status were summarized as described above. These were also analyzed using analysis of variance with etiology of heart failure, study site, and their interaction, as factors (if significant at the 0.15 level). The primary analysis was done using observed cases only and is presented here. A secondary analysis was done using the method of last observation carried forward for imputing missing data, and produced similar results.

Descriptive statistics were also used to summarize demographic variables, medical history, leg discomfort, worsening of condition, and area and pressure ratio. Adverse events were summarized within each etiology of heart failure by incidence, causal relation, intensity, relationship, and action taken.

Results

The demographics and characteristics of the study population are shown in Table I. Of the 32 patients enrolled in the study, 21 had an ischemic etiology for their heart failure and 11 had idiopathic cardiomyopathy. The majority of patients were men. Six of the 32 patients were withdrawn before receiving study treatment, five because they met an exclusion criterion during the baseline period and one because of an adverse event that occurred shortly after the initial visit. The remaining 26 patients (19 with ischemic cardiomyopathy and seven with idiopathic cardiomyopathy) received EECP, but three of these patients were discontinued during the treatment period because of adverse events. The remaining 23 patients completed the treatment, including the 1-week follow-up. Of these, 18 completed the whole study per protocol, and one had a minor protocol violation that did not prevent evaluation of efficacy data at the 6-month follow-up. Four patients had protocol violations sufficient to affect efficacy data at the 6-month follow-up. Thus, a total of 19 patients were evaluated for efficacy at the 6-month follow-up.

Exercise Tolerance Tests

In the 23 patients who had a 1-week post-treatment follow-up, peak oxygen uptake (Figure 1) increased significantly from the mean baseline value of 14.99 to a mean of 15.98 mL/kg/min (change+7.45%; SE±3.57%, min–27.09%, max+43.57%; $p=0.05$).

In the 19 patients who had a 6-month post-treatment follow-up including an exercise tolerance test, peak oxygen uptake increased significantly from the mean baseline value of 14.78 to a mean of 18.41 mL/kg/min (change+27.09%, SE±4.71%, min–0.46%, max+62.76%; $p<0.001$).

At the 1-week follow-up, overall mean exercise duration increased significantly from 627.63 sec at baseline to 732.96 sec (change+20.53%, SE±4.89%, min–7.66%, max+89.58%; $p<0.001$).

At the 6-month follow-up, overall mean exercise duration increased significantly from 637.13 sec at baseline to 715.17 sec (change+15.55%, SE±6.53%, min–15.20%, max+81.02%; $p=0.028$).

Exercise capacity, as measured by peak oxygen uptake and exercise duration, increased similarly in the idiopathic and the ischemic groups (between-population *p* values: peak oxygen uptake 0.408; exercise duration 0.218).

Quality of Life

Quality of life was assessed using the MLHFQ (Figure 2). The MLHFQ was administered at baseline and again 1 week after the end of the treatment period and after the 6-month follow-up.

A total of 24 patients had a post-treatment MLHFQ (including one who was discontinued from the study but had this test 1 month after the end of treatment). In these 24 patients, the overall changes between the test results at baseline and 1-week post-treatment were significant ($p<0.01$) for total score, physical dimension and emotional dimension.

In the 22 patients who completed the study and had the MLHFQ at 6-month follow-up visit, total score showed persistent improvement over baseline values, but only the change in emotional dimension remained significant ($p<0.01$).

Adverse Events

The population available for safety analysis consisted of the 32 patients enrolled in the study, 26 of whom received one or more EECP treatment sessions. Overall, there were 844 treatment sessions performed during which the safety of EECP was assessed by monitoring oxygen saturation and cardiac condition. Except for three instances in the same subject of a worsening of preexisting arrhythmia, there were no cases of worsening of heart condition, particularly heart failure, as a result of the application of EECP in this patient population. As described in Table II, there were 46 adverse events reported in 23 patients; 22 occurred during the treatment period (i.e., between the first treatment session and the first follow-up 1 week after the end of treatment), and 24 during 6-month follow-up. Of these 46 adverse events, 14 were classified as serious (led to hospitalization) and involved eight patients. One occurred during the baseline period; three occurred during the treatment period and, although serious, none were related to the EECP device. Ten cardiovascular adverse events occurred during the 6-month follow-up period.

Of the 22 adverse events that occurred during the treatment period, 10 occurred during the actual application of EECP (treatment session), and 12 outside a treatment session. Two of the 10 adverse events that occurred during a session resulted in treatment discontinuation. One subject was discontinued from the study after 22 treatment sessions for worsening back pain; the other was hospitalized twice for worsening of heart failure and discontinued from the study after the second episode. A third subject had increased frequency of arrhythmia requiring treatment discontinuation after 23 sessions; this subject remained in the study and had follow-up evaluations.

None of the adverse events reported during the 6-month post-treatment follow-up period were related to direct or indirect effects of EECP therapy. No significant changes in clinical laboratory values were identified.

Discussion

The present study showed that when carefully applied and monitored, the application of 35 1-hour daily sessions of EECP over approximately 7 weeks was safe and well-tolerated in patients with relatively stable heart failure and no fluid overload. Although EECP treatments were limited by arrhythmias in some patients, there were no clinically significant problems associated with the administration of EECP in this group of patients with symptomatic heart failure. Furthermore, adverse events that were seen in patients outside the EECP treatment sessions could not be attributed to EECP itself but rather were expected consequences of the disease. Although it had been intended to enroll patients who were stable and presumably NYHA class II–III, the median ejection fraction of the population was 23%, consistent with clinical trials enrolling NYHA class II–IV heart failure patients.

The results of the present study, albeit in a small population and in the absence of a control group, also suggest that EECP can provide short- and long-term benefits to selected patients with chronic stable heart failure. These benefits, consisting of significant and persistent increases in exercise capacity (peak oxygen uptake and exercise duration), were observed 1 week after the end of the 35-session EECP treatment period and were still present at the 6-month follow-up. It appears that the study subjects benefited from EECP to a similar degree, regardless of the ischemic or nonischemic etiology of their heart failure.

While a diminution in the ischemic burden in patients with ischemic heart failure may explain the salutary effects in patients with coronary disease, the mechanism of the beneficial effects of EECP in patients with idiopathic cardiomyopathy remains less obvious. Patients with dilated cardiomyopathy have lower perfusion pressure resulting in decreased coronary flow and insufficient oxygen supply at the cellular level secondary to ventricular hypertrophy. By increasing perfusion pressure and decreasing cardiac work, EECP might improve oxygen supply and promote the recovery of otherwise non-functional areas of the myocardium.

Other possible mechanisms of benefit from EECP in heart failure include the effects of EECP on endothelial function. One such effect is an increase in nitric oxide,[12,13] with subsequent coronary and systemic vasodilation.

Alternatively, EECP has been shown also to decrease levels of endothelin-1, a potent endothelium-derived vasoconstrictor that is felt to contribute to the pathogenesis of heart failure.[13,14]

Conclusions

EECP appears safe when applied as adjunct therapy for heart failure. The efficacy results of this study must be viewed with care, as the size of the study population was small and comparisons were not made with a control group. However, the salutary effects of EECP on functional capacity in the ab-

sence of adverse events are intriguing. The efficacy results suggest that EECP can improve exercise capacity, quality of life and functional status, both in the short term and also for a period of 6 months after completion of the EECP therapy.

Importantly, the results of this feasibility study warrant the initiation of a randomized controlled study to ascertain the efficacy of EECP as an adjunctive therapy in the management of patients with chronic stable heart failure.

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