Vasomedical Inc. EECP®

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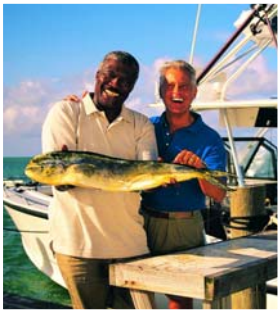
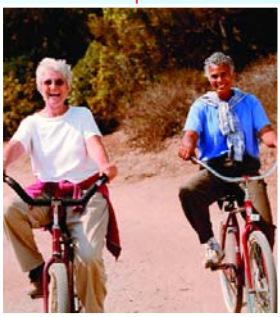




The Physician's Guide to EECP® Therapy

Table of Contents

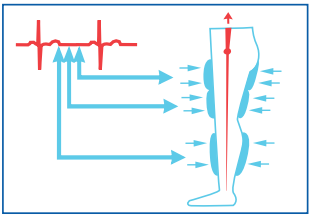
A patient receiving EECP® therapy.

Overview of EECP Therapy

EECP therapy, offered exclusively by Vasomedical, Inc., is a noninvasive treatment for patients with angina and heart failure. EECP treatment is typically provided on an outpatient basis in 35 one-hour sessions over a period of approximately seven weeks. Additional hours may be safely and effectively added to this standard regimen if physician evaluation of the patient's response to treatment determines additional hours will result in greater relief of angina symptoms, i.e., "treating to target." In certain circumstances, adjusting the patient's treatment regimen to two hours per day can make it more comfortable for the patient to complete a course of EECP therapy.

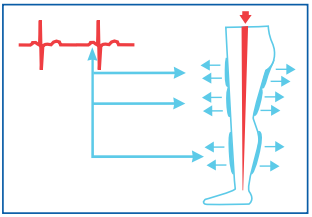
To receive EECP therapy the patient lies on a treatment table. Compressive cuffs (similar to large blood pressure cuffs) are securely wrapped around the patient's calves, thighs and buttocks. These cuffs inflate in a distal to proximal sequence in early diastole, and deflate simultaneously in late diastole just prior to the onset of systole. Inflation and deflation are specifically timed to the patient's ECG to optimize therapeutic benefit. The sequential cuff inflation creates a retrograde pressure wave that augments diastolic pressure, increasing coronary perfusion pressure and venous return to the right heart (increasing preload and cardiac output). Rapid, simultaneous cuff deflation decreases systemic vascular resistance, afterload, and cardiac workload.

Sequential Inflation



- Retrograde aortic pressure wave
- Increased diastolic pressure
- Increased intracoronary perfusion pressure
- Increased myocardial perfusion
- Increased venous return
- Increased preload
- Increased cardiac output

Simultaneous Deflation



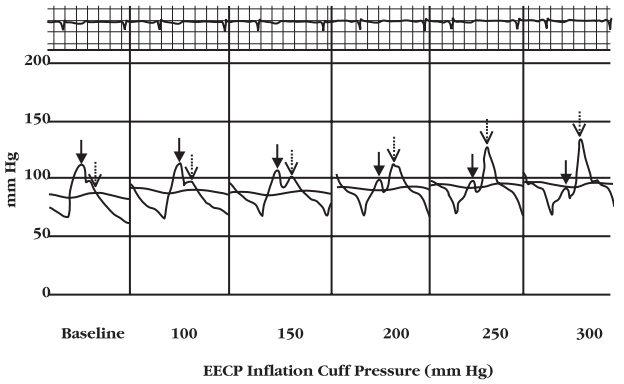
- Decreased systemic vascular resistance
- Decreased cardiac workload
- Decreased myocardial oxygen consumption
- Decreased afterload

Clinical studies and data from the International EECP Patient Registry (IEPR), coordinated by the Epidemiology Data Center at the University of Pittsburgh, continue to demonstrate that 70–80% of patients realize therapeutic benefit immediately upon completion of a course of EECP therapy. At patient follow-up, therapeutic benefit is enhanced at six months and sustained at 24 months post treatment.[1] Quality-of-life measures from a randomized trial and registry studies show significant improvement in the patients' ability to resume activities of daily living, social interaction, and recreational pursuits.[1,2]

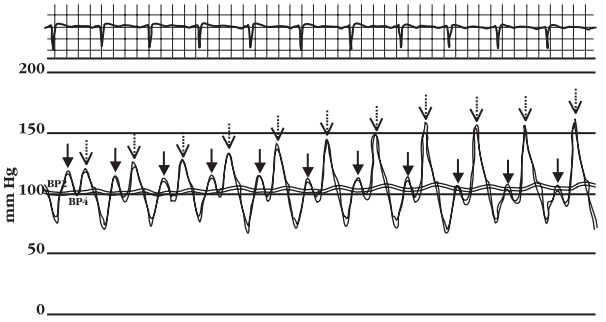
Hemodynamic Effects of EECP Therapy

Studies have shown that the hemodynamics of EECP therapy closely resemble those of the intra-aortic balloon pump (IABP),[3] long held as the "gold standard" for circulatory support of hemodynamically compromised patients. The magnitude of diastolic augmentation that can be achieved with EECP therapy was found comparable to that of the IABP, resulting in improved coronary blood flow with decreased cardiac workload. Michaels, et al,[4] in a landmark cath lab study, demonstrated the hemodynamics of EECP in the central vasculature:

"Treatment with EECP unequivocally and significantly increases central aortic and intracoronary diastolic pressure and intracoronary blood flow velocity. Mean aortic and intracoronary pressure is increased, and left ventricular systolic unloading occurs during EECP."



Intracoronary phasic and mean pressure tracings at baseline and at increasing cuff inflation pressure in the EECP device. With increasing inflation pressure, the diastolic (dashed arrows) and mean pressures increase, whereas the systolic pressure (solid arrows) decreases. Paper speed is 25 mm/sec.[4]



Simultaneous central aortic and intracoronary pressures demonstrate a gradual increase in peak diastolic (dashed arrows) and mean pressures with a decrease in peak systolic pressure (solid arrows) attributable to systolic unloading as the inflation pressure is increased to 300 mm Hg in the EECP device. Paper speed is 25 mm/sec.[4]

Patient Selection and Treatment

EECP therapy is a safe and effective treatment that provides sustained duration of benefit in patients with disabling angina and angina equivalents,[1,5,6] left ventricular dysfunction (LVD),[7] and heart failure.[8,9,10] EECP therapy is indicated for use in stable and unstable angina pectoris, congestive heart failure, acute myocardial infarction, and cardiogenic shock.

Patients that may benefit from EECP Therapy

Patients with angina or angina equivalents who:[7,11,12]

- No longer respond to medical therapy
- Restrict their activities to avoid angina symptoms
- Are unwilling to undergo additional invasive revascularization procedures
- Have LVD (EF <35%)
- Have co-morbid conditions that increase the risk of revascularization procedures (e.g., diabetes, heart failure, pulmonary disease, renal dysfunction)
- Have coronary anatomy unsuitable for surgical or catheter-based revascularization
- Are considered inoperable or at high risk of operative/interventional complications
- Suffer with microvascular angina (Cardiac Syndrome X)

Heart failure patients in a euvolemic state with:[7,9,10]

- Ischemic or idiopathic cardiomyopathy
- LVD (EF <35%)
- Co-morbid conditions that increase the risk of complications of revascularization procedures

Diabetic patients known to be at greater risk for post-procedural complications.[13]

Elderly patients at high risk for morbidity and mortality associated with invasive coronary interventions.[14]

Physician Guidelines

• Conduct a comprehensive pre-EECP therapy clinical status assessment by history and physical examination.	Verify that the patient remains clinically stable before beginning EECP treatment. The heart failure patient should be stabilized for at least two weeks prior to initiating EECP therapy.
• Fluid volume status must be managed, especially in the heart failure/LVD patient population.	Monitor and adjust medical therapy to achieve and maintain a euvolemic state to prevent the onset or recurrence of fluid retention and pulmonary congestion.
• EECP treatment should be withheld if there is exacerbation of heart failure symptoms.	Treatment may be resumed once symptoms have subsided and the patient has been stabilized.
• Patients with severely compromised cardiac function appear to be at greater risk for adverse clinical events with EECP treatment.	The rates of occurrence of adverse events are within expectations for patients with the types and severity of illnesses indicated for EECP treatment.[15]
• Careful patient selection, thorough patient evaluation before, during and after each treatment, vigilant patient monitoring, a properly equipped facility, and appropriate medical supervision will serve to optimize the safety and effectiveness of EECP therapy and minimize the possibility of adverse events.	An EECP therapist skilled in patient assessment, experienced in caring for angina and heart failure patients, and trained in the appropriate delivery of EECP therapy is key to the safe and effective administration of EECP treatments.

Treatment Guidelines

• Instruct the patient to refrain from eating or drinking 90 minutes before treatment.	A large meal or excess fluid intake may cause discomfort during EECP therapy.
• All medications should be taken as prescribed unless approved by the physician.	Diuretics may be held with physician approval to avoid multiple interruptions during treatment.
• Patients should wear pantyhose under treatment tights to minimize the possibility of skin irritation.	Significant skin irritation could require cessation of therapy until lesions are healed.
• Weigh the patient prior to each EECP therapy session. The patient should be weighed in treatment pants for accuracy.	Change in body weight is a reliable gauge for monitoring short-term changes in fluid status. An increase in weight of two pounds or more overnight, or four pounds in a week, may indicate exacerbation of heart failure. Withhold treatment and advise the physician. With a five-pound increase in weight from pre-treatment baseline, withhold treatment and notify the physician.
• Check degree of peripheral edema.	If ≥ 1+, withhold treatment and notify the physician.
• Take and record the pulse, noting heart rate and rhythm.	Resting tachycardia, changes in heart rate or rhythm may indicate changes in clinical status. Hold treatment and advise the physician.

• Take and record blood pressure.	For pressures ≥ than 180/110, ≤ than 80/50, or with a narrow pulse pressure, withhold treatment and advise the physician.
• Count and chart respiratory rate and characteristics.	Rapid, shortened breaths or a new wet cough may be indicative of fluid overload. Withhold treatment, advise the physician, and monitor the patient.
• Listen to breath sounds.	Rales, "crackles," or any change in breath sounds may be indicative of fluid overload. Withhold treatment, advise the physician, and monitor the patient.
• Oxygen saturation should be monitored before, during and after each EECP treatment hour. Frequency of assessment is guided by the patient's clinical status – every 20 minutes at a minimum is recommended.	If a 3–5% decrease in SpO_2 is noted pretreatment from day to day, withhold treatment, notify the physician, and monitor the patient. A decrease in SpO_2 may be noted during EECP therapy as the patient relaxes and/or treatment pressure induces shallow respirations. If oxygen saturation decreases without accompanying symptoms of pulmonary congestion, encourage the patient to take several deep breaths. If this does not return oxygen saturation to the pretreatment level, stop treatment, notify the physician, and monitor the patient.
• Initiate EECP treatment and achieve full treatment pressure within five minutes of applying pressure.	Effective systolic unloading of increased venous return diminishes the possibility of pulmonary congestion.
• Elevate the patient's head to maximize comfortable breathing.	Avoids dyspnea and promotes patient comfort and relaxation.
• Record during-session plethysmography tracings every 20 minutes at a minimum.	Evaluate and adjust inflation/deflation timing to ensure optimum therapeutic benefit.
• Observe the patient throughout the treatment hour.	***Patients should never be left unattended while receiving EECP therapy.*** Changes in symptoms, vital signs, weight, oxygen saturation, skin color, mentation, the presence of edema, rales, jugular venous distention, or S3 gallop should be communicated to the physician. EECP treatment should be withheld until physician assessment has been completed.
• Repeat physical assessment and vital signs before the patient leaves the treatment area.	The ability to monitor and treat potential complications and rapid access to emergency care are significant factors in the decision to treat patients with a higher risk of procedural morbidity.
• Adjust treatment regimen to two hours per day to accommodate transportation difficulties, distance traveled, or work-related time constraints.	Treatments should be limited to one hour per day for the first week to gauge the patient's tolerance. Two-hours-per-day treatments should be separated by a minimum 30-minute break.

Contraindications and Precautions

Contraindications

- Arrhythmias that interfere with machine triggering
- Bleeding diathesis
- Active thrombophlebitis
- Severe lower extremity vaso-occlusive disease
- Presence of a documented aortic aneurysm requiring surgical repair
- Pregnancy

Precautions

- Patients with blood pressure higher than 180/110 mmHg should be controlled prior to treatment with enhanced external counterpulsation.
- Patients with a heart rate of more than 120 bpm should be controlled prior to treatment with enhanced external counterpulsation.
- Patients at high risk of complications from increased venous return should be carefully chosen and monitored during treatment with enhanced external counterpulsation. Decreasing cardiac afterload by optimizing cuff inflation and deflation timing may help minimize increased cardiac filling pressures and the possibility of pulmonary congestion due to increased venous return.
- Patients with clinically significant valvular disease should be carefully chosen and monitored during treatment with enhanced external counterpulsation. Certain valve conditions, such as significant aortic insufficiency or severe mitral or aortic stenosis, may prevent the patient from obtaining benefit from diastolic augmentation and reduced cardiac afterload in the presence of increased venous return.

Key Clinical Studies

Clinical trials of external counterpulsation date back to the 1960's and have been ongoing since then. While most of the clinical trials have focused on the application of EECP therapy in the angina patient population, the earliest studies were done in the areas of cardiogenic shock and acute myocardial infarction. The Multicenter Study of Enhanced External Counterpulsation (MUST-EECP,[6] 1995–1997), remained the only randomized, double-blinded, placebo-controlled EECP study until the PEECH™ trial (Prospective Evaluation of EECP in Congestive Heart Failure) was initiated in March 2001. This blinded, randomized, multicenter trial of EECP in the heart failure patient population was preceded by a heart failure pilot study,[9] the positive results of which prompted the larger, multicenter effort.

Reduction in anginal episodes, increased exercise times,[6] and significant improvement in health-related quality-of-life scores[2] have been demonstrated in controlled studies and in the outcome data from Phases 1 and 2 of the International EECP Patient Registry (IEPR). Since January 1998, the IEPR has documented day-to-day clinical practice with EECP therapy, resulting in rapid expansion of the data set available on the outcomes of EECP treatment. In addition, significant benefits were demonstrated in such diverse patient populations as the elderly,[14] diabetics,[13] patients with LVD,[7] patients refractory to medical therapy, and patients no longer candidates for surgical or catheter-based revascularization.

MUST-EECP

Arora, et al,[6] Columbia-Presbyterian Medical Center, New York, NY

The MUST-EECP trial was a randomized, controlled, double-blinded study carried out at seven leading university hospitals in the United States. The study evaluated the effect of EECP therapy testing the reproducibility of benefits observed in earlier studies. Patients were randomized to active (full EECP treatment pressure) or sham (minimal EECP pressure) treatment groups.

Patients in the active EECP therapy group demonstrated a statistically significant increase in time to exercise-induced ST segment depression when compared to sham and baseline, and reported a statistically significant decrease in the frequency of angina episodes when compared to sham and baseline. Exercise duration increased significantly in both groups; however, the increase was greater in the active EECP group.

IEPR Phases 1 and 2

In January 1998, Phase 1 of the IEPR was established to document patient characteristics, safety, efficacy, and the long-term outcomes of EECP therapy for patients treated in the clinical practice setting. The IEPR is a voluntary registry enrolling consecutive angina patients, open to all EECP providers. At the close of Phase 1 in July 2001 more than 5,000 patients had been enrolled. The treatment outcomes (decrease in anginal symptoms and nitroglycerine usage, improvement in quality of life) reported by the IEPR confirm those seen in the MUST-EECP trial, as well as results seen in other studies. Analysis of long-term outcomes demonstrates that the clinical benefits achieved with EECP therapy are sustained up to at least 24 months following an initial course of treatment.[1]

IEPR Phase 2, initiated in January 2002 with a 2,500-patient enrollment goal, augments the chronic stable angina patient data of Phase 1 with the addition of heart failure-specific data points.

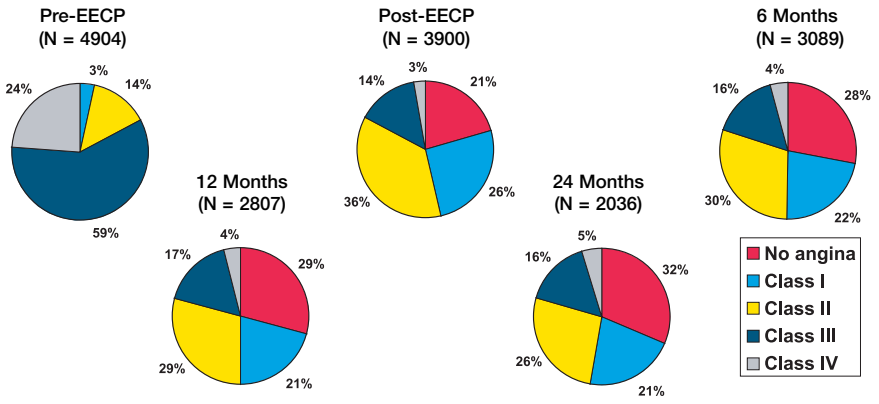
The IEPR has provided data for abstract presentations at all major cardiology meetings held worldwide in addition to providing data for publications in the major peer-reviewed cardiology journals. Information about the IEPR and the complete IEPR bibliography can be found at: www.edc.gsph.pitt.edu/iepr.

EECP Therapy: Duration of Clinical Benefit

Post-EECP, 6, 12 and 24-month data reflect patients who completed the prescribed course of EECP treatment (at least 35 hours) from 99 sites.

		Pre-EECP (N = 4904)	Post-EECP (N = 3900)	At 6 months (N = 3089)	At 12 months (N = 2807)	At 24 months (N = 2036)
Patient Demographics:	No angina (%)	-	20.5	28.0	29.1	31.5
Mean age 66.9 years	Class I (%)	3.2	25.8	22.1	20.9	21.4
Age > 65 60.4%	Class II (%)	13.9	36.6	29.7	29.3	26.5
Male gender 75.3%	Class III (%)	58.9	14.4	16.0	16.8	16.0
Medical History:	Class IV (%)	24.0	2.7	4.1	4.0	4.7
Duration of CAD 10.9 years	Improved by ≥ 1 class (%)		82.3			
Prior PCI/CABG 85.7%	Improved by ≥ 2 classes (%)		43.9			
Prior MI 67.6%	No increase in angina since post-EECP (%)			78.3	73.7	73.8
CHF 31.4%	Decrease in angina since pre-EECP (%)		82.3	80.8	79.8	80.9
Diabetes 42.2%	prn Nitro use (%)	69.0	35.5	42.0	43.8	44.5

N = number of patients reporting angina at these time points



Congestive Heart Failure Feasibility Study
Soran, et al,[7] University of Pittsburgh, PA

This multicenter feasibility study demonstrated that with judicious patient selection, careful application, and monitoring, EECP therapy was well-tolerated in euvolemic, stable heart failure patients. EECP therapy appears safe when applied as an adjunct therapy in this patient population. Efficacy data suggest that EECP therapy may improve exercise capacity and functional status, and enhance quality of life in the short-term and for six months after completion of a course of therapy. The results of this feasibility study supported findings of reports from the IEPR on angina patients with LVD or a history of heart failure,[7,10] and prompted initiation of the PEECH trial.

Central Hemodynamics
Michaels, et al,[4] University of California, San Francisco, CA

In this cath lab study Michaels, et al demonstrated that:

> *"EECP unequivocally and significantly increases diastolic and mean pressures and reduces systolic pressure in the central aorta and coronary artery. Coronary artery flow, determined by both Doppler and angiographic techniques, is increased during EECP. The combined effects of systolic unloading and increased coronary perfusion pressure provide evidence that EECP may serve as a potential mechanical assist device."*

*Summary of Study Results**

Biochemical Markers	Functional Measures	Clinical Outcomes
Increase in Nitric Oxide Levels	Increase in Time to ST Depression	Improvements in CCS** Angina Class
Decrease in Endothelin Levels	Increase in Exercise Tolerance	Reduction in Anginal Episodes
Decrease in BNP Levels	Improvement in Peak Oxygen Consumption	Reduction in Use of Nitrates
Increase in VEGF Levels	Increase in Cardiac Contractility	Improvement in Quality of Life
	Reduction in Systemic Vascular Resistance	Clinical Benefits Sustained Long-Term
	Decrease in Peak Systolic Pressure	
	Increase in Ejection Fraction	
	Increase in Cardiac Output	
	Increase in Intracoronary Pressure and Blood Flow Velocity	** Canadian Cardiovascular Society

*see pp. 14–15

State-of-the-Art Paper
Summary of Evidence Supporting EECP Benefits
Bonetti, et al,[16] Mayo Clinic, Rochester, MN

This review paper recognizes that a growing knowledge base supports the safety and beneficial effects associated with EECP, and recognizes the therapy as the only noninvasive technique for which both a reduction in anginal symptoms and an improvement in objective measures of myocardial ischemia have been shown in patients with refractory angina.

Published Controlled & Uncontrolled Trials of EECP in Patients With Stable Angina[1]								
Study (Ref.)	Year	N	Treatment Duration (h)	Angina (% ≥1 CCS)*[2]	Nitrate Use	Exercise Tolerance	Time to ST Depression	Cardiac Perfusion (%)*
Lawson et al.	1998	60	35	↓		↑		↑ (75)
Arora et al.	1999	139	35	↓	↓	↑	↑	
Lawson et al.	2000	33	35-36	↓	↓			↑ (79)
Lawson et al.	2000	2,289	35	↓ (74)*				
Urano et al.	2001	12	35			↑	↑	↑
Masuda et al.	2001	11	35			↑	↑	↑
Stys et al.	2001	395	35	↓ (88)*				
Barsness et al.	2001	978	35	↓ (81)*	↓			
Stys et al.	2002	175	35	↓ (85)		↑		↑ (83)

1 Adapted from Bonetti, et al[16]
*% of patients for whom this criterion applies are listed in parentheses.
2 CCS = Canadian Cardiovascular Society
↓ = Reduced, ↑ = Increased

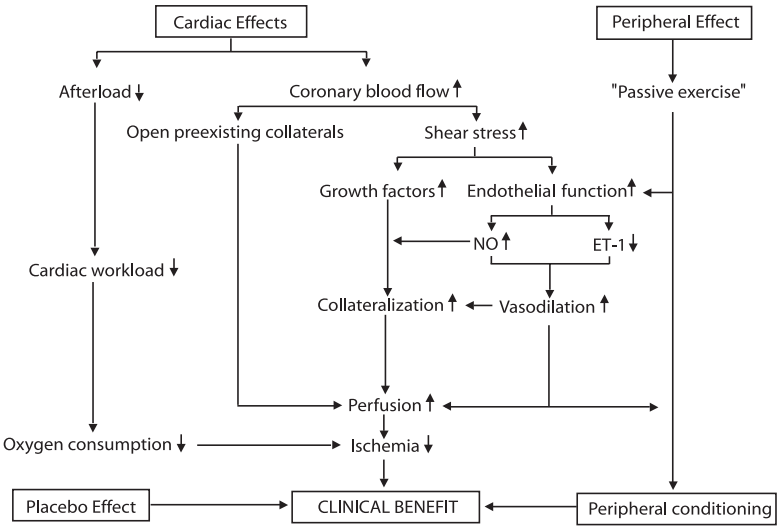
Mechanisms of Action[16,18,20-24]

The specific mechanisms of action of EECP therapy are not as yet defined. However, clinical studies continue to identify components of the physiological, neurohumoral, and hemodynamic cascades initiated by the sequential cuff inflation and simultaneous cuff deflation during treatment.

EECP therapy produces beneficial effects that appear mediated through arterial diastolic augmentation. The retrograde arterial pressure wave increases coronary perfusion pressure, creating a gradient between ischemic and non-ischemic areas of the myocardium that may recruit latent conduits and enhance myocardial perfusion. Increased endothelial shear stress releases growth factors. Increased nitric oxide (NO) and atrial natriuretic peptide (ANP) levels, and decreased endothelin (ET-1) and brain natriuretic peptide (BNP) levels, also ascribed to increased shear stress and improved endothelial function, raise the possibility of peripheral benefits as well as restored coronary flow reserve.

Bonetti, et al[16] point out the suggested mechanisms contributing to the clinical benefit of EECP including improvement in endothelial function, promotion of collateralization, enhancement of ventricular function, and peripheral effects similar to those observed in response to regular physical exercise.

In a study evaluating the effects of EECP therapy using reactive hyperemic-peripheral arterial tonometry (RH-PAT) as a measure of peripheral endothelial function, Bonetti, et al[22] conclude, “that EECP is associated with an acute improvement in peripheral endothelial function, as is demonstrated by the acute increase in RH-PAT index observed in response to EECP on the first three study days. Moreover, the significant difference between RH-PAT indices before the course of EECP and at one-month follow-up suggests that EECP also exerts a beneficial medium-term effect on endothelial function.”



Possible mechanisms responsible for the clinical benefit associated with EECP. By increasing coronary blood flow, EECP is thought to promote myocardial collateralization via opening of pre-formed collaterals. Increased blood flow and shear stress may also improve coronary endothelial function favoring vasodilation and myocardial perfusion. Besides a peripheral training effect, a minor placebo effect is considered to contribute to the symptomatic benefit of EECP. ET= Endothelin; NO = Nitric Oxide.
Adapted from Bonetti, et al.[16]

Reimbursement

Under Medicare Coverage Policy (35–74) reimbursement is provided for the use of EECP therapy for patients diagnosed with disabling angina (Canadian Cardiovascular Society Classification Class III or Class IV, or equivalent classification) who, in the opinion of a cardiologist or cardio-thoracic surgeon, are not readily amenable to surgical intervention such as PTCA or cardiac bypass because: (1) their condition is inoperable, or at high risk of operative complications or post-operative failure; (2) their coronary anatomy is not readily amenable to such procedures; or (3) they have co-morbid conditions which create excessive risk.

Most private insurance carriers offer reimbursement for EECP therapy and have established coverage criteria similar to Medicare.

References

[1] Michaels AD, Linnemeier G, Soran O, Kelsey SF, & Kennard ED. (2004). Two-Year Outcomes After Enhanced External Counterpulsation for Stable Angina Pectoris (from the International EECP Patient Registry [IEPR]). *American Journal of Cardiology*, 93, 461–464.

[2] Arora RR, Chou TM, & Jain D, et al. (January, 2002). Effects of Enhanced External Counterpulsation on Health-Related Quality of Life Continue 12 Months After Treatment: A Substudy of the Multicenter Study of Enhanced External Counterpulsation. *Journal of Investigative Medicine, 50*(1), 25–32.

[3] Taguchi I, Ogawa K, & Oida A, et al. (November 15, 2000). Comparison of Hemodynamic Effects of Enhanced External Counterpulsation and Intra-Aortic Balloon Pumping in Patients with Acute Myocardial Infarction. *The American Journal of Cardiology, 86*(10), 1139–1141.

[4] Michaels AD, Accad M, & Ports TA, et al. (September 3, 2002). Left Ventricular Systolic Unloading and Augmentation of Intracoronary Pressure and Doppler Flow During Enhanced External Counterpulsation. *Circulation, 106*(10), 1237–1242.

[5] Lawson WE, Hui JCK, & Cohn PF. (April, 2000). Long-Term Prognosis of Patients with Angina Treated with Enhanced External Counterpulsation: Five-Year Follow-Up Study. *Clinical Cardiology, 23*(4), 254–258.

[6] Arora RR, Chou TM, & Jain D, et al. (June, 1999). The Multicenter Study of Enhanced External Counterpulsation (MUST-EECP): Effect of EECP on Exercise-Induced Myocardial Ischemia and Anginal Episodes. *The Journal of the American College of Cardiology, 33*(7), 1833–1840.

[7] Soran O, Kennard ED, Holubkov R, Strobeck J, & Feldman AM. (2002). Enhanced External Counterpulsation as a Treatment for Chronic Angina Patients with Left Ventricular Dysfunction: A Report from the International EECP Patient Registry (IEPR). *CHF, 8*, 297–302.

[8] Soran O. (January-February, 2004). A New Treatment Modality in Heart Failure. *Cardiology In Review, 12*(1), 15–20.

[9] Soran O, Fleishman B, & DeMarco T, et al. (July-August, 2002). Enhanced External Counterpulsation in Patients with Heart Failure: A Multicenter Feasibility Study. *Congestive Heart Failure, 8*(4), 204–8, 227.

[10] Lawson WE, Kennard ED, Holubkov R, Kelsey SF, Strobeck JE, Soran O, & Feldman AM. (2001). Benefit and Safety of Enhanced External Counterpulsation in Treating Coronary Artery Patients with a History of Congestive Heart Failure. *Cardiology, 96*, 78–84.

[11] Kronhaus KD, & Lawson WE. (2002). Is Enhanced External Counterpulsation the Treatment Choice for Microvascular Angina? *Journal of Investigative Medicine, 51*(Suppl 2), S360 (26).

[12] Crawford LE. (2002). Experience with Enhanced External Counterpulsation (EECP) in Coronary Artery Disease. *Today's Therapeutic Trends, 20*(3), 243–252.

The complete EECP bibliography can be found at www.eecp.com.

[13] Linnemeier GC, Kennard ED, & Soran O, et al. (2003). Enhanced External Counterpulsation for the Relief of Angina in Patients With Diabetes: A One-Year Clinical Outcome Study. *American Heart Journal, 146*(3), 453–8.

[14] Linnemeier G, et al. (2003). Enhanced External Counterpulsation in the Management of Angina in the Elderly. *American Journal of Geriatric Cardiology, 12*(2), 90–94.

[15] Lawson WE, & Hui JCK. (November, 2000). Enhanced External Counterpulsation for Chronic Myocardial Ischemia. *The Journal of Critical Illness, 15*(11), 629–636.

[16] Bonetti PO, et al. (June 4, 2003). Enhanced External Counterpulsation for Ischemic Heart Disease. *Journal of the American College of Cardiology, 41*(11), 1918–25.

[17] Urano H, Ikeda H, & Ueno T, et al. (January, 2001). Enhanced External Counterpulsation Improves Exercise Tolerance, Reduces Exercise-Induced Myocardial Ischemia and Improves Left Ventricular Diastolic Filling in Patients with Coronary Artery Disease. *Journal of the American College of Cardiology, 37*(1), 93–99.

[18] Urano H, Lida S, & Fukami K, et al. (October, 2000). Intermittent Shear Stimuli by Enhanced External Counterpulsation (EECP) Restores Endothelial Function in Patients with Coronary Artery Disease. *Circulation, 102*(18 Suppl 2), II–57(266).

[19] Masuda D, Nohara R, & Hirai T, et al. (August, 2001). Enhanced External Counterpulsation Improved Myocardial Perfusion and Coronary Flow Reserve in Patients with Chronic Stable Angina; Evaluation by 13N-Ammonia Positron Emission Tomography. *European Heart Journal, 22*(16), 1451–1458.

[20] Wu GF, Qiang SZ, & Zheng ZS, et al. (1999). A Neurohormonal Mechanism for the Effectiveness of Enhanced External Counterpulsation. *Circulation, 100*(18), I-832(4390).

[21] Feldman AM. (December, 2002). Enhanced External Counterpulsation: Mechanism of Action. *Clinical Cardiology, 25*(12 Suppl 2), II 11–15.

[22] Bonetti PO, Barsness GW, & Keelan PC, et al. (May 21, 2003). Enhanced External Counterpulsation Improves Endothelial Function in Patients with Symptomatic Coronary Artery Disease. *Journal of the American College of Cardiology, 41*(10), 1761–1768.

[23] Michaels AD, Kennard ED, & Kelsey SE, et al. (June, 2001). Does Higher Diastolic Augmentation Predict Clinical Benefit from Enhanced External Counterpulsation?: Data from the International EECP Patient Registry (IEPR). *Clinical Cardiology, 24*(6), 453–458.

[24] Lakshmi M, Kennard ED, Kelsey SF, Holubkov R, & Michaels AD. (2002). Relation of the Pattern of Diastolic Augmentation During a Course of Enhanced External Counterpulsation (EECP) to Clinical Benefit [from the International EECP Patient Registry (IEPR)]. *American Journal of Cardiology, 89*, 1303–1305.

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