Enhanced external counterpulsation for chronic myocardial ischemia

How to use a nonpharmacologic, noninvasive treatment for patients with angina

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ABSTRACT: Enhanced external counterpulsation (EECP) produces sequenced compression of the lower extremities during diastole. The "milking" effect on the arterial beds of the lower extremities augments aortic diastolic pressure and thereby increases coronary perfusion pressure and blood flow. The simultaneous compression of venous beds increases venous return and cardiac filling pressure. EECP treatment of patients with chronic angina can improve myocardial perfusion as demonstrated by radionuclide stress imaging, time to significant ST-segment depression during exercise testing, and functional status. EECP may be especially worth considering for patients with severe, diffuse coronary artery disease in whom targets for surgical revascularization are lacking and those who have persistent angina after 1 or more revascularization procedures. (*J Crit Illness.* 2000;15(11):629-636)

Enhanced external counterpulsation (EECP) and intra-aortic balloon counterpulsation serve similar purposes and have the same historic roots. Almost 35 years ago, it was shown that withdrawing blood from the arterial system during systole and returning it during diastole could improve coronary perfusion, decrease cardiac workload, and reduce myocardial oxygen consumption.[1] Dr Richard Gorlin coined the term "counterpulsation" to describe this process.[1] Before long, counterpulsation methods had diverged into the internal and the external. Internal counterpulsation evolved into the currently used intra-aortic balloon technique.

Initially, external counterpulsation was accomplished with cumbersome, uncomfortable hydraulic devices that encased the patient's lower limbs as if in a water-filled suit of armor. As external counterpulsation evolved, pneumatic devices supplanted hydraulic ones. In 1992, we and our colleagues first described "enhanced" external counterpulsation (EECP): the current sequenced, pneumatic system using 3 pairs of cuffs (Figure 1).[2] In this article, we explain the working principles of EECP, which patients with chronic myocardial ischemia are candidates for EECP treatment, and how the procedure is performed.

Principles of EECP

EECP has a marked effect on cardiovascular hemodynamics. Sequenced (from the calves upward) compression of the lower extremities during diastole drives arterial blood backward, augmenting aortic diastolic pressure and causing retrograde flow in the descending aorta (Figure 2). Augmenting aortic diastolic pressure increases coronary perfusion pressure (which is the difference between aortic diastolic pressure and left ventricular [LV] intracavitary pressure) and, consequently, coronary blood flow. This acute effect is similar to that achieved with the intra-aortic balloon pump. The increase in coronary pressure and flow causes an increase in shear stress in the coronary arteries.

Unlike the intra-aortic balloon pump, EECP also increases venous return, since the lower extremity venous beds are sequentially compressed, with a "milking" ef-

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fect, at the same time as the arterial beds. Hemodynamic studies have shown that EECP increases cardiac filling pressures. This results, presumably through the Starling mechanism, in a greater increase in cardiac output than is achieved with the intra-aortic balloon pump.[3]

After an extended course of EECP treatment, myocardial perfusion as demonstrated by stress radionuclide imaging improves, an effect that suggests recruitment or development of collateral vessels. Patients with coronary artery disease (CAD) who have 1 or more proximally patent coronary arteries or bypass grafts demonstrate higher rates of response to EECP. This observation has generated the hypothesis that the proximally patent vessel or vessels allow transmission of the augmented diastolic pressure and coronary blood flow to the distal coronary bed, enhancing the recruitment or development of collaterals.[4,5]

Beneficial neurohormonal effects also occur over the course of treatment, possibly through the effect of increased shear stress on endothelial cells.[6-8] Progressive increases in nitric oxide levels and decreases in endothelin-1 levels suggest a tendency toward normalization of endovascular function. Reductions in malondialdehyde levels reflect decreased lipid peroxidation. These effects are sustained after treatment and may be responsible for some observed post-treatment changes, including a peripheral conditioning effect resembling that of exercise[9] as well as improved coronary vasomotor tone and function and cardiac perfusion. All of these mechanisms may contribute to the persistent clinical benefits demonstrated for up to 5 years after treatment.[10,11]

Indications

Patients with CAD and angina often have a choice of effective treatments. Surgical revascularization and angioplasty may offer the greatest relief of symptoms and improvement in exercise tolerance. Reductions of mortality after revascularization, in comparison with medical therapy, are more difficult to ascertain. A survival advantage after revascularization has been reliably demonstrated for certain subsets of patients with CAD: those with stenosis of the left main or proximal left anterior descending (LAD) coronary artery or with 3-vessel disease, particularly when the arterial disease is accompanied by LV dysfunction.

Which patients, then, are candidates for EECP? Although definitive, evidence-based indications have not been established, the benefits of EECP for certain groups of patients seem clear. EECP may be the therapeutic procedure of choice for patients with severe, diffuse CAD involving 1 or more vessels when targets for surgical revascularization are lacking or endovascular reconstruction with multiple small-diameter stents is the alternative. For these patients, surgery would not be expected to improve mortality, and the success rate associated with angioplasty is low. Patients with chronic angina, preserved LV function, and 1- or 2-vessel CAD not involving the left main or proximal LAD artery, for whom revascularization offers no demonstrated survival advantage, may also be candidates for EECP, as an alternative to revascularization or medical therapy. For these patients, EECP can be considered when medical therapy alone does not provide satisfactory control of angina

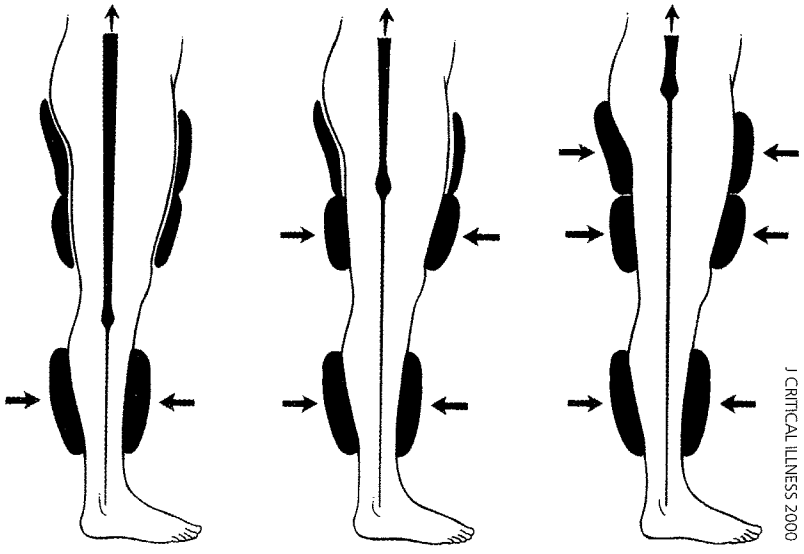

*Figure 1 – **Three sets of paired, inflatable cuffs** that are wrapped around the patient's legs are currently used to perform enhanced external counterpulsation (EECP). The cuffs are inflated during diastole in a timed sequence: the calves first, followed by the thighs and then the upper thighs. All pressure is released at the onset of systole. The sequential compression of the lower extremities increases venous return and augments arterial diastolic pressure. The R wave of the patient's ECG is used as the trigger signal for inflation and deflation. The control panel of the EECP device, which is used to adjust cuff pressure and the timing of cuff inflation and deflation, also provides both ECG monitoring and monitoring of pulse augmentation by finger plethysmograph.*

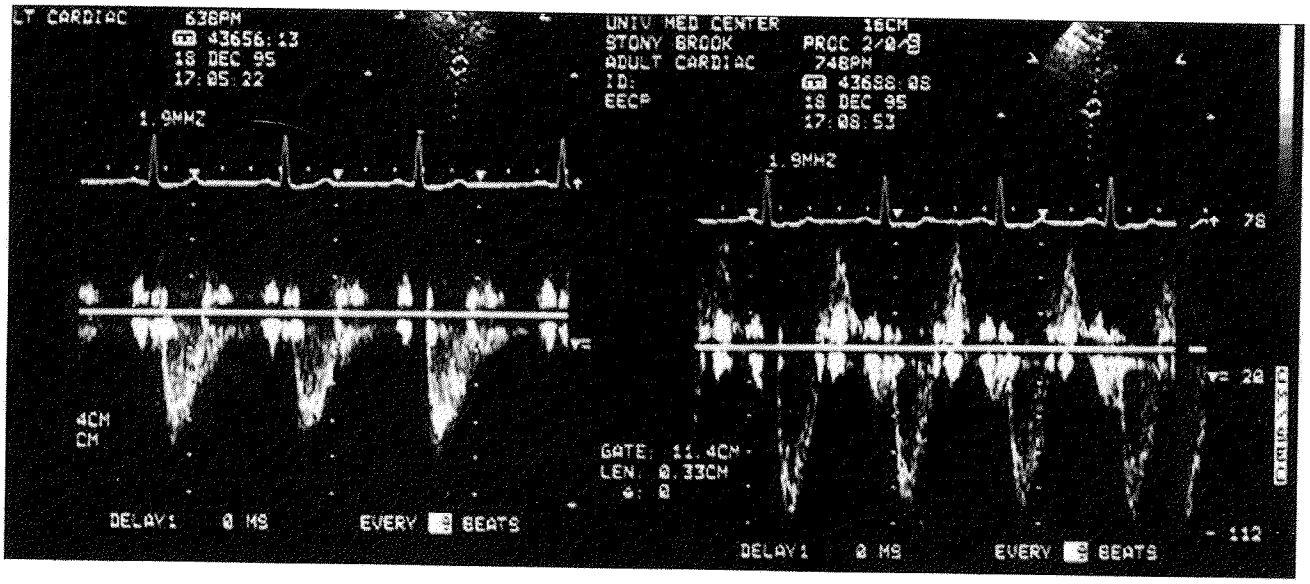

*Figure 2 – **Retrograde flow in the descending aorta** during diastole is seen in the pulsed wave Doppler echocardiogram on the right, obtained during enhanced external counterpulsation (EECP). The left panel is a control echocardiogram of the descending aorta in the same patient just before activation of the EECP system. The right panel shows that the anterograde systolic stroke distance is increased during EECP, reflecting increased cardiac output.*

and revascularization is not necessary or not possible.

Another large group of patients for whom EECP is worth considering comprises those who have undergone 1 or more revascularization procedures but have persistent angina and still-unrevascularized areas of myocardium. Even small amounts of myocardium at risk may be associated with chronic, limiting angina. Patients in this category have a very favorable rate of response (70% to 80%) to EECP treatment,[4,5,12] which may improve both the perfusion defects seen in stress imaging studies and the associated anginal symptoms.

The physician's or patient's individual preference is an important consideration. Some physicians have found EECP a useful means of decreasing the "ischemic burden" when it is desirable to postpone or avoid definitive revascularization therapy. Patients have also elected EECP with the goal of lessening their requirement for antianginal medications,[2] avoiding revascularization,[12] or improving functional status[13] and quality of life.[14]

Not all patients with angina can be treated with EECP (Table 1). Patients with uncontrolled congestive heart failure (CHF), severe pulmonary hypertension or systemic hypertension (blood pressure greater than 180/110 mm Hg), moderate or severe aortic insufficiency, significant coagulopathy (for example, an international normalized ratio greater than 2.0), severe peripheral vascular disease, active thrombophlebitis, or uncontrolled arrhythmia are not candidates for EECP.

The ability to monitor and treat potential complications dictates whether EECP can be considered for certain patients with a higher risk of procedural morbidity.

Ready access to inpatient care facilities is a prerequisite for the treatment of higher-risk patients with EECP. Patients with a moderately to severely decreased LV ejection fraction or a history of CHF are in this category, since the increase of venous return during EECP may precipitate pulmonary congestion or edema. The pretreatment evaluation of such patients must ascertain that they have no pretibial or presacral edema or pulmonary

Table 1 – Contraindications for EECP

•Uncontrolled CHF
•Severe pulmonary hypertension
•Severe systemic hypertension (blood pressure > 180/110 mm Hg)
•Moderate or severe aortic insufficiency
•Significant coagulopathy (for example, INR > 2.0)
•Severe peripheral vascular disease
•Active thrombophlebitis
•Uncontrolled arrhythmia

EECP, enhanced external counterpulsation; CHF, congestive heart failure; INR, international normalized ratio.

Table 2 – Important points in performing EECP

Conduct pretreatment examination before each EECP session
Question patient about changes in health, especially pattern of angina
Assess lower extremities (look for areas of erythema, ecchymoses, edema, abrasions, ulcerations, or other signs of trauma)
Exclude fluid overload (look for evidence such as pretibial or presacral edema, ascites, or pulmonary congestion)
Measure the patient's resting blood pressure, respiratory rate, and pulse to exclude severe hypertension, respiratory compromise, tachycardia, and significant arrhythmia
Patients must urinate immediately before treatment because the cuffs exert pressure on the bladder and EECP may promote diuresis
A spandex body stocking or bicycle pants covering the lower extremities decreases the risk of barotrauma during EECP; avoid additional padding, except over bony prominences
The EECP console provides ECG and finger plethysmographic monitoring; pulse oximetry monitoring should be added for higher-risk patients, such as those with a moderately to severely decreased LV ejection fraction or a history of CHF

EECP, enhanced external counterpulsation; LV, left ventricular; CHF, congestive heart failure.

rales. The periprocedural monitoring should include pulse oximetry, since oxygen desaturation may be a marker of incipient acute CHF. Current reports indicate that EECP, used with appropriate precautions, is safe and effective for treating angina in patients with LV dysfunction[15,16] and those with a history of CHF.[13]

Patients with unstable, crescendo, or rest angina may also have an unpredictable response to EECP treatment. Again, monitoring with pulse oximetry is desirable.

Technique

Current EECP systems include a treatment bed, compressor, and control console. The inflation/deflation valves, which are incorporated into the bed, have 3 outlets to which the 3 pairs of cuffs are attached via hoses. The R wave of the patient's ECG is used as the trigger signal for inflation and deflation: On detection of the R wave,

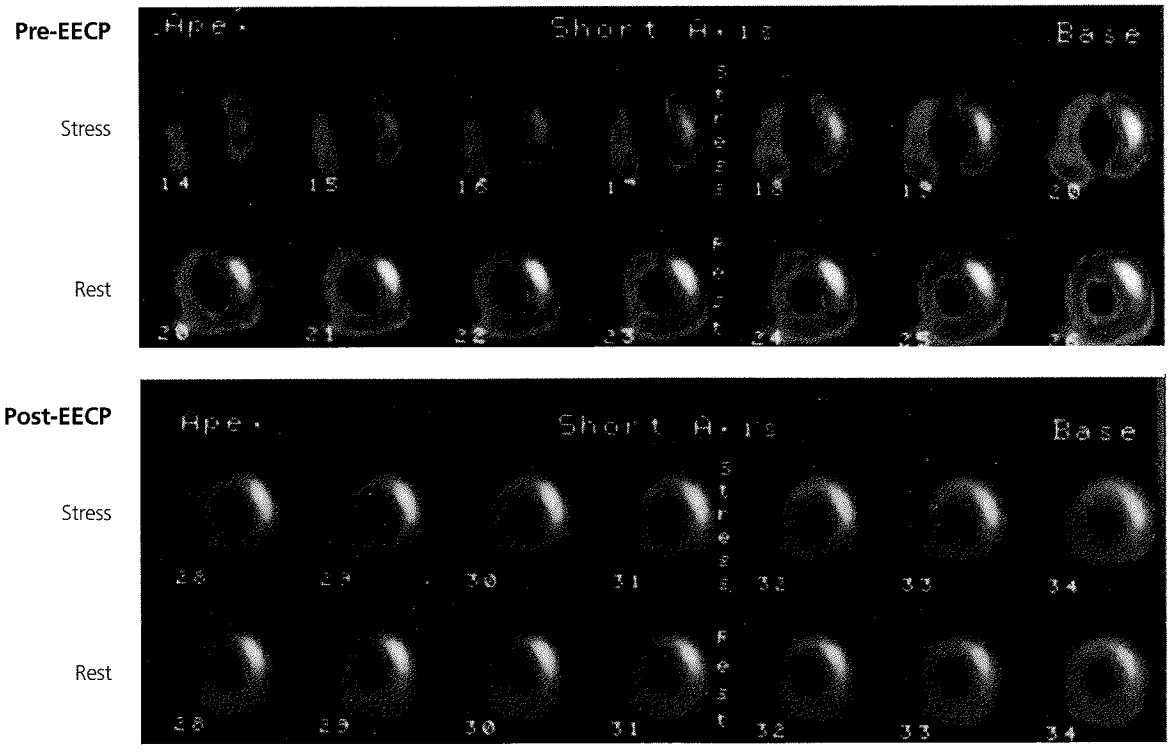

*Figure 3 – **Myocardial perfusion in the anterior and posterolateral walls** improved after a 35-hour course of enhanced external counterpulsation (EECP) treatment. The pre- and post-EECP tests reveal septal and inferoposterior scarring.*

a delay of 0.4 times the square root of the preceding R-R interval is used to initiate sequential inflation. Simultaneous deflation of all cuffs occurs 50 milliseconds before the next anticipated R wave. The control panel, which is used to adjust cuff pressure and the timing of cuff inflation, also provides both ECG monitoring and monitoring of pulse augmentation by finger plethysmograph. Currently available EECP devices are capable of generating pressures up to 350 mm Hg; in clinical practice, the usual upper limit is 250 to 275 mm Hg.

With the patient lying supine on the treatment bed, the cuffs are inflated during diastole in a timed sequence: the calves first, followed by the thighs and then the upper thighs. Inflating the cuffs on the lower extremities increases venous return and augments arterial diastolic pressure. Unlike hydraulic instruments, a pneumatic device using multicompartmental, sequential inflation does not cut off arterial inflow or trap venous outflow. Such a device allows more effective arterial and venous bed compression, optimizing venous return and arterial counterpulsation.

EECP is performed in a variety of settings, including physicians' offices, rehabilitation facilities, free-standing centers that perform EECP exclusively, and hospitals. In general, trained personnel, oxygen, nitroglycerin, and a sphygmomanometer, stethoscope, electrocardiograph, and emergency communication system should be at hand wherever EECP is performed.

Before each treatment session, the patient should be questioned about changes in his or her health, with special attention to the pattern of angina (Table 2). A physical examination should be performed, including assessment of the lower extremities for areas of erythema, ecchymoses, edema, abrasions, ulcerations, or other signs of trauma or vascular compromise. To exclude fluid overload, look for evidence such as pretibial or presacral edema, ascites, or pulmonary congestion. Measure the patient's resting blood pressure, respiratory rate, and pulse to exclude severe hypertension, respiratory compromise, tachycardia, and significant arrhythmia.

Patients must urinate immediately before treatment because the cuffs exert pressure on the bladder and because treatment may promote diuresis by increasing cardiac output, renal blood flow, and atrial natriuretic peptide levels.[17,18] A spandex body stocking or bicycle pants covering the lower extremities is the most comfortable garment for the patient to wear during EECP and decreases the risk of barotrauma. Additional padding, except over bony prominences, is generally to be avoided, since it lessens the effectiveness of EECP.

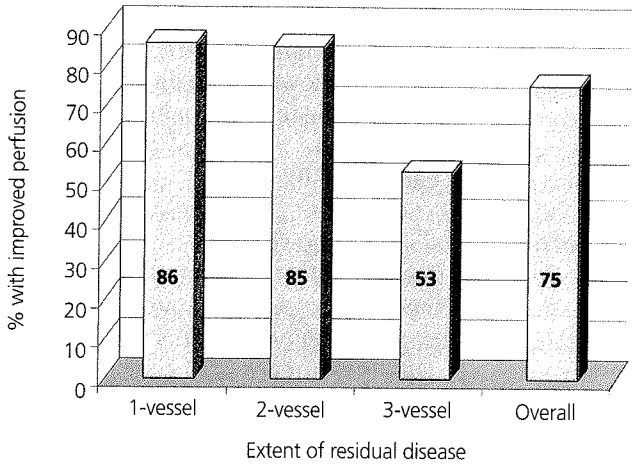

*Figure 4 – **The response to enhanced external counterpulsation** was influenced by the extent of residual (unrevascularized) coronary artery disease. Patients with unrevascularized 3-vessel disease or 3-vessel disease with occluded bypass grafts showed the lowest rate of improvement in radionuclide stress test results. (Data from Lawson WE et al.* Clin Cardiol. *1998.[5])*

Efficacy and safety

The efficacy of EECP in treating angina has been evaluated in case reports, case-control studies, and 1 randomized, blinded, placebo-controlled study (Multicenter Study of EECP [MUST-EECP]).[19] In addition, 2 registry studies have addressed questions of efficacy, patient selection, and adverse effects of therapy.[13,14] The results have been consistent for all end points: time to ST-segment depression, exercise tolerance, improvement of radionuclide stress perfusion defects, severity of angina (Canadian Cardiovascular Society [CCS] functional class), and quality of life.

The MUST-EECP trial enrolled 139 patients, at 7 university hospitals, who had documented CAD, angina, and positive stress test results.[19] Patients received active EECP (pressure, 300 mm Hg) or, as a sham (placebo) treatment, inactive EECP (pressure, 75 mm Hg) for a 35-hour course of treatment. After treatment, time to ST-segment depression of 1 mm or more on stress testing increased significantly ($P = .01$ for the difference between groups) in the active-EECP group (from 337 ± 18 to 379 ± 18 s; $P < .002$) compared with the control group (326 ± 21 to 330 ± 20 s; $P < .74$). There was a parallel decrease in anginal episodes and nitroglycerin use in the active-treatment group. The most common adverse events during treatment were local traumatic effects (skin abrasion, bruising, blisters, leg and back pain).

A placebo effect was observed in the MUST-EECP

study, with patients who received inactive EECP also demonstrating an improvement in post-treatment exercise tolerance. There was, however, no difference after treatment in this group's time to 1-mm or greater ST-segment depression. At 6 and 12 months, quality-of-life measures (bodily pain, social functioning, and health and functional status indexes from the Medical Outcomes Study Short Form [SF-36]) showed increasing separation between the active- and inactive-treatment patients, with the former showing progressive improvement and the latter remaining at baseline.[20,21] This suggests the emergence of a clear EECP treatment benefit after the initial masking effect of a transient placebo benefit in the inactive-treatment group.

Case-control studies have evaluated exercise tolerance, functional change, and myocardial stress perfusion after EECP treatment in patients with chronic angina. In a group of 27 patients treated with EECP,[9] exercise duration on a Bruce-protocol stress test increased by a mean of 1.9 minutes. Improved exercise tolerance was seen in 81% of the patients and improved radionuclide stress test results in 78%.

In a large, single-center, case-control study of 60 patients with known coronary anatomy, treatment benefit was assessed by radionuclide stress testing.[10] Overall, 75% of patients demonstrated improvement in radionuclide stress perfusion test results after treatment (Figure 3). Patients with and without prior revascularization responded similarly. The rate of response was lowest among the patients with the most diffuse disease and the fewest patent proximal conduits: namely, those with unrevascularized 3-vessel disease or 3-vessel disease with occluded bypass grafts (Figure 4). Clinical follow-up has demonstrated initial and sustained (3-year) improvement in stress myocardial perfusion and angina in patients who responded to treatment[10] as well as a reduction of the 5-year incidence of major adverse cardiovascular events.[11]

The results reported by the EECP Clinical Consortium[14] have been consistent with those of the published short-term studies: about 69% of angina patients who were treated with EECP improved by 1 or more CCS functional classes (Figure 5). Preliminary results from the International EECP Patient Registry (IEPR), in which almost 2000 consecutively treated patients are currently enrolled, reveal a similar rate of response to EECP treatment and a low incidence of adverse effects.[22]

Overall, the available data suggest that EECP is an effective treatment for chronic angina that is refractory to medical therapy or revascularization. With treatment, there is improvement in objective measures of ischemia (time to ST-segment depression, myocardial stress perfusion) as well as more subjective measures (such as functional status, severity of angina, nitroglycerin use,

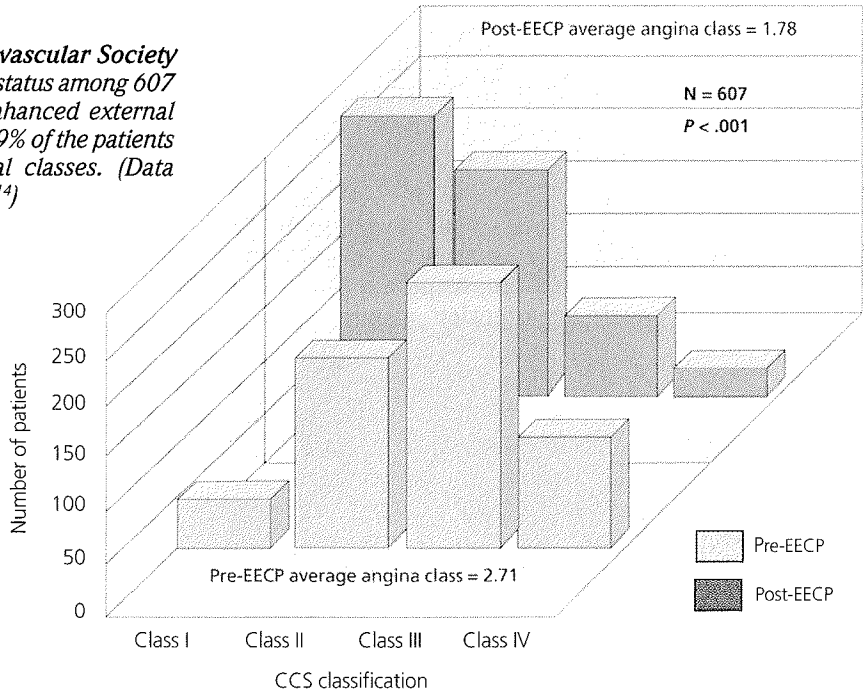

*Figure 5 – **Change in Canadian Cardiovascular Society (CCS) class** reflected improved functional status among 607 patients with angina who underwent enhanced external counterpulsation (EECP). Approximately 69% of the patients improved by 1 or more CCS functional classes. (Data from Lawson WE et al.* Eur Heart J. *1998.[14])*

and quality of life) in 70% to 80% of treated patients. The observed adverse effects of EECP treatment, which are rare and mainly related to local trauma, include edema, skin breakdown, and musculoskeletal pain.

Expanding treatment horizons

Wider application of EECP to the treatment of cardiovascular disease appears to be in the offing. Preliminary multicenter studies coordinated by the University of Pittsburgh,[15] as well as IEPR data,[16] suggest that EECP can be administered safely to selected patients with moderate or severe LV dysfunction. Other preliminary data suggest a potential role for EECP in treating patients with cardiomyopathy.[23]

Another area of interest is the use of EECP in the management of acute coronary syndromes and acute myocardial infarction. In these settings, the procedure might serve as a bridge to more definitive revascularization, providing increased myocardial perfusion and circulatory support during the interim. It might also facilitate clot lysis and platelet disaggregation by increasing pulse pressure and flow in the coronary arteries. Work is under way to develop mobile EECP units that could be used in emergency departments and chest pain units and during interhospital transfers.

The EECP-induced increase in venous return might help maintain cardiac output and blood pressure in patients with shock. With units combining EECP and intra-aortic balloon counterpulsation, the 2 modalities might be sequentially activated in such a way that the retrograde aortic wave initially produced by compression of the peripheral vascular bed could be augmented by intra-aortic balloon pump displacement of blood from the descending aorta. This might produce a greater magnitude of diastolic pressure augmentation and increased venous return and thus provide maximal circulatory support during cardiopulmonary resuscitation or profound shock.

If it is shown to be cost-effective, EECP may move farther toward the front line of therapy for angina. The procedure is noninvasive, easy to perform, and safe. It may be particularly useful as a treatment alternative when revascularization must be delayed or avoided. For patients whose angina is controlled by medical therapy, EECP may have potential to lessen the need for medications, improve function, and perhaps reduce costs.

Preliminary investigations of the neurohormonal effects of EECP have demonstrated changes that suggest improved endovascular function. If EECP can help normalize endothelial function, it might reduce the frequency of major adverse cardiovascular events in a fashion similar to that associated with the 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors.[24] If these findings are supported by future investigations, EECP may eventually be incorporated into the practice of primary and secondary cardiovascular disease prevention and cardiac rehabilitation.

CLINICAL CONCLUSIONS:

Using EECP to manage chronic myocardial ischemia

1 Enhanced external counterpulsation (EECP) augments aortic diastolic pressure and causes retrograde flow in the descending aorta. The acute effect is similar to that achieved with intra-aortic balloon counterpulsation: an increase in coronary perfusion pressure and blood flow. However, EECP also augments venous return, producing a greater increase in cardiac output than is achieved with the intra-aortic balloon technique.

2 EECP may be the therapeutic procedure of choice for patients with severe, diffuse coronary artery disease (CAD) involving 1 or more vessels when targets for surgical revascularization are lacking. EECP is also worth considering for patients who have undergone 1 or more revascularization procedures but have persistent angina. Patients with chronic angina, preserved left ventricular (LV) function, and 1- or 2-vessel CAD may be candidates for EECP.

3 Before each EECP treatment session, question the patient about changes in his or her health, with special attention to the pattern of angina. To exclude fluid overload, look for evidence such as pretibial or presacral edema, ascites, or pulmonary congestion. Measure the patient's resting blood pressure, respiratory rate, and pulse to exclude severe hypertension, respiratory compromise, tachycardia, and significant arrhythmia.

4 Ready access to inpatient care facilities is a prerequisite for the EECP treatment of higher-risk patients. In this category are patients with a moderately to severely decreased LV ejection fraction or a history of congestive heart failure and those with unstable, crescendo, or rest angina. The periprocedural monitoring for such patients should include pulse oximetry.

5 Reported adverse effects of EECP are rare and mainly related to local trauma. They include edema, skin breakdown, and musculoskeletal pain.

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