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EECP and Coronary Artery Disease

For patents with symptomatic coronary artery disease, enhanced external counterpulsation is a newer option that is the only available noninvasive outpatient treatment.

By Marta Bill, PA-C, PhD

Coronary artery disease is the leading cause of death in the United States, responsible for about 500,000 deaths each year.[1] The U.S. Public Health Service first began to address the escalating incidence of cardiovascular disease in the late 1950s. Researchers there decided to undertake a large-scale study to find out why heart disease was becoming the leading cause of death in the United States. The study sought to learn which biological and environmental factors were responsible for the rapid increase of morbidity and mortality since 1930s.

A large-scale study known as the Framingham Heart Study was conceived. Two generations of more than 300 families—the residents of Framingham, Mass.—were the study subjects.[2] The study continued for 50 years and provided valuable epidemiologic data.

Before Framingham, it had been largely believed that heart disease was a part of the aging process. The study identified major risk factors of CAD: hypertension, smoking and hypercholesterolemia. This research greatly impacted preventive medicine in the United States and around the world. According to Framingham, 7 million people in the United States experience angina pectoris annually. The prevalence of the disease for persons older than 30 is 213 cases for every 100,000.[3]

Conventional Treatments

CAD treatment involves primary and secondary prevention with modification of manageable risk factors (hypercholesterolemia, hypertension and smoking). The benefits of preventive medicine, although important, remain limited, since only half of coronary events are related to these factors.[4]

Pharmacologic options for symptomatic CAD include beta-blockers, calcium-channel blockers and long-acting nitrates. Interventional methods include percutaneous coronary interventions (PCIs) such as stents or angioplasty, and coronary artery bypass grafting (CABG). Some patients may not be candidates for these options because of unsuitable coronary anatomy, multiple previous revascularization attempts or comorbid conditions.[4-6]

The limitations of these strategies include adverse effects of medications, procedure-related morbidity and mortality, restenosis after PCI and time-dependent graft attrition after CABG surgery. Patient preferences may also limit conventional options.[4] Previous revascularization and repeated angioplasty procedures significantly increase the morbidity and mortality associated with the next procedure. These patients are not considered for further interventional procedure.

Many patients have tried all available therapeutic options and still continue to have symptoms that significantly impair quality of life.[1,3,5]

Patients With Refractory CAD

The population with refractory CAD grows as more patients survive a primary coronary event.[1,7] The increasing prevalence of CAD is also a contributing factor. According to the American Heart Association, nearly 1 million patients underwent CABG or PCI.[8] Of these patients, more than 125,000 continued to have symptomatic angina.

Enhanced external counterpulsation (EECP) is a noninvasive outpatient treatment for CAD refractory to medical and surgical therapy. The technique provides augmentation of diastolic blood flow and coronary blood flow, similar to an intraaortic balloon pump. It utilizes serial inflation of three sets of cuffs wrapped around the calves, lower thighs and upper thighs.[9,10] During diastole, the cuffs inflate sequentially (applying 250 to 300 mm Hg of external pressure) from the calves to the lower thighs and upper thighs to raise diastolic aortic pressure and increase coronary perfusion flow.[11] Compression of the vascular bed of the legs also increases venous return. Rapid simultaneous deflation of the cuffs at the onset of systole creates significant left ventricular unloading and thereby decreases cardiac workout. Inflation and deflation are timed to the patient's electrocardiogram. Decreased myocardial oxygen demand and recruitment of collateral vessels are the additional benefits of EECP.[12]

The exact mechanisms of benefits of EECP remain unclear, but available data have led to three hypotheses: enhanced diastolic flow, changes in the neurohormonal environment and changes in ventricular function independent of changes in a cardiac load.[13]

Enhanced Diastolic Flow

The arterial hemodynamic effect of EECP is similar to that seen with the intraaortic balloon pump, with the generation of a retrograde venous

pulse. Enhanced diastolic flow subsequently increases shear stress, which is a factor known for activating release of antigenic growth factors promoting angiogenesis and new collateral vessel development.[13,14] Increased shear stress studied in vitro activates the tyrosine-kinase pathway in the endothelium, which modifies the actin cytoskeleton. This modification is responsible for endothelial and smooth muscle migration, resulting in new vessel formation to ischemic area of the heart muscle.

The effect of counterpulsation on collateral vessel formation has been studied since 1963, when Jacobey and colleagues investigated the development of collaterals in dogs under a number of different stimuli. The collateral formation was monitored with coronary angiography. At that time the cellular and molecular mediators responsible for angiogenesis were yet unknown, and the results of the study were received with skepticism. The discovery of vascular endothelial growth factor (VEGF), a very potent angiogenic factor, allowed for advances in the angiogenesis study.

Recently, the influence of the single episode of EECP therapy on a significant increase in VEGF level that persists for up to a month was assessed.[13] Another recent study on ocular blood velocities during EECP in healthy volunteers and patients with atherosclerosis assessed fundus hemodynamic with Doppler flow flowmetry. The results revealed significant blood flow improvement in atherosclerotic patients and no significant change in healthy subjects.[13] This finding may explain the increase of perfusion in patients with retinal ischemia after EECP treatment.

Changes in Neurohormonal Environment

This neurohormonal environment hypothesis implies the improvement of vascular reactivity as a beneficial effect of EECP. The effect of the method is compared with exercise. The increased vascular shear forces created centrally and peripherally during EECP improve endothelial function and vascular reactivity. The vascular effect seems to be mediated by neurohormonal environment changes.[13,15]

The EECP treatment decreases levels of plasma endothelial particles, which are a potent vasoconstrictor, although only during the episode of treatment, and increases nitric oxide, a potent vasodilator. The increase in NO level persists for up to one month. The influence of the EECP on the level of natriuretic peptidesatrial natriuretic factor and brain natriuretic peptidehas been investigated, as well. Both hormones promote diuresis; in addition, BNP is a very sensitive predictor of left ventricular dysfunction. Serum ANF significantly raised and peaked after 35 hours, then the level of hormone declined, remaining above baseline after two months post-treatment.[15] BNP level decreased markedly after one day of treatment and continued to lower after one week. This finding may reflect the improvement of ventricular function. EECP may restore distributed vascular function by down-regulation of endothelin and promoting NO formation. The direct effect on myocardium can be conducted from decreased BNP, and the recent positron emission tomography studies reviled enhanced myocardial perfusion effect of EECP.[3]

Changes in Ventricular Function

The ventricular function changes hypothesis is based on the assumption that EECP changes ventricular function independently of changes in the coronary vasculature. The first clinical studies on efficacy of the method excluded the patients with left ventricular dysfunction. However, the concern that EECP would worsen symptoms in patients with left ventricular dysfunction was revised in other studies. The data suggested that EECP could be applied safely in cardiac failure.[8]

The patients with refractory angina and left ventricular dysfunction benefited the same way as the patients with refractory angina without cardiac failure. The effects of EECP were measured with a shift in angina classification, improvement in oxygen uptake and mean exercise duration noted one week and six months post-treatment.

The prospective studies undertaken to define the role of EECP in patients with heart failure used echocardiography and finger plethysmography and showed a few interesting findings. The heart rate following EECP at three and six months was reduced markedly, the left ventricular maximal power increased, and at six months there was a significant improvement in the left ventricular force or contractility.[13]

Clinical Evidence of Beneficial Effects

Clinical benefits associated with EECP treatment include increased time until onset of ischemia, reduction in severity and frequency of anginal episodes, decrease in nitrate use, improvement in angina class and greater exercise workout.[8,16] The large, randomized, double-blinded analysis, the MUST-EECP trial, reported the improvement in quality of life. The changes in ability to perform activities of daily living, ability of work, bodily pain, confidence in health, energy, ability to engage in social activities, anxiety and depression were measured and continued at one year.[7]

Recent trials have included more diverse populations, and the results have shown that some factors may influence the positive outcome of the EECP treatment. Diabetes, a history of smoking and left ventricle dysfunction predict reduced effectiveness of EECP,[6,11,17] although one study reported no differences in a favorable outcome between diabetic and nondiabetic populations.[16] Diabetes correlates with more extensive vascular damageimpaired endothelium function, angiogenesis and propensity to thrombosis. A nonsmoking history as a positive predictor of EECP effectiveness surprised researchers, because the effects of smoking, such as vasoconstriction, endothelial damage and hypercoagulability, are considered relatively transient.[15] Regardless of the presence of these predictors, the effectiveness of the EECP treatment in these subgroups is approximately 70%.[12,18]

EECP Indications

The FDA has approved EECP for chronic stable angina and congestive heart failure.[8] In December 1999, the American College of Cardiology evaluated and endorsed EECP therapy for the treatment of patients with debilitating (functional class III and IV) refractory angina who are symptomatic despite maximal pharmacotherapy, are not candidates for angioplasty or bypass graft surgery and have no contraindications to

EECP use.[6-8] The most common cause of left ventricle dysfunction in the United States is ischemic cardiomyopathy. As more patients survive stroke and MI, the number of patients with severe ventricular dysfunction has increased, and congestive heart failure has become epidemic. In June 2002, the FDA cleared EECP for CHF treatment.[8]

Contraindications, Safety Issues

Although EECP is considered a relatively safe method, some precautions should be followed.

The first few years after EECP was introduced, there was a great concern about patients with coexisting CHF. EECP increases preload by increasing venous return and therefore could lead to CHF worsening. Early studies excluded all patients with history of heart failure with an ejection fraction less than 35%. As EECP became more intensively investigated, the positive reports encouraged researches to include the patients with heart failure into the studies.[6] EECP is an approved method for treatment of CHF. However, the patient with any evidence of decompensation should be stabilized with medical therapy before EECP implementation.

Moderate and severe aortic decompensation is a contraindication to EECP treatment. The concern is similar to those with the use of an intraaortic balloon pump. The increased diastolic pressure may augment end-diastolic pressure and cause pulmonary congestion. Despite these concerns, many patients with moderate aortic insufficiency as with mitral insufficiency have been treated successfully. The improvement of ischemic or cardiomyopathic component may explain benefit of EECP.

At the beginning of the use of EECP, patients with atrial fibrillation or frequent ectopy that interfered with timing, pacemaker or automated internal cardioverter-defibrillators (AICDs) were excluded. According to the new EECP protocols, patients with atrial fibrillation and with a controlled ventricular response can be treated, unless the heart rate is very irregular. Patients with pacemakers or AICDs may be qualified, since this population has been benefited from EECP in previous studies. The effect of EECP on ectopy is not clear. Some studies report decrease, others increase of ectopy.[6]

Severe peripheral arterial disease, especially if the patient has ulceration or resting pain, is another contraindication to EECP treatment. Although the exact mechanism is unknown and requires more clinical trials, there is concern that reduced vascular volume and muscle mass may prevent effective counterpulsation. EECP may increase risk of thromboembolism as well. Venous diseases (phlebitis, varicose veins, prior or current vein thrombosis, stasis ulcer or pulmonary embolism) raise the concern about possibility of releasing or dislocating a clot, and the impaired blood flow raises questions about the efficacy of the treatment and remains a contraindication to EECP.

Severe hypertension (180/110 mm Hg) and aortic aneurysm or dissections are contraindications for EECP, since diastolic pressure created during the treatment may exceed safe limits. Augmentation of diastolic pressure in aortic aneurysm and dissection may be catastrophic for the patient.

Because high pressure created during inflation of the cuffs can increase the risk of hematoma, coagulopathy with INR of prothrombin time more then 2.0 is also contraindication to EECP.

Pregnancy, childbearing age, and severe chronic obstructive pulmonary disease are considered contraindications.

Compared with invasive techniques, adverse effects of EECP are limited. The most common events include leg discomfort or pain, back pain, abrasions, edema, blisters, bruising and paresthesia.[6,7,17-19] The major adverse cardiovascular events, including unstable angina, MI, exacerbation of CHF, need for emergent revascularization (CABG or PCI) and death, were studied with three-year follow-up and five-year follow-up independently.[11] The frequency of major adverse cardiovascular events was similar to those reported in pharmacologic and revascularization trials groups and seemed to be slightly higher in patients with CHF.[6,17]

Research Trends

Rapid development of EECP over the last few years brought an increasing interest in expanding of EECP applications. The new different approach to the use of EECP can change the present algorithm, which states that the method is used when all other treatments failed. Currently, EECP is considered as an adjunctive to conventional revascularization or as an alternative after medications have been tried.[5] Other future indications for EECP may include treatment of MI in the acute phase to prevent adverse remodeling and to facilitate functional recovery, treatment of ischemic and nonischemic cardiomyopathy, and secondary and primary prevention of vascular disease progression.[5,6]

Many years of Chinese experience and similar encouraging outcome of the stroke treatment in the Caribbean Islands imply another possible use of EECP in prevention and treatment of stroke.[18] Increased perfusion to the body organs, including the brain, explains more rapid and higher degree of recovery from ischemic stroke.

There is a preliminary report of EECP as effective treatment of erectile dysfunction.[20] Several studies have demonstrated with Doppler sonography increases in the flow of the internal iliac artery with significant improvement of penile rigidity after EECP treatment.

Diabetes mellitus is associated with a twofold increase in risk of CHF.21 Therefore, diabetic patients are one of the target populations in EECP research. The studies report an improvement of angina in diabetic patients either with or without CHF.[6]

Because EECP can be used in cardiogenic shock, the portable device seems to become the next generation of EECP. Mobile units could be used in emergency departments, coronary care units or catheterization labs.[22]

Ongoing Studies

A number of planned and ongoing studies of EECP investigate responses of many functions of the body to this method. These include central hemodynamics (measured in the catheterization lab); renal function; cognition; effect on the heart rate variability and silent ischemia (measured by Holter monitor); vascular reactivity; microcirculatory responses in brain, eye, kidney, skin, platelet response and coagulation; myocyte metabolic responses; and effects on baroreceptors and neurohormones. All of these studies are designed to help better understand the mechanism of action and to provide data for the more focused research.

There is special interest in a few populations of the patients: patients with MI, CHF and diabetes.

The purpose of the ongoing study of the application of EECP in MI includes testing of the feasibility of EECP to augment right ventricular performance in order to eliminate or to reduce the need of fluid loading.

Another study investigates whether EECP improves large-vessel compliance and hyperemic reflex and whether response correlates with exercise tolerance test. Prospective Evaluation of EECP in Congestive Heart Failure (PEECH) is an important multicenter study investigating conclusively the efficacy of EECP for new indication, New York Heart Association class II/III CHF, with left ventricular ejection fraction equal or less than 35%.

One of the studies of the application of EECP in patients with angina pectoris and type 2 diabetes will measure hemoglobin A1C and fructosamine in order to determine whether EECP facilitates blood glucose control. In the other study, patients with type 2 diabetes will be tested for microalbuminuria to determine the specific benefit of EECP.[23]

An Innovative Treatment

EECP is an option for patients who do not respond to pharmacologic treatment and revascularization. Although in eradicating angina pectoris, revascularization remains superior to EECP,[23] EECP offers an alternative option or an adjuvant therapy for refractory cases. Since an economic factor is involved in every medical decision, this aspect of the medical treatment should also be considered. Medicare partially reimburses EECP patients who have failed pharmacologic treatment and who are not good candidates for revascularization, amounting to a reimbursement of approximately $133 dollars per session.[24] Medicare does not reimburse EECP treatment for CHF in the absence of refractory angina.

EECP is an innovative treatment that can allow the 8 million Americans with serious CAD a safe and noninvasive treatment by creating a "natural bypass" for the heart.

Marta Bill is a PA at Tri-City Cardiology in Mesa, Ariz.

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For more on the historical development of EECP, go to www.advanceweb.com/pa.

New Therapies for Refractory CAD

It took pharmacology 20 years to introduce a new class of drugs for angina. Partial fatty acid oxidation (pFOX) inhibitors inhibit the heart's ability to burn fatty acids and increase its use of glucose.[1] Burning glucose yields extra energy for the heart and leaves fatty acids to be used as the source of energy during stress. Because of oxygen deprivation to the mitochondria during ischemia, oxidative glycolysis decreases. The excess of lactate leads to acidosis and impaired cell contractility. In this setting, fatty acids can provide a source of energy and inhibit competitively glucose and lactate uptake and glycolysis.

The study of the pFOX inhibitor ranolazine in combination with atenolol, amlodipine or diltiazem on exercise tolerance and angina frequency in patients with severe chronic angina (the CARISA study) showed an increased exercise tolerance and anti-anginal relief without adverse effects in one to two years of follow-up.[2] Ranolazine is in the advanced stages of gaining FDA approval.[3]

The American College of Cardiology/American Heart Association guidelines recommend three alternative therapies for patients with chronic stable angina who are not candidates for PCI or CABG: surgical laser transmyocardial revascularization, spinal cord stimulation and EECP.[4]

Laser transmyocardial revascularization is performed either in the operating room or by a percutaneous approach with a specialized catheter. The purpose of the treatment is to create a series of transmural endomyocardial channels to improve myocardial revascularization. The mechanism of this action is still unclear, but increased myocardial perfusion, stimulation of angiogenesis and denervation of myocardium are considered as possible effects. The technique provides symptomatic relief for end-stage chronic angina. So far, eight prospective randomized trials have showed improvement in angina symptoms, but long-term efficacy has not yet been published.[4] The procedure has not been approved by the FDA.

Spinal cord stimulation since 1987 has been proposed as a method of analgesia for patients with chronic angina pectoris refractory to other treatment. The process consists of the placement of a stimulating electrode in the dorsal epidural space at the C7-T1 level. The efficacy of the method depends on the accurate placement of the electrode. There is still insufficient data on the efficacy and long-term benefit of the procedure.[4]

Marta Bill, PA-C, PhD

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The International EECP Patient Registry

Ever since the 1960s, when the first reports of beneficial effects of counterpulsation on angina and cardiogenic shock were published, the interest in the possible application of the method has increased. Many studies on EECP had been performed. However, they came from the little centers and studied some small cohorts. It clearly appeared a few years ago that there was a need for registry of EECP treatment outcomes in order to analyze safety, efficacy, and pattern of the use of the treatment.[1,2]

The International Patient Registry (IEPR) housed at the University of Pittsburgh's Department of Epidemiology was initiated in January 1998 in the fashion similar to the NHLBI's angioplasty registry.[3] The IEPR collets data from more than 100 centers in the United States and other countries. Since the registry collects a wide spectrum of data about patients, the criteria for entry are only the informed consent for participation and at least one hour of treatment with EECP.[4] The registry collects data on patient demographics, medical history, CAD status and assessment of quality of life. After EECP, treatment data on achieved diastolic augmentation are collected (measurements from the device diastolic-to systolic ratio, peak pressure using finger plethysmography), anginal status, quality of life assessment, and adverse clinical events.[5]

Phase I of long-term study analyzed by IEPR included 5,200 consecutive patients to July 2001 with follow-up for a minimum of three years. The study showed that about 70% of the patients with refractory CAD improved at least one class in angina classification, and 80% of patients maintained the beneficial effects at one year. In order to continue investigation of the use of EECP in refractory angina, phase II of the registry will add 2,500 patients. This will allow conducting few substudies on particular populations of the patientsfor example, testing of microalbuminuria to determine EECP benefit in diabetics.[6]

Marta Bill, PA-C, PhD

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