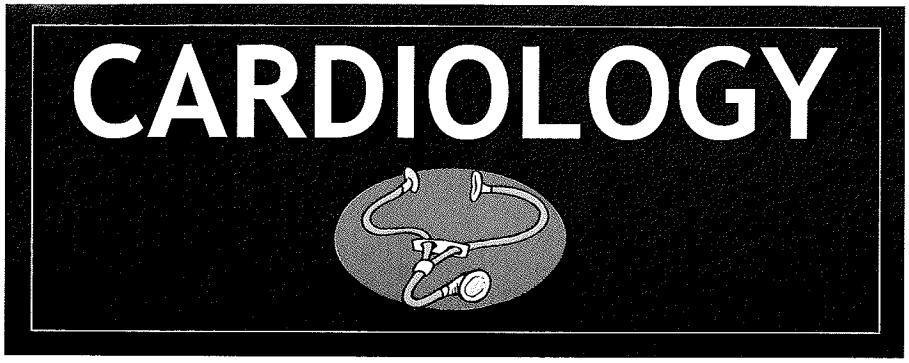

Enhanced External Counterpulsation

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The treatment of coronary atherosclerosis has changed remarkably in this century. Medical management was the sole treatment prior to advent of coronary artery bypass graft surgery in the mid 1960s. Percutaneous coronary intervention developed nearly a decade later paved the way for a minimally invasive method of coronary revascularization. Coronary stenting, rotational and directional atherectomy, and minimally invasive techniques of coronary bypass graft surgery rapidly followed in the 1980s and 90s. As a new millennium approaches, scientific study is ongoing with vascular endothelial growth factor(s) to promote growth of new vessels, and percutaneous transmyocardial laser therapy to treat patients that are either not candidates for, or refractory to, these earlier forms of revascularization. Another new form of therapy, enhanced external counterpulsation (Vasomedical Inc., Westbury, N.Y.), has emerged as a potential therapy for patients suffering from severe disabling angina. We recently cared for a patient treated with enhanced external counterpulsation with dramatic relief of angina.

Patient Presentation

History, Physical, and Laboratory Examinations

A 63-year-old male was referred with severe substernal chest discomfort, present at rest and with minimal exertion (Canadian Cardiovascular Society grade IV angina, see table 1, complete problem list). Even routine activities such as shaving, showering and walking were limited and interrupted by angina on a routine basis. He had sustained three prior myocardial infarctions and underwent two coronary artery bypass grafting procedures. He had a permanent cardiac pacemaker. His was taking aspirin, long-acting nitrates, a calcium-channel blocker and an HMG-CoA reductase inhibitor. He took 10-20 sublingual nitroglycerin tablets daily to relieve his chest discomfort. His physical and laboratory examinations were unremarkable.

Treatment

Due to diffuse coronary atherosclerosis, the patient was not a candidate for either percutaneous coronary angioplasty or further open heart surgery. He completed a 35-treatment course of enhanced external counterpulsation therapy over a seven-week period. Enhanced external counterpulsation was performed using a pneumatic cuff on the calves, thighs and buttocks (Fig 1). These cuffs were inflated sequentially from calf to thigh to buttocks with a 50-msec delay during diastole with rapid deflation of all cuffs at the beginning of systole. This increased diastolic pressure and cardiac output and decreased myocardial oxygen demand and cardiac afterload. He had no side effects from therapy and achieved appropriate levels of diastolic to systolic augmentation during treatment. He is now doing well and has very infrequent episodes of angina usually

Table 1. Complete Cardiac Diagnosis

Etiology:	Atherosclerosis	
Anatomy:	Severe three vessel coronary disease	
Physiology:	A.	Placement of a permanent pacemaker
	B.	Pre-EECP: grade IV angina
	C.	Post-EECP: grade I angina
	D.	ETT: exercised 5 minutes (Bruce protocol), 1 mm ST-segment depression
Objective:	Severely compromised	
Functional:	Mildly compromised	

occurring with only maximal exertion (Canadian Cardiovascular Society grade I angina). Long-acting nitrates have been stopped, and remarkably, he now takes only one to two sublingual nitroglycerin tablets per month. He now walks regularly and enjoys deer hunting, yard work and traveling. On the most recent treadmill exercise stress test he completed five minutes on a Bruce protocol achieving 90% of his age predicted maximal heart rate. He had 1mm ST-segment depression at peak exercise, and the test was terminated secondary to dyspnea. There was no exercise induced chest discomfort.

Discussion

Diastolic Augmentation – History and Physiology

In 1953, Kantrowitz and Kantrowitz related the augmentation of the diastolic arterial pressure to improved coronary artery flow.[1] The intra-aortic balloon pump, developed in the 1960s at the Cleveland Clinic, acutely improves coronary flow, reduces left ventricular work and oxygen demand, reduces cardiac afterload and improves coronary artery patency rates following complex coronary revascularization procedures.[2,3] The mechanism of long-term improvement seen with enhanced external counterpulsation is related possibly to increased production of vascular endothelial growth factor(s) or the recruitment of existing coronary collateral channels thereby improving coronary collateral circulation.[4,5]

Enhanced External Cardiac Counterpulsation – Initial Clinical Studies

In the early 1970s, a hydraulically activated external device improved survival in patients with cardiogenic shock.[6] Additional clinical benefit of external cardiac counterpulsation was noted in the late 1970s when mortality was reduced from 17.5% to 8.3% ($p<0.05$) in 258 patients with acute myocardial infarction treated with external counterpulsation for three hours within 24 hours of admission.[7] Since the 1980s,external counterpulsation devices have been extensively used in China to treat angina.[8]

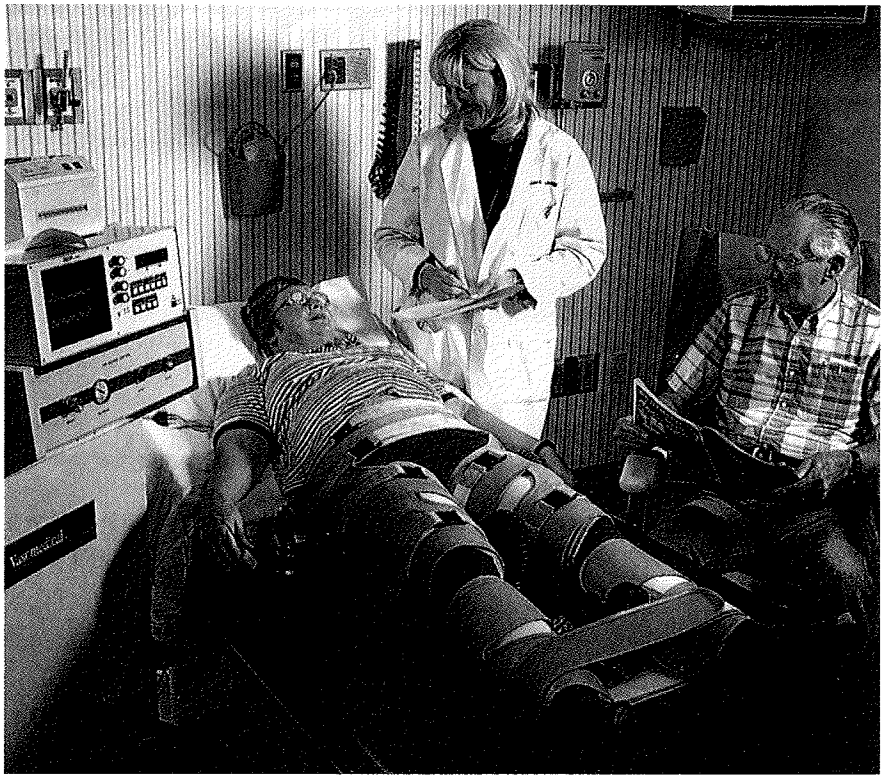

Figure 1. *Enhanced external counterpulsation is performed using a pneumatic cuff on the calves, thighs and buttocks. These cuffs are inflated sequentially from calf to thigh to buttocks with a 50-msec delay during diastole with rapid deflation of all cuffs at the beginning of systole. Diastolic blood pressure and cardiac output are increased and myocardial oxygen demand and cardiac afterload are decreased. Long-term benefit is perhaps related to an increase in coronary collateral development due to expression of vascular growth factors.*

Enhanced External Cardiac Counterpulsation – Clinical Trials in the United States

In 1995, Lawson reported his experience in 18 patients with disabling angina who received 36 one-hour treatments.[9] Three years after this initial treatment, 16 of these 18 patients were free of angina or had their doses of anti-angina medications reduced. With radionuclide testing, ischemic defects were completely resolved in 12 patients (67%), improved in two patients (11%) and were unchanged in four patients (22%). Five-year follow-up of the first 33 angina patients treated with enhanced external counterpulsation showed nearly a 90% survival rate and a 60% freedom from death or myocardial infarction.[10]

The results of the Multicenter Study of Enhanced External Counterpulsation (MUST-EECP) trial were recently reported. [11] One hundred and thirty-nine patients with severe angina were randomized to receive either hemodynamically inactive or active enhanced external counterpulsation. There was a significant improvement in time to ST-segment depression on a treadmill stress test (active group, pre-treatment 337 ±18 seconds→ post-treatment 379 ±18 seconds, $p < 0.0016$; inactive group, pre-treatment 326 ± 21 seconds→post-treatment 330 ± 20 seconds, p=NS). Duration and frequency of angina were decreased with enhanced external counterpulsation. There was a statistical insignificant trend towards less nitrate use in the active counterpulsation group compared to the inactive group. No serious complications occurred in either group. Sustained clinical benefit is seen one year following therapy.[12] Medicare now reimburses enhanced external counterpulsation therapy for treatment of patients with severe angina not amenable to percutaneous or surgical revascularization.

Conclusion

Enhanced external counterpulsation offers promise to improve the quality of life for patients with debilitating angina

not amenable to percutaneous or surgical revascularization. There is ongoing study in patients with coronary disease (including candidates for percutaneous coronary intervention or coronary artery bypass graft surgery and in those with incomplete coronary revascularization) and also those with severe peripheral vascular disease in the cerebral, renal or lower extremity vessels.

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