Can Angiographic Findings Predict Which Coronary Patients Will Benefit from Enhanced External Counterpulsation?

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Through timed, sequential pneumatic inflation of lower-extremity cuffs during diastole, enhanced external counterpulsation augments diastolic pressure, increases venous return to the heart, and decreases left ventricular afterload.[1,2] Augmenting diastolic pressure increases the coronary perfusion pressure and may facilitate the development of collaterals (and/or increase flow through existing collaterals) in patients with coronary artery disease. Most reported patients with chronic angina benefit from treatment with enhanced external counterpulsation as demonstrated by a diminution in their anginal symptoms, improvement in exercise tolerance, and a decrease in their exercise-induced reversible radionuclide perfusion defects.[3] In most of these patients benefits were maintained over a 3-year follow-up period.[4] The present study evaluates the hypothesis that a patent vascular conduit is necessary for efficacy of this therapy via transmission of the augmented diastolic perfusion pressure to the distal coronary artery.

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Fifty consecutive patients with chronic stable angina (despite medical or surgical therapy), angiographic coronary disease (>70% stenosis in a major vessel), and exercise-induced reversible radionuclide perfusion defect(s) were included in this retrospective study, which included 15 of the 18 patients reported previously.[3,4] Patients who had undergone revascularization were included if subsequent angiography showed significant residual disease (>70% stenosis in a major native coronary artery or bypass graft). Exclusion criteria included unstable angina, myocardial infarction within the prior 3 months, clinical congestive failure, significant valvular heart disease, significant arrhythmia or permanent pacemaker, nonischemic cardiomyopathy, severe peripheral vascular disease, active or recurrent thrombophlebitis, uncontrolled hypertension (>180/110 mm Hg), or bleeding diathesis.

Patients were interviewed to ascertain information about prior myocardial infarctions, revas-

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TABLE I Relation of Extent of Coronary Disease to Stress-Induced Radionuclide Defects After Enhanced External Counterpulsation

Radionuclide Deficit	Extent of Coronary Disease		
	1 Vessel	2 Vessels	3 Vessels
Resolved	14	11	4
Partial	4	6	1
No effect	1	2	7
Total	19	19	12

Chi-square analysis for 3 × 3 table: $p < 0.01$.

cularization, cardiac risk factors, and current cardiac medications. Patients were instructed to continue their usual medications as prescribed. No risk factor modifications were introduced. Radionuclide stress testing was performed to peak exercise tolerance at baseline. Patients then completed 35 hours of enhanced external counterpulsation therapy (5 hours/week for 7 weeks), followed by repeat radionuclide stress testing within 1 week of cessation of therapy to the same cardiac workload as baseline. Frequency, severity, and duration of anginal episodes and exercise tolerance were monitored throughout therapy.

Radionuclide images were evaluated by 2 independent interpreters blinded to patient identities. Images were graded as being unchanged or showing partial or complete resolution of reversible perfusion defects. Angiograms were reviewed by 2 similarly blinded observers for quantification of ejection fraction, extent, and severity of coronary disease. Patients without bypasses were classified as having 1-, 2-, or 3-vessel disease. Based on graft and native coronary artery disease, postrevascularization patients were similarly classified as having 1-, 2-, or 3-vessel residual disease.

The overall benefit of counterpulsation was examined by comparing perfusion stress results before and after therapy using a 2 × 2 chi-square test. Cardiac risk factors (hypertension, diabetes mellitus, smoking, hyperlipidemia, and family history of premature ischemic heart disease) and prior myocardial infarction were treated as bivariate and compared with radionuclide stress test results using a 3 × 3 chi-square test. Coronary artery disease (revascularized and unrevascularized) was divided into groups with 1-, 2-, and 3-vessel disease and compared with stress perfusion changes using a 3 × 3 chi-square test. Statistical significance was assumed at the $p < 0.05$ level.

There were 46 men and 4 women in the treatment group, with a mean age of 61 years (range, 45 to 75). Thirty-one patients had had ≥1 prior revascularization attempt with coronary artery bypass grafting (CABG) or percutaneous transluminal coronary angioplasty (PTCA) (6 had CABG alone, 15 had PTCA alone, and 10 had both CABG and PTCA). Nineteen patients had not had prior attempted revascularization.

All patients receiving counterpulsation therapy reported a decrease in anginal symptoms after treatment. Medications remained the same or were adjusted to a lower dose during treatment in all patients receiving counterpulsation treatment.

There was a statistically significant improvement in radionuclide stress perfusion imaging after counterpulsation therapy ($p < 0.001$). Analysis of coronary extent versus change in stress radionuclide perfusion showed a significant ($p < 0.01$) inverse relation between coronary disease extent and therapeutic benefit (Table I, Figure 1). While perfusion defects improved in 95% of patients (18 of 19) with 1-vessel coronary artery disease and in 90% of patients (17 of 19) with 2-vessel coronary artery disease, in those with 3-vessel disease improvement was seen in only 42% (5 of 12).

Cardiac risk factors, age, prior myocardial infarction, and ejection fraction variables were not significantly correlated with the response to counterpulsation therapy.

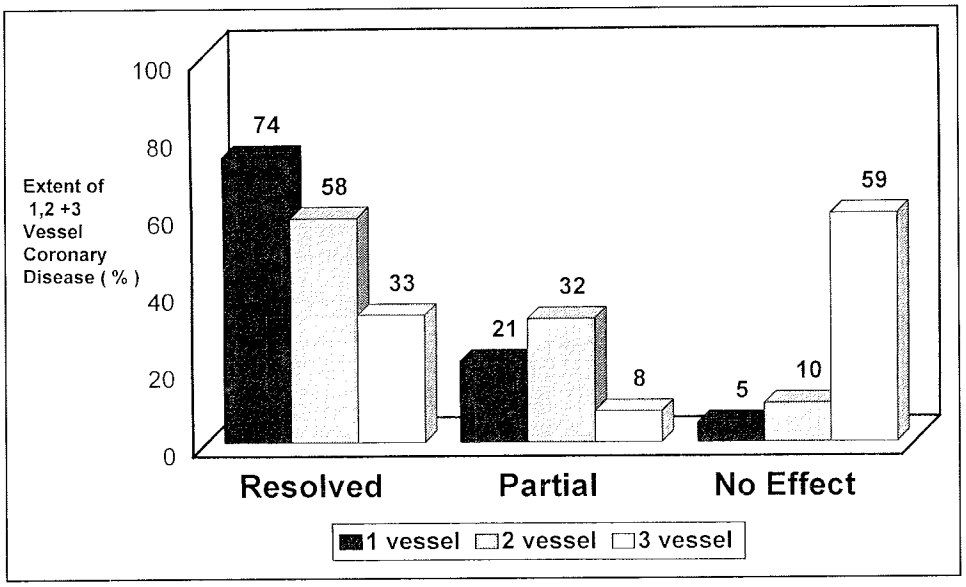

FIGURE 1. Relation of extent of coronary disease to results of radionuclide stress testing after enhanced external counterpulsation. Patients are divided into groups with 1-, 2-, and 3-vessel residual disease. Postcounterpulsation therapy stress perfusion deficits are shown as either resolved, partial improvement, or no effect compared with pretherapy baseline. The percentage and type of response is shown in relation to the extent of residual disease. Chi-square analysis, $p < 0.01$.

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Enhanced external counterpulsation is an effective therapy for patients with chronic stable angina, many of whom are otherwise refractory to medical therapy. The present study supports the hypothesis that a patent vascular conduit is a major determinant of the effectiveness of this therapy.

The hemodynamic effects of counterpulsation on arterial pressures are similar to those produced with the intraaortic balloon pump (IABP). Prior work studying the effects of intraaortic balloon pumping on coronary blood flow has shown the IABP to be effective in increasing proximal coronary blood flow velocity. Coronary blood flow velocity in the distal coronary artery in the presence of a significant proximal stenosis was diminished and was not improved despite diastolic augmentation of aortic pressures by IABP.[5] After successful PTCA, IABP resulted in significant augmentation of distal diastolic flow velocities.[6]

In the present study, patients with residual 1- and 2-vessel coronary artery disease had a significantly better response to counterpulsation treatment than those with 3-vessel coronary artery disease (the no-patent conduit group). The presence of a patent vascular conduit, whether native coronary or bypass graft, appears predictive of a favorable response to therapy. This also offers promise for patients in whom complete revascularization is not feasible. In such cases, palliative PTCA or CABG may decrease the coronary artery disease extent to residual 1- or 2-vessel disease, and it is these patient groups that demonstrate high benefit rates from counterpulsation treatment.

In summary, enhanced external counterpulsation is presumed to work by facilitating the development, or opening of coronary collaterals, or enhancing flow through existing collaterals. Improvement in radionuclide stress perfusion imaging was seen in 80% of treated patients and was inversely related to coronary disease extent. Furthermore, transmission of diastolic pressure and volume to the distal vessel appears to require a patent proximal conduit.

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