Case Studies: Enhanced External Counterpulsation

The effectiveness of EECP, a noninvasive outpatient treatment for angina pectoris, is described in this series of case reports from a community hospital. In a diverse patient population, EECP appears to provide significant relief of angina pectoris even in cases when CAD is fairly advanced and when other revascularization procedures have failed. The procedure appears most effective when at least one patent conduit, either graft or native coronary artery, can provide sufficient coronary flow to ischemic areas of myocardium. The benefits appear to be sustained over time.

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Improved technology in PTCA and CABG has made it possible to successfully treat patients with chronic angina pectoris. However, a large number of patients remain for whom these treatments continue to be ineffective or associated with an unacceptable risk of complications.

EECP augments diastolic pressure, increasing coronary blood flow and perfusion pressure with sequential pneumatic compression of the lower limbs and buttocks. Its effectiveness in reducing the symptoms of chronic angina pectoris have been demonstrated in clinical and research experience.[1,2] A second generation system (EECP-MC2), developed in China, and cleared by the FDA for marketing, has demonstrated significant efficacy in augmenting diastolic flow when compared to its earlier counterparts. This system has been in use at a community hospital since July 1995.[3] Several patients in whom EECP has provided significant clinical benefit are described here. The most noteworthy results appear to be in patients with at least one patent conduit (native coronary artery or bypass graft) that can bring blood flow to the distal ischemic myocardium. It is believed that EECP recruits collateral channels to ischemic areas of myocardium, diminishing angina and reducing or eliminating areas of poor perfusion as demonstrated on nuclear scan.

Case Reports

Case #1. A 78-year-old white female with a history of cerebrovascular accidents, which had caused partial paralysis of the right lower extremity, had persistent angina pectoris 5 years after CABG. The patient experienced 10 episodes of angina pectoris per month, each of which required up to 3 sublingual nitroglycerin tablets to relieve pain, forcing re-

ABBREVIATIONS AND ACRONYMS

CABG	Coronary artery bypass grafting
CAD	Coronary artery disease
EECP	Enhanced external counterpulsation
LAD	Left anterior descending
PTCA	Percutaneous transluminal coronary angioplasty

striction of daily activities. Despite taking long-acting nitroglycerin atenolol 50 mg, and monopril 10 mg daily, she remained a Canadian Class III angina patient. In July 1996, a cardiac catheterization revealed severe native triple-vessel CAD. The saphenous vein graft to the right coronary artery and the native right coronary artery were totally occluded. The LAD had a 90% occlusion proximally and 70% distally; the saphenous vein graft to the LAD was 100% occluded. The graft to the ramus branch and the graft to the 2 obtuse marginal branches of the 2 circumflex arteries were patent. The patient underwent successful PTCA of the 2 sequential LAD lesions with 10% residual stenosis. Despite this, angina persisted. A dipyridomole stress test with sestamibi revealed a large zone of inferior wall ischemia. In October, 1996, the patient received 35 hours of EECP. At the completion of treatment, the patient reported no need for nitroglycerin. A repeat dipyridamole stress sestamibi revealed no inferior wall ischemia.

In this case, advanced age and comorbid illness made the patient an undesirable candidate for repeat CABG. Angioplasty had failed to relieve her angina, as collateral circulation to the ischemic area at the inferior wall was insufficient. EECP may have increased vasculogenesis (enlargement of native intercoronary anastomotic channels) increasing circulation to the ischemic areas of myocardium, enhancing flow from the distal LAD to the inferior wall. In this case, EECP appeared to complement angioplasty.

Case #2. This 58-year-old white male with a cholesterol of 264, a 30 pack per year cigarette smoker, with a father who died of a myocardial infarction at age 63, developed angina pectoris and dyspnea on exertion. A stress test was prescribed. After 3 minutes of the Bruce protocol (113 bpm, 5.0 Mets) the patient developed chest pain and 3 mm horizontal ST segment depression in the inferior lateral lead. Sestamibi scintigraphy revealed a large inferolateral ischemic defect with a dilated left venticle. Cardiac catheterization in July 1995, demonstrated a long 75% proximal LAD lesion, a 75% proximal right coronary artery lesion and a 60% lesion of the left ventricular branch of the right coronary artery. The left ventricular ejection fraction was normal by ventriculogram. Despite metoprolol 25 mg, long acting nitrates and simvastatin 10 mg daily the patient continued to experience intermittent anginal episodes with minimal exertion (Canadian Class III). The recommendation of CABG was rejected.

The patient was treated with 35 hours of EECP in August 1995. Following treatment, the patient experienced significant relief from anginal attacks and was able to perform moderate exercise free of the need for sublingual nitroglycerin. The patient achieved 5 minutes and 30 seconds of a Bruce protocol before having angina (133 bpm, 7.0 Mets) associated with 2 mm horizontal ST segment depression at the end of stage 2. The angina was relieved quickly by rest, without the use of sublingual nitroglycerin. Sestamibi scintigraphy revealed a small fixed inferior basal wall defect.

EECP improved the patient's ability to perform activities of daily living and reduced the need for antianginal medications, without surgery, despite multivessel disease. After EECP a significant reduction in perfusion defect was demonstrated on repeat stress sestamibi. The presence of collaterals has been shown to shorten the time to resolution of exercise–induced ST segment depression.[4] As of April 1997, this patient has not required CABG (Fig. 1).

Case #3. This 72-year-old white male suffered an inferior wall myocardial infarction in July 1985 and underwent CABG for triple-vessel CAD in 1986. A cardiac catheterization found normal left ventricular function, a total occlusion of the right coronary artery with minimal hypokinesis of the inferior wall, a severe, long stenosis of the LAD, and stenosis of a small atrioventricular groove branch of the circum-

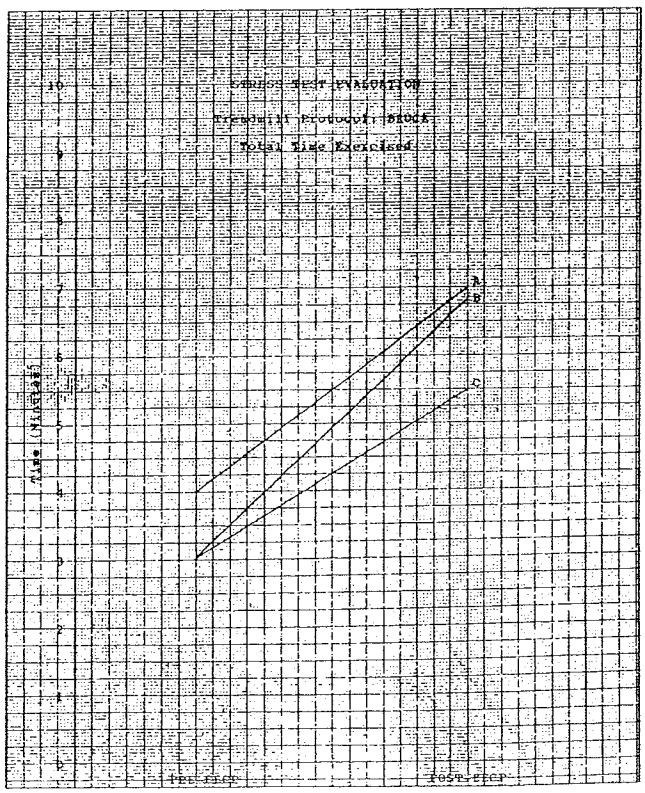

Figure 1. A-Case Study Number 3; B-Case Study Number 5; C-Case Study Number 2.

flex. The left internal mammary artery was used to graft the diagonal artery and a saphenous vein graft was placed from the aorta to the right coronary artery. Daily medications included glipizide 5 mg, simvastatin 10 mg, aspirin 325 mg, and digoxin 0.25 mg for paroxysmal atrial fibrillation. A non-insulin dependent diabetic, the patient had chronic stable angina, until January 1995, when he began to experience angina with minimal exertion, which persisted despite increased medication. A repeat cardiac catheterization was recommended and declined. The patient underwent an exercise nuclear test in November 1995; developing severe angina pectoris and 1 mm ST segment depression in the inferolateral leads after 4 minutes of the Bruce protocol. Angina was relieved with rest and one sublingual nitroglycerin. Sestamibi scintigraphy revealed an inferior wall fixed defect with a moderate zone of perinfarct ischemia. In December 1995, he received 35 hours of EECP achieving a maximum diastolic to systolic ratio of 1.8. The patient improved exercise tolerance and was able to achieve 7 minutes of a Bruce protocol before developing 1 mm ST depression in the anterior lateral leads. He did not develop chest pain, required no sublingual nitroglycerin, and the electrocardiographic abnormalities resolved in less than two minutes. Repeat sestamibi scintigraphy revealed a small area of inferior lateral wall infarction.

This case demonstrates the potential for dramatic improvement with EECP. The patient's exercise tolerance improved significantly with a sustained increase in coronary flow to the inferior wall. As of April 1997, no further intervention has been required (Fig. 1).

Case #4. A 79-year-old white female with a long history of angina pectoris, hypertension, and hyperlipidemia was being treated unsuccessfully with multiple cardiac medications for angina pectoris, including daily diltiazem 180 mg, atenolol 100 mg, lisinopril 20 mg, aspirin 81 mg, hydrochlorothiazide 25 mg, simvastatin 20 mg, and Isordil 20 mg twice daily. A dipyridamole stress test demonstrated a moderate zone of inferior posterior and inferior lateral ischemia. Cardiac catheterization in September 1996, revealed diffuse LAD disease. A 50% mid-circumflex lesion, a totally occluded right coronary artery, and an ejection fraction of 50%. The patient presented with Canadian Class IV angina having 3–5 episodes of angina pectoris daily with minimal exertion. She declined CABG, agreeing to 35 hours of EECP. At completion of 35 hours of treatment, the patient no longer required daily nitroglycerin, improving to Canadian Class II. A repeat dipyridamole stress test with sestamibi showed a small zone of inferior posterior and inferior lateral ischemia.

This case demonstrates how EECP improved this patient's quality of life without the need for CABG, which would have carried a high risk with an associated high morbidity due to her age and medical history. Even an angina patient with severe, triple-vessel disease may improve, although this group, is reported as less likely to respond to EECP (Fig. 2). [5]

Case #5. A 45-year-old female with a strong family history of CAD, hypertension, and hypercholesterolemia underwent cardiac catheterization in August 1995, after an anterior wall myocardial infarction (for which she received thrombolysis). Cardiac catheterization found evidence of a mildly impaired ventricle with an ejection fraction of 40–50% and severe hypokinesis of the anterior lateral wall. The LAD was a vessel of moderate caliber with a discrete 95% stenosis of the proximal segment. Post angioplasty of this lesion in the proximal LAD left a residual 20% obstruction; with normal distal flow. The right coronary artery, left circumflex artery, and left main coronary artery were free of disease. Following angioplasty, the patient had a rest and stress sestamibi myocardial perfusion scan, which revealed a moderate fixed anterolateral region of diminished perfusion. She was able to achieve 7 minutes of a Bruce protocol before developing 1 mm ST segment depression in the inferior lateral leads without chest pain. However, in October 1995, she began to experience angina pectoris. Stress testing revealed ST segment depression with chest pain at 3 minutes of a Bruce protocol. In November, the patient underwent repeat cardiac catheterization which revealed 100% stenosis of the mid LAD. After PCTA she was symptom free for several weeks until she again began to notice chest pain with minimal exertion (Canadian Class III). Repeat stress testing in December 1995, induced angina with 2 mm downsloping ST segment depression in the inferior lateral leads 4 minutes into the Bruce protocol. Repeat cardiac catheterization revealed a 100% mid-LAD obstruction. The distal vessel was poorly visualized, and distal flow was retrograde from a left circumflex collateral. The vessel caliber proved inadequate for a stent. After the third angioplasty, a residual 20% lesion remained in the mid LAD and distal flow was normal. The patient was maintained on Metoprolol (100 mg) and 1 aspirin daily.

Despite medical treatment, the patient remained Canadian Class III, requiring daily nitroglycerin. Rather than undergo CABG for single vessel dis-

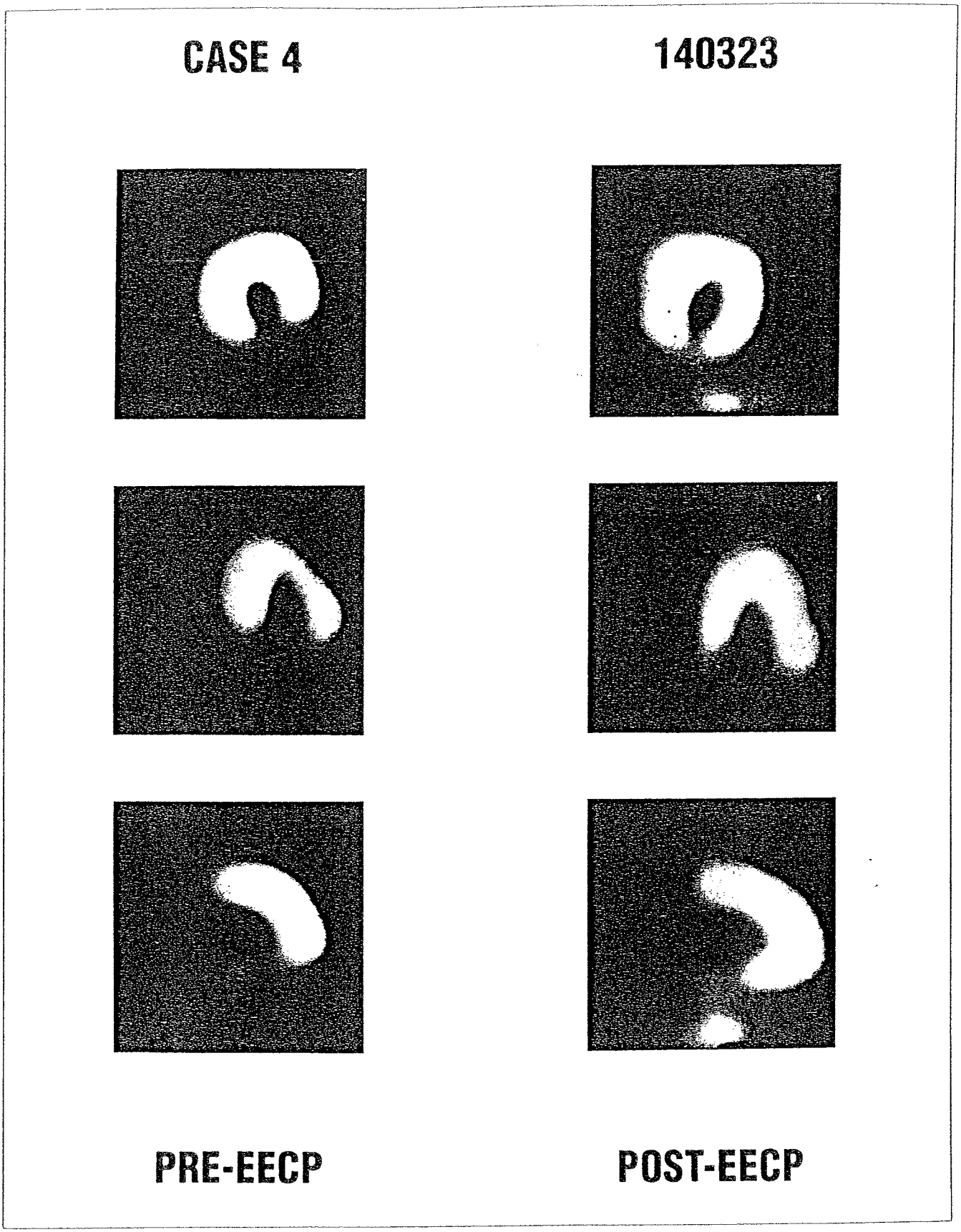

Figure 2. Case #4. Selected tomographic slices of the sestamibi dipyridamole stress images illustrating reduction in the size of the inferior posterior lateral zone of ischemia.

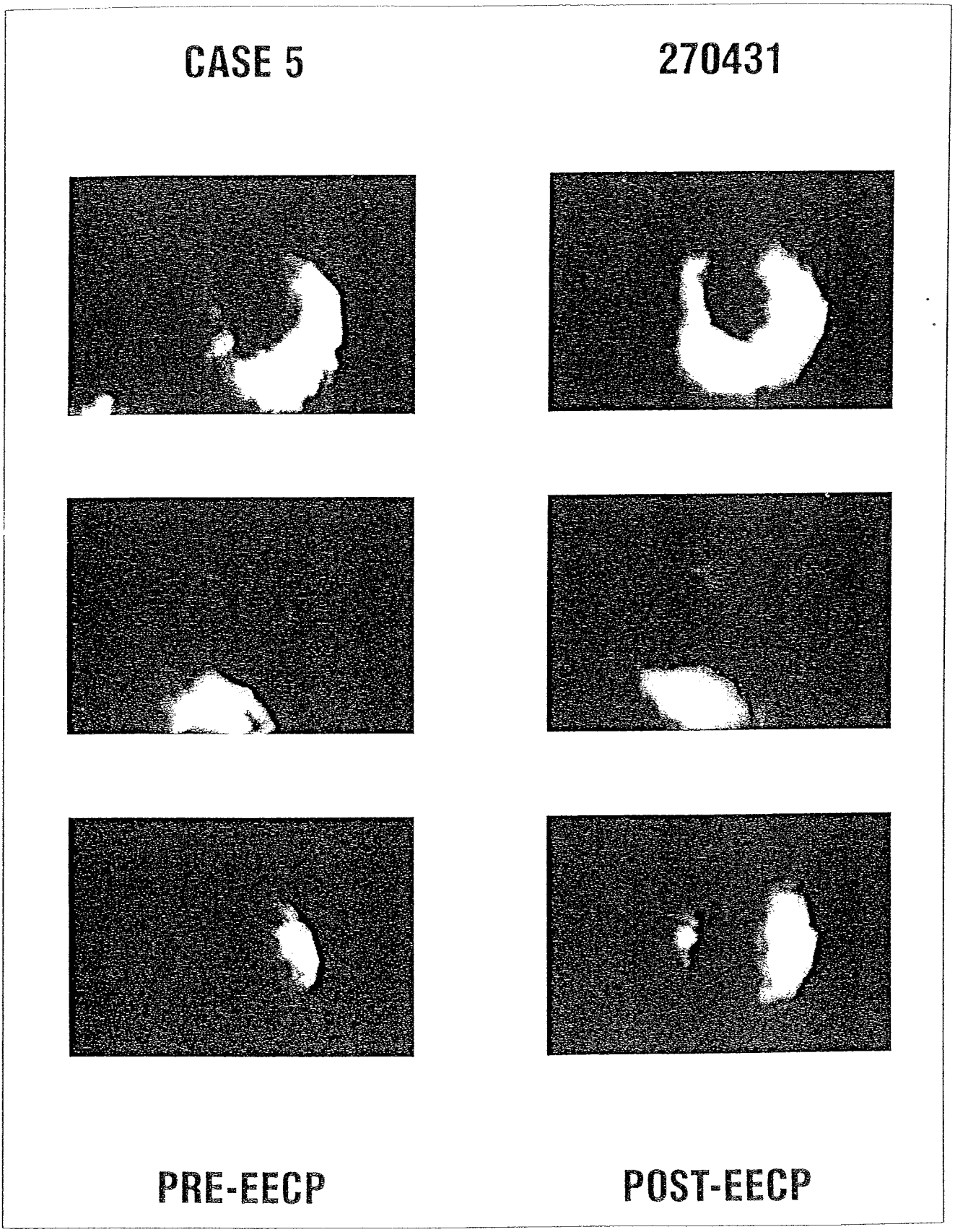

Figure 3. Case #5. Selected tomographic slices of the exercise sestamibi stress test illustrating improvement in perfusion of the anterior apical wall.

ease, the patient elected EECP. In January 1996, she received 35 hours of treatment. Following EECP, her exercise tolerance increased sufficiently to achieve 6 minutes and 50 seconds of a Bruce protocol before developing chest pain and 2 mm horizontal ST segment depression in the lateral chest leads. The chest pain and the ST segment resolved without the use of nitroglycerin. A perfusion scan revealed a moderate zone of anterior septal infarction and perinfarct ischemia. The patient had improved to Canadian Class I.

Again, in this case EECP appeared to prolong the time to ST segment depression. After treatment, angina was less pronounced and often was relieved quickly with rest. Her exercise tolerance increased enough to allow her to perform most activities without using nitroglycerin. Although the patient developed some collaterals, prior to EECP the left circumflex collateral to the distal left anterior descending artery proved inadequate. At this writing (April 1997), the patient has not required any further intervention (Fig. 1 & 3).

Conclusion

The cases reported illustrate a probable mechanism of action: the enhancement of collateral circulation to ischemic areas of myocardium. Patients without a patent graft or native vessel to transmit augmented diastolic flow to ischemic myocardium may not respond as well as those patients with at least one patent conduit. By improving proximal coronary flow, angioplasty may enhance the potential of EECP to improve collateral flow. Patients who have developed some collateral circulation may have existing collateralization enhanced with EECP (Case #5).

In triple-vessel disease, it may be difficult to predict symptomatic response to EECP solely by angiographic appearance of the coronary arteries. In Case #4, despite a 50% circumflex lesion there appeared to be adequate flow to stimulate distal collateral development with a subsequent significant reduction in angina symptoms. Direct measure of coronary flow in the catheterization laboratory may prove a useful predictor.[6]

Since the inception of the EECP program in our institution July 1995, only 2 of 17 patients failed to respond to EECP; both patients had severe, triple-vessel CAD. Our experience mirrors the study reporting that patients with severe triple vessel CAD have a response rate of approximately 42%.[7]

EECP is generally well tolerated. Of the 17 patients, only one could not complete 35 hours (due to the development of dyspnea after the first treatment). This patient was treated successfully with lasix and suffered no persistent residual symptoms.

EECP has demonstrated effectiveness as a primary therapy as well as an effective adjunct to PTCA and CABG, indicating significant potential for assuming a major role in the treatment of angina pectoris.

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