Myocardial Protection During Coronary Angioplasty With an Autoperfusion Balloon Catheter in Humans

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An autoperfusion balloon catheter was developed to allow passive myocardial perfusion during inflation through a central lumen and multiple side holes in the shaft proximal and distal to the balloon. We report its safety and efficacy in 11 patients undergoing elective angioplasty to a single coronary lesion. Each lesion was dilated three times with the autoperfusion inflation bracketed between two inflations by standard angioplasty catheters. Chest pain score, 12-lead electrocardiogram, heart rate, and mean aortic pressure were recorded before each inflation and at 1-minute intervals after inflation. Inflation duration during autoperfusion angioplasty (513 ± 303 seconds) was longer than for the pre- (107 ± 55 seconds, p = 0.0004) and post- (139 ± 71 seconds, p = 0.0006) standard dilatations. The maximum ST-segment elevation and depression in any lead during autoperfusion angioplasty (0.3 ± 0.5 and 0.6 ± 0.8 mm) was significantly less than for the pre- (2.4 ± 1.7 mm, p = 0.002 and 2.2 ± 1.3 mm, p = 0.0004) or post- (1.9 ± 1.3 mm, p = 0.002 and 1.6 ± 1.3 mm, p = 0.018) standard dilatations at the same point in time. Maximal chest pain score during autoperfusion (3.2 ± 3.5) was lower than for the pre- (6.1 ± 2.1, p = 0.003) but not the post- (5.2 ± 3.1, p = 0.07) standard angioplasty. All 11 patients underwent successful, uncomplicated procedures. We conclude that this autoperfusion catheter significantly reduces ischemic symptoms and signs during coronary angioplasty, allowing prolonged periods of balloon inflation. (Circulation 1988;78:1128–1134)

Maximum duration of balloon inflation during conventional percutaneous transluminal coronary angioplasty is usually short (30–60 seconds) because it is limited by the development of symptoms and signs of myocardial ischemia. Theoretical advantages of more prolonged inflation periods, for which there is some experimental and clinical support, include improved initial angiographic results and lower restenosis rates.1–3 Autoperfusion balloon catheters, which allow continued passive myocardial perfusion during periods of balloon inflation, provide us with the means potentially to achieve this goal. Previous studies with similar catheters have demonstrated adequate myocardial perfusion during prolonged balloon inflation in an animal model.4,5 The aim of this study was to determine the efficacy and safety of the autoperfusion balloon catheter in mitigating subjective and electrocardiographic indexes of myocardial ischemia during coronary angioplasty in humans.

Patients and Methods

The autoperfusion angioplasty system used in this study consists of a 4.5F polyethylene double lumen catheter (Stack Perfusion Catheter, Advanced Cardiovascular Systems, Mountain View, California) with a central lumen open to multiple side holes proximal and distal to the balloon portion (Figure 1). Approval for its use in human studies was obtained from the Investigational Review Board at Duke University Medical Center, Durham, North Carolina.

Patient Selection

Male and female patients undergoing elective coronary angioplasty, who gave informed consent, were included in the study if the following angio-
graphic exclusion criteria were absent: 1) ostial stenosis of 50% or greater (luminal diameter narrowing); 2) target lesion length of 1.5 cm or greater; 3) stenosis (≥50%) 2 cm or less distal to target lesion; 4) major side branch 1 cm or less proximal or distal to target lesion; 5) target lesion situated 2 cm or less proximal to a sharp angulation (≥60°); 6) target lesion greater than 5 cm from vessel orifice. In addition, patients with electrocardiographic evidence of bundle branch block, left ventricular hypertrophy, or recent transmural myocardial infarction were excluded, as were patients with significant anemia, hypoxemia, heart failure, or ongoing ischemia (angina with or without transient electrocardiographic changes) at the start of the procedure.

Protocol

Premedication before angioplasty included aspirin 325 mg daily and persantine 75 mg t.i.d. in all patients. Systemic anticoagulation was achieved with heparin 10,000 units i.v. at the beginning of each procedure. Angioplasty was performed by the Judkins femoral approach. Each lesion was dilated three times with the autoperfusion inflation bracketed between two conventional dilatations. Initial dilatation (SR1) was performed with an appropriately sized standard Simpson-Robert catheter (Advanced Cardiovascular Systems) (2.5–3.0 mm balloon diameter) inflated to 6 atm over a 0.018-in. Hi-Torque Floppy (Advanced Cardiovascular Systems) exchange guidewire. Inflation was terminated when one of the following end points was reached: 1) severe angina (chest pain score ≥7); 2) electrocardiographic or hemodynamic signs of severe ischemia (≥4 mm ST-segment elevation or depression, widened QRS complexes, hypotension <90 mm Hg); 3) significant arrhythmia (bradycardia <40, sequential ventricular premature beats or more than 10 beats/min, ventricular tachyarrhythmia). The standard balloon was then deflated and removed. A second dilatation (Perfusion balloon catheter [PBC]) was subsequently performed at the identical site with an autoperfusion catheter of equal balloon size. Immediately after correct positioning, the guidewire was withdrawn from the distal 10 cm of the catheter to allow passive perfusion of blood to the distal myocardium. Inflation (also to 6 atm) was maintained until limited by any of the above indexes of ischemia or for a period of 10 minutes. In some patients, this arbitrary target period was exceeded to determine whether even longer inflations were feasible. During autoperfusion angioplasty, 2 ml of a heparin solution (500 units/ml) was administered every 2 minutes through the central lumen. After balloon deflation, the guidewire was repositioned in the distal coronary artery, and the catheter was removed. Finally, a third dilatation (SR2) was performed after repositioning of the standard Simpson-Robert catheter. Once again, this inflation was terminated when one of the above end points was reached.

Data Acquisition and Analysis

Before, and at 1-minute intervals during, each balloon inflation, mean aortic pressure and 12-lead electrocardiogram were recorded along with a subjective grading (scale 0–10) of the degree of pain experienced by the patient. When questioned about chest pain, patients were not aware whether standard or autoperfusion dilatation was being performed. Medications that might ameliorate perception of ischemia (e.g., morphine, pethidine, nitroglycerin, or calcium channel blockers) were withheld during the study period. Repeat coronary arteriography was performed just before each balloon deflation (after electrocardiographic recording) to confirm vessel occlusion or passive perfusion. Adequacy of myocardial perfusion during autoperfusion angioplasty was judged according to the Thrombolysis in Myocardial Infarction study group’s (TIMI) grading system. TIMI grade 0 indicates no antegrade flow; grade 1 indicates penetration of contrast beyond the obstruction without filling of the distal coronary vessel; grade 2 indicates slow antegrade flow with visualization of the distal coronary artery; grade 3 indicates normal flow. After the SR2 dilatation, arteriography was again performed, and the procedure was terminated if a successful dilatation (i.e., ≤50% residual diameter narrowing) was achieved. Angiograms were reviewed by a panel of at least three experienced angiographers. Coronary luminal diameter narrowing was graded on an ordinal scale (0%, <25%, 25%, 50%, 75%, 95%, and 100%) as previously described. This method has been validated in a previous angiographic and pathological study.

Electrocardiograms were analyzed by one observer blinded to the timing of the recording and type of inflation (standard or autoperfusion) that was performed. The degree of ST-segment elevation or the J point and ST-segment depression 0.08 seconds after the J point was recorded in 0.5-mm increments (0.1 mV=1 mm) for all leads except
TABLE 1. Extent of Electrocardiographic Ischemic Changes and Duration of Inflation With Standard and Autoperfusion Angioplasty

<table>
<thead>
<tr>
<th>Patient</th>
<th>Vessel</th>
<th>ST elevation</th>
<th>ST depression</th>
<th>Inflation duration (sec)</th>
<th>Deflation reason</th>
<th>ST elevation</th>
<th>ST depression</th>
<th>Inflation duration (sec)</th>
<th>Deflation reason</th>
<th>ST elevation</th>
<th>ST depression</th>
<th>Inflation duration (sec)</th>
<th>Deflation reason</th>
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<tr>
<td>2</td>
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<td>1LV&lt;inf&gt;2&lt;/inf&gt;</td>
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<td>...</td>
<td>900 T+</td>
<td>...</td>
<td>23F</td>
<td>1LV&lt;inf&gt;2&lt;/inf&gt;</td>
<td>120 CP</td>
<td>RCA</td>
<td>23F V&lt;inf&gt;2&lt;/inf&gt;-&lt;inf&gt;3&lt;/inf&gt;</td>
</tr>
<tr>
<td>3</td>
<td>Cx</td>
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<td>60 ECG</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>300 *</td>
<td>...</td>
<td>23F</td>
<td>1LV&lt;inf&gt;2&lt;/inf&gt;</td>
<td>120 CP</td>
<td>Cx</td>
<td>23F V&lt;inf&gt;2&lt;/inf&gt;-&lt;inf&gt;3&lt;/inf&gt;</td>
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<tr>
<td>4</td>
<td>LAD</td>
<td>V&lt;inf&gt;1&lt;/inf&gt;-&lt;inf&gt;3&lt;/inf&gt;</td>
<td>23FV&lt;inf&gt;2&lt;/inf&gt;-&lt;inf&gt;6&lt;/inf&gt;</td>
<td>180 CP</td>
<td>...</td>
<td>...</td>
<td>900 T+</td>
<td>...</td>
<td>23F</td>
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<td>120 CP</td>
<td>LAD</td>
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<td>40 CP</td>
<td>...</td>
<td>...</td>
<td>120 CP</td>
<td>...</td>
<td>23FV&lt;inf&gt;2&lt;/inf&gt;-&lt;inf&gt;6&lt;/inf&gt;</td>
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<td>30 ECG</td>
<td>RCA</td>
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<td>...</td>
<td>300 VPB</td>
<td>...</td>
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<td>...</td>
<td>...</td>
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<td>600 T</td>
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<td>600 T</td>
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<td>120 CP</td>
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<td>120 CP</td>
<td>...</td>
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<td>600 T</td>
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<td>V&lt;inf&gt;1&lt;/inf&gt;-&lt;inf&gt;5&lt;/inf&gt;</td>
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<td>180 CP</td>
<td>LAD</td>
<td>V&lt;inf&gt;1&lt;/inf&gt;-&lt;inf&gt;5&lt;/inf&gt;</td>
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</tbody>
</table>

SR1, standard dilatation before PBC; PBC, autoperfusion angioplasty; SR2, standard dilatation after PBC; ST elevation or depression, standard electrocardiographic (ECG) leads showing >0.5 mm ST elevation or depression; Cx, left circumflex coronary artery; RCA, right coronary artery; LAD, left anterior descending coronary artery; CP, chest pain score ≥7; ECG, ST elevation or depression ≥4 mm; VPB, sequential or multiple ventricular premature beats; T, target inflation period (10 min); T+, extended target inflation period (15 min); *, inflation terminated before definitive end point; 1,2,3, standard ECG limb leads; L,F, augmented ECG limb leads AVL, AVF; V<inf>1</inf>-<inf>6</inf>, left ventricular ECG leads 1–6.

Results

Eleven consecutive patients (eight men, three women) with a mean age of 55 years were enrolled in the study. A single lesion was dilated in each patient, involving the right coronary artery in five, the left anterior descending coronary artery in three, and the circumflex coronary artery in three patients. Resting 12-lead electrocardiogram was normal in seven patients. Three patients had evidence of previous inferior myocardial infarction (Q waves only), and one had nonspecific T wave changes with normal ST segments. The target lesion was situated in the proximal segment of the vessel in six and in the middle segment in five patients. The mean diameter stenosis before angioplasty was 84 ± 10%.

In Table 1, the extent of electrocardiographic abnormality during balloon inflation, along with the duration and primary reason for termination of each dilatation, is listed for the 11 patients. The maximum ST elevation and depression in any lead was calculated at end inflation (just before balloon deflation) for SR1 (107 ± 55 seconds) and for PBC and SR2 at the same point in time (Table 2). The degree of electrocardiographically documented ischemia was markedly reduced during PBC inflation when compared with both standard inflations. Figure 2 illustrates the reduction in electrocardiographic ischemia in one patient during autoperfusion angioplasty. Mean maximal chest pain score was significantly lower during PBC inflation (3.2 ± 3.5) when compared with SR1 (6.1 ± 2.1, p = 0.003) but not when compared with SR2 (5.2 ± 3.1; p = 0.07).

In addition, the sum of the changes in ST elevation and depression in all leads was calculated at

Table 2. Comparison of Electrocardiographic and Clinical Data During Standard and Autoperfusion Angioplasty at the Same Point in Time

<table>
<thead>
<tr>
<th>ST elevation (mm)</th>
<th>ST depression (mm)</th>
<th>Chest pain</th>
<th>Heart rate (beats/min)</th>
<th>Mean aortic pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SR1</td>
<td>2.4 ± 1.7</td>
<td>2.2 ± 1.3</td>
<td>6.1 ± 2.1</td>
<td>70 ± 17</td>
</tr>
<tr>
<td>PBC</td>
<td>0.3 ± 0.5</td>
<td>0.6 ± 0.8</td>
<td>3.2 ± 3.5</td>
<td>71 ± 14</td>
</tr>
<tr>
<td>SR2</td>
<td>1.9 ± 1.3</td>
<td>1.6 ± 1.3</td>
<td>5.2 ± 3.1</td>
<td>76 ± 13</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

ST elevation or depression, maximum ST elevation or depression in any lead; SR1, standard dilatation before PBC; PBC, autoperfusion angioplasty; SR2, standard dilatation after PBC; NS, not significant. *p, PBC vs. SR1; †p, PBC vs. SR2.
end inflation for standard and autoperfusion angioplasty in each patient (Figure 3). Mean total ST elevation and depression was 1.6 ± 1.8 and 2.6 ± 2.9 mm at 8.5 ± 5.0 minutes for PBC, 7.0 ± 5.5 (p = 0.009) and 5.4 ± 3.5 mm (p = 0.02) at 1.7 ± 0.9 minutes for SR1, and 6.5 ± 4.6 (p = 0.006) and 4.7 ± 3.7 mm (p = 0.06) at 2.3 ± 1.1 minutes for SR2. There was a trend toward less ST change, lower chest pain score, and longer inflation times during SR2 when compared with SR1. However, this did not attain statistical significance.

Distal perfusion during PBC inflation was graded as TIMI grade 3 in seven patients and as grade 2 in three. In one patient, inadequate opacification of the proximal vessel prevented angiographic assessment of perfusion. The primary target PBC inflation period of 10 minutes was achieved in six patients, and dilatation was arbitrarily continued for a further 5 minutes in three. In five patients, PBC inflation was terminated early (three patients at 5 minutes and two at 2 minutes) because of angina (chest pain score ≥ 7) in three patients and sequential ventricular premature beats in one. In the fifth patient, dilatation was terminated at 5 minutes before a definitive end point was reached. Of the 22 standard inflations, 13 were terminated because of angina (chest pain score ≥ 7), seven because of ischemic electrocardiographic changes (≥ 4 mm ST elevation or depression) and two because of multiple ventricular premature beats. There were no sustained ventricular arrhythmias or severe hemodynamic changes during any balloon inflation. All patients underwent successful, uncomplicated angioplasty.

Discussion

Limitations of Conventional Angioplasty

The mechanism of balloon angioplasty involves a process of plaque fracture, intimal dissection, and medial stretching. Although this usually results in a widely patent lumen, it can also produce an unpredictable progressive dissection of the vessel wall leading to acute coronary occlusion. Even after successful dilatation, restenosis occurs in 30–40% of patients within 6 months. Consequently, considerable interest has been directed toward modifications of current techniques that might prevent or reduce these complications. It has been suggested that prolonged inflation periods result in improved short-term success rates and less restenosis. Theoretically, gradual balloon expansion over a prolonged period of time could cause less abrupt tearing of the plaque and vessel wall, resulting in a more stable initial condition. Alternatively, prolonged periods of vessel compression might desiccate plaque or compromise nutrient supply to the media by interruption of flow through vasa vasora, thereby inhibiting proliferation of smooth muscle cells that can lead to restenosis. Standard balloon catheters are obviously limited in their application in this situation because of the ischemia that results when blood flow is interrupted during inflation. Apart from subjective patient discomfort, previous studies have documented a profound disturbance of systolic and diastolic indexes of ventricular function during, and for up to 12 minutes after, a standard angioplasty inflation.
Functional recovery can be delayed for up to 6 hours in dogs after ischemic periods greater than 5 minutes, with repeated 5-minute episodes actually producing myocardial necrosis.

Methods of Myocardial Protection

Early attempts to prolong inflation time involved the use of pharmacological agents, particularly β-blocking drugs. Clinical and electrocardiographic indexes of myocardial ischemia were significantly diminished or delayed in onset by intravenous or intracoronary propranolol administration, though the magnitude of benefit was small. Another approach consisted of infusing oxygen-carrying perfluorocarbons, such as Fluosol-DA, through the central lumen of a balloon catheter during inflation. This method also achieved only limited success and was associated with significant ventricular arrhythmias. Lehmann and others recently reported preliminary experience with a pump-driven perfusion system with autologous whole blood. In 15 patients, active perfusion resulted in a decrease in pain and ST-segment elevation when compared with standard angioplasty. Although no complications were noted, concern was expressed about the possible adverse effects of high exit velocities, required to maintain physiological flow rates, on neighboring atherosclerotic plaque. Myocardial perfusion during coronary occlusion has also been accomplished with synchronized retrograde perfusion through the coronary sinus. This technique is of potential value during prolonged coronary dilatations, but the safety and efficacy of this approach have not yet been established.

In 1986, Erbel and others reported the first use on humans of a coronary angioplasty catheter capable of delivering blood to the distal myocardium during inflation by passive perfusion of blood through the catheter. In 11 patients, average inflation time was increased from 40 to 81 seconds until appearance of angina pectoris, whereas in three patients (30%), no improvement was noted. However, conventional angioplasty was performed before, and not after, autoperfusion angioplasty in that study, raising the possibility that preconditioning of the myocardium may have accounted for some, if not all, of the observed improvement. The term "fading ischemia" has been used to describe the prolongation of time to appearance of electrocardiographic ischemia that occurs with repeated transient coronary occlusion in humans. The mechanism underlying this phenomenon is unclear. Cohen and colleagues demonstrated that improved collateral filling occurred immediately after coronary occlusion in humans, which limited myocardial ischemia. In an animal study, Murry and others found that preconditioning the myocardium with multiple brief ischemic episodes protected the heart from a subsequent sustained ischemic insult, and they proposed that slowing of adenosine 5'-triphosphate depletion and limitation of catabolite accumulation were possible involved mechanisms.

Our observation that ST changes were less pronounced (though not significantly) during the second standard inflation than during the first is consistent with the concept of fading ischemia. Although this effect may have exaggerated the improvement noted during autoperfusion angioplasty, the marked difference between autoperfusion inflation and the subsequent standard inflation precludes it as a major contributing factor. All of our patients tolerated at least a 100% increase (mean, 327%; range, 100–900%) in inflation time during autoperfusion angioplasty over both standard balloon dilatations, with a
significant reduction in both extent and severity of ischemic ST change. Electrocardiographic changes have been shown to be useful and practical indexes of the extent of myocardium at risk during coronary occlusion. Cohen and others reported a close correlation between the degree of ST-segment elevation and extent of left ventricular asynergy during coronary angioplasty. More recently, Aldrich and colleagues developed formulas, based on quantitative measurements of ST change on the admission electrocardiogram, predictive of final QRS-estimated infarct size. Although antegrade flow was documented arteriographically during autoperfusion angioplasty, chest pain or electrocardiographic changes did occur in seven patients, indicating inadequate oxygen supply. In four of these, small to moderate-sized side branches in close proximity to the target lesion were occluded during the autoperfusion inflation and may have contributed to the ischemic changes observed.

Limitations

The primary aim of this study was to demonstrate the ability of this catheter to allow passive perfusion of blood during inflation. Our study design did not permit a definitive evaluation of its performance with respect to tracking characteristics or ability to traverse a lesion compared with a standard Simpson-Robert catheter. The deflated balloon profile is 18% larger than a corresponding standard catheter (0.065 vs. 0.055 in. for a 3.0-mm balloon), perhaps posing a difficulty with severe stenoses. Further studies are underway to address this aspect.

Current design specifications place additional restraints on the applicability of this catheter for routine angioplasty at present as evidenced by the angiographic exclusion criteria for this study. Vessels with tandem lesions, or target lesions in close proximity to a severe angulation, are best avoided because of the possibility of trauma from the catheter tip. Major side branch occlusion by the balloon portion might reduce the potential efficacy of the catheter but is not a contraindication to its use, although necessarily so for the purposes of this study. Severe systemic hypotension might also limit effective perfusion, although this is less likely to occur as a consequence of autoperfusion rather than conventional angioplasty, and care should be taken to maintain adequate systemic pressure by judicious use of intravenous fluids and strict attention to pharmacological therapy, particularly in relation to nitroglycerin administration. The balloon length is slightly shorter than a standard balloon (1.8 vs. 2.0 cm), making correct positioning somewhat more difficult and reducing the range of lesion length that may be attempted. Withdrawing and repositioning the guidewire are potentially hazardous; however, these have not caused problems in our experience. Further design modifications (lower profile, longer balloon length, etc.) are currently being made to alleviate some of these limitations.

Acknowledgments

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References


**KEY WORDS** • transient ischemia • prolonged dilatation
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