Restoration of Normal Coronary Hemodynamics and Myocardial Metabolism after Percutaneous Transluminal Coronary Angioplasty

DAVID O. WILLIAMS, M.D., RAYMON S. RILEY, M.D., ARUN K. SINGH, M.D., AND ALBERT S. MOST, M.D.

SUMMARY  Regional coronary blood flow and myocardial metabolism were evaluated in a patient who underwent percutaneous transluminal coronary angioplasty (PTCA). Angioplasty increased coronary luminal diameter and reduced trans-stenotic gradient. Before PTCA, angina pectoris developed during sustained rapid atrial pacing and was associated with abnormal lactate metabolism and a mild increase in coronary flow and myocardial oxygen consumption. After PTCA, angina was absent during pacing and lactate extraction was preserved. Coronary flow and oxygen consumption were increased to a greater degree than before PTCA. The temporal response of changes in coronary blood flow due to an abrupt increase in heart rate was also evaluated. Flow reached peak value more rapidly after PTCA. These observations suggest that PTCA may result in improved regional coronary blood flow and restoration of normal flow regulatory mechanisms and myocardial metabolism.

PERCUTANEOUS transluminal coronary angioplasty (PTCA) has been successfully performed in patients with symptomatic coronary artery disease. The goal of this innovative technique is to improve myocardial blood flow by reducing the extent of localized atherosclerotic coronary narrowing. The efficacy of this procedure has been assessed by symptomatic response, changes in degree of angiographically visualized coronary stenosis, trans-stenotic pressure gradient, thallium scintigraphy and electrocardiographic response to exercise. No information is available, however, regarding the effects of coronary angioplasty on regional coronary blood flow or myocardial metabolism. In this report, we describe a patient who underwent successful PTCA in whom a coronary hemodynamic evaluation was performed immediately before and after PTCA.

Case Report and Methods

A 57-year-old white male retailer was referred because of the development of progressive and severe angina pectoris over the prior 2 months. Coronary angiography demonstrated an 85% narrowing of the proximal left anterior descending before the origin of the first diagonal and septal branches. He had no evidence of recent or prior myocardial infarction. PTCA was offered as a therapeutic alternative. The patient elected to participate in a prospective investigation of the procedure. Written informed consent was obtained. In accordance with our protocol, the patient also agreed to undergo emergency coronary bypass surgery if required.

Immediately before PTCA, a #20 cannula was placed in the left brachial artery for pressure monitoring and blood sampling. A similar cannula was inserted into a left antecubital vein for i.v. fluid and drug administration. Leads II and V_a of the ECG were monitored. A thermodilution flow catheter (Wilton Webster Laboratories) was inserted into a right antecubital vein and advanced into the great cardiac vein. Radiopaque contrast was injected into the great cardiac vein to determine the location of major veins and permit proper catheter position for sampling drainage of the anterior descending vein. Atrial pacing was initiated at 10 beats/min faster that the resting heart rate. Great cardiac vein and systemic arterial blood were also obtained for oxygen content and lactate concentration. Then the pacing rate was increased 20 beats/min every 2 minutes until angina pectoris of moderate severity developed. After 2 minutes of angina, great cardiac vein flow and blood sampling were repeated and pacing was stopped.

Ten minutes later the time course of coronary flow response to abrupt increase in oxygen demand was evaluated. Atrial pacing was resumed (90 beats/min) for 5 minutes. Great cardiac vein flow was then recorded. After a steady-state flow recording was obtained, the atrial rate was abruptly increased to 130 beats/min and great cardiac vein flow was continuously recorded for 30 more seconds.

PTCA was performed using a polyurethane Teflon guiding catheter and a Grünzig DG-20-30 dilating catheter (USCI Cardiology and Radiology Products). Initially the left coronary artery system was visualized by injection of radiopaque contrast through the guiding catheter. This was accomplished before and after sublingual nitroglycerin in order to exclude spasm. A #7F pacing catheter, placed in the pulmonary artery, served as a reference for the location of the stenosis (fig. 1). The dilating catheter was then advanced down the left anterior descending coronary artery. Vigorous coughing was required for the catheter to cross the stenosis. In crossing the stenosis, mean pressure...
recorded at the catheter tip fell 62 mm Hg (table 1). After the first balloon inflation (4 atmospheres) and deflation, mean intracoronary pressure distal to the stenosis increased 20 mm Hg. After a second inflation (6 atmospheres) and deflation, the trans-stenotic gradient on pull-back was 15 mm Hg. The dilating catheter was then removed and angiography repeated using the guiding catheter. No adverse reactions were noted during the procedure.

After PTCA, regional coronary flow and myocardial metabolic studies were repeated in a fashion similar to that before the dilatation. At this time, typical angina pectoris was not present during rapid atrial pacing at the heart rate that previously had resulted in angina. Sixty-two minutes elapsed between nitroglycerin administration and repeat coronary flow and metabolic measurements, longer than hemodynamic effects (18 minutes) resulting from nitroglycerin.

Results

Angiography

Before PTCA, the extent of left anterior descending coronary artery stenosis was quantitated in two views with an optical comparator and a 10-mm reticle. The extent of reduction in luminal diameter, obtained from an average of the two views, was 86% (fig. 1A). Immediately after PTCA, the extent of narrowing was reduced to 17% (fig. 1B). There was no evidence of intimal tearing or arterial dissection.

Regional Coronary Blood Flow

Great cardiac vein blood flow was initially measured at 84 beats/min (table 2). Rate-pressure (peak systolic) product at this time was \(13.02 \times 10^8\) mm Hg/min. Great cardiac vein flow was 148 ml/min and regional myocardial oxygen consumption was 20 ml O$_2$/min. Angina pectoris occurred at a paced rate of 144 beats/min, with a rate-pressure product of \(22.32 \times 10^8\) mm Hg/min. At this time, great cardiac vein flow had increased to 191 ml/min, with oxygen consumption of 23.7 ml O$_2$/min. Immediately after PTCA, at 84 beats/min, great cardiac vein flow was 146 ml/min and the rate-pressure product 13.44 mm Hg/min. Myocardial oxygen consumption was 18.0 ml O$_2$/min. During atrial pacing at 144 beats/min, myocardial blood flow increased to 223 ml/min and oxygen consumption to 26.3 ml O$_2$/min.

The time course of coronary blood flow response during an abrupt increase in myocardial oxygen demand was also evaluated (fig. 2). Before PTCA, there was a 15-second delay before coronary blood flow reached peak value after an abrupt increase in atrial pacing rate. After PTCA, peak flow was achieved in 10 seconds.

Myocardial Metabolism

Myocardial lactate extraction in the distribution of the left anterior descending artery was 22% during atrial pacing at a rate of 84 beats/min before PTCA (fig. 3). During pacing-induced angina, lactate production was observed (−2.4% extraction). Immediately after PTCA, again at a rate of 84 beats/min, regional lactate extraction was again observed (30.3% extraction). During rapid atrial pacing, lactate production did not occur; lactate extraction remained 28.8%.

![Figure 1](http://circ.ahajournals.org/) Single frames taken from left coronary cineangiogram before (A) and after (B) coronary angioplasty. Arrows designate site of dilatation.

| Table 1. Intracoronary Pressure and Degree of Stenosis |
|---------------------------------|---------------------------------|------------------|
|                   | Intracoronary pressure (mm Hg) | Degree of stenosis (%) |
|                   | Proximal  | Distal  | Gradient |
| Before dilatation | 112       | 50      | 62       | 86        |
| After dilatation  | 120       | 105     | 15       | 17        |
TABLE 2. Regional Coronary Blood Flow and Myocardial Metabolism

<table>
<thead>
<tr>
<th></th>
<th>Pre-PTCA</th>
<th></th>
<th>Post-PTCA</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Angina</td>
<td>% change</td>
<td>Rest</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>84</td>
<td>144</td>
<td>4</td>
<td>84</td>
</tr>
<tr>
<td>Rate-pressure product (× 10^6 mm Hg/min)</td>
<td>13.02</td>
<td>22.32</td>
<td>70</td>
<td>13.44</td>
</tr>
<tr>
<td>GCV flow (ml/min)</td>
<td>148</td>
<td>191</td>
<td>29</td>
<td>146</td>
</tr>
<tr>
<td>Oxygen consumption (ml O2/min)</td>
<td>20.0</td>
<td>23.7</td>
<td>19</td>
<td>18.0</td>
</tr>
<tr>
<td>Lactate extraction (%)</td>
<td>22.0</td>
<td>-2.4</td>
<td>-24.4</td>
<td>30.3</td>
</tr>
</tbody>
</table>

Abbreviations: PTCA = percutaneous transluminal coronary angioplasty; GCV = great cardiac vein.

Discussion

Grüntzig and colleagues offer PTCA as an alternative therapy for the treatment of symptomatic coronary artery disease. Their investigations have demonstrated that successful application of the procedure can result in an increase in coronary artery diameter, a reduction in trans-stenotic pressure gradient, an increase in myocardial perfusion as assessed by thallium scintigraphy and improved electrocardiographic and symptomatic response to upright bicycle exercise. The present investigation extends the objective evaluation of PTCA to measurement of regional coronary blood flow and myocardial metabolism.

Regional coronary blood flow was determined in our patient by thermodilution. The catheter was positioned to quantitate regional coronary flow to myocardium perfused by the stenosed left anterior descending coronary artery. Before dilatation at a heart rate of 84 beats/min, great cardiac vein flow was within normal limits. With the development of pacing-induced angina, regional coronary flow and myocardial oxygen consumption increased. Abnormal lactate metabolism indicated that the myocardium was ischemic. The increase in regional coronary blood flow before PTCA was considered subnormal because after PTCA, at a similar rate-pressure product, flow was substantially greater. Thus, successful PTCA in this patient was associated with an enhanced capacity to increase coronary blood flow and myocardial oxygen consumption. Flow increase may have resulted from the combined influence of a reduction in flow-limiting stenosis and a concomitant enhancement of systolic contractile function.

The possible effect of nitroglycerin on these observations deserves comment. Nitroglycerin was administered sublingually after pre-PTCA metabolic and blood flow results were obtained and thus could have potentially influenced post-PTCA values. When considering the time elapsed from the administration of nitroglycerin to the performance of the post-PTCA

![Figure 2](image2.png)

**Figure 2.** Time course of great cardiac vein flow response to an abrupt increase in heart rate. Flow is expressed as percent change from control heart Pre = before angioplasty; Post = after angioplasty.

![Figure 3](image3.png)

**Figure 3.** Effect of rapid atrial pacing on lactate extraction before (Pre) and after (Post) coronary angioplasty. Values below broken line indicate lactate production.
evaluation, however, such a consideration is most unlikely. The duration of hemodynamic effects after nitroglycerin was 18 minutes, whereas the post-PTCA evaluation was performed 62 minutes after nitroglycerin administration. Thus, the repeat metabolic and blood flow results obtained after PTCA were remote from the hemodynamic effects of nitroglycerin.

A previous study has raised the possibility that withdrawal of resting α-adrenergic tone from the coronary circulation contributes to the rapidity of myocardial blood flow increase during stress. Patients with denervated hearts are reported to have a slower-than-normal blood flow response to a sudden increase in heart rate. Moreover, it is postulated that patients with coronary artery disease may have chronically reduced α-adrenergic tone in the basal state as a compensation for flow-limiting stenosis. These patients might then be expected to behave much as patients with denervated hearts, increasing coronary blood flow more slowly than normal in response to stress. The PTCA procedure offered a unique opportunity to examine this interesting thesis in the patient, with (pre-PTCA) and without (post-PTCA) a significant coronary stenosis.

Before PTCA, peak flow after the initiation of pacing was achieved in 15 seconds, whereas after successful dilatation 10 seconds elapsed before peak flow was achieved in response to the same heart rate challenge. These results are consonant with the thesis that there is a delayed peak flow response in the presence of a significant coronary stenosis. Because of the multiple potential factors that could influence the time to peak coronary blood flow, additional studies will be necessary to substantiate this initial observation in man.

In summary, we have evaluated the effect of PTCA on regional coronary blood flow and myocardial metabolism. Successful PTCA was associated with an enhancement of coronary blood flow and oxygen consumption during rapid atrial pacing. Furthermore, abnormalities in lactate metabolism were reversed, suggesting a shift from anaerobic to aerobic metabolism. The temporal responsiveness of the coronary circulation was also assessed and results suggest that normal coronary regulatory controls may be restored after release of a significant coronary stenosis.

Acknowledgment

The authors acknowledge the assistance of Karen Jessop, John Larney, Christine Abatiello, Barbara Roberts, M.D., and the staff of the Cardiovascular and Cardiac Research Laboratories.

References

Restoration of normal coronary hemodynamics and myocardial metabolism after percutaneous transluminal coronary angioplasty.
D O Williams, R S Riley, A K Singh and A S Most

Circulation. 1980;62:653-656
doi: 10.1161/01.CIR.62.3.653

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1980 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/62/3/653

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/