Value of the intracoronary electrocardiogram to monitor myocardial ischemia during percutaneous transluminal coronary angioplasty

PETER L. FRIEDMAN, M.D., PH.D., THOMAS L. SHOOK, M.D., JAMES M. KIRSHENBAUM, M.D., ANDREW P. SELWYN, M.D., AND PETER GANZ, M.D.

ABSTRACT To enhance detection of ischemia during percutaneous transluminal coronary angioplasty (PTCA), unipolar intracoronary electrocardiograms (ECGs) were recorded during PTCA in 25 patients from the tips of guidewires positioned distal to stenoses being dilated. Surface electrocardiographic leads chosen to reflect likely areas of reversible ischemia during PTCA were recorded simultaneously. In 21 of 29 stenoses dilated (72%), ST segment elevation and/or T wave peaking in intracoronary ECG appeared during balloon inflation and disappeared after deflation, accompanied by transient angina on 19 occasions. Two patients had transient ST segment elevation in intracoronary ECGs during PTCA without associated angina. ST changes in the surface ECG during PTCA were seen on only nine occasions (31%), always accompanied by ST segment elevation in the intracoronary ECG that appeared earlier and was of much greater magnitude. Five patients with prior myocardial infarction and aneurysm formation had fixed ST segment elevation in the intracoronary ECG unrelated to balloon inflation. Myocardial ischemia during PTCA can be detected easily with intracoronary ECGs and with greater sensitivity than that of the surface ECG. Furthermore, intracoronary ECGs may help to clarify the nature of chest pain during balloon inflation or during suspected complications.


MYOCARDIAL ISCHEMIA is frequently provoked during percutaneous transluminal coronary angioplasty (PTCA). In most cases ischemia is only transient, appearing after advancement of the angioplasty catheter across a critical stenosis or during balloon inflation and disappearing promptly upon balloon deflation. Occasionally, when angioplasty results in occlusion of a vessel or intimal dissection that compromises antegrade blood flow, ischemia may be long lasting. Traditionally, the presence or absence of angina and the surface electrocardiogram (ECG) have been relied upon to monitor myocardial ischemia during PTCA. Such an approach is not entirely satisfactory. Patients may not experience angina, even though profound myocardial ischemia is present. Alternatively, chest pain during PTCA may not be caused by ischemia. The surface ECG is also an unreliable index of ischemia. Ischemia in some regions of the heart may not be reflected by ST segment or T wave changes in the particular leads being monitored during PTCA or may be present in an area of myocardium too small to be reflected in any of the standard surface leads. Con- versely, previous myocardial infarction and aneurysm formation may cause fixed ST segment and T wave abnormalities that can mimic or obscure changes caused by transient ischemia.

Acute ischemia in myocardial cells leads to a decline in resting membrane potential and abbreviation of action potential duration. One result of these abnormalities is the generation of current flow between ischemic and nonischemic regions. It is this current flow that leads to deviation of the ST segment in the ECG. Of note, the magnitude of such “currents of injury” is influenced greatly by the distance from the recording electrode to the region of ischemia. Thus an electrode on the body surface distant from a region of ischemia would be expected to record less ST segment deviation than an electrode placed on the surface of the heart, near the ischemic area. In the present study, local unipolar intracoronary ECGs were recorded during PTCA directly from guidewires positioned across coronary stenoses to be dilated. By virtue of the proximity of the guidewires to regions of potential ische-
mia, it was thought that intracoronary ECGs recorded in this fashion might prove to be more sensitive and reliable indicators of transient myocardial ischemia than the standard ECG recorded from the body surface.

**Methods**

Twenty-five patients undergoing routine PTCA at the Brigham and Women’s Hospital had local unipolar intracoronary ECGs recorded during the course of their procedures. Before catheterization each patient had given informed written consent in accordance with the guidelines of the Human Subjects Committee of the hospital. The methods for routine PTCA in our laboratory have been described in detail previously. We ordinarily use a sequential balloon technique, the initial dilation being performed with an angioplasty balloon catheter of small diameter (2.0 to 2.5 mm) that is passed over a 0.014 inch diameter or 0.018 inch diameter steerable guidewire (Hi-Torque Floppy; Advanced Cardiovascular Systems, Inc., Mountain View, CA). The shafts of such guidewires are coated with Teflon, leaving only the distal 30 cm uninsulated. After dilation with the initial balloon catheter, the guidewire is withdrawn and replaced by a longer exchange guidewire. The initial balloon catheter is then exchanged for one or more balloon catheters of larger diameter until a satisfactory result has been achieved. In the present study, all 25 patients underwent PTCA by this standard technique via a femoral approach. Multiple standard surface electrocardiographic leads chosen to reflect likely areas of reversible myocardial ischemia during balloon inflation were filtered between 0.1 to 100 or 0.1 to 500 Hz and monitored continuously throughout the procedure. Except for one patient, at least two limb leads and one precordial lead were monitored in every patient. In one patient (subject 20, table 1) three limb leads were monitored without a precordial lead. During PTCA of the left anterior descending coronary artery, leads I and aVL were always monitored along with one or more of the left precordial leads (V3, V4, V5, or V6). For PTCA of the left circumflex artery, the limb leads most frequently chosen were II and III, although in some instances I or aVF were monitored in place of one of the inferior leads. V5 or V6 were also monitored in each of these patients. At least two of the three inferior limb leads (II, III, or aVF) along with V3 or V6 were monitored during PTCA of the right coronary artery. After passage of the guidewire through a target stenosis and then advancement of the balloon catheter across the stenosis, the guidewire was withdrawn slightly, taking care to ensure that only a short (1 to 2 cm) distal segment of guidewire protruded beyond the lumen of the balloon catheter. A local intracoronary unipolar ECG from myocardium distal to the stenosis to be dilated was then ob-

**TABLE 1**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>PTCA vessel</th>
<th>Monitored leads</th>
<th>Prior MI</th>
<th>Angina</th>
<th>ECGΔ</th>
<th>IC-ECGΔ</th>
<th>Collaterals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>41</td>
<td>Distal RCA</td>
<td>II, aVF, V5</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Proximal RCA</td>
<td>II, aVF, V5</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>54</td>
<td>LAD</td>
<td>I, aVL, V6</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>49</td>
<td>LAD</td>
<td>I, aVL, V6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>53</td>
<td>LAD</td>
<td>I, aVL, V5</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>50</td>
<td>LAD</td>
<td>I, aVL, V5</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>43</td>
<td>LAD</td>
<td>I, aVL, V3</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>LAD diagonal</td>
<td>I, aVL, V5</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>62</td>
<td>LCX</td>
<td>II, III, V6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>49</td>
<td>LAD</td>
<td>I, aVL, V6</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>66</td>
<td>LAD</td>
<td>I, aVL, V6</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>M</td>
<td>61</td>
<td>LCX</td>
<td>II, III, V6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>63</td>
<td>LCX</td>
<td>II, III, aVL, V6</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>60</td>
<td>LAD</td>
<td>I, aVL, V6</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>52</td>
<td>Distal LAD</td>
<td>I, aVL, V4</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Proximal LAD</td>
<td>I, aVL, V4</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>77</td>
<td>LCX</td>
<td>I, aVL, V6</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>15</td>
<td>M</td>
<td>51</td>
<td>LAD</td>
<td>I, aVL, V6</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>69</td>
<td>LAD</td>
<td>I, aVL, V3, V6</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>F</td>
<td>68</td>
<td>LAD</td>
<td>I, II, V6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>18</td>
<td>F</td>
<td>78</td>
<td>LCX</td>
<td>II, III, V6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>19</td>
<td>F</td>
<td>64</td>
<td>LCX</td>
<td>II, III, V6</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>20</td>
<td>M</td>
<td>40</td>
<td>RCA</td>
<td>II, III, aVF</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>21</td>
<td>F</td>
<td>61</td>
<td>LAD</td>
<td>I, aVL, V5</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>59</td>
<td>LAD</td>
<td>I, aVL, V5</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>23</td>
<td>M</td>
<td>60</td>
<td>RCA</td>
<td>III, aVF, V3</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>24</td>
<td>M</td>
<td>57</td>
<td>LCX</td>
<td>II, aVL, V5</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>25</td>
<td>M</td>
<td>42</td>
<td>LAD</td>
<td>I, aVL, V3</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

RCA = right coronary artery; LAD = left anterior descending coronary artery; LCX = left circumflex coronary artery; ECGΔ = reversible ST segment or T wave abnormalities in surface leads during balloon inflation; IC-ECGΔ = reversible ST segment or T wave abnormalities in intracoronary ECG during balloon inflation.
tained by connecting the proximal end of the guidewire as it exited from the balloon catheter to a precordial lead of a standard surface ECG cable by means of a sterile double-alligator connector. Wilson's central terminal was used as an indifferent electrode. The unipolar intracoronary ECG was filtered between 0.1 to 100 or 0.1 to 500 Hz and then displayed simultaneously with the standard surface leads being monitored. Surface and intracoronary ECGs were recorded simultaneously by means of a photographic multichannel oscillographic recorder (Electronics for Medicine VR16, Pleasantville, NY) at paper speeds of 10, 25, and 50 mm/sec. Initial recordings were made as soon as the balloon catheter was positioned across the target stenosis. Recordings were then made continuously during initial balloon inflation and deflation as well as during subsequent inflation and deflations. Throughout the procedure all patients were questioned repeatedly about presence or absence of chest pain; these responses were duly recorded along with the surface and intracoronary ECGs.

Results

A profile of the clinical and angiographic features of the 25 patients from whom local intracoronary unipolar ECGs were recorded during PTCA are listed in table 1. There were 18 men and seven women, ranging in age from 40 to 78 years (average 57). PTCA in a single coronary artery was performed in 23 of the 25 patients. In two of these patients (subjects 1 and 13, table 1) two separate stenoses in the same artery were dilated. Two patients underwent PTCA of two separate vessels (subjects 6 and 25, table 1). Thus 29 separate stenoses were dilated. Since local intracoronary ECGs were recorded with each of these dilations, 29 such recordings were available for analysis. Of the 29 stenoses being subjected to PTCA, collateral flow to distal portions of the vessel being dilated was present in 12 (table 1). For the purposes of this study, collateral flow was considered to be present when the majority of the vessel being treated by PTCA had been at least faintly visualized angiographically during the patient's initial diagnostic arteriogram.

In the first several patients from whom unipolar intracoronary ECGs were recorded during PTCA, an effort was made to characterize the ability of such ECGs to represent local electrical events. After engagement of a coronary ostium with the guiding catheter, the balloon catheter was advanced inside the guiding catheter until it was in the aortic root near the coronary ostium. If a guidewire was then advanced inside the balloon catheter so that it extended nearly to but not beyond the tip of the balloon catheter, unipolar ECGs recorded from the wire in this position were low-amplitude signals in which the QRS and T waves were obscured by high-frequency electrical noise (figure 1, left). Although the guidewire in such a position was actually much closer to the left ventricle than a precordial lead recorded from the body surface, the latter lead yielded a signal that was free of noise and of greater amplitude (figure 1, left). In contrast, if the balloon catheter was advanced into the coronary artery and the wire was advanced so that a short segment of wire protruded beyond the distal lumen of the balloon catheter, unipolar ECGs recorded from the wire were virtually free of noise and much greater in amplitude, far exceeding the amplitude of limb and precordial leads recorded from the body surface (figure 1, right).

Effect of PTCA on the intracoronary ECG. The most common pattern of response of the local intracoronary unipolar ECG observed during PTCA is illustrated in figure 2. Before advancement of the guidewire and balloon catheter across a left anterior descending coro-

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Surface leads I, aVL, and V₅ as well as the unipolar intracoronary electrocardiogram (IC-ECG) recorded during PTCA in subject 6 (table 1). Left, The IC-ECG was recorded from a guidewire inside a balloon catheter positioned near but not in the left coronary artery ostium. Right, IC-ECG signal recorded from the guidewire after advancement across a stenosis in the left anterior descending coronary artery (LAD). Square-wave calibrations = 1.0 mV; time lines = 1.0 sec.
nary artery stenosis, this patient’s surface ECG revealed only minor nonspecific ST segment abnormalities with upright T waves in leads I, aVL, and V₅. After advancement of the guidewire and balloon catheter across the stenosis but before balloon inflation, these surface leads were unchanged (figure 2, top left). The intracoronary ECG recorded from the guidewire tip beyond the stenosis before balloon inflation was characterized by an isoelectric ST segment and a biphasic T wave of low amplitude (figure 2, top left). Several seconds after balloon inflation, ST segment elevation appeared in the intracoronary ECG, accompanied by the onset of chest pain (figure 2, top right). At this time the ST segments and T waves were unchanged in the standard surface leads (figure 2, top right). After 40 sec of balloon inflation there was ST segment elevation of 2 mV in the intracoronary ECG. Only a trace of ST segment elevation was present in aVL at this time, and leads I and V₅ remained unchanged (figure 2, bottom left). After balloon deflation, ST segment elevation in the intracoronary ECG and chest pain disappeared within seconds (figure 2, bottom right). This pattern of transient ST segment elevation in local intracoronary ECGs appearing with balloon inflation and disappearing after balloon deflation was observed during PTCA of 20 of the 29 stenoses that were dilated. In these 20 instances, the range of ST segment elevation in the intracoronary ECG was between 0.5 and 8.0 mV. During dilation of one additional stenosis, transient peaking of the T wave in the intracoronary ECG without ST segment elevation was noted after balloon inflation and disappeared upon deflation. Thus transient changes in the intracoronary ECG occurred in 21 of the 29 stenoses dilated (72%). In none of these instances were ventricular arrhythmias provoked during balloon inflation. Of the 29 stenoses dilated, transient ST segment or T wave abnormalities in the surface ECG leads being monitored appeared in only nine instances (31%). Eight of these nine demonstrated transient ST segment elevation in one or more of the surface leads being monitored, whereas one episode of transient ST segment depression was noted. All nine instances in
FIGURE 3. Surface leads II, III, and V₆ and intracoronary electrocardiogram (IC-ECG) during PTCA of left circumflex artery stenosis (subject 7, table 1). Records were obtained after 40 sec of balloon inflation. Note marked ST segment elevation in the IC-ECG and less striking ST segment elevation in surface leads. Square-wave calibrations = 1.0 mV; time lines = 1.0 sec.

which the surface leads demonstrated transient ST segment abnormalities during balloon inflation also revealed transient marked ST segment elevation in the local intracoronary ECG. In all nine of these instances, ST segment abnormalities appeared earlier and were of much greater magnitude in the intracoronary ECG compared with the surface leads (figure 3). We never observed transient ST segment and T wave abnormalities in the surface ECG during PTCA unaccompanied by ST segment elevation in the intracoronary ECG. The greater sensitivity of intracoronary ECGs for detection of local ischemia as compared with the surface ECG was real, not apparent, since surface ECGs were always recorded at much higher gain than the intracoronary ECGs (figure 3), and yet the magnitude of ST segment elevation was always much greater in the latter.

Of the 29 stenoses subjected to PTCA, dilation was not associated with any new transient ST segment or T wave abnormalities in either the surface ECG or the local intracoronary ECG on eight occasions. One such instance was encountered in subject 25 (table 1) during PTCA of a right coronary artery stenosis. This patient had no history of prior myocardial infarction. The surface ECG showed evidence only of left ventricular hypertrophy with minor nonspecific ST segment and T wave abnormalities, and left ventriculography revealed normal segmental wall motion throughout. During balloon inflation in the right coronary artery the patient developed mild angina, although neither the surface nor the local intracoronary ECG revealed any changes compared with baseline. In view of the appearance of ischemic chest pain during balloon inflation, the absence of ST segment elevation in the local intracoronary ECG in this patient was thought to represent a false-negative result. Subjects 1 and 6 (table 1), individuals with stenoses of the right coronary artery and a diagonal branch of the left anterior descending coronary artery, respectively, and no evidence of prior infarction, also failed to develop any ST segment or T wave abnormalities in the intracoronary ECG during balloon inflation. These patients, however, also failed to develop chest pain during balloon inflation. Of note, both patients had extensive collateral flow to the distal portions of the vessels being subjected to PTCA and therefore may have failed to develop significant ischemia during balloon inflation. The remaining five patients in whom balloon inflation was not associated with transient ST segment abnormalities in the intracoronary ECG all had angiographic evidence of prior infarction in the distribution of the artery being subjected to PTCA (subjects 2, 13, 15, 20, and 23, table 1). These patients typically also had electrocardiographic evidence of prior infarction, including Q waves and ST segment elevation suggestive of aneurysm formation that were apparent in both the surface ECG and the intracoronary ECG at rest (figure 4, left). These abnormalities remained unchanged during balloon inflation (figure 4, right). Of note, none of these patients developed chest pain during balloon inflation.

During PTCA of the 21 stenoses associated with transient ST segment or T wave abnormalities in the local intracoronary ECG, chest pain developed after balloon inflation on 19 occasions. In these cases, ST segment elevation in the intracoronary ECG preceded onset of chest pain by several seconds or occurred simultaneously with the onset of chest pain. After balloon deflation, local intracoronary ST segment elevation and chest pain subsided together within several seconds in 15 patients. In four patients, however, intracoronary ST segment elevation and chest pain persisted after balloon deflation, resolving only after withdrawal of the balloon catheter back from the stenosis (see below, figure 7). Two patients with transient ST segment elevation in the intracoronary ECG during balloon inflation failed to develop chest pain (patients 9 and 16, table 1). Conversely, one patient (patient 25) developed chest pain during balloon inflation that subsided after balloon deflation, despite absence of any transient ST segment or T wave abnormalities in either the surface ECG or the local intracoronary ECG. The presence or absence of angiographically demonstrable collateral blood flow to arterial segments beyond the stenosis being dilated did not appear to influence whether chest pain or intracoronary ST segment elevation occurred during balloon inflation.

Diagnostic utility of the intracoronary ECG. On several
occasions, monitoring ST segment abnormalities in
the local intracoronary ECG during PTCA yielded use-
ful diagnostic information that would not have been
apparent from inspection of the surface ECG alone.
Subject 8 (table 1), a 49-year-old man who underwent
PTCA of a proximal left anterior descending artery
stenosis, was noted to have ST segment elevation in
the surface ECG that suggested ischemia at the start of
his angioplasty, even though chest pain was absent at
that time (figure 5, left). The intracoronary ECG be-
fore balloon inflation revealed no ST segment eleva-
tion (figure 5, left). After balloon inflation, chest pain
and ST segment elevation in the intracoronary ECG
appeared; these were not associated with any addition-
al ST segment changes in the surface ECG (figure 5, middle). Although ST segment elevation in the surface
ECG persisted after balloon deflation, the disappear-
ance of chest pain and resolution of local intracoronary
ST segment elevation (figure 5, right) suggested that
the ST segment abnormalities in the surface ECG rep-
resented normal early repolarization rather than isch-
emia.

Subject 4 (table 1) was another individual in whom
the local intracoronary ECG provided information that
helped to guide the course of his angioplasty. This
patient, a 53-year-old man undergoing PTCA of a ste-

FIGURE 4. Surface leads I, aVL, and V₄ and intracoronary electrocardiogram (IC-ECG) recorded after advancement of
guidewire and balloon catheter across a left anterior descending coronary artery stenosis before balloon inflation (left) and after
balloon inflation (right) in a patient with prior anterior myocardial infarction (subject 13, table 1). Q waves and ST segment
elevation suggestive of aneurysm were present in V₄ and in the IC-ECG before inflation and were unchanged during inflation.
Square-wave calibrations = 1.0 mV; time lines = 1.0 sec.

FIGURE 5. Surface leads I, aVL, and V₆ and intracoronary electrocardiogram (IC-ECG) recorded during PTCA of a left anterior
descending coronary artery stenosis in subject 8 (table 1). Left, After advancement of guidewire and balloon catheter across the
stenosis but before inflation. Middle, During balloon inflation. Right, After balloon deflation. See text for description. Square-
wave calibrations = 1.0 mV; time lines = 1.0 sec.
nosis in the left anterior descending artery, had flat T waves in surface lead aVL and nonspecific ST segment depression of less than 0.1 mV in surface leads I and V6 before balloon inflation (figure 6, top left). The intracoronary ECG before balloon inflation revealed no ST segment deviation with upright T waves (figure 6, top left). During inflation with a 2 mm balloon, chest pain along with ST segment elevation of 0.7 mV amplitude appeared in the intracoronary ECG. ST segment elevation in the surface ECG at this time was apparent only in aVL and was only 0.09 mV in amplitude (figure 6, top middle). After balloon deflation, the balloon was pulled back from the stenosis, leaving the wire across the stenosis. Injection of radiographic contrast medium revealed what appeared to be an acceptable angiographic result. However, the patient continued to complain of chest pain, and inspection of the intracoronary ECG revealed local T wave inversion that had not been present before balloon inflation (figure 6, top right). Of interest, the surface ECG at this time was unchanged compared with the surface ECG before balloon inflation (figure 6, top right). Chest pain and T wave inversion in the intracoronary ECG were still present several minutes later (figure 6, bottom left). Accordingly, the stenosis was dilated a second time with a balloon of larger diameter (figure 6, bottom middle). After this larger balloon was pulled back, both the patient’s chest pain and local intracoronary T wave inversions resolved completely (figure 6, bottom right).

As mentioned above, in four patients (subjects 1, 7, 12, and 17, table 1), chest pain and local intracoronary ST segment elevation that had been absent before balloon inflation (figure 7, top left) appeared during balloon inflation but failed to resolve after balloon deflation (figure 7, top right). As illustrated in figure 7, in two of these patients inspection of the surface ECG alone after balloon deflation would not have suggested persistent ischemia as the cause of chest pain. Because of persistent ST segment elevation in the local intracoronary ECG, however, ischemia was suspected. When the balloon catheter was withdrawn in each of these

![Figure 6](http://circ.ahajournals.org/)

**FIGURE 6.** Surface leads I, aVL, and V6 and intracoronary electrocardiogram (IC-ECG) recorded during PTCA of a left anterior descending coronary artery stenosis (subject 4, table 1). *Top left,* After advancement of the guidewire and a 2.0 mm diameter balloon catheter across the stenosis, before balloon inflation. Note upright T waves in the IC-ECG. *Top middle,* During balloon inflation. *Top right,* After balloon deflation and withdrawal of balloon catheter back into the guiding catheter, leaving the guidewire across the stenosis. Note return of surface leads to baseline configuration but appearance of new T wave inversion in the IC-ECG. *Bottom left,* Three minutes after withdrawal of 2.0 mm diameter balloon catheter, angina and T wave inversion in the IC-ECG were still present. *Bottom middle,* During inflation of 3.0 mm diameter balloon catheter advanced across stenosis. *Bottom right,* After balloon deflation and pullback of balloon catheter into guiding catheter. Note resolution of angina and return of T wave morphology to normal in the IC-ECG. Square-wave calibrations = 1.0 mV; time lines = 1.0 sec.
four cases, chest pain and local intracoronary ST segment elevation resolved completely (figure 7, bottom), suggesting that the presence of the balloon had been responsible for compromised blood flow in the dilated artery rather than residual stenosis or intimal dissection.

Discussion

In the present study, Teflon-coated guidewires advanced across coronary stenoses being subjected to PTCA were used as intracoronary leads to record local unipolar ECGs in the hope that such leads might be sensitive indicators of local ischemia. Coronary stenoses that are suitable targets for PTCA typically are located in the large epicardial segments of the coronary arterial tree. Thus signals recorded from guidewires positioned across such stenoses should represent local epicardial electrograms.

Previous studies in normal canine hearts have characterized in detail the relationships that exist among intracellular action potentials of endocardial and epicardial fibers, extracelluar electrograms recorded from these regions, and the currents that flow between these regions during inscription of the ST segment and T wave as recorded from the body surface. Action potential duration of epicardial cells typically is less than that of endocardial cells, a characteristic that underlies the normal sequence of repolarization from epicardium to endocardium. Because of these differences in action potential duration, current flow between endocardium and epicardium during repolarization results in inscription of an upright T wave in electrograms recorded from the epicardium. In contrast, significant differences in membrane potential between endocardial and epicardial cells are not normally present during phases 4 and 2 of the action potential; accordingly current flow between these two regions is negligible during the ST and TQ segments. In the presence of acute ischemia, resting membrane potential declines in cells within the ischemic region and action potential duration of such cells shortens because of abbreviation of both the plateau phase as well as phase 3. This results in abnormal current flow between ischemic and the surrounding nonischemic myocardium during the ST and TQ segments. By convention, the direction of current flow is that in which positive charges move. Thus one would predict a positive deflection or elevation of the ST segment in any lead recorded directly from the region of ischemia. In contrast, leads distant from the region of ischemia would reflect events principally from nonischemic regions and thus would inscribe ST segment depression.

Our results suggest that intracoronary ECGs recorded from guidewires during PTCA do in fact represent local electrograms. As predicted for epicardial leads recorded within regions of ischemia, the intracoronary ECG revealed transient ST segment elevation rather than depression during balloon inflation. In most instances this transient ST segment elevation was accompanied by the onset of angina, further supporting the conclusion that ischemia was the cause of the local
ST segment elevation. Although other independent indexes of ischemia such as myocardial lactate production were not monitored during the study, it seems likely that ischemia was present during balloon inflation in most instances.

Further evidence that the unipolar intracoronary ECG reflects predominantly local electrical events arising from myocardium near the guidewire tip was the observation that ischemic ST segment elevation in such leads after balloon inflation was of much greater magnitude than that in standard leads recorded from the body surface. Indeed, ST segment elevation often was obvious in the intracoronary ECG at times when the surface ECG leads were unchanged. Previous studies of potentials recorded from the epicardium and from the body surface of chronically instrumented closed-chest chimpanzees have demonstrated a marked decline in the magnitude of recorded potentials with increasing distance between the heart and the recording electrode. The differences in the magnitude of ST segment elevation in surface and intracoronary leads noted in this study are therefore as one would predict from prior animal studies. In our study, all 12 standard surface electrocardiographic leads were not monitored during PTCA. It is possible that in some patients ST segment abnormalities during balloon inflation might have been present in one or more surface leads that were not being monitored and thus may have escaped detection. On the other hand, an attempt was made to monitor at least the three surface leads thought most likely to reflect regions of transient ischemia. In those patients whose surface ECGs failed to reveal evidence of transient ischemia during balloon inflation, one would have expected to see reversible ST segment or T wave abnormalities in at least one of the surface leads being monitored.

Although the unipolar intracoronary ECG appears to be more sensitive than the standard surface ECG for detecting ischemia, it may not be reliable in all circumstances. In this study, one patient undergoing PTCA of an artery supplying noninfarcted myocardium developed typical angina during balloon inflation that disappeared after balloon deflation, and yet failed to demonstrate evidence of transient ischemia in the intracoronary ECG. Of note, the surface ECG in this patient also failed to reveal transient ischemia. The explanation for this apparently false-negative result is uncertain. If a guidewire were positioned in a branch of an artery that was receiving collateral blood flow whereas other branches of the artery were inadequately supplied by collaterals, one might still expect to elicit angina during balloon inflation, but without accompa-


Value of the intracoronary electrocardiogram to monitor myocardial ischemia during percutaneous transluminal coronary angioplasty.
P L Friedman, T L Shook, J M Kirshenbaum, A P Selwyn and P Ganz

doi: 10.1161/01.CIR.74.2.330

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 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/74/2/330

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/