Acute occlusion after percutaneous transluminal coronary angioplasty — a new approach

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ABSTRACT  Between July 1980 and November 1982, there were 935 coronary angioplasties attempted at Emory University Hospital. Of these patients, 20 developed acute occlusion. Of these 20, 19 presented within 3 hr of surgery or within 3 hr after stopping a continuous heparin infusion. Five patients required emergency surgery, but in 15 nitrates, nifedipine, and/or repeat angioplasty reopened the artery and the patient could be stabilized on continuous infusions of heparin and nitroglycerin. In only one case was an occluding thrombus evident on angiographic examination. The mechanism of acute occlusion is unknown, but coronary artery spasm may play a role.

Circulation 68, No. 4, 725-732, 1983.

EMERGENCY BYPASS SURGERY was required after percutaneous transluminal coronary angioplasty (PTCA) in about 5% to 6% of patients entered into the National Registry of the National Heart, Lung and Blood Institute (NHLBI).1, 2 The two principal reasons for emergency bypass surgery are acute occlusion and coronary artery dissection.3 Coronary artery dissection with compromise of blood flow occurs either as a result of subintimal passage of the balloon catheter4 or as a direct complication of the balloon injury process that can, by itself, result in intimal tearing.4 Intra-aortic balloon counterpulsation followed by emergency coronary bypass surgery5 might minimize myocardial injury in the PTCA patient who has coronary artery dissection. At present, no effective treatment short of these measures is available to treat the patient with coronary dissection.

Acute occlusion after otherwise unremarkable PTCA was the most common reason given in the NHLBI registry for emergency bypass surgery.1 Earlier it had been recommended that all patients with acute occlusion undergo emergency bypass surgery.6 Unlike in the subset of patients with coronary dissection, we no longer perform emergency bypass surgery in all our patients with acute occlusion.

The purpose of this report is to review the treatment, hospital course, complications, and follow-up data from 20 consecutive patients with acute coronary occlusion.

Methods

All patients undergoing angioplasty at Emory University Hospital between July 1981 and November 1982 were examined for evidence of acute occlusion after a successful angioplasty. To be included, patients had to have chest pain after angioplasty and either angiographic evidence of complete occlusion at the angioplasty site or ST segment elevation of at least 2 mm in the electrocardiographic lead corresponding to the dilated vessel. In addition, the initial angiographic and pressure-gradient response must have been judged to be satisfactory, i.e., the diameter narrowing must have been reduced by at least 20% and the pressure gradient across the stenosis correspondingly reduced. Twenty of 935 patients met these criteria during this period. The basic method of angioplasty has been described previously.5, 7

The following is a list of certain details germane to the treatment of acute occlusion.

(1) All patients receive 10,000 units of heparin intravenously as soon as both the pacing catheter and guiding catheter are in place. An additional 5000 units of heparin is given 1 hr later if angioplasty is not completed by that time.

(2) Heparin is never neutralized by the administration of protamine sulfate. Arterial and venous sheaths are left in the puncture site for 2 to 3 hr after angioplasty. (No major local arterial problems have been observed when the sheaths have been removed by 4 hr after angioplasty.)

(3) Nitroglycerin, 0.4 mg, and nifedipine, 10 mg, are administered sublingually at the beginning of the procedure.

(4) Intracoronary nitroglycerin, 100 to 200 mg/ml, is administered just before crossing the lesion. This is repeated when the operator believes it necessary.

(5) After angioplasty all patients without contraindications are placed on sublingual and dermal nitrates and oral nifedipine. This medication is continued as tolerated as long as the patient is hospitalized — usually for about 2 days after the angioplasty.
<table>
<thead>
<tr>
<th>EUH study patient No.</th>
<th>Sex</th>
<th>Date</th>
<th>Patient No. (this study)</th>
<th>Vessel dilated</th>
<th>% Diameter narrowing Before After</th>
<th>Pressure gradient Before After</th>
<th>Time from PTCA to occlusion (hr)</th>
<th>Documentation of occlusion</th>
<th>Predisposing factors to occlusion</th>
<th>Treatment of patients</th>
<th>Emerg. CABG</th>
</tr>
</thead>
<tbody>
<tr>
<td>394</td>
<td>M</td>
<td>7/6</td>
<td>1</td>
<td>LAD</td>
<td>100</td>
<td>1</td>
<td>41</td>
<td>10</td>
<td>56* ECG; CA</td>
<td>Change from heparin to coumadin</td>
<td>No</td>
</tr>
<tr>
<td>434</td>
<td>F</td>
<td>8/1</td>
<td>2</td>
<td>LAD</td>
<td>80</td>
<td>20</td>
<td>54</td>
<td>21</td>
<td>72* CA</td>
<td>Repeat PTCA no change</td>
<td>No</td>
</tr>
<tr>
<td>492</td>
<td>M</td>
<td>9/15</td>
<td>3</td>
<td>LAD</td>
<td>35</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>&lt;1 ECG; CA</td>
<td>Repeat PTCA good (without resolution)</td>
<td>Yes</td>
</tr>
<tr>
<td>398</td>
<td>M</td>
<td>9/16</td>
<td>4</td>
<td>LAD</td>
<td>76</td>
<td>29</td>
<td>63</td>
<td>35</td>
<td>20 ECG; CA Intimal tear, d/c heparin 2 hr earlier</td>
<td>Repeat PTCA (without dissection)</td>
<td>Yes</td>
</tr>
<tr>
<td>523</td>
<td>M</td>
<td>10/12</td>
<td>5</td>
<td>LAD</td>
<td>61</td>
<td>26</td>
<td>39</td>
<td>6</td>
<td>&lt;1 ECG; CA Intimal tear</td>
<td>IC NTG followed by PTCA; IC NTG</td>
<td>No</td>
</tr>
<tr>
<td>541</td>
<td>M</td>
<td>11/4</td>
<td>6</td>
<td>Cx</td>
<td>67</td>
<td>29</td>
<td>49</td>
<td>&lt;1 CA Intimal tear</td>
<td>IC NTG; without transient relief; streptokinase</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>576</td>
<td>M</td>
<td>11/18</td>
<td>8</td>
<td>LAD</td>
<td>73</td>
<td>20</td>
<td>54</td>
<td>8</td>
<td>2 CA None seen</td>
<td>Attempted PTCA; IC NTG; no response</td>
<td>Yes</td>
</tr>
<tr>
<td>741</td>
<td>M</td>
<td>3/9</td>
<td>9</td>
<td>RCA</td>
<td>99</td>
<td>43</td>
<td>51</td>
<td>9</td>
<td>24* CA Intimal tear</td>
<td>Repeat PTCA (without resolution)</td>
<td>No</td>
</tr>
<tr>
<td>746</td>
<td>F</td>
<td>3/11</td>
<td>10</td>
<td>LAD</td>
<td>74</td>
<td>13</td>
<td>55</td>
<td>1</td>
<td>&lt;1 ECG; CA Hypotension; intimal tear</td>
<td>Repeat PTCA (without resolution)</td>
<td>No</td>
</tr>
</tbody>
</table>

*Actual time of occlusion is not known. Time indicated is time from PTCA to discovery of total occlusion by ECG or coronary angiography.

EUH = Emory University Hospital; CA = coronary angiography; LV = left ventricular; Cx = circumflex; neg = negative; EF = ejection fraction; mod = moderate; ETT = exercise treadmill testing; wnl = within normal limits; CABG = coronary artery bypass graft; cath = catheterization; NTG = nitroglycerin; MI = myocardial infarction; CK = creatine kinase; IC = intracoronary.

(6) All patients receive 0.65 aspirin orally the night before angioplasty and 0.325 g daily for 6 months after angioplasty.

Patients at high risk of developing acute occlusion after angioplasty (i.e., patients with large intimal tears, or patients with chest pain after PTCA) are monitored in the coronary care unit for at least 24 hr after PTCA. They are placed on continuous heparin infusions (600 to 1000 units/hr) sufficient to maintain the partial thromboplastin time at 2 to 2½ times control. We also treat such high-risk patients with continuous intravenous infusions of nitroglycerin rather than relying on dermal or sublingual preparations of long-acting nitrates. These intravenous...
infusions are generally tapered and discontinued at 12 to 24 hr if the patient shows no sign or symptoms of ischemia. Myocardial infarction by Q wave criteria was based on the appearance of new Q waves in any lead. Differences between groups were analyzed with the chi-square test.

Results

The clinical data from the 20 study patients are summarized in tables 1A and 1B. A typical case is illustrated in figure 1. Of these 20 patients, 11 (55%) had left anterior descending artery (LAD) lesions, six (30%) had right coronary artery (RCA) lesions, and three (15%) had circumflex artery lesions. During this period, 64% of the total number of patients were treated for LAD lesions, 26% were treated for RCA lesions, and 10% were treated for circumflex artery lesions. In 16 patients, acute occlusion was demonstrated with coronary angiography and in 11 it was suggested by electrocardiographically measured ECG ST segment elevation of more than 2 mm. Seven patients met both criteria. In 16 patients the onset of symptoms occurred after they left the cardiac catheterization laboratory. In these 20 patients the mean diameter narrowing was reduced from 74 ± 15% to 23 ± 11% and the mean pressure gradient was reduced from 52 ± 15 to 11 ± 8 mm Hg. Four of five patients undergoing surgery had electrographic (ECG) evidence of infarction. Two of these 20 patients (Nos. 4 and 14) were undergoing repeat angioplasty for recurrent stenosis.

Fourteen of these 20 patients demonstrated acute occlusion within 3 hr of an otherwise successful coronary angioplasty. Of the other six patients, three (Nos. 1, 5, and 17) developed acute occlusion within 3 hr after being weaned from continuous heparin infusions. Patients 2 and 9 had angina less than 2 hr after PTCA, but initial normal (unchanged) ECGs led to false-negative diagnoses. From a second ECG recorded the following morning a diagnosis of infarction was possible in patient 2, but in patient 9, who had good collateral vessels, a repeat catheterization performed to investigate patency before a second vessel PTCA was necessary to obtain the diagnosis.

The only patient who demonstrated onset of inclusion criteria later than 3 hr after stopping heparin was patient 16. This patient experienced a vagal reaction associated with hypotension and bradycardia followed by chest pain at 16 hr after PTCA. This was also the only patient who fulfilled angiographic criteria for coronary thrombosis.

The most prominent predisposing factor to acute occlusion is the presence of an intimal tear. This is manifested angiographically as a double line present on the angiogram. This angiographic finding was present in 14 of these 20 patients (70%). Three patients had vagal reactions, hypotension, and bradycardia just before they presented with chest pain and ST segment elevation. One patient had undergone an ergonovine provocative test (total dose 0.35 mg) just before successful angioplasty.

Treatment. In three patients the chest pain and ST segment elevations resolved without invasive therapy.

TABLE 1A

(Continued)

<table>
<thead>
<tr>
<th>Q wave MI</th>
<th>Peak CK</th>
<th>Follow-up data</th>
</tr>
</thead>
<tbody>
<tr>
<td>No 499</td>
<td>+</td>
<td>Reoccluded on 10/7/81 by cath; underwent bypass surgery 10/26/81</td>
</tr>
<tr>
<td>Yes 850</td>
<td>+</td>
<td>11/8/81, minimal luminal irregularities by cath; 5/82, LV wnl except slight apical dyskinesia; 11/10/81, thallium ETT, wnl</td>
</tr>
<tr>
<td>Yes 1827</td>
<td>+</td>
<td>10/27/81, neg ETT; 6/17/82, asymptomatic, cath at 8 days EF 48% mild anterior, mod apical hypokinesia</td>
</tr>
<tr>
<td>Yes 1749</td>
<td>+</td>
<td>2/4/82, bypass graft stenosis, PTCA of bypass graft stenosis; 8/6/82, closed LAD, patent bypass graft and good result from graft angioplasty; EF = 62% mod apical, septal hypokinesia</td>
</tr>
<tr>
<td>No 707</td>
<td>+</td>
<td>No chest pain</td>
</tr>
<tr>
<td>No 1266</td>
<td>+</td>
<td>Stress test positive 4/12/82; 2 mm ST depression and chest pain; refused angiogram</td>
</tr>
<tr>
<td>Yes 2730</td>
<td>+</td>
<td>3/25/82, EF 53% at 4 months; no wall motion abnormality at rest</td>
</tr>
<tr>
<td>No 102</td>
<td>+</td>
<td>Restudy 7/22/82, RCA 49%; PTCA repeated; no chest pain</td>
</tr>
<tr>
<td>No 396</td>
<td>+</td>
<td>No chest pain</td>
</tr>
</tbody>
</table>
**TABLE 1B**
Clinical characteristics of acute occlusion patients 11 through 20

<table>
<thead>
<tr>
<th>EUH study patient No.</th>
<th>Sex</th>
<th>Date</th>
<th>Patient No. (this study)</th>
<th>Vessel dilated</th>
<th>% Diameter narrowing Before</th>
<th>Pressure gradient Before</th>
<th>Time from PTCA to occlusion (hr)</th>
<th>Documenta-</th>
<th>Predisposing factors to occlusion</th>
<th>Treatment of patients</th>
<th>Emerg. CABG</th>
</tr>
</thead>
<tbody>
<tr>
<td>759</td>
<td>M</td>
<td>3/17</td>
<td>11</td>
<td>RCA</td>
<td>85</td>
<td>17</td>
<td>&lt;2</td>
<td>ECG</td>
<td>Intimal tear</td>
<td>Spontaneous resolution</td>
<td>No</td>
</tr>
<tr>
<td>789</td>
<td>M</td>
<td>4/7</td>
<td>12</td>
<td>LAD</td>
<td>70</td>
<td>41</td>
<td>&lt;1</td>
<td>ECG</td>
<td>Intimal tear</td>
<td>Spontaneous resolution</td>
<td>No</td>
</tr>
<tr>
<td>796</td>
<td>M</td>
<td>4/12</td>
<td>13</td>
<td>LAD</td>
<td>75</td>
<td>30</td>
<td>&lt;1</td>
<td>CA</td>
<td>None</td>
<td>Repeat PTCA (without resolution)</td>
<td>No</td>
</tr>
<tr>
<td>463</td>
<td>F</td>
<td>4/13</td>
<td>14</td>
<td>LAD</td>
<td>70</td>
<td>12</td>
<td>&lt;2</td>
<td>ECG; CA</td>
<td>Intimal tear</td>
<td>ICU NTG; PTCA (without resolution)</td>
<td>No</td>
</tr>
<tr>
<td>899</td>
<td>M</td>
<td>6/9</td>
<td>15</td>
<td>RCA</td>
<td>64</td>
<td>22</td>
<td>&lt;2</td>
<td>ECG</td>
<td>Intimal tear</td>
<td>None</td>
<td>No</td>
</tr>
<tr>
<td>932</td>
<td>M</td>
<td>6/21</td>
<td>16</td>
<td>Cx</td>
<td>90</td>
<td>32</td>
<td>16</td>
<td>CA</td>
<td>Intimal tear</td>
<td>ICU streptokinase (without resolution)</td>
<td>No</td>
</tr>
<tr>
<td>972</td>
<td>M</td>
<td>7/16</td>
<td>17</td>
<td>LAD</td>
<td>66</td>
<td>20</td>
<td>18</td>
<td>ECG</td>
<td>Intimal tear</td>
<td>No treatment except bypass</td>
<td>No</td>
</tr>
<tr>
<td>1051</td>
<td>M</td>
<td>8/20</td>
<td>18</td>
<td>RCA</td>
<td>74</td>
<td>34</td>
<td>&lt;2</td>
<td>CA</td>
<td>Intimal tear</td>
<td>ICU NTG</td>
<td>No</td>
</tr>
<tr>
<td>1052</td>
<td>M</td>
<td>8/20</td>
<td>19</td>
<td>RCA</td>
<td>66</td>
<td>31</td>
<td>&lt;1</td>
<td>CA</td>
<td>Intimal tear</td>
<td>Recrossed dilated</td>
<td>No</td>
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<tr>
<td>1151</td>
<td>M</td>
<td>9/30</td>
<td>20</td>
<td>Cx</td>
<td>56</td>
<td>22</td>
<td>&lt;1</td>
<td>ECG; CA</td>
<td>None</td>
<td>ICU NTG</td>
<td>No</td>
</tr>
</tbody>
</table>

*Actual time of occlusion is not known. Time indicated is time from PTCA to discovery of total occlusion by ECG or coronary angiography. EUH = Emory University Hospital; CA = coronary angiography; LV = left ventricular; Cx = circumflex; neg = negative; EF = ejection fraction; mod = moderate; ETT = exercise treadmill testing; wnl = within normal limits; CABG = coronary artery bypass graft; cath = catheterization; NTG = nitroglycerin; MI = myocardial infarction; CK = creatine kinase; IC = intracoronary.*

Only sublingual nitrates, nifedipine, and repeat boluses of heparin were given. All three had elevated enzyme levels, but the ECG criteria for infarction were not met. In 11 patients repeat angioplasty was attempted and it reopened the artery, relieved the chest pain, and reversed the ST segment elevation in eight patients. In one patient the syndrome was transient; repeat PTCA was necessary every 10 min to keep the vessel from reoccluding. In one patient the lesion could not be recrossed and in another an attempt to recross resulted in coronary artery dissection.

Intracoronary nitroglycerin alone reopen the vessel in three patients (Nos. 5, 18, and 20). In patient 5 this relief was transient. Repeat bolus infusions were required every 5 min to prevent recurrence of occlusion. Streptokinase was given to three patients (1, 14, and 16); in one of these three (16) streptokinase was judged to be useful in resolving the arterial occlusion.

**Discussion**

The treatment these patients received represents a significant departure from that previously used; there was a time when all patients with acute occlusion underwent emergency bypass surgery. The discovery that most of these patients, once their arteries were reopened, could be stabilized with 24 hr continuous intravenous infusions of heparin and nitroglycerin has allowed safe treatment without bypass surgery.

To be included in this study patients had to have had a satisfactory initial result from angioplasty. In 16 of the study patients the onset of symptoms occurred after they left the cardiac catheterization laboratory. For this
TABLE 1B  
(Continued)  

<table>
<thead>
<tr>
<th>Q wave CK</th>
<th>Follow-up data</th>
</tr>
</thead>
<tbody>
<tr>
<td>MI N &lt; 170 MB+</td>
<td></td>
</tr>
</tbody>
</table>

No 82 – No chest pain active  
No 282 – 6/4/82, ETT negative 7½ min  
No 77 – No follow-up  
No 53 – 6 month angiogram showed no recurrence  
No 1626 + Symptoms recurred 2 months after PTCA, no objective follow-up  
No 1510 + Negative treadmill, negative angiogram  
Yes 2612 + 8/20, radionuclear EF 48% minimal apical, septal hypotension  
No 65 – Intermittent chest pain  
No 50 – No chest pain  
No 65 – No chest pain

reason, careful monitoring of patients during the initial 3 to 4 hr after angioplasty is particularly important.

Although five of these 20 patients underwent emergency surgery, 15 of these patients did not and previously all 20 patients would have undergone emergency surgery. Myocardial infarction occurred relatively frequently in the patients who underwent surgery (4/5 or 80%), but the rate is only 20% (4/20) when all patients are considered. Data from the NHLBI registry shows that nine of 29 (31%) patients with acute occlusion fulfilled ECG criteria for infarction. In all five of our patients who had infarctions, follow-up assessment of left ventricular function demonstrated at least some wall motion in the region supplied by the vessel in which PTCA failed, thus suggesting that emergency bypass surgery is useful in salvaging functioning myocardium. None of these five received intra-aortic balloon counterpulsation: patients 3, 4, 5, and 8 were treated before our first experi-
ence with this technique and patient 17 did not receive counterpulsation for logistic reasons.

Creatine kinase levels were elevated in 12 patients. These values might be higher than would be expected after a traditionally treated myocardial infarction because of the washout phenomena associated with reperfusion. The benign clinical course in these patients after their arteries were reopened suggests that the infarctions were small and of no clinical consequence.

Mechanism of acute occlusion. The mechanisms of acute occlusion may well be multiple. It is not possible to definitely distinguish between coronary spasm, coronary thrombosis, and subintimal hemorrhage by angiographic criteria. Schoffer et al. suggested that acute occlusion was due to coronary thrombosis because three of their patients responded to intracoronary streptokinase and discontinuation of the infusion was associated with reclosure in one patient. Our study differs from that of Schoffer et al. in that all our patients had undergone elective PTCA and in all the procedure was initially successful. In their study PTCA was performed immediately after successful streptokinase recanalization of an acute myocardial infarction in one patient and another patient in their group did not have any balloon inflations but had arterial closure associated with attempted passage of the dilatation catheter. All the occlusions they observed occurred while the patients were still in the catheterization laboratory. Sixteen of our patients had left the laboratory. As additional evidence for coronary thrombosis, these authors cite the work of Block et al. in which coronary thrombosis at the site of PTCA was demonstrated at autopsy of two patients who died after PTCA and emergency coronary bypass surgery.

There are several reasons to question the diagnosis of coronary thrombosis. First, all patients in our series were pretreated with aspirin and heparin. Second, angiographic evidence of thrombosis was demonstrated in only one patient in whom occlusion was observed 16 hr after successful PTCA. In a prospective study of patients with acute myocardial infarction, DeWood et al. found thrombus to be present at surgery in 88.1% of patients in whom thrombus was demonstrated angiographically. Moreover, only five of 20 patients (25%) had thromboses at surgery that were not apparent on their angiograms. Since the lumens of these vessels were relatively open (residual diameter narrowing 23 ± 11%), it is unlikely that a thrombus large enough to occlude these vessels would escape angiographic detection. Intra-arterial thrombi tend to form in vessels with slow flow; the minimal residual pressure gradient of 11 ± 8 mm Hg by itself would not
FIGURE 1. Illustrative case (patient 20). A, Baseline ECG recorded at 12:15 P.M. (lead V5). B, Lesion before angioplasty. C, Balloon inflated in lesion. D, ECG changes at 12:28 P.M. with balloon inflation; mild chest pain present. Gradient with balloon inflated, 100 mm Hg. E, Result immediately after angioplasty. Pain-free at 12:45 P.M. F, Patient returned to laboratory with severe chest pain and ST segment elevation at 1:31 P.M. G, Angiogram taken a few minutes later showing total occlusion at the site of balloon inflation. H, Result after 200 μg intracoronary nitroglycerin; chest pain less severe. I, Result after two further doses of intracoronary nitroglycerin and 10 mg sublingual nifedipine. Chest pain relieved. A later angiogram showed appearance similar to E. The patient was stabilized for 24 hr with a continuous infusion of intravenous nitroglycerin and heparin. He had no evidence of myocardial damage on creatine kinase enzyme testing or ECG.
seem enough to predispose to slow flow and thrombus formation.

Third, distal coronary embolism was detected in only one patient (No. 16) who also had angiographic evidence of thrombosis. Fourth, although Block et al. have shown thrombus in the postmortem coronary arteries of patients who died after successful angioplasty, these were relatively small nonocclusive thrombi. Their patients died after bypass surgery and one died 90 hr after PTCA. Also, considerable differences in coagulation status may have been present.

Coronary arterial spasm is likely important in many of these patients since arterial spasm is the common response of an artery to injury. Failure to immediately respond to sublingual and/or intracoronary nitroglycerin or nifedipine does not exclude the possibility of a more delayed response to these medications, although coronary spasm refractory to sublingual nitroglycerin has been reported. Eight of our patients responded to sublingual nifedipine and sublingual and intracoronary nitrates. In patient 5 about 15 coronary injections of nitroglycerin were used, each transiently opening the artery for 2 to 3 min.

While coronary spasm plays a role in these patients, the close relationship between the discontinuation of heparin and the occurrence of acute occlusion warrants further discussion. For reasons listed above, we do not believe that coronary thrombosis plays an important obstructive role in patients presenting early with acute coronary occlusion. This does not exclude the possibility that coagulation or some other process inhibited by heparin may play an important role in acute occlusion. Heparin has been used successfully in the treatment of unstable angina, a syndrome associated with coronary spasm and, in some cases, nonoccluding coronary thrombosis. Perhaps in situ coagulation, while not occluding the artery, may contribute to the local initiation of coronary arterial spasm through a yet-unrecognized trigger.

In a preliminary communication Peterson et al. have demonstrated a nearly 10-fold rise in coronary sinus thromboxane B level (the measured metabolite of the powerful vasoconstrictor thromboxane A) in a patient with acute coronary occlusion. An insignificant rise was observed in five patients who did not have acute occlusion. Others have demonstrated, in dog carotid arteries, that angioplasty can decrease vascular prostaglandin synthesis and can stimulate lipooxygenase metabolism, thus predisposing to coronary spasm.

Although these findings are intriguing, they do not establish the cause of coronary occlusion after angioplasty. In vasospastic angina thromboxane inhibition has failed to decrease the number of attacks. Our patients all received aspirin (650 mg) the night before angioplasty, and this should have inhibited thromboxane production. In patients with unstable angina, it is not known whether the associated elevation of thromboxane in the coronary sinus is a primary or secondary event. Similarly, in those with acute occlusion the thromboxane elevation may be a primary or a secondary event. Perhaps such a release is important only in the perpetuation of occlusion.

The presence of an intimal tear that is visible angiographically may mean that a large amount of collagen is exposed to platelets. In any case, because of these observations, we now place all patients with a large intimal tear on their post-PTCA control angiograms on continuous intravenous infusions of nitroglycerin and heparin for at least 24 hr.

Coronary occlusion occurs in about 2% of patients undergoing successful angioplasty. Most patients present within 3 hr after receiving 10,000 units of heparin or within 3 hr after the discontinuation of a heparin infusion. Although bypass surgery is required in some patients, in most patients acute occlusion can be reopened with nitrates, calcium antagonists, or repeat angioplasty. Once acute occlusion has been resolved, patients may be effectively stabilized with continuous infusions of nitroglycerin and heparin. The mechanism of acute occlusion is still not precisely known, but it is doubtful that occluding thrombosis plays a significant role. It is more likely that acute occlusion represents a refractory form of local coronary arterial spasm.

We thank Kathy Galan for assistance with the follow-up, Sharon Lane for technical assistance, and William Proudfit, M.D., for his critical review of the manuscript.

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Vol. 68, No. 4, October 1983

731
Acute occlusion after percutaneous transluminal coronary angioplasty--a new approach.
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Circulation. 1983;68:725-732
doi: 10.1161/01.CIR.68.4.725
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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