Coronary Occlusion During Coronary Angiography


SUMMARY
Between January 1, 1970, and December 31, 1974, 2981 patients underwent coronary arteriography. Twelve acute coronary dissections or embolizations occurred, an incidence of 0.4%. The incidences of acute occlusions for the Sones and Judkins techniques were 0.19% (4/2077 studies) and 0.88% (8/940), respectively. No instance of acute occlusion has occurred during the past 490 studies performed by the Judkins technique. Eight patients with right coronary artery dissections or circumflex emboli were treated medically. All survived, but in seven a myocardial infarction evolved. Four patients underwent emergency saphenous venous bypass grafting because of refractory ventricular fibrillation (two patients) or because large amounts of myocardium were thought jeopardized (two patients). All patients in this group had interruption of flow supplying the left anterior descending coronary artery. Despite surgical intervention in less than three hours in all patients, survivors all sustained transmural myocardial infarctions. Three patients survived surgery and were discharged home.

One of the most serious complications of coronary angiography is coronary artery occlusion from coronary dissection or embolization. Now that myocardial revascularization can be performed with an acceptable risk, even in the most critically ill patient, at issue is whether to treat these coronary angiographic complications medically or surgically. With this question in mind, the experience of the cardiac catheterization laboratory and cardiac surgery unit at the Massachusetts General Hospital was reviewed for a five-year period (January, 1970 — December, 1974) in an effort to determine the place of emergency myocardial revascularization for acute coronary occlusion during coronary angiography. The results of this experience are described.

Results
Between January 1, 1970, and December 31, 1974, 2981 patients underwent coronary angiography either as part of a right and left heart catheterization or as the only procedure. All injections of contrast media were hand injections. Twelve instances of catheter-induced coronary occlusion occurred, an over-all incidence of 0.4%. Of 2077 patients studied by the Sones technique, 4 four patients sustained dissections of the right coronary artery (RCA) (table 1), an incidence of 0.19%. Of 904 patients studied by the Judkins technique, 8 eight patients (0.88%) sustained acute coronary occlusions: dissections of the main left coronary artery (MLCA) in one patient; dissection of the RCA in two patients; a fat embolus to the MLCA in one patient; an embolus to the left anterior descending (LAD) in one patient; an embolus to the circumflex coronary artery (CMFX) in three patients. Since June of 1973, when systemic heparinization was instituted (0.5 — 0.1 mg/kg), no acute occlusions have occurred in the catheterization laboratory in 490 cases using the Judkins technique. Of the 521 coronary arteriograms performed using the Sones technique since June of 1973, one RCA dissection (0.19% acute occlusion) has occurred.

Eleven of twelve patients developed severe precordial pain within minutes of the occlusion. The twelfth patient (patient 6, table 2) had no chest discomfort but developed ECG changes immediately and an acute transmural infarct evolved. All 12 patients developed ECG changes within minutes, and the location of these changes could be predicted by the coronary artery or the collateral supply involved in all patients except patient 11 (table 2).

Criteria for the diagnosis of acute myocardial infarction required the occurrence of two of the following three findings: 1) prolonged, severe precordial pain; 2) appearance of new Q waves on ECG; 3) appropriate enzyme elevations (SGOT, CPK, LDH).

Criteria for coronary dissection included the appearance of flow in two channels separated by a thin radiolucent septum and slow or delayed run-off from the false channel. As the central vessel cleared, the false channel rimmed the vessel periphery. In some
because they were felt to have a minimum of myocardium in jeopardy and because their hemodynamics, electrical activity, and clinical status remained or quickly returned to normal (tables 2 and 3). Seven had severe atherosclerotic coronary artery involvement of at least two vessels, and one patient (patient 5, table 2) had normal coronary arteries.

One patient (patient 1, table 2) developed ventricular fibrillation in the laboratory after completion of the procedure and was easily defibrillated to normal sinus rhythm. Other complications occurring within the first 12 hours included brief runs of ventricular tachycardia in two patients, easily controlled by lidocaine, mild ventricular irritability in three patients; and transient hypotension in two patients. Two patients had no arrhythmias (table 4).

All patients survived (fig. 1). Seven of the eight patients evolved an acute myocardial infarction in areas corresponding to the occluded coronary vessel. Four patients with RCA dissections developed transmural inferior infarctions. One patient with an embolus to a left dominant CMFX, which supplied branches to the inferior wall, developed a transmural inferior infarct. Two patients with CMFX emboli developed apical and lateral infarcts of the subendocardium. One patient (patient 7) with a RCA dissection developed

### Table 1

**Five-year Incidence: Acute Coronary Occlusion during Coronary Angiography**

<table>
<thead>
<tr>
<th></th>
<th>Sones</th>
<th>Judkins</th>
<th>Judkins (antiocoagulation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cases</td>
<td>2077</td>
<td>904</td>
<td>490</td>
</tr>
<tr>
<td>% Acute occlusion</td>
<td>0.19%</td>
<td>0.88%</td>
<td>0.0%</td>
</tr>
<tr>
<td>Dissection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MLCA</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>RCA</td>
<td>4</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Embolization</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MLCA</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>LAD</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>CMFX</td>
<td>0</td>
<td>3</td>
<td>0</td>
</tr>
</tbody>
</table>

**Abbreviations:** MLCA = main left coronary artery; RCA = right coronary artery; LAD = left anterior descending coronary artery; CMFX = circumflex coronary artery.

cases only the false channel opacified. Coronary emboli were demonstrated by the total occlusion of a vessel with a normal arterial caliber up to the point of an abrupt occlusion. In many of these patients, a previous injection had shown patency of the vessel.

### Medically Treated Patients

Eight patients were treated medically (five patients with RCA dissections and three with CMFX emboli)

### Table 2

**Acute Coronary Occlusion During Angiography**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Technique</th>
<th>Coronary occlusion</th>
<th>Surgical procedure</th>
<th>Result</th>
<th>Survival in months to Jan., 1975</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medically Treated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. E.B.</td>
<td>51</td>
<td>M</td>
<td>Judkins</td>
<td>CMFX embolus</td>
<td>none</td>
<td>Subendocardial apical MI</td>
<td>35</td>
</tr>
<tr>
<td>2. F.D.</td>
<td>48</td>
<td>M</td>
<td>Judkins</td>
<td>CMFX embolus</td>
<td>none</td>
<td>Transmural inferior MI</td>
<td>21</td>
</tr>
<tr>
<td>3. W.K.</td>
<td>43</td>
<td>M</td>
<td>Judkins</td>
<td>RCA dissection</td>
<td>none</td>
<td>Transmural inferior MI</td>
<td>35</td>
</tr>
<tr>
<td>4. J.W.</td>
<td>52</td>
<td>M</td>
<td>Judkins</td>
<td>RCA dissection</td>
<td>none</td>
<td>Transmural inferior MI</td>
<td>34</td>
</tr>
<tr>
<td>5. M.F.</td>
<td>61</td>
<td>F</td>
<td>Judkins</td>
<td>CMFX embolus</td>
<td>none</td>
<td>Subendocardial apical MI</td>
<td>19</td>
</tr>
<tr>
<td>6. K.R.</td>
<td>32</td>
<td>F</td>
<td>Sones</td>
<td>RCA dissection</td>
<td>none</td>
<td>Transmural inferior MI</td>
<td>27</td>
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<tr>
<td>7. E.C.</td>
<td>49</td>
<td>F</td>
<td>Sones</td>
<td>RCA dissection</td>
<td>none</td>
<td>Transient inferior ST-elevation</td>
<td>33</td>
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<tr>
<td>8. K.K.</td>
<td>50</td>
<td>M</td>
<td>Sones</td>
<td>RCA dissection</td>
<td>none</td>
<td>Transmural inferior MI</td>
<td>6</td>
</tr>
<tr>
<td>Surgically Treated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. V.D.</td>
<td>53</td>
<td>F</td>
<td>Judkins</td>
<td>MLCA dissection</td>
<td>CABG to LAD</td>
<td>Transmural anteroseptal MI</td>
<td>39</td>
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<td>10. A.P.</td>
<td>40</td>
<td>M</td>
<td>Judkins</td>
<td>MLCA fat embolus</td>
<td>CABG to LAD</td>
<td>Died in O.R.</td>
<td>died</td>
</tr>
<tr>
<td>11. B.M.</td>
<td>47</td>
<td>M</td>
<td>Judkins</td>
<td>LAD embolus</td>
<td>CABG to LAD</td>
<td>Transmural antero-lateral MI</td>
<td>19</td>
</tr>
<tr>
<td>12. C.S.</td>
<td>52</td>
<td>M</td>
<td>Sones</td>
<td>RCA dissection</td>
<td>CABG to LAD &amp; RCA</td>
<td>Transmural antero-septal MI</td>
<td>49</td>
</tr>
</tbody>
</table>

**Abbreviations:** MLCA = left main coronary artery; LAD = left anterior descending coronary artery; CMFX = circumflex coronary artery; RCA = right coronary artery; MI = myocardial infarction; CABG = coronary artery bypass graft (saphenous vein).
severe pain and initial inferior lead ST elevation. The arterial lumen was not totally occluded, and distal run-off could be seen angiographically (fig. 2). This patient did not develop enzyme elevations, and the ECG reverted to its precatheterization pattern within 24 hours. All eight patients had a clinically benign course after the first 12 hours, and all patients were alive as of January, 1975.

Surgically Treated Patients

Four patients underwent emergency myocardial revascularization with saphenous vein bypass grafts (tables 2, 3, and fig. 1). All patients but one with a MLCA dissection and normal distal vessels had severe three vessel disease. Bypass grafts were limited to the acutely involved vessel in patients 9, 10, and 11, but all technically bypassable and diseased vessels were grafted in patient 12.

The decision to perform emergency revascularization was dictated by the occurrence of refractory ventricular fibrillation in two patients, both with acute occlusions of the MCLA. Both patients were placed on cardiopulmonary bypass in less than one hour after coronary occlusion. One patient had a fat embolus to the MCLA (documented at postmortem examination) and could not be sustained off cardiopulmonary bypass despite an embolectomy and saphenous vein bypass graft. The second patient, who survived surgery, had a dissection of the MLCA; and surgery consisted of a vein graft to the LAD coronary artery. Despite early surgical intervention, postoperatively this patient developed new Q waves in the anterior precordial leads and enzyme elevations consistent with an acute myocardial infarction. In the other two patients the decision to pursue surgery was based upon the presence of severe hypotension and significantly jeopardized myocardium in the distribution of the LAD coronary artery. One of these patients had an embolus to the LAD artery (fig. 3). The patient was placed on cardiopulmonary bypass within one and one-half hours after the acute occlusion. Despite early surgical intervention and demonstration of LAD graft patency during a study performed two weeks postoperatively, the patient evolved an anterolateral infarct postoperatively with new Q waves and an aki-netic anterolateral wall.

The fourth surgically treated patient is especially interesting because almost all of the distal LAD blood supply came from RCA collaterals. When a RCA dissection occurred, there were marked ST and T wave abnormalities in the anterior precordial leads consistent with anterior ischemia. Despite emergency placement of a vein graft to the LAD and RCA in less than three hours after the acute occlusion, the patient developed new Q waves in precordial leads V1–V4 and enzyme elevations consistent with an acute myocardial infarction.

In all four patients, infarct patterns were limited to the distribution of the acutely involved vessel as documented by ECG or angiographic evidence.

The three survivors were discharged home and are alive as of January, 1975. Except for a resolving hip
flexor paresis in patient 9 (table 2), survivors had clinically benign postoperative courses.

Discussion

Myocardial infarction related to coronary angiography is fortunately a relatively rare complication. Our incidence of acute coronary occlusion during coronary angiography (0.4% for all coronary studies) is similar to other large centers where the occurrence is between 0.3 and 0.9%. Although coronary complications have been reported in some centers to occur in 1.1 to 4.3% of studies. The number of acute occlusions for the Sones technique (4/2077, 0.19%) was considerably less than for the Judkins technique (8/904, 0.88%) and reflects the higher risk of the Judkins technique seen in many institutions. Two years after initiating the Judkins technique in our laboratory, systemic heparinization was instituted. Since that time, no thromboembolic coronary artery complications have occurred in 490 cases. This reduction in coronary occlusive complications with heparinization in our most recent cases has been seen in other laboratories also but may reflect a decrease in the incidence of coronary dissections rather than the effect of heparin. In our laboratory, the Judkins technique now appears to be as safe as the Sones technique.

In all cases of coronary dissection nonocclusive
pressure recording and test injections had demonstrated adequate run-off of contrast agent prior to full coronary opacification. Dissections originated proximally near the catheter tip, and all patients had intrinsic disease of the right coronary artery (RCA), visualized angiographically, although the exact site of intimal tear could not always be correlated with an angiographically demonstrated plaque. In most cases, an atherosclerotic intima was probably lifted up. Avoidance of dissection by this mechanism may not be possible despite the most meticulous attention to detail and safety. One main left coronary artery (MLCA) dissection occurred in a patient with normal coronary arteries and may be related to problems inherent in the Judkins technique such as the limited fine control of the catheter and the wedging of the catheter tip in the coronary ostium.

Several minutes elapsed before the development of chest pain after coronary dissections. It is postulated that it takes a matter of minutes before the subintimal hematoma totally occludes the lumen. Interestingly, in the one patient (patient 7, table 2) with a RCA dissection who did not develop an infarction, total luminal occlusion did not occur, and distal run-off of contrast medium was observed. This observation has been previously reported in a patient displaying resolution of the process on repeat catheterization five days later. One of us (S. Guss, unpublished observations from another institution) has also observed a similar patient with a RCA dissection without total luminal occlusion. No pain or ECG changes occurred, and no evidence of dissection could be detected on repeat catheterization 48 hours later.

The decision to perform emergency myocardial revascularization was dictated by refractory ventricular fibrillation in two patients. One patient with a fat embolus to the MLCA died in the operating room after embolectomy and saphenous vein bypass surgery, despite the institution of cardiopulmonary bypass within one hour of the acute occlusion. The second patient with refractory ventricular fibrillation is alive today because of emergency surgery. Nevertheless, despite prompt institution of cardiopulmonary bypass, within 50 minutes after the acute episode this patient evolved an acute anterior infarct. In the other two patients, a surgical decision was predicated upon an assumption that a large segment of myocardium was jeopardized and because of significant hypotension. Despite early intervention (cardiopulmonary bypass in 1.5 hours in patient 11 and three hours in patient 12), both patients evolved postoperative infarcts by ECG (new Q waves) and enzyme elevations. In spite of this, in patient 11, postoperative graft patency was demonstrated angiographically at two weeks.

The importance of coronary collaterals has been in dispute for a long time. In our patient 12 in whom the distal LAD was supplied principally by collaterals from the RCA (although some bridging collaterals were visualized), surgery to bypass an occlusion of the RCA was followed by development of a new anteroseptal infarction. In this case the presence of collateral pathways did not prevent development of permanent injury in an acutely ischemic area.

This study reinforces the work of others who have demonstrated the feasibility of placing patients on cardiopulmonary bypass rapidly after an acute occlusion and the ability to operate safely on patients with acute infarcts. The real question is whether surgical intervention prevents the inevitability of myocardial infarction after an acute coronary occlusion or limits the necrotic process by revascularizing marginally perfused areas. We were unable to demonstrate any prophylaxis against infarction in our small series as all patients with total occlusions developed infarcts regardless of the therapy employed.

As for infarct size reduction and myocardium salvage, dog experiments are contradictory. Beller et al. have shown nonhomogeneous myocardial blood distribution while Bresnahan et al. demonstrated hemorrhagic infarct extension in 44% of animals reperfused after coronary occlusion. However, Ross has recently shown improvement in ventricular function after the initial postoperative period has passed. Species differences may be important, and animal studies may not be applicable to humans who have chronic ischemia and coronary collateralization.

Until more is known about infarct prevention and myocardium salvage with immediate surgical revascularization for acute coronary occlusion, we are recommending medical therapy for clinically stable patients. We would reserve immediate coronary artery bypass surgery for medically unstable patients with refractory ventricular fibrillation, severe hypotension, or pulmonary edema. Patients with MLCA lesions or LAD lesions will most frequently fall in this category. We would treat clinically stable patients with large amounts of myocardium in jeopardy medically until data suggesting that tissue salvage is likely to occur with surgical intervention becomes available. The risk of patients operated upon, often within an hour of acute coronary occlusion, developing new infarctions is too great.

Acknowledgment

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References

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