Selective Coronary and Left Ventricular Cineangiography during Intraaortic Balloon Pumping for Cardiogenic Shock

By ROBERT C. LEINBACH, M.D., ROBERT E. DINSMORE, M.D., ELDRED D. MUNDTH, M.D., MORTIMER J. BUCKLEY, M.D., W. BRUCE DUNKMAN, M.D., W. GERALD AUSTEN, M.D., and CHARLES A. SANDERS, M.D.

SUMMARY
Eleven patients underwent selective coronary and left ventricular cineangiography during intraaortic balloon pumping for acute myocardial infarction and cardiogenic shock. Studies were performed when the clinical and hemodynamic trend predicted in-hospital death. Angiograms were reviewed by dividing the left ventricle into six segments, each with its characteristic coronary perfusion. No patient with avascular and akinetic segments survived with or without surgery. One patient with residual perfusion of all segments showed massive necrosis at operation that eventually led to his death. In four patients the findings of residual contraction and perfusion of most segments suggested reversible ventricular dysfunction. Three of these underwent coronary surgery with two excellent survivors.

Additional Indexing Words:
Ventricular segments Infarct size Bypass grafts Criteria for operation

THE INTRAARTIC BALLOON pump is an effective device for support of the circulation. It has become our treatment of choice for patients with cardiogenic shock complicating acute myocardial infarction. Its effect is often dramatic, and in most cases shock is reversed. The impact on hospital and long-term mortality, however, has been less impressive.1 For this reason we have performed selective coronary and left ventricular cineangiography on a series of these patients during intraaortic balloon pumping (IABP) in a search for surgically reversible disease. The first 11 of these patients are described in this report.

Materials and Methods
The patients are listed in table 1. All were male with an average age of 54 years. All had acute or remote infarction involving the anterior wall. The average time from onset of infarction to shock was 56 hours. The patients were transferred to the Myocardial Infarction Research Unit Intensive Study Area where a radial or femoral artery cannula and a Swan-Ganz flow-directed pulmonary artery catheter were inserted. The depth of shock was quantitated after correction of acidosis, arrhythmias, and hypoxemia by measuring arterial pressure, pulmonary wedge pressure, and cardiac index (dye dilution). If catecholamines could not be withdrawn, or if in the absence of catecholamines the product of the mean arterial pressure and cardiac index was less than 120 mm Hg liters/min/m² and the wedge pressure was 18

From the Medical and Surgical Services and the Department of Radiology of the Massachusetts General Hospital, and the Departments of Medicine, Surgery, and Radiology, Harvard Medical School, Boston, Massachusetts.

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Address for reprints: Dr. Robert C. Leinbach, Cardiac Catheterization Unit, Massachusetts General Hospital, Boston, Massachusetts 02114.

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### Clinical Data

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<th>Reason for angiogram</th>
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<td>Second balloon assistance in 3 weeks</td>
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<td>congestion 12 days after MI</td>
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<td>M</td>
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<td>ALMI; old AMI</td>
<td>45</td>
<td>M</td>
<td>90</td>
<td>Balloon dependence</td>
</tr>
</tbody>
</table>

Abbreviations: AMI = anterior myocardial infarction; AS = anteroseptal; AL = anterolateral; I = inferior.

*Operated, survived.
†Operated, died.

mm Hg or more, the AVCO-MGH three-segment intraaortic balloon\(^2\) was placed without further delay. The average time from the onset of shock to institution of IABP was nevertheless 37 hours, primarily because of delayed referral. In four of these 11 patients IABP was begun less than 12 hours after onset of shock.

### Criteria for Angiography

Angiograms were performed after variable periods of IABP if the clinical and hemodynamic trend predicted in-hospital death. Criteria for this outcome were based on experience with 25 patients who did not undergo revascularization surgery. The criteria were: (1) failure to improve significantly after 12–24 hours of IABP and catecholamine infusion, (2) continued high or increasing catecholamine requirements in spite of IABP, (3) refractory pump failure more than 1 week after myocardial infarction, and (4) balloon dependence after 48 hours of assistance. Balloon dependence was assessed by performing hemodynamic measurements in the absence of catecholamines after abrupt discontinuance of IABP. If the mean arterial pressure fell to less than 55 mm Hg, the cardiac index fell to less than 2.0 liters/min/m\(^2\), the pulmonary wedge pressure rose above 20 mm Hg in spite of diuretics, or angina pectoris developed, balloon dependence was diagnosed. At the time of angiography, six patients were still receiving catecholamine infusions. The average hemodynamic values just prior to angiography were: cardiac index 2.2 liters/min/m\(^2\), mean arterial pressure 73 mm Hg, and pulmonary wedge pressure 23 mm Hg.

The patients were transferred to the Cardiac Catheterization Laboratory without interruption of IABP utilizing a battery-operated AVCO pump console. Angiograms were performed by the Sones technic. The coronary arteries were opacified early in the series with 69% sodium diatrizoate (Hypaque, 69%) and later with 66% meglumine diatrizoate and 10% sodium diatrizoate (Renographin, 76%). Left ventriculograms were performed with 34% meglumine diatrizoate and 35% sodium diatrizoate (Renovist, 69%). Single-plane 35-mm cineradiography was used. In most cases the left ventricle was opacified first in the right anterior oblique position. Following a 15-min rest period a repeat ventriculogram in a high left anterior oblique position was performed in four of these cases. The study was completed with selective opacification of both coronary arteries in both oblique positions.

Coronary arteries were judged to be severely obstructed if the projected diameter was narrowed by 75% or more. Ventricleograms were analyzed by tracing the end-diastolic and end-systolic cavity margins in the 30° right anterior oblique view and the 60° left anterior oblique view. Since the studies were performed with a 6-inch image intensifier, panning was necessary to film the entire chamber. The reconstructed cavity outlines were planimetered, and the results were expressed as approximate areas (corrected for magnification). Volume measurements were not
made because such calculations compound the error introduced by panning, and because all cases during systole showed asymmetry and akinesis.3

Postmortem examinations were performed as previously described.4 The formalin-fixed hearts were sectioned horizontally from apex to atrioventricular groove producing six sections of equal thickness. Sections for light microscopy were taken from each slice. The extent of the infarction was diagrammed and the percent necrosis of the left ventricle was estimated. The angiograms of these patients were reviewed, and avascular and akinetic areas were plotted in an attempt to correlate the radiographic findings with the extent and distribution of infarction.

Results

The angiographic studies were remarkably well tolerated. There was one episode of ventricular fibrillation during injection of the right coronary artery (Hypaque), which was easily reverted with countershock. The first patient in this series (A.K.) was the only fatality possibly attributable to angiography. Thirty minutes after an uneventful study when the patient had stabilized, IABP had to be discontinued for technical reasons relating to transport. The patient sustained a cardiac arrest and could not be resuscitated. Since this experience, interruptions of IABP have not been necessary, and all patients have remained stable during transfer.

Systemic arterial pressure fell transiently in several instances following ventriculography as shown in figure I. Left ventricular end-diastolic pressure also rose but returned to control within 10 min. With continuation of IABP and a 15-min rest between ventriculograms, pulmonary edema was not produced, and the 10 patients who returned to the Intensive Study Area showed no ill effects.
Table 2

Coronary Arteries with Significant Obstruction of Occlusion

<table>
<thead>
<tr>
<th>No.</th>
<th>Pt</th>
<th>Vessels 75% or more obstructed</th>
<th>Major open vessel</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A.W.*</td>
<td>RCA LAD</td>
<td>C-CM</td>
</tr>
<tr>
<td>2</td>
<td>E.P.†</td>
<td>RCA LAD</td>
<td>C None</td>
</tr>
<tr>
<td>3</td>
<td>C.M.*</td>
<td>RCA LAD</td>
<td>CM</td>
</tr>
<tr>
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<td>A.K.</td>
<td>RCA LAD</td>
<td>C-CM</td>
</tr>
<tr>
<td>5</td>
<td>H.M.</td>
<td>RCA LAD</td>
<td>C CM</td>
</tr>
<tr>
<td>6</td>
<td>A.S.†</td>
<td>RCA LAD</td>
<td>C None</td>
</tr>
<tr>
<td>7</td>
<td>J.W.*</td>
<td>RCA LAD</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>J.D.†</td>
<td>RCA LAD</td>
<td>C None</td>
</tr>
<tr>
<td>9</td>
<td>G.P.</td>
<td>LAD RCA-C</td>
<td>RCA-C</td>
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<tr>
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<td>RCA Main L</td>
<td>None</td>
</tr>
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<td>11</td>
<td>A.F.†</td>
<td>LAD C</td>
<td>RCA</td>
</tr>
</tbody>
</table>

Abbreviations: RCA = right coronary artery; LAD = left anterior descending; C = circumflex; CM = circumflex marginal.

*Operated, survived.
†Operated, died.

The major coronary artery obstructions are listed in Table 2. The left anterior descending coronary artery was narrowed by greater than 75% or occluded in all cases. Nine also showed obstruction of the right coronary artery proximal to the origin of the posterior descending branch. High-grade obstruction of all major vessels was present in four. Patient H.M. showed three- vessel obstruction, but the circumflex coronary artery was narrowed distal to a large circumflex marginal branch supplying an active posterolateral segment.

Table 3 ranks these patients in order of increasing left ventricular diastolic area. Normal ventricles filmed with the same equipment in the right anterior oblique position show end-diastolic areas up to 45 cm² and end-systolic areas of approximately 20 cm². Two of these patients showed no cardiac dilatation by this method. All showed diminished ejection.

Description of the Left Ventricle in Segments

In order to describe more fully the manner of left ventricular contraction and its relation

Table 3

Planimetered Left Ventricular Cavity Areas

<table>
<thead>
<tr>
<th>No.</th>
<th>Pt</th>
<th>RAO (cm²)</th>
<th>LAO (cm²)</th>
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<td></td>
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<td>Syst. area</td>
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<td>39</td>
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<td>E.P.†</td>
<td>44</td>
<td>43</td>
</tr>
<tr>
<td>3</td>
<td>C.M.*</td>
<td>50</td>
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</tr>
<tr>
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<td>A.K.</td>
<td>51</td>
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<td>H.M.</td>
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<td>48</td>
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<tr>
<td>6</td>
<td>A.S.†</td>
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<td>47</td>
</tr>
<tr>
<td>7</td>
<td>J.W.*</td>
<td>53</td>
<td>48</td>
</tr>
<tr>
<td>8</td>
<td>J.D.†</td>
<td>63</td>
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<td>G.P.</td>
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<td>H.W.</td>
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</tr>
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<td>11</td>
<td>A.F.†</td>
<td>75</td>
<td>71</td>
</tr>
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</table>

Abbreviations: RAO = right anterior oblique; LAO = left anterior oblique; Δ = diastolic minus systolic area; % = Δ/diastolic area × 100.

*Operated, survived.
†Operated, died.
to impaired perfusion, we have chosen to
describe the left ventricle in six segments
determined by the usual normal coronary
perfusion pattern.\textsuperscript{5} In figure 2, the basis for
this subdivision is shown with the course of
the coronary arteries as they most often relate
to the ventricular cavity. The left anterior
descending coronary artery and its branches
perfuse the anterolateral, apical, and antero-
septal segments. The posterior descending
branch of the right coronary artery usually
supplies the inferior segment. Posteromedial
segments may be supplied by posterior left
ventricular branches of the right coronary
artery or distal branches of the circumflex.
Finally, the posterolateral segment is perfused
most commonly by the circumflex marginal
artery. Some patients deviate from this
scheme since the precise location of the water-
shed between feeding vessels may vary and since in 10% of cases the posterior
descending artery is a branch of the left
coronary artery.\textsuperscript{5}

Angiographically demonstrated high-grade
stenosis of coronary arteries supplying a
segment may not produce a corresponding
reduction in contraction.\textsuperscript{3} However, when left
ventricular dysfunction is caused by coronary
artery disease, poor contraction of a segment
usually predicts obstruction of the correspond-
ing coronary artery. Contraction and perfu-
sion of each segment of these 11 patients is
shown in table 4. The order is again that of
increasing left ventricular area. Where numer-
ators are missing no left anterior oblique
ventriculogram was performed. A question
mark indicates insufficient clarity of coronary
opacification. Correspondence between seg-
mental contraction and perfusion was good
with an occasional exception, and in general
increasing cavity size was associated with
increasingly severe segmental disease. Patient
E.P. was the exception. In spite of segmental
disease as severe as any other patient, his
diastolic left ventricular area was normal. The
severity of his disease, however, was reflected
by the remarkably small change in cavity size
during ejection (table 3).

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Subdivision of the left ventricular angiographic silhouette into six segments each with its
typical coronary perfusion. The segments are named antero-lateral, apical, inferior, postero-
medial, antero-septal, and postero-lateral. Abbreviations: RAO and LAO = right and left
anterior oblique; RCA and LCA = right and left coronary arteries; LAD = left anterior de-
scending; DIAG = diagonal; C and CM = circumflex and circumflex marginal; Post. LV =
posterior left ventricular branches; and Post. Desc. = posterior descending coronary artery.}
\end{figure}

\textit{Circulation}, Volume XLV, April 1972
Table 4

<table>
<thead>
<tr>
<th>No.</th>
<th>Pt</th>
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<th>AS</th>
<th>I</th>
<th>PM</th>
<th>PL</th>
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<td>2/4</td>
<td>2/2</td>
<td>2/7</td>
<td>1/1?</td>
</tr>
<tr>
<td>2</td>
<td>E.P.‡</td>
<td>4/5</td>
<td>5/5</td>
<td>/5</td>
<td>3/4</td>
<td>3/3</td>
<td>/3</td>
</tr>
<tr>
<td>3</td>
<td>C.M.†</td>
<td>4/3</td>
<td>4/3</td>
<td>/3</td>
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<td>2/2</td>
<td>/1</td>
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<tr>
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<td>3/3</td>
<td>4/4</td>
<td>3/4</td>
<td>3/?</td>
<td>1/3</td>
<td>1/2</td>
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<td>/3</td>
</tr>
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<td>/5</td>
<td>3/1</td>
<td>3/4</td>
<td>/3</td>
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</table>

Abbreviations: PL = posterolateral; PM = posteromedial; others as in table 1.
*The numerator represents contraction. Scale: 1 = normal; 2 = mildly reduced; 3 = moderately reduced; 4 = absent; 5 = paradoxical. The denominator represents perfusion from the corresponding coronary artery, described in figure 2. Scale: 1 = normal; 2 = moderate obstruction (50-75%); 3 = severe obstruction (> 75%); 4 = occlusion with filling by collateral; 5 = occlusion without collateral.
†Operated, survived.
‡Operated, died.

Clinical Course following Angiography

Four patients died without operation, one following angiography (A.K.), another from a recurrent arrhythmia (H.W.), a third was considered inoperable (G.P.), and the fourth refused operation (H.M.).

Seven underwent aortocoronary vein bypass grafting, three with associated infarctectomy. There were three survivors. Two of these survivors had excellent results from right coronary artery and left anterior descending vein grafts (A.W., C.M.). Both have been discharged without cardiomegaly and are essentially asymptomatic. The third recovered following a large anterior infarctectomy and right coronary artery bypass but died 3 months after discharge with congestive failure (J.W.). The others died in the intraoperative or postoperative period (E.P., J.D., A.F.).

Postmortem Correlations

Postmortem examinations were performed on six of these patients. It was found that infarction could be present in areas where the angiogram appeared to show some remaining perfusion and that failure of a segment to contract did not necessarily indicate necrosis. The combination of akinesis and avascularity, however, was a highly reliable predictor of necrosis. All four cases (E.P., G.P., J.D., and H.W.) in whom the angiographic extent of avascularity and akinesis exceeded an estimated 40% of the left ventricular mass showed greater than 40% left ventricular necrosis. This group represents the highest operative risk and may not be salvageable with present surgical techniques short of cardiac replacement.

Discussion

Patients presenting with cardiogenic shock secondary to acute myocardial infarction (CS-MI) who are treated with IABP fall generally into four groups: (1) There are those who recover with IABP alone. Some recover quickly and are discharged without cardiomegaly or significant symptoms. Such patients are presumably in a vicious cycle of hypotension and myocardial depression with relatively small areas of necrosis. Others recover more slowly but do achieve stable independence from IABP over several days of support. The cardiomegaly and congestion that follow bespeak a larger infarct size. (2) A second group shows dependence on IABP after recovery from cardiogenic shock with signs of myocardial ischemia when support is withdrawn. After coronary artery bypass surgery these patients may return to active life without cardiomegaly. The final infarct size is presumably small. (3) A third group also recovers from shock but remains dependent on IABP. Myocardial ischemia during interruption of circulatory support is not clinically evident. The infarct is usually large. (4) Finally, a few patients have sustained such extensive infarction that even IABP and catecholamines will not relieve shock. Death for these patients may be inevitable.

The majority of patients with CS-MI who are assisted with IABP after variable delay are in groups 2 and 3. All patients described in this report were so grouped except one who remained in shock despite IABP (group 4, Circulation, Volume XLV, April 1972
J.D.). It is important to try to distinguish among these groups because of the implications regarding infarct size. The infarct size in turn will probably be the most significant single factor limiting the success of surgical revascularization.

Three of the seven patients who underwent operation, including both long-term survivors, were in group 2. Both of the surviving patients complained of chest discomfort when IABP was interrupted briefly prior to operation, and both are now free of angina without X-ray evidence of cardiomegaly. The completeness of recovery in these cases suggests a relatively small infarct size. The third patient in this group became restless and complained of nausea during a 10-min period off IABP. He died 11 days after operation with pneumonia and a clotted graft presumably leading to recurrent ventricular ischemia. At postmortem examination only 25% necrosis of the left ventricle was found.

The angiographic features characterizing this favorable group were the lack of any avascular segments and in two instances some persistent contraction of the large anterolateral segment in spite of anterior infarction. This in turn was associated with better left ventricular ejection and no more than moderate ventricular dilatation (table 3, A.W., C.M., A.S.). A fourth patient fits angiographically into this group, but no postmortem confirmation is available (H.M.).

The distribution of coronary artery disease did not distinguish among groups. The left anterior descending coronary artery was always involved (with invariable apex dysfunction) and, in all but one patient, another major coronary artery was severely stenotic, most commonly the right. The severity of vascular obstruction, however, appeared critical. All three patients who underwent operation in spite of proximal anterior descending occlusion without collateral circulation (avascular anterolateral, apical, and anteroseptal segments) died within 3 hours of the procedure. Necrosis of over 40% of the left ventricle was the probable cause of death. The extent of necrosis was probably also related to consistently high-grade obstruction of the adjacent circumflex marginal artery.

Finally, some patients cannot be precisely classified preoperatively. Patient J.W. showed only moderate ventricular dilatation, residual perfusion of all segments either directly or by collateral, and preservation of contraction in the anteroseptal segment. Yet, massive anterolateral necrosis was found at operation that eventually led to his death in spite of successful acute surgery. Perhaps a retrospective clue in evaluating his subsequent course was the presence of obstruction in the circumflex marginal artery adjacent to the fresh anterior infarction.

Early application of IABP to patients with myocardial infarction and documented shock will probably increase the ratio of ischemic to necrotic tissue and, while the clinical and hemodynamic state is stable, prevent progression of necrosis. During this interval (usually the first 24–48 hours) angiography may be performed with reasonable risk. Analysis of coronary perfusion and ventricular contraction segment by segment can identify certain patients in whom ventricular necrosis is not massive. Such patients may greatly benefit from emergency coronary surgery.

Acknowledgment

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References


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