Intraaortic Balloon Pumping for Ventricular Septal Defect or Mitral Regurgitation Complicating Acute Myocardial Infarction

By Herman K. Gold, M.D., Robert C. Leinbach, M.D., Charles A. Sanders, M.D., Mortimer J. Buckley, M.D., Eldred D. Mundth, M.D., and W. Gerald Austen, M.D.

SUMMARY
The intraaortic balloon pump (IABP) has been employed in the management of five patients with ventricular septal rupture (VSD) and six patients with acute mitral regurgitation (AMR) following myocardial infarction. All patients were in cardiogenic shock which responded poorly to medical therapy including pressor and inotropic agents. IABP resulted in significant clinical and hemodynamic improvement in all cases.

In patients with VSD, IABP produced a fall in wedge (PCW) pressure from 17 ± 4 (sd) to 13 ± 4 mm Hg (P < 0.01) while mean arterial pressure increased from 88 to 73 mm Hg. Systemic A-V O₂ difference fell from 9.7 ± 2.4 to 8.1 ± 2.4 vol % (P < 0.05) while pulmonary A-V O₂ difference was unchanged. Thus the pulmonic/systemic flow ratio (P/S) declined in all patients. In patients with AMR, PCW fell from 25 ± 4 to 20 ± 4 mm Hg (P < 0.02) with a significant diminution in "V"-wave amplitude. Cardiac output (CO) rose from 3.1 ± 0.9 to 3.7 ± 1.0 liters/min (P < 0.01). All patients underwent coronary angiography without complication in preparation for emergency surgery.

IABP reduces AMR following acute myocardial infarction and reduces the P/S in VSD by a selective augmentation of systemic CO. Such direct therapy acutely stabilizes these severely ill patients. Detailed angiography may then be performed safely.

Additional Indexing Words:
Mechanical circulatory assistance
Left-to-right shunting

A C U T E M I T R A L regurgitation or rupture of the interventricular septum following myocardial infarction may lead to rapid clinical deterioration with pulmonary edema, hypotension, and a high early mortality.¹⁻² Pressor agents which increase afterload can aggravate mitral regurgitation and will increase left-to-right shunting. Inotropic agents can increase systemic output but at

the possible cost of increased myocardial ischemia.⁴ In contrast, counterpulsation can decrease ischemia by raising coronary perfusion pressure and will reduce afterload.⁵⁻¹² This report summarizes our experience in attempting to stabilize critically ill patients with these mechanical complications utilizing the intraaortic balloon pump (IABP).

Material and Methods
Hemodynamic studies were performed in 11 patients admitted to the Myocardial Infarction Research Unit Intensive Study Area. In all patients, acute myocardial infarction was documented by a typical history, diagnostic electrocardiographic changes, and serum enzyme studies. Five patients had ventricular septal defect (VSD) 1–9 days after acute infarction. In two cases the infarction was anterior and in three inferior. Six patients developed acute mitral regurgitation (AMR) 1–14 days after infarction. In three patients the infarction was anterior and in three inferior (table 1).

At the time of study all patients with VSD showed vasoconstriction, systemic hypotension, and low central venous oxygen saturation. Patients with AMR all
demonstrated pulmonary edema, hypotension, and low cardiac output. Both groups responded poorly to medical therapy consisting of digitalis, diuretics, and oxygen, and all but one were receiving catecholamine infusion at the time of study.

Pulmonary artery and pulmonary capillary wedge (PCW) pressures were monitored with the Swan-Ganz flow-directed catheter and arterial pressures through an indwelling radial artery cannula. Pulmonary-to-systemic (P/S) flow ratio was determined by oxygen saturation. Systemic venous samples were drawn from the superior vena cava. In patients with AMR, cardiac output was estimated by the dye-dilution technic with indocyanine green injected into the pulmonary artery and sampled from the radial artery.

In all patients an IABP “on-off” study was performed daily. Systemic and pulmonary artery pressures, PCW pressure, and electrocardiogram were recorded followed by duplicate determinations of cardiac output and oxygen saturation. IABP was then interrupted; 3–10 min were allowed to elapse during which pressures were monitored continuously. Before resuming IABP, cardiac output and oxygen saturation determinations were repeated. IABP was then resumed and after 5 min a third set of measurements were taken.

In three patients (two VSD, one AMR) angiotensin was then infused in low dose (1.25 μg/min) during continuous IABP to abolish the effect of IABP on afterload. When systolic arterial pressure had been matched to the level prior to the institution of pumping, a fourth set of measurements was obtained.

**Table 1**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Date of infarct</th>
<th>Site of infarct</th>
<th>Date murmur appeared</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.B.</td>
<td>62</td>
<td>M</td>
<td>5/6</td>
<td>Inferior</td>
<td>5/11</td>
<td>MR; ruptured posterior papillary muscle</td>
</tr>
<tr>
<td>A.S.</td>
<td>54</td>
<td>M</td>
<td>8/31</td>
<td>Anterior</td>
<td>9/8</td>
<td>MR; infarction anterior and posterior papillary muscle</td>
</tr>
<tr>
<td>R.L.</td>
<td>45</td>
<td>M</td>
<td>6/24</td>
<td>Inferior</td>
<td>6/26</td>
<td>MR; infarction posterior papillary muscle</td>
</tr>
<tr>
<td>M.A.</td>
<td>39</td>
<td>F</td>
<td>3/5</td>
<td>Anterior</td>
<td>3/5</td>
<td>MR; infarction anterior and posterior papillary muscle</td>
</tr>
<tr>
<td>W.J.</td>
<td>56</td>
<td>M</td>
<td>11/6</td>
<td>Inferior</td>
<td>11/9</td>
<td>MR; ruptured posterior papillary muscle</td>
</tr>
<tr>
<td>H.A.</td>
<td>62</td>
<td>M</td>
<td>10/6</td>
<td>Inferior</td>
<td>10/8</td>
<td>VSD</td>
</tr>
<tr>
<td>J.S.</td>
<td>65</td>
<td>M</td>
<td>4/5</td>
<td>Inferior</td>
<td>4/10</td>
<td>VSD</td>
</tr>
<tr>
<td>A.J.</td>
<td>63</td>
<td>M</td>
<td>6/30</td>
<td>Antero-septal</td>
<td>7/3</td>
<td>VSD</td>
</tr>
<tr>
<td>B.A.</td>
<td>68</td>
<td>M</td>
<td>9/15</td>
<td>Inferior</td>
<td>9/26</td>
<td>VSD</td>
</tr>
<tr>
<td>J.G.</td>
<td>54</td>
<td>F</td>
<td>3/14</td>
<td>Anterior</td>
<td>3/23</td>
<td>VSD</td>
</tr>
<tr>
<td>M.P.</td>
<td>63</td>
<td>F</td>
<td>6/17</td>
<td>Antero-septal</td>
<td>6/22</td>
<td>VSD</td>
</tr>
</tbody>
</table>

Abbreviations: MR = mitral regurgitation; VSD = ventricular-septal defect.

**Figure 1**

*The hemodynamic response to IABP in five patients with acute ventricular septal rupture. During IABP, the fall in pulmonary wedge (PCW) pressure is accompanied by a small but significant rise in mean arterial pressure. Vertical bars represent 1 sd.*

**Figure 2**

*The maximal effect of IABP on systemic and pulmonary A-V oxygen difference after ventricular septal rupture. During IABP the pulmonary/systemic (P/S) flow ratio fell from 5.2/1 to 4.1/1. Asterisks represent the mean.*

*Circulation, Volume XLVII, June 1973*
heart rate (fig. 1). Systemic A-V O₂ difference fell from an average of 9.7 ± 2.4 (sd) to 8.1 ± 2.4 vol % (P < 0.05) while pulmonary A-V O₂ difference remained low and unchanged. Thus the mean P/S flow ratio fell from 5.1/1 to 4.2/1 (P < 0.05) secondary to a selective increase in systemic output (fig. 2).

The effect on the intracardiac shunt was produced by a reduction in left ventricular afterload. When angiotensin was infused during IABP and systolic arterial pressure was matched to the level present prior to the institution of balloon pumping, the P/S flow ratio returned to prepumping levels in spite of a marked increase in coronary perfusion pressure (fig. 3).

In the patients with AMR, the mean PCW pressure fell from 25 to 20 mm Hg (P < 0.02) with initiation of IABP. Mean arterial pressure remained unchanged while the heart rate fell from 99 to 95 beats/min (P < 0.02). This was associated with an average rise in cardiac output from 3.1 to 3.7 liters/min (P < 0.01, fig. 4). Interruption of IABP was accompanied by a rise in PCW pressure and an increase in the amplitude of the systolic regurgitant “V” waves (fig. 5). Angiotensin was infused at 1.25 µg/min during IABP in one patient, and cardiac output, PCW pressure, and regurgitant “V” waves all returned to prepumping levels.

All patients in both groups improved hemodynamically and showed maximum stabilization by 24 hours. Nevertheless, at this point all continued to show balloon dependence with inadequate circulatory dynamics during test interruption of IABP. Two patients with VSD and three with AMR were continued on IABP for a longer period prior to angiography. All showed hemodynamic deterioration despite continued circulatory assistance.

The angiograms revealed extensive coronary disease in both groups. All patients with VSD showed high-grade proximal left anterior descending coronary obstruction. Five of six showed high-grade proximal obstruction of the right coronary artery, and two showed three-vessel disease. The left ventriculograms in the right anterior oblique position were partially obscured by the left-to-right shunt and opacification of the right ventricle. When segmental contraction could be appraised, abnormalities were found similar to those reported elsewhere. All patients with AMR showed both high-grade obstruction of the proximal left anterior descending and right coronary arteries. Two showed three-vessel disease. The angiographic severity of mitral regurgitation was considered
The effect of IABP on acute mitral regurgitation. At the arrow the balloon pump is turned off. The increase in systolic arterial pressure is accompanied by a prompt rise in PCW pressure and an increase in the amplitude of systolic regurgitant "V" waves.

Emergency surgery was performed in four patients with VSD and five with AMR. Two patients with anterior myocardial infarction and VSD who received IABP within 24 hours of septal rupture survived. One patient with AMR secondary to papillary muscle survived.

Discussion

The onset of circulatory collapse associated with abrupt appearance of a systolic murmur after acute myocardial infarction presents a diagnostic and therapeutic problem. In most cases the diagnosis is mitral regurgitation secondary to papillary muscle dysfunction or rupture or ventricular septal defect. The diagnosis can be established at the bedside with the passage of a Swan-Ganz flow-directed catheter. Oxygen samples from the right atrium, pulmonary artery, and radial artery will identify the presence of a VSD and the inscription of the PCW wave form will confirm mitral regurgitation.

Medical therapy for these complications often produces only a limited effect. In VSD, infusion of an inotropic agent may increase systemic output. However, this intervention is associated with concomitant increase in left-to-right shunting without change in the P/S flow ratio, thereby resulting in a marked increase in left ventricular work. Agents which raise arterial pressure by increasing afterload augment pulmonary flow, increase left-to-right shunting, and may produce a critically low-output state. In AMR the degree of MR is

Figure 5

radial artery and left ventricular (LV) pressures prior to angiography in a patient with ventricular septal rupture. During IABP the peak LV pressure is 55 mm Hg while the peak diastolic pressure is maintained at 75 mm Hg. The patient is in sinus tachycardia. Electrocardiographic (ECG) artifacts are timing indicators.
aggravated by pressor agents. Inotropic agents
which may increase systemic output do so at
the expense of increased myocardial oxygen demand
and may further impair myocardial contraction in
ischemic areas.16

In both situations, the desired physiologic re-
response possibly could be achieved by vasodilator
therapy. Reduction in systemic resistance in ventric-
ular septal rupture would favor systemic flow and
decrease the left-to-right shunt. Moreover in
patients with papillary muscle dysfunction or
rupture, reduction in systemic resistance would
favor forward flow and reduce the degree of mitral
regurgitation. However, in these patients whose
clinical condition is characterized by cardiogenic
shock, any further reduction in systemic and
coronary perfusion pressure is hazardous.

IABP can selectively reduce systemic resistance
during left ventricular ejection thus producing the
beneficial effects of a vasodilator without a
reduction in mean arterial pressure. This is
accompanied by an increase in coronary perfusion
pressure which can reduced ischemic dysfunction.

This study confirms the effects of IABP in
patients in whom mechanical lesions have resulted
in cardiogenic shock. Although IABP produced
clinical and hemodynamic improvement in all
patients, a completely satisfactory correction of the
impaired circulatory dynamics could not be ob-
tained. The peak improvement occurred within the
first 24 hours of pumping, and in no patient did
prolonged pumping produce further benefit. It was
not possible to maintain patients into a chronic
phase and thus avoid the high risk of emergency
surgery.17–20

IABP does produce a sufficient level of hemody-
namic stability to permit both selective coronary
and left ventricular cineangiography in these
critically ill patients. The studies can be performed
with low risk. In addition to demonstrating the
mechanical abnormality, complete angiography also
uncovers multiple significant proximal coronary
artery obstructions amenable to surgical revascular-
zation. The angiographic results in these patients
suggest that shock is the result of a mechanical
lesion superimposed upon an ischemic and infarct-
ed ventricle. In this setting the greatest chance for a
therapeutic success lies in the early application of
circulatory assistance and the accurate identification
of both mechanical and coronary artery lesions
responsible.

Acknowledgment
We are indebted to Mr. John P. Hinckley, Jr., Mr. John F.
Drake, and Mr. Marc S. Newman for skilled technical
assistance, to the MIHU Nursing Staff, and to Mrs. J. A.
Kowalczyk-Beckworth and Mrs. B. Levin for their help in
the preparation of this manuscript.

References
1. DeBusk RF, Harrison DC: The clinical spectrum of
papillary muscle disease. New Eng J Med 251: 1458,
1969
2. Sanders RF, Neuberger KT, Ravin A: Rupture of
posterior papillary muscles: Occurrence of rupture of
posterior muscle in posterior myocardial infarction.
Dis Chest 31: 316, 1957
3. Sanders RF, Kersh WH, Blount SG: Perforation of
the interventricular septum complicating myocardial
infarctions. Amer Heart J 51: 736, 1966
4. Maroko PR, Kjekshus JK Sobel BE, Watanabe T,
Covell JW, Ross J, Braunwald E: Factors influencing
infarct size following experimental coronary occlusion.
Circulation 46: 465, 1971
5. Moulopoulos ST, Topas S, Kolff WJ: Diastolic
balloon pumping (with carbon dioxide) in the aorta:
A mechanical assistance to the failing circulation.
Amer Heart J 63: 689, 1962
SJ, Free PS, Butner AN: Mechanical intra-aortic
cardiac assistance in cardiogenic shock. Arch Surg
(Chicago) 97: 1000, 1968
7. Buckley MJ, Leinbach RC, Kastor JA, Labad CA,
Austen WG: Hemodynamic evaluation of intra-
aortic balloon pumping in man. Circulation 41 (suppl
II): II-130, 1970
8. Mueller H, Ayres SM, Conklin EF, Giannelli S,
Mazzara JT, Grace WT, Nealon TF: The effects of
intra-aortic counterpulsation on cardiac performance
and metabolism in shock associated with acute
9. Powell WJ, Daggett WM, Magro AR, Bianco JA,
Buckley MJ, Sanders CA, Kantrowitz AR, Austen
WG: Effects of intra-aortic balloon counterpulsation
on cardiac performance, oxygen consumption, and
10. Leinbach RC, Buckley MJ, Austen WG, Petscheh
HE, Kantrowitz AR, Sanders CA: Effects of intra-
aortic balloon pumping on coronary flow and
metabolism in man. Circulation 43 (suppl I): I-77,
1971
11. Dunkman WB, Leinbach RC, Buckley MJ, Mundth
ED, Kantrowitz AR, Austen WG, Sanders CA:
Clinical and hemodynamic results of intraaortic
balloon pumping and surgery for cardiogenic shock.
Circulation 46: 465, 1972
12. Maroko PR, Bernstein EF, Libby P, Delara GA,
Covell JW, Ross J Jr, Braunwald E: Effects of
intra-aortic balloon counterpulsation on the severity
of myocardial ischemic injury following acute
coronary occlusion: Counterpulsation and myocardial
injury. Circulation 45: 1150, 1972

Circulation, Volume XLVII, June 1973
Intraaortic Balloon Pumping for Ventricular Septal Defect or Mitral Regurgitation Complicating Acute Myocardial Infarction

HERMAN K. GOLD, ROBERT C. LEINBACH, CHARLES A. SANDERS, MORTIMER J. BUCKLEY, ELDRED D. MUNDTH and W. GERALD AUSTEN

Circulation. 1973;47:1191-1196
doi: 10.1161/01.CIR.47.6.1191

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 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/47/6/1191

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/