Hemodynamic Profile in Acute Myocardial Infarction

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Hemodynamic and clinical evaluation of 50 patients with acute myocardial infarction were performed. A majority of patients had depressed left ventricular function, but the level of depression was not uniform and a wide spectrum existed. Left ventricular function curves appeared to demonstrate a peak or plateau with a pulmonary arterial end-diastolic pressure (PAEDP) from 17 to 19 mmHg. There was negative correlation between stroke volume index and total peripheral vascular resistance (TPRI). Mean arterial blood pressure (MABP), TPRI, PAEDP were decreased significantly and cardiac index (CI) was markedly increased after administration of nifedipine. Seventeen patients had congestive heart failure. These patients showed a marked increase in PAEDP and a wide degree of variability in CI and left ventricular stroke work index (LVSWI). Sixteen of 17 patients with congestive heart failure showed a PAEDP greater than 18 mmHg. Six patients developed cardiogenic shock. In these patients CI and LVSWI were markedly reduced. Two patients showed normal PAEDP. Three patients with septic shock showed a marked increase in CI and a decrease in TPRI. Hemodynamic subsets established by a PAEDP of 18 mmHg and a mean right atrial pressure (MRAP) of 100 mmH2O was beneficial to evaluate the right ventricular function. Four patients with the findings of elevated MRAP out of proportion to PAEDP were diagnosed as right ventricular infarction. Three of them showed normal PAEDP. Two patients who showed hypotension and low CI died with shock.

It is concluded that hemodynamic evaluation of patients with acute myocardial infarction is useful in understanding the extent of altered cardiac function, classifying the severity of the clinical state, measuring the response to various treatments and predicting the prognosis of the patient.

In recent years the mortality rate from arrhythmias in acute myocardial infarction has been markedly reduced, but mortality from the complications of pump failure has not been significantly altered. Pump failure now stands as the primary cause of death in acute myocardial infarction. Although overt signs of pump failure are usually clinically obvious, its early signs may be subtle and the timing of therapy difficult.

Monitoring of the hemodynamic measurement in acute myocardial infarction can reflect early changes in cardiac performance. Furthermore, the management of pump failure by pharmacologic intervention and the availability of mechanical cardiac assist devices also requires hemodynamic monitoring.1,2

The development of a bedside method for hemodynamic monitoring has made it possible to evaluate cardiac performance in patients with acute myocardial infarction.3

Key Words:
Acute myocardial infarction
Hemodynamic monitoring
Left ventricular function
Right ventricular function
Patient prognosis

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Diagnostic and Therapeutic Procedures in Acute Myocardial Infarction

Fig. 1. Relation of LVSWI to PAEDP at the time of admission. stippled area = the normal range

Fig. 2. Relation between PAEDP and CI at the time of admission. The dotted lines are placed at the levels of 18 mmHg for PAEDP and 2.2 liters per minute per square meter for CI. open circle = uncomplicated myocardial infarction, solid circle = congestive heart failure, triangle = cardiogenic shock, RV = right ventricular infarction

Fig. 3. Left ventricular function curves constructed by altering preload with the use of dextran and/or furosemide. stippled area = the optimal level of PAEDP

SUBJECTS

Fifty patients (47 men and 3 women), admitted to the Kanazawa Medical University Hospital Coronary Care Unit with acute myocardial infarction within 72 hours of the onset of symptom, were included in this study. Their ages ranged from 35 to 78 years (mean 62 years). Twenty-six patients had anterior infarction, 12 inferior infarction and 12 antero-inferior infarction. The diagnosis of acute myocardial infarction was based on the presence of at least two of the following three criteria: (1) a typical history, (2) electrocardiographic changes, and (3) prompt rise and fall of the serum creatine phosphokinase, lactic acid dehydrogenase and glutamic-oxalacetic transaminase.

METHODS

After clinical assessment, electrocardiography and chest radiograph, right pulmonary arterial and right atrial pressure were continuously monitored through a Swan-Ganz balloon-tipped triple lumen catheter (Edwards Laboratories), SIMENS pressure transducers and SIMENS mingograf 34. Cardiac output (CO) was measured by thermodilution techniques with the use of ice cold water. Computations of CO were performed by a bedside computer (Model 9529A cardiac output computer, Edwards Laboratories).

Fig. 4. Relation between TPRI and SVI. solid circle = cardiogenic shock

Fig. 5. Hemodynamic effects of nifedipine in 10 patients.

Arterial blood pressure was measured by cuff readings. Mean arterial blood pressure (MABP) was estimated from the following formula; 
MABP = diastolic pressure + (systolic pressure - diastolic pressure)/3. Pulmonary arterial end-diastolic pressure (PAEDP) was used to determine left ventricular filling pressure. The following parameters of cardiac performance were
Fig. 6. Hemodynamic values in patients with congestive heart failure. open circle = patient with the improvement of congestive heart failure during convalescence, solid circle = in-hospital nonsurvivor, triangle = patient in whom remained impaired left ventricular function, stippled area = the values in patients with uncomplicated acute myocardial infarction (mean ± S.D.)

calculated: cardiac index (Cl) = CO/body surface area (BSA) (L/min/m²); stroke volume index (SVI) = CI/HR × 100 (ml/beat/m²), where HR is heart rate (beats/min); left ventricular stroke work index (LVSWI) = SVI × (MABP - PAEDP) × 0.136 (g.m/m²); right ventricular stroke work index (RVSWI) = SVI × (MPAP - MRAP) × 0.136 (g.m/m²), where MPAP is mean pulmonary arterial pressure and MRAP is mean right atrial pressure; and total peripheral resist-

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Hemodynamic values in patients with cardiogenic shock or septic shock. Solid symbol = nonsurvivor, open symbol = survivor, circle = patient with anterior infarction and cardiogenic shock, triangle = patient with inferior infarction and cardiogenic shock, square = patient with septic shock, stippled area = the values in patients without cardiogenic shock (mean ± S.D.)

\[ \text{TPRI} = \frac{(MABP - MRAP)}{CI} \times 80 \] (dyne·sec·cm\(^{-3}\)·m\(^2\)).

Serial hemodynamic measurements have been made over period of 12 hours to several days in all patients. In 21 patients, the left and right ventricular function curves were constructed by altering preload with the use of low molecular weight dextran and/or furosemide. In 10 pa-
TABLE 1 ACUTE MYOCARDIAL INFARCTION ASSOCIATED WITH SEPTIC SHOCK: CLINICAL FEATURE

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>Predisposing Factor</th>
<th>Causative Organism</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>46</td>
<td>M</td>
<td>Cholelithiasis, Cholecystitis</td>
<td>E. coli</td>
<td>Dead</td>
</tr>
<tr>
<td>2.</td>
<td>48</td>
<td>M</td>
<td>Cholelithiasis, Cholecystitis</td>
<td>Enterobacter</td>
<td>Cured</td>
</tr>
<tr>
<td>3.</td>
<td>47</td>
<td>M</td>
<td>?</td>
<td>Acinetobacter</td>
<td>Cured</td>
</tr>
</tbody>
</table>

patients cardiac performance was examined by altering afterload with the use of nifedipine.

Seventeen patients showed clinical sings of congestive heart failure. Congestive heart failure was diagnosed by auscultatory and radiographic pulmonary congestion. Six patients had signs of cardiogenic shock at the time of study. Cardiogenic shock was diagnosed by definition of the Myocardial Infarction Research Unit of the National Heart and Lung Institute.

RESULTS

Left Ventricular Function

Fig. 1 shows the relation between LVSWI and PAEDP. Ten patients showed normal LVSWI in relation to PAEDP. Only one patient showed increased LVSWI in relation to PAEDP. Thirty-eight patients showed reduced LVSWI in relation to PAEDP. Fig. 2 shows the relation between CI and PAEDP. There was a wide degree of variability in the relation between CI and PAEDP. CI ranged from 0.72 to 4.39 (L/min/m²) and PAEDP from 5 to 32 (mmHg). Based on the hemodynamic subsets defined by Forrester et al., 26 patients were classified in subset H-I, 14 patients in subset H-II, 2 patients in subset H-III and 8 patients in subset H-IV.

Influence of Preload

Fig. 3 shows left ventricular function curves in 21 patients. The function curves appeared to demonstrate a peak or plateau with a PAEDP from 17 to 19 mmHg. Further elevation in PAEDP beyond 20 mmHg produced descending limbs to the function curves in 3 patients.

Influence of Afterload

There was negative correlation between SVI and TPRI (Fig. 4). Hemodynamic response to nifedipine, which acted on systemic arteriolar beds as vasodilator, was examined in 10 patients. MABP, TPRI and PAEDP were decreased significantly, and CI was markedly increased (Fig. 5).

Congestive Heart Failure

One of 26 patients was considered to have congestive heart failure in subset H-I, 12 of 14 patients in subset H-II and 4 of 8 patients in subset H-IV (Fig. 2). The patients with congestive heart failure showed a marked increase in PAEDP, but there was wide variability in CI and SWI. Sixteen patients with congestive heart failure showed a PAEDP greater than 18 mmHg, MABP and TPRI in patients with congestive heart failure were slightly increased (Fig. 6).

Cardiogenic Shock

One of 26 patients developed cardiogenic shock in subset H-I, one of 2 patients in subset H-III and 4 of 8 patients in subset H-IV (Fig. 2). The patients with cardiogenic shock showed a marked decrease in CI and SWI. Two of 6 patients showed normal PAEDP (Fig. 7).

Septic Shock associated with Acute Myocardial Infarction

Three patients developed septic shock during the acute illness. Bacteriological and clinical date in these 3 patients are shown in Table I. In 3 patients complicated with septic shock CI was markedly increased and TPRI was decreased significantly (Fig. 7). After developing septic shock, one of 3 patients was reclassified from subset H-II to H-I (Fig. 8).

TABLE II  RIGHT VENTRICULAR INFARCTION: CLINICAL AND HEMODYNAMIC FEATURE

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Sex</th>
<th>ECG Findings</th>
<th>Art. BP mmHg</th>
<th>MRAP mmHg</th>
<th>Pul.BP mmHg</th>
<th>CI L/min/m²</th>
<th>RYSWI g·m/m²</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>35</td>
<td>M</td>
<td>IMI</td>
<td>108/80</td>
<td>12</td>
<td>24/12</td>
<td>2.31</td>
<td>3.2</td>
<td>Cured</td>
</tr>
<tr>
<td>2.</td>
<td>39</td>
<td>M</td>
<td>AMI</td>
<td>137/98</td>
<td>8</td>
<td>13/ 7</td>
<td>3.22</td>
<td>1.3</td>
<td>Cured</td>
</tr>
<tr>
<td>3.</td>
<td>51</td>
<td>M</td>
<td>AMI</td>
<td>92/70</td>
<td>14</td>
<td>20/13</td>
<td>1.94</td>
<td>0.9</td>
<td>Shock, Dead</td>
</tr>
<tr>
<td>4.</td>
<td>73</td>
<td>M</td>
<td>IMI (+ old AMI)</td>
<td>70/50</td>
<td>24</td>
<td>40/22</td>
<td>1.30</td>
<td>0.5</td>
<td>Shock, Dead</td>
</tr>
</tbody>
</table>

Abbreviations: IMI = inferior myocardial infarction, AMI = anterior myocardial infarction, Art.BP = arterial blood pressure, Pul.BP = pulmonary arterial blood pressure.
### TABLE III

<table>
<thead>
<tr>
<th>Subset</th>
<th>RV Function</th>
<th>LV Function</th>
<th>In-hospital Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Normal</td>
<td>↓ ↓</td>
<td>43%</td>
</tr>
<tr>
<td>B</td>
<td></td>
<td>↓</td>
<td>31%</td>
</tr>
<tr>
<td>C</td>
<td></td>
<td></td>
<td>10%</td>
</tr>
<tr>
<td>D</td>
<td>↓ ~ ↓ ↓</td>
<td>Normal</td>
<td>29%</td>
</tr>
</tbody>
</table>

Fig. 10. Mortality rates in hemodynamic subsets. Solid circle = the patient died of shock, asterisk = patient died of congestive heart failure, double asterisk = patient died of cardiac rupture, parentheses = in-hospital mortality rate

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**Right Ventricular Function**

Fig. 9 plots PAEDP versus MRAP. There was a wide degree of variability in relation between PAEDP and MRAP. Four hemodynamic subsets were defined by using a PAEDP of 18 mmHg and a MRAP of 100 mmHg. Seven patients were classified in subset A (PAEDP ≥ 18 mmHg, MRAP < 100 mmHg), 13 patients in subset B (PAEDP ≥ 18 mmHg, MRAP ≥ 100 mmHg), 21 patients in subset C (PAEDP < 18 mmHg, MRAP < 100 mmHg) and 7 patients in subset D (PAEDP < 18 mmHg, MRAP ≥ 100 mmHg). Patients in subset D showed a marked decrease in RVSWI compared with increased MRAP. Patients in subset C showed a decrease in RVSWI compared with normal MRAP. Patients in subset A and B showed a decrease in LVSWI compared with an increase in PAEDP. The studies in construction of right and left ventricular function curves by altering preload indicated that the left ventricular function in subset A, B and the right ventricular function in subset C, D were depressed (Table II).

**Right Ventricular Infarction**

In 4 patients, right ventricular filling pressure (MRAP) was equal to or greater than the simultaneously measured left ventricular filling pressure (PAEDP) (Fig. 9). These 4 patients were diagnosed clinically as right ventricular infarction. Two of these 4 patients were classified in subset H-I, one in subset H-III and one in subset H-IV (Fig. 2). Clinical and hemodynamic observations in these 4 patients are outlined in Table II. Two patients had electrocardiographic findings compatible with inferior wall infarction. Patient 4 had a previous myocardial infarction and high value for PAEDP (22 mmHg), but the PAEDP was no greater than MRAP. In the other 3 patients PAEDP were within the normal range. CI was reduced in 2 patients. In Patient 1 and 2 there was a depression in the right ventricular function curves and a normal slope in the left ventricular function curves, in Patient 4 a depression in bilateral ventricular function curves and in Patient 3 a marked depression in the right ventricular function curve associated with depressed left ventricular function curve and a descending limb. Two patients with hypotension died with shock.

**Patient Prognosis**

There was a 24 percent (12 of 50 patients) inhospital mortality rate in the total population. This mortality was not evenly distributed among the four hemodynamic subsets defined by Forrester et al. The mortality rate increased from 11.5 percent in subset H-I to 21.4 percent in H-II, 50 percent in H-III and 62.5 percent in H-IV. As with hypoperfusion (CI < 2.2 L/min/m²), there was a fourfold increase in mortality rate associated with depressed CI (15 percent in...
H-I and H-II versus 60 percent in H-III and H-IV). Causes of 12 deaths in patients with acute myocardial infarction were distributed as follows: 5 were due to cardiogenic shock; 3 were due to congestive heart failure; 3 were due to heart rupture and one was due to septic shock (Fig. 10).

The mortality rate in the four hemodynamic subsets established by PAEDP and MRAP is presented in Table II. It is remarkable that there was a high mortality rate (29 percent) in subset D.

**DISCUSSION**

Patients with acute myocardial infarction are generally evaluated by clinical history, physical examination and available laboratory studies. Recently, the application of invasive cardiac catheterization techniques to patients with acute myocardial infarction has produced useful physiologic data on the mechanical performance of the heart. These data have proved useful in understanding the extent of altered cardiac function, classifying the severity of the clinical state, measuring the response to various treatment and predicting the prognosis of the patient.

**Left Ventricular Function**

There is no doubt that clinical prognosis after acute myocardial infarction is determined by the degree of functional impairment of the left ventricle. Therefore, the hemodynamic evaluation of left ventricular function is clinically important.

The relation of LVSWI to PAEDP in our patients indicates that a majority of patients with acute myocardial infarction is determined by the ventricular function, but the level of depression is not uniform and a wide spectrum exists. On the other hand, approximately one half of the patients had normal level of left ventricular performance in the relation between PAEDP and CI. It may be possible that this discrepancy occurs since cardiac output in some patients with acute myocardial infarction is maintained by increased heart rate resulted from reflex sympathoadrenal activation.

The principal peripheral vascular determinants of cardiac function consist of two important mechanisms: systemic venoomotor change (preload) and systemic arterial resistance alteration (afterload).

The level of left ventricular filling pressure is a determinant of cardiac performance since it affects diastolic ventricular stretch. This pressure are of vital clinical importance in patients with acute myocardial infarction. If the left ventricular filling pressure is low, cardiac output may be inadequate. Conversely, if the left ventricular filling pressure is elevated, pulmonary congestion and edema will occur. Our investigation into the hemodynamic changes by altering preload indicates that the optimal level of PAEDP is between 17 mmHg and 19 mmHg. Furthermore, on the basis of the relation between the clinical signs of congestive heart failure and PAEDP, it was thought that the optimal level of PAEDP was 18 mmHg. From the standpoint of practical therapeutics, attempts should be made initially to optimize the level of left ventricular filling pressure.

Left ventricular afterload is determined by left ventricular volume and aortic impedance. The latter is regulated by the compliance of large arteries and total peripheral vascular resistance. Total peripheral vascular resistance was increased in a majority of our patients. It was thought that increased peripheral vascular resistance was caused by increased sympathetic tone and high level of circulating catecholamine in acute myocardial infarction. The relation between TPRI and SVI in our study indicates that increased peripheral vascular resistance results in decreased stroke volume. Increased peripheral vascular resistance causes further rise of myocardial oxygen consumption (MVO2). This rise of MVO2 leads to additional ischemia and continued impairment of pump function. In our observations of nifedipine, it is evident that afterload reduction reduces MVO2 and improves cardiac performance in acute myocardial infarction. On the other hand, it may be possible that afterload reduction results in lowering coronary perfusion pressure and the oxygen supply to the myocardium reduces. Therefore, it is difficult to optimize the level of arterial blood pressure clinically.

PAEDP was elevated (over 18 mmHg) in 16 of 17 patients who had the clinical signs of congestive heart failure. Conversely, only 2 of 27 clinically uncomplicated patients had a elevated PAEDP. These observations indicate that PAEDP provides a reliable objective measure of left ventricular failure and is, therefore, an excellent guide to therapy. A high PAEDP signifies not only depressed contractility, but also decreased distensibility of the left ventricle. A
number of patients with congestive heart failure had a normal cardiac output.\textsuperscript{1,3} It was thought that cardiac output in these patients was maintained by an increase in left ventricular filling pressure and in heart rate which represented compensatory mechanism. A high PAEDP and increased peripheral vascular resistance in congestive heart failure justify the use of vasodilating agents for the treatment of this clinical situation. It may be that LVSWI is the most important determinant of predicting the prognosis in congestive heart failure.

Although cardiac output was markedly reduced in our patients with cardiogenic shock, PAEDP was not always elevated.\textsuperscript{1,3,5} In 4 patients with a elevated PAEDP a marked reduction of cardiac output and left ventricular stroke work indicate severe impairment of left ventricular function. Two others with cardiogenic shock had electrocardiographic findings compatible with inferior wall infarction and showed bradycardia, a normal PAEDP and slightly reduced left ventricular stroke work. Based on these findings, it was thought that these 2 patients had right ventricular infarction. Thus, recognition of cardiogenic shock associated with right ventricular infarction is important because it implies specific therapy.\textsuperscript{8-10} It may be that an increase in peripheral vascular resistance, which might be expected as a compensatory response to a decrease in cardiac output, is not uniformly present in patients with cardiogenic shock.\textsuperscript{1,13,15,16} Whether this failure of peripheral vascular resistance to increase in some patients is an important factor in cardiogenic shock or merely represents a degree of intensity of sympathetic reflex cannot be determined. CI and LVSWI are important determinants of predicting the prognosis in cardiogenic shock.

Patients with acute myocardial infarction complicated by septic shock showed high cardiac output and low peripheral vascular resistance. These findings indicate that hemodynamic monitoring is useful in differentiating septic shock from cardiogenic shock in acute myocardial infarction and in management of this clinical situation.

**Right Ventricular Function**

For many years it was considered that the left and right ventricle in acute myocardial infarction functions in parallel fashion and that disparate performance of the two ventricles could not occur without pulmonary edema or circulatory collapse. However, in the recent studies separation of the levels of right and left ventricular performance was demonstrated experimentally and clinically.\textsuperscript{10,17} In our study the right and left ventricular function curves constructed by altering preload also confirm the existence of unilateral depression of either right or left ventricular performance, and indicate that hemodynamic classification on the basis of the relation between MRAP and PAEDP is clinically beneficial to evaluate the right ventricular function. Impaired right ventricular function in acute myocardial infarction can result from not only right ventricular infarction but also the strain on the right ventricle, namely, increased pulmonary arterial pressure.\textsuperscript{10} Right ventricular infarction is rarely recognized clinically and there are no agreed electrocardiographic criteria of this entity.\textsuperscript{8-10} The clinical diagnosis of right ventricular infarction has been limited to cardiac catheterization. The findings of elevated right atrial pressure out of proportion to left atrial (or pulmonary arterial end-diastolic) pressure has been suggested as the characteristic hemodynamic profile of right ventricular infarction.\textsuperscript{8,9} Based on our findings of the right and left ventricular function curves, it was thought that this hemodynamic profile was diagnostically sensitive. The hypotension and reduced cardiac output associated with right ventricular infarction could be attributed to inadequate left ventricular filling. In the patients with increased left ventricular filling pressure one should consider right ventricular infarction associated with extensive left ventricular infarction as a diagnostic possibility.\textsuperscript{10,18} A number of patients with right ventricular infarction are effectively treated with plasma volume expansion.\textsuperscript{8,10} Conversely, some patients with high left ventricular filling pressure and/or a descending limb of right ventricular function curves are treated with plasma volume depletion or vasodilating agents.\textsuperscript{10} Therefore, the right and left ventricular function curves should be utilized in selecting therapy.

**Hemodynamic Classification**

From our study it was thought that Forrester's hemodynamic subset was clinically useful in understanding the impairment of left ventricular function, selecting therapy and predicting the prognosis of the patient. However, these subset system cannot evaluate the right ventricular function. On the other hand, our hemodynamic subset system established by a
PAEDP of 18 mmHg and a MRAP of 100 mm H₂O is beneficial to evaluate right ventricular function, to demonstrate the separation of right and left ventricular function, and to select appropriate therapy.

REFERENCE

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