Short-term Survival after Acute Myocardial Infarction Predicted by Hemodynamic Parameters

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SUMMARY
In the acute phase of myocardial infarction, short-term prediction of the likelihood of survival helps the physician choose the appropriate therapy for individual patients. Of 122 patients admitted to the coronary care unit of the Thoraxcenter, University Hospital, Rotterdam, with an acute myocardial infarction, 16 died from pump failure. In these and the 106 survivors, the predictive value of peripheral systolic (SP) and diastolic (DP) blood pressure, pulmonary capillary wedge pressure (PCW), mixed venous oxygen saturation (MVO₂sat) in the pulmonary artery and heart rate (HR), both alone and in combination, was evaluated at the time of admission and 24 hours later.

When, at admission, (DP × MVO₂sat)/PCW exceeded 250%, 97/99 patients survived, whereas values below 250% were associated with death in 14/23. All other parameters, taken alone or in other combinations, showed less discriminatory power. The mean value of this index in survivors (549%) was statistically different (P < 0.001) from the mean value in nonsurvivors (183%). Twenty-four hours later all survivors with admission values lower than 250% had an improved index. Of the 14 nonsurvivors with admission values lower than 250%, seven had already died, and in seven others the index had decreased still further. Linear discriminant analysis showed that (0.024 SP − 0.217 PCW + 0.234 MVO₂sat) was the most powerful prognostic index at the time of admission; its time course did not provide a more effective prediction of ultimate fatality than (DP × MVO₂sat)/PCW.

Determination of (DP × MVO₂sat)/PCW in patients hospitalized for acute myocardial infarction provides a reliable prognosis for short-term patient survival. Its practical value in guiding patient management, more particularly for initiating mechanical circulatory assistance or for emergency surgery, must be further assessed.

Myocardial Infarction (MI) in man leads to death primarily by one of three mechanisms: primary ventricular fibrillation, heart rupture, and cardiogenic shock. Coronary care units (CCU) were initiated primarily to prevent mortality due to electrical instability of the myocardium,1,2 and have been extremely successful in this endeavor: primary ventricular arrhythmias are no longer a major cause of death in the CCU. Cardiac ruptures are notoriously unpredictable: the patient is usually well until his sudden demise,3 and even emergency surgery comes too late.4 Pump failure resulting from the loss of more than 40% of the muscle mass of the left ventricle5,6 may sometimes be reversed by timely institution of circulatory assist,7–8 and/or by emergency heart surgery.9,10 This last procedure, however, carries a definite risk itself11 and should be used only if pharmacological therapy is unlikely to prevent a fatal outcome. A further reduction of mortality will therefore be achieved only if choice of treatment in critically ill patients is related to the likelihood of short-term survival.

Cardiogenic shock in MI is characterized by a low systemic blood pressure, by sinus tachycardia, by high left ventricular filling pressures and by a low pulmonary artery oxygen saturation.12–14 Continuous bedside monitoring of pulmonary artery pressures and of mixed venous oxygen saturation has been possible since the advent of balloon-tipped flow-directed catheters,15–17 while rhythm monitoring and regular measurement of arterial blood pressure have long been standard procedures.

Any effective treatment for cardiogenic shock must be done very early in order to salvage as much viable myocardium as possible. The present study was therefore undertaken to investigate whether appropriate hemodynamic monitoring could predict impending fatality in acute MI. To this end a prospective study was undertaken involving 122 patients hospitalized for acute MI: 16 of these died in cardiogenic shock and 106 patients survived their stay in...
the CCU. All patients included had sustained an acute MI according to WHO criteria. In all 16 patients dying in cardiogenic shock, autopsy confirmed the clinical diagnosis of extensive myocardial necrosis.

Materials and Methods

Patients

The present study was carried out on 122 consecutive patients admitted to the CCU less than 24 hours after the first symptoms of their acute MI. Patients who subsequently died of cardiac rupture were excluded from this study.

Ninety-seven patients were male and 21 female; 11 men and 5 women died; the mean age of the survivors was 58.5 and of the nonsurvivors 63.9; the difference was not statistically significant, and there was a marked overlap for both groups.

There was no difference in admission delay (median: 4 hours) between survivors and nonsurvivors. Most patients were first seen by a general practitioner and received pain sedation with opiates and prophylactic lidocaine (100 mg i.v. or i.m.) before being sent to the hospital. At home bradycardia was treated with atropine (0.25–0.50 mg i.v.); no patient was given digoxis glycosides; furosemide 40 mg i.v. was administered for pulmonary edema; maintenance therapy prior to infarction was similar in both groups of patients.

After admission to the CCU, management was identical in all patients, except in cases of left ventricular failure where ouabain and furosemide were given, guided by electrocardiographic monitoring, diuresis, and left ventricular filling pressures. All patients received intravenous lidocaine (2 mg/min). Cardiogenic shock was treated with a variety of drugs.18

A typical history and an abnormal electrocardiogram were considered sufficient indication for hemodynamic monitoring; this was initiated within 30 minutes after arrival in the CCU. With the exception of lidocaine and oxygen, no medication was given during this period of time. When serial enzyme changes and the evolution of the electrocardiogram failed to confirm the admission diagnosis of MI, the patient was excluded from the study. No patient was on respiratory assistance.

Survival was assessed during the usual four to five day stay in the CCU; after discharge from the CCU, patients were followed until they went home two to three weeks after their acute MI.

Technique

From an antecubital vein, a 5F Swan-Ganz catheter was positioned in the pulmonary artery.18 Pulmonary capillary wedge pressure (PCW) was taken to reflect mean left ventricular filling pressure.13–17 Mixed venous oxygen saturation (MVO2sat) was determined on a 3 ml blood sample taken from the pulmonary artery and measured in vitro by hemoreflectometry (American Optical).19

Heart rate (HR) was counted from the electrocardiographic monitoring lead at the time of the hemodynamic measurements, systolic and diastolic blood pressure (SP and DP, respectively) were measured with a sphygmomanometer; in the presence of cardiogenic shock, systemic arterial pressures were measured by means of an indwelling arterial catheter connected to a Statham P23dB manometer mounted at the bedside. In six patients all systemic arterial pressures were thus measured invasively; in ten others early measurements were made with a cuff and arterial catheterization carried out only upon hemodynamic deterioration.

Measurements were performed at four-hourly intervals. Particular attention was paid to pulmonary artery pressure recordings. Whenever the quality of the tracing was technically inadequate this information was excluded from the study. In critically ill patients where pulmonary artery pressures were regarded as vitally important in guiding patient therapy, unreliable pressure tracings were considered an indication for replacing the Swan-Ganz catheter with a new one. This was not done in patients doing well hemodynamically.

Measurements were performed at least 15 minutes after a serious ventricular dysrhythmia. Oxygen administration was also discontinued during at least 15 minutes prior to oxygen saturation determinations.

Data Analysis

For this study a comparison was made between survivors and nonsurvivors by utilizing only data for HR (min−1), SP (mm Hg), DP (mm Hg), PCW (mm Hg) and MVO2sat (%), compiled on admission and 24 hours later, although many more data points were available. Various combinations of these parameters were also evaluated: DP/PCW, MVO2sat/PCW, (SP × MVO2sat)/PCW, (DP × MVO2sat)/PCW, and (DP × MVO2sat)/(PCW × HR). Student’s t-test was used to compare the two groups. P values exceeding 0.05 were considered to be statistically not significant (NS). In addition threshold values were determined below which survival was unlikely.

Linear discriminant function analysis was used to find out if any linear combination of two or three of the following parameters would provide a better discrimination: age, HR, SP, DP, PCW, and MVO2sat. The number of patients wrongly classified by any of these combinations were computed.

Results

Observations on Admission to the CCU

All measurements are given in detail in tables 1 and 2. Heart rate and diastolic blood pressure did not allow any discrimination between survivors and nonsurvivors. Mean SP was significantly higher in survivors than in nonsurvivors (P < 0.005), but overlap was such that measuring SP on admission predicted survival only when it was in excess of 150 mm Hg.

There was a significant difference (P < 0.001) in PCW between survivors and nonsurvivors (fig. 1), although there was a considerable overlap as eight out of 18 patients with a PCW > 20 mm Hg survived. No deaths occurred in the 66 patients with a PCW < 15 mm Hg at the time of admission (table 2).

Similarly, the difference in MVO2sat between survivors and nonsurvivors was significant (P < 0.001). When MVO2sat > 70%, 58 out of 58 patients survived, while in eight out of the nine with a MVO2sat < 55% there was a fatal outcome. When MVO2sat was between 55 and 70% at the time of admission death occurred in eight of 55 patients.

A combination of parameters improved the accuracy of prognosis. DP/PCW > 4.25 suggested sur-
Table 1

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Survivors (N = 106)</th>
<th>Nonsurvivors (N = 16)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>84.2 ± 18.2</td>
<td>90.8 ± 17.4</td>
<td>NS</td>
</tr>
<tr>
<td>SP (mm Hg)</td>
<td>134.7 ± 26.6</td>
<td>107.8 ± 23.9</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>DP (mm Hg)</td>
<td>84.9 ± 16.6</td>
<td>73.1 ± 16.1</td>
<td>NS</td>
</tr>
<tr>
<td>PCW (mm Hg)</td>
<td>13.0 ± 5.4</td>
<td>22.4 ± 6.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>MVO_{sat} (%)</td>
<td>68.1 ± 6.2</td>
<td>51.4 ± 12.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>0.024SP - 0.217PCW</td>
<td>8.47 ± 6.76</td>
<td>3.39 ± 0.99</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>(SP × MVO_{sat})/PCW (%)</td>
<td>910 ± 680</td>
<td>270 ± 128</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>(DP × MVO_{sat})/PCW (%)</td>
<td>549 ± 334</td>
<td>153 ± 90.0</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>(DP × MVO_{sat})/(PCW × HR) (%/min)</td>
<td>6.3 ± 3.79</td>
<td>2.07 ± 0.98</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: HR = heart rate; SP = systolic pressure; DP = diastolic pressure; PCW = pulmonary capillary wedge pressure; MVO_{sat} = mixed venous oxygen saturation; SD = standard deviation; NS = not significant.

vival in 94/95 observations, and MVO_{sat}/PCW greater than 3.75%/mm Hg predicted survival in 89/91. The same was true for (DP × MVO_{sat})/PCW greater than 250%, as survival took place in 97/99, and (SP × MVO_{sat})/PCW > 400%, where survival occurred in 94/96. When all four parameters were combined, (DP × MVO_{sat})/(PCW × HR) > 3.2%/min correctly predicted survival in 96/97.

Linear discriminant analysis showed that the most powerful combination of parameters was 0.024SP - 0.217 PCW + 0.234 MVO_{sat} (tables 1 and 2), which predicted survival in 96/99.

Barring reinfarction and heart rupture, initial hemodynamic observations above the threshold levels suggested ultimate survival. In critically ill patients these threshold values, measured at the time of admission, defined a group in which a roughly 50% probability of fatal outcome existed.

Observations 24 Hours After Admission

By 24 hours after admission 7/16 in the nonsurvivor group had died. The PCW tracings were no longer of sufficient quality in 40 survivors and these data were therefore discarded as being unreliable.

Analysis of the trends for HR, SP, DP, and MVO_{sat} showed that none of these had changed significantly since admission in patients still alive at that moment. Only PCW had decreased significantly (P < 0.001). Moreover in the group of 66 survivors, ten had an admission PCW > 20 mm Hg, while 24 hours later only one patient still presented this elevated left ventricular filling pressure.

When the parameters were combined, mortality could be forecasted more precisely. The most powerful prognostic index for immediate survival was the trend of (DP × MVO_{sat})/PCW of the 23 patients at risk when its value was 250% or less at admission (fig. 2). All nine survivors had an improvement in this index, whereas all nonsurvivors showed a deterioration, except for two who evidenced transient signs of amelioration. Two of 57 survivors with an index exceeding 250% at the time of admission showed a fall in index to less than 250% by 24 hours. In both instances this reflected a slight drop in diastolic blood pressure. The trend of (SP × MVO_{sat})/PCW (table 3) was only slightly less effective in predicting im-

Table 2

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Threshold for survival</th>
<th>S (N = 106)</th>
<th>NS (N = 16)</th>
<th>Threshold for fatality</th>
<th>S (N = 106)</th>
<th>NS (N = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SP (mm Hg)</td>
<td>≥ 150</td>
<td>29</td>
<td>1</td>
<td>&lt; 150</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>PCW (mm Hg)</td>
<td>&lt; 15</td>
<td>66</td>
<td>0</td>
<td>&gt; 20</td>
<td>8</td>
<td>10</td>
</tr>
<tr>
<td>MVO_{sat} (%)</td>
<td>≥ 70</td>
<td>58</td>
<td>0</td>
<td>&lt; 55</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>DP/PCW (mm Hg/mm Hg)</td>
<td>≥ 4.25</td>
<td>94</td>
<td>1</td>
<td>&lt; 4.25</td>
<td>12</td>
<td>15</td>
</tr>
<tr>
<td>MVO_{sat}/PCW (%/mm Hg)</td>
<td>≥ 3.75</td>
<td>89</td>
<td>2</td>
<td>&lt; 3.75</td>
<td>17</td>
<td>14</td>
</tr>
<tr>
<td>(SP × MVO_{sat})/PCW (%)</td>
<td>≥ 0.024</td>
<td>94</td>
<td>2</td>
<td>&lt; 0.024</td>
<td>12</td>
<td>14</td>
</tr>
<tr>
<td>(DP × MVO_{sat})/(PCW × HR) (%/min)</td>
<td>≥ 3.2</td>
<td>96</td>
<td>1</td>
<td>&lt; 3.2</td>
<td>10</td>
<td>15</td>
</tr>
<tr>
<td>0.024SP - 0.217PCW + 0.234MVO_{sat} - 13.1</td>
<td>≥ 0</td>
<td>96</td>
<td>3</td>
<td>&lt; 0</td>
<td>10</td>
<td>13</td>
</tr>
</tbody>
</table>

Abbreviations: S = survivors; NS = nonsurvivors; HR = heart rate; SP = systolic pressure; DP = diastolic pressure; PCW = pulmonary capillary wedge pressure; MVO_{sat} = mixed venous oxygen saturation.

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mediate survival: 10/12 increased but only 6/12 reached the threshold level of 400%.

The most powerful index obtained by linear discriminant analysis (0.024 SP - 0.217 PCW + 0.234 MVO₂sat) was as effective as the two others (table 3). These indices showed transient increases in some patients who ultimately died. On the other hand, a decrease in either (DP × MVO₂sat)/PCW or (0.024 SP - 0.217 PCW + 0.234 MVO₂sat), despite therapy, correctly indicated imminent patient demise.

The trends in all other combined indices showed no greater discriminatory power.

| Table 3 |
| Trend of Hemodynamic Prognostic Indices in Wrongly Classified Survivors and Nonsurvivors 24 Hours after Admission |

<table>
<thead>
<tr>
<th>Index</th>
<th>#&lt;br/&gt;8</th>
<th>NS*&lt;br/&gt; #&lt;br/&gt;16</th>
</tr>
</thead>
<tbody>
<tr>
<td>(SP × MVO₂sat)/PCW</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>(DP × MVO₂sat)/PCW</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>0.024 SP - 0.217 PCW + 0.234 MVO₂sat</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

*Still alive 24 hours after admission.

Abbreviations: 8 = wrongly classified survivors; NS = wrongly classified nonsurvivors; ↑ = increase; ↓ = decrease.

Figure 1

Admission values for PCW of 106 survivors (first column) and 16 nonsurvivors (third column). The difference in PCW for both groups is statistically significant (P < 0.001). However, the most significant feature is that none of the 66 patients with admission PCW < 15 mm Hg died. The second and fourth column give the PCW for the patients from whom these values could still be obtained 24 hours later (66 survivors and 9 nonsurvivors). Only 1/12 survivors with an admission PCW > 20 mm Hg still showed such a high value 24 hours later.

Figure 2

(DP × MVO₂sat)/PCW at admission for 106 survivors and 16 nonsurvivors (first and third column respectively). There is a significant difference between the two groups (P < 0.005). The threshold values above which survival is very likely is at 250% (97 of 99 patients). For all patients with an index < 250% at the time of admission and still alive 24 hours later the trend of (DP × MVO₂sat)/PCW is indicated by a straight line. All survivors show increased values, whereas the index had deteriorated even further in 4/6 nonsurvivors.

Patient Survival

All 106 MI patients surviving their stay in the CCU were discharged from the hospital two to three weeks after their heart attack. Death from cardiogenic shock occurred in seven patients within 24 hours, in eight others on the second day and in one on the third day.

Discussion

Many indices have been proposed to predict survival after acute myocardial infarction. Most parameters used in calculating these indices such as age, sex, location of the infarct, etc. are factors which cannot be influenced by therapy. Heart rate and hemodynamic parameters, on the contrary, are variables reflecting pump function, and improvement or deterioration during medical or surgical intervention.
are measurements of the degree of left ventricular failure and the likelihood of a fatal outcome. The predictive value of these measurements is implied in all the aforementioned indices.50-54

Hemodynamic parameters are relatively easy to measure, and routine determinations possess sufficient accuracy; any combination of parameters can be calculated rapidly and used in patient management, as they immediately reflect changes of left ventricular function.

Sphygmomanometric determinations of systemic blood pressure are reasonably accurate in normotensive and hypertensive patients; in shock, however, an indwelling arterial catheter must be used, positioned in the aorta because vasoconstriction renders peripheral blood pressure determinations unreliable, not reflecting the true performance of the left ventricle. No complications were encountered in the limited experience presented here; however, few patients in this group survived. Very few complications were reported from an intensive care unit where radial artery catheterization was performed routinely.26 In the gray area, where patient survival is not a certainty, accurate determination of systemic blood pressure with an indwelling catheter is to be recommended.

Only invasive techniques can be used for measuring pulmonary artery pressures and mixed venous oxygen saturation. At the site of incision inflammation is usual and after a few days the indwelling catheter produces sterile phlebitis. Infections due to the procedure are very rare and reflect poor technique, as does the occurrence of ventricular dysrhythmias during the passage of the catheter tip through the right ventricle. A more worrisome complication is the occurrence of fan-shaped pulmonary shadows in 10% of 200 consecutive patients monitored with the Swan-Ganz catheter,27 probably caused by pulmonary emboli or by thrombosis of the pulmonary artery; similar complications have been described by others.28, 29 Since we started using systemic anticoagulation with heparin, a prospective monitoring of this complication shows that incidence decreased dramatically, thereby indicating that clotting, and not prolonged positioning of the catheter in wedge position, is the cause.

Hemodynamic monitoring of the critically ill is not a novelty. Various on-line bedside computerized monitoring systems have been described in order to assess patient response to therapy.30, 31 In cardiogenic shock due to myocardial infarction a combination of arterial diastolic pressure, stroke index and central blood volume was found to be a reliable indicator of the severity of shock31 and could be used in patient management. A further refinement of this method32 takes into account not only hemodynamic parameters but also metabolic factors such as blood gasses12, 18 and arterial lactate concentrations reflecting tissue perfusion deficits.

Quite a different approach consists in calculating left ventricular contractility without left ventricular catheterization. This was done33 by measuring systemic diastolic blood pressure, left ventricular filling pressure, and the pre-ejection period measured from the phonocardiogram, the electrocardiogram, and carotid artery pulse tracings. A careful analysis34 of these data showed that the pre-ejection period added very little to the prognostic significance of this index, so that when omitted the method was reduced to a simple determination of two factors, reflecting mainly preload and afterload.

This was the starting point of the present investigation: estimating left ventricular function by measuring preload and afterload, taking into account heart rate and mixed venous oxygen saturation,19 which reflects the adequacy of systemic blood flow in relation to tissue oxygen need.

Early results35 of this investigation showed that hemodynamic measurements at the time of admission did not provide sufficient information on patient prognosis. Different combinations of preload, afterload, and MVO2sat correctly reflect the hemodynamic state of the patient at that moment. A poor pump function was found in a group with a 50% mortality; a more precise prognosis was not possible at the time of admission because the effectiveness of patient therapy could not be foreseen. An improvement of hemodynamic parameters should precede obvious clinical amelioration and this was observed in the present investigation.

Linear discriminant analysis heavily weighted MVO2sat over either SP or PCW; however, all three were needed to evaluate hemodynamic improvement or deterioration. MVO2sat and PCW are independent variables;12 the importance of MVO2sat indicates the usefulness of its on-line monitoring by means of fiberoptic catheters.13

The question remains whether invasive monitoring is required in these patients or whether a clinician at the bedside using auscultation and chest radiography would not be just as effective. Auscultation alone is insufficient as it does not permit detection of interstitial edema and must be associated with frequent chest roentgenograms if the evolution of this stage of pulmonary edema is to be evaluated. Furthermore, radiological changes are much slower in their onset or disappearance than changes in left ventricular filling pressure, or even changes in P terminal forces.36

Pulmonary capillary wedge pressure must remain significantly elevated for prolonged periods of time before alveolar flooding occurs, manifesting clinically as moist rales. Conversely, rales may still be present

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even though PCW has returned to normal. This apparent discrepancy between auscultation, radiography, and hemodynamic monitoring merely reflects the time required for the reabsorption of excess extravascular lung fluids, a process that may take 12 to 24 hours, even when left ventricular filling pressure has returned to normal as the result of treatment. 

Hemodynamic monitoring therefore is a jump from a qualitative evaluation to a quantitative measurement of left ventricular function; furthermore, the effectiveness of therapeutic interventions can be determined immediately. Our data suggest that when pump function remains good during the first few hours after infarction, cardiogenic shock is unlikely to develop unless a new coronary event supervenes. Early therapy may however prevent the onset of the vicious cycle of cardiogenic shock and the loss of ischemic myocardium. The failure to observe instances of pump-failure late after the acute episode favors this interpretation.

The severity of pump failure is clearly related to the extent of the necrotic area.7–9 Serial enzyme determinations,9, 10 radionuclide imaging of the heart9, 11 and precordial ST-segment mapping12 can be used to measure infarct size. Recently evidence has been accumulating13–15 that medical intervention may influence the extent of myocardial necrosis. Of these techniques, only ST-segment mapping may be used directly in patient management to measure the degree of ischemia rather than pump function. The information obtained by this method, measuring degree of ischemia, may complement the findings derived from hemodynamic monitoring, which measures pump function.

All nonsurvivors ultimately died in class IV cardiogenic shock according to the classification used by the myocardial infarction research units (MIRU).16 This classification is based on clinical symptoms and hemodynamic parameters, including the cardiac output. The index presented here combines quantitative information required to make this classification (except the cardiac output) and in addition takes into account MVO₂sat. The aim is for a trend-analysis of an index combining significant and independent variables for assessing left ventricular pump function; in the future, this should include the cardiac output, urine production, plasma colloidosmotic pressure48 reflecting changes in blood volume, and arterial lactate content.49 The clinical usefulness of this or similar indices will have to be assessed in critically ill patients. Carefully controlled studies should determine when mechanical circulatory assist and emergency surgery should be initiated. Conversely the findings from such studies should provide the clinician with evidence when to desist from further massive support therapy since the remaining viable muscle mass cannot support life either on a short or on a long term basis.

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