The Value of Monitoring Pulmonary Artery Pressure for Early Detection of Left Ventricular Failure Following Myocardial Infarction


SUMMARY
Flow-directed catheters recorded serial changes in mean pulmonary artery pressure (PA) every 4 to 6 hours in 25 patients during the first 4 or 5 days following acute myocardial infarction. On the basis of the PA on admission, patients were divided into three groups. Patients in group 1 had normal PA (10–20 mm Hg) and maintained a stroke volume index (SVI) > 35 ml/min/m², a pulmonary artery oxygen saturation (MVSO₂) > 70%, and a normal cardiac index, arterial oxygen saturation, pH, and Pco₂. They developed only minor arrhythmias, no heart failure, and none died. Group 3 consisted of one patient with abnormally low PA (<10 mm Hg) who was hypovolemic. Group 2, those patients with elevated PA (>20 mm Hg) who maintained this elevation over the first 48 hours of monitoring, or showed progressive elevation prior to this, had SVI < 35 ml/min/m², MVSO₂ < 70%, cardiac index < 3 liters/min/m², arterial desaturation, and respiratory alkalosis. They demonstrated clinical evidence of heart failure, had major arrhythmias, and 25% died. Three patients with elevated PA on admission spontaneously returned this pressure to normal over the first 48 hours of monitoring. Each of these patients maintained normal hemodynamics and had a good prognosis. PA was always elevated prior to the usual clinical signs of left ventricular failure. We conclude that PA provides a reliable early objective measure of left ventricular failure and is, therefore, an excellent guide to therapy.

Additional Indexing Words:
Coronary artery disease Flow-directed catheters Cardiac arrhythmias

Despite improved management of arrhythmias following acute myocardial infarction in recent years,¹-⁶ and the consequent improvement in mortality rates,⁷,⁸ little advance has been made in the early recognition and treatment of pump failure.

The prime function of the coronary care unit has been the early detection and therapy of cardiac arrhythmias. But the major causes of morbidity and mortality in these units are heart failure, pulmonary edema, and cardiogenic shock.⁹ MacMillan and associates⁷ reported that over half the patients who have acute myocardial infarction develop either cardiac failure or cardiogenic shock, and the incidence of life-threatening arrhythmias, cardiac arrest, and death in the hospital increases with each of these two serious complications. More patients now die of gradual power failure than of sudden death related to arrhythmia.

Patients admitted to the coronary care unit with acute myocardial infarction may have obvious evidence of left ventricular failure. It is now apparent that the use of noninvasive
and invasive techniques, in addition to careful evaluation of clinical signs, may reveal early evidence of left ventricular dysfunction.  

Heart failure is, however, often a late complication of infarction, and although its overt signs are usually clinically obvious, its early signs may be subtle and the timing of therapy difficult. Furthermore, the use of positive inotropic agents following myocardial infarction is controversial, and we lack a reliable and objective early sign on which to base such therapy.  

In an endeavor to establish some objective early signs of heart failure in the patient who has an acute infarct, and to develop a more rational approach to therapy, we measured the serial changes of pulmonary artery pressure in a group of patients who had acute myocardial infarction, relating the change in pressure to other hemodynamic measurements, physical signs, and clinical progress.

Methods  

We studied 28 patients admitted to the coronary care unit within 24 hours of acute myocardial infarction. Diagnosis was based on typical history of chest pain, classical electrocardiographic changes (Q wave, ST-segment and T-wave changes), and elevation of serum enzymes (creatine phosphokinase, lactic dehydrogenase, and serum glutamic oxaloacetic transaminase). All patients had evidence of transmural infarction, and it was of interest that the elevation of serum enzymes was greater in the group of patients with elevated mean pulmonary artery pressure (PA). The mean values of the maximum creatine phosphokinase, lactic dehydrogenase, and serum glutamic oxaloacetic transaminase for the group with normal PA were 681 units, 282 units, and 124 units respectively, and for the group with elevated PA, 1,361 units, 838 units and 275 units respectively.

Patients’ ages ranged from 28 to 75 years (mean, 58 years); there were 19 men and 9 women. Twenty-four patients were experiencing their first infarction, three patients had one previous infarct, and one patient had two previous infarcts.

Under local anesthesia small polyethylene catheters (Intramedic P.E. 60) were inserted by the Seldinger technique into the medial basilic vein and the brachial artery; the venous catheter was floated under pressure control to the pulmonary artery, which was entered in 25 of the 28 patients. These 25 patients form the basis of this report. Every 4 to 6 hours pulmonary artery and arterial pressures were recorded by Statham P23Db and P23Db transducers and a Century 444 optical recorder. The midchest level was taken as the zero reference point. Arterial and venous samples were taken for determination of oxygen saturation, \( P_{CO_2} \), and \( pH \). Patients breathed room air for 15 min prior to drawing the samples. They were analyzed using Clark and Severinghaus electrodes and a Radiometer-Copenhagen blood-gas analyzer.

Cardiac output was determined by the dye-dilution technique after the injection of 5 mg indocyanine green into the pulmonary artery; sampling was from the brachial artery. Dye curves were recorded by the use of a Waters X-250 cuvette and densitometer and a Texas Instruments recorder.

We calculated total peripheral resistance by dividing the difference of mean systemic arterial pressure and mean right atrial pressure (in mm Hg) by cardiac output (liters/min). The results were expressed in units.

The catheters remained in position a maximum of 7 days (average time, 4 days), usually being removed when the mean pulmonary artery pressure returned to normal.

Ventricular premature contractions were frequently precipitated by the catheter passing through the right ventricle; in two patients, ventricular tachycardia required direct current countershock for control. It should be pointed out that this work was carried out before the introduction of the Swan-Ganz catheter. This catheter has been reported to induce no sustained arrhythmias and would, therefore, be recommended in place of the polyethylene catheter used in this study. One patient developed transient bacteremia while the catheter was in place. The catheters were kept filled with heparin solution (5,000 units/ml) when pressures were not being determined. Few problems with clotting were encountered.

Portable chest roentgenograms were taken every 24 to 48 hours during the monitoring period. Positive roentgenographic signs of pulmonary venous congestion were septal lines, perihilar haze, and perivascular and peribronchial cuffing. The roentgenograms were read independently of knowledge about the clinical findings. Clinical examination was performed when hemodynamics were assessed with particular attention to the presence or absence of third and fourth heart sounds and basal crepitations. The presence of a fourth heart sound was assessed by bedside auscultation agreed upon by two of the authors (B. D. R. and W. D. McC.) and independently assessed by a staff cardiologist, usually without
prior knowledge of the PA. No phonocardiographic records were obtained. A review of 56 normal right heart catheterizations in patients between 21 and 78 years old (mean, 52 years) was undertaken to determine normal values for PA. All but two of these patients had PA between 10 and 20 mm Hg; the two outside these limits had pressures of 21 and 22 mm Hg, respectively.

Results

The relationship of PA to stroke volume index (SVI), arterial pressure, total peripheral resistance, pulmonary artery oxygen saturation (MVSO2), arterial oxygen saturation, PCO2, and pH was first considered for the entire group.

Arterial blood pressure was within the normal range in 21 of the 25 patients. Two patients were hypotensive (systolic pressure <90 mm Hg). Two other patients remained mildly hypertensive during the monitoring period. No relationship existed between rising PA and falling arterial pressure.

Total peripheral resistance varied between 15 and 58 units. Twenty of the 25 patients maintained a normal range (20 to 40 units).

Patients with low cardiac outputs tended to have the highest peripheral resistance. There was, however, no clear relationship between changes in PA and total peripheral resistance.

Figure 1 indicates the relationship between maximum PA recorded for each patient during the monitoring period and SVI, the SVI being assessed as close to the time the pressure was recorded as possible. Maximum PA varied between 12 and 38 mm Hg (mean, 24.4 ± 8.2 mm Hg). SVI varied between 20 and 60 ml/min/m2 (mean, 35.1 ± 10.5 ml/min/m2). A statistically significant inverse relationship exists between these two variables (P < 0.001).

Patients who had PA below 20 mm Hg maintained SVI above 35 ml/min/m2, and most patients with PA above 20 mm Hg had SVI below 35 ml/min/m2. Three patients had PA above 20 mm Hg with SVI above 35 ml/min/m2.

In figure 2, maximum PA is plotted against percentage of pulmonary artery oxygen saturation (MVSO2). This varied between 50% and 81% (mean, 71.0 ± 7.6%). A statistically significant relationship between rising PA and

![Figure 1](image)

**Figure 1**

Relationship between SVI and PA following acute infarction. Each dot represents maximum PA recorded for each of 24 patients during monitoring period. Values for one patient in hypovolemic shock are excluded.

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progressive desaturation can be seen ($P < 0.001$). Patients with PA less than 20 mm Hg maintained MVSO$_2$ above 70%. Those with pressure over 20 mm Hg tended to have MVSO$_2$ less than 75%, and most were below 70%; three patients with saturations over 75% had high PA on admission, but their pressure rapidly returned to within normal limits.

Figure 3 demonstrates the relationship between maximum PA and percentage of arterial oxygen saturation. Arterial saturation varied between 90% and 98.5% (mean, 95.2 ± 2.2%). Again a statistically significant relationship existed between arterial desaturation and elevated PA ($P < 0.002$). Patients with PA below 20 mm Hg maintained normal arterial oxygen saturation. Of those with PA above 20 mm Hg, the majority had arterial saturation below 95%.

In figure 4 the maximum PA recorded for each patient during the monitoring period is related to arterial P$_{CO_2}$ and pH. The P$_{CO_2}$ varied between 27 and 50 mm Hg (mean, 38.4 ± 4.7 mm Hg), patients with PA below 20 mm Hg having P$_{CO_2}$ above 35 mm Hg and those with PA above 20 mm Hg having, except for one patient, levels below 40 mm Hg. Therefore, rising PA usually coincided with falling P$_{CO_2}$.

![Figure 3](image)

**Figure 3**

Relationship between percentage of systemic arterial oxygen saturation (% Sat) and maximum PA for each of 23 patients. Two values are excluded, one from patient in hypocoelmic shock and the other due to unsatisfactory sampling.

The pH varied between 7.35 and 7.54 (mean, 7.44 ± 0.05); PA elevation is associated with rising pH. The patients with elevated PA were all tachypneic, which probably caused the rising pH and falling P$_{CO_2}$.

These data clearly indicated that elevated PA in a patient who had acute myocardial infarction was associated with impaired hemodynamic status.

On the basis of the PA on admission (within 24 hours of acute infarction) we were able to separate three distinct clinical groups (see table 1): those with normal PA (10 to 20 mm Hg), those with elevated PA (above 20 mm Hg), and those with low PA (below 10 mm Hg).
Table 1

Mean Pulmonary Artery Pressure: Behavior in 25 Patients with Acute Myocardial Infarction

<table>
<thead>
<tr>
<th>Group</th>
<th>Pₐ (mm Hg)</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Normal Pₐ</td>
<td>10-20</td>
<td>8</td>
</tr>
<tr>
<td>2. Elevated Pₐ</td>
<td>&gt;20</td>
<td>16</td>
</tr>
<tr>
<td>Spontaneous return</td>
<td></td>
<td></td>
</tr>
<tr>
<td>normal within 48 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>hours after infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent elevation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>or increasing pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Low Pₐ</td>
<td>&lt;10</td>
<td>1</td>
</tr>
</tbody>
</table>

Group 1 (Patients with Normal Pₐ)

The eight patients in this group maintained cardiac index over 3 liters/min/m², SVI above 35 ml/min/m², MVSO₂ above 70%, normal arterial saturation, and relatively normal pH and Pco₂. One patient had a transient fourth heart sound with Pₐ of 20 mm Hg, but no other patient developed gallop rhythm, none had clinical or roentgenographic evidence of pulmonary venous congestion, and none developed heart failure at a later stage of hospitalization. Figure 5 illustrates the sequence of changing Pₐ and clinical course of one of these patients.

The incidence of arrhythmias in this group was low. Three patients had ventricular premature beats requiring lidocaine for suppression. One other had occasional ventricular premature beats, requiring no therapy. No other serious arrhythmias developed, nor cardiogenic shock, and no deaths occurred—at least up to the time of discharge from the hospital.

These patients, moreover, did not have elevated Pₐ during the monitoring period; all had normal pressures on admission, and all maintained this normality. Therefore, the normal Pₐ appeared to be associated with maintenance of satisfactory hemodynamics and an excellent clinical course, and provided a reliable prognostic index.

Group 2 (Patients with Elevated Pₐ)

Sixteen patients had Pₐ greater than 20 mm Hg within 24 hours of their infarction. This group had two subgroups. The first consisted of three patients who, on admission, had elevated pressure which decreased over the first 24 hours and returned to normal levels within 48 hours. These three patients also had a relatively benign course: two had ventricular arrhythmias and the third had transient pulmonary venous congestion. Figure 6 depicts the changing Pₐ and clinical course of this patient. Furthermore, these patients maintained fairly normal hemodynamics and, despite the initial elevation of Pₐ, maintained MVSO₂ above 75% and SVI above 35 ml/min/m².

Although this subgroup contained only three people, we believe that if the Pₐ begins to decrease from elevated levels within the first 24 hours of monitoring, and falls within normal limits by 48 hours, then this pattern
Spontaneous decrease in pulmonary artery pressure after acute infarction in one patient in group 2; 0 = chest roentgenogram obtained but no abnormality noted, no fourth heart sound heard, and no digoxin given; + = chest roentgenogram shows pulmonary venous congestion. Dots connected by solid lines indicate mean; dashed vertical lines indicate the range between systolic and diastolic pressures.

will be associated with a benign hospital course.

The remaining 13 patients comprised the second subgroup. These patients all had elevated PA on admission, and showed further elevation or maintained this elevation at 48 hours. Figure 7 demonstrates the changing PA in one of these patients.

Only one of these patients had a benign course, a man with severe obstructive lung disease. His PA remained high despite his benign hospital course and was clearly related to his pulmonary status (see fig. 8). This demonstrates the difficulty that may be encountered when interpreting an elevated PA in a patient with chronic obstructive respiratory disease. A similar dilemma may result from pulmonary embolism. In both situations the elevated PA may not be correlated with left ventricular failure, and a study of other hemodynamic parameters and clinical status would be necessary to elucidate the degree of left ventricular dysfunction.

The other 12 patients in this subgroup all developed serious complications and, as a group, had impaired hemodynamics. Cardiac index was consistently below 3 liters/min/m², SVI below 35 ml/min/m². MVSO₂ in four patients was between 70 and 75%, and below 70% in all others. Two patients had MVSO₂ below 55%, and each of these patients had pulmonary edema with markedly elevated PA. Arterial oxygen saturation was low, and pH and P₉₀ reflected the characteristic hyperventilation.
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Figure 8

Mild persistent elevation of pulmonary artery pressure following myocardial infarction due to emphysema in the one patient in group 2 who had a benign hospital course; VPC = ventricular premature contractions. Dots connected by solid lines indicate mean; dashed lines indicate the range between systolic and diastolic pressures.

All 12 patients at some time during their course had signs of pulmonary venous congestion on the chest roentgenogram, and all developed fourth heart sounds and basal crepitations. Figure 9 relates the presence of a fourth heart sound and of roentgenographic pulmonary venous congestion to PA.

The presence of a fourth heart sound (atrial gallop) or signs of pulmonary venous congestion on the chest roentgenogram clearly separated those with elevated PA from those with normal PA. No patient with a PA below 20 mm Hg had a fourth heart sound or pulmonary venous congestion. All of the 12 patients with elevated PA at 48 hours of monitoring had both fourth heart sound and pulmonary venous congestion. In addition, three other patients had fourth heart sounds: two of these were patients who had elevated pressure on admission which rapidly returned to normal. The other was a patient grouped with the normals, who had a transient gallop rhythm with a pressure of 20 mm Hg.

The arrhythmias present in the group with elevated PA were interesting (see table 2). Ten patients developed ventricular extrasystoles, four developed ventricular tachycardia (two of these were catheter-induced), and one developed ventricular fibrillation. Six of the 12 developed supraventricular tachycardias. Of these six, only one patient had the arrhythmia on admission (see table 3). In each instance the arrhythmia was preceded by an elevated or rising PA. In three of the patients, other clinical signs of failure were absent, so elevated PA was the only clue to the potential deterioration of the patient. Figure 10 illustrates some findings from one of these patients. Note that the PA was elevated despite a low normal right atrial pressure.

Three of this group of 12 with elevated PA died in the hospital, one patient of acute pulmonary edema on the third day of monitoring, the other two of chronic congestive heart failure later during their hospital stay.

Table 2

<p>| Relationship Between Mean Pulmonary Artery Pressure (PA) and Cardiac Arrhythmias |
|-----------------------------------------|----------------|----------------|</p>
<table>
<thead>
<tr>
<th></th>
<th>Elevated PA (mm Hg) (No. of patients)</th>
<th>Normal PA (mm Hg) (No. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular extrasystoles</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Supraventricular tachycardia</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Atrial flutter</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Sinus tachycardia &gt;100 beats/min</td>
<td>11</td>
<td>0</td>
</tr>
</tbody>
</table>

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Of most critical interest is the fact that only one patient in this group had clinical evidence of left ventricular failure on admission. Even though this group's PA was elevated from the time of admission, three quarters of the patients did not exhibit clinical signs of heart failure until the third day of monitoring (see table 3). Thus elevated PA appeared to be the first objective sign of a deteriorating hemodynamic status, and, therefore, seems significant in predicting which patients will develop heart failure and supraventricular arrhythmias.

All in this group were digitalized during the monitoring period. Two patients were taking digoxin when admitted, the others were digitalized when definite clinical signs of left ventricular failure developed. All patients, except the three who died, responded to digitalis, and their PA returned to normal.

Table 3

<table>
<thead>
<tr>
<th></th>
<th>1 EPA</th>
<th>2 EPA</th>
<th>4 EPA</th>
<th>5 EPA</th>
<th>2 NPA</th>
<th>4 NPA</th>
<th>5 NPA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supraventricular arrhythmia</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular arrhythmia</td>
<td>11</td>
<td>2</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical heart failure</td>
<td>1</td>
<td>2</td>
<td>5</td>
<td>3</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: EPA = elevated mean pulmonary artery pressure; NPA = normal mean pulmonary artery pressure.
PA PRESSURE FOR DETECTION OF LV FAILURE

One patient died in acute pulmonary edema with a steadily rising PA, unresponsive to digitalis and diuretics. Two other patients died in chronic congestive heart failure, again with persistent elevation of the PA, despite adequate digitalization. This failure of the PA to respond to therapy may be a poor prognostic sign and indicate extensive left ventricular damage.

Group 3 (Low PA)

We had only one patient with abnormally low PA, a 64-year-old man who had a 5 to 6-year history of angina. He was admitted to the hospital with severe chest pain, and over a period of 2 to 3 days of continuous pain he lost inferoapical and lateral forces on the vectorcardiogram. He then rather suddenly developed clinical shock. When first monitored, his PA was 8 mm Hg, and on the basis of this, he was thought to be hypovolemic. After infusion of Hartmann’s solution, his hemodynamics rapidly returned to normal, and he made an excellent recovery. It would appear, therefore, that an abnormally low PA, like a very low CVP, may reflect hypovolemia.

Discussion

Despite the considerable work that has been done in assessing hemodynamic changes following acute myocardial infarction, heart failure in these patients is still usually assessed by indirect methods—clinical, roentgenographic, phonocardiographic, cardiorespiratory, and postmortem. More recently, left heart catheterization has been performed following acute myocardial infarction, and some specific data are now being accumulated on left ventricular function. But the method is hazardous and not advised for general use; Hunt and associates reported four serious embolic phenomena in 25 patients in whom the left ventricle was entered.

Catheterization of the pulmonary artery following acute infarction has been carried out by several workers and is routine practice in some coronary care units. But whether the pulmonary artery pressure clearly reflects changing left ventricular function is debatable. Parker and associates and Wiener and associates, studying patients with exercise-induced angina pectoris, found a parallel rise in left ventricular end-diastolic pressure.
(LVEDP) and PA. McCallister and associates, studying patients with coronary artery disease at rest and during exercise, felt that the PA could not be used as a substitute for LVEDP, but that relative changes in PA could be useful as an index of left ventricular function. Hunt and associates found a parallel rise in LVEDP and PA up to an LVEDP of 20 mm Hg in acute infarct patients.

Bouchard and associates have, however, recently reported that in patients with abnormal left ventricular function the LVEDP was consistently higher than pulmonary artery end-diastolic pressure. Furthermore, when LVEDP was elevated by pharmacological means, the pulmonary artery end-diastolic pressure remained unchanged.

Despite this conflicting evidence, in the present group of patients the rising PA appeared to indicate deteriorating hemodynamics.

We have related the PA to MVSO2, SVI, and arterial oxygen saturation and found that there is a linear fall in each of these parameters with rising pressure. Each has been clearly shown by other workers to give good objective evidence of deteriorating left ventricular function; in particular the mixed venous and arterial desaturations have been related to progressive pulmonary venous congestion.

Goldman and associates relating central venous oxygen saturation to clinical status, found that in patients who had uncomplicated infaracts the saturation remained above 70 ± 1%; in patients with heart failure it was 56 ± 1%, and in those with cardiogenic shock and heart failure, 43 ± 1%. They found that in most patients the desaturation could be corrected by diuretics or by oxygen administration, and they concluded that in most instances the desaturation was due to venous congestion.

Scheinman and associates reported on a group of patients who had had acute myocardial infarction. Mean central venous oxygen saturation was significantly greater than MVSO2, but there was no significant difference and correlation was good between changes in central venous pressure compared to changes in MVSO2 even in patients with heart failure or shock. Patients with heart failure had MVSO2 of 58.2 ± 8.7%.

Our MVSO2 readings for patients in heart failure were higher than those reported by Scheinman and associates, yet the PA was elevated in all these patients, suggesting that the PA may be a more sensitive index and an earlier sign of deteriorating left ventricular function than the MVSO2.

Similarly, arterial desaturation has been used as an index of myocardial dysfunction following acute infarction. McNicol and associates reported that the mean arterial oxygen saturation following uncomplicated myocardial infarction was 93 ± 3%, if associated with heart failure 87 ± 8%, and if shock was present 82 ± 8%. They attributed these findings to pulmonary venous congestion and found that the hypoxemia could be relieved by oxygen administration or diuretic therapy. The arterial oxygen saturations in our group of patients in heart failure were higher than those reported by McNicol and associates, again suggesting that the PA may be a more sensitive index of myocardial dysfunction than the arterial desaturation.

Both alkalosis and acidosis have been reported following acute myocardial infarction, but in a large majority of patients little or no change in pH and Pco2 was observed. Kirby and McNicol found that 48% of infarct patients had plasma bicarbonate below 22 mEq/liter, and that while acidosis was common in patients who had both left ventricular failure and shock, metabolic acidosis was compensated for in patients with heart failure by hyperventilation, a fall in pH being much less common than a fall in plasma bicarbonate. Unfortunately, plasma bicarbonate was not consistently assessed in our patients, but the falling Pco2 and rising pH seen in patients with heart failure and elevated PA undoubtedly were due to hyperventilation caused by progressive pulmonary venous congestion.
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This report is based on a relatively small group of patients so care must be taken in drawing any general conclusions. Yet from this small group we begin to see some patterns of behavior in the PA that may be useful in treating the individual patient with an acute myocardial infarction. We believe that the PA gives an early indication of the patient's subsequent behavior, a normal PA on admission indicating a benign clinical course, possibly with only minor arrhythmia problems. If the PA is elevated on admission, but begins to revert toward normal within the first 24 hours of monitoring, and returns to normal within 48 hours, the subsequent course of the patient will also be benign. But those patients who have elevated PA on admission and subsequent progressive elevation, or who maintain an elevated pressure over the first 48 hours, will develop major complications.

The PA often provides the first warning of impending heart failure or serious arrhythmias, the usual early signs of heart failure (fourth heart sound or pulmonary venous congestion) occurring 6 to 24 hours after the PA becomes elevated. The PA appears to be a reliable index on which to base therapeutic maneuvers. Digoxin or diuretics or both would be indicated if the PA remains elevated 48 hours after admission, or if it showed a progressive tendency to rise prior to this, whether or not other signs of heart failure are present. Early use of such therapy may indeed prevent serious arrhythmias or progressive heart failure.

Should the skill and facilities necessary to place pulmonary-artery catheters at the bedside not be available, the best clinical evidence of elevated PA in acute infarct patients appears to be the presence of a fourth heart sound or definite signs of pulmonary venous congestion on the chest roentgenogram.

References


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