Hypovolemia in Shock Due to Acute Myocardial Infarction

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SUMMARY

Twelve patients with the clinical features of shock following acute myocardial infarction were treated with low molecular weight dextran (LMWD) as a plasma volume expander. Two of the patients had elevated central venous pressures (CVP), and neither responded favorably to plasma volume expansion. The remaining 10 patients had CVPs under 7 mm Hg prior to dextran infusion; five survived. Each patient responded favorably to dextran infusion manifested by an increase in arterial pressure and cardiac index. The average increase in CVP in these patients was 1.0 mm Hg per 100 ml of dextran infused. The other five patients died either without recovering from shock or in chronic cardiac failure. These patients failed to show a significant increase in arterial pressure or cardiac index after dextran infusion; CVP increased by an average of 1.9 mm Hg per 100 ml infused. Hypovolemia must be considered in all patients in whom clinical evidence of shock develops as a complication of acute myocardial infarction, and if the CVP is normal or low, plasma volume expansion should be undertaken with caution. Increase in arterial pressure and evidence of improved cardiac index with little rise in CVP indicate a good response to the infusion and excellent prognosis for survival.

Additional Indexing Words:
Central venous pressure Pulmonary arterial pressure Cardiac index
Dextran

Hypovolemia is not considered to be a basic defect in shock associated with acute myocardial infarction and has seldom been recognized as an early complication. However, through routine monitoring of the CVP, we have been made aware of the frequency of this defect in patients in whom circulatory insufficiency or shock develops after acute myocardial infarction. Because hypovolemia is easily treated, some investigators have advocated plasma volume expansion of all patients in whom shock develops after myocardial infarction. On the other hand, the potential hazards of such therapy in the presence of severe myocardial damage has caused us to attempt to establish some guidelines for using plasma volume expanders in these patients. We have conducted hemodynamic studies on 40 patients with circulatory insufficiency or shock complicating acute myocardial infarction. We report here the clinical and hemodynamic responses to low molecular weight dextran (LMWD) in 12 of these patients, 10 of whom had control CVPs of less than 7 mm Hg (10 cm H2O).

Methods

Patients were selected for study because they presented the clinical features of shock (obtundation, weak pulses, cold extremities, and oliguria) following acute myocardial infarction. Mean...
intra-arterial pressure (measured prior to vasopressor therapy) was less than 80 mm Hg in all but three patients. These three each had historical and clinical evidence of pre-existing hypertension. In addition, at the onset of clinical shock these three patients had blood pressures (obtained by the cuff method) that were significantly less than on admission. The diagnosis of acute myocardial infarction was verified by necropsy in three patients and by history, electrocardiogram, or serum enzyme changes in all.

The decision to expand intravascular volume in 10 of the patients was based on the findings of a normal or low CVP (under 7 mm Hg). In two patients although the CVP was slightly elevated (10.5 and 12 mm Hg), volume expansion was attempted in the hope that a further increase in ventricular filling pressure might improve cardiac output. It should be emphasized that the majority of our patients with circulatory insufficiency or shock after acute myocardial infarction have had elevated control CVPs, and thus the question of plasma volume expansion has not arisen.

Patients were studied at the bedside or in a specially equipped study unit. Arterial pressure (MAP) and CVP were measured with a transducer through catheters threaded into the central aorta and superior vena cava or right atrium after surgical exposure of the right brachial vessels. Cardiac output (CO) was measured by the indicator-dilution method by means of indocyanine green injected through the CVP catheter and sampling from the central aortic catheter. Values for CO were obtained by averaging two to five indicator-dilution curves and were corrected for body surface area (CI).

We have found this method to be reproducible to ± 5% when done in rapid succession on the same patient. Pressure pulses, dilution curves, and a standard electrocardiogram lead were recorded on a photographic recorder. Systemic vascular resistance (SVR) was calculated as follows:

\[
SVR = \frac{MAP - CVP}{CO}
\]

Immediately after control measurements LMWD was given in an amount averaging 580 ml (range 200 to 1,000) at an average infusion rate of 7.6 ml per minute (range 4.4 to 12.0). All measurements were repeated within a few minutes after completion of the LMWD infusion. Infusions of isoproterenol (in two patients) and isoproterenol (in one patient) were continued at a constant rate throughout the LMWD study period. One patient received 10 mg of morphine sulfate during the time LMWD was being given.

Results

Two patients, one of whom was receiving isoproterenol throughout the study period, had control CVPs over 7 mm Hg and did not respond to LMWD by significant increases in CI, urine flow, or evidence of clinical improvement. Although one patient had a modest rise in MAP (14 mm Hg) after LMWD, his ultimate recovery from shock was unrelated to plasma volume expansion, and he later died of refractory cardiac failure. The other patient failed to recover from shock. Both of these patients had massive myocardial infarction demonstrated at necropsy.

The remaining 10 patients all had normal or low CVPs (less than 7 mm Hg) before infusion of LMWD and were divided into two groups on the basis of survival. Five of these patients (group A) showed marked clinical improvement and increased urine flow (average increase being 1.7 ml per minute) after receiving LMWD. These patients all survived their illness and were eventually discharged from the hospital. Four of the remaining five patients (group B) failed to show clinical improvement or significant increases in urine flow after receiving LMWD, and none of these five survived to be discharged from the hospital, although two recovered from shock only to die later of cardiac failure.

Comparison is made between the two groups to determine if additional criteria could aid in the early selection of those patients in shock after acute myocardial infarction who can be expected to benefit from plasma volume expansion.

Certain clinical differences between the two groups were apparent. The average age of group A patients was 48 (range 42 to 52) years, whereas group B patients averaged 69 (range 50 to 85) years. None of the group A patients were known to have had previous myocardial infarction, whereas two patients in group B had histories and electrocardiographic evidence of previous infarction. Inferior wall infarction was present in three of the five group A patients, and anterior infarction occurred in four of the five group B patients. The degree of serum enzyme elevation did not, however, differ between the groups. Although a probable cause for hypovolemia
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Table 1

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<tr>
<th>Hemodynamic Status in Groups A and B</th>
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<td>Patient</td>
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<td>Group A</td>
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<td>Mean ± SD</td>
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<td>Group B</td>
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<td>J.A.</td>
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<td>Mean ± SD</td>
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(NE) Received norepinephrine at constant infusion rate throughout study period.
* Admission blood pressure 190/140 (cuff)—fell to 100/70 (cuff) before study.
† Admission blood pressure 110/80 (cuff)—fell to 85/60 (cuff) before study.
‡ Admission blood pressure 150/110 (cuff)—fell to 40 systolic by palpation before study.

Values for heart rate prior to LMWD were not significantly different between the groups and tended to be slightly lower after infusion of LMWD in most patients in both groups.

Prior to LMWD, CI averaged 2.2 ± 0.2 L/min/m² for group A and 2.9 ± 0.8 L/min/m² for group B. After LMWD infusion every group A patient had increased CI by at least 0.7 L/min/m², with an average increase of 1.5 ± 0.9 L/min/m². Only two patients in group B showed significant increases in CI after LMWD, and the average increase for all five patients was 0.1 ± 0.8 L/min/m².

Left ventricular pressure was measured before infusion of LMWD in two patients, one in each group, by passing a catheter retrograde across the aortic valve. These patients are presented in some detail.

Case 1

A.F., a 42-year-old Negro man with a past history of hypertension and anginal pain was admitted after 4 days of increasingly severe
anterior chest pain unrelieved by rest. On admission the patient complained of severe chest pain and required morphine. Blood pressure was 190/140, and the pulse was 80 and regular. Cardiac enlargement and a presystolic gallop were present. There was no evidence of congestive failure. The electrocardiogram showed changes typical of an acute inferior wall infarction, and serial enzymes were confirmatory. The patient was given reserpine, and his blood pressure decreased to 120/80. He continued to complain of severe chest pain and 2 days after admission atrial fibrillation with a rapid ventricular response occurred. Electrocardiographic changes suggested extension of the original infarct with involvement of the posterior basal left ventricle. Although there was no evidence of cardiac failure, digoxin was given to control the rapid ventricular rate. The patient began to complain of nausea and vomiting, which persisted. Four days after admission blood pressure by cuff was found to be 100/70 mm Hg. The pulse was weak, and the extremities were cold and clammy. Urine flow was 30 ml per hour.

Hemodynamic measurements revealed a CVP of 1.5 mm Hg and a left ventricular end-diastolic pressure (LVEDP) of 4.5 mm Hg. MAP was 103 mm Hg but the pulse pressure was only 32 mm Hg. CI was 1.9 L/min/m². The patient was given 500 ml of LMWD, and the CVP increased by only 3 mm Hg. During this infusion MAP rose to 120 mm Hg, pulse pressure increased to 48 mm Hg, and CI to 2.6 L/min/m². Urine flow increased to 170 ml per hour, and upon returning to the ward blood pressure by the cuff method was 140/90 mm Hg. A few days later sinus rhythm returned spontaneously. Recovery was uneventful, and upon discharge the patient was asymptomatic and did not require digitalis.

**Comment.** In this patient circulatory insufficiency was clearly related to hypovolemia due to inadequate fluid intake, diaphoresis, and vomiting. However, until the CVP was measured, hypovolemia was unrecognized. The normal LVEDP, its rapid improvement when given volume, and his subsequent course all attest to the fact that myocardial failure following acute myocardial infarction was not the cause of the circulatory insufficiency.

**Case 2**

A.P., a 50-year-old Negro man, was admitted for severe substernal pain. The patient had been hospitalized 3 months previously for acute myocardial infarction. For 5 years prior to this he had been treated for hypertension. He had been asymptomatic for 2 months, and this was interrupted by the onset of chest pain 2 days before admission. On admission, the patient was alert and complained of chest pain. Blood pressure was 90/60 mm Hg, pulse 130 per minute, and temperature 101.6 F. The lungs were clear. Cardiac enlargement and a "summation" gallop were present. An electrocardiogram showed evidence of an old anterolateral and recent inferior myocardial infarction. Hemodynamic studies were initiated shortly after admission. After the catheters had been inserted, the patient developed rigors and a further elevation of temperature to 106 F. The catheters were changed, and when the rigors had subsided, measurements were made. CVP, which had been 7 mm Hg, fell to 1.5 mm Hg; however, LVEDP remained elevated at 24 mm Hg. MAP was 63 mm Hg and CI was 3.9 L/min/m². When 700 ml of LMWD had been infused, the CVP had increased to 10.5 mm Hg, MAP had fallen to 56 mm Hg, and CI had fallen to 2.6 L/min/m². The temperature had fallen to 101.8 F. The patient was rapidly digitalized, and this resulted in a rise in MAP and CI, while CVP and HR fell. The patient’s condition stabilized; however, during the remainder of his hospital course he complained of severe dyspnea and orthopnea, which remained refractory to vigorous medical therapy until he died in cardiac failure 2 weeks after admission.

**Comment.** The patient had severe cardiac damage from at least two myocardial infarctions. Although CVP was low after an apparent pyrogen reaction, LVEDP was markedly elevated. Plasma volume expansion was accompanied by an elevation in CVP but MAP and CI fell, indicating that hypovolemia was not responsible for the patient’s circulatory insufficiency. Digitalization, which had been withheld at first because of the low CVP, clearly resulted in hemodynamic improvement.

**Discussion**

Although hypovolemia is not present in the majority of patients with acute myocardial infarction, it should be searched for in all patients in whom shock or circulatory insufficiency develops. From our data, measurement of the CVP and, when normal or low, CVP monitoring during a careful therapeutic trial of plasma volume expansion are safe and practical methods for identifying those patients able to respond to volume expansion.

When the CVP is elevated above normal (above 7 mm Hg), volume administration alone is unlikely to result in significant hemodynamic improvement. These patients usually have severe myocardial damage and fail to increase cardiac output when given...
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intravenous infusions. If plasma volume expansion is to be attempted in such patients, it must be done with extreme caution.

When the CVP is not elevated and yet the patient shows evidence of circulatory insufficiency or shock following acute myocardial infarction, hypovolemia must be suspected. Under these circumstances, a therapeutic trial of plasma volume expansion is indicated. A favorable response to fluid administration can be recognized by an increase in arterial pressure, pulse volume, and urine flow accompanied by clinical improvement. The CVP will remain low or increase minimally. The patients in group A (responding well to volume expansion) showed a mean rise in CVP of only 1.0 mm Hg per 100 ml of LMWD infused, and the highest was only 1.5 mm Hg per 100 ml infused. This response is most likely to be seen in younger patients with minimal myocardial damage and indicates an excellent prognosis. When, however, fluid administration results in a rapid increase in the CVP and shock or circulatory insufficiency persists, severe myocardial damage is probably present. The patients in group B (poor response to volume expansion) had a mean rise in the CVP of 1.9 mm Hg per 100 ml of LMWD infused, and the least rise was 1.3 mm Hg per 100 ml. These patients were elderly, had lower initial blood pressures than group A patients, and had clinical or postmortem evidence, or both, of severe myocardial damage.

Although we did not measure total blood volume in our patients, others have found normal or slightly reduced blood volumes in patients with shock after acute myocardial infarction. It is likely that the hemodynamic alterations present in the shock patient prohibit the use of standard normal values for blood volume in determining the need for fluid administration.

We have used the CVP and its response to plasma volume expansion as a guide to fluid therapy. This type of therapeutic trial and monitoring has been proved to be of great value in other types of shock. It should be stressed that in addition to intravascular volume, the CVP is influenced by changes in venous tone and by changes in the compliance of the right ventricle. Under conditions of increased venous tone or decreased right ventricular compliance, or both, the CVP might be normal in spite of an inadequate intravascular volume.

Since myocardial infarction usually involves the left ventricle, and since a sudden rise in LVEDP during fluid administration can precipitate acute pulmonary edema, it is important to determine the relation between the CVP and LVEDP. Increases in CVP have been shown to reflect increases in LVEDP during experimental volume overload. Cohn, and associates have made simultaneous measurements of the CVP and LVEDP in nine patients with acute myocardial infarction and shock, and in none did LVEDP rise without a concomitant rise in CVP. We have made a similar observation in 23 patients with various types of shock in whom we have measured CVP and LVEDP during plasma volume expansion. Although in the two patients presented here LVEDP was not measured following LMWD, the rapid rise in the CVP in the patient whose initial LVEDP was elevated supports the contention that, even in acute myocardial infarction, monitoring the CVP is a safe guide for a trial of plasma volume expansion.

It is possible to precipitate pulmonary edema in the presence of a normal CVP in patients with acute myocardial infarction by adding small amounts of fluid, and, therefore, measurement of LVEDP is the ideal method for monitoring this form of therapy. However, the CVP measurement can easily be made and, if used as a dynamic measurement, can reflect directional changes in LVEDP if addition of volume is the only therapeutic change being made. The catheter can also be used to withdraw fluid, should pulmonary edema develop.

A normal SVR in spite of a reduced CI has been frequently observed in patients with shock due to acute myocardial infarction. The mechanism for this absence of compensatory vasoconstriction is unclear; however, a
similar mechanism might cause a decrease in venous tone and low CVP in patients having normal or elevated blood volumes. Although our patients in groups A and B had similar values for SVR, it is possible that reduced venous tone was partly responsible for normal or low CVPs in some of our group B patients. Group A patients, however, responded well to fluid therapy, and we postulate that hypovolemia was the major cause for their low CVPs and circulatory insufficiency.

The incidence of hypovolemia complicating acute myocardial infarction is probably low, and the routine administration of significant volume loads to all patients with shock does not seem justified. The use of vasodilator agents coupled with plasma volume loading has been advocated; however, this form of therapy has yet to be evaluated clinically and should be reserved for those patients who although manifesting clinical signs of shock, are able to maintain adequate intra-arterial pressures in spite of vasodilator therapy.

Langsjoen and associates have reported a reduced mortality in patients with acute myocardial infarction treated with low molecular weight dextran. Therapy in these patients was designed to reduce intravascular sludging rather than to expand intravascular volume, and the rate of administration was significantly less than in our patients. They have postulated correction of undetected hypovolemia as contributing to improved survival in these patients. Since we did not compare our infusions with equal amounts of dextrose or saline solution infusions, it is impossible for us to know whether the dextran solution had any effect in addition to its contribution to plasma volume.

Allen and others reported hypovolemia to be present in 20% of patients with cardiogenic shock. Although this figure corresponds closely to our own experience, it has undoubtedly been inflated by exclusion of patients presenting with profound shock due to rapidly progressing pump failure, since such patients frequently do not survive long enough for hemodynamic measurements to be obtained.

There are many mechanisms by which hypovolemia can develop after acute myocardial infarction:

1. Inadequate fluid intake is common, and parenteral fluid administration is frequently kept at a minimum for fear of precipitating cardiac failure.

2. Excessive loss of fluids may occur because of fever and diaphoresis, the use of potent diuretics for pulmonary edema, and as a result of nausea and vomiting secondary to pain or drugs.

3. A shift of fluid from the intravascular to the extravascular compartments, or trapping of red cell aggregates in various capillary beds may result in a reduced effective circulating blood volume. This is most likely to occur in patients who remain vasoconstricted for prolonged periods, either from compensatory mechanisms or from the use of various pressor agents. Also, acute acidosis following temporary circulatory arrest may result in sudden marked loss of intravascular volume.

4. In addition to the primary illness, associated illnesses such as sepsis or gastrointestinal bleeding may cause hypovolemia in patients with acute myocardial infarction and, if unrecognized, may lead to a decrease in coronary flow and extension of the myocardial damage.

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