Left Ventricular Function in Acute Myocardial Infarction and its Clinical Significance

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SUMMARY

Investigations on left ventricular function in patients with acute myocardial infarction and the relationship to clinical findings have shown: (1) limitations in the use and interpretation of central venous pressure; (2) pulmonary artery end-diastolic pressure accurately reflects left ventricular filling pressure in the absence of pulmonary vascular or mitral valve disease; (3) left ventricular filling pressure is frequently elevated in mild or clinical uncomplicated infarction; (4) left ventricular function frequently improves during the immediate as well as late convalescent period; (5) the hemodynamic and clinical evaluations may frequently be at variance; (6) a left ventricular gallop is usually associated with an abnormally elevated left ventricular filling pressure; (7) anterior infarctions present greater depression of left ventricular function than inferior infarctions; and (8) monitoring of hemodynamics can be useful in following the changes in left ventricular function and the response to therapy in patients with heart failure and cardiogenic shock.

The evaluation of patients sustaining an acute myocardial infarction has traditionally involved the description of symptoms, observations on physical examination, review of the electrocardiogram, radiographic assessment of the heart and lungs, and various laboratory determinations. During the past 20 years hemodynamic measurements from the right heart, pulmonary circulation, and left heart have been reported in patients with acute myocardial infarction. This information has proved useful in understanding the extent of altered cardiovascular function, classifying the severity of the clinical state, measuring the response to various forms of treatment, and predicting the prognosis and survival of the patient. The objective of the present review is to describe the methods employed for acquiring hemodynamic data, the various findings in the right heart, pulmonary circulation and left heart, and the relationship of hemodynamic findings to the clinical situation in patients with acute myocardial infarction.

Methods

Techniques utilizing standard cardiac catheterization procedures have been employed to obtain hemodynamic information from the right and left heart in patients with acute myocardial infarction. Catheters have been introduced by the percutaneous technique and advanced to the superior vena cava, right atrium, right ventricle, and pulmonary artery to record pressure. A significant development has been the introduction of the Swan-Ganz balloon-tipped catheter for obtaining hemodynamic information in patients with acute myocardial infarction. This catheter does not require fluoroscopy and can be advanced with pressure monitoring to the right atrium, where the small balloon is inflated, and the balloon flow directs the catheter into the pulmonary artery position. Once in the pulmonary artery the catheter can be deflated and then advanced and reinfated to obtain pulmonary capillary or wedge pressure.

Hemodynamic data have also been obtained from the left heart in patients with acute myocardial infarction, and several catheterization techniques have been used. One study involved transseptal catheterization with recording of left atrial pressure in patients with acute myocardial infarction. This approach required percutaneous entry into the right femoral vein and advancement of the catheter and transseptal needle from...
the right atrium into the left atrium. Other studies have employed percutaneous puncture of the femoral artery and retrograde passage of the catheter into the left ventricle. A percutaneous catheter has been developed which permits blind entry into the left ventricle by this retrograde technique and requires only electrocardiographic monitoring. Additional approaches include percutaneous entry or open arteriotomy of the brachial artery with advancement of catheters into the left ventricle.

The measurement of cardiac output in patients with acute myocardial infarction has involved standard catheterization procedures as well as noninvasive methods. The standard indicator-dilution technique has been employed with the dye being injected on the right side of the circulation and sampled from a peripheral artery. The Fick method has also been used with arteriovenous oxygen difference obtained from the pulmonary artery and a systemic artery with simultaneous measurement of oxygen consumption. The radioisotope method has been employed with precordial counting for the estimation of cardiac output in patients with acute infarction. Recently, techniques have been developed for measuring cardiac output with the use of thermal indicators, which are injected into the right atrium with sampling from the pulmonary artery through a single catheter. The noninvasive technique of ultrasound has been applied in patients with acute infarction to estimate stroke volume and cardiac output.

Cardiovascular Dynamics

Central Venous Pressure: Venous pressure measurements have been recorded for several years in patients with acute myocardial infarction. In earlier studies measurements were obtained from an antecubital vein, but in more recent studies central venous pressure has been measured directly in the right atrium. Freis and co-workers initially reported venous pressure of 90 mm H₂O in patients with mild myocardial infarction. Patients with severe infarction were found to have an average venous pressure of 120 mm H₂O, and those in shock had a pressure of 115 mm H₂O. These investigators concluded that an increase in venoconstriction produced an increase in venous filling pressure in the presence of the failing heart. Gilbert and associates recorded venous pressure in 20 patients with myocardial infarction, and in mild uncomplicated infarction the venous pressure was 130 mm H₂O. In patients with failure the venous pressure was elevated to 167 mm H₂O, and those in shock exhibited an average venous pressure of 215 mm H₂O. Gammill and co-workers reported a much narrower range for venous pressure in patients with acute infarction. These investigators found patients with mild infarctions to have an average value of 102 mm H₂O, while those in heart failure displayed values of 114, and those in shock 124 mm H₂O, respectively.

Gunnar and his group recorded central venous pressure in 23 patients in acute myocardial infarction, 12 of whom presented in cardiogenic shock. Measurements in two of the 11 patients without shock revealed a central venous pressure of 4 and 23 mm Hg, while the 12 patients in shock revealed central venous pressures ranging from 0.5 to 22 mm Hg, with a mean value of 7.9. Smith et al. reported two of 14 patients with cardiogenic shock in myocardial infarction having a central venous pressure less than 6 mm Hg. In 15 patients with acute myocardial infarction, McKenzie and co-workers found the right atrial pressure ranging from 5 to 14 mm Hg in nine patients without shock, while in six patients with shock values varied from 13 to 22 mm Hg. Weil and Shubin reported a central venous pressure of 9.3 mm Hg in 20 patients with acute myocardial infarction and shock, but in seven patients the value exceeded 10 mm Hg. Cohn et al. studied nine patients in shock with acute myocardial infarction and described a range of central venous pressure from −6 to +6 mm Hg, and only two patients had values of 5 and 6 mm Hg. During the infusion of 500 ml of dextran, elevation in right atrial pressure paralleled left atrial pressure. Collins and co-workers found the central venous pressure elevated in 21 of 25 patients presenting acute myocardial infarction.

The mechanism for the elevation of central venous pressure in acute infarction has been explained by venoconstriction, alterations in effective blood volume, and the development of heart failure. Nixon and his group proposed dominance of venoconstriction in
the elevation of central venous pressure in acute infarction, but Cohn et al.17 suggested a relationship between central venous pressure and left ventricular filling pressure. However, Hamosh and Cohn18 have more recently compared right atrial pressures to the left ventricular end-diastolic pressure in 40 patients with acute myocardial infarction and demonstrated a lack of correlation. Rapaport and Scheinman19 studied right heart and pulmonary dynamics in 24 patients with acute infarction and demonstrated a poor correlation between changes in central venous pressure and pulmonary artery end-diastolic pressure. Therefore, the central venous pressure is not regarded as an accurate reflection of the filling pressure in the left ventricle in acute myocardial infarction.

Pulmonary Artery Dynamics: Pulmonary artery pressure has been recorded in patients with acute myocardial infarction. Fluck and associates20 found the pulmonary artery pressure abnormally elevated in 21 of 26 patients. Rutherford and associates21 measured pulmonary artery pressure in 25 patients and found eight patients with normal mean pulmonary artery pressure, while 16 had abnormal elevations of the mean pressure, and one patient presented a low mean pressure in the pulmonary artery. Hunt and associates22 described the risks in measuring pulmonary artery pressure, as well as methods for reducing complications. Since the Swan-Ganz catheter can be advanced to the pulmonary artery with considerable ease, pulmonary artery pressure can be measured in patients with acute infarction, and abnormal elevations have been found more frequently than elevations of central venous pressure.

By advancing a catheter in the pulmonary artery, a wedge or capillary pressure can be recorded, which has been shown to reflect mean left atrial pressure. Clinical studies in various forms of heart disease have demonstrated that the capillary wedge pressure accurately reflects the mean pressure in the left atrium, and in the absence of mitral valve obstruction the left ventricular end-diastolic pressure.23, 24 Experimentally, heart rates must exceed 160–180 beats per minute before a discrepancy develops between mean left atrial pressure and left ventricular end-diastolic pressure.25 Lassers and co-workers26 studied 30 patients with acute myocardial infarction complicated by advanced forms of heart block. The mean pulmonary arterial wedge pressure in this group of complicated infarcts ranged from 4 to 37 mm Hg, and in 24 patients exceeded the 12 mm Hg, the upper limit of normal.

In addition to measurements of pulmonary artery systolic pressure, mean pressure, and wedge pressure, the diastolic pressure of the pulmonary artery has also been used as an indication of left ventricular filling pressure. Kaltman and co-workers27 studied 47 patients with congenital and acquired heart disease and demonstrated a relationship between pulmonary artery end-diastolic and left ventricular end-diastolic pressures over a range of 5 to 20 mm Hg. In two patients with severe elevations of left ventricular end-diastolic pressure the linear relationship was still maintained with pulmonary artery end-diastolic pressure. Hunt and co-workers22 described simultaneous measurements of pulmonary artery and left ventricular end-diastolic pressures in five patients with acute infarction, and reported a linear relationship ranging from 5 to 20 mm Hg. Jenkins and associates28 evaluated the pulmonary artery end-diastolic pressure as an indirect estimate of mean left atrial pressure in 28 patients with acute and chronic disease. These investigators demonstrated a linear relationship between pulmonary artery end-diastolic pressure and left atrial mean pressure with normal pulmonary vascular resistance. However, with abnormal increases in pulmonary vascular resistance, the relationship was somewhat altered. Even in the presence of elevated pulmonary vascular resistance, directional changes still occurred between pulmonary artery end-diastolic and mean left atrial pressure. Pulmonary artery end-diastolic pressure did not exceed left atrial mean pressure in any instance. Finally, acute hypoxia and acidosis may exert important effects upon pulmonary artery pressure and must be corrected.
before interpretations of left ventricular hemodynamics are made from pulmonary artery end-diastolic pressure.

Nevertheless, debate persists over the use of pulmonary artery end-diastolic pressure as an accurate measure of left ventricular filling pressure. Falicov and Resnekov\textsuperscript{20} compared these two pressures in 71 patients with chronic heart disease and found a relationship between pulmonary end-diastolic pressure and pulmonary artery mean wedge pressure. However, the pulmonary artery end-diastolic pressure failed to reflect accurately left ventricular end-diastolic pressure. These investigators did report a relationship between the A wave in the pulmonary artery pressure and the left ventricular end-diastolic pressure. Bouchard and others\textsuperscript{30} further demonstrated a discrepancy in pulmonary artery end-diastolic and left ventricular end-diastolic pressures in chronic heart disease, and the difference was further aggravated with the use of various pharmacologic agents.

The pulmonary artery end-diastolic pressure has been reported to be abnormally elevated in approximately 50% of patients with uncomplicated myocardial infarction exhibiting no clinical evidence of left ventricular failure.\textsuperscript{31, 32, 33} Patients in heart failure consistently demonstrate abnormal elevations in end-diastolic pressure, but the range is large. However, patients in cardiogenic shock frequently present severely elevated pulmonary artery end-diastolic pressures, but on occasion may exhibit normal or slightly elevated pressures.

**Left Heart Dynamics:** Physiologists and investigators have interpreted the level of left ventricular filling pressure as a reflection of cardiac performance. In various forms of chronic heart disease the left ventricular end-diastolic pressure may be altered by a variety of mechanisms and cannot be used as a uniform index of cardiac performance.\textsuperscript{34, 35} Nevertheless, in acute heart failure the elevation of filling pressure of the left ventricle may still parallel the end-diastolic pressure-volume relationship. Left atrial and left ventricular pressures have been recorded in patients with acute myocardial infarction. Kirby et al.\textsuperscript{36} reported right and left heart pressures in two patients with acute myocardial infarction. Right atrial pressures were 12 and 17 mm Hg, while left ventricular end-diastolic pressures were 23 and 33 mm Hg, respectively, in these two patients. Both these patients presented clinical evidence of left heart failure. Later, Nixon and associates\textsuperscript{37} recorded a left atrial pressure of 10 mm Hg in myocardial infarction and shock, and the patient responded dramatically to volume expansion. Nixon\textsuperscript{38} subsequently reported a patient with acute pulmonary edema in myocardial infarction with a left ventricular end-diastolic pressure ranging from 3 to 5 mm Hg. These observations on left ventricular filling pressure in cardiogenic shock demonstrated that pressures could be low, normal, or severely elevated. Furthermore, the lack of significant linear correlation between right atrial and left ventricular end-diastolic pressure in acute myocardial infarction indicated the necessity for recording left ventricular filling pressure in patients with heart failure or cardiogenic shock. Hamosh and Cohn\textsuperscript{18} performed left ventricular catheterization, and recorded pressure in 40 patients with acute myocardial infarction. In 14 patients without evidence of heart failure, left ventricular end-diastolic pressure ranged from 6 to 22.5 with a mean of 15 mm Hg. In 12 patients with heart failure the filling pressure ranged from 19 to 40, with a mean value of 29.9 mm Hg. In 12 patients in cardiogenic shock the left ventricular end-diastolic pressure ranged from 8 to 34, with a mean value of 21.1 mm Hg. This study indicated that measurements of left ventricular end-diastolic pressure were similar to previous observations on pulmonary artery end-diastolic pressure, in that patients with uncomplicated infarction frequently exhibited abnormal elevations. On the other hand, heart failure was associated with severe elevations, and shock revealed a range from mild to severe elevations.

**Cardiac Output:** Although it was initially suspected that cardiac output was significantly reduced in all patients with myocardial

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Infarction, a range of values has been reported by various investigators. The indicator-dilution technique has been the most frequently employed method for measuring cardiac output in acute myocardial infarction. Oriol and McGregor8 have cautioned against the possible errors in measuring output by the indicator-dilution method in patients with shock. Errors can be introduced by a prolonged sampling time and are due in part to recirculation. However, acceptable accuracy can be obtained by injecting into the pulmonary artery and sampling at a closer systemic arterial site. Freis and associates7 found a normal cardiac index averaging 3.4 liters/min/m² in patients with mild myocardial infarction, while those patients with severe infarctions had reduced average index of 2.9 liters/min/m², and in cardiogenic shock even lower values of 1.8 liters/min/m². Similar values were reported by Gilbert and co-workers8 in mild infarction, as well as in heart failure, and shock groups with cardiac indices of 2.6, 1.9, and 1.0 liters/min/m², respectively. Gammill and associates9 reported cardiac-index values of 4.3 liters/min/m² in patients with uncomplicated infarction, while those with severe infarction and shock had values of 2.9 liters/min/m². Patients with intermediate complications exhibited an average index of 3.2 liters/min/m². Brock and co-workers10 in 1959 similarly reported cardiac-index values of 3.03 in uncomplicated infarction, and those with circulatory failure possessed reduced indices averaging 2.04 liters/min/m². Fluck and co-workers20 reported cardiac outputs in seven patients in acute infarction ranging from 2.5 to 5.5 liters/min/m². Gunnar and co-workers10 measured cardiac output in 23 patients with acute infarction, 12 of whom had cardiogenic shock. In the 11 patients without shock the mean index was 3.8. The 12 patients with shock were divided into two groups of six patients each. In the six patients with high peripheral vascular resistance the average index was 1.66, while in the other six patients with low resistance values the index was 2.65 liters/min/m². In nine patients with infarction and shock studied by Cohn et al.14 the average output was 3.2, while Weil and Shubin13 found a mean cardiac index of 1.3 in 20 patients with shock and infarction. Ramo and associates40 measured cardiac output in patients with uncomplicated infarctions as well as those in mild heart failure, pulmonary edema, and cardiogenic shock and observed a wide range of values in each group. Russell and associates41 measured cardiac indices in 19 patients with myocardial infarction, and values ranged from 1.0 to 4.0 liters/min/m² in a series that included uncomplicated patients as well as those with heart failure and cardiogenic shock. Hamosh and Cohn18 studied 40 patients with infarction and included 14 without complications, 12 in heart failure, and 14 in shock. Average cardiac index in the uncomplicated group was 2.98 liters/min/m², with values of 2.63 and 1.59 liters/min/m² in the heart-failure and shock groups, respectively. Rutherford and associates21 studied 25 patients and found eight patients with a cardiac index greater than 3.0 among those with a normal mean pulmonary artery pressure, and a cardiac index less than 3.0 liters/min/m² in 12 patients with an elevated mean pulmonary artery pressure.

Peripheral Resistance: Since the measurement of systemic peripheral vascular resistance is determined by the measurements of pressure and cardiac output, the values obtained in patients with acute myocardial infarction exhibit considerable variation especially in those patients with heart failure and cardiogenic shock. Freis and co-workers7 reported an average calculated peripheral vascular resistance of 1175 dynes-sec-cm⁻⁵ in four patients with mild infarction. In five patients with moderately severe infarction the vascular resistance was 1325, and in four patients with cardiogenic shock the value was 2050 dynes-sec-cm⁻⁵. Similar values were reported by Gilbert and co-workers8 in studies on 20 patients with acute myocardial infarction. Thirteen patients without shock had a range of resistance values from 1300 to 3600 dynes-sec-cm⁻⁵, while seven patients in shock exhibited values ranging from 1600 to 3900 dynes-sec-cm⁻⁵.
dynes-sec-cm⁻². Gammill and his group⁹ reported in patients with mild infarction an average value of 1272, while in patients with moderately severe infarction the value was 1555, and in those with shock the measurement was 1781 dynes-sec-cm⁻². Sixteen of these 39 patients were restudied 4 weeks after admission, and an average decrease in peripheral vascular resistance of 373 dynes-sec-cm⁻² was recorded, which was attributed to a fall in blood pressure during the convalescent period. Gunnar et al.¹⁰ studied 23 patients, 12 of whom were in shock with acute myocardial infarction. In the 11 patients without shock the vascular resistance was 26 mm Hg/liter/min. In 12 patients with cardiogenic shock six had resistance values from 21 to 97, while the remainder of the group had values ranging from 12 to 17. In the study of Cohn et al.¹⁴ in nine patients with shock resulting from infarction, vascular resistance varied from 703 to 4176 dynes-sec-cm⁻². Weil and Shubin¹³ in their evaluation of 20 patients with infarction reported an average value of 2042 ± 826 dynes-sec-cm⁻². Therefore, in patients with acute myocardial infarction the peripheral vascular resistance does vary over a wide range, but there is a definite tendency for patients with uncomplicated infarctions to have lower resistance values than in those patients with heart failure who have elevated values. Patients who develop shock with acute infarction may have normal or severely elevated values for peripheral vascular resistance.

*Left Ventricular Function:* Although the measurement of intracardiac pressures and flow represent the mechanical performance of the heart, studies have been performed in recent years that utilized interventions to alter cardiac performance and thereby derive a measure of left ventricular function based on the response to the intervention. The initial observation of Nixon et al.² in a patient with cardiogenic shock and a normal left ventricular filling pressure supported earlier physiologic hypotheses that relative hypovolemia might be operative in cardiogenic shock, and this was further substantiated by the dramatic response to volume expansion. Cohn and associates¹⁴ infused low molecular weight dextran in patients with cardiogenic shock and related filling pressures to cardiac output before and after volume expansion. They concluded that an increase in filling pressure of 3 cm of H₂O/100 ml of dextran infused was abnormal and suggested a flat portion of a ventricular function curve. Russell and co-workers¹¹ evaluated the effects of raising the left ventricular filling pressure in 19 patients with acute myocardial infarction. Ventricular-function curves were described by relating cardiac output, stroke index, stroke work, or stroke power to the left ventricular filling pressure before and during serial infusions of low molecular weight dextran (fig. 1). A spectrum of function curves was described, and in six patients a descending limb of the function curve represented a fall in cardiac output with further increase in filling pressure, which accompanied the infusion of low molecular weight dextran (fig. 2). Russell and his co-workers¹¹ identified the optimum filling pressure of the left ventricle in patients with acute myocardial infarction between 20 and 24 mm Hg, which tended to produce the highest cardiac output in these patients. Bradley and co-workers¹² compared right and left ventricular-function curves in six patients with acute myocardial infarction and shock. These investigators found that a small increase in right atrial pressure could produce considerable increases in left atrial pressure and resultant improvement in left ventricular performance. Similarly, small decreases in right atrial pressure could result in large decreases in left atrial pressure. In addition to the possible benefits of reducing sludging and platelet coagulation, low molecular weight dextran does provide volume expansion with a modest stretch on the left ventricle in patients with acute infarction. The effects of the low molecular weight dextran infused rapidly in amounts of 400–600 ml have been observed to persist for 6–9 hours in patients with acute infarction (fig. 3). Further observations on ventricular function were made by Scheidt and associates⁴¹ who calculated cardiac work.
from mean left ventricular systolic pressure, left ventricular end-diastolic pressure, and cardiac output in 38 patients with acute myocardial infarction. Survivors had mean cardiac-work values of 5.8 kg-m/min, while the nonsurvivors had values of 2.1 kg-m/min.

**Hemodynamic Monitoring:** Not only have the basic measurements of intravascular pressures and cardiac output been recorded in patients with myocardial infarction, but serial measurements have been made over periods of 24 hours to several days. Shillingford and Thomas\(^4^4\) reported measurements of cardiac output, brachial arterial pressures, stroke volume, peripheral resistance, and heart rate during the initial 3 days of acute infarction in four patients. Subsequent observations were made in 6–10 days and in 3–4 weeks after the acute infarction, and improvement was shown for cardiac output in these four patients. Later, Fluck and co-workers\(^2^0\) recorded serial measurements of pulmonary artery systolic pressure in 22 episodes of acute infarction during the initial 6 days. In six surviving patients pulmonary artery systolic pressure returned to normal at the end of the sixth day, and similar trends toward normal for the right ventricular end-diastolic pressure were also observed by these investigators. Weil and Shubin\(^1^3\) in their study of 20 patients with cardiogenic shock and infarction monitored hemodynamic changes during the initial 24 hours, and survivors were characterized by an increase in mean arterial pressure with a

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A composite of left ventricular function curves in patients with acute myocardial infarction. An increase in stroke index generally continued until left ventricular filling pressure reached 20–24 mm Hg (solid curves), and beyond this pressure the curves tended to flatten or decrease (dashed lines). On the right the first and second function curves in a patient are numbered 1 and 2. A star identified each of the five curves whose ratio of stroke index / filling pressure was less than 0.30 ml/mm Hg. (Reprinted from J Clin Invest, by permission.)

Hunt and associates emphasized the usefulness of monitoring pulmonary artery pressure in acute myocardial infarction to measure not only systolic pressure, but also end-diastolic pressure. Russell and co-workers monitored hemodynamic measurements serially in patients with acute myocardial infarction and assessed left ventricular function during the first 3 days with the infusion of low molecular weight dextran (fig. 4). In six patients with ventricular-function curves recorded on days 1, 2, and 3 after the infarction, hemodynamic improvement was demonstrated by a shift of the function curve upward and to the left. On days 2 and 3 after the infarction the patients responded with a larger increase in cardiac output than elevation in filling pressure for the same amount of infused dextran. These studies suggested that significant improvement in the hemodynamic function of the left ventricle occurred in the first 3 days of myocardial infarction. Rutherford and associates have also advocated the value of monitoring pulmonary artery pressure in acute infarction for the early detection of left ventricular failure. In 25 patients flow-directed pulmonary artery catheters remained in place for 4–5 days, and these investigators reported that the mean pulmonary artery pressure was consistently elevated prior to the usual clinical signs of left ventricular failure. Furthermore, the monitoring of mean pulmonary artery pressure provided a guide to pharmacologic therapy. Porter and associates illustrated the usefulness of pulmonary artery pressure monitoring in a patient with
cardiogenic shock due to a ruptured ventricular septum, and pressures were measured for a period of 10 days. In this patient the monitored pulmonary artery diastolic pressure proved to be an extremely useful guide for pharmacologic intervention and later enabled the patient to undergo successful cardiac surgery.

**Clinical Significance**

Although the central venous pressure has been advocated as a useful guide for the assessment of the blood volume in certain forms of shock, studies have now demonstrated that the central venous pressure does not accurately reflect the left ventricular filling pressure in patients with acute myocardial infarction. On occasion the central venous pressure may be elevated with a normal pulmonary artery pressure in patients with severe diaphragmatic infarction. However, the more frequent hemodynamic abnormality is that of an elevated left ventricular filling pressure in association with a normal central venous pressure. The disparity between central venous pressure and left ventricular filling pressure is exaggerated with the infusion of low molecular weight dextran and the expansion of blood volume. Therefore, the limitations of central venous pressure monitoring in acute myocardial infarction must be appreciated. The central venous pressure can be misleading in the recognition, monitoring, and evaluation of pharmacologic interventions in patients with heart failure or shock complicating acute myocardial infarction. Ramo and associates were not able to correlate rales or left ventricular gallop with elevations in right
Serial ventricular function curves are displayed in four patients on days 1, 2, and 3 after acute infarction. During convalescence the repeat function curve for each patient demonstrated a higher cardiac index and a lower pulmonary artery end-diastolic pressure. (Reprinted from J Clin Invest, 41 by permission.)

Figure 4

atrial pressure. Similarly, Collins and co-workers found little correlation between the central venous pressure and X-ray evidence of pulmonary edema. An elevated central venous pressure was associated with serious cardiac arrhythmias and posterior infarctions, and when this was greater than 20 cm of H2O a fatal outcome was experienced. Therefore, these studies indicate that the right atrial pressure is not a sensitive measure of left ventricular function in patients with acute myocardial infarction.

The measurement of pulmonary artery pressures has been useful in assessing the extent of impairment of left ventricular function. Lassers and associates reported similar values for pulmonary artery wedge pressures in those patients presenting with dyspnea as well as those who were not dyspneic. In addition, the mean pulmonary artery wedge pressure was not significantly different in those patients with or without pulmonary basilar rales. Fluck and his group observed that the presence of an elevated pulmonary artery pressure during the acute stage of infarction proved of prognostic value, and with prolonged elevations five deaths were reported in 21 episodes. The persistence of an elevated pulmonary artery pressure after the first few days of infarction has also been

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associated with clinical evidence of chronic heart failure. In the group of patients with pulmonary artery pressure monitoring studied by Rutherford et al.\textsuperscript{21} those patients that maintained an elevated mean pulmonary artery pressure greater than 20 mm Hg over 48 hours demonstrated clinical evidence of heart failure, experienced major arrhythmias, and also had a mortality of 25%. Therefore, the pulmonary artery pressure in acute myocardial infarction can reflect alterations in left ventricular function but do not consistently correlate with symptoms or radiographic findings.

The measurement of left ventricular filling pressure, either directly or as pulmonary wedge or end-diastolic pressure, has proved to be a sensitive reflection of left ventricular dysfunction in acute myocardial infarction. Since patients with mild or uncomplicated infarctions frequently demonstrate abnormal elevations of the left ventricular filling pressure during the first few days after infarction, the symptoms and physical findings of left heart failure do not necessarily manifest the altered hemodynamics. The presence of gallop heart sounds did not correlate with elevations in pulmonary wedge pressure in the study of Lassers et al.\textsuperscript{26} A high incidence of atrial gallop has been recorded in patients with acute myocardial infarction,\textsuperscript{46} but variations in pulmonary wedge pressure from 4 to 34 mm Hg have been reported.\textsuperscript{26} However, left ventricular gallop is usually associated with an elevation of left ventricular filling pressure,\textsuperscript{47} but patients may have elevated pressures during acute myocardial infarction without the presence of a ventricular gallop. The left ventricular filling pressure has been fairly consistently elevated in those patients manifesting heart failure, but the extent of elevation may be useful in the selection of pharmacologic agents. Based on the observations of the ventricular function curves, mild volume overload may be hemodynamically beneficial to patients with myocardial infarction, and diuretics may be most beneficial in the treatment of pulmonary congestion and excessive elevation of left ventricular filling pressures. In cardiogenic shock measurement of the filling pressure is important in selecting the various pharmacologic agents such as volume expansion or sympathomimetic amines.

Although the measurement of cardiac output has been performed by many investigators in acute myocardial infarction, the range of values and overlap encountered in uncomplicated patients as well as those in heart failure and cardiogenic shock restricts the interpretation of a single measurement. The large collection of data indicates that cardiac output may be normal or subnormal in mild infarctions, and patients in heart failure and shock have more severe depression in cardiac output. Although a range of values for cardiac outputs in cardiogenic shock has been described, those patients with severely reduced cardiac outputs tend to have an extremely high mortality, whereas the small percentage of survivors often have slightly higher values for output. Even though knowledge of the cardiac output may not be clinically useful in uncomplicated patients, this measurement can be helpful in assessing the prognosis and pharmacologic management of patients in cardiogenic shock.\textsuperscript{48, 49}

The calculation of peripheral vascular resistance in acute myocardial infarction is a derived value from cardiac output and pressure and is thus subject to similar clinical limitations as the basic measurement of cardiac output. Since normal as well as elevated values for peripheral vascular resistance may be encountered in cardiogenic shock, this measurement may possess clinical value. Studies do suggest that patients with extremely high peripheral vascular resistance in cardiogenic shock have a much worse prognosis than those with normal or slightly elevated values. Furthermore, the resistance value may facilitate the selection of drugs in the management of cardiogenic shock. However, in patients with heart failure the measurement of peripheral vascular resistance does not correlate with clinical findings and course.

Important clinical information can be derived from the relationship of left ventricular
filling pressure, cardiac output, and aortic pressure before and after pharmacologic intervention in patients with acute myocardial infarction. Left ventricular function has been reported to be more depressed and hospital mortality higher in patients with anterior infarction than those with diaphragmatic infarctions. The ventricular-function curves aid in quantitating the level of depression of left ventricular function particularly in heart failure and cardiogenic shock. Furthermore, the slope of the function curve after volume expansion indicates the reserve of the myocardium. The observation that improvement of the ventricular-function curve can occur without treatment in the first 3 days is important in assessing the effect of any pharmacologic intervention. Since the optimum cardiac output observed from the function curve occurs at a filling pressure between 20 and 24 mm Hg, the use of diuretics can be selected on a precise physiologic basis. The assessment of ventricular function by the dextran infusion not only provides information on the cardiac performance, but the intervention itself can be hemodynamically beneficial.

Serial monitoring of cardiovascular dynamics in acute myocardial infarction probably represents the most useful clinical application of the hemodynamic techniques described. Not only can serial changes be related to baseline measurements, but again the derangements in hemodynamics can be useful in the selection of pharmacologic agents. In heart failure and cardiogenic shock indications for volume expansion or reduction can be precisely identified with the information provided by continuous monitoring of pulmonary artery pressure, cardiac outputs, and systemic blood pressure. In patients with cardiogenic shock the control values of various hemodynamic measurements can be followed more closely with various drugs, and the lack of hemodynamic response can be appreciated more readily. Although patients with mild infarction may reveal definite hemodynamic abnormalities, the generally uncomplicated course and favorable prognosis do not seem to justify widespread application of such monitoring techniques in these situations. However, the experience of several studies now indicates that a left ventricular protodiastolic gallop correlates fairly consistently with abnormal elevations in left ventricular filling pressure and may be the earliest indication for hemodynamic monitoring in acute myocardial infarction. As mentioned earlier, the presence of rales in the chest does not consistently relate to hemodynamic abnormalities, and the rise in central venous pressure in most instances is a very late manifestation of left ventricular failure. Studies further suggest that changes of pulmonary vascular congestion on X-ray may be out of phase with left ventricular and pulmonary artery dynamics. Therefore, the hemodynamic monitoring can provide the most accurate indication for the changes in cardiac function that occur during the first few days of infarction.

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