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(Part 5)

Hemodynamic Spectrum of Myocardial Infarction and Cardiogenic Shock
A Conceptual Model

By H. J. C. Swan, M.B., Ph.D., James S. Forrester, M.D., George Diamond, M.D., Kanu Chatterjee, M.R.C.P., and William W. Parmley, M.D.

SUMMARY
Despite the recent accumulation of a large hemodynamic data base describing myocardial infarction and cardiogenic shock, precise characterization of patient subsets has been elusive. This paper represents an attempt to identify the major factors contributing to this wide hemodynamic spectrum, and their interrelation using a theoretical model based upon currently emerging concepts of this disease. It is proposed that the hemodynamic alterations associated with acute infarction are a consequence both of reduction in contractile mass and alteration in left ventricular compliance. In addition, mitral insufficiency, altered contractility, and the peripheral circulation interact to produce wide divergence between clinical and hemodynamic features from case to case and during the progression of the course of the illness. This model may more rationally explain the genesis and natural history of "heart failure" and the "shock syndrome" associated with acute myocardial infarction and in addition explain the extremely variable responses to both drug therapy and to more aggressive modes of treatment of power failure.

POWER FAILURE of the heart and its extreme manifestation, cardiogenic shock, remains the single most important cause of hospital mortality in acute myocardial infarction. This paper will deal with current concepts regarding power failure, as they are presently understood and employed in the Cedars-Sinai Myocardial Infarction Research Unit. To this end, the paper will review the definition of cardiogenic shock and its variable presentation as determined by structural pathology. Arguments supporting the need for significant alterations in present concepts and a theoretical model of acute myocardial infarction will be proposed. The application of this model to understanding of known hemodynamic alterations which accompany shock and its implications concerning therapy will be described.

The shock state must be regarded as a clinical syndrome. In this context, the Myocardial Infarction Research Units of the National Heart and Lung Institute (Resnekov L, Chairman: Unpublished data, April 1971) have agreed on the following definition:
(1) A systolic arterial pressure less than 90 mm Hg or 30 mm Hg below the previous basal level.

(2) Evidence of reduced blood flow as shown by (all should be present):
   (a) Urine output less than 20 ml/hr, preferably with a low sodium content.
   (b) Impaired mental function.
   (c) Peripheral vasoconstriction associated with a cold, clammy skin.

Specifically excluded are hypotension related to pain, to vasovagal reaction, to serious rhythm disturbances, drug reactions, or hypovolemia. Hence the term "cardiogenic" is used to designate the occurrence of the shock syndrome by reason of primary cardiac dysfunction.

Although the use of a standard definition is mandatory for comparison of data from different institutions and evaluation of therapy, very real problems remain in the practical application of any such definition. Thus, central arterial pressure may exceed the apparent cuff blood pressure by 10-20 mm Hg so that patient classification is in part determined by how pressure is measured. Urine volume may be normal or cerebral function may be preserved in individuals who otherwise appear to have unquestionable shock. In a similar manner, it has not proved possible to describe the shock syndrome in terms of precise hemodynamics, although cardiac function and stroke work are in general depressed.1-3 Finally, there has been no definition of the temporal sequence of hemodynamic and metabolic changes in the evaluation of the shock syndrome, whether to ultimate survival or progression to death.

From a clinical standpoint, patients who develop shock may be divided into two groups. First, a group of patients is admitted to the hospital with severe circulatory collapse and hypotension. These patients die within minutes or early hours of their admission to the hospital. The young patient with a first infarction who develops shock is usually in this group. The second group develops shock while in the hospital. This may be secondary to an unexpected cardiac arrest with subsequent resuscitation, an extension of the area of infarction, or may present as a relatively slow deterioration with progressive hypotension, oliguria, and central nervous system depression. The latter frequently characterizes elderly patients who have had several previous myocardial infarctions.

It is evident that a series of widely differing clinical profiles may precede the development of the shock state.4 The nature, magnitude, and location of pathologic changes in the heart, the functional state of noninfarcted myocardium, the effectiveness of compensatory mechanisms, and the presence of additional factors which increase mechanical load are all involved. For example, total occlusion of the proximal portion of a major coronary artery will immediately render a large portion of the left ventricle ischemic and noncontractile. Clearly, this acute reduction in cardiac efficiency may be of such magnitude that survival for more than minutes or hours is impossible, and indeed many such patients die rapidly. Late shock deaths on the other hand appear to be associated with extensive three-vessel coronary artery disease and a history of previous myocardial infarction. Such hearts characteristically exhibit extensive myocardial destruction: "never" less than 40%, and as much as 70% of the myocardial mass may be destroyed prior to the death of the patient.5 Extension and reinfarction actually may be "dated" when the heart is examined at autopsy. In this group, the degree of initial depression of cardiac function is probably less than in those who succumb immediately. Nonetheless, a protracted course over several days is associated with progressive acidosis, further depression of cardiovascular function, the development of a thrombotic tendency, and finally total circulatory insufficiency. The threshold for the development of the shock state, characterized by loss of effective compensatory mechanisms, therefore may be seen as a product of the magnitude of initial insult and the duration of resulting circulatory insufficiency.

The shock syndrome may be precipitated in patients with acute myocardial infarction by

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secondary factors. Abnormalities of heart rate or rhythm, hypovolemia due to failure of fluid intake, or excessive fluid elimination (diuretics), hypoxemia, and acidosis all serve to depress cardiac function. The true extent of the myocardial factor in the shock syndrome cannot be identified until these factors have been corrected. If, following their correction, the patient continues to exhibit the shock syndrome, then and only then is a diagnosis of cardiogenic shock appropriate. It should be noted, therefore, that in terms of this concept the diagnosis of cardiogenic shock cannot be made in the presence of a decreased LV filling pressure.

Mechanical Model of the Infarcted Heart

The observation of a normal or reduced level of LV stroke work or stroke volume in the presence of an elevated LV filling pressure is commonly regarded as evidence for left ventricular failure. Such data imply a generalized decrease in LV performance, and are appropriate to conditions such as cardiomyopathy or essential hypertension. This concept of power failure has been applied to all forms of heart disease, and is validated by the usual presence of clinical signs of rales and gallop sounds. Patients with acute myocardial infarction, however, may have a normal or increased cardiac output in the presence of pulmonary venous congestion and ventricular gallop sounds. These individuals are generally considered to have “heart failure,” although the majority respond poorly or not at all to inotropic agents such as digitalis. The concept of uniform depression of LV function is also belied by recent angiographic studies of patients with cardiogenic shock, in which areas of myocardium contract vigorously and coexist with large areas of akinesis, paradoxical pulsation, or mitral insufficiency. Since the traditional concept of LV failure, based on an assumed uniform depression of left ventricular function, does not explain these observations, it must be critically reexamined.

To the present, little attention has been paid to the description of the alterations in structure and function in the different parts of the heart following acute infarction. Even such relatively basic variables as anatomic location and the extent and severity of damage have been slow to be reported in detail. Recently, it has been recognized that less than 60% of histologically normal myocardium remains in those patients with acute infarction who succumb to the shock syndrome. Experimental evidence suggests that the residual noninfarcted muscle frequently contracts normally or even at an enhanced level, while the infarcted tissue contracts poorly if at all. Other studies have revealed an alteration in ventricular diastolic compliance, the basis for which is presumed to reside in the passive length-tension characteristics of infarcted muscle. These considerations are highly relevant to the hemodynamics of myocardial infarction and the shock syndrome and are deserving of detailed elaboration.

Because of the inadequacy of the unitarian model, we propose a two-component model of the heart in acute myocardial infarction. This spherical model consists of: (1) a totally noncontractile infarct of variable size, possessing an altered compliance, the temporal and directional course of which remains to be defined in detail; and (2) a residual noninfarcted area with normal contractility, which is able to respond normally both to increases in contractile state and to fiber stretch; this area possesses normal compliance. For the purpose of simplicity, we will limit discussion to a nondilated, nonhypertrophied ventricle with a first infarct and will not consider the behavior of the transitional or “twilight” zone, containing fibers with a wide spectrum of contractile function. In addition, it is assumed that LV function is independent of the anatomic location of similarly sized infarcted segments. Similar general considerations have been applied to chronic coronary artery disease. While several workers have utilized contractile element shortening or velocity of circumferential fiber shortening in the analysis of muscle function, we are herein concerned primarily with the function of the heart as a pump. Hence we have selected the
ratio of stroke volume (SV) to end-diastolic volume (EDV)—the ejection fraction (EF)—as the index of cardiac performance most appropriate to our purposes.

A. Infarct Size

The immediate mechanical alterations following myocardial infarction are illustrated schematically in figure 1. This analysis assumes normal myocardial contractility prior to infarction and, therefore, an EF of 0.67 for the normal noninfarcted ventricle. Such a ventricle would have an EDV of 90 ml/m², an end-systolic volume (ESV) of 30 ml/m², and an SV of 60 ml/m². The area of infarct is considered to be completely noncontractile.

**Figure 1**

Hemodynamics of a noncompliant infarct. (A) Relationship of EF, EDV, and SV to infarct size. The schematic diagram represents the heart in systole and diastole, and the infarct segment (delineated by the solid bars to the right of the inset) is assumed to be totally noncontractile and nondistensible. The resulting EF is a linear function of the remaining quantity of normally contracting myocardium (see text). Thus, with an infarct of 10% (circle), EF would fall from 0.67 to 0.60, and with a 40% infarct to 0.40 (diamond). (B) Left ventricular function curves 10% and 40% nondistensible infarct segments are associated with reductions in EF and SV at a constant EDV of 90 ml/m², and hence a downward shift in function curve representing the left ventricle as a whole. (C) The relationship between EF, EDV, and SV. When EF is reduced by acute myocardial infarction, this may result in either a decreased SV at an unchanged EDV or a compensatory increase in EDV to maintain SV (see text). (D) The obligatory changes in the pressure-volume relationships consequent on a noncompliant infarct. It is assumed that the infarct is rigid in the contracted state. The normal pressure-volume curve is based on values for normal heart volumes and normal diastolic pressures, extrapolated in form according to acute immediate postmortem pressure-volume curves obtained from normal canine hearts.

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and, for the present, absolutely nondistensible. If the model is considered as an infinite number of cones, each based on the surface of the sphere and with its apex at the center, the EF is reduced in direct relation to the proportion of inert elements (fig. 1A). It follows then that there is a linear reduction in EF as the size of the infarct increases. If EDV remains constant, the overall performance of the ventricle, depicted in the form of a classic ventricular function curve, is depressed; however, the contractile state of the noninfarcted myocardial fibers has remained unchanged.

For purposes of comparison, a small infarct will be considered as equivalent to destruction of 10% of LV mass, while a large infarct corresponds to 40% destruction. From figure 1A, an EF of 0.60 would be predicted following a small infarct, while the corresponding EF for a large infarct would be 0.40. To maintain a normal level of external cardiac performance, stroke volume must be increased either through an augmentation of contractile state in the residual normal muscle, by an increase in end-diastolic volume, by a decrease in afterload, or by some combination of these mechanisms. Failing these compensations, cardiac performance must, of necessity, decrease proportionately with increasing infarct size (fig. 1B).

B. Pressure and Volume Requirements to Maintain Stroke Volume at Normal Contractility

Figure 1C describes the relation between EF, EDV, and SV for a 10 and 40% infarct of infinite stiffness (noncompliant). Isopleths of stroke volume are depicted as hyperbolic functions of the axes. It is seen that the development of a 10% infarct produces only a minimal decrease in total cardiac function. If EDV remains unchanged, SV is reduced from 60 to 55 ml. Alternatively, a 9% increase in EDV would allow maintenance of a normal SV. However, the replacement of contractile compliant elements by noncontractile elements which are also noncompliant has a profound effect on the pressure-volume relationship, also in direct relation to the infarct size (fig. 1D). For a 10% infarct at constant EDV, EDP rises from 10 to 12 mm Hg, although maintenance of SV by increasing EDV to 100 ml is accompanied by a rise in EDP. With the development of a 40% infarct and a normal EDV, SV would decrease from 60 to 37 ml, while EDP would increase from 10 to 20 mm Hg. Restoration of SV would require an EDV of 140 ml which is not attainable within the normal range of physiologic pressures.

C. Relationship of Compliance to Ventricular Performance

We will now examine the consequences of the retention of normal compliance characteristics by the infarcted segment, in contrast to the hemodynamics associated with an infarct of infinite stiffness (fig. 2). Since the infarcted segment does not participate in the process of contraction, it is subjected to repetitive passive stretch during each systole as intraventricular pressure rises (fig. 2B). Hence this segment will lengthen to a degree dependent upon its intrinsic length-tension characteristics and the level of systolic pressure. The fibers of the healthy myocardium are protected from stretch by the intrinsic shortening of their contracting sarcomeres (fig. 2A). If the fibers of the infarcted segment follow the normal passive length-tension curve then LV function is further compromised as infarct size increases. Assuming normal compliance to be represented by a ratio of segment length at systolic pressure (PS) divided by segment length at end-diastolic pressure of 1.15 (15% increase in length), then the equivalent change in volume SV/VEDP would be 1.15 or 1.52. On this basis, EF would decrease further to 0.57 and 0.27 for a small and large infarct, respectively, in the presence of normal compliance. In the presence of normal heart size such ejection fractions will permit stroke volumes of 51 and only 24 ml, respectively (fig. 2D). Although the 10% infarct heart is capable of further adjustment via the Starling mechanism (EDV = 100 ml/m²; EDP = 17 mm Hg) (see fig. 1D), the consequence of normal compliance becomes very substantial with infarcts in excess of 30% of LV mass. With a 40% infarct, the stroke volume
Hemodynamics of a compliant infarct. (A) Assuming that one half the spherical model is infarcted, the left ventricular pressure-volume relationship during systole and diastole in the noninfarcted cardiac hemisphere illustrates a normal pressure-volume loop and results in a ΔV (or SV) of 30 ml/m². (B) If the noncontractile infarct segment (right panel) exhibits normal compliance, it will stretch during ventricular systole, according to its passive pressure-volume curve with resultant paradoxical expansion, so that for normal pressure ranges the increase in volume during systole will be approximately equal to the decrease in volume of the contracting hemisphere. If the pressure-volume curve is less steep the penalty is even greater. Forward SV can only be maintained with an increased end-diastolic and a reduced peak systolic pressure. In this model a forward SV of 15 ml/m² is possible at an EDP of 25 mm Hg and a peak systolic pressure of 70 mm Hg. (C) Effect of normal compliance characteristics with paradoxical pulsations on EF. Without outward movement of the infarct segment during systole, a portion of the potential systemic SV will be delivered into this segment. Thus, EF will be further reduced in the presence of a compliant infarct segment (circles for 10% infarct, diamonds for 40% infarct). The values are estimated for peak systolic pressures at the low range of normal. (D) Interrelationship of EF, SV, and EDP. In the presence of paradoxical pulsation, reduction in EF is associated with a decrease in SV exceeding that predicted for a noncompliant infarct as shown in figure 1A. When EDP remains unchanged in the presence of normal overall ventricular compliance, LVEDP is also unchanged (fig. 1D, open circle). The SV of 51 shown here in D may be returned to normal levels by compensatory ventricular dilatation only if the infarct is small (10% infarct). This rise in EDV produces a concomitant rise in LVEDP (fig. 1D, square symbol).

decreases to 24 ml at an EDP and EDV identical to the noninfarcted heart and cannot be improved significantly without substantial increase in EDV. Even if EDV is increased to

Figure 2

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the level associated with a filling pressure of 20 mm Hg, stroke volume would rise only from 24 to 29 ml, in comparison to 37 ml when the infarct was noncompliant.

D. Filling Pressure and Ventricular Function Curves

The filling pressure of the LV depends upon the ESV, the volume of diastolic inflow which usually equals SV, and the stiffness of the LV wall. Assuming a normal EDV, a change in EDP will occur in relation to increased infarct size according to the compliance characteristics of the infarcted segment and of the normal myocardium. The effect of this change on LV function curves based on filling pressure was shown in figure 1D. If a 40% infarct fails to stiffen, EDP and EDV would remain unaltered but SV would be reduced to a greater extent than that predicted from infarct size alone, as previously discussed. Stiffening of the infarct would result in true improvement in ventricular function by elimination of an area of systolic expansion, but would be accompanied by an increase in EDP (fig. 3). This apparent shift in the ventricular function curve occurs in the absence of changes in contractility. Thus, changes in ventricular function may result from alteration in either LV compliance or contractility. In states where compliance may vary, therefore, interpretation of ventricular function curves may be particularly misleading when ventricular pressure is used as an index of ventricular volume.

E. Alteration of Contractile State

The above considerations indicate that early mechanical changes may be sufficient to explain the alterations of cardiac performance in the shock syndrome, as evidenced by reduced stroke volume and stroke work with variable filling pressures. Depression of the contractile state of the noninfarcted myocardium, although certainly a potential factor in the genesis and persistence of the shock state, has not been described with clarity. It would appear, however, that the contractility of noninfarcted muscle is not usually depressed in animals or man in the early hours following infarction. In this respect, the enhancement of performance is related to preexisting contractile state of the myocardium. Hence, while some increase in cardiac performance is possible by augmentation of contractility the potential for improvement to influence external function is progressively reduced with increasing size of the infarct (fig. 4A). When a high level of circulating catecholamines exists (as in myocardial infarction) the contractile state of the residual healthy myocardium may be at, or close to, maximum. Many patients with acute myocardial infarction have a maximum LV dp/dt and contractile element velocity (\(V_{CE}\)) close to or within the normal range.23-25 In those patients in whom severe depression of \(V_{CE}\) or of LV dp/dt has been demonstrated, clinical shock is usually evident, and survival is uncommon.23 The normal or enhanced contractility of surviving myocardium reduces the likelihood that the general therapeutic use of inotropic agents can materially improve overall cardiac function in patients with large myocardial infarcts.

![Figure 3](http://circ.ahajournals.org/)

Figure 3

The effects of normal, or of absence of, compliance in a 40% infarct. If the infarct retains normal compliance, the EDP remains unchanged but SV is profoundly reduced and cannot be significantly increased by elevation of EDP. For the noncompliant infarct, the higher SV is accompanied by a marked increase in EDP. However, a small reduction in EDV will substantially reduce EDP (fig. 1D) without major sacrifice of SV.

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F. Added Mechanical Load

Acute mitral insufficiency due to rupture or severe dysfunction of a papillary muscle and acute perforation of the ventricular septum are well-recognized complications of acute myocardial infarction, usually considered to be infrequent. The already compromised heart is unable to maintain a normal filling pressure in the face of the added regurgitant or shunt burden, and acute pulmonary edema and death occur frequently. Although the physical signs of mitral insufficiency have been observed in a substantial percentage of patients with acute myocardial infarction, it has usually been considered of mild degree and not of great hemodynamic significance. Conversely, however, hemodynamically severe mitral insufficiency may occur in the shock syndrome even in the absence of a detectable cardiac murmur.

The severity of mitral insufficiency has been quantitated by calculation of the regurgitant fraction. This value, \[ RF = \frac{\text{Forward SV}}{\text{EDV} - \text{ESV}} \], expresses the volume regurgitated into the left atrium as a fraction of the total volume ejected by the left ventricle. In patients with rheumatic heart disease, clinically mild mitral insufficiency...
insufficiency corresponds to an RF less than 0.4; moderate, 0.4-0.6; and severe, greater than 0.6. Alteration in the ventricular function curve secondary to moderate mitral regurgitation is illustrated in figure 4B. As with an infarct of normal or increased compliance, mitral insufficiency places an added mechanical burden on the already damaged heart. This added load is of major significance even in the presence of a small infarct, while with larger infarcts, or in a heart compromised by previous disease, the resultant reduction in cardiac performance may become rapidly incompatible with life.

The mechanical burden of either mitral insufficiency or aneurysmal paradoxical movement is probably more complex than simple loss of forward stroke volume. In the normally contracting heart there are only minor changes in shape during isovolumic systole, with minimal fiber shortening. The tension associated with increasing intraventricular pressure is stored in the series elastic elements of muscle while the sarcomeres shorten uniformly. In the presence of mitral insufficiency or aneurysm, however, the unaffected fibers actually shorten during early systole, resulting in a slight reduction in length of the healthy fibers. This may result in a minor reduction in wall stress and oxygen consumption according to the LaPlace relation. This beneficial effect, however, does not offset the obligatory increase in EDV required to maintain an adequate forward flow. In addition, this abnormal shortening of the normal fibers during early systole may place them at a less optimal position for the production of useful work when opening of the aortic valve occurs.

G. Temporal Relationships

The foregoing model has considered several fundamental physiologic and mechanical factors which are responsible for many aspects of altered cardiac performance that characterize cardiogenic shock. Infarct size, contractility, compliance, mechanical load, and heart size each has a temporal course during evolution of acute myocardial infarction. In contrast to the substantial amount of information describing a single point in that course, serial description of these parameters is extremely limited. Much of the following discussion is therefore based upon inference.

Contractility. The most reasonable temporal sequence for the noninfarcted segment would appear to be as follows: Immediately after infarction, no change in contractile state occurs. With stimulation of catecholamine secretion by pain or diminished tissue perfusion, increased contractility of the noninfarcted muscle usually occurs. Aside from iatrogenic alteration, subsequent changes in contractile state relate largely to infarct size. If the patient survives, contractility of the surviving myocardium probably returns toward normal. In nonsurvivors, maximal stimulation of the surviving myocardium is insufficient to sustain life. With loss of all compensatory mechanisms, the quantity of viable muscle is progressively reduced, and its contractility may fall to low levels just before death.

Compliance. In the minutes and early hours following acute myocardial infarction, it seems reasonable to assume that apart from electrolyte and water shifts there is no substantive change which occurs in the supportive elements within the myocardial wall. Those structural elements which are the principal determinants of the passive length-tension relationship, namely the elastic and fibrous tissue, the intercalated discs and the sarcomeruellar membrane clearly cannot immediately increase in number or undergo significant repair. Nevertheless, clear-cut functional changes occur immediately. Direct observations in the operating room and the experimental laboratory indicate that systolic bulging of the area of infarction, coinciding with contraction of the surrounding healthy muscle, develops rapidly with cessation of contraction in the infarct area. Furthermore, pressure-volume relationships determined in our laboratory 1 hour following experimental myocardial infarction indicate that the compliance of the LV as a whole is slightly increased above normal. This probably relates to stress relaxation of the noncontracting infarct tissue, which is repeatedly passively stretched during systole.
Compensatory mechanisms for stress relaxation may exist, particularly in smaller infarcts. If some sarcomeres in the infarct segment remain contractile, they may protect the passive elastic elements from excessive stretch and so prevent stress relaxation. If the hypoxic sarcomeres undergo contracture, or when edema fluid accumulates within infarcted tissue, rapid decreases in compliance may occur. For this reason, the time course of compliance changes following myocardial infarction is probably variable although primarily related to infarct size. The eventual result, however, in surviving patients with acute myocardial infarction, is significant stiffening of the LV wall. Although infarcted tissue removed at surgery from human hearts as late as 1 week after infarction may exhibit normal passive length-tension curves, material removed from patients with chronic ventricular aneurysms invariably is minimally distensible. In animals, stiffening of the area of infarction after single-vessel occlusion occurs by the third to fifth postinfarct day, but extrapolation of such data to man with generalized coronary vascular disease must be made with caution.

The temporal sequence of compliance changes is intimately related to the mechanical consequences of acute myocardial infarction. If the overall contractile property of the infarct is so depressed that paradoxical pulsation occurs, then stress relaxation may result in a vicious cycle of dilatation, increased intramyocardial tension, increased regional myocardial hypoxia, and further infarction. However, if and as the infarct stiffens, the mechanical advantage to the heart is obvious. In such cases even patients with large infarcts may generate an adequate stroke volume from a normal end-diastolic volume, albeit at an elevated filling pressure. To attain similar performance with normal compliance requires a marked, possibly unattainable, increase in end-diastolic volume and pressure.

**Cardiac Dilatation.** In the presence of a large infarct, the heart finds itself with the options of increasing contractile state, or undergoing dilatation to maintain performance. Each of these options is costly in terms of myocardial oxygen consumption. Furthermore, the degree of acute dilatation of which the heart is capable is restricted to fairly narrow limits by ventricular compliance. Perhaps for this reason, serial changes in heart size are not easily documented in acute myocardial infarction, and progressive increase in heart size carries a very poor prognosis.

**Application of the Myocardial Infarction Model to Known Hemodynamics**

Table 1 demonstrates the average of the values of a number of hemodynamic descriptors in patients with acute myocardial infarction with and without the clinical shock syndrome. These values are similar to those previously reported by others. Figure 5

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Shock Mean ± 1 sd</th>
<th>No. pt</th>
<th>Nonshock Mean ± 1 sd</th>
<th>No. pt</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>94 ± 17</td>
<td>30</td>
<td>89 ± 21</td>
<td>93</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>71 ± 20</td>
<td>27</td>
<td>100 ± 21</td>
<td>86</td>
</tr>
<tr>
<td>Cardiac output (liters/min)</td>
<td>2.5 ± 0.8</td>
<td>22</td>
<td>4.6 ± 1.2</td>
<td>53</td>
</tr>
<tr>
<td>LV filling pressure (mm Hg)</td>
<td>27 ± 8</td>
<td>18</td>
<td>15 ± 8</td>
<td>68</td>
</tr>
<tr>
<td>LV dp/dt (mm Hg/sec)</td>
<td>710 ± 216</td>
<td>8</td>
<td>1941 ± 1067</td>
<td>9</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>27 ± 11</td>
<td>22</td>
<td>55 ± 20</td>
<td>53</td>
</tr>
<tr>
<td>Stroke work (g-m/beat)</td>
<td>26 ± 10</td>
<td>22</td>
<td>81 ± 36</td>
<td>52</td>
</tr>
<tr>
<td>Total systemic vascular resistance (dyne-sec-cm^-2)</td>
<td>2530 ± 1190</td>
<td>22</td>
<td>1880 ± 580</td>
<td>52</td>
</tr>
</tbody>
</table>

*Observations were made on admission on a total of 123 patients with acute myocardial infarction.

†P < 0.005.
demonstrates the relation between stroke-work index and left ventricular filling pressure in our patients with myocardial infarction. We have included the range seen in normal subjects and the predicted relation in a 40% noncompliant infarct. Several important conclusions may be derived from consideration of these data which express the relationship between left ventricular filling pressure and function. First, some patients have ventricular function within or above the usual range of normal in the presence of an acute myocardial infarction. In terms of the myocardial infarction model, these patients would be expected to have infarcts of small size which resulted in no significant alteration in ventricular compliance and thus normal filling pressures. The effect of a small infarction upon ejection fraction would also be negligible. In those individuals with increased catecholamine excretion, ventricular function would even exceed the normal range.

Second, individuals with levels of stroke work which are normal or somewhat reduced (<40 g·m/m²) may have a wide range of LV filling pressures, although there is a trend toward a negative correlation. From consideration of the infarction model, it is clear that an isolated decrease in LV compliance may result in an increase in filling pressure with no change in stroke work or true LV function. Since the mean left ventricular filling pressure, transmitted retrograde to the left atrium and pulmonary venous circulation, is the hemodynamic determinant of pulmonary venous congestion, it is possible to have clinical evidence of “heart failure” in the presence of normal left ventricular function. Conversely, it is clear that findings on physical examination may be inadequate to indicate the true state of left ventricular function, a fact which is being reluctantly admitted in a number of recent publications in which hemodynamic and physical parameters have been correlated.

Third, stroke-work levels in many patients with acute myocardial infarction fall below that predicted for a 40% infarct, based upon the infarct model. In all patients with cardiogenic shock, left ventricular stroke work was markedly depressed, and many of these patients had increased LV filling pressure. Several possible explanations may be advanced. Some or even all patients clearly either had an infarct which effectively exceeded 40% of the myocardium or were more compliant than the values suggested in our model. In others a mechanical lesion such as mitral insufficiency added substantially to the reduction of external left ventricular work. True depression of the contractile state of the noninfarcted myocardium also may be an intermediate or preterminal characteristic.

Since stroke work is determined by the relationship between cardiac function and peripheral response, it is to be anticipated that shock is persistently reflected by marked reductions in stroke work. This relationship is shown graphically in figure 6. Values for SV
and mean arterial pressure were available in 22 patients with the shock syndrome. However, LV filling pressure was measured in only seven. Several of the remaining patients had hypovolemia, and responded to fluid loading, with seven survivals. Strictly speaking, the shock syndrome was of mixed etiology in these patients and the term cardiogenic shock may not be truly applicable. As a group, the shock patients are characterized by stroke-work values of less than 40 g-m/beat, but not particularly by the level of total peripheral vascular resistance (PVR) (table 1). It seems likely, therefore, that alterations in PVR are a secondary phenomenon. In our experience, low PVR is most commonly seen in patients at two extremes of the shock spectrum: in relative hypovolemia and in preterminal states just prior to death. Furthermore, marked and rapid reduction in PVR and in the state of organ perfusion have been observed following initiating of successful mechanical circulatory assistance in certain patients. This indicates that the changes in PVR are secondary to the profound deficits of pump function, at least in the early stages of shock.

Compensatory Mechanisms

Maximal cardiac compensation may be reflected by the maximal oxygen consumption or maximal heart rate response to exercise, which both decline with age. It must be assumed that the maximal cardiac compensation is an age-dependent phenomenon (figure 7). This figure depicts in schematic form the likely consequences on maximum cardiovascular function of previous myocardial damage and age. The incidence of shock is common in older patients and in those who have had previous myocardial infarction. This may be related to the lesser reserve for effective compensation available to such patients.

Implications of the Myocardial Infarction Model for Rational Therapy

The use of drugs in patients with cardiogenic shock is discussed in another paper in this symposium. However, from the standpoint of our two-component model, inotropic drugs are not likely to be particularly efficacious since they are unlikely to affect either the contractility or compliance of the infarcted myocardium. If the residual myocardium is already in either a normal or hypercontractile state, then the poor response to inotropic agents usually experienced clinically can be readily understood. Inotropic drugs would not be expected to be of benefit unless the contractile state of the noninfarcted myocardium were depressed. The occasional marked improvement associated with the use of inotropic agents may serve to identify that small group of patients in whom significant, reversible, intrinsic depression of contractile state exists.

The fundamental problem in acute myocardial infarction remains acute hypoxia of a segment of myocardium with resultant changes in the contractility and compliance of that segment. Restoration of blood flow by revascularization of this segment improves overall ventricular function by adding contracting muscle mass. In addition it is now clear that this procedure also alters ventricular compliance. Indeed, we have observed a marked decrease in myocardial stiffness in patients following successful revascularization.
HEMODYNAMIC SPECTRUM OF MI

The effect of age upon compensatory mechanisms in acute myocardial infarction. Schematically illustrated is the temporal change in maximal cardiovascular performance and the influence of myocardial infarctions in a hypothetical patient. Maximum cardiac capacity normally decreases progressively from the age of 20 years. At 50 years of age, the development of acute myocardial infarction produces a significant transient decrease in maximum cardiac capacity with subsequent return to a level commensurate with residual myocardial damage. Later myocardial infarctions produce further decreases in cardiac capacity to levels productive of congestive heart failure, shock, and ultimately death.

Conversion of a muscle from a noncontracting to a contracting segment also tends to eliminate the deleterious effects of repeated stretch during systole. Particularly in a large infarct these ischemic but viable myocardial fibers may be repeatedly stretched by more normal contracting fibers. At higher peak pressures, stretch and tension are greater, increasing the likelihood of destruction of the myocardial supportive elements, closure of residual blood vessels, and further impairment of blood supply to the infarcted area. The permanent sequela of such processes is likely to be aneurysm formation.

Reduction in left ventricular peak pressure by the prevention of systemic hypertension should also minimize stress relaxation. Implicit therefore in this argument is the possible beneficial effects of a limitation on peak systolic pressure and the translation of the available but limited ventricular stroke work into volume work as effectively as possible. In this context, it is presently our policy to treat hypertension in the presence of acute infarction, rather than allow pressure to remain high "to maintain organ perfusion," a clinical practice of dubious benefit.

In addition to the many previously described effects of mechanical circulatory assistance, compliance of the infarct segment may be altered in a manner similar to revascularization. Small quantities of oxygenated blood may reach hypoxic muscle via collaterals during the augmented diastolic phase of the cardiac cycle. Counterpulsation could also be of benefit in reducing left ventricular peak systolic pressure by rapid withdrawal of blood from the aorta during systole, and thus reducing both the wasteful systolic expansion of the infarcted segment and the magnitude of stress relaxation.

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H. J. C. SWAN, JAMES S. FORRESTER, GEORGE DIAMOND, KANU CHATTERJEE and WILLIAM W. PARMLEY

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