EVALUATION OF CARDIAC PERFORMANCE AFTER

ACUTE MYOCARDIAL INFARCTION

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MEDICAL GRAND ROUNDS

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I. Introduction

1. Shillingford, J. P. and Thomas, M.: Cardiovascular and pulmonary changes in patients with myocardial infarction treated in an intensive care and research unit. Am. J. Cardiol. 20:484, 1967.
2. Weil, M. H. and Shubin, H.: Shock following acute myocardial infarction; current understanding of hemodynamic mechanisms. Prog. Cardiovas. Dis. 11:1, 1968.
3. Editorial: Left ventricular function in acute myocardial infarction. Lancet 2:159, 1968.
4. Rapaport, E. and Scheinman, M.: Rationale and limitations of hemodynamic measurements in patients with acute myocardial infarction. Mod. Conc. Cardiovas. Dis. 38:55, 1969.
5. Karliner, J. S. and Ross, J., Jr.: Left ventricular performance after acute myocardial infarction. Prog. Cardiovas. Dis. 13:374, 1971.
6. Rackley, C. E. and Russell, R. O., Jr.: Left ventricular function in acute myocardial infarction and its clinical significance. Circulation 45:231, 1972.
7. Cohn, J. N.: Bedside evaluation of left and right ventricular function in myocardial infarction. Submitted for publication.
8. Russell, R. O., Jr., Hunt, D., Potanin, C. and Rackley, C. E.: Hemodynamic monitoring in a coronary intensive care unit: clinical application. Submitted for publication.

The continuous or serial monitoring of physiological events in patients after acute myocardial infarction is an important advancement in medical care. The continuous recording of the electrocardiogram has played an indispensable role in the significant decline of mortality caused by electrical malfunction or arrhythmias of the heart. This can be attributed to the prompt recognition and early treatment of these abnormalities in rhythm. Mechanical malfunction or failure of the heart as a pump in the circulation now represents the most important cause of death in patients in the coronary care unit. For this reason interest is currently being focused on identifying those cardiovascular physiological parameters that can be best used to evaluate cardiac performance after acute myocardial infarction. The ability to continuously monitor such functions and the ability to recognize impending pump failure immediately are extremely important in determining when to use therapeutic interventions such as volume expansion, diuresis, inotropic agents (digitalis and catecholamines), cardiac assist devices, and emergency surgery.

II. Invasive Techniques

    A. Cardiovascular pressure measurements

1. Cardiac filling pressures

a. Right ventricular filling pressure

4. Loc. cit.
9. Landis, E. M. and Hortenstine, J. C.: Functional significance of venous blood pressure. Physiol. Rev. 30:1, 1950.
10. Cohn, J. N.: Monitoring techniques in shock. Am. J. Cardiol. 26:565, 1970.
11. Cohn, J. N.: Central venous pressure as a guide to volume expansion. Ann. Int. Med. 66:1283, 1967.
12. Borow, M. and Escaro, R.: The reliability of central venous pressure monitoring and errors in its interpretation. Surg., Gynec., & Obstet. 127:1228, 1968.

In cardiovascular dynamics right ventricular end-diastolic pressure is the filling pressure of the right ventricle. To obtain this measurement not only does a catheter have to remain in the right ventricle but also a high fidelity recording system must be used. However, with the exceptions of tricuspid disease and right atrial failure, mean right atrial pressure may be used to represent right ventricular filling pressure. Clinically the measurement of right atrial pressure or of pressure in the great veins well within the thoracic cavity is called the *central venous pressure*.

The filling pressure of the right heart or central venous pressure is the result of several interacting factors including the tone of the systemic venous capacitance vessels, the distensibility of the right ventricle, the contractile state of the right ventricle, the blood volume contained within the central veins, and the intrathoracic pressure. In essence, however, central venous pressure represents the result of the conflict between the venous return on the right side of the circulation and the resistance to right ventricular filling.

b. Left ventricular filling pressure

4. Loc. cit.
13. Cohn, J. N., Khatri, I. M. and Hamosh, P.: Bedside catheterization of the left ventricle. Am. J. Cardiol. 25:66, 1970.
14. Swan, H. J. C., Ganz, W., Forrester, J., Marcus, H., Diamond, G. and Chonette, D.: Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. New England J. Med. 283:447, 1970.
15. Rutherford, B. D., McCann, W. D. and O'Donovan, T. P. B.: The value of monitoring pulmonary artery pressure for early detection of left ventricular failure following myocardial infarction. Circulation 43:655, 1971.

Just as right ventricular end-diastolic pressure is hemody-
namically the filling pressure of the right ventricle, left ven-
tricular end-diastolic pressure is the filling pressure of the
left ventricle. Basically, this pressure represents the interplay
between blood returning from the lungs to the left side of the cir-
culation and the resistance to left ventricular filling.

Left ventricular end-diastolic pressure is also an extremely
difficult measurement to obtain and to accurately record in the
clinical setting. Even though methods have been developed to
place a catheter in the left ventricle without the need of fluoro-
scopic monitoring, measurements of pressure may be of inadequate
fidelity to determine left ventricular end-diastolic pressure. In
the absence of mitral disease and of left atrial failure, mean
left atrial pressure may be used to represent left ventricular
filling pressure. Continuous measurement of left atrial pressure
directly is not feasible in the patient being monitored in the
coronary care unit. However, pressure measurements can now be
made easily and safely in the pulmonary artery and its major
branches in patients being monitored after an acute myocardial in-
farction. This is due to the introduction of the Swan Ganz
catheter.

16. Connolly, D. C., Kirklin, J. W. and Wood, E. H.: The relation-
    ship between pulmonary artery wedge pressure and left atrial
    pressure in man. Circulation Res. 2:434, 1954.
17. Sapru, R. P., Taylor, S. H. and Donald, K. W.: Comparison of
    the pulmonary wedge pressure with the left ventricular end-
   diastolic pressure in man. Clin. Sci. 34:125, 1968.

Both pulmonary artery end-diastolic pressure or pulmonary
wedge pressure have been used as measures of left ventricular end-
diastolic pressure. However, pulmonary wedge pressure has been
shown more convincingly to represent mean left atrial pressure.

     c. Relationship between right heart and left heart
       filling pressure

18. Case, R. B., Berglund, E. and Sarnoff, S. J.: Ventricular
    function. II. Quantitative relationship between coronary flow
    and ventricular function with observations on unilateral
    failure. Circulation Res. 2:319, 1954.
19. Berglund, E.: Ventricular function. VI. Balance of left and
    right ventricular output: relation between left and right
    atrial pressure. Am. J. Physiol. 178:381, 1954.
20. Cohn, J. N., Tristani, F. E. and Khatri, I. M.: Studies in
    clinical shock and hypotension. VI. Relationship between left
    and right ventricular function. J. Clin. Invest. 48:2008,
    1969.
21. Ramo, B. W., Myers, N., Wallace, A. G., Starmer, F., Clark,
    D. O. and Whalen, R. E.: Hemodynamic findings in 123 patients

with acute myocardial infarction on admission. Circulation 42:567, 1970.

22. Forrester, J. S., Diamond, G., McHugh, T. J. and Swan, H. J. C.: Filling pressures in the right and left sides of the heart in acute myocardial infarction. A reappraisal of central-venous-pressure monitoring. New England J. Med. 289:190, 1971.

23. Cohn, J. N.: Monitoring the patient in cardiogenic shock. Submitted for publication.

Figure 1

NORMAL RELATION OF RIGHT AND LEFT CARDIAC FILLING PRESSURES
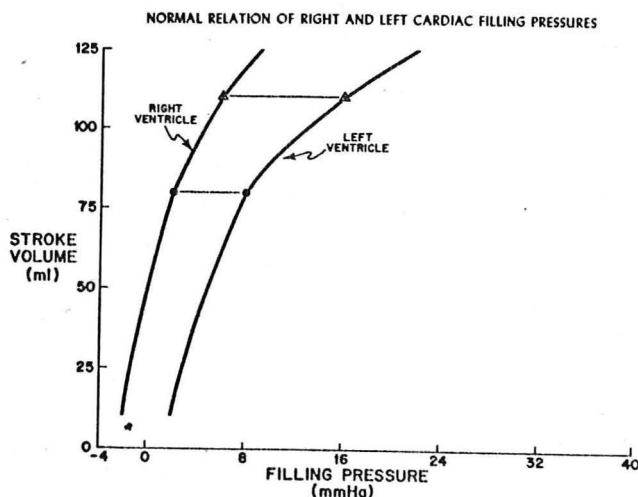
Under normal conditions with balanced function of the right and left ventricles, mean left atrial pressure is higher than mean right atrial pressure (Figure 1). Changes in blood volume when right and left ventricular function are both normal cause parallel changes in right heart filling pressure (central venous pressure) and left heart filling pressure (pulmonary wedge pressure). An increase in vascular fluid volume will cause a parallel increase in filling pressures and stroke volumes of both ventricles. This mechanism is responsible for maintaining the balanced blood volumes between the systemic and pulmonary circulatory systems, respectively. In cases of shock when there is either no effect or an equal effect on the function of the two ventricles, such as

hemorrhagic or septic shock, central venous pressure is a useful guide for the assessment of filling pressures of both ventricles. Therefore, under these conditions central venous pressure is a useful guide for volume therapy.

It has now been clearly shown that in patients after acute myocardial infarction, the central venous pressure does not accurately reflect the filling pressure of the left heart. Forrester, Diamond, McHugh, and Swan have recently published a plot of central venous pressure versus pulmonary wedge pressure measured simultaneously in patients after acute myocardial infarction and this is shown in Figure 2. It is readily seen that there is very little correlation between these two parameters.

Figure 2
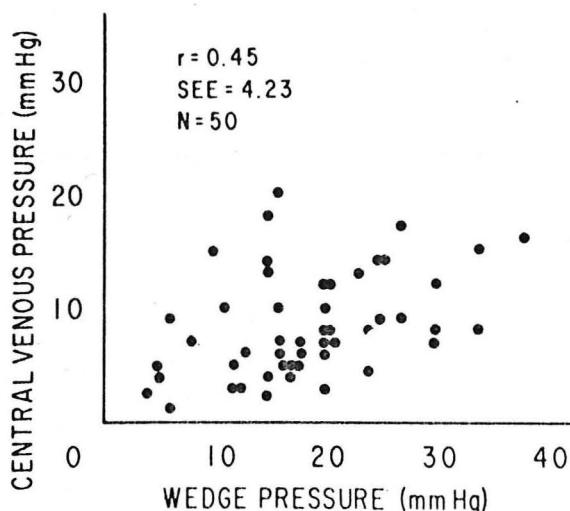
It has further been shown by Rapaport and Scheinman that changes in central venous pressure do not reflect simultaneous changes in pulmonary diastolic pressure and presumably pulmonary wedge pressure (Figure 3). They found a very poor correlation between changes in these two pressures after diuresis, volume expansion, and therapeutic interventions.

Figure 3
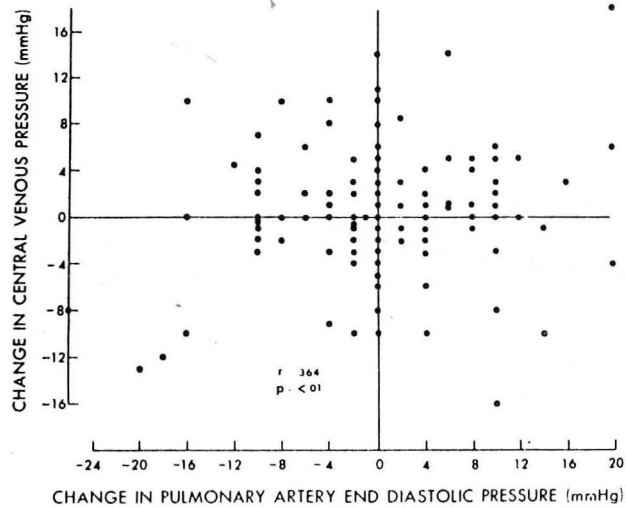
In most patients after an acute myocardial infarction there is principal involvement of the left ventricle with predominant left ventricular failure. The effect of acute myocardial infarction with predominant left ventricular failure on the relation between stroke volume and filling pressure of each ventricle is shown in Figure 4.

Figure 4

RIGHT AND LEFT CARDIAC FILLING PRESSURES AFTER ACUTE MYOCARDIAL INFARCTION WITH PREDOMINANT LEFT VENTRICULAR FAILURE
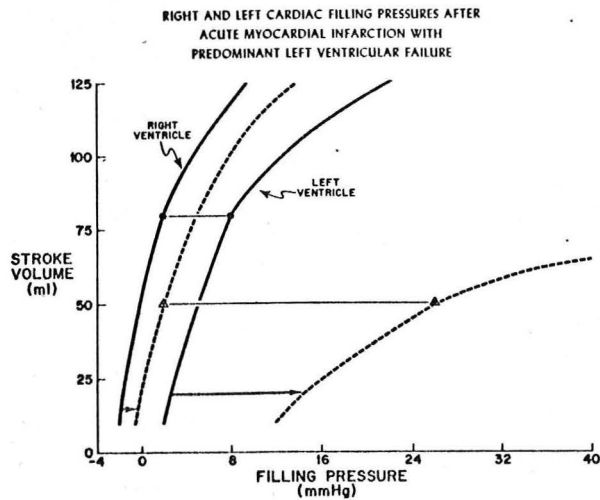
The relations between stroke volume and filling pressure for both the right and left ventricles are shown by the solid lines. Stroke volume of each ventricle is 80 ml and the filling pressure of the right ventricle is 2 mm Hg and that of the left ventricle is 8 mm Hg. After an acute myocardial infarction predominantly involving the left ventricle there is little change in the relation between stroke volume and filling pressure of the right ventricle and a marked change in the relation for the left ventricle (dashed lines). In addition, there is a fall in stroke volume from the control value of 80 ml to 50 ml. These events cause a marked increase in the filling pressure of the left ventricle to 26 mm Hg with no appreciable change in the central venous pressure. Thus in many patients after acute myocardial infarction left ventricular end-diastolic pressure may be significantly elevated while central venous pressure may remain within normal limits.

In a few patients after an acute myocardial infarction there is principal involvement of the right ventricle with predominant right ventricular failure. The effect of acute myocardial infarction with predominant right ventricular failure on the relation between stroke volume and filling pressure of each ventricle is shown in Figure 5.

Figure 5

RIGHT AND LEFT CARDIAC FILLING PRESSURES AFTER
ACUTE MYOCARDIAL INFARCTION WITH
PREDOMINANT RIGHT VENTRICULAR FAILURE
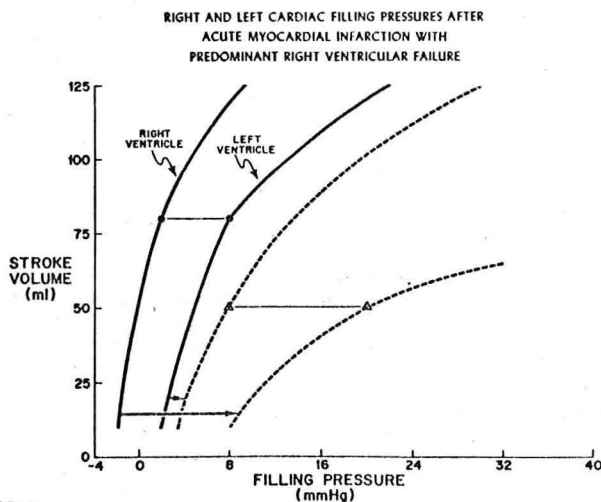
The normal relations are again shown by the solid lines. After an infarction principally involving the right ventricle there is little change in the stroke volume-filling pressure relation of the left ventricle with a marked change in that of the right (dashed lines). With a fall in stroke volume to 50 ml, there is no appreciable change in the filling pressure of the left ventricle; however, right ventricular filling pressure increases from 2 to 20 mm Hg. In such a situation it would be beneficial to give a volume infusion in spite of the elevated central venous pressure to increase the filling pressure of the left heart. This is similar to the condition that may be present in pericardial effusion with cardiac tamponade. A markedly elevated central venous pressure in such clinical situations may be beneficial in supplying the left ventricle with an adequate filling pressure and thereby increasing stroke volume.

      2. Arterial blood pressure and peripheral vascular resistance

21. Loc. cit.
24. Cohn, J. N.: Blood pressure measurement in shock. Mechanism of inaccuracy in auscultatory and palpatory methods. J.A.M.A. 199:972, 1967.

A fall in arterial blood pressure may not occur in the beginning stages of depressed left ventricular function. A marked increase in peripheral vascular resistance in the face of an inadequate cardiac output can maintain a blood pressure within normal limits. A patient with clinical evidence of marked vasoconstriction and with a normal or slightly lowered blood pressure frequently progresses to cardiogenic shock.

    B. Cardiac output

5. Loc. cit.
6. Loc. cit.
21. Loc. cit.
25. Fries, E. D., Schnaper, H. W., Johnson, R. L. and Schreiner, G. E.: Hemodynamic alterations in acute myocardial infarction. I. Cardiac output, mean arterial pressure, total peripheral resistance, "central" and total blood volumes, venous pressure, and average circulation time. J. Clin. Invest. 31:131, 1951.
26. Smith, W. W., Wikler, N. S. and Fox, A. C.: Hemodynamic studies of patients with myocardial infarction. Circulation 9:352, 1954.
27. Thomas, M., Malmcrona, R. and Shillingford, J.: Hemodynamic changes in patients with acute myocardial infarction. Circulation 31:811, 1965.

A rather wide range of values for cardiac output and cardiac index has been reported in studies performed after acute myocardial infarction. In general, patients with acute myocardial infarction have a diminished cardiac output. However, caution must be exercised in the interpretation of values obtained by the usual indicator-dilution technique since errors can be introduced by undetected recirculation and prolonged sampling time.

Ramo, Myers, Wallace, Starmer, Clark, and Whalen have reported hemodynamic findings including cardiac index in 123 patients during acute myocardial infarction and this is shown in Figure 6.

Figure 6

CARDIAC INDEX
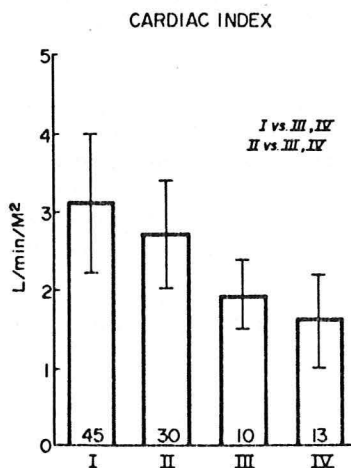
Patients were classified on clinical grounds according to the severity of their heart failure. Class I patients had no heart failure. Class II patients had mild to moderate heart failure (basilar râles and S_3 gallop rhythm). Class III patients had pulmonary edema, and class IV patients were in cardiogenic shock. The cardiac index tended to fall as the clinical severity of the heart failure increased. However, one should note the overlaps of values among the classes and the wide distribution of values within each clinical class.

C. Total body arteriovenous oxygen difference (A-V O_2 difference) and mixed venous oxygen saturation

21. Loc. cit.
28. MacKenzie, G. J., Flenley, D. C., Taylor, S. H., McDonald, A. H., Staunton, H. P. and Donald, K. W.: Circulatory and respiratory studies in myocardial infarction and cardiogenic shock. Lancet 2:825, 1964.
29. Valentine, P. A., Fluck, D. C., Mounsey, J. P. D., Reid, D., Shillingford, J. P. and Steiner, R. E.: Blood gas changes after acute myocardial infarction. Lancet 2:837, 1966.
30. Goldman, R. H., Braniff, B., Harrison, D. C. and Spivack, A. P.: The use of central venous oxygen saturation measurement in a coronary care unit. Ann. Int. Med. 68:1280, 1968.
31. Goldman, R. H., Klughaupt, M., Metcalf, T., Spivack, A. P. and Harrison, D. C.: Measurement of central venous oxygen saturation in patients with myocardial infarction. Circulation 38: 941, 1968.

Changes in arteriovenous oxygen difference are inversely related to changes in cardiac output during acute myocardial infarction. Goldman, Klughaupt, Metcalf, Spivack, and Harrison have suggested that the measurement of central venous oxygen saturation would be useful in assessing cardiac function during acute myocardial infarction since they found a good correlation between the oxygen saturation in the superior vena cava ($CVSO_2$) and the pulmonary artery (mixed venous oxygen saturation, MVO_2). As shown in Figure 7 their studies indicated that as cardiac function deteriorated, $CVSO_2$ falls.

Figure 7
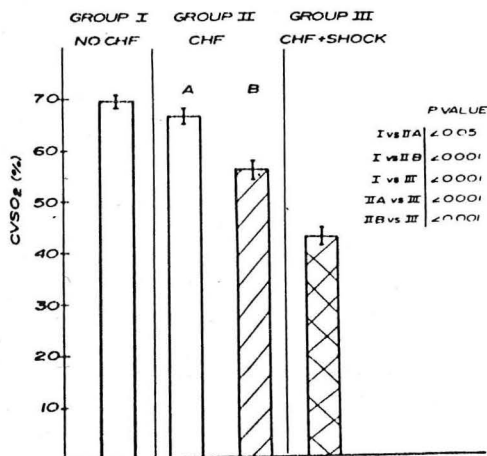
In patients with no evidence of heart failure (group I) $CVSO_2$ was 70%. In group II patients before heart failure (group II, class A) the $CVSO_2$ was near 70% and in patients in heart failure (group II, class B) $CVSO_2$ fell below 60%. In patients with cardiogenic shock and heart failure (group III) the $CVSO_2$ fell below 45%. Other investigators have found as good a correlation between $CVSO_2$ and mixed venous oxygen saturation in patients with cardiac failure or shock or both. However, a good correlation was shown between changes in $CVSO_2$ and $MVSO_2$. Thus serial measurements of changes in $CVSO_2$ may be useful in evaluating changes in cardiac output and cardiac function.

The present ease and safety associated with pulmonary artery catheterization with the Swan Ganz catheter make it possible to follow $MVSO_2$ with little difficulty in the coronary care unit. To my knowledge a study of mixed venous oxygen saturation in patients during acute myocardial infarction to elucidate whether it has prognostic significance has not been carried out. Such a study should be done.

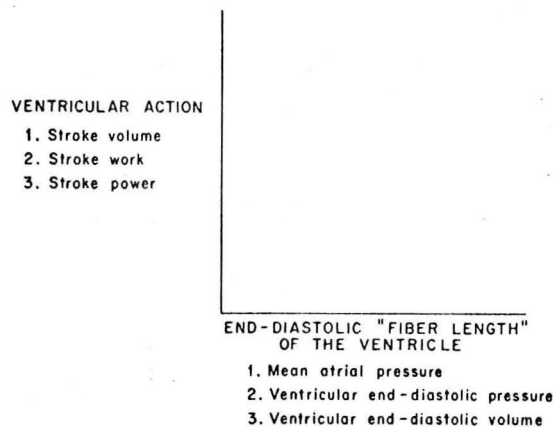D. Ventricular function curves

1. Physiological concepts

32. Frank, O.: Zur Dynamik des Herzmuskels. Ztschr. Biol. <u>32</u>: 370, 1895. (Transl: Am. Heart J. <u>58</u>:282, 467, 1959.)
33. Starling, E. H.: The Linacre Lecture on the Law of the Heart. London: Longmans, Green, 1918.
34. Sarnoff, S. J. and Berglund, E.: Ventricular function. I. Starling's law of the heart studied by means of simultaneous right and left ventricular function curves in the dog. Circulation 9:706, 1954.
35. Sarnoff, S. J. and Mitchell, J. H.: The regulation of the performance of the heart. Am. J. Med. <u>30</u>:747, 1961.
36. Mommaerts, W. F. H. M. and Langer, G. A.: Fundamental concepts of cardiac dynamics and energetics. Ann. Rev. Med. <u>14</u>: 261, 1963.
37. Mitchell, J. H. and Mullins, C. B.: Determinants of ventricular function. In: Pathophysiology of Congenital Heart Disease, ed. by F. H. Adams, H. J. C. Swan and V. E. Hall. Berkeley: University of California Press, 1970, pp. 163-180.

There is a long tradition of considering the left ventricle as a compression pump and of evaluating ventricular function in such terms. The foundation for the evaluation of ventricular function in terms of a compression pump is the Frank-Starling principle. In 1895, Frank found that the tension developed in an isovolumically contracting ventricle was proportional to the initial or end-diastolic tension. In Frank's experiments, the initial length and the initial tension (pressure) were proportional; thus the in-

creased force of contraction might be due either to increased dias-
tolic fiber length or to increased diastolic pressure. Starling
carried out similar studies in a dog heart-lung preparation. From
these experiments he postulated Starling's law of the heart which
stated in its simplest and most general form is as follows: "The
law of the heart is thus the same as the law of muscular tissue
generally, that the energy of contraction, however measured, is a
function of the length of the muscle fiber."

The concept involved in the ventricular function curve ap-
proach to evaluation of ventricular performance is shown in Figure
8. As has been pointed out by Mommaerts and Langer, this ap-
proach follows directly from extrapolation of the length-tension
relation of cardiac muscle. In this method, an estimate of the
"energy of ventricular contraction, however measured", probably
better termed the mechanical activity of the ventricle, is related
to an estimate of the end-diastolic "fiber length" of the ventri-
cle.

Figure 8

VENTRICULAR ACTION
1. Stroke volume
2. Stroke work
3. Stroke power

END-DIASTOLIC "FIBER LENGTH"
OF THE VENTRICLE
1. Mean atrial pressure
2. Ventricular end-diastolic pressure
3. Ventricular end-diastolic volume

Various measurements have been used to quantitate the mechani-
cal activity of the ventricle. Stroke volume was the earliest
used measurement. The amount of work performed by the ventricle
each beat, or the stroke work, has been the most often used esti-

mate of ventricular action. This value can be adequately calcu-
lated by using mean values of stroke volume and ejection pressure.
Stroke power is a more discriminating estimate of the mechanical
activity of the ventricle since it includes a rate component.

Various measurements have also been used to indicate the end-
diastolic "fiber length" of the ventricle. Because of the complex
arrangement of muscle fibers in the wall of the ventricle, it is
not possible to measure this quantity as it is in skeletal muscle
or in a cardiac papillary muscle preparation. Mean atrial pres-
sure has been used as an estimate of the fiber length of the ven-
tricle. More direct measurements, however, are ventricular end-
diastolic pressure or volume.

Changes in the contractile state of the left ventricle can be
defined by utilizing the ventricular function concept, as shown in
Figure 9.

Figure 9
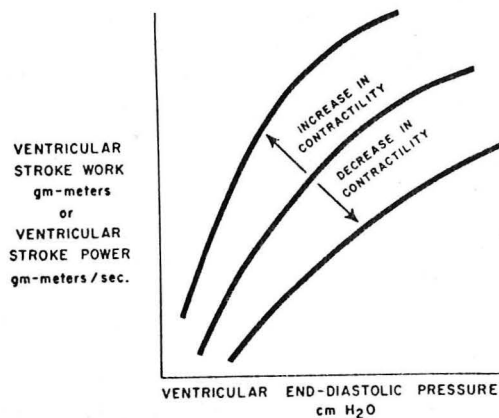
The middle curve represents the control condition. A decrease
in contractility is represented by a movement of the curve down-
ward and to the right; that is, the ventricle is able to perform
less stroke work and less stroke power from any given end-dias-
tolic pressure and presumably fiber length of the ventricle (or,
the performance of a given amount of stroke work or stroke power

requires a higher end-diastolic pressure and fiber length of the ventricle). An increase in the contractile state is represented by a movement of the curve upward and to the left; that is, the ventricle is able to perform more stroke work and more stroke power from any given end-diastolic pressure and fiber length of the ventricle (or, the performance of a given amount of stroke work or stroke power does not require as high an end-diastolic pressure).

2. Clinical use

6. Loc. cit.
7. Loc. cit.
8. Loc. cit.
38. Nixon, P. G. F., Ikram, H. and Morton, S.: Infusion of dextrose solutions in cardiogenic shock. Lancet 1:1077, 1966.
39. Nixon, P. G. F. and Taylor, D. J. E.: Left ventricular diastolic pressure in cardiogenic shock treated by dextrose infusion and adrenalin. Lancet 1:1230, 1968.
40. Cohn, J. N., Luria, M. H., Daddario, R. C. and Tristani, F. E.: Studies in clinical shock and hypotension: V. Hemodynamic effects of dextran. Circulation 35:316, 1967.
41. Russell, R. O., Jr., Rackley, C. E., Pombo, J., Hunt, D., Potanin, C. and Dodge, H. T.: Effects of increasing left ventricular filling pressure in patients with acute myocardial infarction. J. Clin. Invest. 49:1539, 1970.
42. Hamosh, P. and Cohn, J. N.: Left ventricular function in acute myocardial infarction. J. Clin. Invest. 50:523, 1971.
43. Ratshin, R. A., Rackley, C. E. and Russell, R. O., Jr.: Hemodynamic evaluation of left ventricular function in shock complicating myocardial infarction. Circulation 45:127, 1972.

In some cases of acute myocardial infarction with the syndrome of pallor, hypotension, disturbance of consciousness, intense peripheral vasoconstriction, and anemia, the patients recovered after infusion of 5% dextrose solutions. This led to a more careful evaluation of ventricular performance during acute myocardial infarction.

In order to evaluate ventricular performance in patients after acute myocardial infarction, ventricular function curves have been performed by serial infusions of low molecular weight dextran. A study performed by Russell, Rackley, Pombo, Hunt, Potanin, and Dodge is shown in Figure 10.

Figure 10
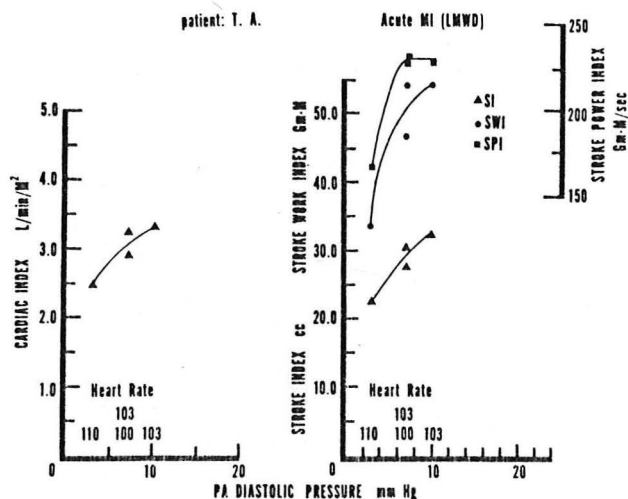
In this study cardiac index, stroke index, stroke work index, and stroke power index were related to left ventricular filling pressure before and after receiving 600 ml low molecular weight dextran in 200 ml increments. With a progressive increase of filling pressure from 3 to 10 mm Hg, there was a progressive increase in cardiac index, stroke index, stroke work index, and stroke power index. This curve demonstrates that the left ventricle responds in a fairly normal fashion to volume infusion and has not been severely damaged by the acute infarction. In patients with markedly depressed left ventricular function, there is little or no increase in cardiac index, stroke index, stroke work index, and stroke power index as ventricular filling is increased by dextran infusions.

Ratshin, Rackley, and Russell have shown that patients with a depressed ventricular function curve have a higher mortality than those with a more normal curve (Figure 11).

Figure 11
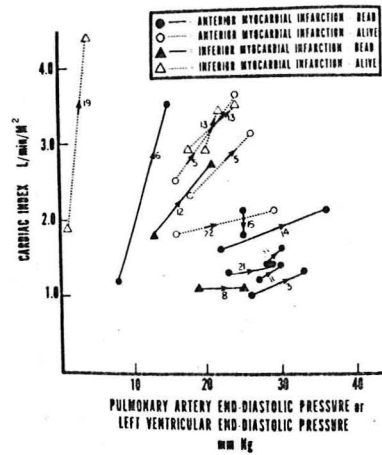
Survivors favorably respond to volume augmentation with a proportionally greater rise in cardiac index and stroke work index than in left ventricular filling pressure. Those not surviving exhibited a proportionally greater increase in left ventricular filling pressure than in cardiac index or stroke work index with dextran infusions.

In addition nonsurvivors tended to have a lower baseline cardiac index and stroke work index and a higher baseline left ventricular filling pressure. Ratshin et al. have also shown that predictions of survival can be made from baseline measurements of cardiac index and left ventricular filling pressure as shown in Figure 12.

Figure 12
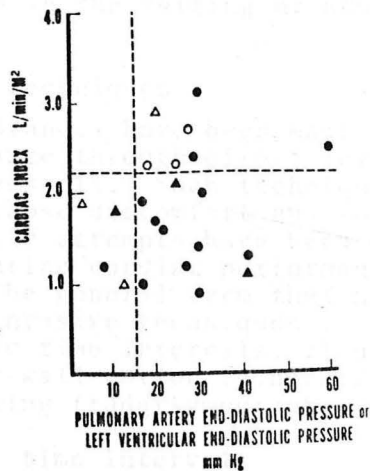
Patients all died who tended to have cardiac indices of less than 2.3 liters/min/m^2 in association with a left ventricular filling pressure of 15 mm Hg or greater. Also patients with an initial filling pressure greater than 28 mm Hg regardless of the cardiac index all expired.

The studies by Russell et al. have also demonstrated that in both patients with and without depressed ventricular function curves the maximal cardiac index and stroke index are obtained when the filling pressure is between 20 and 24 mm Hg. Elevation of left ventricular pressure beyond 25 mm Hg often does not produce further increase in cardiac and stroke index and on occasion may lower them.

It seems important, therefore, to regulate left ventricular filling pressure between 20 and 24 mm Hg in patients with depressed ventricular function after acute myocardial infarction. In patients whose initial filling pressures are above 25 mm Hg, reduction of filling pressure by positive pressure breathing, venous tourniquets, phlebotomy, or diuretics to the optimum range should be accomplished. In patients whose filling pressures are below this optimum range, elevation of filling pressure to between 20 and 24 mm Hg should be reached by dextran infusions. As has been mentioned earlier when making these important adjustments in left ventricu-

lar filling pressure it is extremely important to follow a sensitive measure of this value. Currently left ventricular filling pressure is most easily obtained by catheterization of the pulmonary artery. Central venous pressure does not accurately reflect changes in left ventricular filling pressure and cannot be used for this purpose in the setting of acute myocardial infarction.

III. Noninvasive Techniques

Important advances have been made in our ability to evaluate cardiac performance through direct invasion of the cardiac chambers and great vessels. Such techniques require highly trained physicians and cause discomfort and some degree of hazard for the patient. Recently attempts have been made to develop sensitive methods of measuring cardiac performance without invasion of the body surface. The general term that has been applied to these methods is "noninvasive techniques". Noninvasive techniques include 1) systolic time intervals, 2) ultrasonic measurement of left ventricular wall motion (echocardiography), and 3) heart motion video-tracking (radarkymography).

A. Systolic time intervals

44. Wiggers, C. J.: Studies on the consecutive phases of the cardiac cycle. II. The laws governing the relative durations of ventricular systole and diastole. Am. J. Physiol. 56:439, 1921.
45. Braunwald, E., Sarnoff, S. J. and Stainsby, W. N.: Determinants of duration and mean rate of ventricular ejection. Circulation Res. 6:319, 1958.
46. Wallace, A. G., Mitchell, J. H., Skinner, N. S., Jr. and Sarnoff, S. J.: Duration of the phases of left ventricular systole. Circulation Res. 12:611, 1963.

Investigative studies of the various periods and phases of the cardiac cycle have been carried out for many years. The cardiac cycle is divided into two periods according to events occurring in the ventricle. Relaxation of the ventricle is called diastole and contraction of the ventricle is called systole. Both of these periods can be subdivided into phases based upon physical or mechanical events in the cardiac cycle. Ventricular systole is divided into two principal phases: 1) the isovolumic contraction phase (isometric contraction) and 2) the ejection phase. The isovolumic contraction phase begins with the initiation of ventricular systole and lasts until the aortic valve opens and ejection begins. The ejection phase begins with the opening of the aortic valves and ends with the closing of the aortic valve.

The independent effects of various hemodynamic alterations on duration of the phases of left ventricular systole have been deter-

mined. Increasing stroke volume by increasing left ventricular filling caused a decrease in the duration of isovolumic contraction and an increase in the duration of ejection. An increase in the contractile state caused a decrease in both the duration of isovolumic contraction and the duration of ejection.

47. Katz, L. N. and Feil, H. S.: Clinical observations on the dynamics of ventricular systole. I. Auricular fibrillation. Arch. Int. Med. 32:672, 1923.

48. Weissler, A. M., Peeler, R. G. and Roehll, W. H., Jr.: Relationship between left ventricular ejection time, stroke volume, and heart rate in normal individuals and patients with cardiovascular disease. Am. Heart J. 62:367, 1961.

49. Weissler, A. M., Harris, W. S. and Schoenfeld, C. D.: Bedside technics for the evaluation of ventricular function in man. Am. J. Cardiol. 23:577, 1969.

50. Weissler, A. M. and Garrard, C. L., Jr.: Systolic time intervals in cardiac disease (1). Mod. Conc. Cardiovas. Dis. 40: 1, 1971.

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Systolic time intervals can be obtained from noninvasive or indirect techniques. They are measured from the simultaneously recorded electrocardiogram, phonocardiogram, and carotid artery pulsation. This is shown in Figure 13.

Figure 13
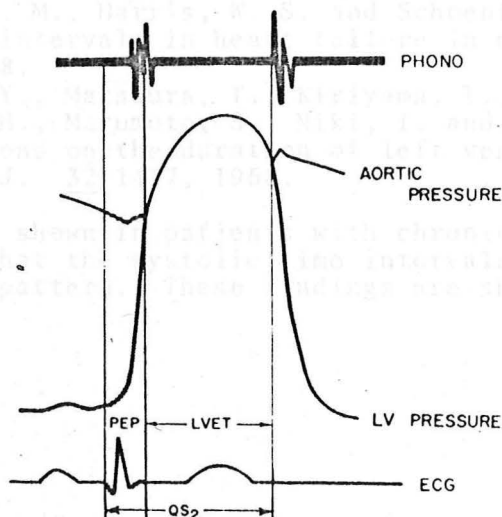
The three intervals which have been most commonly used are: 1) total electrical-mechanical systole (Q-S$_2$), 2) the left ventricular ejection time (LVET), and 3) the pre-ejection phase (PEP). The Q-S$_2$ interval is the time from the onset of ventricular electrical activation to the first high frequency vibrations of the second heart sound. The LVET interval is the time from the beginning of the upstroke of the carotid artery pulsation to the incisural notch which signals the closing of the aortic valve. The PEP interval is the time from the onset of ventricular electrical activation to the beginning of left ventricular ejection. It is calculated by subtracting the LVET from the Q-S$_2$ interval which eliminates the delay in transmission of the arterial pulse from the proximal aorta to the carotid artery.

52. Metzger, C. C., Chough, C. B. and Kroetz, F. W.: True isovolumic contraction time: its correlation with two external indexes of ventricular performance. Am. J. Cardiol. 25:434, 1970.
53. Bush, C. A., Lewis, R. P. and Leighton, R. F.: Verification of systolic time intervals and the true isovolumic contraction time from the apexcardiogram by micromanometric catheterization of the left ventricle and aorta. Circulation 42:III-121, 1970.

The determination of STI from noninvasive recordings of the electrocardiogram, phonocardiogram, and carotid artery pulsation has been shown to accurately reflect direct measurements of STI made from high fidelity pressure recording made during cardiac catheterization.

54. Weissler, A. M., Harris, W. S. and Schoenfeld, C. D.: Systolic time intervals in heart failure in man. Circulation 37:149, 1968.
55. Hishimoto, Y., Matsuura, T., Kiriyama, T., Ochiai, M., Kunishige, H., Marumoto, S., Niki, I. and Endo, N.: Clinical considerations on the duration of left ventricular systoles. Jap. Circ. J. 32:1417, 1968.

It has been shown in patients with chronic arteriosclerotic heart disease that the systolic time intervals are altered in a characteristic pattern. These findings are shown in Figure 14.

Figure 14

Chronic Arteriosclerotic Heart Disease

	$Q-S_2$	PEP	LVET
Weissler et al.	0	↑	↓
Hishimoto et al.	0	↑	↓

Acute Myocardial Infarction

	$Q-S_2$	PEP	LVET
Weissler et al.	↓	↑	↓
Toutouzos et al.	↓	Not measured	Not measured
Halpern et al.	↓	0	↓
Diamant et al.	↓	↑	↓
Heikkilä et al.	↓	0	↓

This consists of an abbreviation of the LVET and a prolongation of the PEP at a time when the $Q-S_2$ remains within normal limits.

56. Schoenfeld, C. D., Robinson, J. L. and Weissler, A. M.: Left ventricular systolic time intervals in myocardial infarction. Clin. Res. 15:411, 1967.
57. Toutouzos, P., Bupta, D. and Samson, R.: Q-second sound interval in acute myocardial infarction. Brit. Heart J. 31: 462, 1969.
58. Halpern, B. L., Hodges, M., Dagenais, G. R. and Friesinger, G. C.: Left ventricular pre-ejection period and ejection time in patients with acute myocardial infarction. Circulation 40:III-100, 1969.
59. Diamant, B. and Killip, T.: Indirect assessment of left ventricular performance in acute myocardial infarction. Circulation 42:579, 1970.
60. Heikkilä, J., Luomanmäki, K. and Pyörälä, K.: Serial observations on left ventricular dysfunction in acute myocardial infarction. II. Systolic time intervals in power failure. Circulation 44:343, 1971.

Attempts have been made to use systolic time intervals to evaluate left ventricular function after acute myocardial infarction. Not everyone agrees on the value of the method in the clinical setting of a myocardial infarction. Investigators who have found the determinations useful have published their data but those who have found little use of the method talk about their findings on the boardwalk but never publish their results so that they can be evaluated. However, even those investigators who have found the method to be useful do not agree on what changes can be used to evaluate left ventricular function. The findings from five reported studies are also shown in Figure 14.

In contrast to chronic arteriosclerotic heart disease, the $Q-S_2$ interval is shortened after acute myocardial infarction. However, the LVET is decreased in both chronic arteriosclerotic heart disease and after acute myocardial infarction. The effect of a myocardial infarction on the PEP interval is controversial. Weissler and his group and Diamant and Killip have reported that this interval is lengthened and Halpern and Heikkilä have reported that it is not changed.

B. Ultrasonic measurement of left ventricular wall motion (echocardiography)

61. Popp, R. L., Wolfe, S. B., Hirata, T. and Feigenbaum, H.: Estimation of right and left ventricular size by ultrasound. Am. J. Cardiol. 24:523, 1969.
62. Pombo, J. F., Troy, B. L. and Russell, R. O., Jr.: Left ventricular volumes and ejection fractions by echocardiography. Circulation 43:480, 1971.
63. Inoue, K., Smulyam, H., Mookherjee, S. and Eich, R. H.: Ultrasonic measurement of left ventricular wall motion in acute myocardial infarction. Circulation 43:778, 1971.

Echocardiography has been used to measure left ventricular posterior wall motion during acute myocardial infarction. A significant reduction in the amplitude of posterior wall motion has been demonstrated in patients with acute myocardial infarction as compared to normal subjects. Further a decrease in the velocity of wall motion during systole was described. These findings suggest a decrease in stroke volume and a decrease in contractile state. Measurement of posterior wall motion appears to be of value as a noninvasive method of evaluating ventricular performance after acute myocardial infarction.

C. Heart motion video-tracking (radarkymography)

64. Cohen, L. S., Simon, A. L., Whitehouse, W. C., Schuette, W. H. and Braunwald, E.: Heart motion video tracking (radarkymography) in diagnosis of congenital and acquired heart disease. Am. J. Cardiol. 22:678, 1968.
65. Kazamias, T. M., Gault, J. H. and Ross, J., Jr.: The continuous measurement of left ventricular size. Circulation 40: III-120, 1969.
66. Kazamias, T. M., Gander, M. P. and Ross, J., Jr.: Serial changes in left heart size in acute myocardial infarction (AMI). Circulation 42 :III-171, 1970.

A method has recently been developed which allows assessment of cardiac shadow size by the use of a radiographic unit in conjunction with a device that translates wall motion into an analog

signal. Left ventricular size after acute myocardial infarction
has been estimated by this method. It has been found that the
left ventricle increases in size immediately after the acute in-
farction and tends to decrease in size during recovery over the
course of several days or weeks.

IV. Conclusions

Little headway has been made in salvaging patients with acute
myocardial infarction who develop pump failure. Careful hemody-
namic monitoring and evaluation of cardiac performance in patients
after acute myocardial infarction are now possible in coronary
care units. Also both medical and surgical therapy for pump fail-
ure are being administered on more solid physiological grounds.
It is hoped that both earlier recognition and earlier treatment of
impending pump failure will have the same impact on mortality in
mechanical failure as has been accomplished in electrical failure
after acute myocardial infarction.