Sequential Circulatory Changes following Acute Myocardial Infarction in Man

Determination of Optimal Heart Rate at Each Stage of the Infarction by Atrial Pacing

By W. Glenn Friesen, M.D., F.R.C.P.

SUMMARY

After informed consent, 12 men with documented acute myocardial infarction had hemodynamic studies performed within 18 hours after onset of symptoms. These were repeated on the second day, at 3–5 weeks, and at 3–5 months in the 10 who survived. One half of the patients were "uncomplicated" and significant differences with the "complicated" patients were seen in pulmonary artery pressure, mean arterial pressure on the first day, acute phase stroke volume and cardiac index, and the total blood volume within 48 hours. The optimal heart rate at each stage was determined by atrial pacing and was found to be 110 ± 10 beats/min. Abnormalities in pH, blood gases, and circulation time were related to the cardiac index. Control groups with and without coronary artery disease were studied prior to angiography to obtain information regarding preinfarction circulatory status, and significant impairment of the ejection fraction was noted even in angina patients without prior infarction. The sequential circulatory changes and possible compensatory mechanisms available were discussed.

Additional Indexing Words:
Isotope Total blood volume Circulation time Blood gases Ejection fraction
End-diastolic volume 131Iodine Gamma spectrometer

ACUTE myocardial infarction remains a frequent cause of death in man. Although the majority of such deaths occur prior to hospitalization, the mortality rate in hospitalized patients has declined by approximately one third, mainly due to control of arrhythmias. Approximately 90% of current in-hospital deaths are associated with myocardial (power) failure. It is hoped that by early recognition of power failure and by institution of rational and effective therapy, including emergency cardiac surgery, the mortality of these hospitalized patients will be further reduced. Characterization of the patient's circulatory status by catheterization technics and measurements serves to identify these patients and provides objective evidence upon which to base therapy in a way not previously possible by general clinical assessment.

Rackley and Russell1 have reviewed the literature pertaining to hemodynamic studies following acute myocardial infarction in man since Pritchard and Hellerstein performed the earliest such cardiac catheterization study in 1950.2 Earlier reports tended to describe only the venous and arterial pressures and cardiac outputs in the severely ill, and descriptions of sequential changes following myocardial infarction have been relatively few and often incomplete.

The purpose of this report is to present the sequential circulatory changes from 12 patients with acute myocardial infarction, half of whom had heart failure, arrhythmias, or both of these complications. In addition, the heart rate associated with the best cardiac output was determined at each stage of the study by means of atrial pacing. Some attempt was made to correlate the clinical course of each patient with the hemodynamic data and to compare these data with observations from normal...
persons and from patients with chronic atherosclerotic coronary artery disease.

Materials and Methods

Subjects

The clinical characteristics and course of the six uncomplicated and the six complicated patients with acute myocardial infarction are illustrated in table 1. After informed consent 12 men were selected for study in the Coronary Care Unit within 18 hours of the onset of symptoms. Standard criteria were used for the diagnosis of acute myocardial infarction and included a positive history, typical electrocardiographic findings, and enzyme studies. There was no evidence for pulmonary or other systemic disease in these patients, and none gave a history of hypertension. All were in sinus rhythm, and none had heart murmurs. The mean age of the uncomplicated group was 59 years and that of the complicated patients 61 years, not a significant difference. The mean peak SGOT for the uncomplicated patients was $159 \pm 87$ and that of the complicated patients was $256 \pm 175$, tending to be higher in the complicated group. Chest roentgenograms were abnormal in four of the six in the complicated group, but were normal in all of the six uncomplicated patients studied.

Bedside Catheterization

All studies were performed at the bedside with the patient supine and resting quietly, clinically stable, and free of pain. Oxygen administration, analgesics, and sedatives were discontinued at least 4 hours prior to hemodynamic studies in all but one patient who was in shock and was receiving oxygen by nasal catheter. Using local anesthesia, both right atrial and pulmonary artery electrode catheters were introduced by way of a cutdown over a median arm vein. The catheters were positioned using electrocardiographic monitoring and the position of the catheters confirmed initially by characteristic pressure responses and later by radiography. A radial artery cannula was inserted in most patients, but in some a short cannula was inserted percutaneously into the brachial artery. The Swan-Ganz balloon flow-guided catheter has been used for recent studies of pulmonary artery and pulmonary capillary wedge pressures.

Figure 1

Measuring the cardiac output and circulation time by the isotope technic using a gamma spectrometer with one probe over the precordium and the other over the femoral artery. A two-pen chart recorder is essential.

Circulation, Volume XLVII, March 1973
Hemodynamic Studies

Pressures were measured utilizing a strain gauge transducer (Statham), mean pressures being obtained electronically. A direct recording system was used, incorporating a simultaneous electrocardiographic write-out. Unipolar atrial pacing was carried out by means of right atrial electrode catheter which was positioned near the junction of the superior vena cava and the right atrium. The paced heart rate was randomly increased or decreased by steps of 15 beats/min, the maximum paced rate being 130/min. At least 10 min were allowed for stabilization at any given rate before hemodynamic measurements were made. Cardiac output and blood volume (TBV) were measured by the indicator-dilution method using radioactive iodine-labeled 131I human serum albumin (RISA) injected through the right atrial catheter with precordial counting utilizing a gamma spectrometer coupled to a chart recorder. Recently we have employed a dual rate computer with high-speed digital printout with one probe positioned over the precordium and the other over the femoral artery to record the circulation time from right atrium to femoral artery (figs. 1 and 2). The principles of the radioisotope-dilution method of determining cardiac output have been described by others and this method was used by Murphy et al. for determining cardiac output serially in patients with acute myocardial infarction. Kloster et al. found that this method correlated well with the Fick method (R = 0.80, P < 0.001; slope = 1.02, intercept = −0.01) and it has the advantage of allowing for repeated measurements without excessive blood sampling.

Kloster's method was used for this study. The mean difference and 1 se of 16 duplicate cardiac output measurements performed at the same heart rate during the 1–2 hour study was 0.25 ± 0.21 liters/min/m². Systemic vascular resistance (mm Hg/liters) was calculated from the equation: (mean arterial pressure − right atrial pressure) / cardiac output.

Arterial and mixed venous blood samples were drawn simultaneously during each study at a given heart rate and from these the pO₂, pCO₂, and pH, and oxygen saturations were obtained. On the first day, hemodynamic studies were made 3 hours after catheters were

Figure 2

With an injection of 10–15 μCi of 131I albumin (RISA) into the central venous catheter and simultaneous recording from the precordial and femoral artery probes at 0.5-sec time response with digital printout the cardiac output and right atrium-to-femoral artery circulation (FCT) time can be calculated. The mean pulmonary circulation time (PCT) is the time between the peak concentrations in right and left ventricle.
**Table 1**

*Clinical Characteristics of Patients Undergoing Hemodynamic Studies*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex/Age (y)</th>
<th>History</th>
<th>BP (mm Hg)</th>
<th>ECG</th>
<th>Chest X-ray</th>
<th>LDH</th>
<th>SGOT</th>
<th>Sequence</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.K.</td>
<td>M/69</td>
<td>0</td>
<td>1 yr</td>
<td>II</td>
<td>Nausea; epigastric distress</td>
<td>170/110</td>
<td>ILMI</td>
<td>Normal</td>
</tr>
<tr>
<td>E.B.</td>
<td>M/51</td>
<td>0</td>
<td>0</td>
<td>I</td>
<td>Chest pain</td>
<td>150/80</td>
<td>AMI</td>
<td>Normal</td>
</tr>
<tr>
<td>A.K.</td>
<td>M/50</td>
<td>0</td>
<td>2 mo</td>
<td>I</td>
<td>Epigastic and arm distress</td>
<td>120/70</td>
<td>IMI</td>
<td>Normal</td>
</tr>
<tr>
<td>M.F.</td>
<td>M/60</td>
<td>0</td>
<td>0</td>
<td>I</td>
<td>Chest and arm pain</td>
<td>150/90</td>
<td>IMI</td>
<td>Normal</td>
</tr>
<tr>
<td>S.O.</td>
<td>M/65</td>
<td>+</td>
<td>14 yr</td>
<td>II</td>
<td>Chest pain</td>
<td>120/80</td>
<td>AMI</td>
<td>Normal</td>
</tr>
<tr>
<td>F.V.</td>
<td>M/58</td>
<td>+</td>
<td>8 yr</td>
<td>I</td>
<td>Chest and arm pain</td>
<td>170/100</td>
<td>ILMI</td>
<td>Normal</td>
</tr>
<tr>
<td>C.B.</td>
<td>M/72</td>
<td>11 yr</td>
<td>12 yr</td>
<td>III</td>
<td>Weakness; chest pain</td>
<td>120/70</td>
<td>Old IMI</td>
<td>Moderate cardiomegaly 730</td>
</tr>
<tr>
<td>R.M.</td>
<td>M/16</td>
<td>0</td>
<td>6 mo</td>
<td>III</td>
<td>Severe chest pain</td>
<td>90/50</td>
<td>LBBB</td>
<td>Marked cardiomegaly; CHF 75</td>
</tr>
<tr>
<td>L.V.</td>
<td>M/46</td>
<td>3 times in last yr</td>
<td>0</td>
<td>I</td>
<td>Pain in chest to jaws and epigastrium</td>
<td>120/80</td>
<td>ALMI</td>
<td>Heart enlarged from day 2 on 2240</td>
</tr>
<tr>
<td>A.M.</td>
<td>M/63</td>
<td>0</td>
<td>0</td>
<td>I</td>
<td>Severe chest and arm pain; nausea</td>
<td>120/80</td>
<td>ILMI</td>
<td>Normal</td>
</tr>
<tr>
<td>P.H.</td>
<td>M/50</td>
<td>2 mo</td>
<td>0</td>
<td>I</td>
<td>Chest and arm pain</td>
<td>140/100</td>
<td>AMI</td>
<td>CHF 411</td>
</tr>
<tr>
<td>R.G.</td>
<td>M/49</td>
<td>0</td>
<td>0</td>
<td>I</td>
<td>Chest pain; prostration</td>
<td>90/60</td>
<td>AMI</td>
<td>— 580</td>
</tr>
</tbody>
</table>

*Abbreviations: M1 = myocardial infarction (A = anterior; IL = inferolateral; AS = anteroseptal); FC = functional classification; CC = chief complaint; BP = blood pressure; ECG = electrocardiogram; 0 = none; + = present; PNCs = premature nodal contractions; PVCs = premature ventricular contractions; LBBB = left bundle-branch block.*
inserted so as to allow the patient to return to a resting state. The electrocardiogram, intravascular pressures, and flow studies were recorded and blood samples obtained at rest and at each heart rate at which the heart was paced. The catheters were left in place following the initial study and were kept open overnight by infusion of a heparinized saline solution. Approximately 500 ml of 0.95% saline was infused in each patient prior to the studies on the second day, after which the catheters were removed. Flow studies were repeated 3–5 weeks later in the patients who recovered and they returned to hospital for more complete studies after 3–5 months.

**Control Group**

The preinfarction hemodynamic status and ventricular function of the patients considered above was not known and therefore a control group was studied. Forty-two patients with anginal chest pain undergoing routine investigation were studied in the catheterization laboratory prior to coronary arteriography. After resting hemodynamic studies were performed these patients underwent cine left ventriculography and coronary arteriography utilizing the Judkins’ technic. Retrospective analysis of the angiographic data permitted patients to be placed in one of three groups. Six patients had both normal coronary arteries and myocardial function; 17 had greater than 50% stenosis of at least one major artery but reasonably normal myocardial contractility, considered coronary artery disease (CAD); and 19 had diseased coronary arteries with an abnormal ventriculogram (hypokinesia or akinesia—CADAVG). None of these 42 patients had valvular dysfunction or evidence for any other cardiac or systemic disorder. None had suffered a myocardial infarction in the preceding 3 months. All patients had both right and left heart catheterization studies performed together with cardiac outputs determined by the radioisotope technic and total blood volume measurements. In addition, left ventricular volume studies including determination of the ejection fraction (stroke volume divided by end-diastolic volume) were performed on 26 of these patients by the isotope-dilution technic using RISA injected into the left ventricle as described by Folse and Braunwald. The mean age of the normal patients was 47 ± 5 years, of the CAD group 54 ± 5 years, and the CADAVG group 52 ± 6 years. Atrial pacing studies were not performed in any of these patients as others have shown both in normal subjects and in patients with chronic stable heart disease with normal sinus rhythm that there is little change in resting cardiac output with induced tachycardia.

**Statistical Analysis**

All groups of data were analyzed using a one-way analysis of variance. The variances were tested for homogeneity. The difference between the means was considered significant if a probability of 0.05 or less was obtained for the F test on the mean squares. A multiple comparison of means was then made using either Schefﬁes’ or Newman-Keuls’ method to detect where a significant difference occurred ($P < 0.05$). Program ANOVI5, Division of Educational Research Services, University of Alberta, was used for the computations.

**Results**

### Pressures

Comparative pressure data at the designated time intervals following acute myocardial infarction are shown in figure 3. Patients whose illness was complicated tended to have higher right atrial pressures, particularly on the second day, but there is considerable overlap. On the first day, the pulmonary artery systolic pressure (mm Hg) in patients without complications was 31 ± 5, and 42 ± 7 ($P < 0.04$) in those with complications. The pulmonary artery diastolic pressures in the two groups were 15 ± 3 and 21 ± 3 mm Hg, respectively ($P < 0.04$). During convalescence (3–5 months) the pulmonary artery systolic pressure in patients without complications was 26 ± 9 and in those with

---

**Figure 3**

Sequential changes and comparison of the resting right atrial and arterial pressures and systemic vascular resistance in the uncomplicated and complicated patients with acute myocardial infarction. Mean values ± 1 so are shown.
complications, $44 \pm 10 \text{ mm Hg (} P < 0.04\text{)$. Comparable pulmonary artery diastolic pressures were $12 \pm 4$ and $25 \pm 7 \text{ mm Hg (} P < 0.04\text{)$. The mean arterial pressure on the first day was $105 \pm 18$ in patients without complications and $71 \pm 5 \text{ mm Hg in those with complications (} P < 0.01\text{). There was no significant difference in the subsequent time periods (fig. 3). Systemic vascular resistances were similar in the two subgroups, tending to decline more by the second day in the group without complications.

Flow Data

Cardiac output, cardiac index, and stroke volume for each subgroup are shown in figure 4 along with the influence of atrial pacing. The optimal heart rate is defined as the rate at which the higher cardiac output was recorded. In the patients without complications, no significant differences in the output were found at the optimal paced heart rate but the resting cardiac output proved to be greater by 48 hours than the cardiac output during convalescence ($P < 0.001$). In the acute phase the stroke volume was higher in the patients without complications ($P < 0.05$), and in those with complications it was found to be increased by the second day and also at 3-5 weeks, but fell again to borderline-low values in convalescence (fig. 4). Atrial pacing augmented the cardiac output in the patients without complications by 10% on the first day as the heart rate was increased from 84 to 102 beats/min (mean values). On the second day the increase was 20% as the heart rate was increased from 90 to 113, and at 3-5 weeks by 43% as the rate was increased from 78 to 109 beats/min. At 3-5 months the increase was 36% as the rate was increased from 79 to 109 (fig. 4). Atrial pacing in the patient with complications resulted in an increase in cardiac output of 10% on the first day as the heart rate was increased from 84 to 105, of 35% on the second day as the rate was increased from 93 to 108, of 27% at 3-5 weeks as the rate was increased from 81 to 106, and of 34% at 3-5 months as the rate increased from 77 to 112 beats/min (fig. 4).

**Figure 4**

The sequential flow studies at resting heart rate and at the optimally paced heart rate are illustrated. Mean values $\pm SD$ are given. Note the difference in scale used for stroke volume in the two groups.

_Circulation, Volume XLVII, March 1973_
Total Blood Volume
There was no difference between the subgroups of patients with and without complications on the first day, but by the second day a significant increase was found in patients with complications (P < 0.005) (fig. 5).

pH and Blood Gases
Table 2 relates the arterial (A) and mixed venous (MV) blood gases and pH to the cardiac index on the first day and the convalescent period (3–5 months) in both subgroups. Patients R.M., A.M., and R.G., who had a cardiac index of 1.51 liters/min/m² or less had wide A-MV pH and pCO₂ differences and the lower MV pO₂ (24–25 mm Hg). As the cardiac index rose above 2.2 liters/min/m² in patients R.M. and A.M., these differences became normal. The ApO₂ in the patients without complications tended to be higher than in those with complications in the acute phase, but there was some overlap. Patient R.G., who was in shock, had a metabolic acidosis not corrected by respiratory alkalosis.

Circulation Time
An inverse relation was found between the cardiac index and the right atrium–femoral artery circulation time as determined by the isotope method (fig. 6), r = −0.731. These are preliminary data and are based on additional patients studied in the Coronary Care Unit and in the catheterization laboratory.

Patient in Shock (R.G.)
This patient’s hemodynamic data were not included in the above analyses since he appears to be in an entirely different category. He was in sinus rhythm with a heart rate of 120 beats/min. His right atrial pressure was 10 mm Hg, pulmonary artery pressure 40/14, arterial pressure 70/55 (mean 60), and his systemic vascular resistance was 18 units. His cardiac index was 1.41 liters/min/m², stroke volume was 25 ml/min, and TBV was 83 ml/kg. His blood gases and pH measurements are listed in table 2. Increasing his heart rate to 130 beats/min with atrial pacing resulted in the cardiac index rising to 1.99 liters/min/m² (an increase of 41%). He died 18 hours after the onset of symptoms and an autopsy was found to have occlusion of the proximal main left coronary artery by an atherosclerotic plaque with an additional recent small thrombus resulting in a massive left ventricular myocardial infarction. The other coronary branches and the distal vessels were normal.

Control Groups
Comparative data are presented in figure 7 of the cardiac index, end-diastolic volume index, and ejection fraction (stroke volume/end-diastolic volume) in the normal group, the CAD group, and in the CADAVG group. The TBV (ml/kg) was normal in all three, being 60 ± 9 in the normal individuals, 65 ± 6 in those with coronary artery disease alone (CAD group), and 64 ± 9 in those with abnormal ventriculograms in addition (CADAVG group). The resting left ventricular
Blood Gases and pH Related to Cardiac Index

<table>
<thead>
<tr>
<th></th>
<th>Acute phase (Day 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt</td>
<td>ApH</td>
</tr>
<tr>
<td>W.K.</td>
<td>7.47</td>
</tr>
<tr>
<td>E.B.</td>
<td>7.44</td>
</tr>
<tr>
<td>A.K.</td>
<td>7.43</td>
</tr>
<tr>
<td>M.F.</td>
<td>7.50</td>
</tr>
<tr>
<td>S.O.</td>
<td>7.42</td>
</tr>
<tr>
<td>F.V.</td>
<td>7.49</td>
</tr>
<tr>
<td>Mean</td>
<td>7.46</td>
</tr>
<tr>
<td>sd</td>
<td>±0.03</td>
</tr>
</tbody>
</table>

**Uncomplicated**

| C.B.             | 7.46    | 7.44    | 0.02| 62          | 33           | 29          | 39          | 39           | 0          | 2.1             |
| R.M.*            | 7.54    | 7.37    | 0.17| 57          | 24           | 33          | 25          | 47           | 22         | 1.3             |
| L.V.             | 7.42    | 7.40    | 0.02| 58          | 32           | 26          | 37          | 38           | 1          | 3.7             |
| A.M.             | 7.42    | 7.37    | 0.05| 55          | 25           | 30          | 35          | 44           | 9          | 1.5             |
| P.H.             | 7.41    | 7.40    | 0.05| 34          | 24           | 10          | 40          | 43           | 3          | 3.0             |
| Mean             | 7.45    | 7.40    | 0.05| 53          | 28           | 27          | 35          | 42           | 7          | 2.1             |
| sd               | ±0.05   | ±0.03   | ±0.07| ±11         | ±5           | ±9          | ±6          | ±9           | ±1.1       |

**Complicated**

| R.G.             | 7.27    | 7.15    | 0.12| 75          | 25           | 50          | 28          | 61           | 33         | 1.4             |

Abbreviations: A = arterial; MV = mixed venous; d = difference; CI = cardiac index; pO2 and pCO2 = blood gases; sd = standard deviation.

*Values listed for convalescence in this patient are second-day values.

end-diastolic pressure (LVEDP) was not significantly different among these three groups in spite of the larger end-diastolic volume in the CAD and CADAVG. The LVEDP (mm Hg) was 8 ± 5 in the normal individuals, 8 ± 4 in the CAD group, and 11 ± 6 in the CADAVG group. Although a trend was found toward a lower cardiac index in patients whose coronary disease was more severe, the difference was not statistically significant. The end-diastolic volume was found to be significantly greater in the CADAVG group than in the group of normal patients ($P < 0.05$). The ejection fraction was significantly lower in both the CAD and the CADAVG groups than in the normal individuals ($P < 0.05$). The cardiac index in patients without complications in the series with acute myocardial infarction was higher in the first 48 hours than the indexes of the CADAVG group ($P < 0.05$).

**Discussion**

In this study, patients who had valvular malfunction, anemia, overt pulmonary disease, or systemic disease which might affect the metabolic rate of the tissues were excluded so that a more precise determination could be made of the circulatory changes resulting from the acute myocardial infarction alone. Patients with angina or those who have suffered previous myocardial infarctions already have impaired left ventricular function (fig. 7), and the effects of the new infarct are additive. If the areas of infarction involve 40% or more of the...
left ventricular mass, cardiogenic shock or “power failure” of the heart occurs. The amount of the left ventricular myocardial mass rendered akinetic and the intrinsic compensatory (autoregulatory) mechanisms operative at the time of study will determine the circulatory changes noted following acute myocardial infarction.

A marked increase in sympathetic nervous system activity usually occurs during the first week following myocardial infarction as evidenced by the markedly elevated levels of urinary catecholamines which have previously been demonstrated during this period. The resultant increase in contractile force and velocity of the remaining viable myocardium may more than adequately compensate for the akinetic cause by the infarction, particularly when the infarcted area is small. This may partially account for the normal-to-high flow studies found in the patients without complications during the acute phase. Increased venomotor tone would increase the venous return to the heart if the peripheral arterial resistance was not markedly elevated, and in this regard it is of interest to note that the peripheral vascular resistance measurements in the present study tended to fall by the second day. Further research is necessary to explain the “resetting” of the baroreceptors following acute myocardial infarction which occurs in spite of the presence of elevated catecholamines. Reduction of the peripheral vascular resistance may be an important compensatory mechanism permitting maintenance of the cardiac output while the work of the heart is reduced.

Renal retention of sodium and water leading to increased total blood volume with resultant increase in venous return, end-diastolic volume, and increased force of contraction (through the Frank-Starling law) is particularly evident in the patients with complications (fig. 5). In fact, this may be one of the important mechanisms operating in these patients which gives rise to the observed improvement in stroke volume and cardiac output by 48 hours. Earlier volume expansion in the patient with power failure by intravenous infusion and titrating the volume against the pulmonary pressure would seem to be a rational form of therapy in this group.

The optimal heart rate with respect to cardiac output at each stage of the infarction was found by atrial pacing to be approximately 110 ± 10 beats/min. Although atrial pacing at this rate may be useful to treat arrhythmias or the patients whose cardiac output is low the induced tachycardia is not recommended in the patient without complications because the increased myocardial oxygen consumption may enlarge the infarct area. An optimal heart rate appears to be particularly important when the increased sympathetic nervous

---

Circulation, Volume XLVII, March 1973
system activity and expanded total blood volume fail to compensate for impaired tissue perfusion.

The bedside catheterization technics and hemodynamic studies described in this report can be used to evaluate therapy objectively in patients with acute myocardial infarction and can identify the patient before "pump failure" becomes clinically manifest. In the absence of pulmonary disease, a pulmonary artery systolic pressure (mm Hg) of over 40 and a pulmonary artery diastolic pressure (or pulmonary wedge pressure) of over 20 would indicate severe impairment of left ventricular compliance. In the acute phase, a cardiac index of less than 2.5 liters/min/m² with a stroke volume of less than 50 ml/beat, arterial hypotension, and a right atrial-femoral artery circulation time beyond 18 sec suggest pump failure. With further tissue perfusion failure, the A-MV pH and pCO₂ differences widen (see Results) and the MV pO₂ falls below 28 mm Hg. The degree of arterial hypoxemia is roughly related to left ventricular failure but is not as reliable as the above indices since there is a rather wide variation in the normal ApO₂ in patients in this age group and particularly those on bed rest. The development of metabolic acidosis in the shock syndrome can best be studied using the MV blood as the associated respiratory alkalosis may normalize the ApH.

Keon has reported on the use of emergency vein-bypass grafts in cardiogenic shock patients and, in retrospect, patient R.G. would have been a very good candidate since he had only one occlusive lesion in the proximal main left coronary artery. His severe pump failure was recognized by these hemodynamic studies hours before his demise.

Acknowledgment

The author wishes to thank Dr. L. W. Ritzmann of the Portland, Oregon Veterans Administration Hospital who enabled me to initiate these studies in that hospital's Coronary Care Unit. Betoe Nyheim and Diane Coiner of Oregon and Geraldine Makarenko and Dr. J. R. Hill of Edmonton provided expert technical assistance. Drs. J. Dvorkin, R. E. Rossall, R. F. Taylor, S. Lee, and R. S. Fraser of the University of Alberta Hospital, Edmonton, Alberta, provided advice and were supportive during the studies performed on their patients. I am grateful to M. Grace, Ph.D., for his statistical analysis of the data presented in this paper. My wife, Elizabeth Howey Friesen, R.N., was helpful in many ways and her encouragement throughout was invaluable.

References

8. Murphy GW, Glick C, Schreiner BF, Yu PN: Cardiac output in acute myocardial infarction: Serial determination by precordial radioisotope dilution curves. Amer J Cardiol 11: 587, 1963

Circulation, Volume XLVI, March 1973


Sequential Circulatory Changes following Acute Myocardial Infarction in Man: Determination of Optimal Heart Rate at Each Stage of the Infarction by Atrial Pacing

W. GLENN FRIESEN

Circulation. 1973;47:465-475
doi: 10.1161/01.CIR.47.3.465

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/47/3/465

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/