Hemodynamic Findings in 123 Patients with Acute Myocardial Infarction on Admission

By Barry W. Ramo, M.D., Nelson Myers, Andrew G. Wallace, M.D., Frank Starker, Ph.D., David O. Clark, and Robert E. Whalen, M.D.

SUMMARY

Hemodynamic and clinical evaluations of 123 patients with acute myocardial infarction were performed during the first hour of admission to the hospital. In the 123 patients, the right atrial pressure was less than 10 mm Hg in 49 patients, the right atrial oxygen saturation was less than 70% in 97 patients, the arteriovenous oxygen difference was greater than 5.0 vol% in 78 patients. The arterial P_o2 was less than 90 mm Hg in 101 of 107 patients who could be evaluated while breathing room air. The cardiac index was depressed below 3.0 L/min/m^2 in 65 of 98 patients.

The hemodynamic findings generally correlated with the clinical status of the patient; however, within each clinical class of patients there was a wide spectrum of values for each measurement evaluated. There was also considerable overlap of the values found within each clinical classification. It is concluded that hemodynamic evaluation of patients with acute myocardial infarction presents a profile of the patient which is frequently different from the profile that clinical evaluation presents. An objective hemodynamic classification of patients with acute myocardial infarction may provide a more useful index for the evaluation of the patient’s prognosis and for the assessment of preventative therapy.

Additional Indexing Words:
Right atrial pressure, Right atrial oxygen saturation, Cardiac output, Heart failure, Arteriovenous oxygen difference

DOCUMENTATION of the types, incidence, and consequences of cardiac arrhythmias by pioneering coronary care units several years ago provided the vital information necessary to define antiarrhythmic programs which have virtually eliminated arrhythmias as a primary cause of death on coronary care units.1–3 Hemodynamic deterioration now stands as the primary cause of death; yet there is no body of information to document the spectrum of hemodynamic derangements seen early in the course of acute myocardial infarction. Although several groups have reported hemodynamic observations in a small number of selected patients studied at variable periods after hospitalization, no large series of unselected patients has been hemodynamically evaluated during the early period of their hospitalization.4–20

The present study was designed to meet this need for early characterization of hemodynamic findings in a large number of unselected patients with acute myocardial infarction and to evaluate these findings in relation to the clinical findings. It is hoped that information generated from this study will allow a more precise classification of patients with acute myocardial infarction based on objective measurements and will ultimately allow the development of a plan of therapy.

From the Department of Medicine, Duke University School of Medicine, Durham, North Carolina.

This study was supported by Contract No. Ph-43-67-1440 from the National Institutes of Health, and Grant HE 05736 from the U. S. Public Health Service.

Address for reprints: Dr. Barry Ramo, Cardiac Care Unit, Duke University Medical Center, Durham, North Carolina 27706.

Received March 20, 1970; revision accepted for publication May 27, 1970.

Circulation, Volume XLII, October 1970
analogous to that developed after the full significance of various cardiac arrhythmias was recognized.

Methods

Clinical Material

In all patients, the diagnosis of acute myocardial infarction was based on the presence of at least two of the following three criteria: (1) a typical clinical history, (2) electrocardiographic changes of transmural infarction (Q waves and ST changes), and (3) a prompt rise and fall of the serum creatine phosphokinase, lactate acid dehydrogenase, and serum glutamic-oxalacetic transaminase. One hundred twenty-three patients ranging in age from 37 to 84 years who fulfilled these criteria were evaluated. The patient population consisted of 95 males and 28 females. Two patients were below the age of 40, 21 were between 40 and 49 years, 36 between 50 and 59, 44 between 60 and 69 years, and 20 were above the age of 69 years. Seventy-four patients were studied within 12 hours after the onset of symptoms, 24 between 13 and 24 hours, eight during a period between 24 and 48 hours after the onset of symptoms, and four were studied more than 48 hours after the onset of symptoms. In 13 patients the onset of symptoms could not be clearly defined because of pre-infarction angina (nine patients) or the development of shock following infarction (four patients) which may have resulted from the extension of a recent infarction. All patients were examined prior to hemodynamic evaluation and 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, that is, basilar rales or an S3 gallop rhythm, or both. Class III patients had overt pulmonary edema, and class IV patients were in cardiogenic shock as defined by a systolic blood pressure of less than 90 mm Hg, oliguria with a urine output less than 20 cc/hr, cold extremities, and disorientation. No patient with an oral temperature greater than 38.0 C was included in this analysis.

\[
\text{Oxygen saturation} = -13.692 + 1.137 \times (\text{oximeter reading})
\]
\[
(t = 0.997; \text{standard error of estimate}, 1.7%)
\]

Laboratory Techniques

After obtaining informed consent from the patient or his family, hemodynamic studies were carried out on 180 patients within 2 hr of admission to the hospital. Of these 180 patients, 123 subsequently met the previously described criteria for the diagnosis of acute myocardial infarction and serve as the basis for this report. All studies were performed in the cardiac catheterization laboratory on the coronary care unit. Patients who had received analgesics were not studied for at least 30 min after the last medication. One hundred eight patients were studied while breathing room air; 15 patients were too ill to be taken off oxygen. Under local anesthesia a no. 6 Teflon or a no. 7 Zucker catheter* was passed through a basilic or femoral vein and positioned under fluoroscopic control in the high right atrium. A no. 16 Longdwell† Teflon arterial needle was placed in the radial or femoral artery. Arterial and right atrial pressure was measured using as a zero reference 10 cm above the surface of the catheterization table. Arterial and right atrial pressures were measured with Hewlett-Packard transducers and were recorded on a Hewlett-Packard data acquisition system and on analog tape. Cardiac output was estimated by the indicator-dilution technic employing indocyanine-green dye. Dye was injected through the right atrial catheter while arterial blood was being continuously withdrawn by a Sage pump‡ through a Waters densitometer§ at a rate of 30 cc/min. The indicator-dilution curve was recorded on both analog tape and a direct writing recorder. Curves were calibrated by using the patient’s blood and a known amount of dye. The cardiac outputs were calculated on a Sigma V computer** by the method of Thompson and associates.21 Arterial and venous blood samples were taken for determination of hemoglobin, hematocrit, pH, $P_{O_2}$, and $P_{CO_2}$, using an Instrumentation Laboratory system.‡‡ Oxygen saturation was measured on reflection oximeter, model no. 1824.‡‡ The reproducibility of oxygen saturation was ±1% on 200 duplicate samples. The oximeter was calibrated using standards supplied with the machine and by the method of Van Slyke and McNeil.22 The oximeter was linear when the oxygen saturation was between 37 and 100%. A linear regression equation derived from the correlation between the oximeter values and the Van Slyke values was developed to correct the oximeter values to the Van Slyke reading.23 The equation used was:

†Becton, Dickinson Co., Rutherford, New Jersey.
§Waters Co., Rochester, Minnesota.
**Scientific Data System, El Segundo, California.
‡‡Instrumentation Laboratory, Inc., Lexington, Massachusetts.
††American Optical Corp., Bedford, Massachusetts.
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Calculations

The oxygen-carrying capacity was calculated from the product of the hemoglobin times 1.34 cc/g carrying capacity and the oxygen saturation. The arteriovenous oxygen difference was the difference between the calculated arterial and venous oxygen content in volumes per cent. The total peripheral vascular resistance was calculated according to the formula:

\[
\text{Total peripheral vascular resistance} = \frac{\text{Mean aortic pressure} - \text{Mean right atrial pressure (mm Hg)}}{\text{Cardiac output (L/min)}}.
\]

Statistical Analysis

The comparison of selected variables across all patient classes was performed using an analysis of variance. In addition, Scheffé contrasts were used to compare each pair of patient classes. The use of Scheffé contrast has the effect of adjusting the individual \( P \) values associated with each two-class comparison for the multiple comparisons.\(^{23, 24} \)

Results

Hemodynamic Measurements on 123 Patients with Documented Myocardial Infarction

The intra-arterial systolic pressure (fig. 1) ranged considerably. It was 95 mm Hg or less in 26 patients despite the fact that only 15 of these 26 showed other evidence of shock. Twenty-three patients (19%) were hypertensive (BP > 140/90) on admission. The arterial diastolic pressure ranged from 40 to 120 mm Hg, and the mean arterial pressure ranged from 50 to 150 mm Hg.

The right atrial pressure was greater than 10 mm Hg in 49 of 123 (40%) patients (fig. 1). There were only 18 patients with right atrial pressure of > 15 mm Hg. These findings are in contrast to the observation that 69 of 123 patients (55%) had clinical evidence of left ventricular failure or shock.

The arterial pH was between 7.37 and 7.50 in 94 patients. It was above 7.50 in 15 patients

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*Figure 1*

The values of arterial systolic pressure and mean right atrial pressure are shown for 123 patients with acute myocardial infarction. Each dot represents a single measurement in this figure and in figures 2 and 3.

*Figure 2*

Arterial \( \text{PO}_2 \) for 107 patients and right atrial \( \text{O}_2 \) saturation and arteriovenous \( \text{O}_2 \) content difference for 123 patients are noted.
The finding that 97 of 123 patients had an abnormally low right atrial oxygen saturation and only 78 of 123 had a wide arteriovenous O₂ difference can be accounted for by the concomitant arterial desaturation.

The cardiac output was measured in 98 patients. It was below 5.0 L/min in 53 patients (54%), and less than 3.0 L/min in 10 patients. The cardiac index (fig. 3) was less than 3.0 L/min/m² in 67 (68%) patients and below 2.0 in 21 (21%). The stroke volume was <40 cc/beat in 21 patients, between 40 and 80 cc/beat in 51 patients, and >80 cc in 26 patients. The stroke index was <40 cc/beat in 69 patients.

The peripheral vascular resistance (fig. 3) was <15 Wood units in 28 of 98 patients, was in the normal range in 38 patients, and was >20 Wood units in 32 patients.

**Comparison of Hemodynamic and Clinical Findings**

The comparison of the hemodynamic findings of each clinical class is noted in table 1. It is apparent that as the clinical severity of heart failure increases, the measured hemodynamic abnormalities become more prominent.

Figure 4 compares the right atrial pressure and right atrial oxygen saturation (RA O₂ sat) with patient class. There was an increase in RAP as the clinical severity of heart failure increased; however, the degree of overlap among the classes was striking. There was no significant difference in the level of RAP in the patients with no evidence of heart failure and those with mild to moderate heart failure. There was a significant difference between patients in classes I and II and those in pulmonary edema or shock. The RA pressure was above 10 mm Hg in 19% of class I, 33% of class II, 75% of class III, and 82% of class IV patients. Thus, elevations of RAP may be seen without clinical heart failure and clinical evidence of elevated left ventricular end-diastolic pressure may be present with a normal RAP.

RA O₂ saturation decreased as the clinical severity of heart failure increased. There was hemodynamic evidence suggestive of heart failure (RA O₂ sat <70%) in 32 of 54 class I patients and 33 of 37 class II patients. All class
### Table 1

**Comparison of Hemodynamic Measurements by Patient Class**

<table>
<thead>
<tr>
<th>Clinical class</th>
<th>Blood pressure (mm Hg)</th>
<th>RA o₂ sat (%)</th>
<th>pH</th>
<th>Pco₂ (mmHg)</th>
<th>P_O₂ (mmHg)</th>
<th>A-V o₂ diff (vol %)</th>
<th>CO (L/min)</th>
<th>CI (L/min/m²)</th>
<th>SV (ml/min)</th>
<th>SI (ml/min)</th>
<th>TPR (unit)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>123</td>
<td>123</td>
<td>123</td>
<td>123</td>
<td>115</td>
<td>115</td>
<td>107</td>
<td>123</td>
<td>98</td>
<td>98</td>
<td>98</td>
</tr>
<tr>
<td>I</td>
<td>146</td>
<td>78</td>
<td>101</td>
<td>7.6</td>
<td>67.3</td>
<td>7.46</td>
<td>35</td>
<td>71</td>
<td>5.2</td>
<td>6.0</td>
<td>3.1</td>
</tr>
<tr>
<td></td>
<td>±3.4</td>
<td>±16</td>
<td>±19</td>
<td>±4.2</td>
<td>±7.6</td>
<td>±0.05</td>
<td>±5</td>
<td>±14</td>
<td>±1.5</td>
<td>±1.8</td>
<td>±9</td>
</tr>
<tr>
<td></td>
<td>147</td>
<td>77</td>
<td>102</td>
<td>9.3</td>
<td>61.9</td>
<td>7.44</td>
<td>35</td>
<td>64</td>
<td>5.5</td>
<td>5.0</td>
<td>2.7</td>
</tr>
<tr>
<td></td>
<td>±39</td>
<td>±19</td>
<td>±23</td>
<td>±4</td>
<td>±8.2</td>
<td>±0.06</td>
<td>±6</td>
<td>±17</td>
<td>±1.3</td>
<td>±1.3</td>
<td>±0.7</td>
</tr>
<tr>
<td></td>
<td>128</td>
<td>75</td>
<td>92</td>
<td>15.2</td>
<td>47.5</td>
<td>7.46</td>
<td>32</td>
<td>49</td>
<td>7.0</td>
<td>3.5</td>
<td>1.9</td>
</tr>
<tr>
<td></td>
<td>±35</td>
<td>±16</td>
<td>±22</td>
<td>±7</td>
<td>±4.5</td>
<td>±0.06</td>
<td>±5</td>
<td>±9</td>
<td>±1.6</td>
<td>±0.6</td>
<td>±0.5</td>
</tr>
<tr>
<td></td>
<td>92</td>
<td>56</td>
<td>69</td>
<td>13</td>
<td>42.2</td>
<td>7.39</td>
<td>29</td>
<td>49</td>
<td>8.5</td>
<td>2.7</td>
<td>1.6</td>
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<tr>
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<td>±27</td>
<td>±8</td>
<td>±13</td>
<td>±7</td>
<td>±12.1</td>
<td>±0.18</td>
<td>±10</td>
<td>±13</td>
<td>±3.5</td>
<td>±1.1</td>
<td>±0.6</td>
</tr>
</tbody>
</table>

P value <

| I vs. II       | NS                      | NS            | NS  | NS          | NS          | NS              | NS         | NS           | NS           | NS          | NS        |
| I vs. III      | 0.001                   | 0.001         | NS  | NS          | 0.001       | 0.001           | NS         | NS           | 0.001        | 0.001       | 0.001     |
| I vs. IV       | 0.001                   | 0.001         | 0.001| 0.001       | NS          | 0.05           | 0.001      | 0.001        | 0.001        | 0.001       | 0.001     |
| II vs. III     | NS                      | NS            | NS  | NS          | 0.05        | 0.005           | NS         | NS           | 0.05         | 0.001       | 0.010     |
| II vs. IV      | 0.01                    | 0.001         | 0.001| 0.001       | NS          | 0.05           | 0.005      | 0.001        | 0.001        | 0.001       | 0.001     |
| III vs. IV     | NS                      | 0.03          | NS  | NS          | NS          | NS              | NS         | NS           | NS           | NS          | NS        |
III and IV patients had values in the abnormal range. Although there was a statistically significant difference among class I, II, and III patients, the figure illustrates the...
considerable degree of overlap which exists among them. The findings of a given patient within any clinical class are quite variable.

The arterial $P_{O_2}$ while the patient was breathing room air fell as the severity of heart failure increased (fig. 5). The class III patients did not differ significantly from class IV patients. The arterial $P_{O_2}$ was $< 70$ mm Hg in 20 of 51 class I patients, 20 of 31 class II patients, and in all but three of the 22 class III and IV patients.

The A-V $O_2$ difference (fig. 6) widened as the severity of heart failure increased. The class I and II patients differed significantly from the class III and IV patients. Twenty-four of the 54 class I patients showed hemodynamic evidence of heart failure in that the A-V $O_2$ difference was $> 5.0$ vol%. Twenty-five of 37 class II patients, 13 of 15 class III patients, and 15 of 17 class IV patients had an A-V $O_2$ difference $> 5.0$ vol%. The normal A-V $O_2$ difference noted in class III and IV patients was associated with extreme hypoxia (arterial $P_{O_2} < 45$ mm Hg). The arterial pH and $P_{CO_2}$ are noted for each clinical class in table 1. There was a fall in pH as the severity of clinical heart failure increased; however, in the majority of patients within each clinical class the pH tended to be in the upper normal range. The arterial $P_{CO_2}$ fell as the severity of heart failure increased; however, the overlap in the classes was apparent and there was no significant difference among the classes. A mild respiratory alkalosis was the most common abnormality noted. Severe acidosis was uncommon even in the more critically ill patients.

The cardiac index (fig. 6) fell as the clinical severity of heart failure increased. There was a significant difference between class I and II patients and class III and IV patients ($P < 0.05$). There was no difference in cardiac index between those patients with no heart failure and those with mild to moderate heart failure. The cardiac index was $< 3.0$ L/min in 23 of 45 patients (51%) in class I, in 19 of 30 patients (63%) in class II, and in all of the patients in classes III and IV.

The stroke index (fig. 6) generally followed the cardiac index. There was an inverse correlation of stroke index with patient class. In class I patients the stroke indices were

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**Figure 6**

Cardiac index and stroke index are noted in relation to clinical class. The overlap of values among the classes and the wide distribution of values within each clinical class are apparent.
The peripheral resistance and heart rate did not correlate with patient class, and there was no significant difference among the classes.

Discussion

The role of hemodynamic measurements in acute myocardial infarction has not been clearly defined. In 1934, Fishberg and associates measured venous pressure and blood volume in patients with acute myocardial infarction. In 1941 Grishman and Master noted a depression in cardiac output in five patients with acute myocardial infarction studied using the Wezler-Boeger method of indirect cardiac output measurement. No systematic approach to the hemodynamic evaluation of patients with acute myocardial infarction was performed until Fries and associates evaluated 15 patients with acute myocardial infarction utilizing dye-dilution measurements of cardiac output and central venous pressure. Subsequently, a number of investigators have evaluated the changes in cardiac output and blood gases in selected patients with acute myocardial infarction at variable times after admission to the hospital. The results of these studies have indicated that most patients with acute myocardial infarction demonstrate some depression of cardiac output, a widening of arteriovenous oxygen difference, a depression in arterial P\textsubscript{O}_2 and a variable change in peripheral resistance. These studies have been useful in our understanding of the hemodynamic changes which accompany acute myocardial infarction. The value of hemodynamic measurements in classifying patients with acute myocardial infarction cannot be readily ascertained from these studies because most studies only evaluated a single variable, for example, cardiac index or arterial P\textsubscript{O}_2. Also, the small number of patients in each group, the varying time intervals between admission and the performance of the hemodynamic studies, and the selectivity of the studies with the frequent purposeful omission of patients without clinical evidence of heart failure makes an overall analysis difficult. The present report was concerned with the hemodynamic characterization of unselected patients with acute myocardial infarction studied at a similar time after the onset of symptoms and the relationship of the hemodynamic findings to the clinical classification.

The right atrial pressure has been measured frequently in patients with acute myocardial infarction. In the present study the right atrial pressure increased as the degree of clinical heart failure increased, but there was no difference between class I and II patients. Twenty-nine per cent of class II patients and 20% of class III patients had RA pressure of <8 mm Hg. Thus, the presence of left ventricular failure sufficient to produce rales or an S3 gallop, or both, frequently is not associated with elevated RA pressure. These findings are consistent with those of Rapaport and Scheinman who noted that one third of their patients with obvious signs of left ventricular failure had a normal right atrial pressure (<10 cm H\textsubscript{2}O). Cohn and associates have shown that the left ventricular end-diastolic pressure in patients with shock ranged from 12 to 44 mm Hg even when the RA pressure was normal. Thus, the right ventricle may remain competent despite gross evidence of left ventricular failure. The presence of an elevated RA pressure may imply a more severe degree of hemodynamic impairment.

The arterial \textit{P}\textsubscript{O}_2 is frequently depressed in association with acute myocardial infarction. MacKenzie and associates evaluated arterial blood gas changes in 15 patients studied during the first 24 hr after the onset of myocardial infarction. The arterial \textit{P}\textsubscript{O}_2 was less than 80 mm Hg in all patients despite the absence of heart failure in some. Our findings confirm these observations. Both the present study and the study by MacKenzie's group note that the degree of arterial hypoxemia became more severe as the severity of clinical heart failure increased.

The mechanism for arterial hypoxemia in...
the patient in cardiogenic shock has been shown to be due to a right-to-left shuntlike effect, probably secondary to pulmonary congestion and low cardiac output. The mechanism for arterial hypoxemia in certain class I patients is less clear. Recently, Valencia and Burgess found right-to-left shunting and a decrease in the diffusion capacity in class I patients. They postulated that the shunting may be due in part to absence of ventilation of large areas of lung secondary to lack of periodic deep breathing. This hypoventilation results in micro-atelectasis. The present study, indicating that hemodynamic evidence of heart failure was present in one half of the class I patients, suggests that left ventricular end-diastolic pressure may be elevated in many of these patients. Thus, the hypoxia may be related in part to subclinical pulmonary congestion.

The arteriovenous oxygen difference has frequently been used as a measure of cardiac function in patients with various types of heart disease. Surprisingly, there is very little information concerning this measurement in patients with acute myocardial infarction. The A-V O\textsubscript{2} difference was above 5.0 vol% in 44% of class I patients in the present study. This finding indicates a degree of heart failure which was not clinically apparent in a number of "uncomplicated" cases. By contrast, the value was < 5.0 vol% in 32% of class II, 13% of class III, and 11% of class IV patients. Thus, many patients with overt clinical heart failure maintained a normal A-V O\textsubscript{2} difference. The previous studies in which this measurement was evaluated were those of MacKenzie, Valentine, and Fluck and their associates. They noted that the A-V O\textsubscript{2} difference was > 5.0 vol% in almost all patients at some time during their hospital course.

The right atrial O\textsubscript{2} saturation was assessed as an index of cardiac performance. It provides a reflection of the arteriovenous O\textsubscript{2} difference. Barratt-Boyes and Wood noted that the RA O\textsubscript{2} saturation and the pulmonary artery saturation did not systematically differ. They found that the normal value at either site was above 75%. A value at this level was present in only 14 of our 123 patients. Since the RA O\textsubscript{2} saturation is influenced by the arterial O\textsubscript{2} saturation and the hemoglobin content, as well as the peripheral O\textsubscript{2} extraction, it was frequently in the abnormal range when the A-V O\textsubscript{2} difference was normal. The frequent presence of arterial hypoxemia depressed the RA O\textsubscript{2} saturation below the normal level. In the present group of patients, if the arterial P\textsubscript{O}\textsubscript{2} was above 55 mm Hg and the hemoglobin was above 12.0 g, an RA O\textsubscript{2} saturation below 70% was generally associated with widened A-V O\textsubscript{2} difference. Previous investigators have attempted to evaluate the central venous (superior vena caval) O\textsubscript{2} saturation and have found it to be generally below 60% in clinical heart failure. This finding was true in the present study, but a number of patients with A-V O\textsubscript{2} differences greater than 5.0 vol% and clinical heart failure had RA O\textsubscript{2} saturations between 60 and 70%. An RA O\textsubscript{2} saturation below 70% usually reflects a widened A-V O\textsubscript{2} difference in patients with acute myocardial infarction.

The relative rarity of severe metabolic acidosis in our group of patients was similar to that reported by MacKenzie and associates. In the present study, we found the pH to be above 7.35 in all class I and II patients and only rarely depressed in the class III and IV patients. Thus, the arterial pH did not serve to separate the majority of patients from one another. The arterial P\textsubscript{CO}\textsubscript{2} generally tended to fall as the clinical severity of heart failure increased, but as with the pH, there was considerable overlapping from class to class.

Cardiac output has been the most frequently reported hemodynamic measurement in patients with acute myocardial infarction. The estimation of cardiac output has been performed by indicator-dilution methods using the dye-dilution method with intermittent sampling, ear oximeter, continuous sampling, and also precordial detection of radioactive labelled human serum albumin. The cardiac index (> 2.5 L/min/m\textsuperscript{2}) was noted in only 52 of 98 patients examined in the present study. Nineteen of these patients were in clinical class II. All but one of the class III

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and IV patients had low cardiac indices. These findings are in agreement with previous studies suggesting that the degree of clinical heart failure correlates well with the depression in cardiac index, but the presence of clinical heart failure does not necessarily imply an abnormal cardiac index. The range of cardiac index values within each patient class was wide and indicated that, although there was a significant correlation, variation of measured values within each clinical class was sufficient to make clinical class alone a poor predictor of cardiac index.

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25 Years Ago
Christening of a Carotid Vibration

In nine patients in whom subsequent clinical, cardiographic, and cardioscopic examination, established the presence of aortic stenosis and incompetence, we observed a characteristic sign, which by itself permitted a diagnosis of the combined aortic lesion from inspection of the neck. At the height of the carotid pulse, large on account of aortic incompetence, we noticed a quiver or vibration which lasted for a moment only. This effect we have named the carotid shudder for it best describes the visual clinical sign.

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BARRY W. RAMO, NELSON MYERS, ANDREW G. WALLACE, FRANK STARMER, DAVID O. CLARK and ROBERT E. WHALEN

doi: 10.1161/01.CIR.42.4.567

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