Detection of Coronary Heart Disease Using Radionuclide Determined Regional Ejection Fraction at Rest and During Handgrip Exercise: Correlation with Coronary Arteriography

Monty M. Bodenheimer, M.D., Vidya S. Banka, M.D., Colleen M. Fooshee, M.S., John A. Gillespie, M.D., and Richard H. Helfant, M.D.

SUMMARY  The detection of regional asynergy provides strong evidence for a critical reduction of coronary blood flow to that zone. In the present study, the usefulness of combining computer-assisted radionuclide angiography and isometric handgrip exercise testing to detect coronary heart disease (CHD) was evaluated. One hundred twenty-nine patients with chest pain undergoing cardiac catheterization were evaluated using radionuclide angiography. Thirty-four patients were found to have severe contraction abnormalities during the initial radionuclide angiographic study. Of these, 33 had significant CHD. Ninety-five patients had normal or borderline normal left ventricular contraction and therefore underwent a second radionuclide angiogram during handgrip. Radionuclide angiogram data were quantitatively analyzed by computer to determine regional left ventricular contribution to ejection fraction during handgrip stress. Of the 95 patients, 30 had normal coronary arteries of whom 26 (87%) had normal relative regional ejection fraction. Sixty-five patients had CHD; 20 had single and 45 had two or three vessel obstructive disease. Of the 20 with single vessel disease, 16 (nine at rest and an additional seven during handgrip) had an area of decreased relative regional ejection fraction ranging from 31-87% in the corresponding segment during radionuclide angiography. Of the 45 patients with two or three vessel disease, 40 had regional abnormalities in ejection fraction during handgrip of from 31-100% (24 at rest and an additional 16 during handgrip). Moreover, 24 of these patients had multiple abnormalities in relative regional ejection fraction indicating multivessel disease. Overall, of the 95 patients who underwent isometric handgrip stress, sensitivity was 86% for detection of CHD and specificity was 87% for accurately defining the patients with normal coronary arteries.

The results of the present study suggest that the radionuclide angiographic assessment of relative regional ejection fraction during isometric handgrip exercise may provide a useful new diagnostic approach for patients with suspected CHD as well as providing important additional data concerning its location and severity.

EXPERIMENTAL AND CLINICAL STUDIES have demonstrated that regional abnormalities of myocardial contraction are sensitive manifestations of myocardial ischemia. Experimentally, studies by Tennant and Wiggers1 and by others2-3 have shown that immediate and dramatic contraction abnormalities appear literally seconds after a reduction in coronary artery flow. Clinically, the detection of regional asynergy provides strong evidence for a critical reduction of coronary blood flow to that zone. However, a significant number of patients with coronary heart disease (CHD) do not exhibit regional asynergy at rest.4

Isometric exercise (handgrip) is an easily applied and safely administered stress which has been shown to induce abnormal hemodynamics5-7 and asynergy8 on contrast ventriculography in patients with CHD.

Recent advances in isotopic imaging have now permitted an accurate evaluation of regional and global left ventricular performance which correlates well with contrast ventriculography.9-11 In the present study an evaluation was made of the utility of combining computer-assisted radionuclide angiography and isometric handgrip exercise testing to detect CHD in patients admitted for evaluation of chest pain.

Methods

All patients admitted for evaluation of chest pain undergoing cardiac catheterization, contrast ventriculography and coronary arteriography were entered into the study. Patients with valvular, congenital or primary myocardial disease based on cardiac catheterization were excluded. The study was approved by the research council and explained to the patients before performance.

Radionuclide angiograms were obtained using a computerized multicrystal scintillation camera (Baird Atomic System Seventy-Seven) and a one-inch thick parallel hole collimator. After administration of 200 mg of potassium perchlorate, the patient was positioned in the right anterior oblique (RAO) view and a 12-18 mCi dose of technetium-99 pertechn-
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netate, in a volume less than 0.7 ml, was rapidly administered intravenously and flushed with 10–15 ml of D5W to obtain a bolus injection. Counts were recorded at a framing interval of 50 msec (20 frames/sec) during the "first pass" of the isotope. Data were recorded on computer disc for processing and magnetic tape for long-term storage.

The initial raw data were qualitatively reviewed to determine the presence of contraction abnormalities. After 10 minutes, those patients considered to have normal or borderline normal left ventricular contraction were asked to squeeze a hand dynamometer (CH Stoelting and Co) to the maximum extent possible and then maintain contraction at one-third of the predetermined maximum for 3–4 minutes while blood pressure and heart rate were monitored. Care was taken not to allow the patient to perform a Valsalva maneuver. Before the termination of handgrip and after an increase in systolic pressure of greater than 20 mm Hg had been obtained, a background frame was collected, a second injection of pertechnetate administered, and a radionuclide angiogram was recorded as above. Chest pain or serious arrhythmias did not occur in any of the patients. Data acquisition required less than 30 seconds for each injection and resulted in 500,000–750,000 counts whole image during the left ventricular phase.

Right and left heart catheterization was performed using standard techniques. Contrast left ventriculography was performed in the RAO projection using 30–40 ml of meglumine diatrizoate (Renografin-76) injected into the left ventricle. Selective cine coronary arteriography was performed in multiple views using the Judkins or Sones technique. Hemodynamics were monitored and recorded on an Electronics for Medicine oscillographic recorder.

All radionuclide angiogram data were first corrected for non-uniformity of field using a uniform source each day and dead time. For the handgrip study, the background frame was obtained immediately before it was used to correct for preexisting counts. The study was then reviewed in a serial format on an oscilloscope. Frames of data containing the left ventricle were displayed, and using a zone grid representing the individual crystals a region of interest comprising the left ventricle was selected and a time-activity curve generated. The peaks (diastole) and valleys (systole) were utilized to derive a computer-generated end-diastolic and end-systolic frame.

A computer derived image of regional ejection fraction was based on the formula, 

\[
\text{CD} - \text{CS} \div \text{CD} - \text{b}
\]

where \(\text{CD} =\) counts in diastole, \(\text{CS} =\) counts in systole and \(b =\) background. The computer derived end-diastolic and end-systolic images were used to derive stroke volume (fig. 1). This frame was then divided by the end-diastolic frame corrected for background.

In a series of 44 patients with and without coronary disease, background contribution was determined as follows.

Using the computer-generated left ventricular end-diastolic frame, the region of the left ventricle was flagged and the counts displayed as a time activity curve. Accurate depiction of the aortic valve plane has been found possible in our laboratory as well as in others in the RAO projection during the first pass, thus avoiding counts over the aorta. In addition, accuracy of flagging was also confirmed by use of a computer-generated frame which delineated regions surrounding the left ventricle, thereby helping to avoid inadvertent flagging of lung tissue. Background subtraction ranging from 30–50% in 5% increments was applied in all patients and the newly derived time activity curves were utilized to determine ejection fraction by using three peaks and valleys (the maximum peak and the peaks on either side). The ejection fraction so obtained was correlated with the radionuclide ejection fraction determined in the left anterior oblique LAO view in the same patient similar to the approach described by Marshall et al. and, in addition, with contrast ventriculography ejection fraction performed within 24 hours of the radionuclide angigram. Using this approach, a background subtraction of 35% in the RAO projection of all patients provided an excellent correlation with the LAO projection \((r = 0.839; y = 0.9x + 0.05)\) and with contrast ventriculography \((r = 0.801; y = 0.73x + 0.16)\) for a wide range of patients with normal and abnormal coronary arteries and varying severity of left ventricular asynergy.

The image resulting from this technique represents the relative contribution to ejection fraction of different zones of the left ventricle (fig. 2). In addition, for aid of localization of abnormalities, a computer derived end-diastolic perimeter was added to the image of relative regional ejection fraction. The left ventricle was further divided into an anterior and inferior half using a line from the midpoint of the aortic valve plane to the apex.

Changes in regional ejection fraction were quantified as follows: The computer generated the images of relative regional ejection fraction similarly for both the rest and handgrip studies. The full range of 0% activity level to 100% was used. The computer displayed these counts normalized according to the N max (the maximum number of counts in any of the matrix elements of the image) over the full range according to a computer-generated 16 color isocount display. This permitted assessment of relative regional differences in ejection fraction as small as 6.25%. For purposes of analysis, relative decrease of \(\geq 25\%\) (a four-color shift) involving one-third by area of either the anterior or inferior zones was considered indicative of an abnormal regional reduction in ejection fraction.

The severity of a coronary occlusion was assessed by comparing its diameter with the diameter of the vessel immediately proximal to it. The obliquity in which the decrease in diameter was maximal was selected for analysis in each case with \(\geq 75\%\) considered evidence of significant coronary arterial narrowing for purposes of analysis.

Twelve lead electrocardiograms performed routinely on all patients were analyzed for the pres-
ence or absence of pathologic Q waves before cardiac catheterization. Q waves ≥ 0.04 seconds duration were considered abnormal except for isolated Q waves in lead III.

Changes in radionuclide, angiographically-determined relative regional and global ejection fraction were interpreted independently of knowledge of coronary arteriography and were then correlated with the independently determined presence of angiographically-quantified CHD. Statistical analysis was performed using the t test for paired and unpaired data and chi square test where appropriate. All data are given as mean ± SEM.

**Results**

One hundred twenty-nine patients evaluated by catheterization for CHD underwent radionuclide angiography. Of these, 34 patients were considered to have severe contraction abnormalities by radionuclide angiography based on initial qualitative assessment and, therefore, handgrip was not performed. Of these 34 patients, 33 had CHD (four with single vessel, 13 with two vessel and 16 with three vessel obstructive disease). Contrast ventriculography revealed asynergy in 32 of the 34 patients and pathologic Q waves were also present in 22 patients.

Ninety-five patients were considered to have either normal or borderline normal left ventricular contraction during the initial qualitative radionuclide angiographic assessment of wall motion and thus underwent a second radionuclide angiographic study during handgrip stress. Coronary arteriography showed that 30 of these patients had normal coronary arteries. Twenty patients had single vessel disease, and contrast ventriculography revealed normal contraction in 12 and asynergy in eight (five hypokinetic, three akinetic). Forty-five patients had two or three vessel coronary obstructive disease, with normal contraction on contrast ventriculography in 23 and asynergy in 22 (14 hypokinetic, eight akinetic). Of these 95 patients, three patients demonstrated pathologic Q waves; one with single vessel, one with two and one with three vessel disease.

Isometric handgrip exercise resulted in a significant increase in systemic pressure in all patients. Systolic
pressure increased by an average of 31.8 \pm 2.8 \text{ mm Hg} in the normal group, 44.5 \pm 5.8 \text{ mm Hg} in the single vessel and 35.5 \pm 2.7 \text{ mm Hg} in the multivessel group (table 1). None of the patients developed chest pain.

Relative Regional Ejection Fraction at Rest and During Handgrip Stress

Normal Coronary Arteries

Of the 30 patients with normal coronary arteries, 27 had normal relative regional ejection fraction images at rest, showing less than 25% reduction in regional ejection fraction, with a range of 0–19% (fig. 2). Three patients had a single area of relatively decreased regional ejection fraction of 31%, 37% and 37%, respectively. During handgrip stress, 26 remained normal; however, in one patient a new area of relative reduction in ejection fraction of 37% occurred. Of the three patients with abnormalities in relative regional ejection fraction at rest, two remained unchanged with handgrip, while one abnormal area worsened. None of the 30 patients showed more than one area of relatively depressed ejection fraction. Thus, 26 of the

<table>
<thead>
<tr>
<th>Number of Obstructed Vessels</th>
<th>Control</th>
<th>Handgrip</th>
<th>Control</th>
<th>Handgrip</th>
<th>Control</th>
<th>Handgrip</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>128.9 ± 3.5</td>
<td>175.5 ± 10.3*</td>
<td>141 ± 2.9</td>
<td>176.5 ± 4.3*</td>
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<tr>
<td>Diastolic pressure (mm Hg)</td>
<td>82 ± 2.3</td>
<td>110.6 ± 6.2*</td>
<td>86.9 ± 2</td>
<td>108.4 ± 2.2*</td>
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<tr>
<td>Heart rate (beats/min)</td>
<td>78.2 ± 2.3</td>
<td>67.1 ± 2.6</td>
<td>66.2 ± 2.9</td>
<td>90.3 ± 6.9*</td>
<td>80.1 ± 4.6*</td>
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</tr>
</tbody>
</table>

*P < 0.001 compared to control.
All values are mean ± SEM.
of the 20 patients developed depressed regional ejection fraction with handgrip stress averaging $53.8\% \pm 5.3\%$ with a range of 37–87% (table 3). Four of the 20 patients had normal relative regional ejection fraction both at rest and with handgrip (table 2). Three patients had greater than 90% obstructive lesions in the LAD, while one had a 90% lesion in a nondominant left circumflex. Three had normal left ventricular contraction by contrast ventriculography. One patient had a small isolated apical aneurysm. These patients increased their systolic pressures between 35–50 mm Hg.

**Multivessel Disease**

Forty of 45 patients with two or three vessel obstructive coronary artery disease manifested abnormal relative regional ejection fractions (15 had two vessel disease, while 30 had three vessel disease) (table 2). Twenty-four of these patients had areas of abnormal relative regional ejection fraction at rest averaging $46.2 \pm 4.2\%$ with a range of 37–100%. After handgrip exercise, 16 of the 21 patients with a normal relative regional ejection fraction at rest developed a

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**Figure 3.** Images of computer derived left ventricular regional ejection fraction obtained during control (C) and handgrip (HG) exercise. The control image obtained at rest reveals a small area of blue and maroon in the inferior region representing a borderline abnormality compared to the anterior region (red, yellow and white). During handgrip, a marked increase in the maroon area is seen inferiorly which, based on the count pattern, represents a disparity of 37% compared to the anterior area.
involved anterior and inferior zones in 24 of 45 patients with multiple vessel disease (fig. 4). Of the patients with single vessel disease only one demonstrated abnormalities in more than one zone (table 2). This patient had an 80% lesion in the right coronary artery and a 40% lesion in the LAD with a corresponding decrease in ejection fraction in both regions.

Overall, of the 129 patients, analysis of rest data alone detected 66 of 98 patients (67%) with CHD. The addition of handgrip increased the sensitivity to 89 of 98 patients with CHD (91%). Of the 65 patients with CHD who underwent isometric handgrip stress, sensitivity was 86%. Specificity for all patients was 84% and for those undergoing isometric stress was 87% for accurately defining the patients with normal coronary arteries.

Global Ejection Fraction at Rest and During Handgrip Stress

Global ejection fraction at rest was similar in patients with normal coronary arteries and those with either single or multiple vessel obstructive disease. During handgrip, the normal group showed a slight

Figure 4. Images of computer derived left ventricular regional ejection fraction obtained during control (C) and handgrip (HG). The control study demonstrates a large maroon area of decreased ejection fraction involving the inferior zone which compared to the anterior zone represents a 44% disparity in regional ejection fraction. During handgrip, this area is seen to enlarge. In addition, a new area of abnormally reduced ejection fraction is seen involving the anterior area (purple and maroon) indicating a decrease in this zone's contribution to ejection fraction as well.

Table 3. Decrease in Regional Ejection Fraction at Rest and During Handgrip in Patients with Coronary Heart Disease

<table>
<thead>
<tr>
<th>Number of Obstructed Vessels</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regional ejection fraction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rest* mean</td>
<td>44.5 ± 3.7%</td>
<td>46.2 ± 4.2%</td>
</tr>
<tr>
<td>range</td>
<td>31-56%</td>
<td>37-100%</td>
</tr>
<tr>
<td>Handgrip* mean</td>
<td>53.8 ± 5.3%</td>
<td>55.8 ± 3.4%</td>
</tr>
<tr>
<td>range</td>
<td>37-57%</td>
<td>37-100%</td>
</tr>
</tbody>
</table>

*Numbers represent mean and range of new areas of abnormality. All values are given as mean ± SEM.
but insignificant increase in ejection fraction from 0.54 ± 0.02 to 0.57 ± 0.02. In contrast, in patients with CHD, ejection fraction decreased from 0.49 ± 0.01 to 0.45 ± 0.01 (P < 0.005).

**Discussion**

Considerable experimental data have shown that regional myocardial ischemia results in a virtually immediate abnormality of myocardial contraction1-3 preceding both metabolic17 and electrographic changes.18 Thus, a noninvasive assessment of regional contraction during stress to unmask ischemic heart disease has considerable theoretical appeal. Recent advances in computer-assisted radionuclide angiography have provided an accurate means of assessing global and regional left ventricular contraction using either first pass or equilibrium-gated techniques.9-11, 13, 19-21

Recent studies have shown that first pass radionuclide angiography is of considerable value when combined with instrumentation which permits high count rates to be obtained. Marshall et al. have evaluated this technique in the antero-posterior and LAO projection and found it to be highly accurate for both global and regional left ventricular function.10 Since the RAO projection provides the best projection for optimal visualization of the left ventricle, we have recently evaluated the first pass technique in this view.11 Determination of accurate background contribution was an important consideration in this study, although this problem is considerably less with the first pass technique due to the relatively low background level, particularly when compared to equilibrium techniques. Several approaches to this problem have been proposed, as described by Pierson et al.18 The method standardized in our laboratory determines ejection fraction using the change in counts from end-diastole to end-systole, thereby avoiding the problems of geometry inherent in the area length method. Using this approach, an excellent correlation of ejection fraction determined in the RAO view was found compared with either radionuclide angiographically determined ejection fraction in the LAO view as standardized by Marshall et al. and contrast ventriculography.11 Moreover, an excellent correlation was found for the depiction of segmental asynery, as has been confirmed by others.11

Several clinical studies during cardiac catheterization have shown that isometric handgrip exercise is a valuable means of unmasking hemodynamic6-7 and contractile abnormalities.8 In normal patients, handgrip stress results in no significant change in left ventricular filling pressure, stroke volume6-7 or ejection fraction.8 However, in patients with CHD, left ventricular filling pressure generally increases, ejection fraction decreases6-7 and areas of asynery can be unmasked or worsened as determined by contrast ventriculography.8 Several aspects of handgrip stress are particularly important in evaluating patients with CHD. It is safe and simple to perform, requiring only a few minutes, and yields predictable increases in arterial pressure and heart rate which return to control levels within seconds of release.6-8 Moreover, handgrip stress did not result in either patient fatigue, angina pectoris, serious ventricular arrhythmia, motion artifacts or other symptoms or signs which necessitated cessation of the study before interpretable data could be obtained.

In a recent study, bicycle ergometry stress was combined with gated equilibrium radionuclide imaging in the LAO projection.22,23 Using these techniques, wall motion abnormalities were detected in all 47 patients with CHD, including three patients without a history of either angina or myocardial infarction.23 In order to acquire statistically reliable results using the equilibrium technique, imaging for 2 minutes or longer was required.22 A practice session was required to determine the exercise capacity of the individual so that imaging could be performed at or close to peak stress which in turn results in potential motion artifact during the 2-minute period of data acquisition.24 However, despite these problems of suboptimal view and data acquisition, the results were excellent. Handgrip stress does not produce a degree of ischemic stress manifested by either anginal symptoms or ST segment abnormalities comparable to bicycle ergometry.24 However, it is clear that both forms of stress can produce distinct wall motion abnormalities in patients with CHD.8,25

In the present study, the choice of handgrip as the stress in association with radionuclide angiography was not associated with any complications. The consistency of the blood pressure response combined with the ability of all patients to lie quietly while maintaining the level of stress eliminated the potential problem of motion artifact. Currently, radionuclide techniques permit a framing rate of 20-25/sec which is considered adequate for heart rates of up to 105 beats/min.26 Although handgrip is advantageous in this regard due to the relatively low heart rate obtained at peak stress, this factor combined with count characteristics of current systems27 may be responsible for the few false positives, including two patients with incorrectly diagnosed asynery and/or false negatives observed in this study. It is also conceivable that a more rigorous stress as during maximal bicycle ergometry might be useful to detect the few false negatives. An additional problem inherent in any single projection is geometric overlap of left ventricular zones. This, in addition to current limitations in resolution, may be responsible for failure to detect small areas of induced asynery and may lead to false negatives.

The results of the present study suggest that the use of computer-assisted assessment of relative regional ejection fraction at rest and during handgrip stress provides a potentially valuable new approach to the detection, localization and quantitation of CHD. Of the 20 patients with single vessel disease, 80% were detected by assessment of relative regional ejection fraction during handgrip while 89% of 45 patients with two or three vessel disease were detected (table 2). Thus, overall sensitivity for patients undergoing rest
and handgrip radionuclide angiograms was 86%. Conversely, only four of 30 patients without CHD demonstrated abnormalities on regional ejection fraction during handgrip exercise yielding the relatively high concomitant specificity of 87%.

An additional interesting observation was the localizing and quantitative information yielded by this technique. Of the patients with single vessel disease, the relative decrease in regional ejection fraction corresponded to the area supplied by the obstructed coronary artery. Moreover, only one of the patients with single vessel CHD had multiple defects. In contrast, of the 45 patients with two or three vessel disease, 24 (53%) showed multiple abnormal areas during handgrip stress, thereby correctly identifying them as having multivessel coronary artery disease. This finding suggests that the additional patients with single vessel disease compared to multivessel disease.

An important consideration in the evaluation of the results obtained from a radionuclide angiogram is the underlying etiology of the contraction abnormality. Although regional contraction abnormalities at rest suggest CHD, while diffuse abnormalities suggest a cardiomyopathy, this demarcation is not invariable or absolute. Moreover, if one zone is asynergic at rest, a second zone may worsen with handgrip and thus appear to simulate the diffuse asynergy associated with a cardiomyopathy. In this regard, a recent study by Bulkeley et al. has shown that the combined approach of assessing LV function by radionuclide angiography and myocardial perfusion using thallium-201 yields additional diagnostic information. While the sensitivity and specificity of the method reported in the present study appears higher than those reported using thallium-201 stress imaging, no studies have been performed directly comparing these two techniques. Thus, the relative role of thallium-201 imaging during stress and radionuclide angiography during stress remain to be defined by further studies. Indeed, it may well be that these two techniques will be complementary in the evaluation of patients with suspected coronary artery disease.

In summary, the results of the present study suggest that radionuclide angiographic assessment of relative regional ejection fraction during isometric handgrip exercise may provide a useful diagnostic approach for patients with suspected CHD as well as providing important additional data concerning its location and severity.

References

Hemodynamic and Myocardial Metabolic Effects of Ergonovine in Patients with Chest Pain

R. Charles Curry, Jr., M.D., Carl J. Pepine, M.D., Michael B. Sabom, M.D., Robert L. Feldman, M.D., Leonard G. Christie, M.D., James H. Varnell, M.D., and C. Richard Conti, M.D.

SUMMARY The effect of ergonovine on left ventricular hemodynamic and lactate-pyruvate measurements was studied in twenty-six patients. Patients were divided into two groups: Group 1 (seven patients) had a well-documented variant angina syndrome and Group 2 (19 patients) had other chest pain syndromes. Ergonovine was given as the following were evaluated: symptoms, electrocardiographic changes, left ventricular pressure, myocardial lactate-pyruvate metabolism and coronary artery diameter changes. Chest pain and ST elevation occurred following ergonovine in all seven Group 1 patients. Left ventricular end-diastolic pressure increased (14–26 mm Hg mean, \( P < 0.05 \)) and lactate extraction decreased (24% to −7%, \( P < 0.05 \)). Subtotal or total dynamic obstruction of a major coronary artery occurred in each of the six Group 1 patients in whom coronary angiography was repeated during pain. In each case the location of ST elevation corresponded to the area perfused by the dynamically obstructed vessel. In Group 2 following ergonovine 13 patients remained asymptomatic, while six developed chest pain without ST changes. Left ventricular systolic and end-diastolic pressure increased (126–138 mm Hg and 14–17 mm Hg mean, respectively, both \( P < 0.05 \)) associated with a minimal diffuse coronary vasoconstriction. Lactate-pyruvate metabolism remained unchanged. No differences were noted between Group 2 patients with and without chest pain following ergonovine.

Thus, only in patients with documented variant angina did ergonovine induce chest pain with ST elevation associated with hemodynamic and metabolic evidence for myocardial ischemia concomitant with subtotal or total dynamic coronary artery narrowing. In other patients only minimal generalized coronary vasoconstriction without metabolic evidence for myocardial ischemia occurred following ergonovine, regardless of the presence or absence of chest pain.

HEMODYNAMIC CHANGES during spontaneous chest pain with ST elevation, reported by Guazzi et al.,1 2 Gaasch et al.,3 and Maseri et al.,4 include a fall in dp/dt, cardiac output and systolic arterial pressure associated with a rise in left ventricular end-diastolic pressure. No significant change occurs in heart rate. Weiner et al.5 have shown that spontaneous attacks of chest pain and ST segment elevation were associated with myocardial lactate production. Recently, ergonovine maleate has been shown to provoke attacks of chest pain with ST elevation.6 11 However, left ventricular hemodynamics and myocardial lactate-pyruvate metabolism have not been systematically studied in these patients or in other patients with various chest pain syndromes after ergonovine infusion. The purpose of this study was to compare the left ventricular hemodynamic and metabolic effects of ergonovine in seven patients with variant angina and 19 patients with other chest pain syndromes.
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Circulation. 1978;58:640-648
doi: 10.1161/01.CIR.58.4.640
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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