Relationship of Regional Myocardial Perfusion to Segmental Wall Motion
A Physiologic Basis for Understanding the Presence and Reversibility of Asynergy

BARRY M. MASSIE, M.D., ELIAS H. BOTVINICK, M.D., BRUCE H. BRUNDAGE, M.D.,
BARRY GREENBERG, M.D., DAVID SHAMES, M.D., AND HARRIS GELBERG, M.D.

SUMMARY Experimental work has shown that even small reductions in myocardial perfusion impair contractile performance. We, therefore, studied the relationship between regional perfusion, assessed by thallium-201 scintigraphy and segmental wall motion, quantitated on biplane contrast ventriculograms, in patients with coronary artery disease. We evaluated 270 segments in 54 patients, including 27 without evidence of myocardial infarction. Most normally perfused regions (125 of 140) contracted normally, whereas those with scintigraphic defects at rest were usually asynergic (42 of 46). Surprisingly, 57% (48 of 84) of regions with exercise-induced perfusion defects were also asynergic, including 48% (25 of 52) of those in patients without myocardial infarction. In 22 patients who had intervention ventriculograms, improvement of perfusion abnormalities at rest correlated closely with reversibility of asynery. Although there was an association between the location and severity of coronary artery stenosis and segmental wall motion, myocardial perfusion during exercise was a significantly better predictor of asynery.

These findings suggest that resting asynery may occur even in patients without previous infarction, predominately in regions with jeopardized perfusion. Asynery in regions with exercise-induced perfusion abnormalities may, therefore, be an indicator of resting ischemia and may be reversible by coronary artery revascularization.

ASYNERGY, as defined by the presence of localized abnormalities of left ventricular contraction, is a distinguishing characteristic of coronary artery disease.1–3 Segmental wall motion disorders often result from myocardial infarction, but they may also be present in patients without clinical or electrocardiographic evidence of previous necrosis.1–5 In particular, asynergic areas which demonstrate normal or improved contractions resulting from interventions that increase the inotropic state of the myocardium or reduce its oxygen requirements, appear histologically to be composed predominantly of viable myocardium.6 Furthermore, these segments frequently display improved performance after coronary artery revascularization.5, 7–12

In patients without previous myocardial infarction, asynery occurs more commonly in the distribution of the most severely stenosed coronary arteries.4, 13 However, coronary anatomy alone may be misleading in predicting ventricular dysfunction, since many regions supplied by apparently equally diseased vessels contract normally.4, 13

Myocardial perfusion scintigraphy is a recently developed and widely available technique which detects regions of relatively decreased perfusion. Regions with diminished radionuclide uptake at rest generally correspond to areas of prior myocardial infarction, while decreased segmental uptake appearing only during exercise is usually considered indicative of stress-induced ischemia.14–22

The purpose of our study was to determine the extent to which coronary anatomy and myocardial perfusion, as assessed by thallium-201 scintigraphy during exercise and at rest, predict the presence of asynery and its reversibility.

Methods

Patient Population and Patient Subgroups

The 54 patients included in this study were selected from a larger group of 79 consecutive patients who underwent both cardiac catheterization and myocardial perfusion scintigraphy for the evaluation of chest pain and who were subsequently shown to have significant coronary artery disease. Twenty-two patients were excluded because 1) they were studied after coronary bypass surgery, 2) they had accompanying significant valvular disease or 3) their left ventricular angiograms were technically inadequate for quantitation. The other three patients were excluded because they experienced chest pain or manifested ischemic electrocardiographic changes in the catheterization laboratory before ventriculography. The mean age of the patients was 53 years (range 34–71 years). Twelve were women and 42 were men. None of these patients had heart failure, but six had radiographic evidence of
cardiomegaly. Patients underwent both scintigraphy and catheterization within a two-week period.

To clarify the relationship between myocardial perfusion and asynergy, the patients were divided into two subgroups based on the presence of previous myocardial infarction. Group 1 consisted of 27 patients without significant electrocardiographic Q waves, hospitalization for documented infarction or a history of prolonged chest pain suggestive of necrosis. Group 2 was composed of 27 patients with prior myocardial infarction. Eighteen group 2 patients had diagnostic Q waves, two had documented inferior wall infarction with subsequent disappearance of Q waves, five had confirmed nontransmural infarction and two gave a history of infarction without documentation.

**Myocardial Perfusion Scintigraphy**

Perfusion scintigraphy was performed according to the protocol we have previously described.23 In brief, 2 mCi of thallium-201 were injected through an indwelling intravenous infusion line at near maximal treadmill exercise, which was then continued for an additional 30-60 seconds. Exercise was discontinued only when patients experienced severe chest pain, dyspnea or fatigue. Thirty-seven of 54 patients achieved 85% of maximum predicted heart rate. The exercise ECG was positive (1 mm new horizontal or downsloping ST segment depression) in 32 patients, and only five patients had both negative tests and failed to achieve 85% of predicted heart rate. Immediately after recovery, scintigraphy was performed with either an Ohio Nuclear Series 120 or a Searle PhoGamma IV scintillation camera, using converging or linear collimation and a 20% window centered at approximately 75 kev. The initial image was taken in the anterior projection to 200,000 counts, and subsequent 45° left anterior oblique (LAO) and left lateral projections were taken to equal time. Scintigraphy was completed within 30 minutes after injection. Patiens with abnormal stress images were restudied within one week at rest.

The unprocessed scintigrams were evaluated by two independent observers who were unaware of the patients’ clinical status or angiographic findings. The readers agreed initially in their assessment of over 95% of regions and were able to reach a consensus in the remaining ones. Stress and rest scintigrams were initially assessed individually and were then compared. A region was considered to be scintigraphically abnormal when relatively decreased perfusion was present during exercise. Regional abnormalities during stress were further classified as unchanged, improved or abolished at rest.

**Cardiac Catheterization**

Left heart catheterization was performed by the Seldinger percutaneous technique in the postabsorptive state, after light medication with intramuscular diazepam or meperidine and promethazine hydrochloride. Biplane left ventriculography was performed in the 30° right anterior oblique (RAO) and 60° LAO projections, alternately exposing each projection at 60 frames/sec. The angiograms were obtained using diatrizoate meglumine 0.8 ml/kg up to a maximum dose of 60 ml, injected over 4 seconds through a #7 French pigtail catheter.

In five patients with asynergy, 0.4 mg sublingual nitroglycerin was administered 15 minutes after completion of the first ventriculogram. The left ventricular angiogram was then repeated 10-20 minutes later, when a decrease in left ventricular end-diastolic or arterial pressure was noted. During ventriculography, 17 additional patients had ventricular ectopic beats which were followed by compensatory pauses, at least one and one-half times the preceding sinus R-R interval and thus could be evaluated for postextrasystolic potentiation of asynergic segments.11, 23

Outlines of the ventriculograms were traced at end-diastole and at the end of the following systole. The ventricular silhouette with the largest total area immediately before mitral valve closure was traced for end-diastole, and the frame with the smallest area before mitral valve opening was considered end-systole. The same beat was used to trace the RAO and LAO views. In both the initial and postnitroglycerin ventriculograms, only sinus beats which occurred within four contractions following contrast injection and which did not follow ectopic beats were drawn. The first sinus beat following a ventricular ectopic beat was traced to evaluate postectopic potentiation.

The coronary arteriograms were interpreted subjectively by two independent observers. Each coronary artery and its major branches was classified as not significantly obstructed, moderately narrowed (stenosis of 70-90% of the luminal diameter), or severely obstructed (> 90% stenosis).

**Segmental Analysis of Scintigrams and Ventriculograms**

As illustrated in figure 1, five regions of the left ventricle were defined on the perfusion scintigrams. The apex and the anterior and inferior walls were evaluated in the anterior and left lateral projections, and the interventricular septum and posterolateral wall were evaluated in the LAO projection.

Segmental wall motion in the left ventricular angiogram was analyzed by a segmental area ejection fraction method developed in our laboratory.24 The biplane ventricular outlines were divided into eight regions, as indicated in figure 1. The long axes were drawn from the midpoint of the aortic valve plane to the apex in the RAO projection and from the aortic-mitral valve junction to the apex in the LAO view. The long axis was trisected by two perpendicular short axes in the RAO projection, and the two resulting apical regions were then combined into a single area. In the LAO view, a perpendicular was drawn from the midpoint of the long axis to the posterolateral wall, thus dividing the ventricular silhouette into three areas. The area ejection fractions were determined by tracing the ventricular outlines on a digitizer on line to
a programmed desk top calculator. By definition, this method does not require superimposition of systolic and diastolic silhouettes. Ten ventriculograms were analyzed independently by two observers, and the results were highly reproducible. Segmental area ejection fractions varied by less than 10%, and no region was found to be abnormal by one observer and normal by the second observer.

A given segment was defined as having normal wall motion when its area ejection fraction was greater than the mean minus 1.75 SD (the 95% confidence level by the 1-tail t distribution) for that segment among a group of 17 normal subjects. An initially abnormal segment was considered improved post-nitroglycerin or post-ectopic beat only when its area ejection fraction both rose into the normal range and manifested an increase of at least 1 SD.

Correlation of Segmental Perfusion, Wall Motion and Coronary Anatomy

The regions of the perfusion scintigrams were compared with corresponding areas on ventriculograms, as indicated in figure 1. The anterior wall was compared to the ventriculographic areas 1 and 2, the apex to area 3, the inferior wall to areas 4 and 5, the posterolateral wall to areas 6 and 7, and the interventricular septum to area 8. If either of the two areas constituting a region was abnormal or subsequently improved postintervention, that region was considered to be ventriculographically abnormal or improved.

As a best approximation, the left anterior descending coronary artery was considered to perfuse the anterior wall, apex and interventricular septum. The right coronary artery, or a dominant left circumflex coronary artery, was considered to supply the inferior wall and the circumflex was considered to perfuse the posterolateral wall.

Statistical Analysis

The comparison of the association between coronary anatomy and wall motion with that between perfusion and wall motion was made employing McNemar’s test for related samples. All other statistical analyses used contingency tables with the chi square statistic.

Results

Comparison of Patient Groups by Coronary Anatomy

Both the noninfarction and infarction groups had similar coronary arteriographic findings, each averaging 2.4 stenosed vessels per patient and each including similar proportions of patients with one-, two- and three-vessel disease. The prior infarction group had a higher incidence of severe (> 90%) occlusions, 74% compared with 58% in group 1. The mean number of myocardial regions per patient supplied by normal, moderately stenosed, and severely stenosed vessels was 0.7, 1.7 and 2.6 in group 1 and 1.0, 1.0, and 3.0 in group 2, respectively.

Relationship of Myocardial Perfusion to Wall Motion (table 1)

There was a strong correlation between myocardial perfusion and segmental wall motion. Figures 2 and 3
Table 1. Relationship Between Myocardial Perfusion and Segmental Wall Motion

<table>
<thead>
<tr>
<th>TI-201</th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No infarct (n = 135 regions)</td>
<td>Infarct (n = 135 regions)</td>
</tr>
<tr>
<td></td>
<td>Normal</td>
<td>Ex Ind*</td>
</tr>
<tr>
<td>No asynergy</td>
<td>73</td>
<td>25</td>
</tr>
<tr>
<td>Asynergy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reversible</td>
<td>6</td>
<td>27</td>
</tr>
<tr>
<td>Irreversible</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>No intervention</td>
<td>3</td>
<td>14</td>
</tr>
</tbody>
</table>

*Either normal or improved at rest.
†Numbers in parentheses indicate regions with corresponding Q waves.
Abbreviations: Ex Ind = exercise-induced abnormality; Unchg Abn = unchanged abnormality.

illustrate the exercise and rest perfusion images, together with the basal and intervention ventriculographic outlines in two patients. Although the first patient had no history of prior infarction and a normal resting ECG, he displayed extensive abnormalities of perfusion during exercise and corresponding areas of asynery at rest. However, his perfusion abnormalities filled in at rest, and his ventriculogram displayed normal segmental wall motion following an ectopic beat. The second patient had two prior myocardial infarctions and Q waves in both the anterior and inferior leads. He also displayed marked abnormalities of exercise perfusion and segmental wall motion, but neither were reversible.

Table 1 presents our findings for the entire study population. Most (125 of 140) regions with normal perfusion during exercise contracted normally. Regions with abnormal stress perfusion which did not

Figure 2. Studies from a group 1 patient demonstrating extensive areas of exercise-induced abnormal perfusion (indicated by arrows) and corresponding regions with asynery which improve in the post-ectopic beat. Projections are the same as those in figure 1.
FIGURE 3. Studies in a patient with two previous myocardial infarctions. The perfusion defects are unchanged at rest and segmental wall motion abnormalities do not improve in the post-ectopic beat. The lower portion of the LAO scintigram is shielded to minimize hepatic activity. Projections are the same as those in figure 1.

improve at rest, were almost invariably asynergic (42 of 46). These were found predominantly (42 of 46) in the prior infarction patients, and generally corresponded to regions with pathologic Q waves. A significant proportion (over one-half in group 1 and about two-thirds in group 2) of the regions displaying exercise-induced scintigraphic defects were also asynergic. Q waves were not present on the ECG in the leads corresponding to these regions.

There was also close agreement concerning the reversibility of the perfusion and wall motion abnormalities in those patients who had intervention ventriculograms. Asynergy was reversible in 17 of 19 regions with exercise-induced scintigraphic defects, which normalized or improved at rest. In significant contrast ($P < 0.001$), asynergy was not reversible in any of the 21 regions with stress image abnormalities which did not improve.

Relationship of Coronary Anatomy to Myocardial Perfusion and Segmental Wall Motion (tables 2 and 3):

We examined coronary anatomy to ascertain whether the interrelationship between segmental perfusion and wall motion reflected predominantly the location and severity of underlying stenoses. In each

<table>
<thead>
<tr>
<th>Table 2. Relationship Between Severity of Stenosis and Myocardial Perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>% Stenosis</td>
</tr>
<tr>
<td>Normal Tl-201</td>
</tr>
<tr>
<td>NS</td>
</tr>
<tr>
<td>17</td>
</tr>
<tr>
<td>Abnormal exercise Tl-201</td>
</tr>
<tr>
<td>Rest normal</td>
</tr>
<tr>
<td>Rest improved</td>
</tr>
<tr>
<td>Rest unchanged</td>
</tr>
</tbody>
</table>

*Numbers in parentheses indicate regions with corresponding Q waves.
Abbreviation: NS = not significant.
patient group, both the proportion of regions displaying perfusion abnormalities and the severity of these abnormalities grew with increasing degrees of coronary artery obstruction. Thus, perfusion defects were rare in the distribution of vessels without significant stenoses, occurring in only one of 18 and three of 29 such regions in the two patient groups. In contrast, 45 of 70 supplied by vessels with severe stenoses in group 1 and 62 of 78 of those in group 2 patients displayed abnormal scintigraphic perfusion; and 31% and 78% of these, respectively, did not improve at rest. Nonetheless, a significant number of segments supplied by vessels with >70% stenoses, 62 of 117 in group 1 and 35 of 106 in group 2, displayed normal relative myocardial perfusion.

As would be expected, 52 of 56 scintigraphic defects in the noninfarction group completely or partially filled in at rest, while only 32 or 74 of those in the infarction group did so (P < 0.001). Thirty-six of 45 regions with corresponding Q waves manifested fixed perfusion defects, while Q waves were present in only six of 30 regions with exercise-induced defects and three of 63 with normal perfusion (P < 0.001).

The prevalence of asynergy also increased with the severity of coronary artery stenosis. Asynergy was present in only five of a total of 47 segments supplied by normal or insignificantly stenosed vessels, while abnormal wall motion was present in 30 of 70 regions perfused by vessels with greater than 90% narrowing in group 1 patients and in 55 of 78 such regions in group 2 patients (P < 0.001). More noteworthy, perhaps, was the finding of asynergy on the resting ventriculogram in nearly one-third (35 of 117) of the jeopardized regions in patients with no historical or electrocardiographic evidence of previous myocardial infarction. But, since most regions perfused by moderately stenosed vessels in both patient groups displayed normal function, the majority (82 of 117) of the jeopardized regions in group 1 patients and many (41 of 106) of those in group 2 contracted normally.

Among patients with intervention ventriculograms, the reversibility of the asynergy was clearly more closely related to the lack of a previous infarction than to the severity of the stenosis. Wall motion of all 16 asynergic regions in group 1, as opposed to only five of 29 in group 2 patients, normalized post-nitroglycerin or post-ectopic beat (P < 0.001). Q waves were present in 22 asynergic regions in which postintervention assessment could be made, and in 21 of these, the asynergy was not reversible.

**Comparison of Myocardial Perfusion and Coronary Anatomy as Predictors of Asynergy (Table 4)**

Although there was an obvious association between coronary anatomy and segmental wall motion, many segments perfused by moderately or even severely obstructed vessels contracted normally. As shown in table 4, myocardial perfusion, as assessed by exercise thallium-201 scintigraphy, was a significantly better predictor of asynergy than coronary anatomy alone (P < 0.001).

### Discussion

Experimental studies have shown that a reduction of coronary blood flow resulting from transient occlusion or progressive constriction of a coronary artery results in segmental dysfunction of the subserved myocardium.25-29 The studies of Gould and coworkers have shown that such a reduction of coronary blood flow is not present at rest until the severity of stenosis exceeds 85%, but under conditions of diminished distal coronary resistance, lesser obstructions will significantly impair blood flow.30 Restoration of normal blood flow after transient periods of ischemia is associated with eventual return to normal function.31

In man, segmental abnormalities of ventricular contraction have been recognized as a characteristic of coronary artery disease since the advent of ventriculography.14 Such asynergy has been most frequently noted in regions of previous myocardial infarction. Asynergy has also been observed in patients

### Table 3. Relationship Between Severity of Stenosis and Segmental Wall Motion

<table>
<thead>
<tr>
<th>% Stenosis</th>
<th>Group 1 (n = 135 regions)</th>
<th>Group 2 (n = 135 regions)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No infarct</td>
<td>70-90%</td>
</tr>
<tr>
<td>No asynergy</td>
<td>NS</td>
<td>18</td>
</tr>
<tr>
<td>Asynergy</td>
<td>Reversible</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Irreversible</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>No intervention</td>
<td>4</td>
</tr>
</tbody>
</table>

*Numbers in parentheses indicate regions with corresponding Q waves.

Abbreviation: NS = not significant.

### Table 4. Comparison Between Severity of Stenosis and Thallium-201 Myocardial Perfusion as Predictors of Asynergy

<table>
<thead>
<tr>
<th>Stenosis</th>
<th>Perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;70%</td>
<td>≥70%</td>
</tr>
<tr>
<td>No asynergy</td>
<td>42</td>
</tr>
<tr>
<td>Asynergy</td>
<td>5</td>
</tr>
</tbody>
</table>

P <0.001 P <0.001
without evidence of infarction and in regions without corresponding electrocardiographic or pathologic findings of necrosis. In particular, regional wall motion disorders, which normalize following interventions that improve contractility or reduce the imbalance between myocardial oxygen supply and demand, have been thought to be more likely due to ischemia than to scar. Studies by Helfant and coworkers have shown that fibrosis is less extensive or absent in regions with reversible asynchrony. Measurements of regional myocardial blood flow, however, have not demonstrated decreased perfusion in patients without infarction; thus, no direct evidence for resting ischemia is available.

Relationship of Coronary Anatomy to Segmental Wall Motion and Perfusion

Our findings on the relationship between coronary anatomy and segmental wall motion are similar to those reported by others. Asynergic regions were more common in the distribution of severely stenosed vessels and were present most frequently in patients with prior infarction. In addition, asynchrony was present in 15 of the 27 patients without evidence of previous infarction and was found in nearly one-third (35 of 117) of the regions perfused by stenosed vessels in these patients. Asynchrony was always reversible in the noninfarction patients, but only infrequently in our group 2 patients. Unlike Helfant et al., we did not find the reversibility of asynchrony to be related to the severity of coronary stenosis per se.

Thallium-201 myocardial perfusion scintigraphy is an accurate measure of myocardial perfusion in experimental preparations. Clinically, it is a reliable method for detecting regions of infarction (diminished uptake at rest) and exercise-induced ischemia (diminished uptake during exercise). The presence of normal scintigrams in seven of 54 patients with significant coronary artery disease in this study is comparable to other reports. Regions supplied by severely narrowed (> 90%) vessels were more likely to display reduced perfusion than those supplied by less diseased arteries (70-90% stenosis). Overall, 58% of segments supplied by significantly stenosed vessels demonstrated decreased perfusion.

Relationship of Myocardial Perfusion to Regional Wall Motion

The principal objective of the present study was to examine the relationship between myocardial perfusion and asynchrony. Our findings are consistent with experimental studies which have shown that segmental dysfunction increases with decreasing coronary blood flow. In both patient groups, asynchrony occurred predominantly in abnormally perfused regions. In the infarction group, the incidence of asynchrony increased and the likelihood of its reversibility decreased as the rest scintigrams became more abnormal. Previous studies have noted the correlation between regions manifesting abnormal relative myocardial perfusion at rest and asynchrony.

Myocardial perfusion, as assessed by exercise and rest thallium-201 scintigrams, correlated significantly better with segmental wall motion than did coronary anatomy; 123 of 223 (55%) regions supplied by significantly narrowed vessels contracted normally, compared with only 40 of 130 (31%) regions with exercise perfusion abnormalities (P < 0.01). This closer agreement of perfusion with the status of segmental contraction is probably in part explained by inaccuracies in the subjective grading of stenoses. In addition, perfusion scintigraphy is a physiologic indicator of blood flow and thus reflects the variability of regional blood supply, as well as collateral circulation.

The findings in patients with intervention ventriculograms suggest that the relationship between regional myocardial perfusion and function has even greater clinical importance. With only two of 19 exceptions, initially abnormal regions which displayed normal or improved perfusion at rest, also contracted normally postintervention. Conversely, no asynergic region without improved perfusion at rest normalized postintervention. Similar findings have also been reported by others in preliminary form.

In patients with previous infarction, the presence of myocardial regions which displayed both abnormal perfusion and wall motion was expected. Many manifested perfusion defects, both at rest and during exercise, electrocardiographic Q waves, and little change in function postintervention. A few, however, predominantly in patients with nontransmural or poorly documented infarctions and those in whom Q waves had disappeared, displayed both perfusion abnormalities which improved at rest and reversible asynchrony. Presumably, such areas have only subendocardial or patchy fibrosis or are adjacent to regions of transmural infarction.

The underlying pathology in the patients with asynchrony but no prior infarction is less clear for this reason, they were considered separately. Their ECGs were often entirely normal. In these patients, both perfusion and wall motion abnormalities were reversible. It is conceivable that some of these regions might be partially fibrotic due to silent infarction, but previous studies have indicated that some asynergic segments have both appeared normal at the time of surgery and have been free of significant fibrosis on pathologic examination. It seems likely, at least in the noninfarction group, that regional contraction disorders occur secondary to ischemia. While these, for the most part, are in the distribution of severely (> 90%) stenosed coronary arteries, they also occur predominantly in the subset of those jeopardized regions which also manifest abnormal perfusion during exercise. Several studies have now documented improved regional wall motion in some patients after coronary artery revascularization.
exercise-induced perfusion defects are those most likely to improve. Indeed, our preliminary studies have indicated that changes in regional perfusion post-bypass surgery correlate well with changes in segmental wall motion.44

One point requiring further discussion is the correlation between asynergy at rest and perfusion abnormalities during exercise. If resting asynergy reflects ischemia, as hypothesized, then one might ask why myocardial perfusion at rest appears normal. Although in some patients the stress of catheterization may have provoked symptomatically or electrocardiographically silent ischemia, a more likely explanation is the relative insensitivity of thallium-201 scintigraphy as a measure of perfusion. Experimental studies have demonstrated that regional function is depressed with as little as a 25% reduction in blood flow.28, 29 In contrast, a recent study has suggested that nearly a twofold ratio of normal to reduced regional flow is required before abnormal perfusion is apparent.44 Such imbalances in coronary blood flow do not often occur at rest in patients with stable angina, in the absence of previous infarction, even when severe obstructive disease is present.33–36 A few studies have noted scintigraphic abnormalities at rest in patients with coronary artery spasm and unstable angina,46–48 but in these conditions myocardial perfusion may be more severely compromised. During exercise and other hyperemic states, variations in regional blood flow are exaggerated.39–42 Thus, one might speculate that regions with exercise-induced perfusion abnormalities are those most likely also to have reduced perfusion at rest, although the latter may not be detectable by thallium-201 scintigraphy. Another possible explanation for resting asynergy without underlying fibrosis is that dysfunction may persist after repeated transient periods of ischemia. Animal studies have demonstrated such a delay in functional recovery following brief coronary occlusions.40

Limitations of Methodology

Several methodologic limitations of the present study should be mentioned. The interpretation of coronary arteriograms is subject to both considerable interobserver variability39 and to occasional significant error when compared to postmortem studies.40, 41 In addition, the distribution of the coronary vasculature assumed for the comparison with regional perfusion and wall motion, while generally valid, is only an approximation.

The interpretation of thallium-201 scintigrams is also quite subjective. However, in the present study the two blinded readers agreed initially in approximately 95% of all segments. Thallium-201 scintigraphy is a relative, rather than an absolute, technique. Thus, regions with impaired perfusion might appear scintigraphically normal relative to other regions with more severely compromised blood flow. Experimental studies have also indicated that small (< 5 g) areas of abnormal perfusion are probably beyond the present resolution of this technique.51

Regional wall motion analysis is fraught with methodologic difficulties and depends on unproven assumptions concerning the geometry of left ventricular contraction. The area ejection fraction method used in this study, however, does not depend on changes in shortening of a small number of arbitrarily placed cords. In addition, the variance for each region in a group of normal subjects was small, permitting strict (95%) confidence limits. This area ejection fraction method proved to be the most sensitive for detecting abnormal segmental wall motion in our preliminary study.24 The ventriculograms were not performed in precisely the same projections as the scintigrams, since adequate resolution is not possible in RAO thallium-201 scintigrams because of tissue attenuation. Nonetheless, the regions evaluated by the two studies are closely contiguous and represent the distribution of the same coronary arteries.

Conclusions

In this study we investigated the relationship between coronary artery anatomy, regional myocardial perfusion and segmental wall motion. Our results indicate: 1) Abnormal perfusion and function are both more prevalent in the distribution of severely obstructed vessels, but they are frequently not present in other regions supplied by similarly diseased arteries; 2) myocardial perfusion correlates more closely than coronary anatomy with segmental wall motion; 3) resting asynergy in patients without prior myocardial infarction is not uncommon and occurs almost exclusively in regions with jeopardized perfusion, as indicated by exercise-induced perfusion abnormalities; 4) asynergy, when present in regions with stress-induced abnormalities of perfusion, is usually reversible on intervention ventriculography and potentially so by myocardial revascularization.

Thus, our findings suggest that a relationship between myocardial perfusion and function, similar to that demonstrated in experimental models, also exists in man and thus provides a better understanding of the existence of asynergy in some regions with jeopardized blood flow.

Acknowledgments

We would like to express our gratitude to Ms. Rebecca Fernandez and to the other technicians of the Cardiac Catheterization Laboratory and Nuclear Medicine Laboratory, and to Mrs. Kathleen Hecker for her editorial assistance. We are also indebted to Drs. Kanu Chatterjee and William W. Parmley for their encouragement and helpful criticism.

References


42. See JR, Holman BL, Maddox DE, Adams DF, Cohn PF: Markedly reduced regional myocardial blood flow as predictor of non-viable myocardium in areas of abnormal wall motion. (abstr) Circulation 56 (suppl III): III-51, 1977


46. Maseri A, Parodi O, Severi S, Pesola A: Transient transmural...
Emergency Revascularization for Unstable Angina

LEONARD A. R. GOLDMING, M.D., FLOYD D. LOOP, M.D., WILLIAM C. SHELDON, M.D., PAUL C. TAYLOR, M.D., LAURENCE K. GROVES, M.D., AND DELOS M. COSGROVE, M.D.

SUMMARY Emergency revascularization for unstable angina (defined according to criteria of the National Cooperative Study Group) was performed in 100 consecutive patients. The mean interval from onset of pain to operation was one day. Nineteen patients had single-vessel narrowing of greater than 70% of lumen diameter, 32 double-vessel obstruction and 49 triple-vessel disease. Fourteen of these patients had left main trunk obstruction. Four patients died within 30 days, three from complications of myocardial infarction. Seventeen of 96 (18%) early survivors sustained perioperative infarction. After a mean follow-up of 42 months, four late deaths and three late infarctions occurred. Postoperative angiography in 47 patients (mean interval 14 months) showed 86% graft patency. Of 92 survivors, 72 are symptom-free. Three of the four operative deaths occurred within 24 hours postoperatively; in each of these, postmortem examination confirmed a recent myocardial infarction which antedated the operation, despite the absence of new infarction in the preoperative electrocardiogram or elevation of cardiac enzymes. Results from this emergency series suggest that, although myocardium may be salvaged in some instances, in other cases infarction has already occurred and treatment might better be directed toward alleviation of acute ischemia to provide a stable period in which diagnostic studies are performed and acute myocardial infarction may be ruled out.

SINCE MYOCARDIAL INFARCTION is a retrospective diagnosis, anginal pain that may precede myocardial infarction is unpredictable and difficult to classify. Depending on criteria for definition, the syndromes of unstable angina probably have varying prognoses. The National Cooperative Study Group has created a homogeneous category for definition purposes and undertaken a randomized prospective study of medical and surgical treatment. Their protocol required a period of stabilization and observation to rule out infarction. Until recently, our therapeutic approach to unstable angina included immediate operation whenever the angiograms were completed, i.e., to dispense with the traditional "cooling off" period.

Our protocol for surgical treatment of unstable angina has been relatively uniform over the past five years. Patients who were thought to have unstable angina were admitted to the coronary care unit, treated with sedation and nitrates, and if severe coronary atherosclerosis was documented by angiography, the patient was offered emergency revascularization. The interval from admission to operation was brief in most instances, and the objective was to provide expeditious emergency revascularization. In this analysis, we selected consecutive patients who met the criteria of the National Cooperative Study, except that we included patients with severe left main coronary disease. The results reported here represent our initial approach to the surgical treatment of unstable angina.

Clinical Material

One hundred consecutive patients who underwent emergency revascularization for unstable angina between December 1970 and January 1976 were reviewed retrospectively. Excluded from this report are patients with unstable angina who became stable during an initial period of observation and had elective surgery later and patients who had previous graft surgery.

Criteria used to define the study group were: 1) episodes of angina at rest, either of new onset or a changing pattern, more frequent, more severe, more prolonged, or occurring at rest, and which was

From the Department of Thoracic and Cardiovascular Surgery, The Cleveland Clinic Foundation, Cleveland, Ohio.

Address for reprints: Leonard A. R. Golding, M.D., Department of Thoracic and Cardiovascular Surgery, The Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, Ohio 44106.

Received May 24, 1978; revision accepted August 10, 1978.

Circulation 58, No. 6, 1978.
Relationship of regional myocardial perfusion to segmental wall motion: a physiologic basis for understanding the presence and reversibility of asynergy.
B M Massie, E H Botvinick, B H Brundage, B Greenberg, D Shames and H Gelberg

Circulation. 1978;58:1154-1163
doi: 10.1161/01.CIR.58.6.1154

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/58/6/1154

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/