The Relationship between Abnormal Echocardiographic Septal Motion and Myocardial Perfusion in Patients with Significant Obstruction of the Left Anterior Descending Artery

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SUMMARY This study assesses the relationship between segmental myocardial function and coronary perfusion in patients with high-grade stenosis of the left anterior descending artery. Twenty-five patients with critical lesions (> 70%) were divided into two groups according to the absence or presence of normal echocardiographic septal motion. Twelve patients had abnormal echocardiographic septal motion (AESM) and 13 patients had normal septal motion. Septal perfusion was evaluated by intracoronary injections of radiolabeled macroaggregated albumin (MAA) particles. Of the parameters analyzed abnormal septal perfusion was best related to AESM. Among the 12 patients with AESM, ten had absent resting septal perfusion. Of the 13 patients with normal septal motion, only two had abnormal septal perfusion. Septal width was also significantly thinner in patients with AESM. When angiographic collateralization was associated with septal perfusion as detected by injection of MAA into the right coronary artery, normal septal motion was present (five patients). When no septal perfusion resulted from right coronary injection, even though collaterals were seen angiographically, AESM was found (four patients). Thus, in patients with severe left anterior descending stenosis the presence of abnormal echocardiographic septal motion strongly suggests absent septal perfusion and most likely infarction. On the other hand, normal echocardiographic septal motion implies that resting septal perfusion is normal.

ECHOCARDIOGRAPHY HAS NOW BECOME a useful and accepted noninvasive procedure for evaluating left ventricular performance.1-7 Although the procedure has been most helpful in assessing left ventricular function when the ventricle is diffusely diseased, the echocardiogram is also of proven benefit in defining abnormal contractility in patients with coronary artery disease.8-10 However, since all segments of the ventricle are not accessible by echocardiography and since coronary disease may produce segmental ventricular dysfunction, it is not always possible to detect all diseased segments of the ventricle. On the other hand, the echocardiogram can provide useful information regarding the interventricular septum, a structure which is not easily evaluated by conventional angiographic techniques.11, 12

An important and common cause of abnormal echocardiographic septal motion (AESM) is high grade obstruction of the left anterior descending artery.5, 8, 10, 13 This abnormality of the septum when seen in the presence of significant obstruction of the left anterior descending would seem to imply myocardial ischemia and/or infarction or fibrosis of the septum. A recent report which includes pathological studies related septal thinning to scarring of the septum.12 Some authors have further suggested that AESM implies not only a critical left anterior descending lesion, but that the lesion is proximal to the first septal perforator branch.10, 13

Despite the usefulness of coronary angiography in defining the presence of significant obstruction of the major coronary arteries, the procedure does not allow visualization of small vessels and therefore does not provide complete information concerning resting myocardial perfusion. Accordingly, a more precise definition of myocardial perfusion and function seems necessary to enhance understanding of the significance of an obstruction of a major coronary artery.

A procedure which can be utilized to study myocardial perfusion in patients with coronary artery disease is that of selective intracoronary injection of radiolabeled macroaggregated albumin (MAA) particles.14-18 The distribution of these particles is dependent upon coronary blood flow throughout the heart and as such the procedure provides information regarding distribution of myocardial blood flow at the capillary level. This study was therefore designed to evaluate more critically the significance of severe narrowing of the left anterior descending lesion on both septal perfusion and function.

Materials and Methods

Patients suspected of having coronary artery disease and who were referred to The Ohio State University Hospitals for coronary angiography from October 1973 through June 1975 were considered as potential candidates for this study. Twenty-five patients with significant obstruction (defined as greater than 70% luminal narrowing) of the left anterior descending artery, technically adequate echocardiograms, and myocardial scintigraphy were included in this study.

Echocardiography is routinely performed in all patients undergoing coronary angiography. M-Mode echocardiograms were recorded by the same technician using the same technique and employing a conventional ultrasonoscope and strip chart recorder. Patients who were known to have other recognized causes of AESM such as left bundle branch block19-21 and right ventricular volume overload22 were excluded. Septal thickness and motion were recorded just below the mitral valve using sweeps toward the apex. Normal septal thickness was defined as greater than 7 mm;11-12 normal septal motion was defined as greater than 3 mm of posterior-directed systolic motion.9 Septal motion was mea-
All scans were performed in three projections using a gamma scintillation counter and a medium energy collimator. The images were recorded on X-ray films and defects were assessed qualitatively by three independent observers. Figure 2 is a normal perfusion scan showing the 45º left anterior oblique (LAO) view in a normal patient.

Left ventricular angiography was performed in the 30º right anterior oblique (RAO) projection using 36–42 cc of 90% hypaque. In addition to calculating the left ventricular volume and ejection fraction, left ventricular wall motion was subjected to segmental analysis. The observed abnormal anterior wall motion was compared to AESM. Statistical analysis was based on Fisher’s exact t-test for contingency and Student’s t-test of paired data using Hewlett-Packard 9600-B computer with a standard program.

**Results**

The 25 patients were divided into two groups on the basis of septal motion defined by echocardiography. AESM was recorded in 12 patients (48%) and normal septal motion in 13 patients (52%). Table 1 indicates the various parameters analyzed in relation to the absence or presence of normal septal motion.

Figure 3 shows septal motion measurements of all patients. Of the 12 patients with AESM seven patients had no motion or paradoxical motion of the septum. Although the septal width was significantly thinner in this group AESM was not invariably associated with an abnormally thin septum. Figure 4 shows a septum with abnormal motion but normal thickness. Figure 5 demonstrates a more common abnormality of the septum in which there is both abnormal systolic motion and a thin septum. The mean septal thickness in patients with AESM was 0.9 cm with a range of 0.5 to 1.3 cm. Only one of these patients had a septal width greater than 1.0 cm. On the other hand the septal thickness in the group with normal septal motion averaged 1.1 cm with a range of 0.9 to 1.2 cm. Eleven of these patients had septal widths of 1.0 cm or greater.

Of all the factors analyzed (table 1) AESM best correlated with abnormal septal perfusion as detected by myocardial scintigraphy. Ten of the 12 patients with AESM had perfusion defects of the septum on their scintigrams. Figure 6 shows a scintigram of a patient with AESM. There is a gross perfusion defect of the interventricular septum consistent with complete absence of septal perfusion. This image is typical of the 10 patients who had absent septal perfusion and AESM.
TABLE 1. Septal Motion in 25 Patients

<table>
<thead>
<tr>
<th></th>
<th>Abnormal septal motion (N = 12)</th>
<th>Normal septal motion (N = 13)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septal perfusion defect</td>
<td>10/12</td>
<td>2/13</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Septal thickness</td>
<td>0.9 cm ± .20</td>
<td>1.1 cm ± .1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Past history of infarction</td>
<td>8/12</td>
<td>5/13</td>
<td></td>
</tr>
<tr>
<td>Anteroseptal infarction on ECG/VCG</td>
<td>7/12</td>
<td>4/13</td>
<td></td>
</tr>
<tr>
<td>Anterior wall hypokinesis</td>
<td>9/12</td>
<td>9/13</td>
<td></td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>47 ± 15</td>
<td>52 ± 15</td>
<td></td>
</tr>
<tr>
<td>Lesion proximal to first septal perforator</td>
<td>3/12</td>
<td>8/13</td>
<td></td>
</tr>
<tr>
<td>Angiographic collateral flow (100% obstruction LAD)</td>
<td>5/6</td>
<td>7/7</td>
<td></td>
</tr>
</tbody>
</table>

Figure 7 shows a perfusion scan in a patient in whom the interventricular septum was totally perfused by collaterals from the right coronary artery. Even though the septum was not being supplied from the anterior descending artery, collateral flow from the right coronary artery was adequate to maintain normal septal motion and in such cases we consider septal perfusion to be present. This can best be appreciated in the dual view. In the two patients with AESM but normal septal perfusion, normal septal thickness was present in both patients. Neither of these two patients had evidence of anteroseptal myocardial infarction by the electrocardiogram, whereas seven of the ten patients with AESM and perfusion defects had electrocardiographic evidence of infarction. Those patients having both AESM and evidence of a previous anteroseptal myocardial infarction invariably had septal perfusion defects. In the 13 patients who had normal echocardiographic septal motion 11 demonstrated normal perfusion of the interventricular septum. Of particular note is the observation that no patient with a septal excursion of greater than 5 mm had abnormal perfusion (fig. 3). In the two patients from the group having a perfusion defect and normal septal motion, one had an anteroseptal myocardial infarction on the electrocardiogram and the other had evidence of anteroseptal infarction on his vectorcardiogram.

Twenty-three of the 25 patients in this study had angina pectoris and 13 had a clinical history of myocardial infarction. Eight of the 12 patients with AESM had a clinical history of an infarction whereas only five of 13 patients with normal septal motion had a history of infarction. The ECG diagnosis of anteroseptal infarction was based upon the presence of frank Q waves in the anterior precordial leads. In the 12 patients with AESM the ECGs showed the following: six anteroseptal infarction, two poor R wave progression, two inferior infarction, and two normal. Nineteen of 25 patients had vectorcardiograms (VCG). The VCG revealed an anteroseptal infarction in one of the two patients with poor R wave progression and left posterior hemiblock in one patient with anterior Q waves. Thus, seven patients with

**Figure 3.** This graph shows septal motion measurements of all patients. The patients above the broken line were considered to have normal septal motion and those below AESM. The open circles and triangles represent those patients in whom perfusion and septal motion did not correlate.

**Figure 4.** This echocardiogram demonstrates normal septal thickness; however, the posterior motion of the septum is abnormal (3–4). The motion of the posterior wall is normal (1–2). This patient did not have normal septal perfusion.
The severity of the left anterior descending lesion was the same in both groups as mean percent luminal narrowing of 92%. Six patients with AESM had complete obstruction of the left anterior descending artery, whereas seven patients with normal septal motion had 100% obstruction of the left anterior descending artery. Location of the lesion was proximal to the septal perforator in eight patients with normal septal motion and in only three with AESM. Thus, neither the severity of narrowing nor the location of the lesion were related to AESM.

Angiographic evidence of collateral vessels in the patients with 100% obstructive lesions occurred with nearly equal frequency in the two groups as seven patients with normal septal motion and five patients with AESM had collateralization of the left anterior descending artery from the right coronary artery. Five of the seven patients who had normal septal motion and angiographic collateral flow to the septum had normal perfusion as detected by scintigraphy. On the other hand, four of the five patients with AESM and collateral flow to the septum had abnormal perfusion scans despite the fact that angiographic evidence of collateral flow was well visualized in this group also.

**Discussion**

In patients with coronary artery disease, abnormal motion of the interventricular septum by echocardiography nearly always indicates abnormal myocardial blood flow to the septum as detected by selective scintigraphy. This observation is in agreement with a recent study performed acutely in dogs whereby segmental dyskinesis correlated with impaired myocardial perfusion as determined by the use of radioactive labeled microspheres. There were two patients in our study with AESM and normal septal perfusion. Kerber et al. also demonstrated apparently normal myocardial perfusion in the presence of abnormal left ventricular wall motion in dogs. These investigators proposed that this seemingly paradoxical situation might be due to either undetected areas of ischemia or passive influence of adjacent severely ischemic myocardium upon normally perfused myocardium. This explanation might also apply to our patients. It is noteworthy that normal septal thickness was present in both of these patients.

There were two patients who had normal echocardiographic septal motion and a definite perfusion defect. The lesions were distal to the first septal perforator in both patients and each patient had either ECG or VCG evidence of a previous anteroseptal myocardial infarction as well as anterior wall contraction abnormalities on the ventriculo-
gram. It is possible that the area of AESM was present but missed by the echocardiogram or that the perfusion defects were artifacts produced by selective streaming of the MAA particles.

In addition to nearly always being seen with perfusion defects, AESM was also associated with a significantly thinner septum. This finding is not surprising as it has been shown recently that a thin septum in a patient with coronary artery disease does represent an area of myocardial infarction. Our study lends further support to these findings in that the four patients with a septal width of 0.7 cm or less and AESM had perfusion defects. On the other hand, eight patients with AESM had normal septal width but still had perfusion defects. It would therefore appear that a thin septum further supports lack of perfusion but a septum of normal thickness does not necessarily imply that blood supply to the septum is normal particularly when its motion is abnormal.

When attempting to relate AESM to various other parameters, it is apparent that AESM is associated with only the absence of septal perfusion as detected by scintigraphy and with septal thinning. Although a clinical history of myocardial infarction and the presence of ECG/VCG evidence of anteroseptal infarction were more common in patients with AESM, these data were not reliable in predicting whether or not the septum was perfused. Two patients demonstrated ECG evidence of anteroseptal infarction but had normal septal motion and perfusion suggesting that the septum was spared of infarction even though the ECG indicated otherwise. This observation indicates that the presence or absence of septal motion and perfusion defects may not correlate strictly with the ECG findings of myocardial infarction.

Likewise, the presence of anterior wall motion abnormalities on the ventriculogram did not correlate with septal motion or perfusion. Since the anterior left ventricular wall and septum are both supplied by the LAD one might expect both structures to have abnormal motion with significant LAD disease particularly when the lesion is proximal. Anterior hypokinesia is certainly a common finding among patients with high-grade LAD lesions as demonstrated in 18 of 25 patients in this group. Yet, only one-half of these patients had AESM and septal perfusion defects. There are two possible explanations: 1) the anterior wall was infarcted but not the septum, or 2) collaterals had re-established septal perfusion in these cases but had not re-established perfusion to the anterior wall.

It is particularly noteworthy that the presence of collateral flow from the right coronary artery to the left anterior descending artery as detected by angiography did not necessarily indicate perfusion of the septum as detected by scintigraphy. In the seven patients with a completely obstructed left anterior descending artery and normal septal motion, five had normal perfusion of the septum by way of collateral flow from the right coronary artery. On the other hand, of five patients with completely obstructed left anterior descending arteries and abnormal septal motion, four had no evidence of septal perfusion despite the presence of collateral flow on the angigram. Thus, collaterals appear to be effective in perfusing the septum in some patients but not in others and where they are effective normal septal motion is usually preserved.

Our study agrees with the results of other investigators in finding a relationship between AESM and left anterior descending coronary disease. The usefulness of the echocardiogram in predicting the location of the LAD lesion is controversial. This study does not show a relationship between AESM and the location of the disease proximal to the first septal perforator and is in agreement with a recent publication by Gordon and Kerber. In our study, it was not the location of the left anterior lesions, but the presence of a perfusion defect that was best related to echocardiographic septal motion.

The pathophysiological significance of abnormal resting scintigraphy remains uncertain. Weller et al., using technetium human albumin microspheres, and Shelburt using technetium MAA particles produced experimental infarction in dogs that were later studied by selective scintigraphy. Pathological correlation indicated that the perfusion defects seen were identifying areas of infarction. Correlative pathological studies have not been done in humans. Hamilton et al., using technetium MAA selectively injected into the coronary arteries, felt that the resultant perfusion defects represented myocardial scarring secondary to infarction as these defects correlated with Q waves or a history of myocardial infarction. In two patients who died and two others who had aneurysmectomy, pathological studies of the infarcted tissue correlated well with the area of perfusion defects seen at the time of scanning. In another recent study, Rothbaum et al. used rubidium to evaluate moyo-
cardiac perfusion in patients subsequently undergoing saphenous vein graft surgery. Surgical mortality was significantly higher in those patients with the largest areas of impaired perfusion suggesting extensive myocardial scarring. Therefore, it would appear from these studies that a perfusion defect, particularly when seen in the presence of electrocardiographic evidence of a myocardial infarction, most likely represents fibrotic tissue.

However, perfusion defects and areas of abnormal contractility do not uniformly indicate areas of fibrosis. Hamilton described perfusion defects in several patients with preinfarction angina but without evidence of infarction.25 The same investigators in a later study were able to produce perfusion defects by injecting the isotope immediately after hyperemia induced by intracoronary contrast media.27, 28 In many of these patients, no defects were noted in the resting scan. We have recently reported that perfusion defects in patients with unstable angina may show improved perfusion and left ventricular wall motion following coronary artery bypass surgery.29 These studies with stress scanning and in patients with severe angina suggest that not all perfusion defects are static or permanent, but in some individuals may represent areas of relative ischemia.

Similarly, contraction abnormalities detected by the echocardiogram may not always be the result of a myocardial infarction. Abnormal motion of the posterior left ventricular free wall has been recorded during episodes of angina.30 In a recent report regarding a patient with Prinzmetal angina AESM was recorded only during an episode of chest pain and marked ST-segment elevation.31 In our study we feel that abnormal septal motion and the presence of perfusion defects in patients with chronic disease most likely do represent infarction of the septum particularly when the septum is thin. While we feel that most septal perfusion defects signify infarction of the septum, reversibility of such perfusion defects has not been tested. Such information might be provided by studying changes in perfusion following nitroglycerin or coronary bypass surgery. A more complete understanding of the relationships between perfusion defects and contraction abnormalities in critical stenosis of a coronary artery will hopefully lead to better prognostication and selection of appropriate therapy in patients with coronary artery disease.

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