Determinants of Reversible Asynergy

Effect of Pathologic Q Waves, Coronary Collaterals, and Anatomic Location

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SUMMARY
To determine which factors may be of value in determining whether or not asynergic zones have residual contractile ability, the responsiveness of these zones to sublingual nitroglycerin (1/150 grs) was studied angiographically in 36 patients. The responsiveness of asynergy was correlated with the presence or absence of pathologic Q waves and coronary collaterals in the corresponding zones, as well as with anatomic location. Of the 25 asynergic segments which had corresponding pathologic Q waves, 11 (44.0%) responded to nitroglycerin while 14 (56%) remained unresponsive. In contrast, 30 (83.3%) of the 36 segments which did not have associated Q waves improved while only 6 (16.7%) did not (P < 0.005). Akinetic segments with Q waves were associated with a significant decrease in responsiveness (P < 0.02) compared to hypokinetic segments. Of the 26 segments with angiographically demonstrable collaterals, 22 (84.6%) improved and only 4 (15.4%) remained unchanged (P < 0.02). In contrast, of the 25 segments without collaterals, 19 (54.3%) were responsive and 16 (45.7%) did not respond. Seven (77.8%) of the 9 akinetic segments with collaterals exhibited improvement compared to only 5 (33%) of the 15 segments without collaterals (P < 0.05). In segments with pathologic Q waves, 70% of those associated with collaterals improved compared to only 27% without collaterals (P < 0.02). Relative to anatomic location, of 29 anterior wall segments, 24 (82.8%) responded compared to only 11 (45.8%) of 24 apical segments (P < 0.005). These data indicate that the presence of coronary collaterals and absence of pathologic Q waves in the corresponding zones are associated with a higher incidence of residual contractile ability of asynergic segments. Apical asynergy responds less frequently than asynergy in other anatomic zones.

Additional Indexing Words:
Nitroglycerin
Myocardial viability
Left ventriculogram
Myocardial ischemia
Ventricular aneurysm

Although aortocoronary bypass surgery is being increasingly utilized for patients with coronary heart disease and angina pectoris, its effect on left ventricular function has remained controversial.1-4 The ability of this procedure to improve left ventricular function depends in large part on the potential for improvement in zones of asynergy.5 There has been, therefore, mounting interest in developing a method which will allow an assessment of residual contractile ability of asynergic zones.6-9 Nitroglycerin has been found to unmask residual contractile ability of asynergic zones as depicted ventriculogramically, presumably by favorably altering the balance between oxygen demand and supply.6, 8, 10-12 This method has been utilized in the present report to allow an evaluation of the factors which may be of value in determining the ability of asynergic zones to improve their contraction pattern.

Material and Methods
Studies were performed in 36 patients undergoing cardiac catheterization for evaluation of coronary heart disease. Criteria for admission to the study were: 1) asynergy on ventriculography (defined as a localized abnormality of left ventricular contraction), 2) significant (≥75% decrease in diameter) obstruction of one or more of the three major coronary arteries (left anterior descending, right and circumflex arteries), 3) absence by catheterization of other etiologic heart disease. All patients were postabsorptive and premedicated with 50 mg nembutal, 50 mg demerol and 0.4 mg atropine.

Right heart catheterization was performed via an antecubital vein cutdown and left heart catheterization either percutaneously through a femoral artery or via a right brachial arteriotomy. Following recording of left ventricular
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pressure (using Statham P23 dB transducers) and cardiac output (dye dilution method using indocyanine green), left ventriculography (control ventriculogram) was performed in 45° right anterior oblique projection using 30 to 40 cc of meglumine diatrizoate (Renograin-76) injected into the left ventricle. In no case was this performed during or immediately after anginal symptoms. When asynergy was observed, nitroglycerin (gr 1/150 sublingual) was administered 15–20 min following the initial ventriculogram. When the characteristic hemodynamic effects of nitroglycerin were observed (fall in systolic and end-diastolic pressure and increase in heart rate), the ventriculogram (TNG ventriculogram) was repeated in the same degree of obliquity, using the same amount of contrast material and tube to table top distance. Selective cine coronary arteriography was then performed by either the Judkins or Sones technique. Cines were taken on a 10 x 6 inch dual-field image intensifier (Siemens) at 64 frames/sec using 35 mm Kodak Shellburst film. Hemodynamics were monitored and recorded on an Electronics for Medicine oscillographic recorder.

Ventriculograms were analyzed with respect to location and severity of asynergy. Location was determined according to the anatomic areas of the left ventricle perfused by each of the three major coronary arteries. The anterior wall and apical zone was defined as the "left anterior descending segment"; the portion of the inferior wall between the mitral valve and posterior papillary muscle was considered the "right coronary segment"; and the inferior wall between the posterior papillary muscle and the apex was taken to be a representative portion of the "circumflex segment." The severity of the contraction abnormality of each segment was defined as follows: hypokinetic indicated diminished contraction, akinetic referred to absence of contraction and dyskinetic to paradoxical systolic expansion.

A quantitative analysis was performed by superimposing tracings of end-diastolic and end-systolic frames using the cardiac apex to mid-aortic valve as fixed points. Hemiaxes were drawn which bisected the long axis at right angles to it. Each hemiaxis was measured and recorded as a percentage change from end-diastole to ascertain the amount of regional contractile motion. Apical motion was calculated on the basis of percent change of the apex to base axis. An asynergic segment was considered to have responded following nitroglycerin when it either normalized or changed to a lesser degree of severity, e.g., a dyskinetic segment becoming akinetic, an akinetic segment becoming hypokinetic, etc. To ascertain whether the apparent improvement of asynergy was not related primarily to a decrease in wall stress in the asynergic area, hemodynamic and ventricular volume data before and after administration of nitroglycerin were examined with respect to their relationship to the responsiveness of the asynergic area. Left ventricular volumes were determined by the single plane method of Sandler and Dodge.14

Angiographic criteria for the presence of coronary collateral vessels20 were as follows: direct visualization of accessory blood vessels either filling the distal segment of an occluded or severely stenotic artery, or subserving the area of myocardium that would ordinarily be supplied by the severely stenotic vessel; and visualization of a coronary artery after injection of contrast material into the contralateral vessel. Collaterals were further classified as "threatened" or "unthreatened" depending on whether the parent vessel exhibited a significant (≥75%) stenosis proximal to the origin of the collateral channel.

Twelve lead electrocardiograms, performed routinely on all patients, were analyzed for the presence of pathologic Q waves prior to cardiac catheterization by a staff cardiologist without knowledge of the patient. Q waves were considered pathologic when they were of ≥0.04 sec duration.18 The presence of a Q wave in lead III unaccompanied by a similar Q in aVS was not considered pathologic. Q waves in leads II, III and aVF were considered to correspond to an asynergic right coronary or circumflex segment while Q waves in leads I, aVL, V7-V8 were considered to correspond anatomically to the left anterior descending segment.17 Statistical data were derived using the Chi-square (χ²) method.

Results

Of the 36 patients studied, 20 had a history of myocardial infarction. Pathologic Q waves were also demonstrated on the electrocardiogram of 20 patients, 8 representing anterior wall infarction, 10 inferior wall and 2 both anterior and inferior wall.

There were 61 asynergic segments of which 36 were hypokinetic, 25 akinetic and one dyskinetic. Quantitative systolic wall motion analysis (percent hemiaxis shortening) with respect to the responsiveness or unresponsiveness of the asynergic zones to nitroglycerin is shown in table 1. These measurements were based on single plane (right anterior oblique view) ventriculograms. The qualitative analysis correlated closely with the quantitative analysis.

<table>
<thead>
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<th>Table 1</th>
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<tr>
<td>Effect of Nitroglycerin (TNG) on Systolic Wall Motion*</td>
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<tr>
<td>Control</td>
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<td>Unresponsive</td>
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*Numbers indicate % hemiaxis shortening.
†(P < 0.001).
The relationship of pathologic Q waves (≥ 0.04 sec) to improvement in asynergy of corresponding zones. Asynergic zones associated with Q waves respond less frequently than those without Q waves (P < 0.005). Responsive = improvement of asynergy during the effect of nitroglycerin. Unresponsive = no improvement in asynergy during nitroglycerin effect.

Pathologic Q Waves

Figure 1 demonstrates the relationship of pathologic Q waves to improvement of asynergy. Of the 25 asynergic segments which had corresponding pathologic Q waves, 11 (44%) improved while the other 14 (56%) did not respond. In contrast, 30 (83.3%) of the 36 segments which did not have corresponding Q waves responded and only 6 (16.4%) did not. Of the 30 improved segments, 9 completely normalized. The difference in responsiveness of the two groups was highly significant (P < 0.005).

When the influence of Q waves was analyzed in relation to severity of asynergy, they did not appear to influence the responsiveness of hypokinetic segments (fig. 2). Of the 10 hypokinetic segments with Q waves, 7 (70%) responded, while of the 26 segments without Q waves 22 (84.6%) demonstrated improvement. In contrast, the presence of Q waves was associated with a decreased incidence of responsiveness in akinetic segments. Only 4 (28.6%) of the 14 akinetic segments with Q waves responded to nitroglycerin in contrast to 8 (80%) of the 10 akinetic segments without Q waves (P < 0.02).

Coronary Collaterals

Figure 3 demonstrates the relationship of coronary collaterals to the improvement of asynergic zones. Of the 26 segments in which collaterals were demonstrable, 22 (84.6%) were responsive and only 4 (15.4%) were unresponsive to nitroglycerin. Seven of the 22 responsive segments completely normalized. Of the 4 segments which exhibited collaterals but did not show improvement, 2 had threatened collaterals and the other two had total occlusion of the primary vessel. All 4 zones had associated pathologic Q waves. In contrast, of the 35 segments in which collaterals were not demonstrable, only 19 (54.3%) improved and 16 (45.7%) did not. The difference in improvement between segments with and without collaterals was statistically significant (P < 0.02).

When the severity of asynergy was analyzed, no difference was found in responsiveness of the hypokinetic segments with or without collaterals. Thus, 88.2% of hypokinetic zones with collaterals improved, compared to 73.7% hypokinetic zones without

![Figure 1](image1.png)

![Figure 2](image2.png)

![Figure 3](image3.png)
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Figure 4

Relationship of coronary collaterals to improvement of hypokinetic and akinetic zones. The incidence of improvement of akinetic zones with collaterals is significantly higher than those without collaterals (P < 0.05).

collaterals (fig. 4). In contrast, akinetic segments subserved by collaterals responded more frequently than those without collaterals. Seven of 9 akinetic segments exhibited improvement when collaterals were present in contrast to only 5 of the 15 segments without collaterals (P < 0.05).

Collaterals Associated with Q Waves

As shown in figure 5, of the 10 segments with Q waves which exhibited collaterals, 7 improved and only 3 were unresponsive. In contrast only 4 of the 15 segments with Q waves and without collaterals responded (P < 0.02). Conversely, when the influence of Q waves on the reversibility of asynergic segments associated with collaterals was tested, there was no significant difference between the responses of the two groups.

Anatomic Location

As indicated in figure 6, 24 (82.8%) of the 29 anterior wall segments responded to nitroglycerin and only 5 (17.2%) did not (P < 0.005). Conversely, only 11 (45.8%) of the 24 apical segments responded while 13 (54.2%) did not. The difference in responsiveness between anterior wall segments, right coronary segments and circumflex segments was not statistically significant (fig. 6).

Hemodynamic Changes

The left ventricular hemodynamic and volume changes were similar for those segments which did and did not respond to nitroglycerin (table 2).

Discussion

An asynergic zone consisting essentially of chronically ischemic but viable myocardium should logically have residual contractile ability, while an extensively infarcted area should remain noncontractile. The presence of pathologic Q waves on the surface electrocardiogram is generally recognized to be indicative of transmural infarction and correlates well with the presence of scar tissue in corresponding zones of the left ventricle at autopsy. Recent studies from our laboratory indicate that pathologic Q waves are associated with severe degrees of asynergy (i.e.,

Table 2

<table>
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<th>Mean decrease LVSP (mm Hg)</th>
<th>Mean decrease LVEDP (mm Hg)</th>
<th>Mean decrease LVEDV (cc)</th>
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<tbody>
<tr>
<td>Responsive</td>
<td>22.3 ± 1.8</td>
<td>6.6 ± 4.5</td>
<td>47.9 ± 10.4</td>
</tr>
<tr>
<td>Unresponsive</td>
<td>22.7 ± 3.6</td>
<td>4.4 ± 3.8</td>
<td>53.0 ± 15.8</td>
</tr>
</tbody>
</table>

Abbreviations: LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; LVEDV = left ventricular end-diastolic volume.
akinesis and dyskinesis) in 72% of cases. These types of asynergy have been taken as the ventriculographic equivalent of a ventricular aneurysm and are less likely to respond to an intervention which unmasks residual contractile ability than hypokinetic segments. The present study shows that the majority of asynergic zones which exhibited corresponding Q waves did not respond to nitroglycerin, whereas only one-sixth of those asynergic segments not associated with Q waves were unresponsive (fig. 1). Of the akinetic segments, 71.4% of those associated with Q waves were unresponsive while only 20% without Q waves did not change (fig. 2). Thus, the presence of pathologic Q waves adversely affects the chances of improvement of asynergy when severe asynergy exists, presumably by denoting extensiveness of zonal scar formation.

The clinical importance of the coronary collateral circulation is controversial. Previous studies have shown that presence of these collateral channels does not protect from the development of asynergy or hemodynamic abnormalities. The present study, however, indicates that collaterals may serve an important protective function in preserving myocardial viability in these asynergic segments (fig. 3). Thus, these zones retain the ability to improve their contraction pattern in response to an intervention which improves the balance between oxygen supply and demand. The response of the akinetic zones was particularly striking. While more than three-fourths of the akinetic segments with collaterals showed improvement, only one-third without collaterals responded (fig. 4). It should be pointed out, however, that these data could be explained conversely. It is possible that the presence of viable, ischemic myocardium in potentially reversible asynergic zones stimulates the development of collaterals and that this explains the association of the two. In this regard, several studies have suggested that ischemia is a major stimulus for the development of collaterals.

It was interesting to find that 70% of asynergic segments exhibiting both Q waves and collaterals improved compared to only 26.7% without collaterals (fig. 5). Therefore, in this situation a significant amount of myocardium in or more likely surrounding these areas may be viable despite the presence of pathologic Q waves in the corresponding zone. In this regard, recent experimental studies in our laboratory utilizing tension and length gauges have shown that only the border zones of an infarct exhibit improvement in contractile characteristics following nitroglycerin administration while there is no change in the central infarct zone.

The finding that the apex was less responsive than the remaining zones of left ventricle (fig. 6) is of interest. Previous postmortem studies have indicated that the most frequent site of aneurysm and scar tissue formation is in the apical segment. This zone is supplied by the terminal portion of the left anterior descending artery and is thus unique. It may be that this anatomic dependence on the anterior descending and the paucity of additional potential sources of blood flow accounts for its decreased incidence of contractile improvement.

The assessment of asynergy by single plane cineangiography has inherent limitations. The right anterior oblique projection employed in this study provides no information regarding the wall motion of the true posterior wall and the septum. Although biplane angiography would be ideal in the absence of such a facility, this would require four ventriculograms to accurately delineate the true posterior and the septal movement and would not be practical for routine clinical usage in most laboratories. The right anterior oblique ventriculogram before and after administration of nitroglycerin would adequately reflect changes in wall motion of the anterior and inferior wall. Its limitations should thus be kept in mind when assessing asynergy by single plane ventriculography.

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