The Effect of Collateral Circulation on Segmental Left Ventricular Contraction

By ROBYN J. CARROLL, MARIO S. VERANI, M.D., AND HERMAN L. FALSETTI, M.D.

SUMMARY

The anatomy of the coronary circulation was correlated with left ventricular segmental contractile motion in 162 patients: 43 with normal coronary arteriograms, 119 with significant (>75%) obstruction of the right coronary artery and/or left anterior descending coronary artery. Collateral vessels were identified in 69% of the patients with coronary artery disease (82/119). The cases with collaterals were graded poor, fair, or good according to the number of vessels and the quality of opacification. Segmental contractile motion was determined angiographically by the percent shortening of each of the six chords of the left ventricle in the right anterior oblique projection. The range of normal shortening (mean ± one standard deviation) for each chord was established in the 43 normal patients as follows:

<table>
<thead>
<tr>
<th>Anterior Wall</th>
<th>Posterior Wall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basal</td>
<td>Basal</td>
</tr>
<tr>
<td>Middle</td>
<td>Middle</td>
</tr>
<tr>
<td>Apical</td>
<td>Apical</td>
</tr>
</tbody>
</table>

Patients were grouped according to the site of coronary artery obstruction and quality of collateral circulation to the area supplied by the obstructed coronary artery. The percent shortening and number of abnormal segments were determined for each patient; there was no consistent difference in segmental contraction between patients with and without collaterals. The quality of collateral vessels did not correlate with either the number of abnormal segments or the percent of chord shortening.

In summary, the development of collateral vessels is not associated with the restoration of normal segmental wall motion in patients with coronary artery disease.

Additional Indexing Words:
Coronary atherosclerosis
Left ventriculogram
Coronary arteriography
Cardiac ischemia
Left ventricular segmental motion

collateral vessels correlates with segmental left ventricular contraction.

Method

The coronary circulation was evaluated in 162 patients, a group which included 43 patients with normal coronary arteriograms and normal left ventriculograms and 119 patients with significant (>75%) obstruction of the right coronary artery and/or left anterior descending coronary artery. The following patients were excluded from the study: patients with less than 75% obstruction, patients with a solitary lesion of the left circumflex coronary artery, and patients with atrial fibrillation. All patients included in the study had undergone diagnostic cardiac catheterization because of chest pain and/or congestive heart failure. Informed consent was obtained from all patients. Catheterization was carried out with patients under mild sedation (sodium pentobarbital, meperidine, or promethazine hydrochloride), local anesthesia, and in the fasting state. Twenty-five to forty milliliters of meglumine diatrizoate and sodium diatrizoate (Renografin-76) were injected from a catheter passed retrogradely across the aortic valve. Cineangiograms were made at 60 frames per second with the patients in the right anterior oblique position during deep inspiration. Selective coronary arteriography was performed by either the Sones' technique.
Collaterals were angiographically defined as distinct anastomotic channels between segments of the same artery (intracoronary collateral) or different arteries (intercoronary collateral). The area supplied and the direction of flow was determined angiographically: 1) by observing the filling of the vessel from its origin to the area supplied and 2) by observing that the vessels originated from a patent artery or proximal to an arterial occlusion and terminated distal to an occlusion. Collateral vessels were observed in 82 patients with coronary artery disease (69%) and graded according to the caliber and number of vessels. On independent observation of two investigators, collaterals were judged poor, fair, or good. A thread-like, poorly opacified collateral was considered "poor," a large, brightly opacified collateral was considered "good," and "fair" collaterals were those in the middle of this spectrum. If there was a difference in grading, a third opinion was obtained. The classification was based on a combination of quantity and quality as illustrated in figure 1. All patients in the "good" class had at least two good collaterals plus any number of fair or poor vessels. Patients in the "fair" group had at least two fair collaterals, any number of poor collaterals, but no more than one good collateral.

The segmental motion of the left ventricle was measured in all patients. The percent shortening was determined from an end-diastolic and end-systolic outline as illustrated in figure 2. Cardiac cycles during or immediately following premature ventricular contractions were not analyzed. The plane of the aortic valve was drawn and bisected with a long axis to the apex on both outlines. The long axis was then equally divided with three perpendicular lines, creating six chords. The difference of each chord from diastole to systole was calculated as a percent shortening.

Patients were not grouped according to lesions of the left circumflex artery. All patients with solitary lesions of the left circumflex were excluded because left anterior oblique left ventriculograms were not uniformly available to analyze the posterior lateral surface of the left ventricle. Only a small number of cases (5/119) had a dominant left circumflex with significant obstruction.

The main goal of the present study was to determine if the collateral circulation had a beneficial effect on the contractile motion of the region supplied. Patients were subgrouped according to the involvement of the coronary artery as well as the collateral circulation. These subgroups were then examined regarding the number of abnormal segments as well as mean percent segmental shortening.

**Results**

A normal range of motion for each chord was established from the 43 normal patients, using the mean

### Table 1

<table>
<thead>
<tr>
<th>Obstructed artery</th>
<th>No collaterals</th>
<th>Poor collaterals</th>
<th>Fair collaterals</th>
<th>Good collaterals</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA</td>
<td>6</td>
<td>6</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>LAD</td>
<td>12</td>
<td>6</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>RCA &amp; LAD</td>
<td>19</td>
<td>15</td>
<td>11</td>
<td>25</td>
</tr>
</tbody>
</table>

Abbreviations: RCA = right coronary artery; LAD = left anterior descending artery.
and one standard deviation. Figure 3 shows the normal left ventricular shortening. Normal ventricles had a mean of 47–51% contraction of the anterior wall segments and 39–49% contraction of the posterior wall. The widest variation was in the posterior wall, particularly the apical posterior area. The mean and standard deviation of the long axis was 24 ± 7.5% but is not shown in the figure.

Patients were grouped according to quality of collateral circulation and site of coronary artery obstruction (area supplied by collaterals) into 12 subgroups. The distribution of CAD patients with and without collateral circulation is listed in Table 1. The largest number of patients had both right coronary artery and left anterior descending artery occlusions.

The mean percent shortening of all chords in each group are illustrated in Figures 4 to 6. As can be seen in Figure 4, the posterior wall had decreased contraction in right coronary artery obstructions. The basal and apical areas contracted best in the patients with fair collaterals, and worst in patients with poor, good, or no collaterals.

Figure 5 shows mean percent coronary artery shortening in patients with left anterior descending obstruction. Patients with good collateral circulation had the highest contraction in the basal-anterior wall and the lowest contraction in the apical-anterior wall.

Figure 6 shows patients with both right coronary artery and left anterior descending coronary artery obstructions. Those with poor collaterals showed the most shortening in the entire anterior wall, and the good collateral group showed the least anterior wall contraction.
shortening. There was little difference among groups regarding the posterior wall.

Patient groups were statistically analyzed by both univariate and multivariate analysis of variance techniques. The results from the univariate analysis are summarized in table 2. As can be seen, patients with isolated right coronary artery disease had statistically significant ($P < 0.05$) decreased shortening of the $R_4$ segment. There was no difference between patients with and without collaterals, nor any difference among patient groups. The patients with isolated disease of the left anterior descending coronary artery had decreased shortening of the anterior segments $R_1 - R_3$. Again there was no statistically significant difference among patient groups. Patients with obstructive disease of both the right and left coronary artery had decreased shortening of all segments; there was no significant difference with or without collaterals.

Shortening of each left ventricular chord was regarded as abnormal when motion was 2% less than one standard deviation from the normal mean. Using this criterion, 101 of the 119 patients (85%) had at least one abnormally contracting segment. It should be noted that no patients with large anatomic ventricular aneurysms were included in this study. Only five patients had paradoxical motion of one segment. The mean number of abnormal chords of the left ventricular wall supplied by collaterals in all patients is listed in table 3. There was no significant difference among patients without collaterals and the three groups of patients with graded collaterals.

### Table 2

<table>
<thead>
<tr>
<th>Statistical Analysis</th>
<th>N</th>
<th>$R_1$</th>
<th>$R_2$</th>
<th>$R_3$</th>
<th>$R_4$</th>
<th>$R_5$</th>
<th>$R_6$</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCA – all patients</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>0.001</td>
<td>0.027</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + no collateral</td>
<td>6</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>0.004</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + poor collateral</td>
<td>6</td>
<td>NS</td>
<td>0.011</td>
<td>NS</td>
<td>0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + fair collateral</td>
<td>3</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + good collateral</td>
<td>8</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>0.001</td>
<td>0.020</td>
<td>NS</td>
</tr>
<tr>
<td>LAD – all patients</td>
<td>0.037</td>
<td>0.004</td>
<td>0.006</td>
<td>NS</td>
<td>NS</td>
<td>0.042</td>
<td></td>
</tr>
<tr>
<td>LAD + no collateral</td>
<td>12</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LAD + poor collateral</td>
<td>6</td>
<td>0.009</td>
<td>0.002</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LAD + fair collateral</td>
<td>5</td>
<td>NS</td>
<td>0.039</td>
<td>NS</td>
<td>0.006</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LAD + good collateral</td>
<td>3</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + LAD – all patients</td>
<td>0.001</td>
<td>0.017</td>
<td>0.002</td>
<td>0.001</td>
<td>0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + LAD + no collateral</td>
<td>19</td>
<td>0.044</td>
<td>NS</td>
<td>0.022</td>
<td>0.009</td>
<td>0.049</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + LAD + poor collateral</td>
<td>15</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>0.001</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + LAD + fair collateral</td>
<td>11</td>
<td>NS</td>
<td>0.032</td>
<td>0.006</td>
<td>NS</td>
<td>0.005</td>
<td>NS</td>
</tr>
<tr>
<td>RCA + LAD + good collateral</td>
<td>25</td>
<td>0.001</td>
<td>0.004</td>
<td>0.001</td>
<td>0.001</td>
<td>0.013</td>
<td>NS</td>
</tr>
</tbody>
</table>

Numbers denote $P$ values derived from univariate analysis of normal and the 12 patient subgroups. NS = not significant. $N =$ number of patients; RCA = right coronary artery; LAD = left anterior descending artery; $R_1$–$R_6$ refers to chords shown in figure 2.

### Discussion

Various methods of measuring segmental left ventricular contraction have been established. In those studies, the range of normal segmental motion was defined for 8 to 13 patients. The present study reports the range of segmental wall motion in 43 normal patients and is consistent with normal values reported by the above investigators.

When viewing a large number of arteriograms, a striking difference is seen in the opacification of collaterals. These vessels may range from a scant, thread-like collateral to a wide, well-defined collateral. Also, the number of channels may vary greatly among patients. One of the goals of this study was to classify patients on the basis of the size and the number of collaterals.

Collaterals were graded visually, as were arterial occlusions, and the limitations of visual impression have been considered. In this institution, medications

### Table 3

| Average Number of Abnormal Left Ventricular Chords Supplied by Collaterals in Each Patient |
|-----------------------------------------------|-------------------------------|-------------------------------|-------------------------------|-------------------------------|
| Obstructed artery                            | No collaterals | Poor collaterals | Fair collaterals | Good collaterals |
| RCA                                           | 1.3            | 1.3             | 0.7             | 1.7             |
| LAD                                           | 1.0            | 1.7             | 1.2             | 0.7             |
| RCA & LAD                                      | 2.2            | 2.1             | 2.4             | 2.4             |

Abbreviations: RCA = right coronary artery; LAD = left anterior descending artery.
are discontinued 48 hours before catheterization and nitroglycerine is administered routinely prior to arteriograms. Therefore, we believe left ventricular contractile motion was not effected by medication. Also, any effects of nitroglycerine on collateral visualization would apply to all patients studied. However, there may be unavoidable limitations in comparing a large number of angiograms such as the amount of contrast material injected or the quality of films. Consequently, there may be slight overlap between the patient groups. But it should be noted that all patients in the “good” group had at least two good collaterals, and all the patients in the “fair” group had at least two fair collaterals as indicated in figure 1. Therefore, rather than group a patient with one fair collateral with a patient having four fair collaterals, we attempted a more reasonable grouping by collateral caliber and number. In addition, only patients with severe coronary artery disease (> 75%) were selected for this study. This has been done in an attempt to match the severity of the coronary artery disease in patients with and without collaterals.

In regard to the mean percent shortening of the left ventricle, there was no statistically significant difference proportional to collateral quality (poor, fair, or good). In other words, contractile motion was not best in patients with good collateral circulation and worst in those with poor or no collateral circulation; nor was an inverse relationship found. Although collateral vessels were sometimes associated with normal segmental motion (figs. 3 to 5), in the majority of patients there was no demonstrable improvement in the segmental wall motion. Although collateral circulation may limit the amount of tissue necrosis or preserve other aspects of myocardial metabolism, it is our conclusion that the development of collateral circulation does not restore normal segmental wall motion.

Acknowledgment

The authors wish to acknowledge the technical assistance of James Cramer and the biostatistical assistance of Larry Linn, Biostatistics Section, Department of Preventive Medicine and Environmental Health.

References

The Effect of Collateral Circulation on Segmental Left Ventricular Contraction
ROBYN J. CARROLL, MARIO S. VERANI and HERMAN L. FALSETTI

Circulation. 1974;50:709-713
doi: 10.1161/01.CIR.50.4.709

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

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/50/4/709

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/