Pathways and Functional Significance of the Coronary Collateral Circulation

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SUMMARY
Two hundred coronary arteriograms and left ventriculograms of patients having significant coronary artery disease were reviewed. Collateral circulation was seen only when the degree of arterial narrowing exceeded 90%. Ten different collateral pathways were noted in patients having right coronary obstruction; seven different pathways were noted in left anterior descending obstruction; five different pathways were noted in left circumflex obstruction.

The role of collateral circulation in preserving myocardial function was assessed by comparing regional left ventricular contractility in 86 instances of total arterial occlusion and adequate collateral circulation with that in 80 instances of total arterial occlusion and inadequate collateral circulation. Among the group with adequate collaterals, regional left ventricular contraction was normal in 43%, mildly impaired (hypokinetic) in 52% and severely impaired (akinetetic or dyskinetic) in only 5%. Among the group lacking adequate collaterals, regional contraction was normal in 11%, mildly impaired in 16% and severely impaired in 73%. Chi square analysis reveals that the difference between the two groups is significant at the .01 level.

These data indicate that collateral circulation plays an important role in preserving myocardial contractility in patients with coronary artery disease.

Additional Indexing Words:
Angiography Coronary artery disease Ventricular wall motion
Atherosclerotic heart disease Ischemic heart disease Ventricular function
Coronary arteriography Ventriculography

THE ADVENT OF BYPASS SURGERY has necessitated a more thorough understanding of the coronary collateral circulation on the part of all physicians concerned with coronary artery disease (CAD). It has been shown by several groups of investigators that the likelihood of a successful bypass is considerably enhanced if good flow is demonstrated angiographically in the distal segment of an obstructed coronary artery as a result of antegrade filling or collateral circulation or both.

Current concepts of the process by which collateral circulation develops are based largely upon careful anatomic studies, such as those of James and Baroldi and Scomazzoni.6 The normal human heart has been found to contain a profusion of small, interconnecting anastomotic vessels (IAVs) which are generally less than 200 micra in diameter and which serve as the precursors of the collateral circulation. Baroldi and Scomazzoni have classified these IAVs as “homocoronary” anastomoses if they connect segments of the same artery and “intercoronary” anastomoses if they connect branches of different arteries. Although the IAVs can be identified in autopsy specimens, they cannot be demonstrated angiographically when normal or only mildly impaired circulation is present because they carry only minimal flow and their small size is beyond the limits of resolution of presently available imaging systems for coronary arteriography. If obstruction of a major artery occurs, however, a transanastomotic pressure gradient is created in those IAVs connecting the distal segment of the involved artery with either the proximal segment of the same artery or nearby segments of other arteries. Because of this gradient, an increased volume of blood is propelled through the IAVs, which progressively dilate and eventually become visible angiographically as collateral channels. The development of collateral circulation is therefore not the result of formation of new vessels, but rather of the utilization of vessels which are extant but nonfunctional until the need arises. The most potent stimulus to this process is the presence of coronary artery con-
striction\textsuperscript{7,8} and the resultant transanastomotic pressure gradient, but other factors undoubtedly also have an effect. These include the size and state of the distal segmental lumen, coronary vascular resistance, blood viscosity, myocardial contractility, and physical activity of the subject.\textsuperscript{9}

The purpose of this study is twofold; first, to define the specific pathways of the coronary collateral circulation, and second, to determine the effect of this collateral circulation upon left ventricular myocardial function.

Method

Coronary arteriograms and left ventriculograms were analyzed in 200 consecutive patients having significant CAD without valvular disease. All had at least 75\% stenosis of at least one major vessel. Coronary arteriography was performed by the percutaneous transfemoral approach originally described by Judkins.\textsuperscript{10} Both 35 mm cine filming and rapid serial radiographs of the coronary arteries were obtained in the right and left anterior oblique positions. Cine left ventriculography was routinely performed in the resting state in the right anterior oblique position.

The various collateral pathways were noted and tabulated. The effect of these collaterals upon myocardial

\[ \text{LAD} \rightarrow \text{RCp/OM} \]

\[ \text{RC} \rightarrow \text{PD} \]

\[ \text{RAO-LC} \text{ Injection (28)} \]

\[ \text{LAO-LC} \text{ Injection (24)} \]

\[ \text{LAO-LC} \text{ Injection (17)} \]

\[ \text{Conus} \]

\[ \text{RC} \rightarrow \text{AM} \]

\[ \text{PD} \]

\[ \text{AM} \]

\[ \text{Kugel's} \]

\[ \text{RC} \rightarrow \text{AV} \]

\[ \text{PLV} \]

\[ \text{PD} \]

\[ \text{PLV} \]

\[ \text{RC} \rightarrow \text{PD} \]

\[ \text{LAO-RC} \text{ Injection (9)} \]

\[ \text{LAO-RC} \text{ Injection (9)} \]

\[ \text{RAO-LC} \text{ Injection (9)} \]

\[ \text{LAO-LC} \text{ Injection (9)} \]

\[ \text{LAO-RC} \text{ Injection (6)} \]

\[ \text{LAO-RC} \text{ Injection (6)} \]

\[ \text{LAO-RC} \text{ Injection (2)} \]

\[ \text{LAO-RC} \text{ Injection (2)} \]

Figure 1

Collateral pathways in right coronary artery (RC) obstruction. RAO = right anterior oblique projection; LAO = left anterior oblique projection; LC = left coronary artery; AM = acute marginal branch of the right coronary artery; PD = posterior descending branch of the right coronary artery; PLV = posterior left ventricular branch of the right coronary artery; A-V = artery to the atroventricular node; LAD = left anterior descending artery; C = circumflex artery; OM = obtuse marginal branch of the circumflex artery. Numbers in parenthesis signify frequency with which the given pathway occurred in this series.

Circulation, Volume 50, October 1974
contractility was studied in 166 instances in which major coronary arteries were completely occluded and therefore entirely dependent upon collateral circulation to provide “runoff” flow in their distal segments. The size and degree of opacification of the distal segments, as seen angiographically, was taken as an indication of the adequacy of collateral circulation. It was considered adequate if the average caliber of the distal segment measured greater than 1.0 mm in diameter after correction for angiographic magnification. Where inflow to the distal segment was absent or so reduced that its average caliber was only 1.0 mm or less, collateral circulation was considered inadequate. By this criterion, collateral circulation was adequate in 86 instances and inadequate in the other 80.

The performance of the various regions of the left ventricle was evaluated by studying contractility of the chamber as seen at ventriculography. Regional function was considered normal, according to the criteria of Herman et al., if there was uniform, forceful and nearly concentric contraction of the endocardial surface. It was considered mildly impaired if hypokinesia was present, and severely impaired if akinesia or dyskinesia occurred. Regional contractility of the affected myocardial segments in the groups with adequate and inadequate collateral circulation was then compared.

The vast majority of the 166 arteries under consideration in this section of the study were left anterior descending (LAD) and right coronary (RC) arteries. These arteries generally supply the anterior and diaphragmatic aspects of the left ventricle, which are the areas best demonstrated by ventriculography in the right anterior oblique projection. The left circumflex generally supplies only the posterolateral aspect of the ventricle, an area not well visualized in this projection. Therefore, a number of left circumflex lesions were excluded from consideration. These were included only when the circumflex artery was dominant and supplied a large portion of the diaphragmatic aspect of the ventricle.

**Collateral Pathways**

There was no case in the entire series in which collateral circulation was seen at angiography when the degree of arterial narrowing was less than 90% of the lumen diameter. This bears out similar observations by previous authors. Although the development of collateral circulation is obviously a progressive phenomenon, it thus seems that narrowing of a lesser degree does not generate sufficient flow through the IAVs to render them visible radiographically.

A number of the pathways which were visualized have not been described in previous papers dealing with this subject. Among the 200 patients, 105 had severe narrowing (greater than 90%) or complete obstruction of the RC artery. Collateral circulation was visualized in 74 of these patients. Ten specific pathways were observed, as listed below and shown in correspondingly lettered diagrams in figure 1.

A. **LAD artery to posterior descending branch of the RC artery via the ventricular septal branches (28 cases).**

B. **Distal circumflex artery to distal RC artery (24 cases).**

C. **Obtuse marginal branch of the circumflex artery to posterior left ventricular branch of RC artery (17 cases).**

D. **Proximal acute marginal or conus branch of the RC artery to a more distal acute marginal branch (9 cases).**

E. **Kugel’s artery (9 cases).** This artery passes from either the proximal right or left coronary artery down along the anterior margin of the atrial septum to anastomose with the A-V node branch of the distal RC artery.

F. **Distal LAD artery around the cardiac apex to posterior descending branch of the RC artery (9 cases).**

G. **Distal circumflex artery or its left atrial circumflex branch to A-V node branch of the RC artery (6 cases).**

H. **Acute marginal branch of the RC artery to posterior descending branch of the RC artery via the diaphragmatic surface of the right ventricle (6 cases).**

I. **SA node branch of the RC artery around the lateral wall of the left atrium to left atrial circumflex branch, then to distal RC artery (2 cases).**

J. **Right ventricular branch of the LAD artery to acute marginal branch of the RC artery (2 cases).**

One hundred twenty seven patients had severe narrowing or complete obstruction of the LAD artery and 70 of them had demonstrable collaterals. Seven different pathways were noted as listed below and shown in correspondingly lettered diagrams in figure 2.

A. **Acute marginal branch of the RC artery to LAD artery (28 cases).**

B. **Proximal ventricular septal branch of the LAD artery to a more distal septal branch (27 cases).**

C. **Obtuse marginal branch of the circumflex artery to LAD artery (17 cases).**

D. **Conus branch of the RC artery to LAD artery (15 cases).**

E. **Diagonal branch of the LAD artery to distal LAD artery (6 cases).**

F. **Posterior descending branch of the RC artery around the cardiac apex to LAD artery (3 cases).**

G. **Posterior descending branch of the RC artery to LAD artery via the ventricular septal branches (3 cases).**

Severe stenosis or complete obstruction occurred much less commonly in the left circumflex artery than in the other two major vessels. This artery was involved in only 48 patients, of whom 21 demonstrated
collateral formation. The five pathways are listed below and shown in correspondingly lettered diagrams in figure 3.

A. Left atrial circumflex branch to distal circumflex artery (7 cases).
B. Proximal obtuse marginal branch of the circumflex artery to a more distal obtuse marginal branch (6 cases).
C. Diagonal branch of the LAD artery to obtuse marginal branch of the circumflex artery (5 cases).
D. Distal RC artery to distal circumflex artery (2 cases).
E. Posterior left ventricular branch of the RC artery to obtuse marginal branch of the circumflex artery (2 cases).

Figure 3

In addition to the homocoronary and intercoronary pathways discussed above, a network of very small and short channels is frequently seen bridging a localized obstruction of a major coronary artery. James'5 feels that these do not represent IAVs but rather are enlarged vasa vasorum or adventitial arteries which form a multi-channel cuff around the obstructed segment.

Functional Significance of Collateral Circulation

Ventriculographic analysis of contractility of the myocardial segments perfused by the 166 totally
obstructed arteries revealed that in 62 cases (37%), severe impairment of contractility was noted, as manifested by segmental areas of akinesia or dyskinesia. In 58 (35%), there was a mild degree of impairment, manifested by segmental areas of hypokinesia. In 46 (28%), regional contractility was entirely normal. Since regional left ventricular dysfunction is generally assumed to be the result of coronary artery obstruction, the question is thus posed as to why perfectly normal contractility was found nearly as frequently as severe dysfunction in the distribution of these obstructed vessels.

When these patients were subdivided into groups having adequate or inadequate collateral circulation and runoff, the difference in associated regional left ventricular contractility was striking, as shown in figure 4. It was found that among 86 obstructed arteries with adequate collateral circulation, regional left ventricular contraction was normal in 37 cases (43%) and hypokinetic in 45 (52%). In only 4 instances (5%) were akinesia or dyskinesia seen. On the other hand, among 80 obstructed arteries lacking adequate collateral circulation, normal regional contractility was present in only 9 (11%) and hypokinesia in 13 (16%). The vast majority, 58 cases or 73%, demonstrated akinesia or dyskinesia of the corresponding myocardial segments. Using Chi-square analysis, the difference between the two groups is significant at the .01 level.

**Discussion**

This study indicates that collateral circulation plays a major role in preserving myocardial function in

![Figure 3](http://circ.ahajournals.org/)

Collateral pathways in circumflex artery obstruction. Abbreviations as in figure 1.

![Figure 4](http://circ.ahajournals.org/)

Incidence of normal contractility, mildly impaired contractility (hypokinesia) and severely impaired contractility (akinesia — dyskinesia) in regions of the left ventricle supplied by totally obstructed coronary arteries. The set of bars on the left reflects the regional contractility patterns of the entire group of 166. The other two sets reflect the same data with the original 166 divided according to whether collateral circulation and distal runoff were adequate or inadequate. Note that where collateralization and runoff were adequate, regional contractility was normal or mildly impaired in most instances. Where collateralization and runoff were inadequate, regional contractility was severely impaired in most instances.

*Circulation, Volume 50, October 1974*
patients with coronary artery disease, a view which has been substantiated in material presented recently by several other groups. These findings are at variance, however, with an angiographic study by Helfant, Kemp, and Gorlin who compared ventricular contractility in patients having single or multiple stenoses greater than 75% without collateral circulation with ventricular contractility in patients having single or multiple stenoses greater than 75% with collateral circulation. These investigators found that contraction abnormalities were slightly more prevalent among the group with collaterals and concluded, therefore, that collateral circulation did not protect against development of an abnormal left ventricular contraction pattern.

There appear to be two principal reasons why their findings tend to underestimate the importance of collateral circulation. First, the mere presence of visible collateral vessels does not indicate whether these vessels are effective or not. To properly assess the role of collateral circulation, it is necessary to differentiate those cases of effective collateralization from those where it may be present but ineffective. Such a differentiation was not made in their study. To be effective, collaterals must provide enough inflow to the distal segment of an obstructed artery to produce reconstitution of a nearly normal caliber vessel and perfusion of the smaller branches within its distribution. If the distal segment of the obstructed artery remains unopacified or shows only minimal flow, its collateral supply is ineffective. The present study differentiates adequate or effective collateral circulation which provides good distal runoff from inadequate or ineffective collateral circulation which provides little or no distal runoff.

Second, patients with stenoses of greater than 75% do not constitute a homogeneous population, as has been assumed by Helfant, Kemp and Gorlin. Although stenotic lesions of between 75% and 90% undoubtedly can produce myocardial ischemia and anginal pain, they are of only moderate degree from a hemodynamic viewpoint since they are virtually never accompanied by angiographically visible collateral circulation. To stimulate development of angiographically visible collaterals, a more severe narrowing (greater than 90%) or complete obstruction is necessary. Thus, although patients were included in their study whenever at least a 75% stenosis was present, it is apparent that the group with collaterals consisted exclusively of individuals with severe stenoses (greater than 90%) or complete obstructions. The group without collaterals would contain some individuals with severe stenoses or complete obstructions, but would also contain all patients having only moderate stenoses (75-90%). As a group, therefore, the patients with collaterals had more advanced CAD and might logically be expected to have a higher incidence of left ventricular contraction abnormalities. The study reported herein concentrated on a more homogeneous group of vascular lesions — those producing total occlusion.

Although the present study and recent work of other groups have indicated that effective collateral circulation helps preserve myocardial function in patients with CAD, several additional aspects may require further clarification.

First, it must be determined whether the existence of a well-developed collateral circulation can prevent future infarction or death. Helfant, Vokonas and Gorlin have investigated this point and concluded that it does not provide such protection. On the other hand, Franklin has stated that patients with adequate collateral circulation emanating from non-stenotic arteries have a significantly better prognosis than patients with poorly developed collaterals or collaterals which are jeopardized by lesions in the donor vessels. A recent study by Webster, Moberg, and Rincon has confirmed that an improved long term prognosis is associated with good collateralization in patients with angiographically documented single and double vessel CAD. Further investigation of a prospective nature is advisable to clarify this important matter. Systems currently being used to grade the severity of CAD do not take into account the adequacy of collateral circulation and probably should be revised to do so.

The surgical significance of collateral circulation likewise has not yet been properly evaluated. On the one hand, by preserving both left ventricular contractility and patency of the distal segments of obstructed arteries, collateral circulation maintains conditions which are favorable for the construction of successful coronary bypass grafts. On the other hand, there may be cases in which the auto-revascularization of collateral circulation is sufficient to obviate the necessity of surgical revascularization. Once formed, collaterals may well be less likely to occlude spontaneously than a saphenous vein bypass. Postoperative angiography has shown that in some cases coronary bypass may lead to loss of previously well developed collaterals or that thrombosis of a vein graft may propagate and obstruct the collaterals, leaving the patient with a much more severe perfusion deficit than had existed previously.

**Acknowledgment**

A number of the diagrams in figures 1-3 are reproduced with permission from Levin DC et al.: Am J Roentgenol Rad Ther Nuc Med 119: 469, 1973.
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