IT HAS LONG BEEN KNOWN that collateral blood vessels which are rudimentary in the normal heart develop with increasing frequency and size in the presence of coronary disease. During recent years a controversy has arisen concerning the effectiveness of these vessels as a compensatory mechanism for the loss of blood flow consequent on coronary obstruction. Both sides of this debate rest on observations of electrical or mechanical function of the left ventricle in patients with coronary disease. Some workers fail to observe any differences in function whether collaterals are present or not in patients with apparently equal degrees of coronary obstruction. This leads to the conclusion, implicit or stated, that the collaterals are functionless. 1-4 Others, in contrast, find that function may be substantially improved when collaterals have developed. 7-10

The direct evidence that collateral vessels can largely compensate for coronary obstruction seems so overwhelming that it would appear that the "problem" must be artificial. In the dog a main coronary branch may be slowly occluded without damage to the distal muscle. Within days flow is rapidly restored to the muscle mass previously supplied by the obstructed vessel through collateral channels. 11, 12 Likewise, in man one may observe a comparable situation as a consequence of coronary disease, namely a complete obstruction of a major coronary artery without gross infarction of the distal muscle, even though some evidence of coronary insufficiency may be elicited by stress. Thus it seems beyond debate that collateral vessels may frequently function so as to compensate, partially or completely, for the loss of normal flow. Why then have such apparently contradictory results been obtained in human angiographic studies?

First, there is a question of experimental design. Ideally, if in a single individual, collateral flow could be manipulated, a direct relationship with ventricular function might be observed. The angiographers have, of course, been forced to adopt another approach. That is, ventricular function in two groups of patients is compared, each group having an equal degree of coronary obstruction but only one with observed collateral vessels.

Whether a valid conclusion can be reached from such a study depends on several assumptions. First, it must be assumed that "equal degrees of obstruction" will cause an equivalent amount of ischemia.

Even if one could precisely measure the degree of narrowing of a coronary artery, the rise in resistance this would cause could only be guessed at. Is there one site of narrowing or several? How long is each? How comparable is the smoothness of the inner wall, the presence of turbulence and of flow vortices. To estimate the exact rise in resistance from the visual appearance of the angiograms is thus questionable.

Even if one could precisely estimate the increase of resistance in each vessel from the angiographic appearance, however, one would still not know the flow through it unless one also knew the driving pressure. In the presence of ischemia, a diastolic pressure of 90 mm Hg should produce a better coronary flow than one of 70 mm Hg for exactly equal degrees of coronary narrowing.
Furthermore, even with a knowledge of the flow one could no more than guess the degree of ischemia. One would need to know the mass of muscle supplied and its oxygen consumption. Likewise, differences in oxygen carrying capacity of the blood would influence the degree and frequency of ischemia. Thus to establish equal degrees of ischemia, in addition to the resistance of the diseased vessel one would want to know the blood pressure, the hemoglobin level and the smoking habits of the patient concerned.

There are other factors which make comparison of two groups difficult. Collateral flow must depend not only on the presence of collaterals but also on their number and size. Likewise, the fact that they can be filled with contrast does not necessarily reflect their function. Effective flow depends also on the existence of a pressure gradient. Thus there is likely to be a difference in the effectiveness of collateral flow in any particular heart depending on whether the ischemic territory is bounded by an area with or without coronary disease. Accordingly the distribution of coronary disease is probably as important as the degree of disease in determining the effectiveness of collateral vessels. Likewise, methods of estimating regional ventricular wall function are relatively crude and quantitatively inexact.

There are thus abundant reasons why the presence or absence of collateral vessels may appear not to influence the degree of muscle dysfunction in patients with apparently equal degrees of coronary obstruction. In view of the direct evidence that collateral vessels can at least partially compensate for the loss of normal coronary flow, it does not seem reasonable to infer that they may be functionless from the angiographic evidence supplied. Further attempts to establish this point by this extremely indirect approach are unlikely to be profitable.

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References

The coronary collateral circulation. A significant compensatory mechanism or a functionless quirk of nature.
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