Is There a Left Main Equivalent?

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LEFT MAIN coronary artery disease has received considerable attention in the past few years. Studies have indicated that the risk of death due to coronary artery disease correlated best with the extent of coronary artery disease and left ventricular function. The extent of coronary artery disease has been and still is generally categorized as one-, two-, three- and left main coronary artery disease. Earlier studies have found a 1-year mortality after identification at angiography of 25–33% for left main coronary artery disease, 14–19% for three-vessel disease, 7–9% for two-vessel disease, 7–8% for one-vessel disease involving the left anterior descending coronary artery, and 4–5% for one-vessel disease involving the right coronary artery or circumflex marginal arterial system.6–8 Studies of 5-year mortality have also indicated a marked difference in risk, depending on the extent of coronary artery disease. Thus, the annual mortality over a 5-year period for three-vessel disease was around 11%, that for two-vessel disease 7% and that for one-vessel disease 3%.6–11

These earlier studies were based on data obtained in the late 1960s and early 1970s. Since then, mortality from coronary artery disease has fallen in the overall population,12 medical treatment has improved, patient selection for coronary angiography has changed, and more detailed attention is given to left ventricular function. Prognosis for any given extent of coronary artery disease is greatly influenced by left ventricular function. For example, McNeer and associates found a 2-year survival in patients with three-vessel disease of 87% if they had normal left ventricular function and 76% if they had poor left ventricular function.13 Recent experience with medical therapy, while demonstrating the improved survival, still indicates that the extent of coronary artery disease has a major influence on risk. The randomized Veterans Administration (VA) Study in Stable Angina provides survival curves of males with stable angina and minimal left ventricular abnormalities who were randomized to medical therapy.14 The study showed a 4-year mortality of 35% for left main disease, 27% for three-vessel disease, 12% for two-vessel disease and 2% for one-vessel disease. The annual mortality rate over this period was about 9%, 7%, 3% and 0.5%, respectively.

Other forms of evaluation have not provided clear-cut differences in prognosis. For example, the initial clinical manifestation of ischemic heart disease does not discriminate subsequent prognosis very well. The Framingham Study revealed a 1-year mortality of 4.5% in patients whose first manifestation of coronary artery disease was angina as well as those who survived for 30 days after a myocardial infarction.15 The Health Insurance Plan of Greater New York found similar results in a separate large general population.16 Exercise electrocardiography has been extremely useful in identifying patients with ischemic heart disease. It can also add significant information concerning prognosis when combined with knowledge about the anatomic extent of coronary artery disease. For example, Platia et al. found that the 5-year mortality of patients with one-vessel left anterior descending coronary artery disease was higher in those patients with a positive exercise test than in those with a negative test.17 However, the ability of the exercise ECG alone to predict subsequent survival is modest. In a large series of over 2700 patients, Ellestad and Wan18 found a 3.3% annual mortality over 4 years in patients with a positive exercise test (defined as ST depression of 1.5 mm or more) and an annual mortality of 0.3% in patients with a negative test (defined as less than 0.5 mm ST depression), a difference far less than that yielded by coronary angiographic findings. Most studies indicate that prognosis in patients with ischemic heart disease who survive their initial clinical manifestation is better determined by the anatomic extent of coronary artery disease than by clinical or electrocardiographic criteria. This information, coupled with knowledge of left ventricular function, allows the most accurate prediction of subsequent prognosis.
The extent of coronary artery disease also provides the basis for important therapeutic considerations as has now been established by large, well-designed, prospective randomized studies that compare medical and surgical therapy. Although aspects of their study are controversial, the VA Group has provided impressive evidence that coronary artery bypass surgery is associated with significantly improved survival compared with medical therapy in male veteran patients with left main coronary artery disease, mild stable angina and mildly abnormal left ventricular function. The Cooperative European Randomized Study compared medical and surgical therapy in males younger than age 65 years with mild stable angina, an ejection fraction of greater than 50% and multivessel disease. In this study, 407 patients with three-vessel disease were randomized and the results indicate a significantly improved survival with surgical therapy compared with medical therapy at 2 years. In the VA Study, the survival curves of medical and surgical patients with three-vessel disease have crossed over so that surgical survival is better at 4 years. The differences in the VA Study are not yet significant, but the trend toward the improved surgical survival appears to be continuing. No randomized study has yet shown that survival (as opposed to relief of symptoms) is better achieved with surgical than with medical therapy in patients with two- and one-vessel disease.

Thus, the classification of the extent of coronary artery disease as one-, two-, three- and left main coronary artery disease has proved extremely useful not only in determining prognosis but also in selecting patients whose survival may be significantly enhanced by coronary artery bypass surgery. Most of our reports and the ongoing national trials use this method of classification in describing comparability of anatomy. However, the concept of one-, two-, three-vessel and left main coronary artery disease may be a bit restrictive. The coronary artery anatomy of different patients varies. A good example is the difference between dominant and nondominant right coronary artery anatomy. In about 95% of females and 85% of males the right coronary artery gives off the atrioventricular (AV) nodal artery, the posterior descending artery and a posterior left ventricular artery. In these people the circumflex artery is usually small and supplies relatively little muscle. The other 10% of the population has a dominant left system, i.e., the AV nodal artery, the posterior descending artery and the posterior left ventricular branches arise from the circumflex artery. In these persons the right coronary artery is small and supplies little muscle. The distribution of the left anterior descending coronary artery itself may vary. In some, the left anterior descending artery is a huge artery that gives off a large diagonal branch supplying part of the lateral wall, continues on to supply the anterior wall and the anterior two-thirds of the interventricular septum, and then extends over to the apex to supply the inferior surface to a variable extent. These patients usually have a relatively short, although still dominant, right coronary artery and a modest circumflex–marginal artery system. In contrast, other patients have a short left anterior descending artery that does not reach the apex and supplies relatively little muscle. These patients usually have a huge right coronary artery and an extensive circumflex–marginal arterial system. Although the amount of myocardium supplied by the left anterior descending artery obviously varies considerably in these patients, present nomenclature combines all these patients into the category of one-vessel left anterior descending artery disease.

The variations in the coronary artery anatomy make identification of comparable extent of coronary artery disease even more difficult in patients with multivessel disease. Attempts have been made to develop more discriminatory classifications of patients on the basis of the anatomic location and severity of coronary artery obstruction. Friesinger, Humphries and their colleagues at The Johns Hopkins used a scoring system that weighted the degree of obstruction found in each of the three major coronary systems, graded as 0–5, and then added the three numbers for a total score (maximum of 15). However, the relative size and distribution of individual arteries was not taken into account. In an attempt to develop a more subtle indication of the amount of myocardium put at risk, a “jeopardy score” was developed by Johnson and co-workers at the Massachusetts General Hospital. The coronary arteries were divided into six segments. Each segment was given a score of 2 if it was at ischemic risk from an obstructive lesion that caused more than 70% narrowing of the luminal area. The left anterior descending coronary artery was divided into three segments, the circumflex-marginal arteries into two segments, and the right coronary artery was considered as one segment. In patients with left coronary arterial dominance, the two points assigned to the posterior descending artery were considered in the circumflex system rather than in the right coronary arterial system. Thus, the maximum score could be 12. This system is a significant improvement in considering the total proportion of myocardium in jeopardy from all possible ischemic events, but does not identify those patients with high scores whose risk depends on a single event rather than on multiple events.

In addition to the total proportion of myocardium put at risk by all occlusive coronary artery disease, one should consider the number of ischemic events required to infarct the threatened muscle and the proportion of muscle infarcted by one occlusive event. For example, the fact that patients with left main coronary artery disease (fig. 1) have a much worse 1-year prognosis than patients with three- and two-vessel disease even though many of the latter patients may have the same percentage of live muscle at risk from all occlusive lesions may be explained by the concept that patients with left main coronary artery disease have a large proportion of muscle at risk from one occlusive event. This is especially true for patients with a dominant left system. Studies by Page and co-workers have shown that the common denominator of cardiogenic
shock after myocardial infarction is loss of 40% of cardiac muscle. Occlusion of a left main coronary artery usually puts at least that much muscle in jeopardy. To lose a comparable percentage of live muscle at one time in three-vessel disease, at least two occlusive events would have to occur simultaneously. Thus, a "left main equivalent" is probably not the simple presence of obstructive disease in the left anterior descending artery and circumflex-marginal system as has been inferred by some, but may be an anatomic situation that places a large proportion of live muscle at risk (perhaps more than 40%) from one event (fig. 2). Figure 2A shows a complete occlusion of the left anterior descending coronary artery without myocardial infarction because the distal vessel is being supplied through well-developed collaterals from the right coronary artery. However, the right coronary artery has a 95% narrowing. If the right coronary artery completely occludes, the whole inferior wall as well as the apical and anteroseptal walls would infarct, far more than would infarct if the distal left anterior descending artery were supplied through collaterals from an intact marginal artery. Yet both types of anatomy are described as simple two-vessel disease in our present nomenclature. Figure 2B shows a similar situation, but the right coronary artery is completely occluded and the muscle in its distribution is living because of flow through collaterals from the left anterior descending artery that is significantly narrowed proximally.

Even in patients with one-vessel disease, there are occasional anatomic situations that may place almost as much muscle at risk as that in main left coronary artery disease. Figure 3 shows a patient who has a partial proximal obstruction of a large left anterior descending coronary artery, which gives off a big diagonal branch and then extends over the apex to supply a major part of the inferior wall. In contrast to the examples in which a large percentage of myocardium is put in jeopardy by one lesion, the anatomy shown in figure 4 depicts two- and three-vessel disease with each narrowed artery jeopardizing only its own area of muscle. The arteries are without collateral connections between each other and are therefore independent of each other.

The role of collaterals is obviously very important in this concept. Well-developed collaterals may protect an area of myocardium from infarction, although the vessel normally supplying it is completely occluded if blood flow from the second coronary artery connected by the collaterals is unimpeded. The second artery, by the same token, would then be supplying a large proportion of myocardium, sometimes equivalent to the area supplied by the main left coronary artery.
Figure 3. One-vessel disease with a large proportion of live myocardium in jeopardy by a single stenotic lesion because of anatomic variation of size and distribution of the left anterior descending coronary artery. See text for discussion. LCA = left coronary artery; Diag. = diagonal artery; Cx = circumflex coronary artery; RCA = right coronary artery; LAD = left anterior descending coronary artery.

Although the concept of one-, two-, three-vessel and left main coronary artery disease is still pervasive, many clinicians faced with decisions of medical or surgical therapy in patients give considerable weight to the proportion of live myocardium put at risk from one occlusive event. Myocardial segments that contract normally or are hypokinetic are considered to be alive; those that are dyskinetic, dead; and those that are akinetic, probably dead. Although the concept of the proportion of live myocardium put at risk from one event seems reasonable, its validity has not been proved. Careful investigations must be performed to identify patients with this anatomic subset and to determine whether their prognosis is worse than that of patients with a comparable number of vessels diseased but with far less live myocardium at risk from any one event. In addition, the relative value of medical and surgical therapy will have to be assessed in these subgroups. If this concept is far more discriminant than that of simple one-, two- and three-vessel disease, a more sensitive tool for determining prognosis may evolve. Also, better selection can be made of comparable patients for medical and surgical trials, and significant advances might be made relative to the proper selection of patients whose survival may be enhanced with coronary artery bypass surgery.

The ongoing national cooperative medical and surgical trials, including the National Unstable Angina Pectoris Study, the VA Study in Stable Angina and the Coronary Artery Surgical Study, to be published later, may provide angiographic information and follow-up of medical and surgical cohorts. Although the angiographic forms in each of these studies were not designed to identify easily patients with more than 40% of live muscle at risk from one occlusive event, the films are available for the patients already prospectively randomized, and they could be reviewed to look for this anatomic subgroup. In the medical cohorts the patients identified with one-, two- or three-vessel disease who have more than 40% of live muscle at risk from one occlusive event could be compared with those with one-, two- and three-vessel disease who have far less live muscle at risk from any one event (perhaps less than 20%). Medical and surgical comparisons could be made by selecting appropriate patients in the various subgroups. In addition, individual large referral centers could review their own data to test this concept.

The exact quantification of the amount of muscle at risk might be difficult, but the method selected should be simple and reproducible in different centers to allow comparison of data. One approach might be to divide the left ventricular wall into six segments in the

Figure 4. Two- and three-vessel disease in which each stenosed vessel supplies only the myocardium in its own distribution because of lack of collateral connections. LCA = left coronary artery; Cx = circumflex coronary artery; RCA = right coronary artery; LAD = left anterior descending coronary artery.
right anterior oblique view, with three equidistant segments from the base to the apex along the superior anterolateral surface and three equidistant segments from the base to the apex along the inferomedial surface. On the left anterior oblique view, two equidistant segments might be measured from base to apex on the anteroseptal surface and two on the posteromedial surface, for a total of 10 segments (fig. 5), each of which might be considered to represent 10% of the left ventricular muscle. The distribution of the different arteries would then be expressed in terms of how many segments are dependent on flow through that artery, either directly or via collaterals.

We have learned a great deal about coronary artery disease in the 1970s. The importance of coronary anatomy and left ventricular function has been established. Medical therapy and surgical techniques have improved greatly over the past 10 years. The relative roles of these complementary forms of therapy are being delineated with respect to relief of symptoms and improvement in survival in patients with certain anatomic lesions. Further advances might be hastened if we can identify subsets of patients with one-, two- and three-vessel disease with very high-risk anatomy, i.e., a true “left main equivalent.”

References


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