EDITORIALS

Left Ventricular Performance
Following Direct
Myocardial Revascularization

FEW DATA are presently available concerning the long-term influence of direct myocardial revascularization on left ventricular performance. This subject is of great practical importance. First of all, in the experience of many centers, moderate to severe left ventricular dysfunction carries a significant risk of increased early and late postoperative mortality. Secondly, due to the placebo effect of any intervention on angina, evaluation of the results of bypass operations based on subjective data alone is unreliable. Moreover, improvement in the functional state of the myocardium might be an important objective to be sought following aorto-coronary revascularization. Regional abnormalities of left ventricular contraction are frequent and can be detected, at cineventriculography, in about 60% of patients with coronary artery disease. These abnormalities correspond, with few exceptions, to significant proximal obstructions of the coronary arteries. The direct relationship of these contraction defects to myocardial fibrosis and ischemia has been stressed. In several instances, the heart muscle may be structurally normal but partly inactivated because of ischemia.

In a recent issue of Circulation, Chatterjee et al. have described marked improvement two weeks after operation in various parameters of left ventricular function in 12 patients without and seven patients with previous myocardial infarction. Although some abnormalities in wall motion persisted in the area of infarction, as determined in the preoperative electrocardiogram, improvement of dyskinesis was observed in almost all other areas. Thus, postoperatively, 25 of 29 patients had both normal segmental wall motion and a normal ejection fraction. Grafts in all patients were patent two weeks after operation. Although our results were less striking, our group first reported that regional changes in myocardial perfusion following aortocoronary bypass have a significant influence on the contraction pattern of the left ventricle. Considering the total number of wall segments abnormal before operation and adequately revascularized, we found that more than half of preoperative ventricular wall motion defects showed early improvement (two weeks after operation) and that this frequently persisted one year or more after operation. Localized ventricular hypokinesis was the underlying defect in most instances. As noted by Chatterjee et al., improvement in wall motion abnormalities was much more frequent when the dyskinesis involved the anteropapical walls (79%) than the inferior wall (21%) of the left ventricle. When the residual volume and ejection fraction were abnormal, these values reverted to normal two weeks after operation in patients with totally corrected hypokinesis and improved in most patients with partly corrected

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dyskinesis. Early postoperative improvement has also been noted by other investigators.

However, these early results have not as yet been confirmed in the few presently available late postoperative studies. In a recent investigation performed at our institution, no significant improvement of ventricular volumes and ejection fraction was found in patients with patent grafts one year or more after operation, whereas deterioration was always evident in patients in whom grafts to the anterior descending coronary artery were occluded. Similar data have been reported by others. In addition, there is strong evidence, at the present time, that significant preoperative left ventricular dysfunction is associated with a markedly decreased late survival rate of patients after aortocoronary bypass operations. For example, the mortality rate over a 12- to 18-month period, based on cineangiographic assessment of preoperative ventricular contraction, was 5% in patients with normal wall motion, 12% in patients with localized single wall impairment and 25% to 30% in patients with localized double wall or diffuse impairment. Similarly, the two-year mortality after operation rose from 11% in patients with an ejection fraction between 0.45 and 0.50 to 29% or greater in patients with an ejection fraction less than 0.45.

When one attempts to account for these differences between early and late results after surgery, various factors, related or unrelated to myocardial revascularization, must be considered. For example, during the immediate postoperative period, increased contractility (particularly evident in patients with previously depressed function) possibly occurs partly as a result of stress. Most patients show a significant increase in heart rate during the first postoperative month. Left ventricular end-diastolic volumes are frequently above the upper limits of normal (90 to 95 ml/m²) and frequently remain unchanged postoperatively, suggesting that the left ventricular cavity remains dilated. On the other hand, factors such as structural changes in vein grafts and modifications of the native coronary circulation almost certainly influence the late results.

Thus, although limited improvement in ventricular dyskinesis can be demonstrated late after the operation, impaired left ventricular function may not be significantly improved. On the other hand, the functional state of the myocardium at the time of operation is possibly the major determinant of long-term survival in patients undergoing direct myocardial revascularization. These results warrant our present attitude of accepting for surgery only patients with reasonably good ventricular function and aiming at preservation of function late after myocardial revascularization. More data are needed to determine whether this objective can be accomplished in a significant number of patients.

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