Increase in Severity of Proximal Coronary Disease after Successful Distal Aortocoronary Grafts

Its Nature and Effects

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SUMMARY

Aortocoronary vein grafts were placed in seven patients to bypass severe proximal stenosis in nine coronary arteries. Routine postoperative angiography showed patent grafts in all patients and substantial increase of proximal occlusive disease, diffusely or at the points of narrowing, in six of nine arteries (four patients), with complete obstruction of four of the six vessels. Two of the four patients experienced improvement in angina which was sustained despite the advanced proximal disease. The third patient suffered a late postoperative myocardial infarction and the fourth had recurrence of angina, both probably as a result of the increased proximal disease. The possibility is considered that a successful vein graft, by diverting flow from the poststenotic segment, may accelerate its occlusion and that consequences of advancing occlusive disease may not be prevented by vein-grafting surgery.

Additional Indexing Words:
Direct revascularization
Coronary atherosclerosis
Coronary arteriography
Myocardial infarction
Coronary blood flow
Patient selection for surgery
Angina

THE RESULTS of aortocoronary bypass surgery have proven so far satisfactory but the long-term benefits will be affected by closure of grafts or by the natural progression of the underlying coronary atherosclerosis. Aldridge and Trimble found increase of proximal incomplete arterial lesions to complete occlusion after successful distal grafting and suggested that progression of coronary disease may be accelerated in the grafted arteries. Similar observations by our group have prompted further examination of this important problem and of its consequences.

Patients and Methods

Nine aortocoronary saphenous vein grafts were carried out in seven patients afflicted with severe angina pectoris for nine severe (over 70%), but incomplete, proximal coronary arterial occlusions. Three more grafts were placed in these patients for complete proximal obstructions and these will not be considered further. Cardiac catheterization and coronary and graft arteriography were performed by the Sones method preoperatively and about 6 months after surgery (7 months after the preoperative study). All grafts were found to be patent in these patients.
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Results

Six of the nine grafted arteries which were severely narrowed before surgery appeared occluded in the postoperative coronary arteriograms. However, in two there was no real obstruction, but rather an impediment to forward flow as suggested by the following angiographic features: (1) the opaque medium stopped not at the point of maximal narrowing but distal to it (fig. 1, top); (2) there was a to-and-fro dilution effect at the arrested opaque front with each systole presumably by the large flow of blood without dye coming through the graft; and (3) contrast material injected through the graft filled not only the distal coronary artery but also, retrogradely, the segment of the vessel proximal to insertion of the graft past the point of simulated occlusion and, in the illustrated example, even past the severe narrowing (fig. 1, bottom). By contrast, in patients 1 and 2 (table 1) there was lack of antegrade or retrograde opacification of a segment of the left anterior descending coronary artery between a point just distal to the narrowing and the point of insertion of the graft, suggesting true obliteration of this segment (fig. 2). In patient 3 (table 1) contrast material stopped at the points of the severe, but incomplete, obstruction of the left anterior descending and circumflex arteries (fig. 3). We think that this was due to real rather than simulated occlusion because of its location (i.e. at rather than distal to the narrowing), lack of flow across these points in either direction (fig. 4), and absence of a to-and-fro dilution effect.

In patients 1 and 4 (table 1) the right coronary artery, which preoperatively had one severe localized narrowing with few mild additional lesions, at restudy showed severe diffuse irregularity of the lumen with several points of significant stenosis (fig. 5). Injection of the graft demonstrated minimal retrograde flow which probably was responsible for the arrest of antegrade opacification, but is unlikely to have interfered with forward flow so as to result in the observed proximal diffuse luminal abnormalities.

There was no increase in severity of disease in three of the nine grafted arteries as evidenced by unchanged angiographic appearance of the segment proximal to the anastomosis with the graft. This segment opacified retrogradely only, after injection of the graft, in two of the three arteries which were initially described as exhibiting simulated occlusion. In the third artery the segment opacified both antegrade and retrogradely (a total of three arteries in three patients—not included in table 1).

Six coronary arteries in the four patients of table 1 were not grafted. Two of these had been found occluded in the first study and the remaining four, being normal or slightly diseased, showed no significant change in the period between the two angiograms.

In spite of the anatomic deterioration there was no change in the condition of patients 1 and 4 whose relief from angina after the operation was sustained. However, patient 2 experienced anterolateral myocardial infarction 2 months after surgery, the residua of which were apparent during the postoperative study (a major area of anteroapical akinesis). This infarction occurred possibly because of involvement of important side branches of the occluded middle segment or because the patent graft could not provide sufficient distal flow to prevent infarction.

Patient 3, after initial improvement, experienced partial recrudescence of angina, possibly as the occlusion of the anterior descending and circumflex arteries became complete. This patient deserves special mention because his nondominant right coronary artery had been found occluded at the time of the first study. Following the closure of the left anterior descending and circumflex arteries, coronary perfusion sufficient to maintain near-normal left ventricular function (table 1) has been provided essentially by the two venous grafts (fig. 4, bottom).

Discussion

The above observations document significant increase of proximal coronary artery disease after successful distal bypass and
20° RAO projection. (Top) Postoperative angiogram. Narrowing of the anterior descending artery is shown by the arrow. Arrest of the dye distal to the narrowing. (Bottom) Injection of the graft (broken arrow) fills the anterior descending artery both antegrade and retrogradely. In this patient retrograde opacification extended past the narrowing (solid arrow) and overflowed into the circumflex system.
### Table 1

<table>
<thead>
<tr>
<th>Pt</th>
<th>Restudied after surgery (mos)</th>
<th>Interval between two angiographies (mos)</th>
<th>Type of surgery</th>
<th>Condition of CA and LV Preop</th>
<th>Condition of CA and LV Postop</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11</td>
<td>12</td>
<td>Graft to RC and to LAD</td>
<td>RC: 90% localized narrowing</td>
<td>Diffuse, severe involvement proximal and distal to narrowing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAD: 70% localized narrowing</td>
<td>100% long segment</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>LC: Normal</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>VG: Normal</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LVed: 8 mm Hg</td>
<td>10 mm Hg</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>RC: Occluded</td>
<td>No change</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LAD: 80% occluded</td>
<td>100% long segment</td>
</tr>
<tr>
<td>2</td>
<td>4</td>
<td>6</td>
<td>Graft to LAD and Resection of small lateral aneurysm</td>
<td>LC: 50% occluded</td>
<td>No change</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>LC: Normal and contractility despite small aneurysm</td>
<td>Large segment of anteroapical akinesis</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>VG: Normal</td>
<td>Normal</td>
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<td></td>
<td></td>
<td></td>
<td>LVed: 4 mm Hg</td>
<td>12 mm Hg</td>
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<td></td>
<td></td>
<td>RC: Occluded</td>
<td>No change</td>
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<td></td>
<td></td>
<td></td>
<td>LAD: 90% occluded</td>
<td>100%</td>
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<td></td>
<td></td>
<td></td>
<td>LC: 90% occluded</td>
<td>100%</td>
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<td></td>
<td></td>
<td></td>
<td>VG: Mild generalized hypokinesis</td>
<td>No change</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>LVed: 18 mm Hg</td>
<td>14 mm Hg</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RC: 90% localized narrowing</td>
<td>Diffuse severe narrowing of the vessel</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>LAD: Occluded</td>
<td>No change</td>
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<td></td>
<td></td>
<td></td>
<td>LC: Normal</td>
<td>No change</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>VG: Normal</td>
<td>Normal</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>LVed: 5 mm Hg</td>
<td>11 mm Hg</td>
</tr>
</tbody>
</table>

Abbreviations: RC = right coronary artery; LC = left circumflex artery; LAD = left anterior descending artery; VG = ventriculogram; LVed = left ventricular end-diastolic pressure at rest; CA = coronary arteries; LV = left ventricle.
Case 2, 20° RAO projection. (Top) Preoperative angiogram of left coronary artery. Most severe lesion is shown by the arrow. Large septal branch is seen. Broken arrow shows approximate position of graft insertion as judged from landmarks (sternal wires) in the postoperative study. (Bottom, left) Postoperative angiogram. Occlusion of the artery (broken arrow) distal to the severe narrowing (solid arrow) after the origin of the large septal branch. (Bottom, right) Filling of the distal anterior descending artery by the graft. There is no retrograde flow to the occluded segment of the anterior descending artery (nor was any visible at 40° LAO projection).
suggest that this is most likely a consequence of grafting rather than progression of coronary atherosclerosis, for the following reasons: (1) the occlusive process increased more rapidly than one would expect from the natural course of atherosclerotic disease; (2) in most patients long, previously uninvolved, segments of vessels were affected rather than the stenotic sites; and (3) there was no significant progression of disease in arteries that were not grafted. Also, we think that true occlusion occurred rather than closing down of these vessels, since such collapsed segments should have filled retrogradely from the graft, as observed in the two patients with the simulated obstruction.

Effects of experimental bypass grafts on pressure and flow in the recipient coronary circulation have been recently reported. These investigators placed a vein graft to the circumflex coronary artery in dogs and measured blood flow through the artery and through the graft, when the latter was open, before and after production of varying degrees of proximal artery stenosis with an adjustable snare. With the graft occluded, stenosis of the circumflex artery by less than 75% produced no significant change in distal pressure or flow. When the narrowing exceeded 75%, and depending on the degree of constriction, it produced a varying decrease of pressure and flow in the distal artery. While maintaining the stenosis the graft was opened and this restored distal coronary flow, but then the flow through the subtotally occluded proximal artery decreased further, sometimes down to zero. This work suggests that slowing or stasis as a result of decreased blood flow through the proximal narrowing may be the most important cause for the occlusions we have described. The high incidence of this complication in our cases (six of nine arteries)
and in those of Aldridge and Trimble (four of eight arteries) may also be considered to support the above concept. The observations of the present study should not be considered as arguments against direct revascularization but, rather, as points

![Image of coronary artery angiograms](image_url)

**Figure 3**

Case 3. 40° LAO projection. (Top) Preoperative angiogram of the left coronary artery. Severe narrowing of the circumflex (solid arrow) and anterior descending arteries (broken arrow). (Bottom) Postoperative angiogram shows complete occlusion at the above points.
Case 3. 20° RAO projection. (Top) Postoperative angiogram of the left coronary artery. The black arrow points to the stump of the circumflex artery and the white arrow points to the stump of the anterior descending artery. It should be noted that opacification of the anterior descending artery stops before the posterior end of the nearest sternal wire which will be used as a landmark. (Bottom) Postoperative graft injection. Filling of the anterior descending and circumflex systems is seen. The solid black arrow shows the end of the retrograde filling of the anterior descending artery proximal to anastomosis with the graft. It should be noted that the point is close to the anterior end of the same sternal wire used as a landmark in figure 4, above (although at a different vertical level, presumably due to unequal depth of inspiration in the two runs). The interrupted black arrow points to the anastomosis of the other limb of the graft to the left circumflex coronary artery. This point also corresponds to the posterior end of the index sternal wire and thus to the point of arrest of contrast material in the stump of the anterior descending artery. A segment of the anterior descending coronary artery between these two arrows is presumed to have been occluded.
Case 4. 30° RAO projection. (Top) Preoperative angiogram of right coronary artery, with 90% narrowing shown by the arrow. Two moderate (50%) lesions are seen proximal to it. Broken arrow points to large right ventricular branch. (Bottom) Postoperative angiogram. Diffuse severe decrease in caliber of the artery is seen. Arrest of dye is not due to obstruction, but to retrograde flow from the graft (not shown). As in top, narrowing is shown by the solid arrow and the right ventricular branch by the broken arrow.
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underscoring the need for careful patient selection. The objectives of aortocoronary graft surgery are: (1) to restore decreased blood flow to one or more parts of the distal coronary arterial tree, and in so doing to relieve angina, and (2) to prevent consequences of advancing occlusive or ischemic disease, such as myocardial infarction. Despite occasional failures surgery appears indicated in severe angina because it offers worthwhile symptomatic relief and also because such improvement appears to be maintained as long as the grafts remain patent, despite graft-induced or spontaneous increase in proximal disease. However, our observations suggest that effects of advancing occlusive or ischemic disease may not always be prevented and might even be precipitated by grafting. Thus, we think that until controlled studies demonstrate significant improvement in prognosis, surgery should not be recommended prophylactically in most patients with few or no symptoms.

Acknowledgment

We thank Mrs. M. Steffek and Mr. S. Dees, cardiac catheterization technicians, Mr. R. Szembrot, Medical Illustration Department, and Mrs. M. L. Peplowski, secretary, for their assistance.

References

Increase in Severity of Proximal Coronary Disease after Successful Distal Aortocoronary Grafts: Its Nature and Effects
GEORGE BOUSVAROS, ABDUL R. PIRACHA, MUHAMMAD A. CHAUDHRY, COLIN GRANT, THOMAS M. OLDER and ROQUE PIFARRE

_Circulation_. 1972;46:870-879
doi: 10.1161/01.CIR.46.5.870

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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