COMMENTARY (This commentary provides editorial perspectives on the report which follows)

Aortocoronary Vein Graft Spasm*

A Clinical Entity?

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The concept that saphenous veins which have been transplanted into the arterial circuit are passive, unreactive conduits may be an oversimplification. The unique report by Victor et al in this issue (see page 413) proves that such vein grafts can go into severe spasm. Their report describes a patient who suffered recurrent angina pectoris after undergoing a triple coronary artery bypass operation. Postoperative catheterization demonstrated total obstruction of one saphenous vein graft, multiple severe obstructions distal to two patent grafts, and transient severe spasm in the graft to the circumflex artery. Their report raises several challenging questions.

Did vein graft spasm really occur in this case?

Laminar flow patterns in saphenous vein grafts may produce artifacts which mimic narrowing of the graft. These flow patterns are more easily detected in cine studies than in single pictures. I reviewed the original 35 mm angiograms of this case with four other angiographers (combined experience more than 30,000 coronary arteriograms). We measured 85 percent transient narrowing of luminal diameter in the saphenous vein graft, and we agreed that the narrowing was not due to a flow artifact.

What was the clinical significance of vein graft spasm in this case?

This is not known. The patient did not manifest angina or ischemic ECG changes during spasm, and therefore, there is no proof that the patient's symptoms were related to saphenous vein graft spasm. The postoperative catheterization revealed severe obstructions in all three coronary arteries beyond the bypass grafts. Therefore, there was enough disease in the native circulation to produce angina.

What is the likelihood that saphenous vein graft spasm occurs in other patients?

Severe spasm has never been documented among more than 5,000 saphenous grafts visualized angiographically at the Cleveland Clinic. Therefore, the phenomenon is probably rare. One case of severe saphenous vein graft spasm has been observed during surgery at the Cleveland Clinic. The spasm in this patient occurred soon after both anastomoses had been established, and it subsided after papaverine was infused into the graft. Repeat catheterization one week after operation demonstrated occlusion of this graft.

Do the histologic and physiologic changes which occur in saphenous vein grafts predispose to spasm?

After excision, normal human saphenous veins still contract in response to serotonin, norepinephrine, and histamine. The contractile response is stronger in veins obtained from the lower leg, and this corresponds to the thicker musculature of the media in these veins, compared to veins in the thigh.1 Within one month after surgery, human aortocoronary saphenous vein grafts demonstrate inflammatory cell infiltration and variable necrosis of smooth muscle cells in the media. After six months, these grafts show a marked decrease in the number of smooth muscle cells in the media, with replacement by fibrous tissue and collagen. Thus, the saphenous vein bypass graft becomes a stiff, fibrous tissue conduit. All saphenous vein grafts show similar changes, but the degree of these changes varies from one patient to another, from one graft to another in the same patient, and from one segment to another in the same graft.2-4 Similar histologic changes occur in saphenous vein grafts in dogs.5 After six months, such grafts demonstrate a marked decrease in contractility to one-sixth or less of nor-

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Spasm of a Saphenous Vein Bypass Graft*

A Possible Mechanism for Occlusion of the Venous Graft

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The phenomenon of spasm in a venous graft was documented by angiographic study of the graft. This phenomenon has not been reported previously, and, therefore, its frequency of occurrence is unknown. Spasm of a venous graft may prove to play a significant role in the early development of myocardial infarction, closure of the graft, or recurrence of angina after initially successful surgery for aortocoronary bypass when venous grafts remain patent.

We report a case of spasm involving a saphenous venous graft in a patient who had accelerated recurrent exertional angina and pain at rest. Three months earlier, he had undergone surgery for aortocoronary bypass because of left main coronary arterial disease. As far as we know, this is the first case of spasm of a venous graft to be reported in the literature.

CASE REPORT

A 66-year-old man with previously stable angina pectoris for the past two or three years had symptoms that had worsened over a two-month period. One week prior to admission, the patient had experienced ventricular tachycardia while undergoing an exercise stress test. The results of the test were strongly positive, with a 5-mm to 6-mm downward-sloping ST-segment depression. The results of physical examination were normal, except for a fourth heart sound.

Cardiac catheterization and angiographic studies were performed; they showed an 80 percent luminal narrowing of the left main coronary artery, a 40 percent narrowing of the diagonal branch of the left anterior descending artery, 80 percent narrowing of the left circumflex artery before the major marginal branch, and total occlusion of the right