Treatment of Coronary Artery Disease: Sisyphus' Work?

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A 37-year-old male with a hyperlipidemia type IIa underwent three conventional percutaneous transluminal coronary angioplasty procedures and three surgical revascularizations for recurrent restenosis of the saphenous vein bypass graft. Despite these interventions, a long-term success was never achieved. Stent implantation at the site of a rerestenosis was successful. However, a second stent implantation to prevent further progression of another previously nonmanipulated atheromateous lesion resulted in a stenosis within the stent for which a new surgical intervention was indicated. At surgery the stented graft was replaced by a fresh venous graft. The partly removed venous graft containing the two stents provides a unique opportunity to study the long-term histologic effects of intravascular stenting. (J Interven Cardiol 1992; 5:5–14)

Introduction

Percutaneous transluminal coronary angioplasty (PTCA) has become the treatment of choice for most patients with symptomatic single vessel disease and selected multivessel disease. Another rapidly growing indication for PTCA consists of patients with recurrent angina after coronary bypass surgery, who may undergo either dilatation of a native coronary artery stenosis or of a bypass graft stenosis. However, in approximately 20%–40% of patients, evidence of myocardial ischemia reappears within 6 months of the dilatation.[1–3] Restenosis rate within the bypass graft is even higher than the rate observed in the native coronary vessels[4,5] and remains one of the main limitations of this therapeutic procedure.

Recently, intravascular stenting for prevention of restenosis was introduced.[6] Initial data suggest that stenting may offer an alternative treatment to selected patients with recurrent angina due to graft stenosis after previous coronary bypass surgery.[7]

This case report highlights the problems of patient management with recurrent restenosis of saphenous vein bypass grafts after several angioplasties and surgical revascularization procedures. Despite these various modes of treatment a long-term success was never achieved. Stent implantations were performed initially to treat a recurrent stenosis accompanied by symptoms and later to prevent progression of a stenosis not associated with symptoms. However, after 3 months angina recurred and angiography revealed a stenosis in the second stent. Another surgical revascularization was indicated. Again at 3 months, angina recurred due to a stenosis at the anastomosis between the two grafts. A third stent was successfully implanted.

This anecdotal case gives us the opportunity to review the changing spectrum of pharmacological and interventional treatment of ischemic heart disease over the last decade.

Case Report

Clinical History. In 1977 a 37-year-old active smoker with a hyperlipedimia type IIa and a posi-

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Submitted for publication February 22, 1991; accepted with revisions June 7, 1991; revisions received July 9, 1991.

Table 1. Case History

Date	Intervention
September 1977	CABG; jump to LAD, RD, RM, RPD.
January 1982	PTCA of a totally occluded graft.
June 1982	CABG; bypass on the bypass.
November 1982	PTCA of bypass on bypass.
May 1984	rePTCA of bypass on bypass. PTCA stenosis in distal part of the bypass.
September 1988	1st STENT rerestenosis of bypass on bypass.
April 1989	2nd STENT to prevent progression of an atheromatous plaque.
July 1989	CATH; within stent stenosis in 2nd stent, patent 1st stent.
July 1989	CABG; venous interposition in proximal jumpgraft. LIMA to distal jump graft, bypass to RPL.
December 1989	3rd STENT for a stenosis in the interposed graft.

CABG = coronary artery bypass grafting; CATH = catheter; LAD = left anterior descending coronary artery; LIMA = left internal mammary artery; PTCA = percutaneous transluminal coronary angioplasty; RD = diagonal branch of the left coronary artery; RM = marginal coronary artery; and RDP = descending posterior coronary artery.

tive family history, presented with an inferior-myocardial infarction. Table 1 summarizes the major events in the 12-year time frame. Briefly, coronary angiography revealed an occlusion of the left anterior descending coronary artery (LAD) and left circumflex coronary artery. The marginal arteries were severely diseased. Collateralized circulation from the right coronary artery to the distal part of the left descending coronary artery was visualized.

Despite optimal pharmacological treatment and intraaortic balloon pumping he remained unstable and received one jump graft with four distal anastomoses at urgent coronary artery bypass grafting.

Recovery was uneventful and the patient was discharged on dipyridamol and aspirin therapy. Repeat angiography and thallium exercise testing were performed 1 and 4 years later as part of an ongoing study of the functional result of bypass surgery on left ventricular (LV) function.[8] At 4 years (March 1981) angiography revealed a 70% stenosis of the distal anastomosis to the second obtuse marginal branch. Because of persistent elevation of serum cholesterol, he was referred to our lipid clinics. Initial treatment consisted of colestyramine. An optimal dose regimen was never given because of nausea. Since 1988 he was treated with symvastatine, a HMG CoA reductase inhibitor. The resultant decrease in the cholesterol levels is shown in Table 2.

In January 1982, balloon angioplasty was performed on a totally occluded venous jump graft for recurrent angina. Because of an extensive collateral network, the patient was spared a recurrent myocardial infarction. Repeat attempts at surgical revascularization was not performed because of anticipated risk exceeding those accruing to the performance of balloon angioplasty.[9,10] The occlusion was mechanically recanalized by means of a 0.035-inch teflon coated guidewire and successfully dilated, although a long nonobstructive dissection was documented at the site of the dilatation.

Recurrence of angina was noted at 2 months

Table 2. Serum Cholesterol Level

Date	Cholesterol (mmol/L)	Triglyceride (mmol/L)	Medication
1977	7.6	1.40	
1978	12.7	1.45	
1979	8.8	0.96	
1982			Colestyramine 4 × 4 g
1983	9.5	1.06	
4/13/88	10.4	1.30	
4/18/88	8.6	1.63	
5/02/88	9.7		Colestyramine 1 × 4 g
6/21/88	10.2		Colestyramine 1 × 4 g
8/02/88	10.6		
10/06/88	10.8	1.66	
11/17/88	8.2	1.23	Simvastatine 1 × 10 mg
12/29/88	7.9	1.14	Simvastatine 1 × 20 mg
2/14/89	7.8	1.31	
4/05/89	6.8	2.29	
7/18/89	7.1		Simvastatine 1 × 30 mg
10/23/89			Simvastatine 1 × 40 mg
12/27/89	5.0		

Normal values; total cholesterol, 3.3–8.0 mmol/L; triglycerides, 0.3–1.6 mmol/L.

follow-up. A second balloon angioplasty was considered contraindicated because of the dissection flap and a surgical reintervention was recommended. At surgery (June 1982) a bypass graft was implanted on the proximal part of the preexisting bypass.

In November 1982, he again experienced exertional angina due to a stenosis of the anastomosis of the bypass on the bypass that was successfully dilated.

In May 1984, a third PTCA procedure was necessary to dilate a restenosis at the anastomosis of the bypass on the bypass.

In April 1988, he again became increasingly symptomatic despite optimal pharmacological treatment. This time coronary angiography disclosed a total occlusion of the distal jump graft between the two marginal branches and a rerestenosis of the bypass—jump graft anastomosis. With our surgical team, the decision was taken to perform PTCA with stent implantation.

In September 1988, a fourth PTCA procedure was performed. The rerestenosis of the bypass—jump graft was successfully dilated with a 4.2-mm balloon at 12 atmospheres (atm). After dilatation a residual stenosis of 20%–30% was present and there upon a 4.5-mm diameter stent (Medinvent, Lausanne, Switzerland) was implanted (Fig. 1). At 6 months angiography there was no recurrent stenosis but a plaque encroaching the lumen at distance of the previous implanted stent was documented. To prevent further progression of this lesion in the single remaining conduit supplying the entire heart it was decided to implant another stent. This asymmetric encroaching plaque was successfully reached and a 4.5-mm stent (Medinvent) implanted (Fig. 2). At 4 months he presented again with unstable angina. At coronary angiography a severe restenosis within the second stent was found. Another PTCA procedure was considered inappropriate mainly because the single remaining stenotic bypass graft was considered a main stem equivalent. In addition, the fibrocellular and proliferative nature of the stenosis let us foresee a high probability of an unfavorable short-term result.

In July 1989, the proximal part of the jump graft was resected and replaced by a new venous graft. The left internal mammary artery (LIMA) was grafted distally on the jump graft and a new venous graft was connected to a posterolateral branch.

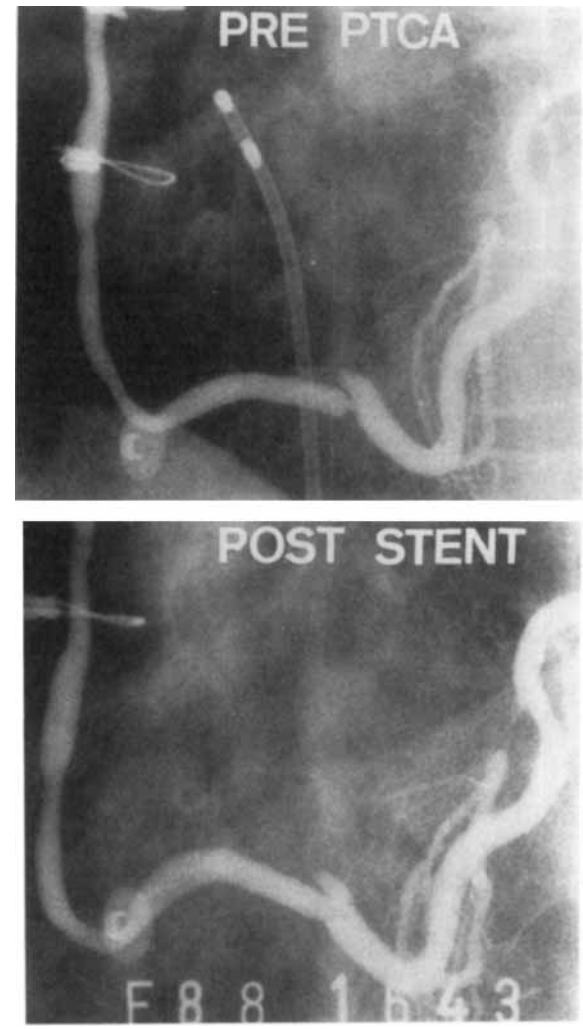

Figure 1. Successful implantation of the first stent. In the upper panel, the pre PTCA angiogram is shown with the rerestenosis in the proximal bypass graft. The lower panel shows the post stent implantation angiogram.

In December 1989 he again experienced exertional angina. Repeated coronary angiography revealed a severe proximal stenosis in the jump graft and a functional occlusion of the LIMA. The angiographic appearance of the stenosis suggested a cicatricial retraction at the site of the suture had occured. A fourth surgical revascularization was considered inappropriate since the internal mammary artery had already been used. Subsequently, a third stent implantation was per-

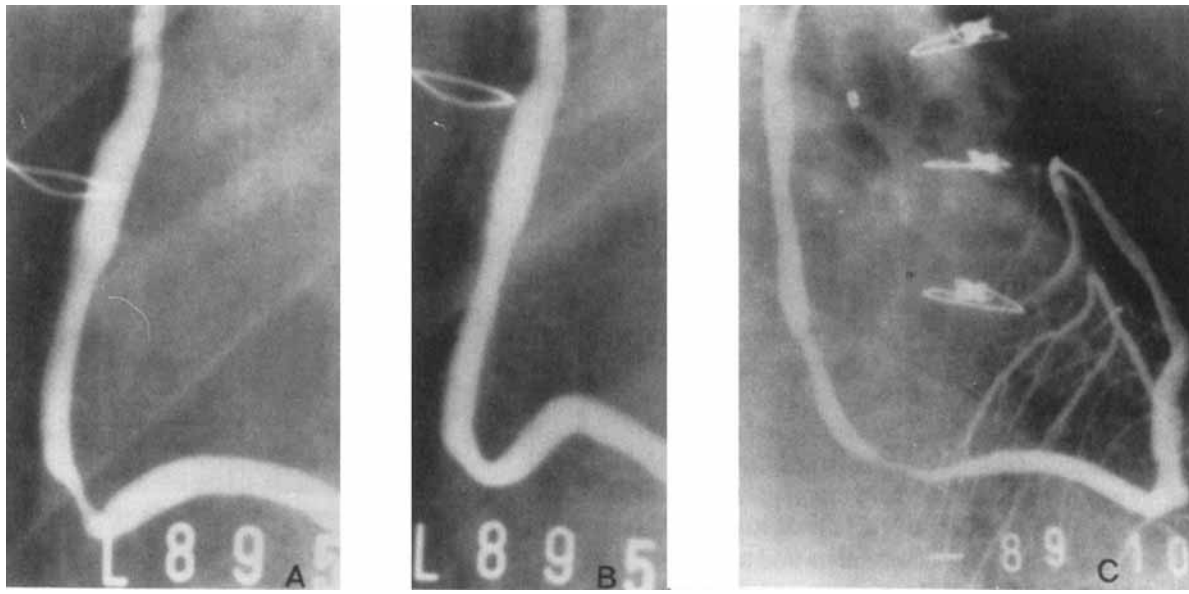

Figure 2. Cineangiogram of the second stent implantation procedure. (A) The primary stenosis before stenting is shown. The first stent is widely patent without signs of a restenosis. After stent implantation (B) no residual stenosis is seen. (C) Three-month follow-up angiogram after the second stent implantation (10 months after the first stent implantation). The second stent contains a restenosis while the first stent is widely patent.

formed without complications (Fig. 3). At 3 months angiographic follow-up, the stent was patent without signs of abnormal hyperplasia.

Histology. Data from our animal experiments have shown that following stent implantation major histologic changes occur.[11] The embedded stent wires create a deformation and outstretching of the media while the internal elastic membrane is interrupted. Within 48 hours after stent implantation massive deposition of leucocytes, platelets, erythrocytes, and fibrin, occur in the "dead spaces" surrounding the stent struts. This process in combination with the presence of the nondegradable wires and hemostasis at the site of the struts results in chemotaxis of monocytes. These monocytes subsequently transform into macrophages, which act as scavengers and are ultimately transformed into foam cells. At 4 weeks a neointima composed of disorganized layers of myofibrillar cells covers the stent wires. At the luminal side two distinct layers of smooth muscle layers are present, one in a circular orientation immediately below the endothelium and a deeper layer in a longitudinal orientation. Finally, complete coverage with endothelial cells will occur. After 3 months, a more extensive neointima has formed with only a small area adjacent to the stent wires containing leucocytes and cellular debris.

Histologic cross section of a porcine coronary artery 1 week after stent implantation thus reveals a combination of thrombotic and myoproliferative process, which was initiated in the "dead spaces" near the stent struts. In the stented venous bypass graft, the spaces directly around the stent struts become filled with depositions of leucocytes, macrophages, and fibrin that were all attracted by chemotactical forces. During formation of the neointima these "dead spots" are burled and become triangular in shape. We believe that in these triangular spaces the mystery of restenosis following stent implantation may be found. Therefore in this report, named after an ancient Grecian myth, we selected the name "Bermuda Triangle" for these regions because these "dead spaces" may play the pivotal role in stent induced restenosis.

Light microscopic examination of the stented human venous bypass graft of our patient showed the same histologic changes as seen in pigs. The

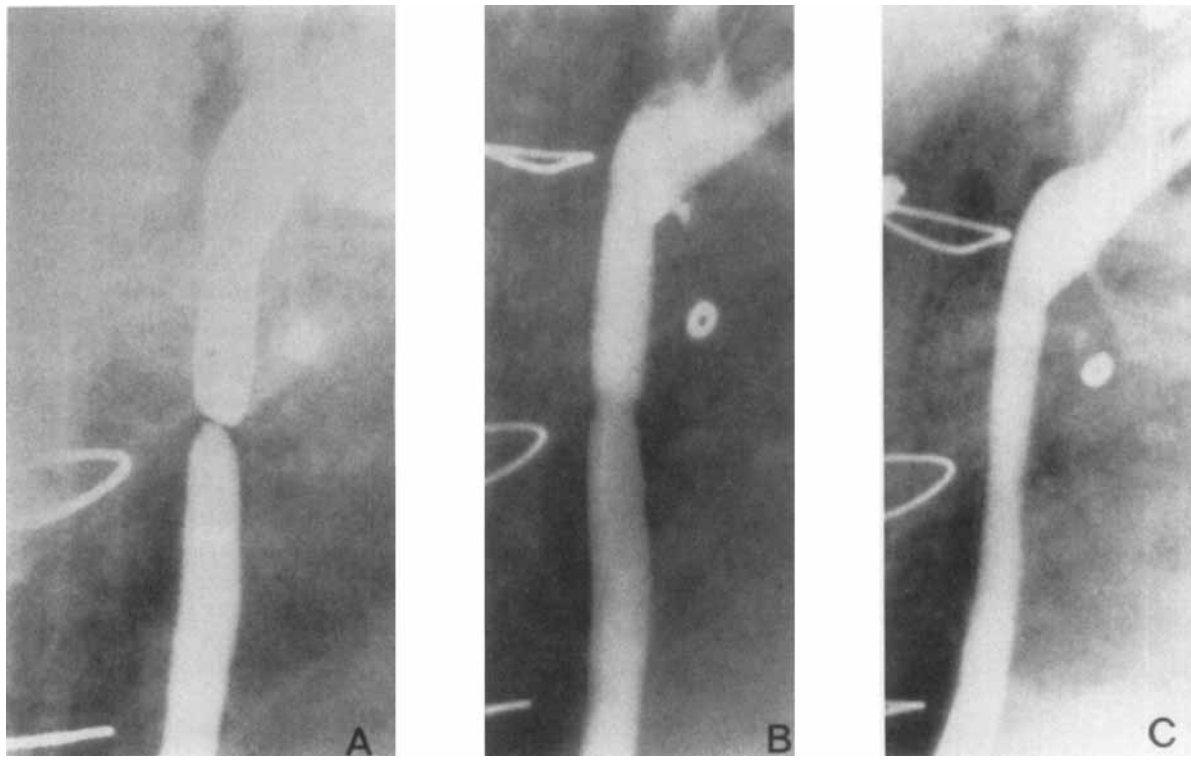

Figure 3. Cineangiogram of the third stent implantation. Frames A and B show the stenosis before and after stenting. Frame C shows the 3-month follow-up result.

first stent was completely covered by a smooth endothelial lining and the neointima consists of a few layers of elongated smooth cells with interpersed collagen and some elastin fibers (Fig. 4). In the vicinity of the stent wires, now seen as voids, necrotic tissue is seen and only a few foam cells are demonstrated.

The second stent is also completely covered by an endothelial lining; however, the neointima is less densely organized and considerably thicker compared with the first stent (Fig. 5). This neointima is vascularized and consists of many layers of ill-arrayed smooth muscle cells and collagen. Around the voids, in the "Bermuda Triangle," many leucocytes, foam cells, and elongated cells with lipid inclusions are found (Fig. 6). This area initially contained the corpse of leucocytes, erythrocytes, and platelets, which were phagocyted by attracted macrophage that ultimately transformed into foam cells.

Discussion

Coronary artery bypass grafting (CABG) is an effective therapeutic treatment for symptomatic coronary artery disease. Although more than 80% of the patients become angina free, recurrence of symptoms occurs in approximately 5% to 10% of the patients annually. Repeat CABG provides symptomatic relief in only 60% to 70% of the patients compared with more than 80% after the first CABG procedure.[9] Moreover, repeat CABG is technically more difficult and mortality rates are higher than after initial operation.[10]

We report our therapeutic strategy to prevent repeat bypass surgery in a patient with a bypass on a bypass.[12] Although all kind of interventional techniques have been applied, a long-term success was never achieved and two reoperations had to be performed. Reviewing this case history one can speculate whether another strategy

Figure 4. Histology specimen of the venous bypass graft containing the first stent 10 months after implantation. A smooth endothelial lining covers the neointima, which consists of a few well-arrayed layers of smooth muscle cells.

would have resulted in a more favorable long-term result. Some limitations and possible alternatives to the currently used strategy will be discussed briefly.

Should We Have Intervened More Aggressively on the Lipid Metabolism In This Patient? Studies on lipid lowering drugs in young males have shown cholesterol reduction has a beneficial effect on cardiovascular risk.[13] It has also been clearly demonstrated that late coronary vein graft stenosis is due to progression of atherosclerosis.[14–16] Regression of coronary artery lesions due to lipid lowering interventions has been documented and is the subject of several ongoing trials.[17–19] At the initial presentation of this patient a severe hypercholesterolemia was found. Initial treatment with colestyramine was unsuccessful in effectively reducing the cholesterol level and was accompanied by side-effects. The advent of a new class of cholesterol lowering drugs has contributed to normalize the cholesterol levels. In retrospect, more intense efforts at cholesterol lowering should have been made earlier.

Today Would We Have Attempted PTCA in the First Place? A point of consideration is whether an initial PTCA procedure would have been more beneficial to the patient. Since 1977 the treatment of acute myocardial infarction and unstable angina has changed dramatically. As an alternative to surgical revascularization, PTCA is being attempted in many instances as an emergency intervention in patients with unstable angina and suitable coronary anatomy. Dilatation of the culprit stenosis has a 89% success ratio while complication and restenosis rates are within acceptable limits.[20] Unfortunately, at the time of his initial angiography we were confronted with two totally occluded coronary arteries. With the introduction of more sophisticated catheters it has been demonstrated that PTCA of totally occluded arteries is feasible although a high restenosis rate is ob-

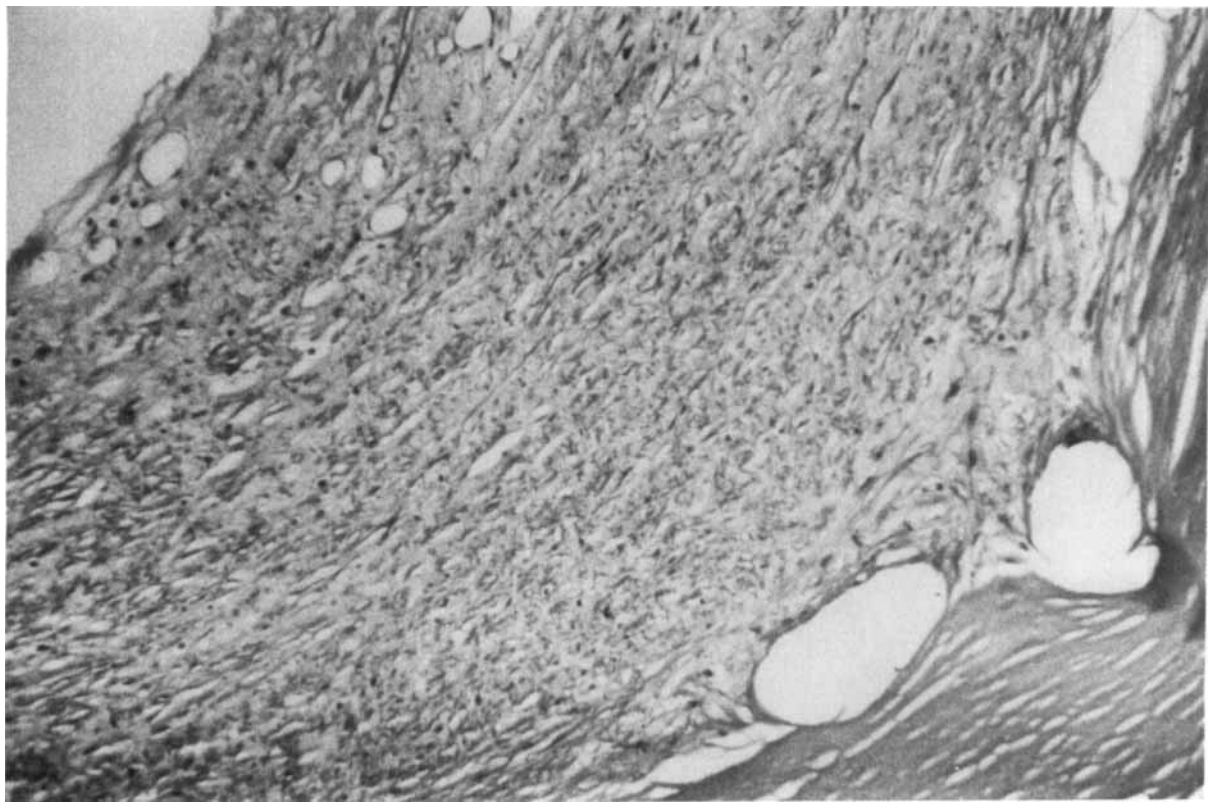

Figure 5. Histology specimen of the venous bypass graft containing the second stent 3 months after implantation (hematoxylin-azofloxine staining). The neointima is vascularized and consists of many layers of ill-arrayed smooth muscle cells and collagen.

served.[21–23] A recent study from our institution shows that recanalization and dilatation of totally obstructed arteries in unstable patients has a favorable clinical outcome.[24] Today, in our initial strategy for this young man, we would contemplate an attempt at recanalization of the culprit lesion in order to avoid or to postpone surgery.

Should We Have Used An Arterial Conduit Instead of a Saphenous Vein Graft? Initial surgical revascularization was achieved by saphenous vein bypass grafting. Currently, long-term follow-up studies comparing venous grafts to internal mammary artery grafts demonstrate a superior long-term patency in mammary artery grafts over the vein grafts.[15,16,25,26] Actuarial survival comparison showed superior survival with a LIMA–LAD graft.[26] Also, the LIMA is an obvious choice in patients undergoing reoperation for failed vein grafts to the LAD.[15] At the last operation the LIMA was used as a graft to the jump graft, and parts of the original venous graft were replaced by new venous grafts. Although the LIMA became occluded, the ideal revascularization procedure might have included the use of the left and right internal mammary artery instead of venous autogenous grafts.

Stent: Chance of the Last Resort Or Start of a New Iatrogenic Era? Lastly, stenting has to be reviewed. Although in selected cases it appears to be a promising new technique several problems remains to be solved. The mechanism by which stent implantation may prevent restenosis is unknown. From a pathophysiological view point the stent may act as a scaffolding device optimizing the dilatation process,[27] or may create a smooth endovascular lining reducing tubulent and laminar resistances.[27,28] Several hypotheses have been proposed to explain the ability of the stent to prevent progression of atherosclerosis. Ac-

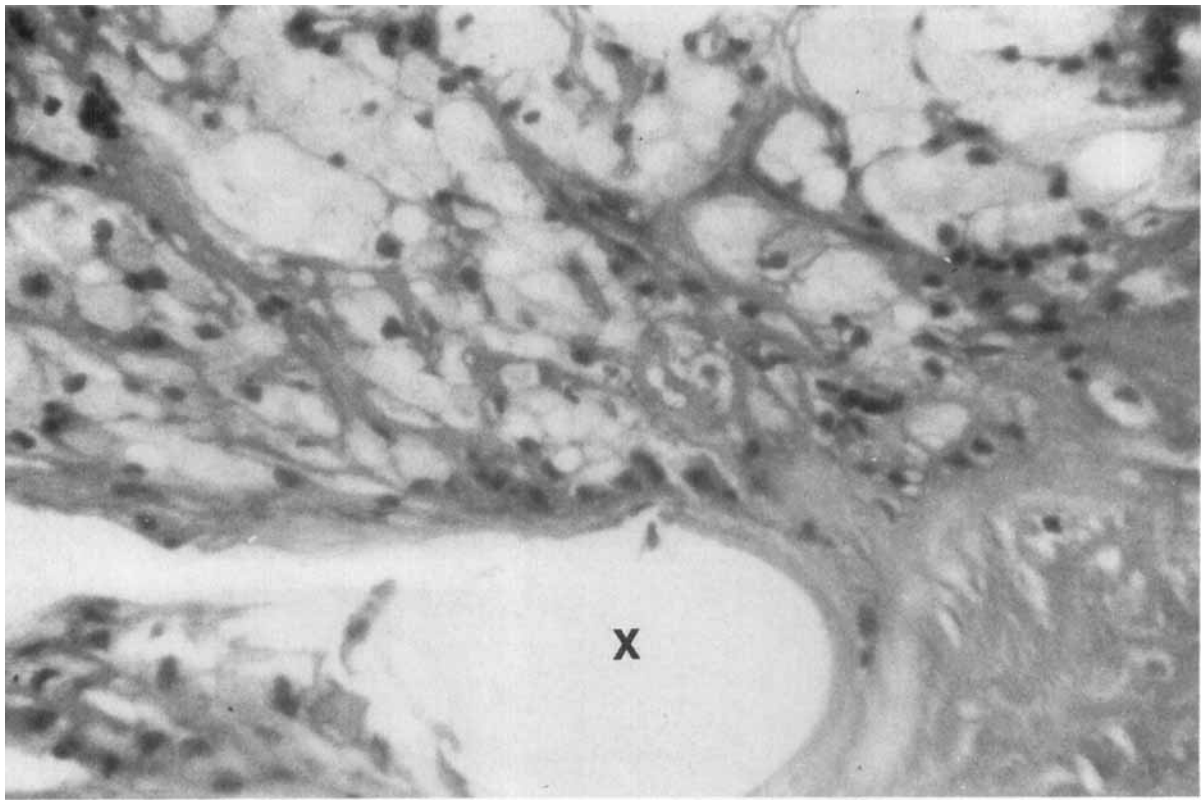

Figure 6. Light microscopic cross section through the "Bermuda triangle." Hematoxylin-azofloxine staining. The voids marked by X originally contained the stent wires. Around the struts many foam cells and leucocytes are present.

cording to these theories the stent may act as a mysterious "tissue barrier" preventing the migration of cellular structures although no scientific proof supports this thesis. Second, the stent may induce ischemia in the vessel wall through compression and occlusion of the nutrient vasa vasorum and thereby limit progression of atherosclerosis.[29] Finally, the stent by acting as a macroscopic screen prevents the protrusion of the atherosclerotic plaque into the lumen of the vessel.[30] On the other hand stent-induced restenosis might result from damage of the internal elastic membrane, thereby inducing cell migration and proliferation.[31]

At PTCA, substantial endothelial damage is induced resulting in splitting the arterial intima or even the media.[32,33] This may be the start of a vicious circle resulting in macrophage attraction and smooth muscle proliferation to form a neointima. The presence of an intravascular stent may become an additional trigger for continued mediator release, resulting in macrophage migration through the internal elastic membrane and foam cell formation.[31]

Histology of a Stent: An Open Window on the Restenosis Phenomenon. The unique pathology specimen retrieved from this patient at surgery shows the long-term effects of intravascular stenting, and thus contributes in our understanding of the basic process of atherosclerosis and the cellular events involved in this case. In the first stent no signs of a proliferative process was found while in the second stent an aggressive cell migration and proliferation was seen.

The cause of these later histologic features are unknown. We speculate that two factors may be important. First, the newly formed endothelium covering the embedded stent wires may be dysfunctional and thus permit abnormal and excessive macrophage penetration and lipid accumulation across the endothelial barrier. Second, important chemotactic substances may be released

in the "Bermuda Triangle," presumably due to pressure necrosis.

As a result of this process, the neointima in the second stent had become an extremely thickened layer mainly consisting of lipid containing cells. Alternatively, the histologic changes observed in the second stent might have started with a thrombotic process resulting in colonialization by myofibroblastic cells. This results in an organized thrombus mimicking some features of the hyperplastic process.

The major cellular difference between the two stents is the abundance of foam cells in conjunction with a proliferative process resulting in intimal hyperplasia in the stent containing the restenosis. With electron microscopy it is clearly apparent that the monocytes are the precursor since these cells penetrate into the endothelial cells. This might be the onset of the vicious circle of chemotaxis, cellular attraction, foam cell transformation, and proliferation. Since the development of restenosis is thought partly to be related to hyperlipidemia, a pharmacological intervention resulting in lowering serum lipid levels might prevent foam cell accumulation and might be recommended to accompany interventional techniques like PTCA or intravascular stenting.

Lessons Learned From Stenting In This Case. It remains a rather interesting question why in only one of the two stents restenosis occurred. One explanation might be that the first stent was implanted because of the development of rerestenosis after several PTCA procedures, while the second was implanted into a nonmanipulated vessel segment containing an aggressive, proliferative plaque. The progression of the atheromatous process at the site of the second stent might have been too aggressive to be contained by the stent. Confronted with this observation we refrain from preventive stenting. These observations are in accordance with the theory of Schatz[30] that the stent may act as a sieve. In our histologic specimen no signs of ischemia induced changes in the vessel wall were found. In contrast, the neointima of the second stent was highly vascularized, which gives no support to the thesis concerning restenosis prevention proposed by Williams.[29]

This case report emphasizes evolution of management with problems of integration of palliative interventional strategies into a chronic disease. Several times decisions were made when long-term data were not available. Although there exists an enlarging experience with interventional techniques, a pharmacological approach appears to be a necessary adjunct to overcome the problem of restenosis.

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