In-stent recurrent stenosis in stents placed in the lower extremity venous outflow tract

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Purpose: **This study was undertaken to describe development of in-stent recurrent stenosis (ISR) in stents placed in the iliocaval outflow tract and to examine possible contributing factors.**

Method: **After iliocaval balloon angioplasty and stent insertion to treat chronic nonmalignant obstruction, single-plane transfemoral venography was performed at least once in 324 limbs, twice in 123 limbs, three times in 40 limbs, and four times in 4 limbs. ISR was measured with a caliper, and expressed as percentage diameter reduction of patent lumen on the venograms. Left-right limb ratio was 2.3:1; thrombotic-nonthrombotic disease ratio, 1.2:1; negative-positive thrombophilia test ratio, 1.6:1; and above-below inguinal ligament stent placement ratio, 4.5:1. Median stent length was 9 cm (range, 4-35 cm), and median lumen area before and after stenting was 0.41 cm^2 (range, 0-1.65 cm^2) and 1.70 cm^2 (range, 0.65-4.00 cm^2), respectively. Limbs were divided into groups with no ISR, any degree of ISR, greater than 20% diameter reduction, and greater than 50% diameter reduction. Cumulative ISR and patency rates were analyzed. Possible contributing factors were examined.**

Results: **At 42 months, only 23% of limbs demonstrated no ISR. Cumulative rate of limbs with greater than 20% diameter reduction was 61%, and of limbs with greater than 50% diameter reduction was 15%. Patient gender or sidedness of the treated extremity did not affect outcome. At 36 months, limbs with thrombotic disease had higher ISR rates than did limbs without thrombotic disease (63% and 41% of limbs with >20% narrowing, and 23% and 4% of limbs with >50% narrowing, respectively; $P < .01$). Similarly, higher rates of ISR were found in patients with thrombophilia and long stents extending below the inguinal ligament. Primary, assisted primary, and secondary patency rates for the entire population at 3 years were 75%, 92%, and 93%, respectively. There was a significant increase in ISR in individual limbs, but analysis of groups of stents did not unequivocally show progression.**

Conclusion: **Severe (>50%) ISR of iliofemoral venous stents is uncommon over the short term. The three major risk factors appear to be presence of thrombotic disease, positive thrombophilia test results, and stent extending below the inguinal ligament (long stents). Although stented limbs that eventually became occluded during the study demonstrated similar risk factors, no conclusion regarding a cause-effect relationship can be drawn from the present data. Whether late occlusion is due to acute thrombosis or to gradual development of true intimal hyperplasia requires further study. (J Vasc Surg 2004;39:181-8.)**

Stenting of the iliofemoral venous outflow tract is emerging as the preferred treatment of venous outflow obstruction. Several studies have shown encouraging patency rates and clinical results after stenting to treat chronic, nonmalignant obstruction of iliac veins over short-term to intermediate follow-up.[1-4] In general, the patency rate for stents placed in the venous system may be influenced by a variety of factors, such as anatomic location, cause of obstruction, development of in-stent restenosis (ISR), amount of venous inflow, and presence of concomitant diseases. The reasons for occlusion of venous stents are not fully understood, and the long-term effects of a stent in the venous system are unknown. We undertook this study to describe development of ISR in stents placed in the lower extremity outflow tract and to evaluate factors that may influence this process.

From River Oaks Hospital.

Competition of interest: none.

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MATERIAL AND METHOD

Between December 1997 and July 2001, balloon angioplasty and stenting of the venous outflow tract, including the common femoral vein, external and common iliac veins, or inferior vena cava, was performed in 455 limbs with chronic, nonmalignant obstruction. Limbs with acute deep vein thrombosis were excluded, and no patients had undergone pre-stenting thrombolytic therapy. Stents in 6 patients became occluded in the immediate postoperative period (<30 days). One stent was reopened with catheter-directed thrombolysis, and remained open 3 months later, as observed at venography. The other five limbs remained blocked, precluding any possibility of assessment of development of ISR. Data for these limbs were excluded from that aspect of the study, but were included in calculation of patency rates.

A single-plane transfemoral venogram was obtained early (2-3 months) after stenting, again 9 months later, and annually thereafter as routine surveillance. If the patient returned with recurrent symptoms, transfemoral venography was always used to assess the venous outflow tract. ISR was assessed as percentage diameter reduction of patent lumen of the stent on the venogram. The degree of stenosis was measured with a caliper and calculated as *diameter of*

stent − diameter of patent area/diameter of stent × 100 (%). Distribution of stenosis in the stent was also noted. At least one venogram was obtained in 324 limbs (324 of 450, 72%) in 316 patients at 2 to 60 months postoperatively (median, 5 months; mean, 10 ± 9 [SD]). An additional venogram was obtained in 123 limbs, two additional venograms were obtained in 40 limbs, and three additional venograms were obtained in 4 limbs. These 316 patients were included in the study. Their median age was 52 years (range, 14-83 years); 229 were female patients, and 87 were male patients (2.6:1). The right lower limb was treated in 91 patients, the left limb in 217 patients, and both limbs in 8 patients (left-right limb ratio 2.3:1 [225: 99]).

The obstructive lesion was considered postthrombotic (175 limbs in 170 patients) if the patient had a history of deep vein thrombosis diagnosed with duplex ultrasound scanning or ascending venography and subsequently treated with anticoagulation, or findings at venography (eg, occlusion, stenosis, collateral vessels) or duplex ultrasound scanning indicative of previous deep vein thrombosis below the inguinal ligament (direct visualization of thrombus or indirect indication by partial or total inability to compress the vein). The remaining 149 limbs in 146 patients were considered nonthrombotic. Screening for thrombophilia was performed in 261 of 316 patients (267 limbs), and was positive for one or more factors in 103 patients (104 limbs). The screening included determination of levels of proteins C and S, antithrombin III, anticardiolipin immunoglobulin IgM and immunoglobulin G, and homocysteine, and detection of lupus anticoagulant, factor V Leiden, and homocysteine gene and prothrombin gene mutation. The test was positive for one or more factors in 74 of 145 postthrombotic limbs (51%), compared with 30 of 122 nonthrombotic limbs (23%; $P < .001$, χ^2 test).

Indications for iliofemoral venous stenting, diagnosis, and clinical results have been discussed in detail elsewhere,[4] and are outside the scope of this article. Technical details of the intervention have been outlined.[5,6] Venous stenting was performed when more than 50% morphologic stenosis was found at transfemoral venography or intravascular ultrasound (IVUS). Crosscut area of stenosis was measured with IVUS before and after stenting and compared with the area of the normal vein below the stenosis.[7]

Wallstents (Boston Scientific, Natick, Mass) were used exclusively in the 324 limbs. Stent diameter was 14 to 16 mm in 81% of limbs (264). In most limbs (187, 58%) one stent was inserted. When two or more stents were inserted (2 stents in 94 limbs, 29%; and 3 to 6 stents in 43 limbs, 13%), the stents were overlapped by at least 1 cm to ensure adequate stent cover of the diseased vein without skipped areas. Median length of the stented vein was 9 cm (range, 4-35 cm). The stent involved the common iliac vein in 307 limbs (95%). In 55% of these 307 limbs (168) stenting was limited to the iliocaval segment; in 33% (102 limbs) the lower part of the stent was in the external iliac vein; and in 12% (37 limbs) the stent reached the common femoral vein. Only two stents were limited to the inferior vena cava, and 15 stents were placed with the upper end in the external iliac vein.

For analysis, the limbs were arbitrarily divided into three groups of similar size, with stent length 4 to 8 cm (n = 100), 9 to 12 cm (n = 108), and 13 to 35 cm (n = 108). The stents reached below the inguinal ligament in 59 limbs, and terminated above the inguinal ligament in 265 limbs. Only 5 of 59 (8%) limbs with stents below the inguinal ligament had short stents isolated to the external and common femoral iliac veins. Most stents placed across the inguinal ligament (54 of 59, 92%) formed the lower part of a long stent originating in the common iliac vein or the inferior vena cava. Consequently the median length of stented vein was longer in this group compared with the group with stents ending above the inguinal ligament (18 cm [range, 4-35 cm] vs 9 cm [range, 4-29 cm], respectively; $P < .001$). The rate of postthrombotic limbs was greater in those with stenting across the inguinal ligament compared with above the inguinal ligament (81% vs 48%; $P < .001$). Similarly, postthrombotic limbs were overrepresented in the group with longer stents (70% in stented vein >12 cm; Table I).

The median transverse lumen area obtained at IVUS during the intervention before balloon angioplasty and stenting was 0.41 cm^2 (range, 0-1.65 cm^2). For analysis, limbs were arbitrarily divided into three groups of similar size, with area 0.0 to 0.30 cm^2 (n = 91), 0.31 to 0.60 cm^2 (n = 68), and greater than 0.61 ^{2} (n = 67), respectively. These three groups had similar distribution of thrombotic and nonthrombotic limbs. After the procedure, median transverse lumen area was 1.70 cm^2 (range, 0.65-2.85 cm^2). Similarly, these limbs were also divided into three groups, with area less than 1.5 cm^2 (n = 71), 1.50 to 1.85 cm^2 (n = 80), and greater than 1.85 cm^2 (n = 70), respectively.

Statistical analysis was performed with GraphPad Prism version 3.00 for Windows (GraphPad Software, San Diego, Calif). To compare groups, a paired or unpaired nonparametric Wilcoxon rank test was used as appropriate. Rates for primary patency, assisted primary patency (patency after preemptive intervention), and secondary patency (patency after intervention for occlusion), as defined by the reporting standards of The Society for Vascular Surgery/International Society for Cardiovascular Surgery (ISCVS),[8] were calculated with survival analysis with the Kaplan-Meier method. Cumulative survival curves were also used to analyze and compare development of in-stent restenosis. The log-rank test was used to assess significance between survival curves and to identify trends of multiple curves. $P < .05$ was considered significant.

RESULTS

Thirteen of 324 limbs (4%) became occluded 2 to 30 months (median, 3 months) after the endovascular procedure. Three limbs were successfully recanalized with catheter-directed thrombolysis. Venograms obtained before and after thrombolysis in these three limbs showed 32%, 33%, and 50% remaining stenosis, respectively. One limb

Table I. Factors that possibly influence in-stent restenosis rate

	Overrepresentation of limbs with thrombotic disease	*Cumulative stenosis rate (%) (>20% stenosis)*	*Cumulative stenosis rate (%) (>50% stenosis)*	*Month*
Gender				36
Male	Yes	52	14	
Female	No	59*	15*	
Side				30
Left	No	47	8	
Right	No	49*	20*	
Thrombotic disease				36
Present	—	63	23	
Not present	—	41†	4†	
Thrombophilia				36
Positive	Yes	66	18	
Negative	No	42†	12‡	
Inguinal ligament				24
Above	No	44	9	
Below	Yes	75†	40†	
Length (cm)				36
2–8	No	42	5	
13–35	Yes	86†	25†	
Pre-stent area (cm^2)				33
0.00–0.30	No	56	15	
>0.61	No	37*	8*	
Poststent area (cm^2)				33
<1.50	No	71	23	
>1.85	No	27§	5*	

Log-rank test: * *NS*, not significant; † $P < .001$; ‡ $P < .05$; § $P < .001$. Frequency of limbs with thrombotic disease was calculated in subgroups and compared with frequency in total limbs (comparison with χ^2 test, $P < .05$ considered significant).

had evidence of increasing ISR before occlusion 18 months after intervention. One limb had a stable ISR of 43% on several venograms before finally becoming occluded after 49 months. No venogram was obtained in eight limbs before or after development of occlusion, and therefore development of ISR could not be assessed. These eight limbs were excluded from grading of in-stent recurrent stenosis; thus 316 limbs were study.

To assess development of ISR, limbs were divided into groups, with no ISR, any degree of ISR, ISR greater than 20% diameter reduction, and ISR greater than 50% diameter reduction. More than 50% recurrence of in-stent stenosis was considered severe, inasmuch as this degree of stenosis was the indication for stenting in the first place. In 13 limbs, only patency could be ascertained, but degree of ISR could not be adequately assessed because of poor quality of venogram. Cumulative ISR rate curves were calculated (Fig 1). At 42 months only 23% of limbs remained ISR-free. However, many had only minimal narrowing. At the same interval, 61% of limbs had greater than 20% diameter reduction and 15% of limbs had >50% diameter reduction. Stenosis in the stent most frequently involved the complete stent uniformly (47%, 143 of 303), sometimes combined with focal stenosis (6%, 18 of 303). The remaining limbs had focal stenosis in the common iliac vein segment (24%, 73 of 303) or the external iliac vein segment (16%, 48 of 303), or at the junction of the two segments (7%, 21 of 303).

Several factors with potential to influence development of ISR were analyzed with cumulative survival (Table I). Gender did not affect outcome. A similar rate of in-stent recurrent stenosis was observed in the left and right lower extremities. In thrombotic limbs ISR developed at a higher rate than in nonthrombotic limbs (Fig 2). At 36 months these rates were 63% and 41% in veins with greater than 20% narrowing, and were 23% and 4% in veins with greater than 50% narrowing in limbs with thrombotic and nonthrombotic disease, respectively ($P < .01$). No difference in in-stent recurrent stenosis rate was found in comparison of a subset of right and left limbs with thrombotic or nonthrombotic obstruction.

In patients positive for thrombophilia, ISR developed more rapidly than in patients with negative test results (Fig 3). At 36 months of follow-up the rates of ISR greater than 20% were 66% and 42%, respectively, in patients with positive and negative test results ($P = .0005$). This difference was maintained with the rate of limbs with severe (>50%) ISR, but was less pronounced (18% and 12%, respectively; $P = .0247$]).

Data concerning length of stented area and placement below the inguinal ligament appear intimately connected, and had a similar effect on development of ISR (Table I; Fig 4). The longest stents and those ending below the inguinal ligament exhibited a rapid increase in ISR during the first 6 months. IRS did not show any preponderance of occurrence at the level of the inguinal ligament. There was a striking difference in the number of limbs with severe stenosis (>50%) at 24 months when limbs with stents terminating above and below the ligament were compared (9% and 40%, respectively; $P < .0001$). Similarly, at 24

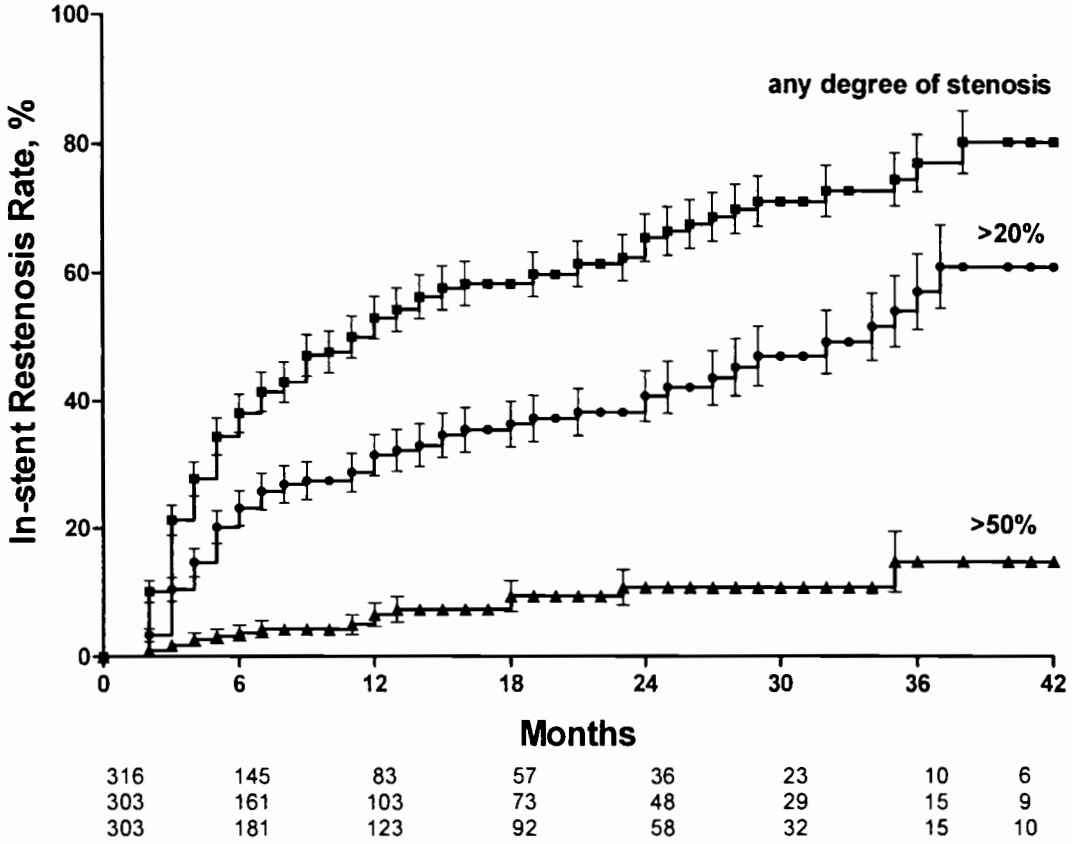

Fig 1. Cumulative in-stent restenosis rates for all limbs with any degree of stenosis, greater than 20% narrowing, and greater than 50% narrowing. *Bottom numbers,* Total limbs at risk for each time interval (SEM <10%).

months postintervention, limbs with the longest stents (>13 cm) had ISR >20% in 74% of limbs, as compared with limbs with intermediate length stents (9-13 cm) and short stents (4-8 cm): 31% and 15% of limbs, respectively). The log-rank test for trend was highly significant ($P <$ .0001). Development of severe ISR (>50%) was similar (Table I). Only 11 of 59 limbs with stents terminating below the inguinal ligament had nonthrombotic causes. At 12 months, there was no difference in cumulative IRS rate. All of these nonthrombotic limbs remained patent during follow-up.

The morphologic cross-cut area of the original obstruction did not appear to influence development of ISR (Table I). ISR was more prevalent with smaller cross-cut area poststenting. At 33 months after intervention, limbs with the smallest lumen (<1.50 cm^2) had ISR greater than 20% in 71% of limbs, as compared with limbs with intermediate lumen (1.50-1.85 cm^2) and large lumen (>1.85 cm^2): 52% and 27%, respectively. The result was mimicked for severe recurrent stenosis (>50%), but did not attain statistical significance. The log-rank test for trend was significant ($P = .0135$).

Groups of limbs with multiple venograms at follow-up without interval interventions were compared to assess progress of ISR over time (Table II). As a group, there was no increase in stenosis over time in limbs with three venograms, although the increase from median degree of stenosis of 9% to 14% was statistically significant in limbs with two venograms. In the intervals between venograms 1 and 2 and venograms 2 and 3, degree of ISR increased by more than 5% in 21% and 13% of limbs, respectively.

Twenty-five secondary endovascular procedures were performed during follow-up. Limbs treated for IRS were excluded from analysis of rate and degree of ISR after intervention, but were included in patency analysis. For the entire population at 3 years, primary patency rate was 75%, assisted primary rate was 92%, and secondary patency rate was 93%. Similarly, values for limbs with nonthrombotic and thrombotic disease at 36 months were 89% and 65%, 100% and 85%, and 100% and 88%, respectively. Nonthrombotic limbs had significantly better patency rate compared with thrombotic limbs ($P = .035$ for primary patency).

It may be of interest to characterize the 13 stented limbs that became occluded during follow-up. All limbs had thrombotic disease, with stent length greater than 13 cm, and 9 of 13 limbs had stents terminating below the inguinal ligament. The iliac veins were completely occluded or had tight, nonyielding stenoses at balloon angioplasty

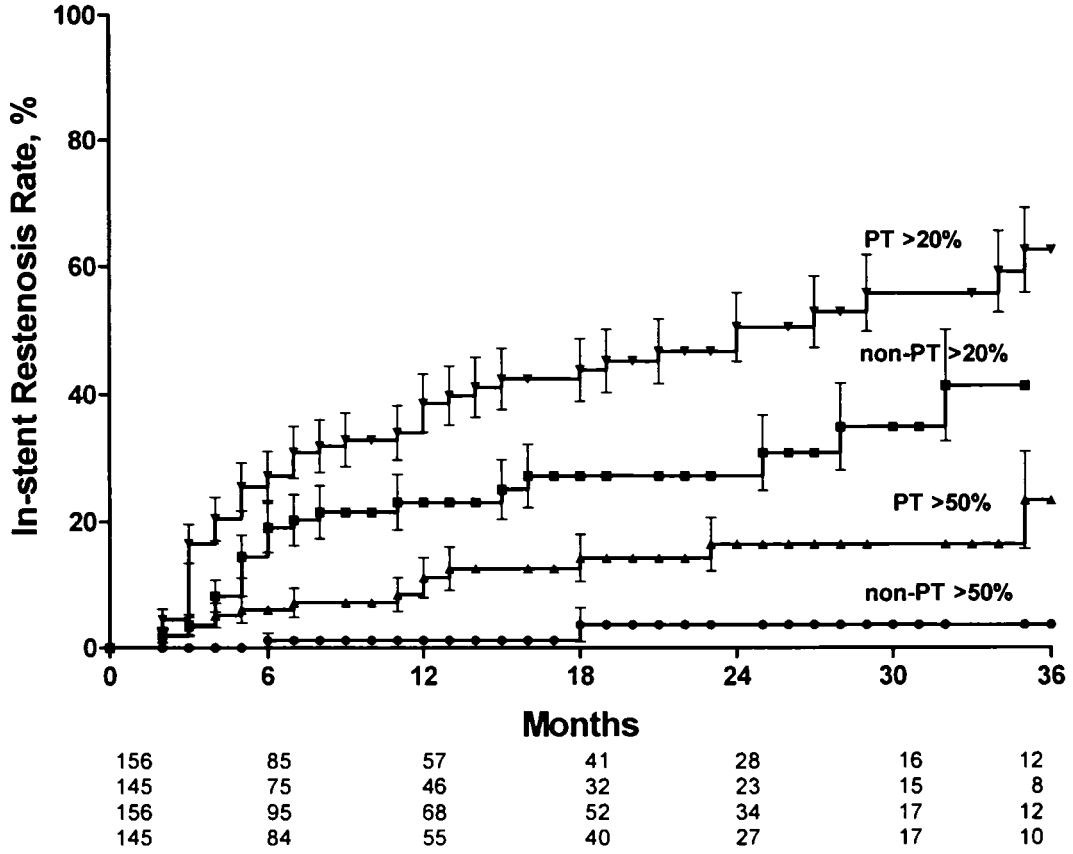

Fig 2. Cumulative in-stent restenosis rates for limbs with thrombotic *(PT)* and nonthrombotic *(non-PT)* disease with greater than 20% and greater than 50% stenosis, respectively. *Bottom numbers,* Total limbs at risk for each time interval (SEM <10%).

Table II. Development of in-stent restenosis in 111 limbs with two subsequent venograms and in 32 limbs with three subsequent venograms

	Venogram 1		*Venogram 2*		*Venogram 3*	
	Median	*Range*	*Median*	*Range*	*Median*	*Range*
111 Limbs						
Months after intervention	5	1–32	16	0–95		
In-stent restenosis (%)	9	0–63	14	0–95*		
32 limbs						
Months after intervention	5	2–31	14	3–37	26	3–60
In-stent restenosis (%)	19	0–31	18	0–65†	18	0–80‡

*P = .0014 compared with venogram 1.
†P = .1763 compared with venogram 1 (not significant).
‡P = .3591 compared with venogram 1 (not significant).

and stenting. Thrombophilia testing yielded positive results in eight patients and negative results in three patients, and was not done in two patients. A similar group of patients also appeared to be at higher risk for development of significant ISR. Limbs with high risk, that is, with thrombotic disease, positive thrombophilia test results, and stent placement below the inguinal ligament (n = 19) were compared with limbs with low risk, that is, without thrombotic disease or stent placement below the ligament and negative for thrombophilia (n = 85). At 24 months postintervention, limbs at high risk had ISR >20% and severe stenosis (>50%) to a greater degree, compared with limbs at low risk (77% and 61% vs 19% and 0%, respectively).

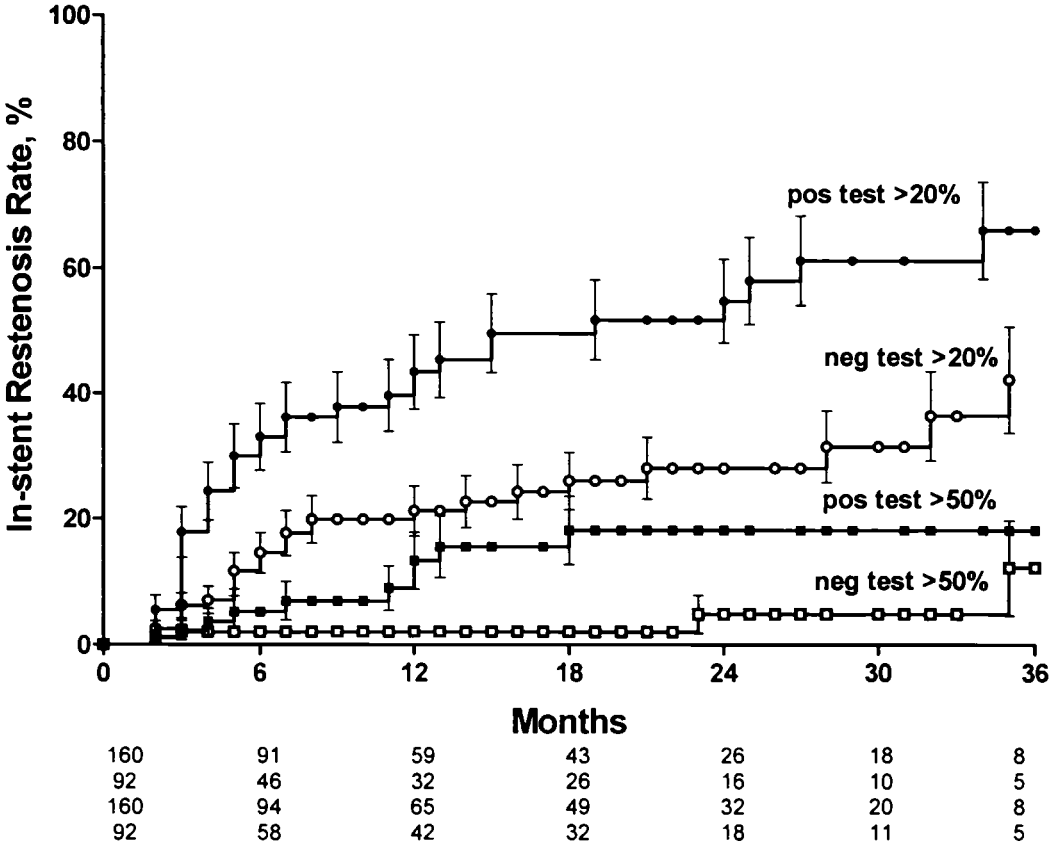

Fig 3. Cumulative in-stent restenosis rates for limbs with greater than 20% and greater than 50% stenosis, respectively, in patients positive and negative for thrombophilia. *Bottom numbers,* Total limbs at risk for each time interval (SEM <10%).

DISCUSSION

To our knowledge, the rate of ISR in stents placed in the venous outflow of the lower extremities to treat chronic, nonmalignant obstruction has not previously been described. In this study ISR developed to some degree in most stents (80% at 42 months). Only a few (17%) had severe (>50%) stenosis during the first 3 years. Measurement of degree of in-stent stenosis as diameter reduction on single-plane venograms may, however, underestimate the degree of intrastent stenosis.[7] The optimal assessment would be to use IVUS to directly measure wall thickness, total area of stenosis, and any vessel remodeling that may have occurred. Cost and logistics prevented regular use of IVUS, and it was performed in only a few patients scheduled for a secondary procedure. The nature of ISR is not known. It may be a lining of thrombosis or a true reaction of the vessel wall, with formation of neointimal hyperplasia as observed in stented arteries.[9] Early in this series, a few patients with early significant ISR underwent thrombolysis in an effort to dissolve any thrombus. These attempts were all unsuccessful. Invariably, balloon angioplasty of in-stent stenosis revealed a very hard stenosis that was difficult to dilate. Any improvement in lumen size appeared to be secondary to remodeling rather than to true decrease in wall thickness. These clinical observations indicate that the reaction to stenting of the vein is possibly a cellular response rather than formation of thrombosis.

As expected, gender and treated limb side did not influence development of ISR. Conversely, the presence of chronic thrombotic disease, as indicated by history or morphologic findings, anywhere in the involved lower extremity resulted in a high rate of ISR. Severe stenosis (>50%) developed in the stent in 23% of these extremities, as compared with 4% of nonthrombotic limbs. This strong impact of thrombotic disease on development of ISR appears to be reflected in the analysis of other potential contributing factors. There was an overrepresentation of limbs with thrombotic disease in patients with thrombophilia, stents terminating below the inguinal ligament, and long stents. Thus a higher rate of ISR was found in patients positive for thrombophilia, which was also more frequently seen in limbs with previous thrombotic disease. The high recurrent stenosis rate in limbs with long stents, especially stents extending below the inguinal ligament, may reflect treatment of more severe and extensive disease demonstrated in limbs with thrombotic disease.[10] A higher rate of chronic thrombotic obstruction was also observed in these limbs.

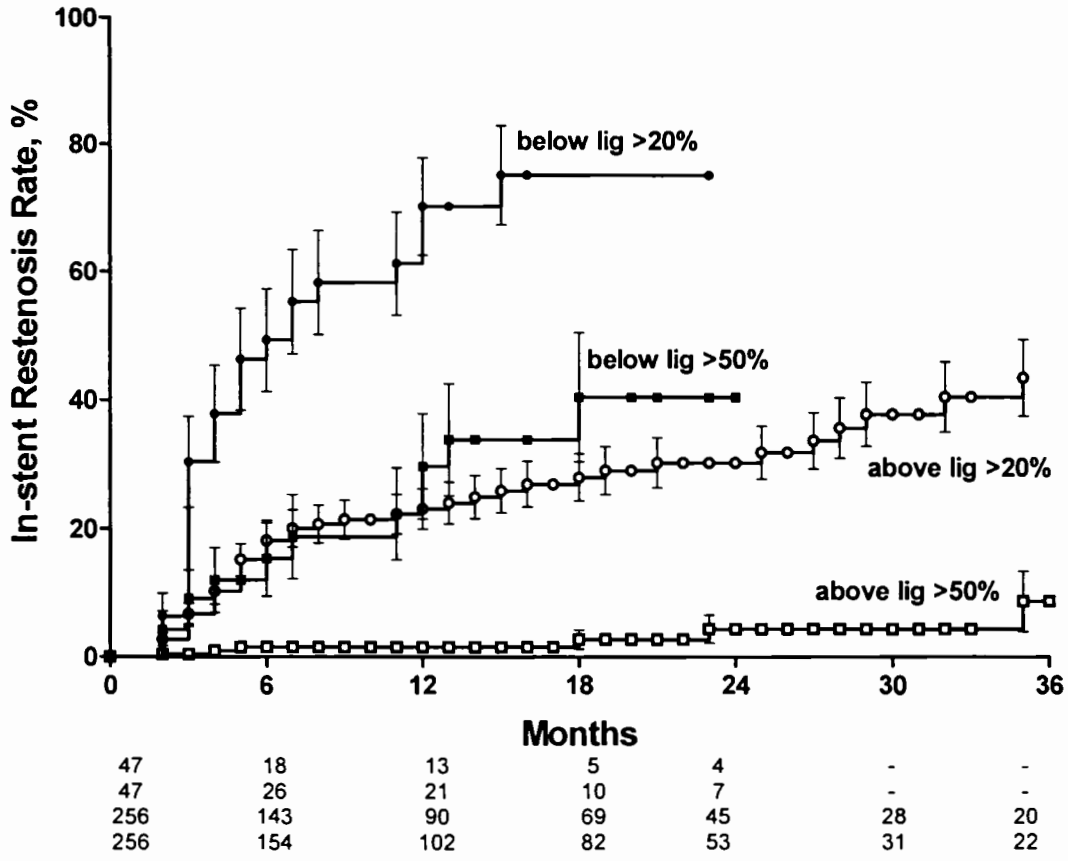

Fig 4. Cumulative in-stent restenosis rates for stents placed below and above inguinal ligament in limbs with greater than 20% and greater than 50% stenosis, respectively. *Bottom numbers,* Total limbs at risk for each time interval (SEM <10%).

It may have been expected that when a narrow vessel is markedly dilated, injury to the vessel wall will be greater and will lead to greater reaction from the stented wall. However, there was no increased rate of recurrent stenosis in stented vessels with tight pre-stenting stenosis. The increased rate of ISR in stents with smaller lumen after dilation may result because the same degree of wall thickening will have a relatively greater effect in a smaller stented vessel compared with a larger vessel. The rate of severe stenosis (>50%), however, was not significantly different.

It is unclear from our data whether there is continuous progress of ISR over time. Despite the fact that groups of limbs with multiple follow-up venograms did not unequivocally exhibit an increase in stenosis, no doubt there is significant progress in individual cases.

The three major risk factors for development of severe ISR appear to be presence of thrombotic disease, positive results of thrombophilia test, and placement of long stents terminating below the inguinal ligament. At 24 months poststenting, limbs with these risk factors demonstrated a 61% rate of severe ISR, whereas no ISR developed in the absence of these risk factors. Although stented limbs that eventually became occluded during this study had similar risk factors, no conclusion regarding a cause-effect relationship can be drawn from the present data. Whether late occlusion occurs as a result of acute recurrent thrombosis or gradual development of true intimal hyperplasia requires further study.

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CORRECTIONS

In: Brown OW, Medicolegal implications of board certification (J Vasc Surg 2003;38:407-8).
The Editors would like to clarify that the author, O. William Brown, MD, JD, is the defendant in the case described in the Editorial.

In: Lauder C, Kelly A, Thompson MM, London NJM, Bell PRF, Naylor AR. Early and late outcome after carotid artery bypass grafting with saphenous vein (J Vasc Surg 2003;38:1025-30).
On page 1030 of the Lauder article, the last line should read, "See Invited Commentary on page 1038."

In: Camiade C, Maher A, Ricco J-B, Roumy J, Febrer G, Marchand C, et al. Carotid bypass with polytetrafluoroethylene grafts: A study of 110 consecutive patients (J Vasc Surg 2003;38:1031-8).
The last line on page 1037 of Camiade et al should read, "See Invited Commentary on page 1038."

In: Myers DD, Hawley AE, Farris DM, Wrobleski SK, Thanaporn P, Schaub RG, et al. P-selectin and leukocyte microparticles are associated with venous thrombogenesis (J Vasc Surg 2003;38:1075-89).
The Myers et al article contains the following errors:
The legend for Fig 5 should read, ". . .shams at day 6 include. . .^CT(23.7 ng/mg)." Under "*CT vs WT, PKO, EPKO P<.05," Fig 5 *B* should include, "**WT vs PKO, EPKO P<.01."
A double asterisk should appear above the column PKO(n5) in Fig 6 *A*.
On page 1084, column one, the text should read, "PKO animals demonstrated significantly reduced levels of E-selectin at day 2 compared with WT ($P<.01$) and nonsignificant reduced levels at day 6 (706 ± 64 vs 1168 ± 312 pg/mg total protein; $P=.15$, ns).
On page 1085, column 2, the text should read, "However, Ab-PSGL-1 did result in a statistically significant decline at day 2 (33 ± 2 to 23 ± 1 ng/mg total protein, $P < .01$) and no decline at day 6 (24 ± 5 to 26 ± 2 ng/mg total protein, ns).
An incorrect version of Fig 8 *B* was submitted for publication. The following is the correct figure: