Do preoperative finger pressures predict early arterial steal in hemodialysis access patients? A prospective analysis

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Background: **Hand ischemia resulting from arterial steal is a serious complication in patients undergoing hemodialysis access, but specific risk factors for steal remain in dispute. The purpose of this study was to determine whether plethysmographically derived finger pressures (FPs) or digital-brachial indices (DBIs) are predictive of symptomatic arterial steal.**

Methods: **We prospectively studied 72 patients (37 men, 35 women; mean age, 57 ± 10 years) who were undergoing brachial artery–based hemodialysis access. All patients had complete pre- and postoperative hand examinations and FP determinations. Surgeons were blinded to preoperative FP results.**

Results: **Prosthetic graft was used in 60 patients (6-mm polytetrafluoroethylene [PTFE] in 50, tapered PTFE in 10), and brachial-based arteriovenous fistulas were created in 12. Fourteen (19%) patients developed arterial steal symptoms. The mean preoperative FP was significantly lower in steal patients than in those without steal (131 ± 27 vs 151 ± 31 mm Hg, $P < .03$). Nine (64%) of the patients with steal had DBIs <1.0, compared to 18 (31%) of the patients without steal ($P = .02$). However, there was no absolute FP or DBI threshold below which steal was inevitable. The occurrence of steal was attributed to proximal arterial stenoses in seven, to distal arterial disease in five, and was unknown in two. When comparing the 14 patients who developed steal to the 58 who did not, we noted that a higher proportion of steal patients had coronary artery disease (57% vs 17%, $P = .005$). Steal was more likely to develop in patients with arteriovenous fistulas than in patients with prosthetic grafts (43% vs 14%, $P = .009$). There were no significant differences in demographic factors, atherosclerotic risks (diabetes, smoking, hypertension, dyslipidemia), prevalence of peripheral vascular disease, cerebrovascular disease, shunt location, tapered vs straight graft, or number of prior grafts placed.**

Conclusions: **These data indicate that preoperative FPs are lower in patients who develop steal syndrome after hemodialysis access. Patients with preoperative DBIs <1.0 are more likely to develop steal, but there is no DBI threshold below which steal is inevitable. Steal is more likely in patients undergoing brachial-based arteriovenous fistulas than in those receiving prosthetic grafts. (J Vasc Surg 2002;36:351-6.)**

The onset of hand ischemia is a devastating complication of upper extremity hemodialysis access. Arteriovenous shunts are nearly always associated with some degree of reduced arterial flow to the distal circulation,[1-3] but severe hemodynamic changes leading to ischemic neuropathy can affect up to 8% of patients.[3,4] Left untreated, this so-called arterial steal syndrome can progress to weakness, ulceration, and gangrene.[5,6] A number of techniques have been proposed to correct arterial steal following hemodialysis access,[7,8] but none have been uniformly successful. Prevention remains the ideal solution for this problem. Identification of risk factors might lead to avoidance of arm access in patients likely to develop steal, but specific risk factors remain in dispute.[3-6]

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Most patients who develop steal syndrome have angiographic evidence of arterial stenoses affecting the circulation proximal or distal to the arterial anastomosis.[3,9,10] Previously asymptomatic patients may develop ischemic symptoms as the high flows associated with arteriovenous shunts "unmask" these lesions. Blood pressure differentials often detect arterial stenoses in the circulation proximal to the brachial artery. We hypothesized that subtle hemodynamic changes may be detectable in the distal circulation of asymptomatic patients who are predisposed to develop arterial steal. Photoplethysmographically derived finger pressures (FPs) represent a simple noninvasive study that may discern minor pressure decreases in the hand and forearm circulations. The purpose of this study was to evaluate whether FP could be used to identify patients at risk for arterial steal.

PATIENTS AND METHODS

This study was approved by the institutional review boards of The University of Texas Southwestern Medical Center and the Dallas Department of Veterans Affairs Medical Center. All patients who underwent brachial artery–based hemodialysis access between July 1998 and July 2001 were considered for enrollment. Following informed

consent, patients underwent complete history taking and detailed hand examinations. All patients were evaluated for the presence of atherosclerotic risk factors (history of smoking, hypertension, diabetes mellitus, and dyslipidemia), clinically overt coronary artery disease (CAD: angina, documented myocardial infarction, or prior coronary revascularization), peripheral arterial occlusive disease (PAOD: claudication, ischemic rest pain, prior revascularization, or amputation for ischemia), and cerebrovascular disease (transient ischemic attacks or stroke). Medical records were reviewed to determine previous symptoms of arterial steal following hemodialysis access. The cause of renal failure was documented, as were the duration of hemodialysis, site of prior indwelling venous catheters, and number and location of previous access.

Hand examinations included assessment of sensory function using light touch, two-point discrimination, and sharp-dull stimuli. Motor examination included assessment of the radial, median, and ulnar nerves. Doppler-derived brachial artery blood pressures were determined in both arms, and FPs were measured by using photoplethysmography (Model 2100 Flo-Lab, Parks Medical Electronics, Inc, Aloha, Ore). Mean FP was calculated after excluding the highest and lowest recorded FP values, and then averaging the remaining three FP. Digital-brachial indices (DBIs) were calculated by dividing the mean FP by the highest recorded brachial artery pressure.

Surgeons were blinded to the results of FP and chose the access site on the basis of routine clinical data. Patients who underwent creation of hemodialysis access by using the brachial artery as inflow were eligible for this study, regardless of the location, configuration, or use of prosthetic graft vs autogenous vein. Follow-up included postoperative hand examinations and repeated FP determinations. Each subject underwent a single postoperative FP determination on the side ipsilateral to the hemodialysis access at the time of the first postoperative visit, usually within 3 weeks of the operation. The presence of steal was documented by a reduction of mean FP of $>$ 20 mm Hg compared to the preoperative value in a patient with symptoms of ischemia (pain, paresthesias, or gangrenous changes) in the hand ipsilateral to the access. Confirmation required improvement in symptoms and an increase in mean FP with temporary graft compression. An attempt was made to document the cause of steal in every case by using arteriography.

Patients undergoing hemodialysis access that used inflow from arteries proximal or distal to the brachial artery were excluded from the analysis. Patients were also excluded if follow-up studies were not performed for any reason or if the hemodialysis access was thrombosed at the time of the first postoperative visit.

Statistics. Continuous data are expressed as mean ± standard deviation. Statistical comparisons between categorical parameters were performed using one-tailed Fisher exact test, and comparisons between groups of unpaired data were made using Student *t* test. Differences were considered to be statistically significant at the $P < .05$ level.

Table I. Demographics for the 72 study subjects

Male	37 (51)
Mean age (y)	57 ± 10
Race	
White	20 (28)
African American	37 (51)
Hispanic	15 (21)
Smoking	22 (31)
Hypertension	69 (96)
Diabetes mellitus	48 (67)
Dyslipidemia*	25 (35)
Cause of renal failure	
HTN	25 (35)
DM	10 (14)
Both DM and HTN	31 (43)
Other	6 (8)

Data are n (%) unless indicated otherwise.
DM, Diabetes mellitus; *HTN*, arterial hypertension.
*Receiving lipid-lowering medication or serum cholesterol > 240 mg/dL, triglycerides > 350 mg/dL.

RESULTS

A total of 108 patients underwent brachial artery-based hemodialysis access during the study period. Thirty-four patients were excluded from the analysis because they did not keep follow-up appointments, and two others were excluded because they had thrombosed grafts at the time of the first postoperative visit. The remaining 72 patients (37 men, 35 women) met the inclusion criteria during the study period. Demographics, atherosclerotic risk factors, and cause of renal failure for the 72 study subjects are shown in Table I.

The mean duration of hemodialysis in the study group was 6 ± 13 months (range, 0-84 months; median, 1 month). Twenty-nine (40%) subjects had previous arm access, including 21 with one previous access, four with two previous accesses, and four with three or more. Sixteen (22%) had prior access on the side ipsilateral to the new access. The mean preoperative FP was 148 ± 31 mm Hg (range, 59-224 mm Hg), and the mean preoperative DBI was 0.99 ± 0.15 (range, 0.54-1.3). Twenty-seven patients (38%) had DBI $<$ 1.0, including 19 subjects with DBI between 0.8 and 0.99; seven with DBI between 0.6 and 0.79; and one with DBI $<$0.6. No neuromuscular abnormalities were detected by detailed hand examination in any patient. None of the subjects had a difference $>$20 mm Hg between brachial blood pressures.

Prosthetic graft was used in 60 (83%) patients, including 6 mm polytetrafluoroethylene (PTFE) in 50 and tapered 4-to-7 mm PTFE in 10. Prosthetic grafts were placed as forearm loop configuration in 50 and as straight upper arm configuration in 10. Autogenous, brachial-based arteriovenous fistulas were created in the other 12 (17%).

The mean postoperative FP for all 72 patients was 94 ± 45 mm Hg (range, 0-186 mm Hg), and the mean DBI was 0.65 ± 0.29 (range, 0-1.3). Fourteen patients (18%) developed symptomatic steal during the postoperative period. Symptoms ranged from digital paresthesias (n = 3) to

Table II. Underlying causes and interventions used to correct ischemia in 14 patients who developed arterial steal

Patient no.	*Cause*	*Graft type*	*Intervention*	*Graft salvage*
1	Subclavian stenosis	V	PTA and stent	Yes
2	Axillary stenosis	V	PTA	Yes
3	Axillary stenosis	P	PTA	Yes
4	Mid-brachial stenosis	P	Endarterectomy, vein patch angioplasty	Yes
5	Brachial stenosis just proximal to graft	V	DRIL	Yes
6	Diffuse brachial artery disease	V	Graft ligated	No
7	Diffuse brachial artery disease	P	Graft ligated	No
7	Distal ablation	V	DRIL	Yes
8	Distal ablation	P	DRIL	Yes
9	Distal ablation	V	Graft banded	No
10	Distal ablation	P	Graft ligated	No
11	Distal ablation	P	Graft ligated	No
12	Distal ablation	P	Graft ligated	No
13	Unknown	P	Graft ligated	No
14	Unknown	P	Graft ligated	No

Distal ablation refers to forearm arterial disease.
DRIL, Distal revascularization-interval ligation; *P*, prosthetic graft; *PTA*, percutaneous balloon angioplasty; *V*, autogenous, brachial-based arteriovenous fistula.

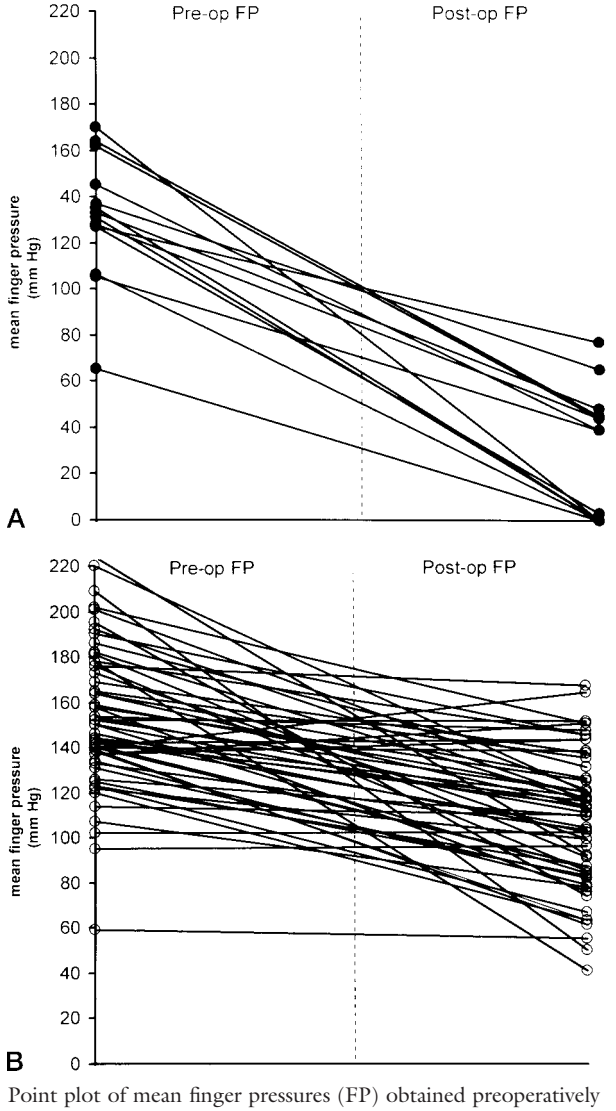
severe hand pain and weakness (n = 11); improvement occurred in every case with graft compression. None of the 58 subjects without steal symptoms had evidence of motor or sensory neural dysfunction, and none developed digital ulceration or gangrene. The mean postoperative FP was 29 ± 27 mm Hg in patients with steal and 110 ± 33 mm Hg in patients without steal.

All 14 patients with steal required interventions to relieve symptoms. The time to intervention depended on the degree of symptoms. The three patients with digital paresthesias were not improved after a mean observation period of 4.5 months. Severe hand pain prompted more rapid intervention (2-6 weeks) in the other 11 patients. The type of intervention depended on the cause of steal. Arteriography was performed in 12 of 14 patients (two refused): steal was attributed to proximal stenoses in seven (three subclavian/axillary, four proximal brachial artery) and to distal arterial disease in five (Table II). Three of the seven patients with proximal disease underwent successful endovascular intervention (angioplasty/stent) with resolution of steal symptoms and graft salvage; two underwent successful open repair, one underwent graft banding, and one required graft ligation for severe ischemia. The single graft that was banded thrombosed within 48 hours. Two of the five patients with distal disease underwent successful distal revascularization-interval ligation (DRIL) procedures with graft salvage, and three underwent graft ligation. The two who did not undergo arteriography also underwent graft ligation. Steal symptoms were resolved in all patients following these procedures. In all, access grafts were salvaged in seven (50%) of the steal patients (Table II). No patient required amputation in this series.

Evaluation of potential risk factors. The mean preoperative FP of patients with steal was 131 ± 27 mm Hg, which was significantly lower than 151 ± 31 mm Hg for patients without steal ($P < .03$; Fig). Nine (64%) of the steal patients had a DBI < 1.0, compared to 18 (31%) of the patients without steal ($P = .02$). However, the difference in DBI between the two groups did not reach statistical significance (0.93 ± 0.18 vs 1.0 ± 0.14, $P = .08$). There was no absolute threshold for FP or DBI below which development of steal was inevitable. By using a threshold value of DBI < 1.0 to predict steal, we determined a sensitivity of 64% and a specificity of 69%; the positive predictive value was 33%, and the negative predictive value was 89%. For DBI <0.8, the sensitivity was 29%, the specificity was 93%, the positive predictive value was 50%, and the negative predictive value was 84%.

Upon comparing the 14 patients who developed steal to the 58 patients who did not, we noted a higher proportion of steal patients with CAD ($P = .005$; Table III). Steal patients tended to have a higher incidence of PAOD (29% vs 12%), prior indwelling central venous lines (79% vs 62%), and prior steal (14% vs 1%). However, these differences did not reach statistical significance (Table III). There were no statistically significant differences in demographic factors, atherosclerotic risk factors, cerebrovascular disease, or duration of hemodialysis (Table III). Eight (57%) steal patients had prior hemodialysis access grafts (two ipsilateral), which was not significantly different compared to 21 (36%) patients without steal who had prior grafts (14 ipsilateral) ($P = .13$).

Steal developed in six (50%) of the 12 patients who had autogenous, brachial-based arteriovenous fistulas and in eight (13%) of the 60 patients who had brachial-based prosthetic grafts ($P < .001$). The mean preoperative FP was 119 ± 32 mm Hg in the 12 patients with autogenous fistulas and 153 ± 28 mm Hg in the 60 patients with prosthetic grafts ($P = .009$). Nine (75%) patients with autogenous fistulas and 20 (33%) patients with prosthetic grafts had prior access procedures ($P = .009$).



Point plot of mean finger pressures (FP) obtained preoperatively and postoperatively in 14 patients who developed steal (**A**), and 58 patients who did not develop steal (**B**) after hemodialysis access.

Steal developed in seven (14%) of the 50 patients who underwent placement of forearm loop grafts and none of the 10 patients with straight upper arm grafts; this difference was not statistically significant. Steal developed in one (7%) patient with a tapered graft, which was not significantly different compared to seven (14%) patients with prosthetic grafts that were not tapered.

DISCUSSION

In 1997, we reported 18 patients who presented with arterial steal syndrome after hemodialysis access, five of who underwent arteriograms that demonstrated correctable inflow stenoses.[10] It is gratifying that none of the 18 patients experienced tissue loss, but the majority experienced graft thrombosis after attempts at banding or other procedures

Table III. Comparison between subjects who developed steal and those who did not

	Steal (n = 14)	*No steal (n = 58)*	P *value*
Female	7 (50)	28 (48)	.86
Age ≥ 60 y	9 (64)	24 (41)	.21
Smoking	3 (21)	19 (33)	.67
Diabetes	9 (64)	39 (54)	.50
Hypertension	14 (100)	55 (95)	.90
Dyslipidemia	5 (36)	19 (26)	.56
Duration of HD	10 ± 16 mo	5 ± 12 mo	.17
≥2 ips. op.*	2 (14)	14 (24)	.66
Prior steal	2 (14)	1 (2)	.17
CAD	8 (57)	10 (17)	.005
PAOD	4 (29)	7 (12)	.11
CVD	2 (14)	6 (10)	.50
AVF	6 (43)	6 (10)	.009
Tapered graft†	1 (7)	9 (16)	.41

Data are n (%) unless indicated otherwise.
AVF, Brachial-based arteriovenous fistula; *CAD*, coronary artery disease; *CVD*, cerebrovascular disease; *HD*, hemodialysis; *ips. op.*, ipsilateral operation; *PAOD*, peripheral vascular disease; *PTFE*, polytetrafluoroethylene.
*Two or more previous access attempts on the ipsilateral limb.
†Tapered 4- to 7-mm PTFE graft.

to correct the steal. We have since seen a number of patients from other institutions with advanced complications of arterial steal, including digital gangrene and hand amputations. These individuals would have fared better if upper extremity access had been avoided altogether, but there were no clear signs that they were at risk for steal syndrome in the preoperative period. In fact, there are no proven risk factors for steal. Published studies have implicated diabetes mellitus and smoking,[5,6,11] but others have shown no difference in the incidence of smoking and diabetes in patients with steal vs those without steal.[3] Peripheral vascular disease has also been suggested as a risk factor for development of steal.[5] PAOD was not a significant risk factor for steal in the present study, probably because many patients with overt vascular lesions were excluded on the basis of unequal arm blood pressures. CAD was significantly more frequent in patients with steal, however. This suggests that, compared to lower extremity involvement, atherosclerosis in the smaller coronary vessels may be a more important marker for atherosclerosis in the upper extremity circulation.

The purpose of the present study was to evaluate a simple noninvasive test that could distinguish patients who might be at risk for steal after hemodialysis access placement. Although patients with steal had lower preoperative FP compared to patients who did not develop steal, there was no threshold below which steal was inevitable. Patients with DBI < 1.0 were more likely to develop steal, but this was not universal. In fact, this threshold had a relatively low sensitivity and specificity; lowering the threshold to .8 increased the specificity but reduced the positive and negative predictive values. Nevertheless, because unsuspected inflow lesions accounted for steal in half of the patients in the present study, it seems reasonable to screen patients

with DBI <1.0 for inflow lesions with a noninvasive study such as duplex scanning. Although all lesions may not be identified with duplex scanning, five of our nine patients with DBI <1.0 who developed steal had inflow lesions that should have been detectable.

It is somewhat surprising that half of our patients with steal had inflow lesions identified on arteriography, especially given that preoperative brachial blood pressures were equal. This unexpected finding may be the result of our rigorous search for the cause of steal in the 14 patients. In our previous study of 18 patients with steal, all five patients who underwent arteriography had inflow lesions measuring at least 50% diameter loss in the donor circulation.[10] It should be pointed out that four of the five had equal brachial blood pressures documented before access placement. We believe that inflow lesions account for a larger proportion of patients with steal syndrome than previously reported, probably because treatment has focused on reduction of shunt diameter (ie, banding) rather than correction of the underlying problem. Others have reported that inflow stenoses account for steal in up to 30% of patients, confirming that angiography remains an important part of evaluating steal syndrome.[2,9] Unfortunately, the presence of symmetrical brachial pressures does not exclude the possibility of occult inflow lesions. Calligaro et al[12] have reported equal blood pressures in some patients with significant stenoses in the axillosubclavian arterial circulation. Crawford et al[13] reported that one fourth of patients with chronic upper extremity ischemia had symmetrical brachial pressures. We believe that the high flows associated with the arteriovenous shunt circuits unmasked these inflow lesions.

Goff et al[3] have shown that a DBI <0.6 identifies a patient at risk for steal syndrome. We cannot dispute this because only one of our patients had a DBI <0.6. We believe that some patients with lower DBI may have been excluded in the present study because they had symptoms of upper extremity ischemia or brachial blood pressure discrepancies. In the rare asymptomatic patient with DBI <0.6, we would recommend screening for inflow lesions. It should be pointed out that the single patient with DBI <0.6 in this study did not develop steal (Fig 1).

The calculated incidence of steal in this study is higher than previously reported.[4,5,9] We acknowledge that our reported incidence is probably overestimated, given that there were 34 additional patients who underwent prosthetic graft placement during the study period who did not return for follow-up FP determinations. Assuming that these patients did not have steal, the recalculated incidence among patients with prosthetic graft would be 8% (14/106), which is in the range reported by others.[3,4] Much of the increased incidence of steal in the present study can be attributed to the use of brachial-based arteriovenous fistulas, which accounted for 43% of the steal cases. The difference between vein and prosthetic grafts is particularly pronounced if one considers only the upper arm conduits, which otherwise should have similar anatomy and hemodynamics: steal occurred in six of 12 vein patients and none of 10 upper arm graft patients. Although these findings may have important implications for National Kidney Foundation—Dialysis Outcomes Quality Initiative guidelines, it should be noted that there were important differences between the patients with autogenous fistulas and those with prosthetic grafts, including lower mean FP and greater proportion with prior grafts in the former group. An in-depth hemodynamic analysis is beyond the scope of this study, but we speculate that at least part of the difference in the incidence of steal is because of lower resistance in the vein conduits. The exaggerated hemodynamic changes may be result in a tendency to shunt blood from the periphery. Since four of the six patients with arteriovenous fistulas who developed steal were women, we speculate that arterial size is another possible contributing factor.

Another possible explanation for our higher reported incidence of steal is related to our definition. "Steal syndrome" covers a wide spectrum of clinical scenarios, ranging from an asymptomatic decrease in finger pressures to advanced ischemia with gangrene. We intentionally based our definition on three factors: the presence of symptoms, evidence of a hemodynamic flow reduction (reduction of mean FP >20 mm Hg), and improvement with graft compression (to distinguish from neuropathy). The presence of symptoms was considered necessary to distinguish from "asymptomatic" steal. We concede that a reduction of 20 mm Hg is somewhat arbitrary in defining hemodynamic significance. However, we feel that this threshold represents clear evidence of hemodynamic flow reduction, whereas smaller values might be open to dispute. Although relief of symptoms during graft compression has not been evaluated for reliability, we feel that it helped to distinguish steal syndrome from primary neurogenic problems. Our results suggest that this definition was adequate to distinguish patients with steal from those who did not. Because all 14 patients ultimately required interventions to control severe or persistent symptoms, we feel that our definition was not overly sensitive.

Despite being a relatively large prospective study of patients undergoing hemodialysis access, there were limitations. First, there is a relatively limited period of formal follow-up. We cannot determine the long-term outcome of our interventions to correct steal, nor do we know whether there is a correlation between FP or DBI and long-term patency in patients who do not develop steal. Nevertheless, the present data gives some indication of the limited usefulness of FP and DBI in patients undergoing hemodialysis access to predict early steal, as well as associated risk factors such as CAD and use of brachial-based arteriovenous fistulas.

A second limitation is that the natural history of arterial steal remains ill-defined. We intervened in all patients with steal, regardless of the severity of symptoms. It is possible that some patients would have improved spontaneously as the arterial circuit dilated and extremity flows increased. However, given the poor medical condition of these patients, their tendency to miss follow-up appointments, and

the grave consequences of untreated steal in some individuals, we did not think that it was ethical to postpone intervention. Although some surgeons might have recommended banding as the procedure of choice in the steal patients, we have generally abandoned this technique because of the high incidence of graft thrombosis.[10]

A third limitation is that some segments of the dialysis population may be underrepresented by the present study group. Specifically, the high incidence of diabetes mellitus may have prevented analysis of this condition as a risk factor for steal. However, the study population came from three hospitals in our institution and represented a diverse socioeconomic group. The increasing incidence of diabetes mellitus in the U.S. dialysis population has recently been acknowledged.

Finally, the relatively small number of patients who developed steal in this study may have prevented identification of important differences between the two groups. We acknowledge the risk of a type II error in the evaluation of risk factors such as PAOD, duration of hemodialysis, and number of prior grafts. However, we do not believe that this should detract from the main findings of this study.

In summary, the present data indicate that preoperative FPs are significantly lower in patients who develop arterial steal syndrome after hemodialysis access. Patients with DBI < 1.0 are statistically more likely to develop steal, but there is no DBI threshold below which steal is inevitable. Although DBI < 1.0 is associated with substandard positive and negative predictive values, the high incidence of inflow lesions in our steal patients suggests that it may useful to indicate which patients should undergo evaluation of the inflow circulation by noninvasive means. Regardless of the preoperative FP or DBI, there was a higher incidence of steal after autogenous, brachial-based arteriovenous fistulas in this study. This group deserves consideration for more careful preoperative screening if the DBI if <1.0.

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