Original Report: Patient-Oriented, Translational Research

American Journal of Nephrology

Am J Nephrol 2003;23:403–408
DOI: 10.1159/000074297

Received: May 7, 2003
Accepted: July 22, 2003
Published online: October 17, 2003

Vascular Access Surveillance: Evaluation of Combining Dynamic Venous Pressure and Vascular Access Blood Flow Measurements

Heidi Hoeben[c] Ali K. Abu-Alfa[a] Robert F. Reilly[a] John E. Aruny[b]
Koenraad Bouman[c] Mark A. Perazella[a]

Departments of [a]Medicine and [b]Radiology, Yale University, New Haven, Conn., USA;
[c]Department of Nephrology, Middelheim Hospital, Antwerp, Belgium

Key Words
Vascular access surveillance · Arteriovenous fistula · Arteriovenous graft · Thrombosis, vascular access · Access blood flow · Venous pressure · Angioplasty

Abstract
Background/Aims: Vascular access thrombosis is one of the most morbid problems encountered by hemodialysis patients. Surveillance protocols utilizing venous pressure (Vp) and vascular access blood flow (VABF) measurements have been employed to preserve vascular access. We undertook a study to evaluate combined dynamic Vp and VABF measurements in the identification of vascular access impairment. We also assessed the effect of preventive repair on thrombosis rates in impaired vascular accesses identified by surveillance. ***Methods:*** Eighty-six chronic hemodialysis patients with a functioning vascular access were enrolled into the surveillance protocol. All vascular accesses with greater than 50% of monthly Vp readings >120 mm Hg or VABF <500 ml/min in arteriovenous fistulas (AVFs) and VABF <650 ml/min in arteriovenous grafts (AVGs), or a decrease in VABF >25% compared to the highest previously measured value, were considered positive. Stenosis >50% on fistulography or a thrombotic event were defined as a 'vascular access impairment episode' while a stenosis <50% or the absence of a thrombotic event was defined as 'no vascular access impairment episode'. Thrombosis rates and intervention rates were calculated per access year at risk. ***Results:*** The sensitivity and specificity of the combined surveillance protocol for AVFs were 73.3 and 91%, respectively. In AVGs, they were 68.8 and 87.5%, respectively. The rate of thrombotic events was lower in patients who underwent early repair. The addition of dynamic Vp did not reduce the thrombosis rate any further than surveillance based on VABF alone. ***Conclusion:*** Combined monitoring for surveillance of AVFs improved sensitivity but had little benefit in AVGs over VABF monitoring alone. Raising VABF cutoff levels might increase and improve identification of vascular access risk for thrombosis, but at the expense of lower specificity.

Introduction

Hemodialysis vascular access thrombosis is the most common cause of access impairment [1]. Early studies addressing this problem demonstrated that uncorrected stenosis is ultimately associated with thrombosis [2–4]. A few studies have suggested that preventive repair of stenotic lesions decreases thrombosis and may increase the longevity of the vascular access [4–6]. It is therefore logical that prospective surveillance protocols aimed at the detection and subsequent correction of these stenotic lesions have been developed. Venous pressure (Vp) mea-

KARGER
Fax + 41 61 306 12 34
E-Mail karger@karger.ch
www.karger.com

0250–8095/03/0236–0403$19.50/0

Accessible online at:
www.karger.com/ajn

Mark A. Perazella, MD, FACP
Associate Professor of Medicine, Section of Nephrology
Yale University School of Medicine, LMP 2071, 333 Cedar Street
New Haven, CT 06520-8029 (USA)
Tel. +1 203 785 4184, Fax +1 203 785 4904, E-Mail mark.perazella@yale.edu

surement (static or dynamic) is one form of surveillance that has been shown to be useful in identifying arteriovenous grafts (AVGs) with stenosis [2, 7]. However, Vp measurements are an insensitive predictor of failing native arteriovenous fistulas (AVFs) [3]. More recently, the ability to measure vascular access blood flow (VABF) employing the ultrasound dilution technique has improved our ability to identify vascular accesses at risk for thrombosis [8, 9]. In several studies, VABF was an effective surveillance tool to identify patients at risk of developing thrombosis in both AVGs and native AVFs [9–14]. Moreover, it has clearly been shown that screening based on VABF is superior to Vp monitoring in reducing the thrombosis rate in AVGs and AVFs [15, 16]. In AVGs, static Vp monitoring appears to be equivalent to VABF in reducing the thrombosis rate [15]; however, this finding has not been confirmed in other studies [13]. In fact, one publication suggested that VABF measurements and dynamic Vp measurements did not correlate [17]. In light of these data, we implemented a surveillance protocol based on the combination of dynamic Vp and VABF in AVGs and AVFs. Our objective was to assess the combination of these two methods as compared with either alone in the detection of access impairment and the prevention of access thrombosis.

Patients and Methods

Vascular Access Protocol

A vascular access surveillance protocol was initiated on July 1 2000 to identify vascular accesses at risk for impairment (stenosis >50% by fistulography or access thrombosis) and to reduce the thrombosis rate by intervening with early repair (angioplasty alone, angioplasty plus stenting or surgical revision). The surveillance protocol was based on the combination of dynamic Vp measurements (Cobe Centry 3 hemodialysis machines) and VABF measurements. Dialysis machine Vp measurements were recorded at a blood flow rate of 200 ml/min during the first 5 min of each hemodialysis session using 15-gauge needles, as previously described [7]. VABF was measured with the ultrasound dilution technique (HD01 Monitor, Transonic Systems, Inc., Ithaca, N.Y., USA) at a hemodynamically stable time during the hemodialysis treatment [8]. All VABF measurements were performed in duplicate to assure accuracy. When 2 measurements differed by more than 10%, a third measurement was taken and the average of the measurements was calculated. The VABF was measured monthly in AVGs and bimonthly in AVFs, though some AVFs were also screened monthly. Patients who met positive surveillance criteria were referred for fistulography. Positive criteria were defined as follows: (1) vascular accesses with greater than 50% of monthly Vp readings >120 mm Hg, (2) VABF <500 ml/min in AVFs, (3) VABF <650 ml/min in AVGs, and (4) a decrease in VABF >25% compared to the highest previously measured value in AVFs and AVGs [18, 19]. Abnormal VABF measurements were confirmed at the start of one of the following hemodialysis sessions to increase accuracy [20]. Other fistulography referral indications were prolonged bleeding and aspiration of excessive clots during needling of the vascular access. Patients were scheduled for fistulography within 8 weeks following a positive surveillance test. A stenosis >50% on fistulography was considered significant and an indication for angioplasty, stent placement or surgical revision.

Study Design

One hundred and five adult patients with a functioning AVF or AVG as their primary vascular access for at least 3 months were undergoing hemodialysis at the Gambro Healthcare-Yale University Dialysis center during the period of July 1 2000 through March 31 2001. Eighty-six of them were enrolled into the surveillance study. Nineteen patients were excluded from study for the following reasons: refusal to undergo VABF measurements (n = 4), immeasurable VABF (n = 2) or less than 3 months of follow-up (n = 13) due to transplantation (n = 1), death (n = 2) and late entry into the study (n = 10).

Each individual patient was only enrolled once. Demographic data such as age, race, gender and pertinent characteristics were obtained, as well as the time on dialysis, the age of the vascular access and the type of vascular access. All episodes of thrombosis and the number and type of interventions (fistulography, surgical revision or thrombolysis) were recorded. The thrombosis rate and intervention rate for the vascular accesses were calculated as the total number of thrombotic events and interventions per access over the total access years at risk.

The results of the surveillance protocol were evaluated in the following manner. Each surveillance measurement was examined over the following 8 weeks in relation to (1) fistulogram result, (2) thrombotic event or (3) absence of a thrombotic event. Presence of stenosis >50% on fistulography or occurrence of a thrombotic event were defined as a 'vascular access impairment episode', while a stenosis <50% or the absence of a thrombotic event were defined as 'no vascular access impairment episode'. An 8-week period was arbitrarily determined as a practical interval within which evaluation based on the surveillance measurement was undertaken. Every positive surveillance measurement associated with either a stenosis as demonstrated by fistulography or a thrombotic event within 8 weeks met the criteria for a vascular access impairment episode (true positive surveillance measurement). Negative surveillance results associated with either a stenosis on fistulography or a thrombotic event within 8 weeks also met the criteria for a vascular access impairment episode (false negative surveillance measurement). Accesses not associated with either stenosis or thrombosis during the 8 weeks following a positive surveillance measurement (Vp or flow) were considered to have no vascular access impairment episode (false positive surveillance measurement). Accesses not associated with either stenosis or thrombosis during the 8 weeks following a negative surveillance measurement were considered to have no vascular access impairment episode (true negative surveillance measurement). Each test was independently analyzed.

To allow calculation of thrombosis rates, patients were divided into 3 groups based on the following criteria: those who never had positive surveillance criteria (group 1), those who had positive surveillance criteria and underwent an elective repair within 8 weeks (group 2), and those who had positive surveillance criteria but did not always follow the study protocol for repair within 8 weeks (group 3). Patients who developed access thrombosis prior to undergoing repair (within 8 weeks following a positive surveillance test) were

Table 1. Sensitivity and specificity of combined and separate surveillance measurements (VABF, dynamic Vp) in AVFs

Criteria	VA impairment	No VA impairment	
VABF or dynamic Vp[1]			
Positive	22	25	
Negative	8	254	
Total	30	279	309
VABF alone[2]			
Positive	17	16	
Negative	13	188	
Total	30	204	234
Dynamic Vp alone[3]			
Positive	14	9	
Negative	16	258	
Total	30	267	297

VA = Vascular access.
1 Sensitivity: 73.3%; specificity: 91%.
2 Sensitivity: 56.6%; specificity: 92.2%.
3 Sensitivity: 46.6%; specificity: 96.6%.

Table 2. Sensitivity and specificity of combined and separate surveillance measurements (VABF, dynamic Vp) in AVGs

Criteria	VA impairment	No VA impairment	
VABF or dynamic Vp[1]			
Positive	62	30	
Negative	28	209	
Total	90	239	329
VABF alone[2]			
Positive	55	17	
Negative	35	199	
Total	90	216	306
Dynamic Vp alone[3]			
Positive	24	13	
Negative	66	187	
Total	90	200	300

VA = Vascular access.
1 Sensitivity: 68.8%; specificity: 87.5%.
2 Sensitivity: 61.1%; specificity: 92.2%.
3 Sensitivity: 26.6%; specificity: 93.5%.

placed into group 3. Subgroup analysis for the different types of vascular access (AVFs and AVGs) was performed.

Statistical Analysis

The two-tailed, unpaired Student's t test and the χ^2 analysis were used. Statistical significance was reached at $p < 0.05$. Confidence intervals were constructed using 95% boundaries around the mean values, and expressed as standard deviations. All calculations were performed using the SPSS 9.0 Windows software package.

Results

The surveillance data of 86 patients were used for analysis. Forty-four patients had AVGs (51.2%) and 42 patients had AVFs. A total of 76 fistulograms were performed; 73 (96%) met the criteria for stenosis (stenosis >50%). 92% of the stenotic lesions were located in the venous limb of the vascular access, while only 4% were located on the arterial side of the access. 46 stenoses were identified as a result of surveillance fistulography, 8 stenoses were identified when evaluated for prolonged access bleeding and 19 stenoses were noted following thrombolysis for an episode of access thrombosis.

The sensitivity and specificity of the vascular access surveillance protocol were calculated separately for AVFs and AVGs (tables 1, 2). In AVFs, 12 stenoses were predicted by 22 positive combined surveillance criteria. Seventeen positive VABF measurements (only 1 identified by a 25% decrease in access flow) revealed 10 stenoses and 14 positive dynamic Vp measurements revealed 7 stenoses. Twelve positive VABF measurements were not followed by either fistulography or access thrombosis (false positive). Three positive VABF measurements in 2 patients with an AVF were false positive, as proven by fistulography (stenosis <50%). Nine positive Vp measurements were not associated with access impairment (false positive). Eight significant stenoses were not predicted by either surveillance test (false negative). These included 4 stenoses noted following a thrombotic event and 4 stenoses identified by prolonged access bleeding.

In AVGs, 39 stenoses were predicted by 62 positive combined surveillance criteria. Fifty-five positive VABF measurements revealed 37 stenoses (3 identified by a 25% decrease in access flow) and 24 positive dynamic Vp measurements revealed 18 stenoses. Two positive Vp measurements in 1 patient with an AVG were noted to be false positive by fistulography (stenosis <50%). Fistulography did not demonstrate any false positive VABF measurements in the AVGs, whereas 17 positive VABF measurements were not associated with access thrombosis (in the absence of fistulography). Thirteen positive Vp measurements were not associated with vascular access impairment (false positive). Fourteen significant stenoses were not predicted by either surveillance measurement; 10 ste-

Table 3. Baseline characteristics and vascular access data

	Total group (n = 86)	AVFs (n = 42)	AVGs (n = 44)
Age, years	55 ± 15	53 ± 17	57 ± 14
Female gender, %	48.8	50	47.7
AA race, %	53.5	57.1	50
DM, %	34.9	21.4	47.7
Time in HD, days	1,577 ± 1,454	1,996 ± 1,901	1,207 ± 736
VA age, days	1,238 ± 1,298	1,564 ± 1,665	920 ± 678
Observation time, days	256 ± 35	258 ± 35	254 ± 36
VABF, ml/min	1,130 ± 530	1,290 ± 650	980 ± 330
Number of thrombotic events	19	5	14
Number of interventions	76	22	54
Intervention rate, n/patient years at risk	1.26	0.74	1.76
Thrombosis rate, n/patient years at risk	0.32	0.17	0.46

AA = African American; DM = diabetes mellitus; HD = hemodialysis; VA = vascular access.

noses were revealed by fistulography following thrombotic events and 4 stenoses were identified by prolonged vascular access bleeding.

The baseline characteristics of the 86 patients are summarized in table 3. As noted in table 3, the thrombosis rate (number per patient years at risk) in all 86 patients was 0.32 and the intervention rate was 1.26. The thrombosis and intervention rates for AVFs were 0.17 and 0.74, respectively, and for AVGs, they were 0.46 and 1.76, respectively.

Of the 86 patients enrolled in the surveillance protocol, 57 patients (66.3%) completely followed the surveillance protocol (groups 1 and 2), while 29 did not (group 3). Of the 57 patients following the protocol, 32 (37%) never met positive VABF or Vp criteria (group 1) and 25 patients (29%) met positive surveillance criteria and underwent preventive repair of access stenoses (group 2). Twenty-nine patients (group 3) did not strictly follow the surveillance protocol (fistulography and preventive access repair). Five out of 29 patients developed thrombosis within 8 weeks when they did not undergo a preventive repair following identification by positive surveillance criteria. Five out of 17 patients who did not have a preventive repair following positive VABF criteria developed access thrombosis. Two out of 21 who did not have a repair following positive dynamic Vp criteria also developed access thrombosis. The effects of early access repair on the thrombosis and intervention rates are shown in table 4. The number of thrombotic events was significantly higher in group 3 (n = 12) versus groups 1 (n = 4; p = 0.007) and 2 (n = 3; p = 0.049). The number of interventions was significantly higher in group 2 (n = 32) compared to group 3 (n = 28) (p = 0.001). This was the same for the thrombosis rate and intervention rate, calculated as thrombotic events and interventions per access year at risk (table 4).

Discussion

The principal objective of our prospective observational study was to evaluate the effect of a surveillance protocol based on the combination of VABF and dynamic Vp measurements on the identification of vascular access impairment (stenosis >50% or thrombosis). In addition, we were interested in examining the effect of preventive access repair on the rate of vascular access thrombotic episodes, since this is an important aspect of early identification of access impairment.

We combined dynamic Vp and VABF measurements as the foundation for our surveillance protocol. In a previous report, we demonstrated that dynamic Vp monitoring successfully identified AVGs at risk for impairment (stenosis or thrombosis) and allowed interventions to be performed that reduced the thrombosis rate (approximately 50%) compared with a historical control group [7]. In other studies, VABF measurements have been recognized as a more accurate monitoring tool for both AVGs [10, 12, 14, 15, 19] and AVFs [10, 14] as compared with dynamic Vp measurements. A recent survey showed an unequalled reduction in thrombosis rate, complication rate and medical cost using a surveillance protocol based on VABF measurements [16]. As Bosman et al. [17] did

Table 4. The influence of access surveillance on vascular access thrombosis

	Group 1 (n = 32)	Group 2 (n = 25)	Group 3 (n = 29)	p values
Age, years	48 ± 12	60 ± 15	58 ± 16	0.05#
Female, %	34	44	62	0.03#
AA race, %	53	40	31	NS
DM, %	28	48	31	NS
AVFs, %	31	76	52	NS
Time in HD, days	1,842 ± 1,686	1,491 ± 1,774	1,389 ± 713	NS
VA age, days	1,392 ± 1,218	1,373 ± 1,903	982 ± 683	NS
Observation time, days	262 ± 29	250 ± 39	254 ± 38	NS
Number of thrombotic events	4	3	12	0.007# 0.049*
Number of interventions	5	32	28	0.0001# 0.001* 0.0001+
Thrombosis rate, number of thrombotic events/access year at risk	0.17 ± 0.74	0.17 ± 0.46	0.62 ± 0.98	0.023# 0.032*
Intervention rate, number of interventions/access year at risk	0.22 ± 0.97	2.54 ± 1.11	1.39 ± 1.17	0.02# 0.00* 0.00+

AA = African American; DM = diabetes mellitus; HD = hemodialysis; VA = vascular access; NS = not significant.

Comparing groups 1 and 3. * Comparing groups 2 and 3. + Comparing groups 1 and 2.

not find any correlation between Vp and VABF measurements, we undertook a study to examine whether combining the two monitoring methods would improve vascular access surveillance. Although static Vp is more reliable than dynamic Vp in identifying patients with significant venous stenoses [3], we chose dynamic Vp monitoring for practical reasons and because this method was successful in our unit [7].

Utilizing our screening protocol and preventive access repair, we noted a decrease in the vascular access thrombosis rate that was below the rate suggested by the Kidney Disease Outcomes Quality Initiative guidelines [21]. However, our vascular access thrombosis rates were similar to those previously reported where only VABF screening was utilized [15, 16]. Although we did not have a true control group, which limits our ability to completely assess our surveillance results, our 'control' group consisted of patients who did not have fistulography following a positive surveillance test and in whom we were able to observe the natural history of no intervention.

We recognize that to correctly calculate sensitivity and specificity, fistulograms should have been performed in all patients regardless of their test results. In view of this limitation, we calculated sensitivity and specificity using vascular access impairment as defined by access stenosis (>50%) or thrombosis as the end point. Positive surveillance tests not followed by fistulography or thrombosis within 8 weeks of the positive test were classified as false positive. By doing so, the false positive rate became an overestimation, likely reducing the specificity of the surveillance tests. Combining the two screening methods resulted in a higher sensitivity in both AVGs and AVFs. Interestingly, the combination of screening methods offers a higher sensitivity for the detection of stenosis, but this trend does not hold for thrombosis risk.

The major question that exists is whether stenosis noted on fistulography is a reasonable surrogate for vascular access failure (i.e. predicting thrombosis). In our survey, 96% of the fistulograms performed revealed a significant stenosis. Supported by previous studies [13], one can safely assume that not every stenotic lesion is ultimately associated with vascular access thrombosis. Only hemodynamically significant stenoses, which decrease VABF, increase the risk for access thrombosis. This finding is actually supported by our data. Only 2 out of 21 patients with positive Vp measurements developed vascular ac-

cess thrombosis when they did not undergo an elective access repair; however, these 2 patients both met positive VABF criteria. By combining the two methods, we increased the number of preventive interventions; however, 4 were probably unnecessary. Review of our data reveals that we missed 3 thrombotic events in AVFs and 3 thrombotic events in AVGs by employing VABF cutoff levels that were too low. It is notable that 3 out of 4 unpredicted (by surveillance criteria) thrombotic events in AVFs were preceded by VABF measurements between 500 and 600 ml/min, and 3 out of 10 unpredicted thrombotic events in AVGs were preceded by VABF measurements between 650 and 750 ml/min. The sensitivity of VABF measurements would have been increased if higher cutoffs (600 ml/min for AVFs and 750 ml/min for AVGs) had been employed.

To improve sensitivity, it appears that adjusting the VABF cutoffs to higher levels would provide a greater benefit than combining VABF with dynamic Vp measurements.

In summary, the vascular access surveillance protocol that combined VABF and dynamic Vp measurements provided reasonable sensitivity and specificity in the detection of stenoses in AVFs and AVGs. The combined surveillance improved sensitivity for AVFs from 56.6 to 73.3%, but made little improvement in AVG sensitivity. However, the combination did not appear to further decrease the thrombosis rate as compared with the protocol employing VABF screening alone. Increasing the VABF referral threshold for AVFs to 600 ml/min and for AVGs to 750 ml/min could potentially improve sensitivity and reduce vascular access thrombosis rates, especially in AVFs. This approach will unfortunately reduce specificity. Early access repair following identification by surveillance reduced the overall thrombosis rate, but required a relatively high intervention rate. The addition of dynamic Vp monitoring probably resulted in unnecessary elective interventions and cannot be supported as an adjunct to VABF screening.

Acknowledgement

H.H. was supported by a grant from the Belgian American Educational Foundation.

References

1 Hakim R, Himmelfarb J: Hemodialysis access failure: A call to action. Kidney Int 1998;54: 1029–1040.
2 Schwab SJ, Raymond JR, Saeed M, Newman GE, Dennis PA, Bollinger RR: Prevention of hemodialysis fistula thrombosis. Early detection of venous stenoses. Kidney Int 1989;36: 707–711.
3 Besarab A, Sullivan KL, Ross RP, Moritz MJ: Utility of intra-access pressure monitoring in detecting and correcting venous outlet stenoses prior to thrombosis. Kidney Int 1995;47:1364–1373.
4 Sands JJ, Miranda CL: Prolongation of hemodialysis access survival with elective revision. Clin Nephrol 1995;44:329–333.
5 Ethier J, Falardeau P, Vandeville B, Roy L, Legault L, Gascon M, Beroniade V, Morin C: Access flow monitoring of native fistula using ultrasound dilution technique: 30 months experience. J Am Soc Nephrol 2000;11:183A.
6 Safa AA, Valji K, Roberts AC, Ziegler TW, Hye RJ, Oglevie SB: Detection and treatment of dysfunctional hemodialysis access grafts: Effect of surveillance program on graft patency and the incidence of thrombosis. Radiology 1996;199:653–657.
7 Cayco AV, Abu-Alfa AK, Mahnensmith RL, Perazella MA: Reduction in arteriovenous graft impairment: Results of a vascular access surveillance protocol. Am J Kidney Dis 1998; 32:302–308.
8 Krivitski NM: Novel method to measure access flow during hemodialysis by ultrasound velocity dilution technique. ASAIO J 1995;41: M741–M745.
9 Bosman PJ, Boereboom FT, Eikelboom BC, Koomans HA, Blankestijn PJ: Graft flow as a predictor of thrombosis in hemodialysis grafts. Kidney Int 1998;54:1726–1730.
10 Sands JJ, Jabyac PA, Miranda CL, Kapsick BJ: Intervention based on monthly monitoring decreases hemodialysis access thrombosis. ASAIO J 1999;45:147–150.
11 Strauch BS, O'Connell RS, Geoly KL, Grundlehner M, Yakub YN, Tietjen DP: Forecasting thrombosis of vascular access with Doppler color flow imaging. Am J Kidney Dis 1992;19: 554–557.
12 May RE, Himmelfarb J, Yenicesu M, Knights S, Ikizler TA, Schulman G, Hernanz-Schulman M, Shyr Y, Hakim RM: Predictive measures of vascular access thrombosis: A prospective study. Kidney Int 1997;52:1656–1662.
13 Neyra NR, Ikizler TA, May RE, Himmelfarb J, Schulman G, Shyr Y, Hakim RM: Change in access blood flow over time predicts vascular access thrombosis. Kidney Int 1998;54:1714–1719.
14 Schwab SJ, Oliver MJ, Suhocki P, McCann R: Hemodialysis arteriovenous access: Detection of stenosis and response to treatment by vascular access blood flow. Kidney Int 2001;59:358–362.
15 Smits JH, van der Linden J, Hagen EC, Modderkolk-Cammeraat EC, Feith GW, Koomans HA, van den Dorpel MA, Blankestijn PJ: Graft surveillance: Venous pressure, access flow, or the combination? Kidney Int 2001;59:1551–1558.
16 McCarley P, Wingard RL, Shyr Y, Pettus W, Hakim RM, Ikizler TA: Vascular access blood flow monitoring reduces access morbidity and costs. Kidney Int 2001;60:1164–1172.
17 Bosman PJ, Boereboom FT, Smits HF, Eikelboom BC, Koomans HA, Blankestijn PJ: Pressure or flow recordings for the surveillance of hemodialysis grafts. Kidney Int 1997;52:1084–1088.
18 Ethier JH, Lindsay RM, Barre PE, Kappel JE, Carlisle EJ, Common A: Clinical practice guidelines for vascular access. Canadian Society of Nephrology. J Am Soc Nephrol 1999; 10(suppl 13):S297–S305.
19 Lindsay R, Leypoldt J: Monitoring vascular access flow. Adv Ren Replace Ther 1999;6: 273–277.
20 DeSoto DJ, Ram SJ, Faiyaz R, Birk CG, Paulson WD: Hemodynamic reproducibility during blood flow measurements of hemodialysis synthetic grafts. Am J Kidney Dis 2001;37:790–796.
21 National Kidney Foundation: III. NKF-K/DOQI Clinical Practice Guidelines for Vascular Access: Update 2000. Am J Kidney Dis 2001;37(suppl 1):S137–S181.