

Influence of Luminal Diameters on Flow Surveillance of Hemodialysis Grafts: Insights from a Mathematical Model

John J. White,* Sunanda J. Ram,[†] Steven A. Jones,[‡] Steve J. Schwab,* and William D. Paulson*

*Section of Nephrology, Hypertension, and Renal Transplantation, Medical College of Georgia, Augusta, Georgia;

[†]Division of Nephrology and Hypertension, Department of Medicine, Louisiana State University Health Sciences Center, Shreveport, Louisiana; and [‡]Biomedical Engineering, Louisiana Tech University, Ruston, Louisiana

Randomized controlled trials have not shown that surveillance of graft blood flow (Q) prolongs graft life. Because luminal diameters affect flow resistance, this study examined whether the influence of diameters on Q can explain the limitations of surveillance. Inflow artery and outflow vein diameters were determined from duplex ultrasound studies of 94 patients. These diameters were applied to a mathematical model for determination of how they affect the relation between Q and stenosis. Also determined was the correlation between Q (by ultrasound dilution) and diameters, stenosis, and mean arterial pressure in 88 patients. Artery and vein diameters varied widely between patients, but arteries generally were narrower than veins. The model predicts that the relation between Q and stenosis is sigmoid: as stenosis progresses, Q initially remains unchanged but then rapidly decreases. A narrower artery increases flow resistance, causing a longer delay followed by a more rapid reduction in Q. In a multiple regression analysis of data from patients, Q correlated with artery and vein diameters, sum of largest stenoses from each circuit segment, and mean arterial pressure ($R = 0.689$, $P < 0.001$). This study helps to explain why Q surveillance predicts thrombosis in some patients but not others. Luminal diameters control the relation between Q and stenosis, and these diameters vary widely. During progressive stenosis, the delay and then rapid reduction in Q may impair recognition of low Q before thrombosis occurs. Surveillance outcomes might be improved by taking frequent measurements so that there is no delay in discovering that Q has decreased.

Clin J Am Soc Nephrol ●●: ●●●-●●●, ●●●●. doi: 10.2215/CJN.00580206

The hemodialysis synthetic graft is prone to developing progressive stenosis, thrombosis, and ultimately graft abandonment. The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (K/DOQI) guidelines recommend that grafts undergo surveillance of function combined with preemptive correction of stenosis so that graft failure can be prevented (1). Measurement of graft blood flow (Q) has been the preferred method of measuring function. This preference is based on the assumption that progressive stenosis causes a gradual reduction in Q that leads to stasis and thrombosis (2). Landmark nonrandomized studies reported that surveillance of function improves graft outcomes (3,4), but more recent randomized controlled trials have not confirmed this (5–7). Moreover, monthly Q measurements have proved to be an inaccurate predictor of thrombosis (8–11). In our patients, 44% of thromboses occur without a preceding reduction in Q (10,11). Consequently, the efficacy of flow surveillance has been debated at several recent nephrology symposia and in the literature (12–14).

We believe that the time has come to reexamine the basic

assumption that flow surveillance can detect progressive stenosis before thrombosis occurs. The feasibility of surveillance depends on the relations among Q, stenosis, and other variables, such as luminal diameters, mean arterial pressure (MAP), and hematocrit (Hct). For example, the shape of the Q versus stenosis curve may influence the success of surveillance. A linear relation between Q and stenosis would favor an early reduction in Q as stenosis progresses, whereas a sigmoid relation would favor a delayed reduction in Q followed by a rapid decrease that might impair timely detection of stenosis. As another example, if diameters of the inflow artery and outflow vein vary from patient to patient, then resistance to flow also will vary. Such variation in resistance will alter the relation between Q and stenosis, so surveillance may be more effective in some patients than in others. Spergel *et al.* (15) similarly suggested that variation in diameters may impair the effectiveness of venous pressure surveillance.

Evaluation of these issues requires a thorough analysis of relations among Q, stenosis, luminal diameters, and other key variables in the graft circuit. This has not been done in clinical studies because such relations are complex and not easily discerned from data on patients. Alternatively, a mathematical model of the graft provides an ideal method to analyze such relations. We previously proposed such a model on the basis of pressure-flow equations from the engineering literature (16) and then used an *in vitro* apparatus to refine and validate the

Received February 16, 2006. Accepted May 26, 2006.

Published online ahead of print. Publication date available at www.cjasn.org.

Address correspondence to: Dr. William D. Paulson, BA 9413, Medical College of Georgia, 1120 15th Street, Augusta, GA 30809. Phone: 706-721-9655; Fax: 706-721-7136; E-mail: wpaulson@mcg.edu

model (17). In this study, we applied representative diameters of inflow arteries and outflow veins to the model and determined how variations in diameters, MAP, and Hct affect the relation between Q and stenosis. The model improves understanding of graft hemodynamics and the factors that affect Q surveillance, and may help in designing programs that optimally detect and treat progressive stenosis.

Materials and Methods

Model of Graft Vascular Circuit

The model includes the inflow artery, arterial and venous anastomoses, graft, stenosis, and outflow vein (Figure 1) (16,17). It is designed to resemble a loop graft that is anastomosed end to side to the radial or brachial artery and cubital or cephalic vein. Nevertheless, the principles developed herein also apply to a straight graft (see Discussion section). Given that the model is nonpulsatile, calculations of Q and pressure should be considered time averaged. The absence of pulsatility may have caused at most a mild overestimation of Q and should not affect the general validity of the model (17). For simplicity, the model assumes that the artery and vein distal to the anastomosis are ligated or that flows in these vessels can be ignored because they are small compared with graft flow. The model ignores special conditions, such as tortuous vessels and nonuniform diameters.

The model is defined by a total pressure drop equation (ΔP_{TOTAL}) that is the sum of pressure drops across all of the segments of the circuit (16,17): $\Delta P_{\text{TOTAL}} = \text{MAP} - \text{CVP} = \Delta P_{\text{ARTERY}} + \Delta P_{\text{AA}} + \Delta P_{\text{GRAFT}} + \Delta P_{\text{VA}} + \Delta P_{\text{STENOSIS}} + \Delta P_{\text{VEIN}}$, where CVP is central venous pressure, AA is arterial anastomosis, and VA is venous anastomosis. This equation is based on the concept that pressure falls as blood flows through the circuit, and the total pressure drop is $\text{MAP} - \text{CVP}$. The ΔP_{TOTAL} equation determines relations between the variables that characterize the circuit: Q, circuit pressures, luminal diameters, stenosis, MAP, and Hct. When diameters are uniform throughout, ΔP_{TOTAL} largely is caused by friction-induced losses of fluid energy. Changes in vascular resistance of the access circuit directly affect Q, whereas changes in systemic vascular resistance affect Q through their effect on MAP.

We showed previously that, with some refinements, pressure-flow equations from the engineering literature can be used to model the graft circuit (17). These equations give ΔP as a function of Q, luminal diameter, MAP, viscosity, and other variables and constants. We related the

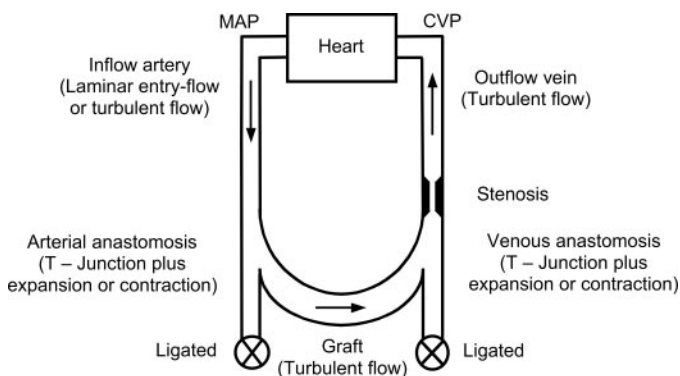


Figure 1. Model of graft vascular circuit. Figure indicates pressure-flow equations that were used to compute pressure drops (ΔP) at each segment of circuit. Sum of all the ΔP equations equals mean arterial pressure (MAP) minus central venous pressure (CVP). Adapted from reference (17) with permission of the American Society of Mechanical Engineers.

viscosity of blood (μ , in g/cm per s) to Hct (%) with the following equation: $\mu = 0.01 + (5.6 \times 10^{-4})\text{Hct}$ (18). The lengths of the artery, graft, vein, and stenosis were set equal to 40, 34, 40, and 1 cm, respectively. The artery and vein lengths represent the vessels of a forearm loop graft before they develop wider diameters in the chest (where they do not contribute significantly to flow resistance). We selected artery and vein diameters that were representative of our patients (see Measurements in Patients section). We defined stenosis as percentage reduction in luminal diameter.

The ΔP equations for the artery, graft, and vein depend on whether flow is laminar or turbulent. Thus, the type of flow influences Q. A minimum “entrance length” is required for laminar flow to develop fully. For most large arteries, the entrance length approaches the length of the artery, so laminar flow usually is not developed fully. Therefore, we used Shah’s laminar entry-flow equation to represent such flow (19,20). We used a modified Blasius equation to represent turbulent flow (17,21).

The *in vitro* study showed that the graft and the vein exhibit turbulent flow, whereas the artery may exhibit laminar entry flow or turbulent flow, depending on the Reynolds number: $\text{Re} = \rho Q / 15\pi D\mu$ (ρ is blood density in g/cm³, Q is in ml/min, and D is diameter in cm) (16). We found that as Re increased above 1500 in the artery, flow became turbulent (17). Therefore, we used Shah’s equation when Re was <1500 (19,20) and used the modified Blasius equation when Re was ≥ 1500 (17,21).

We modeled the stenosis with a modified Young’s equation (17,22). The anastomoses were modeled by adding two equations together: a T-junction equation that defines ΔP across the junction of two tubes (23), plus ΔP caused by increases (24) or decreases (25) in luminal diameter (Bernoulli’s Law [16]).

Measurements in Patients

To ensure that we used representative diameters of the artery and the vein, we reviewed duplex ultrasound studies of 109 patients who had grafts and were evaluated for eligibility in a previous clinical trial (5). A single qualified ultrasound technologist performed studies with a Siemens Sonoline Versa Scanner and a 7.5- or 5.0-MHz linear transducer (Siemens Medical Systems Inc., Issaquah, WA). The graft, arterial and venous anastomoses, inflow artery, and outflow vein were evaluated in sagittal and transverse planes with and without color. Images were recorded on videotape for subsequent analysis. The largest percentage stenosis in each circuit segment was recorded. Luminal diameters were measured where they were most representative of the artery and vein within a few centimeters of the anastomoses. These locations had the smallest diameters of arteries and veins in the circuit and therefore largely controlled inflow and outflow resistances.

Patients who entered the previous trial (5) underwent measurement of Q by ultrasound dilution (Transonic Systems, Inc., Ithaca, NY) (26) a mean of 5 d after the duplex ultrasound study. Q and MAP were determined together as described previously (5).

Analysis

The ΔP_{TOTAL} equation was used to compute relations among Q, pressures, luminal diameters, stenosis, MAP, and Hct. After fully defining the equation (Figure 1), we used Microsoft Excel Solver (the Generalized Reduced Gradient nonlinear optimization code) to determine these relations.

Statistical Analyses

For data from patients, standard least-squares regression analysis was used to determine correlations among the variables in the model.

Differences between means were tested by paired *t* test. $P < 0.05$ was considered significant.

Results

Selecting Luminal Diameters

Selection of luminal diameters is a key step in modeling the graft circuit. For simplicity, we assumed a uniform graft diameter of 0.60 cm. In selecting artery and vein diameters, we considered the results of duplex ultrasound studies in 109 patients. We excluded 15 patients because grafts were not in the arm or the forearm or because duplicated outflow veins or other abnormalities prevented a representative measurement of diameters (Tables 1 and 2). The mean graft age in the remaining 94 patients was >1 yr. Diameters varied widely, but, in general, the artery was narrower than the vein (Figure 2). The median ratio of artery/vein diameters was 0.77, and 80 of 94 patients had ratios of <1.0 . We considered the influence of low, median, and high ratios on the model (Table 3).

Pressures in Graft Circuit

The model is based on equations that relate pressure to Q and other key variables. Figure 3 shows the pressure from beginning to end of the circuit as percentage of initial value. The artery/vein ratio was set at the median value of 0.77, so the artery was narrower than the vein. The model predicts that in the nonstenotic circuit, pressure falls 49% by the time the arterial limb of the graft is reached. Figure 3 shows, however, that as stenosis progresses, pressure upstream to the stenosis increases, and the pressure drop across the stenosis eventually accounts for most of the pressure drop in the circuit.

Relations between Variables in Model

We determined the relation between Q and stenosis while varying MAP from 70 to 140 mmHg (Figure 4). This MAP range was observed in a previous study of 51 patients (27). Figure 4 shows that the relation between Q and stenosis is sigmoid: as stenosis progresses, Q initially remains unchanged but then rapidly decreases as critical stenosis (60 to 80%) is reached. Figure 4 also shows that MAP has a strong effect on Q: Dou-

bling MAP will increase Q by 50% at all levels of stenosis. In contrast, Hct has a small effect on Q. The model predicts that an increase in Hct from 15 to 45% will decrease Q by only 4 to 6%.

Artery and vein diameters control the relation between Q and stenosis (Figure 5). As the artery narrows (lower artery/vein ratios), Q is reduced and the curve is flattened and shifted to the right. This is illustrated further in Figure 6, which shows Q as a percentage of initial value. Ratios below the median value shift the curve to the right, promoting a delay and then rapid reduction in Q. Conversely, a high ratio shifts the curve to the left, thereby promoting a slightly earlier decrease.

Correlations between Variables in Patients

We evaluated the correlation between Q and luminal diameters in 88 patients from Table 1 who had the following measurements: Q (by ultrasound dilution), artery and vein diameters, largest percentage stenosis in each circuit segment (artery and anastomosis, graft, venous anastomosis, and vein), and MAP. Individual stenoses were NS when compared with sum of stenoses, so only the sum was considered. With Q as the dependent variable, the simple correlation coefficients were sum of stenoses ($r = -0.531$, $P < 0.001$), vein diameter ($r = 0.461$, $P < 0.001$), artery diameter ($r = 0.378$, $P < 0.001$), and MAP ($r = 0.190$, $P = 0.08$). All four independent variables were significant when included in a multiple regression analysis (all $P < 0.005$); the multiple correlation coefficient was 0.689 ($P < 0.001$).

Discussion

This study used a mathematical model of the graft vascular circuit to determine relations among Q, luminal diameters, stenosis, MAP, and Hct. The access circuit is unique in that it bypasses the arteriolar resistances. This yields a high Q shunt with a large dissipation of energy and pressure before the arterial limb of the graft is reached. Therefore, it is unlike the normal circulation in which pressures in the arm are only slightly lower than in the ascending aorta.

This study improves understanding of graft hemodynamics and helps to explain why Q surveillance predicts thrombosis in some patients but not others. The most important new insight is that diameters of the inflow artery and outflow vein vary widely, and the diameters of these vessels control the relation between Q and stenosis. Patients generally have narrower arteries than veins, so the inflow artery dominates circuit resistance until stenosis is well advanced. Artery/vein diameter ratios below the median (narrower arteries) are predicted to delay the stenosis-induced reduction in Q until critical stenosis (60 to 80%) is reached (Figures 5 and 6). Assuming stenosis progresses at a constant rate, the delay and then rapid reduction in Q helps to explain why monthly Q measurements often fail to warn of thrombosis (8–10).

In applying Q surveillance, it is important to avoid unnecessary procedures. Therefore, grafts should not be referred for angiography and preemptive correction of stenosis unless a significant decrease in Q (ΔQ) has occurred. If $P < 0.05$ is required, then ΔQ must be $>33\%$ (28), whereas the K/DOQI guidelines recommend $\Delta Q >25\%$ (1) ($P < 0.11$ [28]). As the

Table 1. Patient and graft characteristics

Characteristic	Value
No. of patients	94
Patient age (mean \pm SEM)	54.3 \pm 1.6
Black	90 (96%)
Female	55 (59%)
Diabetes	43 (46%)
Hypertension	94 (100%)
Graft age (d; mean \pm SEM)	405 \pm 57
Graft location	
forearm	32 (34%)
arm	62 (66%)
Configuration	
loop	34 (36%)
straight	60 (64%)

Table 2. Duplex ultrasound measurements of luminal diameters in 94 patients

Luminal Diameters	Mean \pm SEM	Median	Range
Inflow artery (cm)	0.592 \pm 0.013 ^a	0.575	0.34 to 1.15
Outflow vein (cm)	0.803 \pm 0.026	0.745	0.28 to 1.67
Ratio of artery/vein	0.793 \pm 0.028	0.769	0.32 to 2.29

^aArtery versus vein: $P < 0.001$.

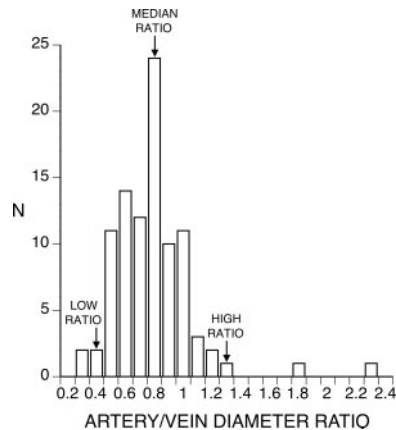


Figure 2. Distribution of ratios of artery/vein luminal diameters in 94 patients. Arrows indicate ratios used in mathematical model. Median = 0.77; low (0.40) and high (1.28) ratios indicate limits that enclose 95% of patients.

dashed line in Figure 6 shows, ΔQ will not be $>25\%$ until stenosis is >51 , 54 , and 71% , for high, median, and low artery/vein ratios, respectively. By the time the $K/DOQI$ threshold is reached, Q is on the most rapidly falling portion of the sigmoid curve, and the curve is steepest for ratios that are below the median.

Krivitski and Gantela (29) used Poiseuille's law to analyze graft hemodynamics. Poiseuille's law assumes fully developed laminar flow in a rigid straight tube of constant diameter, and none of these assumptions is satisfied in the graft. Also, their model assigned the large inflow artery resistance to the arterial anastomosis. Nevertheless, Krivitski and Gantela's (29) analysis provides a useful preliminary approximation. Our model is based on an application of rigorous pressure-flow equations from the engineering literature that have been refined and validated by an *in vitro* model (16,17). This study is the first of

which we are aware to have applied these equations collectively in the context of the hemodialysis access.

It is important to confirm the model with data from patients. In a multiple regression analysis, we found that Q correlated with four variables from the model: the sum of largest stenoses from each circuit segment, artery and vein diameters, and MAP (all $P < 0.005$). The multiple correlation coefficient (R) was 0.689, indicating that approximately 50% of Q variation between patients was due to the relation between Q and these four variables ($R^2 = 0.48$). As predicted by the model, narrower diameters were associated with lower Q . This analysis does not take into account a number of factors that affect Q , such as specific geometries of access circuit, lengths of stenoses, luminal irregularities, and minor stenoses. Considering these limitations, this provides strong clinical confirmation for the model.

As further confirmation, Sullivan *et al.* (30) measured pressures in the graft circuits of 83 patients and correlated these with stenoses that were measured by angiography (Figure 7). It is notable that pressures in the model with no stenosis (Figure 3) are similar to pressures in patients with low-grade ($\leq 40\%$) stenosis (which should have a minimal effect on pressures). The model predicts that for no stenosis, pressure falls to 51% of initial value by the time the arterial limb of the graft is reached (Figure 3), whereas in patients with low-grade stenosis, pressure fell slightly more to 45% of initial value (Figure 7). Therefore, the model agrees remarkably well with patient data. This comparison also suggests that, as in our patients, patients in the study of Sullivan *et al.* (30) had arteries that generally were narrower than veins. Comparison of the model with data from patients who had high-grade stenosis at the venous anastomosis ($>40\%$; Figure 7) is problematic because patients had additional stenoses at other locations, which would alter pressures throughout the circuit. With this caution in mind, the predictions in Figure 3 seem to be consistent with patients with high-grade stenosis in Figure 7.

Table 3. Hypothetical luminal diameters and luminal diameter ratios used in the mathematical model^a

Type of Artery/ Vein Ratio	Inflow Artery Diameter (cm)	Outflow Vein Diameter (cm)	Value of Artery/ Vein Ratio
Low ratio	0.400	1.000	0.40
Median ratio	0.575	0.745	0.77
High ratio	0.950	0.745	1.28

^aArtery and vein luminal diameters were selected so that artery/vein ratios were median and lower and upper limits that enclose 95% of patients in Figure 2.

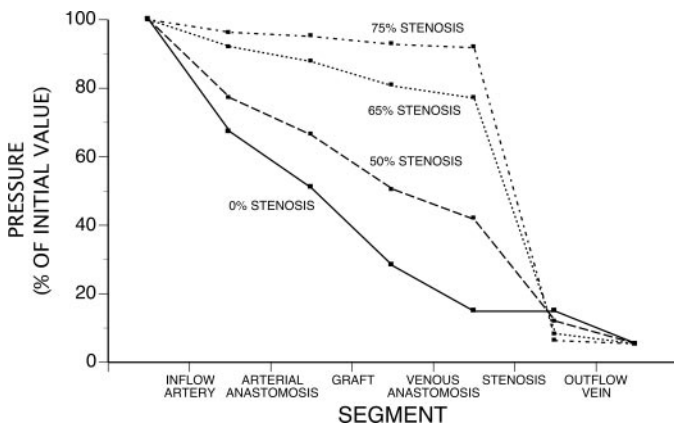


Figure 3. Predicted pressure along segments of graft vascular circuit at different levels of stenosis. Conditions: median ratio of artery/vein diameters, initial pressure (MAP) = 93 mmHg, CVP = 5 mmHg, hematocrit (Hct) = 36%. MAP of 93 mmHg is equivalent to 120/80 mmHg.

This study confirms the clinical observation that MAP has a large effect on Q and underscores the importance of avoiding hypotension in preventing thrombosis. It often has been proposed that Q should be normalized for MAP (Q/MAP) or systolic pressure so as to offset the effect of hemodynamic changes on Q (31–33). This adjustment assumes that a change in MAP causes a proportionally equal change in Q. However, this adjustment impairs the accuracy of Q in predicting thrombosis (10), and the model explains why. It predicts that doubling MAP will increase Q by only 50%, so Q/MAP is an overcorrection. In any case, it is unclear whether adjusting Q for MAP is helpful because a reduction in MAP is itself an independent risk factor for thrombosis, probably because it lowers Q (34).

In contrast to MAP, Hct has a small effect on Q. Because viscosity increases with Hct, one might expect that a rise in Hct

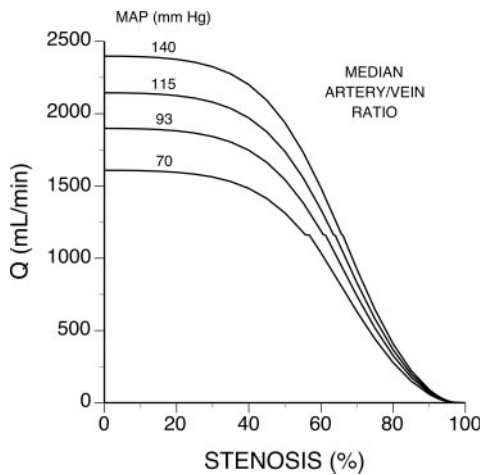


Figure 4. Predicted relation between graft blood flow (Q) and stenosis at different levels of MAP (CVP = 5 mmHg, Hct = 36%). Discontinuities represent transition from turbulent to laminar entry flow in artery as stenosis causes Q to fall (see Materials and Methods).

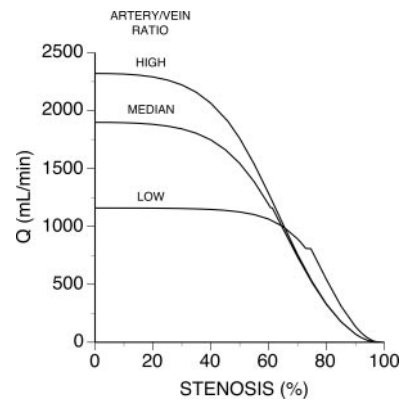


Figure 5. Model predicts that artery and vein diameters control relation between Q and stenosis (MAP = 93 mmHg, CVP = 5 mmHg, Hct = 36%). Q falls as the artery becomes narrower (lower artery/vein diameter ratios).

would increase resistance to flow and cause a reduction in Q (16). The effect of viscosity on Q is greatest in laminar flow. However, the model has turbulent flow in the graft and the vein, and laminar entry flow or turbulent flow in the artery. Therefore, turbulent flow dominates the model, which does not favor a strong effect for Hct. The model is consistent with the observation that increases in Hct during dialysis do not affect Q (35).

Finally, we should note that in applying the model, we assumed that the inflow artery and the outflow vein have an equal length of 40 cm; these lengths most closely approximate a forearm loop graft. Nevertheless, the importance of luminal diameters applies generally. For example, although an arm loop graft has shorter arteries and veins (which minimizes the influence of the relatively narrow vessels in the arm), a straight graft has a longer artery than vein (which enhances the influence of the narrower artery). Moreover, note that the most common access of patients in Table 1 was the straight graft.

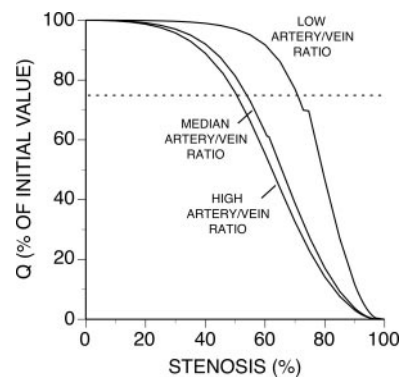


Figure 6. As artery becomes narrower (lower artery/vein ratios), sigmoid curve shifts to the right (MAP = 93 mmHg, CVP = 5 mmHg, Hct = 36%). This promotes delay and then rapid reduction in Q. Values below dashed line represent decrease in Q of >25% (Kidney Disease Outcomes Quality Initiative referral threshold [1]).

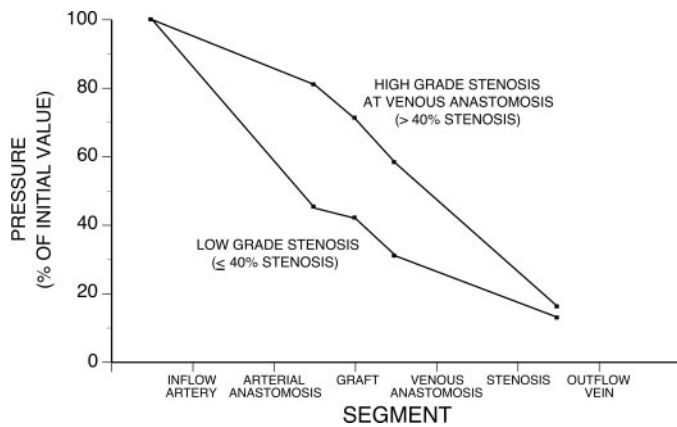


Figure 7. Pressure along segments of graft vascular circuit in patients with low-grade stenosis (any location) and high-grade stenosis (at venous anastomosis). Adapted from reference (30) with permission of the Radiological Society of North America.

Conclusion

The model predicts that a significant decrease in Q does not occur until stenosis is well advanced. Then, assuming stenosis progresses at a constant rate, Q falls so rapidly that there may be insufficient time to detect the decrease before thrombosis occurs. These results question the viability of Q surveillance as currently practiced on a monthly schedule. Outcomes might be improved by measuring Q more frequently so that there is no delay in discovering that Q has decreased. However, measurement every 2 wk has not improved prediction of thrombosis (36). Rates of stenosis progression provide an additional clue to the necessary frequency: we have found that in grafts that thrombose, percentage of stenosis increases at a mean rate of 26%/mo (37). These considerations suggest that measurements might need to be made at least weekly. Another important issue is that the influence of luminal diameters on Q addresses only one potential contributor to the poor results of surveillance. For example, it does not address whether preemptive angioplasty is an effective treatment once Q surveillance has identified stenosis (14). It is possible that even if measurements were done more frequently, outcomes might not be improved.

Acknowledgments

We thank Dialysis Clinic, Inc., for a grant that funded part of this study.

This study was presented in part at the annual meeting of American Society of Nephrology; November 8 through 13, 2005; Philadelphia, PA.

References

- National Kidney Foundation: K/DOQI clinical practice guidelines for vascular access. *Am J Kidney Dis* 37[Suppl 1]: S137–S181, 2001
- Paulson WD: Blood flow surveillance of hemodialysis grafts and the dysfunction hypothesis. *Semin Dial* 14: 175–180, 2001
- Schwab SJ, Raymond JR, Saeed M, Newman GE, Dennis PA, Bollinger RR: Prevention of hemodialysis fistula thrombosis. Early detection of venous stenoses. *Kidney Int* 36: 707–711, 1989
- 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* 47: 1364–1373, 1995
- Ram SJ, Work J, Eason JM, Caldito GC, Pervez A, Paulson WD: A prospective randomized controlled trial of blood flow and stenosis surveillance of hemodialysis grafts. *Kidney Int* 64: 272–280, 2003
- Moist LM, Churchill DN, House AA, Millward SF, Elliott JE, Kribs SW, DeYoung WJ, Blythe L, Stitt LW, Lindsay RM: Regular monitoring of access flow compared with monitoring of venous pressure fails to improve access survival. *J Am Soc Nephrol* 14: 2645–2653, 2003
- Dember LM, Holmberg EF, Kaufman JS: Randomized controlled trial of prophylactic repair of hemodialysis arteriovenous graft stenosis. *Kidney Int* 66: 390–398, 2004
- McDougal G, Agarwal R: Clinical performance characteristics of hemodialysis graft monitoring. *Kidney Int* 60: 762–766, 2001
- Paulson WD, Ram SJ, Birk CG, Work J: Does blood flow accurately predict thrombosis or failure of hemodialysis synthetic grafts? A meta-analysis. *Am J Kidney Dis* 34: 478–485, 1999
- Paulson WD, Ram SJ, Birk CG, Zapczynski M, Martin SR, Work J: Accuracy of decrease in blood flow in predicting hemodialysis graft thrombosis. *Am J Kidney Dis* 35: 1089–1095, 2000
- Paulson WD, Ram SJ, Work J: Use of vascular access blood flow to evaluate vascular access [Letter]. *Am J Kidney Dis* 37: 451–452, 2001
- Sands JJ: Vascular access monitoring improves outcomes. *Blood Purif* 23: 45–49, 2005
- Paulson WD: Access monitoring does not really improve outcomes. *Blood Purif* 23: 50–56, 2005
- White JJ, Bander SJ, Schwab SJ, Churchill DN, Moist LM, Beathard GA, Vesely TM, Paulson WD, Huber TS: Is percutaneous transluminal angioplasty an effective intervention for A-V graft stenosis? *Semin Dial* 18: 190–202, 2005
- Spergel LM, Holland JE, Fadem SZ, McAllister CJ, Peacock EJ: Static intra-access pressure ratio does not correlate with access blood flow. *Kidney Int* 66: 1512–1516, 2004
- Paulson WD, Jones SA: Hemodynamics of the vascular access: Implications for clinical management. *Contrib Nephrol* 142: 238–253, 2004
- Jones SA, Jin S, Kantak A, Paulson WD: A mathematical model for pressure losses in the hemodialysis graft vascular circuit. *J Biomech Eng* 127: 60–66, 2005
- Cokelet GR: The rheology of human blood. In: *Biomechanics: Its Foundations and Objectives*, edited by Fung YC, Perrone N, Anliker M, Englewood Cliffs, NJ, Prentice-Hall, 1972, pp 63–103
- Shah RK: A correlation for laminar hydrodynamic entry length solutions for circular and noncircular ducts. *J Fluids Engineering* 100: 177–179, 1978
- White FM: Solutions of the Newtonian viscous-flow equations. In: *Viscous Fluid Flow*, edited by Beamesderfer L, Morriss J, Boston, McGraw Hill, 1991, pp 104–217
- White FM: Lamina boundary layers. In: *Viscous Fluid Flow*,

- edited by Beamesderfer L, Morriss J, Boston, McGraw Hill, 1991, pp 218–334
22. Young DF, Tsai FY: Flow characteristics in models of arterial stenosis: I. Steady flow. *J Biomech* 6: 395–410, 1973
 23. White FM: Viscous flow in ducts. In: *Fluid Mechanics*, edited by Kane KT, Boston, McGraw Hill, 1999, pp 325–424
 24. Potter MC, Wiggert DC: *Mechanics of Fluids*, Upper Saddle River, NJ, Prentice Hall, 1997, pp 303–304
 25. Munson BR, Young DF, Okiishi TH: *Fundamentals of Fluid Mechanics*, New York, John Wiley & Sons, 1994, p 497
 26. Krivitski NM: Theory and validation of access flow measurement by dilution technique during hemodialysis. *Kidney Int* 48: 244–250, 1995
 27. DeSoto DJ, Ram SJ, Faiyaz R, Birk CG, Paulson WD: Hemodynamic reproducibility during blood flow measurements of hemodialysis synthetic grafts. *Am J Kidney Dis* 37: 790–796, 2001
 28. Ram SJ, Nassar R, Sharaf R, Magnasco A, Paulson WD: Thresholds for significant decrease in hemodialysis access blood flow. *Semin Dial* 18: 558–564, 2005
 29. Krivitski NM, Gantela S: Relationship between vascular access flow and hemodynamically significant stenoses in arteriovenous grafts. *Hemodial Int* 7: 23–27, 2003
 30. Sullivan KL, Besarab A, Bonn J, Shapiro MJ, Gardiner GA, Moritz MJ: Hemodynamics of failing dialysis grafts. *Radiology* 186: 867–872, 1993
 31. Agharazii M, Clouatre Y, Nolin L, Leblanc M: Variation of intra-access flow early and late into hemodialysis. *ASAIO J* 46: 452–455, 2000
 32. Besarab A, Lubowski T, Vu A, Aslam A, Frinak S: Effects of systemic hemodynamics on flow within vascular accesses used for hemodialysis. *ASAIO J* 47: 501–506, 2001
 33. Tonelli M, Hirsch DJ, Chan CT, Marryatt J, Mossop P, Wile C, Jindal K: Factors associated with access blood flow in native vessel arteriovenous fistulae. *Nephrol Dial Transplant* 19: 2559–2563, 2004
 34. Paulson WD, Ram SJ, Faiyaz R, Caldito GC, Atray NK: Association between blood pressure, ultrafiltration, and hemodialysis graft thrombosis: A multivariable logistic regression analysis. *Am J Kidney Dis* 40: 769–776, 2002
 35. Ronco C, Brendolana A, Crepaldia C, D'Intinia V, Sergeyevab O, Levin NW: Noninvasive transcutaneous access flow measurement before and after hemodialysis: Impact of hematocrit and blood pressure. *Blood Purif* 20: 376–379, 2002
 36. Ram SJ, Birk CG, Work J, Paulson WD: Role of frequent blood flow measurements in predicting hemodialysis graft thrombosis. In: *Vascular Access for Hemodialysis: VII*, edited by Henry ML, Chicago, Precept Press, 2001, pp 239–247
 37. Atray NK, Ram SJ, Work J, Eason JM, Oder TF, Paulson WD: A longitudinal study of hemodialysis graft stenosis: Role of progressive stenosis in thrombosis [Abstract]. *J Am Soc Nephrol* 12: 281A, 2001