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Hemodynamics of the Hemodialysis Access: Implications for Clinical Management

William D. Paulson^a, Steven A. Jones^b

^aInterventional Nephrology Section, Division of Nephrology and Hypertension, Department of Medicine, Louisiana State University Health Sciences Center, Shreveport, La., and ^bBiomedical Engineering, Louisiana Tech University, Ruston, La., USA

Introduction

Optimum prevention and management of access complications require an understanding of access hemodynamics. The hemodialysis access is unique in that it creates a low resistance shunt between the arterial and venous circulations. Thus, it is unlike the arterial synthetic graft used in atherosclerotic vascular disease in which the arterioles continue to provide the main source of resistance and blood flow regulation. This chapter describes access hemodynamics for nephrologists, interventionists, surgeons, and others who are involved in the management and preservation of the hemodialysis access. It develops a hemodynamic model that provides insight into access complications and management. Definitions and units of measurement are listed in the Appendix.

Basic Fluid Mechanics

Energy is required to drive flow in the access vascular circuit [1–3]. The total fluid energy per unit volume equals potential energy plus kinetic energy. Potential energy per unit volume exists as pressure (P), which is the force (F) exerted by fluid against an area A (F/A). Pressure is produced by ejection of blood from the left ventricle, by the force of elastic vessels pressing against the blood, and by differences in blood height. Kinetic energy per unit volume is defined as $\rho v^2/2$ where ρ is blood density and v is blood velocity.

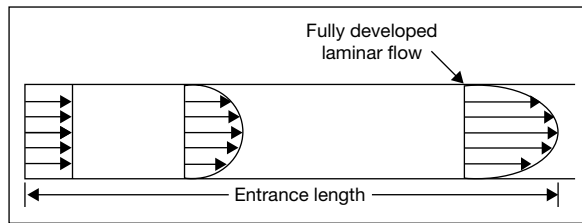


Fig. 1. Development of laminar flow in a tube. Length of arrows indicates velocity. Velocity of lamina immediately adjacent to tube wall is 0.

In the absence of friction, total fluid energy remains constant. This concept is known as Bernoulli's equation:

$$\text{fluid energy/volume} = P + \rho v^2/2 = \text{constant}$$

For example, as blood enters a stenosis, velocity increases and pressure drops (ΔP), indicating energy has been converted from potential energy to kinetic energy. In reality, friction causes dissipation of energy as blood flows through the access circuit. Thus, the fluid energy in the inflow artery is greater than in the outflow vein. Energy loss within a circuit is conventionally approximated as the ΔP between entry and exit from the circuit.

It is important to characterize the type of flow in the access circuit. Laminar flow is organized motion in which fluid travels as a series of cylindrical laminae parallel to the wall of a tube. The lamina in contact with the wall is stationary, whereas each successive lamina slides against the friction of adjacent lamina. As fluid enters a perfectly cylindrical tube, these parallel laminae gradually develop a parabolic velocity profile in that the lamina closest to the wall remains motionless whereas the lamina in the center has the highest velocity (fig. 1). A minimum 'entrance length' is required for laminar flow to fully develop. Before this length is reached, the velocity profile is blunted. For most large arteries in the normal systemic circulation, the entrance length approaches the length of the artery, so that laminar flow is usually not fully developed. In these situations, laminar 'entry-flow' models are used to characterize flow [4].

The concepts of shear rate and shear stress are needed in order to define fluid friction in the access circuit [2, 3]. Shear rate is the change in flow velocity (Δv) per change in distance (Δx) perpendicular to the direction of flow:

$$\text{shear rate} = \Delta v / \Delta x$$

In figure 1, the shear rate near the wall is lowest for fully developed laminar flow because the difference in velocity of adjacent laminae is small in this parabolic profile. In contrast, the shear rate is extremely high at the tube

entrance where the velocity abruptly increases from 0 at the wall to the velocity indicated by the blunt profile.

Shear stress is the force that is necessary to move a layer of fluid of area A across another layer of fluid (or vessel wall) of equal area:

$$\text{shear stress} = F/A$$

An increase in friction between fluid layers will increase the amount of force needed to move the fluid.

Viscosity connects the concepts of shear rate and shear stress, and is a measure of friction between contiguous layers of fluid [2, 3]. Dynamic viscosity (η) is defined as the ratio of shear stress to shear rate:

$$\eta = (\text{shear stress})/(\text{shear rate}), \text{ or } \text{shear stress} = \eta(\text{shear rate})$$

Thus, if more force is required to move layers of fluid across each other at a given shear rate, then η is higher. Alternatively, for a given η , a higher shear rate indicates a higher shear stress is applied to overcome friction. This is the friction that causes dissipation of energy in the access circuit.

Poiseuille's law describes the ΔP caused by viscous losses in fully developed laminar (nonpulsatile) flow in a rigid tube with constant luminal radius (r) [2, 3]:

$$\Delta P = 8\eta LQ/\pi r^4$$

L is length along the tube and Q is flow.

Poiseuille's law provides an expression for estimating vascular resistance (R), which is defined as $\Delta P/Q$:

$$R = \Delta P/Q = 8\eta L/\pi r^4 = 128\eta L/\pi D^4$$

where D is luminal diameter. This equation shows that for a given ΔP , a smaller R will result in a larger Q . R increases linearly with η and L , but D has by far the largest influence since R is proportional to $1/D^4$. Thus, reductions in diameter can cause very large increases in R . Poiseuille's law is based on several assumptions that are generally not completely satisfied in the access circuit [2], so that it usually underestimates ΔP and R . Nevertheless, it is a valuable tool for illustrating hemodynamic principles.

Turbulent flow is characterized by random irregular motion rather than the organized motion of laminar flow. Sometimes flow is disturbed and has both laminar and turbulent characteristics. In models of turbulent or disturbed flow, ΔP is proportional to Q^2 and Q raised to other powers such as $Q^{7/4}$ [4]. This indicates that turbulent and disturbed flow cause a higher ΔP and R than predicted by Poiseuille's law. In these models, R is not constant but rather is a function of Q . Thus, an increase in Q causes an increase in R .

One of the most important predictors of whether flow is laminar or turbulent is the Reynolds number (Re) [2, 3]:

$$Re = 2\rho r v / \eta$$

In classical fluid mechanics, as Re increases above 2,000, laminar flow makes a transition to turbulent flow. Nevertheless, 2,000 is not a strict threshold. Turbulent flow may occur with Re as low as 1,000 and laminar flow may persist for Re above 3,000, depending on conditions.

Re can be rewritten in terms of Q rather than v. Note that $Q = vA = v\pi r^2$, in which A is vessel cross-sectional area. It follows that:

$$Re = 2\rho r Q / \pi r^2 \eta = 2\rho Q / \pi r \eta$$

when the variables are expressed in consistent units (ρ in g/cm^3 , Q in ml/s, r in cm, and η in $\text{g/cm}\cdot\text{s}$). In clinical practice, Q is usually expressed in ml/min, which must be converted to ml/s by dividing by 60. Thus, when Q is in ml/min, the equation takes various forms:

$$Re = 2\rho Q / 60\pi r \eta = \rho Q / 30\pi r \eta = \rho Q / 15\pi D \eta$$

This equation illustrates that when fluid passes through smaller and larger vessels at the same Q, the smaller vessels have higher Re and thus are more prone to turbulence. Additional conditions that promote turbulence include wall irregularities, abrupt changes in tube dimensions, and disturbed flow upstream to the region of interest.

Consider the example of an access with $\rho = 1.056 \text{ g/cm}^3$, $Q = 1,600 \text{ ml/min}$, $D = 0.5 \text{ cm}$, and $\eta = 0.035 \text{ poise}$ [corresponding to hematocrit (Hct) = 33%]. It follows that $Re = 2,049$. Thus, Re is at the transition threshold, and flow may be laminar, turbulent, disturbed, or alternate between types of flow. Because resistance is higher in turbulent than laminar flow, these transitions contribute to variations in Q.

Stenoses may occur along the access circuit in a manner analogous to resistances in series in an electrical circuit [3]. Provided these stenoses are a minimum distance apart [5], the total vascular resistance equals the sum of the individual resistances:

$$R_{\text{total}} = R_1 + R_2 + R_3 + \dots = \Sigma R_i$$

At the entrance to a stenosis, flow accelerates and pressure falls (recall the Bernoulli equation). The conversion of pressure (potential energy) into kinetic energy is efficient, so that there is minimal energy loss at the entrance to the stenosis. However, as blood exits the stenosis and decelerates, there is frictional energy loss because the conversion of kinetic energy back into pressure is less efficient [5].

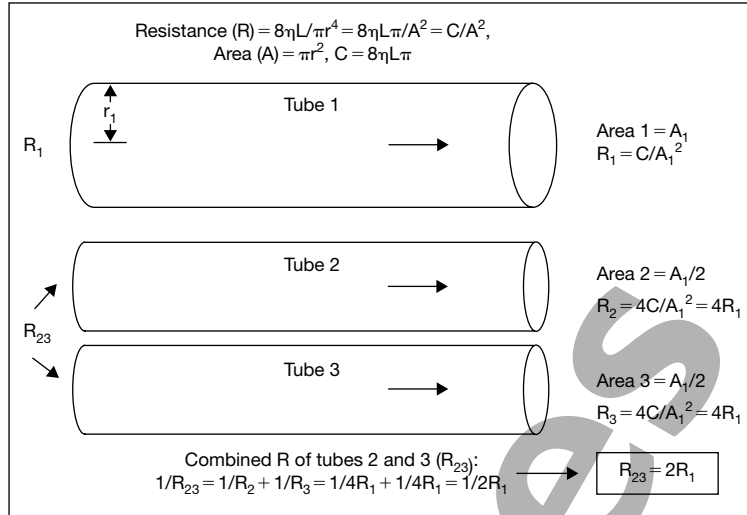


Fig. 2. Although luminal cross-sectional area of tube 1 (A_1) equals total cross-sectional area of tubes 2 and 3, resistance of tube 1 (R_1) is 1/2 combined resistance of tubes 2 and 3 (R_{23}). L is length along tube, r is radius, η is dynamic viscosity.

Because of this frictional loss, ΔP just downstream of the stenosis exit is greater than ΔP within the stenosis (where frictional loss is induced by wall shear stress). Thus, a single stenosis with length L has less resistance than two stenoses that both have length $L/2$ because the two stenoses contribute two larger exit ΔP s. This suggests that correcting two short stenoses may yield a better result than correcting a single longer stenosis.

Continuing the analogy with electrical circuits, resistances can also be in parallel [3]. A clinical example is outflow from an autogenous arteriovenous (AV) fistula that is carried in two veins rather than in a single vein. For parallel resistances, the total R is given by the equation:

$$1/R_{total} = 1/R_1 + 1/R_2 + 1/R_3 + \dots = \Sigma(1/R_i)$$

Recall that from Poiseuille's law, $R = 8\eta L / \pi r^4$. It follows that if two tubes have a total luminal cross-sectional area equal to that of a single larger tube, then the total R of the two tubes is twice that of the single tube (fig. 2). Four of the smaller tubes would be needed to provide R as small as the larger tube. Thus, a fistula with two outflow veins has a higher R than a fistula with one outflow vein of an equal overall cross-sectional area. It follows that a single outflow vein may provide an adequate Q whereas two outflow veins may not.

Dilatation in Access Vascular Circuit

Creation of an access activates mechanisms that cause dilatation of the inflow artery and outflow vein. Q initially increases because the new circuit has a low resistance. This increase in Q causes a high shear stress on the endothelial surface. The shear stress can be estimated from Poiseuille's law [2]:

$$\text{shear stress} = 4\eta Q/\pi r^3$$

Because shear stress is proportional to $1/r^3$, small increases in diameter can offset the effect of an increase in Q .

Arteries adjust their diameters to maintain shear stress between 10 and 20 dyn/cm² [6, 7]. Although dilatation of the outflow vein has been attributed to increased pressure, it is possible that the vein also dilates in response to high shear stress [8]. This mechanism may explain why procedures that occlude accessory veins promote AV fistula maturation. Since these veins divert part of the outflow, occlusion increases Q through the small main outflow vein, thereby increasing shear stress. A larger diameter fistula is then produced when dilatation causes shear stress to return to normal.

The mechanism of acute dilatation is primarily endothelial release of nitric oxide, which relaxes smooth muscle [9]. Over a longer period, remodeling of the vascular wall changes its cellular and matrix composition [10, 11].

Model of Graft Vascular Circuit

The access circuit is a low resistance shunt between the arterial and venous circulations. The mean ΔP that drives flow in this shunt is the same as in the normal systemic circulation: mean arterial pressure (MAP) minus central venous pressure (CVP). Since $\Delta P = QR$, it follows that the large reduction in R must be matched by a large increase in Q . In addition, pressure must fall rapidly in the large arteries and veins of the access circuit so that CVP is reached by the time flow reaches the vena cava. This large ΔP contrasts with the normal systemic circulation in which up to 60% of ΔP is in the arterioles and 15% is in the capillaries, but only approximately 10% is in the arteries and 15% is in the veins [2].

Thus, the access vascular circuit has unique hemodynamic properties that must be taken into account when managing access problems. A model of the circuit can provide valuable insight into these properties. The engineering literature provides pressure-flow equations for hydrodynamic elements that are analogous to elements in the access circuit. We have applied these equations to a model of the synthetic graft vascular circuit in the upper extremity.

Table 1. Dimensions of segments in model of synthetic graft vascular circuit

Segment	Length cm	Luminal diameter, cm
Artery	40.0	0.5
Graft	30.0	0.5
Stenosis at venous anastomosis	1.0	0–0.5
Vein	40.0	0.7

The circuit can be modeled as a series of ΔP s. The total ΔP equals the sum of ΔP s from each segment:

$$\text{total } \Delta P = \text{MAP} - \text{CVP} = \Delta P_A + \Delta P_{AA} + \Delta P_G + \Delta P_{VA} + \Delta P_S + \Delta P_V$$

The subscripts indicate: A = artery, AA = arterial anastomosis, G = graft, VA = venous anastomosis, S = stenosis at venous anastomosis, V = vein. This model includes the stenosis that commonly occurs at the venous anastomosis. Given that flow is normally pulsatile, calculations of Q and pressure in the model should be considered time-averaged. The model ignores special circumstances, such as vessel elasticity, tapering or tortuous vessels, and variable diameters.

Dimensions of model segments and stenosis are shown in table 1. The luminal diameters are similar to values we have measured in our patients [12]. In the graft, the inflow artery usually has a smaller diameter than the outflow vein. In contrast, in the AV fistula, the two diameters are generally approximately equal. Thus, the fistula differs from the model in that the inflow and outflow resistances are approximately equal.

We modeled each segment with an appropriate ΔP equation (table 2). These equations are more complex than Poiseuille's law, and involve Q raised to various powers. Since R is defined as $\Delta P/Q$, it is clear that resistance is not constant, but rather increases with Q. The inflow artery and graft have luminal diameters that may yield Reynolds numbers above and below the 2,000 transition (recall that Re is proportional to $1/D$). Thus, we considered both laminar entry flow and fully developed turbulent flow in these segments [4]. In the outflow vein, we considered only laminar entry flow because the vein has a larger diameter that should yield a lower Re. The ΔP s of the two anastomoses were modeled with a T junction equation [13]. A T junction treats ΔP across the junction of two tubes, which is analogous to the anastomoses. Viscosity was adjusted for Hct [14].

Table 2. Equations used to model pressure drop (ΔP) at each segment of graft vascular circuit

Inflow artery and graft

Shah's laminar entry-flow equation (for Reynolds No. $<2,000$) [4]

$$\Delta P_A = h_1 Q^{3/2} + (h_2 Q + h_3 Q^2 + h_4 Q^{3/2}) / (1 + h_5 Q^2)$$

Blasius's fully developed turbulence equation (for Reynolds No. $>2,000$) [4]

$$\Delta P_A = (0.0665 \rho^{3/4} L \eta^{1/4} / \pi^{7/4} r^{4.75}) Q^{7/4}$$

Arterial anastomosis: T junction equation [13]

$$\Delta P_{AA} = (8 \rho K_1 / \pi^2 D_A^4) Q^2$$

Venous anastomosis: T junction equation [13]

$$\Delta P_{VA} = (8 \rho K_1 / \pi^2 D_G^4) Q^2$$

Stenosis at venous anastomosis: Young's equation [5]

$$\Delta P_S = (8 K_1 \rho / \pi^2 D^4) [(D_V / D_S)^2 - 1]^2 Q^2 + (4 K_V \eta / \pi D_V^3) Q$$

Outflow vein: Shah's laminar entry-flow equation [4]

$$\Delta P_A = h_1 Q^{3/2} + (h_2 Q + h_3 Q^2 + h_4 Q^{3/2}) / (1 + h_5 Q^2)$$

D_A , D_G , D_S , D_V = Luminal diameters of artery, graft, stenosis, and vein, respectively; L = length of circuit segment; h = variables that depend upon viscosity, density, length of tube or circuit segment, and luminal diameter. K_1 depends on luminal diameter; K_V depends on luminal diameters of vein and stenosis, and length of stenosis.

The ΔP equation for the entire circuit is a function of Q and the other variables and constants that characterize the circuit (table 2):

$$\text{MAP} - \text{CVP} = f(Q, Q^Z, X_i, C_i)$$

in which Z indicates various powers of Q , and X_i and C_i represent variables and constants. We assumed $\text{MAP} = 100 \text{ mm Hg}$ ($1.33 \times 10^5 \text{ dyn/cm}^2$) and $\text{CVP} = 0 \text{ mm Hg}$.

Quantitative Predictions of Model

Figure 3 shows the predicted ΔP s for circuits with and without stenosis (percent reduction in luminal diameter). Laminar entry flow was assumed for the inflow artery and graft. The figure demonstrates the unique hemodynamics of the access circuit. In the normal systemic circulation, ΔP from the ascending aorta to the origin of the arterioles is normally small. In the access circuit,

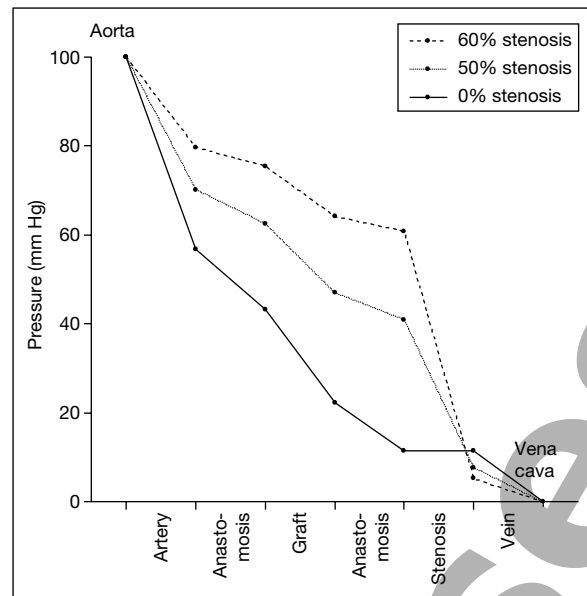


Fig. 3. Pressure drops (ΔP s) across each segment in model of graft vascular circuit. Total ΔP is MAP - CVP. MAP = 100 mm Hg, CVP = 0 mm Hg, Hct = 33%.

however, the high Q is accompanied by a large dissipation of energy so that a large ΔP occurs before flow reaches the graft. This agrees with the in vivo observation that the pressure in the arterial limb of a graft is 45% of MAP [15]. Because of the relatively small luminal diameter of the inflow artery [12], the stenosis ΔP does not match the artery ΔP until stenosis is $>50\%$.

We also computed the inverse of the ΔP equation: $Q = f(\Delta P_i, X_i, C_i)$. Figure 4 shows the solution for Q as a function of stenosis and MAP. The curve is sigmoid, so that it is initially relatively flat. As stenosis increases, however, the rate of decrease in Q accelerates. The slope is greatest at approximately 50% stenosis, where the risk of thrombosis becomes significant. The sigmoid (rather than linear) relation is important because it reduces the time interval during which Q monitoring can warn of impending thrombosis. Figure 4 also shows that MAP has a large effect on Q . Thus, a stable and reproducible Q measurement requires hemodynamic stability. Discontinuity in Q is shown because the model assumes transition from turbulent to laminar entry flow occurs as the Reynolds number falls below 2,000. Because resistance is lower in laminar flow, Q increases at this transition.

Figure 5 shows Q as a function of stenosis and Hct. Laminar entry flow was assumed for the inflow artery and graft. Hct influences Q because viscosity increases with Hct [14]. The influence is substantial at low stenosis, but

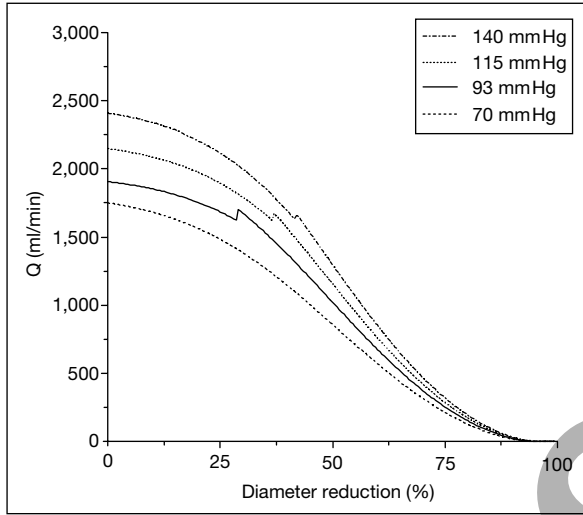


Fig. 4. Effect of MAP and stenosis on Q in model of graft vascular circuit. Hct is 35%.

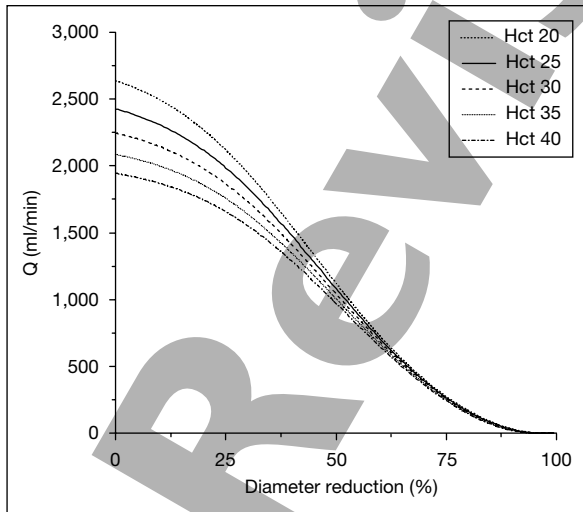


Fig. 5. Effect of Hct and stenosis on Q in model of graft vascular circuit. MAP is 100 mmHg.

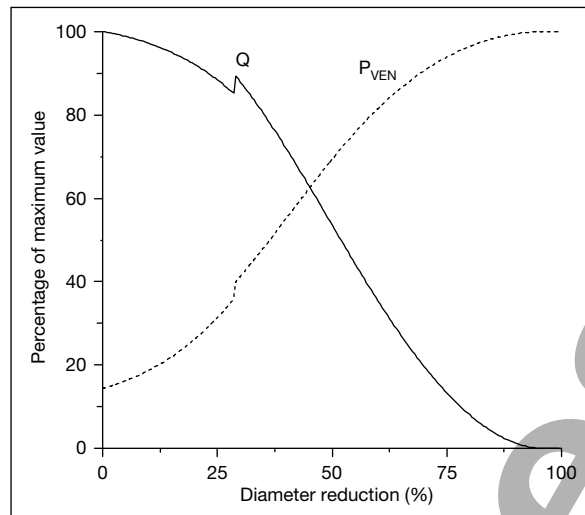


Fig. 6. Effect of stenosis on Q and intragraft pressure (P_{VEN}) in model of graft vascular circuit. Pressure is just upstream to stenosis at venous anastomosis. MAP is 100 mm Hg, Hct is 35%.

decreases as stenosis progresses. Thus, Hct should not have a significant effect on Q when stenosis is severe. Hct should have a lesser effect on Q in turbulent flow because viscous effects are reduced (not shown).

Venous drip-chamber pressure is also used to detect stenosis [16]. By turning off the dialysis blood pump, the drip-chamber pressure becomes equal to intra-access pressure (the pressure must be adjusted for the height relative to the graft). Figure 6 compares Q with intragraft pressure just upstream to the stenosis at the venous anastomosis. Pressure is largely the inverse of Q , indicating that venous pressure monitoring should, in principle, be equivalent to Q monitoring. Q has an advantage, however, in that it does not depend on the location of stenosis. For example, a stenosis that is upstream to the venous dialysis needle will not cause an increase in venous drip-chamber pressure. As in figure 4, discontinuities in curves are due to transition from turbulent to laminar entry flow.

Clinical Correlations

It is widely recommended that surveillance programs be used to detect access dysfunction so that stenosis can be corrected before thrombosis [17]. Grafts are most likely to benefit from such programs because they have a higher failure rate than established AV fistulae. The most popular surveillance method has been monthly Q measurements. Although low Q and decrease in Q are

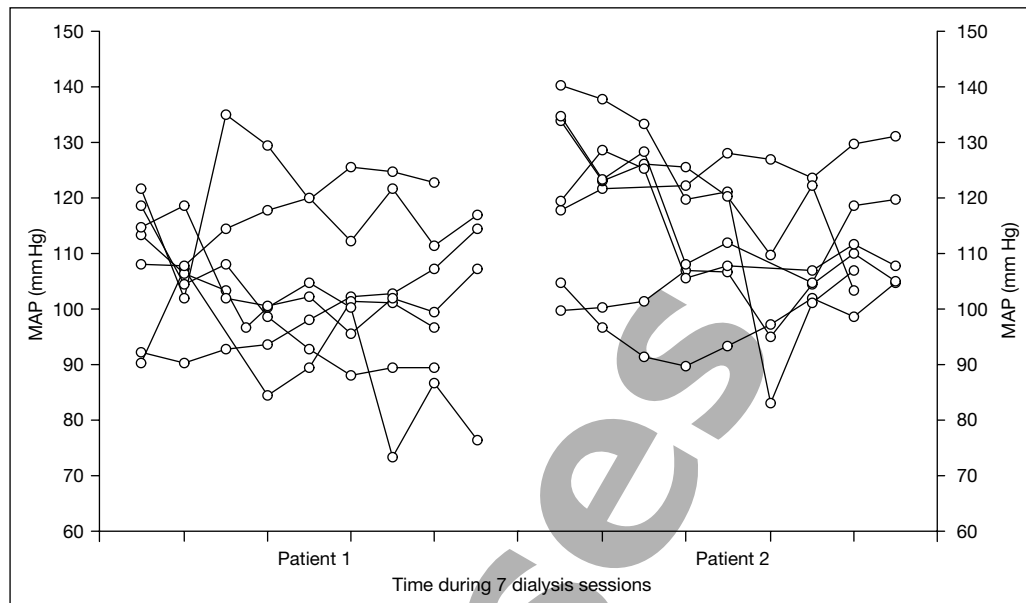


Fig. 7. MAPs of two representative patients during 7 consecutive dialysis sessions [reproduced with permission of the National Kidney Foundation, 22].

important risk factors for graft thrombosis, we and others have shown that Q has a poor predictive accuracy [18–21]. This result is consistent with the model, which predicts that variable hemodynamic conditions will strongly impair the relation between Q and stenosis.

Several studies have documented that variable hemodynamic conditions are indeed present during dialysis, when Q is usually measured [22–24]. The wide MAP range of 70–140 mmHg in figure 4 is similar to the range we observed in 51 patients during 7 dialysis sessions [22]. The pooled within-patient MAP SD was 13.7 mmHg, so that the average range within individual patients was approximately ± 27 mmHg. Figure 7 shows MAPs from two representative patients [22].

Resistance also varies widely during dialysis (fig. 8). Much of this variation is probably caused by changes in constriction of vessels that carry blood to and from the graft [25]. Transitions between laminar and turbulent flow may also contribute. Since $Q = (\text{MAP} - \text{CVP})/R$, it follows that wide variation in MAP and R should cause considerable variation in Q .

Several studies have confirmed that Q varies widely within a single dialysis session [22–24]. For example, figure 9 shows changes in Q_a and MAP between the first 30 and 90 min of a session [24]. Twenty-four percent of patients

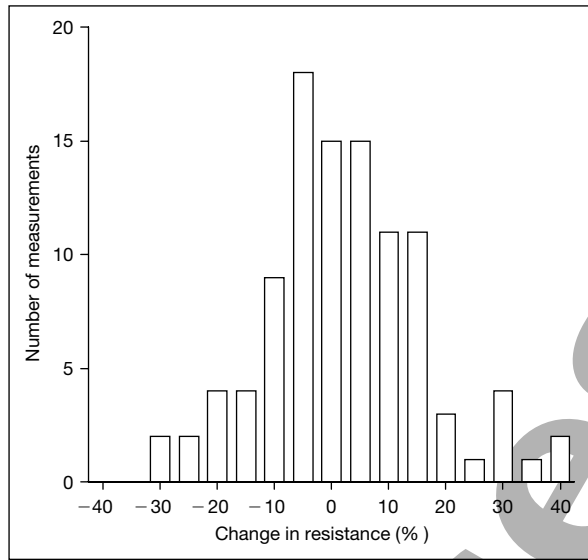


Fig. 8. Percent change in vascular resistance during dialysis in 51 patients with grafts. Data were obtained from a study of DeSoto et al. [22]. Changes in resistance were computed between first and middle thirds, and first and last thirds, of dialysis session. Resistance was defined as $(MAP - CVP)/Q$, with CVP set equal to 0.

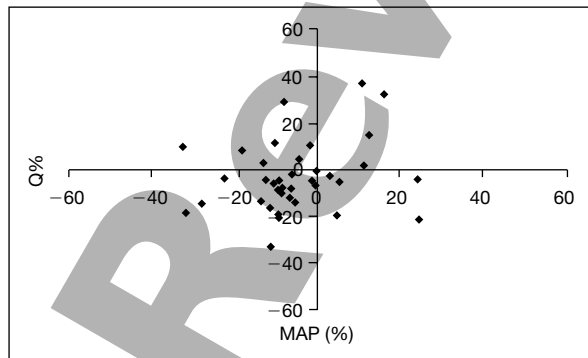


Fig. 9. Percent change in Q vs. percent change in MAP from 30 to 90 min after start of a dialysis session. Twenty patients had grafts, 12 had AV fistulae [reproduced with permission of the National Kidney Foundation, 24].

had changes in Q of at least 18%. Changes in Hct may also contribute to Q variation over a period of weeks or months. Measuring Q early at the same time in a session will not improve Q reproducibility because most of MAP variation is already present with the first MAP measured at the beginning of dialysis [22].

The foregoing illustrates the challenge in using access function to predict risk of thrombosis or failure. Access hemodynamics are unstable and subject to rapid changes during dialysis, when function is most commonly assessed. Measurement of Q more frequently than the standard monthly protocol would improve recognition of trends. However, this is generally impractical at this time and will probably have to await availability of easy and accurate on-line Q measurements.

There are other lessons to be drawn from hemodynamics of the access circuit. For grafts, the most severe stenoses usually occur at the venous anastomosis or outflow vein. However, because luminal diameters are usually largest in the vein [12], stenosis must be severe before it dominates resistance in the circuit. On the other hand, stenoses do commonly occur upstream to the venous anastomosis. Given the narrower luminal diameters in the upstream segments, it follows that even mild stenoses can have a significant impact on resistance. Thus, the interventionist has many issues to consider when treating stenoses with angioplasty. Not only must relative luminal diameters be considered, but as previously discussed, two or more short stenoses may have a greater influence on resistance than a single longer stenosis.

Access circuit hemodynamics can also be an important cause of complications, such as the steal syndrome. Consider a forearm graft anastomosed to the proximal radial artery. As figure 3 shows, low resistance causes a high Q with large ΔP before flow reaches the graft. Thus, the pressure in the radial artery distal to the graft is much lower than in the normal circulation. This low pressure induces retrograde flow from the digital and palmar arch arteries through the radial artery back to the graft. Reduced hand perfusion may result in ischemia, especially if arterial vascular disease is present in the hand [26]. A common method of preventing the high Q steal syndrome is to ligate the radial artery distal to the access.

Conclusion

The access vascular circuit has unique hemodynamic properties that can be analyzed according to principles of fluid mechanics. Concepts such as vascular resistance and shear stress help explain access function and maturation. The model of the graft circuit describes the factors that influence Q and pressure measurements, and explains why graft surveillance often fails to predict graft thrombosis and failure. The large ΔP in the access circuit plays an essential role

in circuit hemodynamics and explains reduced distal perfusion in the steal syndrome. Consideration of hemodynamic principles can help in planning an optimum approach to preventing and treating access problems.

Appendix: Definitions and Units of Measurement

A	Cross-sectional area of tube or access (cm ²)
CVP	Central venous pressure (1 mm Hg = 1,333 dyn/cm ²)
D	Luminal diameter (cm)
ΔP	Pressure drop across part of access vascular circuit (1 mm Hg = 1,333 dyn/cm ²)
L	Length of tube or circuit segment (cm)
MAP	Mean arterial pressure (1 mm Hg = 1,333 dyn/cm ²)
η	Dynamic viscosity (1 poise = 1 dyn·s/cm ² = 1 g/cm/s)
P	Pressure, defined as force exerted by fluid against an area A (1 mm Hg = 1,333 dyn/cm ² = 1,333 g·cm/s ² ·cm ²)
ρ	Density of blood (1.056 g/cm ³)
Q	Rate of flow = vA = vπr ² (1 cm ³ /s = 1 ml/s = 60 ml/min)
R	Vascular resistance [1 peripheral resistance unit (PRU) = 1 mm Hg/ml/min = 8 × 10 ⁴ dyn·s/cm ⁵]
r	Luminal radius (cm)
Re	Reynolds number, defined as ratio of inertial to viscous forces = $2\rho rv/\eta = \rho Q/30\pi r\eta$ (dimensionless)
Shear rate	$\Delta v/\Delta x$ (s ⁻¹) = change in fluid velocity (Δv) per change in distance (Δx) perpendicular to the direction of flow
Shear stress	F/A (dyn/cm ²) = force needed to move two layers of fluid of equal area A across each other
v	Mean cross-sectional velocity of fluid (cm/s)

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William D. Paulson, MD,
Department of Medicine,
Louisiana State University Health Sciences Center,
1501 Kings Highway, Shreveport, LA 71130 (USA)
Tel. +1 318 675 5911, Fax +1 318 675 5913, E-Mail wpauls@lsuhsc.edu