

Original Article

## Inflow stenosis obscures recognition of outflow stenosis by dialysis venous pressure: analysis by a mathematical model

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### Abstract

**Background.** Recent studies have shown that inflow stenosis of haemodialysis grafts is more common than previously realized. The influence of inflow stenosis on graft haemodynamics and venous pressure (VP) surveillance has not been previously systematically studied.

**Methods.** We used a well-established mathematical model to determine the relation between inflow stenosis and static VP (adjusted for mean arterial pressure, VP/MAP), outflow stenosis and artery and vein luminal diameters. We applied low, median and high ratios of artery/vein diameters from 94 patients with grafts. The median ratio was 0.77, indicating that the artery was generally narrower than the vein.

**Results.** The model shows that inflow stenosis reduces VP/MAP. More importantly, however, as outflow stenosis progresses, fixed inflow stenosis causes a delayed increase in VP/MAP followed by a rapid increase at critical outflow stenosis. When both stenoses progress together, their relative rates determine whether and how rapidly VP/MAP increases. The increase in VP/MAP is remarkably abrupt when the rate of inflow stenosis approaches that of outflow stenosis. No increase occurs when inflow stenosis progresses as fast or faster than outflow stenosis.

**Conclusion.** Inflow stenosis exerts its most important haemodynamic effect through its interaction with outflow stenosis. As outflow stenosis progresses, inflow stenosis causes a delayed and then rapid increase in VP/MAP at critical outflow stenosis. This increase may not be detected before thrombosis unless measurements are very frequent. Inflow stenosis has an important impact on graft haemodynamics and surveillance because of its location in the relatively narrow inflow tract.

**Keywords:** access surveillance; arteriovenous graft; haemodialysis; haemodynamics

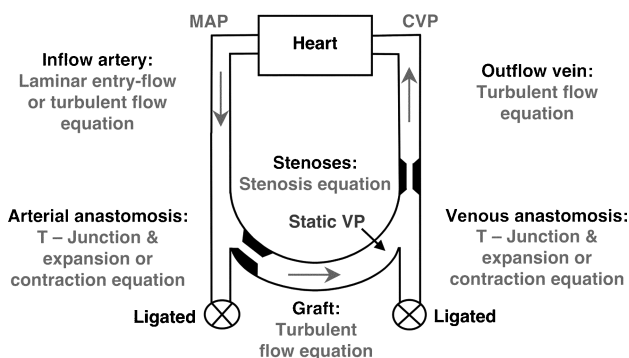
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### Introduction

Stenosis of haemodialysis synthetic grafts most commonly develops at the venous anastomosis or outflow vein. Dialysis venous pressure (VP) measurements are often used to detect such stenosis so that it can be corrected before thrombosis [1–3]. The National Kidney Foundation's K/DOQI Guidelines [4] recommend referral when the ratio of static VP to mean arterial pressure (VP/MAP) is  $>0.50$ . However, the ratio of artery/vein luminal diameters varies widely between patients, and the ratio independently controls VP/MAP [5]. Thus, the standard referral threshold of 0.50 does not indicate a particular level of stenosis. Intervention referrals should be based upon whether VP/MAP has significantly increased rather than whether a particular threshold has been crossed [4,5]. The importance of trend analysis further follows from the observation that the inflow artery is generally narrower than the outflow vein, and a narrower artery increases flow resistance [5,6]. This resistance causes an initially lower VP/MAP, with a longer delay followed by a more rapid increase in VP/MAP as critical stenosis is reached and thrombosis becomes likely. Thus, prevention of thrombosis requires recognition of a rapid increase in VP/MAP.

Although the outflow tract is the most common location of stenosis, recent studies have shown that inflow stenosis is more common than previously realized [7–9]. Asif *et al.* [7] found stenosis near or within the arterial anastomosis in 36/122 (29%) of grafts referred for intervention. Duijm *et al.* [9] targeted more central lesions that were probably atherosclerotic and found 3/35 (8.6%) of referred grafts had inflow stenosis. Other common forms of inflow stenosis are tapered grafts with a narrow inflow [10] and treatment of steal syndrome with a band applied to the inflow of a graft.

Because inflow stenosis increases vascular resistance, it is widely accepted that detection of inflow stenosis is obscured because it reduces rather than increases VP/MAP [3,4,11]. However, 77–100% of patients with inflow stenosis reportedly also have outflow stenosis [7,12]. Thus, the



**Fig. 1.** Model of graft vascular circuit. Figure indicates equations used to compute pressure drops ( $\Delta P$ s) across each segment of circuit. Sum of all  $\Delta P$ s equals MAP – CVP. Static VP indicates pressure at venous dialysis needle with blood pump turned off. Adapted from Figure 1 of Jones *et al.* [12] with permission of American Society of Mechanical Engineers.

haemodynamic effect of inflow stenosis largely depends on its interaction with outflow stenosis. The importance of this interaction is supported by clinical data that document the opposing influences of inflow and outflow stenosis on VP/MAP [12,13].

The fact that the inflow artery and graft are generally narrower than the outflow vein [5,6] further emphasizes the importance of inflow stenosis. Inflow stenosis has a larger influence on resistance than the same percentage stenosis at the venous anastomosis or outflow vein. It follows that inflow stenosis is particularly likely to promote thrombosis. Moreover, the transition from a usually narrow inflow artery to a wider graft makes it difficult to assess the significance of such stenoses during intervention procedures. In discussing these issues, Khan *et al.* [8] concluded that identification and treatment of inflow stenosis are at least as important as treatment of outflow stenosis.

The foregoing shows that it is important to determine the influence of inflow stenosis on surveillance. Specifically, one would predict that because inflow stenosis reduces VP/MAP, it impairs the increase in VP/MAP induced by outflow stenosis. The effect of inflow stenosis is likely to be complex when the interacting influences of outflow stenosis and luminal diameters are considered. This effect has not been evaluated in clinical studies because such complex interactions are not easily discerned from data on patients. However, a mathematical model allows one to control conditions so that key relationships can be determined. In this study, we addressed these issues by using a mathematical model to determine the relation between inflow stenosis and static VP/MAP, outflow stenosis and luminal diameters.

## Subjects and methods

The mathematical model and its validation have been previously described [5,6,14]. The model includes the inflow artery, arterial and venous anastomoses, graft and outflow vein (Figure 1). It resembles a loop graft anastomosed end-to-side to the brachial artery and cubital vein for forearm configuration, or brachial artery and basilic or axillary vein for upper arm configuration. For sim-

licity, 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 to graft flow. The model predicts pressures in the graft circuit that are in good agreement with clinical studies [5,6].

In previous applications of the model, the only stenosis was placed in the vein just downstream to the venous anastomosis. In this study, we added an inflow stenosis to the graft adjacent to the arterial anastomosis. This location downstream to the anastomosis simplified the model by avoiding the complex interaction of stenosis with the anastomosis or with the junction of the graft and ligated artery.

The luminal diameters of vessels in the graft circuit have a strong influence on graft haemodynamics, and the ratio of artery/vein diameters controls the relation between VP and stenosis [5]. We assumed a uniform graft diameter of 0.60 cm, and used previously selected artery and vein diameters from duplex ultrasound studies of 94 patients with grafts (Table 1) [5,6]. Luminal diameters varied widely, but the artery was generally narrower than the vein. We used the median artery/vein ratio (0.77), and low (0.40) and high ratios (1.28) that enclose 95% of patients.

The model uses pressure-flow equations from the engineering literature that have been refined by data from an *in vitro* apparatus [14]. The model is defined by a total pressure drop equation ( $\Delta P_{\text{TOTAL}}$ ) that is the sum of pressure drops across all segments of the circuit [5,6,14]:




$$\begin{aligned} \Delta P_{\text{TOTAL}} = \text{MAP} - \text{CVP} = & \Delta P_{\text{ARTERY}} + \Delta P_{\text{AA}} \\ & + \Delta P_{\text{INFLOW STENOSIS}} + \Delta P_{\text{GRAFT}} + \Delta P_{\text{VA}} \\ & + \Delta P_{\text{OUTFLOW STENOSIS}} + \Delta P_{\text{VEIN}}. \end{aligned}$$

CVP denotes central VP; subscript AA denotes arterial anastomosis; VA denotes venous anastomosis. The pressure just upstream to the venous anastomosis was taken to be static VP. Static VP is the intragraft pressure at the venous dialysis needle with the blood pump turned off.

The  $\Delta P_{\text{TOTAL}}$  equation determines relations between the variables that characterize the circuit: flow rate, circuit pressures, stenoses, luminal diameters, haematocrit and other variables and constants. The lengths of the artery, graft, vein and stenoses were set equal to 40, 34, 40, and 1 cm, respectively. We defined stenosis as percentage reduction in luminal diameter when compared with the adjacent graft for inflow stenosis and adjacent vein for outflow stenosis. Conditions were haematocrit = 36%, CVP = 5 mmHg and MAP = 93 mmHg (corresponding to systolic pressure = 120 mmHg and diastolic pressure = 80 mmHg).

The  $\Delta P$  equations for the artery, graft and vein depend upon the nature of the flow in each segment (laminar or turbulent). A minimum entrance length is required for laminar flow to fully develop. For most large arteries, the entrance length approaches the length of the artery, so that laminar flow is usually not fully developed. We used Shah's laminar entry-flow equation to model such flow [15,16]. We used a modified Blasius equation to model turbulent flow [14,17]. An *in vitro* study [14] showed that the graft and vein exhibit turbulent flow whereas the artery may exhibit laminar

**Table 1.** Hypothetical luminal diameters and diameter ratios that were used in mathematical model

| Type of artery/vein ratio | Inflow artery diameter (cm) | Outflow vein diameter (cm) | 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  |

Ratios were obtained from duplex ultrasound studies of 94 patients [6]. Low and high ratios enclose 95% of patients.

entry-flow or turbulent flow, depending on the Reynolds number:  $Re = \rho Q / 15\pi D \mu$  ( $\rho$  is blood density in  $\text{g/cm}^3$ ,  $Q$  is flow in mL/min and  $D$  is diameter in cm) [18]. We used Shah's equation when  $Re < 1500$  [15,16] and used the modified Blasius equation when  $Re \geq 1500$  [14,17]. Transitions between laminar entry-flow and turbulent flow account for discontinuities in some of the VP/MAP figures in the Results section. We modeled stenoses with a modified Young's equation [14,19]. Anastomoses were modelled by adding two equations together: a T-junction equation that defines  $\Delta P$  across the junction of two tubes [20], plus  $\Delta P$ s caused by increases [21] or decreases [22] in luminal diameter (Bernoulli's law [18]).

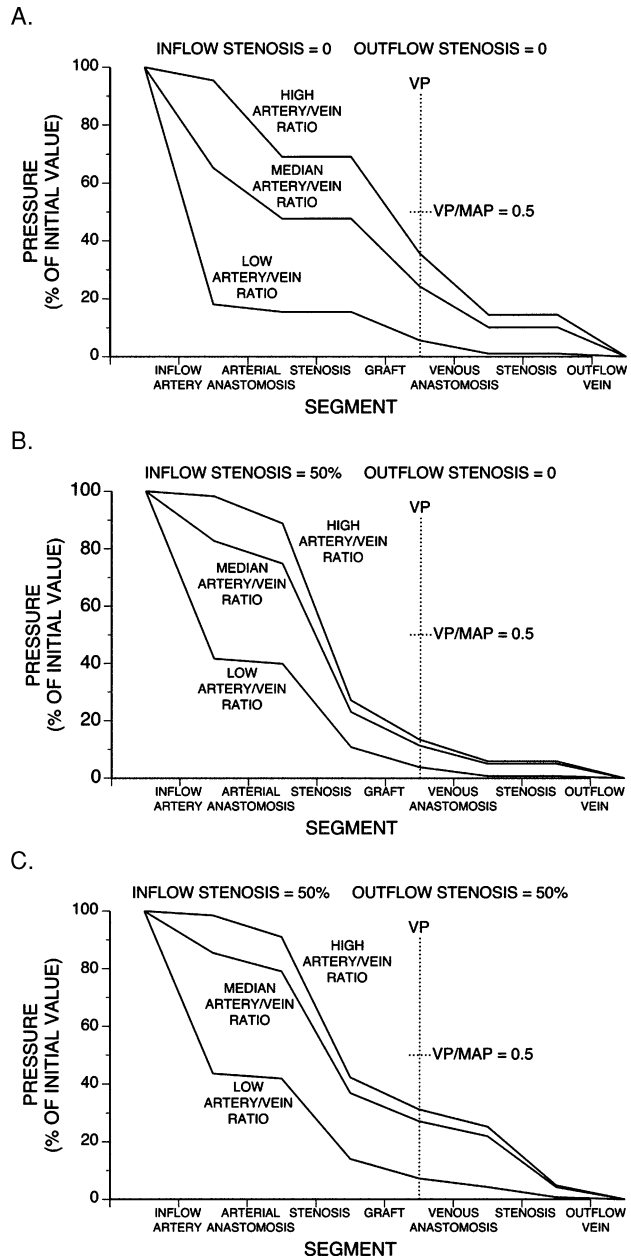
### Analysis

The  $\Delta P_{\text{TOTAL}}$  equation was used to compute relations between inflow and outflow stenoses, pressures, flow rate and luminal diameters. After fully defining the equation (Figure 1 and Table 1), we used Microsoft Excel Solver (the generalized reduced gradient nonlinear optimization code) to determine these relations. Solver (Frontline Systems, Inc., Incline Village, NV, USA) is an add-in to Microsoft Excel that uses iterative methods to optimize solutions to nonlinear equations.

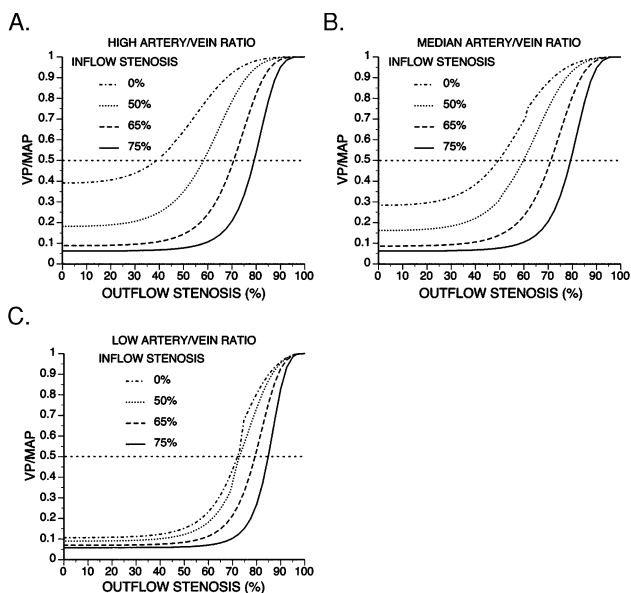
### Results

This study determined the relation between inflow stenosis and static VP/MAP, outflow stenosis and the artery/vein diameter ratio. We used low, median and high ratios from previous studies (Table 1) [5,6]. The mathematical model predicts that as blood flows through the circuit, energy is dissipated and pressure falls from the initial level (MAP) to the final level (CVP). In the absence of stenosis, luminal diameters control the pressures in the circuit through their influence on vascular resistance (Figure 2A). At the median artery/vein ratio, the artery is narrower than the graft and vein, so the largest pressure drop is in the artery. Flow resistance increases as the artery narrows (lower artery/vein ratios), so that the artery accounts for a larger proportion of the total pressure drop.

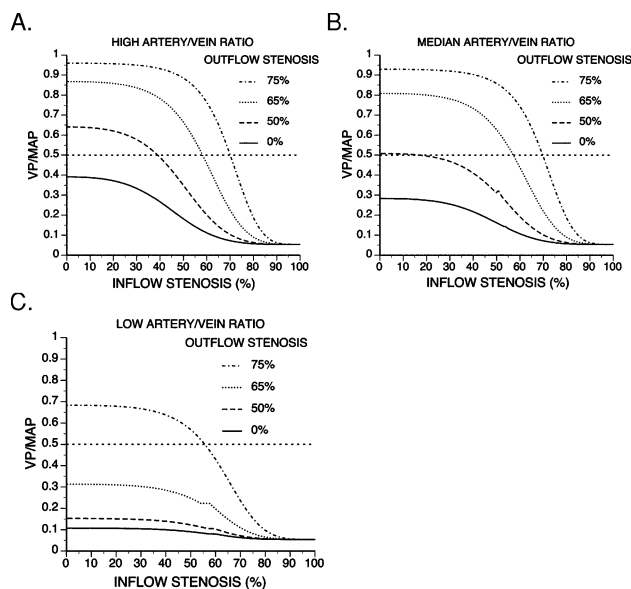
Addition of 50% inflow stenosis yields a lower VP because the stenosis increases resistance (Figure 2B). At high artery/vein ratios, the majority of the total pressure drop is caused by the inflow stenosis. Addition of 50% outflow stenosis to the inflow stenosis increases VP (Figure 2C).



**Fig. 2.** Predicted pressure along segments of graft circuit at artery/vein ratios in Table 1. Conditions in this and following figures were: initial pressure = MAP = 93 mmHg, final pressure = CVP = 5 mmHg, hematocrit = 36%. Vertical dotted line indicates location of static VP. VP/MAP = 0.50 indicates standard referral threshold [4].



**Fig. 3.** Predicted effect of fixed inflow stenosis and artery/vein ratio on relation between VP/MAP and outflow stenosis. As inflow stenosis is increased or artery/vein ratio is decreased, VP/MAP decreases and curve shifts to the right. Dotted line indicates VP/MAP = 0.50 (standard referral threshold [4]).



**Fig. 4.** Predicted effect of fixed outflow stenosis and artery/vein ratio on relation between VP/MAP and inflow stenosis. As outflow stenosis or artery/vein ratio is increased, VP/MAP increases. Progressive inflow stenosis reduces and ultimately prevents this increase. Dotted line indicates VP/MAP = 0.50 (standard referral threshold [4]).

However, VP remains less than when there is no stenosis in the circuit (Figure 2A), so that VP/MAP remains far below the standard referral threshold of 0.50 [4]. Thus, the model predicts that inflow stenosis obscures detection of outflow stenosis by reducing VP/MAP.

It is important to determine how VP/MAP varies at different levels of inflow and outflow stenosis. First, consider the influence of fixed inflow stenosis on the relation between VP/MAP and progressive outflow stenosis (Figure 3). The figure shows that as inflow stenosis increases, VP/MAP decreases and the curve shifts to the right. A lower artery/vein ratio (narrower artery) has a similar effect. Thus, VP/MAP may not cross the 0.50 referral threshold until critical outflow stenosis is reached.

Further insight is obtained by considering the influence of fixed outflow stenosis on the relation between VP/MAP and progressive inflow stenosis (Figure 4). The figure shows that VP/MAP rises as outflow stenosis increases. However, VP/MAP falls as inflow stenosis progresses, so that even critical outflow stenosis eventually fails to increase VP/MAP above the referral threshold. This effect is augmented by a low artery/vein ratio.

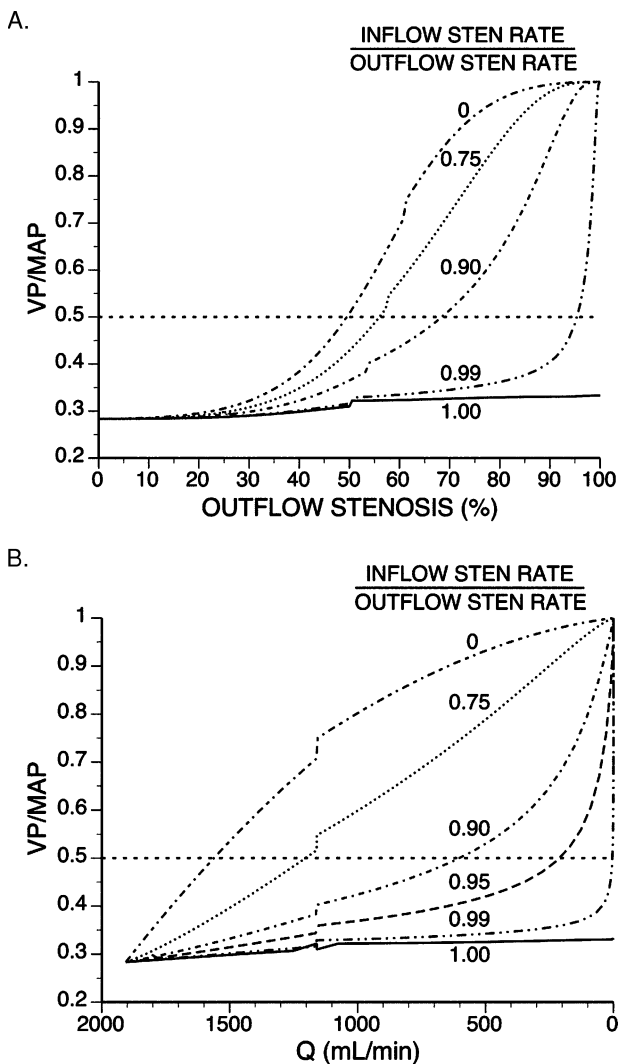
The foregoing analysis varies one of the stenoses while holding the other fixed. It is informative to consider the effect on VP/MAP when both stenoses progress together (Figure 5). Each curve in the figure represents a different rate of inflow stenosis relative to outflow stenosis. As the rate of inflow stenosis approaches that of outflow stenosis, the VP/MAP versus outflow stenosis curve shifts to the right (Figure 5A). Thus, inflow stenosis delays the increase in VP/MAP induced by outflow stenosis. Moreover, inflow stenosis prevents any significant increase when the two stenoses progress at the same rate.

Figure 5B shows the relation between VP/MAP and flow when the two stenoses progress as in Figure 5A. Flow falls to 0 as outflow stenosis progresses to 100%. However, as the rate of inflow stenosis approaches that of outflow stenosis, a given flow is associated with lower VP/MAP. Moreover, when the two stenoses progress at the same rate, flow falls to 0 but VP/MAP does not significantly increase. Thus, flow falls regardless of stenosis locations, but VP/MAP may or may not increase depending on their locations relative to the venous dialysis needle.

### Discussion

This study was prompted by recent observations that inflow stenosis is more common than previously realized [7–9]. Moreover, the inflow tract dominates vascular resistance in the graft circuit because it is generally narrower than the outflow tract. The mathematical model confirms the concept that inflow stenosis reduces VP/MAP [12,13]. The key result, however, is that inflow stenosis obscures detection of progressive outflow stenosis because it causes a delay followed by a rapid increase in VP/MAP at critical outflow stenosis. Assuming outflow stenosis progresses at a constant rate, this rapid increase may be difficult to detect before thrombosis unless VP measurements are very frequent. Inflow stenosis that progresses as fast or faster than outflow stenosis prevents any increase in VP/MAP.

The effect of inflow stenosis on VP/MAP follows from the influence of vascular resistance on pressures in the graft circuit. As blood flows through the circuit, energy is dissipated and pressure decreases [14,18]. Thus, large vascular resistance, whether caused by stenosis or a narrow



**Fig. 5.** (A) Relation between VP/MAP and outflow stenosis when inflow and outflow stenosis increase together. The ratio of inflow/outflow stenosis rates varies from 0 to 1.0. VP/MAP increase is delayed and ultimately prevented as the rate of inflow stenosis approaches and finally equals rate of outflow stenosis. (B) Relation between VP/MAP and flow (Q). As the rate of inflow stenosis approaches the rate of outflow stenosis, a given flow is associated with lower VP/MAP. The artery/vein ratio has the median value of 0.77. Dotted line indicates VP/MAP = 0.50 (standard referral threshold [4]).

vessel, causes a large drop in pressure. If this resistance is upstream to the venous dialysis needle, then VP/MAP is reduced. This effect delays the increase in VP/MAP caused by outflow stenosis until critical stenosis is reached.

The importance of stenosis in a narrow inflow tract is shown by comparing pressure drops in the circuit when the inflow and outflow have the same percentage stenosis (Figure 2C). Recall that stenosis is defined as percentage reduction in luminal diameter when compared with normal adjacent vessel. The figure shows that the pressure drop across the inflow stenosis is larger than across the outflow stenosis. In the model, the graft had a luminal diameter of 0.60 cm whereas the outflow vein had a diameter of 0.745 or 1.00 cm. Thus, an inflow stenosis placed in the graft adjacent to the arterial anastomosis yields a higher

resistance than the same percentage stenosis in the vein adjacent to the venous anastomosis. When we consider that the median diameter of the inflow artery in our patients (0.575 cm) [6] is less than that of the graft (0.60 cm), it follows that inflow stenosis located upstream within the artery often has an even larger impact on VP/MAP.

Figure 3 shows how inflow stenosis may delay or prevent an increase in VP/MAP induced by outflow stenosis. In the presence of progressive outflow stenosis, fixed inflow stenosis flattens the VP/MAP versus outflow stenosis curve and shifts it to the right. This delays the increase in VP/MAP until critical stenosis is reached. VP/MAP then increases so rapidly that it may be difficult to detect stenosis before thrombosis during conventional monthly or twice monthly surveillance. If inflow stenosis is severe enough, it may completely prevent the increase in VP/MAP induced by outflow stenosis (Figure 4). This effect is augmented if inflow stenosis is in the generally narrower inflow artery.

Figure 5A shows the effect on VP/MAP when both stenoses progress together. As the rate of inflow stenosis approaches that of outflow stenosis, the increase in VP/MAP is delayed. This is followed by a remarkably abrupt increase as the rate of inflow stenosis reaches 99% of the rate of outflow stenosis. Then, as the two rates become equal, VP/MAP does not significantly increase at all.

Flow surveillance has an advantage in detecting such combined stenoses because flow falls regardless of stenosis locations. For example, consider a graft with flow of 500 mL/min, which is associated with a high risk of thrombosis. Figure 5B predicts that if there is no inflow stenosis, then outflow stenosis is recognized because VP/MAP has increased to 0.93. On the other hand, if inflow and outflow stenoses have increased at the same rate, then inflow stenosis causes VP/MAP to remain as low as 0.32 and the stenoses are not recognized at this low flow. Note, however, that inflow stenosis can also obscure detection of outflow stenosis by flow [23]. Inflow stenosis delays the decrease in flow until critical outflow stenosis is reached. The flow then falls so rapidly that stenosis may not be detected before thrombosis. It follows that both VP and flow are vulnerable to the inflow versus outflow stenosis interaction. Further studies are needed to improve understanding of the relative merits of the two surveillance methods.

We should emphasize that neointimal hyperplasia is not the only form of inflow stenosis that is relevant to this study. Atherosclerotic vascular disease that causes narrowing of the inflow or more central arteries [9] has a similar effect on VP. Tapered grafts with a narrow inflow are a form of inflow stenosis [10] as is treatment of steal syndrome with a band applied to the inflow of a graft. These all have the same effect on VP as inflow stenosis evaluated in this study: they obscure detection of progressive outflow stenosis.

The model makes a number of assumptions that facilitate the analysis. For example, inflow stenosis can occur at a number of locations. Asif *et al.* [7] defined the inflow tract as the arterial anastomosis, the inflow artery adjacent to the anastomosis and the graft within 2 cm of the anastomosis. They found stenoses in all three segments, but the most common location was within the anastomosis. In order to simplify the model, we placed the stenosis in the graft adjacent to the anastomosis rather than within or upstream

to the anastomosis. This avoided the complex interaction of stenosis with the anastomosis or with the junction of the graft and ligated artery (Figure 1). Similarly, although outflow stenosis most commonly occurs at the venous anastomosis [13], we simplified the model by placing the outflow stenosis in the vein adjacent to the anastomosis. In addition, stenoses in the model are symmetrical, circumferential and of uniform length. The model ignores flow in arteries and veins in the arm distal to the graft, and it considers only one configuration of anastomoses. Finally, the model ignores tortuous or branching vessels and nonuniform diameters. However, these various factors should not alter the general principles described herein [5,6,14]. Moreover, the model has received strong support from the observation that predicted pressures in the graft circuit are in good agreement with clinical studies [5,6].

In conclusion, this study improves understanding of the influence of inflow stenosis on graft haemodynamics and VP surveillance. Inflow stenosis has an effect that is similar to a narrow inflow artery: it lowers VP/MAP and causes a delay followed by a rapid increase as critical outflow stenosis is reached. Thus, the standard once or twice monthly VP measurement may not be frequent enough to detect an increase in VP/MAP before thrombosis. Future studies of VP surveillance should consider more frequent measurements. Detection of an increase in VP may require that measurements be taken as frequently as every dialysis session.

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**Conflict of interest statement.** None declared.

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