Understanding Strategies for the Treatment of Ischemic Steal Syndrome after Hemodialysis Access

Christopher L Wixon, MD, John D Hughes, MD, Joseph L Mills, MD, FACS

The recently published guidelines of the National Kidney Foundation–Dialysis Outcome Quality Initiative have focused on improving patient outcomes and survival by providing recommendations for optimal clinical practice. These guidelines firmly endorse the establishment of autogenous hemodialysis access and recommend a 40% to 50% prevalence of autogenous fistulas among all hemodialysis patients. As surgeons strive to meet these guidelines it will be necessary to extend autogenous reconstruction to older individuals, diabetics, and patients with suitable vein only in the upper arm. These individuals are at increased risk for the development of the ischemic steal syndrome. It is paramount that surgeons who perform vascular access procedures have a firm understanding of the symptoms, diagnostic maneuvers, and treatment options for the ischemic steal syndrome after hemodialysis access procedures. (J Am Coll Surg 2000;191:301–310. © 2000 by the American College of Surgeons)

More than 300,000 people suffer from end-stage renal disease in this country and approximately 180,000 use hemodialysis.¹² As vascular surgeons seek to create an optimal access for longterm hemodialysis, they are faced with a considerable challenge in hemodynamic engineering. That is, the fistula should be of adequate diameter to permit flow sufficient for hemodialysis but it should minimize the potential complications of the ischemic steal syndrome.

Although the reported incidence of ischemic steal syndrome after creating a fistula ranges from 1.6% to 8% the actual incidence may be somewhat higher secondary to a general reluctance for clinicians to recognize the syndrome.³⁻¹⁰ Preoperative factors that place individuals at risk include: female gender, age greater than 60 years, diabetes, multiple operations on the same limb, construction of an autogenous fistula, and the use of the brachial artery as the donor vessel.⁷⁻¹⁰ These three articles comprise the largest clinical reports of steal syndrome in the literature. The mean age of people in whom steal developed was 57.3, 61, and 61 years, respectively. Diabetes mellitus existed in 66%, 78%, and 81% of patients, respectively. More specific physiologic preoperative predictors of distal ischemia have not been identified.

The development of ischemic symptoms distal to an arteriovenous fistula can occur in the early (< 30 days) or late (> 30 days) postoperative interval. Although most symptoms that occur acutely are self-limiting and resolve with observation,⁹ symptoms that occur late are frequently progressive and demand more aggressive medical attention.

Most commonly patients present with extremity coolness and vague neurosensory changes—often incorrectly diagnosed as diabetic neuropathy. In more severe cases patients may report ischemic rest pain, ischemic ulceration of the digits, or intrinsic muscle weakness. Physical examination may reveal diminished peripheral pulses, pallor, and evidence of sensory or motor neuropathy. Because many of the signs and symptoms are nonspecific, clinicians should maintain a high index of suspicion in all patients with a functioning
arteriovenous fistula who present with distal extremity complaints.

Confirmatory testing can be performed noninvasively by performing digital photoplethysmography (PPG) with the fistula open and after manual compression of the fistula. Although it is quite normal to have a reduction in the amplitude of digital waveforms distal to a patent proximal fistula, the nonischemic hand should demonstrate normal pulsatile waveform contours. Patients with pronounced ischemia have monophasic, or flat, waveform contours that augment with the compression of the fistula (Fig. 1).

HEMODYNAMICS

The hemodynamics of an arteriovenous fistula have interested surgeons and physiologists since the work of William Hunter in the late 18th century. It was not until Mont Reid’s and Emile Holman’s studies in the early- to midpart of this century that the physiologic effects of arteriovenous fistulas were fully appreciated. A thorough understanding of these physiologic effects provides the necessary foundation for understanding the pathophysiology of the arterial steal syndrome.

The magnitude of blood flow through an arteriovenous fistula is a function of diameter of both the fistula and the donor artery. Classically fistulas have been classified based on the fistula diameter relative to that of the donor artery. Small fistulas are defined as having a diameter less than 75% of the diameter of the inflow artery. In such fistulas the primary determinant of the magnitude of fistula flow is fistula resistance, which varies with the fourth power of fistula diameter. The natural history of small fistulas is that the relatively sluggish flow through the fistula eventuates in thrombosis. Large fistulas have a diameter greater than 75% of that of the donor artery and the magnitude of blood flow tends to be independent of fistula resistance and diameter (Fig. 2). In large fistulas flow is primarily determined by the relative resistances at the level of the peripheral vascular bed, the donor artery, and the collateral circulation. Because most surgically created fistulas are necessarily of the large variety to provide sufficient blood flow to support hemodialysis (400 to 600 mL/min), the following discussion pertains only to this group.

Figure 2. Sigmoid curve reflecting the flow through an arteriovenous fistula as a function of fistula diameter. Blood flow in small fistulas (20% to 75% of the donor artery diameter) is directly proportional to fistula diameter. Blood flow in large fistulas is independent of the fistula diameter and depends more on the resistance of the inflow artery, the peripheral circulation, and the collateral network.
The basic components of an arteriovenous fistula (Fig. 3A) include: a common inflow (donor artery) and outflow conduit (outflow vein); a low-flow, high-resistance connection (the peripheral vascular bed); a high-flow, low-resistance connection (the fistula); and a parallel set of inflow and outflow conduits (the arterial and venous collaterals).

The creation of an arteriovenous fistula has several important effects on the flow patterns of the arterial and venous circulation. The high-flow, low-resistance arteriovenous fistula causes a transient drop in proximal arterial pressure that is rapidly compensated by an increased heart rate and cardiac output. The temporary occlusion of a chronic arteriovenous fistula results in a transient increase in the blood pressure, which is compensated by a reduction in the heart rate and cardiac output (the Nicoladoni-Branham sign). In all circumstances the direction of blood flow in the proximal artery remains toward the periphery (centrifugal) and the direction of blood flow in the proximal vein remains toward the heart (centripetal). The direction of blood flow in the artery distal to the fistula is variable and may be antegrade, retrograde, or bidirectional.

The pressure gradients that govern the direction and magnitude of blood flow in the artery distal to the fistula are compared with the flow through the crossarm of a Wheatstone bridge and are a function of the resistance ratios of the proximal artery, fistula, collateral circulation, and the periphery.
the fistula bear a complex relationship to the hemodynamic resistances of the proximal artery, the arterial collaterals, the fistula, and the peripheral vascular bed ($\delta dP = QR$, where $\delta dP$ is the pressure gradient, $Q$ is the flow, and $R$ is the resistance). Sumner’s description of the relationship using a simple electrical analogue (Ohm’s Law) provides the best conceptual understanding. In this model the artery immediately distal to the fistula is represented by the crossarm of a Wheatstone bridge (Fig. 3B). The problem of flow through the crossarm of the Wheatstone bridge was first solved by Charles Wheatstone in 1843. Traditional methods of solving the equation require solving six simultaneous loop equations. A more recent technique, Maxwell’s method, uses both of Kirchoff’s rules and reduces the number of simultaneous equations to three. The solution demonstrates that the pressure gradient between the two points is dictated by the resistance ratios of inflow to fistula and collateral to peripheral vascular bed. The pressure in the artery at the level of the fistula (Fig. 3A, point C) is a function of the ratio between the resistance of the proximal artery (Fig. 3A, segment BC) and the fistula (Fig. 3A, segment CE). The pressure in the artery distal to the fistula (Fig. 3A, point D) is a function of the ratio of resistance of the arterial collaterals (Fig. 3A, segment BD) to the distal vascular bed (Fig. 3A, segment DE). The importance of these relationships cannot be overemphasized because the pressure gradient between these two points will dictate the direction and magnitude of the blood flow in the artery distal to the fistula (Table 1).

Using this model it is possible to predict the effect of varying individual portions of the circuit. For example, the model correctly predicts that increasing one’s peripheral vascular resistance would favor the development of steal; increasing fistula resistance would favor antegrade flow in the distal artery. From a practical standpoint, deprivation of adequate distal perfusion by the presence of either retrograde flow or low magnitude antegrade flow in the artery distal to the fistula defines ischemic steal syndrome.

Theoretically the presence of a large arteriovenous fistula always reduces the perfusion to more peripheral tissues. This is evidenced by the fact that the perfusion pressure is always lower distal to an arteriovenous fistula. Under usual circumstances arterial collaterals and compensatory peripheral vasodilatation are sufficient to maintain peripheral perfusion at adequate levels. The relevance and magnitude of steal becomes important only if distal arterial perfusion pressure is insufficient to satisfy distal metabolic requirements. Stated more plainly, although the direction and magnitude of blood flow in the artery just distal to the fistula is of academic interest it is the adequacy of more distal perfusion that is of practical interest.

With this in mind let us examine several clinical scenarios as they relate to the ischemic steal syndrome.

### Ischemic symptoms on dialysis

A somewhat confusing clinical scenario involves a subset of patients who experience ischemic symptoms only when being dialyzed. A common misconception concerning this phenomenon is that an increase in fistula blood flow during dialysis increases the brachial shunt fraction (fistula blood flow/brachial artery blood flow). The high capacitance outflow vein quickly dampens the pressure gradient generated by the dialysis pump, and the resistance of the fistula, inflow, outflow, and collateral vessels remains unchanged. It is unlikely that fistula shunt fractions are substantially augmented during dialysis. Rather, these patients experience a marked reduction in systemic blood pressure sec-

<table>
<thead>
<tr>
<th>Direction of flow in the artery distal to fistula (segment CD, Fig. 3A)</th>
<th>Ratio of resistance</th>
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<tr>
<td>Antegrade</td>
<td>$R_{\text{fistula}}/R_{\text{proximal artery}} &gt; R_{\text{distal vascular bed}}/R_{\text{arterial collaterals}}$</td>
</tr>
<tr>
<td>No flow</td>
<td>$R_{\text{fistula}}/R_{\text{proximal artery}} = R_{\text{distal vascular bed}}/R_{\text{arterial collaterals}}$</td>
</tr>
<tr>
<td>Retrograde</td>
<td>$R_{\text{fistula}}/R_{\text{proximal artery}} &lt; R_{\text{distal vascular bed}}/R_{\text{arterial collaterals}}$</td>
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$R$, resistance.
ondary to diminished myocardial preload. The normal physiologic response to the reduction in perfusion pressure at the inflow artery is a concomitant reduction in peripheral vascular resistance to preserve peripheral perfusion. In extremities with an arteriovenous fistula the additive effect of reduced systemic pressure and a chronically reduced distal perfusion pressure may exceed the compensatory mechanisms of the peripheral vascular bed. The relative reduction in proximal perfusion establishes a temporary condition of global distal ischemia that usually promptly resolves on cessation of dialysis. In general, for individuals who experience mild to moderate ischemic symptoms while on hemodialysis, our first line of therapy is to hold antihypertensive medicines the morning of dialysis.

Changes with chronicity
Establishing an arteriovenous fistula for hemodialysis has several important physiologic consequences. The increased flow velocity in both the donor artery and outflow vein serves as a potent longterm stimulus for these vessels to dilate to normalize transmural shear stress. The chronic distal ischemia will tend to maximize peripheral vasodilation and stimulate the maturation of a rich collateral network. To predict how these changes will influence the steal syndrome we must recall the relationship that exists between the vessel radius and resistance (Poiseuille’s Law). Although the principles of Poiseuille’s Law (ie, ideal fluid, laminar flow, rigid tube of fixed diameter) cannot be strictly satisfied by the current model we must recognize that the resistance offered by a conduit varies inversely with the fourth power of the radius: 

\[ R = \frac{8hL}{\pi r^4} \]

where \( h \) = viscosity constant, \( L \) = length of stenosis, and \( r \) = radius of stenosis.

The consequences of these changes in resistance can then be predicted by reexamining the ratios in Table 1. The model predicts that both lower fistula resistance and decreased collateral resistance are likely to augment retrograde flow in the artery distal to the fistula. These factors are partially compensated by the decrease in the resistance of the inflow artery. More peripherally there must be a point (presumably at the entrance of a large collateral artery) at which the flow becomes antegrade (Fig. 3A, point D).

In reality a strict boundary between antegrade and retrograde flow does not exist; there is bidirectional flow with each systole and diastole. Physiologically the location of this point is arbitrary, but anatomically this location may represent an area of low shear stress at increased risk for accelerated atherogenesis as previously described in the particle resonance theory of carotid plaque formation. This curious observation was made in a previously published report that described substantial occlusive disease in this segment of the artery in all 11 patients in whom the ischemic syndrome developed chronically after arteriovenous fistula construction.

Despite the hemodynamic changes that favor the development of steal at the level of the artery distal to the fistula, most individuals remain entirely asymptomatic. In fact augmented collateral circulation may account for the gradual improvement of distal perfusion pressure noted by some authors. Surgical intervention cannot be recommended for the mere presence of reversal of flow, but rather is reserved for patients with symptomatic, disabling, or limb-threatening ischemia.

Inflow stenosis
It has been previously documented that the presence of a proximal inflow stenosis contributes to the steal syndrome in 20% to 30% of patients who present with distal extremity ischemia. The preceding model supports this observation, because any process that increases the resistance of the inflow artery changes the resistance ratios in Table 1 to increasingly favor steal. It is for this reason that selective arteriography of the donor artery remains a critical portion of the evaluation before embarking on surgical revision to correct symptomatic steal syndrome.

Traditional anatomic predictors of hemodynamic significance may not apply to lesions proximal to an arteriovenous fistula. The increased flow velocity in the proximal artery generates nonlaminar flow patterns and increased mural shear stress, both of which contribute to an increased viscosity constant. The importance of this consideration is suggested through a derivation of Poiseuille’s Law, which predicts the pressure gradient across a stenosis:

\[ \Delta P = \frac{8hV}{\pi r^2} \]
where $L$ = length of stenosis, $\eta$ = viscosity constant of blood, $V$ = mean velocity of blood flow across the stenosis, and $r$ = radius of stenosis.

Consequently the increased viscosity constant and increased mean velocity in the proximal artery contribute to an increased pressure gradient across a lesion, and the traditional predictors of significance, based only on percent stenosis, may fail to identify hemodynamically significant lesions. It is recommended that pressure gradients across all suspicious lesions be carefully measured.

**Treatment of ischemic steal syndrome**

Successful treatment of the ischemic steal syndrome mandates that the surgeon recognize the disparity that exists between the resistances of the peripheral circulation and the fistula. Ideally the treatment would reconcile the conflicting end points of long-term fistula patency and restoration of distal perfusion.

The most commonly suggested procedure involves banding, plicating, or lengthening the fistula to increase the resistance that it offers the circuit. To gauge the precise degree of narrowing such that adequate peripheral perfusion is restored, investigators have used digital PPG testing during banding procedures. Experimentally such banding procedures demonstrate a reduction in the development of myointimal thickness. Review of clinical series in which this technique has been used demonstrates inconsistent restoration of distal perfusion and high rates of fistula thrombosis.

The inability to reconcile the seemingly competing hemodynamic effect of the fistula and the metabolic demand of the distal capillary beds was studied by Schanzer and coworkers in 1988. Although these authors also recognized that the ischemic steal syndrome resulted from a discordant relationship between the resistance in the fistula and periphery, they suggested working the equation from the side of the peripheral circulation. They suggested that a potential mechanism of inadequate peripheral circulation resulted from a poorly formed collateral network. In an attempt to reduce the resistance in the peripheral circuit, a bypass graft (in essence, a low-resistance collateral) was created between the artery proximal to the fistula and the artery distal to the fistula. This reduced the resistance ratio between the peripheral circulation and the fistula, reduced the brachial shunt fraction, and directed a greater proportion of blood flow toward the periphery. To prevent retrograde flow up the native artery distal to the fistula, the artery distal to the fistula was ligated (Fig. 4A).

Since this initial report, patients in whom this technique has been used have experienced excellent graft patency and limb salvage. Despite these results, the distal revascularization, interval ligation (DRIL) procedure has been slow to gain acceptance because of a lack of understanding of the benefit the procedure provides. It is our hope that the preceding discussion of arteriovenous fistulas will provide a framework within which the hemodynamic changes associated with this innovative procedure can be explained.

**Why it works**

The DRIL procedure offers substantial benefits over attempts at fistula banding. Although both procedures recognize and work to correct a relative disparity in the resistance of the fistula and the peripheral arteries, they do so from opposing sides of the equation.

Recall that surgically created fistulas are of the large variety and that the primary determinant of magnitude of blood flow through the fistula remains a function of the diameter of the donor artery. By definition the process of banding, or plication, must narrow the fistula sufficiently to convert a large, flow-independent fistula to a small, flow-dependent fistula, the natural history of which is to eventually thrombose.

Because the plication technique increases the resistance of the fistula it necessarily increases the resistance of the total circuit. In the face of a fixed inflow pressure the result is a global reduction in circuit blood flow, of which a smaller fraction is shunted through the fistula. Although frequently sufficient to improve peripheral perfusion, it creates a low-flow state in the fistula and induces thrombosis. The resultant graft thrombosis, which usually occurs subsequent to banding, remains predictable.

In contradistinction the DRIL procedure (Fig. 4A) reduces resistance not only within the peripheral circulation, but also within the total system. In the most simple terms the bypass graft functions as a low-resistance collateral in parallel circuitry (Fig. 4B). Recalling that the inverse of the total resistance
of a parallel circuit is the sum of the inverse of the individual resistors,

\[
\frac{1}{R_{\text{total}}} = \frac{1}{R_1} + \frac{1}{R_2}
\]

the total resistance of the parallel circuit is always lower than that of each individual resistor. By reducing the ratios of resistance between the systemic circulation and the fistula, the brachial shunt fraction is decreased and peripheral perfusion is augmented. The overall reduction in resistance in the system augments the total blood flow in the extremity such that fistula blood flows are maintained.

If a distal bypass graft was performed without concomitant ligation of the artery distal to the fistula, it could certainly augment the retrograde flow in the distal brachial artery. In the face of ligation the blood flow is left with no other choice but to follow the pressure gradient downstream to the peripheral vascular bed.

**Technical considerations**

Surgical reconstruction for the ischemic steal syndrome demands a physiologic approach. Clinical suspicion should first be confirmed with digital PPG analysis. Afterward selective arteriography of the affected limb should be performed to rule out a potential arterial inflow stenosis and to delineate the distal arterial anatomy.

At the time of reconstruction the location of the

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**Figure 4.** Distal revascularization, interval ligation (DRIL). (A) The origin of the bypass graft is several centimeters proximal to the origin of the fistula. The interposing segment of artery functions as a low-capacitance conduit and maintains near-systemic blood pressures at the origin of the bypass graft. (B) Resistance analogue of the revised circuit. The bypass graft functions as a low-resistance bypass in parallel configuration to the collateral network. This serves to reduce the total resistance of the peripheral circulation and the total circuit.
proximal bypass anastomosis in relation to the arterial pressure sink of the fistula is of critical importance. Although previously described, the significance of the arterial pressure sink has been poorly recognized. It exists secondary to the large capacitance in the outflow veins of the fistula causing the pressure on the venous side of a fistula to fall off quickly (the pressure on the venous side of the fistula approximates the central venous pressure within 1 cm of the anastomosis). Because the systemic to venous pressure gradient must exist in a continuum, a pressure sink necessarily exists on the arterial side of the fistula (Fig. 5).

In our experience locating the origin of the bypass graft 3 cm proximal to the origin of the fistula appears to be sufficient and conveniently avoids the need to approach the artery through a reoperative field. In essence the interposing segment of artery distal to the bypass graft origin, but proximal to the fistula origin, serves functionally to lengthen the fistula with a low-capacitance, low-resistance conduit. The net benefit removes the origin of the bypass graft from the pressure sink region near the fistula origin.

Given the preceding explanation there can be little logic to the argument that it is necessary to use a segment of reversed vein that contains a competent valve to prevent retrograde flow up the bypass graft. If the origin of the bypass graft is a sufficient distance proximal to the fistula origin retrograde flow could only occur if the resistance in the bypass graft exceeds the resistance of the collateral circulation. Although the reversed saphenous vein is our bypass graft of choice, we have performed several procedures with polytetrafluoroethylene (PTFE) and have noted excellent restoration of distal perfusion.

Of unclear consequences are the small collaterals between the ligated portions of the native brachial artery. Some authors have advocated ligating these branches, arguing that the augmented pressure in the more distal artery could serve to reflux flow up these collaterals back into the segment of artery that leads to the fistula. In our experience this has not been necessary.

Finally, in a minority of patients, it may not be necessary to perform both distal revascularization and interval ligation. In our recent series all 11 individuals who presented with late symptoms of ischemia after the creation of the fistula also had hemodynamically significant forearm arterial occlusive disease distal to the fistula. In these individuals, performing the bypass grafting procedure alone is probably sufficient to restore adequate perfusion (Fig. 6). Previous articles by our group have documented the technical aspects and clinical outcomes of the procedure. In light of the National Kidney Foundation–Dialysis Outcome Quality Initiative guidelines, which recommend a higher frequency of autogenous fistulas, the frequency with which steal syndromes occur is likely to increase. Additionally, hemodialysis access continues...
to be practiced by a large number of general surgeons. As such, we believe that a clear understanding of the mechanisms and treatments of the steal syndrome is paramount.

A fraction of patients exists in whom there is substantial retrograde flow in the artery distal to the fistula.\(^5,19\) That is, in addition to diverting the entire blood flow of the more proximal donor artery, the fistula also consumes flow from the arterial collateral network through retrograde flow up the artery distal to the fistula. In this situation the theoretical benefit of ligating the artery just distal to the fistula would eliminate the steal and improve the peripheral perfusion pressure. Although this has been demonstrated in an animal model,\(^19\) its clinical utility remains uncertain and we have applied it only in

**Figure 6.** (A) Angiogram demonstrating an autogenous arteriovenous fistula at the level of the brachial artery. There exists a double outflow system by the basilic and cephalic veins. Severe distal ischemia developed in this patient 18 months after creation of the fistula. Attempts to limit venous outflow by ligating the basilic vein failed to alleviate the symptoms. (B) Selective arteriogram demonstrated an occlusive lesion in the previously patent brachial artery (arrow). Because of the dynamic flow patterns distal to the fistula, this segment of artery may be at risk for accelerated atherosclerosis. (C) Intraoperative arteriogram of the completed reconstruction performed from the more proximal brachial artery to the ulnar artery. The bypass graft provided excellent restoration of distal perfusion and the patient experienced immediate relief of the ischemic symptoms.
cases of steal resulting from an end-side Cimino fistula at the wrist.

CONCLUSION
The development of the ischemic steal syndrome remains an important complication after the creation of an arteriovenous fistula. Before the description of the DRIL procedure attempts at surgical correction often failed to correct the distal ischemia or resulted in fistula thrombosis. As a result there was a general reluctance for clinicians to fully recognize the syndrome. In many centers this reluctance persists because persistent attempts at fistula banding continue to disappoint both the patient and the surgeon. The innovative design of the DRIL procedure uniquely provides a reliable means to reestablish distal perfusion without compromising the patency of the fistula and is strongly recommended as the procedure of choice in ischemic steal syndrome.

References