

Surgical salvage of the autogenous arteriovenous fistula (AVF)

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ABSTRACT

Numerous studies have shown that the native arteriovenous fistula (AVF) has better long-term outcomes than other forms of access for hemodialysis. However, complications may require challenging salvage procedures. The present review summarizes and discusses surgical solutions for AVF salvage recently proposed within the Fistula First Breakthrough Initiative. These include strategies to salvage primary failed fistulae due to early thrombotic events or lack of maturation, ischemia, venous hypertensive problems, aneurysms and infections.

Key words: Arteriovenous fistula, Primary failure, Surgical revisions, Vascular access outcomes

INTRODUCTION

The value of salvage procedures for arteriovenous fistula (AVF) dysfunction and complications has been well established (1-4). Thus, those in charge of vascular access surgery must have the knowledge and skills necessary to salvage an AVF that is failing, has thrombosed or is threatened by any other complications. Although most of these procedures can be performed under local anesthesia, usually with monitored sedation, some revisional interventions require regional or even general anesthesia because they are more challenging and longer than primary constructions. The introduction of prosthetic material for autogenous AVF revisional surgery should be avoided when possible. The present review discusses the surgical salvage techniques recommended for the most common complications of the autogenous access.

EARLY FAILURE

The incidence of early AVF failure is reported to be as high as 30%-50%, when both early thrombosis and failure-to-mature (within 90 days of surgery) are included (1, 4-6). Thrombosis that occurs immediately following surgery is usually secondary to (i) technical errors, (ii) errors in judging the vessel choice or (iii) a period of extreme hypotension. A thorough physical examination, use of preoperative vessel mapping by either the operating surgeon or a technologist experienced in vessel mapping for dialysis, and meticulous attention to detail

at surgery - along with experience - should significantly reduce these causes of early failure. At centers which have adopted an aggressive or all-autogenous approach, a higher early failure rate can be expected (7). However, a majority of these early failures can be salvaged by either endovascular or surgical intervention, or both (1-4). In cases of immediate thrombosis, the case should be re-evaluated with respect to preoperative vessel mapping findings and the intraoperative findings, including the condition of the vessels chosen. Reoperation should be immediately performed, with attention to any possible technical error or unrecognized vessel disease or inadequate vessel caliber. Thrombectomy and a contrast study should be performed to examine the vein and artery. If a lesion is not defined, revision to the more proximal artery or vein, or both, will likely be indicated. In addition, a short course of anticoagulation should be considered, especially if the patient is hypotensive immediately following the procedure. If the thrombosed AVF is beyond the immediate postoperative period, endovascular thrombectomy and fistulography is an ideal approach to both restore patency without delay as well as to identify, and often treat, the cause of the thrombosis.

FAILURE TO MATURE

Based on the Fistula First Breakthrough Initiative (FFBI) (8) and K/DOQI (1), an AVF that (i) does not show evidence of maturation (thickened wall/arterialization and dilatation) by 4-6 weeks or (ii) has signs of maturity within this time frame but not enough to permit routine event-free, 2-needle dialysis by 3 months, requires investigation and intervention. Failure of an AVF to mature or provide adequate flow for dialysis can be due to (a) a focal anatomic lesion, (b) a generalized sclerosis of the vein already damaged or diseased or (c) inadequate inflow due to low baseline systemic blood pressure and/or significant arterial occlusive disease. When an obstructing lesion is not identified, access flow should be measured to rule out low flow as the cause. Low flow is a common cause for early failure, although it is often overlooked when an anatomic cause is not found, with resulting delays in intervention, thrombosis and the prolonged use of catheters.

All AVFs should be routinely evaluated at 4 weeks to determine whether or not the AVF is maturing adequately as well as to examine for any problems (8). This practice is extremely important, since the success rate for salvage procedures is considerably higher if the dysfunctional AVF

is still patent. This 4-week interval is based on reports and opinions that the majority if not all of the causes of AVF dysfunction are readily identifiable by this time, most on physical exam alone (1, 9). In addition, it is reported that the majority of AVFs reach maximum flow by 4 weeks (1, 10, 11).

Assessment of a nonmaturity problem must be based on vessels utilized, the site chosen, type of AVF construction, systemic factors and surgeon experience. A duplex ultrasound (DU) should be performed first. After 8 weeks, it is reported that the likelihood of an AVF reaching maturity to support dialysis can be predicted if the fistula has both a minimum diameter of 4 mm and a flow of 500 ml/min (10). The most common causes for nonmaturity in the *radial-cephalic AVF* are juxta-anastomotic lesions, usually stenotic or sclerotic lesions involving the segment of vein between the anastomosis and the first venous tributary (1). On the other hand, the obstructing lesions in the *brachial-cephalic and transposed brachial-basilic AVF* are most commonly found in the body of the vein or proximal vein near the shoulder. The anatomic locations of stenoses causing AVF failure have been identified and mapped by Turmel-Rodrigues et al (12). Although early failure usually involves the vein (1, 4), an inflow etiology, i.e. focal arterial lesions, inadequate inflow due to peripheral arterial disease or diameter, or a technical anastomotic error is often the cause of AVF failure. Treatment of a *focal* stenosis, arterial or venous, will be dictated by location and suitability for balloon angioplasty (percutaneous transluminal angioplasty; PTA). PTA will usually be the initial treatment, and failure of PTA will require surgical intervention. For *extensive* venous stenosis, there are a number of surgical salvage options, whereas inadequate inflow due to an artery that is small or that has extensive disease will require moving the anastomosis to a larger, more proximal inflow artery. For *venous disease*, the type of remedial surgical revision will be based on the extent of the diseased vein segment, and will entail one of the solutions listed in Table I. As an example, the dysfunctional radial-cephalic AVF that is discovered at 4 weeks with a typical stenosis between the anastomosis and first tributary may first lead to an attempt at PTA. If PTA is not feasible or is not successful, definitive success can be expected with surgical option no. 1, 2, 3A or 3B in Table I, the option chosen depending on the extent of the diseased segment and the anatomic options. Option 3B is a common, definitive remedial procedure but will sacrifice a segment of vein used for cannulation. In the case of a dysfunctional brachial-cephalic or transposed brachial-basilic AVF with

poor flow associated with a high-grade stenosis where the vein turns downward toward the axilla ("swing-point" stenosis), PTA is indicated. Should PTA be unsuccessful or be effective only short-term, surgical intervention will be required, based on one of the surgical options shown in Table I.

LATE FAILURE

Following an extended period on dialysis with a normally functioning access, if dysfunction is identified, or thrombosis occurs, a fistulogram should be performed to clearly delineate the anatomy, both to identify the cause as well as to visualize the surrounding vessels that may be useable for a salvage procedure. Further, the incidence of multiple stenoses increases over time. In addition to possible stenoses described in the above section, additional sites of stenosis often develop at the sites of repeated cannulations, in patients where either the site-rotation or "buttonhole" (same-site) techniques have not been practiced correctly. In current practice, venous as well as arterial lesions are usually approached first by endovascular methods. However, many of these lesions are not definitively resolved without surgical revision. In any case and as a general rule, referral of AVF thrombosis for fistulography plus endovascular thrombectomy/ thrombolysis or other intervention should be done within 24 hours to increase the chance of success of any type of salvage (1, 13).

Following a period on dialysis using a radial-cephalic AVF, if the cephalic vein becomes extensively stenotic, one

option is to transpose the forearm basilic vein to the radial artery. A new antecubital AVF utilizing the outflow vein in the upper arm as the new AVF conduit can usually be constructed since this vein will already be arterialized from being the outflow of the original AVF. This will permit either a new antecubital brachial-cephalic or brachial-basilic AVF that can usually be used as soon as the postoperative swelling has subsided. Transposition of the basilic vein and possibly the cephalic vein will often be necessary. At the point where AVF salvage opportunities are becoming limited, plans for a new AVF, either using the proximal AVF vein as described above or concomitant vessel mapping for construction of a new AVF at another site, should be undertaken without delay before the new AVF opportunity is definitively lost. The patient should be evaluated for a new AVF before the old AVF is abandoned to avoid delays in obtaining the tests needed, to schedule surgery and to allow enough time for the new AVF to reach maturity.

INFECTION

Infections in autogenous fistulae are uncommon, reported to occur in less than 0.4% of cases in the postoperative period (vs. 3% in arteriovenous grafts; AVGs) and 0.2% per year thereafter (vs. 5% in AVGs) (14). Immediate postoperative infection at the surgical incision site is typically the result of a break in aseptic technique during the operative procedure. Later infections are usually the result of contamination during cannulation and may present as a superficial focal cannulation puncture site infection or, especially in diabetics or when treatment is delayed, an abscess, cellulitis, bacteriemia and sepsis. It is recommended that AVF infections be treated with vancomycin and an aminoglycoside initially, while awaiting blood and tissue culture results, at which time the choice of antibiotics can be modified (1). Antibiotic therapy should be continued for a minimum of 6 weeks, as for the treatment of subacute bacterial endocarditis (1, 15). In addition, local treatment is indicated, the nature of which should be based on the extent and location of the infection. Focal infections treated early will usually resolve with systemic antibiotics and local treatment and avoidance of cannulation near the infected site. An abscess requires immediate surgical drainage, and if the anastomosis is involved in the abscess, the AVF can usually be salvaged by taking down the anastomosis, debriding the infected tissue and vein, and constructing a more proximal AVF. If additional

TABLE I
SURGICAL OPTIONS FOR VENOUS LESIONS UNDERLYING FAILURE TO MATURE

1. Excision of the stenotic/sclerotic lesion with simple primary vein reanastomosis
2. Revision utilizing a patch or interposition vein segment
3. Exclusion of an extensively diseased vein segment and
 - A) use of an interposition vein graft or
 - B) turning down the more proximal vein or a nearby venous tributary for anastomosis to the artery
4. Extension or revision using a prosthetic segment
5. Conversion of the proximal vein to a more proximal AVF
6. Abandonment and construction of a new AVF in a different location

vein length is needed after excluding and removing the infected segment, a vein graft or nearby tributary can be used to add vein length. Aggressive treatment is required for extensive infections and abscesses, since bacteremia or sepsis can quickly lead to septic emboli, with devastating consequences.

ANEURYSM

A focal aneurysm, either single or multiple, usually results from either weakness or degeneration of the vessel wall by repeated cannulations in the same area ("one site-itis"), or proximal obstruction or both. Infection has also been implicated in some cases. Although uncommon, AVF aneurysms can rupture or become infected, thereby posing an immediate and serious threat to the patient's life. The gradual, generalized dilatation of a longstanding AVF is common and does not require intervention unless associated with complications, such as prolonged cannulation site bleeding or signs of impending skin ulceration.

However, in the extreme, the vein may exhibit diffuse, extreme aneurysmal dilatation along its entire length, usually due to chronic proximal venous obstruction. This

diffuse, aneurysmal, tortuous dilatation should raise the suspicion of a high-grade proximal outflow obstruction, especially central venous. Physical examination of such an obstructed AVF will usually reveal a hyperpulsatile vein, minimal if any thrill and a high-pitched discontinuous bruit with little or no diastolic component. Since outflow obstruction is often the cause of, or a contributing factor to, the development and progression of an aneurysm, it is critically important to obtain a fistulogram initially and treat or bypass an underlying obstruction as well as the aneurysm.

If an aneurysm has normal overlying skin and does not compromise cannulation, management consists simply of avoiding the aneurysm when cannulating, and continued observation. If an aneurysm enlarges and the overlying skin becomes thinned out, infected or compromises the ability to cannulate because of the size and extent of the aneurysm, surgical intervention is indicated. If intervention is deemed necessary, history and physical exam is important to rule out "one site-itis" and infection as possible etiologies or contributing factors, and physical examination can also often identify the signs of outflow obstruction as a cause. After obtaining a fistulogram, and addressing any abnormalities, the surgical approach should be based on the size and extent of the aneurysm (Tab. II).

TABLE II
TREATMENT OF ANEURYSMAL LESIONS

Lesion size/number	Treatment
Small, few and discrete lesions	1) Resection with direct anastomosis of the free ends of normal vein (made possible by chronic elongation of the vein), or 2) excision of the anterior wall of the aneurysmal vein with suture repair or vein patch
Larger	1) Excision of the anterior wall of the aneurysm with suture closure of the edges to approximate the lumen size of the remainder of the normal AVF or 2) interposition of a segment of autogenous vein, or prosthetic as a less ideal alternative
Extensive, multiple or confluent lesions	1) Excision of the anterior wall with primary tubular reconstruction to match the size of the normal AVF vein; or 2) a bypass of vein or prosthetic graft (possibly leaving the long aneurysmal vein segment in place to avoid meddlesome, traumatic and time-consuming dissection requiring general anaesthesia)
Diffuse, progressive degeneration of the entire AVF	1) Tubular reconstruction, possibly in 2 stages; 2) bypass with either vein or prosthetic; or 3) abandonment and vessel mapping and construction of a new AVF at a different location
Signs of bleeding, infection or ulceration; darkly colored, blister skin	Surgical intervention should be immediate to prevent life-threatening hemorrhage; any of the above options including AVF closure

VENOUS HYPERTENSION

Venous hypertension in the access extremity can result from either a side-to-side anastomosis or a high-grade proximal venous obstruction (usually central in location) or both. In the first case (typically seen with side-to-side radiocephalic AVFs), swelling, discomfort, skin discoloration and eventually ulceration can occur. After obtaining a fistulogram to rule out proximal obstruction as a causative or contributing factor, the vein just beyond the anastomosis should be ligated, thereby converting the side-to-side anastomosis to a functional end vein to side artery anastomosis. If proximal venous obstruction is the cause of venous hypertension, it is usually due to a central venous obstructing lesion involving the subclavian or nearby vein, often secondary to prior central catheter use. Focal venous obstructions more distally do not typically cause venous hypertension and swelling, since there are usually adequate venous collaterals to drain the obstructed flow. In severe cases, typically due to complete central occlusions, the entire extremity will usually be swollen, and there will often be distended collateral veins and varicosities visible on the chest wall and upper arm. On occasion, over time, collaterals may be sufficient to minimize swelling and symptoms. Initial treatment for central venous stenosis and complete occlusion is endovascular, utilizing angioplasty and possibly stents. In cases of severe venous hypertension, *bypass to the internal jugular vein* or a *turndown of the external jugular vein* to bypass the obstruction and redirect flow through the internal jugular vein are considered if (a) endovascular methods and subclavian vein reconstruction have failed or are not feasible and (b) the internal jugular and more central veins are not involved.

STEAL SYNDROME / HAND ISCHEMIA

Steal syndrome, or hemodialysis access-induced ischemia, is a clinical condition caused by profound arterial insufficiency distal to a hemodialysis AV access caused by diversion of blood into the access. Although usually associated with reversal of flow, this is not always a component, and the key causative factors include diversion of blood into a low-pressure, low-resistance access away from a high-resistance peripheral vascular bed, in the absence of adequate collateral circulation. This explains why the most severe cases of ischemia are in diabetics and other patients with distal arterial occlusive disease.

An excellent detailed description of the physiology of vascular steal and ischemia is provided by Wixon (16). Physiological steal is an expected result of most access constructions currently performed and occurs in the majority of patients with an AVF or AVG, although only a small percentage of patients demonstrate the symptoms and signs of ischemia, and an even smaller percentage present clinically with a severe ischemic syndrome, i.e. arterial steal syndrome (16-18). Approximately 10% of patients can be expected to manifest signs and symptoms of ischemia that are sensory and minimal and tolerable, such as coldness, paresthesias and mild pain (1, 17). Many of these patients improve spontaneously with time, and others may respond to simple treatment, such as wearing a glove during dialysis for hand pain. However, at the other end of the ischemia spectrum are steal syndrome and ischemic monomelic neuropathy (IMN), where delay in recognition and treatment can result in irreversible neuropathy, muscle atrophy, loss of function, gangrene and amputation. The incidence of steal syndrome is generally reported to be 1%-9% of all accesses, and it is more common with AVGs than AVFs, and with brachial and femoral artery constructions (1, 19-23).

Prevention

Since many cases of ischemia are preventable, it is critical that the surgeon be aware of the risk factors, the most significant of which include diabetes (especially with neuropathy), peripheral occlusive arterial disease, use of the brachial or femoral artery, size of the anastomosis, prior access procedures and age. As would be expected, conditions that are associated with underlying peripheral arterial disease represent the common denominator (24). Patients considered to be at high risk for developing significant ischemia, by history and physical exam, should have additional testing, which may include 1 or more of the following: Doppler-assisted Allen's test, pulse oximetry, DU, arteriography and other tests based on availability, such as measurement of digital/brachial pressure ratios (DBI). With respect to specific surgical considerations, the location and size of the anastomosis are critical. Any patient considered to be at high risk should prompt a plan prior to surgery that avoids use of the brachial or femoral arteries as inflow, if feasible. If either the distal radial or ulnar artery is not suitable for a distal AVF construction, an AVF based on the proximal radial artery (PRA) will usually serve as an excellent inflow associated with high patency rates and rarely associated with any

significant ischemic complications (25). If an upper arm AVF is being constructed and the vein will not reach distally to the PRA, the gap can be bridged by swinging over a nearby distal tributary of the AVF vein, if available, or using a short segment of vein or prosthetic graft. Although autogenous vein is the preferred choice for extension of the AVF vein, excellent outcomes without significant complications have been reported when a 6-mm segment of polytetrafluoroethylene (PTFE) was used to extend the vein, wherein the prosthetic segment was not used for cannulation (26, 27). If either the brachial or femoral artery is used as inflow, the size of the anastomosis should be limited to 4-5 mm to limit flow. Finally, if the technology is available, such as DU or a flow probe, flow measurement at surgery is extremely helpful in deciding if flow needs to be restricted - and by how much. Since the newly constructed AVF will have a higher flow at full maturity, a measured flow in a high-risk patient that is already high at surgery, i.e. >1 l/min, should prompt a flow-limiting technique at that time. It is important to note that an autogenous fistula will remain patent at low flows, so only enough access flow is needed that will just exceed the dialysis prescription. In the United States, this would be a flow of 500-600 ml/min, since most centers currently dialyze patients at a blood pump flow rate (Q_b) of 400-500 ml/min for routine dialysis.

Treatment

An ideal treatment option for access-induced ischemia is one which reverses the ischemia while preserving the access. The major treatment options that are currently being used and reported below should be evaluated based on achieving the above dual objectives, and in light of whether the ischemia is related to a low- or high-flow access in a given case (Tab. III).

Among these, AVF ligation and distal radial artery ligation are problematic because the former sacrifices the AVF whereas the latter can have serious ischemic consequences in patients with peripheral arterial occlusive disease involving the ulnar artery and palmar arch - either at the time of ligation or later in the patient's life (28). PTA is indicated for arterial stenosis, reported to be responsible for 30% of access-induced ischemia (22, 29, 30).

Distalization of arterial inflow (DAI) is an option suited to high-flow brachial-based AVFs. It is performed ligating the vein close to the brachial artery anastomosis. The vein is then extended distally to the proximal radial (or ulnar) artery as inflow. Ideally, the extension is performed using a tributary of the vein or a vein graft; otherwise a short segment of prosthetic can be used as bridge but not for cannulation. Excellent outcomes in terms of both dual objectives have been reported with this DAI method, which pro-

TABLE III
TREATMENT OF ISCHEMIC LESIONS

Treatment type	Indication
AVF ligation	The most severe cases of progressive ischemia, where IMN is suspected, where no time can be lost in attempting a remedial procedure, or where a remedial operation has failed and the ischemia continues to be severe and progressive
Distal artery ligation (DAL)	Ischemia associated with a radiocephalic AVF; to be considered only for those patients documented to have pure physiologic steal and an intact ulnar artery, palmar arch and collaterals, with no underlying arterial occlusive disease
Angioplasty	Arterial inflow stenotic lesions
Distalization of arterial inflow (DAI)	Ischemia related to a high-flow AVF (usually based on the brachial artery)
Proximalization of arterial inflow (PAI)	For brachial AVF when no distal options are available
Flow-reduction revision	Ischemia associated with high-flow accesses, i.e. > 1 l/min
Distal revascularization-interval ligation (DRIL)	Brachial artery constructions (less often for radial artery AVF) with low-to-normal access flow or high-flow access that fails flow-reduction procedure

IMN = ischemic monomelic neuropathy.

vides the benefit of restoring normal perfusion to the hand by using a smaller arterial inflow (27, 31, 32). The advantages of this procedure include its relative simplicity and limited invasiveness. In addition, the natural anatomy and distal blood flow are maintained without the need for a bypass and artery ligation. The disadvantage is that if the ischemia is associated with a diseased, normal or low-flow brachial artery, revising to a smaller distal artery may result in drastic flow reduction or marginal or inadequate access flow. Therefore, preoperative studies should measure access flow as well as evaluate the suitability of the radial and ulnar arteries.

Proximalization of arterial inflow (PAI) involves moving the anastomosis from the brachial artery in an upper arm access to the axillary artery, creating a loop configuration. Excellent results and patency have been reported (33, 34), but data for this procedure are limited. In essence, the axillary artery provides an additional high-pressure blood supply for the access and does not significantly compromise the natural flow to the extremity. The advantages of this procedure, as with the DAI construction, are that the anatomy and natural arterial flow to the extremity are preserved, and ligation of an artery (with dependence on a bypass, see revascularization) are avoided.

A flow-reduction revision procedure is recommended as an ideal initial treatment for ischemia associated with high-flow accesses (1, 34). The premise is that, since the excessive access flow has compromised distal perfusion, simply increasing the resistance in the access will reduce access flow and physiologic steal significantly and thereby permit adequate distal arterial flow to be restored. The procedure is minimally invasive and can ideally be performed under local anesthesia with a minimum of sedation. The effectiveness of this treatment is optimized if flow is measured during surgery to ensure that distal perfusion is restored and access flow is reduced to a level that is just above that which is required for adequate dialysis and to maintain patency. A safe range would be 500-600 ml/min for AVFs. If access flow is reduced to these levels and ischemia is not reversed adequately, then the patient will require a different procedure, likely a PAI or a revascularization. The benefit of being able to have the patient awake or mildly sedated, is that the resolution of the ischemic symptoms, as well as the signs, can be assessed intraoperatively - and any modification to the surgical plan can be made at that time. To ensure the greatest likelihood of success of a flow-reduction revision, access flow should first be measured as part of the preoperative assessment, to confirm that the access has high flow. In the operating

room, access flow should be measured again and distal perfusion assessed immediately following the procedure. Access flow can easily be measured using flow probe technology (Transonic Systems Inc, Ithaca, NY, USA) or DU. Clinical assessment of perfusion is accomplished through questioning the patient and use of other methods, including the change in pulse oximetry readings and wave forms preprocedure and postprocedure, as well as measuring the digital-brachial pressure index (DBI), looking for a substantial increase from the preoperative baseline (35). A number of investigators have reported success with flow-reduction procedures based on a variety of intraoperative methods used to assess flow and perfusion (34, 36-40). Classical banding performed by either ligature, tape, clip or similar focal restrictive technique in an empirical way, usually results in thrombosis or inadequate flow to support effective dialysis and is therefore ill-advised (41, 42). Failure by these methods can be expected for a number of reasons: (a) flow reduction, regardless of method, is the wrong treatment for a patient with ischemia associated with a low-flow access, and therefore, if ischemia is reversed, the access can be expected to thrombose or have inadequate flow for dialysis, (b) focal banding reaches critical stenosis abruptly (Poiseuille's law), at which point flow becomes inadequate. However, when performed properly, flow reduction can be an effective, reliable treatment for ischemia that is associated with high-flow accesses, while maintaining adequate flow for dialysis. The first order of business is to identify the patient as a candidate for a flow-reduction procedure, i.e. with a high-flow access. Access flow can easily be measured by a number of methods and technologies. A flow-reduction procedure based on a tapering or a gradual reduction in lumen diameter over at least 1 cm, along with an intraoperative method to quantify access flow and assess perfusion, is an excellent, minimally invasive and reliable method. A small vascular clamp with narrow blades is useful to determine the degree of tapering needed, and then running a baseball-type suture back and forth under the clamp. When the clamp is removed, there will be slightly higher flow than when the clamp was in place. Flow reduction cannot be expected to be as definitive as the distal revascularization-interval ligation procedure in all cases, since the latter eliminates the physiologic steal completely. However, flow reduction is minimally invasive, can be accomplished quickly and efficiently under local anesthesia - with the patient usually able to provide subjective evidence of success - and does not preclude going to a more invasive, complicated procedure if flow

reduction does not produce satisfactory results.

Since its introduction in 1998, the distal revascularization-interval ligation (DRIL) procedure has been widely used with excellent results and is generally considered the procedure of choice for access-induced ischemia (16, 20, 21, 23, 33, 42-44). Typically used for brachial artery constructions with ischemia, the procedure has also been used for radial artery constructions (30, 42). The operation involves a bypass from the artery above the access anastomosis to an artery below the access in order to perfuse the hand - along with a ligation between the origin of the access and the distal anastomosis of the bypass, to prevent distal reversal of flow into the access (Fig. 1). The preferred bypass conduit is the saphenous vein, but a prosthetic has also been used. Gradman and Pozrikidis (33) have developed a model comparing the flows and potential benefits of various procedures currently used to treat access-induced ischemia, and have found the DRIL procedure to provide the greatest increase in distal blood flow. Some surgeons choose not to perform the DRIL because of concern about the potential consequences of ligation of an artery in an already compromised extremity as well as reliance on a bypass for permanent distal perfusion. Failure of the bypass has been reported, often requiring reoperation for ischemia, but no cases of limb or digit loss have been reported with a successful, functioning DRIL. As an alternative to the DRIL, the revascularization portion of the DRIL procedure (DR), without interval ligation (IL), has been found to provide adequate restoration of distal flow in some cases, and may be considered as an initial option for those surgeons reluctant to ligate a feeding artery (33). In these cases, the take-off of the bypass should be as proximal as feasible. If the DR is not effective in reversing the ischemia, the IL can easily be added under local anesthesia.

Although it is more likely for high-flow accesses to induce ischemia, as with brachial vs. radial artery constructions, low-flow accesses can also induce ischemia in a patient with advanced peripheral arterial occlusive disease - the finding of which should prompt the surgeon to select the best initial AVF option that will avoid significant compromise of blood flow to the distal extremity in favor of the access, such as a brachial artery-based AVF. The presence of significant ischemia does not indicate whether the access has high or low flow, and it is the baseline access flow which should dictate the surgical option chosen. Choosing a flow-reduction procedure for a low-flow access will result in access failure if the flow is reduced enough to reverse the ischemia. On the other hand, a

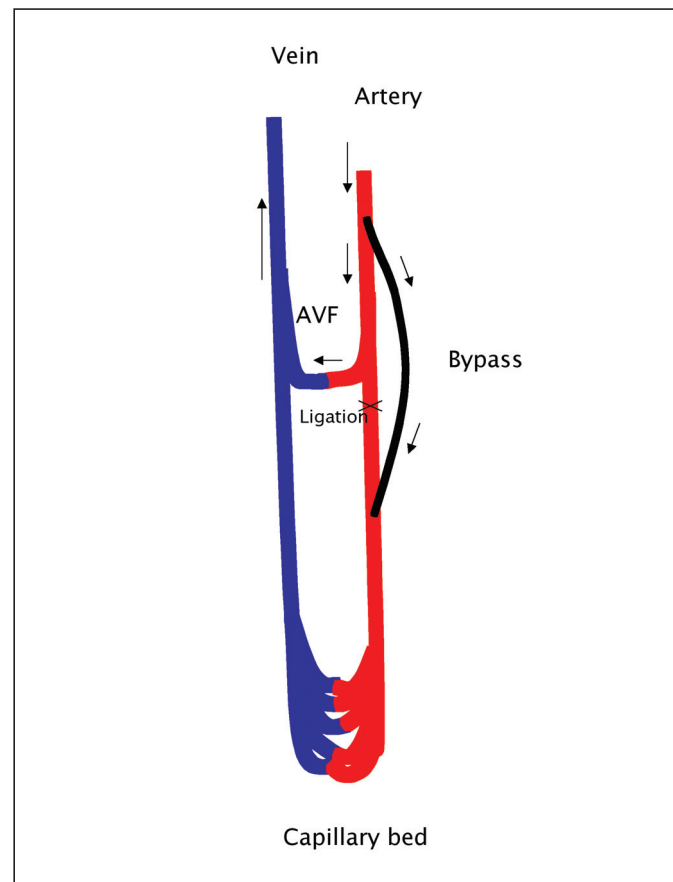


Fig. 1 - Distal revascularization-interval ligation (DRIL).

revascularization procedure such as the DRIL will work regardless of the baseline access flow. However, a revascularization procedure performed in the setting of a high-flow AVF does not address the high access flow that is also likely to cause high-output cardiac failure in many patients (45). In high-flow AVFs, the cardiac output should also be measured.

ISCHEMIC MONOMELIC NEUROPATHY

Ischemic monomelic neuropathy (IMN) is a devastating, usually irreversible ischemic injury confined to the 3 major nerves of the forearm, with profound sensorimotor dysfunction but no tissue necrosis, and thought to represent an acute ischemic injury to the vasa nervosum (44, 46). IMN, although rare and often unrecognized, has been described by a number of investigators (44, 46-49). The lesion begins at the level of the anastomosis and extends

distally, as does the degree of dysfunction. It typically presents immediately following access placement and is usually associated with normal distal perfusion and pulses. The symptoms and signs of neurologic dysfunction and injury are out of proportion to any ischemia that might be present, and the soft tissues are always spared. IMN typically occurs in diabetics, most with longstanding neuropathy, and mostly in females (1, 44, 46). Of note is the fact that all reported cases have been associated with brachial artery constructions. Symptoms include immediate onset of severe pain distal to the anastomosis, sensory loss, weakness of the hand and fingers, and then paralysis of the muscles innervated by the radial, ulnar and median nerves. The diagnosis is often missed or delayed. Diagnosis is confirmed by nerve conduction studies revealing axon loss and markedly reduced sensorimotor nerve velocities (49) and acute distal denervation of all 3 nerves at electromyography (50). The neuromuscular deterioration of IMN is considered to be independent of any coexisting ischemia, and generally proceeds regardless of surgical intervention. For these reasons, some practitioners advise no need for surgical intervention on the access, while others recommend surgery to reverse any possible access-induced ischemia, and still others recommend immediate ligation of the access (1, 44). Physical therapy is the mainstay of ongoing management for patients with IMN.

Although the progression of injury caused by IMN is not considered reversible by any surgical procedure to treat ischemia, and ligating the access also cannot be expected to reduce the impact of IMN, these authors recommend ligation because it is difficult, if not impossible, in the immediate postoperative period, to ascribe all of the acute signs and symptoms solely to IMN. In fact, some of these high-risk patients have some degree of access-induced ischemia as well. If the IMN patient has concurrent ischemia, this may lead to tissue injury and tissue loss, which is not part of IMN, but which will add to the already devastating injuries and disability related to IMN. Considering the devastating nature of IMN, and the fact that there is no way to either ensure the success of an operation for ischemia or be able to exclude that some degree of ischemia persists, ligation is the prudent option. An exception to ligation would be a patient who has no feasible remaining permanent access sites. In such a case, the IMN patient should be evaluated for any degree of concomitant ischemia and any ischemia treated by a revascularization procedure such as the DRIL. Finally, the ravages caused by severe ischemia and IMN often lead to

malpractice litigation. With respect to IMN, although the clinical decision that may be best for the patient should prevail, the decision not to intervene on the access in a patient with IMN may be indefensible if any degree of ischemia is present or develops at a later date. Therefore, thorough, expeditious assessment and discussion with patient and family about the options and associated risks are critical.

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