www.nephrologyrounds.org
JANUARY 2008
Volume 6, Issue 1

NEPHROLOGY Rounds®

Vascular Access for Hemodialysis

By DIRK M. HENTSCHEL, MD

According to the most recent United States Renal Data System (USRDS) data collection, in 2005, there were 341,319 prevalent dialysis patients.[1] Over 90% of these patients received hemodialysis, necessitating an arteriovenous fistula (AVF), arteriovenous graft (AVG), or tunneled catheter (TC) to provide access to high-volume blood flow. Adequate performance by any of these access options is required for successful hemodialysis, and creating and maintaining functional access for the large and growing number of dialysis patients poses economic challenges.

Although in 2005, patients with end-stage renal disease (ESRD) represented only 1.2% of the Medicare population, 8.2% of Medicare expenditures ($17 billion) were associated with their care. This 6.8-fold overrepresentation in cost places ESRD as a disease group ranking higher than congestive heart failure (CHF; 3-fold), chronic kidney disease (CKD; 2.9-fold), or diabetes (1.7-fold).[1] Expenditures for vascular access placement and access complications alone were over $1.5 billion. One may speculate that these costs will rise out of proportion, since diabetes and hypertension both affect the peripheral vasculature with potential detriment to long-term access options and increased vascular access complications.

In the US, vascular access care is the responsibility of numerous groups. General, vascular, or transplant surgeons place AVFs, AVGs, and TCs, and often perform endovascular procedures to treat access complications. Interventional radiologists are predominantly concerned with placement of TCs and the endovascular treatment of access complications (eg, stenoses and thromboses). More recently, nephrologists with training in endovascular or surgical procedures have begun to provide access care to dialysis patients. With so many physicians contributing to the management of vascular access, important questions are raised about the quality of care delivered to patients with ESRD. Unfortunately, prospective randomized controlled studies in this field are rare, often leaving physicians to rely on opinion and small retrospective trials. The challenge for practicing nephrologists is to become knowledgeable in conducting hemodialysis care for their patients. This issue of *Nephrology Rounds* provides the knowledge base for understanding vascular access decisions and procedures.

History of vascular access

Vascular access for hemodialysis is closely tied to the history of dialysis. In 1924, Georg Haas in Germany performed the first hemodialysis treatment in humans. In a 15-minute procedure, he used glass needles to access the radial artery and return blood into the cubital vein.[2] Until 1929, with 11 treatments in uremic patients, Haas used fractionated dialysis to clear ~400 mL of blood at a time, using an artificial kidney made up of 3 glass cylinders with U-shaped collodion tubes. In 1943, Willem Kolff, a young physician from The Netherlands, developed a "rotating drum kidney" with a larger filter surface area made of cellophane membrane. The first patient he dialyzed was a 29-year-old housemaid with CKD. She received 12 dialysis treatments, but the therapy was stopped because of a lack of access sites, since placing each cannula required a cut down to the artery.[3] In 1946, Kolff came to the US, and eventually to Boston, where he worked with John P. Merril and Carl Walter, to construct the "Brigham Artificial Kidney."

The challenge of repetitive vascular access prevented dialysis from becoming a routine method for treatment in chronic renal insufficiency. This changed dramatically in the 1960s when the idea of connecting artery and vein with rubber tubing and a glass cannula, originally from Nils Alwall, Sweden, was developed by Quinton, Dillard, and Scribner into an arteriovenous Teflon® shunt.[4] Their first patient, Clyde Shields, survived for >10 years after the insertion of his first Teflon AV shunt in March 1960. The tapered ends of 2 thin-walled Teflon cannulas were inserted into the radial artery and the adjacent cephalic vein in the distal forearm. While not on dialysis, the external ends were connected by a curved Teflon bypass tube and later

AS PRESENTED IN THE ROUNDS OF
THE NEPHROLOGY DIVISION OF
BRIGHAM AND WOMEN'S HOSPITAL
BOSTON, MASSACHUSETTS

BRIGHAM AND
WOMEN'S HOSPITAL

HARVARD
MEDICAL SCHOOL
TEACHING AFFILIATE

Brigham and Women's Hospital
Website: www.brighamandwomens.org/renal

***Nephrology Rounds* is approved by the Harvard Medical School Department of Continuing Education to offer continuing education credit**

replaced by a flexible silicon rubber tubing. As with the availability of cellophane for the earliest artificial kidneys and heparin to maintain anticoagulation, the invention of Teflon and silicon allowed for new medical devices.

In 1966, Brescia, Cimino, Appell, and Hurwich published their landmark account of 14 side-to-side-anastomoses between the radial artery and the cephalic vein at the wrist,[5] the native AVF was born. One year later, Sperling presented 15 patients with end-to-end-anastomoses[6] and, in 1968, Röhl presented results from 30 patients with radial-artery-side-to-vein-end anastomoses.[7] The different anastomosis techniques each fit specific anatomic needs and physiological considerations. In 1977, the Gracz-fistula[8] was presented and then modified by Klaus Konner;[9] this was a proximal forearm fistula that relied on the perforating vein from the superficial to the deep forearm venous system to limit blood flow in the fistula and prevent steal in patients with peripheral artery disease due to age, hypertension, or diabetes.

The Teflon shunt is an early example of an implanted medical device that is partly internal and partly external. In 1961, Shaldon, unable to find a surgeon to place the necessary dialysis cannulae, inserted catheters into the femoral artery and vein with the Seldinger-technique.[10,11] Now, dialysis catheters have evolved for placement with a cutaneous tunnel in which a cuff at the catheter's neck ensures secure placement and a relative seal against microorganisms. Catheters for acute dialysis are made of the relatively rigid polyethylene, while TCs are made of silicone, polyurethane, or carbothane (polyurethane/polycarbonate co-polymers). Silicone catheters are very soft, require a larger wall thickness to prevent collapse, and are greatly weakened by iodine. Polyurethane has high material strength that allows for thinner catheter walls and, therefore, thinner catheters with equal flow characteristics. However, polyurethane is weakened by alcohol, which today is the preferred catheter exit site cleansing solution. Carbothane combines the exceptional material advantages of polyurethane with even greater strength and tolerance of alcohol, iodine, and peroxide cleaning solutions.[12] Tunneled dialysis catheters are now also heparin-coated, based on studies in central venous catheters documenting decreased rates of thrombosis and infection.[13] For two decades, subclavian placement of dialysis catheters was the preferred route;[14] however, this was abandoned, after venograms revealed the frequent presence of severe stenosis or venous occlusion at subclavian cannulation sites.

Material sciences have continued to evolve in developing grafts for use in hemodialysis. In 1969, George Thomas attached Dacron® patches to the common femoral artery and vein, which were then connected with a silastic tube and brought to the surface of the anterior thigh.[15] The Thomas shunt was soon replaced by the expanded polytetrafluoroethylene (PTFE) graft, when LD Baker, in 1976, presented the first results in 72 hemodialysis patients.[16] The PTFE graft has remained the graft-material of choice, although grafts made of biological materials have been available since 1972.[17] Recent strategies are aiming at drug-eluting grafts to enhance the functional life of the access,[18] and tissue-engineered blood vessels from autogenous fibroblasts and endothelial cells.[19]

In parallel with the advances in surgical techniques and material science, the diagnostic techniques for vascular access care have expanded dramatically, primarily the technology around minimally invasive imaging and endovascular procedures. Seldinger's idea of an endovascular guide wire over which diagnostic and therapeutic devices can be positioned is the basis for much of this development.[10] In 1972-1973, Thelen[20] and Staple[21] independently described the angiographic retrograde imaging of AVFs in hemodialysis patients. Ergun, in 1979, first described a computerized fluoroscopy technique that revolutionized noninvasive cardiovascular imaging;[22] it is now widely used in the diagnostic and therapeutic evaluation of dialysis access. Dotter[23] and Grüntzig[24] advanced the use of catheter-guided balloon angioplasty and, in 1982, it was first used in hemodialysis AVFs and AVGs by Gordon and Glanz.[25]

Physiology of types of vascular access and clinical considerations

The appropriate access for a particular patient should be determined prior to placement, taking into consideration patient factors such as life expectancy, co-morbidities, and status of the venous and arterial vascular system. Other factors are determined by the type of access itself, as AVF, AVG, and TC differ in their effect on the circulatory system, the duration of their functionality, and the risk for infection and thrombosis.

Arteriovenous fistula

A mature AVF is the preferred type of vascular access; it has the lowest long-term complication rates for thrombosis (~ one-sixth of AVGs) and infection (~ one-tenth of AVGs).[26] In fact, the primary patency rate of AVFs 5 years after creation is >50%, while <10% of AVGs are still patent.[27] There are 3 types of AVFs:

- Simple direct fistulas, where artery and vein are connected in their natural position, either with a side-to-side or a side-artery-to-vein-end anastomosis.
- Transposed vein fistulas, where a vein is moved to connect to an artery in end-to-side fashion to either bridge a larger anatomical distance, or to bring the vein to the surface where it is accessible for cannulation. This procedure requires a tunnel to position the vein in its new location.
- Translocated vein fistulas, where a vein is removed from its anatomical location and, similar to the placement of a graft, is connected to an artery and vein in end-to-end fashion and requires the formation of a tunnel.

Each type of surgical anastomosis has inherent advantages and disadvantages.[28] A large anastomosis in the side-to-side technique, without ligating the distal venous limb, may result in venous hypertension, a problem often seen in the original Brescia-Cimino fistula. The side-to-side technique, however, is technically simple. End-to-end anastomoses, popular in the 1970s for their good hemodynamic properties, are now rarely performed, since the complete disruption of the artery imposes a high risk for peripheral ischemia and thrombosis of the fistula results in arterial thrombosis. The most common surgical technique today is the side-to-end anastomosis; it allows a greater distance between artery and vein and the surgical suturing technique is straightforward. However, it is often difficult to determine the appropriate angle between artery and vein and difficult to prevent torsion of the vein along its axis. This is of particular concern with the transposition of veins, where the vein is mobilized over a long distance and placed in a new tunnel. Torsion of the vein will prevent maturation and lead to stenosis. In addition, the diameter of the anastomosis may need to be adjusted by cutting the end of the vein in an oblique angle. In general, an anastomosis more proximal in the arterial system should be smaller to prevent steal and limit maximal fistula flow, with the inherent complication of ischemic steal or heart failure.

While fistula creation is a procedure with low morbidity and often performed under local anesthesia, fistulas require time for maturation. Data from the Dialysis Outcomes and Practice Patterns Study (DOPPS) indicate that AVFs should mature at least 14 days before use.[29] Early access of AVFs may need to be combined with lower initial blood flow rates in the range of 200–300 mL/min and smaller dialysis needles. Flow can then be increased over the course of 8–12 weeks, and needles advanced to regular size as fistula maturation is monitored by physical examination. Fistula size and flow increase over time, which is good, but in some patients these increases may pose an undue demand on the cardiovascular reserve and, in extreme cases, fistulas may require ligation.

Placement of AVFs should be initiated when the patient reaches CKD stage 4, or within 1 year of the anticipated start of dialysis. Blood draws in patients beginning with CKD stage 3 should spare both extremities and take place in the hands. A physical examination should document blood pressure differences between the upper extremities[30] and an Allen test should be performed, since the lack of a well-developed palmar arch potentially leads to steal symptoms with forearm fistulas. Ultrasound can provide valuable information for maximal surgical success by mapping arteries and veins; eg, a preoperative arterial lumen diameter >2 mm is associated with successful fistula maturation,[30] while a diameter of <1.6 mm predicts failure.[31] Venous lumen diameter >2.5 mm, absence of obstruction, and continuity with the central veins are venous predictors of AVF success.[30] The rule of 6s, as stated in the Kidney Disease Outcomes Quality Initiative (KDOQI) Vascular Access guidelines, suggests that a working AVF should have a blood flow >600 mL/min, a diameter >0.6 cm with discernible margins, and be at a depth of 0.6 cm (between 0.5 and 1.0 cm) from the surface 6 weeks after creation. In fistulas that are maturing successfully, flow increases rapidly post-surgery, from baseline values of 30–50 mL/min to 200–800 mL/min within 1 week, generally reaching flows >480 mL/min at 8 weeks.[32,33] As a result, AVFs must be evaluated 4–6 weeks after placement, and experienced examiners (eg, dialysis nurses) can identify non-maturing fistulas with 80% accuracy.[34] The physical examination of vascular hemodialysis access is a skill that nephrologists should develop.

Complications of AVFs can be divided into early and late causes. Early causes include inflow problems such as small or atherosclerotic arteries, or juxta-anastomotic stenosis. The etiology of this acquired lesion is not entirely clear, but may be related to manipulating the free end of the vein, torsion, poor angulation, or loss of the vasa vasorum during anatomic dissection. This lesion often can be adequately treated with angioplasty[35,36] or by surgical revision.[37] Outflow problems may include accessory veins that divert blood flow from the intended superficial vessel to deeper conduits, or central venous stenosis in patients with prior central venous catheters. Accessory veins can be ligated if they appear clinically significant. Vessels smaller than one-fourth of the fistula diameter are usually not hemodynamically relevant. Juxta-anastomotic stenosis and accessory veins are the most common causes for early failure AVFs when pre-operative evaluations for suitable access sites have been performed.[38]

Late causes for failure of AVFs include venous stenosis, thrombosis, and acquired arterial lesions such as aneurysms or stenosis. Venous stenosis may become apparent as flow decreases over time, worsening weekly Kt/V ([dialyzer clearance × time]/body volume) or increasing recirculation. Native fistulas typically will not thrombose until flow is severely diminished. Static pressure measurements, which are helpful in graft monitoring, do not appear as helpful in AVFs, since collaterals surrounding the stenosis area often develop, effectively masking the rise in fistula outflow resistance. Stenotic lesions can be treated by angioplasty. Thrombectomy of fistulas, although technically more challenging than in AVGs, is often successful and if flow is re-established, primary patency is longer than in grafts.[39] Aneurysms may form over the course of years as the fistula increases with increased flow and, unless associated with stenotic lesions, are more a cosmetic than functional concern. If the skin overlying the aneurysm is blanching or atrophic, or if there are signs of ulceration or bleeding, surgical evaluation should be obtained urgently. Rupture of such aneurysms in high-flow fistulas can lead to exsanguination and death.

Much has been written about the differences in prevalence of fistulas in the US and elsewhere in the world. In the first DOPPS study of vascular access conducted between 1996 and 2000, only 24% of US dialysis patients were dialyzed with an AVF, but 58% with an AVG.[40] Comparatively, the use of AVFs in Europe ranged from 67% in the United Kingdom (UK) to 90% in Italy. The "fistula first" effort has successfully increased the prevalence of AVFs and decreased that of AVGs.[41] However, the number of TCs has also increased, and those placed for bridging a patient to a functional AVF may stay in place longer.[1] Studies about fistula placement success from the US and European countries differ significantly in the primary patency rate of AVFs at one year. US studies that include diabetic patients report patency rates as low as 40%–43%.[42,43] An important difference is that European studies are usually conducted in centers that perform a large number of hemodialysis access procedures each year, whereas 2 US studies included 87 AVFs from a 9-year period (9 per year) and 150 from 5 years (30 per year). Konner from Germany, reports a primary patency rate in diabetic patients of 69%–81%, depending on gender and age, and he performs 150 AVFs per year (results reported from 748 AVFs over 5 years).[44] Chemla's team in London, UK, performed 552 AVFs in 4 years, achieving a primary patency rate at 22 months of 80% in 153 patients with radiocephalic fistulas.[45] Interestingly, his personal results as the experienced consultant are superior to that of the junior surgeons performing surgery under his direct supervision (93% vs 81% secondary patency at 22 months, $P<0.01$). These data suggest that case number and experience matter for successful AVF placement, and that nephrologists should be striving to build strong relationships with a limited number of access surgeons who are dedicated to dialysis patients and knowledgeable in access care (Table 1).

Arteriovenous graft

Until very recently, AVGs were the most commonly used type of dialysis access in the US;[1] however, they do not

Table 1: Primary patency of AVFs is related to procedures performed per year

Author	Total no. of AVF (years)	AVFs per year	1° patency at 1 year (%)
Hodges[42]	87 (9)	9	43
Leapman[43]	150 (5)	30	41
Konner[44]	748 (5)	150	69-80
Fassiadis[45]	552 (4)	130	80

last as long as AVFs and have higher rates of infection and thrombosis.[26] Grafts can be placed in the forearm, the upper arm, and the thigh, and can have a straight, curved, or loop configuration. Due to their long subcutaneous course, they offer a large surface area for cannulation and can be used about 2 weeks after placement. This interval is needed to allow for the post-surgical edema to recede and, more importantly, for the surrounding tissue to adhere to the PTFE conduit, such that bleeding into the tissue after needle removal is minimized.

The natural course of AVGs is thrombosis due to venous stenosis caused by neointimal hyperplasia. This lesion is histologically characterized by the presence of smooth muscle cells, myofibroblasts, and vascularization within the neointima. There is also adventitial angiogenesis and, in contrast to venous stenosis in AVFs, there are numerous macrophages in the tissue around the graft.[46,47] Within the neointimal lesion, growth factors (GF) such as PDGF (platelet derived), VEGF (vascular endothelial), and basic FGF (fibroblast) are present.[46] Vascular endothelium is regulated by the presence of shear stress,[48,49] and it is likely that flow within AVGs is different from native veins. Understanding the pathophysiology of neointimal hyperplasia would allow for targeted therapy, and make AVGs a better alternative for patients without suitable vessels for fistula placement. Current studies are evaluating radiation,[50] decoy peptides against transcription factors,[51] and local delivery of drugs with cell-cycle inhibitory effects (eg, paclitaxel[52] and sirolimus) to prevent neointimal proliferation. Cell-based strategies seek to take advantage of endothelial progenitor cells that release endogenous inhibitors of proliferation and thrombosis, such as nitric oxide (NO) and prostacyclin. The challenge is to direct these cells in sufficient numbers to the intended location, but a trial utilizing a receptor-coated stent was unsuccessful.[53] It is likely that advances in science and technology will generate treatment options to enhance durability of AVGs; however, it should be recognized that AVFs with proper surgical planning and in experienced hands already provide outcomes that AVGs hope to achieve.

Venous stenosis in AVGs leads to decreased flow and thrombosis, at a rate of 1–1.5 times/patient/ year.[26] In most cases, thrombosis is associated with anatomical stenoses, which are mostly located at the venous anastomosis (60%), followed by the peripheral vein (37%), and within the graft (38%).[54] The percentages indicate that many grafts have >1 stenosis at the time of diagnosis. Percutaneous angioplasty is safe and effective in treating venous stenosis.[55] Success rates in dilating a venous stenosis range from 80%–94%, and primary patency (ie, no further intervention) is around 60% at 6 months and 40% at 1 year; venous stenosis recurs. Placement of self-expanding nitinol endovascular stents appears to prolong patency in cases where focal lesions are resistant to repeated angioplasty and recur.[56] In this context, it is important to realize that treatment of stenotic lesions is not based on abnormal angiograms alone, but requires concomitant clinical finding.[57] Central stenosis is technically more difficult to treat, and stenotic lesions often recur within 6 months.

Thrombosis of an AVG is usually the result of >1 factor; in addition to a stenosis, there may be hypotension, complications of dialysis needle placement, and excessive compression for hemostasis. It is helpful to inquire about the presence of such factors to prevent their recurrence in the future. The risk for thrombosis increases with decreasing blood flow (BF); for example, May found a 19% risk of thrombosis in any 3-month period for an AVG with BF between 1010 and 1395 mL/min.[58] This risk increased continuously with decreased BF; 1.67-fold at a BF of 650 mL/min, and 2.39-fold at a BF of 300 mL/min.

The AVG thrombus consists of a relatively firm arterial plug and a softer red thrombus. The arterial plug often adheres to the wall of the graft at the site of least shear stress (= high turbulence). Graft thrombosis can be treated in outpatients by endovascular therapy. Angiographic search for a venous stenosis is always appropriate, and angioplasty is often indicated. Timely pharmacological thrombolysis or mechanical removal of the thrombus with a Fogarty catheter, and thromboaspiration or thrombectomy with a mechanical device[59] can prevent placement of a dialysis catheter.

In contrast to AVFs, flow in AVGs does not commonly increase over time, since the luminal diameter does not increase. Pressure drops slowly along the graft, in comparison to AVFs, where most of the pressure drop occurs at the arterial anastomosis, rendering the fistula itself a low-pressure capacity vessel. The continuous drop in pressure and the lack of side branches make AVGs suitable for surveillance of function by obtaining static intra-access pressure ratios, a less costly method of surveillance than flow measurement by ultrasound dilution. Normal intra-access pressure ratios have been determined for AVGs (and AVFs), and dialysis access thrombosis is decreased when patients with abnormal measurements are referred for angiography and angioplasty of stenotic lesions.

AVG infections are serious complications; they contribute prominently to vascular access difficulties and are the second leading cause for a loss of dialysis access. The incidence of hemodialysis-related bacteremia is more than 10-fold higher in AVGs than AVFs: 2.5 episodes per 1000 dialysis procedures versus 0.2.[60] Patient hygiene is the most important modifiable risk factor,[61] but needle-insertion techniques and skin cleaning prior to cannulation are important skills to teach and review.

Pseudoaneurysms should be referred to a surgeon for resection when they are >2 times wider than the graft, are rapidly increasing in size, or the overlying skin appears under duress (thin, bleeding, blanching).

Ischemia as a result of access placement is more common for AVGs than AVFs. Two important clinical entities to distinguish are:

- vascular steal syndrome; and
- ischemic monomelic neuropathy.

Physiologic steal occurs in 73% of AVFs and 90% of AVGs, since blood will flow towards the least resistance. Thus, in a radiocephalic fistula, arterial blood from the palmar arch may also deliver blood into the fistula. Unless there is the capacity for collateralization, this can lead to ischemia in the hand, ranging from complaints about cold hands to necrotic fingertips. Most of these complaints improve over time, but 1% of AVFs and up to 4% of AVGs require surgical revision.[62] Ischemic monomelic neuropathy is characterized by warm hands with a good pulse, but the hands are tender and swollen, usually immediately after surgery, and there is muscle weakness.[63] The cause is likely ischemia of the nerves and these patients need rapid surgical re-evaluation.

Tunneled hemodialysis catheter

TCs are associated with the highest infection rate and they are not a long-term access option. Ultrastructural studies have revealed that central venous catheters are colonized within 10 days of placement; however, colonization of the catheter biofilm does not correspond to positive blood cultures or clinical signs of bacteremia.[64] Usually a bridge to another type of access, they may be the only access possible for some patients with severe peripheral vascular disease or very low cardiac output.

Early causes of catheter dysfunction include kinking and unsuitable positioning of the catheter tip, both of which can be addressed under fluoroscopic guidance. Among late causes of failure, prominent features are fibrin sheaths and thrombi around or at the catheter tip. Fibrinous sheaths can be disrupted by balloon angioplasty with improved flow through a new catheter in the same location. Symptomatic occlusions of the central veins usually require the removal of the catheter and system anticoagulation and must be weighed in the context of a continued need for dialysis and other available access options.

Physical examination of the dialysis access: forgotten skills?

The physical examination of vascular hemodialysis access is a rarely practiced skill among nephrologists. This is unfortunate, as it is easily performed and can provide detailed information for the physician with trained eyes, ears, and hands.

In the physical examination of the AVF, first look for signs of prior central venous access, such as scars or pacemaker pockets. The presence of swelling or collateral veins in the chest, breast, and upper extremities should be noted. A continuous thrill, a soft pulse, and a deflation of the venous outflow with extremity elevation characterize the normal AVF. A juxta-anastomotic venous stenosis has a prominent water-hammer pulse at the anastomosis, a thrill limited to the systole, and a sudden loss of a pulse at the site of the stenosis. Occluding the venous fistula outflow and palpating for a thrill as one moves the point of occlusion up the arm can identify accessory veins that prevent maturation of the main venous outflow. Once the occlusion point is beyond the branch point of the accessory vein, the thrill will be palpable and a ligation of the identified vessel can be initiated. Augmentation is a test where one finger palpates the pulse on the arterial side of an AVF, while a finger of the other hand occludes the venous outflow. The strength of the pulse without occlusion corresponds to the outflow, while the strength of the pulse with manual occlusion is a measure of the inflow into the fistula. If the pulse is normal, then the augmentation with occlusion indicates the quality of the inflow. If there is a water-hammer pulse to begin with, then lack of augmentation indicates a severe stenosis, while moderate augmentation indicates moderate stenosis. A collapse of the AVF downstream of the lesion when the extremity is elevated, and a dilated and pulsatile segment upstream of the stenosis are also characteristics of a venous stenosis.

It is important in the examination of loop AVGs to determine the arterial and venous limb. Occluding the graft at the apex and palpating for a loss of pulse on both sides is the technique to use. The arterial side will have an augmentation in the pulse. In a normal AVG, the thrill is only palpable at the arterial anastomosis, the pulse is soft and compressible, and the bruit is low-pitched and continuous in diastole and systole. With stenosis, a thrill will be audible at the site, there will be a water-hammer pulse, and the bruit will be high-pitched and discontinuous in the systole only.

Vascular access care for hemodialysis patients is not optimal. Nephrologists should identify dedicated vascular access surgeons and interventionalists, and work to foster mutual trust and collaboration. Dialysis patients only have a limited number of vessels, and squandering any one of them is unacceptable.

References

1. USRDS. The 2007 USRDS Annual Data Report. http://www.usrds.org/adr.htm. Accessed September 30, 2007.
2. Haas G. Versuche der Blutauswaschung am Lebenden mit Hilfe der Dialyse. *Klin Wochenschr.* 1925;4:13-14.
3. ISN: Video Legacy Project: Interview with Dr. Willem J Kolff. Available: http://cybernephrology.ualberta.ca/isn/vlp/trans/kolff.htm. Accessed September 30, 2007.
4. Quinton W, Dillard D, Scribner B. Cannulation of blood vessels for prolonged hemodialysis. *Trans Am Soc Artif Intern Organs* 1960;6:104-113.
5. Brescia M, Cimino J, Appel K, Hurwich BJ. Chronic hemodialysis using venipuncture and a surgically created arteriovenous fistula. *N Engl J Med.* 1966;275:1089-1092.
6. Sperling M, Kleinschmidt W, Wilhelm A, Heidland A, Klütsch K. [Subcutaneous arteriovenous shunt for intermittent hemodialysis treatment]. *Dtsch Med Wochenschr.* 1967;92:425-426.
7. Röhl L, Franz HE, Möhring K, et al. Direct arteriovenous fistula for hemodialysis. *Scand J Urol Nephrol.* 1968;2:191-195.
8. Gracz KC, Ing TS, Soung LS, et al. Proximal forearm fistula for maintenance hemodialysis. *Kidney Int.* 1977;11:71-75.
9. Konner K. Primary vascular access in diabetic patients: an audit. *Nephrol Dial Transplant.* 2000;15:1317-1325.
10. Seldinger SI. Catheter replacement of the needle in percutaneous arteriography: a new technique. *Acta Radiol.* 1953;39:368-376.
11. Shaldon S, Chiandussi L, Higgs B. Hemodialysis by percutaneous catheterization of the femoral artery and vein with regional heparinization. *Lancet* 1961;2:857-859.

12. Ash SR. Fluid mechanics and clinical success of central venous catheters for dialysis – answers to simple but persisting problems. *Semin Dial.* 2007;20:237-256.
13. Long DA, Coulthard MG. Effect of heparin-bonded central venous catheters on the incidence of catheter-related thrombosis and infection in children and adults. *Anaesth Intensive Care.* 2006;34:481-484.
14. Erben J, Kvasnicka J, Bastecky J, et al. Long-term experience with the technique of subclavian and femoral vein cannulation in hemodialysis. *Artif Organs.*1979;3:241-244.
15. Thomas GI. A large-vessel applique A-V shunt for hemodialysis. *Trans Am Soc Artif Intern Organs.* 1969;15:288-292.
16. Baker LD Jr., Johnson JM, Goldfarb D. Expanded polytetrafluoroethylene (PTFE) subcutaneous arteriovenous conduit: an improved vascular access for chronic hemodialysis. *Trans Am Soc Artif Intern Organs.* 1976;22:382-387.
17. Chinitz JL, Tokoyama T, Bower R, Swartz C. Self-sealing prosthesis for arteriovenous fistula in man. *Trans Am Soc Artif Intern Organs.* 1972;18:452-457.
18. Fleser PS, Nuthakki VK, Malinzak LE, et al. Nitric oxide-releasing biopolymers inhibit thrombus formation in a sheep model of arteriovenous bridge grafts. *J Vasc Surg.* 2004;40:803-811.
19. L'Heureux N, McAllister T, de la Fuente L. Tissue-engineered blood vessel for adult arterial revascularization. *N Engl J Med.* 2007;357:1451-1453.
20. Thelen M, Frotscher U, Frommhold H, Kozuschek W. [Angiographic demonstration of arteriovenous shunts in dialysis patients]. *Fortschr Geb Rontgenstr Nuklearmed.* 1972;117:438-444.
21. Staple TW. Retrograde venography of subcutaneous arteriovenous fistulas created surgically for hemodialysis. *Radiology.* 1973;106:223-224.
22. Ergun DL, Mistretta CA, Kruger RA, et al. A hybrid computerized fluoroscopy technique for noninvasive cardiovascular imaging. *Radiology.* 1979;132:739-742.
23. Dotter CT, Judkins MP, Rösch J. Nonoperative treatment of arterial occlusive disease: a radiologically facilitated technique. *Radiol Clin North Am* 1967;5:531-542.
24. Grüntzig A, Bollinger A, Brunner U, Schlumpf M, Wellauer J. [Dotter's percutaneous recanalization in chronic arterial occlusions – a nonsurgical catheter technic]. *Schweiz Med Wochenschr* 1973;103:825-831.
25. Gordon DH, Glanz S, Butt KM, Adamsons RJ, Koenig MA. Treatment of stenotic lesions in dialysis access fistulas and shunts by transluminal angioplasty. *Radiology.* 1982;143:53-58.
26. Beathard G. Complications of vascular access, in *Complications of Dialysis - Recognition and Management*, edited by Lameire N, Mehta R, New York, Marcel Dekker, 2000, pp 1-27.
27. Mehta S. Statistical summary of clinical results of vascular access procedures for haemodialysis, in Vascular Access for Hemodialysis-II, edited by Summer B, Henry M, W.L. Gore & Associates, 1991, pp 145-157.
28. Konner K. The initial creation of native arteriovenous fistulas: surgical aspects and their impact on the practice of nephrology. *Semin Dial.* 2003;16:291-298.
29. Rayner HC, Pisoni RL, Gillespie BW, et al. Creation, cannulation and survival of arteriovenous fistulae: data from the Dialysis Outcomes and Practice Patterns Study. *Kidney Int.* 2003;63:323-330.
30. Silva MB Jr., Hobson RW 2nd, Pappas PJ, et al. A strategy for increasing use of autogenous hemodialysis access procedures: impact of preoperative noninvasive evaluation. *J Vasc Surg.* 1998;27:302-307; discussion 307-308.
31. Wong V, Ward R, Taylor J, et al. Factors associated with early failure of arteriovenous fistulae for haemodialysis access. *Eur J Vasc Endovasc Surg.* 1996;12:207-213.
32. Malovrh M. Non-invasive evaluation of vessels by duplex sonography prior to construction of arteriovenous fistulas for haemodialysis. *Nephrol Dial Transplant.* 1998;13:125-129.
33. Yerdel MA, Kesenci M, Yazicioglu KM, et al. Effect of haemodynamic variables on surgically created arteriovenous fistula flow. *Nephrol Dial Transplant.* 1997;12:1684-1688.
34. Robbin ML, Chamberlain NE, Lockhart ME, et al. Hemodialysis arteriovenous fistula maturity: US evaluation. *Radiology.* 2002;225:59-64.
35. Beathard GA, Arnold P, Jackson J, Litchfield T. Aggressive treatment of early fistula failure. *Kidney Int.* 2003;64:1487-1494.
36. Beathard GA, Settle SM, Shields MW. Salvage of the nonfunctioning arteriovenous fistula. *Am J Kidney Dis.* 1999;33:910-916.
37. Romero A, Polo JR, Garcia Morato E, et al. Salvage of angioaccess after late thrombosis of radiocephalic fistulas for hemodialysis. *Int Surg.* 1986;71:122-124.
38. Beathard GA. Strategy for maximizing the use of arteriovenous fistulae. *Semin Dial.* 2000;13:291-296.
39. Turmel-Rodrigues L, Pengloan J, Baudin S, et al. Treatment of stenosis and thrombosis in hemodialysis fistulas and grafts by interventional radiology. *Nephrol Dial Transplant.* 2000;15:2029-2036.
40. Pisoni RL, Young EW, Dykstra DM, et al. Vascular access use in Europe and the United States: results from the DOPPS. *Kidney Int.* 2002;61:305-316.
41. Beasley C, Rowland J, Spergel L. Fistula first: an update for renal providers. *Nephrol News Issues.* 2004;18:88,90.
42. Hodges TC, Fillinger MF, Zwolak RM, et al. Longitudinal comparison of dialysis access methods: risk factors for failure. *J Vasc Surg.* 1997;26:1009-1019.
43. Leapman SB, Boyle M, Pescovitz MD, et al. The arteriovenous fistula for hemodialysis access: gold standard or archaic relic? *Am Surg.* 1996;62:652-656; discussion 656-657.
44. Konner K, Hulbert-Shearon TE, Roys EC, Port FK. Tailoring the initial vascular access for dialysis patients. *Kidney Int.* 2002;62:329-338.
45. Fassiadis N, Morsy M, Siva M, et al. Does the surgeon's experience impact on radiocephalic fistula patency rates? *Semin Dial.* 2007;20:455-457.
46. Roy-Chaudhury P, Kelly BS, Miller MA, et al. Venous neointimal hyperplasia in polytetrafluoroethylene dialysis grafts. *Kidney Int.* 2001;59:2325-2334.
47. Swedberg SH, Brown BG, Sigley R, et al. Intimal fibromuscular hyperplasia at the venous anastomosis of PTFE grafts in hemodialysis patients. Clinical, immunocytochemical, light and electron microscopic assessment. *Circulation* 1989;80:1726-1736.
48. Paszkowiak JJ, Dardik A. Arterial wall shear stress: observations from the bench to the bedside. *Vasc Endovascular Surg.* 2003;37:47-57.
49. Corpataux JM, Haesler E, Silacci P, Ris HB, Hayoz D. Low-pressure environment and remodelling of the forearm vein in Brescia-Cimino haemodialysis access. *Nephrol Dial Transplant.* 2002;17:1057-1062.
50. Misra S, Bonan R, Pflederer T, Roy-Chadhury P. BRAVO I: a pilot study of vascular brachytherapy in polytetrafluoroethylene dialysis access grafts. *Kidney Int.* 2006;70:2006-2013.
51. Corgentech: Edifoligide – Phase I/II trial in arteriovenous grafts for dialysis. 2004.
52. Melhem M, Kelly B, Zhang J, et al. Development of a local perivascular paclitaxel delivery system for hemodialysis vascular access dysfunction: polymer preparation and in vitro activity. *Blood Purif.* 2006;24:289-298.
53. Rotmans JI, Heyligers JM, Verhagen HJ, et al. *In vivo* cell seeding with anti-CD34 antibodies successfully accelerates endothelialization but stimulates intimal hyperplasia in porcine arteriovenous expanded polytetrafluoroethylene grafts. *Circulation.* 2005;112:12-18.
54. Beathard GA. Angioplasty for arteriovenous grafts and fistulae. *Semin Nephrol.* 2002;22:202-210.
55. Vesely TM. Endovascular intervention for the failing vascular access. *Adv Ren Replace Ther.* 2002;9:99-108.
56. Sreenarasimhaiah VP, Margassery SK, Martin KJ, Bander SJ. Salvage of thrombosed dialysis access grafts with venous anastomosis stents. *Kidney Int.* 2005;67:678-684.
57. Ahya SN, Windus DW, Vesely TM. Flow in hemodialysis grafts after angioplasty: do radiologic criteria predict success? *Kidney Int.* 2001;59:1974-1978.
58. May RE, Himmelfarb J, Yenicesu M, et al. Predictive measures of vascular access thrombosis: a prospective study. *Kidney Int.* 1997;52:1656-1662.
59. Vesely TM. Mechanical thrombectomy devices to treat thrombosed hemodialysis grafts. *Tech Vasc Interv Radiol.* 2003;6:35-41.
60. Taylor G, Gravel D, Johnston L, et al. Prospective surveillance for primary bloodstream infections occurring in Canadian hemodialysis units. *Infect Control Hosp Epidemiol.* 2002;23:716-720.
61. Kaplowitz LG, Comstock JA, Landwehr DM, Dalton HP, Mayhall CA. Prospective study of microbial colonization of the nose and skin and infection of the vascular access site in hemodialysis patients. *J Clin Microbiol.* 1988;26:1257-1262.
62. Schanzer H, Schwartz M, Harrington E, Haimov M. Treatment of ischemia due to "steal" by arteriovenous fistula with distal artery ligation and revascularization. *J Vasc Surg.* 1988;7:770-773.
63. Hye RJ, Wolf YG. Ischemic monomelic neuropathy: an under-recognized complication of hemodialysis access. *Ann Vasc Surg.* 1994;8:578-582.
64. Raad I, Costerton W, Sabharwal U, et al. Ultrastructural analysis of indwelling vascular catheters: a quantitative relationship between luminal colonization and duration of placement. *J Infect Dis.* 1993;168:400-407.

Upcoming Scientific Meeting

2-6 April 2008
The National Kidney Foundation
Spring Clinical Meetings
Dallas, Texas
Contact: Tel. 212-889-2210
Email: clinicalmeetings@kidney.org
Website: www.kidney.org

Disclosure: *Dr. Hentschel has no disclosures to announce in association with the contents of this issue.*

This activity is supported by an educational donation provided by

Amgen

SNELL

304-059