Vascular Access: Concepts for the 1990s

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ABSTRACT
Modern hemodialysis requires repeated reliable access to blood vessels capable of providing rapid extracorporeal blood flow. This necessity for reliable access to the circulation is the Achilles' heel of modern hemodialysis. Native and synthetic arteriovenous fistulas remain the preferred method of maintaining long-term hemodialysis access. Vascular access dysfunctions (thrombosis and infection) are the most common complications encountered in the care of end-stage renal disease patients. This article focuses on the mechanisms by which permanent vascular access for hemodialysis is obtained and deals with the common complications that result. This review will evaluate these common complications and outline new methods for improving vascular access patency. New concepts focusing on the prospective detection and correction of venous stenoses as well as on the prevention of other factors that predispose to access failure are explored.

Key Words: Hemodialysis, vascular access, thrombosis, angioplasty, fistula

Nephrologists face a serious challenge in the establishment and maintenance of vascular access for hemodialysis. Indeed, vascular access placement and complications account for approximately one fourth of all admissions and hospitalization days for hemodialysis patients (1,2). With increasing numbers of elderly or seriously ill patients on maintenance dialysis, vascular anatomy and comorbid conditions such as diabetes mellitus frequently restrict the type and number of available access sites. These limitations place additional importance on the preservation of existing grafts. Even patients with ample access options must be carefully managed because long-term survival depends on continuous angioaccess.

TYPES OF VASCULAR ACCESS
First described by Brescia and Cimino, the arteriovenous (AV) fistula constructed from endogenous vessels (Figure 1) remains the preferred form of vascular access. Typically constructed with an end-to-side vein-artery anastomosis of the cephalic vein and radial artery, these fistulas have good long-term patency and infrequently develop infectious or thrombotic complications. However, these accesses require 6 to 8 wk to mature.

Unfortunately, unacceptable arterial or, more commonly, venous anatomy frequently precludes native AV fistula construction, particularly in elderly and diabetic patients. Late referral for vascular access and the indiscriminant use of cephalic veins for phlebotomy or i.v. cannula also reduce the likelihood of successful endogenous fistula placement. Even with careful screening of patients for native fistula placement, as many as 30 to 40% of these accesses fail to develop adequately (3). Because of these limitations, less than 30% of the dialysis population of most centers have native AV fistulas.

Fortunately, AV fistulas constructed with synthetic material, most commonly polytetrafluoroethylene (PTFE), provide excellent vascular access in most patients who fail endogenous AV fistula placement (Figure 2). PTFE has good surgical handling characteristics, and grafts of this material mature in only 2 wk. Unfortunately, these grafts have higher complication rates than native fistulas (4-6). Furthermore, access survival is limited because the PTFE conduit eventually wears out from repeated cannulations.

Fistula construction with biological materials, such as saphenous vein or umbilical vein autografts or bovine carotid artery heterografts, has proven disappointing. These materials have many disadvantages including limited availability, variable size and quality, and high cost. In addition, fistula construction and revision are technically more difficult with biological grafts. Reported patency rates for these grafts vary widely but show no clear advantage over PTFE (7-10). As a result, these materials are rarely used.

Double-lumen silastic catheters with felt cuffs
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Figure 1. Endogenous AV fistula with radial artery and cephalic vein anastomosis at the wrist. Reprinted with permission from The Principles and Practice of Nephrology (3).

Figure 2. Synthetic AV fistula with PTFE graft from radial artery to cephalic vein. Reprinted with permission from Schwab et al. (65).

Figure 3. Cuffed double-lumen hemodialysis catheter inserted through a s.c. tunnel into the right internal jugular vein. Reprinted with permission from Schwab et al. (38).

(Percath [Quinton Instrument Co., Seattle, WA] and others) are primarily used as intermediate duration vascular access to allow maturation of endogenous fistulas placed in dialysis-dependent patients (Figure 3). However, these catheters can also provide acceptable long-term access (11–14) in patients who have exhausted all available access sites. These catheters are also the preferred access in patients who cannot tolerate the hemodynamic consequences of AV fistula placement, such as those with extensive peripheral vascular disease or severe cardiomyopathies. Although insufficient flow frequently complicates the use of these catheters, infection remains the major limitation to their long-term utility (14,15).

Scribner shunts (external plastic AV fistulas) have enjoyed a substantial rebirth for acute vascular access because of their function as access for continuous arteriovenous hemodialysis (CAVHD) as well as acute hemodialysis in acutely ill intensive care unit patients. Their drawbacks for outpatient use include high infection and dislodgement rates as well as permanent use of a potential fistula access site (3). Conventional percutaneously inserted, noncuffed, double-lumen hemodialysis catheters remain the preferred access for acute hemodialysis (3).

FISTULA BLOOD FLOWS

Vascular access patency and adequacy depend on fistula blood flow. Insufficient flow increases the risk of thrombosis and decreases dialysis efficacy by limiting extracorporeal blood flow. Inadequate fistula flow results in recirculation of blood through the dialyzer as dialyzed blood reenters the dialysis circuit rather than the systemic circulation. The higher extracorporeal flows necessitated by high-efficiency hemodialysis exacerbate this problem because recirculation increases as dialyzer blood flow increases (17). In severe cases, the increased recirculation and subsequent reduction in urea clearance may offset the benefit of the increased extracorporeal flows.

Unfortunately, fistula blood flows remain difficult to measure directly. Fistulograms effectively define fistula anatomy but do not evaluate function. Electromagnetic flow probes can provide direct flow measurements, but are not practical for routine clinical use. Duplex Doppler ultrasound flow studies may be useful but have not been validated by concomitant flow probe measurements. In addition, they are limited by the technical difficulties posed by the turbulent blood flow in fistulas and are extremely operator dependent. Other techniques such as thermodilution, dye dilution, or isotope dilution are also poorly vali-
dated or technically impractical for routine clinical use.

Although available data are limited, endogenous radial artery fistula blood flow, measured intraoperatively by flow probe, ranges from 200 to 300 mL/min (18). Blood flow presumably increases over the next few weeks as the proximal artery and venous runoff system dilate. In fact, flow probe studies have documented such an increase in maturing saphenous vein grafts (19). Furthermore, Doppler flow studies suggest that mature native fistulas may attain mean flows of 800 mL/min, which remain stable despite further fistula vein dilation (20).

Unfortunately, no flow probe study directly compares endogenous and synthetic fistulas and methodological differences make comparisons between existing studies difficult. However, available data suggest that synthetic grafts rapidly achieve high blood flow. Flow probe measurements of newly constructed synthetic fistulas (21) and Doppler flow studies of mature PTFE grafts show mean flows of 800 mL/min (22). However, synthetic graft flow probably decreases progressively as neointimal hyperplasia and subsequent stenoses obstruct venous outflow.

The location of the fistula may affect blood flow because more proximal arterial anastomoses appear to yield higher flows (18,22). In general, radial fistulas appear to have lower blood flows than brachial or upper arm fistulas. These differences probably relate to the size of the arterial vessel used in fistula construction. The role of venous anastomotic site in fistula flow remains unknown.

In general, dual lumen cuffed silastic catheters can not achieve extracorporeal flows comparable to native or synthetic AV fistulas. Depending on the specific intraluminal dimensions of the particular catheter, mean blood flows range from 190 to 300 mL/min with recirculation of 5 to 14% (13,14,23). In our center, we have usually obtained blood flows of 300 mL/min, with approximately 50% of catheters attaining 400 to 450 mL/min while maintaining recirculation ratios of less than 20%.

ACCESS PATENCY

Cumulative access patency rates vary considerably between dialysis centers. The differences probably reflect multiple factors such as the demographics of the local dialysis population, the expertise of the dialysis staff, and the skill and preferences of the vascular surgeons. However, most series show cumulative patency rates of approximately 60 to 65% for endogenous fistulas at 1 yr and 50 to 60% at 2 yr (5,24) and a mean patency of 3 yr (6). Cumulative patency rates for PTFE grafts range from 67 to 83% at 1 yr to 50 to 77% at 2 yr (4,5,24–26).

Native fistulas have a high rate of early graft fail-
access. Approximately 50% of these stenoses develop at the venous anastomosis of the fistula, whereas the rest occur more proximally in the venous circulation, including up to 20% that involve central veins (Table 2) (27). Lesions at the venous anastomosis develop from progressive neointimal hyperplasia (9,28) or, rarely, atherosclerosis (29). However, the histology of more proximal stenoses remains poorly characterized.

The pathogenesis of venous stenoses is incompletely understood but may be related to endothelial injury from turbulent blood flow, calcification or fibrosis of venous valves, or endothelial trauma at certain anatomic pressure points such as the elbow or axilla. Lesions within the fistula itself may be due to an organized clot at sites of frequent cannulation (30). Central vein stenosis may be related to antecedent central vein cannulation (31). Approximately one third of access thromboses occurs in the absence of an anatomic lesion (25). These episodes may result from conditions that decrease fistula blood flow, such as hypotension, decreased cardiac output, hypovolemia, or prolonged compression of the fistula during sleep. However, nonstenosis–associated access thrombosis is usually due to excessive fistula compression by dialysis staff to achieve hemostasis after dialysis. Accurate and complete records allow vigilant monitoring of this serious quality assurance issue. Dialysis staff responsible for multiple nonstenosis–associated thromboses should be identified and retrained. These measures may significantly improve thrombosis rates.

Recent data suggest that anticardiolipin antibodies may be associated with recurrent graft thrombosis (32). Other hypercoaguable states may also play a role because hemodialysis alters levels and activity of several components of the coagulation system including protein C, protein S, and antithrombin III (33,34). However, the clinical significance of these observations remains to be determined. Increased hematocrits with erythropoetin therapy may also play a role in fistula thrombosis, though available data suggest that such an effect, if any, is minimal if hematocrits are maintained at recommended levels (35,36).

Fistula thrombosis occurs more frequently in synthetic than in endogenous grafts. This complication develops in more than 50% of PTFE grafts but in only 10 to 15% of native fistulas (5,6,25). Although multiple factors such as the thrombogenicity of PTFE, turbulent blood flow, or progressive deterioration of the graft with use may be involved, the precise causes of this marked difference remain unclear.

Catheter thrombosis, with resultant insufficient flow, complicates up to 10% of dialysis with chronic indwelling cuffed catheters (15). Early catheter thrombosis is usually due to improper placement or catheter kinking. Subsequent clotting complications include sleeve, ball valve, or mural thrombus (37) or central vein thrombosis (13,15). These complications may be reduced by placement of the catheter tip in the right atrium (14). Although thrombosis occurs in over 50% of these catheters and frequently recurs, catheter removal is rarely necessary. The instillation of 5,000 U of urokinase in a volume sufficient to fill the catheter lumen restores patency within 20 min in over 90% of thrombotic episodes (38). Higher doses and longer incubations may be tried if the initial attempt is unsuccessful.

**Infection**

Infection accounts for approximately 20% of vascular access complications and is second only to thrombosis as a cause of fistula loss (4,5,39). In fact, the vascular access is the source of the majority of bacteremias in hemodialysis patients (40–43). Staphylococcus aureus and, less commonly, Staphylococcus epidermidis are the predominant pathogens (40–43). Other gram-positive cocci and various gram-negative bacteria are responsible for occasional infections (Table 3) (40–43).

Although the overall incidence is low, vascular access infection remains an important quality assurance issue because many cases develop after bacterial inoculation because of poor needle insertion technique (25,39). In addition, improper graft cannulation may lead to pseudoaneurysms or perifistular hematomas, which are predisposed to infection.

In our experience, the incidence of access infection correlates with the needle insertion skills of the dialysis personnel. This association is most striking for new hemodialysis units where infection rates fall rapidly as the staff gains expertise. Sporadic outbreaks of access-associated bacteremia are frequently associated with the poor needle insertion technique of a single staff member. A review of graft

**TABLE 2. Location of venous stenoses**

<table>
<thead>
<tr>
<th>Intrafistular and Venous-Fistular Anastomosis</th>
<th>Proximal Peripheral Vein</th>
<th>Central Vein</th>
<th>Arterial-Fistular Anastomosis</th>
</tr>
</thead>
</table>

**TABLE 3. Common pathogens in vascular access infection**

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Species</th>
</tr>
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<tbody>
<tr>
<td>S. aureus</td>
<td>S. epidermidis</td>
</tr>
<tr>
<td>Streptococcus Species</td>
<td>Gram-Negative Organisms</td>
</tr>
</tbody>
</table>
Invasive or reconstructive procedures such as angioplasty, thrombectomy, or graft revision increase the risk of access infection and necessitate periprocedural antibiotic coverage (39). Other predisposing factors for infection include i.v. drug use, dermatitis overlying the fistula, excessive scratching of needle insertion sites, and poor hygiene (43).

Although endogenous fistulas rarely become infected, access-associated bacteremia occurs with 20 to 35% of PTFE grafts (25,39) and with 12 to 38% of chronic indwelling cuffed catheters (13–15). In fact, infection is the primary limitation on catheter survival. Access location also affects the risk of infection because lower extremity accesses, particularly grafts in the groin area, have a significantly higher incidence of infectious complications (44).

Native fistula infections are usually localized and resolve with local care and appropriate antibiotic therapy (5). However, infected PTFE grafts frequently require surgical intervention in addition to antibiotics. Depending on the extent of the infection, procedures range from simple incision and drainage to partial graft excision with bypass grafting to total graft excision (5,25). These interventions can salvage 25 to 50% of infected PTFE grafts (4,5,25). Infection usually necessitates the removal of chronic indwelling cuffed catheters. However, infection has been successfully managed by changing the catheter over a guidewire with concomitant antibiotic treatment (45).

Cardiac Complications

Vascular access–related cardiac decompensation occurs rarely in patients with underlying cardiac dysfunction, particularly in those with severe cardiomyopathies (51). Such patients can develop high-output cardiac failure after access placement if fistula flow exceeds 20% of the cardiac output. Limiting fistula flow by banding may be attempted but frequently results in access thrombosis. Flow can also be reduced by the placement of a tapered inter-
Venous Dialysis Pressures

Access patency can be assessed by measurement of the pressure in the venous return line of the dialyzer circuit or venous dialysis pressure (VDP). Fistula venous outflow obstruction presumably increases intrafistula pressure, which subsequently elevates VDP. However, VDP is also dependent on the rate of extracorporeal blood flow as well as the flow and compliance characteristics of the tubing and needle of the venous return line. VDP may also vary with access type, location, and configuration (1) because VDP in PTFE grafts is reportedly higher than in endogenous fistulas (56). These confounding variables can be minimized by measuring VDP by established protocols (Table 5). For conventional hemodialysis, persistently elevated venous pressures can reliably identify patients at high risk for significant venous stenosis (54). Early correction of venous stenosis with angioplasty or fistula revision reduces thrombosis rates and prolongs access viability (54).

Unfortunately, increasing intravascular turbulence at higher extracorporeal blood flow appears to cause greater variability in VDP with resultant overlap between patients with patent fistulas and those with venous stenosis. As a result, VDP are currently of reduced utility for predicting venous stenosis in patients on high-efficiency dialysis. However, the measurement of VDP without extracorporeal blood flow (static VDP), a potentially superior diagnostic technique for these patients, is currently being evaluated (56).

Urea Recirculation

Direct recirculation of dialyzed blood through the extracorporeal circuit reduces effective urea clearance. Increased recirculation frequently occurs when the dialysis needles are improperly placed, resulting in inadequate distance between the arterial inflow and venous outflow sites. However, increased dialyzer blood flow also causes increased recirculation (17), presumably because fistula flow is insufficient to meet dialyzer blood requirements. This association is most dramatic for high-efficiency hemodialysis patients with limited fistula flow. Despite moderate or even low recirculation rates at conventional dialyzer blood flows, these patients develop extremely high recirculation rates at extracorporeal blood flows that exceed the fistula capacity because all additional flow must come from recirculated blood.

Urea recirculation is calculated from the formula

\[
\text{PERIPHERAL UREA} / \text{VENOUS (DIALYZER OUTFLOW) UREA} \times 100 = \% \text{ RECIRCULATION}
\]

\[
P = \text{PERIPHERAL UREA}
\]

\[
A = \text{ARTERIAL (DIALYZER INFLOW) UREA}
\]

\[
V = \text{VENOUS (DIALYZER OUTFLOW) UREA}
\]

Figure 4. Formula for the calculation of urea recirculation.

<table>
<thead>
<tr>
<th>TABLE 5. VDP monitoring protocol</th>
</tr>
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<tbody>
<tr>
<td>Cannulate Fistula With 15- to 16-Gauge Needles</td>
</tr>
<tr>
<td>Measure VDP at Extracorporeal Blood Flow of 200 to 225 ml/min During the First 30 min of Dialysis</td>
</tr>
<tr>
<td>If VDP &gt;150 mm Hg for three Consecutive Dialysis Sessions, Proceed With Fistulogram</td>
</tr>
</tbody>
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TABLE 6. Factors affecting urea recirculation

<table>
<thead>
<tr>
<th>Factor</th>
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<tbody>
<tr>
<td>Needle Placement</td>
</tr>
<tr>
<td>Extracorporeal Blood Flow</td>
</tr>
<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Decreased Cardiac Output</td>
</tr>
<tr>
<td>Intravascular Volume Depletion</td>
</tr>
<tr>
<td>Venous Stenosis</td>
</tr>
<tr>
<td>Arterial Stenosis</td>
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</tbody>
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TABLE 7. Urea recirculation analysis (Qb 400 mL/min)$^a$

<table>
<thead>
<tr>
<th>Dialysis Duration (min)</th>
<th>Stop-Flow Technique</th>
<th>Contralateral Arm Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>30</td>
<td>16 ± 3</td>
<td>17 ± 3</td>
</tr>
<tr>
<td>60</td>
<td>17 ± 2</td>
<td>17 ± 3</td>
</tr>
<tr>
<td>120</td>
<td>19 ± 3</td>
<td>24 ± 4$^a$</td>
</tr>
</tbody>
</table>

$^a$ Reprinted with permission from Collins et al. (55).

$^b$ $P < 0.05$.

arm accesses. In our study, we identified stenotic lesions in 80% of patients with upper arm fistulas and urea recirculation ratios more than 15% (55).

Recirculation ratios obtained by stop-flow and peripheral venous sample methods were similar at 30 and 60 min. In fact, ratios calculated by the stop-flow method remained stable throughout dialysis. However, at 120 min, the peripheral venous sample technique yielded significantly higher values. The clinical significance of differences in urea recirculation values as determined by various techniques remains to be defined. However, these results demonstrate the importance of consistent technique and timing of recirculation measurements (55).

Duplex Doppler Color Flow Studies

Duplex Doppler color flow studies may accurately measure fistula flow (20,22) and may be useful in monitoring vascular access patency (62). Serial measurements of fistula flow may provide an additional means of prospectively identifying venous stenoses. However, test utility is presently limited by cost and operator dependence.

Intravascular Ultrasound

Intravascular ultrasound provides information on the composition and precise intraluminal dimensions of stenotic lesions (Figure 5) (18). This information allows better assessment of angioplasty results and may help identify appropriate lesions for specialized interventions such as atherectomy catheters. In addition, the combined use of intravascular ultrasound and electromagnetic flow velocity probe can potentially provide accurate measurements of fistula blood flow.

Combined Fistula Assessment Monitoring

Recently, a preliminary study compared a fistula assessment monitor with clinical fistula assessment and angiography (64). This monitor measures basal dialyzer arterial and venous line pressures, intradialysis pressure flow characteristics, and recirculation (via optical density technique). Abnormal monitor parameters correlated with the presence of venous stenotic lesions and subsequent fistula thrombosis.

Fistulogram

Long considered the "gold standard" for the assessment of vascular access patency, the fistulogram provides detailed visualization of the fistula lumen, venous anastomosis, and proximal venous system. Although it allows the clinician to identify any anatomic abnormality, this study provides no information on fistula function. Furthermore, fistulogram is of limited utility as a screening test because the overall prevalence of venous stenoses in the dialysis population is relatively low. Screening techniques such as VDP and urea recirculation are used to select cases for fistulogram.

THERAPEUTIC INTERVENTIONS

Angioplasty

Percutaneous transluminal angioplasty is an excellent means of correcting venous stenoses in both...

Figure 5. Intravascular ultrasound of a brachial venous dissection (arrow) noted following successful PTA. Reprinted with permission from Davidson et al. (63).
native and synthetic fistulas (Figure 6) (65–68). Angioplasty correction of all angiographically determined >50% stenoses improves fistula function and prolongs access survival (65). Angioplasty can be performed on both anastomotic and more proximal stenoses. In fact, this intervention is the preferred procedure for central vein stenoses, which are difficult to correct surgically because of their intrathoracic location (Figure 6). Angioplasty can be performed on both stenotic and occlusive lesions. Even total central vein stenoses can sometimes be opened by angioplasty, either alone or with adjunctive thrombolytic infusion to the affected area (69). Only extremely long lesions are not amenable to angioplasty. Stenoses longer than 5 cm usually have poor technical results and should be surgically revised.

In most series, angioplasty successfully corrects over 80% of stenoses involving the fistula venous anastomosis or proximal peripheral veins (65–68) and up to 95% for central vein lesions (27). However, stenosis recurs more frequently after angioplasty than after surgical fistula revision (70). Reported 1-yr patency rates vary widely, ranging from 25 to 80% (27,66–68). Restenosis rates may vary by location, with recurrence occurring more commonly with proximal and central lesions (27). However, the great majority of restenoses are successfully treated by repeat angioplasty (27). Interventions such as metallic venous stents (71) or atherectomy catheters (72) may reduce restenosis rates. Complication rates range from 5 to 10% (66–68). The most common complications include postprocedure bleeding and venous rupture. However, the complications rarely require surgical intervention (66–68). In fact, angioplasty is usually performed as an outpatient procedure.

**Thrombolytic Therapy**

Attempts to treat fistula thrombosis with infusions of thrombolytic agents have yielded disappointing results. Access patency could be reestablished in fewer than 60% of cases, and nearly all patients required surgical access revision for recurrent thrombosis (73–77). In addition, small preliminary trials were marked by bleeding complications (74,77). However, recent dosing adjustments and technical advances have led to improved results. The “pulsed-spray technique,” which combines thrombolytic therapy with mechanical clot disruption, rapidly reestablishes access patency in 90% of cases with minimal incidence of complications (78). When combined with angiography and angioplasty of identified stenoses, 50% of fistulas remain patent at 1 yr although approximately half require repeat thrombolysis (78). However, direct comparisons of efficacy and cost with surgical thrombectomy will be needed to better define the clinical utility of this therapy.

Thrombolytic agents are extremely useful in maintaining the patency of chronic indwelling dialysis catheters. As previously described, the instillation of thrombolitics into catheter ports is frequently necessary to dissolve thrombus within the lumen or at the catheter tip. Occasionally, systemic infusions may be necessary to reestablish patency (13).
Surgical Fistula Revision and Thrombectomy

Surgical fistula revision remains the most definitive intervention for venous stenosis. This procedure has the lowest stenosis recurrence rates but has the disadvantages of necessitating hospitalization and also extension of the fistula, thereby reducing the number of sites available for later vascular access placement. Bypass of the stenosis achieves better long-term patency than patch grafting or simple thrombectomy (5). Surgical thrombectomy remains the procedure of choice for clotted fistulas. It is an outpatient procedure and retains the option of revision or angioplasty if stenoses are found.

Potential Therapeutic Advances in Maintenance of Access Patency

Pharmacological interventions to improve access patency are currently under investigation. Recent studies suggest that the renin-angiotensin system may play a role in the pathogenesis of fistula stenosis. For example, angiotensin-converting enzyme inhibitors can prevent myointimal proliferation after vascular injury (79). Antiplatelet agents may also prove useful. Combination therapy with low-dose aspirin and sulfipyrazone reduced thrombosis rates in patients with recurrent hemodialysis access thrombosis in a preliminary clinical trial (80). However, the clinical utility of pharmacological agents in maintaining fistula patency requires further evaluation. Additional therapeutic maneuvers under investigation include the endothelial seeding of grafts (81) or the use of graft matrices impregnated with fibroin (82) or other materials that will hopefully reduce graft thrombogenicity and improve patency rates.

SUMMARY AND CONCLUSIONS

The establishment and maintenance of vascular access have become increasingly important and are now significant factors in patient survival. Native and synthetic fistulas provide adequate vascular access in the majority of dialysis patients, whereas dual lumen silastic catheters offer the best option in certain patients. Thrombotic and infectious complications are the leading causes of vascular access loss. In addition, recent observations by Windus et al. confirm that comorbid conditions such as diabetes have a pronounced negative effect on fistula patency (83).

We recommend complete needle insertion and removal records to facilitate quality assurance monitoring of access thrombosis and infection secondary to poor technique by dialysis personnel. Staff members should be retrained as needed. In addition, VDP should be closely monitored for patients on conventional hemodialysis. Urea recirculation ratios should be measured periodically for patients on high-efficiency regimens. Patients identified at high risk for venous stenoses by either method should undergo fistulogram promptly. All >50% stenoses identified should be corrected by angioplasty or surgical revision before actual fistula failure. Close attention to access complications due to technical factors, new methods of prospective detection of venous stenosis, and early and aggressive correction of stenotic lesions should improve and prolong access function and patency. There does not appear to be significant difference in access thromboses between conventional and rapid hemodialysis when prospective monitoring and preventive therapy are employed (55).

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